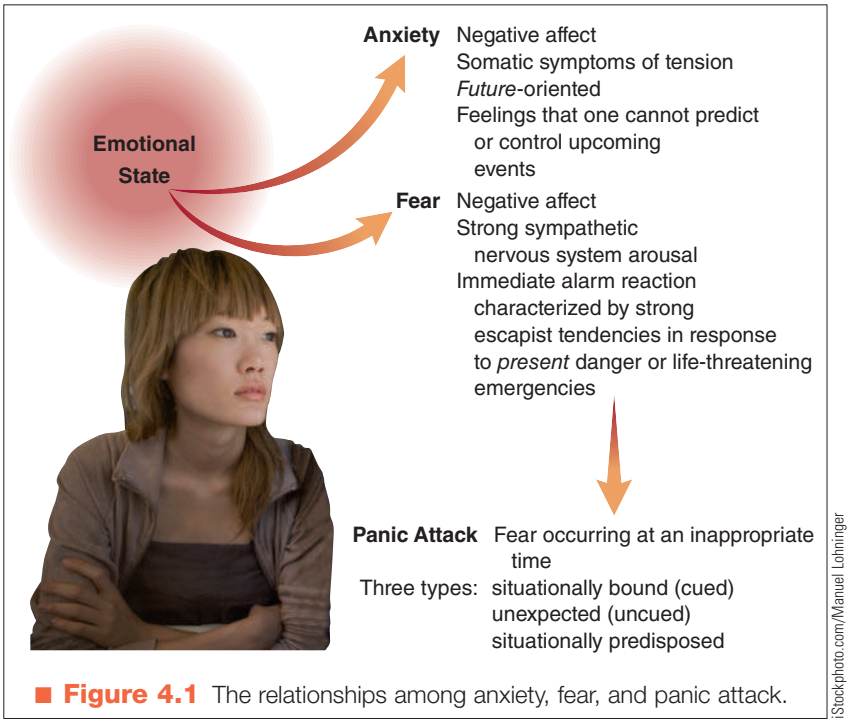
predisposed (this type of panic attack may be dropped in *DSM-5* just to make differentiation simpler). We mention these types of attacks because they play a role in several anxiety disorders. Unexpected and situationally predisposed attacks are important in panic disorder. Situationally bound attacks are more common in specific phobias or social phobia (see ■ Figure 4.1).

Remember that fear is an intense emotional alarm accompanied by a surge of energy in the autonomic nervous system that motivates us to flee from danger. Does Gretchen's panic attack sound like it could be the emotion of fear? A variety of evidence suggests it is (Barlow, 2002; Barlow, Chorpita, & Turovsky, 1996; Bouton, 2005), including similarities in reports of the experience of fear and panic, similar behavioral tendencies to escape, and similar underlying neurobiological processes.



■ **Figure 4.1** The relationships among anxiety, fear, and panic attack.

Causes of Anxiety Disorders

You learned in Chapters 1 and 2 that excessive emotional reactions have no simple one-dimensional cause but come from multiple sources. Next, we explore the biological, psychological, and social contributors and how they interact to produce anxiety disorders. (See the Discussing Diversity box.)

Biological Contributions

Increasing evidence shows that we inherit a tendency to be tense, uptight, and anxious (Clark, 2005; Eysenck, 1967; Gray & McNaughton, 1996). The tendency to panic also seems to run in families and probably has a genetic component that differs somewhat from genetic contributions to anxiety (Barlow, 2002; Craske, 1999; Craske & Barlow, 2008; Kendler et al., 1995). As with almost all emotional traits and psychological disorders, no single gene seems to cause anxiety or panic. Instead, contributions from collections of genes in several areas on chromosomes make us vulnerable when the right psychological and social factors are in place. Furthermore, a genetic vulnerability does not cause anxiety and/or panic directly—that is, stress or other factors in the environment can "turn on" these genes, as we reviewed in Chapter 2 (Gelernter & Stein, 2009; Kendler, 2006; Rutter, Moffitt, & Caspi, 2006; Schumacher et al., 2005; Smoller, Block, & Young, 2009).

Anxiety is also associated with specific brain circuits and neurotransmitter systems. For example, depleted levels of gamma-aminobutyric acid (GABA), part of the GABA–benzodiazepine system, are associated with increased anxiety, although the relationship is not quite so direct. The noradrenergic system has also been implicated in anxiety, and evidence from basic animal studies, and studies of normal anxiety in humans, suggest the serotonergic neurotransmitter system is also involved (Lesch et al., 1996; Maier, 1997; Stein, Schork, & Gelernter, 2007). But increasing attention is focusing on the role of the corticotropin-releasing factor (CRF) system as central to

DSM Disorder Criteria Summary
Panic Attack

A panic attack involves experiencing four or more of the following symptoms during a specific period:

- Palpitations, pounding heart, or accelerated heart rate
- Sweating
- Trembling or shaking
- Sensations of shortness of breath or smothering
- Feeling of choking
- Chest pain or discomfort
- Nausea or abdominal distress
- Feeling dizzy, unsteady, lightheaded, or faint
- Derealization (feelings of unreality) or depersonalization (being detached from oneself)
- Fear of losing control or going crazy
- Fear of dying
- Paresthesias (numbness or tingling sensations)
- Chills or hot flushes

Source: Based on *DSM-IV-TR.* Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision). © 2000 American Psychiatric Association.

fear Emotion of an immediate alarm reaction to present danger or life-threatening emergencies.
panic Sudden, overwhelming fright or terror.
panic attack Abrupt experience of intense fear or discomfort accompanied by several physical symptoms, such as dizziness or heart palpitations.

Discussing Diversity

Cultural Influences on Anxiety

Anxiety is experienced by people all over the world. However, significant differences are found in the reported rates of anxiety disorders and how people experience them across different countries. When asked whether they had suffered from an anxiety disorder in the past year, approximately 3% of those in China reported having an anxiety disorder, whereas 18% of those in the United States reported such a disorder (WHO World Mental Health Survey Consortium, 2004). This could reflect a true difference in the rate of anxiety across these countries but may largely result from how anxiety is experienced and reported.

For instance, the criteria used to assess the presence of anxiety disorders around the world typically are based on the Western conceptualization of each disorder. In the case of panic disorder, this includes the experience of extreme physiological distress (such as heart racing and sweating), along with fears that one is going crazy or losing control. The physiological symptoms associated with panic appear to be universal and are reported in virtually all countries. However, the psychological manifestations of panic differ across countries, and this may lead to lower numbers of people in other countries meeting diagnostic criteria for panic disorder as defined by Western standards. In many third-world countries, for example, anxiety and mood disorders are experienced as bodily sensations but symptoms like fear and dread are not reported (Hinton, Chhean, Fama, Pollack, & McNally, 2007).

In addition to differences in the experience of anxiety, some people may be more willing to report anxiety than others. This appears to be the explanation behind the large sex differences in the prevalence of anxiety disorders in the United States—that is, women consistently report higher rates of fears and phobias, whereas men are less inclined to admit to such disorders (Arrindell et al., 2003a). So what happens to men with severe anxiety that goes unnoticed and untreated? A large portion of them seem to cope with their anxiety by drinking alcohol, often leading to the development of an alcohol use disorder (Kushner, Abrams, & Borchardt, 2000).

Although more is known about sex differences in rates of anxiety disorders, researchers are just beginning to learn how cultural factors can influence the experience and reporting of anxiety disorders. It will be important for future work to continue to enhance our ability to understand and treat anxiety disorders around the world.

the expression of anxiety (and depression) and the groups of genes that increase the likelihood that this system will be turned on (Heim & Nemeroff, 1999; Khan, King, Abelson, & Liberzon, 2009; Ladd, et al., 2000; Smoller, Yamaki, & Fagerness, 2005; Sullivan, Kent, & Coplan, 2000). This is because CRF activates the hypothalamic–pituitary–adrenocortical (HPA) axis, which is part of the CRF system, and this CRF system has wide-ranging effects on areas of the brain implicated in anxiety, including the emotional brain (the limbic system), particularly the hippocampus and the amygdala; the locus coeruleus in the brain stem; the prefrontal cortex; and the dopaminergic neurotransmitter system. The CRF system is also directly related to the GABA–benzodiazepine system and the serotonergic and noradrenergic neurotransmitter systems.

The area of the brain most often associated with anxiety is the limbic system (Britton & Rauch, 2009; Charney & Drevets, 2002; Gray & McNaughton, 1996; LeDoux, 1996, 2002; see Figure 2.7c), which acts as a mediator between the brain stem and the cortex. The more primitive brain stem monitors and senses changes in bodily functions and relays these potential danger signals to higher cortical processes through the limbic system. The late Jeffrey Gray, a prominent British neuropsychologist, identified a brain circuit in the limbic system of animals that seems heavily involved in anxiety (Gray, 1982, 1985; McNaughton & Gray, 2000) and may be relevant to humans. This circuit leads from the septal and hippocampal area in the limbic system to the frontal cortex. The system that Gray calls the **behavioral inhibition system (BIS)** is activated by signals from the brain stem of unexpected events, such as major changes in body functioning that might signal danger. Danger signals in response to something we see that might be threatening descend from the cortex to the septal–hippocampal system. The BIS also receives a big boost from the amygdala (Davis, 1992; LeDoux, 1996, 2002). When the BIS is activated by signals that arise from the brain stem or descend from the cortex, our tendency is to freeze, experience anxiety, and apprehensively evaluate the situation to confirm that danger is present.

The BIS circuit is distinct from the circuit involved in panic. Gray (1982; Gray & McNaughton, 1996) and Graeff (1987, 1993; Deakin & Graeff, 1991) identified what Gray calls the **fight/flight system (FFS)**. This circuit originates in the brain stem and travels through several midbrain structures, including the amygdala, the ventromedial nucleus of the hypothalamus, and the central gray matter. When stimulated in animals, this circuit produces an immediate alarm-and-escape response that looks very much like panic in humans (Gray & McNaughton, 1996). Gray and McNaughton (1996) and Graeff (1993) think the FFS is activated partly by deficiencies in serotonin.

It is likely that factors in your environment can change the sensitivity of these brain circuits, making you more or less susceptible to developing anxiety and its disorders, a finding that has been demonstrated in several laboratories (Francis, Diorio, Plotsky, & Meaney, 2002; Stein et al., 2007). For example, one important study suggested that

cigarette smoking as a teenager is associated with greatly increased risk for developing anxiety disorders as adults, particularly panic disorder and generalized anxiety disorder (Johnson et al., 2000). One possible explanation is that chronic exposure to nicotine, an addictive drug that increases somatic symptoms, and respiratory problems, triggers additional anxiety and panic, thereby increasing biological vulnerability to develop severe anxiety disorders.

Research into the neurobiology of anxiety and panic is still new, but we have made exciting progress by implicating two seemingly different brain systems and confirming the crucial role of the CRF system and the amygdala. Brain-imaging procedures will undoubtedly yield more information in the years to come, and this has already begun to happen (Britton & Rauch, 2009; Charney & Drevets, 2002). For example, there is now general agreement that in people with anxiety disorders the limbic system, including the amygdala, is overly responsive to stimulation or new information (abnormal bottom-up processing); at the same time, controlling functions of the cortex that would downregulate the hyperexcitable amygdala are deficient (abnormal top-down processing), consistent with Gray's BIS model (Britton & Rauch, 2009; Ochsner et al., 2009).

Psychological Contributions

Evidence is accumulating (see, for example, Barlow, 2002; Suárez, Bennett, Goldstein, & Barlow, 2009) that supports an integrated model of anxiety involving a variety of psychological factors. In childhood we may acquire an awareness that events are not always in our control (Chorpita & Barlow, 1998). The continuum of this perception may range from total confidence in our control of all aspects of our lives to deep uncertainty about ourselves and our ability to deal with upcoming events. If you are anxious about schoolwork, you may worry you will do poorly on the next exam, even though all your grades have been As and Bs. A general "sense of uncontrollability" may develop early as a function of upbringing and other disruptive or traumatic environmental factors.

Interestingly, the actions of parents in early childhood seem to do a lot to foster this sense of control or a sense of uncontrollability (Chorpita & Barlow, 1998; Gunnar & Fisher, 2006). Generally, it seems that parents who interact in a positive and predictable way with their children by responding to their needs, particularly when the child communicates needs for attention, food, relief from pain, and so on, perform an important function. These parents teach their children that they have control over their environment and their responses have an effect on their parents and their environment. In addition, parents who provide a "secure home base" but allow their children to explore their world and develop the necessary skills to cope with unexpected occurrences enable their children to develop a healthy sense of control (Chorpita & Barlow, 1998). In contrast, parents who are overprotective and overintrusive and who "clear the way" for their children, never letting them experience any adversity, create a situation in which children never learn how to cope with adversity when it comes along. Therefore, these children don't learn that they can control their environment. A variety of evidence has accumulated supporting these ideas (Barlow, 2002; Chorpita & Barlow, 1998; Chorpita, Brown, & Barlow, 1998; Gunnar & Fisher, 2006; Lieb et al., 2000; Nolen-Hoeksema, Wolfson, Mumme, & Guskin, 1995; White, Brown, Somers, & Barlow, 2006). A sense of control (or lack of it) that develops from these early experiences is the psychological factor that makes us more or less vulnerable to anxiety in later life.

Most psychological accounts of panic (as opposed to anxiety) invoke conditioning and cognitive explanations that are difficult to separate (Bouton, Mineka, & Barlow, 2001). Thus, a strong fear response initially occurs during extreme stress or perhaps as a result of a dangerous situation in the environment (a true alarm). This emotional response then becomes associated with a variety of external and internal cues. In other words, these cues, or conditioned stimuli, provoke the fear response and an assumption of danger, even if the danger is not actually present (Bouton, 2005; Bouton et al., 2001; Martin, 1983; Mineka & Zinbarg, 2006; Razran, 1961), so it is really a learned or false alarm. This is the conditioning process described in Chapter 2. External cues are places or situations similar to the one where the initial panic attack occurred. Internal cues are increases in heart rate or respiration that were associated with the initial panic attack, even if they are now the result of normal circumstances, such as exercise. Thus, when your heart is beating fast you are more likely to think of and, perhaps, experience a panic attack than when it is beating normally. Furthermore, you may not be aware of the cues or triggers of severe fear—that is, they are unconscious.

Social Contributions

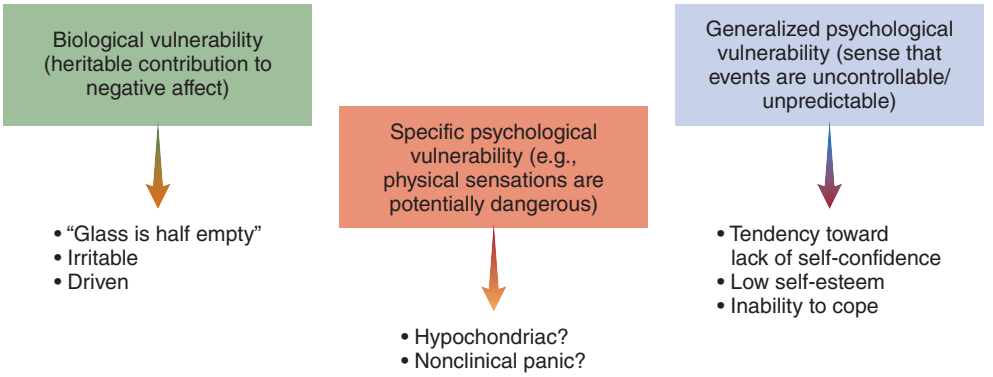
Stressful life events trigger our biological and psychological vulnerabilities to anxiety. Most are social and interpersonal in nature—marriage, divorce, difficulties at work, death of a loved one, pressures to excel in school, and so on. Some might be physical, such as an injury or illness.

The same stressors can trigger physical reactions such as headaches or hypertension and emotional reactions such as panic attacks (Barlow, 2002). The particular way we react to stress seems to run in families. If you get headaches when under stress, chances are other people in your family also get headaches. If you have panic attacks, other members of your family probably do also. This finding suggests a possible genetic contribution, at least to initial panic attacks.

behavioral inhibition system (BIS) Brain circuit in the limbic system that responds to threat signals by inhibiting activity and causing anxiety.
fight/flight system (FFS) Brain circuit in animals that when stimulated causes an immediate alarm-and-escape response resembling human panic.

An Integrated Model

Putting the factors together in an integrated way, we have described a theory of the development of anxiety and related disorders called the *triple vulnerability theory* (Barlow, 2000, 2002; Suárez et al., 2009). The first vulnerability (or diathesis) is a *generalized biological vulnerability.* We can see that a tendency to be uptight or high-strung might be inherited. But a generalized biological vulnerability to develop anxiety is not sufficient to produce anxiety itself. The second vulnerability is a *generalized psychological vulnerability*—that is, you might also grow up believing the world is dangerous and out of control and you might not be able to cope when things go wrong based on your early experiences. If this perception is strong, you have a generalized psychological vulnerability to anxiety. The third vulnerability is a *specific psychological vulnerability* in which you learn from early experience, such as being taught by your parents that some situations or objects are fraught with danger (even if they really aren't). Possible examples are dogs if one of your parents is afraid of dogs, or being evaluated negatively by others if this is something your parents worry about. These triple vulnerabilities are presented in ■ Figure 4.2. If you are feeling a lot of pressure, particularly from interpersonal stressors, a given stressor could activate your biological tendencies to be anxious and your psychological tendencies to feel you might not be able to deal with the situation and control the stress. Once this cycle starts, it tends to feed on itself, so it might not stop even when the particular life stressor has long since passed. Anxiety can be general, evoked by many aspects of your life, but it is usually focused on one area, such as social evaluations or grades (Barlow, 2002).



■ **Figure 4.2** The three vulnerabilities that contribute to the development of anxiety disorders. If individuals possess all three, the odds are greatly increased that they will develop an anxiety disorder after experiencing a stressful situation. (From Barlow, D. H., 2002. *Anxiety and its disorders: The nature and treatment of anxiety and panic* (2nd ed.). New York: Guilford Press.)

Comorbidity of Anxiety Disorders

Before describing the specific anxiety disorders, it is important to note that they often co-occur. As we described in Chapter 3, the co-occurrence of two or more disorders in a single individual is referred to as *comorbidity.* The high rates of comorbidity among anxiety disorders (and depression) emphasize how all of these disorders share the common features of anxiety and panic described here. They also share the same vulnerabilities, biological and psychological, to develop anxiety and panic. The various anxiety disorders differ only in what triggers the anxiety and, perhaps, the patterning of panic attacks. Of course, if each patient with an anxiety disorder also had every other anxiety disorder, there would be little sense in distinguishing among the specific disorders. But this is not the case, and, although rates of comorbidity are high, they vary somewhat from disorder to disorder (Allen et al., 2010; Bruce et al., 2005; Tsao, Mystkowski, Zucker, & Craske, 2002). A large-scale study completed at one of our centers examined the comorbidity of *Diagnostic and Statistical Manual of Mental Disorders,* fourth edition *(DSM-IV-TR)* anxiety and mood disorders (Brown & Barlow, 2002; Brown, Campbell, Lehman, Grisham, & Mancill, 2001). Data were collected from 1,127 patients. If we examine just rates of comorbidity at the time of assessment, the results indicate that 55% of the patients who received a principal diagnosis of an anxiety or depressive disorder had at least one additional anxiety or depressive disorder at the time of the assessment. If we consider whether the patient met criteria for an additional diagnosis at any time in his or her life, rather than just at the time of the assessment, the rate increases to 76%.

By far, the most common additional diagnosis for all anxiety disorders was major depression, which occurred in 50% of the cases over the course of the patient's life. This becomes important when we discuss the relationship of anxiety and depression later in this chapter. Also important is the finding that additional diagnoses of depression or alcohol or drug abuse makes it less likely that you will recover from an anxiety disorder and more likely that you will relapse if you do recover (Bruce et al., 2005; Huppert, 2009).

Concept Check 4.1

Complete the following statements about anxiety and its causes with the following terms: (a) comorbidity, (b) panic attack, (c) situationally bound, (d) neurotransmitter, (e) brain circuits, and (f) stressful.

1. A _____ is an abrupt experience of intense fear or acute discomfort accompanied by physical symptoms, such as chest pain and shortness of breath.

2. A _____ panic attack often occurs in certain situations but not anywhere else.
3. Anxiety is associated with specific _____ (for example, behavioral inhibition system or fight/flight system) and _____ systems (for example, noradrenergic).
4. The rates of _____ among anxiety disorders are high because they share the common features of anxiety and panic.
5. _____ life events can trigger biological and psychological vulnerabilities to anxiety.

Generalized Anxiety Disorder

› **What are the essential features, possible causes, and available treatment approaches for generalized anxiety disorder?**

Specific anxiety disorders are complicated by panic attacks or other features that are the focus of the anxiety. In generalized anxiety disorder, the focus is generalized to the events of everyday life. Therefore, we consider generalized anxiety disorder first.

Clinical Description

Most of us worry to some extent. As we have said, worry can be useful. It helps us plan for the future, make sure that we're prepared for that test, or double-check that we've thought of everything before we head home for the holidays. But what if you worry indiscriminately about everything? Furthermore, what if worrying is unproductive? No matter how much you worry, you can't seem to decide what to do about an upcoming problem or situation. And what if you can't stop worrying, even if you know it is doing you no good and probably making everyone else around you miserable? These features characterize **generalized anxiety disorder (GAD)**. Consider the case of Irene.

Irene ✦ Ruled by Worry

Irene, a 20-year-old college student, came to the clinic complaining of excessive anxiety and general difficulties in controlling her life. Everything was a catastrophe for Irene. Although she carried a 3.7 grade point average, she was convinced she would flunk every test she took.

Irene worried until she dropped out of the first college she attended after 1 month. She felt depressed for a while, then decided to take a couple of courses at a local junior college, believing she could handle the work there better. After achieving straight As at the junior college for 2 years, she enrolled once again in a 4-year college as a junior. After a short time, she began calling the clinic in a state of extreme agitation, saying she had to drop this or that course because she couldn't handle it. With great difficulty, her therapist and parents persuaded her to stay in the courses and to seek further help. In any course Irene completed, her grade was between an A and a B-minus, but she still worried about every test and every paper.

Irene was also concerned about relationships with her friends, and whenever she was with her new boyfriend she feared making a fool of herself and losing his interest. She reported that each date went extremely well but she knew the next one would probably be a disaster. As the relationship progressed and some sexual contact seemed natural, Irene was worried sick that her inexperience would make her boyfriend consider her naive and stupid. Nevertheless, she reported enjoying the early sexual contact and admitted that he seemed to enjoy it also, but she was convinced that the next time a catastrophe would happen.

Irene was also concerned about her health. She had minor hypertension, probably because she was somewhat overweight. She then approached every meal as if death itself might result if she ate the wrong types or amounts of food. She became reluctant to have her blood pressure checked for fear it would be high or to weigh herself for fear she was not losing weight. She severely restricted her eating and as a result had an occasional episode of binge eating.

Although Irene had an occasional panic attack, this was not a major issue to her. As soon as the panic subsided, she focused on the next possible catastrophe. In addition to high blood pressure, Irene had tension headaches and a "nervous stomach,"

generalized anxiety disorder (GAD) Anxiety disorder characterized by intense, uncontrollable, unfocused, chronic, and continuous worry that is distressing and unproductive, accompanied by physical symptoms of tenseness, irritability, and restlessness.

with a lot of gas, occasional diarrhea, and some abdominal pain. Irene's life was a series of impending catastrophes. Her mother reported that she dreaded a phone call from Irene, let alone a visit, because she knew she would have to see her daughter through a crisis. For the same reason, Irene had few friends.

Irene suffered from GAD. The *DSM-IV-TR* criteria specify that at least 6 months of excessive anxiety and worry (apprehensive expectation) must be ongoing more days than not. Furthermore, it must be difficult to turn off or control the worry process. Most of us worry for a time but can set the problem aside and go on to another task. Even if the upcoming challenge is a big one, as soon as it is over the worrying stops. For Irene, it never stopped. She turned to the next crisis as soon as the current one was over.

The physical symptoms associated with generalized anxiety and GAD differ somewhat from those associated with panic attacks and panic disorder (covered next). Whereas panic is associated with autonomic arousal, presumably as a result of a sympathetic nervous system surge (for instance, increased heart rate, palpitations, perspiration, and trembling), GAD is characterized by muscle tension, mental agitation (Brown, Marten, & Barlow, 1995), susceptibility to fatigue (probably the result of chronic excessive muscle tension), some irritability, and difficulty sleeping. Focusing attention is difficult as the mind quickly switches from crisis to crisis. People with GAD mostly worry about minor, everyday life events, a characteristic that distinguishes GAD from other anxiety disorders. When asked, "Do you worry excessively about minor things?" 100% of individuals with GAD respond "yes" compared to approximately 50% of individuals whose anxiety disorder falls within other categories (Barlow, 2002). Such a difference is statistically significant. Major events quickly become the focus of anxiety and worry, too. Adults typically focus on possible misfortune to their children, family health, job responsibilities, and more minor things such as household chores or being on time for appointments. Children with GAD most often worry about competence in academic, athletic, or social performance and about family issues (Albano & Hack, 2004; Furr, Tiwari, Suveg, & Kendall, 2009; Weems, Silverman, & La Greca, 2000). Older adults tend to focus, understandably, on health (Ayers, Thorp, & Wetherell, 2009; Beck & Averill, 2004; Person & Borkovec, 1995); they also have difficulty sleeping, which seems to make the anxiety worse (Beck & Stanley, 1997).

Statistics

Although worry and physical tension are common, the severe generalized anxiety experienced by Irene is rare. Approximately 3.1% of the population meets criteria for GAD during a given 1-year period (Kessler, Chiu, Demler, & Walters, 2005) and 5.7% at some point during their lifetime (Kessler, Berglund, Demler, Jin, & Walters, 2005). This is still a large number, making GAD one of the most common anxiety disorders. Similar rates are reported from around the world—for example, from rural South Africa (Bhagwanjee, Parekh, Paruk, Petersen, & Subedar, 1998). However, relatively few people with GAD come for treatment compared to patients with panic disorder. Anxiety clinics like ours report that only approximately 10% of their patients meet criteria for GAD compared to 30% to 50% for panic disorder. This may be because most patients with GAD seek help from their primary care doctors, where they are found in large numbers (Roy-Byrne & Katon, 2000).

About two-thirds of individuals with GAD are female in both clinical samples (Woodman, Noyes, Black, Schlosser, & Yagla, 1999; Yonkers, Warshaw, Massion, & Keller, 1996) and epidemiological studies (where individuals with GAD are identified from population surveys), which include people who do not necessarily seek treatment (Blazer, George, & Hughes, 1991; Carter, Wittchen, Pfister, & Kessler, 2001; Wittchen, Zhao, Kessler, & Eaton, 1994). But this sex ratio may be specific to developed countries. In the South African study mentioned here, GAD was more common in males.

Some people with GAD report onset in early adulthood, usually in response to a life stressor. Nevertheless, most studies find that GAD is associated with an earlier and more gradual onset than most other anxiety disorders (Anderson, Noyes, & Crowe, 1984; Barlow, 2002; Brown et al., 1994; Sanderson & Barlow, 1990; Woodman et al., 1999). The median age of onset based on interviews is 31 (Kessler, Berglund, et al., 2005), but like Irene, many people have felt anxious and tense all their lives. Once it develops, GAD, like most anxiety disorders, follows a chronic course, characterized by waxing and waning of symptoms.

GAD is prevalent among older adults. In the large national comorbidity study, GAD was found to be most common in the group over 45 years of age and least common in the youngest group, ages 15 to 24 (Wittchen et al., 1994).

DSM Disorder Criteria Summary
Generalized Anxiety Disorder

Features of generalized anxiety disorder include the following:

- Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least 6 months about a number of events or activities
- Difficulty in controlling the worry
- At least three of these symptoms: (1) restlessness or feeling keyed up or on edge; (2) being easily fatigued; (3) difficulty concentrating or mind going blank; (4) irritability: (5) muscle tension; (6) sleep disturbance
- Significant distress or impairment
- Anxiety is not limited to one specific issue

Source: Based on *DSM-IV-TR.* Reprinted with permission from Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision). © 2000 American Psychiatric Association.

Flint (1994) reported prevalence rates of GAD in older adults to be as high as 7%. We also know that the use of minor tranquilizers in the elderly is high, ranging from 17% to 50% in one study (Salzman, 1991). It is not entirely clear why drugs are prescribed with such frequency for the elderly. One possibility is that the drugs may not be entirely intended for anxiety. Prescribed drugs may be primarily for sleeping problems or other secondary effects of medical illnesses. In any case, benzodiazepines (minor tranquilizers) interfere with cognitive function and put the elderly at greater risks for falling down and breaking bones, particularly their hips (Barlow, 2002). In a classic study, Rodin and Langer (1977) demonstrated that older adults may be particularly susceptible to anxiety about failing health or other life situations that begin to diminish whatever control they retain over events in their lives. This increasing lack of control, failing health, and gradual loss of meaningful functions may be a particularly unfortunate by-product of the way the elderly are treated in Western culture. The result is substantial impairment in quality of life in older adults with GAD (Wetherell et al., 2004). If it were possible to change our attitudes and behavior, we might well reduce the frequency of anxiety, depression, and early death among our elderly citizens.

Causes

What causes GAD? We have learned a great deal in the past several years. As with most anxiety disorders, there seems to be a generalized biological vulnerability, as is reflected in studies examining a genetic contribution to GAD. This conclusion is based on studies showing that GAD tends to run in families (Noyes, Clarkson, Crowe, Yates, & McChesney, 1987; Noyes et al., 1992). Twin studies strengthen this suggestion (Kendler, Neale, Kessler, Heath, & Eaves, 1992). Kendler and colleagues (1995; Hettema, Prescott, Myers, Neale, & Kendler, 2005) confirmed that what seems to be inherited is the tendency to become anxious rather than GAD itself.

For years, clinicians thought that people who were generally anxious had simply not focused their anxiety on anything specific. Thus, such anxiety was described as "free floating." But now scientists have looked more closely and have discovered some interesting distinctions from other anxiety disorders.

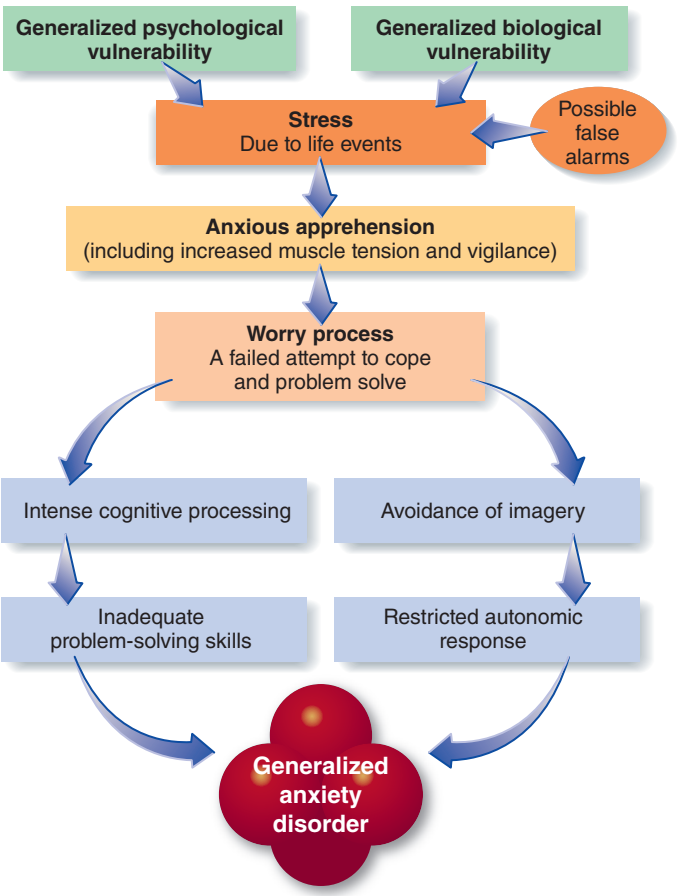
The first hints of difference were found in the physiological responsivity of individuals with GAD. It is interesting that individuals with GAD do not respond as strongly to stressors as individuals with anxiety disorders in which panic is more prominent. Several studies have found that individuals with GAD show *less responsiveness* on most physiological measures, such as heart rate, blood pressure, skin conductance, and respiration rate (Borkovec & Hu, 1990; Hoehn-Saric, McLeod, & Zimmerli, 1989; Roemer, Orsillo, & Barlow, 2002), than do individuals with other anxiety disorders. Therefore, people with GAD have been called *autonomic restrictors* (Barlow et al., 1996; Thayer, Friedman, & Borkovec, 1996).

When individuals with GAD are compared to nonanxious "normal" participants, the one physiological measure that consistently distinguishes the anxious group is muscle tension (Marten et al., 1993). People with GAD are chronically tense. For this reason, one proposal for *DSM-5* is to highlight muscle tension as the principal physical symptom in diagnosing GAD (Andrews et al., 2010). To understand this phenomenon of chronic muscle tension, we may have to know what's going on in the minds of people with GAD. With new methods from cognitive science, we are beginning to uncover the sometimes-unconscious mental processes ongoing in GAD (McNally, 1996).

There is evidence that individuals with GAD are highly sensitive to threat in general, particularly to a threat that has personal relevance—that is, they allocate their attention more readily to sources of threat than do people who are not anxious (Aikins & Craske, 2001; Barlow, 2002; Bradley, Mogg, White, Groom, & de Bono, 1999; MacLeod, Mathews, & Tata, 1986; Mathews, 1997). This high sensitivity may have arisen in early stressful experiences where they learned that the world is dangerous and out of control, and they might not be able to cope (generalized psychological vulnerability). Furthermore, this acute awareness of potential threat seems to be entirely automatic or unconscious. Using the Stroop color-naming task described in Chapter 2, MacLeod and Mathews (1991) presented threatening words on a screen for only 20 milliseconds and still found that individuals with GAD were slower to name the colors of the words than were nonanxious individuals. Remember that in this task words in colored letters are presented briefly and participants are asked to name the *color* rather than the word. The fact that the colors of threatening words were named more slowly suggests the *words* were more relevant to people with GAD, which interfered with their naming the color—even though the words were not present long enough for the individuals to be conscious of them.

How do mental processes link up with the tendency of individuals with GAD to be autonomic restrictors? Tom Borkovec and his colleagues noticed that although the peripheral autonomic arousal of individuals with GAD is restricted, they showed intense cognitive processing in the frontal lobes as indicated by EEG activity, particularly in the left hemisphere. This finding would suggest frantic, intense thought processes or worry without accompanying images (which would be reflected by activity in the right hemisphere of the brain rather than the left) (Borkovec, Alcaine, & Behar, 2004). Borkovec suggests that this kind of worry may be what causes these individuals to be autonomic restrictors (Borkovec, Shadick, & Hopkins, 1991; Roemer & Borkovec, 1993)—that is, they are thinking so hard about upcoming problems that they don't have the attentional capacity left for the all-important process of creating images of the potential threat, images that would elicit more substantial negative affect and autonomic activity. They *avoid* images associated with the threat (Borkovec et al., 2004; Craske, 1999; Fisher & Wells, 2009). But from the point of view of therapy, it is important to "process" the images and negative affect associated with anxiety (Craske & Barlow,

2006; Zinbarg, Craske, & Barlow, 2006). Because people with GAD do not seem to engage in this process, they may avoid much of the unpleasantness and pain associated with the negative affect and imagery, but they are never able to work through their problems and arrive at solutions. Therefore, they become chronic worriers, with accompanying autonomic inflexibility and severe muscle tension. In summary, some people inherit a tendency to be tense (generalized biological vulnerability), and they develop a sense early on that important events in their lives may be uncontrollable and potentially dangerous (generalized psychological vulnerability). Significant stress makes them apprehensive and vigilant. This sets off intense worry with resulting physiological changes, leading to GAD (Roemer et al., 2002; Turovsky & Barlow, 1996). This model is consistent with our view of anxiety as a future-oriented mood state focused on potential danger or threat, as opposed to an emergency or alarm reaction to actual present danger. A model of the development of GAD is presented in ■ Figure 4.3.



■ **Figure 4.3** An integrative model of GAD. (© Cengage Learning 2013)

Treatment

GAD is quite common, and available treatments, both drug and psychological, are reasonably effective. Benzodiazepines are most often prescribed for generalized anxiety, and the evidence indicates that they give some relief, at least in the short term. Few studies have looked at the effects of these drugs for a period longer than 8 weeks (Mathew & Hoffman, 2009). But the therapeutic effect is relatively modest. Furthermore, benzodiazepines carry some risks. First, they seem to impair both cognitive and motor functioning (see, for example, Hindmarch, 1986, 1990; O'Hanlon, Haak, Blaauw, & Riemersma, 1982; van Laar, Volkerts, & Verbaten, 2001). The drugs may impair driving, and in older adults they seem to be associated with falls (Ray, Gurwitz, Decker, & Kennedy, 1992; Wang, Bohn, Glynn, Mogun, & Avorn, 2001). More important, benzodiazepines seem to produce both psychological and physical dependence, making it difficult for people to stop taking them (Mathew & Hoffman, 2009; Noyes, Garvey, Cook, & Suelzer, 1991; Rickels, Schweizer, Case, & Greenblatt, 1990). There is reasonably wide agreement that the optimal use of benzodiazepines is for the short-term relief of anxiety associated with a temporary crisis or stressful event, such as a family problem (Craske & Barlow, 2006). Under these circumstances, a physician may prescribe a benzodiazepine until the crisis is resolved but for no more than a week or two. There is stronger evidence for the usefulness of antidepressants in the treatment of GAD; these drugs may prove to be a better choice (Brawman-Mintzer, 2001; Mathew & Hoffman, 2009).

In the short term, psychological treatments seem to confer about the same benefit as drugs in the treatment of GAD, but psychological treatments are probably more effective in the long term (Barlow, Allen, & Basden, 2007; Barlow & Lehman, 1996; Borkovec, Newman, Pincus, & Lytle, 2002; Roemer et al., 2002). Recent reports of innovations in brief psychological treatments are encouraging. Because we now know that individuals with GAD seem to avoid "feelings" of anxiety and the negative affect associated with threatening images, clinicians have designed treatments to help patients with GAD process the threatening information on an emotional level, using images, so that they will feel (rather than avoid feeling) anxious. In the early 1990s, we developed a cognitive-behavioral treatment (CBT) for GAD in which patients evoke the worry process during therapy sessions and confront anxiety-provoking images and thoughts head-on. The patient learns to use cognitive therapy and other coping techniques to counteract and control the worry process (Craske & Barlow, 2006; Wetherell, Gatz, & Craske, 2003). In a major study, a brief adaptation of this treatment was also used successfully to decrease anxiety and improve quality of life in a primary care office (family doctors and nurses) where GAD is a frequent complaint (Rollman, Belnap, & Mazumdar, 2005). Studies indicate that brief psychological treatments such as these alter the sometimes-unconscious cognitive biases associated with GAD (Mathews, Mogg, Kentish, & Eysenck, 1995; Mogg, Bradley, Millar, & White, 1995).

Despite this success, it is clear we need more powerful treatments, both drug and psychological, for this chronic, treatment-resistant condition. Recently, a new psychological treatment for GAD has been developed that incorpo-

rates procedures focusing on acceptance rather than avoidance of distressing thoughts and feelings in addition to cognitive therapy. Meditational approaches help teach the patient to be more tolerant of these feelings (Orsillo, Roemer, & Barlow, 2003; Roemer & Orsillo, 2002; Roemer et al., 2002). Preliminary results are encouraging (Roemer & Orsillo, 2007).

There is particularly encouraging evidence that psychological treatments are effective with children who suffer from generalized anxiety (Albano & Hack, 2004; Furr et al., 2009). Kendall and colleagues (1997) randomly assigned 94 children from 9 to 13 years of age to CBT or a wait-list control group. The majority of the children were diagnosed with GAD, but some had social phobia or separation anxiety. Based on teacher ratings, 70% of the treated children were functioning normally after treatment, gains maintained for at least 1 year. In a major clinical trial with children, CBT and the antidepressant drug sertraline (Zoloft) were equally effective immediately following treatment compared to taking placebo pills for children with GAD and other related disorders, but the combination of CBT and sertraline was even better, with 80% showing substantial improvement versus 24% on placebo (Walkup et al., 2008). Progress is also being made in adapting psychological treatments for older adults (Beck & Stanley, 1997; Stanley et al., 2003; Wetherell, Lenze, & Stanley, 2005). One large clinical trial demonstrated very clearly the efficiency of this treatment for adults over 60 compared to the usual care they received (Stanley et al., 2009).

After trying a number of different drugs, Irene was treated with the CBT approach developed at our clinic and found herself more able to cope with life. She completed college and graduate school, married, and is successful in her career as a counselor in a nursing home. But even now Irene finds it difficult to relax and stop worrying. She continues to experience mild to moderate anxiety, particularly when experiencing stress; she occasionally takes minor tranquilizers to support her psychological coping skills.

Concept Check 4.2

True (T) or false (F)?

1. ___ GAD is characterized by muscle tension, mental agitation, irritability, sleeping difficulties, and susceptibility to fatigue.
2. ___ Most studies show that in the majority of cases of GAD, onset is early in adulthood as an immediate response to a life stressor.
3. ___ GAD is prevalent in the elderly and in females in our society.
4. ___ GAD has no genetic basis.
5. ___ Cognitive-behavioral treatment and other psychological treatments for GAD are probably better than drug therapies in the long run.

Panic Disorder with and without Agoraphobia

› **What are the essential features of panic disorder?**

Did you have a relative, an eccentric aunt, for example, who never seemed to leave the house? Family reunions or visits always had to be at her house. She never went anywhere else. Most people attributed their old aunt's behavior to her being a little odd or perhaps just not fond of travel. She was warm and friendly when people came to visit, so she retained contact with the family.

Your aunt may not have been just odd or eccentric. She may have suffered from a debilitating anxiety disorder called **panic disorder with agoraphobia (PDA)**, in which individuals experience severe, unexpected panic attacks; they may think they're dying or otherwise losing control. Because they never know when an attack might occur, they develop **agoraphobia**, fear and avoidance of situations in which they would feel unsafe in the event of a panic attack or symptoms. These situations include those from which it would be hard or embarrassing to escape to get home or to a hospital. In severe cases, people with PDA are unable to leave the house, sometimes for years on end, as in the example of Mrs. M.

Mrs. M. • Self-Imprisoned

Mrs. M. was 67 years old and lived in a second-floor walk-up apartment in a lower-middle-class section of the city. Her adult daughter, one of her few remaining contacts with the world, had requested an evaluation with Mrs. M.'s consent. I rang the bell and entered a narrow hallway; Mrs. M. was nowhere in sight. Knowing that she lived on the second floor, I walked up the stairs and knocked on the door at the top. When I heard Mrs. M. ask me to come in, I opened the door.

panic disorder with agoraphobia (PDA) Fear and avoidance of situations the person believes might induce a dreaded panic attack.
agoraphobia Anxiety about being in places or situations from which escape might be difficult.

She was sitting in her living room, and I could quickly see the layout of the rest of the apartment. The living room was in the front; the kitchen was in the back, adjoining a porch. To the right of the stairs was the one bedroom, with a bathroom opening from it.

Mrs. M. was glad to see me and friendly, offering me coffee and homemade cookies. I was the first person she had seen in 3 weeks. Mrs. M. had not left that apartment in 20 years, and she had suffered from PDA for more than 30 years.

As she told her story, Mrs. M. conveyed vivid images of a wasted life. And yet she continued to struggle in the face of adversity and to make the best she could of her limited existence. Even areas in her apartment signaled the potential for terrifying panic attacks. She had not answered the door herself for the past 15 years because she was afraid to look into the hallway. She could enter her kitchen and go into the areas containing the stove and refrigerator, but for the past 10 years she had not been to the part of the room that overlooked the backyard or out onto the back porch. Thus, her life for the past decade had been confined to her bedroom, her living room, and the front half of her kitchen. She relied on her adult daughter to bring groceries and visit once a week. Her only other visitor was the parish priest, who came to deliver communion every 2 to 3 weeks when he could. Her only other contact with the outside world was through the television and the radio. Her husband, who had abused both alcohol and Mrs. M., had died 10 years earlier of alcohol-related causes. Early in her stressful marriage she had her first terrifying panic attack and had gradually withdrawn from the world. As long as she stayed in her apartment, she was relatively free of panic. Therefore, and because in her mind there were few reasons left near the end of her life to venture out, she declined treatment.

Clinical Description

At the beginning of the chapter, we talked about the related phenomena of anxiety and panic. In PDA, anxiety and panic are combined with *phobic avoidance* in an intricate relationship that can become as devastating as it was for Mrs. M. Many people who have panic attacks do not necessarily develop panic disorder. Similarly, many people experience anxiety and panic without developing agoraphobia. In those cases, the disorder is called **panic disorder without agoraphobia (PD)**.

To meet criteria for panic disorder (with or without agoraphobia), a person must experience an unexpected panic attack and develop substantial anxiety over the possibility of having another attack or about the implications of the attack or its consequences. In other words, the person must think that each attack is a sign of impending death or incapacitation. A few individuals do not report concern about another attack but still change their behavior in a way that indicates the distress the attacks cause them. They may avoid going to certain places or neglect their duties around the house for fear an attack might occur if they are too active.

The Development of Agoraphobia

Many people with panic disorder develop agoraphobia. The term *agoraphobia* was coined in 1871 by Karl Westphal, a German physician, and, in the original Greek, refers to fear of the marketplace. This is an appropriate term because the *agora,* the Greek marketplace, was a busy, bustling area. One of the most stressful places for individuals with agoraphobia today is the shopping mall, the modern-day agora.

Almost all agoraphobic avoidance behavior is simply a complication of severe, unexpected panic attacks (Barlow, 2002; Craske & Barlow, 1988, 2008). Simply put, if you have had unexpected panic attacks and are afraid you may have another one, you want to be in a safe place or at least with a safe person who knows what you are experiencing if another attack occurs so that you can quickly get to a hospital or at least go into your bedroom and lie down (the home is usually a safe place). We know that anxiety is diminished for individuals with agoraphobia if they think a location or person is "safe," even if there is nothing effective the person could do if something bad did happen. For these reasons, when they do venture outside their homes, people with agoraphobia always plan for rapid escape (for example, by sitting near the door). A list of typical situations commonly avoided by someone with agoraphobia is found in Table 4.1.

Although agoraphobic behavior almost always is closely tied to the occasions of panic initially, it can become relatively independent of panic attacks (Craske & Barlow, 1988; Craske, Rapee, & Barlow, 1988; White & Barlow, 2002). In other words, an individual who has not had a panic attack for years may still have strong agoraphobic avoidance, like Mrs. M. Agoraphobic avoidance seems to be determined for the most part by the extent to which you think or expect you might have another attack rather than by how many attacks you actually have or how severe they are. Thus, agoraphobic avoidance is simply one way of coping with unexpected panic attacks.

Other methods of coping with panic attacks include using (and eventually abusing) drugs and/or alcohol. Some individuals do not avoid agoraphobic situations but endure them with "intense dread." For example, people who must go to work each day or, perhaps, travel as part of the job will suffer untold agonies of anxiety and panic simply to achieve their goals. Thus, *DSM-IV-TR* notes that agoraphobia may be characterized either by avoiding the situations or by enduring them with marked distress. More recently, epidemiological surveys have identified a group of people who seem to have agoraphobia without ever having a panic attack or any fearful spells whatsoever (Wittchen, Gloster, Beesdo-Baum, Fava, & Craske, 2010). Further research is needed to confirm this condition.

Most patients with severe agoraphobic avoidance (and some with little) also display another cluster of avoidant behaviors that we call *interoceptive avoidance,* or avoid-

Table 4.1 Typical Situations Avoided by People with Agoraphobia

Shopping malls	Being far from home
Cars (as driver or passenger)	Staying at home alone
Buses	Waiting in line
Trains	Supermarkets
Subways	Stores
Wide streets	Crowds
Tunnels	Planes
Restaurants	Elevators
Theaters	Escalators

Source: Adapted, with permission, from Barlow, D. H., & Craske, M. G. (2007). *Mastery of your anxiety and panic* (4th ed., p. 5). New York: Oxford University Press.

ance of internal physical sensations (Barlow & Craske, 2007; Brown, White, & Barlow, 2005; Craske & Barlow, 2008; Shear et al., 1997). These behaviors involve removing yourself from situations or activities that might produce the physiological arousal that somehow resembles the beginnings of a panic attack. Some patients might avoid exercise because it produces increased cardiovascular activity or faster respiration that reminds them of panic attacks and makes them think one might be beginning. Other patients might avoid sauna baths or any rooms in which they might perspire. Psychopathologists are beginning to recognize that this cluster of avoidance behaviors is every bit as important as more classical agoraphobic avoidance.

Statistics

PD or PDA is fairly common. Approximately 2.7% of the population meet criteria for PD or PDA during a given 1-year period (Kessler, Chiu, et al., 2005; Kessler, Chiu, Jin, et al., 2006) and 4.7% met them at some point during their lives, two-thirds of them women (Eaton, Kessler, Wittchen, & Magee, 1994; Kessler, Berglund, et al., 2005). Another smaller group (1.4% at some point during their lives) develops agoraphobia without ever having a full-blown panic attack. Typically, these individuals will have only one or two severe symptoms, such as dizziness, rather than the minimum of four required to be called a panic attack. This condition is called *agoraphobia without a history of panic disorder,* but it looks much the same as PDA and is treated with the same treatments (Craske & Barlow, 2008; Kessler, Chiu, Jin, et al., 2006).

Onset of panic disorder usually occurs in early adult life—from midteens through about 40 years of age. The median age of onset is between 20 and 24 (Kessler, Berglund, et al., 2005). Most initial unexpected panic attacks begin at or after puberty. Furthermore, many prepubertal children who are seen by general medical practitioners have symptoms of hyperventilation that may well be panic attacks. However, these children do not report fear of dying or losing control—perhaps because they are not at a stage of their cognitive development where they can make these attributions (Nelles & Barlow, 1988).

Important work on anxiety in the elderly suggests that health and vitality are the primary focus of anxiety in the elderly population (Wisocki, 1988; Wolitzky-Taylor, Castriotta, Lenze, Stanley, & Craske, 2010). Lindesay (1991) studied 60 confirmed cases of phobic disorder in the elderly and found that they differed from younger adults in several ways, such as age of onset and prevalence. The primary phobia in this group was agoraphobia, which had a late onset (after age 50) and was often related to a stressful life event, usually an illness or injury. In general, the prevalence of PD or PDA decreases among the elderly, from 5.7% at ages 30–44 to 2.0% or less after age 60 (Kessler, Berglund, et al., 2005).

As we have said, most (75% or more) of those who suffer from agoraphobia are women (Barlow, 2002; Myers et al., 1984; Thorpe & Burns, 1983). The most logical explanation is cultural (Arrindell et al., 2003a; Wolitzky-Taylor et al., 2010). It is more accepted for women to report fear and to avoid numerous situations. Men, however, are expected to be stronger and braver—to "tough it out." The higher the severity of agoraphobic avoidance, the greater the proportion of women. For example, in our clinic, out of a group of patients suffering from panic disorder with mild agoraphobia, 72% were women; but if the agoraphobia was moderate, the percentage was 81%. Similarly, if agoraphobia was severe, the percentage was 89%.

What happens to men who have severe unexpected panic attacks? Is cultural disapproval of fear in men so strong that most of them simply endure panic? The answer seems to be

DSM Disorder Criteria Summary
Panic Disorder with Agoraphobia

Features of panic disorder with agoraphobia include the following:

- Recurrent unexpected panic attacks
- One or more of the following during the month after a panic attack: (1) persistent concern about having additional attacks; (2) worry about the implications of the attack; (3) a significant change in behavior related to the attacks.
- Anxiety about being in places or situations from which escape might be difficult or embarrassing, such as being in a crowd, traveling on a bus, or waiting in line.

Source: Based on *DSM-IV-TR.* Reprinted with permission from Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision). © 2000 American Psychiatric Association.

panic disorder without agoraphobia (PD) Panic attacks experienced without development of agoraphobia.

"no." A large proportion of males with unexpected panic attacks cope in a culturally acceptable way: They consume large amounts of alcohol. The problem is that they become dependent on alcohol, and many begin the long downward spiral into serious addiction. Thus, males may end up with an even more severe problem than PDA.

Cultural Influences

Panic disorder exists worldwide, although its expression may vary from place to place. In Lesotho, Africa, the prevalence of panic disorder (and GAD) was found to be equal to or greater than in North America (Hollifield, Katon, Spain, & Pule, 1990). In a more comprehensive study, prevalence rates for panic disorder were remarkably similar in the United States, Canada, Puerto Rico, New Zealand, Italy, Korea, and Taiwan, with only Taiwan showing somewhat lower rates (Horwath & Weissman, 1997). Rates are also similar among different ethnic groups in the United States, including African Americans. Furthermore, black and white patients with panic disorder show no significant differences in symptoms (Friedman, Paradis, & Hatch, 1994). However, note that panic disorder often co-occurs with hypertension in African American patients (Neal, Nagle-Rich, & Smucker, 1994; Neal-Barnett & Smith, 1997).

Somatic symptoms of anxiety may be emphasized in Third World cultures. Subjective feelings of dread or angst may not be a part of some cultures—that is, individuals in these cultures do not attend to these feelings and do not report them, focusing more on bodily sensations (Asmal & Stein, 2009; Lewis-Fernández et al., 2010). An anxiety-related, culturally defined syndrome prominent among Hispanic Americans, particularly those from the Caribbean, is called *ataques de nervios* (Hinton, Chong, Pollack, Barlow, & McNally, 2008; Hinton, Lewis-Fernández, & Pollack, 2009; Liebowitz et al., 1994). The symptoms of an *ataque* seem similar to those of a panic attack, although such manifestations as shouting uncontrollably or bursting into tears may be associated more often with *ataque* than with panic.

Finally, Devon Hinton, a psychiatrist/anthropologist, and his colleagues have recently described a fascinating manifestation of panic disorder among Khmer (Cambodian) and Vietnamese refugees in the United States. Both of these groups seem to suffer from a high rate of panic disorder. But a substantial number of these panic attacks are associated with orthostatic dizziness (dizziness if one stands up quickly) and "sore neck." What Hinton's group discovered is that the Khmer concept of *kyol goeu* or "wind overload" (too much wind or gas in the body, which may cause blood vessels to burst) becomes the focus of catastrophic thinking during panic attacks (Hinton & Good, 2009; Hinton, Pollack, Pich, Fama, & Barlow, 2005; Hinton, Hofmann, Pitman, Pollack, & Barlow, 2008).

Nocturnal Panic

Think back to the case of Gretchen, whose panic attack was described earlier. She was sound asleep when it happened. Approximately 60% of the people with panic disorder have experienced such nocturnal attacks (Craske & Rowe, 1997; Uhde, 1994). In fact, panic attacks occur more often between 1:30 A.M. and 3:30 A.M. than any other time (Taylor et al., 1986). In some cases, people are afraid to go to sleep at night. What's happening to them? Are they having nightmares? Research indicates they are not. Nocturnal attacks are studied in a sleep laboratory. Patients spend a few nights sleeping while attached to an electroencephalograph machine that monitors their brain waves (see Chapter 3). We all go through various stages of sleep that are reflected by different patterns on the electroencephalogram. We have learned that nocturnal panics occur during delta wave or slow wave sleep, which typically occurs several hours after we fall asleep and is the deepest stage of sleep. People with panic disorder often begin to panic when they start sinking into delta sleep, then they awaken amid an attack. Because there is no obvious reason for them to be anxious or panicky when they are sound asleep, most of these individuals think they are dying (Craske & Barlow, 1988; Craske & Rowe, 1997).

What causes nocturnal panic? Currently, our best information is that the change in stages of sleep to slow wave sleep produces physical sensations of "letting go" that are frightening to an individual with panic disorder (Craske, Lang, Mystkowski, Zucker, & Bystritsky, 2002). This process is described more fully later when we discuss causes of panic disorder. Several other events also occur during sleep that resemble nocturnal panic and are mistakenly thought by some to be the cause of nocturnal panic. Initially, these events were thought to be nightmares, but nightmares and other dreamlike activity occur only during a stage of sleep characterized by rapid eye movement (REM) sleep, which typically occurs much later in the sleep cycle. Therefore, people are not dreaming when they have nocturnal panics, a conclusion consistent with patient reports. A related phenomenon occurring in children is called *sleep terrors,* which we describe in more detail in Chapter 8 (Durand, 2006). Often children scream and get out of bed as if something were after them. However, they do not wake up and have no memory of the event in the morning. In contrast, individuals experiencing nocturnal panic attacks do wake up and later remember the event clearly. Sleep terrors also tend to occur at a later stage of sleep (stage 4 sleep), a stage associated with sleepwalking.

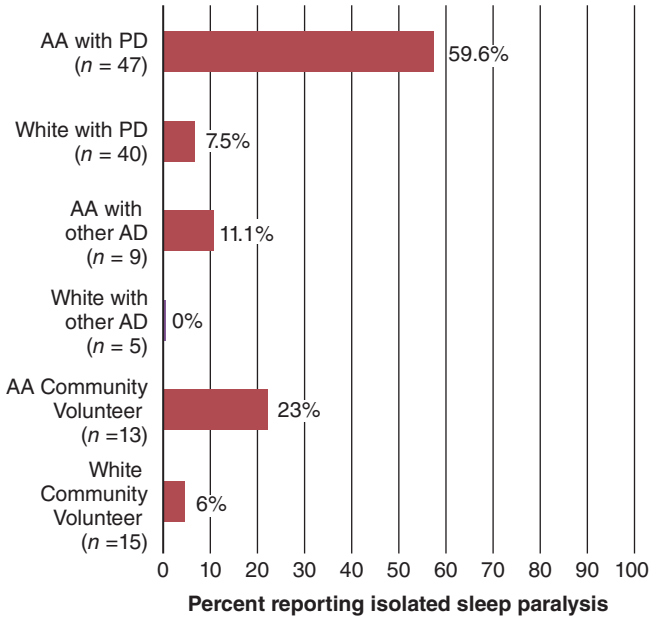
Finally, there is a fascinating condition called *isolated sleep paralysis* that seems culturally determined. Isolated sleep paralysis occurs during the transitional state between sleep and waking, when a person is either falling asleep or waking up but mostly when waking up. During this period, the individual is unable to move and experiences a surge of terror that resembles a panic attack; occasionally, there are also vivid hallucinations. One possible explanation is that REM sleep is spilling over into the waking cycle. This seems likely because one feature of REM sleep is lack of bodily movement. Another is vivid dreams, which could account for the experience of hallucination. Paradis, Friedman, and Hatch (1997) confirmed that the occurrence of isolated sleep paralysis was significantly higher in African Americans with panic disorder (59.6%) as compared with other groups (see ■ Fig-

ure 4.4). More recently, Ramsawh and colleagues (2008) replicated this finding and discovered that African Americans with isolated sleep paralysis had a history of trauma and more frequent diagnoses of panic disorder and posttraumatic stress disorder than African Americans without isolated sleep paralysis.

Causes

It is not possible to understand panic disorder (with or without agoraphobia) without referring to the triad of contributing factors mentioned throughout this book: biological, psychological, and social. Strong evidence indicates that agoraphobia, for the most part, develops after a person has unexpected panic attacks (or panic-like sensations); but whether agoraphobia develops and how severe it becomes seem to be socially and culturally determined, as we noted earlier. Panic attacks and panic disorder, however, seem to be related most strongly to biological and psychological factors and their interaction.

According to the triple vulnerability model (Bouton et al., 2001; Bouton, 2005; Suárez et al., 2009; White & Barlow, 2002), we all inherit—some more than others—a vulnerability to stress, which is a tendency to be generally neurobiologically overreactive to the events of daily life (generalized biological vulnerability). But some people are also more likely than others to have an emergency alarm reaction (unexpected panic attack) when confronted with stress-producing events. (Remember that other people might be more likely to have headaches or high blood pressure in response to the same kinds of stress.) Particular situations quickly become associated in an individual's mind with external and internal cues that were present during the panic attack (Bouton et al., 2001). The next time the person's heart rate increases during exercise, she might assume she is having a panic attack (conditioning). Harmless exercise is an example of an internal cue or a conditioned stimulus for a panic attack. Being in a movie theater when panic first occurred would be an external cue that might become a conditioned stimulus for future panics. Because these cues become associated with a number of different internal and external stimuli through a learning process, we call them *learned alarms*.



■ **Figure 4.4** Isolated sleep paralysis in African Americans (AA) and Caucasian Americans with panic disorder (PD), other anxiety disorder (AD) but not panic disorder, and community volunteers with no disorder. (Adapted from Paradis, C. M., Friedman, S., & Hatch, M., 1997. Isolated sleep paralysis in African-Americans with panic disorder. *Cultural Diversity & Mental Health, 3,* 69–76.)

But none of this would make much difference without the next step. An individual must be susceptible to developing anxiety over the possibility of having another panic attack (a generalized psychological vulnerability)—that is, he or she thinks the physical sensations associated with the panic attack mean something terrible is about to happen, perhaps death. This is what creates panic disorder. This tendency to believe that unexpected bodily sensations are dangerous reflects a specific psychological vulnerability to develop panic and related disorders. This causal sequence is depicted in ■ Figure 4.5.

Approximately 8% to 12% of the population has an occasional unexpected panic attack, often during a period of intense stress during the past year (Kessler et al., 2006; Mattis & Ollendick, 2002; Norton, Harrison, Hauch, & Rhodes, 1985; Suárez et al., 2009; Telch, Lucas, & Nelson, 1989). Most of these people do not develop anxiety (Telch et al., 1989). They seem to attribute the attack to events of the moment, such as an argument with a friend, something they ate, or a bad day, and go on with their lives.

The influential cognitive theories of David Clark (1986, 1996) explicate in more detail some cognitive processes that may be ongoing in the development of panic disorder. Clark emphasizes the specific psychological vulnerability of people with this disorder to interpret normal physical sensations in a catastrophic way. In other words, although we all typically experience rapid heartbeat after exercise, if you have a psychological or cognitive vulnerability, you might interpret the response as dangerous and feel a surge of anxiety. This anxiety, in turn, produces more physical sensations because of the action of the sympathetic nervous system, you perceive these additional sensations as even more dangerous, and a vicious cycle begins that results in a panic attack. Thus, Clark emphasizes the cognitive process as most important in panic disorder.

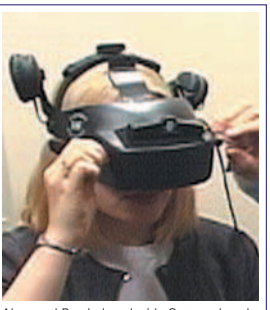
Treatment

As we noted in Chapter 1, research on the effectiveness of new treatments is important to psychopathology. Responses to certain specific treatments, whether drug or psychological, may indicate the causes of the disorder. We now discuss the benefits and some drawbacks of medication, psychological interventions, and a combination of these two treatments.

Virtual Reality Therapy: A New Technique on the Treatment of Anxiety Disorders

"I just feel really closed in, I feel like my heart is going to start beating really fast. . . . I won't be able to get enough air, I won't be able to breathe, and I'll pass out."

Go to Psychology CourseMate at www.cengagebrain.com to watch this video.



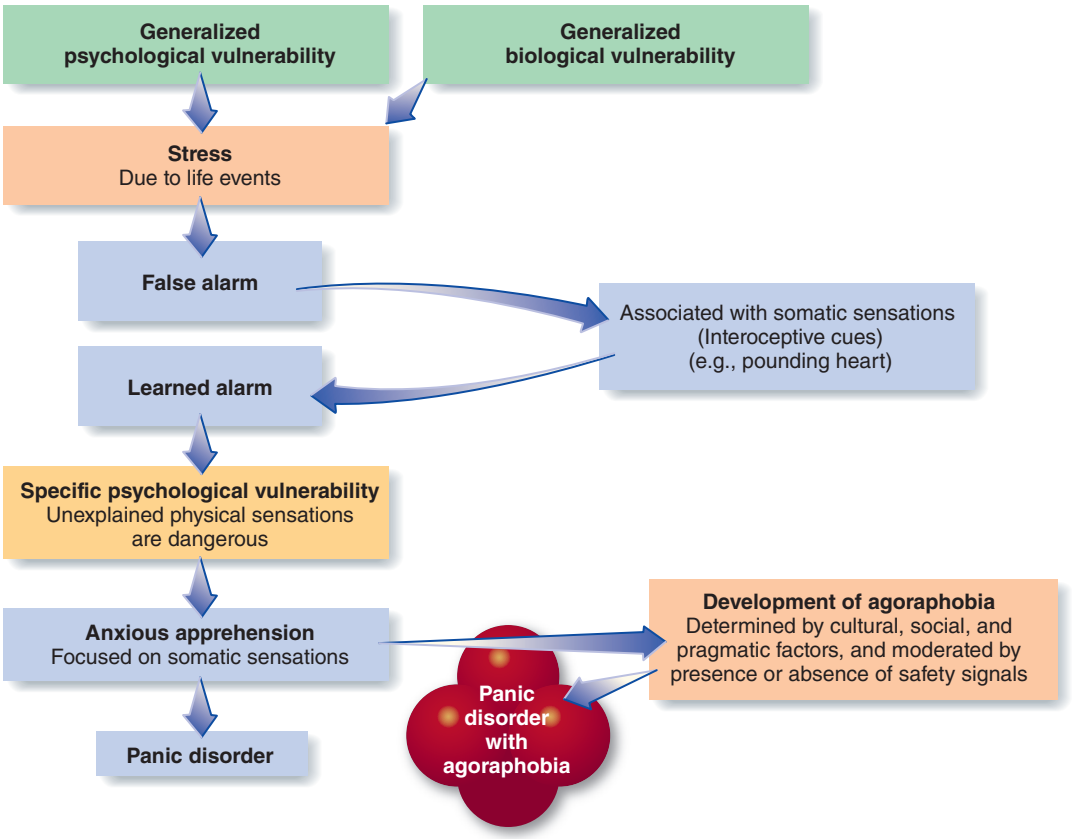
Abnormal Psychology Inside Out. produced by Ira Wohl, Only Child Motion Pictures

Medication

A large number of drugs affecting the noradrenergic, serotonergic, or GABA–benzodiazepine neurotransmitter systems or some combination seem effective in treating panic disorder, including high-potency benzodiazepines, the newer serotonin-specific reuptake inhibitors (SSRIs) such as Prozac and Paxil, and the closely related serotonin-norepinephrine reuptake inhibitors (SNRIs) such as venlafaxine (Barlow, 2002; Barlow & Craske, 2007; Pollack, 2005; Pollack & Simon, 2009; Spiegel, Wiegel, Baker, & Greene, 2000).

Each class of drugs has advantages and disadvantages too. SSRIs are currently the indicated drug for panic disorder based on all available evidence, although sexual dysfunction seems to occur in 75% or more of people taking these medications (Lecrubier, Bakker, et al., 1997; Lecrubier, Judge, et al., 1997). However, high-potency benzodiazepines such as alprazolam (Xanax), commonly used for panic disorder, work quickly but are hard to stop taking because of psychological and physical dependence and addiction. Therefore, they are not recommended as strongly as the SSRIs. Nevertheless, benzodiazepines remain the most widely used class of drugs in practice (Bruce et al., 2003). Also, all benzodiazepines adversely affect cognitive and motor functions to some degree. Therefore, people taking them in high doses often find their ability to drive a car or study somewhat reduced.

Approximately 60% of patients with panic disorder are free of panic as long as they stay on an effective drug



■ **Figure 4.5** A model of the causes of panic disorder with or without agoraphobia. (Reprinted, with permission, from White, K. S., & Barlow, D. H., 2002. A model of the causes of panic disorder with or without agoraphobia. In D. H. Barlow, *Anxiety and its disorders: The nature and treatment of anxiety and panic,* 2nd ed. New York: Guilford Press, © 2002 by Guilford Press.)

(Lecrubier, Bakker, et al., 1997; Pollack & Simon, 2009), but 20% or more stop taking the drug before treatment is done (Otto, Behar, Smits, & Hofmann, 2009), and relapse rates are high (approximately 50%) once the medication is stopped (Hollon et al., 2005; Spiegel et al., 2000). The relapse rate is closer to 90% for those who stop taking benzodiazepines (see, for example, Fyer et al., 1987).

Psychological Intervention

Psychological treatments have proved effective for panic disorder. Originally, such treatments concentrated on reducing agoraphobic avoidance, using strategies based on exposure to feared situations. The strategy of exposure-based treatments is to arrange conditions in which the patient can gradually face the feared situations and learn there is nothing to fear. Sometimes the therapist accompanies the patients on their exposure exercises. At other times, the therapist simply helps patients structure their own exercises and provides them with a variety of psychological coping mechanisms to help them complete the exercises, which are typically arranged from least to most difficult. A sample of these is listed in Table 4.2.

Gradual exposure exercises, sometimes combined with anxiety-reducing coping mechanisms such as relaxation or breathing retraining, have proved effective in helping patients overcome agoraphobic behavior. As many as 70% of patients undergoing these treatments substantially improve as their anxiety and panic are reduced and their agoraphobic avoidance is greatly diminished. Few, however, are cured because many still experience some anxiety and panic attacks, although at a less severe level.

Effective psychological treatments have recently been developed that treat panic attacks directly (Barlow & Craske, 2007; Clark et al., 1994; Craske & Barlow, 2008; Klosko, Barlow, Tassinari, & Cerny, 1990). **Panic control treatment (PCT)** developed at one of our clinics concentrates on exposing patients with panic disorder to the cluster of interoceptive (physical) sensations that remind them of their panic attacks. The therapist attempts to create "mini" panic attacks in the office by having the patients exercise to elevate their heart rates or perhaps by spinning them in a chair to make them dizzy. A variety of exercises have been developed for this purpose. Patients also receive cognitive therapy. Basic attitudes and perceptions concerning the dangerousness of the feared but objectively harmless situations are identified and modified. Follow-up studies of patients who receive PCT indicate that most of them remain better after at least 2 years (Craske & Barlow, 2008; Craske, Brown, & Barlow, 1991). Remaining agoraphobic behavior can then be treated with more standard exposure exercises. Although these treatments are quite effective, they are relatively new and not yet available to many individuals who suffer from panic disorder because administering them requires therapists to have advanced training (Barlow, Levitt, & Bufka, 1999; McHugh & Barlow, 2010).

Table 4.2 Situation-Exposure Tasks (From Least to Most Difficult)

Shopping in a crowded supermarket for 30 minutes alone
Walking five blocks away from home alone
Driving on a busy highway for 5 miles with spouse and alone
Eating in a restaurant, seated in the middle
Watching a movie while seated in the middle of the row

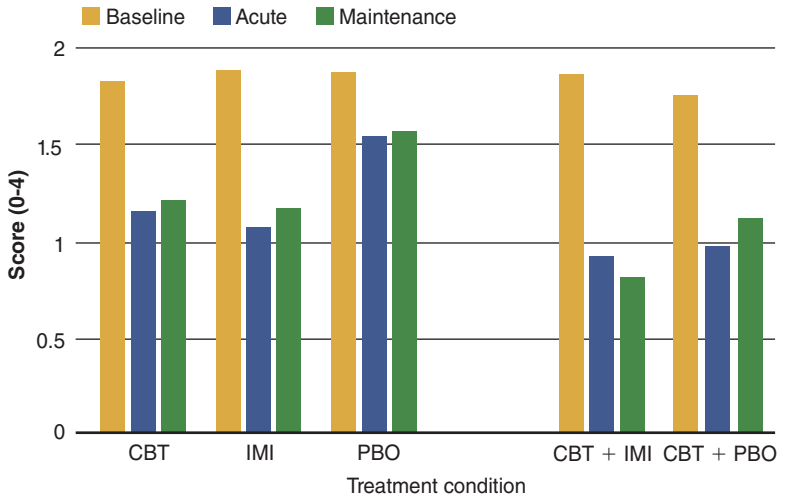
Source: Adapted, with permission, from Barlow, D. H., & Craske, M. G. (2007). *Mastery of your anxiety and panic* (4th ed., p. 133). New York: Oxford University Press.

Combined Psychological and Drug Treatments

Partly because primary care physicians are usually the first clinicians to treat those suffering from panic disorder and psychological treatments are not available in those settings, when patients do get referred for psychological treatment, they are often already taking medications. So, important questions are as follows: How do these treatments compare to each other? And do they work together? One major study sponsored by the National Institute of Mental Health looked at the separate and combined effects of psychological and drug treatments (Barlow, Gorman, Shear, & Woods, 2000). In this double-blind study, 312 carefully screened patients with panic disorder were treated at four sites, two known for their expertise with medication treatments and two known for their expertise with psychological treatments. The purpose of this arrangement was to control for any bias that might affect the results because of the allegiance of investigators committed to one type of treatment or the other. Patients were randomly assigned into five treatment conditions: psychological treatment alone (CBT); drug treatment alone (imipramine—IMI—a tricyclic antidepressant, was used because this study was begun before the SSRIs were available); a combined treatment condition (IMI + CBT); and two "control" conditions, one using placebo alone (PBO), and one using PBO + CBT (to determine the extent to which any advantage for combined treatment was caused by placebo contribution).

■ Figure 4.6 shows the results in terms of the percentage of patients who had responded to treatment by the end of 3 months of active treatment (termed the acute response), during which patients were seen weekly. Data were based on the judgment of an independent evaluator using the Panic Disorder Severity Scale and include patients who

panic control treatment (PCT) Cognitive–behavioral treatment for panic attacks, involving gradual exposure to feared somatic sensations and modification of perceptions and attitudes about them.



■ **Figure 4.6** Responders based on the panic disorder severity scale average item score after acute and after maintenance conditions. (Adapted from Barlow, D. H., Gorman, J. M., Shear, K. M., & Woods, S. W., 2000. Cognitive-behavioral therapy, imipramine, or their combination for panic disorder: A randomized controlled trial. *Journal of the American Medical Association, 283*(19), 2529–2536.)

dropped out along the way and were counted as failures. The data indicate that all treatment groups were significantly better than placebo, with some evidence that, among those who responded to treatment, people taking the drug alone did a little better than those receiving the CBT alone, but approximately the same number of patients responded to both treatments. Combined treatment was no better than individual treatments.

Figure 4.6 also presents the results after 6 additional months of maintenance treatment (9 months after treatment was initiated), during which patients were seen once per month. At this point, the results looked much as they did after initial treatment, except there was a slight advantage for combined treatment at this point and the number of people responding to placebo had diminished. Six months after treatment was discontinued, patients on medication, whether combined with CBT or not, had deteriorated somewhat, and those receiving CBT without the drug had retained most of their gains. Thus, psychological treatments seemed to perform better in the long run (six months after treatment had stopped).

Most studies show that drugs, particularly benzodiazepines, may interfere with the effects of psychological treatments (Craske & Barlow, 2008). Because of this, our multisite collaborative team asked whether a sequential strategy where one treatment was delayed until later and only given to those patients who didn't do as well as hoped would work better than giving both treatments at the same time. In this study, currently in preparation for publication, 256 patients with PD or PDA completed 3 months of initial treatment with CBT. Fifty-eight of those patients did not reach an optimal level of functioning (high end-state functioning) and entered a trial where they either received continued CBT or paroxetine. The paroxetine was administered for up to 1 year, whereas the CBT was delivered for 3 months. At the end of the 1-year period, there was a strong suggestion, represented as a statistical trend, that more of the patients receiving paroxetine responded compared to those receiving continued CBT. Looking at it the other way, another study (Craske et al., 2005) found that in the primary care setting, adding CBT to the treatment of patients already on medications resulted in significant further improvement compared to those patients on medication who did not have CBT added. This suggests that a "stepped-care" approach, where one treatment, such as a psychological treatment, is offered first, followed by a second treatment, such as a drug treatment, for those patients who do not respond adequately, may be superior to combining treatments from the beginning.

Investigation is beginning on a different drug that, unlike existing drugs, looks like it might enhance the effects of the best psychological interventions. This drug, an antibiotic, is called D-cycloserine (DCS). Neuroscientists such as Michael Davis at Emory University have made some interesting discoveries about this drug. Davis found that when rats who had learned a fear response, such as when a light was paired with a brief electric shock, were put into extinction trials in which the light that they had learned to fear was no longer paired with a frightening electric shock, the animals gradually learned not to fear the light (their fear extinguished), as expected. However, giving them DCS during these extinction trials made extinction work faster and last longer (Walker, Ressler, Lu, & Davis, 2002). Further research indicated that this drug works in the amygdala, a structure in the brain involved in the learning and unlearning of fear and anxiety. DCS affects neurotransmitter flow in a way that strengthens the extinction process (Hofmann, 2007).

Most recently, several investigators have used this drug with humans suffering from social anxiety disorder or panic disorder. DCS is given approximately an hour before the extinction or exposure trial, and the individual does not take the drug on an ongoing basis. For example, Michael Otto and his colleagues in one of our clinics (Otto et al., 2010) administered the most effective cognitive-behavioral intervention to patients with panic disorder either with or without the drug. (That is, one group got the drug and the other group got a placebo, and neither the patients nor the therapists knew which group was getting the drug and which was not, making it a double-blind experiment.) The people who got the drug improved significantly more during treatment than those who didn't get the drug. This is particularly noteworthy because the feared cues for people with panic disorder are physical sensations, and the drug DCS helped extinguish anxiety triggered by sensations such as increased heart rate or respiration. Stefan Hofmann and colleagues

(2006) found a similar result with social anxiety disorder. If these results are replicated, we may have an important advance in treating anxiety disorders.

Concept Check 4.3

True (T) or false (F)?

1. ___ PD is a disorder in which an individual experiences anxiety and panic with phobic avoidance of what that person considers an "unsafe" situation.
2. ___ About 40% of the population meet the criteria for panic disorder at some point in their lives.
3. ___ Some individuals with panic disorder are suicidal, have nocturnal panic, and/or are agoraphobic.
4. ___ Psychological treatments such as PCT or CBT are highly effective for treating panic disorder.

Specific Phobia

- **What are the principal causes of specific phobia?**
- **What strategies are typically used to treat specific phobia?**

Remember Judy in Chapter 1? When she saw a film of the frog being dissected, Judy began feeling queasy. Eventually she reached the point of fainting if someone simply said "Cut it out". Judy has what we call a specific phobia.

Clinical Description

A **specific phobia** is an irrational fear of a specific object or situation that markedly interferes with an individual's ability to function. Many of you might be afraid of something that is not dangerous, such as going to the dentist, or have a greatly exaggerated fear of something that is only slightly dangerous, such as driving a car or flying. Surveys indicate that specific fears of a variety of objects or situations occur in a majority of the population (Myers et al., 1984). But the very commonness of fears, even severe fears, often causes people to trivialize the psychological disorder known as a specific phobia. These phobias, in their severe form, can be extremely disabling, as we saw with Judy.

For many people, phobias are a nuisance—sometimes an extremely inconvenient nuisance—but people can adapt to life with a phobia by simply working around it somehow. In upstate New York and New England, some people are afraid to drive in the snow. We have had people come to our clinics who have been so severely phobic that during the winter they were ready to uproot, change their jobs and their lives, and move south. That is one way of dealing with a phobia. We discuss some other ways at the end of this chapter.

Judy's phobia meets the *DSM-IV* criterion of marked and persistent fear that is set off by a specific object or situation. She recognized that her fear and anxiety are excessive or unreasonable and went to considerable lengths to avoid situations in which her phobic response might occur.

DSM Disorder Criteria Summary
Specific Phobia

Features of specific phobia include the following:

- Persistent, excessive or unreasonable fear of a specific object or situation (e.g., heights, animals, seeing blood) with a duration of at least 6 months
- Immediate anxious or fearful response upon exposure to the phobic object or situation
- Recognition that the fear is excessive or unreasonable, or marked distress about having the phobia
- The phobic situation or object is avoided or is endured with intense anxiety or distress

Source: Based on *DSM-IV-TR.* Reprinted with permission from Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision). © 2000 American Psychiatric Association.

There are as many phobias as there are objects and situations. The variety of Greek and Latin names contrived to describe phobias stuns the imagination. Table 4.3 gives only the phobias beginning with the letter "a" (Maser, 1985).

Before the publication of *DSM-IV* in 1994, no meaningful classification of specific phobias existed. However, we have now learned that distinct types of specific phobia differ in major ways. Four major subtypes of specific phobia

specific phobia Unreasonable fear of a specific object or situation that markedly interferes with daily life functioning.

Table 4.3 Phobias Beginning with "A"

Term	Fear of:
Acarophobia	Insects, mites
Achluophobia	Darkness, night
Acousticophobia	Sounds
Acrophobia	Heights
Aerophobia	Air currents, drafts, wind
Agoraphobia	Open spaces
Agyiophobia	Crossing the street
Aichmophobia	Sharp, pointed objects; knives; being touched by a finger
Ailurophobia	Cats
Algophobia	Pain
Amathophobia	Dust
Amychophobia	Laceration; being clawed, scratched
Androphobia	Men (and sex with men)
Anemophobia	Air currents, wind, drafts
Anginophobia	Angina pectoris (brief attacks of chest pain)
Anthropophobia	Human society
Antlophobia	Floods
Apeirophobia	Infinity
Aphephobia	Physical contact, being touched
Apiphobia	Bees, bee stings
Astraphobia	Thunderstorms, lightning
Ataxiophobia	Disorder
Atephobia	Ruin
Auroraphobia	Northern lights
Autophobia	Being alone, solitude, oneself, being egotistical

Source: Reprinted, with permission, from Maser, J. D. (1985). List of phobias. In A. H. Tuma & J. D. Maser (Eds.), *Anxiety and the anxiety disorders* (p. 805). Mahwah, NJ: Erlbaum, © 1985 Lawrence Erlbaum Associates.

have been identified: blood–injury–injection type, situational type (such as planes, elevators, or enclosed places), natural environment type (for example, heights, storms, and water), and animal type. A fifth category, "other," includes phobias that do not fit any of the four major subtypes (for example, situations that may lead to choking, vomiting, or contracting an illness or, in children, avoidance of loud sounds or costumed characters). Although this subtyping strategy is useful, we also know that most people who suffer from phobia tend to have multiple phobias of several types (Hofmann, Lehman, & Barlow, 1997). This fact weakens the utility of subtyping, but subtyping remains useful enough to most likely be retained in *DSM-5* (LeBeau et al., 2010).

Blood–Injury–Injection Phobia

How do phobia subtypes differ from each other? We have already seen one major difference in the case of Judy. Rather than the usual surge of activity in the sympathetic nervous system and increased heart rate and blood pressure, Judy experienced a marked drop in heart rate and blood pressure and fainted as a consequence. Many people who suffer from phobias and experience panic attacks in their feared situations report that they feel like they are going to faint, but they never do because their heart rate and blood pressure are actually increasing. Therefore, those with **blood–injury–injection phobias** almost always differ in their physiological reaction from people with other types of phobia (Barlow & Liebowitz, 1995; Craske, Antony, & Barlow, 2006; Hofmann, Alpers, & Pauli, 2009; Öst, 1992). We also noted in Chapter 2 that blood–injury–injection phobia runs in families more strongly than any phobic disorder we know. This is probably because people with this phobia inherit a strong vasovagal response to blood, injury, or the possibility of an injection, all of which cause a drop in blood pressure and a tendency to faint. The phobia develops over the possibility of having this response. The average age of onset for this phobia is approximately 9 years (Antony, Brown, & Barlow, 1997a; LeBeau et al., 2010; Öst, 1989).

Situational Phobia

Phobias characterized by fear of public transportation or enclosed places are called **situational phobias**. Claustrophobia, a fear of small enclosed places, is situational, as is a phobia of flying. Psychopathologists first thought that situational phobia was similar to panic disorder with agoraphobia (PDA). Both situational phobia and PDA tend to emerge from midteens to mid-20s (Antony et al., 1997a; Craske et al., 2006; LeBeau et al., 2010) and have been shown to run in families (Curtis, Hill, & Lewis, 1990; Curtis, Himle, Lewis, & Lee, 1989; Fyer et al., 1990). But some analyses do not support the similarity as anything more than superficial (Antony et al., 1997a; Antony, Brown, & Barlow, 1997b). The main difference between situational phobia and PDA is that people with situational phobia never experience panic attacks outside the context of their phobic object or situation. Therefore, they can relax when they don't have to confront their phobic situation. People with panic disorder, in contrast, might experience unexpected, uncued panic attacks at any time.

Natural Environment Phobia

Sometimes very young people develop fears of situations or events occurring in nature. These fears are called **natural environment phobias**. The major examples are heights, storms, and water. These fears also seem to cluster together (Antony & Barlow, 2002; Hofmann et al., 1997): If

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▲ People who develop a natural environment phobia intensely fear such places as heights and events such as lightning.

you fear one situation or event, such as deep water, you are likely to fear another, such as storms. Many of these situations have some danger associated with them and, therefore, mild to moderate fear can be adaptive. It is entirely possible that we are somewhat prepared to be afraid of these situations; as we discussed in Chapter 2, something in our genes makes us sensitive to these situations if any sign of danger is present. In any case, these phobias have a peak age of onset of about 7 years. They are not phobias if they are only passing fears. They have to be persistent and to interfere substantially with the person's functioning, leading to avoidance of boat trips or summer vacations in the mountains where there might be a storm.

Animal Phobia

Fears of animals and insects are called **animal phobias**. Again, these fears are common and become phobic only if severe interference with functioning occurs. For example, we have seen cases in our clinic in which people with snake or mice phobias are unable to read magazines for fear of unexpectedly coming across a picture of one of these animals. These people are unable to go many places, even if they want to very much, such as to the country to visit someone. The fear experienced by people with animal phobias is different from an ordinary mild revulsion. The age of onset for these phobias, like that of natural environment phobias, peaks around 7 years (Antony et al., 1997a; LeBeau et al., 2010; Öst, 1987).

Separation Anxiety Disorder

All anxiety disorders described in this chapter may occur during childhood (Rapee, Schniering, & Hudson, 2009), and there is one additional anxiety disorder that is identified more closely with children. **Separation anxiety disorder** is characterized by children's unrealistic and persistent worry that something will happen to their parents or other important people in their life or that something will happen to the children themselves that will separate them from their parents (for example, they will be lost, kidnapped, killed, or hurt in an accident). Children often refuse to go to school or even to leave home, not because they are afraid of school but because they are afraid of separating from loved ones. These fears can result in refusing to sleep alone and may be characterized by nightmares involving possible separation and by physical symptoms, distress, and anxiety (Barlow, Pincus, Heinrichs, & Choate, 2003).

All young children experience separation anxiety to some extent; this fear usually decreases as they grow older. Therefore, a clinician must judge whether the separation anxiety is greater than would be expected at that particular age (Barlow et al., 2003; Ollendick & Huntzinger, 1990). It is also important to differentiate separation anxiety from school phobia. In school phobia, the fear is clearly focused on something specific to the school situation; the child can leave the parents or other attachment figures to go somewhere other than school. In separation anxiety, the act of separating from the parent or attachment figure provokes anxiety and fear.

There is now evidence that separation anxiety, if untreated, can extend into adulthood in approximately 35% of cases (Shear, Jin, Ruscio, Walters, & Kessler, 2006). Furthermore, very recent evidence suggests that this disorder occurs in approximately 6.6% of the adult population over the course of a lifetime (Shear et al., 2006). In some cases, the onset is in adulthood rather than carrying over from childhood. The focus of anxiety in adults is the same: that harm may befall loved ones during separation (Manicavasagar et al., 2010; Silove, Marnane, Wagner, Manicavasagar, & Rees, 2010).

Statistics

Specific fears occur in a majority of people. The ones most commonly found in the population at large, categorized by Agras, Sylvester, and Oliveau (1969), are presented in Table 4.4. Not surprisingly, fears of snakes and heights rank near the top. Few people who report specific fears qualify as hav-

blood–injury–injection phobia Unreasonable fear and avoidance of exposure to blood, injury, or the possibility of an injection. Victims experience fainting and a drop in blood pressure.
situational phobia Anxiety involving enclosed places (for example, claustrophobia) or public transportation (for example, fear of flying).
natural environment phobia Fear of situations or events in nature, especially heights, storms, and water.
animal phobia Unreasonable, enduring fear of animals or insects that usually develops early in life.
separation anxiety disorder Excessive, enduring fear in some children that harm will come to them or their parents while they are apart.

Image Source/JupiterImages

▲ A child with separation anxiety disorder persistently worries that parting with an important person drastically endangers either the loved one or the child.

ing a phobia, but for approximately 12.5% of the population, their fears become severe enough to earn the label "phobia." During a given 1-year period the prevalence is 8.7%. This is a high percentage, making specific phobia one of the most common psychological disorders in the United States and around the world (Arrindell et al., 2003b; Kessler, Berglund et al., 2005). In addition, 4.1% of children have separation anxiety at a severe enough level to meet criteria for a disorder (Shear et al., 2006). As with common fears, the sex ratio for specific phobias is, at 4:1, overwhelmingly female; this is also consistent around the world (Arrindell et al., 2003b; Craske et al., 2006; LeBeau et al., 2010).

Even though phobias may interfere with an individual's functioning, only the most severe cases come for treatment because more mildly affected people tend to work around their phobias; for example, someone with a fear of heights arranges her life so she never has to be in a tall building or other high place. People with situational phobias of such things as driving, flying, or being in small enclosed places most often come for treatment.

Once a phobia develops, it tends to last a lifetime (run a chronic course) (see, for example, Antony et al., 1997a; Barlow, 2002; Kessler, Berglund, et al., 2005); thus, the issue of treatment, described shortly, becomes important.

Although most anxiety disorders look much the same in adults and in children, clinicians must be aware of the types of normal fears and anxieties experienced throughout childhood so that they can distinguish them from specific phobias (Albano et al., 1996; Silverman & Rabian, 1993). Infants, for example, show marked fear of loud noises and strangers. At 1 to 2 years of age, children quite normally are anxious about separating from parents, and fears of animals and the dark also develop and may persist into the fourth or fifth year of life. Fear of various monsters and other imaginary creatures may begin about age 3 and last for several years. At age 10, children may fear evaluation by others and feel anxiety over their physical appearance. Generally, reports of fear decline with age, although performance-related fears of such activities as taking a test or talking in front of a large group may increase with age. Specific phobias seem to decline with old age (Ayers et al., 2009; Blazer et al., 1991; Sheikh, 1992).

The prevalence of specific phobias varies from one culture to another (Hinton & Good, 2009). Hispanics are two times more likely to report specific phobias than white Americans (Magee et al., 1996) for reasons not entirely clear. A variant of phobia in Chinese cultures is called *Pa-leng,* sometimes *frigo phobia* or "fear of the cold." *Pa-leng* can be understood only in the context of traditional ideas—in this case, the Chinese concepts of *yin* and *yang* (Tan, 1980). Chinese medicine holds that there must be a balance of yin and yang forces in the body for health to be maintained. Yin represents the cold, dark, windy, energy-sapping aspects of life; yang refers to the warm,

Table 4.4 Prevalence of Intense Fears and Phobias

Intense Fear	Prevalence per 1,000 Population	Sex Distribution	SE by Sex
Snakes	253	M: 118 F: 376	M: 34 F: 48
Heights	120	M: 109 F: 128	M: 33 F: 36
Flying	109	M: 70 F: 144	M: 26 F: 38
Enclosures	50	M: 32 F: 63	M: 18 F: 25
Illness	33	M: 31 F: 35	M: 18 F: 19
Death	33	M: 46 F: 21	M: 21 F: 15
Injury	23	M: 24 F: 22	M: 15 F: 15
Storms	31	M: 9 F: 48	M: 9 F: 22
Dentists	24	M: 22 F: 26	M: 15 F: 16
Journeys alone	16	M: 0 F: 31	M: 0 F: 18
Being alone	10	M: 5 F: 13	M: 7 F: 11

SE, standard error.

Source: Adapted, with permission, from Agras, W. S., Sylvester, D., & Oliveau, D. (1969). The epidemiology of common fears and phobias. *Comprehensive Psychiatry,* 10, 151–156, © 1969 Elsevier.

Joel Gordon 1993

▲ Chinese medicine is based on the concept that *yin* (dark, cold, enervating forces) and *yang* (bright, warm, energizing forces) must harmonize in the body. In this traditional representation of the yin–yang balance, note that each aspect contains something of the other.

bright, energy-producing aspects of life. Individuals with *Pa-leng* have a morbid fear of the cold. They ruminate over loss of body heat and may wear several layers of clothing even on a hot day. They may complain of belching and flatulence (passing gas), which indicate the presence of wind and therefore of too much yin in the body.

Causes

For a long time, we thought that most specific phobias began with an unusual traumatic event. For example, if you were bitten by a dog, you would develop a phobia of dogs. We now know this is not always the case (Barlow, 2002; Craske et al., 2006; Öst, 1985; Rachman, 2002). This is not to say that traumatic conditioning experiences do not result in subsequent phobic behavior. Almost every person with a choking phobia has had some kind of a choking experience. An individual with claustrophobia who recently came to our clinic reported being trapped in an elevator for an extraordinarily long period. These are examples of phobias acquired by *direct experience,* where real danger or pain results in an alarm response (a true alarm). This is one way of developing a phobia, and there are at least three others: *experiencing* a false alarm (panic attack) in a specific situation; *observing* someone else experience severe fear (vicarious experience); or, under the right conditions, *being told* about danger.

Studies show that many people with a phobia do not necessarily experience a true alarm resulting from real danger at the onset of their phobia. Many initially have an unexpected panic attack in a specific situation, perhaps related to current life stress. A phobia of that situation may then develop. Munjack (1984; Mineka & Zinbarg, 2006) studied people with specific phobias of driving. He noted that about 50% of the people who could remember when their phobia started had experienced a true alarm because of a traumatic experience such as a car accident. The others had had nothing terrible happen to them while they were driving, but they had experienced an unexpected panic attack during which they felt they were going to lose control of the car. Their driving was not impaired, and their catastrophic thoughts were simply part of the panic attack.

We also learn fears vicariously. Seeing someone else have a traumatic experience or endure intense fear may be enough to instill a phobia in the watcher. Remember, we noted earlier that emotions are contagious. If someone you are with is either happy or fearful, you will probably feel a tinge of happiness or fear also. Öst (1985) describes how a severe dental fear developed in this way. An adolescent boy sat in the waiting room at the school dentist's office partly observing, but fully hearing, his friend who was being treated. Evidently, the boy's reaction to pain caused him to move suddenly, and the drill punctured his cheek. The boy in the waiting room who overhead the accident bolted from the room and developed a severe and long-lasting fear of dental situations. Nothing actually happened to the second person, but you can certainly understand why he developed his phobia. Sometimes just being warned repeatedly about a potential danger is sufficient for someone to develop a phobia. Öst (1985) describes the case of a woman with an extremely severe snake phobia who had never encountered a snake. Rather, she had been told repeatedly while growing up about the dangers of snakes in the high grass. She was encouraged to wear high rubber boots to guard against this imminent threat—and she did so even when walking down the street. We call this mode of developing a phobia *information transmission.*

Terrifying experiences alone do not create phobias. As we have said, a true phobia also requires anxiety over the possibility of another extremely traumatic event or false alarm and we are likely to avoid situations in which that terrible thing might occur. If we don't develop anxiety, our reaction would presumably be in the category of normal fears experienced by more than half the population. Normal fear can cause mild distress, but it is usually ignored and forgotten. This point is best illustrated by Peter DiNardo and his colleagues (1988), who studied a group of dog phobics and a matched group who did not have the phobia. Like Munjack's (1984) driving phobics, about 50% of the dog phobics had had a frightening encounter with a dog, usually involving a bite. However, in the other group of individuals who did not have dog phobia, about 50% had also had a frightening encounter with a dog. Why hadn't they become phobics? They had not developed anxiety about another encounter with a dog, unlike the people who did become phobic (reflecting a generalized psychological vulnerability). A diagram of the etiology of specific phobia is presented in ■ Figure 4.7.

In summary, several things have to occur for a person to develop a phobia. First, a traumatic conditioning experience often plays a role (even hearing about a frightening event is sufficient for some individuals). Second, fear is more likely to develop if we are "prepared"—that is, we seem to carry an inherited tendency to fear situations that have always been dangerous to the human race, such as being threatened by wild animals or trapped in small places (see Chapter 2).

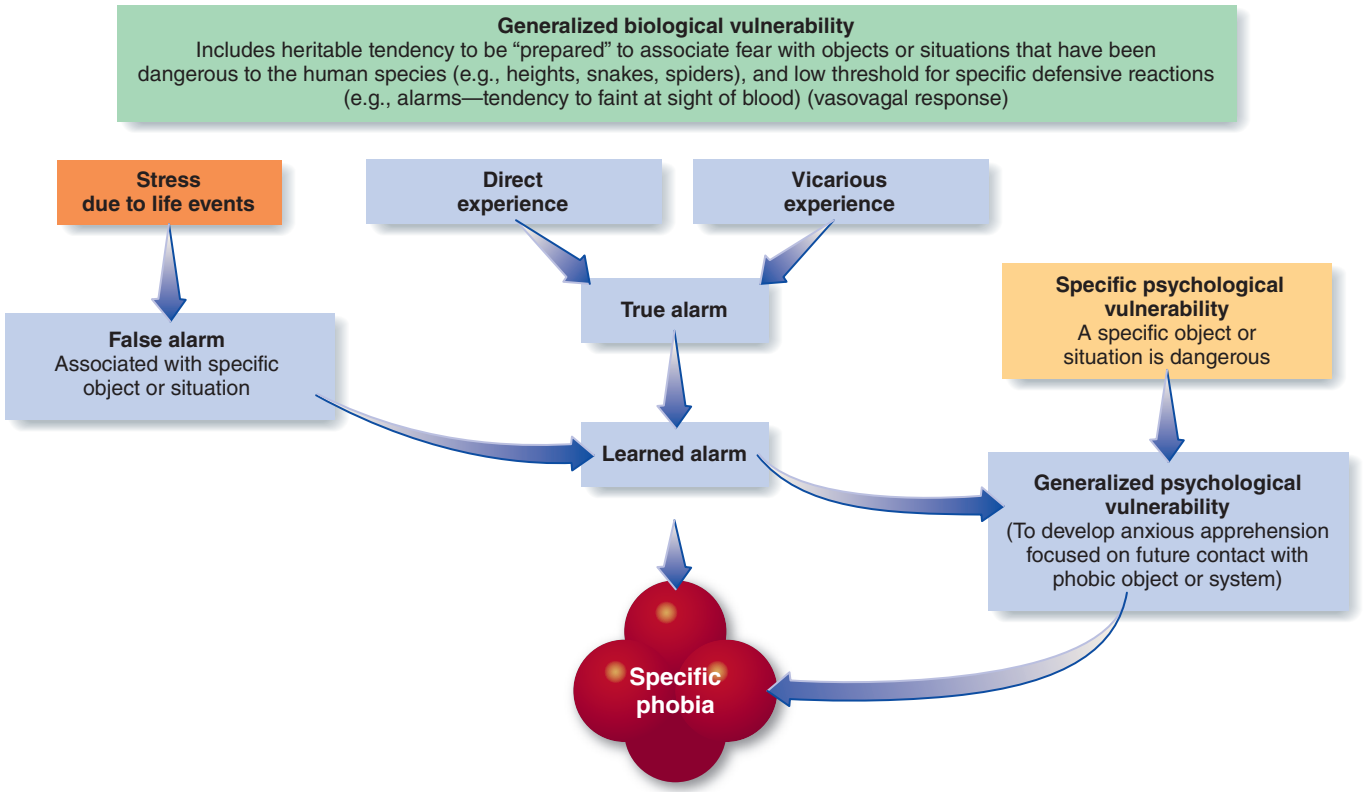
Third, we also have to be susceptible to developing anxiety about the possibility that the event will happen again. We have discussed the biological and psychological reasons for anxiety and have seen that at least one phobia, blood–injury–injection phobia, is highly heritable (Öst, 1989; Page & Martin, 1998). Patients with blood phobia probably also inherit a strong vasovagal response that makes them susceptible to fainting. This alone would not be sufficient to ensure their becoming phobic, but it combines with anxiety to produce strong vulnerability.

Several years ago, Fyer and colleagues (1990) demonstrated that approximately 31% of the first-degree relatives of people with specific phobias also had a phobia, compared with 11% of the first-degree relatives of "normal" controls. More recently, in a collaborative study between Fyer's clinic and our center, we replicated these results, finding a 28% prevalence in the first-degree relatives of patients with phobia compared to 10% in relatives of controls. More interestingly, it seems that each subtype of phobia "bred true," in that relatives were likely to have identical types of phobia. Kendler, Karkowski, and Prescott (1999a and b) and Page and Martin (1998) found relatively high estimates for heritability of individual specific phobias. We do not know for sure whether the tendency for phobias to run in families is caused by genes or by modeling, but the findings are at least suggestive of a unique genetic contribution to specific phobia (Antony & Barlow, 2002; Hettema et al., 2005; Smoller et al., 2005).

Finally, social and cultural factors are strong determinants of who develops and reports a specific phobia. In most societies, it is almost unacceptable for males to express fears and phobias. Thus, the overwhelming majority of reported specific phobias occur in women (Arrindell et al., 2003b; LeBeau et al., 2010). What happens to men? Possibly they work hard to overcome their fears by repeatedly exposing themselves to their feared situations. A more likely possibility is that they simply endure their fears without telling anyone about them and without seeking treatment (Antony & Barlow, 2002).

Treatment

Although the development of phobias is relatively complex, the treatment is fairly straightforward. Almost everyone agrees that specific phobias require structured and consis-



■ **Figure 4.7** A model of the various ways a specific phobia may develop. (Reprinted, with permission, from White, K. S., & Barlow D. H., A model of the various ways a specific phobia may develop. In Barlow, D. H., 2002. *Anxiety and its disorders: The nature and treatment of anxiety and panic.* (2nd ed.). New York: Guilford Press.)

tent exposure-based exercises (Barlow, Moscovitch, & Micco, 2004; Craske et al., 2006). Nevertheless, most patients who expose themselves gradually to what they fear must be under therapeutic supervision. Individuals who attempt to carry out the exercises alone often attempt to do too much too soon and end up escaping the situation, which may strengthen the phobia. In addition, if they fear having another unexpected panic attack in this situation, it is helpful to direct therapy at panic attacks in the manner described for panic disorder (Antony, Craske, & Barlow, 2006; Craske et al., 2006). For separation anxiety, parents are often included to help structure the exercises and also to address parental reaction to childhood anxiety (Choate, Pincus, Eyberg, & Barlow, 2005). More recently, an intensive 1-week program for girls ages 8 to 11 developed at one of our clinics in which the girls end up having a sleepover at the clinic has proven highly successful (Pincus, Santucci, Ehrenreich, & Ryberg, 2008; Santucci, Ehrenreich, Trosper, Bennett, & Pincus, 2009).

Finally, in cases of blood–injury–injection phobia, where fainting is a real possibility, graduated exposure-based exercises must be done in specific ways. Individuals must tense various muscle groups during exposure exercises to keep their blood pressure sufficiently high to complete the practice (Ayala, Meuret, & Ritz, 2009; Öst & Sterner, 1987). New developments make it possible to treat many specific phobias, including blood phobia, in a single, daylong session (see, for example, Antony & Barlow, 2002; Antony et al., 2006; Craske et al., 2006; Öst, Ferebee, & Furmark, 1997; Öst, Svensson, Hellström, & Lindwall, 2001). Basically, the therapist spends most of the day with the individual, working through exposure exercises with the phobia object or situation. The patient then practices approaching the phobic situation at home, checking in occasionally with the therapist. It is interesting that in these cases not only does the phobia disappear, but the tendency to experience the vasovagal response at the sight of blood also lessens considerably. It is also now clear based on brain-imaging work that these treatments change brain functioning by modifying neural circuitry—that is, these treatments "rewire" the brain (Paquette et al., 2003).

Social Phobia (Social Anxiety Disorder)

- **What are the principal causes of social phobia?**
- **What strategies are used to treat social phobia?**

Are you shy? If so, you have something in common with 20% to 50% of college students, depending on which survey you read. A much smaller number of people, who suffer severely around others, have **social phobia**, also called social anxiety disorder (SAD), which most likely will be the name adopted in the *DSM-5*. Consider the case of Billy, a 13-year-old boy.

Billy Too Shy

Billy was the model boy at home. He did his homework, stayed out of trouble, obeyed his parents, and was generally so quiet and reserved he didn't attract much attention. However, when he got to junior high school, something his parents had noticed earlier became painfully evident. Billy had no friends. He was unwilling to attend social or sporting activities connected with school, even though most of the other kids in his class went to these events. When his parents decided to check with the guidance counselor, they found that she had been about to call them. She reported that Billy did not socialize or speak up in class and was sick to his stomach all day if he knew he was going to be called on. His teachers had difficulty getting anything more than a yes-or-no answer from him. More troublesome was that he had been found hiding in a stall in the boy's restroom during lunch, which he said he had been doing for several months instead of eating. After Billy was referred to our clinic, we diagnosed a severe case of social phobia, an irrational and extreme fear of social situations. Billy's phobia took the form of extreme shyness. He was afraid of being embarrassed or humiliated in the presence of almost everyone except his parents.

Clinical Description

SAD is more than exaggerated shyness (Bögels et al., 2010; Hofmann et al., 2009; Schneier et al., 1996). The cases described here are typical of many that appear occasionally in the press over the years.

social phobia Extreme, enduring, irrational fear and avoidance of social or performance situations.

Rapid Behavioral Treatment of a Specific Phobia (Snakes)

"Since I remember, I remember being afraid of snakes. . . . I have dreams of snakes; it's horrible."

Go to Psychology CourseMate at www.cengagebrain.com to watch this video.

Abnormal Psychology Inside Out, produced by Ira Wohl, Only Child Motion Pictures

Steve and Chuck — Star Players?

In the second inning of an All-Star game, Los Angeles Dodger second baseman Steve Sax fielded an easy grounder, straightened up for the lob to first, and bounced the ball past first baseman Al Oliver, who was less than 40 feet away. It was a startling error even in an All-Star game studded with bush-league mishaps. But hard-core baseball fans knew it was one more manifestation of a leading mystery of the 1983 season: Sax, 23, the National League Rookie of the Year, could not seem to make routine throws to first base. (Of his first 27 errors that season, 22 were bad throws.)

Chuck Knoblauch won the Golden Glove Award in 1997 but led the league in errors in 1999 with 26, most of them throwing errors. Announcers and reporters observed that his throws would be hard and on target to first base if he made a difficult play and had to quickly turn and throw the ball "without thinking about it." But if he fielded a routine ground ball and had time to think about the accuracy of his throw, he would throw awkwardly and slowly—and often off target. The announcers and reporters concluded that, because his arm seemed fine on the difficult plays, his problem must be "mental." For the 2001 season, he was moved to left field to avoid having to make that throw and by 2003 was out of baseball.

Whereas Knoblauch continued to struggle, Sax overcame his problem. Many other athletes are not so fortunate. This problem is not limited to athletes but is also experienced by well-known lecturers and performers. Actress Scarlett Johansson avoided doing Broadway for many years because of intolerable performance anxiety. The inability of a skilled athlete to throw a baseball to first base or a seasoned performer to appear on stage certainly does not match the concept of "shyness" with which we are all familiar. What holds these seemingly different conditions together within the category of social anxiety disorder? Billy, Knoblauch, Sax, and Johansson all experienced marked and persistent anxiety focused on one or more social or performance situations. In Billy's case, these situations were any in which he might have to interact with people. For Knoblauch and Johansson, they were specific to performing some special behavior in public. Individuals with performance anxiety usually have no difficulty with social interaction, but when they must do something specific in front of people, anxiety takes over and they focus on the possibility that they will embarrass themselves.

The most common type of performance anxiety, to which most people can relate, is public speaking. Other situations that commonly provoke performance anxiety are eating in a restaurant or signing a paper in front of a clerk. Anxiety-provoking physical reactions include blushing; sweating; trembling; or, for males, urinating in a public restroom ("bashful bladder" or paruresis). Males with this problem must wait until a stall is available, a difficult task at times. What these examples have in common is that the individual is very anxious only while others are present and maybe watching and, to some extent, evaluating their behavior. This is truly social anxiety disorder because the people have no difficulty eating, writing, or urinating in private. Only when others are watching does the behavior deteriorate.

Individuals who are extremely and painfully shy in almost all social situations meet *DSM-IV-TR* criteria for the subtype *social phobia generalized type*. It is particularly prominent in children. In the child program in one of our clinics, 100% of children and adolescents with social phobia met criteria for the generalized type (Albano, DiBartolo, Heimberg, & Barlow, 1995), as did Billy in the example above (Schneier et al., 1996). Because most people with SAD meet criteria for this subtype to at least some degree, it may be dropped in *DSM-5* as being unnecessary (Bögels et al., 2010).

Statistics

As many as 12.1% of the general population suffer from social phobia at some point in their lives (Kessler, Berglund, et al., 2005). In a given 1-year period, the prevalence is 6.8% (Kessler, Chiu, et al., 2005). This makes social phobia second only to specific phobia as the most prevalent anxiety disorder, afflicting more than 35 million people in the United States alone, based on current population estimates. Many more people are shy, but not severely enough to meet criteria for social phobia. Unlike other anxiety disorders for which females predominate (Hofmann et al., 2009; Magee et al., 1996), the sex ratio for social phobia is nearly 50:50 (Hofmann & Barlow, 2002; Marks, 1985). Overall, 45.6% of people suffering from social phobia sought professional help in a recent 12-month period (Wang et al., 2005). Social phobia usually begins during adolescence, with a peak age of onset around 13 years (Kessler, Berglund, et al., 2005). Social phobia also tends to be more prevalent in people who are young (18–29 years), undereducated, single, and of low socioeconomic class. It is less than half as prevalent among individuals older than age 60 (6.6%) as it is among individuals 18–29 (13.6%) (Kessler, Berglund, et al., 2005).

DSM Disorder Criteria Summary
Social Phobia

Features of social phobia include the following:

- Marked and persistent fear of one or more social or performance situations in which the person is exposed to unfamiliar people or to possible scrutiny by others, with the fear that one will be embarrassed or humiliated
- Exposure to the feared social situation almost invariably provokes anxiety, sometimes as a panic attack
- Recognition that the fear is excessive or unreasonable
- The feared social or performance situation is avoided or are endured with intense anxiety or distress
- The avoidance, anxious anticipation, or distress in the feared social or performance situation(s) interferes significantly with the person's life and healthy functioning

Source: Based in *DSM-IV-TR*. Reprinted with permission from Diagnostic and Statistical Manual of Mental Disorders (4th ed., text revision). © 2000 American Psychiatric Association.

Considering their difficulty meeting people, it is not surprising that a greater percentage of individuals with social phobia are single than in the population at large. Social phobias distribute relatively equally among different ethnic groups (Magee et al., 1996). In Japan, the clinical presentation of anxiety disorders is best summarized under the label *shinkeishitsu*. One of the most common subcategories is referred to as *taijin kyofusho* (Hofmann et al., 2009; Kirmayer, 1991; Kleinknecht, Dinnel, Kleinknecht, Hiruma, & Harada, 1997). Japanese people with this form of social phobia strongly fear looking people in the eye and are afraid that some aspect of their personal presentation (blushing, stuttering, body odor, and so on) will appear reprehensible. Thus, the focus of anxiety in this disorder is on offending or embarrassing others rather than embarrassing oneself as in social phobia, although these two disorders overlap considerably (Dinnel, Kleinknecht, & Tanaka-Matsumi, 2002). Japanese males with this disorder outnumber females by a 3:2 ratio (Takahasi, 1989). More recently it has been established that this syndrome is found in many cultures around the world, including North America. The key feature once again is preoccupation with a belief that one is embarrassing oneself and offending others with a foul body odor. This set of symptoms is now called "olfactory reference syndrome" and has been proposed as a possible new disorder pending further study to be published in the appendix of *DSM-5* (Feusner, Phillips, & Stein, 2010).

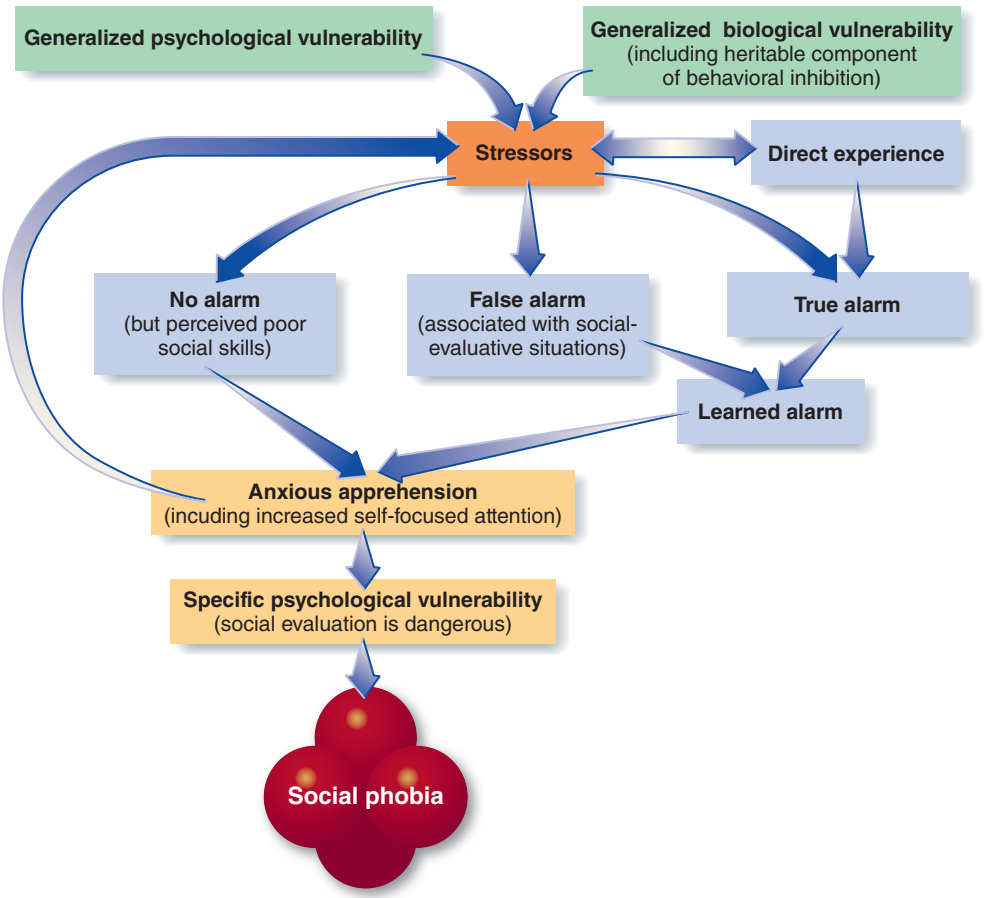
Causes

We have noted that we seem to be prepared by evolution to fear certain wild animals and dangerous situations in the natural environment. Similarly, it seems we are also prepared to fear angry, critical, or rejecting people (Blair et al., 2008; Mineka & Zinbarg, 1996, 2006; Mogg, Philippot, & Bradley, 2004; Öhman, 1986). In a series of studies, Öhman and colleagues (see, for example, Dimberg & Öhman, 1983; Öhman & Dimberg, 1978) noted that we learn more quickly to fear angry expressions than other facial expressions, and this fear diminishes more slowly than other types of learning. Lundh and Öst (1996) demonstrated that people with social phobia who saw a number of pictures of faces were likely to remember critical expressions; Mogg and colleagues (2004) showed that socially anxious individuals more quickly recognized angry faces than "normals," whereas "normals" remembered the accepting expressions (Navarrete et al, 2009). Fox and Damjanovic (2006) demonstrated that the eye region specifically is the threatening area of the face.

Why should we inherit a tendency to fear angry faces? Our ancestors probably avoided hostile, angry, domineering people who might attack or kill them. In all species, dominant, aggressive individuals, high in the social hierarchy, tend to be avoided. Jerome Kagan and his colleagues (see, for example, Kagan, 1994, 1997; Kagan, Reznick, & Snidman, 1988; Kagan & Snidman, 1991, 1999) have demonstrated that some infants are born with a temperamental profile or trait of inhibition or shyness that is evident as early as 4 months of age. Four-month-old infants with this trait become more agitated and cry more frequently when presented with toys or other age-appropriate stimuli than infants without the trait. There is now evidence that individuals with excessive behavioral inhibition are at increased risk for developing phobic behavior (Biederman et al., 1990; Essex, Klein, Slattery, Goldsmith, & Kalin, 2010; Hirschfeld et al., 1992).

A model of the etiology of social phobia would look somewhat like models of panic disorder and specific phobia. Three pathways to social phobia are possible, as depicted in ■ Figure 4.8.

First, someone could inherit a generalized biological vulnerability to develop anxiety, a biological tendency to be socially inhibited, or both. The existence of a generalized psychological vulnerability—such as the belief that events, particularly stressful events, are potentially uncontrollable—would increase an individual's vulnerability. When under stress, anxiety and self-focused attention could increase to the point of disrupting performance, even in the absence of a false alarm (panic attack). Second, when under stress, someone might have an unexpected panic attack in a social situation that would become associated (conditioned) to social cues. The individual would then become anxious about having additional panic attacks in the same or similar social situations. Third, someone might experience a real social trauma resulting in a true alarm. Anxiety would then develop (be conditioned) in the same or similar social situations. Traumatic social experiences may also extend back to difficult periods in childhood. Early adolescence—usually ages 12 through 15—is when children may be brutally taunted by peers who are attempting to assert their own dominance. This experience may produce anxiety and panic that are reproduced in future social situations. For example, McCabe, Anthony, Summerfeldt, Liss,



■ **Figure 4.8** A model of the various ways a social phobia may develop. (Reprinted, with permission, from White, K. S., & Barlow D. H., A model of the various ways a specific phobia may develop. In Barlow, D. H., 2002. *Anxiety and its disorders: The nature and treatment of anxiety and panic.* (2nd ed.). New York: Guilford Press.)

and Swinson (2003) noted that 92% of adults with social phobia in their sample experienced severe teasing and bullying in childhood, compared to only 35% to 50% among people with other anxiety disorders.

But one more factor must fall into place to make it a social anxiety disorder. The individual with the vulnerabilities and experiences just described must also have learned growing up that social evaluation in particular can be dangerous, creating a specific psychological vulnerability to develop social anxiety. Evidence indicates that some people with social phobia are predisposed to focus their anxiety on events involving social evaluation. Some investigators (Bruch & Heimberg, 1994; Rapee & Melville, 1997) suggest that the parents of patients with social phobia are significantly more socially fearful and concerned with the opinions of others than are the parents of patients with panic disorder and that they pass this concern on to their children (Lieb et al., 2000). Fyer, Mannuzza, Chapman, Liebowitz, and Klein (1993) reported that the relatives of people with social phobia had a significantly greater risk of developing it than the relatives of individuals without social phobia (16% versus 5%)—thus, the specific psychological vulnerability depicted in Figure 4.8. As you can see, a combination of biological and psychological events seems to lead to the development of social phobia.

Treatment

Effective treatments have been developed for social phobia only in the past several years (Barlow & Lehman, 1996; Hofmann, 2004; Taylor, 1996; Turk, Heimberg, & Magee, 2008). Rick Heimberg and colleagues developed a cognitive-behavioral group therapy (CBGT) program in which groups of patients rehearse or role-play their socially phobic situations in front of one another (Heimberg et al., 1990; Turk et al., 2008). The group members participate in the role-playing, for example, acting as audience for someone who has extreme difficulty giving a speech. At the same time, the therapist conducts rather intensive cognitive therapy aimed at uncovering and changing the automatic or unconscious perceptions of danger that the socially phobic client assumes to exist. These treatments have proved to be more effective than comparison treatments involving education about anxiety and social phobia and social support for stressful life events. More important, a follow-up after 5 years indicates that the therapeutic gains are maintained (Heimberg, Salzman, Holt, & Blendell, 1993). Clark and colleagues (2006) evaluated a new and improved cognitive therapy program that emphasized more real-life experiences during therapy to disprove automatic perceptions of danger. This program substantially benefited 84% of individuals receiving treatment, and these results were maintained at a 1-year follow-up.

We have adapted these protocols for use with adolescents, directly involving parents in the group treatment process. Preliminary results suggest that severely socially phobic adolescents can attain relatively normal functioning in school and other social settings (Albano & Barlow, 1996) and that including the parents in the treatment process produces better outcomes than treating the adolescents alone (Albano, Pincus, Tracey, & Barlow, in preparation).

Effective drug treatments also have been discovered (Van Ameringen, Mancini, Patterson, & Simpson, 2009). Tricyclic antidepressants and, particularly, monoamine oxidase (MAO) inhibitors have been found to be more effective than placebo in the treatment of severe social anxiety (Liebowitz et al., 1992). Since 1999 the SSRIs Paxil, Zoloft, and Effexor have received approval from the Food

and Drug Administration for treatment of social anxiety disorder based on studies showing effectiveness compared to placebo (see, for example, Stein et al., 1998).

Several major studies have compared psychological and drug treatments. One large and important study compared MAO inhibitors to the psychological treatments described earlier. In this study (Heimberg et al., 1998; Liebowitz et al., 1999) 133 patients were randomly assigned to phenelzine (the MAO inhibitor), CBGT, a placebo drug, or an educational-supportive group therapy that served as a placebo for the psychological treatment because it did not contain the cognitive-behavioral component. Results show that both active treatments are highly and equally effective compared to the two placebo conditions but that relapse tends to be more common after treatment stops among those taking medication. Another impressive study compared Clark's cognitive therapy described earlier to the SSRI drug Prozac, along with instructions to the patients with generalized social phobia to attempt to engage in more social situations (self-exposure). A third group received placebo plus instructions to attempt to engage in more social activities. Assessments were conducted before the 16-week treatment, at the midpoint of treatment, posttreatment, and then after 3 months of booster sessions. Finally, researchers followed up with patients in the two treatment groups 12 months later (Clark et al., 2003). Both treatments did well, but the psychological treatment was substantially better at all times. This study is also notable because of the *extent* of change in treatment (most patients were cured or nearly cured with few remaining symptoms).

The evidence is mixed on the usefulness of combining SSRIs or related drugs with psychological treatments. Davidson, Foa, and Huppert (2004) found that a cognitive-behavioral treatment and an SSRI were comparable in efficacy but that the combination was no better than the two individual treatments; Blanco and colleagues (2010), however, did find an additive effect. As noted earlier, an exciting study suggests that adding the drug D-cycloserine (DCS) to cognitive-behavioral treatments significantly enhances the effects of treatment (Hofmann et al., 2006). Unlike SSRIs, this drug is known to facilitate the extinction of anxiety, an important part of cognitive-behavioral treatments, by modifying neurotransmitter flow in the glutamate system as described in Chapter 2.

Concept Check 4.4

Identify the following specific phobias: (a) blood–injury–injection, (b) acrophobia, (c) animal, (d) social, (e) natural environment, and (f) other. The same phobia may apply to more than one statement.

1. Mark had no friends at school and hid in the boys' bathroom during both lunch and recess. _____
2. Dennis fears and strenuously avoids storms. Not surprisingly, on his first oceangoing cruise, he found that deep water terrified him, too. _____
3. Rita was comfortable at the zoo until the old terror gripped her at the insect display. _____
4. Armando would love to eat fish with his fishing buddies, but he experiences an inordinate fear of choking on a bone. _____
5. John had to give up his dream of becoming a surgeon because he faints at the sight of blood. _____
6. Rachel turned down several lucrative job offers that involved public speaking and took a low-paying desk job instead. _____
7. Farrah can't visit her rural friends because of her fear of snakes. _____

Posttraumatic Stress Disorder

› **What are the essential features and possible causes of posttraumatic stress disorder?**
› **What treatment approaches are available for posttraumatic stress order?**

In recent years, we have heard a great deal about the severe and long-lasting emotional disorders that can occur after a variety of traumatic events. For Americans, perhaps the most notorious traumatic events have been war, the tragedy of the World Trade Center and the Pentagon on September 11, 2001, or Hurricane Katrina in 2005. Still, emotional disorders also occur after physical assault (particularly rape), car accidents, natural catastrophes, or the sudden death of a loved one. One emotional disorder that follows a trauma is known as **posttraumatic stress disorder (PTSD)**.

posttraumatic stress disorder (PTSD) Enduring, distressing emotional disorder that follows exposure to a severe helplessness- or fear-inducing threat. The victim reexperiences the trauma, avoids stimuli associated with it, and develops a numbing of responsiveness and an increased vigilance and arousal.

Clinical Description

DSM-IV-TR describes the setting event for PTSD as exposure to a traumatic event during which someone feels fear, helplessness, or horror. Afterward, victims reexperience the event through memories and nightmares. When memories occur suddenly accompanied by strong emotion and the victims find themselves reliving the event, they are having a *flashback*. Victims most often avoid anything that reminds them of the trauma. They display a characteristic restriction or numbing of emotional responsiveness, which may be disruptive to interpersonal relationships. They are sometimes unable to remember certain aspects of the event. It is possible that victims unconsciously attempt to avoid the experience of emotion itself, like people with panic disorder, because intense emotions could bring back memories of the trauma. Finally, victims typically are chronically overaroused, easily startled, and quick to anger.

Consider the case of the Joneses from one of our clinics.

The Joneses: One Victim, Many Traumas

Mrs. Betty Jones and her four children arrived at a farm to visit a friend. (Mr. Jones was at work.) Jeff, the oldest child, was 8 years old. Marcie, Cathy, and Susan were 6, 4, and 2 years of age. Mrs. Jones parked the car in the driveway, and they all started across the yard to the front door. Suddenly Jeff heard growling somewhere near the house. Before he could warn the others, a large German shepherd charged and leapt at Marcie, the 6-year-old, knocking her to the ground and tearing viciously at her face. The family, too stunned to move, watched the attack helplessly. After what seemed like an eternity, Jeff lunged at the dog and it moved away. The owner of the dog, in a state of panic, ran to a nearby house to get help. Mrs. Jones immediately put pressure on Marcie's facial wounds in an attempt to stop the bleeding. The owner had neglected to retrieve the dog, and it stood a short distance away, growling and barking at the frightened family. Eventually, the dog was restrained and Marcie was rushed to the hospital. Marcie, who was hysterical, had to be restrained on a padded board so that emergency room physicians could stitch her wounds.

This case is unusual because not only did Marcie develop PTSD but so did her 8-year-old brother. In addition, Cathy, 4, and Susan, 2, although quite young, showed symptoms of the disorder, as did their mother (Table 4.5) (Albano, Miller, Zarate, Côté, & Barlow, 1997). Jeff evidenced classic survivor guilt symptoms, reporting that he should have saved Marcie or at least put himself between Marcie and the dog. Both Jeff and Marcie regressed developmentally, wetting the bed (nocturnal enuresis) and experiencing nightmares and separation fears. In addition, Marcie, having been strapped down and given a local anesthetic and stitches, became frightened of any medical procedures and even of such routine daily events as having her nails trimmed or taking a bath. Furthermore, she refused to be tucked into bed, something she had enjoyed all her life, probably because it reminded her of the hospital board. Jeff started sucking his fingers, which he had not done for years. These behaviors, along with intense separation anxiety, are common, particularly in younger children (Eth, 1990; Silverman & La Greca, 2002). Cathy, the 4-year-old, displayed considerable fear and avoidance when tested but denied having any problem when she was interviewed by a child psychologist. Susan, the 2-year-old, also had some symptoms, as shown in Table 4.5, but was too young to talk about them. However, for several months following the trauma she repeatedly said, without provocation, "Doggy bit sister."

Table 4.5 Symptoms of Posttraumatic Stress Disorder (PTSD) Evidenced by Marcie and Her Siblings

Symptoms	Jeff	Marcie	Cathy	Susan
Repetitive play—trauma themes		X	X	X
Nightmares	X	X	X	X
Reexperiencing	X			
Distress at exposure to similar stimuli	X	X	X	X
Avoidance of talk of trauma	X	X		
Avoidance of trauma recollections	X			
Regressive behavior	X	X		
Detachment	X	X		
Restricted affect	X	X		
Sleep disturbance	X	X	X	X
Anger outbursts	X	X		
Hypervigilance	X	X		
Startle response	X	X		
DSM-III-R PTSD diagnosis met	X	X		

Source: From Albano, A. M., Miller, P. P., Zarate, R., Côté, G., & Barlow, D. H. (1997). Behavioral assessment and treatment of PTSD in prepubertal children: Attention to developmental factors and innovative strategies in the case study of a family. *Cognitive and Behavioral Practice,* 4, 245–262.

As indicated in the criteria, PTSD is subdivided into acute and chronic. *Acute PTSD* can be diagnosed 1 month after the event occurs. When PTSD continues longer than 3 months, it is considered chronic. *Chronic PTSD* is usually associated with more prominent avoidance behaviors (Davidson, Hughes, Blazer, & George, 1991) and with the more frequent co-occurrence of additional diagnoses, such as social phobia. In *PTSD with delayed onset* individuals show few or no symptoms immediately after a trauma, but later, perhaps years afterward, they develop full-blown PTSD. Why onset is delayed in some individuals is not yet clear.

As we noted, PTSD cannot be diagnosed until a month after the trauma. New to *DSM-IV-TR* is a disorder called **acute stress disorder**. This is really PTSD occurring within the first month after the trauma, but the different name emphasizes the severe reaction that some people have immediately. PTSD-like symptoms are accompanied by severe dissociative symptoms, such as amnesia for all or part of the trauma, emotional numbing, and derealization, or feelings of unreality. According to one study, 63% to 70% of individuals with acute stress disorder from motor vehicle accidents went on to develop PTSD up to 2 years after the trauma. In addition, 13% who did not meet criteria for acute stress disorder went on to develop PTSD. If the victim experienced strong arousal and emotional numbing as part of acute stress disorder, the likelihood of later developing PTSD was greater (Harvey & Bryant, 1998).

DSM Disorder Criteria Summary
Posttraumatic Stress Disorder

Features of PTSD include the following:

- Exposure to a traumatic event in which the person experienced, witnessed, or was confronted by a situation involving death, threatened death, or serious injury, in response to which the person reacted with intense fear, helplessness, or horror
- The traumatic event is persistently reexperienced in one or more of the following ways: (1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions; (2) recurrent distressing dreams of the event; (3) a sense that the traumatic event is recurring, including illusions, hallucinations, and dissociative flashbacks; (4) intense psychological distress at exposure to internal or external cues that call to mind the event; (5) physiological reaction to cues that call to mind the event
- Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness
- Persistent symptoms of increased arousal, such as difficulty sleeping, irritability, and hypervigilance
- Clinically significant distress or impairment in social, occupational, or other important areas of functioning
- Duration of the disturbance for more than 1 month

Source: Based on *DSM-IV-TR.* Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision). © 2000 American Psychiatric Association.

Statistics

A number of studies have demonstrated the remarkably low prevalence of PTSD in populations of trauma victims. Rachman (1978), in a classic study, reported on the British citizenry who endured numerous life-threatening air raids during Word War II. He concluded that "a great majority of people endured the air raids extraordinarily well, contrary to the universal expectation of mass panic. Exposure to repeated bombings did not produce a significant increase in psychiatric disorders. Although short-lived fear reactions were common, surprisingly few persistent phobic reactions emerged" (Rachman, 1991, p. 162). Similar results have been observed from classic studies following disastrous fires, earthquakes, and floods (e.g., Green, Grace, Lindy, Titchener, & Lindy, 1983).

Phillip Saigh (1984) made some interesting observations when he was teaching at the American University in Beirut, Lebanon, just before and during the Israeli invasion in the early 1980s. Saigh had been collecting questionnaires measuring anxiety among university students just before the invasion. When the invasion began, half these students escaped to the surrounding mountains and were safe. The other half endured intense shelling and bombing for a period. Saigh continued administering the questionnaires and found a surprising result. There were no significant long-term differences between the group in the mountains and the group in the city, although a few students in the city who were closely exposed to danger and death did develop emotional reactions that progressed into PTSD.

In contrast, some studies have found a high incidence of PTSD after trauma. Kilpatrick and colleagues (1985) sampled more than 2,000 adult women who had personally experienced such trauma as rape, sexual molestation, robbery, and aggravated assault. Participants were asked whether they had thought about suicide after the trauma, had attempted suicide, or had a *nervous breakdown* (a lay term that has no meaning in psychopathology but is commonly used to refer to a severe psychological upset). The authors also analyzed the results based on whether the attack was completed or attempted. Rape had the most significant emotional impact. Compared to 2.2% of nonvictims, 19.2% of rape victims had attempted suicide and 44% reported suicidal ideation at some time following the rape. Similarly, Resnick, Kilpatrick, Dansky, Saunders, and Best (1993) found that 32% of rape victims met criteria for PTSD at some point in their lives. Taylor and Koch (1995) found that 15% to 20% of people experiencing severe auto accidents developed PTSD. Other surveys indicate that among the population as a whole, 6.8% have experienced PTSD at some point in their life (Kessler, Berglund, et al.,

acute stress disorder Severe reaction immediately following a terrifying event, often including amnesia about the event, emotional numbing, and derealization. Many victims later develop posttraumatic stress disorder.

2005) and 3.5% during the past year (Kessler, Chiu, et al., 2005), and combat and sexual assault are the most common precipitating traumas (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). The fact that a diagnosis of PTSD predicts suicidal attempts independently of any other problem, such as alcohol abuse, has recently been confirmed (Wilcox, Storr, & Breslau, 2009).

What accounts for the discrepancies between the low rate of PTSD in citizens who endured bombing and shelling in London and Beirut and the relatively high rate in victims of crime? Investigators have now concluded that during air raids many people may not have directly experienced the horrors of dying, death, and direct attack. Close exposure to the trauma seems to be necessary to developing this disorder (Friedman, 2009; Keane & Barlow, 2002; King, King, Foy, & Gudanowski, 1996). But this is also evident among Vietnam veterans, where 18.7% developed PTSD, with prevalence rates directly related to amount of combat exposure (Dohrenwend, Turner, & Turse, 2006). Surveys of 76 victims of Hurricane Katrina also report a doubling of severe mental illness (Kessler, Galea, Jones, & Parker, 2006). The connection between proximity to the traumatic event and the development of PTSD was starkly evident following the tragedy of 9/11. Galea and colleagues (2002) contacted a representative sample of adults living south of 110th Street in Manhattan and found that 7.5% reported symptoms consistent with a diagnosis of acute stress disorder or PTSD. But among respondents who lived close to the World Trade Center (south of Canal Street) the prevalence of the disorder was 20%. Again, those who experienced the disaster most personally and directly seemed to be the ones most affected.

But is this the whole story? It seems not. Some people experience the most horrifying traumas imaginable and emerge psychologically healthy. For others, even relatively mild stressful events are sufficient to produce a full-blown disorder. To understand how this can happen, we must consider the etiology of PTSD.

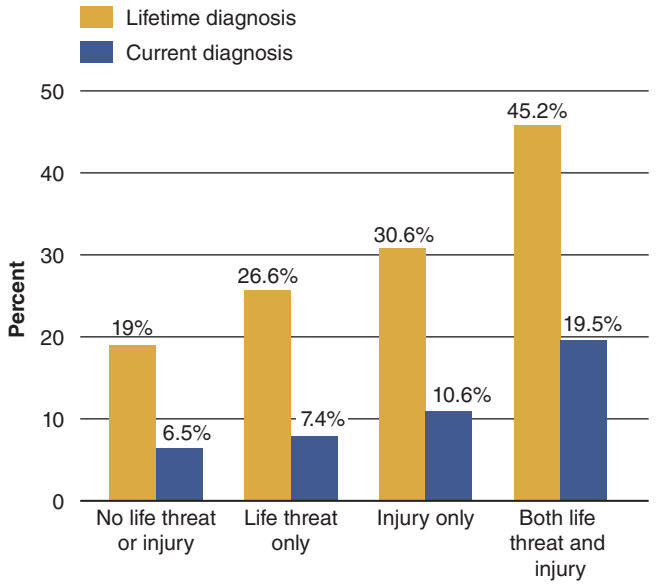
Causes

PTSD is the one disorder for which we know the cause at least in terms of the precipitating event. Someone personally experiences a trauma and develops a disorder. However, whether a person develops PTSD is a surprisingly complex issue involving biological, psychological, and social factors. David Foy and his colleagues (Foy, Sipprelle, Rueger, & Carroll, 1984) concluded that the intensity of combat exposure contributed to the etiology of PTSD in a group of Vietnam War veterans, a finding recently confirmed, as noted earlier (Dohrenwend et al., 2006; Friedman, 2009), but did not account for all of it. For example, approximately 67% of prisoners of war developed PTSD (Foy, Resnick, Sipprelle, & Carroll, 1987). This means that 33% of the prisoners who endured long-term deprivation and torture *did not* develop the disorder; perhaps the best known among the group is Senator John McCain. Similarly, Resnick and colleagues (1993) demonstrated that the percentage of female crime victims who developed PTSD increased as a function of the severity of the trauma (see ■ Figure 4.9). Finally, children experiencing severe burns are likely to develop PTSD in proportion to the severity of the burns and the pain associated with them (Saxe et al., 2005). At lower levels of trauma, some people develop PTSD, but most do not. What accounts for these differences?

As with other disorders, we bring our own generalized biological and psychological vulnerabilities with us. The greater the vulnerability, the more likely we are to develop PTSD. If certain characteristics run in your family, you have a much greater chance of developing the disorder (Davidson, Swartz, Storck, Krishnan, & Hammett, 1985; Foy et al., 1987). A family history of anxiety suggests a generalized biological vulnerability for PTSD. True and colleagues (1993) reported that, given the same amount of combat exposure and one twin with PTSD, a monozygotic (identical) twin was more likely to develop PTSD than a dizygotic (fraternal) twin. The correlation of symptoms in identical twins was between 0.28 and 0.41, whereas for fraternal twins it was between 0.11 and 0.24, which suggests some genetic influence in the development of PTSD. Nevertheless, as with other disorders, there is little or no evidence that genes directly cause PTSD (Norrholm & Ressler, 2009). Rather, genetic factors predispose individuals to be easily stressed and anxious, which then may make it more likely that a traumatic experience will result in PTSD.

Andy Nelson/The Christian Science Monitor via Getty Images

▲ Exposure to a traumatic event may create profound fear and helplessness. People who suffer from PTSD may reexperience such feelings in flashbacks, involuntarily reliving the horrifying event.



■ **Figure 4.9** Prevalence of lifetime and current PTSD associated with assault characteristics. (Reprinted, with permission, from Resnick, H. S., Kilpatrick, D. G., Dansky, B. S., Sanders, B. E., & Best, C. L., 1993. Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. *Journal of Consulting and Clinical Psychology, 81,* 984–991, © 1993 American Psychological Association.)

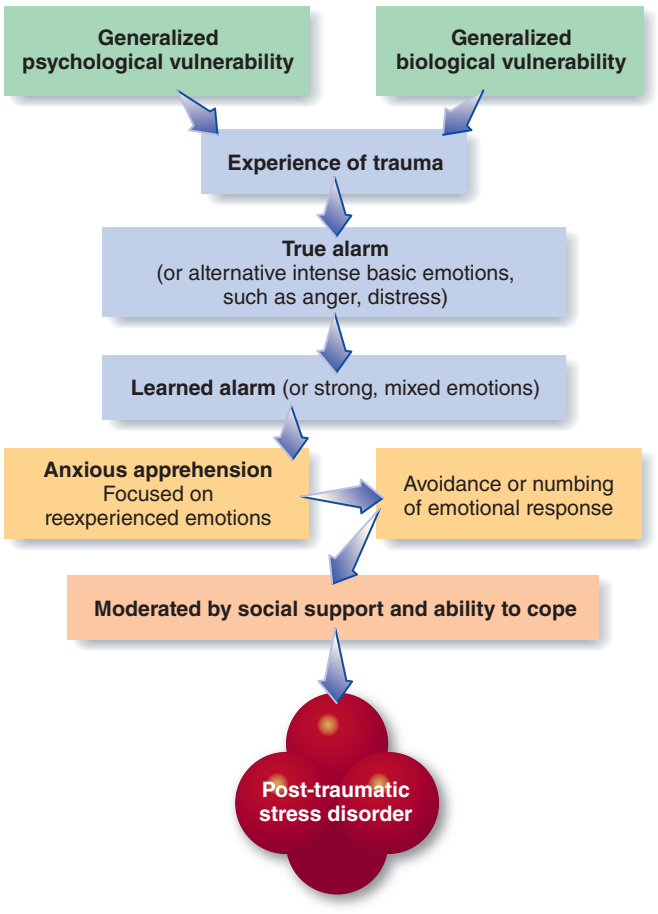
Breslau, Davis, and Andreski (1995) demonstrated among a random sample of 1,200 individuals that characteristics such as a tendency to be anxious, and factors such as minimal education, predict exposure to traumatic events in the first place and therefore an increased risk for PTSD. Breslau, Lucia, and Alvarado (2006) elaborated on this finding by showing that 6-year-old children with externalizing (acting out) problems were more likely to encounter trauma (such as assaults), probably because of their acting out, and later develop PTSD. Higher intelligence predicted decreased exposure to these types of traumatic events—that is, personality and other characteristics, some of them at least partially heritable, may predispose people to the experience of trauma by making it likely that they will be in (risky) situations where trauma is likely to occur (Norrholm & Ressler, 2009). Also, there seems to be a generalized psychological vulnerability described in the context of other disorders based on early experiences with unpredictable or uncontrollable events. Foy and colleagues (1987) discovered that at high levels of trauma, these vulnerabilities did not matter as much because the majority (67%) of prisoners of war they studied developed PTSD. However, at low levels of stress or trauma, vulnerabilities matter a great deal in determining whether the disorder will develop. Family instability is one factor that may instill a sense that the world is an uncontrollable, potentially dangerous place (Chorpita & Barlow, 1998; Suárez et al., 2009), so it is not surprising that individuals from unstable families are at increased risk for developing PTSD if they experience trauma. Family instability was found to be a prewar risk factor for the development of PTSD in a study of more than 1,600 male and female Vietnam veterans (King et al., 1996).

Finally, social factors play a major role in the development of PTSD (see, for example, Carroll, Rueger, Foy, & Donahoe, 1985). The results from a number of studies are consistent in showing that, if you have a strong and supportive group of people around you, it is much less likely you will develop PTSD after a trauma (Friedman, 2009). These factors seem to be true around the world because the reaction to trauma is similar across cultures, as a study comparing American and Russian adolescents demonstrated (Ruchkin et al., 2005). In a particularly interesting study, Vernberg, La Greca, Silverman, and Prinstein (1996) studied 568 elementary school children 3 months after Hurricane Andrew hit the coast of south Florida. More than 55% of these children reported moderate to severe levels of PTSD symptoms, a typical result for this type of disaster (La Greca & Prinstein, 2002). When the authors examined factors contributing to who developed PTSD symptoms and who didn't, social support from parents, close friends, classmates, and teachers was an important protective factor. Similarly, positive coping strategies involving active problem solving seemed to be protective, whereas becoming angry and placing blame on others were associated with higher levels of PTSD. The broader and deeper the network of social support, the less chance of developing PTSD.

Why is this? As you saw in Chapter 2, we are all social animals, and something about having a loving, caring group of people around us directly affects our biological and psychological responses to stress. A number of studies show that support from loved ones reduces cortisol secretion and hypothalamic–pituitary–adrenocortical (HPA) axis activity in children during stress (see, for example, Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). It seems clear that PTSD involves a number of neurobiological systems, particularly elevated corticotropin-releasing factor (CRF), which indicates heightened activity in the HPA axis, as described earlier in this chapter and in Chapter 2 (Amat et al., 2005; Charney, Deutch, Krystal, Southwick, & Davis, 1993; Gunnar & Fisher, 2006; Heim & Nemeroff, 1999; Ladd et al., 2000; Shin et al., 2004; Shin et al., 2009; Sullivan et al., 2000). You may remember that primates studied in the wild under extreme stress also have elevated levels of CRF and cortisol, the stress hormones. Chronic activation of stress hormones in these primates seems to result in permanent damage to the hippocampus, which regulates the stress hormones. Thus, chronic arousal and some other symptoms of PTSD may be directly related to changes in brain function and structure (Bremner, 1999; Bremner et al., 1997; McEwen & Magarinos, 2004). Evidence of damage to the hippocampus has appeared in groups of patients with war-related PTSD (Gurvits et al., 1996; Wang et al., 2010), adult survivors of childhood sexual abuse (Bremner et al., 1995), and firefighters exposed to extreme trauma (Shin et al., 2004). The hippocampus is a part of the brain that plays an important role in learning and memory. Thus, if there is damage to the hippocampus,

we might expect some disruptions in learning and memory. Disruptions in memory functions, including short-term memory and recalling events, have been demonstrated in patients with PTSD (Sass et al., 1992). Bremner, Vermetten, Southwick, Krystal, and Charney (1998) suggest that the fragmentation of memory often seen in patients with PTSD may account for difficulties in recalling at least some aspects of their trauma.

Earlier we described a panic attack as an adaptive fear response occurring at an inappropriate time. We have speculated that the "alarm reaction" that is a panic attack is similar in both panic disorder and PTSD but that in panic disorder the alarm is false. In PTSD, the initial alarm is true in that real danger is present (Jones & Barlow, 1990; Keane & Barlow, 2002). If the alarm is severe enough, we may develop a conditioned or learned alarm reaction to stimuli that remind us of the trauma (for example, being tucked into bed reminded Marcie of the emergency room board). We may also develop anxiety about the possibility of additional uncontrollable emotional experiences (such as flashbacks, which are common in PTSD). Whether or not we develop anxiety partly depends on our vulnerabilities. This model of the etiology of PTSD is presented in ■ Figure 4.10.



■ **Figure 4.10** A model of the causes of PTSD. (Reprinted, with permission, from White, K. S., & Barlow, D. H., A model of the causes of PTSD. In Barlow, D. H., 2002. *Anxiety and its disorders: The nature and treatment of anxiety and panic* (2nd ed.). New York: Guilford Press, © 2002 Guilford Press.)

Treatment

From the psychological point of view, most clinicians agree that victims of PTSD should face the original trauma, process the intense emotions, and develop effective coping procedures to overcome the debilitating effects of the disorder (Barlow & Lehman, 1996; Keane & Barlow, 2002; Najavits, 2007; Resick, Monson, & Rizvi, 2008). In psychoanalytic therapy, reliving emotional trauma to relieve emotional suffering is called *catharsis*. The trick is in arranging the reexposure so that it will be therapeutic rather than traumatic again. Unlike the object of a specific phobia, a traumatic event is difficult to recreate, and few therapists want to try. Therefore, *imaginal exposure,* in which the content of the trauma and the emotions associated with it are worked through systematically, has been used for decades under a variety of names. At present, the most common strategy to achieve this purpose with adolescents or adults is to work with the victim to develop a narrative of the traumatic experience that is then reviewed extensively in therapy. Cognitive therapy to correct negative assumptions about the trauma, such as blaming oneself in some way, feeling guilty, or both, is often part of treatment (Najavits, 2007; Resick et al., 2008).

Another complication is that trauma victims often repress the emotional side of their memories of the event and sometimes, it seems, the memory itself. This happens automatically and unconsciously. Occasionally, with treatment, the memories flood back and the patient dramatically relives the episode. Although this may be frightening to both patient and therapist, it can be therapeutic if handled appropriately. Evidence is now accumulating that early, structured interventions delivered as soon after the trauma as possible to those who require help are useful in preventing the development of PTSD (Bryant, Moulds, & Nixon, 2003; Ehlers et al., 2003; Litz, Gray, Bryant, & Adler, 2002). For example, in the study by Ehlers and colleagues (2003) of patients who had experienced a scary car accident and were clearly at risk for developing PTSD, only 11% developed PTSD after 12 sessions of cognitive therapy, compared with 61% of those receiving a detailed self-help booklet or 55% of those who were just assessed repeatedly over time but had no intervention. All patients who needed it were then treated with cognitive therapy. However, there is evidence that subjecting trauma victims to a single debriefing session, in which they are forced to express their feelings whether they are distressed or not, can be harmful (Ehlers & Clark, 2003).

Both Marcie, the young girl bitten by the dog, and her brother were treated simultaneously at our clinic. The primary difficulty was Marcie's reluctance to be seen by a doctor or to undergo any physical examinations, so a series of experiences was arranged from least to most intense (Table 4.6). Mildly anxiety-provoking procedures for Marcie included having her pulse taken, lying on an examination table, and taking a bath after accidentally cutting herself. The most intense challenge was being strapped on a restraining board. First Marcie watched her brother go

Table 4.6 Fear and Avoidance Hierarchy for Marcie

	Pretreatment Fear Rating	Posttreatment Fear Rating
Being strapped on a board	4	0
Having an electrocardiogram	4	0
Having a chest X ray	4	0
Having doctor listen to heart with stethoscope	3	0
Lying on examination table	3	0
Taking a bath after sustaining an accidentally inflicted cut	3	0
Allowing therapist to put bandage on a cut	2	0
Letting therapist listen to heart with stethoscope	1	0
Having pulse taken	1	0
Allowing therapist to examine throat with tongue depressor	1	0

Source: From Albano, A. M., Miller, P. P., Zarate, R., Côté, G., & Barlow, D. H. (1997). Behavioral assessment and treatment of PTSD in prepubertal children: Attention to developmental factors and innovative strategies in the case study of a family. *Cognitive and Behavioral Practice,* 4, 254, © 1997 Association for Advancement of Behavior Therapy.

through these exercises. He was not afraid of these particular procedures, although he was anxious about being strapped to a board because of Marcie's terror at the thought. After she watched her brother experience these situations with little or no fear, Marcie tried each one in turn. The therapist took instant photographs of her that she kept after completing the procedures. Marcie was also asked to draw pictures of the situations. The therapist and her family warmly congratulated her as she completed each exercise. Because of Marcie's age, she was not adept at imaginatively recreating memories of the traumatic medical procedures. Therefore, her treatment offered experiences designed to alter her current perceptions of the situations. Marcie's PTSD was successfully treated, and her brother's guilt was greatly reduced as a function of helping in her treatment.

Drugs can also be effective for symptoms of PTSD (Dent & Bremner, 2009). Some of the drugs, such as SSRIs (e.g., Prozac and Paxil), that are effective for anxiety disorders in general have been shown to be helpful for PTSD, perhaps because they relieve the severe anxiety and panic attacks so prominent in this disorder.

Concept Check 4.5

Match the correct preliminary diagnosis with the following cases: (a) acute posttraumatic stress disorder, (b) acute stress disorder, and (c) delayed onset posttraumatic stress disorder.

1. Judy witnessed a horrific tornado level her farm 3 weeks ago. Since then, she's had many flashbacks of the incident, trouble sleeping, and a fear of going outside in storms. _____
2. Jack was involved in a car accident 6 weeks ago in which the driver of the other car was killed. Since then, Jack has been unable to get into a car because it brings back the horrible scene he witnessed. Nightmares of the incident haunt him and interfere with his sleep. He is irritable and has lost interest in his work and hobbies. _____
3. Patricia was raped at the age of 17, which was 30 years ago. Just recently, she has been having flashbacks of the event, difficulty sleeping, and fear of sexual contact with her husband. _____

Obsessive-Compulsive Disorder

› **What are the symptoms of obsessive-compulsive disorder?**

A client with an anxiety disorder who needs hospitalization is likely to have **obsessive-compulsive disorder (OCD)**. A client referred for psychosurgery (neurosurgery for a psychological disorder) because every psychological and pharmacological treatment has failed and the suffering is unbearable probably has OCD. OCD is the devastating culmination of the anxiety disorders. It is not uncommon for someone with OCD to experience severe generalized anxiety, recurrent panic attacks, debilitating avoidance, and major depression, all occurring simultaneously with obsessive-compulsive symptoms.

Clinical Description

In other anxiety disorders the danger is usually in an external object or situation—or at least in the memory of one. In OCD the dangerous event is a thought, image, or impulse that the client attempts to avoid as completely as someone with a snake phobia avoids snakes (Clark & O'Connor, 2005). For example, has anyone ever told you not to think of pink elephants? If you really concentrate on not thinking of pink elephants, using every mental means possible, you will realize how difficult it is to suppress a suggested thought or image. Individuals with OCD fight this battle all day, every day, sometimes for most of their lives, and they usually fail miserably. In Chapter 3 we discussed the case of Frank, who experienced involuntary thoughts of epilepsy or seizures and prayed or shook his leg to try to distract himself. **Obsessions** are intrusive and mostly nonsensical thoughts, images, or urges that the individual tries to resist or eliminate. **Compulsions** are the thoughts or actions used to suppress the obsessions and provide relief. Frank had both obsessions and compulsions, but his disorder was mild compared to the case of Richard.

Richard | Enslaved by Ritual

Richard, a 19-year-old college freshman majoring in philosophy, withdrew from school because of incapacitating ritualistic behavior. He abandoned personal hygiene because the compulsive rituals that he had to carry out during washing or cleaning were so time consuming that he could do nothing else. Almost continual showering gave way to no showering. He stopped cutting and washing his hair and beard, brushing his teeth, and changing his clothes. He left his room infrequently and, to avoid rituals associated with the toilet, defecated on paper towels, urinated in paper cups, and stored the waste in the closet. He ate only late at night when his family was asleep. To be able to eat he had to exhale completely, making a lot of hissing noises, coughs, and hacks, and then fill his mouth with as much food as he could while no air was in his lungs. He would eat only a mixture of peanut butter, sugar, cocoa, milk, and mayonnaise. All other foods he considered contaminants. When he walked he took small steps on his toes while continually looking back, checking and rechecking.

Like everyone with OCD, Richard experienced intrusive and persistent thoughts and impulses; in his case they were about sex, aggression, and religion. His various behaviors were efforts to suppress sexual and aggressive thoughts or to ward off the disastrous consequences he thought would ensue if he did not perform his rituals. Compulsions can be either behavioral (handwashing or checking) or mental (thinking about certain words in a specific order, counting, praying, and so on) (Foa et al., 1996; Purdon, 2009; Steketee & Barlow, 2002). The important thing is that they are believed to reduce stress or prevent a dreaded event. Compulsions are often "magical" in that they often bear no logical relation to the obsession.

Types of Obsessions and Compulsions

Based on statistically associated groupings, there are four major types of obsessions (Bloch, Landeros-Weisenberger, Rosario, Pittenger, & Leckman, 2008; Mathews, 2009) and each is associated with a pattern of compulsive behavior. Symmetry obsessions account for most obsessions (26.7%), followed by "forbidden thoughts or actions" (21%), cleaning and contamination (15.9%), and hoarding (15.4%) (Bloch et al., 2008). Symmetry refers to keeping things in perfect order or doing something in a specific way. As a child, were you careful not to step on cracks in the sidewalk? You and your friends might have kept this up for a few minutes before tiring of it. But what if you had to spend your whole life avoiding cracks, on foot or in a car, to prevent something bad from happening? You wouldn't have much fun. People with aggressive (forbidden) obsessive impulses may feel they are about to yell out a swear word in church. One patient of ours, a young and moral woman, was afraid to ride the bus for fear that if a man sat down beside her she would grab his crotch! In reality, this would be the last thing she would do, but the aggressive urge was so horrifying that she made every attempt possible to suppress it and to avoid riding the bus or similar situations where the impulse might occur.

Certain kinds of obsessions are strongly associated with certain kinds of rituals (Bloch et al., 2008; Calamari et al., 2004; Leckman et al., 1997). For example, forbidden thoughts or actions seem to lead to checking rituals. Checking rituals serve to prevent an imagined disaster or catastrophe. Many are logical, such as repeatedly checking the stove to see whether you turned it off, but severe cases can be illogical. For example, Richard thought that if he did not eat in a certain way he might become possessed. If he didn't take small steps and look back, some disaster might happen to his family. A mental act, such as counting, can also be a compulsion. Obsessions with symmetry lead to ordering and arranging or repeating rituals; obsessions with contamination lead to washing rituals that may restore a sense of safety and control (Rachman, 2006). Like Richard, many patients have several kinds of obsessions and compulsions.

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▲ People with obsessive-compulsive hoarding are so afraid they may throw something important away that clutter piles up in their homes.

Tic Disorder and OCD

It is also common for tic disorder, characterized by involuntary movement (sudden jerking of limbs, for example), to co-occur in patients with OCD (particularly children) or in their families (Grados et al., 2001; Leckman et al., 2010). More complex tics with involuntary vocalizations are referred to as Tourette's disorder (Leckman et al., 2010; see Chapter 13). In some cases, these movements are not tics but may be compulsions, as they were in the case of Frank in Chapter 3 who kept jerking his leg if thoughts of seizures entered his head. Approximately 10% to 40% of children and adolescents with OCD also have had tic disorder at some point, leading to a suggestion that tic-related OCD be categorized as a subtype of OCD in *DSM-5* (Leckman et al., 2010). The obsessions in tic-related OCD are almost always related to symmetry.

Hoarding

Recently, a group of patients have come to the attention of specialty clinics because they compulsively hoard things, fearing that if they throw something away, even a 10-year-old newspaper, they then might urgently need it (Frost, Steketee, & Williams, 2002; Grisham & Barlow, 2005; Samuels et al., 2002; Steketee & Frost, 2007a, 2007b). It is not uncommon for some patients' houses and yards to come to the attention of public health authorities. One patient's house and yard was condemned because junk was piled so high it was both unsightly and a fire hazard. Among her hoard was a 20-year collection of used sanitary napkins!

Basically, these individuals usually begin acquiring things during their teenage years and often experience great pleasure, even euphoria, from shopping or otherwise collecting various items. Shopping or collecting things may be a re-

DSM Disorder Criteria Summary
Obsessive-Compulsive Disorder

Features of OCD include the following:

- Obsessions: Recurrent and persistent thoughts, impulses, or images that are experienced as intrusive and inappropriate and cause marked anxiety or distress; more than just excessive worries about real-life problems; the person attempts to ignore or suppress or neutralize them; the person recognizes that the thoughts, impulses, or images are a product of his or her own mind
- Compulsions: Repetitive behaviors (e.g., frequent handwashing or checking) or mental acts (e.g., praying or counting) that the person feels driven to perform in response to an obsession or according to rules that must be applied rigidly
- Recognition that the obsessions or compulsions are excessive or unreasonable
- The thoughts, impulses, or behaviors cause marked distress, consume more than an hour a day, or significantly interfere with the person's normal functioning or relationships

Source: Based on *DSM-IV-TR.* Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision). © 2000 American Psychiatric Association.

obsessive-compulsive disorder (OCD) Anxiety disorder involving unwanted, persistent, intrusive thoughts and impulses, as well as repetitive actions intended to suppress them.
obsessions Recurrent intrusive thought or impulse the client seeks to suppress or neutralize while recognizing it is not imposed by outside forces.
compulsions Repetitive, ritualistic, time-consuming behavior or mental act a person feels driven to perform.

Obsessive-Compulsive Disorder: Chuck

"I'm a little bit obsessive-compulsive. . . . It's a little difficult to deal with. The obsessive part—I'll get a thought in my head, and I can't put it out. It's just there all the time. I think about it when I go to bed, I think about it when I get up. . . . I'm a 'checker'—I have to check things. . . . I don't cook, but I have to check the stove every morning . . . not always really rational."

Go to Psychology CourseMate at www.cengagebrain.com to watch this video.

Abnormal Psychology: Inside Out, Vol. 111: produced by Ira Wohl, Only Child Motion Pictures

sponse to feeling down or depressed and is sometimes called, facetiously, "retail therapy." But unlike most people who like to shop or collect, these individuals then experience strong anxiety and distress about throwing anything away because everything has either some potential use or sentimental value in their minds, and their homes or apartments may become almost impossible to live in. Most of these individuals don't consider that they have a problem until family members or authorities insist that they receive help. The average age when these people come for treatment is approximately 50, after many years of hoarding (Grisham, Frost, Steketee, Kim, & Hood, 2006). Often they live alone.

Recent careful analysis of the rapidly increasing knowledge of hoarding suggests that it has both similarities and differences with OCD as well as with impulse control disorders, and that, perhaps, it should be listed as a separate disorder in *DSM-5* (Mataix-Cols et al., 2010). This is unlikely, although it may be assigned to the appendix of *DSM-5* for further study. New treatments are in development at our clinic that teach people to assign different values to objects and to reduce anxiety about throwing away items that are somewhat less valued (Steketee & Frost, 2007a). Preliminary results are promising, but more information on long-term effects of these treatments is needed.

Statistics

The lifetime prevalence of OCD is approximately 1.6% (Kessler, Berglund, et al., 2005), and in a given 1-year period the prevalence is 1% (Kessler, Chiu, et al., 2005). Not all cases meeting criteria for OCD are as severe as Richard's. Obsessions and compulsions can be arranged along a continuum, like most clinical features of anxiety disorders. Intrusive and distressing thoughts are common in nonclinical ("normal") individuals (Boyer & Liénard, 2008; Clark & Rhyno, 2005; Fullana et al., 2009). Spinella (2005) found that 13% of a "normal" community sample of people had moderate levels of obsessions or compulsions that were not severe enough to meet diagnostic criteria for OCD.

It would also be unusual *not* to have an occasional intrusive or strange thought. Many people have bizarre, sexual, or aggressive thoughts, particularly if they are bored—for example, when sitting in class. Gail Steketee and her colleagues collected examples of thoughts from ordinary people who do not have OCD. Some of these thoughts are listed in Table 4.7.

Have you had any of these thoughts? Most people do, but they are passing worries. Certain individuals, however, are horrified by such thoughts, considering them signs of an alien, intrusive, evil force. The majority of individuals with OCD are female, but the per-

Table 4.7 Obsessions and Intrusive Thoughts Reported by Nonclinical Samples*

Harming
Impulse to jump out of high window
Idea of jumping in front of a car
Impulse to push someone in front of train
Wishing a person would die
While holding a baby, having a sudden urge to kick it
Thoughts of dropping a baby
The thought that if I forget to say goodbye to someone, they might die
Thought that thinking about horrible things happening to a child will cause it
Contamination or Disease
Thought of catching a disease from public pools or other public places
Thoughts I may have caught a disease from touching toilet seat
Idea that dirt is always on my hand
Inappropriate or Unacceptable Behavior
Idea of swearing or yelling at my boss
Thought of doing something embarrassing in public, like forgetting to wear a top
Hoping someone doesn't succeed
Thought of blurting out something in church
Thought of "unnatural" sexual acts
Doubts About Safety, Memory, and So On
Thought that I haven't locked the house up properly
Idea of leaving my curling iron on the carpet and forgetting to pull out the plug
Thought that I've left the heater and stove on
Idea that I've left the car unlocked when I know I've locked it
Idea that objects are not arranged perfectly

*Examples were obtained from Rachman and deSilva (1978) and from unpublished research by Dana Thordarson, PhD, and Michael Kyrios, PhD (personal communications, 2000). *Source:* Reprinted, with permission, from Steketee, G., & Barlow, D. H. (2002). Obsessive-compulsive disorder. In D. H. Barlow, *Anxiety and its disorders: The nature and treatment of anxiety and panic* (2nd ed., p. 529), © 2002 Guilford Press.

centage is not as large as for some other anxiety disorders. Rasmussen and Tsuang (1984, 1986) reported that 55% of 1,630 patients with OCD were female. An epidemiology study noted 60% females in their sample of people with OCD (Karno & Golding, 1991). Interestingly, in children the sex ratio is reversed, with more males than females (Hanna, 1995). This seems to be because boys tend to develop OCD earlier. By mid-adolescence, the sex ratio is approximately equal before becoming predominantly female in adulthood (Albano et al., 1996). Age of onset ranges from childhood through the 30s, with a median age of onset of 19 (Kessler, Berglund, et al., 2005). The age of onset peaks earlier in males (at 13 to 15) than in females (at 20 to 24) (Rasmussen & Eisen, 1990). Once OCD develops, it tends to become chronic (Eisen & Steketee, 1998; Steketee & Barlow, 2002).

OCD looks remarkably similar across cultures. Insel (1984) reviewed studies from England, Hong Kong, India, Egypt, Japan, and Norway and found essentially similar types and proportions of obsessions and compulsions, as did Weissman and colleagues (1994) reviewing studies from Canada, Finland, Taiwan, Africa, Puerto Rico, Korea, and New Zealand.

Causes

Many of us sometimes have intrusive, even horrific, thoughts and occasionally engage in ritualistic behavior, especially when we are under stress (Parkinson & Rachman, 1981a, 1981b). But few of us develop OCD. Again, as with panic disorder and PTSD, someone must develop anxiety focused on the possibility of having additional intrusive thoughts.

The repetitive, intrusive, unacceptable thoughts of OCD may well be regulated by the hypothetical brain circuits described in Chapter 2. However, the tendency to develop anxiety over having additional compulsive thoughts may have the same generalized biological and psychological precursors as anxiety in general (Suárez et al., 2009).

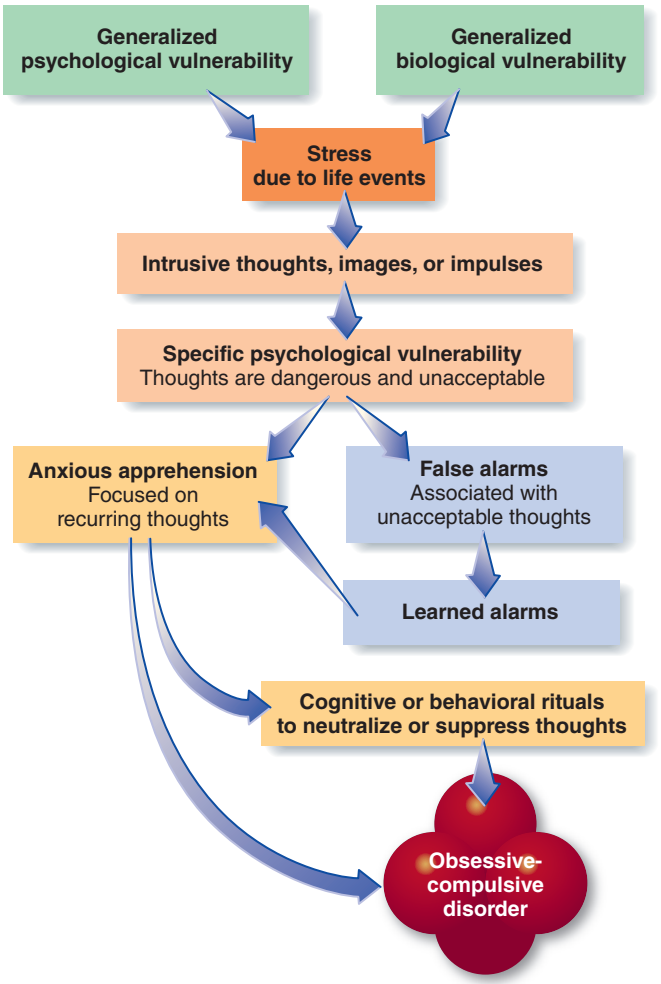
Why would people with OCD focus their anxiety on the occasional intrusive thought rather than on the possibility of a panic attack or some other external situation? One hypothesis is that early experiences taught them that some thoughts are dangerous and unacceptable because the terrible things they are thinking might happen and they would be responsible. These early experiences would result in a specific psychological vulnerability to develop OCD. When clients with OCD equate thoughts with the specific actions or activity represented by the thoughts, this is called *thought–action fusion*. Thought–action fusion may, in turn, be caused by attitudes of excessive responsibility and resulting guilt developed during childhood when even a bad thought is associated with evil intent (Clark & O'Connor, 2005; Salkovskis, Shafran, Rachman, & Freeston, 1999; Steketee & Barlow, 2002). They may learn this through the same process of misinformation that convinced the person with snake phobia that snakes were dangerous and could be everywhere. One patient believed thinking about abortion was the moral equivalent of having an abortion. Richard finally admitted to having strong homosexual impulses that were unacceptable to him and to his minister father, and he believed the impulses were as sinful as actual acts. Many people with OCD who believe in the tenets of fundamental religions, whether Christian, Jewish, or Islamic, present with similar attitudes of inflated responsibility and thought–action fusion. Several studies showed that the strength of religious belief, but not the type of belief, was associated with thought–action fusion and severity of OCD (Rassin & Koster, 2003; Steketee, Quay, & White, 1991). Of course, most people with fundamental religious beliefs do not develop OCD. But what if the most frightening thing in your life was not a snake or speaking in public but a terrible thought that happened to pop into your head? You can't avoid it as you would a snake, so you resist this thought by attempting to suppress it or "neutralize" it using mental or behavioral strategies, such as distraction, praying, or checking. These strategies become compulsions, but they are doomed to fail in the long term, because these strategies backfire and actually increase the frequency of the thought (Purdon, 1999; Wegner, 1989).

Again, generalized biological and psychological vulnerabilities must be present for this disorder to develop. Believing some thoughts are unacceptable and therefore must be suppressed (a specific psychological vulnerability) may put people at greater risk of OCD (Amir, Cashman, & Foa, 1997; Parkinson & Rachman, 1981b; Salkovskis & Campbell, 1994). A model of the etiology of OCD that is somewhat similar to other models of anxiety disorders is presented in ■ Figure 4.11.

Treatment

Studies evaluating the effects of drugs on OCD are showing some promise (Steketee & Barlow, 2002; Stewart, Jenike, & Jenike, 2009). The most effective seem to be those that specifically inhibit the reuptake of serotonin, such as clomipramine or the SSRIs, which benefit up to 60% of patients with OCD, with no particular advantage to one drug over another. However, the average treatment gain is moderate at best (Greist, 1990), and relapse often occurs when the drug is discontinued (Lydiard, Brawman-Mintzer, & Ballenger, 1996).

Highly structured psychological treatments work somewhat better than drugs, but they are not readily available. The most effective approach is called *exposure and ritual prevention (ERP),* a process whereby the rituals are actively prevented and the patient is systematically and gradually exposed to the feared thoughts or situations (Barlow & Lehman, 1996; Franklin & Foa, 2008; Steketee & Barlow, 2002). Richard, for example, would be systematically exposed to harmless objects or situations that he thought were contaminated, including certain foods and household chemicals, and his washing and checking rituals would be prevented. Usually this can be done by simply working closely with patients to see that they do not wash or check. In severe cases, patients may be hospitalized and the faucets removed



■ **Figure 4.11** A model of the causes of obsessive-compulsive disorder. (Reprinted, with permission, from White, K. S., & Barlow, D. H., A model of the causes obsess-compulsive disorder. In Barlow, D. H., *Anxiety and its disorders: The nature and treatment of anxiety and panic* (2nd ed., p. 536). New York: Guilford Press, © 2002 Guilford Press.)

from the bathroom sink for a period to discourage repeated washing. However the rituals are prevented, the procedures seem to facilitate "reality testing," because the client soon learns, at an emotional level, that no harm will result whether he carries out the rituals or not.

Studies are now available examining the combined effects of medication and psychological treatments. In one large study (Foa et al., 2005) ERP was compared to the drug clomipramine, as well as to a combined condition. ERP, with or without the drug, produced superior results to the drug alone, with 86% responding to ERP alone versus 48% to the drug alone. Combining the treatments did not produce any additional advantage. Also, relapse rates were high from the medication-only group when the drug was withdrawn.

Psychosurgery is one of the more radical treatments for OCD. "Psychosurgery" is a misnomer that refers to neurosurgery for a psychological disorder. Jenike and colleagues (1991) reviewed the records of 33 patients with OCD, most of them extremely severe cases who had failed to respond to either drug or psychological treatment. After a specific surgical lesion to the cingulate bundle (cingulotomy), approximately 30% benefited substantially. Similarly, Rück et al. (2008) performed a related surgery (capsulotomy) on 25 patients who had not responded to 5 years of previous treatment; 35% (9 patients) benefited substantially, but 6 of those 9 patients suffered from serious adverse side effects of the surgery. Considering that these patients seemed to have no hope from other treatments, surgery deserves consideration as a last resort.

Concept Check 4.6

Fill in the blanks to form facts about OCD.

1. _____ are intrusive and nonsensical thoughts, images, or urges an individual tries to eliminate or suppress.
2. The practices of washing, counting, and hoarding to suppress obsessions and provide relief are called _____.
3. The lifetime prevalence of OCD is approximately _____ or even lower.
4. _____ is a radical treatment for OCD involving a surgical lesion to the cingulate bundle.

On the Spectrum Emerging Views of Anxiety Disorders

In Chapter 3 we introduced the idea that emerging conceptions of psychopathology move us away from an emphasis on categorical (individual) diagnoses to a consideration of larger dimensions, or spectra, in which similar and related diagnoses might be grouped. One such spectrum consists of what some call emotional disorders, including anxiety and depression (Leyfer & Brown, 2011). But how would this dimensional approach to psychopathology change the way we make diagnoses? Recently, we speculated on how a future diagnostic system using dimensional approaches for emotional disorders might work (Brown & Barlow, 2009). To illustrate this approach, let's first consider a case from our clinic.

Mr. S was a high school teacher in his midfifties who had been in a very serious car accident several months before com-

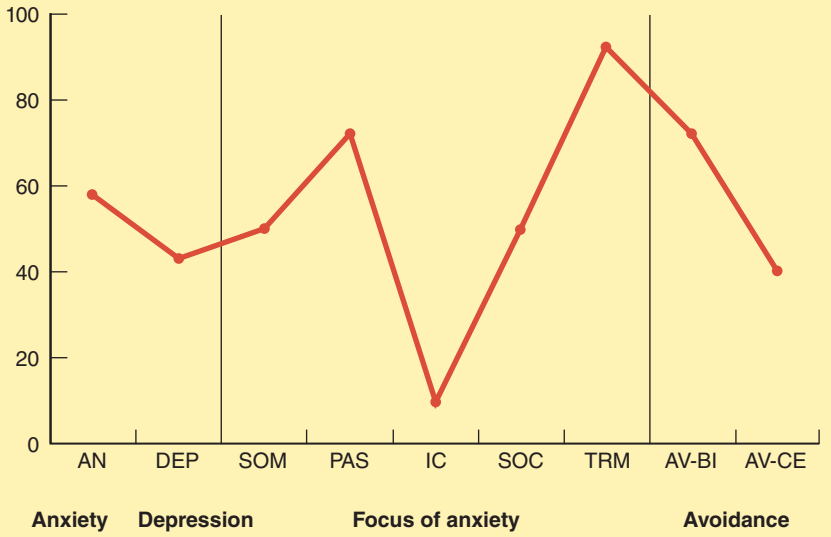
ing in and was suffering from symptoms related to that accident. These included intrusive memories of the crash, "flashbacks" of the accident itself that were very intense emotionally, and images of the cuts and bruises on his wife's face. He also had a strong startle reaction to any cues that reminded him of the accident and avoided driving in certain locations that were somewhat similar to where he had his accident. These symptoms intermingled with a similar set of symptoms emerging from a series of traumatic experiences that had occurred during his service in the Vietnam War. In addition to these trauma symptoms, he also spent a lot of his day worrying about various life events including his own health and that of his family. He also worried about his performance at work and whether he would be evaluated poorly by other staff members, despite the fact that he received consistently high evaluations for his teaching.

After considering everything he said and evaluating him clinically, it was clear that he met criteria for PTSD. He also met criteria for GAD given his substantial worry that was occurring every day about life events unrelated to the trauma. In addition he had some mild depression, perhaps due in part to all of the anxiety he was experiencing. In summary, the patient could be diagnosed with PTSD, although he had substantial features of GAD and depression. But what would it look like if we attempted to describe his symptoms on a series of dimensions rather than on whether they meet criteria for one category or another? ■ Figure 4.12 displays a simplified version of one possible dimensional system (Brown & Barlow, 2009). In this dimensional scheme, "anxiety" (AN) is represented on the left because all individuals with anxiety or depressive disorders have some level of anxiety. Many individuals, but not all, are also depressed (DEP) (as was Mr. S). Mr. S would score fairly high on anxiety and somewhat lower on depression. Looking to the far right of the figure, Mr. S displayed a lot of behavioral avoidance and avoidance of physical sensations (interoceptive avoidance) (AV-BI). Mostly he was having difficulty driving and also would avoid cues connected with his earlier trauma by refusing if at all possible to engage in activities or conversations associated with the war. Another related type of avoidance is when you avoid experiencing intense emotions or thoughts about emotional experiences. We call this cognitive and emotional avoidance (AV-CE) and Mr. S also scored relatively high on this aspect of avoidance.

But what was the focus of Mr. S's anxiety? Here we look at five characteristics that currently categorize anxiety disorder diagnoses. Looking first at trauma (TRM) focus, obviously, this earned the highest score on Mr. S's profile. He also was suffering from frequent flashbacks to his traumatic experiences, which as you may remember, are similar to panic attacks and consist of strong autonomic surges, such as rapidly increasing heart rate. Thus, he scored high on panic and related autonomic surges (PAS). Other kinds of intrusive obsessive thoughts were not present and he scored low on this dimension (IC). His worry about his health and the health of his family caused him to score moderately high on somatic anxiety (SOM), but social anxiety (SOC) was not particularly high.

As you can see, this dimensional profile provides a more complete picture of Mr. S's clinical presentation than simply noting that he met criteria for PTSD. This is because the profile captures the relative severity of a number of key features of anxiety and mood disorders that are often present together in patients who might meet criteria for only a single diagnosis in the current categorical system. This profile also captures the fact that Mr. S had some depression that was below the severity threshold to meet criteria for mood disorder. Knowing all of this by glancing at Mr. S's profile in Figure 4.12 should help clinicians match therapy more closely to his presenting problems.

This is just one possible example, but it does provide some idea of what a diagnostic system might look like in the future. Although this system would not be ready for *DSM-5* (to be published in 2013) because we would need to do much more research on how best to make it work, a system like this might be ready for *DSM-6*.



■ **Figure 4.12** Proposed *DSM-5* (or *6*) Dimensional Diagnosis of a Patient with PTSD. *AN,* anxiety; *DEP,* unipolar depression; *SOM,* somatic anxiety; *PAS,* panic and related autonomic surges; *IC,* intrusive cognitions; *SOC,* social evaluation; *TRM,* past trauma; *AV-BI,* behavioral and interoceptive avoidance; *AV-CE,* cognitive and emotional avoidance. Higher scores on the *y*-axis (0–100) indicate higher levels of the *x*-axis dimension, but otherwise the *y*-axis metric is arbitrary and is used for illustrative purposes. (Adapted from Brown, T. A., & Barlow, D. H., 2009. A proposal for a dimensional classification system based on the shared features of the DSM-IV anxiety and mood disorders: Implications for assessment and treatment. *Psychological Assessment, 21*(3), 267. © 2009 by American Psychological Association. Reprinted with permission.)

Summary

The Complexity of Anxiety Disorders

What are the similarities and differences among anxiety, fear, and panic attacks?

- Anxiety is a future-oriented state characterized by negative affect in which a person focuses on the possibility of uncontrollable danger or misfortune; in contrast, fear is a present-oriented state characterized by strong escapist tendencies and a surge in the sympathetic branch of the autonomic nervous system in response to current danger.
- A panic attack represents the alarm response of real fear, but there is no actual danger.
- Panic attacks may be (1) unexpected (without warning), (2) situationally bound (always occurring in a specific situation), or (3) situationally predisposed (likely but unpredictable in a specific situation).
- Panic and anxiety combine to create different anxiety disorders.

Generalized Anxiety Disorder

What are the essential features, possible causes, and available treatment approaches for generalized anxiety disorder (GAD)?

- In generalized anxiety disorder, anxiety focuses on minor everyday events, not one major worry or concern.
- Both genetic and psychological vulnerabilities seem to contribute to the development of GAD.
- Although drug and psychological treatments may be effective in the short term, drug treatments are no more effective in the long term than placebo treatments. Successful treatment may help individuals with GAD focus on what is really threatening to them in their lives.

Panic Disorder With and Without Agoraphobia

What are the essential features of panic disorder?

- In panic disorder with or without agoraphobia (a fear and avoidance of situations considered to be "unsafe"), anxiety is focused on the next panic attack.
- We all have some genetic vulnerability to stress, and many of us have had a neurobiological overreaction to some stressful event—that is, a panic attack. Individuals who develop panic disorder then develop anxiety over the possibility of having another panic attack.
- Both drug and psychological treatments have proved successful in the treatment of panic disorder. One psychological method, panic control treatment, concentrates on exposing patients to clusters of sensations that remind them of their panic attacks.

Specific Phobia

What are the principal causes of specific phobia?

- In phobic disorders, the individual avoids situations that produce severe anxiety, panic, or both. In specific phobia, the fear is focused on a particular object or situation.
- Phobias can be acquired by experiencing some traumatic event; they can also be learned vicariously or even be taught.

What strategies are typically used to treat specific phobia?

- Treatment of phobias is rather straightforward, with a focus on structured and consistent exposure-based exercises.

Social Phobia

What are the principal causes of social phobia?

- Social phobia is a fear of being around others, particularly in situations that call for some kind of "performance" in front of other people.

What strategies are used to treat social phobia?

- Although the causes of social phobia are similar to those of specific phobias, treatment has a different focus that includes rehearsing or role-playing socially phobic situations. In addition, drug treatments have been effective.

Posttraumatic Stress Disorder

What are the essential features and possible causes of posttraumatic stress disorder?

- Posttraumatic stress disorder (PTSD) focuses on avoiding thoughts or images of past traumatic experiences.
- The precipitating cause of PTSD is obvious—a traumatic experience. But mere exposure to trauma is not enough. The intensity of the experience seems to be a factor in whether an individual develops PTSD; biological vulnerabilities, as well as social and cultural factors, appear to play a role as well.

What treatment approaches are available for posttraumatic stress disorder?

- Treatment involves reexposing the victim to the trauma and reestablishing a sense of safety to overcome the debilitating effects of PTSD.

Obsessive-Compulsive Disorder

What are the symptoms of obsessive-compulsive disorder?

- Obsessive-compulsive disorder (OCD) focuses on avoiding frightening or repulsive intrusive thoughts (obsessions) or neutralizing these thoughts through the use of ritualistic behavior (compulsions).
- As with all anxiety disorders, biological and psychological vulnerabilities seem to be involved in the development of OCD.
- Drug treatment seems to be only modestly successful in treating OCD. The most effective treatment approach is a psychological treatment called exposure and ritual prevention (ERP).

Key Terms

anxiety, 117
fear, 118
panic, 118
panic attack, 118
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obsessions, 152
compulsions, 152

Answers to Concept Checks

4.1

1. b; 2. c; 3. e, d; 4. a; 5. f

4.2

1. T; 2. F (more gradual); 3. T; 4. F; 5. T

4.3

1. F (with agoraphobia); 2. F (3.5%); 3. T; 4. T

4.4

1. d; 2. e; 3. c; 4. f; 5. a; 6. d; 7. c

4.5

1. b; 2. a; 3. c

4.6

1. obsessions; 2. compulsions; 3. 1.6%; 4. psychosurgery

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- *Steve, a Patient with Panic Disorder:* Steve discusses how panic attacks have disrupted his life.
- *Chuck, a Client with Obsessive-Compulsive Disorder:* Chuck discusses how his obsessions affect his everyday life, going to work, planning a vacation, and so on.
- *Virtual Reality Therapy:* A virtual reality program helps one woman overcome her fear of riding the subway.
- *Snake Phobia Treatment:* A demonstration of exposure therapy helps a snake phobic overcome her severe fear of snakes in just 3 hours.

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Video Concept Reviews

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- Anxiety
- Fear
- Characteristics of Anxiety Disorders
- Panic
- Panic Attacks
- Generalized Anxiety Disorder (GAD)—Description
- Panic Disorder
- Panic Control Treatment
- Concept Check: Medical Versus Psychological Treatment
- Specific Phobia
- Phobia Subtypes
- Social Phobia
- Posttraumatic Stress Disorder (PTSD)
- Obsessive-Compulsive Disorder (OCD)

Chapter Quiz

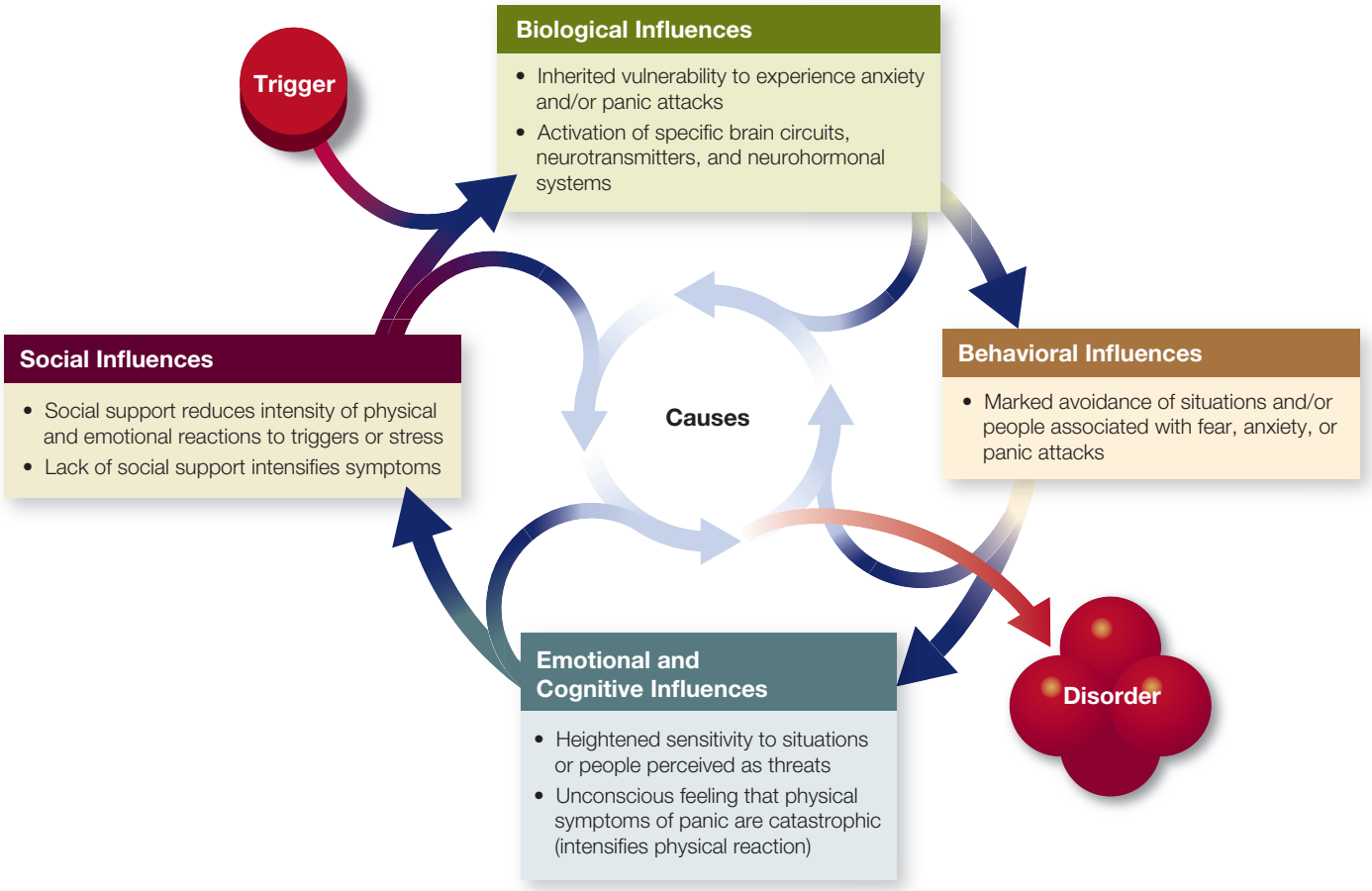
1. _____ is a psychological experience characterized by concern about future events, and _____ is characterized by concern about current circumstances.
   a. Panic; anxiety
   b. Fear; anxiety
   c. Anxiety; fear
   d. Depression; anxiety
2. In an integrated model of anxiety, which childhood experience appears to make an individual more vulnerable to anxiety in adulthood?
   a. negative and inconsistent attention from parents
   b. exposure to situations that reinforce a rigid sense of personal control
   c. interactions with peers that are violent
   d. academic failures in preschool
3. Which of the following is true about generalized anxiety disorder?
   a. It is most common in individuals aged 15–24 years.
   b. Its course tends to be chronic.
   c. It is the least common of the anxiety disorders.
   d. It is more common in men.
4. Why are the majority of people who suffer from agoraphobia women?
   a. Chromosomal features related to sensitivity of the hypothalamus–pituitary axis are more common in women.
   b. Women are more likely to use cognitive distortions in which they appraise events as threatening.
   c. The hormonal system in women sensitizes the female nervous system to stress.
   d. Cultural factors make it more acceptable for women to avoid situations and to report their fears.
5. Marty has a fear of dogs. Which of the following suggests that his fear qualifies as a specific phobia rather than just an everyday fear?
   a. Marty's fear of dogs comes and goes following an episodic pattern.
   b. Marty owns a cat but no dog.
   c. Marty believes that his fear of dogs is reasonable and appropriate.
   d. Marty will only work night shifts, a time when he thinks all dogs will be safely inside.

6. Which technique appears to be the most effective treatment for phobias?
   a. exposure to the feared stimulus under therapeutic supervision
   b. rapid and repeated exposure to the feared stimulus followed by immediate escape
   c. hypnosis during which fear-related conflicts are banished from the unconscious
   d. challenging the client to see that the fears are irrational, unrealistic, and excessive
7. Which of the following is the most essential characteristic of social phobia?
   a. fear of being in public places
   b. fear of being left alone
   c. fear of evaluation by other people
   d. fear of having a panic attack
8. Which feature differentiates posttraumatic stress disorder from acute stress disorder?
   a. the time since the traumatic event occurred
   b. the severity of the symptoms
   c. the nature of the symptoms
   d. the presence of emotional numbing
9. Every morning when he leaves for work Anthony has recurring doubts about whether he locked his front door. He continues thinking about this throughout the day, to the distraction of his work. Anthony is experiencing:
   a. obsession
   b. derealization
   c. panic
   d. compulsion
10. When a person believes that thinking about hurting someone is just as bad as actually hurting someone, that person is experiencing:
   a. obsession
   b. a false alarm
   c. a panic attack
   d. thought-action fusion

Exploring Anxiety Disorders

People with anxiety disorders:

- Feel overwhelming tension, apprehension, or fear when there is no actual danger
- May take extreme action to avoid the source of their anxiety



TREATMENT FOR ANXIETY DISORDERS

Cognitive-Behavioral Therapy

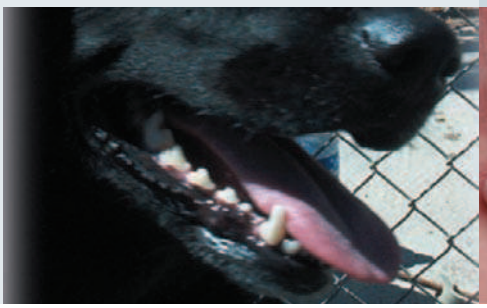
- Systematic exposure to anxiety-provoking situations or thoughts
- Learning to substitute positive behaviors and thoughts for negative ones
- Learning new coping skills: relaxation exercises, controlled breathing, etc.

Drug Treatment

- Reduces the symptoms of anxiety disorders by influencing brain chemistry
  —antidepressants (Tofranil, Paxil, Effexor)
  —benzodiazepines (Xanax, Klonopin)

Other Treatments

- Managing stress through a healthy lifestyle: rest, exercise, nutrition, social support, and moderate alcohol or other drug intake



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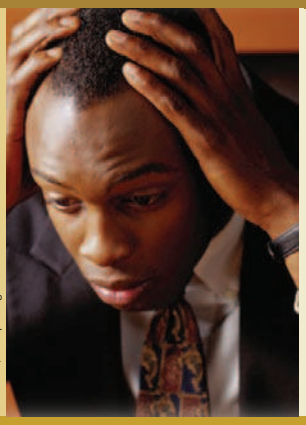
TYPES OF ANXIETY DISORDERS

Panic

People with panic disorders have had one or more panic attacks and are anxious and fearful about having future attacks.



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What is a panic attack?

A person having a panic attack feels:

- Apprehension leading to intense fear
- Sensation of "going crazy" or of losing control
- Physical signs of distress: racing heartbeat, rapid breathing, dizziness, nausea, or sensation of heart attack or imminent death

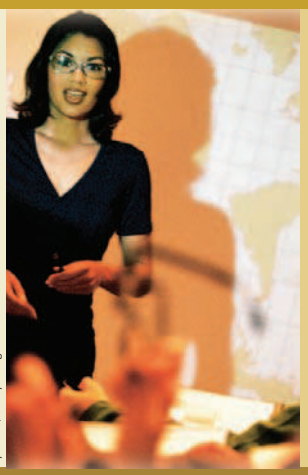
When/why do panic attacks occur?

Panic attacks can be:

- Situationally bound: Always occurring in the same situation, which may lead to extreme avoidance of triggering people, places, or events (see specific and social phobias)
- Unexpected: Can lead to extreme avoidance of any situation or place felt to be unsafe (agoraphobia)
- Situationally predisposed: Attacks may or may not occur in specific situations (between situationally bound and unexpected)

Phobias

People with phobias avoid situations that produce severe anxiety and/or panic. There are three main types:



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Agoraphobia

- Fear and avoidance of situations, people, or places where it would be unsafe to have a panic attack: malls, grocery stores, buses, planes, tunnels, etc.
- In the extreme, inability to leave the house or even a specific room
- Begins after a panic attack but can continue for years even if no other attacks occur

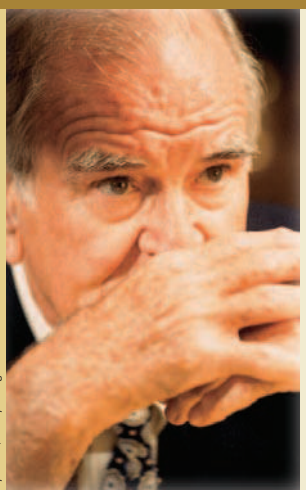
Specific Phobia

- Fear of specific object or situation that triggers attack: heights, closed spaces, insects, snakes, or flying
- Develops from personal or vicarious experience of traumatic event with the triggering object or situation or from misinformation

Social Phobia

- Fear of being called for some kind of "performance" that may be judged: speaking in public, using a public restroom (for males), or generally interacting with people

Other Types



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Generalized Anxiety

- Uncontrollable unproductive worrying about everyday events
- Feeling impending catastrophe even after successes
- Inability to stop the worry–anxiety cycle: e.g., Irene's fear of failure about school relationships and health even though everything seemed fine
- Physical symptoms of muscle tension

Posttraumatic Stress

- Fear of reexperiencing a traumatic event: rape, war, life-threatening situation, etc.
- Nightmares or flashbacks (of the traumatic event)
- Avoidance of the intense feelings of the event through emotional numbing

Obsessive-Compulsive

- Fear of unwanted and intrusive thoughts (obsessions)
- Repeated ritualistic actions or thoughts (compulsions) designed to neutralize the unwanted thoughts: e.g., Richard's attempts to suppress "dangerous" thoughts about sex, aggression, and religion with compulsive washing and cleaning rituals

CHAPTER 5

Somatoform and Dissociative Disorders

Chapter Outline

Student Learning Outcomes*

Use the concepts, language, and major theories of the discipline to account for psychological phenomena.	› Describe behavior and mental processes empirically, including operational definitions *(see textbook pages 166–168, 170–171, 172–176, 178–181, 183–189)*
Identify appropriate applications of psychology in solving problems, such as:	› Origin and treatment of abnormal behavior *(see textbook pages 168–169, 171–172, 176–177, 181–182, 189–193)*

*Portions of this chapter cover learning outcomes suggested by the American Psychological Association (2007) in their guidelines for the undergraduate psychology major. Chapter coverage of these outcomes is identified by APA Goal and APA Suggested Learning Outcome (SLO).

Many people continually run to the doctor even though there is nothing really wrong with them. This is usually a harmless tendency that may even be worth some good-natured jokes. But for a few individuals, the preoccupation with their health or appearance becomes so great that it dominates their lives. Their problems fall under the general heading of **somatoform disorders**. *Soma* means body, and the problems preoccupying these people seem, initially, to be physical disorders. What the somatoform disorders have in common is that there is usually no identifiable medical condition causing the physical complaints. Thus, these disorders are grouped under the shorthand label of "medically unexplained physical symptoms" (olde Hartman et al., 2009; Woolfolk & Allen, 2011).

Have you ever felt "detached" from yourself or your surroundings? ("This isn't really me," or "That doesn't really look like my hand," or "There's something unreal about this place.") During these experiences, some people feel as if they are dreaming. These mild sensations that most people experience occasionally are slight alterations, or detachments, in consciousness or identity, and they are known as *dissociation* or *dissociative experiences*. For a few people, these experiences are so intense and extreme that they lose their identity entirely and assume a new one or they lose their memory or sense of reality and are unable to function. We discuss several types of **dissociative disorders** in the second half of this chapter.

Somatoform and dissociative disorders are strongly linked historically, and evidence indicates they share common features (Kihlstrom, Glisky, & Anguilo, 1994; Prelior, Yutzy, Dean, & Wetzel, 1993). They used to be categorized under one general heading: "hysterical neurosis." You may remember (from Chapter 1) that the term *hysteria* suggests that the cause of these disorders, which were thought to occur primarily in women, can be traced to a "wandering uterus." But the term *hysterical* came to refer more generally to physical symptoms without known organic cause or to dramatic or "histrionic" behavior thought to be characteristic of women. Sigmund Freud (1894–1962) suggested that in a condition called *conversion hysteria* unexplained physical symptoms indicated the conversion of unconscious emotional conflicts into a more acceptable form. The historical term *conversion* remains with us (without the theoretical implications); however, the prejudicial and stigmatizing terms *hysteria* and *hysterical* are no longer used.

The term *neurosis,* as defined in psychoanalytic theory, suggested a specific cause for certain disorders. Specifically, neurotic disorders resulted from underlying unconscious conflicts, anxiety that resulted from those conflicts, and the implementation of ego defense mechanisms. *Neurosis* was eliminated from the diagnostic system in 1980 because it was too vague, applying to almost all nonpsychotic disorders, and because it implied a specific but unproven cause for these disorders.

Somatoform and dissociative disorders are not well understood, but they have intrigued psychopathologists and the public for centuries. A fuller understanding provides a rich perspective on the extent to which normal, everyday traits found in all of us can evolve into distorted, strange, and incapacitating disorders.

somatoform disorder Pathological concern of individuals with the appearance or functioning of their bodies, usually in the absence of any identifiable medical condition.
dissociative disorder Disorder in which individuals feel detached from themselves or their surroundings and feel reality, experience, and identity may disintegrate.

Somatoform Disorders

› What are the defining features of somatoform disorders?
› What treatments have been developed for somatoform disorders?

The *Diagnostic and Statistical Manual of Mental Disorders,* 4th edition, Text Revision *(DSM-IV-TR)* lists five basic somatoform disorders: hypochondriasis, somatization disorder, pain disorder, conversion disorder, and body dysmorphic disorder. In each, individuals are pathologically concerned with the appearance or functioning of their bodies. The first three disorders covered in this section—hypochodriasis, somatization disorder, and pain disorder—overlap considerably, and the proposal for *DSM-5* is to combine these three disorders into a new category called complex somatic symptom disorder (American Psychiatric Association, 2010f).

Hypochondriasis

Like many terms in psychopathology, **hypochondriasis** has ancient roots. To the Greeks, the *hypochondria* was the region below the ribs, and the organs in this region affected mental state. For example, ulcers and other gastric disorders were once considered part of the hypochondriac syndrome. As the actual physical causes of such disorders were discovered they were no longer considered a mental disorder, but physical complaints without a clear cause continued to be labeled *hypochondriasis* (Barsky, Wyshak, & Klerman, 1986; Taylor & Asmundson, 2009; Woolfolk & Allen, in press). In hypochondriasis, as we know it today, severe anxiety is focused on the possibility of having a serious disease. The threat seems so real that reassurance from physicians does not seem to help. Consider the case of Gail.

Gail | Invisibly Ill

Gail was married at 21 and looked forward to a new life. As one of many children in a lower-middle-class household, she felt weak and somewhat neglected and suffered from low self-esteem, but she believed that marriage would solve everything; she was finally someone special. Unfortunately, it didn't work out that way. She soon discovered her husband was continuing an affair with an old girlfriend.

Three years after her wedding, Gail came to our clinic. Although she complained initially of anxiety and stress, it soon became clear that her major concerns were about her health. Any time she experienced minor physical symptoms such as breathlessness or a headache, she was afraid she had a serious illness. A headache indicated a brain tumor. Breathlessness was an impending heart attack. Other sensations were quickly elaborated into the possibility of AIDS or cancer. Gail was afraid to go to sleep at night for fear that she would stop breathing. She avoided exercise, drinking, and even laughing because the resulting sensations upset her.

The major trigger of uncontrollable anxiety and fear was the news in the newspaper and on television. Each time an article or show appeared on the "disease of the month," Gail found herself irresistibly drawn into it, intently noting symptoms that were part of the disease. For days afterward she was vigilant, looking for the symptoms in herself and others. She even watched her dog closely to see whether he was coming down with the dreaded disease. Only with great effort could she dismiss these thoughts after several days. Real illness in a friend or relative would incapacitate her for days at a time.

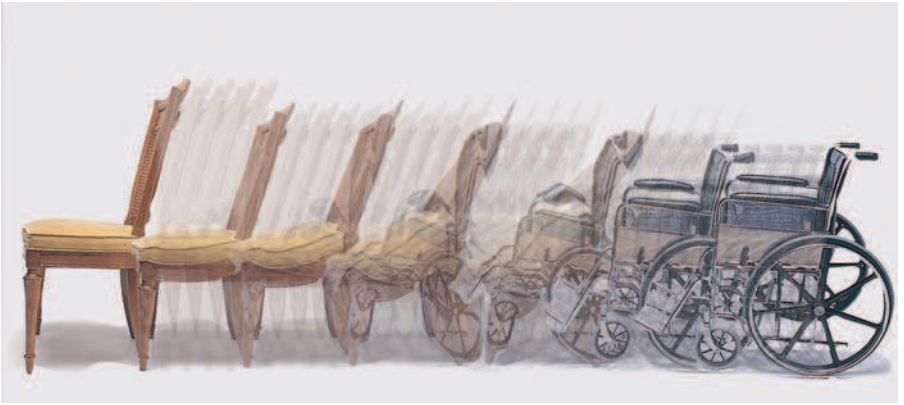
Gail's fears developed during the first year of her marriage, around the time she learned of her husband's affair. At first, she spent a great deal of time and more money than they could afford going to doctors. Over the years, she heard the same thing during each visit: "There's nothing wrong with you. You're perfectly healthy." Finally, she stopped going, as she became convinced her concerns were excessive, but her fears did not go away and she was chronically miserable.

Clinical Description

Gail's problems are fairly typical of hypochondriasis. Research indicates that hypochondriasis shares many features with the anxiety and mood disorders, particularly panic disorder (Craske et al., 1996; Creed & Barsky, 2004), including similar age of onset, personality characteristics, and patterns of familial aggregation (running in families). Indeed, anxiety and mood disorders are often comorbid with hypochondriasis——that is, if individuals with a hypochondriacal disorder have additional diagnoses, these most likely are anxiety or mood disorders (Côté et al., 1996; Creed & Barsky, 2004; Rief, Hiller, & Margraf, 1998; Simon, Gureje, & Fullerton, 2001). The *DSM-5* committee is even considering the possibility that many individuals with hypochondriasis might be better considered to have an anxiety disorder, a position that receives wide support (Taylor & Asmundson, 2009).

Hypochondriasis is characterized by anxiety or fear that one has a serious disease. Therefore, the essential problem is anxiety, but its expression is different from that of the

other anxiety disorders. In hypochondriasis, the individual is preoccupied with bodily symptoms, misinterpreting them as indicative of illness or disease. Almost any physical sensation may become the basis for concern for individuals with hypochondriasis. Some may focus on normal bodily functions such as heart rate or perspiration, and others may focus on minor physical abnormalities such as a cough. Some individuals complain of vague symptoms, such as aches or fatigue. Because a key feature of this disorder is preoccupation with physical symptoms, individuals with hypochondriasis almost always go initially to family physicians. They come to the attention of mental health professionals only after family physicians have ruled out realistic medical conditions as a cause of the patient's symptoms.



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▲ In hypochondriasis, normal experiences and sensations are often transformed into life-threatening illnesses.

Another important feature of hypochondriasis is that reassurances from numerous doctors that all is well and the individual is healthy have, at best, only a short-term effect. It isn't long before patients like Gail are back in the office of another doctor on the assumption that the previous doctors have missed something. This is because many of these individuals mistakenly believe they have a disease, a difficult to shake belief sometimes referred to as "disease conviction" (Côté et al., 1996; Haenen, de Jong, Schmidt, Stevens, & Visser, 2000). Therefore, along with anxiety focused on the possibility of disease or illness, disease conviction is a core feature of hypochondriasis (Benedetti et al., 1997; Kellner, 1986; Woolfolk & Allen, in press).

Minor, seemingly hypochondriacal concerns are common in young children, who often complain of abdominal aches and pains that do not seem to have a physical basis. In most cases, these complaints are passing responses to stress and do not develop into a full-blown chronic hypochondriacal syndrome.

Statistics

Prevalence of hypochondriasis in the general population is estimated to be from 1% to 5% (APA, 2000). A review of five studies in primary care settings suggests that the median prevalence rate for hypochondriasis in these settings is 6.7% (Creed & Barsky, 2004). Although historically considered one of the "hysterical" disorders unique to women, the sex ratio is actually closer to 50:50 (Creed & Barsky, 2004; Kellner, 1986; Kirmayer & Robbins, 1991; Kirmayer, Looper, & Taillefer, 2003). It was thought for a long time that hypochondriasis was more prevalent in elderly populations, but this does not seem to be true (Barsky, Frank, Cleary, Wyshak, & Klerman, 1991). In fact, hypochondriasis is spread fairly evenly across various phases of adulthood. Naturally, more older adults go to see physicians, making the *total number* of patients with hypochondriasis in this age group somewhat higher than in the younger population, but the proportion of all those seeing a doctor who have hypochondriasis is about the same. Hypochondriasis may emerge at any time of life, with the peak age periods found in adolescence, middle age (40s and 50s), and after age 60 (Kellner, 1986). As with most anxiety and mood disorders, hypochondriasis is chronic (Taylor & Asmundson, 2009).

As with anxiety disorders, culture-specific syndromes seem to fit comfortably with hypochondriasis (Kirmayer & Sartorius, 2007). Among these is the disorder of *koro,* in which there is the belief, accompanied by severe anxiety and sometimes panic, that the genitals are retracting into the abdomen. Most victims of this disorder are Chinese males, although it is also reported in females; there are few reports of the problem in Western cultures. Why does *koro* occur in Chinese cultures? Rubin (1982) points to the central importance of sexual functioning among Chinese males. He notes that typical sufferers are guilty about excessive masturbation, unsatisfactory intercourse, or promiscuity. These kinds of events may predispose men to focus their attention on their sexual organs, which could exacerbate anxiety and emotional arousal, much as it does in the anxiety disorders.

Another culture-specific disorder, prevalent in India, is an anxious concern about losing semen, something that obviously occurs during sexual activity. The disorder, called *dhat,* is associated with a vague mix of physical symptoms, including dizziness, weakness, and fatigue. These low-grade depressive or anxious symptoms are simply attributed to a physical factor, semen loss (Ranjith & Mohan, 2004). Other specific culture-bound somatic symptoms associated with emotional factors would include hot sensations in the head or a sensation of something crawling in the head, specific to African patients (Ebigno, 1986), and a sensation of burning in the hands and feet in Pakistani or Indian patients (Kirmayer & Weiss, 1993).

Medically unexplained physical symptoms may be among the more challenging manifestations of psychopathology.

hypochondriasis Somatoform disorder involving severe anxiety over belief in having a disease process without any evident physical cause.

DSM Disorder Criteria Summary

Hypochondriasis

Features of hypochondriasis include the following:

- Preoccupation with fears of having a serious disease
- Preoccupation persists despite appropriate medical evaluation and reassurance
- Preoccupation is not of delusional intensity and is not restricted to concern over physical appearance
- Clinically significant distress or impairment because of preoccupation
- Duration of at least 6 months

Source: Based on *DSM-IV-TR.* Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision). © 2000 American Psychiatric Association.

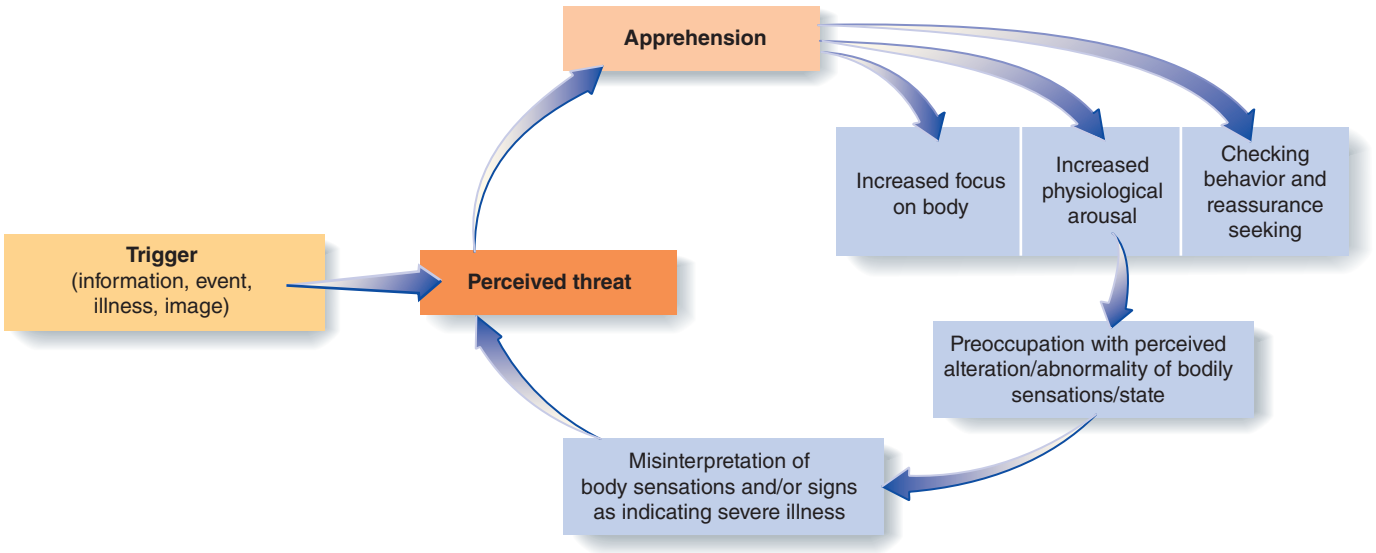
First, a physician must rule out a physical cause for the somatic complaints before referring the patient to a mental health professional. Second, the mental health professional must determine the nature of the somatic complaints to know whether they are associated with a specific somatoform disorder or are part of some other psychopathological syndrome, such as a panic attack. Third, the clinician must be acutely aware of the specific culture or subculture of the patient, which often requires consultation with experts in cross-cultural presentations of psychopathology.

Causes

Investigators with otherwise differing points of view agree on psychopathological processes ongoing in hypochondriasis. Faulty interpretation of physical signs and sensations as evidence of physical illness is central, so almost everyone agrees that hypochondriasis is basically a disorder of cognition or perception with strong emotional contributions (Adler, Côte, Barlow, & Hillhouse, 1994; Barsky & Wyshak, 1990; Kellner, 1985; olde Hartman et al., 2009; Rief et al., 1998; Salkovskis & Clark, 1993; Taylor & Asmundson, 2004, 2009).

Individuals with hypochondriasis experience physical sensations common to all of us, but they quickly focus their attention on these sensations. Remember that the very act of focusing on yourself increases arousal and makes the physical sensations seem more intense than they are (see Chapter 4). If you also tend to misinterpret these as symptoms of illness, your anxiety will increase further. Increased anxiety produces additional physical symptoms and becomes a vicious cycle (■ Figure 5.1) (Salkovskis, Warwick, & Deale, 2003; Warwick & Salkovskis, 1990).

Using procedures from cognitive science such as the Stroop test (see Chapter 2), a number of investigators (Hitchcock & Mathews, 1992; Pauli & Alpers, 2002) have confirmed that participants with hypochondriasis show enhanced perceptual sensitivity to illness cues. They also tend to interpret ambiguous stimuli as threatening (Haenen et al., 2000). Thus, they quickly become aware (and frightened) of any sign of possible illness or disease. A minor headache, for example, might be interpreted as a sure sign of a brain tumor. Smeets, de Jong, and Mayer (2000) demonstrated that individuals with hypochondriasis, compared to "normals," take a "better safe than sorry" approach to dealing with even minor physical symptoms by getting them checked out as soon as possible. More fundamentally, they have a restrictive concept of health as being symptom free (Rief et al., 1998).



■ **Figure 5.1** Integrative model of causes of hypochondriasis. (Based on Warwick, H. M., & Salkovskis, P. M., 1990. Hypochondriasis. Behavior Research Therapy, 28, 105–117.)

What causes individuals to develop this pattern of somatic sensitivity and distorted beliefs? Although it is not certain, the cause is unlikely to be found in isolated biological or psychological factors. There is every reason to believe the fundamental causes of hypochondriasis are similar to those implicated in the anxiety disorders (Barlow, 2002; Suárez et al., 2009). For example, evidence shows that hypochondriasis runs in families (Kellner, 1985) and that there is a modest genetic contribution (Taylor, Thordarson, Jang, & Asmundson, 2006). But this contribution may be nonspecific, such as a tendency to overrespond to stress, and thus may be indistinguishable from the nonspecific genetic contribution to anxiety disorders. Hyperresponsivity might combine with a tendency to view negative life events as unpredictable and uncontrollable and, therefore, to be guarded against at all times (Noyes et al., 2004; Suárez et al., 2009). As we noted in Chapter 4, these factors would constitute biological and psychological vulnerabilities to anxiety.

Why does this anxiety focus on physical sensations and illness? We know that children with hypochondriacal concerns often report the same kinds of symptoms that other family members may have reported at one time (Kellner, 1985; Kirmayer et al., 2003; Pilowsky, 1970). It is therefore quite possible, as in panic disorder, that individuals who develop hypochondriasis have *learned* from family members to focus their anxiety on specific physical conditions and illness.

Three other factors may contribute to this etiological process (Côté et al., 1996; Kellner, 1985). First, hypochondriasis seems to develop in the context of a stressful life event, as do many disorders, including anxiety disorders. Such events often involve death or illness (Noyes et al., 2004; Sandin, Chorot, Santed, & Valiente, 2004). (Gail's traumatic first year of marriage seemed to coincide with the beginning of her disorder.) Second, people who develop hypochondriasis tend to have had a disproportionate incidence of disease in their family when they were children. Thus, even if they did not develop hypochondriasis until adulthood, they carry strong memories of illness that could easily become the focus of anxiety. Third, an important social and interpersonal influence may be operating (Noyes et al., 2003; Suárez et al., 2009). Some people who come from families where illness is a major issue seem to have learned that an ill person often gets a lot of attention. The "benefits" of being sick might contribute to the development of the disorder in some people. A "sick person" who receives increased attention for being ill and is able to avoid work or other responsibilities is described as adopting a "sick role."

Treatment

Unfortunately, relatively little is known about treating hypochondriasis. Clinical reports indicate that reassurance and education seems to be effective in some cases (Haenen et al., 2000; Kellner, 1992)—which is surprising because, by definition, patients with hypochondriasis are not supposed to benefit from reassurance about their health. However, reassurance is usually given only briefly by family doctors who have little time to provide the ongoing support and reassurance that might be necessary. Mental health professionals may be able to offer reassurance in a more effective and sensitive manner, devote sufficient time to all concerns the patient may have, and attend to the "meaning" of the symptoms (for example, their relation to the patient's life stress). Fava, Grandi, Rafanelli, Fabbri, and Cazzaro (2000) tested this idea by assigning 20 patients who met diagnostic criteria for hypochondriasis to two groups. One received "explanatory therapy" in which the clinician went over the source and origins of their symptoms in some detail. These patients were assessed immediately after the therapy and again at a 6-month follow-up. The other group was a wait-list control group that did not receive the explanatory therapy until after their 6 months of waiting. All patients received usual medical care from their physicians. In both groups, taking the time to explain in some detail the nature of the patient's disorder in an educational framework was associated with a significant reduction in hypochondriacal fears and beliefs and a decrease in health-care usage, and these gains were maintained at the follow-up. For the wait-list group, treatment gains did not occur until they received explanatory therapy, suggesting this treatment is effective.

Evaluations of more robust treatments have now appeared (Clark et al., 1998; Kroenke, 2007; Thomson & Page, 2007). For example, in the best study to date, Barsky and Ahern (2005) randomly assigned 187 patients with hypochondriasis to receive either six sessions of cognitive-behavioral treatment (CBT) from trained therapists or treatment as usual from primary care physicians. CBT focused on identifying and challenging illness-related misinterpretations of physical sensations and on showing patients how to create "symptoms" by focusing attention on certain body areas. Bringing on their own symptoms persuaded many patients that such events were under their control. Patients were also coached to seek less reassurance regarding their concerns. CBT was more effective after treatment and at each follow-up point for both symptoms of hypochondriasis and overall changes in functioning and quality of life. But results were still "modest," and many eligible patients refused to enter treatment because they were convinced their problems were medical rather than psychological.

A few recent reports suggest that drugs may help some people with hypochondriasis (Fallon et al., 2003; Kjernisted, Enns, & Lander, 2002; Kroenke, 2007; Taylor et al., 2005). Not surprisingly, these same types of drugs (antidepressants) are useful for anxiety and depression. In one study, CBT and the drug paroxetine (Paxil), a serotonin-specific reuptake inhibitor (SSRI), were both effective, but only CBT was significantly different from a placebo condition. Specifically, 45% in the CBT group, 30% in the Paxil group, and 14% in the placebo group responded to treatment among all patients who entered the study (Greeven et al., 2007).

Somatization Disorder

In 1859, Pierre Briquet, a French physician, described patients who came to see him with seemingly endless lists of somatic complaints for which he could find no medical basis (American Psychiatric Association, 1980). Despite his negative findings, patients returned shortly with either the same complaints or new lists containing slight variations. For many years, this disorder was called *Briquet's syndrome* before being changed in 1980 to **somatization disorder**. Consider the case of Linda.

Linda ♦ Full-Time Patient

Linda, an intelligent woman in her 30s, came to our clinic looking distressed and pained. As she sat down she noted that coming into the office was difficult for her because she had trouble breathing and considerable swelling in the joints of her legs and arms. She was also in some pain from chronic urinary tract infections and might have to leave at any moment to go to the restroom, but she was extremely happy she had kept the appointment. She said she knew we would have to go through a detailed initial interview, but she had something that might save time. At this point, she pulled out several sheets of paper and handed them over. One section, some five pages long, described her contacts with the health-care system for *major difficulties only.* Times, dates, potential diagnoses, and days hospitalized were noted. The second section, one-and-a-half single-spaced pages, consisted of a list of all medications she had taken for various complaints.

Linda felt she had any one of a number of chronic infections that nobody could properly diagnose. She had begun to have these problems in her teenage years. She often discussed her symptoms and fears with doctors and clergy. Drawn to hospitals and medical clinics, she had entered nursing school after high school. However, during hospital training, she noticed her physical condition deteriorating rapidly: She seemed to pick up the diseases she was learning about. A series of stressful emotional events resulted in her leaving nursing school.

After developing unexplained paralysis in her legs, Linda was admitted to a psychiatric hospital, and after a year she regained her ability to walk. On discharge she obtained disability status, which freed her from having to work full time, and she volunteered at the local hospital. With her chronic but fluctuating incapacitation, on some days she could go in and on some days she could not. She was currently seeing a family practitioner and six specialists, who monitored various aspects of her physical condition. She was also seeing two ministers for pastoral counseling.

Clinical Description

Do you notice any differences between Linda, who presented with somatization disorder, and Gail, who presented with hypochondriacal disorder? Linda was more severely impaired and had suffered in the past from symptoms of paralysis. Also, Linda did not seem as *afraid* as Gail that she had a disease. Linda was concerned with the symptoms themselves, not with what they might mean. Individuals with hypochondriasis most often take immediate action on noticing a symptom by calling the doctor or taking medication. People with somatization, however, do not feel the urgency to take action but continually feel weak and ill, and they avoid exercising, thinking it will make them worse (Rief et al., 1998).

Furthermore, Linda's entire life revolved around her symptoms. She once told her therapist that her symptoms were her identity— without them she would not know who she was. By this she meant that she would not know how to relate to people except in the context of discussing her symptoms much as other people might talk about their day at the office or their kids' accomplishments at school. Her few friends who were not health-care professionals had the patience to relate to her sympathetically, through the veil of her symptoms, and she thought of them as friends because they "understood" her suffering. Linda's case is an extreme example of adopting the "sick role" described earlier.

Statistics

Somatization disorder is rare. *DSM-III-R* criteria required 13 or more symptoms from a list of 35, making diagnosis difficult. The criteria were greatly simplified for *DSM-IV,*

DSM Disorder Criteria Summary
Somatization Disorder

Features of somatization disorder include the following:

- A history of many physical complaints beginning before the age of 30, which occur over several years and result in treatment being sought, or significant impairment in important areas of functioning
- Each of the following: (1) four pain symptoms; (2) two gastrointestinal symptoms other than pain (e.g., nausea, diarrhea, bloating); (3) one sexual symptom (e.g., excessive menstrual bleeding, erectile dysfunction); (4) one pseudo-neurological symptom (e.g., double vision, impaired coordination or balance, difficulty swallowing)
- Physical symptoms cannot be fully explained by a known general medical condition or the effects of a substance (for example, a drug of abuse or a medication) *or* where there is a related general medical condition, the physical complaints or impairment are in excess of what would be expected
- Complaints or impairment are not intentionally produced or feigned

Source: Based on *DSM-IV-TR.* Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision). © 2000 American Psychiatric Association.

with only eight symptoms required (Cloninger, 1996). Katon and colleagues (1991) demonstrated that somatization disorder occurs on a continuum: People with only a few medically unexplained physical symptoms may experience sufficient distress and impairment of functioning to be considered to have a disorder that is called *undifferentiated somatoform disorder*. But this disorder is just somatization disorder with fewer than eight symptoms, and for that reason the label is likely to be eliminated in *DSM-5*.Using between four and six symptoms as criteria, Escobar and Canino (1989) found a prevalence of somatization disorder of 4.4% in one large city. The median prevalence in six samples of a large number of patients in a primary care setting meeting these criteria was 16.6% (Creed & Barsky, 2004).

Linda's disorder developed during adolescence, which is the typical age of onset. A number of studies have demonstrated that individuals with somatization disorder tend to be women, unmarried, and from lower socioeconomic groups (see, for example, Creed & Barsky, 2004; Lieb et al., 2002; Swartz, Blazer, George, & Landerman, 1986). For instance, 68% of the patients in a large sample studied by Kirmayer and Robbins (1991) were female. In addition to a variety of somatic complaints, individuals may have psychological complaints, usually anxiety or mood disorders (Adler et al., 1994; Kirmayer & Robbins, 1991; Lieb et al., 2002; Rief et al., 1998). Obviously, individuals with somatization disorder overuse and misuse the health-care system, with medical bills as much as 9 times more than the average patient (Barsky, Orav, & Bates, 2005; Hiller, Fichter, & Rief, 2003; Woolfolk & Allen, in press). In one study, 19% of people with this disorder were on disability (Allen, Woolfolk, Escobar, Gara, & Hamer, 2006). Although symptoms may come and go, somatization disorder and the accompanying sick role behavior are chronic, often continuing into old age.

The rates are relatively uniform around the world for medically unexplained physical symptoms, as is the sex ratio (Gureje, Simon, Ustun, & Goldberg, 1997). When the problem is severe enough to meet criteria for disorder, the sex ratio is approximately 2:1 female to male.

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▲ In somatization disorder, primary relationships are often with medical caregivers; one's symptoms are one's identity.

Causes

Somatization disorder shares some features with hypochondriasis, including a history of family illness or injury during childhood. But this history is a minor factor at best because countless families experience chronic illness or injuries without passing on severe anxiety of being ill or the sick role to children. Something else contributes strongly to somatization disorder.

Given the past difficulty in making a diagnosis, few studies of causes of somatization disorder have been done. Early studies of possible genetic contributions had mixed results. For example, in a sophisticated twin study, Torgersen (1986) found no increased prevalence of somatization disorder in monozygotic (identical) pairs, but most studies find substantial evidence that the disorder runs in families and may have a heritable basis (Bell, 1994; Guze, Cloninger, Martin, & Clayton, 1986; Katon, 1993). A more startling finding emerged from these studies, however. Somatization disorder is strongly linked in family and genetic studies to *antisocial personality disorder (ASPD)* (see Chapter 11), which is characterized by vandalism, persistent lying, theft, irresponsibility with finances and at work, and outright physical aggression. Individuals with ASPD seem insensitive to signals of punishment and to the negative consequences of their often impulsive behavior, and they apparently experience little anxiety or guilt.

ASPD occurs primarily in males and somatization disorder in females, but they share a number of features. Both begin early in life; typically run a chronic course; predominate among lower socioeconomic classes; are difficult to treat; and are associated with marital discord, drug and alcohol abuse, and suicide attempts, among other complications (Cloninger, 1978; Goodwin & Guze, 1984; Lilienfeld, 1992; Mai, 2004). Both family and adoption studies suggest that ASPD and somatization disorder tend to run in families and may well have a heritable component (see, for example, Bohman, Cloninger, von Knorring, & Sigvardsson, 1984; Cadoret, 1978), although it is also possible that the behavioral patterns could be learned in a maladaptive family setting.

Yet, the aggressiveness, impulsiveness, and lack of emotion characteristic of ASPD seem to be at the other end of the spectrum from somatization disorder. What could

somatization disorder Somatoform disorder involving extreme and long-lasting focus on multiple physical symptoms for which no medical cause is evident.

these two disorders possibly have in common? Although we don't yet have the answers, Scott Lilienfeld (1992; Lilienfeld & Hess, 2001) has reviewed a number of hypotheses; although they are speculative, we look at some of them here because they are a fascinating example of integrative biopsychosocial thinking about psychopathology.

One model with some support suggests that somatization disorder and ASPD share a neurobiologically based disinhibition syndrome characterized by impulsive behavior (see, for example, Cloninger, 1987; Gorenstein & Newman, 1980). Evidence indicates that impulsiveness is common in ASPD (see, for example, Newman, Widom, & Nathan, 1985). How does this apply to people with somatization disorder? Many of the behaviors and traits associated with somatization disorder also seem to reflect the impulsive characteristic of short-term gain at the expense of long-term problems. The continual development of new somatic symptoms gains immediate sympathy and attention (for a while) but eventually leads to social isolation (Goodwin & Guze, 1984). One study confirmed that patients with somatization disorder are more impulsive and pleasure seeking than patients with other disorders such as anxiety disorders (Battaglia, Bertella, Bajo, Politi, & Bellodi, 1998).

If individuals with ASPD and somatization disorder share the same underlying neurophysiological vulnerability, why do they behave so differently? The explanation is that social and cultural factors exert a strong effect. Both Cathy Spatz Widom (1984) and Robert Cloninger (1987) have pointed out that the major difference between the disorders is their degree of dependence. Aggression is strongly associated with males in most mammalian species, including rodents (Gray & Buffery, 1971). Dependence and lack of aggression are strongly associated with females. Thus, both aggression and ASPD are strongly associated with males, and dependence and somatization disorder are strongly associated with females. In support of this idea, Lilienfeld and Hess (2001), working with college students, found tendencies for females with antisocial and aggressive traits to report more somatic symptoms. Gender roles are among the strongest components of identity. It is possible that gender socialization accounts almost entirely for the profound differences in the expression of the same biological vulnerability among men and women.

Might these assumptions apply to Linda or her family? Linda's sister had been married briefly and had two children. She had been in therapy for most of her adult life. Occasionally, Linda's sister visited doctors with various somatic complaints, but her primary difficulty was unexplained periods of recurring amnesia that might last several days; these spells alternated with blackout periods during which she was rushed to the hospital.

There were signs of sexual impulsivity and ASPD in Linda and her family. The sister's older daughter, after a stormy adolescence characterized by truancy and delinquency, was sentenced to jail for violations involving drugs and assault. Amid one session with us, Linda noted that she had kept a list of people with whom she had had sexual intercourse. Linda's list numbered well over 20, and most of the sexual episodes occurred in the offices of mental health professionals or clergy!

This development in Linda's relationship with caregivers was important because she saw it as the ultimate sign that the caregivers were concerned about her as a person and she was important to them. But the relationships almost always ended tragically. Several of the caregivers' marriages disintegrated, and at least one mental health professional committed suicide. Linda herself was never satisfied or fulfilled by the relationships but was greatly hurt when they inevitably ended. The American Psychological Association has decreed that it is *always* unethical to have *any* sexual contact with a patient at any time during treatment. Violations of this ethical canon have nearly always had tragic consequences.

Treatment

Somatization disorder is exceedingly difficult to treat. Although there are treatments with proven effectiveness, mostly cognitive-behavioral ones (Woolfolk & Allen, in press), the effectiveness is somewhat lower than for other disorders such as anxiety and mood disorders. In our clinic, we concentrate on initially providing reassurance, reducing stress, and, in particular, reducing the frequency of help-seeking behaviors. One of the most common patterns is the person's tendency to visit numerous medical specialists to address the symptom of the week. There is an extensive medical and physical workup with every visit to a new physician (or to one who has not been seen for a while) at an extraordinary cost to the health-care system (Barsky et al., 2005; Hiller et al., 2003). In treatment, to limit these visits, a gatekeeper physician is assigned each patient to screen all physical complaints. Subsequent visits to specialists must be specifically authorized by this gatekeeper. In the context of a positive therapeutic relationship, most patients are amenable to this arrangement.

Additional therapeutic attention is directed at reducing the supportive consequences of relating to significant others on the basis of physical symptoms alone. More appropriate methods of interacting with others are encouraged, along with additional procedures to promote healthy social and personal adjustment without relying on being "sick." In this context, CBT may then be the most helpful (Allen et al., 2006; Mai, 2004; Woolfolk & Allen, in press). Because Linda, like many patients with this disorder, was receiving disability payments from the state, additional goals involved encouraging at least part-time employment, with the ultimate goal of discontinuing disability.

Pain Disorder

A related somatoform disorder about which little is known is **pain disorder**. Pain disorder refers to pain in one or more sites in the body that is associated with significant distress or impairment. In pain disorder, there may have been clear physical reasons for pain, at least initially, but psychological factors play a major role in maintaining it, particularly anxiety focused on the experience of pain (Asmundson &

Carleton, 2009). In *DSM-5,* the proposal is to make this condition part of a larger category called "complex somatic symptom disorder," as described in the beginning of the chapter. But the clinician could still specify complaints of chronic pain (and associated anxiety) as the principal focus. The three subtypes of pain disorder in *DSM-IV-TR* run the gamut from pain judged to result primarily from psychological factors to pain judged to result primarily from a general medical condition. Several studies suggest that this is a fairly common condition, with 5% to 12% of the population meeting criteria for pain disorder (Asmundson & Carleton, 2009; Frohlich, Jacobi, & Wittchen, 2006; Grabe et al., 2003).

An important feature of pain disorder is that the pain is real and it hurts, regardless of the causes (Aigner & Bach, 1999; King & Strain, 1991). Consider the two cases described here.

The Medical Student ♦ Temporary Pain

During her first clinical rotation, a 25-year-old third-year medical student in excellent health was seen at her student health service for intermittent abdominal pain of several weeks' duration. The student claimed no past history of similar pain. Physical examination revealed no physical problems, but she told the physician that she had recently separated from her husband. The student was referred to the health service psychiatrist. No other psychiatric problems were found. She was taught relaxation techniques and given supportive therapy to help her cope with her current stressful situation. The student's pain subsequently disappeared, and she successfully completed medical school.

DSM Disorder Criteria Summary
Pain Disorder

Features of pain disorder include the following:

- Prevalence of serious pain in one or more anatomical sites
- Pain causes clinically significant distress or impairment in functioning
- Psychological factors are judged to play a primary role in the onset, severity, exacerbation, or maintenance of the pain
- Pain is not feigned or intentionally produced

Source: Based on *DSM-IV-TR.* Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision). © 2000 American Psychiatric Association.

The Woman with Cancer ♦ Managing Pain

A 56-year-old woman with metastatic breast cancer who appeared to be coping appropriately with her disease had severe pain in her right thigh for a month. She initially obtained relief from a combination of drugs and subsequently received hypnotherapy and group therapy. These treatment modalities provided additional pain relief and enabled the patient to decrease her narcotic intake with no increase in pain.

The medical student's pain was seen as purely psychological. In the case of the second woman, the pain was probably related to cancer. But we now know that whatever its cause, pain has a strong psychological component. If medical treatments for existing physical conditions are in place and pain remains, or if the pain seems clearly related to psychological factors, psychological interventions are appropriate. Because of the complexity of pain itself and the variety of narcotics and other medications prescribed for it, multidisciplinary pain clinics are part of most large hospitals. (In Chapter 7, we delve more deeply into types of pain disorders, their causes, and treatment.)

Conversion Disorder

The term *conversion* has been used off and on since the Middle Ages (Mace, 1992) but was popularized by Freud, who believed the anxiety resulting from unconscious conflicts somehow was "converted" into physical symptoms to find expression. This allowed the individual to discharge some anxiety without actually experiencing it.

Clinical Description

Conversion disorders generally have to do with physical malfunctioning, such as paralysis, blindness, or difficulty speaking (aphonia), without any physical or organic pathology to account for the malfunction. Most conversion symptoms suggest that some kind of neurological disease is affecting sensory–motor systems, although conversion symptoms can mimic the full range of physical malfunctioning. For this reason, and because the term "conversion" implies a specific etiology for which there is limited evidence, the proposal for *DSM-5* is to change the name to "functional neurological disorder" (with "functional" referring to a symptom without organic cause) (Stone, LaFrance, Levenson, & Sharpe, 2010).

Conversion disorders provide some of the most intriguing, sometimes astounding, examples of psychopathology. What could possibly account for somebody going blind when all visual processes are normal or experiencing paralysis of the arms or legs when there is no neurological damage? Consider the case of Eloise.

pain disorder Somatoform disorder featuring true pain but for which psychological factors play an important role in onset, severity, or maintenance.
conversion disorder Physical malfunctioning, such as blindness or paralysis, suggesting neurological impairment but with no organic pathology to account for it.

Eloise | Unlearning Walking

Eloise sat on a chair with her legs under her, refusing to put her feet on the floor. Her mother sat close by, ready to assist her if she needed to move or get up. Her mother had made the appointment and, with the help of a friend, had all but carried Eloise into the office. Eloise was a 20-year-old woman of borderline intelligence who was friendly and personable during the initial interview.

Eloise's difficulty walking developed over 5 years. Her right leg had given way and she began falling. Gradually, the condition worsened to the point that 6 months before her admission to the hospital Eloise could move around only by crawling on the floor. Physical examinations revealed no physical problems.

Eloise presented with a classic case of conversion disorder. Although she was not paralyzed, her specific symptoms included weakness in her legs and difficulty keeping her balance, with the result that she fell often.

Eloise lived with her mother, who ran a gift shop in the front of her house in a small rural town. Eloise had been schooled through special education programs until she was about 15; after this, no further programs were available. When Eloise began staying home, her walking began to deteriorate.

In addition to blindness, paralysis, and aphonia, conversion symptoms may include total mutism and the loss of the sense of touch. Some people have seizures, which may be psychological in origin, because no significant electroencephalogram (EEG) changes can be documented. Another relatively common symptom is *globus hystericus,* the sensation of a lump in the throat that makes it difficult to swallow, eat, or sometimes talk (Finkenbine & Miele, 2004).

DSM Disorder Criteria Summary
Conversion Disorder

Features of conversion disorder include the following:

- One or more conditions affecting voluntary motor or sensory function that suggest a neurological or general medical condition
- Psychological factors are judged to be associated with the condition because of preceding conflicts or other stressors
- The condition cannot otherwise be explained by a general medical condition, effects of a substance, or as a culturally sanctioned behavior or experience
- The condition causes clinically significant distress or impairment

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Closely Related Disorders Distinguishing among conversion reactions, real physical disorders, and outright **malingering** (faking) is sometimes difficult. Several factors can help. Conversion symptoms are often precipitated by marked stress. C. V. Ford (1985) noted that the incidence of marked stress preceding a conversion symptom occurred in 52% to 93% of the studied patients. Often this stress takes the form of a physical injury. In one large survey, 324 out of 869 patients (37%) reported prior physical injury (Stone, Carson, Aditya, et al., 2009). Thus, if the clinician cannot identify a stressful event preceding the onset of the conversion symptom, the clinician might more carefully consider the presence of a true physical condition. In addition, although people with conversion symptoms can usually function normally, they seem truly unaware either of this ability or of sensory input. For example, individuals with the conversion symptom of blindness can usually avoid objects in their visual field, but they will tell you they can't see the objects. Similarly, individuals with conversion symptoms of paralysis of the legs might suddenly get up and run in an emergency and then be astounded they were able to do this. It is possible that at least some people who experience miraculous cures during religious ceremonies may have been suffering from conversion reactions. These factors may help in distinguishing between conversion and organically based physical disorders, but clinicians sometimes make mistakes, although it is not common with modern diagnostic techniques. In any case, ruling out medical causes for the symptoms is crucial to making a diagnosis of conversion and, given advances in medical screening procedures, will become the principal diagnostic criterion in *DSM-5* (APA, 2010; Stone et al., 2010).

It can also be difficult to distinguish between individuals who are truly experiencing conversion symptoms in a seemingly involuntary way and malingerers who are good at faking symptoms. Once malingerers are exposed, their motivation is clear: They are either trying to get out of something, such as work or legal difficulties, or they are attempting to gain something, such as a financial settlement. Malingerers are fully aware of what they are doing and are clearly attempting to manipulate others to gain a desired end.

More puzzling is a set of conditions called **factitious disorders**, which fall somewhere between malingering and conversion disorders. The symptoms are under voluntary control, as with malingering, but there is *no obvious reason* for voluntarily producing the symptoms except, possibly, to assume the sick role and receive increased attention. Tragically, this disorder may extend to other members of the family. An adult, almost always a mother, may purposely make her child sick, evidently for the attention and pity given to her as the mother of a sick child. When an individual deliberately makes someone else sick, the condition is called *factitious disorder by proxy* or, sometimes, *Munchausen syndrome by proxy,* but it is really an atypical form of child abuse (Check, 1998). Table 5.1 presents differences between typical child abuse and Munchausen syndrome by proxy.

The offending parent may resort to extreme tactics to create the appearance of illness in the child. For example, one mother stirred a vaginal tampon obtained during men-

Table 5.1 Child Abuse Associated with Munchausen Syndrome by Proxy versus Typical Child Abuse

	Typical Child Abuse	Atypical Child Abuse (Munchausen Syndrome by Proxy)
Physical presentation of the child	Results from direct physical contact with the child; signs often detected on physical examination	Misrepresentation of an acute or accidental medical or surgical illness not usually obvious on physical examination
Obtaining the diagnosis	The perpetrator does not invite the discovery of the manifestation of the abuse	The perpetrator usually presents the manifestations of the abuse to the health-care system
Victims	Children are either the objects of frustration and anger or are receiving undue or inappropriate punishment	Children serve as the vector in gaining the attention the mother desires; anger is not the primary causal factor
Awareness of abuse	Usually present	Not usually present

Source: Reprinted, with permission, from Check, J. R. "Munchausen Syndrome by Proxy: An Atypical Form of Child Abuse." *Journal of Practical Psychiatry and Behavioral Health,* 1998, p. 341, Table 6.2. Copyright © 1998 Lippincott Williams & Wilkins.

struation in her child's urine specimen. Another mother mixed feces into her child's vomit (Check, 1998). Because the mother typically establishes a positive relationship with a medical staff, the true nature of the illness is most often unsuspected and the staff members perceive the parent as remarkably caring, cooperative, and involved in providing for her child's well-being. Therefore, the mother is often successful at eluding suspicion.

Helpful procedures to assess the possibility of Munchausen syndrome by proxy include a trial separation of the mother and the child or video surveillance of the child while in the hospital. An important study has appeared validating the utility of surveillance in hospital rooms of children with suspected Munchausen syndrome by proxy. In this study, 41 patients presenting with chronic, difficult-to-diagnose physical problems were monitored by video during their hospital stay. In 23 of these cases, the diagnoses turned out to be Munchausen syndrome by proxy, where the parent was responsible for the child's symptoms, and in more than half of these 23 cases, video surveillance was the method used to establish the diagnosis. In the other patients, laboratory tests or "catching" the mother in the act of inducing illness in her child confirmed the diagnosis.

DSM Disorder Criteria Summary

Factitious Disorders

Features of factitious disorders include the following:

- Intentional production or feigning of physical or psychological problems
- Behavior motivated by desire to assume the sick role
- Absence of external incentives (such as economic gain, avoiding legal responsibility)

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Unconscious Mental Processes Unconscious cognitive processes seem to play a role in much of psychopathology (although not necessarily as Freud envisioned it), but nowhere is this phenomenon more readily and dramatically apparent than when we attempt to distinguish between conversion disorders and related conditions. Information reviewed in Chapter 2 on unconscious cognitive processes becomes important in this context. We are all capable of receiving and processing information in a number of sensory channels (such as vision and hearing) without being aware of it. Remember the phenomenon of blind sight or unconscious vision? Weiskrantz (1980) and others discovered that people with small, localized damage to certain parts of their brains could identify objects in their field of vision but that they had no awareness whatsoever that they could see. Could this happen to people without brain damage? Consider the case of Celia.

Celia | Seeing through Blindness

A 15-year-old girl named Celia suddenly was unable to see. Shortly thereafter, she regained some of her sight, but her vision was so severely blurred that she could not read. When she was brought to a clinic for testing, psychologists arranged a series of sophisticated vision tests that did not require her to report when she could or could not see. One of the tasks required her to examine three triangles displayed on

malingering Deliberate faking of a physical or psychological disorder motivated by gain.
factitious disorder Nonexistent physical or psychological disorder deliberately faked for no apparent gain except, possibly, sympathy and attention.

three separate screens and to press a button under the screen containing an upright triangle. Celia performed perfectly on this test without being aware that she could see anything (Grosz & Zimmerman, 1970). Was Celia faking? Evidently not, or she would have purposely made a mistake.

Sackeim, Nordlie, and Gur (1979) evaluated the potential difference between real unconscious process and faking by hypnotizing two participants and giving each a suggestion of total blindness. One participant was also told it was extremely important that she appear to everyone to be blind. The second participant was not given further instructions. The first participant, evidently following instructions to appear blind at all costs, performed far below chance on a visual discrimination task similar to the upright triangle task. On almost every trial, she chose the wrong answer. The second participant, with the hypnotic suggestion of blindness but no instructions to "appear" blind at all costs, performed perfectly on the visual discrimination tasks—although she reported she could not see anything.

How is this relevant to identifying malingering? In an earlier case, Grosz and Zimmerman (1965) evaluated a male who seemed to have conversion symptoms of blindness. They discovered that he performed much more poorly than chance on a visual discrimination task. Subsequent information from other sources confirmed that he was almost certainly malingering. To review these distinctions, someone who is truly blind would perform at a chance level on visual discrimination tasks. People with conversion symptoms, however, can see objects in their visual field and therefore would perform well on these tasks, but this experience is dissociated from their awareness of sight. Malingerers and, perhaps, individuals with factitious disorders simply do everything possible to pretend they can't see.

Statistics

We have already seen that conversion disorder may occur with other disorders, particularly somatization disorder, as in the case of Linda. Linda's paralysis passed after several months and did not return, although on occasion she would report "feeling as if" it were returning. Comorbid anxiety and mood disorders are also common (Pehlivanturk & Unal, 2002; Rowe, 2010; Stone, Carson, Duncan, et al., 2009). Conversion disorders are relatively rare in mental health settings, but remember that people who seek help for this condition are more likely to consult neurologists or other specialists. The prevalence estimate in neurological settings is high, averaging about 30% (Allin, Streeruwitz, & Curtis, 2005; Rowe, 2010; Stone, Carson, Duncan, et al., 2009). One study estimated that 10% to 20% of all patients referred to epilepsy centers have psychogenic, nonepileptic seizures (Benbadis & Allen-Hauser, 2000).

Like somatization disorder, conversion disorders are found primarily in women (Deveci et al., 2007; Folks, Ford, & Regan, 1984; Rosenbaum, 2000) and typically develop during adolescence or slightly thereafter. However, they occur relatively often in males at times of extreme stress (Chodoff, 1974). Conversion reactions are not uncommon in soldiers exposed to combat (Mucha & Reinhardt, 1970). The conversion symptoms often disappear after a time, only to return later in the same or similar form when a new stressor occurs. In other cultures, some conversion symptoms are common aspects of religious or healing rituals. Seizures, paralysis, and trances are common in some rural fundamentalist religious groups in the United States (Griffith, English, & Mayfield, 1980), and they are often seen as evidence of contact with God. Individuals who exhibit such symptoms are thus held in high esteem by their peers. These symptoms do not meet criteria for a "disorder" unless they persist and interfere with an individual's functioning.

Causes

Freud described four basic processes in the development of conversion disorder. First, the individual experiences a traumatic event—in Freud's view, an unacceptable, unconscious conflict. Second, because the conflict and the resulting anxiety are unacceptable, the person represses the conflict, making it unconscious. Third, the anxiety continues to increase and threatens to emerge into consciousness, and the person "converts" it into physical symptoms, thereby relieving the pressure of having to deal directly with the conflict. This reduction of anxiety is considered to be the *primary gain* or reinforcing event that maintains the conversion symptom. Fourth, the individual receives greatly increased attention and sympathy from loved ones and may also be allowed to avoid a difficult situation or task. Freud considered such attention or avoidance to be the *secondary gain,* the secondarily reinforcing set of events.

We believe Freud was basically correct on at least three counts but probably not on the fourth, although firm evidence supporting any of these ideas is sparse and Freud's views were far more complex than represented here. Most often, individuals with conversion disorder have experienced a traumatic event that must be escaped at all costs (Stone, Carson, Aditya, et al., 2009). This might be combat, where death is imminent, or an impossible interpersonal situation. Because simply running away is unacceptable in most cases, the socially acceptable alternative of getting sick is substituted; but getting sick on purpose is also unacceptable, so this motivation is detached from the person's consciousness. Finally, because the escape behavior (the conversion symptoms) is successful to an extent in obliterating the traumatic situation, the behavior continues until the underlying problem is resolved. One study confirms these hypotheses, at least partially (Wyllie, Glazer, Benbadis, Kotagal, & Wolgamuth, 1999). In this study, 34 child and adolescent patients, 25 of them girls, were evaluated after receiving a diagnosis of psychologi-

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▲ The seizures and trances that may be symptomatic of conversion disorder are also common in some rural fundamentalist religious groups in the United States.

cally based pseudo-seizures (psychogenic nonepileptic seizures). Many of these children and adolescents presented with additional psychological disorders, including 32% with mood disorders and 24% with separation anxiety and school refusal. Other anxiety disorders were present in some additional patients.

When the extent of psychological stress in the lives of these children was examined, it was found that most of the patients had substantial stress, including a history of sexual abuse, recent parental divorce or death of a close family member, and physical abuse. The authors concluded that major mood disorders and severe environmental stress, especially sexual abuse, are common among children and adolescents with the conversion disorder of pseudo-seizures, as have other studies (Roelofs et al., 2002).

The one step in Freud's progression of events about which some questions remain is the issue of primary gain. The notion of primary gain accounts for the feature of *la belle indifférence* (cited previously), where individuals seem not the least bit distressed about their symptoms. In other words, Freud thought that because symptoms reflected an unconscious attempt to resolve a conflict, the patient would not be upset by them. But formal tests of this feature provide little support for Freud's claim. For example, Stone and colleagues (2006) in the study described earlier on "indifference" to conversion symptoms found no difference in distress over symptoms among patients with conversion disorder compared to patients with organic disease.

Social and cultural influences also contribute to conversion disorder, which, like somatization disorder, tends to occur in less educated, lower socioeconomic groups where knowledge about disease and medical illness is not well developed (Binzer, Andersen, & Kullgren, 1997; Kirmayer, Looper, & Taillefer, 2003; Woolfolk & Allen, in press). For example, Binzer and colleagues (1997) noted that 13% of their group of 30 adult patients with motor disabilities resulting from conversion disorder had attended high school compared to 67% in a control group of patients with motor symptoms because of a physical cause. Prior experience with real physical problems, usually among other family members, tends to influence the later choice of specific conversion symptoms—that is, patients tend to adopt symptoms with which they are familiar (see, for example, Brady & Lind, 1961). Furthermore, the incidence of these disorders has decreased over the decades (Kirmayer et al., 2003). The most likely explanation is that increased knowledge of the real causes of physical problems by both patients and loved ones eliminates much of the possibility of secondary gain so important in these disorders.

Finally, many conversion symptoms seem to be part of a larger constellation of psychopathology. Linda had broad-ranging somatization disorder, as well as the severe conversion symptoms, that resulted in her hospitalization. In similar cases, individuals may have a marked biological vulnerability to develop conversion disorder when under stress, with biological processes like those discussed in the context of somatization disorder. Neuroscientists are increasingly finding a strong connectivity between the conversion symptom and parts of the brain regulating emotion, such as the amygdala, using brain-imaging procedures (Rowe, 2010; Voon et al., 2010).

For countless other cases, however, biological contributory factors seem to be less important than the overriding influence of interpersonal factors (the actions of Eloise's mother, for example), as we will discuss in the next section.

Treatment

Because conversion disorder has much in common with somatization disorder, many of the treatment principles are similar.

A principal strategy in treating conversion disorder is to identify and attend to the traumatic or stressful life event, if it is still present (either in real life or in memory). As in the case of Anna O., therapeutic assistance in reexperiencing or "reliving" the event (catharsis) is a reasonable first step.

The therapist must also work hard to reduce any reinforcing or supportive consequences of the conversion symptoms (secondary gain). For example, it was quite clear that Eloise's mother found it convenient if Eloise stayed in one place most of the day while her mother attended to the store in the front of the house. Eloise's immobility was thus strongly reinforced by motherly attention and concern. Any unnecessary mobility was punished. The therapist must collaborate with both the patient and the family to eliminate such self-defeating behaviors.

Body Dysmorphic Disorder

Some people think they are so ugly they refuse to interact with others or otherwise function normally for fear that people will laugh at their ugliness. This curious affliction is called **body dysmorphic disorder (BDD)**, and at its center is a preoccupation with some imagined defect in appearance by someone who actually looks reasonably normal. The disorder has been referred to as "imagined ugliness" (Phillips, 1991). Consider the case of Jim.

DSM Disorder Criteria Summary
Body Dysmorphic Disorder

Features of body dysmorphic disorder include the following:

- Preoccupation with an imagined defect in appearance, or gross exaggeration of a slight physical anomaly
- Preoccupation causes significant distress or impairment in functioning
- Preoccupation is not better accounted for by another mental disorder (e.g., anorexia nervosa)

Source: Based on *DSM-IV-TR.* Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision). © 2000 American Psychiatric Association.

Jim — Ashamed to Be Seen

In his mid-20s, Jim was diagnosed with suspected social phobia. He had just finished rabbinical school and had been offered a position at a synagogue in a nearby city. However, he found himself unable to accept because of marked social difficulties. Lately he had given up leaving his small apartment for fear of running into people he knew and being forced to stop and interact with them.

Jim was a good-looking young man with dark hair and eyes and of about average height. Although he was somewhat depressed, a mental status exam and a brief interview focusing on current functioning and past history did not reveal any remarkable problems. There was no sign of a psychotic process (he was not out of touch with reality). We then focused on Jim's social difficulties. We expected the usual kinds of anxiety about interacting with people or "doing something" (performing) in front of them. But this was not Jim's concern. Rather, he was convinced that everyone, even his good friends, was staring at a part of his body that he found grotesque. He reported that strangers would never mention his deformity and his friends felt too sorry for him to mention it. Jim thought his head was square! Like the Beast in *Beauty and the Beast* who could not imagine people reacting to him with anything less than revulsion, Jim could not imagine people getting past his square head. To hide his condition as well as he could, Jim wore soft floppy hats and was most comfortable in winter, when he could all but completely cover his head with a large stocking cap. To us, Jim looked normal.

Clinical Description To give you a better idea of the types of concerns people with BDD present to health professionals, the locations of imagined defects in 200 patients are shown in Table 5.2. The average number of body areas of concern to these individuals was five to seven (Phillips, Menard, Fay, & Weisberg, 2005). A variety of checking or compensating rituals are common in people with BDD in attempts to alleviate their concerns. For example, excessive tanning is common, with 25% of one group of 200 patients tanning themselves in an attempt to hide skin defects (Phillips, Menard, Fay, & Weisberg, 2005). Many people with this disorder become fixated on mirrors (Veale & Riley, 2001). They often check their presumed ugly feature to see whether any change has taken place. Others avoid mirrors to an almost phobic extent. Quite understandably, suicidal ideation, suicide attempts, and suicide itself are typical consequences of this disorder (Phillips, Menard, Fay, & Weisberg, 2005; Zimmerman & Mattia, 1998). People with BDD also have "ideas of reference," which means they think everything that goes on in their world somehow is related to them—in this case, to their imagined defect. This disorder can cause considerable disruption in the patient's life. Many patients with severe cases become housebound for fear of showing themselves to other people.

Table 5.2 Location of Imagined Defects in 200 Patients with Body Dysmorphic Disorder*

Location	%	Location	%
Skin	80	Overall appearance of face	19
Hair	58	Small body build	18
Nose	39	Legs	18
Stomach	32	Face size or shape	16
Teeth	30	Chin	15
Weight	29	Lips	14.5
Breasts	26	Arms or wrists	14
Buttocks	22	Hips	13
Eyes	22	Cheeks	11
Thighs	20	Ears	11
Eyebrows	20		

Adapted from Phillips, K. A., Menard, B. A., Fay, C., & Weisberg, R., (2005). Demographic characteristics, phenomenology, comorbidity, and family history in 200 individuals with body dysmorphic disorder. *Psychosomatics, 46*(4), 317–325. © 2005 The Academy of Psychosomatic Medicine.

If this disorder seems strange to you, you are not alone. For decades, this condition, previously known as *dysmorphophobia* (literally, fear of ugliness), was thought to represent a psychotic delusional state because the affected individuals were unable to realize, even for a fleeting moment, that their ideas were irrational. Whether this is true is still debated.

Abnormal Psychology Inside Out, produced by Ira Wohl, Only Child Motion Pictures

Body Dysmorphic Disorder: Doug

"I didn't want to talk to anybody. . . . I was afraid because what I saw on my face . . . they saw. . . . If I could see it, they could see it. And I thought there was like an arrow pointing at it. And I was very self-conscious. And I felt like the only time I felt comfortable was at night because it was dark time.

Go to Psychology CourseMate at www.cengagebrain.com to watch this video.

For example, in 200 cases examined by Phillips, Menard, Fay, and Weisberg (2005) and in 50 cases reported by Veale, Boocock, and colleagues (1996), between 33% and 50% of participants were convinced their imagined bodily defect was real and a reasonable source of concern. Is this delusional? The *DSM-IV* task force wrestled long and hard with this issue and decided that individuals with BDD whose beliefs are so firmly held that they could be called delusional should receive a second diagnosis of delusional disorder, somatic type (see Chapter 12) in addition to BDD. Phillips, Menard, Pagano, Fay, and Stout (2006) looked closely at differences that may exist between delusional and nondelusional types and found nothing significant, beyond the fact that the delusional type was more severe and found in less educated patients. Other studies have supported this lack of meaningful differences between these two groups (Mancuso, Knoesen, & Castle, in press; Phillips et al., 2010). It is also the case that these two groups both respond equally well to treatments for BDD and that the "delusional" group does not respond to drug treatments for psychotic disorders (Phillips et al., in press). Thus, in *DSM-5,* the proposal is that patients would receive just a BDD diagnosis, whether they are "delusional" or not, and the practice of giving them a second diagnosis of delusional disorder (a psychotic disorder) should be dropped (Phillips et al., in press).

Statistics

The prevalence of BDD is hard to estimate because by its very nature it tends to be kept secret. However, the best estimates are that it is far more common than we had previously thought. Without some sort of treatment, it tends to run a lifelong course (Phillips, 1991; Veale, Boocock, et al., 1996). One of the patients with BDD reported by Phillips and colleagues (1993) had suffered from her condition for 71 years, since the age of 9. If you think a college friend seems to have at least a mild version of BDD, you're probably correct. Studies suggest that as many as 70% of college students report at least some dissatisfaction with their bodies, with 4% to 28% of these appearing to meet all the criteria for the disorder (Fitts, Gibson, Redding, & Deiter, 1989; Phillips, 2005). However, this study was done by questionnaire and may well have reflected the large percentage of students who are concerned simply with weight.

Another study investigated the prevalence of BDD specifically in an ethnically diverse sample of 566 adolescents between the ages of 14 and 19. The overall prevalence of BDD in this group was 2.2%, with adolescent girls more dissatisfied with their bodies than boys and African Americans of both genders less dissatisfied with their bodies than Caucasians, Asians, and Hispanics (Mayville, Katz, Gipson, & Cabral, 1999; Roberts, Cash, Feingold, & Johnson, 2006). Overall, about 1% to 2% of individuals in community samples and from 2% to 13% of student samples meet criteria for BDD (Koran, Abujaoude, Large, & Serpe, 2008; Phillips, Menard, Fay, & Weisberg, 2005; Woolfolk & Allen, in press). A somewhat higher proportion of individuals with BDD are interested in art or design compared to individuals without BDD, reflecting, perhaps, a strong interest in aesthetics or appearance (Veale, Ennis, & Lambrou, 2002).

In mental health clinics, the disorder is also uncommon because most people with BDD seek other types of health professionals, such as plastic surgeons and dermatologists. BDD is seen equally in men and women. In the larger series of 200 individuals reported by Phillips, Menard, Fay, and Weisberg (2005), 68.5% were female, but 62% of a large number of individuals with BDD in Japan were males. Generally, there are more similarities than differences between men and women with BDD, but some differences have been noted (Phillips, Menard, & Fay, 2006). Men tend to focus on body build, genitals, and thinning hair and tend to be more severe. A focus on muscle defects and body building is nearly unique to men with the disorder (Pope et al., 2005). Women focus on more varied body areas and are more likely to also have an eating disorder.

As you might suspect, few people with this disorder get married. Age of onset ranges from early adolescence through the 20s, peaking at the age of 16 or 17 (Phillips, Menard, Fay, & Weisberg, 2005; Veale, Boocock, et al., 1996; Zimmerman & Mattia, 1998). Individuals are somewhat reluctant to seek treatment. In many cases, a relative will force the issue, demanding the individual get help; this insistence may reflect the disruptiveness of the disorder for family members. One study of 62 consecutive outpatients

body dysmorphic disorder (BDD) Somatoform disorder featuring a disruptive preoccupation with some imagined defect in appearance ("imagined ugliness").

Discussing Diversity

The Cost of Beauty

What is beauty? How would you describe the perfect body? Your perception of beauty and body image has no doubt been influenced by the social and cultural environment in which you have grown up. Such perceptions vary widely across different cultures and can often lead to behaviors aimed at enhancing one's beauty that may seem strange or even pathological to those from a different culture. For instance, having freckles or a birthmark on one's face is considered by many to be an attractive trait in the United States (such as among popular models and actresses like Cindy Crawford and Lindsay Lohan). However, over the centuries, freckles generally have not been very popular, and in many cultures chemical solutions were used to remove them, sometimes leading to significant damage to layers of one's skin (Liggett, 1974).

In many other cultures, devices are worn during infancy, childhood, or even adulthood that are intended to reshape a person's bone structure—such as by reshaping the head or nose of newborns in order to make them more attractive (e.g., Fallon, 1990; Liggett, 1974). Perhaps one of the best known examples of such a practice is the binding of girls' feet in China in order to prevent them from growing. This binding, which occurred from the 10th to the 20th century, forced women to walk in a way thought seductive by men in that culture. Although this may seem to some to be a foreign concept, can you think of anything similar done in the U.S. culture in an attempt to enhance one's beauty? Although less extreme than foot binding, many women in the United States wear tight-fitting shoes with high heels and have bags of chemicals implanted under their skin to enhance the size of their breasts or even buttocks (some celebrities in the United States voluntarily choose to undergo multiple cosmetic surgeries at one time; reality television star Heidi Montag reportedly had 10 such procedures performed in one day)—all in an effort to appear more attractive. Of course, men also do things to enhance their appearance, such as wearing hair-pieces or getting hair plugs and removing fat via liposuction. Men and women alike will go as far as to knowingly risk their health for what they believe will be increased beauty. In fact, roughly 20% of adults aged 18 to 29 years in the United States reported using indoor tanning in the past year (Heckman, Coups, & Manne, 2008), essentially accepting the health risks (i.e., melanoma and squamos cell cancer) associated with tanning lamps in exchange for darker skin; indoor tanning is widely available in the United States (Hoerster et al., 2009). Such behavior leads one to question the costs associated with beauty. More extreme examples of body modification also exist in the United States, such as skin-piercing and tattooing, which have been quite rare historically in this country but have increased significantly over the past several decades (Laumann & Derick, 2006). All of these practices are quite commonplace in our culture, but each may seem strange to those in other parts of the world.

with BDD found that the degree of psychological stress, quality of life, and impairment were generally worse than comparable indices in patients with depression, diabetes, or a recent myocardial infarction (heart attack) on several questionnaire measures (Phillips, Dufresne, Wilkel, & Vittorio, 2000). Similar results were reported on a larger sample of 176 patients (Phillips, Menard, Fay, & Pagano, 2005). Thus, BDD is among the more serious of psychological disorders, and depression and substance abuse are common consequences of BDD (Gustad & Phillips, 2003; Phillips et al., in press).

Further reflecting the intense suffering that accompanies this disorder, Veale (2000) collected information on 25 patients with BDD who had sought cosmetic surgery in the past. Of these, nine patients who could not afford surgery, or were turned down for other reasons, had attempted by their own hand to alter their appearance dramatically, often with tragic results. One example was a man preoccupied by his skin, who believed it was too "loose." He used a staple gun on both sides of his face to try to keep his skin taut. The staples fell out after 10 minutes and he narrowly missed damaging his facial nerve. In a second example, a woman was preoccupied by her skin and the shape of her face. She filed down her teeth to alter the appearance of her jawline. Yet another woman who was preoccupied by what she perceived as the ugliness of multiple areas of her body and desired liposuction, but could not afford it, used a knife to cut her thighs and attempted to squeeze out the fat. BDD is also stubbornly chronic. In a recent prospective study of 183 patients, only 21% were somewhat improved over the course of a year, and 15% of that group relapsed during that year (Phillips, Pagano, Menard, & Stout, 2006).

Individuals with BDD react to what they think is a horrible or grotesque feature. Thus, the psychopathology lies in their reacting to a "deformity" that others cannot perceive. Social and cultural determinants of beauty and body image largely define what is "deformed." (Nowhere is this more evident than in the greatly varying cultural standards for body weight and shape, factors that play a major role in eating disorders, as you will see in Chapter 8.)

For example, concerns with the width of the face, so common in BDD, can be culturally determined. Until recently, in some areas of France, Africa, Greenland, and Peru, the head of a newborn infant was reshaped, either by hand or by tight caps secured by strings. Sometimes the face was elongated; other times it was widened. Similarly, attempts were made to flatten the noses of newborn infants, usually by hand (Fallon, 1990; Liggett, 1974).

Also, many are aware of the old practice in China of binding girls' feet, often preventing the foot from growing to more than one-third of its normal size. Women's bound feet forced them to walk in a way that was thought seductive, as mentioned earlier. As Brownmiller (1984) points out, the myth that an unnaturally small foot signifies extraordinary beauty and grace is still with us. Can you think of the fairy tale where a small foot becomes the identifying feature of the beautiful heroine?

What can we learn about BDD from such practices? The behavior of individuals with BDD seems remarkably strange because they go *against* current cultural practices that put less emphasis on altering facial features. In other words, people who simply conform to the expectations of their culture do not have a disorder (as noted in Chapter 1). Nevertheless, aesthetic plastic surgery, particularly for the nose and lips, is still widely accepted and, because it is most often undertaken by the wealthy, carries an aura of elevated status. In this light, BDD may not be so strange. As with most psychopathology, its characteristic attitudes and behavior may simply be an exaggeration of normal culturally sanctioned behavior.

Causes and Treatment

We know little about either the etiology or the treatment of BDD. What little evidence we do have on etiology comes from a weak source: the pattern of comorbidity of BDD with other disorders. BDD is a somatoform disorder because its central feature is a psychological preoccupation with somatic (physical) issues. For example, in hypochondriasis the focus is on physical sensations, and in BDD the focus is on physical appearance. We have already seen that many of the somatoform disorders tend to co-occur. Linda presented with somatization disorder but also had a history of conversion disorder. However, BDD does not tend to co-occur with the other somatoform disorders, nor does it occur in family members of patients with other somatoform disorders.

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▲ In various cultures, a child's head or face is manipulated to produce desirable features, as in the addition of rings to lengthen the neck of this Burmese girl.

A disorder that does often co-occur with BDD and is found among other family members is obsessive-compulsive disorder (OCD) (Chosak et al., 2008; Gustad & Phillips, 2003; Phillips et al., in press; Phillips & Stout, 2006; Tynes, White, & Steketee, 1990; Zimmerman & Mattia, 1998). Is BDD a variant of OCD? There are a lot of similarities. People with BDD complain of persistent, intrusive, and horrible thoughts about their appearance, and they engage in such compulsive behaviors as repeatedly looking in mirrors to check their physical features. BDD and OCD also have approximately the same age of onset and run the same course. One recent brain-imaging study demonstrated similar abnormal brain functioning between patients with BDD and patients with OCD (Rauch et al., 2003).

Perhaps most significantly, there are two, and only two, treatments for BDD with any evidence of effectiveness. First, drugs that block the reuptake of serotonin, such as clomipramine (Anafranil) and fluvoxamine (Luvox), provide relief to at least some people (Hadley, Kim, Priday, & Hollander, 2006). One controlled study of the effects of drugs on BDD demonstrated that clomipramine was significantly more effective than desipramine, a drug that does not specifically block reuptake of serotonin, for the treatment of BDD, even BDD of the delusional type (Hollander et al., 1999). A second controlled study reported similar findings for fluoxetine (Prozac), with 53% showing a good response compared to 18% on placebo after 3 months (Phillips, Albertini, & Rasmussen, 2002). Intriguingly, these are the same drugs that have the strongest effect in OCD.

Second, exposure and response prevention, the type of cognitive-behavioral therapy effective with OCD, has also been successful with BDD (McKay et al., 1997; Rosen, Reiter, & Orosan, 1995; Veale, Gournay, et al., 1996; Wilhelm, Otto, Lohr, & Deckersbach, 1999). In the Rosen and colleagues (1995) study, 82% of patients treated with this approach responded, although these patients may have been somewhat less severely affected by the disorder than in other studies (Wilhelm et al., 1999; Williams, Hadjistavropoulos, & Sharpe, 2006). Another interesting lead on causes of BDD comes from cross-cultural explorations of similar disorders. You may remember the Japanese variant of social phobia, *taijin kyofusho* (see Chapter 4), in which individuals may believe they have horrendous bad breath or body odor and thus avoid social interaction. But people with *taijin kyofusho* also have all the other characteristics of social phobia. Patients who would be diagnosed with BDD in our culture might simply be considered to have severe social phobia in Japan and Korea. Possibly, then, social anxiety is fundamentally related to BDD, a connection that would give us further hints on the nature of the disorder. Studies of comorbidity indicate that social phobia, along with OCD, is also commonly found in people with BDD (Phillips & Stout, 2006). For all of these reasons, a major proposal for *DSM-5* is to include BDD with the anxiety disorders, perhaps as a variant of OCD (Phillips et al., in press).

Plastic Surgery and Other Medical Treatments Patients with BDD believe they are physically deformed in some way and go to medical doctors to attempt to correct their deficits (Woolfolk & Allen, in press). Phillips, Grant, Siniscalchi, and Albertini (2001) studied the treatments sought by 289 patients with BDD, including 39 children or adolescents, and found that fully 76.4% had sought this type of treatment and 66% were receiving it. Dermatology (skin) treatment was the most often received (45.2%), followed by plastic surgery (23.2%). Looking at it another way, in one study of 268 patients seeking care from a dermatologist, 11.9% met criteria for BDD (Phillips, Dufresne, Wilkel, & Vittorio, 2000).

Because the concerns of people with BDD involve mostly the face or head, it is not surprising that the disorder is big business for the plastic surgery profession—but it's bad business. These patients do not benefit from surgery and may return for additional surgery or, on occasion, file malpractice lawsuits. Investigators estimate that as many as 8% to 25% of all patients who request plastic surgery may have BDD (Barnard, 2000; Crerand et al., 2004). The most common procedures are rhinoplasties (nose jobs), face-lifts, eyebrow elevations, liposuction, breast augmentation, and surgery to alter the jawline. Between 2000 and 2009, according to the American Society of Plastic Surgeons, the total number of cosmetic procedures increased 69%. In 2009, there were 203,000 eyelid surgeries and 289,000 breast enlargement surgeries. The problem is that surgery on the proportion of these people with BDD seldom produces the desired results. These individuals return for additional surgery on the same defect or concentrate on some new defect. Phillips, Menard, Fay, and Pagano (2005) report that 81% of 50 individuals seeking surgery or similar medical consults were dissatisfied with the result. In 88% of a large group of people with BDD seeking medical rather than psychological treatment, the severity of the disorder and accompanying distress either did not change or *increased* after surgery. Similar discouraging or negative results are evident from other forms of medical treatment, such as skin treatments (Phillips et al., 2001). It is important that plastic surgeons screen out these patients; many do so by collaborating with medically trained psychologists (Pruzinsky, 1988).

▲ The world-famous pop singer Michael Jackson as a child and as an adult. Many people alter their features through surgery. However, people with body dysmorphic disorder are seldom satisfied with the results.

Concept Check 5.1

Diagnose the somatoform disorders described here by choosing one of the following: (a) pain disorder, (b) hypochondriasis, (c) somatization disorder, (d) conversion disorder, and (e) body dysmorphic disorder.

1. Emily constantly worries about her health. She has been to numerous doctors for her concerns about cancer and other serious diseases, only to be reassured of her well-being. Emily's anxiousness is exacerbated by each small ailment (for example, headaches or stomach pains) that she considers to be indications of a major illness. _____
2. D. J. arrived at Dr. Blake's office with a folder crammed full of medical records, symptom documentation, and lists of prescribed treatments and drugs. Several doctors are monitoring him for his complaints, ranging from chest pain to difficulty swallowing. D. J. recently lost his job because he was using too many sick days. _____
3. Sixteen-year-old Chad suddenly lost the use of his arms with no medical cause. The complete paralysis slowly improved to the point that he could slightly raise them. However, Chad cannot drive, pick up objects, or perform most tasks necessary for day-to-day life. _____
4. Loretta is 32 and has been preoccupied with the size and shape of her nose for 2 years. She has been saving money for plastic surgery, after which, she is sure, her career will improve. The trouble is that three honest plastic surgeons have told her that her nose is fine as it is. _____
5. Betty had considerable pain when she broke her arm. A year after it healed and all medical tests indicate her arm is fine she still complains of the pain. It seems to intensify when she fights with her husband. _____

Dissociative Disorders

- What are the five types of dissociative disorders?
- What factors influence the etiology and treatment of dissociative disorders?

At the beginning of the chapter, we said that when individuals feel detached from themselves or their surroundings, almost as if they are dreaming or living in slow motion, they are having dissociative experiences. Morton Prince, the founder of the *Journal of Abnormal Psychology,* noted more than 100 years ago that many people experience something like dissociation occasionally (Prince, 1906–1907). It might be likely to happen after an extremely stressful event, such as an accident (Spiegel, 2010). It also is more likely to happen when you're tired or sleep deprived from staying up all night cramming for an exam (Giesbrecht, Smeets, Leppink, Jelicic, & Merckelbach, 2007). Perhaps because you knew the cause, the dissociation may not have bothered you much (Barlow, 2002; Noyes, Hoenk, Kuperman, & Slymen, 1977). However, it may have been extremely frightening. Transient experiences of dissociation will occur in about half of the general population at some point in their lives, and studies suggest that if a person experiences a traumatic event, between 31% and 66% will have this feeling at that time (Hunter, Sierra, & David, 2004; Keane, Marx, Sloan, & DePrince, 2011). Because it's hard to measure dissociation, the connection between trauma and dissociation is controversial (Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008).

These kinds of experiences can be divided into two types. During an episode of *depersonalization,* your perception alters so that you temporarily lose the sense of your own reality, as if you were in a dream and you were watching yourself. During an episode of **derealization**, your sense of the reality of the external world is lost. Things may seem to change shape or size; people may seem dead or mechanical. These sensations of unreality are characteristic of the dissociative disorders because, in a sense, they are a psychological mechanism whereby one "dissociates" from reality. Depersonalization is often part of a serious set of conditions in which reality, experience, and even identity seem to disintegrate.

As we go about our day-to-day lives, we ordinarily have an excellent sense of who we are and a general knowledge of the identity of other people. We are also aware of events around us, of where we are, and of why we are there. Finally, except for occasional small lapses, our memories remain intact so that events leading up to the current moment are clear in our minds. But what happens if we can't remember why we are in a certain place or even who we are? What happens if we lose our sense that our surroundings are real? Finally, what happens if we not only forget who we are, but also begin thinking we are somebody else—somebody who has a different personality, different memories, and even different physical reactions, such as allergies we never had? These are examples of disintegrated experience (Cardeña & Gleaves, 2003; Dell & O'Neil, 2009; Spiegel & Cardeña, 1991; Spiegel, in press; van der Hart & Nijenhuis, 2009). In each case, there are alterations in our relationship to the self, to the world, or to memory processes.

Although we have much to learn about these disorders, we briefly describe four of them—depersonalization disorder, dissociative amnesia, dissociative fugue, and dissociative trance disorder—before examining the fascinating condition of dissociative identity disorder. As you will see, the influence of social and cultural factors is strong in dissociative disorders. Even in severe cases, the expression of the pathology does not stray far from socially and culturally sanctioned forms (Giesbrecht et al., 2008; Kihlstrom, 2005).

Depersonalization Disorder

When feelings of unreality are so severe and frightening that they dominate an individual's life and prevent normal functioning, clinicians may diagnose the rare **depersonalization disorder**. Consider the case of Bonnie.

Bonnie | Dancing Away from Herself

Bonnie, a dance teacher in her late 20s, was accompanied by her husband when she first visited the clinic and complained of "flipping out." When asked what she meant, she said, "It's the most scary thing in the world. It often happens when I'm teaching my modern dance class. I'll be up in front and I will feel focused on. Then, as I'm demonstrating the steps, I just feel like it's not really me and that I don't really have control of my legs. Sometimes I feel like I'm standing in back of myself just watching. Also I get tunnel vision. It seems like I can only see in a narrow space right in front of me and I just get totally separated from what's going on around me. Then I begin to panic and perspire and shake." It turns out that Bonnie's problems began after she smoked marijuana for the first time about 10 years before. She had the same feeling then and found it scary, but with the help of friends she got through it. Lately the feeling recurred more often and more severely, particularly when she was teaching dance class.

derealization Situation in which the individual loses a sense of the reality of the external world.
depersonalization disorder Dissociative disorder in which feelings of depersonalization are so severe they dominate the individual's life and prevent normal functioning.

You may remember from Chapter 4 that during an intense panic attack many people (approximately 50%) experience feelings of unreality. People undergoing intense stress or experiencing a traumatic event may also experience these symptoms, which characterize the newly defined *acute stress disorder.* Feelings of depersonalization and derealization are part of several disorders (Boon & Draijer, 1991; Giesbrecht et al., 2008). But when severe depersonalization and derealization are the primary problem, the individual meets criteria for depersonalization disorder (APA, 2010; Steinberg, 1991). Surveys suggest that this disorder exists in approximately 0.8% of the population (Johnson, Cohen, Kasen, & Brook, 2006). Simeon, Knutelska, Nelson, & Guralnik (2003) described 117 cases approximately equally split between men and women. Mean age of onset was 16 years, and the course tended to be chronic. All patients were substantially impaired. Anxiety, mood, and personality disorders are also commonly found in these individuals (Simeon et al., 2003; Johnson et al., 2006). Among the 117 patients described, 73% suffered from additional mood disorders and 64% from anxiety disorders at some point in their lives.

Two studies (Guralnik, Schmeidler, & Simeon, 2000; Guralnik, Giesbrecht, Knutelska, Sirroff, & Simeon, 2007) have compared patients with depersonalization disorder to matched normal-comparison participants on a comprehensive neuropsychological test battery that assessed cognitive function. Although both groups were of equal intelligence, the participants with depersonalization disorder showed a distinct cognitive profile, reflecting some specific cognitive deficits on measures of attention, processing of information, short-term memory, and spatial reasoning. Basically, these patients were easily distracted and were slow to perceive and process new information. It is not clear how these cognitive and perceptual deficits develop, but they seem to correspond with reports of "tunnel vision" (perceptual distortions) and "mind emptiness" (difficulty absorbing new information) that characterize these patients.

Specific aspects of brain functioning are also associated with depersonalization (see, for example, Sierra & Berrios, 1998; Simeon, 2009; Simeon et al., 2000). Brain-imaging studies confirm deficits in perception (Simeon, 2009; Simeon et al., 2000) and emotion regulation (Phillips et al., 2001). Other studies note dysregulation in the hypothalamic–pituitary–adrenocortical (HPA) axis among these patients, compared to normal controls (Simeon, Guralnik, Knutelska, Hollander, & Schmeidler, 2001), suggesting, again, deficits in emotional responding.

DSM Disorder Criteria Summary
Depersonalization Disorder

Features of depersonalization disorder include the following:

- Persistent or recurrent feelings of being detached from one's body or mental processes (e.g., feeling like one is in a dream)
- Reality testing remains intact during the depersonalization experience
- Depersonalization causes clinically significant distress or impairment in functioning
- The condition does not occur exclusively as part of another mental disorder, such as schizophrenia, panic disorder, or acute stress disorder

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Dissociative Amnesia

Perhaps the easiest to understand of the severe dissociative disorders is one called **dissociative amnesia**, which includes several patterns. People who are unable to remember anything, including who they are, are said to suffer from **generalized amnesia**. Generalized amnesia may be lifelong or may extend from a period in the more recent past, such as 6 months or a year previously. Consider the case study described here.

The Woman Who Lost Her Memory

Several years ago a woman in her early 50s brought her daughter to one of our clinics because of the girl's refusal to attend school and other severely disruptive behavior. The father, who refused to come to the session, was quarrelsome, a heavy drinker, and abusive on occasion. The girl's brother, now in his mid-20s, lived at home and was a burden on the family. Several times a week a major battle erupted, complete with shouting, pushing, and shoving, as each member of the family blamed the others for all their problems.

The mother, a strong woman, was clearly the peacemaker responsible for holding the family together. Approximately every 6 months, usually after a family battle, the mother lost her memory and the family had her admitted to the hospital. After a few days away from the turmoil, the mother regained her memory and went home, only to repeat the cycle in the coming months. Although we did not treat this family (they lived too far away), the situation resolved itself when the children moved away and the stress decreased.

Far more common than general amnesia is **localized or selective amnesia**, a failure to recall specific events, usually traumatic, that occur during a specific period. Dissociative amnesia is common during war (Cardeña & Gleaves, 2003; Loewenstein, 1991; Spiegel & Cardeña, 1991). Sackeim and Devanand (1991) describe the interesting case of a woman whose father had deserted her when she was young. She had also been forced to have an abortion at the age of 14. Years later, she came for treatment for frequent headaches. In therapy she reported early events (for example, the abortion) matter-of-factly, but under hypnosis she would relive, with intense emotion, the early abortion and remember that

subsequently she was raped by the abortionist. She also had images of her father attending a funeral for her aunt, one of the few times she ever saw him. Upon awakening from the hypnotic state, she had no memory of emotionally reexperiencing these events, and she wondered why she had been crying. In this case, the woman did not have amnesia for the *events themselves* but rather for her intense *emotional reactions to the events*. Absence of the subjective experience of emotion that is often present in depersonalization disorder and confirmed by brain-imaging studies (Phillips et al., 2001) becomes prominent here. In most cases of dissociative amnesia, the forgetting is selective for traumatic events or memories rather than generalized.

DSM Disorder Criteria Summary

Dissociative Amnesia

Features of dissociative amnesia include the following:

- One or more episodes of inability to recall important personal information, usually of a traumatic or stressful nature, that is too extensive to be explained by ordinary forgetfulness
- Episodes are not related to a medical condition, physiological effects of a substance (e.g., a drug of abuse), or a separate psychological disorder
- Inability to recall causes clinically significant distress or impairment in functioning

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Dissociative Fugue

A closely related disorder that will almost certainly become a subtype of dissociative amnesia in *DSM-5* (APA, 2010; Ross, 2009) is referred to as **dissociative fugue**, with *fugue* literally meaning "flight" (*fugitive* is from the same root). In these curious cases, memory loss revolves around a specific incident—an unexpected trip (or trips). In most cases, individuals just take off and later find themselves in a new place, unable to remember why or how they got there. Usually they have left behind an intolerable situation. During these trips, a person sometimes assumes a new identity or at least becomes confused about the old identity. Consider the case of Jeffrey Ingram, a 40-year-old male from Washington state, who found himself unexpectedly in Denver.

Jeffrey | A Troubled Trip

An amnesia sufferer who had been searching for his identity for more than a month was back in Washington state with his fiancée on Tuesday, but he still doesn't remember his past life or what happened, his mother said.

Jeffrey Alan Ingram, 40, was diagnosed in Denver with dissociative fugue, a type of amnesia.

He has had similar bouts of amnesia in the past, likely triggered by stress, once disappearing for 9 months. When he went missing this time, on September 6, he had been on his way to Canada to visit a friend who was dying of cancer, said his fiancée, Penny Hansen.

"I think that the stress, the sadness, the grief of facing a best friend dying was enough, and leaving me was enough to send him into an amnesia state," Hansen told KCNC-TV.

When Ingram found himself in Denver on September 10, he didn't know who he was. He said he walked around for about 6 hours asking people for help, then ended up at a hospital, where police spokeswoman Virginia Quinones said Ingram was diagnosed with a type of amnesia known as dissociative fugue.

Searched for his identity. Ingram's identity came to light last weekend after he appeared on several news shows asking the public for help: "If anybody recognizes me, knows who I am, please let somebody know."

"Penny's brother called her right away and told her 'Did you watch this newscast?' and 'I think that's Jeff that they're showing on television,'" said Marilyn Meehan, a spokeswoman for Hansen.

Hansen had filed a missing person report after Ingram failed to show up at her mother's home in Bellingham, Washington, on his way to Canada, but officials searching for him had turned up nothing.

On Monday night, two Denver police detectives accompanied Ingram on a flight to Seattle, where he was reunited with his fiancée.

His mother, Doreen Tompkins of Slave Lake, Alberta, was in tears as she talked about the struggle her son and the family still face.

"It's going to be very difficult again, but you know what, I can do it," she told CTV news of Edmonton, Alberta. "I did it before, I can do it again. I'll do it as many times as I have to just so I can have my son."

Memory never fully regained. Ingram had experienced an episode of amnesia in 1995 when he disappeared during a trip to a grocery store. Nine months later, he was found in a Seattle hospital, according to Thurston County, Washington, officials. His mother said he never fully regained his memory.

dissociative amnesia Dissociative disorder featuring the inability to recall personal information; usually of a stressful or traumatic nature.
generalized amnesia Loss of memory of all personal information, including identity.
localized or selective amnesia Memory loss limited to specific times and events, particularly traumatic events.
dissociative fugue Dissociative disorder featuring sudden, unexpected travel away from home, along with an inability to recall the past, sometimes with assumption of a new identity.

Meehan, who works with Hansen at the state Utilities and Transportation Commission, said the couple would not give interviews because they want to concentrate on Ingram's effort to regain his memory.

"They're taking it one step at a time," Meehan said.

"He said that while her face wasn't familiar to him, her heart was familiar to him," she said. "He can't remember his home, but he said their home felt like home to him."

Dissociative amnesia and fugue states seldom appear before adolescence and usually occur in adulthood. It is rare for these states to appear for the first time after an individual reaches the age of 50 (Sackeim & Devanand, 1991). However, once they do appear, they may continue well into old age.

DSM Disorder Criteria Summary

Dissociative Fugue

Features of dissociative fugue include the following:

- Sudden, unexpected travel from home or customary place of work, with inability to recall one's past
- Confusion about personal identity or assumption of new identity (partial or complete)
- The disturbance does not occur exclusively during the course of dissociative identity disorder and is not caused by a substance or a general medical condition
- The disturbance causes clinically significant distress or impairment of functioning

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▲ Jeffrey Alan Ingram found himself in Denver not knowing who he was or why he was there after having gone missing a month earlier from Washington state.

Fugue states usually end rather abruptly, and the individual returns home, recalling most, if not all, of what happened. In this disorder, the disintegrated experience is more than memory loss, involving at least some disintegration of identity, if not the complete adoption of a new one.

An apparently distinct dissociative disorder not found in Western cultures is called *amok* (as in "running amok"). Most people with this disorder are males. Amok has attracted attention because individuals in this trancelike state often brutally assault and sometimes kill people or animals; sometimes the person in this state is killed. Those with the disorder probably will not remember an episode occurring. Running amok is only one of a number of "running" syndromes in which an individual enters a trancelike state and suddenly, imbued with a mysterious source of energy, runs or flees for a long time. Except for amok, the prevalence of running disorders is somewhat greater in women, as with most dissociative disorders. Among native peoples of the Arctic, running disorder is termed *pivloktoq*. Among the Navajo tribe, it is called *frenzy witchcraft*. Despite their different culturally determined expression, running disorders seem to meet criteria for dissociative fugue, with the possible exception of amok.

Dissociative Trance Disorder

Dissociative disorders differ in important ways across cultures. In many areas of the world, dissociative phenomena may occur as a trance or possession. The usual sorts of dissociative symptoms, such as sudden changes in personality, are attributed to possession by a spirit important in the particular culture. Often this spirit demands and receives gifts or favors from the family and friends of the victim. Like other dissociative states, trance disorder seems to be most common in women and is often associated with stress or trauma, which, as in dissociative amnesia and fugue states, is current rather than in the past.

Trance and possession are common parts of some traditional religious and cultural practices and are not considered abnormal in that context. Dissociative trances commonly occur in India, Nigeria (where they are called *vinvusa*), Thailand *(phii pob),* and other Asian and African countries (Mezzich et al., 1992; Saxena & Prasad, 1989; van Duijil, Cardeña, & de Jong, 2005). In the United States, culturally accepted dissociation commonly occurs during African American prayer meetings (Griffith et al., 1980), Native American rituals (Jilek, 1982), and Puerto Rican spiritist sessions (Comas-Diaz, 1981). Among Bahamians and African Americans from the South, trance syndromes are often referred to colloquially as "falling out." The personality profiles of 58 cases of dissociative trance disorder in Singapore, derived from objective testing, revealed that these individuals tended to be nervous, excitable, and emotionally unstable relative to "normals" in Singapore (Ng, Yap, Su, Lim, & Ong, 2002). Although trance and possession are almost never seen in Western cultures, they are among the most common forms of dissociative disorders elsewhere. When the state is *undesirable* and considered

pathological by members of the culture, particularly if the trance involves a perception of being possessed by an evil spirit or another person (described next), the proposal for *DSM-5* is to diagnose **dissociative trance disorder (DTD)** as a subtype of dissociative identity disorder (APA, 2010; Spiegel, in press).

Dissociative Identity Disorder

People with **dissociative identity disorder (DID)** may adopt as many as 100 new identities, all simultaneously coexisting, although the average number is closer to 15. In some cases, the identities are complete, each with its own behavior, tone of voice, and physical gestures. But in many cases, only a few characteristics are distinct because the identities are only partially independent. Therefore, the name of the disorder was changed in *DSM-IV* from multiple personality disorder to DID. Consider the case of Jonah, originally reported by Ludwig, Brandsma, Wilbur, Bendfeldt, and Jameson (1972).

Jonah | Bewildering Blackouts

Jonah, 27 years old and black, suffered from severe headaches that were unbearably painful and lasted for increasingly longer periods. Furthermore, he couldn't remember things that happened while he had a headache, except that sometimes a great deal of time passed. Finally, after a particularly bad night, when he could stand it no longer, he arranged for admission to the local hospital. What prompted Jonah to come to the hospital, however, was that other people told him what he did during his severe headaches. For example, he was told that the night before he had a violent fight with another man and attempted to stab him. He fled the scene and was shot at during a high-speed chase by the police. His wife told him that during a previous headache he chased her and his 3-year-old daughter out of the house, threatening them with a butcher knife. During his headaches, and while he was violent, he called himself "Usoffa Abdulla, son of Omega." Once he attempted to drown a man in a river. The man survived, and Jonah escaped by swimming a quarter of a mile upstream. He woke up the next morning in his own bed, soaking wet, with no memory of the incident.

Clinical Description

During Jonah's hospitalization, the staff was able to observe his behavior directly, both when he had headaches and during other periods that he did not remember. He claimed other names at these times, acted differently, and generally seemed to be another person entirely. The staff distinguished three separate identities, or **alters**, in addition to Jonah. (*Alters* is the shorthand term for the different identities or personalities in DID.) The first alter was named Sammy. Sammy seemed rational, calm, and in control. The second alter, King Young, seemed to be in charge of all sexual activity and was particularly interested in having as many heterosexual interactions as possible. The third alter was the violent and dangerous Usoffa Abdulla. Characteristically, Jonah knew nothing of the three alters. Sammy was most aware of the other personalities. King Young and Usoffa Abdulla knew a little bit about the others but only indirectly.

In the hospital, psychologists determined that Sammy first appeared when Jonah was about 6, immediately after Jonah saw his mother stab his father. Jonah's mother sometimes dressed him as a girl in private. On one of these occasions, shortly after Sammy emerged, King Young appeared. When Jonah was 9 or 10, he was brutally attacked by a group of white youths. At this point, Usoffa Abdulla emerged, announcing that his sole reason for existence was to protect Jonah.

DSM-IV-TR criteria for DID include amnesia, as in dissociative amnesia and dissociative fugue. It is proposed that this symptom be given even more prominence in *DSM-5* (Spiegel, in press). In DID, however, identity has also fragmented. How many personalities live inside one body is relatively unimportant, whether there are 3, 4, or even 100 of them. Again, the defining feature of this disorder is that certain aspects of the person's identity are dissociated, accounting for the change in the name of this disorder in *DSM-IV-TR* from multiple personality disorder to DID. This change also corrects the notion that multiple people somehow live inside one body.

Characteristics The person who becomes the patient and asks for treatment is usually a "host" identity. Host personalities usually attempt to hold various fragments of identity together but end up being overwhelmed. The first personality to seek treatment is seldom the original personality of the person. Usually, the host personality develops later (Putnam, 1992). Many patients have at least one impulsive alter who handles sexuality and generates income, sometimes by acting as a prostitute. In other cases, all alters may abstain from sex. Cross-gendered alters are not uncommon. For example, a small agile woman might have a strong powerful male alter who serves as a protector.

dissociative trance disorder (DTD) Altered state of consciousness in which people firmly believe they are possessed by spirits; considered a disorder only where there is distress and dysfunction.
dissociative identity disorder (DID) Disorder in which as many as 100 personalities or fragments of personalities coexist within one body and mind. Formerly known as *multiple personality disorder.*
alters Shorthand term for *alter ego,* one of the different personalities or identities in dissociative identity disorder.

DSM Disorder Criteria Summary
Dissociative Identity Disorder

Features of dissociative identity disorder include the following:

- The presence of two or more distinct identities or personality states, each with its own relatively enduring pattern
- At least two of these identities or personality states recurrently take control of the person's behavior
- Inability to recall important personal information that is too extensive to be explained by ordinary forgetfulness
- The disturbance is not caused by direct physiological effects of a substance (e.g., alcohol intoxication) or general medical condition

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The transition from one personality to another is called a *switch*. Usually, the switch is instantaneous (although in movies and on television it is often drawn out for dramatic effect). Physical transformations may occur during switches. Posture, facial expressions, patterns of facial wrinkling, and even physical disabilities may emerge. In one study, changes in handedness occurred in 37% of the cases (Putnam, Guroff, Silberman, Barban, & Post, 1986).

Can DID Be Faked? Are the fragmented identities "real," or is the person faking them to avoid responsibility or stress? As with conversion disorders, it is difficult to answer this question for several reasons (Kluft, 1999). First, evidence indicates that individuals with DID are suggestible (Bliss, 1984; Giesbrecht et al., 2008; Kihlstrom, 2005). It is possible that alters are created in response to leading questions from therapists, either during psychotherapy or while the person is in a hypnotic state.

Kenneth • The Hillside Strangler

During the late 1970s, Kenneth Bianchi brutally raped and murdered 10 young women in the Los Angeles area and left their bodies naked and in full view on the sides of various hills. Despite overwhelming evidence that Bianchi was the "Hillside Strangler," he continued to assert his innocence, prompting some professionals to think he might have DID. His lawyer brought in a clinical psychologist, who hypnotized him and asked whether there were another part of Ken with whom he could speak. Guess what? Somebody called "Steve" answered and said he had done all the killing. Steve also said that Ken knew nothing about the murders. With this evidence, the lawyer entered a plea of not guilty by reason of insanity.

The prosecution called on the late Martin Orne, a distinguished clinical psychologist and psychiatrist who was one of the world's leading experts on hypnosis and dissociative disorders (Orne, Dinges, & Orne, 1984). Orne used procedures similar to those we described in the context of conversion blindness to determine whether Bianchi was simulating DID or had a true psychological disorder. For example, Orne suggested during an in-depth interview with Bianchi that a true multiple personality disorder included at least three personalities. Bianchi soon produced a third personality. By interviewing Bianchi's friends and relatives, Orne established that there was no independent corroboration of different personalities before Bianchi's arrest. Psychological tests also failed to show significant differences among the personalities; true fragmented identities often score differently on personality tests. Orne concluded that Bianchi responded like someone simulating hypnosis, not someone deeply hypnotized.

Some investigators have studied the ability of individuals to fake dissociative experiences. Spanos, Weeks, and Bertrand (1985) demonstrated in an experiment that a college student could simulate an alter if it was suggested that faking was plausible, as in the interview with Bianchi. All the students in the group were told to play the role of an accused murderer claiming his innocence. The participants received exactly the same interview as Orne administered to Bianchi, word for word. More than 80% simulated an alternate personality to avoid conviction. Groups given vaguer instructions, and no direct suggestion an alternate personality might exist, were much less likely to use one in their defense.

Objective assessment of memory, particularly implicit (unconscious) memory, reveals that the memory processes in patients with DID do not differ from "normals" when the methodologies of cognitive science are used (Allen & Movius, 2000; Huntjens et al., 2002; Huntjens, Postma, Peters, Woertman, & van der Hart, 2003). Huntjens and colleagues (2006) showed that patients with DID acted more like simulators concerning other identities, about which they profess no memory (interidentity amnesia), suggesting the possibility of faking. This is in contrast to reports from interviews with patients with DID that suggest that memories are different from one alter to the next. Furthermore, Kong, Allen, and Glisky (2008) found that, much as with normal participants, patients with DID who memorized words as one identity could remember the words just as well after switching to another identity, contrary to their self-report of interidentity amnesia.

These findings on faking and the effect of hypnosis led Spanos (1996) to suggest that the symptoms of DID could mostly be accounted for by therapists who inadvertently suggested the existence of alters to suggestible individuals, a model known as the "sociocognitive model" because the possibility of identity fragments and early trauma is so-

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▲ Chris Sizemore's history of dissociative identity disorder was dramatized in *The Three Faces of Eve*.

cially reinforced by a therapist (Kihlstrom, 2005; Lilienfeld et al., 1999). A survey of American psychiatrists showed little consensus on the scientific validity of DID, with only one-third in the sample believing that the diagnosis should have been included without reservation in *DSM-IV* (Pope, Oliva, Hudson, Bodkin, & Gruber, 1999). (We return to this point of view when we discuss false memories.)

However, some objective tests suggest that many people with fragmented identities are not consciously and voluntarily simulating (Kluft, 1991, 1999). Condon, Ogston, and Pacoe (1969) examined a film about Chris Sizemore, the real-life subject of the book and movie *The Three Faces of Eve*. They determined that one of the personalities (Eve Black) showed a transient microstrabismus (difference in joined lateral eye movements) that was not observed in the other personalities. These optical differences have been confirmed by S. D. Miller (1989), who demonstrated that DID patients had 4.5 times the average number of changes in optical functioning in their alter identities than control patients who simulated alter personalities. Miller concludes that optical changes, including measures of visual acuity, manifest refraction, and eye muscle balance, would be difficult to fake. Ludwig and colleagues (1972) found that Jonah's various identities had different physiological responses to emotionally laden words, including electrodermal activity, a measure of otherwise imperceptible sweat gland activity, and EEG brain waves.

Using functional magnetic resonance imaging (fMRI) procedures, changes in brain function were observed in one patient while switching from one personality to another. Specifically, this patient showed changes in hippocampal and medial temporal activity after the switch (Tsai, Condie, Wu, & Chang, 1999). A number of subsequent studies confirm that various alters have unique psychophysiological profiles (Cardeña & Gleaves, 2003; Putnam, 1997). Kluft (1999) suggests a number of additional clinical strategies to distinguish malingerers from patients with DID, including the observations that malingerers are usually eager to demonstrate their symptoms and do so in a fluid fashion. Patients with DID, on the other hand, are more likely to attempt to hide symptoms.

Statistics

Jonah had 4 identities, but the average number of alter personalities is reported by clinicians as closer to 15 (Ross, 1997; Sackeim & Devanand, 1991). Of people with DID, the ratio of females to males is as high as 9:1, although these data are based on accumulated case studies rather than survey research (Maldonado, Butler, & Spiegel, 1998). The onset is almost always in childhood, often as young as 4 years of age, although it is usually approximately 7 years after the appearance of symptoms before the disorder is identified (Maldonado et al., 1998; Putnam et al., 1986). Once established, the disorder tends to last a lifetime in the absence of treatment. The form DID takes does not seem to vary substantially over the person's life span, although some evidence indicates the frequency of switching decreases with age (Sackeim & Devanand, 1991). Different personalities may emerge in response to new life situations, as was the case with Jonah.

There are not good epidemiological studies on the prevalence of the disorder in the population at large, although investigators now think it is more common than previously estimated (Kluft, 1991; Ross, 1997). For example, semistructured interviews of large numbers of severely disturbed inpatients found prevalence rates of DID of between 3% and 6% in North America (Ross, 1997; Ross, Anderson, Fleisher, & Norton, 1991; Saxe et al., 1993) and approximately 2% in Holland (Friedl & Draijer, 2000). In the best survey to date in a nonclinical (community) setting, a prevalence of 1.5% was found during the previous year (Johnson et al., 2006).

A large percentage of DID patients have simultaneous psychological disorders that may include anxiety, substance abuse, depression, and personality disorders (Giesbrecht et al., 2008; Johnson et al., 2006; Kluft, 1999; Ross et al., 1990).

Causes

Life circumstances that encourage the development of DID seem clear in at least one respect. Almost every patient presenting with this disorder reports being horribly, often unspeakably, abused as a child.

Sybil ✦ Continual Abuse

You may have seen the movie that was based on Sybil's biography (Schreiber, 1973). Sybil's mother had schizophrenia, and her father refused or was unable to intervene in the mother's brutality. Day after day throughout her childhood, Sybil was sexually tortured and occasionally nearly murdered. Before she was 1 year old, her mother began tying her up in various ways and, on occasion, suspending her from the

ceiling. Many mornings, her mother placed Sybil on the kitchen table and forcefully inserted various objects into her vagina. Sybil's mother reasoned, psychotically, that she was preparing her daughter for adult sex. In fact, she so brutally tore the child's vaginal canal that scars were evident during adult gynecological exams. Sybil was also given strong laxatives but prohibited from using the bathroom. Because of her father's detachment and the normal appearance of the family, the abuse continued without interruption throughout Sybil's childhood.

Imagine you are a child in a situation like this. What can you do? You're too young to run away. You're too young to call the authorities. Although the pain may be unbearable, you have no way of knowing it is unusual or wrong. But you can do one thing. You can escape into a fantasy world; you can be somebody else. If the escape blunts the physical and emotional pain just for a minute or makes the next hour bearable, chances are you'll escape again. Your mind learns there is no limit to the identities that can be created as needed. You do whatever it takes to get through life. Not all the trauma is caused by abuse. Putnam (1992) describes a young girl in a war zone who saw both her parents blown to bits in a minefield. In a heart-wrenching response, she tried to piece the bodies back together, bit by bit.

Such observations have led to wide-ranging agreement that DID is rooted in a natural tendency to escape or "dissociate" from the unremitting negative affect associated with severe abuse (Kluft, 1984, 1991). A lack of social support during or after the abuse also seems implicated. A study of 428 adolescent twins demonstrated that a surprisingly major portion of the cause of dissociative experience could be attributed to a chaotic, nonsupportive family environment. Individual experience and personality factors also contributed to dissociative experiences (Waller & Ross, 1997).

The behavior and emotions that make up dissociative disorders seem related to otherwise normal tendencies present in all of us to some extent. It is quite common for otherwise normal individuals to escape in some way from emotional or physical pain (Butler, Duran, Jasiukaitis, Koopman, & Spiegel, 1996; Spiegel & Cardeña, 1991). Noyes and Kletti (1977) surveyed more than 100 survivors of various life-threatening situations and found that most had experienced some type of dissociation, such as feelings of unreality, a blunting of emotional and physical pain, and even separation from their bodies. Dissociative amnesia and fugue states are clearly reactions to severe life stress. But the life stress or trauma is in the present rather than the past, as in the case of the overwrought mother who suffered from dissociative amnesia. Many patients are escaping from legal difficulties or severe stress at home or on the job (Sackeim & Devanand, 1991). But sophisticated statistical analyses indicate that "normal" dissociative reactions differ substantially from the pathological experiences we've described (Waller, Putnam, & Carlson, 1996; Waller & Ross, 1997) and that at least some people do not develop severe pathological dissociative experiences, no matter how extreme the stress. These findings are consistent with our diathesis–stress model in that only with the appropriate vulnerabilities (the diathesis) will someone react to stress with pathological dissociation.

You may have noticed that DID seems similar in its etiology to posttraumatic stress disorder (PTSD).

One perspective suggests that DID is an extreme subtype of PTSD, with a much greater emphasis on the process of dissociation than on symptoms of anxiety, although both are present in each disorder (Butler et al., 1996). Some evidence also shows that the "developmental window" of vulnerability to the abuse that leads to DID closes at approximately 9 years of age (Putnam, 1997). After that, DID is unlikely to develop, although severe PTSD might. If true, this is a particularly good example of the role of development in the etiology of psychopathology.

We also must remember that we know relatively little about DID. Our conclusions are based on retrospective case studies or correlations rather than on the prospective examination of people who may have undergone the severe trauma that seems to lead to DID (Kihlstrom, 2005; Kihlstrom, Glisky, & Anguilo, 1994). Therefore, it is hard to say what psychological or biological factors might contribute, but there are hints concerning individual differences that might play a role.

Suggestibility Suggestibility is a personality trait distributed normally across the population, much like weight and height. Some people are more suggestible than others; some are relatively immune to suggestibility; and the majority fall in the midrange.

Did you ever have an imaginary childhood playmate? Many people did, and it is one sign of the ability to lead a rich fantasy life, which can be helpful and adaptive. But it also seems to correlate with being suggestible or easily hypnotized (some people equate the terms *suggestibility* and *hypnotizability*). A hypnotic trance is also similar to dissociation (Bliss, 1986; Butler et al., 1996; Carlson & Putnam, 1989). People in a trance tend to be focused on one aspect of their world, and they become vulnerable to suggestions by the hypnotist. There is also the phenomenon of self-hypnosis, in which individuals can dissociate from most of the world around them and "suggest" to themselves that, for example, they won't feel pain in one of their hands.

According to the *autohypnotic model,* people who are suggestible may be able to use dissociation as a defense against extreme trauma (Putnam, 1991). As many as 50% of DID patients clearly remember imaginary playmates in childhood (Ross et al., 1990); whether they were created before or after the trauma is not entirely clear. According to this view, when the trauma becomes unbearable, the person's very identity splits into multiple dissociated identities. Children's ability to distinguish clearly between real-

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▲ A person in a hypnotic trance is suggestible and may become absorbed in a particular experience.

ity and fantasy as they grow older may be what closes the developmental window for developing DID at approximately age 9. People who are less suggestible may develop a severe posttraumatic stress reaction but not a dissociative reaction.

Biological Contributions As in PTSD, where the evidence is more solid, there is almost certainly a biological vulnerability to DID, but it is difficult to pinpoint. For example, in the large twin study mentioned earlier (Waller & Ross, 1997), none of the variance or identifiable causal factors was attributable to heredity: All of it was environmental. As with anxiety disorders, more basic heritable traits, such as tension and responsiveness to stress, may increase vulnerability. However, much as in PTSD, there is some evidence of smaller hippocampal and amygdala volume in patients with DID compared to "normals" (Vermetten, Schmahl, Lindner, Loewenstein, & Bremner, 2006).

Head injury and resulting brain damage may induce amnesia or other types of dissociative experience. But these conditions are usually easily diagnosed because they are generalized and irreversible and are associated with an identifiable head trauma (Butler et al., 1996). Finally, strong evidence exists that sleep deprivation produces dissociative symptoms such as marked hallucinatory activity (Giesbrecht et al., 2007).

Real and False Memories One of the most controversial issues in the field of abnormal psychology today concerns the extent to which memories of early trauma, particularly sexual abuse, are accurate. Some suggest that many such memories are simply the result of strong suggestions by careless therapists who assume people with this condition have been abused. The stakes in this controversy are enormous, with considerable opportunity for harm to innocent people on each side of the controversy.

On the one hand, if early sexual abuse did occur but is not remembered because of dissociative amnesia, it is crucially important to reexperience aspects of the trauma under the direction of a skilled therapist to relieve current suffering. Without therapy, the patient is likely to suffer from PTSD or a dissociative disorder indefinitely. It is also important that perpetrators are held accountable for their actions because abuse of this type is a crime and prevention is an important goal.

On the other hand, if memories of early trauma are inadvertently created in response to suggestions by a careless therapist but seem real to the patient, false accusations against loved ones could lead to irreversible family breakup and, perhaps, unjust prison sentences for those falsely accused as perpetrators. In recent years, allegedly inaccurate accusations based on false memories have led to substantial lawsuits against therapists resulting in awards of millions of dollars in damages. As with most issues that reach this level of contention and disagreement, it is clear that the final answer will not involve an all-or-none resolution. There is irrefutable evidence that false memories *can* be created by reasonably well-understood psychological processes (Bernstein & Loftus, 2009; Ceci, 2003; Geraerts et al., 2009; Lilienfeld et al., 1999; Loftus, 2003; Loftus & Davis, 2006; McNally, 2001, 2003; Schacter, 1995). But there is also good evidence that early traumatic experiences can cause selective dissociative amnesia, with substantial implications for psychological functioning (Gleaves, 1996; Gleaves, Smith, Butler, & Spiegel, 2004; Kluft, 1999; Spiegel, 1995).

Evidence supporting the existence of distorted or illusory memories comes from experiments like one by the distinguished cognitive psychologist Elizabeth Loftus and her colleagues (Loftus, 2003; Loftus & Davis, 2006). Loftus, Coan, and Pickrell (1996) successfully convinced a number of individuals that they had been lost for an extended period when they were approximately 5 years old, which was not true. A trusted companion was recruited to "plant" the memory. In one case, a 14-year-old boy was told by his older brother that he had been lost in a nearby shopping mall when he was 5 years old, rescued by an older man, and reunited with his mother and brother. Several days after receiving this suggestion, the boy reported remembering the event and even that he felt frightened when he was lost. As time went by, the boy remembered more and more details of the event, beyond those described in the "plant," including an exact description of the older man. When he was finally told the incident never happened, the boy was surprised, and he continued to describe details of the event as if they were true. More recently, Bernstein & Loftus (2009) reviewed a series of experiments demonstrating that, for example, creating a false memory of becoming ill after eating egg salad led to eating less egg salad and reporting a distaste for egg salad up to 4 months later dur-

ing a test in which the participants didn't know they were being tested for food preferences.

Young children are quite unreliable in reporting accurate details of events (Bruck, Ceci, Francouer, & Renick, 1995), particularly emotional events (Howe, 2007). In one study (Bruck et al., 1995), 35 girls who were 3 years old were given a genital exam as part of their routine medical checkup; another 35 girls were not (the control group). Shortly after the exam, with her mother present, each girl was asked to describe where the doctor had touched her. She was then presented with an anatomically correct doll and asked again to point out where the doctor had touched her. The findings indicated that the children were inaccurate in reporting what happened. Approximately 60% of those who were touched in the genital region refused to indicate this, whether the dolls were used or not. Of the children in the control group, however, approximately 60% indicated genital insertions or other intrusive acts by the doctor, even though nothing of the sort had occurred.

In another set of studies (Ceci, 2003) preschool children were asked to think about actual events that they had experienced, such as an accident, and about fictitious events, such as having to go to the hospital to get their fingers removed from a mousetrap. Each week for 10 consecutive weeks an interviewer asked each child to choose one of the scenes and to "think very hard and tell me if this ever happened to you." The child thus experienced thinking hard and visualizing both real and fictitious scenes over an extended period. After 10 weeks, the children were examined by a new interviewer who had not participated in the experiment (Ceci, 1995, 2003).

In one of Ceci's studies, 58% of the preschool children described the fictitious event as if it had happened. Another 25% of the children described the fictitious events as real a majority of the time. Furthermore, the children's narratives were detailed, coherent, and embellished in ways that were not suggested originally. More telling was that in one study 27% of the children, when told their memory was false, claimed that they really did remember the event.

But there is also plenty of evidence that therapists need to be sensitive to signs of trauma that may not be fully remembered in patients presenting with symptoms of dissociative disorder or PTSD. Even if patients are unable to report or remember early trauma, it can sometimes be confirmed through corroborating evidence (Coons, 1994). In one study, Williams (1994) interviewed 129 women with previously documented histories, such as hospital records, of having been sexually abused as children. Thirty-eight percent did not recall the incidents that had been reported to authorities at least 17 years earlier, even with extensive probing of their abuse histories. This lack of recall was more extensive if the victim had been young and knew the abuser. But Goodman and colleagues (2003) interviewed 175 individuals with documented child sexual abuse histories and found that most participants (81%) remembered and reported the abuse. Older age when the abuse ended and emotional support following initial disclosure of the abuses were associated with higher rates of disclosures. McNally and Geraerts (2009) also present evidence suggesting that some people, after many years, simply forget these early experiences and recall them after encountering some reminders outside of therapy. In this group, then, it's not necessary to invoke the concepts of repression, trauma, or false memory. It is simple forgetting. In summary, among those individuals reporting memories of sexual abuse, some may have experienced it and remembered it all along, some people may have false memories, some may have recovered memories in therapy of "repressed" sexual abuse, and some may have simply forgotten the incident but remember later.

As Brewin, Andrews, and Gotlib (1993) also point out, the available data from cognitive science would suggest that an extreme reconstructive model of (false) memory induced by careless therapists would be rare because most individuals can recall important details of their childhood, particularly if they are unique and unexpected.

How will this controversy be resolved? Because false memories can be created through strong repeated suggestions by an authority figure, therapists must be fully aware of the conditions under which this is likely to occur, particularly when dealing with young children. This requires extensive knowledge of the workings of memory and other aspects of psychological functioning and illustrates, again, the dangers of dealing with inexperienced or inadequately trained psychotherapists. Elaborate tales of satanic abuse of children under the care of elderly women in day care centers are most likely cases of memories implanted by aggressive and careless therapists or law enforcement officials (Lilienfeld et al., 1999; Loftus & Davis, 2006; McNally, 2003). In some cases, elderly caregivers have been sentenced to life in prison.

However, many people with dissociative disorder and PTSD have suffered documented extreme abuse and trauma, which could then become dissociated from awareness. It may be that future research will find that the severity of dissociative amnesia is directly related to the severity of the trauma in vulnerable individuals, and this type of severe dissociative reaction is also likely to be proved as qualitatively different from "normal" dissociative experiences we all have occasionally, such as feeling unreal or not here for a moment or two. (See, for example, Kluft, 1999; Waller et al., 1996.) Advocates on both sides of this issue agree that clinical science must proceed as quickly as possible to specify the processes under which the implantation of false memories is likely and to define the presenting features that indicate a real but dissociated traumatic experience (Gleaves et al., 2004; Kihlstrom, 1997, 2005; Lilienfeld et al., 1999; Pope, 1996, 1997). Until then, mental health professionals must be extremely careful not to prolong unnecessary suffering among both victims of actual abuse and victims falsely accused as abusers.

Treatment

Individuals who experience dissociative amnesia or a fugue state usually get better on their own and remember what they have forgotten. The episodes are so clearly related to

current life stress that prevention of future episodes usually involves therapeutic resolution of the distressing situations and increasing the strength of personal coping mechanisms. When necessary, therapy focuses on recalling what happened during the amnesic or fugue states, often with the help of friends or family who know what happened, so that patients can confront the information and integrate it into their conscious experience. For more difficult cases, hypnosis or benzodiazepines (minor tranquilizers) have been used, with suggestions from the therapist that it is okay to remember the events (Maldonado et al., 1998).

For DID, however, the process is not so easy. With the person's very identity shattered into many elements, reintegrating the personality might seem hopeless. Fortunately, this is not always the case. Although no controlled research has been reported on the effects of treatment, there are some documented successes of attempts to reintegrate identities through long-term psychotherapy (Brand et al., 2009; Ellason & Ross, 1997; Kluft, 2009; Putnam, 1989; Ross, 1997). Nevertheless, the prognosis for most people remains guarded. Coons (1986) found that only 5 of 20 patients achieved a full integration of their identities. Ellason and Ross (1997) reported that 12 of 54 (22.2%) patients had achieved integration 2 years after presenting for treatment, which in most cases had been continuous. These results could be attributed to other factors than therapy because no experimental comparison was present (Powell & Howell, 1998).

The strategies that therapists use today in treating DID are based on accumulated clinical wisdom, as well as on procedures that have been successful with PTSD (Gold & Seibel, 2009; Keane, Marx, & Sloan, in press; Maldonado et al., 1998; see Chapter 4). The fundamental goal is to identify cues or triggers that provoke memories of trauma, dissociation, or both and to neutralize them. More important, the patient must confront and relive the early trauma and gain control over the horrible events, at least as they recur in the patient's mind (Kluft, 1996, 1999, 2009; Ross, 1997). To instill this sense of control, the therapist must skillfully, and slowly, help the patient visualize and relive aspects of the trauma until it is simply a terrible memory instead of a current event. Because the memory is unconscious, aspects of the experience are often not known to either the patient or the therapist until they emerge during treatment. Hypnosis is often used to access unconscious memories and bring various alters into awareness. Because the process of dissociation may be similar to the process of hypnosis, the latter may be a particularly efficient way to access traumatic memories (Maldonado et al., 1998). (There is as yet no evidence that hypnosis is a *necessary* part of treatment.) DID seems to run a chronic course and seldom improves spontaneously, which confirms that current treatments, primitive as they are, have some effectiveness.

It is possible that reemerging memories of trauma may trigger further dissociation. The therapist must be on guard against this happening. Trust is important to any therapeutic relationship, but it is essential in the treatment of DID. Occasionally, medication is combined with therapy, but there is little indication that it helps much. What little clinical evidence there is indicates that antidepressant drugs might be appropriate in some cases (Coons, 1986; Kluft, 1996; Putnam & Loewenstein, 1993).

Concept Check 5.2

Diagnose the dissociative disorders described here by choosing one of the following: (a) dissociative fugue, (b) depersonalization disorder, (c) generalized amnesia, (d) dissociative identity disorder, and (e) localized amnesia.

1. Ann was found wandering the streets, unable to recall any important personal information. After searching her purse and finding an address, doctors were able to contact her mother. They learned that Ann had just been in a terrible accident and was the only survivor. Ann could not remember her mother nor any details of the accident. She was distressed. _____
2. Karl was brought to a clinic by his mother. She was concerned because at times his behavior was strange. His speech and his way of relating to people and situations would change dramatically, almost as if he were a different person. What bothered her and Karl most was that he could not recall anything he did during these periods. _____
3. Terry complained about feeling out of control. She said she sometimes felt as if she were floating under the ceiling and just watching things happen to her. She also experienced tunnel vision and felt uninvolved in the things that went on in the room around her. This always caused her to panic and perspire. _____
4. Henry is 64 and recently arrived in town. He does not know where he is from or how he got here. His driver's license proves his name, but he is unconvinced it is his. He is in good health and not taking any medication. _____
5. Rosita cannot remember what happened last weekend. On Monday she was admitted to a hospital, suffering from cuts, bruises, and contusions. It also appeared that she had been sexually assaulted. _____

On the Spectrum Somatoform Disorders in *DSM-5*

The grouping of somatoform disorders under the heading of "somatoform" is based largely on the assumption that "somatization" is a common process in which a mental disorder manifests itself in the form of physical symptoms. The specific disorders, then, simply reflect the different ways in which symptoms can be expressed physically. Recently, major questions have arisen concerning the classification of somatoform disorders, and a proposal now exists that would radically revise the classification of these disorders in *DSM-5* (APA, 2010; Noyes, Stuart, & Watson, 2008; Voigt et al., 2010).

Specifically, somatization disorder, hypochondriasis, undifferentiated somatoform disorder, and pain disorder all share presentations of somatic symptoms accompanied by cognitive distortions in the form of misattributions of or excessive preoccupation with symptoms. These cognitive distortions may include excessive anxiety about health or physical symptoms, a tendency to think the worst or "catastrophize" about these symptoms, and very strong beliefs that physical symptoms might be more serious than health-care professionals have recognized. Also, people presenting with these disorders often make health concerns a very central part of their lives; in other words, they adopt the "sick role." For this reason, *DSM-5* may focus on the severity and number of physical symptoms, and the severity of anxiety focused on the symptoms, and group them into a new category entitled "complex somatic symptom disorder" (CSSD). Preliminary explorations of the validity and utility of this strategy indicate that this new dimensional approach, reflecting both physical and psychological symptom severity, would be helpful to clinicians (Noyes et al., 2008; Voigt et al., 2010).

Another advantage of this approach is that there would be less burden on physicians to make tricky determinations on whether the symptoms have physical causes. Rather, the combination of chronic physical symptoms accompanied by the psychological factors of misattributing the meaning of the symptoms and excessive concern would be sufficient to make the diagnosis. The *DSM-5* proposal also suggests that the name of the larger category should be changed from "somatoform disorders" to "somatic symptom disorders"; the new category would include not only the *DSM-IV* somatoform disorders, but also psychological factors affecting medical conditions (see Chapter 7) and the factitious disorders because all involve the presentation of physical symptoms and/or concern about medical illness.

Summary

Somatoform Disorders

What are the defining features of somatoform disorders?

- Individuals with somatoform disorders are pathologically concerned with the appearance or functioning of their bodies and bring these concerns to the attention of health professionals, who usually find no identifiable medical basis for the physical complaints.
- There are several types of somatoform disorders. Hypochondriasis is a condition in which individuals believe they are seriously ill and become anxious over this possibility. Somatization disorder is characterized by a seemingly unceasing and wide-ranging pattern of physical complaints that dominate the individual's life and interpersonal relationships. In conversion disorder, there is physical malfunctioning, such as paralysis, without any apparent physical problems. In pain disorder, psychological factors are judged to play a major role in maintaining physical suffering. In body dysmorphic disorder (BDD), a person who looks normal is obsessively preoccupied with some imagined defect in appearance (imagined ugliness).
- Distinguishing among conversion reactions, real physical disorders, and outright malingering (or faking) is sometimes difficult. Even more puzzling can be factitious disorder, in which the person's symptoms are feigned and under voluntary control, as with malingering, but for no apparent reason.
- The causes of somatoform disorders are not well understood, but some, including hypochondriasis and BDD, seem closely related to anxiety disorders.

What treatments have been developed for somatoform disorders?

- Treatment of somatoform disorders ranges from basic techniques of reassurance and social support to those meant to reduce stress and remove any secondary gain for the behavior. Recently, specifically tailored cognitive-behavioral therapy has proved successful with hypochondriasis. Patients suffering from BDD often turn to plastic surgery or other medical interventions, which more often than not increase their preoccupation and distress.

Dissociative Disorders

What are the five types of dissociative disorders?

- Dissociative disorders are characterized by alterations in perceptions: a sense of detachment from one's own self, from the world, or from memories.

› Dissociative disorders include depersonalization disorder, in which the individual's sense of personal reality is temporarily lost (depersonalization), as is the reality of the external world (derealization). In dissociative amnesia, the individual may be unable to remember important personal information. In generalized amnesia, the individual is unable to remember anything; more commonly, the individual is unable to recall specific events that occur during a specific period (localized or selective amnesia). In dissociative fugue, memory loss is combined with an unexpected trip (or trips). In the extreme, new identities, or alters, may be formed, as in dissociative identity disorder (DID). Finally, the newly defined dissociative trance disorder is considered to cover dissociations that may be culturally determined.

What factors influence the etiology and treatment of dissociative disorders?

› The causes of dissociative disorders are not well understood but often seem related to the tendency to escape psychologically from memories of traumatic events.
› Treatment of dissociative disorders involves helping the patient reexperience the traumatic events in a controlled therapeutic manner to develop better coping skills. In the case of DID, therapy is often long term and may include antidepressant drugs. Particularly essential with this disorder is a sense of trust between therapist and patient.

Key Terms

somatoform disorder, 165
dissociative disorder, 165
hypochondriasis, 166
somatization disorder, 170
pain disorder, 172
conversion disorder, 173
malingering, 174
factitious disorder, 174
body dysmorphic disorder (BDD), 178
derealization, 183
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localized or selective amnesia, 184
dissociative fugue, 185
dissociative trance disorder (DTD), 187
dissociative identity disorder (DID), 187
alters, 187

Answers to Concept Checks

5.1

1. b; 2. c; 3. d; 4. e; 5. a

5.2

1. c; 2. d; 3. b; 4. a; 5. e

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› *Rachel, an Example of Dissociative Identity Disorder:* These three clips explore her multiple personalities, how she copes with them, and how they emerge in response to threats within the environment.
› *Doug, an Example of Body Dysmorphic Disorder:* This interview by Katharine Phillips, an authority on this disorder, shows how it cripples this man's life until he seeks treatment for it.

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CengageNOW also contains Mark Durand's *Video Concept Reviews* on these challenging topics.

- Somatoform Disorders
- Hypochondriasis
- Concept Check: Hypochondriasis versus Other Disorders
- Conversion Disorder
- Body Dysmorphic Disorder
- Dissociative Disorders
- Depersonalization Disorder
- Dissociative Amnesia
- Dissociative Fugue
- Dissociative Trance Disorder
- Dissociative Identity Disorder
- False and Recovered Memories, Malingering

Chapter Quiz

1. The primary symptom of hypochondriasis is:
   a. fear of developing a disease
   b. fear of spreading a disease
   c. fear of contact with diseased individuals
   d. fear of currently having a disease
2. Someone who presents with the following symptoms might have hypochondriasis.
   a. interpreting momentary flutters in the stomach as a sign of illness
   b. reluctance to visit the doctor for fear of having a panic attack
   c. enjoyment of the immediate attention received when visiting a doctor
   d. realization that the presence of an illness could qualify the individual for full-time disability benefits
3. Choose the scenario that best demonstrates a somatization disorder.
   a. Lisa reports that she has continuous nausea and is unable to work, but a medical exam finds no sign of illness. Lisa claims she only feels better when her husband stays home to nurse her.
   b. Eddie visits 11 different physicians in 6 months but is frustrated that no doctor seems able to make an adequate diagnosis.
   c. Sherry has physical complaints that have lasted at least 10 years. Her symptoms include pain in her feet, hands, and neck; alternating diarrhea and constipation; and difficulty walking. Sherry's physician cannot find any illness to account for these complaints.
   d. Pedro stops working because he thinks that his ears are twice the size they should be and that he looks like a freak. His therapist observes, however, that Pedro's ears are a normal size.
4. Hypochondriasis is related to _____, whereas somatization disorder is linked to _____.
   a. obsessive-compulsive disorder; schizotypal personality disorder
   b. dissociative disorder; obsessive-compulsive disorder
   c. psychotic disorders; anxiety disorders
   d. anxiety disorder; antisocial personality disorder
5. In factitious disorder:
   a. the individual is faking symptoms for personal gain
   b. the individual is voluntarily producing the symptoms without any obvious financial or other external incentives
   c. the individual is not in control of the symptoms but there is no physical explanation
   d. the symptoms are caused by a yet-to-be-identified virus
6. Jorge, a 19-year-old male, was hospitalized after his legs collapsed under him while walking to class. He could not regain his stance and has been unable to walk since, although he desperately wants to walk again. A neurological exam revealed no medical problem. Jorge's behavior is consistent with:
   a. somatization disorder
   b. conversion disorder
   c. malingering
   d. body dysmorphic disorder

7. Mrs. Thompson brought her 4-year-old daughter, Carmen, to the emergency room, stating that the child had been vomiting nonstop throughout the morning. Carmen's condition improved over the course of several days. On the day of her discharge from the hospital, a nurse walked in as Mrs. Thompson was giving Carmen a drink of floor cleaner. Mrs. Thompson's behavior is consistent with:
   a. parental hypochondriasis
   b. Munchausen syndrome by proxy
   c. conversion syndrome by proxy
   d. parental somatization
8. _____ describes the experience of losing a sense of your own reality, whereas _____ describes losing your sense of reality of the external world.
   a. Depersonalization; derealization
   b. Derealization; somatization
   c. Derealization; depersonalization
   d. Somatization; derealization
9. Michael's wife, Jennifer, reported him missing to the police in 1998. Two years later she saw Michael in an airport. He lived two states away from Jennifer, was married to another woman, and had two children with her. Michael told Jennifer that his name was Danny, not Michael, and that he had never met her before. Michael's presentation is consistent with:
   a. multiple personality disorder
   b. dissociative trance disorder
   c. dissociative identity disorder
   d. dissociative fugue
10. The different identities or personalities in dissociative identity disorder are called _____, whereas the change from one personality to another is called a _____.
   a. masks; transition
   b. faces; switch
   c. façades; transition
   d. alters; switch

(See Appendix A for answers.)

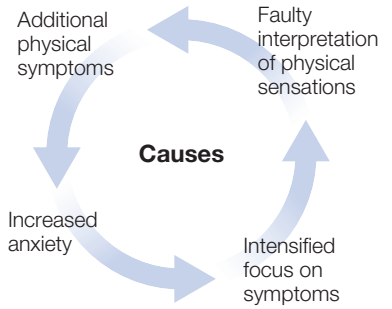
Exploring Somatoform and Dissociative Disorders

These two sets of disorders share some common features and are strongly linked historically as "hysterical neuroses." Both are relatively rare and not yet well understood.

SOMATOFORM DISORDERS

Characterized by a pathological concern with physical functioning or appearance

Hypochondriasis



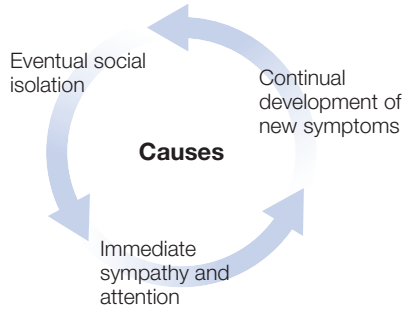
Characteristics

- Severe anxiety over physical problems that are medically undetectable
- Affects women and men equally
- May emerge at any age
- Evident in diverse cultures

Treatment

- Psychotherapy to challenge illness perceptions
- Counseling and/or support groups to provide reassurance

Somatization Disorder



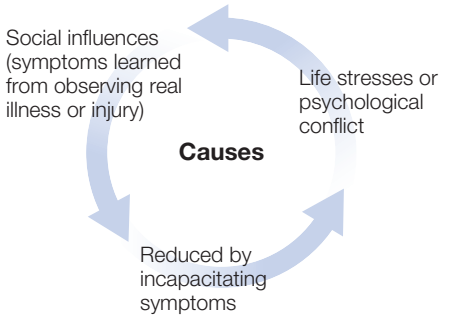
Characteristics

- Reports of multiple physical symptoms without a medical basis
- Runs in families; probably heritable basis
- Rare—most prevalent among unmarried women in low socioeconomic groups
- Onset usually in adolescence; often persists into old age

Treatment

- Hard to treat
- Cognitive-behavioral therapy (CBT) to provide reassurance, reduce stress, and minimize help-seeking behaviors
- Therapy to broaden basis for relating to others

Conversion Disorder



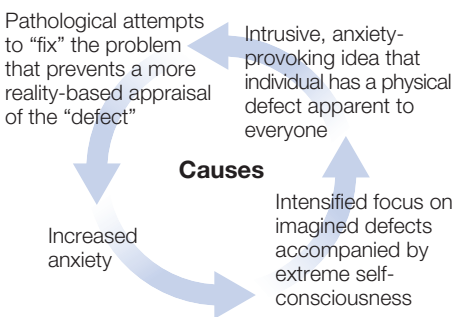
Characteristics

- Severe physical dysfunctioning (e.g., paralysis and blindness) without corresponding physical pathology
- Affected people are genuinely unaware that they can function normally
- May coincide with other problems, especially somatization disorder
- Most prevalent in low socioeconomic groups, women, and men under extreme stress (e.g., soldiers)

Treatment

- Same as for somatization disorder, with emphasis on resolving life stress or conflict and reducing help-seeking behaviors

Body Dysmorphic Disorder (BDD)



Characteristics

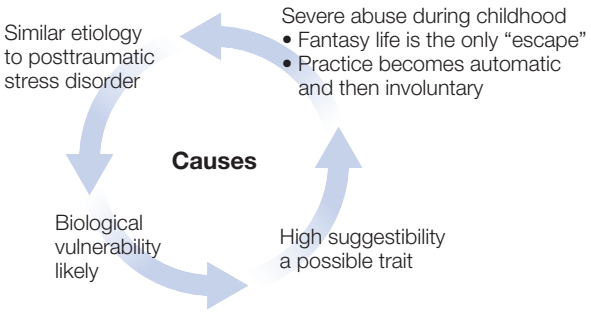
- Socially disabling preoccupation with a normal physical feature that is believed to be hideous (“imagined ugliness”)
- Prevalence is not known; affects men and women equally
- Associated with obsessive-compulsive disorder

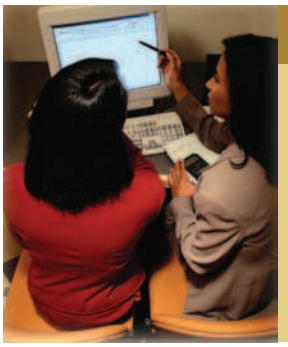
Treatment

- CBT treatments seem most effective
- Drug treatments can provide relief for some sufferers
- Without treatment, BDD lasts a lifetime

DISSOCIATIVE DISORDERS

Characterized by detachment from the self (depersonalization) and objective reality (derealization)





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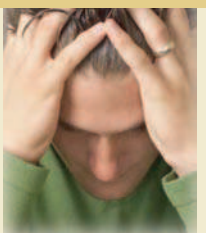
Controversy

The scientific community is divided over the question of whether multiple identities are a genuine experience or faked. Studies have shown that "false memories" can be created ("implanted") by therapists. Other tests confirm that various alters are physiologically distinct.

Disorder	Characteristics	Treatment
Dissociative Identity Disorder (DID)	• Affected person adopts new identities, or alters, that coexist simultaneously; the alters may be complete and distinct personalities or only partly independent • Average number of alters is 15 • Childhood onset; affects more women than men • Patients often suffer from other psychological disorders simultaneously • Rare outside of Western cultures	• Long-term psychotherapy may reintegrate separate personalities in 25% of patients • Treatment of associated trauma similar to posttraumatic stress disorder; lifelong condition without treatment
Depersonalization	• Severe and frightening feelings of detachment dominate the person's life • Affected person feels like an outside observer of his or her own mental or body processes • Causes significant distress or impairment in functioning, especially emotional expression and deficits in perception • Some symptoms are similar to those of panic disorder • Rare; onset usually in adolescence	• Psychological treatments similar to those for panic disorder may be helpful • Stresses associated with onset of disorder should be addressed • Tends to be lifelong
Dissociative Fugue	• Memory loss accompanies an unplanned journey • Person sometimes assumes a new identity or becomes confused about an old identity • Usually associated with an intolerable situation • Fugue states usually end abruptly • Typically adult onset	• Usually self-correcting when current life stress is resolved • If needed, therapy focuses on retrieving lost information
Dissociative Amnesia	• Generalized: Inability to remember anything, including identity; comparatively rare • Localized: Inability to remember specific events (usually traumatic); frequently occurs in war • More common than general amnesia • Usually adult onset for both types	• Usually self-correcting when current life stress is resolved • If needed, therapy focuses on retrieving lost information
Dissociative Trance	• Sudden changes in personality accompany a trance or "possession" • Causes significant distress and/or impairment in functioning • Often associated with stress or trauma • Prevalent worldwide, usually in a religious context; rarely seen in Western cultures • More common in women than in men	• Little is known



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CHAPTER 6

Mood Disorders and Suicide

Chapter Outline

Student Learning Outcomes*

Demonstrate knowledge and understanding representing appropriate breadth and depth in selected content areas of psychology.	› Biological bases of behavior and mental processes, including physiology, sensation, perception, comparative, motivation, and emotion (APA SLO 1.2.a [3]) ***(see textbook pages 215–217)*** › Variability and continuity of behavior and mental processes within and across species (APA SLO 1.2.d [2]) ***(see textbook pages 211–214, 236–238)***
Use the concepts, language, and major theories of the discipline to account for psychological phenomena.	› Describe behavior and mental processes empirically, including operational definitions (APA SLO 1.3.a) ***(see textbook pages 202–210)*** › Integrate theoretical perspectives to produce comprehensive and multifaceted explanations (APA SLO 1.3.e) ***(see textbook pages 224–226, 238–240)***
Identify appropriate applications of psychology in solving problems, such as:	› Origin and treatment of abnormal behavior (APA SLO 4.2.b) ***(see textbook pages 227–235, 241–243)***

*Portions of this chapter cover learning outcomes suggested by the American Psychological Association (2007) in their guidelines for the undergraduate psychology major. Chapter coverage of these outcomes is identified by APA Goal and APA Suggested Learning Outcome (SLO).

Understanding and Defining Mood Disorders

- **What is the difference between a depressive episode and a manic or hypomanic episode?**
- **What are the clinical symptoms of major depressive disorder, dysthymic disorder, and bipolar disorder?**

Think back over the past month of your life. It may seem normal in most respects; you studied during the week, socialized on the weekend, and thought about the future once in a while. Perhaps you were anticipating with some pleasure the next school break or seeing an old friend. But maybe sometime during the past month you also felt kind of down because you got a lower mark than you expected on a test after studying hard or broke up with your boyfriend or girlfriend or, worse yet, one of your grandparents died. Think about your feelings during this period. Were you sad? Perhaps you remember crying. Maybe you felt listless, and you couldn't seem to get up the energy to study or go out with your friends. It may be that you feel this way once in a while for no good reason you can think of and your friends think you're moody.

If you are like most people, you know your mood will pass. You will be back to your old self in a few days or a week. If you never felt down and always saw only what was good in a situation, it would be more unusual (and would also seem so to your friends) than if you were depressed once in a while. Feelings of depression (and joy) are universal, which makes it all the more difficult to understand disorders of mood, disorders that can be so incapacitating that violent suicide may seem by far a better option than living. Consider the case of Katie.

Katie | Weathering Depression

Katie was an attractive but shy 16-year-old girl who came to our clinic with her parents. For several years, Katie seldom interacted with anybody outside her family because of her considerable social anxiety. Going to school was difficult, and as her social contacts decreased her days became empty and dull. By the time she was 16, a deep, all-encompassing depression blocked the sun from her life. Here is how she described it later:

> The experience of depression is like falling into a deep, dark hole that you cannot climb out of. You scream as you fall, but it seems like no one hears you. Some days you float upward without even trying; on other days, you wish that you would hit bottom so that you would never fall again. Depression affects the way you interpret events. It influences the way you see yourself and the way you see other people. I remember looking in the mirror and thinking that I was the ugliest creature in the world. Later in life,

when some of these ideas would come back, I learned to remind myself that I did not have those thoughts yesterday and chances were that I would not have them tomorrow or the next day. It is a little like waiting for a change in the weather.

But at 16, in the depths of her despair, Katie had no such perspective. She often cried for hours at the end of the day. She had begun drinking alcohol the year before, with the blessing of her parents, strangely enough, because the pills prescribed by her family doctor did no good. A glass of wine at dinner had a temporary soothing effect on Katie, and both she and her parents, in their desperation, were willing to try anything that might make her a more functional person. But one glass was not enough. She drank increasingly more often. She began drinking herself to sleep. It was a means of escaping what she felt: "I had very little hope of positive change. I do not think that anyone close to me was hopeful, either. I was angry, cynical, and in a great deal of emotional pain."

For several years, Katie had thought about suicide as a solution to her unhappiness. At 13, in the presence of her parents, she reported these thoughts to a psychologist. Her parents wept, and the sight of their tears deeply affected Katie. From that point on, she never expressed her suicidal thoughts again, but they remained with her. By the time she was 16, her preoccupation with her own death had increased.

I think this was just exhaustion. I was tired of dealing with the anxiety and depression day in and day out. Soon I found myself trying to sever the few interpersonal connections that I did have, with my closest friends, with my mother, and my oldest brother. I was almost impossible to talk to. I was angry and frustrated all the time. One day I went over the edge. My mother and I had a disagreement about some unimportant little thing. I went to my bedroom where I kept a bottle of whiskey or vodka or whatever I was drinking at the time. I drank as much as I could until I could pinch myself as hard as I could and feel nothing. Then I got out a very sharp knife that I had been saving and slashed my wrist deeply. I did not feel anything but the warmth of the blood running from my wrist.

The blood poured out onto the floor next to the bed that I was lying on. The sudden thought hit me that I had failed, that this was not enough to cause my death. I got up from the bed and began to laugh. I tried to stop the bleeding with some tissues. I stayed calm and frighteningly pleasant. I walked to the kitchen and called my mother. I cannot imagine how she felt when she saw my shirt and pants covered in blood. She was amazingly calm. She asked to see the cut and said that it was not going to stop bleeding on its own and that I needed to go to the doctor immediately. I remember as the doctor shot novocaine into the cut he remarked that I must have used an anesthetic before cutting myself. I never felt the shot or the stitches.

After that, thoughts of suicide became more frequent and more real. My father asked me to promise that I would never do it again and I said I would not, but that promise meant nothing to me. I knew it was to ease his pains and fears and not mine, and my preoccupation with death continued.

Clearly, Katie's depression was outside the boundaries of normal experience because of its intensity and duration. In addition, her severe or "clinical" depression interfered substantially with her ability to function. Finally, a number of associated psychological and physical symptoms accompany clinical depression.

Because of their sometimes tragic consequences, we need to develop as full an understanding as possible of **mood disorders**. In the following sections, we describe how various emotional experiences and symptoms interrelate to produce specific mood disorders. We offer detailed descriptions of different mood disorders and examine the many criteria that define them. We discuss the relationship of anxiety and depression and the causes and treatment of mood disorders. We conclude with a discussion of suicide.

An Overview of Depression and Mania

The fundamental experiences of depression and mania contribute, either singly or together, to all the mood disorders. The most commonly diagnosed and most severe depression is called a **major depressive episode**. The *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision; *DSM-IV-TR)* criteria indicate an extremely depressed mood state that lasts at least 2 weeks and includes cognitive symptoms (such as feelings of worthlessness and indecisiveness) and disturbed physical functions (such as altered sleeping patterns, significant changes in appetite and weight, or a notable loss of energy) to the point that even the slightest activity or movement requires an overwhelming effort. The episode is typically accompanied by a general loss of interest in things and an inability to experience any pleasure from life, including interactions with family or friends or accomplishments at work or at school. Although all symptoms are important, evidence suggests that the most central indicators of a full major depressive episode are the physical changes (sometimes called *somatic* or *vegetative* symptoms) (Bech, 2009; Buchwald & Rudick-Davis, 1993; Keller et al., 1995; Kessler & Wang, 2009), along with the behavioral and emotional "shutdown," as reflected by low scores on behavioral activation scales (Kasch, Rottenberg, Arnow, & Gotlib, 2002; Rottenberg, Gross, & Gotlib, 2005). *Anhedonia* (loss of en-

ergy and inability to engage in pleasurable activities or have any "fun") is more characteristic of these severe episodes of depression than are, for example, reports of sadness or distress (Kasch et al., 2002) or the tendency to cry, which occurs equally in depressed and nondepressed individuals (mostly women in both cases) (Rottenberg, Gross, Wilhelm, Najmi, & Gotlib, 2002). This anhedonia reflects that these episodes represent a state of low positive affect and not just high negative affect (Brown & Barlow, 2009; Kasch et al., 2002). The duration of a major depressive episode, if untreated, is approximately 4 to 9 months (Hasin, Goodwin, Stinson, & Grant, 2005; Kessler & Wang, 2009).

DSM Disorder Criteria Summary
Major Depressive Episode

Features of a major depressive episode include the following:

- Depressed mood for most of the day (or irritable mood in children or adolescents)
- Markedly diminished interest or pleasure in most daily activities
- Significant weight loss when not dieting or weight gain, or significant decrease or increase in appetite
- Ongoing insomnia or hypersomnia
- Psychomotor agitation or retardation
- Fatigue or loss of energy
- Feelings of worthlessness or excessive guilt
- Diminished ability to think or concentrate
- Recurrent thoughts of death, suicide ideation, or suicide attempt
- Clinically significant distress or impairment
- Not associated with bereavement
- Persistence for longer than 2 months

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The second fundamental state in mood disorders is abnormally exaggerated elation, joy, or euphoria. In **mania**, individuals find extreme pleasure in every activity; some patients compare their daily experience of mania to a continuous sexual orgasm. They become extraordinarily active (hyperactive), require little sleep, and may develop grandiose plans with the belief that they can accomplish anything they desire. A proposal for *DSM-5* is to highlight this feature by adding "persistently increased activity or energy" to the "A" criteria. Speech is typically rapid and may become incoherent because the individual is attempting to express so many exciting ideas at once; this feature is typically referred to as *flight of ideas*.

DSM-IV-TR also defines a **hypomanic episode**, a less severe version of a manic episode that does not cause marked impairment in social or occupational functioning and need only last 4 days rather than a full week. (*Hypo* means "below"; thus the episode is below the level of a manic episode.) A hypomanic episode is not in itself necessarily problematic, but its presence does contribute to the definition of several mood disorders.

The Structure of Mood Disorders

Individuals who experience either depression or mania are said to have a *unipolar mood disorder* because their mood remains at one "pole" of the usual depression–mania continuum. Mania by itself (unipolar mania) does occur (Bech, 2009; Solomon et al., 2003) but is rare because most people with a unipolar mood disorder go on to develop depression eventually. Someone who alternates between depression and mania is said to have a bipolar mood disorder traveling from one "pole" of the depression–elation continuum to the other and back again. However, this label is somewhat misleading because depression and elation may not be at exactly opposite ends of the same mood state; although related, they are often relatively independent. An individual can experience manic symptoms but feel depressed or anxious at the same time. This combination is called a **dysphoric manic episode** or a **mixed manic episode** (Angst, 2009; Angst & Sellaro, 2000; Cassidy, Forest, Murry, & Carroll, 1998; Hantouche, Akiskal, Azorin, Chatenet-Duchene, & Lancrenon, 2006). The patient usually experiences the symptoms of mania as being out of control or dangerous and becomes anxious or depressed about this uncontrollability. Research suggests that manic episodes are characterized by dysphoric (anxious or depressive) features more commonly than was thought, and dysphoria can be severe (Cassidy et al., 1998). In one study, 30% of 1,090 patients hospitalized for acute mania had mixed episodes (Hantouche et al., 2006). In a more recent, carefully constructed study of more than 4,000 patients, as many as two thirds of patients with bipolar depressed episodes also had manic symptoms, most often racing thoughts (flight of ideas), distractibility, and agitation. These patients were also more severely impaired (Goldberg et al., 2009) than those without concurrent depression and manic symptoms. The rare individual who suffers from manic episodes alone also meets criteria for bipolar mood disorder because experience shows that

mood disorders One of a group of disorders involving severe and enduring disturbances in emotionality ranging from elation to severe depression.
major depressive episode Most common and severe experience of depression, including feelings of worthlessness, disturbances in bodily activities such as sleep, loss of interest, and inability to experience pleasure, persisting at least 2 weeks.
mania Period of abnormally excessive elation or euphoria associated with some mood disorders.
hypomanic episode Less severe and less disruptive version of a manic episode that is one of the criteria for several mood disorders.
mixed manic episode or dysphoric manic episode Condition in which the individual experiences both elation and depression or anxiety at the same time. Also known as *dysphoric manic episode.*

Major Depressive Disorder: Barbara

"I've been sad, depressed most of my life. . . . I had a headache in high school for a year and a half. . . . There have been different periods in my life when I wanted to end it all. . . . I hate me, I really hate me. I hate the way I look, I hate the way I feel. I hate the way I talk to people. . . . I do everything wrong. . . . I feel really hopeless."

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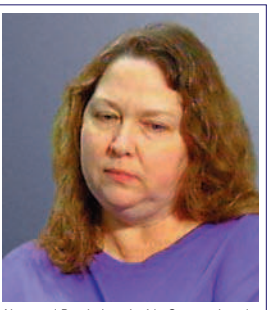

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most of these individuals can be expected to become depressed at a later time (Goodwin & Jamison, 2007; Miklowitz & Johnson, 2006). It is likely that in *DSM-5* the term "mixed episode" will be eliminated in favor of specifying whether a predominantly manic or predominantly depressive episode is present and then noting "with mixed features" to be more precise (American Psychiatric Association, 2010b).

Depressive Disorders

DSM-IV-TR describes several types of depressive disorders. These disorders differ from one another in the frequency with which depressive symptoms occur and the severity of the symptoms.

Clinical Descriptions

The most easily recognized mood disorder is **major depressive disorder**, **single episode**, defined by the absence of manic or hypomanic episodes before or during the disorder. An occurrence of just one isolated depressive episode in a lifetime is now known to be relatively rare (Angst, 2009; Eaton et al., 2008; Judd, 1997, 2000; Kessler & Wang, 2009).

If two or more major depressive episodes occurred and were separated by at least 2 months during which the individual was not depressed, **major depressive disorder**, **recurrent**, is diagnosed. Otherwise, the criteria for major depressive disorder, single episode are the same. Recurrence is important in predicting the future course of the disorder and in choosing appropriate treatments. From 35% to 85% of people with single-episode occurrences of major depressive disorder later experience a second episode and thus meet criteria for major depressive disorder, recurrent (Angst, 2009; Eaton et al., 2008; Judd, 1997, 2000), based on follow-ups as long as 23 years (Eaton et al., 2008). In the first year following an episode, the risk of recurrence is 20%, but it increases as high as 40% in the second year (Boland & Keller, 2009). Because of this finding and others reviewed later, clinical scientists have recently concluded that unipolar depression is often a chronic condition that waxes and wanes over time but seldom disappears. The median lifetime number of major depressive episodes is 4 to 7; in one large sample, 25% experienced six or more episodes (Angst, 1988, 2009; Angst & Preizig, 1996; Kessler & Wang, 2009). The median duration of recurrent major depressive episodes is 4 to 5 months (Boland & Keller, 2009; Kessler et al., 2003), somewhat shorter than the average length of the first episode.

On the basis of these criteria, how would you diagnose Katie? Katie suffered from severely depressed mood, feelings of worthlessness, difficulty concentrating, recurrent thoughts of death, sleep difficulties, and loss of energy. She clearly met the criteria for major depressive disorder, recurrent. Katie's depressive episodes were severe when they occurred, but she tended to cycle in and out of them.

Dysthymic disorder shares many of the symptoms of major depressive disorder but differs in its course. The symptoms are somewhat milder but remain relatively unchanged over long periods, sometimes 20 or 30 years or more (Angst, 2009; Klein, 2008; Klein, Schwartz, Rose, & Leader, 2000; Klein, Shankman, & Rose, 2006).

Dysthymic disorder is defined as a persistently depressed mood that continues at least 2 years, during which the patient cannot be symptom free for more than 2 months at a time. Dysthymic disorder differs from a major depressive episode only in the severity, chronicity, and number of its symptoms, which are milder and fewer but last longer. In a 10-year prospective follow-up study described later, 22% of people suffering from dysthymia eventually experienced a major depressive episode (Klein et al., 2006).

DSM Disorder Criteria Summary

Manic Episode

Features of a manic episode include the following:

- A distinct period of abnormally and persistently elevated, expansive, or irritable mood lasting at least 1 week
- Significant degree of at least three of the following: inflated self-esteem, decreased need for sleep, excessive talkativeness, flight of ideas or sense that thoughts are racing, easy distractibility, increase in goal-directed activity or psychomotor agitation, excessive involvement in pleasurable but risky behaviors
- Mood disturbance is severe enough to cause impairment in normal functioning or requires hospitalization, or there are psychotic features
- Symptoms are not caused by the direct physiological effects of a substance or a general medical condition

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Double Depression

Recently, individuals have been studied who suffer from both major depressive episodes and dysthymic disorder and who are therefore said to have **double depression**. Typically, dysthymic disorder develops first, perhaps at an early age, and then one or more major depressive episodes occur later (Boland & Keller, 2009; Klein et al., 2006). Identifying this particular pattern is important because it is associated with severe psychopathology and a problematic future course (Boland & Keller, 2009; Klein et al., 2006). For example, Keller, Lavori, Endicott, Coryell, and Klerman (1983) found that 61% of patients with double depression had not recovered from the underlying dysthymic disorder 2 years after follow-up. The investigators also found that patients who had recovered from the superimposed major depressive episode experienced high rates of relapse and recurrence. Consider the case of Jack.

Jack | A Life Kept Down

Jack was a 49-year-old divorced white man who lived at his mother's home with his 10-year-old son. He complained of chronic depression, saying he finally realized he needed help. Jack reported that he had been a pessimist and a worrier for much of his adult life. He consistently felt kind of down and depressed and did not have much fun. He had difficulty making decisions, was generally pessimistic about the future, and thought little of himself. During the past 20 years, the longest period he could remember in which his mood was "normal" or less depressed lasted only 4 or 5 days.

Despite his difficulties, Jack had finished college and obtained a master's degree in public administration. People told him his future was bright and he would be highly valued in state government. Jack did not think so. He took a job as a low-level clerk in a state agency, thinking he could always work his way up. He never did, remaining at the same desk for 20 years.

Jack's wife, fed up with his continued pessimism, lack of self-confidence, and relative inability to enjoy day-to-day events, became discouraged and divorced him. Jack moved in with his mother so that she could help care for his son and share expenses.

About 5 years before coming to the clinic, Jack had experienced a bout of depression worse than anything he had previously known. His self-esteem went from low to nonexistent. From indecisiveness, he became unable to decide anything. He was exhausted all the time and felt as if lead had filled his arms and legs, making it difficult even to move. He became unable to complete projects or to meet deadlines. Seeing no hope, he began to consider suicide. After tolerating a listless performance for years from someone they had expected to rise through the ranks, Jack's employers finally fired him.

After about 6 months, the major depressive episode resolved and Jack returned to his chronic but milder state of depression. He could get out of bed and accomplish some things, although he still doubted his own abilities. However, he was unable to obtain another job. After several years of waiting for something to turn up, he realized he was unable to solve his own problems and that without help his depression would continue. After a thorough assessment, we determined that Jack suffered from a classic case of double depression.

DSM Disorder Criteria Summary
Dysthymic Disorder

Features of dysthymic disorder include the following:

- Depressed mood for most of the day, on most days, for at least 2 years (or at least 1 year in children and adolescents)
- The presence, while depressed, of at least two of the following: poor appetite or overeating, insomnia or hypersomnia, low energy or fatigue, low self-esteem, poor concentration or difficulty making decisions, feelings of hopelessness
- During the 2 years or more of disturbance, the person has not been without the symptoms for more than 2 months at a time
- No major depressive episode has been present during this period
- No manic episode has occurred, and criteria have not been met for cyclothymic disorder
- The symptoms are not caused by the direct physiological effects of a substance or a medical condition
- Clinically significant distress or impairment of functioning

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major depressive disorder, single or recurrent episode Mood disorder involving one major depressive episode; mood disorder involving multiple (separated by at least 2 months without depression) major depressive episodes.
dysthymic disorder Mood disorder involving persistently depressed mood, with low self-esteem, withdrawal, pessimism, or despair, present for at least 2 years, with no absence of symptoms for more than 2 months.
double depression Severe mood disorder typified by major depressive episodes superimposed over a background of dysthymic disorder.

Onset and Duration

Generally the risk for developing major depression is fairly low until the early teens, when it begins to rise in a steady (linear) fashion. The mean age of onset for major depressive disorder is 30 years, based on a large (43,000) and representative sample of the population of the United States, but 10% of all people who develop major depression are 55 or older when they have their first episode (Hasin et al., 2005). An alarming finding is that the incidence of depression and consequent suicide seem to be steadily increasing. Kessler and colleagues (2003) compared four age groups and found that fully 25% of people 18 to 29 years had already experienced major depression, a rate far higher than the rate for older groups when they were that age. Recent evidence suggests that this trend is beginning to level off (Hasin et al., 2005).

As we noted previously, the length of depressive episodes is variable, with some lasting as little as 2 weeks; in more severe cases, an episode might last for several years, with the typical duration of the first episode being 4 to 9 months if untreated (Angst, 2009; Boland & Keller, 2009; Hasin et al., 2005; Kessler et al., 2003). Although 9 months is a long time to suffer with a severe depressive episode, evidence indicates that even in the most severe cases, the probability of remission of the episode within 1 year approaches 90% (Kessler & Wang, 2009). Even in those severe cases in which the episode lasts 5 years or longer, 38% can be expected to eventually recover (Mueller et al., 1996). Occasionally, however, episodes may not entirely clear up, leaving some residual symptoms. In this case, the likelihood of a subsequent episode with another incomplete recovery is much higher (Boland & Keller, 2009). Knowing this is important to treatment planning because treatment should be continued much longer in these cases.

Recent evidence also identifies important subtypes of dysthymic disorder, mostly based on when symptoms began. Although the typical age of onset has been estimated to be in the early 20s, Klein, Taylor, Dickstein, and Harding (1988) found that onset before 21 years of age, and often much earlier, is associated with three characteristics: (1) greater chronicity (it lasts longer), (2) relatively poor prognosis (response to treatment), and (3) stronger likelihood of the disorder running in the family of the affected individual. These findings have been replicated (Akiskal & Cassano, 1997). A greater prevalence of concurrent personality disorders has been found in patients with early-onset dysthymia than in patients with major depressive disorder (Klein, 2008; Pepper et al., 1995). These findings may account for the insidiousness of the psychopathology in early-onset dysthymia. Investigators have found a lower (0.07%) prevalence of dysthymic disorder in children compared to adults (3%–6%) (Klein et al., 2000), but symptoms tend to be stable throughout childhood (Garber, Gallerani, & Frankel, 2009). Kovacs, Akiskal, Gatsonis, and Parrone (1994) found that 76% of a sample of children with dysthymia later developed major depressive disorder.

Dysthymic disorder may last 20 to 30 years or more, although studies have reported a median duration of approximately 5 years in adults (Klein et al., 2006) and 4 years in children (Kovacs et al., 1994). Klein and colleagues (2006), in the study mentioned earlier, conducted a 10-year follow-up of 97 adults with dysthymic disorder and found that 74% had recovered at some point but 71% of those had relapsed. The whole sample of 97 patients spent approximately 60% of the 10-year follow-up period meeting full criteria for a mood disorder. This compares to 21% of a group of patients with nonchronic major depressive disorder also followed for 10 years. These findings demonstrate the chronicity of dysthymia. Even worse, patients with dysthymia were more likely to attempt suicide than a comparison group with episodes of major depressive disorder during a 5-year period. It is relatively common for major depressive episodes and dysthymic disorder to co-occur (double depression) (Boland & Keller, 2009; McCullough et al., 2000). Among those who have had dysthymia, as many as 79% have also had a major depressive episode at some point in their lives.

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▲ Queen Victoria remained in such deep mourning for her husband, Prince Albert, that she was unable to perform as monarch for several years after his death. Singer Alicia Keys has spoken of her struggles with depression.

From Grief to Depression

If someone you love has died—particularly if the death was unexpected and the person was a member of your immediate family—you may, after your initial reaction to the trauma, have experienced most of the symptoms of a major depressive episode: anxiety, emotional numbness, and denial (Kendler, Myers, & Zisook, 2008). The frequency of severe depression following the death of a loved one is so high (approximately 62%) that mental health professionals at present do not consider it a disorder unless severe symptoms appear, such as psychotic features or suicidal ideation, or the less alarming symptoms last longer than 6 months (Maciejewski, Zhang, Block, & Prigerson, 2007). Some grieving individuals require immediate treatment because they are so incapacitated by their symptoms (for example, severe weight loss or no energy) that they cannot function.

We must confront death and process it emotionally. All religions and cultures have rituals, such as funerals and burial ceremonies, to help us work through our losses with the support and love of our relatives and friends (Bonanno & Kaltman, 1999; Shear, 2006). Usually the natural grieving process has peaked within the first 6 months, although some people grieve for a year or longer (Clayton & Darvish, 1979; Currier, Neimeyer, & Berman, 2008; Maciejewski et al., 2007). Grief often recurs at significant anniversaries, such as the birthday of the loved one, holidays, and other meaningful occasions, including the anniversary of the death. Mental health professionals are concerned when someone does not grieve after a death because grieving is our natural way of confronting and handling loss. When grief lasts beyond typical time, mental health professionals again become concerned (Neimeyer & Currier, 2009). After a year or so, the chance of recovering from severe grief without treatment is considerably reduced, and for approximately 10% to 20% of bereaved individuals (Bonanno, 2006; Jacobs, 1993; Middleton, Burnett, Raphael, & Martinek, 1996), a normal process becomes a disorder. At this stage, suicidal thoughts increase substantially (Stroebe, Stroebe, & Abakoumkin, 2005).

Many of the psychological and social factors related to mood disorders in general, including a history of past depressive episodes (Horowitz et al., 1997; Jacobs et al., 1989), also predict the development of a typical grief response into a **pathological or impacted grief reaction**, although this reaction can develop without a preexisting depressed state (Bonanno, Wortman, & Nesse, 2004). In children and young adults, the sudden loss of a parent makes them particularly vulnerable to severe depression beyond the normal time for grieving, suggesting the need for immediate intervention (Brent, Melhem, Donohoe, & Walker, 2009). Particularly prominent symptoms include intrusive memories and distressingly strong yearnings for the loved one and avoiding people or places that are reminders of the loved one (Horowitz et al., 1997; Lichtenthal, Cruess, & Prigerson, 2004; Shear, 2006). Although a recent analysis suggests that the similarities to major depression outweigh the differences (Kendler et al., 2008), brain-imaging studies indicate that areas of the brain associated with close relationships and attachment are active in grieving people, in addition to areas of the brain associated with more general emotional responding (Gündel, O'Connor, Littrell, Fort, & Lane, 2003).

In cases of long-lasting grief, the rituals intended to help us face and accept death were ineffective. As with victims suffering from posttraumatic stress, one therapeutic approach is to help grieving individuals reexperience the trauma under close supervision. Usually, the grieving person is encouraged to talk about the loved one, the death, and the meaning of the loss while experiencing all the associated emotions, until that person can come to terms with reality. This would include incorporating positive emotions associated with memories of the relationship into the intense negative emotions connected with the loss and arriving at the position that it is possible to cope with the pain and life will go on (Bonanno & Kaltman, 1999; Currier et al., 2008). Several studies have demonstrated that this approach is successful compared with alternative psychological treatments that also focus on grief and loss (Neimeyer & Currier, 2009; Shear, Frank, Houck, & Reynolds, 2005).

Bipolar Disorders

The key identifying feature of bipolar disorders is the tendency of manic episodes to alternate with major depressive episodes in an unending roller-coaster ride from the peaks of elation to the depths of despair. Beyond that, bipolar disorders are parallel in many ways to depressive disorders. For example, a manic episode might occur only once or repeatedly. Consider the case of Jane.

Jane: Funny, Smart, and Desperate

Jane was the wife of a well-known surgeon and the loving mother of three children. They lived in an old country house on the edge of town with plenty of room for the family and pets. Jane was nearly 50 years old. The older children had moved out; the youngest son, 16-year-old Mike, was having substantial

hallucinations Psychotic symptom of perceptual disturbance in which something is seen, heard, or otherwise sensed although it is not actually present.
delusions Psychotic symptom involving disorder of thought content and presence of strong beliefs that are misrepresentations of reality.
catalepsy Motor movement disturbance seen in people with some psychoses and mood disorders in which body postures can be "sculpted" to remain fixed for long periods.
seasonal affective disorder (SAD) Mood disorder involving a cycling of episodes corresponding to the seasons of the year, typically with depression occurring during the winter.
pathological or impacted grief reaction Extreme reaction to the death of a loved one that involves psychotic features, suicidal ideation, or severe loss of weight or energy or that persists more than 2 months. Also known as an *impacted grief reaction.*

academic difficulties in school and seemed anxious. Jane brought Mike to the clinic to find out why he was having problems.

As they entered the office, I observed that Jane was well-dressed, neat, vivacious, and personable; she had a bounce to her step. She began talking about her wonderful and successful family before she and Mike even reached their seats. Mike, by contrast, was quiet and reserved. He seemed resigned and perhaps relieved that he would have to say little during the session. By the time Jane sat down, she had mentioned the personal virtues and material achievement of her husband, and the brilliance and beauty of one of her older children, and she was proceeding to describe the second child. But before she finished she noticed a book on anxiety disorders and, having read voraciously on the subject, began a litany of various anxiety-related problems that might be troubling Mike.

In the meantime, Mike sat in the corner with a small smile on his lips that seemed to be masking considerable distress and uncertainty over what his mother might do next. It became clear as the interview progressed that Mike suffered from obsessive-compulsive disorder, which disturbed his concentration both in and out of school. He was failing all his courses.

It also became clear that Jane herself was in the midst of a hypomanic episode, evident in her unbridled enthusiasm, grandiose perceptions, "uninterruptable" speech, and report that she needed little sleep these days. She was also easily distracted, as when she quickly switched from describing her children to the book on the table. When asked about her own psychological state, Jane readily admitted that she was a "manic depressive" (the old name for bipolar disorder) and that she alternated rather rapidly between feeling on top of the world and feeling depressed; she was taking medication for her condition. I immediately wondered if Mike's obsessions had anything to do with his mother's condition.

Mike was treated intensively for his obsessions and compulsions but made little progress. He said that life at home was difficult when his mother was depressed. She sometimes went to bed and stayed there for 3 weeks. During this time, she seemed be in a depressive stupor, essentially unable to move for days. It was up to the children to care for themselves and their mother, who they fed by hand. Because the older children had now left home, much of the burden had fallen on Mike. Jane's profound depressive episodes would remit after about 3 weeks, and she would immediately enter a hypomanic episode that might last several months or more. During hypomania, Jane was mostly funny, entertaining, and a delight to be with—if you could get a word in edgewise. Consultation with her therapist, an expert in the area, revealed that he had prescribed a number of medications but was so far unable to bring her mood swings under control.

Jane suffered from **bipolar II disorder**, in which major depressive episodes alternate with hypomanic episodes rather than full manic episodes. As we noted earlier, hypomanic episodes are less severe. Although she was noticeably "up," Jane functioned pretty well while in this mood state. The criteria for **bipolar I disorder** are the same, except the individual experiences a full manic episode. As in the criteria set for depressive disorder, for the manic episodes to be considered separate, there must be a symptom-free period of at least 2 months between them. Otherwise, one episode is seen as a continuation of the last.

The case of Billy illustrates a full manic episode. This individual was first encountered when he was admitted to a hospital.

DSM Disorder Criteria Summary
Bipolar II Disorder

Features of bipolar II disorder include the following:

- Presence (or history) of one or more major depressive episodes
- Presence (or history) of at least one hypomanic episode
- No history of a full manic episode or a mixed episode
- Mood symptoms are not better accounted for by schizoaffective disorder or superimposed on another disorder such as schizophrenia
- Clinically significant distress or impairment of functioning

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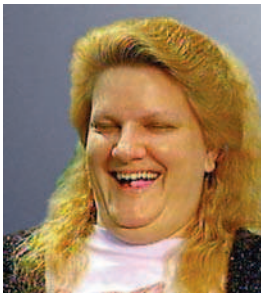
Billy The World's Best at Everything

Before Billy reached the ward, you could hear him laughing and carrying on in a deep voice; it sounded like he was having a wonderful time. As the nurse brought Billy down the hall to introduce him to the staff, he spied the Ping-Pong table. Loudly, he exclaimed, "Ping-Pong! I love Ping-Pong! I have only played twice but that is what I am going to do while I am here; I am going to become the world's greatest Ping-Pong player! And that table is gorgeous! I am going to start work on that table immediately and make it the finest Ping-Pong table in the world. I am going to sand it down, take it apart, and rebuild it until it gleams and every angle is perfect!" Billy soon went on to something else that absorbed his attention.

The previous week, Billy had emptied his bank account, taken his credit cards and those of his elderly parents with whom he was living, and bought every piece of fancy stereo equipment he could find. He thought that he would set up the best sound studio in the city and make millions of dollars by renting it to people who would come from far and wide. This episode had precipitated his admission to the hospital.

During manic or hypomanic phases, patients often deny they have a problem, which was characteristic of Billy. Even after spending inordinate amounts of money or making foolish business decisions, these individuals, particularly if they are in the midst of a full manic episode, are so wrapped up in their enthusiasm and expansiveness that their behavior seems reasonable to them. The high during a manic state is so pleasurable that people may stop taking their medication during periods of distress or discouragement in an attempt to bring on a manic state again.



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Bipolar Disorder: Mary

"Whoo, whoo, whoo—on top of the world! . . . It's going to be one great day! . . . I'm incognito for the Lord God Almighty. I'm working for him. I have been for years. I'm a spy. My mission is to fight for the American way . . . the Statue of Liberty. . . . I can bring up the wind, I can bring the rain, I can bring the sunshine, I can do lots of things. . . . I love the outdoors."

Go to Psychology CourseMate at www.cengagebrain.com to watch this video.

Returning to the case of Jane, we continued to treat Jane's son Mike for several months. We made little progress before the school year ended. Because Mike was doing so poorly, the school administrators informed his parents that he would not be accepted back the next year. Mike and his parents wisely decided it might be a good idea if he got away from the house and did something different for a while, and he began working and living at a ski and tennis resort. Several months later, his father called to tell us that Mike's obsessions and compulsions had completely lifted since he'd been away from home. The father thought Mike should continue living at the resort, where he had entered school and was doing better academically. He now agreed with our previous assessment that Mike's condition might be related to his relationship with his mother. Several years later, we heard that Jane, in a depressive stupor, had killed herself, an all-too-tragic outcome in bipolar disorder.

A milder but more chronic version of bipolar disorder called **cyclothymic disorder** is similar in many ways to dysthymic disorder (Akiskal, 2009). Like dysthymic disorder, cyclothymic disorder is a chronic alternation of mood elevation and depression that does not reach the severity of manic or major depressive episodes. Individuals with cyclothymic disorder tend to be in one mood state or the other for years with relatively few periods of neutral (or euthymic) mood. This pattern must last for at least 2 years (1 year for children and adolescents) to meet criteria for the disorder. Individuals with cyclothymic disorder alternate between the kinds of mild depressive symptoms Jack experienced during his dysthymic states and the sorts of hypomanic episodes Jane experienced. In neither case was the behavior severe enough to require hospitalization or immediate intervention. Much of the time, such individuals are just considered moody. However, the chronically fluctuating mood states are, by definition, substantial enough to interfere with functioning. Furthermore, people with cyclothymia should be treated because of their increased risk to develop the more severe bipolar I or bipolar II disorder (Akiskal, 2009; Akiskal & Pinto, 1999; Alloy & Abramson, 2001; Goodwin & Jamison, 2007; Otto & Applebaum, 2011).

Onset and Duration

The average age of onset for bipolar I disorder is from 15 to 18 and for bipolar II disorder from 19 and 22, although cases of both can begin in childhood (Angst, 2009; Judd et al., 2003; Merikangas & Pato, 2009). This is somewhat younger than the average age of onset for major depressive disorder, and bipolar disorders begin more acutely—that is, they develop more suddenly (Angst & Sellaro, 2000; Johnson, Cuellar, & Miller, 2009). About one third of the cases of bipolar disorder begin in adolescence, and the onset is often preceded by minor oscillations in mood or mild cyclothymic mood swings (Goodwin & Ghaemi, 1998; Goodwin & Jamison, 2007; Merikangas et al., 2007). The illness of between 10% and 25% of people with bipolar II disorder will progress to full bipolar I disorder (Birmaher et al., 2009; Coryell et al., 1995).

Although unipolar and bipolar disorder have been thought distinct disorders, Angst and Sellaro (2000), in reviewing some older studies, estimated the rate of depressed individuals later experiencing a full manic episode at closer to 25%. And Cassano and colleagues (2004), along with Akiskal (2006), found that as many as 67.5% of patients with unipolar depression experienced some manic symptoms. These studies raise questions about the true distinction between unipolar depression and bipolar disorder and suggest they may be on a continuum (called a "spectrum" in psychopathology) (Johnson et al., 2009; Merikangas & Pato, 2009).

bipolar II disorder Alternation of major depressive episodes with hypomanic episodes (not full manic episodes).
bipolar I disorder Alternation of major depressive episodes with full manic episodes.
cyclothymic disorder Chronic (at least 2 years) mood disorder characterized by alternating mood elevation and depression levels that are not as severe as manic or major depressive episodes.

It is relatively rare for someone to develop bipolar disorder after the age of 40. Once it does appear, the course is chronic—that is, mania and depression alternate indefinitely. Therapy usually involves managing the disorder with ongoing drug regimens that prevent recurrence of episodes. Suicide is an all-too-common consequence of bipolar disorder, almost always occurring during depressive episodes, as it did in the case of Jane (Angst, 2009; Valtonen et al., 2007). Estimates of suicide attempts in bipolar disorder range from 12% to as high as 48% over a lifetime, and this rate is approximately 20 times higher than for individuals without bipolar disorder (Goodwin & Jamison, 2007). Rates of completed suicide are 4 times higher in people with bipolar disorder than for people with recurrent major depression (Brown, Beck, Steer, & Grisham, 2000; Miklowitz & Johnson, 2006). Even with treatment, patients with bipolar disorder tend to do poorly, with one study showing 60% of a large group experiencing poor adjustment during the first 5 years after treatment (Goldberg, Harrow, & Grossman, 1995; Goodwin et al., 2003). A more comprehensive and longer follow-up of 219 patients reported that only 16% recovered; 52% suffered from recurrent episodes, 16% had become chronically disabled, and in one study 8% had committed suicide (Angst & Sellaro, 2000); in another study with a lengthy, 40-year follow-up, 11% had committed suicide (Angst, Angst, Gerber-Werder, & Gamma, 2005).

In typical cases, cyclothymia is chronic and lifelong. In about one third to one half of patients, cyclothymic mood swings develop into full-blown bipolar disorder (Kochman et al., 2005). In one sample of cyclothymic patients, 60% were female and the age of onset was often during the teenage years or before, with some data suggesting the most common age of onset to be 12 to 14 years (Goodwin & Jamison, 2007). The disorder is often not recognized, and sufferers are thought to be high-strung, explosive, moody, or hyperactive (Akiskal, 2009; Biederman et al., 2000; Goodwin & Jamison, 2007). One subtype of cyclothymia is based on the predominance of mild depressive symptoms, one on the predominance of hypomanic symptoms, and another on an equal distribution of both.

DSM Disorder Criteria Summary
Cyclothymic Disorder

Features of cyclothymic disorder include the following:

- For at least 2 years, numerous periods with hypomanic symptoms and numerous periods with depressive symptoms that do not meet the criteria for a major depressive episode
- Since onset, the person has not been without the symptoms for more than 2 months at a time
- No major depressive episode, manic episode, or mixed episode has been present during the first 2 years of the disturbance
- Mood symptoms are not better accounted for by schizoaffective disorder or superimposed on another disorder such as schizophrenia
- The symptoms are not caused by the physiological effects of a substance or a general medical condition
- Clinically significant distress or impairment of functioning

Source: Based on *DSM-IV-TR.* Reprinted with permission from *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text revision). © 2000 American Psychiatric Association.

Concept Check 6.1

Match each description or case by choosing its corresponding disorder: (a) mania, (b) double depression, (c) dysthymic disorder, (d) major depressive episode, and (e) bipolar I disorder.

1. Last week, as he does about every 3 months, Ryan went out with his friends, buying rounds of drinks, socializing until early morning, and feeling on top of the world. Today Ryan will not even get out of bed to go to work, see his friends, or even turn on the lights. _____
2. Feeling certain he would win the lottery, Charles went on an all-night shopping spree, maxing out all his credit cards without a worry. We know he's done this several times, feeling abnormally extreme elation, joy, and euphoria. _____
3. Ayana has had some mood disorder problems in the past, although some days she's better than others. Many days it seems like she has fallen into a rut. Although she manages to get by, she has trouble making decisions because she doesn't trust herself. _____
4. For the past few weeks, Jennifer has been sleeping a lot. She feels worthless, can't get up the energy to leave the house, and has lost a lot of weight. Her problem is the most common and extreme mood disorder. _____
5. Sanchez is always down and a bit blue, but occasionally he becomes so depressed that nothing pleases him. _____

Prevalence of Mood Disorders

› **How does the prevalence of mood disorders vary across a life span?**

Several large epidemiological studies estimating the prevalence of mood disorder have been carried out in recent years (Kessler et al., 1994; Kessler & Wang, 2009; Merikangas & Pato, 2009; Weissman et al., 1991). The best estimates of the worldwide prevalence of mood disorders suggest that approximately 16% of the population experience major depressive disorder over a lifetime and approximately 6% have experienced a major depressive disorder in the past year (Hasin et al., 2005; Kessler et al., 2003, 2005). For dysthymia the prevalence rates are approximately 3.5%, both for lifetime and in the past year (Kessler & Wang, 2009; Wittchen, Knäuper, & Kessler, 1994). And for bipolar disorder the estimates are 1% lifetime prevalence and 0.8% during the past year (Merikangas & Pato, 2009). The similarity of the lifetime and past-year rates for dysthymia and bipolar disorders reflects the fact that these disorders are chronic conditions that last much of one's life. Studies indicate that women are twice as likely to have mood disorders as men (Kessler, 2006; Kessler & Wang, 2009), but the imbalance in prevalence between males and females is accounted for solely by major depressive disorder and dysthymia because bipolar disorders are distributed approximately equally across gender (Merikangas & Pato, 2009). Although equally prevalent, there are some sex-based differences in bipolar disorder. As noted previously, women are more likely than men to experience rapid cycling, but also to be anxious, and to be in a depressive phase rather than a manic phase (Altshuler et al., 2010).

It is interesting that the prevalence of major depressive disorder and dysthymia is significantly lower among blacks than among whites (Hasin et al., 2005; Kessler et al., 1994; Weissman et al., 1991), although, again, no differences appear in bipolar disorders. One study of major depressive disorder in a community sample of African Americans found a prevalence of 3.1% during the previous year (Brown, Ahmed, Gary, & Milburn, 1995) and another found a prevalence of 4.52% during the previous year (Hasin et al., 2005), compared with 5.53% among whites. Fair or poor health status was the major predictor of depression in African Americans. Few of these individuals received appropriate treatment, with only 11% coming in contact with a mental health professional (Brown et al., 1995). Native Americans, however, present with a significantly higher prevalence of depression (Hasin et al., 2005), although difficulties in translating the concept of depression to Native American cultures suggest this finding needs more study (Beals et al., 2005; Kleinman, 2004).

Prevalence in Children, Adolescents, and Older Adults

Estimates on the prevalence of mood disorders in children and adolescents vary widely, although more sophisticated studies are beginning to appear. The general conclusion is that depressive disorders occur less often in prepubertal children than in adults but rise dramatically in adolescence (Brent & Birmaher, 2009; Costello, Foley, & Angold, 2006; Garber & Carter, 2006; Garber et al., 2009; Rudolph, 2009). Among very young children ages 2 to 5, rates of major depression are about 1.5% and a bit lower later in childhood (Garber et al., 2009), but as many as 20% to 50% of children experience some depressive symptoms that are not frequent or severe enough to meet diagnostic criteria but are nevertheless impairing (Kessler, Avenevoli, & Reis Merikangas, 2001; Rudolph, 2009). Adolescents experience major depressive disorder about as often as adults (Rudolph, 2009). In children, the sex ratio for depressive disorders is approximately 50:50, but this changes dramatically in adolescence. Major depressive disorder in adolescents is largely a female disorder, as it is in adults, with puberty seemingly triggering this sex imbalance (Garber & Carter, 2006; Garber et al., 2009; Nolen-Hoeksema & Hilt, 2009). Of note, this sex imbalance is not evident for more mild depression.

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▲ Among adolescents, severe major depressive disorder occurs mostly in girls.

The overall prevalence of major depressive disorder for individuals older than age 65 is about half that of the general population (Blazer & Hybels, 2009; Byers, Yaffe, Covinsky, Friedman, & Bruce, 2010; Fiske, Wetherell, & Gatz, 2009; Hasin et al., 2005; Kessler et al., 2003), perhaps because stressful life events that trigger major depressive episodes decrease with age. But milder symptoms that do not meet criteria for major depressive disorder seem to be more common among the elderly (Beekman et al., 2002; Ernst & Angst, 1995; Gotlib & Nolan, 2001) and may be associated with illness and infirmity (Delano-Wood & Abeles, 2005; Roberts, Kaplan, Shema, & Strawbridge, 1997).

Bipolar disorder seems to occur at about the same rate (1%) in childhood and adolescence as in adults (Brent & Birmaher, 2009; Merikangas & Pato, 2009). However, the rates of diagnosis of bipolar disorder in clinics has increased substantially as a result of greater interest and a controversial tendency to broaden the diagnostic criteria in children to accommodate what might be developmental variations in how bipolar disorder appears, as discussed further later in this chapter (Leibenluft & Rich, 2008; Youngstrom, Youngstrom, & Starr, 2005).

Considering the chronicity and seriousness of mood disorders (Gotlib & Hammen, 2009), the prevalence in all age groups is high indeed, demonstrating a substantial impact not only on the affected individuals and their families, but also on society.

Life Span Developmental Influences on Mood Disorders

You might assume that depression requires some experience with life, that an accumulation of negative events or disappointments might create pessimism, which then leads to depression. Like many reasonable assumptions in psychopathology, this one is not uniformly correct. There is some evidence that 3-month-old babies can become depressed. Infants of depressed mothers display marked depressive behaviors (sad faces, slow movement, lack of responsiveness) even when interacting with a nondepressed adult (Garber et al., 2009; Guedeney, 2007). Whether this behavior or temperament is caused by a genetic tendency inherited from the mother, the result of early interaction patterns with a depressed mother or primary caregiver, or a combination is not yet clear.

Most investigators agree that mood disorders are fundamentally similar in children and in adults (Brent & Birmaher, 2009; Garber et al., 2009; Weiss & Garber, 2003). Therefore, no "childhood" mood disorders in *DSM-IV-TR* are specific to a developmental stage, unlike anxiety disorders. However, it also seems clear that the "look" of depression changes with age. For example, children younger than age 3 might manifest depression by sad facial expressions, irritability, fatigue, fussiness, and tantrums and by problems with eating and sleeping. In children between the ages of 9 and 12 many of these features would not occur. Also, for preschool children (6 years old and under), Luby and colleagues (2003) report the necessity of setting aside the strict 2-week duration requirement for major depression because it is normal for mood to fluctuate at this young age. Furthermore, if these children clearly have the core symptoms of sadness or irritability and anhedonia (loss of pleasure), then a total of four symptoms rather than five seems sufficient. But even these core symptoms of anhedonia, hopelessness, and excessive sleep and social withdrawal seem to change with age, typically becoming more severe (Garber & Carter, 2006; Weiss & Garber, 2003).

Looking at mania, children younger than age 9 seem to present with more irritability and emotional swings rather than classic manic states, particularly irritability (Fields & Fristad, 2009; Leibenluft & Rich, 2008), but it is also important to recognize that irritability alone is insufficient to diagnose mania because it is associated with many different types of problems in childhood (it is nonspecific to mania). "Emotional swing" or oscillating manic states that are less distinct than in adults may also be characteristic of children, as are brief or rapid-cycling manic episodes lasting only part of a day (Youngstrom, 2009).

One developmental difference between children and adolescents compared with adults concerns patterns of comorbidity. For example, children, especially boys, tend to become aggressive and even destructive during depressive or manic episodes. Therefore, childhood depression (and mania) is often associated with and sometimes misdiagnosed as attention deficit hyperactivity disorder (ADHD) or, more often, conduct disorder in which aggression and even destructive behavior are common (Fields & Fristad, 2009; Garber et al., 2009). Often conduct disorder and depression co-occur in bipolar disorder. In patients with bipolar disorder, between 60% and 90% of children and adolescents also meet criteria for ADHD (Biederman et al., 2000; Singh, DelBello, Kowatch, & Strakowski, 2006; Youngstrom, 2009). In any case, successful treatment of the underlying depression (or spontaneous recovery) may resolve the associated ADHD or conduct disorder in these patients. Adolescents with bipolar disorder may also become aggressive, impulsive, sexually provocative, and accident prone (Carlson, 1990; Keller & Wunder, 1990).

Is Bipolar Disorder in Youth Overdiagnosed?

Children and adolescents are being diagnosed with bipolar disorder at greatly increasing rates over the past several years. In fact, the rates of bipolar diagnoses in youth have doubled in outpatient clinical settings (up to 6%) and have quadrupled in U.S. community hospitals (up to 40%) (Youngstrom et al., 2005; Leibenluft & Rich, 2008). Why the increase? Many clinicians are now using much broader diagnostic criteria that would not correspond to current definitions of bipolar I or bipolar II disorder but rather fall under the relatively vague category of bipolar disorder not otherwise specified (NOS) and include children with chronic irritability, anger, aggression, hyperarousal, and frequent temper tantrums. Although these broader definitions of symptoms do display some similarities with more

classic bipolar disorder symptoms and might respond to current treatments for bipolar disorder (Biederman et al., 2005; Biederman et al., 2000), the danger is that these children are being misdiagnosed when they might better meet criteria for more classic diagnostic categories such as ADHD or conduct disorder (see Chapter 13). In that case, the very potent drug treatments for bipolar disorder with substantial side effects would pose more risks for these children than they would benefits.

Based on the large multisite study Course and Outcome of Bipolar Youth (COBY; Axelson et al., 2006; Birmaher et al., 2006), which followed 263 youths with bipolar disorder for 2 years, the American Academy of Child and Adolescent Psychiatry has made the recommendation that most children meeting these broader criteria should not be diagnosed with bipolar disorder unless they also meet more classic definitions that involve clearly defined episodes of fluctuating and extreme moods (depression or mania) (Leibenluft & Rich, 2008; McClellan, Kowatch, & Findling, 2007). Thus, youths who have clear manic episodes that nevertheless might be too short to meet current *DSM* criteria (either elevated or irritable mood that may last only 4 hours but reoccurs a number of times) could be included in the bipolar spectrum, but youths who present with chronic, severe, impairing irritability and anger that is unremitting and not episodic, and is accompanied by symptoms of hyperarousal, temper tantrums, and/or aggression could be better described as having severe mood dysregulation (SMD), rather than bipolar disorder. Instead of receiving inappropriately strong medications, this group might benefit more from the more usual treatments for ADHD and/or depression and anxiety including psychological treatments. A proposal to make changes along these lines has been made for *DSM-5*.

Whatever the presentation, mood disorders in children and adolescents are serious because of their likely consequences (Garber et al., 2009). Fergusson and Woodward (2002), in a large prospective study, identified 13% of a group of 1,265 adolescents who developed major depressive disorder between 14 and 16 years of age. Later, between ages 16 and 21, this group was significantly at risk for occurrence of major depression, anxiety disorders, nicotine dependence, suicide attempts, drug and alcohol abuse, educational underachievement, and early parenting compared with adolescents who were not depressed. Weissman and colleagues (1999) identified a group of 83 children with an onset of major depressive disorder before puberty and followed them for 10 to 15 years. Generally, there was also a poor adult outcome in this group, with high rates of suicide attempts and social impairment compared with children without major depressive disorder. Of note, these prepubertal children were more likely to develop substance abuse or other disorders as adults rather than continue with their depression, unlike adolescents with major depressive disorder. Fergusson, Horwood, Ridder, and Beautrais (2005) found that extent and severity of depressive symptoms as an adolescent predicted extent of depression and suicidal behaviors as an adult. Clearly, becoming depressed as a child or adolescent is a dangerous, threatening event to be treated immediately or prevented if possible.

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▲ Depression among the elderly is a serious problem that can be difficult to diagnose because the symptoms are often similar to those of physical illness or dementia.

Age-Based Influences on Older Adults

Only recently have we seriously considered the problem of depression in the elderly. Some studies estimate that 14% to 42% of nursing home residents may experience major depressive episodes (Djernes, 2006; Fiske et al., 2009). In one large study, depressed elderly patients between 56 and 85 years of age were followed for 6 years; approximately 80% did not remit but continued to be depressed (or cycled in and out of depression) even if their depressive symptoms were not severe enough to meet diagnostic criteria for a disorder (Beekman et al., 2002). Late-onset depressions are associated with marked sleep difficulties, hypochondriasis (anxiety focused on possibly being sick or injured in some way), and agitation (Baldwin, 2009). It can be difficult to diagnose depression in older adults because elderly people who become physically ill or begin to show signs of dementia might become depressed about it, but the signs of depression or mood disorder would be attributed to the illness or dementia and thus missed (see, for example, Blazer & Hybels, 2009; Delano-Wood & Abeles, 2005). As many as 50% of patients with Alzheimer's disease suffer from comorbid depression, which also makes life more difficult for their families (Lyketsos & Olin, 2002).

Anxiety disorders accompany depression in from one third to one half of elderly patients, particularly generalized anxiety disorder and panic disorder (Fiske et al., 2009; Lenze et al., 2000), and when they do, patients are more severely depressed. One third will also suffer from comorbid alcohol abuse (Devanand, 2002). Several studies have shown that entering menopause also increases rates of depression among women who have never previously been depressed (Cohen, Soares, Vitonis, Otto, & Harlow, 2006; Freeman, Sammel, Lin, & Nelson, 2006). This may be because of biological factors, such as hormonal changes, or

the experience of distressing physical symptoms or other life events occurring during this period. Depression can also contribute to physical disease and death in the elderly (Blazer & Hybels, 2009). Being depressed doubles the risk of death in elderly patients who have suffered a heart attack or stroke (Schulz, Drayer, & Rollman, 2002).

Wallace and O'Hara (1992), in a longitudinal study, found that elderly citizens became increasingly depressed over a 3-year period. They suggest, with some evidence, that this trend is related to increasing illness and reduced social support; in other words, as we become frailer and more alone, the psychological result is depression, which increases the probability that we will become even frailer and have even less social support. Bruce (2002) confirmed that death of a spouse, caregiving burden for an ill spouse, and loss of independence because of medical illness are among the strongest risk factors for depression in this age group. This vicious cycle is deadly because suicide rates are higher in older adults than in any other age group (Conwell, Duberstein, & Caine, 2002), although rates have been decreasing lately (Blazer & Hybels, 2009).

The earlier gender imbalance in depression reduces considerably after the age of 65. In early childhood, boys are more likely to be depressed than girls, but an overwhelming surge of depression in adolescent girls produces an imbalance in the sex ratio that is maintained until old age, when just as many women are depressed but increasing numbers of men are also affected (Fiske et al., 2009). From the perspective of the life span, this is the first time since early childhood that the sex ratio for depression is more closely balanced.

Across Cultures

We noted the strong tendency of anxiety to take somatic (physical) forms in some cultures; instead of talking about fear, panic, or general anxiety, many people describe stomachaches, chest pains or heart distress, and headaches. Much the same tendency exists across cultures for mood disorders, which is not surprising given the close relationship of anxiety and depression. Feelings of weakness or tiredness particularly characterize depression that is accompanied by mental or physical slowing or retardation (Kleinman, 2004; Ryder et al., 2008). Some cultures have their own idioms for depression; for instance, the Hopi, a Native American tribe, say they are "heartbroken" (Manson & Good, 1993).

Although somatic symptoms that characterize mood disorders seem roughly equivalent across cultures, it is difficult to compare subjective feelings. The way people think of depression may be influenced by the cultural view of the individual and the role of the individual in society (Jenkins, Kleinman, & Good, 1990; Kleinman, 2004; Ryder et al., 2008). For example, in societies that focus on the *individual* instead of the *group,* it is common to hear statements such as "I feel blue" or "I am depressed." However, in cultures where the individual is tightly integrated into the larger group, someone might say, "Our life has lost its meaning," referring to the group in which the individual resides (Manson & Good, 1993).

Weissman and colleagues (1991) looked at the lifetime prevalence of mood disorders in African American and Hispanic American ethnic groups. For each disorder, the figures are similar in the two groups (although, as noted earlier, it is somewhat lower for African Americans in major depressive disorder and dysthymia), indicating no particular difference across subcultures. However, these figures were collected on a carefully constructed sample meant to represent the whole country.

In specific locations, results can differ dramatically. Kinzie, Leung, Boehnlein, and Matsunaga (1992) used a structured interview to determine the percentage of adult members of a Native American village who met criteria for mood disorders. The lifetime prevalence for any mood disorder was 19.4% in men, 36.7% in women, and 28% overall, approximately 4 times higher than in the general population. Examined by disorder, almost all the increase is accounted for by greatly elevated rates of major depression. Findings in the same village for substance abuse are similar to the results for major depressive disorder (see Chapter 10). Hasin and colleagues (2005) found a somewhat lower overall percentage of 19.17% in a different village, which was still 1.5 times higher than the percentage found in Caucasians, a significant difference. Beals and colleagues (2005), however, reported a considerably lower prevalence in two tribes they studied, perhaps because of differences in interviewing methods or because conditions and culture can differ greatly from tribe to tribe. Still, appalling social and economic conditions on many reservations fulfill all requirements for chronic major life stress, which is so strongly related to the onset of mood disorders, particularly major depressive disorder.

Concept Check 6.2

Identify each of the following statements related to prevalence of mood disorders as either true (T) or false (F).

1. _____ Women are approximately twice as likely as men to be diagnosed with a mood disorder.
2. _____ Depression requires some life experience, indicating that babies and young children cannot experience the disorder.
3. _____ It's often difficult to diagnose depression in the elderly because its symptoms are similar to those of medical ailments or dementia.
4. _____ Somatic symptoms characterizing mood disorders are nearly equivalent across cultures.

Causes of Mood Disorders

› **What biological, psychological, and sociocultural factors contribute to the development of mood disorders?**

Psychopathologists are identifying biological, psychological, and social factors that seem strongly implicated in the etiology of mood disorders, whatever the precipitating factor. An integrative theory of the etiology of mood disorders considers the interaction of biological, psychological, and social dimensions and notes the strong relationship of anxiety and depression. Before describing these interactions, we review evidence pertaining to each contributing factor.

Biological Dimensions

Studies that would allow us to determine the genetic contribution to a particular disorder or class of disorders are complex and difficult to do. But several strategies—such as family studies and twin studies—can help us estimate this contribution.

Familial and Genetic Influences

In *family studies,* we look at the prevalence of a given disorder in the first-degree relatives of an individual known to have the disorder (the *proband*). We have found that, despite wide variability, the rate in relatives of probands with mood disorders is consistently about 2 to 3 times greater than in relatives of controls who don't have mood disorders (Gershon, 1990; Klein, Lewinsohn, Rohde, Seeley, & Durbin, 2002; Levinson, 2009). Increasing severity, recurrence of major depression, and earlier age of onset in the proband is associated with the highest rates of depression in relatives (Kendler, Gatz, Gardner, & Pedersen, 2007; Klein et al., 2002; Weissman et al., 2005).

The best evidence that genes have something to do with mood disorders comes from *twin studies,* in which we examine the frequency with which identical twins (with identical genes) have the disorder, compared to fraternal twins who share only 50% of their genes (as do all first-degree relatives). If a genetic contribution exists, the disorder should be present in identical twins to a much greater extent than in fraternal twins. A number of twin studies suggest that mood disorders are heritable (see, for example, Kendler, Neale, Kessler, Heath, & Eaves, 1993; McGuffin et al., 2003). One of the strongest studies demonstrates that an identical twin is 2 to 3 times more likely to present with a mood disorder than a fraternal twin if the first twin has a mood disorder (66.7% of identical twins compared to 18.9% of fraternal twins if the first twin has bipolar disorder; 45.6% versus 20.2% if the first twin has unipolar disorder) (McGuffin et al., 2003). But if one twin has unipolar disorder the chances of a co-twin having bipolar disorder are slim to none.

Two reports have appeared suggesting sex differences in genetic vulnerability to depression. Bierut and colleagues (1999) studied 2,662 twin pairs in the Australian twin registry and found the characteristically higher rate of depressive disorders in women. Estimates of heritability in women ranged from 36% to 44%, consistent with other studies. But estimates for men were lower and ranged from 18% to 24%. These results mostly agree with an important study of men in the United States by Lyons and colleagues (1998). The authors conclude that environmental events play a larger role in causing depression in men than in women.

Note from the studies just described that bipolar disorder confers an increased risk of developing some mood disorder in close relatives—but not necessarily bipolar disorder. This conclusion supports an assumption noted previously that bipolar disorder may simply be a more severe variant of mood disorders rather than a fundamentally different disorder. Then again, of identical twins both having (concordant for) a mood disorder, 80% are also concordant for polarity. In other words, if one identical twin is unipolar, there is an 80% chance the other twin is unipolar as opposed to bipolar. This finding suggests these disorders may be inherited separately and therefore be separate disorders after all (Nurnberger & Gershon, 1992).

McGuffin and colleagues (2003) conclude that both points are partially correct. Basically, they found that the genetic contributions to depression in both disorders are the same or similar but that the genetics of mania are distinct from depression. Thus, individuals with bipolar disorder are genetically susceptible to depression and independently genetically susceptible to mania. This hypothesis still requires further confirmation.

Although these findings do raise continuing questions about the relative contributions of psychosocial and genetic factors to mood disorders, overwhelming evidence suggests that such disorders are familial and almost certainly reflect an underlying genetic vulnerability, particularly for women. As described in some detail in Chapter 2 (see p. 15), studies are now beginning to identify a small group of genes that may confer this vulnerability, at least for some types of depression (Bradley et al., 2008; Caspi et al., 2003; Garlow, Boone, Li, Owens, & Nemeroff, 2005; Levinson, 2009). In this complex field, it is likely that many additional patterns of gene combinations will be found to contribute to varieties of depression.

In conclusion, the best estimates of genetic contributions to depression fall in the range of approximately 40% for women but seem to be significantly less for men (around 20%). Genetic contributions to bipolar disorder seem to be somewhat higher. This means that from 60% to 80% of the causes of depression can be attributed to environmental factors. Also, recent findings underscore the

enormous heterogeneity of genetic associations with any mental disorder. So these percentages (40% for women, 20% for men) may not reflect any one pattern of genetic contribution associated with specific groups of genes, but perhaps many different patterns from different groups of genes (McClellan & King, 2010). As we noted in Chapter 3, behavioral geneticists break down environmental factors into events shared by twins (experiencing the same upbringing in the same house and, perhaps, experiencing the same stressful events) and events not shared. What part of our experience causes depression? There is wide agreement that it is the unique nonshared events rather than the shared ones that interact with biological vulnerability to cause depression (Bierut et al., 1999; Plomin, DeFries, McClearn, & Rutter, 1997).

Depression and Anxiety: Same Genes?

Evidence supports the assumption of a close relationship among depression, anxiety, and panic (and other emotional disorders). For example, data from family studies indicate that the more signs and symptoms of anxiety and depression there are in a given patient, the greater the rate of anxiety, depression, or both in first-degree relatives and children (Hudson et al., 2003; Leyfer & Brown, 2011). In several important reports from a major set of data on more than 2,000 female twins, Ken Kendler and his colleagues (Kendler, Heath, Martin, & Eaves, 1987; Kendler, Neale, Kessler, Heath, & Eaves, 1992b; Kendler et al., 1995) found that the same genetic factors contribute to both anxiety and depression. Social and psychological explanations seemed to account for the factors that differentiate anxiety from depression rather than genes. These findings again suggest that, with the possible exception of mania, the biological vulnerability for mood disorders may not be specific to that disorder but may reflect a more general predisposition to anxiety or mood disorders. The specific form of the disorder would be determined by unique psychological, social, or additional biological factors (Kilpatrick et al., 2007; Rutter, 2010).

Neurotransmitter Systems

In Chapter 2, we observed that we now know that neurotransmitter systems have many subtypes and interact in many complex ways with one another and with neuromodulators (products of the endocrine system). Research implicates low levels of serotonin in the causes of mood disorders, but only in relation to other neurotransmitters, including norepinephrine and dopamine (see, for example, Spoont, 1992; Thase, 2005, 2009). Remember that the apparent primary function of serotonin is to regulate our emotional reactions. For example, we are more impulsive, and our moods swing more widely, when our levels of serotonin are low. This may be because one of the functions of serotonin is to regulate systems involving norepinephrine and dopamine. According to the "permissive" hypothesis, when serotonin levels are low, other neurotransmitters are "permitted" to range more widely, become dysregulated, and contribute to mood irregularities, including depression. A drop in norepinephrine would be one of the consequences. Mann and colleagues (1996) used sophisticated brain-imaging procedures (PET scans) to confirm impaired serotonergic transmission in patients with depression, but subsequent research suggested that this relationship holds only for more severe patients with suicidal tendencies (Mann, Brent, & Arango, 2001; Thase, 2009). Current thinking is that the balance of the various neurotransmitters and their interaction with systems of self regulation are more important than the absolute level of any one neurotransmitter (Carver, Johnson, & Joormann, 2009).

In the context of this delicate balance, there is continued interest in the role of dopamine, particularly in relationship to manic episodes, atypical depression, or depression with psychotic features (Dunlop & Nemeroff, 2007; Garlow & Nemeroff, 2003; Thase, 2009). For example, the dopamine agonist L-dopa seems to produce hypomania in bipolar patients (see, for instance, Van Praag & Korf, 1975), along with other dopamine agonists (Silverstone, 1985). Chronic stress also reduces dopamine levels and produces depressive-like behavior (Thase, 2009). But, as with other research in this area, it is difficult to pin down any relationships with certainty.

The Endocrine System

During the past several years, most attention has shifted to the endocrine system and the "stress hypothesis" of the etiology of depression (Nemeroff, 2004). This hypothesis focuses on overactivity in the hypothalamic–pituitary–adrenocortical (HPA) axis (discussed later), which produces stress hormones. Again, notice the similarity with the description of the neurobiology of anxiety in Chapter 4 (see, for example, Britton & Rauch, 2009; Charney & Drevets, 2002). Investigators became interested in the endocrine system when they noticed that patients with diseases affecting this system sometimes became depressed. For example, hypothyroidism, or Cushing's disease, which affects the adrenal cortex, leads to excessive secretion of cortisol and often to depression (and anxiety).

In Chapter 2, and again in Chapter 4, we discussed the brain circuit called the HPA axis, beginning in the hypothalamus and running through the pituitary gland, which coordinates the endocrine system (see Figure 2.10). Investigators have also discovered that neurotransmitter activity in the hypothalamus regulates the release of hormones that affect the HPA axis. These **neurohormones** are an increasingly important focus of study in psychopathology (see, for example, Garlow & Nemeroff, 2003; Nemeroff, 2004; Thase, 2009). There are thousands of neurohormones. Sorting out their relationship to antecedent neurotransmitter systems (and determining their independent effects on the central nervous system) is likely to be a complex task indeed. One of the glands influenced by the pituitary is the cortical section of the adrenal gland, which produces the stress hormone cortisol that completes the HPA axis. Cortisol is called a *stress hormone* because it is elevated during stressful life events. (We discuss this sys-

tem in more detail in Chapter 7.) For now, it is enough to know that cortisol levels are elevated in depressed patients, a finding that makes sense considering the relationship between depression and severe life stress (Bradley et al., 2008; Thase, 2009).

Recognizing that stress hormones are elevated in patients with depression (and anxiety), researchers have begun to focus on the consequences of these elevations. Preliminary findings indicate that these hormones can be harmful to neurons in that they decrease a key ingredient that keeps neurons healthy and growing. You saw in Chapter 4 on anxiety disorders that individuals experiencing heightened levels of stress hormones over a long period undergo some shrinkage of a brain structure called the *hippocampus*. The hippocampus, among other things, is responsible for keeping stress hormones in check and serves important functions in facilitating cognitive processes such as short-term memory. But the new finding, at least in animals, is that long-term overproduction of stress hormones makes the organism unable to develop new neurons (neurogenesis). Thus, some theorists suspect that the connection between high stress hormones and depression is the suppression of neurogenesis in the hippocampus (Heim, Plotsky, & Nemeroff, 2004; McEwen, 1999; Thase, 2009). Now, new evidence reveals that healthy girls at risk for developing depression because their mothers suffer from recurrent depression have reduced hippocampal volume compared to girls with nondepressed mothers (Chen, Hamilton, & Gotlib, 2010). This finding suggests that low hippocampal volume may precede and perhaps contribute to the onset of depression. Scientists have already observed that successful treatments for depression, including electroconvulsive therapy, seem to produce neurogenesis in the hippocampus, thereby reversing this process (Duman, 2004; Santarelli et al., 2003; Sapolsky, 2004).

Sleep and Circadian Rhythms

We have known for several years that sleep disturbances are a hallmark of most mood disorders. Most important, in people who are depressed, there is a significantly shorter period after falling asleep before *rapid eye movement (REM) sleep* begins. As you may remember from your introductory psychology or biology course, there are two major stages of sleep: REM sleep and non-REM sleep. When we first fall asleep, we go through several substages of progressively deeper sleep during which we achieve most of our rest. After about 90 minutes, we begin to experience REM sleep, when the brain arouses, and we begin to dream. Our eyes move rapidly back and forth under our eyelids, hence the name *rapid eye movement* sleep. As the night goes on, we have increasing amounts of REM sleep. (We discuss the process of sleep in more detail in Chapter 8.) In addition to entering REM sleep more quickly, depressed patients experience REM activity that is more intense, and the stages of deepest sleep, called *slow wave sleep,* don't occur until later if at all (Jindal et al., 2002; Kupfer, 1995; Thase, 2009). It seems that some sleep characteristics occur only while we are depressed and not at other times (Riemann, Berger, & Voderholzer, 2001; Rush et al., 1986). But other evidence suggests that, at least in more severe cases with recurrent depression, disturbances in sleep continuity,, and reduction of deep sleep, may be present even when the individual is not depressed (Kupfer, 1995; Thase, 2009).

Sleep pattern disturbances in depressed children are less pronounced than in adults, perhaps because children are very deep sleepers, illustrating once again the importance of developmental stage to psychopathology (Brent & Birmaher, 2009; Garber et al., 2009). But sleep disturbances are even more severe among depressed older adults. In fact, insomnia, frequently experienced by older adults, is a risk factor for both the onset and persistence of depression (Fiske et al., 2009; Perlis et al., 2006). In an interesting new study, researchers found that treating insomnia directly in those patients who have both insomnia and depression may enhance the effects of treatment for depression (Manber et al., 2008). Sleep disturbances also occur in bipolar patients, where they are particularly severe and are characterized not only by decreased REM latency, but also by severe insomnia and hypersomnia (excessive sleep) (Goodwin & Jamison, 2007; Harvey, 2008; Harvey, Talbot, & Gershon, 2009).

Another interesting finding is that depriving depressed patients of sleep, particularly during the second half of the night, causes temporary improvement in their condition (Giedke & Schwarzler, 2002; Thase, 2009), particularly for patients with bipolar disorder in a depressive state (Johnson et al., 2009; Harvey, 2008), although the depression returns when the patients start sleeping normally again. In any case, because sleep patterns reflect a biological rhythm, there may be a relationship among seasonal affective disorder (SAD), sleep disturbances in depressed patients, and a more general disturbance in biological rhythms (Soreca, Frank, & Kupfer, 2009). This would not be surprising if it were true, because most mammals are exquisitely sensitive to day length at the latitudes at which they live and this "biological clock" controls eating, sleeping, and weight changes. Thus, substantial disruption in circadian rhythm might be particularly problematic for some vulnerable individuals (Moore, 1999; Sohn & Lam, 2005; Soreca et al., 2009).

Psychological Dimensions

Thus far we have reviewed genetic and biological factors including findings from studies of neurotransmitters, the endocrine system, sleep and circadian rhythms, and relative activity in certain areas of the brain associated with depression. But these factors are all inextricably linked to psychological and social dimensions where scientists are

neurohormones Hormone that affects the brain and is increasingly the focus of study in psychopathology.

also discovering strong associations with depression. We now review some of these findings.

Stressful Life Events

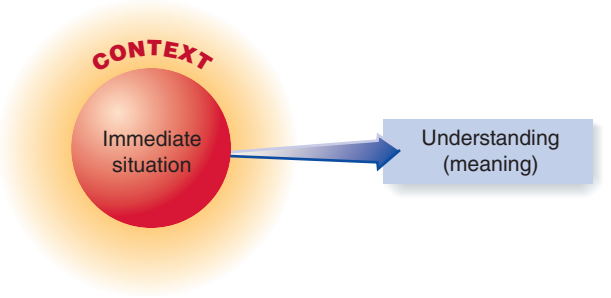
Stress and trauma are among the most striking unique contributions to the etiology of all psychological disorders. This is reflected throughout psychopathology and is evident in the wide adoption of the diathesis–stress model of psychopathology presented in Chapter 2 (and referred to throughout this book), which describes possible genetic and psychological vulnerabilities. But in seeking what activates this vulnerability (diathesis), we usually look for a stressful or traumatic life event.

Stress and Depression. You would think it would be sufficient to ask people whether anything major had happened in their lives before they developed depression or some other psychological disorder. Most people who develop depression report losing a job, getting divorced, having a child, or graduating from school and starting a career as a possible precipitating factor. But, as with most issues in the study of psychopathology, the significance of a major event is not easily discovered (Kessler, 1997; Monroe & Reid, 2009; Monroe, Slavich, & Georgiades, 2009), so most investigators have stopped simply asking patients whether something bad (or good) happened and have begun to look at the context of the event and the meaning it has for the individual.

For example, losing a job is stressful for most people, but it is far more difficult for some than others. A few people might even see it as a blessing. If you were laid off as a manager in a large corporation because of a restructuring but your wife is the president of another corporation and makes more than enough money to support the family, it might not be so bad. Furthermore, if you are an aspiring writer or artist who has not had time to pursue your art, becoming jobless might be the opportunity you have been waiting for.

Now consider losing your job if you are a single mother of two young children and living from paycheck to paycheck and, on account of a recent doctor's bill, you have to choose between paying the electric bill or buying food. The stressful life event is the same, but the context is different and transforms the significance of the event substantially. To complicate the scenario further, think for a minute about how various women in this situation might react to losing their job. One woman might decide she is a total failure and thus becomes unable to carry on and provide for her children. Another woman might realize the job loss was not her fault and take advantage of a job training program while scraping by somehow. Thus, both the context of the life event and its meaning are important. This approach to studying life events, developed by George W. Brown (1989b) and associates in England, is represented in ■ Figure 6.1.

Brown's study of life events is difficult to carry out, and the methodology is still evolving. Psychologists such as Scott Monroe and Constance Hammen (Hammen, 2005; Monroe et al., 2009; Monroe, Rohde, Seeley, & Lewinsohn, 1999; Dohrenwend & Dohrenwend, 1981) have developed



■ **Figure 6.1** Context and meaning in life stress situations. (Reprinted, with permission, from Brown, G. W., 1989b. Life events and measurement. In G. W. Brown & T. O. Harris, Eds., *Life events and illness.* New York, NY: Guilford Press, © 1989 New York, NY: Guilford Press.)

new methods. One crucial issue is the bias inherent in remembering events. If you ask people who are currently depressed what happened when they first became depressed more than 5 years ago, you will probably get different answers from those they would give if they were not currently depressed. Because current moods distort memories, many investigators have concluded that the only useful way to study stressful life events is to follow people *prospectively* to determine more accurately the precise nature of events and their relation to subsequent psychopathology.

In any case, in summarizing a large amount of research, it is clear that stressful life events are strongly related to the onset of mood disorders (Grant, Compas, Thurm, McMahon, & Gipson, 2004; Hammen, 2005; Kendler, Karkowski, & Prescott, 1999b; Kessler, 1997; Mazure, 1998; Monroe et al., 2009; Monroe & Reid, 2009). Measuring the context of events and their impact in a random sample of the population, a number of studies have found a marked relationship between severe and, in some cases, traumatic life events and the onset of depression (Brown, 1989a; Brown, Harris, & Hepworth, 1994; Kendler et al., 1999b; Mazure, 1998). Severe events precede all types of depression except, perhaps, for a small group of patients with melancholic or psychotic features who are experiencing subsequent episodes where depression emerges in the absence of life events (Brown et al., 1994). Major life stress is a somewhat stronger predictor for initial episodes of depression compared to recurrent episodes (Lewinsohn, Allen, Seeley, & Gotlib, 1999). In addition, for people with recurrent depression, the clear occurrence of a severe life stress before or early in the latest episode predicts a poorer response to treatment and a longer time before remission (Monroe et al., 2009; Monroe, Kupfer, & Frank, 1992) and a greater likelihood of recurrence (Monroe et al., 2009; Monroe, Roberts, Kupfer, & Frank, 1996).

Although the context and meaning are often more important than the exact nature of the event itself, some events are particularly likely to lead to depression. One of them is the breakup of a relationship, which is difficult for

both adolescents (Monroe, Rohde, Seeley, & Lewinsohn, 1999) and adults (Kendler, Hettema, Butera, Gardner, & Prescott, 2003). Kendler and colleagues (2003) demonstrated in an elegant twin study that if one twin experienced a loss, such as the death of a loved one, that twin was 10 times more likely to become depressed than the twin who didn't experience the loss. But if one twin is also humiliated by the loss, as when, for example, a boyfriend or husband leaves the twin for a best friend and the twin still sees them all the time, then that twin would be 20 times more likely to get depressed than a twin with the same genes who didn't experience the event. Scientists have confirmed that humiliation, loss, and social rejection are the most potent stressful life events likely to lead to depression (Monroe et al., 2009).

Clearly there is a strong relationship between stress and depression, and scientists are discovering that the cause-and-effect connection between the two might go both ways. Remember in Chapter 2 where we noted that our genetic endowment might increase the probability that we will experience stressful life events? We referred to this as the *reciprocal gene–environment model* (Saudino, Pedersen, Lichenstein, McClearn, & Plomin, 1997). One example would be people who tend to seek difficult relationships because of genetically based personality characteristics that then lead to depression. Kendler and colleagues (1999a) report that about one third of the association between stressful life events and depression is not the usual arrangement where stress triggers depression but rather individuals vulnerable to depression who are placing themselves in high-risk stressful environments, such as difficult relationships or other risky situations where bad outcomes are common. What is important about the reciprocal model is that it can happen both ways in the same individual; stress triggers depression, and depressed individuals create or seek stressful events. It is interesting that, if you ask mothers, they tend to say their depressed adolescents created the problem, but adolescents blame the stressful event itself (Carter, Garber, Cielsa, & Cole, 2006). According to the reciprocal model, the truth lies somewhere between these two views.

Stress and Bipolar Disorder. The relationship of stressful events to the onset of episodes in bipolar disorder is also strong (Alloy & Abramson, 2010; Ellicott, 1988; Goodwin & Jamison, 2007; Johnson, Gruber, & Eisner, 2007; Johnson et al., 2008; Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999). However, several issues may be particularly relevant to the causes of bipolar disorders (Goodwin & Ghaemi, 1998). First, typically negative stressful life events trigger depression, but a somewhat different more positive set of stressful life events seems to trigger mania (Johnson et al., 2008). Experience associated with striving to achieve important goals, such as getting accepted into graduate school, obtaining a new job or promotion, or getting married, trigger mania in vulnerable individuals. Second, stress seems to initially trigger mania and depression, but as the disorder progresses, these episodes seem to develop a life of their own. In other words, once the cycle begins, a psychological or pathophysiological process takes over and ensures the disorder will continue (see, for example, Post, 1992; Post et al., 1989). Third, some precipitants of manic episodes seem related to loss of sleep, as in the postpartum period (Goodwin & Jamison, 2007; Harvey, 2008; Soreca et al., 2009) or as a result of jet lag—that is, disturbed circadian rhythms. In most cases of bipolar disorder, nevertheless, stressful life events are substantially indicated not only in provoking relapse, but also in preventing recovery (Alloy, Abramson, Urosevic, Bender, & Wagner, 2009; Johnson & Miller, 1997).

Finally, although almost everyone who develops a mood disorder has experienced a significant stressful event, most people who experience such events do not develop mood disorders. Although the data are not yet as precise as we would like, somewhere between 20% and 50% of individuals who experience severe events develop mood disorders. Thus, between 50% and 80% of individuals do *not* develop mood disorders or, presumably, any other psychological disorder. Again, data strongly support the interaction of stressful life events with some kind of vulnerability: genetic; psychological; or, more likely, a combination of the two influences (Barlow, 2002; Hankin & Abramson, 2001; Kendler, Kuhn, Vittum, Prescott, & Riley, 2005; Thase, 2009).

Given a genetic vulnerability (diathesis) and a severe life event (stress), what happens then? Research has isolated a number of psychological and biological processes. To illustrate one, let's return to Katie. Her stressful life event was attending a new school. Katie's feeling of loss of control leads to another important psychological factor in depression: learned helplessness.

Katie | No Easy Transitions

I was a serious and sensitive 11 year old at the edge of puberty and at the edge of an adventure that many teens and preteens embark on—the transition from elementary to junior high school. A new school, new people, new responsibilities, new pressures. Academically, I was a good student up to this point but I didn't feel good about myself and generally lacked self-confidence.

Katie began to experience severe anxiety reactions. Then she became quite ill with the flu. After recovering and attempting to return to school, Katie discovered that her anxieties were worse than ever. More important, she began to feel she was losing control.

As I look back I can identify events that precipitated my anxieties and fears, but then everything seemed to happen suddenly and without cause. I was reacting emotionally and physically in a way that I didn't understand. I felt out of control of my emotions and body. Day after day I wished, as a child does, that whatever was happening to me would magically end.

Learned Helplessness

To review our discussion in Chapter 2, Martin Seligman discovered that dogs and rats have an interesting emotional reaction to events over which they have no control. If rats receive occasional shocks, they can function reasonably well as long as they can cope with the shocks by doing something to avoid them, such as pressing a lever. But if they learn that nothing they do helps them avoid the shocks, they eventually become helpless, give up, and manifest an animal equivalent of depression (Seligman, 1975).

Do humans react the same way? Seligman suggests we seem to but only under one important condition: People become anxious and depressed when they decide that they have no control over the stress in their lives (Abramson, Seligman, & Teasdale, 1978; Miller & Norman, 1979). These findings evolved into an important model called the **learned helplessness theory of depression**. Often overlooked is Seligman's point that anxiety is the first response to a stressful situation. Depression may follow marked hopelessness about coping with the difficult life events (Barlow, 1988, 2002). The depressive attributional style is (1) *internal,* in that the individual attributes negative events to personal failings ("it is all my fault"); (2) *stable,* in that, even after a particular negative event passes, the attribution that "additional bad things will always be my fault" remains; and (3) *global,* in that the attributions extend across a variety of issues. Research continues on this interesting concept, but you can see how it applies to Katie. Early in her difficulties with attending school, she began to believe events were out of her control and that she was unable even to begin to cope. More important, in her eyes the bad situation was all her fault: "I blamed myself for my lack of control." A downward spiral into a major depressive episode followed.

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▲ According to the learned helplessness theory of depression, people become depressed when they believe they have no control over the stress in their lives.

But a major question remains: Is learned helplessness a cause of depression or a correlated side effect of becoming depressed? If it were a cause, learned helplessness would have to exist *before* the depressive episode. Results from a 5-year longitudinal study in children shed some light on this issue. Nolen-Hoeksema, Girgus, and Seligman (1992) reported that negative attributional style did not predict later symptoms of depression in young children; rather, stressful life events seemed to be the major precipitant of symptoms. However, as they grew older, children under stress tended to develop more negative cognitive styles, which did tend to predict symptoms of depression in reaction to additional negative events. Nolen-Hoeksema and colleagues speculate that meaningful negative events early in childhood may lead to negative attributional styles, making these children more vulnerable to future depressive episodes when stressful events occur. Indeed, most studies support the finding that negative cognitive styles precede and are a risk factor for depression (Alloy & Abramson, 2006; Garber & Carter, 2006; Garber et al., 2009).

This thinking recalls the types of psychological vulnerabilities theorized to contribute to the development of anxiety disorders (Barlow, 1988, 2002; Suárez, Bennett, Goldstein, & Barlow, 2009)—that is, in a person who has a nonspecific genetic vulnerability to either anxiety or depression, stressful life events activate a psychological sense that life events are uncontrollable (Barlow, 2002; Chorpita & Barlow, 1998). Evidence suggests that negative attributional styles are not specific to depression but also characterize people with anxiety (Barlow, 2002; Hankin & Abramson, 2001; Suárez et al., 2009). This may indicate that a psychological (cognitive) vulnerability is no more specific for mood disorders than a genetic vulnerability. Both types of vulnerabilities may underlie numerous disorders.

Abramson, Metalsky, and Alloy (1989) revised the learned helplessness theory to deemphasize the influence of negative attributions and highlight the development of a sense of hopelessness as a crucial cause of many forms of depression. Attributions are important only to the extent that they contribute to a sense of hopelessness. This fits well with recent thinking on crucial differences between anxiety and depression. Both anxious and depressed individuals feel helpless and believe they lack control, but only in depression do they give up and become hopeless about ever regaining control (Alloy & Abramson, 2006; Barlow, 1991, 2002; Chorpita & Barlow, 1998).

Negative Cognitive Styles

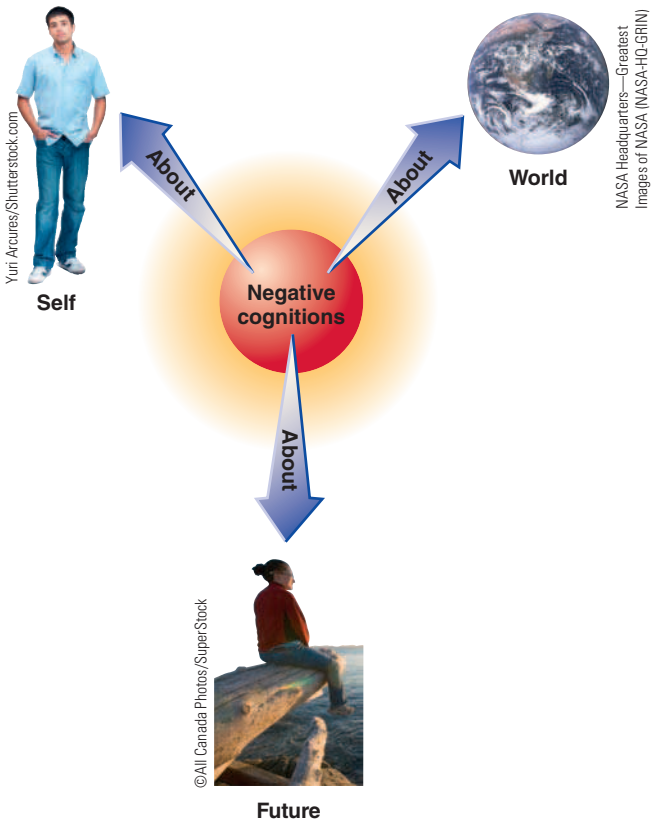
In 1967, Aaron T. Beck (1967, 1976) suggested that depression may result from a tendency to interpret everyday events in a negative way. According to Beck, people with depression make the worst of everything; for them, the

smallest setbacks are major catastrophes. In his extensive clinical work, Beck observed that all of his depressed patients thought this way, and he began classifying the types of "cognitive errors" that characterized this style. From the long list he compiled, two representative examples are *arbitrary inference* and *overgeneralization*. Arbitrary inference is evident when a depressed individual emphasizes the negative rather than the positive aspects of a situation. A high school teacher may assume he is a terrible instructor because two students in his class fell asleep. He fails to consider other reasons they might be sleeping (up all night partying, perhaps) and "infers" that his teaching style is at fault. As an example of overgeneralization, when your professor makes one critical remark on your paper, you then assume you will fail the class despite a long string of positive comments and good grades on other papers. You are overgeneralizing from one small remark. According to Beck, people who are depressed think like this all the time. They make cognitive errors in thinking negatively about themselves, their immediate world, and their future, three areas that together are called the **depressive cognitive triad** (■ Figure 6.2).

In addition, Beck theorized, after a series of negative events in childhood, individuals may develop a deep-seated *negative schema,* an enduring negative cognitive belief system about some aspect of life (Beck, Epstein, & Harrison, 1983; Gotlib & Krasnoperova, 1998; Gotlib, Kurtzman, & Blehar, 1997; Gotlib & MacLeod, 1997; Young, Rygh, Weinberger, & Beck, 2008). In a self-blame schema, individuals feel personally responsible for every bad thing that happens. With a negative self-evaluation schema, they believe they can never do anything correctly. In Beck's view, these cognitive errors and schemas are automatic—that is, not necessarily conscious. Indeed, an individual might not even be aware of thinking negatively and illogically. Thus, minor negative events can lead to a major depressive episode.

A variety of evidence supports a cognitive theory of emotional disorders in general and depression in particular (Goodman & Gotlib, 1999; Ingram, Miranda, & Segal, 2006; Mazure, Bruce, Maciejewski, & Jacobs, 2000; Reilly-Harrington et al., 1999). The thinking of depressed individuals is consistently more negative than that of nondepressed individuals (Gotlib & Abramson, 1999; Hollon, Kendall, & Lumry, 1986; Joormann, 2009) in each dimension of the cognitive triad—the self, the world, and the future (see, for example, Garber & Carter, 2006; Joormann, 2009). Depressive cognitions seem to emerge from distorted and probably automatic methods of processing information. People prone to depression are more likely to recall negative events when they are depressed than when they are not depressed or than are nondepressed individuals (Gotlib, Roberts, & Gilboa, 1996; Joormann, 2009; Lewinsohn & Rosenbaum, 1987).

The implications of this theory are important. By recognizing cognitive errors and the underlying schemas, we can correct them and alleviate depression and related emotional disorders. In developing ways to do this, Beck became the father of cognitive therapy, one of the most important developments in psychotherapy in the past 50 years (see p. 230). Individuals with bipolar disorder also exhibit negative cognitive styles—but with a twist. Cognitive styles in these individuals are characterized by ambitious striving for goals, perfectionism, and self-criticism in addition to the more usual depressive cognitive styles (Alloy & Abramson, 2010; Johnson et al., 2008).



■ **Figure 6.2** Beck's cognitive triad for depression.

Cognitive Vulnerability for Depression: An Integration

Seligman and Beck developed their theories independently, and good evidence indicates their models are independent in that some people may have a negative outlook (dysfunctional attitudes), whereas others may explain things negatively (hopeless attributes) (Joiner & Rudd, 1996; Spangler, Simons, Monroe, & Thase, 1997). Nevertheless, the basic

learned helplessness theory of depression Martin Seligman's theory that people become anxious and depressed when they make an attribution that they have no control over the stress in their lives (whether or not they actually have control).
depressive cognitive triad Thinking errors by depressed people negatively focused in three areas: themselves, their immediate world, and their future.

premises overlap a great deal and considerable evidence suggests depression is always associated with pessimistic explanatory style and negative cognitions. Evidence also exists that cognitive vulnerabilities predispose some people to view events in a negative way, putting them at risk for depression (see, for example, Ingram, Miranda, & Segal, 2006; Mazure, Bruce, Maciejewski, & Jacobs, 2000; Reilly-Harrington et al., 1999).

Good evidence supporting this conclusion comes from the Temple-Wisconsin study of cognitive vulnerability to depression conducted by Lauren Alloy and Lyn Abramson (Alloy & Abramson, 2006; Alloy, Abramson, Safford, & Gibb, 2006). University freshmen who were not depressed at the time of the initial assessment were assessed every several months for up to 5 years to determine whether they experienced any stressful life events or diagnosable episodes of depression or other psychopathology. At the first assessment, the investigators determined whether the students were cognitively vulnerable to developing depression or not on the basis of their scores on questionnaires that measure dysfunctional attitudes and hopelessness attributions. Results indicated students at high risk because of dysfunctional attitudes reported higher rates of depression in the past compared to the low-risk group. But the really important results come from the prospective portion of the study. Negative cognitive styles do indicate a vulnerability to later depression. Even if participants had never suffered from depression before in their lives, high-risk participants (who scored high on the measures of cognitive vulnerability) were 6–12 times more likely than low-risk participants to experience a major depressive episode. In addition, 16% of the high-risk participants versus only 2.7% of the low-risk participants experienced major depressive episodes, and 46% versus 14% experienced minor depressive symptoms (Alloy & Abramson, 2006). In another important study, Abela and Skitch (2007) demonstrated that children at high risk for depression because of a depressed mother showed depressive cognitive styles when under minor stress, unlike children not at risk. The data are suggestive that cognitive vulnerabilities to developing depression do exist and, when combined with biological vulnerabilities, create a slippery path to depression.

Social and Cultural Dimensions

A number of social and cultural factors contribute to the onset or maintenance of depression. Among these, marital relationships, gender, and social support are most prominent.

Marital Relations

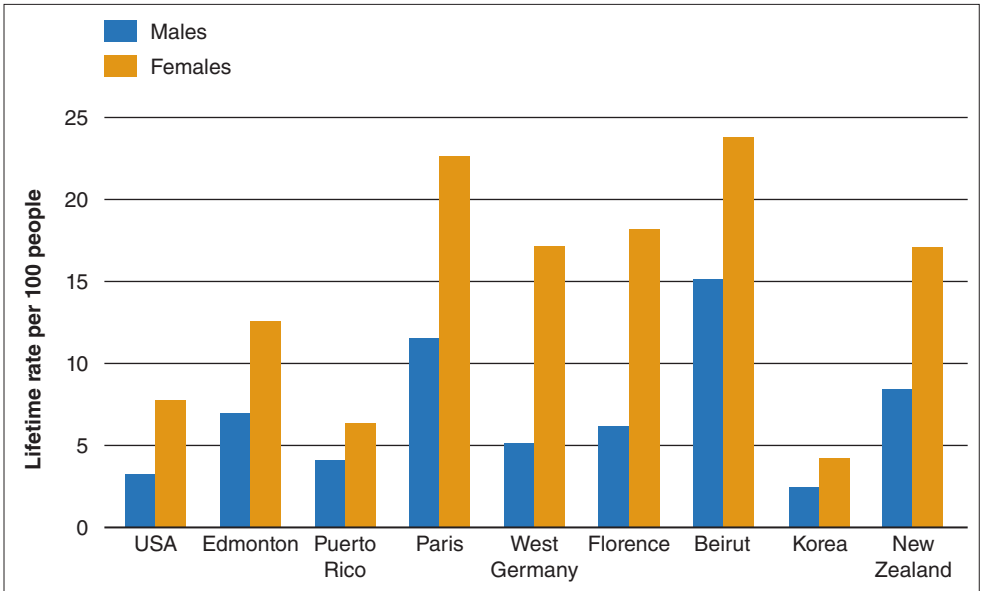
Marital dissatisfaction and depression including bipolar disorder are strongly related, as suggested earlier when it was noted that disruptions in relationships often lead to depression (Davila, Stroud, & Starr, 2009). Bruce and Kim (1992) collected data on 695 women and 530 men and then reinterviewed them up to 1 year later. During this period, a number of participants separated from or divorced their spouses, although the majority reported stable marriages. Approximately 21% of the women who reported a marital split during the study experienced severe depression, a rate 3 times higher than that for women who remained married. Nearly 17% of the men who reported a marital split developed severe depression, a rate *9 times* higher than that for men who remained married. However, when the researchers considered only those participants with no history of severe depression, 14% of the men who separated or divorced during the period experienced severe depression, as did approximately 5% of the women. In other words, *only the men* faced a heightened risk of developing a mood disorder for the first time immediately following a marital split.

Another finding with considerable support is that depression including bipolar disorder, particularly if it continues, may lead to substantial deterioration in marital relationships (Beach, Jones, & Franklin, 2009; Beach, Sandeen, & O'Leary, 1990; Davila et al., 2009; Gotlib & Beach, 1995; Paykel & Weissman, 1973; Uebelacker & Whisman, 2006). It is not hard to figure out why. Being around someone who is continually negative, ill tempered, and pessimistic becomes tiring after a while. Because emotions are contagious, the spouse probably begins to feel bad also. These kinds of interactions precipitate arguments or, worse, make the nondepressed spouse want to leave (Joiner & Timmons, 2009; Whisman, Weinstock, & Tolejko, 2006).

But conflict within a marriage seems to have different effects on men and women. Depression seems to cause men to withdraw or otherwise disrupt the relationship. For women, however, problems in the relationship most often cause depression. Thus, for both men and women, depression and problems in marital relations are associated, but the causal direction is different (Fincham, Beach, Harold, & Osborne, 1997), a result also found by Spangler, Simons, Monroe, and Thase (1996). Given these factors, Beach, Jones, & Franklin (2009) suggest that therapists treat disturbed marital relationships at the same time as the mood disorder.

Mood Disorders in Women

Data on the prevalence of mood disorders indicate dramatic gender imbalances. Although bipolar disorder is evenly divided between men and women, almost 70% of the individuals with major depressive disorder and dysthymia are women (Bland, 1997; Hankin & Abramson, 2001; Kessler, 2006; Weissman et al., 1991). What is particularly striking is that this gender imbalance is constant around the world, even though overall rates of disorder may vary from country to country (Kessler, 2006; Weissman & Olfson, 1995; ■ Figure 6.3). Often overlooked is the similar ratio for most anxiety disorders, particularly panic disorder and generalized anxiety disorder. Women represent an even greater proportion of specific phobias, as we noted in Chapter 2. What could account for this?



■ **Figure 6.3** Lifetime international rate per 100 people for major depression. (Reprinted, with permission, from Weissman, M. M., & Olfson, M., 1995. Depression in women: Implications for health care research. *Science, 269,* 799–801, © 1995 American Association for the Advancement of Science.)

It may be that gender differences in the development of emotional disorders are strongly influenced by perceptions of uncontrollability (Barlow, 1988, 2002). If you feel a sense of mastery over your life and the difficult events we all encounter, you might experience occasional stress but you will not feel the helplessness central to anxiety and mood disorders. The source of these differences is cultural, in the sex roles assigned to men and women in our society. Males are strongly encouraged to be independent, masterful, and assertive; females, by contrast, are expected to be more passive, sensitive to other people, and (perhaps) to rely on others more than males do (needs for affiliation) (Cyranowski, Frank, Young, & Shear, 2000; Hankin & Abramson, 2001). Although these stereotypes are slowly changing, they still describe current sex roles to a large extent. But this culturally induced dependence and passivity may put women at heightened risk for emotional disorders by increasing their feelings of uncontrollability and helplessness.

Evidence has accumulated that parenting styles encouraging stereotypic gender roles are implicated in the development of early psychological vulnerability to later depression or anxiety (Chorpita & Barlow, 1998; Suárez et al., 2009), specifically, a smothering, overprotective style that prevents the child from developing initiative. Also interesting is the "sudden surge" in depression among girls mentioned earlier that occurs during puberty. Many thought this might be biologically based. However, Kessler (2006) notes that low self-esteem emerges quickly in girls in seventh grade if the school system has a seventh- through ninth-grade middle school, but low self-esteem among girls does not emerge until ninth grade when the school has a kindergarten through eighth-grade primary school and a 4-year high school (Simmons & Blyth, 1987). These results suggest that the younger girls just entering a new school, whether it is seventh, ninth, or some other grade, find it stressful. Also, girls who mature physically early have more distress and depression than girls who don't (Ge, Conger, & Elder, 1996).

Women tend to place greater value on intimate relationships than men, which can be protective if social networks are strong, but it may also put them at risk. Disruptions in such relationships, combined with an inability to cope with the disruptions, seem to be far more damaging to women than to men (Nolen-Hoeksema & Hilt, 2009; Rudolph & Conley, 2005). Cyranowski and associates (2000) note that the tendency for adolescent girls to express aggression by rejecting other girls, combined with a greater sensitivity to rejection, may precipitate more depressive episodes in these adolescent girls compared to boys. Kendler, Myers, and Prescott (2005) also observed that women tend to have larger and more intimate social networks than men and that emotionally supportive groups of friends protect against depression. However, data from Bruce and Kim (1992), reviewed earlier, suggest that if the disruption in a marital relationship reaches the stage of divorce, men who had previously been functioning well are at greater risk for depression.

Another potentially important gender difference has been suggested by Susan Nolen-Hoeksema (1990, 2000; Nolen-Hoeksema, Larson, & Grayson, 1999; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Women tend to ruminate more than men about their situation and blame themselves for being depressed. Men tend to ignore their feelings, perhaps engaging in activity to take their minds off them (Addis, 2008). This male behavior may be therapeutic because "activating" people (getting them busy doing something) is a common element of successful therapy for depression (Dimidjian, Martell, Addis, & Herman-Dunn, 2008; Jacobson, Martell, & Dimidjian, 2001).

As Strickland (1992) pointed out, women are at a disadvantage in our society: They experience more discrimination, poverty, sexual harassment, and abuse than do men. They also earn less respect and accumulate less power. Three fourths of the people living in poverty in the United States are women and children. Women, particularly single mothers, have a difficult time entering the workplace. Of note, married women employed full time outside the home report levels of depression no greater than those of employed married men. Single, divorced, and widowed women experience significantly more depression than men

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▲ Of the impoverished people in the United States, three-fourths are women and children.

in the same categories (Davila et al., 2009; Weissman & Klerman, 1977). This does not necessarily mean that anyone should get a job to avoid becoming depressed. Indeed, for a man or woman, feeling mastery, control, and value in the strongly socially supported role of homemaker and parent should be associated with low rates of depression.

Finally, other disorders may reflect gender role stereotypes, but in the opposite direction. Disorders associated with aggressiveness, overactivity, and substance abuse occur far more often in men than in women (Barlow, 1988, 2002). Identifying the reasons for gender imbalances across the full range of psychopathological disorders may prove important in discovering causes of disorders.

Social Support

In Chapter 2, we examined the powerful effect of social influences on our psychological and biological functioning. We cited several examples of how social influences seem to contribute to early death, such as the evil eye or lack of social support in old age. In general, the greater the number and frequency of your social relationships and contacts, the longer you are likely to live (see, for instance, House, Landis, & Umberson, 1988). It is not surprising, then, that social factors influence whether we become depressed (Beach et al., 2009).

In an early landmark study, Brown and Harris (1978) first suggested the important role of social support in the onset of depression. In a study of a large number of women who had experienced a serious life stress, they discovered that only 10% of the women who had a friend in whom they could confide became depressed, compared to 37% of the women who did not have a close supportive relationship. Later prospective studies have also confirmed the importance of social support (or lack of it) in predicting the onset of depressive symptoms at a later time (see, for instance, Joiner, 1997; Kendler, Kuhn et al., 2005; Lin & Ensel, 1984; Monroe, Imhoff, Wise, & Harris, 1983; Monroe et al., 2009; Phifer & Murrell, 1986). The importance of social support in preventing depression holds true in China (Wang, Wang, & Shen, 2006) and every other country in which it has been studied. Other studies have established the importance of social support in speeding recovery from depressive episodes (Keitner et al., 1995; McLeod, Kessler, & Landis, 1992; Sherbourne, Hays, & Wells, 1995). In an interesting twist, several studies examined the effects of social support in speeding recovery from both manic and depressive episodes in patients with bipolar disorder, and they came up with a surprising finding. A socially supportive network of friends and family helped speed recovery from depressive episodes but not from manic episodes (Johnson, Winett, Meyer, Greenhouse, & Miller, 1999; Johnson et al., 2008, 2009). This finding highlights the uniquely different quality of manic episodes (McGuffin et al., 2003). In any case, these and related findings on the importance of social support have led to an exciting new psychological therapeutic approach for emotional disorders called interpersonal psychotherapy, which we discuss later in this chapter.

Let's return again to Katie. In reflecting on her turbulent times and the days when death seemed more rewarding than life, one thing sticks out clearly in her mind:

> My parents are the true heroes of these early years. I will always admire their strength, their love, and their commitment. My father is a high school graduate and my mother has an eighth-grade education. They dealt with complicated legal, medical, and psychological issues. They had little support from friends or professionals, yet they continued to do what they believed best. In my eyes there is no greater demonstration of courage and love.

Katie's parents did not have the social support that might have helped them through these difficult years, but they gave it to Katie. We return to her case later.

An Integrative Theory

How do we put all this together? Basically, depression and anxiety may often share a common, genetically determined biological vulnerability (Barlow, 2002; Barlow, Chorpita, & Turovsky, 1996; Suárez et al., 2009) that can be described as an overactive neurobiological response to stressful life events. Again, this vulnerability is simply a general tendency to develop depression (or anxiety) rather than a specific vulnerability for depression or anxiety itself. But

only between 20% and 40% of the causes of depression can be attributed to genes. For the remainder, we look at life experience.

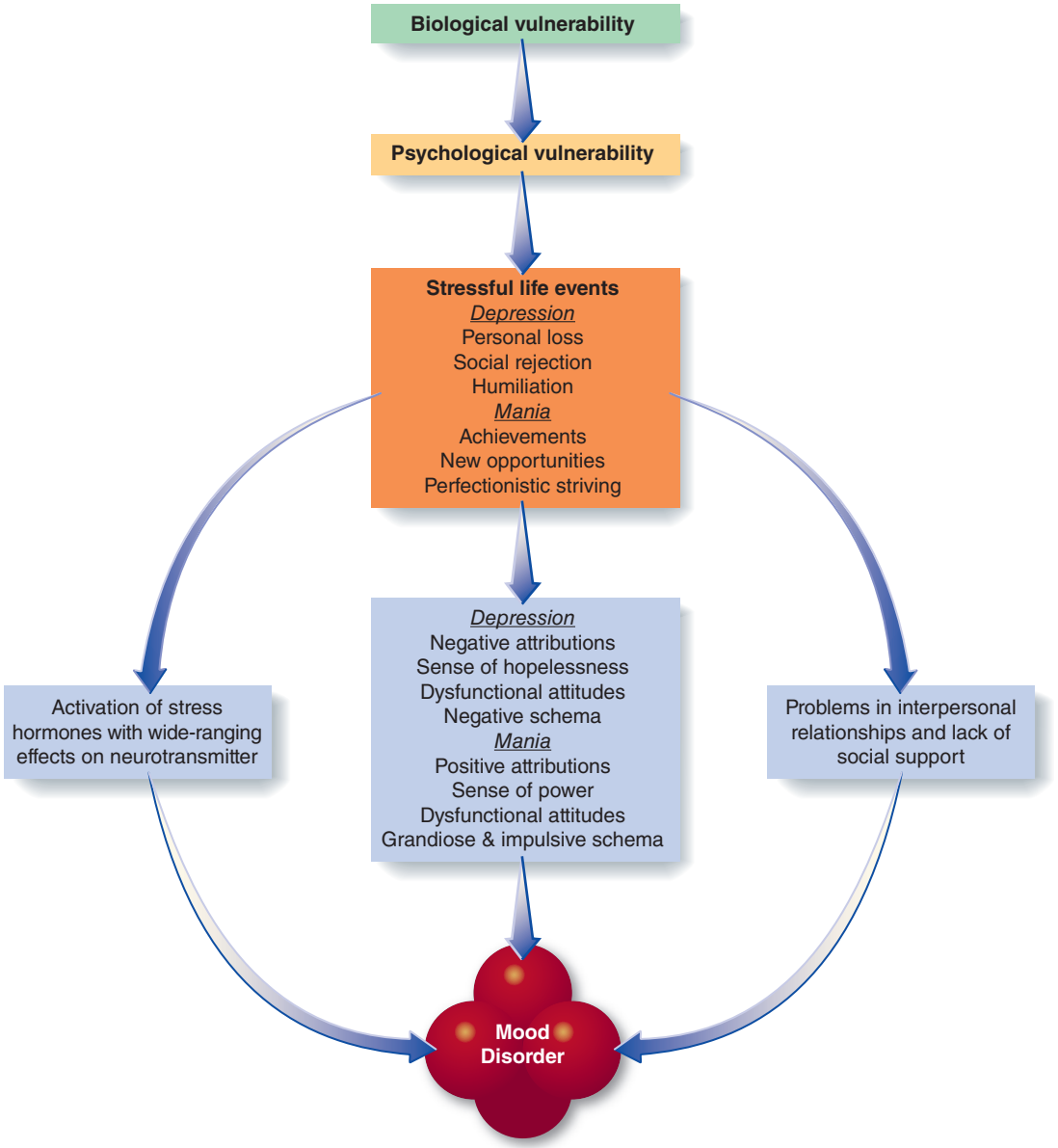
People who develop mood disorders also possess a psychological vulnerability experienced as feelings of inadequacy for coping with the difficulties confronting them and depressive cognitive styles. As with anxiety, we may develop this sense of control in childhood (Barlow, 2002; Chorpita & Barlow, 1998). It may range on a continuum from total confidence to a complete inability to cope. When vulnerabilities are triggered, the pessimistic "giving up" process seems crucial to the development of depression (Alloy, Kelly, Mineka, & Clements, 1990; Alloy et al., 2000; Alloy & Abramson, 2006).

These two vulnerabilities of inadequate coping and depressive cognitive style in combination comprise the temperament of neuroticism or negative affect. You will remember from Chapter 4 that neuroticism is associated with biochemical markers of stress and depression (see, for example, Nemeroff, 2004; Thase, 2009) and different levels of arousal in different hemispheres in the brain (hemispheric lateral asymmetry) (Davidson, 1993; Heller & Nitschke, 1997), and activation of specific brain circuits (Davidson et al., 2009; Elliott, Rubinsztein, Sahakian, & Dolan, 2002; Liotti, Mayberg, McGinnis, Brannan, & Jerabek, 2002). There is also good evidence that stressful life events trigger the onset of depression in most cases in these vulnerable individuals, particularly initial episodes. How do these factors interact? Current thinking is that stressful life events activate stress hormones, which, in turn, have wide-ranging effects on neurotransmitter systems, particularly those involving serotonin, norepinephrine, and the corticotropin-releasing factor system. Booij and Van der Does (2007) have illustrated how neurotransmitter function and negative cognitive styles interact. They collaborated with 39 patients who had suffered an episode of major depression but had recovered. These patients participated in two biological test or "challenge" procedures called acute tryptophan depletion (ATD) that had the effect of temporarily lowering levels of serotonin. This is accomplished fairly easily by altering diet for 1 day by restricting intake of tryptophan (a precursor to serotonergic functioning) and increasing a mixture of essential amino acids. Participants in the experiment, of course, were fully informed of these effects and collaborated willingly.

What Booij and Van der Does (2007) found was that this biological challenge was, as usual, effective in temporarily inducing a variety of depressive symptoms in some of these individuals, but that these symptoms were more pronounced in those who also had evidence of the cognitive vulnerability marker. That is, cognitive vulnerability assessed before the biological challenge clearly predicted a depressive response. Interestingly, a challenge with ATD causes no significant changes in mood in healthy samples; rather, it is limited to those individuals who are vulnerable to depression.

What we have so far is a possible mechanism for the diathesis–stress model. Finally, it seems clear that factors such as interpersonal relationships or cognitive style may protect us from the effects of stress and therefore from developing mood disorders. Alternatively, these factors may at least determine whether we quickly recover from these disorders or not. But remember that bipolar disorder, and particularly activation of manic episodes, seems to have a somewhat different genetic basis and a different response to social support. Scientists are beginning to theorize that individuals with bipolar disorder, in addition to factors outlined so far, are also highly sensitive to the experience of life events connected with striving to reach important goals, perhaps because of an overactive brain circuit called the behavioral approach system (BAS) (Alloy & Abramson, 2010; Gruber, Johnson, Oveis, & Keltner, 2008). In these cases, stressful life events that are more positive but still stressful, such as starting a new job, or pulling all-nighters to finish an important term paper, might precipitate a manic episode in vulnerable individuals instead of a depressive episode. Individuals with bipolar disorder are also highly sensitive to disruptions in circadian rhythm. So individuals with bipolar disorder might possess brain circuits that predispose them to both depression and mania. Research of this hypothesis is just commencing.

In summary, biological, psychological, and social factors all influence the development of mood disorders, as depicted in ■ Figure 6.4. This model does not fully account for the varied presentation of mood disorders—seasonal, bipolar, and so on—although mania in bipolar disorder seems to be associated with unique genetic contributions and is triggered by relatively unique life events as noted above. But why would someone with an underlying genetic vulnerability who experiences a stressful life event develop a mood disorder rather than an anxiety or somatoform disorder? As with the anxiety disorders and other stress disorders, specific psychosocial circumstances, such as early learning experiences, may interact with specific genetic vulnerabilities and personality characteristics to produce the rich variety of emotional disorders.



■ **Figure 6.4** An integrative model of mood disorders. (© Cengage Learning 2013)

Concept Check 6.3

Answer these questions about the various causes of mood disorders.

1. List five biological sources that can contribute to mood disorders. _____ _____ _____ _____ _____
2. What psychological factors can have an impact on mood disorders? _____ _____ _____ _____
3. Name several social and cultural dimensions that contribute to mood disorders. _____ _____ _____

Treatment of Mood Disorders

› **What medical and psychological treatments have been successful in treating mood disorders?**

Researchers have learned a great deal about the neurobiology of mood disorders during the past several years. Findings on the complex interplay of neurochemicals are beginning to shed light on the nature of mood disorders. As we have noted, the principal effect of medications is to alter levels of these neurotransmitters and other related neurochemicals. Other biological treatments, such as electroconvulsive therapy, dramatically affect brain chemistry. A more interesting development alluded to throughout this book, however, is that powerful psychological treatments also alter brain chemistry. Despite these advances, most cases of depression go untreated because neither health care professionals nor patients recognize and correctly identify or diagnose depression. Similarly, many professionals and patients are unaware of the existence of effective and successful treatments (Delano-Wood & Abeles, 2005; Hirschfeld et al., 1997).

Medications

A number of medications are effective treatments for depression. New information often becomes available on new medications or the latest estimates of effectiveness of older medications.

Antidepressants

Four basic types of antidepressant medications are used to treat depressive disorders: selective serotonin reuptake inhibitors (SSRIs), mixed reuptake inhibitors, tricyclic antidepressants, and monoamine oxidase (MAO) inhibitors. It is important to note at the outset that there are few, if any, differences in effectiveness among the different antidepressants; approximately 50% of patients receive some benefit, with about half of the 50% coming very close to normal functioning (remission). If dropouts are excluded and only those who complete treatment are counted, the percentage of patients receiving at least some benefit increases to between 60% and 70%.

The class of drugs currently considered the first choice in drug treatment for depression seems to have a specific effect on the serotonin neurotransmitter system (although such drugs affect other systems to some extent). These *selective*-serotonin reuptake inhibitors specifically block the presynaptic reuptake of serotonin. This temporarily increases levels of serotonin at the receptor site, but again the precise long-term mechanism of action is unknown, although levels of serotonin are eventually increased (Gitlin, 2009; Thase & Denko, 2008). Perhaps the best-known drug in this class is *fluoxetine* (Prozac). Like many other medications, Prozac was initially hailed as a breakthrough drug; it even made the cover of *Newsweek* (Cowley & Springen, 1990). Then reports began to appear that it might lead to suicidal preoccupation; paranoid reactions; and, occasionally, violence (see, for example, Mandalos & Szarek, 1990; Teicher, Glod, & Cole, 1990). Prozac went from being a wonder drug in the eyes of the press to a potential menace to modern society. Neither conclusion was true. Findings indicated that the risks of suicide with this drug for the general population were no greater than with any other antidepressant (Fava & Rosenbaum, 1991), and the effectiveness is about the same as that of other antidepressants.

Recently, concerns about suicidal risks (increased thoughts, and so on) have surfaced again, particularly among adolescents, and this time it looks like the concerns are justified, at least for adolescents (Baldessarini, Pompili, & Tondo, 2006; Berman, 2009; Fergusson, Doucette, et al., 2005; Hammad, Laughren, & Racoosin, 2006; Olfson, Marcus, & Schaffer, 2006). These findings have led to warnings from the Food and Drug Administration (FDA) and other regulatory agencies around the world about these drugs. However, Gibbons, Hur, Bhaumik, and Mann (2006) found that actual suicide rates were lower in sections of the United States where prescriptions for SSRIs were higher. In addition, the SSRIs were also associated with a small but statistically significant *decrease* in actual suicides among adolescents compared to depressed adolescents not taking these drugs based on a large community survey (Olfson, Shaffer, Marcus & Greenberg, 2003). These findings are *correlational,* meaning we can't conclude that increased prescriptions for SSRIs caused lower suicide rates. Research will continue on this important question. One possible conclusion is that SSRIs cause increased thoughts about suicide in the first few weeks in some adolescents but, once they start working after a month or more, may prevent the depression from leading to suicide (Berman, 2009; Simon, 2006). Prozac and other SSRIs have their own set of side effects, the most prominent of which are physical agitation, sexual dysfunction, low sexual desire (which is prevalent, occurring in 50% to 75% of cases), insomnia, and gastrointestinal upset. But these side effects, on the whole, seem to bother most patients less than the side effects associated with tricyclic antidepressants, with the possible exception of the sexual dysfunction. Studies suggest similar effectiveness of SSRIs and tricyclics with dysthymia (Lapierre, 1994).

Two newer antidepressants (sometimes termed *mixed reuptake inhibitors*) seem to have somewhat different mechanisms of neurobiological action. Venlafaxine (Effexor) is related to tricyclic antidepressants, but acts in a slightly different manner, blocking reuptake of norepinephrine and serotonin. Some side effects associated with the SSRIs are reduced with venlafaxine, as is the risk of

damage to the cardiovascular system. Other typical side effects remain, including nausea and sexual dysfunction.

Nefazodone (Serzone) is closely related to the SSRIs but seems to improve sleep efficiency instead of disrupting sleep. Both drugs are roughly comparable in effectiveness to older antidepressants (American Psychiatric Association, 2000; Nemeroff, 2006; Preskorn, 1995; Thase & Kupfer, 1996), but nefazodone has now been withdrawn because of the risk of liver damage (Gitlin, 2009).

MAO inhibitors work differently. As their name suggests, they block the enzyme MAO that breaks down such neurotransmitters as norepinephrine and serotonin. The result is roughly equivalent to the effect of the tricyclics. Because they are not broken down, the neurotransmitters pool in the synapse, leading to a down-regulation. The MAO inhibitors seem to be as effective as or slightly more effective than the tricyclics (American Psychiatric Association, 2000; Depression Guideline Panel, 1993), with somewhat fewer side effects. Some evidence suggests they are relatively more effective for depression with atypical features (Thase & Kupfer, 1996). But MAO inhibitors are used far less often because of two potentially serious consequences: Eating and drinking foods and beverages containing tyramine, such as cheese, red wine, or beer, can lead to severe hypertensive episodes and, occasionally, death. In addition, many other drugs that people take daily, such as cold medications, are dangerous and even fatal in interaction with an MAO inhibitor. Therefore, MAO inhibitors are usually prescribed only when other antidepressants are not effective.

Tricyclic antidepressants were the most widely used treatments for depression before the introduction of SSRIs, but are now used less commonly (Gitlin, 2009; Thase & Denko, 2008). The best-known variants are probably imipramine (Tofranil) and amitriptyline (Elavil). It is not yet clear how these drugs work, but initially, at least, they block the reuptake of certain neurotransmitters, allowing them to pool in the synapse and, as the theory goes, desensitize or down-regulate the transmission of that particular neurotransmitter (so less of the neurochemical is transmitted). Tricyclic antidepressants seem to have their greatest effect by down-regulating norepinephrine, although other neurotransmitter systems, particularly serotonin, are also affected. This process then has a complex effect on both presynaptic and postsynaptic regulation of neurotransmitter activity, eventually restoring appropriate balance. Side effects include blurred vision, dry mouth, constipation, difficulty urinating, drowsiness, weight gain (at least 13 pounds on average), and, sometimes, sexual dysfunction. Therefore, as many as 40% of these patients may stop taking the drug, thinking the cure is worse than the disease. Nevertheless, with careful management, many side effects disappear over time. Another issue clinicians must consider is that tricyclics are *lethal* if taken in excessive doses; therefore, they must be prescribed with great caution to patients with suicidal tendencies.

Because the SSRI and other drugs relieve symptoms of depression to some extent in about 50% of all patients treated but eliminate depression or come close to it in only 25% to 30% of all patients treated (termed *remission*) (Trivedi et al., 2006), the question remains: What do clinicians do when depression does not respond adequately to drug treatment, often called treatment-resistant depression? A large study called the Sequenced Treatment Alternatives to Relieve Depression (STAR*D) examined whether offering those individuals who did not achieve remission the alternatives of either adding a second drug or switching to a second drug is useful. Among those who were willing, approximately 20% (for switching) to approximately 30% (for adding a second drug) achieved remission. When repeating this with a third drug among those who had failed to achieve remission with the first two drugs, the results weren't as good (between 10% and 20% achieved remission) (Insel, 2006; Menza, 2006; Rush, 2007), and very few clinicians would go to a third drug in the same class after failing on the first two (Gitlin, 2009). The conclusion is that it's worth being persistent as long as individuals with depression are still willing to try a second drug because some people who don't improve with the first drug could improve with a different drug. Later, we report on combining psychological treatments with drugs. In summary, all antidepressant medications work about the same in large clinical trials, but sometimes a patient will not do well on one drug and respond better to another.

Current studies indicate that drug treatments effective with adults are not necessarily effective with children (American Psychiatric Association, 2000; Geller et al., 1992; Kaslow, Davis, & Smith, 2009; Ryan, 1992). Sudden deaths of children younger than age 14 who were taking tricyclic antidepressants have been reported, particularly during exercise, as in routine school athletic competition (Tingelstad, 1991). The causes imply cardiac side effects. But evidence indicates that at least one of the SSRIs, fluoxetine (Prozac), is safe and has some evidence for efficacy with adolescents both initially (Kaslow et al., 2009; Treatment for Adolescents with Depression Study [TADS] Team, 2004), and at follow-up (TADS Team, 2009), particularly if combined with cognitive behavioral therapy (CBT) (March & Vitiello, 2009). Traditional antidepressant drug treatments are usually effective with the elderly, but administering them takes considerable skill because older people may suffer from a variety of side effects not experienced by younger adults, including memory impairment and physical agitation (Blazer & Hybels, 2009; Delano-Wood & Abeles, 2005; Deptula & Pomara, 1990; Fiske et al., 2009; Marcopulos & Graves, 1990). Use of a depression care manager to deliver care to depressed elderly patients right in the office of their primary medical care doctor including encouraging compliance with drug taking, monitoring side effects unique to older adults, and delivering a bit of psychotherapy was more effective than usual care (Alexopoulos et al., 2005; Unutzer et al., 2002).

Clinicians and researchers have concluded that recovery from depression, although important, may not be the most important therapeutic outcome (Frank et al., 1990; Thase, 2009). Most people eventually recover from a major de-

pressive episode, some rather quickly. A more important goal is often to delay the next depressive episode or even prevent it entirely (National Institute of Mental Health, 2003; Prien & Potter, 1993; Thase, 2009; Thase & Kupfer, 1996). This is particularly important for patients who retain some symptoms of depression or have a past history of chronic depression or multiple depressive episodes. Because all these factors put people at risk for relapse, it is recommended that drug treatment go well beyond the termination of a depressive episode, continuing perhaps 6 to 12 months after the episode is over, or even longer (American Psychiatric Association, 2000; Insel, 2006). The drug is then gradually withdrawn over weeks or months. Long-term administration of antidepressants has not been studied extensively, and there is even some evidence that long-term treatment lasting several years may worsen the course of depression (Fava, 2003).

Antidepressant medications have relieved severe depression and undoubtedly prevented suicide in tens of thousands of patients around the world. Although these medications are readily available, many people refuse them or are not eligible to take them. Some are wary of long-term side effects. Women of childbearing age must protect themselves against the possibility of conceiving while taking antidepressants because of possible damage to the fetus, although the best evidence suggests that preterm birth, but not damage, is the most likely outcome (Wisner et al., 2009). In addition, approximately 30% to 40% of patients who take a full course of treatment do not respond adequately to these drugs, and a substantial number of the remainder are left with residual symptoms.

Lithium

Another type of antidepressant drug, *lithium carbonate,* is a common salt widely available in the natural environment (Nemeroff, 2006). It is found in our drinking water in amounts too small to have any effect. However, the side effects of therapeutic doses of lithium are potentially more serious than those of other antidepressants. Dosage has to be carefully regulated to prevent toxicity (poisoning) and lowered thyroid functioning, which might intensify the lack of energy associated with depression. Substantial weight gain is also common. Lithium, however, has one major advantage that distinguishes it from other antidepressants: It is also often effective in preventing and treating manic episodes. Therefore, it is most often referred to as a **mood-stabilizing drug**. Antidepressants can induce manic episodes, even in individuals without preexisting bipolar disorder (Goodwin & Ghaemi, 1998; Goodwin & Jamison, 2007; Prien et al., 1984), and lithium remains the gold standard for treatment of bipolar disorder (Thase & Denko, 2008).

Results indicate that 50% of bipolar patients respond well to lithium initially, meaning at least a 50% reduction in manic symptoms (Goodwin & Jamison, 2007). Thus, although effective, lithium provides many people with inadequate therapeutic benefit. Patients who don't respond to lithium can take other drugs with antimanic properties, including anticonvulsants such as carbamazepine and valproate (Divalproex) and calcium channel blockers such as verapamil (Keck & McElroy, 2002; Sachs & Rush, 2003; Thase & Denko, 2008). Valproate has recently overtaken lithium as the most commonly prescribed mood stabilizer for bipolar disorder (Goodwin et al., 2003; Keck & McElroy, 2002; Thase & Denko, 2008) and is equally effective, even for patients with rapid-cycling symptoms (Calabrese et al., 2005). But newer studies show that these drugs have one distinct disadvantage: They are less effective than lithium in preventing suicide (Thase & Denko, 2008; Thies-Flechtner, Muller-Oerlinghausen, Seibert, Walther, & Greil, 1996; Tondo, Jamison, & Baldessarini, 1997). Goodwin and colleagues (2003) reviewed records of more than 20,000 patients taking either lithium or valproate and found the rate of completed suicides was 2.7 times higher in people taking valproate than in people taking lithium. Thus, lithium remains the preferred drug for bipolar disorder (Goodwin & Ghaemi, 1998; Goodwin & Jamison, 2007). This finding was confirmed in a large trial demonstrating no advantage to adding a traditional antidepressant drug such as an SSRI to a mood stabilizer such as lithium (Sachs et al., 2007).

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▲ Kay Redfield Jamison, an internationally respected authority on bipolar disorder, has suffered from the disease since adolescence.

For those patients who do respond to lithium, studies following patients for up to 5 years report that approximately 70% relapse, even if they continue to take the lithium (Frank et al., 1999; Gitlin, Swendsen, Heller, & Hammen, 1995; Peselow, Fieve, Difiglia, & Sanfilipo, 1994). Nevertheless, for almost anyone with recurrent manic episodes, maintenance on lithium or a related drug is recommended to prevent relapse (Yatham et al., 2006). Another problem with drug treatment of bipolar disorder is that people usually like the euphoric or high feeling that mania produces and they often stop taking lithium to maintain or regain the state; that is, they do not comply with the medication regimen. Because the evidence now clearly indicates that individuals who stop their medication are at considerable risk for relapse, other treatment methods, usually psychological in nature, are used to increase compliance.

mood-stabilizing drug A medication used in the treatment of mood disorders, particularly bipolar disorder, that is effective in preventing and treating pathological shifts in mood.

Electroconvulsive Therapy and Transcranial Magnetic Stimulation

When someone does not respond to medication (or in an extremely severe case), clinicians may consider a more dramatic treatment, **electroconvulsive therapy (ECT)**, the most controversial treatment for psychological disorders after psychosurgery. In Chapter 1, we described how ECT was used in the early 20th century. Despite many unfortunate abuses along the way, ECT is considerably changed today. It is now a safe and reasonably effective treatment for severe depression that has not improved with other treatments (American Psychiatric Association, 2000; Gitlin, 2009; National Institute of Mental Health, 2003; Nemeroff, 2006).

In current administrations, patients are anesthetized to reduce discomfort and given muscle-relaxing drugs to prevent bone breakage from convulsions during seizures. Electric shock is administered directly through the brain for less than a second, producing a seizure and a series of brief convulsions that usually lasts for several minutes. In current practice, treatments are administered once every other day for a total of 6 to 10 treatments (fewer if the patient's mood returns to normal). Side effects are generally limited to short-term memory loss and confusion that disappear after a week or two, although some patients may have long-term memory problems. For severely depressed inpatients with psychotic features, controlled studies indicate that approximately 50% of those *not responding* to medication will benefit. Continued treatment with medication or psychotherapy is then necessary because the relapse rate approaches 60% or higher (American Psychiatric Association Practice Guideline, 2000; Depression Guideline Panel, 1993; Fernandez, Levy, Lachar, & Small, 1995; Gitlin, 2009). For example, Sackeim and colleagues (2001) treated 84 patients with ECT and then randomly assigned them to follow-up placebo or one of several antidepressant drug treatments. All patients assigned to placebo relapsed within 6 months compared to 40% to 60% on medication. Thus, follow-up treatment with antidepressant drugs or psychological treatments is necessary, but relapse is still high. Nevertheless, it may not be in the best interest of psychotically depressed and acutely suicidal inpatients to wait 3 to 6 weeks to determine whether a drug or psychological treatment is working; in these cases, immediate ECT may be appropriate.

Recently, another method for altering electrical activity in the brain by setting up a strong magnetic field has been introduced. This procedure is called *transcranial magnetic stimulation (TMS)*, and it works by placing a magnetic coil over the individual's head to generate a precisely localized electromagnetic pulse. Anesthesia is not required, and side effects are usually limited to headaches. Initial reports, as with most new procedures, showed promise in treating depression (Fitzgerald et al., 2003, 2006; George, Lisanby, & Sackheim, 1999), and recent reviews have confirmed that TMS can be effective (Schutter, 2009). But results from several important clinical trials with severe or treatment-resistant psychotic depression reported ECT to be clearly more effective than TMS (Eranti et al., 2007). It may be that TMS is more comparable to antidepressant medication than to ECT (Gitlin, 2009).

Several other nondrug approaches for treatment-resistant depression are in development. Vagus nerve stimulation involves implanting a pacemaker-like device that generates pulses to the vagus nerve in the neck, which, in turn, is thought to influence neurotransmitter production in the brain stem and limbic system (Gitlin, 2009; Marangell et al., 2002). Sufficient evidence has accumulated so that the FDA has approved this procedure, but results are generally weak and it has been little used. Deep brain stimulation has been used with a few severely depressed patients. In this procedure, electrodes are surgically implanted in the limbic system (the emotional brain). These electrodes are also connected to a pacemaker-like device (Mayberg et al., 2005). Time will tell if this is a useful treatment.

Psychological Treatments

Of the effective psychological treatments now available for depressive disorders, two major approaches have the most evidence supporting their efficacy. The first is a cognitive-behavioral approach; Aaron T. Beck, the founder of cognitive therapy, is most closely associated with this approach. The second approach, interpersonal psychotherapy, was developed by Myrna Weissman and Gerald Klerman.

Cognitive-Behavioral Therapy

Beck's **cognitive therapy** grew directly out of his observations of the role of deep-seated negative thinking in generating depression (Beck, 1967, 1976; Beck & Young, 1985; Young et al., 2008). Clients are taught to examine carefully their thought processes while they are depressed and to recognize "depressive" errors in thinking. This task is not always easy because many thoughts are automatic and beyond clients' awareness. Clients are taught that errors in thinking can directly cause depression. Treatment involves correcting cognitive errors and substituting less depressing and (perhaps) more realistic thoughts and appraisals. Later in therapy, underlying negative cognitive schemas (characteristic ways of viewing the world) that trigger specific cognitive errors are targeted, not only in the office but also as part of the client's day-to-day life. The therapist purposefully takes a Socratic approach (teaching by asking questions—see the following dialogue), making it clear that therapist and client are working as a team to uncover faulty thinking patterns and the underlying schemas from which they are generated. Therapists must be skillful and highly trained. Following is an example of an actual interaction between Beck and a depressed client named Irene.

Beck and Irene ✦ A Dialogue

Because an intake interview had already been completed by another therapist, Beck did not spend time reviewing Irene's symptoms in detail or taking a his-

tory. Irene began by describing her "sad states." Beck almost immediately started to elicit her automatic thoughts during these periods.

THERAPIST: What kind of thoughts go through your mind when you've had these sad feelings this past week?

PATIENT: Well . . . I guess I'm thinking what's the point of all this. My life is over. It's just not the same. . . . I have thoughts like, "What am I going to do? . . . Sometimes I feel mad at him, you know my husband. How could he leave me? Isn't that terrible of me? What's wrong with me? How can I be mad at him? He didn't want to die a horrible death. . . . I should have done more. I should have made him go to the doctor when he first started getting headaches. . . . Oh, what's the use. . . ."

T: It sounds like you are feeling quite bad right now. Is that right?

P: Yes.

T: Keep telling me what's going through your mind right now.

P: I can't change anything. . . . It's over. . . . I don't know. . . . It all seems so bleak and hopeless. . . . What do I have to look forward to . . . sickness and then death. . . .

T: So one of the thoughts is that you can't change things and that it's not going to get any better?

P: Yes.

T: And sometimes you believe that completely?

P: Yeah, I believe it, sometimes.

T: Right now do you believe it?

P: I believe it—yes.

T: Right now you believe that you can't change things and it's not going to get better?

P: Well, there is a glimmer of hope but it's mostly. . . .

T: Is there anything in your life that you kind of look forward to in terms of your own life from here on?

P: Well, what I look forward to—I enjoy seeing my kids but they are so busy right now. My son is a lawyer and my daughter is in medical school. . . . So, they are very busy. . . . They don't have time to spend with me.

By inquiring about Irene's automatic thoughts, the therapist began to understand her perspective—that she would go on forever, mostly alone. This illustrates the hopelessness about the future that is characteristic of most depressed patients. A second advantage to this line of inquiry is that the therapist introduced Irene to the idea of looking at her own thoughts, which is central to cognitive therapy (Young et al., 2008).

Between sessions, clients are instructed to *monitor and log* their thought processes carefully, particularly in situations where they might feel depressed. They also attempt to change their behavior by carrying out specific activities assigned as homework, such as tasks in which clients can test their faulty thinking. For example, a client who has to participate in an upcoming meeting might think, "If I go to that meeting, I'll just make a fool of myself and all my colleagues will think I'm stupid." The therapist might instruct the client to go to the meeting, predict ahead of time the reaction of the colleagues, and then see what really happens. This part of treatment is called *hypothesis testing* because the client makes a hypothesis about what's going to happen (usually a depressing outcome) and then, most often, discovers it is incorrect ("My colleagues congratulated me on my presentation"). The therapist typically schedules other activities to *reactivate* depressed patients who have given up most activities, helping them put some fun back into their lives. Cognitive therapy typically takes from 10 to 20 sessions, scheduled weekly.

The late Neil Jacobson and colleagues have shown that increased activities alone can improve self-concept and lift depression (Dimidjian, Martell, Addis, & Herman-Dunn, 2008; Jacobson et al., 1996). This more behavioral treatment has been reformulated because initial evaluation suggests it is as effective as, or more effective than, cognitive approaches (Jacobson, Martell, & Dimidjian, 2001). The new focus of this approach is on preventing avoidance of social and environmental cues that produce negative affect or depression and result in avoidance and inactivity. Rather, the individual is helped to face the cues or triggers and work through them and the depression they produce, with the therapist, by developing better coping skills. Similarly, programmed exercise over the course of weeks or months is surprisingly effective in treating depression (Stathopoulou, Powers, Berry, Smits, & Otto, 2006). Babyak and colleagues (2000) demonstrated that programmed aerobic exercise 3 times a week was as effective as treatment with antidepressive medication (Zoloft) or the combination of exercise and Zoloft after 4 months. More important, exercise was *better* at preventing relapse in the 6 months following treatment compared to the drug or combination treatment, particularly if the patients continued exercising. This general approach of focusing on activities is consistent with findings about the most powerful methods to change dysregulated emotions (see the "On the

electroconvulsive therapy (ECT) Biological treatment for severe, chronic depression involving the application of electrical impulses through the brain to produce seizures. The reasons for its effectiveness are unknown.

cognitive therapy Treatment approach that involves identifying and altering negative thinking styles related to psychological disorders such as depression and anxiety and replacing them with more positive beliefs and attitudes—and, ultimately, more adaptive behavior and coping styles.

Spectrum" box later in this chapter) (Barlow, Allen, & Choate, 2004; Campbell-Sills & Barlow, 2007), and we are likely to see more research on this approach in the near future.

Interpersonal Psychotherapy

We have seen that major disruptions in our interpersonal relationships are an important category of stresses that can trigger mood disorders (Joiner & Timmons, 2009; Kendler et al., 2003). In addition, people with few, if any, important social relationships seem at risk for developing and sustaining mood disorders (Beach et al., 2009; Sherbourne et al., 1995). **Interpersonal psychotherapy (IPT)** (Bleiberg & Markowitz, 2008; Klerman, Weissman, Rounsaville, & Chevron, 1984; Weissman, 1995) focuses on resolving problems in existing relationships and learning to form important new interpersonal relationships.

Like cognitive-behavioral approaches, IPT is highly structured and seldom takes longer than 15 to 20 sessions, usually scheduled once a week. After identifying life stressors that seem to precipitate the depression, the therapist and patient work collaboratively on the patient's current interpersonal problems. Typically, these include one or more of four interpersonal issues: *dealing with interpersonal role disputes,* such as marital conflict; *adjusting to the loss of a relationship,* such as grief over the death of a loved one; *acquiring new relationships,* such as getting married or establishing professional relationships; and *identifying and correcting deficits in social skills* that prevent the person from initiating or maintaining important relationships.

To take a common example, the therapist's first job is to identify and define an interpersonal dispute (Bleiberg & Markowitz, 2008; Weissman, 1995), perhaps with a wife who expects her spouse to support her but has had to take an outside job to help pay bills. The husband might expect the wife to share equally in generating income. If this dispute seems to be associated with the onset of depressive symptoms and to result in a continuing series of arguments and disagreements without resolution, it would become the focus for IPT.

After helping identify the dispute, the next step is to bring it to a resolution. First, the therapist helps the patient determine the stage of the dispute.

1. *Negotiation stage.* Both partners are aware it is a dispute, and they are trying to renegotiate it.
2. *Impasse stage.* The dispute smolders beneath the surface and results in low-level resentment, but no attempts are made to resolve it.
3. *Resolution stage.* The partners are taking some action, such as divorce, separation, or recommitting to the marriage.

The therapist works with the patient to define the dispute clearly for both parties and develop specific strategies for resolving it. Studies comparing the results of cognitive therapy and IPT to those of antidepressant drugs and other control conditions have found that psychological approaches and medication are equally effective immediately following treatment, and all treatments are more effective than placebo conditions, brief psychodynamic treatments, or other appropriate control conditions for both major depressive disorder and dysthymia (Beck, Hollon, Young, Bedrosian, & Budenz, 1985; Blackburn & Moore, 1997; Hollon et al., 1992; Hollon & Dimidjian, 2009; Miller, Norman, & Keitner, 1989; Paykel & Scott, 2009; Schulberg et al., 1996; Shapiro et al., 1995). Depending on how "success" is defined, approximately 50% or more of people benefit from treatment to a significant extent, compared to approximately 30% in placebo or control conditions (Craighead, Hart, Craighead, & Ilardi, 2002; Hollon & Dimidjian, 2009).

Similar results have been reported in depressed children and adolescents (Kaslow et al., 2009). In one notable clinical trial, Brent et al. (2008) demonstrated that, in over 300 severely depressed adolescents who had failed to respond to an SSRI antidepressant, CBT was significantly more effective than switching to another antidepressant. Kennard et al. (2009) showed that this was particularly true if the adolescents received at least nine sessions of the CBT.

Furthermore, studies have not found differences in treatment effectiveness based on severity of depression (Hollon et al., 1992; Hollon, Stewart, & Strunk, 2006; McLean & Taylor, 1992). For example, DeRubeis, Gelfand, Tang, and Simons (1999) carefully evaluated the effects of cognitive therapy versus medication in severely depressed patients only, across four studies, and found no advantage for one treatment or the other. O'Hara, Stuart, Gorman, and Wenzel (2000) reported positive effects for IPT in a group of women with postpartum depression, demonstrating that this approach is a worthwhile strategy in patients with postpartum depression who are reluctant to go on medication because, for example, they are breastfeeding. In an important related study, Spinelli and Endicott (2003) compared IPT to an alternative psychological approach in 50 depressed pregnant women unable to take drugs because of potential harm to the fetus. Fully 60% of these women recovered, leading the authors to recommend that IPT should be the first choice for pregnant depressed women, although it is likely that CBT would produce similar results. IPT has also been successfully administered to depressed adolescents by school-based clinicians trained to deliver IPT right in the school setting (Mufson et al., 2004). This practical approach shows good promise of reaching a larger number of depressed adolescents.

Prevention

In view of the seriousness of mood disorders in children and adolescents, work has begun on preventing these disorders in these age groups (Horowitz & Garber, 2006; Muñoz, 1993; Muñoz, Le, Clarke, Barrera, & Torres, 2009). The Institute of Medicine (IOM) delineated three types of programs: *universal* programs, which are applied to everyone; *selected* interventions, which target individuals at risk for depression because of factors such as divorce, family alcoholism, and so on; and *indicated* interventions, where the individual is already showing mild symptoms of depression (Muñoz et al., 2009). As an example of selected

interventions, Gillham, Reivich, Jaycox, and Seligman (1995) taught cognitive and social problem-solving techniques to 69 fifth- and sixth-grade children who were at risk for depression because of negative thinking styles. Compared to children in a matched no-treatment control group, the prevention group reported fewer depressive symptoms during the 2 years they were followed. More important, moderate to severe symptoms were reduced by half, and the positive effects of this program increased during the period of follow-up.

Now, results from a major clinical trial which combined "selected" and "indicated" approaches have been reported on adolescents at risk for depression (Garber et al., 2009). Three hundred sixteen adolescent offspring of parents with current or prior depressive disorders were entered into the trial and randomized to a CBT prevention program or to usual care. To be included, adolescents had to have either a past history of depression, or current depressive symptoms that would not be severe enough to meet criteria for a disorder, or both. The adolescents in the CBT prevention group received eight weekly group sessions and six monthly continuation sessions. The usual care group included a fairly active use of mental health or other health care services that, however, did not include any of the procedures used in the CBT group. The results indicated that the CBT prevention program was significantly more effective than usual care in preventing future episodes of depression but only for those adolescents whose parents were not currently in a depressive episode themselves. If the parents were in a depressive episode while the adolescents were receiving care, the adolescents became somewhat less depressed based on their own report but did not have significantly fewer depressive episodes during the follow-up period. These results are important because they show the potential power of preventive programs and that living with a depressed parent reduces the power of this preventive program to some degree (Hammen, 2009). The results also suggest that to prevent future depressive episodes it is necessary to treat depression in the whole family in a coordinated manner.

Another recent study also demonstrated that meeting in an integrated fashion with families that included parents who had a history of depression and their 9- to 15-year-old children (who were at risk because of their parents' depression) was successful in preventing depression in these families during a follow-up period (Compas et al., 2009). Additional studies have indicated that preventing depression is possible in older adults in primary care settings (van't Veer-Tazelaar et al., 2009) and also in poststroke patients, a particularly high-risk group (Reynolds, 2009; Robinson et al., 2008).

Combined Treatments

One important question is whether combining psychosocial treatments with medication is more effective than either treatment alone in treating depression or preventing relapse. In a large study reported by Keller and colleagues (2000) on the treatment of chronic major depression, 681 patients at 12 clinics around the country were assigned to receive antidepressant medication (nefazodone), a CBT constructed specifically for chronically depressed patients (CBASP, discussed earlier) (McCullough, 2000), or the combination of two treatments. Researchers found that 48% of patients receiving each of the individual treatments went into remission or responded in a clinically satisfactory way compared to 73% of the patients receiving combined treatment. Because this study was conducted with only a subset of depressed patients, those with chronic depression, the findings would need to be replicated before researchers could say combined treatment was useful for depression generally. In addition, because the study did not include a fifth condition in which the CBT was combined with placebo, we cannot rule out that the enhanced effectiveness of the combined treatment was the result of placebo factors. Nevertheless, a meta-analysis summarizing all studies conducted to date concludes that combined treatment does provide some advantage (Pampallona, Bollini, Tibaldi, Kupelnick, & Munizza, 2004). Notice how this conclusion differs from the conclusion in Chapter 4 on anxiety disorders, where no advantage of combining treatments was apparent. Combining two treatments is also expensive, however, so many experts think that a sequential strategy makes more sense, where you start with one treatment first (maybe the one the patient prefers or the one that's available) and then switch to the other only if the first choice was not entirely satisfactory (see, for example, Schatzberg et al., 2005).

Preventing Relapse

Given the high rate of recurrence in depression, it is not surprising that more than 50% of patients on antidepressant medication relapse if their medication is stopped within 4 months after their last depressive episode (Hollon, Shelton, & Loosen, 1991; Thase, 1990). Therefore, one important question has to do with **maintenance treatment** to prevent relapse or recurrence over the long term. In a number of studies, cognitive therapy reduced rates of subsequent relapse in depressed patients by more than 50% over groups treated with antidepressant medication (see, for example, Evans et al., 1992; Hollon et al., 2005, 2006; Kovacs, Rush, Beck, & Hollon, 1981; Simons, Murphy, Levine, & Wetzel, 1986; Teasdale et al., 2000).

interpersonal psychotherapy (IPT) Brief treatment approach that emphasizes resolution of interpersonal problems and stressors, such as role disputes in marital conflict, forming relationships in marriage, or a new job. It has demonstrated effectiveness for such problems as depression.
maintenance treatment Combination of continued psychosocial treatment, medication, or both designed to prevent relapse following therapy.

In one of the most impressive studies to date, patients were treated with either antidepressant medication or cognitive therapy compared to placebo (see DeRubeis et al., 2005), then the study began (Hollon et al., 2005; Hollon, Stewart, & Strunk, 2006). All patients who had responded well to treatment were followed for 2 years. During the first year, one group of patients who were originally treated with antidepressant medication continued on the medication but then stopped for the second year. A second group of patients originally receiving cognitive therapy were given up to three additional (booster) sessions during that first year but none after that. A third group was also originally treated with antidepressant medication but then switched to placebo.

Outcomes during the first year, patients who were withdrawn from medication and placed onto pill placebo were considerably more likely to relapse over the ensuing 12-month interval than were patients continued on medication (23.8% did not relapse on placebo versus 52.8% on medication). In comparison, 69.2% of patients with a history of cognitive therapy did not relapse. At this point, there was no statistically significant difference in relapse rates among patients who had received cognitive therapy versus those who continued on antidepressant medication. This suggests that prior cognitive therapy has an enduring effect that is at least as large in magnitude as keeping the patients on medications. In the second year, when all treatments had stopped, patients who had continued to receive medications during the first year were more likely to experience a recurrence (56.3%) than patients who had originally received cognitive therapy (17.5%). These studies would seem to confirm that psychological treatments for depression are most notable for their enduring ability to prevent relapse or recurrence.

Psychological Treatments for Bipolar Disorder

Although medication, particularly lithium, seems a necessary treatment for bipolar disorder, most clinicians emphasize the need for psychological interventions to manage interpersonal and practical problems (for example, marital and job difficulties that result from the disorder) (Otto & Applebaum, 2011). Until recently, the principal objective of psychological intervention was to increase compliance with medication regimens such as lithium. We noted before that the "pleasures" of a manic state make refusal to take lithium a major therapeutic obstacle. Giving up drugs between episodes or skipping dosages during an episode significantly undermines treatment. Therefore, increasing compliance with drug treatments is important (Goodwin & Jamison, 2007; Scott, 1995). For example, Clarkin, Carpenter, Hull, Wilner, and Glick (1998) evaluated the advantages of adding a psychological treatment to medication for inpatients and found it improved adherence to medication for all patients and resulted in better overall outcomes for the most severe cases compared to medication alone.

More recently, psychological treatments have also been directed at psychosocial aspects of bipolar disorder. In a new approach, Ellen Frank and her colleagues are testing a psychological treatment that regulates circadian rhythms by helping patients regulate their eating and sleep cycles and other daily schedules and cope more effectively with stressful life events, particularly interpersonal issues (Frank et al., 2005; Frank et al., 1997; Frank et al., 1999). In an evaluation of this approach, called *interpersonal and social rhythm therapy* (IPSRT), patients receiving IPSRT survived longer without a new manic or depressive episode compared to patients undergoing standard, intensive clinical management. Initial results with adolescents are also promising (Hlastala, Kotler, McClellan, & McCauley, 2010).

David Miklowitz and his colleagues found that family tension is associated with relapse in bipolar disorder. Preliminary studies indicate that treatments directed at helping families understand symptoms and develop new coping skills and communication styles do change communication styles (Simoneau, Miklowitz, Richards, Saleem, & George, 1999) and prevent relapse (Miklowitz, 2008; Miklowitz & Goldstein, 1997). Miklowitz, George, Richards, Simoneau, and Suddath (2003) demonstrated that their family-focused treatment combined with medication results in significantly less relapse 1 year following initiation of treatment than occurs in patients receiving crisis management and medication over the same period. Specifically, only 35% of patients receiving family therapy plus medication relapsed compared to 54% in the comparison group. Similarly, family therapy patients averaged over a year and a half (73.5 weeks) before relapsing, significantly longer than the comparison group. Rea, Tompson, and Miklowitz (2003) compared this approach to an individualized psychotherapy in which patients received the same number of sessions over the same period and continued to find an advantage for the family therapy after 2 years. In another important study, Lam et al. (2003) and Lam, Hayward, Watkins, Wright, and Sham (2005) showed that patients with bipolar disorders treated with cognitive therapy plus medication relapsed significantly less over both a 1-year follow-up and a 2-year follow-up compared to a control group receiving just medication. Reilly-Harrington et al. (2007) found some evidence that CBT is effective for bipolar patients with the rapid-cycling feature. In view of the relative ineffectiveness of antidepressant medication for the depressive stage of bipolar disorder reviewed earlier, Miklowitz et al. (2007) reported an important study showing that up to 30 sessions of an intensive psychological treatment was significantly more effective than usual and customary best treatment in promoting recovery from bipolar depression and remaining well. The specificity of this effect on bipolar depression, which is the most common stage of bipolar disorder, combined with the lack of effectiveness of antidepressants, suggest that these procedures will provide an important contribution to the comprehensive treatment of bipolar disorder. Otto et al. (2008a, 2008b) have synthesized these evidence-based psychological treatment procedures for bipolar disorder into a new treatment protocol.

Let us now return to Katie, who, you will remember, had made a serious suicide attempt amid a major depressive episode.

Katie The Triumph of the Self

Like the overwhelming majority of people with serious psychological disorders, Katie had never received an adequate course of treatment, although she was evaluated occasionally by various mental health professionals. She lived in a rural area where competent professional help was not readily available. Her life ebbed and flowed with her struggle to subdue anxiety and depression. When she could manage her emotions sufficiently, she took an occasional course in the high school independent study program. Katie discovered that she was fascinated by learning. She enrolled in a local community college at the age of 19 and did extremely well, even though she had not progressed beyond her freshman year in high school. At the college, she earned a high school equivalency degree. She went to work in a local factory. She continued to drink heavily and to take Valium, however; occasionally, anxiety and depression would return and disrupt her life.

Finally, Katie left home, attended college full time, and fell in love. But the romance was one-sided, and she was rejected.

> One night after a phone conversation with him, I nearly drank myself to death. I lived in a single room alone in the dorm. I drank as much vodka as quickly as I could. I fell asleep. When I awoke, I was covered in vomit and couldn't recall falling asleep or being sick. I was drunk for much of the next day. When I awoke the following morning, I realized I could have killed myself by choking on my own vomit. More importantly, I wasn't sure if I fully wanted to die. That was the last of my drinking.

Katie decided to make some changes. Taking advantage of what she had learned in the little treatment she had received, she began looking at life and herself differently. Instead of dwelling on how inadequate and evil she was, she began to pay attention to her strengths. "But I now realized that I needed to accept myself as is, and work with any stumbling blocks that I faced. I needed to get myself through the world as happily and as comfortably as I could. I had a right to that." Other lessons learned in treatment now became valuable, and Katie became more aware of her mood swings:

> I learned to objectify periods of depression as [simply] periods of "feeling." They are a part of who I am, but not the whole. I recognize when I feel that way, and I check my perceptions with someone that I trust when I feel uncertain of them. I try to hold on to the belief that these periods are only temporary.

Katie developed other strategies for coping successfully with life:

> I try to stay focused on my goals and what is important to me. I have learned that if one strategy to achieve some goal doesn't work there are other strategies that probably will. My endurance is one of my blessings. Patience, dedication, and discipline are also important. None of the changes that I have been through occurred instantly or automatically. Most of what I have achieved has required time, effort, and persistence.

Katie dreamed that if she worked hard enough she could help other people who had problems similar to her own. Katie pursued that dream and earned her PhD in psychology.

Concept Check 6.4

Indicate which type of treatment for mood disorders is being described in each statement.

1. The controversial but somewhat successful treatment involving the production of seizures through electrical current to the brain. _____
2. This teaches clients to carefully examine their thought process and recognize "depressive" styles in thinking. _____
3. These come in three main types (tricyclics, MAO inhibitors, and SSRIs) and are often prescribed but have numerous side effects. _____
4. This antidepressant must be carefully regulated to avoid illness but has the advantage of affecting manic episodes. _____
5. This therapy focuses on resolving problems in existing relationships and learning to form new interpersonal relationships. _____
6. This is an effort to prevent relapse or recurrence over the long run. _____

Suicide

› **What is the relationship between suicide and mood disorders?**

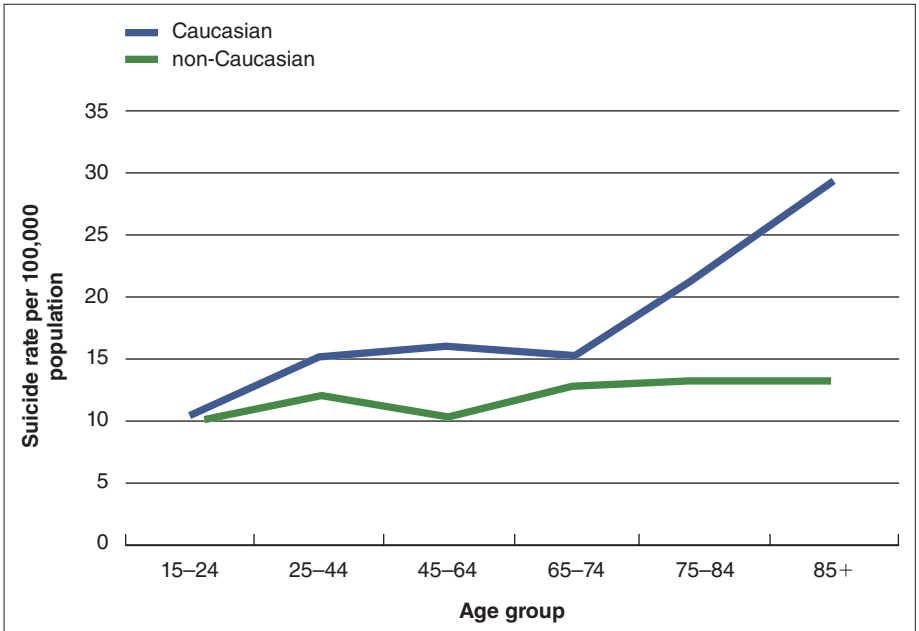
Most days we are confronted with news about the war on cancer or the frantic race to find a cure for AIDS. We also hear never-ending admonitions to improve our diet and to exercise more to prevent heart disease. But another cause of death ranks right up there with the most frightening and dangerous medical conditions. This is the inexplicable decision to kill themselves made by approximately 40,000 people a year in the United States alone.

Statistics

Suicide is officially the 11th leading cause of death in the United States (Nock, Borges, Bromet, Cha, et al., 2008), and most epidemiologists agree that the actual number of suicides may be 2 to 3 times higher than what is reported. Many of these unreported suicides occur when people deliberately drive into a bridge or off a cliff (Blumenthal, 1990). Around the world, suicide causes more deaths per year than homicide or HIV/AIDS (Nock, Borges, Bromet, Cha, et al., 2008).

Suicide is overwhelmingly a white phenomenon. Most minority groups, including African Americans and Hispanics, seldom resort to this desperate alternative, as is evident in ■ Figure 6.5. As you might expect from the incidence of depression in Native Americans, however, their suicide rate is extremely high, far outstripping the rates in other ethnic groups (Beals et al., 2005; Hasin et al., 2005; Nock, Borges, Bromet, Cha, et al., 2008); although there is great variability across tribes (Berlin, 1987). Even more alarming is the dramatic increase in death by suicide beginning in adolescence. In the United States, rates of death by suicide per 100,000 people rise from 1.29 in the 10–14 age group to 12.35 in the 20–24 age group (Centers for Disease Control and Prevention [CDC], 2010b; Nock, Cha, & Dour, 2011). For teenagers, suicide was the *third* leading cause of death behind unintentional injury such as motor vehicle accidents and homicide in 2007 (CDC, 2010b; Minino et al., 2002; Ventura, Peters, Martin, & Maurer, 1997). Prevalence differs greatly depending on ethnic group, as is evident in Figure 6.5. This fact underscores the importance of attending to cultural considerations in the prevention and treatment of adolescent suicide (Goldston et al., 2008).

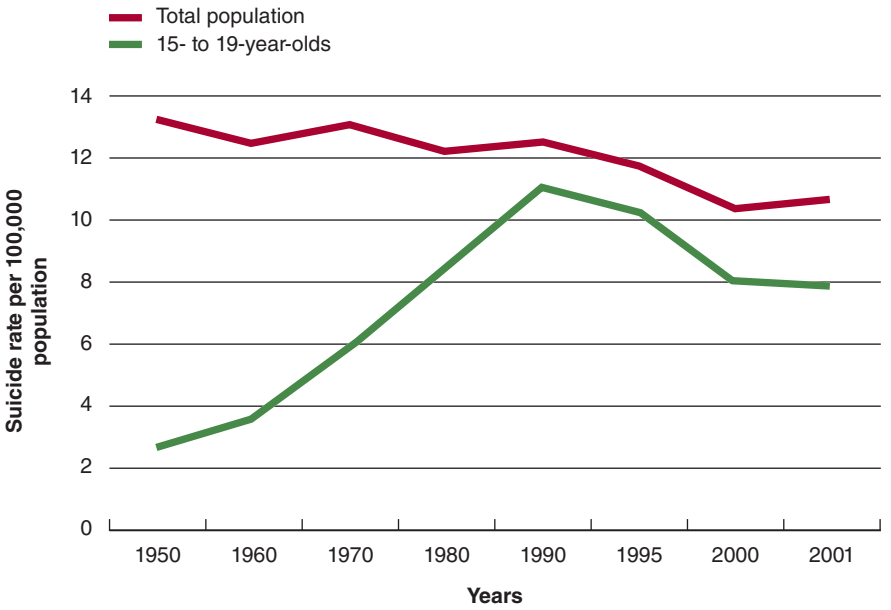
Note also the dramatic increase in suicide rates among the elderly compared to the rates for younger age groups shown in ■ Figure 6.6. This rise has been connected to the growing incidence of medical illness in our oldest citizens and to their increasing loss of social support (Conwell, Duberstein, & Caine, 2002), and resulting depression (Fiske et al., 2009). As we have noted, a strong relationship exists between illness or infirmity and hopelessness or depression.



■ **Figure 6.5** United States suicide rates for Caucasians and non-Caucasians by age group, sexes combined (data from the National Center for Health Statistics, Vital Statistics of the United States, 2001). (From Centers for Disease Control and Prevention, 2003. Deaths: Final data for 2001. *National Vital Statistics Reports,* 52(3). Hyattsville, MD: National Center for Health Statistics.)

Regardless of age, in every country around the world except China males are 4 times more likely to *commit* suicide than females (CDC, 2010b; Nock et al., 2011; World Health Organization, 2010). This startling fact seems to be related partly to gender differences in the types of suicide *attempts*. Males generally choose far more violent methods, such as guns and hanging; females tend to rely on less violent options, such as drug overdose (Gallagher-Thompson & Osgood, 1997; Nock et al., 2011). More men commit suicide during old age and more women during middle age, partly because most attempts by older women are unsuccessful (Berman, 2009; Kuo, Gallo, & Tien, 2001).

Uniquely in China more women commit suicide than men, particularly in rural settings (Murray, 1996; Murray & Lopez, 1996; Nock, Borges, Bromet, Cha, et al., 2008; Phillips, Li, & Zhang, 2002). What accounts for this culturally determined reversal? Chinese scientists agree that China's suicide rates, probably the highest in the world, are the



■ **Figure 6.6** Suicide rates per 100,000 in population for total population and for 15 to 19 year olds (data from the National Center for Health Statistics, Vital Statistics of the United States, 2001). (From Centers for Disease Control and Prevention, 2003a. Deaths: Final data for 2001. *National Vital Statistics Reports,* 52(3). Hyattsville, MD: National Center for Health Statistics.)

result of an absence of stigma. Suicide, particularly among women, is often portrayed in classical Chinese literature as a reasonable solution to problems. A rural Chinese woman's family is her entire world, and suicide is an honorable solution if the family collapses. Furthermore, highly toxic farm pesticides are readily available and it is possible that many women who did not necessarily intend to kill themselves die after accidentally swallowing poison.

In addition to completed suicides, three other important indices of suicidal behavior are **suicidal ideation** (thinking seriously about suicide), **suicidal plans** (the formulation of a specific method for killing oneself), and **suicidal attempts** (the person survives) (Kessler, Berglund, Borges, Nock, & Wang, 2005; Nock et al., 2011). Also, Nock and Kessler (2006) distinguish "attempters" (self-injurers with the intent to die) from "gesturers" (self-injurers who intend not to die but to influence or manipulate somebody or communicate a cry for help). In a carefully done cross-national study using consistent definitions, the prevalence of suicide ideation has been estimated at 9.2%; 3.1% reported a suicide plan and 2.7% attempted suicide during their lifetime (Nock, Borges, Bromet, Alonso et al., 2008). Although males *commit* suicide more often than females in most of the world, females *attempt* suicide at least 3 times as often (Berman & Jobes, 1991; Kuo et al., 2001). And the overall rate of nonlethal suicidal thoughts, plans, and (unsuccessful) attempts is 40% to 60% higher in women than in men (Nock et al., 2011). This high incidence may reflect that more women than men are depressed and that depression is strongly related to suicide attempts (Berman, 2009). It is also interesting that despite the much higher rate of completed suicides among whites, there are no significant ethnic or racial differences in rates of suicide ideation, plans, or attempts (Kessler et al., 2005). Among adolescents the ratio of *thoughts* about suicide to *attempts* is also between 3:1 and 6:1. In other words, between 16% and 30% of adolescents who think about killing themselves actually attempt it (Kovacs, Goldston, & Gatsonis, 1993; Nock, Borges, Bromet, Cha, et al., 2008). "Thoughts" in this context do not refer to a fleeting philosophical type of consideration but rather to a serious contemplation of the act. The first step down the dangerous road to suicide is thinking about it.

In a study of college students (among whom suicide is the second leading cause of death), approximately 10% to 25% had thoughts about suicide during the past 12 months (Brener, Hassan, & Barrios, 1999; Meehan, Lamb, Saltzman, & O'Carroll, 1992; Schwartz & Whitaker, 1990). Only a minority of these college students with thoughts of suicide (perhaps around 15%) attempt to kill themselves, and only a few succeed (Kovacs et al., 1993). Nevertheless, given the enormity of the problem, suicidal thoughts are taken seriously by mental health professionals.

suicidal ideation Serious thoughts about committing suicide.
suicidal plans The formulation of a specific method of killing oneself.
suicidal attempts Effort made to kill oneself.

AP Photo/Mark J. Terrill

▲ Men often choose violent means of suicide. Nirvana's Kurt Cobain shot himself.

Causes

The great sociologist Emile Durkheim (1951) defined a number of suicide types based on the social or cultural conditions in which they occurred. One type is "formalized" suicides that were approved of, such as the ancient custom of *harakiri* in Japan, in which an individual who brought dishonor to himself or his family was expected to impale himself on a sword. Durkheim referred to this as *altruistic suicide*. Durkheim also recognized the loss of social supports as an important provocation for suicide; he called this *egoistic suicide*. (Older adults who kill themselves after losing touch with their friends or family fit into this category.) Magne-Ingvar, Ojehagen, and Traskman-Bendz (1992) found that only 13% of 75 individuals who had seriously attempted suicide had an adequate social network of friends and relationships. *Anomic suicides* are the result of marked disruptions, such as the sudden loss of a high-prestige job. (*Anomie* is feeling lost and confused.) Finally, *fatalistic suicides* result from a loss of control over one's own destiny. The mass suicide of 39 Heaven's Gate cult members in 1997 is an example of this type. Durkheim's work was important in alerting us to the social contribution to suicide. Sigmund Freud (1917/1957) believed that suicide (and depression, to some extent) indicated unconscious hostility directed inward to the self rather than outward to the person or situation causing the anger. Indeed, suicide victims often seem to be psychologically "punishing" others who may have rejected them or caused some other personal hurt. Current thinking considers social and psychological factors but also highlights the potential importance of biological contributions.

Risk Factors

Edward Shneidman pioneered the study of risk factors for suicide (Shneidman, 1989; Shneidman, Farberow, & Litman, 1970). Among the methods he and others have used to study those conditions and events that make a person vulnerable is **psychological autopsy**. The psychological profile of the person who committed suicide is reconstructed through extensive interviews with friends and family members who are likely to know what the individual was thinking and doing in the period before death. This and other methods have allowed researchers to identify a number of risk factors for suicide.

Family History

If a family member committed suicide, there is an increased risk that someone else in the family will also (Berman, 2009; Kety, 1990; Mann, Waternaux, Haas, & Malone, 1999; Mann et al., 2005; Nock et al., 2011). Brent and colleagues (2002) noted that offspring of family members who had attempted suicide had 6 times the risk of suicide attempts compared to offspring of nonattempters. If a sibling was also a suicide attempter, the risk increased even more (Brent et al., 2003). This may not be surprising because so many people who kill themselves are depressed or have some related mental disorder, and these disorders run in families (Nock et al., 2011). Nevertheless, the question remains: Are people who kill themselves simply adopting a familiar solution that they've witnessed in family members, or does an inherited trait, such as impulsivity, account for increased suicidal behavior in families? It seems both factors may contribute. If individuals have an early onset of their mood disorder, and aggressive or impulsive traits, then their families are at a greater risk for suicidal behavior (Mann et al., 2005). The possibility that something is inherited is also supported by several adoption studies. One found an increased rate of suicide in the biological relatives of adopted individuals who had committed suicide compared to a control group of adoptees who had not committed suicide (Nock et al., 2011; Schulsinger, Kety, & Rosenthal, 1979; Wender et al., 1986). This suggests some biological (genetic) contribution to suicide, even if it is relatively small, although it may not be independent of genetic contribution to depression or associated disorders.

Neurobiology

A variety of evidence suggests that low levels of serotonin may be associated with suicide and with violent suicide attempts (Asberg, Nordstrom, & Traskman-Bendz, 1986; Cremniter et al., 1999; Winchel, Stanley, & Stanley, 1990). As we have noted, extremely low levels of serotonin are associated with impulsivity, instability, and the tendency to overreact to situations. It is possible then that low levels of serotonin may contribute to creating a vulnerability to act impulsively. This may include killing oneself, which is sometimes an impulsive act. The studies by Brent and colleagues (2002) and Mann and colleagues (2005) suggest

Discussing Diversity

Suicide Behaviors around the World

Suicide is one of the leading causes of death worldwide, responsible for approximately 1 million deaths per year and 1 death every 40 seconds (Nock, Borges, Bromet, Cha, et al., 2008). Although suicide is a leading cause of death, it is difficult to predict and prevent, partly because the person who dies by suicide obviously can no longer provide information about the factors leading up to the death. In addition, many people who die by suicide leave behind few clues about the factors that influenced their decision to end their life. In an effort to better understand and predict suicide, researchers now often study people who have experienced suicidal thoughts and have made nonlethal suicide attempts. The results of such studies have provided interesting information about suicidal behaviors around the world.

Data from a recent survey of people in 17 countries indicate that approximately 9% of all adults report that they have seriously considered suicide at some point in their life, 3% have made an actual suicide plan, and just under 3% have made a suicide attempt (Nock, Borges, Bromet, Alonso, et al., 2008). However, the rate of suicidal thoughts and attempts varies significantly across countries. For instance, only 3% of people in Italy have had suicidal thoughts, compared to 16% of people in New Zealand. Researchers are still trying to understand why these rates are so dissimilar across countries. Of note, once people report that they have had suicidal thoughts or made a suicide attempt, the characteristics look quite similar across countries. For instance, in every country examined, the rate of suicidal thoughts sharply increases during adolescence, approximately one third of those with suicidal thoughts go on to make a suicide attempt, and more than 60% of those who do attempt suicide do so within the first year after they initially had suicidal thoughts. In addition, risk factors for suicidal thoughts and attempts are quite consistent cross-nationally and include being female, younger, less educated, and unmarried and having a mental disorder (Nock, Borges, Bromet, Alonso, et al., 2008). Unfortunately, although an increasing number of risk factors for suicidal behaviors have been identified, to date no theories of suicide have been able to tie them together into a clear model that allows us to accurately predict who is most at risk and how to prevent them from engaging in suicidal behavior.

that transmission of vulnerabilities for a mood disorder, including the trait of impulsivity, may mediate family transmission of suicide attempts.

Existing Psychological Disorders and Other Psychological Risk Factors

More than 80% of people who kill themselves suffer from a psychological disorder, usually mood, substance use, or impulse control disorders (Berman, 2009; Brent & Kolko, 1990; Conwell et al., 1996; Joe, Baser, Breeden, Neighbors, & Jackson, 2006; Nock, Hwang, Sampson, & Kessler, 2009). Suicide is often associated with mood disorders—and for good reason. As many as 60% of suicides (75% of adolescent suicides) are associated with an existing mood disorder (Berman, 2009; Brent & Kolko, 1990; Oquendo et al., 2004). Many people with mood disorders do not attempt suicide, however, and, conversely, many people who attempt suicide do not have mood disorders. Therefore, depression and suicide, although strongly related, are still independent. Looking more closely at the relationship of mood disorder and suicide, some investigators have isolated hopelessness, a specific component of depression, as strongly predicting suicide (Beck, 1986; Beck, Steer, Kovacs, & Garrison, 1985; Goldston, Reboussin, & Daniel, 2006). A recent important theoretical account of suicide termed the "interpersonal theory of suicide" cites a perception of oneself as a burden on others and a diminished sense of belonging as powerful predictors of hopelessness and subsequently suicide (van Orden et al., 2010).

Alcohol use and abuse are associated with approximately 25% to 50% of suicides and are particularly evident in adolescent suicides (Berman, 2009; Brener et al., 1999; Conwell et al., 1996; Hawton, Houston, Haw, Townsend, & Harriss, 2003; Woods et al., 1997). Brent and colleagues (1988) found that about one third of adolescents who commit suicide were intoxicated when they died and that many more might have been under the influence of drugs. Combinations of disorders, such as substance abuse and mood disorders in adults or mood disorders and conduct disorder in children and adolescents, seem to create a stronger vulnerability than any one disorder alone (Conwell et al., 1996; Nock et al., 2009; Woods et al., 1997). For example, Nock and colleagues (2009) noticed that depression alone did not predict suicidal ideation or attempts, but depression combined with impulse control problems and anxiety/agitation did. For adolescents, Woods and colleagues (1997) also found that substance abuse with other risk-taking behaviors, such as getting into fights, carrying a gun, or smoking, were predictive of teenage suicide, possibly reflecting impulsivity in these troubled adolescents. Esposito and Clum (2003) also noted that the presence of anxiety and mood disorders predicted suicide attempts in adolescents. Past suicide attempts are another strong risk factor and must be taken seriously (Berman, 2009). Cooper and colleagues (2005) followed almost 8,000 individuals who were treated in the emergency room for deliberate self-harm for up to 4 years. Sixty of these people had killed themselves, which is 30 times the risk compared to population statistics.

psychological autopsy Postmortem psychological profile of a suicide victim constructed from interviews with people who knew the person before death.

A disorder characterized more by impulsivity than depression is borderline personality disorder (see Chapter 11). Individuals with this disorder, known for making manipulative and impulsive suicidal gestures without necessarily wanting to destroy themselves, sometimes kill themselves by mistake in as many as 10% of the cases. The combination of borderline personality disorder and depression is particularly deadly (Soloff, Lynch, Kelly, Malone, & Mann, 2000).

The association of suicide with severe psychological disorders, especially depression, belies the myth that it is a response to disappointment in people who are otherwise healthy.

Stressful Life Events

Perhaps the most important risk factor for suicide is a severe, stressful event experienced as shameful or humiliating, such as a failure (real or imagined) in school or at work, an unexpected arrest, or rejection by a loved one (Blumenthal, 1990; Conwell et al., 2002; Joiner & Rudd, 2000). Physical and sexual abuse are also important sources of stress (Wagner, 1997). Evidence confirms that the stress and disruption of natural disasters increase the likelihood of suicide (Krug et al., 1998). Based on data from 337 countries experiencing natural disasters in the 1980s, the authors concluded that the rates of suicide increased 13.8% in the 4 years after severe floods, 31% in the 2 years after hurricanes, and 62.9% in the first year after an earthquake. Given preexisting vulnerabilities—including psychological disorders, traits of impulsiveness, and lack of social support—a stressful event can often put a person over the edge. An integrated model of the causes of suicidal behavior is presented in ■ Figure 6.7.

Is Suicide Contagious?

Most people react to hearing the news of a suicide with sadness and curiosity. Some people react by attempting suicide themselves, often by the same method they have just heard about. Gould (1990) reported an increase in suicides during a 9-day period after widespread publicity about a suicide. Clusters of suicides (several people copying one person) seem to predominate among teenagers, with as many as 5% of all teenage suicides reflecting an imitation (Gould, 1990; Gould, Greenberg, Velting, & Shaffer, 2003).

Why would anyone want to copy a suicide? First, suicides are often romanticized in the media: An attractive young person under unbearable pressure commits suicide and becomes a martyr to friends and peers by getting even with the (adult) world for creating such a difficult situation. Also, media accounts often describe in detail the methods used in the suicide, thereby providing a guide to potential victims. Little is reported about the paralysis, brain damage, and other tragic consequences of the incomplete or failed suicide or about how suicide is almost always associated with a severe psychological disorder. More important, even less is said about the futility of this method of solving problems (Gould, 1990, 2001; O'Carroll, 1990). To prevent these tragedies, mental health professionals must intervene immediately in schools and other locations with people who might be depressed or otherwise vulnerable to the contagion of suicide. But it isn't clear that suicide is "contagious" in the infectious disease sense. Rather, the stress of a friend's suicide or some other major stress may affect several individuals who are vulnerable because of existing psychological disorders (Joiner, 1999).

Treatment

Despite the identification of important risk factors, predicting suicide is still an uncertain art. Individuals with few precipitating factors unexpectedly kill themselves, and many who live with seemingly insurmountable stress and illness and have little social support or guidance somehow survive and overcome their difficulties.

Mental health professionals are thoroughly trained in assessing for possible suicidal ideation (Joiner et al., 2007). Others might be reluctant to ask leading questions for fear of putting the idea in someone's head. However, we know it is far more important to check for these "secrets" than to do nothing because the risk of inspiring suicidal thoughts is small to nonexistent and the risk of leaving them undiscovered is enormous (Berman, 2009). Gould and colleagues (2005) found that more than 1,000 high school students who were asked about suicidal thoughts or behaviors during a screening program showed no risk of increased suicidal thoughts compared to a second group of 1,000 students who had the screening program without the questions about suicide. Therefore, if there is any indication that someone is suicidal, the mental health professional will inquire, "Has there been any time recently when you've thought that life wasn't worth living or had some thoughts about hurting yourself or possibly killing yourself?"

One difficulty with this approach is that sometimes these thoughts are implicit or out of awareness. Now Cha, Najmi, Park, Finn, and Nock (2010) have developed measures of implicit (unconscious) cognition, adapted from the labs of cognitive psychology, to assess implicit suicidal ideation. In this assessment using the Stroop test described in Chapter 2, people who demonstrated an implicit association between the words *death/suicide* and *self,* even if they weren't aware of it, were 6 times more likely to make a suicide attempt in the next 6 months than those without this specific association; thus, this assessment is a better predictor of suicide attempts than both patients' own predictions and clinicians' predictions (Nock et al., 2010). The mental health professional will also check for possible recent humiliations and determine whether any of the factors are present that might indicate a high probability of suicide. For example, does a person who is thinking of suicide have a detailed plan or just a vague fantasy? If a plan is discovered that includes a specific time, place, and method, the risk is high. Does the detailed plan include putting all personal affairs in order, giving away possessions, and other final acts? If so, the risk is higher still. What specific method is the person considering? Generally, the more lethal and violent the method (guns, hanging, poison, and so on), the greater the risk it will be used. Does the person understand what might actually happen? Many people do not understand the effects of the pills on which they might overdose. Finally, has the person taken any precautions against being discovered? If so, the risk is extreme (American Psychiatric Association, 2003).

If a risk is present, clinicians attempt to get the individual to agree to or even sign a no-suicide contract. Usually this includes a promise not to do anything remotely connected with suicide without contacting the mental health professional first. If the person at risk refuses a contract (or the clinician has serious doubts about the patient's sincerity) and the suicidal risk is judged to be high, immediate hospitalization is indicated, even against the will of the patient. Whether the person is hospitalized or not, treatment aimed at resolving underlying life stressors and treating existing psychological disorders should be initiated immediately.

In view of the public health consequences of suicide, a number of programs have been implemented to reduce the rates of suicide. Most research indicates that such curriculum-based programs targeting the general population (universal programs) in schools or organizations on how to handle life stress or increase social support are not effective (Berman, 2009; Garfield & Zigler, 1993; Shaffer, Garland, Vieland, Underwood, & Busner, 1991). More helpful are programs targeted to at-risk individuals, including adolescents in schools where a student has committed suicide. The Institute of Medicine (2002) recommends making services available immediately to friends and relatives of victims. An important step is limiting access to lethal weapons for anyone at risk for suicide. A recent analysis suggests that this may be the most powerful part of a suicide prevention program (Mann et al., 2005). Telephone hotlines and other crisis intervention services also seem to be useful. Nevertheless, as Garfield and Zigler (1993) point out, hotline volunteers must be backed up by competent mental health professionals who can identify potentially serious risks. One large health maintenance organization carefully screened all of its 200,000 members who came in for services for suicide risk and then intervened if any risk was noted. Suicides were greatly reduced in the very promising program (Hampton, 2010).

Specific treatments for people at risk have also been developed. For example, Salkovskis, Atha, and Storer (1990) treated 20 patients at high risk for repeated suicide attempts with a cognitive-behavioral problem-solving approach. Results indicated that they were significantly less likely to attempt suicide in the 6 months following treatment. Marsha Linehan and colleagues developed a noteworthy treatment for the type of impulsive suicidal behavior associated with borderline personality disorder (see Chapter 11). David Rudd and colleagues developed a brief psychological treatment targeting young adults who were at risk for suicide because of the presence of suicidal ideation accompanied by previous suicidal attempts, mood or substance use disorders, or both (Rudd et al., 1996). They randomly assigned 264 young people either to this new treatment or to treatment as usual in the community. Patients undergoing the new treatment spent approximately 9 hours each day for 2 weeks at a hospital treatment facility. Treatment consisted of problem solving, developing social competence, coping more adaptively with life's problems, and recognizing emotional and life experiences that may have precipitated the suicide attempt or ideation. Patients were assessed up to 2 years following treatment, and results indicated reductions

in suicidal ideation and behavior, and marked improvement in problem-solving ability. Furthermore, the brief experimental treatment was significantly more effective at retaining the highest-risk young adults in the program. This program has been expanded into a psychological treatment for suicidal behavior with empirical support for its efficacy (Rudd, Joiner, & Rajab, 2001). One of the more important studies to date has demonstrated that 10 sessions of cognitive therapy for recent suicide attempters cuts the risk of additional attempts by 50% over the next 18 months (Brown et al., 2005). Specifically 24% of those in the cognitive therapy group made a repeat attempt compared to 42% in the care-as-usual group. Because cognitive therapy is relatively widely available, this is an important development in suicide prevention.

Concept Check 6.5

Match each of the following summaries with the correct suicide type, choosing from (a) altruistic, (b) egoistic, (c) anomic, and (d) fatalistic.

1. Ralph's wife left him and took the children. He is a well-known television personality, but, because of a conflict with the new station owners, he was recently fired. If Ralph kills himself, his suicide would be considered _____.
2. Sam killed himself while a prisoner of war in Vietnam. _____
3. Sheiba lives in a remote village in Africa. She was recently caught in an adulterous affair with a man in a nearby village. Her husband wants to kill her but won't have to because of a tribal custom that requires her to kill herself. She leaps from the nearby "sinful woman's cliff." _____
4. Mabel lived in a nursing home for many years. At first, her family and friends visited her often; now they come only at Christmas. Her two closest friends in the nursing home died recently. She has no hobbies or other interests. Mabel's suicide would be identified as what type? _____

On the Spectrum A New Unified Transdiagnostic Treatment for Emotional Disorders

The mood disorders discussed in this chapter and the anxiety disorders discussed in Chapter 4 are often lumped together under the term "emotional disorders." This is because these disorders have many common features, including difficulty in managing intense emotional experiences. This difficulty is often referred to as "emotion dysregulation." It is also the case that these groups of disorders share common risk factors including the same set of genetic risk factors (described earlier). They are also highly comorbid—that is, many people with an anxiety disorder also have, or have had, depressive disorders, and most people with depression either have, or have had, an anxiety disorder. The presence of significant anxiety is associated with more severe forms of depression and bipolar disorders and a longer course and poorer outcomes from treatment (Coryell et al., 2009; Fava et al., 2008). Individuals with both anxiety and mood disorders experience more frequent and intense negative moods than healthy individuals and view these experiences as much more aversive. They also experience similar negative cognitive processes.

Psychological treatments for the mood and anxiety disorders differ from disorder to disorder, but all share common components. These include changing negative attitudes, cognitive styles, and attributions; preventing avoidance of situations or experiences that might provoke intense emotions such as anxiety or depression; and encouraging therapeutic activities and exposure to experiences that trigger strong emotions. Recognizing these advances, we have developed a new unified transdiagnostic psychological treatment in one of our clinics that is designed to be applicable to anyone suffering from emotional disorders even if they have more than one emotional disorder (comorbidity), unusual variations of an anxiety or mood disorder, or for some reason don't quite meet the criteria for one of the diagnostic categories of anxiety and mood disorders (Barlow et al., 2011a, 2011b). The term "unified" is used because it integrates principles that are relevant to all emotional disorders. The new treatment, called the unified protocol (UP) for *Transdiagnostic Treatment of Emotional Disorders* (Barlow et al., 2011a, 2011b), consists of five core modules: (1) increasing awareness of emotional experiences (because people with emotional disorders are uncomfortable with their emotions and often try to suppress or ignore them); (2) encouraging greater flexibility in appraisals and attributions concerning emotional situations (because people with anxiety and mood disorders usually just think the worst when they are experiencing intense emotions); (3) identifying and preventing tendencies to avoid certain situations and intense emotions (because patients are often unaware of many of their avoidant tendencies such as trying to distract themselves when they are feeling anxiety); (4) engaging patients in exercises designed to evoke physical sensations analogous to those typically associated with their anxiety and distress; and (5) encouraging patients to increase their tolerance of intense

or uncomfortable emotions through exposure to both the situational cues or triggers as well as their own internal physical cues associated with intense emotions. Preliminary results show that this treatment is effective across a broad range of disorders (Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010). If these results hold up upon further testing, then the UP should eliminate the necessity for applying a different treatment to every single variation of an emotional disorder and, perhaps, be more effective across a broad range of emotional disorders.

Other recent research is making the idea of transdiagnostic treatment more interesting. Researchers have found that the most important function of antidepressant drugs may not be changes in neurotransmitter activity, although this obviously occurs, but rather changes in the neuropsychological process of regulating emotional reactions that seem to occur very soon after beginning antidepressant drugs and before full therapeutic effect is noted (Harmer, 2010). If this is the case, the fundamental mechanism of action of transdiagnostic psychological treatments, and antidepressant drugs (which work equally well in anxiety disorders) may be more similar than different in that they both target emotion dysregulation.

Summary

Understanding and Defining Mood Disorders

What is the difference between a depressive episode and a manic or hypermanic episode?

- Mood disorders are among the most common psychological disorders, and the risk of developing them is increasing worldwide, particularly in younger people.
- Two fundamental experiences can contribute either singly or in combination to all specific mood disorders: a major depressive episode and mania. A less severe episode of mania that does not cause impairment in social or occupational functioning is known as a hypomanic episode. An episode of mania coupled with anxiety or depression is known as a dysphoric manic or mixed episode.
- An individual who suffers from episodes of depression only is said to have a unipolar disorder. An individual who alternates between depression and mania has a bipolar disorder.

What are the clinical symptoms of major depressive disorder, dysthymic disorder, and bipolar disorder?

- Major depressive disorder may be a single episode or recurrent, but it is always time limited; in another form of depression, dysthymic disorder, the symptoms are somewhat milder but remain relatively unchanged over long periods. In cases of double depression, an individual experiences both depressive episodes and dysthymic disorder.
- Approximately 20% of bereaved individuals may experience a pathological, complicated grief reaction in which the normal grief response develops into a full-blown mood disorder.
- The key identifying feature of bipolar disorders is an alternation of manic episodes and major depressive episodes. Cyclothymic disorder is a milder but more chronic version of bipolar disorder.
- Patterns of additional features that sometimes accompany mood disorders, called specifiers, may predict the course or patient response to treatment, as does the temporal patterning or course of mood disorders. One pattern, seasonal affective disorder, most often occurs in winter.

Prevalence of Mood Disorders

How does the prevalence of mood disorders vary across a life span?

- Mood disorders in children are fundamentally similar to mood disorders in adults.
- Symptoms of depression are increasing dramatically in our elderly population.
- The experience of anxiety across cultures varies, and it can be difficult to make comparisons, especially, for example, when we attempt to compare subjective feelings of depression.

Causes of Mood Disorders

What biological, psychological, and sociocultural factors contribute to the development of mood disorders?

- The causes of mood disorders lie in a complex interaction of biological, psychological, and social factors. From a biological perspective, researchers are particularly interested in the stress hypothesis and the role of neurohormones. Psychological theories of depression focus on learned helplessness, the depressive cognitive schemas, and interpersonal disruptions.

Treatment of Mood Disorders

What medical and psychological treatments have been successful in treating mood disorders?

- A variety of treatments, both biological and psychological, have proved effective for the mood disorders, at least in the short term. For those individuals who do not respond to antidepressant drugs or psychosocial treatments, a more dramatic physical treatment, electroconvulsive therapy, is sometimes used. Two psychosocial treatments—cognitive therapy and interpersonal therapy—seem effective in treating depressive disorders.
- Relapse and recurrence of mood disorders are common in the long term, and treatment efforts must focus on maintenance treatment—that is, on preventing relapse or recurrence.

Suicide

What is the relationship between suicide and mood disorders?

› Suicide is often associated with mood disorders but can occur in their absence or in the presence of other disorders. It is the 11th leading cause of death, but among adolescents, it is the 3rd leading cause of death.

› In understanding suicidal behavior, three indices are important: suicidal ideation (serious thoughts about committing suicide), suicidal plans (a detailed method for killing oneself), and suicidal attempts (that are not successful). Important, too, in learning about risk factors for suicides is the psychological autopsy, in which the psychological profile of an individual who has committed suicide is reconstructed and examined for clues.

Key Terms

mood disorders, 202
major depressive episode, 202
mania, 203
hypomanic episode, 203
mixed manic episode or dysphoric manic episode, 203
major depressive disorder, single or recurrent episode, 204
dysthymic disorder, 204
double depression, 205
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seasonal affective disorder (SAD), 207
pathological or impacted grief reaction, 207
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bipolar I disorder, 208
cyclothymic disorder, 209
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learned helplessness theory of depression, 220
depressive cognitive triad, 221
mood-stabilizing drug, 229
electroconvulsive therapy (ECT), 230
cognitive therapy, 230
interpersonal psychotherapy (IPT), 232
maintenance treatment, 233
suicidal ideation, 237
suicidal plans, 237
suicidal attempts, 237
psychological autopsy, 238

Answers to Concept Checks

6.1

1. e; 2. a; 3. c; 4. d; 5. b

6.2

1. T; 2. F (it does not require life experience); 3. T; 4. T

6.3

1. genetics, neurotransmitter system abnormalities, endocrine system, circadian or sleep rhythms, neurohormones
2. stressful life events, learned helplessness, depressive cognitive triad, a sense of uncontrollability
3. marital dissatisfaction, gender, few social supports

6.4

1. electroconvulsive therapy; 2. cognitive therapy; 3. antidepressants; 4. lithium; 5. interpersonal psychotherapy; 6. maintenance treatment

6.5

1. c; 2. d; 3. a; 4. b

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› *Evelyn, a Patient with Major Depressive Disorder:* Evelyn has a major depressive disorder that gives a more positive view of long-term prospects for change.
› *Mary, a Client with Bipolar Disorder:* Mary is shown in both a manic and depressive phase of her illness. You may notice the similarity of the delusions in both phases of her illness.

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- Dysthymia
- Double Depression
- Bipolar I Disorder
- Bipolar II Disorder
- Cyclothymic Disorder
- Concept Check: Dysthymia Versus Major Depression
- Mood Disorders: Course Specifiers
- Learned Helplessness
- Electroconvulsive Therapy (ECT)
- Suicide

Chapter Quiz

1. An individual who is experiencing an elevated mood, a decreased need for sleep, and distractibility is most likely experiencing:
   a. panic disorder
   b. mania
   c. depersonalization
   d. hallucinations
2. What is the general agreement among mental health professionals about the relationship between bereavement and depression?
   a. Bereavement is less severe than depression in all cases.
   b. Depression can lead to bereavement in many cases.
   c. Bereavement can lead to depression in many cases.
   d. Symptoms of bereavement and depression rarely overlap.
3. Bipolar I disorder is characterized by _____, whereas bipolar II is characterized by _____.
   a. full manic episodes; hypomanic episodes
   b. hypomanic episodes; full manic episodes
   c. both depressive and manic episodes; full manic episodes
   d. full manic episodes; both depressive and manic episodes
4. Treatment for bereavement often includes:
   a. finding meaning in the loss
   b. replacing the lost person with someone else
   c. finding humor in the tragedy
   d. replacing sad thoughts about the lost person with more happy thoughts
5. Which statement best characterizes the relationship between anxiety and depression?
   a. Anxiety usually precedes the development of depression.
   b. Depression usually precedes the development of anxiety.
   c. Almost all depressed patients are anxious, but not all anxious patients are depressed.
   d. Almost all anxious patients are depressed, but not all depressed patients are anxious.
6. Which theory suggests that depression occurs when individuals believe that they have no control over the circumstances in their lives?
   a. attribution theory
   b. learned helplessness
   c. social learning theory
   d. theory of equifinality
7. In treating depressed clients, a psychologist helps them think more positively about themselves, about their place in the world, and about the prospects for the future. This psychologist is basing her techniques on whose model of depression?
   a. Sigmund Freud
   b. Carl Rogers
   c. Rollo May
   d. Aaron Beck
8. Maintenance treatment for depression can be important because it can prevent:
   a. transmission
   b. bereavement
   c. incidence
   d. relapse
9. Which of the following explains why some people refuse to take medications to treat their depression or take those medications and then stop?
   a. The medications are in short supply and are unavailable.
   b. The medications don't work for most people.
   c. For some people the medications cause serious side effects.
   d. The medications work in the short term but not the long term.
10. Which of the following is a risk factor for suicide?
    a. having a relative who committed suicide
    b. playing aggressive, full-contact sports
    c. a history of multiple marriages
    d. an abstract, philosophical cognitive style

(See Appendix A for answers.)

Exploring Mood Disorders

People with mood disorders experience one or both of the following:

- **Mania:** A frantic "high" with extreme overconfidence and energy, often leading to reckless behavior
- **Depression:** A devastating "low" with extreme lack of energy, interest, confidence, and enjoyment of life

- Negative or positive life changes (death of a loved one, promotion, etc.)
- Physical illness

Trigger

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Social Influences

- Women and minorities—social inequality and oppression and a diminished sense of control
- Social support can reduce symptoms
- Lack of social support can aggravate symptoms

Biological Influences

- Inherited vulnerability
- Altered neurotransmitters and neurohormonal systems
- Sleep deprivation
- Circadian rhythm disturbances

Causes

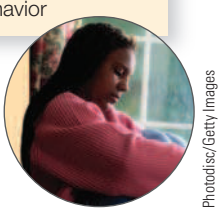
Behavioral Influences

Depression

- General slowing down
- Neglect of responsibilities and appearance
- Irritability; complaints about matters that used to be taken in stride

Mania

- Hyperactivity
- Reckless or otherwise unusual behavior



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Emotional and Cognitive Influences

Depression

- Emotional flatness or emptiness
- Inability to feel pleasure
- Poor memory
- Inability to concentrate
- Hopelessness and/or learned helplessness
- Loss of sexual desire
- Loss of warm feelings for family and friends
- Exaggerated self-blame or guilt
- Overgeneralization
- Loss of self-esteem
- Suicidal thoughts or actions

Mania

- Exaggerated feelings of euphoria and excitement

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Disorder

TYPES OF MOOD DISORDERS

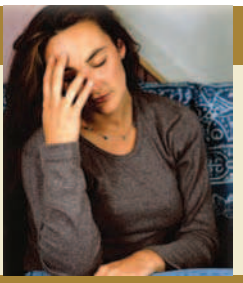
Depressive

Major Depressive Disorder
Symptoms of major depressive disorder:
- begin suddenly, often triggered by a crisis, change, or loss
- are extremely severe, interfering with normal functioning
- can be long term, lasting months or years if untreated

Some people have only one episode, but the pattern usually involves repeated episodes or lasting symptoms.

Dysthymia
Long-term unchanging symptoms of mild depression, sometimes lasting 20 to 30 years if untreated. Daily functioning not as severely affected, but over time impairment is cumulative.

Double Depression
Alternating periods of major depression and dysthymia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Bipolar

People who have a bipolar disorder live on an unending emotional roller coaster.

Types of Bipolar Disorders
- **Bipolar I:** major depression and full mania
- **Bipolar II:** major depression and mild mania
- **Cyclothymia:** mild depression with mild mania, chronic and long term

During the **Depressive Phase**, the person may:
- lose all interest in pleasurable activities and friends
- feel worthless, helpless, and hopeless
- have trouble concentrating
- lose or gain weight without trying
- have trouble sleeping or sleep more than usual
- feel tired all the time
- feel physical aches and pains that have no medical cause
- think about death or attempt suicide

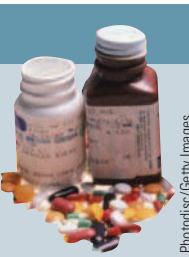
During the **Manic Phase**, the person may:
- feel extreme pleasure and joy from every activity
- be extraordinarily active, planning excessive daily activities
- sleep little without getting tired
- develop grandiose plans leading to reckless behavior: unrestrained buying sprees, sexual indiscretions, foolish business investments, etc.
- have "racing thoughts" and talk on and on
- be easily irritated and distracted

TREATMENT OF MOOD DISORDERS

Treatment for mood disorders is most effective and easiest when it's started early. Most people are treated with a combination of these methods.

Treatment

Medication
Antidepressants can help to control symptoms and restore neurotransmitter functioning.
Common types of antidepressants:
- Tricyclics (Tofranil, Elavil)
- Monamine oxidase inhibitors (MAOIs): (Nardil, Parnate); MAOIs can have severe side effects, especially when combined with certain foods or over-the-counter medications
- Selective-serotonin reuptake inhibitors or SSRIs (Prozac, Zoloft) are newer and cause fewer side effects than tricyclics or MAOIs
- Lithium is the preferred drug for bipolar disorder; side effects can be serious; and dosage must be carefully regulated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Cognitive-Behavioral Therapy
Helps depressed people:
- learn to replace negative depressive thoughts and attributions with more positive ones
- develop more effective coping behaviors and skills

Interpersonal Psychotherapy
Helps depressed people:
- focus on the social and interpersonal triggers for their depression (such as the loss of a loved one)
- develop skills to resolve interpersonal conflicts and build new relationships

Electroconvulsive Therapy (ECT)
- For severe depression, ECT is used when other treatments have been ineffective. It usually has temporary side effects, such as memory loss and lethargy. In some patients, certain intellectual and/or memory functions may be permanently lost.

Light Therapy
- For seasonal affective disorder


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CHAPTER 7

Physical Disorders and Health Psychology

Chapter Outline

Student Learning Outcomes*

Demonstrate knowledge and understanding representing appropriate breadth and depth in selected content areas of psychology:	› Biological bases of behavior and mental processes, including physiology, sensation, perception, comparative, motivation, and emotion (APA SLO 1.2.a (3)) ***(see textbook pages 251–257)***
Use the concepts, language, and major theories of the discipline to account for psychological phenomena:	› Identify antecedents and consequences of behavior and mental processes (APA SLO 1.3.b) ***(see textbook pages 257–270)***
Identify appropriate applications of psychology in solving problems, such as:	› Origin and treatment of abnormal behavior (APA SLO 4.2.b) ***(see textbook pages 270–278)***

*Portions of this chapter cover learning outcomes suggested by the American Psychological Association (2007) in their guidelines for the undergraduate psychology major. Chapter coverage of these outcomes is identified by APA Goal and APA Suggested Learning Outcome (SLO).

Psychological and Social Factors That Influence Health

- **What is the difference between behavioral medicine and health psychology?**
- **How are immune system function, stress, and physical disorders related?**

At the beginning of the 20th century the leading causes of death were from infectious diseases such as influenza, pneumonia, diphtheria, tuberculosis, typhoid fever, measles, and gastrointestinal infections. Since then, the percentage of yearly total deaths from these diseases has been reduced greatly, from 38.9% to 5.5% (Table 7.1). This reduction represents the first revolution in public health that eliminated many infectious diseases and mastered many more. But the enormous success of our health-care system in reducing mortality from disease has revealed a more complex and challenging problem: At present, some major contributing factors to illness and death in this country are *psychological and behavioral.*

In Chapter 2, we described the profound effects of psychological and social factors on brain structure and function. These factors seem to influence neurotransmitter activity, the secretion of neurohormones in the endocrine system, and, at a more fundamental level, gene expression. We have repeatedly looked at the complex interplay of biological, psychological, and social factors in the production and maintenance of psychological disorders. But psychological and social factors are important to a number of additional disorders, including endocrinological disorders such as diabetes, cardiovascular disorders, and disorders of the immune system such as acquired immune deficiency syndrome (AIDS). These and the other disorders discussed in this chapter are clearly *physical disorders.* They have known (or strongly inferred) physical causes and mostly observable physical pathology (for example, genital herpes, damaged heart muscle, malignant tumors, or measurable hypertension). Contrast this with the somatoform disorders discussed in Chapter 5: In conversion disorders, for example, clients complain of physical damage or disease but show no physical pathology. In the fourth edition, text revision, of the *Diagnostic and Statistical Manual (DSM-IV-TR),* physical disorders such as hypertension and diabetes are coded separately on Axis III. However, there is a provision for recognizing "psychological factors affecting medical condition."

The study of how psychological and social factors affect physical disorders used to be distinct and somewhat separate from the remainder of psychopathology. Early on, the field was called *psychosomatic medicine* (Alexander, 1950), which meant that *psychological* factors affected *somatic* (physical) function. The label *psychophysiological disorders* was used to communicate a similar idea. Such terms are less often used today because they are misleading. Describing as psychosomatic a disorder with an obvious physical component gave the impression that psychological (mental) disorders of mood and anxiety did not have a strong biological component. As we now know, this assumption is not viable. Biological, psychological, and social factors are implicated in the cause and maintenance of every disorder, both mental and physical.

The contribution of psychosocial factors to the etiology and treatment of physical disorders is widely studied.

Table 7.1 The 10 Leading Causes of Death in the United States in 1900 and in 2007 (Percentage of Total Deaths)

1900	Percentage	2007	Percentage
1. Pneumonia and influenza	11.8	**1.** Coronary heart disease	25.4
2. Tuberculosis	11.3	**2.** Malignant neoplasms (cancer)	23.2
3. Diarrhea, enteritis, and ulceration of the intestines	8.3	**3.** Stroke and other cerebrovascular diseases	5.6
4. Diseases of the heart	8.0	**4.** Chronic lower respiratory diseases	5.3
5. Intracranial lesions of vascular origin	6.2	**5.** Accidents (unintentional injury)	5.1
6. Nephritis (kidney disease)	5.2	**6.** Alzheimer's disease	3.1
7. Accidents (unintentional injury)	4.2	**7.** Diabetes mellitus	2.9
8. Cancer and other malignant tumors	3.7	**8.** Influenza and pneumonia	2.2
9. Senility	2.9	**9.** Nephritis (kidney disease)	1.9
10. Diphtheria	2.3	**10.** Septicemia	1.4
Other*	36.1	Other*	23.9

*Includes hemophilia, blood transfusion, perinatal exposure, transmission within health-care settings, and risk not reported or identified.

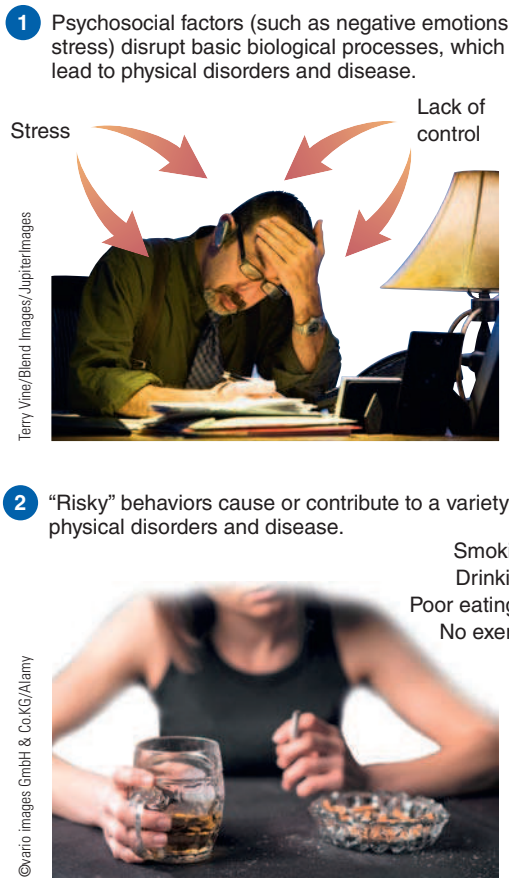
Source: Figures for the world adapted from UNAIDS (2009, November), *AIDS epidemic update.* Figures for the U.S. adapted from Centers for Disease Control and Prevention (2010), *Diagnosis of HIV infection and AIDS in the U.S. and dependent areas, 2008* (HIV Surveillance Report, Volume 20).

Some of the discoveries are among the more exciting findings in all of psychology and biology. For example, we described briefly in Chapter 2 that lowering stress levels and having a rich social network of family and friends is associated with better heath, living longer, and less cognitive decline as one ages (Cohen & Janicki-Deverts, 2009). Remember, too, the tragic physical and mental deterioration among elderly people who are removed from social networks of family and friends (Hawkley & Capioppo, 2007).

Health and Health-Related Behavior

The shift in focus from infectious disease to psychological factors has been called the second revolution in public health. Two closely related new fields of study have developed. In the first, **behavioral medicine** (Agras, 1982; Meyers, 1991), knowledge derived from behavioral science is applied to the prevention, diagnosis, and treatment of medical problems. This is an interdisciplinary field in which psychologists, physicians, and other health professionals work closely together to develop new treatments and preventive strategies (Schwartz & Weiss, 1978). A second field, **health psychology**, is not interdisciplinary, and it is usually considered a subfield of behavioral medicine. Practitioners study psychological factors that are important to the promotion and maintenance of health; they also analyze and recommend improvements in health-care systems and health policy formation within the discipline of psychology (Feuerstein, Labbe, & Kuczmierczyk, 1986; Stone, 1987; Taylor, 2009).

Psychological and social factors influence health and physical problems in *two* distinct ways (■ Figure 7.1). First, they can affect the basic biological processes that lead to illness and disease. Second, long-standing behavior patterns may put people at risk to develop certain physical disorders. Sometimes both these avenues contribute to the etiology or maintenance of disease (Kiecolt-Glaser & Newton, 2001; Miller & Blackwell, 2006; Schneiderman, 2004; Taylor, Repetti, & Seeman, 1997; Williams, Barefoot, & Schneiderman, 2003). Consider the example of *genital herpes*. There's a chance that someone you know has genital herpes and hasn't told you about it. It's not difficult to understand why: Genital herpes is an incurable sexually transmitted disease. Estimates indicate that more than 50 million Americans—about 20% of the entire population—have been infected by the herpes simplex virus affecting either oral or genital areas (Brentjens, Yeung-Yue, Lee, & Tyring, 2003). Because the disease is concentrated in young adults, the percentage in that group is much higher. The virus remains dormant until it is reactivated periodically. When it recurs in the genital region, infected individuals usually experience any of a number of symptoms, including pain, itching, vaginal or urethral discharge, and, most commonly, ulcerative lesions (open sores) in the genital area. Lesions recur approximately four times each year but can appear more often. Cases of genital herpes have increased dramatically in recent years, for reasons that are as much psychological and behavioral as biological. Although genital herpes is a biological disease, it spreads rapidly because people choose not to change their behavior by simply using a condom.



■ **Figure 7.1** Psychosocial factors directly affect physical health in two ways.

Stress also plays a role in triggering herpes recurrences (Chida & Mao, 2009; Goldmeier, Garvey, & Barton, 2008; Pereira et al., 2003). Stress-control procedures, particularly relaxation, seems to decrease recurrences of genital herpes, and the duration of each episode, most likely through the positive effects of such practices on the immune system (Burnette, Koehn, Kenyon-Jump, Huttun, & Stark, 1991; Pereira et al., 2003).

Consider also the tragic example of AIDS. AIDS is a disease of the immune system that is directly affected by stress (Cohen & Herbert, 1996; Kennedy, 2000), so stress may promote the deadly progression of AIDS. This is an example of how psychological factors may directly influence biological processes. We also know that a variety of things we may choose to do put us at risk for AIDS—for example, having unprotected sex or sharing dirty needles. Because there is no medical cure for AIDS yet, our best weapon is large-scale behavior modification to *prevent acquisition* of the disease.

Other behavioral patterns contribute to disease. Fully 50% of deaths from the 10 leading causes of death in the United States can be traced to behaviors common to certain lifestyles (Centers for Disease Control and Prevention [CDC], 2003b; Taylor, 2009). Smoking is the leading preventable cause of death in the United States and has been estimated to cause 20% of all deaths (CDC, 2007). Other unhealthy behaviors include poor eating habits, lack of exercise, and insufficient injury control (not wearing seat belts, for example). These behaviors are grouped under the label *lifestyle* because they are mostly enduring habits that are an integral part of a person's daily living pattern (Lewis, Statt, & Marcus, 2011; Oyama & Andrasik, 1992). Available evidence suggests that the same kinds of causal factors active in psychological disorders—social, psychological, and biological—play a role in some physical disorders (Mostofsky & Barlow, 2000; Uchino, 2009). But the factor attracting the most attention is *stress,* particularly the neurobiological components of the stress response.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▲ Hans Selye suggested in 1936 that stress contributes to certain physical problems.

The Nature of Stress

In 1936, a young scientist in Montreal, Canada, named Hans Selye noticed that one group of rats he injected with a certain chemical extract developed ulcers and other physiological problems, including atrophy of immune system tissues. But a control group of rats who received a daily saline (salty water) injection that should not have had any effect developed the *same* physical problems. Selye pursued this unexpected finding and discovered that the daily injections themselves seemed to be the culprit rather than the injected substance. Furthermore, many types of environmental changes produced the same results. Borrowing a term from engineering, he decided the cause of this nonspecific reaction was *stress.* As so often happens in science, an accidental or serendipitous observation led to a new area of study, in this case, *stress physiology* (Selye, 1936).

Selye theorized that the body goes through several stages in response to *sustained stress.* The first phase is a type of *alarm* response to immediate danger or threat. With continuing stress, we seem to pass into a stage of *resistance,* in which we mobilize various coping mechanisms to respond to the stress. Finally, if the stress is too intense or lasts too long, we may enter a stage of *exhaustion,* in which

behavioral medicine Interdisciplinary approach applying behavioral science to the prevention, diagnosis, and treatment of medical problems. Also known as *psychosomatic medicine.*
health psychology Subfield of behavioral medicine that studies psychological factors important in health promotion and maintenance.

our bodies suffer permanent damage or death (Selye, 1936, 1950). Selye called this sequence the **general adaptation syndrome (GAS)**.

The word *stress* means many things in modern life. In engineering, stress is the strain on a bridge when a heavy truck drives across it; stress is the *response* of the bridge to the truck's weight. But stress is also a *stimulus*. The truck is a "stressor" for the bridge, just as being fired from a job or facing a difficult final exam is a stimulus or stressor for a person. These varied meanings can create some confusion, but we concentrate on **stress** as the physiological response of the individual to a stressor.

The Physiology of Stress

In Chapter 2, we described the physiological effects of the early stages of stress, noting in particular its activating effect on the sympathetic nervous system, which mobilizes our resources during times of threat or danger by activating internal organs to prepare the body for immediate action, either fight or flight. These changes increase our strength and mental activity. We also noted in Chapter 2 that the activity of the endocrine system increases when we are stressed, primarily through activation of the hypothalamic–pituitary–adrenocortical (HPA) axis (see p. 46 in Chapter 2). Although a variety of neurotransmitters begin flowing in the nervous system, much attention has focused on the endocrine system's neuromodulators or neuropeptides, hormones affecting the nervous system that are secreted by the glands directly into the bloodstream (Chaouloff & Groc, 2010; Owens, Mulchahey, Stout, & Plotsky 1997; Taylor, Maloney, Dearborn & Weiss, 2009). These neuromodulating hormones act much like neurotransmitters in carrying the brain's messages to various parts of the body. One of the neurohormones, *corticotropin-releasing factor (CRF)*, is secreted by the hypothalamus and stimulates the pituitary gland. Farther down the chain of the HPA axis, the pituitary gland (along with the autonomic nervous system) activates the adrenal gland, which secretes, among other things, the hormone *cortisol*. Because of their close relationship to the stress response, cortisol and other related hormones are known as the *stress hormones*.

Remember that the HPA axis is closely related to the limbic system. The hypothalamus, at the top of the brain stem, is right next to the limbic system, which contains the hippocampus and seems to control our emotional memories. The hippocampus is responsive to cortisol. When stimulated by this hormone during HPA axis activity, the hippocampus helps to *turn off* the stress response, completing a feedback loop between the limbic system and the various parts of the HPA axis.

This loop may be important for a number of reasons. Working with primates, Robert Sapolsky and his colleagues (see, for example, Sapolsky & Meaney, 1986; Sapolsky, 2000a, 2000b, 2007) showed that increased levels of cortisol in response to chronic stress may kill nerve cells in the hippocampus. If hippocampal activity is thus compromised, excessive cortisol is secreted and, over time, the ability to turn off the stress response decreases, which leads to further aging of the hippocampus. These findings indicate that chronic stress leading to chronic secretion of cortisol may have long-lasting effects on physical function, including brain damage. Cell death may, in turn, lead to deficient problem-solving abilities among the aged and, ultimately, dementia. This physiological process may also affect susceptibility to infectious disease and recovery from it in other pathophysiological systems. Sapolsky's work is important because we now know that hippocampal cell death associated with chronic stress and anxiety occurs in humans with, for example, posttraumatic stress disorder (see Chapter 4) and depression (see Chapter 6). The long-term effects of this cell death are not yet known.

Contributions to the Stress Response

Stress physiology is profoundly influenced by psychological and social factors (Kemeny, 2003; Taylor et al., 2009). This

▲ Baboons at the top of the social hierarchy have a sense of predictability and control that allows them to cope with problems and maintain physical health; baboons at the bottom of the hierarchy suffer the symptoms of stress because they have little control over access to food, resting places, and mates.

link has been demonstrated by Sapolsky (1990, 2000a, 2000b, 2007). He studied baboons living freely in a national reserve in Kenya because their primary sources of stress, like those of humans, are psychological rather than physical. As with many species, baboons arrange themselves in a social hierarchy with dominant members at the top and submissive members at the bottom. And life is tough at the bottom! The lives of subordinate animals are made difficult (Sapolsky calls it "stressful") by continual bullying from the dominant animals, and they have less access to food, preferred resting places, and sexual partners. Particularly interesting are Sapolsky's findings on levels of cortisol in the baboons as a function of their social rank in a dominance hierarchy.

Remember from our description of the HPA axis that the secretion of cortisol from the adrenal glands is the final step in a cascade of hormone secretion that originates in the limbic system in the brain during periods of stress. The secretion of cortisol contributes to our arousal and mobilization in the short run but, if produced chronically, it can damage the hippocampus. In addition, muscles atrophy, fertility is affected by declining testosterone, hypertension develops in the cardiovascular system, and the immune response is impaired. Sapolsky discovered that dominant males in the baboon hierarchy ordinarily had *lower* resting levels of cortisol than subordinate males. When an emergency occurred, however, cortisol levels rose more quickly in the dominant males than in the subordinate males.

Sapolsky and his colleagues sought the causes of these differences by working backward up the HPA axis. They found an excess secretion of CRF by the hypothalamus in subordinate animals, combined with a diminished sensitivity of the pituitary gland (which is stimulated by CRF). Therefore, subordinate animals, unlike dominant animals, continually secrete cortisol, probably because their lives are so stressful. In addition, their HPA system is less sensitive to the effects of cortisol and therefore less efficient in turning off the stress response.

Sapolsky also discovered that subordinate males have fewer circulating lymphocytes (white blood cells) than dominant males, a sign of immune system suppression. In addition, subordinate males evidence less circulating high-density lipoprotein cholesterol, which puts them at higher risk for atherosclerosis and coronary heart disease, a subject we discuss later in this chapter.

What is it about being on top that produces positive effects? Sapolsky concluded that it is primarily the psychological benefits of having *predictability* and *controllability* concerning events in one's life. Parts of his data were gathered during years in which a number of male baboons were at the top of the hierarchy, with no clear "winner." Although these males dominated the rest of the animals in the group, they constantly attacked one another. Under these conditions, they displayed hormonal profiles more like those of subordinate males. Thus, dominance combined with stability produced optimal stress hormone profiles. But the most important factor in regulating stress physiology seems to be a sense of control (Sapolsky & Ray, 1989), a finding strongly confirmed in subsequent research (Kemeny, 2003; Sapolsky, 2007). Control of social situations and the ability to cope with any tension that arises go a long way toward blunting the long-term effects of stress.

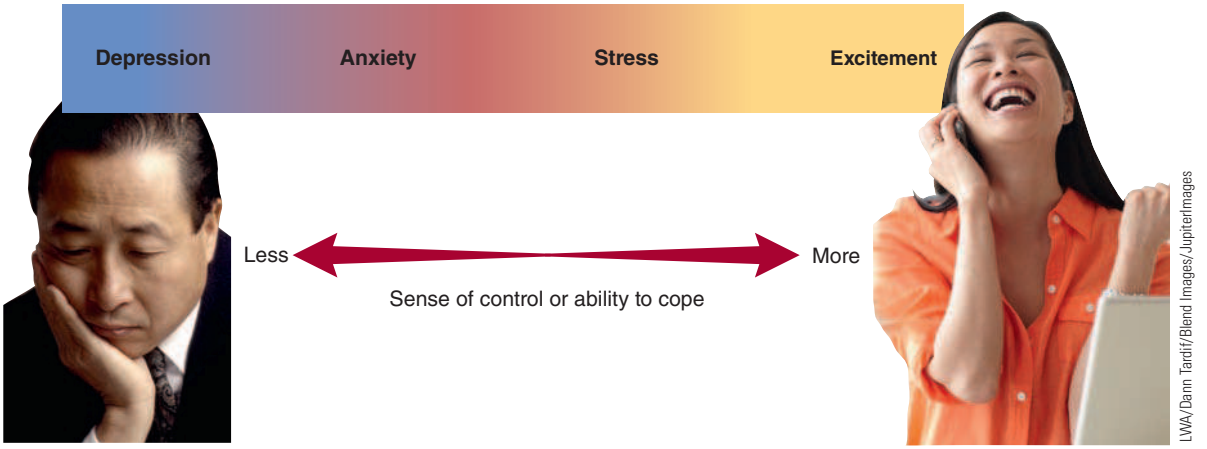
Stress, Anxiety, Depression, and Excitement

If you have read the chapters on anxiety, mood, and related psychological disorders, you might conclude, correctly, that stressful life events combined with psychological vulnerabilities such as an inadequate sense of control are a factor in both psychological and physical disorders. Is there any relationship between psychological and physical disorders? There seems to be a strong one. In a classic study, George Vaillant (1979) studied more than 200 Harvard University sophomore men between 1942 and 1944 who were mentally and physically healthy. He followed these men closely for more than 30 years. Those who developed psychological disorders or who were highly stressed became chronically ill or died at a significantly higher rate than men who remained well adjusted and free from psychological disorders, a finding that has been repeatedly confirmed (see, for example, Katon, 2003; Robles et al., 2005). This suggests that the same types of stress-related psychological factors that contribute to psychological disorders may contribute to the later development of physical disorders and that stress, anxiety, and depression are closely related. Can you tell the difference among feelings of stress, anxiety, depression, and excitement? You might say, "No problem," but these four states have a lot in common. Which one you experience may depend on your *sense of control* at the moment or how well you think you can cope with the threat or challenge you are facing (Barlow, 2002; Barlow, Chorpita, & Turovsky, 1996; Suárez, Bennett, Goldstein, & Barlow, 2009). This continuum of feelings from excitement to stress to anxiety to depression is shown in ■ Figure 7.2.

Consider how you feel when you are excited. You might experience a rapid heartbeat, a sudden burst of energy, or a jumpy stomach. But if you're well prepared for the challenge—for example, if you're an athlete, up for the game and confident in your abilities, or a musician, sure you are going to give an outstanding performance—these feelings of *excitement* can be pleasurable.

Sometimes when you face a challenging task, you feel you could handle it if you only had the time or help you need, but because you don't have these resources, you feel pressured. In response, you may work harder to do better and be perfect, even though you think you will be all right in the end. If you are under too much pressure, you may

general adaptation syndrome (GAS) Sequence of reactions to sustained stress described by Hans Selye. These stages are alarm, resistance, and exhaustion, which may lead to death.
stress Body's physiological response to a stressor, which is any event or change that requires adaptation.



■ **Figure 7.2** Responses to threats and challenges. Our feelings range along a continuum from depression to anxiety to stress to excitement, depending partly on our sense of control and ability to cope. (Adapted, with permission, from Barlow, D. H., Rapee, R. M., & Reisner, L. C., 2001. *Mastering stress 2001: A lifestyle approach.* Dallas, TX: American Health, © 2001 American Health Publishing.)

become tense and irritable or develop a headache or an upset stomach. This is what *stress* feels like. If something really is threatening and you believe there is little you can do about it, you may feel *anxiety.* The threatening situation could be anything from a physical attack to making a fool of yourself in front of someone. As your body prepares for the challenge, you worry about it incessantly. Your sense of control is considerably less than if you were stressed. In some cases, there may not be a difficult situation. Sometimes we are anxious for no reason except that we feel certain aspects of our lives are out of control. Finally, individuals who always perceive life as threatening may lose hope about ever having control and slip into a state of *depression,* no longer trying to cope.

To sum up, the underlying physiology of these particular emotional states seems relatively similar in some basic ways. This is why we refer to a similar pattern of sympathetic arousal and activation of specific neurotransmitters and neurohormones in discussing anxiety, depression, and stress-related physical disorders. However, there seem to be some differences. Blood pressure may increase when the challenges seem to overwhelm coping resources, resulting in a low sense of control (anxiety, depression), but blood pressure will be unchanged during excitement or marked stress (Blascovich & Tomaka, 1996). Nevertheless, it is psychological factors—specifically a sense of control and confidence that we can cope with stress or challenges, called **self-efficacy** by Bandura (1986)—that differ most markedly among these emotions, leading to different feelings (Taylor et al., 1997).

Stress and the Immune Response

Have you had a cold during the past several months? How did you pick it up? Did you spend the day with someone else who had a cold? Did someone sneeze nearby while you were sitting in class? Exposure to cold viruses is a necessary factor in developing a cold, but, as mentioned briefly in Chapter 2, the level of stress you are experiencing at the time seems to play a major role in whether the exposure results in a cold. Sheldon Cohen and his associates (Cohen, 1996; Cohen, Doyle, & Skoner, 1999; Cohen, Tyrrell, & Smith, 1991, 1993) exposed volunteer participants to a specific dosage of a cold virus and followed them closely. They found that the chance a participant would get sick was directly related to how much stress the person had experienced during the past year. Cohen and colleagues (1995) also linked the intensity of stress and negative affect at the time of exposure to the later *severity* of the cold, as measured by mucus production. In an interesting twist, Cohen, Doyle, Turner, Alper, and Skoner (2003) have demonstrated that how sociable you are—that is, the quantity and quality of your social relationships—affects whether you come down with a cold when exposed to the virus, perhaps because socializing with friends relieves stress (Cohen & Janicki-Devarts, 2009).

Almost certainly, the effect of stress on susceptibility to infections is mediated through the **immune system**, which protects the body from any foreign materials that may enter it. Humans under stress show clearly increased rates of infectious diseases, including colds, herpes, and mononucleosis (Cohen & Herbert, 1996; Taylor, 2009). Direct evidence links a number of stressful situations to lowered immune system functioning, including marital discord or relationship difficulties (Kiecolt-Glaser et al., 2005; Kiecolt-Glaser & Newton, 2001; Uchino, 2009), job loss, and the death of a loved one (Hawkley & Cacioppo, 2007; Morris, Cook, & Shaper, 1994; Pavalko, Elder, & Clipp, 1993).

We have already noted that psychological disorders seem to make us more susceptible to developing physical disorders (Katon, 2003; Robles et al., 2005; Vaillant, 1979). In fact, direct evidence indicates that depression lowers immune system functioning (Herbert & Cohen, 1993;

Miller & Blackwell, 2006; Stone, 2000; Weisse, 1992), particularly in older adults (Herbert & Cohen, 1993). It may be that the level of depression—and, more importantly, the underlying sense of uncontrollability that accompanies most depressions—is the crucial mechanism in lowering immune system functioning, a mechanism present during most negative stressful life events, such as job loss (Miller & Blackwell, 2006; Robles et al., 2005; Weisse, 1992). Depression can also lead to poor self-care and a tendency to engage in riskier behaviors. Like Sapolsky's baboons, for humans the ability to retain a sense of control over events in our lives may be one of the most important psychological contributions to good health.

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Studying the Effects of Emotions on Physical Health

People with the lowest level of sociability are most likely to get a cold, whereas people with the highest level of sociability are least likely to develop a cold.

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Most studies concerning stress and the immune system have examined a sudden or acute stressor. But *chronic stress* may be more problematic because the effects, by definition, last longer (Schneiderman, 2004). For example, lowered immune system functioning has been reported for people who care for chronically ill family members, such as Alzheimer's disease patients (Holland & Gallagher-Thompson, 2011; Mills et al., 2004).

To understand how the immune system protects us, we must first understand how it works. We take a brief tour of the immune system next, using ■ Figure 7.3 as a visual guide, and then we examine psychological contributions to the biology of two diseases strongly related to immune system functioning: AIDS and cancer.

How the Immune System Works

The immune system identifies and eliminates foreign materials, called **antigens**, in the body. Antigens can be any of a number of substances, usually bacteria, viruses, or parasites. But the immune system also targets the body's own cells that have become aberrant or damaged in some way, perhaps as part of a malignant tumor. Donated organs are foreign, so the immune system attacks them after surgical transplant; consequently, it is necessary to suppress the immune system temporarily after surgery.

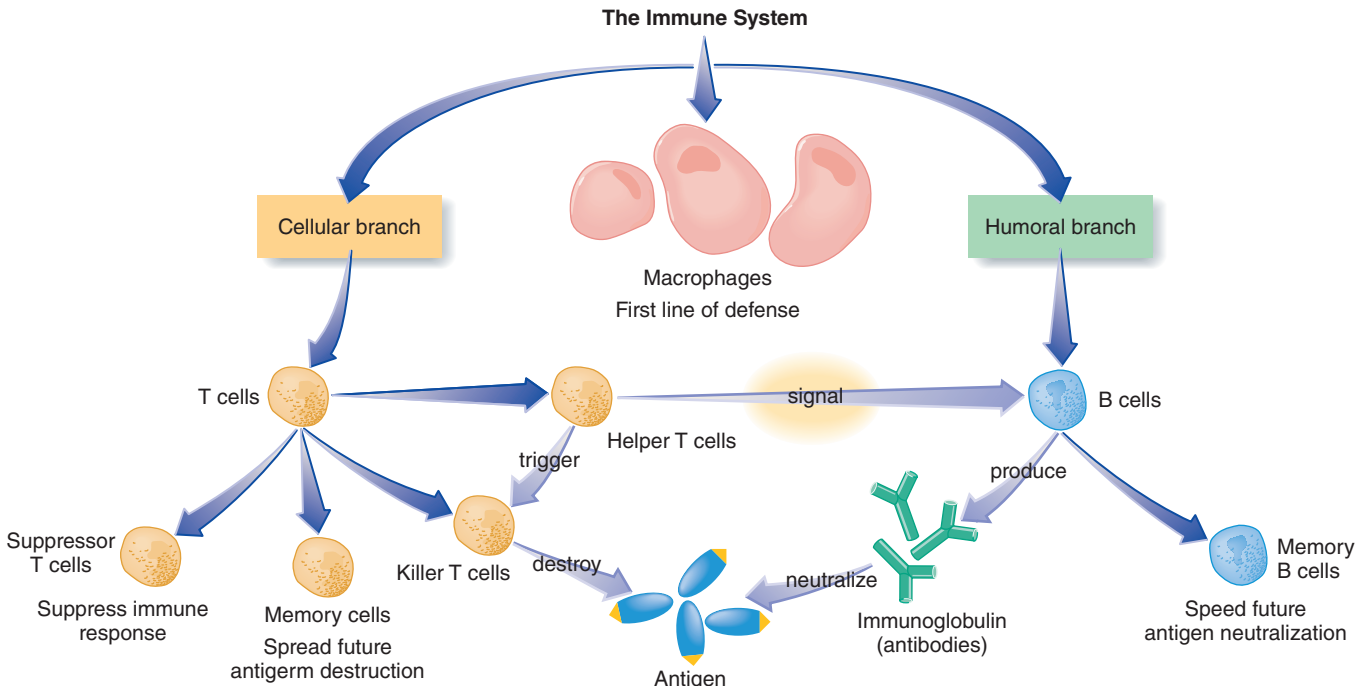
The immune system has two main parts: the humoral and the cellular. Specific types of cells function as agents of both. White blood cells, called *leukocytes,* do most of the work. There are several types of leukocytes. *Macrophages* might be considered one of the body's first lines of defense: They surround identifiable antigens and destroy them. They also signal *lymphocytes,* which consist of two groups, B cells and T cells.

The *B cells* operate within the humoral part of the immune system, releasing molecules that seek antigens in blood and other bodily fluids with the purpose of neutralizing them. The B cells produce highly specific molecules called *immunoglobulins* that act as *antibodies,* which combine with the antigens to neutralize them. After the antigens are neutralized, a subgroup called *memory B cells* are created so that the next time that antigen is encountered, the immune system response will be even faster. This action accounts for the success of inoculations, or vaccinations, you may have received for mumps or measles as a child. An inoculation contains small amounts of the targeted organism but not enough to make you sick. Your immune system then "remembers" this antigen and prevents you from coming down with the full disease when you are exposed to it.

Members of the second group of lymphocytes, called *T cells,* operate in the cellular branch of the immune system. These cells don't produce antibodies. Instead, one subgroup, *killer T cells,* directly destroys viral infections and cancerous processes (Dustin & Long, 2010; Wan, 2010). When the process is complete, *memory T cells* are created to speed future responses to the same antigen. Other subgroups of T cells help regulate the immune system. For example, *T4 cells* are called *helper T cells* because they enhance the immune system response by signaling B cells to produce antibodies and telling other T cells to destroy the antigen. *Suppressor T cells* suppress the production of antibodies by B cells when they are no longer needed.

We should have twice as many T4 (helper) cells as suppressor T cells. With too many T4 cells, the immune system is overreactive and may attack the body's normal cells rather than antigens. When this happens, we have what is called an **autoimmune disease**, such as **rheumatoid arthritis**. With too many suppressor T cells, the body is subject to invasion by a number of antigens. The human immunodeficiency virus (HIV) directly attacks the helper T cells, lymphocytes that are crucial to both humoral and cellular immunity, thereby severely weakening the immune system and causing AIDS.

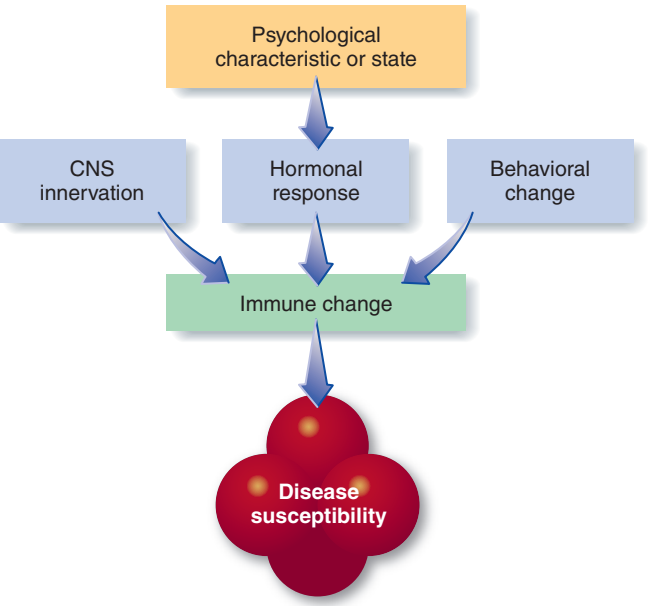
self-efficacy Perception of having the ability to cope with stress or challenges.
immune system Body's means of identifying and eliminating any foreign materials (for example, bacteria, parasites, and even transplanted organs) that enter.
antigens Foreign material that enters the body, including bacteria and parasites.
autoimmune disease Condition in which the body's immune system attacks healthy tissue rather than antigens.
rheumatoid arthritis Painful, degenerative disease in which the immune system essentially attacks itself, resulting in stiffness, swelling, and even destruction of the joints. Cognitive–behavioral treatments can help relieve pain and stiffness.



■ **FIGURE 7.3** An overview of the immune system. (© Cengage Learning 2013)

Until the mid-1970s most scientists believed the brain and the immune system operated independently of each other. However, in 1974, Robert Ader and his colleagues (see, for example, Ader & Cohen, 1975, 1993) made a startling discovery. Working with a classical conditioning paradigm, they gave sugar-flavored water to rats, together with a drug that suppresses the immune system. Ader and Cohen then demonstrated that giving the same rats only the sweet-tasting water produced similar changes in the immune system. In other words, the rats had "learned" (through classical conditioning) to respond to the water by suppressing their immune systems. We now know there are many connections between the nervous system and the immune system. These findings have generated a field of study known as **psychoneuroimmunology**, or **PNI** (Ader & Cohen, 1993), which simply means the object of study is *psycho*logical influences on the *neuro*logical responding implicated in our *immune* response.

Researchers have learned a great deal recently about pathways through which psychological and social factors may influence immune system functioning. Direct connections among the brain (central nervous system), the HPA axis (hormonal), and the immune system have already been described. Behavioral changes in response to stressful events, such as increased smoking or poor eating habits, may also suppress the immune system (Cohen & Herbert, 1996; ■ Figure 7.4). Now scientists have uncovered a chain of molecules that connects stress to the onset of disease by turning on certain genes (Cole et al., 2010). Basically, stress seems to activate certain molecules in cells that activate genes (called



■ **Figure 7.4** Pathways through which psychological factors might influence onset and progression of immune system–mediated disease. For simplicity, arrows are drawn in only one direction, from psychological characteristics to disease. No lack of alternative paths is implied. CNS = central nervous system. (From Cohen, S., & Herbert, T. B., 1996. Health psychology: Psychological factors and physical disease from the perspective of human psychoneuroimmunology. *Annual Review of Psychology, 47,* 113–142.)

a transcription factor), in this case the GABA-1 transcription factor that activates the interleukin-6 gene. This gene makes a protein that turns on the inflammatory response, which brings infection-fighting cells of the immune system to the area. This is great if you've cut yourself, but very damaging if it occurs over a long period. It is this chronic inflammatory response that exacerbates cancer, heart disease, and diabetes and shortens life. Of course, other genes, such as the serotonin transporter gene mentioned in Chapter 2, are also implicated in making one vulnerable to certain types of stressors (Way & Taylor, 2010). Undoubtedly many more groups of genes and integrative psychobiological paths implicated in the effects of the stress response will be discovered (Segerstrom & Sephton, 2010).

Concept Check 7.1

Assess your knowledge of the immune system by matching components of the immune system with their function in the body: (a) macrophages, (b) B cells, (c) immunoglobins, (d) killer T cells, (e) suppressor T cells, and (f) memory B cells.

1. This subgroup targets viral infections within the cells by directly destroying the antigens. _____
2. A type of leukocyte that surrounds identifiable antigens and destroys them. _____
3. Highly specific molecules that act as antibodies. They combine with antigens to neutralize them. _____
4. Lymphocytes that operate within the humoral part of the system and circulate in the blood and bodily fluids. _____
5. These are created so that when a specific antigen is encountered in the future, the immune response will be faster. _____
6. These T cells stop the production of antibodies by B cells when they are no longer needed. _____

Psychosocial Effects on Physical Disorders

- **How is stress related to AIDS, cancer, and cardiovascular disease?**
- **What are the potential causes of acute and chronic pain, and how do the two types of pain differ?**

With an enhanced understanding of the effects of emotional and behavioral factors on the immune system, we can now examine how these factors influence specific physical disorders. We begin with AIDS.

AIDS

The ravages of the AIDS epidemic have made this disease the highest priority of public health systems around the world. The number of people worldwide living with HIV continues to grow, reaching an estimated 33.4 million in 2008, which is 20% higher than in 2000 (UNAIDS, 2009). Only in 2004 did adult and child deaths begin to level off with aggressive treatment and prevention efforts in some parts of the world (Bongaarts & Over, 2010). Despite this modest success, 2.7 million new HIV infections occurred worldwide in 2008, and 2 million people died of AIDS (Grabbe & Bunnell, 2010; UNAIDS, 2009). An estimated 430,000 new infections occurred among children under the age of 15 in 2008, with most new infections stemming from transmission in utero, during delivery, or as a result of breast feeding (UNAIDS, 2009). In the hardest hit regions in southern Africa, between 20% and 40% of the adult population are believed to be HIV positive, comprising two thirds of cases worldwide, with approximately 18 million children orphaned by the disease (Klimas, Koneru, & Fletcher, 2008). AIDS is also spreading rapidly to the densely populated regions of India and China (Normile, 2009), and in Latin America, rates are projected to increase from 2 million in 2006 to 3.5 million by 2015 (Cohen, 2006).

Once a person is infected with HIV, the course of the disease is variable. After several months to several years with no symptoms, patients may develop minor health problems such as weight loss, fever, and night sweats, symptoms that make up the condition known as **AIDS-related complex (ARC)**. A diagnosis of AIDS itself is not made until one of several serious diseases appears, such as pneumocystis pneumonia, cancer, dementia, or a wasting syndrome in which the body literally withers away. The median time from initial infection to the development of full-blown AIDS has been estimated to range from 7.3 to

psychoneuroimmunology (PNI) Study of psychological influences on the neurological responding involved in the body's immune response.
AIDS-related complex (ARC) Group of minor health problems such as weight loss, fever, and night sweats that appears after HIV infection but before development of full-blown AIDS.

10 years or more (Pantaleo, Graziosi, & Fauci, 1993). Clinical scientists have developed powerful new combinations of drugs referred to as highly active antiretroviral therapy (HAART) that suppress the virus in those infected with HIV, even in advanced cases (Hammer et al., 2006; Thompson et al., 2010). This has been a very positive development that has slowed disease progression and decreased mortality. For example, most people with AIDS die within 1 year of diagnosis without treatment, as is the case in many developing countries (Zwahlen & Egger, 2006). But the proportion of people who receive treatment surviving with AIDS 2 years or longer increased to 85% by 2005, and the death rate from AIDS declined 80% since 1990 (Knoll, Lassmann & Temesgen, 2007).

Nevertheless, HAART does not seem to be a cure because the most recent evidence suggests the virus is seldom eliminated but rather lies dormant in reduced numbers; thus, infected patients face a lifetime of taking multiple medications (Buscher & Giordano, 2010; Cohen, 2002; Hammer et al., 2006). Also, the percentage who stop using HAART because of severe side effects, such as nausea and diarrhea, is high—61% in one study (O'Brien, Clark, Besch, Myers, & Kissinger, 2003). For this reason, earlier recommendations were to postpone treatment until those infected are in imminent danger of developing symptomatic disease (Cohen, 2002; Hammer et al., 2006), but in view of the success of this treatment regimen with cases of newly acquired HIV, current recommendations are to start as early as possible after detecting infection and to work closely with patients to increase adherence to the schedule for the medication (Thompson et al., 2010). Unfortunately, drug-resistant strains of HIV are now being transmitted.

Because AIDS is a relatively new disease and takes at least several years to develop, we are still learning about the factors, including possible psychological factors, that extend survival (Klimas et al., 2008; Taylor, 2009). Investigators identified a group of people who have been exposed repeatedly to the AIDS virus but have not contracted the disease. A major distinction of these people is that their immune systems, particularly the cellular branch, are robust and strong (Ezzel, 1993), most likely because of genetic factors (Kaiser, 2006). Therefore, efforts to boost the immune system may contribute to the prevention of AIDS.

Because psychological factors affect immune system functioning, investigators have begun to examine whether these psychological factors influence the progression of HIV. For example, high levels of stress and depression and low levels of social support have been associated with a faster progression to disease (Leserman, 2008; Leserman et al., 2000). But an even more intriguing question is whether psychological interventions can slow the progression of the disease, even among those who are symptomatic (Cole, 2008; Gore-Felton & Koopman, 2008). In fact, several important studies suggest that cognitive-behavioral stress-management (CBSM) programs may have positive effects on the immune systems of individuals who are already symptomatic (Antoni et al., 2000; Carrico & Antoni, 2008; Lutgendorf et al., 1997). Specifically, Lutgendorf and colleagues (1997) used an intervention program that significantly decreased depression and anxiety compared to a control group that did not receive the treatment. More important, there was a significant reduction in antibodies to the herpes simplex virus II in the treatment group compared to the control group, which reflects the greater ability of the cellular component of the immune system to control the virus.

In a study by Antoni and colleagues (2000), 73 gay or bisexual men already infected with HIV and symptomatic with the disease were assigned to a CBSM program or a control group receiving usual care without the program. As in previous studies, men receiving the stress-management treatment showed significantly lower posttreatment levels of anxiety, anger, and perceived stress than those in the control group, indicating the treatment was effective. More important, as long as a year after the intervention had ended, men who had received the treatment evidenced better immune system functioning as indicated by higher levels of T cells.

Similarly, Goodkin and colleagues (2001) reported that a 10-week psychological treatment significantly buffered against an increase in HIV viral load, which is a powerful and reliable predictor of progression to full-blown AIDS, when compared to a control group. Antoni and colleagues (2006) took their important line of research a step further. HIV-positive men on HAART drug regimens received 10 weeks of training in how to take their medication properly by taking the exact amount prescribed as closely as possible to the assigned times. Half of this group also received the investigators' CBSM program. Men receiving CBSM actually showed a decrease in viral load 15 months later compared to those with medication training only, who showed no change. This reduction in viral load was primarily the result of decreases in depression, which, in turn, reduced the stress hormone cortisol. Thus, even in progressed, symptomatic HIV disease, psychological interventions may not only enhance psychological adjustment, but also influence immune system functioning, and this effect may be long lasting.

It is too early to tell whether these results will be strong or persistent enough to translate into increased survival time for AIDS patients, although results from Antoni and colleagues (2000, 2006) suggest they might. If stress and related variables *are* clinically significant to immune response, functioning, and disease progression in HIV-infected patients, as suggested by a number of studies (Cole, 2008; Leserman, 2008), then psychosocial interventions to bolster the immune system might increase survival rates and, in the most optimistic scenario, prevent the slow deterioration of the immune system (Carrico & Antoni, 2008; Kennedy, 2000). Of course, the most effective interventions focus on changing behavior to prevent acquiring HIV in the first place, such as reducing risky behavior and promoting safe sexual practices (Temoshok, Wald, Synowki, & Garzino-Demo, 2008).

Cancer

Among the more mind-boggling developments in the study of illness and disease is the discovery that the development and course of different varieties of **cancer** are subject to psychosocial influences (Emery, Anderson, & Andersen, 2011;

Williams & Schneiderman, 2002). This has resulted in a new field of study called **psycho-oncology** (Andersen, 1992; Antoni & Lutgendorf, 2007; Helgeson, 2005; Lutgendorf, Costanzo, & Siegel, 2007). *Oncology* means the study of cancer. In a widely noted study, David Spiegel, a psychiatrist at Stanford University, and his colleagues (Spiegel, Bloom, Kramer, & Gotheil, 1989) studied 86 women with advanced breast cancer that had metastasized to other areas of their bodies and was expected to kill them within 2 years. Clearly, the prognosis was poor indeed. Although Spiegel and his colleagues had little hope of affecting the disease itself, they thought that by treating these people in group psychotherapy at least they could relieve some of their anxiety, depression, and pain.

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Breast Cancer Support and Education

Women who had low self-esteem, low body image, feelings of low control, low optimism, and a lack of support at home were even more likely to benefit from an education intervention.

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All patients had routine medical care for their cancer. In addition, 50 patients (of the 86) met with their therapist for psychotherapy once a week in small groups. Much to everyone's surprise, including Spiegel's, the therapy group's survival time was significantly longer than that of the control group who did not receive psychotherapy but otherwise benefited from the best care available. The group receiving therapy lived twice as long on average (approximately 3 years) as the controls (approximately 18 months). Four years after the study began, one third of the therapy patients were still alive and all the patients receiving the best medical care available *without* therapy had died. Subsequently, a careful reanalysis of medical treatment received by each group revealed no differences that could account for the effects of psychological treatment (Kogon, Biswas, Pearl, Carlson, & Spiegel, 1997). These findings do not mean that psychological interventions cured advanced cancer. At 10 years, only three patients in the therapy group still survived.

Subsequent studies seemed to support these findings on increased survival and reduced recurrence with different types of cancer (Fawzy, Cousins, et al., 1990; Fawzy, Kemeny, et al., 1990). But other studies did not replicate the finding that psychological treatments prolong life (Coyne, Stefanek, & Palmer, 2007). One such study confirmed that psychological treatments reduced depression and pain and increased well-being, but did not find the survival-enhancing effects of treatment (Goodwin et al., 2001).

However, recently in an important study, Andersen et al. (2008) randomly assigned 227 patients who had been surgically treated for breast cancer to a psychological intervention plus assessment, or to an assessment-only condition. The intervention included strategies to reduce stress, improve mood, alter important health behaviors (reducing smoking, increasing exercise, etc.), and maintain adherence to cancer treatment and care. The treatment was successful in reducing stress and increasing positive mood and healthy behavior (Andersen et al., 2007). More important, after a median of 11 years follow-up, patients receiving the psychological intervention reduced their risk of dying by breast cancer by 56% and their risk of breast cancer recurrence by 45%, supporting once again the survival-enhancing potential of psychological treatments.

As a result of these studies, psychosocial treatment for various cancers to reduce stress, improve quality of life, and perhaps even to increase survival and reduce recurrence are now more readily available (Manne & Ostroff, 2008; Penedo, Antoni, & Schneiderman, 2008). The initial success of these psychological treatments on length of survival in at least some studies generated a great deal of interest in exactly

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▲ Psychological preparation reduces suffering and facilitates recovery in children who undergo surgery.

cancer Category of often-fatal medical conditions involving abnormal cell growth and malignancy.
psycho-oncology Study of psychological factors involved in the course and treatment of cancer.

how they might work, if they do work (Antoni et al., 2009; Antoni & Lutgendorf, 2007; Emery et al., 2011; Helgeson, 2005). Possibilities include better health habits, closer adherence to medical treatment, and improved endocrine functioning and response to stress, all of which may improve immune function (Antoni et al., 2006, 2009; Classen, Diamond, & Spiegel, 1998; Emery et al., 2011; Foley, Baillie, Huxter, Price, & Sinclair, 2010; Nezu et al., 1999).

Anything that promotes closer, more supportive relationships in cancer patients also is very important because it slows disease progression (Antoni et al., 2006; Foley, Baillie, Huxter, Price, & Sinclair, 2010; Nezu, et al., 1999; Weighs, Enright, & Simmens, 2008). There is even preliminary evidence that psychological factors may contribute not only to the *course* but also to the *development* of cancer and other diseases (Antoni & Lutgendorf, 2007; Lutgendorf et al., 2007). Perceived lack of control, inadequate coping responses, overwhelmingly stressful life events, or the use of inappropriate coping responses (such as denial) may all contribute to the development of cancer, probably through changes in immune function, but also through regulating the activity of cancer-causing viruses, deoxyribonucleic acid (DNA) repair processes, and the expression of genes that control the growth of tumors (Antoni & Lutgendorf, 2007; Lutgendorf et al., 2007; Williams & Schneiderman, 2002).

These studies have also led to a renewed emphasis on an overlooked result of cancer—that is, some people discover some positive consequences. For example, many patients with breast cancer experience an enhanced sense of purpose, deepening spirituality, closer ties to others, and changes in life priorities (Lechner & Antoni, 2004; Park, Edmondson, Fenster, & Blank, 2008; Yanez et al., 2009). These experiences have been called "benefit finding" and may reflect the types of traits, such as coping skills, a sense of control, and optimism, that underlie resiliency and reduce the harmful effect of stress (Bower, Moskowitz, & Epel, 2009). It is these traits and skills that are among the most important goals of psychological treatment. Antoni and colleagues (2006) targeted these goals in 199 women with nonmetastatic breast cancer using a CBSM program, and they found substantially improved quality of life in the year following treatment.

Psychological factors are also prominent in treatment and recovery from cancer in children (Koocher, 1996). Many types of cancer require invasive and painful medical procedures; the suffering can be difficult to bear, not only for children, but also for parents and health-care providers. Children usually struggle and cry hysterically, so to complete many of the procedures, they must be physically restrained. Not only does their behavior interfere with successful completion, but also the stress and anxiety associated with repeated painful procedures may have their own detrimental effect on the disease process. Psychological procedures designed to reduce pain and stress in these children include breathing exercises, watching films of exactly what happens to take the uncertainty out of the procedure, and rehearsal of the procedure with dolls, all of which make the interventions more tolerable and therefore more successful for young patients (Brewer, Gleditsch, Syblik, Tietjens, & Vacik, 2006; Hubert, Jay, Saltoun, & Hayes, 1988; Jay, Elliott, Ozolins, Olson, & Pruitt, 1985). Much of this work is based on the pioneering efforts of Barbara Melamed and her colleagues, who demonstrated the importance of incorporating psychological procedures into children's medical care, particularly children about to undergo surgery (see, for example, Melamed & Siegel, 1975). In any case, pediatric psychologists are making more routine use of these procedures.

Reducing stress in parents who could then provide more supportive care is important because almost all parents develop posttraumatic stress symptoms after hearing that their children have cancer (Kazak, Boeving, Alderfer, Hwang, & Reilly, 2005). Sahler and colleagues (2005) treated mothers of children with newly diagnosed cancer with a cognitive-behavioral problem-solving intervention and compared the results to the usual care available to these mothers. Mothers in the problem-solving group became less negative, less stressed, and better problem solvers, certainly a positive outcome in parents who have to deal with the tragedy of cancer in their own children.

Cardiovascular Problems

The *cardiovascular system* comprises the heart, blood vessels, and complex control mechanisms for regulating their function. Many things can go wrong with this system and lead to **cardiovascular disease**. For example, many individuals, particularly older individuals, suffer **strokes**, also called **cerebral vascular accidents (CVAs)**, which are temporary blockages of blood vessels leading to the brain or a rupture of blood vessels in the brain that results in temporary or permanent brain damage and loss of functioning. People with Raynaud's disease lose circulation to peripheral parts of their bodies such as their fingers and toes, suffering some pain and continual sensations of cold in their hands and feet. The cardiovascular problems receiving the most attention these days are hypertension and coronary heart disease, and we look at both. First, let's consider the case of John.

John ♦ The Human Volcano

John is a 55-year-old business executive who is married with two teenage children. For most of his adult life, John has smoked about a pack of cigarettes each day. Although he maintains a busy and active schedule, John is mildly obese, partly from regular meals with business partners and colleagues. He has been taking several medications for high blood pressure since age 42. John's doctor has warned him repeatedly to cut down on his smoking and to exercise more often, especially because John's father died of a heart attack. Although John has episodes of chest pain, he continues his busy and stressful lifestyle. It is difficult for John to slow down because his business has been doing extremely well during the past 10 years.

Moreover, John believes that life is too short so there is no time to slow down. He sees relatively little of his family and works late most evenings. Even when he's at home John typically works into the night. It is difficult for him to relax; he feels a constant urgency to get as many things done as possible and prefers to work on several tasks simultaneously. For instance, John often proofreads a document, engages in a phone conversation, and eats lunch all at the same time. He attributes much of the success of his business to his working style. Despite his success, John is not well liked by his peers. His coworkers and employees often find him to be overbearing, easily frustrated, and, at times, even hostile. His subordinates in particular claim he is overly impatient and critical of their performance.

Most people would recognize that John's behaviors and attitudes make his life unpleasant and possibly lethal. Some of these behaviors and attitudes appear to operate directly on the cardiovascular system and may result in hypertension and coronary heart disease.

Hypertension

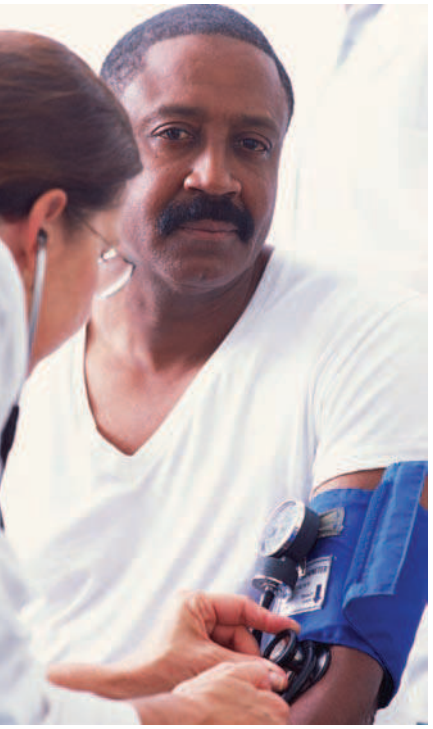
Hypertension (high blood pressure) is a major risk factor not only for stroke and heart disease, but also for kidney disease. This makes hypertension an extremely serious medical condition. Blood pressure increases when the blood vessels leading to organs and peripheral areas constrict (become narrower), forcing increasing amounts of blood to muscles in central parts of the body. Because so many blood vessels have constricted, the heart muscles must work much harder to force the blood to all parts of the body, which causes the increased pressure. These factors produce wear and tear on the ever-shrinking blood vessels and lead to cardiovascular disease. A small percentage of cases of hypertension can be traced to specific physical abnormalities, such as kidney disease or tumors on the adrenal glands (Chobanian et al., 2003; Papillo & Shapiro, 1990), but the overwhelming majority (close to 90%) have no specific verifiable physical cause and are considered **essential hypertension**. Blood pressure is defined as high by the World Health Organization if it exceeds 160 over 95 (Papillo & Shapiro, 1990), although measures of 140/90 or higher are cause for concern and more usually used to define hypertension (Chobanian et al., 2003; Taylor, 2009; Wolf-Maier et al., 2003). The first value is called the *systolic blood pressure,* the pressure when the heart is pumping blood. The second value is the *diastolic blood pressure,* the pressure between beats when the heart is at rest. Elevations in diastolic pressure seem to be more worrisome in terms of risk of disease.

According to a comprehensive survey, 27.6% of individuals between the ages of 35 and 64 suffer from hypertension in North America, with a corresponding and shocking figure of 44.2% in six European countries (Wolf-Maier et al., 2003). A more recent survey puts the hypertension prevalence at 29% of everyone older than age 18 in the United States (Egan, Zhao, & Axon, 2010). These are extraordinary numbers when you consider that hypertension, contributing to as many fatal diseases as it does, including stroke, has been called the "silent killer" because there are few, if any, symptoms and most people don't know they have it. These numbers are much higher than for any single psychological disorder. Even more striking is that African Americans, both men and women, are nearly *twice* as likely to develop hypertension as whites (Brannon & Feist, 1997; Egan et al., 2010; Lewis et al., 2006; Yan et al., 2003). More important, African Americans have hypertensive vascular diseases at a rate 5 to 10 times greater than whites. This makes hypertension a principal disorder of concern among the African American population. Saab and colleagues (1992) demonstrated that during laboratory stress tests African Americans without high blood pressure show greater vascular responsiveness, including heightened blood pressure. Thus, African Americans in general may be at greater risk to develop hypertension.

You will not be surprised to learn there are biological, psychological, and social contributions to the development of this potentially deadly condition. It has long been clear that hypertension runs in families and likely is subject to marked genetic influences (Papillo & Shapiro, 1990; Taylor, 2009; Williams et al., 2001). When stressed in the laboratory, even individuals with *normal* blood pressure show greater reactivity in their blood pressure if their parents have high blood pressure than do individuals with normal blood pressure whose parents also had normal blood pressure (Clark, 2003; Fredrikson & Matthews, 1990). In other words, it doesn't take much to activate an inherited vulnerability to hypertension. The offspring of parents with hypertension are at twice the risk of developing hypertension as children of parents with normal blood pressure (Brannon & Feist, 1997; Taylor, 2009).

Studies examining neurobiological causes of hypertension have centered on two factors central to the regulation of blood pressure: autonomic nervous system activity and mechanisms regulating sodium in the kidneys. When the sympathetic branch of the autonomic nervous system becomes active, one consequence is the constriction of

cardiovascular disease Afflictions in the mechanisms, including the heart, blood vessels, and their controllers, responsible for transporting blood to the body's tissues and organs. Psychological factors may play important roles in such diseases and their treatments.
stroke/cerebral vascular accident (CVA) Temporary blockage of blood vessels supplying the brain, or a rupture of vessels in the brain, resulting in temporary or permanent loss of brain functioning.
hypertension Major risk factor for stroke and heart and kidney disease that is intimately related to psychological factors. Also known as *high blood pressure.*
essential hypertension High blood pressure with no verifiable physical cause, which makes up the overwhelming majority of high blood pressure cases.



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▲ African Americans suffer from hypertension in disproportionately high numbers.

blood vessels, which produces greater resistance against circulation—that is, blood pressure is elevated (Guyton, 1981). Because the sympathetic nervous system is responsive to stress, many investigators have long assumed that stress is a major contributor to essential hypertension. Sodium and water regulation, one of the functions of the kidneys, is also important in regulating blood pressure. Retaining too much salt increases blood volume and heightens blood pressure. This is one reason that people with hypertension are often told to restrict their intake of salt.

Psychological factors, such as personality, coping style, and (again) level of stress, have been used to explain individual differences in blood pressure (Taylor, 2009; Winters & Schneiderman, 2000). For example, in a review of 28 studies, Uchino and colleagues (1996) found a strong relationship between levels of social support and blood pressure. Loneliness, depression, and feelings of uncontrollability are psychological mechanisms that may contribute to the association between hypertension and social support. In fact, one recent study with married couples found that practicing a "warm touch" (frequent affectionate touching) as a way of communicating love and support significantly decreased blood pressure (Holt-Lunstad, Birmingham, & Light, 2008).

A long-term study identified two psychological factors, each of which almost doubles the risk of hypertension: hostility, particularly in interpersonal relations, and a sense of time urgency or impatience. To reach this conclusion, more than 5,000 adults (including both blacks and whites) were followed for 15 years in the Coronary Artery Risk Development in Young Adults (CARDIA) study (Yan et al., 2003). It is likely that the combination of these two factors is an even more powerful risk factor. Also, both anger and hostility have been associated with increases in blood pressure in the laboratory setting (Jamner, Shapiro, Goldstein, & Hug, 1991; King, Taylor, Albright, & Haskell, 1990; Miller, Smith, Turner, Guijarro, & Hallet, 1996).

The notion that hostility or repressed hostility predicts hypertension (and other cardiovascular problems) can be traced back to Alexander (1939), who suggested that an inability to express anger could result in hypertension and other cardiovascular problems. What may be more important is not whether anger is suppressed but rather how often anger and hostility are experienced during stressful situations and expressed to others (Brondolo et al., 2003; Ironson et al., 1992; Miller et al., 1996; Winters & Schneiderman, 2000). Let's return to the case of John for a moment. John clearly suffered from hypertension. Do you detect any anger in John's case study? John's hypertension may be related to his stressful lifestyle, frustration levels, and hostility. The ability to control anger by expressing these feelings constructively is associated with markedly lower blood pressure in the general population (Davidson, MacGregor, Stuhr, Dixon, & MacLean, 2000; Taylor, 2009), suggesting it might help patients with hypertension, too. So the causes of hypertension seem to include the interaction of high stress reactivity (possibly genetically based), high exposure to stress, and inappropriate coping skills and reactions, often involving hostility and anger (al'Absi & Wittmers, 2003; Taylor, 2009).

Coronary Heart Disease

It may not surprise you that psychological and social factors contribute to high blood pressure, but can changes in behavior and attitudes prevent heart attacks? The answers are still not entirely clear, but increasing evidence indicates that psychological and social factors are implicated in coronary heart disease (Emery et al., 2011; Winters & Schneiderman, 2000).

Coronary heart disease (CHD), quite simply, is a blockage of the arteries supplying blood to the heart muscle (the *myocardium*). A number of terms describe heart disease. Chest pain resulting from partial obstruction of the arteries is called *angina pectoris* or, usually, just *angina*. *Atherosclerosis* occurs when a fatty substance or plaque builds up inside the arteries and causes an obstruction. *Ischemia* is the name for deficiency of blood to a body part caused by the narrowing of the arteries by too much plaque. And *myocardial infarction,* or *heart attack,* is the death of heart tissue when a specific artery becomes clogged with plaque. Arteries can constrict or become blocked for a variety of reasons other than plaque. For example, a blood clot might lodge in the artery.

It seems clear that we inherit a vulnerability to CHD (and to many other physical disorders), and other factors such as diet, exercise, and culture make important contributions to our cardiovascular status (Thoresen & Powell, 1992). But what sort of psychological factors contribute to CHD?

A variety of studies suggest strongly that stress, anxiety, and anger, combined with poor coping skills and low social support, are implicated in CHD (Emery et al., 2011; Lett et al., 2005; Matthews, 2005; Suls & Bunde, 2005; Taylor, 2009; Winters & Schneiderman, 2000). Severe stress, as in learning that a family member suddenly died, can lead on rare occasions to a condition called *myocardial stunning,* which is basically heart failure (Wittstein et al., 2005). Some studies indicate that even healthy men who experience stress are later more likely to experience CHD than low-stress groups (Rosengren, Tibblin, & Wilhelmsen, 1991). For such individuals, stress-reduction procedures may prove to be an important preventive technique.

There is a great deal of evidence on the value of stress-reduction procedures in preventing future heart attacks (Emery et al., 2011; Williams & Schneiderman, 2002). In one report summarizing results from 37 studies and using analytic procedures that combine the results from these studies (meta-analysis), the effects of stress-reduction programs on CHD were apparent. Specifically, as a group, these studies yielded a 34% reduction in death from heart attacks; a 29% reduction in the recurrence of heart attacks; and a significant positive effect on blood pressure, cholesterol levels, body weight, and other risk factors for CHD (Dusseldorp, van Elderen, Maes, Meulman, & Kraaij, 1999). Another major clinical study confirmed the benefits of stress reduction and exercise in reducing emotional distress and improving heart function and risk for future attacks in a group of individuals with established heart disease (Blumenthal et al., 2005). This brings us to an important question: Can we identify, before an attack, people who are under a great deal of stress that might make them susceptible to a first heart attack? The answer seems to be yes, but the answer is more complex than we first thought.

Clinical investigators reported several decades ago that certain groups of people engage in a cluster of behaviors in stressful situations that seem to put them at considerable risk for CHD. These behaviors include excessive competitive drive, a sense of always being pressured for time, impatience, incredible amounts of energy that may show up in accelerated speech and motor activity, and angry outbursts. This set of behaviors, which came to be called the **type A behavior pattern**, was first identified by two cardiologists, Meyer Friedman and Ray Rosenman (1959, 1974). The **type B behavior pattern**, also described by these clinicians, applies to people who basically do not have type A attributes. In other words, the type B individual is more relaxed, less concerned about deadlines, and seldom feels the pressure or perhaps the excitement of challenges or overriding ambition.

The concept of the type A personality or behavior pattern is widely accepted in our hard-driving, goal-oriented culture. Indeed, some early studies supported the concept of type A behavior as putting people at risk for CHD (Friedman & Rosenman, 1974). But the most convincing evidence came from two large prospective studies that followed thousands of patients over a long period to determine the relationship of their behavior to heart disease. The first study was the Western Collaborative Group Study (WCGS). In this project, 3,154 healthy men, age 39 to 59, were interviewed at the beginning of the study to determine their typical behavioral patterns. They were then followed for 8 years. The basic finding was that the men who displayed a type A behavior pattern at the beginning of the study were at least twice as likely to develop CHD as the men with a type B behavior pattern. When the investigators analyzed the data for the younger men in the study (age 39 to 49), the results were even more striking, with CHD developing approximately 6 times more often in the type A group than in the type B group (Rosenman et al., 1975).

A second major study is the Framingham Heart Study that has been ongoing for more than 40 years (Haynes, Feinleib, & Kannel, 1980) and has taught us much of what we know about the development and course of CHD. In this study, 1,674 healthy men and women were categorized by a type A or type B behavior pattern and followed for 8 years. Again, both men and women with a type A pattern were more than twice as likely to develop CHD as their type B counterparts (in men, the risk was nearly 3 times as great). For women with type A behavior pattern, the likelihood of developing CHD was highest for those with a low level of education (Eaker, Pinsky, & Castelli, 1992).

Population-based studies in Europe essentially replicated these results (De Backer, Kittel, Kornitzer, & Dramaix, 1983; French-Belgian Collaborative Group, 1982). It is interesting that a large study of Japanese men conducted in Hawaii did *not* replicate these findings (Cohen & Reed, 1985). The prevalence of type A behavior among Japanese men is much lower than among men in the United States (18.7% versus approximately 50%). Similarly, the prevalence of CHD is equally low in Japanese men (4%, compared to 13% in American men in the Framingham study) (Haynes & Matthews, 1988). In a study that illustrates the effects of culture more dramatically, 3,809 Japanese Americans were classified into groups according to how "traditionally Japanese" they were (in other words, they spoke Japanese at home, retained traditional Japanese values and behaviors, and so on). Japanese Americans who were the "most Japanese" had the lowest incidence of CHD, not significantly different from Japanese men in Japan. In contrast, the group that was the "least Japanese" had a 3 to 5 times greater incidence of CHD levels (Marmot & Syme, 1976; Matsumoto, 1996). Clearly, sociocultural differences are important.

Despite these straightforward results, at least in Western cultures, the type A concept has proved more complex and elusive than scientists had hoped. First, it is difficult to determine whether someone is type A from structured interviews, questionnaires, or other measures of this construct, because the measures often do not agree with one another. Many people have *some* characteristics of type A but not all of them, and others present with a mixture of types A and B. The notion that we can divide the world into two types of people—an assumption underlying the early work in this area—has long since been discarded. As a result, subsequent studies did not necessarily support the relationship of type A behavior to CHD (Dembroski & Costa, 1987; Hollis, Connett, Stevens, & Greenlick, 1990).

coronary heart disease (CHD) Blockage of the arteries supplying blood to the heart muscle; a major cause of death in Western culture, with social and psychological factors involved.
type A behavior pattern Cluster of behaviors including excessive competitiveness, time-pressured impatience, accelerated speech, and anger, originally thought to promote high risk for heart disease.
type B behavior pattern Cluster of behaviors including a relaxed attitude, indifference to time pressure, and less forceful ambition; originally thought to promote low risk for heart disease.

The Role of Chronic Negative Emotions

At this point, investigators decided that something might be wrong with the type A construct itself (Matthews, 1988; Rodin & Salovey, 1989). A consensus developed that some—but not all—behaviors and emotions representative of the type A personality might be important in the development of CHD. One factor that seems to be responsible for much of the type A–CHD relationship is anger (Miller et al., 1996), which will come as no surprise if you read the Ironson study in Chapter 2 and the previous section on hypertension. As you may remember, Ironson and colleagues (1992) compared increased heart rate when they instructed individuals with heart disease to imagine situations or events in their own lives that made them angry with heart rates when they imagined other situations, such as exercise. They found that anger impaired the pumping efficiency of the heart, putting these individuals at risk for dangerous disturbances in heart rhythm (arrhythmias). This study confirms earlier findings relating the frequent experience of anger to later CHD (Dembroski, MacDougall, Costa, & Grandits, 1989; Houston, Chesney, Black, Cates, & Hecker, 1992; Smith, 1992). Results from an important study strengthen this conclusion. Iribarren and colleagues (2000) evaluated 374 young, healthy adults, both white and African American, over a period of 10 years. Those with high hostility and anger showed evidence of coronary artery calcification, an early sign of CHD.

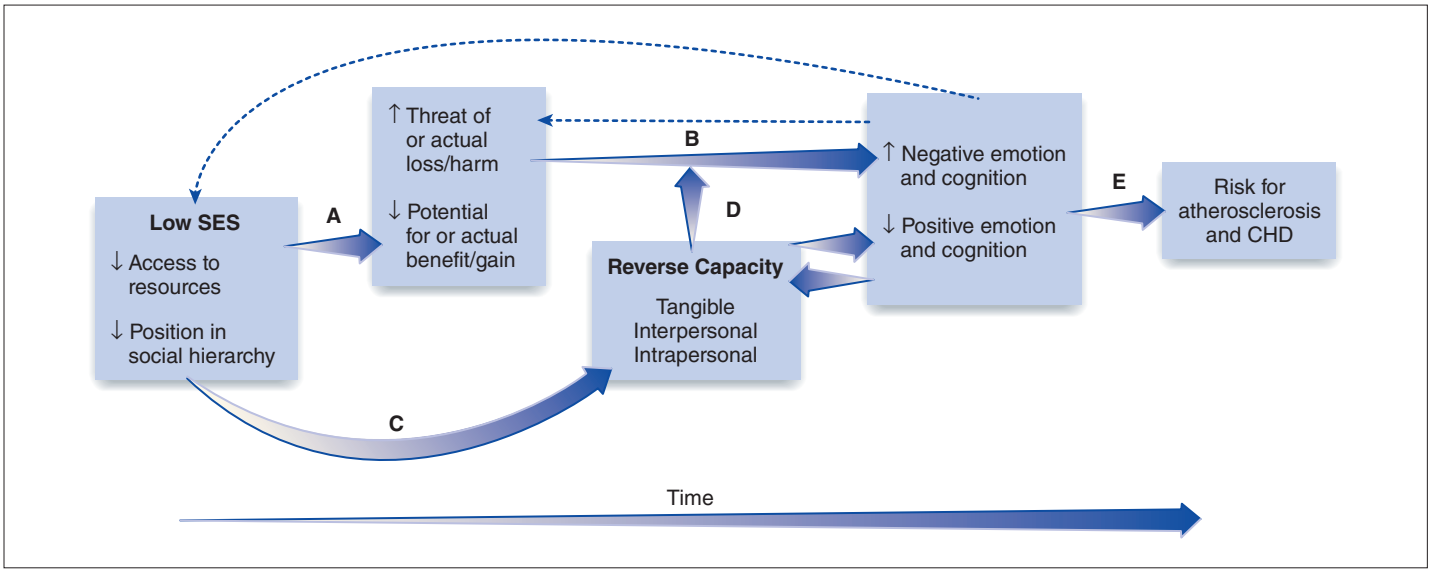
Is type A irrelevant to the development of heart disease? Most investigators conclude that some components of the type A construct are important determinants of CHD, particularly a chronically high level of negative affect (such as anger) and the time urgency or impatience factor (Matthews, 2005; Thoresen & Powell, 1992; Williams, Barefoot, & Schneiderman, 2003; Winters & Schneiderman, 2000). Recall again the case of John, who had all the type A behaviors, including time urgency, but also had frequent angry outbursts. But what about people who experience closely related varieties of negative affect on a chronic basis? Look back to Figure 7.2 and notice the close relationship among stress, anxiety, and depression. Some evidence indicates that the physiological components of these emotions and their effects on the cardiovascular system may be identical or at least similar (Suls & Bunde, 2005). We also know that the emotion of anger, so commonly associated with stress, is closely related to the emotion of fear, as evidenced in flight or fight response. Fight is the typical behavioral action tendency associated with anger, and flight or escape is associated with fear. But our bodily alarm response, activated by an immediate danger or threat, is associated with both emotions.

Some investigators, after reviewing the literature, have concluded that anxiety and depression are as important as anger in the development of CHD (Albert, Chae, Rexrode, Manson, & Kawachi, 2005; Barlow, 1988; Frasure-Smith & Lesperance, 2005; Strike & Steptoe, 2005; Suls & Bunde, 2005), even anxious and depressive features noticeable at an early age (Grossardt, Bower, Geda, Colligan, & Rocca, 2009). In a study of 896 people who had suffered heart attacks, Frasure-Smith and colleagues (Frasure-Smith, Lesperance, Juneau, Talajic, & Bourassa, 1999) found that patients who were depressed were 3 times more likely to die in the year following their heart attacks than those who were not depressed, regardless of how severe their initial heart disease was. In a study of 1,017 patients with CHD, Whooley and colleagues (2008) found a 31% higher rate of cardiovascular events such as heart attacks or arrhythmias in patients with depressive symptoms compared to those without depressive symptoms. Severe depression, as in major depressive episodes, is particularly implicated in cardiovascular damage (Agatisa et al., 2005; Emery et al., 2011). Thus, it may be that the chronic experience of the negative emotions of stress (anger), anxiety (fear), and depression (ongoing) and the neurobiological activation that accompanies these emotions provide the most important psychosocial contributions to CHD, and perhaps to other physical disorders.

Investigators are also learning more about the process through which negative emotions contribute to CHD. Once again, the inflammatory processes associated with the stress response (and with all negative emotions) play a major role because inflammation directly contributes to atherosclerosis and heart failure (Matthews et al., 2007; Taylor, 2009). Gallo and Matthews (2003; Matthews, 2005) provide a model of the contribution of psychosocial factors to CHD (see ■ Figure 7.5). Lower socioeconomic status and relatively few resources or low prestige is in the first box. Stressful life events are in the second. Coping skills and social support contribute to a reserve capacity that may buffer the effects of stress, as represented in the third box. Both negative emotions and negative cognitive styles then constitute a major risk factor. Positive emotions and an optimistic style, on the other hand, reduce the risk of CHD (Giltay, Geleijnse, Zitman, Hoekstra, & Schouten, 2004) and may turn out to be just as important as negative emotions in their effects on CHD. Both negative and positive emotions are in the fourth box. This model summarizes nicely what we know about the influence of psychosocial factors on CHD.

Chronic Pain

Pain is not in itself a disorder, yet for most of us it is the fundamental signal of injury, illness, or disease. If we can't relieve the pain ourselves or we are not sure of its cause, we usually seek medical help. The National Institutes of Health has identified chronic pain as the costliest medical problem in America, affecting nearly 100 million individuals (Byrne & Hochwarter, 2006; Otis & Pincus, 2008). Overall the total cost of chronic pain, including treatments and indirect costs such as loss of productivity at work, have been estimated between $100 billion and $260 billion annually (Byrne & Hochwarter, 2006; Edwards, Campbell, Jamison, & Wiech, 2009). Americans spend at least $125 billion annually on treatment for chronic pain, including over-the-counter medication to reduce temporary pain from headaches, colds, and other minor disorders (Gatchel, 2005; Taylor, 2009). Pain is the cause of 80% of all visits to physicians (Gatchel, Peng, Peters, Fuchs, & Turk, 2007;



■ **Figure 7.5** The reserve capacity model for associations among environments of low socioeconomic status (SES), stressful experiences, psychosocial resources, and emotions and cognitions, which represent pathways to increased risk for CHD. Note: Arrow A depicts the direct influence of SES on exposure to stressful experiences. Arrow B indicates the direct impact of stress experiences on emotion and cognition. Arrow C shows that SES conditions and shapes the bank of resources (that is, the reserve capacity) available to manage stress. Arrow D shows that reserve capacity represents a potential moderator of the association between stress and emotional–cognitive factors. Arrow E indicates the direct effects of emotional–cognitive factors on intermediate pathways and risk for atherosclerosis and CHD. The dashed lines note the possible reverse influences. (Adapted from Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role?" *Psychological Bulletin, 129,* 34 (Figure 1), © 2003 American Psychological Association. Reprinted, with permission, from Matthews, K. A. (2005). Psychological perspectives on the development of coronary heart disease. *American Psychologist, 60*(8), 791 (Figure 2), © 2005 American Psychological Association.)

Turk & Gatchel, 2002), making it by far the most common reason to see a primary care physician (Otis, MacDonald, & Dobscha, 2006). Yet most researchers now agree that the cause of chronic pain and the resulting enormous drain on our health-care system *are substantially psychological and social* (Dersh, Polatin, & Gatchel, 2002; Gatchel et al., 2007; Taylor, 2009; Turk & Monarch, 2002).

There are two kinds of clinical pain: acute and chronic. **Acute pain** typically follows an injury and disappears once the injury heals or is effectively treated, often within a month. **Chronic pain**, by contrast, may begin with an acute episode but *does not decrease* over time, even when the injury has healed or effective treatments have been administered. Typically, chronic pain is in the muscles, joints, or tendons, particularly in the lower back. Vascular pain because of enlarged blood vessels may be chronic, as may headaches. Pain caused by the slow degeneration of tissue, as in some terminal diseases, and pain caused by the growth of cancerous tumors that impinge on pain receptors also may be chronic (Melzack & Wall, 1982; Otis & Pincus, 2008; Taylor, 2009).

To better understand the experience of pain, clinicians and researchers generally make a clear distinction between the subjective experience termed *pain,* reported by the patient, and the overt manifestations of this experience, termed *pain behaviors.* Pain behaviors include changing the way one sits or walks, continually complaining about pain to others, grimacing, and, most important, avoiding various activities, particularly those involving work or leisure. Finally, an emotional component of pain called *suffering* sometimes accompanies pain and sometimes does not (Fordyce, 1988; Liebeskind, 1991). Because they are so important, we first review psychological and social contributions to pain.

Psychological and Social Aspects of Pain

In mild forms, chronic pain can be an annoyance that eventually wears you down and takes the pleasure out of your life. Severe chronic pain may cause you to lose your job, withdraw from your family, give up the fun in your life, and focus your entire awareness on seeking relief. What is interesting for our purposes is that the *severity* of the pain does not seem to predict the *reaction* to it. Some individuals experience intense pain frequently yet continue to work productively, rarely seek medical services, and lead reasonably normal lives; others become invalids. These differences appear to be primarily the result of psychological factors (Dersh et al., 2002; Gatchel, 2005; Gatchel & Turk, 1999; Keefe, Dunsmore, & Burnett, 1992; Turk & Monarch,

acute pain Pain that typically follows an injury and disappears once the injury heals or is effectively treated.
chronic pain Enduring pain that does not decrease over time; may occur in muscles, joints, and the lower back; and may be caused by enlarged blood vessels or degenerating or cancerous tissue. Other significant factors are social and psychological.

2002). It will come as no surprise that these factors are the same as those implicated in the stress response and other negative emotional states, such as anxiety and depression (Ohayon & Schatzberg, 2003; Otis, Pincus, and Murawski, 2010) (see Chapters 4 and 6). The determining factor seems to be the individual's general sense of control over the situation: whether or not he or she can deal with the pain and its consequences in an effective and meaningful way. When a positive sense of control is combined with a generally optimistic outlook about the future, there is substantially less distress and disability (Bandura, O'Leary, Taylor, Gauthier, & Gossard, 1987; Gatchel & Turk, 1999; Keefe & France, 1999; Otis & Pincus, 2008; Zautra, Johnson, & Davis, 2005). Positive psychological factors are also associated with active attempts to cope, such as exercise and other regimens, as opposed to suffering passively (Gatchel & Turk, 1999; Lazarus & Folkman, 1984; Otis et al., 2010; Turk & Gatchel, 1999; Zautra et al., 2005), and successfully treating depression diminishes the experience of chronic pain (Teh, Zaslavsky, Reyolds, & Cleary, 2009).

To take one example, Philips and Grant (1991) studied 117 patients who suffered from back and neck pain after an injury. Almost all were expected to recover quickly, but fully 40% of them still reported substantial pain at 6 months, thereby qualifying for "chronic pain" status. Of the 60% who reported no pain at the 6-month point, most had been pain free since approximately 1 month after the accident. Furthermore, Philips and Grant report that the relationship between the experience of pain and the subsequent disability was not as strongly related to the intensity of the pain as other factors, such as personality and socioeconomic differences and whether the person planned to initiate a lawsuit concerning the injury. Preexisting anxiety and personality problems predict who will suffer chronic pain (Gatchel, Polatin, & Kinney, 1995; Taylor, 2009). Generally, a profile of negative emotion such as anxiety and depression, poor coping skills, low social support, and the possibility of being compensated for pain through disability claims predict most types of chronic pain (Dersh et al., 2002; Gatchel et al., 2007; Gatchel & Dersh, 2002; Gatchel & Epker, 1999). Conversely, developing a greater sense of control and less anxiety focused on the pain results in less severe pain and less impairment (Burns, Glenn, Bruehl, Harden, & Lofland, 2003; Edwards et al., 2009; Otis et al., 2010). Finally, Zautra and colleagues (2005) observed that positive affect in a group of 124 women with severe pain from arthritis or fibromyalgia predicted that they would have less pain in subsequent weeks than would women with lower levels of positive affect.

Examples of psychological influences on pain are encountered every day. Athletes with significant tissue damage often continue to perform and report relatively little pain. In an important study, 65% of war veterans wounded in combat reported feeling no pain when they were injured. Presumably, their attention was focused externally on what they had to do to survive rather than internally on the experience of pain (Melzack & Wall, 1982).

Social factors also influence how we experience pain (Fordyce, 1976, 1988). For example, family members who were formerly critical and demanding may become caring and sympathetic (Kerns, Rosenberg, & Otis, 2002; Otis & Pincus, 2008; Romano, Jensen, Turner, Good, & Hops, 2000).

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▲ It is not uncommon for people to feel specific pain in limbs that are no longer part of them.

©Bob Daemmrich/The Image Works

▲ Some people with chronic pain or disability cope extremely well and become high achievers.

This phenomenon is referred to as *operant* control of pain behavior because the behavior clearly seems under the control of social consequences. But these consequences have an uncertain relation to the amount of pain being experienced.

By contrast, a strong network of social support may reduce pain. Jamison and Virts (1990) studied 521 chronic pain patients (with back, abdominal, and chest conditions) and discovered that those who lacked social support from their families reported more pain sites and showed more pain behavior, such as staying in bed. These patients also exhibited more emotional distress *without* rating their pain as any more intense than participants with strong socially supportive families. The participants with strong support returned to work earlier, showed less reliance on medications, and increased their activity levels more quickly than the others. Even having just a photo of a loved one to look at reduces the experience of pain (Master et al., 2009).

Although these results may seem to contradict studies on the operant control of pain, different mechanisms may be at work. General social support may reduce the stress associated with pain and injury and promote more adaptive coping procedures and control. However, specifically reinforcing pain behaviors, particularly in the absence of social supports, may powerfully increase such behavior. These complex issues have not yet been entirely sorted out.

Biological Aspects of Pain

No one thinks pain is entirely psychological, just as no one thinks it is entirely physical. As with other disorders, we must consider how psychological and physical factors interact.

Mechanisms of Pain Experience and Pain Control. The *gate control theory of pain* (Melzack & Wall, 1965, 1982) accommodates both psychological and physical factors. According to this theory, nerve impulses from painful stimuli make their way to the spinal column and from there to the brain. An area called the *dorsal horns of the spinal column* acts as a "gate" and may open and transmit sensations of pain if the stimulation is sufficiently intense. Specific nerve fibers referred to as *small fibers* (A-delta and C fibers) and *large fibers* (A-beta fibers) determine the pattern, and the intensity, of the stimulation. Small fibers tend to open the gate, thereby increasing the transmission of painful stimuli, whereas large fibers tend to close the gate.

Most important for our purpose is that the brain sends signals back down the spinal cord that may affect the gating mechanism. For example, a person with negative emotions such as fear or anxiety may experience pain more intensely because the basic message from the brain is to be vigilant against possible danger or threat. Then again, in a person whose emotions are more positive or who is absorbed in an activity (such as a runner intent on finishing a long race), the brain sends down an inhibitory signal that closes the gate. Although many think that the gate control theory is overly simplistic (and it has recently been updated; see Melzack, 1999, 2005), research findings continue to support its basic elements, particularly as it describes the complex interaction of psychological and biological factors in the experience of pain (Edwards et al., 2009; Gatchel et al., 2007; Gatchel & Turk, 1999; Otis & Pincus, 2008; Turk & Monarch, 2002).

Endogenous Opioids. The neurochemical means by which the brain inhibits pain is an important discovery (Taylor, 2009). Drugs such as heroin and morphine are manufactured from opioid substances. It now turns out that **endogenous** (natural) **opioids** exist within the body. Called *endorphins* or *enkephalins,* they act much like neurotransmitters. The brain uses them to shut down pain, even in the presence of marked tissue damage or injury. Because endogenous opioids are distributed widely throughout the body, they may be implicated in a variety of psychopathological conditions, including eating disorders and, more commonly, the "runner's high" that accompanies the release of endogenous opioids after intense (and sometimes painful) physical activity. Bandura and colleagues (1987) found that people with a greater sense of self-efficacy and control had a higher tolerance for pain than individuals with low self-efficacy and that they increased their production of endogenous opioids when they were confronted with a painful stimulus. Most recently, Edwards et al. (2009) have articulated the neurobiological processes underlying the effectiveness of psychological coping procedures that successfully alter the experience of pain. Certain procedures, such as reappraising the significance of the pain instead of catastrophizing or thinking the worst about it, activate a variety of brain circuits that modulate or diminish pain experience and allow for more normal functioning.

Gender Differences in Pain

Men and women seem to experience different types of pain. On the one hand, in addition to menstrual cramps and labor pains, women suffer more often than men from migraine headaches, arthritis, carpal tunnel syndrome, and temporomandibular joint (TMJ) pain in the jaw (Lipchik, Holroyd, & Nash, 2002; Miaskowski, 1999). Men, on the other hand, have more cardiac pain and backache. Both males and females have endogenous opioid systems, although in males it may be more powerful. But women seem to have additional pain-regulating mechanisms that may be different. The female neurochemistry may be based on an estrogen-dependent neuronal system that may have evolved to cope with the pain associated with reproductive activity (Mogil, Sternberg, Kest, Marek, & Liebeskind, 1993). It is an "extra" pain-regulating pathway in females that, if taken away by removing hormones, has no implications for the remaining pathways, which continue to work. One implica-

endogenous opioids Substance occurring naturally throughout the body that functions like a neurotransmitter to shut down pain sensation even in the presence of marked tissue damage. These opioids may contribute to psychological problems such as eating disorders. Also known as an *endorphin* or *enkephalin.*

tion of this finding is that males and females may benefit from different kinds of drugs, different kinds of psychological interventions, or unique combinations of these treatments to best manage and control pain.

Chronic Fatigue Syndrome

Chronic fatigue syndrome (CFS) is prevalent throughout the Western world (Jason, Fennell, & Taylor, 2006; Prins, van der Meer, & Bleijenberg, 2006). The symptoms of CFS, listed in Table 7.2, have been attributed to various causes including viral infection—specifically the Epstein-Barr virus (Straus et al., 1985) or, most recently, XMRV, a retrovirus with some similarities to HIV (Kean, 2010)—and immune system dysfunction (Strauss, 1988), exposure to toxins, or clinical depression (Chalder, Cleare, & Wessely, 2000; Costa e Silva & De Girolamo, 1990). Although promising leads appear on occasion, no evidence has yet to support any of these hypothetical physical causes (Chalder et al., 2000; Jason et al., 2003; Kean, 2010; Prins et al., 2006). Jason and colleagues (1999) conducted a sophisticated study of the prevalence of CFS in the community and reported that 0.4% of their sample was determined to have CFS, with higher rates in Latino and African American respondents com-

Table 7.2 Definition of Chronic Fatigue Syndrome

Inclusion Criteria

1. Clinically evaluated, medically unexplained fatigue of at least 6 months duration that is
   - of new onset (not lifelong)
   - not resulting from ongoing exertion
   - not substantially alleviated by rest
   - a substantial reduction in previous level of activities
2. The occurrence of four or more of the following symptoms:
   - Subjective memory impairment
   - Sore throat
   - Tender lymph nodes
   - Muscle pain
   - Joint pain
   - Headache
   - Unrefreshing sleep
   - Postexertional malaise lasting more than 24 hours

Source: Adapted from Fukuda, K., Straus, S. E., Hickie, I., Sharpe, M. B., Dobbins, J. G., & Komaroff, A. L. (1994). Chronic fatigue syndrome: A comprehensive approach to its diagnosis and management. *Annals of Internal Medicine, 121,* 953–959.

Discussing Diversity

The Influence of Culture on Individual Health and Development: Female Reproduction around the World

The study of Psychology and the many different mental states and possible disorders experienced by individuals requires a high degree of sensitivity to the fact that people are inexorably intertwined with the cultural values and experiences in which they are raised. A fascinating example of varying cultural influences, and the psychological and physical effects of such experiences, can be seen through an examination of female reproduction around the world. It turns out that even this most basic of aspect of human biology is heavily influenced by cultural, environmental, and resulting psychological components. Women's ability to reproduce begins during a process called menarche, which refers to a girl's first menstrual bleeding that signals the beginning of fertility. Women generally are able to reproduce throughout adulthood until they reach menopause, which refers to the cessation of the menstrual cycle and signals the end of fertility. Although all women experience the events of menarche and menopause, the experience can be quite different depending on the cultural, environmental, and resulting psychological context in which these processes occur.

The timing of menarche can be influenced by cultural and environmental factors. For instance, differences in nutrition during the past several decades may have contributed to an earlier age of menarche, and such differences may help explain differences in the age of onset of menarche seen in different parts of the world (Euling et al., 2008). In addition, girls in more stressful home environments and in father-absent homes experience menarche earlier, perhaps because from an evolutionary perspective these events signal the need to leave the home and start one's own family (Weisfeld & Woodward, 2004).

There also are significant cross-cultural differences in the psychological and social experience of menopause, with some cultures celebrating each transition and others considering these issues taboo or cause for shame. For instance, in some cultures the loss of the ability to reproduce may be associated with diminished health or social standing (such as in Italy), whereas in other cultures (such as in China and India) menopause confers a higher and more prestigious place in society (Fu, Anderson & Courtney, 2003; Gifford, 1994). Interestingly, these different perceptions of menopause not only influence how different cultures react to menopausal women but also can affect the occurrence of menopausal symptoms, with those for whom menopause is seen as a positive life transition experiencing fewer or no negative symptoms (Fu et al., 2003). All of these findings point out how sensitive clinicians must be to the various cultural differences clients bring with them to therapy and how culture might influence how clients experience and describe their current problems (e.g., Ryder et al., 2008). Can you think of some ways in which disorders such as Major Depression and Bipolar may be influenced by culture and experienced and expressed differently by those with different backgrounds?