Getting Help

As we mention in this chapter, most people who would meet the diagnostic criteria for a personality disorder do not enter treatment, at least not voluntarily. Although their interpersonal problems are pervasive and deeply ingrained, they are reluctant or completely unable to see the active role that they play in maintaining their own misfortunes (regardless of their origins). The pejorative way in which personality disorders are sometimes portrayed may make some people reluctant to acknowledge that they have a personality disorder. We prefer to discuss these problems in terms of personality limitations or maladaptive response styles. No one is perfect. Being able to recognize your own weaknesses is a sign that you are open-minded and willing to change. This is the first important step toward improvement.

It also helps to have a little compassion for yourself, along with the determination to work toward a lasting change in the way you relate to the people and events of your life. *Lost in the Mirror: An Inside Look at Borderline Personality Disorder* by Richard Moskovitz, provides an insightful and sympathetic guide to the painful emotional experiences associated with borderline personality disorder. It also illustrates ways in which these symptoms affect the lives of patients, their families, and their friends.

If you are interested in help because you have to deal with someone who you think may have a personality disorder, you probably feel confused, frustrated, and angry. You may also feel extremely guilty if you blame yourself for problems in the relationship or for the other person's unhappiness. This may be especially true if you are involved in a romantic relationship or must work closely with someone who might meet the criteria for a PD. Fortunately, it is often possible to adapt to such interactions. Several self-help guides provide advice about getting along with difficult people. One good example is *Fatal Flaws: Navigating Destructive Relationships with People with Disorders of Personality and Character* (Yudofsky, 2005). Most recommend that you begin by learning about the predictable nature of the other person's style. Recognize the presence of personality weaknesses and learn how to adapt to them. You must also accept the limits of your own ability to control the other person or to get him or her to change.

Sometimes the only solution is to end the relationship. At their most extreme, people with personality disorders cannot form reciprocal, mutually satisfying relationships with other people. This is particularly true in the case of antisocial personality disorder. Some unscrupulous people repeatedly abuse, exploit, and cheat others. We all may run across such people from time to time, and we need to learn how to protect ourselves. Robert Hare, an expert on psychopathy, concludes his book *Without Conscience* with a brief "survival guide" that may help you minimize your risk. He notes, for example, that we should be aware of the symptoms and interpersonal characteristics of psychopathy. We should be cautious in high-risk situations and know our own weaknesses. Hare's advice may be extremely helpful to someone who finds himself or herself trapped in a relationship with someone who is a psychopath. In fact, you may want to speak to a therapist or counselor to figure out why you have become involved in such an unequal, nonreciprocal relationship.

SUMMARY

- **Personality disorders** are defined in terms of rigid, inflexible, maladaptive ways of perceiving and responding to oneself and one's environment that lead to social or occupational problems or subjective distress. This pattern must be pervasive across a broad range of situations, and it must be stable and of long duration.
- Personality disorders are controversial for a number of reasons, including their low diagnostic reliability and the tremendous overlap among specific personality disorder categories.
- Many systems have been proposed to describe the fundamental dimensions of human personality. One popular alternative is the five-factor model, which includes the basic traits of neuroticism, extraversion, openness to experience, agreeableness, and conscientiousness. Extreme variations in any of these traits—being either pathologically high or low—can be associated with personality disorders.
- DSM-IV-TR lists 10 types of personality disorder, arranged in three clusters. There is considerable overlap among and between these types. Cluster A includes **paranoid, schizoid,** and **schizotypal personality disorders.** These categories generally refer to people who are seen as being odd or eccentric. Cluster B includes **antisocial, borderline, histrionic,** and **narcissistic personality disorders.** People who fit into this cluster are generally seen as being dramatic, unpredictable, and overly emotional. Cluster C includes **avoidant, dependent,** and **obsessive–compulsive personality disorders.** The common element in these disorders is presumably anxiety or fearfulness.
- Dimensional approaches to the description of personality disorder provide an important alternative to this categorical system. These procedures rate a person on a number of traits, such as those included in the five-factor model. Dimensional

classification systems have the advantage of being better able to account for similarities and differences among people with various combinations of personality traits.

- The overall prevalence of personality disorders among adults in the general population (i.e., the percentage who qualify for the diagnosis of at least one type) is approximately 10 percent. The highest prevalence rates for specific types of personality disorders are usually found for obsessive–compulsive, antisocial, and avoidant personality disorders, which may affect 3 or 4 percent of adults. Prevalence rates for other specific types tend to be approximately 1 or 2 percent of the population (or less).
- The disorders listed in Cluster A, especially schizoid and schizotypal personality disorders, have been viewed as possible antecedents or subclinical forms of schizophrenia. They are defined largely in terms of minor symptoms that resemble the hallucinations and delusions seen in the full-blown disorder, as well as peculiar behaviors that have been observed among the first-degree relatives of schizophrenic patients.
- The most important features of borderline personality disorder revolve around a pervasive pattern of instability in self-image, in interpersonal relationships, and in mood. Research regarding the etiology of borderline personality disorder has focused on two primary areas. One involves the impact of chaotic and abusive families. The other is concerned with the premature separation of children from their parents. Both sets of factors presumably can lead to problems in emotional regulation.
- **Psychopathy** and antisocial personality disorder are two different attempts to define the same disorder. The DSM-IV-TR definition of ASPD places primary emphasis on social deviance in adulthood (repeated lying, physical assaults, reckless and irresponsible behavior). The concept of psychopathy places greater emphasis on emotional and interpersonal deficits, such as lack of remorse, lack of empathy, and shallow emotions.
- Treatment for schizotypal and borderline personality disorders often involves the use of antipsychotic medication or antidepressant medication. Various types of psychological interventions, including dialectical behavior therapy, are frequently employed with borderline patients. People with antisocial personality disorder seldom seek treatment voluntarily. When they do, the general consensus among clinicians is that it is seldom effective.

The Big Picture

CRITICAL THINKING REVIEW

- **What is the difference between being eccentric and having a personality disorder?**

  People who are unconventional are not necessarily disagreeable or difficult interpersonally. The concept of personality disorder, on the other hand, carries the assumption that the characteristic features by which they are defined are also associated with subjective distress or social impairment . . . (see p. 219)

- **In what ways are borderline and narcissistic personality disorders similar?**

  People in both groups can be hypersensitive and self-centered, thinking that others should be especially concerned with their needs and interests. When these expectations are not met, they may react with intense anger . . . (see p. 227)

- **What are the advantages of a dimensional approach that would describe personality problems as variations on normal personality traits?**

  This alternative perspective would minimize problems associated with comorbidity (a person meeting criteria for more than one diagnostic category) and arbitrary cut-off points between normal and abnormal personality . . . (see pp. 228–229)

- **Which personality disorders are least likely to change as a person gets older?**

  Schizoid and schizotypal personality disorders are relatively intractable problems and are associated with enduring social isolation and occupational difficulties . . . (see p. 231)

- **Why are personality disorders so difficult to treat?**

  Many people with personality disorders are unable to recognize the nature of their problems. They may also be uncomfortable with the type of close, personal relationship that must be formed with a therapist if psychological treatment is going to be successful . . . (see p. 235)

- **What is the difference between antisocial personality disorder and psychopathy?**

  The DSM-IV definition of antisocial personality disorder is focused largely on repeated conflict with authorities and failure to conform to social norms, while the definition of psychopathy places greater importance on emotional deficits (lack of remorse, shallow emotions) and personality traits (impulsivity, grandiosity, lack of responsibility) . . . (see p. 241)

KEY TERMS

antisocial personality disorder
avoidant personality disorder
borderline personality disorder
cross-cultural psychology
dependent personality disorder
histrionic personality disorder
impulse control disorders
narcissistic personality disorder
obsessive–compulsive personality disorder
paranoid personality disorder
personality
personality disorder
psychopathy
schizoid personality disorder
schizotypal personality disorder

10 Eating Disorders

◀ Already petite actress Natalie Portman lost 20 pounds to play the lead role in *Black Swan*, an indicator of the pressures on ballerinas, and actresses, to be extremely thin—a risk factor for eating disorders.

Popular culture in the United States is obsessed with physical appearance. We are told that "beauty is only skin deep," but the entertainment, cosmetic, fashion, and diet industries are eager to convince young people that "looks are everything." Perfect men are handsome, muscular, and successful. Perfect women are beautiful and thin—extremely thin. In fact, women's thinness is equated with beauty, fitness, success, and ultimately with happiness. Given our national obsession with appearance, diet, and weight, we should not be surprised that many people, especially young women, become obsessed to the point of developing eating disorders.

The Big Picture

- How can you tell if someone has an eating disorder?
- How do media images of women contribute to eating disorders?
- Do men get eating disorders?
- Why do some girls and women develop eating disorders while others do not?
- What treatments work for anorexia and bulimia?
- Can eating disorders be prevented?

OVERVIEW

Eating disorders are severe disturbances in eating behavior. Some experts suggest that *dieting disorder* is a more accurate term, because dread of weight gain and obsession with weight loss often are central features of eating disorders. DSM-IV-TR lists two major types of eating disorders: anorexia nervosa and bulimia nervosa. The most obvious characteristic of **anorexia nervosa** is extreme emaciation, or more technically, the refusal to maintain a minimally normal body weight. The term *anorexia* literally means "loss of appetite," but this is a misnomer. People with anorexia nervosa *are* hungry, yet they starve themselves nevertheless. Some unfortunate victims literally starve themselves to death.

The images of women portrayed in advertising and the popular media contribute to the development of eating disorders.

Bulimia nervosa is characterized by repeated episodes of binge eating, followed by inappropriate compensatory behaviors such as self-induced vomiting, misuse of laxatives, or excessive exercise. The literal meaning of the term *bulimia* is "ox appetite" ("hungry enough to eat an ox"). But people with bulimia nervosa typically have a normal appetite. Paradoxically, the problem often results from trying to maintain a weight below the body's natural set point, an effort that results in ongoing struggle with binge eating and attempts to compensate. Most sufferers view binge eating as a failure of control, when it really is their body's natural reaction to hunger caused by unnatural weight suppression (Keel et al., 2007).

Both anorexia and bulimia are about 10 times more common among females than males, and they develop most commonly among women in their teens and early twenties. The increased incidence among young people reflects both the intense focus on young women's physical appearance and the difficulties many adolescent girls have in adjusting to the rapid changes in body shape and weight that begin with puberty (Field & Kitos, 2010). According to the National Centers for Disease Control and Prevention, at any point in time, 44 percent of high school females are attempting to lose weight compared with 15 percent of males (Serdula et al., 1993). Many adolescent boys want to *gain* weight in order to look bigger and stronger (see Eating Disorders in Males on p. 252).

A national survey found that almost half of American women have a negative body image, particularly concerning their waist, hips, and/or thighs (Cash & Henry, 1995; see Figure 10.1). European American and Latina women report higher rates of body dissatisfaction than African Americans (Bay-Cheng et al., 2002; Grabe & Shibley-Hyde, 2006), with dissatisfaction increasing from the 1980s to the 1990s among white women (Cash et al., 2004). Fortunately, levels of body dissatisfaction declined between the 1990s and 2000s among both European American and African American women, perhaps indicating a growing resistance to the popular media's culture of thinness (Cash et al., 2004).

In this chapter, we discuss the symptoms of anorexia nervosa separately from those of bulimia nervosa, because they differ considerably. We combine the two disorders when reviewing diagnosis, frequency, and causes, however, because they share many developmental similarities. For example, many people with anorexia nervosa also binge and purge on occasion; many people with bulimia nervosa have a history of anorexia nervosa. When considering treatment, we again discuss the two disorders separately, reflecting the important differences in the focus and effectiveness of therapy for each disorder. We begin with a case study.

CASE STUDY Serrita's Anorexia

Serrita was an attractive, well-dressed, and polite 15-year-old high school sophomore who was living in a friendly joint-custody arrangement. She spent alternating weeks living with each of her successful, middle-class parents. Serrita was an excellent student. Her mother described her as a "sweet girl who never gave me an ounce of worry—until now." When she was first seen by a clinical psychologist for the treatment of anorexia nervosa, Serrita was 5 feet 2 inches tall and weighed 81 pounds. Serrita's gaunt appearance was painfully obvious to anyone who looked at her. Despite her constant scrutiny of her own body, however, Serrita firmly denied that she was too thin. Instead, she insisted that she looked "almost right." She was still on a diet, and every day she carefully inspected her stomach, thighs, hips, arms, and face for any signs of fat. Although Serrita was generally pleased with the image she saw in her bathroom mirror, she remained deathly afraid of gaining weight. She monitored her food intake with incredible detail. She could recite every item of food she had consumed recently and discuss its caloric and fat content.

Serrita began her diet nine months earlier after visiting her family doctor, who told her that she could stand to lose a pound or two. At the time, Serrita weighed 108 pounds, a normal weight for her age, height, and body type. Serrita said that the doctor's comment motivated her to begin a diet. She wanted to look like the women in her favorite magazines, but she felt that she wasn't the "cute, all-American girl"—she saw herself as too short, too dark, and her features as too sharp. Serrita secretly hoped that having a "great body" would compensate for her perceived inadequacies.

Serrita's diet began normally enough. She quickly lost the 6 pounds she wanted to lose, and without really planning to do so, she simply continued her diet. She developed the habit of scouring her image in the mirror and invariably found some spot that was just a bit "too fat." As a result, she continually set a new goal to lose another couple of pounds. Serrita weighed herself constantly and said that the bathroom scale became her "best friend." Her friends' and family's compliments soon turned into worried warnings, but privately Serrita was exhilarated. To her, the concerned remarks only proved that her diet was working.

Serrita's diet became extreme and rigid, and she adhered obsessively to its routine. Breakfast consisted of one slice of dry wheat toast and a small glass of orange juice. Lunch was either an apple or a small salad without dressing. In between meals, Serrita drank several diet colas, which helped control her constant, gnawing appetite. Dinner typically was a family meal whether Serrita was at her mom's or her dad's house. During these meals, Serrita picked at whatever she was served. Sometimes her parents would plead with her to eat more, and Serrita would eat a bit to appease them. On occasion, perhaps once a week, Serrita forced herself to vomit after dinner, because she felt that her parents made her eat too much.

Serrita's parents eventually became so concerned that they made her go to see her family physician. The physician also was very worried about Serrita's low weight, and she discovered that Serrita had not menstruated in over six months. The physician said that Serrita was suffering from anorexia nervosa. She immediately made a referral to a psychologist as well as to a nutritionist, who, the physician hoped, would correct Serrita's extreme views about dieting.

In talking with the psychologist, Serrita agreed that she understood why everyone was concerned about her health. She knew about anorexia nervosa, which she realized was a serious problem. Serrita even hinted that she knew that she was suffering from anorexia nervosa. Nevertheless, she steadfastly denied that she needed to gain weight. Although she was happy to talk with the psychologist, she was not prepared to change her eating habits. Serrita was deathly afraid that eating even a little more would cause her to "lose control" and "turn into a blimp." She was *proud* of her mastery of her hunger. She was not about to give up the control she had fought so hard to gain.

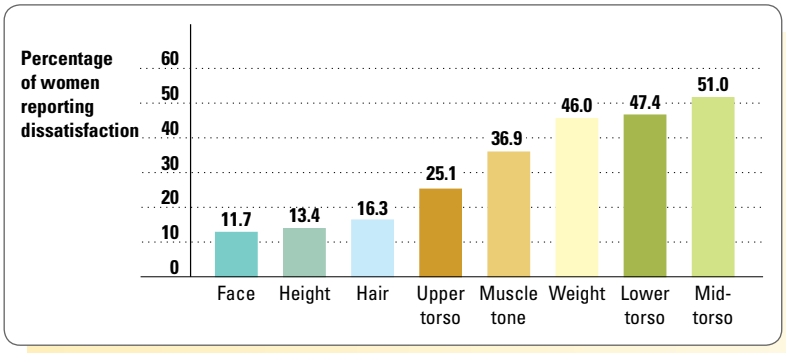


FIGURE 10.1

Percentage of females reporting that they were "very or mostly dissatisfied" with specific physical attributes in a national sample of women aged 18 to 70.

Source: T. F. Cash and P. E. Henry, 1995, "Women's Body Images: The Results of a National Survey in the U.S.A.," *Sex Roles, 33,* pp. 19–28. Copyright © 1995. Reprinted by permission of Springer Science and Business Media.

Symptoms of Anorexia

Serrita showed all the classic symptoms of anorexia nervosa: extreme emaciation, a disturbed perception of her body, an intense fear of gaining weight, and, in women, the cessation of menstruation. Serrita also exhibited a number of problems that are commonly associated with anorexia nervosa but are not defining symptoms: obsessive preoccupation with food, occasional purging, and a "successful" struggle for control over persistent hunger. Finally, Serrita did not suffer from a few important problems that are sometimes associated with anorexia nervosa, particularly mood disturbance, sexual difficulties, a lack of impulse control, and medical problems secondary to the weight loss.

REFUSAL TO MAINTAIN A NORMAL WEIGHT

The most obvious and most dangerous symptom of anorexia nervosa is a *refusal to maintain a minimally normal body weight.* Like Serrita, anorexia nervosa often begins with a diet to lose just a few pounds. The diet goes awry, however, and losing weight eventually becomes the key focus. Weight falls well below the normal range and often plummets to dangerously low levels.

DSM-IV-TR contains no formal cutoff as to how thin is too thin but suggests 85 percent of expected body weight as a rough guideline. The *body mass index,*[1] a calculation derived from weight and height, is another useful way to determine whether someone is significantly underweight. Both DSM-IV-TR and the body mass cutoffs represent weights well beyond "thin" and into the realm of "emaciated," but weight loss often is more extreme: The average victim of anorexia loses 25 to 30 percent of normal body weight. Unlike Serrita, who was fortunate in this sense, many people with anorexia nervosa are not treated until their weight loss becomes life threatening. In fact, about 5 percent of people with anorexia nervosa die of starvation, suicide, or medical complications stemming from their extreme weight loss (Steinhausen, 2002).

Isabelle Caro was a French model and actress who allowed her emaciated image to be used in an Italian advertising campaign against anorexia. She died on November 17, 2010, presumably due to complications associated with her eating disorder.

DISTURBANCE IN EVALUATING WEIGHT OR SHAPE

A second defining symptom of anorexia nervosa can involve one of several related symptoms about weight and shape. Like Serrita, many individuals steadfastly *deny problems with their weight.* Even when confronted with their own withered image in a mirror, some people with anorexia nervosa insist that their weight is not a problem.

Other people with the disorder suffer from *a disturbance in the way body weight or shape is experienced.* Sometimes this may include a **distorted body image,** an inaccurate perception of body size and shape. One early study found that young women with anorexia nervosa overestimated the size of various body parts in comparison to a normal control group (Slade & Russell, 1973). The following excerpt, which we received anonymously from a student, illustrates the symptom.

> I'll try to explain how a person with an eating disorder sees a distorted image of herself. It's almost like she sees herself as bloated–of course she sees herself, she recognizes herself, but as bigger than usual. Also, the skinnier she gets, the more she notices fat deposits around the waist, under the arms, etc., because the more fat is lost, the more attention is drawn to the little bit of fat that still exists. Also, it's the point of reference in the background that may sometimes be distorted. She looks in the bathroom mirror and thinks, "Did I take up that much space against this wall yesterday?"

Not all people with anorexia nervosa suffer from a distorted body image, and many people without the disorder inaccurately estimate the size of their body (Garfinkel, Kennedy, & Kaplan, 1995; Thompson, 1996). These people simply are *unduly influenced by their body weight or shape in self-evaluation.* Whatever its specific form, a defining characteristic of anorexia nervosa is a disturbance in the way one's body or weight is

MyPsychLab VIDEO CASE

ANOREXIA NERVOSA

NATASHA

"I was never skin and bones. Never. I wanted to be. Had I not gone inpatient, I would have been."

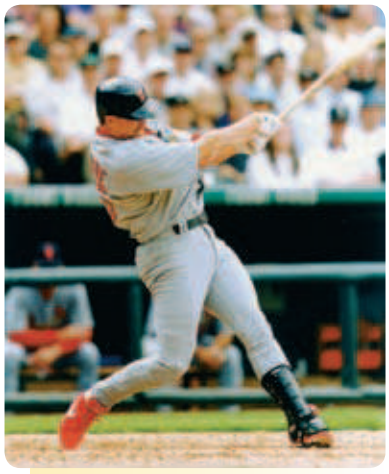
Watch the video "Anorexia: Natasha" on MyPsychLab. As you watch Natasha, listen how her perfectionism and control issues focus on her weight and appearance.

[1]To calculate the body mass index: (1) Multiply weight in pounds by 700; (2) divide this number by height in inches; (3) divide this second number by height in inches. You can interpret the resulting number as follows: Under 16 = extremely underweight; 16–18 = significantly underweight; 20–25 = healthy weight; 27–30 = overweight; 30–40 = significantly overweight; over 40 = extremely overweight.

EATING DISORDERS IN MALES

Our culture clearly values different body types for males than it does for females. Adolescent boys often want to be bigger and stronger, not slimmer. Women rate themselves as being thin only when they are below 90 percent of their expected body weight. In contrast, men see themselves as thin even when they weigh as much as 105 percent of their expected weight (Anderson, 2002). Surveys indicate that the majority of females want to lose weight, but males are about equally divided between those who want to lose weight and those who want to gain weight.

In fact, some experts argue that pressures to be strong and muscular have created a new eating disorder among males. The problem, sometimes called "reverse anorexia" or the "Adonis complex," is characterized by excessive emphasis on extreme muscularity and often accompanied by the abuse of anabolic steroids (Anderson, 2002; Ricciardelli & McCabe, 2004). You may recall that baseball slugger Mark McGwire was taking androstenedione ("andro," an over-the-counter steroid hormone) when he broke the record for most home runs in a single season. McGwire's popularity and success apparently contributed to growing steroid use among young males in the United States, where 3 percent to 12 percent of teenage boys have tried steroids (Ricciardelli & McCabe, 2004).



Mark McGwire took androstenedione ("andro"), an over-the-counter steroid hormone, when he broke the single season home run record. Many teenage boys take steroids to build their bodies, a trend some consider to be a new eating disorder.

More realistic expectations about thinness surely contribute to the lower prevalence of anorexia and bulimia among males. However, men with these eating disorders are less likely to seek treatment than are women, perhaps because they are less likely to recognize the problem or feel more stigmatized because of it (Woodside et al., 2001). Men with anorexia or bulimia deviate far from male norms, and this can lead to rejection and stigmatization by other men, therapists, and even females with eating disorders. The stigma of being a man with an eating disorder also alters one common symptom of anorexia nervosa. Females with anorexia nervosa typically view their appearance positively, perhaps even with a degree of pride. In contrast, anorexia nervosa can negatively affect the self-esteem of men, because weight and/or eating struggles are "unmanly," that is, different from the cultural image of the ideal male (Andersen, 1995).

Anorexia and bulimia are more common among certain subgroups of males. Male wrestlers have a particularly high prevalence of bulimia, a result of the intense pressure to "make weight"—to weigh below the weight cutoffs used to group competitors in a wrestling match. Eating disorders also are more common among gay men, who place more emphasis on appearance (Carlat, Camargo, & Herzog, 1997; Russell & Keel, 2002) and also endure minority stress (Kimmel & Mahalik, 2005).

How are men with eating disorders different from women with eating disorders?

Other aspects of eating disorders are similar for males and females. For both men and women, the disorders typically begin during adolescence or young adulthood. Other than the exceptions we have noted, similar factors also predict a risk for developing eating disorders in both females and males, including "reverse anorexia" (Ricciardelli & McCabe, 2004). Whether the ideal image is unrealistically thin or unrealistically muscular, cultural stereotypes about appearance can be risky for both males and females who internalize them.

Shelves stocked with body building supplements. Legal and illegal supplements are increasingly used by boys and young men to "man up" their bodies.

perceived or evaluated. People with anorexia nervosa do not recognize their emaciation for what it is.

FEAR OF GAINING WEIGHT

An *intense fear of becoming fat* is a third defining characteristic of anorexia. The fear of gaining weight presents particular problems for treatment. A therapist's encouragement to eat more can terrify someone who fears that relaxing control, even just a little, will lead to a total loss of control. Ironically, the fear of gaining weight is not soothed by the tremendous weight loss. In fact, the fear may grow more intense as the individual loses more weight (APA, 2000).

CESSATION OF MENSTRUATION

Amenorrhea, the absence of at least three consecutive menstrual cycles, is the final DSM-IV-TR symptom of anorexia nervosa (in females). The presence of amenorrhea has led to speculation about the role of sexuality and sexual maturation in causing anorexia nervosa. However, amenorrhea typically is a *reaction* to the loss of body fat and associated physiological changes, not a symptom that precedes anorexia (Pinheiro et al., 2007). Sexual disinterest also is a common reaction to severe weight loss (Keys et al., 1950). Furthermore, the presence or absence of menstruation does not differentiate between women who meet other diagnostic criteria for anorexia, and menstrual irregularities are common in bulimia (Attia & Roberto, 2009; Pinheiro et al., 2007; Wilfley et al., 2008). For these reasons, revisions of the DSM are very likely to drop amenorrhea as a diagnostic criterion for anorexia.

MEDICAL COMPLICATIONS

Anorexia nervosa can cause a number of medical complications. People with anorexia commonly complain about constipation, abdominal pain, intolerance to cold, and lethargy. Some of these complaints stem from the effects of semistarvation on blood pressure and body temperature, both of which may fall below normal. In addition, the skin can become dry and cracked, and some people develop *lanugo,* a fine, downy hair, on their face or trunk of their body. Broader medical difficulties may include anemia, infertility, impaired kidney functioning, cardiovascular difficulties, dental erosion, and osteopenia (bone loss) (Mitchell & Crow, 2010). A particularly dangerous medication complication is an *electrolyte imbalance,* a disturbance in the levels of potassium, sodium, calcium, and other vital elements found in bodily fluids. Electrolyte imbalance can lead to cardiac arrest or kidney failure. Anorexia nervosa may begin with the seemingly harmless desire to be a bit thinner, but the eating disorder can lead to serious health problems, including death.

STRUGGLE FOR CONTROL

Some people with anorexia act impulsively, but clinical accounts and some research suggest that more are conforming and controlling. Some theorists speculate that the disorder actually develops out of a desperate sense of having no control. Excessively compliant "good girls" may find that obsessively regulating their diet allows them to be in charge of at least one area of their lives (Bruch, 1982). Certainly, many young people with anorexia nervosa take great pride in their self-denial, feeling like masters of control.

How do people with anorexia nervosa evaluate their weight and shape?

COMORBID PSYCHOLOGICAL DISORDERS

Anorexia nervosa is associated with other psychological problems, including obsessive–compulsive disorder and obsessive–compulsive personality disorder (Halmi, 2010). People with anorexia nervosa are obsessed with food and diet, and they often follow compulsive eating rituals. However, a unique study found that such behavior can result from starvation. In this study, 32 World War II conscientious objectors fulfilled their military obligation by voluntarily undergoing semistarvation for 24 weeks. (The researchers wanted to learn about the effects of starvation on military personnel in the field.) As the men lost more and more weight, they developed extensive obsessions about food and compulsive eating rituals. For many, the obsessions and compulsions continued long after they returned to their normal weight (Keys et al., 1950). This suggests that obsessive–compulsive behavior may be a reaction to starvation, not a risk factor for anorexia.

Most people with anorexia nervosa also show symptoms of depression, such as sad mood, irritability, insomnia, social withdrawal, and diminished interest in sex (Halmi, 2010). Like obsessive–compulsive behavior, however, depression is a common reaction to starvation. Mood disturbances sometimes play a role in the development of anorexia nervosa, but depression can also be a reaction to the eating disorder (Vögele & Gibson, 2010).

Conscientious objectors participating in a study of semistarvation during World War II. Many starving men developed obsessions about food similar to those sometimes found in anorexia nervosa.

Finally, anorexia often co-occurs with the symptoms of bulimia. In some cases, purging follows episodes of binge eating. In other cases, purging may be a means of further controlling eating that already is dramatically restricted. People with anorexia nervosa who do *not* binge eat or purge generally are better adjusted on measures of their mental health—for example, they have lower rates of depression (Braun, Sunday, & Halmi, 1994).

Symptoms of Bulimia

Bulimia nervosa and anorexia nervosa, although different, share similarities. One connection is that many people with bulimia nervosa have a history of anorexia nervosa, as in the following case study.

CASE STUDY Michelle's Secret

Michelle was a sophomore at a state university when she first sought help for a humiliating problem. Several times a week, she fell into an episode of uncontrollable binge eating followed by self-induced vomiting. Michelle had enough control to limit her binge eating to times when her roommate was away. But when Michelle was alone and feeling bad, she would buy a half-gallon of ice cream and perhaps a bag of cookies and bring the food back to her room, where she secretly gorged herself. The binge brought Michelle some comfort at first, but by the time she was finished, she felt physically uncomfortable, sickened by her lack of control, and terrified of gaining weight. To compensate, she would walk across the street to an empty bathroom in the psychology department. There, she forced herself to vomit by sticking her finger down her throat.

The vomiting brought relief from the physical discomfort, but it did not relieve her shame. Michelle was disgusted by her actions, but she could not stop herself. In fact, the pattern of binge eating and purging had been going on for most of the school year. Michelle decided to seek treatment only when a friend from her psychology class discovered her purging in the bathroom. The friend also had a history of bulimia nervosa, but she had gotten her eating under control. She convinced Michelle to try therapy.

Michelle's eating problems began when she was in high school. She had studied ballet since she was 8 years old, and with the stern encouragement of her instructor, she had struggled to maintain her willowy figure as she became an adolescent. At first she dieted openly, but her parents constantly criticized her inadequate eating. In order to appease them, Michelle would eat a more normal meal but force herself to vomit shortly afterwards. When she was a junior in high school, Michelle's parents confronted her and took her to a psychologist, who treated her for anorexia nervosa. She was 5 feet 6 inches tall at the time, but she weighed only 95 pounds. Michelle was furious and refused to talk in any depth with the therapist. She allowed herself to gain a few pounds—to about 105—only to convince her parents that she did not need treatment.

Michelle's weight eventually stabilized between 105 and 110 pounds. Even though she was very thin, Michelle continued to plan her diet with great care. She avoided fat with a vengeance. She counted every calorie at every meal every day. Throughout college, she starved herself all week so she could eat normally on dates during the weekend. Occasionally, she forced herself to vomit after eating too much, but she did not see this as a big problem. Until the previous summer, she had maintained her weight near her goal of 105 pounds. Over the summer, however, Michelle relaxed her diet as she "partied" with old friends. She gained about 15 pounds, a healthy but still quite thin weight for her height and body type. When she returned to college, however, Michelle grew disgusted with her appearance and fearful of gaining even more weight.

Michelle tried to lose weight, but she met with little success. She started to purge more frequently in a desperate attempt to "diet," but she soon found herself binge eating more frequently, too. Michelle was extremely frustrated by her "lack of self-control." Although she now recognized her past problems with anorexia nervosa, Michelle openly longed for the discipline she had once achieved over her hunger and diet.

By all outward appearances, Michelle was a bright, attractive, and successful young woman. Inwardly, she felt like a failure and a "fake." She longed to have a boyfriend but never found one despite many casual dates. She was intensely, if privately, competitive with her girlfriends. She wanted to be more beautiful and intelligent than other girls, but she inevitably felt inferior to one classmate or another. She was determined at least to be thinner than her girlfriends, but she felt that she had lost all control over this goal. Michelle pretended to be happy and normal, but inside she felt as though she was going to explode. Secretly, she was miserable.

BINGE EATING

Michelle's frequent struggles with binge eating and purging, her sense of lost control during a binge, and her undue focus on her weight and figure are the core symptoms that define bulimia nervosa. Depression also is commonly associated with the disorder, as it was for Michelle.

Binge eating is defined as eating an amount of food that is clearly larger than most people would eat under similar circumstances in a fixed period of time, for example, less than two hours. There have been some attempts to define a binge more objectively, such as eating more than 1,000 calories, or subjectively, such as based on the individual's appraisal. Variations in normal eating complicate these alternative definitions, however. Eating a very large number of calories may be normal under certain circumstances (think: Thanksgiving) and having two cookies may be considered a "binge" by other people. Thus, the present DSM-IV-TR definition relies on a clinician's judgment about normal eating patterns.

Sadly, many inappropriate eating behaviors border on being statistically normal—and clearly unhealthy—in our food- and weight-obsessed society. Over 35 percent of people report occasional binge eating. Distressingly large numbers of people also report that they fast (29 percent) and use self-induced vomiting

Lady Gaga, who has struggled with bulimia, is one of many celebrities who have publicly acknowledged battling with an eating disorder.

(8 percent) or laxatives (over 5 percent) in an attempt to compensate for their eating (Fairburn & Beglin, 1990).

Binges may be planned in advance, or they may begin spontaneously. In either case, binges typically are secret. Most people with bulimia nervosa are ashamed and go to elaborate efforts to conceal their binge eating. During a binge, the individual typically eats very rapidly and soon feels uncomfortably full. Although the types of foods can vary widely, the person often selects ice cream, cookies, or other foods that are high in calories. Foods also may be selected for smooth texture to make vomiting easier, one reason why ice cream is a popular binge food.

Binge eating is commonly triggered by an unhappy mood, which may begin with an interpersonal conflict, self-criticism about weight or appearance, or intense hunger following a period of fasting. The binge initially is comforting and alleviates some unhappy feelings, but physical discomfort and fear of gaining weight soon override the positive aspects of binge eating.

A key feature of binge eating is a sense of lack of control during a binge. Some individuals experience a binge as a "feeding frenzy," where they lose all control and eat compulsively and rapidly. Others describe a dissociative experience, as if they were watching themselves gorge. But the lack of control is not absolute. For example, people with bulimia can stop a binge if they are interrupted unexpectedly. In fact, as the disorder progresses, some people feel more in control during a binge but unable to stop the broader cycle of binge eating and compensatory behavior.

INAPPROPRIATE COMPENSATORY BEHAVIOR

Almost all people with bulimia nervosa engage in **purging,** designed to eliminate consumed food from the body. The most common form of purging is self-induced vomiting; as many as 90 percent of people with bulimia nervosa engage in this behavior (APA, 2000). Other less common forms of purging include the misuse of laxatives, diuretics (which increase the frequency of urination), and, most rarely, enemas. Ironically, purging has only limited effectiveness in reducing caloric intake. Vomiting prevents the absorption of only about half the calories consumed during a binge, and laxatives, diuretics, and enemas have few lasting effects on calories or weight (Kaye et al., 1993).

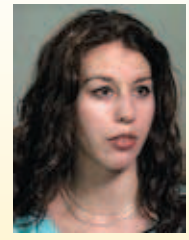
Inappropriate compensatory behaviors other than purging include extreme exercise or rigid fasting following a binge. The extent to which these actions actually compensate for binge eating also is questionable, given what we know about the body's biological regulation of weight (Brownell & Fairburn, 1995).

EXCESSIVE EMPHASIS ON WEIGHT AND SHAPE

People with bulimia nervosa place *excessive emphasis on body shape and weight* in evaluating themselves, a symptom shared with anorexia nervosa (see Table 10.1). Their self-esteem, and much of their daily routine, centers around weight and diet. Some people with bulimia nervosa are exhilarated by positive comments or interest in their appearance, but their esteem plummets if a negative comment is made or if someone else draws more attention. Other people with the disorder constantly criticize their appearance, and the struggle with binge eating and purging only adds to their self-denigration. In either case, the individual's sense of self is linked too closely to appearance instead of personality, relationships, or achievements.

MyPsychLab VIDEO CASE

BULIMIA NERVOSA



JESSICA

"It started out with a diet for me . . ."

Watch the video "Bulimia Nervosa: Jessica" on MyPsychLab. As you watch the video, listen for Jessica's struggles with appearance and weight and how they were magnified—sometimes very directly—by her efforts to pursue a career in the performing arts.

COMORBID PSYCHOLOGICAL DISORDERS

Depression is common among individuals with bulimia nervosa, especially those who self-induce vomiting (APA, 2000).

TABLE 10.1 Anorexia Nervosa and Bulimia Nervosa:Key Differences and Similarities

Issue	Anorexia Nervosa	Bulimia Nervosa
	Differences	
Eating/weight	Extreme diet; below minimally normal weight	Binge eating/compensatory behavior; normal weight
View of disorder	Denial of anorexia; proud of "diet"	Aware of problem; secretive/ashamed of bulimia
Feelings of control	Comforted by rigid self-control	Distressed by lack of control
	Similarities	
Self-evaluation	Unduly influenced by body weight/shape	Unduly influenced by body weight/shape
Comorbidity of AN/BN	Some cases of AN also binge and purge	Many cases of BN have history of AN
SES, age, gender	Prevalent among high SES, young, female	Prevalent among high SES, young, female

Some individuals become depressed prior to developing the eating disorder, and the bulimia may be a reaction to the depression in some of these cases. In many instances, however, depression begins at the same time as or follows the onset of bulimia nervosa (Braun et al., 1994). In such circumstances, the depression is likely to be a reaction to the bulimia. In fact, depression often lifts following successful treatment of bulimia nervosa (Mitchell et al., 1990). Whether depression is an effect or cause of bulimia, eating disturbances are more severe and social impairment is greater when the two problems are comorbid (Stice & Fairburn, 2003).

What are some similarities and differences between anorexia and bulimia?

Other disorders that may co-occur with bulimia nervosa include anxiety disorders, personality disorders (particularly borderline personality disorder), and substance abuse, particularly excessive use of alcohol and/or stimulants. Although each of these psychological difficulties presents special challenges in treating bulimia, the comorbidity with depression is most common and most significant (Halmi, 2010).

MEDICAL COMPLICATIONS

A number of medical complications can result from bulimia nervosa. Repeated vomiting can erode dental enamel, particularly on the front teeth, and in severe cases teeth can become chipped and ragged looking. Repeated vomiting can also produce a gag reflex that is triggered too easily and perhaps unintentionally. One consequence of the sensitized gag reflex—one that is rarely reported in the scientific literature—is *rumination:* the regurgitation and rechewing of food (Parry-Jones, 1994). Another possible medical complication is the enlargement of the salivary glands, a consequence that has the ironic effect of making the sufferer's face appear puffy. As in anorexia nervosa, potentially serious medical complications can result from electrolyte imbalances. Finally, rupture of the esophagus or stomach has been reported in rare cases, sometimes leading to death (Mitchell & Crow, 2010).

Diagnosis of Eating Disorders

BRIEF HISTORICAL PERSPECTIVE

Isolated cases of eating disorders have been reported throughout history. The term *anorexia nervosa* was coined in 1874 by a British physician, Sir William Withey Gull (1816–1890). Still, the history of professional concern with the disorders is very brief. References to eating disorders were rare in the literature prior to 1960, and the disorders have received scientific attention only in recent decades (Fairburn & Brownell, 2002; Striegel-Moore & Smolak, 2001). The term *bulimia nervosa* was used for the first time only in 1979 (Russell, 1979).

The diagnoses of anorexia nervosa and bulimia nervosa first appeared together in DSM in 1980 (DSM-III). Although the diagnostic criteria have changed somewhat, the same eating behaviors remain as the central features of these disorders. The only major change was the creation of a separate diagnostic category for eating disorders. They previously had been listed as a subtype of the Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence, because many eating disorders begin during the teenage years. The new, separate grouping reflects the fact that eating disorders also can begin during adult life.

TABLE 10.2 DSM-IV-TR Diagnostic Criteria for Anorexia Nervosa

A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g.,weight loss leading to maintenance of body weight less than 85 percent of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85 percent of that expected).

B. Intense fear of gaining weight or becoming fat, even though underweight.

C. Disturbance in the way in which one's weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.

D. In postmenarcheal females, amenorrhea, that is, the absence of at least three consecutive menstrual cycles.

SPECIFY TYPE

Restricting type: During the current episode, the person has not regularly engaged in binge eating or purging behavior.

Binge-eating/purging type: During the current episode, the person has regularly engaged in binge eating or purging behavior.

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

CONTEMPORARY CLASSIFICATION

Anorexia Nervosa DSM-IV-TR lists two types of eating disorders: anorexia nervosa and bulimia nervosa. Anorexia nervosa is defined by four symptoms described earlier (see Table 10.2). DSM-IV-TR also includes two subtypes of anorexia nervosa. The *restricting type* includes people who rarely engage in binge eating or purging. In contrast, the *binge eating/purging type* is defined by regular binge eating and purging during the course of the disorder. The validity of the subtypes is questionable, however, as they do not differ in terms of comorbidity, recovery, relapse, or mortality (Wonderlich et al., 2007). Moreover, an eight-year longitudinal study found that 62 percent of the former restrictors met diagnostic criteria for binge eating/purging, and only 12 percent of the restrictors had never regularly engaged in binge eating or purging (Eddy et al., 2002).

Bulimia Nervosa Bulimia nervosa is defined by five symptoms described earlier (see Table 10.3) and also is divided into two subtypes in DSM-IV-TR. The *purging type* regularly uses self-induced vomiting, laxatives, diuretics, or enemas. The *nonpurging type* instead attempts to compensate for binge eating only with fasting or excessive exercise. Like anorexia subtypes,

TABLE 10.3 DSM-IV-TR Diagnostic Criteria for Bulimia Nervosa

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.

C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months.

D. Self-evaluation is unduly influenced by body shape and weight.

E. The disturbance does not occur exclusively during episodes of anorexia nervosa.

Specify Type

Purging type: During the current episode, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.

Nonpurging type: During the current episode, the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.

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

Actress Jessica Biel was *Esquire* magazine's "sexiest woman alive." She is athletic, shapely, and fit, but not abnormally thin.

however, research does not support these bulimia subtypes, and experts have recommended dropping them (Wilfley et al., 2008).

Binge Eating Disorder and Obesity There is debate about whether other eating problems should be considered eating disorders. **Binge eating disorder,** episodes of binge eating but without compensatory behavior, is one possible new diagnosis. Accumulating evidence supports binge eating disorder as a reliable diagnosis distinct from other eating disorders in terms of demographics, correlates, and treatment (Allison & Lundgren, 2010). "Emotional eating," binge eating in response to stress and negative emotion, is a particular problem (Vögele & Gibson, 2010).

Binge eating is associated with a number of psychological and physical difficulties (Hudson et al., 2007). Among these problems is **obesity,** excess body fat corresponding with being 20 percent *above* expected weight. A cutoff of 40 percent above normal is a rough marker of being severely overweight (Brownell, 1995). Calling obesity a "mental disorder" is controversial, especially given its high prevalence in the United States and throughout the world. In fact, some professionals question our society's constant focus on dieting and our castigation of obese people. Obesity is not just a lack of "willpower," as biological factors contribute substantially to body shape and weight (Brownell & Rodin, 1994).

Frequency of Eating Disorders

Estimates of the epidemiology of anorexia and bulimia vary, but both disorders appear to have increased in frequency since the 1960s and 1970s. Figure 10.2 illustrates the surge in new cases of anorexia nervosa based on a compilation of evidence from Northern Europe (Hoek & van Hoeken, 2003). According to this summary, the annual *incidence,* the number of new cases each year, of anorexia nervosa rose from 1 case per million people in 1930–1940 to 54 cases per million people in 1995–1996. Figure 10.2 also shows that anorexia nervosa is rare in the general population with the annual incidence stabilizing in recent decades. Anorexia is far more common among certain groups, however, particularly young women, where the incidence still may be increasing (Keel, 2010). DSM-IV-TR indicates that lifetime prevalence of anorexia nervosa is 0.5 among females, similar to a 0.9 estimate based on a recent U.S. national survey (Hudson et al., 2007). Anorexia nervosa also occurs among males, but most estimates indicate the disorder is about 10 times more common among women than men. Establishing the exact prevalence in males is difficult, because only a few cases are identified even in large national surveys (Hudson et al., 2007).

Recent decades also have witnessed a torrent of new cases of bulimia nervosa. Changes in the frequency of bulimia nervosa are difficult to document, however, because the diagnostic term was introduced only in 1979. Instead, investigators have examined cohort effects in prevalence rates. A **cohort** is a group that shares some feature in common, for example, year of birth; thus, **cohort effects** are differences that distinguish one cohort from another.

Figure 10.3 portrays birth cohort effects in lifetime prevalence rates of bulimia nervosa among a large sample of American women who were born either before 1950, between 1950 and 1959, or in 1960 or after. The figure clearly indicates substantial cohort effects. The lifetime prevalence of bulimia nervosa was far greater among the women born after 1960 than it was for those born before 1950. The risk for women born between 1950 and 1959 was intermediate between the two (Kendler et al., 1991). Figure 10.3 also shows that the risk of developing bulimia declines with increasing age, at least among older cohorts. A recent study of a national U.S. sample also found this declining risk with age and replicated the cohort effects (Hudson et al., 2007).

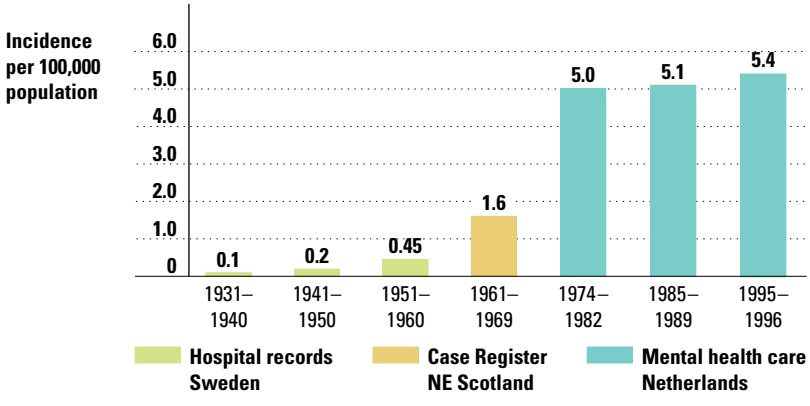


FIGURE 10.2 Annual Incidence of Anorexia Nervosa in Northern Europe from 1931 to 1996

Anorexia nervosa increased dramatically in the 1960s and 1970s, as the ideal media image of women grew thinner. Considerably higher rates are found among population subgroups, particularly young women.

Source: Figure, p. 209, "Incidence of Anorexia Nervosa, 1930–1980" from "The Distribution of Eating Disorders" by H. W. Hoek in *Eating Disorders and Obesity: A Comprehensive Handbook* ed. by K. D. Brownell and C. G. Fairbanks. Copyright © 2003 Wiley Periodicals, Inc.

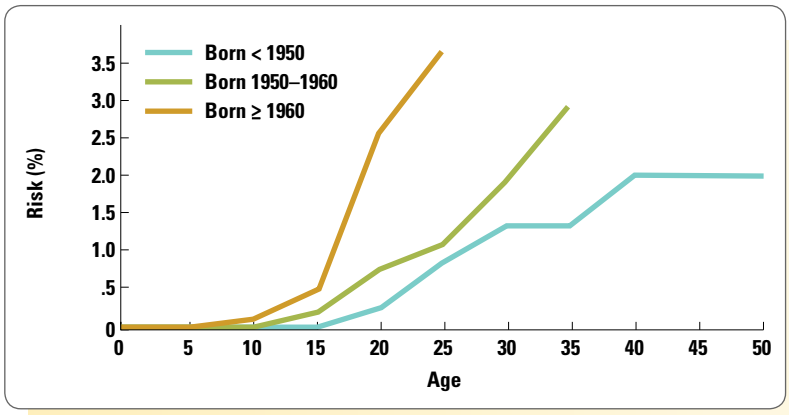


FIGURE 10.3

The lifetime cumulative risk for developing bulimia nervosa is far greater for women born after 1960 than for women born before 1950. The risk for developing the disorder decreases with age, at least among the earlier birth cohort. Later birth cohorts have not yet moved through the entire age of risk.

Source: Figure 1 from K. S. Kendler, C. MacLean, M. Neale, R. Kessler, A. Heath, and L. Eaves, "The Genetic Epidemiology of Bulimia Nervosa," *American Journal of Psychiatry,* Dec 1991; 148: 1627–1637. Reprinted with permission from the American Journal of Psychiatry, (Copyright © 1991). American Psychiatric Association.

Thus, the surge in bulimia nervosa—some say an epidemic—is due to dramatic increases among women born in more recent years. Not coincidentally, cultural standards of beauty changed for this generation of women.

Bulimia nervosa has a lifetime prevalence of 1.5 percent among U.S. women and .5 percent among U.S. men (Hudson et al., 2007). Some evidence suggests that bulimia may be decreasing somewhat, but evidence is variable, with declines perhaps linked to changing cultural standards of beauty (Keel, 2010). Binge eating disorder and occasional binge eating are even more common, with a respective lifetime prevalence of 3.5 percent and 4.9 percent among women and 2.0 percent and 4.0 percent among men (Hudson et al., 2007). Finally, we should note again the overlap between anorexia nervosa and bulimia nervosa. About 50 percent of all people with anorexia nervosa engage in episodes of binge eating and purging (Garfinkel et al., 1995), and many cases of bulimia nervosa have a history of anorexia nervosa (Wonderlich et al., 2007).

STANDARDS OF BEAUTY

Many scientists believe that this huge difference in the prevalence of eating disorders between men and women is explained by gender roles and standards of beauty (Field & Kitos, 2010). Popular attitudes about women in the United States tell us that "looks are everything," and thinness is essential to good looks. In contrast, young men are valued as much for their achievements as for their appearance, and the ideal body type for men is considerably larger and more muscular than for women (see Critical Thinking Matters). In fact, women are much more likely than men to have a negative body image, and that disparity has been growing over time (Feingold & Mazzella, 1998).

The surge in eating disorders also might be explained by changing standards of beauty. *Playboy* centerfolds and Miss America Beauty Pageant contestants—cultural icons but dubious role models for young women—provide the statistics. Between 1959 and 1988, their ratio of weight to height declined dramatically. In fact, 69 percent of *Playboy* centerfolds and 60 percent of Miss America contestants weigh at least 15 percent below expected weight for their height (Garner et al., 1980; Wiseman et al., 1992). Marilyn Monroe, the movie idol of the 1950s, is chunky according to today's "culture of thinness."

Standards of beauty are relative, not absolute. Today, eating disorders are much more common in North America, Western Europe, and industrialized Asian countries; bulimia may be completely culture bound (Keel & Klump, 2003). In other cultures, women who are more rounded are considered to be more beautiful. In Third World countries, where food is scarce, wealth is positively correlated with body weight. Being larger is a symbol of beauty and success. In industrialized nations, where food is plentiful, wealth is negatively correlated with weight. As the saying goes, "You can never be too rich or too thin."

Model Kate Moss. Contemporary images of women place a premium on slimness and suggest that women should be judged by their appearance. Both of these messages apparently contribute to the development of eating disorders.

Critical Thinking Matters

THE PRESSURE TO BE THIN

Critical thinking matters in the classroom—and in everyday life. Consider how young women (and men) embrace standards of beauty based on images in the media, and, in turn, apply fashion model standards to themselves and others.

The images are everywhere. Super-thin models routinely grace the cover and inside of fashion magazines. The average fashion model is 5'11" and 117 pounds; the average woman in the United States is 5'4" and 140 pounds. Television and movie actresses are not only talented and beautiful, but also exceptionally thin. Cameron Diaz and Julia Roberts, for example, both meet the body mass index requirement for anorexia nervosa (Owen & Laurel-Seller, 2000). Advertisements in all kinds of media for all kinds of products use images of sexy, beautiful, and very thin women, images that often are altered not only to erase blemishes, but also to lengthen legs or otherwise distort body shape. Even girls' dolls are unrealistically thin and beautiful. The ubiquitous Barbie Doll has a shape that translates into a 39–18–33 figure in human equivalents. (In 1994, Dolly Parton claimed that she measured 40–20–36.) And real-life GI Joe would have a 55" chest and a 27" bicep. (At his bodybuilding peak, Arnold Schwarzenegger had a 57" chest and 22" biceps.)

In 1994, Dolly Parton claimed that she measured 40-20-36.

The pressure to be thin greatly affects women who make their careers as actresses, models, and singers. The Brazilian model Ana Carolina Reston died of complications due to anorexia in 2006. (In the same year, the Spanish government banned too-thin models from a popular fashion show.) The dubious "who's who" of women in the popular media who have *publicly admitted* to having an eating disorder (often in an attempt to encourage healthier body images among the girls and women who admire them) includes actresses Mary-Kate Olsen, Courtney Thorne-Smith, and Kate Winslet; fashion model Carre Otis; singers Lady Gaga, Ashlee Simpson, Paula Abdul, and Victoria Beckham (Posh Spice of the Spice Girls); and Oprah Winfrey.

Psychological studies repeatedly show that exposure to images of super-thin women increases body image dissatisfaction among girls and young women (Halliwell & Dittmar, 2004). Yet, young women face contradictory messages when parents or others tell them, "Beauty is only skin deep." For one, both extensive psychological research and everyday experience repeatedly tell us that attractiveness *does* matter, not only to romantic attraction (although men prefer a more curvaceous figure than women *think* they like), but also appearance matters in the evaluations of same-gender peers, teachers, employers, and on and on. And even as public health advocates battle the culture of thinness, other public health officials tell young people—rightly—to be careful about what they eat, to not eat too much, and to try to lose weight. In fact, obesity is a much more prevalent public health problem than eating disorders. In the United States, one out of every two adults and 20 percent of children are overweight, and obesity is associated with a great many health risks (Heinberg, Thompson, & Stormer, 2001).

Why are media images of women's bodies unrealistic?

So where does critical thinking come in? Critical thinking is all about being reasonable and thinking for yourself. Appearance does matter, but few of us, female or male, can hope to look like models or movie stars. After all, these professionals literally are one in a million (or a billion). The stars of popular media devote much of their life to their appearance, and they *still* need the help of makeup, camera angles, creative fashions, and various electronic "corrections." Health matters, too, and exercise is a great way to promote health, maintain an attractive physical appearance, and remember that your body is good for something other than being looked at. And most of us are more impressed by what someone does, by who they are, than what they look like. Remembering this is a useful reminder when we start to think critically—instead of using critical thinking—in evaluating our own weight and shape.

Marilyn Monroe, the 1950s movie idol, was the iconic image of the curvy figure that defined beauty in that era.

AGE OF ONSET

Both anorexia and bulimia nervosa typically begin in late adolescence or early adulthood (Hudson et al., 2007). A significant minority of cases of anorexia nervosa begin during early adolescence, particularly as girls approach puberty. The adolescent onset of eating disorders has provoked much past speculation about their etiology, including hormonal changes (Garfinkel & Garner, 1982), autonomy struggles (Minuchin, Rosman, & Baker, 1978), and various sexual problems (Coovert, Kinder, & Thompson, 1989). A more simple explanation is the natural and normal changes in adolescent body shape and weight. Weight gain is normal during adolescence, but the addition of a few pounds can trouble a young woman focused on the numbers on her scale. Breast and hip development not only change body shape, but they also affect self-image, social interaction, and the fit of familiar clothes. Early pubertal timing is a risk factor for anorexia, supporting the importance of self-evaluation and social comparison as girls' shape develops normally in early adolescence (Jacobi & Fittig, 2010).

Weight and dieting become less of a concern, and disordered eating declines, as adolescent girls become women. Changes are particularly sharp following marriage and parenthood (Keel et al., 2007). Men, however, become *more* concerned with weight as they age. As men's metabolism slows with increasing age, losing weight becomes more of a worry than gaining weight.

Causes of Eating Disorders

The culture of thinness clearly contributes to the high rate of eating disorders today. However, other social, psychological, and biological risk factors must play a role, because not every young woman suffers from these problems.

SOCIAL FACTORS

Standards of beauty and the premium placed on young women's appearance contribute to causing eating disorders. This conclusion is supported by epidemiological evidence and other research documenting that

- Eating disorders are far more common among young women than young men (Hoek & van Hoeken, 2003). As Striegel-Moore and Bulik (2007) recently summarized, "The single best predictor of risk for developing an eating disorder is being female . . ." (p. 182).
- The prevalence of eating disorders has risen, as the image of the ideal woman has increasingly emphasized extreme thinness (Hoek & van Hoeken, 2003; Wiseman et al., 1992).
- Eating disorders are even more common among young women working in fields that emphasize weight and appearance, such as models, ballet dancers, and gymnasts (Bryne, 2002).
- Young women are particularly likely to develop eating disorders during adolescence and young adult life, an age during which our culture places a particular emphasis on appearance, beauty, and thinness (Hoek, 2002).
- Eating disturbances are more common among young women who report greater exposure to popular media, endorse more gender-role stereotypes, or internalize societal standards about appearance (Grabe, Ward, & Hyde, 2008).
- Eating disorders are more common among white women, who are more likely to equate thinness with beauty, versus African American women. Eating disorders also may be increasing among well-to-do African Americans, who increasingly hold the thinness ideal (Field & Kitos, 2010; Wildes, Emery, and Simons, 2001).
- Eating disorders are far more prevalent in industrialized societies, where thinness is the ideal, than in nonindustrialized societies, where a more rounded body type is preferred (Keel & Klump, 2003).
- The prevalence of eating disorders is higher among Arab and Asian women living or studying in Western countries than among women living in their native country (Hoek, 2002).

Crystal Renn has found success as a plus-size model. Her prominent fashion modeling is a positive sign for advertisers—and for Renn who formerly battled anorexia. Tony Cenicola/NYT/Redux.

These facts make it clear that adolescent girls and young women are at risk for developing eating disorders, in part because they attempt to shape themselves, quite literally, to fit the image of the ideally proportioned, thin woman. We should note, however, that the culture of thinness plays a stronger role in the development of bulimia than anorexia. Cases of anorexia nervosa are found in the historical literature, occur in non-Western cultures, and appear to have increased less than cases of bulimia nervosa in response to cultural ideals of thinness (Keel & Klump, 2003).

Of course, not every woman in the United States develops an eating disorder, so other factors must interact with culture to produce eating disorders (Striegel-Moore & Bulik, 2007). One basic influence is the individual's *internalization* of the ideal of thinness (Cafri et al., 2005). Same-gender peers can influence internalization (Field & Kitos, 2010), and so can popular media. In one study, ninth- and tenth-grade high school girls randomly received a free subscription to *Seventeen* magazine. One year later, those who received the magazine reported increased negative affect, but only if their body image was negative and they felt pressure to be thin when the study began (Stice, Spangler, & Agras, 2001). These girls apparently were more vulnerable to the media's "thin" message.

How does the culture of thinness contribute to eating disorders?

Twiggy became a popular fashion model in the 1960s. Beginning her career at the age of 16, Twiggy's stick figure helped to usher in the "culture of thinness."

Troubled Family Relationships Troubled family relationships may also increase vulnerability to the culture of thinness (Jacobi et al., 2004). Young people with bulimia nervosa report considerable conflict and rejection in their families, difficulties that also may contribute to their depression. In contrast, young people with anorexia generally perceive their families as cohesive and nonconflictual (Fornari et al., 1999; Vandereycken, 1995).

Although the families of young people with anorexia nervosa appear to be well functioning, some theorists see them as being too close—as *enmeshed families,* families whose members are overly involved in one another's lives. According to the enmeshment hypothesis, young people with anorexia nervosa are obsessed with controlling their eating, because eating is the *only* thing they can control in their intrusive families (Minuchin et al., 1978). However, "intrusive" parental concern is probably an effect, not a cause, of anorexia. Parents of an anorexic adolescent may well become "enmeshed" as a worried reaction to their daughter's emaciation.

Child sexual abuse also is a risk factor for eating disorders (Jacobi & Fittig, 2010). However, sexual abuse may not pose a specific risk. Women with eating disorders report experiencing child sexual abuse more often than normal controls but not more often than women suffering from other psychological problems (Palmer, 1995; Welch & Fairburn, 1996). Sexual abuse increases the risk for a variety of psychological problems, including, but not limited to, eating disorders.

Finally, we should note that there are many direct ways in which parents may influence children toward developing eating disorders. Many parents struggle with diet and thinness themselves, and they are models of preoccupation for their children; other parents directly encourage their children to be extra thin as a part of the general push to compete with their peers (Field & Kitos, 2010; Vandereycken, 2002).

PSYCHOLOGICAL FACTORS

Researchers have hypothesized about many psychological factors contributing to eating disorders. Here we highlight four of the most important: control issues, depression/dysphoria, body image dissatisfaction, and reactions to dietary restraint.

A Struggle for Perfection and Control One of the first and most prolific clinical observers of eating disorders was Hilde Bruch (1904–1984), a physician who fled her native Germany in 1933 and subsequently studied psychiatry in the United States. Bruch viewed a struggle for control as the central psychological issue in the development of eating disorders (Bruch, 1982). Bruch observed that girls with eating disorders seem overly conforming and eager to please. She suggested that they give up too much of the normal adolescent struggle for autonomy. Bruch viewed obsessive efforts to control eating and weight as a way that these overly compliant "good girls" control themselves further. At the same time, Bruch also saw their dieting as an attempt to wrest at least a little control from their parents—control over what they eat. In this *struggle for control,* young people with anorexia nervosa (at least the restricting subtype) "succeed" and take pride in their extreme self-control. In contrast, those with bulimia nervosa continually strive—and fail—to control their eating and weight.

Perfectionism is another part of the endless pursuit of control. Perfectionists set unrealistically high standards, are self-critical, and demand a nearly flawless performance from

themselves. Research demonstrates that young women with eating disorders endorse perfectionist goals both about eating and weight and about general expectations for themselves (Bastiani et al., 1995; Jacobi & Fittig, 2010).

Young people with eating disorders may also try to control their own emotions excessively (Bruch, 1982). They may lack *interoceptive awareness*—recognition of internal cues, including hunger and various emotional states. One large study found that lack of introceptive awareness predicted the development of eating disorders 2 years in the future (Leon et al., 1993, 1995). People with eating disorders appear to be more tuned in to how they look than how they feel—sad, angry, happy, or hungry (Viken et al., 2002).

Depression, Low Self-Esteem, and Dysphoria Depression is often comorbid with eating disorders, particularly bulimia nervosa (Halmi, 2010). Antidepressant medications reduce some symptoms of bulimia nervosa, suggesting that, in some cases, bulimia is a reaction to depression (Mitchell, Raymond, & Specker, 1993). In other cases, depression may instead be a reaction to an eating disorder (Polivy & Herman, 2002). Depression improves markedly following successful group psychotherapy for bulimia (Mitchell et al., 1990). And a study of anorexia nervosa found considerable depression at the time of the original diagnosis but not at a six-year follow-up (Rastam, Gillberg, & Gillberg, 1995).

Depressive *symptoms,* and not necessarily clinical depression, also may play a role in eating disorders. Low self-esteem is a particular concern (Fairburn et al., 1997). In particular, women with eating disorders may be preoccupied with their *social self,* how they present themselves in public and how other people perceive and evaluate them (Striegel-Moore, Silberstein, & Rodin, 1993). Women with bulimia nervosa or a negative body image report more public self-consciousness, social anxiety, and perceived fraudulence (Striegel-Moore et al., 1993). They also show increases in self-criticism and deterioration in mood following negative social interactions (Vögele & Gibson, 2010). In short, people with eating disorders often depend on others for self-esteem.

How do "control issues" differ in anorexia and bulimia?

Depressive symptoms also can play a role in maintaining problematic eating behaviors. *Dysphoria* or negative mood states commonly trigger episodes of binge eating (Vögele & Gibson, 2010). The dysphoria may be brought on by social criticism or conflict, dissatisfaction with eating and diet, or an ongoing depressive episode. In summary, clinical depression can either be a cause or a reaction to eating disorders, while depressed moods, low self-esteem, and dysphoria may contribute to the onset or maintenance of symptoms.

Negative Body Image *A negative body image,* a highly critical evaluation of one's weight and shape, is widely thought to contribute to the development of eating disorders (Polivy & Herman, 2002). One way to assess a negative body image is to compare people's ratings of their "current" and "ideal" size by asking them to pick from the schematics in Figure 10.4. Several longitudinal studies have found negative evaluations of weight, shape, and appearance to predict the subsequent development of disordered eating (Jacobi & Fittig, 2010). A negative body image may be a particular problem when combined with other risk factors, including perfectionism and low self-esteem (Field & Kitos, 2010).

How does depression contribute to eating disorders?

Dietary Restraint Some symptoms of eating disorders may be effects of *dietary restraint,* that is, direct consequences of overly restrictive eating (Heatherton & Polivy, 1992). Ironically, many of the "out-of-control" symptoms of eating disorders are caused by inappropriate efforts to "control" eating! These symptoms include binge eating, preoccupation with food, and perhaps out-of-control feelings of hunger.

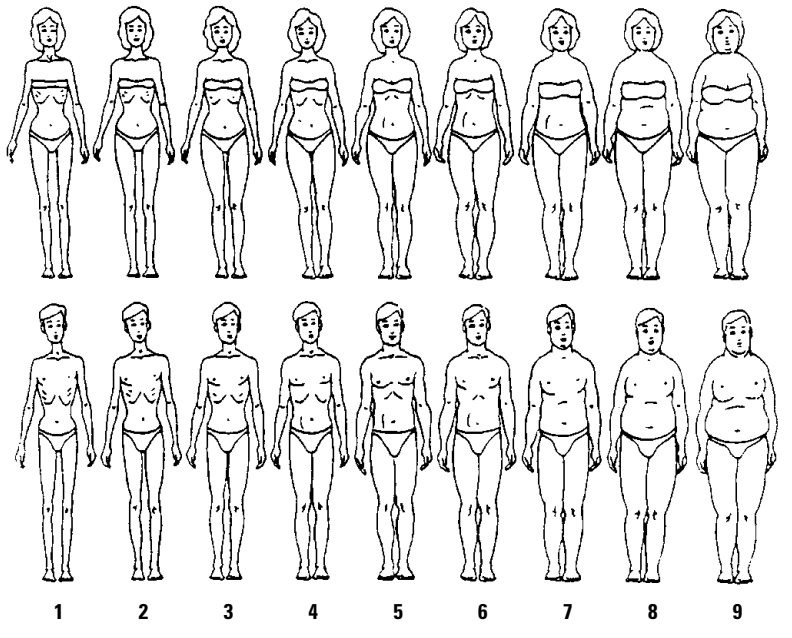


FIGURE 10.4

These figures are often used as one way to assess body image. People pick figures representing their current and ideal shape. The discrepancy between the ratings is a measure of negative body image.

Source: From p. 79 in "Assessing Body Image Disturbance: Measures, Methodology, and Implementation" by J. Kevin Thompson, in *Body Image, Eating Disorders and Obesity,* Ed. by J. K. Thompson. American Psychological Association, Washington, DC, Copyright © 1996, reprinted with permission.

Beyonce is a gorgeous example of a bigger but ideal body type.

Consistent with the dietary restraint hypothesis, adolescent girls who try to lose weight by fasting for 24 hours or more engage in more binge eating and develop bulimia more often five years later (Stice et al., 2008). Similarly, *weight suppression*—defined as highest adult weight minus current weight–predicts the maintenance and onset of bulimia 10 years later (Keel & Heatherton, 2010). An overly restrictive diet increases hunger, frustration, and lack of attention to internal cues, all of which make binge eating more likely. And "quick-fix" diets rarely work, leaving dieters with a sense of failure, disappointment, and self-criticism, negative emotions that lower self-esteem and lead to more binge eating.

Dietary restraint also may directly cause some of the symptoms of anorexia nervosa. The military studies of semistarvation conducted during World War II found that, during refeeding, many men felt intense, uncontrollable hunger, even after eating a considerable amount of food (Keys et al., 1950). Perhaps a similar reaction explains some of the intense fear of losing control and gaining weight found in anorexia nervosa.

While extreme restriction and quick-fix diets are unhealthy, sensible dieting is not. Normal weight women randomly assigned to a low-calorie diet lose weight and decrease binge eating 18 weeks later in comparison to women assigned to no diet (Presnell & Stice, 2003). As with so many things, finding a balanced middle ground is the key.

BIOLOGICAL FACTORS

Our bodies, in fact, seek a middle ground. Physiologically, weight is maintained around **weight set points,** fixed weights or small ranges of weight. Weight regulation around set points results from the interplay between behavior (e.g., exercise, eating), peripheral physiological activity (e.g., digestion, metabolism), and central physiological activity (e.g., neurotransmitter release; Blundell, 1995). The process is very much like the way a thermostat regulates heating and cooling to maintain air temperature at a given setting. Thus, if weight declines, hunger increases and food consumption goes up (Keesey, 1995). There is a slowing of the *metabolic rate,* the rate at which the body expends energy, and movement toward *hyperlipogenesis,* the storage of abnormally large amounts of fat in fat cells throughout the body (Brownell & Fairburn, 1995). All these reactions have obvious survival value and are likely products of evolution. The body does not distinguish between intentional attempts to lose weight and potential starvation.

Genetic factors also contribute to eating disorders. An early twin study of bulimia nervosa found a concordance rate of 23 percent for MZ twins and 9 percent for DZ twins (Kendler et al., 1991). Higher MZ than DZ concordance rates for anorexia nervosa (Bulik et al., 2006) and for dysfunctional eating attitudes have also been reported (Klump, McGue, & Iacono, 2000). Genetic factors also contributed substantially to various symptoms of disordered eating in the only adoption study completed to date (Klump et al., 2009).

Genetic contributions to eating disorders could be explained by several different mechanisms. Eating disorders are unlikely to be directly inherited. Rather, genes may influence personality characteristics such as anxiety that, in turn, increase the risk for an eating disorder (Klump & Culbert, 2007). Or a certain body type may be inherited. As most people assume intuitively, genetics contribute substantially to BMI (Wade, 2010). Inheriting a thin body type may increase the risk for anorexia—when combined with the culture of thinness, internalization of the standard, and perfectionism. Similarly, a more rounded body type may increase the risk for bulimia—when combined with social pressures to maintain a weight below one's natural set point.

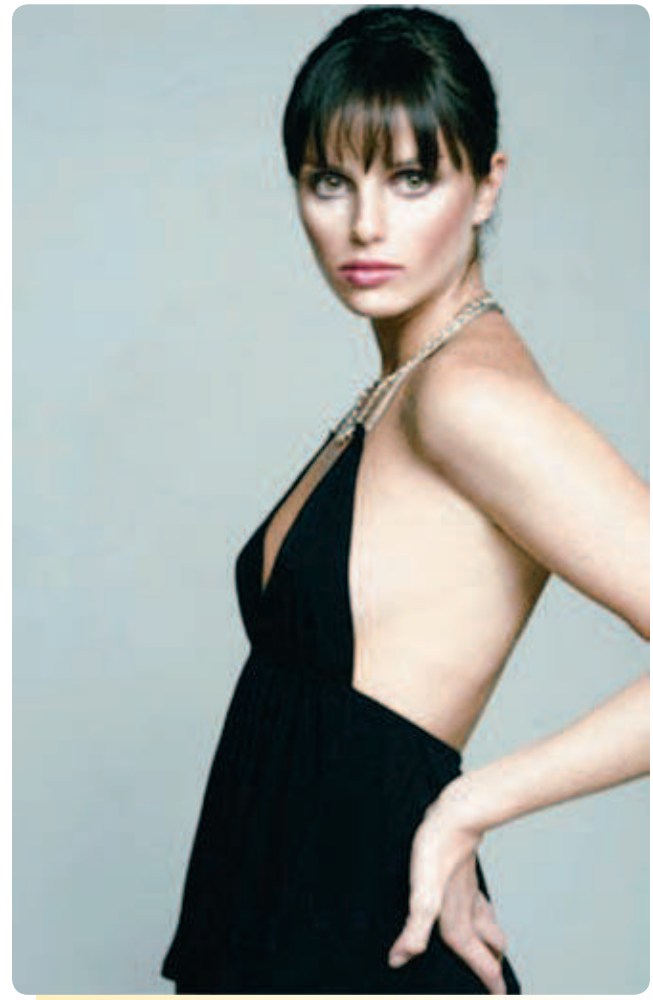
Consistent with these hypotheses, recent evidence shows that genes influence eating pathology *after* puberty but not before (Culbert et al., 2009). Genetic influences also are stronger among women who engage in dietary restraint (Racine et al., 2011). Genes clearly affect weight and body type, but we cannot mindlessly conclude that eating disorders are "genetic" without carefully considering genetic mechanisms and gene-environment interactions.

Several neurophysiological measures also are correlated with eating disorders, including elevations in endogenous opioids, low levels of serotonin, and diminished neuroendocrine functioning (Yates, 1990). Most of these differences in brain functioning, however, appear to be effects of eating disorders and not causes of them. In extremely rare cases, eating disorders have been linked with a specific biological abnormality, such as a hormonal disturbance or a lesion in the *hypothalamus,* the area of the brain that regulates routine biological functions, including appetite. But in most cases, the problem appears to be the result of an interaction of biological, psychological, and social risk factors.

INTEGRATION AND ALTERNATIVE PATHWAYS

Social and cultural values that emphasize thinness, beauty, and appearance over agency are the starting point in understanding eating disorders, particularly among young women. Risk factors that combine with cultural attitudes to produce eating disorders include direct familial and social pressures to be thin, a negative body image, dietary restraint, and genetic influences on body weight and shape (Jacobi et al., 2004; Stice, 2001, 2002). Less obvious risk factors include preoccupation with external evaluation, lack of introceptive awareness, and excessive conformity and self-control.

The etiology of eating disorders underscores the importance of *equifinality*—there are many pathways to developing an eating disorder (Halmi, 1997). Some women are naturally thin, but their perfectionism drives them to become even thinner. Other women may have a more rounded body type determined by genetics, and they struggle, and repeatedly fail, to mold their body into something it was never meant to be. For some people, an eating disorder is an expression of depression. Others may develop an eating disorder because they focus on outward appearances instead of internal values. Finding the middle ground of a healthy weight can be very difficult, particularly when the culture of thinness sets unrealistic standards of beauty, especially for young women.



Brazilian model Ana Carolina Reston died in 2006 as a result of complications due to anorexia nervosa. Bans on super-thin models have been considered in order to protect both the models and their fans.

Treatment of Anorexia Nervosa

The treatments for anorexia nervosa and bulimia nervosa differ in approach and effectiveness; therefore, we consider them separately. The treatment of anorexia nervosa usually focuses on two goals. The first is to help the patient gain at least a minimal amount of weight. If weight loss is severe, the patient may be treated in an inpatient setting. Hospitalized patients may receive forced or intravenous feeding, or participate in strict behavior therapy programs that make rewards contingent on weight gain. Hospitalization also may be needed to prevent suicide, to address severe depression or medical complications, or to remove the patient temporarily from a dysfunctional social circumstance (Garner & Needleman, 1996).

The second goal in treating anorexia nervosa is to address the broader eating difficulties. Many different treatments have been tried, but accumulating evidence indicates that family therapy is more effective than individual treatment, especially for children and adolescents (Lock et al., 2010; Le Grange & Hoste, 2010). The most carefully studied family therapy is the *Maudsley method* (named after Maudsley Hospital in London where the treatment was developed). In the Maudsley method, parents take complete control over the anorexic child's eating, planning meals, preparing food, and monitoring eating. Parents do not blame the adolescent for her problems, but emphasize the uncontrollable nature of anorexia and the importance of taking her "medicine"—food—in order to get better. Age-appropriate autonomy is returned to the teenager as eating and weight improve (Lock et al., 2010; Loeb et al., 2007). Growing evidence supports the effectiveness of the Maudsley method in treating anorexia and perhaps bulimia too (Le Grange & Hoste, 2010).

Many individual therapies also have been tried, including (1) Bruch's (1982) modified psychodynamic therapy designed to increase introceptive awareness and correct distorted perceptions of self; (2) *feminist therapies,* which encourage young women to pursue their own values rather than blindly adopting prescribed social roles (Fallon, Katzman, & Wooley, 1994); and (3) various cognitive behavioral approaches. Unfortunately, little evidence supports the effectiveness of any individual treatment (Wilson, 2010). Even worse, medication (antidepressants often are prescribed) and nutritional counseling not only offer little benefit, but patients also routinely drop out of these treatments (McElroy et al., 2010; Walsh et al., 2006; Wilson, Grilo, & Vitousek, 2007). Clearly, finding effective treatments for anorexia nervosa should be a research and public health priority.

COURSE AND OUTCOME OF ANOREXIA NERVOSA

Evidence on the course and outcome of anorexia nervosa further shows the limited effectiveness of contemporary treatments. At 10- to 20-year follow-up, nearly half of patients have a weight within the normal range, 20 percent remain significantly below their healthy body weight, and the remainder are intermediate in weight (Steinhausen, 2002). Perhaps 5 percent of patients starve themselves to death or die of related complications, including suicide.

Although important, weight gain is not the only measure of the course of anorexia nervosa. In fact, more than half the women with a history of anorexia nervosa continue to have difficulties with eating, notwithstanding gains in weight. Menstruation returns along with weight gain for most women, but many continue to be preoccupied with diet, weight, and body shape. Moreover, people may also develop new problems with social life, depression, or bulimia, as a result of their perfectionism, reliance on external evaluation, or continued struggles with body image (Keel, 2010). Predictors of a better prognosis include an early age of onset, conflict-free parent–child relationships, early treatment, less weight loss, and the absence of binge eating and purging (Steinhausen, 2002). The following account, written by a young woman after her long and, finally, successful struggle with anorexia nervosa, illustrates some of the continuing problems:

> I do not have a story that ends with a miraculous recovery, and I would be suspicious of anyone who claimed that they had completely gotten over an eating disorder. I continue to struggle with worries about food and my body. I exercise every day without fail. I am prone to stress fractures and will most likely encounter early osteoporosis due to the irreversible effects of starvation on my bones. I am lucky that I will be able to have children someday, though many long-term anorexics are never able to. Despite these lingering effects of the disorder, they pale in comparison to what I consider to be the most detrimental of all. When I look back on those six or so years, it sickens me to realize how much of life I missed. I allowed my obsession with my weight to take over my life (Zorn, 1998, p. 21).

Treatment of Bulimia Nervosa

Researchers have developed several approaches to treating bulimia nervosa. The most effective include cognitive behavior therapy, interpersonal psychotherapy, and antidepressant medication.

COGNITIVE BEHAVIOR THERAPY

The most thoroughly researched psychotherapy for bulimia nervosa is cognitive behavior therapy (Wilson et al., 2007). As developed by the British psychiatrist Christopher Fairburn, the cognitive behavioral approach views bulimia as stemming from several maladaptive tendencies, including an excessive emphasis on weight and shape; perfectionism; and dichotomous "black or white" thinking (Fairburn, 1996). Fairburn's cognitive behavioral treatment includes three stages. First, the therapist uses education and behavioral strategies to normalize eating patterns. The goal is to end the cycle where extreme dietary restraint leads to binge eating and, in turn, to purging. Second, the therapist addresses the client's broader, dysfunctional beliefs about self, appearance, and dieting. Techniques include a variation of Beck's cognitive therapy to address perfectionism or depression. Individual problems such as poor impulse control or troubled relationships also may be addressed at this stage. Third, the therapist attempts to consolidate gains and prepare the client for expected relapses in the future. Key goals at this final stage of treatment are to develop realistic expectations about eating, weight concerns, and binge eating, as well as clear strategies for coping with relapses in advance (Fairburn, 2002).

Overall, cognitive behavior therapy leads to a 70 percent to 80 percent reduction in binge eating and purging. Between one-third and one-half of all clients are able to cease the bulimic pattern completely, and the majority of individuals maintain these gains at six-month to one-year follow-up (Agras et al., 2000; Fairburn et al., 1993). Cognitive behavior therapy also may be effective in group (Mitchell et al., 1990) and self-help formats (Carter & Fairburn, 1998), although individual therapy is more effective (Thompson-Brenner, Glass, & Westen, 2003).

INTERPERSONAL PSYCHOTHERAPY

Interpersonal psychotherapy also can be an effective treatment for bulimia nervosa. This is surprising because interpersonal therapy does not address eating disorders directly but instead focuses on difficulties in close relationships. In fact, interpersonal therapy for bulimia initially was studied as a *placebo* treatment. Fairburn and colleagues (1991, 1993) wanted to evaluate whether cognitive behavior therapy had specific effects beyond the general benefits of psychotherapy. They chose interpersonal therapy as a credible placebo, because interpersonal problems often are associated with bulimia nervosa. But they hypothesized that cognitive behavior therapy would outperform the interpersonal approach.

When Fairburn and colleagues (1991) evaluated outcomes shortly after treatment, they found that cognitive behavior therapy was more effective than interpersonal therapy in changing dieting behavior, self-induced vomiting, and attitudes about weight and shape. Cognitive behavior therapy also was more effective than a third condition, behavior therapy alone, in terms of attitude change. However, the results of the two behavioral treatments were similar in other respects.

A very different picture emerged at 12-month follow-up (see Figure 10.5). The behavior therapy alone group deteriorated over time, and a large number of patients dropped out. The cognitive behavior therapy group maintained fairly stable improvements. But the interpersonal therapy group *continued to improve*. At one-year follow-up, in fact, interpersonal therapy equaled cognitive behavior therapy and outdistanced the behavior therapy alone (Fairburn et al., 1993).

The continued improvement for interpersonal therapy was surprising and impressive, for at least two reasons. First, the interpersonal treatments explicitly excluded direct discussions of eating, diet, and related topics. Second, the investigators had lower expectations for interpersonal therapy, and the *allegiance effect* often influences treatment outcome (see Research Methods). A recent, larger study replicated these results, although cognitive behavior therapy again produced more rapid change (Agras et al., 2000).

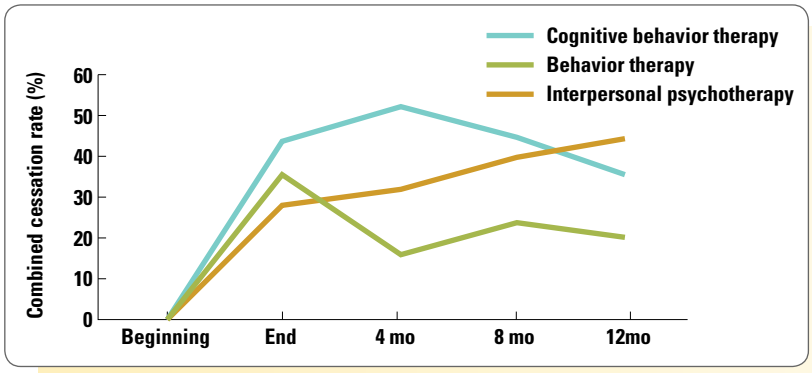


FIGURE 10.5

Percentage of patients who no longer purged or had episodes of bulimia according to objective or subjective reports. Note the decline for the behavior therapy group and continued improvement for the interpersonal therapy group over the 1-year follow-up interval.

Source: From "Psychotherapy and Bulimia Nervosa: Longer-Term Effects of Interpersonal Psychotherapy, Behavior Therapy, and Cognitive Behavior Therapy" by C. G. Fairburn, R. Jones, R. C. Peveler, R.A. Hope, *Archives of General Psychiatry*, (1993), *50,* p. 423. Copyright © 1993. Reprinted by permission of American Medical Association.

RESEARCH METHODS

PSYCHOTHERAPY PLACEBOS

A *placebo* is a treatment that contains no active ingredients for the disorder being treated. A *placebo control group* receives only a placebo treatment. Scientists must include placebo control groups in treatment outcome research, because the mere expectation of change can produce many benefits. New treatments work, in part, because the client and the therapist expect them to work.

Medication placebos are easily administered. Physicians give patients a pill that looks like the real medication but contains no active chemical ingredients. Psychotherapy placebos are much more challenging. How can we create a psychological treatment that contains no active ingredients but increases the client's and the therapist's expectations for change just as much as the real treatment?

One approach is to offer an established, alternative therapy, but one not designed to treat the disorder being studied. In their study of bulimia nervosa, Fairburn et al. (1993) thought interpersonal therapy was a believable placebo psychotherapy. The investigators believed that interpersonal therapy contained no "active ingredients" for treating bulimia nervosa, but thought clients would view it as legitimate.

But this does not fully resolve the problem of creating a psychotherapy placebo. Researchers typically "believe" in their new treatment—otherwise they would not be studying it. Evidence on the *allegiance effect* shows that the *therapist's* beliefs help to make a treatment work. The allegiance effect tells us that cognitive behavior therapy should have been more successful in the Fairburn and colleagues (1993) study, because the investigators were cognitive behavior therapists. In fact, we are particularly impressed by the results for interpersonal therapy, because it overcame the allegiance effect in this study and in a replication (Agras et al., 2000).

What research methods control for the influence of the experimenter's expectations on outcome? In drug research, scientists use the *double-blind study*, where neither the patient nor the therapist knows whether the patient is receiving an active treatment or a placebo. But "real" and placebo psychotherapies are transparent to therapists, making it impossible to conduct double-blind studies of psychotherapy. An alternative approach is to include a pill placebo, a method that also can facilitate comparisons between studies of the effectiveness of drugs and psychotherapy (Klein, 1996). This is a positive step, but even pill placebo effects are not always easy to interpret. For example, medications are more effective when they produce more side effects (Greenberg et al., 1994). There are at least two possible reasons for this. More side effects may increase the patient's expectations for change, because they make the drug seem powerful. Or even in a double-blind study, clinicians may be able to determine whether patients are receiving the real medication based on the side effects.

How can scientists tell if a psychological treatment is more than a placebo?

Another way of addressing the allegiance effect in psychotherapy outcome research is to have investigators who hold *opposing allegiances* participate in the same study. Cognitive behavior therapy is offered by cognitive behavior therapists, interpersonal therapists deliver interpersonal therapy, and so on. This overcomes the allegiance effect but creates a new problem: Because the same therapists cannot deliver the different treatments, effects due to the individual therapists are uncontrolled.

In the absence of a perfect psychotherapy placebo, two conclusions seem clear. First, we must recognize that the expectations of clients, therapists, and experimenters can influence the findings of therapy outcome research. Second, we are particularly impressed when, contrary to expectations, a placebo psychotherapy is as effective as the "real thing."

ANTIDEPRESSANT MEDICATIONS

All classes of antidepressant medications are somewhat effective in treating bulimia nervosa; however, medication alone is *not* the treatment of choice. Binge eating and compensatory behavior improve only among a minority of people treated with antidepressants, and relapse is common when medication is stopped (McElroy et al., 2010). Most importantly, research shows that cognitive behavior therapy is more effective (Hay & Claudino, 2010; Walsh et al., 1997; Wilson et al., 1999). One exception may be treating bulimia in a primary care setting, where most patients fail to complete self-help cognitive behavior therapy programs but are more likely to follow through with antidepressant medication (Walsh et al., 2004). Overall, cognitive behavior therapy is the first-line treatment for bulimia, antidepressant medication may be a useful supplement, and interpersonal therapy is a slower acting alternative (Wilson, 2010).

What treatments are most effective for bulimia?

COURSE AND OUTCOME OF BULIMIA NERVOSA

Bulimia nervosa has a more favorable course than anorexia nervosa, especially with treatment (Thomson-Brenner et al., 2003). About five years following diagnosis, 70 percent of patients are free of symptoms, 20 percent show improvement but continue to struggle, and one in 10 are chronically ill (Keel, 2010). In contrast to anorexia, mortality has been thought to be rare for bulimia, but a recent study found elevated rates, particularly for suicide (Crow et al., 2009). Comorbid psychological disorders also tend to improve with improvements in bulimia nervosa (Keel & Mitchell, 1997). Predictors of continued binge eating include a longer duration, greater emphasis on shape and weight, childhood obesity, poorer social adjustment, persistent compensatory behavior, and comorbid alcohol abuse (Fairburn et al., 2003; Keel, 2010).

Prevention of Eating Disorders

Can eating disorders be prevented? This question is of huge importance, especially given the pervasive body dissatisfaction and disordered eating found among women today. Until recently, the results of prevention research were discouraging. Few, if any, benefits were produced by first-generation prevention efforts, which focused on education about the adverse effects of eating disorders, or by second-generation initiatives offering education about resisting the culture of thinness. However, a third generation of more subtle prevention efforts is promising (Stice & Shaw, 2004).

More successful prevention efforts do not directly focus on body image or disordered eating. Instead, they attack the thinness ideal indirectly, or focus on promoting healthy eating rather than eliminating unhealthy habits (Stice et al., 2006). An important example of the former approach is a "dissonance intervention," in which participants complete tasks inconsistent with the thinness ideal, for example, discussing how to help "younger girls" from becoming obsessed with their appearance. The latter, healthy approach emphasizes the benefits of eating well and exercising. An important study randomly assigned 481 adolescent girls to one of these two prevention programs (each involving only three one-hour group meetings) or to an assessment-only group (no-treatment control group) or three hours of writing about emotional issues (placebo control group; Stice et al., 2006). Results showed improvements due to one or both interventions in body dissatisfaction, internalization of the thin ideal, dieting, and binge eating/purging one month after intervention, and to a lesser extent, at six- and 12-month follow-ups. Figure 10.6 shows the results for binge eating.

Importantly, the results for the dissonance intervention have been replicated in a "real-world" setting (Stice et al., 2009). Moreover, peer-led dissonance groups in sororities also show positive effects (Becker et al., 2008). Psychologists and society clearly have a long way to go to help women, and men, find the right balance between eating too little and too much, being obsessed with appearance, and being lax about health. Still, the results of a new generation of prevention research are an encouraging step in the right direction.

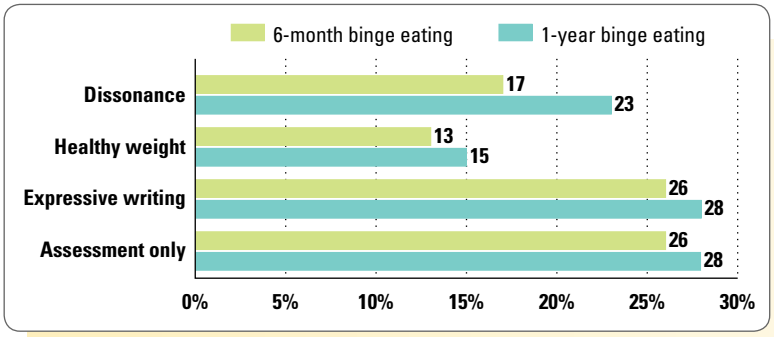


FIGURE 10.6

Binge eating 6 and 12 months after either dissonance, healthy weight, or control interventions. At both times, the healthy weight program produced significantly less binge eating than control conditions. Differences for dissonance training approached significance at 6 months.

Source: From E. Stice, H. Shaw, E. Burton, E. Wade, "Dissonance and Healthy Weight Eating Disorder Prevention Programs: A Randomized Efficacy Trial," in *Journal of Consulting and Clinical Psychology,* 74, 263–275. Copyright © 2006 by the American Psychological Association.

Getting Help

Eating disorders are very common, so it is likely that you or someone close to you may be struggling with eating issues. What can help?

One step is to get more information, but you need to be careful. Some self-help books and websites on eating disorders offer misleading or inaccurate information. And please beware of "pro-ana" and "pro-mia" websites that actually *encourage* eating disorders. One website that we recommend is the homepage of the National Eating Disorders Awareness Association, a nonprofit organization dedicated to increasing the awareness and prevention of eating disorders. The Web page of the National Institute of Mental Health also contains helpful information about eating disorders. An excellent self-help book is *Overcoming Binge Eating*, by Christopher Fairburn, whose treatment research is discussed in this chapter. *Wasted* by Marya Hornbacher is a no-nonsense memoir about struggles with anorexia and bulimia. For parents, we recommend *Help Your Teenager Beat an Eating Disorder* by James Lock and Daniel Le Grange, whose promising new family therapy techniques are also discussed in this chapter.

If you are seriously concerned about your own eating, weight, or body shape, you should talk with a professional. Colleges and universities often have special information and treatments for eating disorders. Call your student health service for information and referral. If no student health resources are available, another option is to talk with your family physician. You should have a physical exam to explore possible medical complications caused by your abnormal eating, and your physician also should know of mental health professionals who specialize in eating disorders. As indicated by the research we review in this chapter, some of the treatment options you should consider include cognitive behavior therapy, the most strongly supported treatment; interpersonal therapy; family therapy; and antidepressant medication. Hospitalization may be another option, but only for very severe weight loss.

If you are concerned about a friend's eating, you should make a plan and talk to her or him. Bring some information on local resources for treating eating disorders. And be prepared to listen as well as to talk! Your friend probably has not confided about his or her problems with many people. If your friend denies a problem, there is no point in arguing. You have done your job by raising the issue. It will be up to him or her to admit to the problem and get help. A good resource before and after talking with a friend is *Surviving an Eating Disorder: Strategies for Friends and Families*, by Michele Siegel, Judith Brisman, and Margot Weinshel.

SUMMARY

- The defining symptoms of **anorexia nervosa** include extreme emaciation, a disturbed perception of one's body, an intense fear of gaining weight, and the cessation of menstruation (in women).
- The defining symptoms of **bulimia nervosa** are **binge eating** and compensatory behavior (**purging** or excessive exercise), a sense of lost control during a binge, and undue focus on weight and shape.
- **Binge eating disorder** is likely to be included in future versions of the DSM, but **obesity** is not.
- The prevalence of both anorexia nervosa and bulimia nervosa has increased dramatically in recent years, particularly among young women.
- Our society's gender roles, standards of beauty, and pubertal changes in body shape and weight all contribute to the onset of eating disorders in young women.
- Four psychological factors in the development of eating disorders are issues of control and perfectionism, dysphoria combined with a lack of introceptive awareness, body image dissatisfaction, and reactions to dietary restraint.
- Biological contributions to eating disorders include the body's attempts to maintain **weight set points,** genetic influences on body weight and shape, and in rare cases, a dysfunction of the hypothalamus.
- There is no clearly effective treatment for anorexia nervosa, which may require inpatient treatment, although a new form of family therapy shows promise at least among adolescents.
- Cognitive behavior therapy is an effective first line treatment for bulimia, while interpersonal psychotherapy and antidepressant medication also can be effective secondary treatments.
- Anorexia and to a lesser extent, bulimia, can be chronic, with a continuation of eating dysfunction even when some symptoms improve.
- Recent research provides hope for the prevention of disordered eating, especially efforts focused on maintaining healthy weight or creating dissonance about the culture of thinness.

The Big Picture

CRITICAL THINKING REVIEW

- **Do men get eating disorders?**
  . . . some experts argue that pressures to be strong and muscular have created a new eating disorder among males . . . (see p. 251)
- **How can you tell if someone has an eating disorder?**
  The most obvious and most dangerous symptom of anorexia nervosa is a *refusal to maintain a minimally normal body weight* . . . (see p. 252)
- **How do media images of women contribute to eating disorders?**
  Popular attitudes about women in the United States tell us that "looks are everything," and thinness is essential to good looks . . . (see p. 259)
- **Why do some girls and women develop eating disorders while others do not?**
  . . . not every woman in the United States develops an eating disorder, so other factors must interact with culture to produce eating disorders . . . (see p. 262)
- **What treatments work for anorexia and bulimia?**
  The treatments for anorexia nervosa and bulimia nervosa differ in approach and effectiveness . . . (see p. 265)
- **Can eating disorders be prevented?**
  This question is of huge importance, especially given the pervasive body dissatisfaction and disordered eating found among women today . . . (see p. 268)

KEY TERMS

anorexia nervosa
binge eating
binge eating disorder
bulimia nervosa
cohort
cohort effects
distorted body image
eating disorders
obesity
purging
weight set point

11

Substance Use Disorders

▶ *Traffic* explores the dark world of international drug trafficking from many perspectives, including a user, a dealer, an enforcement officer, and a politician. The film illustrates vividly the impact of drugs on people's lives and raises challenging questions about the consequences of social policies aimed at prohibiting access to drugs.

The abuse of alcohol and other drugs is one of the most serious problems facing our society today. It is likely that you or someone close to you will be affected by the substance use issues outlined in this chapter. Alcohol and drug problems receive a great deal of attention in the popular media, as illustrated by actress Lindsay Lohan's repeated struggles with alcohol and the drug-related suicide of Kurt Cobain, leader of the rock group Nirvana. Research efforts, treatment priorities, and national publicity have all helped transform national attitudes about the abuse of chemical substances. The picture of the drug addict as a homeless derelict whose personality defects and lack of motivation are largely responsible for the problem is being replaced by a new view in which substance abuse is seen as a chronic mental disorder that affects people from all walks of life.

The Big Picture

- Is each form of addiction a unique disorder?
- Are some drugs more addicting than others?
- Can you be dependent on a drug if you don't experience withdrawal when you stop taking it?
- How does culture influence the ways that people use drugs?
- Why do some people become dependent on alcohol while most are able to drink socially without developing problems?
- Is Alcoholics Anonymous the most effective form of help for people with alcohol-related problems?
- How can you help a person who refuses to acknowledge that he or she has a drinking problem?

OVERVIEW

The costs of substance abuse are astronomical. According to the World Health Organization, alcohol use was responsible for 5 percent of the total burden of disease and disability worldwide in 2004 (Rehm et al, 2009). Cirrhosis of the liver, which is frequently the result of chronic alcoholism, is a leading cause of death in the United States. In addition, alcohol plays a prominent role in many suicides, homicides, and motor vehicle accidents. The rate of deaths attributable to the use of tobacco is growing rapidly, particularly in developing countries, where 50 percent of adult men are regular smokers. By the year 2020, tobacco is expected to kill between 8 million and 9 million people annually worldwide, more than any single disease, including AIDS (Lopez et al., 2006).

DSM-IV-TR uses two terms to describe substance use disorders, and these terms reflect different levels of severity. **Substance dependence,** the more severe of the two forms, refers to a pattern of repeated self-administration that often results in tolerance, the need for increased amounts of the drug to achieve intoxication; withdrawal, unpleasant physical and psychological effects that the person experiences when he or she tries to stop taking the drug; and compulsive drug-taking behavior. **Substance abuse** describes a more broadly conceived, less severe pattern of drug use that is defined in terms of interference with the person's ability to fulfill major role obligations at work or at home, the recurrent use of a drug in dangerous situations, and repeated legal difficulties associated with drug use.

Addiction is another term that is often used to describe problems such as alcoholism. It is essentially synonymous with substance dependence, although it does not appear in DSM-IV-TR. The term addiction is being used more frequently in recent years, partly because the field has become increasingly interested in similarities and distinctions between substance use disorders and other kinds of impulsive behavior problems that involve loss of control or craving in one form or another. These include addiction-like problems such as pathological gambling, excessive use of the Internet, and hypersexual behavior (see the section on Impulse Control Disorders in Chapter 9). The next edition of the diagnostic manual, DSM-V, may include some of these problems in the same section with substance use disorders (Petry, 2006).

A **drug of abuse,** sometimes called a *psychoactive substance,* is a chemical substance that alters a person's mood, level of perception, or brain functioning (Schuckit, 2010). All drugs of abuse can be used to increase a person's psychological comfort level (make one feel "high") or to alter levels of consciousness. The list of chemicals on which people can become dependent is long and seems to be growing longer. It includes drugs that are legally available, whether over the counter or by prescription only, as well as many that are illegal (see Table 11.1).

Depressants of the central nervous system (CNS) include alcohol as well as types of medications that are used to help people sleep, called *hypnotics,* and those for relieving anxiety, known as *sedatives* or *anxiolytics.* The CNS stimulants include illegal drugs like amphetamine and cocaine, as well as nicotine and caffeine. The opiates, also called *narcotic analgesics,* can be used clinically to decrease pain. The *cannabinoids,* such as marijuana, produce euphoria and an altered sense of time. At higher doses, they may produce hallucinations. People with a substance use disorder frequently abuse several types of drugs; this condition is known as **polysubstance abuse.**

One basic question we must address is whether we should view each type of addiction as a unique problem. Experts who answer "yes" to this question point out that each class of abused substance seems to affect the body in distinct ways. For example, when taken orally, some opiates can be used for long periods of time without leading to significant organ damage (Jaffe & Jaffe, 1999). Chronic use of alcohol and tobacco, on the other hand, can have a devastating impact on a person's physical health.

Despite these differences, the various forms of substance abuse share many common elements. All forms of abuse represent an inherent conflict between immediate pleasure and longer-term harmful consequences. The psychological and biochemical effects on the user are often similar, as are the negative consequences for both social and occupational

TABLE 11.1 Commonly Abused Drugs

Class	Examples	Brand Names and Street Names
CNS Depressants	Alcohol	beer, wine, liquor
	Barbiturates	*barbs,* Amytal, Nembutal, Seconal
	Benzodiazepines	*roofies, tanks,* Xanax, Valium, Halcion
	Methaqualone	*quaalude, ludes*
CNS Stimulants	Amphetamine	*black beauties, crosses, hearts*
	Cocaine	*blow, coke, crack, flake, rocks, snow*
	Methamphetamine	*crank, crystal, glass, ice, speed*
	Nicotine	cigars, cigarettes, smokeless tobacco
	Caffeine	coffee, tea, soft drinks
Opiates	Heroin	*horse, smack, H, junk, skag*
	Opium	laudanum, paregoric, dover's powder
	Morphine	Roxanol, Duramorph
	Methadone	Amidone, Dolophine, Methadose
	Codeine	Tylenol w/Codeine, Robitussin A-C
Cannabinoids	Marijuana	*grass, herb, pot, reefer, smoke, weed*
	Hashish	*hash*
Hallucinogens	LSD	*acid, microdot*
	Mescaline	*buttons, cactus, mesc, peyote*
	Psilocybin	*magic, mushroom, purple passion*
	Phencyclidine	PCP, *angel dust, boat, hog, love boat*
	MDMA	ecstasy, XTC, Adam

Note: Street names for drugs appear in italics.

behaviors. The reasons for initial experimentation with a drug, the factors that influence the transition to dependence, and the processes that lead to relapse after initial efforts to change are all similar in many respects. For these reasons, many clinicians and researchers have adopted a view of substance abuse that emphasizes common causes, behaviors, and consequences (Lesch et al., 2011). In fact, DSM-IV-TR employs a single set of diagnostic criteria that defines dependence for all types of drugs.

The variety of problems associated with substance use disorders can be illustrated using a case study of alcohol dependence. Ernest Hemingway (1899–1961), a Nobel Prize–winning writer, was severely dependent on alcohol for many years. The following paragraphs, quoted from an article by Paul Johnson (1989), describe the progression of Hemingway's drinking and the problems that it created. They illustrate many typical features of substance dependence, as well as the devastating impact that alcohol can have on various organs of the body. Johnson's description also raises a number of interesting questions about the etiology of this disorder. Most men and women consume alcoholic beverages at some point during their lives. Why do some people become dependent on alcohol while others do not? What factors influence the transition from social drinking to abuse?

CASE STUDY Ernest Hemingway's Alcohol Dependence

Hemingway began to drink as a teenager, the local blacksmith secretly supplying him with strong cider. His mother noted his habit and always feared he would become an alcoholic. In Italy he progressed to wine, then had his first hard liquor at the officers' club in Milan. His wound [from World War I] and an unhappy love affair provoked heavy drinking: In the hospital, his wardrobe was found to be full of empty cognac bottles, an ominous sign. In Paris in the 1920s, he bought Beaune by the gallon at a wine cooperative and would and did drink five or six bottles of red at a meal. He taught Scott Fitzgerald to drink wine direct from the bottle, which, he said, was like "a girl going swimming without her swimming suit." In New York he was "cockeyed," he said, for "several days" after signing his contract for *The Sun Also Rises,* probably his first prolonged bout.

Hemingway particularly liked to drink with women, as this seemed to him, vicariously, to signify his mother's approval. Hadley [the first of his four wives] drank a lot with him, and wrote: "I still cherish, you know, the remark you made that you almost worshipped me as a drinker." The same disastrous role was played by his pretty 1930s companion in Havana, Jane Mason, with whom he drank gin followed by champagne chasers and huge jars of iced daiquiris; it was indeed in Cuba in this decade that his drinking first got completely out of hand. One bartender there said he could "drink more martinis than any man I have ever seen." On safari, he was seen sneaking out of his tent at 5 A.M. to get a drink. His brother Leicester said that, by the end of the 1930s, at Key West, he was drinking 17 Scotch-and-sodas a day and often taking a bottle of champagne to bed with him at night.

At this period, his liver for the first time began to cause him acute pain. He was told by his doctor to give up alcohol completely, and indeed he tried to limit his consumption to three whiskeys before dinner. But that did not last. During World War II his drinking mounted steadily and by the mid-1940s he was reportedly pouring gin into his tea at breakfast. A. E. Hotchner, interviewing him for *Cosmopolitan* in 1948, said he dispatched seven double-size Papa Doubles (the Havana drink named after him, a mixture of rum, grapefruit, and maraschino), and when he left for dinner took an eighth with him for the drive. And on top of all, there was constant whiskey: His son Patrick said his father got through a quart of whiskey a day for the last 20 years of his life.

Hemingway's ability to hold his liquor was remarkable. Lillian Ross, who wrote his profile for the *New Yorker,* does not seem to have noticed he was drunk a lot of the time he talked to her. Denis Zaphior said of his last safari: "I suppose he was drunk the whole time but seldom showed it." He also demonstrated an unusual ability to cut down his drinking or even to eliminate it altogether for brief periods, and this, in addition to his strong physique, enabled him to survive.

But despite his physique, his alcoholism had a direct impact on his health, beginning with his damaged liver in the late 1930s. By 1959, following his last big drinking bout in Spain, he was experiencing both kidney and liver trouble and possibly hemochromatosis (cirrhosis, bronzed skin, diabetes), edema of the ankles, cramps, chronic insomnia, blood-clotting and high blood uremia, as well as his skin complaints. He was impotent and prematurely aged. Even so, he was still on his feet, still alive; and the thought had become unbearable to him. His father had committed suicide because of his fear of mortal illness. Hemingway feared that his illnesses were not mortal: On July 2, 1961, after various unsuccessful treatments for depression and paranoia, he got hold of his best English double-barreled shotgun, put two canisters in it, and blew away his entire cranial vault.

Why did Hemingway long for death [and why did he drink]? He felt he was failing his art. Hemingway had many grievous faults, but there was one thing he did not lack: artistic integrity. It shines like a beacon through his whole life. He set himself the task of creating a new way of writing English, and fiction, and he succeeded. It was one of the salient events in the history of our language and is now an inescapable part of it. He devoted to this task immense resources of creative skill, energy, and patience. That in itself was difficult. But far more difficult, as he discovered, was to maintain the high creative standards he had set himself. This became apparent to him in the mid-1930s and added to his habitual depression. From then on his few successful stories were aberrations in a long downward slide.

If Hemingway had been less of an artist, it might not have mattered to him as a man. He would simply have written and published inferior novels, as many writers do. But he knew when he wrote below his best, and the knowledge was intolerable to him. He sought the help of alcohol, even in working hours. He was first observed with a drink, a "Rum St. James," in front of him while writing in the 1920s. This custom, rare at first, became intermittent, then invariable. By the 1940s, he was said to wake at 4:30 A.M. [He] "usually starts drinking right away and writes standing up, with a pencil in one hand and a drink in another." The effect on his work was exactly as might be expected, disastrous. Hemingway began to produce large quantities of unpublishable material, or material he felt did not reach the minimum standard he set himself. Some was published nonetheless and was seen to be inferior, even a parody of his earlier work. There were one or two exceptions, notably *The Old Man and the Sea* (1952), which won him the Nobel Prize, though there was an element of self-parody in that, too. But the general level was low, and falling, and Hemingway's awareness of his inability to recapture his genius, let alone develop it, accelerated the spinning circle of depression and drink (Johnson, 1989, pp. 58–59).

Symptoms

Substance use disorders are associated with a host of problems, many of which are illustrated in the life of Ernest Hemingway. Nevertheless, substance dependence is difficult to define. Alcoholism is one important example. George Vaillant (1995), a psychiatrist at Harvard Medical School and the author of an important longitudinal study of alcoholic men, notes that it is difficult to say that one specific problem or set of problems represents the core features of this disorder:

> Not only is there no single symptom that defines alcoholism, but often it is not who is drinking but who is watching that defines a symptom. A drinker may worry that he has an alcohol problem because of his impotence. His wife may drag him to an alcohol clinic because he slapped her during a blackout. Once he is at the clinic, the doctor calls him an alcoholic because of his abnormal liver-function tests. Later society labels him a drunk because of a second episode of driving while intoxicated. (p. 24)

The number of problems that a person encounters seems to provide the most useful distinction between people who are dependent on a substance and those who are not. These problems can be sorted loosely into two general areas: (1) patterns of pathological consumption, including psychological and physiological dependence; and (2) consequences that follow a prolonged pattern of abuse, including social and occupational impairment, legal and financial difficulties, and deteriorating medical condition.

It might seem that the actual amount of a drug of abuse that a person consumes would be the best indication of the existence of a problem. Hemingway, for example, clearly consumed enormous quantities of alcohol over a period of many years. The average person with an alcohol use disorder does drink more frequently and in larger quantities than the average person without an alcohol use disorder (Keyes et al., 2009). Nevertheless, the amount of a drug that a specific person consumes is not a good way to define substance use disorders, because people vary significantly in the amount of any given drug they can consume. Factors such as age, gender, activity level, and overall physical health influence a person's ability to metabolize various kinds of drugs. For example, some people can drink a lot without developing problems; others drink relatively little and have difficulties.

THE CONCEPT OF SUBSTANCE DEPENDENCE

Many psychological features or problems are associated with dependence on chemical substances. One such feature involves *craving*. This word is frequently used to describe a forceful urge to use drugs, but the relationship between craving and drug use is actually very complex (Eliason & Amodia, 2007; Sayette et al., 2000). People who are dependent on drugs often say that they take the drug to control how they are feeling. They need it to relieve negative mood states or to avoid withdrawal symptoms from previous episodes. They may feel compelled to take the drug as a way to prepare for certain activities, such as public speaking, writing, or sex. Some clinicians refer to this condition as **psychological dependence.**

One useful index of craving is the amount of time that the person spends planning to take the drug. Is access to drugs or alcohol a constant preoccupation? If the person is invited to a party or is planning to eat at a restaurant, does he or she always inquire about the availability of alcoholic drinks? If the person is going to spend a few days at the beach in a neighboring state, will he or she worry more about whether liquor stores will be closed on weekends or holidays than about having enough food, clothes, or recreational equipment?

As the problem progresses, it is not unusual for the person who abuses drugs to try to stop. In the case of alcoholism, for example, it is possible for even heavy drinkers to abstain for at least short periods of time. Most clinicians and researchers agree that diminished control over drinking is a crucial feature of the disorder. Some experts have described this issue as "freedom of choice." When a person first experiments with the use of alcohol, his or her behavior is clearly voluntary; the person is not compelled to drink. After drinking heavily for a long period of time, most people with a drinking disorder try to stop. Unfortunately, efforts at self-control are typically short-lived and usually fail.

Tolerance and Withdrawal Two particularly important features of substance dependence are the phenomena known as tolerance and withdrawal. These symptoms are usually interpreted as evidence of *physiological dependence*. **Tolerance** refers to the process through which the nervous system becomes less sensitive to the effects of alcohol or any other drug of abuse. For example, a person who has been regularly exposed to alcohol will need to drink increased quantities to achieve the same subjective effect ("buzz," "high," or level of intoxication).

What evidence is needed to show that a drug is addictive?

The development of drug tolerance seems to be the result of three separate mechanisms (Julien, 2001). Two are pharmacological and the third is behavioral. *Metabolic tolerance* develops when repeated exposure to a drug causes the person's liver to produce more enzymes that are used to metabolize, that is, break down, the drug. The drug, therefore, is metabolized more quickly and the person has to take increasingly larger doses in order to maintain the same level in his or her body. *Pharmacodynamic tolerance* occurs when receptors in the brain (see Figure 2.2 on page 33) adapt to continued presence of the drug. The neuron may adapt by reducing the number of receptors or by reducing their sensitivity to the drug. This process is known as *down regulation*. The third process involved in drug tolerance involves *behavioral conditioning mechanisms* (Siegel, 2005). Cues that are regularly associated with the administration of a drug begin to function as conditioned stimuli and elicit a conditioned response that is opposite in direction to the natural effect of the drug. As this compensatory response increases in strength, it competes with the drug response so that larger amounts of the drug must be taken to achieve the same effect.

Some drugs are much more likely than others to produce a buildup of tolerance (APA, 2000). The most substantial tolerance effects are found among heavy users of opioids, such as heroin, and CNS stimulants, such as amphetamine and cocaine. Pronounced tolerance is also found among people who use

alcohol and nicotine. The evidence is unclear regarding tolerance effects and prolonged use of marijuana and hashish. Most people who use cannabinoids are not aware of tolerance effects, but these effects have been demonstrated in animal studies. Hallucinogens (LSD) and phencyclidine (PCP) may not lead to the development of tolerance.

Withdrawal refers to the symptoms experienced when a person stops using a drug. The symptoms can go on for several days. For example, alcohol is a CNS depressant, and the heavy drinker's system becomes accustomed to functioning in a chronically depressed state. When the person stops drinking, the system begins to rebound within several hours, producing many unpleasant side effects—hand tremors, sweating, nausea, anxiety, and insomnia. The most serious forms of withdrawal include convulsions and visual, tactile, or auditory hallucinations. Some people develop delirium, a sudden disturbance of consciousness that is accompanied by changes in cognitive processes such as lack of awareness of the environment or inability to sustain attention (see Chapter 14). This syndrome is called *alcohol withdrawal delirium* in DSM-IV-TR (more traditionally known as *delirium tremens,* or DTs) if it is induced by withdrawal from alcohol.

The symptoms of withdrawal vary considerably for different kinds of substances. Table 11.2 compares various drugs of abuse in terms of withdrawal and other related characteristics. Unpleasant reactions are most evident during withdrawal from alcohol, opioids, and the general class of sedatives, hypnotics, and anxiolytics (such as Valium and Xanax). Withdrawal symptoms are also associated with stimulants, such as amphetamine, cocaine, and nicotine, although they are sometimes less pronounced than those associated with alcohol and opioids.

Withdrawal symptoms are not often seen after repeated use of cannabis or hallucinogens, and they have not been demonstrated with phencyclidine. Caffeine is the most widely used psychoactive substance in the world. We all know people who crave coffee, especially in the morning. And some heavy coffee users experience severe headaches when they stop drinking caffeine (James & Keane, 2007). You may be surprised to see in Table 11.2 that, according to DSM-IV-TR, the use of caffeine is not considered to lead to dependence or withdrawal symptoms. The authors of DSM-IV-TR acknowledged these symptoms, but they decided that the symptoms did not cause clinically significant distress and impairment and, therefore, should not be included in the manual as a type of mental disorder.

All these problems serve to emphasize the fact that symptoms of substance use disorders fall along a continuum. It is convenient to consider these problems in terms of qualitative distinctions: people who can control their drinking and those who cannot; people who crave alcohol and those who do not; people who have developed a tolerance to the drug and those who have not; and so on. In fact, there are no clear dividing lines on any of these dimensions. Drug use disorders lie on a continuum of severity (Helzer et al., 2008). For this reason it is extremely difficult to define the nature of substance dependence disorders.

People can become dependent on many different kinds of drugs. Although patterns of dependence are similar in some ways for all drugs, each type of drug also has some unique features. In the next few pages we briefly review some of the most important classes of drugs. For each group, we will describe short-term effects on physiology and behavior, as well as the consequences of long-term abuse. Unless otherwise specified, these descriptions are based on information presented by William McKim (2006) in his textbook on drugs and behavior.

TABLE 11.2 Comparison of Various Psychoactive Substances

Substance	Can Produce Dependence	Can Produce Intoxication	Associated Withdrawal	Can Produce Dementia
Alcohol	yes	yes	yes	yes
Amphetamines	yes	yes	yes	no
Caffeine	no	yes	no	no
Marijuana/hashish	yes	yes	no	no
Cocaine	yes	yes	yes	no
Hallucinogens	yes	yes	no	no
Inhalants	yes	yes	no	yes
Nicotine	yes	no	yes	no
Opiates	yes	yes	yes	no
Phencyclidine (PCP)	yes	yes	no	no
Sedatives, hypnotics, and anxiolytics	yes	yes	yes	yes

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

MyPsychLab VIDEO CASE

Alcoholism

CHRIS

"The toughest thing I ever did was admitting that I had a problem."

Watch the video "Alcoholism: Chris" on MyPsychLab. How was Chris's pattern of drinking different from that of his friends?

The negative consequences of alcohol dependence typically include a devastating impact on social relationships and work performance.

ALCOHOL

Alcohol affects virtually every organ and system in the body. After alcohol has been ingested, it is absorbed through membranes in the stomach, small intestine, and colon. The rate at which it is absorbed is influenced by many variables, including the concentration of alcohol in the beverage (for example, distilled spirits are absorbed more rapidly than beer or wine), the volume and rate of consumption, and the presence of food in the digestive system. After it is absorbed, alcohol is distributed to all the body's organ systems. Almost all the alcohol that a person consumes is eventually broken down or metabolized in the liver. The rate at which alcohol is metabolized varies from person to person, but the average person can metabolize about 1 ounce of 90-proof liquor or 12 ounces of beer per hour (Nathan, 1993). If the person's consumption rate exceeds this metabolic limit, then blood alcohol levels will rise.

Short-Term Effects Blood alcohol levels are measured in terms of the amount of alcohol per unit of blood. A "drink" is considered to be 12 ounces of beer, 4 ounces of wine, or 1 ounce of 86-proof whiskey. The average 160-pound man who consumes five drinks in one hour will have a blood alcohol level of 100 milligrams (mg) per 100 milliliters (ml) of blood, or 100 mg percent (Kowalski, 1998). There is a strong correlation between blood alcohol levels and CNS intoxicating effects. According to DSM-IV-TR, the symptoms of alcohol intoxication include slurred speech, lack of coordination, an unsteady gait, nystagmus (involuntary to-and-fro movement of the eyeballs induced when the person looks upward or to the side), impaired attention or memory, and stupor or coma.

In most states, the legal limit of alcohol concentration for driving is 100 mg percent. Some state legislatures have lowered this limit to 80 mg percent, because slowed reaction times and interference with other driving skills may occur at lower blood alcohol levels (Mejeur, 1999). People with levels of 150 to 300 mg percent will almost always act intoxicated. Neurological and respiration complications begin to appear at higher levels. There is an extreme risk of coma leading to toxic death when blood alcohol levels go above 400 mg percent.

Long-Term Consequences The prolonged use and abuse of alcohol can have a devastating impact on many areas of a person's life. The disruption of relationships with family and friends can be especially painful. The impact of Hemingway's drinking on his writing career and his family life is clearly evident. Most critics agree that his literary accomplishments were confined primarily to the early stages of his career, before his alcoholism began to interfere with his ability to write. Drinking also took its toll on his marriages, which were characterized by frequent and occasionally furious conflict in public and by repeated episodes of verbal and physical abuse in private (Johnson, 1989). Also, the heavy use of alcohol by a pregnant woman can cause damage to her fetus (see Chapter 15).

Many people who abuse alcohol experience blackouts. In some cases, abusers may continue to function without passing out, but they will be unable to remember their behavior. An example is the person who drives home drunk from a party and in the morning finds a dent in the car bumper but can't remember how it got there. Sometimes problem drinkers will be told by a friend about how they behaved at the previous night's party, but they cannot remember what they did.

Regular heavy use of alcohol is also likely to interfere with job performance. Coworkers and supervisors may complain. Attendance at work may become sporadic. Eventually, the heavy drinker may be suspended or fired. Related to job performance is the problem of financial difficulties. Losing one's job is clearly detrimental to one's financial stability, as are the costs of divorce, healthcare, liquor, and so on.

Many heavy drinkers encounter problems with legal authorities. These problems may include arrests for drunken driving and public intoxication, as well as charges of spouse and child abuse. Many forms of violent behavior are more likely to be committed when a person has been drinking.

On a biological level, prolonged exposure to high levels of alcohol can disrupt the functions of several important organ systems, especially the liver, pancreas, gastrointestinal system, cardiovascular system, and endocrine system. The symptoms of alcoholism include many secondary health problems, such as cirrhosis of the liver, heart problems (in part, the result of being overweight), and various forms of cancer, as well as severe and persistent forms of dementia and memory impairment or amnestic disorders, such as Korsakoff's

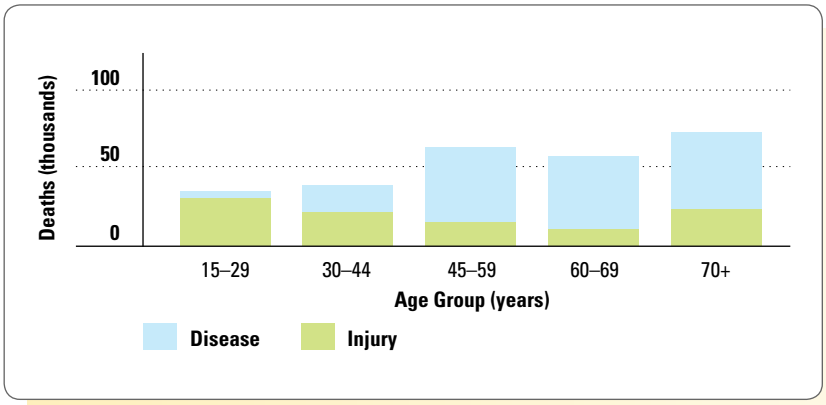


FIGURE 11.1 Male Deaths Caused by Alcohol Use in Established Market Economies

Young men are more likely to be killed by alcohol-related injuries, while older men often die as a result of alcohol-related disease.

Source: C. J. L. Murray and A. D. Lopez, 1997, "Global Mortality, Disability, and the Contribution of Risk Factors: Global Burden of Disease Study," *Lancet, 349,* pp.1436–1442. Copyright © 1997. Reprinted by permission of Elsevier, Ltd.

syndrome (see Chapter 14). Alcoholism is also associated with nutritional disturbances of many types, because chronic abusers often drink instead of eating balanced meals. In fact, over an extended period of time, alcohol dependence has more negative health consequences than does abuse of any other drug, with the exception of nicotine.

The misuse of alcohol leads to an enormous number of severe injuries and premature deaths in every region of the world (Cornelius et al., 2008). The specific impact of alcohol varies among geographic regions, in part because of differences in the age structure of different populations. Deaths that result from alcohol-related injuries are much more common among young men, while deaths from alcohol-related diseases are responsible for more deaths among older men (Murray & Lopez, 1997; see Figure 11.1).

NICOTINE

Nicotine is the active ingredient in tobacco, which is its only natural source. Nicotine is almost never taken in its pure form because it can be toxic. Very high doses have extremely unpleasant effects. Controlled doses are easier to achieve by smoking or chewing tobacco, which provides a diluted concentration of nicotine. Another way of ingesting nicotine is to inhale snuff (powdered tobacco) into the nostrils. When tobacco smoke is inhaled, nicotine is absorbed into the blood through the mucous membranes of the lungs. This route of administration results in the highest concentrations of nicotine because it is carried directly from the lungs to the heart and from there to the brain.

Short-Term Effects The effects of nicotine on the peripheral nervous system (see Chapter 2) include increases in heart rate and blood pressure. In the central nervous system, nicotine has pervasive effects on a number of neurotransmitter systems (Houezec, 1998). It stimulates the release of norepinephrine from several sites, producing CNS arousal. Nicotine also causes the release of dopamine and norepinephrine in the mesolimbic dopamine pathway, also known as the reward system of the brain. The serotonin system, which also mediates the effects of antidepressant medication, is influenced by nicotine. In fact, some people have suggested that nicotine mimics the effects of antidepressant drugs.

Nicotine has a complex influence on subjective mood states. Many people say that they smoke because it makes them feel more relaxed. Some believe that it helps them control their subjective response to stress. This phenomenon is somewhat paradoxical in light of the fact that nicotine leads to increased arousal of the sympathetic nervous system. Various explanations may account for this apparent inconsistency. One involves differences in dosage levels; low doses of nicotine may lead to increased arousal while higher doses lead to relaxation. Another alternative involves withdrawal. Regular smokers may feel relaxed when they smoke a cigarette because it relieves unpleasant symptoms of withdrawal.

Long-Term Consequences Nicotine is one of the most harmful and deadly addicting drugs. Considerable evidence points to the development of both tolerance and withdrawal symptoms among people who regularly smoke or chew tobacco. The physiological symptoms of withdrawal from nicotine include drowsiness, lightheadedness, headache, muscle tremors, and nausea. People who are attempting to quit smoking typically experience sleeping problems, weight gain, concentration difficulties, and mood swings ranging from anxiety to anger and depression (Hughes, 2007b). From a psychological point of view, withdrawal from nicotine is just as difficult as withdrawal from heroin. Many people report that these symptoms disappear after a few months, but some have serious cravings for several years after they quit.

People who smoke tobacco increase their risk of developing many fatal diseases, including heart disease, lung disease (bronchitis and emphysema), and various types of cancer (Kozlowski, Henningfield, & Brigham, 2001). Eighty percent of all deaths caused by lung cancer can be attributed to smoking tobacco. More than 3.5 million people in the world die prematurely each year as a result of tobacco. Large numbers of people are also killed or injured in fires caused by careless smoking. Women who smoke are also more likely to experience fertility problems. Babies born to mothers who smoked during pregnancy are also likely to weigh less than those born to mothers who do not smoke, and they may be more vulnerable to certain types of birth defects.

AMPHETAMINE AND COCAINE

Members of the class of drugs known as **psychomotor stimulants** produce their effects by simulating the actions of certain neurotransmitters, specifically epinephrine, norepinephrine, dopamine, and serotonin (as discussed later in this chapter). Cocaine is a naturally occurring stimulant drug that is extracted from the leaf of a small tree that grows at high elevations, as in the Andes Mountains. The amphetamines (such as dexedrine and methamphetamine) are produced synthetically.

The stimulants can be taken orally, injected, or inhaled. It is easier to maintain a constant blood level when the drugs are taken orally. They are absorbed more slowly through the digestive system, and their effects are less potent. More dramatic effects are achieved by injecting the drug or sniffing it. Cocaine can also be smoked, using various procedures that have been popularized in the past several years. Some people employ a particularly dangerous procedure called "freebasing," in which the drug is heated and its vapors are inhaled. Many people have been seriously burned when these highly combustible chemicals are accidentally ignited.

Short-Term Effects Cocaine and amphetamines are called stimulants because they activate the sympathetic nervous system (Constable, 2004). They increase heart rate and blood pressure and dilate the blood vessels and the air passages of the lungs. Stimulants also suppress the appetite and prevent sleep. These effects have been among the reasons for the popularity and frequent abuse of stimulants. They have been used, for example, by truck drivers who want to stay awake on long trips and by students who want to stay awake to study for exams. Unfortunately, in addition to their addicting properties, large doses of amphetamines can also lead to dizziness, confusion, and panic states, which clearly interfere with activities such as driving and studying.

Many people use (and abuse) stimulants because they induce a positive mood state. When they are injected, amphetamines and cocaine produce very similar subjective effects, but the effects of cocaine do not last as long. Low doses of amphetamines make people feel more confident, friendly, and energetic. At higher doses, the person is likely to experience a brief, intense feeling of euphoria. The rushes associated with snorting or injecting cocaine are frequently described in sexual terms. Although many people believe that cocaine enhances sexual arousal and pleasure, most of the evidence suggests that prolonged use leads to sexual dysfunction (Jaffe, 1995). Tolerance develops quickly to the euphoric effects of stimulant drugs. The feelings of exhilaration and well-being are typically followed, several hours later, by the onset of lethargy and a mildly depressed or irritable mood.

Acute overdoses of stimulant drugs can result in irregular heartbeat, convulsions, coma, and death. The highly publicized overdose deaths of several prominent athletes, such as that of All-American basketball star Len Bias in 1986, indicate that the intense cardiovascular effects of cocaine can be fatal, even among people who are otherwise strong and healthy. Individual differences in sensitivity to the subjective effects of cocaine may play a role in cocaine-related deaths. In other words, people who are resistant to cocaine-induced euphoria may consume unusually large quantities of the drug while trying to achieve the rush that others have described.

Lenny Bruce was an irreverent standup comedian and brilliant social critic. His abuse of various drugs, including amphetamines and heroin, disrupted his personal life and interfered with his ability to perform. He died from an overdose in 1966 at the age of 40.

Long-Term Consequences High doses of amphetamines and cocaine can lead to the onset of psychosis. The risk of a psychotic reaction seems to increase with repeated exposure to the drug (Bolla, Cadet, & London, 1998). This syndrome can appear in people who have no prior history of mental disorder, and it usually disappears a few days after the drug has been cleared. Stimulants can also increase the severity of symptoms among people who had already developed some type of psychotic condition. The symptoms of amphetamine psychosis include auditory and visual hallucinations, as well as delusions of persecution and grandeur.

As with other forms of addiction, the most devastating effects of stimulant drugs frequently center around the disruption of occupational and social roles. The compulsion to continue taking cocaine can lead to physical exhaustion and financial ruin. People who are dependent on cocaine must spend enormous amounts of money to support their habit. They may have to sell important assets, such as their homes and cars, in order to finance extended binges. Some people become involved in a variety of criminal activities in order to raise enough money to purchase drugs.

What are the long-term consequences of abusing psychomotor stimulants?

Prolonged use of amphetamines has also been linked to an increase in violent behavior, but it is not clear whether this phenomenon is due to the drug itself or to the lifestyles with which it is frequently associated. Some violence might be related to a drug-induced increase in paranoia and hostility. Statistics concerning drugs and violent crime are very difficult to interpret. The direct effects of the drug on human behavior are confounded with various economic and social factors that are associated with buying, selling, and using an expensive, illegal drug like cocaine.

People who discontinue taking stimulant drugs do not typically experience severe withdrawal symptoms. The most common reaction is depression. Long-term exposure to high doses of amphetamine can lead to a profound state of clinical depression, which is often accompanied by ideas of suicide.

OPIATES

The **opiates** (sometimes called opioids) are drugs that have properties similar to

those of opium. The natural source of opium is a poppy with a white flower. The main active ingredients in opium are morphine and codeine, both of which are widely used in medicine, particularly to relieve pain. They are available legally only by prescription in the United States. In Canada, small quantities of codeine are available without a prescription in over-the-counter painkillers and cough medicines. Heroin is a synthetic opiate that is made by modifying the morphine molecule. It was originally marketed as an alternative to morphine when physicians believed, erroneously, that heroin is not addictive.

The opiates can be taken orally, injected, or inhaled. Opium is sometimes eaten or smoked. When morphine is used as a painkiller, it is taken orally so that it is absorbed slowly through the digestive system. People who use morphine for subjective effects most often inject the drug because it leads more quickly to high concentrations in brain tissue. Heroin can be injected, inhaled through the nose in the form of snuff, or smoked and inhaled through a pipe or tube.

Short-Term Effects The opiates can induce a state of dreamlike euphoria, which may be accompanied by increased sensitivity in hearing and vision. People who inject morphine or heroin also experience a rush—a brief, intense feeling of pleasure that is sometimes described as being like an orgasm in the entire body.

Laboratory studies of mood indicate that the positive, emotional effects of opiates do not last. They are soon replaced by long-term negative changes in mood and emotion. These unpleasant experiences are relieved for 30 to 60 minutes after each new injection of the drug, but they eventually color most of the rest of the person's waking experience.

The opiates can induce nausea and vomiting among novice users, constrict the pupils of the eye, and disrupt the coordination of the digestive system. Continued use of opiates decreases the level of sex hormones in both women and men, resulting in reduced sex drive and impaired fertility.

Some people mix cocaine and opiates into a mixture known as a *speedball* to enhance these subjective feelings. The following brief case describes the preparation of this combination of drugs and one heroin addict's immediate reaction to the injection of a speedball.

The positive, emotional effects of opiates do not last. They are soon replaced by long-term negative changes in mood and emotion.

BRIEF CASE STUDY

Feelings After Injecting Heroin

He pushes the plunger on the syringe, squirting water into the heroin powder, then strikes a match and waves it just under the metal lid. The liquid bubbles and the heroin quickly dissolves with very little heat required. *That's good,* he thinks. Sometimes the dope is so good it needs hardly any fire to dissolve it. Next, he shakes in a couple of small rocks of cocaine from the foil wrapper and is impressed that they vanish immediately in the solution. He swirls the liquid around, rips open the filter from one of his Marlboros, and uses the white fibers as a strainer through which to draw the liquid speedball into the syringe. He carefully places the loaded syringe between his teeth. He rolls up his sleeve, removes his belt with one hand, and takes a seat on the edge of the toilet. He wraps the belt tight around his right arm and hopes he can get a clean hit on one of the veins he watches come up. *There, I'll go there.*

The needle point feels sharp going in, which is good; it means he's got an unused needle. When he pulls back on the plunger a little stream of blood slithers up into the syringe, discoloring the slightly yellow liquid. He loosens the belt, careful not to dislodge the needle from the vein, takes a breath, and slowly pushes the liquid into his arm. He pulls the needle out and dabs with his finger at the drop of blood left behind on his arm. As he does this, he feels the freeze in his arm from the cocaine. His arm feels numb. Then it reaches his stomach and mouth. His heart races. He tastes the medicinal flavor just as the first wave of rushes is reaching his brain. His stomach heaves. His scalp tingles and he gets a little scared at first—the wave of sensation is stronger than usual. He fights the urge to vomit, the heroin kicks in and the nausea retreats as the warm, heroin heat replaces the heart-thumping freeze caused by the cocaine. His heart starts to slow down, or so it seems. A quiet, hollow siren rages in his head. The familiar beads of perspiration crowd each other on his forehead, and one drops onto his arm when he bends over to begin cleaning everything up. He puts away his paraphernalia, threads his belt into his pants, and sits down again. *Good stuff, very good,* he thinks as he nods for a second.

Back on Houston Street now, he decides to have a cup of espresso in a little coffee shop he comes upon. Sitting back at a table with a view of the street, he savors the thick hot coffee, lights a cigarette, and blows the smoke to the ceiling. *Nothing hurts,* he thinks. The lousy job that he needs to hold onto, the flak he catches from his wife, the fact that he is turning 40 and doesn't have anything to show for his life—none of it fazes him, but he still thinks about it. A spotty work history, no college, and rent that is three weeks late don't matter right now. He feels warm, loose, and sexy. Was the waitress's smile a flirt or was she smiling because she caught him nodding? Doesn't matter. He smiles back and thinks maybe he can buy his wife a gold-plated necklace instead of the real one. It will look just like the one she pointed out anyway.

And that was what he did (Fernandez, 1998, pp. 72–73).

High doses of opiates can lead to a comatose state, severely depressed breathing, and convulsions. The number of people admitted to hospital emergency rooms for treatment of heroin overdoses increased substantially during the 1990s. Between 3,000 and 4,000 people die from accidental overdoses of heroin in the United States each year (Leland, 1996).

Long-Term Consequences The effects of opiates on occupational performance and health depend in large part on the amount of drugs that the person takes. At high doses, people who are addicted to opiates become chronically lethargic and lose their motivation to remain productive. At low doses, some people who use opiates for an extended period of time can remain healthy and work productively in spite of their addiction. This functioning is, of course, dependent on the person's having easy and relatively inexpensive access to opiates. One possibility is being maintained by a physician on methadone, a synthetic opiate that is sometimes used therapeutically as an alternative to heroin.

People who are addicted to opiates become preoccupied with finding and using the drug, in order to experience the rush and to avoid withdrawal symptoms. Tolerance develops rather quickly, and the person's daily dose increases regularly until it eventually levels off and remains steady. Many of the severe health consequences of opiate use are the result of the lifestyle of the addict rather than the drug itself. The enormous expenses and difficulties associated with obtaining illegal opiates almost invariably consume all the person's resources. The person typically neglects housing, nutrition, and health care in the search for another fix. Heroin addicts are much more likely than other people in the general population to die from AIDS, violence, and suicide.

BARBITURATES AND BENZODIAZEPINES

The families of drugs known as barbiturates and benzodiazepines are also known informally as tranquilizers, hypnotics, and sedatives. *Tranquilizers* are used to decrease anxiety or agitation. *Hypnotics* are used to help people sleep. *Sedative* is a more general term that describes drugs that calm people or reduce excitement (other than the relief of anxiety). The **barbiturates,** such as phenobarbital (Nembutal) and amobarbital (Amytal), were used for a variety of purposes, including the treatment of chronic anxiety. The **benzodiazepines,** which include diazepam (Valium) and alprazolam (Xanax), have replaced the barbiturates in the treatment of anxiety disorders, in large part because of their lower potential for producing a lethal overdose.

Short-Term Effects Sedatives and hypnotics can lead to a state of intoxication that is identical to that associated with alcohol. It is characterized by impaired judgment, slowness of speech, lack of coordination, a narrowed range of attention, and disinhibition of sexual and aggressive impulses. Intravenous use of barbiturates can lead quickly to a pleasant, warm, drowsy feeling that is similar to the experience achieved when taking opiates. The benzodiazepines can sometimes lead to an increase in hostile and aggressive behavior. Some clinicians call this a "rage reaction" or aggressive dyscontrol.

Long-Term Consequences People who abruptly stop taking high doses of benzodiazepines may experience symptoms that are sometimes called a *discontinuance syndrome.* These symptoms can include a return—and, in some cases, a worsening—of the original anxiety symptoms, if the medication was being used to treat an anxiety disorder. The person may also develop new symptoms that are directly associated with drug withdrawal. These include irritability, paranoia, sleep disturbance, agitation, muscle tension, restlessness, and perceptual disturbances. Withdrawal symptoms are less likely to occur if the medication is discontinued gradually rather than abruptly.

CANNABIS

Marijuana and hashish are derived from the hemp plant, *Cannabis sativa.* The most common active ingredient in cannabis is a compound called delta-9-tetrahydro-cannabinol (THC). Because every part of the plant contains THC, cannabis can be prepared for consumption in several ways. **Marijuana** refers to the dried leaves and flowers, which can be smoked in a cigarette or pipe. It can also be baked in brownies and ingested orally. **Hashish** refers to the dried resin from the top of the female cannabis plant. It can be smoked or eaten after being baked in cookies or brownies.

Oral administration of cannabis material leads to slow and incomplete absorption. Therefore, the dose must be two or three times larger to achieve the same subjective effect as when it is smoked. Most of the drug is metabolized in the liver.

Short-Term Effects The subjective effects of marijuana are almost always pleasant. "Getting high" on marijuana refers to a pervasive sense of well-being and happiness. Laboratory research has shown that marijuana can have variable effects on a person's mood. Many people begin to feel happy, but some become anxious and paranoid. The mood of other people seems to be especially important. After smoking marijuana, a person's mood may become more easily influenced by how other people are behaving.

Cannabis intoxication is often accompanied by *temporal disintegration,* a condition in which people have trouble retaining and organizing information, even over relatively short periods of time. Conversations may become disjointed because the drug interferes with the people's ability to recall what they have said or planned to say. Lapses in attention and concentration problems are frequent.

Long-Term Consequences The issue of the addictive properties of cannabis remains controversial (Hall & Pacula, 2003; Onaivi, 2002). Some tolerance effects to THC have been observed in laboratory animals. Tolerance effects in humans remain ambiguous. Most evidence suggests that people do not develop tolerance to THC unless they are exposed to high doses over an extended period of time. Some people actually report that they become more sensitive (rather than less sensitive) to the effects of marijuana after repeated use. This phenomenon is called *reverse tolerance.* Although reverse tolerance has been reported casually by frequent users, it has not been demonstrated in a laboratory situation, where dosage levels can be carefully controlled.

Withdrawal symptoms are unlikely to develop among occasional smokers of marijuana. People who have been

exposed to continuous, high doses of THC may experience withdrawal symptoms, such as irritability, restlessness, and insomnia.

Prolonged heavy use of marijuana may lead to certain types of performance deficits on neuropsychological tests, especially those involving sustained attention, learning, and decision making (Pope & Yurgelun-Todd, 1996). These effects should be interpreted cautiously. Follow-up studies of adults who used cannabis over a period of several years did not find evidence of cognitive decline associated with the drug (Lyketsos et al., 1999).

HALLUCINOGENS AND RELATED DRUGS

Drugs that are called **hallucinogens** cause people to experience hallucinations. Although many other types of drugs can lead to hallucinations at toxic levels, hallucinogens cause hallucinations at relatively low doses. There are many different types of hallucinogens, and they have very different neurophysiological effects. The molecular structure of many hallucinogens is similar to the molecular structure of various neurotransmitters, such as serotonin and norepinephrine. The most common hallucinogen is a synthetic drug called *LSD* (D-lysergic acid diethylamide), which bears a strong chemical resemblance to serotonin. It achieves its effect by interacting with certain types of serotonin receptors in the brain. *Psilocybin* is another type of hallucinogen whose chemical structure resembles that of serotonin. It is found in different types of mushrooms, which grow primarily in the southern United States and Mexico. Mescaline is a type of hallucinogen that resembles norepinephrine. It is the active ingredient in a small, spineless cactus called peyote. Mescaline and psilocybin have been used in religious ceremonies by various Native American peoples for many centuries.

MDMA (methylene-dioxy-methamphetamine, also known as ecstasy) is one of several synthetic amphetamine derivates. It could be classified as a stimulant, but most texts list it as a type of hallucinogen (Julien, 2001). MDMA is also known as a "club drug" because it is popular among people who attend "raves" and dance clubs (LSD and methamphetamine are also known as club drugs). MDMA is usually taken as a tablet, but the powder form can be inhaled or injected. Within half an hour of ingesting MDMA orally, the person begins to experience an enhanced mood state and a feeling of well-being that often lasts several hours. Although it does not produce vivid hallucinations, MDMA does lead to changes in perceptual experiences, such as distortions in the sense of time and space, as well as increased sensory awareness. It also produces changes in blood pressure and can interfere with the body's ability to regulate its temperature.

MDMA is known as a "club drug" because it is popular among people who attend "raves" and dance clubs. It causes changes in perceptual experiences, such as distortions in the sense of time and space, as well as increased sensory awareness.

Phencyclidine (PCP) is another synthetic drug that is often classified with the hallucinogens, although its effects are very different than those associated with LSD and mescaline. It was originally developed as a painkiller. Small doses of PCP lead to relaxation, warmth, and numbness. At higher doses, PCP can induce psychotic behavior, including delusional thinking, catatonic motor behavior, manic excitement, and sudden mood changes. The drug is typically sold in a crystallized form that can be sprinkled on leaves, such as tobacco, marijuana, or parsley, and then smoked. Some people snort it or inject it after dissolving the crystals in water.

Short-Term Effects The effects of hallucinogenic drugs are difficult to study empirically because they are based primarily in subjective experience. They typically induce vivid, and occasionally spectacular, visual images. During the early phase of this drug experience, the images often take the form of colorful geometric patterns. The later phase is more likely to be filled with meaningful images of people, animals, and places. The images may change rapidly, and they sometimes follow an explosive pattern of movement.

Although these hallucinatory experiences are usually pleasant, they are occasionally frightening. "Bad trips" are a decidedly unpleasant experience that can lead to panic attacks and the fear of losing one's mind. People can usually be talked through this process by constantly reminding them that the experience is drug-induced and will be over soon.

Most hallucinogens are not particularly toxic. People do not die from taking an overdose of LSD, psilocybin, or mescaline. However, PCP is much more toxic. High doses can lead to coma, convulsions, respiratory arrest, and brain hemorrhage. MDMA (Ecstasy) can damage serotonin neurons on a permanent basis, and it has been associated with some fatalities (Gold, Tabrah, & Frost-Pineda, 2001).

Long-Term Consequences The use of hallucinogens follows a different pattern than that associated with most other drugs. Hallucinogens, with the possible exception of PCP, are used sporadically and on special occasions rather than continuously. If these drugs are taken repeatedly, within two or three days, their effects disappear. Most people do not increase their use of hallucinogens over time. People who stop taking hallucinogens after continued use do not experience problems; there seem to be no withdrawal symptoms associated with the hallucinogens that resemble serotonin and norepinephrine. The perceptual effects of hallucinogenic drugs almost always wear off after several hours. There are cases, however, in which these drugs have induced persistent psychotic behavior. Most experts interpret these examples as an indication that the drug

experience can trigger the onset of psychosis in people who were already vulnerable to that type of disorder. As genes involved in the predisposition toward psychosis are identified, it will become possible to test this hypothesis.

Some people who have taken hallucinogens experience *flashbacks*—brief visual aftereffects that can occur at unpredictable intervals long after the drug has been cleared from the person's body. Scientists do not understand the mechanisms that are responsible for flashbacks. Flashbacks may be more likely to occur when the person is under stress or after the person has used another drug, such as marijuana.

Diagnosis

The problems that we have reviewed indicate that substance dependence represents an extremely diverse set of problems. Everyone—clinicians and researchers, as well as drug abuser and their families—seems to recognize the existence of a serious psychological disorder. But does it have a core? What is the best way to define it? In the following pages we briefly review some of the ways in which alcoholism and drug abuse have been defined. We must begin with the recognition that alcoholism and other types of addictions have not always been viewed as medical conditions that require treatment (Walters, 1999).

BRIEF HISTORY OF LEGAL AND ILLEGAL SUBSTANCES

One of the most widely recognized facts about alcohol consumption is that drinking patterns vary tremendously from one culture to the next and, within the same culture, from one point in time to another. Public attitudes toward the consumption of alcohol have changed dramatically during the course of U.S. history. For example, heavy drinking was not generally considered to be a serious problem in colonial times (Levine, 1978). In fact, it seemed to be an integral part of daily life. The average amount of alcohol consumed per person each year was much higher in those days than it is today. A typical American in the eighteenth century drank approximately 4 gallons of alcohol a year; the corresponding figure for our own society is about 2.5 gallons (Fingarette, 1988). Drunkenness was not considered to be either socially deviant or symptomatic of medical illness.

Public attitudes toward alcohol changed dramatically in the United States during the first half of the nineteenth century. Members of the temperance movement preached against the consumption of alcohol in any form. Temperance workers ardently believed that anyone who drank alcohol would become a drunkard. Their arguments were largely moral and religious rather than medical or scientific, and many of their publications included essays on the personality weaknesses that were associated with such morally reprehensible behaviors (Okrent, 2010). The temperance movement was, in fact, able to persuade many thousands of people to abandon the consumption of alcohol.

The movement finally succeeded in banning the manufacture and sale of alcoholic beverages when Congress approved the Eighteenth Amendment to the Constitution in 1919. During the following years, known as the Prohibition era, the average consumption of alcohol fell substantially, and the incidence of associated medical illnesses, such as cirrhosis of the liver, also declined. Nevertheless, these laws were extremely difficult to enforce, and Prohibition was repealed in 1933.

During the Prohibition era in the United States (1922–1933), it was illegal to manufacture, transport, or sell alcoholic beverages. Nevertheless, alcohol continued to be widely available from illegal sources, and the law was eventually changed. Similar efforts to control access to addicting drugs have failed in other countries.

DSM-IV-TR

As we noted at the beginning of this chapter, DSM-IV-TR divides addictions into two categories: substance abuse and substance dependence, with the latter being the more severe and advanced form of disorder. This distinction is based, in part, on the recognition that many people who suffer serious impairment from substance abuse do not progress to the level of dependence (Bucholz, 1999). The manual lists 11 types of drugs that can lead to problems of abuse and dependence (refer to Table 11.2). Rather than including separate definitions of dependence and abuse for each class of substance, the manual provides one generic set of criteria for substance dependence and another for substance abuse. These criterion sets can be applied to any type of drug.

The DSM-IV-TR criteria for substance dependence are presented in Table 11.3. Tolerance and withdrawal are listed

TABLE 11.3 DSM-IV-TR Criteria for Substance Dependence

A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same 12-month period:

1. **Tolerance, as defined by either of the following:**
   a. A need for markedly increased amounts of the substance to achieve intoxication or desired effect.
   b. Markedly diminished effect with continued use of the same amount of the substance.
2. **Withdrawal, as manifested by either of the following:**
   a. The characteristic withdrawal syndrome for the substance (criteria sets for withdrawal are listed separately for specific substances).
   b. The same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms.
3. **The substance is often taken in larger amounts or over a longer period than was intended.**
4. **There is a persistent desire or unsuccessful efforts to cut down or control substance use.**
5. **A great deal of time is spent in activities necessary to obtain the substance (for example, visiting multiple doctors or driving long distances), use the substance (for example, chain-smoking), or recover from its effects.**
6. **Important social, occupational, or recreational activities are given up or reduced because of substance use.**
7. **The substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (for example, current cocaine use despite recognition of cocaine-induced depression, or continued drinking despite recognition that an ulcer was made worse by alcohol consumption).**

along with five other problems that describe a pattern of compulsive use and loss of control. The person has to exhibit at least three of the seven criteria for a diagnosis of substance dependence to be made. Tolerance and withdrawal are not required for the person to meet this definition of dependence. Their importance is recognized with a subtype designation. If there is evidence of either tolerance or withdrawal (or both), the additional specification of *physiological dependence* is made. Symptoms of withdrawal seem to be more important than symptoms of tolerance in this regard. People with a history of physiological dependence report more severe drug-related problems, greater intensity of exposure to drugs, and more comorbid conditions such as anxiety and depression (Schuckit, 2010).

This approach to the definition of substance dependence is convenient because it points to a unified view of addiction. However, it also has some disadvantages. Perhaps most important is the fact that the use of a single definition of dependence may conceal differences between the kinds of problems that are associated with various classes of drugs (Budney, 2007; Frances, First, & Pincus, 1995). For example, dependence on opiates almost always involves physiological symptoms of tolerance and withdrawal, whereas dependence on cannabis or hallucinogens seldom does.

Where is the boundary between substance abuse and recreational drug use?

Substance abuse is defined in terms of harmful consequences that appear in the absence of tolerance, withdrawal, or a pattern of compulsive use (dependence). The DSM-IV-TR definition of substance abuse is presented in Table 11.4. One difficult issue in defining this condition involves the identification of a boundary between substance abuse and the recreational use of drugs. The diagnostic manual emphasizes the terms *recurrent* and *maladaptive pattern* for this purpose. The problem must be persistent before this diagnosis would be considered. Someone involved in a single drug-related incident would not meet the criteria for this disorder, regardless of how serious the incident might have been (Frances, First, & Pincus, 1995).

Important questions have been raised about the validity of the DSM-IV-TR substance abuse category, especially with regard to alcoholism (Martin, Chung, & Langenbucher, 2008). Many people who receive a diagnosis of alcohol abuse do so on the basis of a single symptom—hazardous use—which usually involves driving while intoxicated. This is certainly a grave problem with enormous negative consequences, but it is not clear whether this form of maladaptive behavior should be considered a mental disorder if it occurs in the absence of other symptoms of alcohol abuse. Perhaps the drunk driver would be better viewed as a person who has persistently chosen, for whatever reason, to engage in reckless and illegal behavior that ignores the safety of other people.

The workgroup that is preparing the next definition of substance use disorders for DSM-V may collapse substance dependence and substance abuse into a single disorder with a continuous range of severity (Helzer et al., 2008; Martin et al., 2008). In the case of alcohol, this disorder would be called "alcohol use disorder." The specific features included in the list of criteria would be essentially a combination of those

TABLE 11.4 DSM-IV-TR Criteria for Substance Abuse

A. A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by one (or more) of the following, occurring within a 12-month period:

1. Recurrent substance use resulting in a failure to fulfill major role obligations at work, school, or home.
2. Recurrent substance use in situations in which it is physically hazardous.
3. Recurrent substance-related legal problems.
4. Continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance.

B. The symptoms have never met the criteria of substance dependence for this class of substance.

currently used to identify dependence and abuse, with at least two of the total features being required to reach threshold for a diagnosis. The rationale for this change is based on research evidence showing that dependence and abuse are not clearly distinct forms of disorder (Harford, Yi, & Grant, 2010).

COURSE AND OUTCOME

It is impossible to specify a typical course for substance dependence, especially alcoholism. Age of onset varies widely, ranging from childhood and early adolescence throughout the life span. Although we can roughly identify stages that intervene between initial exposure to a drug and the eventual onset of tolerance and dependence, the timing with which a person moves through these phases can vary enormously. The best available information regarding the course of substance use disorders comes from the study of alcoholism. The specific course of this problem varies considerably from one person to the next. The only thing that seems to be certain is that periods of heavy use alternate with periods of relative abstinence, however short-lived they may be (Schuckit & Smith, 2011).

One influential study regarding the natural history of alcoholism examined the lives of 456 inner-city adolescents from Boston and 268 former undergraduate students from Harvard University (Vaillant, 2003). Initial information was collected in 1940, when the participants were adolescents. Follow-up information was collected every other year by questionnaire and every fifth year by physical examination. The college group has been followed until 70 years of age, and the core city group has been followed to age 60. At some point during their lives, 21 percent of the college men and 35 percent of the core city men met diagnostic criteria for alcohol abuse, which the investigators defined as the presence of four or more problems in such areas as employer complaints, marital and family difficulties, medical complications, and legal problems. As expected, the mortality rate was higher among men who abused alcohol than among those who did not. Heart disease and cancer were twice as common among the alcohol abusers, perhaps in part because they were also more likely to be heavy cigarette smokers.

Most of the alcoholic men went through repeated cycles of abstinence followed by relapse. The life course of alcohol abuse could be charted most clearly for 121 of the core city men who abused alcohol and remained in the study until age 60 and 46 college men who abused alcohol and remained in the study until age 70. These data are illustrated in Figure 11.2. In the graphs in Figure 11.2, abstinence is defined as less than one drink per month for more than a year. Social drinking refers to problem-free drinking for 10 years or more. Controlled drinking is more than one drink per month for at least two years with no reported problem. The main differences between the groups were that the core city men began abusing alcohol at an earlier age, and they were also more likely than the college men eventually to achieve stable abstinence. The average age of onset of alcohol abuse was 40 years for the college men and 29 years for the core city men.

Many men spent the previous 20 years alternating between periods of controlled drinking and alcohol abuse. The proportion of men who continued to abuse alcohol went down after the age of 40. The proportion of alcoholic men in both groups who became completely abstinent went up slowly, but consistently during the follow-up period. The longer a man remained abstinent, the greater the probability that he would continue to be abstinent. These data indicate that relapse to alcohol abuse was unlikely among men who were able to remain abstinent for at least six years.

Many important questions remain to be answered about the relapse process. Is there a "safe point" that separates a period of high risk for relapse from a period of more stable change? Data from the study of men in Boston suggest that the six-year mark may be important for men who abuse alcohol. Will this suggestion be replicated in other studies? And does it generalize to other drugs? Do relapse rates stabilize over time? Is an addicted person more likely to succeed on a later attempt to quit than on an early attempt? Answers to these questions will be useful in the development of more effective treatment programs.

OTHER DISORDERS COMMONLY ASSOCIATED WITH ADDICTIONS

People with substance use disorders often exhibit other forms of mental disorder as well. Most prominent among these are antisocial personality disorder (ASPD), mood disorders, and anxiety disorders. Conduct disorder (the childhood manifestation of ASPD) is strongly related to concurrent alcohol use in adolescence and the subsequent development of alcohol

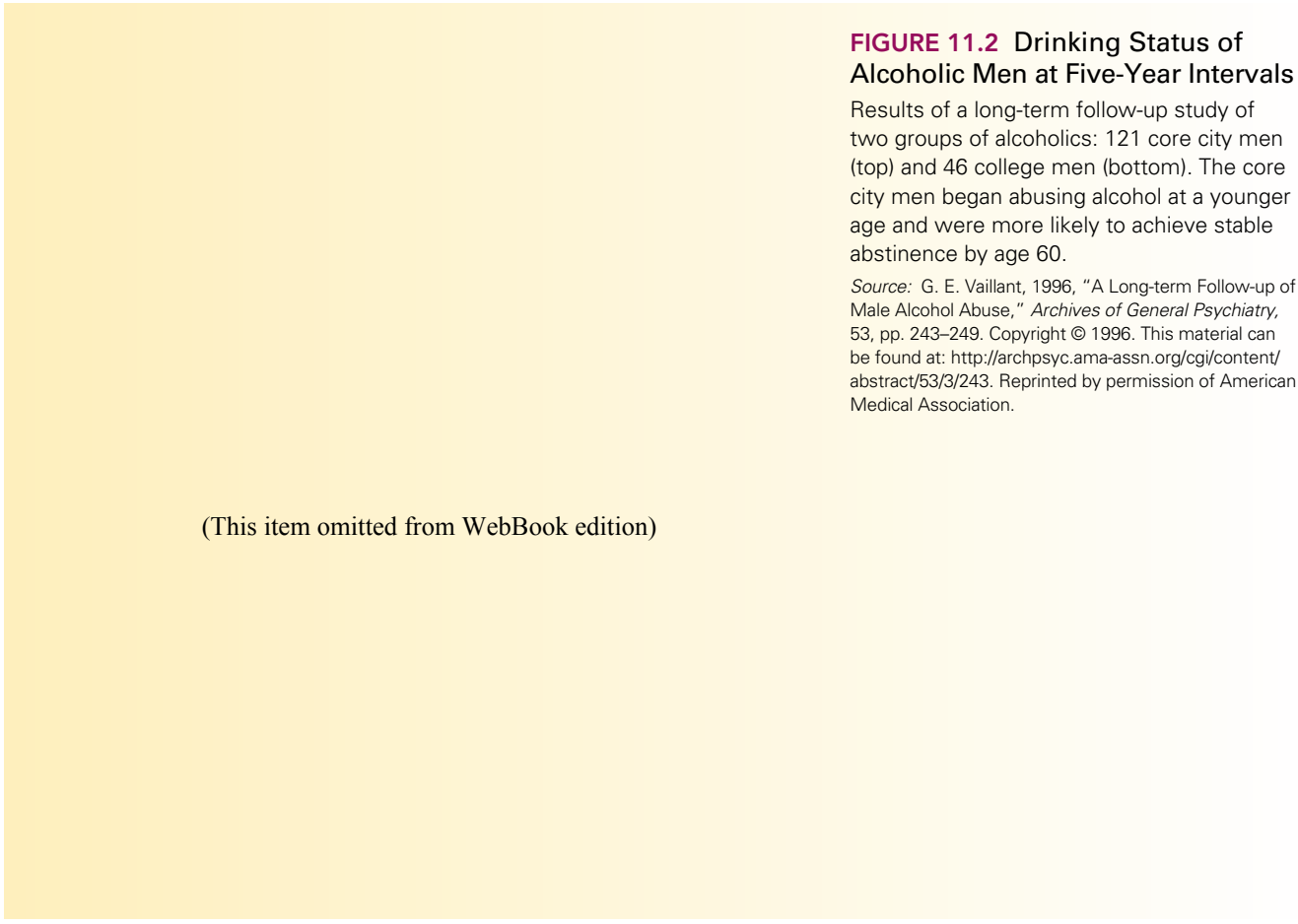


FIGURE 11.2 Drinking Status of Alcoholic Men at Five-Year Intervals

Results of a long-term follow-up study of two groups of alcoholics: 121 core city men (top) and 46 college men (bottom). The core city men began abusing alcohol at a younger age and were more likely to achieve stable abstinence by age 60.

Source: G. E. Vaillant, 1996, "A Long-term Follow-up of Male Alcohol Abuse," *Archives of General Psychiatry,* 53, pp. 243–249. Copyright © 1996. This material can be found at: http://archpsyc.ama-assn.org/cgi/content/abstract/53/3/243. Reprinted by permission of American Medical Association.

dependence (McGue & Iacono, 2008). ASPD and alcohol/drug dependence frequently co-occur, and there is evidence to suggest that they represent alternative manifestations of a general predisposition toward behavioral disinhibition (Kendler et al., 2003).

The complexity of the association between substance use disorders and mood/anxiety disorders makes them difficult to untangle (Grant et al., 2006). In some cases, prolonged heavy drinking or use of psychoactive drugs can result in feelings of depression and anxiety. The more the person drinks or uses drugs, the more guilty the person feels about his or her inability to control the problem. In addition, continued use of alcohol and drugs often leads to greater conflict with family members, coworkers, and other people. Sometimes the depression and anxiety precede the onset of the substance use problem. In fact, some people seem to use alcohol and drugs initially in a futile attempt to self-medicate for these other conditions. Ultimately, the drugs make things worse.

Frequency

Drug-related problems are found in most countries. There are interesting variations, however, in patterns of use for specific types of drugs. The use of specific drugs is determined, in part, by their availability. For example, opium is used most heavily in Southeast Asia and in some Middle Eastern countries, where the opium poppy is cultivated. Cocaine is used frequently in certain countries of South America where coca trees grow; it is also imported into North America, particularly the United States. Use of cannabis is widespread around the world, in part because the plants can grow in many different climates. In contrast, in Japan, where the amount of land available for cultivation is severely limited, the largest drug problem involves amphetamine, a synthetic drug.

The fact that people in some regions are frequent drug users does not necessarily imply that a particular population will have a high rate of substance dependence. Culture shapes people's choices about the use of drugs and the ways in which they are used. It influences such factors as the amount of a drug that is typically ingested, the route of administration, and the person's beliefs about drug effects (Room, 2007). These considerations, in turn, influence the probability that serious problems will develop. Consider, for example, the Indians of South America who produce coca for market. They have traditionally used the leaves as medicines and in religious ceremonies. They also roll the leaves into a ball that can be tucked in the cheek and sucked for an extended period of time. This form of use relieves cold, hunger, and thirst. It does not produce the severe dependence problems that are associated with the use of refined cocaine, a much more potent drug that can be sniffed or injected.

When we consider the frequency of drug addiction, we must keep in mind the distinction between using a drug and becoming addicted to it. Many people who use drugs do not

Actor Charlie Sheen has received considerable media attention for alcohol and drug dependence, as well as related marital problems and allegations of domestic violence. His struggles provide a sad, public illustration of the complex relations among substance use disorders and other mental health problems.

become dependent on them. Nevertheless, people have to use the drug before they can become dependent, and the age at which they *begin* to use drugs is an important risk factor. For example, the prevalence rate for alcoholism among males who began drinking alcohol before the age of 14 is double that found among males who began drinking at age 18 (McGue et al., 2001). The same pattern is found among women; those who begin to use alcohol at an earlier age have a much higher risk of becoming dependent. It is not clear whether earlier initiation leads directly to increased risk of alcohol dependence or whether people who are already predisposed toward the development of drinking problems simply start using earlier.

Most people who occasionally use alcohol and illicit drugs do not become addicted. Dependence almost always develops slowly after extended exposure to a drug. The average time between initial use of illicit drugs and the onset of symptoms of dependence is between two and three years (Anthony & Helzer, 1991). The distinction between people who eventually become addicted and those who use drugs without becoming addicted is an important consideration in the study of psychopathology.

PREVALENCE OF ALCOHOL ABUSE AND DEPENDENCE

Approximately two out of every three males in Western countries drink alcohol regularly, at least on a social basis; less than 25 percent abstain from drinking completely. Among all men and women who have ever used alcohol, roughly 20 percent will develop serious problems—abuse or dependence—at some point in their lives as a consequence of prolonged alcohol consumption (Anthony, Warner, & Kessler, 1994).

The National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) collected information on substance use disorders and related mental health problems in a nationally representative sample of more than 43,000 adults (Grant et al., 2006). This study provides the most recent, comprehensive evidence regarding the prevalence of alcoholism in the United States. Results from the NESARC study, summarized in Figure 11.3, indicate a lifetime prevalence rate of 30 percent for some form of alcohol use disorder (18 percent for alcohol abuse and 12 percent for alcohol dependence). Alcohol-related disorders are clearly among the most common forms of mental disorder in the United States. These problems most often went untreated; only 24 percent of the men and women who were assigned a diagnosis of alcohol dependence had ever received treatment for these problems.

Gender Differences Approximately 60 percent of women in the United States drink alcohol at least occasionally, but, in comparison to men, fewer develop alcoholism. Among people who chronically abuse or become dependent upon alcohol, men outnumber women by a ratio of approximately two to one (see Figure 11.3). This disparity is narrower today than it was 50 years ago, especially among younger people. Although

(This item omitted from WebBook edition)

FIGURE 11.3 Gender Differences in Substance Use Disorders

Lifetime prevalence of substance use disorders in the United States.

Source: From "Prevalence, Correlates, Disabilities, and Comorbidities of DSM-IV-TR Drug Abuse and Dependence in the United States" by W. M. Compton, et al., *Archives of General Psychiatry,* 64 (2007), pp. 566–576. Copyright © 2007. This material can be found at: http://archpsyc.ama-assn.org/cgi/content/full/64/5/566. Reprinted by permission of American Medical Association.

the rate of alcoholism among younger women has increased, prevalence is still higher in men, and the rates do not seem likely to converge (Grant & Weissman, 2007; Grucza et al., 2008). Persistent differences can probably be attributed to social and biological variables. American culture traditionally has held a negative view of intoxication among women. Social disapproval probably explains why women are more likely than men to drink in the privacy of their own homes, either alone or with another person. Women, therefore, may be less likely than men to drink heavily because the range of situations in which they are expected to drink, or in which they can drink without eliciting social disapproval, is narrower.

Biologically, there are also important gender differences in alcohol metabolism. A single standard dose of alcohol, measured in proportion to total body weight, will produce a higher peak blood alcohol level in women than in men. One explanation for this difference lies in the fact that men have a higher average content of body water than women do. A standard dose of alcohol will be less diluted in women because alcohol is distributed in total body water. This may help to explain the fact that women who drink heavily for many years are more vulnerable to liver disorders than are male drinkers.

When it comes to abusing alcohol, men outnumber women 2 to 1, though this proportion may be lower than it was 20 years ago.

PREVALENCE OF DRUG AND NICOTINE DEPENDENCE

The National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) also reported the frequency of problems associated with other kinds of drugs (Compton et al., 2007). The combined lifetime prevalence for abuse of or dependence on any type of controlled substance (those that are illegal or available only by prescription) was 10.3 percent. This is approximately one-third the rate for alcohol abuse and dependence. As in the case of alcohol-related disorders, drug abuse and dependence were significantly more common among men than women. Lifetime prevalence rates generated by this study for substance use disorders associated with specific types of drugs are listed in Figure 11.4.

The lifetime prevalence of nicotine dependence was reported to be 24 percent in the National Comorbidity Survey (Kessler et al., 1994). The percentage of adults in the United States who smoke tobacco has actually declined since 1964, when the U.S. Surgeon General's Report announced it had found a definite link between smoking and cancer and other diseases (see Critical Thinking Matters). The rate of decline has been greatest among men, who traditionally have smoked more than women. Among people between the ages of 18 and 25, however, smoking rates increased during the 1990s (Chaloupka, 2005). Furthermore, although overall tobacco consumption has declined in industrialized countries, it has increased dramatically in the developing countries, where people may be less educated about the health risks associated with smoking (McKim, 2000).

RISK FOR ADDICTION ACROSS THE LIFE SPAN

Older people do not drink as much alcohol as younger people. The proportion of people who abstain from drinking alcohol is only 22 percent for people in their thirties, goes up to 47 percent for people in their sixties, and is approximately 80 percent for people over 80 years of age. Prevalence rates for alcohol dependence are highest among young adults and lowest among the elderly (Hasin et al., 2007). Most elderly alcohol abusers are people who have had drinking problems for many years.

The use of illegal drugs is relatively infrequent among the elderly, but there is a problem associated with their abuse of, and dependence on, prescription drugs and over-the-counter

(This item omitted from WebBook edition)

FIGURE 11.4 Drug Abuse and Dependence

Lifetime prevalence of abuse and dependence associated with specific types of drugs in the United States.

Source: From "Prevalence, Correlates, Disabilities, and Comorbidities of DSM-IV-TR Drug Abuse and Dependence in the United States" by W. M. Compton, et al., *Archives of General Psychiatry,* 64 (2007), pp. 566–576. Copyright © 2007. This material can be found at: http://archpsyc.ama-assn.org/cgi/content/full/64/5/566. Reprinted by permission of American Medical Association.

Critical Thinking Matters

SHOULD TOBACCO PRODUCTS BE ILLEGAL?

In 1996, the U.S. Food and Drug Administration (FDA) issued a regulation prohibiting the sale and distribution of tobacco products to children and adolescents. They remain legally available to adults. Previous efforts to limit smoking had focused more narrowly on restricting smoking in public places, eliminating cigarette advertisements on television, and increasing sales taxes. The new rule asserted that, as a drug, nicotine should be controlled by the government. The decisions behind this regulation raise a number of critical thinking issues with regard to substance use disorders. How does the FDA decide whether a product is a drug? Should the government control people's access to addicting drugs? If so, what is the best way to control access?

The FDA conducted an extensive investigation to examine the effects of tobacco products and to determine whether they were designed by their manufacturers to deliver nicotine to consumers. Independent research studies as well as documents from the tobacco industry's own laboratories pointed to the conclusion that *nicotine is addictive* (Dreyfuss, 1996). People who use tobacco products clearly develop symptoms of dependence, including tolerance, withdrawal, and a pattern of compulsive use. In fact, nicotine is one of the most addicting drugs, viewed in terms of the high proportion of people who become dependent if they use the drug for some period of time.

After officially recognizing that nicotine is an addicting drug, the FDA could have banned tobacco products entirely (because they are not safe for human consumption). Another option would have been to require a complete elimination of nicotine from cigarettes. The FDA did not consider these options to be practical or politically viable. Because so many adults are already addicted to nicotine, extensive black markets would spring up immediately, similar to those involved with other illegal drugs. An outright ban on nicotine would fail, just as efforts to ban other drugs have failed (Husak, 2002; MacCoun, Reuter, & Wolf, 2001).

What evidence supports the conclusion that nicotine is an addicting drug?

The FDA decided instead to approach the nicotine problem by invoking its authority to regulate medical devices (and treating cigarettes as a type of drug-delivery system). The tobacco regulations imposed by the FDA are *prevention* efforts designed to break the cycle of addiction to nicotine. They prohibit the sale of tobacco products to anyone under 18 years old and also severely restrict advertising (Cooper, 1994). Nicotine addiction almost always begins during adolescence. The FDA regulations are intended to reduce the rate at which young people are recruited to become new smokers and to minimize future health casualties from tobacco use.

This policy represents a moderate and thoughtful approach to the problem of nicotine dependence. It is a compromise between two extreme alternatives: allowing completely open access to a dangerous drug or attempting to ban it completely. Preliminary evidence suggests that the FDA regulations regarding tobacco products have been modestly successful. Between 1997 and 2004, prevalence rates for current smoking among adults in the United States dropped from 25 percent to 21 percent (Schiller et al., 2005). Public policy will not be able to eliminate completely the use of harmful drugs by adults in our society. It can reduce the risk of dependence, however, by minimizing their use at an early age.

medications, especially hypnotics, sedatives, anxiolytics, and painkillers. The elderly use more legal drugs than do people in any other age group. One estimate suggested that 25 percent of all people over the age of 55 use psychoactive drugs of one kind or another (Beynon, McVeigh, & Roe, 2007). The risk for substance dependence among the elderly is increased by frequent use of multiple psychoactive drugs combined with enhanced sensitivity to drug toxicity (caused by slowed metabolic breakdown of alcohol and other drugs).

The following case illustrates several issues that are associated with substance use disorders among the elderly, including the abuse of alcohol together with abuse of prescription medications, the presence of prominent symptoms of anxiety and depression, and the tendency to deny the extent of their use or abuse of drugs.

BRIEF CASE STUDY

Ms. E's Drinking

Ms. E is an 80-year-old woman who was brought in for an evaluation by her daughters because they noticed depressive symptoms, appetite disturbance, and memory deficits. She denied all problems related to her daughters' concerns. She had a depressed affect, mild psychomotor agitation, and decrements of recent and remote memory. She was disoriented to time. She verbalized statements of guilt and self-deprecation. She denied ever drinking alcohol, which was corroborated by the daughter with whom she lived but was refuted by her other daughter, who stated that Ms. E drank one or two glasses of brandy almost

every day. She had been taking various barbiturates for "nerves" for over 30 years. The dosage she ingested gradually increased over the years, and she frequently took more medications than were prescribed. Because it was unclear if her symptoms were related to her barbiturate use, she reluctantly agreed to be slowly and gradually detoxified. She refused a dementia work-up. Once detoxification was complete, her affect and appetite were improved, but her cognitive deficits were unchanged. Several months later, she and her family dropped out of treatment. She was reportedly drinking brandy, wine, and "hard liquor" every afternoon and evening, with her hired caregiver mixing the drinks (Solomon et al., 1993).

Diagnostic criteria for substance dependence and abuse are sometimes difficult to apply to the elderly, primarily because drug use has somewhat different consequences in their lives. Tolerance to many drugs is reduced among the elderly, and the symptoms of withdrawal may be more severe and prolonged. They are less likely to suffer occupational impairment because they are less frequently employed than younger people. The probability of social impairment may be reduced because elderly people are more likely to live apart from their families.

In what ways are drug problems different among the elderly?

Causes

Our discussion of causal factors will focus primarily on alcohol dependence and abuse. We have chosen this approach because clinical scientists know more about alcohol and its abuse than about any of the other drugs. Twin studies also suggest that alcohol dependence and other forms of drug dependence share a common etiology (Kendler & Prescott, 2006). Research on alcohol abuse illustrates the factors that are also important in the etiology of other forms of substance dependence.

Most contemporary investigators approach the development of alcoholism in terms of multiple systems (Sher, Grekin, & Williams, 2005). Biological factors obviously play an important role. The addicting properties of certain drugs are crucial: People become addicted to drugs like heroin, nicotine, and alcohol, but they do not become addicted to drugs like the antidepressants or to food additives like Nutrasweet. We must, therefore, understand how addicting drugs affect the brain in order to understand the process of dependence. At the same time, we need to understand the social and cultural factors that influence how and under what circumstances an individual first acquires and uses drugs. Our expectations about the effects of drugs are shaped by our parents, our peers, and the media. These are also important etiological considerations.

The etiology of alcoholism is best viewed within a developmental framework that views the problem in terms of various stages: (1) initiation and continuation, (2) escalation and transition to abuse, and (3) development of tolerance and withdrawal (Leonard et al., 2000; Tarter, Vanyukov, & Kirisci, 2008). In the following pages we review some of the social, psychological, and biological factors that explain why people begin to drink, how their drinking behaviors are reinforced, and how they develop tolerance after prolonged exposure.

SOCIAL FACTORS

People who don't drink obviously won't develop alcoholism, and culture can influence that decision. Some cultures prohibit or actively discourage alcohol consumption. Many Muslims, for example, believe that drinking alcohol is sinful. Other religions encourage the use of small amounts of alcohol in religious ceremonies—such as Jewish people drinking wine at Passover seders—while also showing disdain for those who drink to the point of intoxication (Johnson, 2007). This type of cultural constraint can decrease rates of substance dependence. In one large epidemiological study, for example, Jews had significantly lower rates of alcohol abuse than Catholics and Protestants (Yeung & Greenwald, 1992).

Among those young people who choose to drink alcohol (or smoke cigarettes, or consume other addictive substances), which ones will eventually develop problems? The development of drug dependence requires continued use, and it is influenced by the manner in which the drug is consumed. In other words, with regard to alcohol, will the person's initial reaction to the drug be pleasant, or will he or she become sick and avoid alcoholic beverages in the future? If the person continues drinking, will he or she choose strong or weak drinks, with or without food, with others or alone, and so on?

Several studies have examined social factors that predict substance use among adolescents. Initial experimentation with drugs is most likely to occur among those individuals who are rebellious and extroverted and whose parents and peers model or encourage use (Chassin et al., 2003). The relative influence of parents and friends varies according to the gender and age of the adolescent as well as the drug in question.

The circumstances in which an adolescent is initially exposed to alcohol can influence the person's pattern of drinking. Drinking small amounts of wine with meals or during religious ceremonies may be less likely to lead to alcohol dependence than the sporadic consumption of hard liquor for the purpose of becoming intoxicated.

Parents can influence their children's drinking behaviors in many ways. They can serve as models for using drugs to cope with stressful circumstances. They may also help promote attitudes and expectations regarding the benefits of drug consumption, or they may simply provide access to licit or illicit drugs (Kirisci et al., 2007). Adolescents with alcoholic parents are more likely to drink alcohol than those whose parents do not abuse alcohol. This increased risk seems to be due to several factors, including the fact that alcoholic parents monitor their children's behavior less closely, thereby providing more opportunities for illicit drinking. Parental monitoring and discipline have an important impact on adolescent substance use; higher parental monitoring is associated with reduced risk of tobacco and alcohol use (Latendresse et al., 2008).

The level of negative affect is also relatively high in the families of alcoholic parents. This unpleasant emotional climate, coupled with reduced parental monitoring, increases the probability that an adolescent will affiliate with peers who use drugs (Chassin & Handley, 2006). Peer and sibling substance use are robust predictors of adolescent alcohol and drug use, even more than parental alcohol use. The impact of friends' alcohol use is greater among adolescent girls than adolescent boys.

BIOLOGICAL FACTORS

Initial physiological reactions to alcohol can have a dramatic negative influence on a person's early drinking experiences. For example, millions of people are unable to tolerate even small amounts of alcohol. These people develop flushed skin, sometimes after only a single drink. They may also feel nauseated, and some experience an abnormal heartbeat. This phenomenon is most common among people of Asian ancestry and may affect 30 to 50 percent of this population. The adverse reaction is due to genetic variants in the ADH and ALDH genes, which are involved in the metabolism of alcohol, and are much more common in Asian populations than in other races (Dick & Foroud, 2003). Not coincidentally, the prevalence of alcoholism is unusually low among Asian populations. Research studies indicate a link between these two phenomena. For example, Japanese Americans who experience the fast-flushing response tend to drink less than those who do not flush (Chen & Yin, 2008). The basic evidence suggests that in addition to looking for factors that make some individuals especially vulnerable to the addicting effects of alcohol, it may also be important to identify protective factors that reduce the probability of substance dependence.

What are the most important risk factors for alcoholism?

A person's initial use of addictive drugs is obviously one important step toward the development of substance dependence, but the fact remains that most people who drink alcohol do not develop alcoholism. What accounts for the next important phase of the disorder? Why do some people abuse the drug while others do not? In the following pages we outline several additional biological variables. We begin by examining genetic factors, and then we consider the neurochemical effects of the drugs themselves.

Genetics of Alcoholism An extensive literature attests to the fact that patterns of alcohol consumption, as well as psychological and social problems associated with alcohol abuse, tend to run in families. The lifetime prevalence of alcoholism among the parents, siblings, and children of people with alcoholism is at least three to five times higher than the rate in the general population (MacKillop, McGeary, & Ray, 2010). Of course, this elevated risk among first-degree relatives could reflect the influence of either genetic or environmental factors, because families share both types of influences. Therefore, we must look to the results of twin and adoption studies in an effort to disentangle these variables.

Several twin studies have examined twin concordance rates when the proband meets diagnostic criteria for substance dependence. Concordance rates are higher among MZ than among DZ twin pairs. For example, one study analyzed data from a large sample of twins in Australia. The investigators found concordance rates for alcohol dependence of 56 percent in male MZ twins and 33 percent in male DZ twins (Heath et al., 1997). Corresponding figures for MZ and DZ female twin pairs were 30 percent and 17 percent, respectively. Differences between MZ and DZ concordance rates were significant for both genders. The fact that concordance rates were higher for men than for women reflects the much higher prevalence rate for alcoholism among men. Heritability estimates were the same for both men and women, with approximately two-thirds of the variance in risk for alcoholism being produced by genetic factors.

The strategy followed in an adoption study (see Chapter 2) allows the investigator to separate relatively clearly the influence of genetic and environmental factors. Probands in this type of study are individuals who meet two criteria: (1) They had a biological parent who was alcoholic, and (2) they were adopted from their biological parents at an early age and raised by adoptive parents. Investigators then locate these individuals when they have become adults and determine the frequency of alcoholism as a function of both biological and environmental background. The results of adoption studies are consistent with the data from twin studies and point toward the influence of genetic factors in the etiology of alcohol abuse and dependence (Agrawal & Lynskey, 2008). The offspring of alcoholic parents who are reared by nonalcoholic adoptive parents are more likely than people in the general population to develop drinking problems of their own. Thus, the familial nature of alcoholism is at least partially determined by

genes. Being reared by an alcoholic parent, in the absence of other etiological factors, does not appear to be a critical consideration in the development of the disorder.

What exactly is inherited as the predisposition toward alcohol dependence? Some of the genes that influence the risk of developing alcohol dependence are genes involved in the metabolism of alcohol, such as the ADH and ALDH genes (discussed earlier and related to the skin flushing response). Other genes that alter the risk for alcohol dependence may be genes involved in personality traits (Dick, 2007; Spanagel et al., 2010). For example, to the extent that genes influence novelty seeking and sensation seeking, these genes may also increase the person's risk for alcohol dependence because the person is more likely to participate in dangerous patterns of consumption (such as drinking several shots of liquor in rapid succession rather than sipping beer or wine).

Neuroanatomy and Neurochemistry All of the addicting drugs produce changes in the chemical processes by which messages are transmitted in the brain, including systems that involve catecholamines (for example, dopamine, norepinephrine, and serotonin), as well as the neuropeptides. In the following sections, we will outline some of the ways in which psychoactive drugs influence neural transmission and the areas of the brain in which these effects are most pronounced.

Dopamine and Reward Pathways Scientists who study the biological basis of addiction have devoted a considerable amount of their attention to understanding the rewarding or reinforcing properties of drugs (Koob, 2006; Self & Tamminga, 2004). People may become dependent on psychoactive drugs because they stimulate areas of the brain that are known as "reward pathways" (see Figure 11.5). One primary circuit in this pathway is the medial forebrain bundle, which connects the ventral tegmental area to the nucleus accumbens. Connections from these structures to the frontal and prefrontal cortex as well as areas of the limbic system, such as the amygdala, also moderate the influence of reward. For many years, scientists have known electrical stimulation of the medial forebrain bundle can serve as a powerful source of positive reinforcement for animals as they perform an operant learning task. Natural rewards, such as food and sex, increase dopamine levels in certain crucial sections of this pathway, which is also known as the *mesolimbic dopamine pathway*.

Drugs of abuse have a dramatic effect on brain reward pathways. Some points at which different drugs influence the dopamine pathway between the ventral tegmental area and the nucleus accumbens are illustrated in Figure 11.6. For example, stimulants such as amphetamine and cocaine affect reward pathways by inhibiting the reuptake of dopamine into nerve terminals. Brain imaging studies with human participants have found that the administration of cocaine increases dopamine concentrations in limbic areas of the brain as well as the medial prefrontal cortex (Tomkins & Sellers, 2001). Furthermore, when people who are dependent on cocaine are exposed to cues that have previously signaled drug use, their medial prefrontal cortex becomes activated, suggesting that this area of the brain is involved in feelings of drug craving.

The effects of alcohol on reward pathways in the brain are more complex and less clearly understood than the effects of many other drugs (Durazzo et al., 2010). Alcohol clearly affects several different types of neurotransmitters. It may stimulate the

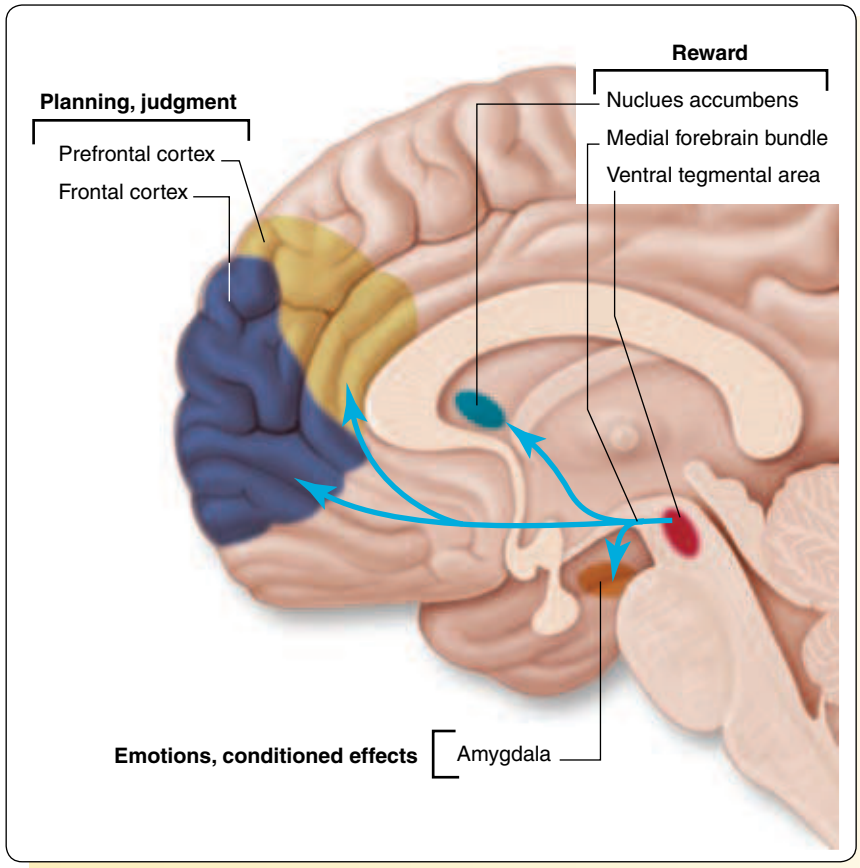


FIGURE 11.5 Reward Pathways in the Brain

The limbic dopamine reward pathways include connections from the ventral tegmental area to the nucleus accumbens and the frontal cortex.

Source: After D. M. Tomkins and E. M. Sellers, 2001, "Addiction and the Brain: The Role of Neurotransmitters in the Cause and Treatment of Drug Dependence," *Canadian Medical Association Journal, 164,* pp. 817–821.

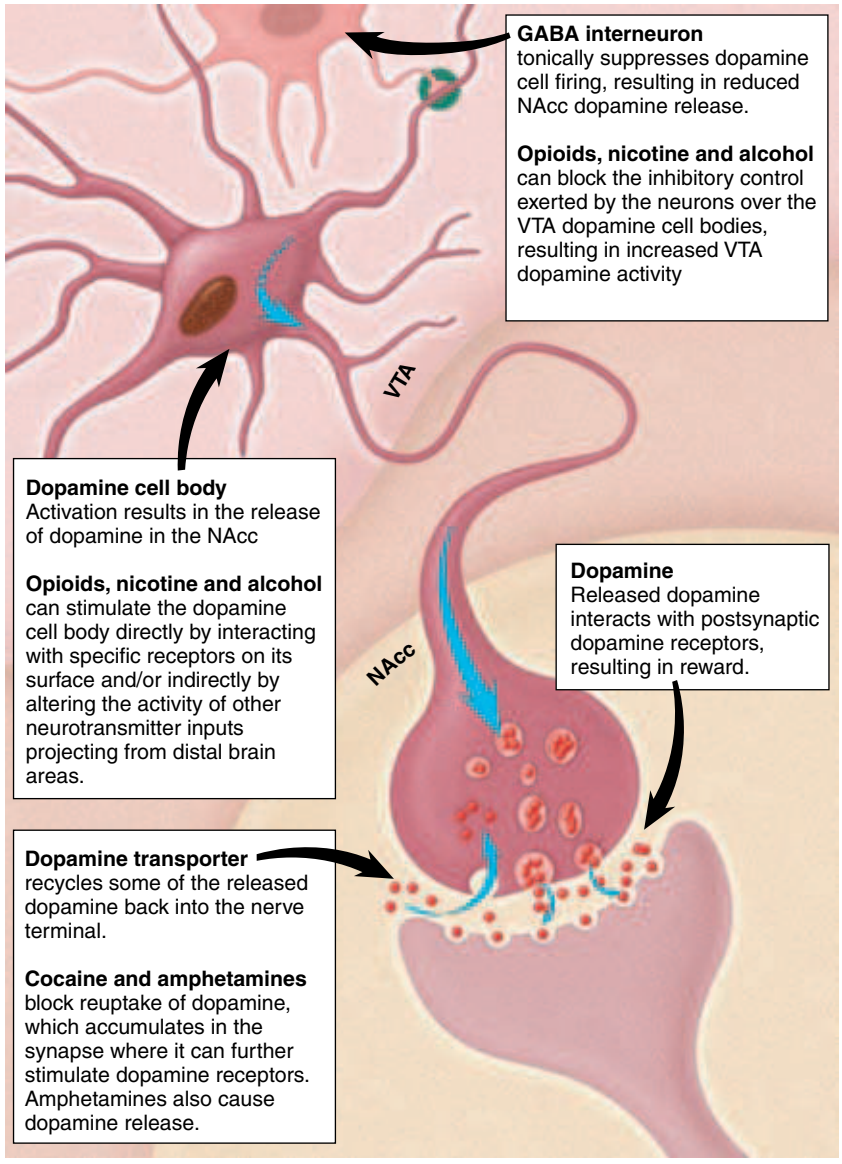


FIGURE 11.6 Neurochemical Mechanisms of Drug Action

Effects of psychoactive drugs on dopamine activity in reward pathway from the ventral tegmental area (VTA) to the nucleus accumbens (NAcc).

Source: After D. M. Tomkins and E. M. Sellers, 2001, "Addiction and the Brain: The Role of Neurotransmitters in the Cause and Treatment of Drug Dependence," *Canadian Medical Association Journal, 164,* pp. 817–821.

mesolimbic dopamine pathway directly, or it may act indirectly by decreasing the activity of GABA neurons (which normally inhibit dopamine neurons). Interesting findings from genetic studies support the latter possibility. Several genes that affect GABA reception have been identified as influencing the risk for alcohol dependence (Covault et al., 2004; Radel et al., 2005).

Endogenous Opioid Peptides One of the most interesting and important advances in neuroscience research was the discovery of the endogenous opioids known as **endorphins** and enkephalins. These relatively short chains of amino acids, or *neuropeptides,* are naturally synthesized in the brain and are closely related to morphine in their pharmacological properties. Opioid peptides possess a chemical affinity for specific receptor sites, in the same way that a key fits into a specific lock. Several types of opioid peptides are distributed widely throughout the brain. They appear to be especially important in the activities associated with systems that control pain, emotion, stress, and reward, as well as such biological functions as feeding and growth (Froehlich, 1997).

Research studies have demonstrated many interesting features of the endorphins. Laboratory animals can develop tolerance to injections of endorphins, just as they develop tolerance to addicting drugs like morphine, and they also exhibit symptoms of withdrawal if the injections are suddenly discontinued. These studies confirm the pharmacological similarity between endogenous and exogenous opioids.

Some theorists associate alcoholism with exaggerated activation of the endogenous opioid system in response to alcohol stimulation (Gianoulakis, DeWaele, & Thavundayil, 1996). Several lines of evidence support this hypothesis. One is that opioid receptor antagonists (drugs that block the

effects of opioid peptides) produce a decrease in alcohol self-administration in laboratory animals. Another important bit of information comes from drug trials with human participants: When alcoholic patients take naltrexone, an antagonist of endogenous opioids, they drink less alcohol and report that the subjective "high" associated with drinking is noticeably diminished (see the section on treatment with medication, later in this chapter). Finally, in both rodents and humans, a genetic predisposition toward increased consumption of alcohol is associated with high levels of opioid system response to the ingestion of alcohol (Froehlich, 1997). For all of these reasons, it seems likely that endogenous opioid peptides are somehow involved in mediating alcohol dependence.

PSYCHOLOGICAL FACTORS

Genetic factors and neurochemistry undoubtedly account for many of the problems associated with addictive drugs, but as the systems perspective indicates, biological explanations are not incompatible with psychological ones. In fact, extensive research over the past several decades has found that the progression of substance dependence depends on an interaction between psychological and biological factors. Drug effects interact with the person's beliefs and attitudes, as well as with the social context in which the drugs are taken.

Expectations About Drug Effects Placebo effects demonstrate that expectations are an important factor in any study of drug effects (see Chapter 3). This is certainly true in the case of alcohol. Expectations account for many effects that are sometimes assumed to be products of the drug itself (Moss & Albery, 2009). For example, subjects who believed that they had ingested alcohol but who had actually consumed only tonic water display exaggerated aggression and report enhanced feelings of sexual arousal (Testa et al., 2006). Much less is known about expectancies for drugs other than alcohol, but there is good reason to believe that these cognitive factors also influence the ways in which people respond to cannabis, nicotine, stimulants, anxiolytics, and sedatives.

Many studies have examined the specific nature of alcohol expectancies (Nicolai et al., 2010). Investigators asked people, Why do you drink? What do you expect to happen after you have consumed a few beers or a couple of glasses of wine? Subjects' answers to these questions fit into six primary categories:

1. Alcohol transforms experiences in a positive way (for example: Drinking makes the future seem brighter).
2. Alcohol enhances social and physical pleasure (for example: Having a few drinks is a nice way to celebrate special occasions).
3. Alcohol enhances sexual performance and experience (for example: After a few drinks, I am more sexually responsive).
4. Alcohol increases power and aggression (for example: After a few drinks, it is easier to pick a fight).
5. Alcohol increases social assertiveness (for example: Having a few drinks makes it easier to talk to people).
6. Alcohol reduces tension (for example: Alcohol enables me to fall asleep more easily).

These expectations may constitute one of the primary reasons for continued and increasingly heavy consumption of alcoholic beverages. In fact, expectancy patterns can help predict drinking behaviors. Longitudinal studies have found that adolescents who are just beginning to experiment with alcohol and who initially have the most positive expectations about the effects of alcohol go on to consume greater amounts of alcoholic beverages (Smith et al., 1995). This type of demonstration is important because it indicates that, in many cases, the expectations appear before the person begins to drink heavily. Therefore, expectations may play a role in the onset of the problem rather than being consequences of heavy drinking (see Research Methods).

Common expectations about the effects of drinking alcohol include the notion that it enhances sexual arousal and experience.

Where do these expectations come from, and when do they develop? In some cases they may arise from personal experiences with alcohol, but they can also be learned indirectly. Many adolescents hold strong beliefs about the effects of alcohol long before they take their first drink. These expectations are influenced by a variety of environmental factors, including parental and peer attitudes as well as the portrayal of alcohol in the mass media (Agrawal et al., 2008). Follow-up studies have demonstrated that adolescents' expectations about the effects of alcohol are useful in predicting which individuals will later develop drinking problems (Jones, Corbin, & Fromme, 2001). Positive expectancies about alcohol, which are likely to encourage people to drink, are especially influential. Negative

RESEARCH METHODS

STUDIES OF PEOPLE AT RISK FOR DISORDERS

We have used the term *risk* informally throughout this book to refer to a hazard—the possibility of suffering harm. In scientific research, a risk is a statement about the probability that a certain outcome will occur. For example, the NCS found that the risk that a person in the United States will develop alcoholism at some point in his or her life is about 14 in 100 (see Figure 11.3). The combined risk for all types of illegal and controlled substances (such as cannabis, cocaine, heroin, and barbiturates) is about 8 in 100. The concept of risk implies only probability, not certainty. Someone who is "at risk" may or may not suffer harm, depending on many other events and circumstances. For example, men are at greater risk than women for the development of alcoholism, but that does not mean that all men will become alcoholics.

Risk factors are variables that are associated with a higher probability of developing a disorder. Notice that this use of the term risk implies association, not causality. The concept of risk simply reflects a correlation between the risk factor and the disorder. Some risk factors are demographic variables, such as gender and race. Others are biological and psychological variables. In the case of alcoholism, and many other types of psychopathology, family history of the disorder is an important risk factor. Expectancies about the effects of drugs represent another important risk factor for alcoholism. People who expect that alcohol will reduce tension or transform experiences in a positive way are more likely to drink frequently and heavily than those who have negative expectancies about the effects of alcohol.

In order to determine whether certain risk factors might play a *causal* role in the development of the disorder, it is often necessary to conduct longitudinal studies (see Research Methods in Chapter 8). The investigator collects information about each person before the onset of the disorder. He or she can therefore determine whether the risk factor is present before or only after the onset of symptoms. In other words, do people believe that alcohol reduces tension before they start to drink heavily, or do they develop this belief after they have been drinking heavily for some time? Longitudinal studies can be extremely expensive, and often they take several years to complete. They also require large numbers of participants because everyone in the study will not go on to develop the disorder in question.

Some of these shortcomings of longitudinal studies are especially relevant to research on substance abuse disorders. The risk for developing such disorders is quite low in the general population. For example, even though alcoholism is one of the most prevalent forms of mental disorder, a longitudinal study that follows the development of 100 randomly selected people from childhood to middle age will find only about 14 alcoholic adults (based on NCS data). Thus, to collect a useful amount of data, researchers need to study a large sample, which can be very expensive.

Recognition of this problem led scientists to develop special methods to increase the productivity of longitudinal research. One important technique is the **high-risk research design.** In high-risk research, subjects are selected from the general population based on a well-documented risk factor (Knop et al., 2003; Tarter & Vanyukov, 2001). A number of risk factors might be used to select subjects: positive family history for a given disorder, the presence of certain psychological characteristics, or perhaps a set of demographic variables such as age, gender, and/or race. High-risk research studies are designed to follow their participants over time, beginning before the onset of serious disorders. They hope to identify factors that increase or decrease the probability that people who are vulnerable to a disorder will eventually develop its active symptoms.

How does the high-risk strategy increase efficiency of longitudinal studies?

expectancies are associated with diminished use but seem to be less powerful.

INTEGRATED SYSTEMS

Alcoholism and other forms of addiction clearly result from an interaction among several types of systems. Various social, psychological, and biological factors influence the person's behavior at each stage in the cycle, from initial use of the drug through the eventual onset of tolerance and withdrawal. Furthermore, it appears that different influences are important at different stages of use. The process seems to progress in the following way. Initial experimentation with drugs is influenced by the environment—the person's family, peers, school, and neighborhood (Rhee et al., 2003). Other people also influence the person's attitudes and expectations about the effects of drugs. Access to drugs, in addition to the patterns in which they are originally consumed, is determined, in part, by cultural factors.

For many people, drinking alcohol leads to short-term positive effects that reinforce continued consumption. The exact psychological mechanisms that are responsible for reinforcing heavy drinking may take several different forms. They may involve diminished self-awareness, stress reduction, or improved mood. These effects of alcohol on behavior and subjective experience are determined, in part, by the person's expectations about the way in which the drug will influence his or her feelings and behavior (Baer, 2002).

Genetic factors play an important role in the etiology of alcoholism. After the person has begun to use alcohol, genetic factors become increasingly important in shaping patterns of use (Dick et al., 2007). There are most likely several different types of genetic influences. Genes interact strongly with environmental events for certain types of the disorders. A genetic predisposition to alcohol dependence probably causes the person to react to alcohol in an abnormal fashion. It is not clear whether those who are vulnerable to alcoholism are initially more or less sensitive than other people to the reinforcing effects of alcohol. Research studies have demonstrated both patterns of response (Sher, Grekin, & Williams, 2005).

The biological mechanisms responsible for abnormal reactions to alcohol seem to involve several interrelated neurotransmitter systems (Hyman & Malenka, 2001). Dopamine activity in the brain's reward pathway is stimulated by alcohol as well as other drugs of abuse. Another important consideration may be a deficiency in serotonin activity in certain areas of the limbic system. Drinking alcohol initially corrects this problem and increases serotonin activity, but the person eventually begins to feel worse after tolerance develops.

Drinking gradually becomes heavier and more frequent. The person becomes tolerant to the effects of alcohol and must drink larger quantities to achieve the same reinforcing effects. After he or she becomes addicted to alcohol, attempts to quit drinking are accompanied by painful withdrawal symptoms. Prolonged abuse can lead to permanent neurological impairment, as well as the disruption of many other organ systems.

Treatment

The treatment of alcoholism and other types of substance use disorders is an especially difficult task. Many people with substance use disorders do not acknowledge their difficulties, and only a relatively small number seek professional help. When they do enter treatment, it is typically with reluctance or on the insistence of friends, family members, or legal authorities. Compliance with treatment recommendations is often low, and dropout rates are high. The high rate of comorbidity with other forms of mental disorder presents an additional challenge, complicating the formulation of a treatment plan. Treatment outcome is likely to be least successful with those people who have comorbid conditions.

The goals of treatment for substance use disorders are a matter of controversy. Some clinicians believe that the only acceptable goal is total abstinence from drinking or drug use. Others have argued that, for some people, a more reasonable goal is the moderate use of legal drugs. Important questions have also been raised about the scope of improvements that might be expected from a successful treatment program. Is the goal simply to minimize or eliminate drug use, or should we expect that treatment will also address the social, occupational, and medical problems that are typically associated with drug problems? Getting Help at the end of this chapter offers additional resources for those seeking help and information on recovering from substance abuse.

DETOXIFICATION

Alcoholism and related forms of drug abuse are chronic conditions. Treatment is typically accomplished in a sequence of stages, beginning with a brief period of **detoxification**—the removal of a drug on which a person has become dependent—for three to six weeks (Coombs, Howatt, & Coombs, 2005). This process is often extremely difficult, as the person experiences marked symptoms of withdrawal and gradually adjusts to the absence of the drug. For many types of CNS depressants, such as alcohol, hypnotics, and sedatives, detoxification is accomplished gradually. Stimulant drugs, in contrast, can be stopped abruptly (Schuckit, 2005). Although detoxification usually takes place in a hospital, some evidence indicates that it can be accomplished with close supervision on an outpatient basis.

People who are going through alcohol detoxification are often given various types of medications, including benzodiazepines and anticonvulsants, primarily as a way of minimizing withdrawal symptoms (O'Brien & McKay, 2007). This practice is controversial, in part because many people believe that it is illogical to use one form of drug, especially one that can be abused itself, to help someone recover from dependence on another drug.

MEDICATIONS DURING REMISSION

Following the process of detoxification, treatment efforts are aimed at helping the person maintain a state of remission. The best outcomes are associated with stable, long-term abstinence from drinking. Several forms of medication are used to help the person achieve this goal.

Disulfiram (Antabuse) is a drug that can block the chemical breakdown of alcohol. It was introduced as a treatment for alcoholism in Europe in 1948 and is still used fairly extensively (Fuller & Gordis, 2004). If a person who is taking disulfiram consumes even a small amount of alcohol, he or she will become violently ill. The symptoms include nausea, vomiting, profuse sweating, and increased heart rate and respiration rate. People who are taking disulfiram will stop drinking alcohol in order to avoid this extremely unpleasant reaction. Unfortunately, voluntary compliance with this form of treatment is poor. Many patients discontinue taking disulfiram, usually because they want to resume drinking or because they believe that they can manage their problems without the drug.

Naltrexone (Revia) is an antagonist of endogenous opioids that has been found to be useful in the treatment of alcohol dependence following detoxification. Research studies have demonstrated that patients who received naltrexone and psychotherapy are less likely to relapse than patients who receive psychotherapy plus a placebo (Carmen et al., 2004). Some clinical patients report that, if they drink while also taking naltrexone, they do not feel as "high" as they would without naltrexone. Naltrexone may dampen the person's craving by blocking alcohol's ability to stimulate the opioid system. In other words, it works by reducing the rewarding effects of alcohol rather than by inducing illness if the person drinks.

Another promising medication for treating alcoholism is acamprosate (Campral). An extensive body of evidence indicates that people taking acamprosate are able to reduce their average number of drinking days by 30 percent to 50 percent (Mann, Lehert, & Morgan, 2004). It also increases

the proportion of people who are able to achieve total abstinence (approximately 22 percent among people taking acamprosate and 12 percent taking placebo after 12 months of treatment). Like naltrexone, acamprosate is intended to be used in conjunction with a psychological treatment program. The dropout rate is very high without these added features (Hart, McCance-Katz, & Kosten, 2001; Malcolm, 2003).

Psychiatrists also use SSRIs, such as fluoxetine, for the long-term treatment of alcoholic patients. Outcome studies suggest that SSRIs have small and inconsistent effects in reducing drinking among those patients who are not also depressed. They do seem to be effective, however, for the treatment of people with a dual diagnosis of alcohol dependence and major depression (O'Brien & McKay, 2002).

SELF-HELP GROUPS: ALCOHOLICS ANONYMOUS

One of the most widely accepted forms of treatment for alcoholism is Alcoholics Anonymous (AA). Organized in 1935, this self-help program is maintained by alcohol abusers for the sole purpose of helping other people who abuse alcohol become and remain sober. Because it is established and active in virtually all communities in North America and Europe, as well as in many other parts of the world, AA is generally considered to be "the first line of attack against alcoholism" (Nathan, 1993). Many members of AA are also involved in other forms of treatment offered by various types of mental health professionals, but AA is not officially associated with any other form of treatment or professional organization. Similar self-help programs have been developed for people who are dependent on other drugs, such as opioids (Narcotics Anonymous) and cocaine (Cocaine Anonymous).

Group therapy is an important part of most inpatient treatment programs. It offers an opportunity for patients to acknowledge and confront openly the severity of their problems.

The viewpoint espoused by AA is fundamentally spiritual in nature (Kaskutas et al., 2003). AA is the original "12-step program." In the first step, the person must acknowledge that he or she is powerless over alcohol and unable to manage his or her drinking. The remaining steps involve spiritual and interpersonal matters such as accepting "a Power greater than ourselves" that can provide the person with direction; recognizing and accepting personal weaknesses; and making amends for previous errors, especially instances in which the person's drinking caused hardships for other people. One principal assumption is that people cannot recover on their own (Emrick, 1999).

The process of working through the 12 steps to recovery is facilitated by regular attendance at AA meetings, as often as every day of the first 90 days after the person stops drinking. Most people choose to attend less frequently if they are able to remain sober throughout this initial period. Meetings provide chronic alcohol abusers with an opportunity to meet and talk with other people who have similar problems, as well as something to do instead of having a drink. New members are encouraged to call more experienced members for help at any time if they experience an urge to drink. There is enormous variability in the format and membership of local AA meetings (Montgomery, Miller, & Tonigan, 1993).

It is difficult to evaluate the effectiveness of AA, for a number of reasons. Long-term follow-up is difficult, and it is generally impossible to employ some of the traditional methods of outcome research, such as random assignment to groups and placebo controls. Early dropout rates are relatively high: About half of all the people who initially join AA leave in less than three months. On the other hand, survival rates (defined in terms of continued sobriety) are much higher for those people who remain in AA. About 80 percent of AA members who have remained sober for between two and five years will remain sober in the next year (Tonigan, Connors, & Miller, 2003).

How does AA differ from other approaches to treating alcoholism?

COGNITIVE BEHAVIOR THERAPY

Psychological approaches to substance use disorders have often focused on cognitive and behavioral responses that trigger episodes of drug abuse. In the case of alcoholism, heavy drinking has been viewed as a learned, maladaptive response that some people use to cope with difficult problems or to reduce anxiety. Cognitive behavior therapy teaches people to identify and respond more appropriately to circumstances that regularly precipitate drug abuse (Finney & Moos, 2002).

Coping Skills Training One element of cognitive behavior therapy involves training in the use of social skills, which might be used to resist pressures to drink heavily. It also

includes problem-solving procedures, which can help the person both to identify situations that lead to heavy drinking and to formulate alternative courses of action. Anger management is one example. Some people drink in response to frustration. Through careful instruction and practice, people can learn to express negative emotions in constructive ways that will be understood by others. The focus in this type of treatment is on factors that initiate and maintain problem drinking rather than the act of drinking itself.

Cognitive events also play an important part in this approach to treatment. Expectations about the effects of alcohol are challenged, and more adaptive thoughts are rehearsed. Negative patterns of thinking about the self and events in the person's environment are also addressed because they are linked to unpleasant emotions that trigger problem drinking.

Relapse Prevention Most people who have been addicted to a drug will say that quitting is the easy part of treatment. The more difficult challenge is to maintain this change after it has been accomplished. Unfortunately, most people will slip up and return to drinking soon after they stop. The same thing can be said for people who stop smoking or using any other drug of abuse. These slips often lead to a full-scale return to excessive and uncontrolled use of the drug. Successful treatment, therefore, depends on making preparations for such incidents.

Relapse prevention is a cognitive behavioral approach to treatment view that has been applied to all forms of substance dependence, ranging from alcoholism to nicotine dependence (Marlatt, Blume, & Parks, 2001; Shiffman et al., 1996; Witkiewitz, Marlatt, & Walker, 2005). It has also been applied to other disorders associated with impulsive behavior, such as bulimia and inappropriate sexual behaviors (see Chapters 10 and 12). It places principal emphasis on events that take place after detoxification and is aimed at helping the addict to deal with the challenges of life without drugs. The therapist helps patients learn more adaptive coping responses, such as applied relaxation and social skills, that can be used in situations that formerly might have triggered drug use.

One important feature of the relapse prevention model is concerned with the *abstinence violation effect,* which refers to the guilt and perceived loss of control that the person feels whenever he or she slips and finds himself or herself having a drink (or a cigarette or whatever drug is involved) after an extended period of abstinence. People typically blame themselves for failing to live up to their promise to quit. They also interpret the first drink or use of the drug as a signal that further efforts to control their drinking will be useless. The following brief case study describes one man's thoughts and feelings, shortly after he returned to the use of heroin. Just prior to this relapse, he had been actively involved in a treatment program and had stayed "clean" for several months.

BRIEF CASE STUDY

Relapse to Heroin Use

It was like goin' home," he tells me later, "and mom's got your favorite dish on the stove, and you smell it, to the back of your tongue, way back. That's the rush of the dope. It's right there, and for like two, three minutes I'm floating. But it was just a quarter of a bag, a baby rush. So I get up and lay down in my bed, put on the (music) again. And I'm feeling dirty, man. I'm thinking, that wasn't nothing, it wasn't worth it. Two, three minutes of this hot euphoria and then I just nod off to sleep."

He slams his fist on his knee. "I can't believe how bad I (screwed) up," Mike wails through his tears. "Damn! I know what happened ain't nobody's fault but mine, and I'm eating myself up over it. I'm scared out of my mind. I mean, it's like I'm afraid of myself. I really see it now, there's so much (stuff) inside me from my past that I ain't worked out yet that I scare even me. So where do I go with that if they kick me out? How do I stay off the dope if I'm alone again?"

Mike looks up, his eyes wide, wet with tears.

"Maybe what they say is true, I'm already a junkie again. It's too late. But I did just one hit, that's all. And I can't be doing more dope, I know that. If I go on a real run of heroin this time, I won't come back, ever. I've seen it now—I *can* blow it, I *can* relapse, I can die. Damn! This is the time I need help more than ever, and this is when they're going to kick me out" (Shavelson, 2001, pp. 161 and 166).

Relapse prevention programs are aimed at exactly this type of conflict. They teach patients to expect that they may slip occasionally and to interpret these behaviors as a temporary "lapse" rather than a total "relapse."

Short-Term Motivational Therapy Many people with substance use disorders do not seek or take full advantage of treatment opportunities because they fail to recognize the severity of their problems. Motivational interviewing is a nonconfrontational procedure that can be used to help people resolve their ambivalence about using drugs and make a definite commitment to change their behavior (Miller, 1995). It is based on the notion that in order to make a meaningful change, people must begin by recognizing the inconsistency between their current behavior and their long-term goals. For example, chronic heavy drinking is not compatible with academic or occupational success.

Motivational interviewing begins with a discussion of problems—issues reported by the patient as well as concerns that have been expressed by others such as friends and family members. The person is asked to reflect on feedback that is provided in a nonthreatening way. Rather than confronting the person, arguing about the reasons for drinking, or demanding action, the therapist responds empathically in an effort to avoid or minimize defensive reactions that will interfere with attempts to change.

The primary goal of this process is to increase the person's awareness of the nature of his or her substance use problems. Central features of motivational interviewing include a comprehensive assessment of the situation and personalized feedback. Emphasis is placed on ways in which the person sees his or her problems rather than assigning diagnostic labels, such as "alcoholism." Various options for creating change are discussed. The therapist and the patient work together to select the most appropriate method to follow. This stage of the interaction is designed to encourage the person's belief in his or her own ability to accomplish positive change.

Motivational interviewing may be most helpful to people whose substance abuse problems are not yet severe or chronic.

It can be used as a stand-alone intervention or in combination with other approaches to treatment. If the person is not ready to abstain completely, short-term motivational therapy can be used to help the person reduce the frequency or intensity of alcohol consumption (Roberts & Marlatt, 1999).

OUTCOME RESULTS AND GENERAL CONCLUSIONS

Although many studies have evaluated the effects of alcohol treatment programs, two deserve special attention because of their large sample sizes and the rigorous methods that the investigators employed. One is known as Project MATCH because it was designed to test the potential value of matching certain kinds of clients to specific forms of treatment (Babor & Del Boca, 2003). In other words, would the outcomes associated with different forms of intervention be related to certain characteristics of the patients (such as the presence or absence of antisocial personality traits)?

The study evaluated three forms of psychological treatment: cognitive behavior therapy (12 sessions focused on coping skills and relapse prevention), 12-step facilitation therapy (12 sessions designed to help patients become engaged in AA), and motivational enhancement therapy (four sessions over 12 weeks designed to increase commitment to change). Most of the people in all three groups attended at least some AA meetings in addition to their assigned form of treatment. More than 1,700 patients were randomly assigned to one of these three conditions. Outcome measures were collected for three years after the end of treatment.

Results indicated that all three forms of treatment led to major improvements in amount of drinking as well as other areas of life functioning (Miller & Longabaugh, 2003). Before treatment, patients in this study averaged 25 drinking days per month. After treatment, they averaged fewer than six days per month (across all forms of treatment). Very few differences were found between the different treatment methods. The one exception favored 12-step facilitation therapy, in which 24 percent of patients were completely abstinent one year after treatment, compared to approximately 15 percent in the other two groups. Analyses that focused on the characteristics of individual clients suggested that there is relatively little reason to try to match certain kinds of patients to specific forms of treatment.

The second study involved a naturalistic evaluation of substance abuse treatment programs administered at 15 sites by the Department of Veterans Affairs (VA) (Finney, Moos, & Humphreys, 1999; Moos et al., 1999). The VA study compared programs that emphasized three approaches to the treatment of substance use disorders: 12-step programs, cognitive behavior therapy, and "eclectic therapy" (a combination of several approaches). The study included more than 3,000 patients. Most of these people had a diagnosis of alcohol dependence, but many also abused other types of drugs. Unlike Project MATCH, they were not randomly assigned to treatments. Despite these differences in methodology, results of the VA study were very similar to those obtained in Project MATCH. Patients in all three groups made significant improvements with regard to both patterns of substance use and levels of social and occupational functioning. People who participated in more treatment sessions had better outcomes than people who received less treatment. When differences were found between different forms of treatment, they tended to favor the 12-step programs. No support was found for the assumption that certain types of patients would do better in one form of treatment than in another.

Comprehensive reviews of these studies and the rest of the research literature regarding treatment of alcoholism and drug abuse point to several general conclusions (Amato et al., 2011; Glasner-Edwards & Rawson, 2010):

- People who enter treatment for various types of substance abuse and dependence typically show improvement in terms of reduced drug use that is likely to persist for several months following the end of treatment. Unfortunately, relapse is also relatively common.
- There is little evidence to suggest that one form of treatment (inpatient or outpatient, professional or self-help, individual or group) is more effective than another. When differences have been found, they tend to favor self-help groups, such as AA, particularly in terms of success in achieving abstinence.
- There is only limited support for the assumption that certain kinds of patients do better in one kind of treatment than another (the matching hypothesis).
- Increased amount of treatment and greater frequency of attendance in self-help meetings and aftercare counseling are associated with better outcomes.
- Among those people who are able to reduce their consumption of drugs, or abstain altogether, improvements following treatment are usually not limited to drug use alone but extend to the person's health in general as well as his or her social and occupational functioning.

What factors predict better long-term outcome for treatment of alcoholism?

Long-term outcome for the treatment of alcoholism is best predicted by the person's coping resources (social skills and problem-solving abilities), the availability of social support, and the level of stress in the environment. These considerations appear to be more important than the specific type of intervention that people receive. Those individuals who are in less stressful life situations, whose families are more cohesive and less supportive of continued drinking, and who are themselves better equipped with active coping skills are most likely to sustain their improvement over several years.

Getting Help

If you have been looking for help in the area of substance dependence, you have probably noticed two things: (1) There are so many different sources of advice and information that the situation can quickly become quite confusing, and (2) the field is sharply divided on a number of crucial issues. Whose advice should you follow? Among all of the self-help books dealing with drugs and alcohol, one stands out on the basis of its strong link to the research literature as well as the extensive clinical experience of the author. Marc Schuckit's book *Educating Yourself About Alcohol and Drugs: A People's Primer* provides sensible answers to the questions asked by people who are wondering about their own, or someone else's, substance use problems.

Denial is a prominent feature of most substance use disorders. It is usually easier to dismiss suggestions that you have begun to use alcohol or drugs in a self-destructive pattern than it is to face the problem directly. Schuckit's book includes a perceptive chapter titled "Is there really a problem?" The bottom line is this: "If you repeatedly have returned to substance use even though that substance has caused a disruption in your life, you do have a problem." Subsequent chapters in Schuckit's book provide thoughtful and practical guidance on topics such as the symptoms of withdrawal, the process of detoxification, the relative merits of self-help groups, outpatient therapy, and hospitalization, and how to find a specific treatment program in your area.

The Internet also provides an enormous amount of information regarding substance use disorders. For information about problems associated with the use of alcohol and drugs, you might want to visit Web pages maintained by the National Institute on Alcohol Abuse and Alcoholism (www.niaaa.nih.gov) and the National Institute on Drug Abuse (www.nida.nih.gov). These websites are primarily concerned with information about federally funded research programs, but they also include answers to frequently asked questions, as well as treatment referral information.

Most people who enter treatment for substance use problems become involved, at least temporarily, with self-help groups such as Alcoholics Anonymous (AA) and Narcotics Anonymous (NA). Related groups, like Alanon and Alateen, are designed for the families and children of people who are dependent on alcohol. You can contact these groups through the Internet. The URL for Alcoholics Anonymous is www.alcoholics-anonymous.org. Many people believe, often passionately, that AA is the most beneficial program for helping people to recover from alcoholism. Others disagree. If you want to consider alternative points of view, visit the website maintained by Stanton Peele, who is one of AA's most persistent, enthusiastic, and articulate critics. The URL for his homepage is www.peele.net. Peele challenges the biological reductionism that often dominates current views of alcoholism, and he promotes approaches to treatment that do not rely exclusively on total abstinence from drinking.

Evidence regarding the long-term outcome of serious substance use disorders can be discouraging, but it is important to remember that a substantial minority of people with these problems do manage to achieve an extended, stable recovery. The research literature does not point to one form of treatment as being clearly superior to another. Therefore, you should consider several alternatives to treatment and select the one that makes most sense in terms of your own life and your own view of the world.

SUMMARY

- DSM-IV-TR uses two terms to describe substance use disorders. **Substance dependence,** the more severe of the two forms, refers to a pattern of repeated self-administration that often results in **tolerance, withdrawal,** or compulsive drug-taking behavior. **Substance abuse** describes a more broadly conceived, less severe pattern of drug use that is defined in terms of interference with the person's ability to fulfill major role obligations at work or at home, recurrent use of a drug in dangerous situations, or repeated legal difficulties that are associated with drug use.
- A **drug of abuse**—sometimes called a *psychoactive substance*—is a chemical substance that alters a person's mood, level of perception, or brain functioning. Although patterns of dependence are similar in some ways for all drugs, each type of drug also has some unique features.
- Prolonged abuse of alcohol can have a devastating impact on social relationships and occupational functioning while disrupting the functions of several important organ systems. Alcohol dependence has more negative health consequences than does abuse of almost any drug, with the possible exception of nicotine.

- Nicotine is one of the most harmful addicting drugs. Recognizing the serious long-term health consequences of exposure to nicotine, the U.S. Food and Drug Administration has prohibited the sale and distribution of tobacco products to children and adolescents. This policy attempts to prevent the development of nicotine addiction rather than trying to ban use of the drug completely.
- The **psychomotor stimulants,** such as amphetamine and cocaine, activate the sympathetic nervous system and induce a positive mood state. High doses of amphetamines and cocaine can lead to the onset of psychosis.
- **Opiates** have properties similar to those of opium and can induce a state of dreamlike euphoria. Tolerance develops quickly to opiates. After repeated use, their positive emotional effects are replaced by long-term negative changes in mood and emotion.
- **Barbiturates** and **benzodiazepines** can be used, as prescribed by a physician, to decrease anxiety (tranquilizers) or help people sleep (hypnotics). People who abruptly stop taking high doses of benzodiazepines may experience withdrawal symptoms, including a return of the original anxiety symptoms.
- **Marijuana** and **hashish** can induce a pervasive sense of well-being and happiness. People do not seem to develop tolerance to THC (the active ingredient in marijuana and hashish) unless they are exposed to high doses over an extended period of time. Withdrawal symptoms are unlikely to develop among people who smoke marijuana occasionally.
- **Hallucinogens** induce vivid visual images that are usually pleasant but occasionally frightening. Unlike other drugs of abuse, hallucinogens are used sporadically rather than continuously. Most people do not increase their use of hallucinogens over time, and withdrawal symptoms are not observed.
- It is impossible to specify a typical course for substance dependence. The specific pattern varies from one person to the next. In the case of alcoholism, the only thing that seems certain is that periods of heavy use alternate with periods of relative abstinence.
- Alcohol dependence and abuse are the most common forms of mental disorder, with a lifetime prevalence of 30 percent in the NESARC study. Among people with alcohol use disorders, men outnumber women by a ratio of approximately two to one.
- Research on the etiology of alcoholism illustrates the ways in which various systems interact to produce and maintain drug dependence. There are several pathways to alcoholism. Social factors are particularly influential in the early phases of substance use. The culture in which a person lives influences the types of drugs that are used, the purposes for which they are used, and the expectations that people hold for the ways in which drugs will affect their experiences and behavior.
- Twin studies indicate that genetic factors influence patterns of social drinking as well as the onset of alcohol dependence. Adoption studies indicate that the offspring of alcoholic parents who are raised by nonalcoholic parents are more likely than people in the general population to develop drinking problems of their own.
- All of the psychoactive drugs cause increased dopamine activity in the reward pathways of the brain. Alcohol may stimulate the mesolimbic dopamine pathway directly, or it may act indirectly by inhibiting GABA neurons. Another focus of neurochemical research has been the role of endogenous opioids known as **endorphins.** Some theorists have argued that alcoholism is associated with excessive production of endorphins.
- Expectations about drug effects have an important influence on the ways in which people respond to alcohol and other drugs. People who believe that alcohol enhances pleasure, reduces tension, and increases social performance are more likely than other people to drink frequently and heavily.
- Treatment of substance use disorders is an especially challenging and difficult task, in light of the fact that many people with these problems do not recognize or acknowledge their own difficulties. Recovery begins with a process of detoxification. Self-help programs, such as Alcoholics Anonymous, are the most widely used and probably one of the most beneficial forms of treatment.

The Big Picture

CRITICAL THINKING REVIEW

- **What evidence is needed to show that a drug is addictive?**

  If repeated use of a drug is associated with the development of tolerance and withdrawal, or if it leads to a pathological pattern of use, then it is considered to be addictive . . . (see pp. 275–276)

- **What are the long-term consequences of abusing psychomotor stimulants?**

  Prolonged use of amphetamines and cocaine increases the person's risk for having a psychotic episode, but the most common and devastating effects of stimulant drugs are the serious disruption of occupational and social roles . . . (see pp. 279–280)

- **Where is the boundary between substance abuse and recreational drug use?**

  The transition to abuse from recreational use can be subtle and is not easily recognized, especially by the person who is using drugs. The distinction hinges on the pattern of use, which must be persistent over time and lead to maladaptive consequences before this diagnosis would be considered . . . (see pp. 283–284)

- **In what ways are drug problems different among the elderly?**

  Tolerance to many drugs is reduced among the elderly, and the symptoms of withdrawal may be more severe and prolonged . . . (see pp. 288–290)

- **What are the most important risk factors for alcoholism?**

  Increased risk is associated with both genetic and environmental factors. Some of the genes that influence risk of alcohol dependence have an impact on the metabolism of alcohol. Others may influence personality traits which increase the tendency to participate in dangerous patterns of consumption . . . (see pp. 295–296)

- **How does AA differ from other approaches to treating alcoholism?**

  Alcoholics Anonymous is a self-help program and is not officially associated with any other form of treatment. Its viewpoint is fundamentally spiritual in nature . . . (see p. 297)

- **What factors predict better long-term outcome for treatment of alcoholism?**

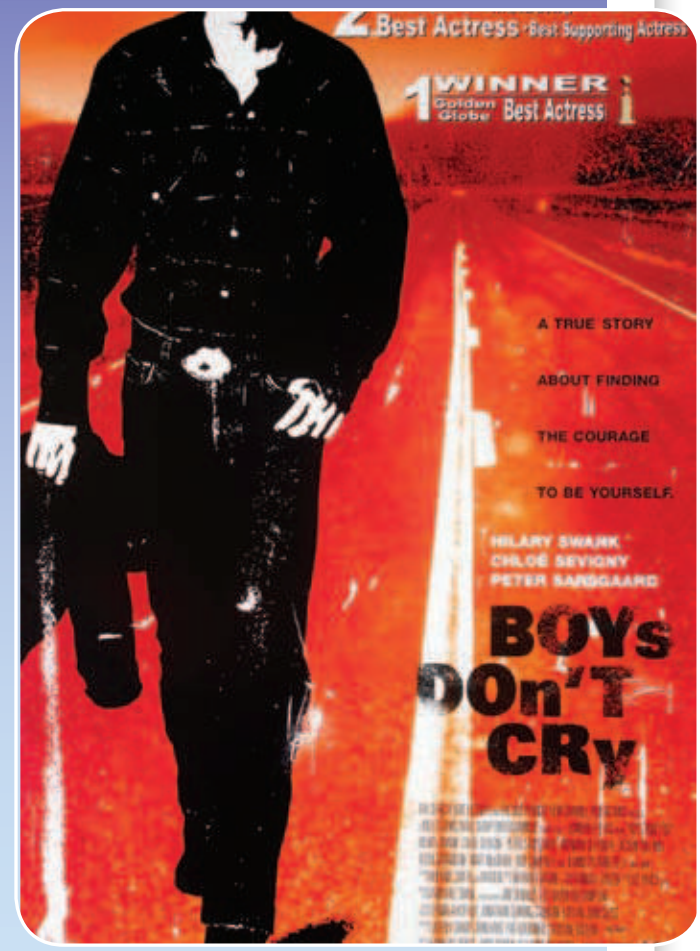
  Long-term outcome is best predicted by the person's coping resources (social skills and problem-solving abilities), the availability of social support, and the level of stress in the environment . . . (see p. 299)

KEY TERMS

barbiturates
benzodiazepines
detoxification
drug of abuse
endorphins
hallucinogens
hashish
high-risk research design
marijuana
opiates
polysubstance abuse
psychological dependence
psychomotor stimulants
substance abuse
substance dependence
tolerance
withdrawal

12

Sexual and Gender Identity Disorders



▶ *Boys Don't Cry* is a dramatization of the real-life story of Brandon Teena, a female-to-male transgender man who paid a terrible price for pursuing a relationship with a young woman.

Sex is often a perplexing area of our lives. Sexual experience can be a source of extreme pleasure, while also providing for the development and expression of intimacy with one's partner. From an evolutionary point of view, reproduction is the key to our survival. Sexual behavior also provides fertile ground for intense feelings of fear and guilt.

When something interferes with our ability to function sexually, it can be devastating both to the person who is affected and to the person's partner. Sometimes a person's inability to enjoy sexual experiences becomes so pervasive or so personally distressing that the person seeks professional help—alone or, more often, with his or her partner.

In other instances, a person may enjoy sex but his or her sexual interest may be triggered by unusual stimuli, or it may involve nonconsenting partners or the pain and suffering of themselves or others. The point at which occasional sexual difficulties become a "sexual dysfunction" is quite subjective and may say as much about sexual norms and expectations as anything else. Similarly, the definition of sexual conduct that is considered deviant has also changed over time. This chapter explores the mix of factors that influence what it means to be a man or a woman and the ways in which we engage in sexual relationships. It also offers a picture of the shifting ground that surrounds what mental health professionals consider to be normal and abnormal sexual practices.

The Big Picture

- Is it true that men focus on sex and women on intimacy?
- Are problems like premature ejaculation and failure to reach orgasm common?
- Is it a problem if you want to have sex too often?
- Why are drugs like Viagra advertised all the time?
- Should sexual offenders be treated or punished?
- Does sex change surgery work?

OVERVIEW

Any discussion of sexual disorders requires some frank consideration of normal sexuality. Such openness has been encouraged and promoted by mental health professionals who specialize in the study and treatment of sexual behavior.

William Masters (1915–2001) and Virginia Johnson (1925–) were undoubtedly the best-known sex therapists and researchers in the United States during the second half of the twentieth century. Their first book, *Human Sexual Response,* published in 1966, was based on their studies of nearly 700 normal men and women. Observations and physiological recordings were made in a laboratory setting while these individuals engaged in sexual activities, including masturbation and intercourse. Masters and Johnson's research received widespread attention in the popular media and helped make laboratory studies of sexual behavior acceptable (Maier, 2009).

On the basis of their data, Masters and Johnson described the human sexual response cycle in terms of a sequence of overlapping phases: excitement, orgasm, and resolution. Analogous processes occur in both men and women, but the timing may differ. Many of the physiological mechanisms involved in this cycle are now understood more clearly, but the general outline described by Masters and Johnson is still useful (Levin, 2008). There are, of course, individual differences in virtually all aspects of this cycle. Variations from the most common pattern may not indicate a problem unless the person is concerned about the response.

Sexual *excitement* increases continuously from initial stimulation up to the point of orgasm. It may last anywhere from a few minutes to several hours. Among the most dramatic physiological changes during sexual excitement are those associated with *vasocongestion*—engorgement of the blood vessels of various organs, especially the genitals. The male and female genitalia become swollen, reddened, and warmed. Sexual excitement also increases muscular tension, heart rate, and respiration rate. These physiological responses are accompanied by subjective feelings of arousal, especially at more advanced stages of excitement.

The experience of *orgasm* is usually distinct from the gradual buildup of sexual excitement that precedes it. This sudden release of tension is almost always experienced as being intensely pleasurable, but the specific nature of the experience varies from one person to the next. The female orgasm occurs in three stages, beginning with a "sensation of suspension or stoppage," which is associated with strong genital sensations. The second stage involves a feeling of warmth spreading throughout the pelvic area. The third stage is characterized

Sexual dysfunctions are best defined in terms of the couple rather than individual persons. They are frequently associated with marital distress.

by sensations of throbbing or pulsating, which are tied to rhythmic contractions of the vagina, the uterus, and the rectal sphincter muscle.

The male orgasm occurs in two stages, beginning with a sensation of ejaculatory inevitability. This is triggered by the movement of seminal fluid toward the urethra. In the second stage, regular contractions propel semen through the urethra, and it is expelled through the urinary opening.

During the *resolution* phase, which may last 30 minutes or longer, the person's body returns to its resting state. Men are typically unresponsive to further sexual stimulation for a variable period of time after reaching orgasm. This is known as the refractory period. Women, on the other hand, may be able to respond to further stimulation almost immediately. They are capable of experiencing a series of distinct orgasmic responses that are not separated by a period of noticeably lowered excitement.

Sexual dysfunctions can involve a disruption of any stage of the sexual response cycle. The following case study, written by Barry McCarthy, a psychologist at American University, is concerned with a man who had difficulty controlling the rate at which he progressed from excitement to orgasm.

CASE STUDY Margaret and Bill's Sexual Communication

Margaret and Bill, both in their late twenties, had been married for two years, and they had intercourse frequently. Margaret seldom reached orgasm during these experiences, but she was orgasmic during masturbation. The central feature of their problem was the fact that Bill was unable to delay ejaculation for more than a few seconds after insertion.

Unbeknownst to Margaret, Bill had attempted a "do-it-yourself" technique to gain better control [of ejaculation]. He had bought a desensitizing cream he'd read about in a men's magazine and applied it to the glans of his penis 20 minutes before initiating sex. He also masturbated the day before the couple had sex.

During intercourse he tried to keep his leg muscles tense and think about sports as a way of keeping his arousal in check. Bill was unaware that Margaret felt emotionally shut out during the sex. Bill was becoming more sensitized to his arousal cycle and was worrying about erection. He was not achieving better ejaculatory control, and he was enjoying sex less. The sexual relationship was heading downhill, and miscommunication and frustration were growing.

Margaret had two secrets that she had never shared with Bill. Although she found it easier to be orgasmic with manual stimulation, she had been orgasmic during intercourse with a married man she'd had an affair with a year before meeting Bill. Margaret expressed ambivalent feelings about that relationship. She felt that the man was a very sophisticated lover, and she had been highly aroused and orgasmic with him. Yet the relationship had been a manipulative one. He'd been emotionally abusive to Margaret, and the relationship had ended when he accused Margaret of giving him herpes and berated her. In fact, it was probably he who gave Margaret the herpes. Margaret was only experiencing herpes outbreaks two or three times a year, but when they did occur, she was flooded with negative feelings about herself, sexuality, and relationships. She initially saw Bill as a loving, stable man who would help rid her of negative feelings concerning sexuality. Instead, he continually disappointed her with the early ejaculation. Bill knew about the herpes but not about her sexual history and strong negative feelings.

Bill was terribly embarrassed about his secret concerning masturbation, which he engaged in on a twice-daily basis. From adolescence on, Bill had used masturbation as his primary means of stress reduction. For him, masturbation was a humiliating secret (he believed married men should not masturbate). The manner in which he masturbated undoubtedly contributed to the early ejaculation pattern. Bill focused only on his penis, using rapid strokes with the goal of ejaculating as quickly as he could. This was both to prevent himself from being discovered and from a desire to "get it over with" as soon as he could and forget about it.

When it came to his personal and sexual life, Bill was inhibited, unsure of himself, and had particularly low sexual self-esteem. As an adolescent, Bill remembered being very interested sexually, but very unsure around girls. Bill's first intercourse at 19 was perceived as a failure because he ejaculated before he could insert his penis in the woman's vagina. He then tried desperately to insert

Margaret had two secrets that she had never shared with Bill.

because the young woman urged him to, but he was in the refractory period (a phenomenon Bill did not understand), and so he did not get a firm erection and felt doubly humiliated (McCarthy, 1989, pp. 151–159).

The case of Bill and Margaret illustrates several important points. First, many sexual problems are best defined in terms of the couple rather than each partner individually. Second, although problems in sexual behavior clearly involve basic physiological responses and behavioral skills, each person's thoughts about the meaning of sexual behavior are also extremely important. Sexual behavior usually takes place in the context of a close, personal relationship. Current views of the sexual response cycle have expanded beyond a simple focus on the mechanisms related to excitement and orgasm (Basson et al., 2005). They begin at a point of sexual neutrality and consider factors that influence whether the person will seek or be receptive to stimuli that might lead to arousal. They also extend beyond the experience of arousal and orgasm to consider feelings of emotional and physical satisfaction, which ultimately serve to build intimacy.

The classification of sexual disorders was revised dramatically in the United States and Western Europe during the twentieth century. This process reflects important changes in the way that our culture views various aspects of sexual behavior. Before describing the disorders that are included in DSM-IV-TR, we outline briefly some of the clinical and scientific perspectives on sexuality that laid the foundation for our current system.

BRIEF HISTORICAL PERSPECTIVE

Early medical and scientific approaches to sexual behavior were heavily influenced by religious doctrines and prevailing cultural values. The exclusive purpose of sexual behavior was assumed to be biological reproduction; anything that varied from that narrow goal was considered a form of psychopathology and was usually subject to severe moral and legal sanctions. Medical authorities were more worried about excessive sexuality and inappropriate or unusual sexual activities than they were about a person's subjective dissatisfaction or impaired sexual performance.

The period between 1890 and 1930 saw many crucial changes in the ways in which society viewed sexual behavior (D'Emilio & Freedman, 1988). A significant number of people were beginning to think of sex as something other than a simple procreative function. If the purpose of sexual behavior was to foster marital intimacy or to provide pleasure, then interference with that goal might become a legitimate topic of psychological inquiry. Changes in prevailing social attitudes led to a change in the focus of systems for the classification of sexual problems. Over the course of the late twentieth century and into the twenty-first, there has been a trend toward greater tolerance of sexual variation among consenting adult partners and toward increased concern about impairments in sexual performance and experience.

Several leading intellectuals influenced public and professional opinions regarding sexual behavior during the first half of the twentieth century. The work of Alfred Kinsey (1894–1956), a biologist at Indiana University, was especially significant. In keeping with his conscious adherence to scientific methods, Kinsey adopted a behavioral stance, focusing specifically on those experiences that resulted in orgasm. In their efforts to describe human sexual behavior, Kinsey and his colleagues interviewed 18,000 men and women between 1938 and 1956 (Jones, 1997). They asked each participant a standard series of questions such as, "How old were you the first time that you had intercourse with another person?" Or, "How many times a week do you masturbate?"

The incredible diversity of experiences reported by his subjects led Kinsey to reject the distinction between normal and abnormal sexual behavior (Robinson, 1976). He argued that differences among people are quantitative rather than qualitative. For example, Kinsey suggested that the distinction between heterosexual and homosexual persons was essentially arbitrary and fundamentally meaningless. This argument was later used in support of the decision to drop homosexuality from DSM-III (see Chapter 1) and to cease regarding homosexuality as a form of abnormal behavior. Kinsey's comments regarding sexual dysfunction reflected a similar view. He believed that low sexual desire was simply a reflection of individual differences in erotic capacity rather than a reflection of psychopathology (Kinsey, Pomeroy, & Martin, 1948).

Sexual Dysfunctions

Sexuality represents a complex behavioral process that can easily be upset. Inhibitions of sexual desire and interference with the physiological responses leading to orgasm are called **sexual dysfunctions.** Problems can arise anywhere, from the earliest stages of interest and desire through the climactic release of orgasm. Some people also experience pain during sexual intercourse.

SYMPTOMS

How do people evaluate the quality of their sexual relationships? Subjective judgments obviously have an important impact on each person's commitment to a partnership. Dissatisfaction sometimes leads the couple to seek help from a mental health professional. It is useful, therefore, to know something about the ways in which normal couples evaluate their own sexual activities before we consider specific symptoms of sexual dysfunction.

One important set of data regarding normal sexual behavior and satisfaction was collected by the National Health and Social Life Survey (NHSLS), the first large-scale follow-up to the Kinsey reports (Laumann, Paik, & Rosen, 1994). The NHSLS research team conducted detailed, face-to-face interviews with nearly 3,500 men and women between the ages of 18 and 59 throughout the United States. Their questionnaire asked about masturbation and four basic sexual techniques involving partners: vaginal intercourse, fellatio, cunnilingus, and anal intercourse. The results indicate that masturbation is relatively common among both men and women. Virtually all of the men (95 percent) and women (97 percent) had experienced vaginal intercourse at some time during their lives. The investigators concluded that the vast majority of heterosexual encounters focus on vaginal intercourse. Most of the men (75 percent) and women (65 percent) also reported that they had engaged in oral sexual activities (as both the person giving and receiving oral-genital stimulation). Most sexual activity occurs in the context of monogamous relationships. Most of these conclusions regarding rates and types of sexual behaviors have been confirmed by a more recent survey conducted by investigators from the Kinsey Institute at Indiana University (Herbenick et al., 2010).

One of the most interesting aspects of the NHSLS results involves the ways in which the participants described the *quality* of their experiences during sexual activity. Figure 12.1 illustrates the proportion of people who said that they always had an orgasm during sexual activity with their primary partner during the past year. Several aspects of these data are worth mentioning. First, there is a very large difference between men and women with regard to the experience of orgasm. Only 29 percent of women reported that they always have an orgasm with a specific partner, compared to 75 percent of men. Second, notice that 44 percent of men reported that their partners always had orgasms during sex. This figure is much higher than the rate reported by women themselves. There are several plausible explanations for this discrepancy. Because female orgasm is sometimes less clearly defined than male orgasm, men may misinterpret some events as signs that their partners have had an orgasm. It may also be the case that women sometimes

Satisfaction in a sexual relationship is influenced by feelings of intimacy and successful communication as well as by the experience of orgasm.

mislead their partners into thinking that they have reached orgasm so that their partners will feel better about their own sexual prowess (Wiederman, 1997).

Figure 12.2 depicts data on participants' ratings of physical and emotional satisfaction. Here the differences between men and women are less marked. Physical and emotional satisfaction in a sexual relationship might reasonably be expected to be influenced by the experience of orgasm, but the relations among these variables are complex. A relationship may be considered intimate and satisfying simply because sexual activity occurs, regardless of whether it always results in orgasm. In fact, a large proportion of both men and women indicated that they were extremely satisfied with their partners, on both the physical and emotional dimensions. Notice in particular that, although only 29 percent of women indicated that they always have an orgasm with their partner, 41 percent of women said that they were extremely physically satisfied with their partners. This pattern suggests that the experience of orgasm is only one aspect of sexual satisfaction, especially for women. Other aspects of the relationship, including tenderness, intimacy, and affection, are also critically important (Mitchell & Graham, 2008; Tiefer, 2001).

Strong negative emotions, such as anger, fear, and resentment, are often associated with sexual dissatisfaction. In some cases, these emotional states appear before the onset of the sexual problem, and sometimes they develop later. Given

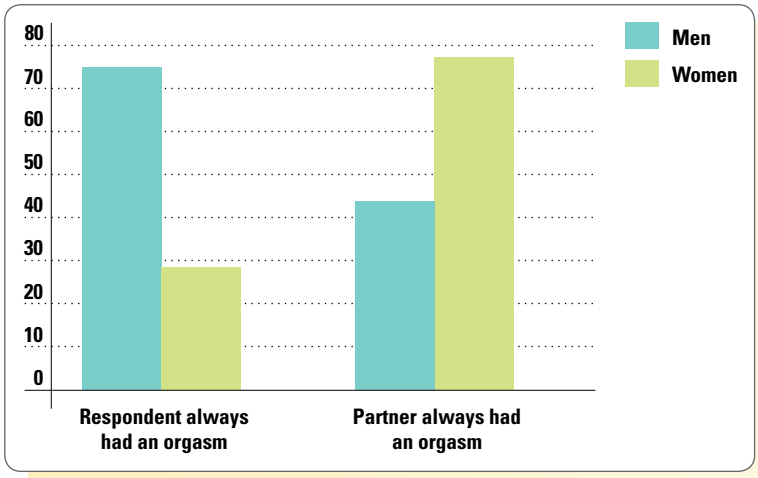


FIGURE 12.1 Sexual Response in Primary Partnership During Previous Year

This graph illustrates the frequency of orgasmic response as well as differences in perception by men and women in their partners' responses.

Source: E. O. Laumann, J. H. Gagnon, R. T. Michael, and S. Michaels, 1994, *The Social Organization of Sexuality: Sexual Practices in the United States,* Chicago: University of Chicago Press. Copyright © 1994. Reprinted by permission of The University of Chicago Press.

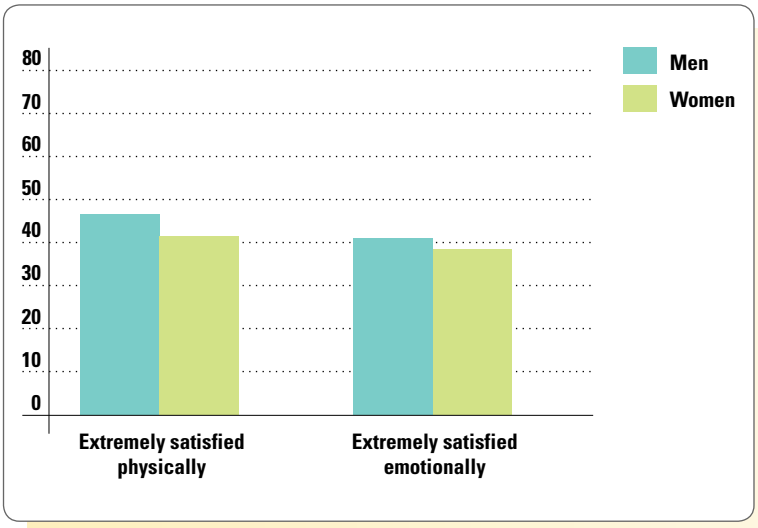


FIGURE 12.2 Sexual Satisfaction in Primary Partnership During the Previous Year

This graph illustrates the physical and emotional satisfaction reported by men and women in their primary partnerships.

Source: E. O. Laumann, J. H. Gagnon, R. T. Michael, and S. Michaels, 1994, *The Social Organization of Sexuality: Sexual Practices in the United States*, Chicago: University of Chicago Press. Copyright © 1994. Reprinted by permission of The University of Chicago Press.

the connection that many cultures make between virile sexual performance and "manhood," it is not surprising that men with erectile difficulties are often embarrassed and ashamed. Their humiliation can lead to secondary problems, such as anxiety and depression. Similar feelings frequently accompany early ejaculation and the recognition that a partner's sexual expectations have not been fulfilled. Women who have trouble becoming aroused or reaching orgasm also frequently experience profound frustration and disappointment. The emotional consequences of sexual problems can be devastating for both partners.

Should sexual problems be defined in terms of difficulty reaching orgasm?

DIAGNOSIS

DSM-IV-TR subdivides sexual dysfunctions into several types (see Table 12.1). The diagnostic criteria for these problems are much less specific than those used to define other kinds of disorders in DSM-IV-TR. Much is left to the judgment of

TABLE 12.1 Sexual Dysfunctions Listed In DSM-IV-TR

Hypoactive Sexual Desire Disorder: Persistently or recurrently deficient (or absent) sexual fantasies and desire for sexual activity.
Sexual Aversion Disorder: Persistent or recurrent extreme aversion to, and avoidance of, all (or almost all) genital sexual contact with a sexual partner.
Female Sexual Arousal Disorder: Persistent or recurrent inability to attain, or to maintain until completion of the sexual activity, an adequate lubrication–swelling response of sexual excitement.
Male Erectile Disorder: Persistent or recurrent inability to attain or maintain until completion of the sexual activity, an adequate erection.
Female Orgasmic Disorder: Persistent or recurrent delay in, or absence of, orgasm following a normal sexual excitement phase.
Male Orgasmic Disorder: Persistent or recurrent delay in, or absence of, orgasm following a normal sexual excitement phase during sexual activity.
Premature Ejaculation: Persistent or recurrent ejaculation with minimal sexual stimulation before, on, or shortly after penetration and before the person wishes it.
Dyspareunia: Recurrent or persistent genital pain associated with sexual intercourse in either a male or a female.
Vaginismus: Recurrent or persistent involuntary spasm of the musculature of the outer third of the vagina that interferes with sexual intercourse.

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

Low sexual desire can reflect the impact of many factors ranging from unhappiness (including anger and worry) to poor physical health.

the individual clinician. Failure to reach orgasm is not considered a disorder unless it is persistent or recurrent and results in marked distress or interpersonal difficulty. The DSM-IV-TR criteria also require that the sexual dysfunction is not better explained by another Axis I disorder (such as major depression) and is not the direct result of a chemical substance (such as alcohol) or a general medical condition.

One diagnostic criterion that is required for all forms of sexual dysfunction defined in DSM-IV-TR is the demonstration that the problem in question leads to marked distress or interpersonal difficulty (Segraves, Balon, & Clayton, 2007). In other words, someone who is not interested in sex or who experiences problems in sexual responsiveness would not qualify for a diagnosis of sexual dysfunction unless this circumstance is upsetting to him or her or causes interpersonal problems. Subjective distress and relationship problems will be evident in many of the brief case studies that are included in this chapter.

We must be cautious, however, in weighing the diagnostic importance of relationship problems and the feelings of the person's partner. A panel of leading experts on women's reproductive health recommended that the only consideration in this regard should be personal distress experienced by the woman (Basson et al., 2000). The satisfaction and concerns of her partner might be an important consideration in terms of their relationship itself, but it should not be grounds for assigning to the woman a diagnosis of sexual dysfunction unless she is personally dissatisfied with her own sexual experience.

Hypoactive Sexual Desire Disorder Sexual desire sets the stage for sexual arousal and orgasm. Some clinicians refer to sexual desire as the person's willingness to approach or engage in those experiences that will lead to sexual arousal. Inhibited, or **hypoactive, sexual desire** is defined in terms of subjective experiences, such as lack of sexual fantasies and lack of interest in sexual experiences. The absence of interest in sex must be both persistent and pervasive to be considered a clinical problem (Carvalheira, Brotto, & Leal, 2010).

The absolute frequency with which a person engages in sex cannot be used as a measure of inhibited sexual desire because the central issue is *interest*—actively seeking out sexual experiences—rather than participation (Warnock, 2002). For example, some people acquiesce to their partners' demands, even though they would not choose to engage in sexual activities if it were left up to them. In the absence of any specific standard, the identification of hypoactive sexual desire must depend on a clinician's subjective evaluation of the level of desire that is expected given the person's age, gender, marital status, and many other relevant considerations.

Almost everyone recognizes that sexual desire fluctuates in intensity over time, sometimes dramatically and frequently, for reasons that we do not understand. The fact that hypoactive sexual desire is listed in DSM-IV-TR as a type of disorder should not lead us to believe that it is a unitary condition with a simple explanation. It is, in fact, a collection of many different kinds of problems. People who suffer from low levels of sexual desire frequently experience other mental and medical disorders. Most men and women seeking treatment for hypoactive sexual desire report other forms of sexual dysfunction, such as problems with arousal or genital pain. Men and women with low sexual desire also have high rates of mood disorders. The mood disorder typically appears before the onset of low sexual desire. It appears likely, therefore, that many cases of low sexual desire develop after the person has experienced other forms of psychological distress.

Sexual Aversion Disorder Some people develop an active aversion to sexual stimuli and begin to avoid sexual situations altogether. This avoidance may be quite general, or it might be limited to certain aspects of sexual behavior, such as kissing, intercourse, or oral sex. This condition is quite different from a lack of interest. Hypoactive sexual desire disorder is defined by the *absence* of desire, while sexual aversion disorder is defined by the *presence* of fear and avoidance. Fear of sexual encounters can occasionally reach intense proportions, at which point it may be better characterized as **sexual aversion disorder.** In fact, this problem might best be viewed as a kind of phobia rather than a form of diminished sexual desire (Kingsberg & Janata, 2003). It may be moved out of the section on sexual dysfunctions in DSM-V because it seems to belong with the anxiety disorders (Brotto, 2010).

Male Erectile Disorder Many men experience difficulties either in obtaining an erection that is sufficient to accomplish intercourse or maintaining an erection long enough to satisfy themselves and their partners during intercourse. Both problems are examples of **erectile dysfunction.** Men with this problem may report feeling subjectively aroused, but the vascular reflex mechanism fails, and sufficient blood is not pumped to the penis to make it erect (Wylie & Machin, 2007). These difficulties can appear at any time prior to orgasm. Some men have trouble achieving an erection during sexual foreplay, whereas others lose their erection around the time of insertion or during intercourse. This phenomenon used to be called *impotence,* but the term has been dropped because of its negative implications.

Erectile dysfunctions can be relatively transient, or they can be more chronic. Occasional experiences of this type are not considered unusual. When they persist and become a serious source of distress to the couple, however, erectile difficulties can lead to serious problems. Consider, for example, the feelings expressed by the man and woman in our next case study, who were treated by Bernie Zilbergeld, an expert in the treatment of sexual dysfunction.

BRIEF CASE STUDY

Male Erectile Disorder

Norm and Linda are both 44 and have been married 15 years, the first marriage for him, the second for her. Linda called to ask if I would be available immediately to work with her and her husband, who had low sexual desire and erection problems. Individually, they are very different. Linda is attractive, vivacious, impulsive, and very critical. Norm seemed generally timid and reluctant to express his feelings in front of his wife, spoke slowly and usually only after some time, and then said only nice things in a nice way, and he struck me as being depressed. (They) had a serviceable relationship in many ways. Neither had close friends and tended to rely on each other for support and companionship. They shared a number of common interests and held similar values about most things.

The only problem, as far as they were concerned, was sex. When they first met, Linda was far more sexually experienced than Norm. She was surprised that a man could have had so little knowledge and experience. He, on the other hand, was somewhat intimidated by her experience and knowledge. But he tried to be a good student and they enjoyed frequent lovemaking at the beginning, although not as frequent or as passionate as she would have liked.

Over the years, however, Norm gradually lost interest in sex and developed erection problems. Either he wouldn't get an erection or he would lose it before or during insertion. Norm sought help from a urologist who, although he found nothing physically wrong, gave him an injection in his penis, which produced an erection that lasted for three hours.

But neither Norm nor Linda was overjoyed by the idea of him taking injections. Norm had always been fearful of needles and almost fainted when the urologist gave him the first injection. Linda didn't like the needles either. In her mind, they weren't "natural or normal." For Norm to have to take shots to get erections meant that he didn't love her or wasn't aroused by her.

Linda appeared to be hurt and angry in my individual session with her. "I know you're sympathetic toward men with erection problems. But what about me? I feel totally defeminized. How can I feel loved or desirable when he can't get it up for me? You don't have any idea what that's like. Even with the help of modern chemistry, he can't do it for me. It's obvious he doesn't want me, doesn't desire me. Even at my age, other men look at me and want me. But not my husband. I feel (awful)."

In my session with Norm, he repeated several times that he loved Linda and wanted to stay with her. When I asked if he found her sexually attractive, he hesitated and then said yes. When I asked about the hesitation, he was silent for a few moments and then said, "No, it's nothing. I am turned on to her." Further prodding on my part yielded nothing, but I thought his arousal was not as unambivalent as he said. As we continued our discussion, I asked how he felt when he was first dating her. He began by saying how beautiful he found her and how surprised he was that a woman like her would take an interest "in a nerd like me." When I asked what else he felt at that time, he answered, "To tell the truth, I was frightened by her experience and sexual openness. It was like I was in kindergarten and she was a professor. I'm not sure I've ever gotten over that. I've always felt at least a little inadequate. And things really got bad after I started having trouble with erections." I wanted to know more about how their sex had been before the erection problem. He said that it had been good and added in a voice so low I could barely hear his words, "She was always teaching and correcting me. Somehow I wasn't able to do exactly what she wanted. I didn't touch her right, my erections were never as hard as she liked, and when we had intercourse, I wasn't passionate enough" (Zilbergeld, 1995, pp. 315–316).

Norm and Linda experienced the frustrations and anxiety that often accompany sexual arousal difficulties. Their relationship also illustrates the marital distress that can develop when people begin to have problems with self-esteem and doubts about the affection of their partner.

Female Sexual Arousal Disorder Sexual arousal can also be impaired in women, but it is somewhat more difficult to describe and identify than is erectile dysfunction in men. Put simply, a woman is said to experience **inhibited sexual arousal** if she cannot either achieve or maintain genital responses, such as lubrication and swelling, that are necessary to complete sexual intercourse. The desire is there, but the responses that characterize sexual excitement are inhibited (Meston & Bradford, 2007).

The capacity for intercourse is less obvious and more difficult to measure for a woman than for a man, whose erect penis usually serves as a signal of readiness (see Research Methods box). Investigators who have studied sexual responses in normal women have reported low correlations between self-reports of subjective arousal and physiological measures, such as the amount of vaginal lubrication or vasocongestion (Meston, Rellini & McCall, 2010). Among women who experience sexual difficulties, the problem may more often be decreased subjective arousal

Sexual arousal is somewhat more difficult to measure for women than for men. Women's subjective feelings of arousal are not always directly connected to physiological responses.

RESEARCH METHODS

HYPOTHETICAL CONSTRUCTS: WHAT IS SEXUAL AROUSAL?

The term *sexual arousal* refers to the state that precedes orgasm. It is defined in terms of two factors: physiological responses, such as vascular engorgement of the genitals, and subjective feelings of pleasure and excitement. Psychologists refer to sexual arousal as a **hypothetical construct.** Many of the concepts that we have discussed in this book are hypothetical constructs: anxiety, depression, psychopathy, and schizophrenia. Hypothetical constructs are theoretical devices. In the field of psychopathology, they refer to events or states that reside within the person and are proposed to help us understand or explain a person's behavior.

Constructs cannot be observed directly, but in order to be scientifically meaningful they must be defined in terms of observable responses (Cronbach & Meehl, 1955; Kimble, 1989). These responses are all associated with the construct, but they are not perfectly related, and the construct is not exhaustively defined by them. For example, an erect penis is not always accompanied by subjective feelings of sexual excitement, and subjective feelings of arousal are not always associated with physiological responses. In other words, the construct of sexual arousal is anchored by feelings and responses that can be measured directly, but it is more than the sum of these parts.

An **operational definition** is a procedure that is used to measure a theoretical construct. Such a definition usually includes measures of the different components of the construct. For men, one obvious component of sexual arousal is penile erection. The most widely accepted procedure for measuring male sexual arousal uses a device called a *penile plethysmograph* (Rosen, Weigel, & Gendrano, 2007). In this procedure, the man places a thin elastic strain gauge around his penis, underneath his clothing. The rubber loop is filled with a column of mercury that changes in its electrical conductance as the circumference of the penis changes. The wire extending from the strain gauge is connected to a plethysmograph, which amplifies the electrical signal passing through the strain gauge and produces a record reflecting changes in penile tumescence.

The *vaginal photometer,* a device shaped like a tampon and inserted into the vagina, is used to measure female sexual arousal. Like the penile strain gauge, the photometer can be placed in position in private and worn underneath clothing during the assessment procedure. As the woman becomes sexually aroused, the walls of the vagina become congested with blood. Vasocongestion causes changes in the amount of red light that can be transmitted through the tissue. The photometer is sensitive to subtle changes in vaginal tissue and is probably most useful in measuring moderate to low levels of sexual arousal (Janssen, 2002; Prouse & Heiman, 2009).

How is sexual arousal measured in the lab?

Clinical scientists must always think carefully about the meaning of their operational definitions. Although the penile strain gauge and the vaginal photometer measure physiological events that are directly related to sexual arousal, the responses that they measure are not the same thing as sexual arousal. They are reflections of the construct, which has many dimensions (Berman et al., 1999). One important goal of scientific studies is to determine more specifically how (and when) these physiological measures are related to the other observable referents of sexual arousal. This process will determine the **construct validity** of the penile strain gauge and the vaginal photometer—that is, the extent to which these specific measures produce results that are consistent with the theoretical construct.

rather than impaired physiological responses. The distinction between desire and subjective arousal is difficult to make. Therefore, some experts have argued that hypoactive sexual desire and sexual arousal disorder should be combined into one diagnostic category for women (Basson & Brotto, 2009).

Premature Ejaculation Many men experience problems with the control of ejaculation. They are unable to prolong the period of sexual excitement long enough to complete intercourse. This problem is known as **premature ejaculation,** but most experts now prefer the term "early ejaculation" because it is less pejorative. Once they become intensely sexually aroused, they reach orgasm very quickly (Metz & Pryor, 2000). Almost all the literature on this topic is concerned with men, but some women are also bothered by reaching orgasm too quickly. Therefore, some clinicians have suggested that "early orgasm" might be a more appropriate description of the problem.

There have been many attempts to establish specific, quantitative criteria for premature ejaculation (Broderick, 2006). None of the attempts has been entirely satisfactory, but certain boundaries identify conditions that can be problematic. If the man ejaculates before or immediately upon insertion, or after only three or four thrusts, almost all clinicians will identify his response as premature ejaculation. Among men suffering from lifelong premature ejaculation, 90 percent routinely ejaculate within 1 minute after insertion of the penis in the vagina (Waldinger, 2009).

Another way to think about premature ejaculation places emphasis on subjective control and the couple's satisfaction rather than on the amount of time required to reach orgasm. The DSM-IV-TR definition defines the problem in terms of recurrent ejaculation shortly after penetration and before the person wishes it. If progression to orgasm is beyond the man's voluntary control once he reaches an intense level of

sexual arousal, he has a problem with premature ejaculation (Symonds et al., 2003).

Female Orgasmic Disorder Some women are unable to reach orgasm even though they apparently experience uninhibited sexual arousal. Women who experience orgasmic difficulties may have a strong desire to engage in sexual relations; they may find great pleasure in sexual foreplay and may show all the signs of sexual arousal. Nevertheless, they cannot reach the peak erotic experience of orgasm. Women whose orgasmic impairment is *generalized* have never experienced orgasm by any means. *Situational orgasmic* difficulties occur when the woman is able to reach orgasm in some situations, but not in others. That might mean that she is orgasmic during masturbation but not during intercourse, or perhaps she is orgasmic with one partner but not with another (Basson, 2002).

Orgasmic disorder in women is somewhat difficult to define in relation to inhibited sexual arousal because the various components of female sexual response are more difficult to measure than are erection and ejaculation in the male. One experienced researcher described this issue in the following way:

> In my experience, many women who have never reached orgasm present the following set of symptoms: They report that when engaging in intercourse they do not have difficulty lubricating and experience no pain. However, they report no genital sensations (hence the term *genital anesthesia*) and do not appear to know what sexual arousal is. Typically they do not masturbate and often have never masturbated. They do not experience the phenomenon that a sexually functional woman would call sexual desire. Most of these women seek therapy because they have heard from others or have read that they are missing something, rather than because they themselves feel frustrated. (Morokoff, 1989, p. 74)

Pain During Sex Some people experience persistent genital pain during or after sexual intercourse, which is known as **dyspareunia.** The problem can occur in either men or women, although it is considered to be much more common in women (Davis & Reissing, 2007). The severity of the discomfort can range from mild irritation following sexual activity to searing pain during insertion of the penis or intercourse. The pains may be sharp and intense, or they may take the form of a dull, aching sensation; they may be experienced as coming from a superficial area near the barrel of the vagina or as being located deep in the lower abdominal area; they may be intermittent or persistent. The experience of severe genital pain is often associated with other forms of sexual dysfunction. Not surprisingly, many women with dyspareunia develop a lack of interest in, or an aversion toward, sexual activity.

The following first-person account was written by a 40-year-old woman who had been experiencing vaginal pain for several months. She had consulted several different health professionals about the problem, and none of their treatments had relieved her discomfort. This passage describes her experience one night when she and the man with whom she had been living seemed to be on the brink of enjoying a renewed interest in their sexual relationship.

BRIEF CASE STUDY

Genital Pain

We went to bed. For a while it was nice—more than nice. It was novel and thrilling, as if we had just met. We hadn't approached each other in more than a month. I was surprised by how wonderful I could feel. I was used to feeling lousy most of the time. The sensations of excitement were overwhelming. I'd forgotten about that. Then he pushed himself into me and it was horrible.

First I felt as if I were being torn or sliced. As he settled into a rhythm, I felt that something was scraping me over and over in the same raw spot, until the rawness and soreness were all I could feel. He didn't notice. He was intent on what he was doing. I decided to let him get on with it, but the pain was really bothering me. I pulled away inside myself, so that the events on the bed were far from where "I" was, and the pain was far away also. That worked, but I didn't like doing it. There was something nasty about it. I had the thought, People who don't like sex must feel this way. Then I realized that now I was somebody who didn't like sex (Kaysen, 2001, pp. 60–61).

Access to the vagina is controlled by the muscles surrounding its entrance. Some women find that whenever penetration of the vagina is attempted, these muscles snap tightly shut, preventing insertion of any object. This involuntary muscular spasm, known as **vaginismus,** prevents sexual intercourse as well as other activities, such as vaginal examinations and the insertion of tampons. Women with vaginismus may be completely sexually responsive in other respects, fully capable of arousal and orgasm through manual stimulation of the clitoris. Women who seek therapy for this condition often report that they are afraid of intercourse and vaginal penetration (Reissing et al., 2004). The problem can be severe or partial in nature. Some couples report that a mild form of vaginismus occurs from time to time, making intercourse difficult and sometimes painful.

Many women experience genital pain during sexual stimulation other than intercourse. Thus, one recommendation made by the Consensus Development Conference on Female Sexual Dysfunction was the addition of a new category for noncoital sexual pain (Basson et al., 2000). This suggestion was made because the DSM-IV-TR definitions of dyspareunia and vaginismus focus exclusively on problems that occur during sexual intercourse. Some experts have suggested that these problems should be viewed as genital pain disorders (similar to pain disorders, such as back pain) that interfere with intercourse rather than as forms of sexual dysfunction (Binik, 2005).

Hypersexual Disorder: A Proposal DSM-IV-TR includes unusually low sexual desire as a sexual dysfunction, but it does not mention unusually *high* sexual desire. In contrast, ICD-10 does include a category called *excessive sexual drive.* Symptoms associated with this condition presumably include such behaviors as seeking new sexual encounters out of boredom with old ones, frequent use of pornography, and legal problems resulting from sexual behaviors. Additional features include obsessive thoughts about sexual encounters, guilt resulting from

problematic sexual behavior, and rationalization for continued reckless sexual behavior. Reckless and uncontrolled sexual activity can obviously disrupt a person's life and cause significant personal distress. But should it be considered a form of abnormal behavior? Some experts believe that the next version of DSM should include some form of this concept (Kafka, 2010).

One approach to the problem views uncontrolled sexual behavior as being similar to an addiction (Bancroft & Vukadinovic, 2004). Consider, for example, the definition of substance dependence presented in Table 11.3 on page 284. Replace the word *substance* with the term *sexual behavior* in this definition and you have one version of a definition of hypersexual disorder. Perhaps most important is the development of a repeated pattern of compulsive sexual behavior. The person spends excessive time being consumed by sexual fantasies and urges, reports repeated unsuccessful attempts to reduce the fantasies and behaviors, and continues to engage in sexual behaviors without regard to the potential for physical or emotional harm to the self or others. The person may also develop a tolerance to previous sexual partners and forms of sexual experience. Some clinicians suggest that sexual addiction can progress from victimless behaviors, such as excessive masturbation, to increasingly harmful forms of behavior, such as exhibitionism, voyeurism, and sexual assault.

There are also some reasons to be skeptical of this concept. One is the somewhat circular definition of the problem, which also plagues the concept of *impulse control disorders* (see page 237 in Chapter 9). Another limitation is the heterogeneous nature of excessive or uncontrolled sexual behavior. Failure to control sexual impulses can be associated with several other disorders, including paraphilias, impulse control disorders, and bipolar mood disorder (Levine, 2010). Many people who admit to compulsive sexual behavior also suffer from major depression, anxiety disorders, and substance use disorders (Black et al., 1997; Guiliano, 2009). The concept obviously includes a diverse set of behavioral problems. The addition of hypersexual disorder to DSM-V should depend on the balance of evidence supporting the construct validity of the disorder (see Research Methods).

FREQUENCY

Surveys conducted among the general population indicate that some forms of sexual dysfunction are relatively common (Christensen et al., 2011). We must keep in mind, however, that this impression is based on self-report questionnaires and judgments made by laypersons, which are less precise than those made by experts. Diagnoses made by experienced therapists would take into account the person's age, the context of the person's life, and whether the person had experienced stimulation that would ordinarily be expected to lead to sustained arousal and orgasm. Clinicians would also take into consideration the amount of distress and interpersonal difficulty associated with the problem before arriving at a diagnosis of sexual dysfunction. Therefore, we must be cautious in our interpretations of survey data (Hayes et al., 2008).

The most extensive set of information regarding sexual problems among people living in the community comes from the National Health and Social Life Survey (NHSLS). Each participant was asked whether during the past 12 months he or she had experienced "a period of *several months or more* when you lacked interest in having sex; had trouble achieving or maintaining an erection or (for women) had trouble lubricating; were unable to come to a climax; came to a climax too quickly; or experienced physical pain during intercourse." For each item, the person was asked for a simple yes or no response. Figure 12.3 indicates the overall percentage of men and women who indicated that they had experienced each of these specific problems. There are obviously significant gender differences in the prevalence of all types of problems. Premature ejaculation is the most frequent form of male sexual dysfunction, affecting almost one out of every three adult men. All the other forms of sexual dysfunction are reported more often by women. One-third of women said that they lacked interest in sex, and almost one-quarter indicated that they experienced a period of several months during which they were unable to reach orgasm (Laumann, Paik, & Rosen, 1999).

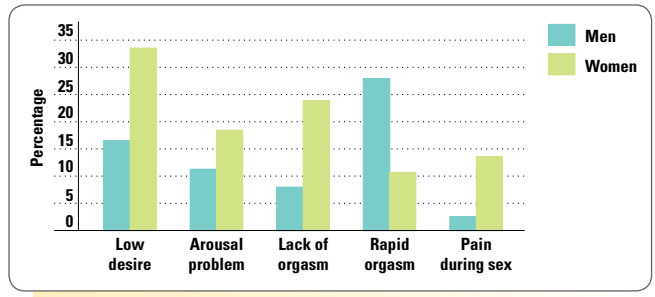


FIGURE 12.3 Prevalence of Sexual Dysfunctions

This graph shows the percentage of NHSLS respondents who reported having sexual difficulties at some time during the previous 12 months. Note the differences in the problems reported by men and women.

Source: E. O. Laumann, J. H. Gagnon, R. T. Michael, and S. Michaels, 1994, *The Social Organization of Sexuality: Sexual Practices in the United States.* Chicago: University of Chicago Press. p. 369. Copyright © 1994. Reprinted by permission of The University of Chicago Press.

Should excessive sexual behavior be considered a disorder in its own right? Or is it a symptom of other mental disorders?

Sexual Behavior Across the Life Span Sexual behavior changes with age. Masters and Johnson devoted considerable attention to this topic in their original studies. Their data challenged the myth that older adults are not interested in, or capable of performing, sexual behaviors. The NHSLS data also indicate that many people remain sexually active later in life. Gender differences become marked in the late fifties, when rates of inactivity increase dramatically for women. Between ages 70 and 74, 65 percent of men are still sexually active, compared to only 30 percent of women. These differences may be, at least partly, the result of differential mortality rates (men die earlier, so many women lose their partners) as well as biological factors that are part of the aging process. They may also reflect the influence of a cultural prejudice against sexual activity among older women.

Differences between younger and older people are mostly a matter of degree. As men get older, they tend to achieve erections more slowly, but they can often maintain erections for longer periods of time. Older men find it more difficult to regain an erection if it is lost before orgasm. As women get older, vaginal lubrication may occur at a slower rate, but the response of the clitoris remains essentially unchanged. The intensity of the subjective experience of orgasm is decreased for older men and women. For both men and women, healthy sexual responsiveness is most likely to be maintained among those who have been sexually active as younger adults (Herbinek et al., 2010).

The prevalence of certain types of sexual dysfunctions increases among the elderly, particularly among men (DeRogatis & Burnett, 2008). In the NHSLS, for example, the proportion of men reporting erectile problems increased from 6 percent in the 18–24 age range to 20 percent in the 55–59 age range. In contrast, several types of sexual problems actually declined in frequency among older women. Women in the 55–59 age range were less likely than women between the ages of 18 and 24 to report pain during sex or inability to reach orgasm, although they did report a slight increase in trouble with lubrication during sexual activity.

The relation between sexual experience and aging is closely related to other health problems that increase with age. People who rate their health as being excellent have many fewer sexual problems than people who rate their health as being only fair or poor (Laumann, Das, & Waite, 2008).

Cross-Cultural Comparisons Patients with sexual disorders seek treatment at clinics all over the world (Steggall, Gann, & Chinegwundoh, 2004). Therefore, these problems are not unique to any particular culture. Cultural and ethnic differences have been reported for sexual practices, beliefs about sexuality, and patterns of sexual decision making. For example, Asians are more conservative than Caucasians in many regards, such as the prevalence and frequency of masturbation (Meston, Trapnell, & Gorzalka, 1996). It is not clear whether variations in sexual behavior are accompanied by cultural differences in the frequency and form of sexual dysfunctions. Cross-cultural studies of prevalence rates for specific sexual dysfunctions have not been reported. This kind of investigation may be difficult to perform because the DSM-IV-TR definitions of sexual dysfunctions may not be well suited to describing the sexual experiences and satisfaction of people living in non-Western cultures (Ghanem & El-Sakka, 2007).

CAUSES

At each stage of the sexual response cycle, a person's behavior is determined by the interaction of many biological and psychological factors, ranging from vasocongestion in the genitals to complex cognitive events involving the perception of sexual stimuli and the interpretation of sexual meanings. Interference with this system at any point can result in serious problems. In the following pages we review some of the factors that contribute to the etiology of various types of sexual dysfunctions.

Biological Factors The experience of sexual desire is partly controlled by biological factors. Sexual desire is influenced by sex hormones for both men and women (LeVay & Valente, 2003). Testosterone is particularly important for male sexual desire. Studies of men with inadequate levels of sex hormones show an inhibited response to sexual fantasies, but they are still able to have erections in response to viewing explicit erotic films. The influence of male sex hormones on sexual behavior is, therefore, thought to be on sexual appetite rather than on sexual performance. This process probably involves a threshold level of circulating testosterone (Schiavi & Segraves, 1995). In other words, sexual appetite is impaired if the level of testosterone falls below a particular point (close to the bottom of the laboratory normal range), but above that threshold, fluctuations in testosterone levels will not be associated with changes in sexual desire. The reduction of male sex hormones over the life span probably explains, at least in part, the apparent decline in sexual desire among elderly males.

Many people remain sexually active later in life. Differences in sexual responsiveness between younger and older people are mostly a matter of degree.

Many cases of erectile dysfunction can be attributed to vascular, neurological, or hormonal impairment (Goldstein, 2004). Erection is the direct result of a threefold increase in blood flow to the penis. Thus, it is not surprising that vascular diseases, which may affect the amount of blood reaching the penis, are likely to result in erectile difficulties. Neurological diseases, such as epilepsy and multiple sclerosis, can also produce erectile difficulties, because erection depends on spinal reflexes. Diabetes may be the most common neurologically based cause of impaired erectile responsiveness.

Various kinds of drugs can also influence a man's erectile response (Clayton & West, 2003). One interesting set of results indicates that men who smoke cigarettes are more likely to experience erectile difficulties than are men in the general population. Many other drugs, including alcohol and marijuana, may have negative effects on sexual arousal.

A number of biological factors can impair a woman's ability to become sexually aroused (Clayton, 2007). Various types of neurological disorders, pelvic disease, and hormonal dysfunction can interfere with the process of vaginal swelling and lubrication. Although relatively little research has been conducted on sexual arousal in women, there is evidence to suggest that genetic factors influence the frequency with which women are able to experience orgasm (Dawood et al., 2005).

Inhibited orgasm, in both men and women, is sometimes caused by the abuse of alcohol and other drugs. The problem may improve if the person is able to stop drinking and maintain a stable period of sobriety (Schiavi et al., 1995). Orgasm problems can also be associated with the use of prescribed forms of medication. For example, as we discussed in Chapter 5, many people who take SSRIs such as fluoxetine (Prozac) for the treatment of depression have difficulty achieving orgasm as a side effect (Werneke, Northey, & Bhugra, 2006).

Psychological Factors Although sexual desire is rooted in a strong biological foundation, psychological variables also play an important role in the determination of which stimuli a person will find arousing. Sexual desire and arousal are determined, in part, by mental scripts that we learn throughout childhood and adolescence (Middleton, Kuffel, & Heiman, 2008; Wiegel, Scepkowski, & Barlow, 2007). These scripts provide structure or context to the otherwise confusing array of potential partners who might become the object of our desires. In other words, there are certain kinds of people to whom we may be sexually attracted, and there are certain circumstances in which sexual behavior is considered appropriate. According to this perspective, the personal meaning of an event is of paramount importance in releasing the biological process of sexual arousal. Both members of the potential couple must recognize similar cues, defining the situation as potentially sexual in nature, before anything is likely to happen.

Beliefs and attitudes toward sexuality, as well as the quality of interpersonal relationships, have an important influence on the development of low sexual desire, especially among women (Nobre & Pinto-Gouveia, 2006). Women seeking treatment for hypoactive sexual desire report negative perceptions of their parents' attitudes regarding sexual behavior and the demonstration of affection. In comparison to other women, they also indicate that they feel less close to their husbands, have fewer romantic feelings, and are less attracted to their husbands. The quality of the relationship is an important factor to consider with regard to low sexual desire (Metz & Epstein, 2002).

Culturally determined attitudes toward sexual feelings and behaviors can also have a dramatic impact on women's ability to become sexually aroused (Al-Sawaf & Al-Issa, 2000). Some societies openly encourage female sexuality; others foster a more repressive atmosphere. Within U.S. culture, there are tremendous variations with regard to women's ability to experience and express their sexuality. For example, many women feel guilty about having sexual fantasies, in spite of the fact that such fantasies are extremely common. Women who feel guilty about fantasizing while they are having intercourse are more likely to be sexually dissatisfied and to encounter sexual problems. The most important factors contributing to failure to reach orgasm involve negative attitudes, feelings of guilt, and failure to communicate effectively (Kelly, Strassberg, & Turner, 2004).

What role do mental scripts play in sexual arousal?

Couples that experience communication problems, power conflicts, and an absence of intimacy and trust are more likely than others to experience sexual problems. Lack of assertiveness and lack of comfort in talking about sexual activities and pleasures are associated with various types of female sexual dysfunctions (Rosen & Leiblum, 1995). The following brief case study provides one example of serious relationship difficulties that were experienced by one couple in which the woman, a married, 34-year-old lawyer, was being treated for long-standing vaginismus as well as alcohol dependence.

BRIEF CASE STUDY

Vaginismus and Alcohol Dependence

Gina speculated that living with Paul exacerbated her sexual anxieties, and she became increasingly dependent on alcohol to "loosen her up" sexually. Paul was sexually naive and did not press Gina to have intercourse, especially when she so visibly panicked at the approach of his penis. He, too, was sexually anxious and was afraid of inflicting pain on her. Sexually, they depended on drinking to disinhibit them, and they developed a sexual script that relied on manual stimulation and oral sex. Although sexual contact was relatively infrequent, both were reasonably content.

This state of affairs continued for many years. It was not without its costs, though. Gina felt inadequate and deficient as a woman and avoided gynecological examinations. Paul would occasionally become enraged at a seemingly small provocation and verbally attack Gina. Internally, he reported feeling humiliated, emasculated, and ashamed about the nonconsummation of their marriage. When his coworkers teased and joked about "getting it on" sexually, he felt alone in the private knowledge that he had never penetrated his wife despite 13 years of living and sleeping together.

Eventually, as Gina's drinking escalated, the marital conflict grew intolerable. When Gina was drunk, she would verbally berate and abuse Paul. Her attacks and complaints about his passivity and lack of assistance with housework and her disparagement of his passion for sports undermined the earlier closeness they had experienced. Although he would usually tolerate her drunken tirades silently, he began to blow up more readily (Leiblum, 1995, p. 256).

Previous harmful or traumatic experiences can also have an important effect on various aspects of sexual interests and arousal. A previous history of sexual abuse can lead to sexual aversion, and it can interfere with a woman's ability to become sexually aroused (Najman et al., 2005). Premature ejaculation and low sexual desire in men have also been linked to various kinds of long-lasting, adverse relationships with adults during childhood (Loeb et al., 2002). For example, boys who grow up in a home in which their father is physically abusive may learn to associate sex with violence and become convinced that they do not want to function—sexually or interpersonally—as their father had.

Performance anxiety and fear of failure are among the most important psychological factors contributing to impaired sexual arousal. People who have experienced inhibited sexual arousal on one or two occasions may be likely to have further problems to the degree that these difficulties make them more self-conscious or apprehensive regarding their ability to become aroused in future sexual encounters. Several prominent and experienced sex therapists have assumed that anxiety and sexual arousal are incompatible emotional states. People who are anxious will presumably be less responsive to sexual stimuli. And men who have sexual arousal disorders are more likely to report feeling high levels of performance anxiety (McCabe, 2005).

Anxiety disrupts sexual performance to the extent that it alters certain cognitive processes. Several studies have compared the responses of sexually dysfunctional men with those of control subjects in laboratory settings. Dysfunctional men experience more negative emotions in the presence of erotic stimuli, and they are also more likely to shift their attention from the arousing properties of sexual stimuli to the threatening consequences of potential failures in sexual performance (Bach, Brown, & Barlow, 1999). In comparison to men without erectile disorder, men with sexual dysfunction rate negative sexual events as being more important and then are more likely to attribute the problem to something about themselves rather than external considerations (Scepkowski et al., 2004).

What are the primary targets of psychological approaches to treating sexual dysfunction?

TREATMENT

Masters and Johnson (1970) were pioneers in developing and popularizing a short-term, skills-based approach to the treatment of sexual dysfunctions. Hundreds of couples who visited their clinic in St. Louis went through a two-week course of assessment and therapy in which they became more familiar with their bodies, learned to communicate more effectively with their partners, and received training in procedures designed to help them diminish their fears about sexuality. The results of this treatment program were very positive and quickly spawned a burgeoning industry of psychosocial treatment for sexual dysfunction. Getting Help at the end of this chapter discusses some of the options and resources available to anyone experiencing problems in sexual functioning or health.

Psychological Procedures Psychological treatments for sexual dysfunction address several of the causes discussed earlier, especially negative attitudes toward sexuality, failure to engage in effective sexual behaviors, and deficits in communication skills. Sex therapy centers around three primary types of activities: sensate focus and scheduling; education and cognitive restructuring; and communication training (Meston & Rellini, 2008; Wincze, Bach, & Barlow, 2008).

Sensate focus exercises help people become aware of physical sensations that are associated with touching and being touched while minimizing demands for sexual performance.

The cornerstone of sex therapy is known as **sensate focus,** a series of simple exercises in which the couple spends time in a quiet, relaxed setting, learning to touch each other. They may start with tasks as simple as holding hands or giving each other back rubs. The rationale for sensate focus hinges on the recognition that people with sexual problems must learn to focus on erotic sensations rather than on performance demands. The goal is to help them become more comfortable with this kind of physical sharing and intimacy, to learn to relax and enjoy it, and to talk to each other about what feels good and what does not.

Another facet of psychological approaches to treating sexual dysfunction involves *scheduling*. This is, in fact, closely related to sensate focus because the technique of sensate focus requires that people schedule time for sex. Couples need a quiet, relaxed, and private environment in order to engage in pleasurable and satisfying sexual behavior.

A third aspect of sex therapy involves education and cognitive restructuring—changing the way in which people think about sex. In many cases the therapist needs to help the couple correct mistaken beliefs and attitudes about sexual behavior. Examples are the belief that intercourse is the only true form of sex, that foreplay is an adolescent interest that most adults can ignore, and that simultaneous orgasm is the ultimate goal of intercourse. Providing information about sexual behaviors in the general population can often help alleviate people's guilt and anxiety surrounding their own experiences. Some people are relieved to know that they are not the only ones who fantasize about various kinds of sexual experiences, or that the fact that they fantasize about these things does not mean that they are going to be compelled to behave in deviant ways.

The final element of psychological treatment for sexual dysfunction is communication training. Many different studies have indicated that people with sexual dysfunction often have deficits in communication skills. They find it difficult to talk to their partners about matters involving sex, and they are especially impaired in the ability to tell their partners what kinds of things they find sexually arousing and what kinds of things turn them off. Therefore, sex therapists often employ structured training procedures aimed at improving the ways in which couples talk to each other.

The outcome results of psychological treatment programs for sexual disorders have generally been considered to be positive (Dutere, Segraves, & Althof, 2007). Early reports from Masters and Johnson's clinic were especially glowing. One summary of their results reported an overall success rate of 85 percent for male patients and 78 percent for female patients. Unfortunately, more recent studies have reported less positive results. Serious questions have been raised about the adequacy of the research methods employed in several outcome studies. Interventions have not been standardized, sample sizes have been relatively small, and long-term follow-up data are often lacking. Therefore, although psychological treatments for sexual dysfunction are frequently successful, empirical support for the efficacy of these procedures is not strong (Heiman, 2002; O'Donohue et al., 1999). Better studies are clearly needed.

Important questions have also been raised about the utility of these procedures for clients in other cultures. Clinics in India, Iran, Japan, Saudi Arabia, and South Africa report that men and women from many different backgrounds seek help for sexual dysfunctions (Verma, Khaitan, & Singh, 1998). Culture dictates the ways in which sexual issues may be discussed, and beliefs about sexuality and reproduction influence decisions about acceptable sexual behaviors. These beliefs vary extensively across cultures. For example, people in some Asian cultures believe that a man's health can be damaged through unnecessary loss of semen (Davis & Herdt, 1997). Such concerns may prohibit use of masturbation as a therapeutic exercise. Implicit rules governing communication patterns between partners are also determined by culture. Some societies value and encourage sharp differences in gender roles, with men being expected to make decisions about the timing and type of sexual activity (Quadagno et al., 1998). Therefore, communication training must be tailored to meet the expectations that each couple holds regarding the nature of their relationship. Mental health professionals must give careful consideration to their clients' cultural background when they conduct an assessment and design a treatment program.

Biological Treatments Biological treatments—primarily medications—are also useful in the treatment of sexual dysfunctions. This is especially true for erectile disorder, the most frequent sexual problem for which men seek professional help. Sildenafil citrate (Viagra) was approved by the FDA in 1998 for the treatment of erectile dysfunction and quickly became one of the most popular drugs on the market. Competing pharmaceutical companies soon developed and began vigorously promoting similar drugs known as tadalafil (Cialis) and vardenafil (Levitra). All three drugs are phosphodiesterase-5 (PDE-5)[1] inhibitors that facilitate erection by increasing blood flow to certain areas of the penis. They increase the man's ability to respond to stimuli that he would ordinarily find sexually arousing, but they do not influence overall sexual desire (Edwards et al., 2006).

Double-blind, placebo-controlled studies have evaluated the use of Viagra in men with erectile problems associated with various conditions, including hypertension, diabetes, and coronary artery disease. It is effective, increasing the number of erections for approximately two-thirds of men with severe erectile dysfunction (Fink et al., 2002). Unfortunately, some men experience negative side effects, such as headache, facial flushing, nasal congestion, and altered vision. Perhaps most important, Viagra can lead to sudden drops in blood pressure if taken with various forms of medication known as "nitrates," which are used in the treatment of heart disease. Some deaths were reported after Viagra was introduced because of this misuse. The research evidence indicates that Viagra and other PDE-5 inhibitors should be used in combination with psychological treatments for sexual dysfunction (see Critical Thinking Matters).

Pharmaceutical companies are also developing and evaluating medications that might be used to treat sexual dysfunction in women (Korda, Goldstein, & Goldstein, 2010; van der Made et al., 2009). One product, known as Intrinsa, is a patch that delivers testosterone through the skin and could serve to increase sexual desire, especially in post-menopausal women and those who have had their ovaries removed. The FDA decided in 2004 to delay approval for Intrinsa because it did not have enough information about its long-term safety, particularly regarding increased risk for cancer and cardiovascular disease. The use of testosterone could also lead to other side effects, such as facial hair growth, deepening of the voice, and the development of other masculine features in women.

Another less frequently used procedure for the treatment of erectile dysfunction involves surgically inserting a penile implant (or prosthesis), which can be used to make the penis rigid during intercourse (Melman & Tiefer, 1992; Schwartz et al., 2000). Several devices have been used. One option is a semi-rigid silicone rod that the man can bend into position for intercourse. Another device is hydraulic and can be inflated for the purpose of sexual activity. The man squeezes a small pump, which forces fluid into the inflatable cylinder and produces an erection. The inflatable device is preferred by partners, but it is also more expensive and can lead to more frequent postsurgical complications, such as infection.

The various forms of treatment that are available for the treatment of sexual dysfunction are certainly promising.

[1]PDE-5 is an enzyme that metabolizes nitric oxide, which triggers sexual arousal.

Critical Thinking Matters

DOES MEDICATION CURE SEXUAL DYSFUNCTION?

Can you remember watching a sporting event on television without seeing an advertisement for Cialis or Levitra? Attractive men and women cuddle and smile as they talk enthusiastically about the satisfaction that can be achieved with pharmacologically induced *strong, lasting erections.* It's difficult to imagine a new form of treatment for a psychological disorder that has been promoted more aggressively, or achieved a more dramatic impact on public awareness, than the PDE-5 inhibitors. In a few short years, they have generated a market that is estimated to approach $2 billion per year. They've become very popular, but do these pills offer a quick fix for all people suffering from arousal disorders?

Is an erect penis the most important consideration in a satisfying sexual relationship? What other ingredients are necessary?

Viagra and similar medications are clearly an important option for men with erectile problems. Countless men and their partners are grateful for their beneficial effects. Unfortunately, in many other cases, they are not a complete solution to sexual dysfunction in the absence of additional treatment. Couples that have experienced sexual problems have often struggled with a number of difficult issues for several years. Increasing the man's capacity for erection will address only one part of the problem. As one expert puts it, "Viagra can increase blood flow to the penis, but it doesn't create intimacy, love, or desire" (Morgentaler, 2003). Most experts recommend a treatment approach that combines the use of medication with cognitive behavior therapy. Therapists need to work with couples to improve intimacy and communication while also helping them to overcome frustrations and anxiety that have accumulated over years (McCarthy, 2004; Rosen, 2000).

A related product for women, which the media have nicknamed the *female Viagra,* may be available soon. Intrinsa, a patch that delivers testosterone through the skin, is being developed to address low desire, the most frequent sexual problem reported by women (see Figure 12.3). Small doses of testosterone can increase sexual desire in some women who have had their ovaries removed, but it seems unlikely that they will be effective with women who have lost interest in sex because of relationship difficulties or other motivational and cognitive factors. Problems with fatigue, scheduling difficulties, anxiety, and low self-esteem are all issues that don't go away simply because testosterone levels increase. Medication may facilitate some of the biological functions that are necessary prerequisites for healthy sexual behaviors, but it cannot guarantee that people will find their partners appealing or that sex will be pleasurable. Consumers and medical professionals all need to think critically about the complex factors that contribute to sexual dysfunction. We should not expect to find a magic bullet that will cure them all at once.

They offer several constructive options for people who are experiencing distress as a consequence of problems in sexual desire or performance. That is the good news with regard to sexual disorders. The bad news is concerned with another type of sexual disorder, which is known collectively as the paraphilias. They are less well understood, in comparison to the sexual dysfunctions, and they are also more difficult to treat. The next section of this chapter reviews the current state of our knowledge regarding these difficult problems.

Paraphilias

For some people, sexual arousal is strongly associated with unusual things and situations, such as inanimate objects, sexual contact with children, exhibiting their genitals to strangers, or inflicting pain on another person. These conditions are known as **paraphilias.** Literally translated, paraphilia means "love" (*philia*) "beyond the usual" (*para*). This term refers to conditions that were formerly called perversions or sexual deviations. According to DSM-IV-TR, the central features of all paraphilias are persistent sexual urges and fantasies that are associated with (1) nonhuman objects, (2) suffering or humiliation of oneself or one's partner, or (3) children or other nonconsenting persons.

Current discussions concerned with these conditions draw an important distinction between the sexual urges and fantasies and the diagnosis of a disorder (Blanchard, 2010). The next version of the diagnostic manual, DSM-V, will place additional emphasis on this issue by recognizing both *paraphilias* (non-normative forms of sexual arousal and behavior) and *paraphilic disorders*. The latter will be diagnosed only when a paraphilia leads to significant distress or impairment. One consequence of this change may be increased recognition

that some forms of non-normative sexual behaviors, such as fetishism and sexual masochism, may not necessarily be pathological if they are practiced voluntarily by consenting adults (Wright, 2010).

In the following pages we summarize a few of the most common types of paraphilias, and we consider some of the factors that might influence the development of unusual sexual preferences.

SYMPTOMS

One hundred years ago, many psychiatrists considered any type of sexual behavior other than heterosexual intercourse to be pathological. Contemporary researchers and clinicians have expanded the boundaries of normal behavior to include a much broader range of sexual behavior. A large proportion of men and women engage in sexual fantasies and mutually consenting behaviors such as oral sex. These experiences enhance their relationships without causing problems (Herbenick et al., 2010).

Problems with sexual appetites arise when a pattern develops involving a long-standing, unusual erotic preoccupation that is highly arousing, coupled with a pressure to act on the erotic fantasy. DSM-IV-TR requires that the erotic preoccupation must have lasted at least six months before the person would meet diagnostic criteria for a paraphilia. Furthermore, the diagnosis of paraphilia is made only if the person's paraphilic urges lead to clinically significant distress or impairment. The person would be considered to be impaired if the urges have become compulsory, if they produce sexual dysfunction, if they require the participation of nonconsenting persons, if they lead to legal problems, or if they interfere with social relationships. For several specific types of paraphilias, the person would qualify for a diagnosis if he acted on the urge (Hilliard & Spitzer, 2002). These include pedophilia, exhibitionism, voyeurism, and frotteurism (see descriptions in the following pages). For sexual sadism, acting on the urge would qualify the person for a diagnosis only if the partner had not consented to the activity. Acting on the other forms of paraphilic urges (masochism, fetishism, and transvestic fetishism) would not be sufficient for a diagnosis unless the urges of fantasies lead to significant personal distress or interfere with the person's ability to function.

It is actually somewhat misleading, or imprecise, to say that paraphilias are defined solely in terms of reactions to unusual stimuli. The central problem is that sexual arousal is dependent on images that are detached from reciprocal, loving relationships with another adult (Levine, Risen, & Althof, 1990). Themes of aggression, violence, and hostility are common in paraphilic fantasies, as are impulses involving strangers or unwilling partners. Rather than focusing on whether the stimuli are common or uncommon, some experts place principal emphasis on the lack of human intimacy that is associated with many forms of paraphilias (Moser, 2001).

Compulsion and lack of flexibility are also important features of paraphilic behaviors. Paraphilias may take up a lot of time and consume much of the person's energy. In that sense, they are similar to the addictions. People with paraphilic disorders are not simply aroused by unusual images or fantasies. They feel *compelled* to engage in certain acts that may be personally degrading or harmful to others, in spite of the fact that these actions are often repulsive to others and are sometimes illegal. The following case describes some of the central features of paraphilias.

BRIEF CASE STUDY

Paraphilia

For the past 40 years, Jon has masturbated to images of barely clad women violently wrestling each other. Periodically throughout his marriage, he has tried to involve his wife in wrestling matches with her friends and, eventually, with their adolescent daughter. When Jon was drunk, he occasionally embarrassed his wife by trying to pick fights between her and other women. On summer vacations, he sometimes jokingly suggested the women wrestle. During much of his sober life, however, his daydreams of women wrestling were private experiences that preoccupied only him. He amassed a collection of magazines and videotapes depicting women wrestling, to which he would resort when driven by the need for excitement.

Jon presented for help with his inability to maintain his erection with his wife for intercourse. With the exception of procreational sex, he was not able to consummate his long marriage. He was able to (become) erect if his wife described herself wrestling other women while he stimulated his penis in front of her, but he always lost his erection when intercourse was attempted (Levine, Risen, & Althof, 1990).

This case illustrates the way in which paraphilias can interfere with a person's life, especially relationships with other people. Jon's preoccupation with fantasies of women wrestling led him to say and do things that disrupted his marriage and his friendships with other people. Many people with paraphilias experience sexual dysfunction involving desire, arousal, or orgasm during conventional sexual behavior with a partner. The wives of men with paraphilias frequently protest that their husbands are not interested in their sexual relationship. In fact, the husband may be actively engaged in frequent masturbation to paraphilic fantasies. Cases of this sort present an interesting diagnostic challenge to the clinician, who must distinguish a paraphilia from what might otherwise appear to be low sexual desire.

How have changing attitudes toward sexuality influenced the definition of paraphilia?

DIAGNOSIS

Although they are listed as distinct disorders, it might be more useful to think of the paraphilias as one diagnostic category, with the specific forms listed in DSM-IV-TR representing subtypes of this single disorder (Fedoroff, 2003). The primary types of paraphilias described in the following pages are the ones most often seen in clinics that specialize in the treatment

Table 12.2 Other Types of Paraphilias	
Name	**Focus of Sexual Urges and Fantasies**
Telephone scatologia	Obscene phone calls
Necrophilia	Corpses
Partialism	One specific part of the body
Zoophilia	Animals
Coprophilia	Feces
Klismaphilia	Enemas
Urophilia	Urine
Stigmatophilia	Piercing; marking body; tattoos

of sexual disorders. Not surprisingly, they are also the ones that frequently lead to a person being arrested. Other types of paraphilias are listed in Table 12.2.

Fetishism Anthropologists use the word *fetish* to describe an object that is believed to have magical powers to protect or help its owner. In psychopathology, **fetishism** refers to the association of sexual arousal with nonliving objects. The range of objects that can become associated with sexual arousal is virtually unlimited, but fetishism most often involves women's underwear, shoes and boots, or products made out of rubber or leather (Darcangelo, 2008). The person may go to great lengths, including burglary, to obtain certain kinds of fetish objects.

Many men find women's clothing attractive or sexy, but for a man with a fetish, sexual arousal is focused *exclusively* on the object. The partner is largely irrelevant.

People who fit the description of fetishism typically masturbate while holding, rubbing, or smelling the fetish object. Particular sensory qualities of the object—texture, visual appearance, and smell—can be very important in determining whether the person finds it arousing. In addition to holding or rubbing the object, the person may wear, or ask his sexual partner to wear, the object during sexual activity. The person may be unable to become sexually aroused in the absence of the fetish object.

Transvestic Fetishism A *transvestite* is a person who dresses in the clothing of the other gender. In DSM-IV-TR, **transvestic fetishism** is defined as cross-dressing for the purpose of sexual arousal. It has been described primarily among heterosexual men and should not be confused with the behavior of some gay men known as *drag queens* (for whom cross-dressing has a very different purpose and meaning). DSM-IV-TR restricts the definition to heterosexual men, but research evidence suggests that a small proportion of transvestites are bisexual, homosexual, or not sexually active with another person (Bullough & Bullough, 1997).

People who engage in transvestic fetishism usually keep a collection of female clothes that are used to cross-dress. Some wear only a single article of women's clothing, such as female underwear, covered by male clothing. Others dress completely as women, including makeup, jewelry, and accessories. Cross-dressing may be done in public or only in private. The person masturbates while he is cross-dressed, often imagining himself to be a male as well as the female object of his own sexual fantasy. Aside from their interest in cross-dressing, men with transvestic fetishism are unremarkably masculine in their interests, occupations, and other behaviors. Most of these men get married and have children (Schott, 1995).

For some men, transvestism may eventually lead to feelings of dissatisfaction with being male (Zucker & Blanchard, 1997). They may eventually want to live permanently as women. These men, who develop persistent discomfort with their gender role or identity, would be assigned a subtype diagnosis of *transvestic fetishism with gender dysphoria.*

Sexual Masochism People who become sexually aroused when they are subjected to pain or embarrassment are called masochists. DSM-IV-TR defines **sexual masochism** as recurrent, intense sexually arousing fantasies, urges, or impulses involving being humiliated, beaten, bound, or otherwise made to suffer (Hucker, 2008; Krueger, 2010). People may act on these impulses by themselves or with a partner. In some large cities, clubs cater to the sexual interests of masochistic men and women, who pay people to inflict pain on them.

The person may become aroused by being bound, blindfolded, spanked, pinched, whipped, verbally abused, forced to crawl and bark like a dog, or in some other way made to experience pain or feelings of shame and disgrace. One relatively common masochistic fantasy takes the form of being forced to display one's naked body to other people. Masochists desire certain types of pain (which are carefully controlled to remain within specified limits, usually unpleasant but not agonizing), but they also go to great lengths to avoid injury during their contrived, often ritualized experiences (Stoller, 1991). They do not enjoy, and are not immune

Some gay men who dress in women's clothes refer to themselves as "drag queens." This is different from transvestic fetishism, which applies only to heterosexual men whose cross-dressing is associated with intense, sexually arousing fantasies or urges.

to, painful experiences that lie outside these limited areas of their lives.

The following first-person account was written by Daphne Merkin (1996), an accomplished writer whose fascinating and controversial essay on masochism appeared in *The New Yorker*.

BRIEF CASE STUDY

Masochism

> The fact is that I cannot remember a time when I didn't think about being spanked as a sexually gratifying act, didn't fantasize about being reduced to a craven object of desire by a firm male hand. Depending on my mood, these daydreams were marked by an atmosphere of greater or lesser ravishment, but all of them featured similar ingredients. Most important among them was a heightened—and deeply pleasurable—sense of exposure, brought about by the fact that enormous attention was being paid to my bottom, and by the fact that there was an aspect of helpless display attached to this particular body part. This scenario, in which my normally alert self was reduced to a condition of wordless compliance via a specific ritual of chastisement, exerted a grip that was the more strong because I felt it to be so at odds with the intellectually weighty, morally upright part of me. (p. 99)

These fantasies and urges made Merkin feel uncomfortable, and she kept them to herself for many years. Being cautious and somewhat inhibited—certainly not prone to illicit sexual adventures—she worried about the boundaries of her masochistic desires. If she ever acted on them, where would she stop? And how would her partner respond? After many years of privately harboring masochistic sexual fantasies, Merkin finally described her fascination with spanking to a man whom she had been dating for several months. She was in her late twenties at the time, and eventually married this man. The following paragraph describes what happened after her admission:

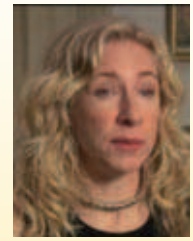
> He appeared delighted at the prospect of implementing my wishes, and so it was that I found myself in the position I had been dreaming of for years: thrust over a man's knee, being soundly spanked for some concocted misdeed. The sheer tactile stimulation of it—the chastening sting—would have been enough to arouse me, but there was also, at last, the heady sense of emotional release: I was and was not a child; was and was not being reduced; was and was not being forced into letting go; was and was not the one in control. I had fantasized about this event for so long that in the back of my mind there had always lurked the fear that its gratification would prove disappointing. I needn't have worried; the reality of spanking, at least initially, was as good as the dream. (pp. 112–113)

Merkin tired of the spankings after she gave birth to her daughter, but the fantasies and urges returned several years later, after she had been separated from her husband. She eventually became involved in a relationship with another man that she described as "a fairly conventional romance that included some light (sadism and masochism)." After their mutual interests and consenting activities had escalated, Merkin found the relationship disturbing:

> It occurred to me that underneath my own limited participation in this world I felt enormous resentment; I was following the steps in a dance I couldn't control. Spanking and its accoutrements may have helped to subdue my simmering rage toward men—as well as theirs toward me—but it also demonstrated how far I was from healthy intimacy, from the real give-and-take that makes a relationship viable. (p. 114)

MyPsychLab VIDEO CASE

Exploring Sexual Sadism and Masochism



JOCELYN

"I really wanted to feel what it was like to be overpowered."

Watch the video, "Exploring Sexual Sadism and Masochism" on MyPsychLab. Do you think that voluntary sexual activities performed with a consenting partner should be considered symptoms of a disorder? In what ways could these behaviors be considered harmful to the person's life (other than the experience of pain)?

This case illustrates the compelling and often contradictory nature of the fantasies that are associated with paraphilias. This successful and independent woman, who did not believe in using corporal punishment with her own daughter, found great pleasure associated with fantasies of being spanked by a man. Merkin would not have qualified for a diagnosis of sexual masochism, even after she had acted on her fantasies, unless she experienced subjective distress or social impairment as a result.

Like Daphne Merkin, many people who engage in masochistic sexual practices are highly educated and occupationally successful. Masochists tend to be disproportionately represented among the privileged groups in society. This pattern leads to the suggestion that masochism may be motivated by an attempt to escape temporarily from the otherwise constant burden of maintaining personal control and pursuing self-esteem (Baumeister & Butler, 1997).

Sexual Sadism Someone who derives pleasure by inflicting physical or mental pain on other people is called a *sadist.* The term is based on the writings of the Marquis de Sade, whose novels describe the use of torture and cruelty for erotic purposes. DSM-IV-TR defines **sexual sadism** in terms of intense, sexually arousing fantasies, urges, or behaviors that involve the psychological or physical suffering of a victim. Sadistic fantasies often involve asserting dominance over the victim; the experience of power and control may be as important as inflicting pain (Hucker, 1997). Some people engage in sadistic sexual rituals with a consenting partner (who may be a sexual masochist) who willingly suffers pain or humiliation. Others act on sadistic sexual urges with nonconsenting partners. In some cases, the severity of the sadistic behaviors escalates over time.

Exhibitionism DSM-IV-TR defines **exhibitionism** in terms of the following criteria: "1. Over a period of at least 6 months, recurrent, intense sexually arousing fantasies, sexual urges, or behaviors involving exposure of one's genitals to an unsuspecting stranger. 2. The person has acted on these sexual urges, or the sexual urges or fantasies cause marked distress or interpersonal difficulty" (APA, 2000, p. 569). This behavior is also known as *indecent exposure.* Many different patterns of behavior fit into this category. About half of these men have erections while exposing themselves, and some masturbate at the time. The others usually masturbate shortly after the experience while fantasizing about the victim's reaction. Their intent usually involves a desire to shock the observer, but sometimes they harbor fantasies that the involuntary observer will become sexually aroused. They rarely attempt to touch or otherwise molest their victims, who are usually women or children (Murphy & Page, 2008).

Exhibitionism is almost exclusively a male disorder. Most exhibitionists begin to expose themselves when they are teenagers or in their early twenties. As adults, most are either married or living with a sexual partner. Exhibitionism is seldom an isolated behavior; men who engage in this type of behavior tend to do it frequently (Abel & Osborn, 1992).

Voyeurism The focus of sexual arousal in **voyeurism** is the act of observing an unsuspecting person, usually a stranger, who is naked, in the process of disrobing, or engaging in sexual activity (Metzl, 2004). Many people, especially men, are sexually aroused by the sight of people who are partially clad or naked. Voyeurs are not aroused by watching people who know that they are being observed. The process of looking ("peeping") is arousing in its own right. The person might fantasize about having a sexual relationship with the people who are being observed, but direct contact is seldom sought. In fact, the secret nature of the observation and the risk of discovery may contribute in an important way to the arousing nature of the situation. The voyeur reaches orgasm by masturbating during observation or later while remembering what he saw. Most keep their distance from the victim and are not dangerous, but there are exceptions to this rule (Långström, 2010).

Frotteurism In **frotteurism**, a person who is fully clothed becomes sexually aroused by touching or rubbing his genitals against other, nonconsenting people. The frotteur usually chooses crowded places, such as sidewalks and public transportation, so that he can easily escape arrest. He either rubs his genitals against the victim's thighs and buttocks or fondles her genitalia or breasts (Horley, 2001; Lussier & Piché, 2008).

Like exhibitionism, frotteurism is a high-frequency form of paraphilia; interviews with people being treated for frotteurism indicate that they may engage in hundreds of individual paraphilic acts. People who engage in frotteurism seek to escape as quickly as possible after touching or rubbing against the other person. They do not want further sexual contact.

Pedophilia People who persistently engage in sexual activities with children exhibit what is undoubtedly the most alarming and objectionable form of paraphilic behavior: pedophilia. Every year, more than 100,000 children in the United States are

To protect women from frotteurs, some railway companies in Japan set aside special women-only cars during peak hours and late at night. This sign in a Tokyo subway station says, "Beware of men who fondle women on crowded trains."

referred to child protective services because of suspected child abuse (see Chapter 18). The effects of child abuse on victims have been the subject of intense debate in recent years. Some victims later engage in excessive and risky sexual activities that lead to additional problems (Browning & Laumann, 1997). One controversial review concluded that negative consequences are neither pervasive nor typically intense (Rind, Tromovitch, & Bauserman, 1998). We must be cautious, however, about accepting the null hypothesis (see Research Methods in Chapter 1). Failure to detect significant differences between victims of abuse and other people may indicate that investigators have not examined the appropriate dependent measures. Harmful consequences of sexual abuse may take many forms, including the disruption of future relationships and discomfort with sexual activity (Emery & Laumann-Billings, 1998). Other forms of mental disorder, such as PTSD and eating disorders, can also be the product of prior sexual abuse (see Chapters 7 and 10).

Pedophilia entails recurrent, intense, sexually arousing fantasies, sexual urges, or behaviors involving sexual activity with a prepubescent child (generally age 13 years or younger). In order to qualify for a diagnosis of pedophilia in DSM-IV-TR, the person must be at least 16 years of age and at least five years older than the child. The terms *pedophile* and *child molester* are sometimes used interchangeably, but this practice confuses legal definitions with psychopathology. A child molester is a person who has committed a sexual offense against a child victim. Therefore, the term depends on legal definitions of "sexual offense" and "child victim," which can vary from one state or country to another. In many locations, a child might be anyone under the age of consent, even if that person has reached puberty. All child molesters are not pedophiles. Furthermore, some pedophiles may not have molested children, because the diagnosis can be made on the basis of recurrent fantasies in the absence of actual behavior (Barbaree & Seto, 1997).

Pedophilia includes a great variety of behaviors and sexual preferences (Cohen & Galynker, 2002; Fagan et al., 2002). Some pedophiles are attracted only to children, whereas others are sometimes attracted to adults. Most pedophiles are heterosexual, and the victims of pedophilia are more often girls than boys. Some offenders are attracted to both girls and boys. Sexual contact with children typically involves caressing and genital fondling. Vaginal, oral, and anal penetration are less common, and physical violence is relatively rare. In many cases, the child willingly and naively complies with the adult's intentions. In most cases, the child knows the person who molests him or her. More than half of all offenses occur in the home of either the child or the offender.

Incestuous relationships, in which the pedophile molests his own children, should perhaps be distinguished from those in which the offender is only casually acquainted with the victim. Incest refers to sexual activity between close blood relatives, such as father–daughter, mother–son, or between siblings. The definition may also be expanded to include stepchildren and their stepparents in reconstituted families. Most reported cases of incest involve fathers and stepfathers sexually abusing daughters and stepdaughters (Cole, 1992).

Many incest perpetrators would not be considered pedophiles, either because their victims are postpubescent adolescents or because they are also young themselves (such as male adolescents molesting their younger sisters). Perhaps as many as half of the men who commit incest have also engaged in sexual activity with children outside their own families (Abel & Osborn, 1992). This subgroup of pedophilic incest perpetrators may be the most harmful and the most difficult to treat. Their personality style is typically passive and dependent. They are unable to empathize with the plight of their victims, perhaps in part because they were absent or uninvolved in early childcare responsibilities (Williams & Finkelhor, 1990).

Rape and Sexual Assault The legal definition of rape includes "acts involving nonconsensual sexual penetration obtained by physical force, by threat of bodily harm, or when the victim is incapable of giving consent by virtue of mental illness, mental retardation, or intoxication" (Goodman, Koss, & Russo, 1993). One conservative estimate of rape prevalence based on a national survey indicated that 14 percent of adult women had been raped (National Victim Center, 1992). The actual rate is probably higher, perhaps in the vicinity of 20 percent (Watts & Zimmerman, 2002). The impact of sexual assault on the victim is described in Chapter 7.

The frequency of coercive sex was studied as part of the NHSLS (Laumann et al., 1994). The 3,500 participants were asked whether they had ever been forced to do something sexually that they did not want to do. The question was focused broadly and did not necessarily focus only on acts involving penetration or threats of violence. Slightly more than one out of every five women in the sample reported that they had been forced by a man to engage in some kind of sexual activity against their will. Among those women who had experienced forced sex, 30 percent said that they had been forced sexually by more than one person.

Some rapes are committed by strangers, but many others—known as *acquaintance rapes*—are committed by men who know their victims. Most female victims know the person who raped them (Wiehe & Richards, 1995). Consider, for example, evidence from women in the NHSLS who had been victims of forced sex. Their relationship to the people who forced them

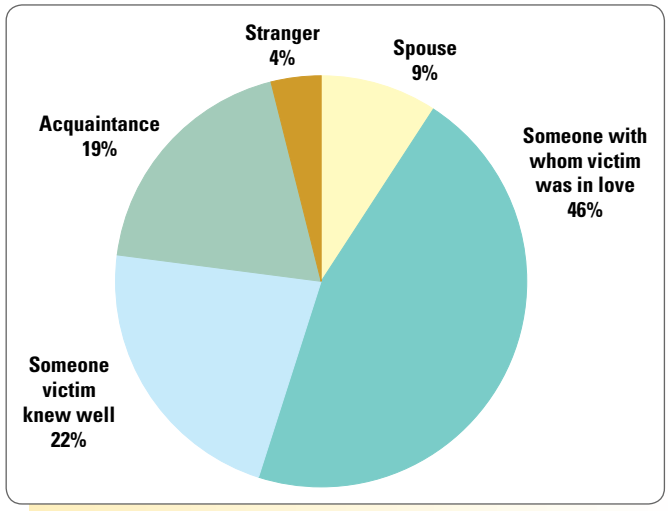


FIGURE 12.4 Forced Sex: Relationship of Perpetrator to Victim

As this chart shows, most NHSLS respondents who were forced into sexual activity knew the person who coerced them.

Source: E. O. Laumann, J. H. Gagnon, R. T. Michael, and S. Michaels, 1994, *The Social Organization of Sexuality: Sexual Practices in the United States,* Chicago: University of Chicago Press, p. 338. Copyright © 1994. Reprinted by permission of The University of Chicago Press.

Does deviant sexual arousal ever play a role in sexual assaults?

to have sex is illustrated in Figure 12.4. Most reported that the person was either someone with whom they were in love or their spouse. Only 4 percent were forced to do something sexual by a stranger.

Rapes are committed by many different kinds of people for many different reasons (Bachar & Koss, 2001). The feminist perspective on rape emphasizes male aggression and violence. The traditional clinical perspective has been concerned with sexual deviance. The authors of DSM-IV-TR considered including rape as a type of paraphilia. This proposal was rejected, primarily because it might imply that rape is *always* motivated by sexual arousal, and it is not. Nevertheless, the behavior of some rapists does include essential features of paraphilias: recurrent, intense sexually arousing fantasies and urges that involve the suffering of nonconsenting persons.

The DSM-V work group for Sexual and Gender Identity Disorders is once again considering these issues, and it has recommended the inclusion of a new diagnostic category that would be called "paraphilic coercive disorder" (Thornton, 2010). The proposal is grounded in the recognition that, for most men, sexual arousal is *inhibited* by obvious clues that their partner is feeling coerced. The new diagnosis would apply to the minority of men for whom the opposite pattern is observed and coercion actually leads to increased sexual arousal. It is not clear, however, that this syndrome can be distinguished reliably from more general sadistic urges and fantasies (Knight, 2010). Further discussions will determine whether this diagnosis is included in the new manual when it is published.

Efforts to classify sexual offenders have attempted to distinguish between those for whom deviant sexual arousal contributes to the act and those whose behavior is motivated primarily by anger or violent impulses. One interesting set of results was produced by studying convicted rapists who were imprisoned at an institution for sexually dangerous persons (Knight & Guay, 2006). Four different types of rapists were identified. Two categories include men whose motivation for sexual assault is primarily sexual in nature. *Sadistic rapists* exhibit features that are close to the DSM-IV-TR definition of a paraphilia. Their behavior is determined by a combination of sexual and aggressive impulses. The *nonsadistic* category also includes men who are preoccupied with sexual fantasies, but these fantasies are not blended with images of violence and aggression. The sexual aggression of these men may result, in part, from serious deficits in the ability to process social cues, such as the intentions of women.

The other two categories describe men whose primary motivation for rape is not sexual. *Vindictive* rapists seem intent on violence directed exclusively toward women. Their aggression is not erotically motivated, as with sadistic rapists. *Opportunistic* rapists are men with an extensive history of impulsive behavior in many kinds of settings and who might be considered psychopaths (see Chapter 9). Their sexual behavior is governed largely by immediate environmental cues. They will use whatever force is necessary to ensure compliance, but they express anger only in response to the victim's resistance. This research program helps to confirm the impression that sex offenders are, in fact, an extremely heterogeneous group (McCabe & Wauchope, 2005).

FREQUENCY

There is very little evidence regarding the frequency of various types of unconventional sexual behavior. This is especially true for victimless or noncoercive forms of paraphilia, such as fetishism, transvestic fetishism, and sexual masochism, because most of these people seldom seek treatment or come to the attention of law enforcement officials. Furthermore, the fact that these forms of behavior are considered deviant or perverse makes it unlikely that people who engage in them will readily divulge their secret urges and fantasies.

With the exception of sexual masochism, paraphilias are almost always male behaviors. Some 95 percent of the people who seek treatment for paraphilic disorders are men. Paraphilias are seldom isolated phenomena. People who exhibit one type of paraphilia often exhibit others (Marshall, 2007). Gosselin and Wilson (1980) surveyed men who belonged to private clubs that cater to fetishists, sadomasochists, and transvestites, and they found that the members of different clubs often shared the same interests. This overlap is illustrated in Figure 12.5. This pattern has been called *crossing* of paraphilic behaviors. There is obviously a considerable amount of crossover among paraphilias.

CAUSES

The high rate of overlap among paraphilias indicates that the etiology of these behaviors might be most appropriately viewed in terms of common factors rather than in terms of distinct pathways that lead exclusively to one form of paraphilia or

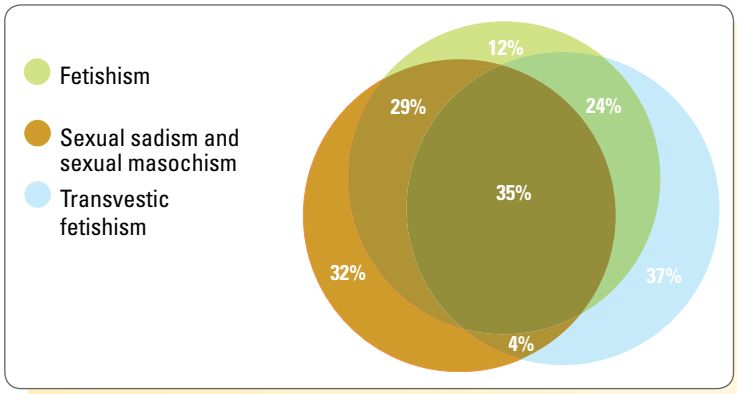


FIGURE 12.5 Overlap Among Three Major Types of Paraphilias

The extent of overlap in the interests of three major types of paraphilias. Note that only 37 percent of men who practice fetishistic transvestism, 32 percent of men who practice sexual sadism and masochism, and 12 percent of those who practice fetishism exhibited those interests exclusively.

Source: G. D. Wilson, 1987, "An Ethological Approach to Sexual Deviation." In G. D. Wilson, ed., *Variant Sexuality: Research and Theory,* p. 92. London: Croom Helm. Copyright © 1987. Reprinted by permission of The Johns Hopkins University Press.

another. Those experiences and conditions that predispose an individual to one form of paraphilia are apparently also likely to lead to another. In the following pages we review a number of proposals regarding the etiology of paraphilias. Some of these have been associated with specific types of paraphilias. For the most part, however, they are concerned more generally with many forms of paraphilias.

Biological Factors Most of the research regarding the role of biological factors in the etiology of paraphilias has focused on the endocrine system (see Figure 2.4 on page 37), the collection of glands that regulate sexual responses through the release of hormones. Some studies of convicted sexually violent offenders have found evidence of elevated levels of testosterone (Langevin, 1992). These reports must be viewed with some skepticism, however, for two reasons. First, the participants in these studies are invariably convicted sexual offenders. Thus, it is not clear that the findings can be generalized to all people with paraphilias. Second, there is a high rate of alcoholism and drug abuse among men convicted of sexual crimes. For that reason, we do not know whether the biological abnormalities observed in these men are causes of their deviant sexual behavior or consequences of prolonged substance abuse.

Neurological abnormalities may also be involved in the development of paraphilias. Structures located in the temporal lobes of the brain, especially the amygdala and the hippocampus, appear to play an important role in the control of both aggression and sexual behavior. These limbic structures, in conjunction with the hypothalamus, form a circuit that regulates biologically significant behaviors that sometimes are whimsically called the four Fs—feeding, fighting, fleeing, and (sexual behavior) (Valenstein, 1973). In 1937, two scientists reported that after extensive bilateral damage to their temporal lobes, rhesus monkeys showed a dramatic increase in sexual activity, as well as a number of related behavioral and perceptual abnormalities. The monkeys apparently tried to copulate with a variety of inappropriate partners, including the investigators. This pattern has subsequently been called the Klüver–Bucy syndrome, named after the scientists who made the original observation.

Inspired by the suggestion that damage to the temporal lobe can lead to unusual patterns of sexual behavior, clinical scientists have studied a number of neurological and neuropsychological factors in convicted sex offenders. Some reports indicate that men with pedophilia and exhibitionism show subtle forms of left temporal lobe dysfunction, as evidenced by abnormal patterns of electrophysiological response and impaired performance on neuropsychological tests (Bradford, 2001; Murphy, 1997).

Social Factors Some types of paraphilias seem to be distortions of the normal mating process when viewed in a broad, evolutionary context. For male primates, sexual behavior involves a sequence of steps: location and appraisal of potential partners; exchange of signals in which partners communicate mutual interest; and tactile interactions that set the stage for sexual intercourse. Voyeurism, exhibitionism, and frotteurism may represent aberrant versions of these social processes. Therefore, the paraphilias have been described as "courtship disorders" (Freund & Blanchard, 1993; Freund & Seto, 1998). Something has apparently gone wrong, disrupting whatever mechanisms facilitate the identification of a sexual partner and govern behaviors used to attract a partner.

Why are paraphilias sometimes called "courtship disorders"?

If people with paraphilias have somehow failed to learn more adaptive forms of courtship behavior, what sort of childhood experiences might have produced such unexpected results? Several background factors have been observed repeatedly among people who engage in atypical sexual behaviors (Seto & Barbaree, 2000; Wincze, 1989). These include the following:

- Early crossing of normative sexual boundaries through a direct experience (for example, sexual abuse by an adult) or an indirect experience (hearing about a father's atypical sexual behavior)
- Lack of a consistent parental environment in which normative sexual behavior and values were modeled
- Lack of self-esteem
- Lack of confidence and ability in social interactions
- Ignorance and poor understanding of human sexuality

All these factors may increase the probability that a person might experiment with unusual types of sexual stimulation or employ maladaptive sexual behaviors.

Although the most notable feature of paraphilias is sexual arousal, ultimately the paraphilias are problems in social relationships. Interpersonal skills may, therefore, play as important a role as sexual arousal. The core feature of unusual sexual behavior may be a failure to achieve intimacy in relationships with other adults (Marshall, 1989; Seidman et al., 1994). According to this perspective, people with paraphilias are lonely, insecure, and isolated and have significant deficits in social skills. Offensive sexual behaviors, such as those observed in pedophilia, are maladaptive attempts to achieve intimacy through sex. These efforts are invariably unsuccessful and self-defeating in the sense that they serve to isolate the person further from the rest of the community. Paradoxically, the pattern may become deeply ingrained because it results in the momentary pleasure associated with orgasm and because it offers the illusory hope of eventually achieving intimacy with another person.

Psychological Factors Another influential perspective on the development of paraphilias has used a geographic metaphor known as a *lovemap* (Money, 2002). A lovemap is a mental picture representing a person's ideal sexual relationship. It might also be viewed as the software that encodes his or her sexual fantasies and preferred sexual practices. These "programs" are written early in life, and they are quite persistent. Children learn their lovemaps during sexual play, by imitation of their parents and other adults, and through messages that they digest from the popular media. According to this theory, when optimal conditions prevail, the child develops a lovemap that includes intercourse as a preferred form of sexual expression. The child learns that love—romantic attachment to another adult—and lust—erotic attraction—can be directed toward the same person.

The lovemap can be distorted, according to this metaphor, if the child learns that romantic attachment and sexual desire are incompatible—that these feelings cannot be directed toward the same person. The inability to integrate these aspects of the lovemap lies at the heart of this explanation for paraphilias. One solution to this dilemma would be to avoid or deny sexual expression altogether. That might explain the development of lack of sexual desire. Sexual impulses are powerful, however, and they are not easily denied. In some cases, they are rerouted rather than being shut off completely. Various types of paraphilias represent alternative strategies through which the person finds it possible to express sexual feelings outside an intimate, loving relationship with another adult. Exhibitionism, voyeurism, and fetishism, therefore, are partial solutions to the perceived incompatibility of love and lust.

TREATMENT

The treatment of paraphilias is different from the treatment of sexual dysfunctions in several ways. Perhaps most important is the fact that most people with paraphilias do not enter treatment voluntarily. They are often referred to a therapist by the criminal justice system after they have been arrested for exposing themselves, peeping through windows, or engaging in sexual behaviors with children. Their motivation to change is, therefore, open to question. Participation in treatment may help them receive reduced sentences or avoid other legal penalties. In many cases, they are being asked to abandon highly reinforcing behaviors in which they have engaged for many years. Their families and other members of society may be much more concerned about change than they are. We mention this issue at the beginning of our discussion because the results of outcome studies in this area are typically less positive than are those concerning the treatment of sexual dysfunctions (McConaghy, 1999; Prentky et al., 1997).

Aversion Therapy For several decades, the most commonly used form of treatment for paraphilias was aversion therapy. In this procedure, the therapist repeatedly presents the stimulus that elicits inappropriate sexual arousal—such as slides of nude children—in association with an aversive stimulus, such as repulsive smells, electric shock, or chemically induced nausea. Revolting cognitive images are sometimes used instead of tangible aversive stimuli. Whatever the exact procedure, the rationale is to create a new association with the inappropriate stimulus so that the stimulus will no longer elicit sexual arousal. Some studies suggested that aversion therapy produces positive effects (Kilmann et al., 1982). This treatment has more recently fallen into disfavor, however, because the studies that were used to evaluate it suffered from design flaws.

Cognitive Behavioral Treatment Current behavioral treatment programs for paraphilic behaviors reflect a broader view of the etiology of these conditions. There is considerable reason to believe that paraphilias are based on a variety of cognitive and social deficits. Marshall, Eccles, and Barbaree (1991) compared two approaches to the treatment of exhibitionists. One was based on aversion therapy and the other employed cognitive restructuring, social skills training, and stress management procedures. The men who received the second type of treatment were much less likely to return to their deviant forms of sexual behavior than were the men who received aversion therapy. Treatment with aversion therapy was no more effective than was treatment with a placebo. These data suggest that broad-based cognitive and social treatment procedures may ultimately be most useful in the treatment of paraphilias and sexual disorders (Marshall et al., 1996).

Unfortunately, research results regarding the effectiveness of psychological treatment for sexual offenders are discouraging. The only large-scale evaluation of such programs that has employed random assignment to treatment conditions is the California Sex Offender Treatment and Evaluation Project (SOTEP; Marques et al., 1993), which was designed for men convicted of either rape or child molestation. Men selected for this comprehensive treatment program are transferred to a special hospital unit, where they remain for several months. They receive education in human sexuality as well as cognitive behavior therapy, including applied relaxation and social skills training and stress and anger management. Treatment also includes a relapse prevention component that is based on procedures used in the treatment of alcoholism (see Chapter 11). Relapse prevention procedures help the men confront personal, social, and sexual difficulties that may increase their risk of relapse after they are released from prison.

The men in the treatment group are compared to those in two control groups. Outcome is measured in several ways, but the most important consideration is being arrested again for similar crimes. Figure 12.6 illustrates some of the results from this study, highlighting the comparison between 138 men who completed the treatment and 184 men who had originally volunteered to participate in the program but were assigned to a no-treatment control group (Marques, 1999). Within four

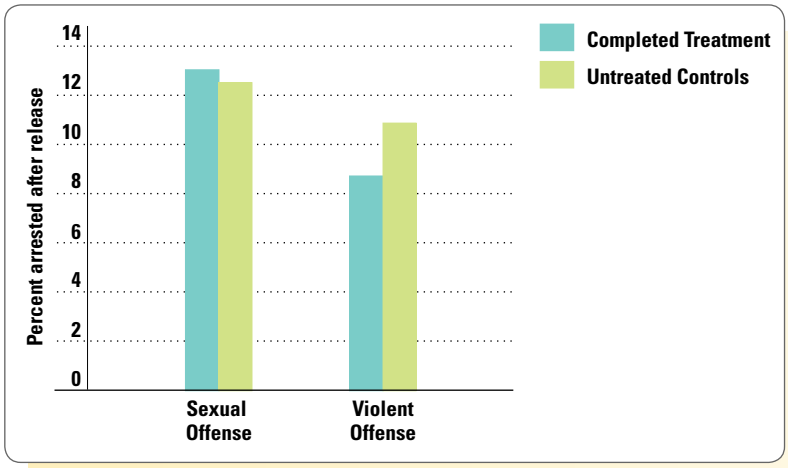


FIGURE 12.6 Outcome of Psychological Treatment for Sex Offenders

Repeat arrest rates among male sex offenders (4 years after treatment).

Source: From "How to Answer the Question: Does Sex Offender Treatment Work?" by J. K. Marques, *Journal of Interpersonal Violence, 14* (1999), pp. 437–451. Copyright © 1999. Reprinted by permission of Sage Publications, Inc.

years after their release from prison, the percentage of men who were arrested for another sexual offense was essentially identical to that of the men who had been treated and of those in the control group (13 percent). The rate of arrest for subsequent violent offenses was somewhat lower for the treatment group than for the controls, but the difference was not significant.

Results were somewhat more encouraging with men convicted of rape than with those who had molested children. Nevertheless, the data from this study are discouraging. They suggest that a broadly based behavioral program focused on education, social skills, and relapse prevention procedures does not lead to obviously better outcomes than a routine period of incarceration (Maletzky, 2002).

Hormones and Medication Another approach to the treatment of paraphilias involves the use of drugs that reduce levels of testosterone, on the assumption that male hormones control the sexual appetite (Hill et al., 2003). One study reported that treatment of paraphilic men with cyproterone acetate, a drug that blocks the effects of testosterone, produced a significant reduction in some aspects of sexual behavior, especially sexual fantasies (Bradford & Pawlak, 1993). Among men with pedophilia, the study found a greater reduction of sexual fantasies of children than of images of sex between consenting adults. Positive results have also been reported for use of triptorelin, which reduces testosterone secretion by inhibiting pituitary–gonadal function. In an uncontrolled trial, 30 male patients (25 with pedophilia) received monthly injections of triptorelin as well as supportive psychotherapy. All of the patients showed a reduction in deviant fantasies and in the number of incidents of paraphilic behaviors (Rosler & Witztum, 1998). We must remember, however, that the absence of double-blind, placebo-controlled studies leaves the efficacy of these drugs in doubt. One review of this literature concludes that treatment programs should never rely exclusively on the use of medications that reduce levels of testosterone (Prentky, 1997).

Antidepressants and antianxiety drugs have also been used to treat paraphilias. Some outcome studies indicate that the SSRIs can have beneficial effects for some male patients (Thibaut et al., 2010). The process by which these drugs manage to alter sexual behavior is open to question. For example, medication may work directly by decreasing deviant sexual interests without affecting other forms of sexual arousal. On the other hand, SSRIs may work by reducing social anxiety, which interferes with the ability to enjoy an intimate sexual relationship with another adult.

Legal Issues The U.S. Congress and all 50 states have passed laws that are intended to protect society from people who have been convicted of violent or repeated sexual offenses. These laws fall into two categories. The first includes *community notification laws* (such as "Megan's Law"), which require the distribution of information to the public regarding the presence of child molesters and sexually violent offenders when they are released from prison or placed on parole. These laws are based on two assumptions: (1) Notification will reduce the offender's opportunities to commit further crimes, and (2) citizens are better able to protect themselves and their children if

U.S. sex offenders can now be tracked on a radar-like app for the iPhone as glowing red dots in the Sex Offender Tracker. Under American law sex offenders must register in their local neighborhoods, and they will now be even more visible as tiny dots on a green screen. The military style radar app will, according to its makers, track sex-offenders in real-time as well as reveal detailed information of their location.

they know that a dangerous person lives in their neighborhood. Critics of community notification laws argue that they violate the former offender's constitutional rights by imposing an additional, unfair penalty after his sentence has been served. These laws are popular, but their impact has not been evaluated. It is not clear that people are actually better able to protect themselves after they have been notified. Furthermore, we do not know whether relapse rates are lower among sexual offenders who live in communities where such laws are strictly enforced (Edwards & Hensley, 2001; Younglove & Vitello, 2003).

The second category includes *sexual predator laws,* which are designed to keep some criminals in custody indefinitely. For example, a Kansas law passed in 1994 and later upheld by the U.S. Supreme Court permits authorities to commit certain sex offenders to a mental hospital after their prison terms are over. Each case is evaluated in a series of steps that end with a civil trial. The person can be hospitalized involuntarily and for an indefinite period of time if the jury decides the person has a "mental abnormality" that will lead him to commit further sexual offenses. Involuntary civil commitment is an infrequent outcome of this law (Fabian, 2011). When it does occur, however, serious questions are raised about the need to balance public safety against the protection of the offender's constitutional rights (see the discussion of civil commitment in Chapter 18).

Gender Identity Disorders

Our sense of ourselves as being either male or female is known as **gender identity.** Gender identity almost always reflects the child's physical anatomy: Toddlers who possess a penis learn that they are boys, and those with a vagina learn that they are girls. Gender identity is usually fixed by the time a child reaches 2 or 3 years of age (Clemans et al., 2010).

What is the difference between gender identity and sex roles?

Gender identity must be distinguished from *sex roles,* which are characteristics, behaviors, and skills that are defined within a specific culture as being either masculine or feminine. For example, certain aspects of appearance and behavior are more often associated with men than with women. These are considered to be masculine. Those behaviors and appearances that are more often associated with women are considered feminine. In our own culture, masculine and feminine sex roles have changed considerably in recent years, and they overlap to a degree (Sczesny et al., 2008).

SYMPTOMS

Some people are firmly convinced that their anatomy and their gender identity do not match up. In males, this means that they feel strongly that they are women trapped in a man's body. For females, the opposite pattern holds. DSM-IV-TR categorizes this sense of discomfort with one's anatomical sex as **gender identity disorder.** It has also been called *transsexualism* or *gender dysphoria.* People with gender identity disturbances do not literally believe that they are members of the other gender. Rather, they feel that, with the exception of their physical anatomy, they are more like the other gender (Becker & Johnson, 2009).

Most transsexuals report that they were aware of these feelings very early in childhood. Many report that they dressed in clothing and adopted sex-role behaviors of the other gender during childhood and adolescence. The intensity of the person's discomfort varies from one individual to the next. Invariably, it becomes more intense during adolescence, when the person develops secondary sexual characteristics, such as breasts and wider hips for girls, and facial hair, voice changes, and increased muscle mass for boys. These characteristics make it more difficult for a person to pass for the other gender. Many transsexuals become preoccupied with the desire to change their anatomical sex through surgical procedures (Paap et al., 2011).

Gender identity disorders should be distinguished from transvestic fetishism, discussed earlier, which is a form of paraphilia in which a heterosexual man dresses in the clothing of the other gender in order to achieve sexual arousal. These are, in fact, very different conditions. Transvestic fetishists do not consider themselves to be women, and transsexuals are not sexually aroused by cross-dressing. They dress as women to feel more comfortable about themselves.

Chaz Bono (born Chastity Bono) is a female-to-male transgender advocate, writer, film-maker, and musician. His documentary film, *Becoming Chaz,* describes his physical and social change from being female to male, which occurred in his late 30s. Bono's famous mother, Cher, was initially uncomfortable with the news of his transition, but she has embraced it and become an outspoken LGBT rights activist.

The relation between gender identity disorder and sexual orientation has been a matter of some controversy. Some clinicians have suggested that transsexuals are homosexuals who claim to be members of the other gender as a way to avoid cultural and moral sanctions that discourage sexual relationships with members of their own sex. This proposal doesn't make sense for two reasons. First, lesbians and gay men are not uncomfortable with their own gender identity. This observation suggests that transsexuals are not simply escaping the stigma of homosexuality. Second, laboratory studies suggest that transsexual and homosexual subjects exhibit different patterns of sexual arousal in response to erotic stimuli.

FREQUENCY

Gender identity disorders are rare in comparison to most of the other disorders that we have considered in this book. Male-to-female transsexuals are apparently more common than

Jazz was a 6-year-old male-to-female transgender child when he and his family appeared on the television news program *20/20*. When Jazz was 2 years old, if his parents praised him as a "good boy," he would correct them, saying he was a good girl.

female-to-male transsexuals, at least based on the numbers of people who seek treatment at clinics. Some studies estimate the prevalence to be approximately one person with gender identity disorder for every 12,000 males and 30,000 females (Olsson & Moller, 2003).

Deeply ingrained cross-gender behaviors and attitudes among children occur infrequently in the general population. Mild forms of cross-gender behavior, such as dressing up in the clothes of the other gender or expressing a desire to be a member of the other sex, are relatively common during the preschool years. Extreme forms of these behaviors are relatively rare, however, especially among boys (Zucker, 2009).

CAUSES

Very little is known about the origins of gender identity in normal men and women, so it is not surprising that the etiology of gender identity disorders is also poorly understood (Richmond, Carroll, & Denboske, 2010). There is some reason to believe that gender identity is strongly influenced by sex hormones, especially during the prenatal period (Diamond, 2009). Much of the research in this area has been done with animals, but an interesting set of data comes from studies of people with a condition that is sometimes called *pseudohermaphroditism*. Individuals with this condition are genetically male, but they are unable to produce a hormone that is responsible for shaping the penis and scrotum in the fetus. Therefore, the child is born with external genitalia that are ambiguous in appearance—thus the term *pseudohermaphrodite*.[2]

Many of these children are raised as girls by their families. When they reach puberty, a sudden increase in testosterone leads to dramatic changes in the appearance of the adolescent's genitals. The organ that had previously looked more like a clitoris becomes enlarged and turns into a penis, and testicles descend into a scrotum. The child's voice becomes deeper, muscle mass increases, and the child quickly begins to consider himself to be a man. The speed and apparent ease with which people with these conditions adopt a masculine gender identity suggest that their brains had been prenatally programmed for this alternative (Hines, 2004).

TREATMENT

There are two obvious solutions to problems of gender identity: Change the person's identity to match his or her anatomy, or change the anatomy to match the person's gender identity. Various forms of psychotherapy have been used in an effort to alter gender identity, but the results have been fairly negative.

MyPsychLab VIDEO CASE

Gender Identity Disorder

DENISE

"I didn't do it for the sexual purpose. It was just to be someone I always thought of myself as—to be the person I wanted to be."

Watch the video "Gender Identity Disorder: Denise" on MyPsychLab. At what age did Denise become dissatisfied with the sexual parts of her body?

One alternative to psychological treatment is *sex-reassignment surgery,* in which the person's genitals are changed to match the gender identity (Sohn & Bosinski, 2007). Medical science can construct artificial male and female genitalia. The artificial penis is not capable of becoming erect in response to sexual stimulation, but structural implants can be used to obtain rigidity. These surgical procedures have been used with thousands of patients over the past 50 or 60 years. Clinics that perform these operations employ stringent selection procedures, and patients are typically required to live for several months as a member of the other gender before they can undergo the surgical procedure.

The results of sex-reassignment surgery have generally been positive (Johansson et al., 2010). Interviews with patients who have undergone surgery indicate that most are satisfied with the results, and the vast majority believe that they do not have trouble passing as a member of their newly assumed gender. Psychological tests obtained from patients who have completed surgery indicate reduced levels of anxiety and depression.

[2]A hermaphrodite has both male and female reproductive organs.

Getting Help

Many sexual problems can be traced to the absence of information regarding the nature of sexual attitudes, feelings, preferences, and behaviors. Fortunately, access to these data, as well as public attitudes toward their discussion, has improved dramatically in recent years. The Sexuality Information and Education Council of the United States (SIECUS) collects and disseminates information and promotes education about sexuality. The council's Internet homepage (www.siecus.org) contains an extensive list of resources, including books and links to other websites, dealing with topics that range from reproduction, women's health, gender identity, and sexual orientation to sexually transmitted diseases and various types of sexual disorders.

If you have been troubled by problems with sexual arousal, inhibited orgasm, or premature orgasm, behavioral procedures can be helpful. Many of these problems can be treated successfully using procedures that were originally developed by Masters and Johnson. Before you seek professional therapy, you may want to try some self-help techniques that have developed from this treatment tradition. Two exceptionally well-written and practical books describe how these procedures can be used by people who want to enhance the pleasure that they experience in their sexual relationships. They are *Becoming Orgasmic: A Sexual and Personal Growth Program for Women,* by Julia Heiman and Joseph LoPiccolo, and *The New Male Sexuality,* by Bernie Zilbergeld.

If you are still experiencing problems after trying self-help procedures, you should seek treatment with a professional sex therapist. The person's professional background is less important than her or his training for treatment of these specific problems. When you contact potential therapists, ask them whether their treatment methods are similar to those developed by Masters and Johnson. Information regarding counseling, therapy, medical attention, and other sexuality resources for people with sexual problems is available from the Sexual Health Network (www.sexualhealth.com). Concise, readable descriptions of various forms of sexual dysfunction and treatments used to address them can be found at the Sexual Health InfoCenter (www.sexhealth.org). These sites can help you increase your knowledge of sexuality, ways in which its expression can be inhibited, and procedures that can be used to improve sexual performance and experience.

Anyone who is interested in additional information regarding gender identity disorder will find help in a book called *True Selves: Understanding Transsexualism—for Families, Friends, Coworkers and Helping Professionals* (Brown & Rounsley, 2003). The authors use extended interviews with patients and families to provide valuable insights regarding important issues encountered by people who struggle with these conditions.

SUMMARY

- DSM-IV-TR recognizes two major forms of sexual disorders. **Sexual dysfunctions** involve an inhibition of sexual desire or disruption of the physiological responses leading to orgasm. **Paraphilias** are defined in terms of extreme forms of unusual sexual behavior, in which sexual arousal is associated with atypical stimuli.
- Sexual dysfunctions are subdivided into several types, based on the stages of the sexual response cycle. These include problems related to sexual desire, sexual arousal, and orgasm. Related difficulties include **sexual aversion disorder** and **premature ejaculation. Dyspareunia** is defined in terms of persistent genital pain during or after sexual intercourse. **Vaginismus** is an involuntary spasm of the muscles surrounding the entrance to the vagina.
- Sexual behavior is dependent on a complex interaction among biological, psychological, and social factors. These factors include cognitive events related to the perception of sexual stimuli, social factors that influence sexual meanings or intentions, and physiological responses that cause vasocongestion of the genitals during sexual arousal.
- Biological factors that contribute to sexual dysfunction include inadequate levels of sex hormones as well as a variety of medical disorders. The effects of alcohol, illicit drugs, and some forms of medication can also contribute to **erectile dysfunction** in men and to **orgasmic disorder** in both men and women.
- Several psychological factors are involved in the etiology of sexual dysfunction. Prominent among these are performance anxiety and guilt. Communication deficits can also contribute to sexual dysfunction. Previous experiences, including sexual abuse, play an important role in some cases of sexual dysfunction.
- Psychological treatments for sexual dysfunction are quite successful. They focus primarily on negative attitudes toward sexuality, failure to engage in effective sexual behaviors, and deficits in communication skills.
- Common characteristics of paraphilias include lack of human intimacy and urges toward sexual behaviors that the person feels compelled to perform. The diversity and range of paraphilic behavior are enormous. DSM-IV-TR describes a few of

the most prominent forms, such as **exhibitionism, fetishism, frotteurism, pedophilia, sexual masochism, sexual sadism, transvestic fetishism,** and **voyeurism**.

- Treatment outcome is generally less successful with paraphilias than with sexual dysfunction. Currently, the most promising approaches to the treatment of paraphilias address a broad range of issues, including deficits in social skills and stress and anger management, as well as knowledge and attitudes regarding sexuality.

- **Gender identity disorder** is a disturbance in the person's sense of being either a man or a woman. People with this problem, which is also known as *transsexualism,* have developed a **gender identity** that is inconsistent with their physical anatomy. These disorders are extremely rare, and very little is known about their etiology. Treatment of gender identity disorders may involve sex-reassignment surgery.

The Big Picture

CRITICAL THINKING REVIEW

- **Should sexual problems be defined primarily in terms of difficulty reaching orgasm?**
  It's important to most people, but it's not the only purpose of sexual activity. The person's satisfaction with intimate relationships is also an important consideration . . . (see pp. 306–308)

- **Should excessive sexual behavior be considered a disorder in its own right? Or is it a symptom of other mental disorders?**
  One problem with the concept of hypersexual disorder is the heterogeneous nature of poorly regulated sexual behavior, which can be associated with many different forms of mental disorder . . . (see pp. 312–313)

- **What role do mental scripts play in sexual arousal?**
  Scripts provide a perceptual structure, helping the person to recognize a situation as being potentially sexual in nature . . . (see p. 315)

- **What are the primary targets of psychological approaches to treating sexual dysfunction?**
  They focus on negative attitudes toward sexuality, failure to engage in effective sexual behaviors, and deficits in communication between partners . . . (see pp. 316–317)

- **How have changing attitudes toward sexuality influenced the definition of paraphilias?**
  A wider range of sexual behaviors is now considered to be normal when these activities occur in the context of an adult, mutually consenting, reciprocal relationship . . . (see p. 319)

- **Does deviant sexual arousal ever play a role in sexual assaults?**
  Although rapes are often motivated by aggressive and violent impulses, some rapists are preoccupied by a sadistic blend of sexual and aggressive impulses that resembles qualities of other paraphilias . . . (see p. 324)

- **Why are paraphilias sometimes called "courtship disorders"?**
  An evolutionary perspective suggests that some forms of paraphilia, such as voyeurism and exhibitionism, may represent maladaptive distortions of normal mating behaviors . . . (see p. 325)

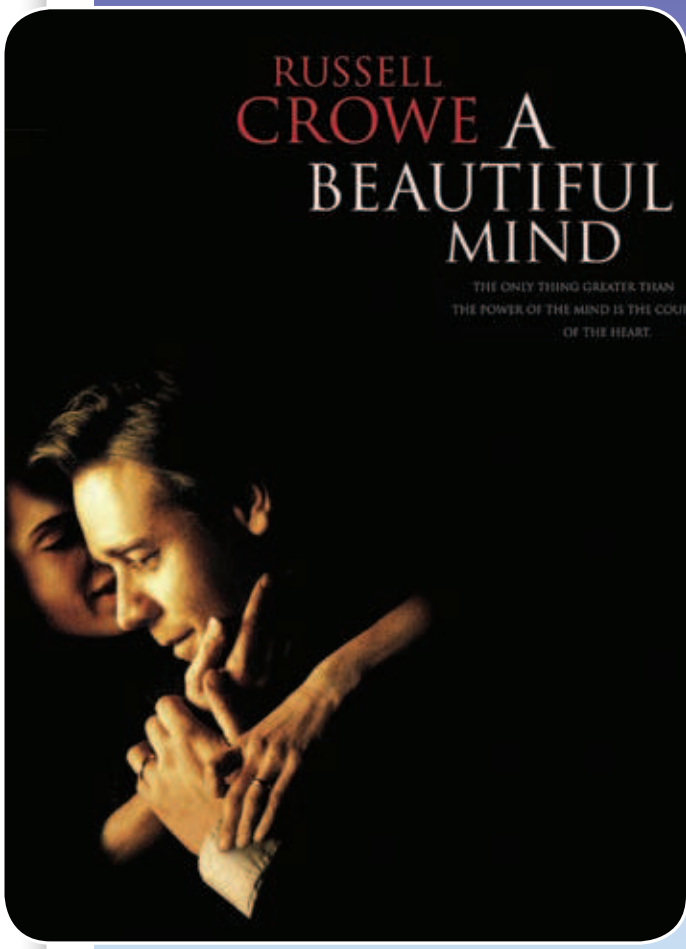
- **What is the difference between gender identity and sex roles?**
  The answer hinges on the distinction between a belief about "who you are" versus "what you do." Gender identity is the fundamental sense of being either male or female. Sex roles are characteristics and behaviors that are considered to be either masculine or feminine . . . (see p. 328)

KEY TERMS

construct validity
dyspareunia
erectile dysfunction
exhibitionism
fetishism
frotteurism
gender identity
gender identity disorder
hypoactive sexual desire
hypothetical construct
inhibited sexual arousal
operational definition
orgasmic disorder
paraphilias
pedophilia
premature ejaculation
sensate focus
sexual aversion disorder
sexual dysfunction
sexual masochism
sexual sadism
transvestic fetishism
vaginismus
voyeurism

13

Schizophrenic Disorders



◀ *A Beautiful Mind* tells the powerful, true story of Nobel Prize–winning mathematician John Nash and his struggle to distinguish fantasy from reality as he copes with schizophrenia.

Schizophrenia is a severe form of abnormal behavior that encompasses what most of us have come to know as "madness." People with schizophrenia exhibit many different kinds of psychotic symptoms, indicating that they have lost touch with reality. They may hear voices that aren't there or make comments that are difficult, if not impossible, to understand. Their behavior may be guided by absurd ideas and beliefs. For example, a person might believe that spaceships from another planet are beaming thoughts into his brain and controlling his behavior. Some people with schizophrenia recover fairly quickly, whereas others deteriorate progressively after the initial onset of symptoms. It is a disorder with "many different faces" (Andreasen, 2001). Because of the diversity

of symptoms and outcomes shown by these patients, many clinicians believe that schizophrenia, or "the group of schizophrenias," may actually include several forms of disorder that have different causes. Others contend that schizophrenia is a single pathological process and that variations from one patient to the next in symptoms and course of the disorder reflect differences in the expression or severity of this process.

The Big Picture

- What does it mean to say that psychotic patients are "out of touch with reality"?
- Does schizophrenia appear in all different parts of the world?
- Can anyone develop schizophrenia, or do you have to be vulnerable in some way?
- If schizophrenia is a disorder of the brain, what part is broken?
- How can we tell if a person is at risk for schizophrenia before the symptoms appear?
- What are the benefits and costs of antipsychotic medication?

OVERVIEW

Many of the disorders that we have discussed in this book strike us as being familiar, at least in form if not in severity. For example, depression and anxiety are experiences with which we can easily empathize. Short-lived versions of these emotions help to shape our responses to daily events. Some clinical scientists speculate that mood and anxiety disorders may be viewed as evolved adaptations or mechanisms that can serve a useful purpose, but the symptoms of schizophrenia represent a different kind of problem. It is much harder for us to understand when someone hears voices that aren't there or speaks sentences that are meaningless. These symptoms seem to stem from a fundamental breakdown in basic cognitive functions that govern the way the person perceives and thinks about the social world (Burns, 2006).

The most common symptoms of schizophrenia include changes in the way a person thinks, feels, and relates to other people and the outside environment. No single symptom or specific set of symptoms is characteristic of all schizophrenic patients. All the individual symptoms of schizophrenia can also be associated with other psychological and medical conditions. Schizophrenia is officially defined by various combinations of psychotic symptoms in the absence of other forms of disturbance, such as mood disorders (especially manic episodes), substance dependence, delirium, or dementia (see Chapter 14).

Schizophrenia is a devastating disorder for both the patients and their families (Bowie et al., 2010). It can disrupt many aspects of the person's life, well beyond the experience of psychotic symptoms. The impact of this disorder is felt in many different ways. For people who develop schizophrenia, it often has a dramatic and lasting impact on their quality of life, both in terms of their own subjective satisfaction and their ability to complete an education, hold a job, and develop social relationships with other people. Approximately 10 percent of schizophrenic patients commit suicide (Heisel, 2008).

For family members of patients with schizophrenia, the consequences can also be cruel. They must come to grips with the fact that their son or daughter, or brother or sister, has developed a severe disorder that may change his or her life forever. One woman whose daughter, then in her mid-thirties, had exhibited symptoms of schizophrenia for 17 years, described her feelings in the following way: "Nothing in (our daughter's) growing up years could have prepared us for the shock and devastation of seeing this normal, happy child become totally incapacitated by schizophrenia" (Smith, 1991, p. 691).

Schizophrenia also has an enormous impact on society (Behan, Kennelly, & O'Callaghan, 2008). Among mental disorders, it is the second leading cause of disease burden (see Figure 1.2 on page 11). Most people who develop the disorder do not recover completely, and many become homeless because long-term institutional care is not available (see Chapter 18). Above and beyond the direct costs of providing treatment to patients and their families, substantial indirect costs are associated with loss of productivity and unemployment. In the United States the financial costs associated with schizophrenia were approximately $63 billion in 2002 (Wu et al., 2005).

In the following case studies we describe the experiences of two people who exhibited symptoms of schizophrenia. DSM-IV-TR divides schizophrenic disorders into several subtypes, based primarily on the type of symptoms that the patient exhibits. Our first case illustrates the paranoid subtype of schizophrenia, which is characterized by a preoccupation with one or more delusions or by frequent auditory hallucinations, most often persecutory.

CASE STUDY A New Mother's Paranoid Schizophrenia

Ann was 21 years old the first time that she was admitted to a psychiatric hospital. She had completed business college and had worked as a receptionist until she became pregnant with her son, who was born six months prior to her admission. She and her husband lived in a small apartment with his 5-year-old daughter from a previous marriage. This was her first psychotic episode.

The first signs of Ann's disturbance appeared during her pregnancy, when she accused her husband of having an affair with her sister. The accusation was based on a conversation that Ann had overheard on a bus. Two women (who were neighbors in Ann's apartment building) had been discussing an affair that some woman's husband was having. Ann believed that this might have been their way of telling her about her husband's infidelity. Although her husband and her sister denied any romantic interest in each other, Ann clung to her suspicions and began to monitor her husband's activities closely. She also avoided talking with her neighbors and friends.

Before this period of time, Ann had been an outgoing and energetic person. Now she seemed listless and apathetic and would often spend days without leaving their apartment. Her husband at first attributed this change in her behavior to the pregnancy, believing that she would "snap out of it" after the baby was born. Unfortunately, Ann became even more socially isolated following the birth of her son. She seldom left her bedroom and would spend hours alone, mumbling softly to herself.

Ann's behavior deteriorated markedly two weeks prior to her hospital admission, when she noticed that some photographs of herself and her baby were missing. She told her husband that they had been stolen and were being used to cast a voodoo spell on her. Ann became increasingly preoccupied with this belief in subsequent days. She called her mother repeatedly, insisting that something would have to be done to recover the missing photographs. Her friends and family tried to reassure Ann that the photographs had probably been misplaced or accidentally discarded, but she was totally unwilling to consider alternative explanations.

Ann finally announced to everyone who would listen that someone was trying to kill her and the children. Believing that all the food in the house had been poisoned, she refused to eat and would not feed the children.

She became increasingly suspicious, hostile, and combative. Her husband and parents found it impossible to reason with her. She was no longer able to care for herself or the children. The family sought advice from their family physician, who recommended that they contact a psychiatrist. After meeting with Ann briefly, the psychiatrist recommended that she be hospitalized for a short period of time.

Believing that all the food in the house had been poisoned, she refused to eat and would not feed the children.

After admission, Ann argued heatedly with the hospital staff, denying that she was mentally disturbed and insisting that she must be released so that she could protect her children from the conspiracy. She had no insight into the nature of her problems.

Painting by a young schizophrenic patient, illustrating his hallucinations. He saw monsters, like the one painted here, crawling on the floor. He also believed that the chairs next to his bed had turned into devils. Patient's description of the picture: "I was very sick at the time I painted this picture. The head represents my fragmented personality and a feeling of being helpless, hopeless, and off balance and of being in a cocoon of unreality. The bright colored rain and outlines represent the level of intensity of myself. The bright colors provided insulation and protected me. The colors felt like microwaves passing through my control center."

The onset of schizophrenia typically occurs during adolescence or early adulthood. The period of risk for the development of a first episode is considered to be between the ages of 15 and 35. The number of new cases drops off slowly after that, with very few people experiencing an initial episode after the age of 55 (Thompson, Pogue-Geile, & Grace, 2004).

The problems of most patients can be divided into three phases of variable and unpredictable duration: prodromal, active, and residual. Symptoms such as hallucinations, delusions, and disorganized speech are characteristic of the active phase of the disorder. The **prodromal phase** precedes the active phase and is marked by an obvious deterioration in role functioning as a student, employee, or homemaker. The person's friends and relatives often view the beginning of the prodromal phase as a change in his or her personality. Prodromal signs and symptoms are similar to those associated with schizotypal personality disorder (see Chapter 9). They include peculiar behaviors (such as talking to one's self in public), unusual perceptual experiences, outbursts of anger, increased tension, and restlessness. Social withdrawal, indecisiveness, and lack of willpower are often seen during the prodromal phase (Woods et al., 2009).

The *residual phase* follows the active phase of the disorder and is defined by signs and symptoms that are similar in many respects to those seen during the prodromal phase. At this point, the most dramatic symptoms of psychosis have improved, but the person continues to be impaired in various ways. Negative symptoms, such as impoverished expression of emotions, may remain pronounced during the residual phase (McGlashan, 1998).

After the onset of schizophrenia, many people do not return to expected levels of social and occupational adjustment. Some prefer social isolation and avoid contact with other people. The man in our second case illustrates this pattern. He is also an example of the disorganized type of schizophrenia. Patients who fit criteria for this category say things that are difficult to understand, behave in a disorganized way, and fail to express expected emotions.

CASE STUDY Edward's Disorganized Schizophrenia

Edward was 39 years old and had lived at home with his parents since dropping out of school after the tenth grade. Edward worked on and off as a helper in his father's roofing business prior to his first psychotic episode at the age of 26. After that time, he was socially isolated and unable to hold any kind of job. He was hospitalized in psychiatric facilities 10 times in the next 14 years. When he was not in the hospital, most of his time at home was spent watching television or sitting alone in his room.

The tenth episode of psychosis became evident when Edward told his mother that he had seen people arguing violently on the sidewalk in front of their house. He believed that this incident was the beginning of World War II. His mother tried to persuade him that he had witnessed an ordinary, though perhaps heated, disagreement between two neighbors, but Edward could not be convinced. He continued to mumble about the fight and became increasingly agitated over the next few days. When he wasn't pacing back and forth from his bedroom to the living room, he could usually be found staring out the front window. Several days after witnessing the argument, he took curtains from several windows in the house and burned them in the street at 2 A.M. A neighbor happened to see what Edward was doing and called the police. When they arrived, they found Edward wandering in a snow-covered vacant lot, talking incoherently to himself. Recognizing that Edward was psychotic, the police took him to the psychiatric hospital.

Although his appearance was somewhat disheveled, Edward was alert and cooperative. He knew the current date and recognized that he was in a psychiatric hospital. Some of his speech was incoherent, and his answers to questions posed by the hospital staff were frequently irrelevant. For example, the following exchange occurred during a structured diagnostic interview. The psychologist asked Edward whether he had any special powers or abilities that other people do not have. He responded by saying that he didn't know because he didn't date women. Puzzled by this tangential response, the psychologist asked him to explain what he meant. Edward responded by asking his own question, "If you had a star in the middle of your head, would you swallow marbles?"

Edward's expressive gestures were severely restricted, and he sat in a relatively motionless position. Although he said that he was frightened by the recent events that he reported to his mother, his face did not betray any signs of emotion. He mumbled slowly in a monotonous tone of voice that was difficult to understand. He said that he could hear God's voice telling him that his father was "the Master of the universe" and he claimed that he had "seen the shadow of the Master."

Other voices seemed to argue with one another about Edward's special calling and whether he was worthy of this divine power.

Other voices seemed to argue with one another about Edward's special calling and whether he was worthy of this divine power. The voices told him to prepare for God's return to earth. At times Edward said that he was a Nazi soldier and that he was born in Germany in 1886. He also spoke incoherently about corpses frozen in Greenland and maintained that he was "only half a person."

Symptoms

In this section we describe in greater detail various types of symptoms that are commonly observed among schizophrenic patients and that are currently emphasized by official diagnostic systems, such as DSM-IV-TR. All of these symptoms can fluctuate in severity over time. Some patients exhibit persistent psychotic symptoms. Others experience symptoms during acute episodes and are better adjusted between episodes.

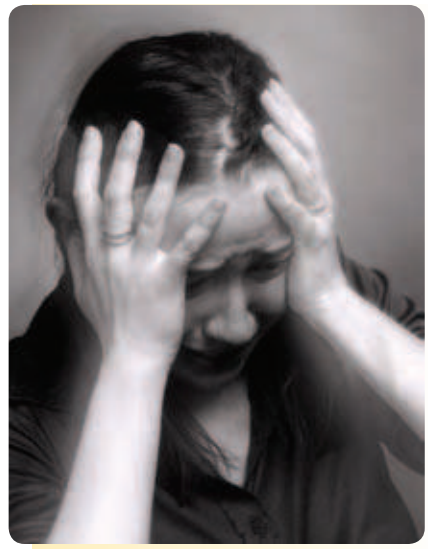
The symptoms of schizophrenia can be divided into three dimensions: positive symptoms, negative symptoms, and disorganization (Lenzenweger, 1999). **Positive symptoms,** also called *psychotic symptoms,* include hallucinations and delusions. In contrast, **negative symptoms** include characteristics such as lack of initiative, social withdrawal, and deficits in emotional responding. Some additional symptoms of schizophrenia, such as incoherent or disorganized speech, do not fit easily into either the positive or negative types. Verbal communication problems and bizarre behavior represent this third dimension, which is sometimes called disorganization. These symptom dimensions overlap and combine in various ways within individual patients. In the following pages, we will describe the most obvious features of these symptoms. It should also be noted, however, that attenuated versions of these symptoms occur relatively frequently in people who are not psychotic (Dominguez et al., 2010). Like other features of psychopathology, these symptoms are not all-or-nothing phenomena; they are best viewed as falling along a continuous dimension of severity.

POSITIVE SYMPTOMS

The term *positive symptoms* of schizophrenia does not imply that these symptoms are beneficial or adaptive. Rather, it suggests that they are characterized by the presence of an aberrant response (such as hearing a voice that is not really there).

Negative symptoms, on the other hand, are characterized by the absence of a particular response (such as emotion, speech, or willpower).

Hallucinations Our senses provide us with basic information that is vital to our notions of who we are, what we are doing, and what others think of us. Many people with schizophrenia experience perplexing and often frightening changes in perception. The most obvious perceptual symptoms are **hallucinations,** or sensory experiences that are not caused by actual external stimuli. Although hallucinations can occur in any of the senses, those experienced by schizophrenic patients are most often auditory. Many patients hear voices that comment on their behavior or give them instructions. Others hear voices that seem to argue with one another. Edward heard the voice of God talking to him. Like Edward, most patients find such voices to be frightening. In some cases, however, hallucinations can be comforting or pleasing to the patient.



Many of the symptoms of schizophrenia, including hallucinations and delusions, can be extremely distressing.

Hallucinations should be distinguished from the transient mistaken perceptions that most people experience from time to time (Brébion et al., 2008). Have you ever turned around after thinking you heard someone call your name, to find that no one was there? You probably dismissed the experience as "just your imagination." Hallucinations, in contrast, strike the person as being real, in spite of the fact that they have no basis in reality. They can vary in terms of both duration and severity. Patients who experience more severe auditory hallucinations hear the voice (or voices) speaking to them throughout the day and for many days at a time.

What is the difference between a delusion and most other false beliefs?

Delusional Beliefs Many schizophrenic patients express **delusions,** or idiosyncratic beliefs that are rigidly held in spite of their preposterous nature (Maher, 2001). Delusions have sometimes been defined as false beliefs based on incorrect inferences about reality. This definition has a number of problems, including the difficulty of establishing the ultimate truth of many situations. Ann's accusation that her husband was having an affair, for example, could easily become a choice between her word and his. This suspicion would not, on its own, be considered a delusion. The judgment that her beliefs were delusional depended to a large extent on their expansion to more absurd concerns about stolen photographs, voodoo spells, and alleged plots to kill her children.

Several additional characteristics are important in identifying delusions (Lincoln et al., 2007). In the most severe cases, delusional patients express and defend their beliefs with utmost conviction, even when presented with contradictory evidence. For example, Ann's belief that the stolen photographs were being used to cast a spell on her was totally fixed and resistant to contradiction or reconsideration. Preoccupation is another defining characteristic of delusional beliefs. During periods of acute psychosis, many patients like Ann find it difficult, if not impossible, to avoid thinking or talking about these beliefs. Finally, delusional patients may be unable to consider the perspective that other people hold with regard to their beliefs. Ann, for example, was unable to appreciate the fact that other people considered her paranoid beliefs to be ridiculous. Taken together, these characteristics describe ways of identifying the severity of delusional beliefs.

Although delusional beliefs can take many forms, they are typically personal. They are not shared by other members of the person's family or cultural group. Common delusions include the belief that thoughts are being inserted into the patient's head, that other people are reading the patient's thoughts, or that the patient is being controlled by mysterious, external forces (Gutierrez-Lobos et al., 2001). Many delusions focus on grandiose or paranoid content. For example, Edward expressed the grandiose belief that his father was the Master of the universe. Ann clung persistently to the paranoid belief that someone was trying to kill her and her children.

In actual clinical practice, delusions are complex and difficult to define (Lesser & O'Donohue, 1999; Oltmanns, 1988). Their content is sometimes bizarre and confusing, as in the case of Edward's insistence that he had witnessed the beginning of World War II. Delusions are often fragmented, especially among severely disturbed patients. In other words, delusions are not always coherent belief systems that are consistently expressed by the patient. At various times, for example, Edward talked about being a Nazi soldier and half a person. Connections among these fragmented ideas are difficult to understand.

The subjective experiences of people who struggle with schizophrenia are an important source of knowledge about this disorder, particularly delusional beliefs. Some of the most fundamental elements of psychosis involve private events that cannot be observed directly by others. Fortunately, many articulate patients have provided compelling accounts of their own internal struggles. The box on the next page is a first person account by a patient who is being treated for schizophrenia. She describes experiences that are part of an elaborate delusional belief system.

MyPsychLab VIDEO CASE

Schizophrenia

LARRY

"My voices had gotten the most of me. . . ."

Watch the video "Schizophrenia: Larry" on MyPsychLab. Pay attention to the nonverbal aspects of Larry's behavior during the interview (lack of expression in face and voice). Notice that, in spite of his symptoms, he does express satisfaction with his life.

FIRST-PERSON ACCOUNT OF DELUSIONAL BELIEFS

At the beginning of my last year at (the university), "feelings" began to descend on me. I felt distinctly different from my usual self. I would sit for hours on end staring at nothing, and I became fascinated with drawing weird, disconnected monsters. I carefully hid my drawings, because I was certain I was being watched. Eventually I became aware of a magical force outside myself that was compelling me in certain directions. The force gained power as time went on, and soon it made me take long walks at 2 or 3 o'clock in the morning down dark alleys in my high-crime neighborhood. I had no power to disobey the force. During my walks I felt as though I was in a different, magical, four-dimensional universe. I understood that the force wanted me to take those walks so that I might be killed.

I do not clearly understand the relationship between the force and the Alien Beings (alas, such a name!), but my universe soon became populated with them. The Alien Beings were from outer space, and of all the people in the world, only I was aware of them. The Alien Beings soon took over my body and removed me from it. They took me to a faraway place of beaches and sunlight and placed an Alien in my body to act like me. At this point I had the distinct impression that I did not really exist, because I could not make contact with my kidnapped self. I also saw that the Aliens were starting to take over other people as well, removing them from their bodies and putting Aliens in their place. Of course, the other people were unaware of what was happening; I was the only person in the world who had the power to know it. At this point I determined that the Aliens were involved in a huge conspiracy against the world.

The Alien Beings were gaining strength and had given me a complex set of rules. The rules were very specific and governed every aspect of my behavior. One of the rules was that I could not tell anyone else about the Aliens or the rules, or else the Aliens would kill me. Another of the rules was that I had to become utterly, completely mad. So now I was living in a world of great fear.

I had a number of other symptoms as well. I felt as though I had been pushed deep within myself, and I had little or no reaction to events or emotions around me. Almost daily the world became unreal to me. Everything outside of me seemed to fade into the distance; everything was miles away from me. I came to feel that I had the power to influence the behavior of animals; that I could, for instance, make dogs bark simply by hooking up rays of thought from my mind to theirs. Conversely, I felt that certain people had the capacity to read my mind. I became very frightened of those people and tried my best to avoid them. Whenever I saw a group of two or three people, I was sure they were talking about me. Paranoia is a very painful emotion! But when I saw crowds of people (as in a shopping mall), I felt an acute longing to wander among them, singing hymns and nursery rhymes (Payne, 1992, pp. 726–727).

NEGATIVE SYMPTOMS

Negative symptoms of schizophrenia are defined in terms of responses or functions that appear to be missing from the person's behavior. In that sense, they may initially be more subtle or difficult to recognize than the positive symptoms of this disorder. Negative symptoms tend to be more stable over time than positive symptoms, which fluctuate in severity as the person moves in and out of active phases of psychosis (Buchanan, 2007; Stahl & Buckley, 2007).

Affective and Emotional Disturbances One of the most common symptoms of schizophrenia involves a flattening or restriction of the person's nonverbal display of emotional responses. This symptom, called **blunted affect,** or *affective flattening,* was clearly present in Edward's case. Blunted patients fail to exhibit signs of emotion or feeling. They are neither happy nor sad, and they appear to be completely indifferent to their surroundings. The faces of blunted patients are apathetic and expressionless. Their voices lack the typical fluctuations in volume and pitch that other people use to signal changes in their mood. Events in their environment hold little consequence for them. They may demonstrate a complete lack of concern for themselves and for others (Blanchard, Cohen, & Carreño, 2007).

Another type of emotional deficit is called **anhedonia,** which refers to the inability to experience pleasure. Whereas blunted affect refers to the lack of outward expression, anhedoniais a lack of positive subjective feelings. People who experience anhedonia typically lose interest in recreational activities and social relationships, which they do not find enjoyable. They may also be unable to experience pleasure from physical sensations, such as taste and touch.

What is the rationale for the distinction between the broad dimensions of positive and negative symptoms?

Longitudinal studies indicate that anhedonia associated with both social and physical experiences is an enduring feature of the disorder for many people with schizophrenia (Herbener & Harrow, 2002). For some people, it may also be an early marker, signaling the onset of the prodromal phase of the disorder (Kwapil, 1998). Like other symptoms of schizophrenia, anhedonia is not unique to this disorder; it is also found among people who are severely depressed.

Apathy, Avolition, and Alogia One of the most important and seriously debilitating aspects of schizophrenia is a malfunction of interpersonal relationships (Meehl, 1993). Many people with schizophrenia become socially withdrawn. In many cases, social isolation develops before the onset of symptoms, such as hallucinations and delusions. It can be one of the earliest signs that something is wrong. This was certainly true in Ann's case. She became socially isolated from her family and friends many weeks before she started to talk openly about the stolen pictures and the plot to kill her children. Social withdrawal appears to be both a symptom of the disorder and a strategy that is actively employed by some patients to deal with

their other symptoms. They may, for example, attempt to minimize interactions with other people in order to reduce levels of stimulation that can exacerbate perceptual and cognitive disorganization (Walker, Davis, & Baum, 1993).

The withdrawal seen among many schizophrenic patients is accompanied by indecisiveness, ambivalence, and a loss of willpower. This symptom is known as *avolition* (lack of volition or will). A person who suffers from avolition becomes apathetic and ceases to work toward personal goals or to function independently. He or she might sit listlessly in a chair all day, not washing or combing his or her hair for weeks.

Another negative symptom involves a form of speech disturbance called *alogia,* which refers to impoverished thinking. Literally translated, it means "speechlessness." In one form of alogia, known as *poverty of speech,* patients show remarkable reductions in the amount of speech. They simply don't have anything to say. In another form, referred to as thought blocking, the patient's train of speech is interrupted before a thought or idea has been completed.

DISORGANIZATION

Some symptoms of schizophrenia do not fit easily into either the positive or negative type. Thinking disturbances and bizarre behavior represent a third symptom dimension, which is sometimes called disorganization (Rietkerk et al., 2008).

Thinking Disturbances One important set of schizophrenic symptoms, known as **disorganized speech,** involves the tendency of some patients to say things that don't make sense. Signs of disorganized speech include making irrelevant responses to questions, expressing disconnected ideas, and using words in peculiar ways (Berenbaum & Barch, 1995). This symptom is also called *thought disorder,* because clinicians have assumed that the failure to communicate successfully reflects a disturbance in the thought patterns that govern verbal discourse. The woman described in the following case exhibited signs of disorganized speech.

CASE STUDY Marsha's Disorganized Speech and Bizarre Behavior

Marsha was a 32-year-old graduate student in political science. She had never been treated for psychological problems.

Marsha called Dr. Higgins, a clinical psychologist who taught at the university, to ask if she could speak with him about her twin sister's experience with schizophrenia. When she arrived at his office, she was neatly dressed and had a Bible tucked tightly under her arm. The next three hours were filled with a rambling discussion of Marsha's experiences during the past 10 years. She talked about her education, her experience as a high school teacher before returning to graduate school, her relationships with her parents, and most of all her concern for her identical twin sister, Alice, who had spent six of the last 10 years in psychiatric hospitals.

Marsha's emotional expression vacillated dramatically throughout the course of this conversation, which was punctuated by silly giggles and heavy sighs. Her voice would be loud and emphatic one moment as she talked about her stimulating ideas and special talents. At other moments, she would whisper in a barely audible voice or sob quietly as she described the desperation, fear, and frustration that she had experienced watching the progression of her sister's disorder. She said that she had been feeling very uptight in recent months, afraid that she might be "going crazy" like her sister. She had been scared to death to go home because her parents might sense that something was wrong with her. Her behavior was frequently inconsistent with the content of her speech. As she described her intense fears, for example, Marsha occasionally giggled uncontrollably.

Dr. Higgins also found Marsha's train of thought difficult to follow. Her speech rambled illogically from one topic to the next, and her answers to his questions were frequently tangential. For example, when Dr. Higgins asked what she meant by her repeated use of the phrase "the ideal can become real," Marsha replied, "Well, after serving the Word of Christ in California for three years, making a public spectacle of myself, someone apparently called my parents and said I had a problem. I said I can't take this anymore and went home. I perceived that Mom was just unbelievably nice to me. I began to think that my face was changing. Something about my forehead resembled the pain of Christ. I served Christ, but my power was not lasting."

Marsha called Dr. Higgins, a clinical psychologist who taught at the university, to ask if she could speak with him about her twin sister's experience with schizophrenia.

At the end of this three-hour interview, Dr. Higgins was convinced that Marsha should be referred to the mental health center for outpatient treatment. He explained his concerns to Marsha, but she refused to follow his advice, insisting that she did not want to receive the medication with which her sister had been treated. She agreed to return to Dr. Higgins's office in three days for another interview, but she did not keep that appointment.

Two weeks later, Marsha called Dr. Higgins to ask if he would talk with her immediately. It was very difficult to understand what she was saying, but she seemed to be repeating in a shrill voice "I'm losing my mind." The door to his office was closed when she arrived, but he could hear her shuffling awkwardly down the hallway, breathing heavily. He opened his door and found Marsha standing in a rigid posture, arms stiffly at her sides. Her eyes were opened wide, and she was staring vacantly at the nameplate on his door. In contrast to her prim and neat appearance at their first meeting, Marsha's hair and clothes were now in disarray. She walked stiffly into the office without bending her knees and sat, with some difficulty, in the chair next to Dr. Higgins's desk. Her facial expression was rigidly fixed. Although her eyes were open and she appeared to hear his voice, Marsha did not respond to any of Dr. Higgins's questions. Recognizing that Marsha was experiencing an acute psychotic episode, Dr. Higgins and one of the secretaries took her to the emergency room at the local hospital.

Marsha's speech provides one typical example of disorganized speech. She was not entirely incoherent, but parts of her speech were difficult to follow. Connections between sentences were sometimes arbitrary, and her answers to the interviewer's questions were occasionally irrelevant.

Several types of verbal communication disruption contribute to clinical judgments about disorganized speech (Docherty, DeRosa, & Andreasen, 1996; Kerns & Berenbaum, 2002). Common features of disorganized speech in schizophrenia include shifting topics too abruptly, called *loose associations* or *derailment;* replying to a question with an irrelevant response, called *tangentiality;* or persistently repeating the same word or phrase over and over again, called *perseveration.* We all say things from time to time that fit these descriptions. It is not the occasional presence of a single feature but, rather, the accumulation of a large number of such features that defines the presence of disorganized speech.

Bizarre Behavior Schizophrenic patients may exhibit various forms of unusual motor behavior, such as the rigidity displayed by Marsha when she appeared for her second interview with Dr. Higgins. *Catatonia* most often refers to immobility and marked muscular rigidity, but it can also refer to excitement and overactivity. For example, some patients engage in apparently purposeless pacing or repetitious movements, such as rubbing their hands together in a special pattern for hours at a time. Many catatonic patients exhibit reduced or awkward spontaneous movements. In more extreme forms, patients may assume unusual postures or remain in rigid standing or sitting positions for long periods of time. For example, some patients will lie flat on their backs in a stiff position with their heads raised slightly off the floor as though they were resting on a pillow. Catatonic patients typically resist attempts to alter their position, even though maintaining their awkward postures would normally be extremely uncomfortable or painful.

Catatonic posturing is often associated with a *stuporous state,* or generally reduced responsiveness. The person seems to be unaware of his or her surroundings. For example, during her acute psychotic episode, Marsha refused to answer questions or to make eye contact with others. Unlike people with other stuporous conditions, however, catatonic patients seem to maintain a clear state of consciousness, and it is likely that Marsha could hear and understand everything that Dr. Higgins said to her. Many patients report after the end of a catatonic episode that they were perfectly aware of events that were taking place around them, in spite of their failure to respond appropriately.

These dementia praecox patients, treated by Emil Kraeplin in the late nineteenth century, display "waxy flexibility," a feature of catatonic motor behavior. "They were put without difficulty in the peculiar positions and kept them, some with a sly laugh, others with rigid seriousness."

Another kind of bizarre behavior involves affective responses that are obviously inconsistent with the person's situation. This symptom is particularly difficult to describe in words. The most remarkable features of *inappropriate affect* are incongruity and lack of adaptability in emotional expression. For example, when Marsha described the private terror that she felt in the presence of her family, she giggled in a silly fashion. The content of Marsha's speech was inconsistent with her facial expression, her gestures, and her voice quality.

Diagnosis

The broad array of symptoms outlined in the previous section have all been described as being part of schizophrenic disorders. The specific organization of symptoms has been a matter of some controversy for many years. Schizophrenic disorders have been defined in many different ways. In the following pages we briefly review some of the more prominent trends that led to the DSM-IV-TR description of these disorders.

BRIEF HISTORICAL PERSPECTIVE

Descriptions of schizophrenic symptoms can be traced far back in history, but they were not considered to be symptoms of a single disorder until late in the nineteenth century (Gottesman, 1991). At that time, Emil Kraepelin, a German psychiatrist, suggested that several types of problems that previously had been classified as distinct forms of disorder should be grouped together under a single diagnostic category called *dementia praecox.* This term referred to psychoses that ended in severe intellectual deterioration (dementia) and that had an early or premature (praecox) onset, usually during adolescence. Kraepelin argued that these patients could be distinguished from those suffering from other disorders (most notably manic–depressive psychosis) largely on the basis of changes that occurred as the disorder progressed over time, primarily those changes involving the integrity of mental functions.

In 1911, Eugen Bleuler (1857–1939), a Swiss psychiatrist and a contemporary of Kraepelin, published an influential monograph in which he agreed with most of Kraepelin's suggestions about this disorder. He did not believe, however, that the disorder always ended in profound deterioration or that it always began in late adolescence. Kraepelin's term *dementia praecox* was, therefore, unacceptable to him. Bleuler suggested a new name for the disorder—*schizophrenia.* This term referred to the *splitting of mental associations,* which Bleuler believed to be the fundamental disturbance in schizophrenia. One unfortunate consequence of this choice of terms has been the confusion among laypeople of schizophrenia with dissociative identity disorder (also known as multiple personality), a severe form of dissociative disorder (see Chapter 7). The two disorders actually have very little in common.

Many other suggestions have been made in subsequent years regarding the description and diagnosis of schizophrenia (Gottesman, 1991; Neale & Oltmanns, 1980). Some clinicians have favored a broader definition, whereas others have argued for a more narrow approach.

TABLE 13.1 DSM-IV-TR Diagnostic Criteria for Schizophrenia

A. Characteristic Symptoms: Two (or more) of the following, each present for a significant portion of time during a one-month period (or less if successfully treated):

1. Delusions
2. Hallucinations
3. Disorganized speech (such as frequent derailment or incoherence)
4. Grossly disorganized or catatonic behavior
5. Negative symptoms, such as affective flattening, alogia, or avolition

(Note: Only one A symptom is required if delusions are bizarre or hallucinations consist of a voice keeping up a running commentary on the person's behavior or thoughts, or two or more voices conversing with each other.)

B. Social/Occupational Dysfunction: For a significant portion of the time since the onset of the disturbance, one or more major areas of functioning such as work, interpersonal relations, or self-care is markedly below the level achieved prior to the onset.

C. Duration: Continuous signs of the disturbance persist for at least six months. This six-month period must include at least one month of symptoms that meet Criterion A (active phase symptoms), and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of the disturbance may be manifested by only negative symptoms or two or more symptoms listed in Criterion A present in an attenuated form (such as odd beliefs, unusual perceptual experiences).

DSM-IV-TR

Several specific criteria for schizophrenia are listed in DSM-IV-TR (see Table 13.1). The first requirement (Criterion A) is that the patient must exhibit two (or more) active symptoms for at least one month. Only one of the characteristic symptoms is required if that symptom is a bizarre delusion or hallucination. Negative symptoms, such as blunted affect, avolition, and social withdrawal, also play a relatively prominent role in the DSM-IV-TR definition of schizophrenia.

The DSM-IV-TR definition takes into account social and occupational functioning as well as the duration of the disorder (Criteria B and C). These criteria reflect the influence of Kraepelin, who argued that the disorder is accompanied by marked impairment in functioning as well as a chronic, deteriorating course. The DSM-IV-TR definition requires evidence of a decline in the person's social or occupational functioning as well as the presence of disturbed behavior over a continuous period of at least six months. Active phase symptoms do not need to be present for this entire period. The total duration of disturbance is determined by adding together continuous time during which the person has exhibited prodromal, active, and residual symptoms of schizophrenia. If the person displays psychotic symptoms for at least one month but less than six months, the diagnosis would be *schizophreniform disorder*. The diagnosis would be changed to schizophrenic disorder if the person's problems persisted beyond the six-month limit.

The final consideration in arriving at a diagnosis of schizophrenia involves the exclusion of related conditions, especially mood disorders. According to DSM-IV-TR, active phase symptoms of schizophrenia must appear in the absence of a major depressive or manic episode. If symptoms of depression or mania are present, their duration must be brief relative to the duration of the active and residual symptoms of schizophrenia.

SUBTYPES

Schizophrenia is a heterogeneous disorder with many different clinical manifestations and levels of severity. The title of Bleuler's classic text referred to "the group of schizophrenias" in an effort to draw attention to the varied presentations of the disorder. It is not clear, however, how best to think about the different forms of schizophrenia. Many clinicians and investigators believe that schizophrenia is a general term for a group of disorders, each of which may be caused by a completely different set of factors. Other clinicians believe that the numerous symptoms of schizophrenia are most likely varying manifestations of the same underlying condition (Gottesman, 1991). Given the current state of evidence, it is not possible to choose between these conceptual options. Nevertheless, most investigators agree that we should at least consider the possibility that there are distinct forms.

DSM-IV-TR recognizes five subtypes of schizophrenia. The subtypes are used to describe the clinical state of the patient during the most recent examination. Only one subtype can be assigned at any point in time. The five subtypes are arranged in a hierarchy so that patients who exhibit symptoms of different subtypes can be diagnosed. The catatonic type is at the top of the hierarchy. Patients who fit this description are diagnosed as catatonic even if they show additional symptoms that are characteristic of other subtypes. The remaining subtypes, in descending order, are the disorganized subtype, the paranoid subtype, the undifferentiated subtype, and the residual subtype (see Critical Thinking Matters).

The *catatonic type* is characterized by symptoms of motor immobility (including rigidity and posturing) or excessive and purposeless motor activity. In some cases, the person may be resistant to all instructions or refuse to speak, for no apparent reason. Catatonic patients may also show a decreased awareness of their environment and a lack of movement and activity.

Critical Thinking Matters

ARE SYMPTOM-BASED SUBTYPES OF SCHIZOPHRENIA USEFUL?

The validity of the traditional subtypes has been debated for many years. The evidence on which they are based is quite weak. Clinicians who favor continued use of subtype diagnoses claim that these categories are *moderately* stable over time (Fenton, 2000). There is also some evidence indicating that patients who fit descriptions of the catatonic and paranoid subtypes have a somewhat better prognosis, whereas those in the disorganized subtype may have—on average—a worse prognosis (McGlashan & Fenton, 1991). If we think critically, this is *not* strong support for the inclusion of these subtypes in the official diagnostic manual.

Critics point to a number of serious problems. Traditional subtypes do not strongly predict either the course of the disorder or response to treatment. The subtypes also have relatively poor diagnostic reliability and are frequently unstable over time. Patients who fit a traditional subcategory during one psychotic episode frequently qualify for a different subtype diagnosis during a subsequent episode. Based on this evidence, it seems reasonable to ask: "How does it help the clinician or the patient to assign a subtype diagnosis such as disorganized type or undifferentiated type?"

Perhaps the most important consideration with regard to the validity of subtypes involves the genetic evidence. Studies of extended families suggest that the subtypes are not etiologically distinct syndromes (Cardno et al., 1998; Linscott et al., 2010). If several members of a family—or two members of a monozygotic twin pair—have developed symptoms of schizophrenia, they will not necessarily exhibit symptoms of the same subtype. That fact argues strongly against the notion that the subtypes are qualitatively different disorders.

Why do clinical scientists say that schizoprenia is a "heterogeneous" disorder?

This is perhaps the greatest irony in research on schizophrenia. For more than 100 years, clinicians and investigators have agreed that the disorder is extremely heterogenous. The diagnostic category that we now recognize as schizophrenia may well be composed of many different kinds of mental disorders. This common opinion stands in contrast to the harsh fact that no one has been able to identify truly meaningful subtypes. Don't take the official system too literally. Be skeptical. At best, the diagnostic subtypes for schizophrenia are placeholders, serving primarily to remind us that the disorder is heterogeneous in nature. We desperately need more knowledge in this area. We need better research that will help us find more meaningful subtypes based on sophisticated measurement procedures that may involve genetic factors, cognitive performance, treatment response, or some other facet of the disorder that has not yet been studied. One thing that does seem to be clear is that it has not been particularly useful to focus on symptoms as the basis for reducing the heterogeneity of the complex disorder.

The *disorganized type* of schizophrenia is characterized by disorganized speech, disorganized behavior, and flat or inappropriate affect. All three features must be present to make this diagnosis. Social impairment is usually quite marked in these patients. The patient's speech is frequently incoherent, and if delusions or hallucinations are present, their content is usually not well organized.

The most prominent symptoms in the *paranoid type* are systematic delusions with persecutory or grandiose content. Preoccupation with frequent auditory hallucinations can also be associated with the paranoid type.

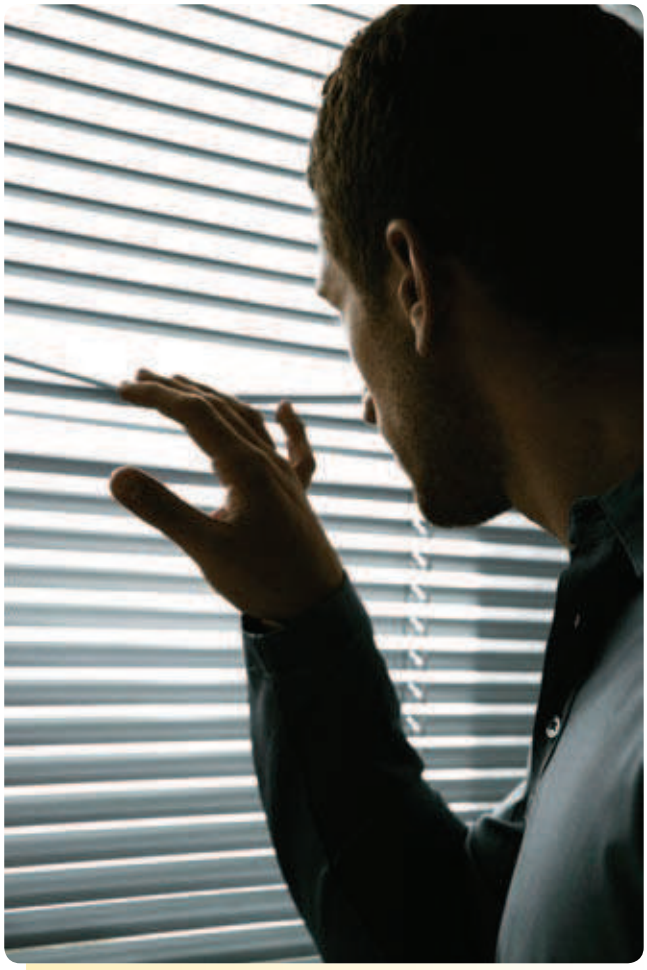
Two additional subtypes are described in DSM-IV-TR, presumably to cover those patients who do not fit one of the traditional types. The *undifferentiated type* of schizophrenia includes schizophrenic patients who display prominent psychotic symptoms and either meet the criteria for several subtypes or otherwise do not meet the criteria for the catatonic, disorganized, or paranoid types. They often exhibit some disorganized symptoms together with hallucinations and/or delusions.

The *residual type* includes patients who no longer meet the criteria for active phase symptoms but nevertheless demonstrate continued signs of negative symptoms or attenuated forms of delusions, hallucinations, or disorganized speech. They are in "partial remission."

RELATED PSYCHOTIC DISORDERS

The U.S. concept of schizophrenia is relatively narrow. The boundaries of the disorder have been refined by excluding patients with certain types of psychotic symptoms from a diagnosis of schizophrenic disorder. Immediately after its description of schizophrenia, DSM- IV-TR lists three additional disorders that are characterized by prominent psychotic symptoms.

Schizoaffective disorder is an ambiguous and somewhat controversial category (Averill et al., 2004; Lake & Hurwitz, 2007). It describes the symptoms of patients who fall on the boundary between schizophrenia and mood disorder with psychotic features. This diagnosis applies only to the description of a particular episode of disturbance; it does not describe the overall lifetime course of the person's disorder. Schizoaffective disorder is defined by an episode in which the symptoms of



People with paranoid delusions are constantly alert to evidence suggesting that they are being victimized. This constant search often leads them to misinterpret others' comments and behaviors.

schizophrenia partially overlap with a major depressive episode or a manic episode. The key to making this diagnosis is the presence of delusions or hallucinations for at least two weeks in the absence of prominent mood symptoms. If the delusions and hallucinations are present only during a depressive episode, for example, the diagnosis would be major depressive episode with psychotic features.

People with **delusional disorder** do not meet the full symptomatic criteria for schizophrenia, but they are preoccupied for at least one month with delusions that are not bizarre. These are beliefs about situations that could occur in real life, such as being followed or poisoned. Ann's delusion, for example, might have fit this description. She believed that someone was trying to kill her and her children and that someone was trying to cast a voodoo spell on them. Ann would not be assigned a diagnosis of delusional disorder, however, because she also displayed negative symptoms, such as avolition. The presence of hallucinations, disorganized speech, catatonic behavior, or negative symptoms rules out a diagnosis of delusional disorder. The definition of delusional disorder also holds that the person's behavior is not bizarre and that social and occupational functioning are not impaired except for those areas that are directly affected by the delusional belief.

How should long-term outcome be measured in schizophrenia?

Brief psychotic disorder is a category that includes those people who exhibit psychotic symptoms—delusions, hallucinations, disorganized speech, or grossly disorganized or catatonic behavior—for at least one day but no more than one month. An episode of this sort is typically accompanied by confusion and emotional turmoil, often (but not necessarily) following a markedly stressful event. After the symptoms are resolved, the person returns to the same level of functioning that had been achieved prior to the psychotic episode. The long-term outcome is good for most patients who experience a brief episode of psychosis (Susser et al., 1998). This diagnosis is not assigned if the symptoms are better explained by a mood disorder, schizophrenia, or substance abuse.

COURSE AND OUTCOME

Schizophrenia is a severe, progressive disorder that most often begins in adolescence and typically has a poor outcome. In fact, Kraepelin considered the deteriorating course to be one of the principal defining features of the disorder. Current evidence suggests that this view may be unnecessarily pessimistic (Hafner et al., 2003; Perkins, Miller-Anderson, & Lieberman, 2006). Many patients experience a more favorable outcome in the sense that their symptoms are improved. For example, Manfred Bleuler (1978) studied a sample of 208 schizophrenic patients who had been admitted to his hospital in Switzerland during 1942 and 1943. After a follow-up period of 23 years, 53 percent of the patients were either recovered or significantly improved. More recent evidence indicates that, while some patients do have a positive outcome, relatively few are able to achieve successful aging (Ibrahim, Cohen, & Ramirez, 2010).

Follow-up studies of schizophrenic patients have found that the description of outcome is a complicated process (Harvey et al., 2009). Many factors must be taken into consideration other than whether the person is still in the hospital. Is the person still exhibiting symptoms of the disorder? Does he or she have any other problems, such as depression or anxiety? Is the person employed? Does she have any friends? How does he get along with other people? The evidence indicates that different dimensions of outcome, such as social adjustment, occupational functioning, and symptom severity, are only loosely correlated. As in most situations where psychologists attempt to predict future behavior, the outcome data regarding schizophrenia suggest that the best predictor of future social adjustment is previous social adjustment. Similarly, the best

MyPsychLab VIDEO CASE

Schizoaffective Disorder

JOSH

"When I was first in the hospital, I thought I was in the middle of a massacre . . ."

Watch the video "Schizoaffective disorders; Josh" on MyPsychLab. In addition to describing bizarre delusional beliefs, Josh has also experienced several symptoms of bipolar mood disorder, such as racing thoughts and grandiosity.

FIRST-PERSON ACCOUNT: A MOTHER'S OBSERVATIONS

The saddest thing of all is to realize that the stories of family life and previous achievements that were a part of the past lives of each of these people are no longer important to them. Nothing in (our daughter's) growing up years could have prepared us for the shock and devastation of seeing this normal, happy child become totally incapacitated by schizophrenia. Coming to grips with the thought of your child living in a mental hospital, possibly for many years, leaves you with a gnawing sense of helplessness that never really dissipates.

In the past year, a new Cindy has emerged. Where once there was a rather unfriendly, often unpleasant girl, there is now an amiable, more responsive person. Cindy smiles more these days, something a person with schizophrenia doesn't do very often. For years her face was a solemn mask, and she could neither give nor receive affection. She knew something terrible had happened to her and could not understand why no one would rescue her from the hell in her head. In the past few months she has become quite loving, and the smiles that now light her face light mine as well (Smith, 1991, pp. 690–691).

predictor of symptom severity at follow-up is severity of psychotic symptoms at initial assessment (Bromet et al., 2005).

The long-term emotional impact of this disorder on parents and families has been described by a woman whose daughter, then in her mid-thirties, had exhibited symptoms of schizophrenia for 17 years. Her statement also indicates that even the most severely disturbed patients may eventually show signs of improvement (see First-Person Account: A Mother's Observations).

Frequency

One of the most informative ways of examining the frequency of schizophrenia is to consider the lifetime prevalence—that is, the proportion of a specific population that will be affected by the disorder at some time during their lives. Most studies in Europe and the United States have reported lifetime prevalence figures of approximately 1.0 percent if they include people who meet diagnostic criteria for schizophrenia as well as related psychotic disorders (Kessler et al., 2005; Saha, Chant, & McGrath, 2008). In other words, approximately one out of every 100 people will experience or display symptoms of schizophrenia at some time during their lives. Of course, prevalence rates depend on the diagnostic criteria that are used to define schizophrenia in any particular study, as well as the methods that are used to identify cases in the general population. Investigators who have employed more narrow or restrictive criteria for the disorder report lower prevalence rates (Messias, Chen, & Eaton, 2007).

The life of John Forbes Nash, a mathematician, was portrayed in the film *A Beautiful Mind*. Nash won the Nobel Prize for Economics in 1994. His thesis, written at the age of 21, revolutionized the field of game theory. He has recovered from paranoid schizophrenia, after experiencing psychotic symptoms for more than 20 years.

GENDER DIFFERENCES

Although experts have believed for many years that men and women are equally likely to develop schizophrenia, this conclusion has been challenged by several recent studies. Current evidence suggests that men are 30 to 40 percent more likely to develop schizophrenia than women (Seeman, 2008).

There are some interesting and widely recognized differences between male and female patients with regard to patterns of onset, symptoms, and course of the disorder. For example, the average age at which schizophrenic males begin to exhibit overt symptoms is younger by about four or five years than the average age at which schizophrenic women first experience problems. A summary of reported gender differences in schizophrenia is presented in Table 13.2. Male patients are more likely than female patients to exhibit negative symptoms, and they are also more likely to follow a chronic, deteriorating course (Atalay & Atalay, 2006; Moriarty et al., 2001).

Gender differences in the age of onset and symptomatic expression of schizophrenia can be interpreted in several ways. The alternatives fall into two types of hypotheses. One approach assumes that schizophrenia is a single disorder and that its expression varies in men and women. A common, genetically determined vulnerability to schizophrenia might be expressed differently in men than in women. Mediating factors that might account for this difference could be biological differences between men and women—perhaps involving certain hormones—or different environmental demands, such as the timing and form of stresses associated with typical male and female sex roles. An alternative approach suggests that there are two qualitatively distinct subtypes of schizophrenia: one with an early onset that affects men more often than women, and another with a later onset that affects women more often than men. Both approaches assume a combination of genetically determined

TABLE 13.2 Typical Gender Differences in Schizophrenia

Variable	Men	Women
Age of onset	Earlier (18–25)	Later (25–35)
Premorbid functioning; adjustment	Poor social functioning; more schizotypal traits	Good social functioning; fewer schizotypal traits
Typical symptoms	More negative symptoms; more withdrawn and passive	More hallucinations and paranoia; more emotional and impulsive
Course	More often chronic; poorer response to treatment	Less often chronic; better response to treatment

Source: Based on J. M. Goldstein, 1995, "The Impact of Gender on Understanding the Epidemiology of Schizophrenia," in M.V. Seeman, Ed., *Gender and Psychopathology,* pp. 159–199. Washington, DC: American Psychiatric Press.

predisposition to disorder with the onset of symptoms being triggered by environmental events. The available evidence does not allow us to favor one of these explanations over the other (Haefner et al., 1998; Taylor & Langdon, 2006).

CROSS-CULTURAL COMPARISONS

Schizophrenia has been observed in virtually every culture that has been subjected to careful scrutiny. Although it is a universal disorder, the frequency of schizophrenia is not constant around the world. The annual incidence of schizophrenia—that is, the number of *new* cases appearing in any given year—varies from one country to the next. Reported estimates range from 8 to 43 cases per 100,000 people (McGrath, 2005). Urban populations have higher rates than rural areas, but incidence is not related to a country's economic status (Saha et al., 2006). As epidemiologists attempt to unravel these differences and explain them, we will learn more about the causes of the disorder.

Substantial cross-cultural differences have also been found with regard to the course of schizophrenia. Two large-scale epidemiological studies, conducted by teams of scientists working for the World Health Organization (WHO), have drawn considerable attention to differences in short- and long-term outcome for schizophrenia in the third world and industrialized countries (Sartorius, 2007). The International Pilot Study of Schizophrenia (IPSS) began in the 1960s and was conducted in nine countries in Europe, North America, South America, Africa, and Asia. It included 1,200 patients who were followed for 15 to 25 years after their initial hospitalization. The Collaborative Study on the Determinants of Outcome of Severe Mental Disorders (DOS) was begun a few years later in six of the same countries that participated in the IPSS, plus four others. The DOS study included more than 1,500 patients. Both the IPSS and DOS projects examined rural and urban areas in both Western and non-Western countries. For purposes of cultural comparison, the countries were divided into those that were "developing" and those that were already "developed" on the basis of prevailing socioeconomic conditions. All the interviewers were trained in the use of a single, standardized interview schedule, and all employed the same sets of diagnostic criteria.

In what way does the long-term course of schizophrenia differ between developing and developed countries? Why?

The IPSS results indicated that patients who exhibited characteristic signs and symptoms of schizophrenia were found in all of the study sites. Comparisons of patients across research centers revealed more similarities than differences in clinical symptoms at the time of entry into the study, which was always an active phase of disorder that required psychiatric treatment. The IPSS investigators found that clinical and social outcomes were significantly better for schizophrenic patients in developing countries than in developed countries, such as the United States, England, and Russia. The DOS study confirmed those results (Hopper et al., 2007).

The WHO studies provide compelling support for the conclusion that, although the frequency of schizophrenia varies around the world, it is expressed in terms of similar symptoms in different cultures. Most experts believe that the more favorable clinical outcome that was observed in India and Nigeria is a product of the greater tolerance and acceptance extended to people with psychotic symptoms in developing countries. This conclusion is consistent with evidence regarding the relationship between frequency of relapse and patterns of family communication, which we consider later in this chapter in the section on expressed emotion. These cross-cultural data certainly testify to the important influence of culture in shaping the experience and expression of psychotic symptoms (Thakker & Ward, 1998; Whaley & Hall, 2009).

Causes

Having considered the defining characteristics of schizophrenia, ways in which it has been classified, and some basic information regarding its distribution within the general population, we now review the evidence regarding factors that might contribute to the development of the disorder, as well as its course and outcome.

BIOLOGICAL FACTORS

Many of the early investigators who defined schizophrenia at the beginning of the twentieth century believed that the disorder was the product of a biological dysfunction. At that time very little was known about human genetics or the

biochemistry of the brain. Research in the areas of molecular genetics and the neurosciences has progressed at an explosive rate in the past decade. Much of what we know today about the biological substrates of schizophrenia has emerged from advances that have taken place in other sciences.

Genetics The role of genetic factors has been studied more extensively with regard to schizophrenia than with any other type of mental disorder. The existing data are based on sophisticated methods that have been refined over many years. The cumulative weight of this evidence points clearly toward some type of genetic influence in the transmission of this disorder (Pogue-Geile & Gottesman, 2007).

Family Studies Figure 13.1 illustrates the lifetime risk for schizophrenia for various types of relatives of a person with schizophrenia. This figure was created by pooling data from 40 European studies that were published between 1920 and 1987 (Gottesman, 1991). All of the studies employed conservative diagnostic criteria for the disorder.

Consider the data for first-degree relatives and second-degree relatives. On average, *siblings* and children share 50 percent of their genes with the schizophrenic proband; nieces, nephews, and cousins share only 25 percent. The lifetime morbid risk for schizophrenia is much greater among first-degree relatives than it is among second-degree relatives. The risk in the second-degree relatives is greater than the 1 percent figure that is typically reported for people in the general population. As the degree of genetic similarity increases between an individual and a schizophrenic patient, the risk to that person increases. The family history data are consistent with the hypothesis that the transmission of schizophrenia is influenced by genetic factors (Goldstein et al., 2010). They do not prove the point, however, because family studies do not separate genetic and environmental events (see Chapter 2).

Twin Studies Several twin studies have examined concordance rates for schizophrenia. The results of these studies are also summarized in Figure 13.1. The average concordance rate for MZ twins is 48 percent, whereas the comparable figure for DZ twins is 17 percent. One study from Finland found a concordance rate of 46 percent among MZ twins and only 9 percent among DZ twins (Cannon et al., 1998). Although the specific rates vary somewhat from study to study, all of the published reports have found that MZ twins are significantly more likely than DZ twins to be concordant for schizophrenia. This pattern suggests strongly that genetic factors play an important role in the development of the disorder.

It should also be pointed out, however, that none of the twin studies of schizophrenia has found a concordance rate that even approaches 100 percent, which would be expected if genetic factors were entirely responsible for schizophrenia. Thus, the twin studies also provide compelling evidence for the importance of environmental events. Some people, like Marsha in the case presented earlier, apparently inherit a predisposition to the development of schizophrenia. Among that select group of vulnerable individuals, certain environmental events must determine whether a given person will eventually exhibit the full-blown symptoms of the disorder.

Adoption Studies Studies of children who were adopted away from their biological parents and reared by foster families provide further evidence regarding the impact of genetic and environmental factors. The first adoption study of schizophrenia began by identifying records for a group of 49 children who were born between 1915 and 1945 while their mothers were hospitalized for schizophrenia (Heston, 1966). All the children were apparently normal at birth and were separated from their mothers within three days of birth. To rule out possible exposure to the environment associated with the mother's psychosis, any child who had been in contact with maternal relatives was excluded from the study. A control group of children was selected using the admission records of foundling homes where many of the target children had originally been placed. These children were matched to the patients' children on a number of variables, including age, sex, type of eventual placement, and amount of time spent in institutions.

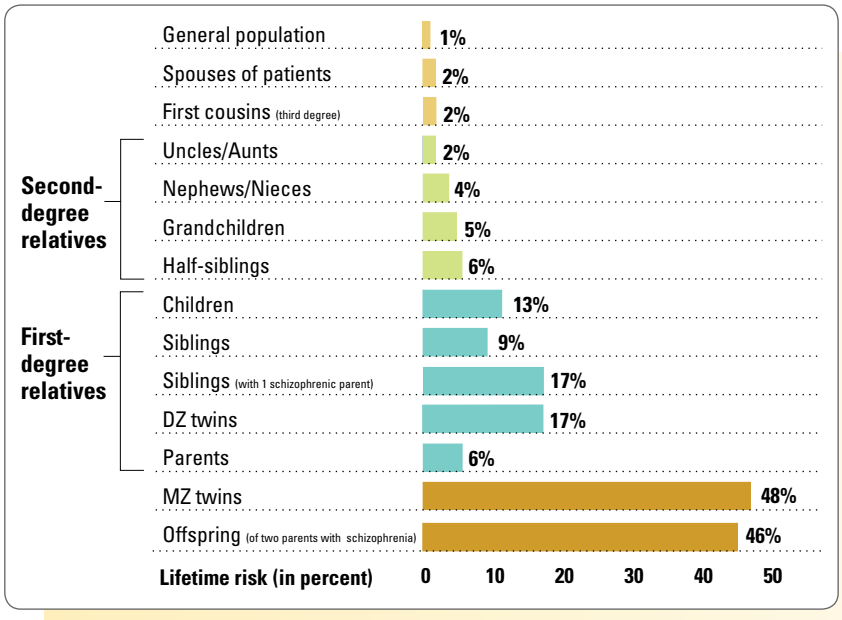


FIGURE 13.1 Average Risk of Developing Schizophrenia

Average risk of schizophrenia among biological relatives of a schizophrenic proband.

Source: I. I. Gottesman, 1991, *Schizophrenia Genesis: The Origins of Madness*, p. 96. New York: Freeman. Copyright © 1991 by Irving I. Gottesman. Reprinted by permission of W. H. Freeman and Company.

Most of the offspring were successfully located and interviewed when they were in their mid-thirties. Five of the adult offspring of schizophrenic mothers received a diagnosis of schizophrenia. Correcting for the fact that most of the participants were still within the period of risk for the disorder, this resulted in a lifetime morbidity risk for schizophrenia of 16.6 percent in the target group, which is almost exactly the rate observed among children of schizophrenic parents who were raised by their biological parents (see Figure 13.1). In contrast, none of the adult offspring in the control group received a diagnosis of schizophrenia. Because the only difference between the two groups was the genetic relationship between the target offspring and their schizophrenic biological mothers, these data indicate that genetic factors play a role in the development of the disorder. Several other adoption studies have been concerned with schizophrenia, and all reach the same conclusion as Heston's original report (Pogue-Geile & Gottesman, 2007).

The Spectrum of Schizophrenic Disorders Results from adoption and twin studies also provide interesting clues regarding the boundaries of the concept of schizophrenia. Several types of psychotic disorders and personality disorders resemble schizophrenia in one way or another, including schizoaffective disorder, delusional disorder, and schizotypal personality disorder (discussed in Chapter 9). Are these conditions a reflection of the same genetically determined predisposition as schizophrenia, or are they distinct disorders caused by different forces? If they are genetically related, then investigators should find that the biological relatives of schizophrenic adoptees are more likely to exhibit these conditions as well as schizophrenia. The overall pattern of results does suggest that vulnerability to schizophrenia is sometimes expressed as schizophrenia-like personality traits and other types of psychoses that are not specifically included in the DSM-IV-TR definition of schizophrenia (van Snellenberg & de Candia, 2009).

Why are some personality disorders considered to be schizophrenia spectrum disorders?

Molecular Genetics The combined results from twin and adoption studies indicate that genetic factors are involved in the transmission of schizophrenia. This conclusion does not imply, however, that the manner in which schizophrenia develops is well understood. We know little beyond the fact that genetic factors are involved in some way. The mode of transmission has not been identified. Most clinical scientists believe that schizophrenia is a polygenic characteristic, which means that it is the product of a reasonably large number of genes rather than a single gene (see Chapter 2).

One of the most exciting areas of research on genetics and schizophrenia focuses on molecular genetics (see Research Methods in Chapter 14 for an explanation of this process). Studies of this type are designed to identify specific genes that are responsible for the disorder (or some important components of the disorder). So far, investigators have not been able to identify any genes that account for a major proportion of the heritability of schizophrenia, but they have found several genes that apparently have a very small but measurable impact (Mitchell & Porteous, 2011; Owen et al., 2010; Sanders et al., 2008). Supporters of the search for specific genes involved in the transmission of schizophrenia contend that the absence of more definitive discoveries is not surprising when we consider the complexity of this process and the magnitude of the search. They feel that the search for a particular gene that causes schizophrenia will simply take more time (Cannon, 2010).

The devastating consequences of war include severe nutritional deficiencies, such as those suffered in Somalia by this mother and her 4-year-old son. The offspring of women who are pregnant during serious famines may be more likely to develop schizophrenia as they reach adulthood.

One specific gene that has attracted considerable research attention is associated with the production of catechol O-methyltransferase (COMT), which is an enzyme that is involved in breaking down the neurotransmitter dopamine. The COMT gene is located on chromosome 22, a region that has been linked to schizophrenia. People who possess a specific form of the COMT gene (called the Val allele) seem to have a small but consistently increased risk for schizophrenia (Glatt, Faraone, & Tsuang, 2003). Scientists believe that this gene may increase risk for schizophrenia by affecting dopamine transmission in the prefrontal cortex of the brain, with the net effect being impaired cognitive ability (Prata et al., 2009; van Haren, Bakker, & Kahn, 2008) (see later section on working memory and vulnerability to schizophrenia).

Pregnancy and Birth Complications People with schizophrenia are more likely than the general population to have been exposed to various problems during their mother's pregnancy and to have suffered birth injuries. Problems during pregnancy include the mother's contracting various types of

diseases and infections. Birth complications include extended labor, breech delivery, forceps delivery, and the umbilical cord wrapped around the baby's neck. These events may be harmful, in part, because they impair circulation or otherwise reduce the availability of oxygen to developing brain regions. Birth records indicate that the mothers of people who later develop schizophrenia experienced more complications at the time of labor and delivery (Cannon, Jones, & Murray, 2002).

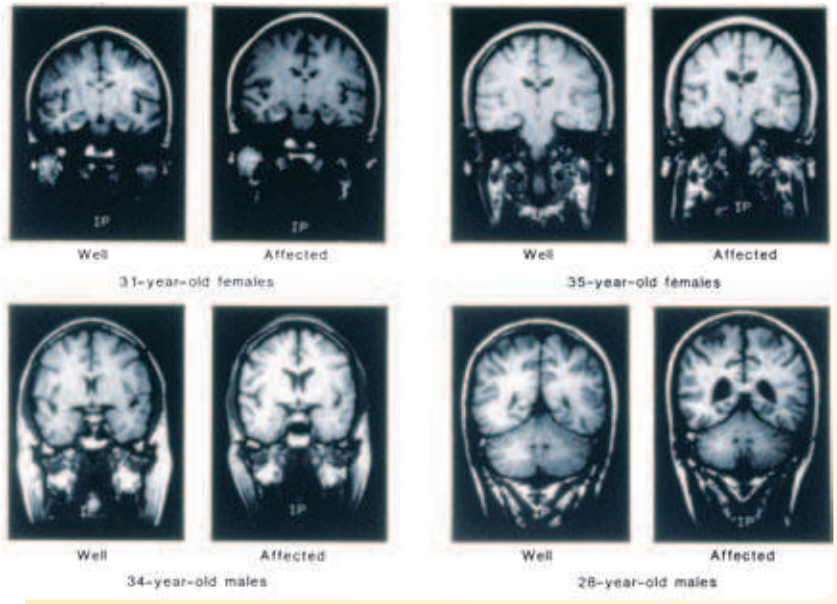
It is not clear whether the effects of pregnancy and birth complications interact with genetic factors. They may produce neurodevelopmental abnormalities that result in schizophrenia regardless of family history for the disorder. Conversely, a fetus that is genetically predisposed to schizophrenia may be more susceptible to brain injury following certain kinds of obstetric difficulties (Walker et al., 2004).

Dietary factors may also play a role in the etiology of the disorder. Severe maternal malnutrition in the early months of pregnancy leads to an increased risk of schizophrenia among the offspring. This conclusion is based on a study of medical and psychiatric records of people who were born in the western part of the Netherlands between 1944 and 1946 (Susser et al., 1996). The German blockade of ports and other supply routes in this area led to a severe famine at the end of World War II. People who were conceived during the worst months of the famine were twice as likely to develop schizophrenia than were people whose mothers became pregnant at other times, including the early months of the famine. These results, coupled with more recent findings, suggest that prenatal nutritional deficiencies may disrupt normal development of the fetal nervous system (Abel et al., 2010; Insel et al., 2008).

Viral Infections Some speculation has focused on the potential role that viral infections may play in the etiology of schizophrenia (Brown & Derkits, 2010). One indirect line of support for this hypothesis comes from studies indicating that people who develop schizophrenia are somewhat more likely than other people to have been born during the winter months (McGrath & Welham, 1999). Some clinicians interpret this pattern to mean that, during their pregnancies, the mothers were more likely to develop viral infections, which are more prevalent during the winter. Exposure to infection presumably interferes with brain development in the fetus. This possibility has received considerable attention in the research literature and remains an important topic of debate (Clarke et al., 2009).

Neuropathology One important step toward understanding the etiology of schizophrenia would be to identify its neurological underpinnings. If people with schizophrenia suffer from a form of neurological dysfunction, shouldn't it be possible to observe differences between the structure of their brains and those of other people? This is a challenging task. Scientists have invented methods to create images of the living human brain (see Chapter 4). Some of these procedures provide static pictures of various brain structures at rest, just as an X-ray provides a photographic image of a bone or some other organ of the body. More recently, sophisticated methods have enabled scientists to create functional images of the brain while a person is performing different tasks. Studies using these techniques have produced evidence indicating that a number of brain areas are involved in schizophrenia (Minzenberg et al., 2009; Reichenberg & Harvey, 2007). You may want to review the description of brain structures in Chapter 2 (Figure 2.3) before reading the next sections of this chapter.

Structural Brain Imaging Many investigations of brain structure in people with schizophrenia have employed magnetic resonance imaging (MRI; see Chapter 4 for an explanation of this process). The disorder is not associated with abnormalities in one specific brain region or in one particular type of nerve cell. Rather, it seems to affect many different regions of the brain and the ways in which they connect or communicate with each other (Niznikiewicz, Kubicki, & Shenton, 2003). Most MRI studies have reported a decrease in total volume of brain tissue among schizophrenic patients. Another consistent finding is that some people with schizophrenia have mildly to moderately enlarged lateral ventricles, the cavities on each side of the brain that are filled with cerebrospinal fluid.



MRI scans from four identical twin pairs discordant for schizophrenia showing varying degrees of increased ventricular size in the twin with the disorder compared to the twin who is well.

These differences seem to reflect a natural part of the disorder rather than a side effect of treatment with antipsychotic medication. In fact, some studies have found enlarged ventricles in young schizophrenic patients before they have been exposed to any form of treatment (Steen et al., 2006). Some studies have also found enlarged ventricles prior to the onset of symptoms. The structural changes seem to occur early in the development of the disorder and therefore may play a role in the onset of symptoms (DeLisi, 2008; Weinberger & McClure, 2002).

The temporal lobes have also been studied extensively using MRI scans. Several studies have reported decreased size of the hippocampus, the parahippocampus, the amygdala, and the thalamus, all of which are parts of the limbic system (Price et al., 2006). These areas of the brain (see Figure 13.2) play a crucial role in the regulation of emotion as well as the integration of cognition and emotion. Decreased size of these structures in the limbic area of the temporal lobes may be especially noticeable on the left side of the brain, which plays an important role in the control of language.

Many questions remain to be answered regarding the relation between structural brain abnormalities and schizophrenia. Does the pattern reflect a generalized deterioration of the brain, or is it the result of a defect in specific brain sites? We don't know. Is the presence of enlarged ventricles and cortical atrophy consistently found in some subset of schizophrenic patients? Some investigators have reported an association between this type of neuropathology and other factors, such as negative symptoms, poor response to medication, and absence of family history of the disorder. These are all interesting possibilities, but none has been firmly established.

Functional Brain Imaging In addition to static pictures of brain structures, clinical scientists use techniques that provide dynamic images of brain functions. One dynamic brain imaging technique, known as positron emission tomography (PET), can reflect changes in brain activity as the person responds to various task demands. Visual stimulation will produce increased cerebral blood flow in the visual cortex; people performing a simple motor task exhibit increased flow in the motor cortex. Functional MRI is another tool that can be used to observe brain activity. The results of studies using these techniques suggest dysfunction in various neural circuits, including some regions of the prefrontal cortex (see Figure 13.3) and several regions in the temporal lobes (Bonner-Jackson et al., 2005; Hall et al., 2010). The problems seem to involve activities within, as well as integration between, a variety of functional circuits rather than a localized abnormality in one region of the brain.

The role of neurological abnormalities in schizophrenia has been highlighted by a study of identical twins conducted by investigators at the National Institute of Mental Health (NIMH). Participants included 27 pairs of twins discordant for schizophrenia and 13 pairs that were concordant for the disorder. Changes in brain structure, measured by MRI, and changes in brain function, measured by cerebral blood flow, were prominent in the twins who had developed schizophrenia. Their well co-twins also exhibited more neurological impairment than a group of normal control participants, but these abnormalities were less marked than those found in the probands. Among discordant monozygotic pairs, the schizophrenic twin typically had the smaller hippocampus and smaller amygdala. The schizophrenic twins always showed

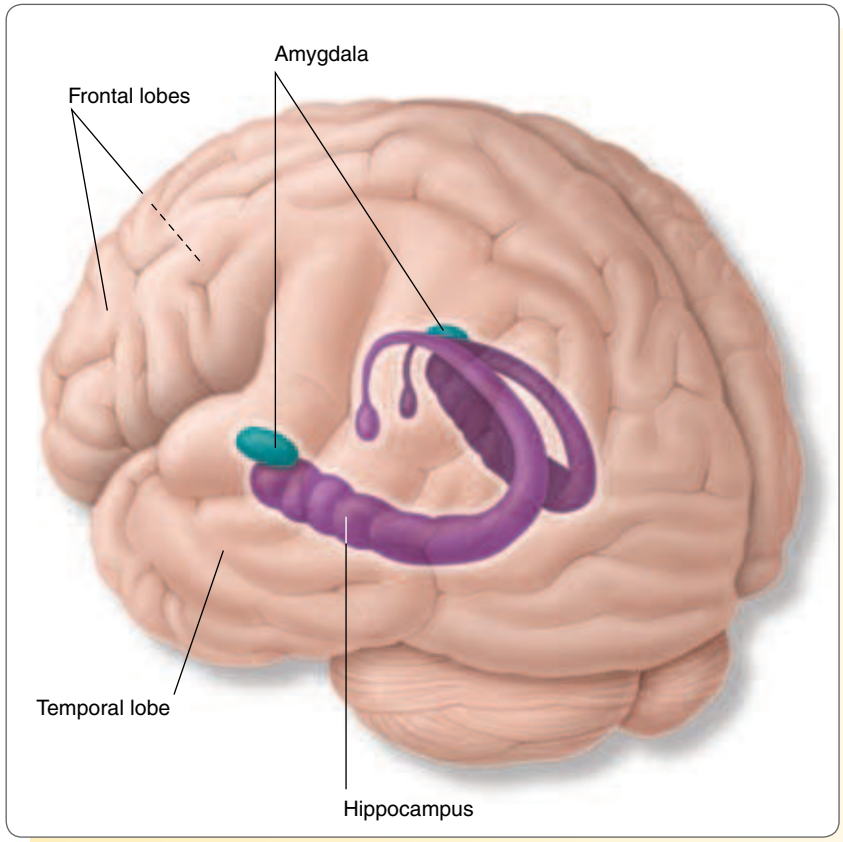


FIGURE 13.2 Structures of the Brain Implicated in Schizophrenia

Structural imaging procedures indicate reduced size of temporal lobe structures, such as the hippocampus and amygdala, among some patients with schizophrenia.

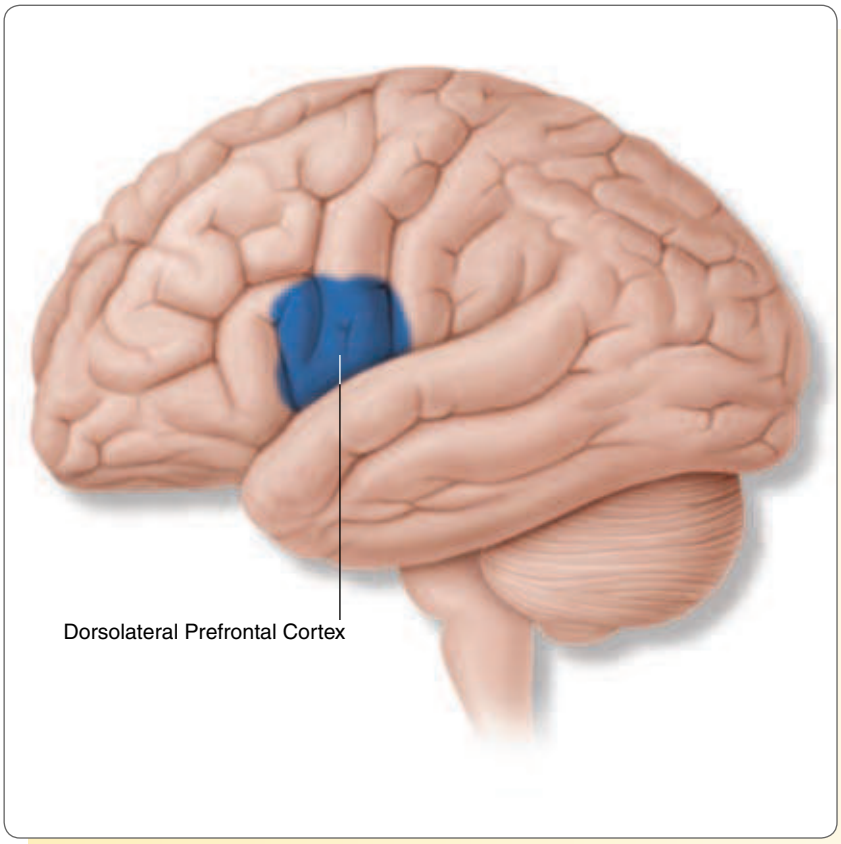


FIGURE 13.3 Areas of Brain Function Implicated in Schizophrenia

Neural circuits in the dorsolateral prefrontal cortex may function improperly in schizophrenia.

reduced frontal lobe activity compared with their unaffected co-twins. Results for enlarged ventricles were less consistent. In general, neurological dysfunction seemed to be associated with the overall severity of the disorder rather than being indicative of an etiologically distinct subgroup of patients (Bridle et al., 2002).

General Conclusions The primary conclusion that can be drawn from existing brain imaging studies is that schizophrenia is associated with diffuse patterns of neuropathology. The most consistent findings point toward structural as well as functional irregularities in the frontal cortex and limbic areas of the temporal lobes, which play an important role in cognitive and emotional processes. The neural network connecting limbic areas with the frontal cortex may be fundamentally disordered in schizophrenia.

Speculation regarding disruptions in neural circuitry must also be tempered with caution. Evidence of neuropathology does not seem to be unique to schizophrenic patients. Many patients with other psychiatric and neurological disorders show similar changes in brain structure and function. Furthermore, a specific brain lesion has not been identified, and it is unlikely that one will be found. It is unlikely that a disorder as complex as schizophrenia will be traced to a single site in the brain. The various symptoms and cognitive deficits that have been observed in schizophrenic patients may be linked to a host of subtle disruptions in neurological functions (Csernansky & Cronenwett, 2008; Green, 2001).

It should also be emphasized that brain imaging procedures are not diagnostically meaningful tests for mental disorders. For example, an MRI showing enlarged ventricles does not prove that a patient has schizophrenia. Brain imaging procedures have identified interesting group differences, but they do not predict the presence of schizophrenia for individuals. The group differences that have been observed are very subtle in comparison to the levels of neuropathology found in disorders such as Alzheimer's disease and Huntington's disease (see Chapter 14). Some schizophrenic patients do not show abnormalities in brain structure or function.

Why can't we use brain imaging to diagnose schizophrenia?

A dramatic example of this point was found in the NIMH study of discordant MZ twins. In one pair, the well twin was a successful businessman who had never had any problems with mental disorder. His twin brother had been severely impaired with schizophrenia for 20 years. The well twin had ventricles that were five times larger than those of the schizophrenic twin. Thus, we should approach all these hypotheses with caution and skepticism.

Neurochemistry The neurological underpinnings of schizophrenia may not take the form of changes in the size or organization of brain structures. They may be even more subtle, involving alterations in the chemical communications among neurons within particular brain circuits.

The Dopamine Hypothesis Scientists have proposed various neurochemical theories to account for the etiology of schizophrenia. The most influential theory, known as the *dopamine hypothesis,* focuses on the function of specific dopamine pathways in the limbic area of the brain. The original version of the dopamine hypothesis proposed that the symptoms of

schizophrenia are the product of excessive levels of dopaminergic activity. This hypothesis grew out of attempts to understand how antipsychotic drugs improve the adjustment of many schizophrenic patients. Animals who receive doses of antipsychotic drugs show a marked increase in the production of dopamine. In 1963, it was suggested that antipsychotic drugs block postsynaptic dopamine receptors. The presynaptic neuron recognizes the presence of this blockade and increases its release of dopamine in a futile attempt to override it (Carlsson & Lindqvist, 1963).

If the dopamine system is dysfunctional in schizophrenic patients, what is the specific form of this problem? One possibility is that certain neural pathways have an elevated sensitivity to dopamine because of increased numbers of postsynaptic dopamine receptors. The potency of various types of antipsychotic drugs is specifically related to their ability to block one type of dopamine receptor, known as receptors. Imaging studies of brain functions in patients with schizophrenia have found that elevated levels of dopamine functioning in the striatum (Howes et al., 2009).

Interactions of Multiple Neurotransmitters A dysregulation and exaggerated response of certain dopamine pathways is certainly involved in schizophrenia, at least for some patients. On the other hand, experts now agree that several other neurotransmitters also play an important role. A neurochemical model focused narrowly on dopamine fails to explain many different aspects of the disorder, including the following: Some patients do not respond positively to drugs that block dopamine receptors; the effects of antipsychotic drugs require several days to become effective, but dopamine blockage begins immediately; research studies that examined the by-products of dopamine in cerebrospinal fluid were inconclusive at best.

Current neurochemical hypotheses regarding schizophrenia focus on a broad array of neurotransmitters (Carlsson et al., 2001). Special interest has been focused on serotonin pathways since the introduction of a new class of antipsychotic drugs such as clozapine (Clozaril) that are useful in treating patients who were resistant to standard antipsychotic drugs. (See the section on treatment.) These "atypical" antipsychotics produce a strong blockade of serotonin receptors and only a weak blockade of D_2 receptors. This pattern leads to speculation that the neurochemical substrates of schizophrenia may involve a complex interaction between serotonin and dopamine pathways in the brain (Downar & Kapur, 2008).

Brain imaging studies that point to problems in the prefrontal cortex have also drawn attention to glutamate and GABA (gamma-aminobutyric acid), the two principal neurotransmitters in the cerebral cortex (Wassef, Baker, & Kochan, 2003). Glutamate is an excitatory neurotransmitter, and GABA is an inhibitory neurotransmitter. As in the case of serotonin, hypotheses regarding the role of glutamate and GABA focus on their interactions with dopamine pathways, especially those connecting temporal lobe structures with the prefrontal and limbic cortexes.

SOCIAL FACTORS

There is little question that biological factors play an important role in the etiology of schizophrenia, but twin studies also provide compelling evidence for the importance of environmental events. The disorder is expressed in its full-blown form only when vulnerable individuals experience some type of environmental event, which might include anything from nutritional variables to stressful life events (Howes et al., 2004; Walker et al., 2004). What sorts of nongenetic events interact with genetic factors and other biological factors to produce schizophrenia? We will review some of the hypotheses that have been proposed and studied.

Social Class One general indicator of a person's status within a community's hierarchy of prestige and influence is social class. People from different social classes are presumably exposed to different levels of environmental stress, with those people in the lowest class being subjected to the most hardships. More than 50 years ago, social scientists working in Chicago found that the highest prevalence of schizophrenia was found in neighborhoods of the lowest socioeconomic status (Faris & Dunham, 1939). Many research studies have subsequently confirmed this finding in several other geographic areas (Boydell & Murray, 2003). The evidence supporting an inverse relationship between social class and schizophrenia is substantial.

There are two ways to interpret the relationship between social class and schizophrenia. One holds that harmful events associated with membership in the lowest social classes, which might include many factors ranging from stress and social isolation to poor nutrition, play a causal role in the development of the disorder. This is often called the *social causation* hypothesis. It is also possible, however, that low social class is an outcome rather than a cause of schizophrenia. Those people who develop schizophrenia may be less able than others to complete a higher level education or to hold a well-paying job. Their cognitive and social impairments may cause downward social mobility. In other words, regardless of the social class of their family of origin, many schizophrenic patients may gradually drift into the lowest social classes. This view is sometimes called the *social selection hypothesis.*

Research studies have found evidence supporting both views. The social selection hypothesis is supported by studies that have compared the occupational roles of male schizophrenic patients with those of their fathers. The patients are frequently less successful than their fathers, whereas the opposite pattern is typical of men who do not have schizophrenia (Jones et al., 1993). It is also true, however, that a disproportionately high percentage of the fathers of schizophrenic patients were from the lowest social class (Harrison et al., 2001). This finding is consistent with the social causation hypothesis.

Migrant Studies Higher rates of schizophrenia have also been found repeatedly among people who have migrated to a new country (Cantor-Graae & Selton, 2005). Several influential studies of this sort focused on African Caribbean people who moved to the United Kingdom from Jamaica, Barbados, and Trinidad. Risk for schizophrenia in these migrant groups was found to be several times higher than the risk observed in the native-born U.K. population. It was also much higher than the risk observed among people living in the migrants' countries of origin. Subsequent studies demonstrated that the effect is not unique to the United Kingdom. Larger effects are reported for migrants from developing rather than developed countries, and they are also larger for migrants from countries where the majority population is black. One possible explanation for this phenomenon is that social adversity increases risk for schizophrenia. Migrants tend to settle in urban areas where they may be exposed to discrimination and other forms of disadvantage (Fearon & Morgan, 2006; Weiser et al., 2008).

In general, the evidence regarding socioeconomic status and schizophrenia indicates that the disorder is, to a certain extent, influenced by social factors. Adverse social and economic circumstances may increase the probability that persons who are genetically predisposed to the disorder will develop its clinical symptoms (van Os & McGuffin, 2003).

PSYCHOLOGICAL FACTORS

Most of the attention devoted to psychological factors and schizophrenia has focused on patterns of behavior and communication within families. Research evidence indicates that family interactions and communication problems are not primarily responsible for the initial appearance of symptoms. Disturbed patterns of communication among family members do not *cause* people to develop schizophrenia. This knowledge is important to parents of schizophrenic patients. They experience enough emotional anguish without also being made to feel that something they did or said was the primary cause of their child's problems.

Expressed Emotion The family environment does have a significant impact on the course (as opposed to the original onset) of schizophrenia. Studies of this effect are concerned with the adjustment of patients who have already been treated for schizophrenic symptoms.

This effect was discovered by people who were interested in the adjustment of patients who were discharged after being treated in a psychiatric hospital. Men with schizophrenia were much more likely to return to the hospital within the next nine months if they went to live with their wives or parents than if they went to live in other lodgings or with their siblings. The patients who relapsed seemed to react negatively to some feature of their close relationship with their wives or mothers.

Subsequent research confirmed this initial impression (Vaughn & Leff, 1976). Relatives of schizophrenic patients were interviewed prior to the patients' discharge from the hospital, and many of the relatives made statements that reflected negative or intrusive attitudes toward the patient. These statements were used to create a measure of **expressed emotion (EE).** For example, many of the relatives expressed hostility toward the patient or repeatedly criticized the patient's behavior. The following comments, made by the stepfather of a young man with schizophrenia, illustrate generalized, hostile criticisms of the patient's behavior. These comments would be considered to be high in expressed emotion.

> **Interviewer**: What seemed different about Stephen's behavior?
> **Stepfather:** Everything and anything. In other words, he's the type of person, you don't tell him, he tells you.
> **Interviewer**: You say that he spent time in a juvenile facility?
> **Stepfather:** Yeah. This kid is a genuine con artist, believe me. I spent time in the service and I've been around con artists. This kid is a first-class, genuine con artist, bar none.
> (Leff & Vaughn, 1985, p. 42)

Other family members appeared to be overprotective or too closely identified with the patient. These phenomena are also rated as being high in expressed emotion. Of course, a certain amount of worrying and concern should be expected from a parent whose child has developed a severe disorder such as schizophrenia. In the assessment of expressed emotion, relatives were considered to be emotionally overinvolved if they reported responses such as extreme anxiety or exaggerated forms of self-sacrifice. For example, the following exchange illustrates emotional overinvolvement (high EE) by the mother of a 24-year-old male patient who had his first onset of the disorder when he was 22:

> **Mother:** He talked to me a lot—because I was his therapist—the person he shared with more than anybody else. He involves me, ruminates with me, because I allow him to do it.
> **Interviewer**: How frequently?
> **Mother:** He would do it constantly. He would do it as much as I would be there with him.
> **Interviewer**: Once or twice a week?
> **Mother:** No, it happened daily. All the time I was with him, particularly in the last four or five months. He would talk to me for hours at a time, worrying and sharing how bad he felt, reporting to me every change in mood or feeling from 5-minute to 5-minute period.
> (Leff & Vaughn, 1985, p. 51)

Patients who returned to live in a home with at least one member who was high in EE were more likely than patients from low EE families to relapse in the first nine months after discharge. This result has been replicated many times (Marom et al., 2005). Approximately half of schizophrenic patients live in families that would be rated as being high in EE. Average relapse rates—defined primarily in terms of the proportion of patients who show a definite return of positive symptoms in the first year following hospital discharge—are 52 percent for patients in high EE families and 22 percent for patients in low EE families. Among the various types of comments that can contribute to a high EE rating, criticism is usually most strongly related to patients' relapse (Hooley & Gotlib, 2000).

Criticism and hostility can increase the risk of relapse for some patients with schizophrenia. Conversely, warmth and family support can serve as a protective factor.

High EE seems to be related, at least in part, to relatives' knowledge and beliefs about their family member's problems. Relatives find it easier to accept the most obvious positive symptoms as being the product of a mental disorder (Brewin et al., 1991). They show less tolerance toward negative symptoms, such as avolition and social withdrawal, perhaps because the patient may appear to be simply lazy or unmotivated.

Understanding Family Attitudes The influence of expressed emotion is not unique to schizophrenia. Patients with mood disorders, eating disorders, panic disorder with agoraphobia, and obsessive–compulsive disorder are also more likely to relapse following discharge if they are living with a high EE relative (Miklowitz, 2004). In fact, EE is an even better predictor of outcome for mood disorders and eating disorders than it is for schizophrenia (Butzlaff & Hooley, 1998). The extension of this phenomenon to other disorders should not be taken to mean that it is unimportant or that the social context of the family is irrelevant to our understanding of the maintenance of schizophrenia (see Research Methods). It may indicate, however, that this aspect of the causal model is shared with other forms of psychopathology. The specific nature of the person's symptoms may hinge on the genetic predisposition.

Cross-cultural evidence suggests that high EE may be more common in Western or developed countries than in non-Western or developing countries (Kymalainen & Weissman de Mamani, 2008). This observation might help explain why the long-term course of schizophrenia is typically less severe in developing countries. Some speculation has focused on family members' attitudes and beliefs: People in developing

RESEARCH METHODS

COMPARISON GROUPS: WHAT IS NORMAL?

Research studies in the field of psychopathology typically involve comparisons among two or more groups of participants. One group, sometimes called "cases," includes people who already meet the diagnostic criteria for a particular mental disorder, such as schizophrenia. Comparison groups are composed of people who do not have the disorder in question. This approach is sometimes called the case control design because it depends on a contrast between cases and control participants. If the investigators find a significant difference between groups, they have demonstrated that the dependent variable is correlated with the disorder (see Research Methods in Chapter 2). They hope to conclude that they have identified a variable that is relevant to understanding the etiology of this condition. Causal inferences are risky, however, in correlational research. Our willingness to accept these conclusions hinges in large part on whether the investigators selected an appropriate comparison group.

People conducting correlational research must make every effort to identify and test a group of people who are just like the cases except that they do not have the disorder in question (Gehlbach, 1988). This typically means that the people in both groups should be similar with regard to such obvious factors as age, gender, and socioeconomic background. If the investigators find differences between people who have the disorder and those who do not, they want to attribute those differences to the disorder itself. Two main types of comparison groups are used in psychopathology research: people with no history of mental disorder, sometimes called "normal participants," and people who have some other form of mental disorder, sometimes called "patient controls."

Selecting normal comparison groups is not as simple as it might seem. In fact, researchers must make several basic decisions. Does "normal" mean that the person has never had the disorder in question, or does it mean a complete absence of *any* type of psychopathology? Should people be included as normal control participants if they have a family history of the disorder, even though they do not have the disorder themselves?

Should we expect to find unique causes for each type of mental disorder?

A second research strategy involves comparing patients with one type of disorder to those who have another form of psychopathology. Investigators usually employ this strategy to determine whether the variable in question is specifically related to the disorder that they are studying. Are enlarged lateral ventricles or family communication problems unique to people with schizophrenia? Lack of specificity may raise questions about whether this variable is related to the cause of the disorder. It might suggest that this particular variable is, instead, a general consequence of factors such as hospitalization, which the patient control group has also experienced.

Many of the causal factors that we have discussed in this chapter are not unique to schizophrenia. For example, expressed emotion predicts relapse among patients with mood disorders as well as among those with schizophrenia. Should this result be taken to mean that EE does not play an important role in the development of schizophrenia? Not necessarily. The answer to this question depends on the specific causal model that is being considered (Garber & Hollon, 1991). All forms of psychopathology depend on the interaction of multiple factors spanning biological, social, and psychological systems. Some of these may be specific to the disorder being studied, and others may be general. The development of schizophrenia may depend on a specific genetically determined predisposition. The environmental events that are responsible for eventually causing vulnerable people to express this disorder might be nonspecific. The fact that similar factors influence people with mood disorders should not be taken to mean that EE is not an important factor in the complex chain of events that explain schizophrenia.

countries may be more tolerant of eccentric behavior among their extended family members. These attitudes may create environments similar to those found in low EE homes in the West. An alternative view places greater emphasis on the culturally determined relationships between patients and other members of their families (Aguilera et al., 2010). Studies of Mexican American families suggest that prosocial aspects of interactions between patients and their families can enhance family cohesion and decrease the stigma associated with serious mental disorders. In some cultures, family warmth serves as a protective factor and reduces the probability of patients' relapse (López et al., 2004).

We must be cautious to avoid a narrow view of this phenomenon. The concept of expressed emotion raises extremely sensitive issues for family members, who have too frequently been blamed for the problems of people with schizophrenia. Expressed emotion is not the only factor that can influence the course of a schizophrenic disorder. Some patients relapse in spite of an understanding, tolerant family environment. Furthermore, research studies have shown that the relationship between patients' behavior and relatives' expressed emotion is a transactional or reciprocal process. In other words, patients influence their relatives' attitudes at the same time that relatives' attitudes influence patients' adjustment. Persistent negative attitudes on the part of relatives appear to be perpetuated by a negative cycle of interactions in which patients play an active role (Goldstein et al., 1997).

INTERACTION OF BIOLOGICAL AND ENVIRONMENTAL FACTORS

A useful etiological model for schizophrenia must include the interaction of genetic factors and environmental events. The heterogeneous nature of the disorder, in terms of symptoms as well as course, also suggests that schizophrenia should be explained in terms of multiple pathways (Tandon, Keshavan, & Nasrallah, 2008). Some forms of the disorder may be the product of a strong genetic predisposition acting in combination with relatively common psychosocial experiences, such as stressful life events or disrupted communication patterns. For other people, relatively unusual circumstances, such as severe malnutrition during pregnancy, may be responsible for neurodevelopmental abnormalities that eventually lead to the onset of psychotic symptoms in the absence of genetic vulnerability (Gilmore, 2010).

Various kinds of environmental events have been linked to the etiology of schizophrenia. Some may operate in interaction with the genotype for schizophrenia; others may be sufficient to produce the disorder on their own. Considerable speculation has focused recently on biological factors, such as viral infections and nutritional deficiencies. Psychosocial factors, such as adverse economic circumstances, may also be involved. These events may be particularly harmful to people who are genetically predisposed to the disorder.

THE SEARCH FOR MARKERS OF VULNERABILITY

Some people apparently inherit a predisposition to schizophrenia. Obviously, it would be useful to be able to identify those people. Studies of molecular genetics will be part of that answer, if several genes are found to be responsible for the disorder. The search for more precise information about the development of the disorder may also hinge on our ability to identify vulnerability markers, which have also been called *endophenotypes* (Gottesman & Gould, 2003; Greenwood et al., 2007). An endophenotype is a component or trait that lies somewhere on the pathway between the genotype, which lays the foundation for the disorder, and full-blown symptoms of the disorder. It can be measured with precise laboratory procedures of many kinds, but it cannot be seen by the unaided eye.

If we are looking for signs of vulnerability—or endophenotypes—that can be detected among individuals who are genetically predisposed to schizophrenia, where should we look? What form will these signs take? Is it possible to detect signs of vulnerability among individuals who approach the threshold for developing schizophrenia spectrum disorders but have not exhibited any kind of overt symptoms? This issue has attracted considerable attention, but we don't have firm answers to these questions.

People who are vulnerable to schizophrenia might be identified by developing measures that could detect the underlying biological dysfunction or by developing sensitive measures of their subtle eccentricities of behavior. The range of possible markers is, therefore, quite large.

Assume that we have selected a specific measure, such as a biochemical assay or a psychological test, and we are interested in knowing whether it might be useful in identifying people who are vulnerable to schizophrenia. What criteria should a **vulnerability marker** fulfill? First, the proposed marker must distinguish between people who already have schizophrenia and those who do not. Second, it should be a stable characteristic over time. Third, the proposed measure of vulnerability should identify more people among the biological relatives of schizophrenic patients than among people in the general population. For example, it should be found among the discordant MZ twins of schizophrenic patients, even if they don't exhibit any symptoms of schizophrenia. Finally, the proposed measure of vulnerability should be able to predict the future development of schizophrenia among those who have not yet experienced a psychotic episode (Braff, Schork, & Gottesman, 2007; Snitz, MacDonald, & Carter, 2006).

What characteristics would define a useful marker of vulnerability to schizophrenia?

Although reliable measures of vulnerability have not been identified, they are being actively pursued by many investigators with a wide variety of measurement procedures. In the following pages we will outline some of the psychological procedures that have been shown to be among the most promising.

Working Memory Impairment Many investigators have pursued the search for signs of vulnerability by looking at measures of cognitive performance in which schizophrenic patients differ from other people. Some of these studies have focused on cognitive tasks that evaluate information processing, working memory, and attention/vigilance (Forbes et al., 2009; Green et al., 2004).

Considerable emphasis has been focused on one aspect of cognitive functioning known as *working memory,* or the ability to maintain and manipulate information for a short period of time. Working memory can be broken down into several more specific processes. Some of these involve memory buffers that provide short-term storage for visual and verbal information. The most important processes in working memory

involve a *central executive component* that is responsible for the manipulation and transformation of data that are held in the storage buffers. Many studies have reported that people with schizophrenia are impaired in their ability to perform laboratory tasks that depend on this central executive component of working memory (Barch, 2005; Gold et al., 2010).

The identification of deficits in working memory is particularly interesting with regard to schizophrenia because it links to other evidence regarding brain functions and this disorder. Processes that are associated with central executive processing are associated with brain activity located in the dorsolateral area of the prefrontal cortex (see Figure 13.3) which seems to be dysfunctional in schizophrenia. Neurochemical hypotheses regarding schizophrenia are also relevant in this regard because the dopamine neurotransmitter system plays a crucial role in supporting activities involved in working memory (Goldman-Rakic, Muly, & Williams, 2000).

Working memory problems seem to be a stable characteristic of patients with schizophrenia; they do not fluctuate over time (Cannon et al., 2002). Furthermore, these cognitive deficits are found with increased prevalence among the unaffected first-degree relatives of schizophrenic persons, including discordant MZ twins (Sitskoom et al., 2004). Finally, children who later receive a diagnosis of schizophrenia are more likely to have been impaired on tests of verbal working memory than are their siblings who do not develop the disorder. Therefore, measures of working memory fulfill several of the criteria for an index of vulnerability. The research indicates that problems in working memory may be useful signs of vulnerability to schizophrenia (Barch, 2005).

Eye-Tracking Dysfunction Another promising line of work involves impairments in eye movements—specifically, difficulty in tracking the motion of a pendulum or a similarly oscillating stimulus while the person's head is held motionless. When people with schizophrenia are asked to track a moving target, like an oscillating pendulum, with their eyes, a substantial number of them show dysfunctions in smooth-pursuit eye movement (Levy et al., 2010). Instead of reproducing the motion of the pendulum in a series of smooth waves, their tracking records show frequent interruptions of smooth-pursuit movements by numerous rapid movements. Examples of normal tracking records and those of schizophrenic patients are presented in Figure 13.4. Only about 8 percent of normal people exhibit the eye-tracking dysfunctions illustrated in part (C) of Figure 13.4, although some studies have reported higher figures.

Approximately 50 percent of the first-degree relatives of schizophrenic persons show similar smooth-pursuit impairments (Calkins, Iacono, & Ones, 2008; Hong et al., 2008). The overall pattern of results seen in people with schizophrenia and their families suggests that poor tracking performance may be associated with the predisposition to schizophrenia. That conclusion becomes even more interesting in light of evidence from additional studies suggesting that tracking ability is stable over time, influenced by genetic factors, and found among people who exhibit features associated with schizotypal personality disorder (Gooding, Miller, & Kwapil, 2000; O'Driscoll & Callahan, 2008).

It is not yet possible to identify people who are specifically predisposed to the development of schizophrenia, but research studies have identified potential vulnerability markers. The real test, of course, will center around predictive validity. Can any of these measures, such as working memory deficits or smooth-pursuit eye-tracking impairment, predict the later appearance of schizophrenia in people whose scores indicate possible vulnerability? High-risk studies will be useful in providing this type of evidence.

Treatment

Schizophrenia is a complex disorder that often must be treated over an extended period of time. Clinicians must be concerned about the treatment of acute psychotic episodes as well as the prevention of future episodes. A multifaceted approach to treatment is typically required. Antipsychotic medication is the primary mode of treatment for this disorder. Because many patients remain impaired between episodes, long-term care must often involve the provision of housing and social support. People with impaired social and occupational skills need special types of training. The treatment of schizophrenia requires attention on all of these fronts and is necessarily concerned with the cooperative efforts of many types of professionals (Lehman et al., 2004). Schizophrenia also takes its toll on families. The Getting Help section at the end of this chapter discusses some of the resources available for patients and families.

ANTIPSYCHOTIC MEDICATION

The many different forms of medication that are used to treat patients with schizophrenia can be divided into two broad categories. The first generation of drugs began to be introduced in the 1950s, and a second generation swept into practice in the 1990s. Both kinds of medication are in standard use today.

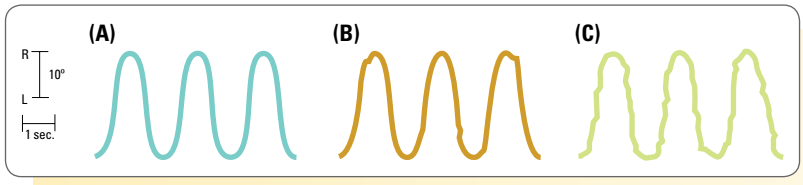


FIGURE 13.4 Eye-Tracking Patterns

This illustration contrasts smooth-pursuit eye-tracking patterns of normal subjects with those of schizophrenic patients. Part (A) shows the actual target. Part (B) illustrates the pattern for people without schizophrenia, and part (C) shows the pattern for people with schizophrenia.

Source: From D. L. Levy et al., Eye-tracking dysfunction and schizophrenia: A critical perspective. *Schizophrenia Bulletin,* (1993), 19, 462.

The first generation of antipsychotic drugs—also called classical or traditional antipsychotics—was discovered accidentally in the early 1950s. Early reports of success in treating chronic psychotic patients quickly led to the widespread use of these drugs, such as chlorpromazine (Thorazine), in psychiatric hospitals throughout Europe and the United States (Shen, 1999). This process quickly changed the way in which schizophrenia was treated. Large numbers of patients who had previously been institutionalized could be discharged to community care (but see Chapter 18 on the effects of deinstitutionalization).

Several related types of drugs were developed in subsequent years. They are called **antipsychotic drugs** because they have a relatively specific effect—to reduce the severity of psychotic symptoms. Some beneficial effects on problems such as agitation and hostility may be noticed within a week after the patient begins taking antipsychotic medication, but it usually takes two or three weeks before improvement is seen with regard to psychotic symptoms (Kutscher, 2008). Positive symptoms, such as hallucinations, respond better to antipsychotic medication than negative symptoms, such as alogia and blunted affect. Viewed from the patient's perspective, medication allows them to be less bothered or preoccupied by troublesome thoughts and perceptual experiences. In other words, they are able to distance themselves from their positive symptoms, even though the medication seldom eliminates hallucinations and delusional beliefs completely.

Double-blind, placebo-controlled studies have confirmed the effectiveness of antipsychotic medication in the treatment of patients who are acutely disturbed. Literally thousands of studies have addressed this issue over a period of more than 40 years (Hadden et al., 2009; Sharif et al., 2007). Most studies find that about half of the patients who receive medication are rated as being much improved after four to six weeks of treatment. Further improvements may continue beyond that point for some patients. In contrast, patients treated with placebos exhibit much smaller rates of improvement, and many of them actually deteriorate.

Unfortunately, a substantial minority of schizophrenic patients, perhaps 25 percent, do not improve on antipsychotic drugs (Conley & Kelly, 2001). Another 30 to 40 percent might be considered partial responders: Their condition improves, but they do not show a full remission of symptoms. Investigators have not been able to identify reliable differences between patients who improve on medication and those who do not. Some experts have suggested that treatment-resistant patients may have more prominent negative symptoms, greater disorganization, and more evidence of neurological abnormalities (Elkis, 2007).

Maintenance Medication After patients recover from acute psychotic episodes, there is a high probability that they will have another episode. The relapse rate may be as high as 65 to 70 percent in the first year after hospital discharge if patients discontinue medication. Continued treatment with antipsychotic drugs can reduce this rate to approximately 40 percent (Davis & Chen, 2003). Therefore, patients with schizophrenia are encouraged to continue taking medication after they recover from psychotic episodes, although usually at a lower dose. Unfortunately, may patients stop taking medication, often to avoid unpleasant side effects (Falkai, 2008).

Motor Side Effects Antipsychotic drugs produce several unpleasant side effects. They come in varying degrees and affect different patients in different ways. The most obvious and troublesome are called *extrapyramidal symptoms* (EPS) because they are mediated by the extrapyramidal neural pathways that connect the brain to the motor neurons in the spinal cord. These symptoms include an assortment of neurological disturbances, such as muscular rigidity, tremors, restless agitation, peculiar involuntary postures, and motor inertia. EPS may diminish spontaneously after the first few months of treatment, but some patients continue to experience EPS for many years.

Prolonged treatment with antipsychotic drugs can lead to the development of a more severe set of motor symptoms called *tardive dyskinesia* (TD). This syndrome consists of abnormal involuntary movements of the mouth and face, such as tongue protrusion, chewing, and lip puckering, as well as spasmodic movements of the limbs and trunk of the body. The latter include writhing movements of the fingers and toes and jiggling of the legs, as well as jerking movements of the head and pelvis. Taken as a whole, this problem is quite distressing to patients and their families. The TD syndrome is induced by antipsychotic treatment, and it is irreversible in some patients, even after the medication has been discontinued. In fact, in some patients, TD becomes worse if antipsychotic medication is withdrawn (Eberhard, Lindström, & Levander, 2006; Lauterbach et al., 2001).

Second-Generation Antipsychotics Several additional forms of antipsychotic medication began to be introduced in the 1990s. Although some clinicians hailed their development as a "second revolution" in the care of patients with schizophrenia, many experts now recognize that this claim has been overstated (Gray & Roth, 2007; Lieberman, 2006). These drugs are frequently called *atypical antipsychotics* because they are less likely than the classical antipsychotics to produce unpleasant motor side effects. The best known of the atypical drugs, clozapine (Clozaril), has been used extensively throughout Europe since the 1970s. The second generation of antipsychotic medications also includes risperidone (Risperdal), olanzapine (Zyprexa), quetiapine (Seroquel), and several other drugs that have recently become available or are waiting for approval by the FDA. Some of these drugs are listed in Table 13.3.

The good news about second-generation antipsychotics is that they are at least as effective as traditional drugs for the treatment of positive symptoms of schizophrenia (Lieberman et al., 2005; Sikich et al., 2008), and they are useful in maintenance treatment to reduce the risk of relapse (Wang et al., 2010). They are also less likely to produce tardive dyskinesia. One review of several outcome studies reported that 13 percent of patients taking second-generation antipsychotics developed tardive dyskinesia, compared to 32 percent patients taking first-generation antipsychotics (Correll & Shenk, 2008). The combination of beneficial effects on positive symptoms and reduced motor side effects makes these forms of medication a reasonable choice in the treatment of schizophrenia.

The bad news is that, contrary to initial claims, second-generation antipsychotics are not significantly more effective for treating negative symptoms (Buckley & Stahl, 2007; Murphy et al., 2006). They also produce additional side effects, and some of them are serious. For example, many of the atypical antipsychotics lead to weight gain and obesity. These problems increase the person's risk for additional medical problems, such as diabetes, hypertension, and coronary artery disease. These adverse reactions lead many patients to discontinue their

TABLE 13.3 Examples of Medications Used to Treat Schizophrenic Disorders

		Modes of Action			
		Selected Side Effects		Selected Receptors	
Drug Class	Generic Name (trade name)	EPS	Weight Gain	D_2	$5HT_{2A}$
First-generation antipsychotics	chlorpromazine (Thorazine)	++	+	++	+
	haloperidol (Haldol)	++++	+	++++	+
Second-generation antipsychotics	clozapine (Clozaril)	+/–	++++	++	++++
	risperidone (Risperdal)	++	++	+++	+++++
	olanzapine (Zyprexa)	+	++++	+++	++++
	quetiapine (Seroqul)	+/–	++	++	+++
	amisulpride (Solian)*	+	++	++++	–

D_2 = dopamine receptors; $5HT_{2a}$ = serotonin receptors.
*Amisulpride is not available in the United States, but it has been used for more than 15 years in France (Leucht et al., 2002).

Source: S. Kapur and G. Remington, 2001, "Atypical Antipsychotics: New Directions and New Challenges in the Treatment of Schizophrenia," *Annual Review of Medicine, 52,* pp. 503–517.

medication, thus increasing risk of relapse. One influential study compared one first-generation antipsychotic with four types of second-generation drugs. The investigators reported that 74 percent of patients stopped taking their prescribed medication before the end of the 18-month treatment period (Lieberman et al., 2005). Poor compliance was found for all of the different drugs. The bottom line is that various kinds of antipsychotic medication are beneficial for patients with schizophrenia, but they all have weaknesses, and none is without adverse side effects.

All antipsychotic medications—both first and second-generation forms—act by blocking dopamine receptors in the cortical and limbic areas of the brain (Factor, 2002). They also affect a number of other neurotransmitters, including serotonin, norepinephrine, and acetylcholine. Table 13.3 includes a comparison of two first-generation and five second-generation antipsychotic drugs in terms of their ability to block specific types of dopamine and serotonin receptors. Most second-generation antipsychotics produce a broader range of neurochemical actions in the brain than do the traditional drugs, which act primarily on dopamine receptors. Clozapine and olanzapine, for example, produce a relatively strong blockade of serotonin receptors and a relatively weaker blockade of dopamine receptors (Richelson, 1999). This increased affinity of some atypical drugs for serotonin receptors might explain why they can have a beneficial effect on symptoms of schizophrenia while producing fewer motor side effects (EPS). This hypothesis is contradicted, however, by the modes of action associated with a newer form of atypical drug, amisulpride, which does not affect serotonin receptors (Leucht, Kissling, & Davis, 2009). Neurochemical differences between different forms of antipsychotic drugs are not completely understood and are currently the topic of interesting debate (Richtand et al., 2007).

Further progress in the pharmacological treatment of schizophrenia will undoubtedly produce new drugs that have varying mechanisms of neurochemical action. The rate of progress in this field is very rapid. You can obtain regularly updated reviews of evidence regarding the treatment of schizophrenia from the Cochrane Library at its website: www.cochrane.org.

PSYCHOSOCIAL TREATMENT

Several forms of psychological treatment have proved to be effective for schizophrenic patients. These procedures address a wide range of problems that are associated with the disorder. Psychological treatments have usually concentrated on long-term strategies rather than the resolution of acute psychotic episodes (Kopelowicz, Liberman, & Zarate, 2002). More recently, several investigators have begun to explore the use of psychosocial interventions in combination with antipsychotic medication for first-episode patients (Grawe et al., 2006; Penn et al., 2005).

Family-Oriented Aftercare Studies of expressed emotion have inspired the development of innovative family-based treatment programs. Family treatment programs attempt to improve the coping skills of family members, recognizing the burdens that people often endure while caring for a family member with a chronic mental disorder. Patients are maintained on antipsychotic medication on an outpatient basis throughout this process. There are several different approaches to this type of family intervention. Most include an educational component that is designed to help family members understand and accept the nature of the disorder (see Getting Help on page 358). One goal of this procedure is to eliminate unrealistic expectations for the patient, which may lead to harsh criticism. Behavioral family management also places considerable emphasis on the improvement of communication and problem-solving skills, which may enhance the family members' ability to work together and thereby minimize conflict.

Several empirical studies have evaluated the effects of family interventions. Most have found reductions in relapse rates for people receiving family treatment (Barrowclough & Lobban, 2008; Girón et al., 2010). Family-based treatment

Patients and families respond in many creative ways to the presence of mental disorder. Brandon Staglin (left) has struggled with schizophrenia for several years. His parents founded an annual Music Festival for Mental Health, which has raised millions of dollars for mental health charities and research.

programs can delay relapse, but they do not necessarily prevent relapse in the long run. In the case of a disorder such as schizophrenia, which is often chronic, difficult decisions have to be made about priorities and the availability of services. Family-based programs can have a positive effect, but we need to find more efficient and more effective ways to integrate this aspect of treatment into an overall treatment program.

Social Skills Training Many patients who avoid relapse and are able to remain in the community continue to be impaired in terms of residual symptoms. They also experience problems in social and occupational functioning. For these patients, drug therapy must be supplemented by psychosocial programs that address residual aspects of the disorder. The need to address these problems directly is supported by evidence that shows that deficits in social skills are relatively stable in schizophrenic patients and relatively independent of other aspects of the disorder, including both positive and negative symptoms.

Social skills training (SST) is a structured, educational approach to these problems that involves modeling, role playing, and the provision of social reinforcement for appropriate behaviors (Heinssen, Liberman, & Kopelwicz, 2000). A general description of this type of approach to treatment is provided in Chapter 3. Controlled-outcome studies indicate that, in combination with neuroleptic medication, SST leads to improved performance on measures of social adjustment. It is not clear, however, that SST has any beneficial effects on relapse rates (Pilling et al., 2002b). That result may not be surprising in light of evidence regarding the course of this disorder, which suggests that various aspects of outcome, including symptom severity and social adjustment, tend to be relatively independent.

Cognitive Therapy One area of treatment that has received much greater emphasis in recent years is the use of various forms of cognitive therapy for schizophrenia (Rathod & Turkington, 2005; Temple & Ho, 2005). In some cases, these interventions have focused on the use of standard cognitive therapy procedures that are designed to help patients evaluate, test, and correct distorted ways of thinking about themselves and their social environments. Other forms of cognitive treatment have become more specialized and are aimed specifically at cognitive deficits that are particularly evident in schizophrenia.

One example of a specialized treatment program is cognitive enhancement therapy (CET) for schizophrenia (Hogarty et al., 2004). This is a comprehensive, integrated program aimed at the improvement of cognitive abilities, including those that are concerned with performance on laboratory tasks (such as attention, working memory, and problem solving) as well as social cognition (such as recognizing the perspectives of other people and appraising social contexts). It is designed for use with people who are also taking antipsychotic medication and have already recovered from active symptoms of psychosis but nevertheless continue to exhibit signs of cognitive disability. Patients spend many hours practicing computerized cognitive exercises. Several weeks after beginning cognitive training exercises, they also participate in an extended series of small group exercises (interpreting verbal messages, recognizing others' emotions, maintaining conversations, and so on). One large-scale two-year outcome study compared patients who received cognitive enhancement therapy with patients in a control group who received enhanced supportive therapy. Those who received CET showed more improvement with regard to performance on measures of cognitive performance, social cognition, overall social adjustment, and employment (Eack et al., 2011). Thus, in the context of ongoing treatment with antipsychotic medication, cognitive therapy can be beneficial for patients with schizophrenia.

Assertive Community Treatment The treatment of a chronic disorder such as schizophrenia clearly requires an extensive range of comprehensive services that should be fully integrated and continuously available. *Assertive community treatment* (ACT) is a psychosocial intervention that is delivered

Many people have made remarkable achievements in spite of suffering from schizophrenia. Tom Harrell has been named jazz trumpeter of the year three times by *Downbeat Magazine.* He hears disturbing auditory hallucinations, but they disappear when he is playing music.

by an interdisciplinary team of clinicians (DeLuca, Moser, & Bond, 2008; Stein & Santos, 1998). They provide a combination of psychological treatments—including education, support, skills training, and rehabilitation—as well as medication. Services are provided on a regular basis throughout the week and during crisis periods (any time of day and any day of the week). The program represents an intensive effort to maintain seriously disordered patients in the community and to minimize the need for hospitalization. It differs from more traditional outpatient services in its assertive approach to the provision of services: Members of an ACT team go to the consumer rather than expecting the consumer to come to them.

Outcome studies indicate that ACT programs can effectively reduce the number of days that patients spend in psychiatric hospitals, while improving their level of functioning (Nordentoft et al., 2010; Thornicroft & Susser, 2001). One study found that only 18 percent of the people in the ACT group were hospitalized during the first year of treatment compared to 89 percent of the people in the control group. ACT is an intensive form of treatment that requires a well-organized and extensive network of professional services. In spite of the expense that is required to maintain this kind of program, empirical studies indicate that it is more cost-effective than traditional services provided by community mental health centers (Lehman et al., 1999). Reduction in costs of inpatient care offsets the expense of the ACT program.

What aspects of schizophrenia are addressed most directly by psychosocial treatments?

Institutional Programs Although schizophrenic persons can be treated with medication on an outpatient basis, various types of institutional care continue to be important. Most patients experience recurrent phases of active psychosis. Brief periods of hospitalization (usually two or three weeks) are often beneficial during these times.

Some patients are chronically disturbed and require long-term institutional treatment. Social learning programs, sometimes called *token economies,* can be useful for these patients (Dickerson, Tenhula, & Green-Paden, 2005). In these programs specific behavioral contingencies are put into place for all of the patients on a hospital ward. The goal is to increase the frequency of desired behaviors, such as appropriate grooming and participation in social activities, and to decrease the frequency of undesirable behaviors, such as violence or incoherent speech. Staff members monitor patients' behavior throughout the day. Each occurrence of a desired behavior is praised and reinforced by the presentation of a token, which can be exchanged for food or privileges, such as time to watch television. Inappropriate behaviors are typically ignored, but occasional punishment, such as loss of privileges, is used if necessary. Carefully structured inpatient programs, especially those that follow behavioral principles, can have important positive effects for chronic schizophrenic patients.

Getting Help

Schizophrenia can be a devastating condition for patients and their families. Fortunately, the past two decades have seen many important advances in treatment for this disorder. Perhaps no other disorder requires such an extensive array of services, ranging from medication and short-term inpatient care to long-term residential facilities and psychosocial help for family members. An extremely useful book, *Coping with Schizophrenia: A Guide for Families,* written by Kim Mueser and Susan Gingrich, offers sound advice on a variety of crucial topics. For example, the authors discuss various forms of antipsychotic drugs, their side effects, their use in preventing relapse, and ways to respond to a patient's reluctance to continue taking necessary medication. They outline available community resources that help patients and their families deal with acute episodes, as well as the long-term challenges of residual symptoms, occupational difficulties, and housing needs.

Another excellent resource is *The Family Face of Schizophrenia,* by Patricia Backlar, who is a mental health ethicist and also the mother of a son who suffers from schizophrenia. This book includes a series of seven stories about people who have struggled with this disorder and the often confusing and sometimes inadequate array of mental health services that are available in many communities. Each story is followed by a commentary that includes advice for patients and their families (e.g., how to obtain insurance benefits for treatment, how to find a missing mentally ill family member, how to cope with suicidal risks, and how to navigate legal issues that can arise in caring for someone with a serious mental disorder). Anyone who must cope with a psychotic disorder will benefit from reading these books carefully.

The National Alliance for the Mentally Ill (NAMI) is an extremely influential grassroots support and advocacy organization that has worked tirelessly to improve the quality of life for patients and their families. It has more than 1,000 state and local affiliates throughout the United States. NAMI is committed to increasing access to community-based services such as housing and rehabilitation for people with severe mental disorders. The address for its website is www.nami.org. It is a comprehensive source of information regarding all aspects of severe mental disorders (especially schizophrenia and mood disorders), including referral to various types of support groups and professional service providers.

SUMMARY

- People who meet the diagnostic criteria for **schizophrenia** exhibit symptoms that represent impairments across a broad array of cognitive, perceptual, and interpersonal functions. These symptoms can be roughly divided into three types. **Positive symptoms** include **hallucinations** and **delusions. Negative symptoms** include **blunted affect, alogia, avolition,** and social withdrawal. Symptoms of disorganization include verbal communication problems and bizarre behavior.
- The onset of schizophrenia is typically during adolescence or early adulthood. The disorder can follow different patterns over time. Some people recover fairly quickly from schizophrenia, whereas others deteriorate progressively after the initial onset of symptoms.
- The negative symptoms of schizophrenia are also important diagnostically. DSM-IV-TR requires evidence of a decline in the person's social or occupational functioning, as well as the presence of disturbed behavior over a continuous period of at least six months for a diagnosis of schizophrenia.
- The lifetime prevalence of schizophrenia is approximately 1 percent in the United States and Europe. Men are 30 to 40 percent more likely than women to be affected by the disorder, and its onset tends to occur at an earlier age in males. Male patients are more likely than female patients to exhibit negative symptoms, and they are also more likely to follow a chronic, deteriorating course.
- Genetic factors clearly play a role in the development of schizophrenia. Risk for developing the disorder is between 10 percent and 15 percent among first-degree relatives of schizophrenic patients. Concordance rates are approximately 48 percent in MZ twins compared to only 17 percent in DZ pairs. Twin and adoption studies indicate that the disorder has variable expressions, sometimes called the schizophrenia spectrum. Related disorders include schizotypal personality disorder and **schizoaffective disorder.**
- A specific brain lesion has not been identified, and it is unlikely that a disorder as complex as schizophrenia will be traced to a single site in the brain. Structural images of schizophrenic patients' brains reveal enlarged ventricles as well as decreased size of parts of the limbic system. Studies of brain metabolism and blood flow have identified functional changes in the frontal lobes, temporal lobes, and basal ganglia in many persons with schizophrenia.
- The discovery of antipsychotic medication stimulated interest in the role of neurochemical factors in the etiology of schizophrenia. The dopamine hypothesis provided the major unifying theme in this area for many years, but it is now considered too simple to account for the existing evidence. Current neurochemical hypotheses regarding schizophrenia focus on a broad array of neurotransmitters, with special emphasis on serotonin.
- Several social and psychological factors have been shown to be related to the disorder. Social class is inversely related to the prevalence of schizophrenia. People who have migrated to a new country are at greater risk for schizophrenia, suggesting the possible influence of social adversity and discrimination.
- Patients from families that are high in **expressed emotion** are more likely to relapse than those from low EE families. Expressed emotion is the product of an ongoing interaction between patients and their families, with patterns of influence flowing in both directions.
- The evidence regarding etiology supports a diathesis-stress model. It should be possible to develop **vulnerability markers** that can identify individuals who possess the genetic predisposition to the disorder. Promising research in this area is concerned with a broad range of possibilities, including laboratory measures of working memory and smooth-pursuit eye-tracking movements.
- The central aspect of treatment for schizophrenia is antipsychotic medication. These drugs help to resolve acute psychotic episodes. They can also delay relapse and improve the level of patients' functioning between episodes. Unfortunately, they often produce troublesome side effects, and a substantial minority of schizophrenic patients are resistant to antipsychotic medication.
- Various types of psychosocial treatments also provide important benefits to schizophrenic patients and their families. Prominent among these are family-based treatment for patients who have been stabilized on medication following discharge from the hospital. Social skills training can also be useful in improving the level of patients' role functioning.

The Big Picture

CRITICAL THINKING REVIEW

- **What is the difference between a delusion and most other false beliefs?**

  Delusional beliefs are more than simply mistaken ideas. They are idiosyncratic, personal in nature, patently absurd, and rigidly held in the face of contradictory evidence . . . (see p. 336)

- **What is the rationale for the distinction between the broad dimensions of positive and negative symptoms?**

  Positive symptoms represent distortions of normal functions, such as auditory perception and belief formation. Negative symptoms reflect the absence of an expected function, such as emotion . . . (see p. 337)

- **Why do clinical scientists say that schizophrenia is a "heterogeneous" disorder?**

  Schizophrenia is a disorder of many faces. It is defined by an extremely diverse set of symptoms that involve distortions of cognition, perception, and emotion. Finally, it can follow many different patterns over time . . . (see pp. 340–341)

- **How should long-term outcome be measured in schizophrenia?**

  It must be viewed from a broad spectrum. Important dimensions include the presence and severity of symptoms, the ability to function socially and occupationally, the need for housing and other social services, and the impact on other family members . . . (see pp. 342–343)

- **In what way does the long-term course of schizophrenia differ between developing and developed countries? Why?**

  Patients in developing countries often have a more favorable outcome than patients in developed countries. This difference may be influenced by more tolerant attitudes toward eccentric behaviors among family members . . . (see p. 344)

- **Why are some personality disorders considered to be schizophrenia spectrum disorders?**

  Some of the symptoms of Cluster A personality disorders represent less severe forms of psychotic symptoms. And the first-degree relatives of patients with schizophrenia are more likely than other people to qualify for a diagnosis of certain types of personality disorders . . . (see p. 346)

- **Why can't we use brain imaging to diagnose schizophrenia?**

  They are useful research tools, but they have not identified any aspects of brain structure or function that are unique to people with this disorder . . . (see pp. 347–349)

- **What characteristics would define a useful marker of vulnerability to schizophrenia?**

  It should be found more frequently among schizophrenic patients and their first-degree relatives, it should be stable over time, and it should be present prior to the onset of psychotic symptoms . . . (see pp. 353–354)

- **What aspects of schizophrenia are addressed most directly by psychosocial treatments?**

  Problems with social cognition (ways of thinking about oneself and others) and interpersonal relationships are often the targets of psychological interventions, which are designed to be used in conjunction with antipsychotic medication . . . (see pp. 356–358)

KEY TERMS

anhedonia
antipsychotic drugs
blunted affect
brief psychotic disorder
delusion
delusional disorder
disorganized speech
expressed emotion (EE)
hallucinations
negative symptoms
positive symptoms
prodromal phase
schizoaffective disorder
schizophrenia
vulnerability marker

14

Dementia, Delirium, and Amnestic Disorders

▶ In *Memento,* a young man desperately tries to solve the mystery of his wife's murder, even though a gunshot wound to the head has left him unable to store any new memories.

Most of us are absent-minded from time to time. We may forget to make a phone call, run an errand, or complete an assignment. Occasional lapses of this sort are part of normal experience. Unfortunately, some people develop severe and persistent memory problems that disrupt their everyday activities and their interactions with other people. Imagine that you have lived in the same house for many years. You go for a short walk, and then you can't remember how to get home. Suppose you are shown a photograph of your parents, and you don't recognize them. These are some of the fundamental cognitive problems discussed in this chapter.

The Big Picture

- What's the difference between being forgetful and being demented?
- Is the impact of dementia limited exclusively to memory impairment?
- How do neuropsychologists test for cognitive impairment?
- Do you have to be old to develop Alzheimer's disease?
- Can memory impairments in dementia be reversed?
- What kinds of challenges confront someone who is caring for a person with dementia?

OVERVIEW

Dementia and delirium are the most frequent disorders found among elderly psychiatric patients. Both conditions involve memory impairments, but they are quite different in other ways. **Dementia** is a gradual worsening loss of memory and related cognitive functions, including the use of language, as well as reasoning and decision making. It is a clinical syndrome that involves progressive impairment of many cognitive abilities (Waldemar & Burns, 2009). **Delirium** is a confusional state that develops over a short period of time and is often associated with agitation and hyperactivity. The most important symptoms of delirium are disorganized thinking and a reduced ability to maintain and shift attention (Gupta et al., 2008). Delirium and dementia are produced by very different processes. Dementia is a chronic, deteriorating condition that reflects the gradual loss of neurons in the brain. Delirium is usually the result of medical problems, such as infection, or of the side effects of medication. If diagnosed and properly treated, delirium is typically short-lived. It can, however, result in serious medical complications, permanent cognitive impairment, or death if the causes go untreated.

What is the difference between cognitive problems in anxiety and those seen in dementia?

People with **amnestic disorders** experience memory impairments that are more limited than those seen in dementia or delirium. The person loses the ability to learn new information or becomes unable to recall previously learned information, but other higher level cognitive abilities—including the use of language—are unaffected.

Dementia, delirium, and amnestic disorders are listed as Cognitive Disorders in DSM-IV-TR. Cognitive processes, including perception and attention, are related to many types of mental disorders that we have already discussed, such as depression, anxiety, and schizophrenia. In most forms of psychopathology, however, the cognitive problems are relatively subtle—mediating factors that help us understand the process by which clinical symptoms are produced. In the case of depression, for example, self-defeating biases may contribute to the onset of a depressed mood. These cognitive schemas are not used, however, as part of the diagnostic criteria for major depression in DSM-IV-TR. They are not considered to be the central, defining features of the disorder. Problems in working memory may represent vulnerability markers for schizophrenia, but again, they are not considered symptoms of the disorder. In dementia, memory and other cognitive functions are the most obvious manifestations of the problem. They are its defining features. As dementia progresses, the person's attention span, concentration, judgment, planning, and decision making become severely disturbed.

Dementia and amnestic disorders are often associated with specific identifiable changes in brain tissue. Many times these changes can be observed only at autopsy, after the patient's death. For example, in Alzheimer's disease, which is one form of dementia, microscopic examination of the brain reveals the presence of an unusual amount of debris left from dead neurons, called *plaque,* and neurofibrillary tangles indicating that the connections between nerve cells had become disorganized. We describe the neuropathology of Alzheimer's disease later in this chapter.

Because of the close link between cognitive disorders and brain disease, patients with these problems are often diagnosed and treated by **neurologists,** physicians who deal primarily with diseases of the brain and the nervous system. Multidisciplinary clinical teams study and provide care for people with dementia and amnestic disorders. Direct care to patients and their families is usually provided by nurses and social workers.

Confusion and disorientation are common symptoms of dementia. This elderly woman may not have been aware that she was walking in front of a line of riot police sent to control demonstrators in Moscow.

Neuropsychologists have particular expertise in the assessment of specific types of cognitive impairments. This is true for clinical assessments as well as for more detailed laboratory studies for research purposes.

The following two case studies illustrate the variety of symptoms and problems that are included in the general category of dementia. The first case describes the early stages of dementia.

CASE STUDY A Physician's Developing Dementia

Jonathan was a 68-year-old physician who had been practicing family medicine for the past 35 years. His wife, Alice, worked as his office manager. A registered nurse, Kathryn, had worked with them for several years. Four months earlier, Alice and Kathryn both noticed that Jonathan was beginning to make obvious errors at work. On one occasion, Kathryn observed that Jonathan had prescribed the wrong medication for a patient's condition. At about the same time, Alice became concerned when she asked Jonathan about a patient whom he had seen the day before. Much to her surprise, he did not remember having seen the patient, in spite of the fact that he spent almost half an hour with her, and she was a patient whom he had treated for several years. Jonathan's personality also seemed to change in small but noticeable ways. He seemed uncharacteristically apathetic about daily activities that he and Alice typically enjoyed together. She also found that he had become increasingly self-centered.

Although Alice tried to convince herself that these were isolated incidents, she finally decided to discuss them with Kathryn. Kathryn agreed that Jonathan's memory was failing. He had trouble recognizing patients whom he had known for many years, and he had unusual difficulty making treatment decisions. These problems had not appeared suddenly. Over the past year or two, both women had been doing more things for Jonathan than they had ever done in the past. They needed to remind him about things that were routine parts of his practice. As they pieced together various incidents, the pattern of gradual cognitive decline became obvious.

Alice talked seriously with Jonathan about the problems that she and Kathryn had observed. He said that he felt fine, but he reluctantly allowed her to make an appointment for him to be examined by a neurologist, who also happened to be a friend. Jonathan admitted to the neurologist that he had been having difficulty remembering things. He believed that he had been able to avoid most problems, however, by writing notes to himself—directions, procedures, and so on. The results of psychological testing and brain imaging procedures, coupled with Jonathan's own description of his experiences and Alice's account of his impaired performance at work, led the neurologist to conclude that Jonathan was exhibiting early signs of dementia, perhaps Alzheimer's disease. He spoke directly with Jonathan regarding his diagnosis and recommended firmly that Jonathan retire immediately. A malpractice suit would be devastating to his medical practice. Jonathan agreed to retire.

Although Jonathan was no longer able to cope with his demanding work environment, his adjustment at home was not severely impaired. The changes in his behavior remained relatively subtle for many months. In short conversations, his cognitive problems were not apparent to his friends, who still did not know the real reason for his retirement. His speech was fluent, and his memory for recent events was largely intact, but his comprehension was diminished. Alice noticed that Jonathan's emotional responses were occasionally flat or restricted. At other times, he would laugh at inappropriate times when they watched television programs together. If Alice asked him about his reaction, it was sometimes apparent that Jonathan did not understand the plot of even the simplest television programs.

Alice found that she had to sew labels into Jonathan's collars to distinguish for him the clothes that he wore to work in the yard from those that he wore if they were going shopping or out to eat. Jonathan had become increasingly literal minded. If Alice asked him to do something for her, she had to spell out every last detail. For example, he began to have trouble selecting his clothes, which had been a source of pride before the onset of his cognitive problems. Alice found that she had to sew labels into Jonathan's collars to distinguish for him the clothes that he wore to work in the yard from those that he wore if they were going shopping or out to eat. His judgment about what was appropriate to wear in different situations had disappeared altogether.

Alice found that she had to sew labels into Jonathan's collars to distinguish for him the clothes that he wore to work in the yard from those that he wore if they were going shopping or out to eat.

It had also become difficult for Jonathan to do things that required a regular sequence of actions or decisions, even if they were quite simple and familiar. Routine tasks took longer than before, usually because he got stuck part of the way through an activity. He had, for example, always enjoyed making breakfast for Alice on weekends. After his retirement, Alice once found him standing in the kitchen with a blank expression on his face. He had made a pot of coffee and some toast for both of them, but he ran into trouble when he couldn't find coffee cups. That disrupted his plan, and he was stymied.

Jonathan's case illustrates many of the early symptoms of dementia, as well as the ways in which the beginnings of memory problems can severely disrupt a person's life. The onset of the disorder is often difficult to identify precisely because forgetfulness increases gradually. Problems are most evident in challenging situations, as in Jonathan's medical practice, and least noticeable in familiar surroundings.

Changes in emotional responsiveness and personality typically accompany the onset of memory impairment in dementia. In some cases, personality changes may be evident before the development of full-blown cognitive symptoms (Duchek et al., 2007). These personality changes may be consequences of cognitive impairment. Jonathan's emotional responses may have seemed unusual sometimes because he failed to comprehend

aspects of the environment that were obvious to his wife and other people.

Our next case illustrates more advanced stages of dementia, in which the person can become extremely disorganized. Memory impairment progresses to the point where the person no longer recognizes his or her family and closest friends. People in this condition are unable to care for themselves, and they become so disoriented that the burden on others is frequently overwhelming. This case also provides an example of delirium superimposed on dementia. Up to 50 percent of dementia patients who are admitted to a hospital are also delirious. It is important for the neurologist to recognize the distinction between these conditions because the cause of the delirium (which might be an infection or a change in the patient's medications) must be treated promptly (Young et al., 2008).

CASE STUDY Dementia and Delirium—A Niece's Terrible Discoveries

Mary was an 84-year-old retired schoolteacher who had grown up in the same small rural community in which she still lived. Never married, she lived with her parents most of her life, except for the years when she was in college. Her parents had died when Mary was in her early sixties. After her retirement at age 65, Mary continued living in her parents' farmhouse. She felt comfortable there, in spite of its relative isolation, and liked the fact that it had plenty of space for animals, including her dog, which she called "my baby," several cats, and a few cows that were kept in the pasture behind the house. Mary's niece, Nancy, who was 45 years old and lived an hour's drive away, stopped to visit her once every two or three months.

Over the past year, Nancy had noticed that Mary was becoming forgetful, as well as more insistent that her routines remain unchanged. Bills went unpaid—in fact, the telephone had been disconnected for lack of payment—and the mail wasn't brought in from the roadside box. Nancy had suggested to Mary that she might be better off in a nursing home, but Mary was opposed to that idea.

At her most recent visit, Nancy was shocked to find that conditions at Mary's home had become intolerable. Most distressing was the fact that some of her animals had died because Mary forgot to feed them. The dog's decomposed body was tied to its house, where it had starved. Conditions inside the house were disgusting. Almost 30 cats lived inside the house, and the smell was unbearable. Mary's own appearance was quite disheveled. She hadn't bathed or changed her clothes for weeks. Nancy contacted people at a social service agency, who arranged for Mary's admission to a nursing home. Mary became furious, refusing to go and denying that there was anything wrong with her own home. Nancy was soon declared her legal guardian because Mary was clearly not competent to make decisions for herself.

Mary grew progressively more agitated and belligerent during the few weeks that she lived at the nursing home. She was occasionally disoriented, not knowing where she was or what day it was. She shouted and sometimes struck people with her cane. She had trouble walking, a problem that was compounded by visual and spatial judgment difficulties. After she fell and broke her hip, Mary was transferred to a general hospital.

Bills went unpaid—in fact, the telephone had been disconnected for lack of payment—and the mail wasn't brought in from the roadside box.

Mary became delirious in the hospital, apparently as a result of medication she was given for her injury. She appeared to be having visual hallucinations and often said things that did not make sense. These periods of incoherence fluctuated in severity throughout the course of the day. During her worse moments, Mary did not respond to her name being spoken, and her speech was reduced primarily to groans and nonsense words. This clouding of consciousness cleared up a few days after her medication was changed. She became less distractible and was once again able to carry on brief conversations. Unfortunately, her disorientation became more severe while she was immobilized in the hospital. When her hip eventually healed, she was moved to a psychiatric hospital and admitted to the geriatric ward.

Although Mary was no longer aware of the date or even the season of the year, she insisted that she did not have any problems with her mind. For the first six weeks at the psychiatric hospital, she would be surprised that she was not in her own home when she woke up each morning. After that time, she acknowledged that she was in a hospital, but she did not know why she was there, and she did not understand that the other patients on the unit were also demented. She didn't recognize hospital staff members from one day to the next. She was completely unable to remember anything that had happened recently. Nevertheless, her memory for events that had happened many years earlier was quite good. Mary repeated stories about her childhood over and over again.

Nurses on the unit were bombarded continuously with her complaints about being removed from her home. Every 20 minutes or so, Mary would approach the nurses' station, waving her cane and shouting, "Nurse, I need to go home. I have to get out of here. I have to go home and take care of my dog." The hospital staff would explain to her that she would have to stay at the hospital, at least for a while longer, and that her dog had died several months earlier. This news would usually provoke sadness, but she seemed unable to remember it long enough to complete the grieving process. Several minutes later, the whole scene would be repeated. Mary also became paranoid, claiming to anyone who would listen that people were trying to steal her things. The most common focus of her concern was her purse. If it was out of her sight, she would announce loudly that someone had stolen it.

In the midst of these obvious problems, Mary retained many other intellectual abilities. She was a well-educated and intelligent woman. Her attention span was reduced, but she was still able to play the piano—pieces that she had practiced over and over again for many years. Poetry had always been one of her special interests, and she was still able to recite some of her favorite poems beautifully from memory. In a quiet room, it was often possible to talk with her and pursue a meaningful conversation. Unfortunately, these lucid periods were interspersed with times of restless pacing and shouting. Her agitation would escalate rapidly unless staff members distracted her, taking her to a quiet room, talking to her, and getting her to read or recite something out loud.

TABLE 14.1 DSM-IV-TR Criteria for Delirium

A. Disturbance of consciousness (i.e., reduced clarity of awareness of the environment) with reduced ability to focus, sustain, or shift attention.

B. A change in cognition (such as memory deficit, disorientation, language disturbance) or the development of a perceptual disturbance that is not better accounted for by a preexisting, established, or evolving dementia.

C. The disturbance develops over a short period of time (usually hours to days) and tends to fluctuate during the course of the day.

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

Symptoms

The symptoms of cognitive disorders are often overlooked in elderly patients. It can be difficult to distinguish the onset of dementia from patterns of modest memory decline that are an expected part of the aging process. Different forms of cognitive disorder can also be confused with one another. Recognition of these disorders and the distinctions among them carries important treatment implications for patients and their families.

DELIRIUM

The DSM-IV-TR criteria for delirium are listed in Table 14.1. The primary symptom of delirium is clouding of consciousness in association with a reduced ability to maintain and shift attention. The disturbance in consciousness might also be described as a reduction in the clarity of a person's awareness of his or her surroundings. Memory deficits may occur in association with impaired consciousness and may be the direct result of attention problems. The person's thinking appears disorganized, and he or she may speak in a rambling, incoherent fashion. Fleeting perceptual disturbances, including visual hallucinations, are also common in delirious patients (Gupta et al., 2008).

The symptoms of delirium follow a rapid onset—from a few hours to several days—and typically fluctuate throughout the day. The person may alternate between extreme confusion and periods in which he or she is more rational and clearheaded. Symptoms are usually worse at night. The sleep/wake cycle is often disturbed. Daytime drowsiness and lapses in concentration are often followed by agitation and hyperactivity at night. If the condition is allowed to progress, the person's senses may become dulled, and he or she may eventually lapse into a coma. The delirious person is also likely to be disoriented with relation to time ("What day, month, or season is it?") or place ("Where are we? What is the name of this place?"). However, identity confusion ("What is your name?") is rare.

It isn't always easy to recognize the difference between dementia and delirium, especially when they appear simultaneously in the same patient. Table 14.2 summarizes several considerations that are useful in making this diagnostic distinction (Insel & Badger, 2002). One important consideration involves the period of time over which the symptoms appear. Delirium has a rapid onset, whereas dementia develops in a slow, progressive manner. In dementia, the person usually remains alert and responsive to the environment. Speech is most often coherent in demented patients, at least until the end stages of the disorder, but it is typically confused in delirious patients. Finally, delirium can be resolved, whereas dementia cannot.

In what ways is delirium different from dementia?

DEMENTIA

The cases at the beginning of this chapter illustrate the changing patterns that emerge as dementia unfolds. Jonathan's cognitive symptoms were recognized at a relatively early stage of development, in part because of his occupational situation and because of his close relationships with other people. Mary's situation was much different, because she lived in a relatively isolated setting without close neighbors or friends. By the time Nancy recognized the full severity of Mary's problems, the cognitive impairment had progressed so far that Mary was no longer able to appreciate the nature of her own difficulties. In the following pages we describe in more detail the types of symptoms that are associated with dementia.

TABLE 14.2 Distinguishing Features of Dementia and Delirium

Characteristic	Delirium	Dementia
Onset	Sudden (hours to days)	Slow (months to years)
Duration	Brief	Long/lifetime
Course	Fluctuating	Stable, with downward trajectory over time
Hallucinations	Visual/tactile/vivid	Rare
Insight	Lucid intervals	Consistently poor
Sleep	Disturbed	Less disturbed

Source: From "Deciphering the 4Ds: Cognitive Decline, Delirium, Depression, and Dementia A Review" by K.C. Insel and T.A. Badger, *Journal of Advanced Nursing,* 38, (2002), pp. 360 368. Copyright © 2002 John Wiley and Sons. Reprinted by permission.

Cognitive Symptoms Dementia appears in people whose intellectual abilities have previously been unimpaired. Both of the people in our case studies were bright, well educated, and occupationally successful before the onset of their symptoms. The earliest signs of dementia are often quite vague. They include difficulty remembering recent events and the names of people and familiar objects. These are all problems that are associated with normal aging, but they differ from that process in order of magnitude (see Memory Changes in Normal Aging below). The distinguishing features of dementia include cognitive problems in a number of areas, ranging from impaired memory and learning to deficits in language and abstract thinking. By the final stages of dementia, intellectual and motor functions may disappear almost completely.

MEMORY CHANGES IN NORMAL AGING

Changes in cognitive abilities are part of the normal aging process. Most elderly adults complain more frequently about memory problems than younger adults do, and they typically perform slower and less efficiently than younger adults on laboratory tests of memory. There are, of course, individual differences in the age at which cognitive abilities begin to decline, as well as in the rate at which these losses take place. Nevertheless, some types of memory impairment are an inevitable consequence of aging (Nilsson, 2003).

In order to understand more clearly the cognitive changes associated with aging, it is useful to distinguish between two general aspects of mental functioning: fluid intelligence and wisdom (Baltes, 1993; Salthouse, 1999). The computer can be used as a metaphor to explain this distinction. Fluid intelligence refers to "the hardware of the mind." These functions are concerned with the speed and accuracy of such basic processes as perception, attention, and working memory. The proficiency of fluid intelligence depends on neurophysiological processes and on the structural integrity of the person's brain.

Wisdom, on the other hand, represents the "culture-based software of the mind." Reading and writing skills, as well as knowledge about the self and ways of coping with environmental challenges, are examples of cognitive abilities that might be included under the general heading of wisdom. These aspects of intelligence represent information about the world that is acquired continually throughout the person's lifetime (Baltes & Smith, 2008).

Fluid intelligence and wisdom follow different trajectories over the normal human life span (Kunzmann & Baltes, 2003). Fluid intelligence develops continuously during childhood and adolescence, reaching a point of optimal efficiency during young adulthood. After that point, it follows a gradual pattern of decline (Bugg et al., 2006). Wisdom also increases throughout adolescence and young adulthood, but it does not become increasingly impaired as the person ages. In fact, it often expands. The erosion of fluid intelligence over time is presumably due to subtle atrophy of brain regions, such as the hippocampus, that take place during normal aging (Head et al., 2008).

The aging mind apparently depends on the coordination of gains and losses. The elderly person strikes a balance through a process that involves selection, optimization, and compensation (Freund & Baltes, 2002). Arthur Rubinstein, the brilliant pianist who performed concerts well into his eighties, provides an example of this process.

Does a decline in working memory capacity and speed of information processing always signal the onset of dementia?

Rubinstein described three strategies that he employed in his old age: (1) He was selective, performing fewer pieces; (2) he optimized his performance by practicing each piece more frequently; and (3) he compensated for a loss of motor speed by utilizing pieces that emphasized contrast between fast and slow segments so that his playing seemed faster than it really was. Successful aging is based on this dynamic process. The person compensates for losses in fluid intelligence by taking advantage of increased knowledge and information.

The fact that an older person begins to experience a reduction in memory capacity and speed of information processing does not necessarily indicate that he or she is becoming demented. Where can we find the line between normal aging and dementia? Is this distinction simply a matter of degree, or is there a qualitative difference between the expected decline in cognitive mechanics and the onset of cognitive pathology? These issues present an important challenge for future research.

Nelson Mandela won the Nobel Peace Prize at age 75. His wisdom and courage provide a remarkable example of successful aging.

Memory and Learning The diagnostic hallmark of dementia is memory loss. In order to describe the various facets of memory impairment, it is useful to distinguish between old memories and the ability to learn new things. **Retrograde amnesia** refers to the loss of memory for events prior to the onset of an illness or the experience of a traumatic event. **Anterograde amnesia** refers to the inability to learn or remember new material after a particular point in time.

Anterograde amnesia is usually the most obvious problem during the beginning stages of dementia. Consider, for example, the case of Jonathan. Alice eventually noticed that he sometimes could not remember things that he had done the previous day. Mary, the more severely impaired person, could not remember for more than a few minutes that her dog had died. Long-term memories are usually not affected until much later in the course of the disorder. Even in advanced stages of dementia, a person may retain some recollections of the past. Mary was able to remember, and frequently described, stories from her childhood.

Verbal Communication Language functions can also be affected in dementia. **Aphasia** is a term that describes various types of loss or impairment in language that are caused by brain damage (Mesulam, 2007). Language disturbance in dementia is sometimes relatively subtle, but it can include many different kinds of problems. Patients often remain verbally fluent, at least until the disorder is relatively advanced. They retain their vocabulary skills and are able to construct grammatical sentences. They may have trouble finding words, naming objects, and comprehending instructions.

In addition to problems in understanding and forming meaningful sentences, the demented person may also have difficulty performing purposeful movements in response to verbal commands, a problem known as **apraxia.** The person possesses the normal strength and coordination to carry out the action and is able to understand the other person's speech but is nevertheless unable to translate the various components into a meaningful action (Ballard, Granier, & Robin, 2008).

Some aspects of the communication problems associated with dementia are captured in the following passage from a novel by J. Bernlef entitled *Out of Mind.* This account provides an insightful and poignant description of the subjective experiences of a man from Holland, named Maarten, who is becoming demented. In this passage, Maarten is listening to his wife, Vera, as she discusses his situation with their physician.

> "Sometimes he's like a stranger to me. I can't reach him. It's a terrible, helpless feeling. He hears me but at such times I don't think he understands me. He behaves as if he were on his own."
>
> I know exactly what she means. Like it was just then, when it all went wrong. All of a sudden I had to translate everything into English first, before I could say it. Only the forms of sentences came out, fragments, the contents had completely slipped away.
>
> Furiously I glare into the front room. I seem to lose words like another person loses blood. And then suddenly I feel terribly frightened again. The presence of everything! Every object seems to be heavier and more solid than it should be (perhaps because for a fraction of a second I no longer know its name). I quickly lie down on the settee and close my eyes. A kind of seasickness in my mind, it seems. Under this life stirs another life in which all times, names and places whirl about topsy-turvy and in which I no longer exist as a person.
>
> "Curious," I say to Vera as she enters the room. "Sometimes I just have to lie down for a moment. I never used to."
>
> "It doesn't matter. Have some time to yourself." She sits down, picks up a book.
>
> "Have some time to yourself." I repeat the phrase because it appears strange to me.
>
> She turns the pages but she isn't reading. I can tell from the look in her eyes that she doesn't understand me.
>
> "It should be: have some time in yourself. That describes the situation better."
>
> "Is that how you feel?"
>
> "Less and less so."
>
> "What do you mean?"
>
> "Like a ship," I say, "A ship, a sailing vessel that is becalmed. And then suddenly there is a breeze, I am sailing again. Then the world has a hold on me again and I can move along with it."
>
> (Bernlef, 1988, pp. 54–55)

Some of the things that Maarten says in this passage reflect subtle problems in verbal communication. His description of the sailing ship, however, is a remarkable analogy that captures the intermittent quality of the cognitive impairment.

MyPsychLab VIDEO CASE

Dementia

ALVIN

"That's one of the real difficulties. There's no sign that goes off and says, Yes, he's understanding, or No, he's not understanding it."

As you watch the interview and the day-in-the-life segment, try to identify the various signs of Alvin's cognitive impairment.

Perception Some patients with dementia have problems identifying stimuli in their environments. The technical term for this phenomenon is **agnosia,** which means "perception without meaning." The person's sensory functions are unimpaired, but he or she is unable to recognize the source of stimulation (Bauer & Demery, 2003). Agnosia can be associated with visual, auditory, or tactile sensations, and it can be relatively specific or more generalized. For example, visual agnosia is the inability to recognize certain objects or faces. Some people with visual agnosia can identify inanimate stimuli but are unable to recognize human faces.

It is sometimes difficult to distinguish between aphasia and agnosia. Imagine, for example, that a clinician shows a patient

a toothbrush and asks, "What is this object?" The patient may look at the object and be unable to name it. Does that mean that the person cannot think of the word "toothbrush"? Or does it mean that the person cannot recognize the object at all? In this case, the distinction could be made by saying to the person, "Show me what you do with this object." A person suffering from aphasia would take the toothbrush in his hand and make brushing movements in front of his mouth, thereby demonstrating that he recognizes the object but cannot remember its name. A person with agnosia would be unable to indicate how the toothbrush is used.

How could a clinician distinguish between aphasia and apraxia?

Abstract Thinking Another manifestation of cognitive impairment in dementia is loss of the ability to think in abstract ways. The person may be bound to concrete interpretations of things that other people say. It may also be difficult for the person to interpret words that have more than one meaning (for example, "pen") or to explain why two objects are alike ("Why are a basketball and a football helmet alike?" Because they are both types of sporting equipment.).

In our opening case, Jonathan became increasingly literal minded in his conversations with other people. After he retired, he had much more time to become involved in routine tasks around the home. Alice found that she had to give him very explicit instructions if she wanted him to do anything. For example, if she asked him to mow the grass, he would do exactly that—nothing more. This was unusual for Jonathan, because he had always enjoyed taking care of their lawn and took great pride in their bushes and flower gardens. Previously, "mowing the grass" would have been taken to include trimming, pulling weeds, raking leaves from under bushes, and all sorts of related details. Now Jonathan interpreted this instruction in concrete terms.

John O'Connor suffered from Alzheimer's disease for several years prior to his death in 2009. In his later years, he was unable to remember that he was married to Sandra Day O'Connor, the first woman to serve on the U.S. Supreme Court. He struck up a romance with a fellow patient after moving into an assisted living center. Justice O'Connor said that she was not jealous and simply pleased that he was comfortable.

Judgment and Social Behavior Related to deficits in abstract reasoning is the failure of social judgment and problem-solving skills. In the course of everyday life, we must acquire information from the environment, organize and process it, and then formulate and perform appropriate responses by considering these new data in the light of past experiences. The disruption of short-term memory, perceptual skills, and higher-level cognitive abilities obviously causes disruptions of judgment. Examples from Jonathan's case include problems deciding which clothes to wear for working around his home as opposed to going out in public, as well as his inability to understand the humor in some television programs. Impulsive and careless behaviors are often the product of the demented person's poor judgment. Activities such as shopping, driving, and using tools can create serious problems.

Assessment of Cognitive Impairment There are many ways to measure a person's level of cognitive impairment. One is the Mini-Mental State Examination, which is outlined in Table 14.3. We include sample items to give you an idea of the types of questions that a clinician might ask in order to elicit the cognitive problems of dementia. Some are directed at the person's orientation to time and place. Others are concerned with anterograde amnesia, such as the ability to remember the names of objects for a short period of time (item 2). Agnosia, aphasia, and apraxia are addressed by items 3 and 4, respectively. Perceptual difficulties are tapped by the last item (11).

Neuropsychological assessment can be used as a more precise index of cognitive impairment. This process involves the evaluation of performance on psychological tests to indicate whether a person has a brain disorder (Weintraub et al., 2009). Neuropsychological testing can involve a variety of tasks that are designed to measure sensorimotor, perceptual, and speech functions. For example, in one tactile performance test, the person is blindfolded and then required to fit differently shaped blocks into spaces in a form board. The time needed to perform this test reflects one specific aspect of the person's motor skills. Complete neuropsychological test batteries are rarely used for the diagnosis of dementia because they are too long and time-consuming. It is more common to use specific tasks that focus on abilities that are impaired in patients with dementia.

Some neuropsychological tasks require the person to copy simple objects or drawings. The drawings illustrated in Figure 14.1 demonstrate this process and the type of impairment typically seen in a patient during the relatively early stages of Alzheimer's disease. The patient was asked to reproduce a drawing. This was done initially while the original figure was still in sight and then repeated after it had been covered up. The performance of the patient indicates two problems associated with the disorder. First, inconsistencies between drawings 1 and 2 reflect perceptual difficulties. Second, the drastic deterioration from drawing 2 to drawing 3 indicates that the patient had a great deal of difficulty remembering the shape of the figure for even a few brief moments.

Personality and Emotion Personality changes, emotional difficulties, and motivational problems are frequently

TABLE 14.3 Sample Items from the Mini-Mental State Examination

Orientation to Time
What is the date?"
Registration
"Listen carefully. I am going to say three words. You say them back after I stop. APPLE (pause), PENNY (pause), TABLE (pause). Now repeat those words back to me." [Repeat up to 5 times, but score only the first trial.]
Naming
"What is this?" [Point to a pencil or pen.]
Reading
"Please read this and do what it says." [Show examinee the words on the stimulus form.] CLOSE YOUR EYES

Source: "Reproduced by special permission of the Publisher, Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549, from the Mini Mental State Examination, by Marshal Folstein and Susan Folstein, Copyright 1975, 1998, 2001 by Mini Mental LLC, Inc. Published 2001 by Psychological Assessment Resources, Inc. Further reproduction is prohibited without permission of PAR, Inc. The MMSE can be purchased from PAR, Inc. by calling (813) 968-3003."

associated with dementia. These problems may not contribute to the diagnosis of the disorder, but they do have an impact on the person's adjustment. They can also create additional burdens for people who care for demented patients.

Hallucinations and delusions are seen in at least 20 percent of dementia cases and are more common during the later stages of the disorder (Savva et al., 2009). The delusional beliefs are typically understandable consequences of the person's disorientation or anterograde amnesia. They are most often simple in nature and are relatively short-lived. Mary's frequent insistence that someone had stolen her purse is a typical example. Other common themes are phantom houseguests and personal persecution (Mizrahi et al., 2006).

The emotional consequences of dementia are quite varied. Some demented patients appear to be apathetic or emotionally flat. Their faces are less expressive, and they appear to be indifferent to their surroundings. Alice noticed, for example, that something seemed a bit vacant in Jonathan's eyes. At other times, emotional reactions may become exaggerated and less predictable. The person may become fearful or angry in situations that would not have aroused strong emotion in the past. Changes like this often lead others to believe that the person's personality has changed.

Depression is another problem that is frequently found in association with dementia (Stroud, Steiner, & Iwuagwu, 2008). In many ways, feelings of depression are understandable. The realization that your most crucial cognitive abilities are beginning to fail, that you can no longer perform simple tasks or care for yourself, would obviously lead to sadness and depression. Mary's case illustrates one way in which cognitive impairment can complicate depression: Her inability to remember from one day to the next that her dog had died seemed to interfere with her ability to grieve for the loss of her pet. Each time that she was reminded of his death was like the first time that she had heard the news.

Motor Behaviors Demented persons may become agitated, pacing restlessly or wandering away from familiar surroundings. In the later stages of the disorder, patients may develop problems in the control of the muscles by the central nervous system. Some patients develop muscular rigidity, which can be accompanied by painful cramping. Others experience epileptic seizures, which consist of involuntary, rapidly alternating movements of the arms and legs.

Some specific types of dementia are associated with involuntary movements, or dyskinesia— tics, tremors, and jerky

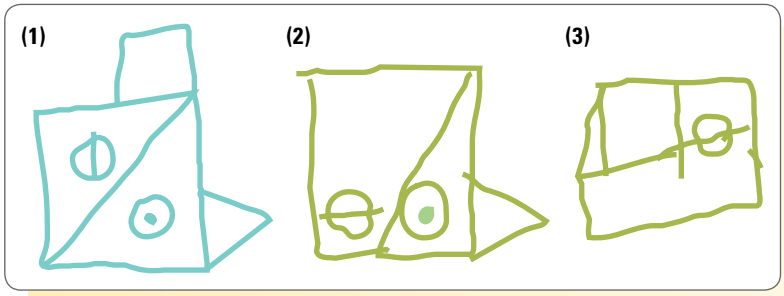


FIGURE 14.1 Neuropsychological Test Performance
These drawings represent part of the neuropsychological test performance of a 75-year-old woman with a diagnosis of Alzheimer's disease. The figure at the left (1) was drawn by the psychologist, who then handed the piece of paper to the patient and asked her to make an exact copy of the figure next to the original. After the patient had completed her replica (2), the piece of paper was turned over and she was asked to draw the figure again, this time from memory. The figure that she drew based on memory is presented at the right (3).

movements of the face and limbs called chorea. These motor symptoms help to distinguish among different types of dementia. We return to this area later in the chapter when we discuss the classification of differentiated and undifferentiated dementias.

AMNESTIC DISORDER

Some cognitive disorders involve more circumscribed forms of memory impairment than those seen in dementia. In amnestic disorders, a person exhibits a severe impairment of memory while other higher level cognitive abilities are unaffected. The memory disturbance interferes with social and occupational functioning and represents a significant decline from a previous level of adjustment. Subtypes of amnestic disorder are diagnosed on the basis of evidence, acquired from the patient's history, from a physical examination, or from laboratory tests, regarding medical conditions or substance use that is considered to be related to the onset of the memory impairment.

The following case, written by Oliver Sacks (1985), illustrates a form of amnestic disorder, involving severe anterograde amnesia, that developed after the patient had been dependent on alcohol for several years.

CASE STUDY Alcohol-Induced Persisting Amnestic Disorder—19 Going on 45

Jimmie G. was admitted to our Home for the Aged near New York City early in 1975, with a cryptic transfer note saying, "Helpless, demented, confused and disoriented." Jimmie was a fine-looking man, with a curly bush of grey hair, a healthy and handsome 49-year-old. He was cheerful, friendly, and warm.

"Hiya, Doc!" he said. "Nice morning! Do I take this chair here?" He was a genial soul, very ready to talk and to answer any questions I asked him. He told me his name and birth date, and the name of the little town in Connecticut where he was born. He described it in affectionate detail, even drew me a map. He spoke of the houses where his family had lived—he remembered their phone numbers still. He spoke of school and school days, the friends he'd had, and his special fondness for mathematics and science. He talked with enthusiasm of his days in the navy—he was 17, had just graduated from high school when he was drafted in 1943. With his good engineering mind he was a "natural" for radio and electronics, and after a crash course in Texas found himself assistant radio operator on a submarine. He remembered the names of various submarines on which he had served, their missions, where they were stationed, the names of his shipmates. He remembered Morse code and was still fluent in Morse tapping and touch-typing.

A full and interesting early life, remembered vividly, in detail, with affection. But there, for some reason, his reminiscences stopped. He recalled, and almost relived, his war days and service, the end of the war, and his thoughts for the future. He had come to love the navy, thought he might stay in it. But with the GI Bill, and support, he felt he might do best to go to college

With recalling, reliving, Jimmie was full of animation; he did not seem to be speaking of the past but of the present, and I was very struck by the change of tense in his recollections as he passed from his school days to his days in the navy. He had been using the past tense, but now used the present and (it seemed to me) not just the formal or fictitious present tense of recall, but the actual present tense of immediate experience.

A sudden, improbable suspicion seized me. "What year is this, Mr. G.?" I asked, concealing my perplexity under a casual manner.

"Forty-five, man. What do you mean?" He went on, "We've won the war, FDR's dead, Truman's at the helm. There are great times ahead."

"And you, Jimmie, how old would you be?"

Oddly, uncertainly, he hesitated a moment, as if engaged in calculation. "Why, I guess I'm 19, Doc. I'll be 20 next birthday."

Looking at the grey-haired man before me, I had an impulse for which I have never forgiven myself—it was, or would have been, the height of cruelty had there been any possibility of Jimmie's remembering it.

"Here," I said, and thrust a mirror toward him. "Look in the mirror and tell me what you see. Is that a 19-year-old looking out from the mirror?"

He suddenly turned ashen and gripped the sides of the chair. "Jesus Christ," he whispered. "Christ, what's going on? What's happened to me? Is this a nightmare? Am I crazy? Is this a joke?" and he became frantic, panicked.

"It's okay, Jimmie," I said soothingly. "It's just a mistake. Nothing to worry about. Hey!" I took him to the window. "Isn't this a lovely spring day. See the kids there playing baseball?" He regained his color and started to smile,and I stole away, taking the hateful mirror with me.

Two minutes later I re-entered the room. Jimmie was still standing by the window, gazing with pleasure at the kids playing baseball below. He wheeled around as I opened the door, and his face assumed a cheery expression.

"Hiya, Doc!" he said. "Nice morning! You want to talk to me—do I take this chair here?" There was no sign of recognition on his frank, open face.

"Haven't we met before, Mr. G.?" I asked casually.

"Am I a patient? Am I sick and don't know it, Doc? It's crazy, it's scary. . . . Is it some sort of joke?"

"No, I can't say we have. Quite a beard you got there. I wouldn't forget you, Doc!"

"Why do you call me 'Doc'?"

"Well, you are a doc, ain't you?"

"Yes, but if you haven't met me, how do you know what I am?"

"You talk like a doc. I can see you're a doc."

"Well, you're right, I am. I'm the neurologist here."

"Neurologist? Hey, there's something wrong with my nerves? And 'here'—where's 'here'? What is this place anyhow?"

"I was just going to ask you—where do you think you are?"

"I see these beds, and these patients everywhere. Looks like a sort of hospital to me. But hell, what would I be doing in a hospital and with all these old people, years older than me. I feel good, I'm strong as a bull. Maybe I work here. Do I work? What's my job?. . . No, you're shaking your head, I see in your eyes I don't work here. If I don't work here, I've been put here. Am I a patient, am I sick and don't know it, Doc? It's crazy, it's scary . . . Is it some sort of joke?" (Sacks, 1985, pp. 22–25).

The preceding case illustrates the most common type of amnestic disorder, alcohol-induced persisting amnestic disorder, also known as Korsakoff's syndrome. In this disorder, which is caused by chronic alcoholism, memory is impaired but other cognitive functions are not. More detailed examinations of the patient's cognitive abilities, using neuropsychological tests, have found evidence of more widespread cognitive deficits, especially those related to visuoperceptual skills and abstract thinking (Brand, 2007).

One widely accepted theory regarding this condition holds that lack of vitamin B_1 (thiamine) leads to atrophy of the medial thalamus, a subcortical structure of the brain, and mammillary bodies (MB). Support for one aspect of this theory comes from studies that used magnetic resonance imaging to compare brain structures in alcoholic patients with amnesia, alcoholic patients without amnesia, and normal controls. Deficits in MB volume occur in both types of alcoholics, and greater volume deficits are found in alcoholic patients with amnesia (Sullivan et al., 1999). Other data suggest, however, that these problems cannot be traced exclusively to thiamine deficiency (Homewood & Bond, 1999). In fact, prolonged exposure to alcohol may have direct toxic effects on cortical and subcortical tissue that are independent of vitamin deficiencies. Alcohol apparently can cause brain damage regardless of the person's nutritional habits (Crews et al., 2004).

Alois Alzheimer (left) on a pleasure cruise with his friend Emil Kraepelin. The form of dementia that Alzheimer described in his famous case was named after him in part because of the influence of Kraepelin's textbook.

DIAGNOSIS

Cognitive disorders have been classified by a somewhat different process than most other forms of psychopathology because of their close link to specific types of neuropathology. Description of specific cognitive and behavioral symptoms has not always been the primary consideration. In the following pages we describe the ways in which these disorders have been defined and some of the considerations that influence the way in which they are classified.

BRIEF HISTORICAL PERSPECTIVE

Alois Alzheimer (1864–1915), a German psychiatrist, worked closely in Munich with Emil Kraepelin, who is often considered responsible for modern psychiatric classification (see Chapters 4, 5, and 13). Alzheimer's most famous case involved a 51-year-old woman who had become delusional and also experienced a severe form of recent memory impairment, accompanied by apraxia and agnosia. This woman died four years after the onset of her dementia. Following her death, Alzheimer conducted a microscopic examination of her brain and made a startling discovery: bundles of neurofibrillary tangles and amyloid plaques. Alzheimer presented the case at a meeting of psychiatrists in 1906 and published a three-page paper in 1907. Emil Kraepelin began to refer to this condition as Alzheimer's disease in the eighth edition of his famous textbook on psychiatry, published in 1910. He distinguished between this form of dementia, which is characterized by early onset, and senile dementia, which presumably has an onset after the age of 65 (Fox, Kelly, & Tobin, 1999).

For many years, there was an argument about the distinction between senile and presenile dementia. As more and more evidence accumulated regarding these conditions, questions were raised about the value of the distinction. For example, several cases were reported in which two siblings developed dementia, but one had the presenile form and the other had the senile form. Clinical symptoms and brain pathology in the siblings were often the same. Katzman (1976) proposed that both types are forms of Alzheimer's disease, which may have either an early or a late onset, and that they are distinctly different from normal aging. Age of onset may be a reflection of the severity of the disorder. Most clinicians and researchers still believe that Alzheimer's disease is a heterogeneous category, and the genetic literature supports that contention.

In previous editions, the diagnostic manual classified the various forms of dementia as Organic Mental Disorders because of their association with known brain diseases. That concept has fallen into disfavor because it is founded on an artificial dichotomy between biological and psychological processes. If we call dementia an organic mental disorder, does that imply that other types of psychopathology are not organically based (Spitzer et al., 1992)? Obviously not. Therefore, in order to be consistent with the rest of the diagnostic manual, and so as to avoid falling into the trap of simplistic mind–body dualism, dementia and related clinical phenomena are now classified as Cognitive Disorders in DSM-IV-TR. These disorders are divided into three major headings: deliria, dementias, and amnestic disorders (see Table 14.4).

TABLE 14.4 Cognitive Disorders Listed in DSM-IV-TR

Delirium	Delirium due to a general medical condition
	Substance-induced delirium
	Delirium due to multiple etiologies
Dementia	Dementia of the Alzheimer's type
	Vascular dementia
	Dementia due to other general medical conditions
	HIV disease
	Head trauma
	Parkinson's disease
	Huntington's disease
	Pick's disease (now subsumed under Frontotemporal Dementia)
	Creutzfeldt-Jakob disease
	Substance-induced persisting dementia
	Dementia due to multiple etiologies
Amnestic disorders	Amnestic disorder due to a general medical condition
	Substance-induced persisting amnestic disorder

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

SPECIFIC DISORDERS ASSOCIATED WITH DEMENTIA

Many specific disorders are associated with dementia. They are distinguished primarily on the basis of known neuropathology—specific brain lesions that have been discovered over the past 100 years. DSM-IV-TR lists several categories of dementia. The criteria for cognitive deficits of dementia are the same for each type, and they are listed in Table 14.5 as they relate to Dementia of the Alzheimer's Type (see Critical Thinking Matters). The only part of this definition that changes from one type of dementia to the next is the description listed under "C" (gradual onset and continuing cognitive decline). In order to qualify for a diagnosis of dementia, the person must exhibit memory impairment (either anterograde or retrograde amnesia) and at least one other type of cognitive disturbance, such as aphasia, apraxia, agnosia, or problems in abstract thinking. There must also be evidence that the person's cognitive impairment interferes with his or her social or occupational functioning. Finally, for all forms of dementia, DSM-IV-TR notes that the cognitive problems must be above and beyond anything that could be attributed solely to delirium.

TABLE 14.5 DSM-IV-TR Criteria for Dementia of the Alzheimer's Type

A. The development of multiple cognitive deficits manifested by both:

1. Memory impairment (impaired ability to learn new information or to recall previously learned information)
2. One (or more) of the following cognitive disturbances:
   a. Aphasia (language disturbance)
   b. Apraxia (impaired ability to carry out motor activities despite intact motor function)
   c. Agnosia (failure to recognize or identify objects despite intact sensory function)
   d. Disturbance in executive functioning (that is, planning, organizing, sequencing, abstracting)

B. The cognitive deficits each cause significant impairment in social or occupational functioning and represent a significant decline from a previous level of functioning.

C. The course is characterized by gradual onset and continuing cognitive decline.

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

Critical Thinking Matters

HOW CAN CLINICIANS ESTABLISH AN *EARLY* DIAGNOSIS OF ALZHEIMER'S DISEASE?

The DSM-IV-TR diagnostic criteria for Dementia of the Alzheimer's Type (DAT) represent an interesting example of a point that we have tried to make throughout this book. In the field of psychopathology, diagnostic criteria usually represent a "work in progress." Do not take any of these definitions as being the final word with regard to the identification of a disorder. Prevailing views about the best way to identify mental disorders will continue to evolve as more evidence is collected and evaluated.

One of the most important problems with regard to the diagnosis of DAT involves the initial identification of the disorder. Symptoms associated with advanced stages of the disorder are obvious. But what are the *earliest* reliable indications that a person has developed the disorder? Are these early signs the same as (although perhaps more subtle than) the symptoms that are present when the disorder has progressed for several years? If the disorder could be identified in its beginning stages, it might be possible to develop more effective treatment procedures.

Do people in the early stages of DAT show changes specifically in memory performance, or does the disorder have a more generalized impact on many different aspects of cognition, such as reasoning and planning, attention, perception, and use of language? In an effort to answer this question, research studies have been conducted to investigate "mild cognitive impairment" in elderly persons (Kawas, 2003). Investigators have tested people who meet various definitions for this condition. The participants are later followed up and retested, in an effort to determine whether specific kinds of problems do, in fact, indicate that the person will go on to develop a more disabling form of dementia.

Is memory impairment the only indication that a person is developing dementia?

The most useful definition of mild cognitive impairment seems to be one that includes evidence of a decline in any area of cognitive performance, not simply memory (Johnson et al., 2009). For example, people who show a decline in *executive functioning* (reasoning and planning) are just as likely to develop DAT three or four years after initial testing. These data suggest that Alzheimer's disease does not always begin as a memory problem.

The definition of DAT that is presented in Table 14.5 may be revised when the next edition of the manual is published. Studies of the progression of mild cognitive impairment suggest that, in the earliest stages of the disorder, memory impairment may not be its only symptom. Increased emphasis may be placed on evidence regarding a decline in executive functioning (Storandt, 2008). Longitudinal studies also indicate that more obvious symptoms, such as aphasia, apraxia, and agnosia, are primarily evident during the advanced stages of the disorder. Critical thinking about this kind of evidence will lead to better refined and more useful diagnostic criteria.

Dementia of the Alzheimer's Type The speed of onset serves as the main feature to distinguish **Alzheimer's disease** from the other types of dementia listed in DSM-IV-TR. In this disorder, the cognitive impairment appears gradually, and the person's cognitive deterioration is progressive (Waldemar & Burns, 2009). If the person meets these criteria, the diagnosis is then made on the basis of excluding other conditions, such as vascular disease, Huntington's disease, Parkinson's disease, or chronic substance abuse.

A definite diagnosis of Alzheimer's disease can only be determined by autopsy because it requires the observation of two specific types of brain lesions: neurofibrillary tangles and amyloid plaques (see Figure 14.2). The brain is composed of millions of neurons. The internal structure of branches that extend from each neuron includes microtubules, which provide structural support for the cell and help transport chemicals used in the production of neurotransmitters (Caselli et al., 2006). These microtubules are reinforced by tau proteins, which are organized symmetrically. Tau proteins are proteins associated with the assembly and stability of microtubules. In patients with Alzheimer's disease, enzymes loosen tau from their connections to the microtubule, and they break apart. The microtubules disintegrate in the absence of tau proteins, and the whole neuron shrivels and dies. The disorganized tangles of tau that are left at the end of this process are known as **neurofibrillary tangles.** They are found in both the cerebral cortex and the hippocampus. Neurofibrillary tangles have also been found in adults with Down syndrome and patients with Parkinson's disease.

The other type of lesion in Alzheimer's disease is known as **amyloid plaques,** which consist of a central core of homogeneous protein material known as *beta-amyloid* surrounded by clumps of debris left over from destroyed neurons. These plaques are located primarily in the cerebral cortex. They are found in large numbers in the brains of patients with Alzheimer's disease, but they are not unique to that condition. The brains of normal elderly people, especially after the age of 75, often contain some neurofibrillary tangles and

(This item omitted from WebBook edition)

FIGURE 14.2 Forms of Brain Tissue Damage Associated with Alzheimer's Disease

The top panel illustrates the appearance of amyloid plaques and neurofibrillary tangles. The bottom panel illustrates the areas of the brain in which each type of lesion is most likely to appear.

Source: (Top) From *What You Need to Know About Alzheimer's* by Medina, John J. Copyright 1999 Reproduced with permission of New Harbinger Publications in the format Textbook and Other Book via Copyright Clearance Center. (Bottom) From J. L. Cummings, *"Alzheimer's Disease," Journal of the American Medical Association,* 287, 2335-2338. Copyright © 2002. This material can be found at: http://jama.ama-assn.org/content/287/18/2335.extract. Reprinted by permission of American Medical Association.

amyloid plaques. A few widely scattered cells of this type do not appear to interfere with normal cognitive functioning.

Brain imaging procedures offer exciting new tools for the measurement of brain lesions associated with dementia. Scientists have developed a technique to detect amyloid plaques using positron emission tomography (PET imaging) in the living brain. This procedure may eventually replace the need to wait for autopsy to verify a diagnosis of Alzheimer's disease (Hinrichs et al., 2010; Klunk, 2008). Some studies have identified nondemented people who have levels of amyloid plaque that are comparable to levels seen in demented people. When these nondemented people are followed over time, high levels of amyloid plaque predict the subsequent onset of obvious symptoms of dementia (Morris et al., 2009). It is not yet possible to create images of (and measure) neurofibrillary tangles in living brains. Nevertheless, advances in the development and validation of these brain imaging tools promise to transform both research and practice related to dementia and other severe forms of cognitive impairment.

Frontotemporal Dementia A rare form of dementia associated with circumscribed atrophy of the frontal and temporal lobes of the brain is known as *frontotemporal dementia (FTD).* This syndrome is very similar to Alzheimer's disease in terms of both behavioral symptoms and cognitive impairment. Patients with both disorders display problems in memory and language. Early personality changes that precede the onset of cognitive impairment are more common among FTD patients. Impaired reasoning and judgment are more prominent than anterograde amnesia in FTD. In comparison to Alzheimer patients, patients with FTD are also more likely to engage in impulsive sexual actions, roaming and aimless exploration, and other types of disinhibited behaviors (Mendez, Lauterbach, & Sampson, 2008).

Huntington's Disease Unusual involuntary muscle movements known as *chorea* (from the Greek word meaning "dance") represent the most distinctive feature of **Huntington's disease.** These movements are relatively subtle at first, with the person appearing to be merely restless or fidgety. As the disorder progresses, sustained muscle contractions become difficult. Movements of the face, trunk, and limbs eventually become uncontrolled, leaving the person to writhe and grimace. A large proportion of Huntington's patients also exhibit a variety of personality changes and symptoms of mental disorders, primarily depression and anxiety. Between 5 and 10 percent develop psychotic symptoms. The symptoms of mental disorder may be evident before the appearance of motor or cognitive impairment (Narding & Janzing, 2003).

The movement disorder and the cognitive deficits are produced by progressive neuronal degeneration in the basal ganglia (Ross & Tabrizi, 2011). This is a group of nuclei, including the caudate nucleus, the putamen, and the globus pallidus, that form a collaborative system of connections between the cerebral cortex and the thalamus (see Figure 14.3).

Dementia appears in all Huntington's disease patients, although the extent of the cognitive impairment and the rate of its progression vary widely. Impairments in recent memory and learning are the most obvious cognitive problems. Patients have trouble encoding new information. Higher-level cognitive functions are typically well preserved, and insight is usually intact. Unlike the pattern of dementia seen in Alzheimer's disease, patients with Huntington's do not develop aphasia, apraxia, or agnosia (Morris, 1995).

The diagnosis of Huntington's disease depends on the presence of a positive family history for the disorder. It is one of the few disorders that are transmitted in an autosomal dominant pattern with complete penetrance. In other words, the person must only inherit one gene—from either parent—to be vulnerable, and an individual who inherits the problematic gene will always develop the disorder (see Research Methods).

Vascular Dementia Many conditions other than those that attack brain tissue directly can also produce symptoms of dementia. The central agent in these problems can be either medical conditions or other types of mental disorders. Diseases that affect the heart and lungs, for example, can interfere with the circulation of oxygen to the brain. Substance abuse can also interfere with brain functions.

One cause of dementia is vascular or blood vessel disease, which affects the arteries responsible for bringing oxygen and sugar to the brain (Roman, 2002). A *stroke,* the severe interruption of blood flow to the brain, can produce various types of brain damage, depending on the size of the affected blood vessel and the area of the brain that it supplies. The area of dead tissue produced by the stroke is known as an *infarct.* The behavioral effects of a stroke are usually obvious and can be distinguished from dementia on several grounds: (1) They appear suddenly rather than gradually; (2) they affect voluntary movements of the limbs and gross speech patterns, as well as more subtle intellectual abilities; and (3) they often result in unilateral rather than bilateral impairment, such as paralysis of only one side of the body.

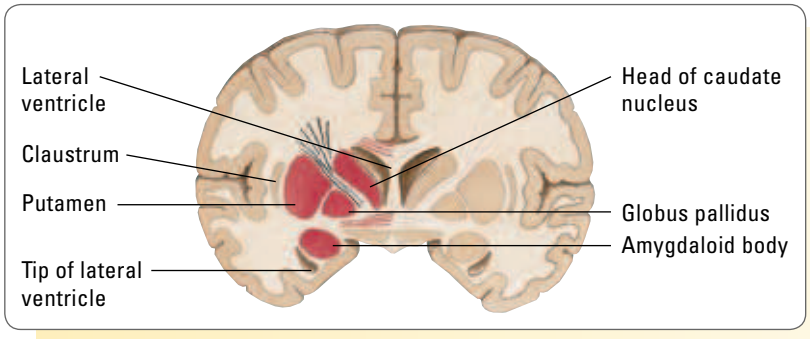


FIGURE 14.3 Areas of the Brain Implicated in Huntington's Disease

Huntington's disease involves deterioration of the basal ganglia (also known as the cerebral nuclei). The primary units of this system are the caudate nucleus, putamen, globus pallidus, and the claustrum.

Source: Adapted from R. Martini, M. Timmons, *Human Anatomy,* © 1995, p. 378. Reprinted by permission of Pearson Education, Inc., Upper Saddle River, NJ.

RESEARCH METHODS

FINDING GENES THAT CAUSE BEHAVIORAL PROBLEMS

Behavior genetic studies have demonstrated that most clinical disorders are under some degree of genetic influence. However, it is one thing to say that genetic factors "are involved" in the development of a disorder, and quite another to identify the specific genes involved. Discovery of the genes that are involved in a disorder would be an exciting step toward explaining the etiology of the disorder. It would also have important implications for developing targeted prevention and intervention programs for those people at greatest risk. Rapid advances in the field of molecular genetics are making it possible for scientists to identify specific genes involved in many disorders.

Finding genes involved in complex behavioral disorders has been difficult because there is no straightforward pattern of inheritance. Many genes are thought to be involved, and each of these genes on its own only increases or decreases risk a small amount. In addition, the environment is known to play an important role in the development of most clinical disorders. Whether an individual develops a disorder is a product of the combination of genetic and environmental risk and protective factors that the individual experiences. This has complicated efforts to identify genes involved in psychiatric disorders because the original methods developed for gene identification were based on simple, Mendelian disorders that are caused by a single defective gene. The application of these methods to complex psychiatric disorders led to many early failures and disappointments. Fortunately, new methods have been developed to take into account the complexities introduced when studying psychiatric phenotypes.

Most cells in the human body have 46 chromosomes grouped in 23 pairs. These chromosomes are transferred from the parents to the child during fertilization, with each parent providing 23 chromosomes. These chromosomes contain a chemical sequence called deoxyribonucleic acid (DNA). The characteristics of an individual that are inherited from one generation to the next are controlled by segments of DNA called genes. Any two human beings are about 99.9 percent identical genetically, but this 0.1 percent difference translates to about 3 million differences in our DNA. Some of these differences in DNA sequence contribute to individual differences in many human characteristics, ranging from eye color to personality. Most of these DNA differences are "silent" and don't appear to have any effect. These locations where the DNA comes in different forms can be used as genetic "markers," and they provide a useful way to find genes.

What do linkage and association studies tell us that twin studies cannot?

One method to find genes is a strategy called *linkage analysis*. **Genetic linkage** studies focus on families that have multiple members affected with the disorder. Investigators systematically search the entire genome by testing for linkage between genetic markers, evenly spaced across all chromosomes, and the expression of a particular disease or behavior. They are looking for stretches of DNA that are more likely to be shared among the affected individuals and less likely to be found in the unaffected individuals, suggesting that there is a gene in that region that contributes to the disorder. One strength of the linkage analysis approach is that it allows susceptibility genes to be identified when we have no, or limited, knowledge about what causes the disorder. This strategy led to the identification of the gene causing Huntington disease, which is a single-gene, Mendelian disorder (Gusella et al., 1983).

Another strategy that is used to identify genes is case-control *association analysis*. This involves identifying two groups of individuals: One group consists of people affected with the disorder (cases) and the other group consists of people who do not have the disorder (controls). The two groups should be matched on factors such as gender, ethnicity, and age, so they only differ on disease status. The frequency with which particular versions of a gene occur in the two groups is then compared. If a gene is involved in the disorder, the "risk variant" should be more frequent among the affected individuals. This approach is often used to test genes that have been targeted as good candidates for involvement in the disorder for biological reasons (for example, genes involved in serotonin reception are considered good candidates for involvement in depression because antidepressants work by altering serotonin levels) or because they lie within a region of linkage identified in family studies, as described above.

It is currently a very exciting time in gene identification efforts for complex disorders. Genes involved in the predisposition to schizophrenia (O'Donovan, Williams, & Owen, 2003), alcoholism (Dick et al., 2006), and ADHD (Faraone, 2003), among others, have all been recently reported, with replications across multiple studies. Another exciting advance is the incorporation of gene-environment interaction into the study of genetic effects. One important study found that a particular version of the serotonin transporter gene contributes to the development of depression, *but only when the individual also experiences stressful life events* (Caspi et al., 2003). Identifying the specific genes involved in clinical disorders, and how these genes interact with environmental risk factors, promises to enhance dramatically our understanding of the etiology of these disorders.

There are instances, however, in which the stroke affects only a very small artery and may not have any observable effect on the person's behavior. If several of these small strokes occur over a period of time, and if their sites are scattered in different areas of the brain, they may gradually produce cognitive impairment. DSM-IV-TR refers to this condition as **vascular dementia.** The cognitive symptoms of vascular dementia that are listed in the diagnostic manual are the same as those for Alzheimer's disease, but DSM-IV-TR does not require a gradual onset for vascular dementia, as it does for dementia of the Alzheimer's type. In addition, the diagnosis of vascular dementia depends on the presence of either focal neurological signs and symptoms associated with the experience of stroke, such as gait abnormalities or weakness in the extremities, or laboratory evidence of blood vessel disease (Paul, Garrett, & Cohen, 2003).

Parkinson's Disease A disorder of the motor system, known as *Parkinson's disease,* is caused by a degeneration of a specific area of the brain stem known as the substantia nigra and loss of the neurotransmitter dopamine, which is produced by cells in this area. Typical symptoms include tremors, rigidity, postural abnormalities, and reduction in voluntary movements (Kontakos & Stokes, 1999). Unlike people with Huntington's disease, most patients with Parkinson's disease do not become demented. Follow-up studies suggest that approximately 20 percent of elderly patients with Parkinson's disease will develop symptoms of dementia. Their risk is approximately double the risk of dementia found among people of similar age who do not have Parkinson's disease (Caviness et al., 2011).

Dementia with Lewy Bodies *Lewy bodies* (also called *intracytoplasmic inclusions*) are rounded deposits found in nerve cells. Named after F. H. Lewy, who first described them in 1912, Lewy bodies are often found in the brain-stem nuclei of patients with Parkinson's disease. Neurologists later discovered occasional cases of progressive dementia in which autopsies revealed Lewy bodies widespread throughout the brain. The development of more sensitive staining techniques that can identify cortical Lewy bodies led to greatly increased interest in this phenomenon during the 1990s.

Boxing legend Muhammed Ali and actor Michael J. Fox joke around before testifying at a government hearing on Parkinson's disease. Both men have the disorder, and they encouraged the committee to increase funds for research.

Clinicians have defined a syndrome known as **dementia with Lewy bodies (DLB),** but the boundaries of DLB are not entirely clear. It overlaps, both in terms of clinical symptoms and brain pathology, with other forms of dementia such as Alzheimer's disease and Parkinson's disease. Many experts now agree that DLB may be the second most common form of dementia, after Alzheimer's disease. Among patients who meet diagnostic criteria for Alzheimer's disease, 30 percent also have evidence of diffuse Lewy bodies in cortical neurons (Andersson et al., 2011).

Symptoms of DLB typically begin with memory deficits followed by a progressive decline to dementia (Cummings, 2004). Patients' cognitive impairment includes problems in attention, executive functions, problem solving, and visuospatial performance. Unlike patients with Alzheimer's disease, patients with DLB often show a fluctuation in cognitive performance, alertness, and level of consciousness. Their episodic confusional states sometimes resemble delirium. These changes may be evident over a period of hours or several days.

The symptom that is most likely to distinguish DLB from Alzheimer's disease and vascular dementia is the presence of recurrent and detailed visual hallucinations (Borroni et al., 2006). The patient usually recognizes that the hallucinations are not real. Many patients with DLB also develop Parkinsonian features, such as muscular rigidity, which appear early in the development of the disorder.

The course of dementia appears to be different between patients with Alzheimer's disease and DLB. Patients with DLB show a more rapid progression of cognitive impairment, and the time from onset of symptoms to death is also shorter.

Dementia versus Depression Another condition that can be associated with symptoms of dementia, especially among the elderly, is depression. There are, indeed, many areas of overlap between these disorders, but the nature of the relationship is not yet clear. Approximately 25 percent of patients with a diagnosis of dementia also exhibit symptoms of major depressive disorder (Steffens & Potter, 2008). The symptoms of depression include a lack of interest in, and withdrawal of attention from, the environment. People who are depressed often have trouble concentrating, they appear preoccupied, and their thinking is labored. These cognitive problems closely resemble some symptoms of dementia. Some depressed patients exhibit poverty of speech and restricted or unchanging facial expression. A disheveled appearance, due to self-neglect and loss of weight, in an elderly patient may contribute to the impression that the person is suffering from dementia.

Why is depression in an elderly person sometimes confused with dementia?

Despite the many similarities, there are important differences between depression and dementia. These are summarized in Table 14.6. Experienced clinicians can usually distinguish between depression and dementia by considering the pattern of onset and associated features (Insel & Badger,

TABLE 14.6 Signs and Symptoms Distinguishing Depression from Dementia

Depression	Dementia
Uneven progression over weeks	Even progression over months or years
Complains of memory loss	Attempts to hide memory loss
Often worse in morning, better as day goes on	Worse later in day or when fatigued
Aware of, exaggerates disability	Unaware or minimizes disability
May abuse alcohol or other drugs	Rarely abuses drugs

Source: "Signs and Symptoms Distinguishing Depression from Dementia" in *The Vanishing Mind: A Practical Guide to Alzheimer's Disease and Other Dementias* by Leonard L. Heston and June A. White © 1991. Reprinted by permission of Henry Holt and Company, LLC.

2002). In those cases where the distinction cannot be made on the basis of these characteristics, response to treatment may be the only way to establish a differential diagnosis. If the person's condition, including cognitive impairments, improves following treatment with antidepressant medication or electroconvulsive therapy, it seems reasonable to conclude that the person was depressed.

The relationship between depression and dementia has been the topic of considerable debate. Is depression a consequence of dementia, or are the symptoms of dementia a consequence of depression? Some clinicians have used the term *pseudodementia* to describe the condition of patients with symptoms of dementia whose cognitive impairment is actually produced by a major depressive disorder. There is no doubt that cases of this sort exist (Raskind, 1998). In fact, depression and dementia are not necessarily mutually exclusive disorders. We know that these conditions coexist more often than would be expected by chance, but we do not know why (Jorm, 2001).

Frequency of Delirium and Dementia

Cognitive disorders represent one of the most pressing health problems in our society. Detailed evidence regarding the prevalence of delirium is not available, but it does seem to be one of the most frequent symptoms of disease among elderly people. At least 15 percent of elderly hospitalized medical patients exhibit symptoms of delirium (Grover et al., 2009). The rate is much higher among nursing home patients, where delirium is often combined with dementia (as in the case study at the beginning of this chapter).

Dementia is an especially important problem among elderly people. Although it can appear in people as young as 40 to 45, the average age of onset is much later. The incidence of dementia will be much greater in the near future, because the average age of the population is increasing steadily (Kukull & Bowen, 2002; Vickland et al., 2010). People over the age of 80 represent one of the fastest growing segments of our population (see Chapter 17). By the year 2030, more than 9 million people in the United States will be affected by Alzheimer's disease. The personal and economic impact of dementia on patients, their families, and our society clearly warrants serious attention from health care professionals, policymakers concerned with health care reform, and clinical scientists seeking more effective forms of treatment.

Epidemiological studies must be interpreted with caution, of course, because of the problems associated with establishing a diagnosis of dementia. Mild cases are difficult to identify reliably. At the earliest stages of the disorder, symptoms are difficult to distinguish from forgetfulness, which can increase in normal aging. Definitive diagnoses depend on information collected over an extended period of time so that the progressive nature of the cognitive impairment, and deterioration from an earlier, higher level of functioning, can be documented. Unfortunately, this kind of information is often not available in a large-scale epidemiological study.

Also bear in mind the fact that the diagnosis of specific subtypes of dementia, such as dementia of the Alzheimer's type, requires microscopic examination of brain tissue after the person's death. Again, these data are not typically available to epidemiologists. With these limitations in mind, we now consider what is known about the frequency of dementia in the general population.

PREVALENCE OF DEMENTIA

The incidence and prevalence of dementia increase dramatically with age. Studies of community samples in North America and Europe indicate that the prevalence of dementia in people between the ages of 65 and 69 is approximately 1 percent. For people between the ages of 75 and 79, the prevalence rate is approximately 6 percent, and it increases dramatically in older age groups. Almost 40 percent of people over 90 years of age exhibit symptoms of moderate or severe dementia (Rocca et al., 2011).

Survival rates are reduced among demented patients. In Alzheimer's disease, for example, the average time between onset of the disorder and the person's death is less than six years. There is considerable variability in these figures. Some patients have survived more than 20 years after the first appearance of obvious symptoms.

There are no obvious differences between men and women with regard to the overall prevalence of dementia, broadly defined. It seems, however, that dementia in men is more likely to be associated with vascular disease or to be secondary to other medical conditions or to alcohol abuse. The incidence of Alzheimer's disease is the same in men and women up to age 90; after that, the number of new cases continues to increase for women while it apparently declines for men (Ruitenberg et al., 2001). Figure 14.4 illustrates gender differences in the number of new cases of Alzheimer's disease and vascular dementia that appear in different age groups. The incidence of vascular dementia is generally lower in women than in men at all age groups.

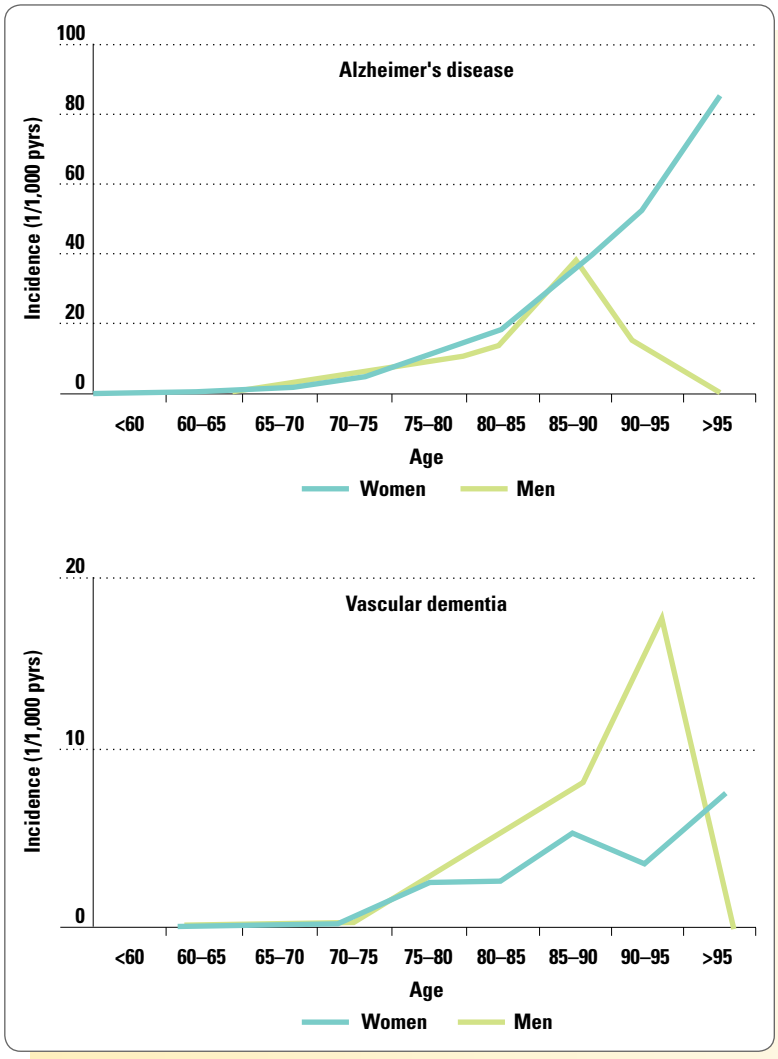


FIGURE 14.4 Gender Differences in the Incidence of Alzheimer's Disease and Vascular Dementia

This figure shows the number of new cases of dementia that appeared among people in specific age groups. The participants included 7,000 people, 55 years and older, in the Netherlands.

Source: Reprinted from *Neurobiology of Aging,* 22, A. Ruitenberg, A. Ott, J. C. van Swieten, A. Hofman, M. M. B. Breteler,"Incidence of Dementia: Does Gender Make a Difference?" Pp. 575–580, Copyright © 2001, with permission from Elsevier.

PREVALENCE BY SUBTYPES OF DEMENTIA

The studies we have already reviewed refer to cross-sectional examinations of populations, which do not allow diagnosis of specific subtypes of dementia. Some clinical studies, based on hospital populations, have allowed investigators to look at the frequency of specific subtypes of dementia. Alzheimer's disease appears to be the most common form of dementia (Waldemar & Burns, 2009), accounting for perhaps half of all cases (depending on the diagnostic criteria employed and the geographic location of the study). Dementia with Lewy bodies may be the second leading cause of dementia; studies report prevalence rates of approximately 20 percent for DLB among patients with primary dementia (Rahkonen et al., 2003). Prevalence rates for vascular dementia are similar to those for DLB (Jellinger & Attems, 2010). Frontotemporal dementia is much less common than Alzheimer's disease, vascular dementia, or DLB. Huntington's disease is quite rare; it affects only 1 person in every 20,000 (Ross & Tabrizi, 2011).

CROSS-CULTURAL COMPARISONS

Several issues make it difficult to collect cross-cultural data regarding the prevalence of dementia. Tests that are used to measure cognitive impairment must be developed carefully to be sure that they are not culturally or racially biased (see Chapter 4 on the validity of assessment procedures). Elderly people in developing countries who have little formal education pose a special challenge, since most cognitive tasks have been developed for use with a different population. Those who follow more traditional ways of life, such as the Australian aboriginal people, may have very different views of old age and its problems. For all these reasons, we must interpret preliminary results on this topic with great caution (Prince et al., 2003).

Some studies have reported that prevalence rates for dementia vary geographically. Alzheimer's disease may be more common in North America and Europe, whereas vascular dementia may be more common in Japan and China (Chiu et al., 1998). There are also some tentative indications that prevalence rates for dementia may be significantly lower in developing

countries than in developed countries. This finding can be misleading, however, because the most common dementias are age-related. Because developing countries have much lower life expectancies, they would also be expected to have lower rates of dementia.

Causes

Delirium and dementia are clearly associated with brain pathology. Damage to various brain structures and neurotransmitter pathways can be the product of various biological and environmental events. In the following pages, we review some of the considerations that guide current thinking about the causes of these disorders.

DELIRIUM

The underlying mechanisms responsible for the onset of delirium undoubtedly involve neuropathology and neurochemistry (Goldstein, 2003). The incidence of delirium increases among elderly people, presumably because the physiological effects of aging make elderly people more vulnerable to medication side effects and cognitive complications of medical illnesses (Jacobson, 1997). Delirium can be caused by many different kinds of medication, including the following:

- Psychiatric drugs (especially antidepressants, antipsychotics, and benzodiazepines)
- Drugs used to treat heart conditions
- Painkillers
- Stimulants (including caffeine)

Delirium also develops in conjunction with a number of metabolic diseases, including pulmonary and cardiovascular disorders (which can interfere with the supply of oxygen to the brain), as well as endocrine diseases (especially thyroid disease and diabetes mellitus). Various kinds of infections can lead to the onset of delirium. Perhaps the most common among elderly people is urinary tract infection, which can result from the use of an indwelling urinary catheter (sometimes necessary with incontinent nursing home patients).

DEMENTIA

In discussing the classification of dementia, we have touched on many of the factors that contribute to the etiology of these problems. Most of the other disorders listed in DSM-IV-TR are classified on the basis of symptoms alone. The classification of dementia is sometimes determined by specific knowledge of etiological factors, even though these may be determined only after the patient's death, as in Alzheimer's disease. In the following discussion we consider in greater detail a few of the specific pathways that are known to lead to dementia.

Genetic Factors Neurologists who treat demented patients have recognized for many years that the disorder often runs in families. Until recently, twin studies have not been used extensively to evaluate the influence of genetic factors in dementia because of the comparatively late age of onset of these disorders. By the time a proband develops symptoms of dementia, his or her co-twin may be deceased. A few studies have capitalized on national samples to find an adequate number of twin pairs. They confirm the impression, based on family studies, that genetic factors play an important role in the development of dementia. One Swedish study, for example, found that the concordance rate in monozygotic twins was over 50 percent, more than double the dizygotic rate (Pedersen et al., 2004). A U.S. study, based on a registry of aging twin veterans of World War II and the Korean War, found an MZ concordance rate of 35 percent in 24 male pairs. None of the 16 DZ pairs was concordant at the time of the report (Breitner et al., 1993).

Most of the research concerned with genetic factors and Alzheimer's disease has focused on gene identification strategies (see Research Methods on page 376). The astounding advances that have been made in molecular genetics have been applied to Alzheimer's disease with fruitful results. Experts now agree that Alzheimer's disease is genetically heterogeneous. In other words, there are several forms of the disorder, and each seems to be associated with a different gene or set of genes. Three genes (located on chromosomes 21, 14, and 1) have been identified that, when mutated, cause early-onset forms of Alzheimer's disease. A fourth gene, located on chromosome 19, serves as a

Chuck Jackson was diagnosed at the age of 50 with a rare, early-onset form of Alzheimer's disease. Speaking at a Congressional hearing on the disease, Jackson showed a family photo because he is the fifth generation of his family to have Alzheimer's.

risk factor for late-onset forms of the disorder (Holmes, 2002; McQueen & Blacker, 2008). The locations of these genes are illustrated in Figure 14.5, along with graphs that indicate the average age of onset for dementia associated with the different genes.

It has been known for many years that amyloid plaques and neurofibrillary tangles are found in the brains of all people who have Down syndrome (see Chapter 15), as well as in people with Alzheimer's disease. This similarity led investigators to search for a link between the gene for Alzheimer's disease and known markers on chromosome 21, because people with Down syndrome possess three copies of chromosome 21 in every cell instead of the normal two. In fact, the gene responsible for producing proteins (amyloid precursor protein, or APP) that serve as precursors to beta-amyloid, found in the core of amyloid plaques, is located on chromosome 21. Several research groups have independently confirmed this association. Therefore, within some families, the gene for Alzheimer's disease is located on chromosome 21.

Mutations on chromosome 14 (presenilin 1, or PS1) and chromosome 1 (presenilin 2, or PS2) have also been found to be associated with early-onset forms of Alzheimer's disease (Plassman & Breitner, 1997). Like the APP gene, both of the presenilin genes are inherited in an autosomal dominant mode of transmission and cause overproduction of beta-amyloid. Mutations in the PS1 gene are probably responsible for 50 percent of early-onset cases of the disorder (which represent less than 3 percent of all patients with Alzheimer's disease).

A fourth gene produces vulnerability to late-onset Alzheimer's disease without having a direct or necessary effect on the development of dementia. In other words, people who carry this gene have an increased risk for Alzheimer's disease, but many people without the gene develop the disorder, and some people who do have the gene do not develop the disorder. The apolipoprotein E (APOE) gene is located on chromosome 19. There are three common alleles (forms) of APOE, called e-2, e-3, and e-4. The APOE-2 allele is correlated with a decreased risk for Alzheimer's disease. People who have the APOE-4 allele at this locus have an increased probability of developing the disorder (Farrer et al., 1997). Although the effect may be weaker in some groups of people (such as Hispanics and African Americans), the finding has been replicated in more than 100 different laboratories. The risk for Alzheimer's disease is between 25 and 40 percent among people who have at least one APOE-4 allele (Mayeux & Ottman, 1998). Because most cases of Alzheimer's disease have a late onset, the APOE gene is probably involved in more cases of the disorder than the genes on chromosomes 21, 14, and 1.

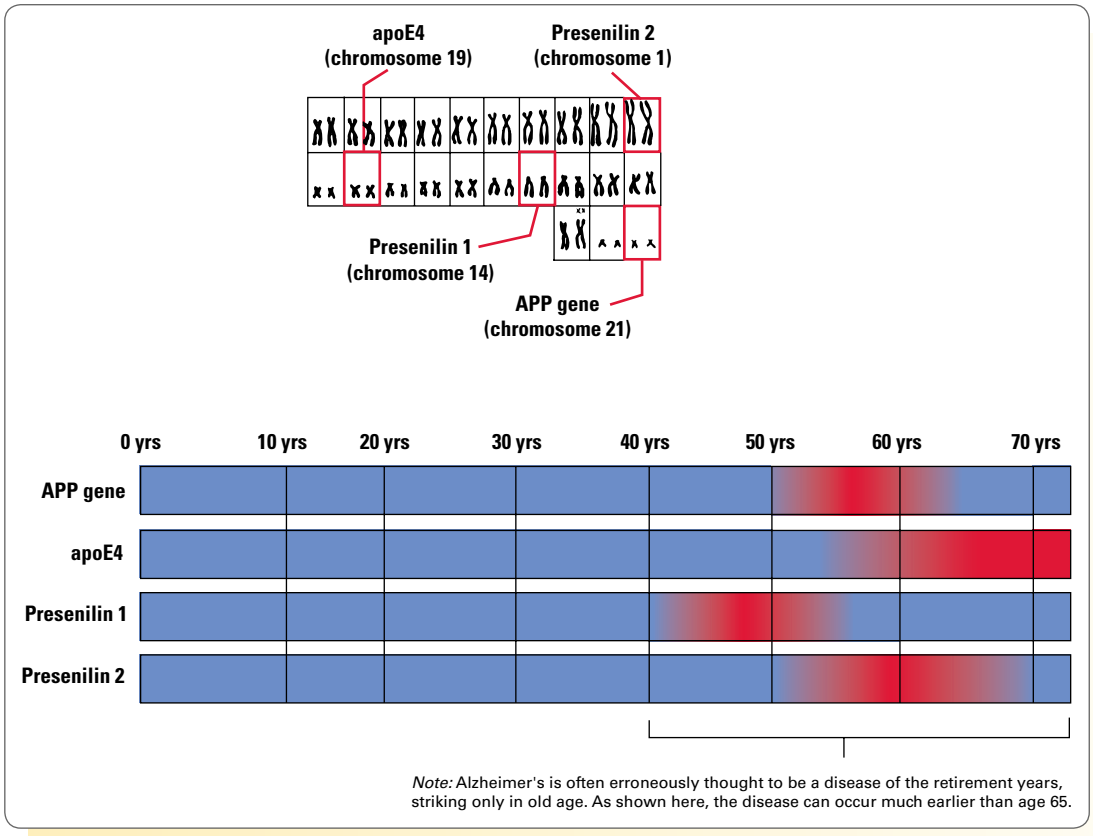


FIGURE 14.5 Genes Associated with Alzheimer's Disease

Four different genes are associated with the creation of plaques found in Alzheimer's disease (AD). The top panel, which illustrates the 23 pairs of human chromosomes, identifies which chromosome carries which AD-related gene. The bottom panel indicates that average age of onset of the disorder depends, in part, on the gene that is involved. The age of first diagnosis is illustrated in red.

Research findings with regard to specific genes and Alzheimer's disease are obviously exciting, but a word of caution is also in order. Although some important genes have been identified, most people who develop the disorder do *not* posses one of these specific genes. In other words, these genes do increase the risk for the disorder, but most cases of the disorder do not follow this pattern. Many questions remain to be answered about the ways in which specific genes interact with other causal factors.

Neurotransmitters In patients suffering from dementia, the process of chemical transmission of messages within the brain is probably disrupted, but the specific mechanisms that are involved have not been identified. We know that Parkinson's disease, which is sometimes associated with dementia, is caused by a degeneration of the dopamine pathways in the brain stem. This dysfunction is responsible for the motor symptoms seen in patients with that disorder. It is not entirely clear, however, that the intellectual problems experienced by patients with Parkinson's disease are directly related to dopamine deficiencies.

Other types of dementia have also been linked to problems with specific neurotransmitters. Huntington's disease may be associated with deficiencies in gamma-aminobutyric acid (GABA). A marked decrease in the availability of acetylcholine (ACh), another type of neurotransmitter, has been implicated in Alzheimer's disease. Reductions in ACh levels, especially in the temporal lobes, are correlated with the severity of dementia symptoms (Kihara & Shimohama, 2004; Raskind & Peskind, 1997).

Viral Infections Some forms of primary dementia are known to be the products of "slow" viruses—infections that develop over a much more extended period of time than do most viral infections. Creutzfeldt-Jakob disease is one example. Susceptibility to infection by a specific virus can be influenced by genetic factors. The demonstration that a condition is transmitted in a familial fashion does not rule out the involvement of viral infection. In fact, familial transmission has been demonstrated for the forms of dementia that are known to be associated with a specific virus.

Immune System Dysfunction The immune system is the body's first line of defense against infection. It employs antibodies to break down foreign materials, such as bacteria and viruses, that enter the body. The regulation of this system allows it to distinguish between foreign bodies that should be destroyed and normal body tissues that should be preserved. The production of these antibodies may be dysfunctional in some forms of dementia, such as Alzheimer's disease. In other words, the destruction of brain tissue may be caused by a breakdown in the system that regulates the immune system.

The presence of beta-amyloid at the core of amyloid plaques is one important clue to the possible involvement of immune system dysfunction. This protein is the breakdown product of a structural component of brain cells. It is made and eliminated constantly as part of normal brain functioning. For some reason, which probably involves genetic factors, some people develop problems with the elimination of beta-amyloid. Clumps of beta-amyloid accumulate. Some clinical scientists believe that immune cells in the brain attempt to destroy these amyloid plaques and inadvertently harm neighboring, healthy brain cells. Some research evidence supports this hypothesis (McGeer & McGeer, 1996; Richardson, 1996).

Environmental Factors Epidemiological investigations have discovered several interesting patterns that suggest that some types of dementia, especially Alzheimer's disease, may be related to environmental factors. One example is head injury, which can cause a sudden increase of amyloid plaque. Elderly people who have been knocked unconscious as adults have an increased risk of developing Alzheimer's disease, compared to people with no history of head injury (Holsinger et al., 2002; O'Meara et al., 1997).

Some studies have reported significant relationships between Alzheimer's disease and variables that seem to protect the person from developing dementia. People who have achieved high levels of education are less likely to develop Alzheimer's disease than are people with less education (Johnson et al.,

Cary Henderson, a patient with Alzheimer's disease, celebrates his 62nd birthday. The drawing and note from his granddaughter illustrate the impact this disorder has on families. Henderson described his experiences as the disorder progressed in *Partial View: An Alzheimer's Journal*.

His granddaughter drew this picture of him and wrote:
My Friend
My Grandpa is my friend. He has glasses with brown rimes around them. He has crystel blue eyes. At first my Grandpa was so very smart. He got his masters degree. Other people called him Docter Cary Henderson. But then he got a dazise. He got Allsimer's dazise. It got worse. Now people have to help him.

1997; Stern et al., 1994). For example, one fascinating study has reported that among elderly Catholic nuns those who graduated from college were much less likely to be cognitively impaired than were those who had less than a college education (Butler, Ashford, & Snowdon, 1996). This finding may be interpreted to mean that increased "brain work" leads to a facilitation of neuronal activation, increased cerebral blood flow, and higher levels of glucose and oxygen consumption in the brain. All of this may increase the density of synaptic connections in the person's cortex and reduce risk for later neuronal deterioration. The discovery of environmental experiences (e.g., going to school) that serve a protective function points to the important role that cultural factors may play in moderating risk for dementia.

Treatment and Management

The most obvious consideration with regard to treatment of the cognitive disorders is accurate diagnosis (Cummings & Cole, 2002). The distinction between delirium and dementia is important because many conditions that cause delirium can be treated. Delirium must be recognized as early as possible so that the source of the problem, such as an infection or some other medical condition, can be treated (Bourne et al., 2008). Some types of secondary dementia can also be treated successfully. For example, if the patient's cognitive symptoms are the products of depression, there is a relatively good chance that he or she will respond positively to antidepressant medication or electroconvulsive therapy.

When the person clearly suffers from a primary type of dementia, such as dementia of the Alzheimer's type, a return to previous levels of functioning is extremely unlikely. No form of treatment is presently capable of producing sustained and clinically significant improvement in cognitive functioning for patients with Alzheimer's disease (Tune, 2007). Realistic goals include helping the person to maintain his or her level of functioning for as long as possible in spite of cognitive impairment and minimizing the level of distress experienced by the person and the person's family. Several treatment options are typically used in conjunction, including medication, management of the patient's environment, behavioral strategies, and providing support to caregivers.

MEDICATION

Some drugs are designed to relieve cognitive symptoms of dementia by boosting the action of acetylcholine (ACh), a neurotransmitter that is involved in memory and whose level is reduced in patients with Alzheimer's disease. One drug that has been approved for use with Alzheimer's patients—donepezil (Aricept)—increases ACh activity by inhibiting acetylcholinesterase, the enzyme that breaks down ACh in the synapse. Research studies have demonstrated that donepezil can provide temporary symptomatic improvement for some patients (Kumagai et al., 2008; Rojas-Fernandez et al., 2001). Unfortunately, it usually works for only six to nine months and is not able to reverse the relentless progression of the disease. Furthermore, its use has been seriously questioned because of the relatively small effects on memory that it is able to produce (Pryse-Phillips, 1999). A statistically significant change in scores on a cognitive task does not necessarily imply a clinically significant improvement in overall clinical condition (see Research Methods in Chapter 6).

New drug treatments are being pursued that are aimed more directly at the processes by which neurons are destroyed (Sabbagh, Richardson, & Relkin, 2008). One possibility involves the use of synthetic peptides and natural proteins that inhibit the formation of amyloid plaques. Others focus on blocking the construction of neurofibrillary tangles by keeping tau protein anchored to microtubules. These alternatives are being developed and tested at a rapid pace. Recent evidence regarding these new treatment options can be obtained on the Web from the Cochrane Library.

How could education help to reduce a person's risk for dementia?

Although the cognitive deficits associated with primary dementia cannot be completely reversed with medication, neuroleptic medication can be used to treat some patients who develop psychotic symptoms (Martinez & Kurik, 2006). These are the same drugs that are used to treat schizophrenia. Low doses are preferable because demented patients are especially vulnerable to the side effects of neuroleptics. Care must be taken to avoid use of these drugs with patients suffering from dementia with Lewy bodies because they may experience a severe negative reaction.

ENVIRONMENTAL AND BEHAVIORAL MANAGEMENT

Patients with dementia experience fewer emotional problems and are less likely to become agitated if they follow a structured and predictable daily schedule. Activities such as eating meals, exercising, and going to bed are easier and less anxiety-provoking if they occur at regular times. The use of signs and notes may be helpful reminders for patients who are in the earlier stages of the disorder. As the patient's cognitive impairment becomes more severe, even simple activities, such as getting dressed or eating a meal, must be broken down into smaller and more manageable steps. Directions have to be adjusted so that they are appropriate to the patient's level of functioning. Patients with apraxia, for example, may not be able to perform tasks in response to verbal instructions. Caregivers need to adjust their expectations and assume increased responsibilities as their patients' intellectual abilities deteriorate.

Severely impaired patients often reside in nursing homes and hospitals. The most effective residential treatment programs combine the use of medication and behavioral interventions with an environment that is specifically designed to maximize the level of functioning and minimize the emotional distress of patients who are cognitively impaired. Several goals guide the design of such an environment (Gauthier et al., 2010). These include considerations that enhance the following aspects of the patient's life.

- *Knowledge of the environment:* For example, rooms and hallways must be clearly labeled, because patients frequently cannot remember directions.

These elderly residents of Havana, Cuba, are participating in an exercise program that is designed to keep the aging population fit and healthy.

- *Negotiability:* In the case of dementia, psychological accessibility is at least as important as physical accessibility. For example, spaces that the person would use (a commons area or the dining room) should be visible from the patient's room if they cannot be remembered.
- *Safety and health:* For example, access to the setting must be secured so that patients who would otherwise wander away can remain as active as possible.

One important issue related to patient management involves the level of activity expected of the patient. It is useful to help the person remain active and interested in everyday events. Patients who are physically active are less likely to have problems with agitation, and they may sleep better. Engaging in a home-based exercise program can reduce functional dependence and delay institutionalization among patients with dementia (Rockwood & Middleton, 2007). Nevertheless, expectations regarding the patient's activity level may have to be reduced in proportion to the progression of cognitive impairment. Efforts should be made to preserve familiar routines and surroundings in light of the inevitable difficulties that are associated with learning new information and recalling past events. Helping the person to cope with these issues may minimize the emotional turmoil associated with the increasing loss of cognitive abilities.

MyPsychLab VIDEO CASE

Wife of Patient with Alzheimer's Disease

SARAH

"You kind a have hope whenever you have cancer, (but with) Alzheimer's you die just a little each day . . ."

Watch the video "Alzheimer's Disease: The case of Wilburn Johnson" on MyPsychLab. What are the special challenges faced by family members and those who care for people suffering from Alzheimer's disease?

SUPPORT FOR CAREGIVERS

A final area of concern is the provision of support to people who serve as caregivers for demented patients. In the United States, spouses and other family members provide primary care for more than 80 percent of people who have dementia of the Alzheimer's type (Ballard, 2007). Their burdens are often overwhelming, both physically and emotionally. Consider, for example, the situation described by Bernlef in *Out of Mind*. In the following passage, Maarten describes the experience of listening to his wife, Vera, describe to a young woman (whom Vera has hired to help care for Maarten) how she has felt while attempting to cope with his progressive cognitive deterioration:

> I hear Vera. "More than 40 years I have been married to him. And then suddenly this. Usually these things happen more slowly, gradually. But with him it came all at once. I feel it has been sprung on me. It's cruel and unfair. Sometimes I get so angry and rebellious when I see him looking at me as if from another world. And then again I feel only sad and I would so much like to understand him. Or I just talk along with him and then I feel ashamed afterwards. I'm glad you're here because it really gets on top of me at times, when I just can't bear watching it any more. At least now I'll be able to get out occasionally."
>
> There is a moment of silence. I feel the tears running under my eyelids and down my cheeks.
>
> "And sometimes, sometimes his face radiates perfect peace. As if he's happy. Like a child can be. Those moments are so brief I sometimes think I imagine them. But I know only too well what I see at such moments: someone who looks exactly like my husband of long ago. At your age it's difficult to understand that. But