Substance Use, Abuse, and Addiction

Ryan, who is 24 years old, plays football for a hard-drinking local pub team. One evening, teammates brought him to the emergency room pale, sweating, in obvious pain, and near collapse. “It’s my chest,” he said to the resident on call and went on to explain that he’d been vomiting profusely for three days. Asked what might have brought this on, a sheepish-looking Ryan explained that his team had made it to the league final the previous weekend and he’d had “a fair bit to drink.” Pressed for specifics, he admitted to drinking more than 20 cans of strong lager beer in the course of one evening of celebration.

Upon examination, Ryan had a fever; a rapid pulse of 120 beats per minute, raised white blood cell count, and was dehydrated with abnormal electrolyte levels. A chest X-ray and CT scan showed the presence of free air along the left border of Ryan’s heart, apparently caused by a traumatic rupture of his esophagus. Spontaneous rupture of the esophagus is very rare, but it is life threatening and has a survival rate that quickly drops to as low as 11 percent if diagnosis and intervention are delayed by as little as 48 hours. Ryan had been vomiting profusely for three days.

Ryan’s perforated esophageal wall was diagnosed as Boerhaave syndrome, the result of forceful vomiting and a sudden increase in pressure within the 12-inch passageway between his stomach and throat. His greatest danger was from extensive sepsis, which occurs when chemicals released into the bloodstream to fight an infection trigger inflammation that spreads throughout the body, potentially causing multiple organs to fail. Fortunately, after surgery and 10 days of hospitalization with broad spectrum antibiotic treatment, Ryan was healthy enough to return home. One month later, he was able to resume playing football, this time swearing that he would never again risk the near-fatal effects that can occur by binge drinking.
Since antiquity, human beings have sought ways to alter their moods, thought processes, and behaviors—often with significantly negative health effects. This chapter examines the different facets of substance abuse—its causes, effects, and prevention, including research pointing to a biopsychosocial common ground in the origins of addiction to many habit-forming substances. We will focus primarily on the two most commonly abused drugs: tobacco and alcohol.

**Some Basic Facts**

Drug abuse is the use of a chemical substance to the extent that it impairs the user’s well-being in any domain of health: biological, psychological, or social. Tobacco and alcohol, two of the most widely used drugs, both pose significant health dangers. Although tobacco use is decreasing in affluent countries, its use is climbing in low- and middle-income countries. Alcohol is the third-largest risk factor for disease in the world (after lack of food for children and unsafe sex), largely due to injuries, with some 2.5 million deaths each year, including 320,000 among young adults, from alcohol-related causes (WHO, 2010a). Although alcohol consumption has recently declined in many developed countries, its use also has been increasing in developing countries. Moreover, alcohol problems are occurring now in many Asian and Western Pacific countries where they did not previously exist.

The abuse of illegal drugs, alcohol, and tobacco causes more deaths, illnesses, and disabilities than any other preventable health condition (United Nations Office on Drugs and Crime [UNODC], 2015). Alcohol, for instance, is implicated in 40 percent of all traffic deaths. Half of all murders in the United States involve alcohol or some other drug, and 80 percent of all suicide attempts follow the use of alcohol. And each year, tobacco use causes nearly 6 million deaths globally (an estimated 8 million by the year 2030) and results in a net loss of over $300 billion in the United States alone due to medical expenses and lost work time (WHO, 2015).

Although the use of most drugs has decreased in the United States over the past 30 years, the use of marijuana has recently undergone a resurgence, which I will discuss at the end of this chapter. Further, abuse of synthetic narcotics (so-called designer drugs) has also increased. Among the most popular designer drugs are hallucinogens such as NBOMes (pronounced en-bombs), stimulants such as bath salts, and spice—a synthetic drug that mimics the effects of marijuana. When a designer drug first appears for sale—often in convenience stores, gas stations, or online—it is technically not illegal, because its chemical makeup is slightly different from the illicit drug it is modeled after.

The abuse of prescribed medications is also on the rise. Over 2 million people in the United States suffer from substance use disorders related to prescription pain relievers (National Institute on Drug Abuse, 2015). Each year, more than 1 million adults are treated in hospital emergency rooms for overdoses due to “nonmedical” use of over-the-counter or prescription drugs (Raffol, 2010). Figure 9.1 shows the 11 most abused prescription drugs.

Why, despite these enormous financial, health, and social costs, do people continue to abuse drugs? To answer this question, you first need to know how drugs move through and affect the body.

**Mechanisms of Drug Action**

Drugs are ingested, or administered, in one of five ways: orally, rectally, by injection, by inhalation, or by absorption through the skin or the mucous membranes. The manner in which a drug is administered can alter its physiological effects. For example, because they
enter the bloodstream faster, drugs that are injected or inhaled usually have stronger and more immediate effects than those that are swallowed.

Within minutes after a drug is absorbed, it is distributed by the bloodstream to its site of action (receptors). How quickly a drug reaches its target receptors depends on the rate of blood flow and how easily the drug passes through cell membranes and other protective barriers. Blood flow to the brain is greater than to any other part of the body. Therefore, drugs that are able to pass through the network of cells that separates the blood and
the brain—the blood–brain barrier (Figure 9.2)—move quickly into the central nervous system. The ease with which a drug passes through this barrier depends on its lipid (fat) solubility.

Fat-soluble drugs that cross the blood–brain barrier are usually also able to permeate the placental barrier, which separates the blood of a pregnant woman from that of her developing child. For this reason, alcohol, nicotine, or other drugs, as well as chemicals in cosmetics, foods, and the environment absorbed by the mother, can affect her unborn child. Scientists now understand a great deal about teratogens—drugs, pollutants, and other substances that cross the placental barrier and damage the developing person. The extent of damaging effects, however, depends on when exposure occurs; the greatest damage occurs during critical periods of development when specific organs and systems are developing most rapidly.

Drugs and Synapses
Once in the brain, drugs affect behavior by influencing the activity of neurons at their synapses. Drugs can achieve their effects in one of three ways: by mimicking or enhancing the action of a naturally occurring neurotransmitter, by blocking its action, or by affecting its reuptake (see Figure 9.3a).

Drugs that produce neural actions that mimic or enhance those of a naturally occurring neurotransmitter are agonists (see Figure 9.3b). Recall that synaptic receptors are cellular locks that wait for neurotransmitters with a particular shape to act like a key and trigger activity within the cell. Partial agonists are neurotransmitters that bind and activate receptors but elicit a smaller response than true or full agonists (see Figure 9.3c).

Drugs that produce their effects by blocking the action of neurotransmitters or agonist drugs are antagonists (see Figure 9.3d). Caffeine, for example, is an antagonist that blocks the effects of adenosine, a neurotransmitter that normally inhibits the release of other transmitters that excite (cause to fire) postsynaptic cells. Thus, the excitatory cells continue firing, resulting in the stimulation felt when caffeine is ingested.

Finally, drugs can alter neural transmission by enhancing or inhibiting the reuptake of neurotransmitters in the synapse (that is, the natural breakdown or reabsorption of the neurotransmitter by the presynaptic neuron). One neurotransmitter that plays a major role in addiction is dopamine. As a chemical messenger, dopamine is similar to adreneline and affects neural processes that control movement, emotions, and the experiences of pleasure and pain. Cocaine, for example, produces its stimulating effect by binding to proteins that normally transport dopamine, thus blocking its reuptake. Because dopamine is not reabsorbed by the sending neuron, it remains in the synapse and continues to excite the receiving neuron. As more dopamine remains to stimulate neurons, the result may be prolonged feelings of pleasure and excitement. Similarly, amphetamine increases dopamine levels and causes overstimulation of the pleasure-pathway nerves in the brain.

Addiction, Dependence, and Tolerance
The American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders (DSM-5) defines substance use disorder as a behavior pattern characterized by impaired control, social impairment, and risky use of a drug.

Dependence is a state in which the body and mind have adjusted to the repeated use of a drug and require its presence in order to maintain “normal” functioning. In this context, normal refers to the absence of the withdrawal symptoms that will appear when use of a
drug is discontinued. Drug withdrawal refers to the unpleasant physical and psychological symptoms that occur when a person abruptly ceases using a drug. The symptoms of withdrawal are generally the direct opposite of a drug’s primary effects. Amphetamines, for example, create a rush of euphoria, while withdrawal triggers the opposing state of depression, along with vomiting, sleep disturbances, anxiety, and even death.

Consistent with the biopsychosocial model, most drugs give rise to both physical and psychological dependence. For example, alcohol, which produces biochemical changes in the brain (see page 256), also seems to improve mood and allow a person to forget his or her problems. For many former stimulant drug users, memories of the “highs” once experienced fade slowly and are constant triggers for relapse. For people who are dependent, obtaining and using a drug can become the day’s focus (see Table 9.1).

The complementary effects of drug use and drug abstinence have led to a general theory—the hypersensitivity theory—which proposes that addiction is the result of efforts by the body and brain to counteract the effects of a drug to maintain an optimal internal state. For example, nicotine accelerates heart rate. To compensate, the brain and nervous system respond by stimulating the vagus nerve, which slows the heart rate. Over time,
regular use of nicotine and the associated vagus nerve stimulation seem to create a new, higher “set point” for vagus nerve activity. If the person quits smoking, vagal activity remains high because there is no nicotine in the system to increase the heart rate.

One sign of dependence is the development of **tolerance**, a state of progressively decreasing behavioral and/or physiological responsiveness to a frequently used drug. The drug user’s brain chemistry adapts to offset the drug’s effect (a process called **neuroadaptation**), and increased dosages are necessary to produce the effect formerly achieved by a smaller dose. With repeated use, some drugs are metabolized at a faster rate by the liver so that more of the drug must be administered simply to maintain a constant level in the body. Also, brain receptors adapt to the continued presence of a particular drug either by increasing the number of receptor sites or by reducing their sensitivity to the drug.

**Psychoactive Drugs**

Chemical substances that act on the brain to alter mood, behavior, and thought processes are known as **psychoactive drugs**. Psychoactive drugs are grouped into three major categories: hallucinogens, stimulants, and depressants.

Also called **psychedelic drugs**, hallucinogens such as marijuana and LSD alter sensory perception, induce visual and auditory hallucinations as they separate the user from reality, and disrupt thought processes.

**Stimulants**, including nicotine, caffeine, cocaine, and amphetamines, make people feel more alert and energetic by boosting activity in the central nervous system. At low doses, the moderate stimulants (nicotine and caffeine) reduce fatigue, elevate mood, and decrease appetite. However, in higher doses of the moderate drugs and with the more extreme drugs in any dosage, stimulants may cause irritability, insomnia, and anxiety. Like all psychoactive drugs, stimulants produce their effects by altering the action of neurotransmitters at synapses. They have a dramatic impact on acetylcholine, the catecholamines, dopamine, and, to a lesser extent, norepinephrine. Because of their powerful reward effects, stimulants are widely abused. Dependence and tolerance develop rapidly, forcing the addict to take progressively higher doses. Withdrawal symptoms associated with amphetamines include increased appetite, weight gain, fatigue, sleepiness, and, in some people, symptoms of paranoia.

**Depressants** (barbiturates, opiates, alcohol, general anesthetics, and antiepileptic drugs) dampen activity in the central nervous system. Low doses reduce responsiveness to sensory stimulation, slow thought processes, and lower physical activity. Higher doses result in drowsiness, lethargy, and amnesia and also can lead to death.

**Barbiturates** are used to block pain during surgery and to regulate high blood pressure. They are also popular street drugs because they produce a long-lasting sense of euphoria. They are highly addictive and considered particularly dangerous because, when taken in combination with another drug, they increase the effects of that drug, a reaction known as **TABLE 9.1**

**Substance Dependence**

According to the *Diagnostic and Statistical Manual of Mental Disorders*, the presence of three or more of the following indicates **dependence** on a substance:

- Tolerance (with repeated use, a drug must be taken in higher doses to achieve the desired effect)
- Withdrawal (discomfort and distress when discontinued)
- Taking the substance longer or in greater amounts than intended
- Failure to regulate use
- Much time devoted to obtaining the substance
- Normal activities abandoned or reduced
- Continued use despite knowledge that using the substance worsens problems
**drug potentiation.** In combination with alcohol, for example, a barbiturate may suppress the brain's respiratory centers and cause death.

Another group of depressants, the opiates (morphine, heroin, and codeine), derive from the opium poppy. Globally, it is estimated that 16.5 million adults used nonprescribed opiates at least once in 2014, including 12 to 14 million heroin users (UNODC, 2015). Opiates produce their effects by mimicking the body's natural opiates, the endorphins, which help regulate our normal experience of pain and pleasure. When the brain is flooded with artificial opiates such as heroin, molecules of these synthetic drugs bind to the receptor sites for the endorphins, and the brain stops producing its own naturally occurring opiates. If the drug is discontinued, withdrawal symptoms soon occur, including rapid breathing, elevated blood pressure, severe muscle cramps, nausea and vomiting, panic, and intense cravings for the drug.

### Models of Addiction

Theories about how people become addicted to drugs can be grouped into three general categories: biomedical models, reward models, and social learning models.

**Biomedical Models: Addiction as Disease**

Biomedical models of addiction view physical dependence as a chronic brain disease caused by the biological effects of psychoactive drugs. The simplest model maintains that addicts inherit a biological vulnerability to physical dependence. Researchers point to evidence from studies that compare the concordance rate, or rate of agreement, of physical dependence among identical and fraternal twins. However, even though these studies suggest that genes play a role in physical dependence, researchers are cautious in interpreting the results because it is impossible to rule out completely possible confounding effects due to other variables. Moreover, twin studies do not pinpoint the specific gene or genes that might promote physical dependence.

Another biomedical model points to altered neurochemistry as the basis for both physical and psychological dependence. According to the withdrawal-relief hypothesis, drug use serves to restore abnormally low levels of dopamine, serotonin, and other key neurotransmitters (Robinson & Berridge, 2003). Depression, anxiety, low self-esteem, and other unpleasant emotional states are indeed associated with neurotransmitter deficiencies and with substance use (Kim, Lim, & Kim, 2003). By elevating the release of presynaptic dopamine, drugs such as cocaine and the amphetamines restore neural functioning and produce a sense of psychological well-being.

For most of the twentieth century, the withdrawal-relief model was based primarily on evidence that opiates trigger dependence by suppressing the brain’s natural production of endorphins. As the first receptor-based theory of addiction, the opiate model was adopted quickly as the basic biomedical model for addiction to all drugs that induce physical dependence. Nicotine acts on acetylcholine receptors, amphetamine and cocaine act on catecholamine receptors, and barbiturates presumably act on receptors for gamma-aminobutyric acid. In each case, addiction might involve the same sequence of receptor adaptation to an artificial source. One glaring exception to the opiate receptor theory as a general model, however, is alcohol, which does not appear to act on specific receptors.

The withdrawal-relief model was appealing because the idea that addicts need more of their drug to relieve physical distress made their intense determination to obtain drugs
seem understandable—a rational response to their withdrawal sickness. However, the model does not explain why addicts begin taking a drug in sufficient dosages and with enough frequency to develop physical dependence in the first place. A second problem with this model is its inability to explain why many users suffer a relapse, even long after withdrawal symptoms have subsided.

Reward Models: Addiction as Pleasure Seeking

Researchers trying to explain the initial motivation for repeated use have focused on the pleasurable effects of psychoactive drugs. Recall from Chapter 3 that some experts believe that certain addictions may stem from a genetic reward deficiency syndrome in which the brain's reward circuitry malfunctions and leads to powerful cravings.

The Brain's Reward System

All major drugs of abuse overstimulate the brain's reward system, which also becomes active when a person engages in pleasurable behaviors that promote survival, such as eating or having sex. Given the choice between psychoactive drugs that put this reward circuit into overdrive—repeatedly activating the neurons until the drug leaves the body—and other, more mundane pleasures, physically dependent animals and human addicts often will choose the former. Rats that were allowed to press a lever to stimulate their reward systems electrically were observed to do so up to 7000 times per hour (Figure 9.4).

According to the reward models, addiction may best be understood as being motivated by pleasure seeking. Psychoactive drugs may induce physical dependence because they increase the availability of dopamine in the brain, overstimulating the reward system (Thompson, 2000). Evidence for the reward system link comes from the fact that people who develop dependence for one drug are more likely to be addicted to others as well. Use of tobacco, alcohol, and marijuana often plays a pivotal role in the development of other drug dependencies and high-risk behaviors. So these drugs are often referred to as gateway drugs because they “open the door” to experimentation with other drugs.

Shortcomings of the Reward Model

Despite its seeming logic, the reward model does not provide the final answer to the question of what causes addiction. It is true that cocaine, heroin, and other highly addictive drugs evoke the most powerful euphoria, but marijuana and other psychoactive drugs that are not considered physically addictive also produce feelings of well-being. In contrast, tobacco, which is highly addictive and as difficult to abstain from as cocaine or heroin, induces a very mild euphoria that is hardly on the same scale as that elicited by cocaine.

Another shortcoming of the reward model concerns the gateway hypothesis. Tobacco and alcohol use, for instance, have historically been considered powerful predictors of marijuana use. However, some newer research indicates that environmental factors may have a stronger influence on subsequent illicit drug use. A 12-year University of Pittsburgh study looked at a group of boys beginning at age 10, 214 of whom eventually used legal or illegal drugs. At 22 years of age, these 214 participants were categorized into three groups: those who had used only tobacco or alcohol, those who first used alcohol or tobacco and later used marijuana (gateway drug use), and those who used marijuana before using alcohol or tobacco. Nearly 25 percent of the participants reported using marijuana first. Three environmental factors differentiated these reverse pattern marijuana users: They were more likely to have lived in economically deprived neighborhood environments, had more exposure to drugs in these neighborhoods, and had less parental

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**FIGURE 9.4**

**Intracranial Self-Stimulation**

Whenever the small lamp on the panel is lit, pressing the lever causes an electrical stimulus to be delivered to the reward system of the rat's brain. Using this experimental arrangement, researchers have found that rats press the lever at rates faster than one response per second.
involvement when they were young children (Tarter, Vanyukov, Kirisci, Reynolds, & Clark, 2006). These data support what’s known as common liability to addiction, which states that the likelihood a person will begin using illegal drugs is determined not by the preceding use of other specific legal drugs (gateway hypothesis), but instead by the particular tendencies and environmental circumstances of the drug user (Vanyukov, Tarter, & Ridenour, 2012).

In addition, reward models by themselves are unable to explain why drug use continues even when unpleasant side effects occur. Why don’t alcohol abusers abstain, given the nausea and vomiting that they often experience? Terry Robinson and Kent Berridge (2000) two-stage theory, known as the wanting-and-liking theory (also called the incentive-sensitization theory), provides a rationale for this. In the first stage, the original good feelings from drug use prevail; in the second stage, drug use becomes an automated behavior. Even though pleasure may not increase (and side effects may even be unpleasant), the reward systems continue to respond to the drug-related cues because they have become conditioned stimuli that evoke dopamine release and craving (Everitt & Heberlein, 2013).

Social Learning Models: Addiction as Behavior

Although psychoactive drugs trigger neurochemical changes and research points to hereditary risk factors in dependence, there is good reason to view addiction as a behavior that is shaped by learning as well as by social-cultural and cognitive factors. For instance, exposure to pro-smoking media, such as point-of-sale displays, magazine advertising, and portrayals of smoking in movies, increases smoking in adolescence (Shadel, Martino, Setodji, & Scharf, 2012). As another example, smokers “learn” to smoke in a variety of situations, such as socializing with friends or after eating a meal. Through conditioning, the pleasurable physiological effects of nicotine, together with other rewarding aspects of social situations, transform these situations into powerful triggers for smoking.

A person’s identification with a particular drug also plays a role in both the initiation and the maintenance of addiction. Adolescence is a time of pronounced self-concept development, and self-concept regulation strongly influences health behaviors (Shepperd, Rothman, & Klein, 2011). Adolescents are preoccupied with their public images and the social implications of their behaviors. Drinking and smoking, for example, may lead to the adoption of a certain lifestyle that makes abstinence a monumental task involving a new sense of self. Similarly, research studies have shown that the more adolescents feel that smoking is a defining aspect of who they are, the more likely they are to smoke (Hertel & Mermelstein, 2012). Conversely, adolescents who are encouraged to think about the positive social consequences of abstaining from alcohol use subsequently have lower levels of willingness to drink when opportunities arise (Moeller and others, 2002).

As another example of social influence on drug use, people—especially young people—may be protected by family, school, religion, and other social institutions. According to the social control theory, the stronger a young person’s attachment to such institutions, the less likely he or she will be to begin using drugs. The University of Michigan’s Monitoring the Future Study is an annual survey—ongoing since 1975—of 50,000 eighth-, tenth-, and twelfth-grade students. The data from this study show that adolescents who do well in school are much more likely never to have used alcohol, tobacco, marijuana, or other drugs later in their lives than students who do poorly (MTF, 2015).

The closely related peer cluster theory maintains that peer groups are strong enough to overcome the controlling influence of family, school, or religious values. Adolescents tend to change their smoking behavior to that of their friends, and the tendency to select friends based on similar smoking behavior is a strong predictor of smoking behavior (Mercken, Steglich, Sinclair, Holliday, & Moore, 2012). This may be partly because some group settings, such as college campuses, induce false beliefs about social norms
Why Adolescence?
As we have seen, there seems to be a window of vulnerability in adolescence for risky behavior. Both the prevalence and incidence of drug use increase every year from age 10 to 25 and then decrease. One exception to this pattern is the use of *inhalants* from cleaning fluids, aerosol containers, and such, which are used more by younger adolescents, perhaps because they can be acquired more easily. Substance *use* before age 18 is a strong predictor of later substance *abuse*, in part because adolescents are limited in their ability to analyze risks cognitively, partly because brain maturation is incomplete (see Chapter 3). For this reason, health psychologists are concerned with findings from the Monitoring the Future Study showing that 25 percent of high school seniors had five drinks in a row in the past two weeks, 11 percent were daily cigarette smokers, and 5 percent were daily marijuana users (MTF, 2015).

Drug use among adolescents varies markedly from place to place, again suggesting the influence of social-cultural factors. For example, the United States has a drinking age of 21, whereas many European countries have younger minimum drinking ages (as young as 16 in France and Italy and 18 in Ireland, Sweden, and the United Kingdom). Throughout most of Europe, alcohol is widely used, even by children. In much of the Middle East, however, alcohol use is illegal, and adolescents rarely drink. In many Asian countries, smoking is permitted everywhere. In the United States, smoking is widely advertised yet banned in most schools and indoor public places. In Canada, cigarette advertising is banned. Could this be a factor in why fewer Canadian teens smoke?

Throughout the world, adolescent boys generally use more drugs than do girls, and they use them more often. Gender differences in *substance use* are tied to self-image and self-presentation and are reinforced by social constructions regarding appropriate male and female behavior. That is, they are influenced by teens’ efforts to appear sophisticated, cool, or tough in the peer environment (Evans, Powers, Hersey, & Renaud, 2006). In the United States, boys and girls are similar in terms of *which* drugs they use, with two exceptions: boys use more illegal drugs and steroids, while girls use more diet drugs (Johnston, O’Malley, Bachman, & Schulenberg, 2013).

Substance abuse among teens often occurs with other unhealthy behaviors as part of a problem behavior syndrome that also includes intercourse in early adolescence, depressive symptoms, and general delinquency (Donahue, Lichtenstein, Langstrom, & D’Onofrio, 2013). Teens who display one or more of these behaviors often have high levels of family conflict and poor self-control and emotional regulation, which suggests that these behaviors may be emotional coping mechanisms in the face of a stressful environment (Goliath and Pretorius, 2016). For some teens, alcohol and other drugs, for instance, allow for a momentary denial of problems. Too often, however, ignored problems don’t go away, a drug is turned to more often, and the teenagers enter a vicious cycle that may lead to addiction.

Findings such as these led some researchers to propose that the stress and strain of adolescence are contributing factors to drug use. In fact, however, prospective studies suggest that drug use *causes* more problems than it solves and often *precedes* depression, rebellion, and anxiety disorders (Meririnne and others, 2010). For example, teenagers who regularly use marijuana may become less motivated to achieve in school and then be more likely to develop other problem behaviors (Ansary & Luthar, 2009).

Fear of addiction may not persuade young adolescents to avoid drug use because, as you may remember from discussions in previous chapters in this book, they often believe that they are invulnerable. They do not know or care that psychoactive drugs excite emotional control centers in the brain’s limbic system, while simultaneously interfering with analytical areas in the prefrontal cortex. These neurological effects make drug users...
more emotional than they otherwise would be, as well as less thoughtful. Every hazard of adolescence—including accidents, unsafe sex, depression, and suicide—is more common among those who are under the influence of a psychoactive drug. For similar reasons, educational campaigns and antidrug advertisements that use scare tactics may backfire and actually increase drug use. Plus, savvy adolescents often recognize exaggerations and other partial truths (Strasburger, Jordan, & Donnerstein, 2009).

An added problem is that many parents and other adult caregivers are unaware of their children’s drug use. In one study, less than 1 percent of the parents of U.S. sixth graders thought their children had ever had alcohol, but 22 percent of the children said they had (O’Donnell, Brydon, Wright, & Steptoe, 2008). In addition to parental monitoring, parents influence their children’s substance use through their own behavior and the social context that they shape at home. Parents who quit smoking by the time their children reach the third grade have children who are more likely to have negative perceptions of smoking, and, in turn, are less likely to smoke themselves (Wyszynski, Bricker, & Comstock, 2011). When parents forbid smoking in their homes, fewer adolescents smoke (Messer, Trinidad, Al-Delaimy, & Pierce, 2008). When parents are careful with their own drinking, fewer teenagers abuse alcohol (Van Zundert, Van Der Vorst, Vermulst, & Engels, 2006). When parents provide guidance about drinking, teenagers are less likely to get drunk or use other substances (Miller & Plant, 2010). Growing up with two married parents reduces cigarette and alcohol use, even when other influences (such as parental smoking and family income) are taken into account (Brown & Rinelli, 2010).

Changing the broader social context also has an impact. Throughout the United States, higher prices, targeted warnings, and better law enforcement have led to a marked decline in cigarette smoking among younger adolescents. In 2014, only 4 percent of eighth graders had smoked cigarettes in the past month, compared with 21 percent 15 years earlier (MTF, 2015).

Now that you understand more about addiction, let’s turn to two of the most common addictions: alcohol and nicotine.

Alcohol Use and Abuse

Alcohol is a depressant that slows the functioning of the central nervous system in a manner similar to tranquilizers. When you drink an alcoholic beverage, approximately 20 percent of the alcohol is rapidly absorbed from the stomach directly into the bloodstream. The remaining 80 percent empties into the upper intestine, where it is absorbed at a pace that depends on whether the stomach is full or empty.

Once alcohol is absorbed, it is evenly distributed throughout body tissues and fluids. It takes a 175-pound man about 1 hour to metabolize the amount of alcohol contained in a 1-ounce glass of 80-proof liquor, a 4-ounce glass of wine, or a 12-ounce bottle of beer (Advokat, Comaty, & Julien, 2014). Drinking at a faster pace results in intoxication because a larger amount of alcohol remains in the bloodstream. Women metabolize alcohol more slowly because they produce less of the enzyme alcohol dehydrogenase, which breaks down alcohol in the stomach. Women also tend to weigh less than men. As a result of both of these factors, women have a higher blood alcohol content than men after consuming the same amount of alcohol.

The amount of alcohol in the bloodstream is your blood alcohol level (BAL). In most states, a BAL of 0.08 grams per 100 milliliters of blood (gpercent) constitutes legal intoxication. It is illegal to attempt to drive an automobile with a BAL at this level or higher. A typical male college student would reach an illegal BAL after consuming one standard drink an hour for every 30 to 35 pounds of body weight. Women would reach an illegal...
Some people develop a higher tolerance and are able to drink larger amounts of alcohol before becoming visibly impaired. For others, however, obvious intoxication may occur with BALs as low as 0.03 or 0.04 gpercent. Regardless of the visible effects, the damaging internal, physiological effects are comparable for all drinkers.

The short-term effects of alcohol are dose-dependent. At BALs ranging from about 0.01 to 0.05 gpercent, a drinker usually feels relaxed and mildly euphoric. As the level increases to 0.10 gpercent, memory and concentration are dulled, and reaction time and motor functioning are impaired significantly. At 0.10 to 0.15 gpercent, walking and fine motor skills become extremely difficult. By 0.20 to 0.25 gpercent, vision becomes blurry, speech is slurred, walking without staggering is virtually impossible, and the drinker may lose consciousness. Death may occur at a BAL of 0.35 or more.

Prevalence of Alcohol Use

As Figure 9.5 shows, 59.4 percent of full-time college students in the United States ages 18 to 22 drank alcohol in the past month compared with 50.6 percent of other persons the same age. The same year, 39 percent of college students engaged in binge drinking (five or more drinks on one occasion) in the past month compared with 33.4 percent of others the same age. Finally, 12.7 percent engaged in heavy drinking (binge drinking on five or more occasions in the past month) compared with 9.3 percent of other nonstudents the same age (SAMHSA, 2015).

The Institute of Medicine considers at-risk drinking as two or more episodes of binge drinking in the past month, or consuming an average of two or more alcoholic drinks per day in the past month. At-risk drinking is a major public health concern because it is linked with poor health, premature death, and a variety of social consequences such as injury, unplanned and unprotected sex, and hostile encounters with police (see Table 9.2). About 1 in 4 college students report academic consequences from their drinking, including missing classes, falling behind, poor exam performance, and getting lower grades overall (National Institute on Alcohol Abuse and Alcoholism, 2015).

The prevalence of the various categories of drinking behavior varies by age, gender, education level, ethnicity, and culture. Adults between 25 and 44 years of age have the highest overall rates of drinking, but the 18 to 24 cohort has the highest rates of binge and heavy drinking (NIAAA, 2015). Alcohol use among adolescents ages 12 to 17 dropped substantially after the legal age for purchasing alcohol was increased to 21 in most states. From the 1980s to 2008, binge drinking among high school students declined by 24 percent (MTF, 2015). Rates of drinking are lowest among older adults (NIAAA, 2015).

**FIGURE 9.5**

Alcohol Use in the United States  A total of 59.4 percent of full-time college students in the United States drank alcohol in the past month compared with 50.6 percent of others the same age. The same year, 39 percent of college students engaged in binge drinking (5 or more drinks on one occasion) in the past month compared with 33.4 percent of others the same age. Finally, 12.7 percent engaged in heavy drinking (binge drinking on 5 or more occasions in the past month) compared with 9.3 percent of other nonstudents the same age.

Compared with women, significantly more men are current drinkers, binge drinkers, and heavy drinkers (15 or more drinks per week for men or 8 per week for women). Binge drinking is defined as having 5 or more drinks for males and 4 or more drinks for females on one occasion or within a short period of time (Centers for Disease Control and Prevention, 2016a). European-Americans have higher rates of drinking than African-Americans, Asian-Americans, or Hispanic-Americans. African-Americans are less likely to be heavy drinkers than European-Americans and Hispanic-Americans.

Although the specific causes of ethnic and cultural group differences in risk are not known, researchers have pointed to several possibilities. A powerful protective factor in the social-cultural environment is racial identity (RI)—an aspect of self-concept and social identity derived from the knowledge and value of group membership. Identifying as part of a group and a sense of belonging and pride are associated with higher levels of psychological well-being and higher self-esteem among minority adolescents and young adults in ethnically diverse settings. Minority adolescents and young adults who have high levels of RI appear to be able to resist or delay substance use initiation, have a more negative attitude toward drugs, and are more likely to perceive substance use as being nonnormative, or atypical, among their racial group than are youths who have low levels of RI (Stock and others, 2013a).

People in certain groups also may be at higher or lower risk because of the way that they metabolize alcohol. One study of Native Americans, for instance, found that they are less sensitive to the intoxicating effects of alcohol (Wall, Garcia-Andrade, Thomasson, Carr, & Ehlers, 1997). People who “hold their liquor” in this way may therefore lack (or can ignore more easily) warning signals that ordinarily make people stop drinking. Asian-Americans, on the other hand, may be less prone to alcohol abuse because they have genetically lower levels of aldehyde dehydrogenase, an enzyme used by the body to metabolize alcohol (Asakage and others, 2007). Without this enzyme, toxic substances build up more quickly after a person drinks alcohol, and they cause flushing, dizziness, and nausea.

Several studies suggest that teen drinking is especially damaging to the brain. Although researchers once thought that the brain is fully developed by age 16 or 17, significant neurological development continues at least until age 21. Heavy drinking at a young age may impair that development.
The Physical Effects of Alcohol Consumption

Alcohol affects all parts of the body. At the most basic level, because cell membranes are permeable to alcohol, alcohol enters the cells and disrupts intracellular communication. Alcohol also affects genes that regulate cell functions such as the synthesis of dopamine, norepinephrine, and other important neurotransmitters.

The craving that some people develop for alcohol, the adverse reactions that occur during withdrawal, and the high rate of relapse are all due to biochemical changes in the brain brought on by heavy, long-term use of alcohol. Prolonged heavy drinking can cause the brain to shrink, especially in women (Mayo Clinic, 2006). Binge drinking inhibits neurogenesis, the process by which neurons are generated, as well as the formation of new synaptic connections (Crews, He, & Hodge, 2007). In addition, alcohol abuse may interfere with the body’s absorption of thiamin, one of the B vitamins. The absence of thiamin may contribute to Korsakoff’s syndrome, a neurological disorder most often caused by alcohol abuse, and characterized by extreme difficulty with memory.

Alcohol has major effects on the hippocampus, a brain area associated with learning, memory, emotional regulation, sensory processing, appetite, and stress (see Chapter 3). It does so by inhibiting neurotransmitters that are strongly associated with emotional behavior and cravings. Dopamine transmission, in particular, is strongly associated with the rewarding properties of alcohol, nicotine, opiates, and cocaine.

Chronic alcohol abuse weakens the immune system, damages cellular DNA, interferes with normal endocrine system development, and disrupts the secretion of growth hormone, which may cause a problematic variety of other endocrine changes. Alcohol abuse has been linked to decreased testosterone levels in men, leading to impotence and decreased fertility. In women, menstrual disturbances, spontaneous abortions, and miscarriages increase with the level of alcohol consumption. Alcohol also may decrease estrogen levels in women, which may partly explain the association between alcohol use and increased risk of breast cancer (Allen, 2009).

Alcohol promotes the formation of fat deposits on heart muscle, which lowers the efficiency of the heart and contributes to cardiovascular disease. It also increases heart rate and causes blood vessels in the skin to dilate, resulting in a loss of body heat. Chronic alcohol abuse may increase blood pressure and serum cholesterol levels and accelerate the development of atherosclerotic lesions in coronary arteries. Although women experience alcohol-related heart damage at lower levels of consumption than men, both men and women who abuse alcohol are equally likely to suffer a fatal heart attack before age 55 (MMWR, 2004).

Excessive use of alcohol contributes to stomach inflammation and the formation of gastrointestinal ulcers. Severe inflammation of the liver (hepatitis) and the replacement of normal liver cells by fibrous tissue (cirrhosis) are two common chronic diseases caused by alcohol abuse. Chronic liver disease and cirrhosis cause approximately 36,000 deaths each year in the United States (CDC, 2015c).

Alcohol freely crosses the placenta of pregnant women, making alcohol a potent teratogen. Alcohol levels in a developing fetus quickly match those of the drinking mother. Pregnant women who drink during critical stages of fetal development place their infants at risk of developing fetal alcohol syndrome (FAS). FAS causes severe birth defects, including low intelligence; microcephaly (small brain); intellectual disability; delayed body growth; facial abnormalities such as malformed eyes, ears, nose, and cheekbones; and congenital heart defects.

The behavioral, psychological, and social effects of alcohol abuse are just as dangerous as its physical effects, as the next section will explain.
Psychosocial Consequences of Alcohol Use

As blood alcohol level initially rises, many drinkers feel cheerful, self-confident, and more sexually responsive. As levels continue to rise, however, higher-order cognitive functions are disrupted. Furthermore, alcohol impairs judgment and facilitates urges that otherwise might be resisted (see Chapter 6). This alcohol-induced sense of confidence and freedom from social constraints is known as behavioral disinhibition, which often results in increased aggressiveness, risk taking, or other behaviors the individual would normally avoid. When drinking, for example, both women and men are more disposed to casual sex, unprotected sex, and other high-risk behaviors (Ebel-Lam, MacDonald, Zanna, & Fong, 2009).

Alcohol also makes it difficult for drinkers to interpret complex or ambiguous stimuli; that is, they find it harder to attend to multiple cues and easier to focus on only the most salient ones (Chermack & Giancola, 1997). This alcohol myopia (nearsightedness) was demonstrated by Antonia Abbey and her colleagues (Abbey, Parkhill, Buck, & Saenz, 2000), who invited unacquainted college students (88 male--female pairs) to converse for 15 minutes after consuming either alcoholic or nonalcoholic drinks.

Both men and women who drank alcohol perceived their partners and themselves as behaving more sexually and in a more disinhibited way than did those who did not drink. However, the effects also depended on the type of cue being evaluated. Intoxicated participants exaggerated the meaning of dating cues and ignored more ambiguous signals that were possibly indicating friendliness or sociability, rather than sexual interest. Thus, alcohol seems to make it even easier for people to concentrate on salient cues that fit their current beliefs (or hopes) and disregard more ambiguous cues that do not. Some researchers believe that these cognitive and perceptual changes are the very basis of alcohol’s addictive capacity.

These results have implications for college prevention programs. Knowing that intoxication is dangerous does not necessarily keep students from drinking heavily and subsequently engaging in risky behaviors. The challenge of health psychology is to make students take these risks seriously, rather than feeling that they are invulnerable.

Alcohol-induced cognitive impairments are especially destructive during adolescence, perhaps because even low doses can impair the judgment of teens who are already distracted by the ongoing psychological, physiological, and social challenges of puberty. Hundreds of research studies have revealed the link between alcohol and risky sex—in particular, that students who use alcohol tend to have more sexual partners, to be less likely to use condoms, and to have more sexually transmitted infections and unwanted pregnancies than their nondrinking counterparts (Cooper, 2006). Frequent drinkers are also absent from school nearly four times more often than nondrinkers, more likely to ride in a car with a driver who has been drinking, and nearly three times as likely to engage in antisocial behaviors such as stealing and vandalizing property (Lammers, Ireland, Resnick, & Blum, 2000).

Alcohol abuse has been associated with a variety of other social problems, including difficulties in interpersonal relationships and various types of violence, including homicides, assault, robbery, suicides, and spousal abuse (Rogers, Boardman, Pendergast, & Lawrence, 2015; Davis, Norris, George, Martell, & Heiman, 2006). Half of all people convicted of rape or sexual assault were drinking before the commission of their crimes. Drinking also increases the chances of being a victim of crime. Approximately 72 percent of rapes committed on college campuses occur when victims are so intoxicated that they are unable to consent or refuse (Wechsler & Nelson, 2008).

Alcohol contributes to violence not only by loosening restraints due to behavioral disinhibition, but also by increasing a person’s sensitivity to pain and frustration. In addition, brain imaging studies show that repeated heavy use of alcohol, stimulants, and
other drugs disrupts frontal lobe activity, which impairs decision making and planning and lowers a person’s normal threshold for violence.

**Alcohol Use Disorder**

For most adults, moderate alcohol use—no more than two drinks a day for men and one for women and older people—is relatively harmless. (A “drink” means 1.5 ounces of spirits, 5 ounces of wine, or 12 ounces of beer, all of which contain 0.5 ounces of alcohol.) Moderate use, however, is at one end of a range of drinking, and more than 18 million people in the United States have an **alcohol use disorder (AUD)** (NIAAA, 2013). Under the newest edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5), a person who meets 2 or more of the 11 problem drinking criteria listed in Table 9.3 would be diagnosed with an AUD.

Various factors have been implicated in explaining why certain people are more likely than others to abuse alcohol. No single factor or influence, however, completely explains the origins of AUD.

**Genes and Alcoholism**

There is no single gene for alcoholism, but genes and alleles that make alcoholism more likely have been found on every chromosome except the Y (Epps and Holt, 2011). Some people inherit a greater tolerance for the aversive effects of alcohol, as well as a genetically

| TABLE 9.3  
Alcohol Use Disorder (based on DSM-5 Criteria)  |
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<td><strong>In the past year, have you:</strong></td>
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<tr>
<td><strong>1</strong> Had times when you ended up drinking more, or longer, than you intended?</td>
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<tr>
<td><strong>2</strong> More than once wanted to cut down or stop drinking, or tried to, but couldn’t?</td>
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<tr>
<td><strong>3</strong> Spent a lot of time drinking? Or being sick or getting over other aftereffects?</td>
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<td><strong>4</strong> Wanted a drink so badly you couldn’t think of anything else?</td>
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<td><strong>5</strong> Found that drinking—or being sick from drinking—often interfered with taking care of your home or family? Or caused job troubles? Or school problems?</td>
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<td><strong>6</strong> Continued to drink even though it was causing trouble with your family or friends?</td>
</tr>
<tr>
<td><strong>7</strong> Given up or cut back on activities that were important or interesting to you, or gave you pleasure, in order to drink?</td>
</tr>
<tr>
<td><strong>8</strong> More than once gotten into situations while or after drinking that increased your chances of getting hurt (such as driving, swimming, using machinery, walking in a dangerous area, or having unsafe sex)?</td>
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<tr>
<td><strong>9</strong> Continued to drink even though it was making you feel depressed or anxious or adding to another health problem? Or after having had a memory blackout?</td>
</tr>
<tr>
<td><strong>10</strong> Had to drink much more than you once did to get the effect you want? Or found that your usual number of drinks had much less effect than before?</td>
</tr>
<tr>
<td><strong>11</strong> Found that when the effects of alcohol were wearing off, you had withdrawal symptoms, such as trouble sleeping, shakiness, restlessness, nausea, sweating, a racing heart, or a seizure? Or sensed things that were not there?</td>
</tr>
</tbody>
</table>

greater sensitivity to the pleasurable effects. Both tendencies may be factors in early excessive drinking, leading to dependence. Consider the evidence:

- When either the mother or father of a male child is alcohol-dependent, that child is significantly more likely to later abuse alcohol himself (Ball, 2008). In fact, for males, alcoholism in a first-degree relative is the single best predictor of alcoholism (Plomin, King, Mainous, & Geesey, 2001).
- Identical twins have twice the concordance rate (76 percent) of fraternal twins for AUD. This is true whether the twins were raised together or apart, and whether they grew up in the homes of their biological parents or with adoptive parents (Ball, 2008).
- The personalities of those most likely to abuse alcohol have several common traits, each of which is, at least in part, genetically determined: a quick temper, impulsiveness, intolerance of frustration, vulnerability to depression, and a general attraction to excitement (MacGregor and others, 2009).

Alcohol, Temperament, and Personality
Research studies that link temperament and personality to alcohol dependence provide another clear indication of the interaction of nature and nurture. Researchers no longer attempt to identify a single “alcoholic personality,” focusing instead on specific personality traits that appear to be linked to alcohol dependence. One such trait is a temperament that includes attraction to excitement and intolerance of frustration. A second is behavioral undercontrol, characterized by aggressiveness, unconventionality, overactivity, and impulsive behavior. A third is negative emotionality, which is characterized by depression and anxiety. Marked by such traits, high-delinquent teens show consistently elevated numbers of alcohol-related problems (Allen and Gabbay, 2013).

Alcohol Expectancy Effects
As is true of all psychoactive drugs, alcohol’s impact depends not only on the dose but also on the circumstances under which the drug is taken—the user’s personality, mood, and alcohol expectancy effects. People who believe they have consumed alcohol behave just like those who have imbibed, whether or not they have (Heinz, de Wit, Lilje, & Kassel, 2013).

Several studies have also found that adolescents’ and young adults’ beliefs about their peers’ alcohol use and attitudes predicted their own alcohol use. Those who are certain that many of their friends drink regularly—and enjoy doing so—are themselves more likely to begin using alcohol (Park, Klein, Smith, & Martell, 2009). As another example, people who believe that alcohol promotes sexual arousal become more responsive to sexual stimuli if they believe they have been drinking (Friedman, McCarthy, Förster, & Denzler, 2005).

Treatment and Prevention of Alcohol Use Disorders
Although some people with AUDs do recover on their own, some cycle in and out of alcohol problems throughout their lives. Those who seek treatment generally receive outpatient rather than inpatient care. The treatments generally involve the use of drugs or therapy, or some combination of the two.

Factors that influence the willingness of a person to enter treatment for an AUD include gender, age, marital status, and ethnicity. Among women, factors that predict entry into treatment include being older and unmarried and having a lower level of

behavioral undercontrol. A general personality syndrome linked to alcohol dependence and characterized by aggressiveness, unconventionality, and impulsiveness; also called deviance proneness.
	negative emotionality. A state of alcohol abuse characterized by depression and anxiety.

alcohol expectancy effects. The effects of an individual’s beliefs about how alcohol affects behavior.

Scott Weiland, former lead singer of Stone Temple Pilots, who overdosed in 2015.
education, employment, and income. For men, factors that predict entry include having experienced alcohol-related social consequences, being older, and belonging to an ethnic minority. Although evaluations of the effectiveness of self-help groups are limited, drinking-related beliefs, readiness and motivation to change, and social support for abstinence are important predictors of the success or failure of treatment.

Drug Treatment
Researchers working to understand the physiological mechanisms by which alcohol affects the brain have uncovered a number of pharmacological treatments for AUDs. Medications include detoxification agents to manage alcohol withdrawal, alcohol-sensitizing agents to deter future drinking, and anticraving agents to reduce the risks of relapse.

As noted, many people with an AUD also suffer from clinical depression. Antidepressants that increase serotonin levels are used sometimes to treat those in the early stages of abstinence from alcohol. The best known of these is fluoxetine (Prozac). Some researchers believe that deficiencies of serotonin may cause alcohol craving (Polina, Contini, Hutz, & Bau, 2009). Other researchers have focused instead on drug treatments that block the release of dopamine, which appears to decrease the reward properties of alcohol (Thompson, 2000).

Another approach involves the drug naltrexone (Revia), which binds to opiate receptors in the brain, prevents their activation, and also decreases the reward that comes from consuming alcohol. A number of studies have found that patients who received naltrexone as part of their treatment experienced less craving than patients who received a placebo, and they were more successful in maintaining their abstinence (Snyder & Bowers, 2008). For instance, the COMBINE project evaluated the effectiveness of naltrexone versus placebo for over 1383 recently alcohol-abstinent volunteers. Over the 16-week duration of the study, different groups received either naltrexone or the placebo alone, or in combination with CBT. Participants who received naltrexone combined with CBT remained abstinent longer than those who received placebos with or without CBT (Kreibel, 2010).

Researchers generally agree that treatment of AUDs is more successful when drugs are combined with behavioral and psychological therapy. One early technique, aversion therapy, is associated with a nauseating drug such as disulfiram (Antabuse) with alcohol. Although the drug does not reduce cravings for alcohol, if the patient takes a single drink within several days of ingesting Antabuse, a variety of unpleasant effects occur, including nausea, sweating, racing heart rate, severe headaches, and dizziness (NIAAA, 2013). Drugs like Antabuse are designed to trigger a conditioned aversion to alcohol. When taken daily, Antabuse can result in total abstinence.

But patient adherence to Antabuse is a major problem, so some therapists prefer to conduct aversion therapy in a controlled clinical setting. The client drinks alcohol and then takes an emetic drug, which induces vomiting. Because the interval between the drink and the emetic drug is carefully timed, the latter functions as an unconditioned stimulus and becomes associated with the taste, smell, and act of taking a drink of alcohol. These stimuli thus become conditioned stimuli and trigger the unpleasant reaction of nausea.

Relapse Prevention Programs
Because of the unusually high rate of relapse in alcohol dependence (roughly 60 percent a year following treatment), many treatments, while helping the person to remain alcohol-free, focus on enabling the person to deal with situations that tempt relapse. Many relapse prevention programs emphasize gaining control over situations that may precipitate a return to drinking.

One form of relapse prevention is based on the gradual extinction of drinking triggers. Treatments have been developed in which drinkers are exposed repeatedly to alcohol-related stimuli, such as the aroma of their favorite drink, but they are not allowed to drink. The patients’ initially powerful, conditioned physical and psychological responses diminish with repeated exposure over a number of sessions (Witteman and others, 2015).
Many relapse prevention programs also incorporate coping and social-skills training, which helps “inoculate” drinkers by teaching specific strategies for coping with high-risk situations without the help of alcohol. Inoculation focuses on improving the person’s assertiveness, listening skills, and ability to give and receive compliments and criticism, as well as on enhancing close relationships (Foxhall, 2001). In addition, the recovered drinker is taught skills that permit him or her to abstain in drinking situations. Drink refusal training entails the modeling and rehearsal of skills needed to turn down offers to drink.

**Controlled Drinking**

Before 1970, virtually all interventions for alcohol dependency focused on total abstinence. Then several research studies reported that a small percentage of recovered alcoholics were able to drink moderately without relapsing into problem drinking. Since that time, the issue has been highly controversial. It does appear that a small percentage of problem drinkers can become moderate drinkers, particularly those who are young, employed, and live in supportive and stable environments (Dawson and others, 2005). Although many intervention programs continue to insist upon total abstinence, they typically have very high dropout rates. For a small sector of problem drinkers, drinking in moderation may be a more realistic social goal than total abstinence. Some intervention programs teach controlled drinking skills to improve the individual’s personal control over drinking. One technique, placebo drinking, involves consuming nonalcoholic beverages in place of, or in alternation with, alcohol-containing ones. Although controlled drinking treatment programs remain controversial, they have gained more acceptance in Europe than in North America, Japan, and other countries where experts are more likely to promote abstinence (Higuchi, Maesato, Yoshimura, & Matsushita, 2014).

**Self-Help Groups**

One of the most widely accepted nonmedical efforts to deal with alcohol dependence is Alcoholics Anonymous (AA). Founded in 1935, AA’s 12-step approach suggests calling on a (not necessarily religious) higher power to help battle what is viewed as an incurable disease. Its theory is that “once an alcoholic, always an alcoholic,” and it opposes “controlled drinking” and disagrees entirely with the belief that alcoholics can be reformed into moderate, responsible drinkers. AA counts more than 2 million members worldwide.

Self-help groups such as AA generally involve group discussions of members’ experiences in recovering from alcohol abuse. Members benefit by connecting with a new, nondrinking network and sharing their fears and concerns about relapse. Another self-help group, Rational Recovery, offers a nonspiritual alternative to treating alcohol dependence.

**Preventing Alcohol Problems**

Alcohol prevention researchers target individual drinkers, as well as the social environments in which drinking occurs. Preventive treatments therefore aim to change attitudes about drinking, strengthen coping skills, and restructure environments to reduce the risk of alcohol-related problems.

Prevention programs are most effective when they target children and adolescents before they have succumbed to the habit. The efforts that have proved at least partly effective include strict enforcement of drunk-driving laws, higher prices of alcohol and cigarettes due to taxes on these items, and harsher punishments for those who sell (or make available) alcohol and cigarettes to minors, as well as classes that inform parents of the hazards of various drugs, help to improve parent–child communication, and/or realistically delineate the potential hazards of drug use.

Several primary prevention programs, including the Alcohol Misuse Prevention Study (AMPS), are based on correcting faulty reasoning about peers’ drug use and improving
social skills in targeted groups. The AMPS was designed to help preadolescent students resist social pressures that lead toward alcohol consumption. For instance, role-playing exercises allow students to practice declining alcohol, marijuana, and other drug offers in various social situations. Should students actually encounter such situations, they will have developed behavioral and cognitive scripts for declining the drug offer.

Realistically, however, health psychologists recognize that so long as drugs are available and are not perceived as serious threats to health, many young people will try them, and many will eventually abuse them. Following this line of reasoning, one strategy is to delay the young person's experimentation as long as possible. Doing so will increase the odds that he or she is informed realistically about the hazards of the drug(s) in question and has the cognitive maturity to avoid the faulty reasoning that often leads to drug abuse. The younger a person is when he or she starts drinking, for example, the more likely that he or she will be to abuse or become dependent on alcohol. One study sponsored by the National Institutes of Health (NIH) found that people who began drinking before they turned 15 were four times more likely to become alcohol abusers than were those who started drinking at the legal age of 21. For every year that drinking alcohol is delayed, the risk of becoming alcohol-dependent decreases by 14 percent.

Tobacco Use and Abuse

Along with caffeine and alcohol, nicotine is one of the three most widely used psychoactive drugs. Native to the New World, the tobacco plant is first represented in history on a Mayan stone carving dated from around 600 to 900 C.E., and tobacco smoking is first mentioned in Christopher Columbus’s log books from his legendary voyage of 1492.

Prevalence of Smoking

Cigarette smoking in the United States peaked in the early 1960s, when half of all adult men and one-third of adult women smoked. From the late 1960s until the mid-1990s, the number of U.S. smokers declined steadily—to about 25 percent of all adults (Grunberg, Brown, & Klein, 1997). However, the decline was not evenly distributed, with most of the decrease occurring among upper socioeconomic status (SES) groups and men. Lower-SES individuals continued to smoke, and the prevalence of smoking among women increased sharply. Today, about 17 percent of adults in the United States smoke. Smoking is most prevalent among American Indian/Alaska natives (26.1 percent), followed by white adults 18 to 24 years of age (19.4 percent), African-American adults (18.3 percent), Hispanic-American adults (12.1 percent), and Asian-American adults (9.6 percent) (CDC, 2015b).

Figure 9.6 shows that socioeconomic status also predicts smoking rates (CDC, 2015b). Smoking rates are highest among adults who have a General Education Development (GED) diploma (41.4 percent) and those with 9 to 11 years of education (24.2 percent). Smoking is least prevalent among those who have an undergraduate degree (9.1 percent) or graduate degree (5.6 percent).
Data from the Monitoring the Future study indicate that cigarette smoking among youth in the United States is now at the lowest level recorded in the 40-year history of the survey (MTF, 2015). Overall increases in perceived risk and disapproval of smoking appear to have contributed to the downturn in cigarette use. Despite this good news, use of electronic cigarettes (e-cigarettes) has risen sharply worldwide, as I’ll discuss later in this chapter.

Although rates continue to decline in some parts of the world, they are skyrocketing in developing countries such as Kenya and Zimbabwe, causing the World Health Organization (WHO) to predict that by the year 2025, 7 out of every 10 tobacco-related deaths will occur in developing countries, where many people are still uninformed about the dangers of smoking (WHO, 2000a).

Physical Effects of Smoking

Cigarette smoking is the single most preventable cause of illness, disability, and premature death in this country and in much of the world. Worldwide, tobacco use causes more than 5 million deaths per year. In the United States, cigarette smoking is responsible for one out of every five deaths—that’s more than the combined number of deaths from murders, suicides, AIDS, automobile accidents, alcohol and other drug abuse, and fires (WHO, 2008). Because each cigarette smoked reduces a person’s life expectancy by an estimated 14 minutes, an adult who has smoked two packs of cigarettes a day (40 cigarettes) for 20 years can expect to lose about 8 years of life.

Each time a person lights up, 4000 different chemical compounds are released. It is these chemicals that provide pleasure and energy, as well as disease and death. For example, the nicotine in cigarette smoke activates specific neural receptors, which in turn causes an increase in heart rate and blood pressure and the constriction of arteries, all of which contribute to the development of cardiovascular disease. The presence of nicotine also causes serum cholesterol levels to rise, hastening the formation of artery-blocking lesions.

Cigarette smoke leads to bronchial congestion by increasing the production of mucus in the throat and lungs while simultaneously damaging the hairlike cilia that line the respiratory tract. This leads to higher-than-normal incidence rates of bronchitis, emphysema, and respiratory infections.

The link between smoking and cancer is no longer a matter of debate. Benzo[a]pyrene (BPDE), a chemical in cigarette smoke, has been identified as a causative agent in lung cancer (Denissenko, Pao, Tang, & Pfeifer, 1996). BPDE damages a cancer suppressor gene, causing a mutation of lung tissue. Smoking is also a significant factor in cancers of the mouth, larynx, stomach, pancreas, esophagus, kidney, bladder, and cervix (CDC, 2008).

Given the same lifetime exposure to tobacco smoke, the risk for developing lung cancer is 20 to 70 percent higher in women than men at every level of exposure, indicating that women are more susceptible to the carcinogens in tobacco. Women who smoke during pregnancy are also more likely to miscarry or to have low-birth-weight infants and infants who die from sudden infant death syndrome (CDC, 2008). Because cigarette smoke reduces the delivery of oxygen to the developing child’s brain, the resulting fetal hypoxia can cause irreversible intellectual damage. Schoolchildren whose mothers smoked during pregnancy have lower IQs and an increased prevalence of attention deficit hyperactivity disorder (ADHD) (Milberger, Biederman, Faraone, Chen, & Jones, 1996).
Smoking Effects Disguised as Aging

Health experts are discovering that a number of disorders once believed to be the normal consequences of aging are, in fact, caused by long-term smoking and other behavioral pathogens. For example, some of the mental decline observed among elderly persons may be caused by tobacco-related bleeding in the brain (“silent strokes”) that goes unnoticed. A meta-analysis of four European studies of 9223 people aged 65 and older compared smokers, nonsmokers, and former smokers once, and then again two years later, on short-term memory, attention, and simple mathematical calculations. All three groups showed a decline in cognitive performance over the two-year period, but the decline was by far the greatest among smokers (Launer & Kalmijn, 1998). In another meta-analysis of 19 prospective studies, current smokers had both an increased risk of dementia, including Alzheimer’s disease and vascular dementia, and greater declines in mental-state testing compared to participants who had never smoked. These findings suggest that cigarette smoke has neurotoxic effects that are associated with an increased risk of dementia (Anstey, von Sanden, Salim, & O’Kearney, 2007).

Secondhand Smoke

The hazards of smoking extend beyond the direct risks to the smoker. Secondhand smoke contains an even higher concentration of many carcinogens than smoke inhaled directly from a cigarette. According to the Centers for Disease Control and Prevention (CDC), nearly 9 out of 10 nonsmoking Americans are exposed to environmental tobacco smoke (ETS). The study reported measurable levels of cotinine (a chemical that the body metabolizes from nicotine) in the blood of 88 percent of the nonsmokers. It is estimated that 49,000 tobacco-related deaths annually in the United States are the result of secondhand smoke exposure (CDC, 2008). Female nonsmokers whose husbands smoke, for instance, stand a 1.32 times greater chance of developing lung cancer than do nonsmoking wives of nonsmoking husbands. Exposure to ETS is also recognized as an independent risk factor for cardiovascular disease (Torpy, Cassio, & Glass, 2005). Children who live with smokers have a significantly higher prevalence of pneumonia, ear and nasal infections, asthma, and the skin disorder eczema. As adults, they also have an increased risk of chronic illness and sickness-related work absences (Eriksen, 2004).

Why Do People Smoke?

To understand why people smoke, we need to consider each of the major stages of smoking behavior: initiation, maintenance, cessation, and relapse (Grunberg, Brown, & Klein, 1997).

Initiation

Initiation of drug use often occurs through social contacts. Initial use of many psychoactive drugs, with the exception of cocaine and amphetamines, is often unpleasant. As a result, a period of experimentation typically precedes the development of regular drug use, thus suggesting that factors other than physical effects are important in the initiation and maintenance of drug use until dependence develops.

Advertising is a powerful influence, and the tobacco industry spends more than $13 billion per year on it in the United States alone. From 1987 to 1997, the R.J. Reynolds company used a cartoon character named Joe Camel to advertise Camel cigarettes. A 1991 study published by the Journal of the American Medical Association showed that children as young as 5 and 6 years of age recognized the character (Campaign for Tobacco-Free Kids, 2006).

In a clever twist of this advertising campaign, Sonia Duffy and Dee Burton showed kindergarten through twelfth-grade students two currently used antismoking messages:

A study in the American Journal of Public Health showed that adolescents who owned a tobacco promotional item and named a cigarette brand whose advertising attracted their attention were twice as likely to become established smokers as those who did neither.
“Smoking kills” and “Smoking causes lung cancer, heart disease, emphysema, and may complicate pregnancy.” The messages were either plain, printed messages or featured a Joe Camel–like cartoon character. All of the cartoon messages received higher ratings of importance and believability than the plain ones (cited in Azar, 1999).

Role modeling and peer influence also lead many teenagers to start smoking. Celebrities who smoke create the image that smoking is linked with success, beauty, and even sexual arousal. Image, smoking among friends, relaxation, and pleasure are most often cited as reasons teens begin smoking (Soldz & Cui, 2002). Having parents, older siblings, and friends who smoke is also predictive of teen smoking (Rodriguez, Romer, & Audrain-McGovern, 2007). In addition, low self-esteem, social isolation, and feelings of anger or depression all increase the likelihood of smoking (Repetto, Caldwell, & Zimmerman, 2005). Among adolescents whose parents and close friends do not smoke, smoking is rare.

The college years are a time when many young adults begin to smoke regularly. A four-year national study identified several personal and environmental factors as important predictors of smoking in college (Choi, Harris, Okuyemi, & Ahluwalia, 2003). Students who were more likely to begin smoking were those who did not like school as much and were more rebellious. High school students who had tried smoking were more likely to become regular smokers if they thought that their college peers approved of smoking and if they believed that experimentation with smoking was not dangerous. Finally, the longer a student avoided smoking, the less likely he or she was to experiment in college. Not surprisingly, students who lived in smoke-free dormitories were less likely to smoke than those who lived where smoking was permitted. The results of this study suggest that interventions that reinforce the message that nonsmoking is the norm and that increase access to smoke-free environments discourage initiation of smoking among college students.

In light of the evidence linking social influences with the initiation of smoking, the U.S. surgeon general has concluded that situational factors are more important than personality factors in explaining why people start smoking. Nevertheless, a number of vulnerability factors differentiate teens who are more likely to become dependent on nicotine and other psychoactive drugs. Smoking is especially prevalent among those who feel less competent and less in control of their future and who perceive a lack of social support (Camp, Klesges, & Relyea, 1993). This is particularly true among people of lower SES, who believe that their personal life and health can only be slightly controlled by their own behavior (Droomers, Schrijvers, & Mackenbach, 2002). In addition, rebelliousness, a strong need for independence, and perceptions of benefits such as weight control, increased alertness, and stress management are also linked to smoking initiation. Teenagers who smoke are also more likely to feel alienated from school, engage in antisocial behavior, have poor physical health, and feel depressed (Kandel & Davies, 1996). They also tend to spend more time in passive activities such as watching television and are more likely to be living with a single parent (Soldz & Cui, 2002).

**Maintenance**

Once a person has begun to smoke, a variety of biological, psychological, and social variables contribute to making it difficult for him or her to abstain.
Heavy smokers are physically dependent on nicotine, which has powerful properties as a reinforcer. Nicotine stimulates the sympathetic nervous system and causes the release of catecholamines, serotonin, corticosteroids, and pituitary hormones (Grunberg, Faraday, & Rahman, 2001). In addition, nicotine induces relaxation in the skeletal muscles and stimulates dopamine release in the brain's reward system (Nowak, 1994).

Stanley Schachter and his colleagues (1977) first advanced the idea of the nicotine-titration model, suggesting that long-term smokers attempt to maintain a constant level of nicotine in their bloodstream. Schachter discovered that smokers smoke roughly the same amount day after day. When they are unknowingly forced to switch to lower-nicotine brands, they compensate by smoking more cigarettes, inhaling more deeply, and taking more puffs (Schachter, 1978). This compensatory smoking behavior has been confirmed in more recent studies and is generally considered to be one of many factors in how nicotine use promotes dependence and contributes to disease (U.S. Department of Health and Human Services [USDHHS], 2010).

Evidence of a genetic component in explaining why people continue to smoke comes from both twin and adoption studies, which estimate heritability for smoking to be as high as 60 percent (Heath & Madden, 1995; Munafo & Johnstone, 2008). Smokers and nonsmokers also appear to differ in a gene for a dopamine transporter—a protein that "vacuums up" dopamine after it has been released by a neuron. Caryn Lerman and her colleagues found that people with one form of the gene (the "9-repeat allele") were less likely to be smokers than people with other forms of it. Other studies have linked the 9-repeat allele to increased levels of dopamine, indicating reduced efficiency at removing excess dopamine. In addition, former smokers are more likely than current smokers to have the 9-repeat allele and the same DRD2 dopamine receptor gene implicated in alcohol dependence, indicating that these genes may boost people's ability to quit smoking (Lerman and others, 1999).

Psychosocial factors also contribute to maintenance. Adolescents who smoke often believe that their behavior is only temporary. When asked, they often report they will no longer be smoking in five years and that the long-term consequences of tobacco will not affect them. Adolescents are also oriented toward the present, so warnings of the long-term health hazards of cigarette smoking generally are not sufficient to deter them from smoking.

For many smokers, coping with stress is a key psychological factor in their habit. Schachter (1978) discovered that nicotine metabolism varies with the smoker's level of stress, providing a physiological explanation for why smokers tend to smoke more when anxious. When a smoker feels stressed, more nicotine is cleared from the body unmetabolized, forcing the smoker to smoke more to get his or her usual amount of nicotine.

Closely related to the idea of the nicotine-titration model, the affect management model proposes that smokers strive to regulate their emotional states. Accordingly, positive affect smokers are trying to increase stimulation, feel relaxed, or create some other positive emotional state. In contrast, negative affect smokers are trying to reduce anxiety, guilt, fear, or other negative emotional states. Evidence for this model comes from research showing that nicotine also affects levels of several neuroregulators, including dopamine, acetylcholine, norepinephrine, vasopressin, and endogenous opioids. Because of these effects, smoking may be used to boost mood, lower anxiety, reduce tension, increase concentration and alertness, and enhance memory temporarily.

In support of the affect management model, researchers also have uncovered a link between nicotine use and depression, naturally leading to questions of whether one causes the other or whether some third factor contributes to both (Nauert, 2008). One longitudinal study of high school students suggests that smoking and depression have a reciprocal effect, triggering a vicious cycle of smoking and negative mood (Windle & Windle, 2001). Every six months, students completed questionnaires assessing their emotional state, cigarette smoking, family dynamics, and friends' drug use. Teens who were
heavy smokers at the beginning of the 18-month study were more likely than those who smoked less to report symptoms of depression. In addition, students who had persistent symptoms of depression at the start of the study were more likely than other students to increase smoking, even when other factors were taken into consideration.

**Prevention Programs**

Because it is so difficult for ex-smokers to remain nicotine free, health psychologists have focused a great deal of energy on primary prevention. Their efforts have included educational programs in schools, public health messages, tobacco advertising bans, increasing tobacco taxes, and campaigns to ban smoking in public places. Over the past three decades, these campaigns have changed in ways that reflect the broader social changes in how smoking is viewed. In the 1970s, for instance, school-based prevention programs focused on providing information regarding the hazards of smoking. In the 1980s, programs were increasingly based on social influence models that portrayed smoking as undesirable and taught the necessary skills to resist social pressure to smoke. Most recently, smoking has been portrayed as an addictive disorder as well as a problem behavior. As a result, smoking interventions have increasingly incorporated some form of nicotine replacement therapy (see Chassin, Presson, Sherman, & Kim, 2003, for a review).

**Information Campaigns**

The most successful antismoking campaigns provide nonsmoking peer role models that change our idea of what behaviors are acceptable and valued (Azar, 1999). Kim Worden and Brian Flynn (1999) followed more than 5000 children in Vermont, New York, and Montana. Half the children participated in a school-based antismoking intervention program and also were exposed to a variety of radio and television commercials featuring nonsmoking role models. The other half only participated in the school program. Instead of focusing on the health hazards of smoking, the commercials featured teens who were enjoying life without smoking, who demonstrated how to refuse a cigarette, and who emphasized that most kids today don’t smoke and don’t approve of smoking. Four years later, children from the commercial intervention group were less likely to smoke than children who participated only in the school program.

As of September 2012, the U.S. Food and Drug Administration (FDA) mandated that cigarette packages in the United States must carry new graphic warning labels showing cancerous lesions and other impacts of smoking. The warnings cover the upper portion of the cigarette pack, both front and back. In addition, at least 20 percent of most cigarette ads must also include the approved warnings. So far, the tobacco industry has managed to delay implementation of this rule (Cohen, 2016).

Researchers have found that neuroimaging data during antismoking messages can predict behavior change, above and beyond self-reporting (Falk, Berkman, Whalen, & Lieberman, 2011). A group of 31 heavy smokers who intended to quit donned liquid crystal display (LCD) goggles and were shown 16 professionally developed TV commercials designed to help smokers quit smoking while neural activity was recorded using fMRI. The participants rated each ad on a 4-point scale, indicating the extent to which each ad promoted a sense of self-efficacy (“This ad makes me feel that I can quit”), increased intentions to quit (“This ad makes me more determined to quit”), and self-relevance (“I can relate to this ad”). One month later, the participants answered a series of questions regarding their efforts to quit smoking, and a biological verification (exhaled carbon monoxide) of their actual recent smoking. This outcome measure is important because self-reports of smoking behavior are often inaccurate due to poor recall, self-presentation concerns to appear consistent with stated intentions, and other
cognitive biases (Pierce, 2009). The researchers found a positive relationship between neural activity in the medial prefrontal cortex (MPFC) and successful quitting (increased activity in the MPFC was associated with a greater decrease in exhaled CO).

Antismoking Campaigns and Ethnic Minorities

Antismoking campaigns have been less effective with ethnic minorities, perhaps partly because tobacco companies have targeted a disproportionate amount of advertising toward minority communities, especially the African-American and Hispanic-American communities. Non-Hispanic members of multiple races (26.8 percent), American Indian/Alaska natives (26.1 percent), and European-Americans (19.4 percent) have the highest smoking rates. The rate for African-American men is slightly lower than for those others (18.3%; CDC, 2015b) but still on the high side among the major racial/ethnic groups in the United States. African-American men also have the highest rates of death due to lung cancer—six times that of European-American men (see Chapter 11).

Overall, Hispanic men smoke at about the same rate as non-Hispanic men, while Hispanic women smoke somewhat less than non-Hispanic women and Hispanic men (CDC, 2015b). Acculturation partly explains these smoking patterns. Traditional Hispanic culture frowns upon smoking in women but not in men. In an unhealthy twist, the generally less rigid American gender roles have meant that smoking rates among more acculturated Hispanic-American women in the United States are higher than those among less acculturated women. Asian-American women and men are less likely to be current smokers than any other single-race group studied (CDC, 2015b).

Increasing Aversive Consequences

Successful primary prevention programs also strive to increase the aversive consequences of smoking. For example, increasing the tax paid on cigarettes is quite effective. Consider the experience of Canadian smokers, whose cigarette tax has increased more than 700 percent since 1980. When a pack of cigarettes costs more than $5, many teenagers think twice about smoking. The tax impact is the sharpest among teenagers, who have less disposable income and are in the age group most vulnerable to smoking behavior. According to a Health Canada survey, the smoking rate among 15- to 19-year-olds dropped to 18 percent in 2003 from 28 percent in 1999 after the tobacco tax was increased by $2.50 per carton (Canadian Tobacco Use Monitoring Survey, 2004).

The price of a pack of cigarettes in the United States increased 90 percent between 1997 and 2003, which may be part of the reason the CDC reports that the percent of high school students who smoke decreased from 36 percent to 22 percent during the same time period. To counter the arguments of those who object to increasing taxation, it is worth noting that tobacco-caused health care costs increase the average American household’s federal tax bill by about $320 each year (Campaign for Tobacco-Free Kids, 2006).

Inoculation

Most effective for deterring smoking in adolescents have been “inoculation” programs that teach practical skills in resisting social pressures to smoke. Because smoking generally begins during the junior high and high school years, prevention programs are typically conducted in schools before children reach their teens.

The most successful programs are based on a social learning model that focuses on three variables: social pressure to begin smoking, media information, and anxiety. A program designed by Richard Evans (2003) used films, role-playing, and rehearsal to help young teens improve their social skills and refusal skills. In the films, same-age models were depicted encountering and resisting social pressure to smoke. The students also role-played situations, such as when someone is called “chicken” for not trying a
cigarette. The students were instructed to give responses such as “I’d be a real chicken if I smoked just to impress you.” After several sessions of “smoking inoculation” during the seventh and eighth grades, these students were only half as likely to start smoking as were those in a control group at another school, even though the parents of both groups had the same rate of smoking.

As noted in Chapter 6, multifaceted community campaigns that intervene on several fronts work better than “single-shot” campaigns. In one Midwestern county school system, two decades of antismoking campaigns have combined school intervention programs with communitywide mass media messages. The results have been gratifying. Between 1980 and 2001, among seventh- through eleventh-grade students, there has been a significant decrease in experimental and regular smoking and a shift in viewing smoking as more addictive and as having more negative social consequences (Chassin and others, 2003).

Cessation Programs

Since 1977, the American Cancer Society has sponsored the annual Great American Smokeout, in which smokers pledge to abstain from smoking for 24 hours. For many smokers, the Smokeout has been a first step in successfully quitting tobacco for good. In existence for over 20 years, “Kick Butts” is a similar program, sponsored by the Campaign for Tobacco-Free Kids, that encourages children and teenagers to avoid tobacco and attacks the image of smoking as being cool (http://kickbuttsday.org/).

Campaigns such as these, along with print and broadcast ads, no-smoking pledge drives, smoking bans, and other programs—many of which have been funded from the $226 billion tobacco settlement in 1998—appear to be working (Pierce & Gilpin, 2004). The settlement originally was between the four largest tobacco companies in the United States and the attorneys general of 46 states. In exchange for exemption from additional, private lawsuits, the tobacco industry agreed to curtail certain tobacco marketing practices and to make annual payments to the states to compensate them for some of the medical costs of caring for persons with smoking-related illnesses.

It is estimated that over 3 million deaths have been prevented as a result of people either quitting smoking or not beginning in the first place. Yet we need to continue these efforts. Each day, more than 4000 kids in the United States try their first cigarette, and 2000 other kids under 18 years of age become new, daily smokers (SAMHSA, 2009).

Smoking cessation programs generally fall into two categories: those based on an addiction model and those with cognitive behavior approaches. Programs based on an addiction model of smoking emphasize the physiological effects and habitual behavior engendered by nicotine (Henningfield, Cohen, & Pickworth, 1993). Cognitive behavior models focus on helping smokers better understand the motivation, conditioning, and other psychological processes that trigger smoking (Lando, 1986). Intervention is aimed at helping smokers develop coping skills to gain control over smoking triggers and to deal with anxiety, stress, and other emotions without smoking.

Addiction Model Treatments

A variety of pharmacological replacement therapy programs have been developed for smokers, including transdermal nicotine patches, nicotine gum, and inhalers. These nicotine replacement programs have helped millions of smokers in their efforts to quit smoking. Once available only as expensive prescription drugs, most now are available over the counter.

People who smoke continuously, day in and day out, are good candidates for transdermal nicotine patches, which have become the most common pharmacological treatment for smoking. Worn during the day, the bandage-like patches release nicotine through the
skin into the bloodstream. Users are able to reduce the daily dose gradually, in a series of steps that minimize withdrawal symptoms and help ensure success in remaining smoke-free (Fiore, Smith, Jorenby, & Baker, 1994; Wetter, Fiore, Baker, & Young, 1995).

However, nicotine patches are only moderately successful as a stand-alone treatment for smoking. After 10 years of research, abstinence rates in patients using the nicotine patch have been found to be about 1.9 times higher than those observed in patients using a placebo (Corelli & Hudmon, 2002). The effectiveness of the patch varies with the user's genotype with respect to the now-familiar dopamine D2 receptor gene. Oxford University researchers genotyped more than 750 people in 1999 and 2000. All had tried to quit smoking during an earlier clinical trial. At the eight-year mark, 12 percent of women with a particular allele of the dopamine D2 receptor gene who had received the patch had remained abstinent. Only 5 percent of women without that D2 receptor gene had maintained their nonsmoking status. Although the same gene variants are found in men, no differences in abstinence based on genotypes were noted (Yudkin and others, 2003).

Like all pharmacological treatments, the effectiveness of nicotine gum varies with the strength of the smoker's dependency on nicotine and his or her particular smoking habits. Nicotine gum appears to be most helpful for smokers who tend to smoke many cigarettes in a short period of time. It may be most effective when used as part of a comprehensive behavioral treatment program. Some researchers believe that the relief of withdrawal symptoms and cravings is largely a placebo effect, rather than a pharmacological effect of the actual nicotine in the gum. Although less effective than the nicotine patch, nicotine gum improves cessation rates by about 50 percent compared with control interventions (Davies, Willner, James, & Morgan, 2004).

Another recent intervention is the oral inhaler, a plastic tube filled with 4 milligrams of nicotine that smokers can “puff” on 2 to 10 times a day. Patients who use the nicotine inhaler are 1.7 to 3.6 times more likely to remain abstinent than patients using a placebo inhaler (Rennard and others, 2006).

A treatment option currently being investigated is bupropion (Zyban), a powerful antidepressant that may curb nicotine cravings by mimicking tobacco's ability to increase brain levels of dopamine (Lerman and others, 2004). Zyban's efficacy in treating nicotine addiction was discovered by accident. Researchers knew that depression was a common symptom of nicotine withdrawal, so they began experimenting with antidepressants to alleviate addiction rather than depression. Cessation rates in patients who use sustained-release bupropion are generally 2.1 times higher than those observed in patients receiving a placebo (Fiore, 2000). As a partial agonist for nicotine receptors, the newer prescription drug varenicline (Chantix) is even more effective than bupropion in reducing cravings for nicotine and in decreasing the pleasurable effects of tobacco use. One study of former smokers found that after one year, the rate of abstinence was 10 percent for participants who received a placebo, 15 percent for those who received bupropion, and 23 percent for those who received varenicline (Jorenby and others, 2006). Most effective is combination therapy, in which one intervention (such as the nicotine patch) provides steady levels of nicotine in the body and a second form (such as bupropion or varenicline) is used as needed to control cravings and suppress nicotine withdrawal symptoms (Corelli & Hudman, 2002; Piper and others, 2009).

### Cognitive Behavior Treatments for Smoking

Given the importance of modeling, reinforcement, and principles of learning in the development of drug abuse, health experts rely on a number of cognitive and behavioral techniques to help people quit smoking. Aversion therapy involves pairing unpleasant consequences with smoking to create an aversion to smoking. In one of the most frequently used techniques, smokers increase their usual smoking rate until the point of satiation,
an unpleasant state of “fullness.” One variation involves rapid smoking, in which a smoker periodically is asked to smoke a cigarette as fast as he or she can, which often leads to an upset stomach. Both techniques are designed to associate nausea with smoking. Aversion strategies have also used electric shock and nausea-inducing drugs. For many smokers, aversion therapy is an effective way to begin to quit.

Cognitive restructuring of health beliefs and smoking attitudes is also important in successful quitting and avoiding relapse. Research studies demonstrate that those who successfully quit smoking typically change their beliefs to see less psychological benefit and more health threat from smoking, while those who relapse may come to view smoking as having more psychological benefits and being less of a personal threat over time (Chassin and others, 2003).

Which Smoking Cessation Programs Are Effective?

There have been relatively few randomly controlled studies examining the effectiveness of smoking cessation programs for adolescents. In general, research studies support the viewpoint that younger smokers, especially those who are heavy tobacco users, are more likely to continue smoking than older smokers (Ferguson, Bauld, Chesterman, & Judge, 2005). A meta-analysis of teen smoking cessation programs revealed that the most effective programs are inexpensive, short-term interventions that include a motivational component, cognitive behavior techniques, and social influence education (Sussman, Sun & Dent, 2006). More specifically, these programs:

- enhanced the intrinsic and extrinsic motivation to quit with rewards and education designed to reduce ambivalence about quitting;
- were tailored to adolescents’ developmental needs (rather than adult programs with only superficial changes) and made intervention programs fun;
- provided social support to help teens persevere and avoid relapse; and
- showed teens how to use community resources for remaining nicotine-free.

In contrast, there is an abundance of research on adult smoking cessation efforts. These studies have found that smoking treatment programs are most effective when two or more methods are used together. For example, treatment programs that combine behavioral methods with nicotine replacement are more effective than either approach used alone (Stead, Perera, Bullen, Mant, & Lancaster, 2008). A meta-analysis of over 100 combination therapy studies concluded that smoking treatment programs that include nicotine replacement have significantly higher quit rates than those that include placebos or no nicotine replacement therapy (Silagy, Lancaster, Stead, Mant, & Fowler, 2005).

Whichever combination of techniques is used, Edward Lichtenstein and Russell Glasgow (1997) of the Oregon Research Institute argue that quitting smoking is determined by three interacting factors: motivation to quit, level of physical dependence on nicotine, and barriers to or supports in remaining smoke-free (Figure 9.7). The extent of a smoker’s physical dependence, for example, certainly will influence both readiness to quit and persistence. The presence or absence of a smoking spouse, workplace smoking bans, a child pressuring a parent to quit, and other barriers and supports also may influence motivation (Hammond, McDonald, Fong, Brown, & Cameron, 2004).

James Prochaska (1996a) suggests that many smoking cessation programs dilute their effectiveness by targeting multiple

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**FIGURE 9.7**  
Factors in Smoking Cessation  
According to this model, readiness motivation is the primary, proximal causal factor in determining whether a person makes a serious attempt to quit. Social and environmental supports or prompts, such as a workplace no-smoking policy, increases in the price of tobacco, persistent reminders from one’s child to stop smoking, or a physician’s advice, also can affect readiness motivation.  

Source: Lichtenstein, E., & Glasgow, R. E. (1997). A pragmatic framework for smoking cessation. Psychology of Addictive Behaviors, 11(2), 142-151 (Figure 1).
behaviors and failing to recognize that different smokers have different needs. Prochaska proposes, instead, that planned interventions be organized according to each smoker’s stage of quitting. His transtheoretical model outlines six stages of behavior change: (1) precontemplation, (2) contemplation, (3) preparation, (4) action, (5) maintenance, and (6) termination. Smokers in the precontemplation stage, for example, are often defensive and resistant to action-oriented programs. They often are demoralized by previous failures to quit smoking and consequently are put off by information campaigns condemning their unhealthy behavior. Historically, health experts considered such people as unmotivated or not ready for therapy. Prochaska, however, would suggest that treatment for people at this stage include reassurance that becoming a nonsmoker—like becoming a smoker in the first place—is not something that happens overnight. Rather, there are stages in its development, and many smokers who attempt to quit are not successful the first time.

The stage approach has several advantages over traditional, nonstage interventions. First, it generates a much higher rate of participation. When free smoking clinics are provided by health maintenance organizations (HMOs), only about 1 percent of subscribers participate. Using the stage approach in two home-based interventions involving 5000 smokers each, Prochaska and his colleagues (2006) generated remarkably high participation rates of 82 to 85 percent. A second strength of stage-based interventions is a dramatic improvement in the number of participants who complete the treatment (retention rate). Other researchers have also reported high retention rates when treatment is individualized according to a stage approach (Aveyard, Johnson, Fillingham, Parsons, & Murphy, 2008; Armitage & Arden, 2008).

The third advantage of stage-based approaches is the most important: Progress in remaining smoke-free is directly related to the stage participants were in at the start of the interventions. This stage effect is illustrated in Figure 9.8. It shows that smokers in the precontemplation phase displayed the smallest amount of abstinence from smoking over 18 months, whereas those in the preparation stage showed the most progress in 6-, 12-, and 18-month follow-ups. As discussed in Chapter 2, stage-based interventions proceed in a gradual series of steps, with the reasonable goal of helping smokers advance one stage at a time.

**FIGURE 9.8** Percentage of Abstinent Former Smokers by Stage of Quitting

The amount of progress that former smokers make in remaining abstinent is directly related to the stage they were in at the start of the intervention. Smokers in the precontemplation phase display the smallest amount of abstinence from smoking over 18 months, whereas those in the preparation stage show the most progress in 6-, 12-, and 18-month follow-ups.


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**Valued Life Activities and Quitting**

You may recall from Chapter 7 that I was able to quit smoking largely because it interfered with my desire to run a marathon and be identified as “a runner.” Several lines of evidence suggest that the restriction of valued activities affects smoking and other health-compromising behaviors. First, low levels of engagement in valued activities (such as swimming, cycling, and running), restriction of valued activities due to physical condition (such as injuries), and the inability to find replacements for restricted valued activities have been linked to depressed mood (Manos, Kanter, & Busch, 2010). Depressed mood, in turn, is more common among smokers and interferes with efforts to quit. Behavioral activation, a counseling treatment that focuses on increasing engagement in valued life activities through guided goal setting, has shown promise as a tool in helping smokers quit (Banducci, Long, & MacPherson, 2014).

Smoking cessation among people with physical disabilities that cause impairment in mobility has been relatively neglected by researchers, despite the fact that cigarette
smoking is more common among this group, with rates as high as 40 percent among nonelderly adults (Altman & Bernstein, 2008). Could it be that restriction of valued life activities due to physical limitations is a barrier to quitting in this population? The answer, according to the findings of a recent study of long-term smokers with physical disabilities, is “yes.” The study found that participants’ readiness to quit smoking was enhanced by an educational intervention that helped identify satisfactory replacements for their most valued restricted activity (Busch & Borrelli, 2012).

Relapse: Back to the Habit

Unfortunately, only a small percentage of people who quit remain smoke-free for very long. Attempts to quit, even within the first weeks of smoking, often fail (DiFranza, 2008). As with other addictions, smokers become dependent, and they develop tolerance. Quitting triggers withdrawal symptoms, including craving, anxiety, irritability, distractibility, and insomnia (Sayette, Loewenstein, Griffin, & Black, 2008). All it takes to relieve this unpleasant state is the negative reinforcement provided by a cigarette. Within seven seconds, the rush of nicotine causes the brain to release epinephrine, norepinephrine, dopamine, and other neurotransmitters that boost alertness and calm anxiety.

The rewards that nicotine delivers keep people smoking, even among the 8 in 10 smokers who say they’d like to quit. Of these smokers, fewer than 1 in 10 are able to do so, and as many as 80 percent of smokers who quit smoking relapse within 1 year (USDHHS, 2012). Many factors are involved in relapse, the most fundamental being the severity of withdrawal symptoms and craving. In one study of 72 long-term smokers (38 men and 34 women), 48 percent relapsed within the first week of quitting. Participants who relapsed experienced greater distress and withdrawal symptoms during the first 24 hours of nicotine abstinence (al’Absi, Hatsukama, Davis, & Wittmers, 2004). Interestingly, the researchers found that stressful experiences affect men and women who are trying to quit smoking differently. For instance, cortisol responses before and after performing a public-speaking test were stronger in the men than in the women.

Ex-smokers may experience other side effects as well, which are immediately eliminated by a return to smoking. For example, some ex-smokers gain weight (perhaps because of slower metabolism, increased preference for sweet-tasting foods, or substituting eating for smoking), have trouble sleeping, are more irritable, and find it difficult to concentrate. Unfortunately, smoking is even used as a weight-control strategy—particularly by adolescents who are also likely to use other unhealthy strategies, such as diet pills and laxatives (Jenks & Higgs, 2007).

Another factor in relapse is the strength of previously conditioned associations to smoking. Smoking behaviors, as well as nicotine’s physiological effects, become conditioned to a variety of environmental stimuli. Many ex-smokers relapse in the face of an irresistible urge (conditioned response) to smoke in certain situations or environments—for example, with that first cup of coffee in the morning or after a meal.

Because of this dismal prognosis for ex-smokers, smoking relapse has received considerable research attention in recent years. A working conference sponsored by the National Institutes of Health (NIH) took a first step in addressing the relapse problem by encouraging health experts to adopt a “stages of change” (see Figure 6.4) model in developing programs to prevent relapse. For example, rather than encouraging ex-smokers to attend an occasional follow-up session reminding them of the hazards of smoking, the NIH group encouraged training in relapse prevention strategies much earlier in the stages of quitting.

Efforts such as these are paying off, and smokers are getting the message that repeated attempts to quit can succeed (see Your Health Assets: You Can Quit Smoking—Here’s How), and success is equally likely whether they quit abruptly or gradually (USDHHS, 2012). After a year’s abstinence from nicotine, only 1 in 10 will relapse in the next year.
PART 3 | Behavior and Health

Your Health Assets
You Can Quit Smoking—Here’s How
Smokers often say, “Don’t tell me why I should quit—tell me how.” There is no one right way to quit, but four factors are key: deciding to quit, establishing a plan and setting a day to quit, developing strategies to deal with withdrawal, and maintaining your success.

Prepare the way for quitting:

- Select a specific date to quit.
- Eliminate cigarettes and ashtrays around you.
- Find replacements for cigarettes such as sugarless gum and other oral substitutes.
- Create a plan. Will you use nicotine-replacement therapy?
- Get social support: Find friends or anyone who will be a support person for you.

On your specific quitting day, do the following:

- Avoid any smoking at all!
- Keep active by walking, exercising, or doing other activities you enjoy.
- Drink lots of water and juices—staying hydrated is key.
- Begin using nicotine replacement if you have chosen to do so.
- Stay away from people who are smoking.
- Stay away from places or situations where you may be tempted to smoke.
- Attend a stop-smoking class, or follow your self-help plan.

(Hughes, 2010). Researchers will continue to study all aspects of substance abuse in an effort to help many more of us become “former users”—or, better yet, to avoid altogether the life disruption that substance abuse brings.

The Rise of e-Cigarettes and the Resurgence of Marijuana

In this final section of the chapter, we turn to two recent developments that merit special attention: the rise of electronic cigarettes and the resurgence of marijuana.

Electronic Cigarettes

Introduced in 2004, electronic, or e-cigarettes (EC) are battery-powered vaporizers that simulate smoking without burning tobacco. Consisting of a liquid-filled cartridge (e-juice) and a heating element, e-cigarettes deliver nicotine, along with flavorings and other chemicals, as an aerosol instead of smoke. The user activates the device by taking a puff or pressing a button and inhaling the atomized nicotine (vaping).
Manufacturers of e-cigarettes make the devices in many shapes, including some that look like traditional cigarettes and pipes, but also some that look like everyday items such as pens and USB flash drives. E-cigarettes also come in more than 7000 flavors, including some—such as mint, candy, and bubble gum—that likely are targeted to the tastes of younger users (Grana, Ling, Benowitz, & Glantz, 2014). This new industry has become an enormous business: Sales of e-cigarettes were about $3.5 billion in 2015 (Szabo, 2015).

In 2014, 12.6 percent of adults in the United States reported having tried e-cigarettes (see Figure 9.9), and about 3.7 percent are current users (Schoenborn & Gindi, 2015). Most EC users still smoke regular cigarettes, but a 2014 study found that more teens in the United States used e-cigarettes in the past 30 days than any other tobacco product, including traditional cigarettes (MTF, 2015).

There are several reasons e-cigarettes are a cause for concern. First, because they deliver vapor without burning tobacco, e-cigarettes may appear to be safer and less toxic than smoking conventional cigarettes (Drummond & Upson, 2014). Although they do not produce tobacco smoke, e-cigarettes still contain nicotine, which is, as we have seen, a highly addictive drug and has a wide range of harmful effects.

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**The Components of an e-Cigarette** A battery-powered vaporizer consisting of a liquid-filled cartridge and a heating element.

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### FIGURE 9.9

**E-Cigarette Usage** Men are more likely than women to have ever tried e-cigarettes but are not more likely to be current users. Younger adults are more likely than older adults to have tried e-cigarettes and to currently use e-cigarettes. Both non-Hispanic AIAN (American Indian or Alaska native) and non-Hispanic white adults are more likely than non-Hispanic black, non-Hispanic Asian, and Hispanic adults to have ever tried e-cigarettes and to be current e-cigarette users.

Another worry is that vaping exposes smokers to other harmful substances. E-cigarette vapor may contain formaldehyde and acetaldehyde—two known carcinogens—as well as toxic metal nanoparticles released from the heating element (National Institute on Drug Abuse, 2015). Harvard University researchers recently tested 51 types of flavoring used in e-liquids and found that most of them contained also diacetyl and acetoin, chemicals that may cause severe lung disease (Allen and others, 2015).

In addition to these issues, several studies suggest that e-cigarette use may encourage the use of other tobacco products (Leventhal and others, 2015). One study showed that students who had used e-cigarettes by the ninth grade were more likely than their non-vaping peers to start smoking traditional cigarettes within one year (Rigotti, 2015). That finding alone should create serious concerns about the rise of electronic cigarettes. Sadly, an even more recent Yale University study of teens who use e-cigarettes cited the low cost of the devices and the promise that vaping can help teens quit smoking as strong predictors of continued use (Barrington-Trimis and others, 2016).

The Resurgence of Marijuana

According to the World Health Organization, cannabis is the most popular recreational drug worldwide (UNODC, 2015). Unlike many other recreational drugs, however, marijuana is also used as a medicine. Since 1996, 23 states and Washington, DC, have passed laws allowing marijuana to be used for a variety of medical conditions, and 17 have reduced punishment for, or fully legalized, possession of small recreational amounts of the drug. From the 1960s until about 2010, public opinion polls showed that a majority of adults thought marijuana (or “pot”) should remain illegal. Spurred by rising claims of its benign nature and medical benefits, by 2014 more than half of all adults in the United States said they supported the legalization of marijuana (see Figure 9.10). While many states have loosened older laws regarding marijuana and others are seriously considering full legalization, marijuana use remains an offense under U.S. federal law.

FIGURE 9.10
Growing Support for Legalization of Marijuana in the United States A 2014 survey reported that 53% of Americans favor legalizing marijuana. As recently as 2006, just 32% supported marijuana legalization, while nearly twice as many (60%) were opposed.

Marijuana: Some Basics about Its Source, How It Is Used, and Its Physical Effects

Marijuana comes from the dried leaves, flowers, stems, and seeds of the hemp plant Cannabis sativa, all of which contain the mild hallucinogen THC (delta-9-tetrahydrocannabinol). A synthetic form of marijuana, called K2 or Spice, mimics THC. In addition to THC, marijuana contains more than 100 other chemical cannabinoids. Marijuana can be smoked in hand-rolled cigarettes (joints), water pipes (bongs), or emptied cigars refilled with the drug (blunts). It can also be inhaled from a vaporizer (vaping), added to food (edibles), and brewed as a tea. A dangerous new method, dabbing, involves smoking or eating THC-rich resins that have been extracted from marijuana using butane (lighter fluid), which can cause serious burns, fires, and explosions.

Marijuana has both short- and long-term effects on the brain (see Figure 9.11). When inhaled, THC quickly passes from the lungs into the bloodstream, which carries the chemical to the brain (in under 10 seconds) and organs throughout the body. THC is absorbed more slowly when eaten, and its effects are typically felt after about 30 minutes to 1 hour. In the brain, THC binds to cannabinoid receptor type 1 (CB1) proteins that ordinarily react to natural THC-like chemicals in the brain. These natural chemicals, called endocannabinoids, are involved in pain perception, memory, mood, and appetite regulation, and also play an important role in brain maturation. This brain maturation issue is of special concern to those who question the wisdom of legalizing marijuana, as I’ll point out in the next section.

THC has many effects on the brain and behavior, including alterations in mood, time perception, and sensations (for example, seeing brighter colors); and impaired thinking, problem-solving and memory. Like alcohol, marijuana also relaxes, disinhibits, and may produce a euphoric high. Unlike alcohol, which is metabolized and eliminated from the body within hours, THC can remain in the body for more than a week. As a result, regular marijuana users are less likely to experience abrupt withdrawal from the drug and may get high with smaller drug amounts than needed when they first used the drug.

The Debate over Legalization

Some people think pot is nothing less than a gift from nature: a nonaddictive drug that lifts the spirit, is safe at any dose, and has important medical applications. Others portray the drug as dangerous, addictive, and with a high potential for abuse. Who is right? Let’s take a look at some of the evidence on both sides of the argument.

Medical marijuana refers to using the plant or its extracts to treat a disease or symptom. Although the FDA has not approved marijuana as medicine, two THC-based drugs, dronabinol and nabilone, have been approved to relieve the pain and nausea caused by AIDS and

<table>
<thead>
<tr>
<th>Brain Area</th>
<th>Potential Effects of THC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nucleus accumbens</td>
<td>euphoria; altered motivation and decision making</td>
</tr>
<tr>
<td>Hypothalamus</td>
<td>altered metabolic process such as increased appetite</td>
</tr>
<tr>
<td>Hippocampus</td>
<td>impaired memory storage recall</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>impaired coordination and balance</td>
</tr>
<tr>
<td>Basal ganglia</td>
<td>impaired motor skills and learning</td>
</tr>
<tr>
<td>Cortex</td>
<td>altered consciousness, perceptual distortions</td>
</tr>
<tr>
<td>Amygdala</td>
<td>altered emotions; anxiety and panic in some cases</td>
</tr>
</tbody>
</table>

cancer, and to increase appetite in patients with AIDS wasting syndrome (Munsey, 2010). THC-based drugs may also help treat seizures and spasticity related to epilepsy and multiple sclerosis, while a related compound called *cannabidiol* (CBD) may be useful in treating autoimmune diseases, inflammation, pain, and substance use disorders (Seppa, 2010).

Advocates for legalizing marijuana (for medical or recreational use) argue that doing so may have important public health benefits, such as reducing the risk of overdoses from other drugs. They point to evidence that death rates from overdoses on prescription painkillers, heroin, and other illicit drugs average 24.8 percent lower in states with medical marijuana laws than in states without similar laws (Bachhuber and others, 2014).

Opponents of legalizing marijuana fear that wider recreational use will adversely affect public health. Case in point: driver safety. Marijuana use has been linked to increased accident risk, likely reflecting marijuana’s disruptive effects on motor coordination and time perception. One study estimated that cannabis intoxication doubles a driver’s risk of an automobile accident (Asbridge, Hayden, & Cartwright, 2012). Some states have expanded their DUI laws to include cannabis intoxication. Washington and Colorado, where marijuana use was legalized in 2012, have a 5-nanogram limit on the level of THC in a driver’s blood. Oregon doesn’t use blood testing and instead relies on police officers’ observations (Kullgren, 2014).

While most experts agree that marijuana is not nearly as addictive as alcohol, tobacco, and opioids, THC does seem to reduce the number of CB1 receptors in the brains of people who use marijuana daily. This may explain why about 1 user in 10 struggles to stop using marijuana and does show signs of becoming dependent on the drug (Hirvonen, Goodwin, & Innis, 2012). Among people who begin using pot as teens, the number showing signs of developing dependency seems to increase to about 1 in 6 (Lopez-Quintero and others, 2011): Heavy, regular users also show signs of developing tolerance for THC, as they need to increase their dose over time to experience the same effects. Some users also experience anxiety and irritability—two other features of addiction according to DSM-5—after stopping heavy use of THC (National Institute on Drug Abuse, 2016).

As to the safety of marijuana, critics of the legalization movement argue that while the drug may not be deadly, it is far from harmless. Marijuana smoke irritates the lungs and contributes to the same breathing problems that tobacco smokers experience, including more frequent lung infections and disease. THC also increases the heart rate for several hours after smoking, which may increase the risk of heart attacks. Marijuana use during pregnancy has been linked to increased risk of brain and behavior problems in babies (National Institute on Drug Abuse, 2016).

In Colorado, where marijuana laws have been loosened for both medical and recreational use, emergency room doctors, law enforcement officers, and legalization opponents have pointed to recent problems, such as rising numbers of marijuana-impaired traffic citations, as cautionary lessons for other states considering more lenient cannabis laws. Proponents of legalization note that the overall rate of crime in the state is down by 10 percent since legalization for recreational use went into effect in 2014 and say critics are cherry-picking statistics to undermine a new industry that is flourishing despite intense scrutiny (Healy, 2014).

Opponents of legalization also note that the amount of THC in marijuana has been increasing steadily over the past few decades. Marijuana legally sold in Colorado, for instance, contains three times as much THC as did plants grown 30 years ago (Mole, 2015). This greater potency may help explain the rise in emergency room visits involving marijuana. The growing popularity of edibles also increases the chance of harmful reactions. Because edibles take longer to produce a high, users may consume more to feel the effects faster, leading to dangerous results.

Perhaps the biggest concern is the potential impact on teenagers, a decreasing number of whom believe that marijuana use is risky (Johnston and others, 2014). As we have seen, the adolescent brain is still growing and refining its neural networks—processes regulated in part by the natural endocannabinoid system in the cortex. Marijuana use at
this vulnerable time may interfere with healthy brain development. Indeed, research has shown that young adults, ages 18 to 25, who use marijuana at least once a week are more likely than nonusers to have structural differences in two brain areas: the nucleus accumbens and the amygdala (Gilman, Kuster, & Breiter, 2014). Heavy use of THC for 20 years or more may also damage the corpus callosum (Rigucci and others, 2015), lead to a reduced volume of gray matter, and shrinkage of cortical areas that process memory (Filbey and others, 2014). However, other researchers are skeptical of the evidence that THC use harms the brain (Rogeberg, 2013; Weiland and others, 2015).

Marijuana use does seem to affect memory, language proficiency, and motivation—all of which are especially important during adolescence and emerging adulthood. Teens who use marijuana heavily are more likely to perform poorly in school and have other problems (Ansary & Luthar, 2009). One study reported that people who started using marijuana heavily as teenagers and had an ongoing cannabis use disorder lost an average of eight IQ points between ages 13 and 38 (Meier and others, 2012). In addition, the more often marijuana is used, especially during adolescence, the greater the risk of anxiety, depression, or addiction (Hurd, Michaelides, Miller, & Jutras-Aswad, 2014). Heavy cannabis use in early adolescence may even accelerate the onset of schizophrenia (Hill, 2015).

Although laws are changing, the biggest obstacle to truly understanding the potential risks and benefits of cannabis is the fact that research has been stifled by years of prohibition and misconceptions about the plant and its active compounds (Grayson, 2015). Major gaps in knowledge continue to exist. For example, while a recent meta-analysis of cannabinoids for medical use supported their use in treating chronic pain and muscle spasticity, other claims about the benefits of THC and CBD were less well supported (Whiting and others, 2015). Until medical marijuana is made a public health priority and more research funding is made available, these knowledge gaps are unlikely to close. In addition to further research, the effects of the recent legalization of recreational marijuana in Colorado, Washington State, and elsewhere will be instructive. The experience of those states should provide a better sense of the social and public health impacts of this policy change.

**Weigh In on Health**

Respond to each question below based on what you learned in the chapter. **(Tip: Use the items in “Summing Up” to take into account related biological, psychological, and social concerns.)**

1. A classmate questions whether a mutual friend is addicted to cocaine. What would you be able to tell the classmate about what addiction is, and how a psychoactive drug like cocaine might affect your friend?
2. You just found out that your cousin, a senior in high school, started smoking cigarettes when she was a sophomore. Now she’s addicted to smoking but wants to quit before she goes off to college. Based on what you read in this chapter, how would you advise her? What if she asked you whether e-cigarette vaping was a safe alternative to smoking cigarettes?
3. Some states have attempted to criminalize drug use during pregnancy or treat it as grounds for terminating parental rights. Should it be illegal for a pregnant woman to purchase or use alcohol or tobacco products? Why or why not?
Summing Up

Some Basic Facts
1. Drug abuse is the use of a chemical substance to the extent that it impairs the user’s well-being in any domain of health: biological, psychological, or social. During pregnancy, many drugs will cross the placenta and act as teratogens to adversely affect fetal development.

2. Drugs affect behavior by influencing the activity of neurons at their synapses. Some (agonists and, to a lesser extent, partial agonists) do so by mimicking natural neurotransmitters, others (antagonists) by blocking their action, and still others by enhancing or inhibiting the reuptake of neurotransmitters in the synapse.

3. Substance use disorder is a behavior pattern characterized by overwhelming involvement with the use of a drug despite its adverse consequences.

4. Psychoactive drugs act on the central nervous system to alter emotional and cognitive functioning. Stimulants, such as caffeine and cocaine, increase activity in the central nervous system and produce feelings of euphoria. Depressants, such as alcohol and the opiates, reduce activity in the central nervous system and produce feelings of relaxation. Hallucinogenic drugs, such as marijuana and LSD, alter perception and distort reality.

Models of Addiction
5. Biomedical models propose that dependence is a chronic disease that produces abnormal physical functioning. One aspect of these models is based on evidence that some people inherit a biological vulnerability toward dependence. The withdrawal-relief hypothesis suggests that drugs deplete dopamine and other key neurotransmitters. Another model proposes that psychoactive drugs are habit-forming because they overstimulate the brain’s dopamine reward system.

6. Reward models suggest that the pleasurable effects of psychoactive drugs provide the initial motivation for their repeated use. All major drugs of abuse overstimulate the brain’s reward system.

7. Most adolescents experiment with drugs, especially tobacco and alcohol. Age, gender, parental factors, community, and national culture each have a powerful influence on drug use in adolescence.

8. Fear of addiction and antitobacco campaigns that use scare tactics may not persuade young adolescents to avoid drug use—they may even backfire.

Alcohol Use and Abuse
9. Alcohol depresses activity in the nervous system, clouds judgment, and is linked to a variety of diseases. Alcohol is also involved in half of all traffic accidents. Genes play a role in alcohol dependence, especially in men. Psychosocial factors such as peer pressure, a difficult home environment, and tension reduction may contribute to problem drinking.

10. The prevalence of drinking varies with ethnic and cultural background. Individuals marked by behavioral undercontrol and negative emotionality are especially prone to alcohol dependence. Alcohol’s impact depends in part on the user’s personality, mood, past experiences with the drug, and expectations regarding its effects.

11. Alcohol use disorder is defined by several specific behaviors, including the need for daily use of alcohol, the inability to cut down on drinking despite repeated efforts to do so, binge drinking, loss of memory while intoxicated, and continued drinking despite known health problems.

12. Alcohol treatment usually begins with detoxification from alcohol under medical supervision. Counseling, psychotherapy, and support groups such as Alcoholics Anonymous (AA) also may help. Pharmacological treatments for alcohol dependence include aversion therapy, which triggers nausea if alcohol is consumed. Antidepressants such as Prozac may help reduce alcohol cravings.

Tobacco Use and Abuse
13. Cigarette smoking is the single most preventable cause of death in the Western world today. A stimulant that affects virtually every physical system in the body, nicotine induces powerful physical dependence and a withdrawal syndrome. Social pressures most often influence the initiation of smoking.

14. Once a person begins smoking, a variety of psychological, behavioral, social, and biological variables contribute to make it difficult to abstain from nicotine. According to the nicotine-titration model, long-term smokers may smoke to maintain a constant level of nicotine in their bodies. Smoking prevention programs that focus on refusal skills and other inoculation techniques prior to the eighth grade may be the best solution to the public health problems associated with smoking.

15. The most successful antismoking advertisements provide culturally sensitive nonsmoking peer role models that shift people’s overall image of what behaviors are “normal” and valuable within one’s peer group.

16. No single treatment has proved most effective in helping smokers quit smoking. Most programs have an extremely high relapse rate. Modern treatments for smoking deal with psychological factors through relapse prevention, and physiological factors through nicotine replacement.
The Rise of E-Cigarettes and the Resurgence of Marijuana

17. E-cigarettes are battery-powered vaporizers that simulate smoking without burning tobacco. Because they deliver vapor without burning tobacco, e-cigarettes may appear to be safer and less toxic than smoking conventional cigarettes.

18. Vaping e-cigarettes exposes smokers to other harmful substances, including formaldehyde and acetaldehyde—two known carcinogens—as well as toxic metal nanoparticles released from the heating element.

19. Spurred by rising claims of marijuana’s benign nature and new laws loosening restrictions on medical and recreational use, more than half of all adults in the United States now support legalizing the drug.

20. Marijuana comes from the hemp plant *Cannabis sativa*, which contains the mild hallucinogen THC (delta-9-tetrahydrocannabinol). THC has many effects on brain and behavior, including alterations in mood, time perception, and sensations; and impaired thinking, problem-solving and memory.

21. While most experts agree that marijuana is not nearly as addictive as many other drugs, THC may reduce the number of CB1 receptors in the brains of people who use marijuana daily, leading to tolerance and dependence.

22. Marijuana use may adversely affect memory, language proficiency, and motivation—all of which are especially important during adolescence and emerging adulthood. Heavy use of THC for 20 years or more may also damage the corpus callosum, lead to a reduced volume of gray matter and shrinkage of cortical areas that process memory.

Key Terms and Concepts to Remember

drug abuse, p. 244  
concordance rate, p. 249  
alcohol use disorder (AUD), p. 258

electronic, or e-cigarettes (EC), p. 274  
behavioral undercontrol, p. 259  
behavioral activation, p. 272

drug–brain barrier, p. 246  
gateway drug, p. 250  
negative emotionality, p. 259

teratogens, p. 246  
common liability to addiction, p. 250  
alcohol expectancy effects, p. 259

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wanting-and-liking theory, p. 251  
aversion therapy, p. 260

antagonist, p. 246  
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To accompany your textbook, you have access to a number of online resources, including quizzes for every chapter of the book, flashcards, critical thinking exercises, videos, and Check Your Health inventories. To access these resources, please visit the Straub Health Psychology LaunchPad solo at: http://www.macmillanlearning.com/launchpadsolo/straub5e/.