

Psychoactive Drugs and Drug Use/Abuse

Marijuana creates a separate brain when you are stoned (kind of like creating a new folder on your computer). Early in a stoner's smoking stature (life), the brain is infant like; simple things and ideas appear to be difficult and the stoned person has impaired movement and gets paranoid easily, etc. The more the person gets stoned, the more advanced the stoner brain becomes. It grows like a child does, and becomes more experienced until a point when it matures and skills are enhanced past the level of a normal stone-free mind. My theory of marijuana brain enhancement along with long-term research concludes that smoking marijuana can make you smarter and better at almost everything than not smoking marijuana. So, in conclusion I recommend getting stoned often because practice makes perfect. (*Anonymous marijuana user quoted at <http://www.angelfire.com/mo/rollitup/quotes.html>*)

I lived in one room in the native quarter of Tangier. I had not taken a bath in a year nor changed my clothes or removed them except to stick a needle every hour in the fibrous grey wooden flesh of terminal addiction. I never cleaned or dusted the room. Empty ampoule boxes and garbage piled to the ceiling. Light and water long since turned off for nonpayment. I did absolutely nothing. I could look at the end of my shoe for eight hours. I was only roused to action when the hourglass of junk ran out. . . Forty, sixty grains a day. And it was still not enough. And I could not pay. (Burroughs, 1959) (*Author William Burroughs describing his addiction to heroin*)

My advice to people today is as follows: if you take the game of life seriously, if you take your nervous system seriously, if you take your sense organs seriously, if you take the energy process seriously, you must turn on, tune in, and drop out. (Leary, 1965, p. 133) (*Harvard psychologist Timothy Leary, proponent of drug use*)

The preceding chapter highlighted the long history of use of psychoactive drugs in society. Contrasting views have always existed about the dangers and values of mind-altering or intoxicating drugs, and these views have fluctuated in different eras of history. As succeeding chapters show, the government's attitudes and policies toward psychoactive drugs were largely *laissez-faire* through the 18th and most of the 19th centuries (except for periodic campaigns against alcohol abuse). However, beginning with the end of the 19th century and through most of the 20th century, we can characterize the policy response to drug use and drug abuse as increasingly emphasizing punitive policies and a law enforcement approach to controlling drug use. Over this period, the balance of policy focus shifted to making more drugs illegal, arresting and incarcerating users and sellers, sending a societal message that use of psychoactive drugs is dangerous to individuals and society, and that the nation's laws and criminal justice policies must do everything possible to deter people from using drugs, and punish those who use and those who sell drugs.

Despite this overarching criminal justice policy emphasis on controlling drugs, there is also growing support for prevention and treatment of drug abuse. Although the vast proportion of funding for drug control still goes toward law enforcement and control of drug supplies, there is also funding for treatment and prevention, as well as some treatment diversion programs for drug offenders. In addition, beginning with the end of the last decade of the 20th century, a number of states started to decriminalize possession of small amounts of marijuana, and in 2012, voters in two states (Colorado and Washington) passed laws legalizing possession of marijuana for personal use. Does this signal a new, less punitive approach to reducing drug abuse?

Given the substantial impacts of drug abuse on health, families, crime, the economy, and children, what is the most effective approach to reducing drug use and the harms caused by drug abuse? Do punitive criminal justice policies deter people from using drugs? Is it more cost effective to develop and implement effective programs to prevent young people from initiating drug use? If drugs are to remain illegal, should persons arrested for using drugs be prosecuted and incarcerated or should they be placed in drug treatment programs instead? Do our nation's drug control policies disproportionately affect the poor and minorities, and, if so, how can these disparities be reduced or eliminated? In sum, if the goals of our society are to reduce harmful drug use, as well as the crime associated with drug use, what are the most effective and fairest ways to achieve those goals?

In order to understand how to begin to answer these questions, it is very important to understand the different types of drugs that concern society. What are the different classes of drugs and how do they affect the health and behavior of users? How do drugs affect the brain and is drug addiction caused by neurochemical changes in the brain or is drug abuse simply a behavioral or moral issue that people can control? Which drugs are more dangerous than others and perhaps deserve more attention from the criminal justice system? Are some drugs less harmful than others and thus should be handled less punitively by the criminal justice system? It is also important to understand the different theories of why people use and abuse drugs. These theories include genetic and biological, psychological, and sociological explanations for drug abuse. Each type of theory has different implications for constructing more effective policies to prevent, treat, and reduce crime associated with drug use. This chapter provides information to help the reader understand the different classes of drugs and the definitions of the terms: drug abuse and addiction. In Chapter 3, we address a related question—that is, how scholars use different theories to understand what causes drug abuse. This background is needed to assess the implications and effectiveness of our criminal justice policies toward illegal drugs, and inform how we think about improving these policies in the future.

Basic Concepts About Psychoactive Drugs

After being taken into the body by oral ingestion, smoking, injection, or snorting, the psychoactive chemicals in drugs move through the bloodstream and pass through the blood-brain barrier. The drugs act at the synaptic level to affect the action of neurotransmitters that control brain function by passing “messages” from one neuron to another. Thus, the action of a drug is at the molecular level and affects nerve endings and receptors in the brain. These neurochemical effects in relation to different drug classes lead to the psychological and behavioral manifestations of drug abuse that are described in this chapter. *For a psychoactive drug to have an effect, it must enter the bloodstream and cross the blood-brain barrier. The faster a drug gets into the brain, the more intense the effect.*

It is these neurochemical actions that produce the euphoria, intoxication, and other psychoactive effects that users seek and which motivates them to initiate and sustain use. Specific types of drugs disrupt normal neurotransmission in different ways. Some drugs directly increase the amount of existing neurotransmitter in certain parts of the brain, thus *increasing* brain activity. Other drugs have a similar effect by mimicking the action of a particular neurotransmitter. Whereas some drugs block the release of a neurotransmitter causing a *decrease* in brain activity, other drugs prevent neurotransmitters from being absorbed after release, thus increasing the amount of neurotransmitter at the synapse. Table 2.1 below shows the different neurotransmitters and areas of the brain that are affected by different types of drugs.

Drugs also differ in the length of time they act on the neurochemical systems in the brain. Some drugs, especially stimulants, such as cocaine, are very short acting and their psychoactive effects only last for 30 minutes or less. Other drugs can have effects that last for hours. In part, the differences in the length of a drug’s effects depend on how rapidly the body breaks down the drug into other chemical substances (called metabolites), which are then excreted from the body over time through urine.

Table 2.1 Key Neurotransmitters and Sections of the Brain Affected by Drugs

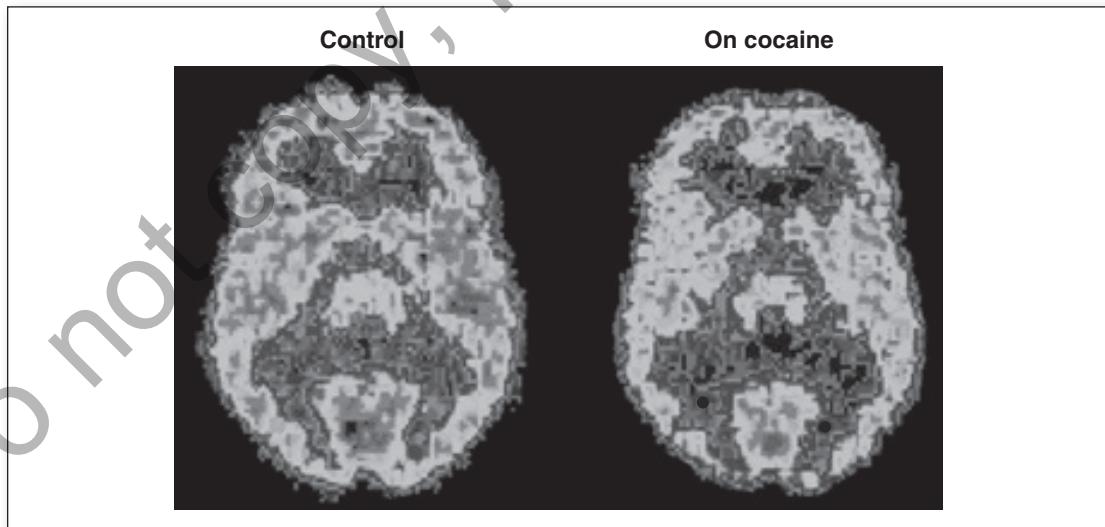
Neurotransmitter or Receptors	Drug	Area of Brain Affected
Dopamine	Cocaine, Ecstasy	Ventral tegmental area, nucleus accumbens
Serotonin	LSD, Ecstasy	Cerebral cortex, locus ceruleus
Norepinephrine	Ecstasy	Regions affecting cognition, emotion, motor function
Opioid receptors	Heroin and other opiates	Limbic system, brain stem
Cannabinoid receptors	Marijuana, hashish	Cerebellum, hippocampus, cerebral cortex, nucleus accumbens, basal ganglia, and other regions to a lesser extent

Source: National Institute on Drug Abuse, www.drugabuse.gov.

The acute and chronic effects of drugs may also vary depending on a number of individual and environmental conditions. For example, drug actions and effects may be conditioned by a person's height and weight, health status, psychological expectations about the drug's effects, or the social setting in which the drug is used. The psychoactive effects of a drug are also influenced by the presence of other drugs or substances in the body; in some cases the effects of two different drugs can be *synergistic* (that is, the combined effect is greater than the sum of the individual drug's effects) and can lead to serious and sometimes fatal consequences for the user. The duration and intensity of a drug's effects are also driven by the dosage taken, the way in which the drug is ingested, and the purity of the drug. For a novice user, the effects of a drug may actually be rather unpleasant, causing disorientation, rapid heartbeat, extreme drowsiness, or distorted vision. The user may ultimately *learn* to experience the effects as positive over time or because of social influences (see the discussion of *subcultural theory* in Chapter 3).

The acute psychoactive effects of a drug typically dissipate after the drug has metabolized in the body. However, many drugs produce chronic effects that can be direct or indirect. In terms of direct effects, repeated use of certain drugs (especially those that work through the brain's dopamine neurotransmitter system) can result in alterations in the neurochemistry of the brain that can last for many months and cause various symptoms, such as drug craving, irritability and sleeplessness, and loss of motivation (see Figure 2.1). Other chronic effects can occur indirectly because chronic drug use can be associated with poor health and nutrition, loss of employment, stress, and, of course, involvement in the criminal justice system.

Figure 2.1 Positive Emission Tomography Scan Comparison of Normal Brain and Cocaine User's Brain



Source: National Institute on Drug Abuse, "Teaching Packet" slide show called "Understanding Drug Abuse and Addiction: What Science Says." Available at <http://www.drugabuse.gov/publications/teaching-packets/understanding-drug-abuse-addiction>.

The legal classifications of psychoactive drugs and the allowable penalties for violating anti-drug laws often vary depending on the amount of the drug possessed or sold by the offenders. Criminal penalties are also driven by the weight and purity of the drug. The underlying assumption in these laws and the way they are enforced and prosecuted is that larger amounts and purer (more concentrated) versions of a drug are deemed more dangerous to the individual and society; thus, under the proportionality (or just deserts) model of sentencing, deserving of greater penalties.

Drugs vary greatly in the amount and purity of the drug needed to achieve a psychoactive effect. They also vary in the threshold of amount and purity beyond which ingestion of the drug can be dangerous or even fatal. The *effective dose* (ED) of a drug is the amount of a drug needed to achieve a specific psychoactive effect: ED50 means that 50% of the population gets the desired effect with that amount of the drug (Goode, 2012). The *lethal dose* (LD) is the amount of drug at which death occurs. Combining these two measures, the ED/LD ratio is an indicator of the relative toxicity or safety of a drug. The closer the ED is to the LD, the more dangerous the drug and the more likely it is that an overdose and death will occur. Generally, a drug with an ED/LD ratio of about 1:10 is considered dangerous. Thus, heroin, with an estimated ED/LD ratio of between 1:10 and 1:15 is one of the more dangerous drugs (Goode, 2012). What makes street drugs particularly dangerous is that the purity (and therefore the dosage) is rarely accurately known. Many drug overdoses, particularly from heroin, occur because the user purchases street drugs that are of higher purity than the user can tolerate (Levinthal, 2012).

Tolerance, Dependence, Abuse, and Addiction

An important aspect of the pharmacology of psychoactive drugs is that the repeated use of the drug may cause changes in the neurochemistry of the brain that can reduce the amount of control the person has over their use of the drug, and repeated use can also alter the drug's psychopharmacological effects. A number of terms are used to describe what happens when a person "loses control" over his or her drug use or has difficulty managing his or her use, and it is important to understand these terms and what they mean. The two key aspects of these effects are: *tolerance* and *dependence*. The overarching question is why people continue to use drugs even when such use has negative consequences for their health, legal status, job, or family. It is important to understand these concepts because they have implications for how our criminal justice system responds to drug abuse, and the different potential sanctions that a judge may impose for a crime committed by a drug user. These issues are discussed more in Chapter 5 and Chapter 8.

Tolerance occurs when the effects of a drug diminish with repeated use. Whereas for a new user of a drug, a certain dose results in the desired psychoactive effects, after multiple uses of the same dose the effects diminish or disappear. The body learns to "tolerate" and adapt to that amount of the drug. Physiological tolerance occurs because the neurons become less sensitive to the drug or the neurotransmitters adapt to the presence of that amount of the drug. Behavioral tolerance is also possible, where after chronic use, the user is able to compensate behaviorally for the drug's effects. Tolerance can be medically dangerous because over time the user may need higher and higher doses to achieve the desired psychoactive effect, and thus move closer to the lethal dose. That is one of the reasons why users overdose after having been abstinent for a period of time. The person takes a similar dose of the drug as used previously, but because the person is no longer tolerant the body can no longer handle the higher dose. A related phenomenon is *cross-tolerance*, which refers to a situation where tolerance develops to one drug, but when the person takes a different drug that is of a similar class (for example, another depressant drug) tolerance to the new drug occurs immediately.

Drug Abuse and Drug Misuse

Several other terms are used to describe drug use that has negative consequences for the user. These include *drug abuse*, *drug dependence*, and *drug misuse*. Drug abuse and drug dependence are psychological terms that are defined in the American Psychiatric Association's *Diagnostic and Statistical Manual* (DSM) of disorders in connection with specific drugs. The DSM defines drug abuse and dependence as two types of substance use disorders. Both are linked to maladaptive use of drugs that lead to clinically identifiable impairment.

The term *drug abuse* is typically used to indicate that a person is experiencing negative consequences as a result of repeated use of a drug. These consequences can include loss of a job, family problems, health issues, or criminal justice involvement. An underlying assumption is that the person is also using a drug regularly or in large amounts. Examples of symptoms of drug abuse include

- failure to fulfill major role obligations;
- legal problems;
- use in situations that are physically hazardous; and
- continued use despite persistent social or interpersonal problems.

Interestingly, the National Institute on Drug Abuse (NIDA) defines any illicit use of a substance as drug abuse; this includes the nonmedical use of prescription drugs. However, the term *drug misuse* is generally used to denote use of a prescription drug for recreational purposes, or for purposes not originally intended by the prescription. For example, taking a prescription opioid analgesic like Vicodin® to get high rather than alleviate pain is an example of drug misuse, as is taking an amphetamine like Adderall® to feel stimulated rather than to reduce symptoms of attention deficit and hyperactivity disorder.

Drug Dependence and Addiction

The term *drug dependence* is usually used to indicate that a person is using the drug compulsively and that the process of finding, buying, and taking drugs has become a central part of the person's life. Some use this term generically to also indicate that the individual is physically dependent on a drug, but drug dependence can be defined from a psychological or a physiological perspective. The term *dependence* is related to what most people think of as *addiction*: the inability to stop using a drug, or loss of control over one's use of a drug. This notion of *loss of control* or *compulsivity* is important from a criminal justice perspective. First, the more a person continues to use drugs and needs higher doses of drugs to obtain a psychoactive effect means that the person is spending more money to buy drugs, which means that it is more likely that the person becomes involved in criminal behavior. Second, many offenders are punished by the criminal justice system because they continue to use drugs despite judicial orders, or probation or parole requirements prohibiting use.

In general, the DSM-IV defines drug dependence as including the following symptoms:

- drug taking in larger amounts than intended;
- inability to cut down on drug use;
- a great deal of time spent in activities necessary to obtain the drug; and
- continued use despite knowledge of health or social problems caused by the drug.

The concept of dependence has specific physiological and psychological components that result in loss of control over use of the drug. Physical dependence on a drug occurs because the alterations in the brain chemistry produce cravings for the drug. The most important element of physical dependence is that when the person stops using a drug, he or she experiences *withdrawal symptoms*. The user is motivated to avoid the unpleasant withdrawal symptoms that occur if usage is stopped. Depending on the drug and the length of time a person has been dependent, withdrawal symptoms (called going “cold turkey” in the vernacular) can be relatively mild (e.g., flu-like symptoms, nausea, diarrhea, difficulty sleeping, cramps and muscle pain) to quite dangerous (e.g., convulsions or death). Withdrawal symptoms result from the pharmacological effects of removing the drug from a brain whose chemistry is now altered by chronic use. Symptoms can begin within a few hours of the last use of the drug and can peak in about 24 to 48 hours, but also can last a week or more. Thus, continued use of the drug is a powerful reinforcer that enables the person to avoid experiencing withdrawal. Withdrawal symptoms can be minimized or avoided by gradually reducing the dosage and/or frequency of use, or by administering a similar class, but safer drug under medical supervision.

Behavioral dependence can also occur as a result of use of classes of drugs that do not cause physical dependence. In this case, the person uses the drug compulsively because of the strong reinforcing effects of the pleasurable feelings it causes. Cocaine is a prime example of a drug that is not known to cause physical dependence, but can readily cause behavioral dependence. Even with psychological or behavioral dependence, a user may experience anxiety, sleeplessness, agitation, or other psychological symptoms on stopping use of the drug, but these symptoms are not necessarily a result of changes in brain chemistry that occur with physical dependence.

Some drug users are not just dependent on, but become addicted to drugs. *Addiction* is defined by the National Institute on Drug Abuse as

a chronic, often relapsing brain disease that causes compulsive drug seeking and use, despite harmful consequences to the individual and to those around him or her. (NIDA, 2012a, p.1)

This definition is similar to the DSM-IV definition of dependence, but the term addiction is used to connote more than just physical dependence. The individual is not only physiologically dependent on the drug (i.e., stopping use produces withdrawal symptoms), but continues using despite serious effects on her or his life and health. The person functions “normally” only when he or she is taking the drug. Central to the consideration of how to define addiction is whether it is (1) a disease over which the individual has little control; (2) a behavioral problem that the person can control; or (3) a moral issue based on the belief that people who use drugs are “bad” people who willingly break the law to indulge in their desire to get high. These complex issues are discussed in detail in Chapter 3.

Factors Influencing a Drug’s Effect on Behavior and Mood

The effects of a drug on mood and behavior are not consistent, and often not predictable. The acute consequences on the level of impairment and on criminal behavior of taking a drug can vary across and within individuals, places, and time. A number of factors are important. First, the way that a drug is ingested affects how quickly it crosses the blood-brain barrier and how quickly it is metabolized in the body, and therefore the rapidity and intensity of its psychoactive effects. As noted earlier, for a psychoactive drug to have an effect, it must enter the bloodstream and cross the blood-brain barrier. The faster a drug gets into the brain, the more intense the effect. In general, the most rapid and intense effects occur

when a drug is injected directly into the bloodstream (intravenous injection). Because of the large number of capillaries in the lungs, smoking a drug also results in rapid absorption into the blood and entry into the brain. Some drugs (notably marijuana) are not soluble in water and thus cannot be injected.

Other routes of administration generally result in a longer time to onset of the drug's effects: these include subcutaneous (under the skin) or intramuscular injection, and snorting or sniffing. Because of the effects of stomach enzymes and relatively slow absorption time, swallowing a drug is the least efficient means of taking a psychoactive drug. Another key factor is the amount of the psychoactive ingredient of the drug taken. There are several factors that are important: **Dosage**, **potency**, and **purity**. Dosage refers to the overall weight of a drug, including any adulterants or additives. Potency refers to the quantity of the actual drug needed to produce the psychoactive effect. Consequently, if 10 milligrams of a drug produces the same effect as 50 milligrams of another drug, then the first drug is considered more potent. Finally, the purity of a drug indicates the percentage of the drug sample that is the actual psychoactive drug ingredient. Street prices of drugs may fluctuate depending on potency and purity, but generally it is the case that street drugs are more potent and of higher purity today than in past decades. These issues are discussed in more detail below in the sections on individual classes of drugs.

In our federal and state penal law systems, it is the overall weight of the drug sold or possessed that determines the allowable charges and penalties. Hence, an offender arrested in possession of 10 grams of heroin is charged according to that overall weight, regardless of whether the confiscated drug is 40% heroin or 10% heroin. As long as the contraband contains any amount of the illegal drug, the criminal charge is based on the total weight.

Classes of Drugs and the Drug Enforcement Administration Schedules

In order to understand how society has responded to the use and abuse of drugs, and why our criminal laws target and punish use of different drugs in different ways, it is important to understand the differences among the various classes of drugs: how they act on the brain, their effects on mood and behavior, their potential to cause abuse or dependence, and their association with crime and criminal behavior.

In October 1970, the previous 56 years of federal drug legislation dating back to the 1914 Harrison Act, was superseded and replaced with passage of the Comprehensive Drug Abuse Prevention and Control Act (Public Law 91-513). In Title II of this Act, the Controlled Substances Act (CSA), illegal drugs were classified and penalties established for violating the provisions of the Act. Up to the present day, this landmark legislation forms the basis for the federal control of drugs. A key aspect of the 1970 Act was its attempt to classify psychoactive drugs according to their potential for abuse and their known medical utility. Although some of these classifications have become controversial over time (e.g., the decision to classify marijuana in the most severe Schedule I category), they represented an important effort to make legal and medical distinctions among the severity of drugs according to medical utility and abuse potential. Ranked in descending order of severity from Schedule I to V, the Act also established penalties for violating the provisions of the law. Drugs in Schedule I cannot be legally prescribed or sold or possessed, and drugs in Schedule II are strictly controlled in terms of prescribing, distributing, or possessing. It is illegal to possess any drug listed in the Controlled Substances Act Schedules without a valid prescription.

Although the 1970 Act generally increased the penalties for drug possession and sale, and established mandatory minimum sentences, it also reduced some penalties, such as those for marijuana

possession. Penalties were further increased under the Anti-Drug Abuse Acts of 1986 and 1988 (see Chapter 5). In addition, the 1970 Act allowed “no-knock” searches by police under certain conditions. Two subsequent Supreme Court cases (*Wilson v. Arkansas* [1995] and *Richards v. Wisconsin* [1997]) ruled that the Fourth Amendment to the U.S. Constitution, which prohibits unreasonable searches and seizures, generally required that the police knock and announce their presence before entering a premise, but that no-knock entries might be allowed on a case-by-case justification.

In addition to forming the basis for federal drug control and enforcement efforts, the 1970 Act also served as a model for state anti-drug laws. As the 1970 law was being planned and drafted, there was a simultaneous effort by the Nixon Administration and Attorney General John Mitchell to pressure states to adopt similar laws (King, 1972). Thus, the National Conference of Commissioners of Uniform State Laws drafted a Uniform Controlled Substances Act to serve as a model for state drug laws (National Conference of Commissioners of Uniform State Laws, 1970). This Uniform Act was modeled after the CSA and states were urged to modify their anti-drug laws to include these basic provisions to conform to the new federal law. Although each state is free to impose its own penalties for violation of its anti-drug laws, the basic provisions of most state laws include some form of distinction among different types of drugs and their associated penalties.

The 1970 Act was not entirely punitive; it contained provisions for increased funding for Public Health Service hospitals to treat drug abuse. It also authorized creation of the National Commission on Marihuana and Drug Abuse to study the drug abuse problem in the United States, report to the President as to the current state of drug abuse and its consequences, and recommend new policies to control drug use and abuse (National Commission on Marihuana and Drug Abuse, 1972). The Commission’s report recommended that state and federal penalties for possession of one ounce or less of marijuana should not be a criminal offense, and possession of any amount of the drug for personal use should not be a crime. It further recommended that under federal law, casual distribution of small amounts of marijuana for insignificant or no remuneration should be decriminalized. The report concluded that

society should discourage use, while concentrating its attention on the prevention and treatment of heavy and very heavy use. The Commission feels that the criminalization of possession of marihuana for personal use is socially self-defeating as a means of achieving this objective. (pp. 210–211)

The report had a major impact on state policies and laws, and 11 states decided to decriminalize marijuana shortly after release of the report. However, Republican President Richard Nixon disagreed with the conclusions of the Commission report and its recommendations were never enacted at the federal level (see Chapter 10 for a discussion of the current movement to decriminalize recreational use of marijuana.)

Table 2.2 summarizes the current criteria used for scheduling controlled substances, and provides examples of drugs included under that schedule. The Drug Enforcement Administration (DEA), with scientific input from the U.S. Department of Health and Human Services, makes the final decision as to whether drugs are scheduled and where drugs are classified (Drug Enforcement Administration, 2011). New drugs may be added to the list if they meet designated Controlled Substances Act criteria. More information about the procedures for scheduling controlled substances and a more complete list of the drugs on each schedule is available in a resource guide on the DEA website (www.justice.gov/dea).

Table 2.2 Schedule of Controlled Substances Under Federal Law

Schedule	Criteria	Examples
I	No established medical usage, cannot be used safely, and have great potential for abuse.	Heroin, LSD, Mescaline, Peyote, Methaqualone, Psilocybin, Marijuana, Hashish
II	High abuse or addiction potential; currently accepted pharmacological or medical use.	Opium, morphine, codeine, cocaine, PCP
III	Accepted medical use, but may lead to high level of psychological dependence or to moderate or low physical dependence.	Many Schedule II drugs in derivative or diluted form
IV	Relatively low potential for abuse, useful in established medical treatments, only limited risk of psychological or physical dependency.	Depressants, minor tranquilizers, some stimulants
V	Prescription drugs with low potential for abuse and only limited risk of psychological or physical dependency.	Cough medicines or anti-diarrhea drugs containing opium, morphine, or codeine

Source: Drug Enforcement Administration.

Classes and Types of Illegal Drugs and Their Effects

Although the scheduling of controlled substances is based in part on the abuse potential and medical utility of a drug, the classification of drugs is often more relevant to law enforcement and criminal justice policies and practices than to the neurobiological actions and chemical structures of these drugs. In this section, we describe the major classes of drugs according to their chemistry and their biological and psychological effects on the body. To fully understand and critically assess the effectiveness of criminal justice responses to drugs, it is important to understand the distinctions among the different classes of drugs, how they affect the brain and behavior, and their chronic effects on behavior as well as health and well-being. The distinctions among these classes of drugs are important to understand, especially because in the past many state and federal laws have erroneously classified specific drugs. For example, marijuana and cocaine have often been listed as “narcotics” in legislation; the term “narcotic” is still commonly used in vernacular by many law enforcement personnel to include any illegal drug, although the term is technically appropriate only for opiates.

Psychoactive drugs are generally classified in the following categories

- Cannabis (Marijuana)
- Central Nervous System Depressants (opiates, sedative-hypnotics)
- Stimulants (cocaine, amphetamines)
- Hallucinogens (LSD, peyote, PCP)
- Club Drugs (Ecstasy, Ketamine, Rohypnol®, GHB)

Marijuana

Marijuana is the name given to the dried and shredded leaves, flowering tips, and buds of the *Cannabis sativa* plant. Typically rolled in cigarette paper and smoked, marijuana is by far the most commonly used illegal drug in the world. It is a unique category of psychoactive drug that has some stimulant, depressant, and hallucinogenic properties. Although the *Cannabis sativa* plant, also known as hemp, has been used to make clothing, rope, and other products for thousands of years, in addition its psychoactive properties have also been known throughout history. There is evidence of pharmaceutical and recreational use of the drug in China in the 3rd century BC, and in the 2nd century BC in India. Use of hashish (the dried resin of the flowering part of the plant, containing high concentrations of delta-9-tetrahydrocannabinol or THC) spread through North Africa and the Middle East beginning in the early part of the first millennium. The use of hashish became well known in Europe as well, beginning in the Middle Ages (Mosher & Akins, 2007).

Beginning in the early colonial days in the United States, hemp plants were widely cultivated and were extensively grown in the 19th century for use in making clothing, rope, and baskets (Brecher & the editors of *Consumer Reports*, 1972). Marijuana was widely dispensed by physicians and pharmacists to treat a number of health problems during the 19th century, and was listed as an acceptable medicine in standard pharmaceutical reference works until the early 1940s (Belenko, 2000a). Many medical books and articles in the 1800s praised marijuana for its health benefits (Bonnie & Whitebread, 1974). However, recreational use of marijuana was relatively unknown in America until the early 1900s, when urban musicians and Mexican immigrant laborers became highly visible users of the drug. Prior to that period, there were various descriptions of “tea pads” or “hashish houses” where less respectable members of society met secretly to smoke or eat hashish (Belenko, 2000a; Kane, 1883). It was the spread of marijuana use by Mexican farm workers that helped spur the passage of the nation’s anti-marijuana laws, first in some states beginning in the 1920s and then at the federal level with passage of the Marihuana Tax Act of 1937 (see Chapter 5).

The primary psychoactive ingredient in marijuana is delta-9-tetrahydrocannabinol (THC), which is chemically similar to endogenous cannabinoids found in the brain. THC binds to cannabinoid receptors in various parts of the brain (primarily the cerebral cortex, hippocampus, nucleus accumbens, and basal ganglia) that control pleasure, memory, motor coordination, and sensory and time perception (NIDA, 2005a). The effects of marijuana are a result of stimulation of the cannabinoid receptors. In addition, as with many other psychoactive drugs, THC also results in release of dopamine in the brain.

Because marijuana is smoked, the THC enters the brain very quickly, and the acute effects last from one to three hours, depending on the THC potency in the marijuana and other factors, such as the setting and the user’s expectancies. These effects include feelings of pleasure and well-being (euphoria), increased heart rate and blood pressure, heightened sensitivity to sensory stimuli, and reduced coordination and fine motor skills. Due to improvements in growing and cultivation processes, the THC content of marijuana (i.e., its potency) has increased substantially since use of marijuana became widespread in the 1960s. Average potency is estimated to have increased from about 1% to 2% in 1980 to about 8% to 10% today (DEA, 2011; Levinthal, 2012), raising concerns about the potential negative health effects of marijuana smoking. However, marijuana is of relatively low toxicity, and there is no evidence that acute marijuana intoxication can cause serious health problems or death.

Although marijuana was traditionally promoted as a benign and safe drug, research over the past two decades has identified a number of potential long-term health effects. Marijuana smoke contains more than 400 chemicals, many of them carcinogenic and similar to those found in tobacco smoke.

Research has not yet shown a causal link between smoking marijuana and lung cancer, but it remains a concern. There is evidence that marijuana causes tolerance and that dependence with mild withdrawal symptoms can occur (Budney & Hughes, 2006). Chronic use of marijuana has also been shown to impair memory and learning skills, as well as cognition. In addition, in the United States in 2011 there were 455,668 emergency department visits involving marijuana; this represented 36.4% of the total visits to emergency departments involving drugs (Substance Abuse and Mental Health Services Administration (SAMHSA), 2013b). This statistic made marijuana the second most common illicit drug mentioned in emergency department admissions after cocaine.

One major reason that marijuana remains an illegal drug, at least in most jurisdictions, and remains the focus of much prevention and law enforcement effort is that most people who use and abuse illegal drugs first started by using marijuana (as well as alcohol). The notion of marijuana as a “gateway drug” means that for many officials and politicians, marijuana is dangerous and needs to be regulated and controlled because it causes or leads to use of more serious drugs, such as cocaine or heroin. Despite the fact that marijuana is generally not viewed as a “dangerous drug” by most people, and does not have strong links to crime (see Chapter 4), marijuana still has a great impact on the criminal justice system due to the large number of marijuana arrests (see Chapter 6). The evidence for and against marijuana as a gateway drug is discussed in the section below titled Focus on an Issue. More recent policy trends toward decriminalizing and legalizing marijuana are discussed in Chapter 10.

Despite its status as a Schedule I drug, marijuana is by far the most widely used illegal drug. In 2012, more than 111 million Americans aged 12 and older reported having ever used marijuana in their lives; this number represented 42.8% of this population (SAMHSA, 2013a). More than 18.9 million (7.3%) reported they were current users (i.e., indicating marijuana use in the past month).

FOCUS ON AN ISSUE: MARIJUANA AS THE “GATEWAY DRUG”?

Over the past two decades, there has been a long and controversial debate as to whether marijuana should be considered a “gateway” drug that increases the risk (especially among adolescents) of moving on to use other illegal drugs, such as heroin or cocaine. This hypothesis is prompted by the fact that most users of other illegal drugs used marijuana as their first “drug” after using alcohol and tobacco. In addition, use of marijuana is highly correlated with use of other illegal drugs. What explains the fact that adolescents usually begin experimenting with marijuana before moving on to other drug use? Is there a causal relationship between marijuana and other drug use? To what extent does marijuana use increase the risk of initiating use of other drugs, and, if so, what explains this relationship?

The “gateway hypothesis” has long been promulgated by government officials as an important reason for the continued criminalization of marijuana. First put forth by Kandel and colleagues (Kandel, 1995; Kandel & Faust, 1975; Kandel & Yamaguchi, 2002), this hypothesis argues that there is a necessary sequential progression in use of psychoactive drugs where individuals gradually initiate using licit and illicit substances. The progression begins with alcohol

and tobacco and then progresses to marijuana and subsequently on to more serious drugs, such as pills, cocaine, and opiates (Kandel, 1995). This model of progressively increasing severity of substance use is argued to be invariant; that is, the stages of increasing drug use must follow the above described pathways, although not all those who use at each stage will continue onto the subsequent stage. Indeed, most marijuana users never progress to use of more serious drugs, which would appear to undermine the gateway hypothesis (Gfroerer, Wu, & Penne, 2002).

Possible explanations for marijuana's perceived role as a gateway drug include (1) pharmacological factors in terms of marijuana's effects on brain development or neurochemical systems in the brain (Winters, 2013); (2) sociocultural factors, such as peer influences that socialize users into other drug use; (3) an increase in cross-tolerance to other drugs; or (4) the fact that use of marijuana connects adolescents to drug dealers who can then provide other drugs.

The debate around the validity of the gateway hypothesis largely stems from the difference between correlation and causation. In order to empirically justify the gateway hypothesis three criteria must be met. First, marijuana use and more serious illicit drug use must be correlated to a statistically significant degree. Second, there must be a temporal relationship where marijuana use precedes use of 'harder' drugs. Finally, no other possible variables could potentially explain this relationship (Gundy & Rebellon, 2010). The first two criteria have reasonably strong support in the literature. Research by Kandel and colleagues' (Kandel 1995; Kandel & Faust, 1975; Kandel & Yamaguchi, 2002) demonstrated the temporal relation of marijuana and other illicit drugs. Moreover, data from national surveys revealed that early onset of marijuana use (i.e., before the age of 15) is strongly correlated with later lifetime use of heroin, cocaine, and amphetamines (Gfroerer et al., 2002).

Using data from the Seattle Social Development Project, Hawkins, Hill, Gui, and Batton-Pearson (2002) found that the initiation pattern of the gateway hypothesis was reasonably well supported, although at each stage of substance use the proportion of those who do not continue on to the next substance grows significantly. They argue that the particular pathway of the gateway hypothesis is the result of social norms—that is, the social acceptability of substances influences their ordering in the pathway.

However, even these more established aspects of the gateway hypothesis have received some criticism in recent years. Degenhardt and colleagues (2009) found that violations of the gateway hierarchical progress do sometimes occur (e.g. use of other illicit drugs before marijuana) and that there is a relationship between this violation of the gateway trajectory and serious mental health disorders. In a later study using an international sample, Degenhardt and colleagues (2010) also found that there is a range in the frequency of violations of the gateway hypothesis, which suggests that cultural and contextual factors may play a role in whether the gateway hypothesis is applicable (Degenhardt et al., 2010). The studies by Degenhardt and his colleagues (2009; 2010) suggest that there may be plausible alternative variables that can explain both earlier marijuana initiation and later lifetime use or abuse of

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more serious drugs. The “third variable” problem was also explored by Gundy & Rebellon (2010), who found that when adjustments to the model correlating marijuana use and other illicit drug use were made for life course and stress related variables (e.g., employment, marriage, age), this relationship was significantly attenuated. Wells and McGee (2008) found in their analysis of the gateway hypothesis that mental health disorders like early onset bipolar disorder also predicted dependence. These findings suggest that it is plausible that other factors may be responsible for both marijuana use and later illicit drug use. A relatively simple explanation for the temporal progression from marijuana to other drugs is that marijuana is more easily available and therefore it is used first.

Finally, Kandel and Jessor (2002) note that there is strong evidence for a developmental sequence of use of different drugs that starts with alcohol and tobacco, and that heavy use of drugs earlier in the sequence increases the risk of use of drugs later in the sequence. However, they conclude that there is no empirical support for a *causal* relationship for use of earlier sequence drugs, such as marijuana and subsequent use of more serious drugs. This relationship is difficult to test empirically, and other potential explanatory factors (including risk and protective factors as well as social and environmental factors) need to be controlled.

Thus, although it is not yet clear which variable or set of variables may play a role in the initiation of drug use, it is apparent that the gateway hypothesis has flaws in its causal logic, and consequently, may not be a sufficient causal explanation for the progression from marijuana to other drugs.

Central Nervous System Depressants: Opiates

The drugs classified as opiates are central nervous depressants that are derived from the opium poppy (*papaver somniferum*). There are both natural and synthetic forms of opium, a milky, sticky substance that is extracted from the seed pods of the opium poppy. The forms naturally derived from opium or a chemical derivate of opium are termed *opiates*. Synthetic drugs that mimic the analgesic and psychoactive effects of morphine are created in the laboratory and are called *opioids*; they are discussed below.

Opium's unique and highly effective ability to reduce pain, as well as its intoxicating effects, has been known for thousands of years. The ancient Egyptians used opium to treat the pains from battle wounds, and Hippocrates wrote about the medical benefits of opium in ancient Greece. Beginning in the 1500s, a tincture of opium mixed in alcohol (called *laudanum*) was introduced into Europe and used for hundreds of years as a soporific (i.e., a sleep-inducing drug), pain killer, and intoxicant.

The primary alkaloid derived from opium is morphine, which was first extracted in the early 19th century. By the mid-1800s, morphine was widely known and prescribed as an effective analgesic (pain killer), especially in the Civil War; its use rapidly spread with the invention of the hypodermic syringe in the 1830s, which made it possible to deliver effective doses of morphine quickly and to large numbers

of patients. Opium and morphine were common ingredients in many popular patent medicines in the late 19th century that were widely marketed and used to treat common ailments, including menstrual cramps, insomnia, and colicky babies (Belenko, 2000a; Musto, 1999). Although the use and sale of opium, morphine, and their derivatives were legal at the federal level until the Harrison Act was passed in 1914, some states had passed laws regulating or banning opium products prior to 1914 (There were earlier restrictions and taxation on the importation of opium; see Chapter 5 and Belenko, 2000a). For example, the first state law against morphine was



▲ An Afghan farmer, accompanied by his daughter, extracts opium resin from poppy heads in Afghanistan's eastern province of Nangargar. Afghanistan became a major producer of opium during the 14 years of civil war.

enacted in Pennsylvania in 1860, and the first ordinance banning opium smoking was passed in San Francisco in 1875 (Belenko, 2000a), followed by Ohio in 1885. These early laws against opium smoking were passed largely in response to concerns about Chinese railroad workers bringing their opium smoking culture to the West (Musto, 1999). Because opiates and their derivatives are such effective analgesics, most of these drugs are categorized in Schedules II to V (depending on dosage and formulation), which means that under certain restrictions they can be legally prescribed by physicians for pain relief. The exception is heroin, which because of its high risk for abuse and dependence is listed as a Schedule I drug. In lower doses, opiates provide pain relief by blocking pain receptors in the brain; they also reduce anxiety and cause feelings of euphoria. In higher doses, these drugs produce drowsiness, induce sleep, and reduce motor coordination. Opiate drugs achieve their psychoactive effect through their effects on opioid receptor sites in the brain that normally are activated by endogenous substances called *endorphins* or *opioid peptides*. These substances are chemically similar to morphine, and comprise several important and basic functions in our brain. Endorphins have powerful reinforcing effects because they can relieve pain and stress and cause feelings of euphoria and well-being. When morphine is introduced into the brain through ingestion of opiates, the morphine molecules bind to the opioid receptors and mimic the effects of the natural endorphins, but on a much stronger scale.

Opiates affect other parts of the central nervous system (CNS) as well. They have an analgesic effect by blocking pain messages transmitted from the body through the spinal cord. They depress breathing and cardiac function by changing neurochemical activity in the brain stem. Finally, the ability of opiates to mimic natural endorphins works in the limbic system to increase feelings of pleasure. The high abuse potential of opiates is in part because their actions mimic the function of endorphins in the brain that provide pleasurable feelings. As a class of drugs, opiates (as well as synthetic opioids) can readily lead to compulsive use, physical dependence, and addiction. This reflects the rapid passage of the drug into the brain, the intensity of the high that occurs through its action on the opioid receptors, and the neurochemical changes in the brain that can occur with repeated use.



▲ Farmers work in a poppy field near the Kabul/Jalalabad road. Afghanistan is the world's biggest producer of poppy-derived opium, which is used to make heroin. During a recent conference in Berlin, the Afghan President Hamid Karzai appealed for international help to combat drug production.

Heroin

Heroin is one of the major drugs of abuse in the United States and is the drug of choice among street opiate users. Controlling heroin manufacture, distribution, and street sales is a major focus for federal and local law enforcement. Heroin is derived from and is chemically similar to morphine, and was first developed and marketed as a cough suppressant by the Bayer Corporation in 1898. The chemical alterations to the morphine molecule speed the crossing of the drug through the blood-brain barrier and thus make it more effective than morphine in lower doses. Once in the brain, heroin is converted quickly into morphine.

Heroin is the most commonly abused opiate, and can be ingested by several different routes. Although from the 1960s to the 1980s injection of heroin into the veins was the most common means of taking the drug, concerns about AIDS risk and the increasing purity of the drug caused many users to avoid injection and instead to smoke, sniff, or snort heroin. Intravenous injection of heroin is the most efficient route of administration, with initial euphoric effects occurring within 7 to 8 seconds. Intramuscular injection causes these effects in about 5 to 8 minutes, and snorting or smoking heroin is the slowest process, taking about 10 to 15 minutes for the drug to pass into the brain and cause euphoric effects. Heroin is sold on the street in diluted powder form, and commonly contains adulterants, such as lactose, quinine, and other drugs that can cause long-term health problems.

As with other opiates, ingestion of heroin rapidly leads to a feeling of euphoria, skin flushing, impaired mental function, slurred speech, and constricted pupils. The drug also causes a release of histamines into the bloodstream, resulting in itching. These immediate effects are followed by several hours of mental and physical relaxation with slowed respiratory and cardiac function.

One of the major concerns about heroin is its rather narrow safety margin, with the lethal dose only about 10 to 15 times greater than the effective dose (Goode, 2012). The recommended dosage of morphine for a new patient, for example, is about 60 mg daily, with about 200 mg considered a lethal dose. Addicted heroin users may consume about 300 to 500 mg per day. Because the purity of a street drug is rarely known to the user, overdose is a serious concern for heroin users. An influx of higher purity heroin from a dealer into a neighborhood can often lead to a spike in the number of overdoses.

The *chronic* effects of heroin use are both direct and indirect. The major direct concerns are the high risks of physiological tolerance and physical dependence. Although it is a myth to say that heroin

use inevitably leads to dependence and addiction (Goode, 2012), changes in brain chemistry as a result of its use can occur relatively quickly and can lead to withdrawal if use of the drug is stopped. Other chronic effects of this drug include lethargy, loss of sexual drive, and constipation. However, aside from the very serious consequence of addiction, the long-term direct physiological effects of heroin and other opiates are actually relatively mild. The major negative consequences occur as a result of the lifestyle associated with chronic heroin use. This can include a high risk of HIV (see Box 2.1) and hepatitis B or C infection as a result of sharing injection equipment, collapsed veins and infections from injection, poor health and nutrition as a result of the addiction lifestyle, and health risks from the impurities and adulterants contained in street heroin.

BOX 2.1 Heroin and HIV/AIDS

Although the percentage of heroin users who inject the drug has decreased since the 1980s as heroin purity has increased, many heroin users continue to engage in this risky means of use. Injection drug use (IDU) is an important risk factor for HIV. According to the Centers for Disease Control and Prevention, 30.3% of AIDS cases among women are related to injection drug use; 25.5% for males (CDC, 2013). These percentages have declined over time, from about 50% of female AIDS cases in the mid-1990s and 34% of male AIDS cases. In the 1980s and early 1990s, half of the heroin addicts in NYC were HIV+ (Des Jarlais et al., 1994). Among Black and Hispanic males, IDU is a more common risk factor (CDC, 2013).

Given its relative toxicity, and interaction with other CNS depressants, it is not surprising that heroin use can lead to serious health consequences. In 2011, there were an estimated 258,482 emergency department visits involving heroin, 20.6% of the total visits involving drugs (SAMHSA, 2013b). In 2012, more than 4.5 million Americans aged 12 and older reported having ever used heroin in their lives, or 1.8% of the population (SAMHSA, 2013a). About 335,000 reported they were current users. (See Box 2.2 for a discussion of heroin use in rural areas of the United States.)

BOX 2.2 Heroin Use in Rural Areas

U.S. heroin use is soaring, especially in rural areas, amid ample supply and a shift away from costlier prescription narcotics that are tougher to get, reports the *Wall Street Journal*. The number of people who said they used heroin in the past year jumped 53.5%, to 620,000, between 2002 and 2011, says the Substance Abuse and Mental Health Services Administration. There were 3,094 overdose deaths in 2010, an increase of 55% from 2000, according to the federal Centers for Disease Control and Prevention.

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Much of the heroin that reaches small towns comes from Mexico, where producers have ramped up production. Heroin seizures at the Southwest border, from Texas to California, jumped to 1,989 kilograms in fiscal 2012 from 487 kilograms in 2008, says the Drug Enforcement Administration. The heroin scourge has been driven largely by a law-enforcement crackdown on illicit use of prescription painkillers like oxycodone, and drug-company reformulations that make pills harder to crush and snort. Those addicted to the pills turned to heroin, which is cheaper and more plentiful. "Basically, you have a generation of ready-made heroin addicts," said Matthew Barnes, DEA agent in charge in Seattle.

Source: Elinson, Z., & Campo-Flores, A., 2013. © Reprinted with permission of *The Wall Street Journal*, Copyright © 2013 Dow Jones & Company, Inc. All Rights Reserved Worldwide. License numbers 3416741336822, 3416750006160.

Synthetic Opiates (Opioids)

Increasingly, abuse of synthetic forms of opiates (also called opioids) that are prescribed for pain relief, and illegal activity around the distribution and sale of these drugs, has become a serious problem for health officials and the criminal justice system. Over the past decade, evidence from law enforcement investigations, national surveys of drug use, and data from emergency department admissions and medical examiner reports indicate a growing epidemic of abuse of opioids, particularly among adolescents and young adults. The illegal use of prescription opioids includes nonmedical use of the drug (i.e., to get high), diversion of the drug to street users, and distribution and sale of the drug without a license. These drugs are typically obtained illegally through theft from pharmacies, diversion from legal distribution routes, forged or fraudulent prescriptions, "doctor shopping," illegal Internet sale, or from unused pills from legal prescriptions obtained by friends or family. A recent national news article highlighted the growing use of prescription opioids by women, and the increasing risk of overdose deaths (Tavernise, 2013). This article indicated that more women die from prescription analgesic overdose than from cervical cancer or homicide. The increased risk for women, which is occurring in rural and suburban areas as well as cities, may reflect a greater likelihood of prescribing opioid pain relievers to women, and the sometimes deadly effects of opioids in combination with alcohol or other sedative drugs (Tavernise, 2013).

Synthetic opiates have similar effects on the brain as heroin and other opiates, and therefore have a high likelihood of abuse and physical dependence when used non-medically. They are similar to morphine in their effects on the brain and behavioral dependence can also occur. When used appropriately, these drugs are highly effective analgesics and have great benefits for managing pain. Unlike heroin, however, these drugs can be legally prescribed; depending on the drug and the formulation, opioids are controlled under Schedules II, III, IV, or V. Prescription opioids are meant to be taken orally, but when abused recreationally to achieve a high, the pills may be crushed and then inhaled, smoked, or mixed with water and injected.

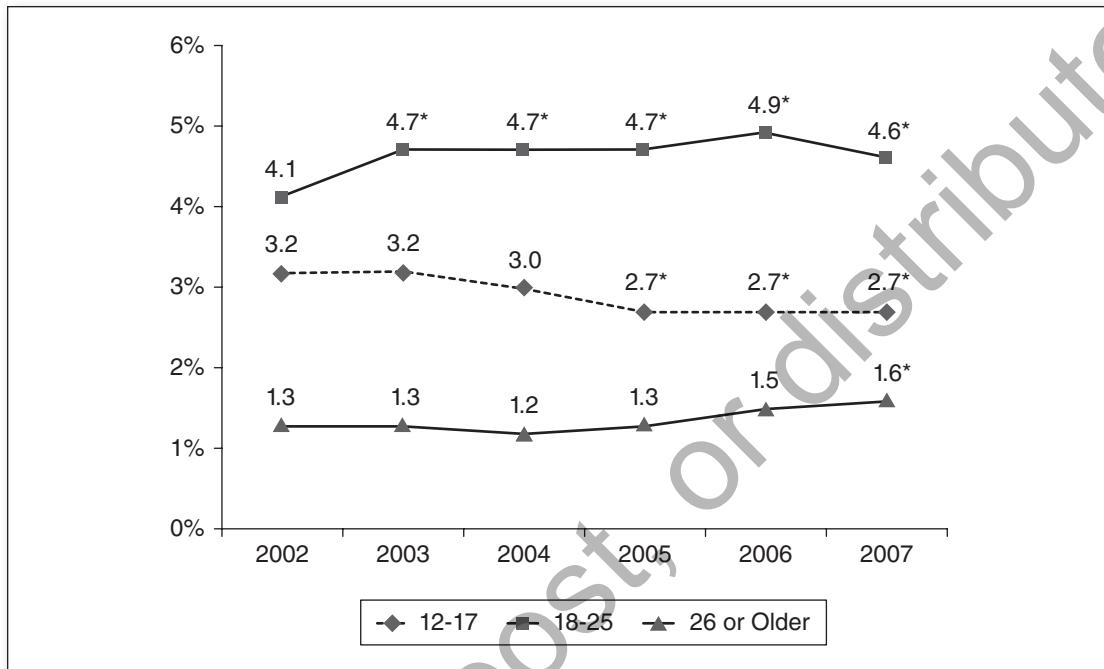
In the following sections, the major examples of commonly abused opioid drugs are briefly described. The most commonly abused opioids are Vicodin®, OxyContin®, and Percocet®. Vicodin® is the trade name for a formulation of hydrocodone with acetaminophen, and is one of the most commonly

prescribed drugs in the United States for pain relief. Although hydrocodone by itself is a Schedule II drug, it is almost always prescribed in combination with acetaminophen or aspirin, and is included in Schedule III. Because of increasing concern over misuse of Vicodin® and other hydrocodone products, there are recent attempts to have the Food and Drug Administration (FDA) recategorize them as Schedule II drugs (Leger, 2013). Oxycodone is a Schedule II opioid that is derived from thebaine, one of the active ingredients of opium. It is a highly effective analgesic that is sold in timed release tablets that vary in potency. The timed release formulation (OxyContin®) contains a high dosage of oxycodone, but was initially thought to have limited abuse potential. But, opiate abusers quickly discovered that the pills could be crushed or ground and then swallowed, snorted, or mixed with water and injected to achieve a high. Oxycodone is also marketed in combination with aspirin (Percodan®) or with acetaminophen (Percocet®). It is one of the most commonly prescribed as well as abused synthetic opiates; production of oxycodone increased from 3.5 tons in 1993 to 41 tons in 2003 (DEA, 2011). There were 151,218 emergency department visits related to nonmedical use of oxycodone products in 2011, the largest category of opioid analgesics by far (SAMHSA, 2013b). Hydromorphone (Dilaudid®) is a Schedule II drug that has a pain-killing effect that is 2 to 6 times greater than morphine. Its effects are shorter acting, however. Fentanyl is a Schedule II drug that has an analgesic effect about 80 times more potent than morphine. It was first synthesized in the 1950s and is commonly used as an anesthetic in surgery and used in patch form for pain management. Because of its potency, it is a particularly dangerous drug when abused; it can be snorted, smoked, or injected. Finally, meperidine is a Schedule II opioid that is commonly marketed as Demerol®. It has similar analgesic effects as morphine, but is shorter acting. It is commonly used in preanesthesia preparation and for postoperative pain relief.

Overall, there were 366,181 emergency department visits involving opioid pain relievers, substantially higher than the number of visits involving heroin (SAMHSA, 2013b). These included 82,480 visits for hydrocodone related emergencies in 2011. Nonmedical use of pain relievers has become a growing problem in society. In 2012, 37 million Americans aged 12 and older reported having used pain relievers nonmedically in their lives (including 6.6 million who had ever used OxyContin® nonmedically), or 14.2% of the population (SAMHSA, 2013a). Almost 4.9 million (1.9% of the population aged 12 and older) reported they were current users. As Figure 2.2 illustrates, abuse of prescription opioids is primarily a problem among adolescents and young adults, with the highest percentage of current users among those aged 16 to 25. (See Box 2.3 for a discussion of the possibility of controlled opiate use.)

BOX 2.3 Is Controlled Opiate Use Possible?

A key question that can affect criminal justice policy, as well as treatment practices, is how culpable heroin users are when under criminal justice supervision. How much control do heroin and other opiate users have over their use patterns? Is addiction to heroin inevitable once someone begins using? As some theorists point out (see discussion below) not all heroin users become dependent, many are able to stop use on their own, and some people can use heroin occasionally without apparently becoming addicted. Similarly, although alcohol is a drug that can cause physical dependence, most people are able to drink alcohol periodically without becoming dependent. What do you think? Is controlled heroin use possible?

Figure 2.2 Past Year Nonmedical Use of Pain Relievers by Age Group

Source: SAMHSA (2009). *Trends in Nonmedical Use of Prescription Pain Relievers* (NSDUH).

Sedative-Hypnotics

The types of drugs known as sedative-hypnotics (also sometimes called tranquilizers or “downers” in the vernacular) have a long history of legitimate medical utility, but also are drugs of abuse and their effects can be quite harmful. The origins of these drugs date back to 1864 when barbaturic acid was discovered and found to induce sleep. The first drug based on this was called Veronal and came on the market in 1903. A number of related drugs, called barbiturates, were developed over the next decades (examples are Phenobarbital, Amytal, Seconal, and Nembutal) and became commonly prescribed for insomnia, seizure disorders, and sedation.

Barbiturates slow down neural activity in the brain, and depress respiration and heart rate. In lower doses, they cause drowsiness, relaxation, reduced anxiety, and mild euphoria. In higher doses, they induce sleep, interfere with muscle coordination, impair perception and cognition, and can induce paranoia and hostility. Some are short-acting drugs, with their effects dissipating in about 6 to 7 hours, and others are long-acting, with effects lasting 12 to 24 hours (Mosher & Akins, 2007). The particular dangers of barbiturate drugs are that they are relatively toxic and can lead to compulsive use and dependence. The ED/LD ratio is relatively high, and fatal overdoses are much more common than with other drugs, especially when alcohol is also consumed due to the synergistic effects of the two drugs. Chronic use of barbiturates produces physical dependence, and serious

health consequences because of the very severe withdrawal symptoms that occur when a user stops taking these drugs. Because of these dangers, most barbiturates are now Schedule II and the number of prescriptions for these drugs has dropped dramatically over the last 40 years.

A new type of barbiturate, methaqualone (with the brand name of Quaalude) was developed in the mid-1960s and was thought to be safer for treatment of insomnia; it became a very popular prescription drug. However, extensive abuse of this drug spread in the 1970s and the DEA moved it to Schedule I in 1985.

A new and safer class of drugs called benzodiazepines was developed beginning in the 1950s and first marketed in 1960. The first of these drugs to market was called Librium®, which was then largely replaced by a more potent benzodiazepine, Valium®, which was aggressively marketed in the 1970s and became one of the most widely prescribed drugs in the country. Today, Xanax® (alprazolam) is the most commonly prescribed of these drugs. Over time, these sedative-hypnotics have largely replaced barbiturates for treatment of insomnia and anxiety disorders. They are substantially safer drugs in terms of overdose risk, are absorbed slowly with gradual onset of effects, and they do not depress respiration. These drugs are listed in Schedule IV.

Benzodiazepines work by increasing the activity of the neurotransmitter gamma-aminobutyric acid (GABA), which has inhibitory effects on the central nervous system (Levinthal, 2012). They also cause release of dopamine. In low doses, benzodiazepines produce relaxation and feelings of euphoria; at higher doses, they induce sleep. However, these drugs are not without health consequences. It is clear that benzodiazepines can be abused and cause dependence and withdrawal symptoms. As with other CNS depressants, they can be dangerous when taken with alcohol.

Both the number of prescriptions and the nonmedical use of barbiturates have declined dramatically over the past 30 years. But, acute medical problems are fairly common from these drugs: there were 421,940 emergency department visits involving sedatives or hypnotics in 2011, and 33.9% of the total visits involving pharmaceutical drugs (SAMHSA, 2013b). In 2012, 23.6 million Americans aged 12 and older reported having used tranquilizers nonmedically in their lives, or 9.1% of the population (SAMHSA, 2013a). About 2.1 million people (0.8%) reported they were current users. Although benzodiazepine drugs are now among the most commonly prescribed drugs in the United States, mainly as antianxiety medications, they are also commonly misused and abused and street sales of these drugs are common.

Stimulant Drugs

Stimulant drugs are among the most commonly abused substances, and they can have quite serious physical and psychological health effects. These drugs have been of particular law enforcement concern since the 1970s because of their association with violent crime and the particularly violent nature of the cocaine trafficking and associated distribution systems (see Chapter 4). Stimulants increase the levels of neurotransmitters in the brain that affect mood and behavior, and can lead to dependence.

Cocaine

Cocaine is derived from the leaves of the coca plant (*Erythroxylon coca*), indigenous to the Andean regions of South America. Indigenous populations in South America have chewed the leaves of the coca plant for thousands of years or brewed coca tea for its mild stimulant effect; its use was quite common in the Incan civilization (Levinthal, 2012) and chewing of coca leaves remains legal in

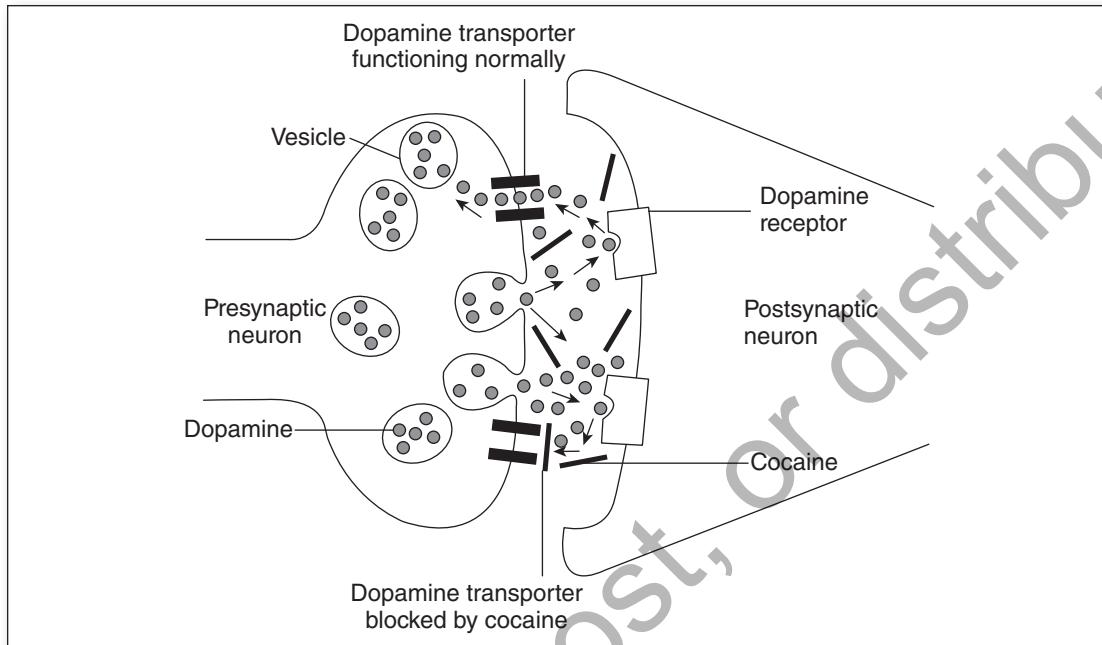
most South American countries. This greatly complicates the ability of the United States to control or eradicate production of the coca plant in those regions. Because the coca leaves contain low doses of the active ingredient *cocaine hydrochloride* and stomach absorption is a slow process, chewing the leaves generally does not cause any abuse or dependence problems.

Cocaine was first extracted from the plant around 1860, and it became a popular ingredient in patent medicines in the late 1800s. Marketed as a “brain tonic” to increase energy and relieve fatigue, cocaine was a key ingredient in Coca Cola and other beverages. It was viewed by many as a “miracle” drug in the late 1800s, curing all sorts of ailments and thought to be relatively safe. Its properties as a stimulant were widely advertised. As is well documented, for many years the eminent psychologist Sigmund Freud was an active user and strong proponent of cocaine for its supposed positive psychological and physiological effects (Freud, 1884). With broad exposés of the patent medicine industry and passage of the Pure Food and Drug Act of 1906 (which required labeling of the ingredients in all medicines), the Coca Cola company began removing the cocaine from the coca leaves. To the present day, the beverage contains “decocainized” extracts of coca leaves.

Despite the concerns about abuse and dependence, cocaine remains a Schedule II drug, because it is sometimes used as a topical anesthetic in eye, nose, or throat surgery. Cocaine hydrochloride is a powerful and short-acting central nervous system stimulant, and is rapidly metabolized in the body. It can be snorted as a powder, mixed with water and injected, or converted to a freebase form and smoked. Its acute effects include a strong feeling of euphoria, self-confidence, suppressed appetite, and energy combined with constriction of the blood vessels, and increases in heart rate, respiration, and blood pressure. In some instances, cocaine can cause cardiac arrhythmia, heart attacks, strokes, or seizures. The acute symptoms are followed by a “crash” in which the user experiences fatigue and depression. The effects of cocaine occur very quickly after ingestion and are quite brief. Smoking cocaine causes psychoactive effects within a few seconds, injection within 15 to 30 seconds, and snorting in a few minutes (DEA, 2011). The effects typically last about 10 to 30 minutes, depending on the route of administration (duration is shortest from smoking). Because the effects of cocaine are so brief, binge use (repeated use over short periods of time) is common.

The effects of cocaine in the brain are primarily a result of increases in the neurotransmitter dopamine in the nucleus accumbens, the part of the brain that responds to rewards and controls feelings of pleasure (NIDA, 2010a). Normally, feelings of pleasure are accompanied by release of dopamine in the synapse, which is then absorbed back into the nerve endings. Cocaine blocks the dopamine transporter in the nerve endings that reabsorb released dopamine from the nerve synapse, resulting in accumulation of the chemical in the synapse that amplifies neural activity, which increases feelings of euphoria and excitation (see Figure 2.3). These effects can be quite powerful and strongly reinforcing. With chronic use, the effects of the dopamine receptors (which absorb released dopamine) are reduced, resulting in lower levels of dopamine in the brain. This is one reason that dependence and cravings occur in the cocaine user; there is a need to increase levels of dopamine in the brain just to feel “normal” (Levinthal, 2012; NIDA, 2010a).

The chronic effects of cocaine can include both psychological and physiological problems. Psychological dependence is possible because of the strong reinforcing effects of the drug, and the depression and irritability that follow use. Compulsive patterns of use are common, but not inevitable, as a result many users are able to use cocaine occasionally and without serious effects (Levinthal, 2012). However, the changes in the brain chemistry caused by repeated cocaine use (i.e., depletion of dopamine) can continue to cause cravings long after the cocaine use has stopped. In addition, tolerance and

Figure 2.3 Effects of Cocaine on Nerve Synapse in the Brain.

Source: National Institutes of Health (NIH). (2010). *The Brain: Understanding Neurobiology through the Science of Addiction*. Bethesda, MD: Author. Available at: <http://science.education.nih.gov/supplements/nih2/addiction/guide/pdfs/Entire.pdf>

sensitization to cocaine can occur with chronic use (NIDA, 2010a). Other chronic effects include irritability, depression, and irritation of the nasal passages and throat.

Crack Cocaine

In its common hydrochloride powder form, cocaine is destroyed by high temperatures and thus cannot be smoked to achieve a high. However, removal of the hydrochloride salt converts cocaine into a form that can be smoked or the vapors inhaled. **Freebase** cocaine is a “pure” form of cocaine that was developed in the 1970s. It is made by treating powdered cocaine with highly flammable chemicals like ether, creating a “base” form of cocaine that can be smoked. In a famous case that probably discouraged many users from using freebase, comedian Richard Pryor was seriously injured in 1980 when the ether he was using to freebase crack cocaine exploded and critically burned him.

A safer and cheaper alternative emerged in the mid-1980s when cocaine powder began to be treated with baking soda and then heated to remove the salt. This process yields small chunks or “rocks” that became known as crack cocaine with a relatively high degree of purity. The crack is heated in a pipe and the vapors are smoked or inhaled. The drug enters the brain almost instantaneously and causes intense, but short-lived euphoria. Because its euphoric effects are shorter-lasting than from snorted

powder cocaine, binge use of crack is more common. Despite the fact that the cost of a single dose is lower, the costs to the user are greater because of increased frequency of use.

The emergence of crack in Los Angeles around 1983 and in New York City around 1985 caused a firestorm of media and political hysteria (Belenko, 1993; Reinerman & Levine, 1989). At a cost of only \$3 to \$10 per vial, it was cheaper than powder cocaine. Numerous media accounts raised concerns that crack users were likely to become irrational and violent, and there were concerns that use of the drug was spreading rapidly across America, and that gang-related crack dealers were spreading through the country and engaging in extensive gun violence.

A sample of media headlines from the late 1980s illustrates the grave concerns about the emergence of crack:

Every day more children surrender their bodies and souls to crack, driven to madness, robbed of their childhood, left desperate and broken and dangerous. (Weinraub, 1989)

Crack is a plague that is eating away at the fabric of America. (*ABC News* as quoted in Belenko, 1993)

Crack has turned many American cities into virtual war zones. ("Slaughter in the Streets," 1988)

One result of the "moral panic" (Goode & Ben-Yehuda, 2009) over crack was the passage of increasingly punitive anti-drug laws in the states and at the federal level (Belenko, 1993). The Anti-Drug Abuse Acts of 1986 and 1988 (discussed in Chapter 5) and the rise in both mandatory sentencing laws for drug offenders and enhanced penalties for crack use and sale (see Chapter 5) can be viewed as a direct result of the concerns about the impacts of crack cocaine that spread in the late 1980s. As with previous moral panics over other drugs (Goode & Ben-Yehuda, 2009), the predictions of an uncontrolled epidemic of crack use never materialized (Belenko, 1993).

In 2011, there were an estimated 505,224 emergency department visits involving cocaine, 40.3% of the total visits involving drugs (SAMHSA, 2013b). Cocaine is the second most commonly used illegal drug in the United States. In 2012, 37.7 million Americans aged 12 and older reported having used cocaine in their lives (9.0 million of whom had ever used crack), or 14.5% of the population (SAMHSA, 2013b). Almost 1.7 million (0.6%) reported as current users. The prevalence of current cocaine use peaked at about 3.0% in 1985 and began declining soon thereafter. By 1992, only 0.7% reported current cocaine use and that percentage has remained relatively stable since then.

Amphetamines

Another important class of CNS stimulants includes the various forms of amphetamine. The first amphetamine, Benzedrine, was synthesized in 1887; it was marketed as a nasal spray to help relieve symptoms of asthma and other respiratory diseases. The drug is derived from the plant-based *ephedra*, which has been used for thousands of years (the first use recorded in China) to treat respiratory problems. Once its stimulant properties became known, Benzedrine was more widely marketed in the 1930s, and was used by soldiers in World War II to increase alertness and reduce fatigue on the battlefield. Other instrumental uses of Benzedrine and other synthesized amphetamines (e.g., Dexedrine) continued in to the early 1970s by truck drivers and students to help them stay awake, by people trying to lose weight (amphetamines suppress the appetite), and by athletes trying to reduce fatigue and

improve performance on the field (Mosher & Akins, 2007). Recreational use peaked in the 1980s. From the 1950s through the 1980s, it was well known that amphetamine pills (known as “greenies”) were handed out liberally in major league baseball locker rooms for their supposed effect on reducing fatigue and improving performance on the field (Curry, 2006). Depending on their formulation and potency, amphetamines can be Schedule II, III, or IV drugs. They are legally prescribed for narcolepsy, obesity and weight loss, attention deficit hyperactivity disorder in children (Ritalin®), and other disorders. All are in pill form and are taken orally. Amphetamines are chemically similar to the neurotransmitters dopamine and norepinephrine, and result in an increased release of dopamine in the brain as well as blocking its reabsorption; thus, the drug results in substantial increases in dopamine levels of the brain accounting for its stimulating effect. Amphetamines, in particular methamphetamine (see below), are metabolized more slowly than cocaine and therefore the acute effects last longer.

In lower doses, amphetamines result in feelings of euphoria, improved motor skills, higher self-confidence, reduced fatigue, alertness, and a sense of well-being. The drug suppresses appetite and increases heart rate and blood pressure. In higher doses, amphetamines can cause impaired mental performance, paranoia, agitation, violent behavior, and psychotic symptoms. Because of their rapid action, prolonged effects, and “crash” once the effects wear off, with chronic use amphetamines carry a high risk of psychological dependence and addiction potential. Binge use is common and tolerance can develop. Withdrawal symptoms following continued use of amphetamines include severe depression, anxiety, fatigue, irritability, and sleeplessness. Chronic effects result from changes in brain chemistry and include insomnia, anxiety, violent behavior, memory loss, hyperactivity, and compulsive behaviors. However, the long term effects on the brain have been shown to be reversible.

Methamphetamine

Methamphetamine is a derivative of amphetamine that is highly addictive and potent. It is a Schedule II drug that can be used medically to treat narcolepsy in lower doses. Its increased abuse potential is because it more rapidly crosses the blood-brain barrier and it is commonly ingested in powder or crystallized form by smoking, snorting, or injecting (NIDA, 2014b). Thus, higher levels of the drug enter the brain more quickly, and the effects last longer than amphetamine. Also known as “ice,” “crank” or “crystal meth,” much of methamphetamine is made in large laboratories in Mexico, but is also easily made in small clandestine labs in rural areas. The raw ingredients for manufacturing methamphetamine are relatively inexpensive and until recently easy to obtain. A 2006 federal law restricted over-the-counter



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▲ Teeth of a methamphetamine user. A strong desire for sugary foods and drinks, compulsive tooth grinding, and general neglect of regular dental care are common among chronic methamphetamine users.

sales of drugs containing pseudoephedrine (such as cold medicines) and required that purchasers show identification and fill out a log (Levinthal, 2012). The amount of this type of drug that can be legally purchased is also restricted. Abuse of methamphetamine became a concern beginning in the late 1960s, when “speed freaks” injected a solution of the drug. Use again escalated beginning in the late 1980s and to this present day concerns about the impacts of methamphetamine use and abuse remain. The acute effects of methamphetamine can last up to 12 hours, and binge use is common because of the severe and unpleasant “crash” that occurs when the effects of the drug wear off. The acute and chronic effects of methamphetamine are similar to other amphetamines, but can be more severe because of its higher potency and longer effects. In addition to high risk of addiction, chronic effects can include psychotic symptoms, such as hallucinations, violent behavior, and structural brain damage in the areas that control memory and emotion (NIDA, 2014b).

Although it is more popular in the western United States, abuse of methamphetamines is found in all regions of the country. Data from the Department of Justice Arrestee Drug Abuse Monitoring program reveal that 57% of female and 40% of male arrestees in Honolulu and 42% of males and 38% of female arrestees in Phoenix test positive for methamphetamine.

In 2011, there were 70,831 emergency department visits involving amphetamines, and 102,961 involving methamphetamine, which equated to 12.8% combined of the total visits involving drugs (SAMHSA, 2013b).

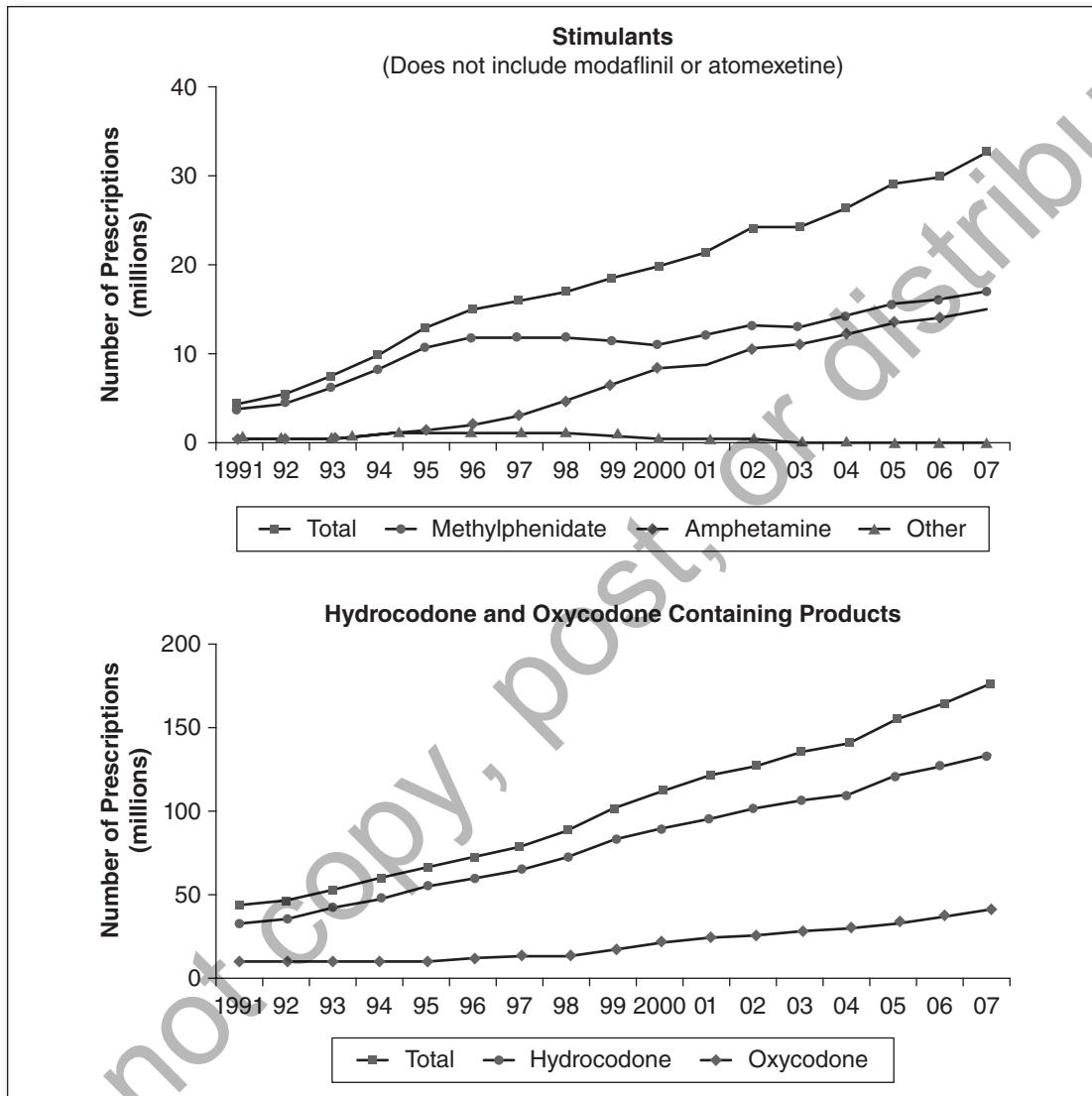
In 2012, 21.5 million Americans aged 12 and older reported having used stimulants nonmedically in their lives (among these persons 12.3 million had ever used methamphetamine nonmedically), or 8.3% of the population (SAMHSA, 2013a). About 1.1 million (0.5%) reported as current users. One concern about amphetamines is the growing number of prescriptions to treat attention hyperactivity disorders and other conditions; as can be seen in Figure 2.4, the number of amphetamine prescriptions has increased dramatically over the past 20 years. This means that there are more opportunities for misuse of these drugs or illegal diversion to recreational use.

Hallucinogens

As a class of drugs, hallucinogens are noted for their effects on sensory perceptions, causing auditory and visual distortions as well as hallucinations. They generally act by disrupting the neurotransmitter serotonin, which affects control of behavioral, perceptual, and regulatory systems in the brain, including sensory perception, mood, and muscle control. Some hallucinogens, such as PCP, also have dissociative properties and affect the distribution of the neurotransmitter glutamate in the brain. Most of these drugs are taken orally, either in pill or powdered form, or in the case of LSD, from paper soaked with the drug. Interestingly, nearly all hallucinogens that are now considered drugs of abuse at one time had legitimate medical uses, and some have a long history of accepted use in religious and cultural ceremonies. Despite this historical legitimacy, hallucinogens are now the focus of strict regulatory control and law enforcement attention due to their intense and sometimes unpredictable psychoactive effects. Use of these drugs is relatively common, with 36.3 million Americans aged 12 or older (14.1%) reporting ever using hallucinogens in their lifetime.

Lysergic Acid Diethylamide (LSD)

LSD is the most widely known and abused hallucinogen, and received broad publicity and law enforcement attention during the 1960s. It is a chemical derived from a fungus (i.e., ergot) that affects rye and

Figure 2.4 Total Number of Prescriptions Dispensed by U.S. Retail Pharmacies, 1991–2007

Source: Verispan VONA.

other grains, and was discovered in 1938 by the Swiss chemist Albert Hofmann. Although he had been searching for new therapeutic drugs based on ergot, Dr. Hofmann accidentally ingested a small amount of one compound and experienced intense perceptual distortions and hallucinations. Some years later, in the 1950s, researchers began experimenting with LSD for its possible use in psychotherapy—it was

thought that its intense psychoactive effects would allow patients to achieve important insights into their psychological problems (Mosher and Akins, 2007). In fact, beginning in 1947 it was actually marketed by the Sandoz drug company as a cure for schizophrenia, alcoholism, criminal behavior, and “sexual perversions” (Mosher & Akins, 2007). It was also tested as a possible treatment for alcoholism, and extensive testing was conducted by the Central Intelligence Agency (often on unsuspecting soldiers) for its possible utility against enemy soldiers.

A highly publicized movement led by Harvard psychology professors Timothy Leary and Richard Alpert arose in the 1960s to promote the recreational use of LSD and other hallucinogens. Connected to the hippie and antiwar movements of that era, use of these drugs was promoted as a way to expand one’s consciousness, rebel against society, and achieve personal insights and satisfaction (Levinthal, 2012). However, negative stories about the effects of LSD began to appear in the media, including false reports that it caused birth defects, chromosome damage, and violent behavior. In addition, the extreme potency of LSD (i.e., intense psychoactive effects lasting 6 to 12 hours occur at a dose of only about 30 micrograms), and the ease with which it could be made in clandestine laboratories, raised concerns about spreading use and abuse of the drug. In 1965, the manufacture and sale of LSD was made illegal, and possession was made a felony; it was declared a Schedule I drug when the Controlled Substances Act was enacted in 1970.

The effects of LSD stem from its stimulation of serotonin receptors in the brain, especially an area in the cerebral cortex called the locus ceruleus that controls receipt and processing of external stimuli. These effects appear within about 30 to 90 minutes of taking the drug, and can be unpredictable and affected by external stimuli, expectations, and presence of other drugs (National Institute on Drug Abuse, 2014a). Various symptoms include an increase in heart rate and blood pressure, dizziness, sweating, elevated body temperature, and nausea. Sounds or visual stimuli may be distorted, and hallucinations may occur (although this is less common). A phenomenon known as *synesthesia* can occur resulting in a “crossover” in sensations: sounds may be seen, and smells may be experienced as lights or colors (NIDA, 2014a). The effects on a specific user can be difficult to predict, and may be experienced as either pleasurable or frightening depending on the user’s experience and expectancies about the drug; similarly depending on the dosage the effects can last up to about 12 hours. Another concern about LSD is that because of the forms in which LSD is usually sold, it is often impossible for the user to know the dosage, and the drug is often mixed with unknown adulterants that can make acute effects uncertain.

Tolerance to the effects of LSD can occur relatively quickly, meaning that users must take increasingly higher doses to achieve the same psychoactive effects over time. However, there is no evidence that LSD causes dependence, and use of the drug (perhaps because of its intense and long-lasting effects) tends to be episodic rather than frequent and regular. One concern is the possibility of Hallucinogen Persisting Perception Disorder (HPPD) in which the user suddenly and unexpectedly experiences “flashbacks” or renewed hallucinations or perceptual distortions months after taking the drug (Levinthal, 2012). Little is known about the cause of HPPD, factors affecting its onset, prevalence, or possible treatment, but the experience can be quite frightening.

Despite its powerful hallucinogenic effects, LSD appears to be relatively nontoxic (see Box 2.4). In 2011, there were 4,819 emergency department visits involving LSD; this represented only 0.4% of the total visits involving drugs (SAMHSA, 2013b). Use of LSD appears to have decreased over time, with 23.8 million Americans aged 12 and older reporting in 2012 having ever used LSD in their lives (9.1% of the population) (SAMHSA, 2013a). Only 184,000 reported they were current users in 2012.

BOX 2.4 Hallucinogens and Moral Panics

There was considerable media attention and societal concern in the 1960s and early 1970s about the effects of LSD on the user (Goode & Ben-Yehuda, 2009). As with other moral panics over drugs, frequent newspaper and magazine articles highlighted LSD-induced “psychoses,” violence committed under the influence of LSD, and the frequency of “bad trips.” Stories emerged from the scientific community that research was showing that use of LSD caused chromosome damage and birth defects; these research findings were later demonstrated to be erroneous. *The New York Times* ran a story about teenagers in Pennsylvania who went blind after staring at the sun while under the influence of LSD (“Six Youths on LSD ‘Trip’ Blinded by Sun,” 1968). The story was later recanted when it was discovered that a Pennsylvania government official had made up the story to raise concerns about the dangers of LSD. Nonetheless, anecdotes like these helped to convince the U.S. Government to reschedule LSD as a Schedule I drug in the early 1970s.

Phencyclidine (PCP)

Phencyclidine (known as PCP) is a Schedule II drug that is termed a dissociative anesthetic, with hallucinogenic and some stimulant properties. It was developed in the 1950s for use as an anesthetic; it was thought to be safer than barbiturates because it did not depress respiratory or cardiac functioning. Although it was never approved for use in humans because of its dissociative and trance-like effects, it is used as an anesthetic in animal surgery. PCP is typically taken in pill or powdered form, and can have unpredictable effects that last for hours. It can also be snorted or smoked (mixed with marijuana). PCP affects the glutamate receptors in the brain and also increases release of dopamine. In higher doses it can be dangerous, causing seizures, and there is evidence for a risk of physical dependence (National Institute on Drug Abuse, 2014a). As a dissociative drug, PCP can produce a feeling of detachment from one’s body, imperviousness to pain, and distortions of thought. Chronic effects can include memory loss and depression.

Concern over the abuse of PCP arose in the 1970s as reports surfaced about its association with violent and unpredictable behavior. Prevalence of PCP use among arrestees was also high in some areas of the country, such as Philadelphia. Accordingly, there was increasing law enforcement attention toward this drug and some media attention to its dangers that caused a minor “moral panic.” Concerns that use of PCP would become widespread and be related to increased violence proved to be unfounded; however, the media coverage of these stories turned out to be erroneous or exaggerated (Goode & Ben-Yehuda, 2009). In 2012, 6.6 million Americans aged 12 or older reported ever using PCP in their lives (2.5% of the population, but only 32,000 reported using it in the past month (SAMHSA, 2013a).

Psilocybin (Mushrooms) and Peyote (Mescaline)

Both psilocybin (contained in various species of mushrooms) and the peyote cactus (active ingredient mescaline) are plants whose hallucinogenic properties have been known for thousands of years.

The ancient Aztecs used psilocybin in religious and cultural ceremonies, as have other Mexican and Central American indigenous cultures. A synthetic form of psilocybin was developed by Dr. Albert Hofmann and marketed by the pharmaceutical company Sandoz through the 1950s as a psychotherapeutic drug until it was made illegal in 1965 (Mosher & Akins, 2007).

The tips of the peyote cactus have been used in religious ceremonies by Native Americans for thousands of years for their mild hallucinogenic properties. Mescaline is one of the active ingredients in peyote and synthetic forms are also produced in clandestine laboratories. Both psilocybin and peyote cause feelings of euphoria and hallucinations that can last from four to six hours, although the effects are not as intense as with LSD (Mosher & Akins, 2007).

Today, psilocybin, peyote, and mescaline are all listed as Schedule I drugs. Despite its illegal status under federal law and under laws in several states, religious use of peyote is still allowed in Native American Church (NAC) ceremonies. Several growers in Texas are licensed to grow peyote for use by the NAC (Levinthal, 2012).

Club Drugs

The use and abuse of the so-called “club drugs” has been a social, health, and criminal justice concern for many years. The general name for these drugs arose because they are typically used by young people at late-night dance parties or clubs to increase energy, enhance enjoyment of music and dancing, and achieve euphoric effects. Club drugs are a heterogeneous group of drugs that include hallucinogens, stimulants, and depressants, and thus are difficult to classify. One concern about these drugs is that the club or “rave” culture views them as relatively safe; however, as discussed under each specific club drug below, these are potentially dangerous drugs that can cause serious acute and chronic health problems.

Trafficking in club drugs (especially Ecstasy) has also drawn considerable attention from domestic and international law enforcement agencies. For example, two major international ecstasy trafficking rings were broken up by the DEA in 2004 and 2005, with the cooperation of a number of local, federal, and international law enforcement agencies. In 2004, Operation Candy Box dismantled an international smuggling ring responsible for distributing about 1 million ecstasy tablets per month in the United States. In 2005, Operation Sweet Tooth resulted in the arrests of 291 persons in the United States and Canada allegedly involved in distributing 1.5 million ecstasy tablets per month. Two common club drugs—Ecstasy (MDMA) and ketamine—are more accurately classified as hallucinogens, but are discussed in this section.

Gamma-hydroxybutyrate (GHB) is a CNS depressant that was first synthesized in the 1960s as a potential anesthetic. In its early years on the market, it was sold in health food stores and gyms as a sleep aid and nutritional supplement for its supposed bodybuilding properties (Levinthal, 2012; Mosher & Akins, 2007). However, it gradually became known as a drug of abuse for its psychoactive effects. In the early 1990s, it was placed on Schedule I and is now only produced in clandestine labs. In smaller doses, GHB causes reduced control over one's behaviors and emotions (disinhibition), an “out-of-body” feeling, and an increased libido; in higher doses, it causes sedation and vomiting and can result in seizures and loss of consciousness. As with other club drugs, its effects are greatly enhanced and become more dangerous when taken with alcohol. Because it is colorless and tasteless, GHB has been used as a date rape drug when placed into a woman's drink.

Rohypnol is a benzodiazepine that is well known as a date rape drug. It is not legally available in the United States, but is legally prescribed in other countries for sleep disorders and as an anesthetic.

It is more potent and its effects last longer than other benzodiazepines. In addition to its use as a date rape drug (slipped into a woman's drink at a bar or club), it has a high risk of abuse and dependence. Its psychoactive effects include drowsiness, confusion, amnesia, and visual disturbances.

The drug methylenedioxymethamphetamine (MDMA), as its name suggests, is related to amphetamines, but also has hallucinogenic properties, so it is commonly classified in the latter category. Known by its street name as Ecstasy, MDMA has been around since the early 1900s, but it was relatively unknown until it began being studied as a potential tool in psychotherapy (see discussion on LSD above) in the 1970s (NIDA, 2010b). Beginning in the early 1980s, MDMA surfaced as a street drug, used for its psychoactive effects, and eventually became an extremely popular drug in the club and rave scene. With growing concern about its abuse potential, and lack of evidence about its medical benefits, the DEA placed MDMA on Schedule I in 1985.

Ecstasy has chemical similarities to methamphetamine, mescaline, and the neurotransmitter norepinephrine (NIDA, 2010b). It is taken in pill form and its effects begin in about 30 minutes and last from three to six hours (DEA, 2011). Ecstasy produces its psychoactive effects by increasing levels of the neurotransmitters serotonin, dopamine, and norepinephrine. The immediate effects include increased heart rate and blood pressure, enhanced sensory perception, reduced anxiety, and a sense of well-being and empathy. The latter effects are in part what accounted for the drug's positive reputation, popularity as a club drug, and nickname as the "hug drug" (Levinthal, 2012). However, MDMA can have substantial negative health and psychological effects as well. There is some evidence that users can become dependent on the drug and that chronic use can result in anxiety and depression, sleep disturbances, and memory loss, in part because it can lead to a depletion of serotonin in the brain (NIDA, 2010b). Adverse acute effects can include nausea and vomiting, muscle cramping, panic attacks, and hyperthermia.

In 2012, 16.2 million Americans aged 12 and older reported having used Ecstasy in their lives (6.2% of the population) (SAMHSA, 2013a), and 628,000 reported use in the past month.

Ketamine (street name Special K or Vitamin K) is a sedative, chemically related to PCP, which is used as an anesthetic, but that also has hallucinogenic effects. It was developed to replace PCP as a less potent, shorter-acting anesthetic. Like PCP, it is considered a dissociative anesthetic with hallucinogenic properties, but is less potent than PCP. It is classified as a Schedule III drug; its benefits as an anesthetic are that patients feel detached from their pain, and in contrast with most anesthetics, it does not depress respiration or the cardiovascular system (Drug Enforcement Administration, 2011). Beginning in the 1990s, the drug became popular in the club scene and concerns over its abuse led to its rescheduling into Schedule III in 1999.

Ketamine is mainly taken orally in powder form, although it can also be smoked or injected. Its effects begin in 15 minutes and last about an hour. Its main acute effects include dreamlike intoxication, an "out-of-body" experience, and hallucinations. It can lead to physical or psychological dependence, depression, and memory or cognitive deficits.



Summary: Psychoactive Drugs and Their Effects

This chapter has reviewed basic principles for understanding the use and abuse of psychoactive drugs. These drugs produce their effects by entering the brain and disrupting or enhancing the actions of various neurotransmitters in different sections of the brain. Medically, drugs are generally classified by

their neurochemical effects in the brain, and by the subsequent effects that these brain changes have on mood, emotions, and behavior. Thus, stimulant drugs, such as cocaine and amphetamines increase neurochemical activity in the brain and produce euphoria, excitement, and increased energy. Depressant drugs, such as opiates or barbiturates slow down neurochemical activity and mimic natural endorphins to produce euphoria; opiates and synthetic opiates also block pain messages. Hallucinogens like LSD stimulate serotonin receptors in parts of the brain that control sensory perceptions and the processing of external stimuli. A number of factors can enhance or reduce the psychoactive effects of drugs, such as height and weight, health status, psychological expectations about the drug's effects, the social setting where the drug is used, and whether the person has taken other drugs at the same time.

Different classes and types of drugs also have different levels of potential for causing dependence on drugs. We have reviewed the various definitions of drug abuse, drug misuse, dependence, and addiction. These definitions vary somewhat according to philosophical and political orientation as well as scientific orientation. Thus, the U.S. government's official agency for drug abuse research, the National Institute on Drug Abuse, defines any use of an illegal drug as *abuse*. In contrast, official professional psychiatric organizations require the presence of a number of conditions and negative effects before terming behavior as drug abuse. These can include loss of a job, family problems, legal issues, and continued use despite negative social or personal consequences.

Clearly, given the generally punitive policies of the criminal justice system, the assumptions there tend more toward treating all illegal drugs as dangerous to the individual and to society, with high potential for abuse. The science of drug addiction, however, provides a specific definition for drug dependence that requires either a physiological change in the brain that triggers withdrawal symptoms when the drug is removed, or evidence of psychological dependence where the user has a compulsion to use and great difficulty in stopping use.

From the point of view of the law enforcement and political systems, the codification of classes of drugs is contained in the scheduling of controlled substances enacted in 1970 as part of the Controlled Substances Act. It is clear that the classification of drugs under these Schedules is not always consistent with medical or scientific evidence. Most notably, marijuana is classified, along with heroin, PCP, and methamphetamine, under Schedule I, which indicates a high potential for abuse and no known medical use. Yet, most research indicates that marijuana, while not a totally harmless drug (especially when used by adolescents) has substantially lower abuse potential and possible medical uses. As discussed in Chapter 10, there is a growing interest in using marijuana for medical purposes, and potential for rescheduling it to Schedule II. It is the Schedule I and II (notably cocaine) drugs that are the principal targets of law enforcement and our criminal justice system. The classification of controlled substances is much more relevant to criminal justice policies and practices than their medical classification.

In contrast to the Controlled Substances Act (and similar state laws), psychoactive drugs are generally classified pharmacologically into five main categories: Cannabis (Marijuana, Schedule I); central nervous system depressants (ranging from Schedule I—heroin—to Schedule V); stimulants (including Schedule I drugs like methamphetamine, Schedule II [cocaine], and other Schedules [various amphetamines]); hallucinogens (mostly in Schedule I); and club drugs (some in Schedule I, such as Ecstasy, GHB, and others in Schedule III).

In the next chapter, we explore the various theoretical perspectives on why individuals use and abuse drugs.

DISCUSSION QUESTIONS

1. Is marijuana a “gateway” drug? Why or why not? What do we mean by “gateway” drug? Is marijuana dangerous? Should it be a focus of law enforcement? Would reducing youth marijuana use reduce initiation into other illegal drugs?
2. Should marijuana be rescheduled to Schedule II under federal law? Why or why not?
3. Discuss the harms caused to society by use and abuse of illegal drugs. How do these harms compare to those caused by legal drugs, such as alcohol and tobacco?
4. What criteria should inform government decisions about which drugs should be legal and under which schedule they should be classified?
5. Is non-prescription instrumental use of stimulants (e.g. Adderall, amphetamines) a problem for society?
6. Should law enforcement focus on the club drug scene?

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