Introduction to Substance Use Disorders for the Eating Disorder Specialist

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Abstract

Substance use disorders and eating disorders co-occur frequently; however, currently, there are no evidence-based treatments to guide the practitioner faced with this comorbid condition. Very few eating disorder programs have incorporated substance abuse protocols into their programs, and likewise, few substance abuse programs can effectively treat the patient with a serious eating disorder. Consequently, clinicians presented with these patients tend to focus on their area of specialty without addressing directly the other comorbid condition. Inadvertently, this can prolong the patient's suffering as they vacillate between their substance use disorder and their eating disorder. The intention of this chapter is to provide a brief overview of substance use disorders for students, clinicians, and researchers in the health and mental health field. It was specifically designed for the eating disorder specialist that has limited knowledge of the psychoactive properties of drugs of abuse, the clinical characteristics of individuals with alcohol and drug abuse problems, the philosophy and/or vernacular of abstinence-based models (e.g., Alcoholics Anonymous [AA], Narcotics Anonymous [NA], or Cocaine Anonymous [CA]), and other evidence-based models/approaches for the treatment of substance use disorders.

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12.1 Introduction

Substance use disorders (SUD) and eating disorders (ED) co-occur frequently; however, currently, there are no evidence-based treatments to guide the practitioner faced with this comorbid condition. Given the high rates of co-occurrence and the complex nature of these disorders, it is surprising to note that most ED clinicians and treatment programs have not incorporated SUD protocols into their practices. Likewise, most substance abuse specialists are not adequately trained in the diagnosis, assessment, and treatment of ED, and very few SUD treatment programs provide comprehensive, integrated services for these dually diagnosed patients. Consequently, clinicians presented with these patients tend to focus on their area of specialty without addressing directly the other comorbid condition. Inadvertently, this can prolong the patient's suffering as they vacillate between their SUD and their ED. Additionally, outpatient clinicians needing to refer patients to a higher level of care or parents seeking treatment for their loved one with both disorders find locating providers or programs that effectively treat both disorders complicated and confusing. The intention of this chapter is to provide a brief overview of SUD for students, clinicians, and researchers in the health and mental health field. It was specifically designed for the ED specialist that has limited knowledge of the psychoactive properties of drugs of abuse, the clinical characteristics of individuals with SUD, the philosophy and/or vernacular of abstinence-based models, and other evidence-based models/approaches for the treatment of SUD. Chapter 11 provides a similar review of ED for the substance abuse specialist. Together, these two chapters are designed to provide a foundation for the reader who is interested in working with individuals who struggle with both ED and SUD. Information on developing a comprehensive, integrated program for these two disorders is discussed in Chap. 21. It is vital that treatment providers in both fields acquire knowledge of the other and that research findings and empirically supported interventions from both specialties be incorporated to create an integrated approach to treatment and recovery (Dennis & Helfman, 2010).

This chapter begins with a discussion of the DSM-5 (American Psychiatric Association, 2013) diagnostic criteria for SUD and a brief discussion of prevalence, clinical characteristics, and medical complications for the following SUD: (1) alcohol; (2) cannabis; (3) sedative, hypnotic, and anxiolytics; (4) stimulants; (5) hallucinogens; (6) opioids; and (7) substances that are frequently used and/or abused by individuals with ED. This will be followed by a definition of recovery, assessment strategies, a review of patient placement criteria (i.e., levels of care),

and psychological and pharmacological interventions for SUD. The chapter will conclude with an examination of the risk factors associated with the development of SUD and a review of the most common psychiatric comorbidities found in individuals with alcohol and/or drug problems.

12.2 Substance Use Disorders

Substance use disorders (SUD) are characterized by a series of cognitive, behavioral, and physiological symptoms that indicate an individual is continuing to use substances without regard to the consequences of repeated use. SUD include ten basic categories: alcohol, caffeine, cannabis, hallucinogens, inhalants, opioids, sedative-hypnotics or anxiolytics, stimulants, and tobacco.

With the publication of DSM-5 (American Psychiatric Association, 2013), a few significant changes have occurred that impact how substance abuse problems are diagnosed. First, the categories of substance abuse and dependence have been collapsed. Second, each diagnosis requires an index of severity (i.e., mild, moderate, or severe). Third, criterion has been grouped into four categories: (1) impaired control, (2) social impairment, (3) risky behavior, and (4) pharmacological criteria. Impaired control implies that the individual is using larger amounts of the substance over longer periods of time than intended; has a desire to reduce or control use but has been unsuccessful; spends a considerable amount of time procuring, using, and recovering from the effects of the substance; and has significant urges to use (craving). Social impairment includes an inability to fulfill role obligations at work, home, or school; significant social or interpersonal problems; and withdrawal from previous relationships, activities, or hobbies in order to use the substance. Risky behaviors include use of the substance in physically hazardous situations and continued use even when faced with persistent physical or psychological problems. Pharmacological criteria include tolerance (requiring increased amounts of the substance to achieve the desired effect) and withdrawal (a significantly unpleasant group of symptoms that occur upon abrupt discontinuation of the substance). Neither of the pharmacological criteria (tolerance or withdrawal) is necessary to make a diagnosis of SUD. Finally, to receive a diagnosis of "mild" SUD, the number of endorsed symptoms has been increased from one to two, and symptoms must lead to clinically significant impairment or distress.

12.3 Alcohol Use Disorders

12.3.1 Prevalence

In 2011, according to the National Survey on Drug Use and Health (SAMHSA, 2012), approximately 52 % of Americans (133.4 million) age 12 and older reported being current drinkers (see Table 12.1). Among that group, a majority was male (57 %). However, the rates of alcohol *use* among youth aged 12–17 were similar for

Table 12.1 2011 Prevalence of substance use and dependence among Americans aged 12 and older

Substance	Use (past month)	Abuse/dependence
Alcohol	133.4	16.7
Illicit drugs	22.5	6.5
Marijuana	18.1	4.2
Psychotherapeutics	6.1	1.8
Cocaine	1.4	0.8
Hallucinogens	1.0	0.3
Inhalants	0.6	0.1
Heroin	0.3	0.4

Adapted from SAMHSA (2012)
Note: Statistics reported in *millions*

both males and females (13 %). The highest rates of alcohol consumption were found in young adults aged 18–25 with approximately 58 % of females and 63 % of males reporting current drinking.

Approximately 16.7 million people (6.5 % of the population age 12 or older) have an alcohol use disorder (AUD) (SAMHSA, 2012). It is estimated that 5 % of adolescents 12–17 years of age and 9 % of adults (18 and older) have AUD. Adult men have higher rates of AUD (12 %) than adult women (5 %), with the highest rates seen in individuals between the ages of 17 and 29 (16 %) and the lowest rates seen in individuals 65 and older (2 %) (American Psychiatric Association, 2013).

12.3.2 Clinical Characteristics

Individuals with AUD have complex and diverse clinical presentations. However, for many individuals, alcohol use and intoxication begins in adolescence. The usual age of first drinking is 15, with the heaviest drinking occurring between age 18 and 22 (Schuckit, 2006). This age pattern is similar between the general population and those who later develop AUD. However, one study found that early-onset drinking (prior to age 15) increased the risk of developing AUD by two to three times compared to individuals that started drinking after age 19 (DeWit, Adlaf, Offord, & Ogbourne, 2000). Early-onset AUD is associated with a family history of alcohol dependence and found in adolescents that have preexisting personality traits of behavioral disinhibition (i.e., an inability to inhibit behavioral impulses, sensation seeking, unconventionality, and rebelliousness) and negative emotionality (i.e., extreme sadness, fear, worry, or anger leading to feelings of isolation, suspiciousness, and interpersonal hostility) (Hicks, Durbin, Blonigen, Iacono, & McGue, 2012). By age 18, more than 60 % of adolescents have experienced drunkenness, and approximately 19 % of individuals between the age of 17 and 20 reported driving under the influence (SAMHSA, 2012).

Alcohol *abuse* often begins in the early to mid-20s with significant signs of AUD apparent in a majority of individuals by the late 30s. In the early stages, daily drinking or frequent binge drinking episodes are common. Drinkers often report a sense of euphoria (“buzz”) after the first few drinks. With continued heavy

drinking, tolerance develops and the individual must increase their consumption to achieve the desired effect. Craving (a strong desire to use the substance) develops and is often triggered by environmental cues (i.e., time of day, bar scenes, parties, celebrations) and/or a need to "self-medicate" or alter negative internal mood states (i.e., life stress, interpersonal conflicts, fatigue, boredom, anxiety, depression). Frequent intoxication can lead to periods of amnesia (blackouts) and debilitating hangovers. Withdrawal symptoms (i.e., tachycardia, psychomotor agitation, insomnia, nausea, diaphoresis, anxiety) often appear between 4 and 12 h after a reduction in alcohol ingestion and often compel the individual to continue consumption to avoid or relieve these unpleasant side effects.

As the illness progresses, occupational, legal, social, psychological, interpersonal, and medical problems become evident. Once a pattern of chronic use develops, the individual becomes increasingly preoccupied with procuring and consuming. Work performance and/or productivity may suffer from drinking on the job or as the result of missed work due to recovering from the effects of alcohol. Legal problems including traffic citations for driving while impaired, arrests due to public intoxication, and domestic violence are common. Daily life maintenance tasks, personal hygiene, and childcare responsibilities may be neglected. Social engagements that do not include alcohol may be avoided, and previously enjoyable activities or hobbies are often abandoned. Profound changes in mood (e.g., depression, anxiety, irritability, aggression, hostility, lability) frequently impact interpersonal functioning and can lead to social withdrawal, violent outbursts and abuse, or suicidal ideations or attempts. Chronic alcohol abuse is associated with significant physical problems (e.g., cirrhosis, ulcers, pancreatitis, cognitive impairment) and may lead to emergency room visits or hospitalizations. Finally, it is not uncommon for individuals with AUD to have other comorbid SUD. Sometimes, alcohol may be used to reduce or eliminate the negative side effects of other drugs (e.g., anxiety, psychomotor agitation, or insomnia from stimulants) or as a substitute when the drug of choice is not readily available.

AUD is a chronic relapsing disorder. Some individuals may have periods of remission (become abstinent) after a crisis (e.g., threat of divorce, loss of a job, arrest, car accident) or a medical emergency (e.g., bleeding ulcers, pancreatitis). However, frequently, these periods of abstinence are followed by controlled or non-problematic drinking but eventually result in the resumption of alcohol abuse and the reemergence of all of the associated consequences.

12.3.3 Medical Complications

Alcohol is considered a central nervous system depressant, and heavy use can have an impact on every organ system in the body. Cardiovascular problems include high blood pressure and an increased risk of an enlarged heart, arrhythmias, heart failure, and stroke. Chronic drinking can also increase LDL cholesterol and increase the risk of cardiomyopathy. Circulatory problems such as anemia (abnormally low number of oxygen carrying red blood cells) can trigger symptoms of fatigue,

light-headedness, and shortness of breath. Numerous gastrointestinal problems can result from alcohol abuse including digestive problems (gastritis, stomach, and esophageal ulcers), pancreatitis, alcohol hepatitis, and cirrhosis. The nutritional status of the individual with moderate to severe AUD may be significantly compromised. Chronic heavy drinking is associated with malnutrition, vitamin deficiencies, and impaired absorption, metabolism, and utilization (See Chap. 23). The neuroskeletal system can also be disrupted, as excessive alcohol use can interfere with the production of new bone and can lead to a reduction in bone density (osteopenia or osteoporosis) and an increased risk for fractures. AUD can affect the nervous system causing peripheral neuropathy (numbness and pain in the hands and feet), cognitive impairment (problems with short-term memory, learning, abstraction, and problem solving), and mild anterograde amnesia (blackouts) or severe anterograde amnesia (Wernicke–Korsakoff syndrome) (See Chap. 15).

12.4 Cannabis Use Disorder

Cannabis is the scientific term for the psychoactive substance derived from the plant Cannabis sativa. The chemical compound it contains that produces the subjective "high" is delta-9-tetrahydrocannabinol (THC) (Budney, Vandrey, & Fearer, 2011). Cannabis is the most frequently used illegal substance worldwide, with approximately 4 % of the adult world population (162 million people) using cannabis annually and 0.6 % (22.5 million) using cannabis daily (United Nations Office on Drugs and Crime, 2006). During the last several decades, over 200 slang terms have been used to describe cannabis including marijuana, Mary Jane, weed, pot, grass, dope, and ganja.

Epidemiological, laboratory, and clinical studies have demonstrated the existence, increasing prevalence, and clinical significance of cannabis use disorders. In 1999, the Institute of Medicine released a report describing the potential negative effects of cannabis including addiction, but also provided a clear statement regarding its potential medical benefits. Currently, in the USA, cannabis remains classified as a *Schedule 1 Substance* (i.e., a substance with high abuse potential that has no accepted medical uses or accepted standards for safe use under medical supervision). However, numerous states have passed laws "legalizing" the medical use of cannabis. In 2012, 66 % of voters approved the legalization of cannabis in Colorado where there are currently more dispensaries for marijuana-infused products (MIP) than liquor stores, Starbucks, or public schools (Osher, 2010).

Controversy regarding its legal status, addictive potential, health consequences, and medical use has pervaded the lay and scientific communities for decades. Ambivalence and debate will likely continue into the foreseeable future.

12.4.1 Prevalence

In 2011, approximately 18.1 million Americans aged 12 and older were current users of cannabis, and it is used by 80.5 % of all illicit drug users. Additionally, 2.6 million persons aged 12 or older used cannabis for the first time within the past year (approximately 7,200 new users every day) (SAMHSA, 2012) (see Table 12.1). The average age of first use among persons aged 12–49 was 17.5 years. Also in 2011, the number of cannabis users in the USA increased from 17.4 million to 18.1 million, and it has the highest abuse rates of any psychoactive substance.

12.4.2 Clinical Characteristics

Cannabis is sold and administered in preparations that vary in potency. The most common preparation is the dried plant form and ranges from 1 to 15 % THC concentration. Hashish refers to the *resin* of the plant which typically contains 10–20 % THC, but may reach as high as 60 % THC. In most instances, cannabis is burned and the smoke inhaled. Cannabis is also dissolved and ingested in the form of candy and baked goods. The cannabis "high" typically occurs within 1 min, reaches a peak in 15–30 min, and persists for up to 4 h (Budney et al., 2011). Subjectively, the user feels an initial euphoric effect characterized by a sense of relaxation. Perception is altered such that time seems to slow and many users report an increased ability to hear, visualize, and appreciate sights and sounds such as artwork, movies, and music. Feelings of anxiety, fear, and paranoia have also been reported in novice users or following the inhalation or ingestion of a higher than usual dose.

Cannabis use is less likely to lead to physical dependence than most other common illicit drugs. In the USA, it is estimated that 9 % of those who try cannabis become dependent compared to 17 % who try cocaine and 23 % who try heroin (Anthony, 2006). Risk of dependence is greater for those who use cannabis more frequently, for those who start using at an earlier age, and for those who have a family history of SUD (Copeland & Swift, 2009). Studies of chronic cannabis users suggest that sustained use may impair attention, memory, and complex cognitive abilities such as problem solving and mental flexibility (Solowij et al., 2002). Abrupt cessation of daily or near-daily cannabis use results in the onset of a cannabis withdrawal syndrome. Common symptoms include anger, anxiety, irritability, decreased appetite or weight loss, restlessness, sleep difficulty, and depressed mood. Withdrawal symptoms generally occur within the first 24 h and last approximately 1–2 weeks (Budney et al., 2011).

In an effort to more definitively understand the physiological and psychological appeal of cannabis, a number of neurobiological studies have been conducted which focus on the effects of THC in brain areas and systems associated with reward, reinforcement, and addiction. THC appears to enhance dopamine (DA) neuronal firing and synaptic DA levels in the reward pathway of the brain. Congruent with these findings, abrupt cessation of chronic THC exposure decreases DA. This effect

has been linked to the dysphoric effects associated with withdrawal from drugs such as alcohol, opiates, and cocaine (Gardner, 2005).

12.4.3 Medical Complications

It is uncommon to require medical care for acute toxicity although potential side effects include drowsiness, dizziness, tachycardia, dysphoria, and on rare occasions, visual hallucinations and drug-induced psychosis. Although rare, case reports have been published suggesting that the acute cardiovascular effects of cannabis can contribute to cardiac-related fatalities (Budney et al., 2011).

12.5 Sedative, Hypnotic, or Other Anxiolytic Use Disorder

This classification of drugs includes benzodiazepines, nonbenzodiazepines, and barbiturates. Like alcohol, these agents are central nervous system depressants. At lower doses, these drugs reduce anxiety and promote sedation; however at high doses, they can produce stupor, amnesia, coma, and death. These drugs can be obtained by prescription or illegally. Both individuals who are prescribed these medications and those who use them recreationally can develop a SUD.

Benzodiazepines. The most common sedative-hypnotic drugs in this category include diazepam (Valium), oxazepam (Serax), clonazepam (Klonopin), lorazepam (Ativan), alprazolam (Xanax), and temazepam (Restoril). These medications are primarily used as muscle relaxants, anticonvulsants, short-term antianxiety agents, sleep aides, and in alcohol withdrawal.

Nonbenzodiazepine ("Z-drugs"). The pharmacodynamics of this class of sedative-hypnotic drugs is nearly the same as benzodiazepine and therefore has similar benefits, side effect profiles, and risks. They have entirely different chemical structures than benzodiazepines and have demonstrated efficacy in treating sleep disorders. There are three primary groups of Z-drugs: (1) imidazopyridines, zolpidem (Ambien, Intermezzo, Stilnox); (2) cyclopyrrolones, zopiclone (Imovane, Zimovane, Imrest) and eszopiclone (Lunesta); and (3) pyrazolopyrimidines, zaleplon (Sonata, Starnoc). As compared to benzodiazepines or barbiturates, Z-drugs promote sleep at doses that produce lower levels of residual sedation and impairment in psychomotor or cognitive functioning, have less pronounced amnesic and antianxiety effects, and are generally considered safer, especially in over dosage. Additionally, they are less likely to produce physical dependence and addiction, although these issues can still occur (Touitou, 2007).

Barbiturates. Prior to the introduction of benzodiazepines, barbiturates were used as anxiolytics, sedative-hypnotic agents, and anticonvulsants. Ultrashort-acting barbiturates such as thiopental sodium (Pentothal) and thiamylal (Surital) are used intravenously to produce unconsciousness in surgical patients. Short-acting barbiturates (3–6 h) such as pentobarbital (Nembutal) and secobarbital (Seconal) are used as sleeping aids. Barbiturates with intermediate duration (6–12 h) include amobarbital (Amytal) and butabarbital sodium (Butisol) and are prescribed to relieve insomnia. The longer-lasting barbiturate phenobarbital (Solfoton) is used primarily to treat and prevent seizures. Slang terms for barbiturates include barbs, dolls, downers, goofballs, and sleepers (Hamid, El-Maliakh, & Vandeveir, 2005).

12.5.1 Prevalence

The 12-month prevalence of sedative, hypnotic, or anxiolytic use disorder is estimated to be 0.3 % among 12- to 17-year-olds and 0.2 % among adults age 18 years and older. The 12-month prevalence rate decreases as a function of age and is greatest among 18- to 29-year-olds (0.5 %). There are differences in the prevalence of sedative, hypnotic, or anxiolytic use disorder across racial/ethnic subgroups in the US population. For adolescents, the rates are greatest among whites (0.3 %). Among adults, the prevalence is greatest among Native Americans and Alaska Natives (0.8 %) (American Psychiatric Association, 2013).

12.5.2 Clinical Characteristics

Drugs in this classification have a potential for misuse if the drug has strong euphoric or mood-altering effects. Among the drugs in this classification, barbiturates produce the greatest pleasant mood alterations (Ciraulo & Knapp, 2011). Recreational users report that barbiturates promote relaxation, contentment, and euphoria. Sedative, hypnotics, and anxiolytics enhance the effects of the more sedating amino acid brain neurotransmitter, gamma-aminobutyric acid (GABA) (Breier & Paul, 1990).

These medications are primarily used in the treatment of insomnia, seizure disorders, and anxiety disorders. They are also prescribed to patients going through alcohol withdrawal and early in the course of treatment of an ED in an attempt to relieve extreme anxiety reported during efforts to return to normal eating behavior. However, even when carefully prescribed and monitored, they can often confound clinical presentations of mood and anxiety disorders at usual dosage ranges. Tolerance often develops rapidly, particularly to the sedating effects of these drugs. As a result, some patients increase their dosage levels (with or without prescriber direction)—even those patients without clinical or family histories of SUD.

12.5.3 Medical Complications

Compared with the other types of sedative-hypnotics, benzodiazepines are relatively safe in overdose but can be particularly dangerous when mixed with alcohol, barbiturates, opioids, or tricyclic antidepressants. The most common symptoms of benzodiazepine overdose include central nervous system (CNS) depression, ataxia, slurred speech, impaired balance, and respiratory depression. Toxic reactions of long-term abuse are characterized by decreased CNS, cardiac, and respiratory functioning; sedation; confusion; disorientation; and impaired memory. The clinical picture resembles severe alcohol intoxication. Although toxic reactions should not be taken lightly, deaths are relatively rare (<1 %) (Schuckit, Smith, Kramer, Danko, & Volpe, 2002).

Z-drugs are notable for producing side effects such as pronounced amnesia and more rarely hallucinations, especially when used in large doses. On rare occasions, these drugs can produce a fugue state where the patient reports sleepwalking or performing acts such as cooking a meal or driving a car while effectively unconscious and with no memory of the event upon waking (Stone, Zorick, & Tsuang, 2007). Daytime-related anxiety can also occur from chronic nightly usage (Fontaine, Beaudry, Le Morvan, Beauclair, & Chouinard, 1990).

The barbiturates produce depression of central nervous system activity that can range from mild sedation to coma. They are not selective in their actions, and their antianxiety effects are not separable from their other depressant effects. Respiratory depression may be a major contributing factor to death in cases of barbiturate overdose (Ciraulo & Knapp, 2011) (See Chap. 15).

12.6 Stimulant Use Disorders

Stimulants are a class of drugs that are prescribed for the treatment of narcolepsy, attention deficit hyperactivity disorder (ADD/ADHD), treatment-resistant depression, HIV-related neuropsychiatric symptoms, and obesity (Hill & Weiss, 2011). Central nervous stimulants activate the psychological reward system in the brain and increase the release of dopamine (DA) resulting in pleasurable effects. Prescription stimulants, including amphetamines (e.g., Dexedrine and Adderall), methamphetamine (e.g., Desoxyn) and methylphenidate (e.g., Ritalin and Concerta), lisdexamfetamine dimesylate (e.g., Vyvanse), and their illegal derivatives including cocaine (also known as coke, coca, snow, flake, or blow), crack cocaine (also known as rock, iron, base, work, cavvy), powder or crystal methamphetamine (also known as meth, crystal, ice, glass, crank, tina), and synthetic cathinones/mephedrone (also known as bath salts, ivory wave, purple wave, bliss, vanilla sky, bloom, red dove, Scarface), are frequently used and abused recreationally by individuals that are not being treated for the above conditions.

Historically, there have been four high-risk groups that have used stimulants to increase energy, improve performance, or lose weight: the military, students, athletes (Hill & Weiss, 2011), and dieters. Since World War II, military forces

have been prescribed stimulants to increase performance and reduce combat fatigue. Nonmedical use of stimulants by students to increase energy levels, decrease the need for sleep, and assist with studying for prolonged periods of time is common. Stimulants and over-the-counter amphetamine "look-alikes" (e.g., caffeine, ephedra, bitter orange, modafinil and adrafinil) are routinely used by collegiate and professional athletes to increase performance, concentration, and reduce weight (McDuff & Baron, 2005). However, individuals in any profession that requires prolonged periods of concentration (e.g., pilots, air traffic controllers, long haul truck drivers) are at risk for stimulant abuse. Finally, both prescription (i.e., ADHD medications and antiobesity drugs) and illicit stimulants (i.e., cocaine, methamphetamine) are used to promote weight loss in dieters. Phentermine, a noradrenergic sympathetic amine, approved by the FDA, is the most widely prescribed medication for the short-term treatment of obesity in the USA. However, there is a high potential for abuse with these drugs (Cochrane, Malcolm, & Brewerton, 1998; Hendricks & Greenway, 2011; Jeffers, Benotsch, & Koester, 2013).

12.6.1 Prevalence

According to the National Survey on Drug Use and Health (SAMHSA, 2012), the prevalence rates for stimulant use for persons aged 12 and older are as follows: nonmedical users of stimulants, 0.4 % or 970,000; cocaine, 0.5 % or 1.4 million; and methamphetamine, 0.2 % or 439,000. Males are more likely than females to use stimulants, and persons between the ages of 18 and 25 have the highest use rates (SAMHSA, 2012). Illicit use of prescription stimulants has become a serious issue on college campuses. It appears that the primary motivation for nonmedical use of stimulants is a belief that these medications will improve academic performance (Teter, McCabe, Cranford, Boyd, & Guthrie, 2005). One study found that 8.1 % of undergraduate students attending a large public Midwestern university reported lifetime illicit use of prescription stimulants, which exceeded the number of students on campus who reported medical use of stimulants for ADHD (McCabe, Teter, & Boyd, 2006). They also identified several risk factors for illicit use of prescription stimulants which included being white, male, a member of a fraternity or sorority, Jewish, having a lower grade point average, and perfectionist personality traits (McCabe et al., 2006). Likewise, Tuttle, Scheurichn and Ranseen (2010) found that 10.1 % of medical students in a public medical college reported using nonmedical prescription stimulants to enhance their performance.

12.6.2 Clinical Characteristics

The clinical effects of stimulant use vary depending on the frequency of use patterns (episodic vs. chronic use), potency of the drug, dosage, and route of administration. In low doses, stimulants increase the libido, alertness,

concentration, and energy and reduce appetite and fatigue. In higher doses, they can cause euphoria, mania, insomnia, anxiety, aggressive behaviors, or stimulant psychosis (e.g., hallucinations, delusions, and thought disorders). Chronic use of stimulants often follows a binge-abstinence pattern (taking the drug repeatedly within a relatively short period of time in escalating doses). For example, cocaine binges can last up to 12 h with methamphetamine binges lasting several days (Ciccarone, 2011). Following the binge, the user often experiences psychological and behavioral withdrawal symptoms (referred to as a "crash") including hypersomnia, depression, lethargy, and powerful cravings.

Prescription stimulants are primarily orally administered; however, some non-medical abusers crush the tablets and inhale (snort) them or dissolve them in water and inject them into the blood stream. Likewise, cocaine is inhaled or injected. Crack is a freebase form of cocaine that is smoked. There are several common routes of administration with methamphetamine including intravenous injection, smoking (i.e., vaporizing it to inhale the fumes, not burning it to inhale the smoke), snorting, and the insertion of suppositories in the anal or vaginal track. "Bath salts" can be swallowed, snorted, smoked, or injected. Heavy use is clearly related to dependency, but smoking and/or injecting stimulants (as opposed to ingesting or snorting) are associated with an increased likelihood of escalating tolerance and addiction (Ciccarone, 2011).

Stimulant abuse is also associated with the abuse of other psychoactive substances including heroin (e.g., cocaine combined with heroin and injected, "speedballs"), tobacco, alcohol, and marijuana. Additionally, it is not uncommon for stimulant abusers to use benzodiazepines or other sedative-hypnotics to manage bouts of anxiety, restlessness, or insomnia. Finally, more than 1/3 of individuals with stimulant use disorder have a lifetime mood or anxiety disorder, and at least 13 % report one or more symptoms of psychosis (Sara et al., 2012). Stimulant use disorder may also be associated with other mental disorders including posttraumatic stress disorder, antisocial personality disorder, and gambling disorder (American Psychiatric Association, 2013).

12.6.3 Medical Complications

Cocaine, amphetamine, and methamphetamine abuse can all be marked by toxic cardiovascular complications including, tachycardia, hypertension, and myocardial infarction. Though longitudinal data linking stimulant abuse to cardiovascular disease is lacking, a study by Yeo et al. (2007) found that chronic stimulant dependence heightens the risk of artherosclerotic heart disease, cardiomyopathies, and sudden death from arrhythmias. Neurological complications of abuse include seizures and a risk for hemorrhagic strokes. Neurobehavioral complications include memory/learning impairment, anxiety, paranoia, psychosis, and anorexia.

Cardiac complications specific to cocaine abuse include an increased myocardial oxygen demand via increased heart rate, coronary artery vasoconstriction, and cocaine-associated chest pain. A medical complication of the respiratory tract

includes a nasal septal perforation. The most acute systemic complication from cocaine abuse is muscle breakdown and renal failure (rhabdomyolysis).

Although abusers of "bath salts" experience many of the same symptoms as other stimulant abusers (e.g., headaches, heart palpitations, nausea, hallucinations, panic attacks, breakdown in skeletal muscle tissue, and paranoia), violent behaviors, heart attacks, kidney failure, liver failure, suicide, and an increased tolerance for pain have been reported (National Institute on Drug Abuse, 2012) (See Chap. 15).

12.7 Hallucinogen Use Disorder

Drugs considered hallucinogens are a diverse group of compounds that includes the phencyclidines (or phencyclidine-like substances) of PCP, ketamine, cyclohexamide, and dizocilpine, as well as the phenylalkylamines (e.g., mescaline): the indoleamines (e.g., psilocybin) and the ergolines, such as LSD (American Psychiatric Association, 2013). MDMA (also called "ecstasy") is technically neither a stimulant nor a hallucinogen. It is a member of the class of drugs called entactogens. However, it is listed under the category of "other hallucinogen intoxication" for diagnostic descriptive purpose in the DSM-5 (American Psychiatric Association, 2013). Recreational users of hallucinogens commonly refer to them as "club drugs." Club drugs are licit and illicit drugs from different classes used in bars, clubs, and concerts to enhance the sensory experience. Recently, the potential therapeutic effects of MDMA for PTSD have been studied in a RCT. Mithoefer and colleagues (Mithoefer et al., 2013; Mithoefer, Wagner, Mithoefer, Jerome, & Doblin, 2011) found that the majority of subjects with severe treatment-refractory PTSD had significant and long-lasting symptomatic relief provided by MDMA-assisted psychotherapy. No subjects reported harm from participation in the study.

12.7.1 Prevalence

Hallucinogen use disorder is the least prevalent of all SUD. In the USA, the 12-month prevalence is estimated to be 0.5 % among 12- to 17-year-olds and 0.1 % among adults. Native American and Alaska Native adolescents have the highest rates (1.2 %) of hallucinogen use disorder; among adults, the prevalence is the same for Native Americans, Alaska Natives, whites, and Hispanics (0.2 %) (American Psychiatric Association, 2013).

12.7.2 Clinical Characteristics

Hallucinogens comprise a group of substances having diverse chemical structures and involve different molecular mechanisms that produce an altered state of consciousness. The phencyclidines or phencyclidine-like substances (e.g., PCP,

ketamine) were first developed as dissociative anesthetics in the 1950s and became street drugs in the 1960s. PCP is known colloquially as angel dust, KJ (Kristal joint), illy, or wet. PCP comes in both powder and liquid form ("embalming fluid"), but typically, it is sprayed onto leafy material such as cannabis, mint, or oregano leaves and then smoked.

The term hallucinogen means "producer of hallucinations." A hallucination causes disturbances in judgment, orientation, intellect, memory, emotion, and level of consciousness (Pechnick & Cunningham, 2011). They produce feelings of separation from mind and body. Hallucinogens are most often taken orally, although some forms can also be smoked, snorted, or injected (e.g., ecstasy). Duration of effects varies across types of hallucinogen: LSD and ecstasy have a long duration such that users spend anywhere from hours to days using and recovering. The hallucinogenic effects in vulnerable individuals may last for weeks and precipitate a persistent psychotic episode resembling schizophrenia (McCann, 2011).

It was in the early 1960s when a psychology instructor from Harvard, Timothy Leary, began experimenting with hallucinogens, particularly LSD. He claimed LSD provided happiness, enhanced creativity, increased self-awareness, and might be useful as an adjunct to psychotherapy (Leary, 1997). By the mid-1960s, more than 1,000 articles on LSD appeared in the medical literature. Sandoz Laboratories stopped distributing the drug in 1966 because of the reported adverse reactions and resulting public fear. Today, LSD and the other hallucinogens (PCP, MDMA [ecstasy]) are classified as Schedule I drugs. Ketamine is used therapeutically as an anesthetic and is currently undergoing research for its antidepressant effects (Pechnick & Cunningham, 2011). A meta-analysis of randomized controlled trials (Krebs & Johansen, 2012) found evidence for the beneficial effects of a single dose of LSD, in decreasing alcohol misuse. There is also research supporting the medical use of psilocybin and LSD to terminate cluster headaches (Sewell, Halpern, & Pope, 2006) as well as psilocybin being safely used with acute reductions in core OCD symptoms (Moreno, Wiegand, Tatano, & Delgado, 2006). Clearly, further research examining accepted medical use of hallucinogens is warranted.

12.7.3 Medical Complications

Behavioral effects of PCP can vary by dosage. Low doses produce numbness in the extremities and intoxication, characterized by staggering and slurred speech. In moderate doses, analgesia and anesthesia are observed. Large doses may produce convulsions.

Hallucinogen toxicity from the so-called "bad trip" may include confusion, anxiety, depression, paranoia, drug-induced psychosis, and respiratory depression (especially with concurrent alcohol use). Acute anxiety or panic reactions usually wear off within 24 h. Depression with suicidal ideation can occur several days after LSD use. Psychosis can develop and persist after hallucinogen use, but it remains

unclear whether hallucinogen use can "cause" long-term psychosis or if it has a role in precipitating the onset of the illness (Pechnick & Cunningham, 2011).

12.8 Opioid Use Disorders

Opioid use disorder, as defined by the DSM-5 (American Psychiatric Association, 2013), includes signs and symptoms indicating a compulsive, prolonged self-administration of opioid substances that are used for no legitimate medical purpose or, if used to treat a medical condition, are used in doses in excess of the amount prescribed for that medical condition.

The term opiates refers to morphine and codeine that occur in opium along with many similarly psychoactive derivatives such as heroin, dilaudid, fentanyl, hydrocodone, and oxycodone (Jaffe & Martin, 1991). Heroin is only available illegally in the USA. Opiates such as codeine, hydrocodone, oxycodone, fentanyl, and morphine are commonly used for pain control.

The use of opium for intoxicant purposes and for pain relief appears to date back to at least ancient Greece where routes of administration were oral ingestion or inhalation of opium vapors. Use of opium via smoking appears to have first become popular in China in the 1600s after a ban on tobacco (Brownstein, 1993). In 1898, the Friedrich Bayer Company first synthesized aspirin and two weeks later, heroin. The pharmacologist at Bayer was most interested in finding a cough suppressant with a more benign side effect profile than morphine. Although this was later determined to be a false conclusion, it was believed that heroin stimulated and strengthened the lungs. Heroin and other opiates were sold as medicines for gynecological, respiratory, and nearly every other conceivable indication (Sneader, 1998). Gradually, reports that the drug was habit forming and used as a means of entertainment resulted in the 1914 passage of the Harrison Narcotic Act, and access to heroin was restricted. By 1924, heroin could no longer legally be prescribed.

12.8.1 Prevalence

Among adults age 18 and over, the 12-month prevalence rate of opioid use disorder is approximately 0.37 % (American Psychiatric Association, 2013). Rates are higher in males than in females (0.49 % vs. 0.26 %), with the male-to-female ratio of 1.5:1 for prescription opioids and 3:1 for heroin. Opioid use disorder is lowest among African Americans (0.18 %), average among whites (0.38 %), Asians or Pacific Islanders (0.35 %), and Hispanics (0.39 %) with the highest rates found in Native Americans (1.25 %) (American Psychiatric Association, 2013). In 2011, among persons 12–49 years, the average age at first use was 21.8 years for pain relievers and 22.1 years for heroin (SAMHSA, 2012). There were approximately 178,000 persons aged 12 or older who initiated heroin use and approximately 1.9 million new nonmedical users of pain relievers in 2011. It is important to note that

over five times as many individuals are dependent on prescription opioid pain relievers than on heroin (SAMHSA, 2012).

12.8.2 Clinical Characteristics

Most recreational users of opiates either snort or inject them intravenously, which produces flushing and an intensely pleasurable, diffuse bodily sensation that resembles orgasm. This initial "rush" is followed by a sense of well-being. With chronic use, these positive effects become unreliable (Epstein, Phillips, & Preston, 2011). Eventually, the effects of heroin and morphine become emotionally numbing and deadening (Marlowe, 1994). Signs of being "high" or intoxicated occur immediately and include psychomotor retardation, drowsiness, inactivity, impaired concentration, and constriction of the pupils, respiratory depression, and slurred speech. Nausea, vomiting, and constipation are common after repeated opiate use. Opiate abusers quickly build tolerance to their drug of choice, requiring increasing doses to achieve the desired effect, which can lead to unintentional overdosing. Withdrawal symptoms usually begin 8–10 h after the last dose and can be highly uncomfortable with a flu-like syndrome that is not life threatening. The most common symptoms are yawning, watery eyes, runny nose, chills, muscle aches, nausea, diarrhea, and cravings. Eating and sleeping are disrupted, and a fever may persist for as long as 2 weeks (Himmelsbach, 1942).

12.8.3 Medical Complications

Opioid use is associated with the slowing of gastrointestinal activity and a decrease in gut motility that produces severe constipation. Persons who sniff or "snort" heroin or other opioids may develop irritation of the nasal mucosa, sometimes resulting in perforation of the nasal septum. Difficulties in sexual functioning are commonly reported with males experiencing erectile dysfunction and females having irregular menses. In those who inject opioids, veins can become severely sclerosed resulting in peripheral edema. When veins become unusable, individuals often inject directly into their subcutaneous tissue with the result of an increased risk of infections such as cellulitis and tetanus. The use of contaminated needles by opioid injectors puts the user at risk for infections that include bacterial endocarditis, hepatitis, HIV infection, and tuberculosis. Infections are less common in opioid use disorder with prescription opioids. The variability in the development of opioid tolerance, as well as the fluctuating levels of purity of illicit opioids, may help explain why even opioid users with some degree of tolerance may experience severe opioid overdoses. The tendency to combine other drugs such as alcohol or sedatives with opioids may also contribute to overdosage (Jaffe, 1992). Opioid overdose is a medical emergency. Signs of acute opioid toxicity include up to complete and unresponsive coma, severe respiratory depression, and pinpoint pupils. There is pulmonary edema associated with the severe respiratory depression

(Jaffe, 1992). Opioid use disorder is associated with a mortality risk as high as 1.5–2 % per year. Death most often results from overdose, accidents, injuries, AIDs, or other general medical complications (American Psychiatric Association, 2013). Opiate overdose can be reversed by opiate antagonists (e.g., naltrexone, nalmefene, and naloxone) (See Chap. 15).

12.9 Substances Frequently Abused by Individuals with Eating Disorders

In addition to alcohol, cannabis, stimulants, sedative-hypnotics, opiates, hallucinogens, and club drugs, individuals with ED frequently abuse over-the-counter drugs, prescription medications, and performance enhancers.

12.9.1 Over-the-Counter Medications

A brief reminder to the reader is indicated in this discussion of over-the-counter medications. Below will be a brief overview of commonly used and abused substances (laxatives, diuretics, and diet pills) by individuals with ED. The term "over the counter" is used to describe substances and preparations that can be purchased in a pharmacy without a prescription, but also include products that can be bought in grocery stores, health food stores, or over the Internet. Unfortunately, these "dietary supplements," "herbal remedies," "energy boosters," "fat busters," and "colonics" are not under the jurisdiction or regulation of the US Food and Drug Administration (FDA) and therefore are not required to demonstrate safety or efficacy. There are serious and potentially life-threatening complications from the chronic use of some of these products (Consumer Reports, 2012; Mascolo, Chu, & Mehler, 2011; Steffen, Mitchell, & Roerig, 2007). Treatment providers should routinely inquire about the use of these medications as patients frequently do not spontaneously disclose the use of over-the-counter products or herbal supplements.

12.9.1.1 Laxatives

Laxatives are the most commonly abused over-the-counter medications used by individuals with ED (Mitchell, Specker, & Edmonson, 1997). In the last decade, prevalence rates for laxative use in ED outpatients have ranged from 26 % (Bryant-Waugh, Turner, East, Gamble, & Mehta, 2006) to 67 % (Steffen et al., 2007).

Despite their ineffectiveness as a weight reduction strategy, individuals with ED report using laxatives to manage anxiety, combat feelings of fullness or bloating, and promote weight loss. It is a common misperception among individuals with ED that laxative use will prevent caloric absorption; however, laxatives do not act on the small intestine where most of the absorption takes place. Any appreciable weight reduction is primarily due to significant fluid loss.

Laxatives can be divided into categories based on their mode of action—(1) stimulants, (2) osmotic agents, (3) lubricants, (4) bulk forming, and (5) softening

(Colton & Woodside, 1999)—and are sold over the counter and increasingly marketed on the Internet as "herbal remedies." Unfortunately, tolerance builds with long-term laxative use, requiring larger and more frequent dosages to obtain the desired results. Laxative abuse can lead to chronic constipation, severe dehydration, edema, bleeding, cathartic colon, electrolyte abnormalities, impaired bowel function, and rectal prolapse.

Most treatment protocols recommend immediate discontinuation of laxative use with no weaning or tapering. During the initial phase of withdrawal, patients often experience fluid retention and rebound edema. Bulk-forming agents such as Metamucil and Prodiem with at least 250 ml of water with each dose are recommended (Colton & Woodside, 1999). Additionally, patients may need increased contact with treatment providers and continued reassurance that a return to normal bowel functions may take up to two weeks.

Clinically speaking, ED patients that abuse laxatives may represent a more pathologically complex subgroup. Relative to non-abusers, ED patients that abuse laxatives are more likely to have symptoms of borderline personality disorder (Johnson, Tobin, & Enright, 1989; Tobin, Johnson, & Dennis, 1992), multi-impulsive behaviors including greater self-injury, and poly-substance abuse (Favaro & Santtonastaso, 1998; Wiederman & Pryor, 1996), a history of sexual abuse (Pryor, Wiederman, & McGilley, 1996), higher rates of depression requiring hospitalization, and past suicide attempts and are significantly more likely to also use diuretics, enemas, and diet pills (Mitchell, Boutacoff, Hatsukami, Pyle, & Eckert, 1986). Waller, Newton, Hardy, and Svetlik (1990) found that laxative abuse was correlated with higher body dissatisfaction and drive for thinness in patients with bulimia nervosa (BN). Finally, two studies have reported that laxative use is a predictor of poor treatment response in patients with BN (Blouin et al., 1994; Maddocks & Kaplan, 1991).

12.9.1.2 Diuretics

Individuals with ED also abuse diuretics. Approximately 31 % of BN patients report diuretic use for weight control purposes (Roerig, Mitchell, & de Zwaan, 2003). Diuretic use is often initiated to manage premenstrual fluid retention. Individuals with ED most commonly abuse over-the-counter diuretics; however, there is a subgroup of ED patients that abuse highly potent prescription diuretics (Pomeroy, Mitchell, Seim, & Seppala, 1998).

Similar to laxatives, diuretics are ineffective in preventing weight gain. Chronic diuretic use can cause nausea, abdominal pain and constipation, polyuria, heart palpitations, fluid and electrolyte imbalances, and kidney damage. Discontinuation of diuretics can cause rebound edema and weight gain. In a majority of cases, patients can be tapered off diuretics over the course of several days. Patients are encouraged to restrict their sodium intake and elevate their legs. However, if there is evidence of significant electrolyte abnormalities (e.g., hypokalemia), treatments need to be individualized and hospitalization may be required (Mitchell, Pomeroy, & Huber, 1988).

12.9.1.3 Appetite Suppressants

Other substances frequently used and abused by individuals with ED are various appetite suppressants including diet pills, caffeine, nicotine, and artificial sweeteners.

One study found that rates of diet pill use among ED patients rose 12 % between 1985 and 2003 (e.g., from 52 to 64 %, respectively) (Roerig et al., 2003). The most common ingredients found in over-the-counter diet aids include ephedrine (Ma Huang), caffeine, chromium, bulk forming, and stimulant laxatives among others. These substances can cause serious medical complications including elevated blood pressure, insomnia, tachycardia, depression, renal failure, neurological problems, stroke, seizures, and cerebrovascular hemorrhage.

In 1999, prescription strength orlistat (Xenical at 120 mg) was approved by the FDA as a weight loss medication to treat obesity. In 2007, the FDA approved the first nonprescription weight loss medication, orlistat (Alli at 60 mg) for over-the-counter use. A large multisite study found that 6 % of ED patients use Alli with approximately 23 % admitting that they had exceeded the maximum recommended dosage (Steffen et al., 2010). They also reported that a majority of Alli users were bulimic or binge eaters (as opposed to restricting anorexics) and were more likely to also use laxatives, diuretics, other diet pills, syrup of ipecac, and herbal fat burners as weight loss aids (Steffen et al., 2010).

Alli is an intestinal lipase inhibitor that works by disrupting the absorption of fat in the small intestines and reduces the absorption of fat-soluble vitamins. Potential side effects associated with the use of orlistat include abdominal pain, flatulence, soft stools, steatorrhea (excessive fat in the stool causing an oily appearance), and fecal urgency/incontinence.

In addition to the use of diet pills, individuals with eating disorders also use nicotine and caffeine to suppress appetite. In a large study of 1,206 monozygotic and 877 dizygotic adult female twins, researchers reported that regular smoking and caffeine disorder were the most prevalent SUD found in women with ED (Baker, Mitchell, Neale, & Kendler, 2010). Approximately 26 % of AN subjects and 23 % of BN subjects met the criteria for caffeine disorder, and 52 % of AN subjects and 45 % of BN subjects were regular smokers. These researchers found that rates of caffeine disorder and regular smoking were more prevalent in women with AN compared to BN (Baker et al., 2010).

Finally, individuals with ED frequently use large quantities of artificial sweeteners with the belief that these products will facilitate weight loss and/or prevent weight gain. In a study by Ohlrich, Aughey, and Dixon (1989), investigators found that 18 out of 21 consecutive ED patients reported the daily use of sorbitol (i.e., sugar-free gum). A more recent study by Klein, Boudreau, Devlin, and Walsh (2006) assessed weekly use of chewing gum; artificially sweetened, low calorie beverages; and packets of artificial sweetener in female patients with ANR, ANBP, and BN. On average, weekly pieces of gum chewed were ANR = 31, ANBP = 27, and BN-31; weekly consumption of 12-oz servings of diet beverages were ANR = 16, ANBP = 40, and BN = 25; and weekly number of packets of artificial sweetener used were ANR = 350, ANBP = 101, and BN = 39.

Taken together, it appears that women with ANBP and BN (the two groups that endorse purging) tend to report a higher use of gum and diet beverages than women with ANR. However, women with ANR reported a much higher use of artificial sweetener packets than either of the two purging groups (Klein et al., 2006).

12.9.2 Prescription Medications

12.9.2.1 Insulin

A meta-analysis of the existing studies on ED individuals with type 1 diabetes found that females with type I diabetes are three times more likely to have BN and two times more likely to have BED compared to their nondiabetic peers (Nielsen, 2002). Approximately 0–11 % of females with type 1 diabetes meet the criteria for a full syndrome ED, most commonly BN or BED, with another 7–35 % reporting symptoms of subthreshold ED (Colton, Rodin, Bergenstal, & Parkin, 2009). Adolescent girls and women with type 1 diabetes tend to have a higher BMI than nondiabetic peers, which may lead to body dissatisfaction, desire to lose weight, and an increased risk of ED (DCCT Research Group, 2001).

Intentional omission of insulin (also known as insulin purging) is a common phenomenon among individuals with ED and type 1 diabetes. Approximately 6 % of type 1 and 2.2 % of type 2 diabetics reported deliberate omission of insulin to control or lose weight (Herpertz, Albus, & Kielman, 2001). Complications of poor glycemic control can include ketoacidosis, vision problems, neuropathy, hearing loss, hypertension, kidney disease, and stroke.

It is important to note that several aspects of the standard protocol for the treatment of diabetes conflict with customary ED interventions. Diabetes treatment requires constant checking of glucose levels before each meal and snack to determine the amount of insulin that should be administered in order to prevent hypo- or hyperglycemia. Patients are educated to focus on food choices, reduce/avoid certain foods, and make lifestyle changes that often include reduced caloric intake and weight loss and encouraged to engage in regular physical activity (American Diabetes Association, 2008).

The co-occurrence of these disorders can negatively affect the course and treatment of an ED. If insulin abuse is suspected, clinicians are advised to work closely with the patient's endocrinologist, who should be considered for inclusion on the patient's treatment team.

12.9.2.2 ADHD Medications

Attention deficit hyperactivity disorder (ADHD) frequently co-occurs in individuals with ED (Ptacek, Kuzelova, & Stepankova, 2010) and in those with SUD (Brook, Brook, Zhang, & Koppel, 2010). Methylphenidate (i.e., Ritalin), amphetamine (i.e., Dexedrine, Adderall), and lisdexamfetamine dimesylate (i.e., Vyvanse) are frequently prescribed for the treatment of ADHD and used to improve concentration and reduce restlessness and impulsivity. However, one of the major side effects of these ADHD medications is reduced appetite and weight loss which

can lead to abuse by individuals with ED. Approximately 33 % of adults with ADHD have histories of AUD, and 20 % have histories of drug use disorders (Waid, LaRowe, Anton, & Johnson, 2004). Another study found that adolescent girls with ADHD were 5.6 times more likely to develop BN and 2.7 times more likely to develop AN than their non-ADHD peers (Biederman et al., 2007).

12.9.2.3 Levothyroxine

Levothyroxine (i.e., Synthroid, Levothroid, Levoxyl, Tirosint, Unithroid) is a medication prescribed to replace a hormone normally produced by the thyroid gland to regulate metabolism and the body's energy. Little is known about the rates of abuse of thyroid medication for weight loss in ED patients; however, there are several anecdotal reports in the literature (Crow, Mitchell, & Kendall, 1997; Fornari, Edleman, & Katz, 1990; Schmidt & O'Donoghue, 1992). An underactive thyroid often produces weight gain, and excessive self-administration of thyroid medication is a weight control strategy used by some patients with ED. Clinicians should routinely screen for levothyroxine use in their ED patients and be aware of the signs and symptoms of hyperthyroidism which include increased basal metabolic rate, polyphagia, weight loss, heat intolerance and sweating, tachycardia and arrhythmias, fatigue, muscle weakness, and simple tremors to severe myopathy (Crow et al., 1997).

12.9.3 Performance Enhancers

12.9.3.1 Steroids

ED or disordered eating is common in athletes and most prevalent in athletes that participate in sports where aesthetics are critical (i.e., dancing, figure skating, diving, gymnastics, body building), "making weight" is necessary to compete (i.e., wrestling, jockeys, rowing), and low body fat is perceived to improve performance (i.e., track and field, cross-country, swimming) (Baum, 2000). Performance-enhancing drugs (i.e., steroids, growth hormones, stimulants, and sports supplements) are often used by athletes to increase strength, decrease fatigue, and build muscle. Many of these substances have been banned by sports-regulating authorities (e.g., International Olympics Committee, Tour de France) and are illegal; however, some are available by prescription or can be purchased over the Internet.

Approximately 13.5 % of female athletes have a diagnosable ED (Sundgot-Borgen & Torstveit, 2004), and roughly 3 % use anabolic steroids to enhance their performance (Johnson, Powers, & Dick, 1999). Muscle dysmorphia is a subtype of body dysmorphic disorder which has been linked to both ED and steroid abuse in men (Strother, Lemberg, Stanford, & Turberville, 2012) (See Chap. 20).

The long-term effects of steroid abuse include adverse cardiovascular effects including hypertension, cardiomyopathy, and arrhythmias; neuroendocrine effects including suppression of the hypothalamic-pituitary-testicular axis, infertility, and prostatic hypertrophy; and neuropsychiatric effects including hypomanic or manic

symptoms, aggressive and/or violent behavior, and depression (Kanayama, Hudson, & Pope, 2008). Withdrawal from steroids should be medically monitored and can cause severe mood swings, depression, suicidal ideations, fatigue, restlessness, loss of appetite, insomnia, reduced libido, and "cravings" for the drug.

12.10 Definition of Recovery

Successful treatment and resultant recovery from a SUD can be defined in a multitude of ways. In the substance abuse field, historical definitions of recovery revolve around complete and sustained abstinence. A broader definition offered by O'Brien and McKay (2007) conceptualizes successful treatment as one that leads to significant reduction in substance use and ultimately to an improvement in the patient's ability to function in society. This definition takes into account the chronicity and remitting nature of SUD and builds on the view of addiction as a disease, much like diabetes. In this model, the aim of treatment is to manage as opposed to cure the disease. For example, individuals that *do not meet* the criteria for AUD but engage in episodes of problem drinking may not be willing to accept abstinence as the goal for treatment.

12.11 Levels of Care

SUD are complex psychiatric disorders that are heterogeneous in etiology and clinical presentation. As a result, this population varies widely, and no single treatment approach or level of care can effectively or efficiently accommodate the diverse clinical needs of these patients. Unfortunately, even today, the general public and many health and mental health-care professionals believe that there is a universally accepted, standardized approach to the treatment of alcohol and drug abuse problems (i.e., 28-day residential treatment). However, the "one size fits all" approach to treatment is neither cost-effective nor consistently successful in producing positive treatment outcome. As a result, the American Society of Addiction created patient placement criteria (ASAM PPC) for the treatment of substance-related disorders, which has undergone two revisions (ASAM PPC-2R) and currently includes criteria for people with co-occurring mental and SUD (Mee-Lee, Shulman, Fishman, Gastfriend, & Griffith, 2001). The purpose of the patient placement process is to match the patient to a specific setting and intensity of treatment by conducting a comprehensive assessment of the severity of the patient's illness and level of functioning. This assessment is conducted upon admission, during the continued stay review, and at discharge (Gastfriend & Mee-Lee, 2011). The current version of the PPC-2R lists five levels of care:

Level 0.5: Early Intervention. This is considered a pretreatment level of care for individuals that have risk factors or problems associated with substance use. Interventions are designed to help the individual recognize the negative

consequences of their substance use and gain skills and strategies to avoid future problems with drug and/or alcohol use.

Level I: Outpatient Treatment. This level of care usually consists of one or two weekly sessions of individual and/or group sessions for individuals with less severe symptoms of substance abuse or for individuals who need continued support for ongoing recovery. The focus of treatment is lifestyle, attitudinal, and behavioral changes that are necessary to reduce the negative consequences associated with substance abuse or issues that could promote relapse.

Level II: Intensive Outpatient/Partial Hospitalization. Intensive outpatient treatment (IOP) or partial hospitalization (PHP) programs provide extended mental health services (9–70 h per week) during the day, after work, in the evenings, and on weekends; however, patients reside at home. This level of care is designed to provide a higher intensity of programming, increased contact with clinical staff, and more support for individuals with SUD than traditional outpatient treatments. The basic services provided by this level of care include medical, psychiatric, and psychopharmacological consultation, medication management, and 24-h crisis services. Upon the completion of residential or inpatient treatment, individuals with SUD are often referred to this level of care to continue the recovery process. However, if medically stable, a patient may be able to start treatment in a "robust" (i.e., 70 h per week with supportive housing) PHP level of care.

Level III: Residential/Inpatient Services. This level of care is provided to individuals that require 24-h, supervised "live-in" care to prevent imminent danger or the negative consequences of continued substance use. Residential services include medication management, counseling and psychoeducation, and skills building to help individuals safely transition to less restrictive levels of care.

Level IV: Medically Managed Intensive Inpatient Services. This level of care is reserved for individuals that have severe mental health and substance-related problems that require 24-h medical management and/or require medically supervised detoxification services. Staffing consists of addiction medicine physicians, skilled nursing staff, and other mental health clinicians who provide specialized biomedical, psychiatric, and nursing services.

This continuum of care is fluid, and patients can move back and forth through the levels depending on their specific needs. Additionally, length of stay in each level of care is not static and depends on severity of illness and progress/response to treatment.

12.12 Assessment

A thorough assessment for SUD should include (1) a systematic and detailed evaluation of current and past alcohol and other substance use and abuse, (2) a comprehensive psychiatric evaluation to determine the presence and extent of other comorbid psychiatric conditions, (3) a physical examination with laboratory tests and drug screens, (4) a review of the individual's environment to determine the

strength of their social support network and determine barriers to treatment, (5) determination of stage of change for each problem, and (6) a discussion of the individual's response to previous psychological and/or pharmacological treatments. A systematic assessment should culminate in a comprehensive treatment plan and assignment to the appropriate level of care.

In addition to the diagnostic criteria set forth in the DSM-5 (American Psychiatric Association, 2013), there are several screening tools that are available to determine the frequency and intensity of alcohol/drug use, symptoms of dependence, and substance-related consequences (screening instruments are detailed elsewhere; See Chap. 14).

A comprehensive psychiatric evaluation should include attention to the most common comorbid conditions found in SUD including mood disorders, anxiety disorders, ED, trauma- and stress-related disorders (particularly posttraumatic stress disorders [PTSD]), impulse control disorders, and personality disorders (i.e., antisocial and borderline personality disorder [BPD]). The identification of all comorbid conditions will assist the clinician in the selection of both psychological and psychopharmacological interventions (Dennis & Sansone, in press). It is not uncommon for individuals (particularly women) with SUD to have multiple comorbid psychiatric conditions (SAMHSA, 2005). Approximately 30 % of alcoholic men (Helzer & Pryzbeck, 1988) and 60–70 % of alcoholic women (Swendsen et al., 2010) have preexisting psychiatric disorders; however, comorbid conditions can occur before, during, or after the onset of an SUD. Additionally, SUD can mask underlying mental illness, which may fully emerge with abstinence from drugs/alcohol.

A complete physical exam should include a review of each organ system to determine the presence and extent of medical complications due to substance abuse. Additionally, laboratory testing at the onset of treatment can provide valuable information to the patient that denies or minimizes their substance use. Periodic testing throughout treatment can also alert the clinician to signs of relapse (See Chap. 15).

Understanding the individual's current social support system and what they perceive as barriers to treatment can assist the clinician in treatment planning, selection of treatment modality, and relapse prevention (Longabaugh, Wirtz, Beattie, Noel, & Stout, 1995). For example, does the individual have emotional, financial, or physical support (e.g., childcare, transportation) from family, friends, partners, employers, the legal system, the medical community, religious, or other local organizations to promote and encourage the treatment and recovery process? Often, the barriers that exist before treatment are the same obstacles that interfere with successful recovery.

Upon intake, determining the "stage of change" can assist the treatment provider in developing a comprehensive treatment plan. A stage of change assessment should be conducted for *each* abused substance and *each* additional comorbid condition, as desire and commitment to modify attitudes and behaviors may vary depending on the issue. For example, the patient may be willing to take medication

to manage their bipolar disorder or stop using alcohol and cocaine, but unwilling to address weight gain which would be necessary for recovery from their ED.

Finally, understanding what previous treatments and/or pharmacological interventions worked or did not work in the past can inform and guide the current treatment team. It is important to create a treatment plan and utilize interventions based on extending or supporting what has worked well in previous interventions.

12.13 Psychological Treatments

In this section, we will briefly review the 12-step approach to the treatment of SUD and several evidence-based (EBT) psychological treatments including motivational interventions (MI), contingency management (CM), and cognitive behavioral therapy (CBT). For a more comprehensive understanding of these and other approaches, please refer to individual chapters in the treatment section of this volume, the American Psychiatric Association (2006), and Connery and Kleber (2007).

12.13.1 12-Step Approach to the Treatment

Historically, the foundation for SUD treatment has been a 12-step, psychoeducational, abstinence model approach, delivered in a group format in intensive outpatient and residential settings. This model views AUD/SUD as a physical, emotional, and spiritual disease that can be arrested, but not cured. Recovery is viewed as a lifelong process that involves working the 12-steps of Alcoholics Anonymous (AA), Narcotics Anonymous (NA), or Cocaine Anonymous (CA) and abstaining from the use of psychoactive substances. In the USA, this is the most common intervention for SUD/AUD and utilized by most all accredited residential and intensive treatment programs (Ries, Galanter, Tonigan, & Ziegler, 2011). Additionally, as of 2013, AA estimates there are over 115,000 self-help groups and 2 million members worldwide (http://www.aa.org, 2013).

Briefly, the 12-Step approach utilizes a set of guiding principles that outline a course of action for recovery from SUD/AUD and other behavioral addictions. According to this approach, the first step toward recovery is acceptance and surrender. The individual must accept that they suffer from a chronic, relapsing, progressive illness (alcoholism/drug addiction) that has made their life unmanageable. Since there is no effective cure for alcoholism or drug addiction, the only viable solution is complete abstinence. In other words, the first step requires the individual to move away from denial and recognize that their prior efforts to reduce or eliminate the negative consequences of their substance use have not been successful.

Steps 2, 3, 6, 7, and 11 focus on developing hope, faith, trust, and a relationship with a power greater than themselves. Although 12-Step programs are based on spiritual principles and utilize the concept of a "higher power," they distinguish

themselves from formal religious practices. Spirituality is defined as "that which gives people meaning and purpose in life" (Puchalski, Dorff, & Hendi, 2004, p. 689), and a "higher power" can be defined as anything that is "greater" than the individual (e.g., God, nature, science, consciousness, existential freedom, or even the *collective* wisdom and experience of an AA group).

Steps 4, 5, 8, 9 and 10 require the individual to engage in a fact-finding and fact-facing process known as a "moral inventory." This process requires the individual to take a fearless journey into their past and compile a list of traits, dysfunctional behaviors, and self-defeating patterns that have compromised personal integrity and important interpersonal relationships. The inventory should also be counterbalanced by the individual's strengths and competencies. Once the inventory is complete, the individual is asked to share their "personal story" with others which promotes humility, fearlessness, and honesty. In Steps 8 and 9, participants are asked to make a list of people they have harmed and make amends where possible. These steps provide the individual with an opportunity to apologize for past harm; remove guilt, shame, and remorse; and promote positive future relationships (Nelson, 1990).

Step 12 asks participants to incorporate these guiding principles into their daily lives and help others who are suffering from similar addictions. The rationale is that helping and supporting others deepens the individual's commitment to the program and lifelong recovery.

12.13.1.1 Twelve-Step Facilitation

Twelve-step facilitation (TSF) is a brief (12–15 sessions), structured, evidence-based (Moos & Timko, 2008), manual-driven approach to facilitating early recovery from AUD and SUD (Nowinski, Baker, & Carroll, 1995). This approach rests on the assumption that peer support is an essential component of the recovery process. The primary goals of this approach are (1) to promote abstinence from substance use and (2) facilitate active participation in a 12-step program (Ries et al., 2011).

TSF can be utilized in both inpatient and outpatient settings and has been successfully used with patients that have other comorbid psychiatric disorders (Ries et al., 2011). Although this approach was originally designed to be applied in individual treatment, several studies have explored its use in a group format. A recently completed large multisite study compared a TSF individual plus group model (i.e., Stimulant Abuser Groups to Engage in 12-Step [STAGE-12]) to treatment as usual (TAU) in intensive outpatient treatment for stimulant abusers. Compared to the TAU group, individuals in STAGE-12 did have higher rates of attendance at 12-step meetings during active TSF treatment and at 6-month follow-up (Donovan et al., 2013).

12.13.2 Motivational Interventions

Alcoholism and drug addiction are chronic, relapsing illnesses. The reinforcing properties of these substances and the psychological and physiological dependence they engender often prevent individuals from acknowledging the frequency and intensity of their substance use problems, which can result in avoidance of treatment. Unfortunately, even today, families as well as many health and mental health professionals view this avoidance as "denial" and believe that individuals must "hit bottom" or experience significant negative consequences from their substance abuse before they will self-refer for treatment. A widely supported belief in the mental health field is that unmotivated patients fair poorly in treatment. As a result, in the past, individuals with low motivation were often turned away from formal treatment programs and referred to self-help programs (e.g., AA) in hopes that peer support and testimony would increase their desire to change (DiClemente, Kofeldt, & Gemmell 2011). However, there have been substantial changes in the field of SUD treatment in the past few decades, and more frequently, treatment professionals are focusing on utilizing evidence-based strategies to *motivate* patients not just educate or medicate.

Motivational interviewing (MI) is an individual, client-centered, humanistic approach infused with change focused strategies borrowed from traditional behavior therapy. MI focuses on helping people explore and resolve their ambivalence about behavioral change. Clinicians are encouraged to avoid giving advice or guiding patients toward specific solutions. Rather, the therapist's role is to be nonjudgmental and empathetic, actively listen, clarify the patient's thoughts and experiences, roll with resistance, elicit change talk, and collaboratively help patients explore the implications of behavioral change (DiClemente, Van Orden, & Wright 2011). MI has demonstrated efficacy as brief intervention and an adjunct to other interventions in the treatment of SUD (Miller & Rose, 2009).

Originally developed by William Miller (1983) as an intervention for problem drinking, MI has evolved into a manual-guided, brief treatment known as motivational enhancement therapy (MET) and has been added to other active individual and group treatments for an array of target problems including cardiovascular rehabilitation, diabetes management, dietary change, hypertension, illicit drug use, infection risk reduction, management of chronic mental disorders, gambling, smoking, and ED (Miller & Rose, 2009) (See Chap. 22).

12.13.3 Contingency Management

Contingency management (CM) is an intervention that involves the systematic application of the operant conditioning principles of reinforcement and punishment. CM was initially implemented in the substance abuse field in the 1960s in methadone clinics to increase counseling attendance and abstinence from opiates. However, over the past two decades, there has been a significant increase in the number of published studies using CM for the treatment of AUD, cocaine, cannabis,

stimulants, polysubstance use disorders, and smoking cessation (Higgins, Sigmon, & Heil, 2011).

CM interventions are carefully designed and implemented to retain patients in treatment, increase therapy attendance, reduce/eliminate drug use, and promote adherence to medications and other therapeutic regimens. The most thoroughly researched form of CM for SUD is voucher-based CM (Higgins, Silverman, & Heil, 2008). In this approach, patients earn vouchers that can be exchanged for retail items, prizes, cash, or privileges. One fundamental principle underlying CM is that behavior that is followed by reinforcing consequences will increase the likelihood of that behavior in the future (i.e., operant conditioning). Providing substitute reinforcement for treatment compliance can help bridge the gap between the elimination of substance abuse and the delayed naturalistic rewards of abstinence which include improved health, interpersonal relationships, and increased productivity. A recent meta-analysis of voucher-based CM found that out of 72 controlled studies published between 2005 and 2009, 88 % reported statistically significant treatment effects (Higgins et al., 2011).

12.13.4 Cognitive Behavioral Therapy

Cognitive behavioral therapy (CBT) refers to a group of therapeutic strategies based on both classical and operant conditioning principles and the work of Beck (1963) and Ellis (1962). The basic model posits that mental disorders and psychological distress are maintained by maladaptive cognitions. The core premise of this approach is that an individual's conscious thoughts (cognitions) are based on beliefs and assumptions (schema) derived from previous life experiences and that these thoughts determine how the person structures and reacts to their environment. The therapist and patient work collaboratively to identify the faulty or maladaptive cognitions that maintain their symptoms and self-defeating behaviors. Through Socratic questioning, hypothesis testing, problem solving, and homework, the patient challenges the validity of their faulty assumptions (i.e., cognitive restructuring) and modifies maladaptive behavioral patterns.

CBT is an active, structured, symptom focused, goal-directed approach that has been found effective in the treatment of mood disorders, anxiety disorders, PTSD, eating disorders, sleep disorders, personality disorders, psychotic disorders, and SUD (Hofmann et al., 2012). Because a significant majority of SUD patients have co-occurring psychiatric disorders, CBT can address multiple disorders in a comprehensive and integrated manner. The skills learned in CBT are designed to be generalizable and will benefit the individual long after they leave treatment (Carroll, 2011).

Over the past three decades, there have been numerous RCT of CBT for a wide range of SUD. CBT has been found effective in the treatment of AUD (Miller & Wilbourne, 2002; Morgenstern & Longabaugh, 2000), cocaine use disorders (Carroll, Rounsaville, Nich, & Gordon, 1994; Monti, Rohsenow, Michalec, Martin, & Abrams, 1997), cannabis use disorder (Copeland, Swift, Roffman, & Stephans, 2001;

MTP Research Group, 2004), opiate use disorder (Linehan et al., 2002), and polysubstance abuse (Linehan et al., 1999; Pollack et al., 2002; Schmitz, Averill, Sayre, McCleary, & Swann, 2002). CBT has also been found effective when combined with CM in the treatment of cocaine use disorders (Higgins et al., 1992) and opiate use disorders (Bickel, Amass, Higgins, Badger, & Esch, 1997) (See Chap. 25).

12.14 Pharmacological Treatments

Pharmacological interventions are commonly used in the withdrawal phase of treatment for AUD and opioid use disorders and post-withdrawal to manage craving and to prevent relapse. A comprehensive discussion of medications used for substance withdrawal is beyond the scope of this chapter; however, it can be found in Chap. 15. In this section, we will briefly review the pharmacological treatments that have an FDA approval for the treatment of SUD.

12.14.1 Alcohol Use Disorders

There are currently four FDA-approved, clinically useful medications for the treatment of AUD. Disulfiram (Antabuse) is an oral medication designed to reduce the likelihood of alcohol consumption due to its adverse effects. This medication produces highly unpleasant symptoms when combined with alcohol, including nausea, vomiting, headache, flushing, hypotension, vertigo, tachycardia, and dysphoria. In several RCT, disulfiram was found to reduce drinking days; however, there is no evidence that it improved relapse rates compared with placebo (Garbutt, West, Carey, Lohr, & Crews, 1999). Naltrexone (Revia [orally administered] and Vivitrol [injectable]) is an opioid receptor antagonist that decreases the reinforcing effects of alcohol ingestion, including reduced feelings of intoxication and fewer cravings. Naltrexone has been found to have short-term benefits in reducing relapse to heavy drinking; however, the evidence for longer-term use is less compelling (Williams, 2005). Acamprosate (Campral) is the most recently FDA-approved medication for AUD. This medication has been found effective in reducing relapse rates in alcohol-dependent individuals when combined with psychosocial treatments (Rosenthal, 2011).

Several other psychopharmacological agents have been studied (off-label investigations) in the treatment of AUD including anticonvulsants (topiramate [Topamax]), antispasmodics (baclofen [Lioresal and Kemstro]), serotonin reuptake inhibitors (fluoxetine [Prozac] and sertraline [Zoloft and Lustral]), and antiemetics (ondansetron [Zofran]). Each of these medications has demonstrated some efficacy for particular AUD subgroups (see Rosenthal, 2011 for a complete review of these agents).

12.14.2 Opioid Use Disorders

The FDA has approved five medications for the treatment of opioid use disorders. Methadone was first introduced in the early 1970s as a treatment for opiate dependence. It is a synthetic opioid and agonist (mimics the action of an opiate) that mitigates withdrawal symptoms, blocks the euphoric and sedating effects of opiates, and relieves craving. Methadone can only be dispensed at outpatient opioid treatment programs (OTP) that are certified by the Substance Abuse and Mental Health Services Administration (SAMHSA) and registered with the Drug Enforcement Administration (DEA). Daily oral administration of methadone, psychosocial interventions, and HIV education are all important components of the comprehensive treatment provided in these programs. Studies suggest that there is strong evidence that methadone maintenance keeps opiate abusers in treatment and reduces opioid use better than treatments without medication (Saxon & Miotto, 2011).

Buprenorphine (Subutex) and buprenorphine/naloxone (Suboxone) were approved by the FDA in 2002, and a new combination of buprenorphine and naloxone (Zubsolv) was approved in 2013. Buprenorphine is an opioid partial agonist, and naloxone is an opioid antagonist used to counter the effects of opiate overdose. Taken sublingually (placed under the tongue and allowed to dissolve), at low doses, buprenorphine produces sufficient agonist effects to prevent withdrawal symptoms and at moderate doses, has a "ceiling" for euphoric effects. Buprenorphine can only be prescribed and dispensed by certified OTP or through an approved program for physicians and pharmacists. Studies have shown that buprenorphine is more effective than placebo and equally as effective as moderate doses of methadone in opioid maintenance therapy. Also, it is known to cause a milder withdrawal syndrome compared to methadone (Stotts, Dodrill, & Kosten, 2009).

Naltrexone (Vivitrol) is a nonaddictive, nonnarcotic opioid antagonist. This medication does not mimic the effects of opioids (like methadone and buprenorphine), rather it blocks the receptor cites in the brain, thus preventing the euphoric effects of the drug. Naltrexone is administered in an injectable, long-acting form once per month to prevent relapse. Although some reviews suggest that retention and relapse rates for opioid use disorders are low, other studies suggest that naltrexone may be the treatment of choice for highly motivated patients (e.g., health-care professionals) that need to remain opiate-free (Veilleux, Colvin, Anderson, York, & Heinz, 2010).

A new form of buprenorphine (Probuphine) for the treatment of opiate addiction is currently under review by the FDA. This long-acting version is implanted under the skin in the upper arm and remains in place for 6 months. Approval was denied in April 2013 pending further information on the effects of higher doses and the development of a comprehensive plan for training physicians that would be inserting and removing the implant (Titan, 2013).

12.15 Risk Factors and Comorbidity in Substance Use Disorders

SUD are the result of the interplay between genes, environment, and developmental risk factors that influence one's susceptibility to psychoactive substance abuse. Although there is robust evidence indicating that genes play a significant role in the development of SUD, important psychological and social factors need to be considered in both the development and maintenance of these disorders.

Results from the 2011 national survey on drug usage found that 20.6 million individuals (8 % of the general population) are abusing or dependent on drugs and/or alcohol, with 14 million alcohol dependent, 4 million abusing or dependent on illicit drugs, and almost 3 million abusing or dependent on both alcohol and other drugs (SAMHSA, 2012) (see Table 12.1).

Gender, age, marital status, ethnicity, and employment status all appear to be risk factors associated with the development of SUD. Sociodemographic data suggest that males initiate substance use earlier than females, and males are more likely than females to abuse psychoactive drugs (approximately 10 vs. 6 %) or be heavy alcohol drinkers (6 vs. 3 %) (SAMHSA, 2012). Among individuals aged 18 or older, age of first use of cannabis (14 or younger) was associated with higher rates of SUD than adults who first used cannabis after age 18. Similarly, individuals that used alcohol at age 14 or younger were 7 times more likely to develop AUD than adults who consumed their first drink at age 21 or older (SAMHSA, 2012). Unmarried individuals have higher rates of substance *use* and approximately 11 % of divorced or separated women, and 16 % of women who have never married have a diagnosable SUD, as compared to only 4 % of married women (SAMHSA, 2004). In 2011, rates of SUD were lowest among Asians (3 %) and blacks (7 %). The highest rates of SUD were found in American Indians and Alaska Natives (17 %), followed by Native Hawaiians or other Pacific Islanders (11 %), Hispanics (9 %), and whites (8 %). Unemployed adults are more likely than employed adults to have SUD (15 vs. 8 %) (SAMHSA, 2012).

There have been several compelling family, twin, and adoption studies that suggest SUD is genetically influenced and that heritability factors place certain individuals at risk for the development of these disorders. Alcohol use disorders (AUD) run in families. McGue (1999) reported that 50–60 % of the phenotypic variance found in alcohol dependence in both men and women is due to genetic factors. In other words, children of alcoholics are five to six times more likely than the general population to develop AUD. Similarly, researchers have found that adolescents of parents that abuse illicit drugs are 45–79 % more likely than the general population to abuse drugs (Agrawal & Lynskey, 2006; Tsuang, Bar, Harley, & Lyon, 2001). In a large twin study done by Kendler, Jacobson, Prescott, and Neale (2003), genetic factors accounted for 73 % of the variance in cannabis abuse, 63 % in cocaine abuse, 63 % in hallucinogens, 51 % in sedative abuse, and 57 % in stimulant abuse. The only psychoactive substance in this study that showed a different phenotypic pattern was opiate abuse, where only 23 % of the variance could be attributed to genetic factors and 77 % to environmental factors. These researchers also found that an increased risk for the development of SUD was

nonspecific (i.e., genetic liability put one at risk to abuse *any* psychoactive drug) (Kendler et al., 2003). Additionally, genes that influence the development of SUD are also relevant to the development of other externalizing psychopathologies including conduct disorders, antisocial personality disorder, and ADHD (Kendler, Prescott, Myers, & Neale, 2003).

One of the strongest predictors of current substance abuse is past substance abuse. Previous epidemiological research on the "gateway pattern" found a predictable sequence of drug use initiation that begins with alcohol and nicotine use followed by cannabis then other illicit drugs (Kandel, Yamaguchi, & Chen, 1992). However, a recent epidemiological survey was conducted in 17 countries, and the results suggest that the pattern found in the USA is not consistently found in other countries. Other factors influence the ordering and progression of drug use around the world including availability of particular substances and cultural attitudes (Degenhardt et al., 2010).

Several environmental factors have been suggested as important in the initiation and maintenance of SUD. First, SUD tend to aggregate in families. In part, this is due to genetic influences, but studies have suggested that parental substance abuse can influence the development of SUD in children (Jennison & Johnson, 2001). In a large national study, looking at the relationship between childhood adversities and adult psychiatric disorders, researchers found that the maladaptive family functioning cluster (i.e., parental mental illness, parental SUD, criminality, family violence, physical abuse, sexual abuse, and neglect) predicted the development of and persistence of SUD (Green et al., 2010; McLaughlin et al., 2010).

There are also strong associations between mental disorders and SUD. Several longitudinal and cross-sectional surveys have confirmed that mental disorders can be conceptualized as risk factors for SUD (Compton, Conway, Stinson, Colliver, & Grant, 2005) because they precede the onset of SUD and can divide the population into high- and low-risk groups (Kraemer et al., 1997). The presence of premorbid mood disorders (particularly bipolar disorder), anxiety disorders (including PTSD), and externalizing disorders (i.e., ADHD, oppositional defiant disorder, conduct disorder, intermittent explosive disorder) predicted the likelihood of future SUD (Glantz et al., 2009). Similarly, Compton, Thomas, Stinson, and Grant (2007) found that individuals who met the criteria for *any* personality disorder were 1.8 times more likely to be substance abusers and 3.3 times more likely to be drug dependent than those who did not have a personality disorder. Finally, in a large national study of DSM-IV comorbidity in borderline personality disorder (BPD), researchers found that half of the respondents (51 %) had a SUD, 51 % had a mood disorder, and 60 % had a comorbid anxiety disorder (Grant et al., 2008).

Conclusions

This chapter was compiled for the ED treatment provider that is not well acquainted with the diagnosis, assessment, and treatment of patients with SUD. In the companion chapter, entitled *Introduction to Eating Disorders for the Substance Abuse Specialist* (See Chap. 11), we provided a basic overview of

diagnosis, clinical characteristics, medical complications, assessment, and evidence-based approaches for individuals with anorexia nervosa, bulimia nervosa, and binge eating disorder. Together, these chapters are designed to begin the cross-training process and prepare the reader to more fully assimilate and apply the content of the remaining chapters in this textbook. See Chap. 21 for a discussion of integrated treatment for patients with both ED and SUD.

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