

CHAPTER 12

ADDICTION AND SUBSTANCE ABUSE

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Abstract

E Addiction and substance abuse represent significant public health concerns with complex biological, psychological, and social dimensions requiring comprehensive understanding and management. Common drugs of abuse span multiple categories with distinct characteristics: stimulants like cocaine and amphetamines produce euphoria, increased energy, and sympathomimetic effects; opioids generate analgesia and euphoria while risking respiratory depression; sedative-hypnotics including benzodiazepines and alcohol cause anxiolysis and sedation; hallucinogens alter perception and cognition; and novel psychoactive substances continuously emerge to evade legal restrictions. Clinical manifestations of substance use disorders follow recognized patterns including compulsive use despite consequences, tolerance requiring escalating doses, withdrawal symptoms upon cessation, and neurobiological adaptations reflecting altered reward circuitry functioning. Acute intoxication and overdose present with substance-specific syndromes requiring targeted interventions, while chronic use leads to characteristic medical, psychological, and social complications. Treatment approaches incorporate pharmacological interventions including medication-assisted treatment for opioid and alcohol use disorders, psychosocial therapies spanning cognitive-behavioral approaches and motivational enhancement, and recovery support services delivered across a continuum from outpatient to residential settings. Prevention programs target different population levels, with universal strategies addressing entire communities, selective approaches focusing on high-risk groups, and indicated interventions targeting individuals showing early substance use patterns, all within a framework that addresses risk factors while enhancing protective factors.

Keywords: *Substance Use Disorder, Addiction, Medication-Assisted Treatment, Withdrawal, Intoxication, Harm Reduction*

Learning Objectives

After completion of the chapter, the learners should be able to:

- Differentiate between major categories of commonly abused substances based on their pharmacological properties, clinical effects, and patterns of use.
- Recognize the clinical manifestations of substance intoxication, withdrawal, and use disorders across different drug classes.
- Implement appropriate acute interventions for substance overdose including naloxone for opioids, benzodiazepines for stimulant toxicity, and supportive care tailored to specific substances.
- Develop comprehensive treatment plans incorporating pharmacological approaches, psychosocial interventions, and recovery support services appropriate to patient needs and substance use patterns.
- Compare the efficacy, indications, and limitations of medication-assisted treatments including methadone, buprenorphine, naltrexone, acamprosate, and disulfiram.
- Design evidence-based prevention strategies addressing substance use across the spectrum from primary prevention to harm reduction, with attention to developmental stages and population needs.

COMMON DRUGS OF ABUSE

Substance use disorders represent complex biopsychosocial conditions with diverse clinical presentations, neurobiology, and treatment implications across different substance categories.

Opioids

Opioids encompass both naturally occurring compounds derived from the opium poppy (*Papaver somniferum*), including morphine and codeine; semi-synthetic derivatives such as heroin, oxycodone, and hydrocodone; and fully synthetic agents including fentanyl, methadone, and tramadol. These substances produce their effects primarily through interaction with the μ -opioid receptor, with secondary activity at δ and κ receptors contributing to their complex pharmacological profile. Activation of these G-protein coupled receptors inhibits adenylyl cyclase, decreases neuronal excitability through potassium channel modulation, and reduces neurotransmitter release, collectively producing analgesia, euphoria, respiratory depression, and other

characteristic effects. Acute intoxication typically manifests with pupillary constriction (miosis), reduced consciousness ranging from sedation to coma, respiratory depression, and constipation, though tolerance to many effects develops rapidly with repeated exposure. Individual substances demonstrate significant pharmacokinetic variation, with heroin (diacetylmorphine) showing rapid brain penetration through enhanced lipophilicity before metabolic conversion to active morphine; fentanyl and analogs demonstrating extreme potency (50-10,000 times morphine) through efficient receptor binding; and methadone exhibiting prolonged half-life (24-36 hours) creating both extended therapeutic duration and withdrawal challenges.



Remember

Substance use disorders involve neurobiological changes in reward circuitry, executive function, and stress response systems, making addiction a chronic brain disorder requiring comprehensive medical and psychosocial treatment rather than simply a behavioral problem

The evolving opioid epidemic demonstrates distinct phases reflecting changing availability and use patterns. Initial prescription opioid misuse expanded substantially in the late 1990s and early 2000s, driven by increased pharmaceutical marketing, shifting pain treatment paradigms

emphasizing opioid therapy, and prescription practices often exceeding appropriate indications or durations. As regulatory restrictions and prescribing practice changes reduced prescription opioid availability, many individuals transitioned to heroin, which offered greater accessibility and lower cost despite increased use risks including uncertain purity, adulterant exposure, and injection-related complications. The current phase involves synthetic opioids, particularly illicitly manufactured fentanyl and its analogs, which have infiltrated both the heroin supply and counterfeit prescription tablet market. These highly potent compounds create extreme overdose risk due to their narrow therapeutic window, variability in illicit products, and rapid onset potentially outpacing naloxone rescue capability. This dynamic landscape necessitates evolving prevention, harm reduction, and treatment approaches addressing these shifting patterns while recognizing the persistent risk for cross-substance transition when availability changes occur.

Opioid tolerance, dependence, and addiction represent distinct but interrelated phenomena with important clinical implications. Tolerance develops through multiple mechanisms including receptor desensitization, internalization, and various intracellular adaptations, manifesting as diminishing effect with repeated dosing and requiring increased amounts to achieve comparable outcomes. Physical

dependence represents neuroadaptation to continuous opioid presence, with homeostatic adjustments creating a new equilibrium state requiring continued substance exposure for normal functioning. Withdrawal emerges when this dependence faces abrupt discontinuation, producing a syndrome typically including autonomic hyperactivity (diaphoresis, piloerection, mydriasis), gastrointestinal distress (nausea, vomiting, diarrhea), generalized pain, and intense craving. While both tolerance and dependence represent expected physiological adaptations to repeated opioid exposure, addiction involves additional behavioral, cognitive, and psychological dimensions including compulsive use despite harmful consequences, impaired control over consumption, and preoccupation with obtaining and using the substance. This distinction explains why patients receiving appropriate long-term opioid therapy may develop tolerance and dependence without necessarily demonstrating addiction, though these phenomena certainly increase vulnerability when additional genetic, psychological, and environmental risk factors exist.

Overdose represents the most immediate life-threatening consequence of opioid misuse, with fatality primarily resulting from respiratory depression through μ -receptor mediated effects on brainstem respiratory centers. Risk factors include high-potency formulations, particularly illicitly manufactured fentanyl analogs; combined depressant use, especially with benzodiazepines or alcohol creating synergistic respiratory effects; decreased tolerance following periods of abstinence such as incarceration or treatment; solitary use without others present to intervene; and comorbid conditions affecting respiratory function including chronic obstructive pulmonary disease or sleep apnea. Clinical presentation typically involves the classic triad of respiratory depression, central nervous system depression, and miosis, though mixed presentations may occur with co-ingestants or in cases of severe hypoxia where pupillary response becomes less reliable. Management centers on respiratory support and opioid antagonism through naloxone, which competitively displaces opioids from receptors restoring respiratory drive. While historically administered primarily by medical personnel, expanding community-based naloxone distribution has demonstrated effectiveness in reducing overdose fatalities through enabling bystander response before professional assistance arrives. Implementation considerations include appropriate dosing for high-potency synthetic opioids potentially requiring multiple administrations; recognition of naloxone's shorter duration compared to many opioids necessitating extended monitoring; and withdrawal precipitation potentially causing distress and rarely, acute pulmonary edema or catecholamine surge with associated cardiovascular complications.

Table 12.1: Commonly Abused Substances and Their Effects

Substance Class	Examples	Acute Effects	Chronic Effects
Opioids	Heroin, fentanyl, oxycodone	Euphoria, sedation, respiratory depression, miosis	Physical dependence, constipation, immunosuppression
Stimulants	Cocaine, methamphetamine, MDMA	Euphoria, hyperalertness, tachycardia, hypertension, hyperthermia	Psychosis, cardiovascular disease, weight loss, dental problems
Sedative-hypnotics	Benzodiazepines, barbiturates, alcohol	Anxiolysis, sedation, disinhibition, respiratory depression	Cognitive impairment, physical dependence, tolerance
Cannabis	Marijuana, hashish, concentrates	Euphoria, relaxation, altered perception, increased appetite	Amotivational syndrome, potential cognitive effects, cannabinoid hyperemesis
Hallucinogens	LSD, psilocybin, DMT	Perceptual alterations, synesthesia, emotional lability	Hallucinogen persisting perception disorder (rare)

Stimulants

Cocaine, derived from *Erythroxylum coca* leaves, produces stimulant effects primarily through blocking reuptake of monoamine neurotransmitters (dopamine, norepinephrine, and serotonin) at synaptic sites, resulting in increased neurotransmitter availability and prolonged receptor activation. This mechanism differs from amphetamine-type stimulants, which both block reuptake and actively promote neurotransmitter release from presynaptic terminals, creating somewhat different clinical profiles though with substantial overlap. Available forms include cocaine hydrochloride powder typically used

intranasally or dissolved for intravenous administration, and alkaloidal "crack" cocaine created through basic extraction enabling vaporization and inhalation. These different preparations create distinct pharmacokinetic profiles, with crack cocaine demonstrating particularly rapid onset (seconds) and brief duration (5-10 minutes) contributing to its highly reinforcing nature and characteristic binge use patterns. Acute effects include euphoria, increased energy, alertness, intense pleasure, and psychomotor agitation alongside physiological manifestations of sympathetic nervous system activation including tachycardia, hypertension, hyperthermia, mydriasis, and diaphoresis. Cocaine-associated medical emergencies primarily involve cardiovascular complications including arrhythmias, myocardial infarction, and hemorrhagic stroke resulting from combined vasoconstrictive effects, platelet activation, enhanced clotting cascade, and direct myocardial toxicity. Additional concerns include seizures through lowered seizure threshold, hyperthermia with potential progression to rhabdomyolysis and renal failure, and psychiatric manifestations including stimulant-induced psychosis characterized by paranoia, agitation, and occasionally hallucinations. Chronic use creates risk for nasal septum perforation with intranasal administration, pulmonary damage with inhalational use, and endocarditis or bloodborne infections with injection. Treatment approaches emphasize supportive care during acute intoxication, with benzodiazepines addressing agitation, seizures, and autonomic hyperactivity, while long-term management remains predominantly psychosocial with no FDA-approved pharmacotherapy specifically for cocaine use disorder.

Amphetamine-type stimulants include both pharmaceutical preparations (dextroamphetamine, lisdexamfetamine, methamphetamine) primarily prescribed for attention-deficit/hyperactivity disorder and narcolepsy, and illicitly manufactured forms, particularly crystal methamphetamine. These compounds increase synaptic concentrations of monoamine neurotransmitters through multiple mechanisms: inhibiting reuptake through transporter proteins, displacing stored neurotransmitters from vesicles, and directly reversing transporter direction causing neurotransmitter efflux into synapses.

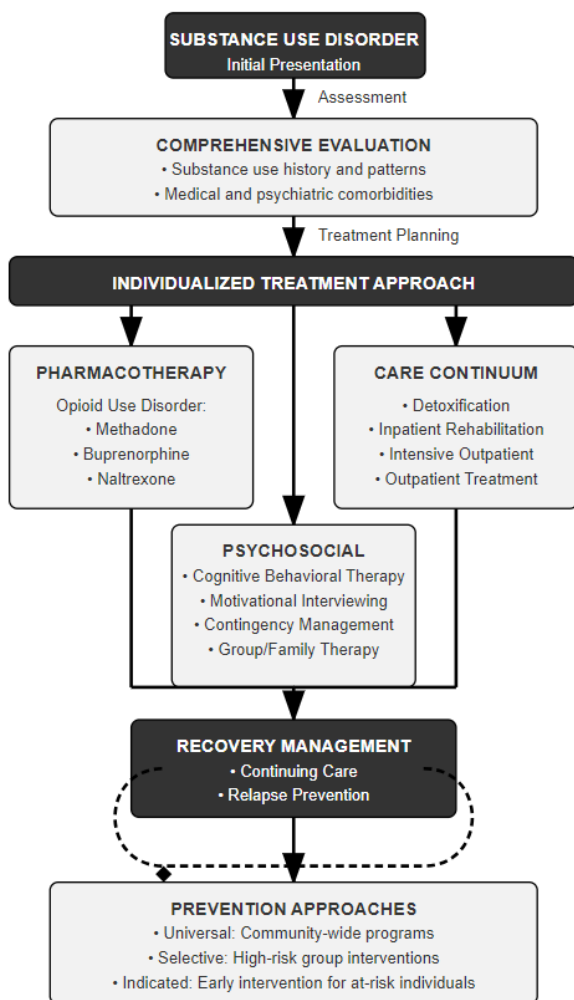


Figure 12.1: Substance Use Disorder Management

Methamphetamine demonstrates high lipophilicity creating rapid brain penetration and prolonged effects, with elimination half-life exceeding 12 hours compared to approximately 2 hours for dextroamphetamine. Route of administration significantly affects pharmacokinetics and associated risks, with oral consumption producing gradual onset over 30-60 minutes; intranasal use creating more rapid effects within 5-10

minutes; smoking enabling almost immediate central nervous system effects similar to intravenous administration while avoiding injection-related complications; and intravenous use creating most rapid and intense effects with associated infectious and vascular risks. Acute intoxication presents with sympathomimetic symptoms including tachycardia, hypertension, hyperthermia, mydriasis, and diaphoresis alongside behavioral manifestations ranging from increased energy, confidence, and sociability to agitation, paranoia, and occasionally frank psychosis with hallucinations and delusions. Medical complications mirror those of cocaine though with typically longer duration reflecting extended half-life, including cardiovascular emergencies, seizures, hyperthermia, and rhabdomyolysis. Distinctive to methamphetamine, chronic use frequently manifests with severe dental deterioration ("meth mouth") resulting from combined xerostomia, bruxism, poor oral hygiene, and sugar cravings; formication with resulting excoriation from sensation of insects crawling under the skin; and distinctive physical appearance changes through combined weight loss, skin picking, and premature aging. Treatment approaches primarily involve behavioral interventions, with limited evidence supporting certain pharmacotherapies including bupropion and naltrexone for reducing use and craving, though none have achieved FDA approval specifically for methamphetamine use disorder.

Novel psychoactive stimulants represent an evolving category including synthetic cathinones (colloquially termed "bath salts") and various phenethylamine derivatives designed to mimic traditional stimulant effects while temporarily circumventing drug control laws through structural modifications. Cathinone, the prototype compound naturally occurring in khat plant (*Catha edulis*) leaves, undergoes various synthetic alterations creating compounds including mephedrone, methylone, and 3,4-methylenedioxypyrovalerone (MDPV). These substances primarily increase synaptic monoamine concentrations through mechanisms similar to cocaine and amphetamines, though with varying selectivity for specific neurotransmitter systems creating diverse effect profiles. Typically marketed under misleading labels as "bath salts," "plant food," or "research chemicals" with deceptive "not for human consumption" disclaimers, these products gained popularity for their initial legal status, easy internet purchasing, and absence from standard drug screens. Clinical presentations generally resemble traditional stimulant intoxication but often with more pronounced psychiatric manifestations including severe agitation, paranoia, hallucinations, and occasionally violent behavior requiring physical restraint and chemical sedation. Medical complications mirror those of other stimulants but sometimes with greater severity, including hyperthermia, rhabdomyolysis, seizures, and cardiovascular

END OF PREVIEW

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