

SWK 5805: Theories and Biological Basis of  
Substance Misuse



# SWK 5805: Theories and Biological Basis of Substance Misuse

*DR. AUDREY BEGUN*



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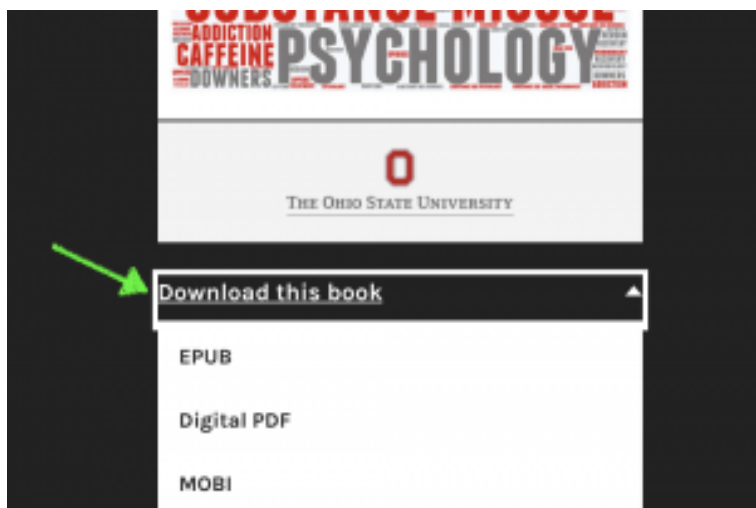
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# Introduction to the Coursebook

Welcome to the online interactive coursebook for our Theories and Biological Basis of Addiction course.

These materials are designed to be read either interactively online or after downloading to your computer (you can print them out in hard copy, too, if you prefer). You have the option of reading the materials interactively on multiple types of devices, including EPUB and MOBI (works best for small screens like phones). The downloads are available on the front page of the book. Click the link to “Download this book” and then select your preferred format.



To read the contents of a module, just click on the “Contents” field in the top-left corner of the web page to extend the accordion. Then click the “+” button to extend the menu and access the rest of the chapters in the module.

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Ch. 1: Genetic Influences

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The embedded interactive exercises require internet connectivity but each can be downloaded for offline work—you simply will not benefit from the immediate feedback the online interactive environment offers. These interactive exercises are presented to help you practice with what you are reading, to challenge yourself, prepare for quizzes, and have a little fun along the way.

Each Module contains a list of key words at the end explaining terms **highlighted in bold italics** throughout the text. If you click on one of these, it will take you to the Key Terms section where you can see a definition/description of the term. Then, you can use the back arrow to return to where you were reading.

Where there are additional outside readings assigned, the links are provided in your Carmen course "Introduction—Tasks" area with the full reference provided in the reference list at the end of each module.

To read the contents of a module, click on the Contents a dropdown menu where there is "+" sign for a list of the contents assigned. This should help you navigate chapters, too.







## PART I

# MODULE 2: KEY DEFINITIONS, DIAGNOSTIC CRITERIA, CLASSIFICATION OF SUBSTANCES, & TRENDING TOPICS

Module 2 readings introduce several key terms with their meaning and use defined. This initial section concludes with an explanation of the biopsychosocial perspective on substance misuse and how the various types of theories we are studying might fit together rather than “compete” with each other to help shape our understanding. This biopsychosocial perspective forms the structure for our course Part 1 analysis of theories. Chapter 2 of our Module 2 readings examines the major diagnostic systems used in the U.S. and internationally for diagnosing various types and severity of substance use disorder (SUD). In Chapter 3, we explore several trending topics in the substance use/misuse and SUD arena.

## Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Define key terms related to substance use, misuse, and use

disorders

- Describe the biopsychosocial perspective on substance misuse
- Describe the diagnostic criteria applied to alcohol and other substance use disorders
- Explain how two different systems apply to the classification of different types of substances
- Identify three key trending topics in the area of substance use/misuse and the different sides of the issues

# Ch. 1: Key Definitions & Diagnostic Criteria

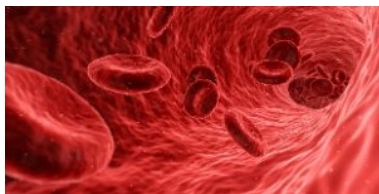
You may be familiar with terms like “alcoholism,” “drug addiction,” and “alcohol or other drug (AOD) dependence.” These terms all relate to the focus of this chapter which presents current thinking about these concepts and how terminology is used in practice and research. First, several key terms are defined and explained. Then, the diagnostic schemes currently in use are described.

In Module 1, you learned about how the concept of psychoactive substances is defined. Now let’s look into how related concepts are used.

## Key Definitions

In Module 1, you learned about how the concept of psychoactive substances is defined—substances (chemicals) “with the potential to cause health and social problems, including addiction” is one way to summarize this (McLellan, 2017, p. 113). Now let’s look into what the concepts of substance use, substance misuse, and substance use disorder actually mean. Because the criteria for substance use disorder include the terms “tolerance” and “withdrawal,” these two terms are also defined for you. Then we can delve into the meaning of “biopsychosocial” with regard to understanding substance misuse and substance use disorders, and the implications of adopting a biopsychosocial perspective in studying different theories.

**Substance Use.** The concept of [substance use](#) is fairly straight-forward: introducing a psychoactive substance into the body/circulating blood stream. As



you will learn in this course, there are many ways these different substances are used: drinking, eating, introducing through oral membranes, or otherwise ingesting; inhaling, “snorting,” or introducing through nasal membranes; smoking or otherwise inhaling through lungs; injecting; and, absorbing through skin are all common modes of introduction. The concept of substance use does not distinguish amounts or consequences of use.

**Substance Misuse.** [Substance misuse](#) implies that substance use occurs in high enough doses or in risky situations such that physical health, mental health, and/or social problems may result (McLellan, 2017). The dose need not be sufficient to cause overdose to be potentially problematic, and the problems may not appear immediately but accumulate over repeated misuse episodes. An example is the difference between using alcohol and binge drinking—the dose consumed during a single drinking episode matters and repeatedly engaging in binge drinking is more problematic than a single episode. In some scenarios, the actual dose consumed may not be as problematic as the situation when/where it is used. For example, using alcohol or cannabis at home might not be problematic but driving under the influence is. Or, a type of substance use might not be problematic for most individuals but is for a woman during pregnancy. And, substance misuse is not defined by the consequences actually experienced but by the potential consequences—many of which can be severe and irreversible, such as exposure to infectious disease, accidental injury (to self or others), legal difficulties/incarceration, and damage to physical or mental health (including, but not limited to overdose or substance use disorder).



## Substance Use

**Disorder (SUD).** In order for an individual to be diagnosed or classified as experiencing a [substance use disorder](#), certain specific criteria must be met. Historically, terms like “alcoholism” and “addiction” were applied, but these terms have been applied unsystematically and



inconsistently. Instead, a substance use disorder used to be called either substance abuse or substance dependence in the American Psychiatric Association’s DSM-IV (*Diagnostic and Statistical Manual of Mental Disorders, fourth edition*). Until recently, and for many years, these diagnostic criteria were applied across much of the U.S. mental and behavioral health system and reported in much of the research literature. At the international level, the World Health Organization’s ICD-10 (International Classification of Diseases and Related Health Problems, version 10) served a similar function.

**Tolerance.** Because of changes in the brain and body (more about this in Module 3), greater amounts of a substance might be needed if certain substances are repeatedly used over time. In other words, when [tolerance](#) to a substance (or type of substance) develops, a person may need to use increasingly higher doses of a drug, medication, alcohol, or other substance to achieve the same psychoactive effects previously experienced at lower doses. (Another way of increasing dose is to use the substance more often—you will see more about this when we look into pharmacokinetic principles). In the DSM-V, acquired tolerance is characterized by either:

- A need for markedly increased amounts of the substance to which tolerance has been developed in order to achieve intoxication or the desired effect; or,
- A markedly diminished effect with continued use of the same amount of the substance.

An example demonstrating where acquired tolerance is particularly problematic occurs with individuals who have developed tolerance, are unable to obtain the substance for a period of time, then resume use again at the same level previously used. For instance, someone may have been regularly using heroin at a certain high level prior to being incarcerated in jail, unable to access heroin during their period of incarceration, and resumes using at community reentry following release from incarceration. If the period of abstinence was long enough, the person's body may have re-adjusted to not having the substance on board, and their tolerance diminished. Resuming use at the previously tolerated level could lead to an overdose in their re-adjusted condition.

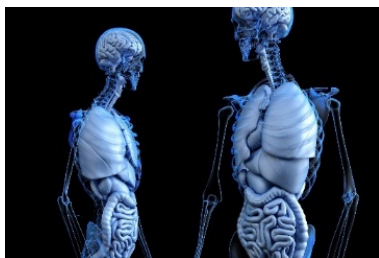
Another example where tolerance matters has to do with alcohol consumption. A person consuming enough alcohol to have a blood alcohol level (BAL)/blood alcohol concentration (BAC) of .20 for the first time likely would experience blackout. However, a person who routinely drinks to this BAL/BAC level might have developed



sufficient tolerance that, while their functioning is significantly impaired, the effects are more reflective of a lower BAL/BAC outcome for individuals who drink less and drink less often.

Base tolerance differs a bit from acquired tolerance in that it is a person's tolerance level for the substance prior to regular substance use. Base tolerance is influenced by a person's biological and genetic makeup and it can be tricky to recognize the implications of this source of individual difference in response to substance use/misuse. For example, individuals who believe "I can hold my liquor better than others" or "I can drink everyone else under the table" also may believe that this is protective from developing an alcohol use disorder or other health consequences related to binge or heavy drinking—believing they have immunity or are "tougher" than others. Unfortunately, this is untrue; in fact, someone who does not feel the effects of alcohol after only a couple of drinks is likely to continue drinking in higher quantities to achieve the desired effect. Meanwhile, the body and organ systems are awash in higher levels of alcohol (and its breakdown/metabolite substances—more about this in Module 3), regardless of the person's psychoactive experience. The brain, heart, liver, and other organ systems are affected by the higher concentrations circulating in the body. This increased concentration of alcohol is doing greater harm, regardless of how the person feels.

**Withdrawal.** With many (but not all) substances, a person's body adapts to the presence of the substances to such an extent that if that substance is no longer available, the person experiences a host of very difficult symptoms (more about this in Module 3). In the DSM-V language, substance [withdrawal](#) is evidenced as:



- the characteristic withdrawal syndrome for the particular

- substance involved, and
- the substance or closely related substances are taken to relieve or avoid withdrawal symptoms (e.g., benzodiazepine withdrawal might be reduced with alcohol).

Examples of alcohol withdrawal symptoms, for instance, include high levels of anxiety, sleep disorders, tremors, nausea/vomiting, sweating, racing heart, physical restlessness, and possible seizures. If a person experiences two or more of these symptoms as a direct result of stopping or reducing alcohol intake, and not as a function of some other condition or other substance the person may have used, the person may be diagnosed with alcohol withdrawal syndrome. Examples of symptoms associated with withdrawal from heavy, prolonged cannabis/marijuana use can include: irritability, anger, aggression, difficulty concentrating, nervousness, anxiety, sleep disturbances, vivid unpleasant dreams, decreased appetite/weight loss, restlessness, depression, shakiness, sweating, fever, chills, and headache. Symptoms associated with opioids, where use has been heavy for several weeks or more, can include: depression, nausea/vomiting, muscle pain, runny eyes, runny nose, sweating, diarrhea, fever, and insomnia. The experience of withdrawal from substances can be fraught with misery.

There exist both differences and similarities in withdrawal from different types of substances, not only in terms of symptoms but also in how soon symptoms might appear and how long they might last. Learning about different substances (as we will in Part 2



of the course) is important because withdrawal from some substances that is not medically managed can be fatal. Just quitting may not always be the safest choice—a person may need to be gradually weaned off certain substances to avoid dangerous

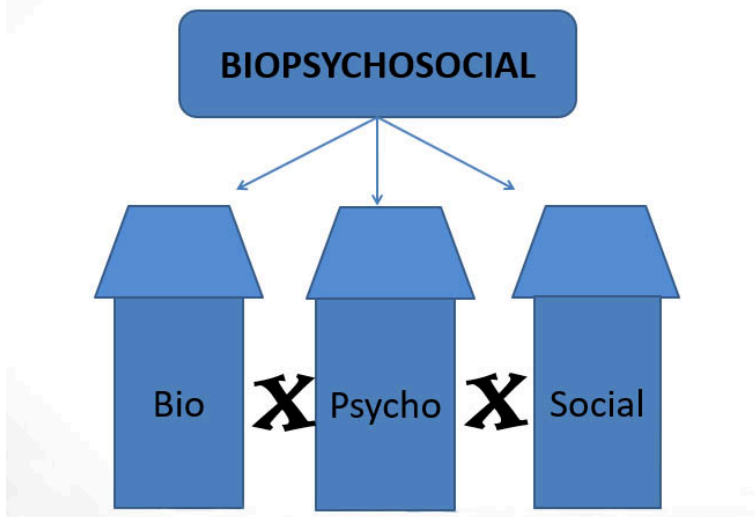
withdrawal effects on heart rate/rhythm, blood pressure, and severe seizures. Consider the public health implications of large community disasters like the combination of Hurricanes Katrina and Rita that made it impossible for some individuals to access alcohol, other substances, or even prescription medications on which their bodies had come to depend—their withdrawal could contribute to loss of life. This is true if a person's access is interrupted by the theft of the substances/prescription medications, inability to pay for medications or a pharmaceutical company's interruption of supply.

Withdrawal symptoms and the experience of withdrawal have profound implications for a person's recovery, especially in the early phase. Withdrawal symptoms may interfere with a person's ability to function as much as (or even more than) the substance misuse did. You will learn more about this in Module 4 when we look into learning principles and their role in addictive behavior.

**Biopsychosocial Perspective.** You may have heard the term “[biopsychosocial](#)” in reference to how we think about complex behavioral health issues and human development. In review (or if the concept is new to you), it means that in order to fully understand a phenomenon like substance misuse it is essential to understand the biological, psychological, and social factors involved in its development, maintenance, and resolution. Unfortunately, because of the different disciplines and professions involved, these three domains are often considered individually or distinctly from each other, rather than as an integrated whole—each domain is often considered as a silo, separate from the others (and not always equal to the others). In reality, the three domains interact in important, mutually influential ways. Whether or not someone engages in substance misuse is influenced by that person's biological makeup and processes, psychological makeup and experiences, and experiences/interactions with the social and physical environment. One thing that all of the research in substance misuse and substance use disorders taken together has taught us is: THERE IS NO ONE SINGLE CAUS, and there is not even any one single

domain involved. These are very complex phenomena with multiple interacting causes. It also explains why the experience can be so different among different individuals and why “one size fits all” treatment approaches do not fit all. In the next several modules of our course, we examine each of these domains separately, because within each there exists a great deal of complexity (Module 3=biological, Module 4=psychological, and Module 5=social/physical environmental). Then, in Module 6, we explore ways to bring these domains back together in a more integrated fashion.

## Major Classifications of Theories



### Current Diagnostic Criteria

In 2013, the American Psychiatric Association adopted a new diagnostic system, the [DSM-5](#) (APA, 2013), informed by decades of additional research into the epidemiology, etiology, and treatment of various psychiatric conditions. This is the main scheme for diagnosing substance use disorders currently used in the U.S.

clinically, and increasingly adopted in research. The ICD-10 is in the process of being replaced by the **ICD-11**. Fairly dramatic changes were seen in the criteria for the diagnosis of substance use disorders. The most dramatic was the change from two distinct categories, abuse, and dependence, to viewing these disorders on a continuum of severity. The list of 11 diagnostic criteria (see Table 1) reflect 4 categories of function:

- impaired control over use [items 1-4]
- social impairment/consequences [items 5-7]
- risky use of the substance(s) [items 8-9]
- pharmacological indicators/symptoms: tolerance, withdrawal [items 10-11]

Table 1. Eleven DSM-5 criteria for diagnosing substance use disorder (SUD)

1	Often taking alcohol or another substance in larger amounts or for a longer period than intended (e.g., planning to limit yourself to 2 beers but ending up drinking 6)
2	A persistent desire or unsuccessful efforts to cut down or control use of alcohol or another substance (e.g., believing your substance use is problematic and attempting to cut back on frequency or amount, but failing to do so).
3	Spending a great deal of time in activities necessary to obtain, use, or recover from the effects of alcohol or another substance (e.g., spending days planning a drinking event/party then drinking before, during, and after the event/party, and needing a day or two to recover from the drinking event/party; or, having to know/plan where you will acquire the substances when you go on vacation or to travel out-of-town)
4	Strong desire, craving, or urge to use alcohol or another substance (e.g., “needing” to smoke a cigarette when driving in the car, with certain friends, or at the end of a meal)
5	Failure to fulfill major role obligations at work, school, or home resulting from recurrent use of alcohol or another substance (e.g., continually “dropping the ball,” disappointing other people, failing to do what is expected of you at work, academically, at home, such as not feeding your children or pets or failing to provide them with adequate supervision because you are intoxicated, high, or recovering from substance use)
6	Continued use of alcohol or another substance despite persistent or recurring problems in social or interpersonal relationships that are caused or made worse by the effects of alcohol or another substance (e.g., continuing to “get high” despite knowing that it is causing relationship problems with your partner/spouse, parents, siblings, or children)
7	Giving up or reducing important social, occupational, or recreational activities because of alcohol or other substance use (e.g., no longer engaging in past hobbies/interests or work, family, fun activities in favor of using substances or recovering from use; replacing your “life” with substance use)
8	Recurrent use of alcohol or another substance in situations where it is physically dangerous to do so (e.g., operating a car/motorcycle/boat or other vehicle while under the influence, engaging in risky sexual practices while under the influence or to acquire substances, risking harm to self or others)

9	Continuing to use alcohol or another substance despite knowledge of having a persistent or recurring physical or psychological problem that could be caused or made worse by its use (e.g., continuing to drink despite its effects on diabetes, liver disease, sleep patterns, or depression)
10	Developing tolerance for alcohol or another substance (see definition of tolerance)
11	Experiencing withdrawal symptoms or taking alcohol or closely related substance in order to relieve or avoid withdrawal symptoms (see definition of withdrawal)

**Severity.** The DSM-5 diagnosis scheme includes the dimension of severity, based on the number of symptoms an individual is experiencing. Severity is determined as follows:

- mild SUD: 2 or 3 symptoms
- moderate SUD: 4 or 5 symptoms
- severe SUD: 6 or more symptoms

**Types of SUD.** While moving away from categorizing SUD in categorical terms (abuse/dependence) the DSM-5 (and the ICD-11) does distinguish between different types of substances involved. Nine types of substance use disorder are identified, each of which utilizes the 11 criteria and severity schedule above (see Table 2).

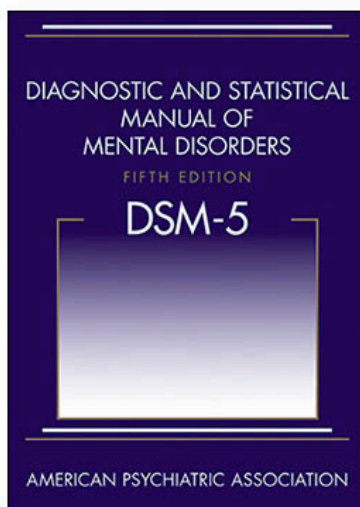
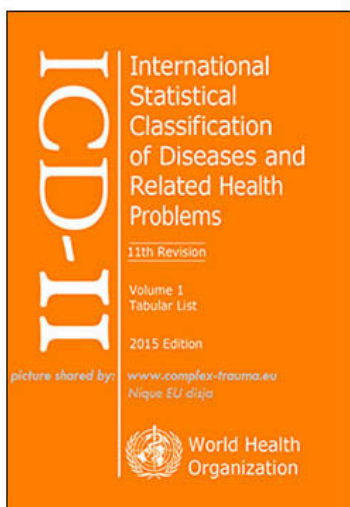
Table 2. Types of substance use disorder classified in DSM-5.

DSM-5 Code	Type of Substance
F10	alcohol
F11	opioid
F12	cannabis/marijuana
F13	sedatives, hypnotics, or anxiolytics
F14, F15	Stimulants (the 14 code is specific for cocaine, 15 for amphetamines)
F16	hallucinogens (other than cannabis)
F17	tobacco
F18	inhalants
F19	Other/unknown substance use disorder

Diagnostic systems make a distinction between a substance use disorder (like alcohol use disorder, AUD, or opioid use disorder, OUD) and a substance-induced disorder. Some of what we see in terms of problems related to the use of alcohol or other substances are caused or exacerbated (made worse) by substance use, but do not reflect a substance use disorder per se. For example, sleep disorders may be induced by substance misuse, or depression may result from use or stopping the use of certain substances. Even psychotic episodes might be induced by substance use despite there not being an underlying psychotic mental condition. Clinicians are quick to admit that it is sometimes very difficult to tell these apart and to make an accurate differential diagnosis. However, the distinctions are clinically important because the different processes need to be treated or managed in different ways.



Additionally, the DSM-5 recognizes that someone may use more than one type of substance, termed “polysubstance” use disorder. Caffeine is a special case in the DSM-5 where it is possible that a substance-related disorder exists, but there is not an actual substance use disorder code associated with caffeine. And, finally, it is important to know that the DSM-5 (and ICD-11) recognizes substance withdrawal as being distinct from a diagnosable SUD—the symptoms of substance withdrawal often warrant a separate diagnosis described in the DSM-5.





**Before you read on, take a moment to jot down your “best guess” answers to the following questions:**

The focus of our course is on substance misuse and substance use disorders. However, many practitioners and scholars argue that the principles apply to other types of behaviors, as well. For example, you may have heard discussions about what some call “process” or “behavioral” addictions:

- Gambling addiction
- Internet/gaming addiction
- Sex addiction
- Shopping addiction

Based on what you have learned so far about defining substance use disorders and addiction, consider the following 3 questions:

1. What do you think might be the similarities or differences between a person who experiences an alcohol use disorder and a person with a gambling disorder?
2. What about “disordered” internet gaming or other “dependence” on technology?

3. What do you think about people using the word “addiction” to describe how they feel about a favorite television show? What about advertisers describing games like Candy Crush as “addicting” to promote its popularity?

# Ch. 2: Classification Systems for Different Types of Substances

As you have learned about what psychoactive substances are, the next step is to consider how they might be classified—classification systems usually highlight similarities between items included within each group and differences between groups. Two major classification schemes have significance and relevance in the U.S. The first relates to the pharmacological and behavioral effects of different substances—this system informed the structure of the second half of our course. The second scheme relates to the legal status of different substances—the Drug Enforcement Agency (DEA) schedule of drugs.

## Classification by Effects

One way of organizing the very long list of psychoactive substances is in terms of their actions on the human body. It would be impossible to list them all because the list is constantly evolving: not only are new nicknames being invented all the time, new formulations (drugs) are being developed on a regular basis. In addition, some substances do not fit neatly into a single category. For example, it is difficult to know how to classify caffeinated energy drink plus alcohol beverages (e.g., Four Loko®, Joose®, Sparks+®, Jaegerbombs, or vodka with Red Bull®) since their components fall into two very different categories: stimulants (caffeine and some other ingredients) and central nervous system depressant (alcohol). (Note that premixed beverages of this type no

longer are sold in most of the United States and many countries but are still produced for consumption in other nations and mixed by individuals on their own.)

In many sources, there is a distinction made between legal or illegal “street” drugs. However, this distinction has two major flaws. First, we have seen a tremendous upsurge in the illegal use of legal substances in recent years—by now, you have heard about the problem of prescription drug abuse in the news. Second, laws can change, as we have witnessed recently with states legalizing various uses of cannabis (medical and recreational uses), and legal drinking age laws that have fluctuated in the United States between 21, 18, 19, and back to 21 just since the 1970s.

The way that clinicians and researchers categorize psychoactive substances is in terms of their effects on the human body or behavior. Tables 3-10 present you with just such a list. Considering some of the substances with which you or people you know may have experience, does it surprise you to see how they are classified? You may be surprised to see alcohol classified as a depressant, or caffeine and tobacco in the same (stimulant) category as cocaine. The different substances within each category may have differences from each other. However, it is important to recognize that they also have shared common features in terms of how they affect the mind, body, and behavior. We will look into each of these different types of substances in the second half of our course. For now, we are aiming for a general overview of the picture concerning “what’s what” in the array of psychoactive substances.

**Table 3. Stimulant Substances.**

Examples of Stimulants	Usual Administration Route & Common Effects
<p>amphetamines (dexadrine, bennies, black beauties, hearts, speed, uppers); attention deficit disorder and narcolepsy medications (e.g., Adderall, Concerta, Ritalin); “bath salts;” caffeine</p>	<p>Administration: Snorted, smoked, injected, swallowed; caffeine also chewed in gum, absorbed through skin in a patch.</p> <p>Effects: Increased heart rate and blood pressure, elevated body temperature, increased body metabolism, reduced appetite, increased energy, feelings of exhilaration and mental alertness, tremors, irritability, anxiety, panic, paranoia, violence and aggression, psychosis. Increased risk of insomnia, weight loss, cardiovascular complications, stroke, seizures, addiction, fatal overdose.</p>
<p>cocaine and “crack” cocaine (blow, C, candy, coke, flake, rock, snow, toot)</p>	<p>Administration: Snorted, smoked, injected.</p> <p>Effects: Nasal damage from snorting, exposure to infectious diseases from injection, poor pregnancy outcomes, and see amphetamines effects above.</p>
<p>methamphetamine (meth, ice, crank, crystal, fire, glass, speed)</p>	<p>Administration: Snorted, smoked, injected, swallowed.</p> <p>Effects: Severe dental problems, poor pregnancy outcomes, explosion/fire risks during production, chemical and environmental contamination from production activities, and see amphetamines effects above.</p>
<p>MDMA (Ecstasy, “club drug” combination of stimulants and hallucinogens of various types)</p>	<p>Administration: Swallowed.</p> <p>Effects: Feelings of euphoria, enhanced mental and emotional clarity, sensations of lightness and floating and other hallucinations, suppression of appetite, thirst, and need for sleep, anxiety, nausea, blurred vision, faintness, high blood pressure, tremors, seizures, elevated body temperature. Increased risk of exhaustion, severe dehydration, sleep disorders, cognitive impairment, confusion, depression, aggression, impulsive behavior, fatal overdose, possible addiction.</p>

tobacco products, nicotine (cigarettes, bidis, cigars, cigarillos, pipe tobacco, e-cigarettes, hookah tobacco, snuff, chew, nicotine patch or nicotine gum)	Administration: Smoked, snorted, chewed; absorbed through skin in a patch. Effects: increased blood pressure and heart rate. Increased risk of chronic lung disease, heart disease, stroke, cancers (mouth, throat, stomach, pancreas, cervix, kidney, bladder, acute myeloid leukemia), poor pregnancy outcomes, overdose (young children), addiction.
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**Table 4. Depressants and Dissociatives**

Examples of Depressant & Dissociative Drugs	Usual Administration Route & Common Effects
alcohol (ethanol, ethyl alcohol, etoh)	<p>Administration: swallowed; some are smoked, chewed, or injected</p> <p>Effects, low dose: euphoria, mild stimulation, relaxation, lowered inhibition;</p> <p>Effects, high dose: drowsiness, slurred speech, nausea, emotional volatility, poor coordination, impaired perception, impaired memory, sexual dysfunction, loss of consciousness, impaired breathing. Increased risk of injury, depression, neurologic and cognitive deficits, memory loss, high blood pressure, liver and heart disease, poor pregnancy outcomes, addiction, fatal overdose.</p>
anti-anxiety medications	
benzodiazepines	
dextromethorphan (DXM) in large amounts (some cough medicine formulations)	
pre-anesthesia medications (rohypnol)	
PCP (phencyclidine; angel dust)	
salvia	
sleep medications	
tranquilizers (“tranqs”)	

**Table 5. Cannabinoids**



Examples of Cannabinoids:	Usual Administration Route & Common Effects
cannabis; marijuana (blunt, dope, ganja, grass, herb, joint, bud, Mary Jane, pot, reefer, smoke, weed); hashish ("hash"); synthetic marijuana compounds	Administration: Smoked, swallowed. Effects: Euphoria, relaxation, slowed reactions, distorted sensory perception, impaired balance and coordination, increased heart rate, increased appetite, impaired learning and memory, anxiety, psychosis. Increased risk of respiratory effects and infections, declining mental health, addiction, unknown effect on pregnancy outcomes. Potential harm from additives.

**Table 6. Opiates, Opioids, & Other Pain Relievers (Analgesics)**

Examples of opiates, opioids, & other pain relievers	Usual Administration Route & Common Effects
heroin, morphine (and morphine derivatives), opium (laudanum, paregoric, gum, big O, block, black stuff), oxycodone, oxycontin, hydrocodone, percodan/percocet, fentanyl, demerol, darvon/darvocet	Administration: Injected, smoked, swallowed, snorted. Effects: Euphoria, drowsiness and sedation, nausea, impaired coordination, confusion, constipation, slowed breathing. Increased risk of exposure to infectious diseases (hepatitis, HIV), poor pregnancy outcomes, fatal overdose, addiction. Potential harm from inconsistent dosing and additives.
methadone	Administration: Swallowed, injected Effects: Like opioids, used to treat opioid addiction; overdose risk, slowed breathing rate

**Table 7. Hallucinogens & Psychotomimetics**

Examples of hallucinogenic & psychotomimetic drug	Usual Administration Route & Common Effects
LSD (lysergic acid diethylamide), mescaline (peyote), psilocybin ("magic" mushrooms)	Administration: swallowed, absorbed through oral tissues Effects: altered perceptions and feelings; hallucination, increased heart rate, blood pressure, body temperature, numbness, dizziness, sleeplessness, possibly paranoia/panic; may develop "flashback" experiences later

**Table 8. Steroids**

Examples of Steroids	Usual Administration Route & Common Effects
anabolic & androgenic steroids ( <i>not to be confused with corticosteroids</i> )	Administration: injected, swallowed, absorbed through the skin Effects: hypertension, changes in blood chemistry, liver damage, aggression, acne, infertility and other reproductive system changes

**Table 9. Inhalants**

Examples of Inhalants	Usual Administration Route & Common Effects
household & industrial aerosols (paint thinner, gasoline, glue, butane, refrigerant gases) nitrous oxide/laughing gas ("whippets," "poppers")	Administration: inhaled Effects: stimulant followed by depression, impaired memory, nervous system disruption, muscle weakness, damage to the cardiovascular system, loss of consciousness; risk of sudden death

**Classification by DEA Schedule of Drugs**

Many drugs, medications, and psychoactive substances are classified by the U.S. Drug Enforcement Agency ([DEA](#)), determining the legal status of their distribution and the rigor with which they need to be controlled. Federal policy assigned this responsibility to the DEA and the controlled substance scheduling system informs law enforcement and criminal justice system responses at local, state, and federal levels. The status of any substance can change according to new, emerging evidence and the DEA is constantly challenged to evaluate new or modified substances as they appear on the ever-changing scene. Additionally, new approved medical uses may emerge—for example, evidence concerning the potential medical applications of cannabis/marijuana, LSD, or “magic mushrooms” may lead to the reclassification of these substances at a federal level (regardless of state and local policy). Let’s take a look at how the DEA controlled substances scheduling system is organized.



Each scheduled substance receives its classification based on evidence concerning (1) its potential for abuse and (2) whether it has current, evidence-supported medical applications in the U.S. The schedule of controlled substances runs from [Schedule I to Schedule V](#)—the value relates to the severity of controls needed. In other words, a Schedule I drug is considered to need the highest degree of control—it is the most addictive category and usually lacks approved medical use in the U.S. A Schedule V drug, on the other hand, is still subject to regulation and controlled access, but the controls required are the least intrusive. For example, heroin is a Schedule I drug and certain prescription-required cold relief products that contain low doses of more heavily controlled substances are Schedule V drugs (see Table 10). Other medications and drugs may be purchased “over-the-counter” (OTC). It is illegal to distribute (“traffic” in) any scheduled drug (I through V) without a proper license to do so (e.g., by prescription from a licensed

pharmacy) and it is illegal to distribute Schedule I drugs at all (with the exception of a few research or specially approved uses).

If you wonder about any specific substances, you can check out the current status at <https://www.dea.gov/drug-scheduling>. In many instances, the DEA has scheduled the precursors or ingredients for making controlled substances, not just the controlled substance products themselves. For example, the Schedule II list includes opium poppy heads, not just opium and lysergic acid is a Schedule III while the LSD (lysergic acid diethylamide) for which it is a precursor is a Schedule I substance. Pseudoephedrine is available OTC but must be registered by a pharmacist since it can only be distributed in controlled amounts, because it is a precursor to the production of methamphetamine. Also, note that the scheduled drugs are not all “bad” drugs—in many cases, they are used in treating physical or mental health conditions. For example, methadone is a Schedule II substance used in treating opioid/heroin use disorders or Adderall® and Ritalin® are used to manage attention deficit disorder (ADD or ADHD). Also, note the situation with fentanyl—the pharmaceutically prepared medication is a Schedule II drug but the “street” or illicitly prepared (often imported) forms are Schedule I drugs.

**Table 10. Scheduled drug examples (adapted from DEA.gov)**

Level	Criteria	Examples
<b>Schedule I</b>	No accepted medical use in the U.S., lack of accepted safety for use under medical supervision, OR some narcotic medications that are used medically; all have a high potential for abuse	heroin, LSD, cannabis (marijuana), peyote, “Ecstasy”/XTC, PCP, synthetic heroin, MDMA, “khat,” “china white fentanyl” and other forms of fentanyl not approved for medical or veterinary use
<b>Schedule II</b>	High potential for abuse, with use leading to severe psychological or physical dependence; has accepted use in the U.S. under medical supervision	cocaine (and crack), methamphetamine, methadone, opium poppy heads/straws/capsules, Seconal®, Tuinal®, Vicodin®, Demerol®, oxycodone (OxyContin®), fentanyl, Dexedrine®, Adderall®, Ritalin®
<b>Schedule III</b>	Potential for abuse exists but is not as high as Schedule I or II; moderate to low dependence potential, but higher risk than Schedule IV	ketamine, anabolic steroids, testosterone, products with less than 90mg codeine per dose (e.g., Tylenol® with codeine), paregoric (combination product containing opium), lysergic acid (precursor for LSD)
<b>Schedule IV</b>	Low potential for abuse or dependence.	Ativan®, Xanax®, Valium®, Darvon®, Darvocet®, Ativan® (lorazepam), Ambien®, Tramadol®, Soma®, Dalmane®, Konopin®, VIBERZI
<b>Schedule V</b>	Potential for abuse is lower than for Schedule IV drugs; preparations containing limited quantities of certain drugs with more stringent scheduling (certain narcotics).	Lomotil®, Lyrica®, cough preparations with less than 200mg codeine per 100ml (e.g., Robitussin AC)

The DEA scheduling system relates to the well-publicized issue of prescription abuse—individuals using prescription (controlled) substances outside of their prescribed use. They acquire the drugs outside of the legal, licensed distribution system.



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<https://ohiostate.pressbooks.pub/swk5805coursebook/?p=122#h5p-40>



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# Ch. 3: Trending Topics

By now you may recognize the critical point that the substance use, misuse, and SUD arena is a dynamic, constantly changing scene and what we think we know about the topic at any point in time is likely to need review again in the future. In this chapter, we examine a couple of trending topics—topics where practitioners, scholars, investigators, and others may not share a common viewpoint. The most enduring point of contention and debate concerns the way the addiction or substance use disorders are viewed. The second concerns the appropriateness of adopting harm reduction approaches to solving the problems associated with substance use and misuse. Third, involves adoption of a recovery orientation in thinking about substance misuse and substance use disorders, particularly in terms of how programs, services, and policies are designed. Note that some contents presented in this chapter are both adapted from and informed the writing of an introductory chapter by Begun and Murray (*in press*), to the *Handbook of Social Work and Addictive Behavior* from Routledge.

## Defining Addiction

There involved in defining substance misuse and SUD than the clinical diagnostic protocols presented in the DSM-5 and ICD-11. As a start, consider the American Society of Addiction Medicine policy statement defining addiction (ASAM, 2011):

*Addiction is a primary, chronic disease of brain reward, motivation, memory and related circuitry. Dysfunction in these circuits leads to characteristic biological, psychological, social and spiritual manifestations. This is reflected in an individual*



*pathologically pursuing reward and/or relief by substance use and other behaviors. Addiction is characterized by inability to consistently abstain, impairment in behavioral control, craving, diminished recognition of significant problems with one's behavior and interpersonal relationships, and a dysfunctional emotional response. Like other chronic diseases, addiction often involves cycles of relapse and remission. Without treatment or engagement in recovery activities, addiction is progressive and can result in disability or premature death (p. 1).*



**ASAM** American Society of  
Addiction Medicine

Important aspects of this definition are recognition of:

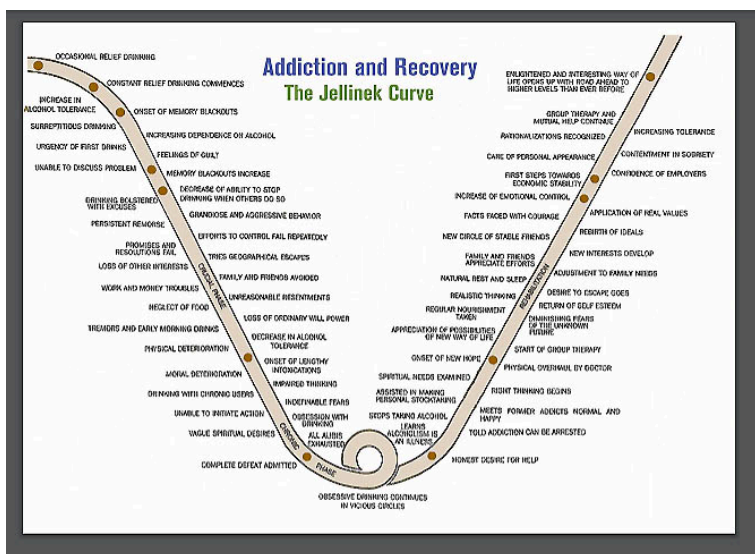
- the impact of addiction on biological, psychological/emotional, social, interpersonal, and spiritual aspects of life;
- the brain-behavior nexus in the development and maintenance of addictive behavior;
- the common experience of cyclical relapse and remission, and
- the potential for problem progression.

The ASAM definition reflects a “disease model” perspective—a model popular in the United States and many other areas, but not without controversy and critics, particularly in other parts of the world.

**Original disease model of addiction.** The original disease model of addiction emerged during the 1950s and 1960s regarding alcoholism, viewing addiction as a primary disease, not secondary to

other psychological conditions (Hartje, 2009). The original disease model of addiction was hailed as an important, less stigmatizing alternative than the prevailing moral model that placed blame on individuals for their addiction and deemed them deserving of its consequences and punishment (Thombs, 2009). Viewing addiction as a disease, instead, allowed the person to be seen as the “victim” of an illness, deserving of compassionate care and medically supervised treatment (Thombs, 2009). In the disease model, an individual’s choice to initially engage in substance may have been freely made; however, once initiated, the disease could take over: “intense cravings are triggered via physiological mechanisms, and these cravings lead to compulsive overuse. This mechanism is beyond the personal control of the addict” (Thombs, 2009, p. 561).

Research by E. Morton Jellinek was credited with providing early support for a disease model of addiction (Hartje, 2009). Based on a non-random sample of surveys completed by 98 men responding to an Alcoholics Anonymous newsletter, later expanded to include 2,000 histories, Jellinek (1952) identified four progressive phases of the disease: the prealcoholic symptomatic, prodromal, crucial, and chronic phases. The “Jellinek Curve” reflects how specific behaviors and experiences relate to the disease’s progression and recovery—its very design reflects the perception of a person “hitting bottom” before being able to recover from addiction (from <https://www.in.gov/judiciary/ijlap/files/jellinek.pdf>).



Despite methodological weaknesses in the evidence, the original disease model became popular with many practitioners and Alcoholics Anonymous programs, introducing significant implications:

- alcoholism was viewed as a chronic, progressive, incurable disease;
- professional treatment was specified as necessary to control this incurable disease;
- abstinence was viewed as the only defense against recurrence and the only reasonable goal for a person with this disease;
- substituting a different drug for alcohol was expected to manifest the same disease symptoms and progression (Hartje, 2009).

The original disease model and principles have greatly influenced assessment and treatment practices over the past 60 to 70 years.

There exist several points around which the original disease model of addiction has been challenged.

### **Heterogeneity**

**challenge to the original disease model.** Longitudinal studies documenting the natural course of alcoholism demonstrated significant inconsistencies with a disease

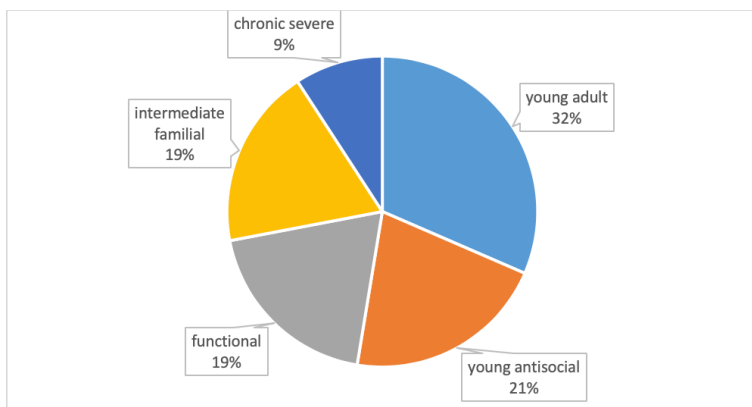


progression premise: multiple patterns were observed among men still alive 60 years after beginning the study, including continued alcohol abuse, stable abstinence, and return to asymptomatic/controlled drinking (Vaillant, 2003). Tremendous individual variation exists in patterns of addictive behaviors, as well as the severity of problems experienced by individuals at different points in time. Jellinek (1952) admitted that his was an “average trend” model in which individuals do not necessarily exhibit all of the symptoms associated with a phase, may differ in the sequencing of symptoms, and may differ in the duration of each phase; furthermore, “nonaddictive alcoholic” individuals may experience the identified negative consequences of alcoholism without experiencing a loss of control over drinking, and women may experience the disease differently.

This high degree of variability (heterogeneity) in expression called into question the perspective that alcoholism (or any substance use disorder) represents a single disease. Emphasis on the addiction/dependence end of the continuum of substance misuse “has resulted in a myopic view of substance abuse problems that has characterized them as progressive, irreversible, and only resolved through treatment” (Sobell, 2007, p. 2). Observed heterogeneity has informed the diagnostic schedules’ differentiations: different substances (and addictive behaviors such as gambling disorder) have distinct diagnostic codes. If “addiction” were a single uniform event there would be no need for multiple diagnostic categories—or different intervention strategies.

**Subtypes versus stages of disease.** There exist marked differences in how substance misuse/SUDs are expressed even within a single substance type. Challenging Jellinek's stage model of alcoholism, for example, is evidence of heterogeneity in "types" of alcoholism derived from a national sample (U.S.). The investigators based their typology on clinical characteristics of individuals meeting criteria for an alcohol dependence per the DSM-IV-R criteria that preceded the DSM-5 (Moss, Chen, & Yi, 2007). This analysis of U.S. National Epidemiological Survey on Alcohol and Related Conditions (NESARC) data led the authors to identify five "subtypes" of alcohol dependence, demonstrating clinical heterogeneity within the single diagnostic classification. The subtypes they identified were based on how participants clustered on diagnostic criteria, age of onset, family history, and presence of other co-occurring disorders. The five statistically determined clusters they identified were labelled: young adult, young antisocial, functional, intermediate familial, and chronic severe subtypes (see Figure 1). The groups demonstrated differences in their patterns of drinking, help-seeking, and response to intervention, as well. This study, based on a large, nationally representative sample reflected heterogeneity among persons engaged in a specific addictive behavior, and the wisdom of avoiding stereotypes about them—for instance, while the chronic severe subtype was the least common, it reflects a common stereotype of alcohol dependence.

**Figure 1. Subtypes of alcoholism (based on data from Moss, Chen, & Yi, 2007).**



### Treatment and the disease

#### model.

Additional important challenges to the disease model of addiction appear in the literature. Asserting that formal treatment for addiction is necessary has been challenged by evidence that many individuals experience significant, long-lasting improvement without engaging in formal treatment—sometimes referred to as “natural recovery” or “self-change”—typically, persons whose alcohol misuse is not of the most severe dependant nature (Sobell, 2007). Little is known about natural recovery in other substance misuse, though some evidence for its existence appears in the literature (e.g., Chen, 2006; Erickson & Alexander, 1989; Price, Risk, & Spitznagel, 2001). Possibly, the necessity for engaging in formal treatment varies by individual, severity of the problem, and characteristics of the substances or addictive behaviors involved.



### **Abstinence only prescription based on disease model.**

Viewing abstinence from substance use as the only defense against “disease” recurrence and the only reasonable goal for a person experiencing a substance use disorder has been challenged. Complete abstinence from all psychoactive substances is at one



end of a continuum in treatment strategies, commonly applied in U.S. medical practice (Glenn & Wu, 2009). A debated position is that the continuum of recovery includes controlled substance use, including the type of substance which a person previously used problematically. Between these positions is a question of whether psychoactive medications used to treat substance use disorders reflects recovery or is only a prelude to recovery not achieved until these medications are no longer needed. This question relates to an assertion that substituting one substance for another, despite its being safer, more controlled, or reducing harm, simply maintains the disease rather than offering a cure.

The word “sobriety” originally, historically implied temperate, moderated indulgence, not necessarily complete abstinence—an abstinence interpretation emerged during the 1900s (Glenn & Wu, 2009). Evidence since the 1970s indicates that some individuals achieve controlled drinking despite having previously engaged in an “out-of-control” drinking pattern, contrary to “the prevailing belief that any alcohol consumption causes an inevitable loss of control over one’s alcohol use” (Klingemann, 2016, p. 436). The debate about “controlled drinking,” “reduced-risk drinking,” and “moderation management” continues, and it is unclear how the evidence for and against it might apply to other substances and addictive behaviors. On the issue of the use of pharmacotherapy to assist in controlled drinking, recent meta-analysis concluded that three medications showed controlled drinking outcomes superior to a placebo, but the effects were small and inconsistent across studies (Palpcuer et al.,

2018). With or without medication, reduced-risk drinking (RRD) is seen in many Western European countries as one pathway out of addiction, and a legitimate treatment goal (Klingemann, 2016). As previously noted, the ability to engage in controlled use following a substance use disorder may vary by individual, severity of the problem, and characteristics of the substances or addictive behaviors involved.

Closely associated with the abstinence issue lies an additional point of contention with the final disease model of addiction, the expectation that substituting a different substance for the primary addictive behavior (e.g., misuse of alcohol, cannabis, or opioids) simply continues manifestation of the same disease of



“addiction” where the symptoms persist, as does the pattern of disease progression. This stance contributes to the hesitancy expressed by some practitioners that medically assisted treatment (MAT) and the use of pharmacotherapies to treat substance use disorders maintains the (incurable) disease rather than treating it. Evidence of the effectiveness of these approaches for many persons, including eventual weaning from medication, contradicts this contention.

**Loss of Control Concept.** The original disease model of addiction expressed another point with which scholars and practitioners have taken issue: applying “loss of control” as a defining criterion. The prior moral model attributed individuals’ use/misuse of alcohol, tobacco, or other drugs to moral failure or personality weakness, holding them “personally responsible for creating suffering for themselves and others” (Thombs, 2009, p. 561). The original disease model, as previously discussed, did not take a position on a person’s initial decision to use a substance, but argued



that the “disease” may take over, eventually rendering an individual helpless to control the behavior. Heather (2017) argued against the “compulsion” aspect of the disease model where addictive behavior “is said to be carried out against the will,” and “marks the turning point from normal, recreational drug use to addictive drug use” (p. 15). His counter-argument does not support a moral failure/ blame stance toward addiction; instead, he emphasized the power of environmental, contextual, and reinforcement paradigms operating to influence behavioral choices related to continued engagement in substance misuse (or other addictive behaviors). One problem with the loss of control concept is that individuals may reframe it in terms of, “I can’t help myself,” excusing themselves from taking responsibility for the behavior or taking steps toward recovery.

Contemporary brain disease model and biopsychosocial perspective. As previously noted, recognition of the brain-behavior nexus in the development and maintenance of addictive behavior is important and necessary to understanding, intervening around, and recovery involving addictive behavior and related problems. Evidence concerning the neurobiology of substance use and mechanisms involved in the transition to substance use disorders has expanded in



many directions over the past two decades, contributing to a widening variety of treatment and prevention intervention strategies (Volkow & Koob, 2015; Volkow, Koob, & McLellan, 2016).

Proponents of a contemporary brain disease model of addiction argue that: “After centuries of efforts to reduce addiction and its

related costs by punishing addictive behaviors failed to produce adequate results, recent basic and clinical research has provided clear evidence that addiction might be better considered and treated as an acquired disease of the brain” (Volkow, Koob, & McLellan, 2016, p. 364). The U.S. National Institute on Drug Abuse applies the following definition of addiction:

*“Addiction is defined as a chronic, relapsing disorder characterized by compulsive drug seeking and use despite adverse consequences. It is considered a brain disorder, because it involves functional changes to brain circuits involved in reward, stress, and self-control, and those changes may last a long time after a person has stopped taking drugs. Addiction is a lot like other diseases, such as heart disease. Both disrupt the normal, healthy functioning of an organ in the body, both have serious harmful effects, and both are, in many cases, preventable and treatable. If left untreated, they can last a lifetime and may lead to death” (NIDA, 2018).*

Chronic, relapsing diseases like diabetes or high blood pressure often have a strong behavioral health component—just as substance use disorders. While these disease conditions may worsen over time, the outcome is not immutable—outcomes can be affected by behavioral health interventions, as well as self-directed changes in behavior and/or environment.

## Biopsychosocial

Biology and psychology intersect where substances altering the brain’s reward and emotional circuits influence individuals’ experiences, learning, memory, affect, executive function, decision-

making, expectancies, withdrawal symptoms, and cravings, with profound implications for continued engagement in addictive behavior, as well as strategies for changing addictive behavior patterns. Understanding brain-behavior processes is necessary; however, this alone does not impart sufficient knowledge. Biological and psychological processes do not occur in a vacuum, but within complex, impactful social contexts and physical environments. For example, evidence that early exposure to alcohol and other substance misuse increases the odds of developing a substance use disorder later in life (Odgers et al., 2008) invokes mechanisms of multiple types: changes to the brain (biology); learning, social learning, and expectancies (psychology); social norms and access (social context/environment). Not only does recovery occur within social contexts (Heather et al., 2018), biological, psychological, and social interventions all may play a role. Furthermore, social and psychological interventions can influence neurobiological processes (Volkow, Koob, & McLellan, 2016); biology does not confer destiny but has a powerful iterative relationship with the other domains. Viewing addictive behaviors from an integrated biopsychosocial framework is required and reflected throughout this book.

### **Harm Reduction**

A somewhat contested topic related to substance misuse and related problems is **harm reduction**. First appearing in the literature during the late 1980s and early 1990s, the term “harm reduction” was used to describe attempts to reduce adverse consequences associated with substance misuse, without necessarily eliminating substance use (Single, 1995). Two general levels of harm reduction effort emerged in the literature: clinical practice and policy interventions. Underlying harm reduction is recognition of the potential harms associated with engaging in addictive behavior (e.g., substance misuse or problem gambling), as well as knowing that some individuals will continue to engage in these behaviors, at least for an unknown length of time, despite the potential for harms to self and others. “The essence of the concept is to ameliorate adverse

consequences of drug use while, at least in the short term, drug use continues” (Single, 1995, p. 287).

Examples of harm reduction strategies at the program/policy level that have at least some support from research evidence are:

- clean needle exchange programs,
- medically supervised injecting facilities (more common in other countries than the U.S.),
- heroin-assisted treatment (more common in other countries than the U.S.),
- distribution of fentanyl testing strips, and
- wide public distribution of opioid overdose reversal kits (Narcan).

These strategies can reduce the risk of infectious disease transmission and drug overdose, among other potential harms (Drucker et al., 2016).



Examples of harm reduction practices at the clinical level include:

- nicotine replacement therapy to reduce harms associated with smoking tobacco products, and
- medication-assisted treatment (MAT) involving opioid substitution drugs (e.g., methadone, buprenorphine) to reduce harms associated with use of unregulated “street” drugs.

While harm reduction as a public health and social work strategy makes intuitive sense on the surface, controversy revolves around philosophy and implementation, led to some degree by a misunderstanding of harm reduction (Drucker et al., 2016). One argument against



harm reduction strategies is that it may be mis-perceived as sanctioning the problematic behavior. The evidence on this is mixed, however. For example, while zero-tolerance/abstinence-based messaging was more effective in curbing college students' future drinking in several studies (Abar, Morgan, Small, & Maggs, 2012; LaBrie, Boyle, & Napper, 2015), in another, this was true only among students who currently consumed two or fewer drinks per week; harm reduction messaging outcomes were more favorable among students currently engaged heavy drinking (Napper, 2019). Thus, the anti-harm reduction argument that it seems to sanction the behavior, thereby contributing to the problematic behavior, is only partially supported by evidence. An argument that harm reduction (reducing the negative consequences) interferes with motivation to seek treatment and/or quit engaging in the problematic behavior is also countered with the argument that, as a result of engaging in harm reduction programming, individuals may then become encouraged to engage in treatment to reduce or cease substance misuse (Drucker et al., 2016). An argument against nicotine or opioid replacement therapies is that the person continues to experience substance dependence. However, use of these therapies may allow the individual to gradually become weaned from dependence in a controlled manner, supported by behavioral therapies. While this argument is offered in support of e-cigarettes/vaping as a harm reduction tool, evidence is mounting that significant risks of harm are associated with these devices (including injury from malfunctions/battery problems, chemical

exposure not being reduced as much as advertised, worsening of the nicotine dependence, and poisoning of children and pets from the liquid nicotine).

## Recovery Orientation

A [recovery orientation](#) refers to a host of values, beliefs, and behaviors related to how individuals engage in and experience the process of recovery from a SUD (Bersamira, in press). The recovery orientation is fundamentally informed by the individuals' own definitions of the problems, solutions, and



subjective experiences, rather than those being imposed by others. Built into this orientation are issues such as having individuals define for themselves what constitutes “recovery”—this may or may not include abstinence as a goal, for instance. Another aspect has to do with adopting a holistic view where individuals’ recovery is embedded in a context of all life structures, functions, and wellness, including their future growth and development as a person, not just changes in past substance use/misuse behavior (Kaskutas et al., 2014). Thus, recovery does not simply mean achieving the absence of disease, it means promoting wellness across all life domains.

Many individuals and professionals actively engage in advocacy related to a general recovery-oriented movement, promoting recovery-oriented services and policy (Bersamira, in press). This orientation includes engaging indigenous and professional services and relationships in supporting individuals’ long-term recovery (and their families), as well as shaping the culture of communities and

policy (White, 2008). For example, peer support systems are often an integral aspect honored and incorporated in a recovery orientation: peers being others who have lived the experience and found their own pathways to recovery. In other words, recovery-oriented systems of care differ quite markedly from traditional treatment systems: their services are more person-centered, self-directed, and strengths-based (Bersamira, in press).



*An interactive H5P element has been excluded from this version of the text. You can view it online here:*

<https://ohiostate.pressbooks.pub/swk5805coursebook/?p=124#h5p-43>

# Ch. 4: Alcohol versus Opioid Deaths

## **Alcohol versus Opioid Deaths**

There is one more important point to make as we conclude this topic of opioid problems. While opioid misuse is a critically important issue in the United States, the topic should not be allowed to totally eclipse attention to other substance use problems. For example, alcohol continues to be the cause of illness, injury, and death at a remarkably high rate. As the next article notes, “more than 33,000 Americans died due to alcohol-induced causes in 2015” (Lopez, 2016). The number of opioid-related deaths has been rising fast over the past 15 years, but has yet to surpass the rate of alcohol-related deaths. Here is the weblink information for the story (and a link is provided in your Carmen coursesite, as well):

**<https://www.vox.com/policy-and-politics/2016/12/9/13898956/alcohol-deaths-2015>**



# Ch. 5: Summary

In the readings for Module 2 you read about:

- Key terms related to substance use, misuse, and use disorders and how these terms are used—substance use, substance misuse, substance use disorder (SUD), tolerance, withdrawal, and a biopsychosocial perspective;
- Current diagnostic criteria applied to alcohol and other substance use disorders (AUD and SUD) in the United States and around the world (DSM-5 and ICD-11);
- Two different systems used in the classification of different types of substances—classification by effects on the brain and body and the DEA schedule of controlled substances; and,
- Three key trending topics in the area of substance use/misuse—the historical and contemporary stances on the disease and brain disease models of addiction, harm reduction, and a recovery orientation.

At this point, you have acquired a great background for engaging with our next topic—Module 3’s examination of the biological models reflected in a biopsychosocial understanding of substance misuse.

# Module 2: Key Terms

**biopsychosocial:** a perspective commonly applied in the substance use arena recognizing the interacting and integrative influences of biological, psychological, and social/physical environment context.

**DEA:** The U.S. Drug Enforcement Agency, setting policy regarding the status of controlled substances.

**DSM-5:** The American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (version 5) used in the diagnosis of substance use disorder and many other mental/psychiatric conditions; widely used across the U.S. and some other nations.

**harm reduction:** An approach to intervention (treatment or policy) where the short-term goal is to reduce potential for harmful outcomes resulting from substance misuse, whether or not the substance misuse is eliminated or reduced [note this does not mean that there is not also a long-term goal of reducing or eliminating the substance misuse, as well].

**ICD-11:** The World Health Organization's International Classification of Diseases and Related Health Conditions used in the diagnosis of substance use disorder and many other physical and mental/psychiatric conditions; widely used in other nations.

**recovery orientation:** An holistic approach to supporting the "whole" person in recovering from substance use disorder that integrates professional, paraprofessional, and natural/indigenous helpers in the process and addresses all aspects of wellness promotion [note that this often includes advocacy efforts].

**Schedule I-Schedule V drugs:** Classification categories for controlled substances established by the U.S. DEA; Schedule I is the most highly controlled class, having the greatest potential for abuse and no recognized medical use in the U.S., and Schedule V

is the least controlled class of substances that remain controlled substances (as compared to over-the-counter/OTC products).

**substance misuse:** Use of psychoactive substances in risky patterns or risky situations.

**substance use:** Introduction of psychoactive substances into the body.

**substance use disorder:** A diagnosable condition, meeting specific criteria, distinguished by degree of severity (number of criteria met) and type(s) of substances involved; discrete from other mental/psychiatric/behavioral health conditions in that the symptoms are influenced by substance use/misuse, and discrete from substance withdrawal syndrome.

**tolerance:** With repeated use, requiring higher doses of a substance (or type of substance) to achieve the same effects or experiencing lesser effects (even withdrawal) when the same dose is used [note that this describes acquired tolerance; base tolerance refers to the amounts initially needed to achieve the same effects experienced by others].

**withdrawal:** Following repeated use of a substance (or type of substance), the body adapts to the presence of the substance such that a person experiences physical and/or psychological effects/symptoms when the substance use stops or markedly decreases [note that withdrawal occurs to a greater extent with some types of substances than others and that unmonitored withdrawal from some substances can be deadly].

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*August 14, 2019 version*





## PART II

# MODULE 1: INTRODUCTION

Module 1 readings introduce major concepts relevant to understanding the theories that explain substance use, substance misuse, and substance use disorders. First, the concept of “psychoactive substances” is defined. Second, general epidemiological data and trends in substance misuse and substance use disorders are presented and explained. Finally, the importance of attending to the kind of language used in discussing substance misuse, individuals engaged in substance misuse, and individuals experiencing substance use disorders is explored. The information presented in these reading materials sets the stage for what is presented throughout the remainder of the course.

## Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Explain what “psychoactive” substances means
- Describe the scope and impact of substance use as a national and global problem (epidemiology)
- Describe historical trends in responses to substance use/misuse in the U.S.
- Identify and resolve where stigmatizing language about substance use and addiction occurs.



## Ch. 2: A Brief History of Substance Use and Policy Responses in the U.S.

While substance misuse is a contemporary social problem, the story of humans experiencing problems related to the use of psychoactive substances is at least 4,000-10,000 years old



(Hanson, Venturelli, & Fleckenstein, 2015; Howard, Garland, & Whitt, 2013; Singer, 2012). United States history is peppered with documentation of problems associated with alcohol and other drugs. For example, the opiate drug morphine was widely used during the Civil War to manage wounded soldiers' pain, leaving many of them experiencing morphine addiction as a result. Subsequently, heroin became available and marketed as a “non-addicting opiate with greater analgesic potency than morphine” (Kornetsky, 2007, p. 96). Prior to the Civil War, 60-75% of Americans experiencing opium or morphine addiction were women, in large part because physicians often prescribed opiates to deal with a wide variety of “female” complaints (Blumenthal, 1998). In addition, physicians of the time often prescribed alcohol as a treatment for opiate addiction, and many socially acceptable and widely accessible medicines contained very high alcohol or opium content (Plant, 1997; Straussner & Attia, 2002; van Wormer & Davis, 2013). Cocaine was also prescribed and marketed in this way. See this historic advertisement promising an instant cure for oral pain (including babies' teething pain).

At around the end of the 19th century, awareness of potential

harms associated with these substances spread. The U.S. (and other nations) has since implemented various policy efforts to reduce both or either supply and demand for different kinds of drugs (Vakharia & Little, *in press*). Protecting public health was not the only motivation in many instances, however.

## Early U.S. Policy and Legislation Efforts.

The first federal policy prohibiting distribution (supply) and non-medical use (demand) of a drug was the Opium Exclusion Act of 1909 (Vakharia & Little, *in press*). Opium used for medical purposes remained legal, but opium prepared for “smoking” no longer was. At this point in history, large numbers of Chinese immigrants were working in the U.S. and opium smoking was associated with this population. The common use was not outlawed, only the form of opium used by Chinese immigrants (Vakharia & Little, *in press*).

The next major federal policy, the Harrison Narcotic Act of 1914, was directed at drugs derived from opium or coca leaves, to control their production, distribution, and use. Possession or use of a narcotic (this included cocaine) without a physician's prescription was a violation that states could criminalize. Cocaine was targeted, possibly for political reasons parallel to the situation with opium (Vakharia & Little, *in press*): up until the early 1900s, cocaine was commonly added to beverages and medicinal tonics because of its energizing properties (and boosting worker productivity). Even though most individuals using cocaine were white, concern grew



over its increasing popularity within the black community, particularly across the Southern U.S. (Vakharia & Little, *in press*). An added public policy motivation: governments could now collect special taxes on the production and distribution of these drugs. Tobacco has been taxed at the federal and state levels since the Civil War, with the amount fluctuating (until 1983) according to governments' need to generate revenue (IOM,1994).

The Harrison Narcotic Act represented early prohibition efforts and laid the foundation for much of the substance-related policy enacted in the U.S. and by individual states or local communities, including marijuana and alcohol control efforts (Vakharia & Little, *in press*). In attempting to prevent the spread of alcohol or other drug addiction, some public policies advocated institutionalization in psychiatric or criminal facilities, as well as forced sterilization as part of the negative eugenics movement (Straussner & Attia, 2002; White, 1998). One effort with which you may be somewhat familiar was passage of the 18th Amendment—commonly known as Prohibition. The 18th Amendment to the United States Constitution banned the manufacture, sale, or transportation of “intoxicating liquors,” but not the drinking of alcoholic beverages. (This picture shows agents pouring liquor confiscated in a New York City raid during Prohibition; it comes from the National Archives).

## Prohibition Era.

Although the combination of the 18th Amendment to the United States Constitution and the Volstead Act (which clarified that beer and wine were included as alcoholic beverages) were implemented beginning in 1920, many states



had already enacted their own local prohibition laws (Hanson, Venturelli, & Fleckenstein, 2015; Kelly, 2017). You might find it interesting to pursue historical literature documenting the intersections of alcohol/drug policy with historical and sociological trends such as the temperance movement, women's suffrage, immigration, organized crime, classism and racism (see for example, Straussner & Attia, 2002; van Wormer & Davis, 2013). Many of these historical policy patterns have implications for today's politics and policy debates, as does the extensive economic impact of both local and international trade in substances such as alcohol, tobacco, coffee, tea, opium, cocaine, and others.

The 21st Amendment repealed the federal alcohol prohibition laws in late 1933; some states and local jurisdictions were slower to change their own prohibition policies. Some states continue to have “dry” communities restricting the sale or distribution of alcohol, and some communities maintain “Sunday” or “blue” laws banning the sale of alcohol during certain hours.

It was also during the 1920s and 1930s that many states developed prohibition-style policies about marijuana, and the federal government got involved in 1937 with the passage of a Marijuana Tax Act and more severe criminalization policies during the 1950s. Marijuana policy concerns cannabis plant products; the word marijuana came



from Mexico, but its use in U.S. policy is becoming recognized as having racist and propagandist connotations by many scholars (Malcolm, *in press*). Historical roots of marijuana prohibition include racial/ethnic concerns about Mexican immigrants and African Americans that parallel opium and cocaine policy regarding Chinese immigrants and Southern black workers (Malcolm, *in press*).

## Evolution of Contemporary U.S. Drug Policy.

During the 1960s, many programs and policies aimed at addressing both the supply and the demand sides of the drug trade were established. The term **“War on Drugs”** began to appear around 1971, referring to stepped-up drug criminalization and law enforcement efforts (McNeece & DiNitto, 2012; Schori & Lawental, 2013). While



these programs focused on our nation’s internal drug problem, it is virtually impossible to separate the U.S. drug war efforts from international policy, international relations, and global economics. It also had political undertones and overtones related to race, age, and the “counter-culture” presence in America at the time.

One criticism of “America’s Longest War” (the title of a 2013 award-winning documentary) has great relevance to social work and disciplines concerned with social justice: the War on Drugs contributed to extreme racial and gender inequities in the nation’s incarceration rates (Bush-Baskette, 1999; Chesney-Lind, 1997). For example, by the early 1990s, 74% of individuals serving prison sentences for drug possession were black, despite accounting for only 13% of individuals who use drugs (Kilty & Joseph, 1999). The War on Drugs also helps explain the relative explosion of women in prison for non-violent, drug possession charges that occurred during the late 1980s to 1990s—leading to a declaration that the War on Drugs became a “War on Women” (Bloom, Chesney Lind, & Owen, 1994). Another criticism of the War on Drugs addresses its high economic costs: the Office of National Drug Control Policy’s (ONDCP) FY 2020 National Drug Control Budget request was \$34.6 billion, an increase of \$1.3 billion over the actual FY 2019 budget (<https://www.whitehouse.gov/briefings-statements/white-house->

[seeks-billions-record-investments-stop-drug-epidemic/](#)). The ONDCP is a component of the President's White House Executive Office, created by the 1988 Anti-Drug Abuse Act.

## *Pregnant Women and Substance Use.*

Part of the concern about a “War on Women” stems from how policy responses (mostly at the state level) to women’s use of alcohol or other drugs (AOD) during pregnancy. States and local communities differ markedly in their policy responses to this issue. The responses run the gamut from dealing with the public health aspects (the health of mother and baby) to criminalization. For example, in some



states, a pregnant woman can be involuntarily committed to a treatment facility, jail, or relative’s home for supervision to prevent her continued use of substances known to be harmful to a developing fetus. Many states have policies relating to the substantiation of child maltreatment allegations when a pregnant mother uses alcohol or other drugs. While intended to help protect the unborn child from potentially harmful drug exposure, these policies are controversial, as they also may discourage women from seeking much-needed prenatal care for fear of discovery and becoming subject to consequences imposed through the courts and child welfare system.



## *Drinking Age Legislation.*

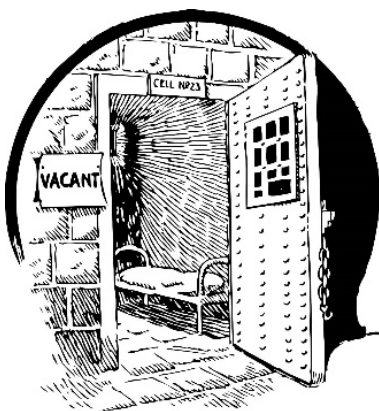
Drinking age legislation in the U.S. currently aims to restrict alcohol use by persons under the age of 21 years. You may find it hypocritical that an 18-year-old person is treated as an adult in other domains (legal rights to marry, join the military, enter into legal contracts), but not legally allowed to purchase alcohol. Drinking establishments are certainly concerned about reduced revenue



from not being allowed to legally serve alcohol to 18- to 20-year-olds. On the other hand, there exists compelling evidence that higher drinking age minimums are associated with lower traffic fatality rates. Another rationale involves an attempt to mitigate the potential harms associated with exposing the still-developing young adult brain to alcohol—major developmental changes in brain structure and function, beginning early in puberty, continue well into the period of early adulthood (Spear, 2000; more about this in our focus on alcohol module). Raising the legal age to be well over 18 eliminates confusion about enforcing alcohol-free zones in high schools and many parts of college/university life, as well. This policy periodically becomes contested, tested, and retested in the United States, including a period during the 1970s when different states had different legal drinking ages of 18, 19, and 21. Drinking age policy is determined at the state level, however federal highway funding is tied to state drinking age policy and governing the states' uniform decision to support a minimum legal drinking age of 21 years.

## *Decriminalization Efforts.*

Our nation has an opportunity to learn from the contemporary “natural experiment” in policy reform whereby several states decriminalized the production, distribution, possession, and/or use of cannabis for medical and/or recreational purposes (more about this in our focus on cannabis module). Some hypothesize that



[decriminalization](#) of substance possession or use reduces economic incentives for illegal production and distribution of drugs, allowing government entities to increase revenue through taxation (McNeece & DiNitto, 2012). Decriminalization is contested, however, as potentially contributing to increased rates of substance use disorders and other health risks associated with substance use, as well as related problems such as driving under the influence and community safety. Law enforcement professionals expressed grave concerns regarding the potential for increased demands on police forces already stretched by the need to manage alcohol-related situations if marijuana is also legally used by the general public. Recent evidence suggests that the presence of legal (medical) marijuana dispensaries are associated with increased violent and property crime rates in adjacent areas (Freisthler, Ponicki, Gaidus, & Gruenwald, 2016).

Addiction treatment providers have expressed concern about the potential impact of easier access on individuals already in recovery from substance use disorders and the potential for further stressing an under-resourced service delivery system with an increase in demand for intervention to address problems with marijuana use. Prevention experts are concerned about the message that

legalization/decriminalization might convey to young people considering initiating substance use. And, there continues to be controversy as to the potential (as yet, unknown) effects on the health care system that might result from an increase in disease or disability resulting from individuals' long-term use of marijuana products—along the lines of what we see with alcohol.



However, social justice advocate relates significant inequities in how the criminalization of cannabis and other drugs is enforced, leading to both a mass incarceration trend and tremendous racial/ethnic (and gender) disparities in who becomes incarcerated in the nation's, states', and local communities' jails and prisons. Mass incarceration beginning in the mid-1970s meant the incarceration rate almost tripled from 1970 (96 per 100,000 population) to 1990 (over 300 per 100,000; Lloyd & Fendrich, *in press*). Not only were War on Drugs policies responsible for this trend, so were policies and policy enforcement stemming from Sentencing Reform Act (1984), Anti-Drug Abuse Act (1986), and Omnibus Anti-Drug Abuse Act (1988) policies concerning sentencing guidelines establishing mandatory minimum penalties for drug crimes (Lloyd & Fendrich, *in press*). Disparities in incarceration of persons of color were further stimulated by differential sentencing for “crack” cocaine (more

commonly used by persons of color) compared to powdered cocaine (used by more affluent and white individuals). Advocating for “smart decarceration” often means advocating for less punitive (and more treatment) responses for low-level and non-violent drug involvement (see Pettus-Davis & Epperson, 2015).

## *Drug courts.*

Traditional drug-control methods of the criminal justice system, such as mandatory incarceration and harsher penalties, along with court-mandated treatment following release from incarceration, have not proven to be sufficiently effective to curb the problems associated with illicit drug use (Broadus, 2009). In



addition, these efforts were wreaking havoc on the court system by creating tremendous backlogs of cases considered to involve relatively minor, non-violent offenses, and pushing jail populations far over capacity at great public expense. In response, a movement emerged during the early-1990s to establish special courts for managing nonviolent drug-related cases. The mission was to engage individuals in court-monitored, structured, evidence-supported treatment and divert them from being incarcerated if they complied with the treatment plan. By 2018, over half of all U.S. counties sponsored at least one of over 3,100 drug courts in operation (Lloyd & Fendrich, *in press*). Each program involves an interdisciplinary team of criminal justice and mental health professionals responsible for creating an individualized comprehensive plan for each program participant and monitoring participant progress. Failure to comply

with the court-treatment plan results in the court levying the traditional sentences for the original offenses. Short-term outcome studies support the drug court model as participants, on average, remain in treatment longer than in traditional treatment settings and experience fewer relapse events, recidivism rates are lower, and participants are able to improve education, housing, and health, as well. Results generally are not as promising for juvenile drug courts as for adult drug court (Lloyd & Fendrich, *in press*).

### *Harm Reduction Policies.*

Some strategies and policy approaches are based on a principle that has come to be known as harm reduction. While the goal always remains reducing substance misuse risk by ending high-risk substance use behaviors (alcohol or other drugs), it is not always wisest to wait for risky behaviors to cease. While waiting, harms to individuals, families, and communities accumulate. Instead, often it is wiser to intervene in ways that reduce the potential risks, harms, and other negative consequences associated with the behaviors in the meantime. The harm reduction approach, derived from public health rather than criminalization motivations, aims to improve quality of life for individuals, families, and communities associated with the risky behaviors (Collins et al., 2012). Some harm reduction policy examples include:

- programs to prevent driving while under the influence of alcohol or other substances, while not necessarily stopping a person from using AOD;
- clean needle and syringe exchange programs to reduce risk of exposure to blood-borne communicable diseases like HIV/AIDS and hepatitis;
- supervised drug-use settings where individuals' drug use and safety are monitored by someone whose judgement is not

- substance-impaired (more common in European nations);
- fentanyl testing of heroin/opioids or other “street” drugs to prevent unexpected opioid overdose;
- supportive housing for which abstinence is not an eligibility requirement;
- making opioid overdose reversal resources (Narcan) available to first responders to save the lives of individuals who might otherwise die before professional treatment is accessible.

On one hand, harm reduction is viewed as being practical and humane. Harm reduction programs may also serve as pathways to enter treatment and reduce substance misuse. On the other hand, some argue that harm reduction is too “soft” on individuals who break the law through substance misuse. Abstinence-only policies are necessary to stop the harms caused by substance misuse, and risk-reduction approaches do not do enough to stop substance misuse. In addition to harm reduction policies, there exist treatment intervention approaches in the harm reduction spirit, as well.

### *Access to Treatment.*

Improving access to treatment for substance misuse and substance use disorders represents another modern policy/advocacy front with great social work significance. In the previous epidemiology sections, you learned about the considerable gap that exists between the need for these services and the numbers of



individuals (and families) able to receive them. A person's ability to engage in formal, professional treatment for these problems often depends on the ability to pay with insurance or self-pay dollars.

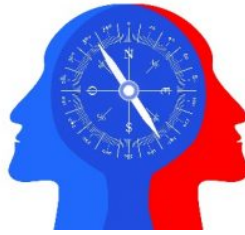
One potential advantage of the Affordable Care Act (ACA) first implemented in the United States during 2013-2014 was the potential for increased access to mental health and substance use disorder treatment services for many individuals. With the passage of the ACA:

- young people could remain on a parent's Medicaid plan until the age of 26 years (remember that 18-25 was the age period the greatest number of individuals engaged in substance misuse);
- subsidies helped more individuals afford health insurance;
- annual and lifetime benefit limits and limits on the number of visits for behavioral health services were eliminated;
- behavioral health care became more affordable by ensuring co-pay expenses could not be greater than those for physical health services; and,
- insurability was protected for individuals experiencing a pre-existing condition in their medical records (having a history of a substance use disorder would be a pre-existing condition necessitating protections, no matter how long the person has been in recovery).

The federal Mental Health Parity and Addiction Equity Act of 2008 also helped regulate the health plan/insurance industry regarding benefits for individuals with substance use disorders in their medical histories.

Despite the excitement over expanded coverage and protections, concerns arose regarding the treatment system's ability to meet the anticipated increase in demand: Do we have enough trained professionals to meet the experienced need?

At the end of 2016, the U.S. Congress passed two major pieces of legislation related to substance use and addiction. The first was the Comprehensive Addiction and Recovery Act (CARA) that provided legal status for many harm reduction strategies, such as increased access by non-physicians to naloxone for reversing an opioid overdose. However, CARA did not provide funding for these approaches. The second was the 21st Century Cures Act that provided federal funding to “accelerate the discovery, development, and delivery of 21st century cures” and other purposes (<https://www.congress.gov/bill/114th-congress/house-bill/34/text>). In addition to ensuring specific funding for the NIH and Federal Drug Administration, the act provided funding for states with a relatively high prevalence of opioid use disorders to develop their responses for addressing the opioid abuse crisis. This included prescription drug monitoring programs, prevention activities, health care provider training about best practices, supporting access to treatment programs, and other public health-related activities to address the identified crisis. The impact of policy revisions regarding health care coverage since the 2017 change in national leadership remains to be seen. The White House created the President’s Commission on Combatting Drug Addiction and the Opioid Crisis (March 2017) with the mission of studying “the scope and effectiveness of the Federal response to drug addiction and the opioid crisis...and make recommendations to the President for improving that response” (<https://www.whitehouse.gov/the-press-office/2017/03/30/presidential-executive-order-establishing-presidents-commission>). However, at the same time, the Office of National Drug Control Policy, a component of the President’s Executive Office, risked being significantly defunded. Clearly, there exists





considerable ambivalence in the policy response to substance misuse and treatment for substance use disorders in the U.S.



**Before you read on, take a moment to jot down your “best guess” answers to the following questions:**

***Thinking About Policy Issues***

For each of the following topics, consider what evidence supports your position, and what evidence might counter your position.

- **Drinking Age Legislation:** What do you think about the current minimum legal drinking age policies in the United States? What do you know about policies in your own community regarding being a minor in possession of alcohol, driving while under the influence as a minor, and the provision of alcohol to underage minors? How might these issues apply to cigarettes, e-cigarettes, and other tobacco products?
- **Drinking or Drug Use during Pregnancy:** What do you think should be the state's policy and why? What are the social justice issues involved? What are the likely “real world” implications of implementing (or not) such ideas in practice.

- **Prohibiting versus Decriminalizing Policies:** Thinking about the historical policy called Prohibition, what are the parallels and differences with regard to policies restricting distribution and use of other substances like marijuana, opioids and heroin. Consider the effectiveness or ineffectiveness of public education strategies that involve “scare tactics” and “Just Say No” policy responses to preventing substance use initiation by young people—what worked and what did not, and for whom were these approaches effective and for whom were they problematic? Why do you think the problems were or were not solved this way?
- **Naloxone access:** Naloxone is not a cure for addiction, but the immediate life-or-death health crisis may be resolved if delivered in time. There is no question that many lives have been saved (in the short-term, at least) with this overdose reversal intervention. The wholesale price for a 3-dose administration (more than one dose is often necessary for individuals who used heroin/fentanyl combinations) can cost over \$4,200. Though the costs to an individual person or family member can be offset to between \$0-\$125 in some communities through donated doses, grants, and public funding, the cost of doses provided by first responders may or may not be offset.
- What do you think about policy allowing laypersons in the community to obtain naloxone to use if they witness an opioid overdose (e.g., a friend, family member, or someone else using drugs together)?
- What about providing it individuals with diagnosed opioid addiction, to carry for others to administer if they overdose?
- How do you feel about doing this for someone yourself (and perhaps conduct rescue breathing during the time it takes to work)?

- How do you feel about these costs affecting city/county/state budgets for first responders?
- How do you feel about pharmaceutical companies charging so much for this life-saving treatment, despite their having invested heavily in its development and testing?

# Ch. 3: Considering the Language We Use

At this point, you have developed a general “big picture” about the topic of our course: substance use, substance misuse, and SUD. Throughout Module 1 so far you have read about alcohol and other substance use. You may not have noticed the language used to describe individuals involved with these substances or who experience substance-related problems. For example, you did not read about “substance users,” you read about individuals who engage in substance use (or misuse), you did not read about “alcoholics” or “addicts,” you read about individuals experiencing alcohol use disorder (AUD) or a substance use disorder (SUD).

Social workers have long been aware of the importance of the way we use language and the deleterious consequences of applying labels to people. You may find that many resources use stigmatizing labels and terms. Not only do labels tend to stereotype, stigmatize, and marginalize people, they also create a pessimistic mindset about the possibility for change. In the field of addictions, awareness about the harms associated with stigmatizing labels like “addict” or “alcoholic” are discussed with increasing frequency. As the field gradually becomes more conscious and aware of this problem in professional writing and speaking, it is important that we all become more conscientious about changing how we discuss individuals involved with substances or affected by someone’s substance use. It is a behavior, not a person’s defining characteristic.

Getting us thinking along these lines is the purpose for assigning the following reading:

- Begun, A.L. (2016). Considering the language that we use: Well worth the effort. *Journal of Social Work Practice in the Addictions*, 16, 332-336.

This final chapter for Module 1, emphasizes the importance of paying attention to the language that we use in discussing and describing people who engage in alcohol or other drug (AOD) use/misuse who experience substance use disorders (SUDs). After reading the assigned article, remember to return here for the chapter and module conclusion.

- [LINK TO THE Carmen COURSEWORK site](#)
- ARTICLE: <http://www-tandfonline-com.proxy.lib.ohio-state.edu/doi/abs/10.1080/1533256X.2016.1201372>

When you are finished reading this brief article:

- Begin to practice ways of changing the language that you use. For example, start by simply identifying stigmatizing labels used by others when you are reading, listening to radio, television, or movies, and talking about social work issues in your classes or with friends.
- As a next step, think about creative ways of editing what you read or heard to remove the labels and describe people in terms of their experiences instead.
- Think about how this might make a difference in how these individuals are viewed and how they might view themselves as a result.

Here is an exercise for you to practice these new skills. Imagine that you are the instructor for our course. First, read this hypothetical student discussion board posting and identify the 6 places where the use of language is of concern. Just click on your choices (some may be two-word phrases, others are single words) and see how you did.



An interactive H5P element has been excluded from this version of the text. You can view it online here:

<https://ohiostate.pressbooks.pub/swk5805coursebook/?p=46#h5p-1>

Now, think about how you would suggest rephrasing each of the six problems. Here is one possible solution—many options exist! The point here is to practice the new skills related to the language that we use. Hopefully, you can better edit your own work before posting in our class discussions in the future.

I think that **persons experiencing addiction** should be able to benefit from treatment for pain, but health care professionals are worried about **providing pain medications when there is a question about the actual need**. It is kind of the same thing as **giving alcohol to someone with an alcohol use disorder** to make them feel better. **People who misuse substances or have an addiction** may believe their pain is worse than they can tolerate, but there may be alternative ways to effectively address pain that doctors and nurses can offer. Treating **a person's pain** should be done with caution **when there is a history of experiencing a substance use disorder**, but it should also be done with respect.

# Ch. 4: Summary

In the readings for Module 1 you read about:

- What “psychoactive substance” means Epidemiological trends in the United States related to substance use and misuse—patterns of use of different types of substances, as well as patterns of use by three different age groups, men versus women, and by racial/ethnic group.
- Major historical and current trends in policy related to substance use and substance use disorders.
- How to monitor the use of language about substance misuse.

In addition, you were introduced to some of the common acronyms used in the field: AUD, SUD, NIDA, NIAAA, and WHO, for example. You are now well prepared to review the list of key terms introduced in these readings.

# Module 1: Key Terms

**binge drinking:** In the NSDUH surveys, this is defined as five or more drinks on the same occasion for men, and four or more for women. The NIAAA definition is a pattern of drinking alcohol that brings a person's blood alcohol concentration (BAC) to or above the 0.08-gram percent (the .08 legal limit for driving). Risky single occasion drinking (RSOD) is another term for describing binge drinking. (Discussed in greater detail in our course module about alcohol).

**blood alcohol concentration:** defined in terms of grams (weight) of alcohol per 100 milliliters of blood, for example 0.08 means 80 milligrams (.08 grams) per 100 milliliters (100 ml=1 deciliter, dL) blood, and can be estimated in breath or urine tests; discussed in greater detail in our course Module 8.

**decriminalization:** the act of repealing, removing, or reducing legal restrictions or criminal penalties associated with a previously illegal act.

**harmful use of alcohol:** the World Health Organization (WHO) definition involves consuming alcohol in a manner *“that causes detrimental health and social consequences for the drinker, the people around the drinker and society at large, as well as the patterns of drinking that are associated with increased risk for adverse health outcomes”* (WHO, p. 2).

**heavy drinking:** Defined in the NSDUH surveys as a pattern of binge drinking on each of five or more days in a month; discussed in greater detail in our alcohol module.

**National Institute on Alcohol Abuse and Alcoholism (NIAAA):** an institute of NIH charged with supporting and conducting research on the impact of alcohol use on human health and well-being and leading the nation's efforts to reduce alcohol-related problems.

**National Institute on Drug Abuse (NIDA):** an institute of NIH



charged with advancing science concerning the causes and consequences of drug use and addiction, as well as applying that knowledge to improve public health.

**National Institutes of Health (NIH):** comprised of 27 institutes and centers, operating through the U.S. Department of Health and Human Services to seek knowledge about the nature and behavior of living systems and application of that knowledge to health enhancement.

**National Institute on Mental Health (NIMH):** an institute of NIH leading research into mental disorders, as well as discovery in the science of brain, behavior, and experience toward the goal of prevention and cure of mental disorders.

**National Survey on Drug Use and Health (NSDUH):** an annual study sponsored by SAMHSA providing national and state-level data concerning mental health status in the United States, and the use of tobacco, alcohol, illicit drugs, and prescription drug misuse.

**Psychoactive (psychotropic) substances:** These are substances that, when consumed, have a significant effect a person's mental processes, mind, mood, and behavior.

**Substance Abuse and Mental Health Services Administration (SAMHSA):** the federal agency in the Department of Health and Human Services (DHHS) charged with leading public health efforts to advance the nation's behavioral health and reduce the impact of substance abuse and mental disorders on communities.

**War on Drugs:** the label applied in 1971 by President Nixon to a campaign of United States government policy actions directed toward controlling trade in illegal drugs.

**World Health Organization (WHO):** part of the United Nation's system, headquartered in Geneva, and leading global efforts to promote health and responses to global health concerns.

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# Ch. 1: Background Facts and Figures

## What Are Psychoactive Substances?

Our course focuses on [psychoactive substances](#). Psychoactive substances are chemicals affecting how the brain functions, and thus have the power to affect a person's mind, mood, and behavior when consumed. The word [psychotropic](#) means the same thing. Many of these substances have important medicinal or other positive purposes when used appropriately. Many also are the subject of concern because of the consequences arising from their misuse and the potential for their use evolving into a substance use disorder. The wide range of psychoactive substances examined in this course include:

- alcohol,
- sedative/hypnotic and central nervous system (CNS) depressants,
- cannabis and other hallucinogenic/dissociative drugs,
- stimulants (including amphetamines, methamphetamine, cocaine, nicotine, and caffeine),
- opioids, inhalants, steroids, commonly misused over-the-counter substances, as well as prescription drug misuse.

## Who (Mis)Uses Psychoactive Substances?

If you are wondering just how common substance use, substance misuse, and substance use disorders are, you will soon find this to

be a more complex question that at first it might appear. The answer varies by:

- type of substance
- age group
- gender
- geographic location
- ethnicity, and
- co-occurring problems.

Based on the popular media, you might have the impression that just about everyone is misusing drugs (except maybe you and a few people in your personal network); this just isn't so. The science of epidemiology can help pinpoint what actually is going on in terms of trends and patterns related to substance use and misuse, as well as the experience of substance use disorders and other negative consequences.



**Before you read on, take a moment to jot down your “best guess” answers to the following questions:**

- *What do you think are the 3 most commonly used substances?*

- *What percentage of individuals do you think use each of the substances you identified?*
- *What do you believe is the distribution of men versus women using these substances?*
- *What age group do you believe is the greatest user of the substances you identified?*
- *What do you believe is the racial/ethnic group most and least likely to use the substances you identified?*
- *How do you think the U.S. compares to other nations in use of the substances you identified?*
- *What percentage of the population do you think has a diagnosable substance use disorder?*
- *What percentage of individuals experiencing substance use disorder do you believe receive treatment for the problem?*

**Epidemiological Studies.** Several large-scale epidemiological studies are routinely relied on to answer questions concerning prevalence and incidence rates and trends in the United States and around the world, as well as other social indicators. These include:



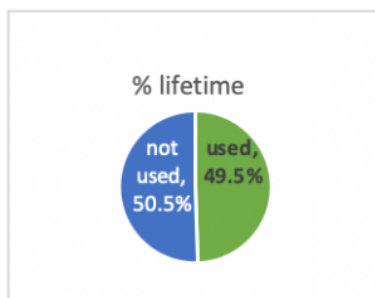
- the National Survey on Drug Use and Health (NSDUH) with regular reports from the **Substance Abuse and Mental Health Services Administration (SAMHSA)**;
- the National Epidemiological Survey on Alcohol and Related Conditions (NESARC) with three waves of data (Wave I from 2001-2002, Wave II from 2004-2005, Wave III from 2012-2103);
- the annual Monitoring the Future Study of 8th, 10th, and 12thgrade students in the U.S., which also has some longitudinal follow-up data for some participants into early

adulthood;

- the United Nations Office on Drugs and Crime (UNODC) which compiles data from global sources, including the **World Health Organization (WHO)** into an annual World Drug Report.

## Scope of the Issue.

The World Drug Report 2019 (WHO, 2019) reported that not only are the adverse health consequences of drug use more severe and widespread than previously believed, the severity of the situation is increasing. Reportedly, an estimated 35 million individuals



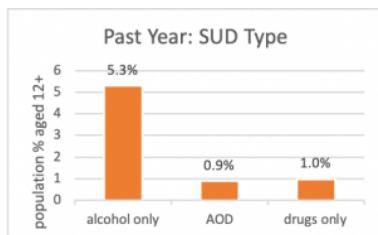
globally experienced drug use disorders requiring treatment services and an estimated 271 million (5.5% of the world's population) used drugs outside of medical recommendation during 2017. The report also concluded that only 1 in 7 persons in need of treatment for a drug use disorder receives it. The report raises alarm over the 25% increased production of cocaine compared to the previous year, reaching an all-time high. It also calls out the 47,000 opioid overdose deaths reported in the United States during 2017 (up 13% from 2016) and 4,000 in Canada (up 33% from the previous year). An opioid crisis is also arising in West, Central, and North Africa although the specific opioid drugs involved may differ in various parts of the world.

In the U.S., based on the 2017 NSDUH data, an estimated 134.7 million individuals over the age of 12 (49.5% of population) used illicit drugs (including prescription drugs outside of medically prescribed use) during their lifetimes (SAMHSA, 2018). The study considered a person to be currently using substances if use was reported during



the past month; almost 30.5 million (11.2% of population) were estimated to have used illicit drugs in the past month.

While substance use/misuse are important to track, it is also important to know about individuals experiencing a substance use disorder. From that 2017 survey, over 19.7 million individuals aged 12 or



older (7.2% of population) were estimated to experience a substance use disorder (SUD) involving alcohol and/or and illicit drug use during the past year, as measured in 2017; the vast majority of SUDs involved alcohol alone (5.3%) or in combination (0.9%) with illicit drugs, leaving 1% with a drugs only SUD.

Just over 4 million individuals (1.5% of population) received substance use treatment during that time (SAMHSA, 2018). You might wonder why such a discrepancy existed. Over 1 million individuals were estimated to have felt a need for treatment related to their substance use, divided about evenly between those who did versus did not make an effort to get treatment; over 17 million individuals needing treatment based on SUD criteria did not feel a need for treatment.

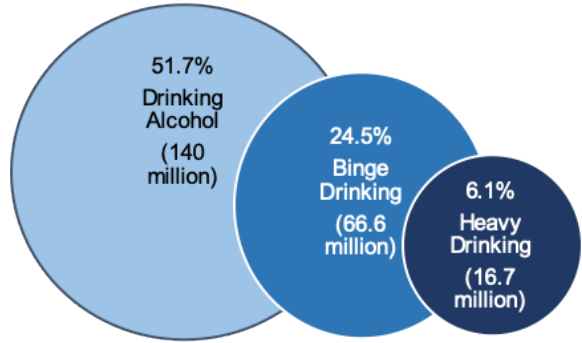
The NSDUH data can be analyzed in somewhat more nuanced ways. Let's look at the differences between types of substances used and who engages in AOD use/misuse in the U.S. (by age, gender, and racial/ethnic group). Despite the emphasis on certain substances in the news and stereotypes stemming from various sources, the answers to these kinds of "what" and "who" questions are informative, and some answers may surprise you.

## Type of Substance: Alcohol.

What is the most commonly used substance? In the U.S., alcohol.

Not illicit drugs like marijuana and heroin and not prescription drug misuse. According to estimates based on the NSDUH data for 2017, more than 140 million individuals (51.7% of population) over the age of 12 used alcohol during the past month—meaning they are considered to be currently using alcohol (SAMHSA, 2018). Not all alcohol consumption occurred in risky or problematic amounts, however—the vast majority of individuals who consume alcohol do so in moderation. This is in contrast to individuals engaging in [binge drinking](#) or [heavy drinking](#) patterns (see Figure 1.1). Binge alcohol use in the past month, defined as “five or more drinks (for males) or four or more drinks (for females) on the same occasion (i.e., at the same time or within a couple of hours of each other),” was attributed to 66.6 million individuals (24.5% of population); heavy alcohol use, defined as “binge drinking on the same occasion on each of 5 or more days in the past 30 days; all heavy alcohol users are also binge alcohol users”, to 16.7 million (6.1% of population) [SAMHSA, 2018, Tables 2.20A & 2.20B].

Figure 1.1. Percent reporting past-month drinking alcohol, binge drinking, and heavy drinking (derived from SAMHSA, 2018 report for persons aged 12+)



You may wonder about the difference in amounts for men and women presented in the binge drinking definition. According to the **National Institute on Alcohol Abuse and Alcoholism (NIAAA)**, drinking in a manner that raises a person's **blood alcohol concentration (BAC)** to 0.08g/dL or higher is binge drinking. Amounts and rates of alcohol consumption will be factors in this outcome, along with aspects of individual differences in constitution. In general, for women this means about four drinks in about two hours or five drinks in two hours for men. This pattern sometimes is referred to as risky single occasion drinking (RSOD).

The World Health Organization (WHO, 2014) identified alcohol as a significant factor in the global burden of disease (and death). The **harmful use of alcohol** was defined as: *“drinking that causes detrimental health and social consequences for the drinker, the people around the drinker and society at large, as well as the patterns of drinking that are associated with increased risk for adverse health outcomes”* (p. 2). Thus, WHO set a goal for a 10% reduction in harmful use of alcohol by the year 2025 around the world because of the many health consequences (and 3 million deaths per year) attributed to this behavior (see Figure 1.2). Reducing and preventing alcohol-related harm is also one of the American Academy of Social Work and Social Welfare (AASWSW) Grand Challenges for Social Work under the umbrella goal called “Close the Health Gap” (Begun, Clapp, and the Alcohol Misuse Grand Challenge Collective, 2015).

Figure 1.2. Infographic produced by WHO (retrieved from [https://www.who.int/images/default-source/departments/substances-abuse/alcohol/infographics/alcohol-3-million-death-every-year.png?sfvrsn=8062967\\_2](https://www.who.int/images/default-source/departments/substances-abuse/alcohol/infographics/alcohol-3-million-death-every-year.png?sfvrsn=8062967_2))

# Alcohol and health



**3 million deaths every year**  
6 deaths every minute from harmful use of alcohol

women 1/4  
men 3/4

## Harmful use of alcohol causes

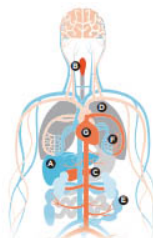
100% of alcohol use disorders

18% of suicides

18% of interpersonal violence

27% of traffic injuries

13% of epilepsy



48% of liver cirrhosis

26% of mouth cancers

26% of pancreatitis

20% of tuberculosis

11% of colorectal cancer

5% of breast cancer

7% of hypertensive heart disease

## Reduce harmful use of alcohol

Best buy interventions

✓ Regulate alcohol distribution

✓ Restrict or ban advertising

✓ Increase prices

More key interventions



Prevent and treat alcohol use disorders



Raise awareness of alcohol-attributable health burden



Support community action to prevent and reduce the harmful use of alcohol



Provide consumer information on alcohol containers



Regulate informally produced alcohol



Implement drink-driving policies



Develop surveillance systems for alcohol consumption, health consequences and policy



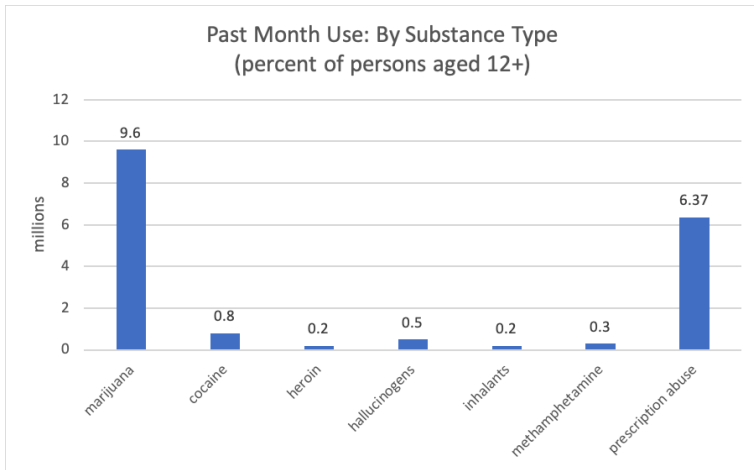
10% reduction in the harmful use of alcohol by 2025

## Type of Substance: Other Drugs.

We looked at data concerning alcohol in the AOD acronym, now let's

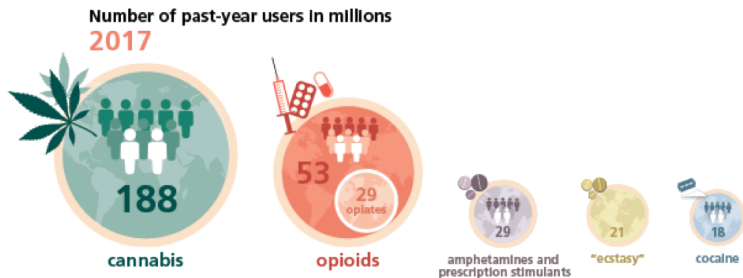
take a look at those other drugs. Over 30 million individuals (11.2% of the population) were estimated to have used illicit drugs in the past month based on 2017 NSDUH data (SAMHSA, 2018). The type of illicit drug most often used in the U.S., by far, was marijuana—an estimated almost 26 million individuals over the age of 12. The next most common was the misuse of prescription psychoactive drugs, including pain relievers, stimulants, tranquilizers, and sedatives, in that order of frequency (an estimated almost 6 million individuals combined). Less commonly used were cocaine, hallucinogens, heroin, and methamphetamine (see Figure 1.3). Note that the percentages in Figure 1.3 add up to more than the 11.2% of the population using illicit drugs; this is because some individuals used more than one type. Comparing these percentages with what you just learned about alcohol, were you surprised that so much greater emphasis seems to be placed on drug problems than alcohol? You may find it curious that the FY 2016 [National Institute on Drug Abuse \(NIDA\)](#) budget for research and development was almost double the National Institute on Alcohol Abuse and Alcoholism (NIAAA) research and development budget; NIDA and NIAAA are two parts of the U.S. [National Institutes of Health \(NIH\)](#) (see <https://officeofbudget.od.nih.gov/pdfs/FY18/Drug%20Control%20Programs.pdf>).

Figure 1.3. Past month use of various substances (SAMHSA, 2018)



The World Drug Report 2019 contains a figure demonstrating the estimated global prevalence of drug use comparing cannabis, opioids, amphetamines/prescription stimulants, ecstasy, and cocaine. As in the U.S., cannabis is the drug most commonly used around the world (see Figure 1.4). We will be studying each of these types of substances in Part 2 of our course.

Figure 1.4. World Drug Report 2019 (UNODC, 2019) past-year use of five types of drug in 2017.



We looked at alcohol (a legal substance) and illicit drugs, but what about the other legal substance so commonly used in the U.S. and around the world—nicotine? Among persons aged 12 and older, based on the 2017 NSDUH data, an estimated 170.5 million individuals have used tobacco products (not including e-cigarettes/vaping) during their lifetimes—62.7% of the population. Current use was attributed to over 61 million, or 22.4% of the population (SAMHSA, 2018). The vast majority of use involved cigarettes; smokeless tobacco, cigars, and pipe tobacco were less common. According to the WHO (<https://www.who.int/news-room/fact-sheets/detail/tobacco>) tobacco kills more than 8 million people annually, 1.2 million of whom were non-smokers exposed to second-hand smoke; 1.1 billion individuals smoke tobacco worldwide; and, tobacco kills up to half its users.

The report also refers to other victims of tobacco: children from poor families employed in tobacco farming absorb nicotine through their skin from handling tobacco leaves and are vulnerable to “green tobacco sickness” as a



result. In addition to concluding that tobacco represents a significant U.S. and global public health concern, this information indicates that deciding to smoke is not just an individual choice matter—it has implications for others nearby (second-hand smoke exposure) and for others of whom we may be unaware (involved in production and distribution). These issues are not unique to tobacco, by the way—it is a relevant social justice consideration regarding all types of drugs.

## Type of Substance by Age Group.

Based on the 2017 NSDUH data (SAMHSA, 2018), patterns of alcohol and illicit drug use can be estimated for each of the following age groups: 12 to 17-year-olds (youth), 18 to 25-year-olds (emerging adults), 26 to 64-year-olds (adults), and individuals aged 65 and older.

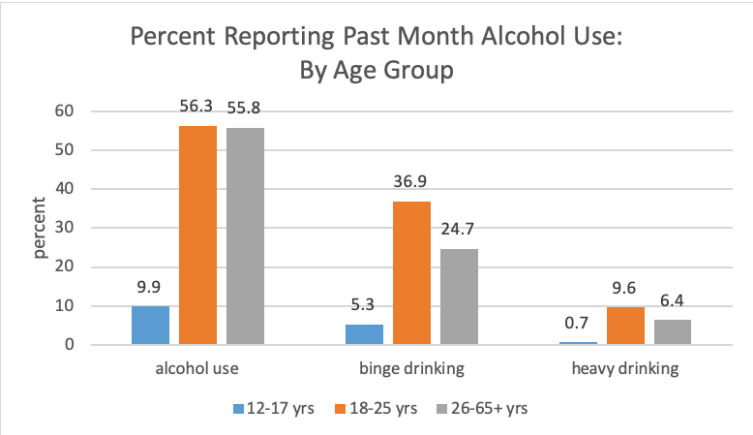
Technically, alcohol is an illicit substance for underage youths (those aged 12-17 and many in the survey's 18-25 group). Figure 1.5 shows the percent reporting past month use of alcohol, binge drinking, and heavy drinking by age group in the 2017 NSDUH data. These numbers all peaked for our emerging adult group.



While the alcohol use percentage remained relatively steady into adulthood (over age 25), binge and heavy drinking percentages declined. Again, these data indicate that the majority of adults who drink generally do so in moderation. However, more than half of individuals in emerging adulthood and adolescence who used alcohol in the past month engaged in binge drinking (considered a risky pattern); slightly less than half of adults did so. About 18% of emerging adults engaged in the riskiest pattern, heavy drinking, compared to about 11% of adults and about 8% of adolescents (SAMHSA, 2018).

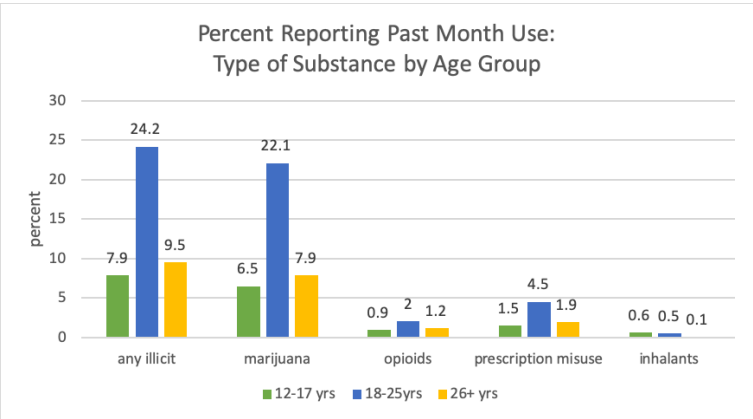
Figure 1.5. Patterns of past month alcohol use by age group.





Past month use of most illicit drugs also was highest among the emerging adulthood group (aged 18-25 years) and declined in percentage after age 26 (see Figure 1.6). The exception was inhalant misuse: this was most common among adolescents, dropped a bit in emerging adulthood, and continued to drop in adulthood. Again, marijuana was the most illicit substance most frequently used; opioids included heroin use and pain reliever misuse.

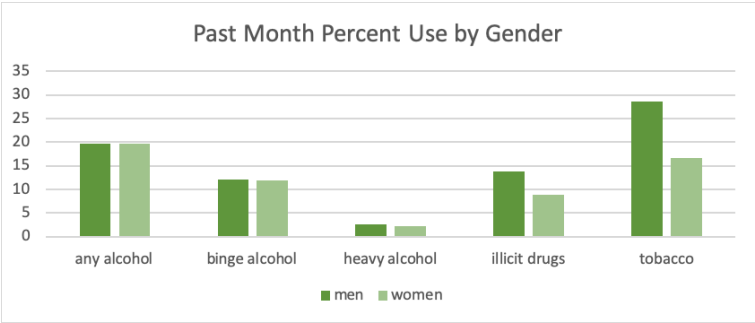
Figure 1.6. Patterns of past month illicit drug use by age group.



Finally, let's consider tobacco use by age group. Adolescents' (aged 12-17 years) past month use of tobacco products was less than the other groups: 4.9% compared to 29.1% of emerging adults (aged 18-25) and 23.4% of adults over the age of 25 (SAMHSA, 2018).

Substance Use by Gender: Illicit drug and tobacco use were more common among men than women aged 12 and older; alcohol use patterns were very similar among men and women (see Figure 1.7).

Figure 1.7. Past month alcohol, illicit drug, and tobacco use by gender for persons aged 12+ years



Of considerable concern is evidence that, despite concentrated public health efforts, about 10% of women worldwide consume alcohol while pregnant (Popova, Rehm, & Shield, in press). Later, in our “Focus on Alcohol Module,” you will learn more about why this so concerning; for now, it is important just to recognize that prenatal alcohol exposure (PAE) potentially has lifelong effects on a person’s health, mental health, and abilities.

## Type of Substance by Race/Ethnicity.

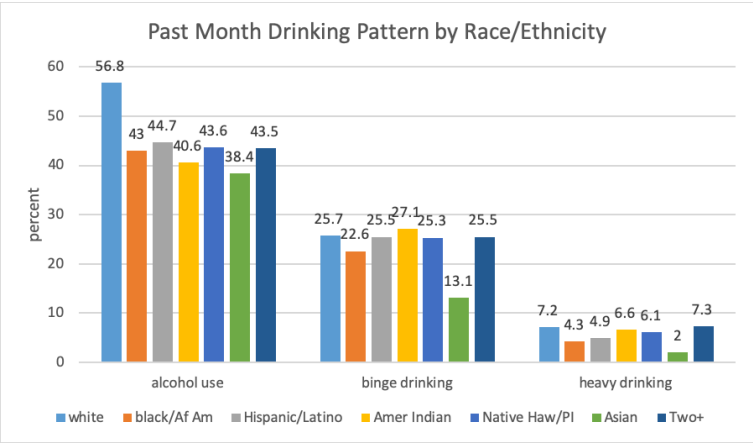
The seven U.S. racial/ethnic groups for whom information is reported in the 2017 NSDUH survey (SAMHSA, 2018 include: white;

black/African American; Hispanic/Latino; Asian; American Indian/Native Alaskan; Native Hawaiian/Other Pacific Islander; and, those of two or more races.

*Alcohol.* The group most likely to report past month use of alcohol was comprised of individuals who identified themselves as white (56.%) and the lowest rates were reported by the group identifying as Asian (38.4%) groups (see Figure 1.8). Looking at these statistics another way, the highest rates of alcohol abstinence (individuals not drinking) in the past month appeared among the Native Hawaiian/Other Pacific Islander, American Indian, and African American groups.

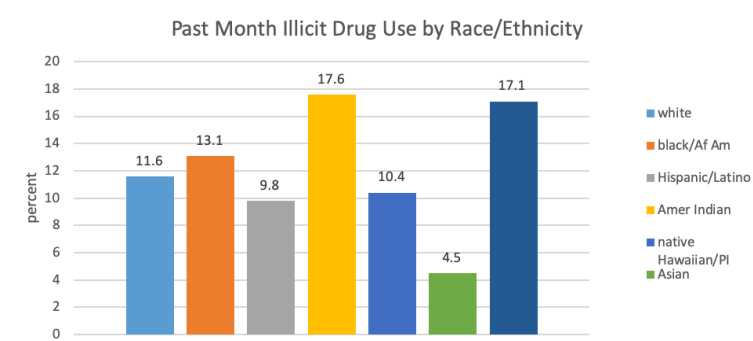
The picture differs somewhat when looking at binge and heavy drinking patterns. Individuals identifying as white remained in the top range of those who engaged in binge drinking during the past month (25.3-27.1) which also included individuals identifying as Hispanic/Latino, American Indian, Native Hawaiian/Pacific Islander, or belonging to two or more groups. Individuals who self-identified as Asian had the lowest rate of binge drinking (13.1%), with a rate in between these extremes reported in the African American group (22.6%). Heavy drinking was at the highest rate among white individuals and those belonging to two or more groups (7.2-7.3%), lowest among the Asian group (2%) and somewhere between 4.3-6.6% for the other groups.

Figure 1.8. Past month drinking patterns reported by race/ethnicity



*Other drugs.* The groups reporting the highest rate of past month illicit drug use were those who self-identified as American Indian and as belonging to two or more races (17.6% and 17.1%, respectively; see Figure 1.9). The lowest rate was reported among those identifying as Asian (4.5%) with the other groups falling in between (9.8% to 13.1%). As you can see, this picture differs somewhat from the story presented by the alcohol data.

Figure 1.9. Past month illicit substance use by race/ethnicity



**Tobacco.** Finally, consider the study data concerning the use of tobacco products (not including e-cigarettes/vaping) across these different groups (see Figure 1.10). The rate was highest among individuals identifying as American Indian (41.5%) and lowest among those identifying as Asian (below 10%) and Hispanic/Latino (16.7%). Still, around a quarter of individuals identifying as white, black/African American, Native Hawaiian/Pacific Islander, and belonging to two or more groups reported using tobacco products during the past month (current use).



**Before you read on, take a moment to jot down your “best guess” answers to the following questions:**

**Now that you have read the information above, take a moment to compare what you learned as answers to the questions below with your pre-reading answers:**

- *What are the 3 most commonly used substances?*
- *What percentage of individuals aged 12 and older use each of those most common substances?*
- *What is the distribution of men versus women using these substances?*
- *What age group is the highest user of these substances?*

- *What are the patterns of substance use among white, black, Hispanic/Latino, and Native American persons?*
- *What percentage of the population has a diagnosable substance use disorder?*
- *What percentage of individuals experiencing substance use disorders receive treatment for the problem?*

Were you surprised by any of the answers? What factors, information, or experiences prior to this course do you think led you to guess the right or wrong answers?

## PART III

# MODULE 3: BIOLOGICAL MODELS OF SUBSTANCE MISUSE, PHARMACOKINETICS & PSYCHOPHARMACOLOGY PRINCIPLES

Module 3 readings introduce a host of biological processes related to substance use, substance misuse, and substance use disorders (SUDs). Biological influences include genetics, neurobiology, and human development (which is a biopsychosocial process). This module begins with an examination of the genetic evidence concerning substance use, misuse, and SUD. Next, we will explore what might be going on in the brain with exposure to alcohol and other drugs (AOD)—basics about neurobiology. In this section, we look at basic information concerning neuroanatomy (parts/areas of the brain) and neurochemistry (neurons and neurotransmitters). Understanding these basic biological processes helps explain the brain-behavior relationship—how what goes on in the human brain relates to the human experience and human behavior. This content reflects a vast difference from the early (1930s) (mis)conception of addiction as resulting from a moral failing or weak willpower (NIDA, 2018). Quoting the director of the National Institute on Drug Abuse (NIDA, 2014), Dr. Nora Volkow:

*“Drug addiction is a brain disease that can be treated.”*

While biopsychosocial processes include additional factors, it is critically important to understand what is happening at the biological level if we are to understand and effectively intervene

around substance use, misuse, and SUD. Substance misuse causes significant and persistent changes in the brain that relate to the experience of addiction (SUD), changes that may persist for long periods of time after substance use stops. Recovery from SUD does not necessarily return the brain to its original pre-SUD state, rather it again changes as it establishes a new state of “normal” functioning—some substance-induced changes are not reversible.

Relevant to discussing the biological basis of substance misuse are elements of human development—exposure to substances at critical developmental periods has a different impact than exposure at other times. In order to understand the biology of substance use, it is helpful to understand certain principles of pharmacokinetics and psychopharmacology—how drugs are processed/metabolized in the body, the biology underlying tolerance and withdrawal, the biology underlying drug actions (agonist, antagonist, and synergism), and how this knowledge might inform pharmacotherapy—the use of medication to help treat substance use disorders.

Portions of our Module 3 content were informed by (and informed) these previous works (see reference list for details): Bares and Chartier (in press), Begun and Brown (2014), and NIDA (2018), as well as a lecture by Dr. David Sackx called Alcohol and the Brain (no longer available on Youtube).

## Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Explain evidence concerning the genetic basis of substance misuse and substance use disorders;
- Describe the roles played by different brain regions (neuroanatomy) in substance misuse and substance use



disorder;

- Describe the roles played by neurotransmitters (neurochemistry/neurophysiology) in substance use, misuse, and use disorders;
- Explain why age at substance use initiation matters in determining substance use disorder outcomes;
- Identify the role of homeostasis processes in acquired tolerance and withdrawal;
- Describe basic principles of pharmacokinetics and psychopharmacology (drug half-life, synergism, agonists, and antagonists) and how this might relate to medication for assisting in treatment of substance use disorder (pharmacotherapy).



# Ch. 1: Genetic Influences

A large body of evidence indicates that substance use disorder (SUD) can follow a familial pattern—but does not necessarily do so. Individuals with genetically close relatives (parents or adult siblings) experiencing a substance use disorder involving opioids, cocaine, cannabis or alcohol have up to an 8 times higher risk of developing a substance use disorder themselves (Merikangas, et al, 1998), and having a biological parent with alcohol use disorder increases the risk of developing problems with alcohol by about 4 times, even if raised by parents without a history of alcohol use disorder (Russell, 1990). Genetic studies paint a picture indicating that genetics are important in both the appearance of and resistance to substance use disorders. However, the single most important message in this module is that genetics alone do not determine a person's destiny: genetic makeup interacts with the environment and a person's lifetime of experiences to determine whether a substance use disorder emerges. It is critically important to note that the majority of individuals with genetic family histories of substance use disorders never develop the problem themselves.

Another fact that has emerged from decades of research is that there is no one specific “addiction gene” that applies to all of the different types of substances. Some of the genes involved are very specific to certain substances—what may “pull” for



an alcohol use disorder may not be “pulling” for a problem with cocaine, for example. Some genes are not specific to substance use disorders per se, but to a class of problems that have substance misuse as an element—for example, depression. The more we learn

about specific combinations of genes that might be involved, exciting new biological tools for treating or even preventing addiction may emerge, including medications and perhaps even immunizations someday. For a little basic background (possibly review) in understanding genetics, see the keywords list for [DNA](#), [alleles](#), [genes](#), [chromosomes](#), [genome](#), [genotype](#), [heritability](#), and [phenotype](#).

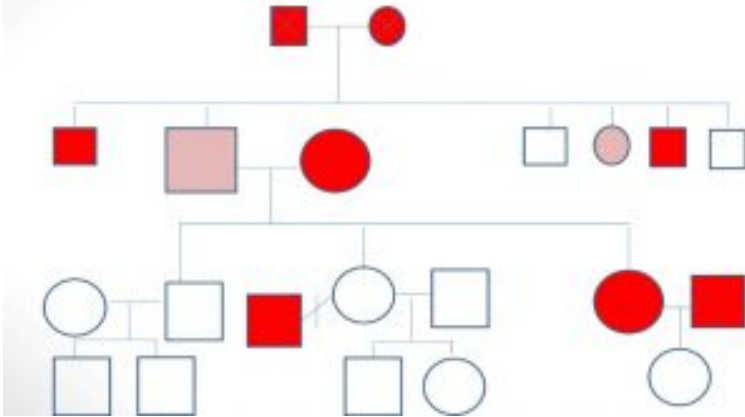
Four general lines of research contribute to our understanding of the role played by genetics in substance misuse and substance use disorder (SUD): family pedigree, twin, adoption, and genome studies.

**Family pedigree studies.** Early genetic influence research relied on tracing the patterns with which a particular phenotype appears in multiple generations of a family—alcohol misuse and alcohol use disorder (AUD) is an example. These familial patterns become apparent when a pedigree chart is created (in social work practice, a family “genogram” is sometimes used in assessment; see Hartman, 1995). The observed pedigree patterns generally supported investigators’ hypothesis that the development of alcoholism has a genetic component—it is not entirely driven or dictated by genetics but is influenced by genetics. The more genetically close (proximal) in relationship, the greater the influence. For example, with alcohol use disorders, the influence of parents is stronger than the influence of aunts/uncles.

Figure 3-1 depicts a family’s pedigree for alcohol use disorder (dark red) and adults’ alcohol misuse (light red) for 3 generations—the youngest generation are still too young to know about. The common notation for a pedigree/genogram is that squares represent males, circles females, triangles unknown sex; lines between shapes represent couple relationships; lines above shapes represent offspring and sibling connections. An “X” through a symbol means the person is deceased and a crossed relationship line means the couple is no longer together.

**Figure 3-1. Sample family pedigree (genogram) tracing alcohol use disorder.**

## Family Pedigree (Genogram)



**Twin studies.** Another source of evidence supporting the theory that alcoholism has a genetic basis comes from twin studies. There exist at least two types of twins, genetically speaking. Identical twins originate from the same single egg/sperm pair ([monozygotic twins](#)), thus they share the same genome. Fraternal twins, on the other hand, originate from two different egg/sperm pairings ([dizygotic twins](#)), thus they share a random amount of genetic coding, just as any sibling pairs might—on average, 50% is shared, but it could be anywhere along the range from almost 0% to almost 100%. The logic behind twin studies is to look at the degree of phenotypic similarity on some trait/condition, called “[concordance](#),” between identical versus fraternal twins—if the degree of

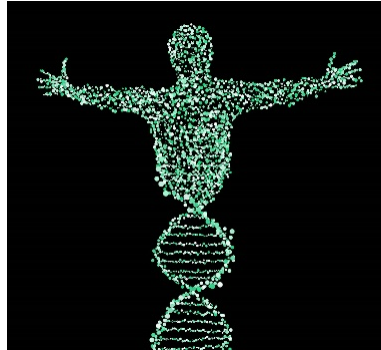


concordance is considerably greater among identical twins, this constitutes strong evidence for a genetic influence. In other words, it has moderate or high heritability. When the phenotypic outcome for identical twins is more than twice as similar as the outcome for fraternal twins, that trait is considered to be under a high degree of genetic control (Bares & Chartier, *in press*). Evidence for a genetic influence on alcohol use disorder is strong, but again—there also is sufficient lack of concordance between identical twins to show that it is not entirely driven by genetics.

**Adoption studies.** Adoption studies represent a third leg in the evidence base supporting a genetic influence on alcohol use disorders. These studies are based on comparing the phenotypic outcome of children raised by their biological parents with children raised by adoptive parents when the biological parent(s) exhibit the phenotype of interest. In this case, children whose biological parent(s) experience an alcohol use disorder who are raised by their biological parents or raised by adoptive parents who do not experience alcohol use disorder. Evidence suggests that among children whose biological father experienced an alcohol use disorder, being raised in an adoptive family was moderately but not entirely protective. In other words, there remains a considerable genetic influence (about 50-60%)—and, the child's environment can confer a great degree of protection (Foroud, Edenberg, & Crabbe, 2010).



**Genome studies.** More recent lines of research go beyond answering the question “do genetics matter” to more specificity about “how genetics matters.” The human genome is a person’s complete set of DNA, represented in virtually every cell of the body. The Human Genome Project, completed in



2003, resulted in a generic “map” of the approximately 20,000-25,000 genes in the human genome (see the national Human Genome Research Institute’s [genome.gov/about-genomics/fact-sheets/A-Brief-Guide-to-Genomics](http://genome.gov/about-genomics/fact-sheets/A-Brief-Guide-to-Genomics)). This knowledge contributes greatly to understanding complex health problems (like substance use disorders) resulting from multiple genetic factors acting together and with the environment. Genome-wise association studies (GWAS) approached the study of substance misuse and SUD (and other phenotypic outcomes) in a unique manner: searching for common variants in allele frequency across the entire genome and then determining what phenotypic differences were associated with those variants (Bares & Chartier, in press). The GWAS approach is credited with identifying a genetic basis for phenotypes including heavy versus light amount of cigarette smoking or alcohol consumption, and developing nicotine or alcohol use disorder (Hancock, Markunas, Bierut, & Johnson, 2018).

Additionally, the Collaborative Studies on Genetics of Alcoholism (COGA) has established a database of information from over 10,000 individuals across multiple sites and over many years. The variables included measures of clinical, neuropsychological, electrophysiological, biochemical, and genetic factors, as well as individual and family histories of drinking behavior, from four groups of individuals (see <http://pubs.niaaa.nih.gov/publications/arh26-3/214-218.htm>):

- those meeting criteria for alcohol dependence (DSM-IV-TR criteria);
- those “at-risk” of alcohol dependence by virtue of their low level of response to alcohol—higher baseline tolerance, needing to consume greater amounts of alcohol than others in order to feel the effects is recognized as a vulnerability factor for developing alcohol use disorder;
- those meeting criteria for depression with or without alcohol dependence (two subgroups);
- “unaffected alcohol users” from families with one or more members experiencing alcohol dependence.

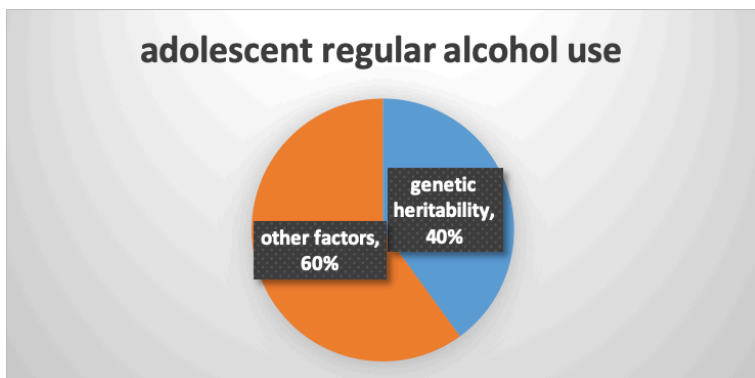
## What Is Known About The Genetics of Substance Misuse

What has been learned from combining evidence from these four different types of studies includes the following:

**Genetics plays a significant role.** As described in terms of the family pedigree, twin, and adoption studies, there is clearly a genetic influence on the development of alcohol use disorders. Less is known about other substances, however, there is convincing evidence from these and genomic studies that the probability of substance use becoming a substance use disorder (SUD) is influenced genetically (heritable) for many different substances. Not only is the emergence of SUD partially directed by genetics, but there appears to be a genetic contribution to the initiation and regular use of at least some substances, as well. For example, initiating tobacco use during adolescence was anywhere between 35%-80% heritable across different studies, regular tobacco use was between 40% to 50% heritable, and regular alcohol use was about 40% heritable (Bares & Chartier, in press). The evidence also demonstrates that it is not entirely driven by



genetics—environmental factors play a significant role, as well (Bares & Chartier, *in press*).



**Multiple are genes involved.** Evidence points to multiple genes contributing to substance use disorder ([\*polygenic\*](#)), even to a single type of substance use disorder



(e.g., alcohol use disorder). Early genetic studies attempted to determine which specific gene or genes were candidates for playing a significant role in substance misuse behavior or SUD based primarily on their control of important, relevant biological processes; candidate gene studies generally showed inconsistent results, however (Bares & Chartier, *in press*). More recent approaches to understanding polygenic phenomena involve aggregating the many small effects each gene might contribute, resulting in a weighted total genetic effect (polygenic score, or PGS) taking into account the vulnerability and protective genes a person might have (Bares & Chartier, *in press*). In other words, we cannot point to any one gene as the “cause” of even a single type of SUD, much less SUDs in general.

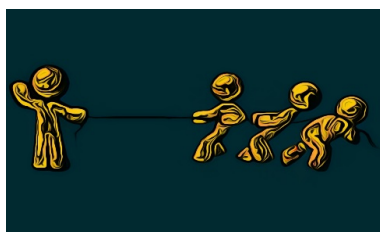
### **Some genes may provide protection.**

At least one gene locus appears to provide protection from alcohol dependence, in contrast to gene sites contributing to vulnerability (Reich et al., 1998). Genes involved in controlling the processes of alcohol metabolism in the human body demonstrate a potential for preventing alcohol use from becoming an alcohol use disorder as exposure to alcohol creates a toxic, highly unpleasant physiological response (Edenberg, Gelernter & Agrawal, 2019). The protective allele (called ALDH2\*2) is most common among individuals of Asian descent. Protective genes may exist for other substances, as well.



**Severity is determined by specific chromosomal regions.**

Genetic influence is not simply occurring at the level of specific chromosome sites, but also in chromosomal regions (areas



where multiple chromosomal sites cluster) and in various polygenetic combinations (multiple genes interacting). What this means is that a person may have various genetic forces pushing for and against developing substance misuse or SUD problems in a kind of genetic tug-of-war. As a result, we see a wide range of phenotypic expression in the population as a whole—the problem is heterogeneous, not “one size fits all.”

**Common versus specific origins.**

Analysis of a vast body of science provided answers to the question of whether SUD involving different types of substances has a shared, common genetic origin or whether each type of substance has its own unique genetic influences (Li et al., 2011). The answer is not simple:



some genomic areas appear to be shared across different types, while other areas are substance-specific (Begun & Brown, 2014). There does appear to be some shared commonality in genetic vulnerability to nicotine, alcohol, and cannabis dependence, at least among men. The underlying common genetic factors, however, fail to explain the high degree of variability in phenotypic expression (Palmer et al., 2012). There exist specific genetic factors, as well, also operating at the same time, including specificity for alcohol, tobacco, marijuana, and cocaine (Palmer et al., 2012). One common underlying genetic factor may be the presence of genetics linked to depression—for some individuals, depression and SUD have common genetic influences, but this is not true for everyone with either/both experiences.

**SUD heritability was stronger among men.**

While heritability of alcohol use disorder was observed for both men and women, the case appeared to be stronger among men. In other words, environmental factors explained a greater proportion of alcohol use disorder among women (Kendler et al., 1992; Jang, Livesly, & Vernon, 1997). However, this gender-based differentiation is less noticeable in recent cohorts than historically, at least for alcohol or nicotine dependence (Palmer et al., 2012).



## Summary

In answer to the “Is it genetic?” question about substance use disorder, the evidence from multiple sources indicates “sort of.” The situation is complex. Some aspects of substance use, substance misuse, and substance use disorder are influenced by genetic forces, but there is a great deal about each of these behaviors/experiences that is influenced by other than genetic forces, too. Furthermore, the genetic forces do not all push in the same

direction or to the same extent—some forces push for and other against the problems emerging. We also know that SUD is not a single phenomenon—susceptibility differs for different substances. For example, a propensity toward alcohol use disorder may or may not align with propensity for nicotine or cocaine use disorder. And, the genetic forces related to certain co-occurring problems may also relate to the propensity for developing a specific type of SUD.



## Your Family Pedigree

Sketch a diagram of your family's genogram for at least three or four generations, as much as you know about. This could be your biological or adoptive family "tree." Use color to highlight everyone you know/suspect had a certain characteristic of interest to you (e.g., nicotine dependence, alcohol use disorder, diabetes, heart disease/stroke) during their lifetime. Is there any pattern to what you see? What are the implications for your own vulnerability? What are the implications for your own resilience? Do you see how genetics are informative but not completely predictive of what happens?

# Ch. 2: Neurobiology and Substance Use

The biological realm of substance use, substance misuse, substance use disorder includes neuroanatomy, neurophysiology, and neurochemistry. Neurobiology investigators are developing increasingly complex, detailed, functional maps of the various regions of the brain involved in substance use, misuse, and SUD. These maps show how the brain's powerful pain, pleasure, reward, and memory systems interact in the process of substance use becoming a substance use disorder—and how psychological learning principles operate at a neurobiological level. This knowledge also helps us understand how difficult it can be to recover from SUD/addiction and why the age/stage of development when substance use is initiated matters in the outcomes.



Learning about the neurochemistry actions of specific substances in neurophysiology also helps us understand the actions of different substances on the brain-behavior link. Here we will look at neurotransmitters



and their role in the experience of substance use/misuse. This knowledge helps investigators develop intervention strategies for treatment, relapse prevention, and even preventing the development of substance use disorders. These biologically based strategies include medications and the use of mindfulness meditation and neurofeedback approaches.

### **Neuroanatomy and Function**

The structure and organization of the central nervous system (CNS) has been studied for a very long time. Current technologies such as functional magnetic resonance imaging (fMRI) help develop our understanding of how different areas of the brain are involved in specific experiences or behaviors, and how exposure to different events or substances might affect specific brain areas and functions. There are certain brain regions identified as having a significant role in the development of SUD. In addition, the brain-behavior link is influenced by and influences the [autonomic nervous system \(ANS\)](#) which controls many bodily functions outside of conscious thought (e.g., heart and breathing rate, blood pressure, and others). Many psychoactive substances not only affect the “mind,” they also affect other organs and systems, including the ANS. When we examine different types of substances, you will see how the health and functioning in other systems is also affected by psychoactive substances.

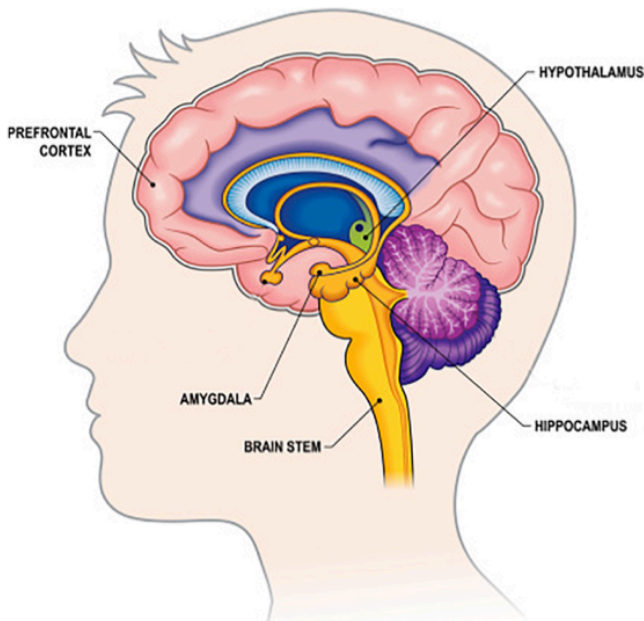
**Limbic system.** The [limbic system](#) helps regulate basic drives, emotions, arousal and attentiveness (Begun & Brown, 2014). As such, it helps coordinate the neurobiological experience of stress and the reward system triggered by exposure to drugs. The [amygdala](#) and [nucleus accumbens](#) are two important components of the limbic

system with regard to substance misuse (Logrip, Zorilla, & Koob, 2012), along with the [hippocampus](#).

*Amygdala.* The amygdala plays a central role in emotional responses to internal and external stimuli—pleasure, fear, anxiety, and anger included. It is central to survival as it manages the “fight or flight” response to perceived threats in the environment, which in turn, is related to the experience of stress. The amygdala is also responsible for the emotional content of our memories—determining not only which experiences related to pain and pleasure become encoded into memory, but also the emotional values attached to the formation of new memories. This area is one target of anti-anxiety medications but is also influenced by the actions of various substances that might be misused.

*Hippocampus.* The hippocampus is involved in memory, as well, particularly memories related to traumatic events and learned responses to environmental cues. This becomes an important factor in the experience of cravings triggered by environmental cues, as well as the relationship between trauma and substance misuse/SUD.

*Nucleus accumbens.* The nucleus accumbens is part of what is called the mesolimbic dopamine system—it is highly involved in positive reinforcement, leading to a person anticipating reward with repetition of the previously positively reinforced behavior. Thus, if a substance increases the release of dopamine in this area, the person comes to anticipate positive reinforcement again with future use. The amount of dopamine increase can far exceed what natural behaviors trigger (eating or sex, for example) and the amount of dopamine directly relates to the degree of pleasure experienced (Volkow et al., 2010). Thus, a person may come to preferentially engage in substance use over naturally rewarding behaviors (like eating or sex).



adapted from Queensland Brain Institute [qbi.uq.edu.au](http://qbi.uq.edu.au)

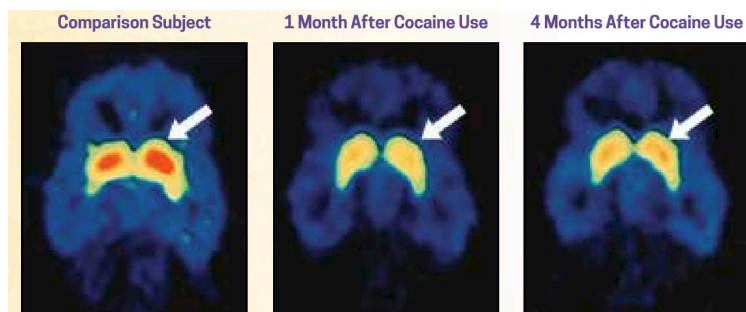
**Prefrontal cortex.** The [prefrontal cortex](#) is linked to the amygdala—they communicate directly. This is a “thinking” part of the brain where functions like cognition, comprehension, concentration, reasoning, planning, and initiating goal-directed behavior takes place (Giancola & Tarter, 1999). The area is responsible for a person’s intentional responses to the experiences the amygdala sends forward. For example, the conscious decision to initially engage in substance use. This part of the brain is also highly susceptible to alteration, even damage, from exposure to many substances, reducing its capacity to mediate responses triggered by the amygdala (Begun & Brown, 2014). As a result, a person might be less able to dampen the amygdala’s push to action, acting more impulsively than thoughtfully/intentionally, especially



in terms of relapse responses. The paradox is that the very area responsible for helping someone control substance misuse is an area impaired by substance misuse (Azmitia, 2001).

## Changes in Brain Function

Changes in how these areas of the brain function following exposure to certain substances, particularly heavy, repeated (chronic) substance misuse, are evident in fMRI (functional magnetic resonance imaging) scans. Additionally, changes remain evident well after the substance use ceases—although the brain does begin to recover and return to more normal appearing functioning. In the following sequence of brain scans, the image on the left is of a person who has not engaged in cocaine use (the “normal” control brain). The other two scans represent a person who has a history of cocaine use disorder 1 month and 4 months after use has ceased. The areas in red represent the density of dopamine receptors in an area of the brain (striatum) responsible for various cognitive functions, including a role in planfulness and self-control—low dopamine receptor density in this region was associated with loss of control. As you can see in these images, there is some improvement at 4 months post-use, but function has not returned to normal (images from NIDA, 2018).



## **Developmental Impact**

A great deal of attention to the developmental effects of exposure to alcohol and other drugs has been directed to two life periods: prenatal exposure and substance use during adolescence/emerging adulthood. These two developmental periods have an important commonality: these are periods when the brain is naturally undergoing rapid developmental growth or change. Thus, introducing substances that affect the brain can have more pronounced, amplified, and pervasive long-term effects.

**Prenatal exposure.** That alcohol exposure during fetal development can cause permanent damage to the brain and other organs has long been recognized, and fetal alcohol syndrome (FAS) was clearly identified as a possible outcome during the 1970s (Jones & Smith, 1973). Subsequent work has led to expansion of the definition and diagnosis of possible prenatal alcohol exposure (PAE) outcomes to reflect a spectrum referred to as fetal alcohol spectrum disorders (FASD) (Streissguth et al., 2000). FASD includes the syndrome (FAS), as well as alcohol-related neurodevelopmental disorder (ARND) and alcohol-related birth defects (ARBD). [Note that ARBD is also used to describe alcohol-related brain damage or ARBI for alcohol-related brain injury experienced by individuals later in life whose drinking patterns leads to brain injury, or ARBI for alcohol-related brain injury.] FASD is perhaps best understood as a “whole-body” diagnosis, as individuals with FASD experience a wide range of health and mental health conditions throughout life (Himmelreich, Lutke, & Hargrove, *in press*). We will learn more about the effects of PAE in our module on alcohol.

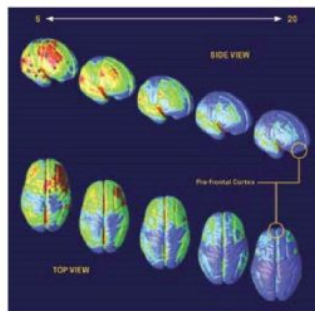
The effects of prenatal exposure to other substances is less well understood. Neonatal abstinence syndrome (NAS) is a known consequence experienced by many, but not all, infants prenatally exposed to opiates/opioids (Reber, Schlegel, Braswell, & Shepherd, *in press*). NAS concerns the infant’s experience of withdrawal from the substances previously circulating from the mother through the placenta and abruptly stopped with birth. The long-term complications of NAS may, but do not necessarily, include

neurocognitive and behavioral effects (Reber et al., *in press*). We will learn more about the known and possible effects of prenatal exposure to different types of substances as we learn about each in Part 2 of our course. It is important to know that many effects of prenatal exposure to alcohol or other substances do not appear right away at birth; some do not appear until children enter school or face increasingly demanding social and cognitive challenges which their brains are ill-equipped to handle. To minimize the negative developmental impacts of prenatal exposure and maximize developmental potential, early diagnosis and intervention is optimal (Loock, Elliott, & Cox, *in press*)—ideally, involving integrated teams of social work, medicine, nursing, physical therapy, occupational therapy, nutrition, and early education professionals.



**Adolescent/emerging adulthood exposure.** Shortly before and during puberty the human brain begins to undergo dramatic remodeling changes. The physical changes, to a large extent, involve reorganization of the connections between neurons and communication pathways between brain regions, particularly in the

Images of Brain Development in Healthy Children and Teens (Ages 5–20)



Source: PMID:10148174; DOI:10.1006/

prefrontal cortex. On one hand, a great deal of neuron “pruning” takes place, trimming out a great many underused or unused connections between neurons. On the other hand, myelination of existing neurons enhances connections between neurons that remain linked (Siegel, 2014). These two processes make the brain more efficient, better integrated, and capable of higher order functioning, but do not happen evenly and at the same time in all brain regions. The result is *emotional* functioning similar to that of adults but *cognitive* functioning that is as yet under-developed in terms of decision making, inhibitory control, planning, and working memory (Meredith & Squeglia, *in press*). Additionally, the adolescent brain is characterized by “heightened reward sensitivity and underdeveloped cognitive control that contribute to risky behaviors, including escalating substance use” (Meredith & Squeglia, *in press*). Heightened reward sensitivity suggests that the positive reinforcement experienced with substance use is experienced as more intensely positive (stronger reinforcement) than what is experienced by individuals later in life. The brain revision process normally tapers off from about ages 20 to 25. This image (from NIDA, 2018) shows how the concentration of grey matter shifts from age 5 to 20—the shift from yellow to blue in these images.

Thus, the brain is quite sensitive to developmental consequences of exposure to psychoactive/psychotropic substances up until age 25. The use of alcohol or other substances during these years can have profound, lasting effects on the still-developing brain; effects which have significant implications for how people think, behave, and feel, as well as for susceptibility to developing substance use disorders later in life. “In studies of drug use, an earlier age at which drug use was initiated is consistently related to a greater level of later drug-related problems,” (Hawkins et al., 1997, p. 281), making a delay in age of substance use initiation an important prevention strategy. Chances of developing severe substance use disorders is higher among individuals whose substance use began before age 15 years; “the biggest reduction in risk with deferred age of onset occurs when first use is postponed beyond age 15” (Robins &

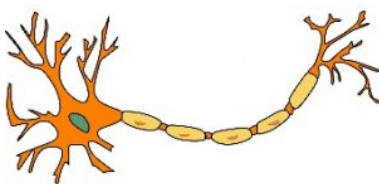
Przybeck, 1975, p. 184). Alcohol dependence was found to be four times more likely and alcohol abuse twice as likely among individuals whose age of drinking onset was before age 15 compared to individuals whose onset was delayed to age 21: “Overall, the risk for alcohol dependence decreased by 14 percent with each increasing year of age of drinking onset” (NIAAA, 1998). Deficits in adolescent brain functions and cognitive performance were observed with as little as 20 drinks per month, particularly if binge drinking was involved (Squeglia, Jacobus, & Tapert, 2009); some but not as great a level of divergence from their peers was detected with marijuana use. Finally, consider that a person’s overall health and development may be affected by poor nutrition, physical trauma or injury, or exposure to diseases that often accompany substance misuse.

### **Neurochemistry/Neurophysiology and Function**

In the previous section we explored what was happening at the level of brain regions. Now we turn attention to what is happening at a more microscopic level—neurons. As you may know from your previous education, the **central nervous system (CNS)** is comprised of about 86 billion nerve cells, called **neurons**, and about an equal number of **glial cells** that provide the energy neurons need to function (BrainFacts/SfN, <https://www.brainfacts.org/in-the-lab/meet-the-researcher/2018/how-many-neurons-are-in-the-brain-120418>). It makes sense to consider neurons and glial cells at this microscopic level because they are the building blocks of the brain regions previously discussed as playing key roles in substance use, substance misuse, and substance use disorder.



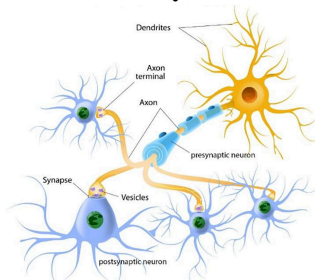
**Neuron activity.** The neurochemistry of substance use operates largely at two points. The first concerns the glial cells and how much energy they can provide to



neurons—the loss of glial cells or impeding their ability to provide energy has a negative impact on neuronal activity. The second concerns the ways that neurons communicate. Neurons physically pass [neurotransmitters](#) (molecules of naturally occurring brain chemicals) between each other as their mechanism for communication. Whether one neuron activates the next one depends on whether neurotransmitters are sent, whether those neurotransmitters are received by the next neuron, the amount of neurotransmitter sent and received, and the rate at which the neurotransmitters are reabsorbed after a “message” has been sent.

**Neurotransmitters.** A neuron’s neurotransmitter molecules are contained in packets called [vesicles](#), located in the terminal area of a neuron’s axon—the area that comes into close contact with the neighboring neurons (see Figure 3-2). The space between the neurons is the [synapse/synaptic cleft](#). This space between neurons is where neurotransmitters are released to work their changes. The “sending” neuron is the [presynaptic neuron](#), while the receiving neuron is the [postsynaptic neuron](#).

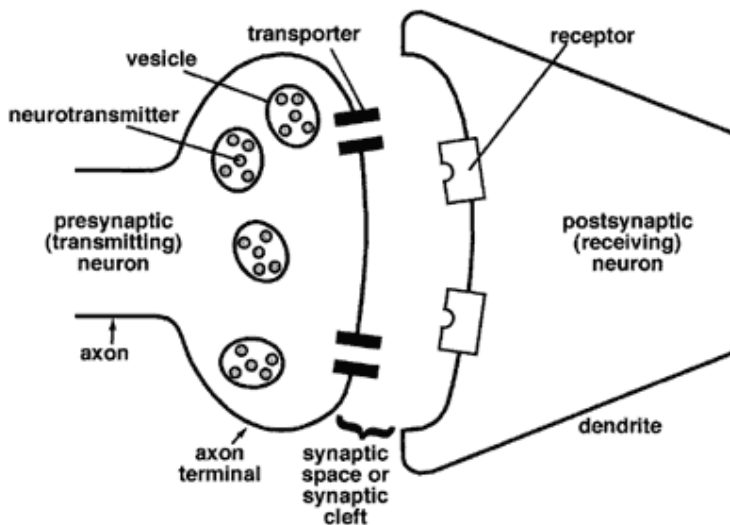
**Figure 3-2. Neurons and how they communicate**



The presynaptic (first) neuron releases neurotransmitter molecules (stored in the vesicles) into the synapse between it and

the postsynaptic (next) neuron. The postsynaptic neuron “receives” the neurotransmitter chemical if it has the right neurotransmitter [receptors](#)—kind of like a lock and key system. Neurotransmitters need the right receptors in order to “dock” and influence the postsynaptic neuron: if the right receptors are available, the neurotransmitter delivers the message but if the right receptors are not available, the neurotransmitter has no effect and just sits in the synapse. If the message is received by the postsynaptic neuron, it can now pass the message along to the next neurons in line. In the meantime, **transporters** retrieve and return the “used” neurotransmitter molecules back into the presynaptic neuron’s vesicles in preparation for sending a future message (see Figure 3-3). If a neuron has released its neurotransmitter molecules, it cannot send new messages until the supply has been restocked.

**Figure 3-3. Diagram of neurotransmission at the synapse** (from [science.education.nih.gov/supplements/webversions/BrainAddiction/other/](http://science.education.nih.gov/supplements/webversions/BrainAddiction/other/))



If the postsynaptic neuron's receptors are already filled, then the sent message will not be received—the neurotransmitters are blocked. This is how some drugs work—they occupy the receptor sites, thereby blocking messages between neurons. Other drugs work to reduce or increase receptor site sensitivity to the neurotransmitters. Still others work to influence the amount of neurotransmitter released into the synapse or affect the transporters' work in returning the neurotransmitter molecules to the vesicles.

### **Types of neurotransmitters.**

Different types of neurotransmitters have different impacts. For example, some play a more [excitatory](#) role, while others play a more [inhibitory](#) role. Excitatory neurotransmitters increase the likelihood that the receiving (postsynaptic) neuron will be triggered into activity; inhibitory



neurotransmitters suppress this kind of activity. Most types of neurotransmitter are either excitatory or inhibitory; a few can be either (e.g., dopamine). Different types of neurotransmitters are more concentrated in specific brain regions—while they may be distributed throughout the brain, they are not evenly distributed. This is why different substances “trigger” certain brain regions more than others—their effects are produced through their influence on the neurotransmitter communication processes and those neurotransmitters are more concentrated in certain regions.

Several types of neurotransmitter are known to play a role in the development, maintenance, and recovery from alcohol or other substance use disorders. Presented alphabetically, these neurotransmitters (and closely related neuropeptides) include:

- [dopamine](#) has both excitatory and inhibitory effects, depending on the nature of the receptor sites involved, is associated with the brain's reward systems, and is increased to



abnormal levels by substances such as alcohol, cocaine, and heroin (influencing their addictive potential);

- [endorphins& enkaphlins](#) are two neuropeptides (rather than neurotransmitters) that play a role in producing some of the rewarding effects experienced with the use of alcohol and some other substances—endorphins relate to opiate receptors causing an analgesic (pain control) effect and enkephalins are similar to endorphins;
- [epinephrine](#) is an excitatory neurotransmitter (also called adrenaline) involved in the “fight or flight” response;
- [GABA\(gamma-aminobutyric acid\)](#) is an inhibitory neurotransmitter widely distributed throughout the brain and plays a critical role in alcohol misuse and alcohol use disorder (and possibly other substances) because alcohol increases the effect of GABA contributing to feeling more calm, relaxed, and even sleepy;
- [glutamate](#) is the most common neurotransmitter found in the human CNS, is excitatory, plays a key role in regulating attention and arousal, and typically acts in opposition to GABA;
- [norepinephrine](#) acts in opposition to epinephrine, as an inhibitory agent, to control “fight or flight” functions stimulated by epinephrine (also called noradrenaline);
- [serotonin](#) is an inhibitory neurotransmitter that helps regulate many functions (sleep, cravings, and pain control, among others) and emotional states, off-setting the effects of excitatory neurotransmitters.

Several things are very important to understand about neurotransmitters and the system of communication in which they are involved:

- We used to believe that each neuron could only release one type of neurotransmitter. More recent research indicates that in many cases the same neuron can release two and possibly more types depending on the frequency of the stimulation it

receives—at one frequency it might release one type of neurotransmitter, at another frequency it might release a different type.

- Most neurotransmitters occur naturally as important chemicals in other parts of the body (including the peripheral nervous system and other organs) where they have other health-related functions, not just in the brain (central nervous system). For example, the human body naturally has opioid and cannabinoid receptors that are meant to respond to naturally occurring (endogenous) chemicals to control pain, reward certain life-supporting behaviors, and influence learning and memory. These receptors are also responsive to introduced chemicals (exogenous) which are often introduced in much higher doses than naturally occur—from using cannabis/marijuana or opioid drugs. Opioid receptors are also involved in responses to alcohol.
- Neurotransmitter release is triggered by many natural behaviors, not just by alcohol and other substances. For example, dopamine release is involved in the natural reward systems associated with food, sex, humor, pair-bonding (mates), listening to music, and video games. The addictive potential of a psychoactive drug increases when the concentration of dopamine released is higher compared to what is released by natural behaviors (Johnson, 2014).
- Fast uptake of a drug, for example getting it to the brain by injection rather than ingesting it orally, produces a stronger “high” and therefore a greater potential for addiction. This is because more dopamine is released at once, so it is more rewarding (Volkow et al., 2010).

## Homeostasis

One hallmark of the human brain is its adaptability (neuroplasticity),

whereby its various functions adjust to conditions in order to maintain overall balance or [homeostasis](#). This adaptability gives rise to acquired tolerance when a substance (or type of substance) is used repeatedly over time. In this chapter we examine how homeostasis plays a role in the development of tolerance, as well as the biological basis of the substance withdrawal experience. In addition, we examine why the age at which the brain becomes exposed to substances matters and a few basic principles concerning pharmacotherapy for treating substance misuse and SUD—we look into this last topic more deeply later in the course.



**Acquired tolerance.** You may recall from Module 2 that we defined acquired tolerance as a person requiring higher doses of a substance (or type of substance) to achieve the same effects or experiencing lesser effects (even withdrawal) when the same dose is used if the substances have been used repeatedly over time. Let's consider what is happening at a neurochemical level. When a person uses a great deal of alcohol often over time, the brain begins to adapt to the presence of the alcohol and its effect on GABA. In attempting to reacquire a state of homeostasis, the brain boosts its arousal systems (glutamate) to offset the overly inhibitory impact of the extra GABA triggered by the alcohol. This is called [upregulation](#) of the glutamate system—additionally activating the system that produces glutamate. In addition, the brain may begin to control the amount of GABA through [downregulation](#) of the GABA system—suppressing the system that produces GABA. In other words, two things are going on to offset the effects of chronic alcohol exposure: downregulating GABA and upregulating glutamate. This means that, in order to experience the same effects at the same level, a person needs to take even more alcohol to boost

the GABA even more. This internal neurophysiological teeter-totter continues to see-saw over time.



**Experience of withdrawal.** At this point, you have developed a basic understanding of how neurotransmitters and homeostasis play a role in the development of a substance use disorder. Up until this point, we have been exploring what happens when the brain is exposed to certain substances. Now, let's look at the other side of the coin: what happens when the brain is no longer exposed to substances to which it has grown accustomed. Remember that the brain has adapted to the chronic presence of the substance (alcohol, in our example) by downregulating GABA and upregulating glutamate systems (see the “Tolerance” section above). Withdrawing the substance (alcohol) means that the GABA and glutamate are going to be out of balance for a while, at least until the GABA begins to upregulate again and the glutamate to downregulate, re-acquiring a state of homeostasis without alcohol being present. The withdrawal of substances can result in the experience of withdrawal symptoms—an experience that may be intense (even potentially deadly) and prolonged. In our next module (Module 4 about psychological models) you will learn more about why withdrawal symptoms might make a difference in a maintaining a “quit” attempt or relapsing to using substances again.

We can draw from content presented in articles published by Koob and Simon (2009) and Trevisan et al (1998). They tell us that:



- A decrease in dopamine or serotonin contributes to the experience of [dysphoria](#) and [anhedonia](#). Dysphoria is the experience of a profound sense of unease, unhappiness, and general dissatisfaction, often associated with major depression and anxiety. Anhedonia refers to a lessening or inability to experience pleasure. Thus, removing substances that stimulated dopamine or serotonin activity can have these effects. A decrease in GABA contributes to the experience of anxiety, even panic attacks, due to the resulting nervous system hyperactivity. An increase in glutamate contributes to hyperexcitability. Thus, removing substances that affected GABA and/or glutamate activity can have these effects.
- An increase in norepinephrine contributes to the experience of stress. Thus, removing substances that affect epinephrine and/or norepinephrine can have this effect.

Why does this matter? These negative emotional and psychological states make it difficult to sustain motivation to avoid using alcohol or other substances and contribute to the pressure a person might feel to relapse into using again. Depending on the nature of the substances involved, withdrawal may lead to decreased dopamine, serotonin, or GABA, as well as increased norepinephrine or



glutamate. Knowing about these links between neurotransmitter changes during prolonged withdrawal from using a substance contributed to the development of several medications to help manage these negative experiences and perhaps help a person sustain a “quit” attempt over time (pharmacotherapy). We will learn about specific pharmacotherapy medications in Module 13. Another reason this matters is that during withdrawal and early recovery from many types of substance use disorders, the risk for suicide is greater than in the general population because of these brain-behavior processes.



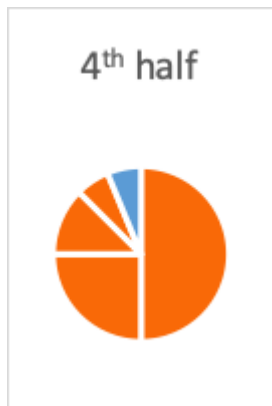
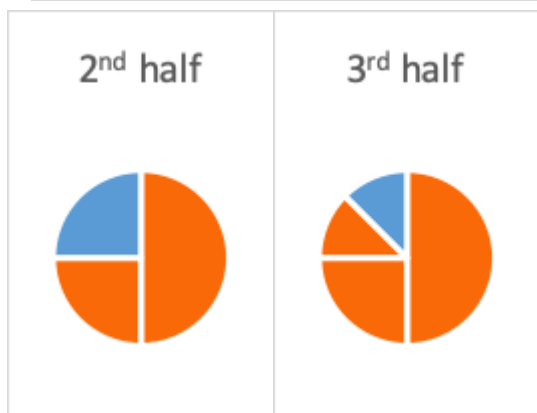
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# Ch. 3: Basic Pharmacokinetic and Psychopharmacology Principles

In this chapter, we introduce some basic principles of [pharmacokinetics](#) and [psychopharmacology](#). Pharmacokinetics is the study of how drugs are distributed and metabolized (broken down) in the body—it represents a branch of pharmacology. We are concerned here with the patterns by which different substances are absorbed, metabolized, and excreted. The principles we examine in this chapter help explain overdose and differences in how quickly different substances begin to have an effect or how long the effects might last. Not only do certain drugs have an effect themselves, but so do their [metabolites](#)—the breakdown products—thereby extending the duration of the effects overall. Psychopharmacology is concerned with how different drugs have their effect on the brain. Our emphasis in this introduction to psychopharmacology concerns how different drugs might influence the actions of different neurotransmitters as agonists, antagonists, and synergistic effects. These actions have implications for medications that can be used to treat substance use disorders ([pharmacotherapy](#)).

**Half-life.** The duration of a drug's effect is measured in terms of its pharmacological [half-life](#) which describes the relationship between the active dose circulating in the body (its concentration) and the variable of time. The first point where time matters is at the front end—from the time of administration, different drugs take a different amount of time to reach peak level. Then, as a drug is metabolized, there comes a point in time when its circulating concentration is half of what it was at its peak level. The time that it takes to achieve this point is what “distribution half-life” refers to.



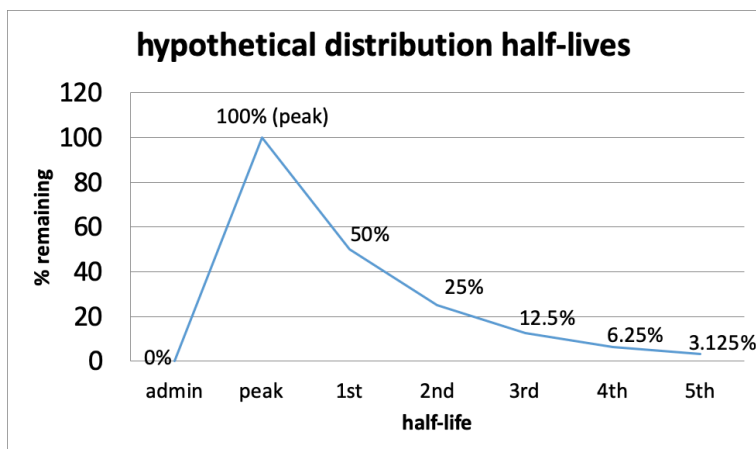
- The first half-life is the point when 50% of the drug is gone;



this means 50% remains ( $100\% - 50\% = 50\%$ ).

- The second half-life is where 50% of what remained after the first half-life is gone—in other words, another 25% is gone (half of 50); together this means the original 50% + next 25% = 75% of the peak level is gone, or only 25% remains ( $100\% - 75\% = 25\%$ ).
- The third half-life is where 50% of what remained after the second half-life is gone—12.5% is half of 25%, so now 75% + 12.5% = 87.5% of the peak level is gone; only 12.5% remains ( $100\% - 87.5\%$ ).
- And so on, until virtually none remains.

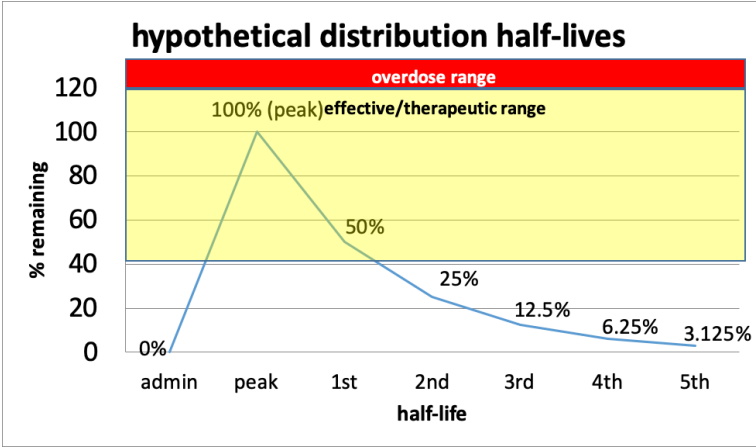
This curve might help you visualize the relationship of half-lives and time for a hypothetical situation. The principles behind the curve are the same for every drug, it is the length of time for each half-life that differs—it could be minutes (e.g., some inhalants), hours, or even days. This also affects how long after using a substance it can still be detected in drug tests. Alcohol can be detected for 7-12 hours after drinking in a urine test (Moeller et al., 2017), or possibly longer depending on how much was consumed and several other factors. Marijuana can be detected in urine for about 3 days for some who uses it occasionally and for more than a month after last use by a person who uses it multiple times a day (Moeller et al., 2017). Opioid detection is possible in urine tests for 2-4 days for the most common forms, and this is about the range for detecting cocaine metabolites in urine, as well (Moeller et al., 2017).



Different drugs, even within the same class of drugs, differ in terms of their half-lives as well as their range of effect—with medicine this would be called the therapeutic range. In other words, one drug might stop having an effect at the first half-life while another may still have an effect at the third half-life. For example, there is a big difference between “short-” and “long-” acting barbiturates and benzodiazepines. Urine tests can detect short-acting pentobarbital for 24 hours and long-acting phenobarbital for 3 weeks, although both are barbiturates; short-acting benzodiazepines (e.g., lorazepam) might be detectable in urine for 3 days, while long-acting benzodiazepines (e.g., diazepam) might be detected for 30 days (Moeller et al., 2017).

This curve shows the relationship of half-lives to dose effect. The distance between peak dose and overdose differs by drug—in some cases, there is very little “wiggle room,” making it very easy to end up with an overdose (the red line in the curve). This is true, for example, of barbiturates and benzodiazepines—the difference between therapeutic and overdose range can be quite narrow. As the dose of a drug increases, so do the risks of side effects, even below the overdose level.

In this hypothetical example, a person might need to take more of the drug at the point where 50% remains (first half-life) in order to maintain an effective dose, but it is going to be important to avoid a peak dose that takes that person into the overdose range. What is known about half-lives, effective dose range, and overdose range is based on averages across individuals—it may differ for a single individual and by various conditions (including the person’s overall health and presence of other drugs). Individuals differ somewhat in how they metabolize drugs. It also is based on drugs of known composition—produced under controlled pharmacy conditions. You can see why drugs manufactured in uncontrolled conditions (e.g., “meth” labs, foreign labs, homemade) can be so much more unpredictable.



In this hypothetical example, consider that it took 1 hour for the drug to reach its peak level and that the half life is 3 hours long. That means at about 4 hours, the person will need to re-dose to maintain a therapeutic dose level or level where the effects remain in the desired range. If we are concerned about withdrawal symptoms, that is the point where the symptoms might begin to be experienced with this hypothetical drug. By about 16 hours, the

person will have very little of the drug remaining in the body (5<sup>th</sup> half-life).

**Metabolites.** The breakdown process for many substances is not as simple as “there and gone.” In many cases, the process of metabolizing a drug or other substances happens in a sequence of steps, and the intermediary products may exert effects themselves. For example, alcohol (ethanol) is first metabolized (broken down) into another chemical called acetaldehyde. Acetaldehyde is toxic and thought to be responsible for many of the “hangover” symptoms associated with alcohol consumption, as well as with the increased risks for cancers. Fortunately, acetaldehyde does not stick around very long as it is metabolized into a less toxic chemical, acetate. Acetate is then metabolized into carbon dioxide and water. The enzymes responsible for the metabolism of ethanol into acetaldehyde (alcohol dehydrogenase, or ADH) and of acetaldehyde into acetate (aldehyde dehydrogenase, or ALDH) are both, to a large extent, under genetic control. This contributes to the observed phenotypic differences in individuals’ responses to drinking that we previously explored in discussing genetics—this is the mechanism through which genetics operate. We will look into this in more detail in our module focused on alcohol.

**Agonists, antagonists, and synergism.** While we have been looking at what happens when one or another substance is used, it is important to understand what happens when two or more substances are involved. This information helps inform strategies for medications used in pharmacotherapy—the use of medications to treat various forms of substance use disorder (including alcohol use disorder). As you may know from warnings on prescriptions you have taken, substances sometimes interact if they are in the body at the same time. Here is how they might influence one another.

**Agonists.** An [\*agonist\*](#) activates specific types of receptor sites in the brain or elsewhere in the body, causing a specific effect. For example, THC is a chemical in cannabis (marijuana) that activates the naturally occurring cannabinoid receptors in the brain. This is how it produces its psychoactive effect. This principle can be

used in treating substance use disorders. For instance, a drug that activates the opioid receptors in the brain can reduce or eliminate withdrawal symptoms by acting like the substance that has ceased to be used. This is why methadone can help in the treatment of heroin/opioid use disorder—it acts enough like the heroin/opioid to help without the added risks and potential harms of using the original substance even if the person continues to experience a dependence on the class of substance involved. (Methadone itself is an addictive substance, but consistency in quality and dosing can be more carefully controlled and it can be more gently weaned over time to further reduce the likelihood of relapse.)

**Antagonists.** Like [antagonists](#) in a story (or superhero/villain comics), two substances may work against each other. Antagonists mostly work by blocking receptor sites in the brain so that a drug cannot trigger its expected response. For example, naloxone is used as an emergency first response to heroin/opioid overdose. This potentially life-saving medication blocks the effects of heroin or other opioids. In other words, naloxone is an opioid antagonist. This antagonist principle is used in developing some of the current medication treatments for alcohol use disorder and other substance use disorders.

**Synergism.** Certain substances, when combined, create a stronger or more prolonged response than either could alone. This is called [synergism](#). For example, the combination of alcohol and barbiturates amplify the CNS depressant effect which is why it is easy to overdose on this combination. It takes less of either when taken together to achieve the same or greater/more prolonged effects as taking either substance alone—however, this goes for side-effects and overdose risk, as well.

# Ch. 4: Summary

In the readings for Module 3, you read a great deal of information about the biological basis of substance use, substance misuse, and substance use disorder. You learned:

- How pedigree, twin, adoption, and genomic studies contribute to our understanding of the genetic basis of substance use and substance use disorders;
- That genetics plays an important role, but that genetics do not operate alone;
- The mechanisms throughout which genetics have their influence on substance use disorders;
- The ways that different areas of the brain might be involved in substance misuse and substance use disorder, particular elements of the limbic system;
- How important age at which the brain is exposed to alcohol and other substances matters greatly and why this matters;
- Basics about how neurotransmitters are involved in substance use and the progression to substance misuse or substance use disorder;
- Basic principles of pharmacokinetics related to drug dosing and metabolism;
- Basic principles of psychopharmacology that explain tolerance and withdrawal, as well we how drugs interact and how medications might help treat substance use disorder.

You are well-prepared to move into our next module which translates much of what we have learned is going on in the brain into what transpires psychologically.

# Module 3: Key Terms

**agonist:** a chemical/substance that activates a specific type of receptor site in the brain or body (opposite of antagonist).

**alleles:** the alternative forms of a gene found at a specific chromosomal location.

**amygdala:** location in the brain associated with emotion.

**anhedonia:** inability to experience pleasure/happiness.

**antagonists:** substances that block or reduce responses by blocking receptors (opposite of agonist)

**autonomic nervous system (ANS):** portions of the nervous system responsible for controlling bodily functions outside of conscious control (e.g., digestion, heart rate, breathing rate, blood pressure).

**central nervous system (CNS):** the brain and spinal cord.

**chromosomes:** sites where genes are located; humans have 23 pairs of chromosomes present in every cell, except egg and sperm cells which have 23 single chromosomes

**chromosomal regions:** sections of a chromosome.

**concordance:** the degree of similarity or agreement in what is being compared (e.g., a pair of twins).

**dizygotic twins:** twins developing from two different fertilized eggs.

**DNA:** the hereditary material (deoxyribonucleic acid) passed from parents to offspring.

**dopamine:** a primary neurotransmitter (and precursor to producing other molecules, like epinephrine)

**downregulation:** reducing or suppressing a response or sensitivity to a substance (opposite of upregulation).

**dysphoria:** experience of unease or dissatisfaction with life which can be intense.

**endorphins & enkaphalins:** peptides in the body with brain and nervous system effects, especially with regard to opiate receptors and pain control.

**epinephrine:** also known as adrenaline, a stimulant/arousing

hormone released in the body that influences autonomic nervous system functions (heart rate, respiration, and muscle preparation for action), acts in opposition to norepinephrine (noradrenaline).

**excitatory neurotransmitters:** neurotransmitters that have an activating effect on postsynaptic neurons.

**GABA (gamma-aminobutyric acid):** a neurotransmitter pervasive throughout the brain which inhibits neuron responses.

**genes:** sections of DNA sequences that direct how/whether biological processes occur.

**genome:** the complete set of genes present in a cell/organism; humans share 99.9% of their genome, with individual difference attributed to that very small remaining percent (NHGRI, 2018)

**genotype:** the set of genes responsible for a certain trait/characteristic.

**glial cells:** a type of cell in the CNS that support neurons.

**glutamate:** an excitatory neurotransmitter.

**half-life:** the period of time it takes for the body to metabolize a drug by half its concentration.

**heritability:** estimate or measure of the contribution of genes (versus environment) to a phenotypic outcome based on a proportion of observed variance in the trait studied

**hippocampus:** area of the brain responsible for emotion, memory, and control of the autonomic nervous system.

**homeostasis:** the tendency in systems to establish and maintain a relatively stable, balanced state; many physiological processes have opposites so they can work in tandem to create this balance.

**inhibitory neurotransmitters:** neurotransmitters that have a suppressing effect on postsynaptic neurons.

**limbic system:** a networked system of brain regions that control basic emotions and drives.

**metabolites:** substances formed in the process of breaking down (metabolizing) other substances.

**monozygotic twins:** twins developing out of the same egg fertilized by a single sperm.



**neuroanatomy:** study of the anatomy (structures) of the nervous system.

**neurochemistry:** study of the biochemical processes occurring in the nervous system.

**neurons:** type of cell in the CNS (nerve cells).

**neurotransmitters:** types of molecules involved in communication between neurons.

**norepinephrine:** also known as noradrenaline, a suppressing/inhibitory hormone released in the body that influences autonomic nervous system functions (heart rate, respiration, and muscle preparation for action), acts in opposition to epinephrine (adrenaline).

**nucleus accumbens:** also called the accumbens nucleus, an area of the brain involved in the reward circuit, primarily using dopamine to stimulate desire and serotonin to establish satiation.

**pharmacokinetics:** branch of pharmacology concerned with how drugs move and are metabolized in the body.

**pharmacotherapy:** providing treatment by the use of medications/drugs.

**phenotype:** an observable/expressed characteristic, trait, behavior, or disease outcome influenced by some combination of genotype and environment.

**polygenic:** a trait, characteristic, or disease attributable to variation in multiple genes.

**postsynaptic neuron:** a neuron receiving communication from another neuron.

**prefrontal cortex:** area of the brain playing a significant role in regulating cognitive processes and higher-order thought, emotion, and behavior.

**presynaptic neuron:** a neuron sending communication to another neuron.

**psychopharmacology:** the study and use of psychoactive/psychotropic medications, drugs, or other substances to create brain changes.

**receptors:** sites on (nerve) cells where neurotransmitters have their

influence if there is a match between type of neurotransmitter and receptor site.

**serotonin:** a neurotransmitter involved in balancing emotion and mood, with a role in social behavior, sleep, memory, appetite, and sexual function.

**synapse/synaptic cleft:** the space between two neurons where communication by neurotransmitters takes place.

**synergism:** the increase in strength or duration of an effect by combining two substances with similar actions.

**transporters:** the route by which neurotransmitter molecules are returned to the presynaptic neuron vesicles.

**upregulation:** enhancing or increasing a response or sensitivity to a substance (opposite of downregulation).

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## PART IV

# MODULE 4: PSYCHOLOGICAL MODELS OF SUBSTANCE MISUSE

## Introduction

In Module 3 we examined what is happening on the brain side of the brain-mind-behavior chain with regard to substance use, substance misuse, and substance use disorder (SUD). Our emphasis in Module 4 centers around psychological theories concerned with the mind-behavior part of this chain. Generally, psychological models in our biopsychosocial framework address cognitive (thoughts, beliefs, attitudes, learning, knowledge) and affective (emotions, feelings) dimensions. Topics we explore in Module 4 include models related to cognition, information processing, learning, social learning, rational/planned behavior, developmental, psychodynamic, attachment, self-medication, personality, psychopathology, expectancies, and cravings theories. Much of what we examine regarding psychological processes directly relates to what we learned in Module 3 about the brain; it is virtually impossible to completely separate “mind” and “brain” functions. By the end of these readings, expect to have developed an appreciation for and understanding of the psychological basis of substance misuse and SUD, including how these theories might help inform prevention, treatment, and recovery-oriented intervention strategies.

Note: Contents of this module both heavily influenced and were influenced by the contents of the Begun (*in press*) chapter listed in the references: Begun, A.L. (*in press*). Psychological models of

addictive behavior. In A.L. Begun & M.M. Murray (Eds.), *Handbook of social work and addictive behavior*. London: Routledge.

## Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Explain how cognition, information processing, learning, social learning, rational/planned behavior, developmental, psychodynamic, attachment, self-medication, personality, psychopathology, expectancies, and cravings model relate to substance misuse;
- Describe the relationship between brain-mind-behavior;
- Identify implications of these theories/models for treatment and recovery efforts.

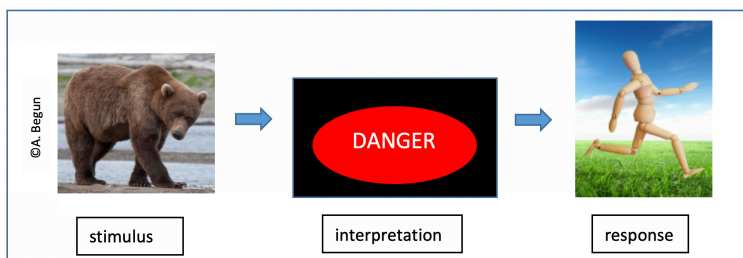


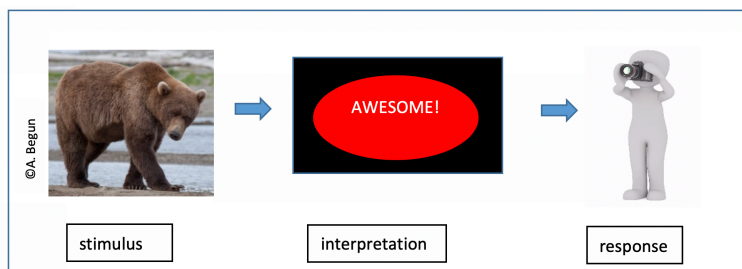
# Ch. 1: Cognitive and Learning Theories

The first group of theories examined in this module are those related to how thinking and learning are both involved in and affected by substance use, substance misuse, and substance use disorders. **Cognition** concerns the mental processes involved in a person's knowledge, thoughts, and understanding of their experiences. Here we are not only interested in *what* a person thinks and believes, but also *how*—the processes and mechanisms that determine what someone knows, thinks, and believes. Psychology even has a word for thinking about thinking—this is metacognition.

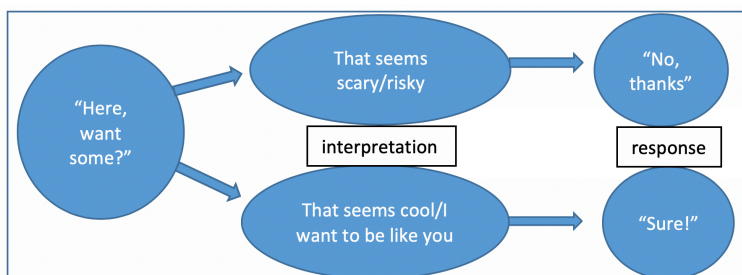
## Cognitive processing

**Cognitive processing** has a great potential to influence human behavior. For example, how a person interprets a situation has a great deal to do with how that person will respond/ behave in the situation. Here are different ways a person might interpret seeing a grizzly bear in the wild (stimulus) and how their response is dependent on that interpretation.





Now let's apply this to an example possibly related to cannabis initiation. What happens when a person is offered alcohol, marijuana, or another psychoactive substance.



Here is another way in which cognitive processing—interpreting situations—is relevant. Consider the body of evidence concerning women becoming less aware of (or less uncomfortable with) situational cues concerning their risk of being sexually assaulted as their blood alcohol concentration rises to or above that specified as unsafe for driving—0.08 (Davis et al., 2009; Testa & Livingston, 2009). Substance use can impair a person's interpretation of the potential riskiness of certain situations, which in turn can diminish their capacity for self-protection and early termination of coercive interactions. Practices like pre-planning for one friend designated as non-drinking during an outing and "having your back" may be

employed by women who plan to engage in drinking activities; effectiveness is dependent on that one friend's power to discern riskiness and effectively deter another from making an unsafe decision.

The [alcohol myopia](#) theory concerning intimate partner violence (IPV) behavior presents another example of how substance use might determine how situations are interpreted, which in turn influences behavior. The theory addresses the fact that IPV incidents are more frequent when one partner in a violent relationship has been drinking alcohol (Mengo & Leonard, in press). With alcohol myopia, a person might focus on immediate circumstances and events rather than placing them in a broader or longer-term context—becoming “nearsighted” in a situation—when alcohol has been consumed; this interferes with reasonable, accurate interpretation of what is happening. Alcohol myopia theory suggests that someone who has been drinking may be more likely to interpret another person's behavior as threatening: the pharmacological properties of alcohol reduce capacity to derive meaning from complex information as happens in most social exchanges (Eckhardt, Parrott, & Sprunger, 2015). Interpreting an innocuous behavior as a threat leads to an aggressive behavioral response, including IPV.



Cognitive processes link to our feelings/emotions/affect, as well. For example, how we label our feelings has an influence on how emotions are experienced and how we behave in response to emotions. For example, if you have only a few labels available for describing and understanding [affect](#) (e.g., mad, sad, glad) then you have relatively few options available for how you behave in

response; having more affective labels cognitively available for the emotions related to an event or experience offers a wider array of behavioral responses. Consider, for instance, what might happen in two different scenarios where someone gets a poor grade on an exam—it feels “bad” but what kind of “bad” or negative affect we identify determines how we might respond to the event. Some of the solutions or options are more productive than others:

Affect Label	Bad=mad	Bad=sad	Bad=frustrated	Bad=disappointed	Bad=guilt/shame
Response options	quit; run away; blame others; threaten others; try to improve mood with exercise or substance use	cry; mope; hide from the situation; try to improve mood with exercise or substance use	problem solve; vent to others; “walk it off,” learn from mistakes for next time; negotiate	problem solve; elicit sympathy from others; “walk it off,” learn from mistakes for next time; negotiate	quit; run away; cheat or lie; apologize; try harder next time; elicit sympathy from others

Individuals differ in how they cognitively label their affective (emotional) experiences which helps explain why they differ so much in how they respond to situations. For example, what is YOUR label for the affect this ambiguous screen bean character is experiencing?

Happy?  
Terrified?  
Excited?  
Dancing?  
Playing a sport?  
Injured?  
Falling?



Identifying what is happening has a lot to do with how we respond behaviorally and understanding this helps us understand a great deal about substance misuse—not only how affect might lead to substance use/misuse but also how substance use might alter emotions and the cognitive processes involved. [Note: the word “affect” here is not about effects—it is pronounced with the “a” like in apple, not like “uh” in apothecary (which is another word for drugstore).]

A great deal of emphasis is in [\*cognitive behavioral therapy \(CBT\)\*](#) and other cognitively-based interventions centers on helping someone reinterpret situations, cues, and stimuli and develop new behavioral responses to those cues. Treatment strategies based on theories or models of the role cognition plays in addictive behavior (e.g., cognitive behavior[al] therapy, rational emotive therapy, cognitive skill building) have a common assumption: “Certain cognitive, emotional, and social skills are particularly useful for voluntarily steering one’s path out of addiction” (Heather et al., 2018, p. 251).

Rotgers (2012) identified a set of common basic assumptions among cognitive behavioral (CB) models and interventions related to substance use disorders, most of which could be applied to other forms of addictive behavior:

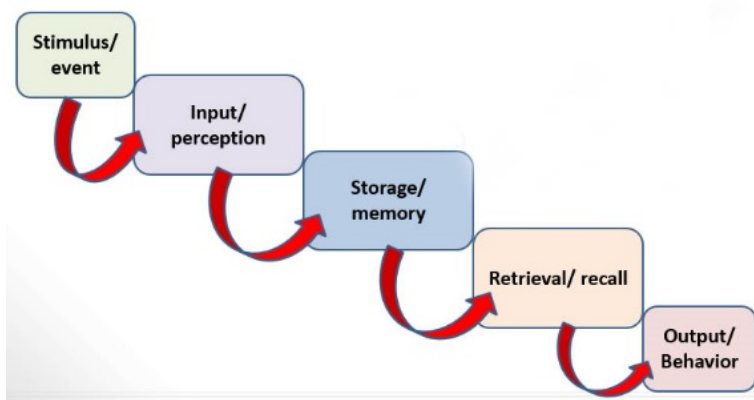
- human behavior is largely learned;
- learning processes leading to problematic behaviors also apply to changing these behaviors (classical conditioning, operant

conditioning, modeling);

- environmental context factors play a major role in determining behavior;
- learning principles apply to changing covert behaviors (e.g., thoughts and feelings), not just overt behaviors;
- critical to changing behavior is the practice of new behaviors within the contexts where they will be performed;
- each individual person is unique and must be assessed with consideration of their experienced contexts;
- “The cornerstone of adequate treatment is a thorough CB assessment” (p. 114); and,
- “A strong working alliance is crucial to effective behavior change, regardless of therapy technique” (p. 115).

## Information Processing

The information-processing model comes from cognitive psychology and helps explain (1) what a person “knows” about a substance, and (2) how a person’s substance use might affect behavior through its influence on perception, short- and long-term memory, and information retrieval. Not only does this model have implications for information/education intervention and how individuals behave while under the acute influence of certain substances, it also has implications concerning long-term (chronic) substance misuse and recovery from SUD. Information processing concerns how we initially take in information about our environment (or from internal biological cues). Then, what happens with that information and does it influence behavior? Let’s look at the information processing steps.



**Perception.** Before information, stimuli, events, or experiences can influence an individual's behavior, several things need to happen in the processing the information. First, the person must attend to and perceive the stimulus through one or more of the five senses—the ways we generally perceive cues from the external environment (seeing, hearing, taste, smell, touch). However, we also perceive myriad cues from internal sources all the time (hunger, fatigue, arousal of “fight or flight” systems, etc.), whether or not we are aware of these internal cues. Regardless of the source, the first step in information processing involves “input” of information.

We know that different types of substances can have different effects on this perception phase of information processing. Have you ever noticed that conversations become progressively louder as individuals in conversation consume more and more alcohol? This is not solely about disinhibition. One effect of alcohol is to reduce the transmission of sound stimuli to the brain—people no longer hear their own voices as loudly so they compensate by talking more loudly. This is only one example of substance use influencing behavior through affecting perception.



**Memory.** Next, perceived information moves into memory storage—or not. Perceptions that do not move into memory are simply gone, eliminated from the system. They no longer have the power to influence an individual's behavior. The first part of memory storage involves short-term (or “working”) memory. There is relatively little storage capacity in this working memory phase—information is lost after about 20-30 seconds unless it is transferred into long-term memory. Long-term memory involves storing information over time. Of interest here is that memories are not necessarily stored intact; they are highly susceptible to distortion and bias as they are stored. This is because humans tend to store memories in terms of their personal meanings and often are combined with other memories. This is part of why eye-witness testimony is so fraught with inaccuracies—the memories become distorted in the storage process. Human memory is not like a digital camera, storing images as they appeared when captured. For one person, some aspects will have more or less salience compared to other individuals, making them more or less memorable.



**Retrieval.** Depending on how memories were stored (long-term), they need to be recalled or retrieved in order to influence behavior. Cues from other stimuli or memories can “trigger” recall of a stored memory—for example, smelling marijuana might “trigger” recall of how it felt to use it or driving the car might “trigger” memory of how it felt to smoke a cigarette while driving. This is an important aspect of cravings. On the other hand, evidence concerning [state-dependent learning](#) suggests that retrieving information is easiest and most accurate when conditions





are very similar to when the information was originally introduced/learned. In other words, information or skills learned and easily retrieved while under the influence of alcohol or other substances may be more difficult to retrieve when a person is in a different (unaltered) state of consciousness (Overton, 1984). Vice versa, what is learned under normal conditions may not be recalled when in an altered state. Thus, a person in recovery may need to relearn information or skills originally learned while under the influence of substances.

**Substance-distorted information processes.** In addition to examples of how each step might be affected by substance use, psychoactive substances can profoundly affect overall information processing. For example, information processing overall is slowed among men engaged in chronic excessive alcohol consumption compared to men who do not drink alcohol excessively, beginning with perception and carrying through the decision-making and response (behavior) phases (Kaur et al., 2016). This, in part, explains delays in reaction time and the risk of driving a vehicle under these conditions. Fortunately, affected cognitive functions improve in many individuals during months to years of abstinent recovery (Cabé et al., 2015). In addition, consider the possibility that individuals in early recovery may not effectively process information delivered through treatment/intervention efforts with a heavy cognitive component—these strategies are better processed a few weeks into recovery (NIAAA, 2001).



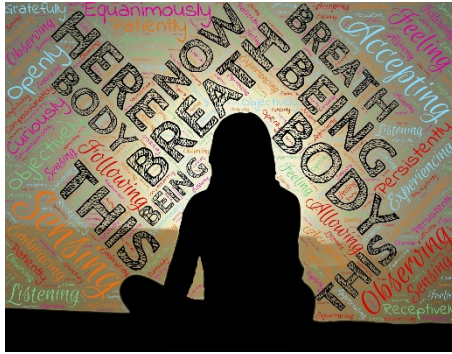
# Learning Theory

Learning theories represent one set of psychological principles that have had a strong influence on our understanding of substance misuse and SUD. Relevant learning theories include both operant and classical conditioning principles.

**Classical Conditioning.** Pavlov demonstrated classical conditioning in his experiments with dogs. The process involved learning where a previously neutral stimulus paired with a naturally potent (unconditioned) stimulus came to elicit the same response (conditioned stimulus) as the natural (unconditioned) stimulus. In Pavlov's experiments, this meant the ability to trigger a salivation response to the sound of a bell after repeatedly pairing the sound with presentation of food. Salivating is a naturally occurring response by dogs to having food presented (unconditioned stimulus). Repeatedly pairing the sound of a bell with the presentation of food, which elicits salivation (unconditioned response), eventually makes the dog salivate in response to the bell alone—the bell has become a conditioned stimulus and salivation to the bell (rather than food) has become a conditioned response.



The same learning principle may apply to certain substance use phenomena. For example, drug paraphernalia, specific settings or environments, certain people, or even certain emotions may become conditioned stimuli eliciting a desire to use the substances previously associated with them. Unfortunately, it is challenging to unlearn strong conditioned pairings, especially those with particularly powerful feelings attached (remember what we learned about what is going on in the brain with learning and memory in Module 3). Fortunately, it is possible to train new pairings, such as training a person to use relaxation, breathing, mindfulness, and delaying techniques in response to the feelings stimulated by the conditioned stimuli.



**Operant Conditioning.** Another set of psychological learning principles with a profound impact on substance misuse is [operant conditioning](#). Operant conditioning is all about rewards and punishments (recall what you learned about the reward circuits in the brain in Module 3—for example that dopamine release is a powerful reward). If someone experiences a positive consequence as the result of using a particular substance, the reward ([positive reinforcement](#)) increases the probability of repeating that behavior again in the future. Experiencing a negative consequence ([punishment](#)) decreases the probability of repeating that behavior again in the future. Considerable confusion revolves around the concept of [negative reinforcement](#), and because this is an important process in substance misuse negative reinforcement warrants some closer attention. Let's start with this chart comparing consequences and effects in operant conditioning.

	Consequence	Effect	Label
BEHAVIOR	provide favorable stimulus (reward)	increased probability of repeating behavior	positive reinforcement
	remove unfavorable stimulus (reward)	increased probability of repeating behavior	negative reinforcement
	provide unfavorable stimulus or remove favorable stimulus (punish)	decreased probability of repeating behavior	punishment

On the far left, we have a person engaging in a specific behavior—exercising, for instance. Looking in the middle and to the right we see the possible consequences, effects of the consequences on future behavior, and what we call this type of operant conditioning learning.

- If the person exercises to the point of experiencing endorphin release in the brain, the positive experience is rewarding. In other words, the exercising behavior was positively reinforced which increases the probability that the person will engage in that behavior again in the future—chasing down that positive reinforcement experience in the form of endorphin release.
- If the person aches and is winded instead, the experience is quite negative. In other words, the exercising behavior was punished which decreases the probability that the person will engage in that behavior again in the future.
- What if the person starts out with negative feelings—may they feel anxious or somewhat depressed (negative experience)—but manages to get active in some form of

exercise (behavior). If the anxiety or depressed mood is removed, the exercising behavior has been rewarded. Rather than providing positive reinforcement (as we saw in the first example with endorphin release), the behavior removed a negative state. This is still a form of reinforcement because it increases the probability that the behavior (exercising) will be repeated in the future. It is not “positive” reinforcement (delivering a positive reward; instead it is called “negative” reinforcement (removing a negative stimulus).

- Technically, punishment could be either positive (delivering something negative) or negative (removing something positive—like making someone pay money in fines). However, we do not use those terms much. Punishment is punishment—the opposite of reinforcement—whether it is taking away something positive or delivering something negative.



Now, let's consider this operant conditioning paradigm in terms of alcohol or other substance misuse.

- A person is offered cigarettes by peers and feels accepted by them (positive reinforcement) when joining them in smoking together. Result: more likely to smoke with friends in the future.
- A person drinks to the point of throwing up (punishment—applying a negative consequence). Result: less likely to drink to excess in the future.
- A person has to pay heavy fines and pay lawyers/legal fees for driving under the influence of marijuana (punishment—taking

away a positive). Result: may be less likely to drive under the influence in the future.

- A person feels nauseous with anxiety and finds that the anxiety and nausea go away when using cannabis (negative reinforcement). Result: may be more likely to use cannabis to dispel anxiety/nausea in the future.

This last example plays a role in what we learned about withdrawal symptoms and tendency to relapse (or at least slip) during recovery from SUD. A person whose body has come to depend on a substance like alcohol or heroin being



regularly administered will experience withdrawal symptoms if the substance is no longer used. Withdrawal symptoms are a very aversive (negative) experience which makes it a quite punishing consequence for quitting use—the person is less likely to maintain the “quit” behavior as a result. Then comes part two of the problem: negative reinforcement. If the person does resume use, even one slip, the punishing withdrawal symptoms momentarily subside—this consequence rewards using again. So, in operant conditioning terms we have two forces pushing for relapse as a result of withdrawal symptoms—the punishment for quitting that the withdrawal symptoms introduce, compounded by the negative reinforcement for using again. You can see why operant conditioning is so important both in the process of substance use becoming substance misuse or SUD and in the difficulty of recovery, as well.

**A little more about reinforcement paradigms.** While operant conditioning can make the story of substance misuse clearer, there do remain some complicating factors. These have to do with (1) consequence salience, (2) consequence timing, and (3) consequence sequencing.

*Salience.* A single reward or punishment may not mean the same thing to everyone—it may have different [salience](#) for different individuals. For example, M&Ms may be perfect rewards for some toddlers in potty training while other toddlers really do not care about candy; they are



better rewarded with smiley faces drawn in marker on their hands and knees. In training a new behavior, it is critically important to find the reinforcements that are most powerful for each individual. As noted in Module 3, heightened reward sensitivity in the adolescent brain might make the reinforcing aspect of drinking, vaping, or using cannabis more rewarding than for older individuals. Likewise, punishments may have different power (salience) for different individuals—charging fines may be more punishing to some than to others, for example. Or, for instance, nicotine withdrawal may be experienced more negatively by some individuals than by others, which has an impact on differences in their ability to cut down or quit smoking.

*Timing.* The strongest effects of reinforcement or punishment on learning and future behavior happen when the time lapse between the behavior and the consequence is very short. In Module 3 you learned that substances that get to the brain quickly through administration methods like inhaling, injecting, or “snorting” have a more powerful influence on the reward circuits than substances arriving through more delayed delivery routes (ingestion requiring digestion). In other words, the faster the substance arrives at the active sites in the brain, the stronger the reinforcement for using it.

On the other side of the timing issue, you may wonder why experiencing a hangover does not always lead to someone learning not to drink, or at least not drinking to excess. Unfortunately, the consequence (hangover) is delayed by many hours from the behavior (drinking). This time lag erodes (ruins) the power of the punishing consequence to be a strong influence on future behavior—“time is the enemy.”



(C) Anthony Ragusa, 2013

*Sequencing.* The other problem with relying on the punishing experience of hangover to influence future behavior is that it is not the first consequence experienced. The positive reinforcements associated with drinking being experienced first imbues them with more power to influence future behavior than the punishing consequences that arrive later. First “place” consequences are usually the winners.



*Negative attention.* One last point about learning theory warrants consideration. The social world around us is a rich source of positive reinforcement, negative reinforcement, and punishment. We would expect that exhibiting a behavior for which the consequence is social approval would likely be repeated—it was positively reinforced. We would expect that a behavior met with scolding would less likely be repeated—it was punished. However, we sometimes see an odd paradox with this latter example. Sometimes, any attention, positive or negative, is rewarding. Instead of a scolding being punishing, it could be reinforcing in some instances. Furthermore, sometimes when a behavior is ignored, the individual interprets the lack of punishing response to be a tacit approval of the behavior—which, in turn, means it is more likely to be repeated. Sometimes ignoring a behavior leads to its extinction. Other times ignoring a behavior leads to its encouragement.





## Social Learning Theory

Classical and operant conditioning theory are somewhat constrained by the necessity for the individuals to directly experience consequence in order for them to have reinforcing or punishing potential. Humans (and many other species) are also capable of learning through observing consequences to others. This is one critical addition from [social learning theory](#). For example, a person does not need to experience a fentanyl-influenced opioid overdose in order to develop concern about fentanyl contamination—witnessing this happening to someone else, or perhaps even learning second-hand about someone else’s experience—[observational learning](#)—can have an influence on their own drug-testing behavior (a harm reduction strategy). Observational learning plays a role in the development of [expectancies](#) discussed later in this module.

Many complex behaviors are learned through modeling and imitation—aspects of observational learning—rather than learning each individual element of the complex behavior one-at-a-time. For example, smoking a cigarette or e-cigarette (“vaping”) is a complex behavior—it involves engaging in a series of coordinated behavioral steps. Learning to do this is not “taught” one step at a time as in an instructional manual for assembling a toy or piece of furniture. It is learned as a behavioral sequence, typically through observation of behavioral models.



The experiments of Albert Bandura demonstrated the power of observational learning through imitation of behavioral models. Children not only learned and imitated specific acts of aggression toward a Bobo doll modelled for them (hitting, kicking, pushing), they learned to express the entire class of aggression toward the Bobo doll—aggressive behaviors that were not specifically modelled for them, like hitting it with another doll. Taking this to the substance use arena, consider a parent modeling alcohol use as a strategy for coping with stress. Children may not learn only to consider using alcohol under stressful circumstances, they may learn to use substances in general—the class of substance use/misuse behavior, beyond the specific drinking behavior. [If you are unfamiliar with Bandura’s Bobo doll aggression research, you might enjoy reviewing the 5-minute video available at [http://www.teachertube.com/viewVideo.php?video\\_id=131805](http://www.teachertube.com/viewVideo.php?video_id=131805) ].



Imitation of modeled behavior is a power mechanism of learning and socialization throughout the lifespan. **Social referencing** concerns a person who, in ambiguous or unfamiliar situations, relies on observing others’ behavior to know how to respond. We see social referencing in young children when they, together with a parent, are approached by a stranger: the child turns to watch and listen to the parent’s reaction to tell them how to interpret and respond in the situation. Social referencing may play a role in how individuals respond to substance-related situations—watching peers, for example, respond to someone offering alcohol or other substances in order to know how they might respond themselves.

Social referencing involves using the other person's behavior as a cue in interpreting a novel or ambiguous situation for oneself.

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Another important aspect of social learning theory concerns that concept of [salience](#), again. This time salience refers to the desirability or relevance of a specific model to the individual—this determines the likelihood of imitating that model. For example, an adolescent might find peers to be more salient models than they find teachers to be; parents remain salient for many adolescents and emerging adults but peers or other highly salient models may become more salient in certain situations. Salience of models might differ in terms of how much “alike” the observer feels they and the model might be—in terms of age, gender, sexual orientation, social status, or other “like me/not like me” variables. It also may differ in terms of how “desirable” (e.g., likeable, “cool,” popular, respected, successful, counter-culture/deviant, from my community) the model appears to the observer. Salience is always in the “eye of the beholder.” Knowing this about social learning theory helps us understand not only why someone might imitate substance use/misuse, but also why they might imitate NOT using/misusing substances. We generally are more likely to imitate salient behavior models—those we wish to be like—than to imitate other models.



These are reasons why adopting a “do as I say, not as I do” strategy is less effective than might be expected: learning is powerfully influenced through social learning principles like observational learning, imitation/modeling, and social referencing.

## Theory of Reasoned Behavior

In many areas of health psychology and health promotion, professional practices are based on theories of [reasoned behavior](#), rational choices and/or behavioral economics. In general, though this is grossly oversimplified, the theory is that individuals will make rational choices when faced with a set of behavioral options. In other words, a person will weigh the pros and cons, advantages and disadvantages, or costs and benefits of each choice before choosing to behave in a certain manner, selecting the option that is most advantageous (or least disadvantageous) among the available choices. A person will choose to engage in an addictive behavior, like substance use or gambling, if they perceive it will better meet a need than the other available options (McNeese & DiNitto, 2012).

In regards to the decision whether or not to use alcohol, cannabis, or some other substance, an individual would engage in an internal mental debate about the possible positive versus negative outcomes—feeling like part of the group using the substance and

positive feelings the substance might create would be weighed against the cost of getting the substances, what happens if your family finds out, possible legal ramifications, and so forth. Interventions from this theory base would be geared towards informing individuals about, and highlighting, the potential health (or other) risks associated with use of the substance(s). The assumption is that if they understand the risks they will make the “wise” decision not to engage in this behavior—the costs would outweigh the benefits. In addition, intervention might be geared toward helping the individual find other means of achieving the desired benefits at less risk/cost (e.g., getting the desired emotional response from exercise rather than substance use).



Unfortunately, we all know instances where someone (maybe even ourselves) made a choice that was not good for us—perhaps for no good reason at all. Perhaps they underestimated or misunderstood the risks/costs or the probability of the negative outcomes. Perhaps they decided the benefits outweighed the risks/costs despite the information provided to them. Or, perhaps, they were motivated by some other reasons to throw caution to the wind and made the disadvantageous decision anyway. The point is that individuals’ decision making does not always seem well-reasoned and rational.

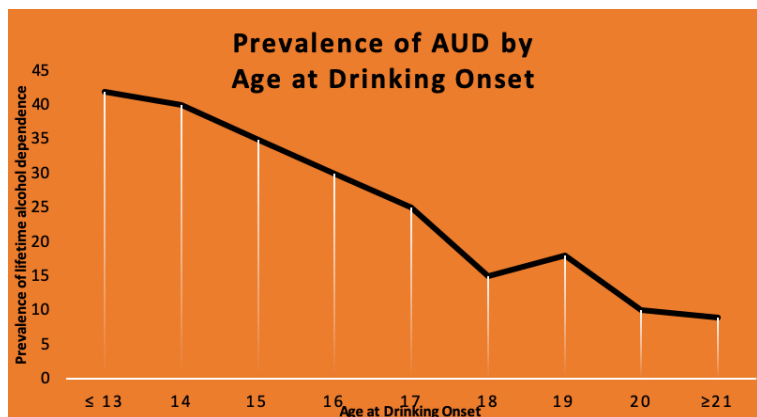
# Ch. 2: Developmental Theories

In recent years, a great deal of research, clinical, policy, and prevention attention has been directed to substance use among young adolescents, adolescents, and emerging adults. Not only do we care about the well-being of these young people in the here and now, while they are young, but because it has profound implications for their future lives, as well. This brings us to look at developmental theories of substance use and addiction.

Relatively recently, scholars have begun to argue for viewing substance use disorder within a developmental framework. Strong arguments are made for considering “the role of genetic, epigenetic, and neurobiological factors alongside experiences of adversity at key stages of development” in approaching the topic of addiction (McCrory & Mayes, 2015). This argument is informed, to a large extent, by evidence concerning the significant role played by adverse childhood events (ACEs) in the emergence of substance use, misuse, and use disorders—exposure to child neglect, child maltreatment, and substance misuse by parents/caregivers (McCrory & Mayes, 2015). For instance, adults who had experienced court-documented child victimization (physical abuse, sexual abuse, neglect) were about 1.5 times more likely to report using illicit substances (especially marijuana), using more types of illicit substances, and experiencing more substance use-related problems compared to adults without this childhood history (Widom, Marmorstein, & White, 2006). In another study, severity of self-reported exposure to childhood physical, sexual, and emotional abuse and other traumas were positively correlated with lifetime drug and alcohol use and this relationship was related to the individuals’ level of emotional dysregulation (Mandavia, et al., 2016).

Regardless of the root causes, it is important to consider developmental processes in substance misuse.

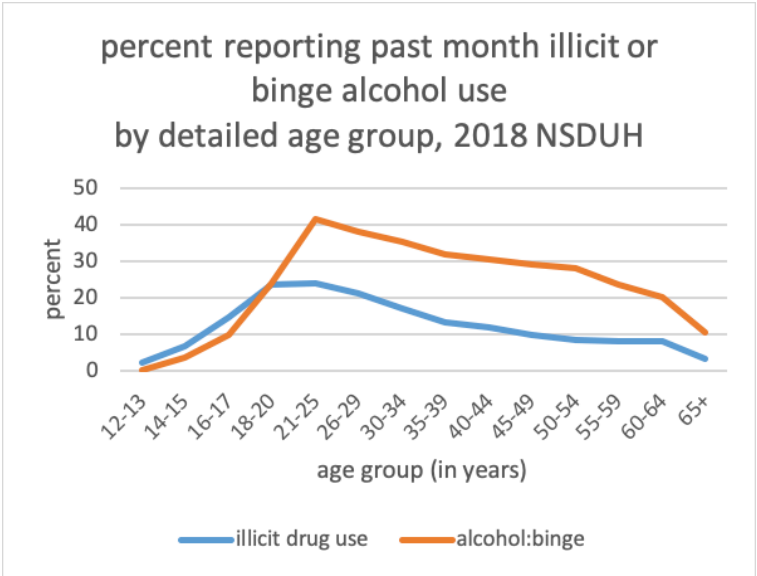
**Developmental trends data.** The following graph displays data from the 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). The data demonstrate a trend in which the younger a person is when beginning to drinking alcohol, the greater the likelihood of developing an alcohol use disorder at some point during that person’s lifetime. The greatest prevalence of alcohol dependence appeared among individuals who began drinking at or before age 13; the lowest prevalence of alcohol dependence appeared among individuals whose drinking began at or after age 21. You may recall from earlier modules that individuals who begin drinking before the age of 15 years are four times more likely to someday develop alcohol dependence than individuals who did not drink before the age of 21 years. Remember that for each year of age that the onset of drinking is delayed, the odds of developing alcohol dependence sometime in life decreases by 14%. This is a pretty important argument for prevention efforts that can help delay drinking onset! This also suggests that something important may be happening developmentally.



You may also recall that some of the impact is due to changes in

the developing brain that occur with exposure to alcohol during the adolescent and emerging adulthood years—this is a period of very rapid brain reorganization under normal developmental conditions so exposure to alcohol during this time may affect the brain more dramatically than alcohol exposure later in brain development. You also may recall that the adolescent brain is more sensitive to the rewarding/reinforcing experience of alcohol exposure than would be true if first exposure occurred later in life.

Consider also that substance use patterns are not consistent or linear in their changes with age, either. Data from the 2018 NSDUH study showed marked differences in substance use by young adults (aged 18-25) compared to younger and older individuals. With most substances, the numbers of individuals engaging in use or misuse increase from early adolescence through adolescence and emerging adulthood, then begin to decline again throughout most of the remaining adulthood period. Here is a graph created using the 2018 NSDUH data for past month illicit drug use by detailed age category:





Because these data are cross-sectional rather than longitudinal, we do not know if the use patterns for each individual followed this type of pattern, only that this pattern reflects the use at one point in time for the different groups. While it suggests a developmental trend, it does not confirm that such exists. For example, it is possible that the declining numbers may be at least partially attributable to attrition—individuals engaging in these behaviors over time may be less likely to survive to represent the later age groups.

**Developmental trends in behavioral control.** However, if the increasing rates during adolescence and early/emerging adulthood are reflective of a developmental trend, it is possible that the principle of [behavioral under-control](#) may be relevant. Thinking back to our biological models module, we learned that the adolescent brain undergoes dramatic developmental changes and functional revisions as part of normal development. The synaptic and myelination revisions do not occur evenly and concurrently throughout the brain. For example, the areas responsible for inhibitory control over behavior do not keep up with the same pace of change as areas responsible for initiating behavior. This explains why adolescents might behave more impulsively, exhibiting less inhibitory control over their behavioral choices—what might appear to be “poor judgment” at times. In other words, adolescents make under-controlled choices at a higher rate than they might have at a younger age or than they will at an older age (assuming that their choices do not prevent their achieving older ages). Thus, it is not surprising that we might see rates of under-controlled drinking behavior rising in this age group compared to other age groups. As the brain continues to mature, and behavioral control (inhibitory) areas catch up to behavior initiation areas, we may expect to see greater behavioral control (inhibition) exhibited. This concept of behavioral under-control as a developmental phenomenon could apply to substance use, aggression, and risk-taking behaviors in general.

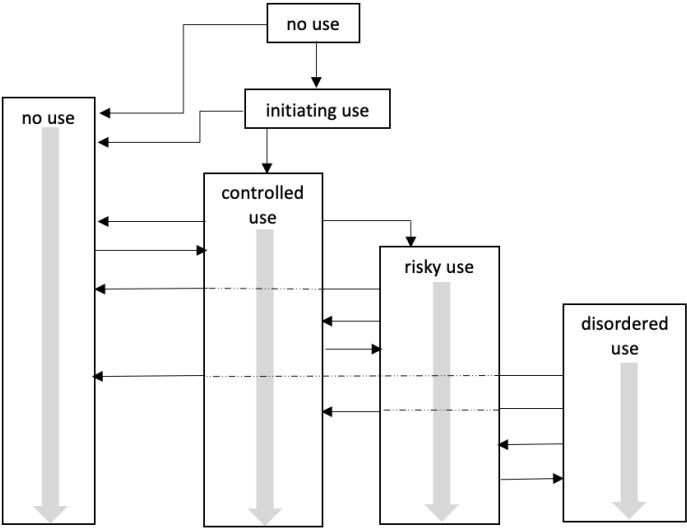
**Developmental trajectories of substance use disorder.**

During the 1950s and 1960s E. Morton Jellinek concluded that alcoholism follows a natural course over time, a course characterized by four qualitatively distinct stages: pre-alcoholic, early alcoholic, middle alcoholic, and late alcoholic (Jellinek, 1952). Despite many years of influence, Jellinek's developmental model has been criticized for being based on a small, select sample (of men in Alcoholic Anonymous programs), and because progressive worsening of symptoms is not universal (see Begun, *in press*): a great deal of clinical heterogeneity exists (Moss, Chen, & Yi, 2007). More recent studies demonstrated the dynamic, constantly changing nature of addictive behaviors: "Addiction can be viewed as a trajectory that emerges, becomes ingrained, and then in most cases evolves further (people quit or learn to control their use) over time" (Heather et al., 2018, p. 251). Yakhnich and Michael (2016) described the trajectory as a process beginning with occasional use of substances and ending with addiction, recognizing that many individuals "mature out" of excessive use at points along the trajectory.

A three-stage cycle of addiction related to the brain-behavior circuit has been offered as a model to consider (Koob & Volkow, 2010; White & Koob, *in press*). The first stage concerns substance use that progresses to binge and/or intoxication. This stage involves the acute reinforcing nature of psychoactive substances on reward systems of the brain. The second stage is called the withdrawal/negative affect stage. As the brain adapts to chronic substance exposure, withdrawal of the substances leaves a person fatigued and experiencing decreased mood, anxiety, stress-related symptoms, and possibly decreased motivation to earn natural rewards. The third stage in this model is a preoccupation/anticipation and craving stage. In this stage, "the individual reinstates drug-seeking behavior after abstinence" (Koob & Volkow, 2010, p. 225). Stress stimuli may heighten the effect. The three-stage model is used to explain what happens when individuals progress to a state of addiction. Not everyone progresses through these stages,

however, just as not everyone progresses from substance use to substance use disorder.

A 60-year longitudinal study of college-aged men whose drinking patterns were identified as “alcoholism” demonstrated widely varied patterns in later adulthood, including stable abstinence, non-problematic/controlled drinking, alcohol abuse, or death (Vaillant, 2003). A typical substance misuse trajectory begins during adolescence or emerging adulthood, declines or escalates during emerging and early adulthood—where it may or may not meet criteria for a substance use disorder—then either declines or extends into adulthood, possibly but not necessarily meeting criteria as a substance use disorder (see figure below, from Begun, *in press*).



Important aspects of this figure are the multiple pathways/trajectories that occur and the iterative nature of the possible trajectories: for example, moving back and forth between

controlled, risky, disordered drinking, and no alcohol use. The probability of different trajectories is affected by a host of individual-specific factors, as well as the “addictive potential” of different substances involved (Upah, Jacob, & Price, 2015) and individuals’ different histories of change attempts over the life course (Begun, Berger, & Salm-Ward, 2011). Similarly, no single, “natural” trajectory to/through recovery exists and there are a multitude of addiction “careers” in individuals’ relationships or involvement with substances over their lifetimes following the emergence of a substance use disorder (DiClemente, 2006).

Multiple factors play a role in “positive outcome” trajectories, including engaging in treatment—but treatment is not a requirement. For example, U.S. combat veterans who experienced both posttraumatic stress disorder and hazardous drinking behavior were less likely to continue hazardous drinking if they had engaged in alcohol-specific treatment, despite persistent/unremitting PTSD symptoms, and particularly if their drinking had led to negative consequences (Possemato et al., 2017). But the field also recognizes “natural” recovery as a studied phenomenon whereby many individuals change their problematic alcohol or other substance use without engaging with formal treatment systems (DiClemente, 2006; Sobell, Ellingstad, & Sobell, 2000), or by combining formal, informal, and natural recovery systems in their change efforts (Begun, Berger, & Salm-Ward, 2011). Surprisingly, this even included a cohort of veterans returning from Viet Nam with heroin use disorders (Robins, 1993).



### **STOP & THINK**

What does it mean to talk about a “trajectory” of a substance use disorder?

How does this kind of developmental thinking inform prevention and intervention strategies?

How would you explain to a group of 12 year olds why it matters not to initiate alcohol, tobacco, or other substance use at least until you are an adult?

# Ch. 3: Theories of the Psyche

The next set of theories to consider can be loosely grouped together under the heading of theories of the psyche—capturing the essence of who a person “is.” Under this heading, we consider psychodynamic, attachment, personality, and psychopathology theories related to substance misuse and substance use disorder. These represent some of the historically earliest psychological models used to explain the phenomenon of addiction.

## Psychodynamic Theory

In a [psychodynamic theory](#) interpretation, addiction is not viewed as being a disease in and of itself but as a *symptom* of intra-psycho conflict, unresolved psychological tension, or psychological turmoil. On one hand, a person

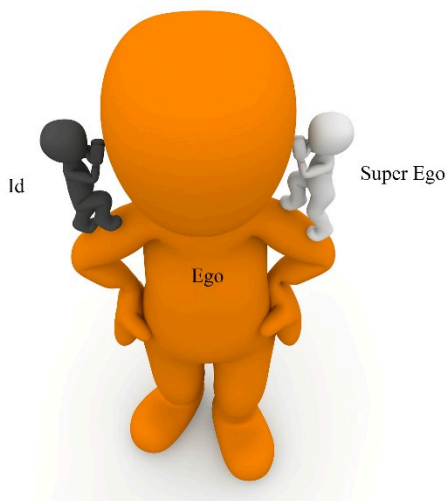


may experience urges to express emotions by behaving in ways that might not be socially acceptable. The urge to handle frustration or anger through aggression and violence are examples of this side of the equation, born in the primal aspects of personality (called the Id). The Id is not just negative, it includes positive feelings, too—think of a really young puppy as a ball of Id—it acts as it feels, positively or negatively, totally in the moment, with no filter, no restraint.

On the other hand, over time and through repeated learning encounters with the physical and social world, a person (and hopefully puppies) develop enough experience to understand and appreciate that acting aggressively or violently is not socially

acceptable and that this behavior is a poor choice. In other words, the super-ego has stepped in to editorialize about the Id response to emotions. This is where sentiments like guilt and shame come into play, helping reign in socially unacceptable behavior choices.

The ego, which develops over time through experience, learning, and social learning, becomes the manager. The ego is faced with the challenge of serving as a referee between strong “act” urges coming from the Id and strong “inhibit” pressures from the Super Ego. As a result, the ego can create appropriate balance between pleasure and control, where emotions and urges are expressed in acceptable ways. The ego also helps prevent someone from acting unwisely or in an unsafe manner.



In this psychoanalytic or psychodynamic model, a person may resolve some of this Id-Superego tension by using alcohol or other drugs for their ability either to “numb” feelings that are triggering the Id response or to silence the super-ego, put it to sleep, thereby removing the unpleasant, tension-filled experience of conflict. Sometimes individuals in conflict feel the need to quiet the “voices” that are always “yelling” in their minds. Additionally, psychoanalytic or psychodynamic theory might suggest that an individual who has experienced trauma might use substances as a means of “numbing”

the powerful negative feelings experienced as a result of reminders of the past trauma experience. This is not the only way the theory has been applied to substance use, however.

**Orality.** Yet another psychoanalytic interpretation of addiction, particularly for cigarette smoking and drinking alcohol, is one related to the concept of oral fixation.



A normal part of infant development involves exploring the world orally, through the taste and touch sensations of the mouth. In psychoanalytic theory, it is part of the normative course of development that a person's libidinal energies become localized at a specific zone of the body at different periods of development. Libido does not only refer to a person's sexual drive—this is true during the developmental period when the libidinal energy localizes in the genital zone.

Earlier in development these libidinal energies localize in the oral zone—the mouth and mouth parts. Stimulation of the oral zone feels good because it relieves the tension in that area caused by the localized libido. Orality is a period of infancy—we expect to see babies using their mouths to explore the world.



According to psychoanalytic theory, if something goes wrong with development at this early orality phase of development then a portion of libidinal energy becomes “stuck” in the oral zone. The person will spend a lifetime trying to satisfy their need for oral stimulation—putting things in the mouth, chewing, or sucking on things. In theory, a need to smoke cigarettes, hookah, e-cigarettes, or cigars—putting them in the mouth and all the ritual that goes into smoking them—and maybe a need to drink alcohol, could represent efforts to curb demands from the trapped libido. Logically, then, a person should be able to substitute one oral tool for another—in other words, chewing gum or drinking from water bottles should resolve a “need” to smoke or to drink alcohol. It is not so simple, though—the tool in the form of cigarettes, hookah, e-cigarettes (vaping) or alcohol comes to cause some needs of its own.

## Attachment Theory

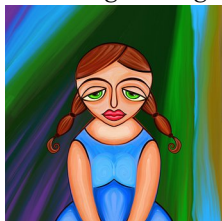
An [attachment theory](#) of addiction is not far removed from psychoanalytic and psychodynamic theory explanations. As explained in the early works of John Bowlby, infants and young children, in the normative course of development, form attachment relationships with others central to their physical and emotional survival—parents, siblings, caregivers, pets, and even special “transitional objects” (like a blankie or stuffed animal). These psychological attachments allow someone to have the sense of security in a great big, unpredictable world. Within these attachment relationships, individuals begin to make sense of their social world.

Sometimes, attachment relationships are disrupted or dysfunctional. They either fail to form, are broken once formed, or develop as insecure and unstable attachments. According to attachment theory, a person experiencing attachment issues is likely to experience significant “holes” in their emotional and

personality development. The world does not seem like a safe, predictable, reliable place to exist, nor are there safe, predictable people on whom the person can rely. Their understanding of and relationship to the world is likely to have significant gaps.

Sometimes these individuals describe themselves as being “full of emptiness.”

As in the case of the psychoanalytic model, this person may come to rely on drugs or alcohol as a means of coping with these gaps, and the associated negative feelings and sense of detachment. It might “numb” the psychic pain for them. The drinking or drug-taking social environment itself may become what they use to fill the emptiness—it is not necessarily the alcohol or drugs at first, but the drinking or drug-taking situations that start the pattern.



Based on these models, the type of intervention that we have available involves attempting to address the root psychic conflicts or deficits and repair the damage to the psyche. Here we are going to try to help the person become whole, to find a way to resolve their internal conflicts and become whole or to fill the empty void and become whole. This is the therapeutic goal of many forms of psychotherapy. The preventive strategy is to help create environments during early infant, child, and adolescent development that nurture the person and help them develop healthy super ego and ego strengths. Furthermore, throughout the life cycle, prevention involves avoiding the disruption of attachment relationships and exposure to traumatizing experiences.

## Self-Medication Theory

The [self-medication theory](#) has, in part, been explained in our discussion of psychodynamic and attachment theory. As discussed, an individual may choose to use substances to quiet psychic conflict, fill emotional emptiness, and/or escape the emotional aftermath of trauma. One thing known about the population who misuse alcohol or other substances is that the incidence of their having experienced injury, trauma, or abuse is much higher compared to the rest of the population. For example, in a study of Vietnam veterans, among individuals meeting criteria for post-traumatic stress disorder (PTSD), 73% also met substance use disorder criteria (Kulka, et al., 1990). Among these veterans, men with PTSD were two times more likely and women with PTSD were five times more likely to also experience a substance use disorder than were their counterparts without PTSD.

Among civilian populations, the experience of trauma is often associated with substance use disorder, particularly among women: in one United Kingdom study, among 146 women engaging in substance misuse, 90% had experienced trauma in the form of intimate partner violence, traumatic grief, sexual abuse, physical abuse, bullying, or neglect (Husain, Moosa, & Khan, 2016). In an Australian sociological study of youth and substance abuse, initiation of substance use was associated with childhood trauma, leaving school (dropout), separation from family, and homelessness, as well as unemployment (Daley, 2016), and a great deal of evidence relates adverse childhood events (ACES) with substance misuse and substance use disorders, as well (Sartor et al., 2018).

***“My whole life went downhill. I was abused, and used alcohol to escape the pain. I became horrible to myself and everyone around me. I honestly didn’t care what happened anymore”*** (quoted in Najavits, 2009, p. 290).

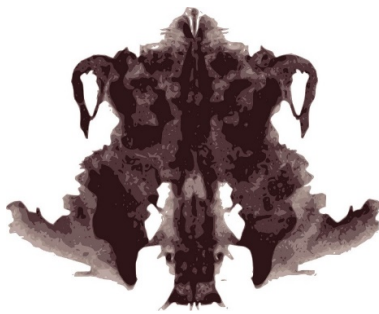
Self-medication theory is somewhat controversial. The prior

examples do not demonstrate a causal relationship whereby self-medication theory is proven; the theory remains a possible explanation for at least some of the co-occurrence. Sometimes trauma events precede substance misuse. Other times, traumatic events occur during a period of substance misuse or after substance misuse was initiated. Self-medication may have more to do with “treating” physical pain from injury or chronic illness than managing psychic or emotional pain. In addition, individuals may use substances to self-medicate a host of other mental health concerns—attention deficit disorder, anxiety, depression, or stress, for example. Clinicians often encounter individuals experiencing substance use problems who have one or another form of chronic pain or depression or anxiety or attention deficit disorders with or without hyperactivity or other problems they believe the substance use can relieve. While this might be a reason why some individuals *initiate* use of one or more substances, it may not explain how the substance use becomes substance use disorder. There are other reasons why individuals initiate substance use, and evidence on this theory is mixed.

A scholar named Lisa Najavits was one of the first to develop intervention approaches specifically designed in an integrated manner to address trauma experiences and substance abuse. She published a book called *Seeking Safety* that is used today as the basis of programs all over the world. How does this relate to the self-medication theory? Since many individuals who misuse alcohol, illicit drugs, or prescription drugs may be attempting to “treat” their own physical and/or psychological pain, finding healthful strategies for doing so might facilitate recovery from substance misuse and substance use disorder. As much as the classic quote about a self-treating physician having a fool for a patient may be true, how more true could it be when individuals in the general population are self-medicating?

# Personality and Psychopathology Theory

Past clinical literature discusses a phenomenon called the “addictive” personality. This concept presumes the existence of a constellation of specific personality traits characterizing individuals who develop substance use disorders (or addiction). In theory, these individuals are predisposed to develop a substance use disorder (or addiction) by virtue of possessing these personality traits—in much the same way genetics may predispose someone to develop a substance use disorder. The question becomes: is there such a thing as an “addictive” personality?



These days, the idea of an addictive personality is considered somewhat dated as it is not well supported by evidence. While there exist some traits or characteristics commonly observed among groups of individuals who experience substance use

disorders, the evidence does not support there being a universal set of personality traits or personality type associated with addiction/substance use disorders. Evidence for the existence of an “addictive personality” type does not exist (per Szalavitz, 2016 citing an interview with George Koob, director of the National Institute on Alcohol Abuse and Alcoholism).

What we know is that pretty much any person can become addicted to something if the right (or, in this case the wrong) circumstances come together. Some individuals may be more vulnerable or at a higher risk of addiction to certain substances, but the potential exists for anyone depending on circumstances. We also know that the circumstances vary somewhat for different types of substances—the vulnerability and risk for developing alcohol use

disorder is not the same as for developing addiction to nicotine or cocaine or opioids or cannabis.

On the other hand, some personality traits or characteristics are shared by many (not all) persons experiencing a substance use disorder/addiction. For example, in her book challenging the addictive personality, Szalavitz (2016) reported research concluding that 18% of persons experiencing an addiction also exhibited “a personality disorder characterized by lying, stealing, lack of conscience, and manipulative antisocial behavior” and that this 18% rate was more than four times the rate observed in the general population. However, arguing against this being the hallmark of an addictive personality are the observations that (1) this leaves 82% of individuals experiencing addiction not expressing this personality disorder and (2) individuals with this personality disorder do not all develop addiction. In other words, the person experiencing addiction is not a separate type of person from the rest of the population. This kind of result is common across many studies of addictive personality traits—the population of individuals experiencing addiction/substance use disorders is tremendously diverse and heterogeneous across many demographic, personal history, and personality factors.

There exists some evidence to suggest that certain temperament or personality characteristics are associated (correlated) with a higher probability of *initiating* substance use, especially early initiation of alcohol or tobacco use during adolescence. For example, studies emphasize the increased odds of using/misusing substances among adolescents who have angry-defiant personality types, as well as the “thrill seeker” personality type (sometimes called the “Type T personality”). Or, evidence indicates that “young people diagnosed with conduct disorders and other oppositional disorders are also at higher risk for developing substance use disorders in adolescence and early adulthood,” as is also true of individuals with bipolar and major depressive mood disorders (Cavaiola, 2009, p. 721).



Again, these personality and psychopathology traits are shared by individuals who develop and do not develop addiction or substance use disorders—they are not traits specific to addiction. Furthermore, it is difficult to determine where the behaviors (e.g., antisocial) preceded the addiction and where the addiction preceded the behaviors—meaning that the trait is not a cause of the addiction but a consequence (Cavaola, 2009).

As a result of newer research methods and ways of analyzing data, some of the earlier correlational studies of personality traits have fallen out of favor. Thus, there is less emphasis these days on personality theory and theories of an addictive personality. In a way, this is a positive development because personality theory leaves very little in the way of intervention tools: personality traits are very resistant to change!





*An interactive H5P element has been excluded from this version of the text. You can view it online here:*

[https://ohiostate.pressbooks.pub/  
swk5805coursebook/?p=136#h5p-44](https://ohiostate.pressbooks.pub/swk5805coursebook/?p=136#h5p-44)



# Ch. 4: Expectancies & Cravings

As a part of the cognitive framework concerning the initiation of substance use/misuse, we can look at the kinds of [expectancies](#) individuals might hold concerning the likely outcomes or effects associated with substance use—what using alcohol or other drugs will do to or for them. To understand continued use of substances over time, particularly when someone experiences the urge to use substances despite consciously not wanting to do so, it is important to look at the psychological phenomenon of [cravings](#).

## Expectancies

Expectancies act as a filter in the appraisals individuals make when faced with a substance use opportunity (stimulus) and their behavioral response. An expectancies process diagram is very similar to what we saw in relation to the cognitive behavioral process; the major difference being that expectancies become part of the interpretation step. What a person has come to expect as the likely outcomes of the behavior becomes part of the interpretation.



Children develop expectancies about alcohol at a very early age—even preschool/kindergarten aged children may already have

developed ideas about the emotional effects of adults' drinking (Kuntsche & Kuntsche, 2018). One source of their expectancies was parental drinking: sons identified positive emotional consequences (e.g., feeling happy, calm, relaxed) if a parent engaged in moderate drinking and they identified negative emotional consequences (e.g., feeling angry, sad, depressed) if a parent engaged in heavy drinking; the effects were less consistent among daughters and were stronger when the parent was the father rather than the mother (Kuntsche & Kuntsche, 2018).

You may find it interesting to see what 8<sup>th</sup>, 10<sup>th</sup>, and 12<sup>th</sup> graders in the U.S. hold as expectancies concerning different substances—and that these expectancies relate to substance use behavior. These data were generated in the annual Monitoring the Future study during 2018 and ask in relation to various substance-related behaviors, “How much do you think people risk harming themselves (physically or in other ways), if they...”; presented here are the percentages responding with “great risk” (<https://www.src.isr.umich.edu/projects/monitoring-the-future-drug-use-and-lifestyles-of-american-youth-mtf/>).

Behavior	8 <sup>th</sup> grade	10 <sup>th</sup> grade	12 <sup>th</sup> grade
try marijuana once or twice	20.3	13.9	12.1
smoke marijuana occasionally	32.1	21.4	14.3
smoke marijuana regularly	52.9	38.1	26.7
try inhalants once or twice	29.6	38.6	—
take inhalants regularly	46.8	57.6	—
take LSD once or twice	20.8	33.8	29.0
take LSD regularly	36.4	54.1	55.2
try cocaine powder once or twice	42.6	52.6	47.9
take cocaine powder occasionally	61.0	70.2	62.1
try heroin once or twice ( <i>without using a needle</i> )	59.5	71.4	63.1
take heroin occasionally ( <i>without using a needle</i> )	72.1	81.0	69.6
try one or two drinks of an alcohol beverage (beer, wine, liquor)	13.6	13.0	10.2
take one or two drinks nearly every day	28.7	30.3	22.8
have five or more drinks once or twice each weekend	52.3	51.8	59.1
smoke one to five cigarettes per day	40.8	49.9	—
smoke one or more packs of cigarettes per day	61.3	69.6	73.9

vape an e-liquid with nicotine occasionally	16.9	17.9	15.8
vape an e-liquid with nicotine regularly	32.4	31.3	27.7

Expectancies, at least those related to alcohol use, do not remain consistent over time. During early adolescence, negative alcohol expectancies tend to diminish while positive expectancies tend to increase (Smit et al., 2018), and positive alcohol expectancies tend to become more stable with progressing age (Wardell & Read, 2013). This is important because alcohol expectancies are predictive of alcohol use initiation, as well as drinking behavior over time (Smit et al., 2018). Among college students, those who held strong positive expectancies about binge drinking (sociability and sexuality) were more likely to engage in binge drinking than students whose positive expectancies endorsement was weaker (McBride et al., 2014).

Besides parental substance use, where do alcohol and other substance use expectancies come from? In some cases, expectancies come from a person's own direct experiences. In others, expectancies emerge from observational learning. Observational learning, especially among children, involves fictional as well as real-world models. For example, consider the scene in the original cartoon Disney movie *Dumbo* where the little elephant gets a big drink of liquor and sees dancing pink elephants on parade. An expectancy might be that alcohol makes you see the world in interesting new ways, or it may seem scary and creepy, depending on the emotions prompted by viewing this scene.



From what individuals see in their homes, neighborhoods, schools

and jobs, media, and social media they develop expectancies about alcohol, drugs, sex, smoking, gambling, and many other types of behavior. These expectancies may influence how situations are appraised and interpreted, which in turn influences choices and behavioral responses. If the expectancy is that using a particular substance will make you feel good/better, substance use is likely to be appraised as a good solution to a bad day, a bad break up, or receiving bad news. If the expectancy is that using these substances will just delay the day of reckoning, and maybe let the problem get worse with time, or that it will make you feel low and depressed, then substance use is likely to be appraised as a bad idea.

## Cravings

As previously discussed, internal and environmental cues can become craving triggers through classical conditioning processes, with exposure to those triggering cues increasing the risk of using substances again. This is called a **cue-induced response**. Cues or “triggers” may involve any combination of the five senses (sight, sound, taste, feel, and smell) or internal states (e.g., anxiety, loneliness, boredom, depression, mania). For example, one woman in treatment for a substance use disorder described loud rock music as a personal trigger for her craving to use alcohol and marijuana because she “learned” to enjoy these substances at rock concerts. Regardless of its nature, craving cues trigger “abnormally strong desires to engage in addictive behaviours,” though not necessarily leading to subsequent use (Heather, 2017, p. 32). One skill addressed in cognitive-based therapies is for individuals to learn to identify their own personal triggers or cues and develop strategies to (1) avoid potentially triggering situations, and (2) respond differently to them when they cannot be avoided. For example, someone might rehearse a series of coping skills, such as relaxation or mindfulness practices, to employ when cravings occur, as a means of

interrupting the “old” behavioral response (called coping skills training, or CST). Cue-exposure treatment is a type of behavioral therapy that involves systematic desensitization to learned cues as a means of reducing the degree to which someone reacts to the triggering stimuli/cues (Monti & Rohsenow, 1999). While this alone may not be sufficient for someone to break the cue-induced response, and the intervention must be delivered very carefully in order not to actually trigger a relapse, this kind of intervention may help decrease an individual’s response to the cues to the point where they can focus on applying their other coping skills.



Think about your personal attitude about getting drunk on alcohol or high on cannabis. What factors in your past and present environment, experiences, and observations contributed to your favorable, unfavorable, and ambivalent attitudes?

Think about the environments and experiences that you have in a typical day. What among them might create an experience of craving for a person in recovery from alcohol or other substance misuse/use disorder? How might a person avoid these kinds of trigger events?

# Ch. 5: Summary

In this module about the second aspect of a biopsychosocial framework, you learned a great deal about what goes on in the minds of individuals considering or engaging in substance use, substance misuse, or experiencing substance use disorder. As a result, you should now be able to explain how cognition, information processing, learning, social learning, rational/planned behavior, developmental, psychodynamic, attachment, self-medication, personality, psychopathology, expectancies, and cravings models relate to substance use, misuse, and use disorders. In discussing many of these theories and models, the relationship between brain and mind was evidenced again (first visited in our earlier biological models module), and linkages were drawn to how the theories and models inform interventions to prevent, treat, and support recovery from substance misuse and substance use disorders. At this point, you are well equipped to explore the final dimension of the biopsychosocial framework: social contexts and physical environments. That is the topic of our next learning module.

# Module 4: Key Terms

**affect** concerns a person's emotions and feelings.

**alcohol myopia** concerns the way a person might focus on immediate circumstances and events rather than placing them in a broader or longer-term context—becoming “nearsighted” in a situation—when alcohol has been consumed; this interferes with reasonable, accurate interpretation of what is happening.

**attachment theory**, as related to an addictive behavior, concerns the role played by dysfunctional attachments or dysfunctional responses to the disruption of positive attachments during the course of human development.

**behavioral under-control** refers to the observation that inhibitory “control” areas/functions of the brain may not be as developed or active as the behavior initiation “action” areas/functions, leading to what appears as impulsiveness, “recklessness,” or high-risk behavior.

**classical conditioning** refers to a learning principle involving the pairing of stimuli whereby a previously neutral stimulus becomes paired with a naturally potent (unconditioned) stimulus such that it elicits the same response (conditioned stimulus).

**cognition** concerns the mental processes involved in a person's knowledge, thoughts, and understanding of their experiences.

**cognitive behavioral therapy (CBT)** includes a class of intervention approaches designed to address a person's cognitive processes as means of changing behavior.

**cognitive processes** concern the link between what a person perceives and how a person responds (behaves)—the important role of situational interpretation.

**craving** refers to an intense, compelling desire to engage in an addictive behavior (e.g., repeated substance use) experienced by someone who has learned positive associations with that behavior; craving triggers may be external cues or internal states.



**expectancies** are cognitions about the likely consequences or outcomes of behaving in a certain manner, with these cognitions having an influence on behavioral choices.

**information-processing** concerns the way that individuals take in (perceive), organize, store (memory), and retrieve information.

**negative reinforcement** a behavioral consequence that involves removing or relieving a negative state such that the behavior is more likely to be repeated in the future (reinforced).

**observational learning** refers to the social learning theory process of learning through either imitating a behavioral model, teaching through modeling, or observing the consequences a model experiences as a result of behaving in a certain manner.

**operant conditioning** is a learning process whereby the consequences of a behavior determine the likelihood of repeating that behavior in the future (positive reinforcement, negative reinforcement increasing the probability, punishment decreasing the probability).

**positive reinforcement** is a behavioral consequence that involves providing a favorable outcome such that the behavior is more likely to be repeated in the future (reinforced).

**psychodynamic theory** explains dysfunctional behavior as a symptom of internal conflict between id, ego, and superego functions, or as an effort to resolve discomfort and stress associated with libido (libidinal energy) that has become fixed in different body locations (e.g., oral or genital) as the result of developmentally disruptive or traumatic experiences.

**punishment** is a behavioral consequence that involves providing an unfavorable outcome such that the behavior is less likely to be repeated in the future.

**reasoned behavior** refers to the tendency of individuals to calculate costs/benefits associated with a behavioral choice with the results of the analysis influencing the choices made.

**salience** refers to how significant or meaningful a consequence or role model might be for a particular individual.

**self-medication theory** reflects a belief that individuals may use

alcohol or other substances as a (potentially harmful or dysfunctional) means of “treating” physical, emotional, or psychic pain.

**social learning theory** is an expansion on learning theory that invokes principles of observing others’ behavior and the consequences of others’ behavior such that these observations influence the observer’s learned behavior.

**social referencing** is a social learning theory construct whereby an individual makes sense of an ambiguous situation by watching how others interpret, react, or respond to the situation.

**state-dependent learning** addresses the tendency for information to be more easily retrieved under conditions similar to when/where/how it was initially gained.

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## PART V

# MODULE 5: SOCIAL CONTEXT & PHYSICAL ENVIRONMENT MODELS OF SUBSTANCE MISUSE

The social context represents one of three pillars in the biopsychosocial framework critical to social work and several other disciplines/professions. Contributing to a biopsychosocial understanding of how substance use, misuse, and use disorders develop, are maintained, and change are the various social and physical environments individuals are exposed to—contexts that can be protective against or predisposing toward substance misuse (Begun, Bares, & Chartier, *in press*; Kendler & Eaves, 1986). In social work, this is partially reflected in adopting a person-in-environment perspective in which an individual's development and behavior is understood only when the individual is considered within the social and physical environmental contexts. Reading for this module introduces concepts essential for understanding many of the social context and physical environment factors believed to play a role in substance use and misuse, as well as recovery from substance use disorder. This online course book includes content that both informed and was informed by the work of Begun, Bares, and Chartier (*in press*).

## Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Explain how social contexts and physical environments influence substance use, substance misuse, substance use disorders, prevention, and recovery;
- Describe the relevance of gene x environment interactions;
- Identify components of the social-ecological model as they relate to substance misuse;
- Describe how social norms, stigma, and microaggression experiences influence substance use, misuse, treatment engagement, and recovery processes;
- Identify social structure models and factors that help explain substance use and misuse and inform intervention/prevention/recovery efforts (e.g., through culture and subculture, labeling theory, deviance, the impact of “isms,” and policy);
- Explain how family systems, peers, and significant others are involved in substance misuse, substance use disorder, and recovery processes;
- Define key terms related to social contexts and physical environments in substance misuse.



# Ch. 1: Social Contexts and Physical Environments

This chapter presents a general overview of theories/models concerning the role of [social contexts](#) and [physical environments](#) in substance misuse and opportunities for prevention or treatment. These are often referred to as [sociocultural theories](#), but that label does not provide sufficient emphasis about the role of physical environments. Here we are concerned with social systems and social structures, physical environments, social norms, culture and subculture, and the impact of “isms” and labeling theory. Evidence points to many relevant social and environmental factors that play a role, such as:

- Stigma
- Policy and global forces
- Family and family system dynamics
- Peer groups
- School and workplace
- Neighborhood and community

## Stigma

Social [stigma](#) refers to negative social attitudes or stereotypes about a type of person or behavior (Begun, Bares, & Chartier, *in press*). Stigma about persons who engage in substance use or substance misuse, experience a substance use disorder, seek treatment for substance-related problems, or are in recovery has an impact on their opportunities and experiences. The stigma could stem from their own beliefs about what they are doing, attitudes expressed by individuals in their immediate social contexts,

attitudes encountered in their interactions with professionals, and/or attitudes and opportunities (or lack of opportunities) expressed through policies. Stigma affects a person's willingness to engage in treatment, which then can translate into further marginalization, blame, and increased barriers to seeking help for substance misuse and related problems (Kulesza et al., 2016). "Explicit bias refers to the beliefs, attitudes, and social norms of which someone is conscious and aware, whereas implicit bias reflects those lying outside of conscious awareness and intentional control; explicit and implicit bias may not fully align even within the same person's belief systems" (Begun, Bares, & Chartier, *in press*). For example, explicitly expressing a belief that someone engaged in injection substance misuse is more deserving of treatment help than punishment as a criminal might not be consistent with what is held as a belief at the implicit level (Kulesza et al., 2016).

Persons experiencing substance use disorders regularly encounter stigma that profoundly impacts their everyday lives (Fraser et al., 2017). For example, they may encounter stigmatized attitudes when they seek health care—either being “blamed” for health conditions related to their substance use or “accused” of deceptively seeking drugs from the healthcare system. Stigma often informs policy at the organizational, local, state, federal, and international levels, as well. Comparing vignettes of successfully treated and untreated addiction led to the conclusion that, since portraying successful treatment was followed by a greater belief in the effectiveness of treatment and less willingness to discriminate against persons experiencing drug addiction, stigma could be reduced through media campaigns and public education (McGinty, Goldman, Pescosolido, & Barry, 2015)—messages along the lines of SAMHSA's message: “prevention works, treatment is effective, and people recover from mental and/or substance use disorders” (<https://www.samhsa.gov/find-help/recovery>).

**Treatment Works. Recovery Happens.**

## Policy as a Context Influence

Social, public, and health policy are tools for influencing outcomes by manipulating the social and physical contexts in which individuals live, develop, and function (Begun, Bares, & Chartier, *in press*). For example, state and federal policies that increased the legal drinking age (Wagenaar & Toomey, 2002), specified the age for legally obtaining tobacco products (Schneider et al., 2016), and established a uniform blood alcohol level (BAL, or blood alcohol concentration, BAC) for intoxicated operation of a motor vehicle are social control actions to influence substance use behavior at the individual level. Lack of social control is also a factor: when first introduced, electronic cigarettes (e-cigarettes, vaping) were not regulated as tobacco products, allowing legal access and use by adolescents who could not legally purchase combustible cigarettes (Cobb, Byron, Abrams, & Shields, 2010). Adolescent e-cigarette use was subsequently related to higher rates of tobacco use (Wills et al., 2017).

Policy restrictions related to advertising of psychoactive substances such as alcohol, tobacco, vaping products, and cannabis/marijuana potentially affect the physical environments in which individuals make choices about substance use. For example, where tobacco advertising appeared in greater numbers, use by young people too young to legally purchase these products nevertheless was increased (Kirchner et al., 2015). Policy can influence substance use patterns through affordability mediated by taxation. Use of tobacco products has a demonstrated relationship to states' taxation rates (Luke, Stamatakis, & Brownson, 2000); alcohol use has similarly been shown to be tax-rate sensitive. Use of tobacco is also related to the density of retail outlets that sell tobacco; density is highly sensitive to local and state policy (Cantrell et al., 2015; Novak, Reardon, Raudenbush, & Buka, 2006).

## Physical Environments

An obvious physical environment aspect important to consider has to do with a person's access to alcohol or other drugs. In general, the physical environment produces opportunities and obstacles that shape the behavior of people living in those spaces and places. For example, the nutritional value of a person's diet is influenced by living in a "food desert" or other conditions of food insecurity versus where healthful foods are easily accessed and affordable. Specific to substance use, consider how difficult or easy it is for someone to gain access to alcohol, tobacco, or other substances in the family home, school, workplace, peer group, or neighborhood. One set of questions tracked over time in the U.S. national survey of middle and high school students called *Monitoring the Future* (Miech et al, 2018) concerns how easy or difficult students believe it is to obtain various substances. As you can see from Table 1, belief in easy access to each of the different substances increased from 8th to 10th to 12th grade.

Table 1. Percent of students responding "fairly" or "very" easy to obtain substances, created from *Monitoring the Future* data 2018, retrieved from <http://www.monitoringthefuture.org/data/data.html>

substance	8th graders	10th graders	12th graders
alcohol	53.9	70.6	85.5
cigarettes	45.7	61.5	75.1
marijuana	35.0	64.5	79.7
vaping device	45.7	66.6	80.5
e-liquid nicotine	37.9	60.4	77.2
LSD	6.5	14.9	28.0
heroin	7.8	9.7	18.4
other narcotics	8.3	16.8	32.5
cocaine	9.8	14.7	23.0
steroids	10.9	14.5	21.1

Access to substances is not the only mechanism through which the physical environment influences substance use and misuse at the individual level. Investigators secondarily analyzing data from large-scale surveys concluded that living in a neighborhood with more opportunities for adolescents to engage in substance use had several effects (Zimmerman & Farrell, 2017):

- detrimental effects of parental substance use/misuse were amplified in the youths' risk;
- detrimental effects of peers' substance use were amplified in the youths' risk;
- protective effects of the youths' perceptions of harmfulness from substance use were diminished.

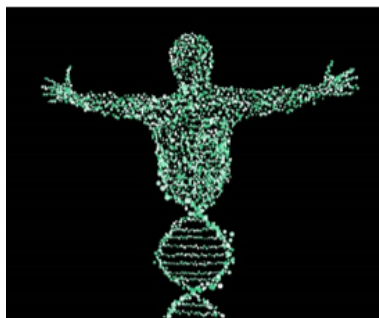
Additionally, the physical and social settings where substance use occurs have an impact on substance use behavior. Among college students, drinking setting was observed to make a difference in drinking behavior (Clapp et al., 2006). Many other patrons or party-goers being intoxicated, drinking games, and illicit substances being present in either public or private drinking settings (versus private parties) were associated with higher alcohol consumption by

individuals attending those settings. Sexual assault by intoxicated persons is also related to drinking setting with “bar culture” being a significant contributor (Davis, Kirwan, Neilson, & Stappenbeck, *in press*).

Consider also the harm reduction practice of providing supervised injection sites/facilities: locations provided in several European countries and Canada suggest that these locations, as opposed to other public or private spaces, reduce needle sharing, promote safer drug use, encourage access to services and entry into treatment, and make available staff to respond in the event of an overdose ([https://harmreduction.org/blog/sif\\_dcr/](https://harmreduction.org/blog/sif_dcr/)). In other words, setting can make a difference in behavior.

## Gene-Environment Interplay

Social and physical environment elements have a great deal of power to potentially modify genetic and psychological influences on health-related outcomes, including substance use initiation, substance misuse, and the development of substance use disorders



(Begun, Bares & Chartier, *in press*). For instance, social and physical environment factors may compound vulnerabilities or impart resilience by either imposing constraints or offering opportunities that enable, trigger, disrupt, or strengthen biological or psychological effects (Bares & Chartier, *in press*). Evidence supports the notion that genetic predisposition to alcohol use/misuse/use disorder and environmental exposures interact to influence alcohol use patterns (Sher et al., 2010). Similarly, this type of interaction was

observed in tobacco use patterns (Chen et al., 2009). The concept of a *gene-by-environment interaction* indicates that a person's genetic makeup can determine sensitivity to environmental effects and whether environmental exposure enhances or diminishes genetic effects (Bares & Chartier, *in press*). A body of research concerning alcohol, cigarette, and other substance use initiation, as well as for regular substance use, generally suggests that the influence of environment is stronger during early adolescence and gradually shifts to genetic factors (heritability) playing a more predominant role in adult development (Bares & Chartier, *in press*). For example, parental monitoring can reduce the influence of genetic heritability in cigarette use (Dick et al., 2007). Additionally, genetic effects on alcohol use are more evident among adolescents receiving low levels of parental monitoring, as well as adolescent affiliating with peers who engage in high levels of deviant behavior (Kendler, Gardner, & Dick, 2011). The interaction between intrinsic (biological and psychological makeup) and extrinsic environmental forces related to substance misuse is further explored in Module 6, reintegrating biological, psychological, and social context models into a more unified biopsychosocial framework.

## Social Systems

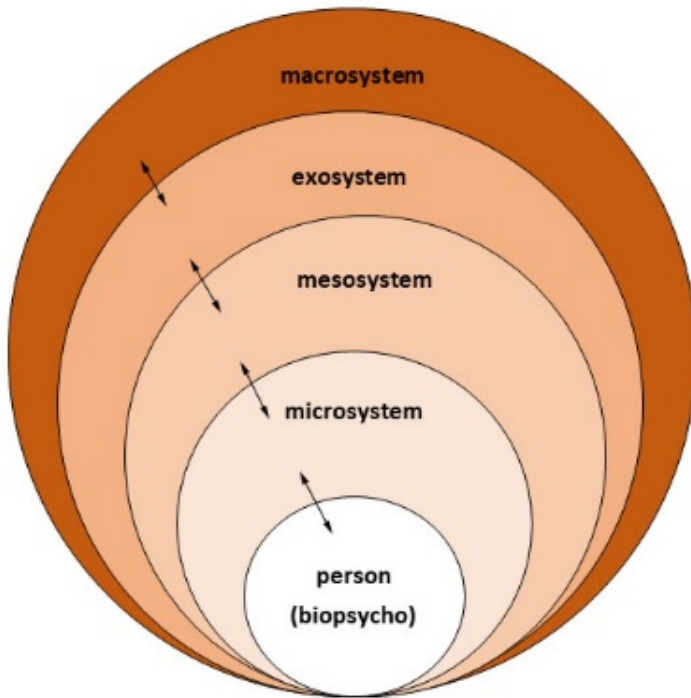
Anthropologists argue that the use of substances can only be properly understood when placed within *a social context*: the family, social, school, work, economic, political and religious systems (Hunt & Barker, 2001). The [\*social ecological model\*](#) (Bronfenbrenner, 1986, 1996) related to human development occurring within social systems at varying levels helps direct attention to social contexts as related to substance misuse—as well as informing interventions for substance misuse prevention and recovery support.

**Social Ecological Model.** In considering how a social ecological model might apply to substance misuse, we can start with the heart

of the matter: the center of the model represents the individual person. This sphere incorporates what we have studied so far in relation to a person's biological and psychological makeup—the bio and psycho components from earlier course modules. This is what the person brings to any interactions or experiences with their social or physical environments. Next, we look at the many contextual spheres of influence, forming an appreciation for an individual's social ecology. These begin at the most intimate, daily connections through a series of progressively more remote spheres of influence: the micro-, meso-, exo-, and macro- system levels (see Figure 1). These social systems influence individuals, individuals influence them, and they influence each other. These multi-directional influences explain why there are arrows between system levels depicted in Figure 1.

Figure 1. Diagram representing social ecological model's multiple system levels





[Microsystem](#) influences include social systems with which individuals directly interact on a regular basis: immediate family members/partners, close friends, and others in the most personal, intimate sphere of daily living. These microsystem members have a powerful effect on an individual's behavior through various mechanisms, including the way that they influence learning through delivering consequences (reinforcing or punishing) behaviors, serving as the models for behavior (social learning theory),

communicating expectations (expectancies and social norms), and possibly triggering cravings. These microsystem members also influence the immediate physical environments. For example, they may make it easier to access alcohol, tobacco, or other drugs. While the microsystem influences an individual's experiences and environments, the individual influences the microsystem, as well. Consider how a person's substance use affects their own behavior and responses to family members or friends; influences on parenting, relating to an intimate partner, or engaging with friends might be affected, along with the effects of bringing illegal activities into the relationship or home environment. This, in turn, has a reciprocal influence on the social context and physical environment experienced by the family and friends in the microsystem. The microsystem of recovery might include one's sponsor in a mutual aid/peer support/12-step type of program.

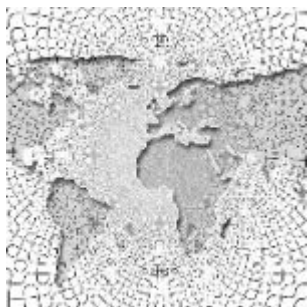


Moving one sphere further out, the microsystem influences and is influenced by the [mesosystem](#). The mesosystem components include elements in the relatively immediate environment with which an individual routinely interacts, but less frequently and intimately than was true of the microsystem. For some individuals this

includes extended family members and peers/friends with whom the relationships are influential but not as close and intimate. It might include the companions in the workplace or at school, and it might include neighbors. For some individuals this might include members of a religious or spiritual community. The mesosystem of recovery might include companions in the peer support community, other members of mutual aid/peer support/12-step type programs. It is also possible that members of the formal health/mental health/addiction treatment system fit into the mesosystem context.



The [exosystem](#) is one more step removed in terms of regular interactions and direct impact. This includes social institutions with which a person directly engages, but somewhat less frequently and intimately. Depending on the nature of the interactions, social institutions designed to provide services might be in the mesosystem for a particular person or family. For example, this might distinguish between the office where someone works (mesosystem) and the company for whom the person works (exosystem). Or, it might distinguish between the person providing recovery treatment (mesosystem) and the agency where treatment is being provided (exosystem). The practices and policies of these social institutions (e.g., zero tolerance policies) influence the individual's experience in the social environment through indirect interactions, often filtered through intervening systems (mesosystem and microsystem). A significant component of the exosystem involves community policing around substance-related activities. For individuals involved with drug court by virtue of their substance-related activities, the team of professionals might be part of the mesosystem and the social service delivery systems as part of the exosystem.

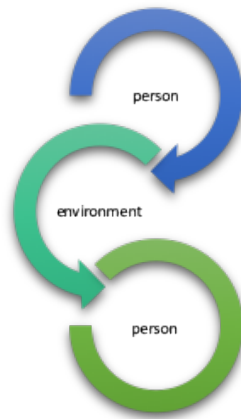


Finally, we have the [macrosystem](#) to consider. While few of individuals directly interact on a routine basis with the elements shaping the cultures and societies in which they live, these elements exert powerful (though indirect) influences on experience. Consider, for example, how changes in the legal status of certain substances influences behavior at the individual level. Popular social media platforms provide an interface between what happens at the macrosystem

(and exosystem) level and the more intimate levels of our social environments. It helps shape attitudes, values, beliefs, stereotypes, and stigma about substance use that are then expressed in the mesosystem and microsystem. Social workers and other professionals cannot afford to ignore the impact of policy, laws, and law enforcement patterns operating at exosystem and macrosystem levels on the social context of substance use at more proximal levels. For example, in many communities there exists a reciprocal relationship between the two problems of heroin use and the abuse of prescription pain medicines: as communities crack down on prescription drug abuse, making the substances more difficult to obtain, problems with heroin seem to explode.

Within this social ecological framework, we can look more closely at theories concerning the mechanisms by which these social ecology elements have their impact, and at evidence concerning these different elements.

**Circularity of Influence.** As noted in the previous discussion, but warranting an emphasis and attention is that individuals being influenced by the social and physical environments is one part of the equation: it is also true that they have an influence on their social and physical environments, as well. Anyone who has cared about a friend or family member experiencing substance use disorder will tell you that the individual's substance use, related behaviors, and consequential problems not only affect that individual but also has an impact on those in the social and physical contexts, as well. The individual's behaviors affect many different types and levels of social and physical



environments; the very environments that influence that individual, too. This iterative pattern of influence continues over time—the environment influences the person who influences the environment, and the changed environment continues to influence the changed person, and so on over time. This is what is meant by the concept of [circularity of influence](#). This perspective acknowledges that individuals are actively engaged with their environments, not simply the passive recipients of environmental influences; furthermore, individuals make choices and decisions from among options available in their social and physical contexts, choices that have consequences for themselves and others in their social/physical contexts, as well (Begun, Bares, & Chartier, *in press*; Shelton, 2019).

## Social Norms

A culture's or group's collective expectations about acceptable behavior are represented in its [social norms](#). Social norms are key social processes related to many types of behavior, including substance use and misuse. Groups may have specific norms about initiating substance use, acceptable patterns for regular use, excessive use or intoxication, seeking treatment for substance-related problems or substance use disorder, and recovery support. For example, most cultures accepting of alcohol use have norms related to the boundaries of its acceptable use—when, where, by whom, and how much. Social norms influence individuals' behavior choices. For example, a person who believes that “everyone else” either uses or approves of using cannabis is far more likely to engage in its use than a person who believes that it is not common or accepted in their social context. Or, for example, social norms against driving under the influence of alcohol (or other substances) influence the behavior of individuals electing to use sober driver strategies when planning to participate in drinking events. On the

other hand, if public education efforts deliver messages that “too many young people” use alcohol, tobacco, or vaping products, the actual message received by that population may be that engaging in this behavior is normative and accepted within their group. In other words, the message could backfire as a preventive strategy because it actually conveys a positive social norm about the behavior. Social norms surrounding substance use are significantly related to substance use behavior, especially among adolescents (Eisenberg et al., 2014). Media campaigns have proven effective in shaping norms and health-related behaviors related to intoxicated driving, use of tobacco products, and parents discussing substance misuse with their children (Wakefield, Loken, & Hornik, 2010). See, for example, the “Don’t Live in Denial Ohio” media campaign (<https://dontliveindenial.org/>).



To understand young cohorts and their norms related to substance use, consider *Monitoring the Future* 2018 study results (Table 2). The survey asked students to rate their own level of disapproval toward people who use various substances. What is interesting in these data is that the trend is substance-dependent. Between 8th, 10th, and 12th grade each group of students was more accepting of alcohol and marijuana use than the next younger group. The opposite was true of heroin, cocaine, LSD, inhalants,

and regular vaping of e-liquids containing nicotine. It is not clear whether these cross-sectional data reflect a true developmental change in youths' opinions. However, it does suggest that as the students progressed in age/grade, they make clearer distinctions between types of substance use.

Table 2. Percent of students who disapprove or strongly disapprove of “people who ...”, created from *Monitoring the Future* data 2018, retrieved from <http://www.monitoringthefuture.org/data/data.html>

<b>Do you disapprove of people who...</b>	<b>8th graders</b>	<b>10th graders</b>	<b>12th graders</b>
try one or two drinks of an alcoholic beverage	47.4	39.6	31.3
take one or two drinks nearly every day	77.9	77.9	74.7
have five or more drinks once or twice each weekend	83.7	80.4	75.8
taking 4 or 5 drinks nearly every day	—	—	91.7
try marijuana once or twice	64.5	47.9	41.1
smoke marijuana occasionally	73.1	57.4	49.2
smoke marijuana regularly	79.3	69.7	66.7
try heroin once or twice without using a needle	85.5	90.6	93.0
take heroin occasionally without using a needle	86.8	91.2	93.4
taking heroin regularly	—	—	96.8
try cocaine once or twice	85.6	87.6	88.9
take cocaine occasionally	88.9	90.9	—
take cocaine regularly	—	—	95.8
take LSD once or twice	55.9	70.5	80.5
take LSD regularly	59.4	76.5	93.4
try inhalants once or twice	75.0	81.8	—
take inhalants regularly	81.3	86.9	—
vape an e-liquid with nicotine occasionally	60.8	58.0	59.2
vape an e-liquid with nicotine regularly	68.9	57.8	70.9

Social norms about alcohol and other substance use are tied to ethnic identity and stereotypes, as well. For example, there exist many drinking-related stereotypes about Irish Americans and Americans with Russian roots. Ethnic stereotypes can have a significant effect on an individual's attitudes and personal decisions about drinking and drinking to excess. On the other hand,



prohibitions around drinking to the point of intoxication or addiction may be strong in an individual's cultural context. For example, norms against alcohol use contribute to primarily Muslim countries having the lowest prevalence rates of alcohol use globally (Michalak & Trocki, 2006). Or, for example, the use of alcohol by members of The Church of Jesus Christ of Latter-day Saints is generally discouraged in the Word of Wisdom which advises members about healthy living.

Alcohol plays an integral role in many religious, ethnic, and cultural ceremonies, but when it is included its use is typically characterized by moderation—drinking alcohol in moderation is permissible but drinking to intoxication is not in alcohol-involved rituals such as Shabbat, Passover, and the marriage ceremony



in Judaism; substitutions for alcohol (grape juice, watered-down wine) are often accepted especially for pregnant women, young children, and persons in recovery from alcohol or other substance use disorder. Social norms disapproving of excessive alcohol use (misuse) can be a protective factor against alcohol use becoming an alcohol use disorder. In an analogous fashion, social norms concerning use of tobacco products, e-cigarettes/vaping, cannabis, and other substances may also have an impact on individuals' decisions about initiating substance use, using substances to excess, or using substances under risky circumstances (e.g., driving or operating dangerous equipment, use during pregnancy, use by adolescents, use in combination with other substances). Shaping and communicating social norms is one target of preventive media campaigns.

Another perspective to keep in mind when thinking about social norms is an observation about [homophily](#). The homophily principle means that, when free to choose, humans tend to congregate and associate with friends, acquaintances, and partners similar to

ourselves. The saying is, “birds of a feather, tend to flock together.” The implication substance use implication is that individuals may choose to spend time in the company of others who engage in similar patterns of substance use/misuse. The homophily tendency shapes and reinforces the individual’s social norms about substance use, misuse, treatment seeking, and recovery—leading the individual to believe that “everyone” holds those similar norms because most everyone in their immediate social context does.



## Social Structures

A number of theories draw from the science of sociology to explain the phenomena of substance use, misuse, and addiction. These theories “view the structural organization of a society, peer group, or subculture as directly responsible for drug use” (Hanson, Ventruelli, & Fleckenstein, 2015, p. 78).

**Culture and subculture.** Cultural systems are significant sources of socialization shaping attitudes, beliefs, and behaviors concerning substance use, misuse, treatment seeking, recovery, and stigma. The content of the values and belief systems of different cultural groups might vary, but many of the socialization



processes by which these values and beliefs are shared and influence behavior are similar across cultural groups. Policy, as a form of intervention, is heavily influenced by a culture's values and belief systems. For example, in the U.S. there has been a history of ambivalent philosophies concerning whether the problem of substance use is better addressed through punishment (criminal justice system responses) or treatment (physical, mental, and behavioral health system responses). Cultural systems are even responsible for defining “drugs” or “substances of abuse” in the first place. For example, in U.S. majority culture, hallucinogenic substances like peyote are defined as drugs of abuse. However, according to anthropologists, peyote religion among certain indigenous North American groups (e.g., Tarahumara Indians of Mexico and various western Native American groups) defines this substance quite differently (Hill, 2013). Its use is acceptable under specific circumstances by specified individuals, including to treat medical conditions and in ritual ceremonies—a clear distinction is made between ritual/medical versus recreational use.

The impact of cultural systems is especially evident among immigrant populations. New Americans experiencing strong cultural identity and/or closeness to their culture of origin may exhibit less susceptibility to alcohol and substance misuse, whereas adapting to the new dominant American culture could be a risk factor for substance related problems (Banks et al., 2019; Perreira et al., 2019). This is particularly true when acculturation pressure impedes family closeness (Begun, Bares, & Chartier, *in press*). The

protective force is dependent on the substance use-related norms of their original culture (Cook, Mulia, & Karriker-Jaffe, 2012). “The combination of having both strong spiritual beliefs and greater religious involvement provides a particularly strong protection against heavy drinking” (Begun, Bares, & Chartier, *in press*).

Subculture is about identifiable groups that form within a larger culture. The values, beliefs, attitudes, and behaviors within a subculture group may complement or contradict those of the larger cultural context. When they are contradictory, [deviance theory](#) may come into play. According to deviance theory, a person (or group) elects to engage in behaviors disapproved of by the conventional “majority” culture, often specifically because of that disapproval. Members embrace their deviance identity—the label becomes an important aspect of personal and group identity. Why would someone want to belong to a deviant subculture or group? For many, it is better to feel a sense of belonging somewhere, anywhere, rather than belonging nowhere—embracing/participating in deviant behavior feels like a small price to pay for admission to the group. For others it is a means of differentiating self from others—particularly from those who represent the conventional culture. It becomes a way of making clear to yourself and the rest of the world that you are your own person, distinct from your parents, siblings, family, neighbors, or others. Having strong prosocial bonds with members of the conventional or majority culture is a protective force against choosing to engage in deviance behavior—the extent to which a person desires approval and wishes to avoid disapproval of the people with whom they have these prosocial bonds helps them make choices that conform to convention (Sussman & Ames, 2008). It is also important to note that what is defined as “deviance” at one point in history, geographical location, or cultural system may later be redefined as the evolution or transition to a new conventional system. For example, attitudes toward cannabis use have shifted dramatically across many parts of the U.S. during the past decades such that a deviance position is now becoming normative.



**Labeling theory** suggests that other people's perceptions of us, the labels they apply to us, have a strong influence on our own self-perceptions (Hanson, Venturelli, & Fleckenstein, 2015). The individual faces the choice of acting in accordance with the labels (e.g., continued drinking to excess when labelled as an “alcoholic”) or differently from/in opposition to the label (e.g., quitting drinking or drinking in moderation). In addition, theory suggests that when individuals have weak bonds to conventional society, there is less motivation to conform to conventional social norms and expectations. Hence, they are more likely to deviate from those norms when they have less “**stake in conformity**” than others who choose to behave in ways that comply with conventional norms (Sherman, Smith, Schmidt, & Rogan, 1992). Similarly, **social control theory** frames it this way:

“According to *social control theory*, strong bonds with family, school, work, religion, and other aspects of traditional society motivate individuals to engage in responsible behavior and refrain from substance use and other deviant pursuits. When such social bonds are weak or absent, individuals are less likely to adhere to

conventional standards and tend to engage in rebellious behavior, such as the misuse of alcohol and drugs” (Moos, 2006, p. 182).

The roots of weak social bonds lie in social disorganization at the family, neighborhood, or school/work levels, and supervisory monitoring of behavior being lax, inconsistent or inadequate (Moos, 2006). On the flip side, strong family, school, work, religion, and other bonds to “traditional society” serve as preventive forces related to substance misuse (Moos, 2006).

**The impact of “isms.”** Issues of racism, classism, sexism, agism, and other forms of “ism” have a powerful impact on individuals’ experience of the social world, as well as on their physical environments. Experiences of oppression, discrimination, and exploitation based on racial, ethnic, social class, gender, gender identity, sexual orientation, religious, disability, or national origin factors are integral to understanding the social context of substance use, substance misuse, and substance use disorders. These forms of societal abuse fall along a complex continuum from the obvious, overt, or explicit to the subtle, covert, or implicit (Edmund & Bland, 2011).



Exposure to repeated instances of [microaggression](#) may contribute to substance use, as well. Ethnic and racial microaggressions are events that leave the person on the receiving end feeling put down or insulted based on their race or ethnicity—regardless of the intent by persons delivering the messages (Blume, Lovato, Thyken, &

Denny, 2011). In a study of undergraduate college students, microaggression experiences were associated with both higher rates of binge drinking and experiencing more of the negative consequences associated with drinking (Blume, Lovato, Thyken, & Denny, 2011). Similarly, a study of college students demonstrated that the odds of regular marijuana use increased as a function of the number of microaggressions experienced (Pro, Sahker, & Marzell,

2017). And, again, the same relationship was observed in a study of Native American students and use of illicit drugs (Greenfield, 2015). Thus, it is important for social workers and other professionals to consider the heavy toll exacted on individuals who experience incidents of societal abuse, and how substance use may be related to these cumulative trauma experiences. Not only does this include those who experience it first-hand, but also those who witness it (second-hand).



“Isms” play a role in creating and maintaining marked disparities in opportunity and resources between social groups at the level of neighborhoods, schools, communities, workplaces, and populations. These include discrepancies in media portrayal, access or barriers to drugs, disparate exposure to advertising and

media portrayals of drugs, access to desirable alternatives to drug use, availability and cultural competence of prevention and treatment options, and the consistency with which sanctions for drug-related activities are imposed (e.g., variable implementation of zero-tolerance policies or criminal justice system sanctions). Recall from Module 1 how the War on Drugs related to tremendous racial and ethnic disparities in the nation’s incarceration rates, for example.

Consider how social justice concerns and disparities function at the neighborhood and community level. The concept of social determinants of health has clear applications in substance use, misuse, and use disorders. Conditions that affect a wide range of health risks and outcomes include social, economic, and environmental factors through their impacts on behavior, risk exposure, and opportunity (CDC, 2018). For example, consider the difference between empowered and distressed neighborhoods to defend against the intrusion of illegal drug trafficking and the crime, violence, and exploitation that accompany drug trafficking, which

in turn affect access to these substances, trauma experiences, and other risk factors for individuals' substance misuse. With its accompanying adversities and deprivations, poverty may create an experience of chronic stress, which is a known contributor to substance misuse and relapse (Shaw, Egan, & Gillespie, 2007; Sinha, 2008). Poverty also may affect access to treatment for substance related problems (Begun, Bares, & Chartier, *in press*). In addition, alcohol and tobacco marketing is disproportionately directed toward low-income communities (Scott et al., 2008). Drinking among young men and women was positively related to the alcohol advertising exposure in their communities (Snyder et al., 2006). For example, men in low marketing exposure communities (5 exposures per month) consumed an average of 15 alcoholic drinks per month; men in high marketing exposure communities (45 exposures per month) consumed an average of 28 drinks per month. While the actual amounts consumed by women were lower (7 and 12 for the low vs high market exposure communities), the pattern was similar to that of men.

Not only do neighborhood factors increase residents' access to substances, they influence social norms about substance use behavior. Also consider how difficult it becomes in many communities to gain access to evidence-supported prevention or treatment services that are accessible in terms of being affordable, close to home, culturally appropriate, and developmentally (age) appropriate.







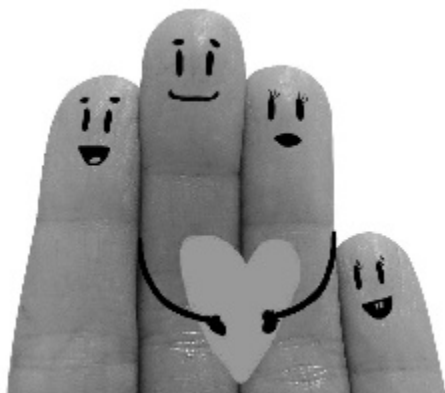
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# Ch. 2: Family as Social Context

There is no doubt that substance misuse and substance disorders often seem to “run” in families. We explored genetic models in Module 3 and learned that expression of genetic vulnerability or resilience to addiction is heavily influenced by environment and experience. The [family system](#) is a powerful source of environmental influence to consider. This chapter explores the family as one influential social context of substance use initiation, substance misuse, substance use disorder, and recovery.

Family forms a context for a great deal of human development—it is a site where individuals learn behaviors through operant conditioning (reinforcement and punishment of their behaviors) and observational learning (behavioral models), as well as become socialized into their culture, social norms, and social roles. The physical environment established by a family can also influence development and behavior through constraints and opportunities provided to individuals—for example, ease of access to alcohol, tobacco products, or other psychoactive substances. Family social relationships influence a person’s motivation for social conformity or deviance, as well. Family can be a source of stress to which a person might respond with substance use, or a source of resilience and protective factors that reduce the probability of engaging in substance misuse.



## Family Systems Theory

Not only do we need to consider how learning, social learning, social norms, and cultural beliefs related to alcohol and other substances operate within families, we also need to consider how family system principles apply to the situation. A prevailing principle in family systems theory concerns families' conscious or subconscious efforts to establish and maintain a stable state of [homeostasis](#) or balance. Just like a biological organism (e.g., the human body), family systems tend to develop practices, roles, rules, norms, patterns of communication and behaviors that serve this homeostatic function. Consider, for example, a family "rule" about not discussing or tending to minimize a member's substance misuse. Making the topic taboo might be dysfunctional in terms of getting the substance misuse problem addressed but may serve the family's need to maintain a stable peace despite the problem. Here is a brief orientation to four facets of the family systems theory as applied to individual members' substance misuse (see Begun, Hodge, & Early, 2017).

### 1. The family is a system embedded in other, larger social

**systems.** Just as we saw the individual embedded in micro- to macro-level systems in the social ecological framework, family systems also are embedded in progressively larger social systems. We cannot hope to understand families or their behavior, functioning, and development without understanding their interactions with their ecological contexts—the influences of extended family, neighborhood, social institutions, culture, society, and large-scale political, economic, and historical trends. Consider, for example, the influence of local, national, and global economy on families in your own community.

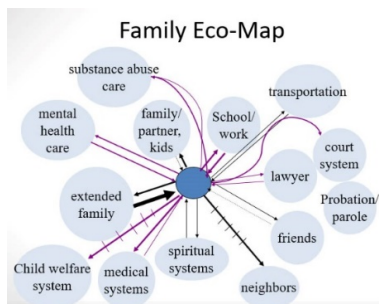


In this framework, consider how family contexts might influence individuals' substance use behavior, treatment seeking, and recovery-related behaviors and the ways that family supports or challenges their substance use behavior or recovery efforts. For example, how might a family's relationships with religious/



spiritual systems, education or workplace settings, neighborhood, criminal justice system, child welfare system, and others be relevant in preventing substance use initiation or substance use from becoming misuse or a substance use disorder? Individuals are not only a product of (and influence on) interactions with the nuclear family but also of the family's interactions with extended family—how might extended kin relationships impact the behaviors of an individual family member? Or, for example, how do kin play a role in caring for children when a parent is engaging in substance misuse or working on recovery (as discussed in a chapter about grandfamilies by Mendoza, Fruhauf, and Hayslip, *in press*). A number of interventions for individuals experiencing substance use disorder are designed to involve families and supportive significant others (SSOs) in the process, as well as provide support to these families/SSOs in their own right (e.g., as discussed in chapters about working with children and families of individuals engaged in substance misuse by Straussner and Fewell, *in press*, and by Petra and Kourgiantakis, *in press*). Considering the neighborhood, organization, and community levels, crime and violence in a neighborhood might be relevant because it affects family stress and distress levels, which in turn may influence substance use at the individual level; access to preventive and treatment interventions in the community are also relevant features of the family's context.

An [eco-map](#) is a visual assessment tool (originally described by Dr. Ann Hartmann) used in social work to help families identify and express the nature and quality of their interactions with the surrounding systems—what is supportive and what is



detrimental, where the energy, emotional, and resource “costs” to the family are excessive compared to what is gained, and where the “gains” are more favorable than the costs. In some cases, a family eco-map may appear overly sparse, leaving the family under-resourced and socially isolated. At the other extreme, a family’s eco-map may be overly saturated with formal institutions that serve the family but at a high “cost” in effort and energy; even informal relationships are supported at some cost since they typically exist within a set of “give and take” expectations. Even if the amount and types of ecological relationships may be reasonably balanced, the qualitative picture may be heavily conflict-ridden as opposed to working peacefully or harmoniously. For example, when a family member engages in substance misuse involvement with criminal justice, child welfare, housing, and health care systems may occur, much of which is demanding despite possibly providing needed resources. All of this relates to the degree of stress which the family system is under in its day-to-day existence. Stress is often translated into individuals’ use of substances, according to [stress and coping theory](#) which emphasizes the stress experienced in contexts characterized by a great deal of social disorganization, distress, and alienation (Moos, 2006).



2. **The family system is greater than the sum of its parts.** “The family” has meaning to its members separate from what each individual family member might mean. In other words, “family” is more than just a conglomeration of individual members living together. A family has an identity and “life” of its own. In many instances, family members act to further the family’s interests, even at individual expense. When an individual engaged in substance misuse violates this family-oriented expectation it is experienced as a gross violation by other family members. On the other hand, the possible impact on family is one potential protective factor stopping an individual from misusing substances—the impact on their family, not just on themselves. It also may be a motivating factor in someone wanting to engage in treatment and recovery—unfortunately, it can be difficult (as seen in the biological and psychological modules) to sustain this kind of family-focused motivation when substance use clouds a person’s mind and drives individuals’ substance seeking behavior.



3. **Family systems are comprised of subsystems.** Family systems do not always operate as a whole;

many interactions, roles, and functions are enacted within subsystems of the larger family system. These might include a couple subsystem, parent-child subsystems, or sibling subsystems. The possible variants are numerous especially when ex-partner, step-parent, step-/half-sibling, and extended family subsystems are involved. In families where a member is engaged in substance misuse, it is possible that the person's "relationship" with the substances themselves functions much like a subsystem. For example, an adult son living with his mother—both of whom experienced active substance use disorders—described their two-person family in terms of there being "three of us" in the relationship—himself, her, and the drugs (quoting Tony from the documentary entitled *Foo Foo Dust*).

**4. Change in any part of the system affects the entire system.**

Family systems are dynamic, changing over time as recognized in the chromosphere aspect of the social ecological model—past, present, and future look different because families are not static or stagnant. It is important to remember that all change—positive and negative in nature—are experienced as stressful, challenging the family system's hard-earned balance and homeostasis. Consider, for example, how stressful happy family development events like marriage, childbirth, and retirement can be for the system; this adaptive pressure might bewilder families who only expected to be stressed by negative changes such as divorce or the death of a family member. Family system changes are a response to pressures from the outside (contexts), family membership, internal subsystems, and changes in individuals. Some pressures are developmental in nature—the dynamics of parenting young children may be very different from parenting adolescents, for example. It is the nature of systems that change in any part of the family system reverberates throughout the entire family system, sending ripples throughout the system.





Despite individual and family developmental changes presenting periods of stress, which in turn may trigger a transition from substance use to misuse or may trigger a relapse during recovery, it is also possible that family changes can lead to a reduction in substance use (Moose, 2006). For example, the transition to parenthood, while creating stress on the family system, also may lead young adults to adapt their substance use to become more aligned with role expectations of parents—certain adult roles are not compatible with alcohol and other substance misuse and therefore may exert pressure to reduce or cease substance use (Moos, 2006). Adolescents and emerging adults maturing and entering into important social roles often is associated with reduced substance use, perhaps due to greater responsibility and/or pressure from partners concerning what is no longer appropriate substance use; “maturing” out of substance misuse is less likely if misuse has progressed to the point of a substance use disorder (Begun, Bares, & Chartier, *in press*).

**Role theory** also has relevance for how a family member’s substance misuse might be experienced by the family system (Begun, Bares, & Chartier, *in press*). Family members adopt and fulfill roles that function to serve the demands of the social environment,

the family as a whole, and the needs of individual family members (Begun, Hodge, & Early, 2017). While specific family roles, tasks, and behaviors vary by culture, context, time, and circumstances, key family functions include (1) obtaining and distributing resources necessary for meeting members' basic needs for food, shelter, and protection, and (2) socialization of family members into family and societal roles (Begun, Bares, & Chartier, *in press*). Family dysfunction is common when one or more family member, particularly a parent, engages in substance misuse (Straussner & Fewell, *in press*). That individual's expected roles could be:

- retained by, but poorly or inconsistently fulfilled by the individual engaged in substance misuse;
- delegated to other family members, potentially creating role overload situations for those individuals or role strain when the designated back-up player is ill-prepared for the role; or,
- unfulfilled, which in turn places the family system in a vulnerable state depending on how critical the role/functions are to the family and family members.

## Recovery as Change Experienced by the Family System

Outside of developmental changes, other individual changes can affect the family system as a whole: for example, a family member moving from substance misuse into recovery. Over time, a family may have adapted to the



individual's unpredictable, unreliable behavior while pursuing, using, and getting over the effects of using substances. Families may

adapt to a member's substance-related unpredictability and erratic functioning by excluding the individual from critical family roles (e.g., caregiving, financial decision making, intimacy). The family system exerts a great deal of energy and effort to achieve and preserve homeostasis under these rapidly shifting and unpredictable conditions. The family system again is challenged by the need to adapt to changes in the individual who engages in recovery efforts—how do family systems reintegrate these individuals and (again) provide them with meaningful roles? Despite recovery being a positive family event, all change in family systems is experienced as stressful—requiring the family to exert energy in (re)establishing balance. The system may not immediately respond in positive, accepting, welcoming, and trusting ways to the individual in early recovery, leaving that family member “on the outside looking in,” especially if they have been challenged to do so through multiple previous recovery efforts—they may be hesitant and not ready to place their trust in the recovery process. One member's recovery can be especially challenging to couples when they have been engaged in substance misuse together; their relationship may have been built around their substance use. What may look like “sabotage” of the recovery process by family members may more realistically represent the family's struggle to regain or retain homeostasis, even at the expense of or sacrificing one member's well-being. For this reason, as well as to support individual family members affected by another member's substance use, interventions at the couples and family system level are often recommended—helping the family to help itself as a whole and to support a member's recovery efforts (McCrary, 2006; McCrary, Epstein, & Sell, 2003).



### **Positive Parenting**

Evidence indicates that strong positive parent-child bonds, family involvement, sanctions against inappropriate behavior, (age appropriate) parental monitoring of their children's behavior and experiences are protective, preventive factors in terms of substance use initiation and substance misuse (Moos, 2006). Parents establishing clear, unambiguous prohibitive norms concerning substance use/misuse, parental monitoring, and warm, positive relationships with their sons and daughters are protective factors against substance use/misuse. Reinforcing the importance of parents' behavior is evidence concerning the preventive potential of parents' own restraint in substance use, child monitoring, and substance-related norm setting (Carpenter, Dobkin, & Warman, 2016; Cook & Tauchen, 1984; Hawkins, Catalano, & Miller, 1992). The presence of positive father-child relationships was shown to decrease the probability of adolescent alcohol use, particularly within African American families (Jordan & Lewis, 2005).

**Compensatory parenting.** A considerable amount of clinical literature discusses the negative developmental outcomes and risks to children growing up in a family where one or both parents engage in substance misuse, potentially leaving children “functionally parentless” (Straussner & Fewell, *in press*). This is the case for about

87 million children aged 17 or younger: about 1 in 10 living in households where a parent experienced alcohol use disorder in the past year and about 1 in 35 in households where a parent experienced a past-year substance use disorder involving illicit substances (Lipari & Van Horn, 2017). Parental substance misuse clearly has the potential to impede parents' ability to provide a safe and nurturing home for their children and raises the likelihood that children will be exposed to an array of environmental stressors (Straussner & Fewell, *in press*).

It is important also to consider the problem from a strengths-perspective, however: assessing the protective and resilience-promoting factors that may be operating in the child's environment, too (Begun & Zweben, 1990). One concept to consider is the possibility that parenting deficits are being otherwise satisfied by significant others in the child's life—children with the ability to elicit this type of [compensatory parenting](#) may be more resilient than others living under the same challenging conditions. For instance, children of parents with alcohol use disorder who elicited positive caregiver experiences from other caring adults showed a reduced probability of poor coping outcomes compared to other “less resilient” children in similar circumstances (Werner, & Johnson, 2004).



In short, it might be helpful to consider how the functions of parenting and caregiving are met when looking at children's experiences of the social and physical environment, rather than focusing only on the persons who are parents. Positive, stable compensatory relationships represent potentially significant contributions to a child's or adolescent's resilience (Begun,

Bares, & Chartier, *in press*). Compensatory parenting is often provided by grandparents (see Mendoza, Fruhauf, & Hayslip, *in press*), other extended family members, other social contexts (e.g., school personnel or peers' family members; Werner & Johnson, 2004), or through formal foster care arrangements.

### **Family Disease Model**

Earlier modules discussed debates concerning the disease model of addiction. Here we consider the [family disease model](#) of addiction. This perspective stems from an awareness of how one family member's substance-related problems affect other family members—especially in couples' and parent-child relationships (McCraday, Epstein, & Sell, 2003). The whole family might be viewed as suffering from the disease of addiction. As a family disease, this might be characterized by family role, communication, and relationship dysfunctions that perpetuate (enable) the individual family member's addictive behavior. The implication is that treating addiction requires intervention with families, not just individuals. In family systems terms, the individual clearly affects the rest of the family and the family clearly affects the individual.

However, the family disease model becomes controversial when relying on a definition of the family disease as [codependency](#). The codependency assumption is based on observations that certain traits and characteristics commonly occur within families experiencing a member's addiction. The defining traits involve family members' behaviors being organized around the one member's addiction-related behaviors. In codependency, family members' behaviors are viewed as supporting or enabling the dysfunctional behavior of the person experiencing addiction because they have come to depend on that person's dysfunctional behavior being maintained. The logical extension that has been inferred involves allowing the person's life to completely fall apart ("hitting rock bottom" or, at least "high bottom") and withholding love (or delivering "tough love") as means of motivating the person to change (Szalavitz, 2016). The evidence supporting this contention is weak and inconsistent, at best; evidence suggests that it actually

inflicts additional psychological harm and many family members refused to engage in this manner without being part of a dysfunctional family system (Szalavitz, 2016).

The codependency model has become highly controversial, with many practitioners and researchers arguing against applying the label or diagnosis of codependence. First, many behaviors identified as codependent can be viewed as reasonable adaptive responses rather than causes (or [enabling](#)) of the family member's addictive behavior. For example, compensatory parenting, while it may remove some negative consequences of a parent's substance-related neglect of their child's caregiving needs is important as a means of ensuring the child is protected and nurtured in important ways—thus, it is adaptive for the child rather than maladaptive for the parent, enabling the substance misuse to continue. Second, many of the observed behaviors also occur in healthy families, or at least in families where no member experiences addiction; the behaviors not being unique to families where a member experiences addiction means they are not diagnostic of a family disease process. Third, the label “codependent” has become overused, imprecise, and “blames” or “shames” family members for the problems they experience as a family. Finally, while “clinical descriptions of codependency are common, empirical support for the concept is lacking...there are no compelling empirical data to support the full construct of codependency” (McCrary, Epstein, & Sell, 2003, p. 120). Despite this level of controversy, the family disease model confounded with codependency constructs continues to underlie some intervention approaches.



## Supportive Significant Others

Family members and others in a person's social context may play a significant role in recovery (Begun, Bares, & Chartier, *in press*); recovery is a process heavily influenced by social processes and occurs within social contexts (Heather et al., 2018). Individuals in recovery from an alcohol or other substance use disorder engages in frequent, proximal, microsystem interactions—these might be with family, friends, co-workers, and members of mutual/peer support groups (Begun, Bares, & Chartier, *in press*). These individuals are considered significant others (SOs) in the person's physical and social recovery contexts. Different SOs, at different times, and through different behaviors may support the individual's recovery efforts, complicate the efforts, or be irrelevant to the recovery efforts. When they are acting in support of recovery, they can be identified as supportive significant others (SSOs). Intervention with SOs might involve training them to be supportive of recovery, to be effective as SSOs. Mutual/peer help programs (e.g., 12-step programs and other recovery support organizations) offer a person in recovery a network of SSOs, as well as creating opportunities for supportive physical environments (e.g., sober housing and social events). A focus of the twelve-step facilitation intervention is to help prepare individuals to effectively engage with



and benefit from participation in mutual/peer support programs available in their communities.

A controversial aspect of family members and friends supporting a person's recovery is represented in the television show *Intervention*. The show demonstrates the implementation of the Johnson Intervention confrontational approach for motivating a person's entry into treatment for a substance use disorder. Members of the person's social network confront the individual about the damage caused by their substance misuse and offer an ultimatum concerning the actions they will take if treatment is not engaged (Loneck, Garrett, & Banks, 1996). A critical review of the television show raises concerns about this use of the SOs (Kosovski & Smith, 2011). These include:

- footage is heavily edited to appeal to a (distorted) reality-television-consuming audience;
- the populations depicted poorly reflect the diversity of individuals engaged in substance misuse or experiencing substance use disorders;
- the treatment options, availability, and accessibility represented are a glaring misrepresentation of what treatments are available, affordable, preferred, and successful;
- data provided by the show concerning the success rate of the interventions in helping individuals enter and complete treatment are misleading and grossly misrepresent the intervention outcomes.

These authors cite literature indicating that fewer than 30% of families encouraged to engage in the confrontational intervention method actually follow through and host such an event, and that a relatively small percentage of individuals enter into additional treatment following such an intervention (Kosovski & Smith, 2011). They concluded that other family-based models used to engage individuals in treatment are more effective and have been evaluated with greater rigor than the Johnson Intervention model. In other

words, this approach does not represent a positive, effective role for SOs to act as SSOs.



Construct an eco-map of your own social contexts.

Identify the relationships you have with your social environment that might “push” toward substance use/misuse.

Identify the relationships you have with your social environment that help resist substance use/misuse.

Think about how this picture might have changed over time and could change in the future.

Consider what you learned from your own eco-map that can help you understand the eco-map of a person experiencing problems with alcohol, tobacco, or other substances.

# Ch. 3: Peer Groups as Social Context

A critical aspect of anyone's microsystem is encompassed by their peer relationships, particularly the friends with whom they interact on a regular and/or relatively intimate basis. In this chapter, we review evidence concerning the power of the peer group both as risk and protective forces concerning individuals' substance use behavior. We also examine the important role peers potentially play in supporting a person's recovery from substance use disorders.

## Peer Influences: Risk and Protection

Like the family, one's peer group provides a proximal context for learning (i.e., behavioral reinforcement and punishment) and social learning (observation, modeling) to operate, access to substances to occur, and social norms concerning substance-related behavior to be expressed and reinforced. "Evidence has also demonstrated a robust relationship between peer substance use and personal substance use," particularly within best friendships, peer cliques, and social crowds (Zimmerman & Farrell, 2017, p. 229). Equally important is evidence that positive peer influences, particularly among close friends, predicts low substance use among adolescents—potentially serving as a protective factor (Coyle et al., 2016).



Peer relationship influences on substance use (among adolescents, at least) are not independent of parental/family influences. The peer group may reinforce family norms against substance use/misuse, serving as a protective factor, or the peer group may contradict those family norms, serving as a risk factor. One mechanism by which low-level parental monitoring may operate to increase the probability of substance use/misuse among youth is the greater opportunity to engage with friends or other peers whose influence supports substance use/misuse (Begun, Bares, & Chartier, *in press*). On the flip side, parental monitoring has the effect of weakening the influence of associating with peers who engage in substance use/misuse (Marschall-Levesque, Castellanos-Ryan, Vitaro, & Seguin, 2014).

Clearly, peer approval and modeling of substance use/misuse is a powerful risk factor for adolescent substance use/misuse (Zimmerman & Farrell, 2017). Adolescents' initiation of substance use, as well as its escalation into problematic substance misuse, is strongly associated with friends' and



peers' substance use (Vink, 2016). This is evident with tobacco, alcohol, and marijuana; it possibly holds true with other substances, as well (Begun, Bares, & Chartier, *in press*). However, peer influences encountered during adolescence may persist well into emerging adulthood, at least as far as active involvement with friends who use alcohol is concerned (Piehler, Véronneau, & Dishion, 2012). In keeping with the previously noted human tendency toward homophily, about half of all people identified as important in the social networks described by individuals entering into treatment for alcohol use disorder were characterized as “drinkers” (24.63%), as were 19% of the most important people in their social networks (Mohr et al., 2001). Conversely, 9 months later, following 3 months of treatment, the rate of “drinkers” had declined significantly (17.98% among important people and 14.81% among the most important) and the rate of “nondrinking friends” had increased significantly from 17.98% to 33.37% of all the important people and from 14.81% to 21.47% among the most important people. Furthermore, the social network constellation of “drinking” and “nondrinking” friends was significantly related to treatment outcomes: the more important “nondrinking” friends became, the greater the individuals' proportion of abstinent days themselves (Mohr et al., 2001).

## Peer Support

As noted, the support of friends and peers as SSOs are important in a person's efforts at quitting substance use (Mohr et al., 2001). In the realm of recovery support, the word "peer" has multiple meanings. It may no longer imply persons of the same age/developmental life stage. Instead, it may take on the meaning of individuals with similar lived experiences in common. In this light, peer support is concerned with assisting others who may be at risk of developing substance use disorder to avoid this outcome or assisting others engaged in recovery to succeed in their efforts (Paquette et al., 2019). Peers are instrumental in assisting in recovery and integral part of recovery programming for adolescents, college students, and adults (Begun, Bares, & Chartier, *in press*; Davidson et al., 2012; Laudet et al., 2014; Paquette et al., 2019). For example, this is an important element of sober schools and sober campus housing programs, as well as mutual aid/recovery support programs. A systematic review of evidence concerning peer-delivered recovery support services concluded that the outcomes were favorable and made positive contributions to the participants' outcomes (Bassuk et al., 2016):

While we can conclude that there is evidence for the effectiveness of peer-delivered recovery support services, additional research is necessary to determine the effectiveness of different approaches and types of peer support services, with regard to the amount, intensity, skill level of the peer, service context, and effectiveness among different target populations" (p. 7).

It is not entirely clear how peer-delivered recovery support services best align with and/or integrates with services delivered by licensed treatment professionals.



*An interactive H5P element has been excluded from this version of the text. You can view it online here:*

<https://ohiostate.pressbooks.pub/swk5805coursebook/?p=146#h5p-49>

## Ch. 4: Summary

In this Module 5 online coursebook, you learned basic principles about social contexts and physical environments as they relate to substance use, substance misuse, substance use disorders, and recovery. We explored numerous ideas related to this complex topic. Not only were you introduced to several theories (social-ecological, deviance, labeling, stigma, microaggression, and stress/coping theories), you also examined some of the systems that are most relevant (family, peers, and cultural systems) and the roles of significant others. Additionally, you were introduced to the controversy surrounding the family disease model, the codependency concept, and the Johnson Intervention approach. Finally, you had a glimpse of gene-environment interactions in substance use initiation, substance misuse, and substance use disorder. This module concludes our separate analysis of the “bio,” “psycho,” and “social” in our biopsychosocial framework. In Module 6 we will be putting them together again into a more unified whole.

You are now ready to review some of the key terms related to substance use disorders that were introduced in this book.



# Module 5: Key Terms

**circularity of influence:** the iterative pattern of mutual influence operating between an individual and their social/physical contexts whereby each influences the other over time.

**codependency:** describes a pattern of dysfunctional behaviors between two individuals, one with a disease/disorder (e.g., addiction) and the other who becomes emotionally and psychologically dependent on the partner's disordered behavior at the expense of his or her own self and needs. Note that this is a controversial concept!

**compensatory parenting:** the assumption of unfulfilled parenting functions by significant others who are not in a parent relationship/role with the child.

**deviance theory:** theory explaining behavior that is outside the bounds of or violates conventional norms of society.

**eco-map:** diagrammatic representation of an individual's (or family's) relationships with its formal and informal systems operating in the environmental context.

**enabling:** providing the opportunity to engage in addictive behavior, particularly with reference to removing negative/punishing consequences that would naturally discourage such behavior. Note: this is a controversial concept!

**exosystem:** elements of the social ecology that have an indirect effect on individual development and behavior without the individual's regular, direct interaction; the effect is often mediated through more intimate systems.

**family disease model:** a perspective about addiction as a disease affecting the entire family, not just the individual experiencing addiction. Note: elements of this model are controversial!

**family system:** the family is viewed in systems dynamic terms where the family is more than a group of related individuals; it involves the interactions, relationships, and roles that exist across

the family, as well as both how individuals affect the system and how the system affects individuals.

**gene-by-environment interaction (or gene x environment interaction):** the interplay between intrinsic genetic and extrinsic social/physical context forces to determine outcomes.

**homeostasis:** the tendency for dynamic systems is to attain/maintain/retain a state of balance where energy expenditure is minimized.

**homophily:** the principle describing a human tendency to engage socially with people similar to ourselves.

**labeling theory:** sociological principal explaining individuals' deviant behaviors as resulting from having a deviant label applied to them; living up to the label applied to them.

**macrosystem:** the broad cultural systems in which individuals live and that influence individual development and behavior.

**mesosystem:** systems that have direct impact on individual development and behavior through their interaction with the more intimate microsystem within which the individual exists.

**microaggression:** insults, dismissal, and degradation of individuals, usually from a group defined by race or ethnicity; while these incidents fall short of physical aggression, they are experienced as a form of violence by the persons targeted.

**microsystem:** the most immediate, direct social system with which individuals interact on a regular basis, having a strong direct impact on individual development and behavior.

**physical environment:** elements of the places and spaces where individuals develop and function on a regular basis, offering opportunities or barriers that influence individual development and behavior.

**role theory:** many behaviors are determined or influenced by the social categories and functions (roles) an individual occupies and fulfills at the time; role expectations are defined by the social context rather than by the individual alone.

**social contexts:** the array of social relationships forming the context for an individual's development and behavior, offering

opportunities or barriers that influence individual development and behavior.

**social control theory:** avoiding deviant behavior and compliance with laws and norms is encouraged by social relationships, commitments, “stake in conformity,” and majority-held norms.

**social-ecological model:** first described by Uri Bronfenbrenner, this model explains the impact of multiple levels of social systems on individual development and behavior; these social systems and institutions interact and include micro-, meso-, exo-, and macro-system elements. Note: this general model can be extended to the family system being at the center and consideration of the family’s micro- to macro-systems.

**social norms:** a culture’s or group’s collective expectations about acceptable behavior.

**sociocultural theories:** theories or models of etiology/causation addressing aspects of the social environment and cultural contexts and their impact on development and/or behavior.

**stake in conformity:** individuals vary in terms of the number and strength of social bonds formed within conventional society; presumably, the greater the cumulative bond strength, the greater the motivation to conform to conventional norms. (see social control theory)

**stigma:** beliefs, values, and actions (behaviors) that set someone apart from others by diminishing that person’s worth by creating a semblance of shame or disgrace.

**stress and coping theory:** theory indicating that life demands create stress to which individuals respond based on the skills that they have for responding to the demands (coping); substance use is one possible coping mechanism although it may ultimately compound stress through increased demands.

# Module 5: References and Image Credits

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## PART VI

# MODULE 6: THEORY INTEGRATION, TRANSTHEORETICAL MODEL, AND VULNERABILITY/RISK/ RESILIENCE/PROTECTIVE FACTORS IN SUBSTANCE MISUSE

In our previous 3 modules we dissected the biopsychosocial framework into the biological, psychological, and social context/physical environment theories and models related to substance use, substance misuse, and substance use disorders. In this module, we consider how the different theories, models, and evidence might be reassembled into a more integrated whole. This effort is important because no one theory or model is sufficient to inform interventions across the board; we need to view them in concert rather than in conflict with one another if we are to build impactful strategies. One emphasis in this module relates to prevention strategies and how theory can inform these kinds of efforts.

After engaging with these reading materials and learning resources, you should be able to:

- Identify key vulnerability, risk, resilience, and protective factors related to substance misuse and substance use disorders and how they apply in an integrated model;

- Explain the continuum of care model and how it relates to prevention strategies;
- Describe a set of evidence-informed prevention intervention strategies;
- Explain theory integration and key principles in the transtheoretical model (TTM) of behavior change;
- Define key terms related to preventing and intervening around substance use, misuse, and use disorders.

# Ch. 1: Theory Integration and Prevention

*\*Note that portions of this chapter were informed by content presented in Begun and Murray (in press), Begun (1993), and Begun (1999).*

A wide range of biological, psychological, and social context theories and models concerning substance use, substance misuse, and substance use disorders can be integrated to inform intervention strategies and future research from a biopsychosocial perspective. A vulnerability, resilience, risk, and protective factors framework is presented here to help conceptualize and integrate multiple, diverse theories and evidence. This integrative framework reflects both a biopsychosocial and social work person-in-environment strengths perspective. Thus, it can inform interventions and policies that help change individuals, their environments, and the interface between individuals and their environments. The framework was derived from E. James Anthony's early work concerning the etiology of schizophrenia.

The vulnerability, resilience, risk, and protective factors framework is applied at the group or population level for purposes of informing/planning intervention strategies and research based on logic models and existing evidence. The state of evidence and assessment tools, at this time, is not sufficiently well-honed to predict individual outcomes, so the framework is not used to assess or predict what will happen for an individual person or family. Here, the framework's four steps are outlined.

**Specify the problem.** The more specific the problem definition, the easier the task of identifying and integrating varied theoretical models becomes (Begun, 1999). For example, "preventing adolescent initiation of alcohol misuse" is reasonably specific, whereas "preventing substance use disorders" is overly general.

Specificity might include specifics of an addictive behavior (e.g., a specific substance, type of technology, or form of gambling) and/or a target population (e.g., a specified age or developmental phase, racial/ethnic group, self-defined gender identity, sexual orientation, co-occurring problems, or problem severity level). It is important, as well, to be specific as to the system level being addressed: individuals, family subsystems, family systems, neighborhoods/communities, institutions, or geographical regions. Specificity about the prevention target hones your aim.



**2. Define the relevant vulnerability/resilience continuum.** Once a prevention problem is clearly specified, evidence concerning known vulnerability and resilience factors can be located and critically analyzed. The [vulnerability/resilience continuum](#) refers to factors *intrinsic* to individuals (or other system level specified in the first step). In other words, factors that individuals bring with them to any new situation or experience, such as those we studied in Module 3 (biological models) and Module 4 (psychological models). These include the factors like: genetics, neurobiology, and other biological processes; temperament and personality characteristics; abilities and disabilities; co-occurring problems; past experiences and learning; and, current attitudes, beliefs, knowledge, and behavior patterns. Some factors reflect individuals' vulnerability to the specified problem, other factors reflect aspects of their resilience. Together evidence concerning these intrinsic factors help determine where along a vulnerability/resilience continuum a group of individuals might be situated. For example, a recognized vulnerability/resilience factor with a great deal of supporting evidence and clear mechanisms by which it happens is the age at

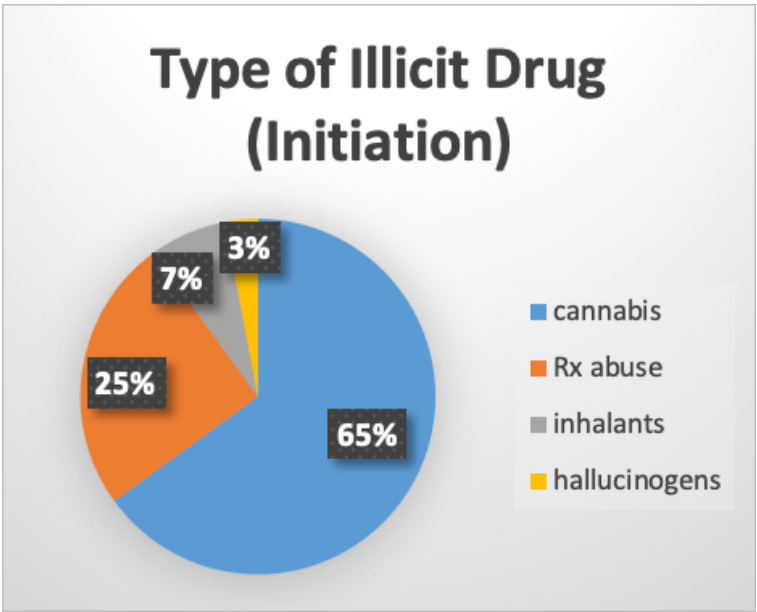


substance initiation—the earlier individuals begin using most types of substances, the greater the probability of developing a substance use disorder during their lifetime. Young age of initiation pushes the continuum toward vulnerability; delaying past emerging adulthood pushes the continuum toward resilience.



At this point, the task involves identifying the theory and evidence related to specific factors. For example, consider evidence related to the [gateway drug theory](#). If the identified problem is to prevent opioid/heroin use among adolescents and emerging adults, multiple theories and pieces of evidence will need to be considered for integration. One of these concerns the conflicting evidence surrounding cannabis use as a “gateway” to use of other, “harder” substances. Early evidence suggested a correlation between initiating heroin use and prior cannabis use—a very large portion of individuals using heroin had this in their past history (a vulnerability factor). However, subsequent and more sophisticated research approaches have called this gateway conclusion into doubt. First, the vast majority of individuals who have used cannabis never progressed to heroin use. Second, the distinction between “mild” and “hard” drugs is arbitrary and subjective. As we have discovered throughout the course so far, any psychoactive substance can be considered potentially addictive—some may have a higher percent

of use or faster progression to addiction than others, but placing them on a single, comparative “seriousness” continuum is not grounded in evidence. A third blow to the gateway drug theory is the tremendous diversity in substance use behavior observed across different geographical areas, ethnic and socio-economic groups, social networks, and cohorts over time. In the 2011 NSDUH survey, about 2/3 of participants who initiated illicit substance use during the study year reported marijuana as the first illicit substance used. However, this means that 1/3 started with something else: almost 25% started with prescription drug abuse instead, 7% started with inhalants, and just under 3% started with hallucinogens. This tremendous variability argues against a gateway drug theory—there are too many openings or access points involved for any one substance to be confidently identified as a gateway drug.



On the other hand, more recent events and evidence suggest that prescription abuse, particularly nonmedical use of opioid drugs,

may represent a gateway to heroin use. Individuals entering treatment for an opioid use disorder (OUD) reported having “progressed” from prescription opioid use and nonmedical use of opioids (NAS, 2017). This sequence was also, by far, the most common pattern observed among surveyed individuals in the general population who reported heroin use each year in analysis of 2003-2014 National Survey on Drug Use and Health data (NAS, 2017). Heroin, in many communities, is more easily accessed and less expensive since prescribing and dispensing restrictions have been introduced in response to the “opioid epidemic” facing many parts of the nation. “A number of studies have yielded evidence strongly supporting the conclusion that the recent prescription opioid epidemic has resulted in a significant increase in domestic heroin use and associated overdose death” (NAS, 2017, p. 207). The gateway theory concerning the relationship between prescription opioid and heroin misuse is bolstered by the fact that these substances have similar psychoactive and pharmacologic effects, including the capacity for [cross-tolerance](#) developing (NAS, 2017). Tolerance to a prescription opioid drug confers some degree of tolerance to heroin. Using higher doses of either/both increases the risk of overdose.

**3. Risk/protective factors continuum.** As with the vulnerability/resilience continuum, evidence concerning known risk and protective factors is identified and analyzed next. The [risk/protective continuum](#) refers to *extrinsic* factors. In other words, factors residing in current social and environmental contexts that we explored in Module 5 (social and physical environment contexts). The risk/protective factors continuum relates to “here and now” contexts and experiences; past interactions with the social context become a part of the vulnerability and resilience continuum—historical experiences of the environment become part of what is brought to new situations. For example, a history of [adverse childhood events \(ACES\)](#) becomes a vulnerability factor related to substance misuse; currently living in a traumatizing environment is a risk factor. Current risk/protective factors might

include the presence (or absence) of alcohol, tobacco/vaping, or cannabis advertising in the neighborhood/community, ease of access to substances of concern, and social norms about substance use/misuse.



4. **Integration.** Consider now how the two continua intersect: bringing together the vulnerability/resilience continuum with the risk/protection continuum. This is conceptually diagrammed as a 2 x 2 grid specifying the general probability (low, moderate, high) for developing the specified problem under these complex circumstances (see Figure below).

		Vulnerability/Resilience	
		low vulnerability/ high resilience	high vulnerability/ low resilience
Risk/ Protection	low risk/ high protection	I low probability	II moderate probability
	high risk/ low protection	III moderate probability	IV high probability

The result is an integration of theory and evidence to inform the development of intervention and policy strategies and logic models. For example, the low probability group (I) needs little attention beyond universal preventive efforts to maintain healthful status—maintaining both resilience and protective factors and minimizing new vulnerability and risk exposure. They are pretty much good to go (green light). On the other hand, the high probability group (IV) warrants a great deal of immediate attention with efforts designed to reduce both vulnerability and risk, as well as promote both resilience and protective factors. This group should stop us in our tracks, getting a great deal of our attention (red light). The two moderate probability groups (II and III) warrant attention in the form of selective or indicated prevention efforts to prevent their shifting into the high probability group (IV). They are the “caution” group (yellow light). Ideally, group II and group III

populations also can be helped to more closely come to resemble the low probability population (group I).

This is where theoretical models and empirical evidence inform both specific interventions (including policy) and planning broader combined intervention strategies, whether the aim is prevention, treatment, or maintenance of gains achieved. A great deal of literature across many disciplines presents detailed and nuanced evidence related to vulnerability, risk, resilience, and protective factors surrounding different addictive behaviors. This framework provides a logical system for organizing the massive literature, only some of which appears in this handbook.

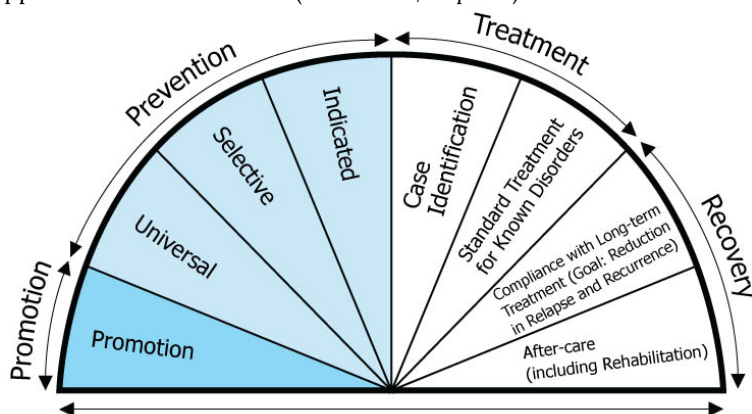


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## Ch. 2: Prevention and the Continuum of Care

The theories explored in our various modules so far have implications for the prevention of substance misuse and substance use disorders, including (but not limited to) delaying or preventing substance use initiation. The Substance Abuse and Mental Health Services Administration (SAMHSA) produced a Fact Sheet through the Center for the Application of Prevention Technologies discussing prevention as part of a behavioral health continuum of care. The Fact Sheet includes a diagram built on the foundational work presented in an earlier Institute of Medicine report diagramming the relationship between prevention, treatment, and maintenance in mental health care (IOM, 1994). This [continuum of care framework](#) is applicable to intervening around substance misuse and substance use disorders, and with the addition of health promotion embraces much of what is important in the recovery support services movement (Bersamira, *in press*).



This is how the Fact Sheet described the different “wedges” of the spectrum:

- **Promotion:** “These strategies are designed to create environments and conditions that support behavioral health and the ability of individuals to withstand challenges. Promotion strategies also reinforce the entire continuum of behavioral health services” (SAMHSA, n.d., p. 2). The [promotion strategies](#) described in the SAMHSA Fact Sheet include interventions that address resilience factors considered in our Chapter 1 discussion; strengths-based strategies designed to promote well-being and positive functioning.
- **Prevention:** “Delivered prior to the onset of a disorder, these interventions are intended to prevent or reduce the risk of developing a behavioral health problem, such as underage alcohol use, prescription drug misuse and abuse, and illicit drug use” (SAMHSA, n.d., p. 2).

- [Universal prevention](#) refers to interventions delivered to the general population without differentiating between persons at different risk levels.



For example, schools may deliver drug awareness and resistance education (DARE) programming to all students regardless of their vulnerability/risk constellation. Mass media campaigns are another example of universal efforts. In much of the prevention literature, the term “primary” prevention is used to describe efforts that occur before any sign of the target problem appear—universal prevention interventions are often applied.



- (C) A. Begun

[Selective prevention](#) is more



targeted than universal, and these interventions would be directed towards populations identified as having a potential somewhat greater than the general population for developing the focal problem. For example, it might be aimed at youth who live with one or more parents/family members engaged in substance misuse. In some prevention literature, the term “secondary” prevention is used to describe efforts that occur before the target problem appears and delivered to populations deemed to be “at risk” of the problem emerging—this could involve selective prevention interventions. Selective prevention is akin to a severe weather “watch” to keep a watchful eye on things, rather than a “warning” that the event is on the verge of happening.



- **Indicated prevention** is even more targeted, delivered to populations/groups of individuals exhibiting/expressing warning signs foreshadowing development of the focal problem. For example, to prevent alcohol use disorder interventions might be directed to youth/emerging adults engaged in binge drinking, preventing this behavior from becoming heavy drinking and a substance use disorder. As the focus increases, preventive interventions may become increasingly resource-intensive and intrusive which makes the focus beneficial. A great deal of effort and resources would be wasted if these intensive interventions were delivered to a large portion of the general population

unlikely to develop the problem anyway. In some prevention literature, the term “tertiary” prevention is used to describe efforts that occur early in emergence of the target problem—this could involve indicated prevention interventions or early intervention in the form of treatment. Indicated prevention is akin to a severe weather “warning” as a more imminent threat than a “watch.”

- **Treatment:** “These services are for people diagnosed with a substance use or other behavioral health disorder” (SAMHSA, n.d., p. 2). Unlike prevention, treatment services are designed to identify individuals experiencing or exhibiting the focal problem—preferably as early in its development as possible, before it becomes increasingly severe and more difficult to treat. Ideally, the treatment services delivered are those with the strongest evidence supporting their use under the circumstances involved.



- **Recovery** (the Fact Sheet reverts to the term “Maintenance” in the text, despite their Recovery label on the diagram): “These services support individuals’ compliance with long-term treatment and aftercare” (SAMHSA, n.d., p. 2). The diagram mentions long-term adherence to treatment as fitting into this category, which may or may not reflect what happens during/following treatment for substance use disorder. For example, engaging in mutual help/support programming (such as Alcoholics Anonymous/AA, Narcotic Anonymous/NA, SMART

Recovery, Women for Sobriety, LifeRing, Celebrate Recovery, and others) may be a part of both the treatment continuum and the recovery/maintenance continuum.

## **TOMORROW STARTS NOW**

Additional points made in the Fact Sheet include the fact that interventions do not necessarily fit into only one category. For example, a universal prevention intervention may take the form of health promotion. The term relapse prevention also may introduce a bit of confusion here: preventing a relapse to the old behavior is not usually considered part of the prevention continuum; it is usually considered part of the recovery/maintenance portion of the continuum of care.

Additionally, the fact sheet suggests that risk and protective factors may be both correlated and cumulative. On one hand, a person with one vulnerability or risk factor may be more likely to have multiple vulnerability and risk factors (positively correlated). This person also may have fewer resilience or protective factors, as well (negatively correlated with risk/vulnerability). On the other hand, a vulnerability or risk factor introduced early on may have developmental impacts that compound the person's vulnerability or risk over time. For example, being known as someone who uses alcohol, tobacco, or other drugs as a young adolescent might lead to that person being labeled, shunned, and stigmatized by peers. This, in turn, leaves that person vulnerable to social isolation and being attracted to a "deviance promoting" peer group, which compound the vulnerability and risk for substance misuse. The risk and vulnerability load just keeps getting heavier and heavier. Risk and vulnerability factors influence one another, underscoring "the importance of (1) intervening early, and (2) developing interventions

that target multiple factors, rather than addressing individual factors in isolation” (SAMHSA, n.d., p. 7).



Just as treatment interventions need to be developmentally appropriate, so do prevention interventions. Children and adolescents are qualitatively different from adults; simplifying or “dumbing down” interventions for adults is not sufficient adaptation for younger populations.



Because the risk and vulnerability factors are different at different periods of the life cycle, preventive efforts need to be tailored to what is relevant and salient at different periods (SAMHSA, n.d.). Preventive interventions also need to be appropriate for the vulnerability/risk mechanisms operating at different life periods. For example, if the concern is ease of access to substances, intervention might be targeted at the neighborhood/community or policy level rather than individuals; if the concern is to build initiation resistance skills, the intervention might be aimed at the individual level.

The SAMHSA Fact Sheet presented a set of tables of risk and protective factors for substance use disorder mapped to broad developmental period. These tables can help inform prevention strategies and used O'Connell, Boat, & Warner (2009) as their source. Their tables are replicated [with minor modifications] here and represent general mental health prevention goals at early ages.

<p><b>Infancy and Early Childhood</b></p> <p><i>Competencies: Infants begin understanding their own and others' emotions, to regulate their attention, and to acquire functional language</i></p>	
Risk Factors	Protective Factors
<ul style="list-style-type: none"> <li>• <i>Individual:</i> difficult temperament</li> <li>• <i>Family:</i> parental drug/alcohol use, cold and unresponsive [caregiver] behavior</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Individual:</i> self-regulation, secure attachment, mastery of communication and language skills, ability to make friends and get along with others</li> <li>• <i>Family:</i> reliable support and discipline for caregivers, responsiveness, protection from harm and fear, opportunities to resolve conflict, adequate socioeconomic resources for the family</li> <li>• <i>School/community:</i> support for early learning, access to supplemental services such as feeding and screening for vision and hearing, stable and secure attachment to childcare provider, low ratio of caregivers to children, regulatory systems that support high quality of care</li> </ul>

<p><b>Middle Childhood</b></p> <p>Competencies: Children learn how to make friends, get along with peers, and understand appropriate behavior in social settings</p>	
Risk Factors	Protective Factors
<ul style="list-style-type: none"> <li>• <i>Individual</i>: poor impulse control, sensation-seeking, lack of behavioral self-control, impulsivity, early persistent behavior problems, attention deficit/hyperactivity disorder, anxiety, depression, antisocial behavior</li> <li>• <i>Family</i>: permissive parenting, parent-child conflict, low parental warmth, parental hostility, harsh discipline, child abuse/maltreatment, substance use among parents or siblings, parental favorable attitudes toward alcohol and/or drug use, inadequate supervision and monitoring, low parental aspirations for child, lack of or inconsistent discipline</li> <li>• <i>School/community</i>: school failure, low commitment to school, peer rejection, deviant peer group, [favorable] peer attitudes toward drugs, alienation from peers, law and norms favorable toward alcohol and drug use, availability and access to alcohol</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Individual</i>: mastery of academic skills (math, reading, writing), following rules for behavior at home and school and in public places, ability to make friends, good peer relationships</li> <li>• <i>Family</i>: consistent discipline, language-based rather than physically-based discipline, extended family support</li> <li>• <i>School/community</i>: healthy peer groups, school engagement, positive teacher expectations, effective classroom management, positive partnering between school and family, school policies and practices to reduce bullying, high academic standards</li> </ul>

<p style="text-align: center;"><b>Adolescence</b></p> <p style="text-align: center;">Competencies: Adolescents focus on developing good health habits, practice critical and rational thinking, seek supportive relationships [and extend autonomy skills]</p>	
Risk Factors	Protective Factors
<ul style="list-style-type: none"> <li>• <i>Individual</i>: emotional problems in childhood, conduct disorder, favorable attitudes toward drugs, rebelliousness, early substance use, antisocial behavior</li> <li>• <i>Family</i>: substance use among parents, lack of adult supervision, poor attachment with parents</li> <li>• <i>School/community</i>: school failure, low commitment to school, not college bound, aggression toward peers, associating with peers [engaged in substance use], societal/community norms about alcohol and drug use</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Individual</i>: positive physical development, academic achievement/intellectual development, high self-esteem, emotional self-regulation, good coping skills and problem-solving skills, engagement and connections (in school, with peers, in athletics, employment, religion, culture)</li> <li>• <i>Family</i>: family provides predictable structure with rules and monitoring, supportive relationships with family members, clear expectations for behavior and values</li> <li>• <i>School/community</i>: presence of mentors and support for development of skills and interests, opportunities for engagement within school and community, positive norms, clear expectations for behavior, physical and psychological safety</li> </ul>

<p><b>Early [Emerging] Adulthood</b></p> <p>Competencies: Individuals learn to balance autonomy with relationships to family, make independent decisions, and become financially independent</p>	
Risk Factors	Protective Factors
<ul style="list-style-type: none"> <li>• <i>Individual:</i> lack of commitment to conventional adult roles, antisocial behavior</li> <li>• <i>Family:</i> leaving home</li> <li>• <i>School/community:</i> attending college, peers [engaged in substance use]</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Individual:</i> identity exploration in love and work and developing a world view, subjective sense of adult status, subjective sense of self-sufficiency, making independent decisions, becoming financially independent, future orientation, achievement motivation</li> <li>• <i>Family:</i> balance of autonomy and relatedness to family, behavioral and emotional autonomy</li> <li>• <i>School/community:</i> opportunities for exploration in work and school, connectedness to adults outside of family</li> </ul>

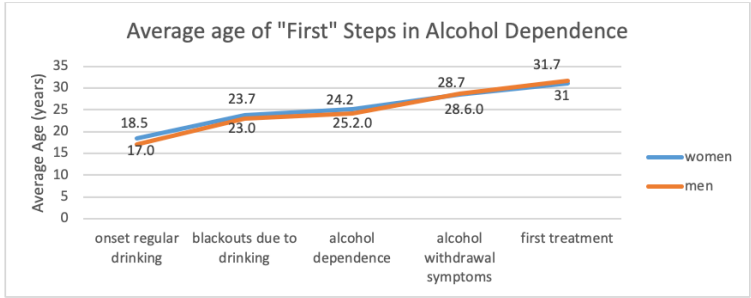
## Harm Reduction as Prevention

You may recall learning about Harm Reduction as a policy strategy way back in our first course module—that the goal is to reduce potential harms to individuals, families, communities, and society associated with substance use/misuse/use disorder, even if the substance use behavior does not end. Harm reduction policies, therefore, represent a type of prevention effort—preventing the associated harms. Harm reduction approaches are not limited to policy efforts: they also are applied at the individual level. For example, strategies to: reduce an individual’s risk of infection, accidental injury, or disease exposure associated with substance



misuse; reduce the chances of accidental overdose; or protect from criminal/sexual violence associated with substance use.

Another possible interpretation of prevention is intervention to slow, halt, or reverse progression from substance use to substance use disorder. As you learned in Module 4, there exists evidence suggestive of a developmental course of substance use disorder/addiction, even if there also exists variability in the course and its expression. Consider the developmental picture of average ages at which different events occurred in the lives of a group of individuals in treatment for alcohol use disorder (Schuckit, et al.,1998). Notice how many years (8!) were present between the average age at when blackouts due to drinking first occurred and when these men and women entered into treatment for alcohol use disorder: a harm reduction strategy might involve shortening this time span to reduce the physical, social, legal, and other harms that might accrue during that lengthy time span.



## Prevention Examples

In their book chapter about preventing alcohol and drug problems, McNeece and Madsen (2012) identified a host of efforts and strategies, including at the policy level. At this point, you should turn to the McNeece and Madsen (2012) chapter to become familiar with how they describe primary, secondary, and tertiary prevention

(aligned with universal, selective, and indicated prevention) and their review of the following types of prevention efforts:

- Public Information and Education
- Programs Directed at Children and Adolescents
- Programs Directed at College and University Students
- Service Measures
- Technologic Measures
- Legislative, Regulatory, and Economic Measures
- Family and Community Approaches
- Spirituality and Religious Factors
- Cultural Factors

Don't forget to return to this coursebook for Chapter 3!



*An interactive H5P element has been excluded from this version of the text. You can view it online here:*

<https://ohiostate.pressbooks.pub/swk5805coursebook/?p=157#h5p-46>

# Ch. 3: Theory Integration in the Transtheoretical Model of Behavioral Change

A great deal of effort both in our course and in research has been directed toward understanding the processes involved in substance use initiation and the progression from use to misuse and substance use disorder. At this point, we examine a model concerned with processes of change and recovery—moving back from problematic and disordered substance use into recovery. The model we focus on in this chapter is known as the [transtheoretical model of behavior change](#) (the TTM for transtheoretical model, or sometimes the TMBC for the transtheoretical model of behavior change). The model originally emerged in transtheoretical analysis of psychotherapies (Prochaska, 1979; Prochaska & DiClemente, 1982) and continued to evolve during the 1980s and 1990s based on research concerning the process of change in smoking behavior (Prochaska & DiClemente, 1983) and expanded to include other addictive behaviors (Prochaska, DiClemente, & Norcross, 1992). It has been applied across disciplines (social work, psychology, medicine, nursing, physical therapy, occupational therapy, and others) and across a wide array of behaviors, including but not limited to individuals making changes in their smoking (tobacco), alcohol use, adhering to a medication or medical treatment regimen, dieting, exercising, safe-sex, and intimate partner violence behaviors.

Use of the word “transtheoretical” in the model name reflects its theoretical inclusiveness and that it integrates and applies across theories. The transtheoretical approach represented an important shift in emphasis away from “horse race” research to find “the winner” among intervention options towards identifying

mechanisms of change and the elements or factors common across a variety of intervention approaches. The TTM's developers distilled from research and clinical observation a set of principles describing behavior change processes and factors that facilitate or pose barriers to achieving change goals. The TTM identified a series of five stages in the typical cycle of change, common processes involved in intentional behavior change, and implications for intervening to support individuals' intentional behavior change efforts.

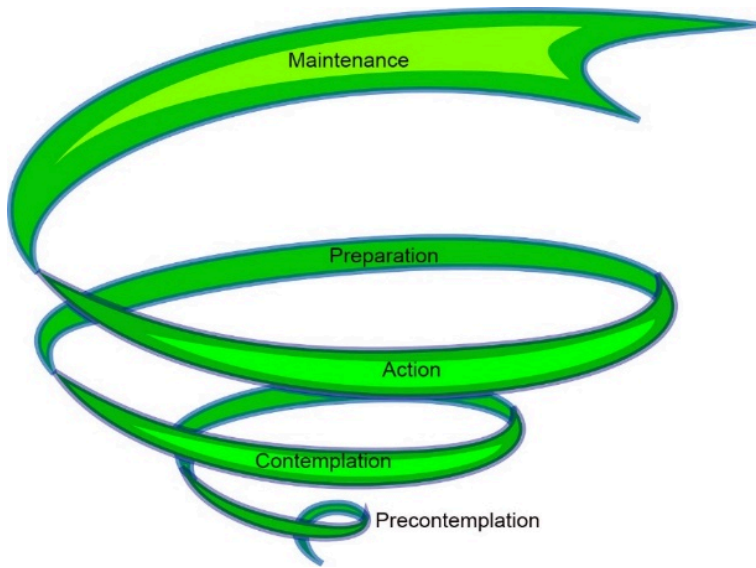


While you review the remainder of this chapter, consider a specific behavior that you have wished or tried to change in the past. See how the model seems to fit your own experience with intentional behavior change (something like getting more sleep, drinking more water, using less electricity, praising your partner

or kids more often, spending less money on coffee, stop biting your fingernails, expressing gratitude for small favors others do for us—it does not have to be about an addictive behavior).

## Stages of Change

Like most stage theories, the TTM identified a series of progressive stages that are qualitatively distinct from each other. Originally, the TTM specified four stages: Precontemplation, Contemplation, Action, and Maintenance; data reanalysis led to specification of a fifth stage, Preparation, between Contemplation and Action. An important difference from many other stage theories is acknowledgement that individuals do not move through the stages in a linear “upward” fashion but that they often cycle upwards and downwards through stages as they work to achieve their change goals (Prochaska, DiClemente, & Norcross, 1992). For example, a person perhaps beginning in Precontemplation may progress through some of the other stages, return to prior stages (including back to Precontemplation), and progress again over time, and that this cycle may repeat multiple times before the desired change goal is ultimately achieved. In research concerning smoking cessation, three to four Action attempts occurred before individuals were able to quit smoking for the long-term (Prochaska, DiClemente, & Norcross, 1992)—in other words, relapsing and falling back to earlier stages is normative, not atypical. A determining factor in how quickly someone is able to again move forward in the process concerns how relapse is handled: if seen as a failed attempt, the person may return to precontemplation and remain there for a lengthy period; if seen as an opportunity to learn from one’s mistakes, identify potential pitfalls and solutions, the person may move more quickly back into action instead. In fact, one criticism of the TTM is that individuals may move between stages so quickly that assessment tools are rendered inaccurate, and that a person may be situated between stages rather than in a single stage.



Another observation made by the model's developers was that what a person learns about changing one type of behavior may help them learn what will or will not help them change a different type of behavior. However, if someone is concerned about changing two or more behaviors at the same time, the change process for each will most likely differ—in other words, a person may be in one stage for one behavior change effort and a different stage for another. Consider, for example, that someone wishes both to quit smoking cigarettes and to quit drinking alcohol to excess. Each of these change attempts, although occurring at the same time, will progress on its own trajectory (Velasquez, Crouch, Stephens, & DiClemente, 2016). The individual may move through the cycle more quickly with one behavior compared to the other and may spiral back and forward more times. It is difficult enough to change an addictive behavior; it is far more difficult to change more than one at a time.

The five stages identified in the TTM distinguish between the

different behaviors, attitudes, experiences, and motivations representing each stage.

**Precontemplation.** The hallmark of **Precontemplation** is the absence of an intent to change the identified behavior, at least not in the foreseeable future. This includes individuals who are un- or under-aware of a



need to make changes. It also may include someone who wishes they could change but does not seriously intend to make the changes wished for. This stage also may involve resistance to change in response to pressure from others. For example, if a person is compelled to quit smoking while incarcerated in jail or prison, that individual may only comply as long as extrinsic (external) pressure is applied. There may be no intention to extend the change in behavior to the post-release period. The kinds of statements endorsed by someone in this stage include denial that a problem exists, that the behavior is not problematic, or that it is “their” business and no one else’s concern—like the proverbial ostrich with its head buried in the sand. On the other hand, they may engage in blame about the problem (“If I drink too much, it is because you are always nagging me”) or focus on an inability to change (“I have tried to quit smoking too many times, face it—I am just a failure” or “It is in my genes, I am destined to die this way.”)

**Contemplation.** A person in the **Contemplation** stage demonstrates awareness of a problem and serious consideration of making a change without making a specific change commitment. One characteristic of the Contemplation stage is the person struggling with the “pros and cons” dilemma—the advantages of making the change



versus the disadvantages. For example, someone might realize the health benefits of changing their binge drinking and appreciate the amount of money that could be saved by making a change, but at the

same time recognize that they like drinking, would be lonely without binge drinking with “buddies,” and that it will take a great deal of effort to make this change (see discussion of decisional balance below). An intention to make significant change within the next six months is considered a characteristic of Contemplation. However, individuals may remain in Contemplation for lengthy periods (despite the “within six months” intent) without moving further in the process—for two years or more among a group of participants in a smoking study (Prochaska, DiClemente, & Norcross, 1992). Examples of statements that a person in Contemplation might endorse generally include awareness of a problem and a desire to make a change: “I think I may have a problem with my drinking,” “I am really starting to feel the effects of my smoking when I try to walk upstairs,” “I am getting to the point where I can’t keep doing this to myself anymore.” A person in Contemplation might engage in information-gathering, exploring options for how to make the desired change (even looking into formal intervention/treatment options), but not actually engage with or commit to any of them.

**Preparation.** The [Preparation](#) stage extends beyond an intent to change to include early change behaviors toward the goal of taking serious action within the next 30 days. They will have set a plan in motion, even if not actively engaged in it yet, and have set a target day/date for the action to begin. For example, the person may enroll in a change-focused program, identify a specific change strategy or plan, and may begin taking “baby-steps” toward the change goal. For example, a person preparing to quit smoking may purchase supply of nicotine replacement “patches” or gum, schedule an appointment for prescription smoking cessation medication, register with a smoking cessation program. In addition, they may break their cigarettes in half to smoke less when they do smoke and gather together all their “stashed” cigarettes into one, visible collection. They may tell friends and family to refuse their requests to “bum” cigarettes and not invite them to share a smoking session.





**Action.** The [Action](#) stage is characterized by a person actively taking very specific, concrete steps to change the target behavior and keep the change momentum going. For a behavior as complex as quitting drinking, for example, the person may engage in a host of strategic alternative behaviors: avoiding the people, places, and situations that tempt them to drink; applying strategies for controlling their mood (e.g., mindfulness practices) and stress management (e.g., exercise); grocery shopping online to avoid impulse alcohol purchases in the store. Additionally, they may have new ways of rewarding themselves for each positive step taken (e.g., putting money that would have been spent on alcohol into an account toward a positive goal; celebrating their “sobriety birthday” each week, then month, then year), and reminding themselves of their accomplishments (e.g., journaling their efforts, experiences, and progress). Action is very often the emphasis in treatment programs—teaching, training, and practicing the new skills. A person in Action has specific skills and behaviors to substitute for and manage the old, problematic behaviors and they consciously act to implement these new behaviors. Action, by definition, lasts for at least 6 months and may last much longer for some individuals and some behaviors. Big changes in complex behaviors do not happen overnight. This is a person engaged in multiple, sometimes heroic, action efforts as they are fighting to achieve their change goals.



**Maintenance.** Once a person has engaged in action behaviors for at least 6 months, they may move into a [Maintenance](#) phase—a period of continued vigilance against relapsing to the past behavior. Individuals

continue to engage in relapse prevention activities, but it differs from the Action period in that the new changed/alternative



behaviors, attitudes, and experiences are becoming routine and feel relatively natural. They require less effort to maintain. During maintenance, a person continues to be aware that it would take only one “slip up” action to undo their hard work but takes many daily “non-actions” to avoid relapse—consistently avoiding temptations and relapse triggers, engaging in competing alternative behaviors, and managing temptations and relapse triggers when they do appear. A person in maintenance is not “cured” as long as there are temptations or craving experiences—the maintenance period may persist for a very long period, possibly indefinitely for some individuals. However, a person who managed to quit smoking cigarettes (for example) may reach a point when there is no longer any desire to pick it up again, none of their old cues trigger a temptation or desire to smoke, and they self-identify as a non-smoke (rather than an ex-smoker), even in periods of stress/distress. At the point where the changed behavior is relatively effortless, the person may be considered to have moved beyond maintenance.

### **Relapse.**

Understanding the change process is incomplete without recognizing what [relapse](#) is and how it might be addressed. Ideally, we want to prevent relapse to the “old” behavior whenever possible; but as the evidence indicates, relapse



happens (may even be a “norm” rather than an exception) and what happens in response to relapse matters very much in the future of a change effort. First, a distinction is made between a recurrence (“slip”) and a full-blown relapse event. A [lapse](#) or “slip” is time/event limited—doing it once or more times for a short period, quickly regretting the lapse, and getting back to renewed action. The circumstances surrounding a lapse can be effectively used as a learning experience to strengthen the ongoing change effort for the

future. Relapse refers to a return to the old pattern of behavior with no intention of changing again—spiraling back to Precontemplation, especially if the person despairs of ever being able to successfully change. A lapse, relapse, or impending relapse can happen at any point in the change process.

Relapse is a process (rather than an event) that starts before substance use occurs again—it is “a gradual process with distinct stages” (Melemis, 2015). The relapse process may begin days or even months before the actual substance use relapse behavior occurs and can be conceptualized in three parts.



- The “emotional” process of relapse is characterized by a lack of emotional, psychological, and physical care (Melamis, 2015). This includes basic physical care (diet, sleep, exercise, hygiene), as well as emotional and social “care” activities. This contributes to the kinds of negative emotional states involved in substance misuse—stress, tension, restlessness, anxiety, fatigue, irritability, and discontent.
- The “mental” relapse process concerns declining cognitive resistance to relapse, increased sensitivity to “use” messages, framing past use more positively (“glamorizing”) and minimizing consequences, entering into bargaining about use (“I’ll only do X and nothing more” or “It will be okay on vacation, just not in my regular life” or “if I stick to beer and avoid “hard” liquor, it will be okay”), scheming/lying, and actually planning a relapse/looking for relapse opportunities (Melamis, 2015). While occasionally thinking about using

substances again is a common experience during recovery, a warning sign is when these thoughts become frequent, detailed/specific, and intrusive/insistent in nature.



- “physical” relapse involves actual substance use/misuse—a return to uncontrolled substance use. One-time substance use may not lead to further uncontrolled use or it may contribute to the emotional and mental relapse processes that, in turn, lead to physical relapse. **Relapse prevention** involves anticipating and addressing all three parts—emotional, mental, and physical—and having in place plans for identifying/assessing and developing exit strategies for the different threats. This likely includes engaging supportive significant others (asking for help from trusted family/friends; participating actively in recovery-oriented or mutual support groups) and engaging in treatment interventions designed specifically around relapse prevention (e.g., cognitive behavioral interventions and skill building).

Concerted intervention effort might be directed toward relapse prevention, particularly during the maintenance stage.

## Change Factors

Threaded throughout the change process are a trio of factors: decisional balance, self-efficacy for change, and timing of different intervention/change promoting strategies.

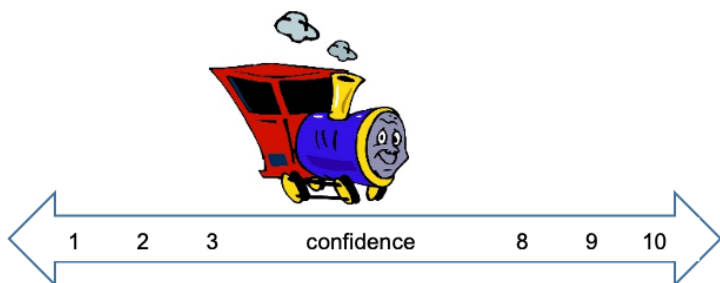
**Decisional balance.** Relevant throughout the change process, but particularly in the Precontemplation and Contemplation stages, is the concept of [decisional balance](#). The TTM relates to motivation for engaging in the change process. It recognizes that a person who is motivated to make an intentional behavior change may also be motivated NOT to make the change. There exist costs and benefits on all sides of the decision and a person may see-saw back up and down as the balance shifts toward or away from making the change effort. There are four dimensions of which the person is aware and that have implications for the likelihood of embarking on a change effort:

Not Changing	Changing	
	Pros	Cons
	Pros	no change
	Cons	ambivalence

Decisional balance underlies the ambivalence identified and addressed in motivational interviewing (MI). Eliciting and sustaining motivation for change often requires addressing ambivalence, not just emphasizing the advantages of changing and disadvantages of

not changing the behavior. Decisional balance is particularly impactful in the Precontemplation, Contemplation, and Preparation stages, but continues to have a role across the process.

**Self-efficacy for change.** Another cognitive process involved in each stage of the intentional behavior change process concerns a person's belief that change (or maintaining change) is possible: their [self-efficacy](#) for making or sustaining the change goal. Like *The Little Engine that Could*, self-efficacy ranges from “I can’t” to “I think I can” to “I know I can” and makes a difference in motivation at all stages of the change process. Someone might be in the Precontemplation stage (no plan to change) because they do not believe it is possible, despite being aware of that their behavior is problematic. This may be because they have made unsuccessful change attempts in the past and feel it is a hopeless goal. Two strategies for assisting with motivation in this situation are (1) focus on ways that they have succeeded in the past, including any positive steps they may have made in changing this behavior or any other behaviors they may have been able to change in the past, and (2) examining how others most like themselves have managed the change process. A conversation that might elicit self-efficacy involves a “change ruler” whereby a person indicates on a scale from 1-10 how confident they are in their ability to make the desired change in a situation of temptation. Rather than focusing on how far from 10 they are, the value lies instead on exploring why the rating is greater than 0—what the person may have going for them.



**Intervention timing.** Matching intervention strategies to “where the person is” with their change process, achieving the right timing, is an important consideration related to the TTM



(Velasquez et al., 2016). “Action-oriented therapies may be quite effective with individuals who are in the preparation or action stages. These same programs may be ineffective or detrimental, however, with individuals in precontemplation or contemplation stages” (Prochaska, DiClemente, & Norcross, 1992, p. 1106). Similarly, individuals ready for action and learning change-based skills may become frustrated and drop out of interventions aimed at raising their awareness of the problem and why they might need to make change—they are already past that point and ready to engage actively in change efforts. In other words, intervention efforts should be timed so as to connect to the relevant change goals at any point in time. Ideally, these fit together like puzzle pieces, and are adapted as the situation changes over time. For example, in efforts to move from Precontemplation to Contemplation, consciousness raising might be appropriate, whereas Action-oriented efforts might include creating a system of positive reinforcement for changed behavior and other change skill sets (Prochaska, DiClemente, & Norcross, 1992; Velasquez et al., 2016). While much of the TTM approach and motivational interviewing reflect the individual’s thoughts, feelings, experiences, and behaviors, it can effectively be applied in group work settings (Velasquez et al., 2016).



Thinking about the material you read in this chapter and the specific change effort example you were considering:

- What did you conclude about how the model seems to fit your own experience with intentional behavior change?
- How did you experience the stages of change and did you follow a single progression or spiral up/down the cycle?
- How did decisional balance, ambivalence, and self-efficacy for change look in your chosen example?
- What did or could have helped and what might have gotten in the way of your change effort?
- What does this tell you about possibly supporting others in their efforts to change, even to change addictive behaviors?



## Ch. 4: Summary

In this module you learned about different ways that the various biological, psychological, and social context/physical environment theories and models related to substance use, substance misuse, and substance use disorders might be integrated into a coherent biopsychosocial framework. We considered how the different theories, models, and evidence might inform prevention and change-promoting strategies. First, you read about four steps in a vulnerability, risk, resilience, and protective factors approach to substance misuse-related problems. The grid that can be created from evidence in the literature can inform prevention strategies at the population/subgroup level (not at the individual assessment level). Next, you were introduced to several key concepts and principles in prevention. Introduction to the continuum of care model included descriptions and examples of health promotion, prevention (universal, selective, indicated), treatment, and recovery/maintenance. Vulnerability/risk and resilience/protective factors related to substance misuse and to different developmental periods were presented as means of informing developmentally appropriate preventive intervention strategies. You read an analysis by McNeece and Madsen (2012) of evidence surrounding various prevention approaches, as well. Finally, you were introduced to the transtheoretical model of behavior change (TTM or TMBC), including stages commonly observed in the intentional behavior change process and factors that are important in the change process. These elements and factors were related to intervention strategies that might facilitate movement through the change process and assist individuals in achieving their behavior change goals.

At this point, we have concluded much of the work related to theories of substance use, misuse, and use disorders. We are now ready to launch into the second half of the course and look at

specific types of substances, as well as topics related to prescription and over-the-counter drug misuse, pharmacotherapy opportunities, and how co-occurring problems might affect substance-related problems and outcomes.

# Module 6: Key Terms

**Action:** the fourth of five stages in the transtheoretical model of behavior change, characterized by taking very specific, concrete, active steps to change the target behavior and keep the change momentum going.

**adverse childhood events (ACES):** potentially traumatizing experiences or events occurring during childhood that can or do have a persistent, negative impact on physical health, emotional health, behavioral/mental health, well-being, or development.

**Contemplation:** the second of five stages in the transtheoretical model of behavior change, characterized by awareness and ownership of that a problem exists and a general intent to change in the relatively near future but no concrete impending plan to change.

**continuum of care framework:** depicting an array of service/intervention options as representing different aspects of health promotion, prevention, treatment, and recovery/maintenance.

**decisional balance:** a process in intentional behavior change whereby an individual is aware of the pros and cons of both changing and not changing the target behavior.

**gateway drug theory:** a theory that use of one type of substance serves as a prelude to use of a different type.

**indicated prevention:** interventions delivered to populations/groups of individuals exhibiting/expressing warning signs foreshadowing development of the focal problem.

**lapse:** engaging in a limited way in a behavior that has been the target of an intentional behavior change effort (distinct from relapse).

**Maintenance:** the fifth of five stages in the transtheoretical model of behavior change, characterized by normalizing changed behaviors and relapse prevention efforts.

**Precontemplation:** the first of five stages in the transtheoretical

model of behavior change, characterized by a lack of intent to change a particular behavior either due to a lack of problem awareness or low self-efficacy for being able to successfully change.

**Preparation:** the third of five stages in the transtheoretical model of behavior change, characterized by efforts to set oneself up to actively engage in change efforts within the next 30 days (one month), potentially including initial change steps which may not be successful.

**promotion strategies:** strengths-based interventions designed to build resilience and promote well-being.

**relapse:** an emotional, mental, physical process whereby an individual returns or risks return to a past behavior pattern that was the target of intentional behavior change.

**relapse prevention:** efforts designed to identify relapse risk factors and intervene before an individual in recovery re-engages with the problem behavior.

**risk/protective continuum:** refers to extrinsic factors that increase (risk) or decrease (protective) the probability of a specific problem emerging, across a continuity of probabilities.

**selective prevention:** interventions directed towards populations identified as having a potential somewhat greater than the general population for developing the focal problem.

**self-efficacy:** a process in intentional behavior change whereby individuals experience differing degrees of belief in their ability to succeed in their change effort and/or to sustain the desired change over time.

**transtheoretical model of behavior change (TTM or TMBC):** a model of the processes and stages typically experienced in the course of intentional behavior change.

**universal prevention:** interventions delivered to the general population without differentiating between persons at different risk levels.

**vulnerability/resilience continuum:** refers to intrinsic factors that

increase (vulnerability) or decrease (resilience) the probability of a specific problem emerging, across a continuity of probabilities.

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## PART VII

# MODULE 7: FOCUS ON ALCOHOL

### Introduction

In this second part of our course, we focus on different types of substances classified in terms of their general actions on the human body, mind, and behavior. We begin with the substance identified as the most commonly used psychoactive substance in the world—alcohol. You may recall from earlier modules that, of all the substances we study, alcohol is the one most commonly used by individuals over the age of 12. “In many of today’s societies, alcoholic beverages are a routine part of the social landscape” (WHO, n.d.). Despite its use being legal in the United States, alcohol misuse causes or contributes to a wide array of serious social and public health problems. In this module, the basic nature of alcohol as a psychoactive substance is described and basic epidemiological study results are presented. Content presented in this module informs and was informed by Begun, A.L. (2020). Introduction to psychoactive substances. In A.L. Begun & M.M. Murray, (Eds.), *Routledge handbook of social work and addictive behavior*. London: Routledge.

### Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Describe the nature of alcohol and its effects on behavioral and physical health;
- Identify the effects of prenatal alcohol exposure on developmental outcomes;
- Explain alcohol consumption guidelines and define alcohol use patterns;
- Describe general conclusions from epidemiological evidence concerning alcohol use, misuse, and use disorders;
- Explain key terms and concepts related to alcohol.

# Ch. 1: The Nature and Effects of Alcohol

“Alcohol is a toxic and psychoactive substance with dependence producing propensities” (WHO, n.d.). Globally, alcohol is a leading risk factor in the burden of disease, contributing to 5.3% of all deaths (an estimated 3 million deaths) in the year 2016 (Popova, Rehm, & Shield, 2020) and accounting for 10% of all premature deaths among individuals aged 15-49 years (WHO, n.d.). “The harmful use of alcohol is a causal factor in more than 200 disease and injury conditions” (Popova, Rehm, & Shield, 2020). Alcohol misuse is also associated with a collection of social problems, including intimate partner violence, sexual assault, child maltreatment, human trafficking, problem gambling, housing insecurity, sexual risk-taking and unintended pregnancy, and suicidality (Begun, Clapp, & The Alcohol Misuse Grand Challenge Collective, 2015).

Recognizing the ubiquitous harms associated with alcohol misuse, the World Health Organization (WHO) established the reduction in harmful use of alcohol as one Global Strategy in 2010 and the United Nations specified substance abuse (including harmful use of alcohol) as a health target among its Sustainable Development Goals (SDGs). Among the Grand Challenges for social work posted by the American Academy of Social Work and Social Welfare, under a Close the Health Gap umbrella, is a challenge entitled *Reducing and Preventing Alcohol Misuse and Its Consequences* (Begun, Clapp, & The Alcohol Misuse Grand Challenge Collective, 2015)

## What We Mean by “Alcohol”

Different types of chemicals are referred to by the general word “alcohol,” including isopropyl (rubbing) alcohol, methyl alcohol (methanol, or “wood alcohol”), butyl alcohol, and ethyl alcohol. Each of these can be used for cleaning/sterilizing purposes, burned as fuel, or antifreeze. [Ethyl alcohol \(ethanol or “grain” alcohol\)](#) is what we think of as drinking alcohol. It is the only form of alcohol among this list that is safe for human consumption. Ethanol is only safe in controlled amounts and if it is not “[denatured](#)” by toxic additives; even ethanol (ethyl alcohol) is toxic when consumed in excess ([alcohol poisoning](#)). Ethanol is the psychoactive ingredient in alcoholic beverages like wine, beer, and spirits/hard liquor. These beverages differ in terms of their alcohol content/concentration and other ingredients or additives.

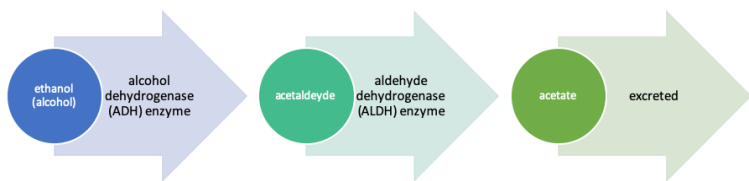
drinking alcohol (ethanol) is a chemical:  
 $\text{CH}_3\text{CH}_2\text{OH}$

Although alcohol is most often consumed in beverages, it can be administered intravenously as a medical intervention in an emergency response to acute alcohol withdrawal syndrome (which can be fatal). This mode of administration is highly discouraged by the lay person—it takes much less alcohol to result in (potentially fatal) alcohol poisoning when the digestive system is circumvented. Recently, powdered forms of alcohol have appeared on the market in some global regions (e.g., Japan, Germany, Netherlands) and are not legally distributed in others (e.g., many states in the U.S. and Australia). Concerns over the powdered form (e.g., Palcohol) include greater potential for overdose, risks associated with consuming it in undiluted form orally, by injection, or by snorting/inhaling, and

difficulty in controlling access in alcohol-restricted venues or by underaged youth because of its portability. The official “dose,” 1 ounce of powdered alcohol mixed in 6 ounces of water, equals one shot of traditional 80 proof spirits.

## Alcohol Metabolism

Alcoholic beverages, when consumed, are broken down (metabolized) by the body—much of the work being performed by enzymatic actions directed by the liver and somewhat involving kidney functions. The ethanol molecules begin to be metabolized by an enzyme called [alcohol dehydrogenase \(ADH\)](#) and others (Zakhari, n.d.). This first-step metabolic process results in the alcohol turning into [acetaldehyde](#) (NIAAA, 2007). Acetaldehyde is a relatively toxic substance responsible for many of the negative health effects associated with drinking alcohol: not only is it carcinogenic (<https://pubs.niaaa.nih.gov/publications/aa72.htm>) and contributing to liver disease, it contributes to “hangover” symptoms (nausea and headache) when alcohol is used in excess. Acetaldehyde is subsequently metabolized by another enzyme, [aldehyde dehydrogenase \(ALDH\)](#) (NIAAA, 2007) and eventually excreted from the body.

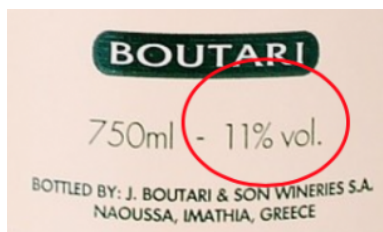


Human genetics play a significant role in directing the control and production of the metabolizing enzymes (ADH and ALDH), reflecting one mechanism in individual differences in alcohol

responses. You may recall from our genetic module how some individuals experience a very negative (punishing) “flushing” response to consuming alcohol—a reaction sometimes considered to be protective against alcohol misuse and alcohol use disorder (AUD). This response is driven by acetaldehyde building up because the person has a deficiency in ALDH2—the enzyme is slow to break down the toxic chemical acetaldehyde. Low ALDH enzyme concentration is also associated with an increased risk of esophageal cancer among individuals who drink alcohol for much the same reason—the acetaldehyde lingers in higher concentrations before being broken down into safer, less toxic chemicals. The medication known as Antabuse works by blocking ALDH activity, allowing the build-up of acetaldehyde which leads to unpleasant side effects (including nausea/vomiting)—this “punishment” is intended to discourage someone from drinking alcohol again in the future.

## Alcohol Dosing

Alcohol by volume (ABV) is a universal measure of alcohol concentration in a beverage. ABV refers to milliliters of pure ethanol in 100 milliliters of the beverage (at 68° F) converted to a percentage. For example, 40% is a typical ABV value for tequila, vodka, and rum. Alcohol proof values convey similar information: in the U.S., the proof value is double (2 times) the ABV percentage. For example, 40% ABV vodka is 80 proof in the U.S. (In the past, some over-the-counter medications had alcohol concentrations at least this high.) Alcohol proof values vary markedly by nation: in the United Kingdom, proof is 1.75 times ABV, making 40% ABV vodka 70 proof in the U.K. This degree of variability led alcohol investigators and clinicians to develop a more systematic measurement strategy which refers to “standard drink” measures.



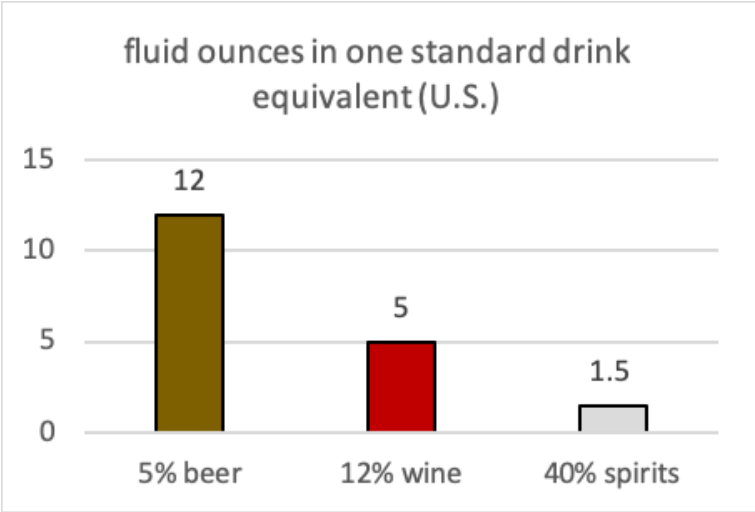
**Standard drink measurement.** If a person indicates that they consumed one drink, do we really know how much alcohol they consumed? “One drink” could mean very different things

under different circumstances. Consider, for instance, the differences in calories consumed in a soft drink. A 12 oz can of Coca-Cola® contains about 140 calories. How does this compare to ordering a Coke® from a fast-food restaurant like McDonald’s® or Burger King®? A “small” drink ordered from McDonald’s® is about 12.5 oz and contains about 150 calories; a “small” drink from Burger King® has about 17.5 oz and contains about 210 calories. The picture is even more confused when you add in the “child” size (110 calories at McDonald’s® or Burger King®), a “medium” (210 calories at McDonald’s®, 290 calories at Burger King®), or “large” (310 calories at McDonald’s®, 390 calories at Burger King®). [Note: beverage sizes may have changed since these data were initially collected.]

Regarding alcohol, servers and even entire countries differ in terms of serving sizes/pour volume. Additionally, the strength of preferred beverages (e.g., beer versus malt liquor, wine versus sake, or different hard liquors) varies markedly between products and nations (Bloomfield, Stockwell, Gmel, & Rehn, 2003). For example, “one beer” might mean a 12 oz can, 16 oz pint, 24 oz Pilsner, or 40 oz bottle; and, beers vary in their alcohol content, as well (e.g., 4.2% ABV in Light/Lite beer to “strong brews” with 29%–67% ABV). Even talking about a “bottle” of vodka can be confusing—is the person referring to the miniature (like those served on airline flights), a pint, a fifth, or more? How much is in “a glass” of wine?

To help address this confusion, the U.S. National Institute on Alcohol Abuse and Alcoholism (NIAAA, 2016) published a [standard drink measure](#) protocol based on 14 grams of pure ethanol per drink equivalent; the World Health Organization (WHO) applies a criterion of 10 grams of pure ethanol that is adopted in most European

nations; other countries vary within the range across these two metrics (Ritchie & Roser, 2018). In the U.S., the standard drink equivalent of 14 grams is 0.6 fl oz of pure alcohol. This chart shows one standard drink equivalent for beer (5% ABV), wine (12% ABV), and distilled spirits/“hard liquor” (40% ABV).



The mathematical formula for calculating standard drink equivalents is as follows:

- multiply the number of fluid ounces by the % alcohol content converted to decimal (40% being 0.40; divide % by 100 or move decimal two places to the left)
- divide that result by 0.6 ounces of pure alcohol per drink-equivalent
- the result is the standard drink equivalents involved.

For example, someone consuming a 40-ounce beer (a “forty”) of 9% ABV has consumed 6 standard drink equivalents [ $1 \times 40 \times .09$  divided by 0.6]. On the other hand, a six-pack of 12 ounce “lite” beers of 4.2%



ABV would be 5 standard drink equivalents  $[6 \times 12 \times .042 \text{ divided by } 0.6]$ .

**Alcohol consumption guidelines and definitions.** According to the 2015-2020 *Dietary Guidelines for Americans* published by the U.S. government (Department of Health and Human Services and Department of Agriculture), alcohol should only be consumed in moderation and only by individuals who have attained the minimum legal drinking age (currently, age 21 years across the U.S.). By definition, moderate drinking means up to 1 standard drink equivalent per day for women and up to 2 drinks per day for men (<https://health.gov/dietaryguidelines/2015/guidelines/appendix-9/>), and since alcohol is not among the foods/beverages in the healthy eating pattern, the calories consumed as alcohol should be accounted for in the overall daily calorie intake. Aging related changes in alcohol metabolism and other health concerns mean that the limit for men or women should be up to 1 per day. The recommendation is no alcohol for women who are pregnant and individuals under the minimum legal drinking age, taking certain types of medication, having certain types of health or mental health conditions, in recovery from an AUD, or find themselves unable to control their drinking when they do drink alcohol. The guidelines also acknowledge individual differences in alcohol metabolism—differences related to body mass, body composition, and metabolizing enzymes.

- ***Binge drinking (heavy episodic drinking)*** is defined as consuming 4 or more drinks within about 2 hours by women and 5 or more drinks in 2 hours by men. “Binge drinking is associated with a wide range of health and social problems, including sexually transmitted diseases, unintended pregnancy, accidental injuries, and violent crime” (Dietary Guidelines 2015-2020).
- ***Heavy drinking*** is defined as 8 or more drinks per week for women and 15 or more drinks per week for men. Half of individuals engaged in 2 or more heavy drinking days per week

experience a diagnosable AUD (NIAAA, 2016).

- **High-risk drinking** is defined as 4 or more drinks on any day (binge drinking) or 8 or more drinks per week for women (heavy drinking), and 5 or more drinks on any day (binge drinking) or 15 or more drinks per week for men (heavy drinking). The “risk” refers to health concerns in general, not simply a risk for alcohol use disorder; it takes into consideration many chronic diseases and risks (e.g., violence) associated with regular binge or heavy drinking.

Low-Risk (Moderate) Drinking Limits (adapted from NIAAA, 2016)		
Time Frame	Men	Women Or Anyone Over Age 65
any single day	no more than 4 standard drink equivalents	no more than 3 standard drink equivalents
	AND	AND
per week	no more than 14 standard drink equivalents	no more than 7 standard drink equivalents
Less or no alcohol may be best depending on health, medications, and how alcohol affects you; none is recommended for adolescents and pregnant women.		

## Alcohol Effects

Alcohol is a potentially addictive substance associated with the development of tolerance and (potentially fatal) withdrawal syndrome: alcohol use disorder (AUD) is a recognized diagnosis

in the DSM-5 and ICD-11. As it is a central nervous system (CNS) depressant, combining alcohol with other substances can be dangerous (especially with other CNS depressants). Furthermore, consuming alcoholic beverages:

- affects a wide range of central nervous system structures and processes,
- increases the risk for intentional and unintentional injuries
- increases the risk for adverse social consequences
- has considerable toxic effects on the digestive- and cardiovascular systems
- is classified as carcinogenic,
- “as an immunosuppressant increases the risk of communicable diseases, including tuberculosis and HIV” (WHO, n.d.).

The probability of experiencing alcohol dependence during a person’s lifetime is 4 times greater if drinking alcohol began before age 15 years (compared to individuals whose drinking is delayed until age 21 years); that probability is reduced by 14% with each increasing year of age first use of alcohol is delayed (Windle & Zucker, n.d.). Children who even sip alcohol (often with parental consent) by the 6<sup>th</sup> grade have significantly greater odds of drinking full drinks, getting drunk, and drinking heavily by the time they are in 9<sup>th</sup> grade (Jackson, Barnett, Colby, & Rogers, 2017). In other words, early sipping is not the protective factor that many parents believe it to be; “offering even just a sip of alcohol may undermine messages about the unacceptability of alcohol consumption for youth” (Jackson, Barnett, Colby, & Rogers, 2017, p. 212).

Risks associated with alcohol use increase in a dose-dependent manner, meaning that the risks increase with greater volumes of alcohol frequently consumed and increase exponentially with high volume consumption on a single occasion—binge drinking (WHO, n.d.). Not only is the amount consumed relevant, but also the rate at which it is consumed. Rate matters because the amount of alcohol circulating in a person’s system is determined by the rate at which

it is metabolized and eliminated. Drinking a great deal of alcohol very quickly means that the circulating alcohol level is temporarily higher than if the same amount were to be consumed gradually over many hours—the body takes time to break down the alcohol as it is consumed.

**Blood alcohol concentration (BAC).** BAC refers to the percent of alcohol (ethanol) circulating in a person's blood stream measured in parts alcohol per 1000 parts of blood. In other words, a [blood alcohol concentration \(BAC\)](https://alcohol.stanford.edu/alcohol-drug-info/buzz-buzz/what-bac) of 0.10% is 1 part alcohol per 1000 parts blood (<https://alcohol.stanford.edu/alcohol-drug-info/buzz-buzz/what-bac>). Sometimes the term blood alcohol level (BAL) is used instead. The U.S. current national guideline used to determine when a person is unable to safely operate a motor vehicle is BAC (or BAL) or 0.08%. However, other nations set the level lower since sufficient impairment may occur at levels of 0.06% to make driving unwise and unsafe—lower BAC criteria for determining a person is under the influence (DUI) or intoxicated (DWI) are being promoted by lobbying groups in the U.S, as well. A BAC of 0.01% is indicative of alcohol consumption which is relevant to assess underage drinking.

BAC is affected by the dose/amount of alcohol consumed, the rate at which it is consumed, and person-specific factors such as body weight, biological sex, medications (and use of other legal or illicit substances), general health status, tolerance, differences in alcohol metabolizing (driven to a great extent by genetics), and to some extent whether food is also consumed. The following table (adapted from <https://alcohol.stanford.edu/alcohol-drug-info/buzz-buzz/what-bac>) identifies what you might expect to see in the behavior of a relatively young, healthy person whose drinking has led to different blood alcohol concentrations (BAC)—outcomes would be different in someone who routinely drinks heavily and has developed some degree of tolerance to alcohol.

BAC	Likely Observed Effects
0.01%-0.03%	No obvious apparent effects; slight mood elevation
0.04%-0.06%	Sense of relaxation, warmth; minor impairment of reasoning and memory
0.07%-0.09%	Mildly impaired balance, speech, vision, and control
0.10%-0.12%	Significantly impaired motor control; poor/loss of judgment; slurred speech
0.13%-0.15%	Grossly impaired motor control; blurred vision; significant loss of balance; anxiety/restlessness
0.16%-0.20%	dysphoria (disturbed mood); nausea; “sloppy drunk” appearance
0.25%-0.30%	Severe intoxication; unable to walk unassisted; mental confusion; nausea/vomiting; dysphoria (disturbed mood)
0.35%-0.40%	Loss of consciousness; brink of coma
>.40%	Coma; likely respiratory failure leading to death

A number of internet tools and phone apps are available for individuals to estimate their BAC. These are not guaranteed to be accurate but can provide information relevant to making decisions about continued drinking and/or driving. For example, the interactive BAC calculator at <https://alcoholaddictioncenter.org/resources/bac-calculator/> uses standard drink equivalents, biological sex, body weight, and time since the first drink to compute an estimated BAC. Working a few examples varying factors results in the following estimates—the last column concerns an alarming practice whereby a person attempts to consume 21 drinks on their 21<sup>st</sup> birthday: this is potentially a lethal act (note above that BAC over 0.40% may be fatal—this is called acute alcohol poisoning).

Many of the other combinations result in BACs over the legal limit (0.08%) for operating a motor vehicle.

male	female	male	female	male	female	male
180 #	180 #	180 #	140 #	180 #	140 #	480
5 drinks	5 drinks	5 drinks	5 drinks	2 drinks	2 drinks	21 drinks
2 hours	2 hours	4 hours	4 hours	2 hours	2 hours	4
BAC estimate: 0.11%	BAC estimate: 0.14%	BAC estimate: 0.08%	BAC estimate: 0.15%	BAC estimate: 0.03%	BAC estimate: 0.05%	BAC estimate: 0.54%

**Alcohol tolerance and withdrawal.** In earlier modules, you learned about the definition and biological processes of developing tolerance and experiencing withdrawal. Both are relevant to repeated use of alcohol over time. Individuals may develop alcohol tolerance such that after drinking at sufficiently high levels frequently enough the body begins to adapt to the presence of alcohol. This, in turn, means that homeostasis pressures are operating, and a person will need to either consume greater amounts of alcohol or drink more quickly in order to achieve the expected effects; or, the person will experience diminishing effects from consuming the same amounts over time. Alcohol withdrawal symptoms can range from relatively mild and unpleasant to very serious and potentially fatal; they may begin within hours of when alcohol use ceases to days after the last drink. Mild symptoms of acute alcohol withdrawal might include:

- tremors (e.g., shaky hands)
- headache
- nausea/vomiting
- anxiety

- sleep disorder (insomnia)
- profuse sweating

More serious/severe symptoms of acute alcohol withdrawal might include:

- hallucinations (tactile, auditory, visual)
- seizures
- confusion/disorientation
- rapid heartbeat, high blood pressure, fever
- [delirium tremens](#) or DTs (a constellation of symptoms involving severe mental and/or nervous system changes and mostly occur in persons who have engaged in heavy drinking for multiple years).

The mortality rate for acute alcohol withdrawal involving the most severe symptoms ranges from 5-25% (Trevisan et al., 1998)—particularly if the withdrawal is not medically supervised or managed. A person in this state may need to engage in a medically managed [detoxification \(detox\)](#) process in order to be safe. Thus, advising or forcing someone who has severe AUD to abruptly stop drinking is unwise and potentially life-threatening. Quite possibly some deaths attributable to regional natural disasters (e.g., severe hurricanes) may be the result of individuals whose bodies have become dependent on alcohol not having access to alcohol and being forced into acute withdrawal.



Using the BAC calculator link above (or a similar calculator), enter your own data to determine different scenarios.

- How much, how fast would your own BAC estimate reach or exceed 0.08%?
- What drinking pattern would get you to a point of significant impairment (0.10%–0.12%), severe intoxication (0.25%–0.30%), or risk of coma and loss of consciousness (0.35%–0.40%)?

**Alcohol's cognitive effects.** As a CNS depressant, alcohol even at relatively low levels slows the speed of cognitive information processing meaning that it can impair driving and reactions to the point where certain activities become dangerous to self and others. "Alcohol is responsible for approximately half of all trauma deaths and nonfatal injuries in the United States" (<https://www.facs.org/~media/files/quality%20programs/trauma/alcoholinjury.ashx>).

For example, alcohol affects most aspects of perception (the first step in information processing). Not only does it have a general effect on the brain, it has a specific effect on the visual and auditory (hearing) areas. Because alcohol blunts lower sound frequencies involved in speech perception, individuals often begin to speak



more and more loudly as their level of intoxication rises—this is only partly about disinhibition of cognitively controlled behavior, it is also a matter of changes in how sound is perceived. Next in the information processing sequence, alcohol affects memory processes. Encoding new memories, a critical aspect of learning something new, is impaired by heavy alcohol use. An alcohol-induced “blackout” involves interference with encoding information into memory—the memory is not “lost,” it is simply never created.

With chronic heavy alcohol use, memory retrieval becomes increasingly impaired, as well. A memory may have been adequately stored, but the individual may have difficulty retrieving it at will. Together these two functions being impaired help explain state dependent learning in



association with alcohol misuse: when a person learns something new or acquires a memory in an intoxicated state, they may have difficulty retrieving the information later in a sober state. Adolescents and emerging adults in recovery from AUD may find it necessary to repeat large portions of their formal education as the information “learned” during the AUD period may no longer be easily retrieved. Furthermore, heavy alcohol use is associated with the onset and progression of dementia in adulthood.

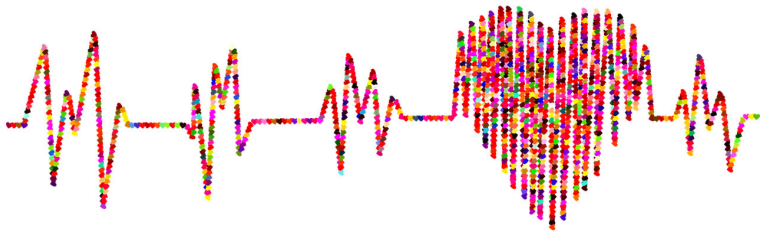
Not only is alcohol implicated in distortions of perception and memory in information processing, it also can impair decision-making and judgment. A significant literature indicates that drinking alcohol impairs a person’s ability to accurately assess risk or feel appropriate anxiety in potentially risk situations. Thus, a person who has been drinking may make risky choices—diving into shallow water, “hooking up” with an unfamiliar sex partner, ignoring “safe sex” practices, deciding to drive despite knowing it is unwise, spending money they cannot really afford.

Alcohol-related brain damage (ARBD) refers to a group of brain changes, resulting in cognitive and other brain impairments, caused by a person's prolonged pattern of alcohol misuse. This occurs in about 30% of persons engaged in prolonged heavy drinking (Dalvi, 2012). An example is diagnosed as Wernicke-Korsakoff syndrome. [Alcohol-related brain damage](#) is differentiated from the acute effects of intoxication because it is a long-term effect. For example, alcohol-related dementia is diagnosed based on symptoms persisting for more than 2 months after cessation of alcohol use. It may be challenging to differentiate from other forms of dementia (e.g., Alzheimers disease or Lewy body dementia). Some reversal of damage is possible with abstinence (Dalvi, 2012).

**Physical health effects.** Not only does alcohol have effects on brain and behavior, it also has effects on physical health. First, alcohol also is a known [teratogen](#), meaning that it disrupts fetal development (see the section concerning fetal alcohol exposure). Second, as previously noted, it plays a role in vulnerability to accidental injury. Third, alcohol use/misuse plays a role in intentional self-harm/suicidality through its disinhibition, impulsivity, and impaired judgment effects (Pompili, et al., 2010). Additionally, alcohol misuse may play a role in suicide risk if it interacts with other, pre-existing mental disorders, stress, social withdrawal/marginalization and loss of social bonds (Pompili et al., 2010). Fourth, alcohol has known effects on cardiac (heart) and circulatory system health. For example, as a CNS depressant alcohol can cause a severe slowing of respiration (breathing), to the point where someone could become oxygen deprived. This risk is multiplied when alcohol is combined with other CNS depressant substances.

Evidence concerning the potential positive effects of drinking one standard drink equivalent of wine daily has come under review—it is not entirely clear that this is an accurate representation of cardiac health promotion and stroke risk reduction. While alcohol use in moderation may help lower “bad” cholesterol (LDL) and/or raise “good” cholesterol (HDL), as well as reduce the tendency to form

blood clots (embolism), these tend to be slight changes and it is not clear that these changes actually improve cardiac health. Furthermore, alcohol misuse is associated with diabetes and high blood pressure. The recommendation is that someone who does not currently drink alcohol should not begin to do so in hopes of improving their health; there are better ways to accomplish this goal (e.g., diet, exercise, medications, and stress-reduction wellness activities).



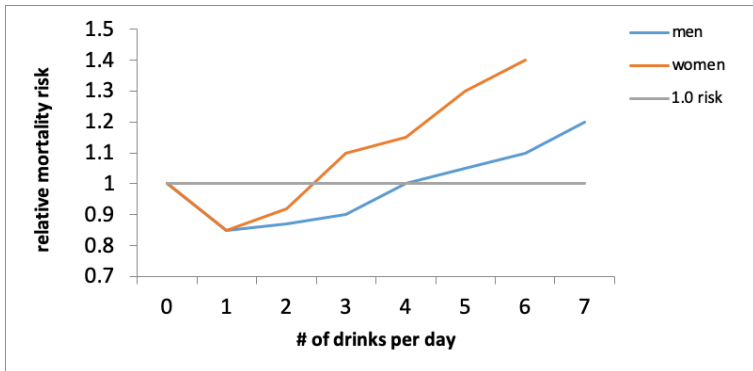
On the other hand, there exists ample evidence that heavy drinking is the enemy of cardiac health. Similarly, heavy alcohol consumption deprives the body of calcium and interferes with vitamin D activation, both of which contribute to bone/skeletal system health. While this can contribute to osteoporosis in men and women, the risk is amplified for women because heavy alcohol consumption also tends to decrease estrogen which accelerates bone loss. Compounding the problem: individuals who engage in heavy drinking also tend toward consuming a diet poor in nutritional value. Fortunately, when a person stops drinking some bone and skeletal health is regained—though not all.

The relationship between alcohol and pain perception is somewhat ambiguous. Alcohol may help raise a person's pain threshold such that it takes a greater amount of stimulation before they experience it as painful. However, individuals



who engage in regular heavy drinking and those whose family history is positive for AUD may be more sensitive to painful stimuli to begin with. Furthermore, they also may be more responsive than other individuals to the pain-reducing effects of alcohol. The degree of overlap between CNS pathways involved in the experienced of pain and the alcohol reward system (pleasure) is notable which may have some power to explain individual differences in response to alcohol.

This graph depicts data presented by O'Keefe, Bybee, and Lavie (2007) concerning the relative risk of death (all causes) in relation to daily alcohol consumption levels. The "neutral" point is a risk value of 1.0 for no daily drinking. The relative risk dropped a bit with one drink per day for both men and women and climbed again for women after 2 and for men after 4 drinks per day. The risk continued to climb more sharply for women but did climb for men, as well, beyond these points. The authors concluded that the effects did not seem to be related to the type of alcohol consumed—the ethanol itself seemed to be the relevant factor. What matters is the amount consumed: too much, too quickly, too often increases health risks.

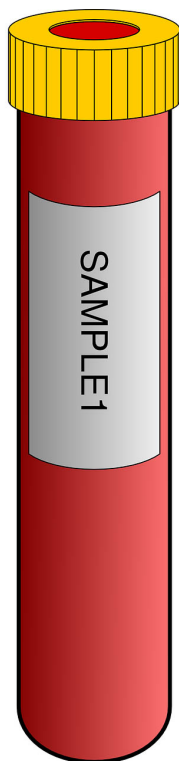


**Alcohol biomarkers.** Because of the way that alcohol is metabolized and the role of its metabolites (breakdown byproducts) in health, it is useful to understand a bit about biomarkers sometimes used in assessment. Biomarkers are objective biological indicators of a substance being present, possibly its concentration, the breakdown products (metabolites) of the substance as it is metabolized, and/or the health of the organ systems affected by the substance (e.g., liver, kidneys, gastrointestinal tract).

**Breathalyzer test.** A breathalyzer is one form of biomarker measure—it provides an estimate of a person's circulating blood alcohol concentration (BAC) or blood alcohol level (BAL). The breathalyzer can detect the presence of alcohol for about 8 to 12 hours after consumption, but it is not clear on a single test whether a person's level is still climbing (the first hour or two after drinking) or dropping as the alcohol is metabolized. It is a somewhat controversial tool. First, some brands/manufacturers have better accuracy than others. Second,



the breath sample must be properly collected. Third, the device must be properly maintained and regularly calibrated.



*Blood, urine, sweat, saliva tests.* A blood test is a direct measure of the amount of alcohol circulating in a person's blood. It is more reliable than a breathalyzer but is also far more invasive. It also relies on two forms of expertise: the blood draw to collect a sample and the laboratory technique to analyze it. Alcohol shows up in a person's urine, sweat, and saliva, as well. These are less reliable as indicators of amount consumed but can detect the presence of alcohol for hours (not days/weeks) after drinking. Some alcohol metabolites, however, can be detected for a few days.

*Clinical tests.* Three tests are commonly utilized to determine the effects of regular heavy alcohol consumption on the liver: the GGT, AST, and ALT. Two tests examine effect on blood and plasma: MCV (size of red blood cells/ability to carry oxygen) and CDT (measuring a serum protein carrying iron through the bloodstream). These tests provide information about a person's health and organ damage; however, disease processes other than AUD may cause their values to be abnormal.

## Fetal Alcohol Exposure

“A significant, pervasive, and persistent alcohol-related public health concern is the potential impact of alcohol use and alcohol use disorders among women of child-bearing age and during pregnancy” (Popova, Rehm, & Shield, 2020). Globally, an estimated 10% of women consumed alcohol during pregnancy, and over 25% engaged in binge drinking during pregnancy (Popova, Lange, Probst, Gmel, & Rehm, 2017). Many adverse pregnancy outcomes have been associated with alcohol use: loss of pregnancy or stillbirth, premature delivery, growth retardation and low birth weight, and fetal alcohol spectrum disorder (Popova, Rehm, & Shield, 2020).

**Fetal Alcohol Spectrum Disorder (FASD).** Prenatal alcohol exposure can cause brain injury resulting in “pervasive, permanent neurodevelopmental differences which impact health, educational, and vocational outcomes” which comprise [\*fetal alcohol spectrum disorder\*](#) (Looock, Elliott, & Cox, 2020). The risk of FASD increases with binge drinking patterns and/or high quantities of alcohol consumption, and when maternal alcohol absorption is increased (low body weight and poor nutrition/fasting); effects are compounded with concurrent exposure to other substances, including use of tobacco products (Looock, Elliott, & Cox, 2020). In one U.S. study, about 1/3 of pregnant women who reported alcohol use in the past 30 days engaged in binge drinking; among pregnant women who engaged in binge drinking did so an average of 4.5 times during the past 30 days (<https://www.cdc.gov/ncbddd/fasd/data.html>).

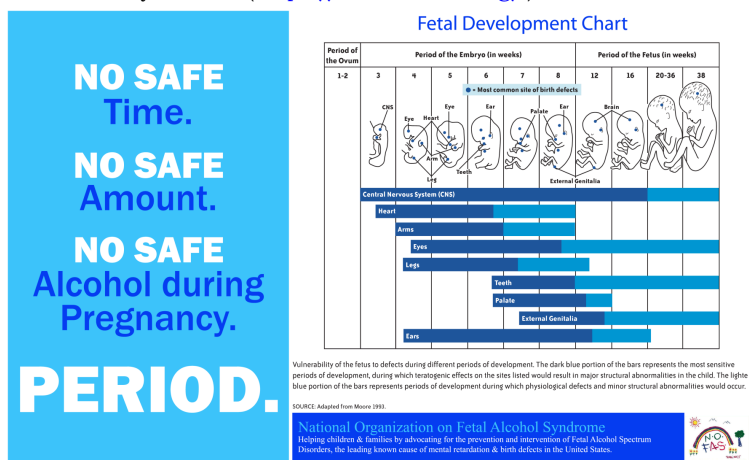


The term “spectrum disorder” is important here because the effects of prenatal alcohol exposure fall extend across a quantitative continuum, as well as varying qualitatively. Some individuals exhibit little or no obvious effects, while others exhibit mild, moderate, or significant differences in physical features or organ system birth defects (malformations of heart, bone, kidney, visual, or hearing systems): [alcohol-related birth defects \(ARBD\)](#). [Note: the abbreviation ARBD is also used to specify [alcohol-related brain damage](#) resulting from alcohol misuse later in life.] Others exhibit [alcohol-related neurodevelopmental disorders \(ARND\)](#), referring to complex differences in neurodevelopment that may affect cognition, information processing, language, behavior, attention, executive function, adaptive skills, mood, hyperactivity, and self-regulation as a child matures (<https://www.niaaa.nih.gov/sites/default/files/>



[ARNDConferenceConsensusStatementBooklet\\_Complete.pdf](#)).

**Fetal alcohol syndrome (FAS)** is considered to be the most severe outcome of prenatal alcohol exposure and may be either full or partial in nature (FAS and pFAS). FAS involves morphological differences (e.g., facial features, growth deficiency) and damage to the central nervous system. Because the effects of alcohol exposure affect so many aspects of fetal growth and development, there is prenatal period when alcohol exposure is considered safe. Because the dose-response is so variable, there is no amount of alcohol considered to be safe during prenatal development—remember that fetal organs are far less efficient at metabolizing substances (including alcohol) than older persons are, thus their relative exposure can be higher for longer than would be true of someone older. Consider this infographic from the National Organization on Fetal Alcohol Syndrome (<https://www.nofas.org/>).



How commonly the full range of FASD occurs in the United States remains unknown; estimates “might number as high as 1 to 5 per 100 school children (or 1% to 5% of the population)” (<https://www.cdc.gov/ncbddd/fasd/data.html>). FAS as the most evident, complicated category is estimated to occur among 3 in 10,000 children aged 7-9 years or up to 90 out of 10,000 children (<https://www.cdc.gov/ncbddd/fasd/data.html>). What this

suggests is that many individuals with whom we engage in daily living or in delivery of human, educational, or health services experience some types of lifelong neurological and/or behavioral effects of prenatal alcohol exposure. Consider also that an infant born with developmental and health challenges related to prenatal alcohol exposure may also enter a social/physical context where parents may be ill-prepared, ill-equipped, or under-responsive to their typical and atypical/exceptional developmental needs.

A group of young adults growing up with FASD conducted a survey of other adults concerning their health as adults prenatally exposed to alcohol (Himmelreich, Lutke, & Hargrove, 2020). They drew several important conclusions from the 541 survey responses:

- Adults with FASD experience vulnerability to a wide range of health conditions, diseases, and disorders, many of which occur at younger ages than in the general population (premature aging).
- FASD over the lifespan is about more than the brain—it is a “whole body” disorder affecting physical and mental health.
- What is “normal” in the general population may not be “normal” for a person with FASD.
- Health and mental health challenges experienced by adults with FASD may be misunderstood, misidentified, misdiagnosed, mistreated, and/or under-served by physical and behavioral health care providers.



### **STOP & THINK**

Answer the following True/False questions based on what you read in Chapter 1.



*An interactive H5P element has been excluded from this version of the text. You can view it online here:*

[https://ohiostate.pressbooks.pub/  
swk5805coursebook/?p=168#h5p-50](https://ohiostate.pressbooks.pub/swk5805coursebook/?p=168#h5p-50)

# Ch. 2: Alcohol Epidemiology & Treatment Needs

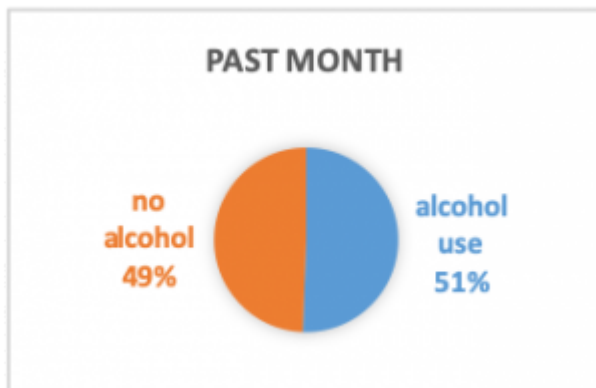
In the United States, a majority of individuals either do not drink alcohol (35%) or do so at low-risk levels (37%), however about 28% drink at levels placing them at risk for AUD or other serious health consequences (NIAAA, 2016).



Before proceeding to read the next section, stop and think about what you expect the epidemiology to say about who engages in alcohol use, binge drinking, and heavy drinking—what age, gender, race/ethnic group data will show and how this might vary by different geographical regions of the United States. When you are done with this section, you will revisit your guesses, so jot them down to refer back to them later.

Here are recent facts and figures for your consideration, derived from the 2018 NSDUH survey data concerning individuals aged 12 and older in the U.S. (SAMHSA, 2019).

Past month alcohol use:



- 51.1% reported having used alcohol in the past month (considered “current use”).
- Past month use rates varied a bit by geographical region: 47.1% in the South, 51.6% in the West, 54.6% in the Midwest, and 54.8% in the Northeast (a more than a 10% difference between the lowest and highest reporting regions).
- The rate was lowest in completely rural areas (43.3%) and highest in large metropolitan areas (52.9%).
- The rate was highest among persons living at 2 times or more of the poverty level (58.4%) and lowest among persons living below the poverty level (34.7%).

Past month binge alcohol use:

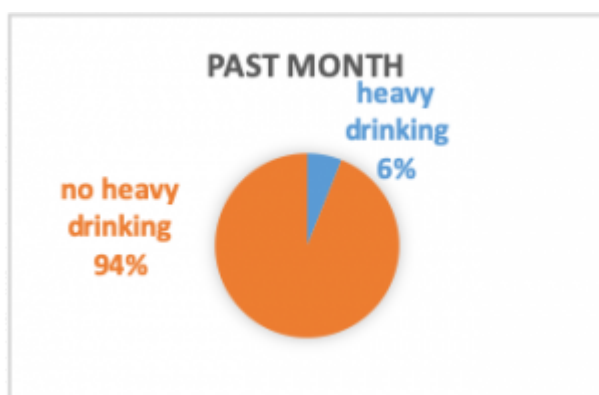


- 24.5%

reported having engaged in binge drinking in the past month.

- Binge drinking rates varied a bit by geographical region: 23.0% in the South, 23.7% in the West, 27.1% in the Midwest, and 25.8% in the Northeast.
- The rate was lowest in completely rural areas (22.0%) and greatest in large metropolitan areas (25.0%).
- The rate was highest among persons living at 2 times or more of the poverty level (25.8%) and lowest among person living below the poverty level (22.3%).

#### Past month heavy alcohol use:



- 6.1% reported heavy alcohol use in the past month.
- Heavy alcohol use varied a bit by geographical region: 5.5% in the South, 6.0% in the West, 6.4% in the Northeast, and 6.8% in the Midwest.
- Heavy alcohol use was higher in completely rural areas (6.6%) than large metropolitan areas (6.0%); it was greatest in less urbanized areas (6.9%) and lowest in small metropolitan areas (5.8%).
- The rate of heavy alcohol use was highest among persons living at 2 times or more of the poverty level (6.6%), lowest among persons living at or up to 2 times the poverty level (5.0%), and between these rates among persons living below the poverty level (5.3%).

In short, it seems a majority of individuals who currently drink alcohol do so within reason—less than binge or heavy drinking patterns. There existed notable variability in alcohol use behavior based on gender, age, and race/ethnicity reported among persons of legal drinking age (21 years) in the NSDUH 2018 data, as well (SAMHSA, 2019).

Demographic Group	Past Month Alcohol Use	Past Month Binge Drinking	Past Month Heavy Drinking
Gender: male female	60.8% 52.4% %	31.5% 22.1%	9.2% 4.4%
Race/Ethnicity: White Black or African American Asian Hispanic or Latino American Indian or Alaskan Native Native Hawaiian or Other Pacific Islander 2 or more races	61.1% 49.0% % 43.9% % 48.2% % 39.7% % 38.5% % 53.7%	27.2% 26.3% 16.0% 28.4% 24.8% 26.8% 26.8%	7.7% 5.0% 2.9% 5.0% 7.1% 5.9% 6.6%
Age: 12-17 18-25 26 and older	9.0% 55.1% 55.3% %	4.7% 34.9% 25.1%	0.5% 9.0% 6.2%

As you can see, men were more likely than women to engage in all three drinking patterns, and the difference was most dramatic in the heavy drinking category (more than twice the rate). The difference between emerging adults (18-25) and adults (26 and older) was apparent in both binge and heavy drinking categories; the rate of binge drinking among adolescents (12-17) was just over half the rate for alcohol use in this age group. The racial/ethnic

group with the highest rate of alcohol use or heavy drinking, and second highest rate of binge drinking, were individuals identifying as white. While alcohol use among black or African American survey respondents was third greatest (behind those identifying with two or more races or as white) and binge drinking tied as second highest, heavy drinking was tied for second lowest (above only Asian respondents). And, while alcohol use was second lowest among American Indian or Alaskan Native respondents, this group was second highest for heavy drinking. All together these statistics contradict some common stereotypes about drinking behavior and shed light on who among the U.S. population is most likely to engage in risky drinking patterns (binge or heavy drinking).



After reading about the epidemiological data, stop and think about what you expected the epidemiology to say and what you just learned about who engages in alcohol use, binge drinking, and heavy drinking—what age, gender, race/ethnic group data showed and how this varied by different geographical regions of the United States.

- Where differences occurred, why do you think your guesses were different from the evidence?



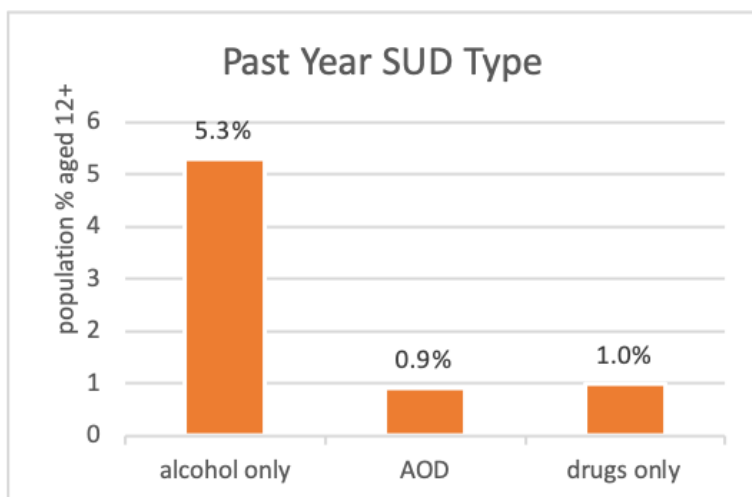
- How do you think the data relate to stereotypes and stigma in our society and policies?

## Treatment Needs

Among all 2018 NSDUH survey respondents, 3.9% perceived a need for specialized alcohol treatment during the past year; 1.6% made an effort to receive treatment (SAMHSA, 2019). The observed discrepancy between needing and seeking treatment persisted across age groups, with the perceived need more than doubling in each older age group.

Age group	Perceived need	Effort to receive	No effort to receive
12-17	1%	0.7%	0.3%
18-25	2.1%	0.7%	1.4%
26 and older	4.6%	1.9%	2.7%

You may recall from Module 1 in our course that individuals who have a diagnosable substance use disorder most commonly have a problem with alcohol. From the 2017 NSDUH survey (SAMHSA, 2018), over 19.7 million individuals aged 12 or older (7.2% of population) were estimated to experience a substance use disorder (SUD) involving alcohol and/or and illicit drug use during the past year; the vast majority involved alcohol alone (5.3%) or in combination (0.9%) with illicit drugs (alcohol and other drugs, AOD), leaving 1% with an illicit drugs-only form of SUD.



**When individuals seek treatment for alcohol problems.** A study involving 47 individuals followed two years after participating in a treatment study for alcohol use disorder (COMBINE Project) reported the ages at which they first experienced diagnostic criteria of alcohol use disorder versus when they first identified their drinking pattern as problematic and first attempted to cut down or abstain from drinking (Begun, Berger, Salm Ward, 2011). The difference in mean “first” age was more than 10 years (see Table adapted from Begun, Berger, Salm Ward, 2011, p. 113), a finding consistent with other literature concerning the discrepancy in ages between when AUD becomes a problem and individuals first seek treatment.

Drinking “First”	Age Range	Mean Age
drinking for longer periods than intended	14-40	21.77
drinking greater amounts than intended	14-45	24.54
family or friend complaints or concerns about the drinking	16-55	27.45
need to consume greater amounts (developing tolerance)	15-55	29.59
physical withdrawal symptoms when not drinking	13-53	32.40
identified drinking pattern as problematic	15-55	32.32
attempted to cut down or abstain from drinking	16-61	34.83

These participants reported having made 1-11 significant attempts to change their drinking behavior over their life course, with the mean number of attempts being 3.4 (median of 3 attempts). Approximately equal numbers of first change attempts were either self-change (32%) or formal treatment attempts (34%); “By the second attempt, the proportion had tipped heavily in favor of formal strategies (51%), and self-change was much less frequent than before (22%)” (Begun, Berger, Salm Ward, 2011, p. 114). Individuals employ a variety of strategies when attempting to change their alcohol use/misuse behavior.

**Is formal treatment necessary?** This question is heavily debated in the literature and in practice: “Researchers and practitioners are only beginning to understand the nature and significance of change attempts that occur outside of formal treatment” (Begun, Berger, & Salm Ward, 2011, p. 105). A convincing body of evidence indicates that many individuals are able to successfully change their problematic drinking behavior without engaging in formal,

specialized alcohol treatment—self-change attempts alone may suffice (Sobell, Cunningham, & Sobell, 1996; Sobell, Cunningham, Sobell, & Tonneato, 1993) or in conjunction with other informal and formal treatment interventions (DiClemente, 2006). Another convincing body of evidence concerns the effectiveness for many individuals of brief intervention delivered outside of specialized alcohol treatment programs (Zweben & West, 2020). These alternatives may suffice for individuals engaged in alcohol misuse without meeting diagnostic criteria for AUD or, perhaps, on the mild end of the continuum for an AUD.

For individuals meeting diagnostic criteria for AUD, particularly in the more severe range, formal AUD treatment may be needed. Options vary and include behavioral counseling (e.g., cognitive behavioral/coping skills training interventions, contingency management, community reinforcement and family training, behavioral couples or family involved therapies), medication-assisted treatment, and combinations of these options (Zweben & West, 2020). The American Society of Addiction Medicine (ASAM) has established a set of guiding decision rules to help determine the appropriate level of care related to assessment of individuals in need of alcohol (or other substance) treatment intervention. The [ASAM levels of care](#) guidelines reflect a continuum of care options indicating increasing intensity in the levels of care on a 5-point scale (0-4):

- No intervention (0)
- Early intervention (0.5)
- Outpatient services (1)
- Intensive outpatient services (2.1)
- Partial hospitalization services (2.5)
- Clinically managed low-intensity residential services (3.1)
- Clinically managed population-specific high-intensity residential services (3.3)
- Clinically managed population-specific high-intensity residential services (3.5)

- Medically monitored intensive inpatient services (3.7)
- Medically managed intensive inpatient services (4)

Detoxification (detox) is an example of a process that could take place at several of these levels, depending on the assessed need (i.e., 2.1 to 4). The goals of detox services are:

(1) safely manage the initial, acute withdrawal period (hours to days after ceasing alcohol use) and ensure the person is medically stabilized;

(2) engage the individual in longer-term treatment plan.

Recovery from alcohol use disorders, with or without formal treatment, may best be supported with appropriate case management or wrap-around supportive services (Zweben & West, 2020), as well as peer support and/or mutual help program participation (Bersamira, 2020; Zweben & West, 2020).

# Ch. 3: Alcohol and Social Contexts

In this module you learned about the ways that social and physical contexts relate to substance use behavior—initiation, use, misuse, and treatment/recovery behaviors. You are assigned two articles here to consider.

**The role of social media (Twitter).** This article looks at Twitter chatter and its potential impact on peer drinking—the mechanisms of influence involve social norms and social learning theory. The article is: Cavazos-Rehg, P.A., Krauss, M.J., Sowles, S.J., & Bierut, L.J. (2015). “Hey everyone, I’m drunk: An evaluation of drinking-related Twitter chatter. *Journal of Studies on Alcohol and Drugs*, 76(4), 635-643. This article addressed:

- the place of online social networks in our understanding alcohol use/misuse behavior
- the “pro alcohol” bias in Tweets
- alcohol marketing in the Twittersphere
- implications for addressing/preventing alcohol misuse

**The role of drinking context on sexual aggression.** This article makes a strong case for how drinking contexts (bars, parties, home) relate to drinking consumption and outcomes. Not only is how much and how fast a person drinks important (dose and BAC/BAL), the social aspects of alcohol use seem to matter, too. In other words, we cannot fully understand the consequences of drinking behavior without understanding the contexts in which it occurs. The article is: Testa, M., & Cleveland, M.J. (2017). Does alcohol contribute to college men’s sexual assault perpetration? Between- and within-person effects over five semesters. *Journal of Studies on Alcohol and Drugs*, 78(1), 5-13. This article addressed:

- evidence concerning [\*heavy episodic drinking \(HED\)\*](#) and sexual aggression
- the role of drinking context (parties and bars) in sexual aggression
- the role of personality characteristics in sexual aggression and drinking behavior

## Ch. 4: Summary

In this module, you were introduced to the first of our examinations of a specific type of substance: alcohol was the focus. You learned what alcohol is and how it is processed in the human body (metabolized). You also learned that much of the physical harm related to drinking to excess has to do with the first-step metabolite, acetaldehyde. Dosing of alcohol was examined in terms of alcohol concentrations (ABV and “proof” designations), as well as in terms of volume consumed—and how standard drink equivalents are calculated. This led to a presentation concerning low-risk drinking guidelines (defining “moderation”) and how binge and heavy drinking are defined. A number of health and cognition/behavior risks associated with alcohol misuse were explored, including the topic of prenatal alcohol exposure and resulting FASD. Biomarkers for alcohol consumption and alcohol effects were presented, as well. You read about the meaning of blood alcohol concentration (BAC) and how it might be estimated using interactive tools. In the second chapter, you explored epidemiological information concerning drinking behavior and possibly confronted some common stereotypes. The gap between the identified need for alcohol treatment and seeking treatment was explored, along with different options for changing one’s alcohol-related behavior—when formal treatment might be in order was considered. One factor that tends to promote alcohol misuse during adolescence and emerging adulthood was next in our list of topics: how social media (Twitter in particular) may play a role by influencing both social norms and social learning. Finally, we examined evidence concerning the significance of drinking contexts in determining drinking consequences; specifically, men’s perpetration of sexual aggression. At this point, you are prepared to review the key terms presented in Module 7 and to move forward to our next module.



# Module 7: Key Terms

**acetaldehyde:** an alcohol metabolite (also present in tobacco smoke) that is carcinogenic, mutagenic, and toxic

**alcohol by volume (ABV):** a universal measure of alcohol concentration in beverages, refers to milliliters of pure ethanol in 100 milliliters of the beverage (at 68o F) converted to a percentage.

**alcohol dehydrogenase (ADH):** an enzyme abundant in the liver and involved in first-step metabolism of alcohol.

**alcohol poisoning:** term used for alcohol overdose resulting from drinking too much too quickly, raising blood alcohol concentration to high levels; potentially fatal.

**alcohol proof:** an indication of alcohol content in a beverage; in the U.S. proof is twice the ABV percentage.

**alcohol-related birth defects (ARBD):** term covering a variety of known morphological and organ system changes resulting from prenatal alcohol exposure. *Note: ARBD as an abbreviation may also refer to alcohol-related brain damage experienced later in life as a result of alcohol misuse.*

**alcohol-related brain damage (ARBD):** term covering a range of central nervous system changes (often characterized by dementia) caused by an adult's prolonged alcohol misuse. *Note: ARBD as an abbreviation may also refer to alcohol-related birth defects.*

**alcohol-related neurodevelopmental disorders (ARND):** refers to a range of neurodevelopmental and behavioral disabilities resulting from prenatal alcohol exposure.

**aldehyde dehydrogenase (ALDH):** an enzyme abundant in the liver involved in second-step metabolism of alcohol (metabolizes acetaldehyde produced in first-step).

**ASAM levels of care:** set of guidelines established by the American Society of Addiction Medicine matching recommended treatment

intensity to assessment of individuals in need of alcohol or other substance misuse/use disorder treatment.

**binge drinking (heavy episodic drinking):** consuming 4 or more drinks within about 2 hours by women and 5 or more drinks in 2 hours by men.

**blood alcohol concentration (BAC):** refers to the percent of ethanol in a person's blood (sometimes referred to as blood alcohol level, or BAL).

**delirium tremens:** confusion and other symptoms (e.g., shaking, shivering, irregular heart rate, sweating) related to alcohol withdrawal in some persons who have a history of heavy drinking.

**denatured alcohol:** ethanol that has been altered by additives to discourage consumption; may be poisonous/toxic or simply unpleasant (taste/smell).

**detoxification (detox):** a first step intervention to manage withdrawal from alcohol (or other substance) and used as a prelude to entering treatment.

**ethyl alcohol (ethanol or “grain” alcohol):** names for the main chemical in alcoholic beverages (beer, wine, spirits).

**fetal alcohol spectrum disorder (FASD):** a continuum of conditions related to prenatal alcohol exposure.

**fetal alcohol syndrome (FAS):** on the FASD continuum, involving brain damage, impaired growth, and specific morphological differences of the face/head; may be full or partial outcome.

**heavy drinking:** 8 or more drinks per week for women and 15 or more drinks per week for men.

**heavy episodic drinking (HED):** see binge drinking

**high-risk drinking:** 4 or more drinks on any day (binge drinking) or 8 or more drinks per week for women (heavy drinking); 5 or more drinks on any day (binge drinking) or 15 or more drinks per week for men (heavy drinking).

**standard drink measure:** a way of indicating alcohol consumption, each standard drink equivalent is determined as 14 grams of pure ethanol in a beverage.

**teratogen:** any factor that disrupts fetal development, such as

chemicals (including alcohol, tobacco, and other drugs), x-rays, viral or bacterial infections.

# Module 7: References and Image Credits

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## PART VIII

# MODULE 8: FOCUS ON SEDATIVE-HYPNOTICS & CNS DEPRESSANTS

### Introduction

In this module concerning sedative-hypnotic and central nervous system (CNS) depressant drugs we extend what was learned about alcohol in Module 7 where we identified alcohol as having a CNS depressant effect. Here in Module 8, we explore what these different types of substances are, how they are typically used and misused (including a group of “date rape” drugs), their effects, and possible effects associated with prenatal exposure. Content presented in this module informs and was informed by Begun, A.L. (2020). Introduction to psychoactive substances. In A.L. Begun & M.M. Murray, (Eds.), *Routledge handbook of social work and addictive behavior*. London: Routledge.

### Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Describe the nature of sedative-hypnotic and CNS depressant drugs and their effects mind, body, behavior and health;
- Describe how these types of drugs might be used and/or misused;
- Identify potential effects on developmental outcomes of

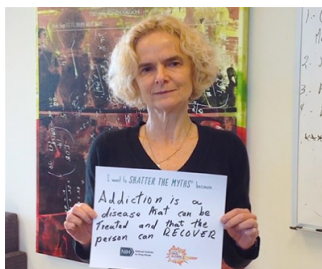
prenatal exposure to these substances;

- Explain patterns of use, misuse, and use disorders associated with these substances and the risks when combining them with alcohol;
- Identify “date rape” and “club” drug related issues;
- Explain key terms and concepts related to this category of substances.

# Ch. 1: Nature and Effects Sedative-Hypnotics and CNS Depressants

The topic of sedative-hypnotics and CNS depressants overlaps considerably with the topic of prescription drug abuse (more about this in Module 12). Whether used as prescribed or used without a prescription, a drug still has the same actions on the brain, body, and behavior. Dr. Nora Volkow, director of the National Institute on Drug Abuse (NIDA) made the following statement to the United States Congress on September 22, 2010:

CNS depressants, typically prescribed for the treatment of anxiety, panic, sleep disorders, acute stress reactions, and muscle spasms, include drugs such as benzodiazepines (e.g.,



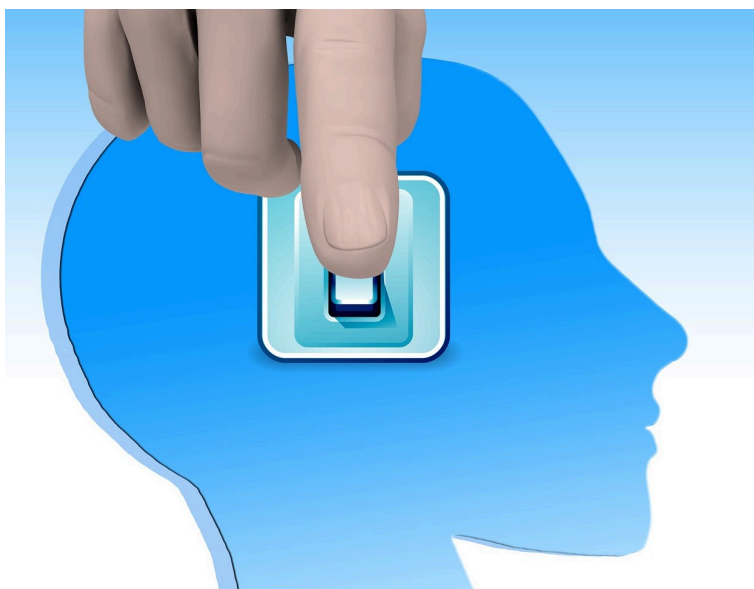
Valium, Xanax) and barbiturates (e.g., phenobarbital)—which are sometimes prescribed for seizure disorders. Most CNS depressants act on the brain by affecting the neurotransmitter gamma-Aminobutyric acid (GABA), which works by decreasing brain activity. CNS depressants enhance GABA's effects and thereby produce a drowsy or calming effect to help those suffering from anxiety or sleep disorders. These drugs are also particularly dangerous when mixed with other medications or alcohol; overdose can suppress respiration and lead to death. The newer non-benzodiazepine sleep medications, such as zolpidem (Ambien), eszopiclone (Lunesta), and zaleplon

*(Sonata), have a different chemical structure, but act on some of the same brain receptors as benzodiazepines and so may share some of the risks—they are thought, however, to have fewer side effects and less dependence potential.*

This statement summarizes many of the key points relevant to Module 8 concerning the nature, effects, and use of sedative-hypnotic and CNS depressant drugs. Let's look into these points in a little more detail.

## What are Sedative-Hypnotics and CNS Depressants?

Sedative-hypnotic, tranquilizer, and central nervous system (CNS) depressant drugs slow down brain activity, calming brain excitability. This effect is typically mediated through enhancing the activity of GABA neurotransmitter activity (Begun, 2020)—GABA (gamma-aminobutyric acid) is one of the brain's main inhibitory neurotransmitters and plays a key role in the regulation of anxiety ([https://thebrain.mcgill.ca/flash/i/i\\_01/i\\_01\\_m/i\\_01\\_m\\_ana/i\\_01\\_m\\_ana.html](https://thebrain.mcgill.ca/flash/i/i_01/i_01_m/i_01_m_ana/i_01_m_ana.html)). The result is a general calming influence on anxiety and acute stress reactions; sleepiness or drowsiness may also be induced. These types of drugs are often used medically in the treatment or management of conditions like anxiety or panic disorders (anxiolytics), acute stress, insomnia (sleeplessness/sleep disorder), epilepsy/seizure disorders, or muscle spasms (tranquilizers). **Sedative** compounds produce a calming effect, reducing excitability in the central nervous system, while **hypnotic** compounds induce sleep or intense drowsiness (Dupont & Dupont, 2005; NIDA, 2018)—switching off brain activity.



Common forms of sedative-hypnotic and CNS depressants, other than alcohol, identified by NIDA (2018) include:

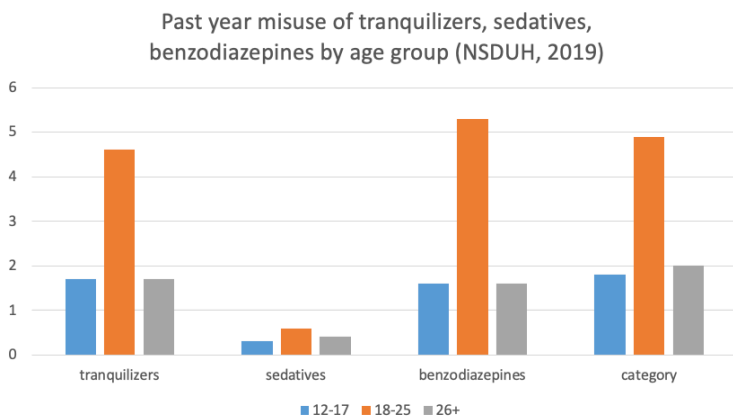
- **benzodiazepines** (e.g., diazepam/Valium®, clonazepam/Klonopin®, alprazolam/Xanax®, lorazepam/Ativan®, triazolam/Halcion®, estazolam/Prosom®, chlorodiazepoxide/Librium®) [street names include: candy, downers, tranks/tranqs] [note that “bennies” are not benzodiazepines, they refer to a brand of amphetamine] [other names for Xanax®: bicycle parts or bicycle handle bars; for Klonopin®: benzos, K, K-Pin, Super Valium  
<https://ndews.umd.edu/sites/ndews.umd.edu/files/dea-drug-slang-terms-and-code-words-july2018.pdf>]
- **barbiturates** (e.g., mephobarbital/Mebaral®, phenobarbital/Luminal®, pentobarbital sodium/Nembutal®, amobarbital/Amytal®, butabarbital/Butisol®) [street names include: barbs, phennies, reds, yellows, yellow jackets]
- **non-benzodiazepine sedative hypnotics/sleep medications** (zolpidem/Ambien®, eszopiclone/Lunesta®, zaleplon/

Sonata®) [street names include: sleep medications or references to sleep/forgetting]

Since these drugs are most often prepared in pill, capsule, or liquid form, they are most often swallowed. However, a form of misuse involves crushing the pills or emptying the contents of a capsule and either inhaling (“snorting”) or injecting the contents. These modes of administration bypass the digestive system and produce a more immediate and possibly more intense response. They also involve additional health risks—such as, risk of infection and communicable disease transmission (HIV, hepatitis) from shared needles. Less commonly used outside of medical/hospital settings are anesthetic drugs, such as propofol (contributing to the death of singer Michael Jackson). Anesthetics used in medical (and veterinary) settings may be in an oral form or a form to be injected, administered intravenously (IV; e.g., propofol), or inhaled as a gas (e.g., nitrous oxide).

## Epidemiology

In the United States, the misuse of [tranquilizers](#) or sedatives is less common than for many other types of psychoactive substances. According to data from the National Survey of Drug Use and Health (NSDUH, 2019), an estimated 0.7% of individuals aged 12 and older engaged in the current (past month) misuse of these types of substances during 2018. However, in the past year, 2.0% reported benzodiazepine misuse, 2.1% reported tranquilizer misuse, and 0.5% reported sedative misuse. The age group most likely to report past year misuse of these substances occurred among emerging adults aged 18-25 years.



## Effects

The CNS effects of sedative-hypnotic compounds occur on a continuum, “depending on the dose, beginning with calming and extending progressively to sleep, unconsciousness, coma, surgical anesthesia, and, ultimately, to fatal respiratory and cardiovascular depression” (Dupont & Dupont, 2005, p. 219). The effects at low doses are not unlike the effects of alcohol—impaired cognitive and motor functioning—and the sedating effect of many antihistamines. Use as prescribed or misuse can be accompanied by the following effects (NIDA, 2018):

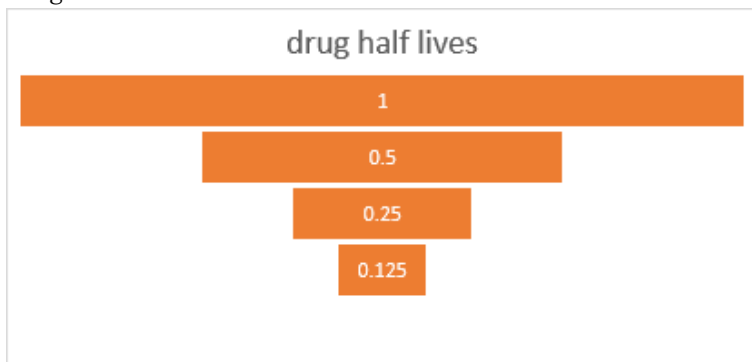
- slurred speech
- poor concentration
- confusion
- memory problems
- headache
- light-headedness/dizziness
- dry mouth

- uncoordinated movements
- low blood pressure
- slowed breathing rate.

Drugs in this group are potentially addictive, some with much greater addictive potential than others. These drugs have different DEA scheduling assignments depending on their addictive potential and approved medical uses in the U.S., ranging from Schedule I to IV (see [https://d14rmgtrwzf5a.cloudfront.net/sites/default/files/nida\\_commonlyabuseddrugs\\_rx\\_final\\_printready.pdf](https://d14rmgtrwzf5a.cloudfront.net/sites/default/files/nida_commonlyabuseddrugs_rx_final_printready.pdf)).

Combining CNS depressants with other substances is potentially dangerous. For example, both alcohol and benzodiazepines have the effect of slowing/suppressing respiration. Thus, if these two substances are combined, the risk of someone's breathing being dangerously slowed or stopped increases since the respiratory effects are additive.

Different forms of sedative-hypnotic and CNS depressant drugs have different half-lives, meaning that some are longer-acting than others. Consider, for example, the news media coverage of celebrities reporting sedating, confusion, coordination, and amnesic effects of non-benzodiazepine sleep medications 8 hours after these drugs were consumed for insomnia (e.g., Tiger Woods, Charlie Sheen, Elon Musk, Sean Penn). Some others in this category of drugs wear off in 2-4 hours.



**Tolerance and withdrawal.** Tolerance is relatively quickly



developed with repeated administration of barbiturates, contributing to a person's likelihood of increasing the dose used over time (Dupont & Dupont, 2005). Tolerance can develop to any of the sedative hypnotic and CNS depressant drugs (NIDA, 2018). In addition, individuals using barbiturates may also develop [cross-tolerance](#) to benzodiazepines and to alcohol (Dupont & Dupont, 2005)—meaning that a person who switches type of drug within this type may already experience tolerance to the new drug. This, too, contributes to the risk of overdose. Overdose with these drugs is dangerous because of the drugs' effects on breathing—slowing it down or stopping breathing to the point of brain damage from hypoxia (lack of sufficient oxygen to the brain), coma, or death (NIDA, 2018). Overdose from benzodiazepines can be treated as an emergency situation with a benzodiazepine receptor antagonist drug (e.g., flumazenil injection). Withdrawal symptoms from CNS depressants include (NIDA, 2018):

- intense cravings
- seizures
- anxiety/agitation
- insomnia
- overly active reflexes, shakiness
- increased heart rate
- increased blood pressure
- increased body temperature
- hallucinations.

Medically managed withdrawal and detoxification from these drugs (particularly barbiturates), just as in the case of alcohol withdrawal, is recommended given the potential severity of acute withdrawal symptoms (including seizures). Ideally, the dose is gradually reduced over time (“weaning” form of detoxification) or safer substitute medications are used to taper off the primary drug.

## Fetal Exposure

The evidence concerning teratogenic effects of benzodiazepines is somewhat unclear and inconsistent, possibly due to variations in study methodology and study participants. An early review indicated that the majority of prenatally exposed infants developed normally and that the few showing neurodevelopmental deficits “caught up” by 4 years of age (McElhatton, 1994). However, a subsequent study demonstrated a behavioral effect (increased internalizing behavior) among toddler/pre-school aged children experiencing long-term prenatal exposure to benzodiazepines compared to unexposed siblings—an effect unlikely to be attributable to environmental factors (Brandlistuen et al., 2017). The authors indicate that these drugs do cross the placental barrier, meaning that there is a potential for affecting fetal development. The greatest health and developmental risks of prenatal exposure to these drugs appear to occur late in the final trimester and during birth with babies exhibiting listlessness (“floppy infant syndrome”), apnea (interrupted breathing), and/or neonatal withdrawal symptoms (McElhatton, 1994).





An interactive H5P element has been excluded from this version of the text. You can view it online here:

<https://ohiostate.pressbooks.pub/swk5805coursebook/?p=180#h5p-51>

# Ch. 2: “Date Rape” and "Club" Drugs

The U.S. National Institute on Drug Abuse (NIDA) identified the following as “club drugs” because they frequently are (mis)used recreationally in nightclub, concert, or rave venues:

- Ecstasy/MDMA,
- GHB,
- ketamine,
- Rohypnol (flunitrazepam),
- methamphetamine, and
- LSD (lysergic acid diethylamide).

Four of these drugs are also specified as “club drugs” by the U.S. Office of National Drug Control Policy (ONDCP):

- MDMA,
- GHB,
- ketamine, and
- Rohypnol (Chakraborty, Neogi, & Basu, 2011).

Some of these drugs are difficult to detect when dissolved in a beverage, being odorless, relatively tasteless, and colorless. Reputable manufacturers are adding dyes to their products so that they will show up in a clear beverage (e.g., Hoffman-La Roche add a

blue dye to Rohypnol pills). Harm/risk reduction strategies related to these substances include avoiding alcohol if their use is



intentional, not leaving a beverage unattended, and having friends in attendance looking out for each other (Chakraborty, Neogi, & Basu, 2011).

In this module focused on sedative-hypnotic and CNS depressant drugs, we take a closer look at the club/date rape drugs GHB, ketamine, and Rohypnol; other club drugs will be explored in future modules concerning stimulant or hallucinogenic substances. Sources for the information below include U.S. Drug Enforcement Administration (DEA) drug facts websites and the National Institute on Drug Abuse (NIDA) drug facts websites.

### **GHB**

The chemical name for GHB is gamma-hydroxybutyrate sodium; “street” or “club drug” names include G, grievous bodily harm, firewater, scoop, poor man’s heroin, liquid ecstasy, and liquid X. The prescription form, Xyrem® (sodium oxybate), is either a liquid or white powder that dissolves in liquids. Illicit preparations include liquids, pills, capsules, and powder forms. GHB is a CNS depressant with intoxicating effects similar to some experienced with alcohol use; GHB amplifies the effects of alcohol when they are combined (Chakraborty, Neogi, & Basu, 2011). It has been approved for prescription use in the U.S. to treat certain forms of narcolepsy; in some European practices, Xyrem® has been used to treat alcohol use disorders and alcohol withdrawal syndrome.

The acute effects of GHB last 2–6 hours and include hallucinations, euphoria, drowsiness, decreased anxiety, aggression, and enhanced libido. At moderately low doses GHB has an amnesic effect and/or causes confusion. At a moderate to high dose it causes hallucinations, respiratory depression and/or apnea (impaired breathing), unconsciousness, and possibly coma or death—especially when combined with alcohol. GHB has relatively high addictive potential and potentially dangerous withdrawal symptoms. GHB overdose is also potentially deadly due to its effects on breathing and potential for coma; there is no antidote or reversal medication for GHB overdose.

### **Ketamine**

Ketamine is a relatively short-acting anesthetic with human and veterinary medicine uses. Ketamine can induce feelings of calmness and relaxation, along with cognitive impairment (attention, learning, memory), as well as pain relief, loss of body control/immobility, and amnesia. It is misused for its hallucinogenic effects, creating a dream-like state and/or altered perceptions. While it could be discussed in the module concerning hallucinogenic substances, it is placed here because (1) it is sometimes used as a “date rape” (sexual assault) drug and (2) in high doses it can cause amnesia, agitation, unconsciousness, depression, and respiratory problems. Flashback experiences have been reported with ketamine use.

The “street” or “club drug” names for ketamine include special K, K, super K, vitamin K, kit kat, keets, jet, purple, and cat valium. Ketamine comes in liquid form that can either be injected or mixed with other liquids, or a powder that is mixed in drinks, snorted, or smoked (often in combination with cannabis or tobacco). Acute effects include rapid heartrate, high blood pressure, and depressed breathing/apnea events, and use is associated with injury because of its suppression of pain responses (Chakraborty, Neogi, & Basu, 2011). Ketamine is potentially addictive and chronic use may lead to reduced pain sensation, loss of coordination, difficulty with concentration, insomnia, drowsiness, slurred speech, and bladder incontinence. Withdrawal symptoms include loss of appetite, fatigue, irregular heartbeat, anxiety, depression, tremors, sweating and chills, as well as nightmare disturbed sleep. Ketamine is often used in a mixed sequence of polydrug use involving stimulants (e.g., methamphetamine, cocaine) or heroin (Chakraborty, Neogi, & Basu, 2011). It is a Schedule III drug in the U.S.

### **Rohypnol**

The generic name for the drug Rohypnol is flunitrazepam; it is known by various “street” or “club drug” names, most commonly as ruffies or rophies, but also as circles, forget me pills, R2, and roche. It can be dissolved in liquids (especially carbonated beverages), swallowed as a pill, or crushed and snorted. Rohypnol is a benzodiazepine depressant, intended as a pre-surgical anesthetic,

muscle relaxant, sleeping, or anxiolytic (anti-anxiety) medication, but is not an approved prescription drug in the U.S. (Schedule I).

It can cause drowsiness/sleep, relaxation/calmness, amnesia for events occurring while under its influence, slurred speech, confusion, impaired mental function/impaired judgement, dizziness, loss of coordination/impaired motor function, aggression, decreased blood pressure, drop in body temperature, and slowed breathing. These effects contribute to its reported use as a “date rape” (sexual assault) drug with the onset of effects being fairly quick and the effects persisting for 8-12 hours. With repeated use, Rohypnol is potentially addictive, and its effects are amplified with alcohol. Overdose with Rohypnol is potentially fatal due to its breathing and coma effects; a benzodiazepine antagonist (flumazenil) may help reverse the effects of flunitrazepam overdose.



Visit the website hosted by the U.S. Department of Health & Human Services concerning date rape drugs at <https://www.womenshealth.gov/a-z-topics/date-rape-drugs>.

Read through the responses to the set of questions linked at this site. Create a 1-page handout/flyer of what you believe is the most relevant information that would be appropriate and important to post in women's restrooms, for shared-ride drivers to hand to

passengers, and for physicians to provide to their patients (including transgender patients).



## Ch. 3: Summary

In this module, you were introduced to the category of sedative-hypnotic, tranquilizer, and central nervous system (CNS) depressant drugs. While these psychoactive substances are among the least commonly misused, they are misused by about 2% of the population over the age of 12—particularly emerging adults in the 18-to-25-year age range. Many effects are similar to those we learned about in the module about alcohol and you learned that combining these drugs with alcohol amplifies some of the risky effects (e.g., breathing complications), contributing to overdose harm. Some of these drugs have significant addictive potential, including the development of tolerance and (potentially dangerous) withdrawal. In this context, you learned the concept of cross-tolerance between drugs of a similar type. Additionally, you learned about the related group of “club drugs” and how these are sometimes used to perpetrate sexual assault (“date rape”). It is difficult to assess the risks of prenatal exposure to drugs in this category, although infants may be born with withdrawal symptoms and some neurodevelopmental effects may persist over time.

# Module 8: Key Terms

**benzodiazepines:** a class of (tranquilizer) psychoactive drug used to treat anxiety, seizures, insomnia, or as a muscle relaxant; may be used in managing alcohol withdrawal under medical supervision.

**barbiturates:** a class of sedative, CNS depressant sleep-inducing drugs, sometimes used for treatment of headache, insomnia, and seizure disorders.

**cross-tolerance:** developing resistance to a specific substance due to repeated exposure to a similar substance, even if that specific substance was not previously used.

**hypnotic:** compound that promotes sleep or drowsiness.

**non-benzodiazepine sedative hypnotics/sleep medication:** drugs with sleep-promoting effects similar to benzodiazepines without or with less significant their common negative effects, such as rebound insomnia (insomnia induces by stopping their use), withdrawal, tolerance, respiratory depression, memory impairment.

**sedative:** compound producing a calming effect and/or reducing excitability in the central nervous system.

**teratogen:** any factor that disrupts fetal development, such as chemicals (including alcohol, tobacco, and other drugs), x-rays, viral or bacterial infections.

**tranquilizers:** medications used to decrease anxiety and increase relaxation/calm state.

# Module 8: References and Image Credits

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## Image Credits

Image of Dr. Nora Volkow with “Addiction is a disease that can be treated...” sign: <https://teens.drugabuse.gov/blog/post/nida-director-kicks-ndafw-and-chat-day> from NIDA, January 22, 2019.

## PART IX

# MODULE 9: FOCUS ON CANNABIS & OTHER HALLUCINOGENIC/ DISSOCIATIVE SUBSTANCES

## Introduction

The next category of substances to study in this course includes cannabis and other **hallucinogenic** substances (including those with **dissociative** effects). A great deal of controversy has emerged in public discourse, policy, public health, and criminal justice concerning the current views and (il)legal status of many of these substances. Consider all that you have been hearing, and perhaps voting, concerning medical and recreational marijuana use, for example. Consider also how cannabis and some of these other hallucinogenic substances are portrayed in the mass media and social media, discussed by public health, mental health, and addiction professionals, and how they might be considered in your own family and peer groups. It is likely that you will conclude that the U.S. society is highly ambivalent concerning cannabis and some of the other hallucinogenic substances most commonly used. Content presented in this module informs and was informed by Begun, A.L. (2020). Introduction to psychoactive substances, appearing in A.L. Begun & M.M. Murray, (Eds.), *The Routledge Handbook of Social Work and Addictive Behaviors*. NY: Routledge.

## Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Describe the nature, common uses, and effects of cannabis and other common hallucinogenic/dissociative substances;
- Describe many of the substances in this category and their effects on humans;
- Identify epidemiological patterns in cannabis and other hallucinogenic/dissociative substance use;
- Explain key terms and concepts related to cannabis and other hallucinogenic or dissociative substances.

# Ch. 1: Cannabis

Similar to alcohol, **cannabis** use has a rather long and storied history in the United States and some other parts of the world. Cannabis has been used in some cultures for hundreds, or even thousands, of years (Malcolm, 2020). Research and clinical practice related to cannabis has had to evolve over time, due to two significant forces. First, the concentration of psychoactive ingredients in cannabis plants has steadily increased over the past few decades (NIDA, 2019a). Potency prior to the 1990s was estimated to be less than 2%; the range in popular strains in 2017 was estimated at 17-28% and some concentrated oil products may even exceed 95% (Stuyt, 2018). Thus, individuals using cannabis products today face a higher relative dose compared to what past generations encountered. It is difficult to determine how much of the active ingredient a person has been exposed to—unlike the standard equivalents we can calculate with alcohol—because (1) different strains and growing conditions produce different concentrations, (2) different preparation methods affect concentrations (e.g., marijuana, hashish resin, hash oil, and parts of the plant used), (3) amounts used/inhaled in any administration vary, and (4) other products may be combined or contaminating the cannabis used (WHO, 2016).

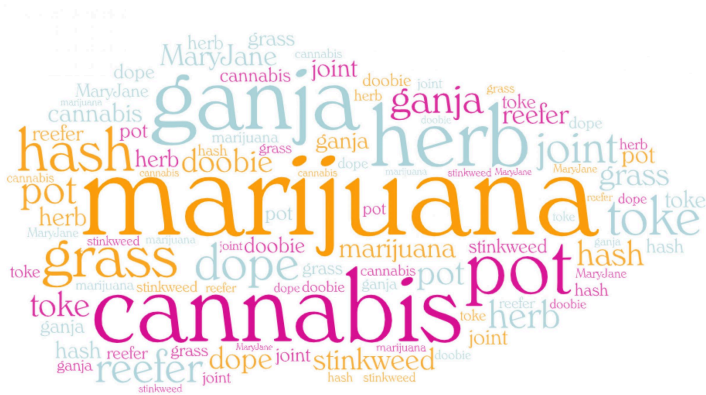


Second are the dramatic shifts, revisions, and retrenchment in local and state policy across much of the United States concerning the use, possession, production, and distribution of both medical and recreational marijuana. Policy revision addressing mass incarceration and promoting smarter “decarceration” in response to the nation’s War on Drugs also have relevance regarding cannabis-related offenses (see Pettus-Davis & Epperson, 2015); federal changes in policy may be on the horizon, as well.

You will notice throughout this module the word “cannabis” is often used, rather than the heavily politicized word **marijuana**. Malcolm (2020), citing Antoine and Douglas (2018) explained that many researchers, scholars, and advocates express a preference for the more scientific term “cannabis,” citing racist and propagandist connotations of the word “marijuana”. He described the deep historical and social significance of cannabis use, noting that “it has been simultaneously reviled, denounced, prohibited, tolerated, and revered,” and that politicization of cannabis use has been heavily tinged with racism concerning African American, Caribbean, and Mexican populations. The word “marijuana,” originating in Mexico,



has often been associated with stigmatized or marginalized groups, hence the stated preference for the less-politicized word “cannabis.” Common “street” names for cannabis (or marijuana) include pot, weed, grass, herb, nuggets, ganga/ganja, dope (though this can also mean heroin in some communities), reefer, ganja, hash (or hashish), Mary Jane, and stinkweed (<https://luxury.rehab.com/marijuana-rehab/street-names-and-nicknames/>).



## How Cannabis is Commonly Used

Cannabis refers to several psychoactive substances derived from either the *cannabis sativa* or the *cannabis indicaplant* (Malcolm, 2020), and less commonly the *cannabis ruderalis* plant. Various parts of these plants may be used: leaves, stems, seeds, and flowers/buds/nuggets (NIDA, 2019). Traditionally, cannabis use involved inhaling smoke either directly as a form of cigarette/cigar (nicknames include joint, blunt, roach, doobie) or through a filtration system (nicknames water pipe, bong). Recent use has included vaping or use of a vape pen/device, however a great deal of public health concern has emerged regarding vaping the

psychoactive ingredients of cannabis. A warning released on October 4, 2019 stated:



In its continued efforts to protect the public, the U.S. Food and Drug Administration (FDA) is strengthening its warning to consumers to stop using vaping products containing THC amid more than 1,000 reports of lung injuries—including some resulting in deaths—following the use of vaping products...A majority of the samples tested by the states or by the FDA related to this investigation have been identified as vaping products containing THC. Through this

investigation, we have also found most of the patients impacted by these illnesses reported using THC-containing products, suggesting THC vaping products play a role in the outbreak (see <https://www.fda.gov/consumers/consumer-updates/vaping-illness-update-fda-warns-public-stop-using-tetrahydrocannabinol-thc-containing-vaping>)

The act of smoking cannabis has a number of nicknames, as well: toking, cheeching, blowing, firing one up, going loco, and others (see <https://americanaddictioncenters.org/marijuana-rehab/slang-names>). Mode of administration matters with cannabis. First, inhaling cannabis in smoke delivers the psychoactive chemicals fairly quickly to the brain. As discussed in previous modules, this has a profound impact on how a person “learns” to anticipate the effects and on the potential for developing a cannabis substance use disorder. Additionally, smoking/inhaling cannabis exposes the mouth, throat, and lungs to hundreds of chemicals present in the plant material or as additives, some of which may have long-term health implications (Begun, 2020).

Use of cannabis' psychoactive ingredients in oils and extracts has recently become popularized. This includes consuming the products in food, as **edibles** (e.g. baked goods, candy, infused cooking oil or butter). The potential problem with edibles is that the action of the psychoactive ingredients is somewhat delayed since the product needs to be digested for absorption to occur. Thus, the psychoactive effects might not be experienced for 1-3 hours post-ingestion. If a person's expectancy involves an immediate effect (as happens with the faster administration route of smoking), that person may continue to consume edibles and end up with an excessive dose when it does begin to take effect.

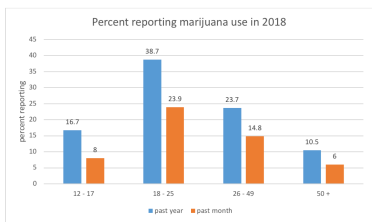


Cannabis products are most often used alone; however, individuals may intentionally or unintentionally (laced) use them in conjunction with other substances such as alcohol, cocaine, or heroin. The combination of alcohol and cannabis products has a **potentiating effect**, meaning that the effects of either alone are heightened, as are their side effects. The combination of cannabis with heroin or other opioids, like alcohol, is also potentiating: the presence of heroin increases the potential for problems in breathing, loss of consciousness, or opioid overdose. The combination of cannabis with cocaine is antagonistic in the sense that one is relatively calming/sedating (cannabis) and the other an intense stimulant (cocaine). This is intended to soften some of the harsh effects of cocaine use. Cannabis may also be contaminated with other products—particularly when illegally supplied—including inert/

inactive plant materials, fungus infested material, bacteria, or other chemicals.

## How Commonly Cannabis is Used

Other than alcohol, cannabis continues to be the most widely used psychoactive substance in the world: an estimated 188 million individuals used cannabis in 2017 (UNODC, 2019). According to the 2018 National Survey of Drug Use and Health (NSDUH; SAMHSA, 2019), 45.3% of individuals over the age of 12 years have used marijuana during their lifetime, 15.9% during the past year, and 10.1% during the past month. This graph depicts past year and past month use rates by age category: the rate is greatest among emerging adults aged 18-25. Still, marijuana use is not the “norm” in any of the age categories since many fewer than half engaged in this behavior.

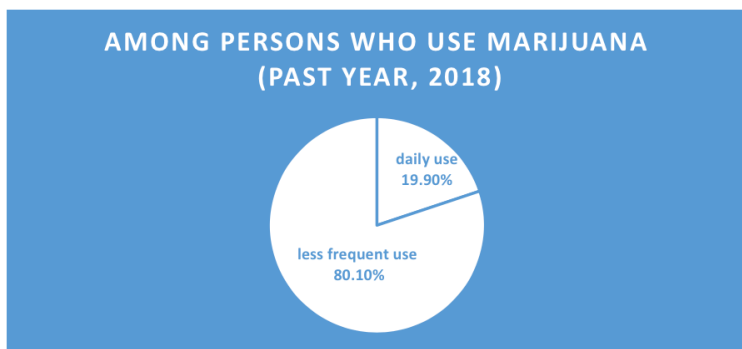


*Click chart to download data in an accessible format*

Data suggest that marijuana use is more common among men than women: past year marijuana use was reported by 18.5% of males and 13.4% of females among persons aged 12 and older. Past year marijuana use was most often reported by individuals identifying as

American Indian or Alaska Native (23.0%) or as being of two or more races (23.4%). The rate was lowest among individuals identifying as Asian (8.9%). This was more commonly reported by individuals who were not Hispanic or Latino (16.4%) compared to those self-identifying as Hispanic or Latino (13.6%), and slightly more commonly among Native Hawaiian or Other Pacific Islander (17.7%) and Black or African American (17.8) respondents than White respondents (16.5%).

The frequency with which cannabis is used varies considerably. The 2018 NSDUH data (SAMHSA, 2019) indicated that the percentage of individuals aged 12 and older who in the past year used marijuana daily or almost daily was 3.2%; among individuals who used marijuana during the past year at all, 19.9% used it daily or almost daily. In other words, the vast majority of individuals who use marijuana do not use it daily or almost daily, but almost 8.7 million individuals do so (SAMHSA, 2019).



*Click chart to download data in an accessible format*

## Cannabis Effects

Cannabis' primary psychoactive chemical is Delta-9-tetrahydrocannabinol or **tetrahydrocannabinol**, both abbreviated as **THC**, which acts on **cannabinoid receptors** making up the **endocannabinoid system** throughout the body (NIDA, 2019a). THC is chemically very similar to the neurotransmitter **anandamide**, known to be involved in regulating mood, memory, appetite, pain perception, cognition, and emotions. Because of this chemical similarity, THC can bind to cannabinoid receptors usually activated by anandamide, thereby affecting similar functions and possibly interfering with normal functioning in the affected areas of the

brain (NIDA, 2007). THC exposure initiates significant dopamine release in the areas of the brain with the highest concentrations of the involved receptors, particularly in brain areas responsible for behavioral reward systems. THC tolerance does develop in humans engaged in regular use (WHO, 2016).

The primary psychoactive effects of cannabis are intoxication, euphoria, relaxation, distorted sensory perception, impaired balance and coordination, and impaired cognitive functions or judgement; effects that potentially contribute to accidental injury (Begun, 2020). The short-range mental, psychological, and information processing effects (NIDA, 2019) include:

- altered sensory perception (e.g., colors seem brighter)
- altered sense of time
- altered mood
- impaired body movement/coordination/reflexive responses
- difficulty thinking and problem-solving (impaired cognitive functions)
- impaired memory
- hallucinations, delusional, and/or paranoid thinking (particularly at higher doses)
- depression
- anxiety
- possible suicidal thoughts
- temporary or persistent psychosis (highest risk with regular use of high potency products; individual vulnerability varies)
- worsening of symptoms among persons with schizophrenia.

Because cannabinoid receptors are distributed throughout the body, additional physical effects occur with use of cannabis products, including:

- respiratory/breathing problems (related to smoking cannabis, can be similar to smoking tobacco products) (NIDA, 2019a);
- increased heart rate (may increase risk of heart attack, with

older individuals at greater risk) (NIDA, 2019a);

- cannabinoid hyperemesis syndrome (regular cycles of severe nausea, vomiting, and dehydration occurs in some individuals engaged in regular, long-term use) (NIDA, 2019); this seems paradoxical in that cannabis products may help reduce nausea in the short-term.

Long-term heavy cannabis use is associated with detectable functional and structural brain changes, particularly those involved in memory and cognitive performance—for example, an average of 8 IQ points lower compared to individuals who did not use cannabis regularly over a long period (WHO, 2016). Additionally, individuals who used cannabis 10 or more times before the age of 18 years were more than twice as likely to later receive a diagnosis of schizophrenia than individuals who did not use cannabis and there existed evidence of a dose-response relationship—heavier use was associated with greater risk (WHO, 2016). Their cannabis use appeared to have preceded the onset of schizophrenia symptoms, suggesting (but not conclusive) that the cannabis use likely was not an effort to self-medicate schizophrenia symptoms (WHO, 2016). Additionally, there exists a significant prevalence of co-morbid cannabis use disorder and other mental health disorders (WHO, 2016).



Because extrinsically introduced substances occur in greater concentrations and release greater amounts of the involved neurotransmitters than naturally occurring stimuli, their effects are experienced more intensely. This contributes to the potential for ongoing misuse of or addiction to these kinds of substances. Chronic cannabis use can result in development of a substance use disorder specified in the DSM-5 as **cannabis use disorder**, following the 11 diagnostic criteria identified in Module 2. The criteria include elements of recurrent use under physically hazardous conditions, tolerance, withdrawal, craving, greater use than intended, and other criteria similar to those in the general list of substance use disorder criteria. The DSM-5 also presents criteria for assessing:

- cannabis intoxication,
- cannabis withdrawal,
- cannabis intoxication delirium,
- cannabis-induced psychotic disorder,
- cannabis-induced anxiety disorder, and
- cannabis-induced sleep disorder.



Worldwide, cannabis use disorder was estimated to affect an estimated 4-8% of adults during their lifetime: approximately 13.1 million persons globally (WHO, 2016). In the 2018 United States National Survey of Drug Use and Health (NSDUH) data, over 4.4 million individuals (1.6% of the population) aged 12 and older were estimated to meet criteria for a substance use disorder involving marijuana during the past year (NSDUH, 2019)—as much as 5.9% of the population of individuals aged 18-25 years. Although women have a lower prevalence of cannabis use disorder than men (0.14% versus 0.23%), women “exhibit an accelerated progression to cannabis-use disorder after first use and show more adverse clinical



problems than men” (WHO, 2016, p. 11). This phenomenon is sometimes referred to as **telescoping**—the rate at which problems appear is collapsed into a shorter time frame.

Remember: the concentration of THC in different cannabis formulations varies markedly, which in turn means that the effects can vary widely, as well. While some efforts to develop THC content standards, THC and other chemical concentrations in different cannabis samples are not easily compared as in the case of standard drink equivalents with alcohol.

## Driving under the influence.

Traffic injuries related to cannabis use are also of concern, particularly as more regions permit its medical and/or recreational use. The risk of car crash is estimated to double or triple with cannabis intoxication (WHO, 2016).



Furthermore, the crash risk “increases substantially if cannabis users also have elevated blood alcohol levels, as many do” (WHO, 2016, p. 20). In the 2018 NSDUH data (SAMHSA, 2019), an estimated 11.8 million individuals aged 16 or older drove under the influence of marijuana during the past year; this figure is more than half the estimated 20.5 million individuals who drove under the influence of alcohol.

## Prenatal cannabis exposure.

Evidence is unclear as to the long-term developmental impact of prenatal exposure to cannabis. It is likely, however, that neurobehavioral and cognitive impairments, as well as alternations in the dopamine neurotransmitter system of some brain regions, are more common among children prenatally exposed to cannabis (WHO, 2016). Negative effects of prenatal cannabis exposure “may not become apparent until later in development. It is, therefore, essential to follow up cannabis-exposed children long into adolescence” (WHO, 2016, p. 16).



## Cannabis, Cannabinoids, and Cannabidiol

As previously noted, cannabis refers specifically to products derived from the cannabis plants of the *sativa*, *indica*, or *ruderalis* type (Malcolm, 2020). The term **cannabinoid** refers to a diverse range of chemicals structurally similar to THC and that act on the same cannabinoid receptors in the human body (UNODC, 2018; WHO, 2016). Many of these are either genetically modified versions of cannabis plants or laboratory manufactured synthetic products; **endocannabinoids** are produced naturally in the human body (WHO, 2016). Modified and synthetically produced cannabinoids may have many times greater relative dose exposure than those occurring naturally, and as a result, potential harm with their use is amplified (Malcolm, 2020). For example, you may hear about **Spice** or **K2**: these are synthetic cannabinoids produced by spraying plant material (not necessarily cannabis) with psychoactive chemicals. Thus, Spice and

K2 tend to be many times more concentrated and bind to the cannabinoid receptors more intensely than cannabis alone—there is no way of knowing what or how much active ingredient is involved or what other toxic substances might be included in these products. “Between 2011 and 2017, U.S. poison control centers received more than 31,000 calls related to synthetic cannabinoid effects” (<https://www.webmd.com/mental-health/addiction/news/20180910/k2-spice-what-to-know-about-these-dangerous-drugs>).

Cannabinoids have recognized medical uses recognized in many, but not all, countries: cannabis remains a Schedule I drug in the U.S. DEA classification system because of its high potential for abuse and the absence of their recognized medical uses in the U.S. This, however, may be changing as increasing evidence emerges supporting its efficacy in treating numerous physical and mental health conditions.

**Cannabidiol (CBD)** is a cannabinoid naturally occurring in cannabis plants, including hemp (low THC-containing cannabis varieties), although it can also be synthetically manufactured (WHO, 2018). In countries outside of the United States, CBD has medical uses in treatment of several medical



conditions; its use is under study for approval as a medical treatment in the United States, as well. CBD does not produce the psychoactive effects seen with THC, seems not to have the abuse potential seen with THC, and appears not to affect heart rate or blood pressure under normal conditions—though it may reduce both under conditions of stress (WHO, 2018). Very preliminary research suggests that CBD may prove helpful in treating opioid, cocaine, cannabis, and tobacco addiction, but “considerably more research is required to evaluate CBD as a potential treatment”

(WHO, 2018, p. 19). At this time, CBD remains in Schedule I among controlled substances in the United States.

**Cannabinol (CBN)** is a cannabinoid with weak psychoactive potential, much less than THC of which it is a metabolite (breakdown product). The term **hemp** refers to cannabis varieties low in THC, thus are considered non-intoxicating. Interest in CBN is more aligned with potential medical uses related to some observed effects on immune and inflammatory processes rather than with psychoactive potential. Globally, hemp also is produced and used in many ways related to its physical characteristics (e.g., textiles, construction and insulation materials, cosmetics, pulp/paper-like products, animal bedding, insect repellent, biodegradable landscape matting, cooking oil, fuel, and others <https://www.hort.purdue.edu/newcrop/ncnu02/v5-284.html>).



### **STOP & THINK**

Review the nine statements below and click on the number in front of only those that you believe are true, accurate statements based on what you read in this chapter.



*An interactive H5P element has been excluded from this version of the text. You can view it online here:*

[https://ohiostate.pressbooks.pub/  
swk5805coursebook/?p=192#h5p-52](https://ohiostate.pressbooks.pub/swk5805coursebook/?p=192#h5p-52)

## Ch. 2: Other Hallucinogenic & Dissociative Substances

This chapter concerns hallucinogenic substances (hallucinogens) and includes several dissociative substances, as well. The chapter contents address both naturally occurring hallucinogens from the plant and animal world, and others that are synthesized. Because of enlightenment or spiritual associations, hallucinogens are among the oldest psychoactive substances known to be intentionally used by humans—possibly for as long as 10,000 years (Knox, 2016). Hallucinogenic **trips (tripping)** can be experienced as either enlightening, fascinating, wonderous or “bad,” fraught with creepiness, terror, distress, and anxiety—“bad” as in the flying monkeys from the Wizard of Oz movie, an alien popping out of your chest from the movie Alien, or a prolonged, inescapable, really bad dream.

The substances discussed in this chapter represent only a fraction of those used worldwide—many forms are used in relatively localized ways. An example of localized misuse comes from guides’ anecdotal accounts in Costa Rica who describe tourists becoming ill (or dying) from licking cane toads who produce poison for self-defense, but which contain hallucinogenic chemicals (bufotenine and possibly DMT), among other toxic chemicals. Toad-produced toxins may be fatal to natural predators (including dogs and crocodiles). Production of hallucinogenic products from close toad relatives in the southwest United States (Colorado River Toad) has led to these species becoming seriously endangered.



## What are Hallucinogenic and Dissociative Substances

Hallucinogenic and dissociative substances are those known to distort a person's perceptions of reality, altering a person's thoughts, feelings, and awareness of their environment (mind-altering) to the point where sensations seem real although they are not (<https://www.drugabuse.gov/publications/drugfacts/hallucinogens>). Dissociative substances also alter a person's sense of reality, inducing a sense of being disconnected or detached (dissociated) from reality and/or disconnected from control over one's own body.

Hallucinogens, whether dissociative or not, are sometimes referred to as **psychotomimetic**—mimicking psychosis (Begun, 2020). Why, you might ask, would anyone *want* to mimic psychosis? The easier-to-answer question is “What are hallucinations like?” Sometimes the effect has been reasonably recreated in films as an altered state of consciousness. In some instances, past memories are re-experienced as current lived realities, seeming as real as when first experienced. This may be good, bad, or neutral quality memories—the problem being that a person cannot control or

predict which will be experienced this way. Or, the experience can involve a sense of movement through space or time—flying, soaring, floating, being pulled along, or otherwise moving—when in reality, no such movement occurs. Someone might experience being in more than one place at the same time or becoming someone/something else: becoming a tree, being the ocean, or being inside the mind of your dog. Objects or people in the real world can take on strange shapes, colors, or sounds while things not present can be seen, felt, or heard. The experience may involve unreal sensations, including the sensory crossover: hearing colors or seeing sounds, for example. The kind of sensory crossover experience is called **synesthesia**.

In addition to their main psychoactive effects, many hallucinogenic substances also cause increased heart rate, blood pressure, and body temperature. They also may cause sleep disorder, and possibly paranoia or acute, severe panic. Hallucinogenic experiences may persist or recur well after the active ingredients have been fully metabolized and the drug's effects have worn off (**drug-related flashback**). Dissociative substances also may cause numbness, amnesia, disorientation, inability to move, and trouble breathing, particularly when used on combination with other respiratory depressing substances. Anxiety and depression (including suicidal thoughts) associated with hallucinogen use may persist long-term after a period of regular use.

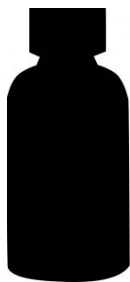




Examples of naturally occurring hallucinogenic substances are **psilocybin** (“**magic mushrooms**”), **mescaline** (**peyote cactus**), ayahuasca (DMT), and *salvia divinorum* (a plant in the mint family). Examples of synthesized hallucinogenic substances are **LSD** (**lysergic acid diethylamide**) and **MDMA/ecstasy**. Examples of synthesized hallucinogenic dissociative substances are **PCP** (**phencyclidine**), **ketamine**, and **dextromethorphan (DXM)**, the substance in many cough medicines. You may also hear about synthetic products called **bath salts** (synthetic cathinones) which have both hallucinogenic and stimulant effects; these new psychoactive substances may temporarily skirt the law and DEA scheduling rules by rapidly changing their ingredients and being sold as plant food, jewelry cleaner, or phone screen cleaner, and labeled “not for human consumption” (NIDA, 2018a).

Let’s look at some of these substances in a little more detail, recognizing that according to the U.S. Drug Enforcement Agency

(DEA), LSD, mushrooms, and ecstasy/MDMA are the most commonly misused hallucinogens ([https://www.dea.gov/sites/default/files/sites/getsmartaboutdrugs.com/files/publications/DoA\\_2017Ed\\_Updated\\_6.16.17.pdf#page=84](https://www.dea.gov/sites/default/files/sites/getsmartaboutdrugs.com/files/publications/DoA_2017Ed_Updated_6.16.17.pdf#page=84)).



**DXM (dextromethorphan).** DXM is a common ingredient in many cough suppressant medications. In high doses, DXM can induce hallucinogenic and dissociative effects. A complicating problem with high dose exposure to DXM in cough suppressant formulations is that the person is also ingesting exceedingly high doses of the other ingredients (antihistamines and decongestants) that can produce risky overdose effects themselves. Even at low doses DXM can cause distorted visual perceptions. Related to the name of one well-known manufacturer of cough medicines, Robo is a common nickname related to DXM misuse (NIDA, 2019b).

**Ketamine.** Ketamine is another of the “club drugs” you read about in our module concerning sedative-hypnotic drugs. It was originally developed for anesthesia purposes and has some structural similarity to PCP (see below). It is briefly revisited here in our module concerning hallucinogenic and dissociative drugs because

of its hallucinogenic effects and its effects on loss of body control and amnesia. Ketamine can create a dream-like state and/or altered perceptions. Flashback experiences have been reported with ketamine use. Other names used for ketamine include special K, K, super K, vitamin K, kit kat, keets, jet, purple, and cat valium. Acute effects include rapid heart rate, high blood pressure, and depressed breathing/apnea events, and use is associated with increased risk of injury because of its suppression of pain responses (Chakraborty, Neogi, & Basu, 2011). Ketamine is potentially addictive and chronic use may lead to reduced pain sensation, loss of coordination, difficulty with concentration, insomnia, drowsiness, slurred speech, and bladder incontinence. Withdrawal symptoms include loss of appetite, fatigue, irregular heartbeat, anxiety, depression, tremors, sweating, and chills, as well as nightmare disturbed sleep. Ketamine is often used in a mixed sequence of polydrug use involving stimulants (e.g., methamphetamine, cocaine) or heroin (Chakraborty, Neogi, & Basu, 2011). It is a Schedule III drug in the U.S. and currently the subject of some research into its potential in treating severe depression.

**Kratom.** *Kratom* powder, pills, and capsules are produced from the leaves of a tropical Asian plant with a mix of effects: at low doses kratom acts more as an energizing stimulant, while at higher doses acts more like an opioid, but can also have hallucinogenic effects. Thus, it is difficult to know where to classify kratom: often, it is listed in opioid discussions because of its pain-control actions. Kratom has potential use in opioid withdrawal but is considered a dangerous addictive substance itself (UNODC, 2012). Although some argue it is a safer alternative than certain prescription medications, its use has proven deadly in some cases. Kratom's abuse potential and lack of evidence-based medical uses lead to its being declared an illicit substance in many countries and several of the United States. In 2016, the U.S. Drug Enforcement Agency (DEA) announced plans to declare kratom a Schedule I substance in the United States; however, intense public reaction contributed to the DEA's reversal of this decision only 2 months later. As of 2017 kratom remains an

unscheduled substance at the federal level but is listed as “a drug of concern” by the DEA.

**LSD (lysergic acid diethylamide).**

LSD is among the most powerful mind-altering substances in common use (NIDA, 2019b). Some common “street” names for LSD include acid, battery acid, blotter acid, boomers, California sunshine, sunshine,



dots, microdot, doses, pane, windowpanes, sugar cubes, Lucy in the sky/Lucy, golden dragon, and mellow yellow—several of these nicknames refer to the ways that LSD is or has in the past been distributed (e.g., drops placed on sugar cubes or on scraps of blotter paper). LSD is synthesized from the product of a fungus known to grow on rye and some other cereal grains: ergot. Ergot-contaminated food/bread is attributed as the cause of historical community-wide mass hysterical/hallucination events; ergotamine is also an ingredient in several medications used to affect blood flow patterns related to migraine headaches and to induce uterine contractions (so it can also cause miscarriage).

**MDMA/ecstasy.** MDMA, commonly called ecstasy (sometimes XTC) or Molly, is a hallucinogen with stimulant effects. The psychoactive effects of MDMA involve increased activity among three neurotransmitters (NIDA, 2018b): dopamine (reward



system reinforcing behavior), norepinephrine, and serotonin (mood, appetite, sleep, sexual arousal, pain sensation). MDMA use can cause dangerously high body temperature, elevated blood pressure, and rapid heart rate, especially when a person’s increased energy leads

to high levels of physical activity. MDMA/ecstasy is often addressed as a “club drug” but its use is not limited to those environments. It is listed as a Schedule I drug by the U.S. DEA; because distribution is illegal, what MDMA an individual acquires to use may be heavily contaminated with toxic chemicals or other drugs. MDMA appears to have an addictive potential, although the evidence is unclear (NIDA, 2018); “almost 60% of people who use ecstasy report withdrawal symptoms” and as many as 43% have met three or more criteria for substance use disorder related to this drug (<https://www.drugs.com/illicit/ecstasy.html>). Confusion, depression, anxiety, sleep disorders, and craving are known effects of either MDMA use or withdrawal, and in nonhuman primates MDMA has proven to be toxic to neurons in the mood, thinking, and judgment areas of the brain—just 4 days of MDMA exposure caused damage that remained evident years later (<https://www.drugs.com/illicit/ecstasy.html>).

**Mescaline (peyote).** While mescaline is naturally occurring in peyote cactus buds (buttons or mescal buttons) and a few other plant types, it can also be synthetically produced (NIDA, 2019b). Generally illegal to possess in the U.S., some religious ceremony uses by Native Americans is allowed.



Mescaline causes “rich visual hallucinations” (<https://www.drugs.com/illicit/mescaline.html>), similar to those experienced with LSD or psilocybin (see below). Use also may be accompanied by a distorted (slowed) sense of time and synesthesia (seeing sounds, hearing colors). Its use may be accompanied by a racing heart rate, acute anxiety, headache, accidental injury, amnesia, vomiting, or seizures (<https://www.drugs.com/illicit/mescaline.html>). Although not considered to be addictive,

individuals may develop tolerance, requiring increasing amounts to achieve the same effects—which also increases the potential for negative side effects.

**PCP (phencyclidine).** PCP is classified as a dissociative substance with hallucinogenic effects. Common “street” names include angel dust, wack, ozone, dust, peace pills, embalming fluid, rocket fuel; cannabis laced with PCP may be called supergrass, superweed, whacko tobacco, or killer joints (<https://www.drugs.com/illicit/pcp.html>). PCP interacts with an array of neurotransmitter sites (including NMDA, glutamate, dopamine, opioid, and nicotinic receptors). Auditory hallucinations may accompany visual distortions with PCP use. Individuals may experience acute anxiety or paranoia which may contribute to them erupting into hostile violence; some individuals experience an overwhelming sense of dread or impending doom which may contribute to attempted suicide (<https://www.drugs.com/illicit/pcp.html>). PCP is potentially addictive and long-term use may lead to memory loss, cognitive/learning difficulties, depression, and significant weight loss. Because PCP interacts with alcohol and other CNS depressants (e.g., benzodiazepines), overdose risk is increased when these substances are combined.

**Psilocybin.** Multiple species of fungi (mushrooms) contain the active ingredient **psilocybin** and/or psilocin, hence they are often called psychedelic or **magic mushrooms**, they may also be referred to as mushies, sacred mushrooms, or zoomers. Because of the potential for abuse and the absence of evidence-based medical uses, psilocybin is a Schedule I



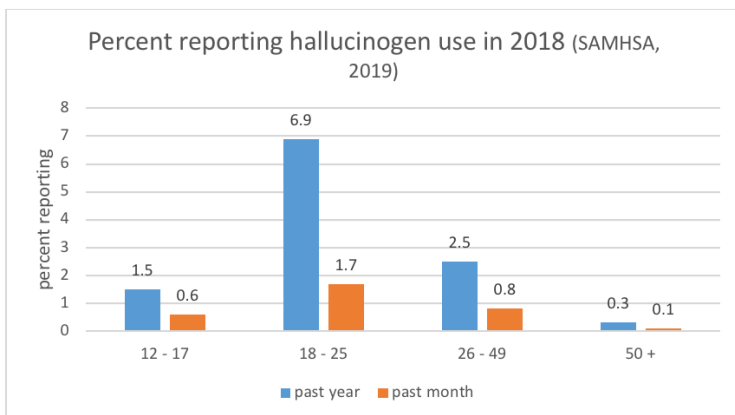
substance in the United States (and many other United Nations countries). However, in recent years increasing scientific interest

has been directed toward the potential for psilocybin to treat various physical and mental conditions, including intense cluster headaches, depression, and extreme anxiety. Should these studies result in strong supporting evidence, it is possible that the DEA could revise the schedule level for this substance. One problem with psilocybin mushrooms is that they closely resemble other types that are poisonous. Another problem is that their potency varies dramatically by mushroom species, growing conditions, and how they are handled/time since harvesting. For example, the concentration in dried mushrooms is considerably higher than in their fresh counterparts (<https://www.medicalnewstoday.com/articles/308850.php#what-is-psilocybin>). Although psilocybin is considered non-addictive, individuals may develop tolerance with regular use and possibly cross-tolerance to other hallucinogenic substances (e.g., LSD and mescaline; <https://www.medicalnewstoday.com/articles/308850.php#abuse-potential>).

**Salvia divinorum.** *Salvia divinorum*'s original use among indigenous groups in Mexico involved ritual divination in spiritual contexts. The plant's leaves can be chewed or smoked and the active ingredient, salvinorin A, induces visual hallucinogenic effects, as well as distorted bodily sensations and movement. *Salvia divinorum* is currently unscheduled by the U.S. DEA but is illegal in some states.

## Epidemiology of Hallucinogenic Use

Based on data from the 2018 National Survey on Drug Use and Health (NSDUH, SAMHSA, 2019) we can see once again that the age group most commonly reporting the use of these substances were emerging adults, 18 to 25-year-olds. However, compared to many other substances (e.g., alcohol, cannabis, stimulants, sedatives, opioids) the rate of hallucinogenic use is considerably lower.

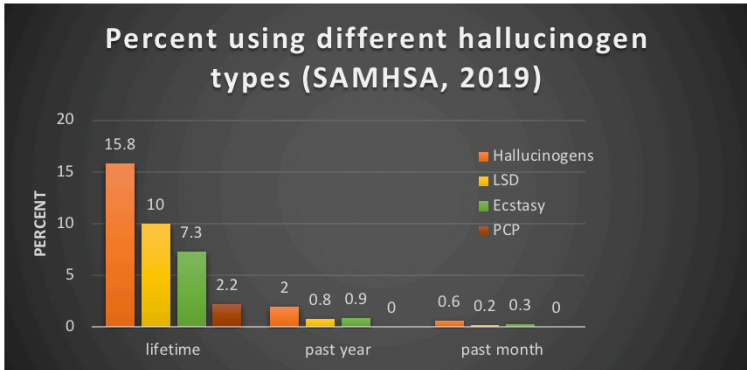


*Click chart to download data in an accessible format*

The rate of overdose deaths from hallucinogens is described by the DEA as “extremely rare” [https://www.dea.gov/sites/default/files/sites/getsmartaboutdrugs.com/files/publications/DoA\\_2017Ed\\_Updated\\_6.16.17.pdf#page=84](https://www.dea.gov/sites/default/files/sites/getsmartaboutdrugs.com/files/publications/DoA_2017Ed_Updated_6.16.17.pdf#page=84). However, the DEA reports that deaths *related* to their use do occur—typically from accidents, engaging in risky/dangerous behaviors, toxicity or poisoning by contaminants, and suicide.

The 2018 NSDUH data (SAMHSA, 2019) can help develop an understanding of which hallucinogenic substances are used most often in the U.S. by individuals aged 12 and older. The results for lifetime, past year, and past month use of hallucinogens in general, LSD, PCP, and ecstasy are presented in the following graph. As you can see, LSD was the most commonly reported of the hallucinogens and PCP the least.





*Click chart to download data in an accessible format*



### **STOP & THINK**

Turn on the iTunes visualizer to display animations of some favorite songs or visit the following YouTube sites to experience how sensory crossover might be experienced—visualizations of music/sound.

- <https://www.youtube.com/embed/QHTjYSC8RWw>

- <https://www.youtube.com/embed/NwHUemOwF0c>

Consider what might it be like for someone to live a unique visual/auditory experience through use of hallucinogens. Then consider what might be like if they were to encounter an anxiety producing visual/auditory experience instead?

Consider what you have been learning about hallucinogenic substance misuse: how would you advise someone who was considering using LSD, peyote, or mescaline for the first time?

## Ch. 3: Summary

In this module you read about cannabis and hallucinogenic substances, including some known for their dissociative effects. The first chapter focused on cannabis, cannabinoids, cannabidiol, and related synthetic cannabis products. You learned about the reason some recommend using the word cannabis rather than marijuana, and you were reminded of the lessons from the beginning of the course about how stigma, prejudice, and stereotypes might influence public health and social policy related to substances like cannabis (marijuana). The nation's current state of ambivalence about cannabis and products from cannabis was pointed out. The nature and effects of THC were presented, and you read about the distinction in effects/experience depending on whether the cannabis is used by inhaling (smoked) or consumed in edibles. The risks associated with driving under the influence of cannabis were mentioned, as well. In the second chapter, emphasis was placed on a varied group of hallucinogenic substances, some that occur naturally and others which are synthetically produced. You learned that these substances differ markedly in the power of their psychoactive effects and that their effects can be highly unpredictable. Some of the hallucinogens we explored have stimulant qualities, as well. Quite a few new terms were presented in this module, and you are now prepared to review them in the next section.

# Module 9: Key Terms

**anandamide:** an endogenous cannabinoid neurotransmitter naturally produced in humans, operating in the endocannabinoid system.

**bath salts:** a type of (questionably legal) synthetically produced hallucinogenic stimulants.

**cannabinoid:** any of the class of chemical compounds acting on cannabinoid receptors in the endocannabinoid system (e.g., THC and CBD).

**cannabinoid receptors:** part of the endocannabinoid system and located throughout the human body, they are involved in psychoactive responses to cannabinoids including appetite, mood, memory, and sensitivity to pain.

**cannabis:** label applied to *cannabis sativa*, *indica*, and *ruderalis* among the many types of plants in the cannabis family; often referred to as marijuana and typically distinguished from hemp based on its concentration of psychoactive substances (specifically, THC).

**cannabis use disorder:** a specific diagnosis in the DSM-5 and ICD-11 defined by a number of substance use disorder diagnostic criteria.

**dextromethorphan (DXM):** a cough suppressing ingredient commonly found in over-the-counter (OTC) formulations; when consumed in large quantities, potentially has hallucinogenic effects but this comes with significant side effect risks.

**dissociative substances (dissociatives):** a type of hallucinogen which, in addition to other psychoactive effects, produce a sense of detachment from one's self/body or environment.

**edibles:** cannabis-infused products containing THC consumed by eating or drinking.

**endocannabinoid system:** an endogenous cell-signaling system in the human body that regulates a variety of mental and physical

processes through response to cannabinoids (whether endogenously produced or exogenously introduced).

**endocannabinoids:** cannabinoids produced endogenously (naturally) in the body.

**flashback (drug-related):** the re-experiencing of a drug's effects without having used it again and after the drug has been fully metabolized (no longer in the body); may occur long after the true drug effects have ceased.

**hallucinogenic substances (hallucinogens):** a varied group of substances, natural or synthetic, with the potential for causing a person to experience a dramatically distorted reality (hallucination), usually in the visual or auditory sphere, but may also affect time sense, tactile sensation, and other mental functions.

**hemp:** low THC content *cannabis sativa* used for its material properties and possible medical applications rather than psychoactive characteristics.

**K2:** synthetically produced cannabinoid produced by spraying dried plant material with psychoactive chemicals; synthetic marijuana (see Spice entry).

**ketamine:** originally developed for use as an anesthetic, has significant dissociative and hallucinogenic effects; often considered one of the "club drugs."

**kratom:** derived from leaves of a specific plant, it has some effects similar to opioids and stimulants, may also have hallucinogenic effects and is potentially addictive.

**LSD (lysergic acid diethylamide):** a synthetically produced, highly concentrated hallucinogen.

**marijuana:** a commonly used name for cannabis used for its psychoactive effects.

**MDMA/ecstasy:** synthetically produced hallucinogenic substance with stimulant effects; considered one of the "club drugs."

**mescaline (peyote cactus):** hallucinogen derived from the peyote cactus and other similar species; may also be synthetically produced.

**PCP (phencyclidine):** originally developed for anesthesia, misused for its psychoactive (hallucinogenic) effects, it also may produce amnesia.

**potentiating effect:** when one substance increases the potency or effectiveness of another.

**psilocybin (“magic mushrooms”):** hallucinogenic substance naturally occurring in specific species of mushroom; over 100 species contain psilocybin at varying degrees of potency (<https://www.livescience.com/psilocybin.html>).

**psychotomimetic:** substance that, in the short-term, induces effects that mimic (imitate) a psychotic episode.

**salvia divinorum:** a plant species with leaves that can produce hallucinogenic effects when chewed or drunk as tea.

**Spice:** synthetically produced cannabinoid produced by spraying dried plant material with psychoactive chemicals; synthetic marijuana (see K2 entry).

**synesthesia:** when one of the senses is perceived by another sense, such as sound being visual or something seen being heard; some versions associate objects with color, flavor, or scent (e.g., the letter A being red and the letter B as blue).

**telescoping:** refers to accelerated rate of progression of substance use disorder symptoms/criteria often seen in women compared to men.

**tetrahydrocannabinol (Delta-9-tetrahydrocannabinol)/THC:** the primary psychoactive ingredient in cannabis.

**trip (tripping):** an altered state-of-consciousness episode induced by use of an hallucinogenic substance.

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## PART X

# MODULE 10: FOCUS ON STIMULANTS (AMPHETAMINES, METHAMPHETAMINE, COCAINE, NICOTINE, & CAFFEINE)

## Introduction

The next class of substances to explore is the large, diverse group that produce central nervous system (CNS) stimulant effects. While you might expect to read about amphetamines (including methamphetamine), you might not have been expecting cocaine, caffeine, and tobacco to be presented here. These are included because they also produce stimulant effects and all of these substances have some degree of addictive potential—in other words, can be objects of substance misuse. There exists considerable controversy among substance misuse/addiction treatment professionals as to whether any of these substances (including coffee and cigarettes) support or interfere with recovery from substance use disorders (SUDs), even those SUDs that do not involve stimulants. As your review the contents from this module, consider (or reconsider) your own perspective on this issue. Content presented in this module informs and was informed by Begun, A.L. (2020). Introduction to psychoactive substances. In A.L. Begun & M.M. Murray, (Eds.), *The Routledge handbook of social work and addictive behaviors*. NY: Routledge.

## Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Describe what stimulant substances are and their effects on humans (amphetamines, methamphetamine, cocaine, caffeine, and tobacco/e-cigarettes);
- Identify epidemiological patterns related to the use/misuse of different stimulant substances;
- Discuss the potential risks associated polydrug use mixing stimulants with other substances (particularly with alcohol);
- Define key terms and concepts related to the use of stimulant substances.

# Ch. 1: What Are Stimulant Substances and Their Effects?

In a previous module we examined central nervous system (CNS) depressants; this time we are looking at their opposite, the CNS stimulants. There exist many forms of stimulant substances, some of which are legal and unregulated in the United States (like coffee and tea), semi-regulated (like age restrictions on tobacco and vaping/e-cigarette products and stimulant medications requiring health provider prescriptions), or highly regulated (like methamphetamine which is illegal to manufacture, distribute, or possess). Many stimulant substances are synthetically produced; many others occur in nature and may have been used by humans from different cultures for hundreds or even thousands of years for their ability to increase alertness, attention, and energy. For example, local practices of chewing betel nuts (practiced in some Asian/Pacific areas) or coca leaves (practiced in parts of South America) are not usually included in reports of psychoactive substance use/misuse, despite these practices having potentially significant public health consequences, such as oral cancers (Begun, 2020). In this chapter we explore the most commonly used and misused of the stimulant substances, keeping in mind that worldwide there are many, many more (e.g., khat and bath salts) that we will not explore. Before we examine specific types of stimulant substances, let's look at the common effects these substances generally produce, then we can look more specifically at their differences.



## Effects of Stimulant Substances

Despite their many differences, stimulant substances share some common effects. Furthermore, when combined, their effects may be amplified—including their side effects (Begun, 2020). Stimulants produce their psychoactive effects through some or all of four major neurotransmitters: serotonin, dopamine, **epinephrine**, and **norepinephrine**. Serotonin and dopamine have appeared in several of our previous modules concerning psychoactive substances, so that should be no surprise. We have not previously much encountered epinephrine and norepinephrine in conjunction with other substances. These two substances have a great deal to do with the process we previously studied where the brain (and other organ systems in the body) attempts to maintain a state of **homeostasis**. The system responsible for controlling many physiological functions throughout the body, ones we do not have to think about, is called the **autonomic nervous system (ANS)**. This includes maintaining proper breathing, heartrate, blood pressure, body temperature, sweating, digestion, and kidney/urinary functions. The autonomic nervous system regulates these functions under dynamic, changing internal and external circumstances through two complementary divisions: the sympathetic and parasympathetic nervous systems.

The **sympathetic nervous system** usually runs things at a relatively even baseline level while the person is functioning within a normal

operational state. The sympathetic nervous system, however, is prepared to gear up, creating a rapid “fight or flight” stress response to perceived threats, events, or stimuli. Once the system is all revved up and the threat has passed, the system needs to calm back down again. That is where the **parasympathetic nervous system** engages and helps bring the body back to its homeostatic resting baseline state again.



How do stimulant substances play a role in all of this arousal and becalming balance dance? Most stimulant substances trigger the sympathetic nervous system to a state of arousal, as if an event warranting a “fight or flight” stress response truly exists. They do this through their influence on two neurotransmitters, epinephrine and norepinephrine. Together these two neurotransmitters play a role in the body’s stress response; epinephrine is also known as adrenaline and norepinephrine as noradrenaline. Just as we have previously discussed in comparing endogenous (naturally occurring internal sources) and exogenous (introduced from the outside)

substances, use of stimulants can cause release of these neurotransmitters in much greater quantity; additionally, some impede neurotransmitter reabsorption so they hang around in the “active” synaptic gap/cleft for an extended duration. This, in turn, keeps the person in a higher state of arousal far longer than nature might have intended as a helpful “fight or flight” response.

The major psychoactive effects across the class of stimulant substances include possible:

- heightened state of alertness, attention, and focus
- wakefulness
- feelings of pleasure, euphoria, enhanced mood (dopamine reward system activity)
- restlessness, nervousness, anxiety, agitation, and jittery feelings
- increased sexual libido
- irritability, aggressiveness, and paranoia
- hallucinations.

Because of their psychoactive effects, stimulant substances are sometimes used in combination with some of the other substances studied in our course. For example, individuals may engage in polydrug use involving stimulants to counter undesirable effects of other substances, such as combining cocaine or methamphetamine with heroin. A potentially problematic trend that has even proven fatal involves combining stimulants (e.g., caffeinated/stimulant beverages) with alcohol in an attempt to delay onset of warning signs from drinking too much too fast—this, in turn, contributes to alcohol poisoning.

In addition to their psychoactive effects, stimulant substances affect multiple organ systems, thus they can have multiple side effects. In addition to mood swings, irritability, and paranoia, side effects of stimulant use might include:

- accelerated heart rate, blood pressure, and body temperature



(especially with physical activity), as well as increased blood sugar

- reduced blood flow to many organs
- appetite suppression
- disrupted sleep patterns
- growth retardation/slowed physical development in fetuses, children, and adolescents
- tremors
- seizures
- amphetamine-induced psychosis.

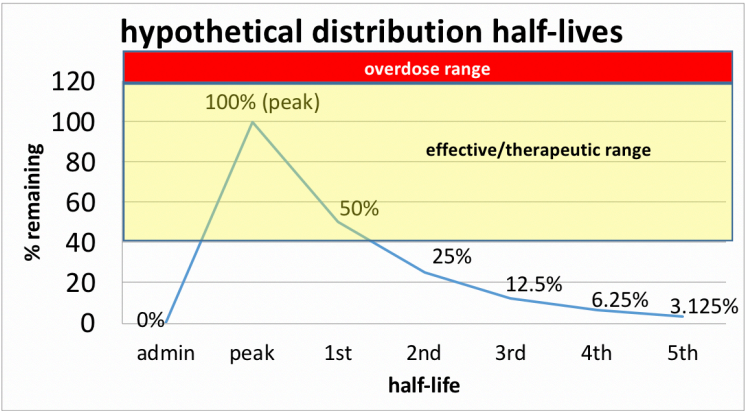
Stimulant misuse can be risky because of the cardiac (irregular and accelerated heart rate), elevated blood pressure, and elevated body temperature effects. Many stimulant substances are potentially addictive, some with greater



addictive potential than others. Individuals do develop tolerance to many forms of stimulant substances and may experience withdrawal symptoms with discontinued use following a period of regular use. Consider how you or others you know describe feeling when they miss their routine coffee or it has been a lengthy period of time since their last cigarette. Withdrawal from any stimulant substance might include fatigue, headaches, depression, and disrupted sleep patterns. The effects may be more intense in withdrawal from some substances compared to others; however, stimulant withdrawal lacks the medical risks associated with alcohol and CNS depressant withdrawal.

Another problem with many stimulant substances is the mood swing (emotional “crash”) that can occur as a dose wears off, before the next dose is scheduled—a rebound effect. Recalling what you learned about the pharmacokinetics of different medications (Module 3), there exists a zone around which the circulating drug

dose achieves therapeutic effects; too high and the risk of overdose or negative side effects rises, too low and the amount circulating in the body cannot have the desired effect—it is below the therapeutic threshold. As stimulant substances are metabolized and the circulating dose drops, individuals may experience a precipitous emotional mood swing where negative emotions (irritability and depressed mood) become overwhelming and difficult to control as the drug “washes out” of their system. While this could be relieved by taking the next dose of medication or using more of the stimulant substance, combined with what remains in the body could exceed the safety zone (risking overdose). In addition, managing stimulant medication schedules is complicated by timing issues: for example, appetite suppression effects mean the person should not take/use it prior to mealtime and the alert/wakefulness effects mean the person should not take use it too late or before bed. Many stimulant substances have relatively short half-lives, so they quickly “wash out” of the system. Some medications (e.g., Ritalin®) come in slow, extended, or time release versions which may “soften the crash” experience.



[Click to download the data from this chart](#)

## Different Types of Stimulant Substances

At this point, let's look at specific characteristics and uses/misuses associated with different types of stimulant substances. Here we consider **amphetamines** (including **methamphetamine**), **cocaine**, **caffeine**, and **nicotine** (including tobacco and vaping/e-cigarette products). In each case, the previously discussed effects remain relevant. Additionally, in each case, the mode of administration matters, too: injection reaches the brain more quickly than ingestion (swallowing), and along with “snorting” and inhaling may have a stronger influence on addictive potential. Furthermore, injection use has associated infectious disease and infection harm risks.

### Amphetamines.

Amphetamines are synthetically produced drugs that are a subject of concern related to prescription abuse (along with opioids). Common prescription drugs of this type include dextroamphetamine (Dexedrine® and Adderall®) and methylphenidate (Ritalin® and Concerta®). Some of the common “street” names include bennies (referring to benzadrine), black beauties, study drugs, speed, uppers, and vitamin R (a reference to Ritalin®). These types of drugs are typically prescribed in managing **attention deficit disorder (ADD)** and **attention deficit hyperactivity disorder (ADHD)**, and less often for narcolepsy, some forms of depression, and some respiratory problems (including asthma). At times, certain amphetamines have also been prescribed for weight management or weight loss purposes, and the military historically distributed amphetamines to troops to help them stay alert and awake for long hours (Rasmussen, 2008). Typically, these drugs are swallowed, but can be crushed and “snorted,” smoked, or injected.

The tendency to develop tolerance and significant side effects associated with the prolonged use of amphetamines, including their considerable addictive potential, has contributed to reconsideration of their recommended medical uses. Amphetamine misuse is one significant aspect of prescription abuse concerns. First, a person misusing these drugs may not gain the same benefits as someone for whom they are prescribed and risks their negative effects. Second, the person for whom they were prescribed is not taking them, therefore is not gaining the anticipated therapeutic benefits.

According to the UNODC report in 2010, clandestine laboratories producing amphetamines were detected in 32 countries. The greatest number were located in the U.S., Canada, Mexico, China, Australia/New Zealand, and several additional European countries.

*The stimulant paradox with ADD/ADHD.*

Why would stimulant medication be prescribed to manage ADD or ADHD? On the surface, it seems rather paradoxical or counter-intuitive to provide stimulants to someone who already naturally exhibits high levels of energy and activity—a bit like



adding fuel to an already burning fire. Use of medications like Ritalin®, Concerta®, Adderall®, and Dexedrine® increases dopamine levels in the brain, a neurotransmitter responsible for cognitive alertness (among other things). This dopamine release improves attention, motivation, and ability to focus. In turn, this directly helps the person with ADD/ADHD improve in a whole lot of performance areas, but it also helps through a more indirect route, as well. For example, it can improve a their ability to respond appropriately to social cues, stay on task in school or work activities, and control their impulsiveness, all of which can positively influence social relationships, self-esteem, self-confidence, and self-image. In some instances, stimulant medication may also increase activity in areas of the brain that help a person inhibit their actions (behavioral

control centers). However, medication alone is not sufficient to successfully manage ADD or ADHD: a great deal of hard work is also involved in learning appropriate coping and self-management skills (behavioral coping). Stimulant medication may provide someone with a better chance for behavioral interventions to be more effective. Without medication, it is more difficult (but not impossible) for a person with ADD or ADHD to generate the focused attention needed to learn these new intentional behaviors and skills.

Individuals with ADD or ADHD vary widely in their level of response and improvement (cognitive and behavioral) with stimulant medication—some improve dramatically, others only slightly, and some not at all. Some experience better outcomes with one type of stimulant medication than with another, while other individuals do better with different medications. In addition, medications may need to be switched if a person develops tolerance to one that previously provided good outcomes. Systematically tested evidence indicates that cognitive performance is not enhanced in individuals who do not have ADD or ADHD—despite widespread popular beliefs (NIDA, 2018). However, a person may have greater wakefulness which may allow a chance to study longer, which contributes to an elevated rate of prescription amphetamine misuse by high school and college students—it does not make them “smarter.” Use of these stimulant drugs by individuals *without* primary ADD or ADHD can stimulate hyperactivity while the drug is in their system. And, of great concern with this form of amphetamine prescription *misuse*: substances with the effect of increasing dopamine also have an increased probability of addiction because of the drug’s effects on the pleasure centers of the brain.

The National Institute on Drug Abuse (NIDA, 2018, however, summarizes data concerning individuals who do have ADD or ADHD and use stimulant medication as prescribed:

*Some people may be concerned about later substance misuse in children and teens who’ve been prescribed stimulant drugs to treat ADHD. Studies so far have not shown a difference*

*in later substance use in young people with ADHD treated with prescription stimulants compared with those who didn't receive such treatment. This suggests that treatment with ADHD medication does not positively or negatively affect a person's risk of developing problem use.*

## Methamphetamine.

Methamphetamine is a specific form of synthetically manufactured amphetamine. Vast amounts of methamphetamine are produced in illegal, foreign, or clandestine labs (Begun, 2020). Not only is methamphetamine a controlled substance (Schedule II in the U.S.

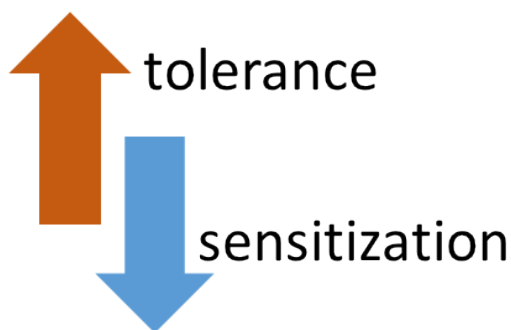


DEA system), many of the ingredients used in its manufacture are also controlled substances making it illegal to distribute or possess in excessive amounts. This is why U.S. pharmacies record and limit the amount of pseudoephedrine over-the-counter product a person attempts to purchase (e.g., Sudafed® and Contac®). Methamphetamine “street” names include meth, ice, crystal, crystal meth, and glass (Stoneberg, Shukla, & Magness, 2018; <https://www.justice.gov/archive/ndic/pubs5/5049/5049p.pdf>).

Because of its rapid effect on dopamine release in the brain's reward system (it is a smoked substance), methamphetamine has high addictive potential (NIDA, 2019a). It also has a relatively long half-life period, meaning that the “high” associated with its use may last 12 or more hours (<https://www.justice.gov/archive/ndic/pubs5/5049/5049p.pdf>). Individuals sometimes engage in binge use (sometimes called “tweaking” or “a run”) which involves repeated dosing over a period of days in an attempt to maintain the “high” from its use. “Because the pleasurable effects of methamphetamine

disappear even before the drug concentration in the blood falls significantly, users try to maintain the high by taking more of the drug,” sometimes foregoing food and sleep for several days while continuing to take the drug (NIDA, 2019b).

Tolerance may develop in both the relatively short term and long term with repetitive methamphetamine use (<https://www.ncbi.nlm.nih.gov/books/NBK64328/>). Repeated use also may produce drug **sensitization**—a term to describe a sort of reverse tolerance phenomenon. Sensitization involves the development of hypersensitivity to the effects of a drug like methamphetamine (Ujike & Sato, 2004). This process may be related to the neurotoxic effects of methamphetamine whereby neuronal dopamine storage vesicles rupture and dopamine leaks into synapses and inside the neurons themselves (<https://www.ncbi.nlm.nih.gov/books/NBK64328/>). In other words, it takes less of the drug to cause some of the psychoactive effects previously experienced at higher doses. And, just as **cross-tolerance** can develop, so too can **cross-sensitization**: it may be that a person will develop the same sensitization response to other substances in the same class/type as the one to which sensitization initially developed, although cross-sensitization may also affect drugs in a different class (Stewart & Badiani, 1993). It is exceedingly difficult to anticipate whether an individual person using a specific substance will develop tolerance or sensitization as a result of “chronic” use (Stewart & Badiani, 1993).



In addition to the risks associated with any stimulant misuse, methamphetamine use is associated with an increased risk of stroke. Magnetic resonance imaging (MRI) studies indicate that regular methamphetamine use is associated with a reduction in the density of grey matter in areas of the brain responsible for certain mental functions, and a significant amount of gray matter recovery is observed in individuals who remain abstinent for at least 6 months (Fowler, Volkow, Kassed, & Chang, 2007). After at least 9 months of methamphetamine abstinence, the number of dopamine transporters available in the brain may have increased again, more closely resembling the number in persons who did not use this drug, but poor memory and slowed motor deficits are not repaired (Fowler et al., 2007). According to the American Dental Association, methamphetamine use can have devastating effects on a person's dental health, resulting in severe tooth decay and gum disease, broken teeth, and tooth loss, sometimes called “meth mouth” (<https://www.mouthhealthy.org/en/az-topics/m/meth-mouth>). Additionally, methamphetamine may induce changes in the immune system which can worsen the consequences/severity of infectious diseases (e.g., HIV) if they are contracted (NIDA, 2019a).

Methamphetamine production introduces safety concerns over and above those related to all illicit drug distribution (“dealing”) activities. Numerous serious and explosive fires are attributed to “meth lab” errors which not only affect those involved in the illicit production of methamphetamine, but also neighbors, first responders, and whole communities.



*The chemicals used to produce methamphetamine are extremely hazardous. Some are highly volatile and may ignite or explode if mixed or stored improperly. Fire and explosion pose risk not only to the individuals producing the drug but also to anyone*

*in the surrounding area, including children, neighbors, and*



passersby. <https://www.justice.gov/archive/ndic/pubs7/7341/7341p.pdf>)

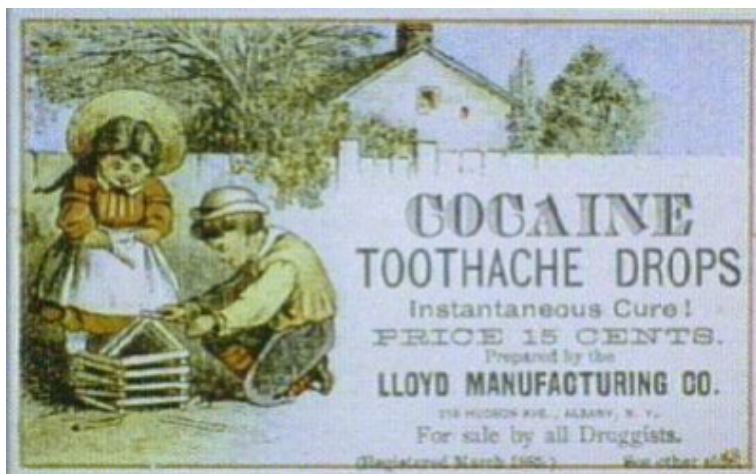
In addition, exposure to the toxic chemicals involved in production of methamphetamine poses significant health risks to the individuals involved in production, as well as to others unknowingly exposed—again, including neighbors, first responders, and whole communities. Trash resulting from production efforts often is heavily contaminated with toxins and polluted waste contaminates the production site. Entire buildings/houses may become contaminated with particulate contaminants spread through ventilation systems, making the site dangerous to unsuspecting occupants. Cleanup of a “meth lab” site often requires expensive and extensive hazardous material procedures. Recent statistics suggest that methamphetamine manufacture across the U.S. has declined as more of the drug is smuggled into the country, most from Mexico (Vestal, 2017). Historical data once suggested that methamphetamine use was more problematic in rural communities; current data indicate that this is no longer the case—methamphetamine use has moved into other, more urban communities, as well (Vestal, 2017).

## Cocaine.

Cocaine is a stimulant substance that also has anesthetic properties and a long history of use (and misuse) in the United States. It is derived from a naturally occurring source: plants in the *erythroxylum* group that include *coca*, *laetevirens*, and *novogranatense* variants. (Note that **coca** and **cacao** plants are different, see caffeine section below.) Cocaine has various “street names,” some of which relate to different forms (powder or crystal): for example, coke, crack, blow, and snow. “Freebasing” refers to heating and smoking a processed crystal form of cocaine (**crack**).

“Speedballing” refers to the practice of combining cocaine with heroin which increases the risk of heroin overdose because the initial effects of the cocaine (a stimulant) offset the sedating effects of the heroin, encouraging higher doses of heroin to be used; as the cocaine wears off quickly, the respiratory effects of the heroin predominate, leading to an overdose outcome (NIDA, 2016).

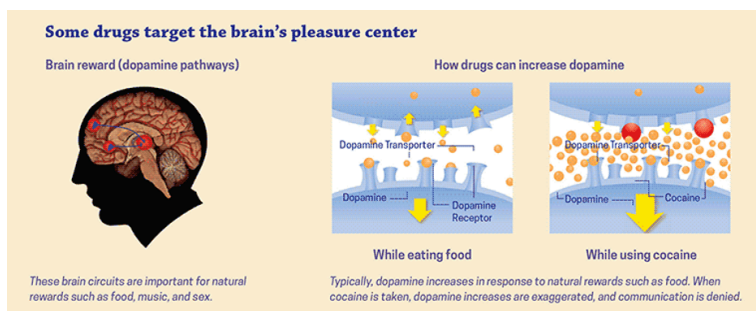
Common methods of cocaine administration lead to quick and intense psychoactive effects; it is usually “snorted,” smoked, or injected, but may be rubbed on gums to be absorbed. As with many other potentially addictive substances we have studied in this course, the intensity of the dopamine (brain reward) response to cocaine is far greater than is elicited endogenously from engaging in natural pleasure behaviors (<https://www.drugabuse.gov/publications/drugs-brains-behavior-science-addiction/drugs-brain>). The historical advertisement here refers to cocaine dissolved in solution intended to be applied (and absorbed) on the gums of a person experiencing dental pain or the gums of babies and young children experiencing pain associated with teething.



Cocaine is a quickly metabolized substance (short half-life), so

its psychoactive effects tend to be short-lived (<https://www.ncbi.nlm.nih.gov/books/NBK64328/>). While the “high” cocaine use produces occurs quickly, the effect also fades quickly, within minutes to an hour (NIDA, 2016). The duration of effect is somewhat dependent on route of administration: “The faster the drug is absorbed, the more intense the resulting high, but also the shorter its duration” (NIDA, 2016). In addition, like many stimulant substances, cocaine tolerance develops readily and individuals experience rebound effects after repetitive use—“the crash” after the high.

Bleeding within the brain and stroke have been associated with cocaine use, and long-term cocaine use is associated with a wide range of cognitive deficits in attention, memory, impulse control, and motor actions (NIDA, 2016). Gray matter density loss in the frontal cortex of the brain (responsible for logical thinking, goal setting, planning, and self-control, among other functions) is observed on MRI scans of individuals who have engaged in chronic cocaine misuse ((Fowler et al., 2007)). The physiological effects of cocaine use on other organ systems include cardiac arrest or seizures, the two most common causes of cocaine-related death (NIDA, 2016). The avenue of use may also increase risks of other health complications, such as infectious disease exposure and infections associated with injection use, or nasal passage deformations from “snorting” cocaine. Tolerance and withdrawal may develop with regular cocaine use, contributing to continued use in order to avoid or relieve withdrawal symptoms and requiring increasingly greater doses to do so.



## Caffeine.

Caffeine is included in this chapter because it is believed to be the stimulant (possibly the “drug”) most widely used globally (NIDA, 2014). “According to the Dietary Guidelines for Americans 2015-2020, more than 95 percent of adults in the United States consume foods and drinks containing caffeine. On average, U.S. adults consume between 110 and 260 milligrams (mg) of caffeine per day” (<https://www.medicalnewstoday.com/articles/324986>).

Caffeine is present in many forms of tea, coffee, chocolate, and “energy” products. Decaffeinated coffee, despite what the name suggests, retains some caffeine content although at low amounts; the word is not to be confused with caffeine-free (no caffeine). One problem with comparison of caffeine content across types and brands of caffeinated products is that serving sizes are often not comparable. For example, a “cup” of coffee might be 6 or 8 ounces, and in terms of specialty coffees like those produced by Starbucks®, serving sizes (8, 10, 12, 16, 20, 24 and 31 ounce containers) are confounded by what else is added (milk, sugars/syrups, and other flavor vehicles). Comparing soft drinks is equally confounded by serving size. For example, in the U.S., small, medium, and large beverages served at McDonald’s are 16, 21, and 32 ounces respectively; small, medium, large, and extra-large beverages served at Burger King are 16, 20, 29, and 38 ounces respectively. (There are significant sizing differences in other countries.)

While caffeine content may be presented per 8 ounces, most containers are consumed as a single serving despite the labeling reference to 2 or more servings per container. This harkens back to the “standard drink equivalent” problem we saw in our focus on alcohol module. (A table is presented below comparing caffeine content from various different sources with corresponding serving size indicated.) In addition, some products include other potentially (but not necessarily proven) stimulant contents, or substances that potentiate the stimulant action of caffeine, in addition to the

caffeine itself: ephedrine, guarana, taurine, and ginseng, for example. Caffeinated beverage products also may contain high sugar content. The “energy” boost from the combination of stimulants and sugar is often followed by the kind of rebound effect previously mentioned: an extreme, precipitous drop in energy when the effects of these combined substances wear off.

While prevalence estimates for caffeine use disorder among the general population vary widely, it likely lies in the neighborhood of 9% (Meredith, Juliano, Hughes, & Griffiths, 2013). Diagnostic criteria for caffeine withdrawal syndrome also are described in the DSM-5 (Meredith et al., 2013):

- that caffeine has been consumed for a prolonged period
- 3 or more symptoms within 24 hours following an abrupt cessation (or significant reduction) in caffeine consumption, including headache, fatigue/drowsiness, depressed mood or irritability, difficulty with concentration, and nausea/vomiting or muscle pain/stiffness
- these symptoms cause significant functional impairment (social, occupational, or other important areas)
- these symptoms are not associated with another condition, mental disorder, intoxication, or withdrawal from other substances.

**Tea.** “Tea” is derived from the leaves of tea plants (*camellia sinensis*) and sold as black (e.g., Early Grey, English breakfast), oolong, green, or white tea. Like other botanically derived substances, caffeine content varies as a function of growing conditions, age of the leaves (more oxidation means more caffeine), handling/storage, and how it is brewed. Matcha is powdered green tea and has levels of caffeine greater than what is present in a brewed green tea preparation: the entire leaf is consumed rather than discarded after producing the infusion made from the leaves (<https://www.oolong.com/life-in-flavor/230831/which-tea-has-the-most-caffeine/>). Decaffeinated tea is one of previously

mentioned types which have undergone one of several processes (carbon dioxide, ethyl acetate, methylene chloride, water processing) to remove as much caffeine as possible (<https://www.cupandleaf.com/blog/pros-and-cons-of-drinking-decaf-tea>). Caffeine-free herbal “teas” may be completely caffeine free because they do not actually contain any tea.

**Coffee.** Coffee is produced from the beans of coffee plants (*coffea*). The caffeine content in a coffee beverage is highly dependent on the type of coffee, different coffee brands, and how it is brewed. For example, cold brew coffee generally contains a somewhat higher caffeine content than typically brewed coffee, and the same amount of espresso contains much more caffeine (<https://www.medicalnewstoday.com/articles/324986#caffeine-content-by-coffee-type>) See the comparison chart below for more specifics.



**Chocolate.** Caffeine is present in the beans of cacao trees (*theobroma cacao*) from which chocolate is produced. (Note that chocolate producing cacao plants and cocaine producing coca plants are very different types.) In general, the darker the chocolate, the greater its caffeine content (<https://greatist.com/eat/does-chocolate-have-caffeine#1>). “White chocolate” is produced from cocoa butter in the cocoa bean rather than from chocolate solids (cacao nibs), hence it does not have the dark brown color of the bean and also does not have caffeine. As you can see from the comparison chart below, the amount of caffeine in chocolate is considerably less than in most of the caffeinated beverages but the amount consumed is dependent on serving size.



## Soft



**drinks and energy drinks.** A tremendous amount of caffeine consumption occurs with certain types of soft drinks, including the diverse class of “energy drinks” and “energy shots” currently on the market. Energy drinks are sold in container sizes similar to those of soft drinks, whereas energy shots are concentrated liquids sold in small containers (about 2 ounces). “Next to multivitamins, energy drinks are the most popular dietary supplement consumed by American teens and young adults” (<https://nccih.nih.gov/health/energy-drinks>). You may recall from our module focused on alcohol the potential problems associated with the practice of mixing alcohol and energy drinks—the stimulant of the energy drink offsetting some of the sedative effects of the alcohol, such that individuals may drink more in a short period of time than is safe (i.e., engage in high intensity binge drinking). The caffeine content in different products varies widely (see comparison chart below) and, adding to the confusion, the contents listing may refer to single serving sizes when the packaging encourages 2 or more servings be consumed at once.

### Other forms of caffeine.

Caffeine has been made available in liquid, powder, and gum forms, and many of these products have received significant warning from the U.S. Food and Drug Administration because of their potential for misuse. Products with very high caffeine content can lead to overdose, which can be fatal just as overdose with any other stimulant substance. Their caffeine content is presented in the comparison table below.



Comparison Chart of Caffeine Content in Different Products			
Product	approximate mg caffeine content	measure (approximate serving size)	approximate mg caffeine per fluid ounce
Tea: <sup>a</sup>			
black <sup>a</sup>	60-90	8 ounces	7.5-11.25
oolong <sup>a</sup>	50-75	8 ounces	6.25-9.38
green <sup>a</sup>	35-70	8 ounces	4.38-8.75
white <sup>a</sup>	30-55	8 ounces	3.75-6.88
matcha <sup>b</sup>	70	1 teaspoon	—
decaffeinated <sup>c</sup>	2	8 ounces	0.25
caffeine-free herbal <sup>c</sup>	0	—	0
Coffee: <sup>d</sup>			
brewed coffee	95	8 ounces	11.88
Starbucks Pike Place roast	235	12 ounces	19.58
Seattle's Best brewed	260	12 ounces	21.67



### Comparison Chart of Caffeine Content in Different Products

Dunkin' Donuts	210	14 ounces	15
cold brewed coffee	153-236	12 ounces	12.75-19.67
Starbucks cold brew & foam	155	12 ounces	12.92
espresso	63	1 ounce	63
Dunkin' Donuts espresso	85	single shot	—
Starbucks cappuccino	75	12 ounces	6.25
Dunkin' Donuts Americano	249	14 ounces	17.8
Seattle's Best mocha	80	12 ounces	6.67
decaffeinated coffee	2	8 ounces	0.25
Starbucks decaf Pike Place	20	12 ounces	1.67

### Comparison Chart of Caffeine Content in Different Products

Dunkin' Donuts decaf	10	14 ounces	0.71
Soft Drink: <sup>e</sup>			
Coca-cola® Classic	34	12 ounces	2.8
Diet Coke®	46	12 ounces	3.8
Coca-cola® caffeine free	0	12 ounces	0
Mountain Dew®	54	12 ounces	4.5
Diet Mountain Dew®	54	12 ounces	4.5
Pepsi-Cola®	38	12 ounces	3.2
Diet Pepsi®	35	12 ounces	2.9
Dr. Pepper®	41	12 ounces	3.4
Sunkist® Orange	19	12 ounces	1.58
Barq's® Root Beer	22	12 ounces	1.8
Tazo® Chai	47	8 ounces	5.9

## Comparison Chart of Caffeine Content in Different Products

Iced tea	47	8 ounces	5.9
Energy Drink/ Shot: <sup>1</sup>			
Spike	300	8.4 ounces	35.71
Redline Extreme	316	8 ounces	39.5
Rockstar Punched	180	12 ounces	15
Red Bull	113.5	12 ounces	9.46
Amp	106.5	12 ounces	8.88
Monster	120	12 ounces	10
Viso	300	17 ounces	7.65
Kickstart	69	12 ounces	5.75
5-hour energy shot	200	2 ounces	100
Chocolate <sup>9</sup>			
milk chocolate	4	1 ounce	4
medium dark (45-59% cacao)	12	1 ounce	12

Comparison Chart of Caffeine Content in Different Products			
dark (60-85% cacao)	23	1 ounce	23
chocolate milk (whole milk)	2	8 ounces	0.25
white chocolate	0	—	0
cocoa mix			
Liquid Caffeine <sup>h</sup>	500	1 ounce	500
Caffeine Gum <sup>i</sup>	40	1 piece	—
Powder <sup>j</sup>	3,200	1 teaspoon	—

## Comparison Chart of Caffeine Content in Different Products

<sup>a</sup> retrieved from <https://www.oola.com/life-in-flavor/2308311/which-tea-has-the-most-caffeine/>

<sup>b</sup> retrieved from <https://www.gotmatcha.com/matcha-and-caffeine/>

<sup>c</sup> retrieved from <https://www.cupandleaf.com/blog/pros-and-cons-of-drinking-decaf-tea>

<sup>d</sup> retrieved from <https://www.medicalnewstoday.com/articles/324986#caffeine-content-by-coffee-type>

<sup>e</sup> retrieved from <https://www.caffeineinformer.com/the-caffeine-database>

<sup>f</sup> retrieved from <https://www.caffeineinformer.com/energy-drinks-caffeine> and <https://spoonuniversity.com/lifestyle/caffeine-in-energy-drinks-ranked>

<sup>g</sup> retrieved from <https://greatist.com/eat/does-chocolate-have-caffeine#1>

<sup>h</sup> retrieved from <https://www.caffeineinformer.com/caffeine-content/5150-juice-caffeine-liquid>

<sup>i</sup> retrieved from <https://www.cnn.com/2013/04/30/health/caffeinated-gum/index.html>

<sup>j</sup> retrieved from <https://www.healthline.com/health-news/fda-cracking-down-on-caffeine-powder#2>

## Nicotine.

Leaves of the **tobacco** plant (*nicotiana tabacum*) also can produce a stimulant effect when chewed, “sniffed,” or smoked. Smoking tobacco or nicotine-containing products include cigarettes, cigars, pipe, hookah, and e-cigarettes (vaping). As of May 2016, federal



regulations on tobacco products were extended to include all these forms; as of December 2019, regulations concerning the sale of tobacco products raised the minimum age from 18 to 21 years; and, as of January 2020, the Food and Drug Administration issued policy regarding the sale of flavored **vaping** cartridges as a means of reducing their attractiveness to minors (NIDA, 2020)a.

Nicotine is the primary psychoactive substance involved, however there are more than 7,000 chemicals produced in tobacco smoke, many of which are known to cause cancer (<https://www.cancer.gov/about-cancer/causes-prevention/risk/tobacco/cessation-fact-sheet>). These include acetaldehyde (we learned about this one in the focus on alcohol module), arsenic, benzene, formaldehyde, and vinyl chloride, and several toxic metals/elements such as polonium-210, cadmium, chromium, and beryllium).

Nicotine has a relatively high addictive potential, being quickly absorbed, causing the release of epinephrine and activating the brain's dopamine reward circuits (NIDA, 2020a). Tobacco smoke contributes to a host of physical health problems/diseases: lung, oral, and other cancers; chronic bronchitis and emphysema; heart disease, heart attack, and stroke; Type 2 diabetes; cataracts; and poor pregnancy outcomes that include miscarriage, stillbirth, premature birth, and low birth weight, as well as learning and

behavioral problems (NIDA, 2020a). Chronic obstructive pulmonary disease (COPD) is an additional health risk associated with smoking (<https://medlineplus.gov/ency/patientinstructions/000696.htm>).

In the immediate short-term, nicotine has the familiar effects of the stimulant class of substances: fast (and sometimes irregular) heart rate, elevated blood pressure, appetite suppression, and increased focus of attention. Withdrawal symptoms include difficulty with paying attention, irritability, disordered sleep, increased appetite, and intense nicotine craving (NIDA, 2020a). Nicotine addiction related to use of tobacco products is discussed in the DSM-5 as a tobacco-use disorder. Many smoking cessation aids currently available offer a gradual withdrawal experience: gums, patches, and prescription medications (Portelli, Munjal, & Leggio, 2020).

*The Surgeon General's Report on Smoking Cessation, released in January 2020, offers evidence that smoking cessation is beneficial at any age, improves health status and enhances quality of life. It also reduces the risk of premature death and can add as much as a decade to life expectancy (NIDA, 2020).*

Smoking cigarettes is more than an individual, personal choice: the respiratory and cardiac health of others is affected by exposure to **second-hand smoke**. Infants and young children are also affected by exposure to **third-hand smoke**: the smoke residue that accumulates on hard and soft surfaces (e.g., carpeting, furniture, car seats) in areas where someone has been smoking (Begun, Barnhart, Gregoire, & Shepherd, 2014).



## E-cigarettes.

Electronic or **e-cigarettes** are devices intended to administer nicotine (and possibly other chemicals/substances) through inhaled vapor—similar to traditional “combustible” cigarette smoking but without actual combustion and tobacco leaves being involved. “There is **substantial evidence** that e-cigarette use results in symptoms of dependence on e-cigarettes,” and moderate evidence that the risk and severity of such dependence is lower than for combustible tobacco cigarettes (NAS, 2018). The term “vaping” is related to e-cigarettes also being named e-vaporizers. Some common nicknames for the devices are e-cigs, e-hookahs, hookah pens, vapes, vape pens, and mods (NIDA, 2020b).

Originally marketed as a tool to facilitate smoking cessation, these devices still deliver the addictive substance (nicotine) in amounts equivalent to that delivered from traditional combustible cigarettes (NAS, 2018). Hence, an individual who begins using e-cigarettes (vaping) remains at high risk of developing a nicotine addiction without ever having used a tobacco product. While they do deliver



fewer and lower levels of many toxic substances compared to combustible tobacco cigarette smoke, most e-cigarettes do emit potentially toxic substances, although the amount and type is variable (NAS, 2018). The evidence surrounding the potential for increased cancer risk with e-cigarette use (compared to no cigarette use) is just beginning to emerge, but is not yet conclusive (NAS, 2018). An additional concern is raised by results of studies showing that use of e-cigarettes in early adolescence was associated with transitioning to established cigarette use within the near future (Chaffee, Watkins, & Glantz, 2018; Levanthal et al., 2015; NAS, 2018).

Despite how they are marketed, e-cigarettes are not approved by the FDA for treatment of nicotine/tobacco addiction because strong evidence supporting use for this purpose is lacking; other approved alternatives are



backed by evidence (NIDA, 2020b; Portelli, Munjal, & Leggio, 2020). In terms of a harm reduction strategy (compared to combustible cigarette smoking), adults' use of e-cigarettes may reduce their exposure to some toxins and carcinogenic substances if used exclusively and not alternated with cigarette use (NAS, 2018).

In addition, there exist significant concerns regarding the safety of these devices. In addition to the health concerns previously noted, the devices themselves pose risks. “There is **conclusive evidence** that e-cigarette devices can explode and cause burns and projectile injuries” (NAS, 2018). As in the case of second-hand (and possibly third-hand) smoke exposure from traditional combustible cigarettes, there exists conclusive evidence that e-cigarette use causes increased concentrations of airborne particulate matter and nicotine in indoor environments and limited evidence of contaminants on indoor surfaces (NAS, 2018). The harm from

second-hand e-cigarette exposure is likely less than the harm associated with second-hand cigarette smoke exposure (NAS, 2018).



Exposure to the nicotine-containing liquids used to fill e-cigarette reservoirs can cause significant health problems if it comes into contact with a person's eyes or skin, or if it is consumed by drinking or injection (NAS, 2018). The most common types of nicotine overdose fatalities occur when young children consume these liquids (NIDA, 2020).

Finally, the FDA and the Centers for Disease Control and Prevention (CDC) have posted public alerts concerning clusters of vaping deaths attributed to poor quality vaping devices or nicotine liquid formulations, and especially to vaping liquids containing other substances such as THC (the psychoactive ingredient in cannabis) or vitamin E acetate (used as a thickening agent). As of February 4, 2020, a total of 2,758 hospitalizations or deaths were reported to the CDC related to **e-cigarette or vaping associated lung injury (EVALI)**; rates are gradually declining following a sharp increase in August and September of 2019 (CDC, 2020).



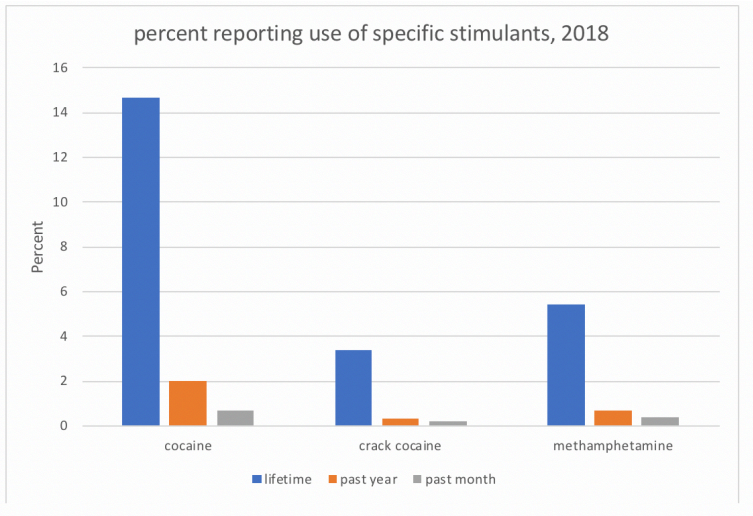
### **STOP & THINK**

Visit the caffeine informer website (<https://www.caffeineinformer.com/the-caffeine-database>) and calculate the caffeine content in the serving sizes of products you enjoy consuming. Consider the following questions:

- What effects do you think this might have on your health and behavior (positive and/or negative)?
- Do you think that a person might develop tolerance to the products you enjoy with a prolonged period of regular use at the levels you like to use them?
- Do you think a person might develop withdrawal symptoms if abruptly stopping the use of these products following a prolonged period of regular use at the levels you like to use them?
- What might be good ways for a person to change the “habit” of using these products?

# Ch. 2: Epidemiology of Stimulant Misuse

Globally, the highest prevalence of amphetamine misuse (separate from other stimulants) occurs in the United States: 2.95% (UNODC, 2018). In the U.S., according to the 2018 National Survey of Drug Use and Health (NSDUH; SAMHSA, 2019), 6.6% of respondents reported any use of stimulant drugs during the past year (this does not include caffeine and tobacco products). Misuse of any stimulants was reported by 1.9% of respondents for the past year (an estimated 4.7 million persons in the population) and 0.6% in the past month (an estimated 1.55 million in the population). Use of illicit stimulants figures were quite similar to the stimulant misuse figures: 1.2% in the past year and 0.4% in the past month. The chart below depicts the figures reported for specific types of stimulant substances: cocaine, crack cocaine, and methamphetamine.



[Click to download data for this chart](#)

## Cocaine.

Among the estimated 40 million persons aged 12 and older who have used cocaine during their lifetime, men are more likely to have done so than women (18.1% versus 11.5%). Persons reporting lifetime use of cocaine are more likely to self-identify their race/ethnicity as White (17.6%) or as two or more races (17.7%) than as American Indian/Alaska Native (16.4%), Native Hawaiian/Other Pacific Islander (14.0%), Hispanic/Latino (11.1%), Black or African American (8.5%), or Asian (5.4%). Lifetime crack cocaine use, however, is more likely reported among individuals who self-identify as American Indian/Alaska Native (5.4%) and as two or more races (5.0%) than the other race categories; White (3.8%) and Black or African American (3.6%) reported rates are fairly similar.

More individuals report having used methamphetamine in their lifetime, the last year, and last month than report having used crack cocaine; however, cocaine use was far more likely to be reported than methamphetamine (crack cocaine makes up a relatively small portion of the cocaine use reported in the United States). Still, an estimated 144,000 individuals aged 18 and older engaged in daily or almost daily use of cocaine during the past year (SAMHSA, 2019). Based on the NSDUH 2018 data, an estimated 977,000 individuals aged 12 and older had a substance use disorder during the past year that involved cocaine (SAMHSA, 2019). In this sample, the perceived risk of harm from using cocaine once or twice a week was rated as “great” by 86.5% and the perception that it was easy to obtain by 22.0% (SAMHSA, 2019).

## Methamphetamine.

Among the estimated 14.9 million persons aged 12 and older who have used methamphetamine during their lifetime, men outnumber

women (6.8% versus 4.2%; SAMHSA, 2019). The race/ethnic groups most likely to report having used methamphetamine during their lifetime are those self-identifying as American Indian/Alaska Native (12.7%), followed by individuals of two or more races (8.5%) and White individuals (6.9%). Least likely are Asian (1.5%) and Black/African American (1.1%) individuals (SAMHSA, 2019). An estimated 228,000 individuals in the U.S. engaged in daily or almost daily use of methamphetamine during the past year (SAMHSA, 2019). Over 1 million individuals aged 12 and older were estimated to have a past year substance use disorder involving methamphetamine based on 2018 NSDUH data (SAMHSA, 2019).

## Caffeine.

Estimates indicate that over 90% of adults in the U.S. regularly use caffeine and that average daily use exceeds 12 ounces of coffee or five 12-ounce servings of soft drinks (Meredith, Juliano, Hughes, & Griffiths, 2013). The Academy of Nutrition and Dietetics reported that an estimated 75% of children, adolescents, and young adults regularly consume caffeine (<https://www.eatright.org/food/nutrition/healthy-eating/is-your-kid-over-caffeinated>).

They also reported that the U.S. Food and Drug Administration (FDA) does not produce recommended limits on caffeine for children (they suggest 400mg for adults), but the Canadian government recommends limits based on age:

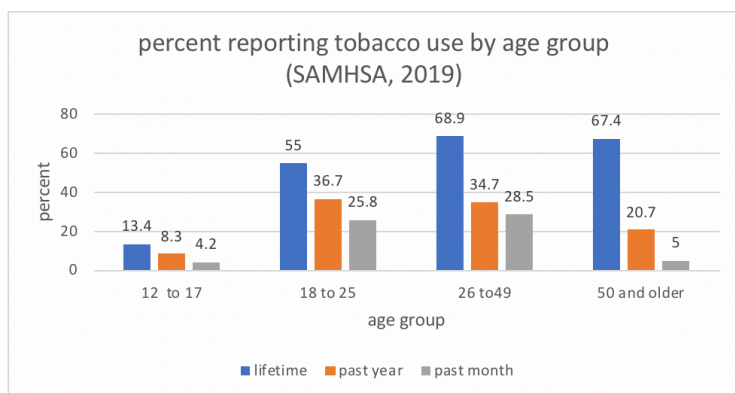


- 45 mg for children aged 4-6 years
- 62 mg for children aged 7-9 years
- 85 mg for children aged 10-12 years

The American Academy of Pediatrics “discourages” caffeine use (and other stimulants) by children and adolescents (<https://www.eatright.org/food/nutrition/healthy-eating/is-your-kid-over-caffeinated>).

## Tobacco/Cigarettes & Vaping:

Based on the 2018 NSDUH data (SAMHSA, 2019), an estimated 168 million individuals aged 12 and over (61.5%) have used tobacco products during their lifetime; 152 million used cigarettes (55.7% of population), and almost 84 million (30.6% of population) used cigarettes on a daily basis during their lifetime. Current tobacco use, as indicated by past month use, was estimated to occur among over 58 million (21.5%) with cigarette use accounting for almost 47 million individuals (17.2%). Unlike most of the other substances we have studied, adults in the 26 to 49-year-old range are very similar in pattern to emerging adults in terms of percent reporting past year or current tobacco use. In fact, this adult group is more likely to report current (past month) use than are emerging adults.



[Click to download data for this chart](#)

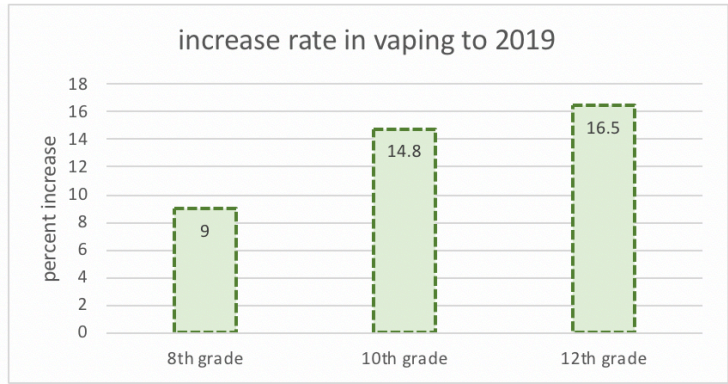
Like most of the other substances we have studied, men were more likely to report use of tobacco products than were women (69.2% versus 54.2% lifetime, 33.4% versus 20.5% past year, and 26.6% versus 16.6% past month). The race/ethnic groups reporting past month tobacco use at the highest rate self-identified as American Indian/Alaska Native (39.8%) or being of two or more races (27.1%). The following groups reported past month use at rates somewhat similar to each other: White (23.9%), Black/African American (23.0%), Native Hawaiian/Other Pacific Islander (24.9%). Study participants self-identifying as Hispanic/Latino (14.5%) and Asian (9%) reported past month use at the lowest rates. Past month use of tobacco products had an inverse relationship to income: persons living at less than 100% of the federally defined poverty threshold reported tobacco use at the highest rate (35.3%) compared to those in the group living at 100-199% of the poverty threshold (30.3%), or those living at 200% or more than the poverty threshold (23.7%).

Perhaps the most telling statistics presented in the NSDUH 2018 data (SAMHSA, 2019) relate to nicotine dependence among persons aged 12 and older. Based on these data, an estimated 26 million experienced past month nicotine dependence. Another way of



looking at the data shows that 55.7% of individuals reporting past month cigarette use experienced nicotine dependence—in other words, current use is not casual among the majority who smoke cigarettes.

With regard to vaping/e-cigarette use, we can turn to the Monitoring the Future (MTF) study results concerning 8th, 10th, and 12th graders surveyed across the United States. Between 2017 and 2019, the rate at which vaping was reported continually increased—by 9% among 8th graders, 14.8% among 10th graders, and 16.5% among 12th graders: “among the largest increases ever recorded for any substance in the 45 years that MTF has tracked adolescent drugs use” (Johnston et al, 2020). Among 12th graders, 35.5% reported engaging in this behavior and nicotine vaping “continues to rank among the lowest of all substances for perceived risk” (Johnston et al, 2020). By comparison, the rates of students in these age groups engaging in traditional combustible cigarette use has been on a “fairly steady and substantial decline” since a peak in 1996 (Johnston et al, 2020).



[Click to download data for this chart](#)

## Ch. 3: Summary

We covered a great deal of territory in this module concerning stimulant substances. You read descriptions of amphetamines (mostly prescription drugs), methamphetamine, cocaine, caffeine, and nicotine-containing products (tobacco and e-cigarettes). A description of the psychoactive and other physical effects of stimulant substances was supplemented in many of the sections with actions and effects more specific to individual substances. A great deal of the effect on addictive potential and health concerns related to the mode of administration, and we were reminded once again that the more quickly and powerfully a substance affects the pleasure areas of the brain, the greater the potential for addiction to that substance. Some discussions were presented concerning what happens when stimulant substances are combined with other drugs or substances (including alcohol). Substance use disorder criteria were referenced, as were some of the symptoms of withdrawal from stimulant substances. The concept of cross-tolerance was presented again in this module, along with the introduction of a new concept: drug sensitization. And, the module concluded with a chapter presenting some of the epidemiological evidence related to use of various stimulant substances. You are now ready to review some of the key terms presented in this module.

# Module 10: Key Terms

**amphetamine:** central nervous system stimulant medications, most by prescription but some are only distributed illicitly in the U.S.

**attention deficit disorder (ADD):** a diagnosis ascribed to individuals exhibiting a specific constellation of behaviors that include distractibility and disorganization, among others.

**attention deficit hyperactivity disorder (ADHD):** a diagnosis ascribed to individuals exhibiting a specific constellation of behaviors that include the attention deficit disorder characteristics, as well as impulsivity and excessive activity, restlessness, and fidgeting, among others.

**autonomic nervous system (ANS):** a part of the nervous system responsible for directing many involuntary bodily functions (e.g., breathing, heart rate, digestion, and glandular activity); it is comprised of the sympathetic and parasympathetic nervous systems that act in concert to maintain a state of homeostasis.

**cacao:** of the plant type used in producing cocoa, chocolate, and cocoa butter (used in making white chocolate); sometime spelled cocoa (not to be confused with coca).

**caffeine:** a stimulant compound naturally occurring in several types of plants around the world, including tea, coffee, and cacao/cocoa.

**coca:** referring to the plant type used in producing cocaine (not to be confused with cacao or cocoa).

**cocaine:** a powdered stimulant substance produced from coca or produced synthetically, having both stimulant and anesthetic effects.

**crack:** a crystal form of cocaine, as opposed to the powdered form.

**cross-sensitization:** drug sensitization developed to one substance through repeated use that carries over as sensitization to another substance despite its never having been used.

**cross-tolerance:** drug tolerance developed to one substance through

repeated use that carries over as tolerance to another substance despite its never having been used.

**e-cigarette:** a device designed to heat nicotine (or other substance) liquid by battery power rather than combustion (burning) as would be the case with traditional cigarettes.

**e-cigarette or vaping associated lung injury (EVALI):** the label assigned to a respiratory illness/symptom complex attributed to harm (or death) from using e-cigarettes/vaping products.

**epinephrine:** a central nervous system neurotransmitter (sometimes called adrenaline) with stimulant effects on the autonomic nervous system and dopamine centers.

**homeostasis:** the state of balance/equilibrium that systems attempt to achieve and maintain to preserve energy; generally, a healthy state in living organisms.

**methamphetamine:** a synthetic form of amphetamine with longer lasting effects; having some recognized medical uses, it is primarily produced, distributed, and used illicitly in the U.S

**nicotine:** the primary psychoactive (stimulant) component in tobacco, also may be produced in liquid or powdered form.

**norepinephrine:** a central nervous system neurotransmitter (sometimes called noradrenaline) with effects on the autonomic nervous system.

**parasympathetic nervous system:** a component of the autonomic nervous system responsible, in part, for regaining a state of homeostasis following an event where the “fight or flight” response has been triggered.

**second-hand smoke:** vapors and residue exhaled by someone who is smoking tobacco products and inhaled by a person who is not smoking.

**sensitization:** repeated use of a substance leading to a decrease in tolerance—it takes less of the substance to produce the same effects previously experienced at higher doses.

**sympathetic nervous system:** a component of the autonomic nervous system responsible, in part, for initiating a “fight or flight” response to trigger events.

**third-hand smoke:** smoke residue that accumulates on hard and soft surfaces (e.g., carpeting, furniture, car seats) in areas where someone has been smoking.

**tobacco:** a specific type of nicotine-rich plant, the leaves of which are processed to make a variety of products (cigarettes, cigars, and smokeless tobacco).

**vaping:** the use of electronic/e-cigarettes.

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## PART XI

# MODULE II: FOCUS ON OPIOIDS

### Reading Objectives

The readings for our module concerned with opioids introduces concepts essential for understanding the nature of these substances, opioid misuse and opioid use disorder (OUD), and the “opioid epidemic” declared by health officials in the United States. After engaging with these reading materials and learning resources, you should be able to:

- Explain similarities and differences between opiates, opioids, narcotics, heroin, fentanyl, and carfentanil;
- Describe effects of opioid substances and how they are administered;
- Identify key features of the recent “opioid epidemic” and overdose statistics;
- Explain basic principles of neonatal withdrawal syndrome (NWS);
- Define key terms related to opioids



# Ch. 1: Introduction to Opioids

In this chapter, we examine opioids—what they are, how they are used, how they are misused, their effects, and some statistics concerning their use/misuse. Note that a portion of this chapter's contents informed and were informed by materials presented in Begun (*in press*), NAS (2017), DEA (n.d.), and NIDA (2018). For more in-depth information, consider reviewing the Council on Social Work Education (CSWE) Learning Academy course *America's Opioid Crisis: A Primer for Social Work Educators* and the review by Reber, Schlegel, Braswell, and Shepherd (*in press*), members of an interdisciplinary team serving children and families in the neonatal intensive care environment.

## What are Opioids?

*Opioids* are substances that interact with naturally occurring opioid receptors in the human body and are either derived from opium or synthetically constructed/manufactured. You may hear the words opioid and opiate being used interchangeably—this is not



entirely correct. [Opiates](#) are a subset of opioids derived from opium—substances such as [heroin](#), morphine, and codeine are produced from the seeds or what is extracted as a resin from the opium poppy, *papaver somniferum* (somniferum meaning “bringer

of sleep”—you may recall the scene in Wizard of Oz where Dorothy and Toto fall asleep in the poppy field outside of the Emerald City). The poppy seeds we eat in various foods (e.g., poppy seed bagels, lemon poppy seed muffins, poppy seed salad dressing) also come from this plant. Many varieties of this plant do not produce opium in any significant amounts and are grown for ornamental purposes. The opium poppy is most heavily (commercially) harvested in Southeast and Southwest Asia, Mexico, and Columbia, supplying pharmaceutical companies (if not others). A great deal of international economics, policy, policing, and trafficking are involved in opioid production and distribution.

Originally, opioids referred to synthetically or semi-synthetically produced substances. Semi-synthetic substances include opium to some extent but there are synthetic components involved, as well; for example, oxycodone and hydrocodone are semi-synthetic opioid drugs. Purely synthetic opioid examples include [methadone](#), tramadol, [fentanyl](#), and [carfentanyl](#). Opioids are manufactured both by licensed/controlled pharmaceutical companies in the U.S. and other countries, as well as illegally produced and distributed by uncontrolled clandestine laboratories around the world (<https://www.cdc.gov/drugoverdose/data/fentanyl.html>). The word opioid is now used to describe the whole group of substances that bind with opioid receptors in the brain and body, whether naturally or synthetically occurring. Hence, opiates are generally considered as a group of opioids these days.

**Narcotics** is another word you may have encountered. Narcotics are substances intended for use in treating moderate to severe pain. Examples of opioid pain medications are:

- oxycodone (OxyContin®, Percocet®)
- hydrocodone (Vicodin®)
- codeine (often combined with acetaminophen or with cough suppressants)
- morphine
- fentanyl, carfentanyl, and other fentanyl analogs

The word narcotic refers to the capacity for these drugs to induce a state of narcosis—a state of stupor or unconsciousness produced by narcotics or other substances (<https://www.merriam-webster.com/dictionary/narcosis>).



The word narcotic is not much used in health or mental health professions anymore once it became so heavily associated with the legal system and illegal drug trafficking.

## Opioid Use

The medical use of opioids is to provide pain relief. Common types include prescription drugs like oxycodone, hydrocodone, codeine, morphine, and fentanyl, as well as combination medications (e.g., aspirin or acetaminophen plus opioid, such as Percodan®, Percocet®, or Tylox®). Prescription misuse of opioids generally involves pills which are either swallowed or crushed and injected or snorted. Some forms are meant to be absorbed through the skin in a controlled dose, such as the fentanyl patch, or to be administered intravenously (IV) under medical supervision. These drugs are used in human and veterinary medicine.



As learned in Module 6, nonmedical use of opioid drugs may represent a gateway to heroin use: based on 2011 data, about 80% of individuals who used heroin misused prescription opioids first (NIDA, 2019). However, “more recent data suggest that heroin is frequently the first opioid people use. In a study of those entering treatment for opioid use disorder, approximately one-third reported heroin as the first opioid they used regularly to get high”

(NIDA, 2019). It is also important to recognize that, in the 2011 data, about 4-6% of individuals who misuse prescription opioids transitioned to heroin: “This suggests that prescription opioid misuse is just one factor leading to heroin use” (NIDA, 2019).

## Opioid Use Disorder (OUD)

You learned in an early course module about the Diagnostic and Statistical Manual of Mental Disorders version 5 (DSM-5) criteria for substance use disorder (SUD). The criteria apply to the diagnosis of [opioid use disorder \(OUD\)](#) as one type of SUD. The criteria are not applied when opioid medication is used strictly as prescribed and under medical supervision. The 11 criteria are as presented in your earlier lesson but specifically related to opioid use (APA, 2013):

- opioids often used in larger amounts or over a longer period of time than intended;
- experiencing a persistent desire or unsuccessful efforts to cut down or control opioid use;
- a great deal of time is spent in activities necessary to obtain opioids, use opioids, or recover from opioid use effects;
- experience of craving, or a strong desire to use opioids;
- recurrent opioid use resulting in failure to fulfill major role obligations at work, school, or home;
- continued opioid use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of opioids;
- important social, occupational or recreational activities given up or reduced because of opioid use;
- recurrent opioid use in situations where it is physically hazardous;
- continued use despite knowledge of having a persistent or recurrent physical or psychological problem likely caused or

- exacerbated by opioid use;
- developing opioid tolerance, as defined by either: (a) a need for markedly increased amounts of opioids to achieve intoxication or desired effect, or (b) markedly diminished effect with continued use at the same opioid amount/dose; and
- experiencing withdrawal, as manifested by either: (a) the characteristic opioid withdrawal syndrome, or (b) the same or closely related opioid substance taken to relieve or avoid withdrawal symptoms.

A diagnosis of OUD is made when 2 or more of these 11 symptoms are present during the same 12-month period. Severity of the diagnosed opioid use disorder (OUD) is determined by the total number of these 11 symptoms experienced:

- mild OUD (2-3 symptoms),
- moderate OUD (4-5 symptoms), or
- severe OUD (6 or more symptoms).

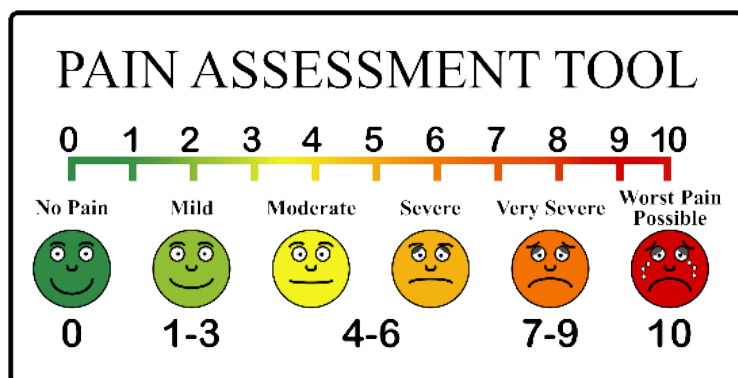
## Opioid Effects

Opioid medications are used medically for their ability to control pain ([analgesic](#) effects), as well as to reduce/control/suppress coughing and diarrhea. In addition to pain relief, opioids confer a general sense of well-being, relaxation/reduced tension, reduced anxiety, reduced aggression, and potentially a state of euphoria. Opioids also cause drowsiness, difficulty concentrating, apathy/lack of motivation, slowed physical activity/reactions, constricted pupils, constipation, nausea/vomiting, and slowed breathing (DEA, n.d.; NIDA, 2019). The psychoactive effects of opioids come from their bonding to naturally occurring opioid receptor sites on neurons, leading to a surging release of dopamine to the pleasure areas of the brain, blocking pain, and rewarding the substance use

behavior. The surge in “pleasure” from [exogenous](#) opioid use is many times greater than naturally occurs at opioid receptor sites from [endogenous](#) sources, such as pleasure from eating or sex. Because the brain adapts to the presence of the extrinsically introduced opioids, tolerance occurs with repeated use and withdrawal symptoms appear when use is stopped. At this point, a person may take the drugs not so much to “get high” as to avoid or reduce the low or negative feelings that occur without the drug.

Opioids are powerfully psychoactive and addictive, with chronic use accompanied by the development of tolerance, as well as withdrawal symptoms: watery eyes, runny nose, yawning, sweating, restlessness, irritability, loss of appetite, nausea, tremors, increased heart rate, increased blood pressure, chills, flushing, drug craving, and severe depression (DEA, n.d.). Withdrawal symptoms generally disappear over days to weeks without additional dosing, depending on the type of substance involved and the severity of the physical/psychological addiction developed. The experience of craving and depression may persist much longer, contributing to a heightened risk of suicide during early recovery. Other than overdose risk, the greatest acute danger of opioid misuse is the effect of slowing (or stopping) a person’s breathing, an effect compounded by combining opioids with some other substances with this same effect (e.g., alcohol, barbiturates). Other opioid effects include sleepiness, confusion, possible pneumonia. Over time, opioid use/misuse can lead to insomnia and increased sensitivity to pain—despite being used to control pain, a person’s pain tolerance threshold may be lowered with chronic opioid use/misuse. Heroin use may also lead to liver and kidney disease (NIDA, 2018).





Physical dependence on opioids can develop even when used as medically prescribed; a person may become dependent on the class of substances, such that heroin and prescription misuse may become intertwined depending on a person's access to the different substances. Heroin and other opioids are frequently combined with other substances to amplify their effects or to counter some of their side or withdrawal effects. Opioids like fentanyl or carfentanil may be added to heroin (or other substances) to amplify effects and increase drug trafficking profits (Taxel, 2019). Because these added substances are many times more potent than the primary substance, their addition greatly increases the risk of overdose.

**Mode of administration.** Depending on the mode of administration, opioid misuse may increase the risk of infection and infectious disease exposure. Since many individuals engage in injection use of these substances, this is of significant concern and worthy of harm reduction attention. Heroin ("horse," "smack") is generally injected, smoked, or sniffed/snorted. The avenue of administration can amplify the addictive potential of heroin or other opioids—the faster the drug reaches the brain in large concentration, the greater the addictive potential (i.e., injection is faster than oral drugs). The avenue of administration also may introduce additional risks, such as injection site infection, injection

site vein collapse, and exposure to infectious diseases, such as HIV and hepatitis (Rassool, 2011).

**Opioid overdose.** The symptoms of a heroin overdose as described by Rassool (2011, p. 73), include:

- shallow breathing or difficulty breathing
- weak pulse and/or low blood pressure
- delirium
- drowsiness
- muscle spasms
- disorientation
- bluish-color of lips and fingernails (from low oxygen levels)
- dry mouth
- pinpoint (small) pupils
- coma.

Obviously, the risk of a dangerous or fatal overdose is tied to the amount of a substance used. However, amounts are relative depending on the potency/strength of the substance. Fentanyl is recognized as contributing to a significant surge in overdose events and deaths—pharmaceutical-grade fentanyl is 50 times stronger than the average potency of heroin (CDC, 2018). One reason it becomes even more dangerous is that it often is added to illegally distributed substances, like heroin (but also other types), without the knowledge of persons using them. The person is unknowingly delivered a more potent dose than accounted for, contributing to their overdose risk: increased opioid overdose death rates “are being driven by increases in fentanyl-involved overdose deaths, and the source of the fentanyl is more likely to be illicitly manufactured than pharmaceutical” (CDC, 2018). Dosing uncertainty is compounded by uncertainty as to the strength/concentration of illegally manufactured opioids, including fentanyl and its variants ([analogs](#)). While not as common an opioid problem as fentanyl, pharmaceutically produced carfentanil, intended for use in large animal veterinary care (e.g., elephants), has 100 times the potency

of fentanyl (5,000 time the potency of heroin). This diagram visually shows these ratios.



Another contributor to overdose happens after a person has developed opioid tolerance and uses these substances at an increasingly higher level (dose) over time. Then, if the person ceases using the drug either as a result of treatment/recovery efforts, hospitalization, during a period of incarceration, or for some other reason (e.g., no access following a community-wide natural disaster disrupting distribution), tolerance reverts to a lower level (dose). Individuals unaware of this change in tolerance may resume use at their prior higher tolerance level, amounting to an overdose at their current lower tolerance level. This phenomenon is suspected as a contributor to relatively high mortality rates observed among formerly incarcerated individuals in the days immediately following release from jail or prison.

### **Opioid overdose reversal.**

Opioid overdose might be reversed by administering [naloxone](#) (Narcan® or Evzio®). As an [opioid antagonist](#), naloxone binds to opioid receptors in the body and blocks the opioid's

effects. The amount of opioid reversal drug needed depends on the dose and strength of the opioid involved in the overdose event. This means the overdose reversal drug needs to be available, available in a quantity sufficient for managing the overdose event, and someone needs to be able to administer it in the event of overdose. The opioid



overdose reversal drug puts the person into immediate withdrawal, which is exactly what the person may have been using the opioids to avoid. It can be life-saving, however, since it can restore normal breathing suppressed by an opioid overdose. Many communities and institutions have adopted naloxone distribution programs, policies, and Good Samaritan laws allowing lay persons on the scene to administer the overdose reversal care before professionally trained first responders can do so and making it easier for individuals to obtain and carry a reversal kit for use in the event of an overdose. Ideally, an overdose incident can be followed up with outreach efforts designed to engage the individual in OUD treatment—handling the event as a “reachable” moment.

#### **Medication for treating opioid use disorder (MOUD).**

Treating opioid use disorder (OUD) often involves prescribed medications as part of an evidence-based intervention plan—[medication assisted treatment \(MAT\)](#). Three medications have been approved by the U.S. Food and Drug Administration (FDA) for this purpose and have a strong evidence base supporting their use: [methadone](#) (itself a synthetic opioid), [buprenorphine](#) (an opioid partial agonist), and [naltrexone](#) (an opioid antagonist). Additional medications are in trial for treating OUD, as well (Portelli, Munjal, & Leggio, 2018). These medications work on the same brain opioid receptor systems affected by opioid misuse—humans naturally have opioid receptors in the brain and some other parts of the body.

Like any medication, there are advantages and disadvantages to their use. The primary take-home messages about medication assisted treatment of opioid use disorder: the success rates in retaining individuals in treatment and reducing the illicit use of opioids is greater with MAT than without (and better than placebo), and MAT (plus counseling) is more cost-effective than treatment without medication. Despite this mounting evidence, medication in treating opioid use disorder currently is grossly underutilized (Portelli, Munjal, & Leggio, *in press*). The Substance Abuse and Mental Health Services Administration, in its Treatment Improvement Protocol (TIP) #63 (SAMHSA, 2018), and Portelli,

Munjal, and Leggio (in press) compared these three options for medication assisted treatment (SAMHSA, 2018).

**Methadone.** Methadone is used both in medically supervised withdrawal from opioids (short-term) and longer-term recovery maintenance. It is a Schedule II controlled substance, having addictive potential itself, and is generally only legally distributed through specially licensed/federally certified opioid treatment programs or inpatient hospital settings treating opioid use disorder. [Methadone maintenance therapy \(MMT\)](#) is the primary and most researched approach to use of methadone in treating OUD.

One main advantage of methadone over heroin is that it is longer acting, so that it is dosed daily rather than heroin which is taken multiple times per day to avoid withdrawal symptoms. This creates a more even psychoneurological experience of withdrawal which supports opioid abstinence goals. As an agonist, it creates some of the same effects as opioids/heroin (respiratory depression, sedation, heart rhythm changes, low blood pressure, constipation), but in a more controlled dosing pattern and at a level insufficient to create a “high” when used as prescribed. Methadone “helps individuals experiencing OUD reduce withdrawal symptoms and craving for opioids by delivering the desired drug effect over a longer period than the abused substances” (Portelli, Munjal, & Leggio, *in press*). Pharmaceutical-grade methadone is more predictable, and presumably safer, than illicit substances. Methadone can be gradually tapered off to eventually leave a person opioid-free and is known to reduce risk of overdose-related death, as well as harms associated with illicit opioid misuse (e.g., HIV/Hepatitis infection and criminal activity; SAMHSA, 2018). Because of its effectiveness, the World Health Organization (WHO) “lists it as an essential medication” (SAMHSA, 2018). An advantage of methadone controls is that programs have the potential to engage recipients in other recovery-support services, as they must attend the program daily to receive the medication.

Methadone is considered a safer alternative to illicit opioids for

opioid use disorder management during pregnancy since it reduces the occurrence/cycles of withdrawal which pose a significant risk to the fetus and viability of the pregnancy (causing premature labor contractions, among other risk events). However, the baby has a high likelihood of experiencing neonatal withdrawal syndrome (NWS) at birth and will need to be weaned off the methadone just as in the case of any other opioid.

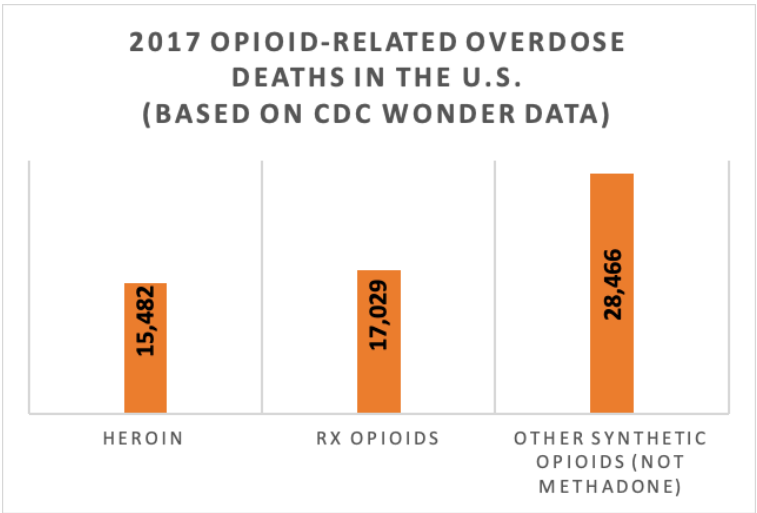
**Buprenorphine.** Buprenorphine is used both in medically supervised withdrawal from opioids (short-term) and longer-term recovery maintenance. It is a Schedule III controlled substance originally introduced for pain management. It may be prescribed outside of federally certified opioid treatment programs by professionals with a prescribing waiver, distributed by a pharmacy, making it more easily accessible than methadone. As a partial agonist, buprenorphine has some of the same effects as opioids (respiratory depression) without creating the “high” when used as prescribed, but it also may precipitate some degree of opioid withdrawal symptoms (nausea, sweating, insomnia, pain). For this reason, adhering to the treatment may be more difficult than with methadone. Buprenorphine does have some addictive potential but less so than methadone. Risks are greater when combined with use of other drugs that affect breathing (e.g., benzodiazepines). It can be delivered by monthly injection as a slow-release option is available. Naloxone (an opioid antagonist that precipitates opioid withdrawal symptoms and used in opioid overdose reversal) is sometimes combined with buprenorphine to help prevent its misuse. Buprenorphine allows for gradual tapering to eventually no longer needing opioids or medication to manage withdrawal. Because of its effectiveness, the World Health Organization (WHO) “lists it as an essential medication” (SAMHSA, 2018). Buprenorphine may cause neonatal withdrawal syndrome (NWS) but is considered safer than alternatives—however, pregnant women may be less likely to remain in buprenorphine treatment than with methadone, which increases risks to the fetus and pregnancy (see CSWE course, *America’s Opioid Crisis*).

*Naltrexone.* Naltrexone is used in opioid use disorder relapse prevention after medically supervised withdrawal. As an opioid antagonist, it blocks opioid receptor sites, acting in a longer-acting manner but similarly to the overdose reversal drug, naloxone. Thus, Naltrexone minimizes the rewarding effects of opioid use, but also can precipitate opioid withdrawal. It requires a prescription but is available through primary care providers without specialty waivers or certification as an opioid treatment program. It does carry a risk of potential side effects (nausea, anxiety, depression, insomnia, liver toxicity, suicidality, sedation, loss of appetite, dizziness, muscle cramping). In addition, because it can precipitate withdrawal, it reduces pre-existing tolerance so that if a person relapses to using opioids, the risk of overdose is increased. Naltrexone in a monthly injectable form was equally effective to oral buprenorphine in maintaining post-withdrawal opioid abstinence in one study, and in another, showed a lower rate of relapse than no medication (SAMHSA, 2018).

## Opioid Use Statistics

Global data from the United Nations Office on Drugs and Crime (UNODC, 2019) indicate that opioid use accounted for 66% of deaths attributed to drug use disorders during 2017 and that 1.1 percent of the global population (53.4 million persons) aged 15–64 years engaged in opiate and/or use of prescription opioids for non-medical purposes. The problem was greatest in North America (4% of the population used opioids in the past year). More than 28,000 deaths in the U.S. during 2017 involved synthetic opioids (not including methadone)—a significant increase from 2016—with the highest death rates in West Virginia, Ohio, and New Hampshire (CDC, 2018). These increases are reported to be driven by illicitly manufactured fentanyl-involved overdose. This figure shows the number of opioid-involved overdose deaths during 2017, comparing

heroin, prescription (Rx) opioids, and other synthetic opioids—mostly fentanyl (<https://www.drugabuse.gov/related-topics/trends-statistics/overdose-death-rates>). These numbers were all greater than the numbers of overdose deaths attributed to cocaine (13,942), benzodiazepines (11,537), psychostimulants other than cocaine (10,333), or antidepressants (5,269). Opioid-related deaths in the U.S. increased by over 290% in the 15 years between 2001 and 2016, from 0.4% of all deaths to 1.5%, and represented 20% of deaths among 24- to 35-year-olds during 2016 (Gomes, et al., 2018). In 2017, the U.S. Department of Health and Human Services declared the opioid crisis a public health emergency, stating that over 130 individuals died every day (47,600 in a year) from opioid-related drug overdose and problems of opioid addiction (<https://www.hhs.gov/opioids/about-the-epidemic/index.html>).



While profoundly disturbing, deaths due to overdose are not the only statistics of concern regarding opioid misuse in the U.S. For example, neonatal withdrawal syndrome (NWS) rates in 2015 multiplied by 8 times the 2006 rate in Ohio

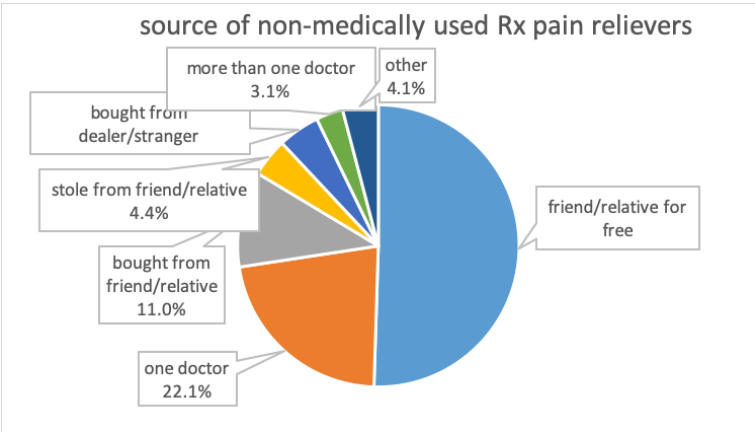


(<https://www.wcbe.org/post/rate-ohio-babies-born-addicted-drugs-increasing>) and children's services in Franklin County Ohio reported 70% of children under one year of age and 28% of all children in their custody had parents using opiates at the time of removal from the home (<https://adamhfranklin.org/opiateactionplan/>). Again in Franklin County Ohio, for every overdose death, there were 32 emergency department visits. According to data from the 2018 National Survey on Drug Use and Health (NSDUH, 2018), over 3 million individuals aged 12 and over were estimated to engage in current (past month) opioid use outside of what was medically prescribed. The good news: the 2018 estimate of 3.042 million is lower than 2017 estimate of 3.549 million, a statistically significant difference. The following table presents statistics by age group for several pieces of information concerning non-prescription use of pain relievers (synthetic opioids) and heroin (estimated numbers across the U.S. and percent of population). Harm perception and lack of access are two important components in preventing substance misuse and the youngest group was least likely to perceive harm, although they were also the least likely to perceive having easy access.

created using NSDUH 2018 data ( <a href="https://www.samhsa.gov/data/report/2018-nsduh-detailed-tables">https://www.samhsa.gov/data/report/2018-nsduh-detailed-tables</a> )			
	12-17 years	18-25 years	26+ years
# past month use pain relievers not prescribed	161,000	475,000	2,216,000
# past month heroin use	8,000	61,000	285,000
#substance use disorder: pain relievers	104,000	248,000	1,343,000
#substance use disorder: heroin	4,000	101,000	421,000
% great harm perceived: heroin try once or twice	64.5%	82.5%	89.3%
% great harm perceived: heroin use once or twice a week	84.0%	93.3%	95.7%
% perceiving heroin fairly or very easy to obtain	8.1%	14.5%	18.0%

The 2018 Monitoring the Future study reported the lowest rates for 12-graders' past-year misuse of prescription opioids since 200—about 3.4%, which represents a 64% decline (<http://www.monitoringthefuture.org/pressreleases/18drugpr.pdf>). According to data from the 20125 NSDUH survey, most individuals (97.5%) prescribed opioid pain relievers did not misuse them (Hughes et al., 2016). Among individuals aged 12 and older who did misuse prescription opioids, according to 2013-2014 NSDUH data, their most common source was from a friend or relative for free; the second most common was from a single prescribing doctor (Lipari & Hughes, 2017; see the figure recreated from data presented in Lipari & Hughes, 2017). Recent initiates and those occasionally engaged in prescription pain reliever misuse

were far more likely to report friend/relative for free as their source than individuals engaged in frequent use; the latter group was more likely than the others to report their source as one doctor or bought (either from friend/relative or drug dealer/stranger) (Lipari & Hughes, 2017).



A variety of responses have potentially cut into the ease-of-access from prescribers (reporting systems, prescriber education), as well as family/friends (drug take-back programs, public education efforts).





*An interactive H5P element has been excluded from this version of the text. You can view it online here:*

[https://ohiostate.pressbooks.pub/  
swk5805coursebook/?p=216#h5p-47](https://ohiostate.pressbooks.pub/swk5805coursebook/?p=216#h5p-47)

# Ch. 2: The “Opioid Epidemic” Narrative

## How the Opioid Epidemic Came to Be

The book *Dreamland: The True Tale of America’s Opiate Epidemic* (Quinones, 2015) traces the interwoven threads and historical events that, combined, created the significant eruption of opioid misuse, opioid use disorder, and opioid-related deaths in the U.S. The number of individuals using heroin, as well as the number with opioid use disorder where heroin was the substance used, more than doubled in the years between 2002 and 2014 (NIDA, 2018). While the timeline presented in the analysis began in 1804 with the distillation of morphine from opium for the first time, the story gained momentum during the 1990s with:

- Xalisco Boys heroin distribution system evolved across multiple cities and states in the U.S.
- OxyContin (time-released oxycodone) becomes available and heavily marketed for treating chronic pain
- first “pill mills” (as pain clinics) emerge on the scene
- health care providers are urged to assess and manage pain as “the fifth vital sign.”

The development and marketing of new (lucrative) opioid formulations, combined with prescribers’ inadequate training about addiction, sincere wish to alleviate patients’ pain, and dependence on positive patient evaluation ratings contributed to opioid overprescribing practices, not to mention ethically questionable “pill mill” practices (NAS, 2017). As the number of opioid prescriptions dispensed in the U.S. nearly tripled from 1991 to 2011,

there was a parallel near-tripling in the number of opioid-related deaths (NIDA, 2018). The intense (and apparently misleading) opioid marketing practices of various drug companies has led to a series of multi-billion dollar lawsuits against the companies by individuals surviving opioid addiction, family members of individuals who died from opioid use, and communities facing staggering costs from law enforcement, emergency response, and health/mental health/addiction care services required in response to increased opioid misuse. Concurrently, Mexican and Columbian heroin sources expanded dramatically across the U.S., making an easily injectable white powder form of heroin easily accessible and relatively low-cost: major factors in heroin use initiation by many individuals (NIDA, 2018). Fentanyl entering the country through Mexico and China are also major contributors to the crisis.

According to the timeline, concerns about overdose deaths began to be expressed in the early 2000s; while heroin addiction and overdose had historically been recognized as problems in urban, minority communities, the problem was emerging in new populations, new geographical areas, and explosively larger numbers. In 2008 drug overdose surpassed auto fatalities as the leading cause of accidental death in the U.S (Quinones, 2015). For instance, in Ohio's Franklin County, the number of accidental drug overdose deaths increased by 71% during the four years between 2012 and 2016 (<https://adamhfranklin.org/opiateactionplan/>). By 2014, concern about the addictive behavior pattern of shifting from pain pills to heroin was evident, too (Quinones, 2015). It is possible that making opioid drugs (OxyContin in 2010, for example) more difficult to misuse—harder to dissolve or crush for injection or “snorting”—may have contributed to an increase in heroin use (Evans, Lieber, & Power, 2017). Again in Ohio's Franklin County, the number of persons infected with Hepatitis C (often associated with intravenous drug use) increased by 68% between 2012 and 2016 (<https://adamhfranklin.org/opiateactionplan/>).

On the illegal drug market, fentanyl is a favored product because what might cost 1-2 cents to purchase from suppliers (the front end

of the distribution chain) might end up being sold “on the street” for \$10–\$20 (the back end of the distribution chain) (NAS, 2017). By comparison, heroin might be less expensive than pharmaceutical opioids on the front end of the distribution chain, fentanyl is much less expensive than heroin, making it a far more lucrative product in which to traffic (NAS, 2017).

Strategies to address the opioid problem traditionally fall into 4 general categories (NAS, 2017):

1. create abuse-deterrent formulations (non-addictive forms) and alternative pain management strategies that may include behavioral health interventions with or without medication;
2. reduce supply/access/availability through efforts such as restricting lawful access through DEA scheduling, influencing prescribing practices and imposing prescription drug monitoring programs, training healthcare practitioners about substance misuse and substance use disorders, preventing diversion from legal to illegal use (e.g., with easy to access, regular drug take-back programs to eliminate access to leftover drugs), and addressing pharmaceutical company marketing practices;
3. reduce demand through patient and public education campaigns, promote access to evidence-supported treatment for OUD, initiate treatment engagement efforts with individuals who experience overdose, need for emergency department care, or other health-related consequences; and,
4. reduce harmful consequences associated with use, such as overdose prevention and response efforts (e.g., dissemination of opioid overdose reversal training and kits), supervised drug injection sites, disperse tools for checking “street” drugs for fentanyl, wound care education for individuals engaged in injection use, providing immunity from prosecution for possession of substances or paraphernalia when first responders treat an overdose event.

A combination of supply, demand, practitioner and public education, and legal/policy actions may be turning the tide. As previously noted, the trend statistics appear to have declined somewhat during 2018 compared to the most recent prior years. Persistence of vigilance is necessary, however, to ensure a continued decline with the hope of continuing to reverse the epidemic. Of concern, for example, are the previously presented data on youthful perceptions of the potential harm being lower than the perceptions of harm held by current adults.



Imagine that a family relative at Thanksgiving dinner asked you to explain the opioid crisis in America. In 4-6 sentences, what would you say? What elements/threads would you need to include in your narrative?



# Ch. 3: Prenatal Opioid Exposure

In this chapter, we look at what is known about prenatal exposure to opioids and a common result: [neonatal withdrawal syndrome \(NWS\)](#) at birth. While the emphasis here is on outcomes for the baby, it is important to recognize that opioid misuse/OD greatly amplifies the risk of maternal health complications and death—representing “a leading cause of pregnancy-related deaths in the U.S.” (Sanjanwala & Harper, 2019, p. 192). Furthermore, opioid misuse during pregnancy often leads to mothers losing child custody, and parents using opioids are less likely to retain child custody than parents using other substances (Hall et al., 2016). Evidence suggests that medication-assisted treatment (MAT) for opioid use disorder increased the odds of parents retaining child custody (Hall et al., 2016).

## Prenatal Opioid Exposure

As the explosion of prescription opioid drug misuse developed, the rate of maternal opiate use during pregnancy increased dramatically. In the U.S., the rate of mothers experiencing opioid use disorder at the time of hospital delivery in 2014 increased by more than 4 times compared to the 1999 rate—and OUD statistics do not fully describe opioid misuse during pregnancy (Haight et al., 2018). Opioid misuse/OD during pregnancy contributes to a multitude of poor infant outcomes: stillbirth, preterm birth, low birth weight, neonatal withdrawal syndrome, and sudden infant death syndrome (Kandall, et al., 1993; Sanjanwala & Harper, 2019). The problem stems from opioids passing to the developing fetus’s

brain and body organs through the placenta, then interacting with the baby's opioid (mu-)receptors which resemble the adult pattern of distribution in the spinal cord by about 24 weeks (Ray & Wadhwa, 1999). Like an adult, the fetus can develop tolerance to the drug and withdrawal symptoms when the drug is no longer available with placenta separation from the mother at birth.

Each time a pregnant mother experiences withdrawal it places stress on the fetus and jeopardizes the pregnancy. For this reason, opioids with longer half-life (such as methadone) deliver a more even dose over 24 hours, reducing the mother's withdrawal episodes, and improving outcomes for the pregnancy compared to a drug like heroin which has a relatively short half-life and multiple experiences of withdrawal daily (Reber et al., *in press*). The presence and severity of NWS is unpredictable, even with controlled dosing guidelines for methadone management: "some babies exposed to relatively low doses may experience severe NAS while other babies exposed to even higher doses may not" (Reber et al., *in press*).

## Neonatal Withdrawal Syndrome

Previously called [\*\*neonatal abstinence syndrome \(NAS\)\*\*](#), the currently preferred term is neonatal withdrawal syndrome (NWS). The shift in terminology from abstinence to withdrawal more accurately described the infant's experience of abruptly transitioning from the prenatal environment involving opioid exposure to the post-birth opioid withdrawal experience. As the rate of maternal opioid misuse during pregnancy has grown, so too has the rate at which NWS occurs: 6 per 1,000 live births in 2013 compared to 1.5 in 1999 (Reber et al., *in press*). The rate also varies by geographical trends in opioid prescribing and OUD; for example, NWS ranges from fewer than 1 per 1,000 births in the District of Columbia to almost 50 per 1,000 in Vermont (Ko et al., 2016).

Withdrawal symptoms may appear up to 5 days following birth;

symptoms following heroin exposure (4-24 hours) is typically more rapid than for methadone (24-48 hours) or buprenorphine (48-72 hours), due to the different half-lives and pharmacokinetic actions of these different substances (Reber et al., *in press*). Babies prenatally exposed to opioids may exhibit difficulty with breathing, meconium aspiration complications, feeding, sepsis (systemic infection), gastrointestinal symptoms (diarrhea leading to dehydration), moderating autonomic nervous system functions (e.g., managing body temperature, sweating), poor sleep, irritability, fussiness, jitteriness, seizures, and even death (Reber et al., *in press*; Sanjanwala & Harper, 2019). Their symptoms make these babies more difficult to care for and contribute to later developmental and health complications, as well as child maltreatment risks; long-term outcome effects of chronic exposure during prenatal development are unclear from the literature, in part because it is difficult to separate the impact of confounding, co-occurring, and post-birth risk and vulnerability factors and social determinants of health (Reber et al., *in press*). Initial newborn hospitalization is typically as long as 20 days for babies affected by NWS, resulting in tremendous direct medical costs across the nation estimated at \$500 million to \$1.5 billion annually (Reber et al., *in press*).

Screening for possible maternal opioid use/OPUD is an important aspect of early detection and intervention during pregnancy and for infants born following prenatal opioid exposure—whether the opioid use was the result of following healthcare provider prescribing protocols, prescription drug misuse, methadone as MAT, or illicit substance use (e.g., heroin). All babies deemed at risk should be carefully screened and monitored throughout the first 3- to 5-day period (Reber et al., *in press*). Other psychoactive substances (polydrug use) can also affect the timing, degree, and outcomes of opioid withdrawal in newborns (Reber et al., *in press*). Treatment typically involves medically managed, step-wise withdrawal protocols using opioid medications (morphine, methadone, buprenorphine) to gradually wean the infant from all substances. However, if symptoms are not severe, supportive

interventions and medical management of symptoms may suffice without escalation to pharmacologic treatment (Reber et al., *in press*). Non-pharmacologic interventions include:

- skin-to-skin contact with mother (and other parent/caregiver);
- low-stimulation environment (light and noise), which may include music and/or massage therapy;
- reduce auto-stimulation with tight swaddling, timely response to hunger and discomfort cues, providing comforting positions like gentle swaying/rocking;
- attending to hydration and increased caloric needs, including involving multi-disciplinary teams to aid in feeding infants with dysregulated suck/swallow/breathe patterns and “sensitive stomach” formulas as a supplement to/replacement for breast milk which can carry opioids to the nursing infant (Reber et al., *in press*).

The benefits of additional family support services following the infant’s release from care have been demonstrated (Reber et al., *in press*).



Imagine that a family member corners you into a conversation

triggered by a local news story about a residential treatment program serving pregnant mothers illicitly using opioids for up to two years after their newborns are released from the hospital. The program diverts the mothers from criminal justice system consequences as long as they follow the program (MAT is used in combination with behavioral counseling and wrap-around support services) and the mothers maintain child custody while child protective services continue to supervise/monitor care of their infants. This family member announces, “I think it is wrong to provide these people free drugs, especially while they are pregnant—making things worse for those babies. They should lock these women up and throw away the key and put those babies up for adoption by people who will love them.” How might you explain such a program to this family member? Why might this be better for the babies?

## Ch. 4: Summary

In this module, you were introduced to several different types of opioid substances and learned definitions of opioids, opiates, and narcotics. How opioids are used/misused and their effects were presented, along with information concerning their potential health and addiction risks—including risks associated with routes of administration (e.g., injection use). The nature of opioid overdose was described and the use of opioid overdose reversal drugs explained. In addition, medications approved for use in medication-assisted treatment (MAT) of opioid use disorder were described along with how they might be used in treatment and recovery (more on this topic in a later module about pharmacotherapy). The story of “how we got into this mess” called the opioid epidemic was outlined; a resource for learning more about the different interwoven threads is the *Dreamland* book (Quinones, 2015). The Preface to the NAS (2017) report represents a strong summary to much of the content presented in this module. They indicate that it is clear “that the opioid epidemic will not be controlled without deploying multiple policy tools. Increasing access to treatment for individuals with OUD is imperative, together with a substantial program of research to develop new nonaddictive treatments for pain.” The report goes on to recommend changes in the health care system where collective responsibility for over-prescribing resides, law enforcement efforts to curtail trafficking in illegally manufactured and distributed opioids (in the U.S. and abroad), and a strong commitment to prevention and treatment of substance use disorders more generally.

This module concluded with a discussion of the effects of opioid exposure during prenatal development and how neonatal withdrawal syndrome (NWS) might be managed. At this point, you are prepared for the next module which looks into other forms of prescription drug misuse and use of medications for managing

and supporting recovery from substance use disorder. Several additional substances are examined, including over-the-counter substances, that did not fit easily into the “types” we have discussed so far.

# Module 11: Key Terms

**analgesic:** having the ability to relieve pain (usually refers to a drug).

**analogs:** having a chemical structure similar to another compound but different in one or more component, often developed and distributed as means of circumventing laws restricting manufacture/distribution of the drug for which it is an analog.

**buprenorphine:** an opioid medication used in the treatment of opioid use disorder (partial mu-opioid receptor agonist).

**carfentanil (or carfentanyl):** an extremely powerful, addictive synthetic opioid originally intended for large animal veterinary practice.

**endogenous:** originating inside the body.

**exacerbated:** meaning that something is made worse.

**exogenous:** originating outside the body.

**fentanyl:** an extremely powerful, addictive synthetic opioid, often mixed with other substances, with a strong presence in illicit drug trafficking but originally intended for prescription pain management in human and veterinary medicine.

**heroin:** a powerful, addictive opioid derived from morphine (naturally derived from opium poppy), produced in various forms (e.g., white powder, brown powder, black tar) and having no recognized medical use in the U.S. (Schedule I drug by the DEA).

**medication assisted treatment (MAT):** use of prescription medications under medical supervision to treat substance use disorders of various types and deter relapse through management of cravings and withdrawal symptoms and/or interrupting the substance-use reward system; recommended that behavioral interventions accompany MAT.

**methadone:** a long-acting opioid used in treating opioid use disorder (originally developed as a strong pain reliever/analgesic narcotic) working as an opioid receptor agonist; because of its addictive potential, it remains a Schedule II drug by the DEA.



**methadone maintenance therapy (MMT):** an integrated treatment protocol for recovery from opioid use disorder, combining long-term prescribing of methadone in combination with behavioral counseling and other social services to support recovery.

**naloxone:** an opioid antagonist drug with low addictive potential used both in the immediate reversal of opioid overdose (causing immediate withdrawal) and in longer-term medication assisted treatment of opioid use disorder.

**naltrexone:** an opioid/opiate antagonist that blocks positive effects from using opioids or alcohol, decreasing the desire to use these substances in the future.

**narcotics:** drugs designed for pain management/relief; the term now commonly refers to illicitly used/trafficked opioids.

**neonatal abstinence syndrome (NAS):** term commonly used for neonatal withdrawal syndrome (see below).

**neonatal withdrawal syndrome (NWS):** a cluster of symptoms frequently observed in newborn infants who have been prenatally exposed to opioids, triggered by separation from the source of these substances via the placenta causing the infant to experience substance withdrawal.

**opiates:** psychoactive substances that interact with opioid receptors and are produced from natural sources (e.g., opium, morphine, codeine); opiates are now considered to fall under the broader opioid category.

**opioids:** psychoactive substances that interact with opioid receptors; may be “natural,” synthetic, or partially/semi-synthetic in origin.

**opioid agonist:** a substance or drug that activates opioid receptors resulting in some (partial agonist) or all (full agonist) opioid effects—heroin, methadone, morphine are full opioid agonists and buprenorphine is a partial agonist.

**opioid antagonist:** a substance or drug that blocks opioid receptors thereby interfering with opioid effects—naloxone is an opioid antagonist.

**opioid use disorder (OUD):** a diagnostic label applied when 2 or

more of 11 criteria listed in the DSM-5 are met within the same 12-month period, with degree of severity determined by the total number of criteria met.

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## PART XII

# MODULE 12: FOCUS ON OTC & PRESCRIPTION DRUGS, INHALANTS, STEROIDS, AND PHARMACOTHERAPY AGENTS

### Introduction

With the exception of two types of substances we have not yet covered (inhalants and anabolic steroids), this module takes a somewhat different turn from the formula we followed in our modules about alcohol, sedative-hypnotic/CNS depressant, cannabis/hallucinogen, stimulant, and opioid substances. In this module we address **prescription drug misuse**, which was touched on in our modules concerning sedative-hypnotic/CNS depressant, stimulant, and opioid misuse. Along with prescription misuse, we address **over-the-counter (OTC) substance misuse**, as well. The module concludes with a chapter concerning the use of medications as therapeutic agents as an option in treating substance misuse/use disorders (pharmacotherapy), particularly in the detoxification (detox) phase of intervention.

## Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Describe the patterns and effects of inhalant and anabolic steroid misuse;
- Identify issues related to prescription and OTC drug misuse;
- Identify basic principles of pharmacotherapy applied in treating substance use disorders, including in “detox” protocols;
- Explain key terms and concepts related to misuse of inhalants and steroids, prescription and OTC substance misuse, and pharmacotherapy.



# Ch. 1: Focus on Inhalants and Anabolic Steroids

In addition to the kinds of substances we have previously studied that individuals use by inhaling (i.e., by smoking or inhaling vapors), a variety of chemicals intended for medical, cleaning, or industrial uses may be misused by inhaling, as well. Additionally, we need to consider the misuse of anabolic steroids. While these are often used/misused for their effect on body shape and athletic performance, they do have psychoactive effects, as well.

## What Is Inhalant Misuse



**Inhalants** are volatile substances at room temperature; in other words, they are in a gas, aerosol, or vapor form. Inhalant misuse involves breathing in these substances, inhaling them, in high concentrations. For example, they may be concentrated in plastic bags, in latex/rubber balloons or gloves, or soaked on cloths held over the nose and/or mouth (Baydala et al, 2010). Many (but not all) inhalant substances are legally accessed and easily accessed in the home, workplace, or stores, making their misuse attractive to youth when there are no age restrictions on their purchase or use. Some common terms to describe inhalant use are sniffing, huffing, and bagging (Baydala et al., 2010; NIDA, 2017a).

Various chemicals included in lists of potentially psychoactive inhalants are (Baydala et al., 2010; NIDA, 2017a):

- nitrous oxide, ether, or chloroform (medical anesthetics, generally not easily accessed legally);
- propane;
- whippets/whipped cream aerosol dispensers;
- nitrites (labeled video head cleaner, room odorizer, leather cleaner, liquid aroma; including amyl nitrites, butyl nitrites called poppers, snappers, amys);
- cleaning fluids, spot remover, degreasers (including benzene);
- gasoline and other fuels or lighter fluid (e.g., butane);
- spray paint, varnish, lacquer, resins;
- paint/lacquer thinner, paint remover, polish remover (including acetone);
- computer keyboard or other electronic contact cleaners;
- felt-tip markers;
- correction fluids (mostly older types);
- glues and adhesive sprays; and,
- other aerosol products, like hair spray, spray deodorant, or vegetable oil sprays.

The psychoactive effects associated with most inhalants are short-lived—a matter of a few minutes. This contributes to a tendency to use the inhalant substance repeatedly over a brief period of time (NIDA, 2017a).

## Inhalant Effects

Inhalants are selected for misuse because of their “ability to rapidly induce euphoria,” as well as their stimulant, disinhibiting, and hallucinatory effects (Baydala et al., 2010, p. 443). As soon as these immediate effects diminish, the individual may experience depression, dizziness, disorientation, loss of coordination, slurred speech, drowsiness, and/or headache (Baydala et al., 2010). Specific to nitrite misuse is a drop in blood pressure (hypotension) that may

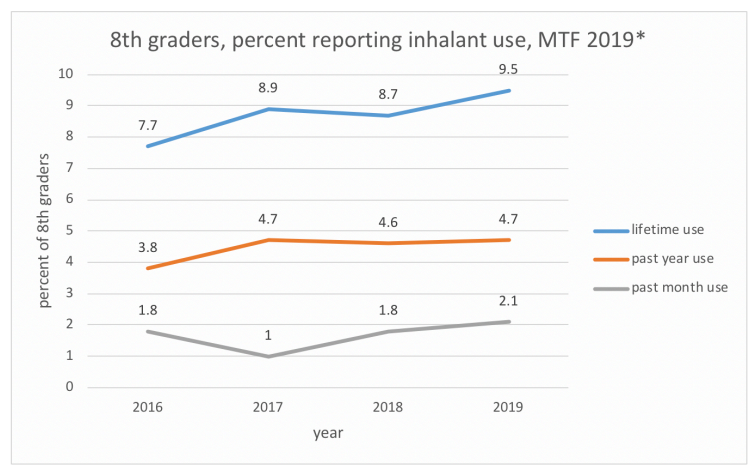
lead to a loss of consciousness (syncope). Their misuse as a club drug is related to sexual effects (penile engorgement and sphincter relaxation conducive to anal sex; Baydala et al., 2010).

Inhalant misuse is extremely concerning as a public health issue because of the considerable potential for brain damage, damage to other organ systems, and lack of oxygen (hypoxia) associated with this form of substance misuse. Baydala et al (2010) report that sudden death may occur as a result of the physical effects of these chemicals (especially the heart); injury may result from engaging in risky behavior due to the disinhibiting effects these substances may have (drowning, falling, burns, or exposure to the elements/cold weather); and, suffocation (hypoxia, lack of oxygen) may occur in the process of using inhalants. In the long term, irreversible damage to the brain and other organ systems (heart, lungs, bone marrow, liver, kidneys) may result, especially (but not only) when chronic misuse occurs (Baydala et al., 2010).

## Epidemiology of Inhalant Misuse

The 2019 Monitoring the Future (MTF; <https://www.drugabuse.gov/trends-statistics/monitoring-future/monitoring-future-study-trends-in-prevalence-various-drugs>) data show a perplexing trend in the comparison of 8th, 10th, and 12thgrade students' report of lifetime inhalant use—the 8th grade cohort reported it more often than did 10th graders or 12th graders: 9.5% compared to 6.8% and 5.30%. With most other substances, lifetime use is greater as students age. (This 8th grade cohort also more often reported lifetime use of cough medicine misuse, heroin use, and methamphetamine use than did the 10th or 12th graders.) They also reported past year (4.70%) and past month (2.10%) inhalant misuse more often than their older peers (2.80% and 1.90% for past year misuse by 10th and 12thgraders; 1.10% and 0.90% for past month misuse by 10th and 12th graders). Data concerning a

trend from 2016-2019 suggests that lifetime and past year inhalant misuse have increased significantly (see the trend chart below).



\*Based on data retrieved from <https://www.drugabuse.gov/trends-statistics/monitoring-future/monitoring-future-study-trends-in-prevalence-various-drugs>

*Click to download chart data*

Inhalants are typically a “youthful” choice for substance misuse (NIDA, 2017a; Baydala et al., 2010). This point is reinforced by the 2019 NSDUH survey data (SAMHSA, 2019). In 2018, almost 25 million individuals (9.1% of the population) aged 12 and older in the U.S. were estimated to have engaged in inhalant misuse during their lifetime, over 2 million (0.7%) during the past year, and almost 700,000 (0.2%) during the past month. The past month (current) misuse of inhalants was reported by 0.7% of adolescents aged 12 to 17 years, 0.4% of emerging/young adults aged 18 to 25 years, and 0.1% of adults aged 26 or older.

Lifetime reported inhalant use by individuals aged 12 and older was more common among men than women (11.8% versus 6.5%), however among the current cohort of 12 to 17-year-olds, the statistics were almost equal with girls reporting at a slightly higher



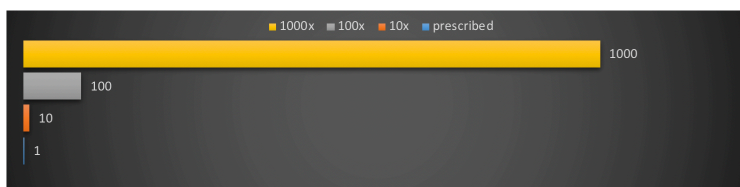
rate than boys (8.6% versus 8.5%). Within this same young cohort, lifetime inhalant use was most often reported by individuals self-identifying as being of two or more races (9.7%), White (9.2%) and Black or African American (8.7%). Unlike most of the other types of substances we have studied, Asian individuals in this young cohort were not the group with the lowest reported lifetime inhalant use (7.8%). This rate was similar to that reported by Hispanic or Latino individuals (8.0%) and greater than the rate reported by American Indian/Alaska Native individuals (5.1%). The 12 to 17-year-old cohort with lifetime inhalant use was estimated across the U.S. to be about 662,000 individuals.

## What is (Anabolic Androgenic) Steroid Misuse

Steroid misuse typically refers to the use of **anabolic steroids** in ways not medically prescribed. Anabolic (androgenic) steroids differ considerably from corticosteroid medications used for treating inflammation, such as occur in autoimmune disorders like rheumatoid arthritis, allergic reactions, eczema, or asthma events. Anabolic androgenic steroids (AAS) are synthetic compounds related to or mimicking testosterone, a hormone naturally occurring in the bodies of both men and women in differing amounts. Anabolic is the common name for these substances: anabolic refers to tissue building and androgenic means promoting

masculine characteristics (DOJ, 2004). Medically, anabolic steroids may be used to address delayed puberty and loss of muscle mass with certain diseases (NIDA, 2018a).

Their misuse by athletes, body builders, and members of certain physically demanding occupations to boost strength and endurance, or recover from muscle injury, taps into the tendency to develop muscle with these compounds and typically involves doses 10 to 1000 times what is medically prescribed (NIDA, 2018a; WHO, 1993). Steroid misuse comes in multiple forms of administration: oral, injection, or topical application to the skin. Common names for anabolic steroids include roids, juice, pumpers, gym candy, arnolds, and weight trainers (DOJ, 2004). Various OTC health supplements introduce anabolic steroid precursor chemicals to the body, leading the body to produce more of the steroid than would normally occur—with some of the same effects as using steroids themselves (DOJ, 2004). The major source of illicit steroids is from other countries where prescriptions are not required (DOJ, 2004), raising the specter of poor quality and/or contaminated drugs (WHO, 1993).



[Click to download chart data](#)

### Effects of (Anabolic) Steroid Misuse

In the short-term, steroid misuse does not have easily recognizable psychoactive effects. Compared to the other substances we have studied in this course:

“The most important difference is that steroids do not directly activate the reward system to cause a ‘high’; they

also do not trigger rapid increases in the brain chemical dopamine, which reinforces most other types of drug taking behavior” (NIDA, 2018a).

The mental effects of steroid misuse are more likely to appear over time with repeated (and high dose) misuse. These include (NIDA, 2018a; WHO, 1993):

- paranoia, extreme jealousy, and delusions;
- impaired judgment;
- mania;
- extreme irritability and aggression (sometime called “roid rage”).

Other organ systems are also likely to be affected by steroid misuse, including the kidneys, liver, cardiac (heart enlargement, high blood pressure, increased risk of stroke or heart attack), and male sexual organs (testicular shrinkage, decreased sperm count/infertility, breast development, and increased risk for prostate cancer). In women, the effects tend to defeminize/masculinize the body (reduced breast size, loss of menstrual cycles, deeper voice, male-pattern baldness, growth of excess facial/body hair).

Steroids do have some degree of addictive potential (NIDA, 2018a) despite their differences from the other substances studied this semester. As a substance use disorder, it falls under the classification of “other substance-related disorder” in the DSM; the International Classification of Diseases (ICD-10) presents criteria for dependence specific to steroid misuse. These criteria include evidence of tolerance, withdrawal syndrome with reduced use, strong desire to take steroids, difficulty in controlling steroid use, and neglecting other interests and persisting in use despite harmful consequences. Steroid withdrawal symptoms include:

- steroid craving
- fatigue
- restlessness

- mood swings
- loss of appetite
- disordered sleep (insomnia)
- decreased sex drive
- depression (including suicidality).

### **Epidemiology of Steroid Misuse**

According to the MTF 2019 data (<https://www.drugabuse.gov/trends-statistics/monitoring-future/monitoring-future-study-trends-in-prevalence-various-drugs>), lifetime, past year, and past month misuse by 12th graders is relatively uncommon (1.50%, 1.60%, and 1.60% respectively). Steroid misuse is considerably more common among men compared to women, being athletic is a significant predictor, and one study identified a considerable number of noncompetitive bodybuilders as being engaged in this practice (AlShareef & Marwaha, 2019). Multiple reports suggested that it is difficult to be much more precise about the epidemiology since so few substance-related studies ask about steroid use/misuse.







*An interactive H5P element has been excluded from this version of the text. You can view it online here:*

[https://ohiostate.pressbooks.pub/  
swk5805coursebook/?p=228#h5p-53](https://ohiostate.pressbooks.pub/swk5805coursebook/?p=228#h5p-53)

## Ch. 2: Focus on OTC and Prescription Drug Misuse

Let's begin with debunking a myth commonly held in the general public: that over-the-counter (OTC) drugs, sold in general drug and grocery stores, are safe because they do not require a prescription to purchase. The simple truth is that ANY drug, whether OTC or prescription, is potentially dangerous if misused. All drugs have potential side effects. This is a common feature of both prescription and OTC drugs. It is also of concern with many herbal remedies and dietary supplements marketed as health or mental health promoting products. Consider, for example, the scientific debate concerning the effectiveness and safety of St. John's Wort for treating depression—in some studies it was no better than placebo, in others it was equally beneficial as prescription medications, and it is clearly dangerous to use in combination with other medications (or substances), including other anti-depressants, birth control pills, pain medication, heart medication, cancer and HIV treatments, and others (NCCIH, 2018). OTC treatments often lack strong evidence of effectiveness, may not be thoroughly evaluated for negative side effects, and may delay a person seeking evidence-supported treatment options.

So, what is the difference between OTC and prescription products? The main difference goes back to what we previously learned about the Drug Enforcement Administration (DEA) controlled substance schedules for classifying drugs. The federal Controlled Substances Act (CSA) defines regulations for the manufacture, importing, possession, use and distribution of different drugs. In some cases, the major difference in classifying a substance as needing a prescription versus being accessible as an



OTC medication lies in the concentration of active ingredients the product contains. Thus, when taken as directed, there is little risk involved. That does not mean there is NO risk, only that the risk level is in a tolerable range for the general population. Risk levels, however, are considerably more difficult to judge for:

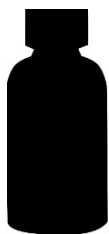
- Children
- Adolescents
- Individuals with certain types of physical or mental conditions (including pregnancy)
- Individuals with a pre-existing substance use disorder.

## What Is OTC Misuse About?

OTC abuse is not as large on the public radar as the topics of prescription misuse and misuse of illicit drugs or alcohol. But OTC products can be just as problematic when misused. This means use beyond or outside of the recommended dosing and/or to experience psychoactive effects with the product alone or mixed with other products (NIDA, 2017b). There are three types of OTC products commonly misused for psychoactive purposes for us to consider in greater detail; many other OTC products are misused for other physical or mental health effects.

**Decongestants.** Until relatively recently, pseudoephedrine (e.g., Sudafed®) was easily purchased as an OTC for managing cold, flu, and allergy symptoms of nasal congestion. Since 2005, it has become more tightly controlled. Although these medications are still available without a prescription, they are no longer fully OTC products in the United States. Their status is as a **behind-the-counter (BTC) medication** in some countries. BTC policy (in the U.S.) means that a person can purchase the substance without a prescription, but only through interacting with a pharmacist and only in small, designated amounts. The reason: pseudoephedrine

can be used as an ingredient in the illegal manufacturing of methamphetamine. (We learned about this in a previous module). However, medications containing pseudoephedrine may be abused on their own for other purposes: to promote weight loss or as a stimulant performance enhancer by athletes.



**Cough Medicines.** Dextromethorphan (DXM) is an ingredient commonly found in many OTC products intended as a cough suppressant. At recommended doses, DXM works on the part of the brain region that controls coughing. However, at extremely high doses (10-50 times the recommended), it becomes a psychotropic drug causing euphoria (sometimes referred to as “robotripping” or “skittling”—see NIDA, 2017b). The effect is dissociative and/or hallucinogenic, like with PCP or ketamine, and also a depressant effect (NIDA, 2017b). (We learned about this in a previous module.) Thus, it may not be alcohol content in cough medicine that leads someone to abuse these products (many forms are alcohol free, including tablets and capsules); it may be about the DXM. These medications are often misused in combination with other substances, like alcohol or cannabis (NIDA, 2017b). DXM misuse is largely a young person’s behavior. One reason is that DXM is legal, easily accessible, and relatively inexpensive. Policies have been enacted in many areas requiring purchasers to provide proof that they are over 18 years of age in an effort to curtail adolescent misuse of DXM. Another reason that adolescents might engage in DXM misuse, rather than some other substances, is that it may be easier to hide from parents who are unaware that it represents a form of substance misuse.



One hazard related to DXM misuse is the potential for acquiring the drug in a highly concentrated form meant for pharmacies to use; this “raw” or “pure” form may easily be taken in much higher doses than intended and usually comes from outside of the U.S. (thus, may not be quality

controlled). The risks of DXM misuse include impaired judgment and mental function (thus, impaired driving and risky decisions about engaging in other high risk behaviors), irregular/rapid heart rate, increased blood pressure (increasing the risk of stroke), vomiting (with the risk of aspiration/choking), and coma or death from overdose.

Another hazard lies in using DXM along with other substances. and many formulations that contain DXM also contain other medications, too. For example, OTC cold/flu medications often contain acetaminophen, which can cause liver damage, heart attack, or stroke in overdose amounts. These formulations also may contain antihistamines and other substances intended to relieve cold/flu symptoms and that are dangerous at high doses. If a person is taking enough of the combination medications to “get high,” there may be enough of these other substances to cause irreversible or deadly damage.

Moving beyond the subject of OTC cough medications, many prescription cough medicines include codeine. (We learned about codeine in the module focusing on opioids.) These prescription cough medications may be abused by individuals because codeine shares the same neurotransmitter receptor sites as opioids and heroin. These are potentially addictive medications because of their impact on the increased dopamine released in the brain’s reward system, hence they are Scheduled drugs requiring a prescription for legal distribution.

**Weight Loss Aids.** You learned about a variety of types of stimulant substances in a previous module, including amphetamines, methamphetamine, cocaine, nicotine, and caffeine. One reason for misuse of stimulant drugs relates to their tendency to suppress appetite, possibly contributing to weight loss. In the past, individuals may have been prescribed stimulant drugs to achieve a weight loss goal. However, this practice has diminished markedly as a result of growing awareness among health care providers of the high addictive potential associated with many stimulant prescription drugs.



There exists a wide range of OTC stimulant products on the market, many with questionable levels of risk and benefit. Until recently banned, OTC products sold in the United States might have included ingredients like ephedrine, pseudoephedrine, ephedra, or phenylpropanolamine. Like other stimulants, these ingredients and others, like bitter orange and ma huang (acting like ephedra), can cause nervousness/anxiety, tremor, rapid/irregular heart rate, increased blood pressure, and stroke, as well as being potentially addictive (Cohen, 2013).

Relatively recently, a new approach to serious weight loss medication has emerged: prescription medications that influence the brain chemistry (serotonin and norepinephrine) of appetite and craving (thus helping reduce caloric intake) without the stimulant effects on heart rate and blood pressure. They largely operate on the serotonin neurotransmitter systems of the brain. These are prescription medications because of their potential risks. Another prescription medication approach works instead on the gastrointestinal (GI) system to reduce fat absorption (thus, calories) from foods consumed.

**Anti-diarrheal medication.** An OTC opioid medication, loperamide (e.g., Imodium®), has the effect of slowing down the lower GI track, like other opioids. It does not, however, have psychoactive effects at recommended doses since it is designed not to enter the brain (NIDA, 2017b). However, consuming it in large quantity and/or combining it with other substances may cause psychoactive effects experienced with other opioids (NIDA, 2017b); combined with alcohol, the effect of both is amplified (<https://www.addictioncenter.com/drugs/over-the-counter-drugs/loperamide-addiction-abuse/>). It is unclear whether misuse of loperamide is common (NIDA, 2017b), but *may* have increased over the past decade in conjunction with the nation’s “opioid epidemic” (<https://www.addictioncenter.com/drugs/over-the-counter-drugs/loperamide-addiction-abuse/>).



## What Is Prescription Drug Misuse About?

In earlier modules you learned a bit about the problem of prescription drug misuse—for example, when we learned about opioids, CNS depressants (like benzodiazepine), amphetamines (like Ritalin® and Adderall®), and others. As cannabis becomes increasingly accepted for medicinal use, it too may become a subject of prescription misuse. The National Institute on Drug Abuse (NIDA, 2018b) defines prescription drugs as:

“...taking someone else’s prescription, even if for a legitimate medical complaint such as pain; or taking a medication to feel euphoria (i.e., to get high). The term *nonmedical use* of prescription drugs also refers to these categories of misuse.”



It is difficult to determine the extent to which prescription drug misuse involves engaging in ***polydrug misuse***, either concurrently or sequentially. In other words, an individual may not stick to just one type of substance and may either

combine them or move from one to another. As we have discussed elsewhere in this course, the safety of combining prescription, OTC, and/or illicit substances is questionable and depends on the type of substances being combined—they may potentiate (heighten or enhance) each other's effects and side-effects which increases overdose risks, or one may block the effectiveness of another leading either to a loss of therapeutic advantage of a medically prescribed treatment or a person taking higher doses than safe in order to achieve a desired effect.

“Multiple studies have revealed associations between prescription drug misuse and higher rates of cigarette smoking; heavy episodic drinking; and marijuana, cocaine, and other illicit drug use among U.S. adolescents, young adults, and college students” (NIDA, 2018b).

Data from the 2018 NSDUH survey (SAMHSA, 2019) indicate the following regarding past-month prescription drug misuse among individuals aged 12 and older:

- Over 5.4 million (2.0%) are estimated to have engaged in misuse of psychotherapeutic drugs;
- Over 2.8 million (1.0%) engaged in misuse of pain relievers
- Almost 1.7 million (0.6%) engaged in misuse of stimulant drugs
- Almost 1.8 million (0.7%) engaged in misuse of tranquilizers or sedatives (including benzodiazepines).





The demographic breakdown of survey participants indicates that:

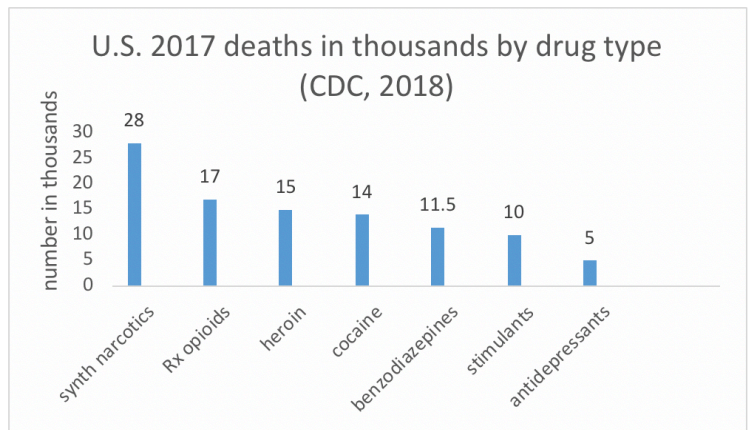
- Emerging adults aged 18-25 reported past month pain reliever misuse at higher rates (1.4%) than adults aged 26 and older (1.0%) or adolescents aged 12-17 years (0.6%);
- Emerging adults aged 18-25 reported past month (prescription) stimulant misuse at higher rates (1.7%) than adults aged 26 and older (0.4%) or adolescents aged 12-17 years (0.5%);
- Emerging adults aged 18-25 reported past month tranquilizer or sedative misuse at higher rates (1.2%) than adults aged 26 and older (0.6%) or adolescents aged 12-17 years (0.4%);
- Past month misuse of pain relievers was almost equally divided between men (1.1%) and women (1.0%);
- Misuse of (prescription) stimulants during the past month was somewhat more often reported by men (0.7%) than women (0.5%);
- Tranquilizer or sedative misuse during the past month was reported somewhat more often by men (2.5%) than women (2.2%);
- Misuse of pain relievers during the past month was most

commonly reported by individuals self-identifying as being of two or more races (1.4%) or American Indian/Alaska Native (1.4%), slightly less frequently by White (1.1%), Native Hawaiian/Other Pacific Islander (1.1%), Black and African American (1.0%), or Hispanic/Latino (0.9%) individuals, and least often by Asian (0.3%) individuals;

- Past month misuse of (prescription) stimulants was most commonly reported by individuals of two or more races (1.0%), and Asian (0.7%) or White (0.7%) individuals, and less often by individuals self-identifying as Black or African American (0.3%), American Indian/Alaska Native (0.3%), or Hispanic/Latino (0.3%)—no data were provided for Native Hawaiian/Other Pacific Islander individuals;
- Tranquilizer or sedative misuse during the past month was more often reported by individuals of two or more races (3.0%) and those self-identifying as American Indian/Alaska Native (3.0%), White (2.7%), Hispanic/Latino (2.3%), or Native Hawaiian/Other Pacific Islander (2.1%) compared to Black or African American (1.1%) and Asian (0.7%) individuals.



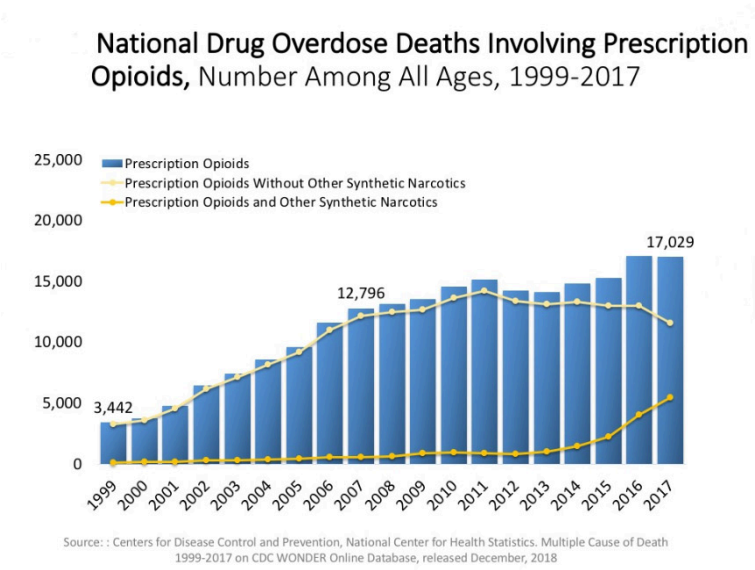
The Centers for Disease Control and Prevention (CDC) produced a report on deaths attributed to drug overdose in the U.S. during 2017 (CDC, 2018). While a great deal of public and policy attention has been directed to the 28,466 deaths reported from fentanyl and other synthetic narcotics (not including methadone), prescription opioids contributed to 17,029 deaths. This is compared to 15,482 deaths from heroin overdose or 13,942 from cocaine. Among the CNS depressants we have studied, benzodiazepines were responsible for 11,537 deaths. On the other hand, 5,269 deaths were from antidepressant overdose and 10,333 from psychostimulants (including methamphetamine). (Not depicted in this chart, note that over 33,000 deaths in 2015 were attributed to alcohol misuse; Lopez, 2016).



*Click to download data from this chart*

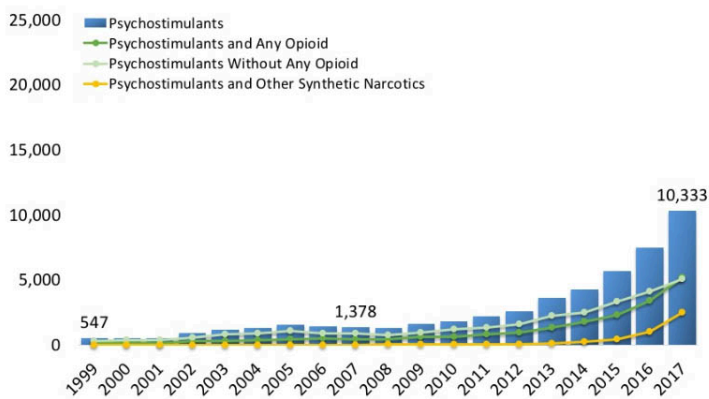
The next chart, produced from a 2018 CDC Wonder report and posted as a series of slides by NIDA (<https://www.drugabuse.gov/related-topics/trends-statistics/overdose-death-rates>), shows the rate of drug overdose deaths related specifically to prescription opioids from 1999 to 2017. Not only is the change in height of the

bars (numbers of deaths in the thousands) showing a dramatic increase, the yellow line across the top dipping down since 2011 and the orange line curving upwards since 2014 show that these prescription opioid deaths increasingly often include other synthetic narcotics (like fentanyl).



The trend in overdose deaths due to psychostimulants increased even more rapidly over the same time period (including methamphetamine deaths, not only prescription stimulants). The blue bars show the dramatic climb in deaths; the pale green line shows deaths due to psychostimulants alone, and the other two lines show the climb in deaths involving opioids or synthetic narcotics (like fentanyl) in combination with the stimulants.

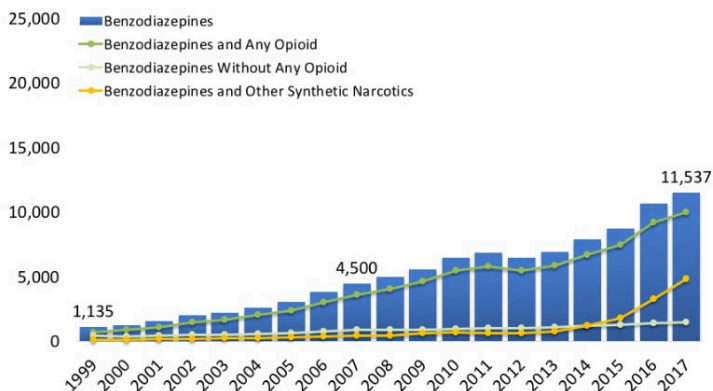
## National Drug Overdose Deaths Involving Psychostimulants With Abuse Potential (Including Methamphetamine), by Opioid Involvement Number Among All Ages, 1999-2017



Source: : Centers for Disease Control and Prevention, National Center for Health Statistics. Multiple Cause of Death 1999-2017 on CDC WONDER Online Database, released December, 2018

Not to be ignored, as the rates do surpass the stimulant-related deaths, are deaths from benzodiazepine overdose. This graph and the corresponding lines lead to parallel conclusions regarding benzodiazepine deaths over time, including those involving only benzodiazepines and those involving opioids or other synthetic narcotics.

## National Drug Overdose Deaths Involving Benzodiazepines, by Opioid Involvement, Number Among All Ages, 1999-2017



Source: : Centers for Disease Control and Prevention, National Center for Health Statistics. Multiple Cause of Death 1999-2017 on CDC WONDER Online Database, released December, 2018

The data do not distinguish between accidental overdose death and suicide by overdose. A review of literature led to the conclusions that: (1) benzodiazepines promote both aggression and impulsivity among individuals who use or misuse them, (2) impulsivity and aggression are both mediators of suicide risk, and (3) there exists a distinct positive correlation, and probably a causal relationship, between prescribed benzodiazepine use and suicide risk (Dodds, 2017). The concern appeared both during the period in which these drugs were used and the period of withdrawal from benzodiazepine use.

## Addressing Prescription Drug Misuse

The United States Department of Health and Human Services (HHS)

produced a report in 2013 containing a list of recommendations for addressing the massive problem of prescription abuse in the United States. Here is a copy of their summarized findings (CDC, 2013):

*As described in this report, current HHS prescription drug abuse activities fall within the following eight domains: 1) surveillance, 2) drug abuse prevention, 3) patient and public education, 4) provider education, 5) clinical practice tools, 6) regulatory and oversight activities, 7) drug abuse treatment, and 8) overdose prevention. Each of these areas contributes to ensuring the safe use of prescription drugs and the treatment of prescription drug dependence. Although significant efforts are already underway, a review of current activities along with a review of the prescription drug abuse literature, identified opportunities to enhance policy and programmatic efforts as well as future research are presented. Below are the overarching opportunities to enhance current activities identified in this report.*

- Strengthen surveillance systems and capacity
- Build the evidence-base for prescription drug abuse prevention programs
- Enhance coordination of patient, public, and provider education programs among federal agencies
- Further develop targeted patient, public, and provider education programs
- Support efforts to increase provider use of prescription drug monitoring programs (PDMPs)
- Leverage health information technology to improve clinical care and reduce abuse
- Synthesize pain management guideline recommendations and incorporate into clinical decision support tools
- Collaborate with insurers and pharmacy benefit managers to implement robust claims review programs

- Collaborate with insurers, and pharmacy benefit managers to identify and implement programs that improve oversight of high-risk prescribing.
- Improve analytic tools for regulatory and oversight purposes
- Continue efforts to integrate drug abuse treatment and primary care
- Expand efforts to increase access to medication-assisted treatment
- Expand Screening, Brief Intervention, and Referral to Treatment services
- Prevent opioid overdose through new formulations of naloxone

*Described more fully in Section III of the report, the opportunities listed above serve to strengthen programs and policies to reduce prescription drug abuse and overdose in the U.S. HHS has been at the forefront of the response to this serious public health issue and is committed to working with our federal, state, local governmental and non-governmental partners to further the actions included in this report.*

You can see from this report that no single, simple solution or strategy will solve the problem of prescription drug misuse. The complex nature of the issues involved dictates applying complex, integrated strategies.





Take a few minutes to conduct a complete inventory of your household:

- What OTC substances are present and what is their potential for misuse by household members or visitors? How might you best secure any that might be problematic? (While you're at it, check for expiration dates and learn how to dispose of them properly using online resources for your community.)
- What prescription substances are present and what is their potential for misuse by household members or visitors? How might you best secure any that might be problematic? (While you're at it, check for expiration dates and learn how to dispose of them properly using online resources for your community.)
- What potential inhalants are present and what is their potential for misuse by household members or visitors? How might you best secure any that might be problematic? (While you're at it, check for expiration dates and learn how to dispose of them properly using online resources for your community.)

# Ch. 3: Focus on Pharmacotherapy

This chapter explores the potential for medications to assist in the treatment of substance misuse and substance use disorders. Two general terms or labels are applied to this kind of intervention strategy: **pharmacotherapy** and **medication assisted treatment (MAT)**. Four basic purposes underlie pharmacotherapy and MAT interventions for substance misuse and substance used disorder:

1. Manage **detoxification (detox)** and **stabilization** processes as the first step in recovery;
2. Reduce substance cravings to prevent relapse and support recovery;
3. Manage short- and long-term withdrawal symptoms to prevent relapse and support recovery;
4. Help improve physical and/or mental health status related to co-occurring conditions.

The first step emphasizes that any necessary detoxification and stabilization be established before longer-term recovery support or treatment is delivered (CSAT, 2006).

As you read this chapter, consider several things:

- First, on the surface, it might seem a bit paradoxical to treat a drug problem with drugs. The pharmacotherapy strategy is not without controversy. Underlying some programs is a philosophy encouraging total abstinence from all types of psychoactive substances, including relying on the medications prescribed in pharmacotherapy—not substituting one drug for another, but eliminating the need for any drugs. However, there exists a sizable (and growing) literature supporting the use of certain medications to facilitate treatment of substance

use disorders.

*“Pharmacotherapies, or prescribed use of medications, have increasingly become an important component in managing the care of individuals experiencing substance use problems” (Zweben & West, 2020, p. 310).*

The underlying assumption is that substance use disorders and addiction are diseases of the brain and brain chemistry, therefore addressing those brain chemistry mechanisms is a reasonable approach to treatment. Furthermore, pharmacotherapy is intended as a means of supporting the transition from substance misuse to substance abstinence through medication assistance the helps individuals overcome cravings and withdrawal (short- and long-term). Individuals who have been self-medicating physical and/or mental disorders can be supported with alternatives to the drugs that were causing so much harm in their lives.



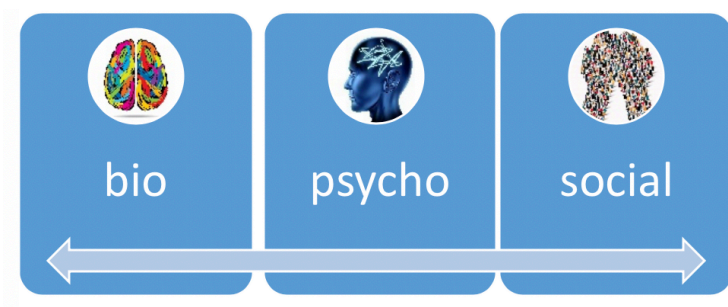
- Second, a preponderance of evidence supports providing psychological, social, and biological approaches in combination—medications alone are not sufficient but can make a difference in a person’s ability to stick with and benefit from behavioral and social interventions.

*“Pharmacotherapy should be accompanied by behavioral intervention to clarify and set expectations of the benefits, side effects management, and potential barriers to compliance with the medication protocol” (medication management counseling) and provide the kind of behavioral, social, and case management interventions necessary to support the recovery process (Zweben & West, 2020, p. 311).*

*“Lacking the requisite social support and social stability*

to sustain treatment gains increases the risk of treatment failure. Adequate exposure to a more intensive behavioral approach is often necessary to attend to treatment needs” (Zweben & West, 2020, p. 312).

“In summary, pharmacotherapy may help reduce withdrawal symptoms and craving during recovery, but behavioral interventions are needed to resolve accompanying or associated social, economic, interpersonal, and intrapersonal problems. Not attending to these issues can present a major impediment to recovery” (Zweben & West, 2020, p. 312).



This is the theme of the guidelines published by the Substance Abuse and Mental Health Services Administration (SAMHSA, 2016) for medication assisted treatment of opioid addiction. These guidelines indicate that the following steps are critical:

- ✓ Evaluate the need for medically managed withdrawal from opioids
- ✓ Addressing co-occurring problems (more about this our final course module)
- ✓ Integrate pharmacologic and nonpharmacologic therapies—medications are a part of a comprehensive, individualized treatment plan that includes counseling and other psychosocial therapies and mutual-help programs.

- Third, medications used to treat substance use disorders are not without side effects and risks themselves: some pharmacotherapy medications have addictive potential themselves (e.g., methadone). The hope is that their known risks can be managed more safely than in the uncontrolled world of illicit substance misuse. In other words, use of managed, monitored, medication assisted treatment is a form of harm reduction strategy.

In short, keep in mind that MAT or pharmacotherapy is a tool in the collection of options available for the treatment of substance use disorders. The science surrounding the development and testing of new medication applications changes rapidly, so new



medications may come into favor just as older and current medications may decline in favor as treatment approaches (Portelli, Munja, & Leggio, 2020). As we have learned throughout this course, no one-size-fits-all approach works for everyone. Like any treatment program, pharmacotherapy/MAT needs to be tailored to individuals and their circumstances, and often need to be modified as a person's circumstances change over time. Furthermore, many individuals engaged in polydrug use or experiencing co-occurring problems may need individualized medication regimens as different medications used in pharmacotherapy/MAT have differing effects, differing effectiveness for addressing misuse of different substances, and interact differently with conditions and other medications (Portelli, Munja, & Leggio, 2020).

*“Individuals experiencing more severe substance use problems typically require more intensive behavioral interventions in*

conjunction with an effective medication” (Zweben & West, 2020, p. 312).

“In contrast, for individuals experiencing less severe substance-related problems, a less intensive medical management (MM) approach, combined with an effective medication might suffice to produce positive change” (Zweben & West, 2020, p. 312).

### **Medication Assisted Detoxification/Stabilization and Management of Cravings and Withdrawal (Relapse Prevention)**

Detoxification (detox) is part of the continuum of care in addressing a person’s substance misuse or substance use disorder and may involve an interdisciplinary team. A great deal of managing neonatal withdrawal syndrome (NWS) involves detoxification and stabilization of the infant, as well as potentially addressing substance-related concerns with parent(s)—sometimes the mother’s use of opioids is medically managed throughout a pregnancy, it is not always a matter of substance misuse.

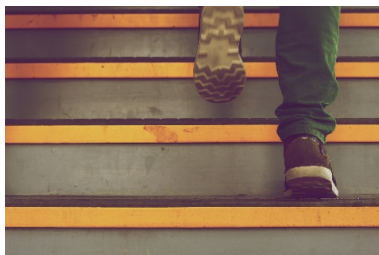
According to TIP #45 (CSAT, 2006):

*“Detoxification is a set of interventions aimed at managing acute intoxication and withdrawal. It denotes a clearing of toxins from the body of a patient who is acutely intoxicated and/or dependent on substances of abuse. Detoxification seeks to minimize the physical harm caused by the abuse of substances” (p. 4).*

The TIP #45 resource also explains that the detoxification process is comprised of 3 essential components:

- Evaluation (e.g., person’s physical and mental status, types and amounts of substances involved)
- Stabilization (establish safe physical and mental status)
- Fostering readiness for engaging in treatment.

Pharmacotherapy and MAT might assist in the detox and stabilization processes: “This often is done with the assistance of medications” (CSAT, 2006, p. 4). Medically supervised detoxification is an important means of managing



potentially life-threatening withdrawal and other physical (or suicidality) concerns that may arise during this early step in recovery. The length of time required to complete detoxification and stabilization varies as a function of the drugs and doses involved, as the **persistence** of different substances being metabolized varies markedly. Detox and stabilization are not considered a completed substance misuse treatment episode but an important component that also provides a person with:

“a point of first contact with the treatment system and the first step to recovery. Treatment/rehabilitation, on the other hand, involves a constellation of ongoing therapeutic services ultimately intended to promote recovery” (CSAT, 2006, p. 4).

A detox/stabilization program may consist of specific stages or phases with different aims at each point in the process (CSAT, 2006). The initial goal is to monitor any acute medical situation or crisis, ensuring safety as the misused substances leave the body (withdrawal). Administering medications to support the person medically could take place during this phase, but only if the stabilization team knows what substances were involved—a polydrug misuse crisis might leave the team unwilling to risk administering medications that might adversely interact with some of the potentially involved substances.



The second phase of stabilization involves a more extended detoxification treatment plan (measured in days) to manage the early withdrawal period and to support the person in obtaining ongoing treatment for their substance misuse/substance use disorder. This might involve a medication assisted treatment (MAT) plan. The third phase of a stabilization plan might continue for days to weeks with the goal of supporting the person in making a successful transition to long-term treatment, often involving counseling, supportive recovery services (e.g., sober housing and other case management services), and MAT.

Let's take a closer look at some of the current Food and Drug Administration (FDA) approved options used in detox and longer-term treatment interventions. First, a little reminder of some pharmacotherapy principles relevant to pharmacotherapy and MAT. The distinction between agonist and antagonist medications is relevant to pharmacotherapy intervention strategies. You may recall from earlier coursework that an **agonist** leads to activation or stimulation of neurons when it binds to the specific receptor sites, and an **antagonist** blocks (or dampens) the neurons' responses when it binds to the specific receptor sites (Portelli, Munja, & Leggio, 2020). This information helps determine which medications are most likely to produce the desired effect in treating misuse of a specific type of substance. Recall also that different drugs even within the same class have different rates at which they are absorbed and metabolized, and dosing is dependent on finding the medication's therapeutic zone without moving into an overdose level where side effects outweigh the benefits. Recall also that a person may develop physical dependence on some pharmacotherapy/MAT medications, just as they did to the misused substances, that tolerance to some pharmacotherapy agents may develop, and that withdrawal from some of these medications may be unpleasant. Critical in all of this discussion is **medication management (MM)**—ensuring that a person has access to the



medications prescribed, is able to and does use them as prescribed, and is able to tolerate the side effects (e.g., see Medication Management Support for Alcohol Dependence, <https://pubs.niaaa.nih.gov/publications/clinicianGuide/guide/tutorial/data/resources/MedMgmtSupportTemplates.pdf>).

### **Common Pharmacotherapy/MAT Agents**

Several pharmacotherapy and MAT agents common in the U.S. are described and discussed in terms of the types of substance misuse/use disorder they are intended to treat.

**Alcohol Use Disorder.** You may recall from our unit focused on alcohol that withdrawal from alcohol can be a complicated, and potentially deadly, process best managed with close medical supervision and management (CSAT, 2006). While the majority of individuals will not need medication to



manage the stabilization process following alcohol intoxication, medications might be helpful for the others (CSAT, 2006).

**Benzodiazepine** has a significant history as a first step in treating alcohol withdrawal (Zweben & West, 2020), but benzodiazepine treatment also introduces significant risks of its own (CSAT, 2006). Three medications used in longer-term treatment of alcohol misuse/use disorder and relapse prevention are not particularly addictive themselves and are otherwise reasonably safe to use: naltrexone, acamprosate, and disulfiram (Portelli, Munja, & Leggio, 2020). Naltrexone was approved by the FDA in 1994 for treatment of alcohol use disorder (Suh, Pettinati, Kampman, & O'Brien, 2006).

**Naltrexone** is an opioid receptor antagonist that decreases the positive reinforcing effects of alcohol use, thereby gradually reducing a person's craving for alcohol as the reward is not as strongly "paired" with the behavior (Suh et al., 2006). Adherence with naltrexone therapy is bolstered by using a long-acting, extended-release injectable form (e.g. Vivitrol®) instead of a daily oral dosing protocol (Portelli, Munjal, & Leggio, 2020).

**Acamprosate** became an FDA-approved alcohol use disorder

treatment medication in 2004 (Suh et al., 2006). It works at the neurotransmitter level, reducing the longer-term negative withdrawal effects of quitting alcohol use—effects associated with relapse, making it easier to stick with a recovery commitment. One of the benefits of acamprosate over some other medications: it is not metabolized by the liver which is important in persons whose liver may be compromised from chronic alcohol misuse or who might have hepatitis or other liver disease (Witkiewitz, Saville, & Hamreus, 2012). It is also a safely tolerated alternative for a person whose treatment goal is to reduce their drinking but not eliminate all alcohol use (Witkiewitz, Saville, & Hamreus, 2012).



In 1951, **disulfiram** (Antabuse®) was approved by the FDA for use in treating alcohol use disorder (Suh et al., 2006). This drug works differently from the previous two: it creates a set of acute physical discomforts when alcohol is consumed in its presence. In learning theory terms, drinking behavior is punished by the physical consequences experienced. It does this by inhibiting the enzyme (ALDH, aldehyde dehydrogenase) responsible for metabolizing the first-order alcohol metabolite, acetaldehyde, thus allowing the acetaldehyde to build up to levels where facial flushing, sweating, headache, nausea/vomiting, rapid and/or irregular heart rate occur soon after drinking alcohol (Suh et al., 2006). Pharmacotherapy with disulfiram for treating alcohol use disorder can be somewhat tricky and complicated. For example, the individual needs to be sufficiently motivated for change and have reliable access to the medication in order to take the medication as prescribed (**medication adherence**); skipping doses means they can drink again without the immediately punishing consequences. Dosing is also an issue, as the aim is to cause mild discomfort rather than significant symptoms when alcohol is consumed, but these consequences are alcohol dose dependent (Suh et al., 2006). The unpleasant reaction may even be triggered by exposure to alcohol in other forms, such as: alcohol-

based hand sanitizer, mouthwash, cleaner, or solvent; cooking wine or wine-based vinegar; flamed/flambé desserts using alcohol as the fuel; and more. Like any medication, it is not without side effects, but these are generally considered mild (Suh et al., 2006).

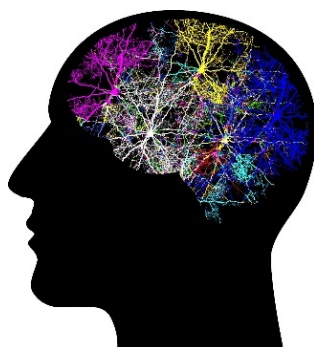
Other pharmacotherapy approaches to alcohol use disorder either in other countries or under study in the United States include:

- nalmefene, used in Europe for this purpose, has moderate evidentiary support;
- baclofen, which has mixed results in studies from several countries;
- varenicline, used to support smoking cessation, has some efficacy support especially for individuals experiencing both alcohol and nicotine use disorder;
- and others under early investigation (Portelli, Munjal, & Leggio, 2020).

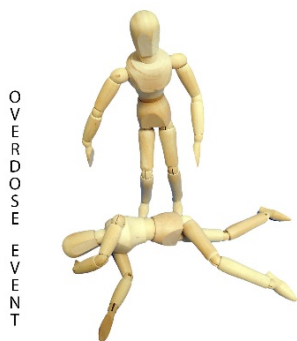
**Sedative-Hypnotic/CNS Depressant Use Disorder.** As in the case of alcohol withdrawal, the process can be complicated and potentially deadly, therefore the process may best be managed with close medical supervision and management (CSAT, 2006). The pharmacotherapy medication of choice has been benzodiazepine—a sedative-hypnotic CNS depressant we previously studied and discussed in relation to alcohol use disorder. This intervention approach is based on transitioning from the effects of one CNS depressant (e.g. alcohol or other CNS depressant) to another (benzodiazepine), then imposing a more controlled physical withdrawal process through gradual tapering of the second CNS depressant (the benzodiazepine; CSAT, 2006).

**Cannabis Use Disorder.** “There are currently no FDA-approved pharmacotherapy treatments for cannabis use disorders” (Portelli, Munjal, & Leggio, 2020, p. 336). Symptoms of cannabis withdrawal may be managed with medications.

**Cocaine Use Disorder.** Individuals helped with severe cocaine (or other stimulant) withdrawal may engage in less future cocaine use when their detox was supported with a medication called amantadine, but this medication did not improve outcomes for individuals with less severe withdrawal symptoms (CSAT, 2006). Disulfiram has been used to



address cocaine use disorder, as well as alcohol use disorder; this is relevant because the two problems often co-occur (Suh et al., 2006). The mechanism by which disulfiram is believed to have its effect on cocaine relapse is different than how it worked against alcohol misuse: it seems to act similarly to an agonist in the dopamine system of the brain (Suh et al., 2006). However, efficacy of disulfiram treatment for cocaine use disorder is debatable (Portelli, Munjal, & Leggio, 2020). Systematic review of literature concerning the use of antidepressant medications to treat primary cocaine use disorder concluded that they “appear to have no effect on cocaine use or treatment retention” (Chan et al., 2019). Bupropion, topiramate, and psychostimulants have some evidence of effectiveness on cocaine abstinence, and antipsychotic medications have moderate evidence of effectiveness in improving treatment retention (Chan et al., 2019).



**Opioid Use Disorder.** As in the case of alcohol and sedative hypnotic withdrawal, the process can be complicated and potentially deadly, therefore the process is best managed with close medical supervision and management (CSAT, 2006). Opioid receptor antagonists are important in the immediate

reversal of opioid overdose—**naloxone** (Narcan® or Evzio®) being the one with which the public is most familiar. As an antagonist, naloxone binds to opioid receptors and blocks the effects of other opioids—heroin, fentanyl, and opiate medications. It is either injected or sprayed into a nostril of a person undergoing (or suspected of) opioid overdose (NIDA, 2020). Naloxone delivery induces immediate opioid withdrawal, so the person will likely experience withdrawal symptoms—this consequence is offset by the lifesaving effects on suppressed breathing. More than one dose might be necessary, depending on the intensity and dose of the opioid that was used in the overdose situation (e.g., fentanyl overdose may require multiple administrations for reversal). In most communities, naloxone can be legally carried and administered by any community member or family member/friend of anyone at risk of opioid overdose; some states require physician prescription, while in other pharmacies can distribute naloxone delivery kits without a prescription. To learn about naloxone access laws in different states, see the Prescription Drug Abuse Policy System website (<http://www.pdaps.org/datasets/laws-regulating-administration-of-naloxone-1501695139>). In addition to use for opioid overdose reversal, naloxone appears in combination with other MAT medications in longer-term pharmacotherapy for opioid use disorder—these formulations allow the different medication combined to offset each other’s negative effects leading to greater recovery support potential (Portelli, Munjal, & Leggio, 2020).

**Naltrexone**, previously discussed in the pharmacotherapy for alcohol misuse/use disorder, is also used in MAT for opioid use disorder. Naltrexone is an opioid antagonist that blocks the euphoric and sedating effects of opioids, and reduces opioid craving (SAMHSA, 2019). If a person taking naltrexone



relapses to opioid use, the reinforcing effect of the substance misuse is blocked. One challenge with MAT involving naltrexone is that a person will need to be free of opioid use for at least 7-10 days before starting naltrexone therapy because the medication will induce opioid withdrawal symptoms (SAMHSA, 2019). Of concern is the high relapse rate occurring before this point of detox: "...abstaining for weeks or months before entry will fail most of the population, who relapse before that point" (Humphreys, Malenka, Knutson, & MacCoun, 2017). Another challenge arises if a person stops taking the medication: their previous opioid tolerance level may have dropped so resuming use of the same amount/dose of opioids previously misused may cause a dangerous overdose event (SAMHSA, 2019). An advantage of naltrexone therapy is that it is available in an extended-release monthly injectable form (Vivitrol®) which helps a person maintain medication compliance over time. Also, because the injections are delivered by health care providers, the individual's status can be monitored, and additional treatments/services can be offered as needed.

**Methadone** is a relatively long-acting opiate, synthetically produced, and the pharmacotherapy approach to opioid use disorder with the longest evidence-supported history (Portelli, Munjal, & Leggio, 2020). The pharmacological effects are similar to opioids commonly misused, allowing prescribers to help a person gradually withdraw and taper off of other opioids. It reduces the experience of withdrawal so that a person is less likely to relapse with the aim of avoiding these miserable symptoms. **Methadone maintenance therapy (MMT)**, the primary form of MAT for opioid use disorder, is delivered by specially licensed clinics and integrates methadone pharmacotherapy with behavioral and other intervention strategies with better outcomes than either methadone detoxification or non-methadone involved strategies alone (Mattick, Breen, Kimber, & Davoli, 2009; Portelli, Munjal, & Leggio, 2020). As long as a person continues to engage in MMT, their progress and health concerns can be monitored and potentially addressed on a regular basis by the clinic team. "In addition to

maintaining abstinence, methadone treatment has been shown to reduce overdose death risk, increase treatment retention, and reduce use of illicit drugs” compared to placebo medication (Portelli, Munjal, & Leggio (2020). Tight control over methadone therapy is required because of its high potential for abuse—it is an addictive substance itself, listed as a Schedule II substance. Unfortunately, the necessity for such tight control reduces ease-of-access to this type of treatment.



In addition to naltrexone and methadone, the FDA has approved **buprenorphine** for the treatment of opioid use disorder. Buprenorphine (Subutex®), nicknamed bupe, is a long-acting (partial) agonist acting on opioid receptor sites—it does not produce the same euphoric or dangerous effects as commonly misused opioids (NIDA, n.d.). Buprenorphine may also be prescribed to manage chronic pain (Portelli, Munjal, & Leggio, 2020). **Suboxone®** is a medication combining buprenorphine and naloxone which reduces “diversion” for misuse of the buprenorphine alone (NIDA, n.d.). Buprenorphine has a lower risk of substance misuse compared to methadone which has allowed a larger range of qualified health care providers to legally prescribe this medication (since the Drug Addiction Treatment Act of 2000). However, requiring specialized training to become a qualified buprenorphine prescriber does

impose a barrier to wider use (Portelli, Munjal, & Leggio, 2020). An opioid use abstinence period of 12-24 hours is recommended prior to beginning buprenorphine treatment (Portelli, Munjal, & Leggio, 2020).



**Nicotine Addiction.** Tobacco use disorder has greater chances for successful treatment when pharmacotherapy and behavioral therapies are delivered in combination (Portelli, Munjal, & Leggio, 2020). Among the pharmacotherapy strategies are **nicotine replacement**

**therapy (NRT)** and medications that influence neurotransmitter systems. NRT options include various forms of administering controlled doses of nicotine (the most addictive of the chemicals in tobacco use: transdermal (skin) patches, gum, lozenges, oral/nasal spray, or inhaler (e.g, vaping). “There exists robust evidence of the efficacy of NRTs in aiding smoking cessation” (Portelli, Munjal, & Leggio, 2020, p. 327). **Bupropion** (e.g., Zyban®) acts on nicotine withdrawal effects, making it easier to avoid relapse. **Varenicline** (e.g. Chantix®) acts by mildly stimulating the same receptor sites activated by nicotine and, at the same time, blocks nicotine’s dopamine releasing capabilities—in other words, it is both an agonist and antagonist medication. Thus, it provides some of the reinforcement previously gained with smoking cigarettes and decreases the reinforcement received from smoking again. Evidence supporting e-cigarettes/vaping as an NRT is mixed: evidence suggests that individuals can effectively use this method to taper off of cigarette smoking, however it is all too common that they simply replace smoking cigarettes with continued e-cigarette use and fail to taper off the nicotine and remain addicted (Portelli,



Munjal, & Leggio, 2020). As previously discussed, there exist significant health and safety concerns related to e-cigarette use.



An interactive H5P element has been excluded from this version of the text. You can view it online here:

<https://ohiostate.pressbooks.pub/swk5805coursebook/?p=910#h5p-54>

## Ch. 4: Summary

This module opened with an examination of issues related to inhalant misuse—what kinds of substances are commonly involved and who tends to engage in this type of substance misuse. The practice was identified as being particularly risky, not as much because of addiction concerns as because of the heavy toll it can take on the brain and other critical organ systems in the body. Then we looked at concerns related to anabolic steroid misuse. The next topics examined in this chapter were over-the-counter (OTC) and prescription drug misuse. The great fallacy of thinking that drugs sold as OTC (or behind-the-counter, BTC) substances are safer than prescription or “street” drugs was addressed, and you learned about some of the most commonly misused OTC products. Prescription drug misuse has appeared in several modules in our course as we looked into specific types of drugs like amphetamines, sedative-hypnotic/CNS depressants, opioids, and cannabis. In this module we looked a bit more deeply into the epidemiology of prescription drug misuse and some policy response ideas. In our final chapter, we (re)visited the topic of pharmacotherapy/medication assisted treatment for alcohol and other substance misuse/use disorders. The topic was discussed to some extent in our prior modules on alcohol and opioids; here we delved more deeply into the topic. In particular, we explored the interdisciplinary detoxification (detox) and stabilization process, and then the subsequent treatment uses of different pharmacotherapy approaches to treatment of substance misuse and substance use disorders. The science in this area continues to develop and new pharmacotherapy strategies are emerging. The evidence does support the combined use of behavioral and case management interventions with pharmacotherapy: “Treating addiction more commonly requires longer-term intervention, such as Alcoholics Anonymous, methadone-buprenorphine maintenance, ‘sober living’ residential

facilities, and extended case monitoring” (Humphreys et al., 2017). At this point, you are prepared to review some key terms used in this module and proceed to our final course topic.

# Module 12: Key Terms

**acamprosate:** a medication used to reduce the desire to drink alcohol by adjusting neurotransmitter balance.

**agonist:** a drug that partially or fully activates specific neurotransmitter receptors, creating a partial or full response that would be triggered by another drug (e.g., illicit or misused substances); used as a substitute for the problematic substances.

**anabolic (androgenic) steroids:** synthetically produced testosterone, potentially misused.

**antagonist:** a drug that blocks another substance's action by binding to the neurotransmitter sites and preventing its action.

**behind-the-counter (BTC) medication:** a level of control imposed on over-the-counter medications that limits amounts obtained and/or records user identification information to reduce potential for misuse; does not involve a prescription control but requires involvement of a licensed distributor (e.g., pharmacist).

**benzodiazepines:** synthetically produced drugs with a tranquilizing effect on the brain, commonly prescribed to treat anxiety, sleep disorders, and alcohol withdrawal; potentially addictive, and may be misused themselves.

**buprenorphine:** a prescribed opioid medication (narcotic) used to treat opioid use disorder; may be combined with naloxone (e.g., Suboxone®). [not to be confused with bupropion, see below]

**bupropion:** an antidepressant medication that also may be used to treat nicotine dependence by reducing cravings and withdrawal effects. [not to be confused with buprenorphine, see above]

**detoxification (detox):** an initial step in treating substance misuse/substance use disorders during which the substances of concern are withdrawn from the body under supervision, the person is medically stabilized, withdrawal symptoms are managed, and longer-term treatment is encouraged.

**disulfiram:** an alcohol antagonist drug that produces unpleasant

physical reaction to alcohol consumption/exposure; serves as a deterrent to drinking (avoiding the punishing consequences); may also be used in pharmacotherapy with cocaine misuse where it likely serves as a cocaine agonist in the dopamine reward system instead.

**inhalants:** volatile substances (gas, aerosol, or vapor) misused by inhalation in high concentrations.

**medication adherence:** the extent to which an individual uses medication as prescribed (adheres to a treatment plan involving medication).

**medication assisted treatment (MAT):** treatment of specific substance misuse/substance use disorder with specific medications; the term often refers to the combination of medication and behavioral intervention approaches, but may refer only to the medication aspect of intervention.

**medication management (MM):** a specific type of intervention designed to support adherence to a medication-involved intervention protocol.

**methadone:** a synthetic opioid agonist drug used to treat opioid use disorder by reducing cravings and withdrawal symptoms, as well as blocking the effects of other opioids that might be used.

**methadone maintenance therapy (MMT):** a coordinated, integrated intervention strategy for treating opioid use disorder that includes concurrent delivery and monitoring of methadone pharmacotherapy with behavioral/rehabilitation interventions.

**naloxone:** an opioid antagonist used for rapid reversal of an opioid overdose event.

**naltrexone:** an opioid antagonist used for treatment of opioid or alcohol misuse/use disorder. [Not to be confused with naloxone, see above]

**nicotine replacement therapy (NRT):** medications or devices that deliver controlled amounts of nicotine that can be gradually tapered to help a person stop using nicotine products (e.g., cigarettes) by minimizing the cravings and withdrawal symptoms associated with cessation efforts; considered a harm reduction

approach if the medication or device eliminates the risks associated with smoking or otherwise consuming the nicotine-containing products.

**over-the-counter (OTC) substance misuse:** intentional misuse of medication/medical products sold without a prescription for the purpose of experiencing their psychoactive effects.

**persistence:** how long a substance remains active in the body; related to the pharmacokinetic principle of drug half-life.

**pharmacotherapy:** use of (prescribed) medications, in this context, for the purpose of treating substance misuse/substance use disorder.

**polydrug misuse:** using two or more psychoactive substances in combination, usually with the intent of achieving a particular effect; alcohol is commonly involved in polydrug use scenarios.

**prescription misuse:** the use of a controlled substance (medication) without a prescription, in a manner other than was prescribed, or for the purpose of altering feelings/experience.

**stabilization:** one major goal of the detoxification (detox) process aimed at ensuring a person is medically and mentally stable without additional use of previously misused substances.

**Suboxone®:** a medication combining buprenorphine and naloxone, used in treating opioid misuse/use disorder.

**varenicline:** a partial nicotine agonist medication used in treating nicotine addiction.

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## PART XIII

# MODULE 13: FOCUS ON CO-OCCURRING PROBLEMS & COURSE CONCLUSIONS

The Module 13 readings introduce concepts essential for understanding problems that commonly co-occur with substance use, misuse, and use disorders, as well as concluding the course with a summary and final points concerning substance misuse. Sources discussing co-occurring problems typically emphasize mental disorders and sometimes physical health concerns; however, many other types of challenging situations and circumstances may co-occur with substance misuse, as well. The frequently used term **dual diagnosis** is somewhat limiting as it refers specifically to diagnosable mental conditions and to only a pair of them co-occurring; considerable numbers of individuals experience more than two co-occurring conditions and/or non-diagnosable challenges (Reedy, 2020). Another term, **comorbidity**, again refers specifically to diagnosable conditions that occur either simultaneously or sequentially (NIDA, 2010). In this module we apply a social work lens in examining a broader, more inclusive host of potentially co-existing or **co-occurring problems**—including but not limited to diagnosable conditions. Challenging situations and conditions may co-occur in complex clusters, with one co-occurring problem impacting another, and co-occurring problems can impede recovery and treatment efforts, as well. The first chapter examines diagnosable physical and mental/behavioral health issues that commonly co-occur with substance misuse. The second addresses a host of additional (non-diagnosable) co-

occurring challenges. The module concludes with a final chapter providing an overview and wrap-up of the entire course.

## Reading Objectives

After engaging with these reading materials and learning resources, you should be able to:

- Explain the relationships between substance misuse/ substance use disorders and several commonly co-occurring physical health, mental/behavioral health, and social circumstance challenges;
- Identify epidemiological trends in their co-occurrence and how this might impact intervention strategies;
- Define several key terms related to problems that tend to co-occur with substance use, substance misuse, and substance use disorders;
- Summarize what was covered in this course and what next steps in further education you might consider.

# Ch. 1: Problems That Commonly Co-occur With Substance Misuse

First, we explore mental/behavioral health challenges that commonly co-occur with substance misuse. The list about which there is available evidence includes:

- mood disorders (e.g., depression, anxiety/panic)
- thought disorders (e.g. schizophrenia and dementia)
- personality disorders
- attention deficit disorder (ADD) with and without hyperactivity (ADHD)
- post-traumatic stress disorder (PTSD)
- risk of suicide
- multiple types of substance use disorder or addictive behaviors (e.g. gambling disorder).

Second, we explore physical health and disability conditions that commonly co-occur with substance misuse, including:

- infectious disease exposure and progression of disease
- non-infectious diseases
- traumatic brain injury
- accidental injury.

As we progress through this module, consider what is meant by co-occurring or **concomitant** problems. These are issues, concerns, or problems that occur together, are associated with one another, and/or coincide closely in time or over the course of time. When two or more problems coincide like this (they co-occur or are concomitants), several possible models explain the possible

relationship between them. Let's consider examples where Problem A is a form of mental disorder and problem B is an alcohol use disorder.



Quite possibly, Problem A causes Problem B, or at least increases the probability that Problem B will arise. For

example, mental disorders can contribute to alcohol (or other substance) misuse and use disorder (NIDA, 2018).

Or, the opposite may be true: Problem B may cause Problem A. For example, alcohol misuse (or other form of substance



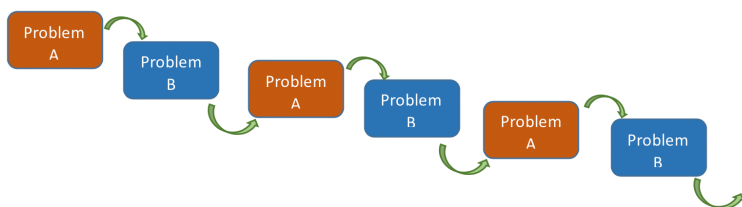
misuse) can contribute to the development or expression of mental disorders (NIDA, 2018).



Yet, another possibility is that some third factor influences the emergence of both

problems A and B—Problem A does not cause Problem B, nor does Problem B cause Problem A. However, each may be caused or influenced by the same third factor. For example, both alcohol misuse and a mental disorder may be influenced by a common genetic thread or from experiencing violence (e.g. intimate partner violence, sexual assault, child maltreatment, or military combat). Conceptually, this situation exists when both problems (A and B) have shared, common risk or vulnerability factors contributing to their development or expression (NIDA, 2018).

The relationship between problems can be even more complex, including that they are iteratively interacting over time. In other words, Problem A might influence Problem B, which then causes a change (worsening) in Problem A, which then changes Problem B, and so forth over time.



**Why does it matter?** Co-occurring disorders and substance misuse/substance use disorders present exceptional life and intervention challenges. “Co-occurring substance use and mental health disorders often



present with more severe mental health problems, earlier relapse, greater difficulty maintaining abstinence, more mental health hospitalizations, and greater risk for suicide attempts,” as well as more symptoms of each disorder (Reedy, 2020, p. 529). Having a substance use disorder may mean a person experiences extreme stress and stressor events associated with living “an addicted” life, circumstances that might include homelessness, exposure to violence, and social isolation from friends and family (van Wormer & Davis, 2013, p. 462). The situation is somewhat reminiscent of *The Sorcerer’s Apprentice*: things were manageable with one or even two brooms but became increasingly challenging as the brooms multiplied.

Unfortunately, individuals with more complex arrays of challenges may be systematically excluded from treatment programs and from research concerning intervention efficacy and effectiveness—these individuals may not meet inclusion criteria by virtue of their exceptional circumstances. Or, one/some of their problems may be masked by the most obvious disorder, rendering

appropriate diagnosis and treatment planning difficult. These complexity issues help explain why evidence-based substance-related interventions may fail at higher rates with individuals experiencing co-occurring problems. Let's look at these commonly co-occurring conditions.

## Mental Disorders and Mental Health Challenges

*Mental illness is terrifying when it spirals out of control. Repeated alcohol or drug misuse often brings misery, regret, and dire consequences. When the two are combined, the result can be a double whammy of troubles that worsens each condition*(van Wormer & Davis, 2013, p. 452).

Based on 2018 National Survey on Drug Use and Health (SAMHSA, 2019a), over 9 million adults aged 18 and older (3.7%) in the U.S. were estimated to have experienced past year co-occurring substance use and mental disorders; for over 3 million (1.3%), their mental disorders were categorized as serious.

The National Co-Morbidity Survey conducted between 2001-2003 involving over 9,000 adults offers some insight into the frequency of co-occurring mental disorders across the U.S., with substance use disorder considered as one form of mental disorder (Kessler et al., 2005). In this report, diagnosable mental conditions were considered co-occurring if they were present during the same 12-month period. Across the sample, 26.2% of participants experienced one or more diagnosable mental disorder. Most often, individuals with a diagnosable mental disorder had only one type (55%), but a considerable number experienced two or more diagnosable mental disorders (45%). The investigators concluded that: "Although mental disorders are widespread, serious cases are concentrated among a relatively small proportion of cases with high comorbidity" (Kessler et al., 2005, p. 617).



1 single diagnosis	2 diagnoses	3 or more diagnoses
55%	22%	23%

The types of mental disorders that most commonly co-occur with substance misuse and substance use disorder include mood disorders (anxiety, depression, and bipolar disorders), thought disorders (schizophrenia and dementia), personality disorders (including antisocial and borderline personality disorders), impulse control disorders (ADD/ADHD and bipolar disorders), post-traumatic stress disorder (PTSD), and gambling disorder.

**Anxiety** . Numerous studies have demonstrated that: “Individuals who are more sensitive to symptoms of anxiety may also be more likely to use substances,” particularly alcohol and sedative substances (Reedy, 2020, p. 531). The co-occurrence of anxiety and substance use disorders is “associated with greater symptom severity, higher levels of disability, and poorer course of illness relative to either disorder alone” (McHugh, 2015, p. 99). According to analysis of the National Epidemiologic Survey on Alcohol and Related Conditions III (NESARC) data, alcohol use disorder was modestly associated with panic disorder, phobias, and generalized anxiety disorder, regardless of the level of alcohol use disorder severity (Grant et al., 2015). Concluded from prior NESARC data was that, among individuals experiencing an anxiety disorder, almost 15% experienced a substance use disorder during the past year; additionally, among individuals experiencing a substance use disorder, almost 18% also experienced an anxiety disorder during the past year, and this rose to between 33% to 43% among individuals engaged in treatment (McHugh, 2015).





Weiss, 2019). Individuals experiencing alcohol use disorder were more than twice as likely to experience past-year major depressive disorder compared to individuals not experiencing alcohol use disorder, and the probability of this co-occurrence increased with alcohol use disorder severity; this association was more prevalent among women than men (McHugh & Weiss, 2019).

There remains a great deal of confusion in diagnosing alcohol-induced depression versus depression that occurs independently of (but co-occurs with) alcohol use disorder. Intervention strategies under review for concurrently treating co-occurring depression and substance use disorder include pharmacotherapy, combined motivational interviewing and cognitive behavioral therapy, and “transdiagnostic” integrated therapies such as behavioral activation (McHugh & Weiss, 2019). Another reason to explore depression and substance misuse, separate from the comorbidity issue, is that depression is an unfortunately common experience during early recovery from substance misuse/use disorder. “Intoxication and/or withdrawal from certain substances can lead to depressive symptoms...symptoms can last as long as an individual continues to take substances and may or may not improve with abstinence”—lasting up to 6 months of abstinence (CSAT, 2014, p. 7). Depressive symptoms are most likely to occur in relation to chronic use or withdrawal from alcohol, opioids, cocaine and other stimulants, cannabis, or sedative-hypnotic substances (CSAT, 2014).

Depression can interfere with a person’s road to recovery and participation in substance-related intervention efforts. Even if they attend intervention sessions, their depression symptoms may interfere with their ability focus, concentrate, remember, or pay attention during intervention (CSAT, 2014). The Center for Substance Abuse Treatment (CSAT) Treatment Improvement Protocol (TIP) #48 addresses depression during early recovery with the following recommendations:

- screening all substance use treatment participants for depressive symptoms and suicidality;

- being aware of how depressive symptoms appear in persons with substance use disorders and how these might affect treatment participation, process, and outcomes;
- delivering client-centered, integrated treatment for co-occurring depressive symptoms and substance misuse/use disorders;
- delivering evidence-supported interventions (e.g., behavioral, cognitive behavioral, supportive, expressive, 12-step facilitation, and motivational interviewing);
- being aware of how one's own attitudes toward clients' depressive symptoms might affect work with these individuals.

*“Depression and hopelessness, combined with alcohol and/or drug use, may also increase the potential for violence to self or others. The client may be at higher risk for thinking about, planning, or acting on suicidal thoughts.” (CSAT, 2014, p. 9)*

**Thought disorder.** Both schizophrenia (including schizoaffective and delusional disorders) and dementia are explored under this heading. In these cases, a person experiences episodes during which it is difficult or impossible for them to distinguish between their external and internal worlds—whether the information they are receiving is coming from the outside world or generated in their minds (van Wormer & Davis, 2013).

*Schizophrenia.* Early research into the co-occurrence of schizophrenia and substance use disorder estimated that 47% of individuals with schizophrenia also met criteria for a substance use disorder involving alcohol or illicit substances—4.6 times greater than among the general population—and at least 70% of individuals with chronic schizophrenia exhibited nicotine dependence (Winklbaaur et al., 2006). The substances most often



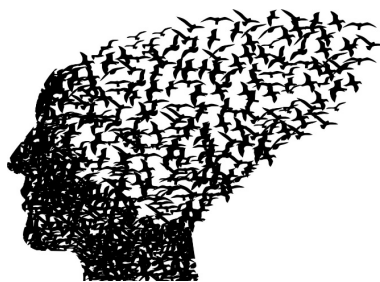
used by individuals with schizophrenia were alcohol, cannabis, nicotine, and cocaine. Among individuals with schizophrenia, schizoaffective disorder, or bipolar disorder with psychotic features, substance use was significantly more prevalent compared to a comparison group of individuals without severe mental illness (Reedy, 2020).

The increased risk of psychosis or psychotic symptoms among some individuals engaging in cannabis misuse was offered as evidence that substance misuse can cause one or more symptoms of mental illness (NIDA, 2010): “in particular, heavy cannabis use may accelerate or exacerbate psychotic symptoms in vulnerable individuals” (Winklbaaur et al., 2006, p. 39). There appears to be a genetic basis to this vulnerability (NIDA, 2010). However, the self-medication hypothesis has tended to dominate explanations of high co-occurrence rates: substance misuse may help a person deal with side effects of medications treating schizophrenia symptoms and/or symptoms of the schizophrenia itself (Winklbaaur et al., 2006). In addition, the third factor of chronic stress also comes into play: it is a common factor in the severity both of schizophrenia and substance misuse (Winklbaaur et al., 2006). And, some reward neural pathways of the brain and neurotransmitters involved in

progression to addiction are also involved in schizophrenia symptoms, suggesting that a person with schizophrenia may be more susceptible to have substance use progress to a substance use disorder (Winklbaaur et al., 2006).

It can be difficult to tell the difference between behaviors and symptoms caused by substance misuse, withdrawal from substances, and symptoms of a primary thought disorder like schizophrenia. Good assessment, diagnosis, and dynamic evaluation processes are critical to making such a determination and to intervention planning. Complicating treatment for either schizophrenia or substance use disorder is the possibility that either problem will interfere with treatment, leading to significant lapses in treatment and/or increased symptom severity. Additionally, a person with either problem alone or in combination may not engage in adequate self-care—nutrition, hygiene, personal safety and shelter—and may lose/lack social relationships and structures that help keep them functional and able to maintain their health, mental health, and substance recovery. Finally, the misused substance may interact badly or dangerously with medications prescribed for treating schizophrenia. Strong case management practices may be important in supporting individuals who experience co-occurring schizophrenia and substance misuse/substance use disorder.

*Dementia.* While schizophrenia tends to first appear in younger adults, most forms of dementia tend to first appear during later adulthood. “Dementia refers to a set of symptoms and signs associated with a progressive deterioration of cognitive functions that affects daily activities. Symptoms may include memory loss and difficulties with thinking, problem-solving or language, as well as changes in mood, perception, personality, or behaviour” (Peprah & McCormack, 2019, p. 3). The most common recognized form of dementia is Alzheimer’s disease (Peprah & McCormack, 2019). There exists some evidence to indicate that several types of substance misuse are associated with a higher risk for later development of dementia.



Prolonged excessive alcohol consumption can lead to permanent changes in brain structures and function, some of which are associated with an alcohol-related form of dementia or diagnosis of **Wernicke-Korsakoff syndrome**

—these may be two distinct syndromes of alcohol-related impairment or they may be variants of the same (Ridley, Draper, & Withall, 2013). Wernicke-Korsakoff syndrome includes some degree of psychosis 80-90% of the time, as well as retrograde amnesia (memory loss), difficulty forming new memories (anterograde amnesia; Martin, Singleton, & Hiller-Sturmhöfel, 2003). The neurological damage and impaired cognition gradually and progressively appear in individuals who have a long history of heavy alcohol misuse. Some behavioral symptoms mimic depression, potentially leading to the erroneous conclusion that an aging person is drinking because of aging-related depression, rather than recognizing that the drinking came first and continued for a long time. If detected early enough, some alcohol-related dementia symptoms can be reversed with proper treatment. A review of literature suggested that alcohol-related dementia may appear at ages younger than many dementia studies include, but across studies the prevalence of alcohol use disorder among individuals with dementia ranges from 9% to 22% and dementia is present in 10% to 24% of individuals with alcohol use disorder (Ridley, Draper, & Withall, 2013).

The evidence surrounding cannabis misuse is less consistent than what we see regarding alcohol misuse. In some studies, long-term heavy cannabis use has been statistically associated with some cognitive deficits involved with dementia later in life—memory, attention, and planful executive functioning tasks have each shown deficits. Shorter term, less frequent cannabis use does not seem to generate the same detrimental effects, and some studies indicated



that cannabis use may help slow the progression of some forms of dementia or improve some dementia symptoms—however, the evidence is inconclusive, only suggestive (Peprah & McCormack, 2019).

**Personality disorder.** Inflexible, enduring patterns of internal thought processes (cognitions), behavior, affect/emotions, interpersonal relations, or impulse control that lead to significant distress or impairment are encapsulated under the broad



category of personality disorder diagnoses (Hassin & Kilcoyne, 2012). Exploring the co-occurrence rates of personality disorders with substance use disorder (and vice versa) is complicated by a bit of pretzel logic since substance misuse is one of the diagnostic criteria for several types of personality disorder.

In a review of NESARC data from two periods, antisocial, borderline, and schizotypal personality disorders consistently predicted alcohol, cannabis, and nicotine use disorders; these three personality types also predicted persistence over time (three years) of cannabis misuse, other illicit substance misuse, and prescription drug misuse disorders (Hassin & Kilcoyne, 2012). Additionally, antisocial personality disorder was associated with smoking behavior, not only with nicotine dependence, and obsessive-compulsive personality disorder was associated with drug and nicotine disorders. Of significant concern was the authors' conclusion that personality disorders predicted poorer course of comorbid substance use (and other mental) disorders. In many instances, a diagnosis of antisocial personality disorder no longer applies after a person is well into recovery—the behaviors and characteristics that warranted the initial antisocial personality disorder diagnosis were part of the survival strategy for maintaining a pattern of substance misuse (van Wormer & Davis, 2013).

Borderline personality disorder is characterized by some of the same traits associated with substance misuse/substance use

disorders; similarly, the intoxication or withdrawal phases of substance use are “characterized by features that resemble” borderline personality disorder (Trull et al., 2018, p. 2). The overlaps might include:

- emotion dysregulation, affective instability,
- impulsive acts,
- disturbed interpersonal relationships, interpersonal problems, and
- suicidal/self-harm behaviors (Trull et al., 2018).

Among individuals diagnosed with substance use disorder or receiving treatment for addiction, 22.1% were also diagnosed with borderline personality disorder when multiple studies were considered in combination; about 17% experienced co-occurring alcohol use disorder, and cocaine and opioid dependence occurred at relatively high rates, as well (Trull et al., 2018). The following table is based on data presented in analysis of Revised NESARC data by Trull et al. (2010).

Personality Disorder	% alcohol dependent	% drug dependent	% nicotine dependent
All types combined	42%	19%	48%
schizoid	38%	20%	40%
antisocial	52%	27%	59%
borderline	47%	23%	54%
narcissistic	39%	17%	44%
obsessive-compulsive	32%	11%	36%

**Impulse control disorders or ADD/ADHD.** Bipolar disorder could have been addressed in the prior section concerning depression and mood disorders, but here it is placed in a category of impulse control disorders. This choice was made because common

symptoms of bipolar disorder (mania and impulsivity) also are associated with substance use disorder (Reedy, 2020): “Bipolar disorder has a high co-occurrence with substance abuse disorders” (Post & Kallivas, 2013, p.172). This co-occurrence often appears with more severe bipolar disorder that is more difficult to treat, and there seems to be an iterative sensitization process between them where each episode of either contributes to progression of the other condition (Post & Kallivas, 2013). Other investigators concluded that alcohol misuse by individuals with bipolar disorder was associated with poorer recovery following their first hospitalization for mania, a greater number of recurrent bipolar episodes and hospitalizations, poorer response to pharmacotherapy, poorer adherence to treatment regimens, and greater progression of an alcohol use disorder (Strakowski et al., 2005). Part of bipolar disorder involves mania, a period during which a person engaged to a great extent in activities which are pleasurable (van Wormer & Davis, 2013)—pleasurable activities which could include substance misuse.

In our prior module concerning CNS stimulants, we introduced the topic of attention deficit disorder with and without hyperactivity—ADD and ADHD. Wilens and Morrison (2011) reviewed the literature concerning the intersection of ADD or ADHD with substance misuse, drawing the following conclusions:

- The rate at which ADHD coincides with substance use disorders is higher than would be expected merely by chance—the rate of ADHD in the general population is about 5-9% but among adults with substance use disorders it is around 25%, and among adolescents with substance use disorders, around 50% have diagnosable ADHD.
- In the group of individuals experiencing substance use disorders, substance misuse generally started earlier among those with ADHD than among those without ADHD. This is relevant because, as you may recall from prior modules, earlier initiation is predictive of more severe problems with substance

misuse/use disorder later down the line.

- Since symptoms of ADHD appear much earlier in life than does substance misuse, ADHD appears to influence the emergence of substance use disorder (not the other way around).
- Individuals with ADHD plus conduct disorder and/or bipolar disorder have the greatest probability of developing substance use disorder.
- Early treatment of ADHD with stimulant medication neither increases nor decreases the risk for subsequent substance use disorder; however, it may delay substance use initiation during adolescence.

Again, why does this evidence matter? It should help inform prevention efforts, for one thing. It helps to know where we might want to focus some of our more specific, targeted efforts at prevention, especially our efforts to delay initiation of substance use. It matters because we may need to rethink our substance misuse/use disorder treatment approaches in terms of how well suited they are for a population with ADHD traits. This includes thinking about pharmacotherapy options, particularly medication management (MM) efforts to ensure better compliance with the medication schedule by individuals who have a lifetime of greater difficulty organizing themselves for reliable follow-through.

**Trauma.** **Post-traumatic stress disorder (PTSD)** symptoms include difficulties with concentration, attention and focus, along with anxiety and insomnia or sleep disruption. Recalling or psychologically reliving trauma events in dreams and waking memories can be experienced as intensely as the original event—the sympathetic nervous system responds and triggers the “fight or flight” responses of increased heart rate, muscles ready for action, shifting blood sugar levels, heightened blood pressure, and so forth. It takes a while for the parasympathetic system to calm things down again, and a person with PTSD may spend an extreme amount of time in a hyper-vigilant, “ready for action” physiological state. From all that we have learned about the role of neurotransmitters

and different regions of the brain regarding substance misuse, we can piece together at least part of the story as to why PTSD and substance misuse might co-occur. The experience of prolonged, chronic stress floods the body and brain with stress hormones and alters brain pathways (e.g. hypothalamus control center and its memory storage areas and the hippocampus and amygdala involved in stress responses). Combined with exposure to various substances, it becomes evident that trauma-related stress can easily lead to substance misuse and substance use disorders.



Early research concerning trauma and PTSD largely stemmed from work with combat veterans. More recently, practitioners and scholars have recognized many of the same trauma-related symptoms (whether or not these meet criteria for a PTSD diagnosis) among numerous other populations, as well: children, adolescents, and adults who have been the target of or witness to family or community violence (e.g., intimate partner violence, sexual assault, child maltreatment); survivors of community-wide natural disasters; and, other survivors of severe and/or life-threatening/life-altering events. The main “take home” lesson from the literature concerning the co-occurrence of trauma/PTSD and substance

misuse/substance use disorder is that trauma histories are very common among individuals experiencing substance use disorders.

Combining numerous studies, it appears that alcohol use disorder occurs among 24% to 52% of individuals with PTSD, nicotine dependence among at least 19%, and cannabis use disorder being up to 6 times more common in this population; cocaine use disorder also co-occurred with PTSD at relatively high rates, as did opioid use disorder, particularly among individuals also experiencing chronic pain as a result of trauma (Bailey & Stewart, 2014).

*“It is now a well-established fact that there is a surprisingly high degree of overlap between substance misuse and PTSD across diverse community and patient samples. This overlap is of clinical significance because individual with comorbid substance use and PTSD show poorer functioning across various indicators and may also suffer from worse long-term clinical trajectories”* (Read & Oimette, 2014, p. 4).

Just as we learned early in this course about the problem of casually using words like “addiction” and “addicted,” it is equally important to address overly casual use of the term “PTSD” that has crept into our society. Like substance use disorders, PTSD is a very real disorder with debilitating symptoms that also can be effectively treated if properly diagnosed and managed. And, many individuals who experience a traumatic event do not develop symptoms associated with PTSD (Bailey & Stewart, 2014). Trauma related to disaster events and experiences of military veterans are introduced here; intimate partner violence, child maltreatment, and sexual assault are presented in a later section.



*Disaster events.* Trauma and PTSD often result from experiencing a disaster event, whether it is a natural disaster (e.g., earthquake, flood, tornado, hurricane, tsunami), health disaster (e.g., epidemic or pandemic), or caused by persons (e.g., terrorist bombing, mass shooting, arson fire). Substance misuse may begin or worsen as a result of disaster exposure. Both immediately and 6 months following the traumatic events that unfolded in New York, Washington DC, and Pennsylvania on 9/11 of 2001, investigators of several different studies identified increased nicotine, alcohol, and cannabis use among residents and first responders/disaster aid workers, with heavy episodic drinking being worse the greater the number of traumatic events and degree of PTSD symptomatology experienced in the aftermath (Bailey & Stewart, 2014). Alcohol misuse increased among first responder firefighters following the Oklahoma City federal building bombing, but primarily those who already experienced an alcohol use disorder; taken together these findings suggest “that increases in substance use may be most strongly related to PTSD status and predisaster substance use” (Bailey & Stewart, 2014, p. 14). Consider also that widespread community disaster events (e.g., Hurricanes Katrina and Rita within one month, or the COVID-19 pandemic) may lead to individuals undergoing unintended, unmanaged (and potentially dangerous) alcohol or other substance withdrawal if the distribution network/supply access is disrupted.

## Military

veterans. Practitioners and health care providers are developing an increased awareness and understanding of the stress symptoms exhibited by men and women experiencing exceptional circumstances in military service. Their symptoms may or may not rise to the level of diagnosable post-traumatic



stress disorder but may still have profound effects on their lives. In a national survey of U.S. veterans, among those with PTSD (whether combat exposed or not), almost 17% also experienced alcohol use disorder and among veterans with alcohol use disorder, over 20% also experienced PTSD (Norman et al., 2018). Veterans experiencing co-occurring alcohol use disorder and PTSD were more likely than their counterparts with alcohol use disorder alone to have positive screening results for major depression (36.8% versus 2.3%), generalized anxiety disorder (43.5% versus 2.9%), suicidal ideation (39.1% versus 7.0%), or suicide attempt (46.0% versus 4.1%). Their overall quality of life was significantly lower, as well. In other research, cannabis use disorder also was significantly associated with PTSD and depression among veterans receiving services (Reedy, 2020). Estimates of the prevalence for substance use disorder (particularly nicotine, alcohol, and cannabis) among individuals with PTSD exposed to combat ranged between 31% to 76%: combat exposure alone is not the predictive factor, instead it is the experience of PTSD following combat exposure that matters (Bailey & Stewart, 2014). Consider that trauma may be experienced by non-combat members of the military, as well (see sexual assault topic below). Members of the military and military veterans may experience a double threat as far as co-occurring substance misuse and PTSD: not only might they engage in alcohol or other substance



misuse as a coping strategy, both internalized and external stigma may impede their willingness/ability to seek behavioral health care (Miller, Pedersen, & Marshall, 2017). Furthermore, they may fear being discharged from their military careers if their difficulties with PTSD and/or substance misuse is discovered.

**Gambling disorder.** In the DSM-5, gambling disorder is a diagnosable mental disorder with neurobiological similarities to certain substance use disorders (Nower, Mills, & Anthony, 2020). It is defined as “a persistent maladaptive pattern of gambling resulting in clinically significant impairment or distress,” such that an individual exhibits four or more from a set of nine symptoms within a 12-month period, including five that are similar to what is involved in substance use disorder diagnosis (Rash, Weinstock, & Van Patten, 2016, p. 3):

- tolerance—gambling with increasing amounts of money to achieve the desired level of excitement;
- loss of control—unsuccessful attempts to control, limit, or stop gambling;
- withdrawal—restlessness and/or irritability when trying to control gambling;
- negative consequences—risked or lost significant relationships or opportunities because of gambling;
- fixation—preoccupation with gambling-related thoughts (e.g., reliving past gambling experiences, planning future experiences, strategizing ways to fund gambling).

The remaining four are somewhat unique to gambling:

- negative affect—frequently gambles in response to negative affect;
- chasing losses—often follows gambling losses by returning another day in attempt to recoup losses;
- lying—lies about gambling or its consequences;
- bailouts—depends on others for money to alleviate desperate

financial situations cause by gambling (p. 4).

Internet gaming and problematic use of technology has much in common with gambling disorder and internet gaming disorder has been proposed a condition relevant for further study as a diagnosable disorder in the DSM-5; gaming disorder is included in the ICD-11 (Anthony, Mills, & Nower, 2020). As of 2013, the types of gambling in which the largest percent of U.S. adults engaged were (Welte et al., 2015):



- lottery (62%)
- office pools/raffles (40.2%)
- casino gambling (26.2%)
- cards, including via internet (19.2%)
- slot machines outside of casino, including via internet (17.4%)
- sports betting, including via internet (16%).

The U.S. prevalence of past year gambling disorder is about 2% and about 3% over the lifetime, however the 6% rate of gambling disorder and 15% for problem gambling is much greater in communities where gambling is integral to economic and social systems (Nower et al., 2020).

The association between gambling disorder and alcohol or other substance use disorder is “well established” (Rash et al., 2016, p. 5): 28% of problem/pathological gamblers experience an alcohol use disorder and 17% experience substance use disorder involving illicit substances. Among individuals seeking treatment for problematic gambling or gambling disorder, over 40% meet criteria for lifetime alcohol use disorder and 21% for substance use disorder involving other substances—including nicotine—and having a lifetime history

of substance use disorder is associated with lower rates of achieving gambling abstinence, just as problems with gambling predict poorer substance-related treatment outcomes (Rash et al., 2016). Of interest is the observation that at-risk alcohol use patterns decreased during gambling treatment and incorporating brief alcohol interventions into gambling treatments may further advance these reductions in drinking behavior (Rash et al., 2016). Screening for problematic gambling is recommended for anyone entering treatment for substance use disorder and, vice versa, substance misuse screening is recommended for anyone entering treatment for problematic gambling (Rash et al., 2016). Similarly, ongoing assessment for suicidality is critically important: rates are considerably higher than in the general population—as much as three times more common than in the general population (Nower et al., 2020).

**Intervening around co-occurring mental disorders and substance misuse.** Both substance misuse and mental disorders can contribute to difficulties in daily living and “generally have a synergistic effect and either can impede treatment of the other,” thus traditional unidimensional sequential or parallel treatment approaches are ill-advised (Reedy, 2020, p. 536). Instead, integrated treatment is recommended for these co-occurring disorders, including when delivered collaboratively by different providers and designed to meet holistic needs over a period of months to years (Reedy, 2020).

Several behavioral therapies judged to have strong evidence supporting their use with co-occurring substance use and mental disorders include (NIDA, 2018):

- cognitive behavioral therapy (CBT)
- dialectical behavioral therapy (DBT)
- assertive community treatment (ACT)
- therapeutic communities (TC)
- contingency management (CM).

An evidence-supported model for addressing women's co-occurring PTSD and substance use disorder was initially presented as *Seeking Safety* (Najavits, 2022).

Pharmacotherapy strategies are also relevant for consideration and may assist in managing one or the other disorder, or perhaps in managing both simultaneously: the example presented by NIDA (2018) is the potential use of bupropion to treat both depression and nicotine dependence. The report also suggests that evidence concerning how these pharmacotherapy agents work singly or in combination for populations experiencing comorbid mental conditions is sorely lacking.

## Physical Health

Aside from the risk of accidental overdose, misuse of alcohol, prescription or OTC drugs, and other substances potentially takes a toll on physical health. Substance misuse “contributes to the risk of developing or complicating other illnesses, as well as the substances interacting negatively with medications used to treat medical conditions” and may also impact adherence to medical treatments (Saunders-Adams, Hechmer, Peck, & Murray, 2020, p.438). Integrated health care systems provide behavioral health and substance-related interventions seamlessly within non-stigmatizing settings where a person might be receiving their general, primary health care services (Saunders-Adams et al., 2020). Advocates of integrated care models point to data indicating that substance misuse and substance use disorders are undertreated in the U.S. and globally, with segregation of physical and behavioral health care systems being largely to blame (Saunders-Adams et al., 2020). For example, NSDUH data from 2018 (SAMHSA, 2019a) led to an estimate that over 20 million individuals aged 12 or older (7.4% of population) experienced a past year substance use disorder involving alcohol or illicit use of substances; however, only 3.7

million (1.4% of population) received alcohol or other substance use treatment during that same year. Many more individuals could potentially be identified early in their substance use-disorder trajectory, supported in recovery, and/or assisted through harm reduction interventions should they enter appropriately prepared integrated healthcare service delivery systems (Saunders-Adams et al., 2020). Physical health concerns that commonly co-occur with different types of substance misuse are worth exploring.

### **Infectious disease exposure, infection, and disease progression.**

The significance of “mode of administration” in substance misuse is a theme woven through several modules in this course. The topic is once again relevant because injection administration increases the risk of both exposure to infectious disease and local, injection site infections: viral hepatitis (hepatitis B/HBV, hepatitis C/HCV) and HIV (human immunodeficiency virus), as well as both bacterial and fungal infections (CDC, 2018). Of concern are viruses transmitted through blood or other body fluids present when individuals share drug equipment, including needles (NIDA, 2019). Injection drug use was a contributing factor in their acquiring HIV at a 20% rate among men with the infection (150,000 cases) and 21% among women (50,000), according to 2016 CDC data (NIDA, 2019). Harm reduction practices known as syringe services programs (SSPs) and pharmacies being allowed to sell sterile needles without a prescription are recommended community-based strategies (NIDA, 2019).

Not only is the concern that someone engaged in injection or non-injection substance misuse might become infected with a disease, but their disease progression and prognosis may be worse, as well. For example,

in a study of 1,712 individuals entering care related to HIV infection, “patients with a history of injection drug use were more likely to advance to AIDS or death than non-users”; the researchers detected



no statistically significant difference in disease progression between individuals engaged in non-injection drug use and those who did not engage in substance misuse (Qian et al., 2011, p. 14).

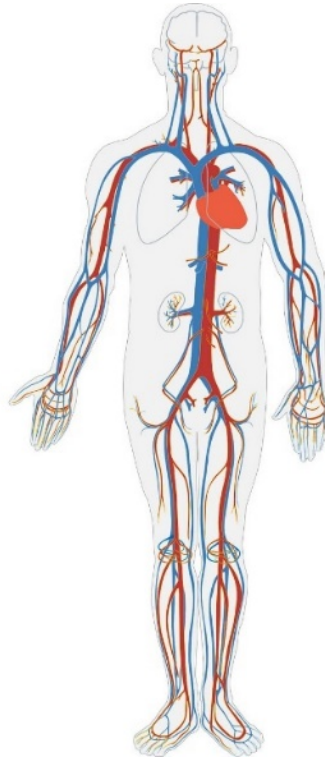
Furthermore, these infectious diseases can be risk factors in developing other serious health problems or diseases. For example, hepatitis (types B and C) may lead to cirrhosis/loss of liver function and is a significant risk factor in liver cancer (NIDA, 2019); tuberculosis risks include antibiotic resistance, and can affect lungs, brain, kidneys, and spine (<https://www.cdc.gov/tb/publications/factsheets/general/tb.htm>). Various factors might explain the disease progression phenomenon, but changes in the immune system, as well as a person's overall health and nutrition status may leave them less able to combat infection or disease. Additionally, substance misuse may worsen the situation: "Drug use can worsen the progression of HIV and its symptoms, especially in the brain. Studies show that drugs can make it easier for HIV to enter the brain and cause greater nerve cell injury and problems" (NIDA, 2019). Individuals engaged in significant levels of substance misuse or experiencing substance use disorder may not receive routine primary health care, testing, and vaccinations that could prevent or minimize the seriousness of infectious diseases; this includes lack of prenatal care among pregnant women.

Individuals engaged in substance misuse, with or without injection use, are also more susceptible to acquiring sexually transmitted infections (STIs) and tuberculosis infection or disease. For example, authors of a literature review concluded that the rate of latent (asymptomatic) tuberculosis among individuals engaged in illicit drug use was 10% to 59% (Deiss, Rodwell, & Garfein, 2009). Also of considerable concern is the increased rate of co-infection between tuberculosis, HIV, and hepatitis (Deiss, 2009). A significant barrier to disease recognition and treatment is the relatively poor access to appropriate primary and specialty care this population experiences (Rodwell, 2009). So too are viruses and other infections sexually transmitted through blood or body fluids, including HIV (NIDA, 2019). Substance use contributes to unsafe sex practices/

failure to consistently employ safe sex practices, especially when judgment is impaired, sexual risk is underestimated, or sex is the currency through which the drugs are acquired.

**Non-communicable diseases/health**

**complications.** As we explored each type of substance, you learned about some of the effects each has on mind, behavior, and health/organ systems. For example, effects on breathing, heart rate/rhythms, blood pressure, immune system, diabetes, stroke risk, fertility/infertility, fetal development, and sleep patterns. Of additional concern is evidence that some types of substance misuse increase the probability of developing cancer.



- *Tobacco products.* Among all cancers diagnosed in the U.S., 40% are linked to tobacco use, including cancers of the mouth and throat, esophagus and voice box, lungs, trachea and bronchus, liver, stomach, kidneys/bladder, uterus/cervix, pancreas, colon and rectum, and blood (acute myeloid leukemia) (<https://www.cdc.gov/media/releases/2016/p1110-vital-signs-cancer-tobacco.html>).
- *Alcohol.* Alcohol increases the probability of developing cancers of the mouth and throat, esophagus and voice box, liver, colon and rectum, and (for women) breasts, and the risk increases as a function of increased alcohol consumption (<https://www.cdc.gov/cancer/alcohol/index.htm>). Cancer

risk increases even more when alcohol and tobacco are both involved (Saunders-Adams et al., 2020).

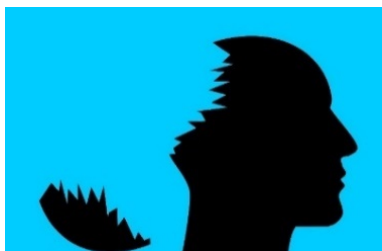
- *Cannabis*. The picture concerning possible cancer risks associated with cannabis/marijuana use is mixed. In a review of literature, authors concluded: “There is currently no consensus on whether marijuana use is associated with cancer risk” (Huang et al., 2015, p. 15). The only consistent evidence they identified suggested that testicular cancer risk increased with frequent marijuana use. Another review drew the same conclusion regarding testicular tumors, and findings for lung cancer were mixed (Ghasemiesfe et al., 2019).
- *Sedative-hypnotics*. There exists some evidence that regular, prolonged use may contribute to oral, liver, and breast cancer (Fang et al., 2019).
- *Opioids*. There exists some evidence that regular, prolonged opioid/opiate use may contribute to cancers of the bladder, kidney, oral, esophagus, and larynx/pharynx (Rashidian et al., 2016).
- *Anabolic steroids*. Steroid misuse typically occurs at high concentrations compared to what is typically prescribed for treating medical conditions; this pattern of misuse is associated with increased risk of developing liver, testicular, prostate, breast, and colon cancers; it is also associated with more aggressive forms of cancer (Tentori & Graziani, 2007).

Not only are these specific substances of concern, so too are possible contaminants and additives that also may be harmful to health—especially drugs that are illicitly manufactured or distributed.

**Traumatic brain injury, disability, and other accidental injury.** Disability is different from disease in that it concerns long-term mild to severe consequences resulting from injury, disease, genetics, or birth-related circumstances rather than from specific pathogens or disease processes. Substance misuse is an all-too-common cause underlying emergency department visits in the U.S. Estimated based



on the Drug Abuse Warning Network (DAWN) 2011 data were 5.1 million drug-related emergency department visits occurred, almost 2.5 million of which were associated with drug misuse or abuse: 51% involved illicit substances, 51% involved nonmedical use of prescription drugs, and over 25% involved drugs combined with alcohol—over 600,000 visits involved drugs combined with alcohol (SAMHSA, 2013). More than 40% of emergency department visits by individuals under the age of 21 involved alcohol (almost 118,000 in this age group involved alcohol alone), particularly among those aged 18-20 years (SAMHSA, 2013).



“Traumatic brain injury (TBI) and substance abuse (SA) are two of the leading causes of disability” (Sacks et al., 2009, p., 405). Substance misuse is a recognized risk factor for TBI and, vice versa, TBI is a risk factor for substance misuse or

substance use disorder; substance misuse also is associated with forms of violence and accidents that result in TBI (Sacks et al, 2009). TBI may result in persistent deficits in executive cognitive functioning, depending on the regions of the brain affected, and this may heighten vulnerability to substance misuse (Bjork & Grant, 2009). Individuals with TBI have demonstrated a propensity to choose immediate small rewards preferentially over delayed larger rewards, and may be impaired in their ability to envision the negative consequences associated with substance misuse, or to recognize their own perception of themselves as having control or self-efficacy over their own behaviors (Bjork & Grant, 2009). These factors not only affect addictive behavior choices, they likely affect recovery and intervention delivery, as well.

Among individuals in treatment for substance use disorders, 38% to 63% also had a traumatic brain injury (Corrigan et al., 2005). Screening for both TBI and substance misuse is relevant because each complicates the course of treatment for the other (Sacks et

al., 2009). It is important to recognize that TBI may not occur all at once, in one severe event, but may result from an accumulation of less severe events; mild TBI represents up to 90% of all TBIs, and more than 60% of individuals experiencing repetitive mild TBI do not seek medical care (Haycraft & Glover, 2018). For example, in a study of 845 individuals entering treatment for alcohol or other substance use services, 54% had positive screening results for a prior TBI (Sacks et al, 2009). Only 24% of participants reported not having received any blows to the head while over 50% reported having received more than two blows to the head and more than 25% reported more than four; sports-related injuries were the most common source (18%), with assault (14.5%), motor vehicle accidents (13%), falls during drug/alcohol blackout (9%), and other falls (12%) also commonly represented. The authors recommend multifaceted (integrated) treatment addressing substance misuse as well as cognitive remediation and addressing emotional and behavioral aspects associated with TBI. They go on to say that individuals with cognitive deficits associated with TBI may be unable to effectively engage in traditionally delivered substance-related treatment programs and relying on narrowly focused approaches result in treatment failure and relapse:

“For these individuals, strategies to facilitate compensation for these cognitive deficits need to be incorporated as core components of their [substance abuse] treatment (e.g., allowing for more repetition of information, prompting individuals to write things down to aid in memory for information, use of memory books, extended treatment duration)” (Sacks et al., 2019, p. 413).

**Driving under the influence (DUI).** Depending on the state or local jurisdiction, the offense of impaired, drugged, or drunk driving may be called:

- driving under the influence (DUI),
- driving while intoxicated (DWI),

- driving while impaired (DWI),
- operating (a vehicle) under the influence (OUI), or
- operating (a vehicle) while intoxicated (OWI).

As noted in our module focused on alcohol, even if a person’s blood alcohol level (BAL)/blood alcohol concentration (BAC) does not measure at the legal limit (0.08%), performing the complex array of tasks involved in driving still may be impaired. Impairment may also occur with other legal prescription and OTC substances, as well as an array of illicit substances. The odds of driving accidents increase sharply with increasing BAC levels, according to calculations presented by the World Health Organization (WHO, 2009)—any fraction over the value of 1.0 is an increased risk (“buzzed driving is drunk driving”):

BAC range	Odds Ratio
0.01% – 0.04%	1.17
0.05% – 0.07%	1.71
0.08%* –0.10%	3.93
0.11% or greater	10.68
*legal limit in U.S.	

The 2018 NSDUH data (SAMHSA, 2019a) led to estimates that over 20 million individuals aged 16 and older (8%) drove under the influence of alcohol during the past year, and 12 million (4.9%) under the influence of illicit substances—most often, marijuana (11.8 million, 4%).

News reports in recent years have identified car crashes where legal non-benzodiazepine sleep medications (e.g. Ambien®) were reportedly involved. While it is clear to most of us that driving under the influence of alcohol, marijuana, or (other) illicit substances is a bad idea (and illegal), it is dangerous to overlook the potential dangers associated with driving under the influence of (other) legal OTC and prescription substances. The Automobile Association of

America (AAA, 2014) reported that, while 66% of people consider driving under the influence of alcohol to be a very serious threat and 56% considered driving under the influence of illegal drugs to be so, only 28% consider driving under the influence of prescription drugs a very serious threat. They reported that the crash risk increased by up to 41% when driving under the influence of certain antidepressants and that even over-the-counter cold and allergy medications can impair driving. The AAA Foundation for Road Safety hosts an interactive informational site where specific prescription and OTC medications can be searched for potential drug interactions, food interactions, driver warnings, and general medication information: <http://www.roadwiserx.com/>. Dangers also apply to operating any dangerous equipment, not only motor vehicles (e.g. boats, snow mobiles, riding mowers, planes, and industrial/construction machinery).





### **STOP & THINK**



An interactive H5P element has been excluded from this version of the text. You can view it online here:

<https://ohiostate.pressbooks.pub/swk5805coursebook/?p=240#h5p-55>

# Ch. 2: Additional Challenges That Co-occur With Substance Misuse

The topics explored in this chapter are not necessarily distinct from those discussed in the first chapter: they often co-occur in multiples, complicating one another and compounding vulnerability and risk for initiation or worsening of substance misuse, as well as a host of additional physical and mental/behavioral health concerns. In this chapter we focus on a variety of situational circumstances: violence (intimate partner violence, sexual assault, and child maltreatment), employment/unemployment concerns, human trafficking and sex work, housing insecurity, and criminal justice system involvement/incarceration. These issues are often significantly intertwined in a person's life, along with some of the issues previously discussed (e.g., mental and physical health concerns, violence exposure). For these reasons, scholars recommend substance-related interventions and programs integrate case management into their design to better meet the complex, dynamic needs participants experience (Zweben & West, 2020).

## Violence Exposure and Perpetration

Thinking about a person's exposure to violence or perpetration of violence against others is an example of why it is important to consider the broader range of co-occurring problems rather than limiting attention to comorbid, dual diagnosis, or co-occurring disorders: violence has no diagnostic category, but certainly has a

strong relationship to substance misuse. The relative risk of injury related to alcohol use is remarkably greater for intentional injury by someone else (RR=21.50) compared to other sources of alcohol-involved injury, and the odds ratio for violence-related injury increased as a function of amounts of alcohol consumed (WHO, 2009):

Number of Drinks	Odds Ratio
0	1.00
1 – 2	11.14
3 – 4	13.76
5 or more	35.57

This leads to an examination of the relationship between alcohol or other substance misuse and intimate partner violence, sexual assault, and child maltreatment

**Intimate partner violence.** A great deal of evidence indicates that alcohol and other substance misuse are related both to perpetrating and experiencing intimate partner violence (Mengo & Leonard, 2020). On the subject of **intimate partner violence (IPV)** perpetration, half of men engaged in programs to address their IPV



behaviors (“batterer” treatment) also engaged in alcohol or other substance misuse, and half of men with partners who entered substance-related treatment perpetrated IPV events during the past year; furthermore, the two groups of men were 8 or 11 times as likely (respectively) to perpetrate IPV on a day they had been drinking (Bennett & Bland, 2008). Men’s alcohol misuse consistently has been shown to be “one of the most consistent risk factors for IPV” (Mengo & Leonard, 2020, p. 547). Furthermore, “violence is more severe

when one or both partners (most often the male partner) has been drinking” (Wilson, Graham, & Taft, 2014, p. 1). Intoxication by or withdrawal from certain substances might increase a person’s overall aggressiveness to anyone in the immediate vicinity, with intimate partners likely being in proximity. Different reviews have specified cocaine and amphetamines/methamphetamine misuse as factors in IPV perpetration, but results are mixed concerning the relationship of IPV with marijuana, sedatives, hallucinogens, and opiates (Begun, 2003; Mengo & Leonard, 2020); withdrawal symptoms from some substances may also be related.

*IPV perpetration.* Relationships between substance misuse and intimate partner violence (IPV) are complex, and no single mechanism explains it. Possible models related to alcohol or other substance misuse and IPV perpetration include (Begun, 2003; Mengo & Leonard, 2020):

- Certain substances may stimulate biological arousal of a person’s “fight or flight” response which becomes cognitively interpreted as calling for “fight.”
- **Alcohol myopia theory** specifies that intoxication distorts cognitive interpretation of situations—neutral events may be interpreted as hostile or threatening, dictating an aggressive response that would not be expressed in a sober state.
- Substance use may dampen or suppress a person’s learned inhibitions against acting aggressively (**disinhibition**).
- **Expectancy theory** suggests that an individual who believes alcohol (or other substance use) “causes” aggression may use alcohol (or other substances) as an “excuse” for acting aggressively toward others/intimate partners, in anticipation of acting this way.
- An intimate partner may be viewed as interfering with or standing in the way of a person’s access to or use of “needed” substances and/or the couple may argue about the substance misuse.
- Antisocial personality and adults’ antisocial behavior are



associated with both substance misuse and perpetration of intimate partner violence—in this model, substance misuse is not a causal factor in perpetrating intimate partner violence but is correlated with it through this third “externalizing” factor.

*IPV victimization.* In terms of the association between a person’s substance use and being the target of intimate partner violence, substance misuse may have preceded the violence or it may have only developed afterward, in response to the traumatic experience or injuries:

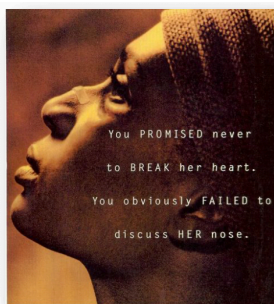


Image from Safe At Home media campaign, Milwaukee, WI

“substance use may be a response to the effects of IPV and/or render individuals more vulnerable to ongoing abuse” (Mengo & Leonard, 2020, p. 551). What we explored earlier about trauma/PTSD and “self-medicating” physical or emotional pain/stress apply to this discussion concerning IPV survivors. Having alcohol problems was reported by 29% of women who experienced intimate partner violence, and ¼ to ½ of women receiving IPV victim services also experienced problems with substance misuse (Bailey & Stewart, 2014; Bennett & Bland, 2008). From 55% to 99% of women who experience substance misuse/use disorder also have experienced IPV during their lives (Bennett & Bland, 2008 ).

*Intervention.* History and data show that if we require a person to successfully complete substance treatment and remain abstinent before engaging them in batterer treatment programming, there is poor follow through: their intimate partners remain at risk for ongoing abuse. On the other hand, it is very difficult to effectively engage a person in the kind of cognitive behavioral interventions commonly employed in programs designed to stop intimate partner violence perpetration: the person’s memory, perceptions, and

judgment may remain substance impaired. The current line of thinking is that it is preferable to address both problems simultaneously and integratively, rather than sequentially, but not all communities have this capacity available (Begun, 2003; Mengo & Leonard, 2020). Screening for substance misuse is advised for anyone engaged in IPV interventions—whether they are the perpetrator or survivor. Similarly, anyone engaged in substance-related intervention should be screened for IPV perpetration and/or victimization/survivorship (Mengo & Leonard, 2020).

**Sexual violence.** As many as 36% of women and 1 in 6 men in the U.S. experienced sexual assault during their lifetimes and “at least 50% of sexual assaults involved alcohol consumption by the victim, perpetrator, or both” (Davis, Kirwan, Neilson, & Stappenbeck, 2020, p. 561). Alcohol or other substances may be consumed either voluntarily or involuntarily—when a perpetrator administers them to a victim (e.g. “date rape” drugs). Either way, a sexual assault victim may be severely intoxicated, incapacitated, unconscious, or otherwise unable to consent—these are termed incapacitated or alcohol/drug facilitated sexual assaults or rapes (Davis et al., 2020). Drinking settings are relevant in that the majority of alcohol-involved sexual assaults occur either around bar and party contexts (Cleveland, Testa, & Hone, 2019) or within the context of intimate partner relationships; much less is known about drug-involved sexual assault (Davis et al, 2020).



Among college men, those with higher bar and party attendance had a greater probability of perpetrating sexual assault than other college men, particularly when they engage in greater number of “hook ups” (Testa & Cleveland, 2017). Alcohol-involved sexual assaults often are of a more severe form, with assault severity increasing as alcohol consumption increases, up to the point of heavy intoxication, and

often have more severe outcomes than assaults not involving alcohol (Davis et al., 2020; Testa, Vanzile-Tamsen & Livingston, 2004). Sex-related alcohol expectancies, increased impulsivity/disinhibition, slowed information processing/situational interpretation, and generally increased inappropriate behavior/aggression may play a significant role in sexual assault perpetration (Davis et al., 2020; Pegram et al., 2018). The alcohol myopia theory previously described regarding IPV may also be relevant to sexual assault perpetration and tendency to ignore or misinterpret the victim's social cues (Davis et al., 2020).

Heavy drinking in bar/party settings place a woman in a context where sexual assault is more common, and “being intoxicated can impair a victim’s ability to mitigate or thwart an attempted sexual assault” (Davis et al., 2020, p. 565). Heavy intoxication increases the risk of penetrating sexual assault (Testa et al., 2004); among college women, “heavy episodic drinking (HED) increases risk for sexual assault victimization, particularly incapacitated rape” (Testa & Cleveland, 2017). Intoxication may diminish a person’s sexual assault risk assessment (Melkonian & Ham, 2018) and increase self-blame for an experienced assault (Norris, Zawacki, Davis, & George, 2018). The fallout from sexual assault victimization can be traumatic—prior discussions of trauma and PTSD are applicable—and may involve physical as well as significant mental health and social relationship consequences. “Women with sexual assault histories may consume alcohol in part to regulate emotional distress and cope with negative affect states” (Davis, et al., 2020, p. 566). Thus, alcohol and other substance misuse may precede, follow, or both precede and follow a sexual assault experience.

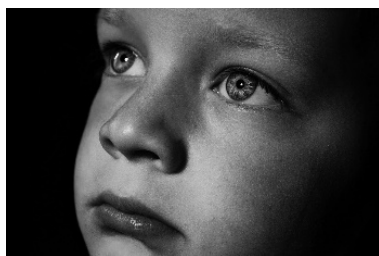


Sexual assault prevention interventions include risk reduction programs—preparing potentially vulnerable individuals with protective behavior strategies, situational awareness, and self-defense skills, as well as efforts to

change community norms surrounding violence against women (Davis et al., 2020). Prevention programs may be designed to intervene with potential perpetrators of sexual assault or to increase bystander awareness and willingness to intervene. Both types of prevention programs too rarely include information about alcohol or other drug involvement (Davis et al., 2020).

**Child maltreatment/  
neglect.**

Many individuals engaged in alcohol or other substance misuse are parents or primary caregivers to children or adolescents, and vice versa, over 12% of children under the age of 18 years (over



8.7 million) live with at least one parent experiencing a substance use disorder (Straussner & Fewell, 2020). These children are at greater risk for neurological, psychological, developmental, and behavioral problems, as well for experiencing some form of child maltreatment (Straussner & Fewell, 2020). Additionally, they may live under conditions of dysfunctional family dynamics, housing, food, and economic instability, and/or unstable legal and custody status.

Estimates of the proportion of child welfare cases involving parental substance misuse range from 34% to 78% and include harms on a continuum, from a parent's inability to function to children's exposure to violence, hazardous environments, and substances (Kepple & Freisthler, 2020). For example, accidental ingestion of drugs leads to a high number of emergency department visits for children, especially among children aged 5 years or under (SAMHSA, 2013). Children are at risk when a parent is intoxicated, experiencing substance withdrawal, or engaging in the illicit manufacture or distribution of substances (Kepple & Freisthler, 2020). Neglectful parenting is generally more common than physical violence: among children who experienced maltreatment in 2015, neglect represented 75% of the cases, 17.2% involved physical abuse,

and 8.4% involved sexual abuse (National Children's Alliance, <https://www.nationalchildrensalliance.org/media-room/nca-digital-media-kit/national-statistics-on-child-abuse/>).



A parent or caregiver engaged in substance misuse may spend long periods of time unaware of or unresponsive to a child's needs for food, protection, cognitive and language stimulation, discipline and guidance, comfort, health

care, school attendance, supervision, and other critical developmental needs. Furthermore, resources that could be used to meet a child's basic needs may be spent instead on substances. Harm may befall a child exposed to substance residue in their physical environment, or a child having access to alcohol, tobacco, or other substances with or without a parent's knowledge/approval. A parent's illegal dealing in drugs may involve a child's exposure to weapons and threats of or witnessing violence. Some children also experience exploitation as they are enlisted by a parent in drug deals or other aspects of a lifestyle involving illegal substance activities.

Among the physical, developmental, emotional and other harms resulting from child maltreatment, early drinking onset (preteen) was identified as three times more common among adolescents who had experienced one or more types of maltreatment, and 54% of adolescents who had experienced sexual abuse engaged in substance misuse (Bailey & Stewart, 2014). The potential child maltreatment risks and harms are not equal across different types of substances; they differ somewhat by type of substance and how substances are misused (Kepple & Freisthler, 2020). It is important for anyone working in the field of substance-related treatment to learn how to recognize signs of child maltreatment, including neglect, and understand what they are legally obligated to do when it is suspected (i.e. mandated reporting policy and procedures).

## Employment Concerns



Heavy and frequent alcohol or other substance misuse prior to or during work hours poses significant employment problems, including absenteeism and frequent job changes (Bush & Lipari, 2015), as well as the potential for workplace accidents and

injuries. In the U.S., the vast majority (70%) of individuals who engage in heavy drinking or other substance misuse are employed full time; however, this information does not inform us about the proportion of individuals in the workforce who engage in these addictive behaviors (Frone, 2012). Based on various national surveys, Frone (2012) concluded that 8.5% to 9.3% of workers frequently engaged in heavy drinking, 4.6 million workers (3.6%) frequently drank to intoxication, and 9.3% of the workforce (12 million workers) may experience an alcohol use disorder. Additionally, 81 million workers (6.3%) frequently engaged in illicit substance use, 3.5% of the workforce (4.5 million workers) frequently were intoxicated from use of an illicit substance, and 2.0% of the workforce (2.6 million workers) may experience a substance use disorder involving illicit substances. Cannabis/marijuana was the illicit substance most commonly used. More difficult to determine is the prevalence of alcohol or other substance misuse just prior to working or during the work period; Frone (2012) concluded that 13 million workers (over 10%) experienced impairment in the workplace from alcohol intoxication or (most frequently) hangover, and 1 million of them did so frequently. Additionally, almost 2 million workers (1.5%) frequently used illicit substances within 2 hours prior to work and 1.8 million (1.4%) frequently used illicit drugs during work.

These figures are inconsistently distributed across job types. In

2008-2012 NSDUH data, Bush and Lipari (2015) indicated that past month heavy alcohol use was reported at the highest rates by workers in mining (17.5%) and construction (16.5%) industries, followed by accommodations and food services workers (11.8%), and those in arts, entertainment, and recreation (11.5%); the rate was lowest among those working in health care and social assistance (4.4%) or educational services (4.7%). Past month use of illicit substances was greatest among workers in accommodations and food service industries (19.1%) or arts, entertainment, and recreation (13.7%); the rate was lowest among workers in public administration (4.3%), educational services (4.8%), mining (5.0%), or health and social assistance (5.5%). Not surprising, based on these patterns, past year substance use disorder was greatest among workers in accommodations and food services (16.9%), construction (14.3%), arts/entertainment/recreation (12.9%), and mining (11.8%).

The issue of substance misuse and employment has two important sides: protecting the workplace and protecting the worker. To the first, many employers and companies have posted policies concerning alcohol or other substance use, whether including nicotine or



not—zero tolerance policies, for example. This approach may be combined with drug testing policies and programs of testing, as well as an employee assistance program (EAP) or services made available to individual employees experiencing substance-related (or other mental/behavioral health) problems (Hall, 2020). A few barriers to implementing these strategies exist, especially for small businesses: the cost of drug testing materials, questionable accuracy of the tests used, legal concerns related to drug searches, privacy issues related to use of EAP services, and costs of providing EAP services. While these issues are often thought about in relation to manufacturing and retail settings, they should be considered in terms of

workplaces where an individual is responsible for driving (or flying), for the welfare of children or adults needing care, carrying weapons, and making decisions that affect others' health, safety, and well-being.

On the employee side, substance misuse may contribute to loss of employment, unemployment, and underemployment. Among persons who were unemployed in 2011, past month use of illicit substances was reported by about 17%: a rate more than double what it was among persons employed full time and almost double the rate among persons employed part time (Badel & Greaney, 2013). Marijuana was, by far, the most common (15% of unemployed persons) and illegally acquired pain relievers were second (just under 5% of unemployed persons). Whether the unemployment was caused by or led to substance misuse is difficult to unravel; it is also possible that other factors contributed to both (e.g. the Great Recession having a greater impact on a class of workers who already engaged in substance misuse at a greater rate).

## Sex Work, Human Trafficking, and Exploitation

“A commonly held view is that women engage in street-level prostitution to obtain money to buy drugs or in direct exchange for drugs. Indeed, existing research demonstrates a strong association between substance abuse and prostitution...The substances that women in prostitution most commonly abuse are heroin, cocaine (crack), marijuana, and alcohol” (Wiechelt & Shdaimah, 2011, p. 161). Substance misuse may precede involvement in prostitution, to fund their substance use and the “degradation and trauma involved in prostitution” may worsen a pre-existing substance use disorder (Wiechelt & Shdaimah, 2011, p. 162). On the other hand, substance misuse may begin after initiation of prostitution, as alcohol and other substance use “appear to make women in prostitution feel more confident and able to manage their interactions with their



“customers” as well as provide them with a mechanism for coping with or numbing their negative feelings” (Wiechelt & Shdaimah, 2011, p. 161). Either model alone is insufficient as they “fail to capture the cascade of factors” involved in the co-occurrence of sex work and substance misuse (Wiechelt & Shdaimah, 2011, p. 162).



Women, men, children, and adolescents engaging in sex work seldom do so voluntarily—typically, a high degree of coercion and abuse is involved. Globally, human trafficking occurs as forced work in agriculture, fishing, manufacturing, mining, forestry, construction, domestic/cleaning, and hospitality service labor sectors, with others forced to serve as beggars, soldiers, “wives” and sex workers (WHO, 2012). This is happening around the world to female and male children, adolescents, and adults. The United Nations defined human trafficking is as follows (WHO, 2012):

*[T]he recruitment, transportation, transfer, harbouring or receipt of persons, by means of the threat or use of force or other forms of coercion, or abduction, or fraud, or deception, of the abuse of power or of a position of vulnerability or of the giving or receiving of payments or benefits to achieve the*

*consent of a person having control over another person, for the purpose of exploitation.*

Exploitation is a key concept involved in the intersection between substance misuse and human trafficking for sex/sex work. Because human trafficking and sex work are generally illegal, they are somewhat invisible in nature, making it difficult to generate accurate data on prevalence. The invisibility also contributes to difficulties in providing for individuals who are trafficked—ensuring that basic needs are adequately met, much less their health, mental health, and protection needs. These children, women, and men:

*“...may encounter psychological, physical and/or sexual abuse; forced or coerced use of drugs or alcohol; social restrictions and emotional manipulation; economic exploitation, inescapable debts; and legal insecurities...Risks often persist even after a person is released from the trafficking situation, and only a small proportion of people reach post-trafficking services or receive any financial or other compensation” (WHO, 2012).*

Alcohol and other drugs are frequently involved with human trafficking—either used to control a person already experiencing substance use disorder, used to coerce someone to initiate use, and/or used to cope with the physical and psychological traumas associated with trafficking (WHO, 2012). Anecdotal cases exist where family members allowed the sexual exploitation/human trafficking of their children by others in exchange for drugs or drug money (Keely, 2020). Among over 23,000 callers to the National Human Trafficking Hotline and Resource Center, substance use was one of the top five risk factors for falling prey to traffickers ([https://humantraffickinghotline.org/sites/default/files/Polaris\\_National\\_Hotline\\_2018\\_Statistics\\_Fact\\_Sheet.pdf](https://humantraffickinghotline.org/sites/default/files/Polaris_National_Hotline_2018_Statistics_Fact_Sheet.pdf)). In the state of Ohio, induced substance abuse was a significant control method reported to the national hotline, and discovered through criminal investigations, drugs were frequently used as a means of

coercion (Roberson, n.d.). Coercion efforts used to compel victims included threatening to call victims' probation officer to report their drug use, creating a debt (fictitious), threats or anticipation of withdrawal sickness, and payment for "services" in drugs—in other words, exploiting a person's addiction.

Recovery advocates recommend supportive services for victims of human trafficking, rather than imposing criminal justice system sanctions. In a qualitative arts-based study of women engaged in prostitution, investigators concluded: "A combination of systemic violence, poverty, ill health, addiction, and prostitution shaped the lives and circumstances of the women in our sample, all of whom described a longing for basic health, security, belonging, and stability" (Shdaimah & Wiechelt, 2017, p. 362). They reported that women "engaged largely in what is often called "survival" sex work; their motivation for engaging in prostitution is to meet their basic needs of shelter, clothing, food, and, often, addiction" (Shdaimah & Wiechelt, 2017, p. 363). Trauma-based and trauma-informed care is in order (Shdaimah & Wiechelt, 2011), potentially delivered within the structure of diversionary treatment-based and specialty drug court programs rather than through traditional criminal justice punitive responses (Begun & Clark-Hammond, 2012).

## Housing Insecurity



That alcohol or other substance misuse leads to homelessness has been a long-standing stereotype in the U.S., much of which has an underlying morality theme. However, evidence more accurately suggests that the causal relationships are much more complex, particular in terms of multiple co-occurring phenomena being inter-twined: housing instability/insecurity,

mental health issues, physical health issues, intimate partner/family violence, economics of poverty, discrimination, and alcohol or other substance misuse (Pynoos, Schafer, & Hartman, 2017). Recognizing that homelessness is a dynamic state, the issue is more appropriately considered under the rubric of housing instability/stability and security over time. Housing stability, with reliable and safe shelter, is important in providing substance-related and recovery support services over time, services that persist across transitions in how/where a person is living. Not only does housing stability affect a person's engagement in substance-related intervention, substance misuse may interfere with a person's housing stability. For example, public housing services may have policies that preclude a person engaged in substance misuse from housing assistance eligibility, and landlords or family/friends may refuse to house someone who is engaging in substance misuse.

The Substance Abuse and Mental Health Services Administration (SAMHSA) recommends engaging in community-based service responses that place housing at the top of the service provision priority list and deliver Permanent Supportive Housing, Housing First, and Recovery Housing service strategies (SAMHSA, 2019b). The goal is to help meet individuals' recovery support needs with stable housing where case management and wraparound care are easily accessible in substance-free environments involving peer support and other recovery support.

## **Incarceration and Other Criminal Justice Involvement**

Much of what surrounds alcohol and other substance misuse involves illegal behaviors, thus there exists a high degree of co-occurrence between substance misuse and criminal justice system involvement—including courts, incarceration in jail, prison, and correctional treatment settings. Criminal activities may include

possession and/or distribution offenses, offenses committed in order to obtain substances, and offenses committed while intoxicated or in withdrawal from substance misuse. For these reasons, substance misuse is termed a **criminogenic factor**, meaning that it contributes to the rate at which criminal offenses are committed. According to a Bureau of Justice Statistics report (Sawyer, 2017), in the U.S. between 2007-2009:

- 21% of persons sentenced to state prisons and local jails were incarcerated for crimes committed to obtain drugs or money for drugs;
- 40% of individuals incarcerated for property crimes committed those crimes for drug-related reasons;
- 14% of individuals incarcerated for violent crimes committed those crimes for drug-related reasons;
- about 40% of sentenced incarcerated persons reported using drugs at the time of the offense for which they were in jail or state prison;
- more than 50% of persons in state prisons and 66% sentenced to jail experienced substance use disorders;
- only about 25% of incarcerated individuals experiencing substance use disorders received substance-related treatment while in jail or state prison.

Others report that an estimated 75% of jail and prison inmates need intervention for substance misuse or substance use disorder, but only about 11% receive these services during their period of incarceration (Pettus-Davis & Epperson,



2015). It is of great concern that a population in great need lacks access to evidence-supported substance-related interventions during incarceration and as they transition to community living

post-release (Begun, Rose, & LeBel, 2011). While the majority of incarcerated adults are men, the statistics concerning substance-involvement and women warrant attention as they do paint a somewhat more dire picture: an estimated 20% of incarcerated women meet criteria for alcohol use disorder and 51% for substance use disorder; this is compared to 30% of incarcerated men (Rose & LeBel, 2020).

1

Why does all of this matter? First, it suggests that incarceration may provide particularly salient “teachable moments” related to substance misuse. In a randomized control study conducted with women in jail preparing for community reentry, investigators reported a significantly greater reduction in post-release alcohol and other drug use scores among women provided with screening and brief intervention near the end of their incarceration compared to women receiving treatment as usual—which meant little to no intervention preparing them to address their problems with alcohol and other substances (Begun, Rose, & LeBel, 2011). Surprisingly, this difference in improvement was not attributed to post-release treatment engagement as many women in both groups were unable to access substance-related treatment; the treatment preparation alone was a meaningful intervention.

2

Second, it is critical to ensure strong transitional support as a person is released to the community, supporting them in maintaining recovery gains out in the community. This transition can be extremely difficult for individuals to manage as there are often problems with gaining secure housing, becoming employed in work that provides a sustaining wage, and access to needed health, mental health, and addiction services. In the previously described study involving women released from jail, the most common barriers to utilizing alcohol or other drug treatment services as: perceiving that treatment programs were full, inability to pay for treatment/no health insurance coverage for treatment, and a lack of transportation to treatment (Begun, Rose, & LeBel, 2011). A pre-

/post-release study involving men and women incarcerated in Ohio's jails, state prisons, and community based correctional facilities (CBCFs, treatment providing residential programs during final months of incarceration) reported that 44% of individuals experienced the need for services to help them address their problems with substance misuse and the most significant barrier to receiving such services was inability to pay (Begun, Early, & Hodge, 2016). Substance misuse is a major driving factor in the return to incarceration, suggesting that a failure to address the problem initially has long-lasting implications for recurrence and re-offending (Rose & LeBel, 2020).

### 3

Third, addressing substance-related problems, and especially changing the criminal justice response to non-violent substance-related offending are consistent themes in a “smart decarceration” movement (Pettus-Davis & Epperson, 2015; Epperson & Pettus-Davis, 2017). As you learned in the beginning of our course, the War on Drugs has driven a great deal of policy concerning responses to substance use/misuse across the nation. Smart decarceration is about reversing the nation's nearly 40-year mass incarceration trend, as it has proven to be unsustainable and harmful to individuals, families, communities, and society alike, as well as perpetuating historical class and race disparities (Epperson & Pettus-Davis, 2017)). While you might be thinking that failing to stop drug trafficking through criminal justice responses would be unwise, it is important to recognize that globally, among individuals serving sentences for drug offenses, over 80% are serving sentences involving drug possession for personal use (Rose & LeBel, 2020). The introduction of drug courts, beginning in 1989, represents an evidence-supported, integrated criminal justice system and drug treatment response (Lloyd & Fendrich, 2020). Drug courts offer as mandated treatment involvement with close case monitoring as an alternative to incarceration (Lloyd & Fendrich, 2020). Thus, evidence-supported

and evidence-based interventions do exist for supporting recovery from substance misuse/use disorder among men and women while incarcerated and during community reentry; policy and funding changes are needed to more fully support their implementation (Epperson & Pettus-Davis, 2017; Rose & LeBel, 2020).



### **STOP & THINK**

**A Perfect Storm:** After completing these two chapters concerned with co-occurring mental, physical, and situational challenges, think about the following situation and its implications for understanding and intervening around substance misuse. In late 2019 and early 2020 the world awoke to a global pandemic: COVID-19 is a potentially serious, even deadly, disease communicated through saliva or “droplets” containing a corona virus. The population of individuals engaged in substance misuse and experiencing substance use disorders may be at particularly high risk for exposure to the virus, contracting the COVID-19 disease, and having poor outcomes if they do become ill.

Based on what you read in these chapters, what might you expect in terms of the following and why?

- Disease transmission among individuals who engage in “party”



or other group alcohol or cannabis consumption.

- Likelihood of successful “social distancing” and disease containment within communities of individuals engaging in substance misuse—consider mental status, health disparities, behavior, life circumstances on a broad level (e.g. what kind of work they may be engaged in, housing, incarceration, and more of the topics discussed in chapter 2).
- Recovery support when health and mental health providers were scrambling to adapt to the pandemic and unstable/unreliable availability of many medications (including those used in pharmacotherapy for mental and substance use disorders).
- Likelihood of more severe outcomes from infection with the COVID-19 virus (e.g., consider the range of physical health concerns explored in chapter 1).

What would you have recommended to public health officials and human services providers with regard to this corona virus/ COVID-19 pandemic among members of these communities?

## Module 13 Summary

In this module you explored a host of issues, concerns, and problems that commonly co-occur with substance misuse and substance use disorder. First, we looked into diagnosable comorbid conditions of mind and body. The mental disorders reviewed include anxiety, depression, bipolar disorder, schizophrenia, dementia, personality disorders, impulse disorder/ADD/ADHD, post-traumatic stress disorder (PTSD), and gambling disorder. Next, we turned our attention to the physical conditions of concern, including both communicable and non-communicable diseases, as well as traumatic brain and other injuries. That section also included content related to driving under the influence of alcohol and other substances. The following chapter explored some situational

contexts that often co-occur with substance misuse: violence (including intimate partner violence, child maltreatment, and sexual assault), employment and unemployment concerns, sex work/human trafficking and related exploitation, housing insecurity, and criminal justice involvement or incarceration in jails or prison.

A common theme throughout this module is the complex nature of the relationships between co-occurring phenomena—that one may cause the other, the other way around may happen, both may be caused by a third factor, or they may interact in an iterative manner over time. Nonetheless, co-occurring problems do have an effect on severity, outcomes, and intervention. In most cases, it is recommended that screening and assessment include their consideration, and that they may best be treated in an integrative approach rather than either sequentially or in a disconnected concurrent manner. At this point, you are prepared to review key terms introduced in this module, and to proceed to the course conclusion chapter.

# Ch. 3: Course Summary

As we conclude our final course module, let's take the chance to briefly review the vast array of information shared and learned, then identify some ideas for continuing to develop knowledge and skills for working in the substance use, substance misuse, and substance use disorders arena.

## Course Overview

The original purpose of this course, evidenced by the title “Theories and Biological Basis of Substance Misuse,” was to explore the theories underlying our understanding of and intervention around substance misuse and to develop a basic understanding of some of the issues and specific substances commonly involved. Some of what you learned along the way included:

- ethical and professional uses of person-first language related to substance use, misuse, and use disorders, and avoiding casual overuse of labeling language and words
- how substance misuse and use disorders are defined and what recovery means
- how different types of substances are classified
- biological theories (basic neurobiology and neurotransmitters, genetics, and pharmacokinetics)
- psychological theories (cognition, information processing, learning, social learning, psychodynamics, expectancies, and cravings)
- social context and environments at the micro-, meso-, and macro-levels, including family, peers, school/workplace, neighborhood/community, and policy
- social norms, stigma, and social theories

- how an individual's substance misuse affects others in the social and physical environment
- integrating theories into a biopsychosocial framework that can inform prevention and the transtheoretical model of behavior change (TMBC)
- alcohol
- sedative-hypnotics and CNS depressants
- cannabis and various hallucinogenic substances
- stimulants, including amphetamines, methamphetamine, cocaine, caffeine, and nicotine
- opioids
- inhalants
- anabolic steroids
- over-the-counter and prescription drug misuse
- mixing alcohol and other drugs, as well as polydrug misuse
- pharmacotherapy approaches
- detoxification
- co-occurring mental and physical conditions, and other situational challenges.

This information represents a strong foundation for understanding and making a difference in substance use, substance misuse, and substance use disorder.

## Future Directions

What comes next? For some, this degree of being informed is sufficiently satisfying. Others may wish to pursue further knowledge and skills. You now have the foundation for future study concerning topics related to:

- screening for substance misuse and related or co-occurring problems

- assessment and diagnosis of substance use disorders
- intervention planning
- delivering and implementing interventions, recovery services
- prevention planning and intervention
- evaluating intervention
- policy related to substance misuse and recovery services

Whether this is the end of study for you (for now) or you plan to continue on in this arena of study and work, take pride in all that you have mastered through this course.

# Module 13: Key Terms

**alcohol myopia theory** suggests that many alcohol-related behaviors are the consequence of alcohol's effect of narrowing perceptual and distorting cognitive functioning.

**comorbidity** refers to having two or more diagnosable conditions either at the same time or in close sequence.

**concomitant** means two or more events that occur together or in close sequence.

**co-occurring problems** refers to two or more difficult or challenging concerns, conditions, or events that happen either at the same time or within close proximity in time.

**criminogenic factor** refers to variables, events, or conditions that are known (based on evidence) to contribute to the probability a person or persons will engage in criminal activity/commit criminal offenses.

**disinhibition** is a process where a person's learned inhibitions against behaving in a certain way are themselves inhibited or suppressed, resulting in an increased probability of that behavior being exhibited.

**driving under the influence (DUI)** refers to the criminal offense of operating a vehicle while impaired or intoxicated by alcohol or other substances.

**dual diagnosis** refers to having two diagnosable (mental) disorders at the same time or within close proximity in time.

**expectancy theory** refers to how a person's beliefs about the likely effects of alcohol or other substance use influences decisions about using the substance(s).

**intimate partner violence (IPV)** refers to the threat or expression of violence toward a person in a present or past (ex-) close couple relationship; the violence may be physical, emotional, sexual, economic, or other forms of exerted control, and may (also or

instead) be directed to children, other loved ones, or pets with the specific intent of affecting the intimate partner.

**post-traumatic stress disorder (PTSD)** is a diagnosable condition, meeting specific criteria, following one or more life- or limb-threatening event or one which is otherwise seriously life altering.

**Wernicke-Korsakoff syndrome** is a diagnosable neurological condition resulting from a history of chronic heavy alcohol misuse.

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