Theories and Biological Basis of Substance Misuse, Part 2

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AUDREY BEGUN

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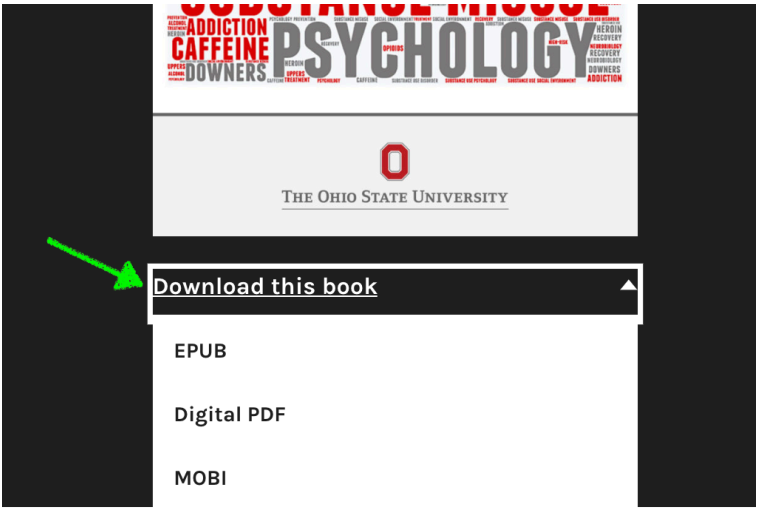
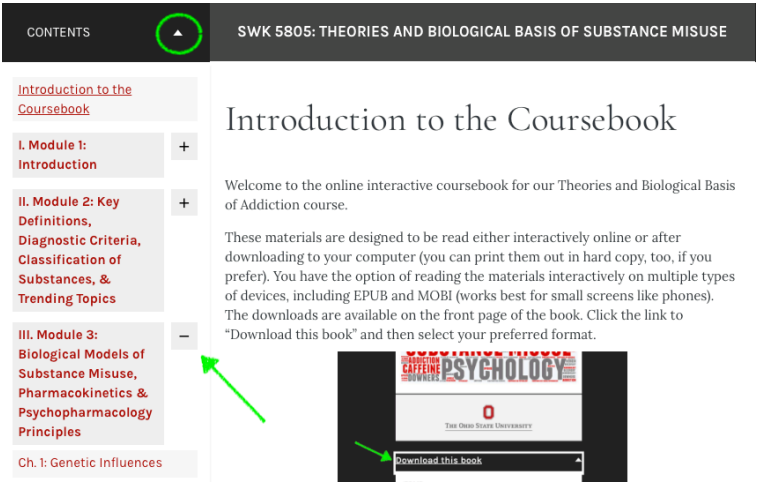
Introduction to the Coursebook

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.



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 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.

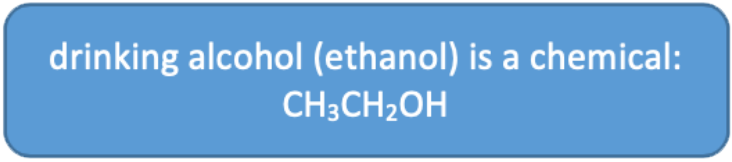
Ch. 1: The Nature and Effects of Alcohol

"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 (ethanolor "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.

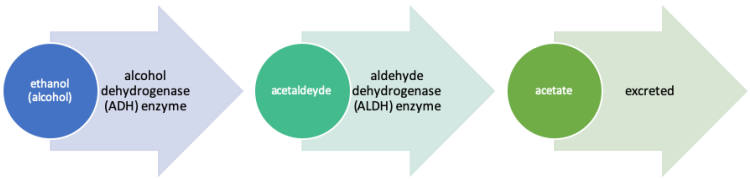


Although alcohol is most often consumed in beverages, it can be administered intravenously as a medical intervention in an emergency response to acute alcohol withdrawal syndrome (which can be fatal). This mode of administration is highly discouraged by the lay person—it takes much less alcohol to result 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.

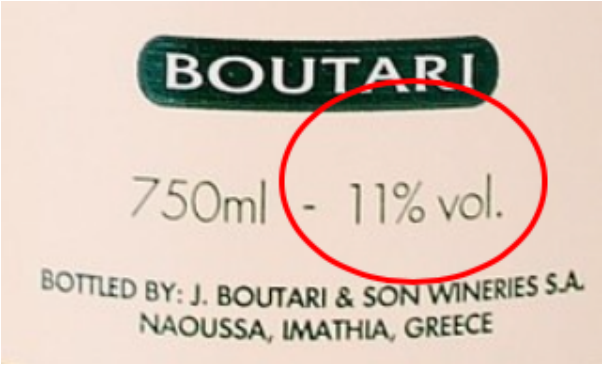


Human genetics play a significant role in directing the control and production of the metabolizing enzymes (ADH and ALDH), reflecting one mechanism in individual differences in alcohol responses. You may recall from our genetic module how some individuals experience a very negative (punishing) "flushing" 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^{O} 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.

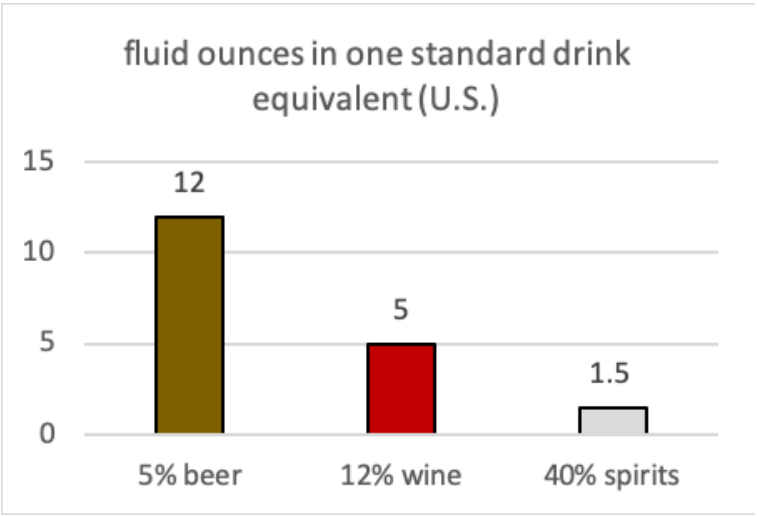


Standard drink measurement. If a person indicates that they consumed one drink, do we really know how much alcohol they consumed? "One drink" could mean very different things under different circumstances. Consider, for instance, the differences in calories consumed in a soft drink. A 12 oz can of Coca-Cola® 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 x 40 x .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 x 12 x .042 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^{th} grade have significantly greater odds of drinking full drinks, getting drunk, and drinking heavily by the time they are in 9^{th} 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)*** 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 21st 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
180 #	180 #	180 #	140 #	180 #	140 #
5 drinks	5 drinks	5 drinks	5 drinks	2 drinks	2 drinks
2 hours	2 hours	4 hours	4 hours	2 hours	2 hours
BAC estimate: 0.11%	BAC estimate: 0.14%	BAC estimate: 0.08%	BAC estimate: 0.15%	BAC estimate: 0.03%	BAC estimat 0.05%

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.

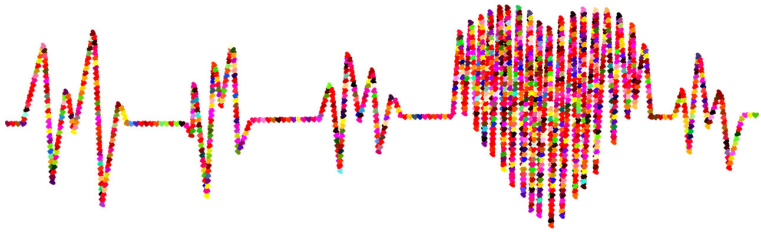
Not only is alcohol implicated in distortions of perception and memory in information processing, it also can impair decision-making and judgment. A significant literature indicates that drinking alcohol impairs a person's ability to accurately assess risk or feel appropriate anxiety in potentially risk 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).

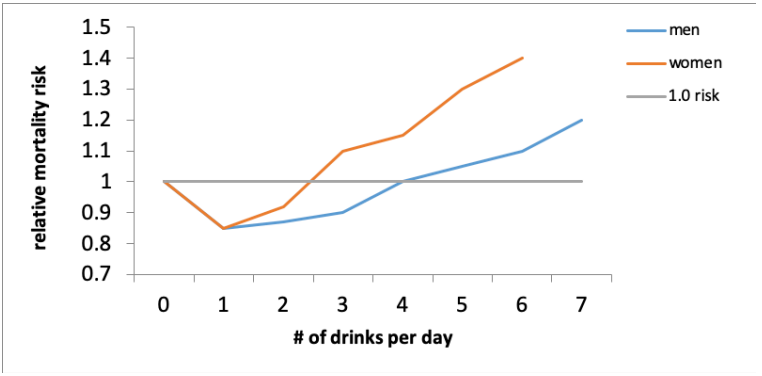


On the other hand, there exists ample evidence that heavy drinking is the enemy of cardiac health. Similarly, heavy alcohol consumption deprives the body of calcium and interferes with vitamin D activation, both of which contribute to bone/skeletal system health. While this can contribute to osteoporosis 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.

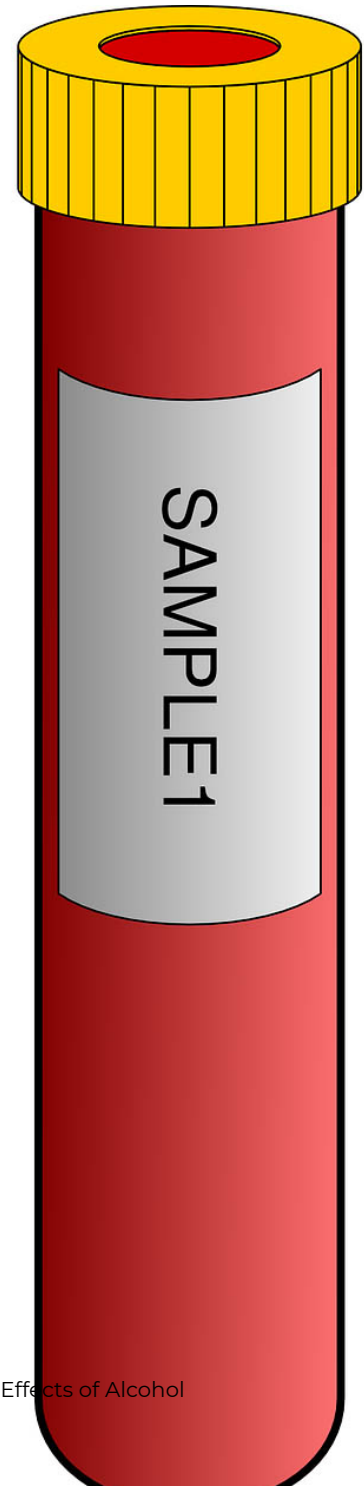
This graph depicts data presented by O'Keefe, Bybee, and Lavie (2007) concerning the relative risk of death (all causes) in relation to daily alcohol consumption levels. The "neutral" point is a risk value of 1.0 for no daily drinking. The relative risk dropped a bit with one drink per day for both men 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.


SAMPLE1

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.

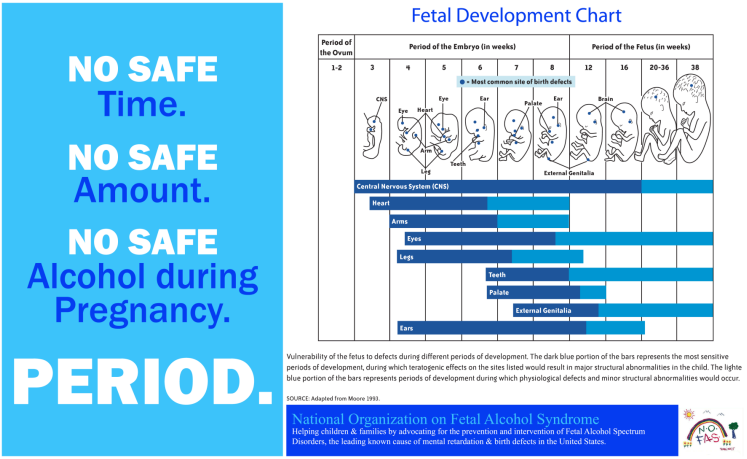
Fetal Alcohol Exposure

"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*** (Loock, 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 (Loock, 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 or media element has been excluded from this version of the text. You can view it online here:

https://ohiostate.pressbooks.pub/substancemisusepart2/?p=288

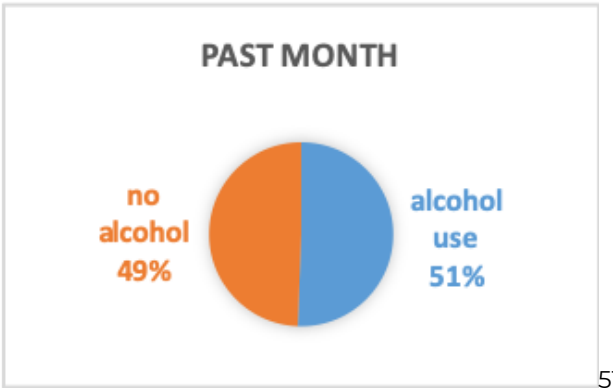
Ch. 2: Alcohol Epidemiology & Treatment Needs

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.

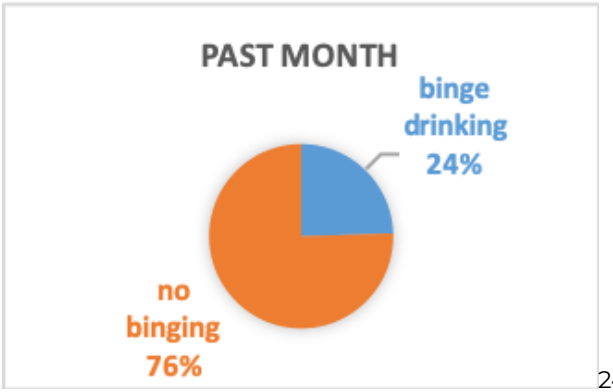
Here are recent facts and figures for your consideration, derived from the 2018 NSDUH survey data concerning individuals aged 12 and older in the U.S. (SAMHSA, 2019).

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:

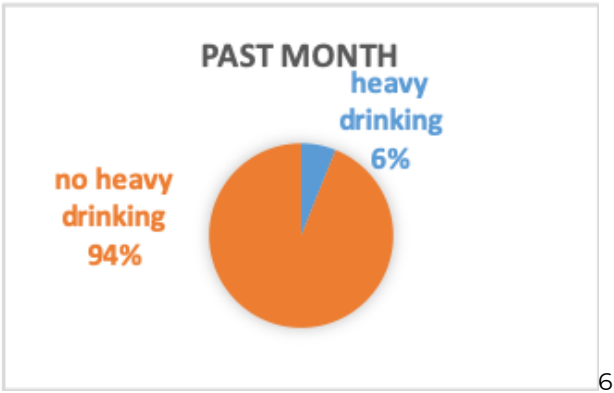
- 

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	60.8%	31.5%	9.2%
female	52.4%	22.1%	4.4%
Race/Ethnicity:			
White	61.1%	27.2%	7.7%
Black or African American	49.0%	26.3%	5.0%
Asian	43.9%	16.0%	2.9%
Hispanic or Latino	48.2%	28.4%	5.0%
American Indian or Alaskan Native	39.7%	26.8%	5.9%
Native Hawaiian or Other Pacific Islander	38.5%	26.8%	6.6%
2 or more races	53.7%		
Age:			
12-17	9.0%	4.7%	0.5%
18-25	55.1%	34.9%	9.0%
26 and older	55.3%	25.1%	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?

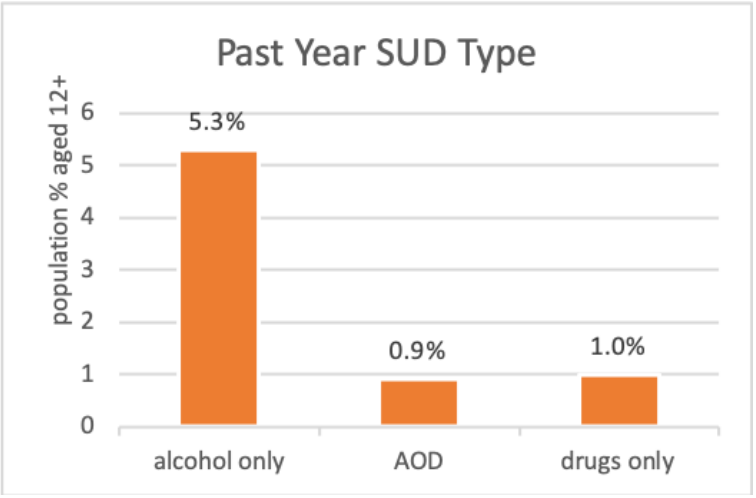
- How do you think the data relate to stereotypes and stigma in our society and policies?

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 II

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:

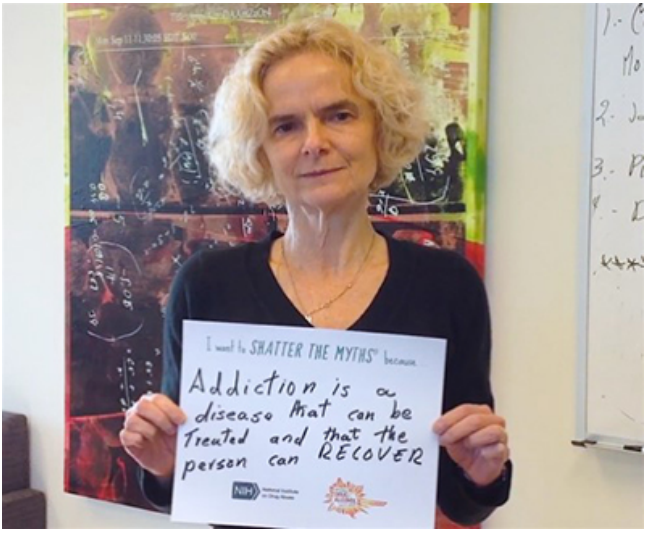
- Describe the nature of sedative-hypnotic and CNS depressant drugs and their effects mind, body, behavior and health;
- Describe how these types of drugs might be used and/or

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

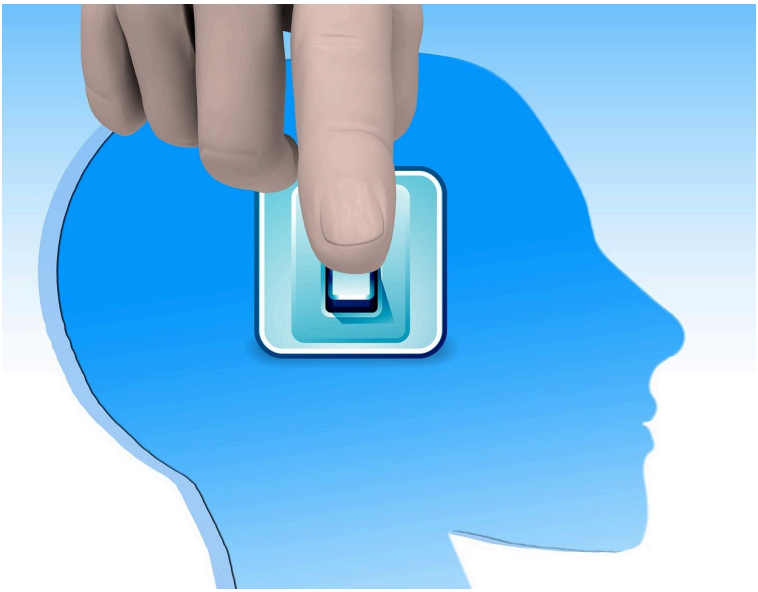
stress reactions, and muscle spasms, include drugs such as benzodiazepines (e.g., Valium, Xanax) and barbiturates (e.g., phenobarbital)—which are sometimes prescribed for seizure disorders. Most CNS depressants act on the brain by affecting the neurotransmitter gamma-Aminobutyric acid (GABA), which works 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). 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®,

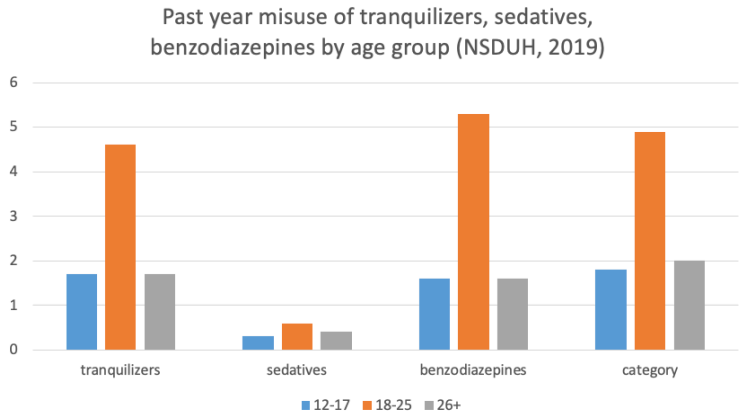
triazolam/Halcion®, estazolam/Prosom®/, chlorodiazepoxide/Librium®) [street names include: candy, downers, tranks/tranqs] [note that "bennies" are not benzodiazepines, they refer to a brand of amphetamine] [other names for Xanax®: bicycle parts or bicycle handle bars; for Konopin®: benzos, K, K-Pin, 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/Lumninal®, pentobarbitol sodium/ Nembutal®, amobarbital/Amytal®, butabarbital/Butisol®) [street names include: barbs, phennies, reds, yellows, yellow jackets]
- ***non-benzodiazipinesedative 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):

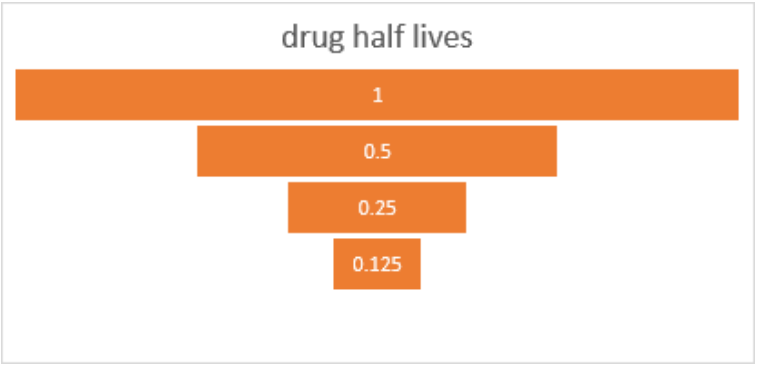
- slurred speech
- poor concentration
- confusion
- memory problems
- headache
- light-headedness/dizziness
- dry mouth
- uncoordinated movements
- low blood pressure
- slowed breathing rate.

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).

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

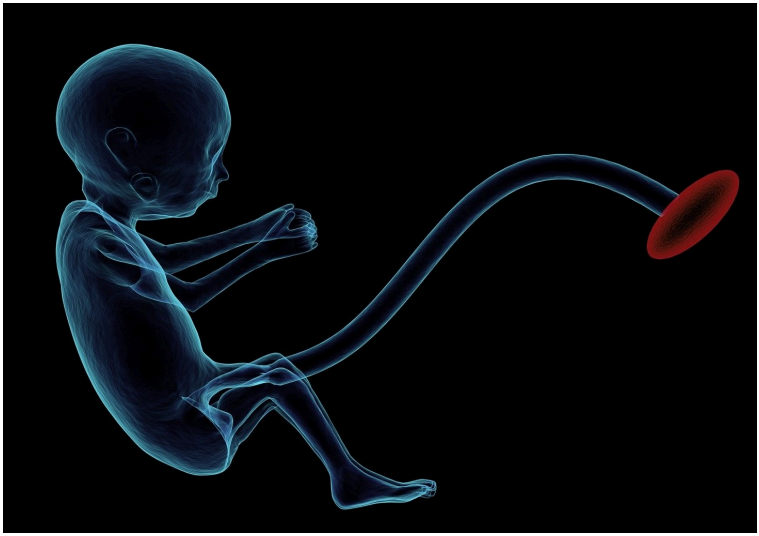
- seizures
- anxiety/agitation
- insomnia
- overly active reflexes, shakiness
- increased heart rate
- increased blood pressure
- increased body temperature
- hallucinations.

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).



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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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https://www.samhsa.gov/data/report/2018-nsduh-detailed-tables

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 III

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.rehabs.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 indica*plant (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

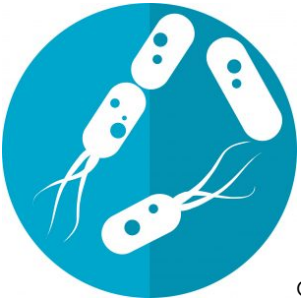
emerged regarding vaping the psychoactive ingredients of cannabis. A warning released on October 4, 2019 stated:

*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.

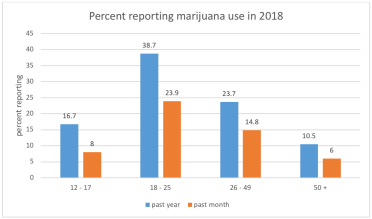


Cannabis products are most often used alone; however, individuals may intentionally or unintentionally (laced) use them in conjunction with other substances such as alcohol, cocaine, or heroin. The combination of alcohol and cannabis products has a ***potentiating effect***, meaning that the effects 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

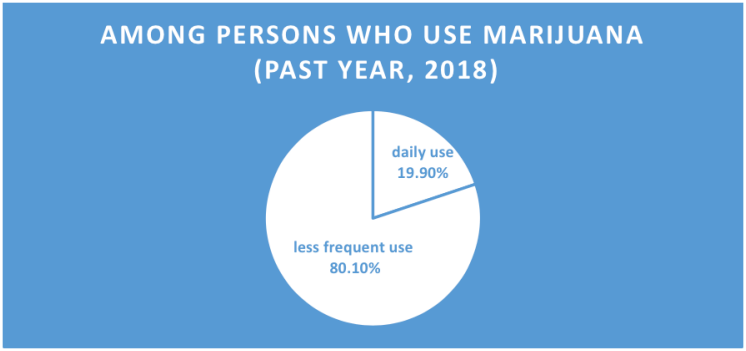
the past year, and 10.1% during the past month. This graph depicts past year and past month use rates by age category: the rate is greatest among emerging adults aged 18-25. Still, marijuana use is not the "norm" in any of the age categories since many fewer than half engaged in this behavior.



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).



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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).

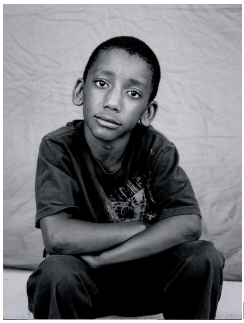
This phenomenon is sometimes referred to as ***telescoping***—the rate at which problems appear is collapsed into a shorter time frame.

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 varietals 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 repellant, 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.

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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

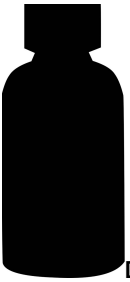
be good, bad, or neutral quality memories—the problem being that a person cannot control or predict which will be experienced this way. Or, the experience can involve a sense of movement through space or time—flying, soaring, floating, being pulled along, or otherwise moving—when in reality, no such 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 divonorum* (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).



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 heartrate, 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 is 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 is 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.

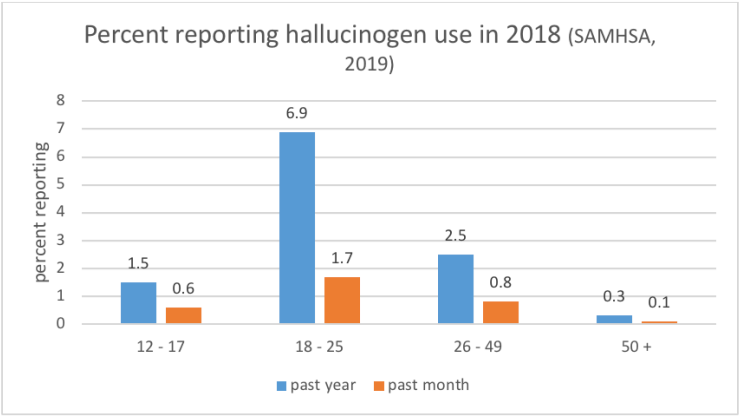
Psilocybin. Multiple species of fungi (mushrooms) contain the active ingredient ***psilocybin*** and/or psilocin, hence they are often called psychedelic or ***magic mushrooms***, they may also be referred to as mushies, sacred mushrooms, or zoomers. Because of the potential for abuse and the absence 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 divornum. ***Salvia divornum***'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 divornum 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.



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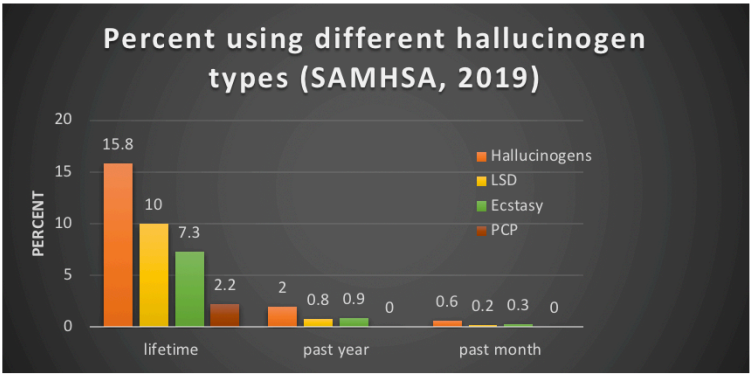
The rate of overdose deaths from hallucinogens is described by the DEA as "extremely rare" https://www.dea.gov/sites/default/files/sites/getsmartaboutdrugs.com/files/publications/DoA_2017Ed_Updated_6.16.17.pdf#page=84.However, the DEA reports that deaths *related* to their use do occur—typically from 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.



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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 divornum: 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.

Module 9: References and Image Credits

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PART IV

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.

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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.

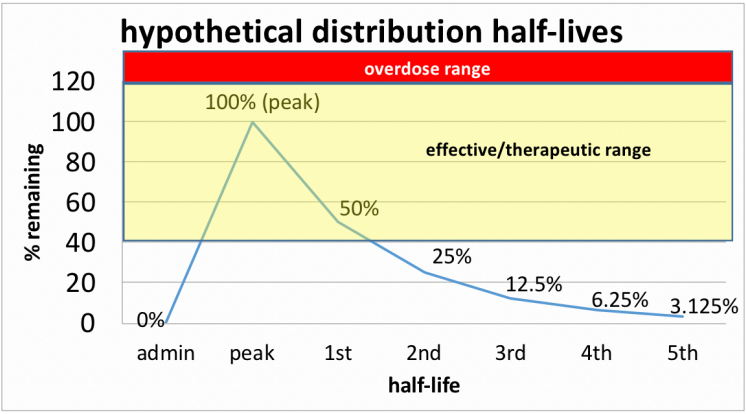
In addition to their psychoactive effects, stimulant substances affect multiple organ systems, thus they can have multiple side effects. In addition to mood swings, irritability, and paranoia, side effects of stimulant use might include:

- 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.



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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 dextroamphateamine (Dexedrine® and Adderall®) and methlphenidate (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 elevate 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.

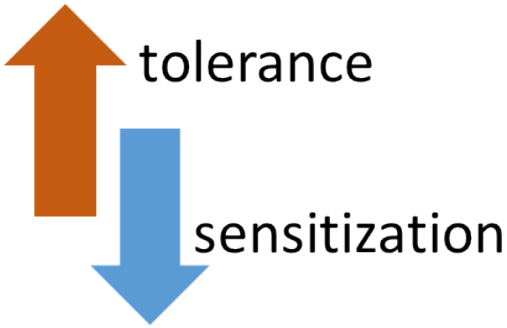
Methamphetamine.

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.

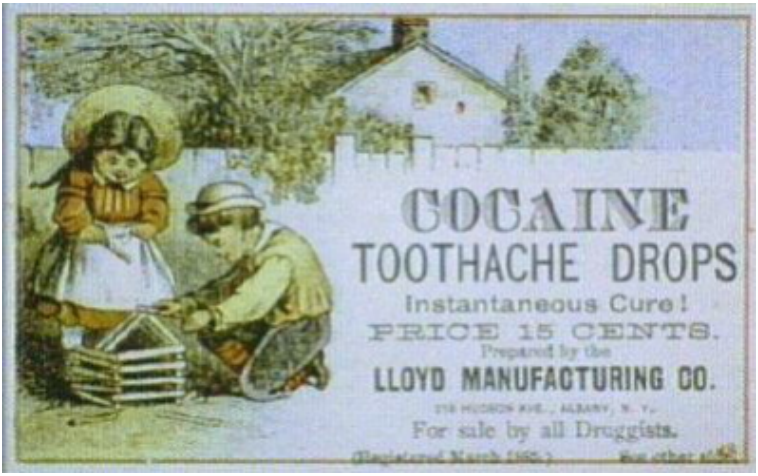
 *The chemicals used to produce methamphetamine are extremely hazardous. Some are highly volatile and may ignite or explode if mixed or stored improperly. Fire and explosion pose risk not only to the individuals producing the drug but also to anyone in the surrounding area, including children, neighbors, 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*, *laetervirens*, and *novogranatense* varients. (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).

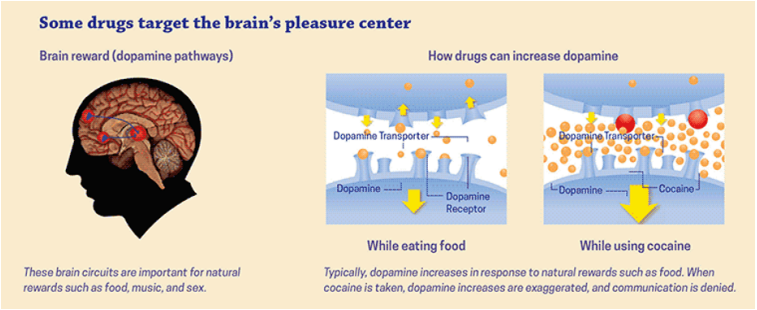
Common methods of cocaine administration lead to quick and intense psychoactive effects; it is usually "snorted," smoked, or injected, but may be rubbed on gums to be absorbed. As with many other potentially addictive substances we have studied in this course, the intensity of the dopamine (brain reward) 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 producs 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.oola.com/life-in-flavor/2308311/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 off-setting 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:[a]			
black[a]	60-90	8 ounces	7.5-11.25
oolong[a]	50-75	8 ounces	6.25-9.38
green[a]	35-70	8 ounces	4.38-8.75
white[a]	30-55	8 ounces	3.75-6.88
matcha[b]	70	1 teaspoon	—
decaffeinated[c]	2	8 ounces	0.25
caffeine-free herbal[c]	0	—	0
Coffee:[d]			
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:[e]			
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:[f]			
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[g]			
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[h]	500	1 ounce	500
Caffeine Gum[i]	40	1 piece	—
Powder[j]	3,200	1 teaspoon	—

Comparison Chart of Caffeine Content in Different Products

[a] retrieved from https://www.oola.com/life-in-flavor/2308311/which-tea-has-the-most-caffeine/

[b] retrieved from https://www.gotmatcha.com/matcha-and-caffeine/

[c] retrieved from https://www.cupandleaf.com/blog/pros-and-cons-of-drinking-decaf-tea

[d] retrieved from https://www.medicalnewstoday.com/articles/324986#caffeine-content-by-coffee-type

[e] retrieved from https://www.caffeineinformer.com/the-caffeine-database

[f] retrieved from https://www.caffeineinformer.com/energy-drinks-caffeine and https://spoonuniversity.com/lifestyle/caffeine-in-energy-drinks-ranked

[g] retrieved from https://greatist.com/eat/does-chocolate-have-caffeine#1

[h] retrieved from https://www.caffeineinformer.com/caffeine-content/5150-juice-caffeine-liquid

[i] retrieved from https://www.cnn.com/2013/04/30/health/caffeinated-gum/index.html

[j] 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) heartrate, 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).

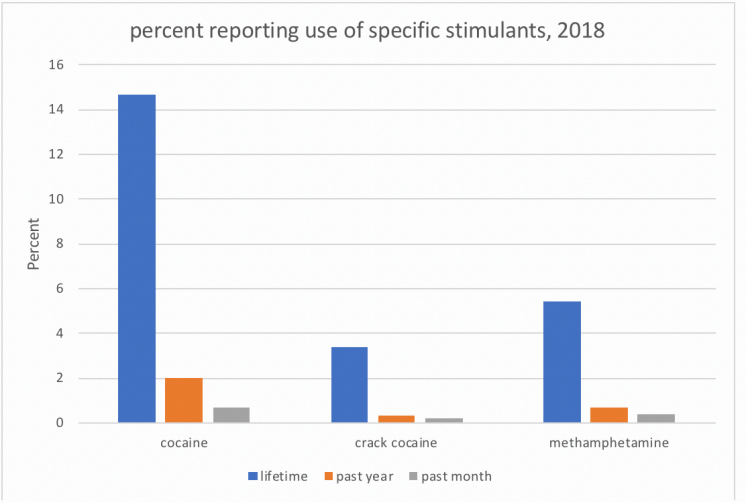
STOP & THINK

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

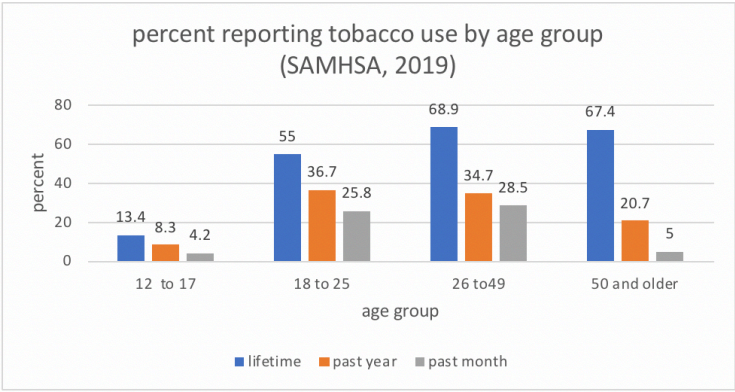
regularly consume caffeine (https://www.eatright.org/food/nutrition/healthy-eating/is-your-kid-over-caffeinated).They also reported that the U.S. Food and Drug Administration (FDA) does not produce recommended limits on caffeine for children (they suggest 400mg for adults), but the Canadian government 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.



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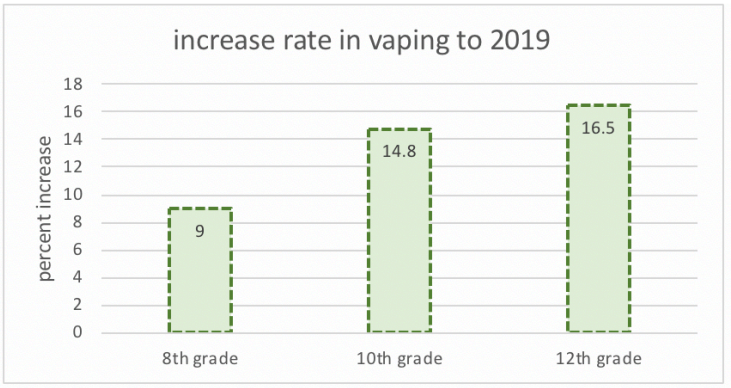
Like most of the other substances we have studied, men were more likely to report use of tobacco products than were women (69.2% versus 54.2% lifetime, 33.4% versus 20.5% past year, and 26.6% versus 16.6% past month). The race/ethnic groups reporting past month tobacco use at the highest rate self-identified 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 12thgraders: "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).



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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.

Module 10: References and Image Credits

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PART V

MODULE 11: 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 ***carfentanil***. 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:

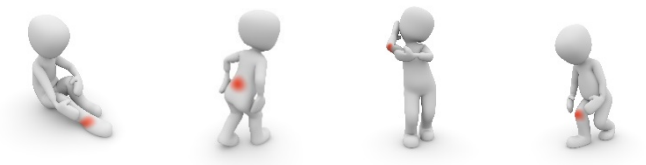
- oxycodone (OxyContin®, Percocet®)
- hydrocodone (Vicodin®)
- codeine (often combined with acetaminophen or with cough suppressants)
- morphine
- fentanyl, carfentanil, and other fentanyl analogs

Narcotics

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:

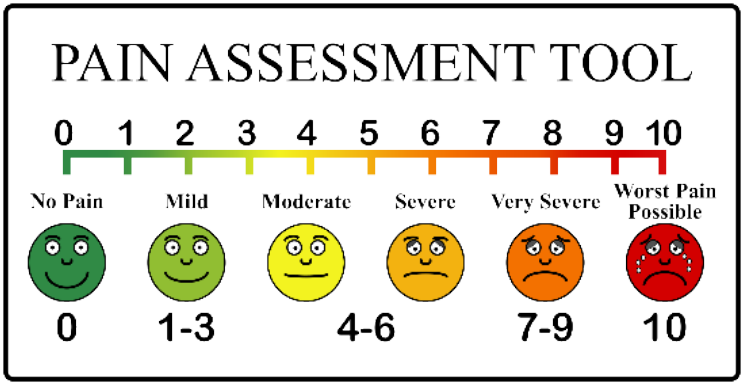
- mild OUD (2-3 symptoms),
- moderate OUD (4-5 symptoms), or
- severe OUD (6 or more symptoms).

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.

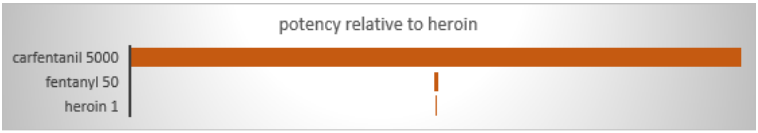
Mode of administration. Depending on the mode of administration, opioid misuse may increase the risk of infection and infectious disease exposure. Since many individuals engage in injection use of these substances, this is of significant concern and worthy of harm reduction attention. Heroin ("horse," "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*).

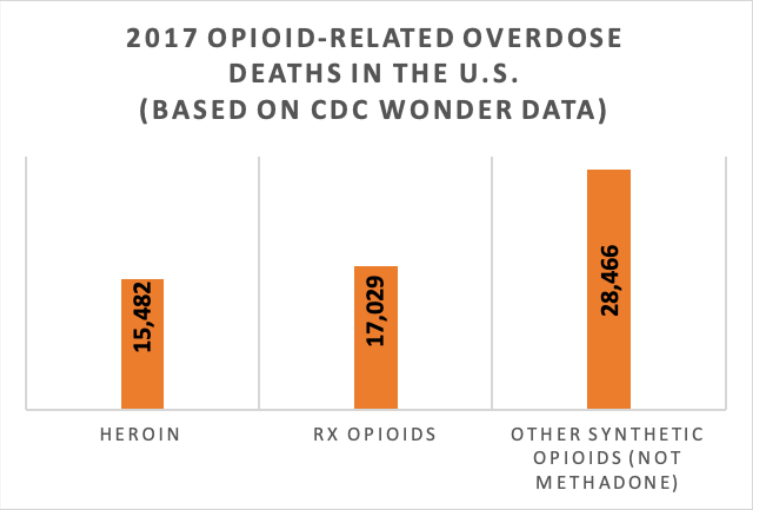
Naltrexone. Naltrexone is used in opioid use disorder relapse prevention after medically supervised withdrawal. As an opioid antagonist, it blocks opioid receptor sites, acting in a longer-acting manner but similarly to the overdose reversal drug, naloxone. Thus, Naltrexone minimizes the rewarding

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).



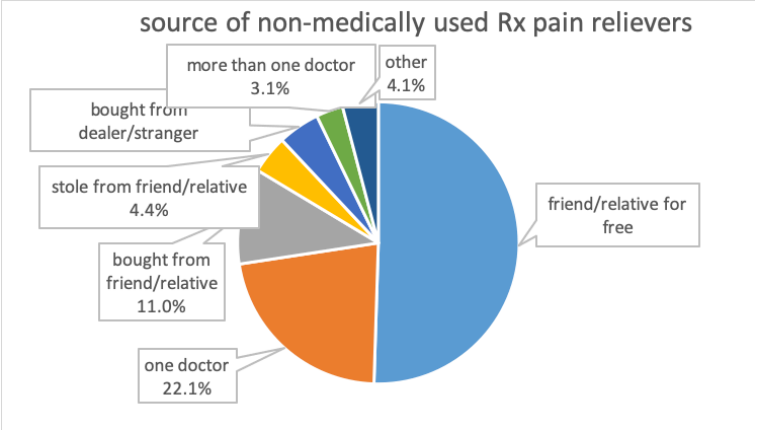
While profoundly disturbing, deaths due to overdose are not the only statistics of concern regarding opioid misuse in the U.S. For example, neonatal withdrawal syndrome (NWS) rates in 2015 multiplied by 8 times the 2006 rate in Ohio (https://www.wcbe.org/post/rate-ohio-babies-born-addicted-drugs-increasing) 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 (https://www.samhsa.gov/data/report/2018-nsduh-detailed-tables)			
	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 or media element has been excluded from this version of the text. You can view it online here: https://ohiostate.pressbooks.pub/substancemisusepart2/?p=405

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 withdrawalsyndrome (NWS)*** at birth. While the emphasis here is on outcomes for the baby, it is important to recognize that opioid misuse/OUD 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/OUD 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/OUD 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.

Module 11: References and Image Credits

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October 23, 2019 version

PART VI

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 used 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

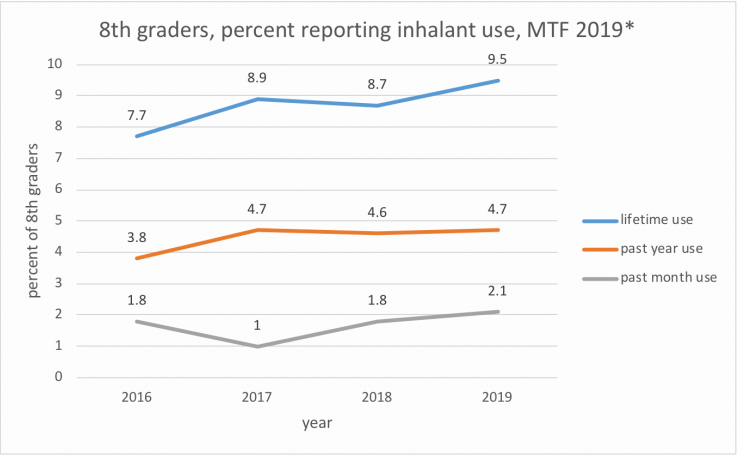
Inhalants are selected for misuse because of their "ability to rapidly induce euphoria," as well as their stimulant, disinhibiting, and hallucinatory effects (Baydala et al., 2010, p. 443). As soon as these immediate effects diminish, the individual may experience depression, dizziness, disorientation, 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

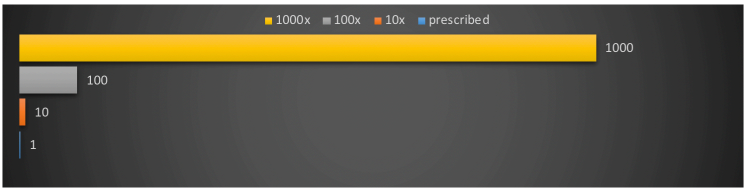
25 million individuals (9.1% of the population) aged 12 and older in the U.S. were estimated to have engaged in inhalant misuse during their lifetime, over 2 million (0.7%) during the past year, and almost 700,000 (0.2%) during the past month. The past month (current) misuse of inhalants was reported 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 to17-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).



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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.

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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:

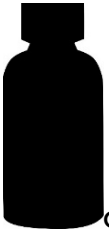
- Children
- Adolescents
- Individuals with certain types of physical or mental conditions (including pregnancy)
- Individuals with a pre-existing substance use disorder.

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 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 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:

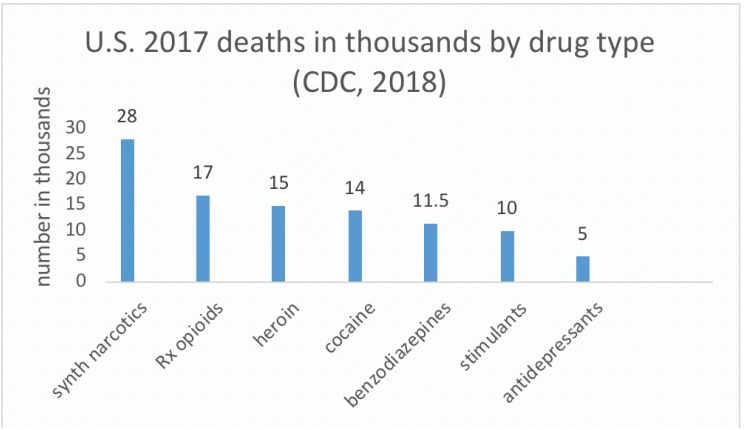
- Over 5.4 million (2.0%) are estimated to have engaged in misuse of psychotherapeutic drugs;
- Over 2.8 million (1.0%) engaged in misuse of pain relievers
- Almost 1.7 million (0.6%) engaged in misuse of stimulant drugs
- Almost 1.8 million (0.7%) engaged in misuse of tranquilizers or sedatives (including 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).



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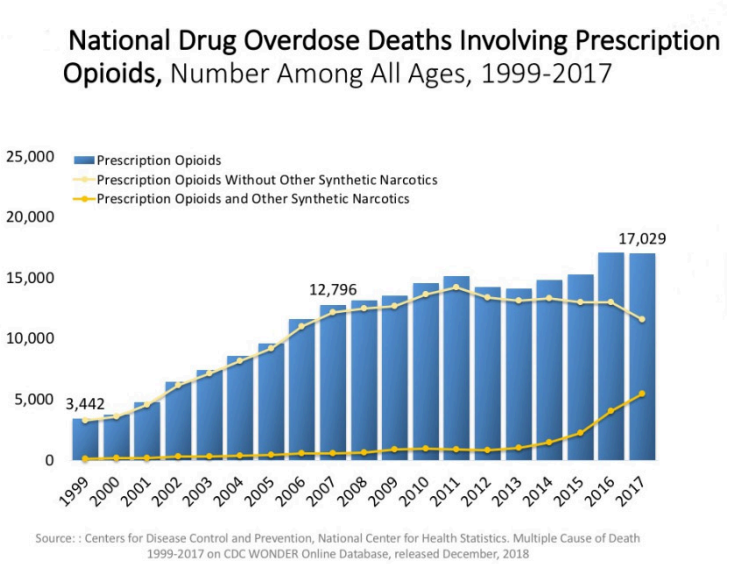
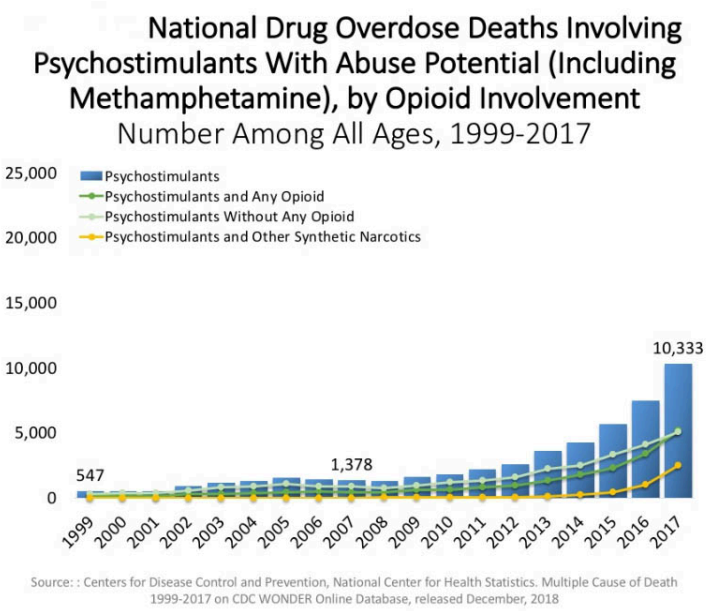
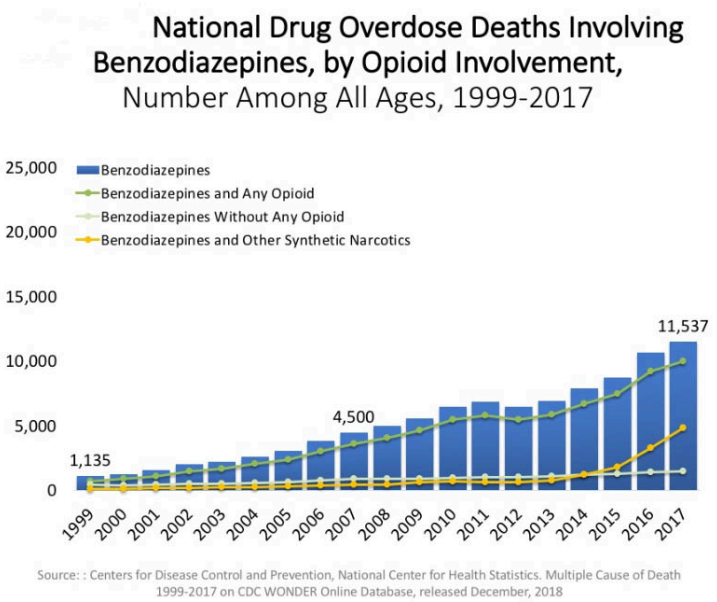
The next chart, produced from a 2018 CDC Wonder report and posted as a series of slides by NIDA (https://www.drugabuse.gov/related-topics/trends-statistics/overdose-death-rates), shows the rate of drug overdose deaths related specifically to prescription opioids from 1999 to 2017. Not only is the change 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.



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.



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 Stated 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).*

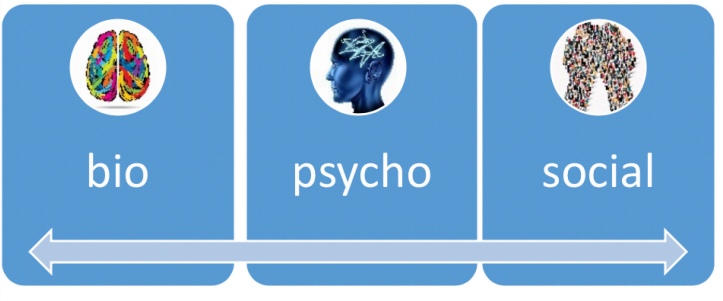
The underlying assumption is that substance use disorders and addiction are diseases of the brain and brain chemistry, therefore addressing those brain chemistry mechanisms is a reasonable approach to treatment. Furthermore, pharmacotherapy is intended as a means of supporting the transition from substance 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.

Alcoho 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, & Hamreaus, 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, & Hamreaus, 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:

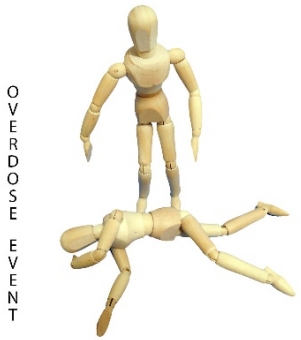
- nalmefene, used in Europe for this purpose, has moderate evidentiary support;
- baclofen, which has mixed results in studies from several countries;
- varenicline, used to support smoking cessation, has some efficacy support especially for individuals experiencing both alcohol and nicotine use disorder;
- 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 it 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). Buproprion, 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 staring 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, Knutxon, & 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.

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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.

Module 12: References and Image Credits

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PART VII

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-

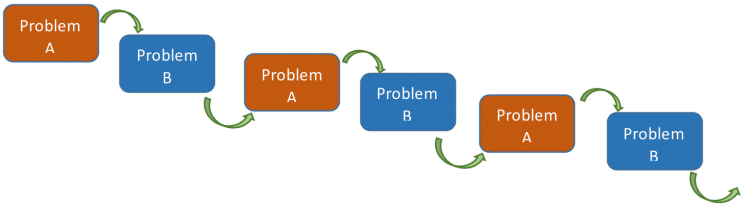
occur or are concomitants), several possible models explain the possible relationship between them. Let's consider examples where Problem A is a form of mental disorder and problem B is an alcohol use disorder.

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.,

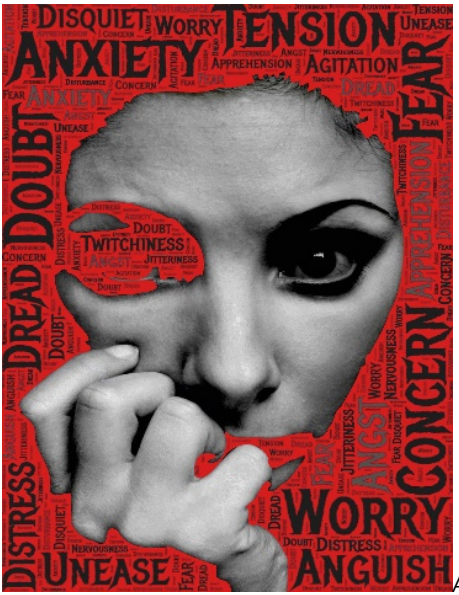
with substance use disorder considered as one form of mental disorder (Kessler et al., 2005). In this report, diagnosable mental conditions were considered co-occurring if they were present during the same 12-month period. Across the sample, 26.2% of participants experienced one or more diagnosable mental 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

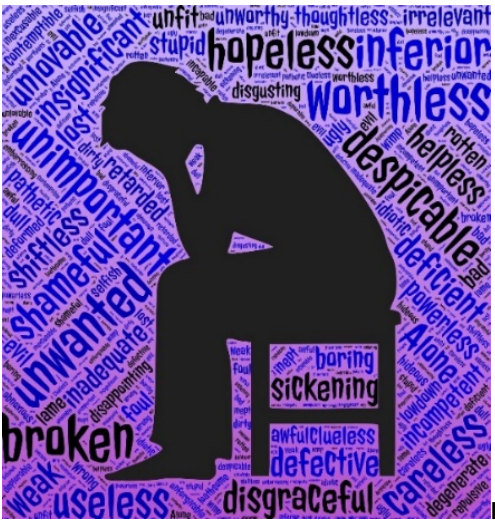
generalized anxiety disorder, regardless of the level of alcohol use disorder severity (Grant et al., 2015). Concluded from prior NESARC data was that, among individuals experiencing an anxiety disorder, almost 15% experienced a substance use disorder during the past year; additionally, among individuals 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).



An anxiety or panic disorder differs markedly from the kind of normative anxiety that helps someone function at their peak performance; it is what happens when anxiety becomes intense and unpredictable, is difficult to control, and affects a person's daily functioning. The experience acute anxiety or panic is both mental and physical, with release of "fight or flight" hormones surging throughout the body, affecting many organ systems. In many instances it is initially difficult to discern between an intense panic attack and a sudden cardiac event (heart attack). The types of anxiety disorder studied in the NESARC-III

included panic disorder, generalized anxiety disorder, and phobias (agoraphobia, social, and other specific phobias). It is not surprising that some individuals seek substance-involved solutions to prevent or reduce these intensely disturbing and disruptive anxiety experiences. Recall from prior modules, however, that anxiety also is one possible symptom of some types of substance misuse or withdrawal from some types of substances. For example, anxiety is often experienced during early recovery from alcohol use disorders. It is important through ongoing assessment and diagnosis processes to discern if a person is experiencing anxiety as a primary mental disorder or if what they experience might be substance or withdrawal induced anxiety: "anxiety can be either masked or exacerbated by the effects of substance intoxication, withdrawal, and chronic administration" (McHugh, 2015, p. 3).

As far as intervening with individuals who experience co-occurring anxiety and substance use disorders, the literature generally indicates that integrated treatments addressing both anxiety and substance misuse may have superior benefits to separate but concurrent treatment efforts (McHugh, 2015). Pharmacotherapy approaches are available for addressing each disorder but may be less well studied for concurrently addressing both conditions. While exposure therapy for anxiety disorders is somewhat controversial, in situations where an individual also engaged in substance misuse and/or experienced a co-occurring substance use disorder, exposure therapy may not be effective. This conclusion is based primarily on animal studies concerning fear extinction which demonstrated a marked impairment of the fear extinction process following chronic exposure to alcohol, nicotine, morphine (opioid), or cocaine (McHugh, 2015). Mindfulness practices have demonstrated a great deal of promise for supporting management of anxiety disorder symptoms and supporting recovery from substance use disorder, both of which involve mind-physiology aspects (Edguer & Taylor, 2020).



Depression. Based on a review of literature, investigators concluded that depression may co-occur among as many as 55% to 85% of individuals engaged in treatment for substance use disorders, depending on each study's methodology and whether the time frame of co-occurrence—at the same time or during the same year (Kingston, Marel, & Mills, 2017). The same literature review concluded that substance use disorder and bipolar disorder co-occurred during the same 12-month period in this population of individuals at about a 10% rate. Authors of the NESARC-III report (Grant et al., 2015) concluded that lifetime alcohol use disorder was significantly associated with persistent depression; among persons with alcohol use disorder, major depressive disorder was the most common of the co-occurring psychiatric disorders (McHugh & 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 used 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

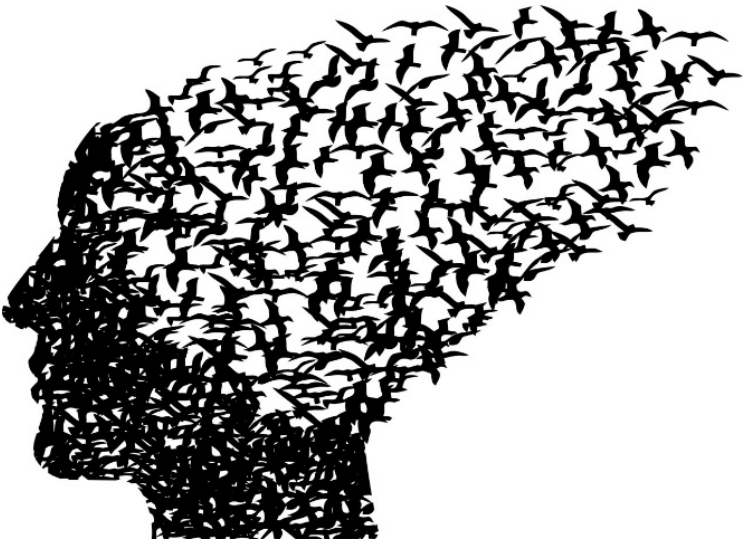
into the co-occurrence of schizophrenia and substance use disorder estimated that 47% of individuals with schizophrenia also met criteria for a substance use disorder involving alcohol or illicit substances—4.6 times greater than among the general population—and at least 70% of individuals with chronic schizophrenia exhibited nicotine dependence (Winklbaur 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" (Winklbaur 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 (Winklbaur 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 (Winklbaur 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 (Winklbaur 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.

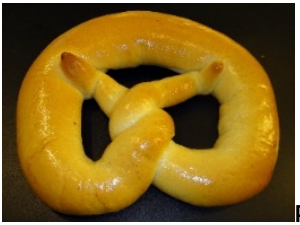
Dementia. While schizophrenia tends to first appear in younger adults, most forms of dementia tend to first appear during later adulthood. "Dementia refers to a set of symptoms and signs associated with a progressive deterioration of cognitive functions that affects daily activities. Symptoms may include 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 all, 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 & Kalllivas, 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 alco 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, 202).

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 medica 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-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).

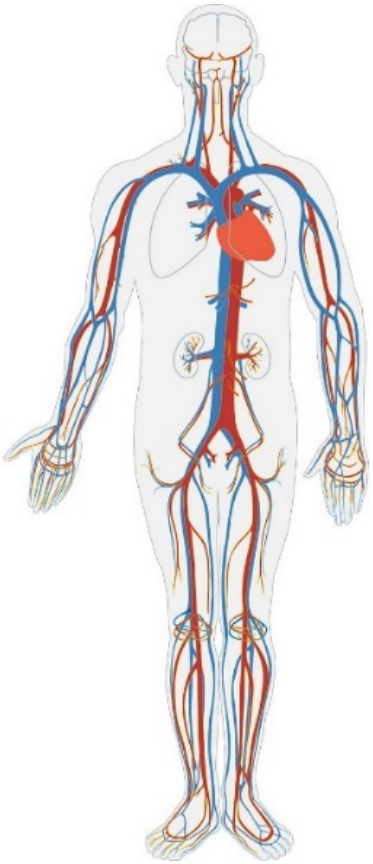
Not only is the concern that someone engaged in injection or non-injection substance misuse might become infected with a disease, but their disease progression and prognosis may be worse, as well. For example, in a study of 1,712 individuals entering care related to HIV infection, "patients with a history 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).
- *Opiods*. 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"):

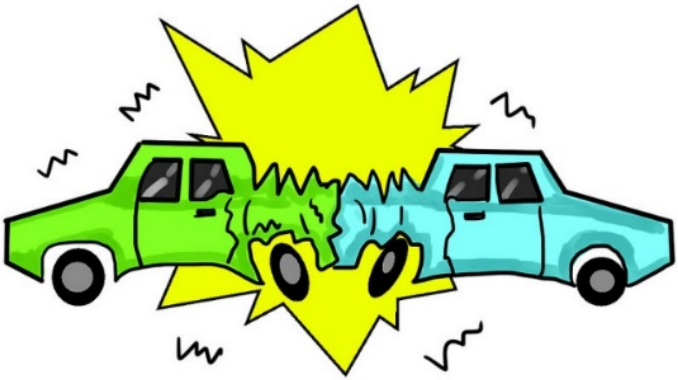
BAC range	Odds Ratio
0.01% – 0.04%	**1.17**
0.05% – 0.07%	**1.71**
0.08%* -0.10%	**3.93**
0.11% or greater	**10.68**

*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

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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.

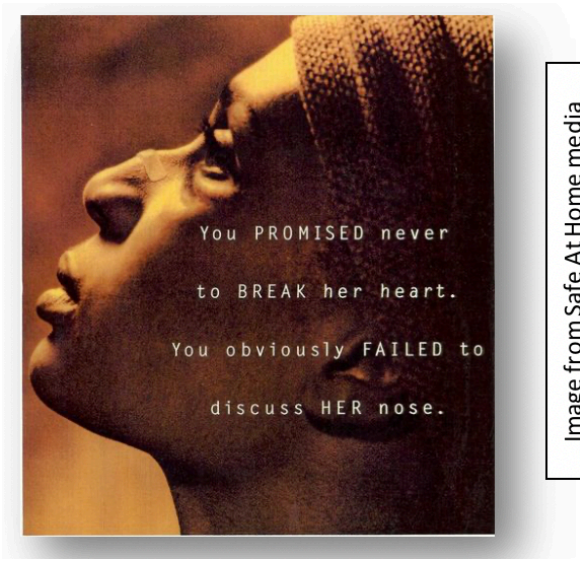

Image from Safe At Home media campaign, Milwaukee, WI

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: “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 abused 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). 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).

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.

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).

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?

s 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 of 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.

Module 13: References and Image Credits

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Appendix - Syllabus

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