Theories and Biological Basis of Substance Misuse, Part 1

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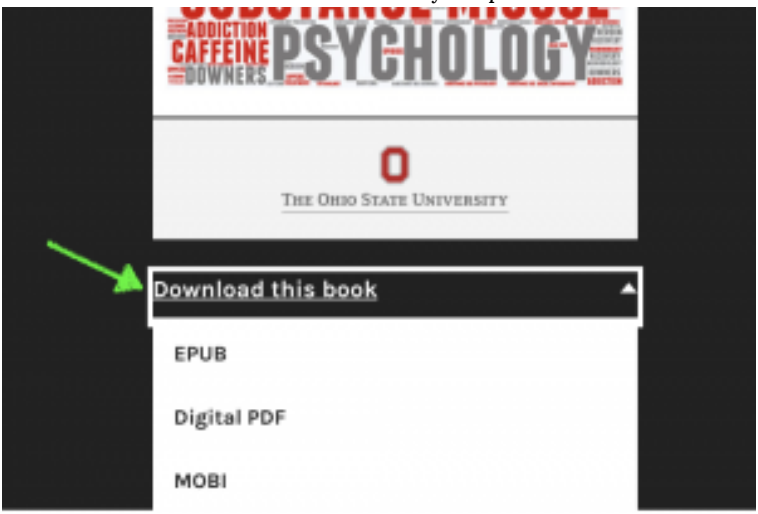
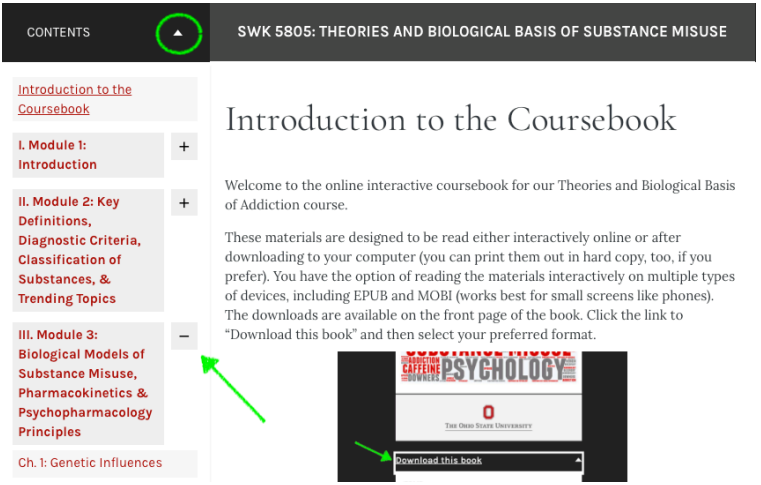
Introduction to the Coursebook

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 1: INTRODUCTION

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

Reading Objectives

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

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

Ch. 1: Background Facts and Figures

What Are Psychoactive Substances?

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

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

Who (Mis)Uses Psychoactive Substances?

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

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

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

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

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

- *What do you think are the 3 most commonly used substances?*
- *What percentage of individuals do you think use each of the substances you identified?*
- *What do you believe is the distribution of men versus women*

Epidemiol

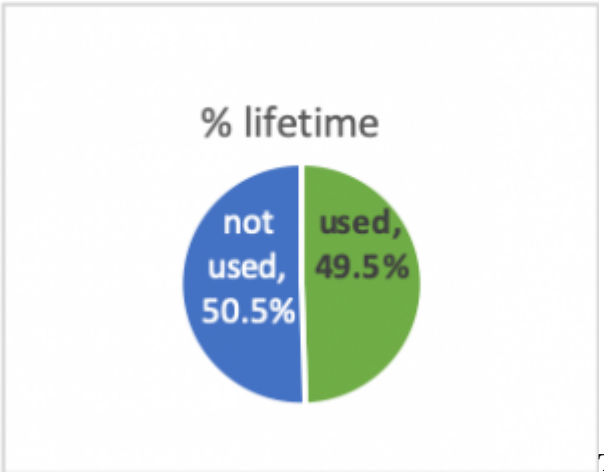
using these substances?

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

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

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

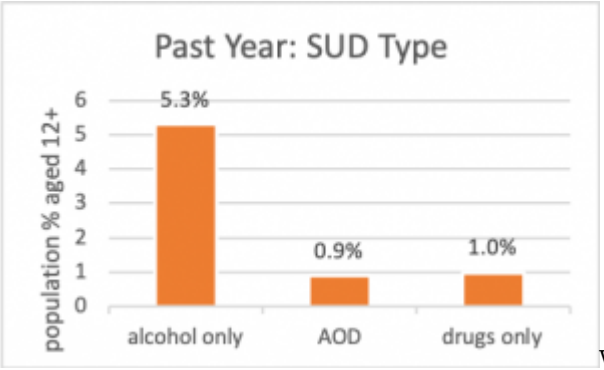
Scope of the Issue.



The World Drug Report 2019 (WHO, 2019) reported that not only are the adverse health consequences of drug use more severe and widespread than previously believed, the severity of the situation is increasing. Reportedly, an estimated 35 million individuals globally experienced drug use disorders requiring treatment services and an estimated 271 million (5.5% of the world's population) used drugs outside of medical recommendation during 2017. The report also concluded that only 1 in 7 persons in need of treatment for a drug use disorder receives it. The report raises alarm over the 25% increased production of cocaine compared to the previous year, reaching an all-time high. It also calls out the 47,000 opioid overdose deaths reported in the United States during 2017 (up 13% from 2016) and 4,000 in Canada (up 33% from the previous year). An opioid crisis is also arising in West, Central, and North Africa although the specific opioid drugs involved may differ in various parts of the world.

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

person to be currently using substances if use was reported during the past month; almost 30.5 million (11.2% of population) were estimated to have used illicit drugs in the past month.



While substance use/misuse are important to track, it is also important to know about individuals experiencing a substance use disorder. From that 2017 survey, over 19.7 million individuals aged 12 or older (7.2% of population) were estimated to experience a substance use disorder (SUD) involving alcohol and/or and illicit drug use during the past year, as measured in 2017; the vast majority of SUDs involved alcohol alone (5.3%) or in combination (0.9%) with illicit drugs, leaving 1% with a drugs only SUD.

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

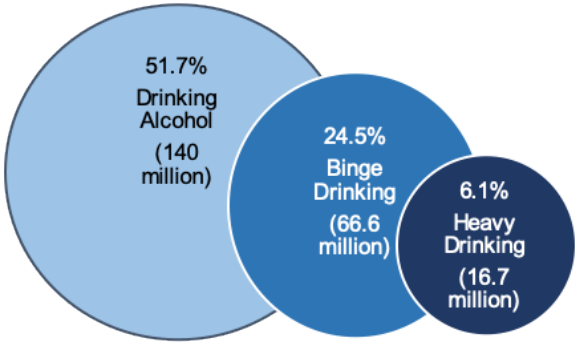
The NSDUH data can be analyzed in somewhat more nuanced ways. Let's look at the differences between types of substances used and who engages in AOD use/misuse in the U.S. (by age, gender, and racial/ethnic group). Despite the emphasis on certain substances in the news and stereotypes stemming from various

sources, the answers to these kinds of "what" and "who" questions are informative, and some answers may surprise you.

Type of Substance: Alcohol.

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

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



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

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

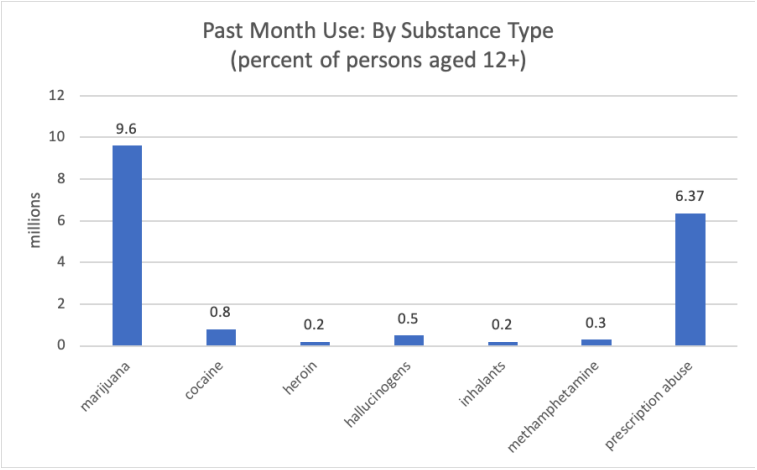
Work under the umbrella goal called “Close the Health Gap” (Begun, Clapp, and the Alcohol Misuse Grand Challenge Collective, 2015).

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

Type of Substance: Other Drugs.

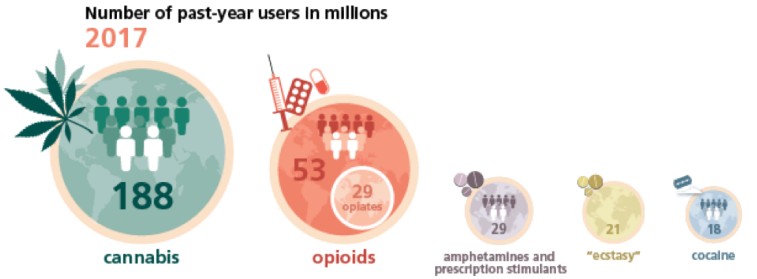
We looked at data concerning alcohol in the AOD acronym, now let's take a look at those other drugs. Over 30 million individuals (11.2% of the population) were estimated to have used illicit drugs in the past month based on 2017 NSDUH data (SAMHSA, 2018). The type of illicit drug most often used in the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

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



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

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



We looked at alcohol (a legal substance) and illicit drugs, but what about the other legal substance so commonly used in the U.S. and around the world—nicotine? Among persons aged 12 and older, based on the 2017 NSDUH data, an estimated 170.5 million individuals have used tobacco products (not including e-cigarettes/vaping) during their lifetimes—62.7% of the population. Current use

was attributed to over 61 million, or 22.4% of the population (SAMHSA, 2018). The vast majority of use involved cigarettes; smokeless tobacco, cigars, and pipe tobacco were less common. According to the WHO (https://www.who.int/news-room/fact-sheets/detail/tobacco) tobacco kills more than 8 million people annually, 1.2 million of whom were non-smokers exposed to second-hand smoke; 1.1 billion individuals smoke tobacco worldwide; and, tobacco kills up to half its users.

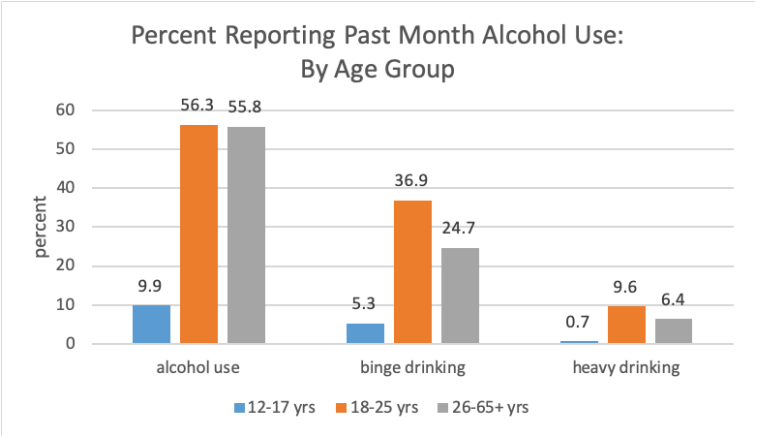
The report also refers to other victims of tobacco: children from poor families employed in tobacco farming absorb nicotine through their skin from handling tobacco leaves and are vulnerable to "green tobacco sickness" as a result. In addition to concluding that tobacco represents a significant U.S. and global public health concern, this information indicates that deciding to smoke is not just an individual choice matter—it has implications for others nearby (second-hand smoke exposure) and for others of whom we may be unaware (involved in production and distribution). These issues are not unique to tobacco, by the way—it is a relevant social justice consideration regarding all types of drugs.

Type of Substance by Age Group.

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

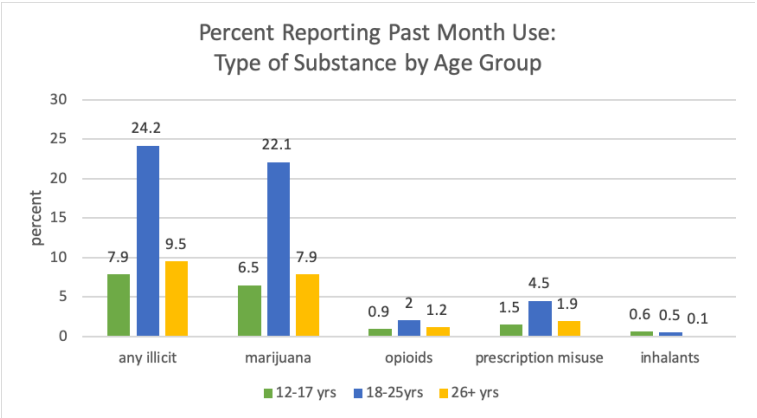
Technically, alcohol is an illicit substance for underage youths (those aged 12-17 and many in the survey's 18-25 group). Figure 1.5 shows the percent reporting past month use of alcohol, binge drinking, and heavy drinking by age group in the 2017 NSDUH data. These numbers all peaked for our emerging adult group. While the alcohol use percentage remained relatively steady into adulthood (over age 25), binge and heavy drinking percentages declined. Again, these data indicate that the majority of adults who drink generally do so in moderation. However, more than half of individuals in emerging adulthood and adolescence who used alcohol in the past month engaged in binge drinking (considered a risky pattern); slightly less than half of adults did so. About 18% of emerging adults engaged in the riskiest pattern, heavy drinking, compared to about 11% of adults and about 8% of adolescents (SAMHSA, 2018).

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



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

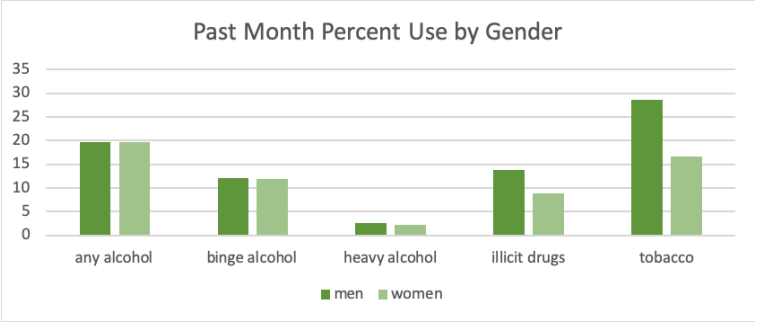
Figure 1.6. Patterns of past month illicit drug use by age group.



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

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

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



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

Type of Substance by Race/Ethnicity.

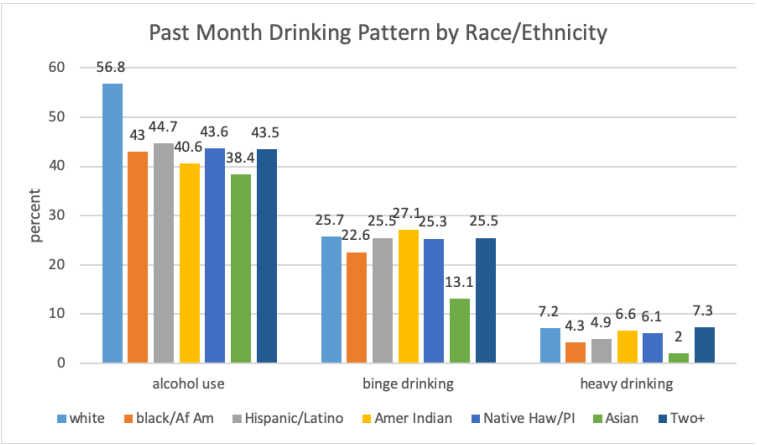
The seven U.S. racial/ethnic groups for whom information is reported in the 2017 NSDUH survey (SAMHSA, 2018 include: white; black/African American; Hispanic/Latino; Asian; American Indian/ Native Alaskan; Native Hawaiian/Other Pacific Islander; and, those of two or more races.

Alcohol. The group most likely to report past month use of alcohol was comprised of individuals who identified themselves as white (56.%) and the lowest rates were reported by the group identifying as Asian (38.4%) groups (see Figure 1.8). Looking at these statistics

another way, the highest rates of alcohol abstinence (individuals not drinking) in the past month appeared among the Native Hawaiian/ Other Pacific Islander, American Indian, and African American groups.

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

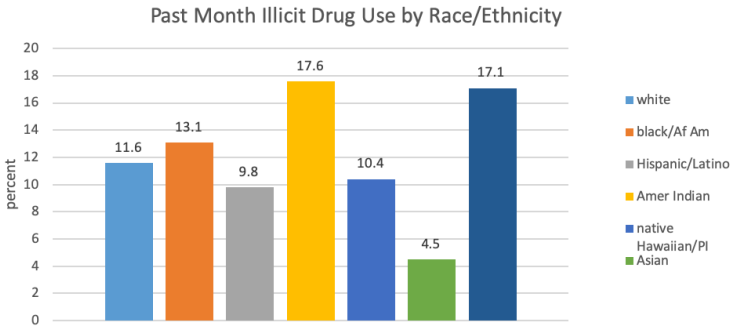
Figure 1.8. Past month drinking patterns reported by race/ ethnicity



Other drugs. The groups reporting the highest rate of past month illicit drug use were those who self-identified as American Indian and as belonging to two or more races (17.6% and 17.1%, respectively; see Figure 1.9). The lowest rate was reported among those identifying as Asian (4.5%) with the other groups falling in between

(9.8% to 13.1%). As you can see, this picture differs somewhat from the story presented by the alcohol data.

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



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

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

Now that you have read the information above, take a moment W e r e y o u s u r p r i

to compare what you learned as answers to the questions below with your pre-reading answers:

- *What are the 3 most commonly used substances?*
- *What percentage of individuals aged 12 and older use each of those most common substances?*
- *What is the distribution of men versus women using these substances?*
- *What age group is the highest user of these substances?*
- *What are the patterns of substance use among white, black, Hispanic/Latino, and Native American persons?*
- *What percentage of the population has a diagnosable substance use disorder?*
- *What percentage of individuals experiencing substance use disorders receive treatment for the problem?*

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

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

While substance misuse is a contemporary social problem, the story of humans experiencing problems related to the use of psychoactive substances is at least 4,000-10,000 years old (Hanson, Venturelli, & Fleckenstein, 2015; Howard, Garland, & Whitt, 2013; Singer, 2012). United States history is peppered with documentation of problems associated with alcohol and other drugs. For example, the opiate drug morphine was widely used during the Civil War to manage wounded soldiers' pain, leaving many of them experiencing morphine addiction as a result. Subsequently, heroin became available and marketed as a "non-addicting opiate with greater analgesic potency than morphine" (Kornetsky, 2007, p. 96). Prior to the Civil War, 60-75% of Americans experiencing opium or morphine addiction were women, in large part because physicians often prescribed opiates to deal with a wide variety of "female" complaints (Blumenthal, 1998). In addition, physicians of the time often prescribed alcohol as a treatment for opiate addiction, and

many socially acceptable and widely accessible medicines contained very high alcohol or opium content (Plant, 1997; Straussner & Attia, 2002; van Wormer & Davis, 2013). Cocaine was also prescribed and marketed in this way. See this historic advertisement promising an instant cure for oral pain (including babies' teething pain).

At around the end of the 19th century, awareness of potential harms associated with these substances spread. The U.S. (and other nations) has since implemented various policy efforts to reduce both or either supply and demand for different kinds of drugs (Vakharia & Little,*in press*). Protecting public health was not the only motivation in many instances, however.

Early U.S. Policy and Legislation Efforts.

The first federal policy prohibiting distribution (supply) and non-medical use (demand) of a drug was the Opium Exclusion Act of 1909 (Vakharia & Little, *in press*). Opium used for medical purposes remained legal, but opium prepared for "smoking" no longer was. At this point in

history, large numbers of Chinese immigrants were working in the U.S. and opium smoking was associated with this population. The common use was not outlawed, only the form of opium used by Chinese immigrants (Vakharia & Little, *in press*).

The next major federal policy, the Harrison Narcotic Act of 1914, was directed at drugs derived from opium or coca leaves, to control their production, distribution, and use. Possession or use of a narcotic (this included cocaine) without a physician's prescription was a violation that states could criminalize. Cocaine was targeted, possibly for political reasons parallel to the situation with opium (Vakharia & Little, *in press*): up until the early 1900s, cocaine was commonly added to beverages and medicinal tonics because of its energizing properties (and boosting worker productivity). Even though most individuals using cocaine were white, concern grew over its increasing popularity within the black community, particularly across the Southern U.S. (Vakharia & Little, *in press*). An added public policy motivation: governments could now collect special taxes on the production and distribution of these drugs. Tobacco has been taxed at the federal and state levels since the Civil War, with the amount fluctuating (until 1983) according to governments' need to generate revenue (IOM,1994).

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

shows agents pouring liquor confiscated in a New York City raid during Prohibition; it comes from the National Archives).

Prohibition Era.

Although the combination of the 18thAmendment to the United States Constitution and the Volstead Act (which clarified that beer and wine were included as alcoholic beverages) were implemented beginning in 1920, many states had already enacted their own local prohibition laws (Hanson, Venturelli, & Fleckenstein, 2015; Kelly, 2017). You might find it interesting to pursue historical literature documenting the intersections of alcohol/drug policy with historical and sociological trends such as the temperance movement, women's suffrage, immigration, organized crime, classism and racism (see for example, Straussner & Attia, 2002; van Wormer & Davis, 2013). Many of these historical policy patterns have implications for today's politics and policy debates, as does

the extensive economic impact of both local and international trade in substances such as alcohol, tobacco, coffee, tea, opium, cocaine, and others.

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

It was also during the 1920s and 1930s that many states developed prohibition-style policies about marijuana, and the federal government got involved in 1937 with the passage of a Marijuana Tax Act and more severe criminalization policies during the 1950s. Marijuana policy concerns cannabis plant products; the word marijuana came from Mexico, but its use in U.S. policy is becoming recognized as having racist and propagandist connotations by many scholars (Malcolm, *in press*). Historical roots of marijuana prohibition include racial/ethnic concerns about Mexican immigrants and African Americans that parallel opium and cocaine policy regarding Chinese immigrants and Southern black workers (Malcolm, *in press*).

Evolution of Contemporary U.S. Drug Policy.

During the 1960s, many programs and policies aimed at addressing both the supply and the demand sides of the drug trade were established. The term **"*War on Drugs*"** began to appear around 1971, referring to stepped-up drug criminalization and law enforcement efforts (McNeece & DiNitto, 2012; Schori & Lawental, 2013). While these programs focused on our nation's internal drug problem, it is virtually impossible to separate the U.S. drug war efforts from international policy, international relations, and global economics. It also had political undertones and overtones related to race, age, and the "counter-culture" presence in America at the time.

One criticism of "America's Longest War" (the title of a 2013 award-winning documentary) has great relevance to social work and disciplines concerned with social justice: the War on Drugs contributed to extreme racial and gender inequities in the nation's incarceration rates (Bush-Baskette, 1999; Chesney-Lind, 1997). For example, by the early 1990s, 74% of individuals serving prison sentences for drug possession were black, despite accounting for only 13% of individuals who use drugs (Kilty & Joseph, 1999). The War on Drugs also helps explain the relative explosion of women in prison for non-violent, drug possession charges that occurred during the late 1980s to 1990s—leading to a declaration that the War on Drugs became a "War on Women" (Bloom, Chesney Lind, & Owen, 1994). Another criticism of the War on Drugs addresses its high economic costs: the Office of National Drug Control Policy's

(ONDCP) FY 2020 National Drug Control Budget request was $34.6 billion, an increase of $1.3 billion over the actual FY 2019 budget (https://www.whitehouse.gov/briefings-statements/white-house-seeks-billions-record-investments-stop-drug-epidemic/). The ONDCP is a component of the President's White House Executive Office, created by the 1988 Anti-Drug Abuse Act.

Pregnant Women and Substance Use.

Part of the concern about a "War on Women" stems from how policy responses (mostly at the state level) to women's use of alcohol or other drugs (AOD) during pregnancy. States and local communities differ markedly in their policy responses to this issue. The responses run the gamut from dealing with the public health aspects (the health of mother and baby) to criminalization. For example, in some states, a pregnant woman can be involuntarily committed to a treatment facility, jail, or relative's home for supervision to prevent her continued use of substances known to be harmful to a developing fetus. Many states have policies relating to the substantiation of child maltreatment allegations when a pregnant mother uses alcohol or other drugs. While intended to help protect the unborn child from potentially harmful drug exposure, these policies are controversial, as they also may discourage women from seeking much-needed prenatal

care for fear of discovery and becoming subject to consequences imposed through the courts and child welfare system.

Drinking Age Legislation.

Drinking age legislation in the U.S. currently aims to restrict alcohol use by persons under the age of 21 years. You may find it hypocritical that an 18-year-old person is treated as an adult in other domains (legal rights to marry, join the military, enter into legal contracts), but not legally allowed to purchase alcohol. Drinking establishments are certainly concerned about reduced revenue from not being allowed to legally serve alcohol to 18- to 20-year-olds. On the other hand, there exists compelling evidence that higher drinking age minimums are associated with lower traffic fatality rates. Another rationale involves an attempt to mitigate the potential harms associated with exposing the still-developing young adult brain to alcohol—major developmental changes in brain structure and function, beginning early in puberty, continue well into the period of early adulthood (Spear, 2000; more about this in our focus on alcohol module). Raising the legal age to be well over 18 eliminates confusion about enforcing alcohol-free zones in high schools and many parts of college/university life, as well. This policy periodically becomes contested, tested, and retested in the United States, including a

period during the 1970s when different states had different legal drinking ages of 18, 19, and 21. Drinking age policy is determined at the state level, however federal highway funding is tied to state drinking age policy and governing the states' uniform decision to support a minimum legal drinking age of 21 years.

Decriminalization Efforts.

Our nation has an opportunity to learn from the contemporary "natural experiment" in policy reform whereby several states decriminalized the production, distribution, possession, and/or use of cannabis for medical and/or recreational purposes (more about this in our focus on cannabis module). Some hypothesize that ***decriminalization*** of substance possession or use reduces economic incentives for illegal production and distribution of drugs, allowing government entities to increase revenue through taxation (McNeece & DiNitto, 2012).

Decriminalization is contested, however, as potentially contributing to increased rates of substance use disorders and other health risks associated with substance use, as well as related problems such as driving under the influence and community safety. Law enforcement professionals expressed grave concerns regarding the potential for increased demands on police forces already stretched by the need to manage alcohol-related situations if marijuana is also legally used by the general public. Recent evidence suggests that the presence of legal (medical) marijuana dispensaries are associated with increased violent and property crime rates in adjacent areas (Freisthler, Ponicki, Gaidus, & Gruenwald, 2016).

Addiction treatment providers have expressed concern about the potential impact of easier access on individuals already in recovery from substance use disorders and the potential for further stressing an under-resourced service delivery system with an increase in demand for intervention to address problems with marijuana use. Prevention experts are concerned about the message that legalization/decriminalization might convey to young people considering initiating substance use. And, there continues to be controversy as to the potential (as yet, unknown) effects on the health care system that might result from an increase in disease or disability resulting from individuals' long-term use of marijuana products—along the lines of what we see with alcohol.

However, social justice advocate relates significant inequities in

how the criminalization of cannabis and other drugs is enforced, leading to both a mass incarceration trend and tremendous racial/ethnic (and gender) disparities in who becomes incarcerated in the nation's, states', and local communities' jails and prisons. Mass incarceration beginning in the mid-1970s meant the incarceration rate almost tripled from 1970 (96 per 100,000 population) to 1990 (over 300 per 100,000; Lloyd & Fendrich, *in press*). Not only were War on Drugs policies responsible for this trend, so were policies and policy enforcement stemming from Sentencing Reform Act (1984), Anti-Drug Abuse Act (1986), and Omnibus Anti-Drug Abuse Act (1988) policies concerning sentencing guidelines establishing mandatory minimum penalties for drug crimes (Lloyd & Fendrich,*in press*). Disparities in incarceration of persons of color were further stimulated by differential sentencing for "crack" cocaine (more commonly used by persons of color) compared to powdered cocaine (used by more affluent and white individuals). Advocating for "smart decarceration" often means advocating for less punitive (and more treatment) responses for low-level and non-violent drug involvement (see Pettus-Davis & Epperson, 2015).

Drug courts.

Traditional drug-control methods of the criminal justice system, such as mandatory incarceration and harsher penalties, along with court-mandated treatment following release from incarceration, have not proven to be sufficiently effective to curb the problems associated with illicit drug use (Broadus, 2009). In addition, these efforts were wreaking havoc on the court system by creating tremendous backlogs of cases considered to involve relatively minor, non-violent offenses, and pushing jail populations far over capacity at great public expense. In response, a movement emerged during the early-1990s to establish special courts for managing nonviolent drug-related cases. The mission was to engage individuals in court-monitored, structured, evidence-supported treatment and divert them from being incarcerated if they complied with the treatment plan. By 2018, over half of all U.S. counties sponsored at least one of over 3,100 drug courts in operation (Lloyd & Fendrich, *in press*). Each program involves an interdisciplinary team of criminal justice and mental health professionals responsible for creating an

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

Harm Reduction Policies.

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

- programs to prevent driving while under the influence of alcohol or other substances, while not necessarily stopping a person from using AOD;
- clean needle and syringe exchange programs to reduce risk of exposure to blood-borne communicable diseases like HIV/AIDS and hepatitis;

- supervised drug-use settings where individuals' drug use and safety are monitored by someone whose judgement is not substance-impaired (more common in European nations);
- fentanyl testing of heroin/opioids or other "street" drugs to prevent unexpected opioid overdose;
- supportive housing for which abstinence is not an eligibility requirement;
- making opioid overdose reversal resources (Narcan) available to first responders to save the lives of individuals who might otherwise die before professional treatment is accessible.

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

Access to Treatment.

Improving access to treatment for substance misuse and substance use disorders represents another modern policy/advocacy front with great social work significance.

In the previous epidemiology sections, you learned about the considerable gap that exists between the need for these services and the numbers of individuals (and families) able to receive them. A person's ability to engage in formal, professional treatment for these problems often depends on the ability to pay with insurance or self-pay dollars.

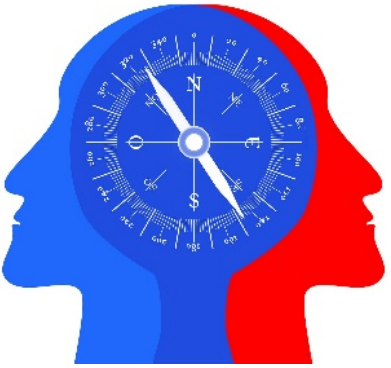
One potential advantage of the Affordable Care Act (ACA) first implemented in the United States during 2013-2014 was the potential for increased access to mental health and substance use disorder treatment services for many individuals. With the passage of the ACA:

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

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

Despite the excitement over expanded coverage and protections, concerns arose regarding the treatment system's ability to meet

the anticipated increase in demand: Do we have enough trained professionals to meet the experienced need?



At the end of 2016, the U.S. Congress passed two major pieces of legislation related to substance use and addiction. The first was the Comprehensive Addiction and Recovery Act (CARA) that provided legal status for many harm reduction strategies, such as increased access by non-physicians to naloxone for reversing an opioid overdose. However, CARA did not provide funding for these approaches. The second was the 21stCentury Cures Act that provided federal funding to "accelerate the discovery, development, and delivery of 21stcentury cures" and other purposes (https://www.congress.gov/bill/114th-congress/house-bill/34/text). In addition to ensuring specific funding for the NIH and Federal Drug Administration, the act provided funding for states with a relatively high prevalence of opioid use disorders to develop their responses for addressing the opioid abuse crisis. This included prescription drug monitoring programs, prevention activities, health care provider training about best practices, supporting access to treatment programs, and other public health-related activities to address the identified crisis. The impact of policy revisions regarding health care coverage since the 2017 change in national leadership remains to be seen. The White House created the President's Commission on Combatting Drug Addiction and the Opioid Crisis (March 2017) with the mission of studying "the scope

and effectiveness of the Federal response to drug addiction and the opioid crisis…and make recommendations to the President for improving that response" (https://www.whitehouse.gov/the-press-office/2017/03/30/presidential-executive-order-establishing-presidents-commission). However, at the same time, the Office of National Drug Control Policy, a component of the President's Executive Office, risked being significantly defunded. Clearly, there exists considerable ambivalence in the policy response to substance misuse and treatment for substance use disorders in the U.S.

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

Thinking About Policy Issues

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

- **Drinking Age Legislation:** What do you think about the current minimum legal drinking age policies in the United States? What do you know about policies in your own community regarding being a minor in possession of alcohol, driving while under the influence as a minor, and the provision of alcohol to underage minors? How might these issues apply to cigarettes, e-cigarettes, and other tobacco products?

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

addiction, to carry for others to administer if they overdose?

- How do you feel about doing this for someone yourself (and perhaps conduct rescue breathing during the time it takes to work)?
- How do you feel about these costs affecting city/county/state budgets for first responders?
- How do you feel about pharmaceutical companies charging so much for this life-saving treatment, despite their having invested heavily in its development and testing?

Ch. 3: Considering the Language We Use

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

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

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

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

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

- ARTICLE: http://www-tandfonline-com.proxy.lib.ohio-state.edu/doi/abs/10.1080/1533256X.2016.1201372

When you are finished reading this brief article:

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

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

An interactive or media element has been excluded from this version of the text. You can view it online here:
https://ohiostate.pressbooks.pub/substancemisusepart1/?p=53

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

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

Ch. 4: Summary

In the readings for Module 1 you read about:

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

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

Module 1: Key Terms

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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PART II

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

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

Reading Objectives

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

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

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

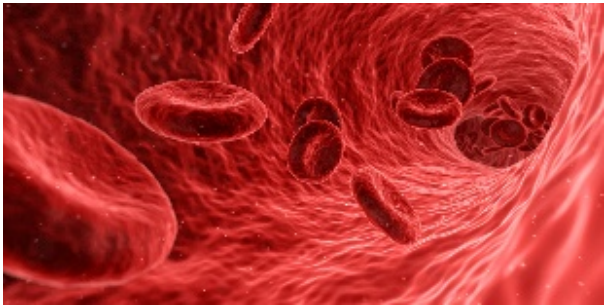
Ch. 1: Key Definitions & Diagnostic Criteria

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

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

Key Definitions

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



Substance Use. The concept of ***substance use*** is fairly straightforward: introducing a psychoactive substance into the body/circulating blood stream. As you will learn in this course, there are many ways these different substances are used: drinking, eating, introducing through oral membranes, or otherwise ingesting; inhaling, "snorting," or introducing through nasal membranes; smoking or otherwise inhaling through lungs; injecting; and, absorbing through skin are all common modes of introduction. The concept of substance use does not distinguish amounts or consequences of use.

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

irreversible, such as exposure to infectious disease, accidental injury (to self or others), legal difficulties/incarceration, and damage to physical or mental health (including, but not limited to overdose or substance use disorder).

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

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

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

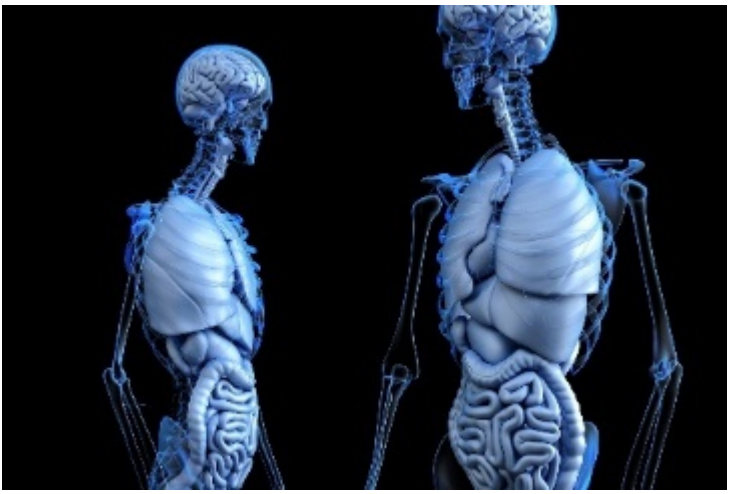
- A need for markedly increased amounts of the substance to which tolerance has been developed in order to achieve intoxication or the desired effect; or,
- A markedly diminished effect with continued use of the same amount of the substance.

An example demonstrating where acquired tolerance is particularly problematic occurs with individuals who have developed tolerance, are unable to obtain the substance for a period of time, then resume use again at the same level previously used. For instance, someone may have been regularly using heroin at a certain high level prior to being incarcerated in jail, unable to access heroin during their period of incarceration, and resumes using at community reentry following release from incarceration. If the period of abstinence was

long enough, the person's body may have re-adjusted to not having the substance on board, and their tolerance diminished. Resuming use at the previously tolerated level could lead to an overdose in their re-adjusted condition.

Another example where tolerance matters has to do with alcohol consumption. A person consuming enough alcohol to have a blood alcohol level (BAL)/blood alcohol concentration (BAC) of .20 for the first time likely would experience blackout. However, a person who routinely drinks to this BAL/BAC level might have developed sufficient tolerance that, while their functioning is significantly impaired, the effects are more reflective of a lower BAL/BAC outcome for individuals who drink less and drink less often.

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

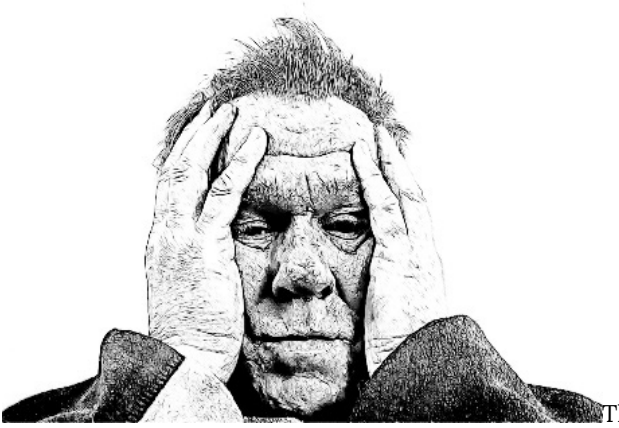


Withdrawal. With many (but not all) substances, a person's body adapts to the presence of the substances to such an extent that if that substance is no longer available, the person experiences a host of very difficult symptoms (more about this in Module 3). In the DSM-V language, substance ***withdrawal***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

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

Examples of alcohol withdrawal symptoms, for instance, include high levels of anxiety, sleep disorders, tremors, nausea/vomiting, sweating, racing heart, physical restlessness, and possible seizures. If a person experiences two or more of these symptoms as a direct result of stopping or reducing alcohol intake, and not as a function of some other condition or other substance the person may have used, the person may be diagnosed with alcohol withdrawal syndrome. Examples of symptoms associated with withdrawal from heavy, prolonged cannabis/marijuana use can include: irritability, anger, aggression, difficulty concentrating, nervousness, anxiety,

sleep disturbances, vivid unpleasant dreams, decreased appetite/ weight loss, restlessness, depression, shakiness, sweating, fever, chills, and headache. Symptoms associated with opioids, where use has been heavy for several weeks or more, can include: depression, nausea/vomiting, muscle pain, runny eyes, runny nose, sweating, diarrhea, fever, and insomnia. The experience of withdrawal from substances can be fraught with misery.



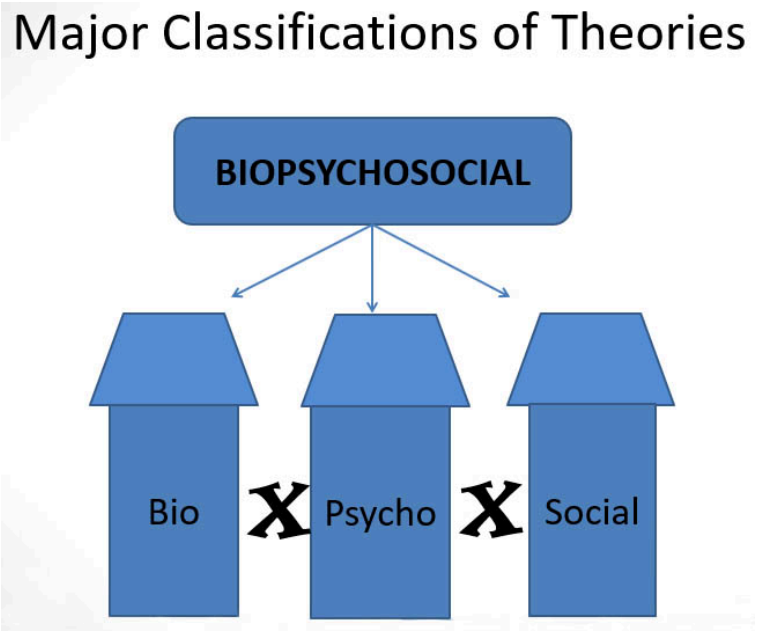
There exist both differences and similarities in withdrawal from different types of substances, not only in terms of symptoms but also in how soon symptoms might appear and how long they might last. Learning about different substances (as we will in Part 2 of the course) is important because withdrawal from some substances that is not medically managed can be fatal. Just quitting may not always be the safest choice—a person may need to be gradually weaned off certain substances to avoid dangerous withdrawal effects on heart rate/ rhythm, blood pressure, and severe seizures. Consider the public health implications of large community disasters like the combination of Hurricanes Katrina and Rita that made it impossible for some individuals to access alcohol, other substances, or even prescription medications on which their bodies had come to depend—their withdrawal could contribute to loss of life. This is true if a person's access is interrupted by the theft of the substances/

prescription medications, inability to pay for medications or a pharmaceutical company's interruption of supply.

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

Biopsychosocial Perspective. You may have heard the term "***biopsychosocial***" in reference to how we think about complex behavioral health issues and human development. In review (or if the concept is new to you), it means that in order to fully understand a phenomenon like substance misuse it is essential to understand the biological, psychological, and social factors involved in its development, maintenance, and resolution. Unfortunately, because of the different disciplines and professions involved, these three domains are often considered individually or distinctly from each other, rather than as an integrated whole—each domain is often considered as a silo, separate from the others (and not always equal to the others). In reality, the three domains interact in important, mutually influential ways. Whether or not someone engages in substance misuse is influenced by that person's biological makeup and processes, psychological makeup and experiences, and experiences/interactions with the social and physical environment. One thing that all of the research in substance misuse and substance use disorders taken together has taught us is: THERE IS NO ONE SINGLE CAUS, and there is not even any one single domain involved. These are very complex phenomena with multiple interacting causes. It also explains why the experience can be so different among different individuals and why "one size fits all" treatment approaches do not fit all. In the next several modules of our course, we examine each of these domains separately, because within each there exists a great deal of complexity (Module 3=biological, Module 4=psychological, and Module 5=social/physical environmental). Then, in Module 6, we

explore ways to bring these domains back together in a more integrated fashion.



Current Diagnostic Criteria

In 2013, the American Psychiatric Association adopted a new diagnostic system, the **DSM-5** (APA, 2013), informed by decades of additional research into the epidemiology, etiology, and treatment of various psychiatric conditions. This is the main scheme for diagnosing substance use disorders currently used in the U.S. clinically, and increasingly adopted in research. The ICD-10 is in the process of being replaced by the ***ICD-11***. Fairly dramatic changes were seen in the criteria for the diagnosis of substance use disorders. The most dramatic was the change from two distinct categories, abuse, and dependence, to viewing these disorders on a continuum of severity. The list of 11 diagnostic criteria (see Table 1) reflect 4 categories of function:

- impaired control over use [items 1-4]
- social impairment/consequences [items 5-7]

- risky use of the substance(s) [items 8-9]
- pharmacological indicators/symptoms: tolerance, withdrawal [items 10-11]

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

1	Often taking alcohol or another substance in larger amounts or for a longer period th to limit yourself to 2 beers but ending up drinking 6)
2	A persistent desire or unsuccessful efforts to cut down or control use of alcohol or an believing your substance use is problematic and attempting to cut back on frequency so).
3	Spending a great deal of time in activities necessary to obtain, use, or recover from th another substance (e.g., spending days planning a drinking event/party then drinking the event/party, and needing a day or two to recover from the drinking event/party; where you will acquire the substances when you go on vacation or to travel out-of-to
4	Strong desire, craving, or urge to use alcohol or another substance (e.g., "needing" to driving in the car, with certain friends, or at the end of a meal)
5	Failure to fulfill major role obligations at work, school, or home resulting from recurr another substance (e.g., continually "dropping the ball," disappointing other people, fa of you at work, academically, at home, such as not feeding your children or pets or fai adequate supervision because you are intoxicated, high, or recovering from substanc
6	Continued use of alcohol or another substance despite persistent or recurring proble interpersonal relationships that are caused or made worse by the effects of alcohol or continuing to "get high" despite knowing that it is causing relationship problems with parents, siblings, or children)
7	Giving up or reducing important social, occupational, or recreational activities becaus substance use (e.g., no longer engaging in past hobbies/interests or work, family, fun substances or recovering from use; replacing your "life" with substance use)
8	Recurrent use of alcohol or another substance in situations where it is physically dan operating a car/motorcycle/boat or other vehicle while under the influence, engagin while under the influence or to acquire substances, risking harm to self or others)
9	Continuing to use alcohol or another substance despite knowledge of having a persist psychological problem that could be caused or made worse by its use (e.g., continuing on diabetes, liver disease, sleep patterns, or depression)
10	Developing tolerance for alcohol or another substance (see definition of tolerance)
11	Experiencing withdrawal symptoms or taking alcohol or closely related substance in o withdrawal symptoms (see definition of withdrawal)

Severity. The DSM-5 diagnosis scheme includes the

dimension of severity, based on the number of symptoms an individual is experiencing. Severity is determined as follows:

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

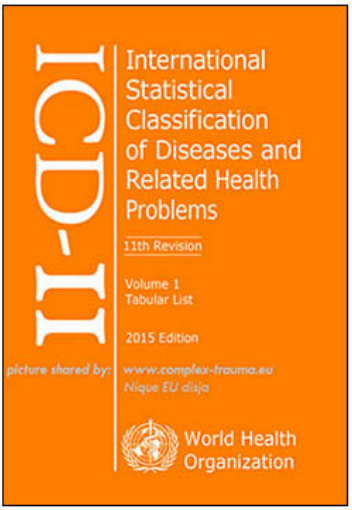
Types of SUD. While moving away from categorizing SUD in categorical terms (abuse/dependence) the DSM-5 (and the ICD-11) does distinguish between different types of substances involved. Nine types of substance use disorder are identified, each of which utilizes the 11 criteria and severity schedule above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

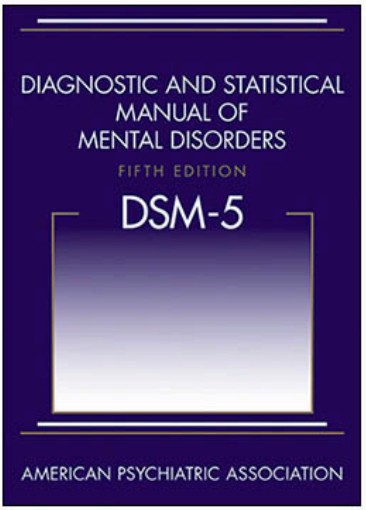
Table 2. Types of substance use disorder classified in DSM-5.

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

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

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





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

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

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

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

1. What do you think might be the similarities or differences between a person who experiences an alcohol use disorder and a person with a gambling disorder?
2. What about "disordered" internet gaming or other "dependence" on technology?
3. What do you think about people using the word "addiction" to describe how they feel about a favorite television show? What about advertisers describing games like Candy Crush as "addicting" to promote its popularity?

Ch. 2: Classification Systems for Different Types of Substances

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

Classification by Effects

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

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

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

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

Table 3. Stimulant Substances.

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

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

Table 4. Depressants and Dissociatives

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

Table 5. Cannabinoids

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

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

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

Table 7. Hallucinogens & Psychotomimetics

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

Table 8. Steroids

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

Table 9. Inhalants

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

Classification by DEA Schedule of Drugs

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

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

license to do so (e.g., by prescription from a licensed pharmacy) and it is illegal to distribute Schedule I drugs at all (with the exception of a few research or specially approved uses).

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

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

Level	Criteria	Examples
Schedule I	No accepted medical use in the U.S., lack of accepted safety for use under medical supervision, OR some narcotic medications that are used medically; all have a high potential for abuse	heroin, LSD, cannab "Ecstasy"/XTC, PCP "china white fentan approved for medic
Schedule II	High potential for abuse, with use leading to severe psychological or physical dependence; has accepted use in the U.S. under medical supervision	cocaine (and crack) opium poppy heads Tuinal®, Vicodin®, (OxyContin®), fenta Ritalin®
Schedule III	Potential for abuse exists but is not as high as Schedule I or II; moderate to low dependence potential, but higher risk than Schedule IV	ketamine, anabolic less than 90mg cod codeine), paregoric opium), lysergic aci
Schedule IV	Low potential for abuse or dependence.	Ativan®, Xanax®, V Ativan® (lorazepam Dalmane®, Konopi
Schedule V	Potential for abuse is lower than for Schedule IV drugs; preparations containing limited quantities of certain drugs with more stringent scheduling (certain narcotics).	Lomotil®, Lyrica®, 200mg codeine per

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

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Ch. 3: Trending Topics

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

Defining Addiction

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

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

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

ASAM American Society *of* Addiction Medicine

Important aspects of this definition are recognition of:

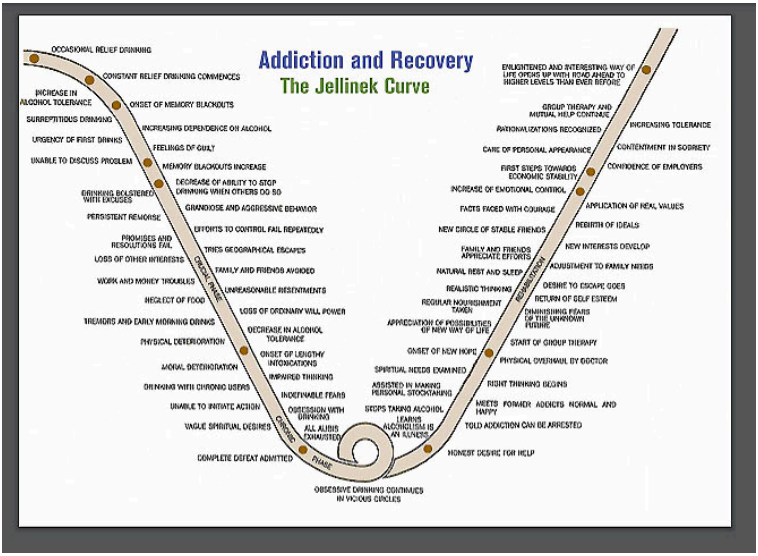
- the impact of addiction on biological, psychological/ emotional, social, interpersonal, and spiritual aspects of life;
- the brain-behavior nexus in the development and maintenance of addictive behavior;
- the common experience of cyclical relapse and remission, and
- the potential for problem progression.

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

Original disease model of addiction. The original disease model of addiction emerged during the 1950s and 1960s regarding alcoholism, viewing addiction as a primary disease, not secondary to other psychological conditions (Hartje, 2009). The original disease model of addiction was hailed as an important, less stigmatizing alternative than the prevailing moral model that placed blame on individuals for their addiction and deemed them deserving of its

consequences and punishment (Thombs, 2009). Viewing addiction as a disease, instead, allowed the person to be seen as the "victim" of an illness, deserving of compassionate care and medically supervised treatment (Thombs, 2009). In the disease model, an individual's choice to initially engage in substance may have been freely made; however, once initiated, the disease could take over: "intense cravings are triggered via physiological mechanisms, and these cravings lead to compulsive overuse. This mechanism is beyond the personal control of the addict" (Thombs, 2009, p. 561).

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



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

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

The original disease model and principles have greatly influenced assessment and treatment practices over the past 60 to 70 years. There exist several points around which the original disease model of addiction has been challenged.

Heterogeneity challenge to the original disease model. Longitudinal studies documenting the natural course of alcoholism demonstrated significant inconsistencies with a disease progression premise: multiple patterns were observed among men still alive 60 years after beginning the study, including continued alcohol abuse, stable abstinence, and return to asymptomatic/controlled

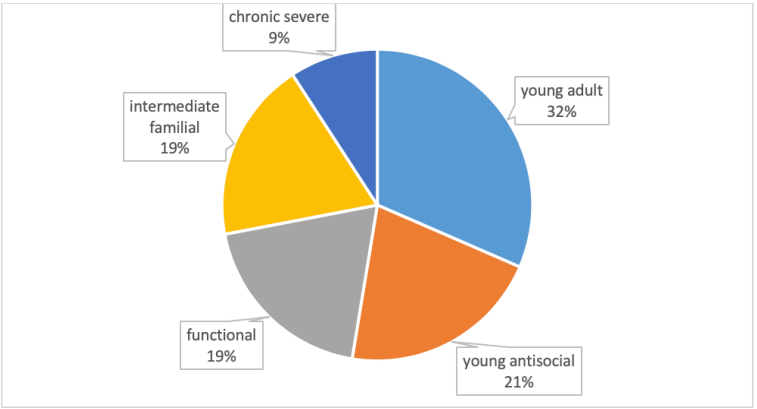
drinking (Vaillant, 2003). Tremendous individual variation exists in patterns of addictive behaviors, as well as the severity of problems experienced by individuals at different points in time. Jellinek (1952) admitted that his was an "average trend" model in which individuals do not necessarily exhibit all of the symptoms associated with a phase, may differ in the sequencing of symptoms, and may differ in the duration of each phase; furthermore, "nonaddictive alcoholic" individuals may experience the identified negative consequences of alcoholism without experiencing a loss of control over drinking, and women may experience the disease differently.

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

Subtypes versus stages of disease. There exist marked differences in how substance misuse/SUDs are expressed even within a single substance type. Challenging Jellinek's stage model of alcoholism, for example, is evidence of heterogeneity in "types" of alcoholism derived from a national sample (U.S.). The investigators based their typology on clinical characteristics of individuals meeting criteria for an alcohol dependence per the DSM-IV-R criteria that preceded the DSM-5 (Moss, Chen, & Yi, 2007). This analysis of U.S. National Epidemiological Survey on Alcohol and Related Conditions (NESARC) data led the authors to identify five "subtypes" of alcohol dependence, demonstrating clinical heterogeneity within the single diagnostic classification. The subtypes they identified were based on how participants clustered

on diagnostic criteria, age of onset, family history, and presence of other co-occurring disorders. The five statistically determined clusters they identified were labelled: young adult, young antisocial, functional, intermediate familial, and chronic severe subtypes (see Figure 1). The groups demonstrated differences in their patterns of drinking, help-seeking, and response to intervention, as well. This study, based on a large, nationally representative sample reflected heterogeneity among persons engaged in a specific addictive behavior, and the wisdom of avoiding stereotypes about them—for instance, while the chronic severe subtype was the least common, it reflects a common stereotype of alcohol dependence.

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



Treatment and the disease model. Additional important challenges to the disease model of addiction appear in the literature. Asserting that formal treatment

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

Abstinence only prescription based on disease model. Viewing abstinence from substance use as the only defense against "disease" recurrence and the only reasonable goal for a person experiencing a substance use disorder has been challenged. Complete abstinence from all psychoactive substances is at one end of a continuum in treatment strategies, commonly applied in U.S. medical practice (Glenn & Wu, 2009). A debated position is that the continuum of recovery includes controlled substance use, including the type of substance which a person previously used problematically. Between these positions is a question of whether psychoactive medications used to treat substance use disorders reflects recovery or is only a prelude to recovery not achieved until these medications are no longer needed. This question relates to an assertion that substituting one substance for another, despite its being safer, more controlled, or reducing harm, simply maintains the disease rather than offering a cure.

The word "sobriety" originally, historically implied temperate, moderated indulgence, not necessarily complete abstinence—an

abstinence interpretation emerged during the 1900s (Glenn & Wu, 2009). Evidence since the 1970s indicates that some individuals achieve controlled drinking despite having previously engaged in an "out-of-control" drinking pattern, contrary to "the prevailing belief that any alcohol consumption causes an inevitable loss of control over one's alcohol use" (Klingemann, 2016, p. 436). The debate about "controlled drinking," "reduced-risk drinking," and "moderation management" continues, and it is unclear how the evidence for and against it might apply to other substances and addictive behaviors. On the issue of the use of pharmacotherapy to assist in controlled drinking, recent meta-analysis concluded that three medications showed controlled drinking outcomes superior to a placebo, but the effects were small and inconsistent across studies (Palpcuer et al., 2018). With or without medication, reduced-risk drinking (RRD) is seen in many Western European countries as one pathway out of addiction, and a legitimate treatment goal (Klingemann, 2016). As previously noted, the ability to engage in controlled use following a substance use disorder may vary by individual, severity of the problem, and characteristics of the substances or addictive behaviors involved.

Closely associated with the abstinence issue lies an additional point of contention with the final disease model of addiction, the expectation that substituting a different substance for the primary addictive behavior (e.g., misuse of alcohol, cannabis, or opioids) simply continues manifestation of the same disease of "addiction" where the symptoms persist, as does the pattern of disease progression. This stance contributes to the hesitancy expressed by some practitioners that medically assisted treatment (MAT) and the use of pharmacotherapies to treat substance use disorders maintains the (incurable) disease rather than treating it. Evidence of the effectiveness of these approaches for many persons, including eventual weaning from medication, contradicts this contention.

Loss of Control Concept. The original disease model of addiction expressed another point with which scholars and practitioners have taken issue: applying "loss of control" as a defining criterion. The prior moral model attributed individuals' use/misuse of alcohol, tobacco, or other drugs to moral failure or personality weakness, holding them "personally responsible for creating suffering for themselves and others" (Thombs, 2009, p. 561).

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

Contemporary brain disease model and biopsychosocial perspective. As previously noted, recognition of the brain-behavior nexus in the development and maintenance of addictive behavior is important and necessary to

understanding, intervening around, and recovery involving addictive behavior and related problems. Evidence concerning the neurobiology of substance use and mechanisms involved in the transition to substance use disorders has expanded in many directions over the past two decades, contributing to a widening variety of treatment and prevention intervention strategies (Volkow & Koob, 2015; Volkow, Koob, & McLellan, 2016).

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

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

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

Biopsychosocial

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

Harm Reduction

A somewhat contested topic related to substance misuse and related problems is ***harm reduction***. First appearing in the literature during the late 1980s and early 1990s, the term "harm reduction" was used to describe attempts to reduce adverse consequences associated with substance misuse, without necessarily eliminating substance use (Single, 1995). Two general levels of harm reduction effort emerged in the literature: clinical practice and policy

interventions. Underlying harm reduction is recognition of the potential harms associated with engaging in addictive behavior (e.g., substance misuse or problem gambling), as well as knowing that some individuals will continue to engage in these behaviors, at least for an unknown length of time, despite the potential for harms to self and others. "The essence of the concept is to ameliorate adverse consequences of drug use while, at least in the short term, drug use continues" (Single, 1995, p. 287).

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

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

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

Examples of harm reduction practices at the clinical level include:

- nicotine replacement therapy to reduce harms associated with

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

While harm reduction as a public health and social work strategy makes intuitive sense on the surface, controversy revolves around philosophy and implementation, led to some degree by a misunderstanding of harm reduction (Drucker et al., 2016). One argument against harm reduction strategies is that it may be mis-perceived as sanctioning the problematic behavior. The evidence on this is mixed, however. For example, while zero-tolerance/abstinence-based messaging was more effective in curbing college students' future drinking in several studies (Abar, Morgan, Small, & Maggs, 2012; LaBrie, Boyle, & Napper, 2015), in another, this was true only among students who currently consumed two or fewer drinks per week; harm reduction messaging outcomes were more favorable among students currently engaged heavy drinking (Napper, 2019). Thus, the anti-harm reduction argument that it seems to sanction the behavior, thereby contributing to the problematic behavior, is only partially supported by evidence. An argument that harm reduction (reducing the negative consequences) interferes with motivation to seek

treatment and/or quit engaging in the problematic behavior is also countered with the argument that, as a result of engaging in harm reduction programming, individuals may then become encouraged to engage in treatment to reduce or cease substance misuse (Drucker et al., 2016). An argument against nicotine or opioid replacement therapies is that the person continues to experience substance dependence. However, use of these therapies may allow the individual to gradually become weaned from dependence in a controlled manner, supported by behavioral therapies. While this is argument is offered in support of e-cigarettes/vaping as a harm reduction tool, evidence is mounting that significant risks of harm are associated with these devices (including injury from malfunctions/battery problems, chemical exposure not being reduced as much as advertised, worsening of the nicotine dependence, and poisoning of children and pets from the liquid nicotine).

Recovery Orientation

A ***recovery orientation*** refers to a host of values, beliefs, and behaviors related to how individuals engage in and experience the process of recovery from a SUD (Bersamira, in press). The recovery orientation is fundamentally informed by the individuals' own definitions of the problems, solutions, and subjective experiences, rather than those being imposed by others. Built into this orientation are issues such as having individuals define for themselves what constitutes "recovery"—this may or may not include abstinence as a goal, for instance. Another aspect has to do with adopting a holistic view where individuals' recovery is embedded in a context of all life structures, functions, and wellness, including their future growth and development as a person, not just changes in past substance use/misuse behavior (Kaskutas et al., 2014). Thus, recovery does not simply mean achieving the absence of disease, it means promoting wellness across all life domains.

Many individuals and professionals actively engage in advocacy

related to a general recovery-oriented movement, promoting recovery-oriented services and policy (Bersamira, in press). This orientation includes engaging indigenous and professional services and relationships in supporting individuals' long-term recovery (and their families), as well as shaping the culture of communities and policy (White, 2008). For example, peer support systems are often an integral aspect honored and incorporated in a recovery orientation: peers being others who have lived the experience and found their own pathways to recovery. In other words, recovery-oriented systems of care differ quite markedly from traditional treatment systems: their services are more person-centered, self-directed, and strengths-based (Bersamira, in press).

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Ch. 4: Summary

In the readings for Module 2 you read about:

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

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

Module 2: Key Terms

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

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

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

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

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

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

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

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

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

substance use: Introduction of psychoactive substances into the body.

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

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

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

Module 2: References and Image Credits

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PART III

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

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

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

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

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

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

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

Reading Objectives

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

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

disorder;

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

Ch. 1: Genetic Influences

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

Another fact that has emerged from decades of research is that there is no one specific "addiction gene" that applies to all of the different types of substances. Some of the genes involved are very specific to certain substances—what may "pull" for an alcohol use disorder may not be "pulling" for a problem with cocaine, for example. Some genes are not specific to substance use disorders per se, but to a class of problems that have substance misuse as an element—for example, depression. The more we learn about specific combinations of genes that might be involved, exciting new biological tools for treating or even preventing addiction may emerge, including medications and perhaps even immunizations someday. For a little basic background (possibly review) in understanding genetics, see the keywords list for ***DNA***, ***alleles***, ***genes***, ***chromosomes***, ***genome***, ***genotype***, ***heritability***, and ***phenotype***.

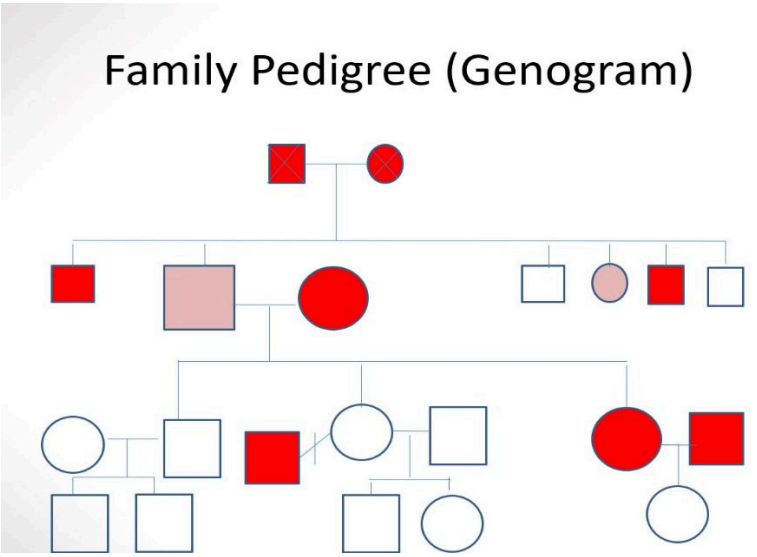
Four general lines of research contribute to our understanding of the role played by genetics in substance misuse and substance use disorder (SUD): family pedigree, twin, adoption, and genome studies.

Family pedigree studies. Early genetic influence research relied on tracing the patterns with which a particular phenotype appears in multiple generations of a family—alcohol misuse and alcohol use

disorder (AUD) is an example. These familial patterns become apparent when a pedigree chart is created (in social work practice, a family "genogram" is sometimes used in assessment; see Hartman, 1995). The observed pedigree patterns generally supported investigators' hypothesis that the development of alcoholism has a genetic component—it is not entirely driven or dictated by genetics but is influenced by genetics. The more genetically close (proximal) in relationship, the greater the influence. For example, with alcohol use disorders, the influence of parents is stronger than the influence of aunts/uncles.

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

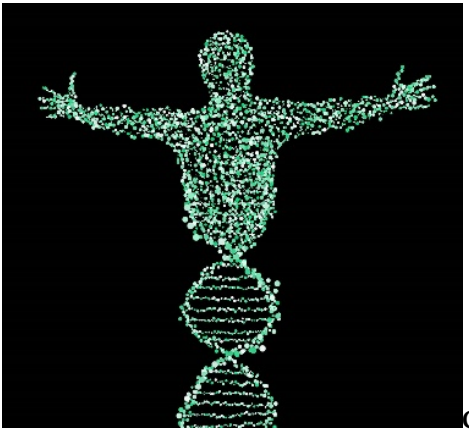
Figure 3-1. Sample family pedigree (genogram) tracing alcohol use disorder.



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

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

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

 **Genome studies.** More recent lines of research go beyond answering the question "do genetics matter" to more specificity about "how genetics matters." The human genome is a person's complete set of DNA, represented in virtually every cell of the body. The Human Genome Project, completed in 2003, resulted in a generic "map" of the approximately 20,000-25,000 genes in the human genome (see the national Human Genome Research Institute's genome.gov/about-genomics/fact-sheets/A-Brief-Guide-to-Genomics). This knowledge contributes greatly to understanding complex health problems (like substance use disorders) resulting from multiple genetic factors acting together and with the environment. Genome-wise association studies (GWAS) approached the study of substance misuse and SUD (and other phenotypic outcomes) in a unique manner: searching for common variants in allele frequency across the entire genome and then determining what phenotypic differences were associated with those variants (Bares & Chartier, in press). The GWAS approach is credited with identifying a genetic basis for phenotypes including heavy versus light amount of cigarette smoking or alcohol consumption, and developing nicotine or alcohol use disorder (Hancock, Markunas, Bierut, & Johnson, 2018).

Additionally, the Collaborative Studies on Genetics of Alcoholism (COGA) has established a database of information from over 10,000

individuals across multiple sites and over many years. The variables included measures of clinical, neuropsychological, electrophysiological, biochemical, and genetic factors, as well as individual and family histories of drinking behavior, from four groups of individuals (see http://pubs.niaaa.hih.gov/publications/arh26-3/214-218.htm):

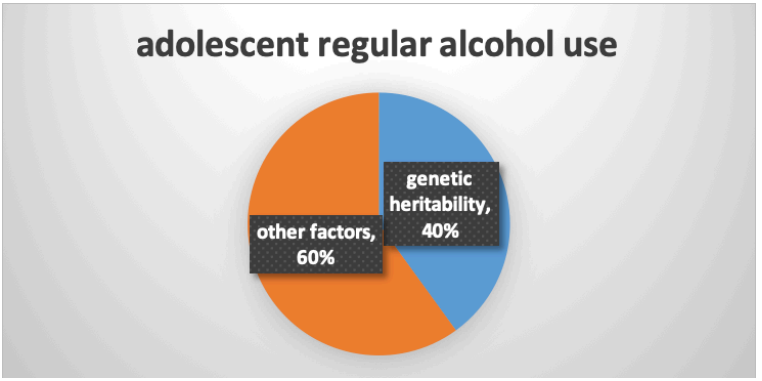
- those meeting criteria for alcohol dependence (DSM-IV-TR criteria);
- those "at-risk" of alcohol dependence by virtue of their low level of response to alcohol—higher baseline tolerance, needing to consume greater amounts of alcohol than others in order to feel the effects is recognized as a vulnerability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

What Is Known About The Genetics of Substance Misuse

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

Genetics plays a significant role. As described in terms of the family pedigree, twin, and adoption studies, there is clearly a genetic influence on the development of alcohol use disorders. Less is known about other substances, however, there is convincing evidence from these and genomic studies that the probability of substance use becoming a substance use disorder (SUD) is influenced genetically (heritable) for many different substances. Not only is the emergence of SUD partially directed by genetics, but there appears to be a genetic contribution to the initiation and

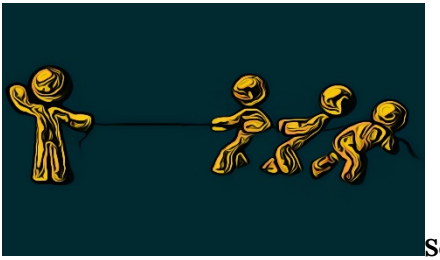
regular use of at least some substances, as well. For example, initiating tobacco use during adolescence was anywhere between 35%-80% heritable across different studies, regular tobacco use was between 40% to 50% heritable, and regular alcohol use was about 40% heritable (Bares & Chartier, in press). The evidence also demonstrates that it is not entirely driven by genetics—environmental factors play a significant role, as well (Bares & Chartier, *in press*).



Multiple genes involved. Evidence points to multiple genes contributing to substance use disorder (***polygenic***), even to a single type of substance use disorder (e.g., alcohol use disorder). Early genetic studies attempted to determine which specific gene or genes were candidates for playing a significant role in substance misuse behavior or SUD based primarily on their control of important, relevant biological processes; candidate gene studies generally showed inconsistent results, however (Bares & Chartier, *in press*). More recent approaches to understanding polygenic phenomena involve aggregating the many small effects each gene might

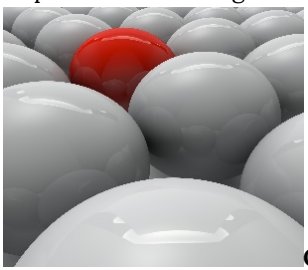
contribute, resulting in a weighted total genetic effect (polygenic score, or PGS) taking into account the vulnerability and protective genes a person might have (Bares & Chartier, *in press*). In other words, we cannot point to any one gene as the "cause" of even a single type of SUD, much less SUDs in general.

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



Severity is determined by specific chromosomal regions. Genetic influence is not simply occurring at the level of specific chromosome sites, but also in ***chromosomal regions*** (areas where multiple chromosomal sites cluster) and in various polygenetic combinations (multiple genes interacting). What this means is that a person may have various genetic forces pushing for and against developing substance misuse or SUD problems in a kind of genetic tug-of-war. As a result,

we see a wide range of phenotypic expression in the population as a whole—the problem is heterogeneous, not "one size fits all."



Common versus specific origins. Analysis of a vast body of science provided answers to the question of whether SUD involving different types of substances has a shared, common genetic origin or whether each type of substance has its own unique genetic influences (Li et al., 2011). The answer is not simple: some genomic areas appear to be shared across different types, while other areas are substance-specific (Begun & Brown, 2014). There does appear to be some shared commonality in genetic vulnerability to nicotine, alcohol, and cannabis dependence, at least among men. The underlying common genetic factors, however, fail to explain the high degree of variability in phenotypic expression (Palmer et al., 2012). There exist specific genetic factors, as well, also operating at the same time, including specificity for alcohol, tobacco, marijuana, and cocaine (Palmer et al, 2012). One common underlying genetic factor may be the presence of genetics linked to depression—for some individuals, depression and SUD have common genetic influences, but this is not true for everyone with either/both experiences.

SUD heritability was stronger among men. While heritability of alcohol use disorder was observed for both men and women, the case appeared to be stronger among men. In other words, environmental factors explained a greater

proportion of alcohol use disorder among women (Kendler et al., 1992; Jang, Livesly, & Vernon, 1997). However, this gender-based differentiation is less noticeable in recent cohorts than historically, at least for alcohol or nicotine dependence (Palmer et al., 2012).

Summary

In answer to the "Is it genetic?" question about substance use disorder, the evidence from multiple sources indicates "sort of." The situation is complex. Some aspects of substance use, substance misuse, and substance use disorder are influenced by genetic forces, but there is a great deal about each of these behaviors/experiences that is influenced by other than genetic forces, too. Furthermore, the genetic forces do not all push in the same direction or to the same extent—some forces push for and other against the problems emerging. We also know that SUD is not a single phenomenon—susceptibility differs for different substances. For example, a propensity toward alcohol use disorder may or may not align with propensity for nicotine or cocaine use disorder. And, the genetic forces related to certain co-occurring problems may also relate to the propensity for developing a specific type of SUD.

Your Family Pedigree

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

Ch. 2: Neurobiology and Substance Use

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

Learning about the neurochemistry

actions of specific substances in neurophysiology also helps us understand the actions of different substances on the brain-behavior link. Here we will look at neurotransmitters and their role in the experience of substance use/misuse. This knowledge helps investigators develop intervention strategies for treatment, relapse prevention, and even preventing the development of substance use disorders. These biologically based strategies include medications and the use of mindfulness meditation and neurofeedback approaches.

Neuroanatomy and Function

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

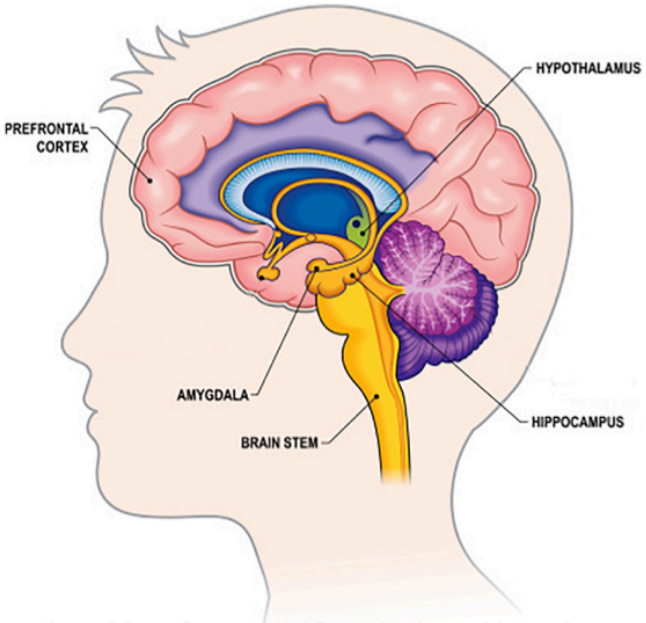
Limbic system. The ***limbic system*** helps regulate basic drives, emotions, arousal and attentiveness (Begun & Brown, 2014). As such, it helps coordinate the neurobiological experience of stress and the reward system triggered by exposure to drugs. The ***amygdala*** and ***nucleus accumbens*** are two important components of the limbic system with regard to substance misuse (Logrip, Zorilla, & Koob, 2012), along with the***hippocampus***.

Amygdala. The amygdala plays a central role in emotional responses to internal and external stimuli—pleasure, fear, anxiety,

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

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

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



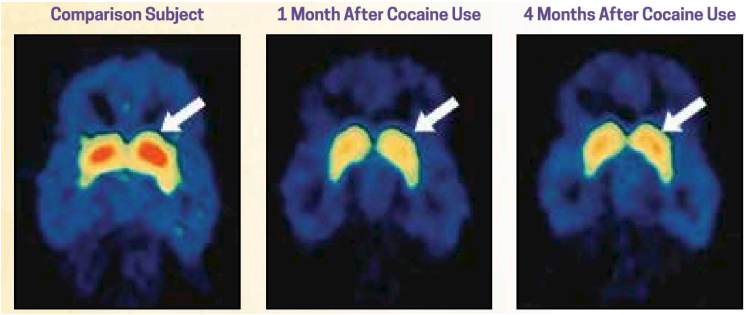
adapted from Queensland Brain Institute qbi.uq.edu.au

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

responsible for helping someone control substance misuse is an area impaired by substance misuse (Azmitia, 2001).

Changes in Brain Function

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



Developmental Impact

A great deal of attention to the developmental effects of exposure to alcohol and other drugs has been directed to two life periods:

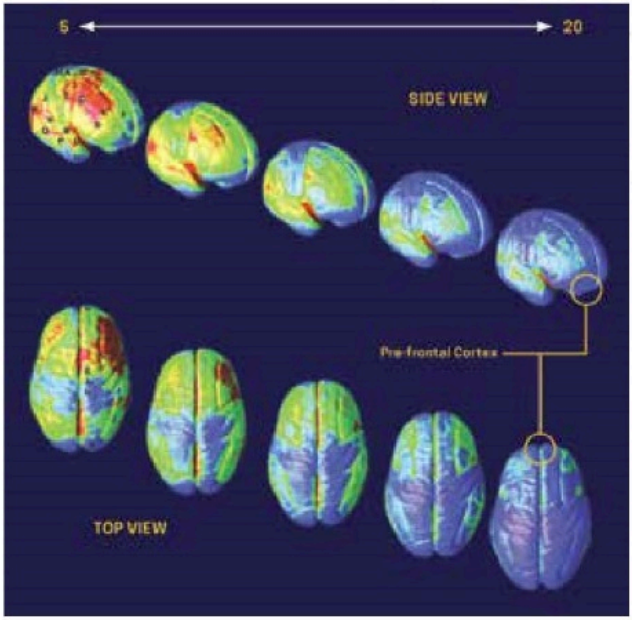
prenatal exposure and substance use during adolescence/emerging adulthood. These two developmental periods have an important commonality: these are periods when the brain is naturally undergoing rapid developmental growth or change. Thus, introducing substances that affect the brain can have more pronounced, amplified, and pervasive long-term effects.

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

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

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

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



Source: PNAS 101:8174–8179, 2004

Adolescent/emerging adulthood exposure. Shortly before and during puberty the human brain begins to undergo dramatic remodeling changes. The physical changes, to a large extent, involve reorganization of the connections between neurons and communication pathways between brain regions, particularly in the prefrontal cortex. On one hand, a great deal of neuron "pruning" takes place, trimming out a great many underused or unused connections between neurons. On the other hand, myelination of existing neurons enhances connections between neurons that remain linked (Siegel, 2014). These two processes make the brain more efficient, better integrated, and capable of higher order functioning, but do not happen evenly and at the same time in all brain regions. The result is *emotional* functioning similar to that of adults but *cognitive* functioning that is as yet under-developed in terms of decision making, inhibitory control, planning, and working memory (Meredith & Squeglia, *in press*). Additionally, the adolescent

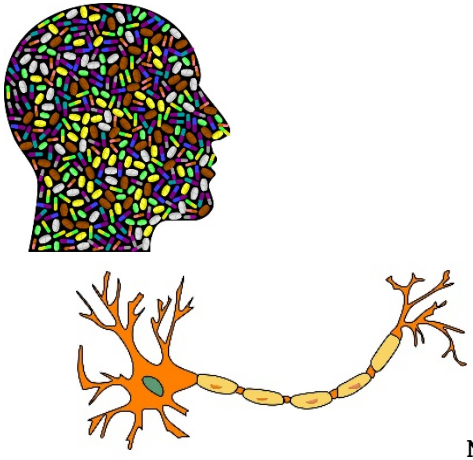
brain is characterized by "heightened reward sensitivity and underdeveloped cognitive control that contribute to risky behaviors, including escalating substance use" (Meredith & Squeglia, *in press*). Heightened reward sensitivity suggests that the positive reinforcement experienced with substance use is experienced as more intensely positive (stronger reinforcement) than what is experienced by individuals later in life. The brain revision process normally tapers off from about ages 20 to 25. This image (from NIDA, 2018) shows how the concentration of grey matter shifts from age 5 to 20—the shift from yellow to blue in these images.

Thus, the brain is quite sensitive to developmental consequences of exposure to psychoactive/psychotropic substances up until age 25. The use of alcohol or other substances during these years can have profound, lasting effects on the still-developing brain; effects which have significant implications for how people think, behave, and feel, as well as for susceptibility to developing substance use disorders later in life. "In studies of drug use, an earlier age at which drug use was initiated is consistently related to a greater level of later drug-related problems," (Hawkins et al., 1997, p. 281), making a delay in age of substance use initiation an important prevention strategy. Chances of developing severe substance use disorders is higher among individuals whose substance use began before age 15 years; "the biggest reduction in risk with deferred age of onset occurs when first use is postponed beyond age 15" (Robins & Przybeck, 1975, p. 184). Alcohol dependence was found to be four times more likely and alcohol abuse twice as likely among individuals whose age of drinking onset was before age 15 compared to individuals whose onset was delayed to age 21: "Overall, the risk for alcohol dependence decreased by 14 percent with each increasing year of age of drinking onset" (NIAAA, 1998). Deficits in adolescent brain functions and cognitive performance were observed with as little as 20 drinks per month, particularly if binge drinking was involved (Squeglia, Jacobus, & Tapert, 2009); some but not as great a level of divergence from their peers was detected with marijuana use. Finally, consider that a person's overall health and

development may be affected by poor nutrition, physical trauma or injury, or exposure to diseases that often accompany substance misuse.

Neurochemistry/Neurophysiology and Function

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

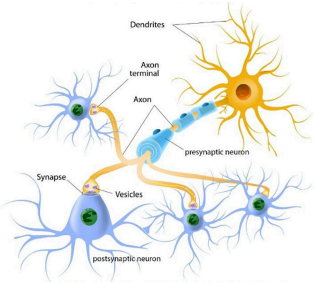


Neuron activity. The neurochemistry of substance use operates largely at two points. The first concerns the glial cells and how much energy they can provide to neurons—the loss of glial cells or impeding their ability to provide energy has a negative impact on neuronal activity. The second concerns the ways that neurons communicate. Neurons physically pass ***neurotransmitters*** (molecules of naturally occurring brain chemicals) between each other as their mechanism for

communication. Whether one neuron activates the next one depends on whether neurotransmitters are sent, whether those neurotransmitters are received by the next neuron, the amount of neurotransmitter sent and received, and the rate at which the neurotransmitters are reabsorbed after a "message" has been sent.

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

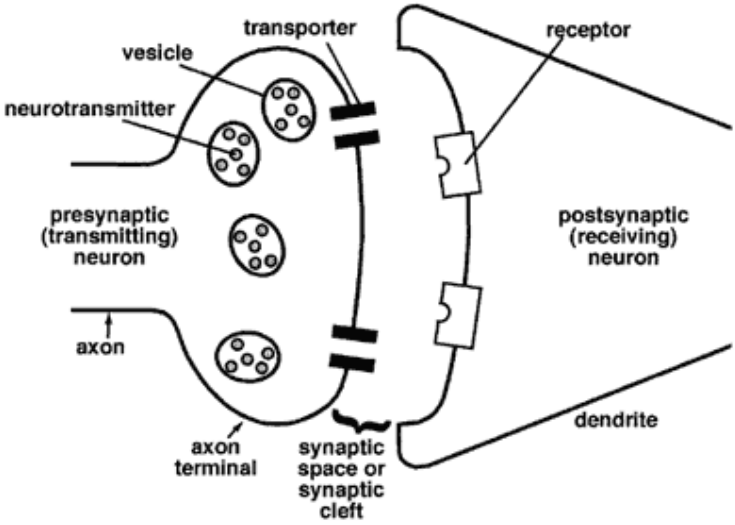
Figure 3-2. Neurons and how they communicate



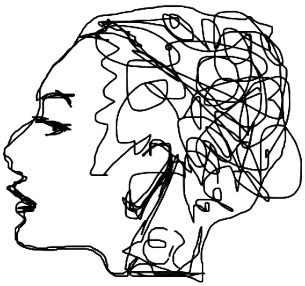
The presynaptic (first) neuron releases neurotransmitter molecules (stored in the vesicles) into the synapse between it and the postsynaptic (next) neuron. The postsynaptic neuron "receives" the neurotransmitter chemical if it has the right neurotransmitter ***receptors***—kind of like a lock and key system. Neurotransmitters need the right receptors in order to "dock" and influence the postsynaptic neuron: if the right receptors are available, the neurotransmitter delivers the message but if the right receptors are not available, the neurotransmitter has no effect and just sits in the synapse. If the message is received by the postsynaptic neuron, it can now pass the message along to the next neurons in line. In the meantime, ***transporters*** retrieve and return the "used" neurotransmitter molecules back into the presynaptic neuron's

vesicles in preparation for sending a future message (see Figure 3-3). If a neuron has released its neurotransmitter molecules, it cannot send new messages until the supply has been restocked.

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



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



Types of neurotransmitters. Different types of neurotransmitters have different impacts. For example, some play a more ***excitatory*** role, while others play a more ***inhibitory*** role. Excitatory neurotransmitters increase the likelihood that the receiving (postsynaptic) neuron will be triggered into activity; inhibitory neurotransmitters suppress this kind of activity. Most types of neurotransmitter are either excitatory or inhibitory; a few can be either (e.g., dopamine). Different types of neurotransmitters are more concentrated in specific brain regions—while they may be distributed throughout the brain, they are not evenly distributed. This is why different substances "trigger" certain brain regions more than others—their effects are produced through their influence on the neurotransmitter communication processes and those neurotransmitters are more concentrated in certain regions.

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

- ***dopamine*** has both excitatory and inhibitory effects, depending on the nature of the receptor sites involved, is associated with the brain's reward systems, and is increased to abnormal levels by substances such as alcohol, cocaine, and heroin (influencing their addictive potential);
- ***endorphins& enkaphlins*** are two neuropeptides (rather than neurotransmitters) that play a role in producing some of the

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

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

- We used to believe that each neuron could only release one type of neurotransmitter. More recent research indicates that in many cases the same neuron can release two and possibly more types depending on the frequency of the stimulation it receives—at one frequency it might release one type of neurotransmitter, at another frequency it might release a different type.
- Most neurotransmitters occur naturally as important

chemicals in other parts of the body (including the peripheral nervous system and other organs) where they have other health-related functions, not just in the brain (central nervous system). For example, the human body naturally has opioid and cannabinoid receptors that are meant to respond to naturally occurring (endogenous) chemicals to control pain, reward certain life-supporting behaviors, and influence learning and memory. These receptors are also responsive to introduced chemicals (exogenous) which are often introduced in much higher doses than naturally occur—from using cannabis/marijuana or opioid drugs. Opioid receptors are also involved in responses to alcohol.

- Neurotransmitter release is triggered by many natural behaviors, not just by alcohol and other substances. For example, dopamine release is involved in the natural reward systems associated with food, sex, humor, pair-bonding (mates), listening to music, and video games. The addictive potential of a psychoactive drug increases when the concentration of dopamine released is higher compared to what is released by natural behaviors (Johnson, 2014).
- Fast uptake of a drug, for example getting it to the brain by injection rather than ingesting it orally, produces a stronger "high" and therefore a greater potential for addiction. This is because more dopamine is released at once, so it is more rewarding (Volkow et al., 2010).

Homeostasis

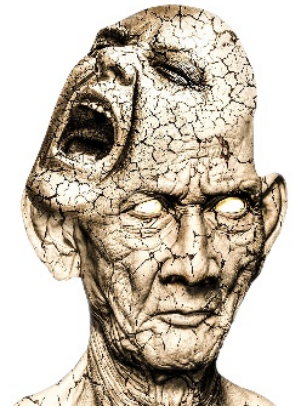
One hallmark of the human brain is its adaptability (neuroplasticity), whereby its various functions adjust to conditions in order to maintain overall balance or ***homeostasis***. This adaptability gives rise to acquired tolerance when a substance (or type of substance) is used repeatedly over time. In this chapter we examine how

homeostasis plays a role in the development of tolerance, as well as the biological basis of the substance withdrawal experience. In addition, we examine why the age at which the brain becomes exposed to substances matters and a few basic principles concerning pharmacotherapy for treating substance misuse and SUD—we look into this last topic more deeply later in the course.

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

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

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



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

Why does this matter? These negative emotional and psychological states make it difficult to sustain motivation to avoid using alcohol or other substances and contribute to the pressure a person might feel to relapse into using again. Depending on the nature of the substances involved, withdrawal may lead to decreased dopamine, serotonin, or GABA, as well as increased norepinephrine or glutamate. Knowing about these links between neurotransmitter changes during prolonged withdrawal from using a substance contributed to the development of several medications to help manage these negative experiences and perhaps help a person sustain a "quit" attempt over time (pharmacotherapy). We will learn about specific pharmacotherapy medications in Module 13. Another reason this matters is that during withdrawal and early recovery from many types of substance use disorders, the risk for suicide is greater than in the general population because of these brain-behavior processes.

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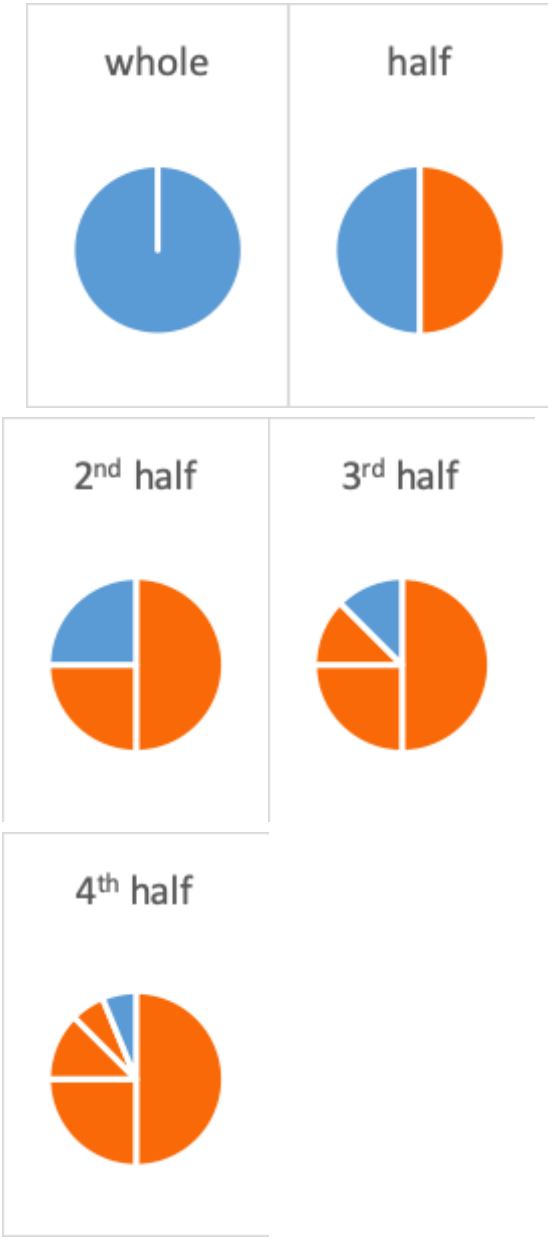
https://ohiostate.pressbooks.pub/substancemisusepart1/?p=112

Ch. 3: Basic Pharmacokinetic and Psychopharmacology Principles

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

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



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

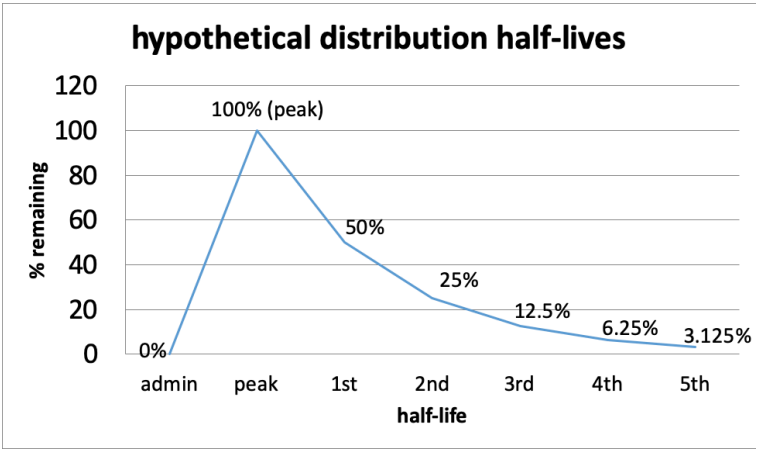
this means 50% remains (100% – 50%=50%).

- The second half-life is where 50% of what remained after the first half-life is gone—in other words, another 25% is gone (half of 50); together this means the original 50% + next 25%=75% of the peak level is gone, or only 25% remains (100% – 75%=25%).

- The third half-life is where 50% of what remained after the second half-life is gone—12.5% is half of 25%, so now 75% + 12.5%=87.5% of the peak level is gone; only 12.5% remains (100%-87.5%).

- And so on, until virtually none remains.

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

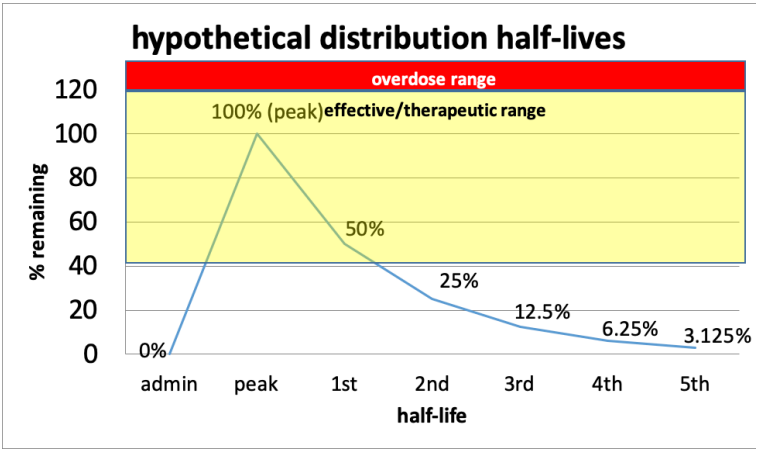


Different drugs, even within the same class of drugs, differ in terms of their half-lives as well as their range of effect—with medicine this would be called the therapeutic range. In other words, one drug might stop having an effect at the first half-life while another may still have an effect at the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

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

In this hypothetical example, a person might need to take more of the drug at the point where 50% remains (first half-life) in order to maintain an effective dose, but it is going to be important to avoid

a peak dose that takes that person into the overdose range. What is known about half-lives, effective dose range, and overdose range is based on averages across individuals—it may differ for a single individual and by various conditions (including the person's overall health and presence of other drugs). Individuals differ somewhat in how they metabolize drugs. It also is based on drugs of known composition—produced under controlled pharmacy conditions. You can see why drugs manufactured in uncontrolled conditions (e.g., "meth" labs, foreign labs, homemade) can be so much more unpredictable.



In this hypothetical example, consider that it took 1 hour for the drug to reach its peak level and that the half life is 3 hours long. That means at about 4 hours, the person will need to re-dose to maintain a therapeutic dose level or level where the effects remain in the desired range. If we are concerned about withdrawal symptoms, that is the point where the symptoms might begin to be experienced with this hypothetical drug. By about 16 hours, the person will have very little of the drug remaining in the body (5^{th} half-life).

Metabolites. The breakdown process for many substances is not as simple as "there and gone." In many cases, the process of metabolizing a drug or other substances happens in a sequence of steps, and the intermediary products may exert effects themselves.

For example, alcohol (ethanol) is first metabolized (broken down) into another chemical called acetaldehyde. Acetaldehyde is toxic and thought to be responsible for many of the "hangover" symptoms associated with alcohol consumption, as well as with the increased risks for cancers. Fortunately, acetaldehyde does not stick around very long as it is metabolized into a less toxic chemical, acetate. Acetate is then metabolized into carbon dioxide and water. The enzymes responsible for the metabolism of ethanol into acetaldehyde (alcohol dehydrogenase, or ADH) and of acetaldehyde into acetate (aldehyde dehydrogenase, or ALDH) are both, to a large extent, under genetic control. This contributes to the observed phenotypic differences in individuals' responses to drinking that we previously explored in discussing genetics—this is the mechanism through which genetics operate. We will look into this in more detail in our module focused on alcohol.

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

Agonists. An ***agonist*** activates specific types of receptor sites in the brain or elsewhere in the body, causing a specific effect. For example, THC is a chemical in cannabis (marijuana) that activates the naturally occurring cannabinoid receptors in the brain. This is how it produces its psychoactive effect. This principle can be used in treating substance use disorders. For instance, a drug that activates the opioid receptors in the brain can reduce or eliminate withdrawal symptoms by acting like the substance that has ceased to be used. This is why methadone can help in the treatment of heroin/opioid use disorder—it acts enough like the heroin/opioid to help without the added risks and potential harms of using the

original substance even if the person continues to experience a dependence on the class of substance involved. (Methadone itself is an addictive substance, but consistency in quality and dosing can be more carefully controlled and it can be more gently weaned over time to further reduce the likelihood of relapse.)

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

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

Ch. 4: Summary

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

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

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

Module 3: Key Terms

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

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

amygdala: location in the brain associated with emotion.

anhedonia: inability to experience pleasure/happiness.

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

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

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

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

chromosomal regions: sections of a chromosome.

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

dizygotic twins: twins developing from two different fertilized eggs.

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

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

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

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

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

epinephrine: also known as adrenaline, a stimulant/arousing

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

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

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

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

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

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

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

glutamate: an excitatory neurotransmitter.

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

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

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

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

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

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

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

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

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

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

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

neurotransmitters: types of molecules involved in communication between neurons.

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

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

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

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

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

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

postsynaptic neuron: a neuron receiving communication from another neuron.

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

presynaptic neuron: a neuron sending communication to another neuron.

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

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

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

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

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

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

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

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

Module 3: References and Image Credits

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PART IV

MODULE 4: PSYCHOLOGICAL MODELS OF SUBSTANCE MISUSE

Introduction

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

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

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

Reading Objectives

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

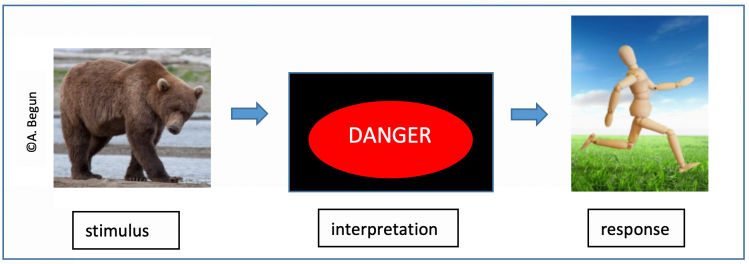
- Explain how cognition, information processing, learning, social learning, rational/planned behavior, developmental, psychodynamic, attachment, self-medication, personality, psychopathology, expectancies, and cravings model relate to substance misuse;
- Describe the relationship between brain-mind-behavior;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

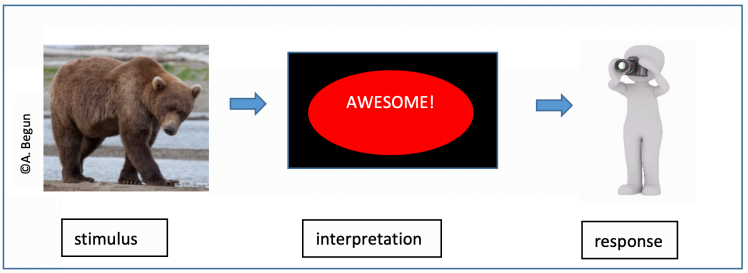
Ch. 1: Cognitive and Learning Theories

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

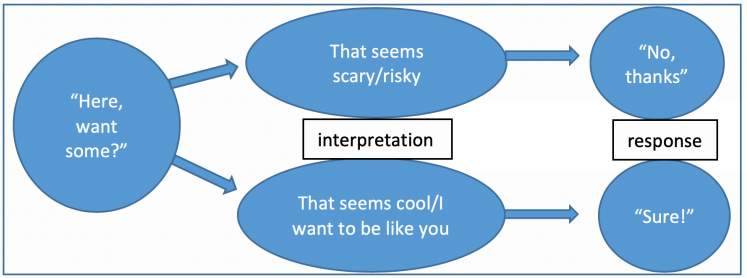
Cognitive processing

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





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



Here is another way in which cognitive processing–interpreting situations–is relevant. Consider the body of evidence concerning women becoming less aware of (or less uncomfortable with) situational cues concerning their risk of being sexually assaulted as their blood alcohol concentration rises to or above that specified as unsafe for driving–0.08 (Davis et al., 2009; Testa & Livingston, 2009). Substance use can impair a person's interpretation of the potential riskiness of certain situations, which in turn can diminish their capacity for self-protection and early termination of coercive

interactions. Practices like pre-planning for one friend designated as non-drinking during an outing and "having your back" may be employed by women who plan to engage in drinking activities; effectiveness is dependent on that one friend's power to discern riskiness and effectively deter another from making an unsafe decision.

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

Cognitive processes link to our feelings/emotions/affect, as well. For example, how we label our feelings has an influence on how emotions are experienced and how we behave in response to emotions. For example, if you have only a few labels available for

describing and understanding ***affect*** (e.g., mad, sad, glad) then you have relatively few options available for how you behave in response; having more affective labels cognitively available for the emotions related to an event or experience offers a wider array of behavioral responses. Consider, for instance, what might happen in two different scenarios where someone gets a poor grade on an exam—it feels "bad" but what kind of "bad" or negative affect we identify determines how we might respond to the event. Some of the solutions or options are more productive than others:

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

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

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

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

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

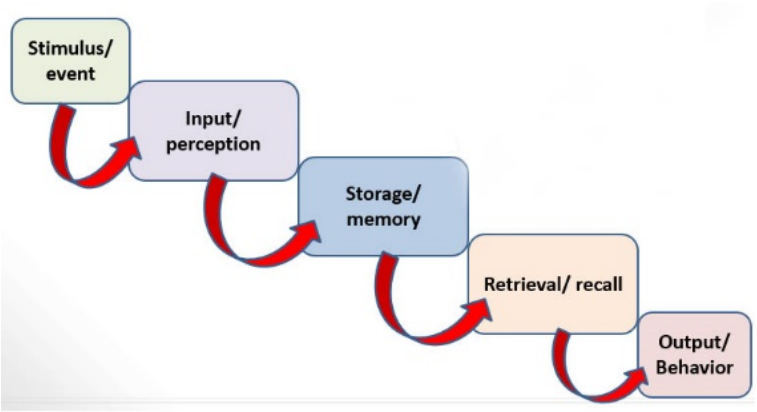
Rotgers (2012) identified a set of common basic assumptions among cognitive behavioral (CB) models and interventions related to substance use disorders, most of which could be applied to other forms of addictive behavior:

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

Information Processing

The ***information-processing*** model comes from cognitive psychology and helps explain (1) what a person "knows" about a substance, and (2) how a person's substance use might affect behavior through its influence on perception, short- and long-term memory, and information retrieval. Not only does this model have implications for information/education intervention and how individuals behave while under the acute influence of certain substances, it also has implications concerning long-term (chronic) substance misuse and recovery from SUD. Information processing

concerns how we initially take in information about our environment (or from internal biological cues). Then, what happens with that information and does it influence behavior? Let's look at the information processing steps.



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

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

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

Retrieval. Depending on how memories were stored (long-term), they need to be recalled or retrieved in order to influence behavior. Cues from other stimuli or memories can "trigger" recall of a stored memory–for example, smelling marijuana might "trigger" recall of how it felt to use it or driving the car might "trigger" memory of how it felt to smoke a cigarette while driving. This is an important aspect of cravings. On the other hand, evidence concerning ***state-dependent learning*** suggests that retrieving information is easiest and most accurate when conditions are very similar to when the information was originally introduced/learned. In other words, information or skills learned and easily retrieved while under the influence of alcohol or other substances may be more difficult to retrieve when a person is in a different (unaltered) state of consciousness (Overton, 1984). Vice versa, what is learned under normal conditions may not be recalled when in an altered state. Thus, a person in recovery may need to relearn information or skills originally learned while under the influence of substances.

Substance-distorted information processes. In addition to examples of how each step might be affected by substance use, psychoactive substances can profoundly affect overall information processing. For example, information processing overall is slowed among men engaged in chronic excessive alcohol consumption compared to men who do not drink alcohol excessively, beginning with perception and carrying through the decision-making and response (behavior) phases (Kaur et al., 2016). This, in part, explains delays in reaction time and the risk of driving a vehicle under these conditions. Fortunately, affected cognitive functions improve in many individuals during months to years of abstinent recovery

(Cabé et al., 2015). In addition, consider the possibility that individuals in early recovery may not effectively process information delivered through treatment/intervention efforts with a heavy cognitive component—these strategies are better processed a few weeks into recovery (NIAAA, 2001).

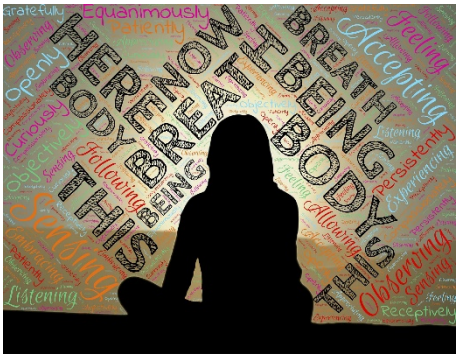
Learning Theory

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

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

(unconditioned response), eventually makes the dog salivate in response to the bell alone—the bell has become a conditioned stimulus and salivation to the bell (rather than food) has become a conditioned response.

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



Operant Conditioning. Another set of psychological learning principles with a profound impact on substance misuse is ***operant conditioning***. Operant conditioning is all about rewards and punishments (recall what you learned about the reward circuits in the brain in Module 3—for example that dopamine release is a powerful reward). If someone experiences a positive consequence as the result of using a particular substance, the reward (***positive reinforcement***) increases the probability of repeating that behavior again in the future. Experiencing a negative consequence

(***punishment***) decreases the probability of repeating that behavior again in the future. Considerable confusion revolves around the concept of ***negative reinforcement***, and because this is an important process in substance misuse negative reinforcement warrants some closer attention. Let's start with this chart comparing consequences and effects in operant conditioning.

	Consequence	Effect
BEHAVIOR	provide favorable stimulus (reward)	increased probability of repeating behavior
	remove unfavorable stimulus (reward)	increased probability of repeating behavior
	provide unfavorable stimulus *or* remove favorable stimulus (punish)	decreased probability of repeating behavior

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

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

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

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

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

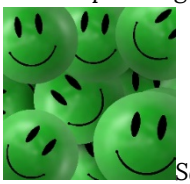
- A person is offered cigarettes by peers and feels accepted by them (positive reinforcement) when joining them in smoking together. Result: more likely to smoke with friends in the future.
- A person drinks to the point of throwing up (punishment—applying a negative consequence). Result: less likely to drink to excess in the future.

- A person has to pay heavy fines and pay lawyers/legal fees for driving under the influence of marijuana (punishment—taking away a positive). Result: may be less likely to drive under the influence in the future.
- A person feels nauseous with anxiety and finds that the anxiety and nausea go away when using cannabis (negative reinforcement). Result: may be more likely to use cannabis to dispel anxiety/nausea in the future.

This last example plays a role in what we learned about withdrawal symptoms and tendency to relapse (or at least slip) during recovery from SUD. A person whose body has come to depend on a substance like alcohol or heroin being regularly administered will experience withdrawal symptoms if the substance is no longer used. Withdrawal symptoms are a very aversive (negative) experience which makes it a quite punishing consequence for quitting use—the person is less likely to maintain the "quit" behavior as a result. Then comes part two of the problem: negative reinforcement. If the person does resume use, even one slip, the punishing withdrawal symptoms momentarily subside—this consequence rewards using again. So, in operant conditioning terms we have two forces pushing for relapse as a result of withdrawal symptoms—the punishment for quitting that the withdrawal symptoms introduce, compounded by the negative reinforcement for using again. You can see why operant conditioning is so important both in the process of substance use becoming substance misuse or SUD and in the difficulty of recovery, as well.

A little more about reinforcement paradigms. While operant conditioning can make the story of substance misuse clearer, there do remains some complicating factors. These have to

do with (1) consequence salience, (2) consequence timing, and (3) consequence sequencing.



Salience. A single reward or punishment may not mean the same thing to everyone—it may have different ***salience*** for different individuals. For example, M&Ms may be perfect rewards for some toddlers in potty training while other toddlers really do not care about candy; they are better rewarded with smiley faces drawn in marker on their hands and knees. In training a new behavior, it is critically important to find the reinforcements that are most powerful for each individual. As noted in Module 3, heightened reward sensitivity in the adolescent brain might make the reinforcing aspect of drinking, vaping, or using cannabis more rewarding than for older individuals. Likewise, punishments may have different power (salience) for different individuals—charging fines may be more punishing to some than to others, for example. Or, for instance, nicotine withdrawal may be experienced more negatively by some individuals than by others, which has an impact on differences in their ability to cut down or quit smoking.

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

On the other side of the timing issue, you may wonder why experiencing a hangover does not always lead to someone learning not to drink, or at least not drinking to excess. Unfortunately, the

consequence (hangover) is delayed by many hours from the behavior (drinking). This time lag erodes (ruins) the power of the punishing consequence to be a strong influence on future behavior—"time is the enemy."

(C) Audrey Begun 2015

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

Negative attention. One last point about learning theory warrants consideration. The social world around us is a rich source of positive reinforcement, negative reinforcement, and punishment. We would expect that exhibiting a behavior for which the consequence is social approval would likely be repeated—it was positively reinforced. We would expect that a behavior met with scolding would less likely be repeated—it was punished. However, we sometimes see an odd paradox with this latter example. Sometimes, any attention, positive or negative, is rewarding. Instead of a scolding being punishing, it could be reinforcing in some instances.

Furthermore, sometimes when a behavior is ignored, the individual interprets the lack of punishing response to be a tacit approval of the behavior—which, in turn, means it is more likely to be repeated. Sometimes ignoring a behavior leads to its extinction. Other times ignoring a behavior leads to its encouragement.

Social Learning Theory

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

Many complex behaviors are learned through modeling and imitation—aspects of observational learning—rather than learning each individual element of the complex behavior one-at-a-time. For example, smoking a cigarette or e-cigarette ("vaping") is a complex behavior—it involves engaging in a series of coordinated behavioral steps. Learning to do this is not "taught" one step at a time as in an

instructional manual for assembling a toy or piece of furniture. It is learned as a behavioral sequence, typically through observation of behavioral models.

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

Imitation of modeled behavior is a power mechanism of learning and socialization throughout the lifespan. ***Social referencing*** concerns a person who, in ambiguous or unfamiliar situations, relies on observing others' behavior to know how to respond. We see social referencing in young children when they, together with a

parent, are approached by a stranger: the child turns to watch and listen to the parent's reaction to tell them how to interpret and respond in the situation. Social referencing may play a role in how individuals respond to substance-related situations—watching peers, for example, respond to someone offering alcohol or other substances in order to know how they might respond themselves. Social referencing involves using the other person's behavior as a cue in interpreting a novel or ambiguous situation for oneself.

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

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

Theory of Reasoned Behavior

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

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

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

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

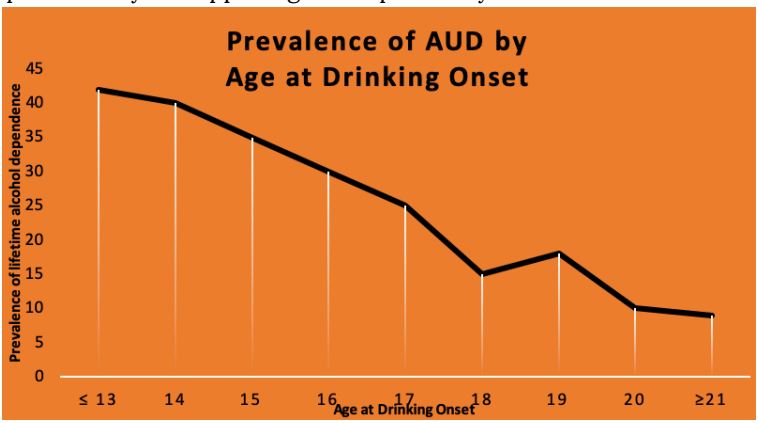
Ch. 2: Developmental Theories

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

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

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

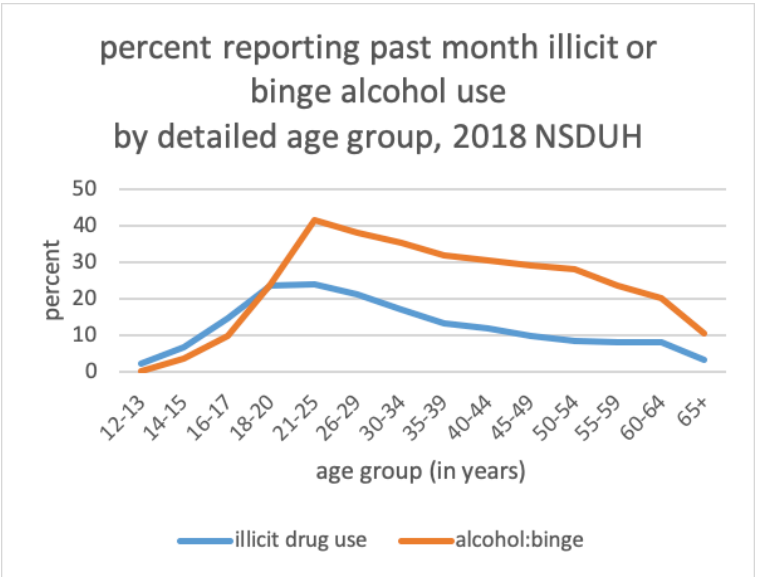
Developmental trends data. The following graph displays data from the 2001-2002 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). The data demonstrate a trend in which the younger a person is when beginning to drinking alcohol, the greater the likelihood of developing an alcohol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



You may also recall that some of the impact is due to changes in the developing brain that occur with exposure to alcohol during the adolescent and emerging adulthood years—this is a period of very rapid brain reorganization under normal developmental conditions

so exposure to alcohol during this time may affect the brain more dramatically than alcohol exposure later in brain development. You also may recall that the adolescent brain is more sensitive to the rewarding/reinforcing experience of alcohol exposure than would be true if first exposure occurred later in life.

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



Because these data are cross-sectional rather than longitudinal, we do not know if the use patterns for each individual followed this type of pattern, only that this pattern reflects the use at one point in time for the different groups. While it suggests a developmental trend, it does not confirm that such exists. For example, it is

possible that the declining numbers may be at least partially attributable to attrition–individuals engaging in these behaviors over time may be less likely to survive to represent the later age groups.

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

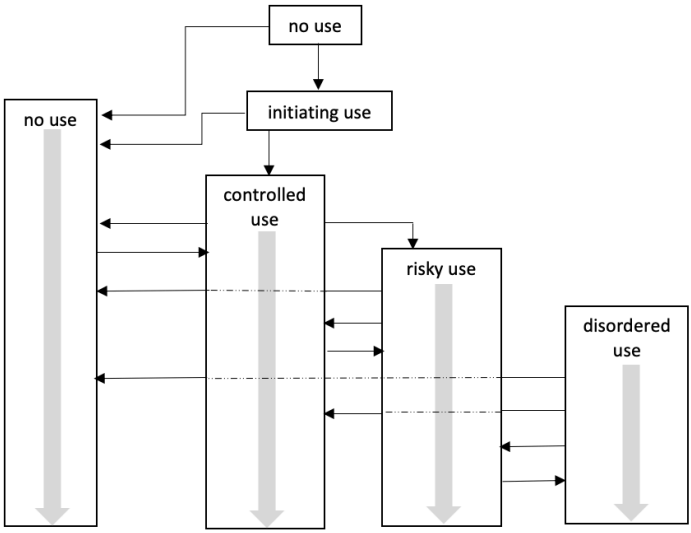
Developmental trajectories of substance use disorder. During the 1950s and 1960s E. Morton Jellinek concluded that alcoholism follows a natural course over time, a course characterized by four qualitatively distinct stages: pre-alcoholic, early alcoholic, middle alcoholic, and late alcoholic (Jellinek, 1952). Despite many years of influence, Jellinek's developmental model

has been criticized for being based on a small, select sample (of men in Alcoholic Anonymous programs), and because progressive worsening of symptoms is not universal (see (see Begun, *in press*): a great deal of clinical heterogeneity exists (Moss, Chen, & Yi, 2007). More recent studies demonstrated the dynamic, constantly changing nature of addictive behaviors: "Addiction can be viewed as a trajectory that emerges, becomes ingrained, and then in most cases evolves further (people quit or learn to control their use) over time" (Heather et al., 2018, p. 251). Yakhnich and Michael (2016) described the trajectory as a process beginning with occasional use of substances and ending with addiction, recognizing that many individuals "mature out" of excessive use at points along the trajectory.

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

A 60-year longitudinal study of college-aged men whose drinking patterns were identified as "alcoholism" demonstrated widely varied patterns in later adulthood, including stable abstinence, non-problematic/controlled drinking, alcohol abuse, or death (Vaillant,

2003). A typical substance misuse trajectory begins during adolescence or emerging adulthood, declines or escalates during emerging and early adulthood—where it may or may not meet criteria for a substance use disorder—then either declines or extends into adulthood, possibly but not necessarily meeting criteria as a substance use disorder (see figure below, from Begun, *in press*).



Important aspects of this figure are the multiple pathways/ trajectories that occur and the iterative nature of the possible trajectories: for example, moving back and forth between controlled, risky, disordered drinking, and no alcohol use. The probability of different trajectories is affected by a host of individual-specific factors, as well as the "addictive potential" of different substances involved (Upah, Jacob, & Price, 2015) and individuals' different histories of change attempts over the life course (Begun, Berger, & Salm-Ward, 2011). Similarly, no single, "natural" trajectory to/through recovery exists and there are a multitude of addiction "careers" in individuals' relationships or

involvement with substances over their lifetimes following the emergence of a substance use disorder (DiClemente, 2006).

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

STOP & THINK

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

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

How would you explain to a group of 12 year olds why it matters

not to initiate alcohol, tobacco, or other substance use at least until you are an adult?

Ch. 3: Theories of the Psyche

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

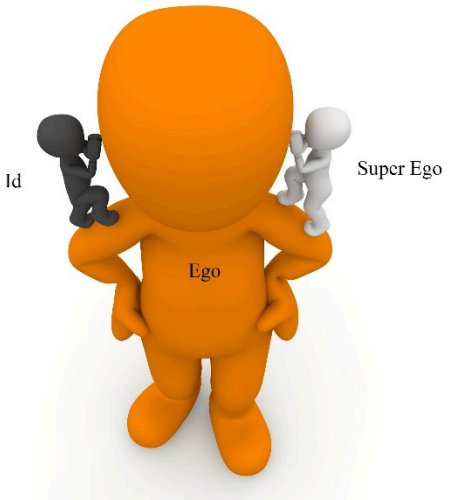
Psychodynamic Theory

In a ***psychodynamic theory*** interpretation, addiction is not viewed as being a disease in and of itself but as a *symptom* of intra-psychic conflict, unresolved psychological tension, or psychological turmoil. On one hand, a person may experience urges to express emotions by behaving in ways that might not be socially acceptable. The urge to handle frustration or anger through aggression and violence are examples of this side of the equation, born in the primal aspects of personality (called the Id). The Id is not just negative, it includes positive feelings, too—think of a really young puppy as a ball of Id—it acts as

it feels, positively or negatively, totally in the moment, with no filter, no restraint.

On the other hand, over time and through repeated learning encounters with the physical and social world, a person (and hopefully puppies) develop enough experience to understand and appreciate that acting aggressively or violently is not socially acceptable and that this behavior is a poor choice. In other words, the super-ego has stepped in to editorialize about the Id response to emotions. This is where sentiments like guilt and shame come into play, helping reign in socially unacceptable behavior choices.

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



In this psychoanalytic or psychodynamic model, a person may resolve some of this Id-Superego tension by using alcohol or other drugs for their ability either to "numb" feelings that are triggering

the Id response or to silence the super-ego, put it to sleep, thereby removing the unpleasant, tension-filled experience of conflict. Sometimes individuals in conflict feel the need to quiet the "voices" that are always "yelling" in their minds. Additionally, psychoanalytic or psychodynamic theory might suggest that an individual who has experienced trauma might use substances as a means of "numbing" the powerful negative feelings experienced as a result of reminders of the past trauma experience. This is not the only way the theory has been applied to substance use, however.

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

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

the developmental period when the libidinal energy localizes in the genital zone.

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

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

Attachment Theory

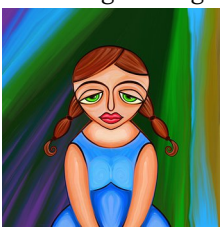
An ***attachment theory*** of addiction is not far removed from psychoanalytic and psychodynamic theory explanations. As explained in the early works of John Bowlby, infants and young children, in the normative course of development, form attachment relationships with others central to their physical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

attachment relationships, individuals begin to make sense of their social world.

Sometimes, attachment relationships are disrupted or dysfunctional. They either fail to form, are broken once formed, or develop as insecure and unstable attachments. According to attachment theory, a person experiencing attachment issues is likely to experience significant "holes" in their emotional and personality development. The world does not seem like a safe, predictable, reliable place to exist, nor are there safe, predictable people on whom the person can rely. Their understanding of and relationship to the world is likely to have significant gaps.

Sometimes these individuals describe themselves as being "full of emptiness."

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



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

healthy super ego and ego strengths. Furthermore, throughout the life cycle, prevention involves avoiding the disruption of attachment relationships and exposure to traumatizing experiences.

Self-Medication Theory

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

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

"My whole life went downhill. I was abused, and

> ***used alcohol to escape the pain. I became horrible to myself and everyone around me. I honestly didn't care what happened anymore"*** (quoted in Najavits, 2009, p. 290).

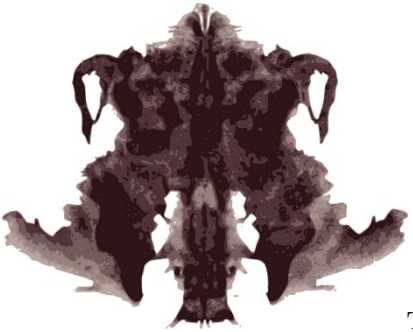
Self-medication theory is somewhat controversial. The prior examples do not demonstrate a causal relationship whereby self-medication theory is proven; the theory remains a possible explanation for at least some of the co-occurrence. Sometimes trauma events precede substance misuse. Other times, traumatic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

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

true could it be when individuals in the general population are self-medicating?

Personality and Psychopathology Theory

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



These days, the idea of an addictive personality is considered somewhat dated as it is not well supported by evidence. While there exist some traits or characteristics commonly observed among groups of individuals who experience substance use disorders, the evidence does not support there being a universal set of personality traits or personality type associated with addiction/substance use disorders. Evidence for the existence of an "addictive personality" type does not exist (per Szalavitz, 2016 citing an interview with George Koob, director of the National Institute on Alcohol Abuse and Alcoholism).

What we know is that pretty much any person can become addicted to something if the right (or, in this case the wrong) circumstances come together. Some individuals may be more vulnerable or at a higher risk of addiction to certain substances, but the potential exists for anyone depending on circumstances. We also know that the circumstances vary somewhat for different types of substances—the vulnerability and risk for developing alcohol use disorder is not the same as for developing addiction to nicotine or cocaine or opioids or cannabis.

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

There exists some evidence to suggest that certain temperament or personality characteristics are associated (correlated) with a higher probability of *initiating* substance use, especially early initiation of alcohol or tobacco use during adolescence. For example, studies emphasize the increased odds of using/misusing substances among adolescents who have angry-defiant personality types, as well as the "thrill seeker" personality type (sometimes

called the "Type T personality"). Or, evidence indicates that "young people diagnosed with conduct disorders and other oppositional disorders are also at higher risk for developing substance use disorders in adolescence and early adulthood," as is also true of individuals with bipolar and major depressive mood disorders (Cavaiola, 2009, p. 721).

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

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

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https://ohiostate.pressbooks.pub/substancemisusepart1/?p=160

Ch. 4: Expectancies & Cravings

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

Expectancies

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

Children develop expectancies about alcohol at a very early age–even preschool/kindergarten aged children may already have developed ideas about the emotional effects of adults' drinking (Kuntsche & Kuntsche, 2018). One source of their expectancies was parental drinking: sons identified positive emotional consequences

(e.g., feeling happy, calm, relaxed) if a parent engaged in moderate drinking and they identified negative emotional consequences (e.g., feeling angry, sad, depressed) if a parent engaged in heavy drinking; the effects were less consistent among daughters and were stronger when the parent was the father rather than the mother (Kuntsche & Kuntsche, 2018).

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

Behavior	8th grade	10th gr
try marijuana once or twice	20.3	13.9
smoke marijuana occasionally	32.1	21.4
smoke marijuana regularly	52.9	38.1
try inhalants once or twice	29.6	38.6
take inhalants regularly	46.8	57.6
take LSD once or twice	20.8	33.8
take LSD regularly	36.4	54.1
try cocaine powder once or twice	42.6	52.6
take cocaine powder occasionally	61.0	70.2
try heroin once or twice *(without using a needle)*	59.5	71.4
take heroin occasionally *(without using a needle)*	72.1	81.0
try one or two drinks of an alcohol beverage (beer, wine, liquor)	13.6	13.0
take one or two drinks nearly every day	28.7	30.3
have five of more drinks once or twice each weekend	52.3	51.8
smoke one to five cigarettes per day	40.8	49.9
smoke one or more packs of cigarettes per day	61.3	69.6
vape an e-liquid with nicotine occasionally	16.9	17.9

vape an e-liquid with nicotine regularly	32.4	31.3

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

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

From what individuals see in their homes, neighborhoods, schools and jobs, media, and social media they develop expectancies about alcohol, drugs, sex, smoking, gambling, and many other types of

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

Cravings

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

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

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

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

Ch. 5: Summary

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

Module 4: Key Terms

affect concerns a person's emotions and feelings.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

self-medication theory reflects a belief that individuals may use

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

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

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

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

Module 4: References and Image Credits

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PART V

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

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

Reading Objectives

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

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

Ch. 1: Social Contexts and Physical Environments

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

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

Stigma

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

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

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

Policy as a Context Influence

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

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

Physical Environments

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

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

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

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

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

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

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

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

Gene-Environment Interplay

Social and physical environment elements have a great deal of power to potentially modify genetic and psychological influences on health-related outcomes, including substance use initiation, substance misuse, and the development of substance use disorders (Begun, Bares & Chartier, *in press*). For instance, social and physical environment factors may compound vulnerabilities or impart resilience by either

imposing constraints or offering opportunities that enable, trigger, disrupt, or strengthen biological or psychological effects (Bares & Chartier, *in press*). Evidence supports the notion that genetic predisposition to alcohol use/misuse/use disorder and environmental exposures interact to influence alcohol use patterns (Sher et al., 2010). Similarly, this type of interaction was observed in tobacco use patterns (Chen et al., 2009). The concept of a *gene-by-environment interaction* indicates that a person's genetic makeup can determine sensitivity to environmental effects and whether environmental exposure enhances or diminishes genetic effects (Bares & Chartier, *in press*). A body of research concerning alcohol, cigarette, and other substance use initiation, as well as for regular substance use, generally suggests that the influence of environment is stronger during early adolescence and gradually shifts to genetic factors (heritability) playing a more predominant role in adult development (Bares & Chartier, *in press*). For example, parental monitoring can reduce the influence of genetic heritability in cigarette use (Dick et al., 2007). Additionally, genetic effects on alcohol use are more evident among adolescents receiving low levels of parental monitoring, as well as adolescent affiliating with peers who engage in high levels of deviant behavior (Kendler, Gardner, & Dick, 2011). The interaction between intrinsic (biological and psychological makeup) and extrinsic environmental forces related to substance misuse is further explored in Module 6, reintegrating biological, psychological, and social context models into a more unified biopsychosocial framework.

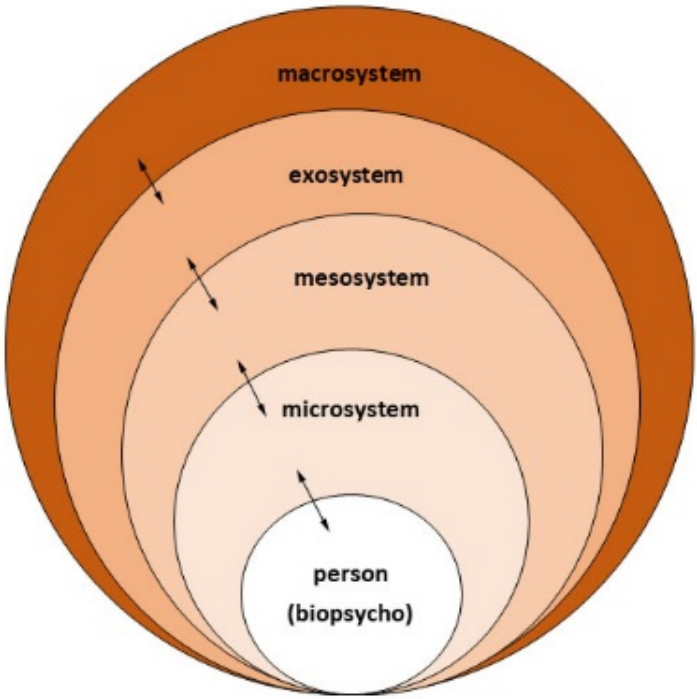
Social Systems

Anthropologists argue that the use of substances can only be properly understood when placed within *a social context: the f*amily, social, school, work, economic, political and religious systems (Hunt & Barker, 2001). The ***social ecological model*** (Bronfenbrenner, 1986,

1996) related to human development occurring within social systems at varying levels helps direct attention to social contexts as related to substance misuse—as well as informing interventions for substance misuse prevention and recovery support.

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

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



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

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

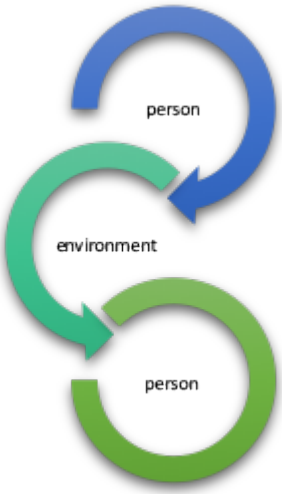
Moving one sphere further out, the microsystem influences and is influenced by the ***mesosystem***. The mesosystem components include elements in the relatively immediate environment with which an individual routinely interacts, but less frequently and intimately than was true of the microsystem. For some individuals this includes extended family members and peers/friends with whom the relationships are influential but not as close and intimate. It might include the companions in the workplace or at school, and it might include neighbors. For some individuals this might include members of a religious or spiritual community. The mesosystem of recovery might include companions in the peer support community, other members of mutual aid/peer support/12-step type programs. It is also possible that members of the formal health/mental health/addiction treatment system fit into the mesosystem context.

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

Finally, we have the ***macrosystem*** to consider. While few of individuals directly interact on a routine basis with the elements shaping the cultures and societies in which they live, these elements exert powerful (though indirect) influences on experience. Consider, for example, how changes in the legal status of certain

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

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



Circularity of Influence.

As noted in the previous discussion, but warranting an emphasis and attention is that individuals being influenced by the social and physical environments is one part of the equation: it is also true that they have an influence on their social and physical environments, as well. Anyone who has cared about a friend or family member experiencing substance use disorder will tell you that the individual's substance use, related behaviors, and consequential problems not only affect that individual but also has an impact on those in the social and physical contexts, as well. The individual's behaviors affect many different types and levels of social and physical environments; the very environments that influence that individual, too. This iterative pattern of influence continues over time—the environment influences the person who influences the environment, and the changed environment continues to influence the changed person, and so on over time. This is what is meant by the concept of ***circularity of influence***. This perspective acknowledges that individuals are actively engaged with their environments, not simply the passive recipients of environmental influences; furthermore, individuals make choices and decisions from among options available in their social and physical contexts, choices that have consequences for themselves and others in their social/physical contexts, as well (Begun, Bares, & Chartier, *in press*; Shelton, 2019).

Social Norms

A culture's or group's collective expectations about acceptable behavior are represented in its ***social norms***. Social norms are key social processes related to many types of behavior, including substance use and misuse. Groups may have specific norms about initiating substance use, acceptable patterns for regular use, excessive use or intoxication, seeking treatment for substance-related problems or substance use disorder, and recovery support.

For example, most cultures accepting of alcohol use have norms related to the boundaries of its acceptable use–when, where, by whom, and how much. Social norms influence individuals' behavior choices. For example, a person who believes that "everyone else" either uses or approves of using cannabis is far more likely to engage in its use than a person who believes that it is not common or accepted in their social context. Or, for example, social norms against driving under the influence of alcohol (or other substances) influence the behavior of individuals electing to use sober driver strategies when planning to participate in drinking events. On the other hand, if public education efforts deliver messages that "too many young people" use alcohol, tobacco, or vaping products, the actual message received by that population may be that engaging in this behavior is normative and accepted within their group. In other words, the message could backfire as a preventive strategy because it actually conveys a positive social norm about the behavior. Social norms surrounding substance use are significantly related to substance use behavior, especially among adolescents (Eisenberg et al., 2014). Media campaigns have proven effective in shaping norms and health-related behaviors related to intoxicated driving, use of tobacco products, and parents discussing substance misuse with their children (Wakefield, Loken, & Hornik, 2010). See, for example, the "Don't Live in Denial Ohio" media campaign (https://dontliveindenial.org/).

To understand young cohorts and their norms related to

substance use, consider *Monitoring the Future* 2018 study results (Table 2). The survey asked students to rate their own level of disapproval toward people who use various substances. What is interesting in these data is that the trend is substance-dependent. Between 8th, 10th, and 12th grade each group of students was more accepting of alcohol and marijuana use than the next younger group. The opposite was true of heroin, cocaine, LSD, inhalants, and regular vaping of e-liquids containing nicotine. It is not clear whether these cross-sectional data reflect a true developmental change in youths' opinions. However, it does suggest that as the students progressed in age/grade, they make clearer distinctions between types of substance use.

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

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

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

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

Alcohol plays an integral role in many religious, ethnic, and cultural ceremonies, but when it is included its use is typically characterized by moderation—drinking alcohol in moderation is permissible but drinking to intoxication is not in alcohol-involved rituals such as Shabbat, Passover, and the marriage ceremony in Judaism; substitutions for alcohol (grape juice, watered-down wine) are often accepted especially for pregnant women, young children, and persons in recovery from alcohol or other substance use disorder. Social norms disapproving of excessive alcohol use (misuse) can be a protective factor against alcohol use becoming an alcohol use disorder. In an analogous fashion, social norms concerning use of tobacco products, e-cigarettes/vaping, cannabis, and other substances may also have an impact on individuals' decisions about initiating substance use, using substances to excess, or using substances under risky circumstances (e.g., driving or operating dangerous equipment, use during pregnancy, use by adolescents, use in combination with other substances). Shaping and communicating social norms is one target of preventive media campaigns.

Another perspective to keep in mind when thinking about social

norms is an observation about ***homophily***. The homophily principle means that, when free to choose, humans tend to congregate and associate with friends, acquaintances, and partners similar to ourselves. The saying is, "birds of a feather, tend to flock together." The implication substance use implication is that individuals may choose to spend time in the company of others who engage in similar patterns of substance use/misuse. The homophily tendency shapes and reinforces the individual's social norms about substance use, misuse, treatment seeking, and recovery—leading the individual to believe that "everyone" holds those similar norms because most everyone in their immediate social context does.

Social Structures

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

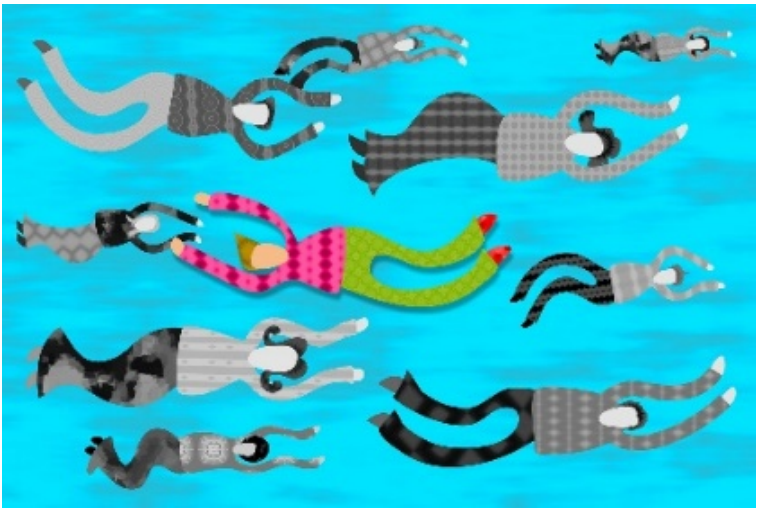
Culture and subculture. Cultural systems are significant sources of socialization shaping attitudes, beliefs, and behaviors concerning substance use, misuse, treatment seeking, recovery, and stigma. The content of the values and belief systems of different cultural groups might vary, but many of the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

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

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

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

past decades such that a deviance position is now becoming normative.



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

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

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

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

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

Exposure to repeated instances of ***microaggression*** may contribute to substance use, as well. Ethnic and racial microaggressions are events that leave the person on the receiving end feeling put down or insulted based on their race or ethnicity—regardless of the intent by persons delivering the messages (Blume, Lovato, Thyken, & Denny, 2011). In a study of undergraduate college students, microaggression experiences were associated with both higher rates of binge drinking and experiencing more of the negative consequences associated with drinking (Blume, Lovato, Thyken, & Denny, 2011). Similarly, a study of college students demonstrated that the odds of regular marijuana

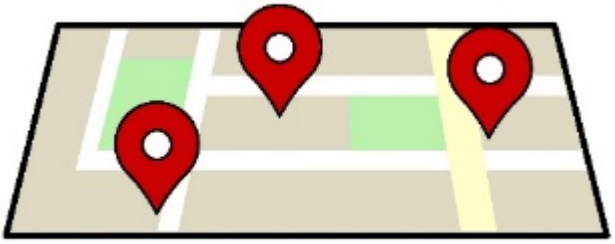
use increased as a function of the number of microaggressions experienced (Pro, Sahker, & Marzell, 2017). And, again, the same relationship was observed in a study of Native American students and use of illicit drugs (Greenfield, 2015). Thus, it is important for social workers and other professionals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

"Isms" play a role in creating and maintaining marked disparities in opportunity and resources between social groups at the level of neighborhoods, schools, communities, workplaces, and populations. These include discrepancies in media portrayal, access or barriers to drugs, disparate exposure to advertising and media portrayals of drugs, access to desirable alternatives to drug use, availability and cultural competence of prevention and treatment options, and the consistency with which sanctions for drug-related activities are imposed (e.g., variable implementation of zero-tolerance policies or criminal justice system sanctions). Recall from Module 1 how the War on Drugs related to tremendous racial and ethnic disparities in the nation's incarceration rates, for example.

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

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

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



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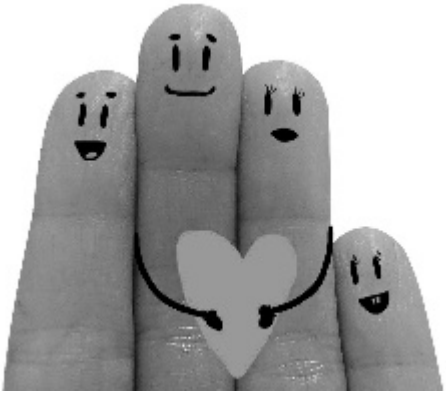
https://ohiostate.pressbooks.pub/substancemisusepart1/?p=184

Ch. 2: Family as Social Context

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

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

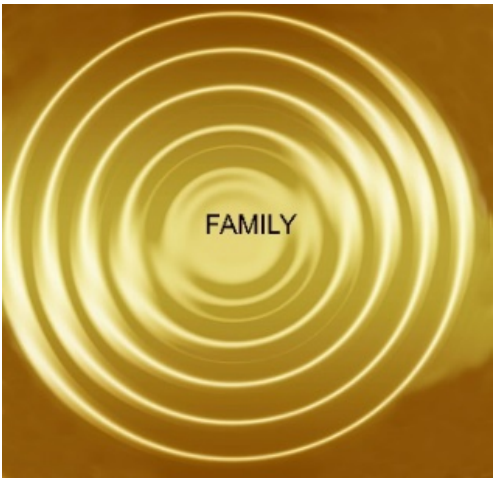


Family Systems Theory

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

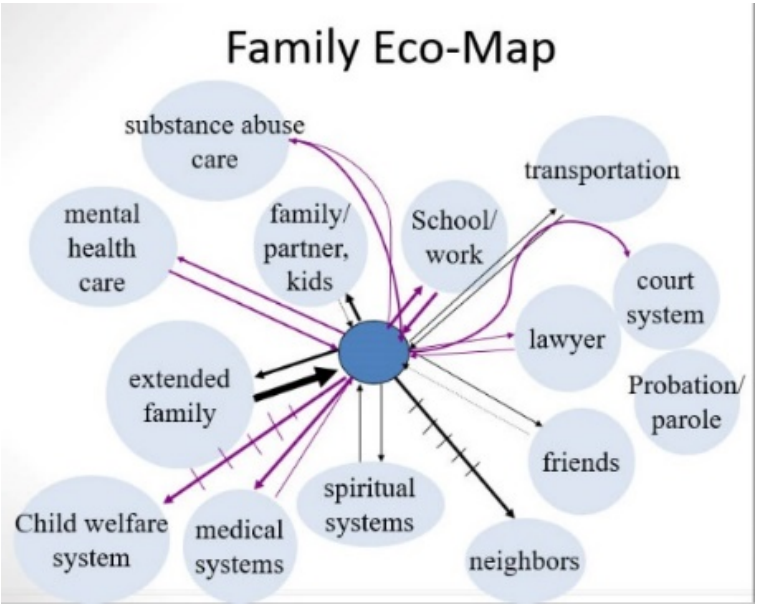
1. **The family is a system embedded in other, larger social**

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



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

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



An ***eco-map*** is a visual assessment tool (originally described by Dr. Ann Hartmann) used in social work to help families identify and express the nature and quality of their interactions with the surrounding systems—what is supportive and what is detrimental, where the energy, emotional, and resource "costs" to the family are excessive compared to what is gained, and where the "gains" are more favorable than the costs. In some cases, a family eco-map may appear overly sparse, leaving the family under-resourced and socially isolated. At the other extreme, a family's eco-map may be overly saturated with formal institutions that serve the family but at a high "cost" in effort and energy; even informal relationships are supported at some cost since they typically exist within a set of "give and take" expectations. Even if the amount and types of ecological relationships may be reasonably balanced, the qualitative picture may be heavily conflict-ridden as opposed to working peacefully or harmoniously. For example, when a family member engages in substance misuse involvement with criminal justice, child welfare, housing, and health care systems may occur, much of which is demanding despite possibly providing needed resources. All of this

relates to the degree of stress which the family system is under in its day-to-day existence. Stress is often translated into individuals' use of substances, according to ***stress and coping theory*** which emphasizes the stress experienced in contexts characterized by a great deal of social disorganization, distress, and alienation (Moos, 2006).

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

3.  **Family systems are comprised of subsystems.** Family systems do not always operate as a whole; many interactions, roles, and functions are enacted within subsystems of the larger family system. These might include a couple subsystem, parent-child subsystems, or sibling subsystems. The possible variants are numerous especially when ex-partner, step-parent, step-/half-sibling, and extended family subsystems are involved. In families where a member is engaged in substance misuse, it is possible that the person's "relationship" with the substances themselves functions much like a subsystem. For example, an adult son living with his mother—both of whom experienced active substance use disorders—described their two-person family in terms of there being "three of us" in the relationship—himself, her, and the drugs (quotingTony from the documentary entitled *Foo Foo Dust*).
4. **Change in any part of the system affects the entire system.** Family systems are dynamic, changing over time as recognized in the chromosphere aspect of the social ecological model—past, present, and future look different because families are not static or stagnant. It is important to remember that all change—positive and negative in nature—are experienced as stressful, challenging the family system's hard-earned balance and homeostasis. Consider, for example, how stressful happy family development events like marriage, childbirth, and retirement can be for the system; this adaptive pressure might bewilder families who only expected to be stressed by negative changes such as divorce or the death of a family member. Family system changes are a response to pressures from the outside (contexts), family membership,

internal subsystems, and changes in individuals. Some pressures are developmental in nature—the dynamics of parenting young children may be very different from parenting adolescents, for example. It is the nature of systems that change in any part of the family system reverberates throughout the entire family system, sending ripples throughout the system.

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

progressed to the point of a substance use disorder (Begun, Bares, & Chartier, *in press*).

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

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

Recovery as Change Experienced by the Family System

Outside of developmental changes, other individual changes can affect the family system as a whole: for example, a family member moving from substance misuse into recovery. Over time, a family may have adapted to the individual's unpredictable, unreliable behavior while pursuing, using, and getting over the effects of using substances. Families may adapt to a member's substance-related unpredictability and erratic functioning by excluding the individual from critical family roles (e.g., caregiving, financial decision making, intimacy). The family system exerts a great deal of energy and effort to achieve and preserve homeostasis under these rapidly shifting and unpredictable conditions. The family system again is challenged by the need to adapt to changes in the individual who engages in recovery efforts—how do family systems reintegrate these individuals and (again) provide them with meaningful roles? Despite recovery being a positive family event, all change in family systems is experienced as stressful—requiring the family to exert energy in (re)establishing balance. The system may not immediately respond in positive, accepting, welcoming, and trusting ways to the individual in early recovery, leaving that family member "on the outside looking in," especially if they have been challenged to do so

through multiple previous recovery efforts—they may be hesitant and not ready to place their trust in the recovery process. One member's recovery can be especially challenging to couples when they have been engaged in substance misuse together; their relationship may have been built around their substance use. What may look like "sabotage" of the recovery process by family members may more realistically represent the family's struggle to regain or retain homeostasis, even at the expense of or sacrificing one member's well-being. For this reason, as well as to support individual family members affected by another member's substance use, interventions at the couples and family system level are often recommended—helping the family to help itself as a whole and to support a member's recovery efforts (McCrady, 2006; McCrady, Epstein, & Sell, 2003).

Positive Parenting

Evidence indicates that strong positive parent-child bonds, family involvement, sanctions against inappropriate behavior, (age appropriate) parental monitoring of their children's behavior and experiences are protective, preventive factors in terms of substance use initiation and substance misuse (Moos, 2006). Parents establishing clear, unambiguous prohibitive norms concerning substance use/misuse, parental monitoring, and warm, positive relationships with their sons and daughters are protective factors against substance use/misuse. Reinforcing the importance of parents' behavior is evidence concerning the preventive potential

of parents' own restraint in substance use, child monitoring, and substance-related norm setting (Carpenter, Dobkin, & Warman, 2016; Cook & Tauchen, 1984; Hawkins, Catalano, & Miller, 1992). The presence of positive father-child relationships was shown to decrease the probability of adolescent alcohol use, particularly within African American families (Jordan & Lewis, 2005).

Compensatory parenting. A considerable amount of clinical literature discusses the negative developmental outcomes and risks to children growing up in a family where one or both parents engage in substance misuse, potentially leaving children "functionally parentless" (Straussner & Fewell, *in press*). This is the case for about 87 million children aged 17 or younger: about 1 in 10 living in households where a parent experienced alcohol use disorder in the past year and about 1 in 35 in households where a parent experienced a past-year substance use disorder involving illicit substances (Lipari & Van Horn, 2017). Parental substance misuse clearly has the potential to impede parents' ability to provide a safe and nurturing home for their children and raises the likelihood that children will be exposed to an array of environmental stressors (Straussner & Fewell, *in press*).

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

In short, it might be helpful to consider how the functions of parenting and caregiving are met when looking at children's experiences of the social and physical environment, rather than focusing only on the persons who are parents. Positive, stable compensatory relationships represent potentially significant contributions to a child's or adolescent's resilience (Begun, Bares, & Chartier, *in press*). Compensatory parenting is often provided by grandparents (see Mendoza, Fruhauf, & Hayslip, *in press*), other extended family members, other social contexts (e.g., school personnel or peers' family members; Werner & Johnson, 2004), or through formal foster care arrangements.

Family Disease Model

Earlier modules discussed debates concerning the disease model of addiction. Here we consider the ***family disease model*** of

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

However, the family disease model becomes controversial when relying on a definition of the family disease as ***codependency***. The codependency assumption is based on observations that certain traits and characteristics commonly occur within families experiencing a member's addiction. The defining traits involve family members' behaviors being organized around the one member's addiction-related behaviors. In codependency, family members' behaviors are viewed as supporting or enabling the dysfunctional behavior of the person experiencing addiction because they have come to depend on that person's dysfunctional behavior being maintained. The logical extension that has been inferred involves allowing the person's life to completely fall apart ("hitting rock bottom" or, at least "high bottom") and withholding love (or delivering "tough love") as means of motivating the person to change (Szalavitz, 2016). The evidence supporting this contention is weak and inconsistent, at best; evidence suggests that it actually inflicts additional psychological harm and many family members refused to engage in this manner without being part of a dysfunctional family system (Szalavitz, 2016).

The codependency model has become highly controversial, with many practitioners and researchers arguing against applying the label or diagnosis of codependence. First, many behaviors identified as codependent can be viewed as reasonable adaptive responses rather than causes (or ***enabling***) of the family member's addictive

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

Supportive Significant Others

Family members and others in a person's social context may play a significant role in recovery (Begun, Bares, & Chartier, *in press*); recovery is a process heavily influenced by social processes and

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

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

- footage is heavily edited to appeal to a (distorted) reality-television-consuming audience;
- the populations depicted poorly reflect the diversity of

individuals engaged in substance misuse or experiencing substance use disorders;
- the treatment options, availability, and accessibility represented are a glaring misrepresentation of what treatments are available, affordable, preferred, and successful;
- data provided by the show concerning the success rate of the interventions in helping individuals enter and complete treatment are misleading and grossly misrepresent the intervention outcomes.

These authors cite literature indicating that fewer than 30% of families encouraged to engage in the confrontational intervention method actually follow through and host such an event, and that a relatively small percentage of individuals enter into additional treatment following such an intervention (Kosovski & Smith, 2011). They concluded that other family-based models used to engage individuals in treatment are more effective and have been evaluated with greater rigor than the Johnson Intervention model. In other words, this approach does not represent a positive, effective role for SOs to act as SSOs.

Construct an eco-map of your own social contexts.

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

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

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

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

Ch. 3: Peer Groups as Social Context

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

Peer Influences: Risk and Protection

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

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

 Clearly, peer approval and modeling of substance use/misuse is a powerful risk factor for adolescent substance use/misuse (Zimmerman & Farrell, 2017). Adolescents' initiation of substance use, as well as its escalation into problematic substance misuse, is strongly associated with friends' and peers' substance use (Vink, 2016). This is evident with tobacco, alcohol, and marijuana; it possibly holds true with other substances, as well (Begun, Bares, & Chartier, *in press*). However, peer influences encountered during adolescence may persist well into emerging adulthood, at least as far as active involvement with friends who use alcohol is concerned (Piehler, Véronneau, & Dishion, 2012). In keeping with the previously noted human tendency toward homophily, about half of all people identified as important in the social networks described by individuals entering into treatment for alcohol use disorder were characterized as "drinkers" (24.63%), as were 19% of the most important people in their social networks (Mohr et al., 2001). Conversely, 9 months later, following 3 months of treatment, the rate of "drinkers" had declined significantly (17.98% among important people and 14.81% among the most important) and the rate of "nondrinking friends" had increased significantly from 17.98% to 33.37% of all the important people and from 14.81% to 21.47% among the most important people. Furthermore, the social network

constellation of "drinking" and "nondrinking" friends was significantly related to treatment outcomes: the more important "nondrinking" friends became, the greater the individuals' proportion of abstinent days themselves (Mohr et al., 2001).

Peer Support

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

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

It is not entirely clear how peer-delivered recovery support

services best align with and/or integrates with services delivered by licensed treatment professionals.

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https://ohiostate.pressbooks.pub/substancemisusepart1/?p=199

Ch. 4: Summary

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

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

Module 5: Key Terms

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

opportunities or barriers that influence individual development and behavior.

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

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

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

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

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

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

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

Module 5: References and Image Credits

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PART VI

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

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

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

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

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

Ch. 1: Theory Integration and Prevention

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

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

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

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

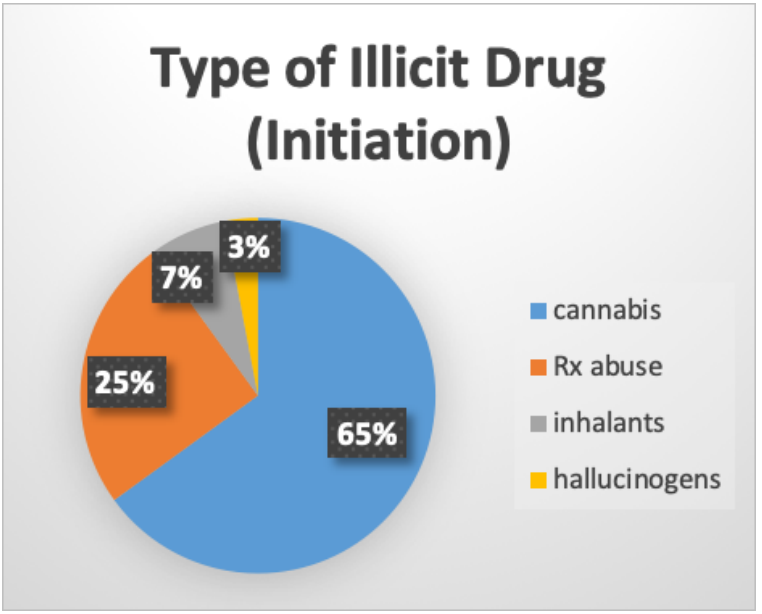
Specificity might include specifics of an addictive behavior (e.g., a specific substance, type of technology, or form of gambling) and/or a target population (e.g., a specified age or developmental phase, racial/ethnic group, self-defined gender identity, sexual orientation, co-occurring problems, or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

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

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

At this point, the task involves identifying the theory and evidence related to specific factors. For example, consider evidence related to the ***gateway drug theory***. If the identified problem is to prevent opioid/heroin use among adolescents and emerging adults, multiple theories and pieces of evidence will need to be considered for integration. One of these concerns the conflicting evidence surrounding cannabis use as a "gateway" to use of other, "harder" substances. Early evidence suggested a correlation between initiating heroin use and prior cannabis use—a very large portion of individuals using heroin had this in their past history (a vulnerability factor). However, subsequent and more sophisticated research approaches have called this gateway conclusion into doubt. First, the vast majority of individuals who have used cannabis never progressed to heroin use. Second, the distinction between "mild" and "hard" drugs is arbitrary and subjective. As we have discovered throughout the course so far, any psychoactive substance can be considered potentially addictive—some may have a higher percent of use or faster progression to addiction than others, but placing them on a single, comparative "seriousness" continuum is not grounded in evidence. A third blow to the gateway drug theory is

the tremendous diversity in substance use behavior observed across different geographical areas, ethnic and socio-economic groups, social networks, and cohorts over time. In the 2011 NSDUH survey, about 2/3 of participants who initiated illicit substance use during the study year reported marijuana as the first illicit substance used. However, this means that 1/3 started with something else: almost 25% started with prescription drug abuse instead, 7% started with inhalants, and just under 3% started with hallucinogens. This tremendous variability argues against a gateway drug theory—there are too many openings or access points involved for any one substance to be confidently identified as a gateway drug.

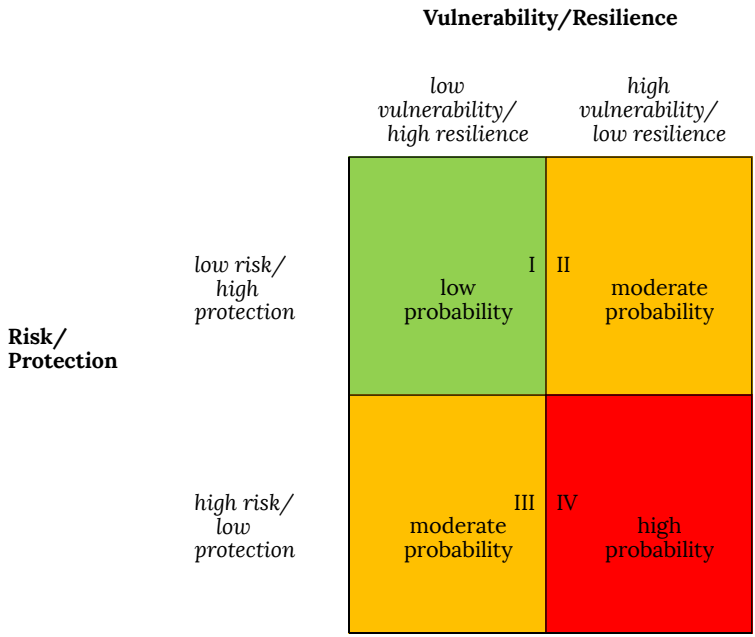


On the other hand, more recent events and evidence suggest that prescription abuse, particularly nonmedical use of opioid drugs, may represent a gateway to heroin use. Individuals entering treatment for an opioid use disorder (OUD) reported having "progressed" from prescription opioid use and nonmedical use of opioids (NAS, 2017). This sequence was also, by far, the most common pattern observed among surveyed individuals in the general population who reported heroin use each year in analysis

of 2003-2014 National Survey on Drug Use and Health data (NAS, 2017). Heroin, in many communities, is more easily accessed and less expensive since prescribing and dispensing restrictions have been introduced in response to the "opioid epidemic" facing many parts of the nation. "A number of studies have yielded evidence strongly supporting the conclusion that the recent prescription opioid epidemic has resulted in a significant increase in domestic heroin use and associated overdose death" (NAS, 2017, p. 207). The gateway theory concerning the relationship between prescription opioid and heroin misuse is bolstered by the fact that these substances have similar psychoactive and pharmacologic effects, including the capacity for ***cross-tolerance*** developing (NAS, 2017). Tolerance to a prescription opioid drug confers some degree of tolerance to heroin. Using higher doses of either/both increases the risk of overdose.

3. **Risk/protective factors continuum.** As with the vulnerability/resilience continuum, evidence concerning known risk and protective factors is identified and analyzed next. The ***risk/protective continuum*** refers to *extrinsic* factors. In other words, factors residing in current social and environmental contexts that we explored in Module 5 (social and physical environment contexts). The risk/protective factors continuum relates to "here and now" contexts and experiences; past interactions with the social context become a part of the vulnerability and resilience continuum—historical experiences of the environment become part of what is brought to new situations. For example, a history of ***adverse childhood events (ACES)*** becomes a vulnerability factor related to substance misuse; currently living in a traumatizing environment is a risk factor. Current risk/protective factors might include the presence (or absence) of alcohol, tobacco/vaping, or cannabis advertising in the neighborhood/community, ease of access to substances of concern, and social norms about substance use/misuse.

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



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

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

An interactive or media element has been excluded from this version of the text. You can view it online here:
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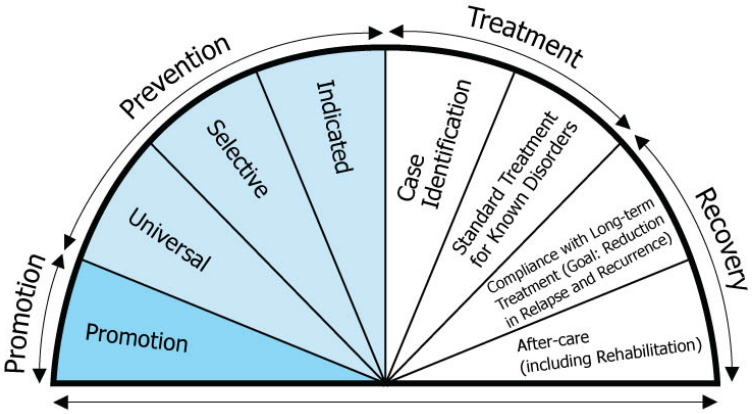
Ch. 2: Prevention and the Continuum of Care

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



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

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

  - ***Universal prevention*** refers to interventions delivered to the general population without differentiating between persons at different risk levels. For example, schools may deliver drug awareness and resistance education (DARE) programming to all students regardless of their vulnerability/risk constellation. Mass media campaigns are another example of universal efforts. In much of the prevention literature, the term "primary" prevention is used to describe efforts that occur before any sign of the target problem appear—universal prevention interventions are often applied.

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

- ***Indicated prevention*** is even more targeted, delivered to populations/groups of individuals exhibiting/expressing warning signs foreshadowing development of the focal problem. For example, to prevent alcohol use disorder interventions might be directed to youth/emerging adults engaged in binge drinking, preventing this behavior from becoming heavy drinking and a substance use disorder. As

the focus increases, preventive interventions may become increasingly resource-intensive and intrusive which makes the focus beneficial. A great deal of effort and resources would be wasted if these intensive interventions were delivered to a large portion of the general population unlikely to develop the problem anyway. In some prevention literature, the term "tertiary" prevention is used to describe efforts that occur early in emergence of the target problem—this could involve indicated prevention interventions or early intervention in the form of treatment. Indicated prevention is akin to a severe weather "warning" as a more imminent threat than a "watch."

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

- **Recovery** (the Fact Sheet reverts to the term "Maintenance" in the text, despite their Recovery label on the diagram): "These services support individuals' compliance with long-term treatment and aftercare" (SAMHSA, n.d., p. 2). The diagram

mentions long-term adherence to treatment as fitting into this category, which may or may not reflect what happens during/following treatment for substance use disorder. For example, engaging in mutual help/support programming (such as Alcoholics Anonymous/AA, Narcotic Anonymous/NA, SMART Recovery, Women for Sobriety, LifeRing, Celebrate Recovery, and others) may be a part of both the treatment continuum and the recovery/maintenance continuum.

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

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

vulnerability factors influence one another, underscoring "the importance of (1) intervening early, and (2) developing interventions that target multiple factors, rather than addressing individual factors in isolation" (SAMHSA, n.d., p. 7).

Just as treatment interventions need to be developmentally appropriate, so do prevention interventions. Children and adolescents are qualitatively different from adults; simplifying or "dumbing down" interventions for adults is not sufficient adaptation for younger populations. Because the risk and vulnerability factors are different at different periods of the life cycle, preventive efforts need to be tailored to what is relevant and salient at different periods (SAMHSA, n.d.). Preventive interventions also need to be appropriate for the vulnerability/risk mechanisms operating at different life periods. For example, if the concern is ease of access to substances, intervention might be targeted at the neighborhood/community or policy level rather than individuals; if the concern is to build initiation resistance skills, the intervention might be aimed at the individual level.

The SAMHSA Fact Sheet presented a set of tables of risk and protective factors for substance use disorder mapped to broad developmental period. These tables can help inform prevention strategies and used O'Connell, Boat, & Warner (2009) as their

source. Their tables are replicated [with minor modifications] here and represent general mental health prevention goals at early ages.

Infancy and Early Childhood

Competencies: Infants begin understanding their own and others' emotions, to regulate their attention, and to acquire functional language

Risk Factors	Protective Factors
• *Individual:* difficult temperament • *Family:* parental drug/alcohol use, cold and unresponsive [caregiver] behavior	• *Individual:* self-regulation, secure attachment, mastery of communication and language skills, ability to make friends and get along with others • *Family:* reliable support and discipline for caregivers, responsiveness, protection from harm and fear, opportunities to resolve conflict, adequate socioeconomic resources for the family • *School/community:* support for early learning, access to supplemental services such as feeding and screening for vision and hearing, stable and secure attachment to childcare provider, low ratio of caregivers to children, regulatory systems that support high quality of care

Middle Childhood Competencies: Children learn how to make friends, get along with peers, and understand appropriate behavior in social settings	
Risk Factors	Protective Factors
• *Individual*: poor impulse control, sensation-seeking, lack of behavioral self-control, impulsivity, early persistent behavior problems, attention deficit/hyperactivity disorder, anxiety, depression, antisocial behavior • *Family*: permissive parenting, parent-child conflict, low parental warmth, parental hostility, harsh discipline, child abuse/ maltreatment, substance use among parents or siblings, parental favorable attitudes toward alcohol and/or drug use, inadequate supervision and monitoring, low parental aspirations for child, lack of or inconsistent discipline • *School/community*: school failure, low commitment to school, peer rejection, deviant peer group, [favorable] peer attitudes toward drugs, alienation from peers, law and norms favorable toward alcohol and drug use, availability and access to alcohol	• *Individual*: mastery of academic skills (math, reading, writing), following rules for behavior at home and school and in public places, ability to make friends, good peer relationships • *Family*: consistent discipline, language-based rather than physically-based discipline, extended family support • *School/community*: healthy peer groups, school engagement, positive teacher expectations, effective classroom management, positive partnering between school and family, school policies and practices to reduce bullying, high academic standards

Adolescence Competencies: Adolescents focus on developing good health habits, practice critical and rational thinking, seek supportive relationships [and extend autonomy skills]	
Risk Factors	Protective Factors
• *Individual*: emotional problems in childhood, conduct disorder, favorable attitudes toward drugs, rebelliousness, early substance use, antisocial behavior • *Family*: substance use among parents, lack of adult supervision, poor attachment with parents • *School/community*: school failure, low commitment to school, not college bound, aggression toward peers, associating with peers [engaged in substance use], societal/community norms about alcohol and drug use	• *Individual*: positive physical development, academic achievement/intellectual development, high self-esteem, emotional self-regulation, good coping skills and problem-solving skills, engagement and connections (in school, with peers, in athletics, employment, religion, culture) • *Family*: family provides predictable structure with rules and monitoring, supportive relationships with family members, clear expectations for behavior and values • *School/community*: presence of mentors and support for development of skills and interests, opportunities for engagement within school and community, positive norms, clear expectations for behavior, physical and psychological safety

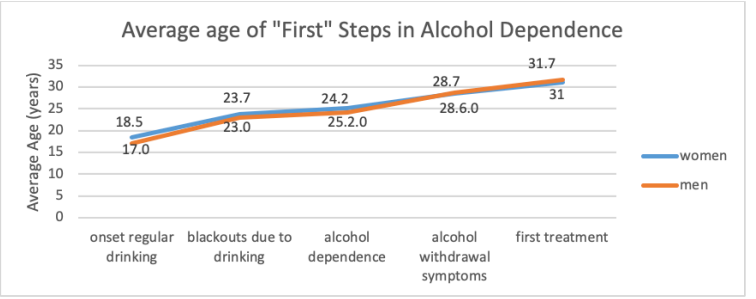
Early [Emerging] Adulthood Competencies: Individuals learn to balance autonomy with relationships to family, make independent decisions, and become financially independent	
Risk Factors	Protective Factors
• *Individual*: lack of commitment to conventional adult roles, antisocial behavior • *Family*: leaving home • *School/community*: attending college, peers [engaged in substance use]	• *Individual*: identity exploration in love and work and developing a world view, subjective sense of adult status, subjective sense of self-sufficiency, making independent decisions, becoming financially independent, future orientation, achievement motivation • *Family*: balance of autonomy and relatedness to family, behavioral and emotional autonomy • *School/community*: opportunities for exploration in work and school, connectedness to adults outside of family

Harm Reduction as Prevention

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

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

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



Prevention Examples

In their book chapter about preventing alcohol and drug problems, McNeece and Madsen (2012) identified a host of efforts and strategies, including at the policy level. At this point, you should turn to the McNeece and Madsen (2012) chapter to become familiar with how they describe primary, secondary, and tertiary prevention

(aligned with universal, selective, and indicated prevention) and their review of the following types of prevention efforts:

- Public Information and Education
- Programs Directed at Children and Adolescents
- Programs Directed at College and University Students
- Service Measures
- Technologic Measures
- Legislative, Regulatory, and Economic Measures
- Family and Community Approaches
- Spirituality and Religious Factors
- Cultural Factors

Don't forget to return to this coursebook for Chapter 3!

An interactive or media element has been excluded from this version of the text. You can view it online here:
https://ohiostate.pressbooks.pub/substancemisusepart1/?p=218

Ch. 3: Theory Integration in the Transtheoretical Model of Behavioral Change

A great deal of effort both in our course and in research has been directed toward understanding the processes involved in substance use initiation and the progression from use to misuse and substance use disorder. At this point, we examine a model concerned with processes of change and recovery–moving back from problematic and disordered substance use into recovery. The model we focus on in this chapter is known as the ***transtheoretical model of behavior change*** (the TTM for transtheoretical model, or sometimes the TMBC for the transtheoretical model of behavior change). The model originally emerged in transtheoretical analysis of psychotherapies (Prochaska, 1979; Prochaska & DiClemente, 1982) and continued to evolve during the 1980s and 1990s based on research concerning the process of change in smoking behavior (Prochaska & DiClemente, 1983) and expanded to include other addictive behaviors (Prochaska, DiClemente, & Norcross, 1992). It has been applied across disciplines (social work, psychology, medicine, nursing, physical therapy, occupational therapy, and others) and across a wide array of behaviors, including but not limited to individuals making changes in their smoking (tobacco), alcohol use, adhering to a medication or medical treatment regimen, dieting, exercising, safe-sex, and intimate partner violence behaviors.

Use of the word "transtheoretical" in the model name reflects its theoretical inclusiveness and that it integrates and applies across theories. The transtheoretical approach represented an important shift in emphasis away from "horse race" research to find "the winner" among intervention options towards identifying

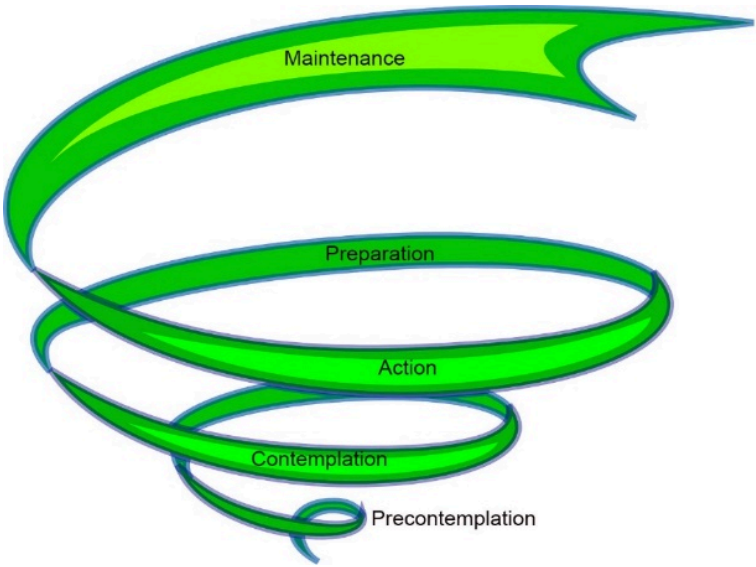
mechanisms of change and the elements or factors common across a variety of intervention approaches. The TTM's developers distilled from research and clinical observation a set of principles describing behavior change processes and factors that facilitate or pose barriers to achieving change goals. The TTM identified a series of five stages in the typical cycle of change, common processes involved in intentional behavior change, and implications for intervening to support individuals' intentional behavior change efforts.

While you review the remainder of this chapter, consider a specific behavior that you have wished or tried to change in the past. See how the model seems to fit your own experience with intentional behavior change (something like getting more sleep, drinking more water, using less electricity, praising your partner or kids more often, spending less money on coffee, stop biting your

fingernails, expressing gratitude for small favors others do for us—it does not have to be about an addictive behavior).

Stages of Change

Like most stage theories, the TTM identified a series of progressive stages that are qualitatively distinct from each other. Originally, the TTM specified four stages: Precontemplation, Contemplation, Action, and Maintenance; data reanalysis led to specification of a fifth stage, Preparation, between Contemplation and Action. An important difference from many other stage theories is acknowledgement that individuals do not move through the stages in a linear "upward" fashion but that they often cycle upwards and downwards through stages as they work to achieve their change goals (Prochaska, DiClemente, & Norcross, 1992). For example, a person perhaps beginning in Precontemplation may progress through some of the other stages, return to prior stages (including back to Precontemplation), and progress again over time, and that this cycle may repeat multiple times before the desired change goal is ultimately achieved. In research concerning smoking cessation, three to four Action attempts occurred before individuals were able to quit smoking for the long-term (Prochaska, DiClemente, & Norcross, 1992)—in other words, relapsing and falling back to earlier stages is normative, not atypical. A determining factor in how quickly someone is able to again move forward in the process concerns how relapse is handled: if seen as a failed attempt, the person may return to precontemplation and remain there for a lengthy period; if seen as an opportunity to learn from one's mistakes, identify potential pitfalls and solutions, the person may move more quickly back into action instead. In fact, one criticism of the TTM is that individuals may move between stages so quickly that assessment tools are rendered inaccurate, and that a person may be situated between stages rather than in a single stage.



Another observation made by the model's developers was that what a person learns about changing one type of behavior may help them learn what will or will not help them change a different type of behavior. However, if someone is concerned about changing two or more behaviors at the same time, the change process for each will most likely differ—in other words, a person may be in one stage for one behavior change effort and a different stage for another. Consider, for example, that someone wishes both to quit smoking cigarettes and to quit drinking alcohol to excess. Each of these change attempts, although occurring at the same time, will progress on its own trajectory (Velasquez, Crouch, Stephens, & DiClemente, 2016). The individual may move through the cycle more quickly with one behavior compared to the other and may spiral back and forward more times. It is difficult enough to change an addictive behavior; it is far more difficult to change more than one at a time.

The five stages identified in the TTM distinguish between the different behaviors, attitudes, experiences, and motivations representing each stage.

Precontemplation. The hallmark of ***Precontemplation*** is the absence of an intent to change the identified behavior, at least not in the foreseeable future. This includes individuals who are un- or under-aware of a need to make changes. It also may include someone who wishes they could change but does not seriously intend to make the changes wished for. This stage also may involve resistance to change in response to pressure from others. For example, if a person is compelled to quit smoking while incarcerated in jail or prison, that individual may only comply as long as extrinsic (external) pressure is applied. There may be no intention to extend the change in behavior to the post-release period. The kinds of statements endorsed by someone in this stage include denial that a problem exists, that the behavior is not problematic, or that it is "their" business and no one else's concern–like the proverbial ostrich with its head buried in the sand. On the other hand, they may engage in blame about the problem ("If I drink too much, it is because you are always nagging me") or focus on an inability to change ("I have tried to quit smoking too many times, face it–I am just a failure" or "It is in my genes, I am destined to die this way.")

Contemplation. A person in the ***Contemplation*** stage demonstrates awareness of a problem and serious consideration of making a change without making a specific change commitment. One characteristic of the Contemplation stage is the person

struggling with the "pros and cons" dilemma—the advantages of making the change versus the disadvantages. For example, someone might realize the health benefits of changing their binge drinking and appreciate the amount of money that could be saved by making a change, but at the same time recognize that they like drinking, would be lonely without binge drinking with "buddies," and that it will take a great deal of effort to make this change (see discussion of decisional balance below). An intention to make significant change within the next six months is considered a characteristic of Contemplation. However, individuals may remain in Contemplation for lengthy periods (despite the "within six months" intent) without moving further in the process—for two years or more among a group of participants in a smoking study (Prochaska, DiClemente, & Norcross, 1992). Examples of statements that a person in Contemplation might endorse generally include awareness of a problem and a desire to make a change: "I think I may have a problem with my drinking," "I am really starting to feel the effects of my smoking when I try to walk upstairs," "I am getting to the point where I can't keep doing this to myself anymore." A person in Contemplation might engage in information-gathering, exploring options for how to make the desired change (even looking into formal intervention/treatment options), but not actually engage with or commit to any of them.

Preparation. The ***Preparation*** stage extends beyond an intent to change to include early change behaviors toward the goal of taking serious action within the next 30 days. They will have set a plan in motion, even if not actively engaged in it yet, and have set a target day/date for the action to begin. For example, the person may enroll in a change-focused program, identify a specific change strategy or plan, and may begin taking "baby-steps" toward the change goal. For example, a person preparing to quit smoking may purchase supply of nicotine replacement "patches" or gum, schedule an appointment for prescription smoking cessation medication, register with a smoking cessation program. In addition, they may break their cigarettes in half to smoke less when they do smoke and gather

together all their "stashed" cigarettes into one, visible collection. They may tell friends and family to refuse their requests to "bum" cigarettes and not invite them to share a smoking session.

Action. The ***Action*** stage is characterized by a person actively taking very specific, concrete steps to change the target behavior and keep the change momentum going. For a behavior as complex as quitting drinking, for example, the person may engage in a host of strategic alternative behaviors: avoiding the people, places, and situations that tempt them to drink; applying strategies for controlling their mood (e.g., mindfulness practices) and stress management (e.g., exercise); grocery shopping online to avoid impulse alcohol purchases in the store. Additionally, they may have new ways of rewarding themselves for each positive step taken (e.g., putting money that would have been spent on alcohol into an account toward a positive goal; celebrating their "sobriety birthday" each week, then month, then year), and reminding themselves of their accomplishments (e.g., journaling their efforts, experiences, and progress). Action is very often the emphasis in treatment programs—teaching, training, and practicing the new skills. A person in Action has specific skills and behaviors to substitute for and manage the old, problematic behaviors and they consciously act to implement these new behaviors. Action, by definition, lasts for at least 6 months and may last much longer for some individuals and some behaviors. Big changes in complex behaviors do not happen overnight. This is a person engaged in multiple, sometimes heroic, action efforts as they are fighting to achieve their change goals.

Maintenance. Once a person has engaged in action behaviors for at least 6 months, they may move into a ***Maintenance*** phase—a period of continued vigilance against relapsing to the past behavior. Individuals continue to engage in relapse prevention activities, but it differs from the Action period in that the new changed/alternative behaviors, attitudes, and experiences are becoming routine and feel relatively natural. They require less effort to maintain. During maintenance, a person continues to be aware that it would take only one "slip up" action to undo their hard work but takes many daily "non-actions" to avoid relapse—consistently avoiding temptations and relapse triggers, engaging in competing alternative behaviors, and managing temptations and relapse triggers when they do appear. A person in maintenance is not "cured" as long as there are temptations or craving experiences—the maintenance period may persist for a very long period, possibly indefinitely for some individuals. However, a person who managed to quit smoking cigarettes (for example) may reach a point when there is no longer any desire to pick it up again, none of their old cues trigger a temptation or desire to smoke, and they self-identify as a non-smoke (rather than an ex-smoker), even in periods of stress/distress. At the point where the changed behavior is relatively effortless, the person may be considered to have moved beyond maintenance.

Relapse. Understanding the change process is incomplete without recognizing what ***relapse*** is and how it might be addressed. Ideally, we want to prevent relapse to the "old" behavior whenever possible; but as the evidence indicates, relapse happens (may even be a "norm" rather than an exception) and what happens in response to relapse matters very much in the future of a change effort. First, a distinction is made between a recurrence ("slip") and a full-blown relapse event. A ***lapse*** or "slip" is time/event limited—doing it once or more times for a short period, quickly regretting the lapse, and getting back to renewed action. The circumstances surrounding a lapse can be effectively used as a learning experience to strengthen the ongoing change effort for the future. Relapse refers to a return to the old pattern of behavior with no intention of changing again—spiraling back to Precontemplation, especially if the person despairs of ever being able to successfully change. A lapse, relapse, or impending relapse can happen at any point in the change process.

Relapse is a process (rather than an event) that starts before substance use occurs again—it is "a gradual process with distinct stages" (Melemis, 2015). The relapse process may begin days or even months before the actual substance use relapse behavior occurs and can be conceptualized in three parts.

- The “emotional” process of relapse is characterized by a lack of emotional, psychological, and physical care (Melamis, 2015). This includes basic physical care (diet, sleep, exercise, hygiene), as well as emotional and social “care” activities. This contributes to the kinds of negative emotional states involved in substance misuse—stress, tension, restlessness, anxiety, fatigue, irritability, and discontent.
- The “mental” relapse process concerns declining cognitive resistance to relapse, increased sensitivity to “use” messages, framing past use more positively (“glamorizing”) and minimizing consequences, entering into bargaining about use (“I’ll only do X and nothing more” or “It will be okay on vacation, just not in my regular life” or “if I stick to beer and avoid “hard” liquor, it will be okay”), scheming/lying, and actually planning a relapse/looking for relapse opportunities (Melamis, 2015). While occasionally thinking about using substances again is a common experience during recovery, a warning sign is when these thoughts become frequent, detailed/specific, and intrusive/insistent in nature.

- “physical” relapse involves actual substance use/misuse—a return to uncontrolled substance use. One-time substance use may not lead to further uncontrolled use or it may contribute to the emotional and mental relapse processes that, in turn, lead to physical relapse. ***Relapse prevention*** involves anticipating and addressing all three parts—emotional, mental, and physical—and having in place plans for identifying/assessing and developing exit strategies for the different threats. This likely includes engaging supportive significant others (asking for help from trusted family/friends; participating actively in recovery-oriented or mutual support groups) and engaging in treatment interventions designed specifically around relapse prevention (e.g., cognitive behavioral interventions and skill building).

Concerted intervention effort might be directed toward relapse prevention, particularly during the maintenance stage.

Change Factors

Threaded throughout the change process are a trio of factors: decisional balance, self-efficacy for change, and timing of different intervention/change promoting strategies.

Decisional balance. Relevant throughout the change process, but particularly in the Precontemplation and Contemplation stages, is the concept of ***decisionalbalance***. The TTM relates to motivation for engaging in the change process. It recognizes that a person who is motivated to make an intentional behavior change may also be motivated NOT to make the change. There exist costs and benefits on all sides of the decision and a person may see-saw back up and down as the balance shifts toward or away from making the change effort. There are four dimensions of which the person is aware and that have implications for the likelihood of embarking on a change effort:

		Changing	
		Pros	Cons
Not Changing	Pros	*ambivalence*	*no change*
	Cons	*yes change*	*ambivalence*

Decisional balance underlies the ambivalence identified and addressed in motivational interviewing (MI). Eliciting and sustaining motivation for change often requires addressing ambivalence, not just emphasizing the advantages of changing and disadvantages of not changing the behavior. Decisional balance is particularly impactful in the Precontemplation, Contemplation, and Preparation stages, but continues to have a role across the process.

Self-efficacy for change. Another cognitive process involved in each stage of the intentional behavior change process concerns a person's belief that change (or maintaining change) is possible: their ***self-efficacy*** for making or sustaining the change goal. Like *The Little Engine that Could*, self-efficacy ranges from "I *can't*" to "I *think I can*" to "I *know I can*" and makes a difference in motivation at all stages of the change process. Someone might be in the Precontemplation stage (no plan to change) because they do not believe it is possible, despite being aware of that their behavior is problematic. This may be because they have made unsuccessful change attempts in the past and feel it is a hopeless goal. Two strategies for assisting with motivation in this situation are (1) focus on ways that they have succeeded in the past, including any positive steps they may have made in changing this behavior or any other behaviors they may have been able to change in the past, and (2) examining how others most like themselves have managed the change process. A conversation that might elicit self-efficacy involves a "change ruler" whereby a person indicates on a scale from 1-10 how confident they are in their ability to make the desired change in a situation of temptation. Rather than focusing on how far from 10 they are, the value lies instead on exploring why the rating is greater than 0—what the person may have going for them.

Intervention timing. Matching intervention strategies to "where the person is" with their change process, achieving the right timing, is an important consideration related to the TTM (Velasquez et al., 2016). "Action-oriented therapies may be quite effective with individuals who are in the preparation or action stages. These same programs may be ineffective or detrimental, however, with individuals in precontemplation or contemplation stages" (Prochaska, DiClemente, & Norcross, 1992, p. 1106). Similarly, individuals ready for action and learning change-based skills may become frustrated and drop out of interventions aimed at raising their awareness of the problem and why they might need to make change—they are already past that point and ready to engage actively in change efforts. In other words, intervention efforts should be timed so as to connect to the relevant change goals at any point in time. Ideally, these fit together like puzzle pieces, and are adapted as the situation changes over time. For example, in efforts to move from Precontemplation to Contemplation, consciousness raising might be appropriate, whereas Action-oriented efforts might include creating a system of positive reinforcement for changed behavior and other change skill sets (Prochaska, DiClemente, & Norcross, 1992; Velasquez et al., 2016). While much of the TTM approach and motivational interviewing reflect the individual's thoughts, feelings, experiences, and behaviors, it can effectively be applied in group work settings (Velasquez et al., 2016).

Thinking about the material you read in this chapter and the specific change effort example you were considering:

- What did you conclude about how the model seems to fit your own experience with intentional behavior change?
- How did you experience the stages of change and did you follow a single progression or spiral up/down the cycle?
- How did decisional balance, ambivalence, and self-efficacy for change look in your chosen example?
- What did or could have helped and what might have gotten in the way of your change effort?
- What does this tell you about possibly supporting others in their efforts to change, even to change addictive behaviors?

Ch. 4: Summary

In this module you learned about different ways that the various biological, psychological, and social context/physical environment theories and models related to substance use, substance misuse, and substance use disorders might be integrated into a coherent biopsychosocial framework. We considered how the different theories, models, and evidence might inform prevention and change-promoting strategies. First, you read about four steps in a vulnerability, risk, resilience, and protective factors approach to substance misuse-related problems. The grid that can be created from evidence in the literature can inform prevention strategies at the population/subgroup level (not at the individual assessment level). Next, you were introduced to several key concepts and principles in prevention. Introduction to the continuum of care model included descriptions and examples of health promotion, prevention (universal, selective, indicated), treatment, and recovery/maintenance. Vulnerability/risk and resilience/ protective factors related to substance misuse and to different developmental periods were presented as means of informing developmentally appropriate preventive intervention strategies. You read an analysis by McNeece and Madsen (2012) of evidence surrounding various prevention approaches, a well. Finally, you were introduced to the transtheoretical model of behavior change (TTM or TMBC), including stages commonly observed in the intentional behavior change process and factors that are important in the change process. These elements and factors were related to intervention strategies that might facilitate movement through the change process and assist individuals in achieving their behavior change goals.

At this point, we have concluded much of the work related to theories of substance use, misuse, and use disorders. We are now ready to launch into the second half of the course and look at

specific types of substances, as well as topics related to prescription and over-the-counter drug misuse, pharmacotherapy opportunities, and how co-occurring problems might affect substance-related problems and outcomes.

Module 6: Key Terms

Action: the fourth of five stages in the transtheoretical model of behavior change, characterized by taking very specific, concrete, active steps to change the target behavior and keep the change momentum going.

adverse childhood events (ACES): potentially traumatizing experiences or events occurring during childhood that can or do have a persistent, negative impact on physical health, emotional health, behavioral/mental health, well-being, or development.

Contemplation: the second of five stages in the transtheoretical model of behavior change, characterized by awareness and ownership of that a problem exists and a general intent to change in the relatively near future but no concrete impending plan to change.

continuum of care framework: depicting an array of service/intervention options as representing different aspects of health promotion, prevention, treatment, and recovery/maintenance.

decisional balance: a process in intentional behavior change whereby an individual is aware of the pros and cons of both changing and not changing the target behavior.

gateway drug theory: a theory that use of one type of substance serves as a prelude to use of a different type.

indicated prevention: interventions delivered to populations/groups of individuals exhibiting/expressing warning signs foreshadowing development of the focal problem.

lapse: engaging in a limited way in a behavior that has been the target of an intentional behavior change effort (distinct from relapse).

Maintenance: the fifth of five stages in the transtheoretical model of behavior change, characterized by normalizing changed behaviors and relapse prevention efforts.

Precontemplation: the first of five stages in the transtheoretical

model of behavior change, characterized by a lack of intent to change a particular behavior either due to a lack of problem awareness or low self-efficacy for being able to successfully change.

Preparation: the third of five stages in the transtheoretical model of behavior change, characterized by efforts to set oneself up to actively engage in change efforts within the next 30 days (one month), potentially including initial change steps which may not be successful.

promotion strategies: strengths-based interventions designed to build resilience and promote well-being.

relapse: an emotional, mental, physical process whereby an individual returns or risks return to a past behavior pattern that was the target of intentional behavior change.

relapse prevention: efforts designed to identify relapse risk factors and intervene before an individual in recovery re-engages with the problem behavior.

risk/protective continuum: refers to extrinsic factors that increase (risk) or decrease (protective) the probability of a specific problem emerging, across a continuity of probabilities.

selective prevention: interventions directed towards populations identified as having a potential somewhat greater than the general population for developing the focal problem.

self-efficacy: a process in intentional behavior change whereby individuals experience differing degrees of belief in their ability to succeed in their change effort and/or to sustain the desired change over time.

transtheoretical model of behavior change (TTM or TMBC): a model of the processes and stages typically experienced in the course of intentional behavior change.

universal prevention: interventions delivered to the general population without differentiating between persons at different risk levels.

vulnerability/resilience continuum: refers to intrinsic factors that

increase (vulnerability) or decrease (resilience) the probability of a specific problem emerging, across a continuity of probabilities.

Module 6: References and Image Credits

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Appendix - Syllabus

Click here to download the master syllabus for this course.