BI 358 Discussion Session 2

:30 ***I.*** ***Group Discussion*****: DLN 2-1 thru 2-6**

A. ***Cannibas in the Clinic: The Medical Marijuana Debate***
http://learn.genetics.utah.edu/content/addiction/cannabis/

B. ***Opioid Addiction***
https://www.pbs.org/newshour/science/brain-gets-hooked-opioids
https://www.pbs.org/newshour/health/a-drug-counselor-sees-the-perils-of-jailing-her-son-for-addiction

:40 ***II.*** ***Class Convenes*****: w/group summary statements.**

:45 ***III.*** ***Regroup*** **(new groups!) for DLN review.**
Try to incorporate general model of addiction after D.O. ***Norris Simplified Homeostatic Model*****.**
#1 Alcohol DLN 2-7 thru 2-14
#2 Cocaine DLN 2-15 thru 2-28
#3 Heroin DLN 2-29 thru 2-34
#4 Kratom DLN 2-35 thru 2-38
#5 Marijuana DLN 2-39 thru 2-47
#6 Methamphetamine DLN 2-48 thru 2-59
#7 Tobacco-Nicotine DLN 2-62 thru 2-74

:10 ***IV.*** ***Informal Group Overhead Presentations***

Paper Topic + 4 copies of Brief Outline due next T!
Be sure to visit the fabulous University of Utah website
http://learn.genetics.utah.edu/content/addiction/mouse/

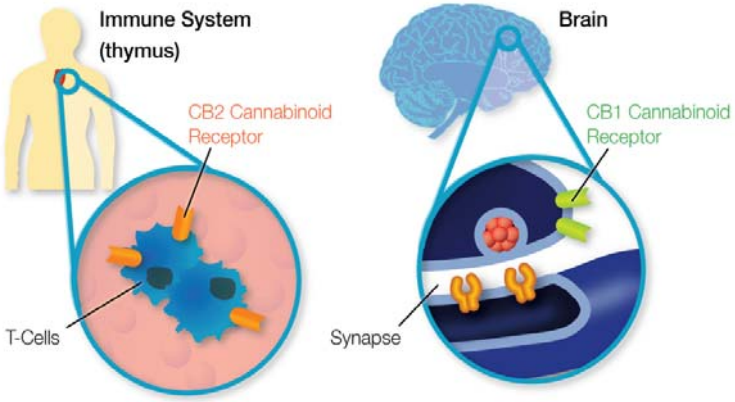
SOURCE: University of Utah Health Sciences. Genetic Science Learning Center, 2017. *http://learn.genetics.utah.edu/content/addiction/cannabis/*

Cannabis in the Clinic? The Medical Marijuana Debate

Until its prohibition in 1937, extract of Cannabis sativa (marijuana) was one of the top three most prescribed medicines in the US. When it became illegal, its use as a medicine became restricted. Despite these regulations, research on the medical use of marijuana continued.

In recent years, when some states decided to legalize smoked marijuana for certain patients, medical marijuana became a subject of contentious debate. Should patients be allowed to grow their own plants? Might medical use inevitably lead to recreational use?

The Endocannabinoid System

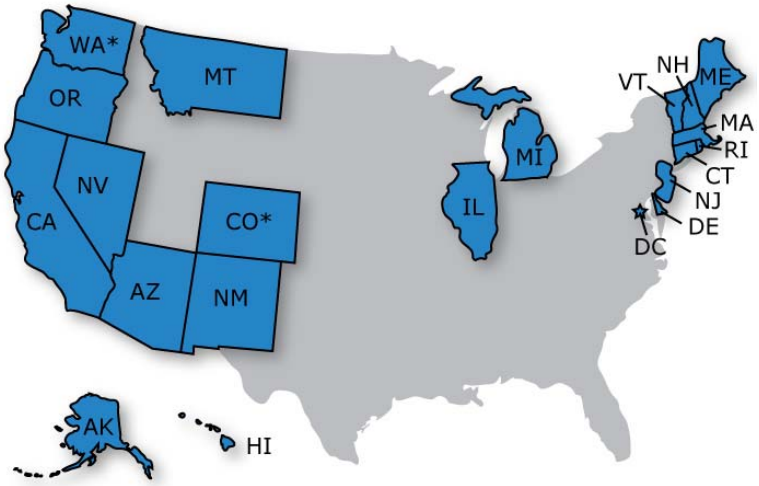


We have two types of cannabinoid receptors: CB1 receptors are expressed on the surface of neurons, and CB2 receptors are expressed on cells of the immune system.

The active compounds in marijuana are similar to a class of molecules in our bodies called endocannabinoids. The endocannabinoid system influences our immune system, protects nerve cells from premature death, and influences mood, memory, appetite, sleep, sensation, and movement. Both endocannabinoids and the compounds in marijuana bind to proteins called cannabinoid receptors in the brain and throughout the body.

"Endocannabinoids regulate every one of the systems in our bodies." --Dr. Robert Melamede

Defining Medical Marijuana



As of October, 2013, 20 states plus Washington, DC, have legalized medical marijuana. Two states (*) also allow recreational use. The trend is moving toward legalization. (The Wikipedia page Medical cannabis in the United States has up-to-date legal information.)

Each state has its own regulations to control details such as allowable quantities and registration requirements. However, in June of 2005, the Supreme Court ruled that individuals in all states can still be prosecuted under federal law, under which medical marijuana remains illegal under the Controlled Substances Act

"There are different kinds of pain." --Dr. Robert Melamede

In 1970, Congress classified cannabis and THC (one of the active compounds in cannabis) as Schedule I drugs under the Controlled Substances Act. Schedule I drugs are defined by Congress as having no medicinal value. The one exception to this classification allows for use in FDA-approved research programs.

Who can benefit?
Research suggests that there are conditions for which medical marijuana may be an effective treatment:

- Cancer - Relieves nausea during chemotherapy treatment, may prevent the spread of some cancers.
- HIV/AIDS - Increases appetite in patients experiencing severe weight loss, eases neurological symptoms.
- Neurological disorders (including spinal cord injury and multiple sclerosis) - Reduces pain and spasticity resulting from nerve damage.
- Inflammatory pain - Cannabinoids seem to be more effective than opiates in treating long-term, chronic pain. (Opiates are better for treating short-term acute pain.)
- Autoimmune diseases (such as arthritis) - Suppresses the immune system, decreasing pain and inflammation.

Cannabinoid Delivery Methods

The active compounds in marijuana are available in several forms and can be administered in a variety of ways. Each delivery method has benefits and disadvantages.

Smoking

(+) Delivers all of the plant's active compounds.
(+) Easy to regulate dose (patients smoke until symptoms are eased, but are not intoxicated).
(–) No standardization. Amounts of active ingredients may vary.
(–) Burning marijuana produces toxins which can cause emphysema and lung cancer.
(–) Illegal in most states.

Marinol - Synthetic THC in pill form

(+) Legal in the US.
(+) Delivers some of the benefits of the whole plant.
(–) Difficult to control dose.
(–) Contains only one of the plant's active compounds (THC).
(–) Patients report fewer of the positive effects and more negative side effects.

Vaporizor

(+) Converts the active compounds to inhalable form without releasing toxins.
(+) Delivers all of the plant's active compounds.
(+) Easy to control dose.
(–) No standardization. Amounts of active ingredients may vary.
(–) Illegal in most states.

Sativex (nabiximols) - Extract from plants delivered as a spray

(+) Contains all of the plant's active compounds.
(+) Concentrations of active ingredients are standardized.
(+) Relatively easy to regulate dose.
(+) Legally approved for the medical treatment of Multiple Sclerosis.
(–) Legal in a limited number of countries.

Marijuana contains approximately 66 active compounds with different properties, collectively called cannabinoids. Scientists are studying cannabinoids to understand their individual and combined effects and their potential benefits.

"Studies show that Sativex (marijuana extract) is not as addicting as legal prescription medications such as opiates." --Dr. William Notcutt, Director of Sativex trials

Social Implications

Some argue that Congress should change marijuana's classification under the Controlled Substances Act. If it were no longer a Schedule I drug, its medicinal benefits could be recognized legally. But if medical marijuana were legalized, there could be repercussions outside the realm of medicine.

Opponents worry that legalizing medical marijuana might lead teens to believe that marijuana is safe for recreational use and increase availability of the drug. On the other hand, some supporters think changing the perception of marijuana from a party drug to a medication might make it less attractive to teens wanting to defy or rebel.

Legal Implications

Legalized medical marijuana also presents lawmakers with challenges. How would federal, state, and local governments control and regulate the production, distribution, and sale of medical marijuana? Who would define what is recreational versus medical use of the drug, and how would that be enforced?

Please see the following link for Discussion on America Addicted:

https://www.pbs.org/newshour/features/america-addicted/

Jot down any comments you would like to make or notes here:

Alcohol Alert

National Institute on Alcohol Abuse and Alcoholism

Number 90

Improving Health Through Translational Alcohol Research

Alcohol misuse has profound effects on the health and well-being of individuals, families, and communities. In the United States, nearly 88,000 people—approximately 62,000 men and 26,000 women—die from alcohol-related causes each year.[1] Alcohol misuse not only increases the risk for alcohol use disorder (AUD),[a] which affects nearly 17 million people in the United States, but it contributes to a wide range of adverse social, vocational, and health conditions. These include traffic fatalities, injuries, poor academic performance, alcoholic liver disease, alcoholic pancreatitis, certain cancers, and fetal alcohol spectrum disorders, among many others.[2] Over the past several decades, scientists have made great strides in illuminating the biological and behavioral underpinnings of alcohol misuse and "translating" this knowledge into effective preventive and treatment interventions for AUD and other substance use disorders.

Translational research is the process of turning observations made in the laboratory, clinic, and community into interventions that improve health—from diagnostics and therapeutics to medical procedures and behavior change.[3] Although translational research has long been a goal of the National Institutes of Health (NIH), in recent years, NIH has expanded its focus on this research area. Translation occurs at multiple points along the scientific research continuum, including from basic to clinical research, from clinical research to clinical practice, and from clinical practice to the implementation of public health interventions (see figure). For example, translational alcohol researchers may seek to demonstrate how alcohol's effects on the brain and on behavior at the fundamental level (e.g., in cells or animals) are relevant to humans. Others seek to translate our understanding of the mechanisms by which alcohol affects human health and well-being into interventions for preventing or treating those effects.

Translation does not stop there, however. Interventions that prove effective in people must then be adopted into healthcare practice. Translational researchers working at this stage of the continuum may study how to increase the adoption of healthy drinking behaviors or increase clinician use of alcohol screening or pharmacotherapies. They may also conduct research to understand how interventions that have been adopted into clinical practice affect health at the population level. Importantly, translation does not proceed in one direction. Just as basic research informs clinical studies and clinical practice, observations made in studies with people and by clinicians at the frontlines of patient care can drive new basic and clinical research questions. Such "reverse" translation is also important for validating basic research models for preventing, diagnosing, and treating disease.

This *Alert* addresses an active area of translational research—the neurobiology of AUD. Drawing on human and animal research, it offers a brief overview of the changes in the brain that underlie the

[a] The *Diagnostic and Statistical Manual of Mental Disorders* (DSM–5) describes AUD as a problematic pattern of alcohol use leading to clinically significant impairment or distress. AUD may be categorized as mild, moderate, or severe depending on the number of diagnostic criteria that a patient meets. American Psychiatric Association (APA). *Diagnostic and Statistical Manual of Mental Disorders (5th Ed.)*. Washington, DC: APA, 2013.

development and progression of AUD—from moderate to excessive to compulsive drinking—and how our understanding of these changes is informing—or being translated into—the development of interventions to help people reduce or abstain from alcohol use altogether.

Alcohol's Influence on the Brain

Scientists have long understood that our brains are adaptable. One type of adaptation occurs through "synaptic plasticity," a process through which connections among brain cells, called synapses, transform over time. When positive, these changes, or neuroadaptations, can enhance the efficiency of brain functioning, such as when learning a new task. But plasticity also can be harmful, causing disruptions in brain circuitry, as can occur with chronic, excessive drinking.

"A growing body of evidence indicates that neuroadaptations in these brain regions drive the development and progression of AUD."

Research has demonstrated that chronic alcohol misuse produces negative changes in three regions of the brain, each associated with one of the three stages of the AUD cycle:[4]

- The *basal ganglia*, which is involved in the *binge/intoxication* stage of AUD. This is the stage at which an individual consumes alcohol and experiences its rewarding or pleasurable effects.
- The *extended amygdala*, which is involved in the negative emotional state that individuals with AUD experience in the absence of alcohol during the *withdrawal/negative* affect stage.
- The *prefrontal cortex*, which is involved in behavioral control and decisionmaking, underlies the "preoccupation/anticipation" stage of AUD during which individuals experience a compulsion to drink.

A growing body of evidence indicates that neuroadaptations in these brain regions drive the development and progression of AUD.[5–8] On the hopeful side, reversing these neuroadaptations may help individuals with AUD maintain sobriety or return to low-risk drinking.

Basal Ganglia: The basal ganglia include two subregions that are particularly important in substance use disorders: the nucleus accumbens, which is involved in motivation and the experience of reward, and the striatum, which is involved in habit formation and other routine behaviors. Repeated activation of the reward system of the nucleus accumbens by alcohol can trigger changes in the striatum, leading to the development of habitual or compulsive alcohol seeking. In addition, the stimuli that are present when people drink—including people, places, and even their own internal mood states—can become associated with the pleasurable effects of alcohol. Over time, these cues may acquire the ability to activate the brain's reward systems even in the absence of alcohol. This helps explain the intense craving and compulsive alcohol seeking that occurs when some people with AUD are exposed to stimuli they have come to associate with drinking.

Evidence for the role of the basal ganglia in AUD comes from research conducted with animals and humans. For example, animal studies show that chronic alcohol consumption disrupts communication between cells within the basal ganglia and in circuits connecting the cortex to the basal ganglia. This abnormal communication is linked to alterations in how animals respond to alcohol and to alcohol-associated cues, such as its smell.[5] Animal studies also show that alterations in the striatum are associated with habitual alcohol seeking and drinking behaviors similar to those observed in people with AUD.[5] Using functional magnetic resonance imaging (fMRI), a noninvasive neuroimaging technique that measures brain activity, researchers found that activity in the striatum increases when heavy[b] drinkers[8] and people with AUD drink or are exposed

[b] Participants in this study reported drinking an average of 11.8 times per month and an average of 5.53 drinks per drinking occasion. The Substance Abuse and Mental Health Services Administration defines heavy drinking as drinking 5 or more drinks on the same occasion on each of 5 or more days in the past 30 days.

to alcohol-associated cues.[6] This activity is highly correlated with alcohol craving. Even patients with a history of AUD who have stopped drinking show alterations in how they process stimuli associated with reward. For example, compared with people who lack a history of AUD, detoxified AUD patients showed greater activity in the striatum when presented with cues associated with alcohol, but less activation in that area of the brain when expecting a monetary reward. Together, these and other studies suggest that repeated alcohol use alters the reward and habit pathways in the brain, making individuals more sensitive to environmental stimuli associated with alcohol and increasing alcohol craving, seeking, and drinking.[8]

Extended Amygdala: AUD is associated with heightened stress sensitivity, anxiety, and depressive symptoms, and individuals with severe AUD often experience these symptoms when they stop drinking.[8] These negative feelings are thought to stem from disruptions in the brain's reward circuits, as well as activation of the brain's stress systems in the extended amygdala. Studies with animals have found that short- and long-term alcohol use disrupts synaptic plasticity in the extended amygdala. They have also found associations between altered neuronal responses in the extended amygdala and stress-triggered drug use. Moreover, stress neurotransmitters—chemical messengers that relay information throughout the nervous system—are activated in the extended amygdala during alcohol withdrawal. Blocking the activation of these chemical messengers has been shown to reduce alcohol consumption both in animal models of AUD and in people with AUD.[9] Additional evidence for a role of the extended amygdala in AUD comes from brain-imaging studies: researchers have found that the volume of the amygdala is smaller in individuals with AUD compared with those without the disorder, and smaller amygdala volume is associated with increased alcohol craving and relapse.[8]

Prefrontal Cortex: The prefrontal cortex (PFC) aids in organizing thoughts, controlling behavior, and making decisions, a set of skills known as "executive function." Executive function is necessary for making appropriate choices about whether or not to drink and for overriding strong urges to drink that people with AUD may experience, particularly when faced with stress or alcohol cues as described above. Brain-imaging studies consistently show that people with severe AUD have structural and functional abnormalities in the PFC.[7]

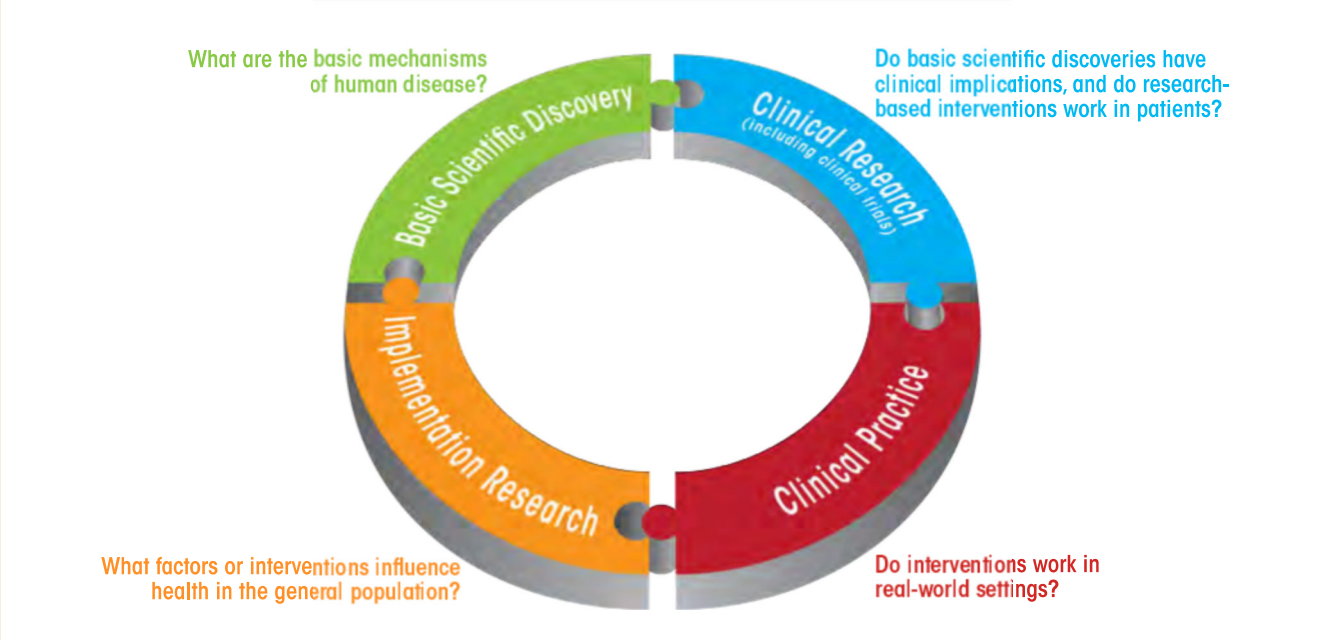


Figure Translational research is the process of transforming discoveries made in the laboratory and clinic into interventions that improve public health. Translation occurs at multiple points along the scientific research continuum, including from basic to clinical research, from clinical research to clinical practice, and from clinical practice to the implementation and dissemination of public health interventions.

They generally have less activity in executive function networks responsible for decisionmaking and impulse control, and higher activity in the areas involved in the rewarding or pleasurable effects of alcohol and other drugs.[6] This pattern is opposite in people who succeed in maintaining abstinence. Moreover, compared with people without a history of substance misuse, people with or recovering from AUD appear to need to marshal more regions of the brain to perform as well on certain cognitive tasks. Recruitment of additional brain regions may be a way of compensating for the compromise of function in brain circuits that normally would be responsible for carrying out such tasks.

Another study found that smaller prefrontal cortex volume predicted a shorter time to relapse in abstinent individuals with AUD. Studies of individuals with damage to the PFC also suggest a role for this brain region in AUD. Using an experimental gambling task, researchers have found that both people with AUD and people with PFC damage tend to make impulsive choices that result in immediate monetary rewards, even if those choices lead to greater future losses.[10] This behavior is similar to the behavior observed in some people with AUD and other substance use disorders who may compulsively use drugs or alcohol in spite of the potential negative consequences.[7] These data suggest that alcohol-induced disruptions in PFC function may underlie the executive function deficits exhibited by individuals with AUD.

From Brain Circuits to Targeted Therapy

As a whole, studies find that brain circuits linking the basal ganglia, extended amygdala, and prefrontal cortex play a pivotal role in the reinforcing effects of alcohol (i.e., those effects that make a person want to continue drinking), habitual alcohol use, stress, and decisionmaking throughout the course of AUD.[8] An ongoing challenge for scientists is to translate these findings into interventions for preventing and treating the disorder. Two of the three medications currently approved by the U.S. Food and Drug Administration for the treatment of AUD appear to work by targeting the processes described above. Naltrexone, for example, helps to reduce heavy drinking by diminishing alcohol's rewarding effects. Acamprosate, in contrast, reduces the negative experiences associated with alcohol withdrawal, and some studies show that it also diminishes craving, making it easier for people to maintain abstinence. Researchers are currently investigating numerous other compounds as potential treatments for AUD, including those that target brain–stress systems. Recognizing that no one AUD treatment is likely to be effective for all people, scientists are studying genetic variants that might help to predict how well a person with AUD will respond to a particular medication.[11] Like developing new AUD treatments, identifying the individuals most likely to benefit from a particular treatment is an important goal of translational alcohol research that will help clinicians tailor treatment strategies to the needs of their patients.

"Researchers are currently investigating numerous other compounds as potential treatments for AUD, including those that target brain–stress systems."

In addition to continuing to develop medications, alcohol researchers are exploring exciting nonpharmacological AUD treatment approaches based on what we have learned about alcohol's effects on brain circuits. One area of investigation involves examining how patterns of brain activity may be used to predict treatment outcomes. For example, using functional magnetic resonance imaging (fMRI), researchers have found that individuals with AUD who maintain abstinence have less synchrony in brain–reward networks and greater synchrony in behavioral control networks than individuals without a history of substance use disorder.[6] fMRI studies have also found that greater activity in the prefrontal cortex and striatum during the performance of tasks designed to measure cognitive control is associated with better outcomes in substance use disorder treatment.[12] Another study found that behavioral therapy resulted in a decrease in the quantity and frequency of adolescent drinking, and these changes were correlated with brain-activation patterns in response to the language the therapist used (i.e., asking youth open-ended vs. "yes"/"no" questions about their substance use).[13]

Researchers are also investigating whether altering the activity of brain networks involved in AUD can influence drinking.[14] One research team found that using transcranial magnetic stimulation (TMS)—a procedure that stimulates the brain using a magnetic coil placed near the scalp—to increase PFC activation made it easier for people to exert control over their alcohol craving.[6] Other investigators are exploring the use of electroencephalography (EEG), a technique that measures patterns of electrical activity in the brain using electrodes attached to the scalp, to treat AUD.[6,8,15] Scientists have found that individuals with AUD have abnormal EEG patterns. Training them to manipulate these signals—a technique called neurofeedback—may hold promise for treating AUD. Studies of EEG neurofeedback as a treatment for substance use disorders are not new. In fact, they date back to the 1970s. Relatively recent advances in the field, however, are allowing researchers to measure EEG activity in *specific* areas of the brain. That, coupled with an improved understanding of the brain regions involved in alcohol and other substance use disorders, may lead to more effective use of this technique.[6]

Conclusion

Alcohol use disorder is an extraordinarily complex and formidable public health problem. Nearly 30 percent of the U.S. population has experienced AUD at some point in their lives.[16] Basic neurobiological and behavioral research has laid the foundation for the development of a host of effective AUD treatments and continues to drive progress in the field. However, the vast majority of people in the United States who have experienced AUD have not received treatment or help. Moreover, existing behavioral and pharmacological interventions, although effective, work better for some people than they do for others. This underscores the need for continued research aimed at further illuminating the mechanisms through which alcohol contributes to disease and disability, translating this knowledge into a broader array of effective interventions, and ensuring that these interventions are delivered to and used by the people who will benefit from them.

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[1] **National Institute on Alcohol Abuse and Alcoholism (NIAAA).** *Alcohol Facts and Statistics.* Bethesda, MD: NIAAA, 2016. Available at: http://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/alcohol-facts-and-statistics. Accessed November 29, 2016. [2] **Center for Behavioral Health Statistics and Quality.** *Behavioral Health Trends in the United States: Results from the 2014 National Survey on Drug Use and Health* (HHS Publication No. SMA 15–4927, NSDUH Series H–50). Rockville, MD: Substance Abuse and Mental Health Services Administration, 2015. Available at: http://www.samhsa.gov/data/sites/default/files/NSDUH-DetTabs2014/NSDUH-DetTabs2014.htm#tab5-8a. Accessed November 29, 2016. [3] **Batman, A.M., and Miles, M.F.** Translating alcohol research: Opportunities and challenges. *Alcohol Research: Current Reviews* 37(1):7−14, 2015. PMID: 26259085 [4] **Koob, G.F., and Volkow, N.D.** Neurocircuitry of addiction. *Neuropsychopharmacology* 35(1):217–238, 2010. PMID: 19710631 [5] **Lovinger, D.M., and Kash, T.L.** Mechanisms of neuroplasticity and ethanol's effects on plasticity in the striatum and bed nucleus of the stria terminalis. *Alcohol Research: Current Reviews* 37(1):109−124, 2015. PMID: 26259092 [6] **Fein, G., and Cardenas, V.A.** Neuroplasticity in human alcoholism: Studies of extended abstinence with potential treatment implications. *Alcohol Research: Current Reviews* 37(1):125−141, 2015. PMID: 26259093 [7] **Naqvi, N.H., and Morgenstern, J.** Cognitive neuroscience approaches to understanding behavior change in alcohol use disorder treatments. *Alcohol Research: Current Reviews* 37(1):29−38, 2015. PMID: 26259087 [8] **Seo, D., and Sinha, R.** Neuroplasticity and predictors of alcohol recovery. *Alcohol Research: Current Reviews* 37(1):143−152, 2015. PMID: 26259094 [9] **Vendruscolo, L.F.; Estey, D.; Goodell, V.; et al.** Glucocorticoid receptor antagonism decreases alcohol seeking in alcohol-dependent individuals. *Journal of Clinical Investigation* 125(8):3193−3197, 2015. PMID: 26121746 [10] **Bechara, A.; Dolan, S.; Denburg, N.; et al.** Decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in alcohol and stimulant abusers. *Neuropsychologia* 39(4):376–389, 2001. PMID: 11164876 [11] **Seneviratne, C., and Johnson, B.A.** Advances in medications and tailoring treatment for alcohol use disorder. *Alcohol Research: Current Reviews* 37(1):15−28, 2015. PMID: 26259086 [12] **Brewer, J.A.; Worhunsky, P.D.; Carroll, K.M.; et al.** Pretreatment activation during Stroop task is associated with outcomes in cocaine-dependent patients. *Biological Psychiatry* 64(11):998−1004, 2008. PMID: 18635157 [13] **Feldstein Ewing, S.W.; Houck, J.M.; Yezhuvath, U.; et al.** The impact of therapists' words on the adolescent brain: In the context of addiction treatment. *Behavioural Brain Research* 297:359−369, 2016. PMID: 26455873 [14] **Mayo Clinic Staff.** *Transcranial Magnetic Stimulation: Overview.* Rochester, MN: Mayo Clinic, 2015. Available at: http://www.mayoclinic.org/tests-procedures/transcranial-magnetic-stimulation/home/ovc-20163795. Accessed November 29, 2016. [15] **Kamarajan, C., and Porjesz, B.** Advances in electrophysiological research. *Alcohol Research: Current Reviews* 37(1):53−87, 2015. PMID: 26259089 [16] **Grant, B.F.; Goldstein, R.B.; Saha, T.D.; et al.** Epidemiology of *DSM-5* alcohol use disorder: Results from the National Epidemiologic Survey on Alcohol and Related Conditions **III.** *JAMA Psychiatry* 72(8):757−766, 2015. PMID: 26039070

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Resources

Source material for this *Alcohol Alert* originally appeared in *Alcohol Research: Current Reviews,* 2015, Volume 37, Number 1.

Through translational research, scientists are turning discoveries made in basic and clinical research laboratories into new and improved applications in health and medicine. Moreover, the information gained through clinical research and practice is stimulating new directions in basic science. This issue of *Alcohol Research: Current Reviews* highlights the bidirectional nature of translational alcohol research and explores how today's investigations are setting the stage for tomorrow's interventions to prevent and treat alcohol misuse and alcohol use disorder.

For more information on the latest advances in alcohol research, visit NIAAA's Web site, **www.niaaa.nih.gov**

Alcohol's Effects on the Body

Drinking too much – on a single occasion or over time – can take a serious toll on your health. Here's how alcohol can affect your body:

Brain:
Alcohol interferes with the brain's communication pathways, and can affect the way the brain looks and works. These disruptions can change mood and behavior, and make it harder to think clearly and move with coordination.

Heart:
Drinking a lot over a long time or too much on a single occasion can damage the heart, causing problems including:

- Cardiomyopathy – Stretching and drooping of heart muscle
- Arrhythmias – Irregular heart beat
- Stroke
- High blood pressure

Liver:
Heavy drinking takes a toll on the liver, and can lead to a variety of problems and liver inflammations including:

- Steatosis, or fatty liver
- Alcoholic hepatitis
- Fibrosis
- Cirrhosis

Pancreas:
Alcohol causes the pancreas to produce toxic substances that can eventually lead to pancreatitis, a dangerous inflammation and swelling of the blood vessels in the pancreas that prevents proper digestion.

Cancer:
Source: National Cancer Institute -- see https://www.cancer.gov/about-cancer/causes-prevention/risk/alcohol/alcohol-fact-sheet:

Based on extensive reviews of research studies, there is a strong scientific consensus of an association between alcohol drinking and several types of cancer. In its Report on Carcinogens, the National Toxicology Program of the US Department of Health and Human Services lists consumption of alcoholic beverages as a known human carcinogen. The research evidence indicates that the more alcohol a person drinks—particularly the more alcohol a person drinks regularly over time—the higher his or her risk of developing an alcohol-associated cancer. Based on data from 2009, an estimated 3.5 percent of all cancer deaths in the United States (about 19,500 deaths) were alcohol related.

Clear patterns have emerged between alcohol consumption and the development of the following types of cancer:

- **Head and neck cancer**: Alcohol consumption is a major risk factor for certain head and neck cancers, particularly cancers of the oral cavity (excluding the lips), pharynx (throat), and larynx (voice box). People who consume 50 or

more grams of alcohol per day (approximately 3.5 or more drinks per day) have at least a two to three times greater risk of developing these cancers than nondrinkers. Moreover, the risks of these cancers are substantially higher among persons who consume this amount of alcohol and also use tobacco.

- **Esophageal cancer**: Alcohol consumption is a major risk factor for a particular type of esophageal cancer called esophageal squamous cell carcinoma. In addition, people who inherit a deficiency in an enzyme that metabolizes alcohol have been found to have substantially increased risks of alcohol-related esophageal squamous cell carcinoma.
- **Liver cancer**: Alcohol consumption is an independent risk factor for, and a primary cause of, liver cancer (hepatocellular carcinoma). (Chronic infection with hepatitis B virus and hepatitis C virus are the other major causes of liver cancer.)
- **Breast cancer**: More than 100 epidemiologic studies have looked at the association between alcohol consumption and the risk of breast cancer in women. These studies have consistently found an increased risk of breast cancer associated with increasing alcohol intake. A meta-analysis of 53 of these studies (which included a total of 58,000 women with breast cancer) showed that women who drank more than 45 grams of alcohol per day (approximately three drinks) had 1.5 times the risk of developing breast cancer as nondrinkers (a modestly increased risk). The risk of breast cancer was higher across all levels of alcohol intake: for every 10 grams of alcohol consumed per day (slightly less than one drink), researchers observed a small (7 percent) increase in the risk of breast cancer.

  The Million Women Study in the United Kingdom (which included more than 28,000 women with breast cancer) provided a more recent, and slightly higher, estimate of breast cancer risk at low to moderate levels of alcohol consumption: every 10 grams of alcohol consumed per day was associated with a 12 percent increase in the risk of breast cancer.
- **Colorectal cancer:** Alcohol consumption is associated with a modestly increased risk of cancers of the colon and rectum. A meta-analysis of 57 cohort and case-control studiesthat examined the association between alcohol consumption and colorectal cancer risk showed that people who regularly drank 50 or more grams of alcohol per day (approximately 3.5 drinks) had 1.5 times the risk of developing colorectal cancer as nondrinkers or occasional drinkers. For every 10 grams of alcohol consumed per day, there was a small (7 percent) increase in the risk of colorectal cancer.

Immune System:
Drinking too much can weaken your immune system, making your body a much easier target for disease. Chronic drinkers are more liable to contract diseases like pneumonia and tuberculosis than people who do not drink too much. Drinking a lot on a single occasion slows your body's ability to ward off infections – even up to 24 hours after getting drunk.

NIAAA: Understanding the impact of alcohol on human health and well-being

National Institute on Drug Abuse
Advancing Addiction Science

Cocaine

What is cocaine?

Photo by ©iStock.com/Rafal Cichawa

Cocaine is a powerfully addictive stimulant drug. For thousands of years, people in South America have chewed and ingested coca leaves (*Erythroxylon coca*), the source of cocaine, for their stimulant effects.[1,2] The purified chemical, cocaine hydrochloride, was isolated from the plant more than 100 years ago. In the early 1900s, purified cocaine was the main active ingredient in many tonics and elixirs developed to treat a wide variety of illnesses and was even an ingredient in the early formulations of Coca-Cola®. Before the development of synthetic local anesthetic, surgeons used cocaine to block pain.[1] However, research has since shown that cocaine is a powerfully addictive substance that can alter brain structure and function if used repeatedly.

Today, cocaine is a Schedule II drug, which means that it has high potential for abuse but can be administered by a doctor for legitimate medical uses, such as local anesthesia for some eye, ear, and throat surgeries. As a street drug, cocaine appears as a fine, white, crystalline powder and is also known as *Coke, C, Snow, Powder,* or *Blow*. Street dealers often dilute (or "cut") it with non-psychoactive substances such as cornstarch, talcum powder, flour, or baking soda to increase their profits. They may also adulterate cocaine with other drugs like procaine (a chemically related local anesthetic) or amphetamine

(another psychoactive stimulant).[2,3] Some users combine cocaine with heroin —called a *Speedball*.[2]

People abuse two chemical forms of cocaine: the water-soluble hydrochloride salt and the water-insoluble cocaine base (or freebase). Users inject or snort the hydrochloride salt, which is a powder. The base form of cocaine is created by processing the drug with ammonia or sodium bicarbonate (baking soda) and water, then heating it to remove the hydrochloride to produce a smokable substance. The term *crack*, which is the street name given to freebase cocaine, refers to the crackling sound heard when the mixture is smoked.[2]

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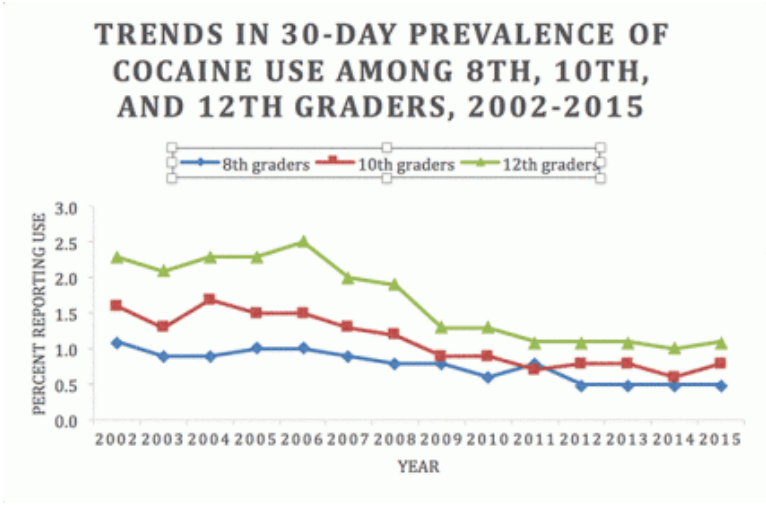
Cocaine

What is the scope of cocaine use in the United States?

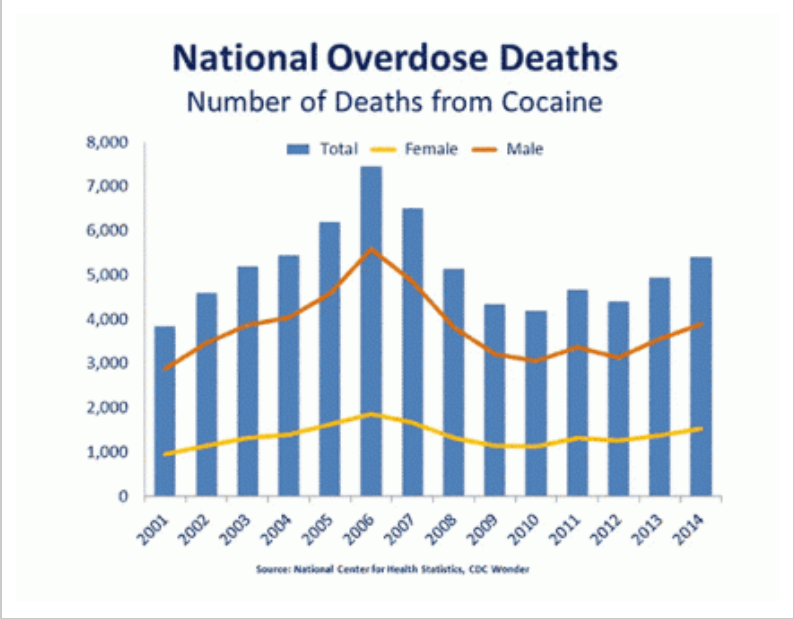
According to the National Survey on Drug Use and Health (NSDUH), cocaine use has remained relatively stable since 2009. In 2014, there were an estimated 1.5 million current (past-month) cocaine users aged 12 or older (0.6 percent of the population). Adults aged 18 to 25 years have a higher rate of current cocaine use than any other age group, with 1.4 percent of young adults reporting past-month cocaine use.[4]



Source: University of Michigan, Monitoring the Future national results on drug use: 1975-2015: overview, key findings on adolescent drug use, 2016.

The 2015 Monitoring the Future survey, which annually surveys teen attitudes and drug use, reports a significant decline in 30-day prevalence of powder cocaine use among 8th, 10th, and 12th graders from peak use in the late 1990s. In 2014, 1.1 percent of 12th graders and only 0.8 percent of 10th and half a percent of 8th graders reported using cocaine in the past month.[5]

Repeated cocaine use can produce addiction and other adverse health consequences. In 2014, according to the NSDUH, about 913,000 Americans met the *Diagnostic and Statistical Manual of Mental Disorders* criteria for dependence or abuse of cocaine (in any form) during the past 12 months. Further, data from the 2011 Drug Abuse Warning Network (DAWN) report showed that cocaine was involved in 505,224 of the nearly 1.3 million visits to emergency departments for drug misuse or abuse. This translates to over one in three drug misuse or abuse-related emergency department visits (40 percent) that involved cocaine.[6]



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How is cocaine used?

Photo by DEA/

Cocaine

Users primarily administer cocaine orally, intranasally, intravenously, or by inhalation. When people snort the drug (intranasal use), they inhale cocaine powder through the nostrils, where it is absorbed into the bloodstream through the nasal tissues. Users also may rub the drug onto their gums (oral use). Dissolving cocaine in water and injecting it (intravenous use) releases the drug directly into the bloodstream and heightens the intensity of its effects. When people smoke cocaine (inhalation), they inhale its vapor or smoke into the lungs, where absorption into the bloodstream is almost as rapid as by injection. This fast euphoric effect is one of the reasons that crack became enormously popular in the mid-1980s.[2]

Photo by DEA/

Crack cocaine

Cocaine use ranges from occasional to repeated or compulsive use, with a variety of patterns between these extremes. Any route of administration can potentially lead to absorption of toxic amounts of cocaine, causing heart attacks, strokes, or seizures—all of which can result in sudden death.[2,7]

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How does cocaine produce its effects?

The brain's *mesolimbic dopamine system*, its reward pathway, is stimulated by all types of reinforcing stimuli, such as food, sex, and many drugs of abuse, including cocaine.[8] This pathway originates in a region of the midbrain called the ventral tegmental area and extends to the nucleus accumbens, one of the brain's key reward areas.[8] Besides reward, this circuit also regulates emotions and motivation.

In the normal communication process, dopamine is released by a neuron into the synapse (the small gap between two neurons), where it binds to specialized proteins called *dopamine receptors* on the neighboring neuron. By this process, dopamine acts as a chemical messenger, carrying a signal from neuron to neuron. Another specialized protein called a *transporter* removes dopamine from the synapse to be recycled for further use.[8]

Drugs of abuse can interfere with this normal communication process. For example, cocaine acts by binding to the dopamine transporter, blocking the removal of dopamine from the synapse. Dopamine then accumulates in the synapse to produce an amplified signal to the receiving neurons. This is what causes the euphoria commonly experienced immediately after taking the drug (see the video "Brain Reward: Understanding How the Brain Responds to Natural Rewards and Drugs of Abuse").

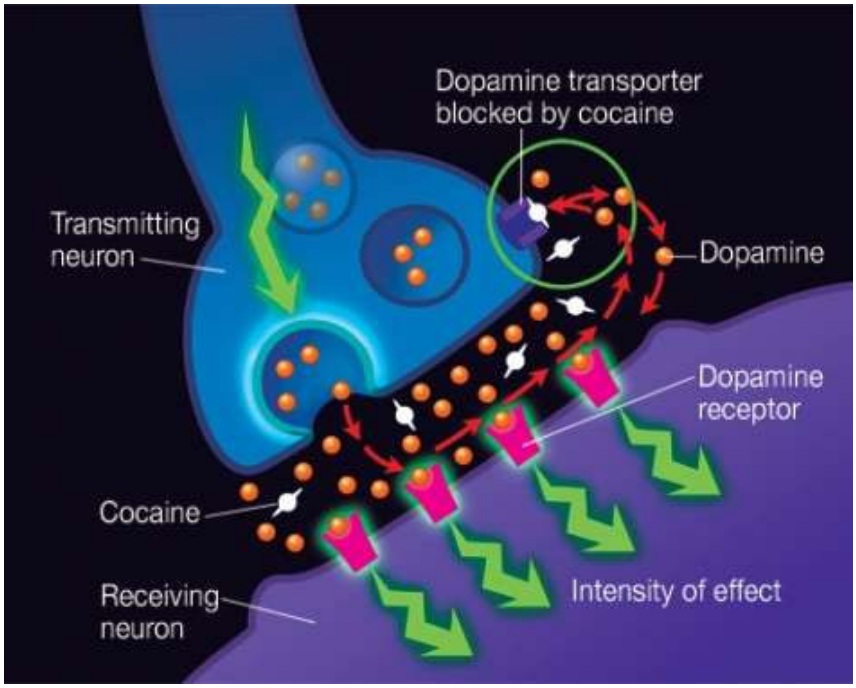

Image by NIDA

Cocaine in the brain: In the normal neural communication process, dopamine is released by a neuron into the synapse, where it can bind to dopamine receptors on neighboring neurons. Normally, dopamine is then recycled back into the transmitting neuron by a specialized protein called the dopamine transporter. If cocaine is present, it attaches to the dopamine transporter and blocks the normal recycling process, resulting in a buildup of dopamine in the synapse, which contributes to the pleasurable effects of cocaine.

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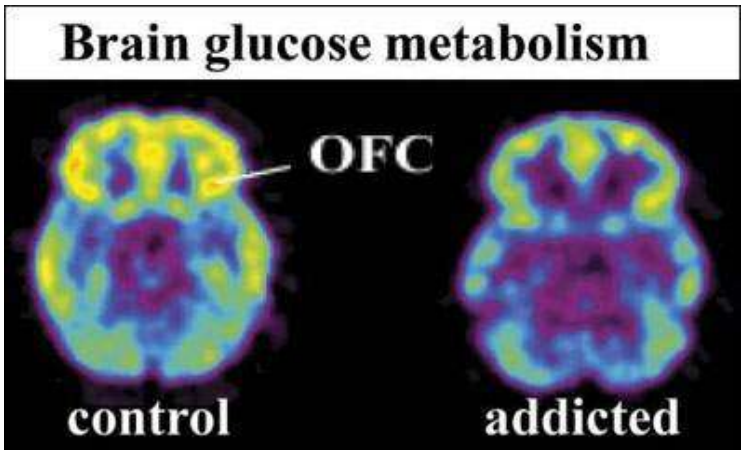
What are some ways that cocaine changes the brain?

Use of cocaine, like other drugs of abuse, induces long-term changes in the brain. Animal studies show that cocaine exposure can cause significant neuroadaptations in neurons that release the excitatory neurotransmitter glutamate.[9,10] Animals chronically exposed to cocaine demonstrate profound changes in glutamate neurotransmission—including how much is released and the level of receptor proteins—in the reward pathway, particularly the nucleus accumbens. The glutamate system may be an opportune target for anti-addiction medication development, with the goal of reversing the cocaine-induced neuroadaptations that contribute to the drive to use the drug.[9]

Although addiction researchers have focused on adaptations in the brain's reward system, drugs also affect the brain pathways that respond to stress. Stress can contribute to cocaine relapse, and cocaine use disorders frequently co-occur with stress-related disorders.[11] The stress circuits of the brain are distinct from the reward pathway, but research indicates that there are important ways that they overlap. The ventral tegmental area seems to act as a critical integration site in the brain that relays information about both stress and drug cues to other areas of the brain, including ones that drive cocaine seeking.[11] Animals that have received cocaine repeatedly are more likely to seek the drug in response to stress, and the more of the drug they have taken, the more stress affects this behavior.[11] Research suggests that cocaine elevates stress hormones, inducing neuroadaptations that further increase sensitivity to the drug and cues associated with it.[11]



Brain images showing decreased glucose metabolism, which indicates reduced activity, in the orbitofrontal cortex (OFC) in a control subject (left) and a cocaine-addicted subject (right). Volkow ND, Want G-J, Fowler JS, Tomasi D, Teland F. Addiction: beyond dopamine reward circuitry. *Proc Natl Acad Sci USA*. 2011;108(37):15037-15042.

Chronic cocaine exposure affects many other areas of the brain too. For example, animal research indicates that cocaine diminishes functioning in the orbitofrontal cortex (OFC), which appears to underlie the poor decision-making, inability to adapt to negative consequences of drug use, and lack of self-insight shown by people addicted to cocaine.[12] A study using optogenetic technology, which uses light to activate specific, genetically-modified neurons, found that stimulating the OFC restores adaptive learning in animals. This intriguing result suggests that strengthening OFC activity may be a good therapeutic approach to improve insight and awareness of the consequences of drug use among people addicted to cocaine.[13]

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What are the short-term effects of cocaine use?

Cocaine's effects appear almost immediately after a single dose and disappear within a few minutes to an hour. Small amounts of cocaine usually make the user feel euphoric, energetic, talkative, mentally alert, and hypersensitive to sight, sound, and touch. The drug can also temporarily decrease the need for food and sleep.[14] Some users find that cocaine helps them perform simple physical and intellectual tasks more quickly, although others experience the opposite effect.

The duration of cocaine's euphoric effects depend upon the route of administration. The faster the drug is absorbed, the more intense the resulting high, but also the shorter its duration. Snorting cocaine produces a relatively slow onset of the high, but it may last from 15 to 30 minutes. In contrast, the high from smoking is more immediate but may last only 5 to 10 minutes.[15]

Short-term physiological effects of cocaine use include constricted blood vessels; dilated pupils; and increased body temperature, heart rate, and blood pressure.[16] Large amounts of cocaine may intensify the user's high but can also lead to bizarre, erratic, and violent behavior. Some cocaine users report feelings of restlessness, irritability, anxiety, panic, and paranoia.[2] Users may also experience tremors, vertigo, and muscle twitches.[2]

Severe medical complications can occur with cocaine use. Some of the most frequent are cardiovascular effects, including disturbances in heart rhythm and heart attacks; neurological effects, including headaches, seizures, strokes, and coma; and gastrointestinal complications, including abdominal pain and nausea.[7] In rare instances, sudden death can occur on the first use of cocaine or unexpectedly

thereafter. Cocaine-related deaths are often a result of cardiac arrest or seizures[2] (see "National Overdose Deaths: Number of Deaths from Cocaine"). Many cocaine users also use alcohol, and this combination can be particularly dangerous. The two substances react to produce cocaethylene, which may potentiate the toxic effects of cocaine and alcohol on the heart.[17] The combination of cocaine and heroin is also very dangerous. Users combine these drugs because the stimulating effects of cocaine are offset by the sedating effects of heroin; however, this can lead to taking a high dose of heroin without initially realizing it. Because cocaine's effects wear off sooner, this can lead to a heroin overdose, in which the user's respiration dangerously slows down or stops, possibly fatally.

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What are the long-term effects of cocaine use?

With repeated exposure to cocaine, the brain starts to adapt so that the reward pathway becomes less sensitive to natural reinforcers[10,18] (see "What Are Some Ways that Cocaine Changes the Brain?"). At the same time, circuits involved in stress become increasingly sensitive, leading to increased displeasure and negative moods when not taking the drug, which are signs of withdrawal. These combined effects make the user more likely to focus on seeking the drug instead of relationships, food, or other natural rewards.

With regular use, tolerance may develop so that higher doses, more frequent use of cocaine, or both are needed to produce the same level of pleasure and relief from withdrawal experienced initially.[10,18] At the same time, users can also develop sensitization, in which less cocaine is needed to produce anxiety, convulsions, or other toxic effects.[7] Tolerance to cocaine reward and sensitization to cocaine toxicity can increase the risk of overdose in a regular user.

Users take cocaine in binges, in which cocaine is used repeatedly and at increasingly higher doses. This can lead to increased irritability, restlessness, panic attacks, paranoia, and even a full-blown psychosis, in which the individual loses touch with reality and experiences auditory hallucinations.[2] With increasing doses or higher frequency of use, the risk of adverse psychological or physiological effects increases.[2,7] Animal research suggests

that binging on cocaine during adolescence enhances sensitivity to the rewarding effects of cocaine and MDMA (Ecstasy or Molly).[19] Thus, binge use of cocaine during adolescence may further increase vulnerability to continued use of the drug among some people.

Specific routes of cocaine administration can produce their own adverse effects. Regularly snorting cocaine can lead to loss of sense of smell, nosebleeds, problems with swallowing, hoarseness, and an overall irritation of the nasal septum leading to a chronically inflamed, runny nose.[15] Smoking crack cocaine damages the lungs and can worsen asthma.[2,3] People who inject cocaine have puncture marks called tracks, most commonly in their forearms,[7] and they are at risk of contracting infectious diseases like HIV and hepatitis C (see "Why Are Cocaine Users at Risk for Contracting HIV and Hepatitis?"). They also may experience allergic reactions, either to the drug itself or to additives in street cocaine, which in severe cases can result in death.

Cocaine damages many other organs in the body. It reduces blood flow in the gastrointestinal tract, which can lead to tears and ulcerations.[7] Many chronic cocaine users lose their appetite and experience significant weight loss and malnourishment. Cocaine has significant and well-recognized toxic effects on the heart and cardiovascular system.[7,16,20] Chest pain that feels like a heart attack is common and sends many cocaine users to the emergency room.[7,20] Cocaine use is linked with increased risk of stroke,[16] as well as inflammation of the heart muscle, deterioration of the ability of the heart to contract, and aortic ruptures.[20]

In addition to the increased risk for stroke and seizures, other neurological problems can occur with long-term cocaine use.[7,18] There have been reports of *intracerebral hemorrhage*, or bleeding within the brain, and balloon-like bulges in the walls of cerebral blood vessels.[7,18] Movement disorders, including Parkinson's disease, may also occur after many years of cocaine use.[7] Generally, studies suggest that a wide range of cognitive functions are impaired

with long-term cocaine use—such as sustaining attention, impulse inhibition, memory, making decisions involving rewards or punishments, and performing motor tasks.[14]

Former cocaine users are at high risk for relapse, even following long periods of abstinence. Research indicates that during periods of abstinence, the memory of the cocaine experience or exposure to cues associated with drug use can trigger strong cravings, which can lead to relapse.[21]

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Heroin

Overview

Heroin is a highly addictive opioid drug, and its use has repercussions that extend far beyond the individual user. The medical and social consequences of drug use—such as hepatitis, HIV/AIDS, fetal effects, crime, violence, and disruptions in family, workplace, and educational environments—have a devastating impact on society and cost billions of dollars each year.

Although heroin use in the general population is rather low, the numbers of people starting to use heroin have been steadily rising since 2007.[1] This may be due in part to a shift from misuse of prescription pain relievers to heroin as a readily available, cheaper alternative[2-5] and the misperception that pure heroin is safer than less pure forms because it does not need to be injected.

Like many other chronic diseases, substance use disorders can be treated. Medications are available to treat heroin use disorder while reducing drug cravings and withdrawal symptoms, thus improving the odds of achieving abstinence. There are now a variety of medications that can be tailored to a person's recovery needs while taking into account co-occurring health conditions. Medication combined with behavioral therapy is particularly effective, offering hope to individuals who suffer from substance use disorders and for those around them.

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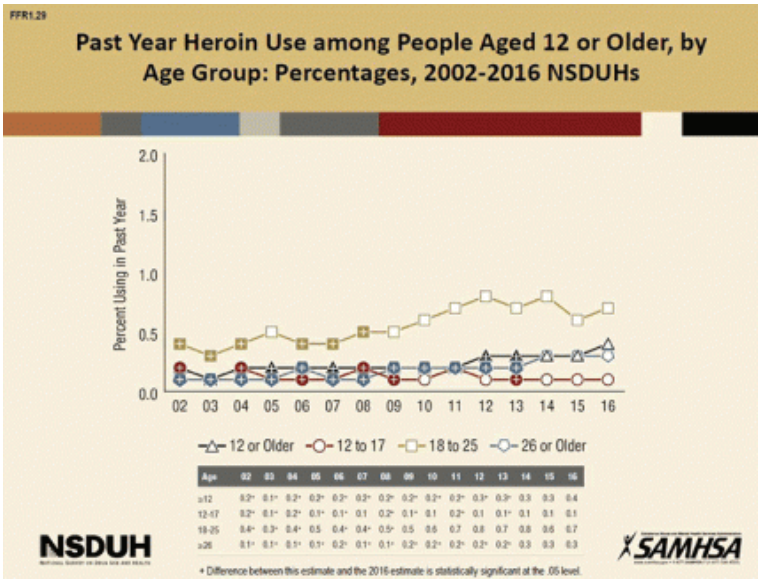
Heroin

What is the scope of heroin use in the United States?

According to the National Survey on Drug Use and Health (NSDUH), in 2016 about 948,000 Americans reported using heroin in the past year,[1] a number that has been on the rise since 2007. This trend appears to be driven largely by young adults aged 18–25 among whom there have been the greatest increases. The number of people using heroin for the first time is high, with 170,000 people starting heroin use in 2016, nearly double the number of people in 2006 (90,000). In contrast, heroin use has been declining among teens aged 12–17. Past-year heroin use among the nation's 8th, 10th, and 12th graders is at its lowest levels since 1991, at less than 1 percent in each grade level.[6]



Source: National Survey on Drug Use and Health: Summary of National Findings, 2016.

It is no surprise that with heroin use on the rise, more people are experiencing negative health effects that occur from repeated use. The number of people meeting Diagnostic

and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) criteria for dependence or heroin use disorder increased dramatically from 214,000 in 2002 to 626,000 in 2016.[1] The fifth and the current version of the DSM, DSM-5, no longer separates substance abuse from dependence, but instead provides criteria for opioid use disorder that range from mild to severe, depending on the number of symptoms a person has.[7] Data on the scope and severity of opioid use disorder in the United States are not yet available for these new criteria.

The impact of heroin use is felt all across the United States, with heroin being identified as the most or one of the most important drug use issues affecting several local regions from coast to coast. The rising harm associated with heroin use at the community level was presented in a report produced by the NIDA Community Epidemiology Work Group (CEWG). The CEWG is comprised of researchers from major metropolitan areas in the United States and selected foreign countries and provides community-level surveillance of drug use and its consequences to identify emerging trends.[3]

Fentanyl

Fentanyl is a synthetic opioid that is 50 to 100 times more powerful than morphine. Recently, traces of fentanyl have been found in many other illegal drugs, including heroin. This is a public health concern because the strength of fentanyl makes overdosing more likely.

Heroin use no longer predominates solely in urban areas. Several suburban and rural communities near Chicago and St. Louis report increasing amounts of heroin seized by officials as well as increasing numbers of overdose deaths due to heroin use. Heroin use is also on the rise in many urban areas among young adults aged 18-25.[8] Individuals in this age group seeking treatment for heroin use increased from 11 percent of total admissions in 2008 to 26 percent in the first half of 2012.

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What effects does heroin have on the body?

The greatest increase in heroin use is seen in young adults aged 18-25.

Heroin binds to and activates specific receptors in the brain called mu-opioid receptors (MORs). Our bodies contain naturally occurring chemicals called neurotransmitters that bind to these receptors throughout the brain and body to regulate pain, hormone release, and feelings of well-being.[9] When MORs are activated in the reward center of the brain, they stimulate the release of the neurotransmitter dopamine, causing a reinforcement of drug taking behavior.[10] The consequences of activating opioid receptors with externally administered opioids such as heroin (versus naturally occurring chemicals within our bodies) depend on a variety of factors: how much is used, where in the brain or body it binds, how strongly it binds and for how long, how quickly it gets there, and what happens afterward.

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What are the immediate (short-term) effects of heroin use?

Once heroin enters the brain, it is converted to morphine and binds rapidly to opioid receptors.[11] People who use heroin typically report feeling a surge of pleasurable sensation—a "rush." The intensity of the rush is a function of how much drug is taken and how rapidly the drug enters the brain and binds to the opioid receptors. With heroin, the rush is usually accompanied by a warm flushing of the skin, dry mouth, and a heavy feeling in the extremities. Nausea, vomiting, and severe itching may also occur. After the initial effects, users usually will be drowsy for several hours; mental function is clouded; heart function slows; and breathing is also severely slowed, sometimes enough to be life-threatening. Slowed breathing can also lead to coma and permanent brain damage.[12]

Opioids Act on Many Places in the Brain and Nervous System

- Opioids can depress breathing by changing neurochemical activity in the brain stem, where automatic body functions such as breathing and heart rate are controlled.
- Opioids can reinforce drug taking behavior by altering activity in the limbic system, which controls emotions.
- Opioids can block pain messages transmitted through the spinal cord from the body.

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What are the long-term effects of heroin use?

Repeated heroin use changes the physical structure[13] and physiology of the brain, creating long-term imbalances in neuronal and hormonal systems that are not easily reversed.[14,15] Studies have shown some deterioration of the brain's white matter due to heroin use, which may affect decision-making abilities, the ability to regulate behavior, and responses to stressful situations.[16-18] Heroin also produces profound degrees of tolerance and physical dependence. Tolerance occurs when more and more of the drug is required to achieve the same effects. With physical dependence, the body adapts to the presence of the drug, and withdrawal symptoms occur if use is reduced abruptly.

Withdrawal may occur within a few hours after the last time the drug is taken. Symptoms of withdrawal include restlessness, muscle and bone pain, insomnia, diarrhea, vomiting, cold flashes with goose bumps ("cold turkey"), and leg movements. Major withdrawal symptoms peak between 24–48 hours after the last dose of heroin and subside after about a week. However, some people have shown persistent withdrawal signs for many months. Finally, repeated heroin use often results in heroin use disorder—a chronic relapsing disease that goes beyond physical dependence and is characterized by uncontrollable drug-seeking, no matter the consequences.[19] Heroin is extremely addictive no matter how it is administered, although routes of administration that allow it to reach the brain the fastest (i.e., injection and smoking) increase the risk of developing heroin use disorder. Once a person has heroin use disorder, seeking and using the drug becomes their primary purpose in life.

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Kratom

Revised April 2019

What is kratom?

Kratom is a tropical tree *(Mitragyna speciosa)* native to Southeast Asia, with leaves that contain compounds that can have psychotropic (mind-altering) effects.

Kratom is not currently an illegal substance and has been easy to order on the internet. It is sometimes sold as a green powder in packets labeled "not for human consumption." It is also sometimes sold as an extract or gum.

Photo by DEA

Kratom sometimes goes by the following names:

- Biak
- Ketum
- Kakuam
- Ithang
- Thom

How do people use kratom?

Most people take kratom as a pill, capsule, or extract. Some people chew kratom leaves or brew the dried or powdered leaves as a tea. Sometimes the leaves are smoked or eaten in food.

How does kratom affect the brain?

Kratom can cause effects similar to both opioids and stimulants. Two compounds in kratom leaves, *mitragynine* and *7-α-hydroxymitragynine*, interact with opioid receptors in the brain, producing sedation, pleasure, and decreased pain, especially when users consume large amounts of the plant. Mitragynine also interacts with other receptor systems in the brain to produce

stimulant effects. When kratom is taken in small amounts, users report increased energy, sociability, and alertness instead of sedation. However, kratom can also cause uncomfortable and sometimes dangerous side effects.

What are the health effects of kratom?

Reported health effects of kratom use include:

- nausea
- itching
- sweating
- dry mouth
- constipation
- increased urination
- loss of appetite
- seizures
- hallucinations

Symptoms of psychosis have been reported in some users.

Can a person overdose on kratom?

There have been multiple reports of deaths in people who had ingested kratom, but most have involved other substances. A 2019 paper analyzing data from the National Poison Data System found that between 2011-2017 there were 11 deaths associated with kratom exposure. Nine of the 11 deaths reported in this study involved kratom plus other drugs and medicines, such as diphenhydramine (an antihistamine), alcohol, caffeine, benzodiazepines, fentanyl, and cocaine. Two deaths were reported following exposure from kratom alone with no other reported substances.* In 2017, the FDA identified at least 44 deaths related to kratom, with at least one case investigated as possible use of pure kratom. The FDA reports note that many of the kratom-associated deaths appeared to have resulted from adulterated products or taking kratom with other potent substances, including illicit drugs, opioids, benzodiazepines, alcohol, gabapentin, and over-the-counter medications, such as cough syrup. Also, there have been some reports of kratom packaged as dietary supplements or dietary ingredients that were laced with other compounds that caused deaths. People should check with their health care providers about the safety of mixing kratom with other medicines.

*(Post et al, 2019. *Clinical Toxicology*).

Is kratom addictive?

Like other drugs with opioid-like effects, kratom might cause dependence, which means users will feel physical withdrawal symptoms when they stop taking the drug. Some users have reported becoming addicted to kratom. Withdrawal symptoms include:

- muscle aches
- insomnia
- irritability
- hostility
- aggression
- emotional changes
- runny nose
- jerky movements

How is kratom addiction treated?

There are no specific medical treatments for kratom addiction. Some people seeking treatment have found behavioral therapy to be helpful. Scientists need more research to determine how effective this treatment option is.

Does kratom have value as a medicine?

In recent years, some people have used kratom as an herbal alternative to medical treatment in attempts to control withdrawal symptoms and cravings caused by addiction to opioids or to other addictive substances such as alcohol. There is no scientific evidence that kratom is effective or safe for this purpose; further research is needed.

Points to Remember

- Kratom is a tropical tree native to Southeast Asia, with leaves that can have psychotropic effects.
- Kratom is not currently illegal and has been easy to order on the internet.
- Most people take kratom as a pill or capsule. Some people chew kratom leaves or brew

the dried or powdered leaves as a tea. Sometimes the leaves are smoked or eaten in food. Two compounds in kratom leaves, mitragynine and *7-α-hydroxymitragynine*, interact with opioid receptors in the brain, producing sedation, pleasure, and decreased pain.

- Mitragynine can also interact with other receptor systems in the brain to produce stimulant effects.
- Reported health effects of kratom use include nausea, sweating, seizures, and psychotic symptoms.
- Commercial forms of kratom are sometimes laced with other compounds that have caused deaths.
- Some users have reported becoming addicted to kratom.
- Behavioral therapies and medications have not specifically been tested for treatment of kratom addiction.

Learn More

For more information about kratom, visit:

- Commonly Abused Drug Charts
- Drug Enforcement Administration (DEA)

This page was last updated April 2019

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Marijuana

Letter From the Director

Changes in marijuana policies across states legalizing marijuana for medical and/or recreational use suggest that marijuana is gaining greater acceptance in our society. Thus, it is particularly important for people to understand what is known about both the adverse health effects and the potential therapeutic benefits linked to marijuana.

Photo by the NIDA

Because marijuana impairs short-term memory and judgment and distorts perception, it can impair performance in school or at work and make it dangerous to drive. It also affects brain systems that are still maturing through young adulthood, so regular use by teens may have negative and long-lasting effects on their cognitive development, putting them at a competitive disadvantage and possibly interfering with their well-being in other ways. Also, contrary to popular belief, marijuana can be addictive, and its use during adolescence may make other forms of problem use or addiction more likely.

Whether smoking or otherwise consuming marijuana has therapeutic benefits that outweigh its health risks is still an open question that science has not resolved. Although many states now permit dispensing marijuana for medicinal purposes and there is mounting anecdotal evidence for the efficacy of

marijuana-derived compounds, the U.S. Food and Drug Administration has not approved "medical marijuana." However, safe medicines based on cannabinoid chemicals derived from the marijuana plant have been available for decades and more are being developed.

This Research Report is intended as a useful summary of what the most up-to-date science has to say about marijuana and its effects on those who use it at any age.

Nora D. Volkow, M.D.
Director
National Institute on Drug Abuse

See Also:

- Message from the NIDA Director - Marijuana's Lasting Effects on the Brain, (Archives) (March 2013)

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Marijuana

What is marijuana?

Image by ©iStock.com/nicoolay

Marijuana—also called *weed, herb, pot, grass, bud, ganja, Mary Jane*, and a vast number of other slang terms—is a greenish-gray mixture of the dried flowers of *Cannabis sativa*. Some people smoke marijuana in hand-rolled cigarettes called *joints*; in pipes, water pipes (sometimes called *bongs*), or in *blunts* (marijuana rolled in cigar wraps).[1] Marijuana can also be used to brew tea and, particularly when it is sold or consumed for medicinal purposes, is frequently mixed into foods (*edibles*) such as brownies, cookies, or candies. Vaporizers are also increasingly used to consume marijuana. Stronger forms of marijuana include sinsemilla (from specially tended female plants) and concentrated resins containing high doses of marijuana's active ingredients, including honeylike *hash oil*, waxy *budder*, and hard amberlike *shatter*. These resins are increasingly popular among those who use them both recreationally and medically.

The main *psychoactive* (mind-altering) chemical in marijuana, responsible for most of the intoxicating effects that people seek, is *delta-9-tetrahydrocannabinol* (THC). The chemical is found in resin produced by the leaves and buds primarily of the female cannabis plant. The plant also contains more than 500 other chemicals, including more than 100 compounds that are chemically related to THC, called *cannabinoids*.[2]

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Marijuana

What is the scope of marijuana use in the United States?

Marijuana is the most commonly used psychotropic drug in the United States, after alcohol. In 2018, more than 11.8 million young adults reported marijuana use in the past year.[3] Its use is more prevalent among men than women.[4]

Marijuana use is widespread among adolescents and young adults. According to the Monitoring the Future survey—an annual survey of drug use and attitudes among the Nation's middle and high school students—most measures of marijuana use by 8th, 10th, and 12th graders peaked in the mid-to-late 1990s and then began a period of gradual decline through the mid-2000s before levelling off. However, in 2019, there was a significant increase in daily use in the younger grades. In addition, teens' perceptions of the risks of marijuana use have steadily declined over the past decade. In 2019, 11.8 percent of 8th graders reported marijuana use in the past year and 6.6 percent in the past month (current use). Among 10th graders, 28.8 percent had used marijuana in the past year and 18.4 percent in the past month. Rates of use among 12th graders were higher still: 35.7 percent had used marijuana during the year prior to the survey and 22.3 percent used in the past month; 6.4 percent said they used marijuana daily or near-daily.[5] With the growing popularity of vaping devices, teens have started vaping THC (the ingredient in marijuana that produces the high), with nearly 4% of 12th graders saying they vape THC daily.[5]

Medical emergencies possibly related to marijuana use have also increased. The Drug Abuse Warning Network (DAWN), a system for monitoring the health impact of drugs, estimated that in 2011, there were nearly 456,000 drug-related emergency department visits in the United States in which marijuana use was mentioned in the medical record (a 21 percent increase over 2009). About two-thirds of patients were male and 13 percent were between the ages of 12 and 17.[6] It is unknown whether this increase is due to increased use, increased *potency* of marijuana (amount of THC it contains), or other factors. It should be noted, however, that mentions of marijuana in medical records do not necessarily indicate that these emergencies were directly related to marijuana intoxication.

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Marijuana

What are marijuana's effects?

When marijuana is smoked, THC and other chemicals in the plant pass from the lungs into the bloodstream, which rapidly carries them throughout the body to the brain. The person begins to experience effects almost immediately (see "How does marijuana produce its effects?"). Many people experience a pleasant euphoria and sense of relaxation. Other common effects, which may vary dramatically among different people, include heightened sensory perception (e.g., brighter colors), laughter, altered perception of time, and increased appetite.

If marijuana is consumed in foods or beverages, these effects are somewhat delayed—usually appearing after 30 minutes to 1 hour—because the drug must first pass through the digestive system. Eating or drinking marijuana delivers significantly less THC into the bloodstream than smoking an equivalent amount of the plant. Because of the delayed effects, people may inadvertently consume more THC than they intend to.

Pleasant experiences with marijuana are by no means universal. Instead of relaxation and euphoria, some people experience anxiety, fear, distrust, or panic. These effects are more common when a person takes too much, the marijuana has an unexpectedly high potency, or the person is inexperienced. People who have taken large doses of marijuana may experience an acute psychosis, which includes hallucinations, delusions, and a loss of the sense of personal identity. These unpleasant but temporary reactions are distinct from longer-lasting psychotic disorders, such as schizophrenia, that may be associated with the use of marijuana in vulnerable individuals. (See "Is there a link between marijuana use and psychiatric disorders?")

Although detectable amounts of THC may remain in the body for days or even weeks after use, the noticeable effects of smoked marijuana generally last from 1 to 3 hours, and those of marijuana consumed in food or drink may last for many hours.

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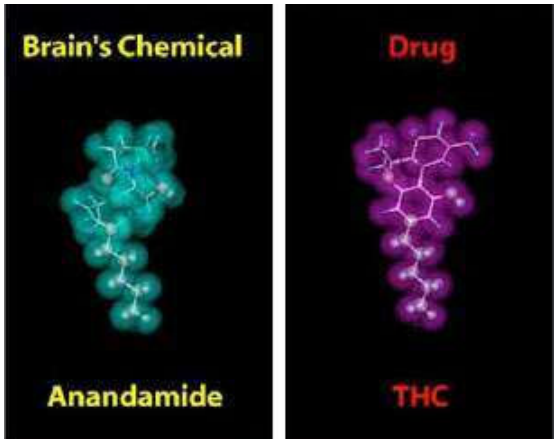
Marijuana

How does marijuana produce its effects?



Courtesy of NIDA

THC's chemical structure is similar to the brain chemical anandamide. Similarity in structure allows drugs to be recognized by the body and to alter normal brain communication.

THC's chemical structure is similar to the brain chemical *anandamide*. Similarity in structure allows the body to recognize THC and to alter normal brain communication.

Endogenous cannabinoids such as anandamide (see figure) function as *neurotransmitters* because they send chemical messages between nerve cells (*neurons*) throughout the nervous system. They affect brain areas that influence pleasure, memory, thinking, concentration, movement, coordination, and sensory and time perception. Because of this similarity, THC is able to attach to molecules called *cannabinoid receptors* on neurons in these brain areas and activate them, disrupting various mental and physical functions and causing the effects described earlier. The neural communication network that uses these cannabinoid neurotransmitters, known as the *endocannabinoid system*, plays a critical role in the nervous system's normal functioning, so interfering with it can have profound effects.

For example, THC is able to alter the functioning of the hippocampus (see "Marijuana, Memory, and the Hippocampus") and orbitofrontal cortex, brain areas that enable a person to form new memories and shift his or her attentional focus. As a result, using marijuana causes impaired thinking and interferes with a person's ability to learn and perform complicated tasks. THC also disrupts functioning of the cerebellum and basal ganglia, brain areas that regulate balance, posture, coordination, and reaction time. This is the reason people who have used marijuana may not be able to drive safely (see

"Does marijuana use affect driving?") and may have problems playing sports or engaging in other physical activities.

People who have taken large doses of the drug may experience an acute psychosis, which includes hallucinations, delusions, and a loss of the sense of personal identity.

THC, acting through cannabinoid receptors, also activates the brain's reward system, which includes regions that govern the response to healthy pleasurable behaviors such as sex and eating. Like most other drugs that people misuse, THC stimulates neurons in the reward system to release the signaling chemical *dopamine* at levels higher than typically observed in response to natural stimuli. This flood of dopamine contributes to the pleasurable "high" that those who use recreational marijuana seek.

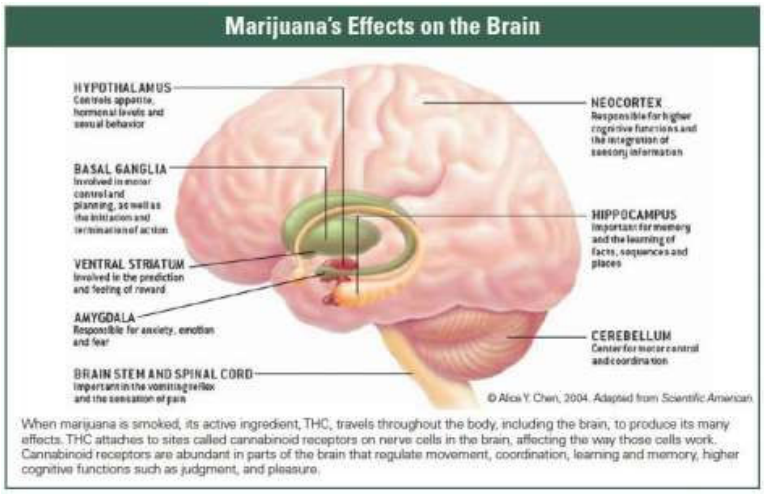


Diagram showing different parts of the brain and describing marijuana's effects on the brain

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Marijuana

Is marijuana addictive?

Marijuana use can lead to the development of problem use, known as a marijuana use disorder, which takes the form of addiction in severe cases. Recent data suggest that 30 percent of those who use marijuana may have some degree of marijuana use disorder.[18] People who begin using marijuana before the age of 18 are four to seven times more likely to develop a marijuana use disorder than adults.[19]

Marijuana use disorders are often associated with *dependence*—in which a person feels withdrawal symptoms when not taking the drug. People who use marijuana frequently often report irritability, mood and sleep difficulties, decreased appetite, cravings, restlessness, and/or various forms of physical discomfort that peak within the first week after quitting and last up to 2 weeks.[20,21] Marijuana dependence occurs when the brain adapts to large amounts of the drug by reducing production of and sensitivity to its own endocannabinoid neurotransmitters.[22,23]

Marijuana use disorder becomes addiction when the person cannot stop using the drug even though it interferes with many aspects of his or her life. Estimates of the number of people addicted to marijuana are controversial, in part because epidemiological studies of substance use often use dependence as a proxy for addiction even though it is possible to be dependent without being addicted. Those studies suggest that 9 percent of people who use marijuana will become dependent on it,[24,25] rising to about 17 percent in those who start using in their teens.[26,27]

In 2015, about 4.0 million people in the United States met the diagnostic criteria for a marijuana use disorder;[3] 138,000 voluntarily sought treatment for their marijuana use.[28]

Rising Potency

Marijuana potency, as detected in confiscated samples, has steadily increased over the past few decades.[2] In the early 1990s, the average THC content in confiscated marijuana samples was roughly 3.8 percent. In 2014, it was 12.2 percent. The average marijuana extract contains more than 50 percent THC, with some samples exceeding 80 percent. These trends raise concerns that the consequences of marijuana use could be worse than in the past, particularly among those who are new to marijuana use or in young people, whose brains are still developing (see "What are marijuana's long-term effects on the brain?").

Researchers do not yet know the full extent of the consequences when the body and brain (especially the developing brain) are exposed to high concentrations of THC or whether the recent increases in emergency department visits by people testing positive for marijuana are related to rising potency. The extent to which people adjust for increased potency by using less or by smoking it differently is also unknown. Recent studies suggest that experienced people may adjust the amount they smoke and how much they inhale based on the believed strength of the marijuana they are using, but they are not able to fully compensate for variations in potency.[30,31]

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Methamphetamine

Overview

The misuse of methamphetamine—a potent and highly addictive stimulant—remains an extremely serious problem in the United States. In some areas of the country, it poses an even greater threat than opioids, and it is the drug that most contributes to violent crime.[36] According to data from the 2017 National Survey on Drug Use and Health (NSDUH), over 14.7 million people (5.4 percent of the population) have tried methamphetamine at least once. NSDUH also reports that almost 1.6 million people used methamphetamine in the year leading up to the survey,[1] and it remains one of the most commonly misused stimulant drugs in the world.[37]

The consequences of methamphetamine misuse are terrible for the individual—psychologically, medically, and socially. Using the drug can cause memory loss, aggression, psychotic behavior, damage to the cardiovascular system, malnutrition, and severe dental problems. Methamphetamine misuse has also been shown to contribute to increased transmission of infectious diseases, such as hepatitis and HIV/AIDS.

Beyond its devastating effects on individual health, methamphetamine misuse threatens whole communities, causing new waves of crime, unemployment, child neglect or abuse, and other social ills. A 2009 report from the RAND Corporation noted that methamphetamine misuse cost the nation approximately $23.4 billion in 2005.[1]

But the good news is that methamphetamine misuse can be prevented and addiction to the drug can be treated with behavioral therapies. Research also continues toward development of new pharmacological and other treatments for methamphetamine use, including medications, vaccines, and noninvasive stimulation of the brain using magnetic fields. People can and do recover from methamphetamine addiction if they have ready access to effective treatments that address the multitude of medical and personal problems resulting from their long-term use of the drug.

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Methamphetamine

What is the scope of methamphetamine misuse in the United States?

According to the 2017 National Survey on Drug Use and Health (NSDUH), approximately 1.6 million people (0.6 percent of the population) reported using methamphetamine in the past year, and 774,000 (0.3 percent) reported using it in the past month. The average age of new methamphetamine users in 2016 was 23.3 years old.[2]

An estimated 964,000 people aged 12 or older (about 0.4 percent of the population) had a methamphetamine use disorder in 2017—that is, they reported clinically significant impairment, including health problems, disability, and failure to meet responsibilities at work, school, or home as a result of their drug use. This number is significantly higher than the 684,000 people who reported having methamphetamine use disorder in 2016.

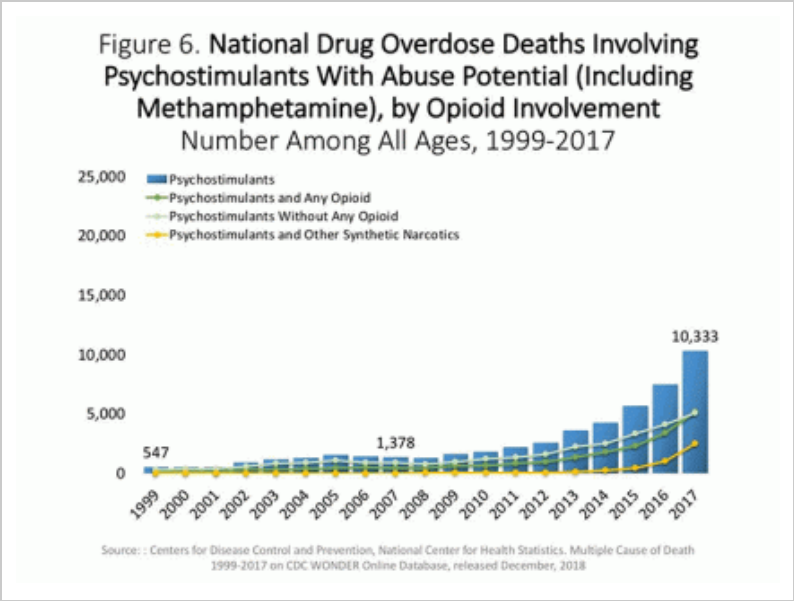
The 2018 Monitoring the Future (MTF) survey of adolescent drug use and attitudes reported that about 0.5 percent of 8th, 10th, and 12th graders had used methamphetamine within the past year. Use of methamphetamine by adolescents has declined significantly since 1999, when this drug was first added to the survey.[3]

The Treatment Episode Data Set (TEDS) provides information on admissions to substance abuse treatment facilities that are licensed or certified by state substance use agencies. According to TEDS data, nationwide treatment admissions for methamphetamine misuse dropped from 68 per 100,000 individuals in 2005 to 49 per 100,000 in 2015.[39]

An important caveat to these national numbers is the degree to which they mask regional variability. While methamphetamine is available across the US, highest

availability is in the western and midwestern regions of the US; more than 70 percent of local law enforcement agencies from the pacific and west central regions of the US report methamphetamine as the greatest drug threat in their area.[41]

NIDA's National Drug Early Warning System (NDEWS), which tracks drug trends in sentinel sites across the country, found that treatment admissions for methamphetamine as the primary substance of use were less than one percent in sites east of the Mississippi River, but ranged from 12-29 percent in the sites west of the Mississippi.[41] Nationwide, overdose deaths from the category of drugs that includes methamphetamine increased by 7.5 times between 2007 and 2017. About 15 percent of all drug overdose deaths involved the methamphetamine category in 2017, and 50 percent of those deaths also involved an opioid.[42] In 2017, 5 of the 12 NDEWS sites reported increases in methamphetamine overdose deaths: Washington, Colorado, Texas, Florida, and Georgia.[41]



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Methamphetamine

How is methamphetamine misused?

Methamphetamine comes in several forms and can be smoked, snorted, injected, or orally ingested. The preferred method of using the drug varies by geographical region and has changed over time.[37,38,43]

Smoking or injecting methamphetamine puts the drug very quickly into the bloodstream and brain, causing an immediate, intense "rush" and amplifying the drug's addiction potential and adverse health consequences. The rush, or "flash," lasts only a few minutes and is described as extremely pleasurable. Snorting or oral ingestion produces euphoria—a high, but not an intense rush. Snorting produces effects within 3 to 5 minutes, and oral ingestion produces effects within 15 to 20 minutes.[4]

As with many stimulants, methamphetamine is most often misused in a "binge and crash" pattern. Because the pleasurable effects of methamphetamine disappear even before the drug concentration in the blood falls significantly, users try to maintain the high by taking more of the drug. In some cases, people indulge in a form of binging known as a "run," foregoing food and sleep while continuing to take the drug for up to several days.[37,38,43]

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Methamphetamine

How is methamphetamine manufactured?

Currently, most methamphetamine in the United States is produced by transnational criminal organizations (TCOs) in Mexico.[44] This methamphetamine is highly pure, potent, and low in price. The drug can be easily made in small clandestine laboratories, with relatively inexpensive over-the-counter ingredients such as pseudoephedrine, a common ingredient in cold medications.

To curb production of methamphetamine, Congress passed the Combat Methamphetamine Epidemic Act in 2005, which requires that pharmacies and other retail stores keep logs of purchases of products containing pseudoephedrine and limits the amount of those products an individual can purchase per day. Restrictions on the chemicals used to make methamphetamine in the United States have dramatically reduced domestic production of the drug. In 2010, there were 15,256 domestic methamphetamine laboratory incidents—a figure that has fallen over 80 percent to 3,036 in 2017.[44] Data on drug seizures indicate that most domestic production of methamphetamine is now conducted in small laboratories that make two ounces or less of the drug using common household items.[44]

Mexico has also tightened its restrictions on pseudoephedrine and other methamphetamine precursor chemicals. But manufacturers adapt to these restrictions via small- or large-scale "smurfing" operations: obtaining pseudoephedrine from multiple sources, below the legal thresholds, using

multiple false identifications. Manufacturers in Mexico are also increasingly using a different production process (called P2P which stands for pseudoephedrine's precursor chemical, phenyl-2-propanone) to make methamphetamine that does not require pseudoephedrine.

When methamphetamine is smuggled into the United States in powder or liquid form, domestic conversion laboratories transform it into crystal methamphetamine. These laboratories do not require a significant amount of equipment, so they can be small in size and thus easily concealed, which presents challenges to law enforcement agencies.[44] Methamphetamine pressed into a pill form intended to resemble ecstasy has also recently emerged, potentially in an effort to make methamphetamine more appealing to people who haven't tried it before.[44] As with other illicit drugs like heroin and cocaine, methamphetamine is also sometimes laced with fentanyl.[44]

Methamphetamine production is also an environmental concern; it involves many easily obtained chemicals that are hazardous, such as acetone, anhydrous ammonia (fertilizer), ether, red phosphorus, and lithium. Toxicity from these chemicals can remain in the environment around a methamphetamine production lab long after the lab has been shut down, causing a wide range of damaging effects to health. Because of these dangers, the U.S. Environmental Protection Agency has provided guidance on cleanup and remediation of methamphetamine labs.

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Methamphetamine

How is methamphetamine different from other stimulants, such as cocaine?

The methamphetamine molecule is structurally similar to amphetamine and to the neurotransmitter dopamine, a brain chemical that plays an important role in the reinforcement of rewarding behaviors, but it is quite different from cocaine.[45] Although these stimulants have similar behavioral and physiological effects, there are some major differences in the basic mechanisms of how they work.

In contrast to cocaine, which is quickly removed from and almost completely metabolized in the body, methamphetamine has a much longer duration of action, and a larger percentage of the drug remains unchanged in the body. Methamphetamine therefore remains in the brain longer, which ultimately leads to prolonged stimulant effects.[46]

Although both methamphetamine and cocaine increase levels of dopamine, administration of methamphetamine in animal studies leads to much higher levels of dopamine, because nerve cells respond differently to the two drugs. Cocaine prolongs dopamine actions in the brain by blocking the re-absorption (re-uptake) of the neurotransmitter by signaling nerve cells. At low doses, methamphetamine also blocks the re-uptake of dopamine, but it also increases the release of dopamine, leading to much higher concentrations in the synapse (the gap between neurons), which can be toxic to nerve terminals.[38,39]

Methamphetamine versus Cocaine

Methamphetamine	Cocaine
Stimulant	Stimulant and local anesthetic
Man-made	Plant-derived
Smoking produces a long-lasting high	Smoking produces a brief high
50% of the drug is removed from the body in 12 hours	50% of the drug is removed from the body in 1 hour
Increases dopamine release and blocks dopamine re-uptake	Blocks dopamine re-uptake
Limited medical use for ADHD, narcolepsy, and weight loss	Limited medical use as a local anesthetic in some surgical procedures

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What are the immediate (short-term) effects of methamphetamine misuse?

As a powerful stimulant, methamphetamine, even in small doses, can increase wakefulness and physical activity and decrease appetite. Methamphetamine can also cause a variety of cardiovascular problems, including rapid heart rate, irregular heartbeat, and increased blood pressure. Hyperthermia (elevated body temperature) and convulsions may occur with methamphetamine overdose, and if not treated immediately, can result in death.[37,38]

The exact mechanisms whereby drugs like methamphetamine produce euphoria (the pleasurable high) are still poorly understood. But along with euphoria, methamphetamine use releases very high levels of the neurotransmitter dopamine in the reward circuit, which "teaches" the brain to repeat the pleasurable activity of taking the drug. Dopamine is involved in motivation and motor function and its release in the reward circuit is a defining feature of addictive drugs. The elevated release of dopamine produced by methamphetamine is also thought to contribute to the drug's deleterious effects on nerve terminals in the brain.

Short-term effects may include:

- increased attention and decreased fatigue
- increased activity and wakefulness
- decreased appetite
- euphoria and rush
- increased respiration
- rapid/irregular heartbeat
- hyperthermia

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Methamphetamine

What are the long-term effects of methamphetamine misuse?

Long-term methamphetamine abuse has many negative consequences, including addiction. Addiction is a chronic, relapsing disease, characterized by compulsive drug seeking and use and accompanied by functional and molecular changes in the brain.

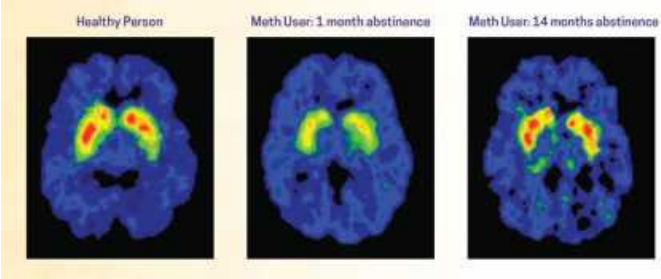
As is the case with many drugs, tolerance to methamphetamine's pleasurable effects develops when it is taken repeatedly. Abusers often need to take higher doses of the drug, take it more frequently, or change how they take it in an effort to get the desired effect. Chronic methamphetamine abusers may develop difficulty feeling any pleasure other than that provided by the drug, fueling further abuse. Withdrawal from methamphetamine occurs when a chronic abuser stops taking the drug; symptoms of withdrawal include depression, anxiety, fatigue, and an intense craving for the drug.[43]

In addition to being addicted to methamphetamine, chronic abusers may exhibit symptoms that can include significant anxiety, confusion, insomnia, mood disturbances, and violent behavior.[47] They also may display a number of psychotic features, including paranoia, visual and auditory hallucinations, and delusions (for example, the sensation of insects creeping under the skin).[48] Psychotic symptoms can sometimes last for months or years after a person has quit abusing methamphetamine, and stress has been shown to precipitate spontaneous recurrence of methamphetamine psychosis in formerly psychotic methamphetamine abusers.[49]

These and other problems reflect significant changes in the brain caused by misuse of methamphetamine. Neuroimaging studies have demonstrated alterations in the activity of the dopamine system that are associated with reduced motor speed and impaired verbal learning.[6,7,8] Studies in chronic methamphetamine users have also revealed severe structural and functional changes in areas of the brain associated with emotion and memory, which may account for many of the emotional and cognitive problems observed in these individuals.[9,10,11]

Research in primate models has found that methamphetamine alters brain structures involved in decision-making and impairs the ability to suppress habitual behaviors that have become useless or

counterproductive. The two effects were correlated, suggesting that the structural change underlies the decline in mental flexibility.[12] These changes in brain structure and function could explain why methamphetamine addiction is so hard to treat and has a significant chance of relapse early in treatment.



Recovery of Brain Dopamine Transporters in Chronic Methamphetamine (METH) Users
Methamphetamine misuse greatly reduces the binding of dopamine to dopamine transporters (highlighted in red and green) in the striatum, a brain area important in memory and movement. With prolonged abstinence, dopamine transporters in this area can be restored.

Methamphetamine misuse also has been shown to have negative effects on non-neural brain cells called microglia. These cells support brain health by defending the brain against infectious agents and removing damaged neurons. Too much activity of the microglial cells, however, can assault healthy neurons. A study using brain imaging found more than double the levels of microglial cells in people who previously misused methamphetamine compared to people with no history of methamphetamine misuse, which could explain some of the neurotoxic effects of methamphetamine.[13]

Some of the neurobiological effects of chronic methamphetamine misuse appear to be, at least, partially reversible. In the study just mentioned, abstinence from methamphetamine resulted in less excess microglial activation over time, and users who had remained methamphetamine-free for 2 years exhibited microglial activation levels similar to the study's control subjects.[14] A similar study found that while biochemical markers for nerve damage and viability persist in the brain through 6 months of abstinence from methamphetamine, those markers return to normal after a year or more without taking the drug.[15] Another neuroimaging study showed neuronal recovery in some brain regions following prolonged abstinence (14 but not 6 months).[16] This recovery was associated with improved performance on motor and verbal memory tests. Function in other brain regions did not recover even after 14 months of abstinence, indicating that some methamphetamine-induced changes are very long lasting. Methamphetamine use can also increase one's risk of stroke, which can cause irreversible damage to the brain. A recent study even showed higher incidence of Parkinson's disease among past users of methamphetamine.[17]

In addition to the neurological and behavioral consequences of methamphetamine misuse, long-term users also suffer physical effects, including weight loss, severe tooth decay and tooth loss ("meth mouth"), and skin sores.[38] The dental problems may be caused by a combination of poor nutrition and dental hygiene as well as dry mouth and teeth grinding caused by the drug. Skin sores are the result of picking and scratching the skin to get rid of insects imagined to be crawling under it.[38]

Long-term effects may include:

- addiction
- psychosis, including:
  - paranoia
  - hallucinations
  - repetitive motor activity
- changes in brain structure and function
- deficits in thinking and motor skills
- increased distractibility
- memory loss
- aggressive or violent behavior
- mood disturbances
- severe dental problems
- weight loss

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Opioids

Brief Description

Opioids are a class of drugs that include the illegal drug heroin, synthetic opioids such as fentanyl, and pain relievers available legally by prescription, such as oxycodone (OxyContin®), hydrocodone (Vicodin®), codeine, morphine, and many others.

Monitoring the Future Study: Trends in Prevalence of Various Drugs for 8th Graders, 10th Graders, and 12th Graders; 2019 (in percent)*

Drug	Time Period	8th Graders	10th Graders	12th Graders
Heroin	Past Year	0.30	0.30	0.40
Narcotics other than Heroin	Past Year	-	-	[2.70]
OxyContin	Past Year	1.20	2.00	1.70
Vicodin	Past Year	0.90	1.10	[1.10]

* Data in brackets indicate statistically significant change from the previous year. Previous MTF Data

National Survey on Drug Use and Health: Trends in Prevalence of Various Drugs for Ages 12 or Older, Ages 12 to 17, Ages 18 to 25, and Ages 26 or Older; 2018 (in percent)*

Drug	Time Period	Ages 12 or Older	Ages 12 to 17	Ages 18 to 25	Ages 26 or Older
Heroin	Past Year	0.30	0.00	0.50	0.30
Pain Relievers	Past Year	3.60	2.80	5.50	3.40

^ indicate low precision; no estimate reported.
Data in brackets indicate statistically significant change from the previous year. Previous NSDUH Data

Summary of the Issue

All opioids are chemically related and interact with opioid receptors on nerve cells in the body and brain. Opioid pain relievers are generally safe when taken for a short time and as prescribed by a doctor, but because they produce euphoria in addition to pain relief, they can be misused (taken in a different way or in a larger quantity than prescribed, or taken without a doctor's prescription). Regular use—even as prescribed by a doctor—can lead to dependence and, when misused, opioid pain relievers can lead to addiction, overdose incidents, and deaths.

An opioid overdose can be reversed with the drug naloxone when given right away. Improvements have been seen in some regions of the country in the form of decreasing availability of prescription opioid pain relievers and decreasing misuse among the Nation's teens. However, since 2007, overdose deaths related to heroin have been increasing. Fortunately, effective medications exist to treat opioid use disorders including methadone, buprenorphine, and naltrexone.

A NIDA study found that once treatment is initiated, both a buprenorphine/naloxone combination and an extended release naltrexone formulation are similarly effective in treating opioid addiction. However, naltrexone requires full detoxification, so initiating treatment among active users was more difficult. These medications help many people recover from opioid addiction.

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Tobacco, Nicotine, and E-Cigarettes

Introduction

In 2014, the Nation marked the 50th anniversary of the first Surgeon General's Report on Smoking and Health. In 1964, more than 40 percent of the adult population smoked. Once the link between smoking and its medical consequences—including cancers and heart and lung diseases—became a part of the public consciousness, education efforts and public policy changes were enacted to reduce the number of people who smoke. These efforts resulted in substantial declines in smoking rates in the United States—to half the 1964 level.[1]

However, rates of cigarette smoking and other tobacco use are still too high,[2] and some populations are disproportionately affected by tobacco's health consequences. Most notably, people with mental disorders—including substance use disorders—smoke at higher rates than the general population.[3–6] Additionally, people living below the poverty line and those with low educational attainment are more likely to smoke than those in the general population. As tobacco use is the leading preventable cause of mortality in the United States,[1] differential rates of smoking and use of other tobacco products is a significant contributor to health disparities among some of the most vulnerable people in our society.

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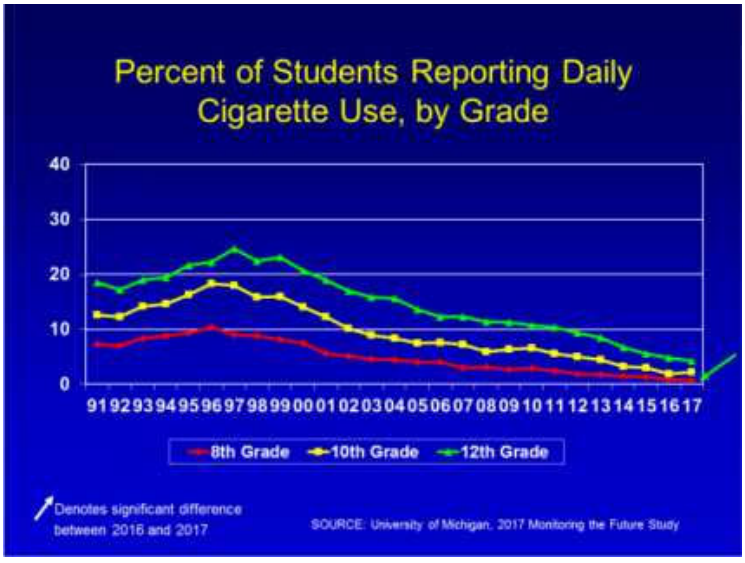
Tobacco, Nicotine, and E-Cigarettes

What is the scope of tobacco use and its cost to society?

Approximately one fourth of the population uses tobacco products, and 19.4 percent smoke cigarettes. According to the 2016 National Survey on Drug Use and Health (NSDUH), an estimated 63.4 million people aged 12 or older used a tobacco product during the past month, including 51.3 million cigarette smokers.[7] Smoking rates continue to go down year to year; the percentage of people over age 18 who smoke cigarettes declined from 20.9 percent in 2005 to 15.8 percent in 2016, according to the 2017 National Health Interview Survey.[8]



However, smoking rates are substantially higher among some of the most vulnerable people in our society. The 25 percent of Americans with mental disorders, including addiction, account for 40 percent of the cigarettes smoked in the U.S.[9] (see "Do people with mental illness and substance use disorders use tobacco more often?"). More than 40 percent of people with a General Education Development certificate (GED) smoke—which is the highest prevalence of any socioeconomic group.[10] Also, people who live in rural areas,

particularly in the South Atlantic states, use all forms of tobacco at higher rates than people who live in urban areas. These differences cannot be fully explained by different levels of poverty or affluence.[11]

Smoking among youth is at historically low levels. According to the NIDA-sponsored Monitoring the Future (MTF) survey,[12] in 2015, an estimated 4.7 million middle and high school students used tobacco products during the past month, according to data from the National Youth Tobacco Survey (NYTS)[13] e-cigarettes) were the most commonly used tobacco products among middle (5.3 percent) and high school (16.0 percent) students in 2015.[13] E-cigarettes deliver synthetic nicotine and do not contain tobacco; however, they are classified as tobacco products for regulatory purposes. These findings are echoed by other studies,[14–17] including the MTF survey.[12] Scientists have not yet determined the medical consequences of long-term e-cigarette use or the secondhand effects of e-cigarette vapor (see "What are electronic cigarettes?").

Between 1964 and 2012, an estimated 17.7 million deaths were related to smoking[18] leads to more than 480,000 deaths annually.[1] If current smoking rates continue, 5.6 million Americans who are currently younger than 18 will die prematurely from smoking-related disease.[13]

In addition to the tremendous impact of premature deaths related to tobacco use, the economic costs are high. Experts estimate that between 2009 and 2012, the annual societal costs attributable to smoking in the United States were between $289 and $332.5 billion. This includes $132.5 to $175.9 billion for direct medical care of adults and $151 billion for lost productivity due to premature deaths. In 2006, lost productivity due to exposure to secondhand smoke cost the country $5.6 billion.[1] About 70 percent of current smokers' excess medical care costs could be prevented by quitting.[19]

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Tobacco, Nicotine, and E-Cigarettes

How does tobacco deliver its effects?

The smoke from combustible tobacco products contains more than 7,000 chemicals. Nicotine is the primary reinforcing component of tobacco; it drives tobacco addiction.[20,21] Hundreds of compounds are added to tobacco to enhance its flavor and the absorption of nicotine.[22] Cigarette smoking is the most popular method of using tobacco; however, many people also use smokeless tobacco products, such as snuff and chewing tobacco, which also contain nicotine (see "Other Tobacco Products"). E-cigarettes, which deliver nicotine in the absence of other chemicals in tobacco, have become popular in recent years (see "What are electronic cigarettes?").

The cigarette is a very efficient and highly engineered drug-delivery system. By inhaling tobacco smoke, the average smoker takes in 1–2 milligrams of nicotine per cigarette. When tobacco is smoked, nicotine rapidly reaches peak levels in the bloodstream and enters the brain. A typical smoker will take 10 puffs on a cigarette over the roughly 5 minutes that the cigarette is lit.[23] Thus, a person who smokes about 1 pack (20 cigarettes) daily gets 200 "hits" of nicotine to the brain each day. Among those who do not inhale the smoke—such as cigar and pipe smokers and smokeless tobacco users—nicotine is absorbed through mucous membranes in the mouth and reaches peak blood and brain levels more slowly.

Immediately after exposure to nicotine, there is a "kick" caused in part by the drug's stimulation of the adrenal glands and resulting discharge of epinephrine (adrenaline). The rush of adrenaline stimulates the body and causes an increase in blood pressure, respiration, and heart rate.[24] Like other drugs, nicotine also activates reward pathways in the brain—circuitry that regulates reinforcement and feelings of pleasure.[20,21]

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Is nicotine addictive?

Yes. Most smokers use tobacco regularly because they are addicted to nicotine. Addiction is characterized by compulsive drug-seeking and use, even in the face of negative health consequences. The majority of smokers would like to stop smoking, and each year about half try to quit permanently. Yet, only about 6 percent of smokers are able to quit in a given year.[25] Most smokers will need to make multiple attempts before they are able to quit permanently.[22] Medications including varenicline, and some antidepressants (e.g. bupropion), and nicotine-replacement therapy, can help in many cases (see "What are treatments for tobacco dependence?").[26]

A transient surge of endorphins in the reward circuits of the brain causes a slight, brief euphoria when nicotine is administered. This surge is much briefer than the "high" associated with other drugs. However, like other drugs of abuse, nicotine increases levels of the neurotransmitter dopamine in these reward circuits,[20,21,27] which reinforces the behavior of taking the drug. Repeated exposure alters these circuits' sensitivity to dopamine and leads to changes in other brain circuits involved in learning, stress, and self-control. For many tobacco users, the long-term brain changes induced by continued nicotine exposure result in addiction, which involves withdrawal symptoms when not smoking, and difficulty adhering to the resolution to quit.[28,29]

The *pharmacokinetic* properties of nicotine, or the way it is processed by the body, contribute to its addictiveness.[24] When cigarette smoke enters the lungs, nicotine is absorbed rapidly in the blood and delivered quickly to the brain, so that nicotine levels peak within 10 seconds of inhalation. But the acute effects of nicotine also dissipate quickly, along with the associated feelings of reward; this rapid cycle causes the smoker to continue dosing to maintain the drug's pleasurable effects and prevent withdrawal symptoms.[30]

Withdrawal occurs as a result of dependence, when the body becomes used to having the drug in the system. Being without nicotine for too long can cause a regular user to experience irritability, craving, depression, anxiety, cognitive and attention deficits, sleep disturbances, and increased appetite. These withdrawal symptoms may begin within a few hours after the last cigarette, quickly driving people back to tobacco use.

When a person quits smoking, withdrawal symptoms peak within the first few days of the last cigarette smoked and usually subside within a few weeks.[31] For some people, however, symptoms may persist for months, and the severity of withdrawal symptoms appears to be influenced by a person's genes.[30,31]

In addition to its pleasurable effects, nicotine also temporarily boosts aspects of cognition, such as the ability to sustain attention and hold information in memory. However, long-term smoking is associated with cognitive decline and risk of Alzheimer's Disease, suggesting that short-term nicotine-related enhancement does not outweigh long-term consequences for cognitive functioning.[32] In addition, people in withdrawal from nicotine experience neurocognitive deficits such as problems with attention or memory.[33] These neurocognitive withdrawal symptoms are increasingly recognized as a contributor to continued smoking.[34] A small research study also suggested that withdrawal may impair sleep for severely dependent smokers, and that this may additionally contribute to relapse.[35]

In addition to the drug's impact on multiple neurotransmitters and their receptors,[30] many behavioral factors can affect the severity of withdrawal symptoms. For many people who smoke, the feel, smell, and sight of a cigarette and the ritual of obtaining, handling, lighting, and smoking the cigarette are all associated with the pleasurable effects of smoking and can make withdrawal or craving worse.[36] Learning processes in the brain associate these cues with nicotine-induced dopamine surges in the reward system[21]—similar to what occurs with other drug addictions. Nicotine replacement therapies such as gum, patches, and inhalers, and other medications approved for the treatment of nicotine addiction may help alleviate the physiological aspects of withdrawal[37–39] (see "What are treatments for tobacco dependence?"); however, cravings often persist because of the power of these cues. Behavioral therapies can help smokers identify environmental triggers of craving so they can use strategies to avoid these triggers and manage the feelings that arise when triggers cannot be.[40,41]

Are there other chemicals that may contribute to tobacco addiction?

Research is showing that nicotine may not be the only ingredient in tobacco that affects its addictive potential.

Smoking is linked with a marked decrease in the levels of monoamine oxidase (MAO), an important enzyme that is responsible for the breakdown of dopamine, as well as a reduction in MAO binding sites in the brain.[42] This change is likely caused by some as-yet-unidentified ingredient in tobacco smoke other than nicotine, because we know that nicotine itself does not dramatically alter MAO levels. Animal research suggests that MAO inhibition makes nicotine more reinforcing, but more studies are needed to determine whether MAO inhibition affects human tobacco dependence.[42]

Animal research has also shown that acetaldehyde, another chemical in tobacco smoke created by the burning of sugars added as sweeteners, dramatically increases the reinforcing properties of nicotine and may also contribute to tobacco addiction.[43]

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What are the physical health consequences of tobacco use?

Cigarette smoking harms nearly every organ in the body,[1,44] and smoking is the leading preventable cause of premature death in the United States. Although rates of smoking have declined, it is estimated that it leads to about 480,000 deaths yearly.[1] Smokers aged 60 and older have a twofold increase in mortality compared with those who have never smoked, dying an estimated 6 years earlier.[45] Quitting smoking results in immediate health benefits, and some or all of the reduced life expectancy can be recovered depending on the age a person quits.[46]

Although nicotine itself does not cause cancer, at least 69 chemicals in tobacco smoke are carcinogenic,[1] and cigarette smoking accounts for at least 30 percent of all cancer deaths.[22] The overall rates of death from cancer are twice as high among smokers as nonsmokers, with heavy smokers having a four times greater risk of death from cancer than nonsmokers.[1]

Foremost among the cancers caused by tobacco use is lung cancer. Cigarette smoking has been linked to about 80 to 90 percent of all cases of lung cancer, the leading cause of cancer death for both men and women, and it is responsible for roughly 80 percent of deaths from this disease.[22,47] Smoking increases lung cancer risk five to tenfold, with greater risk among heavy smokers.[48] Smoking is also associated with cancers of the mouth, pharynx, larynx, esophagus, stomach, pancreas, cervix, kidney, and bladder, as well as

acute myeloid.[1] Cigarette smoking is not the only form of tobacco use associated with cancers. Smokeless tobacco (see "Other Tobacco Products") has been linked to cancer of the pharynx, esophagus, stomach, and lung, as well as to colorectal cancer.[49]

In addition to cancer, smoking causes lung diseases such as chronic bronchitis and emphysema, and it has been found to exacerbate asthma symptoms in adults and children. Cigarette smoking is the most significant risk factor for chronic obstructive pulmonary disease (COPD).[50] Survival statistics indicate that quitting smoking results in repair to much of the smoking-induced lung damage over time. However, once COPD develops, it is irreversible; COPD-related lung damage is not repaired with time.

Smoking also substantially increases the risk of heart disease, including stroke, heart attack, vascular disease, and aneurysm.[51,52] Cardiovascular disease is responsible for 40 percent of all smoking-related deaths.[53] Smoking causes coronary heart disease, the leading cause of death in the United States. Smoking is also linked to many other major health conditions—including rheumatoid arthritis, inflammation, and impaired immune function.[1] Even young smokers aged 26 to 41 report reduced health-related quality of life compared with nonsmoking peers, according to a cross-sectional population study.[54] Recent animal research also identified a pathway between the pancreas and a part of the brain active in nicotine intake, potentially linking cigarette smoking to the risk of developing Type 2 Diabetes.

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What are the effects of secondhand and thirdhand tobacco smoke?

Secondhand smoke is a significant public health concern and driver of smoke-free policies. Also called passive or secondary smoke, secondhand smoke increases the risk for many diseases.[55] Exposure to environmental tobacco smoke among nonsmokers increases lung cancer risk by about 20 percent.[48] Secondhand smoke is estimated to cause approximately 53,800 deaths annually in the United States.[55] Exposure to tobacco smoke in the home is also a risk factor for asthma in children.[56]

Smoking also leaves chemical residue on surfaces where smoking has occurred, which can persist long after the smoke itself has been cleared from the environment. This phenomenon, known as "thirdhand smoke," is increasingly recognized as a potential danger, especially to children, who not only inhale fumes released by these residues but also ingest residues that get on their hands after crawling on floors or touching walls and furniture. More research is needed on the risks posed to humans by thirdhand smoke, but a study in mice showed that thirdhand smoke exposure has several behavioral and physical health impacts, including hyperactivity and adverse effects on the liver and lungs.[57]

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What are electronic cigarettes?

E-cigarettes (electronic nicotine delivery systems) emerged in the U.S. market in 2007 and have rapidly grown in popularity.[78] E-cigarettes, or e-vaporizers, are devices that heat a liquid containing solvents, flavors, and often nicotine.[79] Users inhale the resulting vapor. A variety of designs are available, some mimicking the look of traditional cigarettes. More than 7,000 flavors are available for e-cigarettes,[80] some of which are especially appealing to youth. Many convenience stores, drug stores, grocery stores, and other physical and online retail outlets sell e-cigarettes, although as of August, 2016, it is illegal to sell them to people under 18. Some convenience stores and drugstore chains have also stopped selling e-cigarettes to promote public health.

In 2013, more than one third of cigarette smokers said they had ever used e-cigarettes.[78] According to data from the 2014 Tobacco Products and Risk Perceptions Survey, current cigarette smokers had a greater likelihood of using e-cigarettes. This analysis found that half of cigarette smokers had ever used an e-cigarette and 20.7 percent currently used these devices. However, approximately 10 percent of adults who used e-cigarettes had never smoked previously.[81] Data from the 2014 National Health Interview Survey indicated that 0.4 percent of adults who have never smoked and 0.8 percent of former smokers (abstinent 4 or more years) currently use e-cigarettes.[82] The survey also found that 13 percent of daily e-cigarette users were former smokers who quit during the past year.[82]

As with cigarette use, e-cigarette use is higher among people with mental health conditions—with 3.1 percent currently using compared with 1.1 percent of those without mental illness.[83] It is also a concern that pregnant women are using e-cigarettes, as nicotine exposure during periods of developmental vulnerability

(including prenatal development) has adverse health consequences.[84]

Users report the belief that e-cigarette products are less harmful than traditional cigarettes,[85] and many report using them to help quit smoking traditional cigarettes. While it is not yet clear if e-cigarettes are effective smoking cessation aids, the devices are sometimes marketed for this purpose[86] (see "Are e-cigarettes useful for smoking cessation?"). Some research suggests that older adults use these devices as a tobacco substitute, although not always as a cessation method.[87] Users also cite convenience and being conscientious towards others as reasons for using these products.[88]

Reports of Deaths Related to Vaping

The Food and Drug Administration has alerted the public to thousands of reports of serious lung illnesses associated with vaping, including dozens of deaths. They are working with the Centers for Disease Control and Prevention (CDC) to investigate the cause of these illnesses. Many of the suspect products tested by the states or federal health officials have been identified as vaping products containing THC, the main psychotropic ingredient in marijuana. Some of the patients reported a mixture of THC and nicotine, and some reported vaping nicotine alone. No one substance has been identified in all of the samples tested, and it is unclear if the illnesses are related to one single compound. Until more details are known, FDA officials have warned people not to use any vaping products bought on the street, and they warn against modifying any products purchased in stores. They are also asking people and health professionals to report any adverse effects. The CDC has posted an information page for consumers.

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has identified a thickening agent—Vitamin E acetate—as a chemical of concern among people with e-cigarette or vaping associated lung injuries. They recommend that people should not use any product containing Vitamin E acetate, or any vaping products containing THC; particularly from informal sources like friends, family, or in-person and online dealers. They also warn against modifying any products purchased in stores, or using any vaping products bought on the street. People, including health professionals, should report any adverse effects of vaping products. The CDC has posted an information page for consumers.

How does the federal government regulate e-cigarettes?

The U.S. Food and Drug Administration (FDA), which regulates cigarettes, tobacco, and smokeless tobacco, gained the authority in 2016 to also regulate electronic nicotine delivery systems (such as e-cigarettes and vape pens), all cigars, hookah (waterpipe) tobacco, pipe tobacco, and nicotine gels, among other tobacco products. Under the new regulations, e-cigarette manufacturers must list ingredients, and vendors cannot sell e-cigarettes to people under age 18.[110]

Is it true that e-cigarettes are safer than traditional cigarettes?

Are e-cigarettes useful for smoking cessation?

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