

2009

Issues In-Depth: Advancing Understanding of Drug Addiction and Treatment

Roxanne Greitz Miller

Chapman University, rgmiller@chapman.edu

Follow this and additional works at: http://digitalcommons.chapman.edu/education_articles

 Part of the [Curriculum and Instruction Commons](#), [Curriculum and Social Inquiry Commons](#), [Science and Mathematics Education Commons](#), [Social and Philosophical Foundations of Education Commons](#), and the [Substance Abuse and Addiction Commons](#)

Recommended Citation

Miller, R. G. (2009). Issues In-Depth: Advancing Understanding of Drug Addiction and Treatment. *Science Scope*, 32(6), 14-17.

This Article is brought to you for free and open access by the College of Educational Studies at Chapman University Digital Commons. It has been accepted for inclusion in Education Faculty Articles and Research by an authorized administrator of Chapman University Digital Commons. For more information, please contact laughtin@chapman.edu.

Issues In-Depth: Advancing Understanding of Drug Addiction and Treatment

Comments

This article was originally published in *Science Scope*, volume 32, issue 6, in 2009.

Copyright

National Science Teachers Association

Advancing understanding of drug addiction and treatment

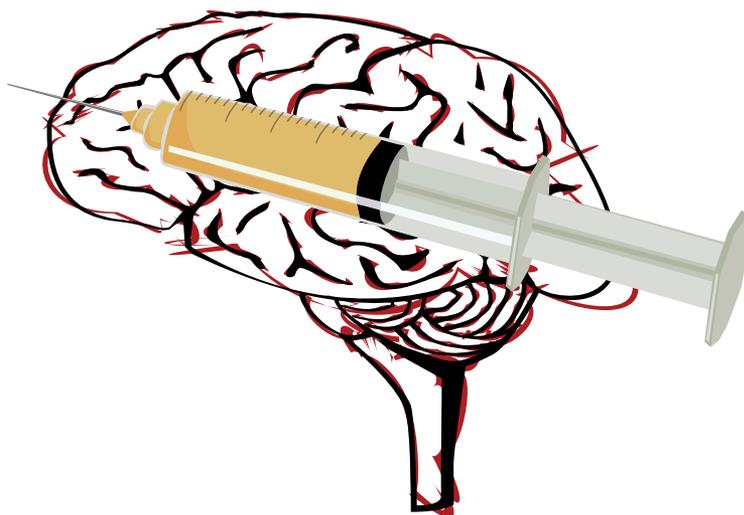
by Roxanne Greitz Miller

While most school districts utilize a drug abuse resistance curriculum, as science teachers, it is our responsibility to understand the science behind drug addiction in order to most effectively educate our students against drug abuse. In the last two decades, increases in scientific technology have permitted significant discoveries surrounding the neurobiology, genetic components, and treatment of drug addiction. This article addresses the latest scientific knowledge about drug addiction and treatment with information that can be used in the middle school setting, focusing on cocaine addiction to illustrate the points discussed.

What is drug addiction?

Most scientists now consider drug addiction a biomedical rather than a psychological condition, or a failure of will, as many laypersons still believe. The American Medical Association, American Psychiatric Association, and other organizations now describe drug addiction as a brain disease that causes an uncontrollable, compulsive drug craving, seeking, and use even in the face of negative health and social consequences. Addiction must be distinguished from the term *dependence*, which refers to whether physical or psychological withdrawal symptoms will occur if drug use is discontinued. Certain drugs, such as heroin and alcohol, create physical dependence, while other drugs, such as cocaine, produce very few symptoms of physical dependence and are therefore referred to as primarily creating psychological dependence.

Drug education programs of the past, particularly in the 1970s and 1980s, highlighted physical versus psychological addiction, an emphasis that is now seen as archaic in the drug treatment world. Addiction can occur whether the drug used creates physical or psychological dependence, and the compulsion to use the two classes of drugs can be equal. Addiction causes physical changes in the brain, regardless of whether the drug used causes physical withdrawal symptoms upon discontinuance, and it is these changes in brain function that result in the addict's compulsion to continue drug use.



An analogy used to illustrate how addiction occurs is that “after a certain amount of a drug is consumed, it is as if a switch in the brain is flipped from normal to addict” (Leshner 2001). The amount of drugs that must be consumed to cause these brain changes is different for everyone, which helps to explain why some people become addicted after low to moderate drug use when others can use higher levels of drugs or use them for longer periods of time and not show symptoms of addiction (Leshner 2001). Specifically, in the case of cocaine and related stimulant drugs (such as crystal methamphetamine), some addicts report that craving began after as few as one to two incidents of use.

Recent scientific evidence suggests that between 50–70% of the variability in susceptibility to becoming addicted is due to genetic factors, while environmental and biological factors also play a role (Leshner 2001). Therefore, while it is accurate to state that addiction can run in families due to their shared genetic makeup, an absence of addicts in a family does not remove the possibility of genetic predisposition to addiction. This is why the phrase “be smart, don’t start” represents the most effective way to prevent addiction. Abstaining entirely from drug use will prevent addiction from occurring, regardless of whether or not a genetic predisposition to addiction is present.

For those who do not heed this advice and become addicted, even if their brain function is returned to normal after treatment, it is possible that the psycho-

logical effects of addiction, including the compulsion to use drugs, can continue for years after drug use has stopped. Stressing these points—that one cannot predict when addiction will develop, that one can carry a genetic predisposition to addiction without knowing it, that addiction can develop even after only low levels of drug use, and that the effects of addiction can last long after treatment—when educating students strengthens the argument that prevention and abstinence from all drug use are the most effective treatment.

While it is true that a drug user initially voluntarily chooses to engage in drug use, the neurobiology research contradicts the notion that the user can simply “decide” to quit. Due to the changes in the brain over time, the addict loses control over what was once voluntary drug use, and that behavior becomes compulsive and truly uncontrollable, much like other behavioral expressions of brain disease. For example, Parkinson’s patients cannot control their trembling, nor can schizophrenics control their hallucinations. It is important for students to understand that due to changes in the brain, outside help and medical treatment are almost always necessary to break addiction. While stories of individuals who are able to break their addiction without outside help are indeed true, it is estimated that less than 1 in 10 former addicts successfully quits without formal treatment. When compared to the total number of current and former addicts and those who have died from drug use, it becomes readily apparent how few drug addicts recover without formal treatment. Understanding the extreme difficulty in quitting without formal treatment helps students to understand why family members are so often unsuccessful in changing the behavior of their loved ones who struggle with addiction, no matter how hard they try or how much they are loved by the addict.

What causes the high?

The sense of a drug “high” comes from the effect of the drug on the process of neurotransmission in the brain. Most science teachers are familiar with information in the brain being delivered from one neuron to another in a nerve pathway using chemicals called *neurotransmitters*. The neurotransmitters carry the signal from the sending neuron to the receiving neuron across the *synapse*, the space between neurons. Common neurotransmitters in the brain include dopamine, serotonin, gamma-aminobutyric acid (GABA), and acetylcholine. *Dopamine*, the specific neurotransmitter affected by cocaine use, is directly or indirectly affected by virtually every addictive substance. Dopamine is released in two ways: (1) in constant, low levels (called tonic levels)

that aid in proper brain and motor function, and (2) in higher levels during a person’s engagement in activities that activate the reward pathway of the brain, such as eating, sex, or addictive drug use.

Drugs of abuse alter normal neurotransmission by increasing the level or effect of the neurotransmitter in the brain. This can be accomplished by increasing the production or release of neurotransmitters, by enhancing the reception of the neurotransmitter at the receiving neuron, or by preventing neurotransmitter reuptake in/return to the sending neuron from which it was released. No matter the exact mechanism, the drug high is a result of increasing the neurotransmitter’s effect, causing an increased stimulus of the receiving neurons. This state of overstimulation of the receiving neurons by the neurotransmitters (specifically, dopamine) causes the feelings of euphoria and satisfaction reported by drug users.

When dopamine is released by the sending neuron to the receiving one, it binds to *dopamine receptors* on the receiving neuron’s surface. After the signal is sent, dopamine is transported back to the sending neuron by a specialized protein, the *dopamine transporter*. Cocaine increases dopamine levels by binding to the dopamine transporters, thus preventing dopamine’s reuptake by the sending neurons and thereby causing a buildup of dopamine in the synapse. This buildup floods the dopamine receptors on the receiving neuron. It is this excess of dopamine in the synapse that causes excessive stimulation of the receiving neurons and the euphoria associated with cocaine use.

What treatments are possible?

Historically, psychosocial intervention (counseling) has been used to help addicts fight their drug addictions. Examples include the counseling groups sponsored by organizations such as Alcoholics Anonymous and Narcotics Anonymous, as well as more individualized inpatient and outpatient counseling programs.

Medical intervention for drug addiction is often combined with psychosocial intervention. All the current medical therapies work by either causing severe negative side effects if the drug of abuse is used, or by substituting a drug that acts on the same receptors as the drug of abuse and therefore allows the drug addict to engage more effectively in psychosocial therapy without constant craving and compulsion to use drugs. For example, Antabuse is a drug commonly prescribed to alcohol-dependent patients; nausea and vomiting occur if alcohol is consumed while taking Antabuse. The nicotine patch, which is actually a source of nicotine and not another type of drug, acts exactly the same way as

tobacco on nicotine receptors. The idea behind its use is that the patch satisfies the addict's nicotine craving while removing the psychological and social cues that reinforce smoking behavior, and decreases the nicotine dosage over time while permitting addicts to engage in psychosocial therapy and ultimately wean themselves from the need for nicotine. Unlike the nicotine patch, methadone, which is used in heroin addiction therapy, is an example of a drug that is similar to the drug of abuse but is not the same chemical compound. A problem with nearly all treatments that substitute another drug for the drug of abuse is that the substitute itself has an approximately equivalent addictive liability if misused; meaning, it is possible to become addicted to the treatment.

Cocaine addiction, until recently, has shown to be highly resistant to medical treatment. The mechanism of cocaine addiction via its effect on dopamine levels has been known for over a decade, but medical treatments aimed at affecting the dopamine receptors have generally been unsuccessful due to the fact that the dopamine pathway is intricately linked to mood, motor control, and proper mental functioning (recall the examples of Parkinson's disease and schizophrenia mentioned above, which result from malfunctions of the dopamine neurotransmitter systems). Blocking the reception of dopamine means that not only is the reward-pathway release of dopamine blocked as a result of drug use, but the normal (tonic) level of dopamine the body needs to function properly also cannot be maintained. Therefore, drug addiction therapies that block the dopamine receptors have often been either unsuccessful at halting addiction or have caused undesirable side effects in patients.

Experimental therapies aimed instead at preventing the release of dopamine during excitation of the reward pathway are providing promise in treating cocaine addiction. Some of these therapies have capitalized on using the neurotransmitter GABA, which inhibits dopamine's release. GABA regulates the overall excitability of the human brain. If dopamine can be considered the gas pedal of a car, GABA is the brake (Lemonick 2007). When GABA is released, it blocks dopamine release via the reward pathway, while still allowing for tonic release, and thereby keeps the neurons in a normal, balanced state. The reasoning for this GABA-based approach to drug addiction therapy is that if reward-activated dopamine release can be inhibited, the excessive levels of dopamine flooding the synapses will be avoided, thus eliminating the high experienced during cocaine use, but normal tonic functioning will still be preserved.

Examples of GABA-active drugs include tranquilizers and antiseizure medications. Among the most promis-

ing of these in fighting cocaine addiction is vigabatrin (gamma-vinyl GABA), which is currently being tested across the United States at multiple treatment centers (ClinicalTrials.gov 2008). Vigabatrin is a drug used outside of the United States to treat epilepsy. It is unique among the GABA-active drugs because it prevents the breakdown of GABA; therefore, when a neuron receives the signal to release GABA there is a vast storehouse of GABA available, far more than would normally be present. The end result of this increased amount of available GABA is that reward-pathway-dopamine release is highly inhibited, which prevents the usual craving for cocaine and the high and feelings of euphoria even if cocaine is still used. In essence, vigabatrin combats the excitability of the neurons normally affected by cocaine and keeps the addict's craving and psychological compulsion to use cocaine in check, even if the addict falls off the wagon, engaging in drug use during psychosocial treatment. This is highly important for addicts who do not check into a residential treatment center and instead opt to engage in outpatient therapy, where the same external stimuli and outside stresses and pressures to use cocaine are present. Vigabatrin should not be viewed as a cure for cocaine addiction; its goal is to decrease or remove the craving and high associated with cocaine in order to make psychological treatment of addiction more effective.

Interestingly, recent research has shown that virtually all addictive substances activate this dopamine neurochemical pathway, and it is this neurochemical pathway that is responsible for sending the "that feels good" signal when an addictive substance is consumed. Dr. Frank Vocci, director of pharmacotherapies at the National Institute of Drug Abuse, was quoted in *Time* magazine regarding vigabatrin, saying optimistically, "If it works, it will probably work on all addictions" (Lemonick 2007). It is expected that results from the clinical trials on vigabatrin will be available in 2009, along with an examination of any side effects and potential dangers of treatment.

Final words

When discussing drugs and addiction with students, there are two primary purposes: (1) preventing use, abuse, and thereby possible addiction in students, now or in the future, and (2) helping students to understand addiction so that they can understand the behavior of others and know the options for treatment. There are still many people—youth and adults—who believe not only that addiction is purely psychological and can be halted at will, but also that because addiction begins with a voluntary act, it does not deserve to be classified as a disease. Thus, discussions of addiction can be emotional and heated, and as teachers we need to be prepared with

scientific evidence to support any claim we might make or concept we might teach. While it is true addiction starts with a voluntary act, the complex neurochemical cascade that follows this voluntary act is what makes addiction a disease rather than a conscious choice.

While focusing on drug avoidance and decision-making strategies is highly important in aiding students to abstain from drug use, early adolescents in middle grades are more able than their younger counterparts to grasp the scientific mechanisms of drug action and to understand the genetic predisposition that some individuals have to addiction. If we employ a total approach to drug education in our curricula, incorporating up-to-date information on the psychological, scientific, and social underpinnings of drug use and addiction, we can aspire to be even more effective than we have been in the past at helping our students live healthy lives.

References

ClinicalTrials.gov. 2008. Vigabatrin treatment for cocaine addiction. <http://clinicaltrials.gov/ct2/show/NCT0061>

1130?term=catalyst+pharmaceuticals&rank=2.

Lemonick, M.D. 2007. How we get addicted. *Time*. July 5. www.time.com/time/magazine/article/0,9171,16404361,00.html.

Leshner, A.I. 2001. Addiction is a brain disease. *Issues in Science and Technology Online*. www.issues.org/17.3/leshner.htm.

Resources

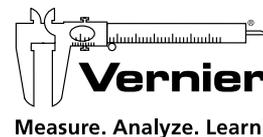
The brain's drug reward system—www.nida.nih.gov/NIDA_Notes/NNVol11N4/Brain.html

The neurobiology of drug addiction—www.nida.nih.gov/Teaching2/teaching5.html

Roxanne Greitz Miller (rgmiller@chapman.edu) is assistant professor of teacher education at Chapman University in Orange, California, and a former middle and senior high school biological and physical science teacher.



"Magnet Finder" project: build a robot to detect a magnet hidden under one of three walnut shells.



Enhance LEGO® NXT Robotics with Vernier Sensors

Is your robotics team looking for deeper investigations and more creativity?

Now you can expand your options beyond the basic sensors included with LEGO® Mindstorms® NXT. Imagine the robotics projects your students could create using a Vernier Magnetic Field Sensor, a Force Sensor, or a Gas Pressure Sensor. The NXT Sensor Adapter can be used with LEGO® Mindstorms® NXT robots and **over 30 Vernier sensors** for sensor-based control systems.

\$39 ORDER CODE
BTA-NXT



Dozens of robotics videos available at
www.vernier.com/nxt

Vernier Software & Technology • Toll Free: 888-837-6437
www.vernier.com