

Research Update is published by the Butler Center for Research to share significant scientific findings from the field of addiction treatment research.

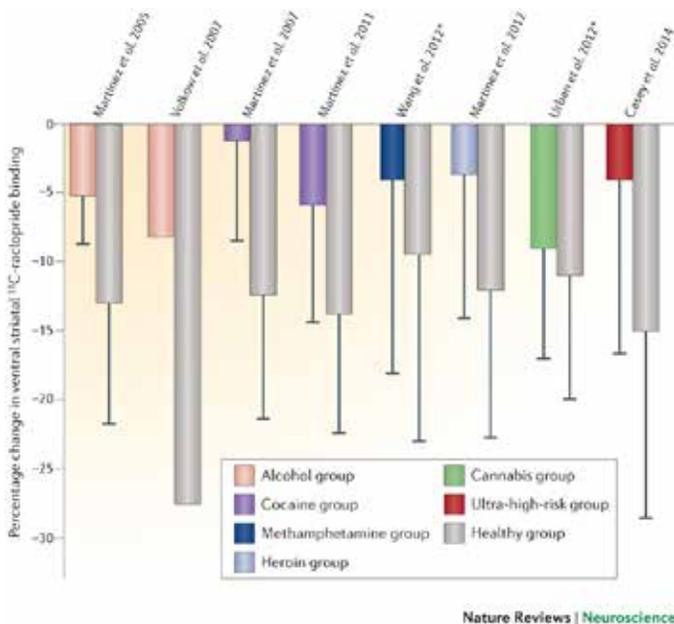
The Brain Disease Model of Addiction

Many people assume that addiction is not a disease but a weakness of character.¹ This misconception contributes to the stigma of addiction and unfairly minimizes the challenge of overcoming chemical dependence. Advances in neuroscience and imaging technology have rapidly evolved our understanding of addiction and demonstrated a great deal of support for what is often referred to as the brain disease model of addiction. This model considers genetic and environmental factors that cause physical changes to the brain and is the basis for many existing and emerging concepts, including (but not limited to) genetic predisposition to addiction, behavioral addictions, psychopharmacological treatment interventions, and cross-addiction.

Defining Addiction

The complexity of addiction makes it difficult to define in a way that allows for precise study. Due to the significant impacts of social, developmental, and other external factors on addiction, symptoms and prognoses vary a great deal from person to person. This interdisciplinary complexity has drawn comparisons between addiction and other complex diseases, including obesity, diabetes, and cardiovascular disease.^{2,3} However, most scientists agree that a number of characteristics are generally present among addicted individuals. In summarizing the disease model of addiction, Volkow, Koob, and McLellan (2016) highlighted three primary symptoms of addiction: (1) desensitization of the reward circuits of the brain; (2) increased conditioned responses related to the substance an individual is dependent upon; and, (3) declining function of brain regions that facilitate decision making and self-regulation. These themes are echoed throughout the neurobiological literature on addiction^{2,4} and are also common in other psychology fields, including behavioral conditioning^{5,6} and behavioral economics research.⁷

By identifying observable symptoms associated with addiction, scientists have been able to narrow research to structures and chemicals associated with these functions and behaviors.



Paired with ever-increasing imaging technology, researchers have been able to identify a number of consistent, physical characteristics in the brain that support the concept of addiction as a brain disease.

Physical Brain Changes Associated With Addiction

Brain physiology plays two major parts in addiction: First, certain hereditary traits can make an individual more vulnerable to developing a physical dependence after exposure to a rewarding stimulus; second, physical

This graph shows the differences in dopamine release between control subjects and individuals with various chemical dependencies. Large negative differences (like those seen among controls) are representative of larger amounts of dopamine in the striatum after administration of a stimulant. Smaller differences (like those seen among the chemically-dependent subjects) are representative of less dopamine in the striatum after stimulant administration.

Data Source: Nutt, D. J., Lingford-Hughes, A., Erntzoe, D., and Stokes, P. R. A. (2015). The dopamine theory of addiction: 40 years of highs and lows. *Nature Reviews Neuroscience*, 16, 305-312.

THE HAZELDEN BETTY FORD EXPERIENCE

Comprehensive Opioid Response with the Twelve Steps (COR-12™)

COR-12™ is Hazelden Betty Ford Foundation's current effort to combine evidence-supported, disease model research with treatment options available to our patients. The COR-12™ treatment path includes group therapy and lectures that focus on opioid addiction in addition to medication-assisted treatment (MAT) as a means to helping people achieve a stable, Twelve Step-based recovery lifestyle and ultimate abstinence from opioids. Components of the MAT plan include the possible use of two medications—buprenorphine/naloxone (Suboxone®) and extended-release naltrexone (Vivitrol®)—which are offered under closely supervised care. These medications have been shown to improve the likelihood of abstinence from opioids, boost retention and engagement in treatment, reduce cravings for opioids, and lower relapse rates. For additional information, visit HazeldenBettyFord.org/COR12.

QUESTIONS & CONTROVERSIES

Why is the brain disease model of addiction so controversial if it is supported by evidence?

One of the major controversies about the disease model of addiction is related to its impact on patients in recovery. While most professionals generally don't argue the fact that behaviors related to addiction are the result of physiological effects on the brain, many worry that educating patients about the disease model of addiction may not reduce the stigma of addiction and will instead erode patients' sense of control over their treatment.^{1,9,10} Others argue that the cost of disease model research does not justify its continued study; disease model research has received a large portion of available research funding in recent years, yet the number of new treatment options resulting from these studies has been lower than some anticipated.¹⁰ Proponents of the disease model point to a number of benefits to support their research, including advances in MAT and significant increases in core understanding of addiction that may pave the way to more precise and effective treatment options in the future.

Why are programs using prescription drugs to treat dependence? Isn't that part of the problem?

It is understandable for individuals seeking treatment to be suspicious of pharmaceutical solutions; however, modern medication-assisted therapies rely on non-habit-forming drugs that are made to counteract the reward-center activation effect that occurs when alcohol and other drugs are used. This reduces cravings and increases the odds for successful recovery. For those who are uncomfortable with MAT, many behavioral interventions, including Twelve Step programs and cognitive-behavioral therapy, have been supported through research to be effective treatments

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changes caused by repeated exposure to rewarding stimuli strengthen the dependence by deteriorating brain function critical to self-regulation and motivation to remain abstinent, even in the face of extreme consequences.^{3,4,8} Different substances have different specific chemical effects on the brain; however, by using a broader definition of addiction, scientists have identified hereditary and conditional physical characteristics that are consistent across all chemical addictions and even include a number of compulsive or addictive behaviors as well.

The large role that the neurotransmitter dopamine plays in addiction is well documented (for more information on this topic, see the *Research Update* “Drug Abuse, Dopamine, and the Brain’s Reward System”). For many years, scientists have known that the flood of dopamine released by addictive substances in the brain is what leads to the feeling of reward (or the “high”), but more recent research has found another critical role of dopamine in addiction. Specific dopamine receptors (called “D2 receptors”) in a region of the brain known as the striatum appear to be responsible for the motivation of individuals to forgo instant gratification in order to work toward a challenging, but larger, reward.⁴ When there is a lower dopamine response among D2 receptors in the striatum, individuals begin to experience impulsivity, including compulsive short-term reward-seeking behavior.^{2,3,4} Some people have a genetic propensity for fewer D2 receptors in the striatum that makes them naturally inclined toward impulsivity and also more vulnerable to addiction.⁸ Conversely, those who have higher D2 receptor availability in the striatum tend to be much more successful in treating addiction through behavioral interventions.⁴ For others, reduced D2 receptors are a result of ongoing alcohol or drug use.^{2,3,4} As individuals continue to regularly engage in activities that prompt a dopamine-related reward response in the brain (e.g., taking a drug, drinking alcohol, smoking a cigarette, or engaging in a compulsive behavior such as overeating), the brain tries to regulate by reducing dopamine levels. This leads not only to a need for more and more of the substance/activity to achieve a “high”, but also to the continual erosion of D2 receptor availability in the striatum, which in turn makes those with addiction more impulsive, more likely to use, and less able to successfully achieve sobriety.^{2,3,4,8}

Important Considerations for Patients

While studies related to the disease model of addiction are generally much newer than many other forms of addiction research, a number of policies and interventions have already been developed as a result of our better understanding of the physiological nature of addiction. Medication-assisted treatment (MAT) has evolved as a result of recent imaging and brain function research, allowing scientists and practitioners to prescribe nonaddictive medications to significantly reduce patients’ cravings for alcohol, tobacco, and opioids.³ Positron emission tomography (PET) scans have tied together impulsivity, motivation, and addiction in a way that is allowing scientists to collaborate on effective treatments for a multitude of disorders affected by striatal D2 receptors (including ADHD, compulsive overeating, and behavioral addictions) and to focus on solutions that can increase an individual’s ability to improve overall self-regulation and motivation to remain in a treatment program.⁴ Public health policy is recognizing addiction as a medical condition, and under the Affordable Care Act, insurance companies must now provide benefits for substance abuse treatment.³

Summary

Despite decades of public misinterpretation of substance dependence as a sign of moral weakness or a simple unwillingness to exert self-control, professionals can now point to common neurobiological patterns that underlie chemical and behavioral addictions. These findings are the foundation of understanding addiction as a treatable disease and may pave the way for more precise and effective treatment interventions in the future.

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for dependence and can be used to support sustained abstinence from drugs and alcohol. It is the abstinence from the substance that is critical to long-term physical improvements in the brain, not the medication itself.

HOW TO USE THIS INFORMATION

Patients: Addiction is a degenerative physical disease of the brain. However, addiction can be treated, and no matter what genetic characteristics you may have, physical changes in the brain can be reversed through sustained abstinence. If you have been unsuccessful with behavioral interventions alone, MAT can help address physical parts of the addiction while you learn critical new behaviors, examine unhealthy attitudes, and address important environmental factors through a treatment program.

Friends and Family: When supporting loved ones with addiction, it is important to remember that their dependence is not a result of character, nor is it something they can “just quit.” Addiction is a complicated and multifaceted disease with physical, social, and psychological components, much like diabetes or heart disease. Sustained abstinence from alcohol and other drugs can reverse the physical damage caused by addiction, and this requires intensive participation in a holistic treatment program, especially for those whose genetic brain structure makes them more prone to substance dependence.

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The Butler Center for Research informs and improves recovery services and produces research that benefits the field of addiction treatment. We are dedicated to conducting clinical research, collaborating with external researchers, and communicating scientific findings.

Bethany Ranes, PhD, Director

If you have questions, or would like to request copies of *Research Update*, please call 800-257-7800, ext. 4405, email ButlerResearch@HazeldenBettyFord.org, or write BC 4, P.O. Box 11, Center City, MN 55012-0011.