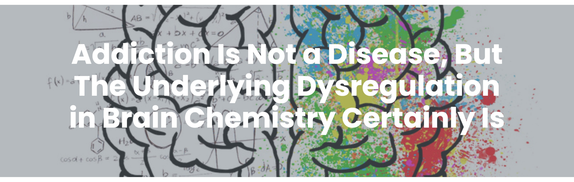


Addiction Is Not a Disease, But The Underlying Dysregulation in Brain Chemistry Certainly Is

By Gaurav Dubey (MS Biotechnology)

Over the course of human history, drug and alcohol addiction has been a powerful force, controlling the lives of its victims. Until recently, addiction has been viewed as a moral failing or character weakness, but scientific studies and clinical trials conducted over the last few decades suggest that an individual's brain chemistry plays a significant role in predisposing them to addiction. Another popular, albeit incomplete, assessment of addiction is simply labeling it as a "disease." In reality, drug and alcohol addiction is but a *symptom* of an underlying disease characterized by dopamine imbalance in the brain. Thus, it is crucial to note that addiction is a disease of the brain's reward system, which heavily relies on the neurotransmitter dopamine. This is an important distinction to draw, as it will help in better understanding a potentially major factor in the etiology of substance use disorder (SUD). This blog will explore dopamine imbalance in the brain and how it relates to the development and progression of SUD.

What Is Dopamine and How Does It Influence The Reward Pathway?

"Brain DA plays a key role in the processing of information about saliency, which is at the heart of its ability to regulate or influence reward, reward expectation, motivation, emotions, and the feelings of pleasure."[1-5] When the brain's dopamine related circuits operate in an integrated and balanced fashion, the outcome "manifests as the execution of appropriate behaviors (proper inhibitory control and decision making) in a broad range of circumstances." Essentially, this means that people with a *normal* brain are able to make "better" decisions and exhibit proper self-control in a wide variety of situations. Rational decision-making and the exercising of self-control is generally attributed to proper functioning of the pre-frontal cortex (PFC). Dopamine levels are normally elevated when we perform actions indispensable to our survival, such as eating and procreating. Ultimately, dopamine is what conditions the human race to do the things we need to do to ensure our survival and the propagation of the species. When our natural, endogenous reward circuitry is introduced and consistently exposed to chemicals that radically alter normal dopamine transmission, chaos can ensue.

High-Quality Clinical Evidence Suggests Etiology of Addiction is Genetic

There is solid evidence now that suggests drugs of abuse affect dopamine transmission.[1] Because only a minority of people who try drugs of abuse develop a substance use disorder (SUD), attempts have been made to identify predisposing neurobiological features. One long-considered hypothesis is that increased susceptibility reflects preexisting perturbations in the mesolimbic dopamine system.[1] That is to say some individuals are highly likely to be more genetically predisposed towards addiction than others. In the "rooms" of Alcoholics Anonymous, a now global fellowship of men and women who help each other live sober and free, this dopamine imbalance is said to manifest as feelings of restlessness, irritability and discontent. Indeed, for such individuals, drugs and alcohol can be a solace at first by temporarily masking their condition by artificially boosting dopamine.

However, as most addicts come to eventually realize about their drug use, it is simply an unsustainable lifestyle. Despite this realization, many find it utterly impossible to quit (as is sadly proved time and again by relapse rates published in the literature). Why exactly do addicts and alcoholics find themselves so *powerless* in the realm of trying to control alcohol or drug use when they can exceed in exercising self-will and control in other areas of life? Science may provide an answer as we explore how repeated exposure to the common drugs of abuse alters brain chemistry and structure.

Neural Hijacking: How Drugs Spike Dopamine Levels, Alter Brain Architecture

To better understand exactly how drugs alter dopamine levels, a brief dive into some basic *neuropharmacology* is warranted. Throughout the pathogenesis of addiction, the inflated value of the drug in the reward, motivation, and memory circuits overcomes the inhibitory control exerted by the prefrontal cortex (PFC).[1] Anhedonia is a hallmark characteristic of post-acute withdrawal syndrome or PAWS (commonly experienced following the acute-phase of opiate withdrawal).[2] From a clinical perspective, anhedonia is marked by diminished interest or pleasure in activities that are naturally rewarding (e.g. food and sex) and a struggle all too common in recovering addicts. It is a common cause of relapse, especially in early recovery.[3]

This results in a positive-feedback loop ignited by drug use and perpetuated by the enhanced activation of the motivation/drive and memory circuits. In plain English, this essentially describes a phenomenon where drugs literally hijack the brains reward circuitry due to their enhanced value and ability to release five to ten times as much dopamine than normal behaviors (e.g. eating a tasty meal, having sex, exercising, etc.) are able to produce. This results in the phenomenon of "tolerance," as habitual drug use requires higher concentrations to try and achieve what has become the "new normal" for the individual abusing drugs and slipping into dependency and addiction.

Why Abstinence Helps Heal the Fastest: All Drugs of Abuse Spike Dopamine, Set Off Cravings

Unfortunately, regular use of common drugs of abuse will continue to stimulate excess dopamine production (in particular, methamphetamine, cocaine, alcohol and opiates) significantly modulates the brains natural ability to perceive reward reinforcement and this has serious physiological and psychological consequences for the struggling addict.[1] It is further important to note here that irrespective of the specific pharmacologic mechanisms of action for each of the aforementioned compounds, they *all release dopamine and light up that reward pathway.* A 2000 study published in PNAS demonstrated, with advanced imaging techniques, how rodents who received an intravenous dose of cocaine "significantly increased dopamine efflux in the NAcc [nucleus accumbens] and amygdala as measured by intracranial microdialysis in a separate group of rats."[1] This spike in dopamine was directly linked to cravings observed in the rodents. Thus, the dopamine spike caused by drugs or alcohol not only sets off treacherous cravings, but further delays the healing process to where healthier things, like making love or eating a delicious meal, take longer to naturally make us happy again. With discipline and time, studies have shown our brains can readjust to utilizing its natural reward pathways in a homeostatic fashion.

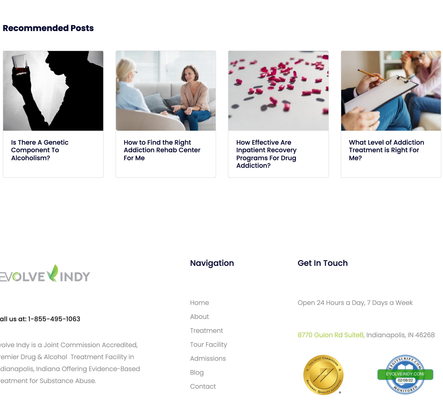
Evidence Suggests Abstinence Provides The Fastest Route to Recovery

This is why a heroin addict attempting recovery that continues to drink alcohol will quickly be faced with the phenomenon of craving setting in. That's all it takes for the dopamine reward pathway to be activated by that first drink. Persistent overstimulation of the dopamine reward system pathway results in dysregulation of dopamine receptor concentration and disrupts homeostatic, endogenous dopamine release. The brain's architecture literally changes as it desperately upregulates the amount of dopamine receptors while one's tolerance rises. Essentially, things like great sex and great food can't possibly compare to the new "normal" level of stimulation in a drug abuser—so it is common for them to lose interest in these things. Upon cessation of the drug of choice, there is often a period of anhedonia, which can certainly be challenging and is a hallmark of PAWS (Post-Acute Withdrawal Syndrome) when referring to cessation of opiate narcotic drugs. Luckily, there is mounting clinical evidence supporting the neurological benefits of sobriety—especially with regards to neurogenesis and balancing one's dopamine levels back to a homeostatic state following a period of abstinence.[1,2]

Closing Thoughts: The Path to Recovery Is Simple, But Not Easy

From a clinical standpoint, this is the strongest evidence to support total abstinence in sobriety. The brain is not able to clearly distinguish between what drug stimulated the dopamine release (and quite frankly, it really doesn't care because you feel good either way).[1] Making the choice to no longer rely on extraneous sources for dopamine can allow your brain to reorient itself toward finding pleasure in life. Indeed, such a doctrine is simple enough to comprehend, but is sure to be anything but easy in its application to our daily lives. While it will take some time to readjust, it is a worthwhile endeavor for anyone who is in the midst of addiction. Knowing that—with the addition of healthy habits into one's life—the brain can heal, one may find the courage to move forward and embrace sobriety in all its beauty.

To learn more about how substance abuse disorder is a symptom of dopamine imbalance in the brain and not a disease in and of itself, listen to James Sweasy Live discuss low-dopamine and it's relationship to substance abuse disorder (SUD) in this podcast. Also, check out his Youtube video exploring dopamine imbalance below!



WORKS CITED

1. Volkow, N. D. *et al.* Addiction: Decreased reward sensitivity and increased expectation sensitivity conspire to overwhelm the brain's control circuit. *BioEssays News Rev. Mol. Cell. Dev. Biol.* **32**, 748–755 (2010).
2. Pacek, L. R. *et al.* Perceived nicotine content of reduced nicotine content cigarettes is a correlate of perceived health risks. *Tob. Control* **27**, 420–426 (2018).
3. Mesolimbocortical and nigrostriatal dopamine responses to salient non-reward events. – PubMed – NCBI. https://www.ncbi.nlm.nih.gov/pubmed/10727783.
4. Tobler, P. N., O'Doherty, J. P., Dolan, R. J. & Schultz, W. Reward value coding distinct from risk attitude-related uncertainty coding in human reward systems. *J. Neurophysiol.* **97**, 1621–1632 (2007).
5. Human striatal response to salient nonrewarding stimuli. – PubMed – NCBI. https://www.ncbi.nlm.nih.gov/pubmed/12954871.
6. Willuhn, I., Wanat, M. J., Clark, J. J. & Phillips, P. E. M. Dopamine Signaling in the Nucleus Accumbens of Animals Self-Administering Drugs of Abuse. *Curr. Top. Behav. Neurosci.* **3**, 29–71 (2010).
7. Leyton, M. & Vezina, P. Dopamine ups and downs in vulnerability to addictions: a neurodevelopmental model. *Trends Pharmacol. Sci.* **35**, 268–276 (2014).
8. Hatzigiakoumis, D. S., Martinotti, G., Giannantonio, M. D. & Janiri, L. Anhedonia and Substance Dependence: Clinical Correlates and Treatment Options. *Front. Psychiatry* **2**, (2011).
9. Your Brain on Drugs: Dopamine and Addiction. *Big Think* https://bigthink.com/going-mental/your-brain-on-drugs-dopamine-and-addiction (2010).
10. Weiss, F. *et al.* Control of cocaine-seeking behavior by drug-associated stimuli in rats: Effects on recovery of extinguished operant-responding and extracellular dopamine levels in amygdala and nucleus accumbens. *Proc. Natl. Acad. Sci. U. S. A.* **97**, 4321–4326 (2000).
11. Boileau, I. *et al.* Rapid Recovery of Vesicular Dopamine Levels in Methamphetamine Users in Early Abstinence. *Neuropsychopharmacology* **41**, 1179–1187 (2016).
12. Garavan, H., Brennan, K. L., Hester, R. & Whelan, R. The Neurobiology of Successful Abstinence. *Curr. Opin. Neurobiol.* **23**, 668–674 (2013).

addiction dopamine opioid addiction reward pathway rewiring the brain

Facebook Twitter LinkedIn

Recommended Posts

Is There A Genetic Component To Alcoholism?

How to Find the Right Addiction Rehab Center For Me

How Effective Are Inpatient Recovery Programs For Drug Addiction?

What Level of Addiction Treatment is Right For Me?