Katherine Belilty, Mark Dodici, Nathan Shin

Professor Yael Niv and Dr. Rachel Bedder

PSY/NEU 338

3 May 2022

A Long and Winding Road: Detailing Pathways to Addiction

Introduction

Per the DSM-5 (the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition), a diagnosable "addiction" has several key characteristics that distinguish it from a simple habit. These requirements include: impaired control — the subject finds himself unable to cut down on substance use or refrain from the problem action, even if he wants to; social problems — engagement in the problem action causes neglect or other problematic treatment of those close to the addict; risky use — the subject engages with the object of abuse in dangerous ways, even when he knows of its negative effects; and physical dependence — the subject undergoes withdrawals or other indications that his body needs the substance of abuse to continue functioning.

With this understanding of what makes addiction unique, we were curious about variations between different types of addictions. Specifically, if we break addiction down into its two main classes — behavioral addictions, marked by engagement in repeated behaviors such as gambling, and substance abuse disorders, marked by abuse of substances such as drugs and alcohol — what differences do we see in the pathways and neurological bases underlying them? Here, we discuss pathways to addiction for both categories, look at neurobiological underpinnings, study computational models, and discuss exceptions that help to clarify the characteristics of addiction.

Reinforcement Learning Models of Addiction

To study the pathways behind behavioral addictions, we chose to focus on problem gambling — a disorder in which one displays persistent gambling despite obvious negative effects on one's life — hoping to better understand the various pressures and neurological underpinnings that lead to a habit of gambling that can be labeled "pathological." Before the turn of the century, much work in this area struggled to provide a quality model for addiction development that suited all types of gamblers. Blaszczynski and Nower (2001) were the first to propose a model including several pathways by which addiction might form in an attempt to better understand and better treat problem gamblers whose pathologies arose from different conditions. Each of their three pathways included environmental effects (e.g. gambling becoming available to a non-gambler), operant conditioning once gambling begins, the formation of a habit, the eventual (and inevitable) accrual of losses, and the chasing of wins that ensues. Their second and third pathways included extra background conditions that could leave someone more prone to pathological gambling — family history, depression, heightened impulsivity, or money issues, for example. The presence of multiple steps and conditions in the development of addiction, and the importance of recognizing the variety of possible backgrounds for proper treatment of problem gambling, are key to our understanding of this particular pathology.

Substance addictions are formed by fundamentally similar pathways. The initial addiction is enforced by the influx of dopamine generated by the initial drug receipt. Following this, addiction is additionally reinforced by both classical and operant conditioning. Repeated pairings between any emotional, environmental, and subjective cues and the physiological effects produced by the substances in question develop strong

associations such that the presence of such cues elicits conditioned withdrawal states or substance cravings in affected individuals. Environmental cues such as drug paraphernalia or subjective cues such as "negative mood states… may become conditioned stimuli capable of triggering craving and other drug-related conditioned responses," playing powerful roles in sustaining substance addiction (Childress et al. 1994). In terms of operant conditioning, simply achieving the intended effects of drug abuse increases the likelihood that an individual will take the same set of actions to acquire and abuse substances under similar circumstances in the future (Center for Substance Abuse Treatment 1999). The underlying mechanism driving these associations and cravings is that these stimuli generate conditioned changes in the central nervous system – the nervous system begins to expect a reward that it will not necessarily receive – which, upon lack of immediate receipt, in turn generate a craving or negative emotional state (which can itself be a stimulus/trigger), encouraging drug use (Wikler 1973). Across these dimensions of conditioning, reinforcement learning both generates and sustains substance addiction.

Neural Bases of Addiction

The brain's 'messenger' molecules are neurotransmitters and neuromodulators. They are the key to understanding how the brain communicates, both within itself and how it spreads messages throughout the body. Some of the most important such molecules related to pathological gambling are noradrenaline (excitation), serotonin (modd, sleep/wake cycle, emotional behavior), dopamine (motor control, TD reward prediction error and reinforcement, and glutamate. During casino blackjack gambling, heart rate and noradrenergic measures were shown to become more intensified in men

with gambling problems as compared to those without (Potenza 2008). Those with PG, impulsive aggression, or other clinically relevant levels of impaired impulse control were also demonstrated to have low levels of a serotonin metabolic acid, and display different behavioral and biochemical responses compared to healthy control subjects to serotonergic drugs. When a reward is expected, such as in gambling, neurons that release dopamine (as in 'dopamine hit') are activated. Glutamate, the most abundant excitatory neurotransmitter, has also been implicated in motivational processes and drug addiction, though its precise role remains unclear.

A blood oxygen level-dependent (BOLD) signal can tell us which brain regions are being activated by temporally comparing the amount of oxygen the region receives. Pathological gamblers (PGers) showed relatively less BOLD signal change in the frontal cortex, basal ganglia, and thalamic brain regions compared to recreational gamblers while viewing gambling tapes, prior to subjective motivational or emotional response onset. These findings are congruent with results from impaired impulse control studies in other behavioral domains, notably aggression (Potenza 2008). Appetitive urge, or craving states, also often immediately precede problematic behavior engagement, such as gambling for PGers or substance abuse in drug addiction.

Computational Models of Addiction

We created a computational model in an attempt to understand how wins and losses can lead to snowball effects in problem gambling based on a simple Rescorla-Wagner learning model. Our agent — let's call him Joe — starts with a pool of money, and he lives his life with two options: gamble, or don't. At every timestep, he looks at his understood value of gambling. If it's positive, he chooses to gamble. If it's

negative, he still might choose to gamble with some low likelihood (representing, perhaps, social encouragement or poor situational decision making). Wins add large sums of money to his pool, while losses detract smaller amounts. After each win or loss, Joe updates his understood value of gambling based on the difference between a reward R and his previously understood value V. The reward is calculated from $R = R_t + R_o$, where R_o is a baseline reward representing the dopamine rush Joe experiences just from the act of gambling. The model can be written

$$V_{new} = V_{old} + \eta(R - V_{old}),$$

where the value is updated every time Joe chooses to gamble. With this model, if the casino sets the win rate and the average winnings and losses to the right values, Joe will consistently lose a lot of money, even if his perceived value of gambling never becomes consistently positive. This is, of course, dependent on Joe occasionally choosing to gamble even when he views it negatively. We feel this is a natural assumption of the state of an average human being. If not, this model doesn't quite work, but casinos could win in other ways. For example, we presume that the baseline adrenaline rush of gambling R_o diminishes over time — if the casino finds a way to keep it up, or even increase it with time, Joe's perceived value could stay consistently positive. There are many, many parameters to be toyed with here, but by varying different ones, this model could be useful for understanding a person's tendency to go against their understood values, for understanding how casinos can set win rates to keep people winning *just* enough, or for understanding the impact of adrenaline and "baseline" positives on gambling addictions.

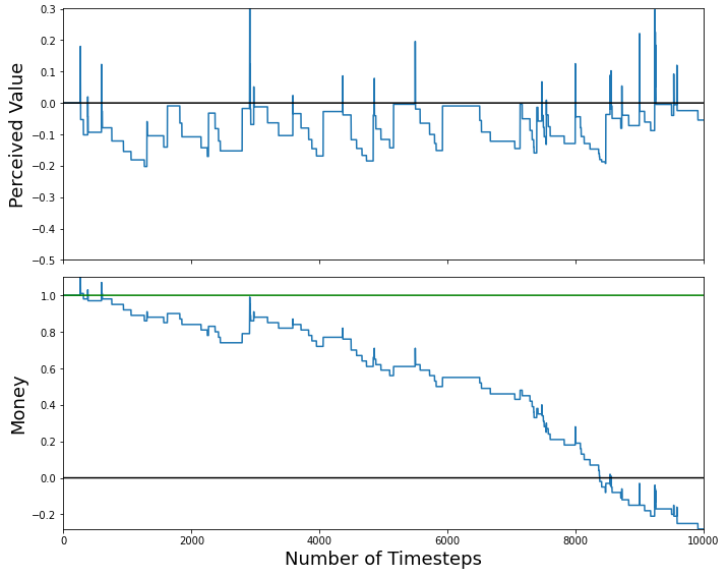

Fig. 1. Tracking Joe's perceived value of gambling and total earnings over time. He begins with a unitless monetary value of 1 and an initial perceived value $V = 0$ (neutral). The casino has set a win rate of 30%. Wins add 0.5 to Joe's perceived value, while losses subtract 0.3. This is proportional to the "monetary" value of wins and losses. There is a baseline reward, representing the adrenaline rush of the act of gambling, which starts at 0.15 and diminishes over time.

Joe will always gamble if he views it positively; when he views it negatively, he will gamble roughly 1 in 100 times. These parameters can all be varied, with different impacts on the outcome. It is difficult to characterize this "parameter space" as it is 4-dimensional (win rate, win value, loss value, rate of going against $V < 0$). Further work could find the median final V and M (money) for some large number of Joes for each possible combination of parameters, performing a sweep of parameter space to understand which values lead to significant financial losses and a pattern of gambling.

A link to the Google CoLab in which we developed this model can be found here.

Substance addiction functions similarly to behavioral addiction, but, due to the lasting impacts and neuropharmacological components of the substances involved, it is useful to model it with temporal difference (TD) learning. One key aspect of substance addiction is "over-selection" — the phenomenon of consistently choosing to take actions that lead to substance receipt over any other actions (which an external observer might perceive as having a higher value). However, the TD learning model does not inherently account for this phenomenon (Redish 2004). This is because, unlike natural rewards,

which become conditioned over time and eventually induce a release of dopamine only upon receipt of the conditioned stimulus (CS), drugs (by their inherent, neuropharmacological mechanisms, and while acting as the unconditioned stimulus (US)) generate an irreducible surge of dopamine at their receipt. This causes there to be a positive prediction error with each instance of drug receipt, increasing the value of the conditioned stimulus without bound. As the drug-related conditioned stimuli are thus ultimately perceived to have an extremely high value relative to any other natural stimuli, users will consistently "over-select" these stimuli. The model, thus, must be edited to account for this distinction and consistently generate this positive prediction error. The adjusted model can look as follows:

$$\delta = max\{\gamma^{d}[R(S_l) + V(S_l)] - V(S_k) + D(S_l), D(S_l)\}$$

where $D(S_l)$ is the dopamine surge upon entry into state S_l. This equation reduces to the normal temporal difference learning equation when there is no dopamine surge, but produces a positive prediction error if there is any dopamine surge (as induced by drugs of abuse). This makes it nearly impossible for the agent to work against or unlearn his drug-induced positive prediction error and learned values. Furthermore, as opposed to regular temporal difference learning where the values of states asymptotically approach a finite value of the total expected future reward, the values of the states leading to drug receipt increase without bound. The more times the agent takes a set of actions leading to drug receipt, the larger the value of the states leading to that sequence will be and the more likely the agent will be to select an action leading to those states again. This creates a state of perpetual learning that makes it increasingly difficult to break out of the

learning cycle and induces over-selection, perpetuating substance use and abuse (Redish 2004).

Exceptions

One population that does not fall under the umbrella of addiction or pathological gambling are professional gamblers. According to what the literature says about neural correlates, we are going to consider all sorts of professional gamblers to be part of this group, so poker players, stock traders, even art dealers who have no interest in the art itself but are focused on flipping them for a profit would all fall under this category.

The main difference is this: professional gamblers make their living by gambling and thus consider it a profession. They are skilled in the games they choose to play and are able to control both the amount of money and time spent gambling. Thus, professional gamblers are not addicted to gambling, in the same way that you wouldn't say a professional hockey player, even though they really love the sport, isn't addicted to hockey. There isn't too much literature directly comparing the two groups, but a good illustration of this is found in a 2013 study by Weinstock et al., in which they found that problem gamblers were more than 6x likelier to play the lottery, a game that is pure luck. Professional gamblers are much more calculating and less susceptible to loss of impulse control.

Counterintuitively, in individuals with a gambling problem, losing money actually triggers the rewarding release of dopamine almost to the same degree that winning does (Linnet et al. 2010). As a result, in problem gamblers, losing sets off the urge to keep playing, rather than the disappointment that might prompt a 'normal' person to walk away, thus making them fall into the trap of chasing losses. Professional gamblers

present less of the craving state, and they also 'dissociate from the result'. In other words, they break the link that our brain creates between the action and the resulting reward or lack thereof. Professional gamblers also have statistically significant higher IQ scores, though the relevancy of this statistic remains unclear. Finally, problem gamblers experience greater levels of psychopathology in comparison to the general population and professional gamblers. They experience elevated levels of psychiatric distress, more stressful events, less social support, and have lower self-esteem than the general population, which could explain the need to chase a source of dopamine and adrenaline rushes.

Conclusion

Throughout this research, we have collected and synthesized recent literature on addiction with a particular focus on pathological gambling and substance abuse. We also engaged with our existing understanding of computational models of addiction, and created our own. It appears that the neural correlates of gambling addiction mimic those of the perhaps more well-researched and well-established issue of drug addiction. Given that there are established treatments for drug addiction, a natural extension (and practical implementation) of our initial research question could be to study whether such treatments are or could be applied to other addictions such as gambling. Further research could also include differentiating habits and addictions in a manner similar to that described in the 'Exceptions' section, as well as extending and developing our model to attempt to understand the more complex dynamics of addiction as currently understood by the scientific community.

This paper represents our own work in accordance with University standards.

/s/ Katherine Belilty, Mark Dodici, Nathan Shin

Contributions

KB researched and wrote the sections describing the reinforcement learning and computational models of substance addiction. MD created the computational model of gambling and wrote the section describing it, as well as the section on the “three pathways” model of gambling addiction. NS researched and wrote the Neural Basis of Addiction and Exceptions sections, as well as the Conclusion. Topics that NS also researched but ultimately were omitted for concision and clarity included relations to Parkinson’s disease, impulse control disorders, habits, and chunking and the basal ganglia.

All authors contributed to the introduction and conclusion sections, as well as the organizing, structuring, and editing of the research information and paper itself.

Works Cited

Blaszczynski, Alex, and Lia Nower. “A Pathways Model of Problem and Pathological Gambling.” *Addiction* 97, no. 5 (2002): 487–99. https://doi.org/10.1046/j.1360-0443.2002.00015.x.

Center for Substance Abuse Treatment. Brief Interventions and Brief Therapies for Substance Abuse. Rockville (MD): Substance Abuse and Mental Health Services Administration (US); 1999. (Treatment Improvement Protocol (TIP) Series, No. 34.) Chapter 4—Brief Cognitive-Behavioral Therapy. Available from: https://www.ncbi.nlm.nih.gov/books/NBK64948/

Childress, Anna Rose, Ronald Ehrman, A.Thomas McLellan, James MacRae, Michael Natale, and Charles P. O'Brien. “Can Induced Moods Trigger Drug-Related Responses in Opiate Abuse Patients?” *Journal of Substance Abuse Treatment* 11, no. 1 (1994): 17–23. https://doi.org/10.1016/0740-5472(94)90060-4.

Linnet, J., E. Peterson, D. J. Doudet, A. Gjedde, and A. Møller. “Dopamine Release in Ventral Striatum of Pathological Gamblers Losing Money.” *Acta Psychiatrica Scandinavica* 122, no. 4 (2010): 326–33. https://doi.org/10.1111/j.1600-0447.2010.01591.x.

Potenza, Marc N. “Review. The neurobiology of pathological gambling and drug addiction: an overview and new findings.” *Philosophical transactions of the Royal Society of London. Series B, Biological sciences* vol. 363,1507 (2008): 3181-9. doi:10.1098/rstb.2008.0100

Redish, A. David. “Addiction as a Computational Process Gone Awry.” *Science* 306, no. 5703 (2004): 1944–47. https://doi.org/10.1126/science.1102384.

Weinstock, Jeremiah, Carrie E. Massura, and Nancy M. Petry. “Professional and Pathological Gamblers: Similarities and Differences” *Journal of Gambling Studies*, no. 29 (2013): 205-216. https://doi.org/10.1007/s10899-012-9308-y

Wikler, Abraham. “Dynamics of Drug Dependence: Implications of a Conditioning Theory for Research and Treatment.” *Arch Gen Psychiatry*. 1973; 28(5):611–616. doi:10.1001/archpsyc.1973.01750350005001