



Review

Behavioral and neuroeconomics of drug addiction: Competing neural systems and temporal discounting processes

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Abstract

We review behavioral- and neuroeconomic research that identifies temporal discounting as an important component in the development and maintenance of drug addiction. First, we review behavioral economic research that explains and documents the contribution of temporal discounting to addiction. This is followed with recent insights from neuroeconomics that may provide an explanation of why drug-dependent individuals discount the future. Specifically, neuroeconomics has identified two competing neural systems that are related to temporal discounting using brain-imaging techniques that examine the relative activation of different brain regions for temporal discounting. According to the competing neural systems account, choices for delayed outcomes are related to the prefrontal cortex (i.e., the “executive system”) and choices for immediate outcomes are related to the limbic brain regions (i.e., the “impulsive system”). Temporal discounting provides a useful framework for future imaging research, and suggests a novel approach to designing effective drug dependence prevention and treatment programs.

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1. Introduction

Samuel Taylor Coleridge
referring to his laudanum addiction

“My case is a species of madness, only that it is a derangement of the Volition, and not of the intellectual faculties.”

This quote, by the esteemed poet from the early 1800s, addresses that aspect of addiction that renders it amongst the most challenging of human disorders. The challenge is understanding how and why someone would make a choice knowing that this choice will later entail receipt of a negative consequence or outcome. Even more challenging is to understand why this same choice is repeatedly made with the negative consequences, in turn, being received over and over again.

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The challenges and frustration associated with this behavior have been, and are experienced by addicted individuals, individuals who know a family member or friend with addiction, and addiction scientists who seek to understand this perplexing behavior.

Fortunately, new insights from the new and emerging translational fields of behavioral- and neuroeconomics have begun to allow us to understand this behavior, and identify potential mechanisms that result in this behavior. In this paper, we review how behavioral economics permits an understanding of the function of this perplexing behavior, how neuroeconomics suggests a mechanism for this behavior (namely, the competing neural systems hypothesis of addiction), and the potential implications of this hypothesis for prevention and treatment efforts.

2. Behavioral economics

Behavioral economics characterizes the allocation of behavior within a system of constraint, with an emphasis on the conditions that influence consumption of commodities (Bickel et al., 2000). One crucial component in the behavioral economics approach is cost—be it monetary value, time to receipt, or effort necessary to obtain a commodity (Bickel et al., 1991). Here, we will focus on the time aspect of cost, and, in particular, how temporally distant events are discounted.

Temporal discounting describes a decrease in the subjective value of a commodity as a function of the amount of and delay to that commodity, or reward (Bickel and Johnson, 2003). For example, if someone would prefer \$1000 now as opposed to that same amount of money available 1 month in the future, we would refer to the temporally distant monetary amount as being discounted. The degree of discounting can be measured by using a choice procedure that is similar to those used in psychophysical experiments (Richards et al., 1997; Stevens, 1975). In such procedures, one stimulus is varied systematically while another remains constant. The task of the subject is to indicate the point at which the two stimuli are subjectively identical. Similarly, the temporal discounting procedure involves the choice between two rewards separated in time. One reward is systematically varied until preference switches from the smaller, immediate reward to the larger, later reward. For example, if the choice presented earlier varied the immediate amount (e.g., \$1000, 950, 900, etc.) and preference switched to the later choice (\$1000 in a month) when the immediate amount was \$800, we could infer that the individual making the choices discounted that amount of money by 20% in a month period. If this process was repeated at a variety of time frames (1 week, 1 month, 6 months, 1 year, etc.), then a discounting curve could be made of these switching points, which are more technically referred to as indifference points or points of subjective equality (cf. Green et al., 1994). These curves have been documented in a variety of settings to be hyperbolic in form (Bickel et al., 1999; Madden et al., 1999; Myerson and Green, 1995).

Hyperbolic discounting refers to a decrease in the value of delayed reward proportional to the delay (Ainslie and Haslam,

1992). Mazur's (1987) hyperbolic discounting equation (Eq. (1)) is used to quantify the discounting rate:

$$V_d = \frac{V}{1 + kd} \quad (1)$$

In this equation, V_d is the discounted value of an outcome, V the undiscounted value or amount, k an empirically derived constant proportional to the degree of temporal discounting and d the delay to reinforcement. The free parameter, k , provides a measure of the tendency to prefer the smaller reward to the larger, delayed reward. Eq. (1) characterizes these findings as a hyperbolic relationship between the subjective value of a reward and time until its delivery. Empirically determined hyperbolic discounting functions are described well by Eq. (1), which often has been found to account for 85% of variance, when real and hypothetical money are used with human subjects (Green et al., 1994; Johnson and Bickel, 2002; Kirby, 1997; Kirby et al., 1999; Madden et al., 1997, 2003; Myerson and Green, 1995; Rachlin et al., 1991). This equation may be employed to examine how drug-dependent individuals discount the future. Some consider this a measure of impulsive, irrational, or present-focused behaviors (e.g., the larger the parameter k , the more impulsive, irrational or present-focused the behavior). In the study by Madden et al. (1997), for example, opioid-dependent patients and non-drug-using control participants chose between hypothetical money available immediately or after a delay. Opioid-dependent patients discounted significantly more than controls and the hyperbolic curve fit, the median indifference points (see Fig. 1, top panel). Additionally, when the opioid-dependent patients chose between heroin available immediately or after a delay, delayed heroin was discounted even further when the choices involved heroin rather than hypothetical money (see Fig. 1, bottom panel).

Indeed, temporal discounting procedures show that individuals with addictions discount more (i.e., are more impulsive) than non-drug-using individuals (cf. Crean et al., 2000). Elevated temporal discounting has been observed in opioid-dependents (Kirby et al., 1999; Madden et al., 1997), cocaine-dependents (Coffey et al., 2003), problem drinkers (Petry, 2001a; Richards et al., 1999a,b), gamblers (Alessi and Petry, 2003; Petry, 2001b; Petry and Casarella, 1999), and cigarette smokers (Baker et al., 2003; Bickel et al., 1999; Mitchell, 1999), collectively demonstrating that drug-dependent individuals discount future rewards more than non-dependent controls. The similarity across types of disorders suggests that this is a behavioral process common to addiction, and it may be a process necessary for addiction to occur.

Interestingly, ex-addicts discount the future less than current addicts and in some cases not different than controls (e.g., Bickel et al., 1999), and this raises the question of whether temporal discounting is a state or a trait. There are mixed views and data regarding discounting status with respect to this distinction (cf. Bickel and Yi, 2006; Perry et al., 2005). For example, Perry et al. (2005) examined temporal discounting rates in female rats using food reinforcement. Once temporal discounting functions were obtained, the rats were grouped as either high or low for impulsivity, and were then trained to self-administer

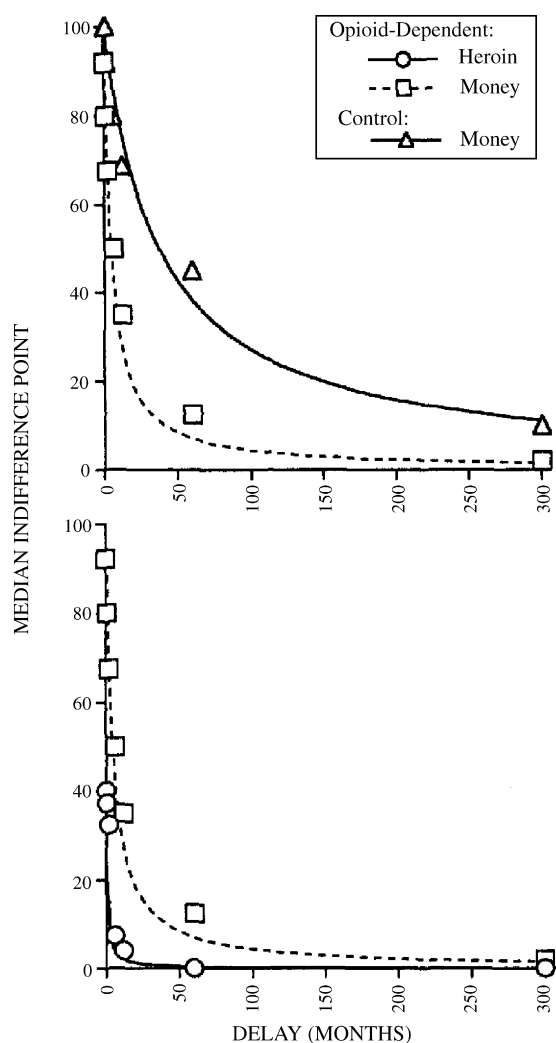


Fig. 1. Median indifference points between large delayed and small immediate heroin and monetary rewards. Opioid-dependent and control participants' monetary choice data are shown at the top, whereas the bottom shows opioid-dependent participants' monetary and heroin choice data. Reprinted from Madden et al. (1997).

cocaine. Cocaine self-administration was acquired by 77% of the rats in the high-impulsivity group, and by only 25% of the rats in the low-impulsivity group; this suggests that discounting is a trait variable. However, psychopharmacological evidence has also demonstrated that experimentally administered amphetamines are able to increase (with chronic use) or decrease (with moderate acute doses) temporal discounting among rats (Richards et al., 1999a,b), and decrease (with moderate acute doses) temporal discounting among humans (e.g., de Wit et al., 2002); this suggests that discounting is a state variable. Whether temporal discounting may be viewed as a state or trait, however, is an empirical question; temporal discounting may function as both. In either case, there would be great utility for the study of addiction in understanding the mechanisms of this behavior, which may be obtained via the application of neuroeconomics (cf. Bickel and Yi, 2006).

3. Neuroeconomics

Previous behavioral and economic research has focused on stimulus input and behavioral output, often ignoring the intermediary steps by which information is processed and decisions are made. Neuroeconomics combines the discipline of neuroscience with economics and psychology to examine brain function in decision making and choice in various circumstances (e.g., under uncertainty, in social situations). Brain imaging techniques such as functional magnetic resonance imaging (fMRI) or positron emission tomography (PET) can be used to determine areas of relative brain activation, and deduce the specific portions of the brain used, during decision making tasks.

fMRI has emerged as the preferred method for functional brain imaging, because it is non-invasive (i.e., does not require injection of any substance) and it provides greater spatial and temporal resolution than PET. fMRI works because oxyhemoglobin (hemoglobin molecules carrying oxygen) and deoxyhemoglobin (hemoglobin molecules not carrying oxygen) have different magnetic properties. The current theory of fMRI is that brain activation requires increased oxygen metabolism, which in turn results in increased blood flow to the activated regions. However, the active regions do not use all of the oxygen delivered by the blood, so the local concentration of oxyhemoglobin increases. The increased oxyhemoglobin concentration causes the fMRI signal intensity to increase in the so-called blood oxygenation level dependent (BOLD) effect in those regions in which blood flow increases due to brain activation.

Neuroeconomics provides an important scientific approach to the study of behavior because of the ability to better understand the role of the human brain in decision making. Research in the field of neuroeconomics suggests a common neural currency (i.e., utility) in the judgment of various reward types as well as other factors that affect value (e.g., reward size, temporal delay to reward, McClure et al., 2004; Montague and Berns, 2002). Neuroeconomics also suggests that decision making is an emergent process that results from the interaction (synergistically or competitively) of independent neuronal subsystems (Sanfey et al., 2006). Among the topics that neuroeconomic methods may be best suited to examine, in combination with imaging techniques, is brain functioning in optimal and suboptimal decision making.

4. Neuroeconomics of addiction: competing neural systems hypothesis

Techniques of neuroeconomics and the resulting knowledge about brain function can be applied to a diverse set of problems affecting society, including drug dependence. Bechara (2005) recently updated and expanded the role of competing brain regions for understanding the seemingly suboptimal decision making of drug-dependent individuals (see also Jentsch and Taylor, 1999). Bechara hypothesizes that the competing interaction of two neural systems may result in behavior consistent with drug dependence. One system, labeled "impulsive" includes the amygdala, nucleus accumbens, ventral pallidum, and related structures. Many sensory components converge on these sub-

cortical nuclei, and this region is thought to be essential for reacting to stimuli and initiating a physiological response, as well as decoding emotions. Studies of humans with lesions of the amygdala indicate an inability of these patients to behave in their long-term interests in the Iowa Gambling Task or develop appropriate somatic responses to risky behavior (Bechara et al., 2003); lesions to comparable areas of a rat's brain also increase impulsive choice behavior (see Winstanley et al., 2003, 2004; Kheramin et al., 2002; Cardinal et al., 2001). Observations of this sort have led to the hypothesis that the amygdala is an important set of nuclei in signaling the valence (positive or negative) of immediate outcomes (Bechara, 2005; Bechara and Damasio, 2005; McClure et al., 2004), with the immediate valence of drug dependence being largely positive.

The competing system is labeled “reflective” by Bechara (2005) and includes the prefrontal cortex (PFC). We prefer, however, the term “executive” and will use it hereafter. The PFC, located on the anterior part of the frontal lobes, is a part of neocortex, an evolutionarily younger brain region found in humans and higher mammals. The PFC is generally thought to be involved in executive functions such as working toward a defined goal, prediction and expectation of outcomes, determining future consequences of current activities, and social control (Barkley, 1997). Lesions to portions of the PFC lead to significant impairments in decision making, resulting in maladaptive behavior that often leads to negative outcomes including social and family problems, as well as money loss. The PFC is likely the location where information from various sources (e.g., nature of stimulus, affect associated with stimulus, memory of previous events; see Bechara et al., 2003) converges to make decisions regarding the behavior necessary to achieve current goals (McClure et al., 2004). Individuals with ventromedial prefrontal cortex (VMPFC) damage, consistently make the same mistake because they cannot incorporate negative feedback from previous behavior into occurrence of future behavior (Bechara and Damasio, 2005). Because VMPFC is the region that stores and signals value of future consequences (the somatic marker hypothesis, Bechara and Damasio, 2005; Glimcher and Rustichini, 2004), damage to VMPFC results in behavior that relies solely on input from other regions (e.g., the amygdala). Consistent with this view, lesions of the PFC prevent appropriate somatic responses to affect-laden, internally generated images (Bechara et al., 2003). Bechara (2005) hypothesizes that the PFC is responsible for signaling the valence of future outcomes.

According to Bechara, the competing impulsive and executive neural systems (referred to henceforth as a competing brain regions hypothesis), could explain addiction as follows: a hyperactive impulsive system (e.g., amygdala) may weaken the relative influence of the executive system (e.g., PFC), and drug dependence results when the impulsive system overwhelms the executive system, with corresponding emphasis on immediate consequences. This is consistent with the previously reviewed literature that has found that drug-dependent individuals temporally discount rewards more than non-dependent controls.

McClure et al. (2004) made that relationship explicit when they examined the discounting of delayed rewards in college

students. Specifically, they proposed that differential activity in the limbic and fronto-parietal regions might be directly related to whether a smaller sooner, or a larger later reward will be selected. To test this hypothesis, participants were asked to perform a temporal discounting task during an fMRI scan. The results showed greater activity in limbic areas for choices that included an immediate reward and, conversely, greater activity in frontal and parietal regions when participants chose the later reward. These two regions may be considered in the context of Bechara's proposed “separate, but interacting” neural systems, in which the limbic area corresponds to the impulsive system and the frontal and parietal regions correspond to the executive system.

Conceptually, Bechara's separate but interacting regions idea, and McClure's validating experiment provide a new way to look at temporal discounting. For instance, the excessive temporal discounting exhibited by individuals with drug addictions (Bickel et al., 1999) may in part be explained by evidence that many drugs produce neurological changes that decrease regions of the cortex associated with the executive system (Verdejo-García et al., 2004). If the choice of a delayed option compared to a more immediate option is related to increased activity in the executive system as McClure et al. (2004) indicated, then it might be expected that individuals who have decreased activity in their executive system might make fewer choices for delayed rewards. The possibility that damage to, or dysfunction of the executive system increases discounting of future rewards is consistent with the observation that certain tasks which challenge executive functioning are able to produce steeper discounting among individuals that are otherwise more likely to choose delayed outcomes (cf. Hinson et al., 2003).

Increased discounting of future rewards among individuals that have been diagnosed with particular psychological disorders may also be explained by this new view of temporal discounting. For instance, increased rates of temporal discounting among teenagers and children who were diagnosed with attention deficit hyperactivity disorder (ADHD; e.g., Barkley et al., 2001) could be explained by the relative difficulties with executive functioning that arise with ADHD. In addition, individuals diagnosed with antisocial personality disorder (ADP) also tend to exhibit greater discounting of delayed rewards than controls, which could be related to relatively weak activation in their executive system (Petry, 2002). Indeed, prefrontal cortex dysfunction has been implicated in these and other disorders, particularly those that, like ADHD, primarily affect young people. Interestingly, the prefrontal cortex has been shown to undergo developmental changes well into the 20s (Giedd, 2004). Adolescents appear to be particularly vulnerable to substance abuse, and this has been hypothesized to be partly due to the ongoing maturation of the prefrontal cortex (see, for example, Chambers et al., 2003). In this context, impulsivity results from the “executive” system not being fully developed. This, in turn, suggests that dysfunction of the “executive” system is exacerbated in adolescence by immaturity as well as the brain changes associated with the pharmacological effects of addictive drugs. According to Kalivas and Volkow (2005) the release of dopamine (possibly an important biochemical connection between the “impulsive” and “execu-

tive” regions) following consumption serves as the initiating step in a cascade of molecular and cellular events that eventually result in the prefrontal cortex attaching extreme value to addictive drugs (also see Redish, 2004). Eventually, prefrontal regulation of behavior is reduced, which leads to the impaired decision making observed in adolescent and adult addicts; the molecular changes that occur following chronic dopamine stimulation cause the “executive” region to be overwhelmed by stimuli from the “impulsive” region. Developmental theories of “impulsive” and “executive” functioning and neuropharmacologic effects of specific drugs have much to contribute to the competing neural systems hypothesis, but given the vast literature on the topics, further discussion is beyond the scope of this paper.

5. Conclusion and implications

The observation that (1) the addicted discount the future more than controls and (2) the shortened temporal focus appears to result from a concomitant increase in activation in the impulsive brain regions and a decrease in activation in the executive brain regions provides a novel basis for understanding addiction. Heretofore, our efforts to research and treat addiction tend to consider these as a unitary phenomenon and, as such, the solution to addiction was to identify and alter the central element. This new competing brain region hypothesis provides a radically different view—it suggests that addiction is the result of the interaction between two neural systems and as such it provides two complementary targets for intervention (cf. Bickel and Potenza, 2006). If this hypothesis is empirically confirmed and replicated, then, in our opinion, it may radically alter our understanding and efforts to treat addiction. Here, we will briefly review the implication of this view for (1) developing new prevention and treatment approaches; (2) evaluating treatment already developed; (3) provide a novel basis for treatment and prevention matching; and (4) suggest the importance of supporting different cultural practices and policies. Given that discounting has some state-like features, note that discounting might both serve as an index to assess the extent of the addiction by summarizing relative control of these two brain regions and provide a useful measure of the success of different interventions.

The competing brain region hypothesis suggests that we need to develop treatments that will (1) decrease activation of impulsive brain regions and (2) increase activity in executive brain regions. As such, this may mean that successful treatment will require the development of two separate treatments, one for each brain region. Whether these treatments could be medications or behavioral therapies or some combination of the two will remain to be seen. Moreover, the competing brain region hypothesis permits consideration of entirely new treatment approaches. For example, perhaps through biofeedback from brain activation, we might be able to increase activity of the frontal brain region and increase its participation in behavior relative to the impulsive brain regions. If so, this treatment might change addictive behaviors. Similarly, prevention programs might be developed that strengthen executive brain regions and/or constrain activity in impulsive brain regions.

Currently existing treatment could be evaluated from the perspective of the competing brain-region hypothesis of addiction. Perhaps, treatments with documented efficacy operate largely at only one or the other of these brain regions. For example, contingency management might decrease addiction by working via impulsive brain regions, while cognitive-behavioral therapies might operate by strengthening executive brain regions. This would suggest, of course, that these two treatments together might produce better outcomes than either alone. Similar considerations would be relevant to prevention efforts. Additionally, the examination of the components of current treatments might show one or more of those components to be ineffective in altering either relevant brain region, and perhaps then those treatment components could be appropriately dropped from utilization and decrease cost and enhance efficiency of treatment.

Another important implication of the competing brain-region hypotheses is the possibility of providing an empirical basis for treatment matching. Patients with more excessive impulsive brain activity than diminished executive brain activity may require a different treatment than patients who have more diminished executive brain activity than excessive impulsive brain activity. Of course, prevention matching could be considered along these same lines.

Finally, there may be value in considering whether our current cultural practices and our current policies tend to support, if not reinforce, short-term considerations (Bickel and Marsch, 2000). If so, does that diminish some aspects of executive functioning? Do cultural practices of immediate gratification strengthen impulsive brain activation? If so, are we rendering those in our population more susceptible to addiction? These challenging questions are worth considering, as is the value of developing practices and policies that support greater consideration of the future and self-control.

In conclusion, the relatively new fields of behavioral- and neuroeconomics working in conjunction may lead to new empirical findings of our subject matter, namely, addiction. In turn, these new findings may suggest a new conceptual reframing of what is relevant for understanding and identifying the mechanisms of addiction. A new conceptual framework immediately suggests new ways to treat and prevent addiction as outlined above. Together, the application of these approaches to addiction are full of promise, and deserve appropriate scientific attention and skepticism, and by testing of these new approaches our field may develop prevention and treatment programs that may lead us to more fully resolve this serious public health problem.

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