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# Changing Delay Discounting in the Light of the Competing Neurobehavioral Decision Systems Theory: a Review

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## Abstract

Excessively devaluing delayed reinforcers co-occurs with a wide variety of clinical conditions such as drug dependence, obesity, and excessive gambling. If excessive delay discounting is a trans-disease process that underlies the choice behavior leading to these and other negative health conditions, efforts to change an individual's discount rate are arguably important. Although discount rate is often regarded as a relatively stable trait, descriptions of interventions and environmental manipulations that successfully alter discount rate have begun to appear in the literature. In this review, we compare published examples of procedures that change discount rate and classify them into categories of procedures, including therapeutic interventions, direct manipulation of the executive decision-making system, framing effects, physiological state effects, and acute drug effects. These changes in discount rate are interpreted from the perspective of the competing neurobehavioral decision systems theory, which describes a combination of neurological and behavioral processes that account for delay discounting. We also suggest future directions that researchers could take to identify the mechanistic processes that allow for changes in discount rate and to test whether the competing neurobehavioral decision systems view of delay discounting is correct.

### Keywords

delay discounting; behavioral neuroscience; impulsivity; executive function; human

The effective value of a reinforcer has long been known to be inversely correlated with the delay to its receipt (e.g., Hull, 1943). More recently, precise mathematical models have been developed to quantify the relationship between the delay to the receipt of a reinforcer and its present value (Mazur, 1987). Hyperbolic (Mazur) or hyperbolic-like models (e.g., Green & Myerson, 2004) have been shown to best describe this relationship, the most common of these in behavioral psychology being Mazur's hyperbolic discounting model

$$V=\frac{A}{1+kD} \quad (1)$$

where $V$ is the present value of a reinforcer, $A$ is the amount of the reinforcer, $D$ is the delay to the receipt of the reinforcer, and $k$ is a free parameter that quantifies the rate at which the reinforcer is devalued as a function of delay. Behavioral psychologists often use psychophysical titration procedures to evaluate the present value of a reinforcer at a series of delays (e.g., Du, Green, & Myerson, 2002). The value of $k$ can then be straightforwardly and

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accurately computed and the rate of discounting for individuals or groups of individuals can be compared. Many neuroscientists favor a double exponential model of discounting (e.g., McClure, Ericson, Laibson, Loewenstein, & Cohen, 2007), whereas economists often assess discounting by simply asking individuals how much a delayed commodity is worth or by asking how much increasing or decreasing the delay to the consumption of a commodity decreases or increases the commodity's value (Loewenstein, 1988).

A sizable body of research has shown that discount rate is an important predictor or correlate of choice behavior in a wide variety of contexts. The most thoroughly documented correlation is between drug dependence and discount rate. Almost without exception, drug-dependent populations discount delayed reinforcers at higher rates than matched controls (see Bickel & Marsch, 2001 for review; Reynolds, 2006). This is true for drug-dependent populations across drug classes, with the possible exception of marijuana dependence (Johnson et al., 2010). Higher than normal discount rates, however, are not limited to drug-dependent populations. A growing body of research suggests that excessive rates of discounting are associated with numerous unhealthy behaviors such as overeating (Weller, Cook III, Avsar, & Cox, 2008) and gambling (e.g., Petry & Casarella, 1999). The observation that excessive discount rates are associated with a myriad of problematic behavior patterns has led some to hypothesize that discount rate may be a trans-disease process related to a variety of disorders (see below).

With delay discounting involved in so many health-related behaviors, researchers have sought to determine whether discount rate is a cause of, or simply a correlate or result of, unhealthy decision-making. Additionally, reports of treatments or manipulations that alter discount rate are increasingly appearing in the literature. Both the causal influences of discount rate and the potential to modify discount rate deal with the question of whether discount rate is a trait or state variable. In other words, is discount rate a result of the current environment or is it a relatively immutable pattern of behavior (Odum, 2011)?

Research suggests that discount rate may typically be regarded as a stable trait, but is mutable under certain circumstances. Discount rate has high test–retest reliability for periods of up to one year (Baker, Johnson, & Bickel, 2003; Beck & Triplett, 2009; Black & Rosen, 2011; Kirby, 2009; Ohmura, Takahashi, Kitamura, & Wehr, 2006; Simpson & Vuchinich, 2000; Takahashi, Furukawa, Miyakawa, Maesato, & Higuchi, 2007), suggesting that discount rates are stable over short to medium timeframes. In addition, individuals' discount rates are correlated across commodities (Bickel, Landes, et al., 2011; Odum, 2011), further suggesting that discount rate is a trait variable. Despite this evidence, various environmental manipulations increase or decrease discount rate, which we describe in detail in this review. The fact that a variety of manipulations alter discount rates suggests that, although discount rate may be regarded as a trait under many circumstances, it may be a fruitful target for intervention. Given the expansive list of unhealthy behaviors associated with discount rate, such attempts are arguably important pursuits.

The results of published experiments to date implicate delay discounting in a wide variety of problematic health behaviors, but the vast majority of these studies are correlational. Therefore, a complete understanding of the role of discount rate in the development or treatment of these associated disorders and behavioral patterns is largely unknown. Furthermore, little is understood about effective ways to alter discount rate, and the mechanisms by which such treatments and manipulations function. Understanding how discount rate interacts with decision-making in individuals that engage in problematic health behaviors is crucial, especially with respect to the impact of altering discount rate on the future choices an individual makes.

Efforts to move beyond correlational studies are important to addressing these issues. For example, high discount rates could cause individuals to make choices that result in poor health; thus changing discount rate will change these choices, resulting in improved health. However, some other characteristic associated with discount rate (e.g., socioeconomic status; Green, Myerson, Lichtman, Rosen, & Fry, 1996; Jaroni, Wright, Lerman, & Epstein, 2004) could cause individuals to make unhealthy choices. In this case, altering discount rate would not affect the underlying problem (low socioeconomic status) or modify the unhealthy behavior. Moving beyond correlational study designs and/or conducting longitudinal experiments (Audrain-McGovern et al., 2009) will help address these issues.

Future studies that manipulate environmental variables that alter discount rate and measure collateral changes in discounting-associated health behavior could imply causality in some of these relationships. The purpose of this review is to classify and compare research on environmental manipulations shown to alter discount rate. To stay at a manageable length, we limit our consideration to research on humans. We make these classifications in light of evidence that excessive rates of delay discounting may be a trans-disease process that underlies a variety of health-related behaviors (Bickel, Jarmolowicz, Mueller, Koffarnus, & Gatchalian, 2012; Bickel & Mueller, 2009), and that discount rate may be a summary measure relevant to the competing neurobehavioral decision systems (CNDS) theory (see Bechara, 2005; Bickel, Jarmolowicz, Mueller, & Gatchalian, 2011; Bickel et al., 2007; Jentsch & Taylor, 1999). Taking all of these factors into consideration, we propose directions that researchers in this area could pursue to refine our understanding of the role of discount rate in the origin and expression of problematic behavior patterns.

## Delay Discounting as a Trans-disease Process

### Trans-disease Processes

The question of whether discount rate is a trans-disease process is critical for our present purposes and for the study of decision-making in general. A trans-disease process is one that operates across a range of disorders. For example, stress has been implicated as an underlying cause or exacerbating factor in a number of disorders with seemingly unique etiologies (Sapolsky, 2005). If discount rate serves as a summary measure of decision-making processes that underlie a range of impulse-control disorders, then the potential value of altering an individual's discount rate becomes immediately apparent. The evidence to date, summarized here (see Bickel et al., 2012; Bickel & Mueller, 2009 for a more detailed discussion), suggests discount rate may serve this role.

There are numerous advantages to seeking out and intervening on trans-disease processes. If a process is the underlying cause of multiple disorders, therapeutic approaches that address that process should result in therapeutic gain across the range of resulting disorders. Researchers studying trans-disease processes would also benefit from findings in other content areas where the same process was implicated. A trans-disease perspective may also demystify comorbidity (i.e., the presence of two of more disorders). Comorbidity is a somewhat puzzling phenomenon if diseases are seen as distinct entities, but is neither surprising nor difficult to understand from the trans-disease process view. Specifically, if the same process contributes to multiple disorders, then the likelihood of these disorders co-occurring would increase.

This notion of trans-disease processes is, in many ways, consistent with the behavior analytic approach. For example, behavior analysis interprets processes such as reinforcement, punishment, and stimulus control as generalized principles that apply across diverse species, organisms, response forms, and occasions. Moreover, disorders such as autism (Drash & Tudor, 2004), depression (Dougher & Hackbert, 1994; Kanter, Busch,

Weeks, & Landes, 2008), and addiction (Bickel et al., in press; Bickel, Jarmolowicz et al., 2011) have been described as disruptions in normal behavioral processes (cf., Sidman, 1960). For behavior analysts, embracing the notion of trans-disease processes may be nothing more than an extension of the notion of generalized principles to the domain of disease and disorder.

## Is Excessive Delay Discounting a Trans-disease Process?

Strong evidence for the position that excessive discount rate may be an example of a trans-disease behavioral process can be found in human behavioral pharmacology (for review, see Madden & Bickel, 2010). For example, individuals addicted to alcohol (Bjork, Hommer, Grant, & Danube, 2004; Bobova, Finn, Rickert, & Lucas, 2009; Finn & Hall, 2004; Mitchell, Fields, D'Esposito, & Boettiger, 2005; Petry, 2001a), cigarettes (Baker et al., 2003; Bickel, Odum, & Madden, 1999; Bickel, Yi, Kowal, & Gatchalian, 2008; Businelle, McVay, Kendzor, & Copeland, 2010; Johnson, Bickel, & Baker, 2007; Mitchell, 1999; Odum, Madden, & Bickel, 2002; Reynolds, Leraas, Collins, & Melanko, 2009; Reynolds, Richards, Horn, & Karraker, 2004; Rezvanfard, Ekhtiari, Mokri, Djavid, & Kaviani, 2010), cocaine (Allen, Moeller, Rhoades, & Cherek, 1998; Bickel, Landes et al., 2011; Camchong et al., 2011; Coffey, Gudleski, Saladin, & Brady, 2003; Heil, Johnson, Higgins, & Bickel, 2006; Kirby & Petry, 2004; Moeller et al., 2002; Petry & Casarella, 1999), methamphetamine (Monterosso et al., 2007), and opioids (Kirby & Petry, 2004; Kirby, Petry, & Bickel, 1999; Vassileva, Georgiev, Martin, Gonzalez, & Segala, 2011) all discount delayed reinforcers more rapidly than do non-using controls. Moreover, consistent with the notion that a common process contributes to the abuse of these substances, polysubstance abuse (i.e., comorbidity) is relatively common (Agrawal, Lynskey, Madden, Bucholz, & Heath, 2007).

Excessive rates of delay discounting also appear to be related to other types of problematic behavior. Numerous studies have linked high rates of delay discounting to pathological and/or problem gambling (Dixon, Marley, & Jacobs, 2003; Ledgerwood, Alessi, Phoenix, & Petry, 2009; MacKillop, Anderson, Castelda, Mattson, & Donovick, 2006; Petry, 2001b; Petry & Casarella, 1999). Interestingly, not only is comorbidity between pathological gambling and substance abuse relatively common (e.g., Petry, 2001b; Petry & Casarella, 1999), rates of discounting are even higher in pathological gamblers with substance abuse disorders than they are in pathological gamblers without substance abuse disorders (Petry, 2001b). This suggests that individuals with the highest rates of discounting may be particularly vulnerable to both substance abuse disorders and pathological gambling.

Obesity is also associated with discount rate in some populations. For example, obese women have higher discount rates than healthy-weight women, but this relation was not replicated with obese and healthy-weight men (Weller et al., 2008). Many factors (e.g., overeating, poor diet, lack of exercise, stress, metabolic issues, etc.) can contribute to obesity (Epstein, Leddy, Temple, & Faith, 2007; Epstein, Salvy, Carr, Dearing, & Bickel, 2010), making the link between obesity and patterns of problematic behavior somewhat indirect. The link between delay discounting and problematic patterns of eating, however, was somewhat clarified by Davis, Patte, Curtis, and Reid (2010), who compared discount rates in obese women with binge eating disorder, obese women without binge eating disorder, and healthy-weight women. They found that only obese women with binge eating disorder had significantly higher discount rates than healthy-weight women. Interestingly, akin to what has been shown with gambling and substance abuse (Petry, 2001b), obese smokers tend to discount at higher rates than healthy-weight smokers (Fields, Sabet, Peal, & Reynolds, 2011), further suggesting that individuals with the highest rates of discounting are at increased risk of developing comorbid disorders.

Higher than normal rates of delay discounting are also seen across a range of developmental and psychiatric disorders including attention deficit hyperactivity disorder (Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001; Scheres, Tontsch, Thoeny, & Kaczkurkin, 2010; Wilson, Mitchell, Musser, Schmitt, & Nigg, 2011), schizophrenia (Heerey, Robinson, McMahon, & Gold, 2007; MacKillop & Tidey, 2011), borderline personality disorder (Bornovalova, Lejuez, Daughters, Zachary Rosenthal, & Lynch, 2005), depression (Yoon et al., 2007), social anxiety disorder (Rounds, Beck, & Grant, 2007), and disruptive behavior disorder (Swann, Bjork, Moeller, & Dougherty, 2002). As noted above in regard to addiction, gambling, and obesity, these disorders are highly comorbid with other discounting-linked patterns of aberrant behavior. For example, ADHD is frequently comorbid with cigarette smoking (e.g., Lambert & Hartsough, 1998; Laucht, Hohm, Esser, Schmidt, & Becker, 2007), obesity (Davis, 2010), problem gambling (Grall-Bronnec et al., 2011), and drug addiction (Carpentier, van Gogh, Knapen, Buitelaar, & De Jong, 2011).

Excessive discount rates have also been linked with a range of behavior patterns that are not necessarily considered disorders, but often result in poor health nonetheless. For example, Odum, Madden, Badger, and Bickel (2000) found that heroin addicts reporting a willingness to share needles tended to discount delayed money more rapidly than heroin addicts that did not report being willing to share needles. The association between discount rate and HIV risk behaviors like needle-sharing (Odum et al., 2000) may help explain why individuals infected with HIV (Dierst-Davies et al., 2011) or Hepatitis C (Huckans et al., 2011) tend to discount delayed reinforcers more rapidly than noninfected controls. Early onset of sexual activity in teenagers is associated with discount rate (Chesson et al., 2006), as is failing to engage in a wide variety of prohealth behaviors such as eating breakfast, wearing sunscreen, wearing a seatbelt, exercising vigorously, checking blood pressure, following physician advice, or having a recent mammogram, Pap smear, prostrate examination, dental visit, cholesterol test, or flu shot (Axon, Bradford, & Egan, 2009; Bradford, 2010).

This broad base of findings, from both within and outside of behavioral psychology, suggests that delay discounting is a trans-disease process. Adopting this view elevates the importance of understanding and developing treatments that alter rates of delay discounting.

## The Competing Neurobehavioral Decision Systems Model

Recent advances in neuroeconomics—an emerging interdisciplinary field that merges behavioral economics, neuroscience, cognitive psychology, and social psychology (Glimcher, Camerer, Poldrack, & Fehr, 2008) to understand human decision-making—have provided novel insights into neural mechanisms associated with the discounting of delayed reinforcers. For example, McClure, Laibson, Loewenstein, & Cohen (2004) had 14 undergraduate students make choices between reinforcers that varied in both their immediacy (ranging from today to 6 weeks from now) and magnitude (ranging from $5 to $40). Functional magnetic resonance imagining (fMRI) during the decisions revealed that choices for immediate reinforcers were associated with relatively high levels of activation in limbic and paralimbic areas (i.e., the ventral striatum, the medial orbitofrontal cortex, medial prefrontal cortex, posterior cingulate cortex) whereas choices for delayed reinforcers were associated with relatively high levels of activation in lateral prefrontal brain regions (i.e., the dorsolateral prefrontal cortex, the ventrolateral prefrontal cortex, and the lateral orbitofrontal cortex).

Work by Knutson and colleagues (Ballard & Knutson, 2009; Knutson, Taylor, Kaufman, Peterson, & Glover, 2005) suggests the involvement of these neural systems in intertemporal choices is related to the attribution of reinforcer value. When delay and amount are presented at separate times, many of the same lateral prefrontal regions are associated with

delay magnitude, while limbic and paralimbic areas are associated with absolute magnitude of the reinforcers. In combination, these studies suggest that these two brain networks participate in the attribution of value to response options in intertemporal choice procedures, thereby affecting choice behavior.

This demonstration that valuation of and choices for immediate and delayed reinforcers are associated with activation in two distinct neural systems (Ballard & Knutson, 2009; McClure et al., 2004) is consistent with a number of dual-system models of decision-making (Bechara, 2005; Bickel, Jarmolowicz, et al., 2011; Bickel et al., 2007; Jentsch & Taylor, 1999; Kahneman, 2011). The CNDS model posits that decision-making reflects the relative balance in activation between two interacting neurobiological systems (Bickel et al., 2007). The evolutionarily older *impulsive system*, made up of portions of the limbic and paralimbic regions (the amygdala, nucleus accumbens, ventral pallidum, and related structures), values immediate reinforcers. By contrast, the more recently evolved executive system, made up of portions of the prefrontal cortex, may be needed for the inhibition of the impulsive system and the associated valuation of delayed reinforcers. The theory posits that relative activation of these two decision systems is associated with behavior in delay discounting procedures, and, by extension, with clinically relevant choice scenarios (e.g., whether to ingest a drug of abuse). If the theory is correct, interventions or treatments that “strengthen” (i.e., increase neuronal firing and regional blood flow) the lateral prefrontal cortices, for example, should be associated with an increased subjective valuation attributed to delayed rewards and resulting increased choice of delayed rewards.

## Support for the Competing Neurobehavioral Decision Systems Model

In addition to McClure, York, and Montague (2004), an array of findings supports the CNDS model. For example, other fMRI studies have demonstrated that choices for immediate reinforcers are associated with activation in the impulsive system (Bickel, Pitcock, Yi, & Angtuaco, 2009b; Kable & Glimcher, 2007, 2010; McClure et al., 2007; Monterosso et al., 2007; Xu, Liang, Wang, Li, & Jiang, 2009) and this activation decreases as the delay to the reinforcer increases (Kable & Glimcher, 2007, 2010; Yan, 2009). Thus, immediate reinforcers may preferentially and systematically engage the impulsive system. Additionally, numerous studies have replicated the finding (McClure et al., 2004) that choices of delayed reinforcers are associated with relatively high levels of activation in the executive system (e.g., Bickel, Pitcock, Yi, & Angtuaco, 2009a; e.g., Bickel et al., 2009b; Hoffman et al., 2008; McClure et al., 2007; Meade, Lowen, Maclean, Key, & Lukas, 2011; Monterosso et al., 2007; Xu et al., 2009; see Kable & Glimcher, 2010 for a contrasting view). Taken together, these fMRI studies support the notion that the CNDS model describes the brain regions correlated with choices for immediate and delayed reinforcers.

Although findings regarding the neural correlates of delay discounting are relatively consistent, causal inference cannot be made from correlational data. Causal inference regarding the brain regions responsible for elevated rates of delay discounting would require observations of alterations in discount rate as brain states were systematically manipulated. Fortunately, recent advances in behavioral neuroscience technology enable the direct and relatively selective manipulation of brain states. For example, transcranial magnetic stimulation (TMS) is an emerging technology that uses noninvasive electromagnetic waves to temporarily increase or decrease neural activation in specified brain regions. Using TMS, researchers have identified brain regions associated with a wide range of complex behaviors such as remembering (Kaminski, Korb, Villringer, & Ott, 2011; Mottaghy et al., 2000; Soto, Rotshtein, Hodsoll, Mevorach, & Humphreys, 2011; Zanto, Rubens, Thangavel, & Gazzaley, 2011), behavioral flexibility (Moser et al., 2002) and, more recently, delay discounting (Cho et al., 2010; Figner et al., 2010). Consistent with the CNDS model, two

studies (Cho et al.; Figner et al., see below for summaries) suggest that the dlPFC modulates discount rate. The deep-brain location of the impulsive system, however, precludes its manipulation via TMS, making information on the impulsive system's role in human delay discounting somewhat more speculative.

The brain imaging and brain stimulation evidence is supplemented by a number of related findings. First, brain structure abnormalities accompany the higher than average discount rates seen in drug addicts. For example, Lyoo et al. (2006) found that opioid-dependent individuals had less gray matter in areas of the executive system (e.g., the prefrontal cortex) than did healthy controls (also see Bjork, Momenan, & Hommer, 2009). A similar shortage in gray matter was not seen in the impulsive system. Because opioid-dependent individuals discount delayed reinforcers more rapidly than healthy controls (Madden, Petry, Badger, & Bickel, 1997), these findings suggest that discounting may relate to structural abnormalities in the brain. Future research should investigate this possibility. Second, concurrent performance of other executive-system-dependent tasks (e.g., remembering) increases rates of discounting. For example, Hinson, Jameson, and Whitney (2003) found that discount rates were higher when college students were required to remember and subsequently report on a string of numbers while performing a delay-discounting task than when no such remembering was required. This finding suggests that an executive system taxed through use in other executive-system-dependent tasks does not effectively compete with the impulsive system. Third, interventions that strengthen other executive-system-dependent performance also decrease rates of discounting. For example, working memory training decreases discount rates in stimulant-dependent individuals (Bickel, Yi, Landes, Hill, & Baxter, 2011; see description below).

## Competing Neurobehavioral Decision Systems Model as a Dimensional View of Behavioral Dysfunction

The CNDS model posits that individuals are at risk of developing behavioral maladies when the relative strength of the impulsive system reliably exceeds the relative strength of the impulsive system (Bickel, Jarmolowicz, et al., 2011). There are, however, many ways that this relative balance could be disrupted. Figure 1 illustrates the logic of this dimensional approach. For example, an individual with a low-strength executive system and a medium-strength impulsive system (X on Figure 1) would run the same risk of developing behavioral maladies as an individual with a medium-strength executive system and a high-strength impulsive system (O on Figure 1). These two scenarios, however, may suggest two completely different approaches to restoring regulatory balance between the competing decision systems. For example, because the individual in scenario "X" has a weak executive system, there is a large potential to improve executive system performance. As a result, techniques that strengthen executive system performance may be maximally effective (see below). By contrast, the individual in scenario "O" already has a medium strength executive system, leaving less room to improve executive system performance. This particular type of competing decision systems dysregulation may be more effectively remediated by decreasing the influence of the impulsive system (see below).

This dimensional view may spur the development of individualized approaches to restoring regulatory balance to these competing decision systems. Although this personalized approach to behavioral maladies is consistent with the individualized approach utilized across various areas of behavioral application (e.g., Iwata, Dorsey, Slifer, Bauman, & Richman, 1994), it may represent an expansion of these sorts of personalized approaches into the domain of behavioral risk factors. This expansion is consistent with a recent call for a greater focus on the behavior-analytic approach to individual differences (Williams, Myerson, & Hale, 2008).

### Study Selection and Summary Procedure

In this review, we have included any human-participants study that reported a change in rate of delay discounting after an experimental manipulation. Presumed changes in discount rate accompanying an extra-experimental event (e.g., development of a drug addiction) were not included. We searched academic databases (e.g., PubMed, Web of Science, Google Scholar) with a variety of search terms, and also searched the reference list of any candidate studies produced by these initial searches.

Although an increasing number of researchers have reported experimentally induced changes in discount rate, the types of manipulations used and methods for assessing discount rate have varied widely. This variability in experimental design makes direct comparisons between studies difficult. To aid in such comparisons, whenever possible we have computed a common measure of effect size (Cohen's *d*, see Cohen, 1988) from the published manuscripts discussed below (Table 1). Cohen's *d*, the number of pooled standard deviations that separate the means of two groups (Cohen, 1988; Rosnow and Rosenthal, 1996), was chosen as it is calculable from a wide variety of experimental designs and with relatively little descriptive data.

Since the focus of all these studies was experimentally induced changes in discount rate, most used within-subject designs. There are opposing views regarding the correct way to compute Cohen's *d* with correlated designs. Calculating *d* from group means and standard deviations is recommended by Dunlop, Cortina, Vaslow, & Burke (1996), while using the *t* or *F* statistic is recommended by Rosenthal (1991). This first approach does not take repeated measures correlations into account and results in relatively lower effect sizes than the second approach which does incorporate intrasubject correlation. We used whichever approach was possible from the available data, although we used the *t* or *F* statistic when both that and the group means and standard deviations were reported. Cohen (1988) has defined a small effect as $d \geq 0.2$, medium as $d \geq 0.5$, and large as $d \geq 0.8$.

## Variables that Impact Discount Rate

### Therapeutic Interventions

Of the growing number of manipulations shown to affect discount rate, four studies have reported therapeutic interventions that altered discount rate (Bickel et al., 2011; Black & Rosen, 2011; Landes, Christensen, & Bickel, in press; Yi et al., 2008). Although these interventions were quite different from one another, each decreased discount rates in their respective active treatment groups. In the CNDS view, therapeutic interventions likely act by enhancing the inhibitory control that the executive system exercises over the impulsive system, although this notion remains untested.

The therapeutic intervention that most straightforwardly targeted the executive system was working memory training (Bickel, Yi, et al., 2011). Individuals in treatment for psychomotor stimulant addiction were assigned to either an active training group (n = 14) or a yoked control training group (n = 13) that was matched on the basis of gender and score on a working memory task (i.e., letter–number sequencing). Working memory training was administered via four modules of a commercially available computer-based memory-training task (PSSCogReHab, Krabbendam & Aleman, 2003). For control training, the programs had been modified such that they presented all of the same stimuli, but indicated to the participant the correct response to the working memory task, preempting the need for the participant to use their working memory to respond correctly. The difficulty of the training programs in the active group progressively increased so working memory would continue to be taxed in each session, and performance was reinforced with monetary bonuses when accuracy was sustained or improved in consecutive sessions. Participants in

the control training condition received compensation in amounts and at times that matched the active training participant to whom they were yoked. The training phase was terminated before the end of 15 sessions for an active training participant and their yoked control participant if the active participant failed to improve their performance on any two programs for three consecutive training sessions (range 4 to 15 sessions). A battery of assessments including some to measure working memory and delay discounting were administered prior to and after the training phase of the experiment. Results indicate that although none of the other pre- and post-training assessment measures in this study were significantly affected by working-memory training, participants in the active training group significantly decreased their discount rate by an average of 50% ($d = -0.54$). This study supports the notion that the executive function system is linked to discount rate, and that discount rate can be decreased by exercising working memory over a period of time.

Yi et al. (2008) observed a change in smokers' discount rates as a result of a contingency management intervention that reduced smoking. Participants exposed to the contingency management intervention were required to visit the laboratory in the morning, afternoon, and evening for five consecutive days and provide a breath sample that was analyzed for carbon monoxide content. If the carbon monoxide level was below 12 ppm, the participant was given $10 in cash. Control participants were asked to continue their regular smoking pattern and were not required to make regular visits to the laboratory. Delay-discounting assessments for hypothetical rewards of $1000 and $1000 worth of cigarettes were administered before and after the contingency-management intervention. Statistically significant decreases in discount rates for both the money ($d = -0.59$) and cigarette ($d = -0.78$) commodities were observed over the course of treatment for the contingency management group only (Table 1).

Analogous to those results (Yi et al., 2008), Landes et al. (in press) found that opioid-dependent participants completing a 12-week multimodal treatment including contingency management had lower discount rates after treatment than before. In two separate experiments, opioid-dependent participants were recruited to participate in a treatment regimen that always included voucher contingency management based on opioid abstinence and the medication burprenorphine, but also may have included either a community reinforcement approach component or standard counseling, depending on group assignment. Independent of group, participants who completed the 12-week treatment in either experiment (n = 159) were significantly more likely to have lower discount rates after treatment than before ($d = -0.41$, Table 1). On an individual basis, 39% of completers showed a significant decrease in discount rate, while 13% showed a significant increase.

Lastly, Black and Rosen (2011) assessed the effects of a money-management-based substance use treatment intervention on discount rate. Adults with histories of cocaine and/or alcohol use received outpatient psychiatric treatment and the Advisor-Teller Money Manager intervention. This intervention addresses participants' substance abuse issues in the context of discussion and instruction about general money management concerns, particularly making and maintaining budgets. Participants were randomly assigned to an active treatment group or a control group. Discount rates were assessed via the Monetary-Choice Questionnaire (Kirby et al., 1999) administered at 0, 8, 20, and 32 weeks after group assignment. Thoughtful responding on the questionnaire was encouraged by giving each participant a one in six chance of receiving a randomly-selected outcome chosen by the participant on the questionnaire. Black and Rosen found that although discount rates generally increased over the course of time in the experiment, this increase was significantly less for the intervention group than the control group ($d$ not calculable, Table 1).

Although fairly different in modality, all four of these treatments demonstrated moderately sized decreases of discount rate in the active treatment groups relative to the appropriate control groups (Bickel, Yi, et al., 2011; Black & Rosen, 2011; Landes et al., in press; Yi et al., 2008). Reducing drug use or achieving abstinence may lead to a decrease in discount rate, independent of the process by which abstinence was achieved. Since each of these experiments measured discount rates very near to the end of treatment, the observed decreases in rate may be due to current, active engagement with treatment independent of the effectiveness of that treatment. Future research should examine these possibilities. Concurrent measurement of the executive and impulsive decision-making systems with treatments such as these could also allow for determining if the CNDS model accurately identifies the role of the executive and impulsive systems in any observed changes.

## Direct Manipulation of the Executive System

Two studies have used noninvasive brain stimulation techniques to modulate activity in the dorsolateral prefrontal cortex (dlPFC). These studies may be the most direct test of the CNDS view of delay discounting, since they directly manipulated the activity of a component of the executive system. Both studies found that modulation of the dlPFC influenced discounting of delayed rewards, while control manipulations did not (Cho et al., 2010; Figner et al., 2010) supporting the CNDS view of delay discounting.

Figner et al. (2010) assessed 52 individuals' delay discounting both immediately after receiving repetitive TMS and after the TMS effects were expected to have worn off (i.e., 30 minutes). Half of the choices were between smaller immediate reinforcers and larger reinforcers delayed 2 weeks, and half were between smaller reinforcers delayed 2 weeks and larger reinforcers delayed 4 weeks. The participants were divided into three groups. One group received 15 min of 1 Hz repetitive TMS to the left dlPFC, a second group received 15 min of 1 Hz repetitive TMS to the right dlPFC, and a third group received 15 minutes of sham stimulation that was designed to mimic the feel and sound of TMS without inducing a magnetic field. Compared to sham, they found that TMS applied to the left dlPFC temporarily increased the participant's tendency to choose the smaller immediate reinforcers (*d* not calculable, Table 1), but did not impact choices when both options were delayed (i.e., 2 weeks versus 4 weeks). TMS applied to the right dlPFC had no effect on choices. Similarly, Cho and colleagues (2010) modulated cortical excitability by applying TMS (i.e., continuous theta burst stimulation [cTBS]) to participants' right dlPFC. They assessed the effect of 3-pulse bouts at 50 Hz with a bout frequency of 5 Hz on delay discounting rates 3 min after the participant received either cTBS or sham (control) stimulation. They found that discount rates obtained after cTBS was applied to the right dlPFC were significantly lower (i.e., a greater tendency to choose the larger–later reinforcer) than those obtained after sham stimulation ($d = -0.52$, Table 1).

Together, these two studies provide confirmatory evidence that the dlPFC is a neural substrate of the process of discounting delayed rewards. The direction of change produced by these two applications of TMS were in opposite directions, but without concurrent fMRI scanning after TMS application, whether a particular TMS procedure increases or decreases brain activity is difficult to determine. The different pulse frequencies employed in the two studies may have been a factor, since high frequency (> 1 Hz) stimulation tends to increase activity while low frequency stimulation tends to depress activity (Lazzaro et al., 2005). Nevertheless, these studies allow one to infer that the dlPFC is not just preferentially active during discounting tasks (McClure et al., 2004), but that altering dlPFC activity also changes discount rate.

## Executive Function Taxation

Another approach to assessing the role of the executive system in delay discounting is to tax some component of the executive system while individuals complete a delay-discounting task. If executive functioning is related to delay discounting, then an executive system that is otherwise occupied or taxed would be less able to exert control over the impulsive system, thereby increasing discount rate. Two experiments have tested this notion (Ebert, 2001; Hinson et al., 2003).

As noted in an above section, Hinson et al. (2003) conducted a series of experiments that demonstrated that an executive system taxed through its concurrent use in a working memory task does not effectively compete with the impulsive system (cf. Franco-Watkins, Rickard, & Pashler, 2010, for a partial replication). In their first experiment, 44 undergraduate psychology majors were required to complete three conditions. The working-memory condition instructed participants to remember a five-digit string of numbers while completing a delay-discounting task. Once the delay-discounting task was complete, participants were asked to recall the memorized string of numbers. In a second condition, participants were required to generate a random number between 1 and 9 after each choice made during the delay-discounting task. The third condition only required participants to type a letter that appeared on the screen after each trial of the delay-discounting task. This third condition, which did not require remembering, acted as a control condition for the other two conditions. Rates of delay discounting were higher during the condition that required remembering the string of numbers ($d$ = 0.47, Table 1).

In their second experiment, working memory load was manipulated by presenting two, three, or four stimuli from which to choose on each trial of the delay-discounting task. Discount rates were increased when either three ($d$ = 0.30, Table 1) or four ($d$ = 0.33, Table 1) alternatives were presented. The third experiment replicated the findings from Experiment 2 in a larger sample of undergraduates (170) and found that discount rate was associated with scores on the Barratt Impulsivity Scale (Patton, Stanford, & Barratt, 1995) and the Dysexecutive Questionnaire (Burgess, Alderman, Evans, Emslie, & Wilson, 1998). The fourth experiment replicated both the effect of remembering a string of digits ($d$ = 0.37, Table 1) and presenting three delay-discounting stimuli ($d$ = 0.71, Table 1) with potentially real rewards (the outcome for one choice was actually delivered to participants). In sum, these findings support the CNDS assertion that executive function, and specifically working memory, is related to delay discounting.

Ebert (2001) conducted a pair of experiments evaluating the impact of two manipulations said to tax cognitive resources while participants simultaneously assessed the value of future events. In the first experiment, half of the participants who completed a delay-discounting task had a limited time to respond (i.e., 3 s), whereas the other half were not under time pressure. Discount rates were lower in the group under time pressure, but only for the first half of the session ($d$ = −0.32, Table 1). In the second experiment, half of the subjects had to perform a tone-monitoring task as they underwent the delay-discounting assessment. Discount rates were lower in the subjects performing the tone-monitoring task ($d$ = −0.63, Table 1). These findings corroborate the notion that executive function is involved in delay discounting, but this effect is in the opposite direction as the Hinson et al. (2003) study. Numerous procedural differences could potentially account for these differences. For example, Hinson et al. used a psychophysical titration procedure that involved many choices between outcomes to assess discount rate, whereas Ebert inferred discount rate by asking participants to state the present value of future events. Perhaps estimating a value does not involve executive function in the same way as making choices between options. In any case, more research is needed before definitive statements can be made about the role of executive-function distraction procedures on discount rate.

### Framing Effects: Time Saliency or Perception

Framing effects refer to manipulations that alter the way a question is phrased or presented, or alters the immediate context of a question. Researchers have employed a variety of framing effects to modify discount rate, and a number of these studies have manipulated the saliency or context of the time that a future event will occur or the intervening time before the future event (LeBoeuf, 2006; Magan, Dweck, & Gross, 2008; Peters & Büchel, 2010; Radu, Yi, Bickel, Gross, & McClure, 2011; Read, Frederick, Orsel, & Rahman, 2005; Ungemach, Stewart, & Reimers, 2011; Zauberman, Kim, Malkoc, & Bettman, 2009). By manipulating salience of delayed outcomes, these manipulations may alter how participants think about and value future outcomes. By encouraging participants to engage in future thinking, these studies may have been altering discount rate by affecting the executive decision-making system, although whether increasing salience of an outcome, by itself, will result in increased valuation of that outcome is unknown.

The first of these studies investigated the effect of episodic future thinking on discount rates (Peters & Büchel, 2010). Discounting assessments for potentially real rewards (i.e., one trial was chosen randomly and the choice option selected by the participant on that trial was actually delivered to the participant) were administered to participants in an fMRI session. A distinguishing aspect of this experiment was its implementation of an “episodic condition.” To create the episodic condition, the experimenters determined subject-specific future events in an extensive prescanning interview with each participant. The experimenters devised episodic cue words that referred to these events. These were intended by the experimenters to evoke episodic future thinking. This episodic thinking was presumed to be spontaneous because the participants were not instructed to use mental imagery. Episodic cues were presented in half of the trials of the discounting assessments, which constituted the episodic condition. Peters and Büchel found that discount rates obtained in the episodic condition were significantly lower than those obtained in the control condition ($d = -0.83$, Table 1), supporting the hypothesis that spontaneous episodic imagery during cue processing reduces discount rates. Importantly, they found that brain activation in the executive system (lateral prefrontal cortex, posterior cingulate cortex, and ventromedial prefrontal cortex) was significantly higher during choices made in the episodic condition than in the control condition. These data support the CNDS view of discounting by confirming that a manipulation that increases activity in the executive system shows a corresponding decrease in discount rate. They also found that the effect of the episodic cues on discount rate was restricted to subjects who were assessed after scanning as high in imagery, suggesting a direct relationship between executive system activation and decrease in discount rate.

In an analogous experiment, Ungemach et al. (2011) altered discount rate by increasing the saliency of an event during the delay associated with monetary rewards. Seventy-five college applicants (most aged 17–18) were first asked how they were going to celebrate their birthday. Indifference points were then obtained between sums of money to be delivered 3 months (smaller–sooner) or 9 months (larger–later) in the future. Participants whose birthdays fell in the 3-to-9 month period between assessment and hypothetical reward delivery had higher discount rates than those with birthdays before or after that period ($d = 0.76$, Table 1). Although Peters and Büchel (2010) found that focusing attention on the time of delivery of a delayed reward can decrease discount rates, Ungemach et al. found that the opposite may be true if attention is focused on the intervening delay period. Since the executive system is likely involved in focusing attention on events in future delay periods, an interesting test of the CNDS view would be to compare executive system activation with fMRI or a similar tool while attention is focused on the time of delivery of a future reward or the intervening delay period. If both future-thinking exercises increase executive system

activation, much could be learned about the relation between discount rate and activation of prefrontal brain regions.

Another experiment that encouraged participants to alter their thinking about future events examined a framing effect called the hidden-zero effect (Magan et al., 2008). Questions in a delay-discounting assessment were worded to be clear to the participant that both of the choice options involved a sequence of events. For example, participants may be asked: "Would you prefer [A] $5 today and $0 in 26 days OR [B] $0 today and $6.20 in 26 days?" (Magan et al., p. 648). The key to this framing scheme is to make the receipt of a zero amount explicit at the time opposite that chosen for the receipt of a monetary reinforcer. They found that the explicit-zero format of question presentation resulted in significantly decreased discount rate compared to question phrasing that did not include an explicit zero opposing option for both hypothetical ($d = -0.84$, Table 1) and potentially real ($d = -0.54$, Table 1) money.

In a follow-up experiment, Radu et al. (2011) conducted a set of four experiments to distinguish between two possible mechanisms for the explicit-zero effect. They hypothesized that this effect is likely either due to a bias for sequences of reinforcers that improve over time, or to participants' attending to the future choice stimuli more closely. They conducted a series of experiments, including one that involved past discounting. Analogous to many tasks that assess the discounting of future events, past discounting assessments ask individuals their preference for having received small reinforcers now, or larger reinforcers at various points in the past (e.g., $500 now versus $1,000 a month ago). Individuals discount past reinforcers in a hyperbolic fashion similar to how they do for future reinforcers (Yi, Gatchalian, & Bickel, 2006). In two experiments, Radu et al. found that the framing questions with explicit zeros also decreases rates of past discounting ($d = -0.80$ and $d = -1.34$, Table 1) as well as future discounting ($d = -0.89$, Table 1). This argues against the hypothesis that participants are biased toward an increasing series of amounts. In a separate experiment, they also found that participants were more likely to choose temporally distant past rewards if they were first primed to attend to significant events in their past ($d = -0.40$, Table 1). Radu et al. concluded that this pattern of results supports the hypothesis that the explicit-zero effect reduces discount rate by encouraging participants to more closely attend to the temporally distant choice options. As attention is an executive function controlled by the executive system, the explicit-zero phrasing of choice options may decrease discount rate by altering activity in this neural system, although this remains untested.

Studies have also examined the effect of stating the choice alternatives in a delay-discounting assessment as either amounts that would be received on specific dates (e.g. a calendar date 2 months after the experiment date) or as the duration of time until their receipt (e.g. "2 months from now"). In a series of experiments, Read et al. (2005) determined that discount rate is lower when delayed options are represented with specific dates instead of durations of time. They replicated this result using hypothetical ($d = -0.58$ to $-1.00$, see Table 1) and potentially real rewards ($d = -1.04$, Table 1), and also when assessing discount rate with choices between smaller–sooner and larger–later rewards or by simply asking participants the present value of a reward available in the future. Additionally, future dates that are represented as compound stimuli including both dates and durations of delay yield discount rates similar to delays alone ($d = -0.67$, Table 1), suggesting that delay format contributed most to the participants' impressions of temporal distance when compounded. Lastly, Read et al. used a computerized choice titration procedure in conditions that displayed future rewards in terms of dates, and other conditions that displayed future rewards in terms of units of delay. Analysis of these data suggest that delay discounting functions are only accurately described as hyperbolic when the delay to future

receipt of reward is stated in terms of delay duration, not when stated in terms of dates. In several experiments, LeBoeuf (2006) replicated this general effect (*d* range = −0.39 to −1.61, Table 1). In these experiments, future events were either described as specific dates or durations of delay, and higher discount rates due to stating delays as durations were manifest in a variety of ways: (a) demand for more compensation to endure a delay; (b) demand that a certificate of deposit be worth more at maturity; (c) unwillingness to wait as long to receive a fixed amount of money; (d) less willingness to wait for an extra payoff; (e) greater willingness to defer payment of a fine; and (f) greater perceived length of a given time interval.

Zauberman et al. (2009) manipulated subjective time perception through a temporal priming procedure that asked the participants to estimate the duration of seven activities. Discount rates assessed after the priming procedure were compared to discount rates assessed after a control condition wherein the participants rated the caloric content of seven foods. Discount rates after the temporal priming condition were lower than those after the control condition (*d* not calculable, Table 1). Zauberman et al. also found that time perception (i.e., rating of how subjectively distant various objective time points were from the present) was broadened in the temporal priming condition.

Time perception is necessarily related to delay discounting. If two people perceive 1 year as subjectively different lengths of time, their apparent discount rates will necessarily differ, all else being equal. Thus, framing effects that alter time perception should also alter apparent discount rate. The implications of timing framing effects on the CNDS model are largely speculative. However, the areas of the brain associated with time perception are distinct from those we have defined as the executive or the impulsive systems (Wittmann et al., 2011). Therefore, timing framing effects may represent a class of effects that effectively alter discount rate, but somewhat outside the implications of the CNDS model.

### Non-timing Framing Effects

A number of studies have altered discount rates with framing effects that are not obviously related to one another. The first of these manipulated discount rate through a reward contrast procedure (Dai, Grace, & Kemp, 2009). Reward contrast occurs when the effectiveness of a reinforcing stimulus varies inversely with the surrounding reinforcement context (Williams, 1983, 2002), and may be analogous to “anchoring effects” which describe a very similar process (Tversky & Kahneman, 1974). Dai et al. showed that delay and probability discounting rates are subject to reward contrast effects. Specifically, they assessed discount rate for delayed $500 following other delay-discounting assessments for a higher ($5,000) or lower ($50) amount. Two groups first answered questions in discounting procedures that assessed the subjective values of hypothetical $50 or $5,000 delayed or probabilistic monetary amounts. Both groups then completed discounting assessments of $500 delayed or probabilistic monetary amounts. They found that the discount rates of the $500 delayed or probabilistic money differed for the two groups. In the assessment of delay discounting, the discount rate for $500 was higher following the $5,000 assessment than the $50 assessment in both a between-subject (*d* = 0.85, Table 1) and within-subject (*d* = 1.00, Table 1) design, reflecting a lesser valuation of $500 subsequent to exposure to questions about $5,000 than subsequent to exposure to questions about $50. Conversely, the probability discounting rate for $500 was higher following the $5,000 assessment than following the $50 assessment. The opposite effect of group on discount rate for delay versus probability discounting is consistent with the opposite nature of the magnitude effect in delay versus probability discounting (Green, Myerson, & Ostaszewski, 1999), suggesting this framing effect is operating by manipulating the subjective value of the $500 reward. This interpretation is

also consistent with the typical effect of reward contrast on the value of a stimulus (Williams, 1983).

Callan, Shead, & Olson (2009) divided 56 undergraduates into two groups. Both groups watched a videotaped interview with a HIV-positive woman, but one group was told she contracted HIV through unprotected sex and the other group was told she contracted HIV through a tainted blood transfusion. Delay-discounting assessments conducted after viewing the video found that the group told that the woman contracted HIV through the tainted blood transfusion discounted delayed money more rapidly than the group that had been told that she had contracted HIV though unprotected sex ($d$ = 0.68, Table 1). A questionnaire that accompanied the delay-discounting task ruled out the influence of fear and other emotions evoked by the scenario. The authors credited this effect to the participants attributing unfairness to the woman having received the tainted transfusion, and the authors relate the ability to delay gratification to the belief that one is living in a just world.

In a follow-up study, 71 undergraduate psychology students provide information (e.g., monthly income, living expenses, etc.) that the experimenters used to ascertain their discretionary income relative to their peers (Callan, Shead, & Olson, 2011). Half of the subjects were told they had less discretionary income than their peers, whereas the other half were told their discretionary income was similar to others. All subjects subsequently completed a delay-discounting assessment. The participants that were told that they had relatively low levels of discretionary income discounted delayed reinforcers more rapidly than the group told they have average discretionary income ($d$ = 0.57, Table 1). As with the earlier study (Callan et al., 2009), the findings were interpreted as being due to a manipulation of “fairness.” Despite this interpretation, the complex psychosocial scenarios of these studies make identifying the reinforcers and relevant stimuli in control of the behavior of the individuals difficult, and therefore identifying whether the results support or refute the CNDS perspective is similarly difficult.

Malkoc, Zauberman & Bettman (2010) conducted a series of brief experiments on the impact of abstract or concrete thinking on rates of delay discounting. In their first experiment, one group of undergraduates chose between two cameras that could be concretely compared (i.e., two digital cameras) whereas a second group of undergraduates chose between two cameras that could not be concretely compared (i.e., an analog and a digital camera). Individuals that could evaluate the cameras concretely required more money to delay their camera delivery than those that could not concretely compare cameras (i.e., had to evaluate “abstractly”), and therefore had increased discount rates ($d$ = 0.18, Table 1). In their second experiment, one group of undergraduates was instructed to either consider the implications of a newly passed law (i.e., the Digital Millennium Copyright Act) for one particular person that they know (concrete thinking group) or for all music consumers (abstract thinking group). Participants were then asked how much money they would pay to avoid a delay to the receipt of a $45 rebate. The concrete thinking group was willing to pay more to avoid the delay to rebate delivery, again indicating increased discount rates. Experiment 3 found a similar effect of searching for concrete (i.e., names of fruit) or abstract (i.e., adjectives about fruit) words in a word search puzzle on subsequent choices about receiving money to delay using a gift certificate. In their fourth experiment, two groups of undergraduates either evaluated two similar private retirement plans (concrete comparison) or dissimilar private and public retirement plans (abstract comparison). The abstract comparison group had higher discount rates (i.e., required more money to avoid a delay to a $200 payout), but only if delays were presented as durations of time instead of concrete dates. While speculative, abstract thinking is sometimes thought of as an executive function (e.g., Alvarez & Emory, 2006), so in the CNDS view, these experiments may have been altering executive function and thereby affecting discount rate.

## Conditioned Stimuli and Discount Rate

Three experiments presented stimuli that likely were conditioned or discriminative stimuli for behavior that could be considered impulsive in conjunction with discounting assessments, and thereby altered discount rates (Dixon & Holton, 2009; Dixon, Jacobs, & Sanders, 2006; Wilson & Daly, 2004). Conditioned stimuli associated with impulsive behavior are likely to alter discount rate by changing activity of the impulsive system, but the three experiments described below do not provide evidence for or against that hypothesis.

Dixon et al. (2006) examined the effect of assessment location on pathological gamblers' discount rates. Participants completed discounting tasks in both a coffee shop and an off-track betting facility. This within-subject research explored whether discount rates were influenced by a highly salient and conditioned context for gamblers. Dixon et al. found that delay discounting rates obtained in the off-track betting facility were greater than those obtained in the coffee shop for 16 of 20 participants ($d$ = 0.51, Table 1). This is strong support for the hypothesis that the immediate context of an assessment may influence discount rates.

Building upon those findings, Dixon and Holton (2009) used a relational training procedure to alter discount rate in pathological gamblers. Delay-discounting assessments were first collected in the presence of neutral stimuli (i.e., a pink square behind the larger later option and a purple square behind the smaller sooner option). This was followed by relational training that altered the function of those neutral stimuli. Namely, the purple stimulus was present during a series of match-to-sample trials where the correct answer was the alternative that was "worse than" the comparison stimulus (e.g., $5 is worse than $20), whereas the pink square was present during match-to-sample trials where the correct answer was "better than" the comparison stimulus (e.g., $75 is better than $20). Delay-discounting rates were then reassessed with the purple stimulus associated with the immediate option and the pink stimulus associated with the delayed option. Discount rates were lower than before relational training ($d$ not calculable, Table 1), suggesting that the function of contextual cues that alter discounting can be manipulated with contextual pairings.

Wilson and Daly (2004) assessed the effect of viewing appealing versus not appealing people or the comparable effect of viewing appealing versus unappealing cars on discount rate. For each comparison, one group viewed photographs of objects that had been predetermined to be appealing and the other group viewed photographs of objects that had been predetermined to be unappealing. Delay discounting was measured before and after the photograph viewings. Wilson and Daly found a significant pre- to post-viewing increase in the discount rates of the men who viewed photographs of appealing women ($d$ = 0.55, Table 1), but no significant changes in the discount rates of either women who viewed appealing or unappealing photos of men, nor the group of men who viewed photographs of unappealing women. Conversely, only women displayed a significant pre- to post-viewing change in discount rates in the contexts of viewing attractive or unattractive cars ($d$ = 0.90, Table 1). Although Wilson and Daly interpreted the observed changes in discount rates in terms of evolutionary theory, the photos of appealing women and photos of appealing cars may also have been conditioned stimuli associated with impulsive behavior patterns.

These studies suggest that context can significantly alter discount rate. The exact relation of these findings to the CNDS, however, is unclear. Future research will clarify the relation between the CNDS theory and these context effects.

### Physiological State Effects

Two experiments found that discount rate increased when participants were experiencing withdrawal from a drug on which they were dependent (Ashare & Hawk, 2011; Giordano et al., 2002). Drug withdrawal increases drug craving (Schuster, Greenwald, Johanson, & Heishman, 1995) and may increase discount rate by increasing the activity of the impulsive system, although this remains untested.

Ashare and Hawk (2011) studied the acute effects of abstinence from smoking among smokers with both high and low levels of ADHD symptoms. They found that discount rates in the individuals with high levels of ADHD symptomatology were significantly higher after an overnight period of smoking abstinence than after smoking as usual (*d* not calculable, Table 1). The direction of this observed effect was similar in the smokers with low levels of ADHD symptoms, but this change was not statistically significant. Similarly, Giordano et al. (2002) assessed the effects of opioid deprivation on opioid-dependent outpatients' discount rates for $1000, $3000, and $10,000 of hypothetical money and $1000, $3000, and $10,000 worth of hypothetical heroin. The six assessments were administered 2 hr after buprenorphine treatment (i.e., when participants were not in opioid withdrawal) and just prior to buprenorphine administration (i.e., when participants were experiencing mild opioid withdrawal). They found that discount rates obtained during deprivation were significantly higher than those when the participants were not deprived (*d* not calculable, Table 1).

Two studies examined the effect of sleep deprivation on discount rate, with mixed results. Reynolds and Schiffbauer (2004) assessed the effects of sleep deprivation on discount rate as assessed by the experiential discounting task. The results of this within-subject study showed that discount rates were significantly higher in the sleep-deprived state compared to the rested state ($d = 0.53$, Table 1), supporting the contention that discount rates can be modulated by sleep deprivation. However, a similar study found that sleep deprivation had no effect on discount rates obtained with the experiential discounting task or a hypothetical discounting task (Acheson, Richards, & de Wit, 2007). Given the broad range of physiological and behavioral effects of sleep deprivation, asserting whether this variable manipulates the executive system or the impulsive system may be premature. Further study is required to determine whether sleep deprivation affects discount rate, and if it does, the nature of the mechanism of this effect.

A final study that changed discount rate via physiological means manipulated participants' blood glucose levels. Wang and Dvorak (2010) assessed the discount rates for monetary rewards in two groups of participants before and after they manipulated the blood glucose level of the test group by having those participants drink a caffeine-free soft drink containing sugar. The control group drank a caffeine-free and sugar-free soft drink. Wang and Dvorak found a significant decrease in the discount rates in the experimental group ($d = -0.45$, Table 1) and a significant increase in the control group ($d = 0.54$, Table 1). This suggests a relation between discount rates and blood glucose levels, with high levels being correlated with lower discount rates.

The physiological effects reviewed above may plausibly manipulate either the impulsive system or executive system or some combination of the two. These possibilities, however, await clarification through future research.

### Acute Drug Effects

Acute drug administration sometimes impacts rates of delay discounting. Mixed effects have been reported with acute alcohol administration (Ortner, MacDonald, & Olmstead, 2003; Reynolds, Richards, & de Wit, 2006; Richards, Zhang, Mitchell, & de Wit, 1999), making

interpretation of these results difficult. The psychomotor stimulants *d*-amphetamine and methylphenidate, both of which are used clinically to treat ADHD, decrease discount rates when administered acutely (de Wit, Enggasser, & Richards, 2002; Shiels et al., 2009), providing support for the use of the delay discounting task as a marker for clinical efficacy.

Acute effects of alcohol on discount rate appear to depend on the specific delay-discounting task used. Richards et al. (1999) did not find statistically significant increases in discount rates due to alcohol intake. Ortner et al. (2003) reported that increasing blood alcohol level actually decreased discount rates ($d$ = −0.35 to −0.40, Table 1), although the main effect of alcohol administration in this study did not meet traditional cutoffs for statistical significance. Perhaps importantly, those two studies assessed discounting via a series of hypothetical binary-choice questions (although one choice out of 140 was randomly chosen and delivered to the participant after the session in Ortner et al.). Reynolds et al. (2006), however, assessed discount rates of participants via both the experiential discounting task and a hypothetical-choice question procedure. Their participants were administered placebo, 0.4 and 0.8 g/kg doses of alcohol prior to separate testing sessions. Results showed a significant increase in discount rates for the 0.8 g/kg dose compared to placebo as assessed by the experiential discounting task ($d$ = 1.01, Table 1). No tested amount of alcohol had a significant effect on the hypothetical-choice delay-discounting task. They suggest that these discrepant finding may be because the experiential discounting task requires the participant to actually experience delays during testing, whereas the question-based assessment requires the participant to predict or imagine the delayed events.

There are opposing hypotheses about the effects of amphetamine on discount rates. Methamphetamine abuse is associated with increased discount rates (Monterosso et al., 2007). However, amphetamine is also a medication used to diminish the symptoms of ADHD, with impulsivity being a primary symptom. Thus, when viewed as a medication, amphetamine could be expected to decrease discount rates. de Wit et al. (2002) examined effects of acute administrations of *d*-amphetamine and found that a 20-mg dose significantly reduced discount rates ($d$ = −0.21, Table 1). Methylphenidate had a similar effect on two measures of delay discounting in 9- to 12-year-old children with ADHD (Shiels et al., 2009). Either of two active doses (0.3 and 0.6 mg/kg) of methylphenidate significantly reduced discount rates as measured by the experiential discounting task ($d$ = −0.57, Table 1), but not as measured via a hypothetical delay discounting task. These effects suggest that acute administration of *d*-amphetamine and methylphenidate decreases discount rates as would be expected by clinical effects when treating ADHD, rather than increasing discount rates as would be expected via the data collected from stimulant abusers. This indicates that increased discount rates seen in stimulant abusers (and by extension, drug abusers) are probably not solely due to direct acute effects of the abused drugs, and may predate drug use or be due to other factors associated with chronic drug use. The effect of amphetamine and related drugs on discount rates requires further study to tease apart these complicated relationships.

Inferring whether these drugs change discount rate by selectively influencing the executive or impulsive decision systems is difficult. Acute administration of amphetamine increases activity over the whole brain, including structures included in the impulsive and executive decision-making systems (Rose et al., 2006). Conversely, alcohol decreases overall brain activity (as measured by glucose metabolism) and disrupts the functionality of connectivity networks between brain regions (Volkow et al., 2008). If Reynolds et al. (2006) are correct that the experiential discounting task is a more appropriate measure of discount rate when under the influence of a drug, the increase in discount rate they observed with alcohol could be explained by a disruption of the network connecting the executive and impulsive systems, preventing the executive system from exerting inhibitory control over the impulsive system.

This is consistent with the observation that while alcohol reduces brain activity as a whole, this reduction is less in areas associated with the impulsive system (Volkow et al.). These effects should be interpreted with caution, however, considering the wide-ranging effects of drugs such as amphetamine and alcohol and the inconsistency in the literature regarding the effects of alcohol on discount rate.

## Future Directions

According to the CNDS view, the value of immediate reinforcers can be manipulated by altering the impulsive decision system, and the value of delayed reinforcers can be manipulated by altering the executive decision system. By influencing reinforcer value, either type of manipulation can produce a change in discount rate. Specifically, increasing activity of the impulsive system should increase discount rate, whereas increasing activity of the executive system should decrease discount rate. Decreasing activity in each decision system should have the opposite effect from increasing activity. These are testable hypotheses, and we can envision a number of approaches to testing each.

There are behavioral and cognitive correlates of both the executive and impulsive decision-making systems that could be used to test whether the CNDS explanation of changes in discount rate is correct. Performance on executive function tasks such as working-memory assessments represent the functional capacity of the executive system (Mottaghy et al., 2000), and future research should develop other tasks that correspond to impulsive system activity. Obtaining measures of the executive and impulsive decision-making systems before and after an environmental manipulation to change discount rate would allow for the concomitant determination of the effect that the single manipulation has on the two decision-making systems of the CNDS theory. Research could also investigate whether such assessments, if found to be valid measures of the two decision-making systems, could be developed into diagnostic tools to predict who may be susceptible to the development of a disorder associated with imbalanced decision-making, or which treatment(s) are likely to be most effective.

Imaging approaches such as fMRI allow for direct measuring of brain states during a behavioral task, and provide information about which areas of the brain are active during the execution of a behavior. fMRI does not imply a causative influence, as the brain is highly interconnected, and an observed activation of any specific area during a task may actually be caused by control of another area (Poldrack, 2006). However, fMRI allows for inferences to be made about brain systems that mediate or are correlates of behaviors, including distinguishing between increases and decreases in activity (for a review, see Cabeza & Nyberg, 2000). Therefore, one test of the CNDS view would be to expose individuals to environmental manipulations known to alter discount rate, such as those reviewed here, while simultaneously measuring brain states with fMRI. Manipulations that expose an individual to certain conditioned stimuli (see above) should be more likely to alter activity in the impulsive system, whereas manipulations thought to improve higher-order cognitive processes such as executive function training (e.g., Bickel, Yi, et al., 2011) should be more likely to alter activity in the executive system. Although still correlational in nature, experiments such as these would provide support for hypotheses of the CNDS view regarding the brain networks associated with choices of immediate and delayed reinforcers.

Emerging technologies allow researchers to move beyond measuring correlated brain states with fMRI and directly manipulate brain states. One of these technologies is TMS. By using magnetic waves to stimulate or depress specific brain areas, TMS provides the ability to determine if activation of a specific region of the brain is simply correlated with or causes a change in behavior. For example, TMS applied to the dlPFC, an area in the executive

system, affects working memory capacity (Mottaghy et al., 2000). Importantly, this same study found that TMS applied to an area of the frontal cortex not thought to be involved in working memory (midline frontal cortex) had no effect. This study confirms that activation of the dlPFC plays a causal role in working memory, and is not simply a correlate of another, unknown variable. TMS could likewise be employed to determine if the CNDS view of changes to discount rate is correct. For example, by directly stimulating or suppressing brain areas in the executive or impulsive systems, one could simultaneously measure changes in discount rate, changes in other behavior mediated by the same systems (e.g., working memory or reinforcer value), and changes in brain activation with fMRI. A combination of measures such as these following TMS of relevant brain areas would provide a thorough test of the CNDS view of delay discounting and change in discount rate. Unfortunately, TMS is unable to manipulate the impulsive system due to deep location in the brain of this system, but future advances in technology will likely make such manipulations possible.

Another emerging technique that allows for manipulation of brain states through a different approach is real-time fMRI biofeedback. Research has demonstrated that individuals are capable of modulating activity of regions of their brains if given ongoing feedback about these activations (Yoo & Jolesz, 2002). Real-time fMRI allows researchers to treat brain activation like a lever press. Biofeedback can be delivered following targeted brain activation, resulting in an increased likelihood of future brain activity in that area. Moreover, both specific, discrete regions of the brain (e.g., Yoo & Jolesz) and the relative activations throughout a connected network of areas (LaConte, Peltier, & Hu, 2007) may be modulated. These modulations in brain activity can have real benefits. For example, real-time fMRI procedures have been used to modulate pain perception among pain patients (deCharms et al., 2005). As with TMS, real-time fMRI could be used to determine whether activation of a specific brain area or network produces the same outcome as environmental manipulations that are associated with brain activity in the same areas. Such experiments would provide evidence regarding the causal effect of brain activation.

## Conclusions

In describing radical behaviorism, Skinner (1945, 1953, 1974) distinguished between private and public events. Public events represent the majority of events and are potentially observable by any individual, whereas private events are those stimuli and actions within an individual's skin and cannot be observed by anyone other than the individual involved. Importantly, Skinner maintained that public and private events are governed by the same principles of behavior, and that they are only distinguished by the number of potential observers (many versus one). Emerging technologies are rapidly reducing the number of private events by moving cognition, emotions, and decision-making processes into the public realm. Imaging technologies such as fMRI permit more direct observation of brain activity. Additionally, real-time fMRI biofeedback technology allows researchers to apply traditional behavioral concepts and procedures, such as reinforcement, to the activity of another individual's brain regions (Yoo & Jolesz, 2002). Uncertainties still exist around the correct interpretation of any particular observed brain activation (Poldrack, 2006) but as imaging technology is improved and the understanding of brain activity is enhanced, these uncertainties are progressively coming into focus.

The CNDS view of decision-making processes posits neural mechanisms for problematic decision-making patterns in a variety of patient populations. With recent advances in technology, these neural mechanisms can be observed, manipulated, and potentially treated when dysfunctional. Here we have systematically outlined the manipulations of discount rate in humans that have been published to date. As a quantitative measure of the decision-

making processes in the CNDS view, discount rate may summarize any change effected in the underlying neural decision-making systems. For some manipulations of discount rate, the implications for the CNDS view are relatively straightforward and have thus far confirmed the hypothesis (see above). However, most of the demonstrated manipulations of discount rate cannot be as easily identified as confirming or disconfirming the CNDS theory. Going forward, we have suggested a number of procedures and experiments that could determine whether these changes in discount rate are a result of underlying changes in the executive and impulsive decision-making systems in the brain.

If delay discounting is a trans-disease process and the CNDS theory is correct about the mechanism of the decision-making processes involved in delay discounting, a number of important implications ensue. First, treatments for a disorder resulting from imbalanced decision-making processes may be made more effective by targeting these processes prior to and/or in conjunction with the other treatment. Second, treatments for one disorder may be effective for other disorders resulting from the same imbalanced decision-making processes. Instead of focusing on a specific disorder to find a treatment that works, a more fruitful approach may be to develop treatments that work across disorders and to share knowledge about the efficacy of treatments across different disorders. Finally, the CNDS theory implies that different treatments should be effective for individuals with different levels of activity in the two decision-making systems, even if these differing levels of activity lead to the same behavioral result and the same discount rate (see above for discussion and Figure 1). If true, effective treatment of disorders resulting from imbalanced decision-making processes will require personalization. To predict one's sensitivity to different treatment approaches, an assessment of discount rate and select neurocognitive assessments may be required. As fMRI may be prohibitively expensive for this purpose, future research should examine behavioral correlates of these processes to develop predictive assessment tools that could be easily applied in clinical situations. A number of remaining questions must be answered before such treatments could be developed, however. First and foremost, research must address whether experimentally induced changes in discount rate result in improvement in clinically relevant behaviors such as drug use. Second, the studies cited in this review do not speak to the duration of time that discount rates are changed with each manipulation. To be useful therapeutically, any intervention to change discount rate would either need to have persistent effects or be shown to be both effective and feasible when repeatedly administered for as long as the treatment is required.

In conclusion, the CNDS theory offers a novel way to conceptualize delay discounting and approach the treatment of disorders related to delay discounting. As a trans-disease process, changing discount rate has potential benefit to a wide range of disorders that may be affected by that process. Although much remains to be learned about the executive and impulsive decision-making systems, the current body of research suggests that the manipulation of discount rate is possible and may yield important therapeutic benefits.

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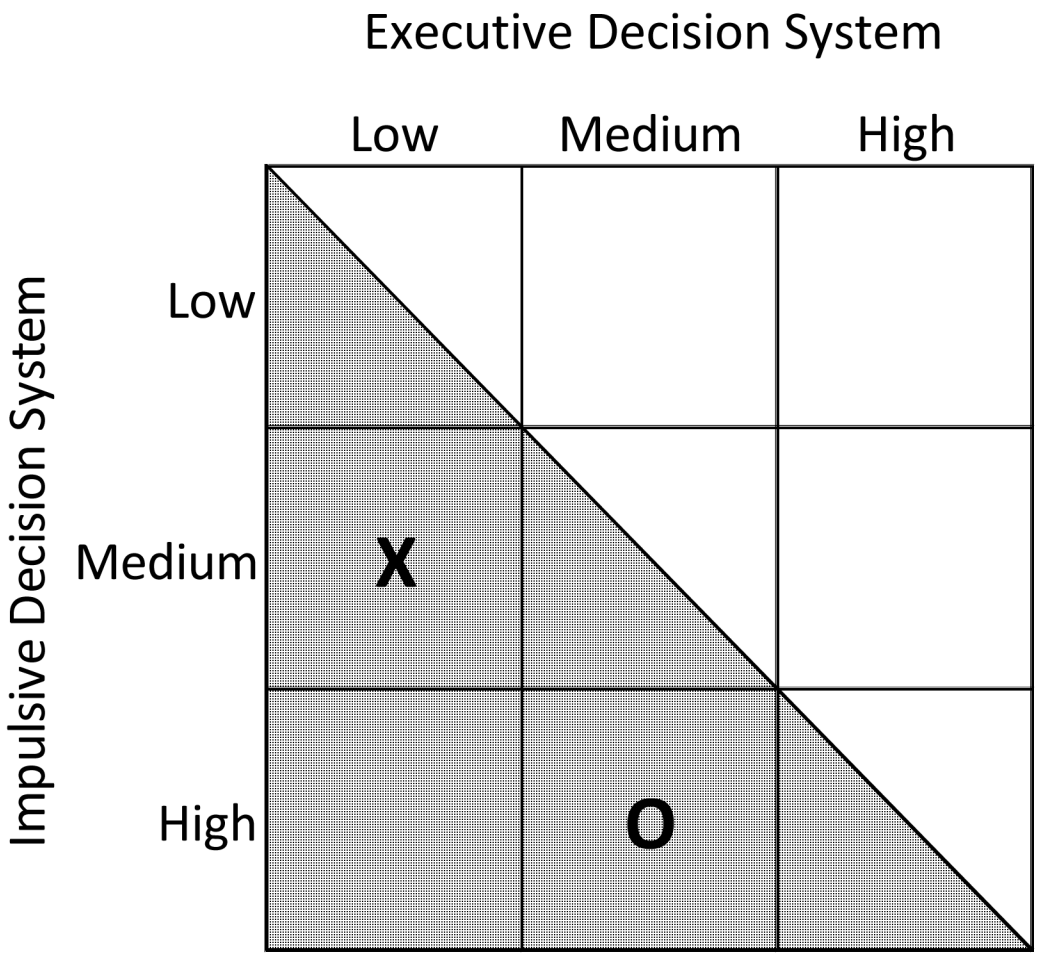


**Fig. 1.**
The hypothetical interactions between the impulsive and executive decision-making systems indicate that propensity to make choices that underlie unhealthy behavior patterns can arise from a variety of combinations of contributions of the two systems. The shaded area indicates an increased risk of developing an addiction, gambling disorder, or other disorder of self-control (adapted from Bickel, Mueller, & Jarmolowicz, in press).

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**Table 1**

Summary of each experiment reviewed here and calculated Cohen's d effect sizes (± 95% confidence interval). Positive effect sizes indicate an increase in discount rate (or analogous measure) while negative effect sizes indicate the opposite.

| Citation | Exp. | n | Population | Manipulation | Dependent variable | *d* (95% CI) |
|---|---|---|---|---|---|---|
| *Therapeutic interventions* | | | | | | |
| Bickel, Yi, et al., 2011 | | 27 | Cocaine addicts | Working memory training | Discount rate | −0.54 (NED) [a] |
| Black & Rosen, 2011 | | 90 | Alcoholics & cocaine addicts | Money management training | Discount rate | decrease (NED) |
| Landes et al., in press | | 159 | Opioid addicts | Contingency management | Discount rate | −0.41 (−0.63 to −0.19) [b] |
| Yi et al., 2008 | | 56 | Cigarette smokers | Contingency management | Discount rate (money) | −0.59 (−1.12 to −0.05) [c] |
| | | 56 | Cigarette smokers | Contingency management | Discount rate (cigarettes) | −0.78 (−1.32 to −0.24) [c] |
| *Direct manipulation of executive system* | | | | | | |
| Figner et al., 2010 | | 52 | Right-handed men | Transcranial magnetic stimulation | Immediate choices | increase (NED) |
| Cho et al., 2010 | | 7 | Right-handed people | Transcranial magnetic stimulation | Discount rate | −0.52 (−1.58 to 0.55) [b] |
| *Executive function taxation* | | | | | | |
| Hinson et al., 2003 | 1 | 44 | College students | Working memory taxation (digit string task) | Discount rate | 0.47 (0.04 to 0.89) [a] |
| | 2 | 50 | College students | Working memory taxation (3 DD options) | Discount rate | 0.30 (−0.09 to 0.70) [a] |
| | 2 | 50 | College students | Working memory taxation (4 DD options) | Discount rate | 0.33 (−0.07 to 0.72) [a] |
| | 4 | 20 | College students | Working memory taxation (digit string task) | Discount rate (real outcomes) | 0.37 (−0.26 to 0.99) [a] |
| | 4 | 20 | College students | Working memory taxation (3 DD options) | Discount rate (real outcomes) | 0.71 (0.07 to 1.34) [a] |
| Ebert, 2001 | 2 | 152 | College students | Time pressure | Ratings of value | −0.32 (NED) [d] |
| | 3 | 88 | College students | Concurrent tone monitoring task | Ratings of value | −0.63 (NED) [d] |
| *Framing effects: Time saliency or perception* | | | | | | |
| Peters & Buchel, 2010 | 1 | 30 | Healthy adults | Episodic future thought | Discount rate | −0.83 (−1.57 to −0.08) [c] |
| Ungemach et al., 2011 | 3 | 75 | College applicants | Episodic future thought between vs. not between delays | Indifference point | 0.76 (0.29 to 1.23) [c] |
| Magen et al., 2008 | | 112 | Healthy adults | Explicit-zero format (hypothetical money) | Immediate choices | −0.84 (−1.22 to −0.45) [c] |
| | | 57 | Healthy adults | Explicit-zero format (real money) | Immediate choices | −0.54 (−1.07 to −0.01) [c] |

| Citation | Exp. | n | Population | Manipulation | Dependent variable | *d* (95% CI) |
|---|---|---|---|---|---|---|
| Radu et al., 2011 | 1 | 26 | College students | Explicit-zero format (past discounting) | Smaller proximate choices | −0.80 (−1.60 to −0.00) [c] |
| | 2 | 47 | College students | Explicit-zero format (past discounting) | Smaller proximate choices | −1.34 (−1.97 to −0.71) [c] |
| | 2 | 47 | College students | Explicit-zero format (future discounting) | Smaller proximate choices | −0.89 (−1.49 to −0.29) [c] |
| | 4 | 111 | College students | Temporal priming (past discounting) | Smaller proximate choices | −0.40 (−0.78 to −0.03) [c] |
| Read et al., 2005 | 1 | 58 | College students | Explicit date (months v. dates) | Delayed choices | −0.75 (−1.36 to −0.14) [e] |
| | 1 | 58 | College students | Explicit date (weeks v. dates) | Delayed choices | −1.00 (−1.66 to −0.34) [e] |
| | 2 | 160 | College students | Explicit date | Implied choices with indifference point self-report | −0.59 (−0.85 to −0.33) [e] |
| | 3 | 58 | College students | Explicit date (potentially real money) | Delayed choices | −1.04 (−1.68 to −0.40) [e] |
| | 4 | 90 | College students | Explicit date | Delayed choices | −0.58 (−1.23 to 0.08) [e] |
| | 5 | 89 | College students | Explicit date | Discount rate | −0.67 (−1.10 to −0.24) [c] |
| LeBoeuf, 2006 | 1A | 356 | College students | Explicit date | Indifference point | decrease (NED) |
| | 1B | 229 | College students | Explicit date | Indifference point | −0.39 (−0.66 to −0.13) [c] |
| | 2 | 253 | College students | Explicit date | Specify time willing to wait | −0.43 (−0.67 to −0.18) [c] |
| | 3 | 133 | College students | Explicit date | Indifference point | −0.58 (−0.93 to −0.24) [c] |
| | 4 | 81 | College students | Explicit date | Amount paid to defer debt | −0.62 (−1.07 to −0.18) [c] |
| | 5 | 35 | College students | Explicit date | Willingness to wait for long-term investment | −1.61 (−2.37 to −0.85) [c] |
| Zauberman, 2008 | 3 | 190 | College students | Temporal priming | Amount paid for immediate delivery | decrease (NED) |
| *Non-timing framing effects* | | | | | | |
| Dai et al., 2009 | 1 | 32 | College students | Reward contrast (between subjects) | AUC | 0.85 (0.13 to 1.58) [c] |
| | 2 | 32 | College students | Reward contrast (within subject) | AUC | 1.00 (0.26 to 1.73) [c] |
| Callan et al., 2009 | | 56 | College students | Perceived unfairness in watched video | AUC | 0.68 (NED) [f] |
| Callen et al., 2011 | 1 | 71 | College students | Perceived unfairness (income disparity) | AUC | 0.57 (NED) [f] |
| Malkoc et al., 2010 | 1A | 102 | College students | Concrete thinking | Amount paid for immediate delivery | increase (NED) |
| | 1B | 521 | College students | Concrete thinking | Amount paid for immediate delivery | 0.18 (0.01 to 0.35) [c] |
| | 2 | 117 | College students | Concrete thinking | Amount paid for immediate delivery | increase (NED) |
| | 3 | 232 | College students | Concrete thinking | Amount paid for immediate delivery | increase (NED) |
| | 4 | 171 | College students | Concrete thinking | Amount paid for immediate delivery | increase (NED) |

| Citation | Exp. | n | Population | Manipulation | Dependent variable | *d* (95% CI) |
|---|---|---|---|---|---|---|
| *Conditioned stimuli and discount rate* | | | | | | |
| Dixon et al., 2006 | | 20 | Pathological gamblers | Gambling context | AUC | 0.51 (NED) [g] |
| Dixon & Holton, 2009 | | 5 | Pathological gamblers | Contextual stimulus training | AUC | decrease (NED) |
| Wilson & Daly, 2003 | | 45 | Males | Viewing attractive vs. unattractive women | Discount rate | 0.55 (−0.04 to 1.15) [c] |
| | | 25 | Females | Viewing attractive vs. unattractive cars | Discount rate | 0.90 (0.08 to 1.73) [c] |
| *Physiological state effects* | | | | | | |
| Ashare & Hawk, 2011 | | 25 | Low ADHD smokers | Cigarette deprivation | AUC | increase (NED) |
| Giordano et al., 2002 | | 13 | Opioid addicts | Opioid deprivation | Discount rate | increase (NED) |
| Reynolds & Schiffbauer, 2004 | | 11 | College students | Sleep deprivation | Discount rate | 0.53 (NED) [g] |
| Wang & Dvorak, 2010 | | 32 | College students | Drink soft drink containing sugar | Discount rate | −0.45 (NED) [f] |
| | | 33 | College students | Drink soft drink containing aspartame | Discount rate | 0.54 (NED) [f] |
| *Acute drug effects* | | | | | | |
| Ortner et al., 2003 | | 28 | Male college students | Alcohol (0.7 g/kg, standard condition) | Discount rate | −0.35 (−1.11 to 0.42) [b] |
| | | 29 | Male college students | Alcohol (0.7 g/kg, impelling cue condition) | Discount rate | −0.40 (−1.19 to 0.40) [b] |
| Reynolds et al., 2006 | | 24 | Social drinkers | Alcohol (0.8 g/kg) | Experiential discount rate | 1.01 (0.16 to 1.86) [c] |
| de Wit et al., 2002 | | 36 | Healthy adults | d-Amphetamine (20 mg) | Discount rate | −0.21 (−0.68 to 0.25) [b] |
| Shiels et al., 2009 | | 49 | Children with ADHD | Methylphenidate administration | Experiential discount rate | −0.57 (NED) [f] |

NED = Not enough data to compute effect size or 95% confidence intervals; AUC: Area under the curve of a delay discounting function; Exp. = Experiment number from the source citation.

[a] Converted from $\eta^2$ with formula in Cohen (1988).

[b] Calculated from group means and standard deviations, not taking into account any within-subject correlation for within-subject designs.

[c] Calculated from tests of statistical inference (*t* or *F*), which would lead to an increased effect size for within-subject designs.

[d] Converted from *r* with formula in Rosenthal (1994).

[e] Calculated from proportion of choices.

[f] Cohen's *d* as reported by authors.

[g] Calculated from raw data reported by authors as the mean of the differences divided by the standard deviation of the differences.