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The Behavioral- and Neuro-Economic Process of Temporal Discounting: A Candidate Behavioral Marker of Addiction

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Abstract

Addiction science would benefit from the identification of a behavioral marker. A behavioral marker could reflect the projected clinical course of the disorder, function as a surrogate measure of clinical outcome, and/or may be related to biological components that underlie the disorder. In this paper we review relevant literature, made possible with the early and sustained support by NIDA, to determine whether temporal discounting, a neurobehavioral process derived from behavioral economics and further explored through neuroeconomics, may function as a behavioral marker. Our review suggests that temporal discounting 1) identifies individuals who are drug-dependent, 2) identifies those at risk of developing drug dependence, 3) acts as a gauge of addiction severity, 4) correlates with all stages of addiction development, 5) changes with effective treatment, and 6) may be related to the biological and genetic processes that underlie addiction. Thus, initial evidence supports temporal discounting as a candidate behavioral marker. Additional studies will be required in several areas for a more conclusive determination. Confirmation that temporal discounting functions as a behavioral marker for addiction could lead to 1) a screen for new treatments, 2) personalization of prevention and treatment interventions, and 3) the extension of temporal discounting as a behavioral marker for other etiologically similar disorders.

1.1 Introduction

Modern behavioral economic and neuro-economic approaches consider addiction to function, in part, as a possible valuation disorder wherein normal decision-making mechanisms become dysfunctional, resulting in pathological reward processing (Bickel et al., 2012a; Bickel et al., 2012b). Pathological valuations stemming from this aberration distort decision-making and lead to 1) overvaluing immediate, drug-associated stimuli and 2) undervaluing longer-term rewards (Bickel et al., 2007; Schultz, 2011). Temporal discounting, considered a measure of one's location on the continuum of impulsive decision-making to self-control, may represent the interaction of these valuation systems and their associated neural networks (Bickel et al., 2007). Substantial evidence has demonstrated that addicted individuals grossly undervalue (i.e., discount) future rewards relative to immediate

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rewards (see below). Moreover, excessive discounting among those with an addiction is associated with clinically important phenomena such as poor treatment outcome and relapse (see below).

Temporal discounting, at a behavioral level, refers to the intertemporal reward preferences often characterized by a decrease in reward value as a function of the delay to its receipt (Ainslie, 1975; Rachlin and Green, 1972). Procedurally, these methods often pit a smaller, more immediate reward against a larger, more delayed reward. In some procedures, choice amounts are titrated until there is no preference between the immediate and delayed reinforcers; this value is referred to as the indifference point. Identifying the indifference points across a range of delays allows the plotting of an indifference curve. From such a curve, the rate at which a reinforcer decreases in value as a function of the delay to its receipt can be estimated. The shape of the resulting curve has been shown in a wide variety of subjects and conditions to approximate a hyperbola (Mazur, 1987) and can be characterized by the equation

$$\mathrm{V_d}=V/(1+\mathrm{kd}).$$

In this equation, V_d is the present discounted value of the reinforcer, V is the objective value of the reinforcer, k is an empirically derived constant that reflects the rate of discounting, and d is the temporal delay to the delivery of the reinforcer (Mazur, 1987; Nevin and Rachlin, 1986).

There are several discounting procedures that have been used in the literature. In addition to variants of the adjusting procedure described above, temporal discounting has been examined with tasks that arrange other sequences of questions, questionnaires, and single-item choice assessments (Bradford, 2010; Kirby et al., 1999; MacKillop, 2013). Not surprisingly, these procedures, although all measuring preferences for smaller sooner versus larger delayed rewards, have been referred to with a variety of different names including delay of gratification, delay discounting, impulsive choice, inter-temporal choice, and time preference. Moreover, quantifications of the resulting data are diverse. Although the single-free-parameter hyperbolic model described above is widely used, other models including two-free-parameter models, hyperbola-like, and exponential-power models have been used (Myerson and Green, 1995; Yi et al., 2009). Additional measures include area under the curve and proportion of the choices allocated to the smaller-sooner choice (Mitchell et al., 2005; Myerson et al., 2001). Across these procedural and analytical variations, the methods and measures with greater resolution are more responsive to addiction-related differences (MacKillop et al., 2011).

In this paper, we consider whether temporal discounting, a neurobehavioral process derived from behavioral economics, further explored with neuroeconomics, and used to understand addiction via early and sustained support by NIDA, may function as a potential or candidate behavioral marker of addiction (Bickel et al., 2007). To explore its candidacy, we will review the rapidly expanding research on temporal discounting in addiction with an emphasis on human studies, and examine the extent to which this measure may function as a behavioral marker. According to Duka et al., (2011), a behavioral marker is more than a risk factor or a mere correlate of disease progression if it also reveals facets of the disorder's mechanism, tracks treatment outcomes, and suggests novel avenues for treatment development. In order to explore the relationship of temporal discounting to addiction, as well as its status as a candidate behavioral marker, we will examine (1) if temporal discounting reflects the clinical course of addiction, (2) the relationship of temporal discounting and treatment outcomes in addiction, and (3) the biological components of

excessive temporal discounting in addiction (Duka et al., 2011; Frank and Hargreaves, 2003; Wiedemann, 2011).

Although we will review the status of temporal discounting as a potential behavioral marker in addiction, we acknowledge that the relationship between temporal discounting and addiction has not been extensively explored in all aspects of addiction research. Therefore, the determination of temporal discounting as a behavioral marker is still incomplete in several areas and awaits additional study. Moreover, we will not review the relationship between addiction and other distinct measures that have been characterized as measures of the multi-faceted construct of impulsivity, and only briefly discuss the role executive function has on discounting, as these have been reviewed elsewhere (e.g., Bickel et al., 2012a; de Wit, 2009; Perry and Carroll, 2008).

2.1 Distinguishing the Drug Dependent from Controls

To serve as a useful behavioral marker, temporal discounting should sufficiently distinguish whether individuals have a current drug dependence disorder. Research comparing temporal discounting rates among current users and community controls has repeatedly shown that those with a drug dependence disorder have a comparatively higher average discount rate. The first published accounts of greater discounting of delayed rewards was conducted among a group of opioid-dependent participants (Madden et al., 1997). The opioid-dependent group in that study was found to discount delayed money more than controls. More specifically, among controls the delayed hypothetical $1,000 lost 50% of its absolute value when the delay was approximately 37 months, while among the opioid-dependent group the same monetary amount lost half its value in only 4.5 months. Subsequent research with additional groups of opioid-dependent participants has since replicated this result (Kirby and Petry, 2004; Kirby et al., 1999; Madden et al., 1999; Odum et al., 2002; Vassileva et al., 2011). This observation extends beyond opioid-dependent participants. Populations that use nearly every common drug of abuse have been shown to discount delayed rewards more rapidly than appropriate controls. This is the case for individuals dependent on alcohol (Bjork et al., 2004; Bobova et al., 2009; Finn and Hall, 2004; Mitchell et al., 2005; Petry, 2001), (but see Kirby and Petry, 2004), cigarettes (Baker et al., 2003; Bickel et al., 1999; Bickel et al., 2008; Businelle et al., 2010; Johnson et al., 2007; Mitchell, 1999; Odum et al., 2002; Reynolds et al., 2009; Reynolds et al., 2004; Rezvanfard et al., 2010), cocaine (Bickel et al., 2011a; Camchong et al., 2011; Coffey et al., 2003; Heil et al., 2006; Kirby and Petry, 2004; Moeller et al., 2002), and methamphetamine (Monterosso et al., 2007). The only commonly abused drug dependence disorder that has been tested and shown to be unassociated with increased discount rates is marijuana dependence (Johnson et al., 2010). While most researchers have examined monetary discounting in relation to substance abuse, cigarette smokers have also been shown to discount delayed health gains at a greater rate than controls (Baker et al., 2003; Odum et al., 2002), suggesting that the delayed negative health-related consequences of smoking may be less impactful to cigarette smokers. Several studies examining discounting of several commodities have shown that the rate of discounting is often correlated across commodities (Odum, 2011a, b), but further research is needed comparing discounting of non-monetary rewards in drug-dependent and control populations.

Overall, a consistent finding observed in this section is that those with addiction discount money more than controls. This pattern of findings has generality across most drugs of abuse and is evident across different sampling techniques, settings, and measurement approaches used in these studies.

2.2 Prediction of Entrance to Drug Use

Few studies have examined whether the rate of delay discounting predicts the likelihood that an individual will use or become dependent on a drug of abuse. This is likely due to the difficulty with measuring this relationship. As one's discount rate is possibly altered by continued drug use (see above), the relationship between delay discounting and drug use onset can only be reliably and accurately measured by assessing delay discounting rate before any drug use occurs and following up at a later date to ascertain which individuals eventually used or abused a drug. To date, only one study has assessed this relationship in humans (Audrain-McGovern et al., 2009a), with another reporting on the relationship between a similar construct (delay of gratification) and later drug use (Ayduk et al., 2000). Both are described below.

The only reported study to directly measure whether a high discount rate predicts whether someone goes on to use or abuse a drug of abuse found a positive relationship between discount rate and subsequent initiation or increased use of cigarettes among high school students (Audrain-McGovern et al., 2009b). A questionnaire version of the delay discounting task, (The Monetary Choice Questionnaire, Kirby et al., 1999) and smoking behavior were assessed among 947 high school students on three occasions between grade 10 and two years post high school. A higher discount rate in grade 10 was associated with a greater use of cigarettes post high school, but was not associated with later marijuana use. A second study used a related delay of gratification task and found it predicted later cocaine use among a subgroup of individuals with high rejection sensitivity (Ayduk et al., 2000). Preschool-age children (n = 152) were offered a larger amount of a consumable treat if they waited a period of time (between 15 and 20 minutes). Children who were unable to wait could ring a bell at any time during the waiting period to receive a smaller amount of the same treat. The children were then asked in adulthood whether they used cocaine or crack. Inability to wait for the consumable treat in childhood significantly predicted cocaine/crack use in adulthood among those individuals who also scored high on a scale of rejection sensitivity, or one's tendency to experience anxiety about interpersonal rejection scenarios. Interestingly, in the cross-sectional studies cited in the section above, marijuana use in adulthood was the only substance use not related to discount rate (Johnson et al., 2010). These results may suggest that the initiation and maintenance of marijuana use occurs without the impulsive decision-making pattern associated with most other drugs of abuse.

The lack of similar studies in this area limits the confidence in this predictive relationship between delay discounting rate and later entrance to drug use across drug abusing populations. Research using animal models of delay discounting and drug self-administration benefits from the relatively greater control possible in non-human subjects research. Researchers employing these animal models have found a similar relationship between intertemporal choice patterns and intake of abused drugs. For example, rats with greater preference for immediate food rewards before any drug exposure were more likely to acquire cocaine self-administration (Anker et al., 2009; Perry et al., 2005; Perry et al., 2008), had more inelastic demand for cocaine (Koffarnus and Woods, 2013) and nicotine but not alcohol (see Diergaarde et al., 2012), and self-administered greater amounts of nicotine when the response requirement for each nicotine injection was high (Diergaarde et al., 2008). Together, these important findings demonstrate that preexisting, inherent differences in choices between immediate and delayed rewards prospectively predict which individuals will subsequently use cigarettes or cocaine/crack. In addition to the two human-subjects studies confirming this relationship, animal models of delay discounting are similarly predictive of later drug self-administration. This ability to prospectively predict who will later develop an addiction disorder is a feature of delay discounting as a candidate behavioral marker that would be of great use in the targeting of preventative treatment interventions to those individuals who are at a greater risk of later developing an addiction.

Further human research is required in this area to confirm the limited research on this prospective relationship in other drug abusing populations.

2.3 Relationship with the Amount of Drug Used

Discount rate has also been shown to be correlated with the quantity of a drug used, indicating it may be an indicator of addiction severity. Among smokers, more cigarettes smoked or a greater amount of nicotine consumed, has generally been related to the extent of temporal discounting (Johnson et al., 2007; MacKillop and Kahler, 2009; Ohmura et al., 2005; Reynolds, 2004). Similarly, the peak amount of cocaine used (Albein-Urios et al., 2012), the number of years spent abusing heroin (Cheng et al., 2012), and the average volume of alcohol consumed (MacKillop et al., 2010; Vuchinich and Simpson, 1998) were all found to positively correlate with one's rate of discounting.

2.4 Relationship with Comorbidities

Temporal discounting has been examined among individuals who demonstrate both substance-dependence and other comorbid diagnoses or symptomatology. For example, adolescent smokers who exhibit a greater number of ADHD symptoms discount more than those who do not (Fields et al., 2009). Similarly, individuals with alcohol dependence who also exhibit a Cluster B personality disorder (Dom et al., 2006) discount more than those without that comorbidity. Furthermore, attempting suicide among substance abusers (Liu et al., 2012) and need-sharing among heroin-dependent adults (Odum et al., 2000) are associated with greater rates of discounting.

Discounting has been shown in some cases to increase with the number of addictive behaviors, although not all studies have found this association. Individuals who abuse both cigarettes and alcohol have been shown to discount more than those who abuse only one of those, (Moallem and Ray, 2012) but the analogous relationship was not found between cigarette smokers and those with generalized substance abuse (Businelle et al., 2010). Further research in poly-substance abusers would help clarify these relationships.

2.5 The Effects of Acute Withdrawal on Delayed Discounting

Most drugs of abuse produce withdrawal syndromes if the amount of use is substantially and abruptly reduced after a period of heavy use. The duration of symptoms and particular symptoms present during this withdrawal syndrome varies among drug classes and also depends on dependence severity (Koob et al., 2004; West and Gossop, 1994). During withdrawal from most drugs, there is a drastically increased risk of relapse to that drug (Baker et al., 2004; Weiss, 2005). Among the few studies that measured discount rate during withdrawal, some have found that discount rate is elevated during the withdrawal period. Giordano et al. (2002) tested opioid-dependent individuals who received daily buprenorphine to alleviate opioid withdrawal symptoms on six delayed discounting procedures. In these procedures, commodity (hypothetical money and hypothetical heroin) and quantity (small, medium, or large amounts of money or bags of heroin) were varied. Participants were tested either in a deprived (where their last dose of buprenorphine was 5 days prior) or sated (after receiving 5 times their usual maintenance dose of buprenorphine) state. Across all six discounting conditions, subjects discounted more when in the deprived state, demonstrating that withdrawal symptoms may affect one's rate of discounting, and that this effect extends to multiple decisions made by the individual.

Four studies have assessed whether nicotine withdrawal effects discounting. Ashare and Hawk (2012), found that low ADHD-symptom smokers discounted future monetary rewards more when in a nicotine deprived state, whereas subjects with high ADHD-symptom showed no change in behavior due to deprivation. Fields et al. (2006) found that smokers

experiencing nicotine withdrawal discounted both hypothetical money and hypothetical cigarettes at an elevated rate when nicotine-deprived. Yi & Landes (2012) found that nicotine-deprived smokers showed greater discounting for hypothetical monetary gains at two separate magnitudes, but discounting for cigarettes was only significantly greater under the largest magnitude when the subjects had to choose between losses of hypothetical money. Finally, Mitchell et al. (2004) asked subjects to decide whether they would rather have 10 dollars now or a variable (0–60) amount of hypothetical money/cigarettes after a delay. Discounting for cigarettes was significantly greater under nicotine-withdrawal conditions compared to sated, but discounting for money was unaffected. The four studies above indicate that nicotine withdrawal may affect the rate of discounting for some commodities, but the commodity and magnitude combination at which this occurs may vary. Further research is needed to clarify these discrepancies.

The effects of withdrawal on discounting have also been studied in the animal literature where the greater level of control exerted by the experimenter in these studies allows for the more straightforward manipulation of withdrawal states. Animal researchers have investigated topics unaddressed in the human literature, such as the amount of time after drug administration required before discounting behavior returns to pre-drug levels and the effect of drug dose on discount rate.

The discounting behavior of pigeons was tested throughout and following a series of increasing, chronic morphine injections (10, 32, 100, and 200 mg/kg/day). The effect of chronic morphine was inconsistent across pigeons, but all six pigeons were more likely to choose the larger later food rewards while in opiate withdrawal. Discounting behavior returned to baseline levels one to five weeks after chronic administration ceased. This study demonstrates that drug withdrawal can affect intertemporal choice, but it is less apparent why withdrawal in this study was in the opposite direction as was observed in human subjects in opiate withdrawal (Eppolito et al., 2013) .

In Dallery & Locey, (2005) rats received chronic injections of nicotine at a dose that was personalized based on acute effects on nicotine for each subject (0.03, 0.1, 0.3, or 1.0 mg/kg/day). When nicotine withdrawal was induced by discontinuing nicotine administration, rats showed significantly greater smaller-sooner choice compared to their baseline performance for 14 consecutive sessions, largely corresponding to the human studies of nicotine withdrawal cited above.

Finally, the effect withdrawal from amphetamine had on discounting behavior was examined in rats (Gipson and Bardo, 2009). Rats allowed extended access (6 hour/day) to either 0.03 or 0.1 mg/kg/injection amphetamine increased their choices of immediate food rewards. Following this 21-day extended access period, discounting behavior gradually returned to baseline levels after approximately 3 days with no apparent exacerbation of immediate choice during acute withdrawal. Further research is needed to examine how stimulant withdrawal affects discount rate in human participants.

The above human and animal studies highlight that the effect of withdrawal from chronic drug use on one’s rate of discounting is largely dependent upon the experimental conditions and specific drug of abuse. Further research will be required to understand the mechanisms that bring about these changes in discounting rate and better relate results from the human and animal literature.

2.6 Distinguishing Current from Former Drug Dependence

The ability of discount rate to distinguish those currently dependent on a drug from those who used to be dependent, but have since quit, is less clear. Ex-smokers have a similar

discount rate as people who have never smoked, with both having lower discount rates than current smokers (Bickel et al., 1999). Similarly, injection heroin and amphetamine users have higher discount rates than ex-users and control subjects (Bretteville-Jensen, 1999). However, cocaine users that have quit only 14 (Kirby and Petry, 2004) or 30 days prior to assessment (Heil et al., 2006) and alcohol users who quit only 14 days prior to assessment (Kirby and Petry, 2004) have similar discount rates as current users, while alcohol users who quit 30 days prior to assessment have lower discount rates than current drinkers (Petry, 2001). Perhaps, discount rate requires some period of time to return to pre-drug use levels, or there may be sampling issues associated with assessing discount rate in a cross-section of currently dependent and ex-dependent individuals. Moreover, another possibility is that those users who successfully initiate and maintain abstinence had lower discount rates throughout their time in dependence, and that discount rates do not decrease after an abstinence episode. Unfortunately, longitudinal studies of discounting in addiction recovery that would conclusively determine whether discount rates decrease during recovery or are a precondition of a successful recovery have not been conducted. Discounting would serve as a useful behavioral marker if either condition is true, but it is important for future research to make this distinction to determine whether discounting is more useful to track the progress of recovery or predict which individuals will be more likely to have a successful recovery attempt.

2.7 Temporal Discounting as a Predictor of Abstinence and Cessation in Laboratory and Clinical Studies

Perhaps most important to the evidence for delay discounting serving as a behavioral marker, discount rate has been shown to correlate with recovery success in a limited set of studies. Since 2007, 11 studies using a variety of approaches and measures have examined the extent to which temporal discounting is a predictor of later abstinence or future drug use among individuals who were engaged in drug use, at least initially. Below we review each of these studies briefly.

Two studies were conducted in the human laboratory. The first examined the relation between temporal discounting and smoking. Specifically, thirty smokers without intention to quit participated in a laboratory model of abstinence reinforcement following random assignment to receive nicotine or placebo patches (Dallery and Raiff, 2007). After 3 hours of smoking deprivation, discount rate for a hypothetical $100 was assessed. Both groups then participated in three delay of gratification sessions. In two of those sessions, participants earned an increasing portion of either $5 or $20 for each 30-second period of abstinence. The third type of session was a control condition where money was provided independent of abstinence. No statistical difference was observed between the nicotine and placebo patch conditions, but those individuals who smoked during the two incentive conditions showed a higher rate of temporal discounting. In the second study, 19 nicotine-deprived cigarette smokers completed a titrating discounting procedure for $10 and $1,000 and a comparable amount of cigarettes. These subjects received monetary rewards for each minute they choose not to resume smoking in a 2-hour session (Mueller et al., 2009). Smokers participated in 4 types of sessions. In three of those sessions, the amount earned increased, decreased, or remained constant throughout the session. In the fourth type of session, there were no contingencies on smoking and smoking by the participants was shown to begin almost immediately after the session began. Time to re-initiation of smoking in the other three sessions was shortest in the decreasing condition and longest in the increasing conditions. Temporal discounting for $10, $1,000 and $1,000 worth of cigarettes, but not for $10 worth of cigarettes, was significantly correlated to the time to re-initiate smoking (Mueller et al., 2009).

Two other studies were conducted with either smoking or substance abusing adolescents. In the first of these studies, 30 adolescent smokers participated in a 4-week school-based cessation program composed of contingency management and cognitive behavior therapy (Krishnan-Sarin et al., 2007). Prior to the date set for the quit attempt, participants completed both a delay discounting questionnaire and an experiential discounting task where participants choose between an adjusting immediate amount and a later standard amount of $0.30 where options and the corresponding delays selected were actually experienced. Participants who were unable to achieve abstinence discounted more on the experiential discounting task than those who achieved abstinence (Krishnan-Sarin et al., 2007), with this relationship yielding a medium effect according to Cohen (1988). The discounting questionnaire did not yield a significant group difference. The second study by Stanger et al. (2012) randomized 165 adolescents to one of three 14-week behavioral treatment programs (either cognitive behavioral therapy alone, cognitive behavioral therapy and contingency management, or cognitive behavioral therapy with contingency management and Family Management Curriculum) designed to treat marijuana abuse or dependence. Discount rate was assessed at intake with four adjusting hypothetical discounting tasks ($100, $1,000, and marijuana equivalents of those two monetary amounts). When controlling for treatment group in multivariate models, delay discounting of $100 and $1,000 both significantly predicted the number of negative drug urine samples; that is, as the k parameter increased, marijuana abstinence decreased. Discounting of $1,000 and discounting of $1,000 worth of marijuana also predicted achieving at least 4 weeks of continuous abstinence, and discounting of $1,000 also predicted achieving at least 8 weeks of abstinence. While statistically significant, none of the above findings exceed the criteria for a small effect according to Cohen (1988). Discounting of $100 of marijuana was not predictive of treatment outcomes.

Five studies sought to decrease the cigarette smoking of adults who smoked or followed those who had quit or sought to quit. The first study by Yoon et al. (2007) identified a group of 48 pregnant smokers who had participated in two treatment groups (contingency management and control). The data from these two groups was pooled and analyzed by the subject's abstinence status. Baseline discounting of a hypothetical $1,000 was obtained in an adjusting procedure. These pregnant mothers were assessed throughout their pregnancy and several months post-partum. Overall, baseline discounting was predictive of smoking status 24 weeks post-partum and this difference exceeded the criteria for a medium effect (Cohen, 1988). The second study measured discounting in a sample of 97 highly-dependent cigarette-smoking adults prior to receiving treatment, which consisted of weekly group cognitive behavioral therapy meetings (Sheffer et al., 2012). Temporal discounting measures from an adjusting procedure were obtained at baseline for hypothetical $100 and $1,000 and a potentially real outcome of $100 (one of the choices made was randomly selected and delivered to the participant). The *k* values from the baseline hypothetical $100 and $1,000 discounting tasks, and the mean of the three discounting procedures was significantly related to abstinence. More specifically, for every standard deviation increase in average value of temporal discounting, abstinence decreased by approximately 40%. Using methods described in Chinn (2000), the effect sizes for each of these three statistical tests were found to exceed the criteria for a small effect (Cohen, 1988). The real $100 condition approached, but did not reach statistical significance. The third study examined the relationship between smoking cessation among 57 alcohol-dependent smokers receiving two treatments (standard treatment consisting of group counseling and nicotine replacement and the standard treatment plus a brief alcohol intervention). Baseline discounting was obtained through the questionnaire form of discounting for three amounts of hypothetical money ($25-$35, $50-$60, and $75-$85). Overall, temporal discounting at all three magnitudes significantly predicted days to first lapse. The hazard ratio (95% confidence intervals) from these associations at the small, medium, and large magnitudes were 1.41 (1.06–1.89), 1.53 (1.16–

2.02), and 1.43 (1.09–1.88), respectively. Therefore, an increase of one standard score on delay discounting was associated with an increased risk of having a lapse by 40–50% (MacKillop and Kahler, 2009). The fourth study measured self-reported time-period for financial planning among 1,248 older smokers who were interviewed as part of English Longitudinal Survey of Aging who were followed over a 4-year period (Adams, 2009). Preference for planning over longer timeframes was associated with a greater likelihood of quitting smoking. With a "small" effect size (see Chinn, 2000; Cohen, 1988) this statistical approach controlled for age, gender, and education. The fifth study systematically replicated the preceding study and examined 1,817 respondents of the Household Income and Labour Dynamics of Australia survey panel who were aged 15–64 years, who responded to at least four waves of data collection between 2001 and 2008 (Brown and Adams, 2013). After controlling for socio-demographic and smoking-related covariates, they found that those respondents who reported preference for longer time periods for financial planning were more likely to have quit in subsequent time periods. The hazard ratio and 95% confidence interval for this statistic was 1.29 (1.02–1.62).

Finally, two studies reported results with other forms of drug dependence. One study examined 37 opioid-dependent individuals entering a community treatment program that included opioid replacement medication who completed a discounting questionnaire and a follow-up session 3 months later (Passetti et al., 2008). Abstinence or drug use was measured by self-report of illicit drug use in the previous 30 days and one or more confirmatory urine drug screen results. Discounting as measured by the questionnaire version of discounting (with one of the choices enforced) did not distinguish the 10 abstinent from the 27 non-abstinent participants. Lastly, Washio et al. (2011) reported the results from 36 cocaine-dependent individuals, split into low and high magnitude abstinence reinforcement groups. Temporal discounting was measured at baseline with an adjusting procedure for a hypothetical $1,000, with higher discounting rate predicting greater cocaine use in the lower magnitude contingency management procedure only. This difference met the criteria for a large effect according to Cohen (1988).

The studies reviewed here are a diverse set using a variety of procedures and measures. Despite those differences, the preponderance of studies found that these measures of temporal discounting are systematically related to measures of abstinence, cessation, or relapse. Discerning if any particular measure, treatment, or drug-dependent population is more or less likely to exhibit this relationship will require additional studies and systematic replications. Moreover, any conclusions drawn from these studies would have to be tempered by the possibility of unpublished studies not demonstrating this relationship.

2.8 Modified By Effective Treatment

In this section, we review five published studies that document change in the temporal discounting of substance abusers in response to interventions. Producing a change in the rate of discounting is especially noteworthy given that measures of temporal discounting have been shown to be stable over varying timeframes ranging from 1 week to 1 year, suggesting that in the absence of an intervention discounting is a stable measure (Baker et al., 2003; Beck and Triplett, 2009; Black and Rosen, 2011; Kirby, 2009; Ohmura et al., 2006; Simpson and Vuchinich, 2000; Takahashi et al., 2007).

Temporal discounting was measured before and after two clinical trials of a multimodal treatment for opioid dependence and reported in a combined analysis across treatment modality (e.g., various combinations of efficacious treatments including buprenorphine, cognitive behavioral therapy, and contingency management) (Landes et al., 2012). One-hundred fifty-nine (159) participants completed discounting assessments at baseline and the end of treatment across the two trials (Chopra et al., 2009; Christensen et al., unpublished

results). Mean discounting at 12 weeks significantly decreased to less than half (44.8%, 95% CI from 27.5% to 73.2%) of the baseline level. Additionally, over 3 times as many participants significantly decreased their discounting from their own baseline levels as significantly increased. Note that discounting was not predictive of treatment outcome in this report.

The third study examined the effects of a money-management intervention or control intervention on temporal discounting and cocaine use in 90 participants receiving outpatient psychiatric treatment (Black and Rosen, 2011). In this random assignment study, the active treatment consisted of a multi-component package addressing substance abuse in the context of prospective money management concerns. Specifically, participants were encouraged to make monthly budgets reflecting long-term goals broken into shorter spending plans. The control group was given a workbook on creating budgets and encouraged to meet with a counselor weekly to review progress in completing budgets. Experimenters used the temporal discounting questionnaire, which they administered four times across the following 32 weeks. The group receiving the money management intervention discounted future monetary rewards less and had more abstinence than the control participants by the end of the measurement period. Additionally, independent of group assignment, those participants who discounted future rewards more over time also increased their cocaine use.

In the fourth study, the effects of 5 days of a contingency management procedure on the temporal discounting of dependent smokers were investigated (Yi et al., 2008). Cigarette smokers were randomly assigned to either a contingency management or a control condition, and temporal discounting rates for money and cigarettes were assessed before or after implementation of the conditions. Completion of contingency management reduced cigarette smoking and produced decreases in breath carbon dioxide. As shown in Figure 1, discounting decreased significantly for both cigarettes and money among the contingency management group, whereas the control group showed no significant change for either commodity.

In the fifth study, the effects of working memory training on the temporal discounting of stimulant-dependent individuals was investigated (Bickel et al., 2011b). This study was based, in part, upon prior observations suggestions that temporal discounting and working memory were correlated (Shamosh et al., 2008). Specifically, stimulant users in treatment at a local treatment facility were assigned to either receive working memory training or control training for up to 15 1-hour sessions. The working memory training consisted of sequence recall of digits, recall of reverse digits, recall of words, and a verbal memory categorization task. The control training used a modified version of this working memory program where the correct answers were cued to the participants. In doing so, the participants in the control group were exposed to the same stimuli as the experimental group and also made similar responses as the experimental group. However, since the correct response was cued, the participants did not have to “work” to obtain the correct answers. Note that this intervention, unlike the others mentioned above, was specifically designed to target discounting and not drug-taking behavior. The training groups showed improvements in working memory throughout the training sessions and exhibited significant reductions in the temporal discounting relative to their own baseline performance and to the control group. Whether this working memory intervention would decrease substance use by modifying one’s discount rate remains to be determined.

Although the reports in this section suggest that discounting changes in response to effective treatment, the number of relevant studies is limited and more will be needed to confirm whether discounting can function as a proxy of outcomes of drug treatments. One important issue to address is the relationship between the results reviewed in this and the preceding

section; that is, the relationship between temporal discounting as a predictor versus as a surrogate of treatment effect. Perhaps, temporal discounting is predictive for treatments with more modest efficacy because those treatments only work in those who discount less (patients with higher discounting having poorer outcomes), while in response to highly-effective treatment, the rate of discounting changes more extensively across the treated group, thereby limiting the predictive ability of pre-treatment discounting. If confirmed, this speculation would support the findings from Washio et al. (2011) that discounting predicts treatment outcomes with a low-magnitude reinforcement procedure, but not with a high-magnitude procedure. Importantly, confirmation of these potential relationships might also suggest that discounting can distinguish between lower and higher efficacy treatments based on whether temporal discounting is predictive prior to, or changes following, the intervention. Testing this hypothesis will require additional studies measuring discounting pre and post intervention.

2.9 The Relationship Between Delayed Discounting and Biological Components of Addiction

The research reviewed thus far suggests that temporal discounting may serve as a behavioral marker of addiction. Given this, there is value in considering whether biological components underlie the empirical relationship between discounting and addiction, as one of the criteria for a behavioral marker is its ability to elucidate a disorder's mechanism of action. If accepted as a behavioral marker of addiction, extension of the biological underpinnings of temporal discounting to the understanding of addiction may suggest novel neurobehavioral indices of the disorder. These mechanisms may be useful as diagnostic assessment tools, predictors of illness or outcome, or they may even guide the implementation of individualized therapy. Here we briefly review neural and genetic correlates of discounting in addicted populations.

To the best of our knowledge, two studies have looked at neural correlates of delayed discounting in stimulant-dependent individuals. In the first study, Monterosso et al. (2007) compared current methamphetamine-dependent individuals to non-substance users. When comparing "easy" choices, where the choices were far from the indifference curve, versus "hard" choices, where the choices were close to the indifference curve, methamphetamine users had less differential activation in frontoparietal regions. Specifically, control participants showed greater cortical activation in the left dorsal lateral prefrontal cortex (dlPFC) and right intraparietal sulcus (IPS) during hard trials than in easy trials, while the methamphetamine-dependent participants had increased activation in these frontoparietal regions during both hard and easy trials. Hoffman et al. (2008) also looked at methamphetamine users, comparing recently abstinent (i.e., abstinent for at least 2 weeks but not more than 8 weeks) individuals to age and gender matched controls. All participants completed a delayed discounting task and a magnitude estimation task as a control in the MRI scanner. These groups differed on two reported findings. First, control participants had more robust cortical activation overall compared to the methamphetamine-dependent group. For example, the controls showed evidence of greater activation in the right dlPFC and anterior cingulate cortex (ACC), both of which are associated with cognitive control and behavioral inhibition (Kerns et al., 2004; Miller and Cohen, 2001; Stuss and Knight, 2013), when contrasting hard choices in the delayed discounting task to the control task. Second, the methamphetatmine-dependent group had greater activation during easy choices than the control group, a finding which mirrors the Monterosso paper (2007). Together, these two studies may indicate that substance-using individuals have inefficient recruitment of cognitive systems compared to normal controls.

In addition to stimulant dependence, neural correlates of temporal discounting have also been examined in alcohol-dependent individuals. In a study looking at 151 participants with

varying severities of alcohol use disorder, Claus et al. (2011) compared immediate versus delayed choices and the corresponding neural activation. Alcohol use disorder severity was positively correlated with greater recruitment of frontoparietal regions, including the ACC, when participants chose the delayed choice over the immediate option. Claus et al. suggest that these findings represent impaired conflict resolution abilities in individuals with severe alcohol use disorder that may, in part, explain their tendency toward impulsive decisions. Similar results were found in a study comparing individuals that met DSM-IV criteria for alcohol use disorder to those who drink but did not meet criteria for diagnosis (Amlung et al., 2013) and in a study comparing sober, self-reported alcoholics to individuals who do not drink (Boettiger et al., 2007). Amlung et al. (2013) found differential activation as a function of diagnosis status, specifically, the group meeting diagnosis criteria exhibited hyperactivity in dlPFC and posterior parietal cortex (PPC), which are associated with cognitive control (Miller and Cohen, 2001; Stuss and Knight, 2013) and prospective thought, respectively. These results suggest that individuals with alcohol use disorders recruit greater neuronal resources to make the same decision as individuals without alcohol use disorders. In line with Amlung et al.'s (2013) findings, Boettiger et al. (2007) reported increased activation in the PPC and dorsal PFC. These findings are consistent with the interpretation of Monterosso et al. (2007) and Hoffman et al. (2008) of an inefficient cognitive system in the substance-dependent groups.

In addition to neuroimaging evidence of neural associations between addiction and discounting, studies of genetic links to discounting in addicted populations highlight an avenue of promising future research. Initial studies looking at heritability of delayed discounting in monozygotic twins (Anokhin et al., 2011) offer a basis for research on healthy controls to inform studies of substance abuse. Currently, only one study specifically looked at the genetic underpinnings of discounting in addicted populations. In this neurogenetics study (discussed previously), sober, self-reported alcoholics, homozygous for the Valine-allele on the *COMTval*^{158}*met* single nucleotide polymorphism of the catechol-O-methyltransferase (COMT) gene (which plays a role in basal frontal dopamine regulation) showed greater dorsal cognitive system activation when completing a delayed discounting task even after controlling for alcohol-abuse history. Furthermore, these participants discounted delayed rewards more steeply than participants with either of the other two genotypes (i.e., $158^{Val/Met}$ and $158^{Met/Met}$) (Boettiger et al., 2007). While this represents a compelling finding linking discounting behavior to genetic precursors, it is far from conclusive. Future research will afford the opportunity to analyze joint effects of multiple polymorphisms, which may offer greater understanding of functional genomics than that captured at the level of single markers.

Taken together, research in neurobiological and, even more so, genetic underpinnings of discounting and addiction are sparse, but warrant further study. With respect to neuroimaging, future research is necessary to specify brain regions and networks that are differentially active in delayed discounting in those with and without drug dependence to greater characterize the relationship between temporal discounting and addiction.

3.1 Conclusion

The purpose of this review was to examine if the empirical evidence supports temporal discounting as a candidate behavioral marker of addiction. Overall, the evidence suggests that temporal discounting should be considered as a candidate behavioral marker for addiction. By stipulating "candidate," we suggest this topic receives active consideration by the field. As we noted earlier, not enough data are available to fully evaluate its status as a behavioral marker in some areas.

In terms of reflecting the clinical course of addiction, temporal discounting (in a very limited number of studies) predicts entry into drug use, identifies individuals who are drug-dependent from controls, reflects the quantity of drugs used, is related to comorbidity with other disorders (but not necessarily other co-morbid dependence disorders), and may distinguish current from former drug dependence. Unfortunately, the relative absence of longitudinal study of discounting limits the conclusions that can be drawn and suggests the importance of future research of this type.

The published data suggest that temporal discounting may serve as a surrogate of treatment outcomes. Several studies found that temporal discounting measured prior to or during treatment predicted treatment outcomes. Additionally, a smaller set of studies showed that discounting changes in response to efficacious treatments. The small number of studies underscores the importance of additional studies to examine the extent and power of temporal discounting functioning as proxy for treatment outcomes.

Lastly, in terms of a biological component of temporal discounting, research supports differential neural activation during temporal discounting in substance users when compared to normal controls. Furthermore, genetic traits associated with discounting behavior in substance using populations is a promising new area of investigation. Both areas are in need of additional research in order to fully understand the biological substrates of temporal discounting and addiction.

Although the data are far from complete, the extant data suggest that temporal discounting warrants further study as a candidate behavioral marker for addiction. If additional studies were found to be supportive, then temporal discounting could be a highly useful tool that could be used as a means to examine the efficacy of novel treatments and could be the basis for personalizing prevention or treatments. In addition, if temporal discounting functions as a trans-disease process as we have speculated (Bickel et al., 2012b), then temporal discounting may serve a potential behavioral marker for other related disorders that are characterized by excessive discounting. Whether subsequent study will be supportive and whether temporal discounting is deemed a behavioral marker, of course, awaits the future efforts of the field.

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Highlights

- A behavioral marker may inform the development, severity, and cause of a disorder
- Temporal discounting is a potentially useful behavioral marker of addiction
- Temporal discounting reflects changes across five stages of addiction development
- Temporal discounting correlates with addiction severity and predicts treatment outcome
- Temporal discounting and addiction may share some genetic and neurological components

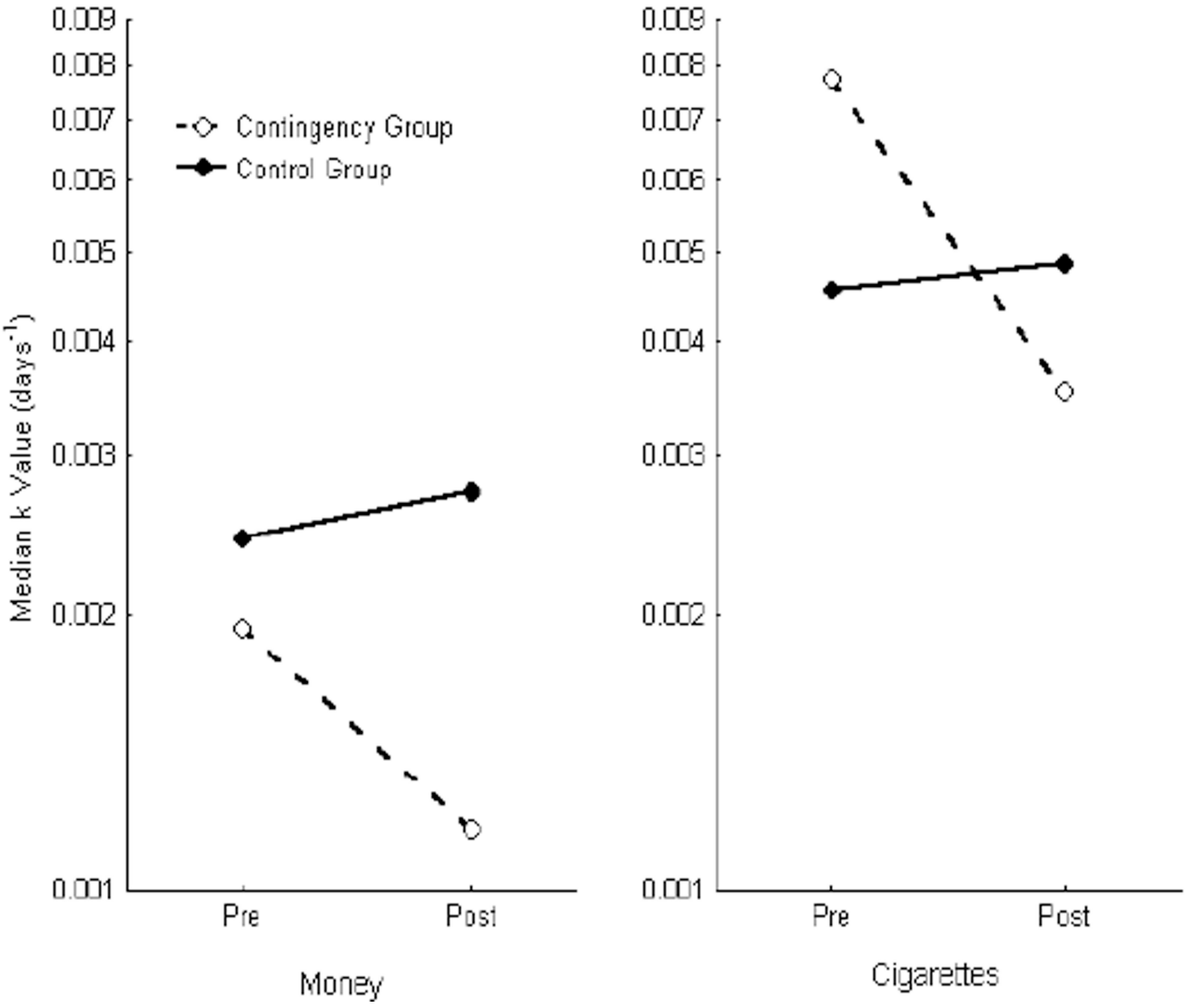

Figure 1.
Temporal discounting of money and cigarettes as measured by the discount rate (k) collected pre-post a contingency management procedure reinforcing smoking abstinence and a control condition (data from Yi et al., 2008).