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Delayed reward discounting predicts treatment response for heavy drinkers receiving smoking cessation treatment

James MacKillop^{a,b,*} and Christopher W. Kahler^b

^aDepartment of Psychology, University of Georgia, Athens, GA, U.S.A.

^bCenter for Alcohol and Addiction Studies, Brown University, Providence, RI, U.S.A.

Abstract

Delayed reward discounting (DRD) is a behavioral economic index of impulsivity that reflects the extent to which an individual devalues a reward based on its delay in time (i.e., preference for smaller immediate rewards relative to larger delayed rewards). Current smokers exhibit greater DRD compared to non-smokers, but also exhibit greater DRD compared to ex-smokers, suggesting that either DRD is inversely associated with successful smoking cessation or that smoking cessation itself reduces DRD. In a sample of treatment-seeking smokers ($n = 57$, 61% male, 85% Caucasian) participating in a randomized controlled smoking cessation trial, the current study prospectively examined DRD for money in general and at three magnitudes in relation to time to the participants' first lapse to smoking. Survival analysis using Cox proportional-hazards regression revealed that DRD predicted days to first lapse ($ps < .05-.01$) and did so beyond nicotine dependence, sensation-seeking, and income in covariate analyses, with the exception of small magnitude discounting. In addition, dichotomous comparisons revealed significantly more impulsive baseline discounting for individuals who had lapsed by the two week and eight week follow-up visits. These findings indicate that high levels of DRD reflect a risk factor for poor smoking cessation treatment response. Interrelationships among the variables assessed and clinical strategies to improve outcomes for smokers who are high in DRD are discussed.

Keywords

Nicotine Dependence; Treatment; Behavioral Economics; Delay Discounting; Impulsivity; Smoking Cessation

1. Introduction

The field of behavioral economics integrates the principles of psychology and microeconomics to study how individuals make transactions with the world (Camerer, 1999). A primary focus of the discipline is characterizing the rational and irrational processes that underlie behavior in general (Loewenstein, 1996; Kahneman, 2003), however, behavioral economics has also been profitably applied to understanding pathological behavior, particularly in terms of

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*Communicating Author: James MacKillop, PhD, Department of Psychology, University of Georgia, Athens, GA 30677, Telephone: (706) 542-3033, Fax: (706) 542-3275, Email: jmackill@uga.edu

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nicotine dependence and other substance use disorders (Vuchinich and Heather, 2003). The approach has made significant contributions by characterizing how environmental factors, such as increases in cost or the presence of alternative reinforcers, can affect substance use (Bigelow et al., 1972; Griffiths et al., 1996; Higgins et al., 1994; Johnson et al., 2004). Behavioral economics also recognizes the importance of individual differences as contributors to substance use. As such, substance use disorders are putatively a function of both individual characteristics (e.g., decision making biases, substance use history, conditioning history), environmental factors (e.g., prices, consequences, alternative reinforcers), and the interaction of the two (Vuchinich & Heather, 2003).

Delayed reward discounting (DRD), or the amount an individual devalues a reward based on its delay in time, is the individual-level behavioral economic variable that has been most extensively studied and is considered to be an index of impulsivity (Ainslie, 1975). Delayed reward discounting is typically measured by assessing an individual's preference for smaller immediate rewards relative to larger delayed rewards using choice tasks (e.g., Rachlin et al., 1991). In the context of nicotine dependence, smokers have consistently demonstrated significantly steeper devaluation of delayed rewards than non-smokers (Baker et al., 2003; Bickel et al., 1999; Mitchell, 1999; Reynolds, 2006; Reynolds et al., 2004). Greater DRD relative to matched controls has also been evident for individuals with alcohol problems (Bjork et al., 2004; Boettiger et al., 2007; Dom et al., 2006; Field et al., 2007; Mitchell et al., 2005; Mitchell et al., 2007; Petry, 2001a; Vuchinich and Simpson, 1998), stimulant dependent individuals (Coffey et al., 2003), opiate dependent individuals (Kirby et al., 1999; Madden et al., 1997), and pathological gamblers (Petry, 2001b). Based on these consistent findings across studies and different types of addictive behavior, Bickel and Johnson (2003) have argued that excessive DRD is a fundamental process in addiction. Clinically, this steep decrease in value for delayed rewards putatively underlies the persistent motivation for the small immediate drug reward and also results in the preference reversals that reflect the characteristic loss of control of addiction (Ainslie, 2001; Bickel and Marsch, 2001). For example, with regard to nicotine dependence, an individual may report a preference for the larger delayed rewards associated with quitting smoking (e.g., improved health, financial benefits), but shift their preference to the smaller but immediate rewards of smoking when a cigarette is immediately available and those larger benefits remain temporally distant.

Delayed reward discounting also appears relevant to treatment. Bickel et al. (1999) examined DRD in current smokers, ex-smokers, and never smokers, and found significantly more impulsive discounting in current smokers compared to both ex-smokers and never smokers, who exhibited almost identical discounting. Similarly, Petry (2001a) examined actively drinking alcoholics, ex-alcoholics, and individuals with no history of alcoholism and found that alcoholics exhibited greater DRD than controls, whereas ex-alcoholics exhibited intermediate levels of discounting. These findings raise an important question, do individuals with lower (i.e., less impulsive) discounting rates have a better prognosis or do individuals who successfully stop using tobacco or alcohol become less impulsive over time? In other words, are the observed differences in discounting between ongoing and former users a cause or consequence of abstinence?

These questions have been provisionally addressed via evidence that DRD is highly reliable over time. Both Simpson and Vuchinich (2000) and Baker et al. (2003) found evidence that DRD is reliable over a one-week period. Furthermore, Takahashi et al. (2007) and Ohmura et al. (2006) found DRD to be reliable over durations of several months. These studies indicate that DRD is largely reliable and suggest that it is more likely that high levels of DRD are associated with poorer outcomes, rather than DRD changing over the course of abstinence. However, the preceding studies only obliquely address the relationship between DRD and clinical prognosis. Several recent studies have directly addressed the relationship between

DRD and treatment response. In a four-week study of adolescent smokers undergoing smoking cessation treatment, lower baseline levels of DRD were associated with treatment success (Krishnan-Sarin et al., 2007). Similarly, in a sample of pregnant smokers undertaking smoking cessation treatment, DRD was found to predict treatment outcome six-months postpartum (Yoon et al., 2007). In addition to these clinical studies, a recent study using a laboratory model of abstinence reinforcement found that greater DRD was associated with choosing to smoke despite contingent alternative reinforcement (Dallery et al., 2007). Taken together, the evidence of relatively high reliability of DRD over time and evidence revealing a relationship between baseline DRD and clinically-relevant outcomes suggest that high levels of this form of impulsivity are associated with less favorable treatment response.

The objective of the current study was to examine further the relationship between DRD and smoking cessation treatment response. Participants were treatment-seeking smokers who were also heavy drinkers and were enrolled in a randomized controlled trial (RCT) of smoking cessation treatment (i.e., combined nicotine replacement and in-person counseling) plus either a brief alcohol intervention or a control placebo treatment module (Kahler et al., 2008). Delayed reward discounting was assessed prior to treatment and its relationship was prospectively examined in relation to treatment outcomes. We predicted that DRD would significantly predict time to first lapse (i.e., smoking any amount on or beyond quit day; Shiffman, Gny, Richards, Paty, Hickcox, and Kassel, 1996) over the six-month period following the individual's quit day. Time to first lapse was selected as the outcome of interest because it has been consistently implicated as a key variable in leading to long-term relapse (Brandon et al., 1990; Kenford et al., 1994; Shiffman et al., 1996). In addition, we examined two other relevant variables, nicotine dependence and sensation-seeking orientation, to determine whether the observed relationships would be specifically attributable to DRD. Nicotine dependence is well established as being associated with treatment response (Piasecki, 2006) and sensation-seeking and DRD both reflect related aspects of reward orientation and are each also related to smoking behavior (e.g., Carton et al., 1994, 2000; Kahler et al., in press). For example, sensation seeking in smokers has been found to be positively related to nicotine dependence has been found to be positively related to anhedonia and fatigue during nicotine replacement therapy (Carton et al., 2000) and, in some studies, has been found to predict treatment outcome (Kahler et al., in press), although not others (Carton et al., 2000). As such, we considered it important to assess the potential overlap among these variables, predicting that DRD would independently predict treatment response.

2. Method

2.1. Participants

Participants were 66 treatment-seeking smokers from a larger sample of 236 who participated in a smoking cessation RCT. To be included in the RCT, participants had to: (a) be at least 18 years of age; (b) have smoked cigarettes regularly for at least one year; (c) currently smoke at least 10 cigarettes a day; (d) currently use no other tobacco products or nicotine replacement therapy; and (e) currently drink heavily according to NIAAA guidelines (National Institute on Alcohol Abuse and Alcoholism, 1995): for men, >14 drinks per week or ≥5 drinks per occasion at least once per month over the past 12 months; for women, >7 drinks per week or ≥4 drinks per occasion at least once per month. Participants were excluded if they: (a) met full DSM-IV criteria for alcohol dependence in the past 12 months; (b) met criteria for other current psychoactive substance abuse or dependence (excluding nicotine dependence and alcohol abuse) in the past 12 months; (c) had a current (past month) affective disorder; (d) were psychotic or suicidal; (e) had an unstable medical condition that would suggest caution in the use of the nicotine patch (e.g., unstable angina, arrhythmia, recent congestive heart failure); (f) were currently pregnant, lactating, or intended to become pregnant. Smokers had to agree

to be available for 6 months and not to seek other smoking cessation treatment during the active phase of treatment (i.e., during the 8 weeks after their quit date while on the nicotine patch). Only a minority of individuals from the larger sample participated in the study because the DRD measure was introduced at a later stage of the RCT. Of the 66 assessed for DRD, 9 participants were lost to follow-up, leaving a final sample of 57. Participants were 61% male and primarily Caucasian (85% Caucasian; 5% African-American, 5% Latino, 4% mixed race, 2% “other”). Participants’ mean age was 41.38 (SD = 13.21) and median income was \$40,000-\$49,999 per year. In terms of tobacco-related variables, the mean score on the FTND was 4.98 (SD = 2.65), participants smoked a mean of 20.92 (SD = 10.70) cigarettes per day, and had been smoking daily for 22.8 years (SD = 12.27). Participants drank approximately 2-3 standard alcoholic drinks per day (M = 2.59, SD = 2.03). Independent samples t-tests were used to compare the participants who were lost to follow-up to those who were assessed for the full six-months. No differences were found in terms of nicotine dependence, cigarettes/day, years of daily smoking, drinks/day, delayed reward discounting, or sensation-seeking (not shown).

2.2. Treatment

Treatment consisted of four individual counseling sessions over three weeks and eight weeks of nicotine replacement therapy (NRT). Standard treatment was based on clinical practice guidelines (Fiore et al., 2000) and focused on problem-solving regarding high-risk situations for smoking relapse, providing empathic support within the treatment, and encouraging participants to seek social support for quitting smoking outside of treatment. The quit date occurred at session two, one week after session one. Participants in the standard treatment condition (ST) were given brief advice to avoid or reduce drinking as much as possible while quitting smoking. In the standard treatment + brief alcohol intervention condition (ST-BI), a more extensive discussion of alcohol use was reserved for the second half of sessions one and two. All participants received NRT consisting of 21 mg for four weeks, followed by two weeks of 14 mg patch, and then two weeks of 7 mg patch. Sessions ranged in length from 70 minutes for session one, 40 minutes for session two, and 20 minutes for sessions three and four. The ST and ST-BI conditions were matched on treatment contact time. In ST, 40 minutes of session one and 20 minutes of session two were dedicated to an active placebo intervention, progressive muscle relaxation, which has not been shown to improve smoking cessation outcomes (Fiore et al., 2000). Sessions three and four contained 5-minute check-ins regarding use of relaxation skills. In ST-BI, the same amount of time was dedicated to discussion of the participant’s alcohol use. Smoking post-quit date was systematically assessed across the follow-up period at two-weeks (end of psychosocial treatment), eight-weeks (end of pharmacological treatment), sixteen weeks, and twenty-six weeks. Additional details of the larger RCT are provided in Kahler et al. (2008).

2.3. Assessment

2.3.1. Demographics—Participants provided demographic and background information, such as age, sex, race and income.

2.3.2. Delayed Reward Discounting—Delayed reward discounting was assessed using the Monetary Choice Questionnaire (MCQ; Kirby et al., 1999), a validated self-report measure of DRD. Individuals made 27 hypothetical choices between smaller immediate rewards and larger delayed rewards that were pre-configured at various levels of hyperbolic discounting. The observed pattern of responding can be used to determine an estimate of the individual’s overall temporal discounting function, commonly referred to as k , and temporal discounting of rewards at three levels of magnitude (Small: \$25-35; Medium: \$50-60; Large: \$75-85).

2.3.3. Nicotine Dependence and Smoking Behavior—Severity of nicotine dependence at enrollment was assessed using the Fagerström Test for Nicotine Dependence (FTND;

Heatherton et al., 1991), a well-validated six-item measure. Smoking behavior during the intervention and follow-up period was assessed via self-reported smoking on a calendar-based assessment and self-reported abstinence was verified by alveolar carbon monoxide (CO) using a Bedfont Scientific Smokerlyzer® breath CO monitor. Abstinence was confirmed by a CO $\leq$ 10 ppm (SRNT Subcommittee on Biochemical Verification, 2002). Significant other report was used to verify smoking status in those instances when an individual did not provide biochemical verification of abstinence. Of 228 total assessments, abstinence from smoking was verified by significant others in three instances.

2.3.4. Sensation Seeking—Participants completed a brief version of the Sensation Seeking Scale (SSS-B; Stephenson et al., 2003), which is a four-item psychometrically validated measure of proneness toward engaging in novel, stimulating, and unusual experiences. The SSS-B had adequate internal reliability in this sample, $\alpha = .80$.

2.3.5. Alcohol Use—Drinking during the eight weeks prior to enrollment was assessed using the Timeline Followback (TLFB) (Sobell et al., 1979), which has been validated for accurately assessing drinking during a circumscribed period of time when administered under the conditions of confidentiality and zero blood alcohol. The TLFB also was conducted at sessions three and four, and at each follow-up interval for the period since its last administration. Smoking and nicotine replacement use also were assessed with the TLFB.

2.4. Procedures

All study procedures were integrated into the larger RCT. Baseline assessments were conducted during the first treatment session, prior to meeting with the therapist for the first time. Time to first lapse was subsequently assessed during treatment and throughout the follow-up period. Participants were assessed for smoking behavior at 2, 8, 16, and 26 weeks.

2.5. Data Analysis

In preliminary analyses, all variables were initially screened for distribution abnormalities and outliers ($Z > 3.29$; Tabachnick & Fidell, 2001). No data were missing. As a validity check of the DRD measure, a magnitude effect (i.e., greater discounting for smaller rewards than larger rewards) was examined using a within-subjects analysis of variance (ANOVA). In addition, t -tests were used to examine potential treatment group and sex differences. Pearson product-moment (r) correlations were used to examine interrelationships between DRD and potential covariates. The principal analyses examined DRD as a predictor of days to smoking lapse using two approaches. First, survival analysis used Cox proportional-hazards regression modeling to examine the independent effect of DRD on survival to a smoking lapse in combination with only treatment condition. All analyses included treatment condition, even when it was determined to be statistically nonsignificant, to reflect the fact that the participants did experience two different treatments. In addition, the same approach was used to examine the effect of DRD on survival to a lapse with treatment condition and other potentially relevant variables as covariates, including nicotine dependence (i.e., FTND), sensation-seeking (i.e., SSS), income, and alcohol use. The covariates were determined by examining the correlations between the indices of discounting and the preceding candidates; variables that were significantly correlated with discounting were covaried. For all Cox proportional-hazards regressions, continuous variables, including DRD, were standardized for interpretational clarity. In models with only DRD and treatment condition, both variables were entered into a single block and significance was determined by the regression coefficient. In models including covariates, DRD was entered into a block beyond the other variables of interest. The relationship of discounting was determined by a χ^2 test of the addition of the variable beyond the initial block and the significance of the observed regression coefficient. Significance of the other variables included in the covariate models was also determined by the significance of the

regression coefficient. In addition to examining survival to a first lapse, a second approach used dichotomous comparisons to examine the role of discounting at the specific follow-up time points. Baseline DRD was compared between individuals who remained abstinent and the individuals who experienced a lapse at each of the four point prevalence assessment points (two weeks, eight weeks, sixteen weeks, twenty-six weeks) using analyses of covariance (ANCOVAs), covarying treatment condition.

A significance criterion of $\alpha \leq .05$ was used for all analyses. Effect sizes reported for were hazard ratios for Cox regression models and η_p^2 for ANCOVAs. All analyses were conducted using SPSS 16.0.

3. Results

3.1. Preliminary Analyses

No significant differences were evident based on treatment for overall discounting ($t[55] = -.60, p = .55$), large magnitude discounting ($t[55] = -1.33, p = .19$), medium magnitude discounting ($t[55] = -.76, p = .45$), and small magnitude discounting ($t[55] = -.003, p = .99$). No sex differences were evident for overall discounting ($t[55] = -.53, p = .60$), large magnitude discounting ($t[55] = -.06, p = .95$), medium magnitude discounting ($t[55] = -.93, p = .36$), and small magnitude discounting ($t[55] = -.006, p = .99$). A one-way three-group ANOVA revealed a magnitude effect ($F[2, 130] = 5.35, p < .006$), with follow-up pairwise comparisons revealing significant differences ($ps < .01$) between large magnitude k values ($M = .045$ [$SE = .01$]) and both medium ($M = .057$ [$SE = .01$]) and small magnitude k values ($M = .062$ [$SE = .01$]), but no differences between medium and small magnitude discounting. Pearson product-moment correlations between discounting and potential covariates are presented in Table 1. Consistent with previous studies, high correlations were observed among the levels of discounting, indicating that, in spite of the magnitude effect, discounting at one magnitude generally corresponded with discounting at another magnitude. In addition, a significant negative correlation was observed between the discounting variables and income, and a significant positive correlation was observed between the discounting variables and nicotine dependence; all three variables were subsequently included in the follow-up analyses as covariates.

3.2. Principal Analyses

The overall six-month smoking lapse survival curve is presented in Figure 1. Cox proportional-hazards regression models of discounting as an independent predictor of smoking lapse, controlling for treatment condition, are presented in Table 2. Overall DRD and all three magnitudes significantly predicted days to first lapse. The relationship was such that an increase of one standard score on delay discounting increased the hazard, or risk of having a lapse, by 40-50%. For illustrative purposes, graphical depictions of the discounting curves for individuals who immediately lapsed to smoking on their quit day ($n = 5$) and individuals who maintained sustained verified abstinence for the six-month follow-up ($n = 7$) are presented in Figure 2. The estimated overall discounting function is presented because it exhibited among the largest effect sizes and is most representative of the individuals' general temporal discounting function. The discounting function is applied to the mean amount of money for the discounting measures choices (\$55) over a 100-day period.

For the covariate models, Cox proportional-hazards regression models of discounting, treatment condition, income, sensation-seeking, and nicotine dependence as predictors of hazard to smoking lapse are presented in Table 3. The addition of overall DRD to the other variables significantly improved the model (χ^2 [DF = 1] = 4.56, $p < .05$) and both discounting and sensation-seeking contributed to the hazard for a cigarette lapse. This was also the case for large magnitude discounting (χ^2 [DF = 1] = 4.58, $p < .05$), medium magnitude discounting

(χ^2 [DF = 1] = 6.72, $p < .01$), but not small magnitude discounting (χ^2 [DF = 1] = 2.00, $p = .16$), where no predictors reached statistical significance. In general, although the level of statistical significance was reduced in the covariate models, the discounting variables' hazard ratios were largely unaffected by the addition of the other variables.

For the dichotomous comparisons, the proportions of individuals who experienced a lapse at each of the point prevalence assessments were as follow: 2 Weeks = 60%, 8 Weeks = 77%, 16 Weeks = 86%, and 26 Weeks = 88%. Logistic regression revealed that treatment condition was not significantly associated with differences in lapse point prevalence at any of the three time points. Table 4 provides means, standard errors, F-ratios and statistical significance for individual ANCOVAs comparing lapsed and abstinent individuals and covarying treatment condition. At two weeks, successfully abstinent individuals had significantly lower overall discounting values compared to individuals who lapsed and also exhibited trend-level differences for large and medium magnitude discounting ($ps = .09$ and $.06$, respectively). At eight weeks, successfully abstinent individuals had reported significantly lower baseline discounting values at all magnitudes compared to individuals who lapsed. At sixteen weeks, no significant differences were evident between groups, although a trend-level difference was evident for medium magnitude discounting. At twenty-six weeks, no significant differences were evident between groups.

To further affirm these findings, the principal analyses were re-run excluding the three individuals for whom abstinence was not biochemically verified, but depended on the significant other reports. Significance and nonsignificance was the same for the vast majority of analyses, with minor reductions in significance levels due to reduced power (not shown). More importantly, effect sizes across analyses were virtually identical, indicating a negligible effect of excluding these individuals.

4. Discussion

The current study examined whether DRD prospectively predicted smoking cessation outcomes in smokers who were also heavy drinkers and were participating in a smoking cessation RCT. Consistent with our predictions, survival analysis revealed that greater impulsivity in terms of DRD was inversely associated with maintaining smoking abstinence. More precipitous discounting of delayed rewards was associated with fewer days until a smoking lapse. This relationship is clearly illustrated in Figure 2, where prominent differences are evident between those individuals who immediately lapsed and those who successfully maintained abstinence for the full six-month follow-up period. These relationships were independent of the treatment condition in the larger RCT and of other potentially overlapping variables. Overall, large magnitude, and medium magnitude discounting still significantly predicted days to first lapse with the inclusion of all candidate covariates, although small magnitude discounting did not. In general, the odds-ratio magnitudes were only modestly affected by the inclusion of the other variables.

A second analytic strategy compared individuals over the course of the follow-ups and may clarify the preceding findings. At two week follow-up, baseline DRD was more impulsive in individuals who had lapsed, as indicated by both significant and statistical trend-level differences in DRD between groups. At eight week follow-up, more impulsive baseline DRD was even more clearly associated with having lapsed, with differences in DRD overall and at all three magnitudes statistically significant. No significant differences were evident at the sixteen week and twenty six week follow-up, which suggests that DRD may be most relevant to early treatment lapses, however, this interpretation should be tempered by the relatively small number of abstinent individuals at the sixteen week follow-up.

These findings support the hypothesis that greater discounting of delayed rewards is associated with poorer smoking cessation treatment response. In particular, in this study we found that DRD was associated with time to initial lapse, a critical element in smoking cessation. Time to first lapse is important because initial lapses have been demonstrated to substantially increase the probability of full relapse to smoking (Brandon et al., 1990; Kenford et al., 1994; Shiffman et al., 1996). For example, two large NRT efficacy trials revealed that 83% and 97% of individuals who had an early initial lapse progressed to regular smoking again (Kenford et al., 1994). As such, these findings complement previous studies of adolescents and pregnant women in demonstrating this negative association (Krishnan-Sarin et al., 2007; Yoon et al., 2007) and substantiate the association observed in a laboratory model of smoking lapse (Dallery & Raiff, 2007). A clear implication of the current study and these previous investigations is that high DRD constitutes a risk factor for poor smoking cessation treatment response.

As a risk factor for treatment failure, these results suggest that smokers who are high in DRD will require modifications to typical smoking cessation treatments in order to successfully quit smoking. More effective treatment for high DRD smokers could come in several forms. On one hand, it may be that high DRD smokers simply need quantitatively more treatment. According to the recent recommendations from the United States Surgeon General (Fiore et al., 2008), there are a number of effective pharmacological and psychosocial treatment elements, and there is also evidence that their effects are additive. The more empirically-supported elements a treatment program has, the better the prospects of individuals attempting to quit. As such, it may be that smokers with high DRD would fare better in response to a more intensive treatment with a larger array of empirically-supported treatment elements (e.g., multiple forms of NRT, multiple forms of behavioral intervention). Unfortunately, such intensive multi-model treatments are likely to be relatively inaccessible to the general public due to increased costs. Nonetheless, smokers who are motivated to quit but refractory to existing standard treatments, such approaches may be necessary.

Alternatively, it may be that highly impulsive smokers will require qualitatively different treatments. The current study included a treatment with many of the recommended treatment components (i.e., first-line pharmacotherapy [i.e., NRT, wellbutrin, and varenicline], problem-solving-based psychosocial counseling, and empathic supportive counseling; Fiore et al., 2008) and those individuals who were high in DRD tended not to fare well. As such, it may be that existing strategies are suboptimal for high-DRD smokers, hence the association with treatment failure in this study, and that new treatment approaches, either behavioral or pharmacological, will be necessary to improve outcomes for these individuals. In particular, approaches that target altering or attenuating preferences for immediate rewards may be the most promising in this capacity. For example, initial preclinical findings found that the opioid antagonist naltrexone has some promise in reducing DRD (Kieres et al., 2004). However, a subsequent study using alcoholics and controls did not observe any effects of naltrexone on discounting (Mitchell et al., 2007) and progress in identifying strategies for reducing DRD is still in its infancy. At this point, however, whether quantitatively greater or qualitatively different treatments would be more useful for more high-DRD smokers is necessarily speculative. The current findings nonetheless further implicate high levels of DRD as a risk factor for treatment failure and suggest that identifying ways to improve treatment response in impulsive smokers should be a priority.

Although this study provides relatively clear evidence of the relationship between DRD and treatment response, what remains unclear is the cause of the variation in DRD. This can be partially addressed by observing whether DRD was meaningfully associated with other variables in the study. A significant correlation was evident between DRD and income, but this association does not provide much insight into the role of DRD. Lower income was

associated with greater discounting (i.e., greater valuation of immediate rewards and devaluation of delayed rewards), which is not surprising, but income was not associated with other smoking-related variables or treatment response, suggesting the relationship reflects a general relationship between income and discounting that is not specific to smoking. Another possibility is that high-levels of discounting develop as a person becomes increasingly dependent on nicotine and repeatedly selects small immediate rewards (i.e., cigarettes) over larger delayed rewards (e.g., physical health). There is some support for this in the current study, where DRD and FTND were significantly correlated. However, nicotine dependence was not associated with time to lapse, indicating that the overlap between the variables was not responsible for the DRD's predictive power. Put another way, DRD was independently associated with both nicotine dependence and time to first lapse in this study. Sensation-seeking proneness provided a similar case. Sensation-seeking also predicted time to first lapse, but sensation-seeking and DRD were not significantly associated with each other, and DRD predicted time to lapse beyond the covariate model. Taken together, the other measured variables provide relatively little clarification into the influence of DRD on treatment outcome, suggesting future studies in this area might benefit from including other potentially relevant variables that were not measured in this study.

Of note, lack of association between DRD and sensation-seeking merits discussion in itself. Although both constructs reflect aspects of reward orientation (i.e., devaluation of delayed rewards versus preference for novel, stimulating experiences, respectively) and both are related to smoking behavior (Baker et al., 2003; Bickel et al., 1999; Carton et al., 1994, 2000), a number of studies have not found a significant positive or negative relationship between the two (Dom et al., 2006; Eisenberg et al., 2007; Perkins et al., 2008). Although it is evidence in the form of a null result and may be specific to the sample studied, this study provides further evidence that sensation-seeking and DRD should be thought of as distinct constructs.

The current findings should be considered in light of the study's strengths and weaknesses. Among the strengths are that the study was conducted within the context of a high-quality RCT using the recommended standard of care for smoking cessation treatment and that participants were closely monitored over a relatively long follow-up period. However, the study also had several limitations. First, the sample size was relatively small, and this may have contributed to the observed changes in statistical significance in covariate models when larger numbers of variables were included. This was also an issue when comparing individuals at later points during follow-up, when cell sizes became increasingly unbalanced and statistical power was substantially diminished. Second, the objective criterion for abstinence of $CO \leq 10\text{ppm}$ is a somewhat lenient standard and could be achieved with some low level of smoking. As such, it is possible a lapse could have been missed. Third, participants were only assessed for DRD at enrollment and a second assessment in individuals who did successfully quit smoking would have allowed a direct test of whether changes in discounting take place over the course of successful cessation. Finally, as noted above, it is worthwhile to note that the findings pertain to the specific sample recruited and treatment provided. Participants were treatment-seeking smokers who were also heavy drinkers, although drinking and discounting were unrelated in this study, and the core treatment was a combination of NRT and psychotherapy. Although this study generated findings in this sample that were similar to samples of adolescent smokers and pregnant female smokers (Krishnan-Sarin et al., 2007; Yoon et al., 2007), to fully establish high DRD as a reliable risk factor for poor smoking cessation outcome, it will be important to establish the study's generalizability via replication in larger and more representative samples, and with other empirically-supported treatments.

These limitations notwithstanding, this study nonetheless extends what is known about the relationship between DRD and smoking cessation. The study prospectively examined the relationship between DRD and smoking cessation treatment response and, as hypothesized,

found that greater devaluation of delayed rewards was indeed associated with poorer treatment response. These findings suggest that individuals who are high in DRD may need quantitatively more or qualitatively different smoking cessation treatments to successfully quit smoking. Further, the study raises the question of the nature of these differences in impulsivity in relation to smoking cessation. Directly examining these questions is a worthwhile prospect for future studies.

References

- Ainslie G. Specious reward: a behavioral theory of impulsiveness and impulse control. *Psychol. Bull* 1975;82:463–496. [PubMed: 1099599]
- Ainslie, G. Breakdown of Will. Cambridge University Press; 2001.
- Baker F, Johnson MW, Bickel WK. Delay discounting in current and never-before cigarette smokers: similarities and differences across commodity, sign, and magnitude. *J. Abnorm. Psychol* 2003;112:382–392. [PubMed: 12943017]
- Bickel, WK.; Johnson, MW. Delay discounting: A Fundamental Behavioral Process of Drug Dependence. In: Loewenstein, G.; Read, D.; Baumeister, RF., editors. *Time and Decision: Economic and Psychological Perspectives on Intertemporal Choice*. Russell Sage Foundation; New York: 2003. p. 419–440.
- Bickel WK, Marsch LA. Toward a behavioral economic understanding of drug dependence: delay discounting processes. *Addiction* 2001;96:73–86. [PubMed: 11177521]
- Bickel WK, Odum AL, Madden GJ. Impulsivity and cigarette smoking: delay discounting in current, never, and ex-smokers. *Psychopharmacology (Berl.)* 1999;146:447–454. [PubMed: 10550495]
- Bigelow G, Cohen M, Liebson I, Faillace LA. Abstinence or moderation? Choice by alcoholics. *Behav. Res. Ther* 1972;10:209–214. [PubMed: 5047678]
- Bjork JM, Hommer DW, Grant SJ, Danube C. Impulsivity in abstinent alcohol-dependent patients: relation to control subjects and type 1-/type 2-like traits. *Alcohol* 2004;34:133–150. [PubMed: 15902907]
- Boettiger CA, Mitchell JM, Tavares VC, Robertson M, Joslyn G, D'Esposito M, Fields HL. Immediate reward bias in humans: fronto-parietal networks and a role for the catechol-O-methyltransferase 158 (Val/Val) genotype. *J. Neurosci* 2007;27:14383–14391. [PubMed: 18160646]
- Brandon TH, Tiffany ST, Obremski KM, Baker TB. Postcessation cigarette use: the process of relapse. *Addict. Behav* 1990;15:105–114. [PubMed: 2343783]
- Camerer C. Behavioral economics: reunifying psychology and economics. *Proc. Natl. Acad. Sci. U S A* 1999;96:10575–10577. [PubMed: 10485865]
- Carton S, Jouvent R, Widlocher D. Sensation seeking, nicotine dependence, and smoking motivation in female and male smokers. *Addict. Behav* 1994;19:219–227. [PubMed: 7942242]
- Carton S, Le Houezec J, Lagrue G, Jouvent R. Relationships between sensation seeking and emotional symptomatology during smoking cessation with nicotine patch therapy. *Addict. Behav* 2000;25:653–662. [PubMed: 11023009]
- Coffey SF, Gudleski GD, Saladin ME, Brady KT. Impulsivity and rapid discounting of delayed hypothetical rewards in cocaine-dependent individuals. *Exp. Clin. Psychopharmacol* 2003;11:18–25. [PubMed: 12622340]
- Dallery J, Raiff BR. Delay discounting predicts cigarette smoking in a laboratory model of abstinence reinforcement. *Psychopharmacology (Berl.)* 2007;190:485–496. [PubMed: 17205320]
- Dom G, D'Haene P, Hulstijn W, Sabbe B. Impulsivity in abstinent early- and late-onset alcoholics: differences in self-report measures and a discounting task. *Addiction* 2006;101:50–59. [PubMed: 16393191]
- Field M, Christiansen P, Cole J, Goudie A. Delay discounting and the alcohol Stroop in heavy drinking adolescents. *Addiction* 2007;102:579–586. [PubMed: 17309540]
- Fiore, MC.; Bailey, WC.; Cohen, SJ.; Dorfman, SF.; Goldstein, MG.; Gritz, ER. Treating tobacco use and dependence: Clinical practice guideline. U.S. Department of Health and Human Services, Public Health Service; Rockville, MD: 2000.

- Treating Tobacco Use and Dependence Guideline Panel. Treating Tobacco Use and Dependence: 2008 Update. U.S. Department of Health and Human Services, Public Health Service; Rockville, MD: 2008.
- Griffiths RR, Rush CR, Puhala KA. Validation of the multiple-choice procedure for investigating drug reinforcement in humans. *Exp. Clin. Psychopharmacol* 1996;4:97–106.
- Heatherton TF, Kozlowski LT, Frecker RC, Fagerstrom KO. The Fagerstrom Test for Nicotine Dependence: a revision of the Fagerstrom Tolerance Questionnaire. *Br. J. Addict* 1991;86:1119–1127. [PubMed: 1932883]
- Higgins ST, Bickel WK, Hughes JR. Influence of an alternative reinforcer on human cocaine self-administration. *Life Sci* 1994;55:179–187. [PubMed: 8007760]
- Johnson MW, Bickel WK, Kirshenbaum AP. Substitutes for tobacco smoking: a behavioral economic analysis of nicotine gum, denicotinized cigarettes, and nicotine-containing cigarettes. *Drug Alcohol Depend* 2004;74:253–264. [PubMed: 15194203]
- Kahler CW, Metrik J, LaChance HR, Ramsey SE, Abrams DB, Monti PM, Brown RA. Addressing heavy drinking in smoking cessation treatment: a randomized clinical trial. *J. Consult. Clin. Psychol* 2008;76:852–862. [PubMed: 18837602]
- Kahler CW, Spillane NS, Metrik J, Leventhal AM, Monti PM. Sensation seeking as a predictor of treatment compliance and smoking cessation treatment outcomes in heavy social drinkers. *Pharmacol. Biochem. Behav.* in press
- Kahneman D. A perspective on judgment and choice: mapping bounded rationality. *Am. Psychol* 2003;58:697–720. [PubMed: 14584987]
- Kenford SL, Fiore MC, Jorenby DE, Smith SS, Wetter D, Baker TB. Predicting smoking cessation. Who will quit with and without the nicotine patch. *JAMA* 1994;271:589–594. [PubMed: 8301790]
- Kirby KN, Petry NM, Bickel WK. Heroin addicts have higher discount rates for delayed rewards than non-drug-using controls. *J. Exp. Psychol. Gen* 1999;128:78–87. [PubMed: 10100392]
- Krishnan-Sarin S, Reynolds B, Duhig AM, Smith A, Liss T, McFetridge A, Cavallo DA, Carroll KM, Potenza MN. Behavioral impulsivity predicts treatment outcome in a smoking cessation program for adolescent smokers. *Drug Alcohol Depend* 2007;88:79–82. [PubMed: 17049754]
- Loewenstein G. Out of control: Visceral Influences on Behavior. *Organ. Behav. Hum. Decis. Process* 1996;65:272–292.
- Madden GJ, Petry NM, Badger GJ, Bickel WK. Impulsive and self-control choices in opioid-dependent patients and non-drug-using control participants: drug and monetary rewards. *Exp. Clin. Psychopharmacol* 1997;5:256–262. [PubMed: 9260073]
- Mitchell JM, Fields HL, D'Esposito M, Boettiger CA. Impulsive responding in alcoholics. *Alcohol. Clin. Exp. Res* 2005;29:2158–2169. [PubMed: 16385186]
- Mitchell JM, Tavares VC, Fields HL, D'Esposito M, Boettiger CA. Endogenous opioid blockade and impulsive responding in alcoholics and healthy controls. *Neuropsychopharmacology* 2007;32:439–449. [PubMed: 17047667]
- Mitchell SH. Measures of impulsivity in cigarette smokers and non-smokers. *Psychopharmacology (Berl.)* 1999;146:455–464. [PubMed: 10550496]
- National Institute on Alcohol Abuse and Alcoholism. Helping patients who drink too much: a clinician's guide. U.S. Department of Health and Human Services, National Institutes of Health, National Institute on Alcohol Abuse and Alcoholism; 2005.
- Ohmura Y, Takahashi T, Kitamura N, Wehr P. Three-month stability of delay and probability discounting measures. *Exp. Clin. Psychopharmacol* 2006;14:318–328. [PubMed: 16893275]
- Perkins KA, Lerman C, Coddington SB, Jetton C, Karelitz JL, Scott JA, Wilson AS. Initial nicotine sensitivity in humans as a function of impulsivity. *Psychopharmacology (Berl)* 2008;200:529–544. [PubMed: 18604520]
- Petry NM. Delay discounting of money and alcohol in actively using alcoholics, currently abstinent alcoholics, and controls. *Psychopharmacology (Berl)* 2001a;154:243–250. [PubMed: 11351931]
- Petry NM. Substance abuse, pathological gambling, and impulsiveness. *Drug Alcohol Depend* 2001b;63:29–38. [PubMed: 11297829]
- Piasecki TM. Relapse to smoking. *Clin Psychol Rev* 2006;26:196–215. [PubMed: 16352382]

- Rachlin H, Raineri A, Cross D. Subjective probability and delay. *J. Exp. Anal. Behav* 1991;55:233–244. [PubMed: 2037827]
- Reynolds B. The Experiential Discounting Task is sensitive to cigarette-smoking status and correlates with a measure of delay discounting. *Behav. Pharmacol* 2006;17:133–142. [PubMed: 16495721]
- Reynolds B, Richards JB, Horn K, Karraker K. Delay discounting and probability discounting as related to cigarette smoking status in adults. *Behav. Processes* 2004;65:35–42. [PubMed: 14744545]
- Shiffman S, Hickcox M, Paty JA, Gnys M, Kassel JD, Richards TJ. Progression from a smoking lapse to relapse: prediction from abstinence violation effects, nicotine dependence, and lapse characteristics. *J. Consult. Clin. Psychol* 1996;64:993–1002. [PubMed: 8916628]
- Sobell LC, Maisto SA, Sobell MB, Cooper AM. Reliability of alcohol abusers' self-reports of drinking behavior. *Behav. Res. Ther* 1979;17:157–160. [PubMed: 426744]
- SRNT Subcommittee on Biochemical Verification. Biochemical verification of tobacco use and cessation. *Nicotine Tob. Res* 2002;4:149–159. [PubMed: 12028847]
- Stephenson MT, Hoyle RH, Palmgreen P, Slater MD. Brief measures of sensation seeking for screening and large-scale surveys. *Drug Alcohol Depend* 2003;72:279–286. [PubMed: 14643945]
- Tabachnick, BG.; Fidell, LS. *Using Multivariate Statistics*. Allyn & Bacon; Needham Heights, MA: 2001.
- Takahashi T, Furukawa A, Miyakawa T, Maesato H, Higuchi S. Two-month stability of hyperbolic discount rates for delayed monetary gains in abstinent inpatient alcoholics. *Neuro. Endocrinol. Lett* 2007;28:131–136. [PubMed: 17435675]
- Vuchinich, RE.; Heather, N., editors. *Choice, behavioural economics and addiction*. Pergamon/Elsevier Science; Amsterdam, The Netherlands: 2003.
- Vuchinich RE, Simpson CA. Hyperbolic temporal discounting in social drinkers and problem drinkers. *Exp. Clin. Psychopharmacol* 1998;6:292–305. [PubMed: 9725113]
- Yoon JH, Higgins ST, Heil SH, Sugarbaker RJ, Thomas CS, Badger GJ. Delay discounting predicts postpartum relapse to cigarette smoking among pregnant women. *Exp. Clin. Psychopharmacol* 2007;15:176–186. [PubMed: 17469941]

Figure 1. Overall survival function of days to first lapse. Days to first lapse is provided on the X-axis, from 0 to 182, and the proportion of individuals who did not lapse is provided on the Y-axis.

Figure 2. Overall discounting curves for individuals who immediately lapsed (zero days abstinent) and individuals who achieved continuous verified abstinence for 182 days. The estimated temporal discounting function (i.e., k) is applied to the mean monetary value (\$55) in the measure over a 100 day period.

Table 1
Correlations among indices of delayed reward discounting, other individual difference measures, and days to first lapse (n =57)

	1	2	3	4	5	6	7	8	9
1. Overall <i>k</i>	1								
2. Large Magnitude <i>k</i>	.88***	1							
3. Medium Magnitude <i>k</i>	.90***	.89***	1						
4. Small Magnitude <i>k</i>	.87***	.81***	.78***	1					
5. Income	-.36**	-.27*	-.19	-.26*	1				
6. FTND	.41**	.33**	.33**	.42***	-.34**	1			
7. SSS	-.15	-.16	-.15	-.08	.28*	.11	1		
8. Daily Alcohol Consumption	.19	.12	.12	.20	-.26*	.38**	.20	1	
9. Days to Lapse	.27*	-.24 [†]	-.28*	-.26 [†]	.08	-.21	-.20	-.24 [†]	1

Note: Temporal discounting functions are denoted by *k*; FTND = Fagerström Test of Nicotine Dependence; SSS = Sensation Seeking Scale

[†]
p ≤ .10
*
p ≤ .05
**
p ≤ .01
***
p ≤ .001

Table 2

Cox proportional-hazard regression models of all indices of delayed reward discounting as an independent predictor of days to first smoking lapse. Each model represents a separate regression analyses (N = 57)

Model	Hazard Ratio	95% CI	<i>p</i>
<i>Overall Discounting</i>			
Treatment Condition	.84	.49-1.44	.52
Overall <i>k</i>	1.48	1.12-1.96	≤ .01
<i>Large Magnitude Discounting</i>			
Treatment Condition	.85	.50-1.46	.55
Large Magnitude <i>k</i>	1.43	1.09-1.88	≤ .01
<i>Medium Magnitude Discounting</i>			
Treatment Condition	.84	.49-1.45	.54
Medium Magnitude <i>k</i>	1.53	1.16-2.02	< .01
<i>Small Magnitude Discounting</i>			
Treatment Condition	.92	.54-1.56	.76
Small Magnitude <i>k</i>	1.41	1.06-1.89	≤ .05

Table 3

Cox proportional-hazard regression models of all indices of delayed reward discounting as an incremental predictor of days to first smoking lapse with treatment condition, nicotine dependence, and income as covariates. Each model represents a separate regression analyses (N = 57)

Model	Hazard Ratio	95% CI	p
<i>Overall Discounting</i>			
Treatment Condition	.81	.47-1.39	.44
Income	1.05	.76-1.45	.78
SSS	1.40	1.05-1.88	<.05
FTND	.86	.63-1.17	.33
Overall <i>k</i>	1.45	1.04-2.01	<.05
<i>Large Magnitude Discounting</i>			
Treatment Condition	.84	.49-1.44	.53
Income	1.09	.79-1.49	.61
SSS	1.42	1.54-1.91	<.05
FTND	.83	6.1-1.13	.26
Large <i>k</i>	1.41	1.05-1.90	<.05
<i>Medium Magnitude Discounting</i>			
Treatment Condition	.83	.48-1.42	.50
Income	1.06	.77-1.46	.72
SSS	1.44	1.07-1.92	<.05
FTND	.83	.61-1.12	.22
Medium <i>k</i>	1.53	1.13-2.07	<.01
<i>Small Magnitude Discounting</i>			
Treatment Condition	.91	.53-1.54	.72
Income	1.10	.80-1.53	.55
SSS	1.33	.99-1.77	.06
FTND	.84	.62-1.14	.26
Small <i>k</i>	1.28	.92-1.78	.14

Note: SSS = Sensation Seeking Scale; FTND = Fagerström Test of Nicotine Dependence; *k* = temporal discounting function.

Table 4

Analyses of covariance comparing individuals who experienced a lapse to smoking and those who remained abstinent at two week, eight week, and sixteen week follow-up following quit date. Treatment condition was covaried in all analyses. Note: k = temporal discounting function

	Lapsed Mean k (SE)	Abstinent Mean k (SE)	F (1, 54)	p	η_p^2
<i>Two Weeks (ns = 34/23)</i>					
Overall k	.07 (.01)	.03 (.02)	4.42	.04	.08
Large k	.06 (.01)	.02 (.02)	3.08	.09	.05
Medium k	.07 (.01)	.03 (.02)	3.78	.06	.07
Small k	.07 (.01)	.04 (.02)	1.95	.17	.04
<i>Eight Weeks (ns = 44/13)</i>					
Overall k	.06 (.01)	.01 (.02)	5.26	.03	.09
Large k	.05 (.01)	.01 (.02)	4.44	.04	.08
Medium k	.07 (.01)	.01 (.02)	4.77	.03	.08
Small k	.07 (.01)	.02 (.02)	4.55	.04	.08
<i>Sixteen Weeks (ns = 49/8)</i>					
Overall k	.06 (.01)	.01 (.03)	2.54	.12	.05
Large k	.05 (.01)	.01 (.03)	2.00	.16	.04
Medium k	.06 (.01)	.01 (.03)	2.93	.09	.05
Small k	.07 (.01)	.02 (.03)	2.32	.13	.04
<i>Twenty-Six Weeks (ns = 50/7)</i>					
Overall k	.06 (.01)	.01 (.03)	1.95	.17	.04
Large k	.05 (.01)	.01 (.03)	1.49	.23	.03
Medium k	.06 (.01)	.01 (.03)	2.33	.13	.04
Small k	.06 (.01)	.02 (.03)	1.97	.17	.04