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Delay discounting rates: A strong prognostic indicator of relapse to smoking

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Abstract

Recent evidence suggests that several dimensions of impulsivity and locus of control are likely to be significant prognostic indicators of relapse. We compared the relative strengths of associations among delay discounting rates, dimensions of trait impulsiveness from the Barratt Impulsiveness Scale - 11, locus of control, nicotine dependence, and stress level with days to relapse among smokers after an intensive multicomponent cognitive-behavioral treatment for tobacco dependence. We used Cox proportional hazard regressions to model days to relapse with each of the following: delay discounting of \$100, delay discounting of \$1,000, six subscales of the Barratt Impulsiveness Scale (BIS), Rotter's Locus of Control (RLOC), Fagerstrom Test for Nicotine

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Dependence (FTND), and the Perceived Stress Scale (PSS). Standardized regression coefficients and hazard ratios (HRs) were used to assess the relative strength and direction of the associations along with a bootstrap method to determine 95% confidence intervals. We then examined the extent to which the measures retained associations with days to relapse while accounting for nicotine dependence and stress level. Our findings indicate that the \$100 delay discounting rate had the strongest association with days to relapse, but was only significantly stronger than nicotine dependence. Discounting rates maintained significant associations with days to relapse when combined with the FTND and the PSS, but the BIS subscales and the RLOC did not. These findings indicate that delay discounting is independently associated with relapse and adds to what is already accounted for by nicotine dependence and stress level. These findings signify that delay discounting is a productive new target for enhancing treatment for tobacco dependence. Adding an intervention designed to decrease discounting rates to a comprehensive treatment for tobacco dependence has the potential to decrease relapse rates.

Keywords

delay discounting rates; impulsivity; smoking cessation; tobacco dependence

1.0 INTRODUCTION

Tobacco use is the greatest cause of preventable death and disease in the United States (CDC, 2005; Mokdad, Marks, Stroup, & Gerberding, 2004). At present, 45 million, fully 1 in 5 Americans, smoke daily (CDC, 2011a). Even though 70% of smokers desire to quit smoking (CDC, 2011b) and about half of all smokers make a quit attempt each year (CDC, 2011b), 94% of those who attempt to quit will relapse within 6 months (CDC, 2011b; Fiore et al., 2008). Even with intensive multicomponent combination treatment, at least 70% of smokers relapse within 12 months (Fiore et al., 2008). Substantial progress has been made in addressing factors identified by well-established, strong prognostic indicators such as nicotine dependence and stress level (Fiore et al., 2008), however, the identification of additional factors would provide new targets for enhancing treatment for tobacco dependence and reducing relapse rates. Evidence is mounting for the prospective role of delay discounting, certain aspects of trait impulsiveness, and locus of control as indicators of relapse to smoking and potential targets for enhancing treatment for tobacco dependence (MacKillop & Kahler, 2009; Sheffer et al., 2012; Stanger et al., 2012; Yoon et al., 2007).

Impulsivity is a multidimensional construct often associated with nicotine dependence and smoking (Chase & Hogarth, 2011; Rezvanfard, Ekhtiari, Mokri, Djavid, & Kaviani, 2010; Sheffer et al., 2012); however, the results supporting impulsivity as a prognostic indicator of relapse have been mixed (Powell, Dawkins, West, & Pickering, 2010). Impulsivity is generally thought to subsume many aspects of reward seeking and disinhibition (Flory & Manuck, 2009; Mitchell, 1999) as well as several aspects of what is often considered trait impulsiveness (Lane et al., 2003; Reynolds et al., 2006; Meda et al., 2009). Consistent with this conceptualization, trait impulsiveness is positively associated with subjective rewarding effects of nicotine (Perkins et al., 2008) as well as explicit expectancies about nicotine reward (Doran, McChargue, & Cohen, 2007). Some have found trait impulsiveness to be

positively associated with another dimension of impulsivity, delay discounting (Audrain-McGovern, et al., 2009; Flory & Manuck, 2009; Powell et al., 2010).

Smoking provides a variety of immediate rewards (e.g., stress relief, relief from withdrawal symptoms, etc.) of value to smokers for limited immediate costs; however, the long-term costs to health, relationships, and long life are substantial. Delay discounting is the degree (i.e., the rate) to which one discounts or de-values a reward as a function of the amount of time to the receipt of that reward. Unsurprisingly, smokers discount the value of future health more than nonsmokers, but smokers also discount the value of monetary rewards more than nonsmokers (Baker, Johnson, & Bickel, 2003b; Bickel, Odum, & Madden, 1999; Bickel & Yi, 2008; Mitchell, 1999; Reynolds, 2004; Reynolds, Karraker, Horn, & Richards, 2003). Higher delay discounting rates are also associated with higher nicotine dependence levels (Sweitzer, Donny, Dierker, Flory, & Manuck, 2008) and a decreased likelihood of long-term abstinence after treatment for tobacco dependence (Krishnan-Sarin et al., 2007; MacKillop & Kahler, 2009; Sheffer et al., 2012; Stanger et al., 2012; Yoon et al., 2007), but are not always strongly associated with dimensions of trait impulsiveness among smokers (Sheffer et al., 2012).

Locus of control is the degree to which one believes that reinforcement or rewards are contingent upon internal versus external factors (Rotter, 1966). Individuals with an internally-focused locus of control believe that reinforcement is contingent upon their own efforts and tend to be less impulsive, more future-oriented, report lower levels of stress, and discount less than externally-focused individuals, while individuals with an externally-focused locus of control believe that reinforcement is contingent upon luck, fate, or powerful others (Erikson & Roberts, 1971; Platt & Eisenman, 1968; Plunkett & Buehner, 2007; Rotter, 1966; Srinivasan & Tikoo, 1992). An internally focused locus of control is a positive prognostic indicator for maintaining abstinence from smoking (Gregor, Zvolensky, McLeish, Bernstein, & Morissette, 2008; McKenna & Higgins, 1997; Rosenbaum & Argon, 1979; Sheffer et al., 2012). Externally-focused individuals appear to experience greater levels of stress (Sheffer et al., 2012).

Perceived stress level is a well-established prognostic indicator of relapse as well as a frequently mentioned barrier to quitting (Fiore et al. 2008). Stress management is, in fact, an evidence-based component in comprehensive tobacco dependence treatment programs (Fiore et al. 2008). We have found weak associations among smokers in previous work between stress level and delay discounting as well as between stress level and the dimensions of trait impulsiveness assessed by the Barratt Impulsiveness Scale with the exception of attentional/cognitive impulsiveness (Sheffer et al., 2012). Locus of control, however, is conceptually and empirically associated with stress level. Perceived stress and locus of control both incorporate perceived control over stressful events. Less perceived control over stressful events contributes to greater levels of stress (Wetter, Smith, Kenford, Fiore, et al. 1994) and an externally-focused locus of control (Rotter, 1966). In some instances, locus of control appears to moderate the relationship between external events and the experience of stress (Carter, Mollen, & Smith, 2013).

Given the tobacco-related disease burden, improving relapse rates has the potential to significantly impact public health. There are a modest number of studies that examine delay discounting rates, trait impulsiveness, and locus of control as prognostic indicators of relapse to smoking, but there is growing support for incorporating these new measures into the assessment and treatment of tobacco dependence and other addictions (Sheffer et al., 2012; Bickel, Koffarnus, Moody, & Wilson, 2014). Arguably, in order to justify these efforts, the strengths of the associations among these measures with relapse need to be at least commensurate with well-established indicators such as nicotine dependence and stress levels. These measures also need to add to what is already provided by these well-established indicators. At present, there are no studies in which these measures have been compared with nicotine dependence and/or stress or other well-established indicators.

We compared the relative strengths of associations among delay discounting, dimensions of trait impulsiveness from the Barratt Impulsiveness Scale – 11 (BIS-11), locus of control, nicotine dependence, and stress level with days to relapse among smokers after an intensive multicomponent cognitive-behavioral treatment for tobacco dependence. Although impulsivity is often associated with nicotine dependence, there are mixed findings with regard to its role as a prognostic indicator of relapse (Powell, Dawkins, West, & Pickering, 2010); therefore, we hypothesized that higher levels of trait impulsiveness would not be strongly associated with relapse. Given the consistent, strong relationship between delay discounting rates and abstinence rates in other studies (Krishnan-Sarin et al., 2007; MacKillop & Kahler, 2009; Sheffer et al., 2012; Stanger et al., 2012; Yoon et al., 2007), we hypothesized that higher delay discounting rates would be a strong indicator of relapse, perhaps as strong as nicotine dependence, a well-established indicator. Because stress and locus of control are conceptually and empirically related, we hypothesized that their associations with relapse would be strong, but not differ significantly. Further, given the conceptual and empirical evidence, we hypothesized that delay discounting and trait impulsiveness, but not locus of control would remain associated with relapse when combined with nicotine dependence and stress level in the models.

2.0 MATERIAL AND METHODS

2.1 Participants

Participants were age 18 years or older, smoked 10 or more cigarettes per day and were recruited by referral from medical center campus tobacco treatment services, word-of-mouth, and print and radio advertisements. For the purposes of this study, only those participants who reached the treatment quit date, the third treatment session, were included. Individuals who were pregnant or lactating, already using medications for smoking cessation (i.e., bupropion, varenicline, nicotine replacement, etc.) or using medications that would interfere with participation or treatment, were drinking >20 alcoholic drinks per week, tested positive for drugs of abuse (amphetamine, benzodiazepines, cannabis, cocaine, opioids, methadone), or had plans to move out of the area were excluded.

2.2 Procedure

This study was approved by the Institutional Review Board of the University of Arkansas for Medical Sciences. Prior to baseline data collection participants were required to smoke one cigarette to standardize time from the last cigarette across participants.

Participants were treated with multi-component cognitive-behavioral therapy and 8 weeks of nicotine replacement therapy (4 weeks of 21mg, 2 weeks of 14mg, and 2 weeks of 7mg patches), a well-established treatment for tobacco dependence which is considered state-of-the-art and consistent with the consensus statement and recommendations of the Public Health Service Clinical Practice Guideline.(Fiore et al., 2008). The treatment was delivered by certified Tobacco Treatment Specialists (TTSs) with a masters degree in psychology or a bachelors degree in social work and 1–2 years of experience. The approach consisted of six weekly, structured, 60-minute, closed group sessions of content delivered in clinical, community and telephone-based settings and research studies (Payne, Smith, Adams, & Diefenbach, 2006; Schmitz, Rosenfarb, & Payne, 1993; Schmitz & Tate, 1994; Sheffer, et al., 2009; Sheffer, Stitzer, Landes, Brackman, & Munn, 2013). A variety of cognitive-behavioral components were employed, including self-monitoring, stimulus control, problem-solving, conflict management, cigarette refusal training, enhancing social support, goal setting, relapse prevention, and stress management. The quit date for all participants was the day of the third treatment session. Treatment fidelity was ensured by: a) using a manual-driven treatment delivery; b) specialized training; c) using only certified TTSs; d) weekly review of all treatment sessions with a licensed psychologist (CES); e) random review of clinical notes; and f) random review of recorded sessions.

2.3 Measures

Standard demographic measures were collected including sex, race, age, years of education, marital status, income, and employment status. Multiple tobacco-related, clinical, and executive function measures were administered, but were not included in these analyses. The following measures, included in these analyses, were administered at baseline, 4 weeks after the quit date, and 27 weeks after the quit date.

Fagerström Test for Nicotine Dependence (FTND)—The FTND is a widely used six-item questionnaire assessing dependence level in smokers with extensive data attesting to its reliability and validity. Scores range from 0–10 with greater values indicating greater dependence levels and a lesser likelihood of abstinence.(Fagerstrom & Schneider, 1989; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) Sample question: “How soon after you wake up do you smoke your first cigarette?”

Perceived Stress Scale (PSS)—The PSS is a widely used 14-item questionnaire assessing stress level. Scores range from 0–56 with greater values indicating greater stress levels and predicting a lesser likelihood of abstinence.(Cohen, Kamarck, & Mermelstein, 1983; Cohen & Lichtenstein, 1990; Glasgow, Klesges, Mizes, & Pechacek, 1985; Kenford et al., 2002) Sample question: “In the last month, how often have you been upset because of something that happened unexpectedly?”

Barratt Impulsiveness Scale - 11 (BIS)—The BIS is a widely used 30-item questionnaire assessing personality/behavioral impulsiveness, often called trait impulsiveness. The instrument is comprised of three impulsivity scales with six subscales: Attentional Impulsivity (Attention, Cognitive Instability), Motor Impulsivity (Motor, Perseverance), and Non-Planning Impulsivity (Self-Control, Cognitive Complexity). Because impulsivity is a multifaceted construct, it is recommended that the scales and/or subscales be utilized whenever possible to account for their individual contributions. We utilized all three scales: Attentional Impulsivity (AI), Motor Impulsivity (MI), and Non-Planning Impulsivity (NI); and one subscale from each scale: Cognitive Instability (CI), Motor (MO), and Self-Control (SC). The BIS is internally consistent and stable measure where higher values indicate more impulsiveness (Barratt, Patton, Stanford, & Barratt, 1995). Sample question: “I plan tasks carefully.”

Delay Discounting Tasks—The delay discounting tasks assessed the degree to which the subjective value of rewards was modulated by the delay to their receipt (Logue, 1988). Participants completed one task using two amounts for delayed hypothetical money: \$100 and \$1,000. For each amount, a series of choices were presented for each of seven delays: 1 day, 1 week, 1 month, 6 months, 1 year, 5 years, and 25 years. Smaller, immediately available rewards were offered against the larger constant delayed amount. The first choice was always between the larger delayed reward and half of the delayed reward available immediately. Subsequent choices adjusted the value of the immediate choice according to whether the participant chose the immediate (adjusted down) or delayed (adjusted up) reward. The adjusted amount for the smaller immediate amount was the mid-point between the previous choice and the hypothetical indifference point as indicated by the participant's previous choices. The indifference point is the value of the immediate reward, expressed as a proportion, subjectively deemed equivalent to the larger, delayed reward. Mazur's (1987) hyperbolic discounting model was then applied to the indifference points across delays where the discounting rate k reflected a participant's degree of preference for the smaller immediate reward. The $\ln(k)$ values from both tasks were also averaged to produce a mean delay discounting score.

2.4 Outcome Assessment

Participants were contacted by telephone once per week from the date of the informed consent to 6 months after the quit date by specially trained interviewers. The Timeline Follow-Back (TLFB) procedure was used to determine the number of cigarettes smoked each day. This procedure, which utilizes a structured interview and a timeline anchored with notable events relevant to the participant, has been shown to be accurate and reliable for recalling daily cigarette use for the last 30 days (Sobell & Sobell, 1992; Toll, Cooney, McKee, O'Malley, 2005). Relapse was defined as any smoking for seven consecutive days (Hughes, 2003). Days to relapse was defined as the number of days from the quit date to relapse or the end of the observation. Participants who did not relapse while under observation, either because they maintained abstinence to the end of the study period or were lost to follow-up were considered abstinent at least as long as the time they were observed to have been abstinent. This is known as right censoring.

2.5 Data analysis

Descriptive statistics (frequency, mean and standard deviation, median and interquartile ranges [25th and 75th percentile values]) were used to characterize participants. Analysis of variance was used to examine differences between participants who reached the third session of treatment and those who did not for the explanatory measures (nicotine dependence, stress level, trait impulsivity, locus of control, and discounting rate). The explanatory measures were standardized to ensure that they had approximately the same scale by dividing the difference of the sample mean from the original value by the sample standard deviation (i.e., the standardized value of variable X from subject j was $Z_j = [X_j - \text{mean}(X)]/\text{SD}(X)$). Because patterns of missing data differed slightly across measures, the number of observations on which the comparisons were made differed in the analyses.

In order to compare the relative ability of the explanatory measures to predict days to relapse after treatment, we used Cox proportional hazard regressions to model days to relapse using each standardized measure as the sole explanatory variable. Standardized regression coefficients and hazard ratios (HRs) were used to assess the strength and direction of the associations between days to relapse and the explanatory variable. Standardized regression coefficients > 0 and/or a HR > 1 indicate an increased hazard of relapse. Larger magnitude coefficients indicate a stronger association than smaller magnitude coefficients. Of note, standardized regression coefficients may be interpreted as HRs by taking the anti-log of the coefficient (i.e. $\text{HR} = \exp(b)$, where b is the standardized regression coefficient). As such, the HR is the fold-change in the hazard of relapse for a 1 SD increase in the standardized variable. We report both standardized regression coefficients and HRs. We also compare standardized regression coefficients among measures using a bootstrap method to supply 95% confidence intervals for the difference between a pair of regression coefficients. We compared the measure with the strongest association (i.e., the greatest standardized regression coefficients) to all the other measures.

We then examined the extent to which the discounting rates, locus of control, and/or trait impulsivity measures retained associations with days to relapse while accounting for nicotine dependence and stress level. For these analyses, the base model included the FTND, the PSS, and number of missed treatment sessions. Likelihood ratio tests were used to compare the base model to base model plus the additional measures (i.e., delay discounting measures, locus of control, and the BIS scales).

3.0 RESULTS

3.1 Participants

One-hundred and thirty-one ($n=131$) participants were enrolled in this study; $n=100$ participants attended at least one session of treatment; and $n=90$ participants attended at least three sessions of treatment (i.e., made it to the scheduled quit day). Participants who reached the quit day were 47% male, 57% partnered, 77% white, 13% African American, and 10% other (American Indian or Alaskan Native, Pacific Islander, multi-racial). They had a mean age of 47.5 (SD 12.7), a median annual household income of \$30,000 (Interquartile range [IQR] \$16,000 – \$45,000), and a median number of years of education of 13 (IQR 12 – 15).

About two-thirds (63%) were employed full- or part-time, and 37% were unemployed. Participants smoked a mean of 23.6 (SD 11.80) cigarettes per day, had a mean FTND score of 6.04 (SD 1.91) and a mean PSS score of 32.94 (SD 5.95). Participants were highly dependent and reported high levels of stress. Means (SD) for all measures are displayed in Table 1. No significant differences were found between participants who attended at least three treatment sessions and those who did not in level of dependence, stress level, locus of control, delayed discounting rates, and any of the BIS scales.

The hazard of relapse increased for all measures as the level or value of the measure increased, or in the case of the FTND, decreased. See Table 1 and Figure 1. Using the standardized regression coefficients to compare the relative strength of the association with days to relapse, the \$100 discounting rate demonstrated the strongest association with relapse; however, only the FTND was significantly weaker than the \$100 discounting rate. See Figure 2. When the FTND, the PSS, and the number of missed sessions were accounted for in the models, the discounting measures continued to be associated with days to relapse. No other measures evidenced an association with the hazard of relapse when added to the base model.

Because the \$100 delay discounting rate was so strongly associated with days to relapse, we conducted an exploratory analysis to examine whether delay discounting rates changed over the course of the observation period. Although the tendency was for discounting rates to decrease over the 6 months of observation, the decrease was not statistically different from baseline. See Table 3.

4.0 DISCUSSION

We compared the relative strengths of associations among delay discounting rates, dimensions of trait impulsiveness from the BIS-11, locus of control, nicotine dependence, and stress level with days to relapse among smokers after an intensive multicomponent cognitive-behavioral treatment for tobacco dependence. Our findings indicate that delay discounting of \$100 demonstrated the strongest association with relapse and that measures of delay discounting and trait impulsiveness have stronger associations with days to relapse than measures of nicotine dependence and stress level. Moreover, delay discounting rates maintained significant associations with days to relapse when combined with measures of nicotine dependence and stress indicating that delay discounting rates add to what is already accounted for by nicotine dependence and stress and are thus, uniquely associated with relapse. Although dimensions of trait impulsiveness and locus of control did not maintain significant associations with relapse when combined with nicotine dependence and stress, the strength of their associations with relapse were not significantly different from nicotine dependence and stress alone and thus they compare favorably with these well-established indicators of relapse. These findings signify that delay discounting rates and measure of impulsiveness are productive new targets for enhancing treatment for tobacco dependence. Adding interventions designed to decrease delay discounting rates and/or decrease impulsiveness to a comprehensive treatment for tobacco dependence has the potential to incrementally decrease relapse rates.

Smokers with higher discounting rates, higher levels of impulsiveness, and a more externally focused locus of control maintained abstinence for fewer days after receiving a comprehensive treatment for tobacco dependence. These results are similar to previous studies (Sheffer et al., 2012; Stanger, et al., 2012; Yoon et al., 2007; MacKillop & Kahler, 2009). This suggests that routine assessment of these factors, delay discounting rates in particular, are likely to be of clinical value. Clinical interventions to address these factors might also be used to personalize treatment for tobacco dependence, similar to the manner in which treatment is now personalized by nicotine dependence level (i.e., dose of nicotine in nicotine replacement) and stress level (i.e., various stress management interventions). Understanding the relative effects of various levels of these factors as well as the mechanisms by which these factors influence relapse will be key to developing and disseminating clinical assessments and treatment intervention components.

Surprisingly, measures of nicotine dependence and stress demonstrated some of the weakest associations with days to relapse. We speculate that there could be several reasons for these results. The comprehensive treatment provided in this study included interventions for nicotine dependence (i.e., nicotine patches, understanding and managing withdrawal) and stress management (i.e., relaxation training, problem-solving, conflict management) as well as other components (i.e., self-monitoring, enhancing social support, goal setting, etc.), that could have attenuated associations among nicotine dependence and stress and relapse during the 6 months of observation. The participants were also highly dependent, reported high levels of stress, and were primarily of lower socioeconomic status. This range of factors could have affected traditional associations between the FTND, the PSS, and relapse. Additionally, we used a very detailed continuous measure of relapse (i.e., days to relapse) rather than the more commonly utilized dichotomous point-prevalence measure of abstinence for treatment outcomes. This might also have affected the expected associations between the FTND, the PSS, and relapse.

Delay discounting of \$100 was clearly the strongest prognostic indicator in this study. These findings support recent characterizations of delay discounting rates as strong prognostic indicators for treatment outcomes for tobacco dependence as well as cocaine dependence, obesity, and asthma (Bickel, Jarmolowicz, Mueller, Koffarnus, & Gatchalian, 2012; Best et al., 2012; Brandt & Dickinson, 2013). If delay discounting rates are generally predictive of a therapeutic response then they might function as a trans-disease process indicator in a wide variety of medical and behavioral health settings in which a diversity of disorders are encountered.

At present, a variety of techniques have shown some efficacy to decrease delay discounting rates and suggest specific lines of research for developing interventions. Laboratory studies show that simply reframing smaller sooner versus larger later choices into two statements that explicitly state the default outcomes (i.e., something now but nothing later versus nothing now but more later) decreases discounting rates (Radu, Yi, Bickel, Gross, & McClure, 2011). Encouraging episodic future thinking with personalized, structured tasks designed to envision events in the future has also been shown to decrease discounting rates by 16% (Peters & Buchel, 2010). Among stimulant users, discounting rates can be decreased by training working memory, an executive function skill (Bickel, Yi, Landes, Hill, & Baxter,

2011). Additionally, increasing activity in the left dorsolateral prefrontal cortex with one session of high frequency repetitive transcranial magnetic stimulation decreases delay discounting rates among smokers and nonsmokers, albeit temporarily (Sheffer, et al., 2013). Finally, biological mechanisms implicated in the subjective valuing of rewards suggest that certain types of medications might be helpful in decreasing delay discounting rates as well (Hamilton & Potenza, 2012). Assuming that discounting is trans-disease process, the same techniques might be useful for a variety of health behaviors.

The relationship of delay discounting rates to therapeutic outcomes in this and other studies raises questions about the mechanisms by which discounting influences relapse. We speculate that there are several possibilities which might be operating simultaneously. Smokers who discount more steeply might simply be unable to project value into the future. Others have found that a cognitive-behavioral intervention designed to increase the future time perspective was effective in increasing physical activity (Hall & Fong, 2003). Smokers who discount more steeply also might be more insensitive to the cognitive-behavioral components of treatment. Perhaps they are unable to use or apply information and techniques outside of treatment or have more difficulty recalling these techniques when they need them. Smokers who discount more steeply might also be more sensitive to influences in their immediate environment. A limited temporal horizon (Bickel et al., 2011; Radu et al., 2011) might influence the perception of immediate stimuli. For example, internal or external cues for smoking might feel stronger when viewed from a limited temporal horizon. This interpretation suggests that long-term behavioral change requires the lengthening of the temporal window with a particular focus on selecting less impulsive delayed alternatives during decision making. Tracking the development of process skills hypothesized to operate during treatment might help to untangle the relationship between delay discounting rates and relapse. Recent research examining similar self-regulation processes in the treatment of problem gambling suggests that participants follow a process of change whereby psychological improvements are achieved prior to behavioral improvements change (Christensen et al., 2013). However, some participants made significant concurrent behavioral and psychological changes suggesting a degree of individual difference and the need for a range of assessments to capture the variety of mechanisms that appear to drive change (Christensen et al., 2013). Several of the mechanisms mentioned above are related to executive function abilities, which indicates that tracking of executive function skills could help untangle these relationships, similar to the work of Bickel et al. among stimulant users receiving working memory training (Bickel et al., 2011).

This study has a number of strengths and limitations. Among the strengths are that this study provided a comprehensive behavioral treatment with a full range of evidence-based components and nicotine replacement therapy for 8 weeks. Our participants were highly dependent, reported high levels of stress, and were of lower SES, and are thus representative of large proportion of smokers who have more difficulty achieving long-term abstinence. More research is needed to develop and test delay discounting assessment methods and cut-off scores for use by clinicians and to adapt current knowledge of how to decrease delay discounting rates to clinical interventions.

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HIGHLIGHTS

- We compared the ability of selected factors to predict days to smoking relapse
- Delay discounting rates had stronger associations with days to relapse than other factors
- Delay discounting added to variance associated with dependence and stress levels
- Delay discounting rates are uniquely associated with relapse
- Delay discounting is a new target for enhancing treatment for tobacco dependence

Figure 1.

Hazard ratios for a one standard deviation increase for each measure/explanatory variable plotted with 95% confidence intervals (CIs). CIs that do not cover the center line (i.e., hazard ratio = 1) are significant at the .05 level. Discounting = Delay discounting measures; BIS = Barratt Impulsiveness Scale-11; LOC=Rotter's Locus of Control; PSS = Perceived Stress Scale; FTND= Fagerstrom Test for Nicotine Dependence; \$100= delay discounting of \$100; Mean = Mean value of delay discounting of \$100 and \$1000; \$1000 = delay discounting of \$1000; CI = Cognitive Instability; SC= Self-control; MI= Motor Impulsivity; MO = Motor; NI=Non-planning Impulsivity; AI=Attentional Impulsivity.

Figure 2.

Standardized regression coefficient estimates for one standard deviation increase for each measure/explanatory variable plotted with 95% confidence intervals (CIs) for a difference from the coefficient for delay discounting of \$100. CIs not covering the center line are significantly different from the coefficient for delay discounting of \$100 ($p < .05$).

Discounting = Delay discounting measures; BIS = Barratt Impulsiveness Scale-11; LOC=Rotter's Locus of Control; PSS = Perceived Stress Scale; FTND= Fagerstrom Test for Nicotine Dependence; \$100= delay discounting of \$100; Mean = Mean value of delay discounting of \$100 and \$1000; \$1000 = delay discounting of \$1000; CI = Cognitive Instability; SC= Self-control; MI= Motor Impulsivity; MO = Motor; NI=Non-planning Impulsivity; AI=Attentional Impulsivity.

Table 1

Results from univariate Cox proportional hazard regressions modeling days to relapse with each measure. Hazard ratios (HR) – the expected fold-change in the hazard of relapse for a one SD increase in the explanatory variable. Note: Hazard ratio = $\exp(\text{Estimated standardized regression coefficient})$.

Measure (N)	Mean (SD)	Estimated standardized regression coefficient ($\times 10^{-3}$)	Standard error ($\times 10^{-3}$)	Hazard Ratio	Confidence Interval (95%) of hazard ratio (lower, upper)	X ² value (1 df)	P. value
Delay Discounting of \$100 [ln(k)] (85)	-4.93 (2.9)	374	158	1.454	1.066, 1.983	5.58	0.018
Delay Discounting mean ln(k) of \$100 and \$1,000 (81)	-5.38 (2.42)	401	174	1.493	1.062, 2.099	5.32	0.021
Delay Discounting of \$1,000 [ln(k)] (86)	-5.81 (2.52)	265	155	1.303	0.961, 1.766	2.91	0.088
BIS Cognitive Instability (88)	5.88 (1.89)	306	157	1.358	0.998, 1.847	3.79	0.052
BIS Self-Control (88)	11.41 (3.6)	267	153	1.306	0.969, 1.761	3.07	0.080
BIS Motor Impulsiveness (88)	22.57 (4.76)	256	142	1.292	0.977, 1.707	3.23	0.072
BIS Motor (88)	15.34 (3.42)	214	135	1.238	0.95, 1.613	2.5	0.114
BIS Nonplanning Impulsivity (88)	22.97 (4.82)	177	147	1.194	0.896, 1.591	1.46	0.227
BIS Attentional Impulsivity (88)	15.93 (4.3)	149	155	1.16	0.856, 1.574	0.92	0.338
Rotter's Locus of Control (89)	9.04 (3.82)	161	154	1.174	0.868, 1.589	1.08	0.298
Perceived Stress Scale (89)	32.94 (5.95)	32	148	1.033	0.773, 1.38	0.05	0.828
Fagerstrom Test for Nicotine Dependence (84)	6.04 (1.91)	-69	152	0.933	0.693, 1.256	0.21	0.649

BIS=Barratt Impulsiveness Scale –11

Table 2B

Model	N	FTNDEST (SE) by $\times 10^{-3}$	PSS EST (SE) by $\times 10^{-3}$	Missed Tx EST (SE) by $\times 10^{-3}$	Measure (s) added to model	EST (SE) by $\times 10^{-3}$	Likelihood ratio test χ^2	p-value
Base model	75	-60 (171)	-90 (173)	326 (198)				
Base + Delay Discounting mean of \$100 and \$1,000	75	-176 (173)	-205 (175)	368 (204)	ln(k) Mean	539 (202)	8.20 (df=1)	.004
Base model	75	-60 (171)	-90 (173)	326 (198)				
Base model + Delay Discounting for \$100 and \$1000	75	-210 (177)	-205 (177)	380 (204)	ln(k) \$100 ln(k) \$1000	181 (221) 456 (255)	8.78 (df=2)	.003
Base model	82	-68 (156)	-46 (160)	379 (193)				
Base + Rotter's Locus of Control	82	-82 (153)	-28 (160)	390 (196)	Rotter's Locus of control	233 (161)	2.09 (df=1)	.148
Base model	81	-89 (159)	-18 (159)	366 (196)				
Base + BIS Total	81	-126 (161)	-45 (157)	361 (201)	BIS-Total	304 (159)	3.57 (df=1)	.059
Base model	81	-89 (159)	-18 (159)	366 (196)				
Base + BIS scales	81	-233 (176)	-207 (182)	472 (211)	BIS-Self- Control BIS-Cognitive Instability BIS- Attentional Impulsivity BIS-Motor Impulsivity BIS- Nonplanning Impulsivity BIS-Motor	525 (383) 668 (366) -638 (376) 922 (500) -395 (420) -639 (438)	11.83 (df=6)	.066

Base model = Cox proportional hazard model of days to relapse with the Fagerstrom Test for Nicotine Dependence (FTND), the Perceived Stress Scale (PSS), and number of missed treatments (Tx) only;
EST=Standardized regression coefficients ($\times 10^{-3}$); SE = Standard error ($\times 10^{-3}$); BIS = Barratt Impulsiveness Scale;

Table 3

Change from baseline discounting for those completing 7 and 27 weeks of treatment

Delay discounting measure	Weeks after quit date	N	Mean Difference from baseline delay discounting rate (95% Confidence Interval)
\$100	7	42	−0.127 (−1.112, 0.859)
	27	31	−0.188 (−1.218, 0.843)
\$1000	7	52	−0.369 (−1.181, 0.442)
	27	38	−0.347 (−1.373, 0.679)