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Delay Discounting, Locus of Control, and Cognitive Impulsiveness Independently Predict Tobacco Dependence Treatment Outcomes in a Highly Dependent, Lower Socioeconomic Group of Smokers

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Abstract

Tobacco use disproportionately affects lower socioeconomic status (SES) groups. Current explanations as to why lower SES groups respond less robustly to tobacco control efforts and tobacco dependence treatment do not fully account for this disparity. The identification of factors that predict relapse in this population might help to clarify these differences. Good candidates for novel prognostic factors include the constellation of behaviors associated with executive function including self-control/impulsiveness, the propensity to delay reward, and consideration and planning of future events. This study examined the ability of several measures of executive function and other key clinical, psychological, and cognitive factors to predict abstinence for highly dependent lower SES participants enrolled in intensive cognitive-behavioral treatment for tobacco dependence. Consistent with predictions, increased discounting and impulsiveness, an external locus of control as well as greater levels of nicotine dependence, stress, and smoking for negative affect reduction predicted relapse. These findings suggest that these novel factors are

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Declaration of Interest

Dr. Bickel is a principal in HealthSI LLC. Drs. MacKillop, McGeary, Landes, Stitzer, Yi, Jones, and Christensen and Ms. Jackson report no competing financial interests or potential conflicts of interest. Dr. Carter is now employed by Jazz Pharmaceuticals, PLC and has a financial interest in Jazz Pharmaceuticals, PLC. The authors alone are responsible for the content and writing of this paper.

clinically relevant in predicting treatment outcomes and suggest new targets for therapeutic assessment and treatment approaches.

INTRODUCTION

Tobacco use continues to be the greatest cause of preventable death and disease in the United States causing 438,000 deaths and incurring \$157 billion in costs annually.^{1, 2} Although smoking prevalence has declined, approximately one in five Americans (19.8%) still smoke daily.³ The smoking prevalence in the United States for those with 221 incomes <\$35K (~30%) is twice that of those with incomes >\$50K (~15%).³ Smokers of lower socioeconomic status (SES) are of considerable public health interest because they respond less robustly to tobacco control efforts, are often difficult to access, use evidence-based tobacco dependence treatments less frequently, and are generally less successful in achieving long-term abstinence.⁴⁻⁹ Current explanations of this disparity do not account for a large proportion of these differences.^{6, 10, 11}

Greater than 60% of smokers make at least one attempt to quit each year, but just 5% of those who attempt remain abstinent 12 months later.^{5, 12} Even with intensive combination treatment, at least 70% of smokers relapse within 12 months.⁵ Smokers, along with treatment providers, close friends, and family members are often mystified as smokers relinquish the prospects of long-term health, relationships, and monetary savings in favor of smoking. Although substantial progress has been made in understanding how several key factors such as dependence, stress, and negative affect levels contribute to relapse, the identification of additional factors that influence relapse might elucidate this conundrum and explain why lower SES smokers have more difficulty quitting as well as suggest new targets for molecular study and lead to new or enhanced therapeutic approaches for addiction.

Good candidates for novel prognostic factors might be found in the constellation of behaviors associated with executive function including self-control/impulsiveness, the propensity to delay reward, and behaviors associated with consideration and planning of future events.¹³⁻¹⁹ In other addictive disorders, relapse is correlated with compromised executive function.²⁰⁻²³ Impulsiveness also interacts with stress level to produce higher vulnerabilities for alcoholic drinking.²⁴ Although findings are dependent upon the particular measures used, nicotine dependence levels are generally positively correlated with impulsiveness and contribute independently to cessation outcomes.²⁵⁻²⁷ Smoking is associated with more impulsive decision making and a greater tendency to prefer smaller immediate rewards over larger later rewards as measured by delayed discounting tasks.^{13, 28-31} Although there is considerable variation, most individuals prefer an immediate reward, but will wait a period of time for a larger later reward.³²⁻³⁴ However, smokers discount the value of rewards received later more steeply, demonstrating an increased preference for smaller immediate rewards.^{13, 28-31} The degree to which rewards are discounted by smokers is positively related to number of cigarettes smoked, nicotine levels, and their experience of withdrawal.^{31, 35, 36} Decreased discounting is also associated with an increased likelihood of long-term abstinence from smoking in pregnant women, adolescents, and heavy drinkers.³⁷⁻³⁹

Locus of control is the degree to which one believes that reinforcement or rewards are contingent upon internal or external factors.⁴⁰ Individuals with an internally focused locus of control believe that reinforcement is contingent upon their own efforts. Individuals with an externally focused locus of control believe that reinforcement is contingent upon luck, fate, or powerful others.⁴⁰ Those with an internally focused locus of control tend to be less impulsive, more future-oriented, and discount less steeply than externally focused

individuals.^{41–44} An internally focused locus of control is also linked to better performance on measures of executive function and greater sensitivity to opioid receptor blockade-induced reductions in delay discounting while an externally focused locus of control is linked to the likelihood of smoking in lower SES groups.^{11, 45} An internally focused locus of control and perceived control over external events are also positive prognostic factors for abstinence from smoking.^{46–48}

The constellation of behaviors associated with executive function are quite plausibly related to cognitive-behavioral treatment (CBT) outcomes for tobacco dependence, the type of counseling that has been shown to be efficacious for the treatment of tobacco dependence.⁵ CBT response clearly involves: (1) believing that rewards or outcomes are contingent on factors that can be managed by self, (2) understanding the role of self and the environment in managing self-control, (3) understanding the relationship between cues, urges, and individual responses, and (4) planning into the future to manage high-risk situations, among other factors. All of the above factors engage executive functions such as the ability to manage your environment, self-regulation, and ability to choose a larger delayed reward (reflecting internal locus of control, self/control impulsiveness, and delay discounting).

Given these putative relationships, this study examined the ability of several key clinical, psychological, and cognitive factors to predict smoking abstinence for highly dependent lower SES participants enrolled in intensive CBT for tobacco dependence. We hypothesized that well-studied factors such as dependence, stress, and negative affect levels would predict abstinence in the expected directions for this group. We also hypothesized that poorer performance on measures of executive function, greater impulsiveness, greater discounting of future rewards, a more externally focused locus of control, and a more present-focused orientation would predict a lesser likelihood of abstinence after treatment.

METHOD

Participants

Participants were recruited by referral from medical center campus tobacco treatment services, word-of-mouth, and print and radio advertisements. Criteria for participation included: age 18 years or older; smoking 16 or more cigarettes per day; meeting criteria for nicotine dependence from the Diagnostic and Statistical Manual for Mental Disorders-IV⁴⁹; not pregnant or lactating; not currently using medications for smoking cessation (i.e., bupropion, varenicline, nicotine replacement, etc.); no current psychiatric diagnosis that would interfere with participation in assessments or treatment (ie, schizophrenia spectrum disorders, social phobia, etc.); drinking 19 or less alcoholic drinks per week; and no plans to move out of the area. Participants also provided a carbon monoxide (CO) breath sample of 15 parts per million (ppm) or more and passed a urine drug screen for drugs of abuse (amphetamine, benzodiazepines, cannabis, cocaine, opioids, methadone). This study was approved by the Institutional Review Board of the University of Arkansas for Medical Sciences.

Procedure

Pretreatment Procedures—Participants took part in two 3-hour sessions of data collection prior to receiving treatment. Prior to each session, participants were required to smoke one cigarette to standardize time from the last cigarette across participants.

The Tobacco Dependence Treatment—Participants were treated with multicomponent cognitive-behavioral therapy with relapse prevention, a well-established, validated 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.^{5, 50} The treatment was manual-driven and delivered by certified Tobacco Treatment Specialists (TTSs). The TTSs had either a masters degree in psychology or a bachelors degree in social work with 1–2 years experience delivering the same treatment in-person and over the telephone in the medical center tobacco dependence treatment programs. They were trained by the first author in an intensive 5-day tobacco dependence treatment training and certified by the ACT Center at the University of Mississippi Medical Center. The approach consisted of six structured, 60-minute, closed-group sessions of content utilized elsewhere.^{51–54} An overview of the biopsychosocial underpinnings of tobacco dependence was presented in lay language addressing the physiological components (eg, tolerance and withdrawal symptoms) and learning components (eg, triggers or cues and tobacco use as reinforcement) of tobacco addiction, as well as the use of tobacco to cope with nicotine-related (eg, lowered plasma nicotine levels) and nicotine-unrelated (eg, managing stress) events. 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. Participants attended sessions once per week for 6 weeks. The quit date was the day of the third treatment session. No medications were provided to participants and participants agreed not to use any medications for cessation for the duration of the study. All treatment sessions were reviewed in weekly supervision meetings with a psychologist (CES) as well as randomly recorded and reviewed for consistency.

Measures

Demographic Measures—Standard demographic measures were collected including sex, race, age, years of education, marital status, income, and employment status.

Tobacco-Related Clinical Factors—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 to 10 with greater values indicating greater dependence levels and a lesser likelihood of abstinence.^{55, 56}

Motivation and Self-efficacy. Motivation and self-efficacy were measured on a scale of 0–10 with 0 = “not at all” and 10 = “the most ever” using the questions: “How much do you want to quit smoking?” and “How confident are you that you can quit using tobacco and stay quit for good?” These items are utilized elsewhere to briefly assess motivation and self-efficacy with greater values predicting a greater likelihood of abstinence.^{51, 57, 58}

Perceived Stress Scale (PSS). The PSS is a widely used 14-item questionnaire assessing stress level. Scores range from 0 to 56 with greater values indicating greater stress levels and predicting a lesser likelihood of abstinence.^{59–62}

Smoking Consequences Questionnaire-Adult (SCQ-A). The SCQ-A is a 55-item questionnaire assessing the expected utility of smoking in adults. Because negative affect is associated with relapse, only the nine items from the Negative Affect Reduction subscale were included.^{61, 63, 64} Scores on this subscale range from 9 to 81 with greater scores indicating a greater expectation that smoking will result in the reduction of negative affect. This scale is highly internally consistent with greater values predicting a lesser likelihood of abstinence.^{64, 65}

Beck Depression Inventory-II (BDI-II). The BDI-II is a well-established 21-item questionnaire assessing depressive symptomatology.⁶⁶ Scores range from 0 to 63 with

greater values indicating greater dysphoric/depressed mood. Increased dysphoric/depressed mood is associated with a lesser likelihood of abstinence.⁶¹

Positive and Negative Affect Scales (PANAS). The PANAS is a 20-item questionnaire with two 10-item subscales: Positive Affect and Negative Affect. Scores on each subscale range from 10 to 50 with greater values indicating greater affect intensity. The two subscales are internally consistent, largely uncorrelated, and stable over a period of 8 weeks.⁶⁷ Greater values on the Negative Affect subscale are associated with a lesser likelihood of abstinence.⁶¹

Executive Function and Impulsiveness—Balloon Analogue Risk Task (BART). The BART is a computerized task assessing risky decision making that is highly correlated with measures of impulsiveness and lowered behavioral constraint.⁶⁸ Smokers tend to make more risky decisions using the BART than nonsmokers.⁶⁸ For each trial, an uninflated balloon appears on the monitor along with a pump. The participant can choose to inflate the balloon one unit and earn 5 cents. The money is collected in a temporary reserve shown on the screen. The participant can also choose to end the trial (and the ability to earn money) by emptying the sum in the temporary reserve to a permanent bank where it cannot be lost. If the participant chooses to continue inflating the balloon (and adding money to the temporary reserve) and the balloon bursts, the participant will lose the amount in the temporary reserve. There is an increasing probability that the balloon will burst with each successive set of pumps (1/128 for 30 trials, 1/32 for 30 trials, and 1/8 for 30 trials [90 trials total]). Each probability is associated with a different color balloon. The first 30 trials present the balloons in a random mix of colors. The next 60 trials present the balloons in 20 trial blocks of the same color (counterbalanced between subjects). Results include three measures: the total amount of money collected in the permanent bank, the total number of pumps on which the balloon did not burst, and the mean number of pumps per trial.

Barratt Impulsiveness Scale 11 (BIS). The BIS is a 30-item questionnaire that yields a total score and three subscale scores for motor impulsiveness (acting without thinking), cognitive impulsiveness (making quick cognitive decisions), and nonplanning impulsiveness (lack of concern about the future). Only the subscale scores were included. The measure is internally consistent and stable over a period of 8 weeks.^{69, 70} Greater values indicate more impulsiveness.

Delay Discounting Tasks. The delay discounting tasks assess the degree to which rewards are modulated by the delay to their receipt.³⁴ Participants completed three tasks: \$100 reward in real money, \$100 reward in hypothetical money, and \$1,000 reward in hypothetical money. For each task, 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 choice adjusts the immediate choice according to whether the participant chose the immediate or delayed reward. The final value at the end of the series is the indifference point for that delay. An indifference point is the value of the immediate reward, expressed as a proportion, subjectively deemed equivalent to the larger, delayed reward. The results for each task are expressed as the natural logarithm of k in Mazur's hyperbolic discounting model, with k increasing as the magnitude of the participant's preference for the smaller, sooner reward increases.⁷¹ The results from each task were also averaged to produce a mean delay discounting score. Data from existing published studies indicate values for log k range from about -12.00 to 4.00.

Eysenck Impulsiveness Scale (EIS). The EIS is a 54-item questionnaire with three subscales: Impulsiveness, Venture-someness (ie, sensation-seeking), and Empathy. Because the Empathy subscale is unrelated to risky decision making, it was not administered. The subscales are internally consistent with good test-retest reliability and convergence with other measures of impulsiveness.⁷²⁻⁷⁴

Frontal Systems Behavior Scale (FrSBe). The FrSBe, a 46-item behavior rating scale intended to measure behavior associated with damage to the frontal systems of the brain, is regarded as one of the most valid and reliable measures of the behavioral aspects of frontal lobe dysfunction.⁷⁵ The FrSBe measures behavior at the present time and before the illness or injury. Only the present time scales were applicable and included in this analysis. The present time scales yield a total score and scores for the subscales of Apathy, Disinhibition, and Executive Dysfunction. Individual subscale scores are highly internally consistent and stable over 12 weeks.⁷⁶⁻⁷⁸ Values are scaled according to reference group norms and reported in *T*-scores. Scores above 65 are considered clinically significant.

Go/No-Go Task. The Go/No-Go Task assesses inhibitory control by developing a prepotent response to one stimulus while requiring inhibition of a different, but similar stimulus. Go/No-Go Tasks have identified inhibitory control impairments in individuals with addictive disorders.⁷⁹⁻⁸¹ The participant learns by trial and error to respond to four of eight two-number pairs (positive stimuli) and not respond to four other two-number pairs (negative stimuli).⁸² When participants correctly respond to a positive stimuli, they gain a point; when they correctly respond to a negative stimuli, no point is lost; when they incorrectly respond to any stimuli, a point is lost. Participants begin the task with 32 points and can earn up to a total of 64 points. Results are presented as a Plus score that reflects the speed with which one correctly responds to positive stimuli, a Minus score that reflects the speed with which one incorrectly responds to negative stimuli, and a total score that reflects the total number of points earned.

Microcog™: Assessment of Cognitive Functioning. This computer-administrated battery of subtests generates composite scores across five cognitive domains: Attention/ Mental Control, Memory, Reasoning/Calculation, Spatial Processing, and Reaction Time. Two factors assessing Information Processing Speed and Accuracy are derived from the speed and accuracy scores of the subtests. The two information processing calculations are used to produce a General Cognitive Functioning score, in which speed and accuracy are given equal weight, and a General Cognitive Proficiency score, in which accuracy is given more weight than speed. Values are scaled according to reference group norms provided by the manufacturer with a mean of 100, a standard deviation of 15.⁸³

Rotter's Locus of Control Scale (LOCS). The LOCS is a 29-item questionnaire assessing the extent to which individuals believe they can control events.⁴⁰ Scores range from 0 to 29 with greater values indicating a more externally focused locus of control.⁴⁰ An internally focused locus of control is associated with less impulsiveness, more future-orientation, the propensity to choose larger later rewards, and better performance on measures of executive function.^{44, 45}

Stanford Time Perception Inventory (STPI). The STPI is a 38-item measure used to assess four aspects of time perspective: Present-Hedonistic, Present-Fatalistic, Future-Oriented, and Past-Oriented.⁸⁴ Greater values indicate greater endorsement of the particular time perspective. The STPI is correlated with insensitivity to future consequences in heroin addicts and pathological gambling symptoms in gamblers.^{85, 86}

Tuckman Procrastination Scale (TPS). The TPS is a 16-item measure used to assess the tendency to procrastinate.⁸⁷ Scores range from 16 to 64 with greater values indicating a greater propensity to procrastinate.

Outcome Assessment—Point prevalence abstinence was confirmed by exhaled CO level of 8 ppm or less using a Bedfont Smokelyzer (Bedfont Scientific Ltd, Kent, England).⁸⁸ Outcomes assessments were conducted 1, 2, 3, 4, 8, 12, and 28 weeks after the quit day.

Data Analysis

Descriptive statistics (means, standard deviations, medians, interquartile ranges, IQRs [25th and 75th percentile values], frequencies) were used to characterize participants. Demographic differences between those who attended treatment and those who did not attend treatment were assessed with tests of significance (*t*-tests, Mann-Whitney tests, and chi-square tests where appropriate).

Similar to other addiction treatment studies, this study incorporated repeated measurements over time, which are often correlated, as well as included a number of missing data points. Generalized estimating equations (GEE) is a set of methods used for modeling correlated or repeated measures in longitudinal designs, but is not often utilized by addiction researchers.⁸⁹ The oft-used classic proportional methods of analysis such as logistic regression can only incorporate one outcome data point in a model, do not recognize correlations among measures and can thus produce incorrect standard errors resulting in invalid hypothesis tests and confidence intervals, and are naïve to the effects of time. Classic proportional methods also handle missing outcome data by requiring penalized imputation (ie, imputing missing outcome data as smoking) or elimination of missing cases. GEE can be thought of as an extension of logistic regression that incorporates all outcome data points in one model, corrects standard errors of estimates by using a working correlational structure based on observed data, and incorporates the effects of time. GEE also handles missing outcome data without penalized imputation or case elimination. Missing data are simply entered as missing. Nonetheless, at least one outcome data point was required for a participant to be included in the models and if a participant was missing a particular baseline predictor, his or her data could not be included in the model for that predictor. See Lee et al. and Hall et al. for more information.^{90,91} Each baseline predictor was individually entered alongside time in a GEE model that included all outcome assessment data points. On the basis of previous studies examining the candidate variables we made a priori hypotheses about the direction of each measure's predictive utility and used one-tailed, $p < .05$ -level tests of significance in our GEE models.^{20–23, 37–39, 46–48, 56, 61, 79, 92–96} The GLIMMIX procedure in SAS[®] Version 9.2 was used in fitting the GEE models.

RESULTS

Participants

The participants ($n = 97$) were 41% male and primarily lower SES with a mean age of 48.16 (SD = 11.62). The median household income was \$17,750 (IQR: \$10,000–\$25,500) and the mean number of years of education was 13.14 (SD 1.84). Sixty-one percent self-identified as white and 51% were unemployed. These participants include a number of notable characteristics: They were highly dependent with a mean FTND score of 6.43 (SD 1.75). They were highly motivated to quit smoking with a median response of 10 on a scale of 0–10 (IQR: 8–10), but reported clinically significant levels of apathy on the FrSBe and relatively high levels of Negative Affect on the PANAS—Negative Affect scale, nearly double those found in other samples.⁶⁷ See Table 1 for details.

Eighty (82%) of the 97 participants attended treatment. Among these, treatment adherence was high with the median number of treatment sessions attended being five out of a possible six sessions (IQR: 3.5–6). Those participants who attended treatment were slightly older (49.2 vs. 43.1 years; $t[95] = 1.97, p = .052$) and had more education (13.3 vs. 12.4 years; $t[95] = 1.91, p = .059$) than those who enrolled but did not attend treatment. Thirty-seven percent of the total number of participants ($n = 97$) attended the 6-month follow-up assessment. Using penalized imputation (ie, imputing missing data as smoking), the abstinence rate for participants who attended treatment ($n = 80$) was 27% on the quit date, 10% four weeks after the quit date, and 7% six months after the quit date. The GEE model estimated abstinence rates were 39.5% on the quit day, 35.1% four weeks after the quit, and 12% six months after the quit day.

Predictors of Treatment Response

In all instances, the effect of time was significant so it will not be reported separately. Measures that significantly predicted abstinence are listed in Table 2. As hypothesized, greater dependence (FTND) and stress (PSS) levels, and greater levels of smoking in response to negative affect (SCQ-Negative affect reduction scale) predicted a lesser likelihood of abstinence. Three different measures of delay discounting predicted abstinence with those participants who discounted more steeply being less likely to be abstinent, as expected. An externally focused locus of control also predicted a lesser likelihood of abstinence. Finally, greater impulsiveness as measured by the BIS 11 cognitive impulsiveness scale predicted a lesser likelihood of abstinence. Note: Table 2 reflects odds ratios an increase of one standard deviation for each measure. For example, every increase in one standard deviation (1.75 points) on the FTND decreased the odds of abstinence by about 40%.

Interrelationships among Predictors

Because the measures were modeled individually, questions arose as to the relatedness of the measures found to be significant. An exploratory correlational analysis was then conducted. The three delay discounting measures were highly and positively correlated. Stress level was positively correlated with impulsiveness, a more externally focused locus of control, and smoking to reduce negative affect, but negatively correlated to generalized negative affect (PANAS—Negative Affect scale). Impulsiveness was positively correlated with a more externally focused locus of control as well as smoking in response to Negative Affect, but negatively correlated with the PANAS—Negative Affect scale. See Table 3 for details.

DISCUSSION

These findings confirm the predictive role of several key clinical, psychological, and cognitive factors in predicting abstinence for this highly dependent lower SES group of smokers. These findings suggest that this population typically manages high levels of negative affect, dependence, and stress levels, and in this context smoking to reduce negative affect plays a significant role in reducing the likelihood of achieving abstinence. This is one of the few studies that demonstrate that an externally focused locus of control, steeper delay discounting, and greater cognitive impulsiveness independently predict a lesser likelihood of abstinence and suggests that these factors play a role in treatment outcomes for lower SES groups.

Externally focused individuals appear to experience greater levels of stress and be less likely to achieve abstinence from smoking after treatment. The positive correlation between locus of control and stress level is unsurprising given that the two constructs are both conceptually

related to perceived control over external events. Greater stress levels are consistently associated with less perceived control over external events.⁹⁷ Several items on the PSS assess perceived control (ie, “How often have you felt that you were unable to control the important things in your life?”).⁵⁹ Because externally focused individuals attribute reinforcement as under the control of external agents such as luck, chance, fate, or powerful others, they are likely to perceive less control over important events in their lives and thus are likely to experience greater levels of stress. Although not a finding here, greater stress levels have also been associated with higher dependence levels and poorer treatment outcomes.^{60, 98} These findings suggest that research is needed to further explicate the relationship between locus of control, stress, and perceived control in order to better understand how to assess and address these important prognostic factors. This might be particularly important for lower SES smokers as they appear to endorse a more externally focused locus of control which may be reinforced by cultural factors.¹¹ Additionally, the cognitive and physiological ramifications of externally focused individuals chronically experiencing higher levels of stress have the potential to be far-reaching.

Baseline measures of delay discounting were strong predictors of abstinence after treatment. Three different delay discounting measures predicted abstinence including hypothetical discounting of \$100 and \$1,000 and the mean discounting rate of hypothetical \$100, \$1,000, and real \$100. (Note: The estimated effect of the discounting rate for real \$100 [OR = .887, one-sided $p = .069$] was nearly as strong as the others, but the reduced variance of real \$100 discounting rates [relative to the other three] might have prevented it from reaching statistical significance.) These results support previous findings that indicate increased discounting predicts less success with abstinence.^{37–39} Given the strong relationships between delay discounting and smoking status in cross-sectional studies, previous studies have questioned whether individuals who, at baseline, discount more steeply (ie, have strong preferences for smaller, sooner rewards) are simply less successful at achieving abstinence, or if individuals who stop using tobacco begin to discount less steeply over time. If the latter statement were true, we would be unable to predict abstinence from the natural variability in discounting rates. Lower discounting rates would emerge from the process of quitting or being quit. If the former statement were true, the natural variability in baseline discounting rates would predict treatment success, as was the case in this study.

These findings suggest that delay discounting is clinically relevant in predicting abstinence outcomes from CBT for tobacco dependence. Delay discounting might be a productive target for new assessment and therapeutic approaches. These findings also suggest that various measures of discounting future rewards (ie, hypothetical and real, \$100 and \$1,000) are similarly effective in predicting treatment outcomes. The propensity to more steeply discount delayed rewards has the potential to become a behavioral marker alerting clinicians that these individuals might have more difficulty quitting and might need additional or special attention. Discounting rates could be evaluated during the initial assessment along with dependence and stress levels as well as the propensity to smoke in response to negative affect, and other factors. More research is needed to determine the levels of discounting that place a smoker at higher risk for relapse after treatment. More research is also needed to discover the manner in which clinicians might tailor treatment for individuals who discount more steeply. Similar to highly dependent smokers, more intensive and longer treatment, including combination drug therapies might be required for better treatment outcomes for steep discounters. Alternatively, similar to highly stressed smokers or smokers prone to dysphoria, CBT that targets cognitive errors and/or reframes cognitions involved with impulsiveness, the propensity to prefer smaller, sooner rewards, and the perceived contingencies on rewards might be required for better treatment outcomes for steep discounters.

Increased impulsiveness and the propensity to delay reward are also associated with decreased activity in the prefrontal cortex relative to the limbic areas, while the preference for immediate rewards is associated with a relative increase in activity in the limbic system relative to the prefrontal cortex.¹³ Because there are distinct areas of brain activity associated with immediate versus delayed rewards in delay discounting tasks and delay discounting tasks predict abstinence, these findings also suggest that abstinence might be associated with these distinct areas of brain activity as well. If so, then increasing or decreasing activity in these specific areas might also lead to new assessment and therapeutic approaches. Increasing activity in the prefrontal cortex using direct stimulation or repetitive transcranial magnetic stimulation has been shown to temporarily reduce risky or impulsive decision making,⁹⁹ and in one case actually reduced the number of cigarettes smoked in the hours after stimulation.¹⁰⁰ Similar to the way that our current repertoire of approved cessation medications assist with cessation by attenuating the experience of withdrawal, these findings suggest that we might be able to augment treatment by attenuating the propensity to make impulsive decisions by increasing activity in the prefrontal cortex.

This study has a number of strengths and limitations. Among the strengths are that this study provided a highly intensive behavioral treatment with a full range of evidence based components in the absence of medications. This allowed us to evaluate the response to cognitive-behavioral therapy alone without confounding the treatment response with response to medications, which might have produced physiological changes that would have confounded our results. Furthermore, if steep discounters are similar to highly dependent individuals and simply respond better to more intensive, combination treatment, the addition of medications might have attenuated the predictive utility of the delay discounting tasks. A further strength of this approach was its ecological validity, as minority and lower income smokers are less likely to use nicotine replacement or other medications for cessation.^{5, 101-106} Moreover, a minority of publicly funded treatment programs consistently offer free or low-cost medications; only about one-third of quitlines in the United States provide free or low-cost medications and then only to eligible participants.¹⁰⁷ Nonetheless, our participants were characterized by factors strongly associated with more difficulty achieving abstinence (ie, high dependence, negative affect, and stress levels and lower SES) and only a small number of participants achieved long-term abstinence. In all likelihood, more participants would have achieved abstinence if medications were included. A larger group of abstinent smokers would have increased our ability to detect statistical significance and would have allowed us more freedom in our choice of analyses.

Additional limitations include the number of participants who were lost to follow-up. While very little dropout occurred during treatment, a smaller group of participants regularly attended the follow-up assessments. As with many lower SES groups, many participants were highly mobile, depended upon pay-as-you-go forms of communication, and were difficult to track. Given that 51% of participants were unemployed at baseline, it is unsurprising that some participants needed to relocate over the course of 6 months to find work. Use of GEE analyses helped to minimize the effects of missing data on the outcomes, but missing outcome data were nonetheless a limitation that must be considered when interpreting these findings. Another consideration is that the study did not examine the effects of SES level. As such, the findings cannot directly address the extent to which these factors are associated with treatment outcomes for individuals of middle and higher SES; however, given the importance of identifying successful treatment strategies for lower SES individuals, this study verifies that these prognostic factors, both established and novel, are indeed important determinants and are potentially important targets for tailoring interventions for this population. Finally, although the use of CBT without pharmacotherapy in this study is a strength in some ways, it is also a limitation. The current findings may

generalize to lower SES smokers receiving pharmacotherapy, but the current data cannot speak to that question directly.

Future research needs to elucidate relationships among delay discounting, impulsiveness, stress, locus of control, SES, and ability to achieve abstinence for tobacco users with and without the use of CBT and medications. These interrelationships are particularly important in light of the fact that some of the largest effect sizes observed in this study were for stress and discounting, and there is evidence that powerful state factors make discounting more impulsive. This may also be the case for stress. Certainly this sample was characterized by high levels of dependence and were thus subject to high levels of nicotine withdrawal which have both been shown to make discounting more impulsive.^{31, 36, 108} Moreover, these smokers also reported high levels of stress which has been shown to interact with impulsiveness to increase the vulnerability for another addictive substance, alcohol.²⁴ Thus, for lower SES smokers, high levels of nicotine dependence, impulsiveness, and the financial stresses of daily life may interact to create adverse intrapersonal conditions for smoking cessation. Paradoxically, there are clear environmental factors to motivate lower SES smokers to quit, such as the associated cost, that need to also be taken into account. Finally, future studies will be necessary to develop and test assessment methods and cut-off scores for use by clinicians for delay discounting, locus of control, and impulsiveness measures as well as the efficacy of new strategies to address impulsiveness, the propensity to discount the value of future rewards, and to reframe an externally focused locus of control. These strategies include both cognitive-behavioral strategies and novel approaches such as neurocognitive training.¹⁰⁹ Progress in these domains will be essential for clarifying the present findings and translating them into improved smoking cessation outcomes for lower SES smokers.

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REFERENCES

1. Mokdad AH, Marks JS, Stroup DF, et al. Actual causes of death in the United States 2000. *JAMA*. 2004; 291:1238–1245. [PubMed: 15010446]
2. CDC. Annual smoking-attributable mortality, years of potential life lost, and economic costs: United States, 1997–2001. *Morb Mortal Wkly Rep*. 2005; 54:625–628.
3. Centers for Disease Control and Prevention. Behavioral risk factor surveillance system survey data. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention; 2010. Retrieved from <http://apps.nccd.cdc.gov/brfss/CDC>
4. Fiore MC, Novotny TE, Pierce JP, et al. Trends in cigarette smoking in the United States. The changing influence of gender and race. *JAMA*. 1989; 261:49–55. [PubMed: 2908994]
5. Treating tobacco use and dependence: 2008 update. Rockville, MD: U.S. Department of Health and Human Services Public Health Service; 2008.
6. Wetter DW, Cofta-Gunn L, Fouladi RT, et al. Understanding the associations among education, employment characteristics, and smoking. *Addict Behav*. 2005; 30:905–914. [PubMed: 15893088]

7. Barbeau EM, Krieger N, Soobader MJ. Working class matters: Socioeconomic disadvantage, race/ethnicity, gender, and smoking in NHIS 2000. *Am J Public Health*. 2004; 94:269–278. [PubMed: 14759942]
8. Barbeau EM, Leavy-Sperounis A, Balbach ED. Smoking, social class, and gender: What can public health learn from the tobacco industry about disparities in smoking? *Tob Control*. 2004; 13:115–120. [PubMed: 15175523]
9. Barbeau EM, McLellan D, Levenstein C, et al. Reducing occupation-based disparities related to tobacco: Roles for occupational health and organized labor. *Am J Ind Med*. 2004; 46:170–179. [PubMed: 15273970]
10. Wetter DW, Cofta-Gunn L, Irvin JE, et al. What accounts for the association of education and smoking cessation? *Prev Med*. 2005; 40:452–460. [PubMed: 15530598]
11. Stronks K, van de Mheen HD, Looman CW, et al. Cultural, material, and psychosocial correlates of the socioeconomic gradient in smoking behavior among adults. *Prev Med*. 1997; 26:754–766. [PubMed: 9327486]
12. Hughes JR. Effects of abstinence from tobacco: Valid symptoms and time course. *Nicotine Tob Res*. 2007; 9:315–327. [PubMed: 17365764]
13. Bickel WK, Yi R. Temporal discounting as a measure of executive function: Insights from the competing neuro-behavioral decision system hypothesis of addiction. *Adv Health Econ Health Serv Res*. 2008; 20:289–309. [PubMed: 19552313]
14. Barkley RA. Adolescents with attention-deficit/hyperactivity disorder: An overview of empirically based treatments. *J Psychiatr Pract*. 2004; 10:39–56. [PubMed: 15334986]
15. Dennis M. Frontal lobe function in childhood and adolescence: A heuristic for assessing attention regulation, executive control, and the intentional states important for social discourse. *Developmental Neuropsychology*. 1991; 7:327–358.
16. Denckla, MB., editor. *Measurement of Executive Function*. Baltimore: Paul H. Brookes Publishing Co.; 1994.
17. Volkow ND, Fowler JS, Wang GJ, et al. Role of dopamine, the frontal cortex and memory circuits in drug addiction: Insight from imaging studies. *Neurobiol Learn Mem*. 2002; 78:610–624. [PubMed: 12559839]
18. Verdejo-Garcia A, Lopez-Torrecillas F, Gimenez CO, et al. Clinical implications and methodological challenges in the study of the neuropsychological correlates of cannabis, stimulant, and opioid abuse. *Neuropsychol Rev*. 2004; 14:1–41. [PubMed: 15260137]
19. Hester R, Garavan H. Executive dysfunction in cocaine addiction: Evidence for discordant frontal, cingulate, and cerebellar activity. *J Neurosci*. 2004; 24:11017–11022. [PubMed: 15590917]
20. Bauer L. Antisocial personality disorder and cocaine dependence: Their effects in behavioural and electroencephalographic measures of time estimation. *Drug Alcohol Depend*. 2001; 63:87–95. [PubMed: 11297834]
21. Pezawas LM, Fischer G, Diamant K, et al. Cerebral CT findings in male opioid-dependent patients: Stereological, planimetric and linear measurements. *Psychiatr Res*. 1998; 83:139–147.
22. Fals-Stewart W, Schafer J. The relationship between length of stay in drug-free therapeutic communities and neurocognitive functioning. *J Clin Psychol*. 1992; 48:539–543. [PubMed: 1325478]
23. Paulus MP, Tapert SF, Schuckit MA. Neural activation patterns of methamphetamine-dependent subjects during decision making predict relapse. *Arch Gen Psychiatry*. 2005; 62:761–768. [PubMed: 15997017]
24. Fox HC, Bergquist KL, Peihua G, et al. Interactive effects of cumulative stress and impulsivity on alcohol consumption. *Alcohol Clin Exp Res*. 2010; 34:1376–1385. [PubMed: 20491738]
25. Rezvafard M, Ekhtiari H, Mokri A, et al. Psychological and behavioral traits in smokers and their relationship with nicotine dependence level. *Arch Iran Med*. 2010; 13:395–405. [PubMed: 20804306]
26. Flory JD, Manuck SB. Impulsiveness and cigarette smoking. *Psychosom Med*. 2009; 71:431–437. [PubMed: 19251874]

27. Powell J, Dawkins L, West R, et al. Relapse to smoking during unaided cessation: Clinical, cognitive and motivational predictors. *Psychopharmacology (Berl)*. 2010; 212:537–549. [PubMed: 20703450]
28. 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]
29. Bickel WK, Madden GJ. A comparison of measures of relative reinforcing efficacy and behavioral economics: Cigarettes and money in smokers. *Behav Pharmacol*. 1999; 10:627–637. [PubMed: 10780504]
30. Mitchell SH. Measures of impulsivity in cigarette smokers and nonsmokers. *Psychopharmacology (Berl)*. 1999; 146:455–464. [PubMed: 10550496]
31. Reynolds B. Do high rates of cigarette consumption increase delay discounting? A cross-sectional comparison of adolescent smokers and young-adult smokers and nonsmokers. *Behav Processes*. 2004; 67:545–549. [PubMed: 15519004]
32. Kirby KN. Bidding on the future: Evidence against normative discounting of delayed rewards. *J Exp Psychol: General*. 1997; 126:54–70.
33. Ainslie G. Specious reward: A behavioral theory of impulsiveness and impulse control. *Psychol Bull*. 1975; 82:483–496.
34. Logue AW. Research on self-control: An integrating framework. *Behav Brain Sci*. 1988; 11:665–709.
35. Ohmura Y, Takahashi T, Kitamura N. Discounting delayed and probabilistic monetary gains and losses by smokers of cigarettes. *Psychopharmacology (Berl)*. 2005; 182:508–515. [PubMed: 16167142]
36. Field M, Santarcangelo M, Sumnall H, et al. Delay discounting and the behavioural economics of cigarette purchases in smokers: The effects of nicotine deprivation. *Psychopharmacology (Berl)*. 2006; 186:255–263. [PubMed: 16609902]
37. Yoon JH, Higgins ST, Heil SH, et al. Delay discounting predicts postpartum relapse to cigarette smoking among pregnant women. *Exp Clin Psychopharmacol*. 2007; 15:176–186. [PubMed: 17469941]
38. Krishnan-Sarin S, Reynolds B, Duhig AM, et al. Behavioral impulsivity predicts treatment outcome in a smoking cessation program for adolescent smokers. *Drug Alcohol Depend*. 2007; 88:79–82. [PubMed: 17049754]
39. MacKillop J, Kahler CW. Delayed reward discounting predicts treatment response for heavy drinkers receiving smoking cessation treatment. *Drug Alcohol Depend*. 2009; 104:197–203. [PubMed: 19570621]
40. Rotter JB. Generalized expectancies for internal versus external control of reinforcement. *Psychol Monogr*. 1966; 80:1–28. [PubMed: 5340840]
41. Erikson RV, Roberts AH. Some ego functions associated with delay of gratification in male delinquents. *J Consult Clin Psychol*. 1971; 36:378–382. [PubMed: 5561483]
42. Srinivasan N, Tikoo S. Effect of locus of control on information search behavior. *Adv Cons Res*. 1992; 19:498–504.
43. Platt JJ, Eisenman R. Internal-external control of reinforcement, time perspective, adjustment, and anxiety. *J Gen Psychol*. 1968; 79:121–128. [PubMed: 5672276]
44. Plunkett H, Buehner M. The relation of general and specific locus of control to intertemporal monetary choice. *Pers Individ Dif*. 2007; 42:1233–1242.
45. De Brabander B, Declerck C. A possible role of central dopamine metabolism associated with individual differences in locus of control. *Pers Individ Dif*. 2004; 37:735–750.
46. Rosenbaum M, Argon S. Locus of control and success in self-initiated attempts to stop smoking. *J Clin Psychol*. 1979; 35:870–872. [PubMed: 512019]
47. McKenna K, Higgins H. Factors influencing smoking cessation in patients with coronary artery disease. *Patient Educ Couns*. 1997; 32:197–205. [PubMed: 9423501]
48. Gregor KL, Zvolensky MJ, McLeish AC, et al. Anxiety sensitivity and perceived control over anxiety-related events: Associations with smoking outcome expectancies and perceived cessation barriers among daily smokers. *Nicotine Tob Res*. 2008; 10:627–635. [PubMed: 18418785]

49. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th edn.. Washington, DC: American Psychiatric Association; 1994.
50. Chambless DL, Baker MJ, Baucom DH, et al. Update on empirically validated therapies II. *Clin Psychologist*. 1998; 51:3–21.
51. Sheffer CE, Stitzer M, Payne TJ, et al. Treatment for tobacco dependence for rural, lower-income smokers: Outcomes, predictors, and measurement considerations. *Am J Health Promot*. 2009; 23:328–338. [PubMed: 19445436]
52. Payne TJ, Smith PO, Adams SG, et al. Pretreatment cue reactivity predicts end-of-treatment smoking. *Addict Behav*. 2006; 31:702–710. [PubMed: 15979814]
53. Schmitz JM, Rosenfarb IS, Payne TJ. Cognitive and affective responses to successful coping during smoking cessation. *J Subst Abuse*. 1993; 5:61–72. [PubMed: 8329881]
54. Schmitz JM, Tate JC. Treatment session frequency and smoking cessation. *J Subst Abuse*. 1994; 6:77–85. [PubMed: 7915923]
55. Fagerstrom KO, Schneider NG. Measuring nicotine dependence: A review of the Fagerstrom Tolerance Questionnaire. *J Behav Med*. 1989; 12:159–182. [PubMed: 2668531]
56. Heatherton TF, Kozlowski LT, Frecker RC, et al. The Fagerstrom Test for Nicotine Dependence: A revision of the Fagerstrom Tolerance Questionnaire. *Br J Addict*. 1991; 86:1119–1127. [PubMed: 1932883]
57. McKee SA, O'Malley SS, Salovey P, et al. Perceived risks and benefits of smoking cessation: Gender-specific predictors of motivation and treatment outcome. *Addict Behav*. 2005; 30:423–435. [PubMed: 15718060]
58. Heishman SJ, Saha S, Singleton EG. Imagery-induced tobacco craving: Duration and lack of assessment reactivity bias. *Psychol Addict Behav*. 2004; 18:284–288. [PubMed: 15482084]
59. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *J Health Soc Behav*. 1983; 24:385–396. [PubMed: 6668417]
60. Cohen S, Lichtenstein E. Perceived stress, quitting smoking, and smoking relapse. *Health Psychol*. 1990; 9:466–478. [PubMed: 2373070]
61. Kenford SL, Smith SS, Wetter DW, et al. Predicting relapse back to smoking: Contrasting affective and physical models of dependence. *J Consult Clin Psychol*. 2002; 70:216–227. [PubMed: 11860048]
62. Glasgow RE, Klesges RC, Mizes JS, et al. Quitting smoking: Strategies used and variables associated with success in a stop-smoking contest. *J Consult Clin Psychol*. 1985; 53:905–912. [PubMed: 4086690]
63. Brandon TH, Baker TB. The smoking consequences questionnaire: The subjective expected utility of smoking in college students. *Psychol Assess*. 1991; 3:484–491.
64. Brandon TH, Copeland AL, Saper ZL. Programmed therapeutic messages as a smoking treatment adjunct: Reducing the impact of negative affect. *Health Psychol*. 1995; 14:41–47. [PubMed: 7737072]
65. Wetter DW, Smith SS, Kenford SL, et al. Smoking outcome expectancies: Factor structure, predictive validity, and discriminant validity. *J Abnorm Psychol*. 1994; 103:801–811. [PubMed: 7822583]
66. Beck, AT.; Steer, RA.; Brown, GK. Manual for the Beck Depression Inventory. 2nd edn.. San Antonio, TX: The Psychological Corporation; 1996.
67. Watson D, Clark L, Tellegen A. Development and validation of brief measures of positive and negative affect. The PANAS scale. *J Pers Soc Psychol*. 1988; 54:1063–1070. [PubMed: 3397865]
68. Bornoalova MA, Cashman-Rolls A, O'Donnell JM, et al. Risk taking differences on a behavioral task as a function of potential reward/loss magnitude and individual differences in impulsivity and sensation seeking. *Pharmacol Biochem Behav*. 2009; 93:258–262. [PubMed: 19041886]
69. Barratt, ES. Impulsiveness defined within a systems model of personality. In: Speilberge, CD.; Butcher, JN., editors. *Advances in Personality Assessment*. Hillsdale, NJ: Erlbaum; 1985. p. 113-132.
70. Patton JH, Stanford MS, Barratt ES. Factor structure of the Barratt impulsiveness scale. *J Clin Psychol*. 1995; 51:768–774. [PubMed: 8778124]

71. Mazur, JE. An adjusting procedure for studying delayed reinforcement. In: Commons, ML.; Nevin, JA.; Rachlin, H., editors. *Quantitative Analysis of Behavior*. Hillsdale, NJ: Erlbaum; 1987. p. 55-73.
72. Eysenck SBG, Pearson PR, Easting G, et al. Age norms for impulsiveness, venture-someness, and empathy for adults. *Pers Individ Dif*. 1985; 6:613–619.
73. Eysenck SB, Eysenck HJ. Impulsiveness and venture-someness: Their position in a dimensional system of personality description. *Psychol Rep*. 1978; 43:1247–1255. [PubMed: 746091]
74. Dickman SJ. Functional and dysfunctional impulsivity: Personality and cognitive correlates. *J Pers Soc Psychol*. 1990; 58:95–102. [PubMed: 2308076]
75. Malloy P, Grace J. A review of rating scales for measuring behavior change due to frontal systems damage. *Cogn Behav Neurol*. 2005; 18:18–27. [PubMed: 15761273]
76. Grace J, Stout JC, Malloy PF. Assessing frontal lobe behavioral syndromes with the frontal lobe personality scale. *Assessment*. 1999; 6:269–284. [PubMed: 10445964]
77. Stout JC, Ready RE, Grace J, et al. Factor analysis of the frontal systems behavior scale (FrSBe). *Assessment*. 2003; 10:79–85. [PubMed: 12675387]
78. Velligan DI, Ritch JL, Sui D, et al. Frontal Systems Behavior Scale in schizophrenia: Relationships with psychiatric symptomatology, cognition and adaptive function. *Psychiatry Res*. 2002; 113:227–236. [PubMed: 12559479]
79. Colder CR, O'Connor R. Attention biases and dis-inhibited behavior as predictors of alcohol use and enhancement reasons for drinking. *Psychol Addict Behav*. 2002; 16:325–332. [PubMed: 12503905]
80. Verdejo-Garcia AJ, Perales JC, Perez-Garcia M. Cognitive impulsivity in cocaine and heroin polysubstance abusers. *Addict Behav*. 2007; 32:950–966. [PubMed: 16876962]
81. Lane SD, Moeller FG, Steinberg JL, et al. Performance of cocaine dependent individuals and controls on a response inhibition task with varying levels of difficulty. *Am J Drug Alcohol Abuse*. 2007; 33:717–726. [PubMed: 17891664]
82. Newman JP, Widom CS, Nathan S. Passive avoidance in syndromes of disinhibition: Psychopathy and extraversion. *J Pers Soc Psychol*. 1985; 48:1316–1327. [PubMed: 3998992]
83. Powell, DH.; Kaplan, EF.; Whitla, D., et al. *MicroCog: Assessment of Cognitive Functioning Manual*. San Antonio, TX: Psychological Corporation; 1993.
84. Zimbardo, PG. *Stanford Time Perception Inventory Manual*. Stanford, CA: Department of Psychology, Stanford University; 1992.
85. Petry NM, Bickel WK, Arnett M. Shortened time horizons and insensitivity to future consequences in heroin addicts. *Addiction*. 1998; 93:729–738. [PubMed: 9692271]
86. MacKillop J, Anderson EJ, Castelda BA, et al. Divergent validity of measures of cognitive distortions, impulsivity, and time perspective in pathological gambling. *J Gambl Stud*. 2006; 22:339–354. [PubMed: 16826455]
87. Tuckman BW. The development and concurrent validity of the Procrastination Scale. *Educ Psychol Meas*. 1991; 51:473–490.
88. Benowitz N. Biochemical verification of tobacco use and cessation. *Nicotine Tob Res*. 2002; 4:149–159. [PubMed: 12028847]
89. Liang KY, Zeger SL. Longitudinal data analysis using generalized linear models. *Biometrika*. 1986; 73:13–23.
90. Lee JH, Herzog TA, Meade CD, et al. The use of GEE for analyzing longitudinal binomial data: A primer using data from a tobacco intervention. *Addict Behav*. 2007; 32:187–193. [PubMed: 16650625]
91. Hall SM, Delucchi KL, Velicer WF, et al. Statistical analysis of randomized trials in tobacco treatment: Longitudinal designs with dichotomous outcome. *Nicotine Tob Res*. 2001; 3:193–202. [PubMed: 11506764]
92. Zvolensky MJ, Bernstein A, Marshall EC. Anxiety vulnerability factors and disorders and tobacco and marijuana use and disorders: Emerging theory and research explicating their relations. *Addict Behav*. 2008; 33:1383–1384. [PubMed: 18722720]

93. Lejuez CW, Aklin WM, Jones HA, et al. The balloon analogue risk task (BART) differentiates smokers and non-smokers. *Exp Clin Psychopharmacol.* 2003; 11:26–33. [PubMed: 12622341]
94. Mackillop J, Anderson EJ, Castelda BA, et al. Convergent validity of measures of cognitive distortions, impulsivity, and time perspective with pathological gambling. *Psychol Addict Behav.* 2006; 20:75–79. [PubMed: 16536668]
95. Cohen LG, Roth BJ, Nilsson J, et al. Effects of coil design on delivery of focal magnetic stimulation. Technical considerations. *Electroencephalogr Clin Neurophysiol.* 1990; 75:350–357. [PubMed: 1691084]
96. Copeland A, Brandon TH, Quinn EP. The Smoking Consequences Questionnaire-Adult: Measurement of smoking outcome expectancies of experienced smokers. *Psychological Assessment.* 1995; 7:484–494.
97. Frazier P, Keenan N, Anders S, et al. Perceived past, present, and future control and adjustment to stressful life events. *J Pers Soc Psychol.* 2011; 100:749–765. [PubMed: 21299308]
98. Berg CJ, Thomas JL, Guo H, et al. Predictors of smoking reduction among Blacks. *Nicotine Tob Res.* 2010; 12:423–431. [PubMed: 20194521]
99. Fecteau S, Knoch D, Fregni F, et al. Diminishing risk-taking behavior by modulating activity in the prefrontal cortex: A direct current stimulation study. *J Neurosci.* 2007; 27:12500–12505. [PubMed: 18003828]
100. Eichhammer P, Johann M, Kharraz A, et al. High-frequency repetitive transcranial magnetic stimulation decreases cigarette smoking. *J Clin Psychiatry.* 2003; 64:951–953. [PubMed: 12927012]
101. Bansal MA, Cummings KM, Hyland A, et al. Stop-smoking medications: Who uses them, who misuses them, and who is misinformed about them? *Nicotine Tob Res.* 2004; 6(Suppl. 3):S303–S310. [PubMed: 15799593]
102. Houston TK, Scarinci IC, Person SD, et al. Patient smoking cessation advice by health care providers: The role of ethnicity, socioeconomic status, and health. *Am J Public Health.* 2005; 95:1056–1061. [PubMed: 15914833]
103. Okuyemi KS, Ahluwalia JS, Richter KP, et al. Differences among African American light, moderate, and heavy smokers. *Nicotine Tob Res.* 2001; 3:45–50. [PubMed: 11260810]
104. Murphy JM, Mahoney MC, Hyland AJ, et al. Disparity in the use of smoking cessation pharmacotherapy among Medicaid and general population smokers. *J Public Health Manag Pract.* 2005; 11:341–345. [PubMed: 15958934]
105. Lopez-Quintero C, Crum RM, Neumark YD. Racial/ethnic disparities in report of physician-provided smoking cessation advice: Analysis of the 2000 National Health Interview Survey. *Am J Public Health.* 2006; 96:2235–2239. [PubMed: 16809587]
106. McMenamin SB, Halpin HA, Bellows NM. Knowledge of Medicaid coverage and effectiveness of smoking treatments. *Am J Prev Med.* 2006; 31:369–374. [PubMed: 17046407]
107. Cummins SE, Bailey L, Campbell S, et al. Tobacco cessation quitlines in North America: A descriptive study. *Tob Control.* 2007; 16(Suppl. 1):i9–i15. [PubMed: 18048639]
108. Mitchell SH. Effects of short-term nicotine deprivation on decision making: Delay, uncertainty and effort discounting. *Nicotine Tob Res.* 2004; 6:819–828. [PubMed: 15700917]
109. Bickel W, Yi R, Landes R, et al. Remember the future: Working memory training decreases delay discounting among stimulant addicts. *Biol Psychiatr.* 2011; 69:260–265.

TABLE 1

Characteristics of the participants

Category	Measure	Mean (SD)	Median (interquartile range)*	
Tobacco-related clinical factors	Fagerström Test for Nicotine Dependence	6.43(1.75)	6(5, 8)	
	Motivation	8.89(1.52)	10(8, 10)	
	Self-efficacy	7.42(2.54)	8(6, 10)	
	Perceived Stress Scale	22.16(8.07)	21(17,27)	
	Beck Depression Inventory II	11.58(10.56)	9(3,16)	
	Smoking Consequences Questionnaire Negative affect reduction scale	55.10(18.53)	59(40.5,70)	
	Positive and Negative Affect Scales	Positive affect scale Negative affect scale	16.96(6.88) 30.96(8.04)	15(12,20) 31.5(25, 37)
Executive function	Balloon Analogue Risk Task	Money earned Pumps Mean pumps	23.29(4.58) 601.32(233.04) 27.56(17.89,38.48)	
	Barratt Impulsiveness Scale	Motor impulsiveness Cognitive impulsiveness Nonplanning impulsiveness	11.43(4.32) 9.51(3.16) 16.82(4.08)	11(8, 14) 9(7,12) 17(14, 20)
	Delay discounting tasks	\$100 real gain	-4.62(2.88)	-4.92(-6.80, -2.48)
		\$100 hypothetical gain	-4.77(3.28)	-4.59(-6.29, -2.67)
		\$1,000 hypothetical gain	-5.86(3.42)	-6.08(-7.75, -3.18)
		Mean of \$100 real and \$100 and \$1,000 hypothetical gains	-5.55(2.93)	-5.68(-7.28, -3.46)
	Eysenck Impulsiveness Scale	Impulsiveness Venturesomeness	8.65(4.49) 5.75(3.23)	7(5, 12) 5(3, 8)
	Frontal Systems Behavior Scales	Executive dysfunction	61.20(14.76)	60(49, 72)
		Apathy	65.76(15.93)	63(52, 76)
		Disinhibition	60.98(16.26)	61(48, 71)
		Total	65.18(16.06)	65(52, 75)
	Go/No-Go Task	Plus milliseconds to respond correctly	416.9(188.5)	423.2(279.5, 558.2)
		Minus milliseconds to respond incorrectly	542.6(204.6)	569.2(481.1, 660.5)
		Total points	37.6(6.9)	37(32, 43)
	Microcog™	Attention/mental control	80.77(39.29)	96.5(73.5, 107)
		Memory	82.39(39.78)	99(79, 108.5)
		Spatial processing	81.18(37.76)	95(76.5, 105.5)
Reasoning/calculation		82.18(39.80)	95(80, 107)	
Reaction time		84.86(41.06)	102(93, 109)	
Information processing accuracy		74.51(36.25)	87(71.5, 99)	
Information processing speed		85.65(41.46)	103.5(81, 112.5)	
Cognitive functioning		88.01(44.40)	98(75, 119)	
	Cognitive proficiency	76.60(37.56)	85(75, 100)	

Category	Measure	Mean (SD)	Median (interquartile range)*
Stanford Time	Present-hedonistic	22.16(4.18)	22(19, 25)
	Perception	21.80(4.74)	22(18, 25)
Inventory	Future-oriented	46.52(7.62)	47(40, 53)
	Past-oriented	16.36(3.05)	16(15, 18)
Rotter's Locus of Control Scale		8.14(3.50)	8(5, 11)
Tuckman Procrastination Scale		42.70(13.26)	45(37, 50)

* Interquartile range: first value = 25th percentile and second value 75th percentile.

TABLE 2

Significant results from the generalized estimating equations models applied to each measure individually*

	Odds ratio for one SD increase	95% CI upper bound	<i>p</i> -Value
Fagerström Test for Nicotine Dependence (SD = 1.75)	.607	.863	.010
Perceived Stress Scale (SD = 8.07)	.571	.878	.017
Delay discounting mean baseline log <i>k</i> of real \$100 and hypothetical \$100 and \$1,000 gains (SD = 2.93)	.623	.912	.021
Delay discounting \$100 hypothetical gains (SD = 3.28)	.662	.942	.027
Delay discounting \$1,000 hypothetical gains (SD = 3.42)	.684	.966	.035
Barratt Impulsiveness Scale Cognitive impulsiveness (SD = 3.16)	.680	.966	.035
Rotter's Locus of Control Scale (SD = 3.50)	.681	.972	.038
Smoking Consequences Questionnaire Negative affect reduction scale (SD = 8.04)	.864	.992	.042

* All tests were one-tailed.

TABLE 3

Correlations among the measures that predicted abstinence

Measure	PSS	DD mean	DD \$100	DD \$1,000	BIS -Cog	LOC	SCQ-N	PANAS-N
FTND	.11	.12	.08	.12	.11	.11	.16	.08
PSS		.17	.09	.17	.37**	.29**	.28**	-.43**
DD Mean			.84**	.86**	.11	.05	.19	.05
DD \$100				.78**	.09	.00	.14	.11
DD \$1,000					-.02	.11	.09	.03
BIS-Cog						.21*	.43**	-.21*
LOC							.12	-.15
SCQ-N								-.05

FTND = Fagerström Test for Nicotine Dependence; PSS = Perceived Stress Scale; DD mean = Delay discounting mean baseline log k of real \$100, hypothetical \$100 and \$1,000 gains; DD \$100 = Delay discounting mean baseline log k of hypothetical \$100 gains; DD \$1,000 = Delay discounting hypothetical \$1,000 gains; BIS-Cog = Barratt Impulsiveness Scale 11-Cognitive impulsiveness; LOC = Rotters Locus of Control; SCQ-N = Smoking Consequences Questionnaire Negative affect scale; PANAS-N = Positive and Negative Affect Scale - Negative affect scale.

* $p < .05$;

** $p < .01$.