Published in final edited form as:
Drug Alcohol Depend. 2009 August 1; 103(3): 99–106. doi:10.1016/j.drugalcdep.2008.12.019.

Does delay discounting play an etiological role in smoking or is it a consequence of smoking?

Janet Audrain-McGovern[1], **Daniel Rodriguez**[1], **Leonard H. Epstein**[2], **Jocelyn Cuevas**[1], **Kelli Rodgers**[1], and **E. Paul Wileyto**[1]

[1]Department of Psychiatry, University of Pennsylvania, 3535 Market St., Suite 4100, Philadelphia, PA 19104

[2]Department of Pediatrics, University of Buffalo School of Medicine & Biomedical Sciences, Farber Hall, Room G56, 3435 Main St., Building #26, Buffalo, NY 14214

Abstract

Although higher delay discounting rates have been linked to cigarette smoking, little is known about the stability of delay discounting, whether delay discounting promotes smoking acquisition, whether smoking contributes to impulsive choices, or if different relationships exist in distinct subgroups. This study sought to fill these gaps within a prospective longitudinal cohort study (N=947) spanning mid adolescence to young adulthood (age 15 to 21 years old). Smoking and delay discounting were measured across time. Covariates included peer and household smoking, academic performance, depression, novelty seeking, inattention and hyperactivity/impulsivity symptoms, and alcohol and marijuana use. The associated processes Latent Growth Curve Modeling (LGCM) with paths from the delay discounting level factor (baseline measure) and the trend factor (slope) to the smoking trend factor (slope) fit the data well, $X^2_{(19,\ n=947)} = 15.37$, p=.70, CFI=1.00, RMSEA=0, WRMR=.36. The results revealed that delay discounting did not change significantly across time. Baseline delay discounting had a significant positive effect on smoking trend (β=.08, z=2.16, p=.03). A standard deviation (SD=1.41) increase in baseline delay discounting resulted in an 11% increase (OR=1.11, 95% CI= 1.03, 1.23) in the odds of smoking uptake. The alternative path LCGM revealed that smoking did not significantly impact delay discounting (p's > .05). Growth Mixture Modeling identified three smoking trajectories: nonsmokers, early/fast smoking adopters, and slow smoking progressors. Delay discounting was higher in the smoking versus nonsmoking trajectories, but did not discriminate between the smoking trajectories, despite different acquisition patterns. Delay discounting may provide a variable by which to screen for smoking vulnerability and help identify subgroups to target for more intensive smoking prevention efforts that include novel behavioral components directed toward aspects of impulsivity.

Keywords

Delay discounting; smoking acquisition

Janet Audrain-McGovern, Ph.D., Tobacco Use Research Center, Department of Psychiatry, University of Pennsylvania, 3535 Market Street, Suite 4100, Philadelphia, PA 19104, USA. Tel.: 1-215-746-7145; fax: 1-215-746-7140. E-mail address: audrain@mail.med.upenn.edu..

1. Introduction

Impulsivity is an important factor influencing cigarette smoking as well as other substances of abuse (Kreek et al., 2005; Mitchell, 1999). It can be defined, in part, by delay discounting, which describes the tendency to discount the value of a reward as a function of the length of delay to its delivery (Madden, 2000). Individuals who score higher on measures of delay discounting prefer more immediate rewards at the expense of larger rewards later (Monterosso and Ainslie, 1999). Across substances of abuse, current substance abusers discount delayed rewards more rapidly than non-users or controls (Kirby et al., 1999; Madden et al., 1997). For example, current smokers tend to discount future monetary reinforcers more than ex-smokers and nonsmokers (Bickel et al., 1999), while light and heavy smokers do not differ in their discounting rates for either money or cigarettes (Johnson et al., 2007). However, little is known about the stability of delay discounting across time (state versus trait), whether delay discounting promotes smoking acquisition, whether smoking contributes to impulsive choices, or if different relationships exist in distinct subgroups.

Is delay discounting a state that may change across time or does it reflect a stable disposition? Within-subjects designs suggest that delay discounting is stable across a 3-month period in healthy young adults (21 years old) (Ohmura et al., 2006) and stable across at least 1-week in nicotine dependent smokers (age 31 years). Between-group cross-sectional comparisons of young adolescents (age 12), young adults (average age 20 years old), and older adults (average age 70 years old) indicate that young adolescents discount monetary rewards more steeply than young adults, who discount at a faster rate than older adults (Green et al., 1994). Research to date has not prospectively examined in a within subject manner whether delay discounting is stable over longer periods of time or developmental periods.

Likewise, only cross-sectional data are available regarding delay discounting, smoking and other substance use. For example, smokers who smoke more cigarettes have higher rates of delay discounting (Reynolds, 2004). Similarly, impulsivity positively correlates with smoking frequency in high school students (Lewinsohn et al., 2000). College students who reported an earlier age of smoking onset had higher delay discounting rate (Kollins, 2003). However, cross-sectional data do not permit a determination of the temporal precedence between smoking and delay discounting. Animal models of delay discounting and substance use have found that rats exhibiting a greater rate of delay discounting acquired cocaine self-administration faster (Perry et al., 2005), suggesting that delay discounting may precede substance use. Alternatively, animal models have not consistently shown that acute administration of drugs (e.g., delta-9-tetrahydrocannabinol, ethanol, stimulants) increases delay discounting rates (McDonald et al., 2003; Richards et al., 1999b; Wade et al., 2000). However, chronic high doses of methamphetamine and cocaine have been shown to increase the degree to which rats discounted the value of delayed rewards (Richards et al., 1999a; Roesch et al., 2007), suggesting that substance use may promote impulsive choices.

We must also consider the possibility that delay discounting and smoking reciprocally influence each other. That is, higher delay discounting predisposes adolescents to make impulsive choices such as smoking, and smoking in turn, accentuates trait levels of delay discounting. Likewise, the relationship between delay discounting and smoking may vary depending on the subgroup; delay discounting may contribute to smoking acquisition in some adolescents whereas smoking contributes to the development of delay discounting in other adolescents. Given the developmental heterogeneity in smoking acquisition, subgroups defined by higher rates of delay discounting may have earlier smoking onset or a faster progression along the smoking uptake continuum than individuals with lower levels of delay discounting. For example, early onset alcoholics discount monetary rewards more than late-onset alcoholics (Dom et al., 2006), needle sharing-heroin users have higher discounting rates than non-needle

sharing heroin users (Odum et al., 2000), and early onset substance use, polysubstance use, and problems associated with substance use were associated with higher delay discounting rates in college students (Kollins, 2003).

The present study sought to fill these critical gaps in the literature by investigating the stability of delay discounting, the temporal precedence between delay discounting and smoking, and potential heterogeneity in the relationship between smoking and delay discounting within a prospective longitudinal cohort study spanning mid adolescence to young adulthood (age 15 to 21 years old). The majority of regular smokers report initiating smoking during adolescence and 75% report smoking regularly by age 18 (USDHHS, 1994). Smoking and delay discounting are repeatedly measured over these two developmental periods. A better understanding of the causal pathways between smoking and impulsive choices may promote distinct treatment implications and help inform smoking prevention and intervention efforts. Based on the ties between delay discounting and the dispositional trait of impulsivity (Mitchell, 1999), we anticipated that delay discounting would be fairly stable over mid adolescence to early adulthood. Given the expected stability in delay discounting, we further anticipated that delay discounting would contribute to smoking uptake rather than smoking progression promoting increases in delay discounting. Finally, we hypothesized that delay discounting would discriminate between smoking and nonsmoking trajectories and between smoking trajectories that differ with respect to smoking onset and magnitude.

2. Methods

2.1. Sample

Participants were high school students (53% female and 65% White) taking part in a longitudinal study of the social, psychological, and genetic determinants of adolescent smoking adoption. Participants were enrolled in one of five public high schools in northern Virginia. This cohort was drawn from the 2,393 students identified through class rosters at the beginning of ninth grade. Students were ineligible to participate in this study if they had a special classroom placement (e.g., severe learning disability). Based on the selection criteria, a total of 2,120 (89%) students were eligible to participate, and of these, 1,533 (72%) parents provided a response regarding their teen's participation. Of the 1,533 parents who provided a response 1,151 (75%) consented to their teen's participation, yielding an overall consent rate of 54%. Analysis of differences between students whose parents did and did not consent revealed that the likelihood of consent was greater for white parents with more than a high school education than for parents with a high school education or less (89% vs.77%) (Audrain et al., 2002).

The majority of adolescents with parental consent provided their assent (99%, N=1136). The adolescent cohort was formed in the 9^{th} grade and was followed until the end of 12^{th} grade. Five data collection waves were completed on-site during compulsory classes during high school: spring 9^{th} grade; fall and spring10^{th} grade; spring 11^{th} grade and spring 12^{th} grade (age range 14 - 18 years old). Each survey took approximately 30 minutes to complete. The cohort was measured annually each spring the four years following high school. Delay discounting was measured at three time points. It was first measured in 10^{th} grade spring and in the first two years after high school. Thus, the current analyses span these time points (~ age 15 to 20 years of age). Participants were individuals (N=947) with complete data on the covariates. University Institutional Review Board approval of the study protocol was obtained. Approximately 147 participants were lost to follow-up. There was no significant difference between those lost to follow-up and those retained on smoking ($p>.05$) although the difference between those lost to follow-up and those retained on delay discounting approached significance ($p=.055$) (-.413 versus -.438).

2.2. Outcome Measures

2.2.1. Smoking—Smoking was assessed at each annual wave with thirteen standard epidemiological questions, such as "Have you tried or experimented with cigarette smoking, even a few puffs?" and "When was the last time you smoked a cigarette?" (CDC, 2006). Based on the responses to these items, a 5-level variable representing increasing levels of smoking within the past 30 days was generated (Rodriguez et al., 2007). The ordered categories were: 0 - did not smoke in the past month; 1 - smoked "1 month ago or less;" 2 - smokes "...at least once a week;" 3 - smokes ≤ 10 cigarettes daily; 4 - smokes > 10 cigarettes daily.

2.2.2. Delay Discounting—Delay discounting was measured from the pattern of choices across 27 questions on a monetary choice questionnaire (Kirby et al., 1999). The 27 choices define 10 ranges of discount rates with delays ranging from 7 days to 186 days. Delay discounting is measured by fitting a hyperbolic function to bivariate data on indifference points between choices of small, medium, and large delayed rewards and the time delay. The resulting estimated parameter (k) is greater for individuals who discount the value of future rewards and thus prefer immediate rewards (Kirby, 1997; Kirby et al., 1999; Madden et al., 1997; Myerson and Green, 1995).

2.3. Covariates

The demographic variables included gender and race (white versus non-white). Psycho-social and behavioral covariates included perceived academic performance, peer smoking, household smoking, depression, novelty-seeking, inattention and hyperactivity symptoms, and alcohol and marijuana use. Perceived academic performance was assessed with a single item that asked, "How do you do in school? Scores ranged from 4= "Mostly A's" to 1 = "Mostly D's and F's" (Audrain-McGovern et al., 2004a; Bergen et al., 2005). Household smoking was measured with an item that asked if any member of the household smokes (no one versus someone). Peer smoking was measured at each annual wave by summating responses to three items asking whether the adolescents' best friend smokes, and whether and, if so, how many of his or her other four best male and four best female friends smoke (range 0 to 9 friends smoking) (Audrain-McGovern et al., 2006a; Choi et al., 1997).

Depression symptoms were measured with the Center for Epidemiologic Studies Depression Scale (Radloff, 1977). This 20-item Likert-style scale has high internal consistency (Cronbach's alpha = .85-.90). Research supports the validity and reliability of the CES-D for use in adolescents and young adults (Lewinsohn et al., 1998; Radloff, 1991; Roberts et al., 1990). Response options range from 0 (none of the time) to 3 (most of the time).

Novelty-seeking was measured with the Temperament & Character Inventory (TCI) (20 True/False items KR-20 = .74) (Cloninger et al., 1994). Novelty-seeking and similar constructs as measured by the TCI, and its predecessor the Temperament Personality Questionnaire, have been linked to adolescent smoking and substance use (Audrain-McGovern et al., 2004a; Wills et al., 1994; Wills et al., 1998).

Inattention and hyperactivity symptoms were assessed with the 18 item Current Symptoms Scale-Self Report Form (Barkley and Murphy, 1998), which asks individuals to describe their behavior during the past 6 months, on 18 clinically relevant attention deficit and hyperactivity symptoms using a four-point Likert scale (0=Never or rarely to 3=often). The two 9-item subscales (inattention symptoms and hyperactivity-impulsivity) and the total score have adequate reliability (α= .78 -.84) and predictive validity (Barkley and Murphy, 1998; Rodriguez et al., 2008).

Marijuana use was assessed with one item asking ”During the past 30 days, how many times have you used marijuana? Alcohol use was assessed with one item asking, “During the past 30 days, on how many days did you have at least one drink (not just a sip) of alcohol?” Response choices ranged from 0 to all 30 days for alcohol use and 0 to 40 or more times for marijuana use (Grunbaum et al., 2004). These covariates were measured at tenth grade (baseline).

2.4. Analysis

2.4.1. Latent Growth Curve Modeling (LGCM)—LGCM is a multivariate method that models repeated observed measures (e.g., smoking) on random coefficients representing baseline level and rate of change (trend) from baseline (Duncan et al., 1999). As such, LGCM allows for assessment of the average developmental trajectory for a given observed measure (e.g., smoking), and the effect of different predictors on baseline level and rate of change. In the present analysis, we conducted associated processes LGCM. Associated processes LGCM extends LGCM by allowing the testing of paths among factors from two or more LGCMs (Duncan et al., 1999). Two associated processes were modeled in the present study, one for the five-level ordered categorical smoking variable and one for the continuous variable delay discounting. Since delay discounting was only measured in 10^{th} grade, and one and two years post high school, the fixed factor loadings from the trend factor, representing time, were set to 0 (baseline), and 1 and 3, for the first and second years post high school, respectively, for both delay discounting and smoking; one unit of time equaled one year. As smoking was an ordered categorical variable, we estimated model parameters with a Weighted Least Squares estimation technique (WLSMV) in which the diagonal weight matrix uses robust standard errors, and the chi-square test statistic is Mean and Variance adjusted (Muthén and Muthén, 1998-2004). Model fit was evaluated with model chi-square, Comparative Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA), and weighted Root Mean Residual (WRMR). Suggested criteria for model fit are non-significant model chi-square, CFI above .95, RMSEA below .05-.08, and a WRMR value below .9 (Loehlin, 2004; Muthén and Muthén, 2001). Analyses were conducted using M*plus*, version 5.1, software (Muthén and Muthén, 1998-2007).

2.4.2. Growth Mixture Modeling (GMM)—GMM extends LGCM by exploring developmental heterogeneity among distinct trajectories of a dependent measure like smoking (Jung and Wickerama, 2008; Muthén, 2004; Nylund et al., 2006) and permits characterization of trajectories on select covariates. Although there are no agreed upon criteria for the optimal number of trajectories, the most widely used criterion is to select the model with the lowest Bayesian Information Criterion (BIC) value (Boscardin et al., 2008; Jung and Wickerama, 2008). A second criterion is average classification probability; the probability that each individual is placed in the correct class. Classification probabilities should be close to 1. A third criterion is the Bootstrap Likelihood ratio test (BLRT), which uses bootstrap samples to empirically estimate the distribution of the likelihood difference between neighboring (k and k-1 classes) models (Nylund et al., 2006). This test assesses whether the addition of a class significantly improves fit, with a non-significant *p* value favoring the k-1 class model (see Nylund et al., 2006, for a description of the BLRT). We use a combination of these criteria, along with a substantive assessment of the trajectory classes to select the optimal number of latent trajectories of smoking (Muthén, 2004). As smoking was measured in 10^{th} through 12^{th} grade, along with the first two years post high school, we will conduct GMM over these five waves. These analyses were conducted using M*plus*, version 5.1, software (Muthén and Muthén, 1998-2007).

2.4.3. Missing Data—To account for missing data, multivariate modeling used all available data. M*plus* allows modeling with missing data using maximum likelihood estimation of the mean, variance, and covariance parameters, when requested, employing the Expectation

Maximization (EM) algorithm, assuming data are missing at random (Muthén, 1998-2004). This approach was considered appropriate as analyses indicated that the data were missing at random (MAR), with no significant relationship between missingness and the dependent variables, only a relationship between missingness and the covariate race (greater missing data among non-whites at follow-up). We only accounted for missing data on the repeated measure of smoking and delay discounting in the associated processes LGCM (three waves) and smoking in the GMM (five waves), not the covariates. Thus, cases with missing data on the covariates were not included in the analysis. As such, the analyses were based on 947 adolescents in the LGCM and 909 adolescents in the GMM since delay discounting was treated as a covariate in the GMM rather than a dependent variable.

3. Results

3.1. Descriptive Statistics

The sample was 53% female and 68% white. At baseline (10th grade) about 25% of the adolescents lived with smokers, 30% used marijuana at least once in the past month, and 14% had at least one alcoholic drink in the past month. On average, the participants perceived their academic performance to be in the B range (M=3.17, SD=.57). On average, participants reported more than one smoking peer (M=1.84, SD= 2.28). The means and standard deviations for the psychological measures are as follows: depression symptoms (M=14.43, SD=10.32), novelty-seeking (M=10.77, SD=3.94), inattention (M=5.85, SD=4.54), and hyperactivity - impulsivity (M=6.56, SD=4.39). At baseline, 5% of adolescents were smoking daily, 7% weekly, and 13% smoked at least once a month. At one and two years post high school, 10% of individuals were smoking daily, 15% weekly, and 25% smoked at least once a month.

3.2. Latent Growth Curve Models

The associated processes LGCM with paths from baseline delay discounting level and trend factors to the smoking trend factor fit the data well, $X^2_{(19,\ n=947)} = 15.37$, p=.70, CFI=1.00, RMSEA=0, WRMR=.36. The associated processes LGCM revealed that the average delay discounting trend was not significant, indicating insignificant change in delay discounting from baseline (i.e., stability). Figure 1 presents the standardized path coefficients for the significant model pathways. The nonstandardized parameter estimates, standard errors, test statistics, and p values for all model paths are presented in Table 1. Baseline delay discounting had a significant positive effect on smoking trend, (β=.08, z=2.16, p=.03). As the path coefficient from a predictor to the dependent variable (latent or measured) is the log odds change in the latter for a unit change in the former, a standard deviation (SD=1.41) increase in baseline delay discounting (level) resulted in a 11% increase (OR=1.11, 95% CI= 1.03, 1.23) in the odds of smoking progression. However, the effect of delay discounting trend on smoking trend was not significant. Regarding the covariates, a standard deviation (SD=3.89) increase in novelty seeking was associated with a 24% increase in the odds of smoking progression (OR=1.24, 95% CI=1.13, 1.36). Alcohol use in the past 30 days (OR=1.41, 95% CI= 1.30, 1.53) and marijuana use in the past 30 days (OR=1.37, 95% CI= 1.23, 1.53) increased the likelihood of smoking at baseline as did and peer smoking (OR=1.18, 95% CI= 1.14, 1.23) and household smoking (OR=1.43, 95% CI= 1.19, 1.71). Higher academic performance (OR=.69, 95% CI= . 59, .81) decreased the odds of smoking at baseline. There were no effects for any covariates on smoking trend.

With respect to delay discounting, being female (β= -.34, z= -3.56, p<.0001) and higher academic performance (β= -.25, z= -2.73, p=.01) were associated with lower delay discounting rates at baseline. Being non-White (β= .25, z= 2.54, p=.01), having more peers whom smoke (β= .05, z= 1.97, p=.05), and higher novelty seeking (β= .04, z= 2.90, p=.004) were associated with higher delay discounting at baseline. With respect to delay discounting trend, depression

symptoms was associated with a decreased rate of change in delay discounting over time (β= -.004, z= -2.26, p=.03).

We also assessed whether smoking influences delay discounting, reversing the paths to test whether baseline smoking and smoking trend significantly affect delay discounting trend. Neither path was significant ($p > .05$).

3.3. Growth Mixture Models

We first determined the optimal number of smoking trajectories. We ran models with two to six latent trajectory classes without covariates. The empirical criteria are presented in Table 2. The BIC continued to decrease from model 1 (BIC=5908.52) to model 5 (5721.17), suggesting model 5. However, although the BLRT was significant through model 4, it was not significant for model 5, suggesting a non significant likelihood ratio difference between the five and six latent class models. Based on a combination of these empirical criteria, we selected the five trajectory class model. However, after adding the covariates to the model, and again evaluating the criteria, the best model included three trajectory classes, as the BLRT failed to reach significance comparing the four to three class models. Thus, we decided upon three trajectory classes (model 7). Based on the distribution of participants in each of the three trajectory classes, they are labeled as non-smokers (n=556), fast smoking adopters (n=112), and slow smoking progressors (n=241).

3.3.1. Characterizing trajectory classes on covariates—We characterized trajectory classes on covariates using multinomial logistic regression analysis, assessing the likelihood of membership to one trajectory class compared to another trajectory class. Table 3 provides odds ratios and 95% confidence intervals for all model comparisons. A one standard deviation (SD=1.41) increase in delay discounting was associated with 31% decrease (OR=.69, 95%CI=.51,.94) in the odds of being a nonsmoker compared to a fast smoking adopter, and a 26% decrease (OR=.74, 95%CI=.57,.96) in the odds of being a nonsmoker compared to a slow smoking progressor. Delay discounting, however, did not discriminate between slow progressors and fast smoking adopters. Similarly, a standard deviation (SD=3.89) increase in novelty seeking was associated with a 35% decrease (OR=.65, 95%CI=.46,.93) in the odds of being a nonsmoker compared to a fast smoking adopter, and a 27% decrease (OR=.73, 95% CI=.55,.96) in the odds of being a nonsmoker compared to a slow progressor. Like delay discounting, novelty seeking also did not discriminate between slow progressors and fast smoking adopters. Regarding the remaining covariates, nonsmokers were lower in past month alcohol use than fast smoking adopters (OR=.33, 95%CI=.18,.59) and slow progressors (OR=.31, 95%CI=.18,.54). They were also lower in peer smoking than fast smoking adopters (OR=.63, 95%CI=.53,.75) and slow smoking progressors (OR=.81, 95%CI=.70,.95). However, nonsmokers were higher in academic performance than fast (OR=2.71, 95%CI=1.49, 4.92) but not slow progressors. Comparing slow smoking progressors and fast smoking adopters, slow smoking progressors used less marijuana (OR=.59, 95%CI=.39,.89) and had fewer smoking peers (OR=.78, 95%CI=.67,.89) than fast smoking adopters. There were no other significant differences comparing trajectory classes. Figure 2 depicts these smoking trajectories.

4. Discussion

To our knowledge, this is the first study to date to examine the longer term stability of delay discounting, the causal relationship between delay discounting and smoking, and the evidence for heterogeneity in the relationship between smoking and delay discounting. The findings indicate that delay discounting is more trait-like than state-like across adolescence to young adulthood. Related to its stability across time, delay discounting appears to promote smoking acquisition, but smoking does not significantly alter delay discounting. Finally, delay

discounting appears to be elevated in those individuals who progress to a more regular pattern of smoking versus those who do not. In fact, delay discounting is a stronger predictor of smoking status than the pattern of smoking acquisition.

As delay discounting is considered to be an aspect of impulsivity and is relatively stable across adolescence and emerging adulthood, it makes sense that delay discounting would play an etiological role in smoking acquisition, rather than smoking playing an etiological role in impulsive choices measured by delay discounting. These results may help us better understand the dynamic nature between impulsive choices and smoking uptake. Adolescents higher in delay discounting may seek out activities that have more immediate rather than more delayed rewards, such as smoking and substance use. These results may also shed light on why some adolescents choose healthy rewarding behaviors (e.g., physical activity) and others choose unhealthy rewarding behaviors (e.g., smoking) (Audrain-McGovern et al., 2003; Audrain-McGovern et al., 2006b). If higher rates of delay discounting contribute to smoking acquisition, then delay discounting may provide a variable by which to screen for smoking vulnerability. Adolescents at higher risk of smoking due to higher delay discounting may be a subgroup to target for more intensive smoking prevention efforts that include novel behavioral components directed toward aspects of impulsivity. Intervention research suggests that impulsive decision making may be moderated by the acquisition of self-control skills. For example, a class-room based behavioral management intervention focused on reducing aggressive (e.g., fighting) and disruptive (e.g., shouting out of turn) behaviors in first and second graders reduced the risk of early onset smoking initiation (age 12), smoking initiation by age 14 years old for boys, and regular smoking in young men (19 - 21 years old) (Kellam and Anthony, 1998; Kellam et al., 2008a; Storr et al., 2002). Proscribed behaviors were met with a team of classmates losing points. Teams received tangible rewards (e.g., classroom activities, stickers, erasers) for their points when no member exhibited the proscribed behaviors during the sessions. The rewards were delivered immediately at first and then delayed to the end of the school day and eventually the end of the school week. Early interventions, such as these may interrupt the development of impulsive behaviors, or reduce their occurrence by bolstering self control skill sets, including delaying gratification.

Delay discounting characterized smoking uptake, but not how individuals progressed along the uptake continuum (average delay discounting scores at baseline: -3.79 for fast adopters versus -4.15 for slow progressors versus -4.58 for nonsmokers). Although not measured in this study, delay discounting may help determine who quits smoking successfully. A recent adolescent smoking cessation study found that adolescents who were not abstinent from smoking at study end (4 weeks) discounted monetary rewards more than those adolescents who were abstinent (Krishnan-Sarin et al., 2007). In addition, elevated impulsivity measured prior to smoking cessation treatment predicted faster time to relapse in adult smokers (Doran et al., 2004).

The higher delay discounting scores of the faster smoking adopters and greater smoking rate is consistent with the cross-sectional research findings showing that individuals who begin using substances early, have greater use, and more poly-substance use tend to have high discount rates (Dom et al., 2006; Kollins, 2003). At age 16, fast adopters were over 50% more likely to have used marijuana in the past month compared to slow progressors, and slow progressors did not differ from nonsmokers on academic performance. Higher delay discounting scores may be a marker for early onset of smoking, heavier smoking rate, and other issues such as concurrent substance use and poorer academic performance. Interventions to disrupt one or a range of problematic behaviors associated with smoking (e.g., poor grades, alcohol use) may indirectly impact smoking acquisition, but such an intervention may be more difficult or less effective than modifying a common etiologically important antecedent early, during developmentally malleable periods (Kellam et al., 2008b). Thus, the prevention of the

direct effect of delay discounting on smoking progression and the indirect effect via behaviors related to smoking may have a significant and meaningful impact on smoking uptake.

Those individuals in the slow smoking progressors trajectory had lower delay discounting scores, slower smoking uptake, and lower overall smoking rates. Is this distinct pattern of uptake a reflection of relatively lower delay discounting or is it the loss of factors that protect against smoking over time and an increase in risk factors associated with smoking progression? Members of this trajectory had more peers who smoked and greater alcohol use at baseline compared to nonsmokers. Our previous research has shown that delay discounting can impact smoking progression indirectly through the choice of other behaviors with more immediate rewards that are associated with smoking, such as substance use (Audrain-McGovern et al., 2004b). Delay discounting may also reflect variability in genetic liability for substance use in general, including smoking. Delay discounting may serve as an endophenotype between gene action and acquisition phenotypes (Audrain-McGovern et al., in press). These results provide some initial evidence of the predictive validity of delay discounting, which is an important criterion for defining a potential endophenotype. Whether delay discounting meets the criteria for an endophenotype for smoking acquisition specifically and substance abuse more generally awaits further investigation.

As the first investigation of the role of delay discounting in smoking acquisition, the present study has both strengths and limitations. The strengths include multiple measures of delay discounting and smoking across five years and two developmental periods, a good retention rate, the assessment of both directional paths (i.e., the path from delay discounting to smoking and the path from smoking to delay discounting), and the assessment of the developmental heterogeneity in smoking and how it is characterized by delay discounting. Despite these strengths, limitations of the study should be noted. One limitation is that there were insufficient numbers of adolescents in other racial groups to conduct meaningful analyses stratified by race. Another potential limitation of this study is the consent rate. Although the difference in those parents who originally provided consent and those who did not was relatively small and few (Audrain et al., 2002) caution is warranted in generalizing the results, despite the sample being regionally and locally representative with respect to demographics and smoking behavior (Audrain-McGovern et al., 2004a). One could also argue that the use of the delay discounting questionnaire rather than a lab task is a limitation, as the questionnaire may be less sensitive. The questionnaire has been shown to correlate highly ($r = .82$) with lab based measures of delayed discounting (Epstein et al., 2003). In addition, the variables, including the smoking data are based on self report. Research supports the validity of adolescent self report of smoking behavior as well as other sensitive behaviors in nontreatment contexts where confidentiality is emphasized (Botvin and Botvin, 1992; Velicer et al., 1992; Wills and Cleary, 1997). Finally, the latent growth curve model is based on only three time points (the minimum number needed) given that delay discounting was measured at three time points. Although more than three time points may have offered greater power and stability of the parameter estimates (Muthen and Muthen, 2008), our model fit the data well, but the results will need to be replicated.

Contrary to previous research in community samples, the present study did not find a significant relationship between ADHD symptoms and smoking (Galera et al., 2005; Kollins et al., 2005). We can only speculate that the presence of novelty-seeking and delay discounting in the model dampened the individual effects of inattention and hyperactivity/impulsivity on smoking progression or the discrimination between smoking and nonsmoking trajectories. Likewise, our statistical models considered the effects of ADHD symptom subtypes on changes in smoking across time, rather than an end state smoking outcome. Our previous research has shown that ADHD symptoms have significant effects on the developmental trajectory of nicotine dependence (Rodriguez et al., 2008).

Despite the limitations noted above, the present study provides initial evidence of the etiological role of delay discounting in smoking acquisition, irrespective of the onset time point or the rate and magnitude of acquisition. It is important to note that the impact of delay discounting on smoking acquisition was somewhat modest (OR = 1.11) while controlling for other influences on smoking behavior, such as novelty-seeking, ADHD symptoms, and other substance use. These findings help define the average contribution of delay discounting to smoking uptake and also indicate that delay discounting makes a unique contribution beyond important personality constructs and externalizing behaviors. It is important to note that we did not measure conduct disorder or delinquency in this cohort study, which could be considered a limitation. However, recent research suggests that 50% of adolescents with early exposure to alcohol and illicit drugs do not have a history of conduct problems, but are still at an increased risk for substance dependence in adulthood (Odgers et al., in press). This suggests that other factors besides the traditional problem behaviors may be involved in adolescent substance use, including cigarette smoking. Future research should determine the joint contributions of impulsive decision making, impulsive behavior, and other mood variables (e.g., depression) that may impact decisions about reward and play a role in smoking uptake (Forbes et al., 2007; Spring et al., 2003; Windle and Windle, 2001). The identification of variables that facilitate and buffer the impact of delay discounting on smoking uptake may also inform intervention planning by identifying who may benefit most from an intervention and what variables could be targeted to prevent smoking progression.

References

Audrain-McGovern J, Nigg JT, Perkins K. Phenotypes and Endophenotypes: Foundations for Genetic Studies of Nicotine Use and Dependence. Endophenotypes for nicotine dependence risk at or before initial nicotine exposure. National Cancer Institute Monograph, No 22. in pressChapter 8

Audrain-McGovern J, Rodriguez D, Moss HB. Smoking progression and physical activity. Cancer Epidemiol Biomarkers Prev 2003;12:1121–1129. [PubMed: 14652270]

Audrain-McGovern J, Rodriguez D, Tercyak KP, Cuevas J, Rodgers K, Patterson F. Identifying and characterizing adolescent smoking trajectories. Cancer Epidemiol Biomarkers Prev 2004a;13:2023–2034. [PubMed: 15598757]

Audrain-McGovern J, Rodriguez D, Tercyak KP, Epstein LH, Goldman P, Wileyto EP. Applying a behavioral economic framework to understanding adolescent smoking. Psychol Addict Behav 2004b; 18:64–73. [PubMed: 15008687]

Audrain-McGovern J, Rodriguez D, Tercyak KP, Neuner G, Moss HB. The impact of self-control indices on peer smoking and adolescent smoking progression. J Pediatr Psychol 2006a;31:139–151. [PubMed: 16467314]

Audrain-McGovern J, Rodriguez D, Wileyto EP, Schmitz KH, Shields PG. Effect of team sport participation on genetic predisposition to adolescent smoking progression. Archives of General Psychiatry 2006b;63:433–441. [PubMed: 16585473]

Audrain J, Tercyak KP, Goldman P, Bush A. Recruiting adolescents into genetic studies of smoking behavior. Cancer Epidemiol Biomarkers Prev 2002;11:249–252. [PubMed: 11895873]

Barkley, RA.; Murphy, KR. Attention-deficit hyperactivity disorder: A clinical workbook. Guilford Press; New York, NY: 1998.

Bergen HA, Martin G, Roeger L, Allison S. Perceived academic performance and alcohol, tobacco and marijuana use: longitudinal relationships in young community adolescents. Addictive Behaviors 2005;30:1563–1573. [PubMed: 16122617]

Bickel WK, Odum AL, Madden GJ. Impulsivity and cigarette smoking: delay discounting in current, never, and ex-smokers. Psychopharmacology 1999;146:447–454. [PubMed: 10550495]

Boscardin CK, Muthén BO, Francis DJ, Baker EL. Early identification of reading difficulties using heterogeneous developmental trajectories. Journal of Educational Psychology 2008;100:192–208.

Botvin GJ, Botvin EM. Adolescent tobacco, alcohol, and drug abuse: prevention strategies, empirical findings, and assessment issues. J Dev Behav Pediatr 1992;13:290–301. [PubMed: 1506471]

Centers for Disease Control and Prevention. Youth Risk Behavior Surveillance, United States, 2005. Surveillance Summaries June 9;2006 55(SS05):1–108.

Choi WS, Pierce JP, Gilpin EA, Farkas AJ, Berry CC. Which adolescent experimenters progress to established smoking in the United States. Am J Prev Med 1997;13:385–391. [PubMed: 9315272]

Cloninger, CR.; Przybeck, TR.; Svrakic, DM.; Wetzel, RD. The temperament and character inventory (TCI): A guide to its development and use. Center for Psychobiology and Personality; St.Louis, MO: 1994.

Dom G, Hulstijn W, Sabbe B. Differences in impulsivity and sensation seeking between early- and late-onset alcoholics. Addictive Behaviors 2006;31:298–308. [PubMed: 15949898]

Doran N, Spring B, McChargue D, Pergadia M, Richmond M. Impulsivity and smoking relapse. Nicotine Tob Res 2004;6:641–647. [PubMed: 15370160]

Duncan, TE.; Duncan, SC.; Strycker, LA.; Li, F.; Alpert, A. An introduction to latent variable growth curve modeling: Concepts, issues, and applications. Lawrence Erlbaum Associates; Mahwah, NJ: 1999.

Epstein LH, Richards JB, Saad FG, Paluch RA, Roemmich JN, Lerman C. Comparison between two measures of delay discounting in smokers. Exp Clin Psychopharmacol 2003;11:131–138. [PubMed: 12755457]

Forbes EE, Shaw DS, Dahl RE. Alterations in reward-related decision making in boys with recent and future depression. Biological Psychiatry 2007;61:633–639. [PubMed: 16920074]

Galera C, Fombonne E, Chastang JF, Bouvard M. Childhood hyperactivity-inattention symptoms and smoking in adolescence. Drug and Alcohol Dependence 2005;78:101–108. [PubMed: 15769563]

Green L, Fry AF, Myerson J. Discounting of delayed rewards: A life-span comparison. Psychological Science 1994;5:33–36.

Grunbaum JA, Kann L, Kinchen SA, Ross JG, Hawkins K, Lowry R, Harris WA, McManus T, Chyen D, Collins J. Youth risk behavior surveillance--United States, 2003. MMWR Surveillance Summaries 2004;53:1–96.

Johnson MW, Bickel WK, Baker F. Moderate drug use and delay discounting: a comparison of heavy, light, and never smokers. Exp Clin Psychopharmacol 2007;15:187–194. [PubMed: 17469942]

Jung T, Wickerama KAS. An introduction to latent class growth analysis and growth mixture modeling. Social and Personality Psychology Compass 2008;1:302–317.

Kellam SG, Anthony JC. Targeting early antecedents to prevent tobacco smoking: findings from an epidemiologically based randomized field trial. Am J Public Health 1998;88:1490–1495. [PubMed: 9772850]

Kellam SG, Brown CH, Poduska JM, Ialongo NS, Wang W, Toyinbo P, Petras H, Ford C, Windham A, Wilcox HC. Effects of a universal classroom behavior management program in first and second grades on young adult behavioral, psychiatric, and social outcomes. Drug and Alcohol Dependence 2008a;95(Suppl 1):S5–S28. [PubMed: 18343607]

Kellam SG, Reid J, Balster RL. Effects of a universal classroom behavior program in first and second grades on young adult problem outcomes. Drug and Alcohol Dependence 2008b;95(Suppl 1):S1–4. [PubMed: 18343051]

Kirby KN. Bidding on the future: Evidence against normative discounting of delayed rewards. J of Exper Psychol 1997;126:54–70.

Kirby KN, Petry NM, Bickel WK. Heroin addicts have higher discount rates for delayed rewards than non-drug-using controls. J Exp Psychol Gen 1999;128:78–87. [PubMed: 10100392]

Kollins SH. Delay discounting is associated with substance use in college students. Addictive Behaviors 2003;28:1167–1173. [PubMed: 12834659]

Kollins SH, McClernon FJ, Fuemmeler BF. Association between smoking and attention-deficit/hyperactivity disorder symptoms in a population-based sample of young adults. Archives of General Psychiatry 2005;62:1142–1147. [PubMed: 16203959]

Kreek MJ, Nielsen DA, Butelman ER, LaForge KS. Genetic influences on impulsivity, risk taking, stress responsivity and vulnerability to drug abuse and addiction. Nat Neurosci 2005;8:1450–1457. [PubMed: 16251987]

Krishnan-Sarin S, Reynolds B, Duhig AM, Smith A, Liss T, McFetridge A, Cavallo DA, Carroll KM, Potenza MN. Behavioral impulsivity predicts treatment outcome in a smoking cessation program for adolescent smokers. Drug and Alcohol Dependence 2007;88:79–82. [PubMed: 17049754]

Lewinsohn PM, Brown RA, Seeley JR, Ramsey SE. Psychosocial correlates of cigarette smoking abstinence, experimentation, persistence and frequency during adolescence. Nicotine Tob Res 2000;2:121–131. [PubMed: 11072450]

Lewinsohn PM, Rohde P, Seeley JR. Major depressive disorder in older adolescents: prevalence, risk factors, and clinical implications. Clinical Psychology Review 1998;18:765–794. [PubMed: 9827321]

Loehlin, JC. Latent Variable Models: An Introduction to Factor, Path, and Structural Equation Analysis. Lawrence Erlbaum Associates, Publishers; Mahwah, NJ: 2004.

Madden, GJ. A behavioral economics primer. In: Bickel, WK.; Vuchinich, RE., editors. Reframing Health Behavior Change with Behavior Economics. Lawrence Erlbaum Associates; Mahwah, NJ: 2000. p. 3-25.

Madden GJ, Petry NM, Badger GJ, Bickel WK. Impulsive and self-control choices in opioid-dependent patients and non-drug-using control participants: drug and monetary rewards. Exp Clin Psychopharmacol 1997;5:256–262. [PubMed: 9260073]

McDonald J, Schleifer L, Richards JB, de Wit H. Effects of THC on behavioral measures of impulsivity in humans. Neuropsychopharmacology 2003;28:1356–1365. [PubMed: 12784123]

Mitchell SH. Measures of impulsivity in cigarette smokers and non-smokers. Psychopharmacology 1999;146:455–464. [PubMed: 10550496]

Monterosso J, Ainslie G. Beyond discounting: possible experimental models of impulse control. Psychopharmacology 1999;146:339–347. [PubMed: 10550485]

Muthén, BO. Mplus technical appendices. Muthén & Muthén; Los Angeles, CA: 19982004.

Muthén, BO. Latent variable analysis: Growth mixture modeling and related techniques for longitudinal data. In: Kaplan, D., editor. Handbook of quantitative methodology for the social sciences. Sage Publications; Nebury Park, CA: 2004. p. 345-368.

Muthen, BO.; Muthen, LK. What is the minimum number of timepoints needed to estimate a growth model?. 2008 [accessed on Oct. 27, 2008]. http://www.statmodel.com/discussion/messages/14/20.html?1218829316

Muthén, BO.; Muthén, LK. Mplus user's guide. Muthen & Muthen; Los Angeles, CA: 2001.

Muthén, LK.; Muthén, BO. Mplus user's guide. Muthén& Muthén; Los Angeles, CA: 19982004.

Muthén, LK.; Muthén, BO. Mplus user's guide. Muthén& Muthén; Los Angeles, CA: 19982007.

Myerson J, Green L. Discounting of delayed rewards, models of individual choice. J Exp Anal Behav 1995;64:263–276. [PubMed: 16812772]

Nylund, KL.; Asparouhov, T.; Muthén, BO. A Monte Carlo simulation study. University of California; Los Angeles: 2006. Deciding on the number of classes in latent class analysis and growth mixture modeling.

Odgers CL, Caspi A, Nagin DS, Piquero AR, Slutske WS, Milne BJ, Dickson N, Poulton R, Moffit TE. Is It Important to Prevent Early Exposure to Drugs and Alcohol Among Adolescents? Psychol Sci. in press

Odum AL, Madden GJ, Badger GJ, Bickel WK. Needle sharing in opioid-dependent outpatients: psychological processes underlying risk. Drug and Alcohol Dependence 2000;60:259–266. [PubMed: 11053760]

Ohmura Y, Takahashi T, Kitamura N, Wehr P. Three-month stability of delay and probability discounting measures. Exp Clin Psychopharmacol 2006;14:318–328. [PubMed: 16893275]

Perry JL, Larson EB, German JP, Madden GJ, Carroll ME. Impulsivity (delay discounting) as a predictor of acquisition of IV cocaine self-administration in female rats. Psychopharmacology 2005;178:193–201. [PubMed: 15338104]

Radloff LS. The CES-D scale: A self-report depression scale for research in the general population. Applied Psychological Measurement 1977;1:385–401.

Radloff LS. The use of the center for epidemiologic studies depression scale in adolescents and young adults. Journal of Youth and Adolescence 1991;20:149–166.

Reynolds B. Do high rates of cigarette consumption increase delay discounting? A cross-sectional comparison of adolescent smokers and young-adult smokers and nonsmokers. Behavioural Processes 2004;67:545–549. [PubMed: 15519004]

Richards JB, Sabol KE, de Wit H. Effects of methamphetamine on the adjusting amount procedure, a model of impulsive behavior in rats. Psychopharmacology 1999a;146:432–439. [PubMed: 10550493]

Richards JB, Zhang L, Mitchell SH, de Wit H. Delay or probability discounting in a model of impulsive behavior: effect of alcohol. J Exp Anal Behav 1999b;71:121–143. [PubMed: 10220927]

Roberts RE, Andrews JA, Lewinsohn PM, Hops H. Assessment of depression in adolescents using the center for epidemiologic studies depression scale. Psychological Assessment 1990;2:122–128.

Rodriguez D, Romer D, Audrain-McGovern J. Beliefs about the risks of smoking mediate the relationship between exposure to smoking and smoking. Psychosomatic Medicine 2007;69:106–113. [PubMed: 17244853]

Rodriguez D, Tercyak KP, Audrain-McGovern J. Effects of inattention and hyperactivity/impulsivity symptoms on development of nicotine dependence from mid adolescence to young adulthood. J Pediatr Psychol 2008;33:563–575. [PubMed: 17956929]

Roesch MR, Takahashi Y, Gugsa N, Bissonette GB, Schoenbaum G. Previous cocaine exposure makes rats hypersensitive to both delay and reward magnitude. J Neurosci 2007;27:245–250. [PubMed: 17202492]

Spring B, Pingitore R, McChargue DE. Reward value of cigarette smoking for comparably heavy smoking schizophrenic, depressed, and nonpatient smokers. The American Journal of Psychiatry 2003;160:316–322. [PubMed: 12562579]

Storr CL, Ialongo NS, Kellam SG, Anthony JC. A randomized controlled trial of two primary school intervention strategies to prevent early onset tobacco smoking. Drug and Alcohol Dependence 2002;66:51–60. [PubMed: 11850136]

USDHHS. Preventing tobacco use among young people. A report of the Surgeon General. Executive summary. MMWR Recomm Rep 1994;43:1–10.

Velicer WF, Prochaska JO, Rossi JS, Snow MG. Assessing outcome in smoking cessation studies. Psychological Bulletin 1992;111:23–41. [PubMed: 1539088]

Wade TR, de Wit H, Richards JB. Effects of dopaminergic drugs on delayed reward as a measure of impulsive behavior in rats. Psychopharmacology 2000;150:90–101. [PubMed: 10867981]

Wills TA, Cleary SD. The validity of self-reports of smoking: analyses by race/ethnicity in a school sample of urban adolescents. Am J Public Health 1997;87:56–61. [PubMed: 9065227]

Wills TA, Vaccaro D, McNamara G. Novelty seeking, risk taking, and related constructs as predictors of adolescent substance use: an application of Cloninger's theory. J Subst Abuse 1994;6:1–20. [PubMed: 8081104]

Wills TA, Windle M, Cleary SD. Temperament and novelty seeking in adolescent substance use: convergence of dimensions of temperament with constructs from Cloninger's theory. J Pers Soc Psychol 1998;74:387–406. [PubMed: 9491584]

Windle M, Windle RC. Depressive symptoms and cigarette smoking among middle adolescents: prospective associations and intrapersonal and interpersonal influences. Journal of Consulting and Clinical Psychology 2001;69:215–226. [PubMed: 11393599]

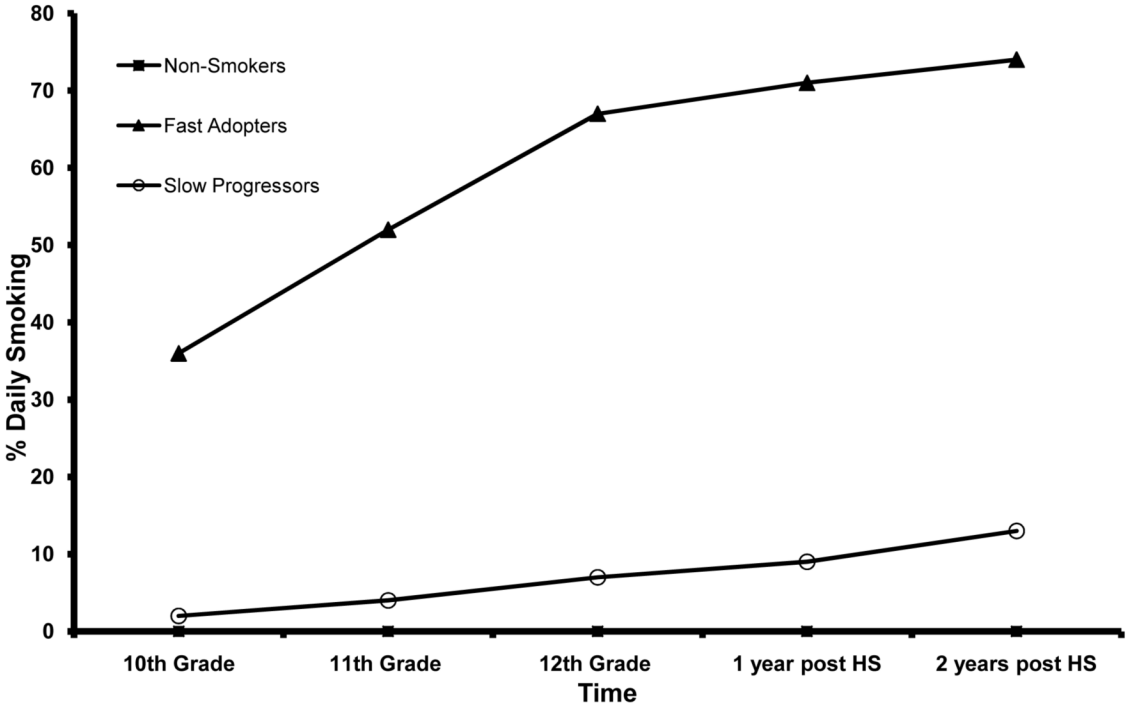

Figure 1.

The etiological role of delay discounting in smoking acquisition.

Note: Associated processes latent growth curve model with standardized path coefficients for significant model paths only, and factor loadings representing 10th grade baseline (0), and the first (3) and second (4) years post high school. DD = delay discounting; MJ = marijuana; DS = depression symptoms; HS = high school. *p<.05, **p<.0001

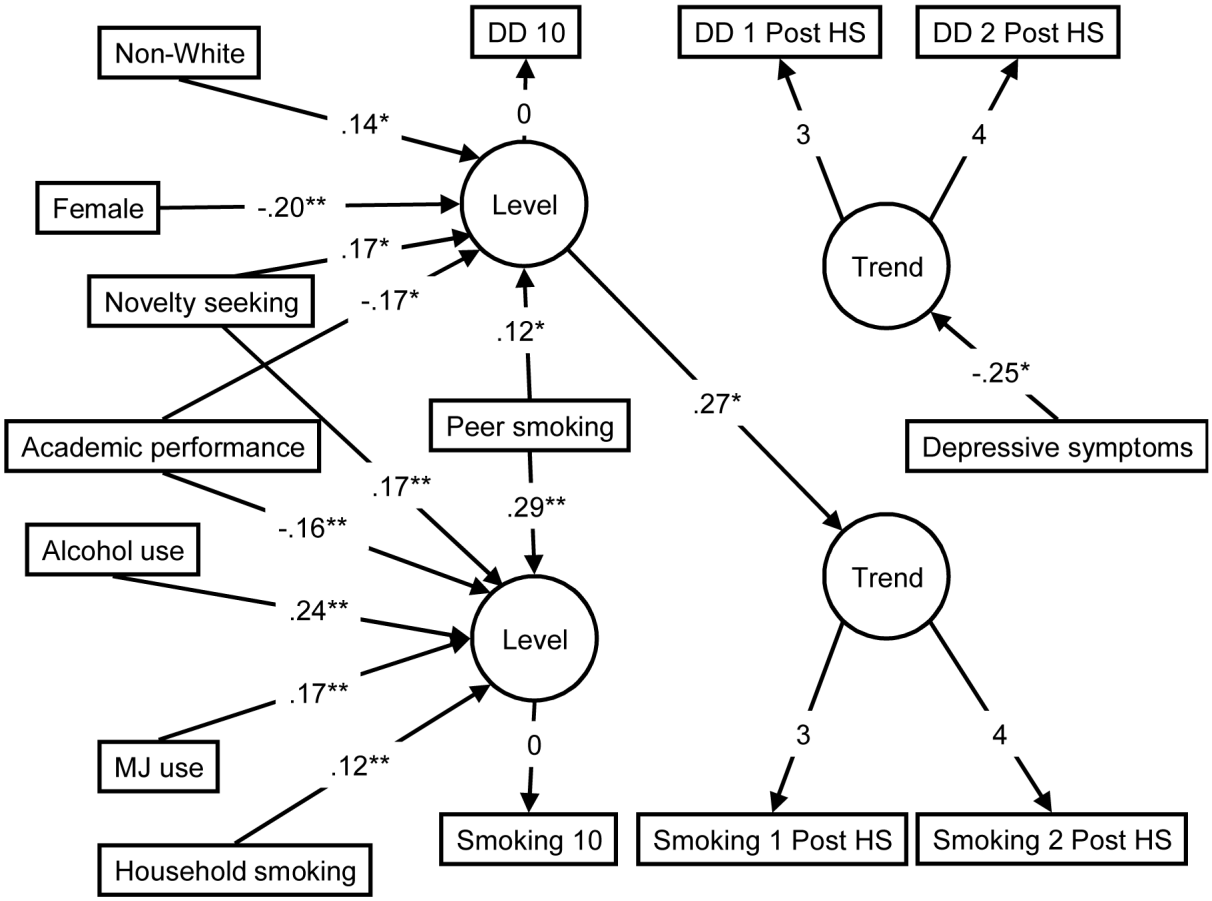

Figure 2.
Smoking trajectories from mid-adolescence to young adulthood.

Table 1

Nonstandardized path coefficients, standard errors, and test statistics with probability values for all model regressions

	Regular Smoking							
	Level				Trend			
	β	SE	z	*p*-value	β	SE	z	*p*-value
Delay discounting level	-	-	-	-	0.08	0.04	2.16	0.03
Delay discounting trend	-	-	-	-	-0.24	0.38	-0.64	0.53
Female	-0.09	0.09	-1.12	0.27	-0.00	0.04	-0.06	0.95
Non-White	-0.01	0.09	-0.15	0.88	-0.02	0.04	-0.43	0.67
Academic performance	-0.37	0.08	-4.63	0.00	0.01	0.04	0.39	0.70
Depression Symptoms	0.01	0.00	1.84	0.07	0.00	0.00	0.17	0.87
Novelty seeking	0.06	0.01	4.62	0.00	-0.00	0.01	-0.14	0.89
Attention	-0.02	0.01	-1.17	0.24	-0.00	0.01	-0.09	0.93
Hyperactivity	0.00	0.01	0.30	0.77	-0.00	0.01	-0.40	0.69
Alcohol use	0.34	0.04	5.62	0.00	-0.00	0.02	-0.09	0.93
Marijuana use	0.31	0.07	9.01	0.00	-0.02	0.03	-0.56	0.57
Peer smoking	0.17	0.02	9.84	0.00	-0.01	0.01	-1.01	0.31
Household smoking	0.36	0.09	3.86	0.00	-0.07	0.04	-1.62	0.11

	Delay Discounting							
	Level				Trend			
	β	SE	z	*p*-value	β	SE	z	*p*-value
Smoking level	-	-	-	-	0.00	0.02	0.01	0.99
Female	-0.34	0.10	-3.56	0.00	0.05	0.03	1.67	0.10
Non-white	0.25	0.10	2.54	0.01	0.04	0.03	1.18	0.24
Academic performance	-0.25	0.09	-2.73	0.01	-0.03	0.03	-1.16	0.25
Depression Symptoms	0.01	0.00	1.83	0.07	0.00	0.00	-2.24	0.03
Novelty seeking	0.04	0.01	2.90	0.00	-0.01	0.00	-1.76	0.08
Attention	-0.02	0.02	-1.39	0.17	-0.01	0.01	-1.01	0.31
Hyperactivity	0.01	0.01	0.30	0.77	0.01	0.01	1.05	0.29
Alcohol use	0.01	0.02	0.89	0.37	-0.00	0.02	-0.15	0.88

	Delay Discounting							
	Level				Trend			
	β	SE	z	*p*-value	β	SE	z	*p*-value
Marijuana use	0.08	0.09	0.90	0.37	0.01	0.03	0.42	0.68
Peer smoking	0.05	0.02	1.97	0.05	-0.01	0.01	-1.86	0.06
Household smoking	0.07	0.12	0.61	0.55	0.04	0.04	1.09	0.28

Note. Level factors represent the baseline level for a given growth process; trend factors represent the rate of change in a given process for a unit change in time. However, as smoking is an ordered categorical variable, the Beta is the log odds change for a unit increase in a predictor variable. Female (1=Male, 2=Female); Non-white (0=white, 1=non-white); Alcohol use (30-day alcohol use, 1=0 days to 7=20 or more days); Marijuana use (30-day marijuana use, 1=0 times to 6=40 or more times); Household smoking (1=at least one household member smokes, 0=nobody living at home smokes); Academic performance (1=Mostly D's and F's to 4=Mostly A's).

Table 2

Growth Mixture Model: Optimization of Trajectory Number

Model	Latent Classes	Covariates	Free parameters	Loglikelihood	BIC	Entropy	BLRT (parameter difference), *p* - value
1	2	No	8	-2926.37	5908.52	.90	1122.09(3), *p* <.0001
2	3	No	11	-2844.65	5766.01	.78	163.440(3), *p* <.0001
3	4	No	14	-2817.08	5731.79	.73	55.137(3), *p* <.0001
4	5	No	17	-2802.60	5723.75	.76	28.96(3), *p* =.0312
5	6	No	20	-2790.85	5721.17	.77	23508(3), *p* =.19
6	4	Yes	50	-2230.96	4802.54	.78	267.98 (19), *p* =.67
7	3	Yes	35	-2276.35	4791.14	.77	213.18 (15), *p* <.0001

Note. BIC, Bayesian information criterion ($-2logL + r\ ln\ n$), where L is the model's maximum likelihood value, n is the sample size, and r is the number of free model parameters; BLRT, Bootstrap Likelihood Ratio Test for k (H0) versus k-1 classes; Entropy ($E_K=1-(p_{ik}log(p_{ik}))/nlog(K)$), where p_{ik} is the conditional probability of individual i in class K, is a measure of classification quality, with values closer to one representing better classification.

Drug Alcohol Depend. Author manuscript; available in PMC 2010 August 1.

Table 3

Characteristics of trajectory class membership

Characteristic	Nonsmokers compared to slow smoking progressors			Nonsmokers compared to fast smoking adopters			Slow smoking progressors compared to fast smoking adopters		
		95% CI			95% CI			95% CI	
	OR	Low	High	OR	Low	High	OR	Low	High
Female	1.26	0.73	2.19	1.44	0.77	2.69	1.14	0.62	2.07
Non-white	1.05	0.59	1.87	1.46	0.71	2.97	1.39	0.68	2.85
Delay discounting	0.74	0.57	0.96	0.69	0.51	0.94	0.95	0.78	1.17
Novelty seeking	0.73	0.55	0.96	0.65	0.46	0.93	0.97	0.89	1.06
ADHD-Attention	1.02	0.94	1.10	1.00	0.91	1.09	0.98	0.91	1.06
ADHD-Hyperactivity	1.01	0.94	1.09	1.07	0.97	1.17	1.06	0.97	1.15
Depression symptoms	0.99	0.96	1.02	0.96	0.93	1.00	0.98	0.95	1.01
Academic performance	1.73	1.00	3.00	2.71	1.49	4.92	1.56	0.87	2.82
Alcohol use	0.31	0.18	0.54	0.33	0.18	0.59	1.04	0.79	1.37
Marijuana use	0.19	0.01	2.81	0.11	0.01	1.55	0.59	0.39	0.89
Peer smoking	0.81	0.70	0.95	0.63	0.53	0.75	0.78	0.67	0.89
Household smoking	1.59	0.81	3.13	0.93	0.45	1.96	0.59	0.30	1.15

Note. The results are from a multinomial logistic regression analysis. Confidence intervals containing 1.00 are not significant, $p < .05$. For delay discounting, a unit is one standard deviation (SD=1.41). Female (1=Male, 2=Female); Non-white (0=white, 1=non-white); Alcohol use (30-day alcohol use, 1=0 days to 7=20 or more days); Marijuana use (30-day marijuana use, 1=0 times to 6=40 or more times); Household smoking (1=at least one household member smokes, 0=nobody living at home smokes); Academic performance (1=Mostly D's and F's to 4=Mostly A's).