

Longitudinal Associations among Adolescent Socioeconomic Status, Delay
Discounting, and Substance Use

Kristin M. Peviani

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Jungmeen Kim-Spoon, Chair
Warren K. Bickel
Brooks King-Casas

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Abstract

Adolescence is a period of heightened risk for substance use and heightened vulnerability to substance exposure. Yet, little is known about how socioeconomic status (SES) influences adolescent decision making and behavior across development to add to these risks. This prospective longitudinal study used latent growth curve modeling (GCM) to examine the contributions of SES on adolescent delay discounting and substance use in a sample of 167 adolescents (52% male). Confirmatory factor analysis (CFA) was used to compute SES factor scores across three waves using a composite of parent and spouse education years and combined annual household income. Adolescent delay discounting and substance use were measured annually across three waves. The main goal of this study is to examine how SES may explain individual differences in growth trajectories of delay discounting and substance use. We used parallel process growth curve modeling with SES as a time-varying and time-invariant covariate to examine the associations between adolescent SES, delay discounting, and substance use onset as well as frequency. These results reveal that delay discounting exhibits a declining linear trend across adolescent development whereas cigarette, alcohol, marijuana, and polysubstance use exhibit increasing linear trends across adolescent development. Furthermore, low SES (as a time-invariant covariate) may lead to earlier onset adolescent alcohol and polysubstance use by way of heightened levels of delay discounting. These findings suggest that delay discounting interventions may be a promising avenue for reducing

socioeconomic disparities in early onset alcohol and polysubstance use, while delay discounting development is still underway.

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General Audience Abstract

Adolescence is a period of heightened risk for substance use and heightened vulnerability to the effects of substances. Yet, little is known about how socioeconomic status (SES) influences adolescent decision making and behavior to add to these risks. This study used latent growth curve modeling (GCM) to examine the role of SES on adolescent decision making and substance use in a sample of 167 adolescents (52% male). Confirmatory factor analysis (CFA) was used to compute SES factor scores across three time points using an average of parent and spouse education years and income. Adolescent delay discounting and substance use were measured annually across three time points. The main goal of this study is to examine how SES may explain individual differences in delay discounting and substance use across adolescence. We used parallel process growth curve modeling with SES as a time-varying and time-invariant covariate to examine the links between adolescent SES, delay discounting, and substance use age of onset and frequency. These results reveal that delay discounting shows linear decreases in growth across adolescence whereas cigarette, alcohol, marijuana, and polysubstance use show increasing linear growth across adolescence. Additionally, low SES may lead to earlier onset adolescent alcohol and polysubstance use by way of heightened levels of delay discounting. These findings suggest that delay discounting interventions may help reduce socioeconomic differences in early onset alcohol and polysubstance use, while delay discounting development is still in progress.

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Introduction

Adolescence is a period of heightened risk for substance use and heightened vulnerability for substance exposure. Low socioeconomic status (SES) may alter brain development associated with reward sensitivity, stress response, and executive functioning to heighten adolescent risky decision making associated with substance use (Bickel, Moody, Quisenberry, Ramey, & Sheffer, 2014; Gordon, 2002; Mani, Mullainathan, Shafir, & Zhao, 2013). Specifically, low SES may impose cognitive strain and heighten perceived resource scarcity to restrict temporal perspective and lead to impaired decision making (Bickel et al., 2014). These impairments may influence substance initiation and escalation by way of delay discounting, which is the preference for relatively smaller immediate gains over larger gains with a temporal delay, and is a strong predictor of substance use (Bickel & Marsch, 2001; Fernie et al., 2013; Kim-Spoon, McCullough, Bickel, Farley, & Longo, 2014). Furthermore, early adolescent substance initiation may lead to neuroadaptations that confer greater risk for developing later substance use disorders (Hanson, Medina, Padula, Tapert, & Brown, 2011). Neuroeconomic models may elucidate how SES influences adolescent suboptimal decision making associated with substance initiation and escalated use, which is vital for reducing socioeconomic substance use disparities (Bickel et al., 2014).

According to the competing neurobehavioral decision systems (CNDS) theory, two distinct systems work in tandem to influence decision making (Bickel et al., 2007). The impulsive system, which includes the limbic and paralimbic systems (including the amygdala, basal ganglia, nucleus accumbens, dorsal striatum, hippocampus, and cingulate cortex), is associated with hedonic response, while the executive system

(including the prefrontal cortex), assists with future planning and abstract reasoning (McClure, Laibson, Loewenstein, & Cohen, 2004). The theory proposes that dysregulation of these competing systems can lead to pathological reinforcement and subsequent substance use disorders, particularly when delay discounting is heightened and harmful rewards (e.g., foods, substances) are overvalued (Bickel et al., 2017). These competing systems develop asynchronously during adolescence; the impulsive system develops first while executive development is protracted and may continue through the mid-twenties (Somerville & Casey, 2010).

Development of Delay Discounting during Adolescence

Delay discounting develops across adolescence, but there is a dearth of longitudinal research examining delay discounting developmental trends. Findings from the limited research examining delay discounting development have been somewhat inconsistent, leading some researchers to suggest that enthusiasm for the predictive utility of delay discounting as an endophenotype for adolescent substance use should be tempered until findings from non-clinical samples are better understood (Isen, Sparks, & Iacono, 2014). Results from a cross sectional study of delay discounting in adolescent and young adult participants demonstrated that delay discounting progresses in a steep, ascending, and linear fashion from early to middle adolescence (from ages 10 to 15) and relatively stabilizes thereafter (from ages 16 to 30 (Steinberg et al., 2009). Results from another cross-sectional study found especially low levels of delay discounting between ages 15 and 16 in a sample consisting of adolescents and young adults ranging in age from 12 to 27 (Water, Cillessen, & Scheres, 2014). A longitudinal study using a community sample of adolescents implemented growth curve modeling to examine

associations between delay discounting and cigarette smoking found that delay discounting was relatively stable between the ages of 15 and 20 (Audrain-McGovern et al., 2009). However, this study measured delay discounting only once during adolescence, during the 10th grade, with two follow-ups during young adulthood (approximately 18-19 and 19-20 years of age), thus developmental trajectories of delay discounting during adolescence could not be examined. Regardless, the results indicated that delay discounting predicted trends in smoking progression, whereas smoking did not significantly influence trends in delay discounting, adding support to the perspective that increases in risk-taking during adolescence may be reflective of limited executive regulation over the reward sensitive impulsive system (Casey, Jones, & Somerville, 2011; Steinberg et al., 2009).

The lack of longitudinal studies on delay discounting development during adolescence warrants additional research incorporating consistent and consecutive measurement of delay discounting beginning in early adolescence, to gain a clearer understanding of the developmental trajectories of delay discounting as it develops over time. Additionally, although the study by Audrain-McGovern et al. (2009) adds important information to the literature on the longitudinal associations between delay discounting and smoking, examination of the longitudinal associations between delay discounting and a broader range of commonly used substances across adolescence is needed. Importantly, although prior work has implicated the significant impact of SES on delay discounting and substance use (Bickel et al., 2014), little is known about the processes through which SES may be related to delay discounting and substance use development. We propose the first study testing whether SES is related to developmental trajectories of delay

discounting and in turn developmental trajectories of substance use during adolescence, a developmental window in which brain development is known to be particularly sensitive to environmental contexts.

SES and Adolescent Substance Use

Low SES has been associated with deficits in natural, physical, or social resources which may work independently or collectively, directly or indirectly, to influence adolescent substance use perceptions, responses, and behaviors. Material deficits associated with low SES include greater exposure to toxins, residential crowding, limited access to green and active spaces, limited access to healthy foods, and poor housing quality, whereas nonmaterial deficits associated with low SES include greater residential instability, greater neighborhood substance use prevalence, poor social monitoring, greater environmental stress, higher levels of unemployment, and ease of access to substances, all of which pose risks for adolescent substance use (Evans & Kantrowitz, 2002; Powell, Slater, Chaloupka, & Harper, 2006; Tucker, Pollard, de la Haye, Kennedy, & Green, 2013).

In a latent growth model of SES on adolescent substance use, lower SES (measured by parental education) predicted increased adolescent cigarette smoking across three time points when adolescents were between the ages of 12 and 18, and the effects were especially prominent in adolescents from low SES residing in more affluent communities (Mathur, Erickson, Stigler, Forster, & Finnegan Jr, 2013). Similar results have been found using annual household income as a time-varying covariate to predict adolescent substance use with growth curve modeling (Cambron, Kosterman, Catalano, Guttmanova, & Hawkins, 2017). Specifically, higher family income was associated with

lower adolescent smoking (but not alcohol use) after controlling for average growth in smoking, and greater neighborhood disadvantage predicted higher levels of adolescent smoking and alcohol use beyond average growth in smoking and alcohol use across four time points, from the 5th to the 9th grade. These findings demonstrate socioeconomic risks, particularly for adolescent smoking, compared to other commonly used substances.

Low SES has also been associated with deficits in opportunities for extracurricular involvement which may place adolescents at a greater risk for substance use (Leventhal et al., 2015). Participation costs and limited access to alternative reinforcement activities may prohibit adolescents from low SES from acquiring the positive, socially rewarding benefits from engaging in extracurricular activities; experiences which might otherwise help buffer against substance use (McCabe, Modecki, & Barber, 2016; Powell et al., 2006). Thus, adolescents growing up in low SES may experience greater barriers to material and non-material resources which may lead to compensatory reward-seeking and substance use. Additionally, adolescents may experience greater exposure to material and non-material risks, which may accumulate to contribute to risks for substance initiation and escalation.

Individual Differences in SES and Adolescent Delay Discounting

Adolescence is a time when contextual sensitivity and reward seeking typically peaks which may poise adolescents to be especially susceptible to their socioeconomic contexts (Belsky & Shalev, 2016; O'Brien, Albert, Chein, & Steinberg, 2011; Somerville & Casey, 2010; Spear, 2011). Adolescents from low SES may experience greater contextual stress disproportionate to their higher SES peers, the effects of which may alter neurodevelopment associated with executive functioning and reward processing

(Birn, Roeber, & Pollak, 2017; Evans & Kantrowitz, 2002; Rabl et al., 2014). Thus, SES may influence adolescent delay discounting through the executive and impulsive systems implicated by CNDS theory: 1) through hedonic reward sensitivity, and 2) through constraints to the executive system.

Socioeconomic adversity may lead to stress effects that accumulate over time to contribute to variations in reward responsivity, which may influence delay discounting preferences. For example, one study found that adolescents exhibiting higher levels of delay discounting also exhibited lower deactivation in response to rewards in the amygdala, insula, vmPFC, and hippocampus and diminished activation in the executive system (Stanger et al., 2013). Results from another study demonstrate links between SES and reward anticipation. Specifically, cumulative years of receipt of aid (from age five to sixteen) predicted heightened responsivity during monetary reward anticipation, but not receipt, at age 16, which may be a risk for substance use (Romens et al., 2015). It is also possible, however, that these effects may be reversible, as indicated by prefrontal functioning plasticity recovery from stress effects (Liston, McEwen, & Casey, 2009). Thus, delay discounting preferences may be malleable and respond to changing socioeconomic conditions.

There are a variety of ways SES may impact cognitive functioning and lead to individual differences in the executive system. Environments that are unpredictable or resource scarce may preoccupy cognitive resources to lead to unfavorable decision making (Mani et al., 2013). Specifically, cognitive strain may impair working memory performance. Socioeconomic adversity may also lead to heightened stress reactivity and espouse greater delay discounting via increasing present orientation (Frankenhuis,

Panchanathan, & Nettle, 2016; Leonard, Mackey, Finn, & Gabrieli, 2015; Sheridan, Sarsour, Jutte, D'Esposito, & Boyce, 2012).

Adolescent Delay Discounting and Substance Use

Substance use disorders are posited to develop progressively from impulsivity to compulsivity (Koob & Le Moal, 2005). The impulsive stage is believed to be motivated by positive reinforcement derived from the pleasurable effects of the substance generated by the brain's reward circuitry, peer approval, or temporary relief from negative emotional states whereas the compulsive stage may be influenced by decreased hedonic response to rewards caused by substance use or relief from the negative effects of substance withdrawal (Koob & Le Moal, 2001).

Delay discounting may be an etiological predictor of, and an endophenotype for, substance use disorders (MacKillop, 2013). Empirical research on delay discounting and substance use suggests that delay discounting influences substance use, but not the other way around (Audrain-McGovern et al., 2009). During adolescence, delay discounting has been associated with frequency of cigarette, alcohol, and marijuana use (Audrain-McGovern et al., 2009; Kim-Spoon, McCullough, Bickel, Farley, & Longo, 2014). Trends in delay discounting increases or decreases may be important indicators of risk for adolescent substance use. For example, one study, although it did not directly examine delay discounting, showed that slower gradual declining rates of impulsivity across adolescence were associated with greater cigarette, alcohol, and marijuana use (Quinn & Harden, 2013). Furthermore, delay discounting preferences may be reversible with changes in substance use; a study of delay discounting among cigarette smokers, former

cigarette smokers, and non-smokers found no significant differences in delay discounting between former smokers and non-smokers (Bickel, Odum, & Madden, 1999).

The Present Study

Given adolescent sensitivity to context and reward as well as neurobehavioral patterns of development associated with SES, it is critical to clarify how SES contributes to individual differences in delay discounting and substance use to identify ways to alter developmental trajectories that lead to serious substance use problems and disorders. Furthermore, because much of the research on delay discounting and substance use has utilized clinical samples with substance disorders (Amlung, Vedelago, Acker, Balodis, & MacKillop, 2017; Karakula et al., 2016; Stanger et al., 2012), less is known regarding the role of delay discounting in more normative development of substance use behaviors in a community sample of adolescents. Statistical modeling capable of examining interindividual differences in intraindividual changes in delay discounting may elucidate transactional processes between the development of delay discounting and the development of substance use behaviors.

We used parallel process latent growth curve modeling (GCM) to examine the potential mechanisms linking SES, delay discounting, and substance use longitudinally across adolescence. Importantly, this study fills in gaps in the literature on the longitudinal effects of SES on delay discounting and substance use, as they develop during adolescence. The main goal of this study is to examine how SES as a context may explain the associations between individual differences in growth trajectories of delay discounting and individual differences in growth trajectories of substance use. By testing these associations of substance specific (cigarette, alcohol, and marijuana separately)

onset and use frequency as well as polysubstance onset and use frequency, these findings may reveal substance use trajectories related to initiation as well as progression to more frequent use. Specifically, the present study aims to 1) identify the role of SES in adolescent delay discounting development, 2) test delay discounting as a mediating process that explains the effects of SES on adolescent substance use initiation and progression, 3) describe how SES is associated with individual differences in delay discounting substance use behaviors at each time; and 4) explore substance specific risks associated with changing SES and delay discounting development.

Hypotheses

Hypothesis 1: As a time-invariant covariate, lower levels of SES at Time 1 will predict high intercept and increasing slope of delay discounting as well as high intercept and increasing slope of substance use from Time 1 to Time 3 (see Figure 2).

Hypothesis 2: Delay discounting growth factors will mediate the link between SES and adolescent substance use such that low SES is related to high intercept and increasing slope of delay discounting from Time 1 to Time 3. In turn, high intercept of delay discounting is related to high intercept and increasing slope of substance use from Time 1 to Time 3, and increasing slope of delay discounting from Time 1 to Time 3 is related to increasing slope of substance use from Time 1 to Time 3 (see Figure 2).

Hypothesis 3: As time-varying covariates, at each time point, lower SES will be associated with higher levels of delay discounting and higher levels of substance use (see Figure 2).

Method

Participants

The final sample used for the current analyses was drawn from an on-going longitudinal study and included 167 adolescents (52% male) and their parents. A total of 157 adolescents were recruited for their participation in the study at Time 1 and an additional 10 adolescents were recruited for participation at Time 2. Over the course of the study, 19 adolescents withdrew participation for reasons such as: lost contact ($n = 8$), declined participation ($n = 8$), ineligibility for neuroimaging tasks ($n = 1$), moved away ($n = 1$) and extenuating circumstances ($n = 1$). Adolescents were excluded from participation for such reasons as claustrophobia, history of head injury resulting in loss of consciousness for more than 10 minutes, orthodontia impairing image acquisition, and contraindications to magnetic resonance imaging. Despite partially missing data from adolescents who did not participate at all time points, the final sample for our analyses included 167 adolescents by using Full Information Maximum Likelihood (FIML) that allows data from all individuals to be included regardless of their pattern of missing data.

Adolescents were between the ages of 13 to 14 at Time 1 ($M = 13.51$, $SD = 0.50$) and participated annually in the longitudinal study over three years ($M = 14.52$, $SD = 0.50$ at Time 2 and $M = 15.55$, $SD = 0.53$ at Time 3). Adolescent races were predominantly Caucasian (79%), African-American (11%), and Other (10%). Median annual household income was between \$35,000 and \$49,999 at Time 1 and \$50,000 and \$74,999 at Times 2 and 3.

Although the sample was not entirely substance naïve at the study outset, adolescent substance use was relatively consistent with 8th grade national averages reflected in the Monitoring the Future survey of having tried cigarettes (14%), alcohol (27%), and marijuana (16%) from 2014 (the first measurement year), albeit low for

marijuana use (Miech et al., 2017). Specifically, in the present sample, 26% of adolescents initiated cigarette, alcohol, or marijuana use prior to Time 1; 15% initiated cigarette use, 19% initiated alcohol use, and 8% initiated marijuana use.

Procedures

Participants were recruited through word of mouth, flyers, and recruitment letters for an ongoing study of adolescent health. Adolescents provided written assent and their parents provided consent in compliance with university institutional review board approved protocol. Data collection took place across three time points, approximately one year apart. Parents and adolescents were interviewed separately by trained researchers at the university offices. Logistic regression was used to test whether adolescents who withdrew their participation and those who continued throughout the study differed on demographic variables such as age, income, race, and sex. General linear modeling was used to test for sex, age, and ethnicity covariates for predicting adolescent substance use in the model.

Measures

SES. Parents provided self-reported demographic information as well as the demographic information of their spouses (when applicable) across all three waves of data collection. The demographic questionnaire included questions about income, education, family size, and receipt of aid (see Appendix A). A factor score consisting of total household income and mean parent spouse education was selected for testing in the model due to extant literature identifying these as prominent indicators of SES during adolescence.

Total household income. Total annual household income before taxes was reported using responses ranging from 1 (*none or \$0 per month*), to 15 (*\$200,000 or more or \$16,667 or more per month*). Higher scores were indicative of higher SES.

Parental Education. Primary caregivers reported the highest number of years of education they completed as well as the highest number of years of education completed by their spouses (with a capped score of 17) at each time point. Composite parental education scores were calculated using an average of the years of education completed by the primary caregiver and spouse (when applicable). Higher scores are indicative of higher SES.

Delay Discounting. Reward-dependent decision making was assessed for adolescents using a computerized delay discounting task (Johnson & Bickel, 2002) across all three time points. Adolescents were given a series of hypothetical monetary decisions in which they made intertemporal choices between an immediate monetary reward and a larger monetary reward with a delay. The amount for the delay was held constant at \$100 across four delays. Choices were presented using the following delays: one day, one week, one month, and one year. Individual discounting functions were calculated using hyperbolic k values (Mazur, 1987) as an index for discounting rate where I is the indifference point, D is the time to delay, and k is the free parameter where discounting rates decline as delays increase in the formula:

$$I = \frac{1}{1 + kD}$$

We used the Johnson and Bickel (2008) algorithm for identifying and excluding nonsystematic discounting from the analysis for violating the assumption of monotonic decreases in discounting function. Nonsystematic data were identified for either of two

reasons: (1) if following the first delay, an indifference point was greater than the previous indifference point by 20% of the larger, later reward, and (2) if the last indifference point (calculated at one year) was not any different from the first indifference point (calculated at one day). Despite the hypothetical nature of the decisions, consistently similar patterns of discounting have been observed between actual and hypothetical rewards, bolstering evidence of external task validity, despite the hypothetical nature of the task (Johnson & Bickel, 2002).

Adolescent Substance Use. Adolescent cigarette, alcohol, and marijuana use questionnaires were administered via Survey Monkey (see Appendix).

Substance Use Frequency. Adolescents were asked to report their frequency of cigarette, alcohol, and marijuana use across all three time points using a substance use index adapted from the Youth Risk Behavior Survey (Kann et al.). This index consists of three items such as, “Which is the most true for you about using alcohol?”, using a 6-point response scale ranging from 1 (*never used*) to 6 (*usually use every day*). A polysubstance use composite score was computed at each time point using an average of adolescent responses across all three items. Higher scores are indicative of greater substance use. Scale reliability was acceptable with internal consistency of $\alpha = .75$ at Time 1, $\alpha = .69$ at Time 2, and $\alpha = .61$ at Time 3.

Adolescent Substance Use Onset. Adolescents were asked to report their age of onset for cigarette, alcohol, and marijuana use at Time 3 using items adapted from the Youth Risk Behavior Survey. This index consists of three items such as, “How old were you when you smoked a whole cigarette for the first time?”, using an 11-point response scale ranging from 1 (*never*) to 11 (*17 years old or older*). Ages were capped at the

maximum age represented by adolescents at each time point and recoded such that higher scores were indicative of earlier age of initiation. Cigarette, alcohol, and marijuana item responses separately indicated age of substance specific onset. A polysubstance use onset composite score was computed using an average of responses across all three items at Time 3.

Statistical Analyses

Correlations and descriptive statistics (see Table 1) for all study variables were analyzed using SPSS version 24.0, prior to conducting CFA and modeling the GCM in *Mplus* version 8 (Muthén & Muthén, 1998-2017).

Structural equation modeling (SEM) was conducted to test the univariate, unconditional, and conditional GCMs (Preacher & Hayes, 2008). To determine whether FIML was acceptable, patterns of missingness on all study variables were examined using Little's MCAR test (Little, 1988). The resulting pattern resembled a missing completely at random pattern (Little's MCAR test on all variables in this study: $\chi^2 = 59.17$, $df = 67$, $p = .74$), thus FIML was used as it is superior to listwise deletion, pairwise deletion, and similar response pattern imputation by retaining statistical power and producing unbiased estimates and robust standard errors (Enders & Bandalos, 2001). GCM estimates were conducted using maximum likelihood including robust standard errors (MLR) and model fit indices were evaluated using the Hu and Bentler (1999) criteria for Root Mean Square Error of Approximation (RMSEA) values of less than (or near) .08 and Comparative Fit Index (CFI) values greater than or near .9.

Parallel process GCM was used to identify changes in delay discounting and substance use over time with the benefit of incorporating time-varying covariates to

identify time-specific associations among SES, delay discounting, and substance use, above and beyond average growth (Curran & Hussong, 2003). Parallel process GCM also allows estimating various growth trajectories of the mediator and outcome by fixing the loadings on the growth rate to be zero (no-growth), linear (linear increase or decrease), or latent by allowing the loadings to estimate freely (Cheong, MacKinnon, & Khoo, 2003). Absolute fit indices were used to assess model fits. Closeness of fit measures including the comparative fit index (CFI), root mean square error of approximation (RMSEA) and chi-square goodness of fit tests were used to assess the best fitting univariate models. Given that there was no reason to expect any pattern (increases or decreases) of SES growth, the effects of Time 1 SES on the growth trajectories of substance use from Time 1 through Time 3 mediated through the growth trajectories of delay discounting from Time 1 through Time 3 were tested (see Figure 2). Additionally, Time 1, Time 2, and Time 3 SES scores were included as time-varying covariates to test whether SES was associated with delay discounting and substance use at each time point, above and beyond average delay discounting and substance use growth trajectories (see Figure 3).

Power

Power estimation started with hypothetical effect sizes representing bivariate correlations of .10, .30 and .50 or values for $f^2 = r^2/[1-r^2]$ as .02, .15 and .35 (Cohen, 1988), using sample sizes of 167 in G-power. Statistical power for each effect size—even for the smallest estimated effects—was sufficient and approached asymptote ($> .995$). Power for the growth curve model was calculated using the sample size recommendation of 5:1 with the assumption of normally distributed variables (Bentler & Chou, 1987). The mediation parallel process GCM with time-invariant covariate in Figure 2 estimates 24

parameters, requiring a minimum sample size of 120. The parallel process GCM model with time-varying covariates in Figure 3 estimates 33 parameters, requiring a minimum sample size of 165. Thus, our sample size of 167 was adequate for the proposed analyses.

Results

CFA was used to compute socioeconomic status factor scores at each time point using a variety of candidate variables. Four of the factor scores tested demonstrated significant loadings from each of the indicators across all three time points and good model fit with CFI values greater than .95 and RMSEA values less than .08. The factor score selected for the model included parent and spouse standardized years of education and standardized total annual household income. The decision to proceed with the factor score used in the analyses was based on prior research emphasizing the role of income and parental education as important indicators of SES. Factor scores that were computed but not included in the final model for lack of conceptual foundation or model fit are reported in Appendix B. Preliminary model results revealed a small, negative, and non-significant residual variance of the parent and spouse education composite variable ($-0.01, p = .95$), thus the residual variance of this variable was fixed to 0 for subsequent analyses. Factor loadings were set to be equal and the factor variance was set to 1 for model convergence, due to having only two indicators. This factor model indicated a good fit at Time 1 ($\chi^2 = 57.17, df = 1, p = .95, CFI = 1.00, \text{ and RMSEA} = .00$), Time 2 ($\chi^2 = 37.79, df = 1, p = .95, CFI = 1.00, \text{ and RMSEA} = .00$), and Time 3 ($\chi^2 = 25.052, df = 1, p = .95, CFI = 1.00, \text{ and RMSEA} = .00$).

Descriptive statistics for all study variables are presented in Table 1. Skewness and kurtosis were assessed for all study variables. Delay discounting rates at all three

time points were log transformed prior to use. Mahalanobis distance was used to detect extreme multivariate outliers across each of the models (i.e., multivariate outliers significant at the $p < .001$ level). Cigarette use frequency ($n = 2$), alcohol use frequency ($n = 1$), marijuana use frequency ($n = 7$) and polysubstance use frequency ($n = 3$) extreme cases were removed prior to the multivariate analyses. Descriptive statistics of delay discounting and substance use frequency appeared to suggest trends showing that delay discounting, on average, declined over time, whereas substance use frequency increased over time.

Multivariate GLM was conducted to test for the effects of demographic covariates on the outcomes in each of the substance use frequency and onset models: delay discounting and cigarette use frequency, delay discounting and alcohol use frequency, delay discounting and marijuana use frequency, and delay discounting and polysubstance use frequency as well as delay discounting and cigarette use onset, delay discounting and alcohol use onset, delay discounting and marijuana use onset, and delay discounting and polysubstance use onset. Analyses revealed significant effects of gender ($p = .02$), but not age ($p = .26$) or race ($p = .49$; dichotomized as white vs. non-white) on delay discounting and polysubstance use frequency outcome variables at Times 1, 2, and 3. For the delay discounting and specific substance use frequency models, analyses revealed significant effects of gender ($p = .00$) and race ($p = .01$; dichotomized as white vs. non-white), but not age ($p = .30$), on marijuana use frequency and delay discounting outcome variables. There were no significant effects of gender ($p = .07$), age ($p = .68$) or race ($p = .81$; dichotomized as white vs. non-white) on the cigarette use frequency and delay discounting outcome variables. Similarly, there were no significant effects of gender ($p =$

.06), age ($p = .19$), or race ($p = .82$; dichotomized as white vs. non-white) on the alcohol use frequency and delay discounting outcome variables. Thus, when covariates were added to the model, gender was included as a time-invariant covariate (TIC) in the conditional parallel process GCMs (see Figures 2 and 3) involving polysubstance use frequency and gender and race were included as TICs in the conditional parallel process GCMs involving delay discounting and marijuana use frequency.

Multivariate GLM was also used to test for significant effects of demographic variables on the delay discounting and substance use onset variables (assessed at Time 3). There were significant effects of gender ($p = .02$), but not age ($p = .27$) or race ($p = .33$; dichotomized as white vs. non-white) on the polysubstance use onset and delay discounting outcome variables. Additionally, there were significant effects of gender ($p = .00$), but not age ($p = .39$) or race ($p = .48$; dichotomized as white vs. non-white) on cigarette use onset and delay discounting variables. Similarly, there were significant effects of gender ($p = .02$), but not age ($p = .39$), or race ($p = .13$; dichotomized as white vs. non-white) on marijuana use onset and delay discounting outcome variables. There were no significant effects of gender ($p = .09$), age ($p = .41$), or race ($p = .57$; dichotomized as white vs. non-white) on the alcohol use onset and delay discounting outcome variables. Thus, gender was only included as a covariate in the substance use onset models involving polysubstance use onset, cigarette use onset, and marijuana use onset. Gender and race were centered prior to their inclusion in the models (when applicable) for clarity of interpretation.

Growth curve modeling was conducted following the guidelines of Bollen and Curran (2006), first analyzing the unconditional univariate growth curve models for delay

discounting, and substance use frequency with no slope (intercept only), then analyzing the model with a linear slope (by specifying the parameters to 0, 1, and 2), and then analyzing the latent model which allows the parameters to be estimated freely (model specification requires the first and last parameters to be fixed, leaving the second parameter to be freely estimated), and evaluating model fit to identify the best-fitting models. Within-process residual variances were set to equality to improve model fits when necessary. Nested model comparisons were conducted for models demonstrating adequate fits across no-growth, linear and latent growth trajectories and for tests of equality constraints on SES parameters using Satorra-Bentler scaled correction factor (Satorra & Bentler, 2001), which is robust to nonnormality compared to alternate difference test statistics (Curran, West, & Finch, 1996).

Results of the nested model comparisons between the no growth, linear growth, and latent growth univariate models indicated linear growth as the best fitting for each of the models (see Table 2). Thus, all subsequent modeling included growth factors reflecting a linear growth structure. The univariate GCMs were then combined and fit into a series of unconditional and conditional (including covariates) parallel process models. The first parallel process model (unconditional) included correlations among all four growth factor intercepts and slopes (see Figure 1). This model provides growth factor intercept and slope mean and variance estimates and correlations as well as meaningful information about how the variables change over time but does not incorporate predictors in the model. Thus, to address the first and second hypotheses, the unconditional parallel process model was extended to a conditional (including covariates) parallel process model by including SES as a time-invariant covariate (and demographic

covariates when applicable; see Figure 2) on delay discounting and substance use intercepts and slopes and between-process regressions. Specifically, between-process slopes were regressed onto between-process intercepts and substance use intercepts and slopes were regressed onto delay discounting intercepts and slopes, respectively (see Figure 2). This allowed us to examine whether initial levels of SES, as a time-invariant covariate, predicted initial levels and slopes of delay discounting and whether initial levels and slopes of delay discounting predicted substance use growth rates in-turn (and vice versa). Next, we tested a series of conditional parallel process models incorporating SES as a time-varying covariate (TVC) (Bollen & Curran, 2006) to address the third hypothesis and estimate whether time-specific measures of SES were associated with growth above and beyond average growth in delay discounting and substance use over time (see Figure 3).

Finally, any models resulting in significant associations among SES and growth factor intercept or slope as well as significant associations among between process growth factor intercept or slope, indicated potentially significant indirect effects, and were tested using the model indirect command in *MPlus* with 5,000 bootstrap confidence intervals (Muthén & Muthén, 1998-2017; Preacher & Hayes, 2008). Maximum Likelihood (ML) estimates were used for conducting bias-corrected bootstrapping tests of indirect effects using delay discounting growth factor intercept as the mediator and substance use growth factor slope (or Time 3 substance use onset, when applicable) as the outcome (see Figure 2), which account for potentially asymmetric distribution estimates (Preacher & Hayes, 2008). The parameter estimates and bootstrap standard

errors from MLR are comparable to those obtained with ML estimation and are robust to non-normality (Muthén & Muthén, 1998-2017).

To examine whether SES predicted average growth and individual differences in changing rates of delay discounting and substance use frequency, covariates were added in two separate GCMs for each of the parallel processes: 1) with SES as a TIC to test the effects of SES at Time 1 on the growth factors of delay discounting and substance use and examine delay discounting as a mediating process between SES and substance use, and 2) with SES as a TVC (and gender and race as demographic TICs when applicable) to determine whether SES was associated with contemporaneous individual differences in delay discounting and substance use at each time point, above and beyond average levels of growth.

To examine the associations among SES, delay discounting, and substance use onset, cigarette, alcohol, marijuana, and polysubstance use onset were tested in a univariate GCM variation of the conditional parallel process models with time-invariant covariates and between-process regressions (see Figure 2). Specifically, delay discounting intercept and slope were regressed onto SES and substance use onset was regressed onto delay discounting intercept, delay discounting slope, and SES. Due to the nature of substance use onset (i.e., onset does not indicate time-dependent growth patterns), onset variables were not tested for growth or with time-varying covariates. Instead, substance use onset variables reported at Time 3 were used as outcomes. Gender was included as a time-invariant covariate in the conditional parallel process models with TICs involving cigarette use onset, marijuana use onset, and polysubstance use onset.

All GCMs provide estimates for the latent growth factor intercepts and slopes. The intercept mean (fixed effect) is the initial status when the time score is zero (time scaling was set to zero at Time 1 to center the intercepts as the initial status) and the intercept variance (random effect) is the individual variability in initial status when the time score is zero. The slope mean is the systematic rate of change with an increase in one unit of time (each time unit represents a year), whereas the slope variance indicates the individual variability in rate of change over time (Muthén & Muthén, 1998-2017). Significant variability on the mean and variance of the intercept and slope indicates individual variability in starting levels and rates of change. Intercept and slope growth factors covaried within factors, indicating the degree to which individual differences in initial status levels of the intercepts and individual differences in the rate of change in slopes are related. TIC models explain variance in the growth processes, whereas TVC models explain variance in individual differences, above and beyond average growth processes (Bollen & Curran, 2006).

Results from the unconditional univariate models demonstrated significant intercept and slope variance for the delay discounting, alcohol use frequency, and polysubstance use frequency models, whereas the unconditional univariate cigarette use frequency model exhibited nonsignificant cigarette use frequency intercept variance and nonsignificant cigarette use frequency slope variance and the marijuana use frequency model exhibited significant marijuana use frequency slope variance but nonsignificant marijuana use frequency intercept variance (see Tables 3-6). Nonetheless, the models were retained for testing in the unconditional and conditional parallel process models to examine whether the inclusion of covariates could potentially describe significant

variances in delay discounting intercept and slope and cigarette use intercept as well as delay discounting intercept and slope and marijuana use frequency slope. Thus, each of the univariate models were fit and tested in the unconditional parallel process models as well as the extended conditional parallel process models with covariates.

Nested model comparisons were conducted to test two competing TVC GCMs: 1) with SES as a TVC where the parameters from each indicator varied across measurement and 2) where equality constraints were imposed on the parameters of SES testing whether the SES effects were comparable over time. If constraining the parameters did not significantly degrade the model fit (indicated by a non-significant chi-square difference value), the more parsimonious model with the equality constraints was preferred. Nested model comparison results (between the null model where the parameters for the SES effects were fixed to be equal and the nested model where the parameters were estimated freely at each time point) indicated that imposing equality constraints did not significantly degrade the model fits for the TVC GCMs involving cigarette use frequency, alcohol use frequency, marijuana use frequency or polysubstance use frequency (see Table 7). Thus, results of the TVC GCMs reflect the imposed SES equality constraints for each of the TVC models.

Cigarette Use Frequency

The unconditional parallel process GCM (see Figure 1) including delay discounting and cigarette use frequency demonstrated acceptable model fit ($\chi^2 = 11.50$, $df = 11$, $p = .40$, CFI = 1.00, and RMSEA = .02). Results from the unconditional parallel process model revealed no significant associations between delay discounting cigarette use frequency intercepts and slopes (see Table 3).

The conditional parallel process GCM (see Figure 2) with SES as a TIC including delay discounting and cigarette use frequency (see Table 3), demonstrated good fits ($\chi^2 = 12.24$, $df = 22$, $p = .51$, CFI = 1.00 and RMSEA = .00). In this model, delay discounting and cigarette use frequency intercepts and slopes were regressed onto SES, and between-process correlations were changed to regressions to examine directional prediction among them. Specifically, cigarette use frequency intercept and slope were regressed onto delay discounting intercept and slope, respectively, cigarette use frequency slope was regressed onto delay discounting intercept, and delay discounting slope was regressed onto cigarette use frequency intercept (see Figure 2). SES significantly predicted delay discounting and cigarette use frequency intercepts, such that lower SES was associated with higher initial levels of delay discounting and higher initial levels of cigarette use frequency. However, SES was not significantly associated with delay discounting or cigarette use frequency slopes, and there were no other significant between-process associations.

The TVC GCM (see Figure 3) including cigarette use frequency and delay discounting, with SES as a time-varying covariate (see Table 3), demonstrated good model fits ($\chi^2 = 28.64$, $df = 26$, $p = .33$, CFI = .99, and RMSEA = .03). However, SES did not significantly predict delay discounting or cigarette use frequency at any time point and there were no other significant associations between delay discounting and cigarette use frequency intercepts and slopes.

Alcohol Use Frequency

The unconditional parallel process GCM (see Figure 1) including delay discounting and alcohol use frequency (see Table 4) demonstrated good model fit ($\chi^2 =$

24.61, $df = 11$, $p = .01$, CFI = .94, and RMSEA = .09). Results from the unconditional parallel process model revealed a significant negative correlation between alcohol use frequency intercept and delay discounting slope, indicating that higher initial levels of alcohol use frequency were associated with steeper declines in delay discounting. There was also a significant positive correlation among delay discounting intercept and alcohol use frequency intercept, indicating that higher initial levels of alcohol use frequency were associated with higher initial levels of delay discounting.

The conditional parallel process GCM (see Figure 2) with SES as a TIC including between-process regressions among the parallel processes (see Table 4), fit the data well ($\chi^2 = 24.73$, $df = 13$, $p = .03$, CFI = .95, and RMSEA = .07). In this model, delay discounting and alcohol use frequency intercepts and slopes were regressed onto SES, and between-process correlations were changed to regressions to examine directional predictions among them. Specifically, alcohol use frequency intercept and slope were regressed onto delay discounting intercept and slope, respectively, alcohol use frequency slope was regressed onto delay discounting intercept, and delay discounting slope was regressed onto alcohol use frequency intercept (see Figure 2). SES significantly predicted delay discounting and alcohol use frequency intercepts, such that lower SES was associated with higher initial levels of delay discounting and higher initial levels of alcohol use frequency. Additionally, the significant negative correlation between alcohol use frequency intercept and delay discounting slope found in the unconditional parallel process model became nonsignificant with the inclusion of SES as a covariate and between-process regressions. However, SES was not significantly associated with and

delay discounting or alcohol use frequency slopes, and there were no other significant between-process associations.

The TVC GCM (see Figure 3) including alcohol use frequency and delay discounting, with SES as a time-varying covariate (see Table 4), demonstrated good model fits ($\chi^2 = 35.31$, $df = 24$, $p = .06$, CFI = .96, and RMSEA = .05). There was a significant positive association between alcohol use frequency intercept and delay discounting intercept such that higher initial levels of delay discounting were associated with higher initial levels of alcohol use frequency. However, SES did not significantly predict delay discounting or alcohol use frequency above and beyond average growth at any time point, and there were no other significant associations between delay discounting and alcohol use frequency intercepts and slopes.

Marijuana Use Frequency

The unconditional parallel process GCM (see Figure 1) including delay discounting and marijuana use frequency (see Table 5) demonstrated acceptable model fit ($\chi^2 = 31.96$, $df = 11$, $p = .00$, CFI = .92, and RMSEA = .11) despite its high RMSEA value since caution should be taken for using strict RMSEA cutoff values for models with few degrees of freedom (Kenny, Kaniskan, & McCoach, 2015). However, there were no significant correlations between delay discounting and marijuana use frequency intercepts and slopes.

The conditional parallel process GCM (see Figure 2) with SES, gender, and race as TICs including between-process regressions among the parallel processes (see Table 5) demonstrated adequate model fit ($\chi^2 = 43.50$, $df = 17$, $p = .00$, CFI = .91, and RMSEA = .10). In this model, delay discounting and marijuana use frequency intercepts and

slopes were regressed onto SES, and between-process correlations were changed to regressions to examine directional predictions among them. Specifically, marijuana use frequency intercept and slope were regressed onto delay discounting intercept and slope, respectively, marijuana use frequency slope was regressed onto delay discounting intercept, and delay discounting slope was regressed onto marijuana use frequency intercept (see Figure 2). There was a significant positive association between delay discounting intercept and marijuana use frequency slope, indicating that higher initial levels of delay discounting were associated with steeper increases in delay discounting. However, SES was not significantly associated with delay discounting or marijuana use frequency intercepts or slopes and there were no other significant between-process associations and no significant associations between gender or race and delay discounting and marijuana use frequency intercepts and slopes.

The SES TVC GCM (see Figure 3) including delay discounting and marijuana use frequency demonstrated acceptable model fit (see Table 5; $\chi^2 = 56.89$, $df = 30$, $p = .00$, CFI = .92, and RMSEA = .07). However, there were no significant associations between SES and delay discounting or marijuana use frequency intercepts and slopes and no significant between-process associations and no significant associations between gender or race and delay discounting and marijuana use frequency intercepts and slopes.

Polysubstance Use Frequency

The unconditional parallel process GCM (see Figure 1) including delay discounting and polysubstance use frequency demonstrated adequate model fit ($\chi^2 = 22.42$, $df = 11$, $p = .02$, CFI = .96, and RMSEA = .08) despite its high RMSEA value since caution should be taken for using strict RMSEA cutoffs for models with few

degrees of freedom (Kenny et al., 2015). However, there were no significant correlations between delay discounting and polysubstance use frequency intercepts and slopes.

The conditional parallel process GCM (see Figure 2) with SES and gender TICs including between-process regressions among the parallel processes (see Table 6) demonstrated acceptable model fit ($\chi^2 = 25.01$, $df = 15$, $p = .05$, CFI = .97, and RMSEA = .06). In this model, delay discounting and polysubstance use frequency intercepts and slopes were regressed onto SES, and between-process correlations were changed to regressions to examine directional predictions among them. Specifically, polysubstance use frequency intercept and slope were regressed onto delay discounting intercept and slope, respectively, polysubstance use frequency slope was regressed onto delay discounting intercept, and delay discounting slope was regressed onto polysubstance use frequency intercept (see Figure 2). There was a significant negative association between SES and polysubstance use frequency slope, above and beyond the effect of gender, such that lower SES was associated with steeper increases in polysubstance use frequency. Furthermore, there was a significant positive association between gender and polysubstance use frequency slope, indicating that being female was associated with steeper increases in polysubstance use frequency. There was also a significant positive association between delay discounting intercept and polysubstance use slope, indicating that higher initial levels of delay discounting were associated with steeper increases in polysubstance use frequency. However, SES was not significantly associated with delay discounting or polysubstance use frequency intercepts or delay discounting slope, and gender was not significantly associated with delay discounting or polysubstance use

frequency intercepts or delay discounting slope and there were no other significant between-process associations.

The SES TVC GCM (see Figure 3) including delay discounting and polysubstance use frequency demonstrated acceptable model fit (see Table 6; $\chi^2 = 43.81$, $df = 31$, $p = .06$, CFI = .96, and RMSEA = .05). Delay discounting intercept was significantly positively associated with polysubstance use frequency slope such that higher initial levels of delay discounting were associated with steeper increases in polysubstance use frequency. However, there were no significant contemporaneous effects of SES on delay discounting or polysubstance use and no other significant between-process regressions or significant associations between gender and delay discounting or polysubstance use.

Cigarette Use Onset

The conditional parallel process GCM (see Figure 2) including cigarette use onset and delay discounting with SES and gender TICs demonstrated acceptable model fit (see Table 8; $\chi^2 = 12.99$, $df = 6$, $p = .04$, CFI = .94, and RMSEA = .08). In this model, delay discounting residual variance set to be equal across time points to achieve acceptable model fit. SES significantly predicted delay discounting intercept and cigarette use onset, above and beyond the effect of gender, such that lower SES was associated with higher initial levels of delay discounting and earlier cigarette use onset. However, neither delay discounting intercept nor delay discounting slope predicted cigarette use onset, precluding further testing of indirect effects of SES on cigarette use onset via delay discounting.

Alcohol Use Onset

The conditional parallel process GCM (see Figure 2) including alcohol use onset and delay discounting with SES and gender TICs demonstrated acceptable model fit (see Table 8; $\chi^2 = 6.51$, $df = 3$, $p = .09$, CFI = .97, and RMSEA = .08). SES significantly predicted delay discounting intercept, such that lower SES was associated with higher initial levels of delay discounting. Higher delay discounting intercept, but not slope, significantly predicted earlier alcohol use onset. SES significantly predicted delay discounting intercept, such that lower SES was associated with higher initial levels of delay discounting. Given the significant associations between SES and delay discounting intercept and between delay discounting intercept and alcohol use onset, the indirect effect from SES to alcohol use onset via delay discounting intercept was tested. Bias-corrected confidence intervals of 5,000 iterations revealed significant mediation effects from SES to alcohol use onset such that lower SES was related to earlier onset alcohol use via higher initial levels of delay discounting ($b = -.07$, $SE = .05$, 95% CI [-.407, -.004]; see Table 8).

Marijuana Use Onset

The conditional parallel process GCM (see Figure 2) including marijuana use onset and delay discounting with SES and gender TICs demonstrated acceptable model fit (see Table 8; $\chi^2 = 13.04$, $df = 6$, $p = .04$, CFI = .94, and RMSEA = .08). In this model, delay discounting residual variance was set to be equal across time points to achieve acceptable model fit. Above and beyond the effect of gender, SES significantly predicted delay discounting intercept, but not slope, such that lower SES was associated with higher initial levels of delay discounting. SES also significantly predicted marijuana use onset such that lower SES was associated with earlier marijuana use onset. However,

neither delay discounting intercept nor delay discounting slope significantly predicted marijuana use onset, precluding testing of mediation effects of delay discounting between SES and marijuana use onset.

Polysubstance Use Onset

The conditional parallel process GCM (see Figure 2) including polysubstance use onset and delay discounting with SES and gender TICs demonstrated adequate model fit (see Table 8; $\chi^2 = 8.00$, $df = 4$, $p = .09$, CFI = .97, and RMSEA = .08). SES significantly predicted delay discounting intercept and polysubstance use onset, above and beyond the effect of gender, such that lower SES was associated with higher initial levels of delay discounting and earlier polysubstance use onset. Furthermore, delay discounting intercept, but not slope, significantly predicted substance use onset such that higher initial levels of delay discounting were associated with earlier polysubstance use onset. Gender, however, did not significantly predict delay discounting intercept, delay discounting slope, or polysubstance use onset. Given the significant associations among SES, delay discounting intercept, and polysubstance use onset, the indirect effect from SES to polysubstance use onset via delay discounting intercept was tested. Bias-corrected confidence intervals of 5,000 iterations revealed significant mediation effects from SES to polysubstance use onset such that lower SES was related to earlier onset polysubstance use via higher initial levels of delay discounting ($b = -.07$, $SE = .05$, 95% CI [-.392, -.001]; see Table 8).

Discussion

Understanding associations among SES, delay discounting, and adolescent substance use may provide vital information for prevention and intervention efforts

aimed at interrupting socioeconomic health disparities across the lifespan. This study examined influences of SES on delay discounting and substance use growth trajectories and substance use onset, during a developmental period when individual differences in substance use may be harbingers of risk for later problematic substance use. We hypothesized that as a time-invariant covariate, low SES would predict high initial levels of both delay discounting and substance use and increasing slopes for both delay discounting and substance use. We further hypothesized that delay discounting growth factors would mediate the link between SES and adolescent substance use development such that low SES is related to high intercept and increasing slope of delay discounting, which in turn, are related to high intercept and increasing slope of substance use. We also examined SES as a time-varying covariate, expecting that SES at each time point would exhibit contemporaneous effects on delay discounting and substance use such that low SES would be related to individual differences in elevated delay discounting and elevated substance use.

Results from the longitudinal latent growth curve model demonstrated unique influences of socioeconomic status on delay discounting and substance use growth trajectories by substance type. Lower SES (as a time-invariant covariate) was associated with higher initial levels of delay discounting, cigarette, and alcohol use frequency (but not higher initial levels of marijuana or polysubstance use frequency) and earlier onset cigarette, marijuana, and polysubstance use (but not alcohol use onset). Additionally, lower SES was associated with steeper increases in polysubstance use frequency. However, SES was not directly significantly associated with changing rates of delay discounting or specific substance use frequency. Delay discounting was significantly

associated with changes in substance use and substance use onset, but these associations varied by substance type. Specifically, higher initial levels of delay discounting were associated with steeper increasing rates of marijuana use frequency, and earlier onset alcohol and polysubstance use (but not cigarette or marijuana onset). Furthermore, delay discounting intercept mediated the link between SES and alcohol and polysubstance use onset such that low SES was related to high delay discounting intercept, which was related to earlier onset alcohol and polysubstance use. Finally, testing the models with SES as a time-varying covariate, we did not find developmental differences in the effects of SES on delay discounting and substance use frequency, indicating that adolescent delay discounting and substance use were not particularly susceptible to socioeconomic influences at any of the assessment time points.

This is the first study, to our knowledge, to use a longitudinal design to examine delay discounting development from early-middle adolescence, when delay discounting preferences are relatively plastic. Prior cross-sectional research has suggested that delay discounting steeply increase from ages 10 to 15, where it peaks, and then gradually declines from 16 to 20, where it reaches relative stability and is comparable to adult preferences (Steinberg et al., 2009). To our knowledge, the only other longitudinal study to examine delay discounting growth utilized an older sample of adolescents (assessments were not measured across equidistant time points, beginning during the second semester of the 10th grade, after delay discounting putatively peaks, and ending 2 years after high-school graduation), and found relatively stable delay discounting growth across time points (Audrain-McGovern et al., 2009). Our results, however, revealed a slightly different picture of adolescent delay discounting development: delay discounting

was at its highest at the time of the first assessment, and descended monotonically across annual assessments. Thus, it is possible that delay discounting may reach its apogee even sooner than previously suspected. Taken together with the findings from Audrain-McGovern et al. (2009), in which delay discounting did not significantly vary from the baseline (in an older sample of adolescents), our results indicate that earlier delay discounting assessments (i.e., across early to middle adolescence) may be necessary to capture significant developmental variation in delay discounting trajectories before rates stabilize. Furthermore, the steep declining patterns of delay discounting growth from ages 13-16 were somewhat consistent (albeit slightly earlier) with declining patterns of delay discounting found in the cross-sectional study by Steinberg et al. (2009).

Our findings revealed unique delay discounting and substance use growth patterns as well as associations among SES, delay discounting, and specific substance use growth, signifying that low SES may directly elevate risks for initial cigarette and alcohol use frequency and for increasing rates of polysubstance use frequency during early adolescence. To evaluate whether SES, delay discounting, and substance use growth patterns varied by substance, we examined cigarette, alcohol, marijuana, and polysubstance use separately. There was significant variation in alcohol and polysubstance use intercepts and slopes (i.e., starting levels and rates of change, respectively) across development, whereas marijuana use frequency exhibited significant variation in growth but not starting levels and there was no significant variation in cigarette use frequency starting levels or rates of change over time. The significant variation in the changing rates of alcohol, marijuana, and polysubstance use indicated significantly different patterns of growth among adolescents emerged over the course of

assessments, whereas non-significant variation in changing rates of cigarette use suggest rather stable developmental trajectory patterns for adolescent smoking.

Substance use exhibited linear growth across cigarette, alcohol, marijuana, and polysubstance use frequency (despite relatively stable cigarette use frequency growth). These trends reflect generally similar national adolescent substance use trends found in the Monitoring the Future Study for 8th, 10th, and 12th grade adolescents spanning the same time period (Johnston, O'Malley, Bachman, Schulenberg, & Miech, 2017). Specifically, national adolescent cigarette, alcohol, and marijuana use trends exhibited a linear gradient across the 8th to 12th grades (i.e., 4%, 6%, and 11% across 8th, 10th, and 12th grades). We also found significant gender differences in changing rates of polysubstance use frequency. Specifically, female adolescents showed significantly steeper increases in polysubstance use frequency than male adolescents.

We examined the effects of SES on delay discounting and substance use as both a time-invariant and time-variant covariate and the indirect effects of SES on adolescent substance use via delay discounting. SES was tested separately as a TIC and TVC to examine 1) socioeconomic influences on delay discounting and substance use levels and 2) whether SES explained additional variance in delay discounting and substance use at specific time points, above and beyond average growth trajectories. These findings revealed substance specific associations with SES related to overall growth but we did not find any unique time-specific associations among SES, delay discounting, and substance use, above and beyond average development. As a time-invariant covariate, lower SES was directly associated with higher initial levels of delay discounting, cigarette, and alcohol use frequency, as well as changing rates of polysubstance use, but

it was not directly associated with initial levels of marijuana or polysubstance use frequency. However, higher initial levels of delay discounting were significantly associated with steeper increases in marijuana and polysubstance use frequency (but not cigarette or alcohol use frequency). SES also directly influenced cigarette, marijuana, and polysubstance use (cigarette, alcohol, and marijuana) onset and indirectly influenced alcohol use onset such that lower SES was associated with earlier cigarette, marijuana, and polysubstance onset directly and earlier alcohol onset indirectly. Results from the mediation model provided insight into the mediating effects of delay discounting in the links between SES and alcohol use onset as well as SES and polysubstance use onset. Specifically, lower SES significantly predicted earlier onset of polysubstance use directly and indirectly via higher initial levels of delay discounting and earlier onset of alcohol use indirectly via higher initial levels of delay discounting. These findings are consistent with prior research suggesting that higher levels of impulsivity may pose heightened risks for substance initiation, in particular (Kim-Spoon et al., 2014; Koob & Le Moal, 2001).

As a time-varying covariate, SES did not significantly explain any unique variance in delay discounting and substance use frequency at any time point. Instead, socioeconomic risks for substance use may unfold differently by substance and risk type: low SES may play an important role in determining the onset of alcohol and polysubstance use. Furthermore, delay discounting may be especially harmful for non-clinical adolescents due to its associations with substance use onset, in particular. The significant indirect effects in this study identify delay discounting as an important process explaining why low SES adolescents may be at elevated risk for early onset alcohol and polysubstance use.

Our findings that low SES was associated with greater delay discounting, align with prior research which suggests that adolescents from low SES may experience disproportionate environmental stress, which may impose cognitive strain to impair decision making (Evans & Kantrowitz, 2002). Our findings demonstrating peak levels of delay discounting at a period when the asynchrony between reward and executive systems are putatively developmentally imbalanced, may provide behavioral evidence in support of CNDs theory, which posits that the imbalance of these competing systems may lead to maladaptive decision making and greater risk for developing substance use disorders (Bickel et al., 2007). Furthermore, low SES may heighten developmental decision making risks by restricting temporal perspective to lead to greater delay discounting and greater substance use, in-turn (Bickel et al., 2014). Specifically, adolescents from low SES exhibited elevated delay discounting from the study outset, which in-turn was associated with earlier onset alcohol and polysubstance use. Although adolescents from low SES also exhibited higher initial levels of cigarette use frequency, we did not find significant associations between delay discounting and cigarette smoking onset or use. These findings support to the possibility that associations between delay discounting and cigarette smoking may be different between adolescents and adults. Indeed, prior research comparing delay discounting differences between adolescent smokers and non-smokers has also found null associations between adolescent delay discounting and cigarette smoking, controlling for SES (Fields, Collins, Leraas, & Reynolds, 2009; Reynolds, Karraker, Horn, & Richards, 2003). Thus, detectable delay discounting differences between adolescent smokers and non-smokers may emerge after

more extensive or prolonged use but SES may be a more reliable predictor of adolescent cigarette smoking onset during early-middle adolescence.

Earlier age of substance use onset has been associated with greater risks for later development of substance use disorders. At the neural level, early onset substance use may strengthen the neural connections between reward (NAcc) and cognitive control networks (rPFC), which may be one pathway from early onset substance use to lead to vulnerability for later substance use disorders (Weissman et al., 2015). Thus, delay discounting interventions aimed at reducing socioeconomic substance use disparities may be especially effective during early-middle adolescence, when delay discounting levels are plastic and prior to substance use onset or escalated use (Daniel, Said, Stanton, & Epstein, 2015; Peters & Büchel, 2010). At the broader level, policies aimed at improving economic stability may also reduce the cognitive strain associated with low SES, and diminish its associations with delay discounting and substance use onset (Mani et al., 2013).

Associations among SES and delay discounting may differ as a function of the economic burdens facing adolescents and their families at any given time (Bickel, Wilson, Chen, Koffarnus, & Franck, 2016). Specifically, abrupt changes to income may restrict an individual's temporal frame of reference and lead to increasingly myopic decision making. Although parent-adolescent perceived SES becomes more similar as adolescents develop (Goodman et al., 2001), adolescents may not be privy to economic challenges facing their families at any given time. Thus, despite our findings that the influences of SES on adolescent delay discounting and substance use were relatively stable (using a standard measure of SES combining parental education and annual

income), alternative adolescent-reported measures of SES (e.g., subjective socioeconomic status), may capture socioeconomic influences less readily filtered by parents. Indeed, parent-adolescent perceived SES becomes more concordant as adolescents develop, which may be one avenue whereby SES confers intergenerationally similar behaviors such as delay discounting and substance use. Although the present study demonstrated significant associations among SES and delay discounting, it is unclear whether socioeconomic resource scarcity, associated with fewer resources available to meet one's needs leads to heightened reward sensitivity, diminished cognitive control, or both, to influence delay discounting rates and substance use (Mani et al., 2013). An important direction for future research is to explore associations among SES and executive function neurodevelopment, delay discounting, and substance use to clarify how SES gets under the skin to influence adolescent health risk behaviors.

Notably, we did not find significant associations between delay discounting and cigarette smoking onset or use despite well-documented differences in delay discounting between adult smokers versus non-smokers (Bickel et al., 1999; Reynolds, Richards, Horn, & Karraker, 2004). It is unclear, however, when these differences emerge and whether they develop during the initial stages of adolescent smoking or manifest after prolonged or more frequent use. For example, one study compared differences in adolescent smokers (smoking at least one cigarette a week over the past 6-months), triers (having tried smoking during the past 6-months) and non-smokers (having never tried) and found no significant differences in delay discounting between the groups (Reynolds et al., 2003). Also, relatively consistent with our results, that study found that higher paternal education was significantly associated with having never tried smoking. Another

study comparing delay discounting between adolescent smokers (smoking at least 4 cigarettes per day for the last 3 months) and non-smokers (having never tried smoking) found significant differences in delay discounting such that smokers discounted significantly more than non-smokers but these differences became nonsignificant after controlling for differences in IQ and income (Fields et al., 2009). Thus, it is possible that delay discounting differences between adolescent smokers and non-smokers may emerge after more prolonged smoking or greater frequency of smoking but that SES may be a more reliable predictor of risk for early adolescent smoking onset. Indeed, we found that lower SES was significantly associated with higher initial levels of delay discounting and greater initial levels of smoking frequency.

Despite the study's strengths, some limitations should also be noted. First, the relatively small sample size and limited time points precluded testing the examination of moderator variables (such as genotype, family history of substance use, parental disclosure about financial circumstances, adolescent gender, pubertal development, reward sensitivity), and potentially differential associations with SES, delay discounting and substance use (Dom, D'haene, Hulstijn, & Sabbe, 2006; Dougherty et al., 2014; Sweitzer et al., 2013). Additional time points would facilitate more complex modeling strategies such as growth mixture modeling (GMM) to test for unique trajectory subgroups among delay discounting and substance use trajectories (Jung & Wickrama, 2008). Second, for adolescents who started using substances prior to Time 1, SES cannot predict their substance use onset, but it is associated with it. Future studies using a substance naïve sample to examine the association between delay discounting and substance use onset may be useful to clarify the directionality of these associations. The

non-significant findings between delay discounting and cigarette smoking in our community sample of adolescents indicate the possibility that there may be a certain threshold for smoking frequency and onset to lead to significant differences in delay discounting. However, how and when detectable differences in delay discounting manifest from smoking, and whether they lead to bidirectional associations between delay discounting and smoking, will be left for future research. Given the previous finding suggesting differences in reward processing activation in the ventral striatum observed between non-smokers and smokers who have smoked even fewer than 10 occasions (Peters et al., 2011; Reynolds, 2004), it is also possible that the associations between delay discounting and smoking are more readily detectable in delay discounting related brain responses, compared to delay discounting behavior, per se.

Finally, our data showed delay discounting at its highest point during the first assessment. Future research spanning a broader range of ages may offer additional insight into developmental patterns of delay discounting growth, leading up to, and descending its peak. Indeed, the current findings dovetail with the declining growth patterns found by Steinberg et al. (2009) and (Water et al., 2014) but the shift from declining to stabilizing growth was not observed across these time points. Thus, whether delay discounting rates continue declining or stabilize after age 16 is yet to be seen.

This is the first study to examine longitudinal growth of delay discounting and substance use trajectories across adolescent development. Our findings indicate that delay discounting declines monotonically across early to middle adolescence and that higher initial levels of delay discounting pose significant risks for early adolescent alcohol and polysubstance use frequency and onset and escalating rates of marijuana use. Significant

socioeconomic risks for delay discounting and substance use onset unfolded directly and indirectly across development and manifested differently by substance type. Low SES was directly associated with greater cigarette and alcohol frequency, steeper increases in polysubstance use frequency, and earlier onset cigarette, marijuana, and polysubstance use. Indirectly, low SES was associated with earlier onset alcohol and polysubstance. Specifically, delay discounting significantly mediated the effects of SES on alcohol and polysubstance use onset such that lower SES was associated with higher delay discounting starting levels and earlier onset alcohol and polysubstance use in-turn. Our findings demonstrating peak levels of delay discounting at a period when the asynchrony between reward and executive systems are putatively developmentally imbalanced, provides behavioral evidence in support of CNDS theory (Bickel et al., 2007). Furthermore, the present study adds empirical support to the perspective that SES may impose cognitive strain and restrict temporal perspective to lead to greater delay discounting (Bickel et al., 2014). These results emphasize the importance of examining adolescent substance use by specific substance, as well as collapsing across substances. Importantly, our findings indicate delay discounting risks for adolescent alcohol use onset and polysubstance use onset, which may be especially important given the increasing popularity of alcohol use across adolescent development. Despite the imposing challenges of low SES, delay discounting interventions may be a promising avenue for reducing socioeconomic disparities in early onset alcohol and polysubstance use, when delay discounting development is still underway.

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Appendix A: Measures

Demographic Interview

Introduction:

I am going to ask you some basic questions about the work and educational experiences of yourself and of the people in your household. These questions are very important and need to be answered honestly. No one outside of the project will ever have access to this information. The information that you provide us will not affect any services or assistance that you might be receiving. This information will only be used for the purposes of our research.

1. What is your relation to the child? (RESNM)

- 1-Mother
- 2-Father
- 3-Grandmother
- 4-Grandfather
- 5-Foster Parent
- 6-Other – Specify who other is _____

2. What is your sex? (R_SEX)

- 0-Male
- 1-Female

3. If respondent is not the biological parent ask:

“How long has this child been in your care?” (RCARE) _____

4. How old are you? (Record age in years.)

AGE _____

5. What are your birth month and year? (MonthBirth)

____/____/____
Month Year

Now I am going to ask you about your current family situation.

7. What is your current marital status- married, widowed, separated, divorced, never married, or living with someone? (RMASTAT) *(If separated, ask “Is this separation legal or not legal?”)*

- 1 never married
- 2 married
- 3 widowed
- 4 divorced
- 5 legally separated
- 6 separated, not legally

7 living with someone as though married

8a. What is the name of the child who is participating with you in this study? (CSEX)

8b. What is the date of birth of the child who is participating with you in this study? (CDOB)

_____ (month) _____ (date) _____ (year)

8c. Starting with your oldest, please tell me all of the children to whom you have given birth (fathered), including the child participating in this study if applicable: (For each child, ask about the child's sex and birth year. Include the child participating in this study if applicable. Do not include children in utero in total but do include deceased children).

Sex	Year of Birth

RBRTKIDS _____

8d. Are there any children who are not your own but who live in your household? (For each child, ask about the child's sex and birth year. Include the child participating in this study if applicable. Include children over age 18 but do not include children who reside in the home part-time (e.g., weekends)).

Sex	Year of Birth

RTOTKIDS _____
Total # of children living with respondent

10. What is the total number of people living in your household?

11a. How would you describe your own race?

RRACE _____

11b. How would you describe the race of the child who is participating with you in this study?

CRACE _____

- 1 = American Indian/Alaska Native
- 2 = Asian
- 3 = Native Hawaiian or Other Pacific Islander
- 4 = Black or African American
- 5 = White
- 6 = More than one race
- 7 = Other _____

11c. How would you describe your own ethnicity?

RETH _____

- 1 Hispanic or Latino
- 2 Not Hispanic or Latino

11d. How would you describe the ethnicity of the child who is participating with you in this study?

CETH _____

- 1 Hispanic or Latino
- 2 Not Hispanic or Latino

12a. How many years of school do you have credit for altogether? (*Use 1-12 for elementary school through high school; 13-16 for college undergraduate work; and use 17 as the cap for the highest grade in school when the respondent has some post undergr years for GED*). Continue on to next page.

REDUC _____
01-17

12b. What is the highest education degree or certificate you hold? RDEGREE

- 0 None
- 1 Elementary School / Junior High
- 2 GED (General Education Development)
- 3 High School Diploma
- 4 Vocational / Technical Diploma
- 5 Associate Degree
- 6 RN Diploma
- 7 Bachelor Degree

- 8 Master Degree
- 9 Doctorate: MD., Ph.D., J.D., etc.

RHED _____
 Hollingshead education score 1-7

13. During the past week, were you working (either full-time or part-time)? RCURWORK

13a. (If Yes) Mark below for full-time or part-time
 (If No, ask) Which one of these best describes your current situation?

- 1 full-time (35+ hrs)
- 2 part-time
- 3 unemployed or laid off and looking for work
- 4 unemployed or laid off and not looking for work
- 5 retired
- 6 in school
- 7 keeping house/taking care of children
- 8 disabled and not looking for work
- 9 other (specify) _____

(In school (6) takes precedence over keeping house (7). If pregnant and unemployed, code as 7.)

For a current spouse / partner (within 1 yr, ask questions 10-12).

14a. Whom do you consider to be your partner? _____

14b. What relation is this person to you? _____

14c. What is this person's age? SAGE _____

14d. What is this person's gender? S_GENDER

- 0 Male
- 1 Female

14e. What is this person's race? SRACE _____ SETH _____ (Q10 on page 4)

15a. How many grades / years of school does your partner have credit for altogether? (Use 1-12 for elementary school through high school; 13-16 for college undergraduate work; and use 17 as the cap for the highest grade in school when the respondent has some post undergraduate work. Do not add years for GED). SEDUC _____ (01-17)

15b. What is the highest education degree or certificate your partner holds?

- 0 None
- 1 Elementary School / Junior High
- 2 GED (General Education Development)
- 3 High School Diploma
- 4 Vocational / Technical Diploma

- G \$10,000 - \$14,999 or \$834 - \$1,249 per month
- H \$15,000 - \$19,999 or \$1,250 - \$1,666 per month
- I \$20,000 - \$24,999 or \$1,667 - \$2,083 per month
- J \$25,000 - \$34,999 or \$2,084 - \$2,916 per month
- K \$35,000 - \$49,999 or \$2,917 - \$4,167 per month
- L \$50,000 - \$74,999 or \$4,168 - \$6,249 per month
- M \$75,000 - \$99,999 or \$6,250 - \$8,333 per month
- N \$100,000 - \$199,999 or \$8,334 - \$16,666 per month
- O \$200,000 or more or \$16,667 or more per month

Please circle the number corresponding with your answer to the following questions about your health.

20. During the last 12 months (one year), would you say that your general health has been...(HEALTH)

- 1 Excellent
- 2 Good
- 3 Fair
- 4 Poor

Continue on to next page.

21. Would you say that you have been SICKLY a large part of your life? (SICKLY)

- 1 Yes
- 2 Somewhat
- 3 No

22. How well off would you say your family is? (RCURWOF)

- 1 very poor (at times no money for food, clothing, and / or shelter)
- 2 poor (limited money for anything more than the basics)
- 3 lower middle class (able to afford necessities for modern life)
- 4 middle class (own house, meet the bills with some extra)
- 5 upper middle class (own nice home, many luxuries)

23. How satisfied are you with your overall financial situation? (RFINSAT)

- 1 very satisfied
- 2 satisfied
- 3 unsatisfied
- 4 very unsatisfied

24. How satisfied are you with your current income? (RINSAT)

- 1 very satisfied
- 2 satisfied
- 3 unsatisfied
- 4 very unsatisfied

25. How satisfied are you with your material possessions, for example, TV's, household appliances, and other things that your family owns? (RPOSSA)

- 1 very satisfied
- 2 satisfied
- 3 unsatisfied
- 4 very unsatisfied

26. How often do you worry about your family's financial situation? (RFINWOR)

- 1 very often
- 2 often
- 3 seldom
- 4 never

Youth Risk Behaviors (Administered Online via Survey Monkey)

The next eight questions ask about tobacco use.

1. Have you ever tried cigarette smoking, even one or two puffs?
 - A. Yes
 - B. No

2. How much do you think people risk harming themselves (physically or in other ways) if they smoke half a pack of cigarettes or more a day?
 - A. Great risk
 - B. Moderate (Medium) risk
 - C. Slight risk
 - D. No risk

3. Which is the most true for you about smoking cigarettes?
 - A. Never used
 - B. Tried once – twice
 - C. Used three – five times
 - D. Usually use a few times a month
 - E. Usually use a few times a week
 - F. Usually use every day

4. How old were you when you smoked a whole cigarette for the first time?
 - A. I have never smoked a whole cigarette
 - B. 8 years old or younger
 - C. 9 years old
 - D. 10 years old
 - E. 11 years old
 - F. 12 years old
 - G. 13 years old
 - H. 14 years old
 - I. 15 years old
 - J. 16 years old
 - K. 17 years or older

5. During the past 30 days, on how many days did you smoke cigarettes?
 - A. 0 days
 - B. 1 or 2 days
 - C. 3 to 5 days
 - D. 6 to 9 days
 - E. 10 to 19 days
 - F. 20 to 29 days
 - G. All 30 days

6. During the past 30 days, on the days that you smoked, how many cigarettes did you smoke per day?
- A. I did not smoke cigarettes during the past 30 days
 - B. Less than 1 cigarette per day
 - C. 1 cigarette per day
 - D. 2 - 5 cigarettes per day
 - E. 6 - 10 cigarettes per day
 - F. 11-20 cigarettes per day
 - G. More than 20 cigarettes per day
7. Which is the most true for you about using any other tobacco products (other than cigarettes) such as cigars; chewing tobacco, snuff, or dip; pipes; bidis and kreteks; hookah, snus, dissolvable tobacco, and electronic cigarettes?
- A. Never used
 - B. Tried once – twice
 - C. Used three – five times
 - D. Usually use a few times a month
 - E. Usually use a few times a week
 - F. Usually use every day
8. During the past 30 days, on how many days did you use any other tobacco products (other than cigarettes) such as cigars; chewing tobacco, snuff, or dip; pipes; bidis and kreteks; hookah, snus, dissolvable tobacco, and electronic cigarettes?
- A. 0 days
 - B. 1 or 2 days
 - C. 3 to 5 days
 - D. 6 to 9 days
 - E. 10 to 19 days
 - F. 20 to 29 days
 - G. All 30 days
9. How old were you when you tried any other tobacco products (other than cigarettes) for the first time?
- A. I have never tried any other tobacco products (other than cigarettes)
 - B. 8 years old or younger
 - C. 9 years old
 - D. 10 years old
 - E. 11 years old
 - F. 12 years old
 - G. 13 years old
 - H. 14 years old

- I. 15 years old
- J. 16 years old
- K. 17 years or older

The next seven questions ask about drinking alcohol. This includes drinking beer, wine, wine coolers, and liquor such as rum, gin, vodka, or whisky. For these questions, drinking alcohol does not include drinking a few sips of wine for religious purposes.

1. Have you ever had a drink of alcohol, other than a few sips?
 - A. Yes
 - B. No

2. How much do you think people risk harming themselves (physically or in other ways) if they take one or two drinks of an alcoholic beverage (beer, wine, liquor) nearly every day?
 - A. Great risk
 - B. Moderate (Medium) risk
 - C. Slight risk
 - D. No risk

3. Which is the most true for you about using alcohol?
 - A. Never used
 - B. Tried once – twice
 - C. Used three – five times
 - D. Usually use a few times a month
 - E. Usually use a few times a week
 - F. Usually use every day

4. How old were you when you had your first drink of alcohol other than a few sips?
 - A. I have never had a drink of alcohol other than a few sips.
 - B. 8 years old or younger
 - C. 9 years old
 - D. 10 years old
 - E. 11 years old
 - F. 12 years old
 - G. 13 years old
 - H. 14 years old
 - I. 15 years old
 - J. 16 years old
 - K. 17 years or older

5. During the past 30 days, on how many days did you have at least one drink of alcohol?

- A. 0 days
 - B. 1 or 2 days
 - C. 3 to 5 days
 - D. 6 to 9 days
 - E. 10 to 19 days
 - F. 20 to 29 days
 - G. All 30 days
6. How many times did you have 5 or more alcoholic drinks on one occasion in the past 30 days?
- A. Never happened
 - B. Happened once
 - C. Happened twice
 - D. Happened three or more times
7. How many times did you have 5 or more alcoholic drinks on one occasion in the past 6 months?
- A. Never happened
 - B. Happened once – twice
 - C. Happened three – five times
 - D. Happened a few times a month
 - E. Happened a few times a week
 - F. Happened every day

The next five questions ask about marijuana use. Marijuana is also known as grass, pot, or weed.

1. Have you ever used marijuana?
- A. Yes
 - B. No
2. How much do you think people risk harming themselves (physically or in other ways) if they smoke marijuana?
- A. Great risk
 - B. Moderate (Medium) risk
 - C. Slight risk
 - D. No risk
3. Which is the most true for you about using marijuana?
- A. Never used
 - B. Tried once – twice
 - C. Used three – five times
 - D. Usually use a few times a month

- E. Usually use a few times a week
 F. Usually use every day
4. How old were you when you tried marijuana for the first time?
 A. I have never tried marijuana
 B. 8 years old or younger
 C. 9 years old
 D. 10 years old
 E. 11 years old
 F. 12 years old
 G. 13 years old
 H. 14 years old
 I. 15 years old
 J. 16 years old
 K. 17 years or older
5. During the past 30 days, on how many days did you have at least one puff of marijuana?
 A. 0 days
 B. 1 or 2 days
 C. 3 to 5 days
 D. 6 to 9 days
 E. 10 to 19 days
 F. 20 to 29 days
 G. All 30 days
6. On the days that you used marijuana during the past 30 days, how many average size joints did you **usually** smoke each day? (If you typically smoke it via another means, please estimate the number of average size joints).
 _____ joints
7. During the past 30 days, on average, how much marijuana per week do you think you used in ounces?
 ___ (0) I did not use marijuana during the past 30 days
 ___ (1) Less than 1/16th
 ___ (2) 1/16th
 ___ (3) 1/8th
 ___ (4) 1/4th
 ___ (5) 3/8th
 ___ (6) 1/2
 ___ (7) 5/8th
 ___ (8) 3/4th
 ___ (9) 7/8th
 ___ (10) 1
 ___ (11) More than 1

The following questions ask about using drugs other than alcohol, cigarette, and marijuana.

1. Which is the most true for you about using drugs other than marijuana?
 - A. Never used
 - B. Tried once – twice
 - C. Used three – five times
 - D. Usually use a few times a month
 - E. Usually use a few times a week
 - F. Usually use every day

2. During your life, how many times have you used any form of cocaine, including powder, crack, or freebase?
 - A. 0 times
 - B. 1 or 2 times
 - C. 3 - 9 times
 - D. 10-19 times
 - E. 20-39 times
 - F. 40 or more times

3. During the past 30 days, how many times did you use any form of cocaine, including powder, crack, or freebase?
 - A. 0 times
 - B. 1 or 2 times
 - C. 3 - 9 times
 - D. 10-19 times
 - E. 20-39 times
 - F. 40 or more times

4. During your life, how many times have you sniffed glue, breathed the contents of aerosol spray cans, or inhaled any paints or sprays to get high?
 - A. 0 times
 - B. 1 or 2 times
 - C. 3 - 9 times
 - D. 10-19 times
 - E. 20-39 times
 - F. 40 or more times

5. During the past 30 days, how many times have you sniffed glue, breathed the contents of aerosol spray cans, or inhaled any paints or sprays to get high?
 - A. 0 times
 - B. 1 or 2 times
 - C. 3 - 9 times
 - D. 10-19 times
 - E. 20-39 times

- F. 40 or more times
- 6. During your life how many times have you used heroin (also called smack, junk, or China White)?
 - A. 0 times
 - B. 1 or 2 times
 - C. 3 - 9 times
 - D. 10-19 times
 - E. 20-39 times
 - F. 40 or more times
- 7. During your life, how many times have you used methamphetamines (also called speed, crystal, crank, or ice)?
 - A. 0 times
 - B. 1 or 2 times
 - C. 3 to 9 times
 - D. 10-19 times
 - E. 20-39 times
 - F. 40 or more times
- 8. During your life, how many times have you used ecstasy (also called MDMA)?
 - A. 0 times
 - B. 1 or 2 times
 - C. 3 - 9 times
 - D. 10-19 times
 - E. 20-39 times
 - F. 40 or more times
- 9. During your life, how many times have you taken steroid pills or shots without a doctor's prescription?
 - A. 0 times
 - B. 1 or 2 times
 - C. 3 - 9 times
 - D. 10-19 times
 - E. 20-39 times
 - F. 40 or more times
- 10. During your life, how many times have you used a needle to inject any illegal drug into your body?
 - A. 0 times
 - B. 1 or 2 times
 - C. 3 - 9 times
 - D. 10-19 times
 - E. 20-39 times
 - F. 40 or more times

11. During your life, how many times have you taken over-the-counter drugs to get high?
- A. 0 times
 - B. 1 or 2 times
 - C. 3 - 9 times
 - D. 10-19 times
 - E. 20-39 times
 - F. 40 or more times
12. During your life how many times have you taken prescription drugs to get high?
- A. 0 times
 - B. 1 or 2 times
 - C. 3 - 9 times
 - D. 10-19 times
 - E. 20-39 times
 - F. 40 or more times

Here are some questions about things that could happen to adolescents.

1. Suppose you were with a group of friends and one of them offered you a *cigarette*. How likely is it that you would *take it and try it*?
- A. not at all likely
 - B. somewhat unlikely
 - C. somewhat likely
 - D. very likely
2. Suppose you were with a group of friends and one of them offered you a *cigarette*. How likely is it that you would *tell them no, thanks*?
- A. not at all likely
 - B. somewhat unlikely
 - C. somewhat likely
 - D. very likely
3. Suppose you were with a group of friends and one of them offered you a *cigarette*. How likely is it that you would *leave the situation*?
- A. not at all likely
 - B. somewhat unlikely
 - C. somewhat likely
 - D. very likely
4. Suppose you were with a group of friends and one of them offered you some *alcohol*. How likely is it that you would *take it and try it*?
- A. not at all likely
 - B. somewhat unlikely
 - C. somewhat likely
 - D. very likely

5. Suppose you were with a group of friends and one of them offered you some *alcohol*. How likely is it that you would *tell them no, thanks*?
- A. not at all likely
 - B. somewhat unlikely
 - C. somewhat likely
 - D. very likely
6. Suppose you were with a group of friends and one of them offered you some *alcohol*. How likely is it that you would *leave the situation*?
- A. not at all likely
 - B. somewhat unlikely
 - C. somewhat likely
 - D. very likely
7. Suppose you were with a group of friends and one of them offered you some *marijuana*. How likely is it that you would *take it and try it*?
- A. not at all likely
 - B. somewhat unlikely
 - C. somewhat likely
 - D. very likely
8. Suppose you were with a group of friends and one of them offered you some *marijuana*. How likely is it that you would *tell them no, thanks*?
- A. not at all likely
 - B. somewhat unlikely
 - C. somewhat likely
 - D. very likely
9. Suppose you were with a group of friends and one of them offered you some *marijuana*. How likely is it that you would *leave the situation*?
- A. not at all likely
 - B. somewhat unlikely
 - C. somewhat likely
 - D. very likely

Appendix B: Confirmatory Factor Analysis Factors

CFA was used to compute socioeconomic status factor scores at each time point using a variety of candidate variables. The four factors demonstrating significant loadings from each of the indicators across all three time points and good model fit with CFI values greater than .95 and RMSEA values less than .08 are listed below.

Factor 1: Primary caregiver and spouse composite years of education, annual income

Factor 2: Primary caregiver and spouse composite years of education, relative income

Factor 3: Primary caregiver and spouse composite years of education, income-to-needs ratio

Factor 4: Primary caregiver and spouse composite years of education, relative income, primary caregiver and adolescent composite reported household chaos

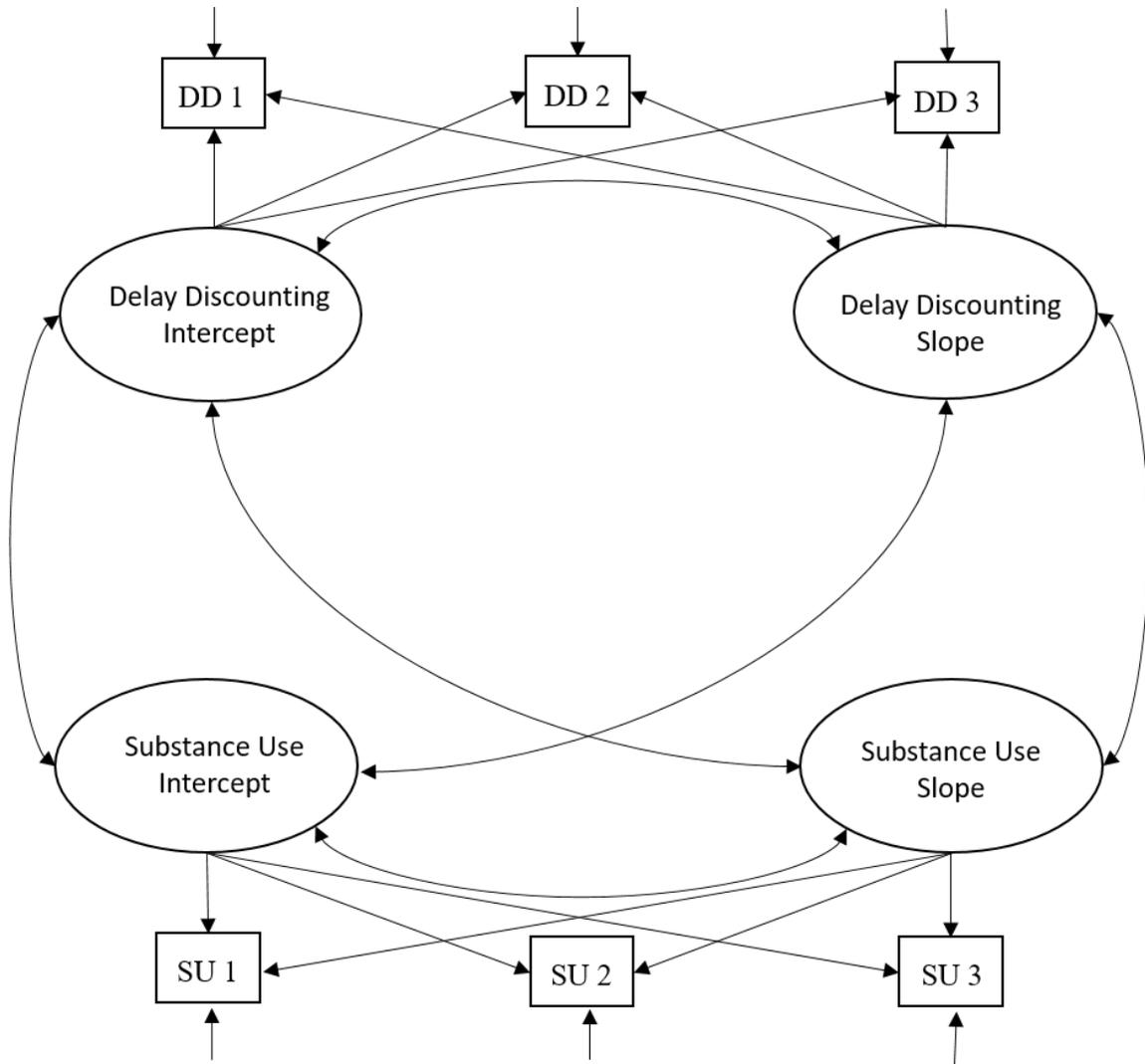


Figure 1. An unconditional parallel process model with between-process correlations. *SU* = substance use; *DD* = delay discounting; delay discounting intercept = initial levels of delay discounting; delay discounting slope = growth rate factor of delay discounting; substance use intercept = initial levels of substance use; substance use slope = growth rate factor of substance use.

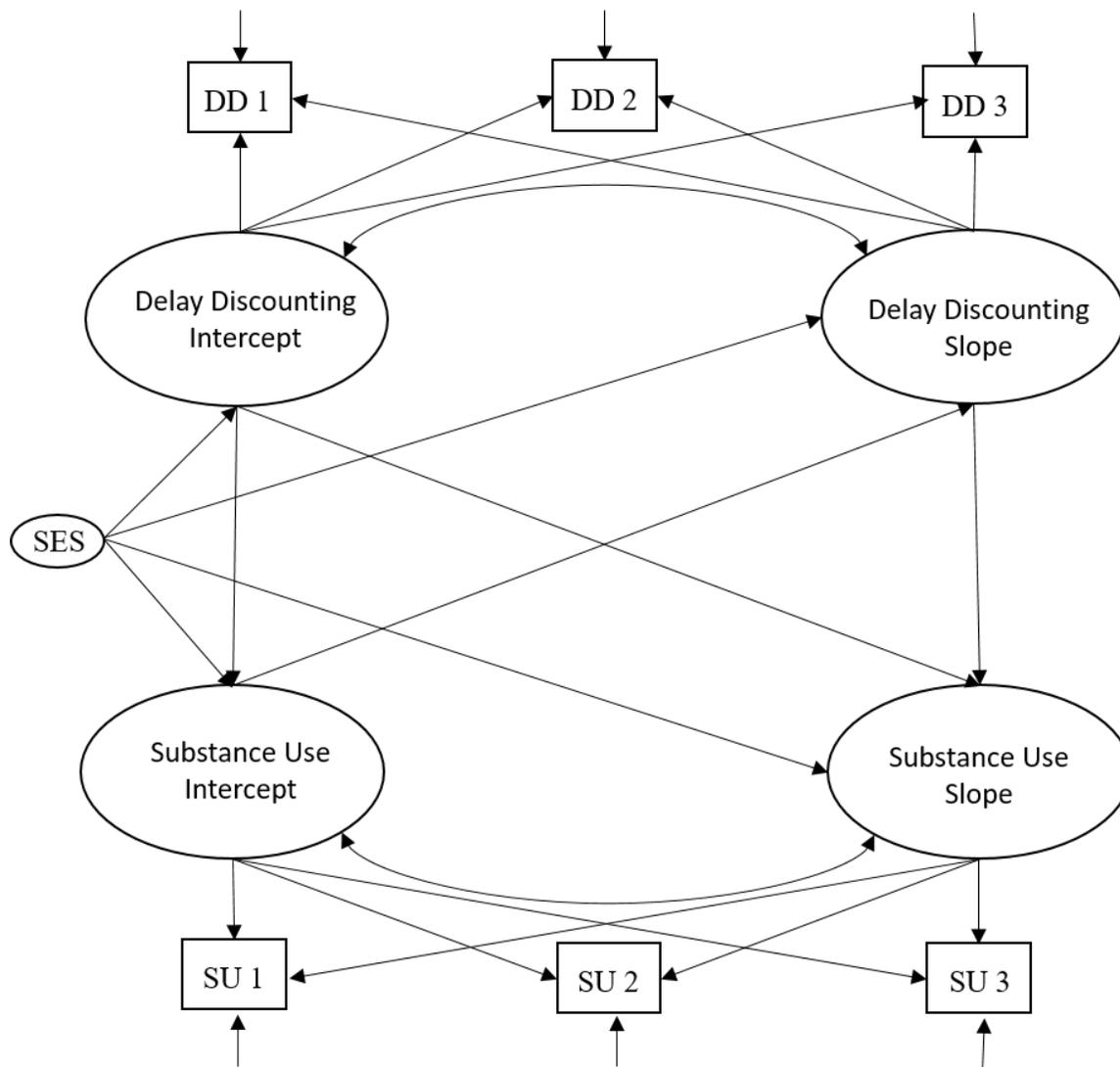


Figure 2. A parallel process GCM model with SES as a time-invariant covariate (TIC) and between-process regressions. *SES* = Socioeconomic status; *SU* = substance use; *DD* = delay discounting; delay discounting intercept = initial levels of mediator; delay discounting slope = growth rate factor of mediator; substance use intercept = initial levels of outcome; substance use slope = growth rate factor of outcome.

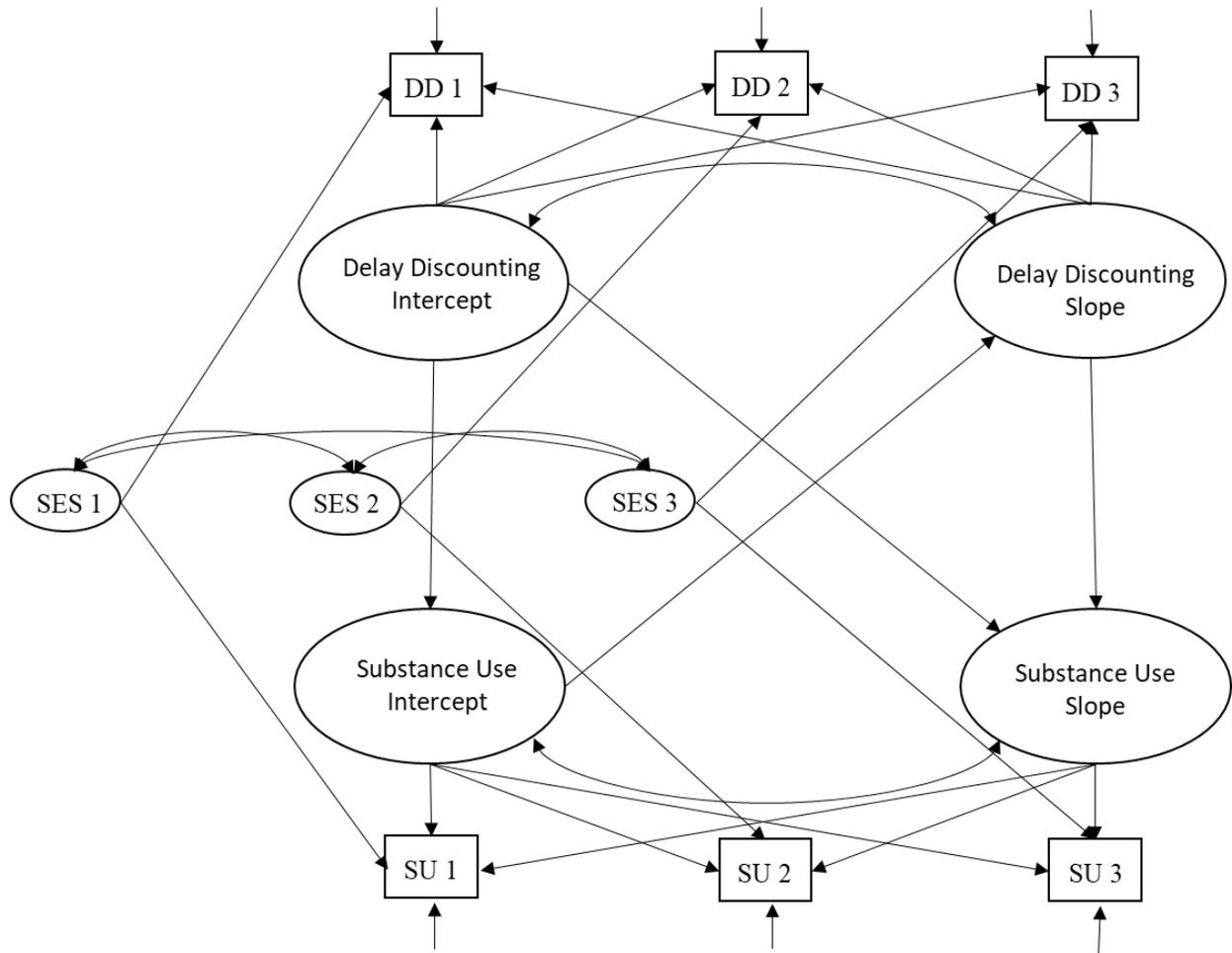


Figure 3. A parallel process model for mediation with SES as a time-varying covariate (TVC) and between-process regressions. *SES* = Socioeconomic status; *SU* = substance use; *DD* = delay discounting; delay discounting intercept = initial levels of mediator; delay discounting slope = growth rate factor of mediator; substance use intercept = initial levels of outcome; substance use slope = growth rate factor of outcome.

Table 1

Socioeconomic Status, Delay Discounting, and Substance Use Descriptive Statistics

Variables	Min	Max	Time 1		Time 2		Time 3	
			M	SD	M	SD	M	SD
K100	<.00	17.11	.45	2.17	.09	0.32	.05	0.17
K100_log	-5.89	1.23	-1.91	1.27	-2.26	1.27	-2.54	1.34
Cig_Freq	1.00	5.00	1.18	0.54	1.28	0.61	1.36	0.71
Alc_Freq	1.00	4.00	1.40	0.65	1.58	0.87	1.91	1.02
Mar_Freq	1.00	6.00	1.15	0.57	1.29	0.86	1.61	1.16
PolySU_Freq	1.00	4.00	1.24	0.48	1.38	0.62	1.63	0.74
Cig_Onset	1.00	10.00	-	-	-	-	1.77	1.93
Alc_Onset	1.00	8.00	-	-	-	-	2.33	1.87
Mar_Onset	1.00	9.00	-	-	-	-	1.72	1.00
PolySU_Onset	1.00	8.33	-	-	-	-	1.94	1.40
PC_Ed	9.00	17.00	14.71	1.95	14.67	1.92	14.85	1.82
SP_Ed	2.00	17.00	14.30	2.19	14.36	2.19	14.01	2.51
PCSPEd	7.00	17.00	14.47	1.82	14.19	1.85	14.52	1.88
Income	1.00	15.00	10.97	2.58	10.94	2.95	11.34	2.82
SES_FScore	-4.00	1.39	0.00	1.00	0.00	1.00	0.00	1.00

Note. K100 = Raw delay discounting k parameter. K100_log = log transformed delay

discounting k parameter. Cig_Freq = cigarette use frequency. Cig_Onset = cigarette use onset.

Alc_Freq = alcohol use frequency. Alc_Onset = alcohol use onset. Mar_Freq = marijuana use

frequency. Mar_Onset = marijuana use onset. PolySU_Freq = composite cigarette, alcohol,

marijuana polysubstance use frequency score. PolySU_Onset = composite cigarette, alcohol,

marijuana polysubstance use onset score. PC_Ed = Primary caregiver years of education (capped

at 17). SP_Ed = Spouse years of education (capped at 17). PCSPEd = Composite primary

caregiver and spouse years of education. Income = Annual combined household income before

taxes. SES_FScore = SES factor score comprised of primary caregiver and spouse education

composite and annual combined household income.

Table 2

*Chi-Square Difference Test Comparisons of Univariate No Growth, Linear Growth, and Latent Growth**Models*

Model label	χ^2	<i>df</i>	<i>p</i>	SB	CFI	RMSEA	Comparison	<i>T</i>	Δdf	<i>p(d)</i>
<i>Delay Discounting</i>										
a. No Growth	45.57	6	.00	.98	.49	.20				
b. Linear Growth	5.33	3	.15	1.05	.97	.07	a vs b	43.02	3	< .00001
c. Latent Growth	1.58	2	.00	.86	1.00	.00	b vs c	2.96	1	.09
<i>Cigarette Use Frequency</i>										
a. No Growth	19.78	6	.00	2.55	.88	.16				
b. Linear Growth	.85	3	.84	.76	1.00	.00	a vs b	12.89	3	.005
c. Latent Growth	.43	2	.81	.67	1.00	.00	b vs c	.32	1	.57
<i>Alcohol Use Frequency</i>										
a. No Growth	82.84	6	.00	1.28	.15	.28				
b. Linear Growth	5.94	3	.11	1.37	.97	.08	a vs b	81.96	3	<.0001
c. Latent Growth	11.79	2	.00	.67	.89	.17	b vs c	2.94	1	.09
<i>Marijuana Use Frequency</i>										
a. No Growth	65.34	6	.00	2.90	.00	.24				
b. Linear Growth	11.14	3	.01	2.78	.85	.13	a vs b	52.50	3	<.00001
c. Latent Growth	30.35	2	.00	.76	.46	.29	b vs c	1.14	1	.29
<i>Polysubstance Use Frequency</i>										
a. No Growth	71.64	6	.00	2.17	.26	.26				
b. Linear Growth	6.15	3	.10	1.43	.97	.08	a vs b	50.47	3	<.00001
c. Latent Growth	11.25	2	.00	.73	.90	.17	b vs c	.19	1	.66

Note. SB = Satorra-Bentler adjusted chi-square value; CFI = comparative-fit index; RMSEA = root mean square error of approximation; *T* = distributed chi-square with difference in *df*; Δdf = difference in *df*; *p(d)* = probability of the difference tests. Best-fitting models are in bold face. Results from the cigarette use frequency latent model are not reported due to non-convergence.

Table 3

Results from the Growth Curve Models for the Associations among SES, Delay Discounting, and Cigarette Use

Frequency

Univariate Linear GCM: Latent Means Analysis

<i>Means</i>	<u>DD Est. (SE)</u>	<i>p</i>	<u>CigSU Est. (SE)</u>	<i>p</i>
Int	-1.92 (.10)	.00	1.18 (.04)	.00
Slope	-.29 (.06)	.00	.10 (.02)	.00
<i>Variances</i>				
Int	.97 (.17)	.00	.19 (.11)	.08
Slope	.22 (.09)	.01	.03 (.02)	.18
<i>Within Process Correlations</i>				
Int ↔ Slope	-.30 (.15)	.05	.40 (.58)	.49

Model 1: Unconditional Parallel Process GCM

<i>Between-Process Correlations</i>	<u>DD ↔ CigSU</u>	<i>p</i>
DD _{Int} ↔ CigSU _{Int}	.19 (.12)	.09
DD _{Int} ↔ CigSU _{Slope}	.03 (.17)	.88
CigSU _{Int} ↔ DD _{Slope}	-.10 (.14)	.46
DD _{Slope} ↔ CigSU _{Slope}	-.14 (.18)	.45

Model 2: Conditional Parallel Process GCM with Between-Process Regressions and SES TICs

<i>Time-Invariant Covariate Regression Estimates</i>	<u>DD Est. (SE)</u>	<i>p</i>	<u>CigSU Est. (SE)</u>	<i>p</i>
SES				
→Int	-.23 (.11)	.03	-.21 (.08)	.01
→Slope	.05 (.14)	.70	-.13 (.12)	.28

Between-Process Regressions

	<u>Est. (SE)</u>	<i>p</i>
DD _{Int} → CigSU _{Int}	.14 (.12)	.25
DD _{Int} → CigSU _{Slope}	-.05 (.17)	.78
CigSU _{Int} → DD _{Slope}	-.05 (.14)	.70
DD _{Slope} → CigSU _{Slope}	-.12 (.18)	.53

Model 3: Conditional Parallel Process GCM with Between-Process Regressions and SES TVCs

<i>Time-Varying Covariate Regression Estimates</i>	<u>DD Est. (SE)</u>	<i>p</i>	<u>CigSU Est. (SE)</u>	<i>p</i>
SES T1				
→T1	-.05 (.23)	.84	-.13 (.07)	.07
SES T2				
→T2	-.05 (.23)	.84	-.11 (.06)	.09
SES T3				

→T3	-04 (.21)	.84	-09 (.05)	.09
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Between-Process Regression Estimates

	<u>Est. (SE)</u>	<u>p</u>
DD _{Int} → CigSU _{Int}	.16 (.14)	.23
DD _{Int} → CigSU _{Slope}	-.02 (.21)	.94
CigSU _{Int} → DD _{Slope}	-.07 (.15)	.66
DD _{Slope} → CigSU _{Slope}	-.11 (.20)	.57

Notes. Int = Intercept; DD = Delay Discounting; CigSU = Cigarette use frequency. Unstandardized estimates used for means and variances. Standardized estimates used for correlations and coefficients. Bold faced values are significant at the $p < .05$ level. Positive and significant intercept, slope associations indicate higher initial status leads to increased growth. Negative significant associations indicate higher initial status leads to decreases in growth.

Table 4

Results from the Growth Curve Models for the Associations among SES, Delay Discounting, and Alcohol Use

Frequency

Univariate Linear GCM: Latent Means Analysis

<i>Means</i>	<u>DD Est. (SE)</u>	<u><i>p</i></u>	<u>AlcSU Est. (SE)</u>	<u><i>p</i></u>
Int	-1.92 (.10)	.00	1.38 (.05)	.00
Slope	-.29 (.06)	.00	.26 (.04)	.00
<i>Variances</i>				
Int	.97 (.17)	.00	.21 (.08)	.01
Slope	.22 (.09)	.01	.08 (.03)	.01
<i>Within Process Correlations</i>				
Int ↔ Slope	-.30 (.15)	.05	.55 (.37)	.14

Model 1: Unconditional Parallel Process GCM

<i>Between-Process Correlations</i>	<u>DD ↔ AlcSU</u>	<u><i>p</i></u>
DD _{Int} ↔ AlcSU _{Int}	.36 (.13)	.01
DD _{Int} ↔ AlcSU _{Slope}	.14 (.17)	.43
AlcSU _{Int} ↔ DD _{Slope}	-.43 (.22)	.045
DD _{Slope} ↔ AlcSU _{Slope}	.39 (.21)	.07

Model 2: Conditional Parallel Process GCM with Between-Process Regressions and SES TICs

<i>Time-Invariant Covariate Regression Estimates</i>	<u>DD Est. (SE)</u>	<u><i>p</i></u>	<u>AlcSU Est. (SE)</u>	<u><i>p</i></u>
SES				
→Int	-.24 (.11)	.03	-.21 (.08)	.01
→Slope	.05 (.14)	.70	-.13 (.12)	.28
<i>Between-Process Regressions</i>	<u>Est. (SE)</u>	<u><i>p</i></u>		
DD _{Int} → AlcSU _{Int}	.14 (.12)	.25		
DD _{Int} → AlcSU _{Slope}	-.05 (.17)	.78		
AlcSU _{Int} → DD _{Slope}	-.05 (.14)	.70		
DD _{Slope} → AlcSU _{Slope}	-.12 (.18)	.53		

Model 3: Conditional Parallel Process GCM with Between-Process Regressions and SES TVCs

<i>Time-Varying Covariate Regression Estimates</i>	<u>DD Est. (SE)</u>	<u><i>p</i></u>	<u>AlcSU Est. (SE)</u>	<u><i>p</i></u>
SES T1				
→T1	-.07 (.17)	.67	-.13 (.08)	.12
SES T2				
→T2	-.07 (.17)	.68	-.09 (.06)	.12
SES T3				
→T3	-.06 (.15)	.68	-.08 (.05)	.12

<i>Between-Process Regression Estimates</i>	<u>Est. (SE)</u>	<u>p</u>
DD _{Int} → AlcSU _{Int}	.22 (.11)	.046
DD _{Int} → AlcSU _{Slope}	.23 (.14)	.11
AlcSU _{Int} → DD _{Slope}	-.28 (.16)	.09
DD _{Slope} → AlcSU _{Slope}	.38 (.21)	.07

Notes. Int = Intercept; DD = Delay Discounting; AlcSU = Alcohol use frequency. Unstandardized estimates used for means and variances. Standardized estimates used for correlations and coefficients. Bold faced values are significant at the $p < .05$ level. Positive and significant intercept, slope associations indicate higher initial status leads to increased growth. Negative significant associations indicate higher initial status leads to decreases in growth.

Table 5

Results from the Growth Curve Models for the Associations among SES, Delay Discounting, and Marijuana Use

Frequency

Univariate Linear GCM: Latent Means Analysis

<i>Means</i>	<u>DD Est. (SE)</u>	<u><i>p</i></u>	<u>MarSU Est. (SE)</u>	<u><i>p</i></u>
Int	-1.92 (.10)	.00	1.12 (.05)	.00
Slope	-.29 (.06)	.00	.25 (.04)	.00
<i>Variances</i>				
Int	.97 (.17)	.00	.19 (.11)	.08
Slope	.22 (.09)	.01	.20 (.05)	.00
<i>Within Process Correlations</i>				
Int ↔ Slope	-.30 (.15)	.05	.39 (.25)	.13

Model 1: Unconditional Parallel Process GCM

<i>Between-Process Correlations</i>	<u>DD ↔ MarSU</u>	<u><i>p</i></u>
DD _{Int} ↔ MarSU _{Int}	-.02 (.14)	.90
DD _{Int} ↔ MarSU _{Slope}	.16 (.11)	.15
MarSU _{Int} ↔ DD _{Slope}	.01 (.29)	.98
DD _{Slope} ↔ MarSU _{Slope}	.13 (.14)	.34

Model 2: Conditional Parallel Process GCM with Between-Process Regressions and SES TICs

<i>Time-Invariant Covariate Regression Estimates</i>	<u>DD Est. (SE)</u>	<u><i>p</i></u>	<u>MarSU Est. (SE)</u>	<u><i>p</i></u>
SES				
→Int	-.14 (.18)	.44	-.03 (.14)	.84
→Slope	.04 (.23)	.87	-.18 (.14)	.21
Gender (0 = male, 1 = female)				
→Int	-.18 (.17)	.32	.10 (.15)	.51
→Slope	.14 (.22)	.52	.16 (.15)	.27
Race (0 = white, 1 = other)				
→Int	.03 (.11)	.80	.11 (.08)	.18
→Slope	-.19 (.13)	.15	.06 (.09)	.48
<i>Between-Process Regressions</i>	<u>Est. (SE)</u>	<u><i>p</i></u>		
DD _{Int} → MarSU _{Int}	-.05 (.15)	.75		
DD _{Int} → MarSU _{Slope}	.22 (.10)	.03		
MarSU _{Int} → DD _{Slope}	.03 (.25)	.89		
DD _{Slope} → MarSU _{Slope}	.21 (.13)	.10		

Model 3: Conditional Parallel Process GCM with Between-Process Regressions and SES TVCs

<i>Time-Varying Covariate Regression Estimates</i>	<u>DD Est. (SE)</u>	<u><i>p</i></u>	<u>MarSU Est. (SE)</u>	<u><i>p</i></u>
--	---------------------	-----------------	------------------------	-----------------

SES T1				
→T1				
SES T2				
→T2				
SES T3				
→T3				
Gender (0 = male, 1 = female)				
→Int				
→Slope				
Race (0 = white, 1 = other)				
→Int				
→Slope				

Between-Process Regression Estimates

	<u>Est. (SE)</u>	<u>p</u>
DD _{Int} → MarSU _{Int}	-.04 (.16)	.78
DD _{Int} → MarSU _{Slope}	.22 (.14)	.12
MarSU _{Int} → DD _{Slope}	.00 (.23)	.99
DD _{Slope} → MarSU _{Slope}	.07 (.16)	.68

Notes. Marijuana residual variance at Time 1 was fixed to 0 due to small, negative, non-significant value. Int = Intercept; DD = Delay Discounting; MarSU = Marijuana use frequency. Unstandardized estimates used for means and variances. Standardized estimates used for correlations and coefficients. Bold faced values are significant at the $p < .05$ level. Positive and significant intercept, slope associations indicate higher initial status leads to increased growth. Negative significant associations indicate higher initial status leads to decreases in growth.

Table 6

Results from the Growth Curve Models for the Associations among SES, Delay Discounting, and Polysubstance

Use Frequency

Univariate Linear GCM: Latent Means Analysis

<i>Means</i>	<u>DD Est. (SE)</u>	<u><i>p</i></u>	<u>PolySU Est. (SE)</u>	<u><i>p</i></u>
Int	-1.92 (.10)	.00	1.23 (.04)	.00
Slope	-.29 (.06)	.00	.20 (.02)	.00
<i>Variances</i>				
Int	.97 (.17)	.00	.16 (.06)	.004
Slope	.22 (.09)	.01	.04 (.02)	.01
<i>Within Process Correlations</i>				
Int ↔ Slope	-.30 (.15)	.05	.51 (.31)	.10

Model 1: Unconditional Parallel Process GCM

<i>Between-Process Correlations</i>	<u>DD ↔ PolySU</u>	<u><i>p</i></u>
DD _{Int} ↔ PolySU _{Int}	.17 (.12)	.16
DD _{Int} ↔ PolySU _{Slope}	.19 (.12)	.12
PolySU _{Int} ↔ DD _{Slope}	-.23 (.18)	.20
DD _{Slope} ↔ PolySU _{Slope}	.13 (.16)	.41

Model 2: Conditional Parallel Process GCM with Between-Process Regressions and SES TICs

<i>Time-Invariant Covariate Regression Estimates</i>	<u>DD Est. (SE)</u>	<u><i>p</i></u>	<u>PolySU Est. (SE)</u>	<u><i>p</i></u>
SES				
→Int	-.14 (.18)	.45	-.11 (.13)	.42
→Slope	.06 (.22)	.80	-.34 (.17)	.045
Gender (0 = male, 1 = female)				
→Int	-.16 (.17)	.36	-.13 (.15)	.37
→Slope	-.01 (.22)	.98	.46 (.18)	.01

Between-Process Regressions

	<u>Est. (SE)</u>	<u><i>p</i></u>
DD _{Int} → PolySU _{Int}	.12 (.13)	.35
DD _{Int} → PolySU _{Slope}	.31 (.13)	.02
PolySU _{Int} → DD _{Slope}	-.19 (.18)	.26
DD _{Slope} → PolySU _{Slope}	.32 (.19)	.09

Model 3: Conditional Parallel Process GCM with Between-Process Regressions and SES TVCs

<i>Time-Varying Covariate Regression Estimates</i>	<u>DD Est. (SE)</u>	<u><i>p</i></u>	<u>PolySU Est. (SE)</u>	<u><i>p</i></u>
SES T1				
→T1	-.09 (.11)	.40	-.01 (.09)	.94
SES T2				

→T2	-0.09 (.11)	.40	-0.01 (.07)	.94
SES T3				
→T3	-0.08 (.10)	.40	-0.00 (.05)	.94
Gender (0 = male, 1 = female)				
→Int	-0.18 (.15)	.23	-0.12 (.08)	.15
→Slope	.04 (.14)	.77	.16 (.11)	.17

Between-Process Regression Estimates

	<u>Est. (SE)</u>	<u>p</u>
DD _{Int} → PolySU _{Int}	.12 (.12)	.34
DD _{Int} → PolySU _{Slope}	.32 (.13)	.01
PolySU _{Int} → DD _{Slope}	-.19 (.17)	.26
DD _{Slope} → PolySU _{Slope}	.32 (.19)	.10

Notes. Int = Intercept; DD = Delay Discounting; PolySU = Polysubstance use composite of cigarette, alcohol, and marijuana use frequency. Unstandardized estimates used for means and variances. Standardized estimates used for correlations and coefficients. Bold faced values are significant at the $p < .05$ level. Positive and significant intercept, slope associations indicate higher initial status leads to increased growth. Negative significant associations indicate higher initial status leads to decreases in growth.

Table 7

*Satorra-Bentler Scaled Chi-Square Difference Test Comparisons of the Effects of SES on Delay**Discounting and Substance Use Frequency*

Model label	χ^2	<i>df</i>	<i>p</i>	SB	CFI	RMSEA	Comparison	<i>T</i>	Δdf	<i>p(d)</i>
<i>SES, Delay Discounting and Cigarette Use Frequency</i>										
a. Equal SES	28.37	26	.33	1.00	.97	.05				
b. No Equality Constraints	25.78	22	.26	1.04	.98	.03	a vs. b	.96	4	.74
<i>SES, Delay Discounting and Alcohol Use Frequency</i>										
a. Equal SES	35.31	24	.06	.87	.96	.05				
b. No Equality Constraints	30.39	20	.06	.84	.96	.06	a vs. b	5.09	4	.28
<i>SES, Delay Discounting and Marijuana Use Frequency</i>										
a. Equal SES	56.89	30	.002	.81	.92	.07				
b. No Equality Constraints	54.21	26	.001	.81	.91	.08	a vs. b	2.68	4	.61
<i>SES, Delay Discounting and Polysubstance Use Frequency</i>										
a. Equal SES	43.81	31	.06	.95	.96	.05				
b. No Equality Constraints	40.76	27	.04	.95	.96	.06	a vs. b	3.05	4	.55

Note. SB = Satorra-Bentler adjusted chi-square value; CFI = comparative-fit index; RMSEA = root mean square error of approximation; *T* = distributed chi-square with difference in *df*; Δdf = difference in *df*; *p(d)* = probability of the difference tests. Best-fitting models are in bold face.

Table 8

Results from the Growth Curve Models for the Associations among SES, Delay Discounting, and Substance Use Onset

<u>Unconditional GCM</u>								
<i>Between-Process Correlations</i>	<u>CigOn Est.</u> <u>(SE)</u>	<i>p</i>	<u>AlcOn Est.</u> <u>(SE)</u>	<i>p</i>	<u>MarOn Est.</u> <u>(SE)</u>	<i>p</i>	<u>PolyOn Est.</u> <u>(SE)</u>	<i>p</i>
DD _{Int} ↔ DD _{Slope}	-.31 (.15)	.04	-.30 (.15)	.05	-.31 (.15)	.04	-.31 (.15)	.04
<i>Between-Process Regression Estimates</i>	<u>DD, CigOn</u> <u>Est. (SE)</u>	<i>p</i>	<u>DD, AlcOn</u> <u>Est. (SE)</u>	<i>p</i>	<u>DD, MarOn</u> <u>Est. (SE)</u>	<i>p</i>	<u>DD, PolyOn</u> <u>Est. (SE)</u>	<i>p</i>
DD _{Int} → SUOnset	.14 (.12)	.23	.25 (.10)	.01	.22 (.10)	.03	.23 (.10)	.02
DD _{Slope} → SUOnset	-.17 (.15)	.25	.00 (.13)	.98	-.07 (.18)	.69	-.10 (.15)	.51
<u>Conditional GCM</u>								
<i>Time-Invariant Covariate Regression Estimates</i>	<u>DD, CigOn</u> <u>Est. (SE)</u>	<i>p</i>	<u>DD, AlcOn</u> <u>Est. (SE)</u>	<i>p</i>	<u>DD, MarOn</u> <u>Est. (SE)</u>	<i>p</i>	<u>DD, PolyOn</u> <u>Est. (SE)</u>	<i>p</i>
SES								
→ DD _{Int}	-.25 (.11)	.02	-.26 (.12)	.03	-.23 (.11)	.04	-.26 (.12)	.03
→ DD _{Slope}	.04 (.12)	.72	.09 (.11)	.45	.04 (.12)	.78	-.16 (.15)	.29
→ SUOnset	-.28 (.08)	.00	-.15 (.09)	.09	-.17 (.08)	.04	-.18 (.09)	.04
Gender (0 = male, 1 = female)								
→ DD _{Int}	.02 (.10)	.82	-	-	.03 (.10)	.79	.04 (.11)	.72
→ DD _{Slope}	-.22 (.13)	.10	-	-	-.22 (.14)	.10	-.23 (.13)	.08
→ SUOnset	-.13 (.10)	.19	-	-	-.14 (.09)	.13	-.16 (.09)	.08
<i>Between-Process Regression Estimates</i>	<u>DD, CigOn</u> <u>Est. (SE)</u>	<i>p</i>	<u>DD, AlcOn</u> <u>Est. (SE)</u>	<i>p</i>	<u>DD, MarOn</u> <u>Est. (SE)</u>	<i>p</i>	<u>DD, PolyOn</u> <u>Est. (SE)</u>	<i>p</i>
DD _{Int} → SUOnset	.06 (.12)	.59	.24 (.10)	.02	.18 (.10)	.07	.26 (.12)	.03
DD _{Slope} → SUOnset	-.18 (.14)	.22	.00 (.13)	.98	-.05 (.18)	.78	-.16 (.15)	.29

Note. Int = Intercept; DD = Delay Discounting; SU = Substance Use; CigOn = Cigarette Use Onset; AlcOn = Alcohol Use Onset; MarOn = Marijuana Use Onset; PolyOn = Average Cigarette, Alcohol, and Marijuana Use Onset. Standardized estimates are reported. Bold faced values are significant. Greater onset score = earlier age of onset.