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Declining Alternative Reinforcers Link Depression to Young Adult Smoking

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Abstract

Aims—Young adulthood represents a period of continued smoking progression and the establishment of regular and long-term smoking practices. Our understanding of the psychological processes that facilitate and solidify regular smoking patterns in this developmental period is limited. We sought to evaluate the role of depression symptoms in young adult smoking uptake and to evaluate whether non-smoking related alternative reinforcers was a mechanism by which depression symptoms influence smoking.

Participants—The sample was composed of 834 young adults who participated in a longitudinal study of smoking adoption (age 18 – 22 years old).

Design and measurements—In this prospective cohort study, smoking, depression, alternative reinforcers and several covariates were measured annually via telephone from emerging adulthood (age 18) to young adulthood (age 22).

Findings—Results of a parallel processes latent growth curve model showed that depression symptoms level (baseline age 18) had a significant negative effect on substitute alternative reinforcers trend ($\beta = -.01$, $z = -3.17$, $p = .002$) and that substitute reinforcers trend had a significant negative effect on smoking trend ($\beta = -.62$, $z = -2.99$, $p = .003$). An assessment of indirect effects revealed that depression symptoms level had a significant positive indirect effect on smoking trend through substitute alternative reinforcers trend ($B = .01$, $z = 2.09$, $p = .04$, 99% CI = .001, .02), such that greater depression symptoms at baseline predicted decreases in substitute reinforcers across time which in turn predicted increases in smoking uptake/rate from emerging to young adulthood.

Conclusions—Depression symptoms in emerging adulthood indirectly influence smoking and mitigating declines and/or promoting greater alternative reinforcers to smoking may prevent smoking uptake and further increases in smoking rate among young adults.

Keywords

Young adult smoking; depression symptoms; alternative reinforcers

INTRODUCTION

Smoking is the greatest source of preventable morbidity and mortality in the United States (1). About 26% of young adults in the U.S. smoke cigarettes (2), which is the highest prevalence rate for any adult age group. Young adulthood represents a period of continued smoking progression and the establishment of regular and long-term smoking practices (3,4). For example, as many as 37% of never smokers initiate smoking, 25% of experimenters progress in their smoking, and 16% became regular smokers in emerging and

young adulthood (3,5). As such, this period presents a unique opportunity to intervene upon smoking behavior to prevent lifelong smoking and the associated morbidity and mortality.

Despite the public health significance of young adults smoking, our understanding of the psychological processes that facilitate further smoking and solidify regular smoking patterns in this developmental period is limited. Depression, clinical episodes and subclinical symptoms, appears to be an important risk factor for young adult smoking. A history of depression predicts the onset of daily smoking and progression to nicotine dependence (6,7). Elevated depression symptoms in late adolescence have been shown to predict the transition from nonsmoking to regular smoking in emerging adulthood (8).

Although the literature supports a relationship between depression and smoking in young adulthood, the mechanisms explaining this comorbidity have gone largely unexplored. Depression is associated with a decline in pleasant activities or alternative reinforcers (9–11). Typically, behavioral treatment for depression includes a component to increase the frequency of reinforcing activities (12), based on behavioral theory that postulates that depressed mood prompts a withdrawal from pleasant activities that then exacerbates depression (13).

The clinical relevance of alternative reinforcers spans the substance use continuum (14). For example, a lower level of alternative reinforcers has been shown to predict adolescent smoking progression, young adult smoking status, and abstinence from other substances of abuse (15–18). As alternative reinforcers decline in those with heightened depression symptoms, the reinforcing value smoking may increase as smoking may be an easy way to increase the level of pleasurable experiences (19,20). Cross-sectional data suggest that expectations that smoking will provide pleasure or reward may account for the relationship between a history of depression and regular smoking in young adults (21). Indeed, cigarette smoking has disproportionate reward value for depressed smokers compared to nondepressed smokers (22). Nicotine may also enhance the reward from those alternatives that are available in the environment. Animal models suggest that nicotine potentiates reward from drug and nondrug reinforcers by increasing the sensitivity of brain reward systems (23).

Based on the literature, it is plausible that depression symptoms contribute to a decline in alternative reinforcers, which in turn, increase the likelihood of smoking uptake in young adulthood. This prospective cohort study sought to clarify whether non-smoking related alternative reinforcers (i.e., substitute alternative reinforcers) explain the link between depression symptoms and smoking in young adulthood. Since elevated depression symptoms are common in smokers and are associated with smoking persistence (24,25), a better understanding of how elevated depression symptoms foster regular smoking in young adulthood may provide important clues to preventing established smoking and offer insight into how to promote smoking cessation in young adulthood to prevent the medical and economic consequences of long-term smoking.

METHODS

Participants and procedures

Participants were young adults (n=834) taking part in a longitudinal study (54% female, 68% White) of the predictors of smoking adoption. Participants were originally enrolled in the cohort at age 14 through school-based recruitment at five public high schools representative of those in a Northern Virginia county. This sample represents a subset (79%) of the initial longitudinal cohort of adolescents that has been described in detail elsewhere (26,27). The participants for the present study were 18 – 19 years old at baseline and were

followed for four years (age 22–23 years old). Participation involved completing an annual 30-minute telephone survey. Participants received \$20 after the completion of each survey. The data for the present study are the 834 participants with complete data on the baseline covariates.

Measures

Smoking—Smoking was assessed at each wave with 13 standard epidemiological questions (28). Participants reporting to have smoked in the past 30 days were asked to estimate the number of cigarettes smoked in the past 30 days, the smoking measure used in the present study. This variable was log (base e) transformed for each wave for an observed continuous measure of the number of cigarettes smoked in the past 30 days at each wave to correct for univariate non-normality.

Depression symptoms—Depression symptoms were assessed at each wave with the Center for Epidemiological Studies Depression (CES-D) inventory. The CES-D is a valid and reliable 20-item self-report measure designed to assess depression symptoms in the general population (11,29–31). Scores range from 0 to 60, with scores above 16 indicative of a clinical level of depressive symptoms. The 20-items were linearly summated to form a single depression symptoms score at each wave.

Substitute alternative reinforcers—The Pleasant Events Schedule (PES) (32,33) was adapted to measure alternative reinforcers. The PES was designed to assess reinforcers that occur in an individual's natural environment. The 320-items are rated once in terms of frequency (0=none to 3=often) and once in terms of enjoyability (0=none to 3=very) over the past 30 days (640 responses). The PES was adapted to reduce response burden by deleting items that would not be relevant (e.g., spending time with grand children) and by collapsing items into content classes. For example items such as art work, refinishing furniture, and photography were collapsed into arts and crafts. This resulted in 78 items. These collapsed categories matched reinforcers generated by responses from young adults (34).

Each item yields a frequency score and an enjoyability score, the cross-product of which provides a measure of the activity's reinforcement. They next were asked whether the activity was associated with smoking. If the activity was not associated with smoking, it was considered a substitute reinforcer. The cross-products of the substitute reinforcers were summed and then standardized to provide standardized scores for substitute alternative reinforcers. Higher scores on this measure discriminate between young adult ex-smokers and current smokers and predict smoking abstinence among young adults in smoking cessation treatment (15).

Covariates—Demographic variables included gender (1= "male," 2= "female") and race (0= "white," 1= "non-white"). Household smoking was assessed with a binary variable (0=nobody living in the household smokes, 1=at least one household member smokes). Peer smoking was measured by summing responses to three items asking whether the adolescent's best friend smokes, and whether and, if so, how many of his or her other four best male and four best female friends smoke (range 0 to 9 friends smoking) (35). Marijuana use and alcohol use were each assessed with one item asking "During the past 30 days, how many times have you used marijuana?" and "During the past 30 days, on how many days did you have at least one drink (not just a sip) of alcohol?" Response choices ranged from 0 to all 30 days for alcohol use and 0 to 40 or more times for marijuana use (28). Stress was assessed with the four-item Perceived Stress Scale (36). Items were summed and then split at the median to provide a dichotomous measure of perceived stress (0=lower perceived

stress, 1=higher perceived stress). We controlled for these covariates in the model as potential confounding variables given their associations with smoking, substitute reinforcers, and depression symptoms (27,37–43).

Analytical Approach

Univariate statistics were generated to describe the study population in terms of demographics, smoking practices, substitute reinforcers, and depression symptoms. Univariate estimates were generated with SAS 9.1.3 software.

Latent Curve Growth Modeling (LGCM)—LGCM was conducted to assess the direct and indirect effects of depression symptoms on smoking through substitute alternative reinforcers. LGCM was conducted using *Mplus* version 5.2 software (44). LGCM is a Structural Equation Modeling method that models repeated observed measures on factors (latent variables) representing random effects (η s). Level factors represent baseline level and trend factors represent growth or rate of change across time. In the present analysis, we conducted associated processes LGCM, which is a multivariate method that allows testing paths among random effects (i.e., levels [η_0] and trends [$\eta_{1,2,\dots}$]) from two or more LGCMs (45). Three associated processes were modeled, one each for the repeated observed measures of depression symptoms, smoking, and substitute alternative reinforcers. We were interested in whether the depression symptoms level and trend factors affected the smoking trend factor directly and indirectly through the substitute reinforcers trend factor. To control for the effects of prior smoking and substitute alternative reinforcers, we regressed the depression symptoms trend factor on baseline levels of smoking and substitute reinforcers.

Evaluating model fit—Model fit was evaluated with model chi-square (X^2), Comparative Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA), and Standardized Root Mean Residual (SRMR). Suggested fit criteria are non-significant X^2 , CFI > .95, RMSEA < .05–.08 (46–48). An RMSEA value zero represents exact model fit (46). We used maximum likelihood robust parameter estimates to correct for multivariate non-normality.

Missing data—To account for missing data, multivariate modeling used all available data. *Mplus* allows modeling with missing data using maximum likelihood estimation of the mean, variance, and covariance parameters, when requested, employing the Expectation Maximization (EM) algorithm, assuming data are missing at random (49). Final analyses were based on 834 young adults.

RESULTS

Descriptive Statistics

Table 1 presents the bivariate correlations, means, and standard deviations for all model variables, including the log transformed values for the dependent repeated measures of cigarettes smoked in the past 30 days, depression symptoms (not log transformed), and the standardized values for the repeated measures of substitute reinforcers. The percentage of young adults smoking at least one cigarette in the past 30 days was 25%, 25%, 23%, and 21% for the first through the fourth year post high school (waves 1 through 4), respectively. The average number of cigarettes smoked in the past 30 days (not log transformed) among participants reporting to have smoked at least one cigarette in that time frame was 108.99 (SD=169.84), 126.33 (SD=199.39), 110.30 (SD=173.58), 101.65 (SD=183.64), for waves 1 to 4, respectively. About 15% of baseline never smokers reported smoking on at least one of the follow-ups. Average depression symptoms scores across waves 1 – 4 were 17.91 (SD=5.14; range 7–36), 18.39 (SD=5.38; range 8–36), 18.17 (SD=5.32; range 6–40), and 17.43 (SD=5.15; range 4–35), respectively. The average non-standardized values for

substitute reinforcers remained relatively stable across the four waves and were 138.32 (SD=46.66) in wave 1, 141.37 (SD=49.34) in wave 2, 139.85 (SD=46.69) in wave 3, and 138.16 (SD=48.76) in wave 4.

Model fit

This three process LGCM fit the data reasonably well, $\chi^2_{(101, 834)} = 222.74, p < .0001$, CFI=.97, RMSEA=.04, 90% CI=.03, .05, probability RMSEA $\leq .05 = 1.00$. SRMR=.04. Please see Figure 1.

Direct Effects

The non-standardized regression coefficients (β), standard errors (SE), z-statistics (β/SE), and associated p -values for all model effects are presented in Table 2. Standardized path coefficients for significant model pathways are presented in Figure 1.

Smoking—Substitute reinforcers trend had a significant negative effect on smoking trend ($\beta = -.62, z = -2.99, p = .003$), indicating that an increase in substitute reinforcers over time (from one wave to the next) was associated with a decrease in the rate of change (deceleration) in smoking over time. In other words, more substitute reinforcers predicted less smoking over time. Among the covariates, household ($\beta = .55, z = 4.31, p < .0001$) and peer smoking ($\beta = .28, z = 8.98, p < .0001$), and alcohol use ($\beta = .14, z = 3.28, p = .001$) and marijuana use ($\beta = .41, z = 5.11, p < .0001$) over the past 30-days each had significant positive effects on smoking level. There were no effects for any of the covariates on smoking trend.

Substitute Alternative Reinforcers—Depression symptoms level (baseline at age 18–19 years old) had a significant negative effect on substitute reinforcers trend ($\beta = -.01, z = -3.17, p = .002$), indicating that the greater the depression symptoms at baseline, the greater the decline in substitute alternative reinforcers over time. There were no significant effects for any covariate on substitute reinforcers trend. Among the covariates, being female ($\beta = .30, z = 4.71, p < .0001$) and the frequency of alcohol consumption in the past 30 days ($\beta = .10, z = 4.41, p < .0001$) each had positive effects on substitute reinforcers level. Non-White race ($\beta = -.19, z = -2.73, p = .01$), household smoking ($\beta = -.15, z = -2.05, p = .04$), peer smoking ($\beta = -.10, z = -6.12, p < .0001$), and perceived stress ($\beta = -.18, z = -2.85, p = .004$) were each negatively associated with substitute reinforcers level.

Depression Symptoms—Substitute reinforcers level had significant positive effect on depression symptoms trend ($\beta = .21, z = 2.57, p = .01$), indicating that higher baseline substitute reinforcers predicted an acceleration in depression symptoms. Among the covariates, being female ($\beta = .74, z = 2.43, p = .02$), past 30-day marijuana use ($\beta = .42, z = 2.18, p = .03$), and perceived stress ($\beta = 3.79, z = 11.17, p < .0001$) each had significant positive effects on baseline depression symptoms level. Perceived stress also had a significant negative effect on depression symptoms trend ($\beta = -.55, z = -3.81, p < .0001$). High baseline perceived stress was associated with a 3.79 point increase (non-standardized values) in baseline depressive symptoms. Given the strong positive effect of perceived stress on baseline depressive symptoms, the effect of perceived stress on depressive symptoms trend (rate of change) was negative, indicating a slowing of growth in depressive symptoms (a .11 decrease in the rate of change (slope) in depressive symptoms from baseline) that could be attributed to having high perceived stress.

Indirect Effects of Depression on Smoking

We evaluated whether there were indirect effects of depression symptoms on smoking through substitute reinforcers. As such, we tested indirect effects from depression symptoms

level and trend to smoking trend through substitute reinforcers trend, for significance with Delta method standard errors. The 95% confidence intervals (CI) and 99% CI (if it did not include zero) also provide a measure of the strength of the indirect effect, suggesting minimum and maximum effects (50). Depression symptoms level had a significant total effect (direct plus indirect paths) on smoking trend ($\beta_{\text{total}} = .01$, $z = 2.08$, $p = .04$). The direct effect of depression symptoms level on smoking trend was not significant ($\beta_{\text{direct}} = .005$, $z = .68$, $p = .49$), but the indirect effect through substitute reinforcers trend was significant ($\beta_{\text{indirect}} = .01$, $z = 2.09$, $p = .04$) with a 99% confidence interval for log change that did not include zero (0.001, 0.02). This indicates that greater depression symptoms at baseline predicted decreases in substitute reinforcers across time which in turn predicted increases in smoking uptake/rate from emerging to young adulthood. Specifically, a unit increase in depression symptoms at baseline was associated with a .01 decline in the rate of change in substitute reinforcers (substitute reinforcers trend) from its intercept value ($\alpha_{\text{subs trend}} = .224$), a 5% decrease. A unit decline in the substitute reinforcers trend (rate of change) was associated with a .62 increase in the smoking trend factor from its intercept value ($\alpha_{\text{cigarettes trend}} = .398$), a 1.6 fold increase in the rate of change in cigarettes smoked. To assess strength of mediation on a continuum from absence of mediation to complete mediation, we estimated the ratio of the indirect effect to the total effect (i.e., effect proportion mediated $\beta_{\text{indirect}} / \beta_{\text{total}}$). The proportion mediated was 100%, indicating that the effect was completely due to the indirect effect (i.e., complete mediation). Together these predictors (latent and observed variables) accounted for 27% of the variance in smoking trend.

DISCUSSION

The present study provides the first longitudinal evidence for processes linking depression symptoms in emerging adulthood to smoking uptake in young adulthood. Higher depression symptoms in emerging adulthood (age 18–19 years old) predicted declines in non-smoking alternative reinforcers across time, which in turn, predicted increases in smoking uptake/rate in young adulthood (age 18–19 years old). These findings highlight that depression in emerging adulthood is a risk factor for subsequent smoking because of its negative influence on alternative reinforcers and suggest that promoting greater alternative reinforcers or mitigating declines may prevent smoking uptake and increases in smoking rate in young adults.

These findings bridge the research showing that reductions in reinforcing activities are a consequence of depression and research showing that substance use is acquired and maintained within the context of fewer alternative reinforcers (12,14,17). Here, we show that declining alternative reinforcers link depression symptoms to the establishment of young adult smoking. Behavioral theory posits that the probability of substance use increases as a function of decreases in alternative sources of reinforcement as well as the availability of substances. As elevated depression symptoms precipitate declines in substitute alternative reinforcers, young adults may be more likely to choose to smoke and to find smoking more rewarding (20). A consistently lower level of substitute alternative reinforcers may promote smoking persistence and continued elevations in depression symptoms. Recent research indicates that young adult smokers have fewer substitute reinforcers than ex-smokers (15) and almost half of smokers seeking smoking cessation treatment have elevated depression symptoms (24).

Depression, even at subclinical levels is often accompanied by withdrawal and less involvement in rewarding activities (12,51). In addition to being an easily available reinforcer, nicotine may mitigate these losses by increasing the salience of rewarding stimuli in the environment and by enhancing reward from the non-smoking reinforcers that are

present in the environment (23,52–54). The role of nicotine in reward processing may explain why depressed smokers are more likely to choose smoking as their preferred activity and why they report needing significantly more alternative reinforcers to quit smoking compared to smokers without a depression diagnosis (22). Our findings suggest that similar relationships exist for subclinical levels of depression and the establishment of smoking as a habit.

From a clinical standpoint these findings emphasize the importance of young adulthood as an opportune time to prevent smoking uptake and promote smoking cessation. About 50% of the sample had elevated depression symptoms, which increased the likelihood of smoking via diminished substitute alternative reinforcers. Interventions directed toward emerging adults with elevated depression symptoms and focused on increasing alternative reinforcers or preventing declines in alternative reinforcers may have a significant impact on preventing young adult smoking. These interventions may involve facilitating the identification and daily involvement in pleasurable activities not linked to smoking or drug use and/or preventing declines in these the pleasurable activities through self-monitoring and time allocation. These interventions also could be informed, in part, by successful depression prevention programs, which target young people with elevated depression symptoms and have an impact on a range of behaviors that commonly co-occur with cigarette smoking (55,56).

In addition, a context of elevated depression symptoms and reduced reinforcers may promote increases in smoking rate, less interest in quitting and a decreased likelihood of successful smoking cessation (15). Incentive-based approaches may increase motivation to enroll and achieve initial abstinence, while behavioral therapy can facilitate smoking cessation skills and a repertoire of substitute alternative reinforcers to smoking. Recent research suggests that these approaches hold promise in promoting smoking cessation and abstinence from other substance of abuse (15,17,51).

It is important to point out that only a portion of those with elevated depression symptoms smoked cigarettes. A feature of depression, not present in all young people with elevated depression symptoms, may be responsible for the observed relationships. Anhedonia, or loss of interest in usual rewarding activities, may help explain the heterogeneity (57). A temporary loss of interest or a dispositional diminished capacity to derive pleasure from activities that others typically find rewarding warrant further investigation (58–60). Identification of the distinct aspect(s) of depression that impact smoking behavior may help to better define young adults who are at risk for smoking and highlight valuable treatment targets.

As our model accounted for about 30% of the variance in smoking, other mechanisms explaining the relationship between depression and young adult smoking should be evaluated in future research. The enhancement of positive affect and the management of negative affect are potential mechanisms to consider. Depression is associated with lower levels of positive affect and higher levels of negative affect (61–63). Lower levels of positive affect predict smoking progression among adolescents (64) and an inability to achieve smoking abstinence (65) and shorter time to relapse in adults (66). Less involvement in reinforcing activities and diminished reward from usual activities (decreased hedonia) are associated with a reduction in positive affect (61,63,67). Likewise, young adults with elevated depression symptoms may smoke because it either relieves negative affect or it decreases the variability in negative affect (68).

It is important to note that we did not find evidence for a direct effect of depression symptoms on smoking uptake, only an indirect effect. Evaluation of more complex

relationships between depression and smoking, such as indirect effects, may provide a better understanding of the comorbidity and highlight targets for smoking prevention and cessation interventions (69). We also considered the possibility that smoking contributed to depression symptoms directly and indirectly through a decline in substitute reinforcers. However, we did not find support for a direct or indirect effect of smoking on depression symptoms in young adulthood. Although evidence exists for reductions in depression among adolescents who progressed along the smoking uptake continuum (70), these mood modulation effects may diminish with greater smoking experience (71).

To our knowledge, this is the first longitudinal investigation of how depression can influence smoking progression and the establishment of regular smoking practices during emerging and young adulthood. Along with the several study strengths (e.g., large sample, repeated measures of key variables, control for many confounding influences), the limitations must be noted. One potential limitation is that we are not able to measure the extent of depression as diagnostic assessments were not completed. Higher levels of depression as measured by the CES-D may have been reflective of clinical episodes rather than subclinical depression symptoms. In addition, we are not able to clearly discern smoking uptake from an increase in smoking rate among more regular smokers in the mediation model. We are not aware of a two-piece model that can accommodate multiple associated processes and indirect effects. Finally, our annual assessment intervals may not have allowed us to capture finer-grained changes in our variables of interest. However, the model does highlight a general and novel connection between subclinical depression, alternative reinforcers, and smoking behavior among young adults. A better understanding of how elevated depression symptoms foster regular smoking in some young adults, but not others may help inform smoking prevention and smoking cessation interventions specifically designed for this age group. A reduction in smoking in this age group could have a significant impact on smoking attributable morbidity and mortality.

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Figure 1.
The indirect effects of depression symptoms on smoking uptake via substitute alternative reinforcers. Standardized path coefficients are shown only for significant effects.

Table 1

Correlation matrix for all model variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Substitute reinforcers 1	1.00									
2. Substitute reinforcers 2	0.64	1.00								
3. Substitute reinforcers 3	0.58	0.60	1.00							
4. Substitute reinforcers 4	0.56	0.59	0.68	1.00						
5. Depression symptoms 1	0.00	-0.01	-0.02	-0.08	1.00					
6. Depression symptoms 2	-0.03	-0.03	-0.07	-0.06	0.46	1.00				
7. Depression symptoms 3	-0.03	-0.02	-0.06	-0.10	0.45	0.46	1.00			
8. Depression symptoms 4	0.04	0.03	-0.02	-0.06	0.47	0.46	0.48	1.00		
9. Cigarettes per month 1	-0.31	-0.31	-0.19	-0.18	0.15	0.15	0.15	0.12	1.00	
10. Cigarettes per month 2	-0.27	-0.35	-0.26	-0.24	0.16	0.18	0.13	0.14	0.79	1.00
11. Cigarettes per month 3	-0.26	-0.27	-0.30	-0.24	0.08	0.18	0.15	0.16	0.72	0.78
12. Cigarettes per month 4	-0.28	-0.29	-0.28	-0.29	0.08	0.16	0.14	0.15	0.68	0.77
13. Female	0.13	0.15	0.15	0.09	0.11	0.05	0.06	0.04	-0.08	-0.08
14. Non-White	-0.13	-0.11	-0.06	-0.09	0.03	0.06	0.08	0.05	-0.05	-0.05
15. Alcohol use	0.04	0.02	0.04	0.06	0.05	0.07	-0.01	0.06	0.41	0.34
16. Marijuana use	-0.12	-0.07	-0.04	-0.04	0.08	0.17	0.13	0.10	0.47	0.41
17. Peers smoking	-0.21	-0.20	-0.12	-0.11	0.07	0.09	0.11	0.08	0.56	0.50
18. Household smoking	-0.11	-0.07	-0.08	-0.04	0.09	0.12	0.16	0.10	0.26	0.22
19. Perceived stress	-0.12	-0.10	-0.08	-0.10	0.42	0.25	0.25	0.26	0.12	0.07
Means	.01	-.04	-.01	-.03	17.91	18.39	18.17	17.43	.86	.90
Standard deviations	1.00	.99	.98	.99	5.14	5.38	5.32	5.15	1.73	1.78
	11.	12.	13.	14.	15.	16.	17.	18.	19.	
11. Cigarettes per month 3	1.00									
12. Cigarettes per month 4	0.83	1.00								
13. Female	-0.12	-0.10	1.00							
14. Non-White	-0.01	-0.01	0.00	1.00						
15. Alcohol use	0.28	0.27	-0.10	-0.23	1.00					

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
16. Marijuana use	0.41	0.35	-0.09	-0.08	0.44	1.00				
17. Peers smoking	0.45	0.42	-0.04	-0.05	0.40	0.42	1.00			
18. Household smoking	0.18	0.14	0.05	0.05	0.14	0.09	0.22	1.00		
19. Perceived stress	0.08	0.06	0.09	0.08	0.03	0.07	0.11	0.12	1.00	
Means	.85	.81	1.54	.32	2.50	1.44	2.22	.28	.41	
Standard deviations	1.71	1.66	.5	.47	1.56	1.04	2.24	.45	.49	

Note. Cigarettes smoked 1 – Cigarettes smoked 4, log transformed number of cigarettes smoked in the past 30 days for waves 1 through 4; Female (Female=2, Male=1); Non White (Non White =1, White =0); Household smoking (1=at least one person living with the participant smokes, 0=nobody living with the participant smokes); Perceived stress (0=lower stress, 1=higher stress); Past 30 day alcohol use; Past 30 day marijuana use; Peers smoking (0 to 9 peers).

Table 2

Non-standardized parameter estimates (β), standard errors (SE), z statistics and probability (p) values for all model regression equations

Substitute reinforcers ^a								
	level				trend			
	β	SE	z-statistic	p-value	β	SE	z-statistic	p-value
Depression symptoms level	-	-	-	-	-0.01	0.00	-3.17	<0.01
Depression symptoms trend	-	-	-	-	-0.28	0.17	-1.64	0.10
Female	0.30	0.06	4.71	<.01	-0.05	0.05	-1.02	0.31
Non-White	-0.19	0.07	-2.73	0.01	0.08	0.06	1.35	0.18
Alcohol use	0.10	0.02	4.41	<.01	-0.02	0.02	-1.07	0.29
Marijuana use	-0.06	0.04	-1.61	0.11	0.03	0.03	1.38	0.17
Peers smoking	-0.10	0.02	-6.12	<.01	0.02	0.01	1.71	0.09
Household smoking	-0.15	0.08	-2.05	0.04	0.09	0.06	1.59	0.11
Perceived stress	-0.18	0.06	-2.85	<.01	-0.11	0.11	-0.97	0.33
Depression symptoms ^a								
	Level				trend			
	β	SE	z-statistic	p-value	β	SE	z-statistic	p-value
Substitute reinforcers level	-	-	-	-	0.21	0.08	2.57	0.01
Smoking level	-	-	-	-	0.07	0.05	1.42	0.16
Female	0.74	0.31	2.43	0.02	-0.23	0.13	-1.74	0.08
Non-White	0.13	0.34	0.37	0.71	0.21	0.16	1.34	0.18
Alcohol use	0.03	0.11	0.27	0.79	-0.07	0.05	-1.29	0.20
Marijuana use	0.42	0.19	2.18	0.03	0.03	0.07	0.33	0.74
Peers smoking	-0.01	0.08	-0.19	0.85	0.02	0.04	0.57	0.57
Household smoking	0.51	0.36	1.42	0.15	0.18	0.15	1.22	0.22
Perceived stress	3.79	0.34	11.17	<.01	-0.55	0.14	-3.81	<.01
Smoking ^a								
	Level				trend			
	β	SE	z-statistic	p-value	β	SE	z-statistic	p-value

Substitute reinforcers ^a									
	level				trend				
Substitute reinforcers level	-	-	-	-	0.11	0.17	0.67	0.50	
Substitute reinforcers trend	-	-	-	-	-0.62	0.21	-2.99	<.01	
Depression symptoms level	-	-	-	-	0.01	0.01	0.68	0.49	
Depression symptoms trend	-	-	-	-	-0.95	0.90	-1.06	0.29	
Female	-0.14	0.09	-1.54	0.13	-0.25	0.24	-1.03	0.30	
Non-White	0.01	0.09	0.13	0.90	0.24	0.23	1.02	0.31	
Alcohol use	0.14	0.04	3.28	<.01	-0.08	0.07	-1.10	0.27	
Marijuana use	0.41	0.08	5.11	<.01	0.02	0.09	0.24	0.81	
Peers smoking	0.28	0.03	8.98	<.01	0.02	0.05	0.42	0.68	
Household smoking	0.55	0.13	4.31	<.01	0.10	0.25	0.40	0.69	
Perceived stress	0.16	0.10	1.62	0.11	-0.59	0.51	-1.15	0.25	

Note. Female (Female=2, Male=1); Non White (Non White=1, White=0); Household smoking (1=at least one person living with the participant smokes, 0=nobody living with the participant smokes); Perceived stress (0=lower stress, 1=higher stress); Past 30 day alcohol use; Past 30 day marijuana use; Peers smoking (0 to 9 peers).

^aThese are the dependent processes in the regression equations. The outcome variables (levels and trend) are latent variables (factors).