Published in final edited form as:

Addiction. 2015 March ; 110(3): 519–529. doi:10.1111/add.12797.

Gender differences in the relationship between affect and adolescent smoking uptake

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Abstract

Aims—To evaluate gender differences in the role of positive and negative affect on smoking uptake.

Design—Prospective longitudinal cohort study of adolescent health behaviors.

Setting—Four suburban secondary schools outside Philadelphia, Pennsylvania, USA.

Participants—Adolescents ($n = 1357$) were surveyed every 6 months for 4 years (age 14–18 years).

Measurements—Smoking and affect were measured via survey at each of the eight time-points.

Findings—A two-group associative process latent growth curve model revealed that baseline positive affect was related negatively to smoking progression for females ($b = -0.031$, $Z = -4.00$, $P < 0.0001$) but not for males ($P = 0.33$). This gender difference was significant, $\chi^2_{(df=1)} = 8.24$, $P = 0.0041$, indicating that for every standard deviation (SD) decrease in positive affect (SD = 2.90), there was a 10% increase [odds ratio (OR) = 1.10, 95% confidence interval (CI) = 1.04, 1.14] in the odds of smoking progression for females. Baseline negative affect was related significantly and positively to smoking progression for males ($b = 0.038$, $Z = 2.874$, $P = 0.004$) and females ($b = 0.025$, $Z = 3.609$, $P < 0.0001$), but the gender difference was not significant, $\chi^2_{(df=1)} = 0.82$, $P = 0.37$. Thus, on average, for every standard deviation (SD = 4.40) increase in baseline negative affect there was a 15% (OR = 1.15, 95% CI = 1.06, 1.26) increase in the odds of smoking progression for males and for females.

Conclusions—The impact of affect on adolescent smoking uptake varies by gender. Low positive affect (low experience of positive feelings or emotions) for females and high negative

affect (high experience of negative feelings or emotions) for both males and females increases the risk for adolescent smoking.

Keywords

Adolescent smoking; gender; positive affect; negative affect

INTRODUCTION

Approximately 20% of adolescents regularly smoke cigarettes in the United States [1], with the percentage of regular smokers doubling and the percentage of daily smokers tripling from mid to late adolescence [1,2]. Increasingly, affective vulnerabilities such as depression are being recognized as important contributors to adolescent smoking initiation and progression [3–9].

While epidemiological research indicates that the prevalence of depression is higher among adolescent females than males [10–12], gender differences in the relation of depression on smoking uptake have received little prospective investigation. Cross-sectional studies support an association between depression and smoking status for females, but not males [13,14]. In contrast, prospective research has shown that depression predicts smoking initiation for males, but not for females [15]. In addition to methodological features, these disparate findings may suggest gender differences in the dimensions that comprise the multi-factorial construct of depression [16].

Most multi-factorial models of depression purport that depression can be parsed into two primary affect dimensions of low positive affect (i.e. diminished levels of happiness, enjoyment and positive moods) and high negative affect (i.e. sadness, fear), and sometimes non-affective dimensions, such as interpersonal problems and somatic symptoms [17,18]. Research indicates that positive and negative affect both relate to smoking in adults, but differ in the degree as well as the smoking characteristic (e.g. status, heaviness, cessation) [19].

Although conceptualizations of the relationship between adolescent smoking and depression have focused on smoking to relieve negative affect, there has been growing interest in the relationship between smoking and positive affect [20]. Adolescents experience increases in positive affect and decreases in negative affect following smoking [21] and greater overall affective changes following smoking predict smoking escalation [22]. Thus, low positive affect and high negative affect reflect empirically distinct processes that may both play a role in smoking. However, the impact of changes in positive and negative affect across adolescence on smoking uptake for males and for females has yet to be investigated. The present study sought to evaluate gender differences in the longitudinal relation of positive and negative affect on smoking uptake. Increasing our understanding of the link between affect and adolescent smoking for males and females can greatly inform affective models of addiction motivation as well as the next generation of smoking prevention interventions.

METHODS

Participants and procedures

Participants were high school students taking part in a longitudinal study of adolescent health behaviors. Participants were enrolled into one of four public high schools in suburban Philadelphia, PA. This cohort was drawn from the 1517 students identified through class rosters at the beginning of 9th grade. Students were ineligible to participate in this study if they had a severe learning disability or if they did not speak fluent English. Based on the selection criteria, a total of 1487 (98%) students were eligible to participate. Parents were mailed a study information letter (active information) with a telephone number to call to obtain answers to any questions and to decline consent for their adolescent to participate (passive consent). Of these 1487 eligible teens, 1478 (99%) had a parent's passive consent to participate. Thirty adolescents were absent on the assent/survey days and 19 did not provide assent due to lack of interest in the study. Thus, 1429 of 1478 teens with parental consent (97%) provided their assent to participate and completed a baseline survey. Adolescents who declined assent or who were absent on the baseline survey day did not differ on race and gender from those who provided assent and completed the baseline survey.

The adolescent cohort was formed in the 9th grade and followed until the end of 12th grade. A self-report 40-minute survey was administered every 6 months (fall and spring semesters) on-site during compulsory classes each year of high school for a total of eight surveys. The sample ($n = 1357$) comprised participants with complete data for the model covariates. University Institutional Review Board approval of the study was obtained.

Measures

Primary variables

Smoking: Smoking progression was derived from evaluating smoking practices at each wave with a series of standard epidemiological questions regarding smoking, such as: 'Have you ever tried or experimented with cigarette smoking, even a few puffs?' and 'Have you smoked a cigarette in the past 30 days?' [23,24]. The seven-category ordered categorical smoking variable used in this study was coded as 0 = never smoked, 1 = puffed but did not smoke a whole cigarette, 2 = smoked a whole cigarette but not in the past month, 3 = smoked in the last month, 4 = smokes weekly, 5 = smokes 10 cigarettes daily and 6 = smokes >10 cigarettes daily.

Positive and negative affect: We assessed positive and negative affect at each wave with 11 items from the 20-item Center for Epidemiologic Studies Depression Scale (CESD) [25], an epidemiological measure assessing four facets of depressive symptoms (positive and negative affect, somatic symptoms and interpersonal distress) on a four-point Likert-style scale. Positive affect was assessed with four items measuring symptoms indicative of happiness and enjoyment in the past week ($\alpha = 0.75$). Negative affect was assessed with seven items ($\alpha = 0.89$).

Covariates—We controlled for the effects of several covariates at baseline. This included race and parental education. Peer smoking was assessed by asking participants whether their

best friend smoked and how many of their four other best male and four other best female friends smoke (range 0–9 friends). Household smoking exposure was assessed by asking if anyone living in the household smokes [26–28]. Past 30-day marijuana and alcohol use were each assessed as in previous studies [24,26]. Perceived parental monitoring was assessed with a five-item Likert-style scale that evaluated adolescent perceptions of parental knowledge of whereabouts, activities and friendships [29–32]. Impulsivity was assessed using the impulsivity subscale of the Temperament and Character Inventory [33]. Baseline interpersonal problems (two items) and somatic symptoms (seven items) were measured by CESD subscales [25].

Statistical analysis

Univariate statistics were generated to describe the study population with SAS version 9.1.3 software.

Latent growth curve modeling (LGCM)—Associated-processes LGCM was conducted to assess the longitudinal relations of repeated measures of positive and negative affect with smoking. LGCM is a structural equation modeling method that models repeated observed measures (measured variables) on factors (latent variables) representing random effects (η s) [34]. A level factor is used to represent baseline and a trend factor is used to represent change across time. Factor loadings (i.e. correlations between the observed and latent variables) are fixed to define baseline and to define trend. Each process in the associated-processes model is a linear growth model composed of one baseline factor and one factor representing rate of change from baseline.

We conducted a two-group associated processes LGCM, dividing the model by gender [35]. This means that two concurrent models were run, one associated-processes LGCM for males and one for females. The two-group associated processes model allowed us to assess the impact of baseline levels of positive affect and negative affect on smoking progression for females and then for males. Further, given that changes in positive and negative affect may parallel changes in smoking across time, we also allowed the trend (slope) factors to correlate freely, and tested whether differences in trend to trend correlations between males and females were significant. The models for each of the three processes included intercept and linear trend factors only, as the complexity of the two group model with repeated measures over eight waves failed to solve with additional growth form factors (e.g. quadratic trends).

Model estimation and fit criteria—We estimated model parameters with a weighted least squares estimation technique (WLSMV) in which the diagonal weight matrix uses robust standard errors, and the χ^2 test statistic is mean and variance-adjusted [36]. Models are evaluated by assessing how well the variance/covariance matrix implied by the model fits the observed data variance/covariance matrix [37]. Suggested criteria for model fit are non-significant model χ^2 , comparative fit index (CFI) above 0.95, root mean square error of approximation (RMSEA) below 0.05–0.08 and a WRMR value below 0.9 [37,38]. To test for gender differences we constrained to equality comparison paths, however, only if the paths were either significant for males and females, or significant for only one gender. We

conducted a χ^2 difference test to determine whether the constraints significantly degraded model fit. If the model fit was worse after the constraint, it was determined that a sex difference exists [37]. Mplus version 7.11 software provides a special method for χ^2 difference testing when dependent variables are categorical, as χ^2 differences from models with WLSMV or WLSM parameter estimates do not have a χ^2 distribution [39].

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, using a full information maximum likelihood estimating procedure which employs the expectation maximization algorithm, assuming data are missing at random [36,39]. This only accounted for missing data on the repeated measure of smoking and positive and negative affect, not the time-invariant covariates. Thus, cases with missing data on the time-invariant covariates were not included in the analysis.

RESULTS

Descriptive statistics

Table 1 presents the univariate statistics. Twenty-one per cent of adolescents progressed along the smoking uptake continuum across 4 years, with females being 33% more likely to have progressed than males [odds ratio (OR)= 1.33, 95% confidence interval (CI) = 1.02, 1.74]. Females had a greater increase in positive affect [mean change = 0.60, standard deviation (SD) = 3.28] than males (mean change = 0.04, SD = 3.38), $t\text{-test}_{(1065)} = -2.78$, $P = 0.006$. Females also had a greater change in negative affect (mean change = -0.95, SD = 5.35) compared to males (mean change = 0.24 SD = 4.82), and in the opposite direction, decreasing for females but increasing for males, $t\text{-test}_{(1064)} = 3.81$, $P = 0.0001$.

Multivariate models

The bivariate statistics supported evaluating gender as a covariate in a single-group model. The single-group associative processes LGCM with three processes (positive affect, negative affect and smoking) fitted the observed data variance/covariance matrix fairly well; $\chi^2_{(505,1357)} = 1990.11$, $P < 0.0001$, CFI=0.95, RMSEA =0.047 (90% CI=0.044, 0.049), WRMR =1.79. The model revealed significant effects for baseline positive affect (OR= 0.97, 95% CI= 0.95, 0.99) and baseline negative affect on smoking trend (OR = 1.04, 95% CI = 1.02, 1.05). Female gender was related to baseline positive affect ($b = -0.25$, $P = 0.04$), positive affect trend ($b = 0.10$, $P = 0.001$) and baseline negative affect ($b = 0.64$, $P < 0.0001$). While gender was a significant predictor of affect, there were no significant gender differences in smoking progression. These findings supported further examination of gender through a two-group model.

The results of the two-group LGCM divided by sex are presented in Table 2. Non-standardized parameter estimates, standard errors, Z-test statistics and P -values for the effects of baseline negative affect and positive affect to smoking trend, and the covariates, were presented first for females and then for males. Within each gender these values were presented first for smoking, followed by positive and then negative affect. Figure 1 presents a graphic depiction of the multivariate model using standardized values for significant

model effects only. Standardized path coefficients are presented for females (above the division line) and males (below the division line). We included paths for the covariates in Fig. 1 only if the gender difference was significant, in an attempt to promote clarity. In contrast to our prior research, we included peer smoking as a baseline covariate and did not examine whether peer smoking had mediating effects [3].

Overall model fit

The two-group associative processes LGCM with three processes (positive affect, negative affect and smoking) fitted the observed data variance/covariance matrix fairly well;

$\chi^2_{(984,1357)}=2389.73$, $P < 0.0001$, CFI = 0.95, RMSEA = 0.05 (90% CI = 0.044, 0.048), WRMR = 2.04. We first tested for differences in the parameter estimates by gender, and then whether the differences between males and females were significant. Results are presented in Table 2.

Path from positive affect level to smoking trend—Baseline positive affect was related significantly to smoking trend for females ($b = -0.031$, $Z = -4.00$, $P < 0.0001$) but not for males ($P = 0.33$). This difference in effects (i.e. significant path for females but not for males) was significant, $\chi^2_{(df=1)} = 8.24$, $P = 0.0041$, indicating that lower baseline positive affect was associated with increased odds of smoking progression for females, but that positive affect had no effect on smoking progression for males. Thus, for every standard deviation decrease in positive affect ($SD = 2.90$), there was a 10% increase ($OR = 1.10$, 95% CI = 1.04, 1.14) in the odds of smoking progression for females.

Path from negative affect level to smoking trend—Baseline negative affect was related significantly and positively to smoking trend for males, ($b = 0.038$, $Z = 2.874$, $P = 0.004$) and females ($b = 0.025$, $Z = 3.609$, $P < 0.0001$). However, the gender difference in effects was not significant, $\chi^2_{(984,1357)} = 2389.73$, $P = 0.37$, indicating that higher baseline negative affect was associated with a greater likelihood of smoking progression regardless of gender. Indeed, for every standard deviation increase in negative affect ($SD = 4.13$) there was a 17% increase ($OR = 1.17$, 95% CI = 1.05, 1.30) in the odds of smoking progressing for males and a 13% increase ($OR = 1.13$, 95% CI = 1.06, 1.21) in the odds of smoking progression for females ($SD = 4.94$). This means that, on average, for every standard deviation ($SD = 4.40$) increase in baseline negative affect there was a 15% ($OR = 1.15$, 95% CI = 1.06, 1.26) increase in the odds of smoking progression for males and for females.

Effects of affect trend on smoking trend—We assessed the relation between negative affect trend and smoking trend to determine whether changes in negative affect were associated with changes in smoking over time. Negative affect trend was associated significantly and negatively with smoking trend for males ($b = -0.018$, $Z = -2.10$, $P = 0.04$), but not for females ($b = -0.005$, $Z = -0.736$, $P = 0.46$). Decreases in negative affect were paralleled by increases in smoking for males, while changes in negative affect had no association with changes in smoking for females. However, this gender difference was not significant. We were not able to assess the relation between positive affect trend and

smoking trend as the positive affect residual trend variance was constrained to equal zero to allow the model to solve without a negative variance.

Effects of covariates on smoking level and trend—White race was associated with a 37% increase in the odds of smoking at a higher level at baseline for females (OR = 1.37, 95% CI = 1.08, 1.74). However, it was not related significantly to baseline smoking for males ($P = 0.24$). This sex difference in the effects was significant, $\chi^2_{(df=1)} = 7.195$, $P = 0.007$, indicating that being white was a risk factor for smoking among females at baseline, but not males.

White race was associated with an increase odds of smoking progression for males ($b = 0.14$, $Z = 3.062$, $P = 0.002$), but not females ($P = 0.279$). This sex difference was significant, $\chi^2_{(df=1)} = 4.565$, $P = 0.0326$, indicating that being white increased the risk of smoking progression for males, but not females. Further, having a household member who smokes increased the odds of smoking progression for males ($b = 0.09$, $Z = 2.218$, $P = 0.027$), but not females ($P = 0.433$). This difference in effects was significant, $\chi^2_{(df=1)} = 5.841$, $P = 0.0157$, indicating that exposure to household members who smoke influenced smoking progression for males, but not females.

DISCUSSION

Adolescent smoking prevention programs to date have been largely ineffective at preventing adolescent smoking [40,41]. While population-based policies have been relatively more successful, almost 20% of adolescents still smoke [1]. The adolescent smoking rate, coupled with the limited effectiveness of smoking prevention approaches to date, underscores the need to identify novel factors that drive smoking and their interactions with gender to inform more effective smoking prevention interventions [40,41]. The present study provides the first evaluation of the relationship between two dimensions of affect and adolescent smoking uptake, and whether the relationships vary by gender. Adolescent females with lower positive affect at age 14 were more likely to progress along the smoking uptake continuum from mid to late adolescence. Positive affect had no relation with smoking progression for males. Greater negative affect during mid-adolescence predicted smoking progression across adolescence regardless of gender. These findings emphasize the importance of higher negative affect in smoking uptake for all adolescents, and highlight a unique role for positive affect in smoking uptake for female adolescents.

Lower positive affect coupled with higher negative affect increased the odds of smoking uptake for adolescent females by 25%. Each analysis modeled these risk pathways simultaneously, offering evidence that positive affect and negative affect reflect distinct sources of smoking vulnerability in females, which also operates over and above other factors, including non-affective depressive symptoms and impulsivity. In an effort to regulate positive affect, adolescent females may seek out potent reinforcers such as smoking in order to enhance positive affect [42,43]. While research suggests that cigarettes alone produce a mild increment in positive emotions, there is evidence that nicotine also enhances pleasure derived from other reinforcers [44–47]. Also, recent studies have shown

that variability in negative affect predicted smoking escalation among girls [48], and that rapid smoking escalation was associated with less negative affect variability [49]. Different strategies may be required to offset the risk carried by low positive affect versus high negative affect [50]. The observation that these affective influences were already present at age 14 years emphasizes the importance of affect regulation skills by mid adolescence to reduce the risk of smoking.

The notion that adolescents begin and continue to smoke as adults to manage negative affect dates back almost two decades [6,51]. Our findings indicate that adolescent males with higher levels of negative affect were 17% more likely to progress in their smoking across mid to late adolescence. Decreases in negative affect across adolescence were paralleled by smoking progression for males, but these changes were not different compared to females. Recent studies have shown that negative affect predicted smoking escalation among boys, but only among boys whose smoking motives were to manage affect [48]. Rapid smoking escalation was associated with improved negative affect among boys [49]. However, this effect was not observed at the 15-month follow-up for boys who had a higher smoking rate and had symptoms of nicotine dependence [49]. Given the complexity of these relationships, further research is warranted. While skills to manage negative affect have not been included in traditional smoking prevention programs, these data suggest that such skills have the potential to decrease smoking uptake for males and females. Addressing expectations that smoking reduces negative affect may be an important starting point [52].

While the results revealed very few gender differences in the covariates, our work raises the possibility that males' smoking uptake may be influenced by higher negative affect, whereas females' smoking may be influenced by both lower positive affect and higher negative affect. Positive and negative affect are distinct dimensions [53–56], associated with different neural underpinnings [57–59], and have different psychological correlates [60–62]. These findings emphasize that mid to late adolescence is a formative period for the relationship between affect regulation and smoking.

The study has several strengths, including an excellent participation rate, a large sample, repeated measures of affect and smoking, smaller measurement intervals than previous adolescent smoking studies and control for confounding influences in the statistical models. However, we relied upon self-reports of smoking. Biochemical verification of smoking status is not typically implemented in epidemiological studies, as adolescent self-reports have been determined to be valid and sufficient, especially when confidentiality is assured [63,64]. Further, the standard cotinine cut-off cannot validate the current definition of adolescent smoking (e.g. one cigarette in the past 30 days) [65]. The present study also used the CESD positive and negative affect subscales to measure affect. The CESD sub-scales are appropriate measures of affective symptomatology [66], but whether our findings extend to other constructs in the affective spectrum, including positive and negative affective states that do not span behavioral and cognitive levels of functioning (e.g. as measured by adjective checklists such as the Positive and Negative Affect Scale), is unknown. None the less, the extensive use of the CESD in adolescent risk behavior research also permits greater opportunity for replication studies.

These findings have important implications, including the potential to stimulate further research on affect, gender and adolescent smoking to inform novel and more effective models of smoking prevention [40,41]. The findings also support a shift in the long-standing focus of smoking prevention models from social intervention targets to affect-related targets. A shift in prevention targets would be consistent with the over-representation of smoking and female gender among affectively vulnerable populations [3,9,67–70]. If our findings are replicated this would suggest that affect may be an important message target in public health media campaigns, that affect management skill building may be important to include in novel smoking prevention programs and that affective components could be tailored easily to gender. Our results are the first to suggest that the mitigation of negative mood for all adolescents, and the maximization of positive mood for female adolescents could prevent the reliance on cigarettes to perform these functions [71]. Indeed, the adult literature has shown us that men and women smoke for different reasons [72] and that they respond differently to specific smoking cessation pharmacotherapies [73].

Acknowledgements

This study was supported by National Cancer Institute RO1 CA126958 (JAM) and National Institute on Drug Abuse K08 DA025041 (AML).

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Figure 1.

The moderating effects of gender on the longitudinal relationship between affect and smoking uptake. Standardized path coefficients are shown for the smoking and affective processes. Standardized path coefficients are only presented for the covariates with significant gender differences. Female values are above the line, male values are below the line. NS = not significant; * denotes significant gender difference, $P < 0.05$

Table 1

Descriptive statistics.

Variable	Level	Male		Female	
		n	%	n	%
Race	<i>White</i>	486	72	511	74
	<i>Non-white</i>	185	28	175	26
Household smoking **	No household smoking	399	59	352	51
	Household smoking	272	59	334	59
Parental education	Both parents > high school	395	24	408	24
	1 parent high school	159	17	162	50
	Both parents high school	117	17	116	17
Alcohol use	0 days	530	79	508	74
	1 or 2 days	94	14	115	17
	3-30 days	47	7	63	9
Marijuana use	0 times	615	92	621	91
	1 or 2 times	26	4	32	5
	3 or more times	30	4	33	4
Smoking (baseline)	Never smoked	496	75	470	69
	Smoked, but not a whole cigarette	63	9	77	11
	Smoked, but not in the past month	50	7	56	8
	Smoked in the past month	15	2	27	4
	Smokes weekly	17	3	15	2
	Smokes daily, 10 cigarettes	20	3	33	5
	Smokes daily, > 10 cigarettes	4	1	3	1
Smoking (wave 8) **	Never smoked	347	68	320	58
	Smoked, but not a whole cigarette	39	8	77	14
	Smoked, but not in the past month	50	10	67	12
	Smoked in the past month	18	4	16	3
	Smokes weekly	11	2	10	2
	Smokes daily, 10 cigarettes	33	6	56	10
	Smokes daily, > 10 cigarettes	9	2	7	1
Smoking progression *	Progressed	122	18	157	23

	Mean	SD	Mean	SD
Positive affect wave 1 **	12.40	2.85	11.91	2.90
Positive affect wave 8	12.59	2.70	12.59	2.62
Negative affect wave 1 ***	3.65	4.13	5.36	4.94
Negative affect wave 8 *	3.64	4.35	4.21	4.54
Positive affect change *	.04	3.38	.60	3.28
Negative affect change **	.24	4.82	-.95	5.35
Parental monitoring	12.23	2.73	12.49	2.57

	Mean	SD	Mean	SD
Impulsivity	8.04	1.39	7.96	1.46
Peers smoking ***	1.20	2.07	1.87	1.68
Interpersonal **	1.18	1.45	1.46	1.52
Somatic ***	6.42	3.81	7.80	3.90

SD = standard deviation.

*
 $P < 0.05$

**
 $P < 0.01$

 $P < 0.0001$

Table 2

Estimates, standard errors, Z-statistic and *P*-values for all model regressions, separated for females and males.

Female model								
	Baseline smoking level				Smoking trend			
	Estimate	SE	Z	P-value	Estimate	SE	Z	P-value
Baseline negative affect	–	–	–	–	0.03	0.01	3.61	<0.0001
<i>Baseline positive affect</i>	–	–	–	–	–0.03	0.01	–4.00	<0.0001
White	0.32	0.12	2.62	0.01	0.04	0.03	1.08	0.28
Parental monitoring	–0.02	0.02	–1.03	0.31	0.00	0.01	–0.15	0.88
Impulsivity	0.06	0.03	1.87	0.06	0.01	0.01	0.73	0.47
Peers smoking	0.19	0.03	7.60	<0.0001	–0.01	0.01	–1.44	0.15
Household smoking	0.58	0.11	5.22	<0.0001	–0.02	0.03	–0.78	0.43
Parental education	0.15	0.06	2.42	0.02	–0.02	0.02	–1.43	0.15
Alcohol use	0.25	0.06	3.98	<0.0001	–0.01	0.02	–0.28	0.78
Marijuana use	0.33	0.07	4.68	<0.0001	0.00	0.02	0.08	0.94
Interpersonal distress	–0.03	0.03	–0.77	0.44	–0.05	0.01	–3.58	<0.0001
Somatic symptoms	0.03	0.01	2.11	0.04	–0.01	0.01	–1.99	0.05

	Baseline positive affect level				Positive affect trend			
	Estimate	SE	Z	P-value	Estimate	SE	Z	P-value
Baseline smoking	–	–	–	–	0.00	0.02	–0.08	0.94
Baseline negative affect	–	–	–	–	–0.08	0.01	–9.10	<0.0001
White	0.25	0.20	1.30	0.20	–0.03	0.05	–0.54	0.59
Parental monitoring	0.21	0.03	6.48	<0.0001	–0.03	0.01	–3.49	<0.0001
Impulsivity	0.05	0.06	0.86	0.39	0.00	0.02	–0.21	0.83
Peers smoking	–0.12	0.04	–3.14	0.002	0.03	0.01	2.49	0.01
Household smoking	–0.28	0.18	–1.58	0.12	0.09	0.05	1.77	0.08
Parental education	–0.18	0.11	–1.66	0.10	0.01	0.03	0.35	0.73
Alcohol use	–0.16	0.11	–1.42	0.16	0.03	0.03	1.02	0.31
Marijuana use	–0.08	0.14	–0.58	0.56	–0.03	0.04	–0.68	0.49
Interpersonal distress	–0.43	0.06	–6.75	<0.0001	0.13	0.02	6.96	<0.0001
Somatic symptoms	–0.05	0.02	–2.04	0.04	0.04	0.01	5.06	<0.0001

	Baseline negative affect level				Negative affect trend			
	Estimate	SE	Z	P-value	Estimate	SE	Z	P-value
Baseline smoking	–	–	–	–	0.02	0.04	0.61	0.54
Baseline positive affect	–	–	–	–	–0.16	0.01	–11.46	<0.0001
White	–0.61	0.28	–2.18	0.03	0.08	0.08	1.01	0.31
Parental monitoring	–0.08	0.04	–1.84	0.07	0.04	0.01	3.09	0.002
Impulsivity	–0.12	0.08	–1.40	0.16	0.01	0.03	0.28	0.78
Peers smoking	0.26	0.05	5.16	<0.0001	–0.04	0.02	–2.22	0.03
Household smoking	0.47	0.25	1.89	0.06	–0.08	0.08	–1.02	0.31
Parental education	0.00	0.15	0.02	0.99	–0.02	0.05	–0.55	0.58

	Baseline negative affect level				Negative affect trend			
	Estimate	SE	Z	P-value	Estimate	SE	Z	P-value
Alcohol use	0.09	0.17	0.52	0.60	−0.06	0.05	−1.07	0.28
Marijuana use	−0.22	0.19	−1.17	0.24	0.01	0.07	0.17	0.86
Interpersonal distress	1.11	0.08	14.82	<0.0001	−0.18	0.03	−7.09	<0.0001
Somatic symptoms	0.47	0.03	14.24	<0.0001	−0.06	0.01	−5.83	<0.0001

Male model								
	Baseline smoking level				Smoking trend			
	Estimate	SE	Z	P-value	Estimate	SE	Z	P-value
Baseline negative affect	−	−	−	−	0.04	0.01	2.87	0.004
Baseline positive affect	−	−	−	−	0.01	0.01	0.97	0.33
White	−0.15	0.13	−1.18	0.24	0.14	0.05	3.06	0.002
Parental monitoring	−0.04	0.02	−2.21	0.03	−0.01	0.01	−1.31	0.19
Impulsivity	0.00	0.05	−0.10	0.92	0.01	0.01	0.65	0.51
Peers smoking	0.24	0.03	9.50	<0.0001	0.01	0.01	0.94	0.35
Household smoking	0.48	0.11	4.24	<0.0001	0.09	0.04	2.22	0.03
Parental education	0.13	0.07	1.80	0.07	0.02	0.02	0.86	0.39
Alcohol use	0.25	0.07	3.73	<0.0001	0.02	0.02	0.70	0.49
Marijuana use	0.23	0.06	4.16	<0.0001	−0.06	0.02	−2.57	0.01
Interpersonal distress	0.04	0.04	0.94	0.35	−0.02	0.02	−0.91	0.36
Somatic symptoms	0.00	0.02	0.17	0.87	−0.01	0.01	−1.90	0.06

	Baseline positive affect				Positive affect trend			
	Estimate	SE	Z	P-value	Estimate	SE	Z	P-value
Baseline smoking	−	−	−	−	−0.02	0.02	−1.16	0.25
Baseline negative affect	−	−	−	−	−0.09	0.01	−7.04	<0.0001
White	0.10	0.19	0.54	0.59	−0.05	0.05	−0.91	0.36
Parental monitoring	0.20	0.03	7.15	<0.0001	−0.01	0.01	−1.06	0.29
Impulsivity	−0.07	0.06	−1.14	0.26	0.00	0.02	0.07	0.94
Peers smoking	−0.09	0.04	−2.10	0.04	0.02	0.01	1.82	0.07
Household smoking	−0.30	0.18	−1.63	0.10	0.03	0.05	0.56	0.57
Parental education	0.01	0.11	0.13	0.90	0.00	0.03	0.07	0.95
Alcohol use	−0.03	0.12	−0.20	0.84	0.01	0.03	0.26	0.79
Marijuana use	−0.04	0.14	−0.31	0.76	0.05	0.04	1.17	0.24
Interpersonal distress	−0.35	0.06	−6.23	<0.0001	0.12	0.02	6.07	<0.0001
Somatic symptoms	−0.08	0.02	−3.35	0.001	0.03	0.01	3.85	0.00

	Baseline negative affect level				Negative affect trend			
	Estimate	SE	Z	P-value	Estimate	SE	Z	P-value
Baseline smoking	−	−	−	−	0.05	0.03	1.43	0.15
Baseline positive affect	−	−	−	−	−0.16	0.02	−10.37	<0.0001
White	−0.38	0.23	−1.64	0.10	0.09	0.08	1.19	0.23
Parental monitoring	−0.07	0.04	−1.91	0.06	0.02	0.01	1.63	0.10

	Baseline negative affect level				Negative affect trend			
	Estimate	SE	Z	P-value	Estimate	SE	Z	P-value
Impulsivity	0.01	0.08	0.07	0.94	0.02	0.02	0.70	0.49
Peers smoking	0.09	0.05	1.79	0.07	-0.04	0.02	-2.12	0.03
Household smoking	0.50	0.22	2.30	0.02	-0.01	0.07	-0.10	0.92
Parental education	-0.02	0.13	-0.13	0.90	-0.01	0.04	-0.20	0.84
Alcohol use	0.30	0.11	2.74	0.01	-0.04	0.05	-0.95	0.35
Marijuana use	0.03	0.13	0.21	0.84	-0.02	0.05	-0.31	0.76
Interpersonal distress	1.01	0.07	14.15	<.0001	-0.14	0.02	-5.96	<0.0001
Somatic symptoms	0.37	0.03	12.91	<.0001	-0.05	0.01	-5.78	<0.0001

SE = standard error