

Stress and Smoking in Adolescence: A Test of Directional Hypotheses

Thomas Ashby Wills and James M. Sandy
Albert Einstein College of Medicine of Yeshiva University

Alison M. Yaeger
Ferkau Graduate School of Psychology of Yeshiva University

The authors conducted a comparative test of the hypotheses that (a) stress is an etiological factor for smoking and (b) cigarette smoking causes increases in stress (A. C. Parrott, 1999). Participants were a sample of 1,364 adolescents, initially surveyed at mean age 12.4 years and followed at 3 yearly intervals. Measures of negative affect, negative life events, and cigarette smoking were obtained at all 4 assessments. Latent growth modeling showed negative affect was related to increase in smoking over time; there was no path from initial smoking to change in negative affect. Comparable results were found for negative life events, with no evidence for reverse causation. Results are discussed with respect to theoretical models of nicotine effects and implications for prevention.

Key words: stress, smoking, adolescence, negative affect, substance use

Studies of several types of populations have noted that stress is linked to cigarette smoking (Wills, 1990). Measures of negative affect and negative life events have been linked to maintenance and relapse for smoking among adults (e.g., Anda et al., 1990; Cohen & Lichtenstein, 1990; Cohen & Williamson, 1988; Shadel & Mermelstein, 1993). A linkage with stress has also been found for alcohol use (e.g., Brown, Vik, Patterson, Grant, & Schuckit, 1995; Hussong & Chassin, 1994) and opiate use (e.g., Shaham, 1996). The observed relationship of stress and smoking has been a basis for theoretical models that include constructs of affect regulation and coping functions (Shiffman et al., 1986; Wills & Cleary, 1995). This concept is consistent with laboratory evidence (Gilbert, 1979, 1995) and with the self-reports of smokers, who have commonly reported that smoking provides coping functions (U.S. Surgeon General, 1988). From a clinical standpoint, understanding the nature of the stress–smoking relationship has significant implications for smoking cessation treatment.

Studies of adolescent populations have also found that smoking is positively related to indices of negative affect and life stress. This has been found in cross-sectional surveys and experience-

sampling studies in the United States and other countries (Castro, Maddahian, Newcomb, & Bentler, 1987; Lloyd & Lucas, 1998; Patton et al., 1996; Sussman et al., 1993; Whalen, Jamner, Henker, & Delfino, 2001). In addition, prospective studies have found stress measures to be predictive of smoking onset and escalation (Dugan, Lloyd, & Lucas, 1999; Seltzer & Oechsli, 1985; Sussman & Dent, 2000; Wills, 1986). Thus, the endeavor to obtain a correct theoretical understanding of the stress–smoking relationship also has implications for smoking prevention research.

The empirical findings noted above are consistent with an etiological model in which negative affect is a risk factor for smoking. However, a recent article by Parrott (1999) has suggested a different perspective, interpreting previous findings as indicating reverse causation (i.e., smoking increases stress) and positing that neurochemical changes induced by smoking make individuals more prone to anxiety, depression, and irritability. Because of the implications for prevention and treatment, the hypothesis that smoking increases negative affect needs empirical examination in studies with appropriate designs, and tests of the hypothesis with adolescents seem particularly warranted because of the implications for prevention research. In the present study, we tested the hypothesis in a representative sample of adolescents followed over time with repeated assessments of affect and smoking. The following sections discuss prior evidence and the methods of the present research.

Prior Evidence on Affect and Smoking in Adolescence

Parrott (1999) cited cross-sectional studies of adolescents that found positive correlations between stress measures and smoking indices (Lloyd & Lucas, 1998; Mitic, McGuire, & Neumann, 1985). For example, Mitic et al. (1985) found that indices of perceived stress from various domains were highest in regular smokers, intermediate in occasional smokers, and lowest in non-smokers. Wills, Vaccaro, and McNamara (1992) found that a measure of negative affect (in the past month) and a measure of negative life events (during the past year) were positively related

Thomas Ashby Wills and James M. Sandy, Department of Epidemiology and Social Medicine, Albert Einstein College of Medicine of Yeshiva University; Alison M. Yaeger, Ferkau Graduate School of Psychology of Yeshiva University.

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Correspondence concerning this article should be addressed to Thomas Ashby Wills, Department of Epidemiology and Social Medicine, Albert Einstein College of Medicine of Yeshiva University, 1300 Morris Park Avenue, Room 1301, Belfer Building, Bronx, New York 10461. E-mail: wills@aecom.yu.edu

to smoking and alcohol use. Parrott noted correctly that the causal interpretation of such findings is ambiguous; the positive correlation could indicate that stress increases smoking, but it could indicate that smoking increases stress.

Also discussed by Parrott (1999) was a retrospective study by Hirschman, Leventhal, and Glynn (1984) in which a sample of school students (Grades 2–10) were interviewed about their first, second, and third smoking experiences. An index termed *helplessness* (i.e., feeling dissatisfied with aspects of life and having given up trying to improve things) was significantly related to movement to second and third cigarettes after initial trial. This was interpreted by Parrott as indicating that an increase in affective distress accompanied the move from experimental to regular smoking. However, it is not clear that this interpretation is warranted, because the interviews were retrospective and the temporal sequence of helplessness and smoking transitions was not indicated by the data.

Finally, Parrott (1999) discussed a study by Wills (1986). In this study, samples of junior high school students were surveyed on four occasions over a 2-year period, and multiple regression was used to predict changes in smoking and alcohol use from stress measures at baseline. Significant prospective effects were found for several stress indices, which were related to increases in smoking and alcohol use over time. Parrott suggested that Wills's (1986) study showed that "stress levels increased in children who became more frequent smokers" (Parrott, 1999, p. 818). However, this summary of the results was incorrect; no such finding was reported in Wills's article.

To summarize, Parrott's (1999) discussion of previous findings, with respect to his theoretical framework, had mixed success. Although ambiguity in the interpretation of cross-sectional findings was appropriately noted, the interpretations of retrospective evidence (Hirschman et al., 1984) and prospective results (Wills, 1986) were not always warranted. However, previous studies have not directly tested whether prior smoking influences negative affect over time. If such a result were found, this would be consistent with Parrott's hypothesis and would not contradict previous empirical findings.

Present Research

We conducted longitudinal analyses to test hypotheses about stress and smoking in adolescence because of the implications for etiology and prevention research. If smoking actually increases negative affect over time, this would lend support to Parrott's (1999) arguments. The hypothesis that negative affect is a risk factor for increase in smoking over time is more consistent with theoretical models positing that smoking has affect-regulation functions (Pomerleau & Pomerleau, 1984; Wills & Shiffman, 1985) and with research showing that among users, measures of perceived coping functions are strongly related to current levels of smoking and alcohol use (Cooper, Frone, Russell, & Mudar, 1995; Wills, Sandy, & Shinar, 1999).

The present research was based on a sample of adolescents who were surveyed on four occasions at yearly intervals over the period from 12 through 16 years of age, the time during which smoking onset and escalation occur for the majority of the population (Johnston, O'Malley, & Bachman, 1995). Measures for two stress indices, negative affect and negative life events, and for cigarette smoking were obtained at all assessments. Thus, the study could

compare the hypotheses (not mutually exclusive) that (a) stress increases smoking and/or (b) smoking increases stress.

For evaluating the hypotheses, we used latent growth curve modeling (Willett & Sayer, 1994). This analytic approach has several advantages over autoregressive approaches (Windle, 1997) because the latent growth approach is based on an individual growth curve for each participant, can distinguish between initial level of a variable (i.e., intercept) and rate of growth of the variable over time (i.e., slope), and does not impose statistical assumptions about the relation between intercept and slope. To test the relationships between constructs, we specified an associative model in which intercept and slope constructs for stress and for smoking were both included. This provided a specific test of whether stress is a risk factor for smoking onset and escalation versus the alternative hypothesis that smoking is related to increase in stress over time. Gender differences in stress–smoking relationships were considered, and analyses for relations of stress with alcohol and marijuana use were also conducted.

Method

Questionnaires were administered to participants in a school-based longitudinal study with follow-ups conducted at yearly intervals; the study began in 7th grade and continued through 10th grade. The questionnaire had measures of negative affect and negative life events and included measures of cigarette smoking and other types of substance use.

Participants

The participants were students in public school districts in a metropolitan area; the students initially were in six junior high schools and subsequently were in four high schools. At the initial survey administration, the mean age was 12.4 years ($SD = 0.7$); the sample was 47% female and 53% male. Ethnic identification was 29% African American, 24% Hispanic, 3% Asian American, 37% Caucasian, 3% mixed ethnicity, and 5% other ethnicity. Data indicated that 53% of participants were living with two biological parents, 34% were in a single-parent structure, and 13% were in a blended family (one biological parent and one stepparent). Data on parental education indicated the mode was high school graduate and the mean on a 1–6 scale was 3.7 ($SD = 1.3$), a level just above high school graduate.

Data collection involved a self-report questionnaire administered to students in classrooms by trained research staff. The staff members followed a standardized protocol in giving instructions to students and answering questions about individual items. The survey was administered under confidential conditions, and participants were informed not to write their names on the questionnaires. The study data were covered by a Certificate of Confidentiality from the U.S. Public Health Service; participants were informed that their responses were totally confidential and would not be known by their parents or teachers. With the questionnaire administration, a sample of expired air was obtained and was analyzed for carbon monoxide (CO) with automated equipment using the Breath CO Analyzer (Vitalograph Corporation, Lenexa, KS). The CO results were labeled with only a code number, and students were informed that these data also were totally confidential.

Students participated under a consent procedure in which parents were sent, by direct mail, a notice that informed them about the purpose and nature of the study. Parents were informed that they could exclude their child from the research, if they wished, by notifying the investigator or the school. Students were informed about the purpose and nature of the research at the time of questionnaire administration and were instructed that they could refuse or discontinue participation. The completion rate (number of students providing completed surveys divided by total eligible

population from school lists) was 92% in 7th grade, 88% in 8th grade, 84% in 9th grade, and 83% in 10th grade. Rates of parental or student exclusion were in the range of 1–3%; the primary source of case loss was from student absenteeism, which increased over the course of the study. As the study continued, students new to the schools were surveyed in addition to continuing participants. The sample size was 1,702 participants in 7th grade, 1,827 in 8th grade, 1,895 in 9th grade, and 1,699 in 10th grade. The overall retention rate for the study variables was approximately 70%.

Measures

Demographics. The participant was asked about his or her age, gender, and ethnicity (five options, multiple responding allowed). An item on family structure asked what adult(s) the participant currently lived with (eight options, multiple responding allowed); this was recoded for analysis to three levels (single parent, blended family, or intact family). Questions about the level of education for mother and father, respectively, had responses on 6-point scales with anchor points *grade school* and *post-college*.

Negative affect. An affective-state inventory used an adaptation of the adjective checklist developed by Zevon and Tellegen (1982). Items were introduced to the participant with the stem “Below are some words that may describe how you felt during the last month. Read each one and circle a number to show if you felt this way.” Responses were on a 0–4 scale with anchor points *not at all true* and *very true*. Negative affect was assessed through 12 items (e.g., tense, dissatisfied with things, sad, worried, hostile, irritated, upset). Internal consistency for the 12-item negative affect scale (Cronbach’s alpha) was .88–.91 over the four assessments.

Negative life events. Stress from life events during the previous year was assessed with a 20-item inventory of negative events based on a previous measure (Wills et al., 1992). Items were introduced with the stem “Here are some things that may happen in people’s lives. Read each one and check a space No or Yes to show whether this happened for you *during the past year*.” The inventory included 11 events that could happen to a family member (e.g., “My father/mother lost his/her job”) and 9 items that could happen directly to the respondent (e.g., “I had a serious illness”). Responses were on a 0–1 scale with response points *No* and *Yes*. The measure was scored for the unweighted total frequency of life events. Alpha for the 20-item inventory was .67–.71 over assessments.

Substance use. Substance use by the participant was assessed with items that asked about the typical frequency of his or her cigarette, alcohol, or marijuana use. Items were introduced to the participant with the stem “Here are some simple questions about substances. Please give the best answer you can for each one. Remember to check only one answer for each question.” The item for cigarette smoking asked “How often have you smoked cigarettes?” Responses were on a 0–5 scale with response points *never smoked in my life, tried one or two cigarettes, used cigarettes four or five times, usually smoke a few cigarettes a month, usually smoke a few cigarettes a week, and usually smoke cigarettes every day*. Similar items asked the respondent how often he or she drank alcohol and how often he or she smoked marijuana. An item on heavy drinking asked “Was there a time in the past month when you had 3 or more drinks (beer, wine, or liquor) on one occasion?” Responses were on a 0–3 scale with anchor points *no, yes, it happened once, yes, it happened twice, and yes, it happened more than twice*.

Results

Data on prevalence of cigarette smoking indicated a steady increase over time. For example, the percentage of participants who indicated they had never smoked decreased from 66% in 7th grade to 39% in 10th grade. The percentage of participants who indicated they usually smoked on a daily basis increased from 1% in 7th grade to 13% in 10th grade. Thus, there was a marked

increase in smoking over the study period. Similar increases were noted for alcohol and marijuana use. Data from the CO measure indicated that self-reports of cigarette smoking had good validity (Wills & Cleary, 1997).

Descriptive statistics for the study variables are presented in Table 1. Distributions for negative affect and negative life events were shifted toward the lower end of the scales (with skewness values positive by convention), but skewness figures were in the moderate–low range. Distributions for substance-use indices were positively skewed because the majority of the sample were not using substances in the seventh grade; this changed as the participants grew older and rates of substance use increased. Overall, skewness values for the smoking and alcohol indices were mostly in the 1.0–2.0 range. Marijuana use was more skewed because there were very low rates of use at baseline. Smoking showed a steady increase over time, as did the other substance use indices. Negative affect also showed a steady increase over time. There was no consistent pattern of change over time for negative life events. The stability correlation for negative affect averaged .57 for 1-year lags, .50 for 2-year lags, and was .43 for the 3-year lag. The stability correlation for negative life events averaged .56 for 1-year lags, .44 for 2-year lags, and was .34 for the 3-year lag.

Tests for attrition were based on *t* tests that contrasted participants who dropped out over various lags with those who continued. The nature of attrition was comparable over 1-, 2-, and 3-year lags. In most cases, there was no significant difference on negative affect between continuing participants and dropouts. There was evidence of differential attrition for life events, where dropouts had higher levels ($p < .0001$ in all tests), and for smoking, where dropouts had higher levels ($p < .001$ in all tests). The magnitude of the attrition effect was that dropouts had approximately one more negative life event and scored approximately 0.3 scale points higher on smoking compared with continuing participants.

Table 1
Descriptives for Study Variables for Four Assessments

Variable	Grade			
	7th	8th	9th	10th
Negative affect				
<i>M</i>	14.78	16.01	16.88	18.21
<i>SD</i>	10.26	11.55	11.78	12.75
Skewness	0.57	0.63	0.54	0.48
Negative life events				
<i>M</i>	4.11	4.18	4.16	4.29
<i>SD</i>	2.88	2.97	3.06	3.09
Skewness	0.87	0.92	0.97	0.88
Smoking				
<i>M</i>	0.54	0.86	1.28	1.47
<i>SD</i>	0.95	1.26	1.60	1.70
Skewness	2.39	1.77	1.24	1.04
Heavy drinking				
<i>M</i>	0.16	0.22	0.37	0.53
<i>SD</i>	0.44	0.53	0.81	0.96
Skewness	2.83	2.33	2.27	1.69
Marijuana use				
<i>M</i>	0.04	0.09	0.37	0.65
<i>SD</i>	0.32	0.48	0.99	1.26
Skewness	10.59	6.79	3.13	2.07

Note. Range for negative affect, 0–48; for life events, 0–20; for smoking and marijuana use, 0–5; for heavy drinking, 0–3.

Latent Growth Model of Directional Effects for Stress and Smoking

To evaluate the directional hypotheses, an associative model was first specified for affect and smoking constructs (Duncan, Duncan, Strycker, Li, & Alpert, 1999). Latent constructs were based on four observations each for negative affect and cigarette smoking (taken at 7th, 8th, 9th, and 10th grades); the analysis was restricted to cases who had at least three measures of substance use over the study period. The construct representing initial level for negative affect (intercept) was specified by setting loadings of the four indicators for negative affect on the intercept construct to 1. The construct representing rate of change in negative affect over time (slope) was specified by setting loadings for the indicators of negative affect at each time point on the slope construct to 0, 1, 2, and 3, reflecting the modeling of linear growth with equal spacing of observations over time. Similarly, a construct representing initial level of smoking (intercept) was specified by setting loadings of the four indicators for smoking on the intercept construct to 1, and a construct representing rate of change in smoking over time (slope) was specified by setting loadings for the indicators of smoking at each time point on the slope construct to 0, 1, 2, and 3.

The associative model was formulated by including, in one model, the intercept and slope constructs for negative affect and the intercept and slope constructs for adolescent smoking. The model included a covariance of the intercepts for affect and smoking, covariances of the intercept and slope constructs for affect and of the intercept and slope constructs for smoking, and a covariance of the slopes for affect and smoking. The directional hypotheses were tested by specifying a path from initial negative affect to change in smoking over time, and a path from initial smoking to change in negative affect over time. The latter path, if significant, would support the reverse-causation argument of Parrott (1999). To control for any correlations with demographics, the model included, as exogenous variables, indices for gender, ethnicity (African American vs. Hispanic or Caucasian, and Hispanic vs. African American or Caucasian), family structure (single vs. blended or intact, and blended vs. intact or single), and parental education.

The model was analyzed in Mplus (Muthén & Muthén, 1998) with the maximum-likelihood method, using the expectation-maximization (EM) algorithm for missing data.¹ Demographic effects were included if they had $p < .05$, and correlated errors if they had $p < .0001$. The model with one correlated-error term had good fit to the data, with χ^2 (62, $N = 1,364$) of 123.46 and root-mean-square error of approximation (RMSEA) of 0.027 (confidence interval [CI] = .020–.034). The model is presented in Figure 1. Correlations of the exogenous variables, included in the model but omitted from the figure for graphical simplicity, are presented in Table 2. The relation of intercept and slope was nonsignificant for both affect and smoking constructs. Effects for demographics were such that single and blended family structures had positive paths to smoking intercept, parental education had an inverse path to smoking intercept, female gender had positive paths to negative affect intercept and slope and to smoking slope, and African American ethnicity and Hispanic ethnicity had inverse paths to smoking slope.

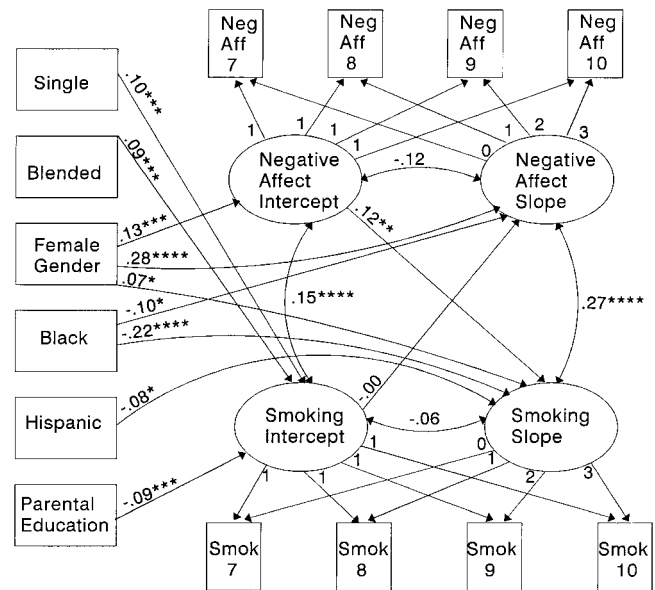


Figure 1. Associative latent growth model with intercept and slope constructs for negative affect and adolescent smoking, with demographic characteristics as exogenous variables. All values are standardized coefficients except for factor loadings, which are fixed. Curved double-headed arrows represent covariances; single-headed arrows represent path effects. Covariances of exogenous variables are presented in Table 2. (Smok = smoking; Neg Aff = negative affect. * $p < .05$. ** $p < .01$. *** $p < .001$. **** $p < .0001$.)

With regard to the directional hypotheses, the path from negative affect intercept to smoking slope was significant ($\beta = .12, p < .01$). There was no significant path from smoking intercept to negative affect slope ($\beta = .00, ns$). There was a significant correlation of the intercept constructs for negative affect and smoking ($r = .15, p < .0001$), and a significant correlation of the slope constructs for affect and smoking ($r = .27, p < .0001$). Thus, the results supported the hypothesis of a relation of negative affect to increase in smoking, which is also consistent with the positive correlation of the slopes. There was no evidence for reverse causation.

¹ Measurement models for the growth constructs were tested in Mplus. All intercept constructs were different from zero and had significant variability ($p < .0001$). (For all t tests, $df = 1,364$.) For negative affect, the measurement model had good fit, $\chi^2(5, N = 1,364) = 12.42$ and root-mean-square error of approximation (RMSEA) = .033 (confidence interval [CI] = .010–.057). The mean slope was 1.21 ($SE = .12$), different from zero, $t = 10.16, p < .0001$; the variance for the slope was 1.21 ($SE = .119$), also significant, $t = 6.49, p < .0001$. For negative life events, the measurement model had reasonable fit, $\chi^2(5, N = 1,364) = 25.43$ and RMSEA = .055 (CI = .035–.077). The mean slope was 0.04 ($SE = .03$), not different from zero, $t = 1.31, ns$; the variance for the slope was 0.51 ($SE = .07$), which was significant, $t = 7.75, p < .0001$. For smoking, the measurement model had reasonable fit, $\chi^2(5, N = 1,364) = 24.45$ and RMSEA = .058 (CI = .039–.086). The mean slope was 0.34 ($SE = .01$), different from zero, $t = 24.94, p < .0001$, and the variance for the slope was 0.21 ($SE = .01$), also significant, $t = 17.33, p < .0001$. Thus, the constructs met the necessary assumptions for latent growth analysis, though it should be noted that the slope for life events was essentially zero.

Table 2
Correlations of Exogenous Variables for Model

Variable	1	2	3	4	5	6
1. Gender	—					
2. African American	.00	—				
3. Hispanic	.02	-.42 ^a	—			
4. Single parent	.04	.23	.03	—		
5. Blended family	.01	.04	.12	-.24 ^a	—	
6. Education	.03	.11	-.28	-.07	-.02	—

Note. $N = 1,364$ for correlations. Approximate significance levels are $r > |.07|$, $p < .01$; $r > |.12|$, $p < .0001$.

^a This correlation is a consequence of the coding procedure.

A similar model was analyzed with negative life events as the stress index.² This associative model with one correlated-error term had good fit to the data, $\chi^2(59, N = 1,364) = 148.91$ and $RMSEA = 0.033$ ($CI = .027-.040$). The relation of intercept and slope was nonsignificant for smoking ($r = -.04$) and was significant for life events ($r = -.39$, $p < .0001$). Effects for demographics (aside from those previously noted) were such that life events intercept had positive paths from single family structure ($\beta = .15$, $p < .0001$), blended family structure ($\beta = .15$, $p < .0001$), African American ethnicity ($\beta = .09$, $p < .01$), and Hispanic ethnicity ($\beta = .11$, $p < .001$), and inverse paths from female gender ($\beta = -.09$, $p < .01$) and parental education ($\beta = -.09$, $p < .001$); life events slope had a positive path from female gender ($\beta = .19$, $p < .0001$).

With regard to the directional hypotheses, the path from life events intercept to smoking slope was significant ($\beta = .23$, $p < .0001$). There was a significant inverse path from smoking intercept to life events slope ($\beta = -.16$, $p < .001$), indicating that persons with higher levels of smoking at baseline had decreases in negative events over time. There was a positive correlation of the intercepts for smoking and life events ($r = .29$, $p < .0001$) and a positive correlation of the slopes ($r = .26$, $p < .0001$). Thus, these results supported the hypothesis of negative life events as a risk factor for increase in smoking, also consistent with the positive correlation of the slopes for life events and smoking. The inverse path from initial smoking to change in life events was inconsistent with the hypothesis of reverse causation.³

Test for Gender Differences

Possible gender differences in the influence of affective variables were tested with multiple-group analysis. The sample was divided into gender subgroups, and the associative model, minus demographics, was analyzed simultaneously in both subgroups with all parameters freely estimated (base model). Across-group equality constraints were then imposed to determine whether the two paths from intercepts to slopes differed by gender, indicated by a significant chi-square difference for the constrained model compared with the base model. The multiple-group models had 644 cases for girls and 720 cases for boys. The base model for negative affect had good fit, $\chi^2(40, N = 1,364) = 66.83$ and $RMSEA = 0.031$ ($CI = .017-.044$), and the base model for negative life events had reasonable fit, $\chi^2(40, N = 1,364) = 123.76$ and $RMSEA = 0.055$ ($CI = .044-.067$). Table 3 presents the coefficients from the base models by gender subgroups. Un-

standardized coefficients are reported because standardized coefficients cannot be directly compared across groups.

Results showed that paths for the stress indices had similar magnitudes for boys and girls. The paths from initial stress levels to increase in smoking were significant for both groups. The path from initial smoking to change in negative affect was nonsignificant for both groups, and the path from initial smoking to change in life events was inverse and significant in both subgroups. Constraining these paths to be equal across subgroups in the model for negative affect resulted in $\Delta\chi^2(2) = 0.27$, *ns* and in the model for negative life events resulted in $\Delta\chi^2(2) = 0.25$, *ns*. These analyses indicated relations between stress and smoking were comparable for boys and girls.

Relations for Other Substances

To examine the possibility that processes in affect–substance use relationships might differ for other substances, the model in Figure 1 (including demographics) was analyzed for indices of alcohol and marijuana use. Results for negative affect and heavy drinking are presented in Figure 2A.⁴ The model had good fit to the data, $\chi^2(67, N = 1,364) = 145.58$ and $RMSEA = 0.029$ ($CI = .023-.036$). There was a significant path from initial negative affect to growth in heavy drinking over time ($p < .05$) but a nonsignificant path from initial heavy drinking to growth in negative affect, together with significant positive correlations of the intercepts ($p < .01$) and the slopes ($p < .0001$). This same model analyzed with negative life events as the stress index had $\chi^2(64, N = 1,364) = 139.77$ and $RMSEA = 0.029$ ($CI = .023-.036$). There was a significant path from initial life events to growth in heavy drinking ($\beta = .17$, $p < .01$) and an inverse path from initial heavy drinking to change in life events ($\beta = -.34$, $p < .0001$), together with significant positive correlations of the intercepts ($r = .36$, $p < .0001$) and of the slopes ($r = .38$, $p < .0001$). These models are consistent with data for smoking in showing an effect of stress on growth in heavy alcohol use, with no evidence for reverse causation.

Results for marijuana use are presented in Figure 2B. The model for negative affect had good fit to the data, $\chi^2(63, N = 1,364) = 120.21$ and $RMSEA = 0.026$ ($CI = .019-.033$). There was a significant path from initial negative affect to growth in marijuana use over time ($p < .01$) but no significant path for initial marijuana use to growth in negative affect. There were positive correlations

² This analysis used an 18-item score for life events, with items on parental divorce and remarriage dropped to avoid confounding with the family structure indices.

³ The study included a 12-item scale for positive affect (from Zevon & Tellegen, 1982), and a latent growth model indicated the path from initial positive affect to growth in smoking was $\beta = -.11$ ($p < .01$), whereas the path from initial smoking to change in positive affect was $\beta = .03$ (*ns*). The correlation of the intercepts was $r = -.18$ ($p < .0001$), and the correlation of the slopes was $r = -.08$ ($p < .05$). Thus, there was evidence that positive affect reduced growth in smoking, with no evidence for reverse causation.

⁴ The heavy drinking index was used for these analyses because it had stronger relations with stress than the overall alcohol use index. Two factor loadings for marijuana slope were freed because growth was not strictly linear.

Table 3
Unstandardized Coefficients From Base Models in Multiple-Group Analysis for Girls and Boys

Path	B		SE		t	
	Girls	Boys	Girls	Boys	Girls	Boys
Affect intercept→ smoking slope	.007	.009	.003	.003	2.11*	2.66**
Smoking intercept→ affect slope	-.045	.032	.191	.210	ns	ns
Events intercept→ smoking slope	.030	.034	.012	.009	2.52**	3.72***
Smoking intercept→ events slope	-.169	-.214	.083	.087	2.02*	2.47**

* $p < .05$. ** $p < .01$. *** $p < .001$.

of the intercepts ($p < .05$) and of the slopes ($p < .01$). This same model analyzed with negative life events as the stress index had $\chi^2(63, N = 1,364) = 150.03$ and RMSEA = 0.032 (CI = .025–.038). There was a significant path from initial life events to growth in marijuana use ($\beta = .31, p < .0001$) and an inverse path from initial marijuana use to change in life events ($\beta = -.24, p < .01$). There were positive correlations of the intercepts ($r = .25, p < .0001$) and of the slopes ($r = .27, p < .0001$). Thus, these models showed paths from stress to growth in marijuana use over

time, with cross-paths that were inconsistent with the hypothesis of reverse causation.

Discussion

This research evaluated directional hypotheses about the relation between stress and smoking on the basis of latent growth analyses of data from a representative sample of adolescents. Self-reports of smoking were accompanied by a biochemical measure, which showed that the reports of smoking had good validity (Wills & Cleary, 1997). Tests of measurement models showed that negative affect constructs and smoking constructs met the statistical assumptions for growth modeling. Results showed significant relations of stress indices to increase over time in smoking, with comparable findings for measures of negative affect and negative life events. There was no evidence that smoking had a role in causing increase in stress over time. These results were obtained in a large sample followed from early through middle adolescence, and the findings were independent of demographics.

The aim of this research was to test alternative theoretical models of the relationship between stress and smoking. The risk-factor model posits that stress increases the likelihood of smoking; the alternative model suggested by Parrott (1999) is that there is no causal effect of life stress but rather that smoking acts to increase negative affect. Studying the relation between stress and smoking over time provides a direct test of the hypotheses, and latent growth modeling provides a fair comparative test of alternative models of the stress–smoking relationship. With this approach, we found consistent evidence for stress as an etiological factor for smoking, and found no support for a reverse-causation model. The observed support for the etiological model is consistent with a range of laboratory and epidemiologic evidence (Gilbert, 1995), lending more confidence to this theoretical model of smoking.

The present findings derive from a research context in which relations between stress and smoking were studied over a 3-year period. This research context is not necessarily mapped onto the time spans examined in laboratory studies or in research on daily episodes of smoking, but we note that it is of interest for prevention research to determine how stress effects operate in such a time frame because prevention studies are typically conducted over periods of several years. It is possible that studies focused on different time spans might provide a different perspective, though commentators on the Parrott (1999) article have noted several inaccuracies in his citations and have noted that there is considerable evidence from laboratory and clinical research that stress

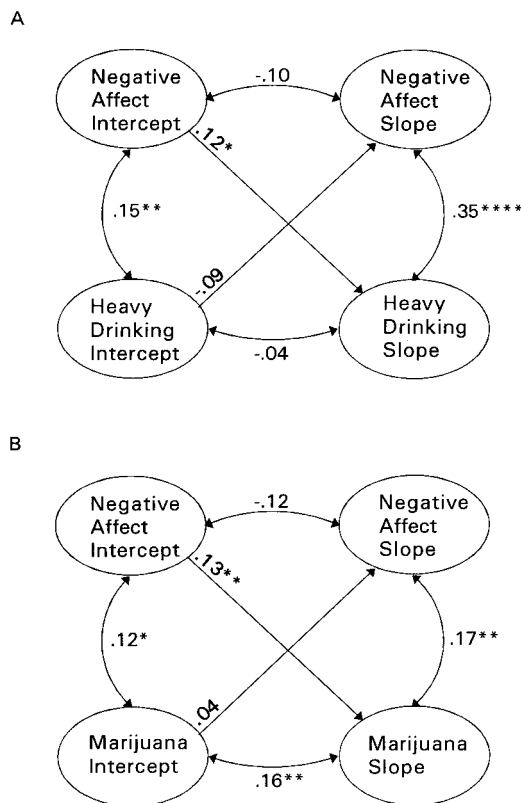


Figure 2. Associative models for negative affect and substance use for heavy drinking (A) and for marijuana use (B). All values are standardized coefficients except for factor loadings, which are fixed. Curved double-headed arrows represent covariances; single-headed arrows represent path effects. Demographic variables were included in the models but are not represented in the figures. * $p < .05$. ** $p < .01$. **** $p < .0001$.

increases smoking and that smoking can ameliorate negative affective states (Gilbert & McClernon, 2000; Kassel, 2000).

Some aspects of the present research could be noted as possible limitations. The study used two indices of stress, and though they are reasonable measures for a test of the theoretical models (Pia-secki & Baker, 2000), other measures of anxiety and depression could be used in further research, including diagnostic measures of affective and anxiety disorders. There was some evidence of differential attrition, but this would operate to reduce the variability of stress within the analytic sample and make it more difficult to confirm the hypothesis. The measure of cigarette smoking focused on frequency of use and did not directly index addictive or dependent smoking; these aspects of smoking behavior could be studied in further research. Finally, the results were obtained during early to middle adolescence, and relationships of stress and smoking may be studied at later ages to test how these processes operate during other parts of the life span.

Demographic Effects

The study showed some noteworthy demographic effects. Gender effects were such that girls had higher initial levels of negative affect and a greater rate of increase in negative affect over time; in addition, there was a path from female gender to rate of increase in smoking. Though multiple-group analyses showed that the impact of negative affect was comparable for boys and girls, it was evident that girls had more negative affect to begin with and were more likely to escalate in smoking once they started. These effects would operate to produce greater vulnerability for smoking (Gritz, 1986; Khoury, 1998). Further longitudinal research may help to clarify the basis for gender differences in smoking.

Ethnic differences were such that African American and Hispanic respondents showed lower rates of increase in smoking over time, even though they experienced higher initial levels of negative life events. This finding suggests the existence of protective factors associated with ethnicity, which may be attitudinal or cultural in nature (Brook, Balka, Brook, Win, & Gursen, 1998). The nature of these factors is not well understood at the present time, and further research may be designed to provide a better understanding of these factors (Brook, 1993; Vega, Gil, & Kolody, 1998).

Evaluation of Alternative Models

The article by Parrott (1999) presented several arguments about the relation between negative affect and smoking. One argument was that smoking may increase negative affect because smokers frequently experience withdrawal states, and Parrott suggested this is typical of adolescents as well as adults. The present results seem inconsistent with this position, because similar effects of stress indices were found for alcohol and marijuana use as well as for smoking. Use of these former substances at levels typical of adolescents has not been suggested as producing withdrawal states; hence, the findings for these substances are incompatible with the argument that negative affect is elevated among users by withdrawal states.

Another suggestion raised by Parrott (1999) is that the relation of stress and smoking may be spurious, actually attributable to a third factor, neuroticism (i.e., stably elevated negative affect). If this argument were correct, the analyses would show negative

affect related to initial levels of smoking but there should be no correlation of changes in affect and smoking over time. The present results are not supportive of this position. Though there was a correlation of initial levels, a path was found from negative-affect intercept to smoking slope, and there was a positive correlation between the slopes for negative affect and smoking. Elevated negative affect may be a predisposing factor for onset, but the present findings are contradictory to a model in which relationships between affect and smoking are spurious. Instead, they support the position that negative affect is an etiological factor for escalation of smoking.

The Relation Between Stress and Smoking

A question raised by the findings concerns the mechanism of the relation between stress and smoking. Several articles have posited that cigarette smoking has functions that are relevant for affect regulation and coping with stress and that these functions are implicated in the relationship between stress and smoking (Gilbert, 1995). Both smoking and alcohol use are perceived to provide functions for increasing concentration, countering boredom, enhancing positive mood, and reducing distress in anxiety-arousing conditions (Wills & Cleary, 1995), and these motive reports represent an individual-difference factor that is strongly related to level of use and use-related problems in later adolescence (Cooper et al., 1995; Wills, Sandy, & Shinar, 1999; Wills, Sandy, & Yaeger, 2002). Although evidence from laboratory studies on stress-reduction effects of smoking is not totally consistent (Kassel, 2000), evidence from physiological research has shown that nicotine influences brain reward systems (Pontieri, Tanda, Orzi, & Di Chiara, 1996), and research conducted in the natural environment has shown that smoking helps to modulate negative affect states (Jamner, Shapiro, & Jarvik, 1999). Thus, there is a sound basis for the proposition that cigarette smoking has functions for affect regulation, and further research with a variety of research designs is warranted to explicate the mechanism of such effects.

A specific issue concerns the relationship between smoking and anxiety disorders. A recent study by Johnson et al. (2000) was based on a sample of adolescents assessed at ages 16 and 22 years. Johnson et al.'s analyses indicated that adolescents who smoked at least 20 cigarettes a day at age 16 were at increased risk for generalized anxiety disorder at age 22, controlling for several other variables. This study raises a question of whether smoking causes clinical anxiety disorder. Similar issues have been raised about suicide because epidemiologic studies have shown cigarette smokers to be at increased risk for suicide, controlling for confounders such as chronic illness (M. Miller, Hemenway, & Rimm, 2000). We suggest that the crucial issue is the effects of poor self-control, which may be an underlying factor in smoking as well as in several types of psychopathology (W. R. Miller & Brown, 1991; Sacks & Nelson, 1994; Sher & Trull, 1994; Wills & Stoolmiller, in press) and was not statistically controlled for in these studies. Research is needed to investigate measures of self-control in relation to affective states and substance use.

Issues for Interpretation

We note two ways in which the present findings could be misinterpreted. Our conclusion that stress influences smoking, and

the suggestion that this may reflect the operation of a coping mechanism, does not mean that we view smoking as a desirable way of coping. In addition to the adverse health consequences of smoking, the adoption of substance use as a coping mechanism may operate to undermine the development of more effortful social coping and individual problem-solving strategies (Wills & Hirky, 1996). Thus, there are no good arguments for advocating cigarette smoking as a desirable way of coping with stress, and we are not presenting such a position.

The second issue is that proposing that smoking has coping functions could be taken as portraying cigarette smoking as positive, hence undermining the antismoking message of prevention programs. Our view is that portrayals of the effects of smoking should be balanced and accurate because participants in prevention programs may reject a message that they see as contrary to their own senses and experiences (or those of their friends). We think that a primary aim for prevention programs is to strengthen effortful coping mechanisms and build engagement in alternative sources of reinforcement (Carroll, 1996; Perry et al., 1996; Wills, Sandy, Shinar, & Yaeger, 1999).

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