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Influences of Mood Variability, Negative Moods, and Depression on Adolescent Cigarette Smoking

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Abstract

Understanding the emotional risk factors for cigarette smoking in adolescence can greatly inform prevention efforts. The current study examined prospective relationships between three affective dimensions – negative mood variability, overall negative mood, and depression, affect-related smoking motives, and future smoking patterns among adolescents. The current study expands on prior research by using real-time methods to assess mood and by focusing on a key developmental transition in smoking behavior: the progression from experimentation or low level, infrequent use to higher use. Ninth and 10th grade students (N = 461; 55% girls) provided data on cigarette use at a baseline and follow-up 15-month wave, and also provided ecological momentary assessments of negative moods via palmtop computers for one week at each wave. Negative mood was examined via the means of negative mood reports at each wave, and mood variability was examined via the intraindividual standard deviations of negative mood reports at each wave. Depressive symptoms and smoking motives were also assessed. Findings supported a complex self-medication model of smoking escalation in adolescence whereby mood-smoking relationships differed by affect dimension and gender. For girls, greater negative mood variability at baseline significantly predicted rapid escalation in smoking over time, whereas depressive symptoms and overall negative mood were unrelated to girls' smoking patterns. In contrast, overall negative mood significantly predicted boys' smoking escalation among those with affect-related motives for smoking. Results thus suggest that inconsistent mood-smoking relations in past work may be driven by the complex interrelationships among affect vulnerabilities, gender, and smoking patterns.

Keywords

cigarette smoking; adolescence; moods; affect regulation; depression; ecological momentary assessments

Understanding the factors influencing escalation in youth smoking, from experimentation to more regular cigarette use, remains one of the most important challenges in the adolescent literature. A considerable proportion of Americans initiate cigarette smoking in adolescence, and the majority of adolescents who progress to daily smoking continue such health compromising behaviors well into adulthood (Chassin, Presson, Pitts, & Sherman, 2000). Thus, delineating the pathways to regular cigarette use in adolescence can greatly inform prevention and intervention efforts. To this end, the current study used a multi-method,

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longitudinal approach to examine the influences of mood variability, mood regulation smoking motives, and depression on adolescent smoking escalation.

Numerous studies have shown relationships between depressive symptoms and smoking initiation and regular cigarette use in adolescence (e.g., Audrain-McGovern, Rodriguez, & Kassel, 2009; Brown, Lewinsohn, Seeley, & Wagner, 1996; Choi, Patten, Gillin, Kaplan, & Pierce, 1997; Hedeker, Mermelstein, Berbaum, & Campbell, 2009; Windle & Windle, 2001). The self-medication (Khantzian, 1997, 1999) model proposes a functional relationship between emotional difficulties and smoking, whereby individuals smoke cigarettes to regulate and alleviate a wide range of emotional distress symptoms and states. In turn, the antidepressant effects of smoking and nicotine (Brody, 2006; Tizabi et al., 1999) reinforce cigarette use and increase the likelihood of future use. In support of these models, research has found that adolescents with high levels of depression are twice as likely to be regular smokers (Patton et al., 1996), and adolescent depressive symptoms prospectively predict future cigarette smoking in longitudinal studies (Audrain-McGovern et al., 2009; Killen et al., 1997; Orlando, Ellickson, & Jinnett, 2001; Repetto, Caldwell, & Zimmerman, 2005; Wills, Sandy, Shinar, & Yaeger, 1999). Yet prospective associations between depressive symptomatology and smoking outcomes are not consistently supported in longitudinal studies (e.g., Brown et al., 1996; Choi et al., 1997; Wu & Anthony, 1999). Thus, unanswered questions remain regarding the role of emotional distress in the development of youth smoking.

Although investigations of youth smoking have focused on measures of depression, difficulties with affect regulation in adolescence – versus depression specifically – may be particularly relevant to the development of smoking. Affect regulation refers to a continuum of involuntary to effortful processes involved in initiating and attenuating the quality and intensity of affective responses (Thompson, 1994), including attention shifting, attributions, accessing coping resources, or ability to self-soothe (Silk, Steinberg, & Morris, 2003; Thompson, 1994). Although self-initiated regulatory processes emerge in early to middle childhood (Cole, Martin, & Dennis, 2004; Thompson, 1994), the maturation of neural systems, cognition, and interpersonal skills during adolescence renders this a salient period for the expansion and sophistication of regulatory responses (Kovacs et al., 2006; Spear, 2000). Moreover, adolescents confront unique tasks (e.g., onset of puberty, school transitions, emerging romantic relationships; Cicchetti & Rogosch, 2002) that may tax their affect management capabilities. Affect regulation vulnerabilities during this period may enhance risk for smoking behavior, as youth with underdeveloped internal resources for regulating emotional states are more likely to seek external and maladaptive means of regulation (Eisenberg & Fabes, 1992). Thus, consistent with the self-medication hypothesis, adolescents with limited affect regulation abilities may use cigarettes to modify a range of unpleasant affective states (Khantzian, 1997, 1999). Indeed, research has shown that affect dysregulation in high school and college students predicts frequency of smoking (Wills, Walker, Mendoza, & Ainette, 2006), experimentation with cigarettes and progression to regular use (Novak & Clayton, 2001), and alcohol- and marijuana use problems (Simons & Carey, 2002; Simons, Carey, & Gaher, 2004; Simons, Gaher, Correia, Hansen, & Christopher, 2005). However, this work has been limited by cross-sectional designs and/or use of global, retrospective self-report questionnaires to index affect dysregulation, and such measures may be subject to recall difficulties and judgment biases (Stone & Shiffman, 1994). Thus, findings warrant replication with more sensitive mood assessment as well as longitudinal designs to examine the progression to problematic cigarette use in adolescence.

In the context of dysregulated affective states, cigarette use may be motivated by an individual's beliefs regarding the mood regulatory functions of substance use (Kassel, Stroud, & Paronis, 2003; Wills et al., 1999b). Emotionally labile youth may choose to

smoke as a means of self-regulation if they anticipate mood benefits of smoking, whereas mood lability in the absence of relevant perceived mood-regulatory outcomes may not increase risk for smoking outcomes (Simons et al., 2005). Much evidence suggests that affect-regulation smoking motives and expectations are related to youth smoking behavior (Baker, Brandon, & Chassin, 2004; Kassel et al., 2007; Mayhew, Mott, & Flay, 2000) and predict future smoking (Wetter et al., 2004). Thus, the strength of mood regulatory smoking motives is important to consider when examining a self-medication function of smoking.

The current study investigated one component of affect dysregulation – operationalized as the level of within-individual fluctuation in negative moods (i.e., negative mood variability) – as a risk factor for smoking escalation. Affect regulation and mood variability have been conceptualized as intertwined processes, such that maladaptive regulation of affect manifests in more variable negative emotional states (Forbes & Dahl, 2005; Hoeksma, Oosterlaan, & Schipper, 2004). That is, an individual with limited affect regulation is expected to experience peak intensities of negative emotions, versus a more stable affective profile of an emotionally regulated individual. We thus conceptualize adolescent mood variability as a byproduct of affect regulation, with high levels of negative mood variability reflecting affect dysregulation.

In recent cross-sectional work on this sample (Hedeker et al., 2009) as well as a longitudinal study examining a younger sample earlier in their smoking careers (Weinstein et al., 2008), we examined how negative mood variability related to smoking level. Real-time methods of data assessment (i.e., ecological momentary assessments, EMA: Stone & Shiffman, 1994) were used to assess adolescent mood as it occurred in daily experience, thereby providing an objective index of actual fluctuations in mood and avoiding retrospective bias or summary judgments of affective experience inherent in self-report scales. Examining eighth and 10^{th} graders' smoking patterns across a one-year period, Weinstein and colleagues (2008) found that high levels of negative mood variability significantly differentiated youth who escalated in their smoking behavior over time from those who never progressed beyond limited experimentation. Additionally, Hedeker et al. (2009) examined cross-sectional relations between variance in moods and current smoking level in the current sample, and found increased consistency of mood with higher levels of smoking experience. The present study extends past work by examining negative mood variability in addition to overall levels of negative mood, depression, and mood-related smoking motives to disentangle the prospective relationships between emotional factors and smoking escalation in adolescence, among a sample further along in the progression of smoking than examined in Weinstein et al (2008). Moreover, we build on Hedeker et al.'s (2009) cross-sectional work by examining emotional factors and affect-related smoking motives as predictors of future change in smoking patterns, and by looking more specifically at gender differences. As in past work, EMA were used to examine adolescent mood and mood variability.

The current study first sought to examine the interrelations among mood constructs, with the expectation that high levels of negative mood variability would be associated with greater depressive symptoms and worse overall mood. A second goal was to examine negative mood variability as a predictor of future smoking. In the present study as well as past work (Weinstein et al., 2008), high levels of negative mood variability were conceptualized as a consequence of affect dysregulation. Guided by a proposed self-medication function of smoking to modify a range of unpleasant emotional states, versus to self-medicate depression specifically (Khantzian, 1999), we hypothesized that high negative mood variability at baseline would prospectively predict smoking escalation, differentiating adolescents who escalated to more frequent cigarette use over time from those who did not progress beyond experimentation. A related goal was to compare the impact of mood variability on smoking with overall mood levels and depressive symptoms, in an effort to

identify the most relevant distress state for smoking escalation. Moreover, this study examined whether such mechanisms operated differentially at various points along the continuum of smoking behavior, from early experimentation to escalation to more frequent use. A final goal was to test the moderational role of mood regulation motives (i.e., perceptions of mood-regulatory functions of smoking) in linking affect dysregulation and adolescent smoking behavior. It was hypothesized that affect regulation smoking motives would strengthen the relationship between mood indices and smoking outcomes. In addition, exploratory analyses examined gender differences in the relationships between affective risk factors and smoking behavior. Given the greater prevalence of depressive symptoms and affective lability among adolescent girls versus boys (Hankin et al., 1998; Silk et al., 2003), the relationship between emotional distress and cigarette use may also vary by gender. However, findings are equivocal and warrant further examination. Some researchers have found that depression predicts smoking for girls but not boys (Costello, Erkanli, Federman, & Angold, 1999; Whalen, Jamner, Henker, & Delfino, 2001), whereas other studies demonstrated the reverse relationship (Repetto et al., 2005; Killen et al., 1997) or no gender differences in depression-smoking relations (Brown et al., 1996; Lloyd-Richardson, Papandonatos, Kazura, Stanton, & Niaura, 2002).

The current study builds on prior work by examining mood-smoking associations among a sample of youth currently experimenting with smoking and thus at-risk for progressing in their cigarette use, in an attempt to prospectively capture escalation to more problematic levels of use and dependence during the study period. The current study design allowed for the examination of finer gradations in behavioral patterns of cigarette use than in previous work. Smoking in adolescence has been conceptualized as a sequence of developmental stages of smoking intensity and frequency (Mayhew, Flay, & Mott, 2000), with adolescents progressing from nonsmoking to contemplation, trying (one or two puffs), experimentation, regular smoking, daily smoking, and dependence. Enhancing knowledge of the predictors of change in substance use, particularly the progression to dependence, has important implications for preventing and treating smoking (Flay, Hu, & Richardson, 1998). Accordingly, this study focused on the critical transition from experimentation to regular cigarette use.

Method

Design Overview

Data for this study come from a longitudinal, multi-method study of the natural history of smoking among adolescents. For this study, participants completed self-report questionnaires and in-depth interviews, in addition to week-long time/event sampling via palmtop computers, at baseline and a 15-month follow-up wave.

Participants

The sample for the longitudinal study included 1,263 9th and 10th grade students from 16 Chicago-area high-schools. All 9th and 10th graders at each school completed a brief screener survey (N = 12,970). Students were eligible to participate in the longitudinal study if they fell into one of four levels of smoking experience: 1) never smokers; 2) former experimenters (smoked in the last 12 months, but not in the last 90 days, and have smoked fewer than 100 cigarettes in their lifetime); 3) current experimenters (smoked in the past 90 days but smoked fewer than 100 cigarettes in their lifetime); and 4) regular smokers (smoked in the past 30 days and have smoked more than 100 cigarettes in their lifetime). Invitation/recruitment packets were mailed to eligible students and their parents, including a random sample of the never smokers and former experimenters, and all current and regular smokers (N = 3,695; valid N = 3,654, as 41 packets were returned due to an incorrect

address). Youth were enrolled into the longitudinal study after written parental consent and student assent was obtained. Of those invited, 1,344 agreed to participate (36.8%), and 1,263 (94.0%) completed the baseline measurement wave. Agreement to participate did not vary by smoking history, race/ethnicity, or parental smoking, but girls were slightly more likely to agree to participate than boys.

The current sample included the subset of youth who provided EMA data at baseline (N = 461). Students were invited to carry palm-top computers if they were former experimenters (n = 112), current experimenters (n = 249), or regular smokers (n = 100); thus, all participants in this study had previous or current smoking experience, although may have refrained from any cigarette use during the study. Adolescents were randomly assigned to this project within school, smoking level, grade, and gender. Participants ranged in age from 13.85 years to 17.29 years (M = 15.67 years, SD = 0.61), 50.7% were 9th graders, 55% were girls, and racial/ethnic composition was as follows: 56.8% White; 15.8% African American; 20% Latino; 2.8% Asian/Pacific Islander; and 4.6% Other/Bi-racial. Average parental education was as follows: 32.3% completed high school or less; 19.5% completed some college; and 36.2% completed college or more. Demographic characteristics of the participants enrolled in this study were representative of the 1,263 students in the total study; no differences were found between the adolescents who did and did not participate in the EMA substudy for grade ($\chi^2 = 3.66$, $p = .16$), gender ($\chi^2 = 0.54$, $p = .46$), race/ethnicity ($\chi^2 = 7.01$, $p = .32$), or age ($t = -1.63$, $p = .10$).

Procedures

All procedures received approval from the Institutional Review Board at the University of Illinois at Chicago. The current study included: 1) self-report questionnaires that assessed smoking behaviors and psychosocial variables, and 2) EMA via hand-held computers. The questionnaires were mailed to the students two weeks prior to each data collection wave, and students were instructed to bring the completed questionnaire to the interview session that occurred at their schools. Students were paid $20 upon receipt of the completed questionnaire.

In addition, EMAs were used to assess daily mood states. All participants were trained on the EMA device at the beginning of the data collection week, and they carried the device for seven consecutive days at each wave. Students were trained to complete three types of interviews on the EMA device: in response to random prompts (“random prompt” interviews) and to actively event record every situation when they decided to smoke (“smoke” interviews) and situations when they either had the opportunity but decided not to smoke or situations when they wanted to smoke but could not smoke because of external restrictions (e.g., in school, lack of availability; “no smoke” interviews). The device randomly prompted the adolescents approximately 5 times per day; in response to each signal, participants were trained to complete a brief interview about their activity, situation, and mood (random prompt). The smoke/no smoke interviews contained all of the questions from the random prompt interview, as well as questions about the participants’ mood both before and after the event. The current study utilized the EMA mood data from the random prompt interviews as well as pre-event mood data from the smoke and no smoke interviews. Participants were also trained to “suspend” (temporarily disable) the random prompt function during situations when they could not use the device. Participants received a payment of $40 at the end of the baseline data week, and $50 at the 15-month wave.

Measures

Demographic information was assessed via questionnaire and included age, grade, gender, race (Hispanic/Latino or not), ethnicity (White, African American, American Indian or Alaska Native, Asian, or Native Hawaiian/Other Pacific Islander), and parental education.

Smoking behavior was assessed via self-report questionnaire with several standardized items from national surveys, including: 1) the number of days smoked in the past 30 days, with responses ranging from 1 (none) to 9 (all 30 days), referred to as "monthly smoking frequency"; 2) the number of cigarettes per day on days smoked in the past 30 days, with responses ranging from 1 (none) to 11 (more than 20 per day), referred to as "monthly smoking quantity"; and 3) lifetime number of cigarettes, with responses ranging from 1 (I have never smoked) to 9 (500 or more). Additionally, continuous measures of monthly smoking frequency and quantity were constructed by computing the mid-point of each response category. The reliability of these retrospective self-reports of smoking behavior is supported by the strong correspondence with both daily diary reports of smoking episodes as well as interview-obtained reports of smoking behavior in our past work (Diviak, Kohler, Mermelstein, & Flay, 2001).

Depression was assessed via the Center for Epidemiological Studies Depression inventory (CES-D; Radloff, 1977). The CES-D is a 20-item measure that assesses the frequency of depressive symptoms on a 0 to 3 point scale. Research supports the validity and utility of the CES-D to measure depressive symptoms in high school adolescents (Radloff, 1991). Coefficient alpha in the current sample = .89.

Mood-Regulatory Smoking Motives, namely the perceived mood-regulatory functions of smoking, were measured via the 4-item Affect Regulation Motives subscale of a shortened version of the Wills Tobacco Motives Inventory (Wills, Sandy, & Shinar, 1999). This scale asks respondents to rate each potential affect-related function for smoking ("Here are some things that people have said about smoking cigarettes") with respect to how true they think it is, with responses ranging from 1 (not at all true) to 5 (very true). Items include: Smoking helps you when you're feeling angry; Smoking helps you calm down when you're feeling tense and nervous; Smoking helps you feel more relaxed; and Smoking cheers you up when you are in a bad mood. Coefficient alpha in the current sample at baseline =.91.

Nicotine Dependence was measured using the seven-item adolescent version of the Fagerstrom Tolerance Questionnaire (mFTQ; Prokhorov, Pallonen, Fava, Ding, & Niaura, 1996). Coefficient alpha for the total mFTQ score = .66. An mFTQ score of 6 or more is considered to represent a high level of nicotine dependence (Prokhorov et al., 1996).

Daily Negative Affect (EMA)—Participants were asked on each EMA interview to rate their mood (e.g., "Before the signal, I felt sad") and responded to mood adjectives using a 10-point Likert-type scale. Mood adjectives were selected based on prior studies, including qualitative (focus groups and in-depth interviews) and quantitative data collection with adolescents. Consistent with factor analyses on the current data set, the following adjectives formed a strong "Negative Affect" (NA) scale: angry, frustrated, irritable, sad, and stressed, all with loadings greater than .79 (Coefficient alpha =.89). EMA have been used effectively in past research to study mood constructs (e.g., Hedeker, Mermelstein, & Demirtas, 2008; Hedeker et al., 2009; Silk et al., 2003).

Mood Variability (EMA)—An index of mood variability was constructed from the EMA mood ratings by computing mean standard deviation scores for the negative affect scale for each participant across the baseline data collection week. Mood variability thus reflected the degree of intraindividual fluctuation within negative affect across the EMA observations

during the week. We conceptualized a wider range of fluctuation to reflect dysfunctional affect regulation abilities. Standard deviations have been used to quantify mood variability in EMA studies with supported reliability and validity (Eid & Diener, 1999), and have shown relations to internalizing and externalizing symptomatology (Silk et al., 2003).

Results

Analytic Approach

To examine smoking patterns over time, groups were created based on the degree and direction of change in smoking behavior (measured via monthly smoking frequency) across the baseline to the follow-up 15 month waves, with attention to *a priori* potential points of escalation (e.g. monthly, weekly, and daily smoking). We identified seven groups of longitudinal smoking patterns, illustrated in Figure 1, including: *nonsmokers*, who did not smoke at baseline and remained abstinent at 15 months, despite history of smoking experience (n = 129, 32%); *triers*, who engaged in low levels of experimentation during the study, as defined by zero to three days smoked in the past month at baseline and 15 months (n = 109, 27%); *experimenters*, who escalated from low levels of use at baseline (i.e., reporting zero to five days of smoking in the past month) to weekly smoking at 15 months (i.e., five to 10 days smoking in the past 30 days; n = 32, 8%); *rapid escalators*, who escalated from low use at baseline (i.e., monthly smoking of zero to five days) to near-daily or daily use at 15 months (i.e., smoking on 11 to 30 or more days in the past month; n = 34, 8%); *infrequent stables*, who maintained a stable level of approximately weekly smoking at baseline and 15 months (n = 37, 9%); *smokers*, who engaged in near daily to daily smoking at baseline and 15 months (n = 43, 11%); and *quitters*, who reported smoking at baseline but reported zero days of smoking at 15 months (n = 21, 5%). Girls and boys were similarly represented across: girls comprised 54 – 58% of each smoking group, with the exception of the quitters (67% girls).

Table 1 displays mean smoking frequency, quantity, and mFTQ scores across smoking groups at baseline and 15 months. A series of one-way between-subjects analyses of variance (ANOVAs) examined group differences in smoking variables at each wave. Analyses confirmed significant differences among the groups for smoking frequency at baseline, F (6, 384) = 599.74, $p < .0001$, $\eta^2_{partial} = .90$, and at 15 months F (6, 374) = 485.04, $p < .0001$, $\eta^2_{partial} = .89$; and baseline smoking quantity, F (6, 384) = 65.39, $p < .0001$, $\eta^2_{partial} = .51$, and 15-months smoking quantity, F (6, 374) = 76.82, $p < .0001$, $\eta^2_{partial} = .55$. Levels of nicotine dependence also significantly differed among groups at baseline, F (6, 384) = 49.78, $p < .0001$, $\eta^2_{partial} = .44$, and at 15 months, F (6, 374) = 67.36, $p < .0001$, $\eta^2_{partial} = .51$. As Table 1 reveals, mFTQ scores at baseline suggest low levels of dependence among all groups except the Smokers; however, by follow-up, Rapid Escalators and Smokers show moderate levels of dependence. Post-hoc Tukey tests examined pairwise comparisons among smoking groups, and findings are displayed in Table 1. As Table 1 reveals, results at each wave were consistent with smoking group status.

In addition, paired t-tests examined changes in monthly smoking frequency, quantity, and nicotine dependence between baseline and follow-up among the primary groups of interest. Consistent with their group status, rapid escalators significantly increased on all smoking measures over time. For the triers, differences in smoking quantity and dependence between waves were not significant, although the slight increase in smoking frequency was significant. Last, the smokers slightly but significantly escalated on all smoking measures over time.

Compliance and Attrition

Participants provided mood reports for a mean of 30.09 (*SD* = 7.76) random prompts on the EMA device per person at baseline. In total, participants responded to 73% of all random prompts. These compliance rates are consistent with prior literature (O'Connell, Gerkovich, Bott, Cook, & Shiffman, 2002). Participants also provided a mean of 2.45 (*SD* = 4.85) smoke interviews and 2.73 (*SD* = 3.43) no smoke interviews at baseline. Attrition in the current study was minimal. At the 15 month wave, 411 adolescents participated in data collection (89.2%). Analyses verified that there were no significant differences in retention for grade, gender, and race/ethnicity, nor for baseline reports of negative mood, negative mood variability, depression, and monthly smoking quantity (effect sizes $\eta^2_{partial}$ ranging from .00 to .01). However, participants who did not complete the 15 month wave reported significantly greater smoking frequency at baseline (i.e., number of days smoked in the past month; *M* = 8.53, *SD* = 10.63) than did those with complete data (*M* = 5.14, *SD* = 8.89), *F* (1, 458) = 6.14, *p* = .01, $\eta^2_{partial}$ = .01.

Descriptive Analyses

A series of independent *t*-tests examined gender differences in the main smoking and mood variables at baseline. No gender differences were found for lifetime cigarettes, smoking frequency or quantity, mFTQ scores, or smoking motives; gender differences remained nonsignificant at follow-up. Findings revealed that girls reported significantly higher levels of depressive symptoms (*M* = 19.84, *SD* = 10.86) than boys (*M* = 14.32, *SD* = 8.35), *t*(458) = 6.00, *p* < .0001. Girls also reported higher levels of negative affect (*M* = 3.79, *SD* = 1.59) and negative mood variability (*M* = 1.73, *SD* = 0.58) than boys (negative affect *M* = 3.21, *SD* = 1.35; negative mood variability *M* = 1.50, *SD* = 0.64), *t*(459) = 4.33 and *t*(459) = 4.47, respectively, *p*s <.01. Analyses also examined bivariate correlations among the mood variables at baseline. In line with expectations, negative mood variability correlated significantly and positively with depression, *r* = .30, *p* < .001, and daily negative affect, *r* = .47, *p* < .001, suggesting that these constructs are related yet distinct dimensions of affect. Additionally, smoking motives were significantly associated with higher levels of depression, negative mood, and negative mood variability (*r*s = .23, .26, and .20, respectively, *p*s < .001).

Prediction of Longitudinal Smoking Patterns: Mood Variability and Negative Mood

We hypothesized that baseline mood variability would prospectively predict escalation in cigarette use, and that affect regulation smoking motives would moderate these relationships. To test these hypotheses, a series of hierarchical binomial logistic regressions were estimated via SPSS REGRESSION. This method allowed for the estimation of odds ratios (OR) associated with specific meaningful contrasts of the longitudinal smoking patterns, including those who experimented with cigarettes at baseline and progressed to heavier levels of use (i.e., rapid escalators) versus those who experimented at similar baseline levels but never escalated to regular use (i.e., triers and experimenters). We were also interested in the associations between mood and smoking outcomes at the lower and upper ends of the smoking continuum. Thus, four planned contrast models were evaluated (of note, the order of the contrast also reflects the coding of 0, 1): (1) triers/experimenters (including the triers and the experimenters) versus rapid escalators; (2) smokers versus rapid escalators; (3) nonsmokers versus smokers; and (4) nonsmokers versus triers/experimenters. Each contrast model included baseline mood variability in the first step, baseline smoking motives in the second step, and Mood Variability x Smoking Motives in the final step. Given the significant gender differences in baseline mood variability and average mood, each contrast model was evaluated for the total sample and also separately by gender. In addition, all models were repeated using average mood level and Mood x Smoking Motives

as predictors of future smoking status. Separate analyses allowed for comparisons between the mood variability-smoking and overall mood-smoking relationships. All interactions were centered to reduce potential multicollinearity (Aiken & West, 1991).

Results of the contrast models for the combined sample and for the analyses stratified by gender are summarized in Table 2. Given the significant gender differences, only analyses stratified by gender are discussed below. In addition, Figure 2 illustrates the differences in baseline negative mood variability mean levels of negative mood among the longitudinal smoking groups under examination and stratified by gender. For ease of presentation, main effects for smoking motives listed and described below refer to the findings from the mood variability models, and thus these values are adjusted for negative mood variability; in the average negative mood models, similar effects and significance levels were obtained for smoking motives and therefore the effects are not described here.

Contrast 1: Triers/Experimenters v. Rapid Escalators (N = 175; Girls, n = 102; Boys, n = 73)—Consistent with hypotheses, girls with higher levels of baseline negative mood variability were more than twice as likely to rapidly escalate to regular smoking than to remain at a low level of cigarette use. As Figure 2 illustrates, girls who escalated in their smoking pattern over time have high levels of mood variability at baseline. In addition, when added to the model, smoking motives significantly predicted rapid escalation, indicating that girls with greater affect-related motives for smoking were more likely to progress in their smoking. Smoking motives did not moderate the mood variability-smoking relationship. Additionally, neither mean negative mood nor Negative Mood x Motives predicted escalation for girls. For boys, neither negative mood variability nor mean negative mood was associated with risk of rapid escalation. However, findings revealed a significant Negative Mood x Motives effect. To identify the source of this interaction, follow-up logistic regression analyses were conducted by level of motives, using a median split. Findings indicated nonsignificant effects for negative mood among those with higher mood motives for smoking (OR = 1.77, *ns*) and lower motives (OR = 0.60, *ns*). Although power is limited in follow-up analyses to demonstrate effects, the significant interaction is suggestive of differing trends for mood-smoking patterns at varying levels of smoking motives.

Contrast 2: Smokers v. Rapid Escalators (*N* = 77; Girls, *n* = 45, Boys, *n* = 32)—Girls with higher baseline negative mood variability were significantly more likely to be rapid escalators versus smokers. Thus, girls engaging in higher use at baseline had low concurrent levels of mood variability (Figure 2). Conversely, girls with higher motives were more likely to be smokers than rapid escalators. In contrast, mean negative mood was not a significant a predictor of smoking pattern, and Mean Negative Mood x Smoking was not significant. For boys, those with higher motives were more likely to be smokers than rapid escalators.

Contrast 3: Nonsmokers v. Smokers (*N* = 170; Girls *n* = 95, Boys *n* = 75)—For girls, smoking motives emerged as the only significant predictor; baseline negative mood variability was not associated with smoking group. Thus, the girl smokers at baseline had levels of negative mood variability as low as the nonsmokers (see Figure 2). Similarly, boys with higher smoking motives were more likely to be smokers than nonsmokers. However, neither mood variability nor mean levels of mood significantly differentiated the boy nonsmokers and smokers.

Contrast 4: Nonsmokers v. Triers/Experimenters (*N* = 268; Girls *n* = 152, Boys *n* = 116)—Analyses stratified by gender revealed no significant predictors of future smoking. Thus, for boys and girls, baseline negative mood variability did not differentiate

the nonsmokers from the triers/experimenters; additionally, smoking motives were unrelated to future smoking.

Prediction of Longitudinal Smoking Patterns: Depressive Symptoms

A series of binary logistic regression models identical to the contrast models specified above were estimated for baseline depressive symptoms. Separate models were conducted for the total sample, girls, and boys; only gender-specific results are described. For girls, no contrasts were significant. For boys, a trend was found for smokers versus rapid escalators (Wald test = 3.77, OR = .92, 95% C.I. 0.84 – 1.00, p = .05), suggesting that boys with higher depressive symptoms at baseline were more likely to be stable smokers than escalators. Additionally, boys with higher depressive symptoms were significantly more likely to be smokers than nonsmokers (Wald test = 7.54, OR = 1.09, 95% C.I. 1.03 – 1.17, p = .006).

Discussion

The current study examined how negative mood variability, overall negative mood, and depression may differentially relate to developmental patterns of youth smoking. Findings supported a complex self-medication model of smoking escalation in adolescence whereby mood-smoking relationships differed by affect dimension and gender. Results thus suggest that inconsistent mood-smoking relations in past research may be driven by the complex interrelationships among affect vulnerabilities, gender, and smoking patterns.

Relations between Adolescent Mood Variability, Depression, and Overall Mood

Consistent with hypotheses, higher levels of negative mood variability were associated with elevated depressive symptoms and overall negative moods. Thus, findings support previous research suggesting that negative mood volatility is inversely related to adolescent emotional functioning (e.g., Kovacs et al., 2006; Silk et al., 2003), but also represents a distinct emotional construct. Additionally, in line with past research (e.g., Silk et al., 2003), girls experienced higher levels of depression and overall negative mood than boys. Girls also experienced higher levels of negative mood variability than boys, indicating that boys may experience more stable moods. Gender differences in various biological and cognitive factors may contribute to greater mood fluctuations among girls as compared to boys, including higher levels of rumination (Broderick & Korteland, 2002), the ability to make finer discriminations in self-reports of emotional experience, and/or hormonal changes related to menstruation and puberty (Buchanan, 1991), although causal mechanisms were not examined in this study.

Findings of lower mood variability in boys compared to girls follows those of Hedeker et al. (2008, 2009), with the same set of participants, but focusing on a different analytic approach and subset of the EMA data. As Hedeker et al. (2008) note, there is substantial heterogeneity in mood variability among these adolescents. The present study goes beyond the work of Hedeker and colleagues, however, in its consideration of other potential moderators of mood variance as well as understanding how mood variance relates to longitudinal patterns of smoking.

Prospective Prediction of Smoking Escalation: Mood Variability and Overall Mood

A primary goal was to investigate whether mood variability, as well as overall mood, influenced adolescents' smoking behavior. Consistent with hypotheses, results suggest that negative mood variability confers risk for girls' smoking escalation. Among girls with low and infrequent smoking at baseline, higher levels of negative mood variability was associated with greater risk of rapidly escalating to daily smoking versus remaining at low levels of use by follow-up. Moreover, girl nonsmokers and girl triers/experimenters had

similar levels of baseline mood variability, further supporting the specificity of mood variability as a risk factor for rapid progression in smoking. Notably, such relationships were unique to mood variability; overall negative mood at baseline did not differentiate girls' future smoking patterns. Thus, findings extend past cross-sectional and longitudinal research demonstrating associations between affect dysregulation and adolescent substance use (Novak & Clayton, 2001; Simons & Carey, 2002; Simons et al., 2004; Weinstein et al., 2008; Wills & Stoolmiller, 2002; Wills et al., 2006) by documenting mood variability as a specific risk factor for the progression to frequent smoking among girls. In line with the self-medication model (Khantzian, 1997, 1999), the present results as well as past research suggest that emotionally labile girls may escalate in their cigarette use as a means of coping with or controlling mood fluctuations. In contrast, the regular smokers at baseline had low levels of mood variability; future work is needed to examine how mood may change as smoking escalates and is maintained at higher levels of use.

However, negative mood variability did not predict boys' smoking escalation. Boys reported low levels of mood variability overall, suggesting that mood lability is not a source of vulnerability for adolescent boys. Rather, findings point to the relevance of overall levels of negative mood as a risk factor for boys' smoking. Specifically, results suggest that higher levels of negative moods predict escalation among boys, but patterns differed by strength of mood-related motives. Although follow-up tests of the significant motives by mood interaction were limited in power to demonstrate these effects, the observed trends are consistent with past work indicating that high negative mood among youth with strong mood-related motives for substance use predicted higher levels of use (Wills et al., 1999b). It will be important to further examine the mood motives-negative mood risk pathways for boys smoking progression in future research.

Overall, results indicated that affect regulation smoking motives also directly corresponded to more problematic patterns of use over time for boys and girls. These findings add to the literature on affect-related smoking motives as a robust risk factor for youth smoking among boys and girls (e.g., Mayhew et al., 2000; Wills et al., 1999a; Wills et al., 2006). Yet smoking motives were found to moderate the mood-smoking link for boys only. We are unaware of other studies that have found interactions of gender, negative mood, and motives on substance use in adolescence. However, results are consistent with the affective functions tapped by the motives measure (Wills et al., 1999a), namely the motivation to improve mean levels of negative affect via smoking versus the desire to stabilize mood volatility. Thus, moderation effects of smoking motives corresponded to the salient affective risk factor for boys' versus girls' smoking, rather than suggesting gender differences in the motives-smoking relationship.

Taken together, results support a complex self-medication model of smoking escalation, whereby adolescents with diverse affective vulnerabilities – manifesting as labile moods for girls and high negative mood for boys – may use cigarettes to stabilize mood volatility or to relieve negative moods, and are at risk for progression to problematic use. Gender-specific relations between various stressors and smoking uptake have been found in previous work (e.g., Byrne & Mazanov, 1999), and underscore the importance of considering gender differences in the etiology of adolescent smoking. Moreover, the examination of multiple affective indices in the current study provides insight into the multifaceted emotional mechanisms underlying smoking escalation, and may shed light on inconsistent mood-smoking relations in the past. Of note, analyses examining girls' negative mood variability revealed large confidence intervals, indicating that mood variability may particularly promote escalation for some individuals versus others. We examined one hypothesized cognitive moderator of these relationships, smoking motives, but findings were not significant. However, the interplay of mood variability and relevant environmental or

genetic factors (e.g., family discord, temperamental characteristics; Wills & Stoolmiller, 2002) in the development of girls' smoking may be of particular importance, and hence the identification of moderators warrants further study.

Depression and Smoking Patterns

In contrast to predictions, depressive symptoms were unrelated to girls' smoking patterns, and only differentiated boys at the extreme ends of the smoking continuum (i.e., stable nonsmokers and smokers). Results are not surprising in light of the mixed literature on depression-smoking relations in adolescence (e.g., Duncan & Rees, 2005; Repetto et al., 2006; Wu & Anthony, 1999). Indeed, discrepant associations between depressive symptomatology and smoking behavior in past work may be attributable, at least in part, to differences in the level of smoking experience across study samples. Significant effects may be found in samples composed mainly of very experienced and non-experienced smokers (e.g., Windle & Windle, 2001) or when making distinctions between gross levels of smoking frequency (e.g., Lloyd-Richardson et al., 2002; Patton et al., 1996) but depressive symptomatology may not distinguish among more precise patterns of smoking beyond experimentation. Alternately, depression-smoking relations may vary by the level of depressive symptoms, with associations emerging primarily among adolescents with persistent, high levels of depressive symptoms (Rodriguez, Moss, & Audrain-McGovern, 2005; Windle & Windle, 2001). Thus, the use of a continuous versus categorical measure of depression may account for the observed findings.

In addition, the lack of depression-smoking effects for girls is consistent with the findings for overall mood level. Collectively, findings suggest that overall mood – as assessed by mean levels of mood or the CES-D (Radloff, 1977), which taps global, persistent low mood – is less relevant for girls' cigarette use. Rather, changes in momentary affective states (i.e., mood volatility), which are not captured in overall measures of mood, may be the critical emotional determinant of girls' progression beyond experimentation. Hence, findings underscore the importance of assessing within-person fluctuations in girls' mood in future studies, as reliance on global mood measures may mask key etiologic processes.

Implications, Limitations, and Future Directions

The current findings highlight important avenues for the design of prevention and intervention efforts aimed at youth smoking. One implication of the prospective data is that dysregulated youth may be a key target for prevention and early intervention efforts. Moreover, intervention programs can include gender-specific components to maximize effectiveness among high risk youth. For girls in the experimental stages of smoking, intervention protocols that focus on enhancing emotion regulation through the use of mindfulness strategies (e.g., Acceptance and Commitment Therapy for Adolescents, ACT-A; Greco, Blackledge, Coyne, & Ehrenreich, 2010; Mindfulness-Based Stress Reduction; Kabat-Zinn, 1994) may be particularly relevant. Additionally, efforts for boy experimenters as well as regular smokers may focus on identifying non-drug methods for alleviating negative affect (Wills et al., 1999b).

Study limitations should be noted. This study attempted to capture only one component of the complex construct of affect regulation via intraindividual mood variability. Although research supports the connection between affect dysregulation and mood variability (Hoeksma et al., 2004; Wills et al., 2006), future work examining broader measures of affect dysregulation and smoking is warranted. Second, this study did not directly examine causal relationships between mood and smoking, and thus interpretations regarding the influence of mood on smoking behavior must be made cautiously. Third, smoking history was assessed via self-report and was not confirmed by biological assay, and thus results may potentially

be biased by over- or under-reporting of smoking behavior. In addition, the use of observed trajectory classifications to create longitudinal smoke groups, rather than an empirical – based distinction, may limit the generalizability of our findings. Further, although agreement to participate in the study did not vary by smoking history, race/ethnicity, or parental smoking, the generalizability of findings is limited by the 37% participation rate for the larger study. Findings may also underestimate smoking-mood relations among the smokers, as those with the higher smoking frequency at baseline – and possibly higher levels of emotional distress – were more likely not to participate in the follow-up wave of data collection. Nonetheless, results are bolstered by the fact that baseline smoking quantity did not differ between study completers and noncompleters. Finally, given the small sample sizes in the analyses stratified by gender, we chose to limit the number of predictor variables under investigation and thus did not control for potential confounds related to smoking and mood (e.g., genetics, family factors, or alcohol/drug use) or other possible moderators (e.g., ethnicity). However, findings are strengthened by the similar patterns found in both the present results and previous studies that did control for potential confounding variables (e.g., Orlando et al., 2001; Wills & Stoolmiller, 2002; Windle & Windle, 2001).

Despite these limitations, the present study revealed valuable information about the emotional factors influencing smoking escalation among adolescents. Strengths of the present study include the use of real-time data, a specific focus on escalation from low levels of use, and considerations of both mood variability and mean levels of mood. Findings add to the growing body of evidence on the importance of affect dysregulation for understanding smoking among adolescents (Novak & Clayton, 2001; Weinstein et al., 2008; Wills et al., 2006), but also highlight gender differences in these processes that must be considered in future work.

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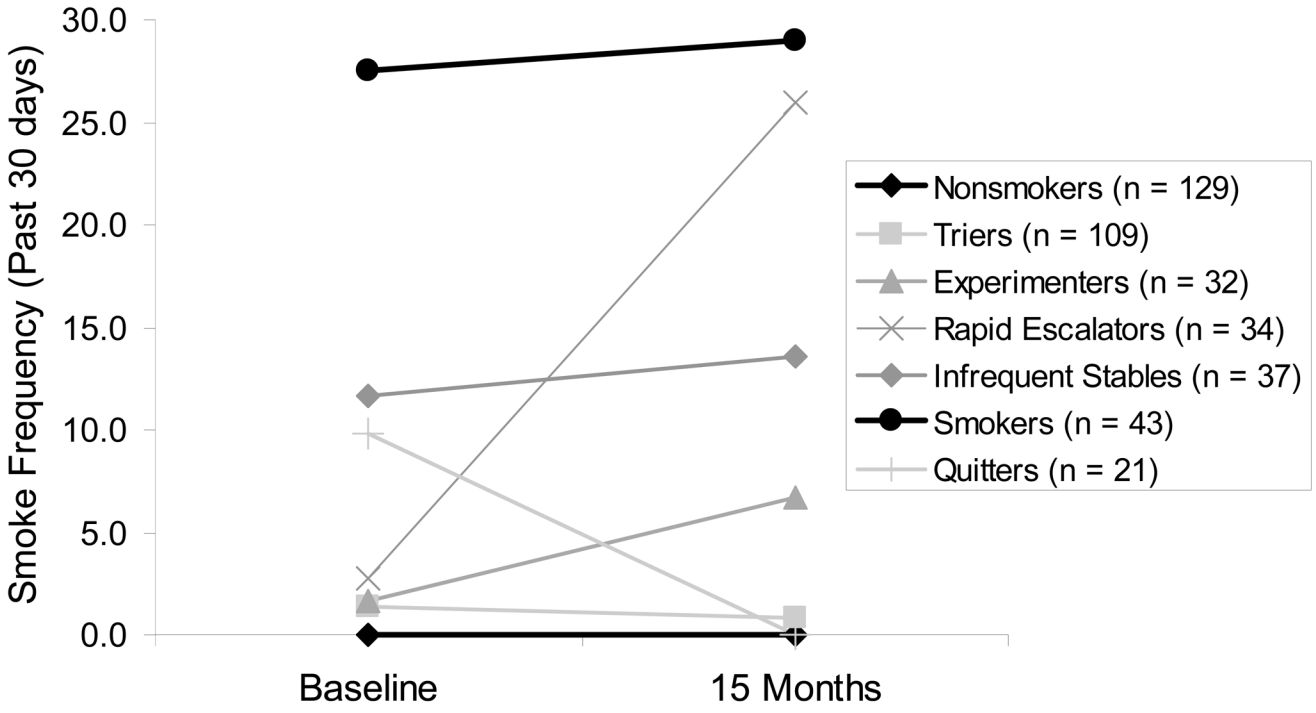

Figure 1.
Monthly smoking frequency (number of days smoked in the past 30 days), from baseline to follow-up, by longitudinal smoking group.

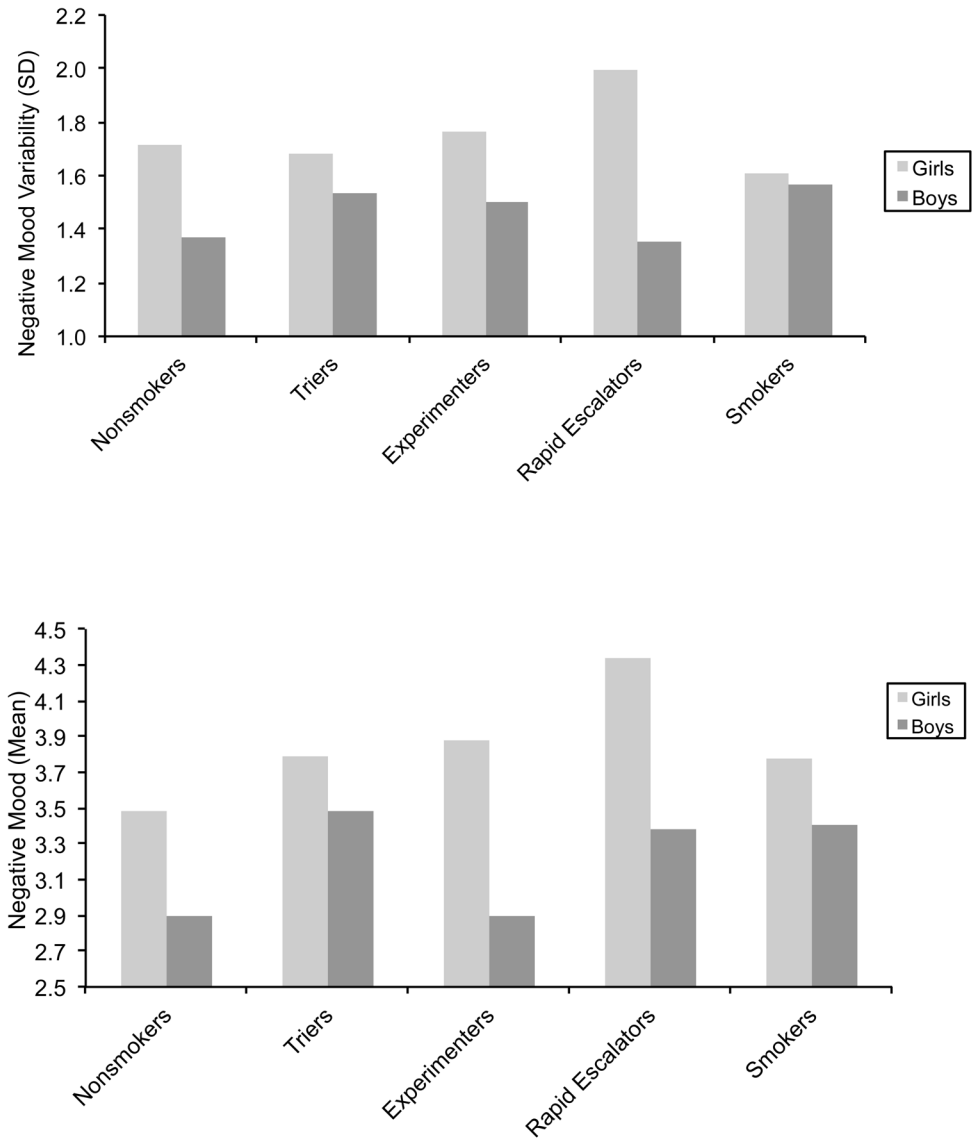

Figure 2.
Estimated negative mood variability at baseline (upper portion) and mean overall negative mood at baseline (lower portion) as a function of longitudinal smoking group and gender. Note: differences between genders within smoke group were not directly compared.

Table 1

Descriptive Statistics and Results of Pairwise Comparisons for Smoking Measures, at Baseline and Follow-up, by Longitudinal Smoking Pattern

	BASELINE							15 Months						
		Smoke Frequency		Smoke Quantity		mFTQ			Smoke Frequency		Smoke Quantity		mFTQ	
	N	*M*	*SD*	*M*	*SD*	*M*	*SD*	*N*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Nonsmokers	129	0.00[a]	0.00	0.00[a]	0.00	0.79[a]	0.85	110	0.00[a]	0.00	0.00[a]	0.00	0.77[a]	0.85
Triers	109	1.40[b]	0.95	0.81[b]	0.83	1.30[b]	0.85	105	0.85[a]	1.03	0.60[a,b]	0.99	1.26[b]	0.70
Experimenters	32	1.69[a,b]	2.12	0.48[a,b]	0.60	1.16[a,b]	0.88	31	6.69	2.02	1.77[b,c,d]	1.48	1.55[b]	0.72
Rapid Escalators	34	2.79[b]	2.39	1.35[b,c]	1.87	1.68[b,c]	0.88	34	25.99	5.77	7.06[e]	5.85	3.27	1.84
Infrequent Stables	37	11.62[c]	5.32	2.18[c,d]	1.28	2.08[c,d]	0.83	37	13.58	10.86	2.95[c]	3.08	2.43	1.29
Smokers	43	27.56	2.53	4.99	3.44	3.51	1.22	43	28.95	2.06	8.95[e]	5.68	4.05	1.43
Quitters	21	9.70[c]	8.27	1.79[b,d]	2.18	1.96[b,d]	1.32	21	0.00[a]	0.00	0.00[a,d]	0.00	1.33[a,b]	1.02

Note. Smoke frequency = # days smoked in the past month; Quantity = # cigarettes smoking on days smoked in the past month; mFTQ= Modified Fagerstrom Tolerance Questionnaire.

Within each smoking measure at each time point, mean values with shared alphabetic superscripts signify nonsignificant pairwise comparisons; all other pairwise comparisons were significant at $p < .05$.

Table 2

Logistic Regression Models of Smoking Outcome Contrasts, for the Total Sample and by Gender: Negative Mood Measures

	TOTAL SAMPLE			GIRLS			BOYS		
Predictor	**Wald Test**	**OR**	**95.0% C.I.**	**Wald Test**	**OR**	**95.0% C.I.**	**Wald Test**	**OR**	**95.0% C.I.**
CONTRAST 1: TRIERS/EXPERIMENTERS v. RAPID ESCALATORS									
NA Variability	0.77	1.33	0.70 – 2.53	4.20	2.61	1.04 – 6.55 *	1.04	0.59	0.22 – 1.62
Motives	3.24	1.08	0.99 – 1.17	5.87	1.15	1.03 – 1.28 *	0.00	1.00	0.88 – 1.15
Variability x Motives	0.02	1.01	0.88 – 1.17	0.42	0.92	0.73 – 1.17	0.11	1.03	0.84 – 1.27
Mean NA	1.20	1.14	0.90 – 1.45	1.73	1.23	0.90 – 1.67	0.01	1.02	0.68 – 1.52
Mood x Motives	0.26	1.02	0.96 – 1.08	2.61	0.94	0.87 – 1.01	5.04	1.14	1.02 – 1.28 *
CONTRAST 2: SMOKERS v. RAPID ESCALATORS									
NA Variability	0.95	1.46	0.69 – 3.09	4.92	4.54	1.19 – 17.30 *	17.30	0.59	0.20 – 1.80
Motives	13.56	0.79	0.70 – 0.90 **	5.15	0.81	0.68 – 0.97 *	0.97	0.79	0.66 – 0.95 *
Variability x Motives	0.00	1.00	0.82 – 1.21	0.00	1.00	0.65 – 1.54	1.54	0.96	0.72 – 1.27
Mean NA	0.77	1.14	0.85 – 1.52	1.36	1.26	0.85 – 1.86	0.00	0.99	0.62 – 1.57
Mood x Motives	0.13	0.99	0.91 – 1.07	3.83	0.86	0.74 – 1.00	1.93	1.11	0.96 – 1.27
CONTRAST 3: NONSMOKERS v. SMOKERS									
NA Variability	0.04	1.06	0.62 – 1.81	0.49	0.77	0.37 – 1.60	0.99	1.51	0.67 – 3.42
Motives	36.78	1.37	1.24 – 1.51 **	21.88	1.38	1.21 – 1.58 **	14.24	1.35	1.15 – 1.57 **
Variability x Motives	0.17	0.97	0.82 – 1.14	0.32	0.93	0.74 – 1.18	0.00	0.99	0.78 – 1.26
Mean NA	2.39	1.20	0.95 – 1.51	0.68	1.13	0.84 – 1.51	2.01	1.34	0.90 – 1.99
Mood x Motives	0.01	1.00	0.93 1.06	0.00	1.00	0.92 – 1.09	0.00	1.00	0.90 – 1.11
CONTRAST 4: NONSMOKERS v. TRIERS/EXPERIMENTERS									
NA Variability	0.67	1.18	0.80 – 1.73	0.01	0.97	0.58 – 1.65	1.47	1.46	0.79 – 2.67
Motives	0.90	1.03	0.97 – 1.08	0.02	1.01	0.93 – 1.08	1.41	1.05	0.97 – 1.15
Variability x Motives	0.80	0.96	0.87 – 1.05	0.00	1.00	0.88 – 1.14	1.73	0.91	0.79 – 1.05
Mean NA	4.56	1.19	1.01 – 1.41 *	1.57	1.14	0.93 – 1.40	3.22	1.29	0.98 – 1.72
Mood x Motives	0.00	1.00	0.96 – 1.04	0.18	0.99	0.94 – 1.04	1.19	1.04	0.97 – 1.11

Note. The listed effects reflect findings from the initial step each variable was entered in each contrast model (i.e., controlling for only prior variables in model).

NA = negative affect; OR = Odds Ration; C.I. = Confidence Interval.

* $p < .05$;

** $p < .01$.