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Mood Variability and Cigarette Smoking Escalation among Adolescents

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Abstract

The current study examined how affect dysregulation, as indexed via within-person negative mood variability, related to longitudinal patterns of smoking among adolescents. Eighth and 10th grade students (N = 517; 56% girls) provided data on cigarette use at baseline, six-, and twelve-month waves, and also provided ecological momentary assessments of negative moods via palmtop computers for one week at each wave. Mood variability was examined via the intraindividual standard deviations of negative mood reports at each wave. As predicted, high levels of negative mood variability at baseline significantly differentiated adolescents who escalated in their smoking behavior over time from those who never progressed beyond low levels of experimentation during the course of the study. Mixed-effects regression models revealed that adolescents who escalated in their smoking experienced a reduction in mood variability as smoking increased, whereas mood variability levels were more stable among those with consistently high or low levels of cigarette use. Results suggest that high negative mood variability is a risk factor for future smoking escalation, and mood stabilizing effects may reinforce and maintain daily cigarette use among youth.

Keywords

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

Although a considerable proportion of youth experiment with smoking, with 26% of 8th graders and 50% of 12th graders reporting having ever smoked cigarettes (Johnston, O'Malley, Bachman, & Schulenberg, 2006), fewer adolescents progress to heavier levels of smoking; 14% of 12th graders report smoking on a daily basis (Johnston et al., 2006). Understanding the factors that differentiate adolescents who progress beyond experimentation with smoking versus those who engage in experimentation but never escalate to heavier levels of cigarette use can greatly inform prevention and intervention efforts. To this end, the current study utilized ecological momentary assessment methods (EMA; Stone & Shiffman, 1994) to examine adolescent mood variability as a predictor of longitudinal smoking patterns.

Researchers have increasingly focused on the role of self-regulation of emotions as a risk factor for smoking initiation and escalation among youth. Emotional self-regulation refers to the involuntary as well as effortful processes involved in initiating and maintaining affective

responses, as well as attempts to modify the intensity and quality of such responses (Forbes & Dahl, 2005; Thompson, 1994). Adolescence is a period of refinement of affect regulation systems (Kovacs et al., 2006; Spear, 2000) and maladaptive affect regulation during this period may confer risk for substance use. As posited by the self-medication model, individuals with self-regulation vulnerabilities, including dysfunctional affect regulation processes and emotional lability, engage in substance use as a means of regulating and alleviating emotional distress (Khantzian, 1997). Further, the mood-regulatory effects of smoking and nicotine (Brody, 2006; Delfino, Jamner, & Whalen, 2001; Mermelstein, Hedeker, Flay, & Shiffman, 2007) may reinforce the use of cigarettes to cope with affective distress.

Consistent with the self-medication model, the current study investigated the role 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. Mood variability reflects the intensity and frequency of intraindividual fluctuations in momentary affective states (Eid & Diener, 1999). 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 varying intensities of negative emotions, with greater peak intensities, yielding a variable affective profile over a period of time; in contrast, an emotionally regulated individual would exhibit a more stable affective profile. We thus conceptualize high levels of negative mood variability as a byproduct of affect dysregulation.

Evidence suggests an association between affect dysregulation and substance use in adolescence, although conceptualizations of affect regulation vary across studies. Wills and colleagues (Wills, Walker, Mendoza, & Ainette, 2006) found that poor emotional control (as indexed by global self-report questionnaire measures of affective lability, rumination, and emotional reactivity) was positively related to the frequency of marijuana, alcohol and cigarette use in adolescence, although effects were mediated by proximal influences such as exposure to negative events and motives for substance use. In addition, deficient emotional regulation (indexed via global self-report questionnaires assessing the ability to control extreme affect and to regulate negative moods) predicted adolescent experimentation with cigarettes as well as progression to regular use (Novak & Clayton, 2001). Moreover, a series of studies by Simons and colleagues demonstrated significant relationships between affect lability (assessed by subjective global questionnaire on the frequency and extremity of mood shifts) and alcohol- and marijuana-use problems among college students (Simons & Carey, 2002; Simons, Carey, & Gaher, 2004).

Taken together, theory and research suggest that labile youth who lack internal regulation resources may use substances as a means of mood regulation. However, additional research is needed to better understand the role of affect dysregulation in predicting smoking behavior. First, previous studies examining affect dysregulation as a predictor of smoking have been cross-sectional (e.g., Novak & Clayton, 2001; Wills et al., 2006). Longitudinal research is needed to examine prospective relations between mood variability and smoking patterns, as well as to investigate dynamic, reciprocal relationships between mood variability and smoking over time. In line with the self-medication model of substance use, we expect that if mood variability improves or stabilizes with increased smoking, such mood improvement may reinforce cigarette use and influence continued use beyond experimentation.

An additional gap in the literature concerns the assessment of mood variability. Each of the previous studies examining affect dysregulation-smoking relations utilized global retrospective self-report questionnaires. Such reports required the adolescent to rate their global experience of emotional lability (e.g., “one minute I can be OK and the next minute I feel tense

and nervous"; Wills et al., 2006) or their overall ability to regulate negative emotions (e.g., Novak & Clayton, 2001). Retrospective reports of mood and coping abilities may be subject to recall difficulties and judgment biases (Stone et al., 1998; Stone & Shiffman, 1994), and may not reflect actual fluctuations in moods. Real-time data capture procedures, such as Ecological Momentary Assessments (EMA; Stone & Shiffman, 1994), sample mood in the moment and therefore minimize retrospective bias or summary judgments of affective experience. Moreover, the random and frequent assessment of mood via EMA allows for the examination of intraindividual fluctuations across individual mood reports, yielding a finer grained and more objective index of actual variability in moods. Ecological momentary assessments have been used effectively in prior studies to study adolescent (e.g., Larson, Csikszentmihalyi, & Graef, 1980; Larson, Rafaelli, Richards, Ham, & Jewell, 1990; Silk, Steinberg, & Morris, 2003), as well as adult (Penner, Shiffman, Paty, & Fritzche, 1994; Ilies & Judge, 2002) mood variability.

The goal of the current study was to explore how mood variability relates to longitudinal smoking patterns in adolescence. Mood variability is defined as the intraindividual standard deviations of mood assessments across EMA observations. Standard deviations have been used to measure mood variability in the majority of experience sampling/ecological momentary assessment studies (e.g., Eid & Diener, 1999; Hoeksma et al., 2004; Larson et al., 1980, 1990; Penner, Shiffman, Paty, & Fritzsche, 1994; Silk et al., 2003). This index of mood variability quantifies the tendency to experience a range of intraindividual fluctuation in negative mood levels within a typical week, such that larger values reflect the experience of frequently varying and intense levels of negative affect. A wider range of fluctuation in negative affect is conceptualized as the result of dysfunctional emotion regulation abilities.

We examined the broad continuum of smoking patterns - including adolescents who never smoked, those who experimented, and those who progressed to more frequent smoking - to better understand the factors influencing escalation in cigarette use. Specifically, we addressed the following questions: (1) Do high levels of intraindividual variability in negative moods predict smoking escalation? and (2) Do levels of negative mood variability change as smoking patterns escalate over time? We hypothesized that baseline levels of negative mood variability would predict future smoking patterns, specifically escalation, and would differentiate youth who progressed to heavier levels of smoking from those who never progressed beyond experimentation. Second, as predicted by the self-medication model of cigarette use (Khantzian, 1997), we expected that increased cigarette use over time would be associated with stabilization of affect regulation (i.e., reduced negative mood variability).

Methods

Design Overview

Data for this study come from a longitudinal natural history study of adolescent smoking. The longitudinal study used a multi-method approach to assess adolescents including 7-day time/event sampling via palmtop computers (ecological momentary assessment), self-report questionnaires, and in-depth interviews at baseline, 6-, and 12-month waves.

Participants

The sample for the longitudinal study was recruited through a multi-stage process. A screening survey was administered to all 8th and 10th graders at 14 Chicago-area schools (N = 5,278); a waiver of written parental consent was used such that parents and students were notified prior to the screening and given the opportunity to decline their child's participation. Survey responses were used to identify students who were either susceptible adolescents who had not yet smoked but indicated a probability of future smoking, or experimenters (never having

smoked more than 100 cigarettes in their lifetime). The sampling rationale was based on our desire to follow youth who might initiate and progress in their experimentation with smoking during the course of the study. Of those who met inclusion criteria (*N* = 2,153), a random sample of 1,457 students was invited to participate. Recruitment packets were mailed to eligible students and their parents. Students were enrolled after written parental consent and student assent were obtained. Of those invited, 713 agreed to participate (48%), and 562 of the 713 (81%) completed the baseline wave. The consent rate is similar to those reported in other EMA and experience sampling studies with adolescents (e.g., Larson, Moneta, Richards, & Wilson, 2002; Silk et al., 2003). Failure to complete the baseline assessment was due to 1) students being sick/absent for their data collection visit and unable to reschedule (*n* = 25, 17%); 2) students not bringing parental consent forms to their appointment (*n* = 12, 8%); or 3) students being turned away by research staff because enrollment in a particular school was overfilled (*n* = 114, 76%). Details regarding the procedures can be found in Diviak, Kohler, O'Keefe, Mermelstein, & Flay, 2006.

The sample for the current study includes eighth and 10th grade students who provided EMA data at baseline (*N* = 517). Due to EMA hardware malfunction during the data week, 8% of the total sample did not provide EMA data; there were no significant differences on any variables for those who did and did not provide EMA data. Mean age of the participants was 14.4 years (*SD* = 1.20); 56% were girls, and racial/ethnic composition was as follows: 74% White; 5% African American; 13% Latino; and 8% Other/Bi-racial. At baseline, 67% (*N* = 371) of the total sample reported ever smoking in their lifetime and 35% (*N* = 192) reported smoking within the past 30 days. Of those reporting past month smoking, 58% (*N* = 115) smoked on 1 - 3 days, 19% (*N* = 37) smoked on 4 - 7 days, 5% (*N* = 9) smoked on 8 - 10 days, and 19% (*N* = 37) smoked on 11 or more days. Average daily smoking rates during the past 30 days ranged from 0 to 15 cigarettes/day (*Mdn* = 0, *M* = 0.29, *SD* = 1.32).

Procedures

All study procedures received approval from the Institutional Review Board at the University of Illinois at Chicago. Data collection occurred via three modalities at each wave: EMA, self-report questionnaires, and in-depth interviews. For the EMA portion, all participants received training on the use of the EMA device at the beginning of the data collection week, and carried the device for seven consecutive days at each wave. The device randomly prompted the adolescents approximately 5-7 times per day to answer questions about their mood, behavior, and situation; only mood items were analyzed in the current study. Participants received a payment of $40 at the end of each data week. In addition, self-report questionnaire packets were mailed to the adolescents two weeks prior to each data wave. The participant was instructed to bring the completed packet to his/her EMA training session and were paid $10 upon receipt of each completed packet.

Finally, timeline follow-back interviews were conducted with each participant at each wave at his/her EMA training session. Participants completed a structured in-depth interview with project staff to develop a continuous calendar of smoking behavior over the prior six months. Participants were first asked to list notable events over the past six months (e.g., birthdays, school exams, vacations, and parties) to aid recall, and then were asked to reconstruct smoking experiences during this time period, noting the amount smoked on specific days.

Measures

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

Smoking Behavior (Interview and Questionnaire) was assessed via structured timeline follow-back interviews conducted with each participant. The interview data were used to create a continuous calendar of each participant's daily smoking behavior (counts of cigarettes per day), from six months prior to baseline through the 12-month wave (referred to as "smoking calendar data"). To better focus on trends over time, the data were aggregated into a summary smoking rate variable: the number of cigarettes smoked per day for each of the two 90-day intervals within the six-month interval, generated by computing the total number of cigarettes smoked in the 90-day period divided by the number of reported days. Given the positively skewed distribution of this variable, we derived an ordinal categorization: 0 = 0 cigarettes in a time period; 1 = greater than 0, but less than 1 cigarette/month; 2 = greater than/equal to 1 cigarette/month, less than 1 cigarette/week (monthly smoking); 3 = greater than/equal to 1 cigarette/week, less than 1 cigarette/day (weekly smoking); and 4 = greater than/equal to 1 cigarette/day (daily smoking). Given the low rates of smoking in this sample, these categories were chosen to reflect meaningful substantive classifications of adolescent smoking behavior.

The reliability of these retrospective reports was supported by the strong correspondence with both daily diary reports of smoking episodes as well as questionnaire reports of smoking behavior over the past 90 days (Diviak, Kohler, Mermelstein, & Flay, 2001). Moreover, by using interview aided-recall for the calendar data, a more accurate measure of smoking activity is obtained than by relying strictly on the questionnaire data.

Smoking history was also assessed via self-report questionnaire at each wave with several items: 1) number of days smoked in the past 30 days, with response categories ranging from 1 (none) to 9 (all 30 days); and 2) number of cigarettes per day on days smoked in the past 30 days, with response categories ranging from 1 (none) to 11 (more than 20 per day). Based on these data, daily smoking rates were generated by computing the average number of cigarettes smoked per day in the past 30 days at each wave (referred to as "daily smoking rates"). A third item assessed the lifetime number of cigarettes, with response categories ranging from 1 (I have never smoked) to 9 (500 or more). The questionnaire data were used to examine participants' smoking behavior descriptively over time, but were not included in the study's main analyses.

Mood Variability (EMA)—Participants were asked on each EMA interview to rate their mood just before the random prompt; for example, "Before the signal, I felt angry." Subjects responded to mood adjectives using a 10-point Likert-type scale, ranging from 1 (not at all) to 10 (very). The adjectives were selected based on pilot work, involving qualitative (focus groups and in-depth interviews) and quantitative data collection with 146 8^{th} and 10^{th} graders recruited from similar schools to those involved in the present study. Confirmatory factor analyses on the current sample revealed a "Negative Affect" (NA) factor, formed by lonely, embarrassed, sad, angry, and left-out, all with factor loadings greater than .50 (Cronbach's alpha ranged from .73 to .78). Other items comprised additional mood factors, but we focus here on only Negative Affect.[1] An index of mood variability was constructed from EMA mood ratings by computing standard deviation scores for the negative affect scale for each participant across the measurement week at each data collection wave. Mood variability thus reflected the degree of intraindividual fluctuation in negative mood states across the week. Research supports the reliability and validity of the intraindividual standard deviation as a measure of mood variability (Eid & Diener, 1999;Penner et al., 1994), and standard deviations have shown

[1]We focus on negative mood variability exclusively for theoretical as well as empirical reasons. Whereas previous research has shown relations between negative mood variability and clinical outcomes (e.g., depressive symptoms and problem behaviors, Silk et al., 2003; substance use, Simons et al., 2004), positive mood variability is associated with adaptive personality traits (Eid & Diener, 1999) and thought to reflect a normative part of adolescent emotional experience (Larson et al, 1980). Additionally, in pilot work, positive mood variability was not significantly related to current or future smoking behavior (Weinstein & Mermelstein, 2007).

relations to internalizing and externalizing symptomatology (Larson et al., 1990;Silk et al., 2003).

Results

Analytic Approach

To examine and predict change in smoking behavior over time, a variant of latent growth curve analysis (i.e., trajectory analysis; Nagin, 1999) modified for ordinal responses (Hedeker, 2000) was used to derive groups of longitudinal patterns of smoking from the smoking calendar data. Trajectory analyses are advantageous because they model longitudinal data in terms of trend parameters and summarize the continuum of trends into a relatively small number, thus statistically identifying the main systematic characteristics of the continuum. The trajectory analysis identified six primary classes of smoking trajectories, and Figure 1 illustrates the patterns of the smoking groups over time, including: nonsmokers, who had never tried a cigarette or at baseline reported having ever tried a cigarette in their life, but did not smoke during the study (n = 210; 37%); triers, who engaged in very low levels of experimentation with cigarettes but never escalated (n = 169, 30%); escalators, who steadily escalated from low levels of use to daily smoking (n = 61; 11%); rapid escalators, who rapidly escalated from low levels of use to daily smoking (n = 36, 6%); quitters (n = 54; 10%); and smokers, who smoked at heavier levels of use (weekly to daily smoking) throughout the study (n = 32; 6%).

As the figure reveals, smoking patterns diverged over time. Longitudinal smoking patterns as derived from the trajectory analyses converged with participants' daily smoking rates, indexed using the self-report questionnaire data at each wave. For example, triers maintained a low level of use throughout the study: at baseline, triers reported having smoked on average 0.30 (SD = 0.84) days in the past 30 days, with an average daily rate of 0.01 (SD = 0.04) cigarettes/day; at 12 months, triers smoked on average 0.16 (SD = 0.60) days in the past 30 days, with a daily rate of 0.01 (SD = 0.06) cigarettes/day. In contrast, escalators and rapid escalators combined smoked on average 5.44 (SD = 8.04) days in the past 30 days at baseline, with a mean rate of 0.65 (SD = 1.57) cigarettes/day, increasing to 10.3 (SD = 11.0) days at 12 months and a mean rate of 2.01 (SD = 3.69) cigarettes/day. Thus, consistent with their group status, all escalators increased in their smoking behavior over time. Paired t-tests confirmed that differences in daily rates between each wave were all significant. The smokers maintained a higher level of use throughout the study: at baseline, smokers averaged 11.7 (SD = 11.5) days of smoking with a daily rate of 2.30 (SD = 4.07) cigarettes; values were similar at 12 months, smoking on average 13.1 days (SD = 12.5), with a daily rate of 2.62 cigarettes (SD = 4.30). For the smokers, differences in daily rates between each wave were not significant.

By identifying longitudinal patterns of smoking, we were able to examine mood variability as a predictor of these patterns. Mood variability was measured via the intraindividual standard deviations of momentary mood assessments across EMA observations; this approach has the same, if not fewer, limitations than alternate measurement approaches (e.g., spectral analysis; Eid & Diener, 1999; Larsen, 1987). Given the focus on mood variability (aggregating across momentary mood assessments within an individual), the person was the unit of analysis in all analyses.

Compliance and Attrition

Participants provided mood reports for a mean of 33.50 (SD = 9.86) random prompts from the EMA device per person per wave and missed a mean of 5.70 (SD = 5.75) prompts. In total, participants responded to 85% of all random prompts (SD = 0.14). Furthermore, 89% of the random prompts were answered within 3 minutes of the signal. Attrition in the current study was minimal. At the final wave (12 months), 507 adolescents (90%) participated in data

collection. Analyses verified that there were no significant differences in retention for grade, sex, race/ethnicity, or smoking status, nor for baseline reports of daily negative mood and negative mood variability (effect sizes *d* ranging from .04 to .10).

Preliminary Analyses

Descriptive statistics for overall negative mood, mood variability, and daily smoking rates at all waves for the total sample, and also stratified by sex, are shown in Table 1. As the table reveals, the sample was not very distressed; mean Negative Mood ratings hovered around 2.4 (higher scores reflect greater negative mood). Girls reported significantly higher levels of mood variability than did boys at each wave. Additionally, daily smoking rates for girls were significantly higher than boys' daily rates. Analyses also examined correlations among the mood variables. Negative mood variability was fairly stable over time, with interwave correlations ranging from $r = .50$ to .56, p's < .01. Mean negative mood and mood variability were strongly and positively correlated; r's range from .61 to .67 over time, p's < .01.

Negative Mood Variability as a Predictor of Future Smoking Patterns

We hypothesized that baseline negative mood variability would differentiate adolescents who escalated in their smoking over time from those who did not progress in their cigarette use. To test this hypothesis, we conducted a one-way between-subjects analysis of variance (ANOVA) to investigate differences in baseline negative mood variability among longitudinal smoking trajectory groups. Given that the smoking variable represents longitudinal smoking patterns, this analysis allowed us to examine mood variability at baseline as a prospective predictor of future smoking patterns. Baseline negative mood variability significantly differed among the smoking groups, F (3, 517) = 3.20, $p = .02$, $\eta^2_{partial} = .03$. Figure 2 displays mean negative mood variability, as well as mean overall negative mood, at baseline as a function of smoking group. As the figure illustrates, those who escalated in their smoking pattern over time (the escalators and the rapid escalators) have the greatest levels of mood variability at baseline.

To further examine our hypotheses, three planned comparisons were conducted: (1) the rapid escalators versus all other smoking groups; (2) all smoking escalators (escalators and rapid escalators) versus those who tried smoking but never progressed beyond experimentation (triers); and (3) all smoking escalators versus the never smokers. We were specifically interested in the rapid escalators as this represented the group with the greatest change in smoking over time, but also focused on differences between all of those who escalated as compared to those who never progressed in their smoking behavior. The rapid escalators had significantly higher levels of negative mood variability at baseline as compared to all other smoking groups, F (1, 38) = 4.16, $p = .04$. In addition, as predicted, adolescents who escalated in smoking over time had significantly higher levels of mood variability at baseline as compared to those who experimented but did not progress in their cigarette use, F (1, 102) = 4.80, $p = .03$, as well as compared to the never smokers, F (1, 89) = 10.43, $p = .002$.

Interestingly, Figure 2 also reveals that the regular smokers - who at baseline have heavier levels of cigarette use - have lower levels of negative mood variability, appearing more similar to the nonsmokers. Indeed, a post hoc comparison confirmed that differences between the never smokers and the regular smokers were not significant, F (1, 31) = 0.01, *ns*. We had no specific hypotheses about differences between the remaining smoking groups, and post hoc Tukey tests indicated that no further pairwise comparisons were significant.

Of note, given the strong correlation between mean negative mood and mood variability, we repeated these analyses using mean negative mood as a covariate for the omnibus test and all follow-up analyses. When controlling for mean mood, the pattern of results was identical and significance levels remained unchanged. Results thus suggest that mood variability,

independent of mean mood levels, differentiates adolescents at risk for smoking escalation. Figure 2 displays the patterns of mood variability versus mean negative mood among the smoking groups. The patterns are generally similar with the exception of the smokers, who have low levels of variability but the highest level of mean negative mood. These findings suggest that overall mood and mood variability are related, but still distinct, dimensions of affect.

Longitudinal Smoking-Mood Relationships

We also hypothesized that increased smoking would lead to subsequent improvements in affect regulation, as indicated by a reduction in negative mood variability over time among the escalating adolescents. To test this hypothesis, we used random intercept and trend mixed-effects regression models for continuous outcomes (MRMs; Laird & Ware, 1982) via SAS PROC MIXED. Mixed effects regression models are well-suited for the analysis of longitudinal data: these models are robust to the data dependency that occurs with the repeated assessments of individuals over time, and also can handle missing data. We used random intercept and trend modeling, a subclass of MRM that accounts for each individual's distinct initial level of mood variability and rate of change across time.

We conducted two MRMs: (1) comparing adolescents who escalated in cigarette use over time (rapid escalators and escalators, collapsed into one group) versus all nonescalators (including the non-smokers and the non-escalating triers, collapsed into one group); and (2) comparing all escalators versus the regular smokers. These analyses thus examined changes in mood variability for those who escalated versus those who maintained a more stable level of high or low cigarette use during the study. Each contrast MRM included the effects of Smoking Group (Escalators v. Nonescalators or Smokers), Time (Baseline, 6-months, 12-months), and Smoking Group × Time on negative mood variability. Given the sex differences in mood variability, sex was included as a control in each model. For inclusion in these analyses, participants had to provide EMA data at 2 or more waves ($N_1 = 435$, yielding 1,214 observations of mood variability; $N_2 = 118$, 331 observations).

Results of the contrast models are presented in Table 2. The first model (Escalators v. Nonescalators) indicated a significant effect of sex, with girls reporting greater negative mood variability than boys overall. Mood variability significantly decreased over time, and escalators reported higher overall levels of mood variability than did the nonescalators. Findings also revealed a trend for the Smoking Group × Time interaction, indicating that changes in mood variability over time differed for the escalators versus the nonescalators. To best illustrate this interaction, Figure 3 displays estimated negative mood variability over time as a function of smoking level. Consistent with our predictions, those who escalated in their smoking also experienced greater changes in mood variability over time, with negative moods stabilizing as smoking increased. In contrast, the nonescalators had lower mood variability at baseline, and remained more stable over time.

Results of the second contrast model (Escalators v. Smokers) revealed similar effects for sex and time; neither the effects of Smoking Group nor the Smoking Group × Time interaction were significant. Examination of Figure 3 suggests that unusual findings for the smokers at the six-month time point likely influenced this analysis and rendered interpretation difficult. Despite the nonsignificant interaction, findings indicated that although the escalators had higher levels of negative mood variability at baseline as compared to the smokers, these values converged by 12 months (i.e., when escalators were smoking at similar levels as the smokers, as indicated by the daily smoking rates: 2.01 cigarettes/day, versus smokers baseline rates of 2.30 and 12-month rates of 2.62 cigarettes/day).

Follow-up analyses focused on the rapid escalators as compared to the nonescalators and the smokers. Again, we were specifically interested in the factors that differentiated youth with the steepest smoking trajectory from the other smoking groups. Thus, we conducted a final MRM that included the effects of Smoking Group (Rapid Escalators, Nonescalators, Smokers), Time, and Smoking Group × Time, controlling for sex (N = 405). Significant effects were found for sex (Estimate = 0.11, SE = .04), F (1, 320) = 7.22, p < .008; time (Estimate = -0.14, SE = .04), F (1, 402) = 10.65, p = .001; and smoking group (Rapid escalator v. Nonescalator, Estimate = -0.26, SE = .12; Rapid escalator v. Smoker, Estimate = -0.24, SE = .09; F (2, 320) = 4.47, p = .01. In addition, findings revealed a trend for the Smoking Group × Time interaction, F (2, 320) = 2.51, p = .08, indicating that changes in negative mood variability over time varied as a function of smoking level. To specify the source of this effect, follow-up smoking group contrasts revealed that the interaction was primarily driven by the group by time interaction at the level of Rapid Escalators v. Nonescalators, Estimate = .10, SE = .05, t (320) = 2.22, p = .02. Thus, the rapid escalators experienced significantly greater changes in mood variability over time as compared to the nonescalators (see Figure 3). However, the Rapid Escalators v. Smokers interaction contrast was also significant at the p < .10 level, Estimate = .11, SE = .07, t (320) = 1.66. In line with our hypothesis, the rapid escalators experienced a steep reduction in negative mood variability over time, whereas both the nonescalators and the smokers (with the exception of the 6 month wave) had lower, more consistent levels of mood variability over time. Thus, findings suggest that mood variability may decrease as a function of smoking escalation.

All longitudinal MRM analyses were repeated using mean negative mood as a covariate. When controlling for mean negative mood, the interactions of Smoking Group × Time were not significant; effects remained in the same direction as the initial models. In light of these findings, we examined specifically the longitudinal patterns of mean negative mood among the smoking groups to investigate the possibility that the observed changes in mood variability over time may be a function of changes in mean levels of negative mood over time. A series of MRMs examined the effects of Time, Smoking Group (examining each contrast), and Smoking Group × Time on mean negative mood, controlling for sex. Findings revealed that the interactions of Smoking Group × Time were not significant, indicating that changes in overall mood over time did not vary by smoking group. Findings revealed a significant main effect for Time (Estimate = -0.045, Standard Error = 0.02, t = -2.24, p < .05), indicating that all adolescents experienced a reduction in negative mood over time. No other effects were significant when examining mean mood levels. These findings suggest that mood variability may show unique relations with smoking over time, but small sample sizes likely influenced the statistical significance of findings in the full model.

Discussion

Our study examined the role of intraindividual mood variability, as assessed in “real time”, as a predictor of adolescent smoking patterns. Despite much research documenting a link between moods and smoking in adolescence (e.g., Choi, Patten, Gillin, Kaplan, & Pierce, 1997; Windle & Windle, 2001), far less is known about the emotional factors influencing the escalation from experimentation to regular smoking. To enhance understanding of the progression to and maintenance of higher levels of cigarette use in adolescence, we investigated prospective as well as reciprocal relationships between smoking patterns and mood variability over time.

Negative Mood Variability: Risk and Maintenance Factor for Smoking Escalation

Consistent with our hypotheses, results suggest that negative mood variability is a risk factor for smoking escalation. Higher levels of mood variability prospectively predicted longitudinal patterns of increased cigarette use and differentiated the escalating adolescents from those who

never progressed beyond low levels of experimentation. In addition, the predictive power of mood variability was distinct from overall negative mood, as effects remained significant when controlling for mean mood levels. Our results are congruent with past research demonstrating the relevance of affect dysregulation for adolescent substance use (Simons & Carey, 2002; Simons et al., 2004; Wills et al., 2006) and also corroborate Novak and Clayton's (2001) cross-sectional findings that deficient emotional regulation predicted transitions from smoking experimentation to daily cigarette use using longitudinal data. Adolescents with greater mood lability may have underdeveloped internal resources for regulating emotional states and thus be more likely to seek external and nonconstructive means of regulation (Eisenberg & Fabes, 1992). In line with the self-medication model (Khantzian, 1997), the present results as well as past research suggest that youth with affect regulation vulnerabilities may escalate in their cigarette use as a means of coping with labile moods.

For the escalating youth, longitudinal analyses suggested a possible self-medication function of smoking: adolescents with increased smoking experienced a trend of improved affect regulation over time, as indicated by the reduction of negative mood variability, compared to the stable mood profile of the non-smoking/non-escalating adolescents. Of note, these trends were distinct from the longitudinal patterns of mean negative mood levels, which did not vary by smoking group. Mood stabilizing effects were particularly apparent among those with the steepest smoking trajectory, and the regular smokers had levels of mood variability as low as the nonsmoking adolescents. Thus, results suggest that smokers may use cigarettes to "normalize" mood volatility. In addition, collective findings suggest that the regulation of affect (i.e., the enhancement of mood stability) may be more important to understanding youth smoking escalation than improvement in overall negative mood levels.

Such mood regulatory benefits may play an important role in the progression to regular smoking and the development of nicotine dependence. Learning models of addiction and substance dependence posit that smoking-related mood improvement, such as reduced emotional distress or lability, acts as a reinforcer for cigarette use and increases the likelihood of future smoking through negative reinforcement (Shadel, Shiffman, Niaura, Nichter, & Abrams, 2000). Indeed, our findings for the regular smokers suggest that mood stabilizing effects may maintain a high level of smoking, independent of improvements in average mood. A reduction in mood variability may be uniquely reinforcing, as mood swings may be a source of discomfort for adolescents. By modulating the frequency and/or intensity of these affective changes (i.e., reducing the swings between emotional extremes), cigarette use serves an important regulatory function and, in turn, may enhance a youth's sense of emotional control (Khantzian, 1997). Thus, transactional relations between mood variability and cigarette use over time may help explain the maintenance of regular cigarette use in adolescence.

Several mechanisms may account for the mood regulatory trends accompanying higher levels of smoking among the adolescent escalators and smokers. Findings from adult brain imaging research indicate that acute and chronic nicotine exposure results in increased dopamine concentration in the ventral striatum/nucleus accumbens as well as inhibited monoamine oxidase (MAO) activity in the basal ganglia, which may mediate the rewarding and mood-palliative effects of smoking (Brody, 2006). Moreover, individuals with affect regulation difficulties may have heightened sensitivity to the therapeutic effects of nicotine (Choi et al., 1997). Such mood-enhancing effects may translate into reduced emotional volatility (i.e., preventing negative moods from peaking) as adolescents increase the frequency of their cigarette use. In addition, smoking may have lasting mood stabilizing effects via nicotinic receptor functioning. Prolonged nicotine exposure has been shown to result in the loss of neuronal nicotinic acetylcholine receptor (nAChR) function, or desensitization of nAChRs (Brody et al., 2006; Quick & Lester, 2002). Evidence suggests that inhibition of nAChRs reduces mood instability (Shytle et al., 2002). As such, it is possible that smoking-related

desensitization of nAChRs may mediate mood stabilization among the adolescent escalators and smokers. It has been proposed that these effects may be state-dependent, whereby nicotine exposure increases mood instability among those of stable mood while stabilizing mood in individuals with pre-existing mood-instability (Shytle et al., 2002).

Withdrawal models of cigarette use offer an alternate explanation of the observed mood variability patterns, asserting that fluctuating moods are a consequence - not a predictor - of smoking escalation (Parrott, 1999). It is possible that the acute affective benefits of cigarette use among heavier smokers signal the reversal of unpleasant withdrawal effects (e.g., irritability, frustration, and anger; APA, 1994), rather than a direct mood improvement. In this vein, erratic cigarette use among the smoking escalators may account for the observed high levels of mood variability at baseline, with the onset and subsequent alleviation of withdrawal symptoms yielding a fluctuating affective profile (Parrott, 1999). As adolescents increased their cigarette use and experienced smaller periods of nicotine deprivation, withdrawal symptoms may not have peaked and thus moods appeared stabilized. According to withdrawal models, the negatively reinforcing effects of smoking via withdrawal symptom relief motivate and maintain regular cigarette use (Baker, Piper, McCarthy, Majeski, & Fiore, 2004).

Although important to consider, the current results argue against a withdrawal model interpretation of mood variability-smoking relationships. First, differences in mood variability were observed at baseline, when the smoking escalators had low levels of cigarette use (e.g., 63% reported smoking less than/equal to 0.08 cigarettes/day during the past 30 days). Thus, prospective findings suggest that mood variability is a predictor of smoking escalation, and not a function of nicotine withdrawal and alleviation patterns. In addition, the majority of mood items in the study (e.g., feeling lonely, embarrassed and left-out) do not reflect typical withdrawal symptoms and therefore would not be expected to worsen as a result of nicotine deprivation.

Implications, Limitations, and Future Directions

In sum, results suggest that transactional mood-smoking relationships may comprise part of the complex etiology of regular smoking among youth. High levels of negative mood variability may confer risk for cigarette use to self-medicate labile moods; in turn, the reduction of mood variability among the smoking escalators may reinforce and maintain their upward smoking trajectory. These findings raise important implications for the prevention and treatment of youth smoking. Our results suggest that dysregulated adolescents may be a key target for prevention and early intervention efforts. Moreover, previous research has found that good emotional control reduced risk for adolescent substance use (Novak & Clayton, 2001; Wills et al., 2006). As such, an important aim for smoking prevention and intervention programs for high risk youth may be enhancing emotion self-regulation via training in cognitive and behavioral regulatory strategies (e.g. Contextual Emotion-Regulation Therapy; Kovacs et al., 2006).

The present investigation extends previous research on the link between emotion self-regulation and adolescent smoking by using a three-wave longitudinal design, real-time methods of mood assessment, and statistical techniques best suited for identifying longitudinal smoking patterns. Nonetheless, several study limitations should be noted. First, the method used did not directly examine causal relationships between smoking and mood and thus interpretations regarding the mood stabilizing influences of smoking escalation must be made cautiously. Future research should continue to explore the causal mechanisms underlying smoking-mood relationships among youth. Second, our sampling strategy focused on the initial stages of cigarette use and, as result, we had fewer experienced smokers. Thus, limited statistical power may account for the marginally significant interactions in some models.

Last, we must consider limitations regarding the use of intraindividual mood variability as a proxy measure of affect dysregulation. Developmental literature suggests that emotion regulation is a complex, multifaceted construct (e.g., Cole, Martin, & Dennis, 2003; Eisenberg & Spinrad, 2004; Forbes & Dahl, 2005), and we only assessed one component of affect dysregulation. Although research supports the connection between emotion dysregulation and mood lability (Hoeksma et al., 2004; Wills et al., 2006), it will be important to replicate the current results with a broader measure of affect dysregulation (e.g., incorporating measures of rumination and soothability, cf. Wills et al., 2006). Along these lines, further investigation of the standard deviation as an index of affect dysregulation is warranted; we considered higher standard deviations to reflect problematic variability, but future work would benefit from operational definitions of normative versus abnormal degrees of variability. Despite these limitations, the present study offers insight into the etiology of adolescent smoking. Our findings provide preliminary evidence of affect dysregulation, specifically negative mood variability, as one factor that differentiates normative versus maladaptive smoking trajectories.

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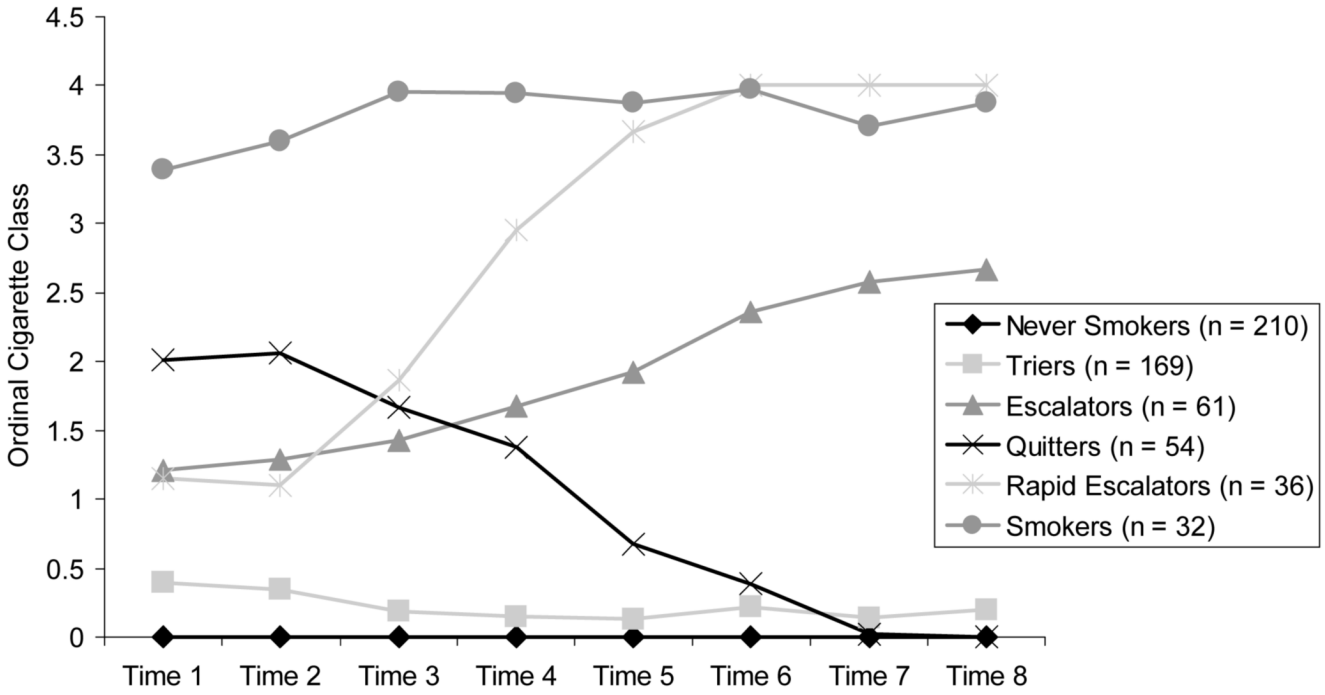

Figure 1.

Longitudinal patterns of cigarette use, indexed via ordinal cigarette class, among the smoking trajectory groups. Each time point reflects smoking over a 3 month period, beginning with six months prior to Baseline (Time 1 = 6 months pre-Baseline to 3 months pre-Baseline; Time 2 = 3 months pre-Baseline to Baseline; Time 3 = Baseline to 3 months; Time 4 = 3 months to 6 months; Time 5 = 6 months to 9 months; Time 6 = 9 months to 12 months; Time 7 = 12 to 15 months; Time 8 = 15 to 18 months). Of note, the figure displays smoking data through 18 months because additional smoking data were collected from participants at an 18 month wave. The EMA data were not collected as this time point and thus the 18 month wave was excluded from all analyses.

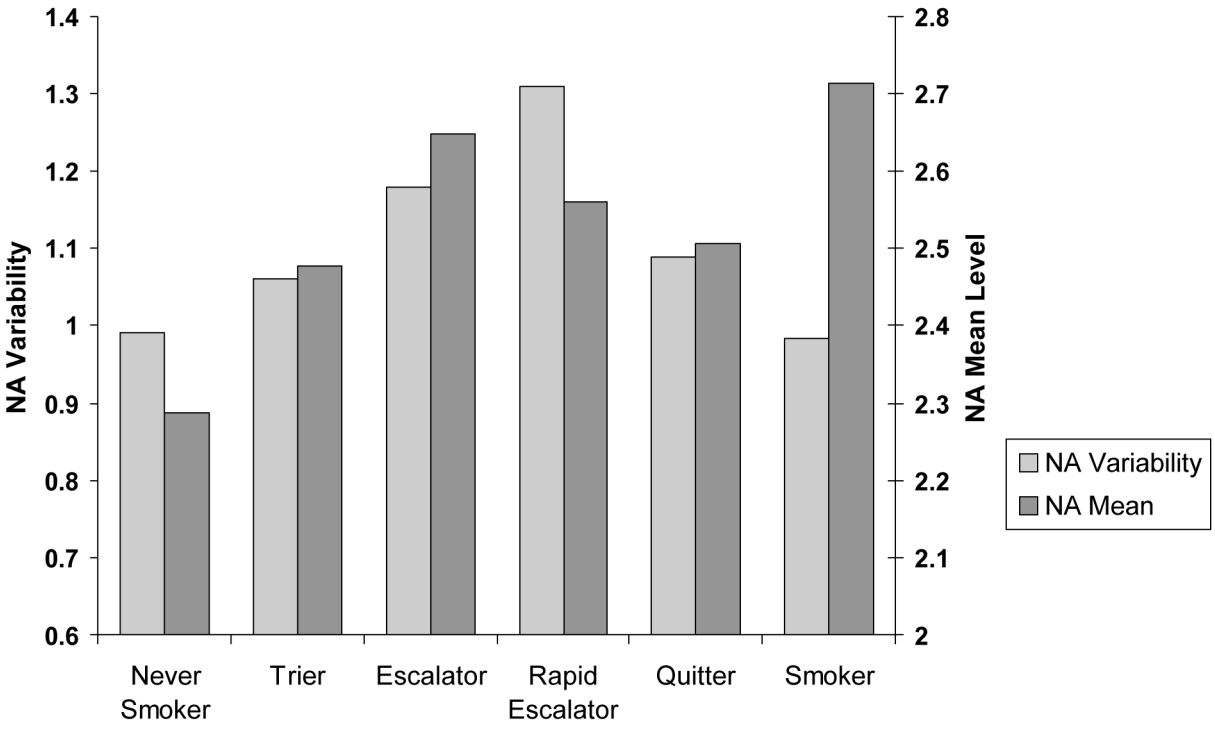

Figure 2.
Estimated negative mood variability (primary Y axis) and mean overall negative mood (secondary Y axis) at baseline as a function of longitudinal smoking group.

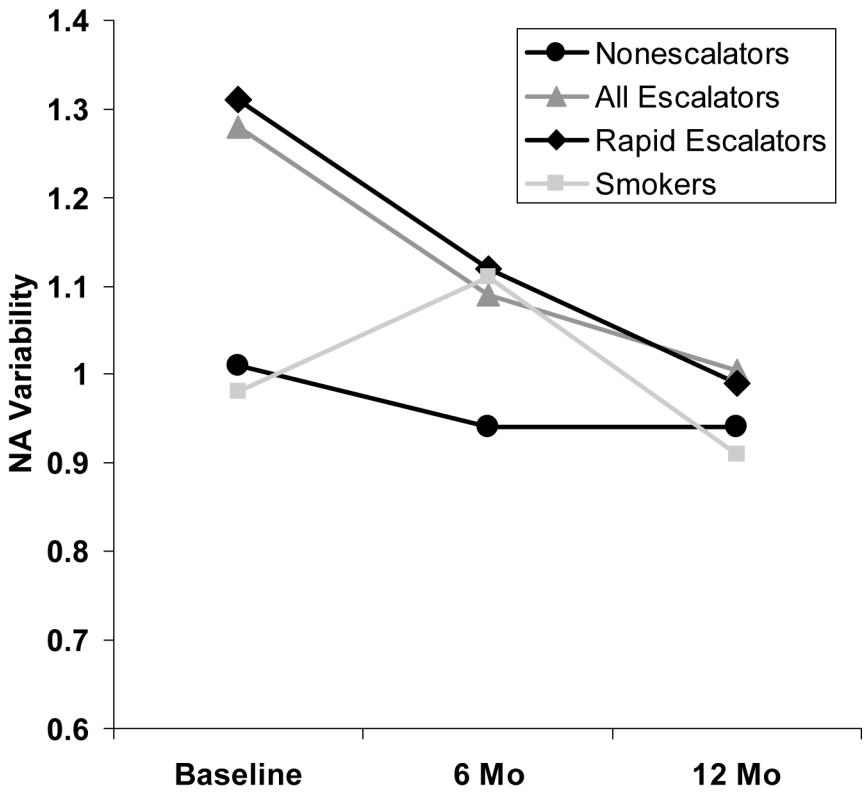

Figure 3.
Estimated negative mood variability over time as a function of smoking group.

Table 1

Descriptive Statistics for Mood and Smoking Variables, for the Total Sample and by Sex

	Total				Girls				Boys			
	N	Range	*M*	*SD*	*N*	Range	*M*	*SD*	N	Range	*M*	*SD*
BASELINE												
Negative Mood	517	1.00 - 6.36	2.44	0.97	287	1.02 - 6.36	2.47	0.93	230	1.00 - 5.83	2.42	1.02
Mood Variability	517	0.00 - 2.73	1.07	0.48	287	0.10 - 2.73	1.13	0.48	230	0.00 - 2.52	0.99	0.48 **
Daily Smoking Rate	512	0.00 - 15.00	0.30	1.36	285	0.00 - 15.00	0.45	1.75	227	0.00 - 6.67	0.12	0.53 **
6 MONTHS												
Negative Mood	487	1.00 - 6.03	2.40	1.07	264	1.00 - 5.58	2.35	0.98	223	1.02 - 6.03	2.45	1.17
Mood Variability	487	0.03 - 3.12	0.99	0.52	264	0.03 - 3.12	1.03	0.51	223	0.07 - 2.61	0.94	0.52 *
Daily Smoking Rate	477	0.00 - 20.00	0.45	1.92	258	0.00 - 20.00	0.68	2.53	219	0.00 - 4.17	0.18	0.60 **
12 MONTHS												
Negative Mood	466	1.01 - 7.13	2.32	1.04	255	1.01 - 4.92	2.25	0.88	211	1.03 - 7.13	2.39	1.20
Mood Variability	466	0.03 - 3.16	0.96	0.50	255	0.03 - 3.16	1.01	0.50	211	0.16 - 2.58	0.89	0.50 *
Daily Smoking Rate	465	0.00 - 20.00	0.60	2.12	251	0.00 - 20.00	0.89	2.70	214	0.00 - 8.00	0.26	1.00 **

Note. Negative Mood: Negative Affect Scale - EMA; Mood Variability: Intraindividual standard deviations of Negative Affect Scale;

Daily Smoking Rate: Average number of cigarettes smoked per day for the past 30 days - Self-Report Questionnaire.

For comparison of Girls versus Boys,

* $p < .05$

** $p < .01$

Psychol Addict Behav. Author manuscript; available in PMC 2008 December 19.

Table 2

Mixed Effects Regression Contrast Model 1 (All Escalators v. Nonescalators, N = 435) and Contrast Model 2 (All Escalators v. Smokers, N = 118) on Negative Mood Variability

	Contrast 1: Escalators v. Nonescalators				Contrast 2: Escalators v. Smokers			
Effect	**Parameter Estimate**	***SE***	***F***	***p***	**Parameter Estimate**	***SE***	***F***	***p***
Intercept	1.11	.06	20.04	<.001	1.04	.08	13.62	< .001
Sex	0.12	.04	9.65	.002	0.25	.08	9.58	.003
Time	-0.09	.03	20.09	<.001	-0.09	.03	3.61	.005
Smoking Group	-0.17	.06	8.91	.003	-0.13	.12	1.10	*ns*
Smoking Group × Time	0.05	.03	3.09	.079	0.06	.07	0.81	*ns*

Note. Contrast 1 Model χ^2 (3, N = 435) =295.60, p < .0001; Contrast 2 Model χ^2 (3, N = 118) = 74.99, p < .0001.

Psychol Addict Behav. Author manuscript; available in PMC 2008 December 19.