Mood Variability and Cigarette Smoking Escalation Among Adolescents

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The current study examined how affect dysregulation, as indexed via within-person negative mood variability, related to longitudinal patterns of smoking among adolescents. Students in the 8th and 10th grades (N = 517, 56% girls) provided data on cigarette use at baseline, 6-, and 12-month waves and provided ecological momentary assessments of negative moods via palmtop computers for 1 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 participants who escalated in their smoking behavior over time from participants who never progressed beyond low levels of experimentation during the course of the study. Mixed-effects regression models revealed that participants who escalated in their smoking experienced a reduction in mood variability as smoking increased, whereas participants with consistently high or low levels of cigarette use had more stable mood variability levels. Results suggest that high negative mood variability is a risk factor for future smoking escalation and that mood-stabilizing effects may reinforce and maintain daily cigarette use among youths.

Keywords: cigarette smoking, adolescence, moods, affect regulation, ecological momentary assessments

Although a considerable proportion of youths experiment with smoking, fewer adolescents progress to heavier levels of smoking. According to Johnston, O’Malley, Bachman, and Schulenberg (2006), 26% of 8th graders and 50% of 12th graders reported having ever smoked cigarettes, whereas 14% of 12th graders reported smoking on a daily basis. Understanding the factors that differentiate adolescents who progress beyond experimentation with smoking from adolescents who engage in experimentation but never escalate to heavier levels of cigarette use can greatly inform prevention and intervention efforts. To this end, in the current study we utilized ecological momentary assessments (EMAs; 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 initiation and escalation of smoking among youths. Emotional self-regulation refers to the involuntary and 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; Hoeksema, Oosterlaan, & Schipper, 2004). That is, an individual with limited affect regulation is expected to experience varying intensities of negative emotions, with greater peak intensities, that yield a variable affective profile over a period of time; in contrast, an emotionally regulated individual would exhibit a more stable affective profile. We thus conceptualized high levels of negative mood variability as a by-product of affect dysregulation.
Evidence suggests an association between affect dysregulation and substance use in adolescence, although conceptualizations of affect regulation vary across studies. Wills, Walker, Mendoza, and Ainette (2006) found that poor emotional control (as indexed by global self-report questionnaire measures of affective lability, re- 
mination, 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 that assessed the ability to control extreme affect and to regulate negative moods) predicted adolescent experiment-
imentation with cigarettes as well as progression to regular use (Novak & Clayton, 2001). Moreover, a series of studies by Simons and colleagues (Simons & Carey, 2002; Simons, Carey, & Gaher, 2004) demonstrated significant relationships between affect labil-
ity (assessed by a subjective global questionnaire on the frequency and extremity of mood shifts) and problems with alcohol and 
marijuana use among college students.

Taken together, theory and research suggest that labile youths who lack internal regulation resources may use substances as a means of mood regulation. However, additional research is needed so we can better understand the role of affect dysregulation in the prediction of smoking behavior. First, previous studies that have examined affect dysregulation as a predictor of smoking have been cross-sectional (e.g., Novak & Clayton, 2001; Wills et al., 2006). There is a need for longitudinal research that examines prospective relations between mood variability and smoking patterns and investigates dynamic, reciprocal relationships between mood variabil-
ity and smoking over time. In line with the self-medication model of substance use, we expected that if mood variability improved or stabilized with increased smoking, such mood improvement might 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 on relations between affect dysregulation and smoking utilized global retrospective self-
report questionnaires. Such reports required adolescents 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 pro-
cedures, such as EMAs (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 EMAs allows for the examination of intraintividual fluctuations across individual mood reports and thus yields a finer grained and more objective index of actual variability in moods. EMAs have been used effectively to study adolescent mood variability (e.g., Larson, Csikszentmihalyi, & Graef, 1980; Larson, Raffaelli, Richards, Ham, & Jewell, 1990; Silk, Steinberg, & Morris, 2003) as well as adult mood variability (Ilie & Judge, 2002; Penner, Shiffman, Paty, & Fritzsche, 1994).

Our goal in the current study was to explore how mood vari-
bility relates to longitudinal smoking patterns in adolescence.
Mood variability is defined as the intraintividual standard devia-
tions of mood assessments across EMA observations. Standard deviations have been used to measure mood variability in the majority of experience sampling/EMA studies (e.g., Eid & Diener, 1999; Hoekema et al., 2004; Larson et al., 1980, 1990; Penner et al., 1994; Silk et al., 2003). This index of mood variability quan-
tifies the tendency of an individual to experience a range of 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 those adolescents who had never smoked, those who had experimented, and those who had progressed to more frequent smoking—to better understand the factors influencing escalation in cigarette use. Specifically, we addressed the following ques-
tions: (a) Do high levels of intraintividual variability in negative moods predict smoking escalation? and (b) 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 youths who progressed to heavier levels of smoking from youths who never progressed beyond experimen-
tation. Second, we expected that increased cigarette use over time would be associated with stabilization of affect regulation (i.e., reduced negative mood variability), as predicted by the self-
medication model of cigarette use (Khantzian, 1997).

Method

Design Overview

Data for this study come from a longitudinal natural history study of adolescent smoking. To assess adolescents, we used a multimethod approach that included 7-day time/event sampling (EMA) via palmtop computers, self-report questionnaires, and in-depth interviews at baseline, 6-month, and 12-month waves.

Participants

The sample for the longitudinal study was recruited through a multistage 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 stu-
dents were notified prior to the screening and parents were given the opportunity to decline their child’s participation. We used survey responses to identify students who were (a) susceptible adolescents who had not yet smoked but who indicated a proba-
bility of future smoking or (b) experimenters who had smoked fewer than 100 cigarettes in their lifetime.

The sampling rationale was based on our desire to follow youths who might initiate smoking and progress in their experimentation with it 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 (a) students being sick/absent for their data collection visit and unable to reschedule (n = 25, 17%); (b) students not bringing parental consent forms to their appointment (n = 12, 8%); or (c) 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, and Flay (2006).

The sample for the current study included 8th- and 10th-grade students who provided EMA data at baseline (N = 517). Due to an 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 or 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/biracial. 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 7 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 present study. Participants received a payment of $40 at the end of each data wave. In addition, self-report questionnaire packets were mailed to the adolescents 2 weeks prior to each data wave. Participants were instructed to bring the completed packet to their EMA training session and were paid $10 upon receipt of each completed packet.

Finally, time line follow-back interviews were conducted with all participants at each wave at their EMA training session. Participants completed a structured in-depth interview with project staff to develop a continuous calendar of smoking behavior over the prior 6 months. Participants were asked to list notable events over the past 6 months (e.g., birthdays, school exams, vacations, and parties), to aid recall, and then were asked to reconstruct their smoking experiences during this time period by noting the amount they had smoked on specific days.

Measures

Demographic information was assessed via questionnaire and included age, grade, gender, 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 time line follow-back interviews conducted with each participant. We used the interview data to create a continuous calendar of each participant’s daily smoking behavior (counts of cigarettes per day), from 6 months prior to baseline through the 12-month wave (referred to as “smoking calendar data”). To improve focus on trends over time, we aggregated the data into a summary smoking rate variable: the number of cigarettes smoked per day for each of the two 90-day intervals within the 6-month interval. We generated the variable by computing the total number of cigarettes smoked in the 90-day period and dividing 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 and 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, one obtains a more accurate measure of smoking activity than is obtained by strict reliance on the questionnaire data.

Smoking history was also assessed via self-report questionnaire at each wave with several items: (a) number of days smoked in the past 30 days, with response categories ranging from 1 (none) to 9 (all 30 days), and (b) 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). On the basis of these data, we generated daily smoking rates 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 in our examination of participant 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 (e.g., “Before the signal, I felt angry”). Subjects responded to mood adjectives using a 10-point Likert-type scale that ranged from 1 (not at all) to 10 (very). The adjectives were selected on the basis of pilot work involving collection of qualitative data (focus groups and in-depth interviews) and quantitative data with 146 students from the 8th and 10th grades who had been recruited from schools similar to those involved in the present study. Confirmatory factor analyses on the current sample revealed a negative affect factor formed by the mood adjectives lonely, embarrassed, sad, angry, and left out, all of which had factor loadings greater than .50 (Cronbach’s α
.73–.78). Other items formed additional mood factors, but we focus here on negative affect.1

We constructed an index of mood variability 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 has supported 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 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, we used a variant of latent growth curve analysis (i.e., trajectory analysis; Nagin, 1999) modified for ordinal responses (Hedeker, 2000) 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, they statistically identify the main systematic characteristics of the continuum. The trajectory analysis identified six primary classes of smoking trajectories. Figure 1 illustrates the patterns over time of the following groups: never smokers, who had never tried a cigarette or at baseline reported having tried a cigarette but who did not smoke during the study \((n = 210, 37\%)\); triers, who engaged in very low levels of experimentation with cigarettes but who 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 Figure 1 shows, smoking patterns diverged over time. Longitudinal smoking patterns, as derived from the trajectory analyses, converged with participants’ daily smoking rates, which we indexed with 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 \((SD = 0.94)\) days in the past 30 days, with an average daily rate of 0.01 \((SD = 0.04)\) cigarettes/day; at 12 months, triers had 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 had 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; at 12 months, they had increased to 10.3 \((SD = 11.0)\) days 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.

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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 is 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).
throughout the study: At baseline, they averaged 11.7 (SD = 11.5) days of smoking and had a daily rate of 2.30 (SD = 4.07) cigarettes. At 12 months, values were similar: Smokers averaged 13.1 days (SD = 12.5) of smoking and had 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 intra-individual standard deviations of momentary mood assessments across EMA observations; this approach has the same, if not fewer, limitations as do alternate measurement approaches (e.g., spectral analysis; see 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 min 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 gender, grade, race/ethnicity, or smoking status, nor for baseline reports of daily negative mood and negative mood variability. Effect sizes (d) ranged from 0.04 to 0.10.

Preliminary Analyses

Descriptive statistics for overall negative mood, mood variability, and daily smoking rates at all waves for the total sample, as stratified by gender, are shown in Table 1. As it shows, 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 were daily rates for boys. Analyses also examined correlations among the mood variables. Negative mood variability was fairly stable over time, with interwave correlations ranging from .50 to .56 (p < .01). Mean negative mood and mood variability were strongly and positively correlated (rs = 0.61–0.67, p < .01).

Table 1

<table>
<thead>
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<th>Wave and measure</th>
<th>Total</th>
<th>Girls</th>
<th>Boys</th>
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<tr>
<td></td>
<td>n</td>
<td>Range</td>
<td>M</td>
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<td>Baseline</td>
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<td></td>
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<td>6 months</td>
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<tr>
<td>Daily smoking rate</td>
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Note. Negative mood was measured by the Negative Affect Scale via ecological momentary assessment (EMA). Mood variability indicates intraindividual standard deviations measured by the Negative Affect Scale (EMA). Daily smoking rate indicates self-reported average number of cigarettes smoked per day for the past 30 days.

*p = .05. **p = .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 adolescents who did not progress in their cigarette use. To test this hypothesis, we conducted a one-way between-subjects analysis of variance 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 differed significantly among the smoking groups, F(3, 517) = 3.20, p = .02, η² 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, participants who escalated in their smoking pattern over time (the escalators and the rapid escalators) had the greatest levels of mood variability at baseline.

To further examine our hypotheses, we conducted three planned comparisons: (a) the rapid escalators versus all other smoking groups; (b) all smoking escalators (escalators and rapid escalators) versus those who tried smoking but never progressed beyond experimentation (triers); and (c) all smoking escalators versus the never smokers. We were specifically interested in the rapid escalators, as their group exhibited the greatest change in smoking over
time, but focused as well on differences between all participants who escalated as compared with those participants who never progressed in their smoking behavior. The rapid escalators had significantly higher levels of negative mood variability at baseline in comparison with 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 than did adolescents who experimented but did not progress in their cigarette use, \( F(1, 102) = 4.80, p = .03 \), or the never smokers, \( F(1, 89) = 10.43, p = .002 \).

It is interesting that, as shown in Figure 2, regular smokers—who at baseline had heavier levels of cigarette use—had lower levels of negative mood variability and appeared more similar to the never smokers. Indeed, a post hoc comparison confirmed that differences between the never smokers and the regular smokers were not significant, \( F(1, 31) = 0.01, \text{ ns} \). 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.

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 we controlled for mean mood, the pattern of results was identical and significance levels remained unchanged. Results of the contrast models are presented in Table 2. The first model (escalators vs. nonescalators) indicated a significant effect of gender, as girls reported greater negative mood variability than did boys overall. Mood variability decreased significantly over time, and escalators reported higher overall levels of mood variability than did nonescalators. Findings also revealed a trend for

\[
\text{Intercept} \quad 1.11 \quad .06 \quad 20.04 \quad <.001 \\
\text{Gender} \quad 0.12 \quad .04 \quad 9.65 \quad .002 \\
\text{Time} \quad -0.09 \quad .03 \quad 20.09 \quad <.001 \\
\text{Smoking group} \quad -0.17 \quad .06 \quad 8.91 \quad .003 \\
\text{Smoking group} \times \text{Time} \quad 0.05 \quad .03 \quad 3.09 \quad .079
\]

Table 2

<table>
<thead>
<tr>
<th>Effect</th>
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<th>( p )</th>
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<td>Time</td>
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<td>Smoking group ( \times \text{Time} )</td>
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<td>.03</td>
<td>3.09</td>
<td>.079</td>
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\[
\text{Intercept} \quad 1.04 \quad .08 \quad 13.62 \quad <.001 \\
\text{Gender} \quad 0.25 \quad .08 \quad 9.58 \quad .003 \\
\text{Time} \quad -0.09 \quad .03 \quad 3.61 \quad .005 \\
\text{Smoking group} \quad -0.13 \quad .12 \quad 1.10 \quad \text{ns} \\
\text{Smoking group} \times \text{Time} \quad 0.06 \quad .07 \quad 0.81 \quad \text{ns}
\]

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

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. NA = negative affect.

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. With the exception of smokers, who had low levels of variability but the highest level of mean negative mood, all groups had similar patterns. 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. MRM 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 can also 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. The first compared adolescents who escalated in cigarette use over time (rapid escalators and escalators, collapsed into one group) with all nonescalators (including the never smokers and the nonescalating triers, collapsed into one group); the second compared all escalators with the regular smokers. These analyses thus examined changes in mood variability for individuals who escalated versus individuals 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 vs. nonescalators or smokers), time (baseline, 6 months, 12 months), and Smoking Group \( \times \) Time on negative mood variability. Given the gender differences in mood variability, gender was included as a control in each model. Participants had to provide EMA data at two or more waves for inclusion in these analyses (\( N_1 = 435 \), yielding 1,214 observations of mood variability; \( N_2 = 118 \), yielding 331 observations).

Results of the contrast models are presented in Table 2. The first model (escalators vs. nonescalators) indicated a significant effect of gender, as girls reported greater negative mood variability than did boys overall. Mood variability decreased significantly over time, and escalators reported higher overall levels of mood variability than did nonescalators. Findings also revealed a trend for
the Smoking Group × Time interaction, which indicated that changes in mood variability over time differed for escalators versus 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, nonescalators had lower mood variability at baseline and remained more stable over time.

Results of the second contrast model (escalators vs. smokers) revealed similar effects for gender and time; neither the effects of smoking group nor the Smoking Group × Time interaction was significant. Examination of Figure 3 suggests that unusual findings for the smokers at the 6-month time point likely influenced this analysis and rendered interpretation difficult. Despite the nonsignificant interaction, findings indicated that, although escalators had higher levels of negative mood variability at baseline than did smokers, these values converged by 12 months (i.e., when escalators were smoking at levels similar to those of smokers, as indicated by the daily smoking rates: 2.01 cigarettes/day for escalators vs. baseline rates of 2.30 cigarettes/day and 12-month rates of 2.62 cigarettes/day for smokers).

Follow-up analyses focused on the rapid escalators as compared with the nonescalators and the smokers. Again, we were specifically interested in the factors that differentiated youths 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 gender (n = 405). Significant effects were found for gender (estimate = 0.11, SE = 0.04), F(1, 320) = 7.22, p < .008; time (estimate = −0.14, SE = 0.04), F(1, 402) = 10.65, p = .001; and smoking group, rapid escalator versus nonescalator (estimate = −0.26, SE = 0.12) and rapid escalator versus smoker (estimate = −0.24, SE = 0.09), 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, which indicated that changes in negative mood variability over time varied as a function of smoking level. Follow-up smoking group contrasts undertaken to specify the source of this effect revealed that the interaction was primarily driven by the Group × Time interaction at the level of rapid escalators versus nonescalators (estimate = 0.10, SE = 0.05), t(320) = 2.22, p = .02. Thus, the rapid escalators experienced significantly greater changes in mood variability over time than did the nonescalators (see Figure 3). However, the rapid escalators versus smokers interaction contrast was also significant at the p < .10 level (estimate = 0.11, SE = 0.07), 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 with mean negative mood used as a covariate. When we controlled 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 the longitudinal patterns of mean negative mood among the smoking groups to investigate the possibility that the observed changes in mood variability over time might 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 gender. The finding that interactions of Smoking Group × Time were not significant indicated that changes in overall mood over time did not vary by smoking group. A significant main effect for time (estimate = −0.045, SE = 0.02), t(402) = −2.24, p < .05, indicated that all adolescents had experienced a reduction in negative mood over time. No other effects were significant when we examined 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. Although much research has documented 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 that influence 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 and 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 adolescents who never progressed beyond low levels of experimentation. In addition, the predictive power of mood variability was distinct from that of overall negative mood, as effects remained significant when we controlled for mean mood levels. Our results are congruent with past research that demonstrated the relevance of affect dysregulation for adolescent substance use (Simons & Carey, 2002; Simons et al., 2004; Wills et al., 2006), and they also corroborate Novak and Clayton’s (2001) cross-sectional findings that deficient emotional regulation predicted transitions from smoking experimentation to daily cigarette use with longitudinal data. Adolescents with greater mood lability may have underdeveloped internal resources for regulation of emotional states and thus may 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 youths with affect regulation vulnerabilities may escalate in their cigarette use as a means of coping with labile moods.

For the escalating youths, 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 with the stable mood profile of the nonsmoking/nonescailing 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 participants with the steepest smoking trajectory, and the regular smokers had levels of mood variability as low as those of 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 for the understanding of youth smoking escalation than is 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, Niura, NIch-ter, & Abrams, 2000). Indeed, our findings for the regular smokers suggest that, independently of improvements in average mood, mood-stabilizing effects may maintain a high level of smoking. 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., by 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 (Khant- zian, 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 that accompany higher levels of smoking among the adolescent escalators and smokers. Findings from adult brain imaging research have indicated that acute and chronic nicotine exposure result in increased dopamine concentration in the ventral striatum/ nucleus accumbens as well as inhibited monoamine oxidase activity in the basal ganglia, both of 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., they may prevent 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 in desensitization of nAChRs (Brody et al., 2006; Quick & Lester, 2002). Evidence suggests that inhibition of nAChRs reduces mood instability (Shytle et al., 2002). 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 and that nicotine exposure increases mood instability among those of stable mood but stabilizes mood in individuals with preexisting mood instability (Shytle et al., 2002).

Withdrawal models of cigarette use offer an alternate explanation of the observed mood variability patterns, which asserts 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; American Psychiatric Association, 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, their withdrawal symptoms may not have peaked and thus their 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, Majeskie, & Fiore, 2004).

The current results argue against a withdrawal model interpretation of mood variability—smoking relationships, although this model is important to consider. 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 constitute part of the complex etiology of regular smoking among youths. 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 youths may be enhancing self-regulation of emotions 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 self-regulation of emotions 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 we used did not directly examine causal relationships between smoking and mood; 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 youths. 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 intradividual 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, 2004; Eisenberg & Spinrad, 2004; Forbes & Dahl, 2005), and we assessed only one component of affect dysregulation. Although research supports the connection between emotion dysregulation and mood lability (Hoekasma et al., 2004; Wills et al., 2006), future research should replicate the current results with a broader measure of affect dysregulation (e.g., one that incorporates 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.

References


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