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Relationship of Self-Reported and Acute Stress to Smoking in Emerging Adult Smokers

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Abstract

Objective—In the current study, we examined subjective and objective measures of stress and their relationship to baseline and future cigarette smoking behaviors over a 1-year follow-up in young adult experimental smokers.

Method—Participants (N = 56) completed two laboratory sessions to determine subjective and objective responses to a controlled laboratory stressor versus a control task. Baseline measures included drug use and smoking histories and a self-report measure of habitual stress (i.e., daily hassles). They were re-contacted 1 year after the laboratory sessions to determine smoking status.

Results—There was wide variability in smoking trajectories, with 34% of participants increasing their smoking over the course of the year. Contrary to predictions, neither daily hassles nor stress reactivity was related to smoking at baseline or change over the year.

Conclusions—These preliminary findings suggest that daily stress or responses to acute social stress are not strong predictors of progression in emerging adult smokers.

Keywords

emerging adults; experimental light smokers; stress; cortisol; smoking trajectory

Emerging adults, aged 18 to 25 years (Arnett, 2004; Riggs, Chou, Li, & Pentz, 2007), have the highest rates of tobacco smoking of any age group in the United States (Substance Abuse and Mental Health Services Administration [SAMHSA], 2010), and exhibit steady increases in smoking behavior and nicotine dependence during this developmental period (Chassin, Presson, Rose, & Sherman, 1996; Jackson, Sher, & Wood, 2000). Understanding risk factors for cigarette use and smoking progression during the vulnerable transitional period from initial experimentation to heavier smoking could lead to more effective prevention and early intervention efforts.

Young adulthood is also associated with new stressors related to changing life roles and responsibilities that may affect smoking. Chronic smokers smoke more when experiencing negative emotions (Kassel, Paronis, & Stroud, 2003; McKennell, 1970; Shiffman & Waters, 2004) and they report that smoking alleviates negative mood (Brandon & Baker, 1991; Copeland, Brandon, & Quinn, 1995; Kassel et al., 2003). Adolescent experimental smokers report similar motivations for smoking, and in laboratory tests smoking actually reduces

negative affect in these individuals (Kassel et al., 2007; Wang, Fitzhugh, Eddy, & Westerfield, 1996). Thus, stress may be a risk factor for initiation and maintenance of smoking during these early stages. Prospective investigations are critical to infer causal conclusions about the relationship between stress and smoking (Kassel et al., 2003).

Several studies have examined whether individual differences in stress response predict smoking among occasional smokers, but results have been mixed. Among college-age experimental smokers, Magid and colleagues (Magid, Colder, Stroud, Nichter, & Nichter, 2009) found that self-reported negative affect, but not objective accounts of stressful events, was associated with smoking. In an earlier prospective self-report study of young adult occasional smokers, Wetter and colleagues (Wetter et al., 2004) found that expecting smoking to relieve negative affect predicted the transition from occasional to regular smoking better than actual experience of negative affect or stress. Finally, in a preliminary prospective study in which occasional smokers were exposed to an acute social stress procedure, we found that higher cortisol responses to the stressor predicted increases in smoking over a 6-month follow-up period (de Wit, Vicini, Childs, Sayla, & Terner, 2007). Overall, these studies have used a variety of measures to conceptualize “stress,” making it difficult to integrate the findings. To our knowledge, however, no study has systematically employed both self-report and objective methods to examine the role of stress in a prospective design with emerging adults who are not yet habitual smokers.

The goal of the current study was to examine whether responses to a valid laboratory stressor, the Trier Social Stress Test (TSST), or self-reported ratings of daily stress and hassles predicted changes in smoking over 1 year, among occasional smokers. We also examined whether individual differences in stress were associated with levels of smoking at baseline.

Method

Participants

Participants were recruited via posters on nearby college campuses, newspaper ads (e.g., The Chicago Reader), and online postings (e.g., Craigslist). Initial screening criteria were as follows: 18–25 years of age, smoking at least one cigarette in the past month but not more than 20 per week, body mass index between 18–29 kg/m², a high school education (to ensure comprehension of the consent process and study questionnaires), and English fluency. At the screening session, eligible participants underwent a modified Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV; SCID-IV; First, Spitzer, Gibbon, & Williams, 1996) with a senior graduate student in clinical psychology, a physical examination by a study nurse, and an electrocardiogram. The study physician reviewed the results of the physical exam along with the self-reported health history to determine medical eligibility. Exclusion criteria were as follows: serious medical conditions contraindicating the stress procedure, use of prescription medications, and current or past year Axis I psychiatric disorder (APA, 2000). For women, these criteria also included taking hormonal contraceptives, as hormonal status influences responses to stress (Childs, Dlugos, & De Wit, 2010; Kajantie & Phillips, 2006), or current

or planned pregnancy. All participants provided informed consent and the study was approved by the University of Chicago Institutional Review Board.

Procedure

Participants first attended an orientation visit during which they received an explanation of the study, provided informed consent, and were given instructions to abstain from alcohol and recreational drugs for 24 hours and smoking and eating for 2 hours prior to each session. During the orientation visit they also completed the Adult Hassle Scale of Daily Stressors (Maybery, Neale, Arentz, & Jones-Ellis, 2007), which measures the frequency of 46 life stressors (e.g., "Disagreement with a friend") on a 5-point Likert scale ranging from 1 (*did not happen*) to 5 (*10+ times in the last month*). This scale was developed to focus on the experience of these types of small daily hassles, as they may be more predictive of negative psychological outcomes than major negative life events (Kanner, Coyne, Schaeffer, & Lazarus, 1981). The dependent variable used in this study was a composite variable, Total Hassle Frequency, which summed scores across all types of daily hassles ($\alpha = 0.80$).

Participants also completed surveys assessing smoking history, smoking exposure, total number of cigarettes smoked in the last 30 days, average number of cigarettes smoked per day in the last 30 days, and nicotine dependence, as measured by the Fagerström Test of Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, 1991). The survey also included items assessing the frequency and quantity of alcohol, marijuana, and other recreational substance use. Participants completed the same survey 12 months after completing laboratory sessions (described below) to assess changes over time.

After the orientation, participants attended two randomized experimental sessions (stress and no stress) in comfortably furnished rooms in the Human Behavioral Pharmacology Laboratory at the University of Chicago. These sessions were separated by at least 48 hours and, on average, were completed 7.5 days apart. Their compliance with drug abstinence instructions was confirmed using urine and breath samples (ToxCup, Branam Medical Corporation, Irvine, CA, Alcosensor III, Intoximeters Inc., St. Louis, MO). Women were also screened for pregnancy (AimStrip, Germaine Laboratories, San Antonio, TX).

After completing baseline subjective, cardiovascular, and saliva measures, participants were given instructions for either the TSST (Kirschbaum, Pirke, & Hellhammer, 1993), which was used as a psychosocial stressor, or control task, as per their initial randomized assignment. In the TSST, participants were told that they had 10 minutes to prepare a 5-minute mock job interview speech to be performed in front of two interviewers and a video camera. They were then escorted to a separate room to perform the speech and a 5-minute mental arithmetic task in front of an unsupportive audience. For the control task, participants were given 10 minutes to prepare to discuss their favorite book, movie, or television program with the research assistant for 5 minutes. They were moved to a different room to do this, where they also played a card game (solitaire) for another 5 minutes, without video cameras or observers.

Following the task, participants returned to the original room, where they immediately completed subjective, cardiovascular and saliva measures. Participants subsequently

completed subjective measurements at 10, 30, 60, and 90 minutes after the end of the task and completed cardiovascular and saliva measures at 10, 20, 30, 60, and 90 minutes after the end of the task. Participants could watch television and movies or read magazines when measures were not being obtained. At the end of the experimental sessions, participants were debriefed, instructed on follow-up procedures, and paid \$100.

Subjective emotional response to the laboratory stressor was measured using the Profile of Mood States (POMS; McNair et al., 1971). Based on previous data suggesting the TSST nonspecifically elevates several scales on the POMS (Childs & de Wit, 2009; de Wit et al., 2007; Hamidovic et al., 2010), we constructed a Negative Emotion composite scale by summing POMS Anxiety, Depression, Anger, and Confusion scales ($\alpha = 0.77$). Blood pressure was measured using a portable monitor (DINAMAP[®] ProCare 100 Vital Signs Monitor, GE Healthcare, Tampa, FL). Measurements of systolic and diastolic blood pressure (SBP and DBP) were used to calculate mean arterial pressure (MAP) using the formula $MAP = (2 \times DBP + SBP) / 3$. Cortisol was measured using saliva samples collected with Salivette[®] cotton wads (Sarstedt Inc. Newton, NC). The GCRC Core Laboratory at The University of Chicago determined levels of cortisol in saliva (Salimetrics LLC, sensitivity = 0.003ug/dL).

Data Analysis Strategy

To compare TSST and control sessions on stress responsivity over time, we conducted a series of repeated measures analyses of variance (ANOVAs) for each laboratory measure. Then, for both the stress and no stress sessions, we created a change score for each time point by subtracting baseline scores from those at subsequent time points. Finally, we created a single session score for each measure by calculating the area under the curve (AUC) for these change scores. Subtracting the AUC for the no stress session from that for the stress session captured summary differences in reactivity to the TSST compared with the control task. This difference in AUC serves as the primary dependent variable for each measure in subsequent analyses.

Pearson correlations were conducted to examine the relationships among the summary scores for laboratory measures (subjective mood, cardiovascular, and cortisol response) and self-report daily hassles at baseline (Total Hassle Frequency). Next, we descriptively characterized smoking patterns at baseline and over the 1-year follow-up in the current sample. Finally, we examined the association between stress measures and smoking variables at baseline and at 12-month follow-up via Pearson's correlations. We also conducted independent sample *t* tests to explore whether stress response differentiated the individuals who showed an increase in cigarette smoking over time compared with those who did not; eta-squared values are included to denote the effect size for each analysis.

Results

Demographics

Sixty-two participants enrolled in the study, but six withdrew because of scheduling conflicts (three withdrew after the acute stressor session and three withdrew after the control

task session). Therefore, the final sample was $N = 56$. There were no differences in demographic or smoking behaviors between participants who completed both sessions and those who did not. As shown in Table 1, most participants were male (59%), European American (70%), full-time students (82%), with a mean age of 20.8 ($SD = 2.2$). None of these demographic variables were related to smoking behaviors at baseline or at follow-up; however, alcohol use at baseline did correlate with smoking at baseline, $r = 0.36$, $p = 0.007$. With regard to smoking trajectory, higher age at baseline predicted a smaller increase in smoking at 12-month follow-up, $r = 0.29$, $p = 0.04$. We examined age and baseline alcohol use as potential covariates and found that controlling for these factors did not change our pattern of results. Therefore, they are not included in the presented analyses.

Participants reported their first cigarette use at 18.0 years ($SD = 2.1$), and the mean time from their first cigarette to study participation was 2.7 years ($SD = 2.3$). Household and social exposure to smoking was moderately high, with 35% (18/52) reporting that at least one other person in their household was a smoker and 63% (33/52) reporting that some or all of their friends smoked. No participants at baseline scored higher than 2 on the FTND (i.e., very low to no nicotine dependence).

Laboratory and Self-Reported Stress Measures

Variables were screened for normality, univariate, and multivariate outliers. The TSST task (vs. control task) effectively elicited acute increases in POMS negative emotion, $F(5, 270) = 22.75$, $p < .001$, $\eta_p^2 = .34$, MAP, $F(6, 330) = 22.32$, $p < .001$, $\eta_p^2 = .29$, and salivary cortisol, $F(6, 318) = 14.07$, $p < .001$, $\eta_p^2 = .21$, as seen in previous studies (de Wit et al., 2007). This was supported by AUC scores, which showed larger increases in the TSST versus control task for POMS negative emotion, $t(54) = 4.04$, $p < .001$, $d = .54$; MAP, $t(55) = 5.20$, $p < .001$, $d = .69$; and cortisol, $t(53) = 2.91$, $p = .005$, $d = .40$. Sample means for these summary stress variables as well as self-reported daily hassles are shown in Table 2. Subjective and objective measures of acute reactivity to stress were generally moderately positively correlated, though self-reported daily hassles did not significantly correlate with any of the laboratory stress measures.

Smoking Patterns and Follow-Up Measures

Follow-up retention at 12 months was high, with 95% (53/56) of participants completing the follow-up online surveys. At baseline, participants smoked a mean of 31.1 cigarettes per month (median = 17.5). This variable was log-transformed in all subsequent analyses due to right skewness. Over the course of the year-long study, there was considerable variability in smoking behavior over time: 21% (11/53) increased smoking by 20+ cigarettes monthly, 13% (7/53) increased smoking by 1–19 cigarettes monthly, 6% (3/53) did not change monthly smoking, 26% (14/53) decreased smoking by 1–19 cigarettes monthly, 15% (8/53) decreased smoking by 20+ cigarettes monthly, and 19% (10/53) reported no smoking at all in the last month. Further, 11% (i.e., six individuals) scored 3 or above (mild to moderate nicotine dependence) on the FTND at follow-up. Finally, hassle scores at baseline and 12-month follow-up were correlated, $r = 0.58$, $p < 0.001$, so stress level was moderately but not entirely stable across the follow-up period.

Stress and Smoking

As described in the statistical analyses section, we examined both baseline smoking and smoking increases at 12 months (as an indicator of smoking progression) in relation to stress measures taken at baseline. Neither acute stress responsivity nor self-reported daily hassles were associated with number of cigarettes smoked per month at baseline (see Table 2). Further, these baseline measures of stress were unrelated to changes in smoking one year later (Table 2). Measures of stress also did not differentiate individuals who increased smoking behavior ($n = 18$) from those who did not ($n = 35$): POMS negative emotion, $t(51) = 0.16$; MAP, $t(51) = 0.88$; cortisol, $t(50) = 0.12$; Hassle Scale, $t(47) = 0.28$.

Discussion

In the current study, there was considerable individual variability in the patterns of change in smoking over the 1-year follow-up period. Approximately one third of the participants increased their smoking during the 1-year follow-up (34%), whereas a few continued to smoke at the same light levels (6%), and others decreased smoking (41%) or stopped completely (19%). However, this variability was not related to measures of stress reactivity: neither the laboratory-derived measures of stress reactivity nor self-reported daily hassles were associated with change in smoking over time.

Based on the literature, the occasional smokers in our study were at risk for acceleration of smoking, as smoking behavior generally increases during young adulthood (Chassin et al., 1996). Although they started smoking slightly later than average smokers (i.e., 18 years of age; SAMHSA, 2010), they were within the typical period for acceleration from first use of cigarettes to regular daily smoking (i.e., about 2 years), and they reported significant environmental smoking exposure by their immediate friends and roommates. However, the individual variability in smoking patterns seen here was consistent with another large-scale study of college-age occasional smokers, in which 14% progressed to daily use after 4 years, 35% remained occasional smokers, and 51% quit smoking (Wetter et al., 2004). Therefore, a method of identifying a priori young smokers most likely to increase their smoking over time would be valuable for prevention or early intervention efforts. The results from our study indicate that neither self-reported daily stress nor psychological and physiological responses to acute stress are useful predictors of smoking progression.

The study had both strengths and limitations. The strengths included a relatively homogeneous sample of young adult, experimental, light smokers, a validated and effective stress induction procedure, and high 1-year follow-up rates. We also examined stress in two distinct ways. First, we used a laboratory-based acute stress procedure that is analogous in some ways (controlled nature of the stressor, elicitation of glucocorticoid responses) to the acute stress procedures used to induce drug self-administration in animal models (Marinelli & Piazza, 2002; Piazza & Le Moal, 1996). Second, we used a daily hassles measure that relates to health outcomes in clinical settings (Romano, Bloom, & Syme, 1991). The primary limitation was that sample size was modest and so could only detect effects of medium size or larger ($r = 0.32$); future studies would need larger samples to detect smaller effects.

A second limitation was that our follow-up period was only one year and focused mainly on self-reports of smoking behavior, though retrospective reporting of cigarette use has generally proven valid (Brigham et al., 2010; Wong, Shield, Leatherdale, Malaisson, & Hammond, 2012). Although smoking increased in a sizeable minority of participants, relatively few progressed to dependence, as measured by the FTND. Thus, longer follow-up periods may be needed to capture the development of heavy smoking behavior and/or nicotine dependence. It would also be valuable to obtain more details about use of other drugs, including alcohol, in relation to stress reactivity, as smoking patterns in college-age and young adult occasional smokers indicate that most smoking takes place on the weekend and is associated with alcohol and other substance use (Colder et al., 2006; Harrison & McKee, 2008; King, Epstein, Conrad, McNamara, & Cao, 2008; Shiffman et al., 2002; Shiffman, Kirchner, Ferguson, & Scharf, 2009). Finally, though mood and anxiety disorders are often comorbid with substance use, we recruited a relatively healthy sample. Therefore, any conclusions about stress effects on smoking among those with psychiatric disorders or symptoms were outside the scope of the present study. Future research could make note of clinical symptoms at baseline and follow-up and examine any relationships between mood disorders and smoking behavior over time.

In conclusion, in a controlled, laboratory-based prospective study, neither self-reported levels of stress nor reactivity to an acute social stress procedure related to either baseline smoking or progression in smoking over time. Thus, although stress has been strongly associated with dependent smoking and relapse, it may not be a primary motivator of smoking or smoking progression in young adult occasional smokers. These findings are consistent with other studies indicating that self-reported stress does not relate to either single incidents of smoking or smoking progression in large samples of college-age occasional smokers (Magid et al., 2009; Wetter et al., 2004). In contrast to these negative findings, expecting that smoking will help regulate negative mood does appear to predict smoking progression (Wetter et al., 2004). It may be necessary to measure stress and smoking expectancies in tandem to elucidate the relationship between smoking and stress. Further, it remains an important empirical question whether young adults with anxiety or depression may initially smoke or increase smoking over time in response to stress, unlike our current healthy sample. Increasing our understanding of such risk factors in young samples remains critically important to prevent continued harmful smoking behaviors and development of nicotine dependence.

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Table 1
Sample Demographics

	N (% of sample) or Mean (SD, range)
<u>Demographics</u>	
Sex (male)	33 (59%)
Race (Caucasian)	39 (70%)
Ethnicity (non-Hispanic)	51 (91%)
Education	44 (78%) Some college
	11 (20%) Bachelor's degree
	1 (2%) Some graduate school
Student status (current students)	46 (82%)
Body mass index (kg/m ²)	23.0 (2.6, range = 19.0–28.7)
Age (year)	20.8 (2.2, range = 18–25)
Alcoholic drinks per week	10.0 (10.0, range = 0–40)
<u>Recreational drug use (10× Lifetime use)</u>	
Cannabis	36 (47%)
Tranquilizers	5 (9%)
Stimulants	5 (9%)
Opiates	0 (0%)
Hallucinogens	2 (4%)
Inhalants	5 (9%)

Note. SD = standard deviation.

Table 2

Stress Variable Means, Correlations, and t-test Results

	Means (SD)		Correlations ^b					
	No stress ^a	Stress ^a	POMS negative emotion	MAP	Cortisol	Hassle Scale	Baseline smoking	Peak smoking
POMS Negative Emotion ^c	-5.0 (7.8)	3.6 (11.6) ***	—	0.09	0.30 *	.15	0.12	-0.07
MAP (mmHg)	-35.8 (54.7)	16.5 (70.3) ***		—	0.35 **	.20	0.11	-0.05
Cortisol (µg/dl)	-0.4 (0.7)	0.1 (0.1) ***			—	.01	0.06	0.04
<u>Self-Report</u>								
Hassle Scale	18.7 (10.5)		0.15	0.20	0.01	—	0.02	-0.22

Note. SD = standard deviation; POMS = Profile of Mood States; MAP = mean arterial pressure.

^aLaboratory session means are reported as area under the curve (AUC) relative to baseline.

^bStress reactivity data were unavailable on some measures for a few participants: insufficient sample quantity or quality for cortisol (n = 2) and computer error on the hassle questionnaires (n = 4). These participants are thus omitted from correlations and *t* tests involving these variables.

^cPOMS Negative Emotion = Sum of Anxiety, Depression, Anger, and Confusion subscales.

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$.