

Smoking is Associated with Worse Mood on Stressful Days: Results from a National Diary Study

Keith R. Aronson, Ph.D. · David M. Almeida, Ph.D. ·
Robert S. Stawski, Ph.D. · Laura Cousino Klein, Ph.D. ·
Lynn T. Kozlowski, Ph.D.

Published online: 6 December 2008
© The Society of Behavioral Medicine 2008

Abstract

Background Many smokers report smoking because it helps them modulate their negative affect (NA). The stress induction model of smoking suggests, however, that smoking causes stress and concomitant NA. Empirical support for the stress induction model has primarily derived from retrospective reports and experimental manipulations with non-representative samples of smokers. Moreover, prior studies have typically not considered contextual factors (e.g., daily stressors) that may impact the smoking–NA relationship.

Purpose The aim of this study was to assess the stress induction model of smoking using a prospective design in a nationally representative sample of smokers while simultaneously examining the impact of daily stressors on the relationship between smoking and NA. We hypothesized that smoking and NA would be positively related, and this relationship would be intensified by exposure to daily stressors.

Methods A national sample of middle-aged smokers ($N=256$) were called on eight consecutive evenings to assess stressor exposure and intensity. Participants also reported on their daily NA and indicated the number of cigarettes they smoked. Analyses were conducted using hierarchical linear modeling

to determine the relationship between daily smoking, NA, and stress.

Results Smoking more than usual was associated with increased NA on days when respondents were exposed to any stressors. Smoking more than usual had no effect on NA on days when no stressors were encountered. Moreover, the moderating effect of stressor exposure remained significant even after controlling for the number and intensity of daily stressors reported.

Conclusions While smokers report that smoking alleviates their NA, our study suggests that the exact opposite may occur, particularly on stressful days. When smokers smoke more than usual on days when the encounter stress, they are likely to feel emotionally worse off.

Keywords Smoking · Smoker · Negative effect · NSDE · Stress

Introduction

Despite the well-documented public health threat and tremendous economic costs associated with cigarette smoking [1], approximately 21% of the US population continues to smoke [2]. Smokers often report that they continue to smoke because it helps them regulate their affective states [3, 4]. In particular, virtually all smokers report that when confronted with stressors and emotional upset, smoking helps reduce negative affect (NA) [3–9]. NA is a general dimension of subjective distress and displeasure in engagement that subsumes various negative affective states such as anger, contempt, disgust, fear, guilt, sadness, and anxiousness [10]. Indeed, smokers have strong expectancies that cigarettes will mitigate aversive affective states and provide anxiolytic effects [11, 12]. These

K. R. Aronson · D. M. Almeida · R. S. Stawski · L. C. Klein
The Pennsylvania State University,
University Park, PA 16802, USA

L. T. Kozlowski
State University of New York,
Buffalo, NY, USA

K. R. Aronson (✉)
Social Science Research Institute,
The Pennsylvania State University,
University Park, PA 16802, USA
e-mail: kra105@psu.edu

findings are consistent with the stress-coping [13] and self-medication [14] models of substance abuse which suggest that drugs are used to maintain emotional equilibrium. However, while smokers believe that smoking helps alleviate NA, some theory and research suggests otherwise.

Parrott and colleagues [15–19] have posited the stress induction model of smoking, which suggests that smokers experience acute nicotine deprivation during the period between one cigarette and the next. Nicotine deprivation results in abstinence symptoms including negative affective states (e.g., anxiety, tension, anger). As abstinence symptoms and NA increase, smoking is once again initiated. The initiation of smoking replenishes nicotine levels, thus, reversing and alleviating the NA associated with the deprivation [16]. Smokers repeat this deprivation-reversal cycle throughout the day, thereby experiencing emotional downs followed by return to affective baseline [20].

The stress induction model has been used to explain paradoxical aspects of the smoking–NA relationship. For example, while smokers report that smoking relaxes them, they also report higher rates of stress than nonsmokers. Parrott has shown, for example, that when smokers provide self ratings of NA before and after each cigarette, they demonstrate repetitive mood fluctuations over the course of the day. Specifically, he found that smokers experienced greater than average NA in between cigarettes with a brief declination after smoke inhalation [21]. Moreover, comparisons with nonsmokers indicated that smokers did not gain a mood advantage from smoking but instead experienced repeated abstinence symptoms. Therefore, smokers report both NA and relief from NA as they smoke. The model also proves useful, although not conclusive, in understanding why stress levels decline after smokers quit. Specifically, Parrott has suggested that with complete and prolonged abstinence, smokers no longer experience the distressing cycle of withdrawal, NA, and reversal [20].

On the other hand, research on the stress induction model has yielded inconsistent results and been criticized on several fronts [22–24]. Most germane to this study is the fact that the model uses the stress and NA constructs interchangeably [24] when, in fact, they are quite different [25]. This lack of differentiation is surprising given Parrott's [26] own recognition that "abstinence symptoms and post-cigarette relief are closely related to the environmental circumstances" (p. 1159). Second, a number of studies have found that smoking occurs in the absence of withdrawal [27, 28] and can be motivated by low arousal states such as boredom [29, 30]. Third, while Parrott and other researchers suggest that smoking and NA are related to acute nicotine withdrawal and reversal [31–33], it is important to note that nicotine regulation only controls smoking within broad bounds, allowing for other environmental or contextual factors to influence smoking [30, 34,

35]. Unfortunately, environmental factors such as exposure to stressors have not been adequately studied within the context of the stress induction model.

Stressors in Smoking Research

In the smoking literature, there is great variability in how stressors are conceptualized and measured (e.g., exposure to an aversive stimuli, engagement in a stressful task such as public speaking, endorsement of negative major life events). However, such definitions ignore the minor yet more frequently occurring stressors of life [36]. Therefore, we operationalize stressors as the routine challenges of day-to-day living, such as the everyday concerns of work, caring for other people, commuting between work and home, and other more unexpected small occurrences that disrupt daily life [37]. Exposure to these kinds of everyday commonplace events, or "quotidian" stressors [38] are strong predictors of psychological well being [36, 39–48].

To understand how one is affected by these everyday stressors, both objective characteristics of the stressor (e.g., frequency) and the individuals' subjective appraisal of the stressor (i.e., perceived stress) must be examined. Perceived stress refers to the meaning that individuals give to daily stressor occurrences in terms of how bothersome and disruptive they are [37]. Both objective and subjective components of daily stressors affect daily well-being [49]; thus, both should be measured in stress studies.

The distinction between frequency of stressor exposure and perceived stress is important because some theories of stress suggest that mere exposure to stressor events (e.g., daily hassles) requires the organism to adapt and change, which ultimately leads to disequilibrium [50, 51]. On the other hand, some theorists suggest that how stressors are perceived and subjectively evaluated determines whether an event will be experienced as stressful [25, 52]. Both the stressor frequency and perceived stress notions have received empirical support. Therefore, in this study, we examine stressors from both perspectives.

Smoking, Stressors, and NA

While the stress induction model suggests that acute nicotine withdrawal causes NA, the evidence is quite mixed regarding this assertion. In a number of cross-sectional studies, smoking is positively related to various manifestations of NA, most typically depression and anxiety [53]. On the other hand, longitudinal studies have produced inconsistent results [3]. Laboratory studies have also yielded inconsistent results. For example, some studies have manipulated affective states (e.g., induced NA) and then observed specific smoking behaviors such as rate, puff duration, and number of puffs [54–56]. These studies,

however, have typically not found an association between smoking and NA, and when found, they tend to be ephemeral [57] or occur inconsistently [58–61]. We hypothesize that one of the reasons for the variable findings from experimental work is that it fails to account for the kinds of “real world” stressors (e.g., problems at work, disagreements with one’s spouse, role overload) that smokers frequently mention as central to their smoking behavior [8]. In other words, smoking and NA may largely be related within the context of these daily stressors. Indeed, one study found that smoking lapses were associated with NA, and this relationship was moderated by environmental stressors which accounted for variance in relapses above and beyond that accounted for by NA alone [62].

Because the stress induction model has not clearly delineated between stressors and NA, it is important to examine the impact of stressors on the relationship between smoking and NA. First, a number of studies have found a positive relationship between various stressors and smoking in adults [63–65]. For example, work demands [66–68], social stressors [69–71], and more generalized stressors [63] are associated with increases in self-reported smoking. On a population level, smokers exposed to more stressors tend to smoke more [72]. Experimental studies have further demonstrated that stressors increase smoking behaviors such as puff rate, volume of smoke inhaled [70, 73–75], nicotine intake [32], and desire to smoke [74]. Not surprisingly, daily stressors are also a reliable positive predictor of NA [36, 76–79]. Because exposure to stressors is related to both smoking and NA, such exposure may be an important moderator of the smoking–NA relationship. Such a notion needs to be more precisely considered within the stress induction model.

Unfortunately, only a few studies have modeled the relationship between smoking, stressor exposure, and NA simultaneously [54, 56], and we could not locate one published prospective study examining these relationships using a nationally representative sample. The studies conducted to date have found that the relationship between smoking and NA remains unchanged in the face of stressors. These studies, however, have several limitations [3, 23, 80]. Most studies have used laboratory tasks which expose participants to a specific contrived stressor (e.g., exposure to an unexpected noise, engagement in a challenging task). Laboratory tasks such as these may lack ecological validity because they do not capture the impact of real world stressors that often occur on a daily basis. Studies to date have also typically been cross-sectional in design, thus, allowing for only between-subject comparisons.

We propose that real world stressors are more likely than laboratory stressors to positively moderate the relationship between smoking and NA. There are three reasons for this. First, nicotine appears to increase sympathetic responsive-

ness to stressors, including increased cortisol, blood pressure, and heart rate [32, 81–84]. Furthermore, exposure to stressors in the absence of nicotine also increases sympathetic arousal. Therefore, stressor exposure may have an additive effect on sympathetic arousal. Indicators of sympathetic arousal (e.g., increased blood pressure and heart rate, heightened vigilance, feelings of tension) are often experienced as distressing and are, themselves, associated with NA [85–88]. Second, as daily real world stressors accumulate, the utility and effectiveness of cigarette smoking as a means of “escaping” from stressful cognitions may decrease. Indeed, several studies have shown that smoking narrows the smoker’s attention to external stressors and, therefore, can act as an anxiolytic agent [60, 61]. However, this effect has been demonstrated only in laboratory tasks. Third, Parrott describes that the nicotine depletion–replenishment cycle that smokers experience throughout the day is, in itself, stressful [15, 16, 19, 26]. Because individuals possess finite internal and external resources to effectively manage their stressors, exposure to additional stressors during the day serves to further challenge the individual’s resources [25, 52]. Thus, cycling smokers exposed to additional stressors may be more likely to tax their resources and experience emotional upset.

Taken as a whole, these findings suggest that exposure to daily stressors may exacerbate any preexisting relationship between smoking and NA. Therefore, we contend that in the face of daily stressors or perceived stress, smoking may result in smokers feeling emotionally worse off.

The Current Study

To more fully explicate the smoking–NA relationship, a number of theorists and researchers have indicated that ecologically valid prospective studies with representative samples and well-validated measures are critically needed [3, 89]. The current study will be the first to examine the relationship between smoking, stressor exposure, and NA using a national sample of adults using well-validated measures. Moreover, this is the first daily diary study to test the hypothesis that stressors positively moderate the relationship between smoking and NA. The prospective design, nationally representative sample, and measurement of objective and subjective daily stressors address a number of the weaknesses in the extant literature.

Methods

Participants

This study is a secondary data analysis from the National Study of Daily Experiences (NSDE) [36]. We received IRB

approval from The Pennsylvania State University prior to conducting this study. Respondents were 1,031 adults (562 women, 469 men), all who had previously participated in the National Survey of Midlife Development in the United States, a national representative telephone–mail survey of 3,032 people, aged 25–74 years. Respondents in the NSDE were randomly selected from the Midlife in the United States (MIDUS) survey and received \$20 for their participation. Of the 1,242 MIDUS respondents that were contacted, 1,031 agreed to participate, yielding a response rate of 83%. Of most relevance to this proposal are the 256 respondents who reported being smokers (female=131, male=125). Demographic information is provided in Table 1.

Measures

Daily negative affect was measured using the six-item Negative Affect Scale of the Nonspecific Psychological Distress Scale [90] which was designed specifically for the MIDUS. The scale was developed from the following well-known and valid instruments: the Affect Balance Scale [91], the University of Michigan's Composite International Diagnostic Interview [92], the Manifest Anxiety Scale [93], and the Centers for Epidemiological Studies Depression Scale [94]. The scale was developed using item response models and factor analysis, yielding a single factor structure representing current general psychological distress. The measure was validated in eight administrations using samples from different populations and has demonstrated good reliability and validity in prior research [90]. Respondents were asked how much of the time today did they feel: worthless; hopeless; nervous; restless or fidgety; that everything was an effort; and so sad that nothing could cheer you up. Respondents rated their response on a 5-point

scale *from none of the time to all of the time*. It is important to note, that measuring negative affect in terms of frequency is in keeping with theory and research that NA is better characterized by its duration than its intensity [95, 96]. Scores across the six items were summed for each day and the scale was internally consistent ($\alpha=.89$).

Daily Stressor Occurrence

Daily stressor occurrence was assessed through the semi-structured Daily Inventory of Stressful Experiences (DISE) [36]. The DISE consists of a series of stem questions asking whether specific types of daily stressors had occurred in the past 24 h and a set of interviewer guidelines for probing affirmative responses. Participants were asked about the occurrence of seven specific stressors: an argument or disagreement with someone; a time where you engaged in a disagreement but decided to let it pass; something happened at work that most people would consider stressful; something happened at home that most people would consider stressful; an experience of discrimination; something happened to a close friend that was upsetting to you; or anything else not previously mentioned. Notably, these events do not represent major life events but instead the minor annoyances of daily life. These seven broad stressor domains were those most frequently mentioned in a pilot study of 1,006 adults [36].

In order to examine these narratives, interviews were tape recorded and transcribed. For each stressor described, trained graduate and advanced undergraduate expert coders rated (a) content classification, (b) focus of who was involved in the event, (c) dimensions of threat, and (d) severity of stress [36]. Inter-rater reliability across the DISE codes ranges from 71% to 95% [97]. In addition, respondents provided subjective assessment of (e) degree of severity and (f) appraisal of areas of life at risk because of the stressor.

The interview-based approach allows one to distinguish between the occurrence of a stressor (e.g., conflict with spouse) and the affective response to the stressor (e.g., crying or feeling sad). Another benefit of this approach is its ability to identify overlapping reports of stressors [98]. In the present study, approximately 5% of the reported stressors were discarded because they were either solely affective responses or they were identical to stressors that were previously described on that day.

The validity of the DISE has been demonstrated in a number of studies. For example, various aspects of daily stressors measured by the DISE are significantly associated with negative mood and physical health symptoms [36], two commonly used outcomes in research on the relationship between daily stressors and health. Stressor level as measured by the DISE is associated with decline in memory function [99], increased family tension [100, 101],

Table 1 Sample descriptives

Age (in years)	M=44 SD=13 Range=25–73
Gender	Male=49.8% Female=50.2%
Race	White=87.3% Black/African American=4.1% Native American=.8% Asian=.4% Other/mixed=7.4%
Education	Less than high school diploma or GED=12.5% High school diploma or GED=33.7% Some college=37.3% College degree or more=16.5%
Household annual income	M=\$45,000 SD=\$39,000 Range=\$0–\$300,000

and decreased marital satisfaction [102]. Socioeconomic status is negatively associated with stressor level as measured by the DISE [43, 103]. The DISE is also sensitive to stressor effects based on age [104–107] and genetic endowment [108]. The validity of the DISE should not be surprising since personal interviews regarding stress improve the assessment of perceived stress (real stories are elicited), provide more precise classification of stressor content, and provide more valid differentiation between severity and stressor appraisal [109].

Stressor Day

For each daily interview, individuals who responded affirmatively to any of the stem questions received a value of 1 on an indicator variable of any stress and were coded 0 otherwise. Respondents' narrative responses to investigator probes provided objective information on the content of the stressful experiences as well as the meaning of the stressor for the respondent.

Perceived Stress

Perceived stress was measured by asking respondents, "How stressful was this [particular stressor] for you—very, somewhat, not very, or not at all?" The average score across all reported stressors throughout the 8-day interview was used in the analyses.

Stressor Frequency

Respondents completed interviews each evening of the 8-day protocol. Because people varied in the number of days they participated in the study, total number of stressors across the 8 days was divided by the number of recorded days. Thus, the total score represented an average stressor frequency across all participant days (e.g., a score of 3 stressors across 8 days yielded a score of .375). At the end of the study, individuals were asked how typical were the number of stressors they had experienced throughout the week. The majority (62%) rated the week as typical to their usual experience, with the remaining respondents equally distributed between more frequent and less frequent than usual.

Smoking behavior was assessed by asking "How many cigarettes did you smoke today?" On average, participants reported smoking 17.3 cigarettes per day. The number of cigarettes smoked in this sample is comparable to smoking rates for adults reported by the Substance Abuse and Mental Health Services Administration, which recently reported that, on average, adult smokers report smoking 15 cigarettes per day [110]. The between person standard deviation was 11.44, while the within person standard

deviation was 4.43, indicating that there were far more individual differences in cigarette consumption across individuals than for any given individual across the eight study days.

Procedures

Over the course of eight consecutive evenings, respondents completed short telephone interviews about their daily experiences. Data collection spanned an entire year (March 1996 to April 1997) and consisted of separate "flights" of interviews, with each flight representing the 8-day sequence of interviews from each of the participants. Participants completed an average of seven of the eight interviews.

Data Analytic Plan

To examine the relationship between stress, smoking, and NA within individuals over time, we used hierarchical linear modeling [111]. The simple form of hierarchical linear modeling (HLM) can be conceived as two separate models, one a within-person model (Level 1) and the other a between-person model (Level 2). A distinctive feature of HLM is that the intercepts and slopes are allowed to vary across persons, allowing estimates of individual differences in within-person effects. To examine the temporal links among stress, smoking, and NA, we fit a within-person model essentially equivalent to 256 (the number of smokers in the sample) regressions assessing daily covariation among each of these variables.

The daily covariation among the three variables of interest with NA as the dependent variable and both stress and smoking as the predictors can be expressed as:

$$\begin{aligned}
 NA_{ij} &= b_{00} + b_{10}(\text{Stressor}_{ij}) + b_{20}(\text{Smoking}_{ij}) + e_{ij} \\
 b_{00} &= \beta_{00} + U_{0j} \\
 b_{10} &= \beta_{10} + U_{1j} \\
 b_{20} &= \beta_{20} + U_{2j}
 \end{aligned}$$

where, NA for person j on day i , is a function of their average level of NA (b_{00}), whether they experience a stressor (b_{10}), the number of cigarettes consumed (b_{20}), and a residual (e_{ij}). β_{00} , β_{10} , and β_{20} are the average within-person intercept, stressors, and smoking effects (i.e., fixed effects), while U_{0j} , U_{1j} , and U_{2j} are the random effects, and reflect person-specific deviations from the average values. Stressors and smoking were both person-centered to reflect deviations from each individual's average levels of stressors and smoking. Thus, the values of these variables are constant across individuals with a mean of 0, and reflect only within-person variability. The person-specific means, or between-person effects, were also included in all models.

Table 2 Between-person correlations for smoking, NA, daily stress, and gender

Variable	M	SD	CC	NA	FSD
Cigarette consumption	17.26	11.44			
Negative affect	2.41	3.36	.15*		
Frequency of stress days	0.41	0.27	-.02	.31**	
Average perceived stress	1.27	1.09	.04	.16*	.83**

CC cigarette consumption, NA negative affect, FSD frequency of stress days

* $p < .05$, ** $p < .01$

Results

Bivariate Relationships among the Variables

Before proceeding to the central hypothesis in the study, we examined the data for consistency with widely reported bivariate relationships in the extant literature (see Table 2). Smoking was positively related to NA, $r(255) = .15$, $p < .05$, but was not reliably associated with stress measured as frequency of stressor days (i.e., days on which at least one stressor is endorsed), $r(255) = -.02$, ns, or perceived stress, $r(255) = .04$, ns. NA was positively associated with stressors, and this was true regardless of whether daily stressors were calculated using frequency of stressor days, $r(255) = .31$, $p < .0001$, or perceived stress, $r(255) = .16$, $p < .05$. Consistent with prior findings, gender was reliably associated with the average number of cigarettes smoked, $r(255) = -.26$, $p < .0001$, indicating that women smoke significantly fewer cigarettes than men. These findings are generally consistent with prior research.

Stressors and Smoking as Predictors of NA

We next examined the degree to which stressors and smoking predicted NA (see Table 3). The first model

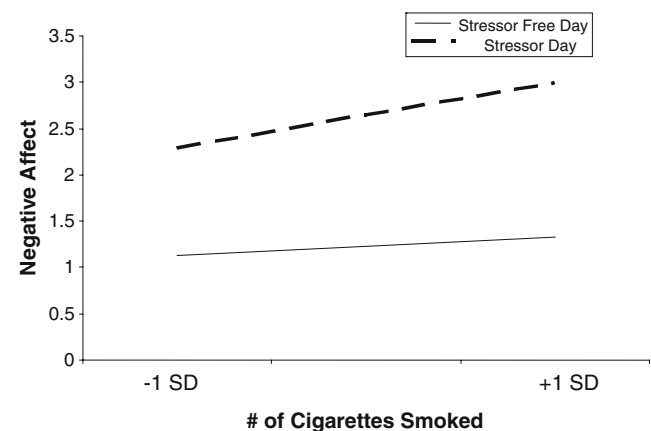
Table 3 Predicting negative affect from stressor frequency/severity and smoking

	Model 1 Estimate (SE)	Model 2 Estimate (SE)
Fixed effects		
Intercept	.69 (.32)*	.65 (.32)*
Daily stressors (WP)	1.79 (.21)**	1.80 (.21)**
Daily stressors (BP)	3.38 (.59)**	3.56 (.59)**
Smoking (WP)	.05 (.02)*	-.01 (.03)
Smoking (BP)	.02 (.01)	.01 (.01)
Daily stress (WP) × smoking (WP)	–	.14 (.04)**
Variance components		
Intercept	2.41 (.48)**	2.41 (.48)**
Daily stress	3.03 (1.07)**	2.86 (1.05)**
Smoking	.05 (.01)**	.06 (.01)**
Residual	8.36 (.37)**	8.29 (.37)**

Smoking estimate reflect effects for the number of cigarettes smoked WP within-person, BP between-person

(Model 1) examined the unique effects of daily stressor exposure and cigarette smoking on daily negative affect. Daily stressor exposure was associated with NA at the between and within person levels. Individuals with a higher frequency of stressor days reported higher NA (estimate = 3.38, SE = .59), and NA was significantly higher on stressor days compared to nonstressor days (estimate = 1.79, SE = .21). For smoking, the between-person effect was not significant indicating the NA of heavy smokers is not significantly different from the NA of people who are not heavy smokers (estimate = .02, SE = .01). However, the within-person effect of smoking on NA was significant, indicating that people reported higher levels of NA on days when they smoked more cigarettes than usual (estimate = .05, SE = .02). We then estimated a second model (Model 2), to examine whether the day-to-day association between smoking and NA differed between stressor and nonstressor days. This was accomplished by adding the interaction between the within-person daily stressors and smoking effects. The interaction was significant (estimate = .14, SE = .04; see Fig. 1) indicating that smoking more than usual was associated with higher NA on stressor days (estimate = .13, SE = .04) but not on nonstressor days (estimate = -.01, SE = .03). Altogether, 17.4% of the variability in daily negative affect was accounted for with this model.

To consider the magnitude of the smoking effect, we calculated the amount of daily variability in NA that

**Fig. 1** Negative affect as a function of cigarette consumption and daily stressor occurrence

smoking accounted for on stressor days and nonstressor days, respectively. This was done following methods describe by Bryk and Raudenbush [112] for calculating the pseudo- R^2 . Across nonstressor days, the daily variation (residual variance) in NA decreased from 5.35 to 5.04 after controlling for smoking, indicating that smoking accounted for 5.7% of the daily variability. Across stressor days, the daily variation in NA was reduced from 14.92 to 12.81 after controlling for smoking, indicating that smoking accounted for 14.1% of the daily variability.

The initial models demonstrated that smoking more than usual was associated with higher levels of NA on stressor days. The next set of analyses explored possible variation in stressors days. The link between smoking and NA could be due to characteristics of the stressor day such as the number and severity of stressors experienced. If the link between smoking and NA on days when people report a stressor is due to the number of stressors being experienced or more severe stressors being experienced, then controlling for these factors should attenuate the effect of smoking on NA. We tested this hypothesis by estimating a model where we examined the effect of smoking on NA across stressor days, controlling for the number and severity of stressors reported following methods described by [113].

The results of this model can be seen in Table 4 (Model 1). Across stressor days, the within-person effect for number of stressors indicates that NA increased 3.27 (SE=.39) units per stressor reported, while the between-person effect shows that individual's whose stressor days are characterized by a greater number of stressors report higher levels of NA (estimate=5.14, SE=.82). Similarly for the severity of stressors, the within-person effect indicates that NA was higher on days when the stressors experienced were reported to be more severe (estimate=1.08, SE=.16), and the between-person effect indicates that NA is highest among individuals who report their stressors to be of greater severity (estimate=2.13, SE=.32). Importantly, the within-person

effect of smoking remained significant (estimate=.10, SE=.04) indicating that the link between smoking and NA across stressor days could not be explained by the number of severity of the stressors reported.

We also estimated a second model (Table 4, Model 2) to test whether the number or severity of the stressors reported moderated the effects of smoking on NA. As can be seen in Table 4, neither the interaction between smoking and number of stressors (estimate=-.09, SE=.09), nor between smoking and stressor severity (estimate=.03, SE=.04) was significant. Together, the results of these models suggest that the effect of smoking on NA across stressor days cannot be attributed to the number or severity of the events reported. Furthermore, the association between smoking and NA is not moderated by the number or severity of stressors reported.

Discussion

The relationship between smoking and NA is complex [23, 114]. The stress induction model posits that, in between cigarettes, smokers experience distressing withdrawal symptoms as the result of acute nicotine deprivation. These withdrawal symptoms are associated with NA. To relieve these feelings, smokers engage in smoking to replenish fallen nicotine levels. After smoking, nicotine levels are restored and smokers experience withdrawal reversal. This cycle is repeated numerous times throughout the day. Parrott suggests that, while smokers report that smoking improves their mood, the stress induction model suggests that smoking only serves to reverse the NA that is associated with acute nicotine deprivation.

Findings from our study lend tentative support to the stress induction model. First, NA was positively associated with cigarette consumption. Moreover, on days when participants smoked more than usual and experienced any

Table 4 Multilevel model estimates of smoking predicting negative affect across stressor days, controlling for the number and severity of the stressors reported

	Model 1	Model 2
	Estimate (SE)	Estimate (SE)
Intercept	2.26 (.90)***	2.29 (.90)***
Smoking (WP)	.10 (.04)***	.10 (.04)***
Smoking (BP)	.04 (.02)*	.04 (.02)*
Number of stressors (WP)	3.27 (.39)***	3.28 (.40)***
Number of stressors (BP)	5.14 (.82)***	5.14 (.82)***
Severity of stressors (WP)	1.08 (.16)***	1.09 (.16)***
Severity of stressors (BP)	2.13 (.32)***	2.14 (.32)***
Smoking (WP) × number of stressors (WP)	–	-.09 (.09)
Smoking (WP) × severity of stressors (WP)	–	.03 (.04)

* $p < .10$, ** $p < .05$, *** $p < .01$

daily stressor, their NA was higher. This finding was not accounted for by the severity or number of daily stressors experiences. This suggests that there may be a threshold effect for the role of daily stressors on the smoking–NA relationship. Experiencing *any* daily stressors, both in terms of frequency and perception, appears sufficient enough to alter the association between smoking and NA. Specifically, the association between smoking and NA is stronger on stressor days than on nonstressor days.

The discovery that exogenous stressors (i.e., daily hassles in various life domains) intensify the relationship between smoking and NA is an important new finding in the literature. It suggests that smokers may experience a “double whammy” on stressful days. Specifically, it appears that the endogenous stress of acute nicotine deprivation is compounded by exposure to exogenous stressors to heighten NA states. In other words, the two sources of stress may compound each other and serve to make smokers feel emotionally worse off. Since smokers tend to believe that smoking ameliorates stress and NA [8–13, 15, 16], they will likely seek relief in smoking more, thus perpetuating a cycle of distress [18, 19, 26]. As long as smokers believe that relief emanates from smoking, they will have little motivation to effectively deal with either the endogenous or exogenous sources of stress. Indeed, there is evidence that stress may shake smokers out of their “boundary of comfort” [35], thus, strengthening the relationship between smoking and NA which in turn leads to increased smoking.

There is other evidence to suggest that smoking increases irritability and distress [17, 18], while sustained abstinence decreases negative affective states [20, 115]. For example, studies with adolescents (a developmental stage when stress is high) have found that smoking is associated with depressive symptoms and greater levels of NA [116, 117]. Some research has suggested that smoking in adolescents does mitigate NA, but as smokers age, smoking exacerbates NA [118]. Our results lend support to this notion since we studied middle aged smokers. Compared with nonquitters, those who sustained abstinence reported less perceived stress, used more positive coping strategies, and engaged in fewer negative coping strategies [119].

There are also several implications of our research for both individual behavior and public health approaches to smoking. To the extent to which smokers perceive that smoking attenuates NA, the more likely it is that they will use smoking as a coping mechanism [120]. Therefore, behavioral interventions designed to prevent or reduce smoking should assist smokers to find healthy alternative coping strategies to deal with stress and NA. To the extent to which the mitigating effects of smoking are “in the mind” of smokers, greater public health efforts will need to be made to widely dispel a potentially dangerous myth. Moreover, given that as cigarette smokers age, they become

less likely to express readiness to quit [10, 121]; better understanding of the smoking–stress–NA relationship needs to occur at all developmental stages [3].

While the results of our study lend support to the stress induction model of cigarette smoking, a number of caveats are in order. First, the stress induction model itself has been called into question. For example, several researchers suggest that the model fails to explain results from studies which suggest that smoking does ameliorate distress and NA [22, 24]. Other criticism include that the stress induction model (a) ignores the problem of selective relapse, (b) does not account for effects of repeated measures, (c) assumes that smokers and nonsmokers are comparable in terms of stress and affect liability, and (d) has not been rigorously assessed for directionality [22, 23, 122]. Recently, however, Parrott has addressed a number of these criticisms [123]. It is also important to note that smoking occurs in the absence of withdrawal symptoms [27, 28], and withdrawal symptoms are highly variable across individuals [124]. Therefore, the stress induction model may only generalize to those smokers most sensitive to withdrawal symptoms. On the other hand, there is evidence that acute withdrawal is an important factor in smoking behavior. First, withdrawal emerges relatively rapidly. This should not be surprising since the half-life of nicotine is about 10 min [125]. For example, experimental studies suggest that withdrawal can occur within 1-h post cessation [126–129]. Second, the typical interval of smoking in the natural environment is under 40 min in heavy smokers [130], a timeframe that is consistent with acute withdrawal.

There are also several methodological limitations in this study. First, we did not measure a number of key variables central to the model (i.e., nicotine, cotinine). Therefore, we assume that acute nicotine deprivation occurred. Second, our results are based on correlation analyses. Therefore, we cannot rule out the influence of unmeasured confounding variables nor are we able to make definitive conclusions about the directionality of the relationships among smoking, stress, and NA. In their exhaustive review of the literature [23], Kassel et al. concluded that it was not clear whether NA precedes or follows smoking [3]. For example, Shiffman et al. examined the antecedents of smoking in naturalistic settings and found no support for the notion that smoking is under control of affective antecedents [114]. However, some studies have found temporal evidence that rapid increases in NA precede smoking when examined on a daily basis [80]. In this study, momentary rating from smokers enrolled in a cessation program found significant NA effects in the hours before smoking lapses. Our study relied solely on self-report. Moreover, respondents relied to a degree on retrospection, although within 1 day. Momentary ratings [114] might have led to differing results. More studies using momentary

ratings of smoking, stress, and NA are needed to help determine the direction of these relationships. Our study also relied heavily on participant compliance. However, by contacting participants via phone each night at a pre-agreed to time, participation rates were excellent. Future studies should attempt to operationalize key constructs using both subjective and objective measurement.

Despite its limitations, the study possessed a number of strengths. The sample was representative of the middle-aged Americans as well as representative of adult smokers in the United States in terms of cigarettes smoked per day. The use of multilevel modeling allowed us to examine both within and between differences in the relationship between stress, smoking, and NA. The assessment of daily stressors using the DISE provides for much more fine-grained assessment of these experiences than checklists which are commonly used. This was also the first study examining stress, NA, and smoking over time. Given the unique strengths of the NSDE data, the results of this study should be instructive to theorists, researchers, and healthcare professionals.

References

- Hyland A, Vena C, Bauer J, Li Q, Giovino GA, Yang J, Cummings KM. Cigarette smoking-attributable morbidity—United States, 2000. *MMWR Morb Mortal Wkly Rep.* 2003; 52: 842–844.
- Centers for Disease Control and Prevention. Tobacco use among adults—United States, 2005. *MMWR Morb Mortal Wkly Rep.* 2006; 55: 1145–1148.
- Kassel JD, Stroud LR, Paronis CA. Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychol Bull.* 2003; 129: 270–304.
- Nesbitt PD. Smoking, physiological arousal, and emotional response. *J Pers Soc Psychol.* 1973; 25: 137–144.
- Gilbert DG, Welser R. Emotion, anxiety and smoking. In: Ney T, Gale A, eds. *Smoking and Human Behavior.* Chichester: Wiley; 1989: 171–196.
- Ikard FF, Tomkins S. The experience of affect as a determinant of smoking behavior: A series of validity studies. *J Abnormal Psychol.* 1973; 81: 172–181.
- Pomerleau O, Adkins D, Pertschuk M. Predictors of outcome and recidivism in smoking cessation treatment. *Addict Behav.* 1978; 3: 65–70.
- Russell M, Peto J, Patel U. The classification of smoking by factorial structure of motives. *J R Stat Soc, Ser A.* 1974; 137: 313–333.
- Shiffman S. Relapse following smoking cessation: A situational analysis. *J Consult Clin Psychol.* 1982; 50: 71–86.
- Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: The PANAS scales. *J Pers Soc Psychol.* 1988; 54: 1063–1070.
- Brandon TH, Baker TB. The smoking consequences questionnaire: The subjective expected utility of smoking in college students. *Psychol Assess.* 1991; 3: 484–491.
- Copeland AL, Brandon TH, Quinn EP. The smoking consequences questionnaire-adult: Measurement of smoking outcome expectancies of experienced smokers. *Psychol Assess.* 1995; 7: 484–494.
- Wills TA, Shiffman S. Coping and substance use: A conceptual framework. In: Shiffman S, Wills TA, eds. *Coping and substance use.* New York: Academic; 1985: 3–24.
- Khantzian EJ. The self-medication hypothesis of substance use disorders: A reconsideration and recent applications. *Harv Rev Psychiatry.* 1997; 4: 231–244.
- Parrott AC. Cigarette smoking: Effects upon self-rated stress and arousal over the day. *Addict Behav.* 1993; 18: 389–395.
- Parrott AC. Stress modulation over the day in cigarette smokers. *Addiction.* 1995; 90: 233–244.
- Parrott AC. Nesbitt's Paradox resolved? Stress and arousal modulation during cigarette smoking. *Addiction.* 1998; 93: 27–39.
- Parrott AC. Does cigarette smoking cause stress? *Am Psychol.* 1999; 54: 817–820.
- Parrott AC. Heightened stress and depression follow cigarette smoking. *Psychol Rep.* 2004; 94: 33–34.
- Parrott AC. Smoking cessation leads to reduced stress, but why? *Int J Addict.* 1995; 30: 1509–1516.
- Parrott AC. Individual differences in stress and arousal during cigarette smoking. *Psychopharmacology (Berl).* 1994; 115: 389–396.
- Gilbert DG, McClernon FJ. A smoke cloud of confusion. *Am Psychol.* 2000; 55: 1158–1159.
- Kassel JD. Smoking and stress: Correlation, causation, and context. *Am Psychol.* 2000; 55: 1155–1156.
- Piasecki TM, Baker TB. Does smoking amortize negative affect? *Am Psychol.* 2000; 55: 1156–1157.
- Lazarus RS. The psychology of stress and coping. *Issues Ment Health Nurs.* 1985; 7: 399–418.
- Parrott AC. Cigarette smoking does cause stress. *Am Psychol.* 2000; 55: 1159–1160.
- Patten CA, Martin JE. Does nicotine withdrawal affect smoking cessation? Clinical and theoretical issues. *Ann Behav Med.* 1996; 18: 190–200.
- Piasecki TM. Relapse to smoking. *Clin Psychol Rev.* 2006; 26: 196–215.
- Shiffman S. Assessing smoking patterns and motives. *J Consult Clin Psychol.* 1993; 61: 732–742.
- Frith CD. Smoking behavior and its relation to the smoker's immediate experience. *Br J Soc Clin Psychol.* 1971; 10: 73–78.
- Balfour DJ, Ridley DL. The effects of nicotine on neural pathways implicated in depression: A factor in nicotine addiction? *Pharmacol Biochem Behav.* 2000; 66: 79–85.
- Pomerleau CS, Pomerleau OF. The effects of a psychological stressor on cigarette smoking and subsequent behavioral and physiological responses. *Psychophysiology.* 1987; 24: 278–285.
- Wise RA. Drug-activation of brain reward pathways. *Drug Alcohol Depend.* 1998; 51: 13–22.
- Eysenck HJ. *Smoking, personality, and stress: Psychosocial factors in the prevention of cancer and coronary heart disease.* New York: Springer; 1991.
- Kozlowski LT, Herman CP. The interaction of psychosocial and biological determinants of tobacco use: More on the boundary model. *J Appl Soc Psychol.* 1984; 14: 244–256.
- Almeida DM, Wethington E, Kessler RC. The daily inventory of stressful events: An interview-based approach for measuring daily stressors. *Assessment.* 2002; 9: 41–55.
- Almeida DM. Resilience and vulnerability to daily stressors assessed via diary methods. *Curr Dir Psychol Sci.* 2005; 14: 64–68.
- Pearlin LI, Skaff MM. Stressors and adaptation in late life. In: Gatz M, ed. *Emerging issues in mental health and aging.* Washington, DC: American Psychiatric; 1995.
- Almeida DM, Kessler RC. Everyday stressors and gender differences in daily distress. *J Person Soc Psychol.* 1998; 75: 670–680.

40. Bolger N, DeLongis A, Kessler RC, Schilling EA. Effects of daily stress on negative mood. *J Person Soc Psychol.* 1989; 57: 808–818.
41. Bolger N, Foster M, Vinokur AD, Ng R. Close relationships and adjustments to a life crisis: The case of breast cancer. *J Person Soc Psychol.* 1996; 70: 283–294.
42. Eckenrode J. Impact of chronic and acute stressors on daily reports of mood. *J Person Soc Psychol.* 1984; 46: 907–918.
43. Grzywacz JG, Almeida DM, Neupert SD, Ettner SL. Socioeconomic status and health: A micro-level analysis of exposure and vulnerability to daily stressors. *J Health Soc Behav.* 2004; 45: 1–16.
44. DeLongis A, Folkman S, Lazarus RS. The impact of daily stress on health and mood: Psychological and social resources as mediators. *J Person Soc Psychol.* 1988; 54: 486–495.
45. Pearlin LI. Stress and mental health: A conceptual overview. In: Horwitz AV, Scheid TL, eds. *Handbook for the Study of Mental Health: Social Contexts, Theories, and Systems.* Cambridge: Cambridge University Press; 1999: 161–175.
46. Pearlin LI. The stress process revisited: Reflections on concepts and their interrelationships. In: Aneshensel CS, Phelan JC, eds. *Handbook on the Sociology of Mental Health.* New York: Springer; 1999: 395–415.
47. Repetti RL. Short-term effects of occupational stressors on daily mood and health complaints. *Health Psychol.* 1993; 12: 125–131.
48. Repetti RL. The effects of workload and the social environment at work on health. In: Goldberger L, Breznitz S, eds. *Handbook of Stress: Theoretical and Clinical Aspects.* New York: Free; 1993: 120–130.
49. Cohen S, Kessler RC, Gordon LU. *Measuring Stress: A Guide for Health and Social Scientists.* New York: Oxford University Press; 1997.
50. Pearlin LI, Menaghan EG, Lieberman MA, Mullan JT. The stress process. *J Health Soc Behav.* 1981; 22: 337–356.
51. Selye H. What is stress? *Metabolism.* 1956; 5: 525–530.
52. Lazarus RS. Puzzles in the study of daily hassles. *J Behav Med.* 1984; 7: 375–389.
53. Patton GC, Hibbert M, Rosier MJ, et al. Is smoking associated with depression and anxiety in teenagers? *Am J Public Health.* 1996; 86: 225–230.
54. Hatch JP, Biemer SM, Fisher JG. The effects of smoking and cigarette nicotine content on smokers' preparation and performance of a psychosocially stressful task. *J Behav Med.* 1983; 6: 207–216.
55. Herbert M, Foulds J, Fife-Schaw C. No effect of cigarette smoking on attention or mood in non-deprived smokers. *Addiction.* 2001; 96: 1349–1356.
56. Jarvik ME, Caskey NH, Rose JE, Herskovic JE, Sadeghpour M. Anxiolytic effects of smoking associated with four stressors. *Addictive Behav.* 1989; 14: 379–386.
57. Perkins KA, Grobe JE, Epstein LH, Caggiula AR, Stiller RL. Effects of nicotine on subjective arousal may be dependent on baseline subjective state. *J Subst Abuse.* 1992; 4: 131–141.
58. Gilbert DG. *Smoking: Individual Differences, Psychopathology, and Emotion. The Series in Health Psychology and Behavioral Medicine.* Philadelphia: Taylor & Francis; 1995.
59. Kassel JD. Smoking and attention: A review and reformulation of the stimulus-filter hypothesis. *Clin Psychol Rev.* 1997; 17: 451–478.
60. Kassel JD, Shiffman S. Attentional mediation of cigarette smoking's effect on anxiety. *Health Psychol.* 1997; 16: 359–368.
61. Kassel JD, Unrod M. Smoking, anxiety, and attention: Support for the role of nicotine in attentionally mediated anxiety. *J Abnorm Psychology.* 2000; 109: 161–166.
62. Shiffman S, Paty JA, Gnys M, Kassel JA, Hickcox M. First lapses to smoking: Within-subjects analysis of real-time reports. *J Consult Clin Psychol.* 1996; 64: 366–379.
63. Conway TL, Vickers RR, Ward HW, Rahe RH. Occupational stress and variation in cigarette, coffee, and alcohol consumption. *J Health Soc Behav.* 1981; 22: 155–165.
64. Ogden J, Mitandabari T. Examination stress and changes in mood and health related behaviors. *Psychol Health.* 1997; 12: 288–299.
65. Steptoe A, Wardle J, Pollard TM, Canaan L, Davies GJ. Stress, social support and health-related behavior: A study of smoking, alcohol consumption and physical exercise. *J Psychosom Res.* 1996; 41: 171–180.
66. Green KL, Johnson JV. The effects of psychosocial work organization on patterns of cigarette smoking among male chemical plant employees. *Am J Publ Health.* 1990; 80: 1368–1371.
67. House JS, Strecher V, Metzner HL, Robbins CA. Occupational stress and health among men and women in the Tecumseh Community Health Study. *J Health Soc Behav.* 1986; 27: 62–77.
68. Mensch BS, Kandel DB. Do job conditions influence the use of drugs? *J Health Soc Behav.* 1988; 29: 169–84.
69. Frone MR, Cooper ML, Russell M. Stressful life events, gender, and substance abuse: An application of tobit analysis. *Psychol Addict Behav.* 1994; 8: 59–69.
70. Todd M. Daily processes in stress and smoking: Effects of negative events, nicotine dependence, and gender. *Psychol Addict Behav.* 2004; 18: 31–39.
71. Todd M, Chassin L, Presson CC, Sherman SJ. Role stress, socialization, and cigarette smoking: Examining multiple roles and moderating variables. *Psychol Addict Behav.* 1996; 10: 211–221.
72. Creson D, Schmitz JM, Arnoutovic A. War-related changes in cigarette smoking: A survey study of health professionals in Sarajevo. *Subst Use Misuse.* 1996; 31: 639–646.
73. Rose JE, Ananda S, Jarvik ME. Cigarette smoking during anxiety-provoking and monotonous tasks. *Addict Behav.* 1983; 8: 353–359.
74. Perkins KA, Grobe JE. Increased desire to smoke during acute stress. *Br J Addict.* 1992; 87: 1037–1040.
75. Payne TJ, Schare ML, Levis DJ, Colletti G. Exposure to smoking-relevant cues: Effects on desire to smoke and topographical components of smoking behavior. *Addict Behav.* 1991; 16: 467–479.
76. Folkman S, Lazarus RS. Stress processes and depressive symptomatology. *J Abnorm Psychology.* 1986; 95: 107–113.
77. Folkman S, Lazarus RS, Gruen RJ, DeLongis A. Appraisal, coping, health status, and psychological symptoms. *J Pers Soc Psychol.* 1986; 50: 571–579.
78. Mroczek DK, Almeida DM. The effect of daily stress, personality, and age on daily negative affect. *J Person.* 2004; 72: 355–378.
79. Serido J, Almeida DM, Wethington E. Chronic stressors and daily hassles: Unique and interactive relationships with psychological distress. *J Health Soc Behav.* 2004; 45: 17–33.
80. Shiffman S, Waters AJ. Negative affect and smoking lapses: A prospective analysis. *J Consult Clin Psychol.* 2004; 72: 192–201.
81. Dembroski TM, MacDougall JA, Cardozo SR, Ireland SK, Krug-Fite J. Selective cardiovascular effects of stress and cigarette smoking in young women. *Health Psychol.* 1985; 4: 153–167.
82. MacDougall JM, Dembroski TM, Slaats S, Herd JA, Eliot RS. Selective cardiovascular effects of stress and cigarette smoking. *J of Hum Stress.* 1983; 9: 13–21.
83. Perkins KA, Epstein LH, Jennings JR, Stiller R. The cardiovascular effects of nicotine during stress. *Psychopharmacology.* 1986; 90: 373–378.
84. Pomerleau OF, Pomerleau CS. Cortisol response to a psychological stressor and/or nicotine. *Pharmacol Biochem Behav.* 1990; 36: 211–213.
85. Buchanan TW, al'Absi M, Lovallo WR. Cortisol fluctuates with increases and decreases in negative affect. *Psychoneuroendocrinology.* 1999; 24: 227–241.

86. Susman EJ, Dorn LD, Chrousos GP. Negative affect and hormone levels in young adolescents: Concurrent and predictive perspectives. *J Youth Adolesc.* 1991; 20: 167–190.
87. Ewart CK, Kolodner KB. Negative affect, gender, and expressive style predict elevated ambulatory blood pressure in adolescents. *J Pers Soc Psychol.* 1994; 66: 596–605.
88. Enkelmann HC, Bishop GD, Tong EW, et al. The relationship of hostility, negative affect and ethnicity to cardiovascular responses: An ambulatory study in Singapore. *Int J Psychophys.* 2005; 56: 185–197.
89. Whalen CK, Jamner LD, Henker B, Delfino RJ. Smoking and moods in adolescents with depressive and aggressive dispositions: Evidence from surveys and electronic diaries. *Health Psychol.* 2001; 20: 99–111.
90. Mroczek DK, Kolarz CM. The effect of age on positive and negative affect: A developmental perspective on happiness. *J Pers Soc Psychol.* 1998; 75: 1333–1349.
91. Bradburn NM. *The Structure of Psychological Well-being.* Chicago: Aldine; 1969.
92. Kessler RC, McGonagle KA, Zhao S. Lifetime and 12-month prevalence of DSM-II-R psychiatric disorders in the United States. *Arch Gen Psychiatry.* 1994; 51: 8–19.
93. Taylor JA. A personality scale of manifest anxiety. *J Abnorm Soc Psychol.* 1953; 48: 285–290.
94. Radloff LS. The CES-D scale: A self-report depression scale for research in the general population. *App Psychol Meas.* 1977; 1: 385–401.
95. Diener E, Larsen RJ. The experience of emotional well-being. In: Lewis M, Haviland JM, eds. *Handbook of Emotions.* New York: Guilford; 1993: 405–415.
96. Diener E, Sandvik E, Pavot W. Happiness is the frequency, not the intensity, of positive versus negative affect. In: Strack F, Argyle M, Schwarz N, eds. *Subjective Well-being: An Interdisciplinary Perspective.* Oxford: Pergamon; 1991: 119–139.
97. Almeida D. *Daily Inventory of Stressful Events (DISE) Expert Coding Manual.* Tucson: University of Arizona; 1998.
98. Wethington E, Almeida DM, Brown GW, Frank E, Kessler RS. The assessment of stressor exposure. In: Vingerhoets A, ed. *Assessment in Behavioral Medicine.* New York: Taylor & Francis; 2001: 113–134.
99. Neupert SD, Almeida DM, Mroczek DK, Spiro A. Daily stressors and memory failures in a naturalistic setting: Findings from the VA Normative Aging Study. *Psychol Aging.* 2006; 21: 424–429.
100. Almeida DM. *Using Daily Diaries to Assess Temporal Friction Between Work and Family.* Mahwah: Erlbaum; 2004.
101. Grzywacz JG, Almeida DM, McDonald DA. Work-family spillover and daily reports of work and family stress in the adult labor force. *Fam Relat.* 2002; 51: 28–36.
102. Ridley CA, Cate RM, Collins DM, Reesing AL, Lucero AA. The Ebb and flow of marital lust: A relational approach. *J Sex Res.* 2006; 43: 144–153.
103. Almeida DM, Neupert SD, Banks SR, Serido J. Do daily stress processes account for socioeconomic health disparities? *J Gerontol B Psychol Sci Soc Sci.* 2005; 60B: 34–39.
104. Almeida DM, Horn MC. Is daily life more stressful during middle adulthood. In: Brim OG, Ryff CD, Kessler RC, eds. *How Healthy are We?* Chicago: University of Chicago Press; 2004: 425–451.
105. Piazza JR, Charles ST, Almeida DM. Living with chronic health conditions: Age differences in affective well-being. *J Gerontol B Psychol Sci Soc Sci.* 2007; 62B: P313–P321.
106. Neupert SD, Almeida DM, Charles ST. Age differences in reactivity to daily stressors: The role of personal control. *J Gerontol B Psychol Sci Soc Sci.* 2007; 62B: P216–P225.
107. Almeida DM, Serido J, McDonald D. Daily life stressors of early and late baby boomers. In: Whitbourne SK, Willis SL, eds. *The Baby Boomers Grow Up.* New York: Routledge; 2006: 165–184.
108. Charles ST, Almeida DM. Genetic and environmental effects on daily life stressors: More evidence for greater variation in later life. *Psychol Aging.* 2007; 22: 331–340.
109. Brown GW. Life events and measurement. In: Brown GW, Harris TO, eds. *Life Events and Illness.* London: Guilford; 1989: 3–45.
110. Department of Health and Human Services. *Results from the 2002 National Survey on Drug Use and Health: National Findings.* Rockville: Substance Abuse and Mental Health Services Administration; 2003.
111. Bryk AS, Raudenbush SW. *Hierarchical Linear Models: Applications and Data Analysis Methods.* London: Sage; 1992.
112. Raudenbush SW, Bryk AS. *Hierarchical Linear Models: Applications and Data Analysis Methods.* Thousand Oaks: Sage; 2002.
113. Stawski R, Sliwinski MJ, Almeida DM, Smyth JM. Reported exposure and emotional reactivity to daily stressors: The roles of adult-age and global perceived stress. *Psychol Aging.* 2008; 23: 52–61.
114. Shiffman S, Gwaltney CJ, Balabanis MH, et al. Immediate antecedents of cigarette smoking: An analysis from ecological momentary assessment. *J Abnorm Psychology.* 2002; 111: 531–545.
115. Ludman EJ, McBride CM, Nelson JC, et al. Stress, depressive symptoms, and smoking cessation among pregnant women. *Health Psychol.* 2000; 19: 21–27.
116. Choi WS, Patten CA, Gillin JC, Kaplan RM, Pierce JP. Cigarette smoking predicts development of depressive symptoms among U.S. adolescents. *Ann Behav Med.* 1997; 19: 42–50.
117. Stein JA, Newcomb MD, Bentler PM. Initiation and maintenance of tobacco smoking: Changing personality correlates in adolescence and young adulthood. *J Appl Soc Psychol.* 1996; 26: 160–187.
118. Orlando M, Ellickson PL, Jinnett K. The temporal relationship between emotional distress and cigarette smoking during adolescence and young adulthood. *J Consult Clin Psychol.* 2001; 69: 959–970.
119. Carey MP, Kalra DL, Carey KB, Halperin S, Richards CS. Stress and unaided smoking cessation: A prospective investigation. *J Consult Clin Psychol.* 1993; 61: 831–838.
120. Vollrath M. Smoking, coping and health behavior among university students. *Psychol Health.* 1998; 13: 431–441.
121. Cataldo JK. Smoking and aging. Clinical implications. Part I: Health and consequence. *J Gerontol Nurs.* 2003; 29: 15–20.
122. Piasecki TM, Baker TB. Any further progress in smoking cessation treatment? *Nicotine Tob Res.* 2001; 3: 311–323.
123. Parrott AC. Nicotine psychobiology: How chronic-dose prospective studies can illuminate some of the theoretical issues from acute-dose research. *Psychopharmacology (Berl).* 2006; 184: 567–576.
124. Shiffman SM, Jarvik ME. Smoking withdrawal symptoms in two weeks of abstinence. *Psychopharmacology (Berl).* 1976; 50: 35–39.
125. Russell MA. Nicotine replacement: The role of blood nicotine levels, their rate of change, and nicotine tolerance. *Prog Clin Biol Res.* 1988; 261: 63–94.
126. Gross J, Lee J, Stitzer ML. Nicotine-containing versus denicotinized cigarettes: Effects on craving and withdrawal. *Pharmacol Biochem Behav.* 1997; 57: 159–165.
127. Hendricks PS, Ditte JW, Drobos DJ, Brandon TH. The early time course of smoking withdrawal effects. *Psychopharmacology.* 2006; 187: 385–396.
128. Schuh KJ, Stitzer ML. Desire to smoke during spaced smoking intervals. *Psychopharmacology.* 1995; 120: 289–295.
129. Tiffany ST, Drobos DJ. The development and initial validation of a questionnaire on smoking urges. *Br J Addict.* 1991; 86: 1467–1476.
130. Hatsukami DK, Pickens RW, Svikis DS, Hughes JR. Smoking topography and nicotine blood levels. *Addict Behav.* 1988; 13: 91–95.