Life Before and After Quitting Smoking: An Electronic Diary Study

Danielle E. McCarthy
University of Wisconsin—Madison

Thomas M. Piasecki
University of Missouri—Columbia

Michael C. Fiore and Timothy B. Baker
University of Wisconsin—Madison

This article describes a multidimensional, multivariate, and multilevel approach to the assessment of nicotine withdrawal. In this prospective study, 70 adult smokers assigned to an active or placebo nicotine patch condition completed multiple daily assessments using an electronic diary. Average and individual growth curves were estimated for affective and nonaffective withdrawal symptoms. All symptoms but hunger increased significantly on the quit day and remained elevated for three weeks. Variability in symptom experiences across individuals increased from pre- to post-quit. Relations between symptom reports (e.g., negative affect or craving) and episodic events (e.g., stressful events or seeing someone smoke) changed from pre-quit to post-quit. Pre-quit increases in negative affect and quit-day increases in craving were inversely related to abstinence three months after the quit day, suggesting that anticipatory and immediate reactions to quitting influence success.

Keywords: ecological momentary assessment system, electronic diary, smoking cessation, withdrawal

There is considerable controversy over the role played by withdrawal in motivating continued drug use and relapse. Addiction theories that were prominent 20 to 50 years ago emphasized the role of negative reinforcement via withdrawal reduction as a primary determinant of drug motivation (Siegel, 1983; Solomon, 1977; Solomon & Corbit, 1974; Wikler, 1948, 1977, 1980). Such models posit that individuals continue to smoke despite the substantial costs of tobacco use (Centers for Disease Control, 2002) and the well-known benefits of quitting (U.S. Department of Health and Human Services, 1990), because smoking is negatively reinforced through the alleviation of aversive withdrawal symptoms. As such, these models depict withdrawal as a potent motivator of continued or renewed drug use.

In support of the withdrawal-relief model of addiction, several studies have found that individual symptoms or withdrawal summary scores predict subsequent smoking status (e.g., Covey, Glassman, & Stetner, 1990; Kenford et al., 2002; Swan, Ward, & Jack, 1996; West, Hajek, & Belcher, 1989). In addition, smokers frequently report that they smoke to relieve negative affect (e.g.,

Gilbert, Sharpe, Ramanaiah, Detwiler, & Anderson, 2000; Wetter et al., 1994), the principal component of nicotine withdrawal (Welsch et al., 1999).

Although there is evidence supporting negative reinforcement accounts of addictive drug use, these models have been supplanted in recent years by alternative models, such as those that emphasize the role of incentive strength or incentive sensitization in addiction (Hutcheson, Everitt, Robbins, & Dickinson, 2001; Robinson & Berridge, 1993, 2003; Stewart, de Wit, & Eikelboom, 1984; Stewart & Wise 1992). The plausibility and apparent corroboration of alternative models of addiction such as these have contributed to waning interest in withdrawal-relief models. In addition, withdrawal theories have lost favor among theorists in part because research has failed to show consistent, strong associations between measures of withdrawal severity and the likelihood of drug use (Hall, Havassy, & Wasserman, 1990; Hughes & Hatsukami, 1986; Jaffe, 1992; Lyvers, 1998; Robinson & Berridge, 1993). Indeed, in a review of 15 prospective smoking cessation studies, Patten and Martin (1996a) concluded that withdrawal severity was not strongly related to relapse risk.

It may be that strong associations between withdrawal and tobacco use have not been detected because tobacco withdrawal is insufficiently understood and inadequately measured. The research described below explores the nature of tobacco withdrawal and its relations with relapse. We begin by identifying unresolved questions concerning the nature of the nicotine withdrawal syndrome.

One important question concerns the extent to which withdrawal symptoms should be collapsed into a global syndrome measure versus examined as specific subcomponents (e.g., Tate, Pomerleau, & Pomerleau, 1993). Recent data have revealed important differences among symptoms. Some data have challenged the syndromal nature of withdrawal by showing that specific symptoms differ in important ways. For instance, hunger persists longer than other symptoms (Hughes, 1992) and craving seems to follow

a different time course than negative affective withdrawal symptoms, with craving sometimes being higher pre-quit than post-quit (e.g., Hughes, 1992). Craving also appears to share less variance with other withdrawal symptoms (Swan et al., 1996). This has led some to question whether craving should be considered a withdrawal symptom (4th ed., text rev., *DSM–IV–TR,* American Psychiatric Association, 2000; Hughes, 1992; Hughes, Higgins, & Hatsukami, 1990). In addition, there is at least one prominent theory that holds that craving should only be weakly or occasionally related to drug self-administration and relapse (Tiffany, 1990). In sum, important questions remain about differences among individual withdrawal symptoms—differences regarding their relations with abstinence, their trajectories, and their motivational significance.

Recent research suggests that there is considerable heterogeneity in the elevation, duration, variability, and volatility of tobacco withdrawal symptoms across individuals (Piasecki et al., 2000; Piasecki, Fiore, & Baker, 1998; Piasecki, Jorenby, Smith, Fiore, & Baker, 2003a). For example, dynamic cluster analyses of longitudinal withdrawal rating data have shown that smokers differ in the pattern and severity of their symptoms (Piasecki et al., 1998, 2000). Other research has shown that smokers' withdrawal symptom profiles can be parsed into several distinct components, each of which shows significant heterogeneity across smokers (Piasecki et al., 2003a). Additionally, research has shown strong independent relations between these various components (elevation, trajectory, and volatility) of the withdrawal syndrome and the likelihood that a smoker will relapse after a quit attempt (Piasecki, Jorenby, Smith, Fiore, & Baker, 2003b). This suggests that motivationally significant information is contained in distinct withdrawal dimensions—information that is lost if withdrawal assessments are not sensitive to heterogeneity in multifactorial and dynamic symptom patterns over time.

This recent research on withdrawal suggests that it has motivational significance and plays a vital role in relapse. However, because of measurement limitations, such as a lack of real-time data acquisition, more research is needed in this area. Given the negative results in earlier research (Patten & Martin, 1996a), it is vital to gather additional evidence that withdrawal measures index vulnerability to subsequent relapse.

More information is also needed about the nature of the substantial variability in withdrawal symptoms observed across individuals after quitting. As noted earlier, when smokers stop using tobacco they differ greatly in symptom elevation, trajectory, and volatility (Piasecki et al., 2003a). We currently know little about predictors or outcomes associated with this heterogeneity. For example, we do not know whether the heterogeneity reflects withdrawal per se, or instead reflects stable individual differences that are relatively unaffected by nicotine deprivation. There is some evidence that individuals' symptom reports in the week preceding a quit attempt are correlated with post-quit reports (Piasecki, Jorenby, Smith, Fiore, & Baker, 2003c). Comparison of extensive pre-quit and post-quit symptom reporting could determine whether the previously observed "withdrawal" heterogeneity is related to drug deprivation or merely reflective of stable differences in symptom experiences or reporting (e.g., trait-like affective lability). If such a comparison reveals that individuals' symptom experiences are influenced by quitting per se, multiple explanations could account for this effect. For example, an increase in heterogeneity upon cessation might reflect individual differences in

deprivation responses, perhaps related to drug dependence level or drug use history. Alternatively, an increase in variability across persons post-quit might reflect a change in individuals' reactivity to episodic events such as exposure to stressors or smoking cues.

Past research has established the relation between environmental events (e.g., stressors) and relapse (Shiffman, 1982, 1986; Shiffman, Paty, Gnys, Kassel, & Hickcox, 1996) and the relation between moods and subsequent smoking (Shiffman et al., 2002; Shiffman, Paty, Gwaltney, & Dang, 2004). However, research has not yet examined relations between environmental events, beyond temptation events that are strongly related to urges (Shiffman et al., 1996, 1997), and withdrawal experiences. Such research is needed to explore the possibility that the increase in relapse risk following stressful events is mediated by event-related increases in symptom distress. As others have noted, additional research is needed to characterize and explain individual differences in symptom reactions to episodic events, and to explore how quitting may influence these reactions (Shapiro, Jamner, Davydov, & James, 2002).

In order to characterize withdrawal phenomena adequately, we need to adopt assessment practices that minimize biases and have high temporal sensitivity. The majority of nicotine withdrawal studies have required participants to rate withdrawal symptoms retrospectively or to integrate experiences over an extended period of time in a single rating (e.g., Piasecki et al., 2003a). Such ratings are susceptible to a number of biases, including recall, recency, and availability biases (Hammersley, 1994; Stone & Shiffman, 1994). For example, Piasecki and colleagues (2003a, 2003b) collected withdrawal data in a traditional daily diary format. The problems of daily diary methods have been noted previously (Stone, Shiffman, Scwhartz, Broderick, & Hufford, 2002). Specifically, we know that participants often fail to adhere to instructions; for example, they complete entries for several days moments before a study visit (Stone et al., 2002). Collecting data in real time may reduce sources of bias in the data and reveal new information about a targeted phenomenon (Hammersley, 1994; Stone & Shiffman, 1994).

As assessment technology advances, an increasing number of investigators are adopting real-time data collection strategies. For example, Shiffman and colleagues (1997) collected real-time urge reports from smokers during ad-lib smoking and for up to 26 days post-quit. Although these authors reported new information about the average pattern of symptoms over time and day-to-day fluctuations in urges, they did not address questions related to individual variability in withdrawal or urge reports (Shiffman et al., 1997). In the current research, we use real-time, ecologically valid assessment methods to explore tobacco withdrawal symptom heterogeneity. Specifically, we explored relations between pre-quit and post-quit symptom reports, between episodic events and symptom reports both pre- and post-quit, and between symptom dimensions and relapse vulnerability.

The current study used real-time assessment procedures and multivariate, multidimensional, and multilevel analytic techniques to address three broad questions related to nicotine withdrawal experiences among regularly smoking adults motivated to quit smoking. First, we sought to describe the withdrawal syndrome and specific withdrawal symptoms (negative affect, hunger, and craving) in multiple dimensions (elevation, shape, and variability across smokers) for an extended period of time (three weeks) both preceding and following a target quit date. Second, we sought to

describe the relation between episodic events (e.g., exposure to people smoking, stressors, recent smoking, and temptations) and symptom ratings. Third, we sought to explore the relation between withdrawal experiences as assessed in real time and subsequent smoking behavior. These exploratory analyses allowed us to investigate whether an individual's estimated withdrawal elevation or trajectory pre- or postcessation carried meaningful information regarding his or her likelihood of abstinence, as one would expect based on a withdrawal-relief model of addictive behavior.

Method

Participants

Ninety English-speaking, heavy-smoking (cigarettes per day $\geq$ 15; breath carbon monoxide $\geq$ 10 ppm) adults ($\geq$ 18 years old) motivated to quit smoking were recruited through newspaper advertisements and from waiting lists. All participants were screened initially for serious psychopathology, use of other forms of tobacco or cessation aids, and serious cardiovascular disease. Participants taking psychoactive medications were excluded because of a possible impact on symptom reports. Only females who were neither pregnant nor breast feeding and who agreed to use birth control throughout the study were enrolled. Ineligible participants were referred to community resources for cessation assistance. Participants received compensation for completion of assessments ($10 x 15 weekly assessments), providing a final breath sample for carbon monoxide (CO) testing at three months postcessation ($25 x 1 test), and returning the electronic diary (ED) at the end of the study ($75) for a total possible remuneration of $250.

Materials and Procedure

Participants who met eligibility requirements during an initial phone screen attended a group orientation meeting to obtain study information and provide informed consent. Participants also provided demographic data, smoking history, scheduling information, and a breath sample for CO testing. An individual session was scheduled for each participant within one week of the group orientation.

At the initial individual session, participants were assigned a quit date either four or seven weeks in advance and received an ED (Palm IIIe Palmtop Computer, Palm, Inc., Santa Clara, CA) with specialized Pen-

dragon Forms software (Pendragon Software Corporation, 1999) and instruction for its use. Participants also provided another CO breath sample and completed the Fagerström Test for Nicotine Dependence (Heatherton, Kozlowski, Frecker, & Fagerström, 1991); Prime MD, for screening purposes (Spitzer et al., 1994); and additional measures (e.g., Beck Depression Inventory-II, Beck, Steer, & Brown, 1996; Affect Intensity Measure, Larsen & Diener, 1987) that will not be discussed further.

Participants returned once per week in the four to seven weeks between ED training and the quit date. The first week of ED recording was for training purposes only; data collected during this period were excluded from all analyses. At each of these pre-quit visits, and every subsequent office visit, participants completed a battery of paper and pencil measures (i.e., withdrawal, life event, and social support measures) that will not be discussed here. Data were uploaded from the ED at every office visit, and feedback regarding missed and delayed responses to ED prompts was provided. The study visit and counseling schedules are shown in Figure 1. Counseling occurred in the two sessions preceding the quit date and all but the last visit following the quit date. These 10-minute counseling sessions entailed education about quitting (e.g., preparing for the quit date) and study medication (e.g., proper use and side effects), motivational enhancement, coping skills training, stress reduction, and social support.

Follow-up interviews were conducted over the phone at two months and three months post-quit to determine participants' smoking status. Participants who reported being abstinent at three months post-quit were asked to return for a final visit to provide a breath sample for CO measurement. Participants were considered abstinent at three months if they reported no smoking in the past seven days and provided a CO reading of $\leq$ 8 ppm; all others were considered to be smoking.

For the 10 weeks that participants carried the ED, they completed brief (2–3 minutes) daily assessments four times each day, or at the participant's initiation. Prior to quitting, participants were instructed to initiate a report if they experienced a significant stressful event. After the quit day, participants were asked to initiate reports following stressful events, strong urges or temptations to smoke, or smoking. User-initiated assessments were not included in analyses for this study, as these might inflate the estimated effects of the smoking, urge, and stress covariates. In addition, there were too few user-initiated records (87 or 1.8% pre-quit and 80 or 1.7% post-quit) to examine statistically the differences between these and ED-prompted records.

ED prompts were designed to capture experiences in a distributed fashion throughout the day. The first recording was programmed to occur
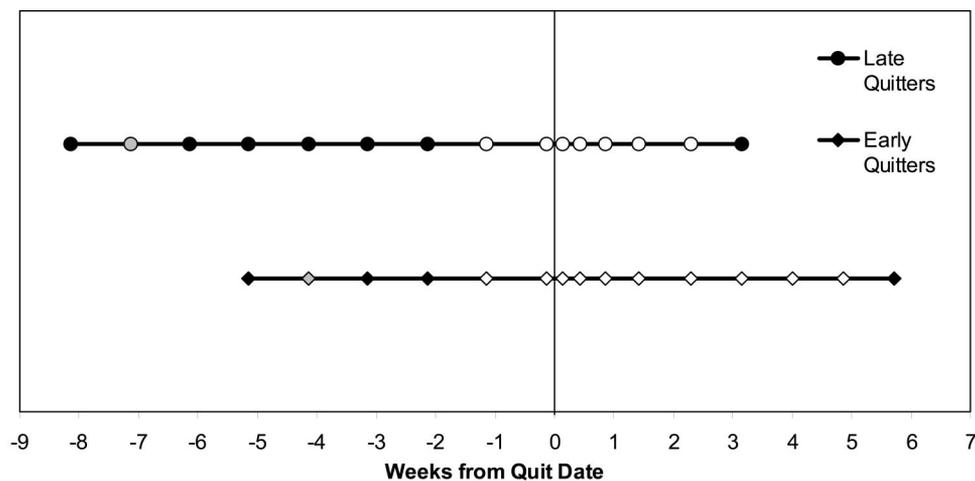

Figure 1. Study time line by quit date group. Study visits occurred at marked points for those assigned early and late quit dates. Electronic diaries were issued at the second visit (shaded in gray). White markers indicate that brief counseling occurred.

at participants' normal wake-up times. The second report occurred at a random time between wake-up and midday, but at least one hour after wake-up. The third report occurred at a random time between midday and one hour before bedtime, and the fourth report occurred at participants' normal bedtimes. Assessments that occurred fewer than 30 minutes after a previous assessment were excluded from analyses because the questions were designed to capture stressors and smoking behavior over a longer time period. A total of 523 cases (3% of all ED records in the 10-week recording period) were eliminated for this reason.

Participants used the ED to complete 10 Wisconsin Smoking Withdrawal Scale (WSWS) items, rating their agreement with each statement on a 1–5 scale (Welsch et al., 1999). The average of the 10 WSWS items constituted a participant's overall withdrawal score. Craving scores reflect the mean of two items that tapped cravings and urges to smoke. Negative affect scores were defined as the mean of six WSWS items tapping anger, anxiety, and sadness. Hunger scores reflect the mean of two items from the hunger subscale of the WSWS. In addition, participants were asked whether they had smoked in the last 15 minutes, whether they had seen someone smoking in the last 15 minutes, whether a stressful event had occurred since their last recording, and whether they had experienced a strong urge or temptation to smoke in the last 15 minutes. Each of these variables was treated as dichotomous. Responses to these items were collected for all participants, regardless of whether or not they returned to smoking.

Design

Two factors were manipulated in this study: nicotine replacement therapy and timing of the quit date. The first 47 participants recruited were aware that they received active nicotine replacement therapy delivered via a transdermal patch. Of the remaining 43 participants, 18 were randomly assigned to receive active patches and 25 to receive inert placebo patches in a double-blind fashion. Participants received a six-week supply of patches (four weeks at 21 mg and two weeks at 14 mg). Patches were offered to participants to increase the odds that a substantial portion of participants would achieve some period of abstinence. In the final sample, 16 (22.9%) of the 70 participants were in the placebo condition. This small placebo control condition was introduced halfway through the study due to concerns that the nicotine patch might be suppressing withdrawal symptoms. The placebo condition was instituted to permit examination of effect sizes as a function of patch condition (i.e., to explore whether patches suppressed symptoms).

The timing of the quit date also varied across participants in order to assess for reactivity to our intensive assessment procedures. Thirty-nine participants (43%) were randomly assigned to set a quit date after four weeks of carrying the ED, whereas the other 51 participants (57%) were assigned to quit during the seventh week of ED assessment. This schedule eliminated a confound between when quitting occurred and duration of self-recording. In the final sample, 70 participants provided observations during the three weeks preceding the quit date and the three weeks following the quit date.

Attrition

Of the 90 participants recruited, 10 withdrew from the study before providing complete demographic information and other baseline measures. Four more participants withdrew before receiving an ED. Another 16 people withdrew before attending all 15 scheduled office visits. Participants who attended most of the office visits were included in the growth-curve models, leaving a total of 70 participants in the final sample. The 20 participants who did not provide sufficient data to be included in the growth-curve analyses were significantly older ($M = 50.15$ years, $SD = 12.58$) than those who better adhered to study procedures ($M = 40.82$ years, $SD = 11.57$, $F(89) = 9.73$, $p < 003$). We heard reports from some older participants that they had difficulty hearing the ED prompts and, thus,

missed many assessments. Adherent and nonadherent participants did not differ in terms of gender, minority status, smoking heaviness, CO level, or timing of the quit date (all $p > .05$).

We observed differential attrition rates across experimental conditions. Among the participants whose quit date was set four weeks into the assessment period, 26 out of the 39 (67%) participants completed the study. The proportion of early-quitting participants completing the study was higher in the active patch conditions (22 out of 28, 79%) compared to the placebo patch condition (4 out of 11, 36%). Among the 51 participants whose quit date was set seven weeks into the assessment period, 37 (73%) completed the study. The proportion of participants with a late quit date who completed the study was comparable in both patch conditions; 25 out of 37 (68%) who received active patches completed the study, as did 8 out of 14 (57%) who received placebo patches.

Sixty-one participants out of 90 (68%) reported on their smoking behavior during the three-month telephone contact. Of these, 28 participants (40% of the total sample) reported point-prevalence abstinence and provided expired breath samples to confirm their self-reported abstinence at the three-month follow-up.

We restricted our analyses to the assessment period common to all participants (three weeks pre-quit and three weeks post-quit). The 70 participants in the final sample provided a total of 9,449 records, out of a possible 11,760 scheduled ED prompts. This represents 80.3% of target assessment occasions. Unfortunately, we do not know how the 20% of occasions that were missed might differ from the 80% that were ascertained. Complete data (four reports) were collected on 58.8% of the possible 2,940 (7 days $\times$ 6 weeks $\times$ 70 participants) assessment days of the study, and partial data were collected on an additional 32.6% of assessment days, leaving only 8.6% of target assessment days unassessed.

Although participants adhered fairly well to the ED prompting protocol, they self-initiated few reports following stressful events, strong urges and temptations, or lapses. Only 167 self-initiated reports occurred (1.7% of all records), generated by only 47 (67.1%) participants. Therefore, data analysis was restricted to ED-prompted assessments.

Growth-Curve Modeling

The within-subjects, repeated measures design of this study made the data suitable for growth-curve modeling, a technique that estimates change over time via multilevel modeling (Bryk & Raudenbush, 1992; Francis, Fletcher, Stuebing, Davidson, & Thompson, 1991; Snijders & Bosker, 1999). With growth-curve analysis, we were able to allow for missing data and unequal intervals between assessments that resulted from pseudorandomly selecting assessment times. In addition, using a discontinuous piecewise modeling technique allowed us to estimate the difference in symptom levels pre- versus post-quit and to estimate different rates of change pre- versus post-quit.[1] We examined pre- and post-quit withdrawal patterns separately within a single model and also estimated the "jump" in symptom reports coincident with the quit day. Growth-curve modeling allowed for estimation of individual variability in both withdrawal patterns as well as between-subjects treatment covariates (1 = active patch, 0 = placebo patch) modeled in post-quit data. We modeled growth in four dependent variables: overall withdrawal, negative affect, hunger, and craving.

---

[1] Although not reported here, models including quadratic growth components were also tested. Quadratic growth was not detected as significant in any model. Given the lack of support for quadratic growth and the change in interpretability of the linear components occasioned by inclusion of the quadratic coefficients, simpler linear models of symptom growth were retained. It is important to note that our results differ somewhat from Piasecki and colleagues' (2003a) finding that a quadratic model of growth achieved good fit for within-subject symptom patterns. This discrepancy may reflect a difference in the length of the follow-up period for the two studies (8 weeks vs. 3 weeks); a longer follow-up period may be necessary to detect quadratic effects.

In a preliminary step, we coded time in such a way that there was a node occurring at 12:01 a.m. on the quit day. Both the pre-quit and post-quit variables were set to zero at this node. In addition, a dummy coded variable indicated whether an observation was recorded after the quit date. This coding allowed us to interpret the pre-quit intercept as the symptom level just prior to the quit date (when all other predictors = 0) and to estimate the change in symptom levels immediately following the quit date (when all predictors except the post-quit dummy variable = 0). Data were analyzed on an occasion basis; that is, we did not aggregate data provided over multiple occasions within a day or any other time period.

Smoking in the last 15 minutes was reported on 2,085 of 4,801 (43.4%) pre-quit assessments and on 198 of 4,648 (4.3%) post-quit assessments. Exposure to someone smoking in the last 15 minutes was reported on 812 of 4,798 (16.9%) occasions pre-quit (3 records were missing this information pre-quit) and 445 of 4,646 (9.6%) occasions post-quit (2 records were missing this information post-quit). Stressful events since the last recording were reported on 193 of 4,801 (4.0%) occasions pre-quit and 205 of 4,648 (4.4%) occasions post-quit.

Using Hierarchical Linear and Nonlinear Modeling Version 5 (HLM 5, Scientific Software International; Raudenbush, Bryk, Cheong, & Congdon, 2001), we specified multilevel models for both the pre- and postcessation periods at the within-subjects level, assuming independent within-person residual terms. Restricted maximum likelihood estimation was used. All growth models contained an intercept term as well as a linear time function in both the pre- and post-quit periods. A forward model-building approach was used in which a single additional parameter was set to be random in each step, to see if this improved model fit. The first model tested was a random-intercept model in which only the intercept (or pre-quit symptom level just before the quit day) was allowed to vary between participants. We then allowed each additional coefficient (e.g., the pre-quit linear slope) to be random across persons, one variable at a time, and compared the resulting change in model deviance to a chi-square distribution to identify significant improvements in model fit. A similar forward model-building approach was used in models estimating episodic event–symptom relations (e.g., first, only the intercept would be allowed to be random, then each additional coefficient would be set to random and the resultant change in model deviance would be assessed). Misspecified models that failed to converge in 100 iterations were rejected in favor of simpler models that converged readily. When the best model fit was achieved, we then conducted exploratory analyses to see whether timing of the quit date or gender could account for variability in model coefficients.

In order to describe how symptom reports changed from pre- to post-quit, piecewise multilevel models of overall withdrawal, affective symptom, hunger, and craving scores were estimated. These models contained the following predictors: an intercept reflecting the symptom level immediately pre-quit, a dummy coded variable indicating that observations occurred post-quit (to capture the jump in symptoms from pre- to post-quit), and weeks from the quit date (linear term) both pre-quit and post-quit. We did not code the data in a way that permitted a direct comparison of the linear slope in symptoms pre- versus post-quit. Nicotine patch condition (0 = placebo condition, 1 = active condition) was entered as a between-subjects control variable.

Separate pre-quit and post-quit models that included intercept and linear growth terms were fit to the data in order to quantify the relations between episodic events (recent smoking, smoker exposure, stress, and strong temptations) and symptom reports. Each model examined the relations between one type of episodic event and one symptom variable in either the pre- or post-quit period, controlling for initial level and rate of change in the target symptom. Concurrent smoking was not controlled in models examining reactivity to other events because we were interested in examining the overall relations between distress and each event as it occurs, rather than examining a partialled, residual relationship. Episodic events were coded so that the coefficients reflect the change in symptoms associated with the presence versus absence of the event.

Results

Sample Characteristics

Summary statistics regarding demographics, smoking history, nicotine dependence, and cessation success for the 70 participants included in the growth-curve models are summarized in Table 1.

Growth-Curve Modeling

Overall withdrawal. The best-fitting piecewise model for mean overall withdrawal score over the three weeks preceding and following a cessation attempt is summarized in Table 2 and depicted graphically in Figure 2.[2] The final model had significantly reduced model deviance relative to earlier, simpler models. In terms of elevation or severity of symptoms, the model suggested that the average score was 1.44 on a 5-point scale just prior to the quit date and 1.71 just after the quit date (due to a significant .27-point increase post-quit). The model did not detect significant linear growth in withdrawal ratings either pre-quit or post-quit. Nicotine patch condition was not significantly related to symptom severity or rate of change post-quit.

Negative affect. The final model of negative affective WSWS symptom ratings is summarized in Table 3 and depicted in Figure 2. There was a significant increase in negative affect ratings on the quit date, on average, but no significant linear change in ratings pre- or post-quit. Nicotine patch condition was not significantly related to symptom severity or rate of change post-quit.

Hunger. The best-fitting model of growth over time in hunger is summarized in Table 4 and depicted graphically in Figure 2. As shown in the table, on average, participants' hunger ratings did not change in a linear fashion either pre- or post-quit, or increase significantly on the quit date. Participants receiving active patches had significantly greater increases in hunger on the quit date than did those receiving placebo patches. Nicotine patch condition was not related to rate of change in hunger.

Craving. The best fitting model for craving is summarized in Table 5 and depicted graphically in Figure 2. Like negative affect and overall withdrawal, craving increased significantly after the quit date, but to a greater extent (over a one-half point increase occurred, on average). Craving ratings did not show significant linear change in either rating period, although those receiving active patches experienced a −.27-point decline in craving per week, on average. Patch condition was not significantly associated with symptom level or slope, however.

Interindividual Variability

There was significant variance in intercepts and linear growth coefficients in all of the final growth-curve models. The estimated

---

[2] Figure 2 was created by applying the estimated average intercept value and linear growth components to various time values. The estimated coefficients were derived from a completely random model. Time values were plugged into the following equation: $Y = \beta_0 + \beta_1$(Weeks before quit-date) $+ \beta_2$(Observation in post-quit period) $+ \beta_3$(Weeks after quit date). Patch condition was included as an explanatory variable for both β_2 and β_3 [e.g., $\beta_2 = \gamma_{20} + \gamma_{21}$(Patch condition) $+ U_2$]. Weeks before the quit date ranged from −3 to 0, and entered values were spaced at .25-week intervals. Weeks after the quit date ranged from 0 to 3 and were similarly spaced.

Table 1

Summary Statistics for 70 Participants With Sufficient Data to Fit Growth-Curve Models

Variable	Mean or %	SD
Female	51.4%	
White	90.0%	
African American	7.1%	
Other minority	2.9%	
Age	40.82	11.57
Cigarettes per day	24.49	10.07
Baseline CO	26.25	13.32
Age started smoking	17.64	4.38
Years smoked	23.18	11.28
Number of past serious quit attempts	2.89	3.23
Readiness to quit (1–7)	2.42	0.58
Fagerström Test of Nicotine Dependence score	5.79	2.00
Beck Depression Inventory-II score	4.79	5.40
Smoke within 30 minutes of waking	24.4%	
Active patch condition	77.1%	
Quit at three months post-quit	37.1%	

standard deviations and associated chi-square statistics, degrees of freedom, and p values for growth coefficients are shown in Tables 2–5 for each of the dependent variables. Coefficients were allowed to be random rather than fixed because allowing them to vary across individuals significantly improved the fit of the model without affecting the model's ability to converge. Results indicated that individuals differed significantly in terms of their estimated pre-quit and quit-day symptom levels and rates of change in symptoms both preceding and following a quit attempt for all the dependent variables modeled. Based on the standard deviations in the growth components derived from the craving growth-curve model (see Table 5), it appears as though the amount of estimated variance in the linear growth components for craving more than doubled after the quit date. Figure 3 depicts the average estimated linear growth in craving ratings pre-and post-quit and estimated slopes pre- and post-quit for all 70 participants in the study, controlling for nicotine patch condition (active vs. placebo) at the individual level during the post-quit period (i.e., both the quit-day jump estimates and post-quit linear change estimates are adjusted for patch condition). The figure suggests that individual smokers' paths diverge from the average more post-quit than pre-quit, beginning on the quit date. Individuals' slopes also appear steeper in the post-quit period than in the pre-quit period, suggesting that there is more systematic linear change at the individual level post-quit, compared with the relative stability estimated pre-quit. In sum, there is greater variability across individuals in craving trends following a quit attempt than preceding it, even when the influence of nicotine replacement condition is controlled. Similar increases in variance of growth estimates were observed for total WSWS scores and hunger (see Tables 2 and 4), but not for affective withdrawal symptoms (see Table 3).

Exploratory analyses were conducted to determine whether variance in growth terms across persons could be explained by gender or quit-day timing. In these analyses, we examined estimated t values for these variables as predictors of the intercept, quit-date jump, and pre- and post-quit linear growth coefficients. We were not able to explain variance in growth coefficients in any dependent variable, either pre- or postcessation, using either of these predictors (all t values < 1.96). No significant gender differences were found in this small sample.

Episodic Events

Separate models for each episodic event and symptom combination were fit to the pre-quit or post-quit data, controlling for symptom level and linear change.

Smoking. We found that reports of recent smoking occurring prior to the quit date were associated with significantly higher overall WSWS scores ($p < .001$), greater negative affect ratings ($p < .001$), and greater craving ratings ($p < .001$), relative to reports of recent abstinence. Precessation, hunger was not significantly related to recent smoking ($p < .11$, Figure 4A). After controlling for patch condition, reports of post-quit smoking (vs. no smoking) in the past 15 minutes were associated with significantly higher overall withdrawal ($p < .04$) and negative affect scores ($p < .03$), but unrelated to craving ($p < .17$) or hunger ratings ($p < .83$). The magnitude of the association between withdrawal and negative affect distress and recent smoking appeared to more than double after the quit date. Although the magnitude of the relation between recent smoking and craving nearly doubled from pre- to post-quit, the smoking coefficient for craving was not significant post-quit. This may be due to a roughly five-fold increase in the estimated standard error for the smoking coefficient from pre- to post-quit that likely resulted from the drop in the frequency of smoking reports post-quit. Being in the active patch condition was not significantly related to symptom associations with reports of recent smoking (all $p > .10$). Standardized coefficients for patch condition were nonsignificant and small in magnitude, ranging from −.17 to .05.

It is important to note that significant variance across individuals was observed in estimated smoking coefficients. Individuals were found to vary significantly in the extent of symptom displacement associated with recent smoking reports both before and after the quit date (all $p < .03$).

Smoker exposure. Participants were asked whether they had been in the presence of someone smoking in the 15 minutes prior to assessment. The average coefficients for smoker exposure pre- and post-quit date are shown in Figure 4B. On average, during the

Table 2

Estimated Fixed Effects and Variance Components for Final Growth Model of Total WSWS Withdrawal Scores

Fixed effects	β	SE	T	df	p	τ
Pre-quit intercept	1.44	.06	25.75	69	<.001	.21
Pre-quit linear slope	.01	.01	.56	69	<.58	.01
Increase in symptoms on quit date	.27	.09	2.82	69	<.005	.13
Active patch condition	.06	.11	.54	68	<.60	
Post-quit linear slope	.02	.05	.36	68	<.73	.04
Active patch condition	−.08	.06	−1.40	68	<.17	

Variance components	SD	Variance	χ^2	df	p
Pre-quit intercept	.46	.21	2029.84	67	<.001
Pre-quit linear slope	.11	.01	319.13	67	<.001
Increase in symptoms on quit date	.36	.13	662.49	66	<.001
Post-quit linear slope	.20	.04	1001.83	66	<.001

Note. WSWS = Wisconsin Smoking Withdrawal Scale; SE = Standard error; τ = Within-person residual variance. Covariance estimates not shown.
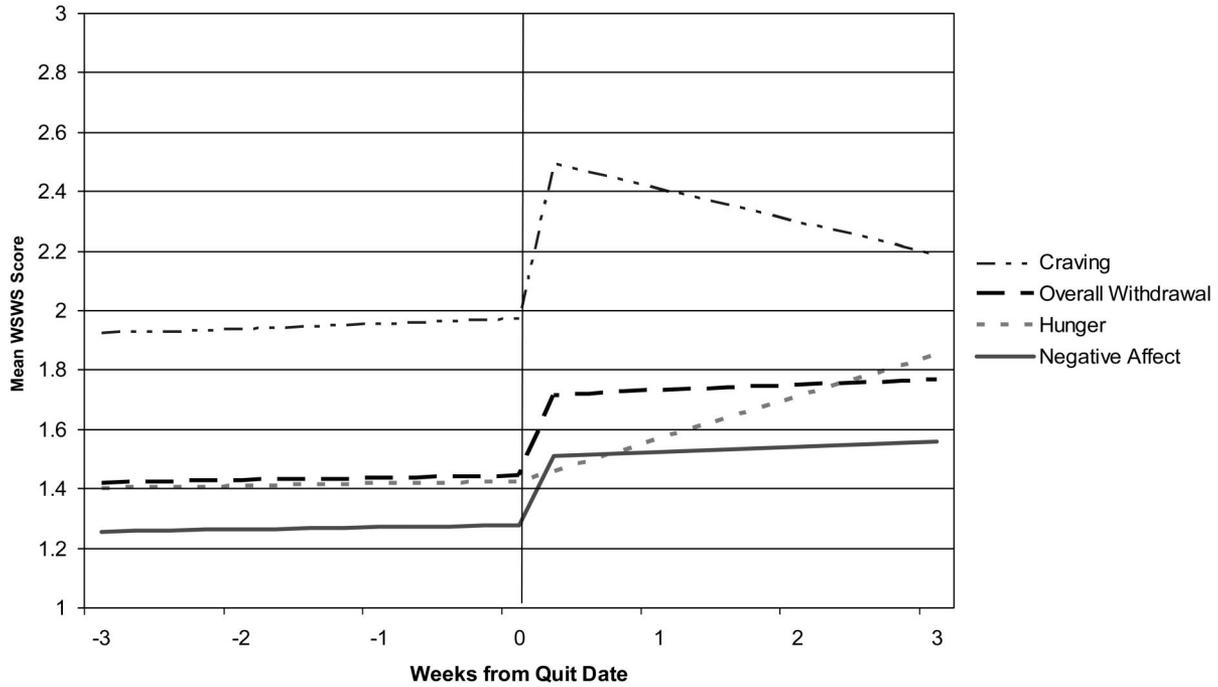
Figure 2. Best fitting growth-curves in symptom ratings. Growth in overall withdrawal score and particular symptom scores were estimated separately. Curves are based on the estimated levels and linear growth occurring in the pre- and post-quit periods in discontinuous piecewise multilevel models.

precessation period, such smoker exposure was associated with significantly higher overall withdrawal ($p < .005$), negative affect ($p < .008$), and craving ($p < .03$) ratings than when smoker exposure did not occur. Hunger ratings were not significantly associated with smoker exposure pre-quit ($p < .33$). After controlling for patch condition, postcessation overall withdrawal ($p < .008$), negative affect ($p < .02$), and craving ratings ($p < .005$) were, on average, significantly higher when recent smoker exposure was reported than when no exposure was reported. No sig-

nificant relationship was observed between smoker exposure and hunger scores postcessation ($p < .65$). As Figure 4B illustrates, smoker exposure was associated with an approximately four-fold increase in craving reports from precessation to postcessation.

The extent of the relation between smoker exposure and symptom ratings was found to vary significantly across individuals. Specifically, significant variance in smoking exposure coefficients was observed for overall withdrawal, craving, and hunger pre-quit, and overall withdrawal and craving coefficients varied signifi-

Table 3
Estimated Fixed Effects and Variance Components for Final Growth Model of Negative Affect

Fixed effects	β	SE	T	df	p	τ
Pre-quit intercept	1.28	.05	25.89	69	<.001	.16
Pre-quit linear slope	.01	.02	.39	69	<.70	.02
Increase in symptoms on quit date	.23	.08	2.78	69	<.007	.10
Active patch condition	−.04	.09	−.43	68	<.68	
Post-quit linear slope	.02	.04	.38	68	<.71	.03
Active patch condition	−.03	.05	−.65	68	<.52	

Variance components	SD	Variance	χ^2	df	p
Pre-quit intercept	.40	.16	1149.00	67	<.001
Pre-quit linear slope	.14	.02	322.67	67	<.001
Increase in symptoms on quit date	.32	.10	463.54	66	<.001
Post-quit linear slope	.17	.03	621.77	66	<.001

Note. SE = Standard error; τ = Within-person residual variance. Covariance estimates not shown.

Table 4
Estimated Fixed Effects and Variance Components for Final Growth Model of Hunger

Fixed effects	β	SE	T	df	p	τ
Pre-quit intercept	1.42	.08	16.95	69	<.001	.48
Pre-quit linear slope	.01	.02	.41	69	<.69	.02
Increase in symptoms on quit date	.00	.11	.009	68	<.99	.16
Active patch condition	.27	.12	2.19	68	<.03	
Post-quit linear slope	.14	.08	1.84	68	<.07	.09
Active patch condition	−.15	.09	−1.67	68	<.10	

Variance components	SD	Variance	χ^2	df	p
Pre-quit intercept	.69	.48	2431.53	67	<.001
Pre-quit linear slope	.13	.02	296.22	67	<.001
Increase in symptoms on quit date	.40	.16	398.85	66	<.001
Post-quit linear slope	.29	.09	1141.32	66	<.001

Note. SE = Standard error; τ = Within-person residual variance. Covariance estimates not shown.

Table 5

Estimated Fixed Effects and Variance Components for Final Growth Model of Craving

Fixed effects	β	SE	T	df	p	τ
Pre-quit intercept	1.97	.12	16.48	69	<.001	.97
Pre-quit linear slope	.02	.02	.69	69	<.50	.03
Increase in symptoms on quit date	.55	.25	2.21	68	<.03	1.04
Active patch condition	.22	.28	.79	68	<.43	
Post-quit linear slope	−.11	.11	−1.04	68	<.30	.18
Active patch condition	−.16	.12	−1.30	68	<.20	

Variance components	SD	Variance	χ²	df	p
Pre-quit intercept	.98	.97	2184.93	67	<.001
Pre-quit linear slope	.16	.03	244.02	67	<.001
Increase in symptoms on quit date	1.02	1.04	1145.74	66	<.001
Post-quit linear slope	.42	.18	1002.51	66	<.001

Note. SE = Standard error; τ = Within-person residual variance. Covariance estimates not shown.

cantly (all $p < .001$) across persons post-quit. Allowing smoker exposure coefficients for hunger to vary across individuals failed to improve model fit. No significant variance was observed in negative affect–smoker exposure coefficients post-quit.

Stressful events. Average coefficients for reported stressors occurring since the last recording are shown separately for the pre- and post-quit periods in Figure 4C. As this figure suggests, stress coefficients increased following the quit date, after controlling for patch condition. Both pre- and post-quit, stress was related to significantly higher overall withdrawal, negative affect, and craving scores (all $p < .005$). Hunger was not significantly related to stress in either time period (pre-quit $p < .55$, post-quit $p < .29$). Being in the active versus placebo patch condition was associated with significantly lower increases in negative affect ratings following reported stressful events ($\beta = -.38$, $SE = .18$, $t(66) = -2.08$, $p < .04$).

The average estimated coefficient for stressful event reports and symptom reports varied across participants. Significant variance in stress coefficients was observed both pre- and post-quit date in models for all dependent variables (all $p < .001$), except hunger.

Strong urges and temptations. Coefficients for recent temptations events pre- and postcessation are shown in Figure 4D. Correlations between craving ratings and temptation reports were not estimated, due to redundancy between the predictor and criterion variables. On average precessation, reporting a recent temptation was associated with significantly higher ratings of overall WSWS ($p < .001$), negative affect ($p < .001$), and hunger ratings ($p < .05$). On average, after controlling for patch condition post-quit, recent temptation reports were associated with higher overall withdrawal ($p < .001$) and negative affect ratings ($p < .001$), but not associated with hunger ratings ($p < .11$).

Once again, these findings regarding significant associations between a variable and symptom ratings are qualified by significant variance in the degree of the association across persons. Allowing the pre- and post-quit date urge and temptation coefficients to vary across individuals significantly improved the fit of
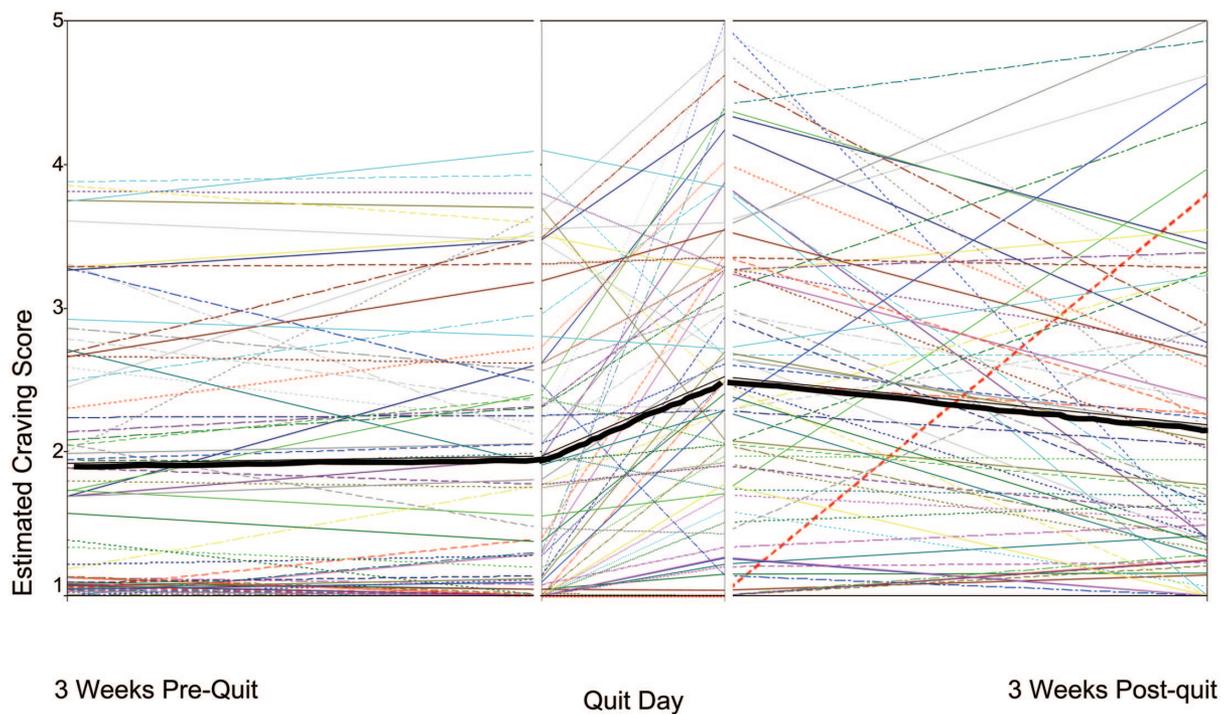

Figure 3. Individual estimated slopes in craving ratings over three weeks pre-quit, from just before to just after midnight on the quit date, and over three weeks post-quit. The heavy black line represents the mean trend in craving ratings.

the models of overall withdrawal, negative affect, and hunger. Each of the coefficients was found to vary significantly across persons (all $p < .001$).

Predicting Abstinence From Symptom Dimensions

In order to explore relations between withdrawal experiences pre- or post-quit and subsequent smoking behavior, we conducted logistic regressions predicting smoking abstinence three months post-quit from individual estimates of symptom elevation and trajectory. These analyses were conducted using data from the 70 participants who provided sufficient data for growth-curve modeling. Empirical Bayesian estimates of individual deviations from the average intercept and linear growth coefficients derived from the models depicted in Figure 2 were used as predictors in logistic regression models. The dependent variable was CO-verified seven-day point prevalence abstinence three months after the quit date. Patch condition effects were factored into the individual estimates

of growth. Patch condition was not entered as a control variable in the logistic regressions. Empirical Bayesian estimates were standardized prior to entry in the logistic regression model to facilitate interpretation of the odds ratios.

Logistic regressions predicting abstinence three-months post-quit, achieved by 37.1% of participants, revealed two significant predictors of abstinence. After controlling for pre-quit craving levels and growth both pre- and post-quit, the jump in craving ratings occurring on the quit day was significantly and inversely related to the likelihood of abstinence ($\beta = -.86$, $SE = .41$, Wald $= 4.40$, odds ratio (OR) $= .42$, 95% confidence interval (CI) $= .19-.95$, $p < .04$). In addition, pre-quit increases in negative affect were significantly and inversely related to abstinence ($\beta = -.69$, $SE = .34$, Wald $= 4.10$, OR $= .50$, 95% CI $= .26-.98$, $p < .05$), after controlling for negative affect severity just prior to the quit date. No other standardized estimates of symptom levels or slopes around the quit date were significantly related to abstinence in this small sample (all $p > .05$).
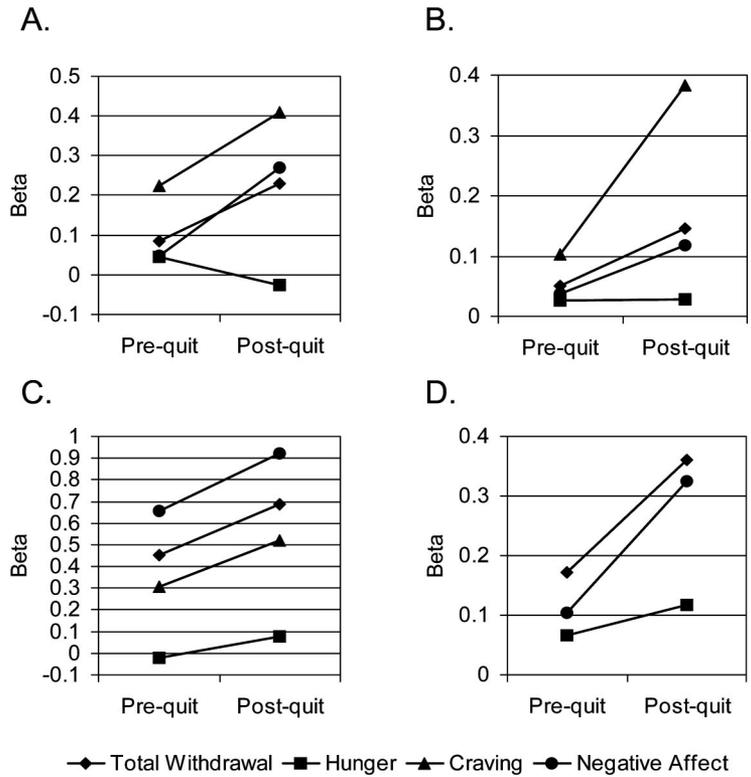

Figure 4. The *y*-axis depicts the magnitude of the average standardized coefficient derived from multivariate, multilevel models in which symptom elevation and linear growth were controlled. Episodic event coefficients were estimated separately in the pre-quit and post-quit periods. Statistical tests of the change in symptom–event association from pre- to post-quit were not conducted. The beta weights shown reflect the degree of symptom displacement associated with a one-unit change in the episodic event variable (i.e., from absent to present). The significance of each symptom–event coefficient is reported in the text. (A) Smoking: Coefficients for smoking in the past 15 minutes in models of overall withdrawal, hunger, craving, and affective withdrawal symptoms. (B) Smoker exposure: Coefficients for recent exposure to smoking behavior in models of overall withdrawal, hunger, craving, and affective withdrawal symptoms. (C) Stress: Coefficients for recent stressful events in models of overall withdrawal, hunger, craving, and affective withdrawal symptoms. (D) Urge/temptation: Coefficients for recent strong urges and temptations in models of overall withdrawal, hunger, and affective withdrawal symptoms.

Discussion

This research characterized life before and after an attempt to quit smoking, in terms of smokers' subjective experience of symptoms. This research describes symptoms as assessed in near real time in participants' natural environments. The data were analyzed in a novel, multidimensional manner encompassing the severity, trajectory, interindividual variability, and associations among episodic events and individual withdrawal symptoms in periods of ad-lib use and self-imposed restriction.

From a phenomenological perspective, results from this study showed that the average smoker's symptom experience changes for the worse immediately on the quit date, or even prior to the quit date. Negative changes associated with quitting were observed both in terms of symptom level and symptom–event associations. Some smokers reported escalating negative affect prior to the quit date, and such escalations were predictive of failure to stop smoking months later. Thus, for some smokers, anticipatory reactions to quitting may increase vulnerability to lapses or relapse. On average, participants reported higher overall withdrawal, including increased negative affect and craving, as soon as the quit day arrived. Individuals reporting larger jumps in craving on their quit date were more likely to fail in their cessation attempt. For the average smoker, symptom distress did not improve in the few weeks following the quit attempt. Few smokers appeared to follow this average pattern, however, particularly after the quit date when variability across individuals was observed to increase, relative to pre-quit levels. Symptom fluctuations associated with recent episodic event reports also increased following the quit date. Reports of recent smoking, exposure to someone smoking, or stress post-quit were all associated with worse craving post-quit versus pre-quit. Finally, comparisons of pre- and postcessation periods suggested that following the quit attempt, reports of events such as smoking, exposure to someone smoking, stress, and temptation events were associated with greater negative affect and withdrawal. Moreover, the magnitude of these symptom–event associations differed greatly from one person to the next.

One goal of this research was to determine with real-time data the extent to which patterns and variance in withdrawal symptoms, measured post-quit, reflect a change from pre-quit states. To examine this question, we quantified diverse symptom elevations both before and after quitting. Our data suggest that, on average, all symptoms except hunger increased significantly from just before, to just after, the beginning of the quit attempt. The significant jumps observed in these symptoms suggest that smokers' subjective distress level does in fact change in a manner that is contingent upon trying to quit. The immediacy of the increase in these symptoms may reflect, in part, psychological reactions to quitting rather than slowly emerging withdrawal processes that are typically thought to peak 3–10 days after a quit attempt (Cummings, Giovino, Jaen, & Emrich, 1985; Patten & Martin, 1996b; West, Hajek, & Belcher, 1987). This jump in distress may help to explain why the majority of cessation failures begin within 48 hours of the quit day (Kenford et al., 1994; Westman, Behm, Simel, & Rose, 1997).

We also sought to describe both pre-quit and post-quit trajectories in diverse symptoms. We did not find evidence of significant, systematic growth in symptoms in either time period, on average. These results suggest that participants' distress does not typically increase prior to quitting, but that it also does not typically diminish quickly in the weeks following the jump that occurs on the quit date.

Another target of the current research was the association between symptom reports and diverse episodic events. Our results suggest that the covariation between events and symptoms tends to increase following a quit attempt. In particular, participants tended to report feeling worse and craving cigarettes more when reporting recent smoking, observation of smoking, stressful events, and temptation events after versus before a quit attempt. Although hunger did not appear strongly related to any of these events, either pre- or post-quit, all the other symptoms (overall withdrawal, negative affect, and craving) appeared to be more tightly and positively linked with event reports following the quit attempt. These findings are important to the extent that they shed light on the aversiveness of withdrawal or the reduced quality of life occasioned by abstinence, and to the extent that they motivate relapse. In many cases, pre-quit associations between events and symptoms were weak and nonsignificant, whereas post-quit associations were of moderate or greater strength, with some associations (e.g., between reports of craving and seeing someone smoke) increasing four-fold from pre- to post-quit. Interestingly, participants tended to report feeling worse (in terms of negative affect) when reporting a recent urge or temptation event after versus before a quit attempt. This suggests that smokers may experience negative affective reactions to fighting urges during a quit attempt. This increasing association is also consistent with the hypothesis that deprivation enhances the coherence of urge and negative affect responses (Baker, Morse, & Sherman, 1987; Sayette, Martin, Hull, Wertz, & Perrott, 2003). Such interpretations are speculative at this point, however, as the results of increasing covariation between symptom reports and recent event reporting may reflect reciprocal relations (e.g., a person is more likely to report a recent episodic stressor when feeling upset).

With regard to smoking, some past research has found that ad-lib smoking is associated with feeling stress (Shapiro et al., 2002), whereas other research suggests that negative affect is not tightly linked with affect prior to smoking (Shiffman et al., 2002). Our data suggest that, prior to quitting, smoking reports are not tightly related to affect, but that this changes after the quit attempt. After the quit date, negative affect and overall withdrawal ratings were found to be significantly higher when recent smoking was reported than not, and this increase was nearly double that observed pre-quit. This finding is consistent with previous research showing that symptoms assessed via daily diary tended to be higher, on average, on days when smoking was reported post-quit than when no smoking was reported, depending on the amount smoked (Piasecki et al., 2003a). Of course, the increased relation between smoking and affect observed post-quit could be due to multiple factors. For example, the stronger relation could be due to disappointment and guilt caused by the lapse or relapse. Alternatively, postcessation smoking may be more likely to be discriminated on stressful circumstances than is precessation smoking. In this case, the postsmoking negative affect would be secondary to the stress that occasioned the smoking (e.g., Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Shiffman et al., 2002).

Interindividual heterogeneity in symptom experiences was a central target of the current research. Replicating previous research (Piasecki et al., 2003a), we found evidence, using real-time data, of significant heterogeneity in symptom elevation, trajectories, and associations between symptoms and environmental event (e.g.,

seeing someone smoke or experiencing a stressful event). These results suggest that patterns of symptom–event covariation, as well as patterns of symptom growth, differ across individuals in ways that have not been appreciated before. Even after taking nicotine patch effects into account, significant heterogeneity in symptom–event and symptom growth relations remained. In addition, we detected a near doubling of the estimated variance in symptom trajectories post-quit, relative to pre-quit (as reflected in Tables 2–5 and Figure 3). Coupled with the significant variability observed in the magnitude of the jump in symptoms occurring on the quit date, these data show that smokers' paths diverge more after than before a quit attempt. Thus, drug removal was associated not only with reliable changes in levels of withdrawal and covariation with event reports, but also with a substantial increase in variation from one person to the next on such measures. Whether the apparent increase in variability post-quit reflects the unmasking of a nicotine dependence- or withdrawal-related phenomenon, or the removal of a homogenizing acute drug effect, cannot be determined by our data.

Another goal of this research was to examine patterns in particular constituents of the withdrawal syndrome, such as negative affect, hunger, and craving. Our results show potentially important differences among these symptoms. Specific symptoms appear to follow different patterns over time and to share different relations with environmental events. For example, whereas negative affect predominates in association with stressful events, craving appears to predominate in association with smoking-related events. This raises questions about the meaningfulness of characterizing broad amalgams of withdrawal symptoms (i.e., using summary scores for diverse symptoms). In particular, craving appears to increase to a greater degree than other symptoms immediately after the quit date. These results suggest that craving, which has been thought to be insensitive to deprivation effects in the past (Hughes, 1992; Swan et al., 1996), is, on the contrary, quite sensitive to trying to quit. Craving also seemed to differ from other symptoms in terms of covariation with episodic events. Craving was more tightly linked with reports of recent smoking than any other symptom and was the symptom most strongly linked with reporting seeing someone smoke post-quit. Recent research has demonstrated that craving or urges to smoke are tightly linked to subsequent smoking during periods of ad-lib smoking, in ways that other symptoms are not (Shapiro et al., 2002; Shiffman et al., 2002; Shiffman et al., 2004). In addition, real-time reports of urges (both tonic urge levels and increased urge upon waking) have been shown to predict subsequent lapses during quit attempts (Shiffman et al., 1997). Our results corroborate this link between urges and relapse; in our sample, greater increases in craving on the quit date were predictive of failure to achieve later abstinence. Taken together, these results suggest that urges to smoke may differ from other symptoms in the withdrawal syndrome in important ways and may play a unique role in continuing smoking and relapse.

An additional objective of this research was to explore relations between symptom experiences as assessed in real-time and subsequent smoking behavior. Despite a lack of power to detect between-subjects effects in our small sample, the jump in craving on the quit day and the rate of increase in negative affect prior to the quit attempt were found to predict biochemically confirmed point-prevalence abstinence three months post-quit. In this small sample, no other predictors of abstinence were detected among the post-quit symptom dimension variables. Some of these odds ratios

were substantial, however, suggesting that low power to detect between-subjects effects may be responsible for these null effects. The significant results obtained from these exploratory analyses suggest that anticipation of quitting and immediate reactions to quitting have motivational significance.

Finally, we explored nicotine patch effects on symptom patterns and covariation with episodic events. One significant patch effect was observed; those participants who received active nicotine patches reported smaller increases in negative affect in conjunction with stressful events than did those receiving placebo patches. These results suggest that nicotine may suppress negative affective reactions to stressors, as others have posited (Kassel, Stroud, & Paronis, 2003), although our data would support reciprocal relations between affect and stress reporting as well. It may be that nicotine replacement pharmacotherapy produces its beneficial effects, in part, by mitigating stress reactivity. Standardized coefficients for the nicotine patch condition in other models explaining variance in symptom patterns or symptom–event relations were small (ranging from .03 to .22). As such, these effects appear small and are likely not of clinical significance.

Limitations

Conclusions based on this research must be tempered by recognition of important study limitations. First and foremost, the sample of smokers was small. Therefore, negative results in this study should be interpreted with caution. Although the repeated measures design and intensive assessment yielded acceptable power for detecting within-subjects effects (e.g., trends over time), the study was underpowered to test between-subjects effects (e.g., predicting abstinence).

A second important limitation in this study relates to interpretation of episodic event coefficients. Although participants were instructed to use a different time frame for some questions, reports of smoking and all other events were gathered contemporaneously with symptom reports. Although we would like to infer that prior smoking affected symptom reports, it is possible that recollection of prior smoking was affected by current symptoms (e.g., affect). Alternatively, it is possible that recollection of prior smoking influenced symptom ratings (i.e., that respondents rated symptoms in a way that helped them explain prior smoking). Finally, the likely autocorrelation of symptom reports over time complicates interpretation, as symptom reports at a given time are likely to be a function of symptom reports at the most recent assessment as well as any intervening episodic events or behaviors (e.g., smoking).

Another shortcoming of this study is its unbalanced and mixed design. Only 27% of participants were assigned to a placebo condition, and one half of the sample received open-label active nicotine patches. Exposure to active patches was treated as a between-subjects explanatory variable in all models in order to control for differences that might be associated with patch condition. We did not control for participants' awareness of their active patch status, however. It is possible that patch effects might differ among those who knew versus did not know that they were wearing active patches.

Finally, assessment reactivity may have colored the results. For instance, participants might have experienced high levels of frustration due to the demands of the ecological momentary assessment system. In order to assess this indirectly, we attempted to

explain variance in multilevel model parameters by timing of the quit date. Analyses did not support the hypothesis that parameter estimates differed among those who completed four versus seven weeks of assessment pre-quit.

Conclusions

This study yielded new information about how a smoking cessation attempt changes smokers' lives. First, smokers' withdrawal symptom ratings increased significantly over the first hours after the quit date. Second, attempting to quit increased interindividual variability in levels and trajectories of withdrawal symptoms including craving and hunger. Third, symptom covariation with smoking and other episodic events appeared to increase from pre-quit to post-quit. For example, following a quit attempt, reports of observing smoking were associated with greater spikes in craving than occurred pre-quit. Taken together, these results highlight the value of research that examines both episodic and emergent processes that occur in a smoking cessation attempt and that may determine its success. In the future, new theories must address the large individual differences seen in emergent processes and reactions to environmental events and clarify how these affect relapse likelihood (e.g., Piasecki, Fiore, McCarthy, & Baker, 2002; Witkiewitz & Marlatt, 2004).

References

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Baker, T. B., Morse, E., & Sherman, J. E. (1987). The motivation to use drugs: A psychobiological analysis of urges. In C. Rivers (Ed.), *Nebraska symposium on motivation* (pp. 257–323). Lincoln: University of Nebraska Press.

Baker, T. B., Piper, M. E., McCarthy, D. E., Majeskie, M. R., & Fiore, M. C. (2004). Addiction motivation reformulated: An affective processing model of negative reinforcement. *Psychological Review, 111,* 33–51.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory–II.* San Antonio, TX: The Psychological Corporation.

Bryk, A., & Raudenbush, S. W. (1992). *Hierarchical linear models for social and behavioral research: Applications and data analysis methods.* Newbury Park, CA: Sage.

Centers for Disease Control (2002). Annual smoking-attributable mortality, years of potential life lost, and economic costs—United States, 1995–1999. *Morbidity and Mortality Weekly Report, 51,* 300–303.

Covey, L. S., Glassman, A. H., & Stetner, F. (1990). Depression and depressive symptoms in smoking cessation. *Comprehensive Psychiatry, 31,* 350–354.

Cummings, K. M., Giovino, G., Jaen, C. R., & Emrich, L. J. (1985). Reports of smoking withdrawal symptoms over a 21-day period of abstinence. *Addictive Behaviors, 10,* 373–381.

Francis, D. J., Fletcher, J. M., Stuebing, K. K., Davidson, K. C., Thompson, N. M. (1991). Analysis of change: Modeling individual growth. *Journal of Consulting and Clinical Psychology, 59,* 27–37.

Gilbert, D. G., Sharpe, J. P., Ramanaiah, N. V., Detwiler, F. R., & Anderson, A. E. (2000). Development of a situation X trait adaptive response (STAR) model-based smoking motivation questionnaire. *Personality and Individual Differences, 29,* 65–84.

Hall, S. M., Havassy, B. E., & Wasserman, D. A. (1990). Commitment to abstinence and acute stress in relapse to alcohol, opiates, and nicotine. *Journal of Consulting and Clinical Psychology, 58,* 175–181.

Hammersley, R. (1994). A digest of memory phenomena for addiction research. *Addiction, 89,* 283–293.

Heatherton, T. F., Kozlowski, L. T., Frecker, R. C., & Fagerström, K. O. (1991). The Fagerström Test for Nicotine Dependence: A revision of the Fagerström Tolerance Questionnaire. *British Journal of Addiction, 86,* 1119–1127.

Hughes, J. R. (1992). Tobacco withdrawal in self-quitters. *Journal of Consulting and Clinical Psychology, 60,* 689–697.

Hughes, J. R., & Hatsukami, D. K. (1986). Signs and symptoms of tobacco withdrawal. *Archives of General Psychiatry, 43,* 289–294.

Hughes, J. R., Higgins, S. T., & Hatsukami, D. K. (1990). Effects of abstinence from tobacco: A critical review. In L. T. Kozlowski, H. Annis, H. D. Cappell, F. Glaser, M. Goodstadt, Y. Israel, H. Kalant, E. M. Sellers, & J. Vingilis (Eds.), *Research advances in alcohol and drug problems* (Vol. 10, pp. 317–398). New York: Plenum Press.

Hutcheson, D. M., Everitt, B. J., Robbins, T. W., & Dickinson, A. (2001). The role of withdrawal in heroin addiction: Enhances reward or promotes avoidance? *Nature Neuroscience, 4,* 934–947.

Jaffe, J. H. (1992). Current concepts of addiction. In C. P. O'Brien & J. Jaffe (Eds.), *Addictive States* (pp. 1–21). New York: Raven Press.

Kassel, J. D., Stroud, L. R., & Paronis, C. A. (2003). Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychological Bulletin, 129,* 270–304.

Kenford, S. L., Fiore, M. C., Jorenby, D. E., Smith, S. S., Wetter, D., & Baker, T. B. (1994). Predicting smoking cessation: Who will quit with and without the nicotine patch. *Journal of the American Medical Association, 271,* 589–594.

Kenford, S. L., Smith, S. S., Wetter, D. W., Jorenby, D. E., Fiore, M. C., & Baker, T. B. (2002). Predicting relapse back to smoking: Contrasting affective and physical models of dependence. *Journal of Consulting and Clinical Psychology, 70,* 216–227.

Larsen, R. J., & Diener, E. (1987). Affect intensity as an individual difference characteristic: A review. *Journal of Research in Personality, 21,* 1–39.

Lyvers, M. (1998). Drug addiction as a physical disease: The role of physical dependence and other chronic drug-induced neurophysiological changes in compulsive drug self-administration. *Experimental and Clinical Psychopharmacology, 6,* 107–125.

Patten, C. A., & Martin, J. E. (1996a). Does nicotine withdrawal affect smoking cessation? Clinical and theoretical issues. *Annals of Behavioral Medicine, 18,* 190–200.

Patten, C. A., & Martin, J. E. (1996b). Measuring tobacco withdrawal: A review of self-report questionnaires. *Journal of Substance Abuse, 8,* 93–113.

Pendragon Software Corporation (1999). Pendragon Forms (Version 3.0) [Computer software]. Buffalo Grove, IL: Author.

Piasecki, T. M., Fiore, M. C., & Baker, T. B. (1998). Profiles in discouragement: Two studies of variability in the time course of smoking withdrawal symptoms. *Journal of Abnormal Psychology, 107,* 238–251.

Piasecki, T. M., Fiore, M. C., McCarthy, D. E., & Baker, T. B. (2002). Have we lost our way? The need for dynamic formulations of smoking relapse proneness. *Addiction, 97,* 1093–1108.

Piasecki, T. M, Jorenby, D. E., Smith, S. S., Fiore, M. C., & Baker, T. B. (2003a). Smoking withdrawal dynamics: I. Abstinence distress in lapsers and abstainers. *Journal of Abnormal Psychology, 112,* 3–13.

Piasecki, T. M, Jorenby, D. E., Smith, S. S., Fiore, M. C., & Baker, T. B. (2003b). Smoking withdrawal dynamics: II. Improved tests of withdrawal-relapse relations. *Journal of Abnormal Psychology, 112,* 14–27.

Piasecki, T. M, Jorenby, D. E., Smith, S. S., Fiore, M. C., & Baker, T. B. (2003c). Smoking withdrawal dynamics: III. Correlates of withdrawal heterogeneity. *Experimental and Clinical Psychopharmacology, 11,* 276–285.

Piasecki, T. M., Niaura, R., Shadel, W. G., Abrams, D., Goldstein, M., Fiore, M. C., & Baker, T. B. (2000). Smoking withdrawal dynamics in unaided quitters. *Journal of Abnormal Psychology, 109,* 74–86.

Raudenbush, S., Bryk, A., Cheong, Y. F., & Congdon, R. (2001). *HLM 5:*

Hierarchical linear and nonlinear modeling. Lincolnwood, IL: Scientific Software International.

Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: An incentive sensitization theory of addiction. *Brain Research Reviews, 18,* 247–291.

Robinson, T. E., & Berridge, K. C. (2003). Addiction. *Annual Review of Psychology, 54,* 10.1–10.29.

Sayette, M. A., Martin, C. S., Hull, J. G., Wertz, J. M., & Perrott, M. A. (2003). The effects of nicotine deprivation on craving response covariation in smokers. *Journal of Abnormal Psychology, 112,* 110–118.

Shapiro, D., Jamner, L. D., Davydov, D. M., & James, P. (2002). Situations and moods associated with smoking in everyday life. *Psychology of Addictive Behaviors, 16,* 342–345.

Shiffman, S. (1982). Relapse following smoking cessation: A situational analysis. *Journal of Consulting and Clinical Psychology, 50,* 71–86.

Shiffman, S. (1986). A cluster-analytic classification of smoking relapse episodes. *Addictive Behaviors, 11,* 295–307.

Shiffman, S., Engberg, J. B., Paty, J. A., Perz, W. G., Gnys, M., Kassel, J. D., & Hickcox, M. (1997). A day at a time: Predicting smoking lapse from daily urge. *Journal of Abnormal Psychology, 106,* 104–116.

Shiffman, S., Gwaltney, C. J., Balabanis, M. H., Liu, K. S., Paty, J. A., Kassel, J. D., Hickcox, M., & Gnys, M. (2002). Immediate antecedents of cigarettes smoking: An analysis from ecological momentary assessment. *Journal of Abnormal Psychology, 111,* 531–545.

Shiffman, S., Paty, J. A., Gnys, M., Kassel, J. A., & Hickcox, M. (1996). First lapses to smoking: Within subjects analysis of real time reports. *Journal of Consulting and Clinical Psychology, 64,* 366–379.

Shiffman, S., Paty, J. A., Gwaltney, C. J., & Dang, Q. (2004). Immediate antecedents of cigarette smoking: An analysis of unrestricted smoking patterns. *Journal of Abnormal Psychology, 113,* 166–171.

Siegel, S. (1983). Classical conditioning, drug tolerance, and drug dependence. In R. G. Smart, F. B. Glaser, Y. Israel, H. Kalant, R. E. Popham, & W. Schmidt (Eds.), *Research advances in alcohol and drug problems* (Vol. 7). New York: Plenum.

Snijders, T., & Bosker, R. (1999). *Multilevel analysis: An introduction to basic and advanced multilevel modeling.* Thousand Oaks, CA: Sage.

Solomon, R. L. (1977). An opponent-process theory of acquired motivation: The affective dynamics of addiction. In J. D. Maser & M. E. P. Seligman (Eds.), *Psychopathology: Experimental models* (pp. 66–103). San Francisco: W. H. Freeman.

Solomon, R. L., & Corbit, J. D. (1974). An opponent-process theory of motivation: 1. Temporal dynamics of affect. *Psychological Review, 81,* 119–145.

Spitzer, R. L., Williams, J. B., Kroenke, K., Linzer, M., deGruy, F. V., Hahn, S. R., et al. (1994). Utility of a new procedure for diagnosing mental disorders in primary care: The PRIME MD 1000 study. *The Journal of the American Medical Association, 272,* 1749–1756.

Stewart, J., de Wit, H., & Eikelboom, R. (1984). Role of unconditioned and conditioned drug effects in the self-administration of opiates and stimulants. *Psychological Review, 91,* 251–268.

Stewart, J., & Wise, R. A. (1992). Reinstatement of heroin self-administration habits: Morphine prompts and naltrexone discourages renewed responding after extinction. *Psychopharmacology, 108,* 79–84.

Stone, A. A., & Shiffman, S. (1994). Ecological momentary assessment (EMA) in behavioral medicine. *Annals of Behavioral Medicine, 16,* 199–202.

Stone, A. A., Shiffman, S., Schwartz, J. E., Broderick, J. E., & Hufford, M. R. (2002). Patient non-compliance with paper diaries. *British Medical Journal, 324,* 1193–1194.

Swan, G. E., Ward, M. M., & Jack, L. M. (1996). Abstinence effects as predictors of 28-day relapse in smokers. *Addictive Behaviors, 21,* 481–490.

Tate, J. C., Pomerleau, O. F., & Pomerleau, C. S. (1993). Temporal stability and within-subject consistency of nicotine withdrawal symptoms. *Journal of Substance Abuse, 5,* 353–363.

Tiffany, S. (1990). A cognitive model of drug urges and drug-use behavior: Role of automatic and nonautomatic processes. *Psychological Review, 97,* 147–168.

U.S. Department of Health and Human Services. (1990). *The health benefits of smoking cessation: A report of the surgeon general* (DHHS Publication CDC No. 90–8416). Atlanta, GA: Author.

Welsch, S. K., Smith, S. S., Wetter, D. W., Jorenby, D. E., Fiore, M. C., & Baker, T. B. (1999). Development and validation of the Wisconsin Smoking Withdrawal Scale. *Experimental and Clinical Psychopharmacology, 4,* 354–361.

West, R. J., Hajek, P., & Belcher, M. (1987). Time course of cigarette withdrawal symptoms during four weeks of treatment with nicotine chewing gum. *Addictive Behaviors, 12,* 199–203.

West, R. J., Hajek, P., & Belcher, M. (1989). Severity of withdrawal symptoms as a predictor of outcome of an attempt to quit smoking. *Psychological Medicine, 19,* 981–985.

Westman, E. C., Behm, F. M., Simel, D. L., & Rose, J. E. (1997). Smoking behavior on the first day of a quit attempt predicts long-term abstinence. *Archives of Internal Medicine, 157,* 335–340.

Wetter, D. W., Smith, S. S., Kenford, S. L., Jorenby, D. E., Fiore, M. C., Hurt, R. D., et al. (1994). Smoking outcome expectancies: Factor structure, predictive validity, and discriminant validity. *Journal of Abnormal Psychology, 103,* 801–811.

Wikler, A. (1948). Recent progress in research on the neurophysiological basis of morphine addiction. *American Journal of Psychiatry, 105,* 329–338.

Wikler, A. (1977). The search for the psyche in drug dependence. *The Journal of Nervous and Mental Disease, 165,* 29–40.

Wikler, A. (1980). *Opioid dependence: Mechanisms and treatment.* New York: Plenum.

Witkiewitz, K., & Marlatt, G. A. (2004). Relapse prevention for alcohol and drug problems. *American Psychologist, 59,* 224–235.