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Smoking Withdrawal Dynamics: III. Correlates of Withdrawal Heterogeneity

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Five parameters of postcessation smoking withdrawal variability derived from clinical data (T. M. Piasecki, D. E. Jorenby, S. S. Smith, M. C. Fiore, & T. B. Baker, 2003a, 2003b) were predicted from baseline measures and pharmacotherapy assignment. Smokers who were more dependent, older, and high in negative affect reported more severe withdrawal. Women, heavier smokers, and those with a history of depression reported more variable symptoms. Smokers treated with nicotine patch, bupropion, or both reported less severe withdrawal than did those given placebo, but medication did not affect the slope of symptoms over time, day-to-day variability of symptoms, or the size of acute changes in symptoms associated with lapses to smoking. Prior research has shown that these symptom facets predict later relapse; thus, current pharmacotherapies may aid cessation by diminishing withdrawal severity, but they do not affect all clinically important aspects of withdrawal.

The withdrawal symptoms that follow discontinuation of tobacco use have clear clinical significance. For instance, they may index the net subjective costs of quitting, reflect the magnitude of physical dependence, or predict the future likelihood of relapse (e.g., Kenford et al., 2002; Piasecki, Fiore, & Baker, 1998). Despite the clinical importance of withdrawal symptoms, researchers have achieved little insight into their determinants.

Although withdrawal symptoms are, no doubt, influenced by tobacco dependence and smoking history, they may also reflect a host of other influences. Mood symptoms, which form the core of the smoking withdrawal syndrome (e.g., Gilbert, Gilbert, & Schulz, 1998; Hughes, 1992) are variable in nonsmokers (e.g., Larsen & Kasimatis, 1990) and in smokers who are not trying to quit (Gilbert, Gilbert, & Schulz, 1998; Parrott, 1995). It follows that any pharmacologic modulation of withdrawal symptoms must be superimposed on the effects of other variables that influence affective responses. Indeed, recent research has revealed that individual differences generally thought to be independent of drug use per se, such as neuroticism (Madden et al., 1997) and psychiatric cofactors (Pomerleau, Marks, & Pomerleau, 2000), are related to postcessation symptoms. Thus, there is evidence that withdrawal symptoms are related to diverse nondrug individual-difference variables, but this evidence is neither complete nor consistent.

Characterizing the individual differences associated with withdrawal symptoms is vital because this information may eventually help researchers understand withdrawal's causal determinants, help explain why some treatments are more effective for some individuals than for others, and reveal why some populations (e.g., women, those with depression) are especially likely to relapse (Glassman et al., 1988; Perkins 1996; Wetter et al., 1999).

Recent research has revealed that smoking withdrawal symptoms are highly variable, both across persons and over time (Piasecki et al., 1998). Ideally, research designed to identify the correlates of withdrawal symptomatology should take this variability into account. In a companion article (Piasecki, Jorenby, Smith, Fiore, & Baker, 2003a), we used repeated measures of withdrawal from a large clinical trial to describe a new statistical approach to representing individual differences in symptom dynamics based

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on growth models computed using hierarchical linear modeling (HLM; Bryk & Raudenbush, 1992). The growth modeling produces continuously scaled parameters of symptomatic distress that are interpretable a priori and that may have unique relations with nonwithdrawal individual-difference variables. These parameters included an *intercept value* tapping the mean severity of symptoms across an 8-week postcessation period, a *linear slope* measure assessing the direction and rate of linear symptomatic change across 8 weeks of the cessation attempt, a *quadratic trend coefficient* that permits curvature in the predicted withdrawal function, and a *cigarette coefficient* (for lapsers only) measuring typical acute symptom changes associated with lapses to smoking. These four model parameters were supplemented with a *volatility index* assessing the degree of symptomatic scatter, or day-to-day variability, around the predicted withdrawal function. Figure 1 illustrates the meaning of the derived parameters by depicting 8 weeks of raw symptom and smoking data from 2 participants, overlaid with the growth functions fitted to the participants' data. Prior research attests to the clinical importance of these withdrawal parameters: higher intercepts, weak negative or positive linear slopes, greater volatility, and negative cigarette coefficients were found to predict relapse at 6 months postcessation (Piasecki, Jorenby, Smith, Fiore, & Baker, 2003b).

In this article, we use baseline data from the same trial in which these five parameters were derived to explore the correlates of postcessation symptom dynamics. The analyses are exploratory in nature, but we posited two broad hypotheses. First, on the basis of prior research and theory (e.g., Baker, Morse, & Sherman, 1987; Covey, Glassman, & Stetner, 1990; Gilbert, Gilbert, & Schulz, 1998; Gilbert, McClernon, et al., 1998; Kenford et al., 2002; Pomerleau et al., 2000) and the nature of subjective symptoms, we expected that affect-relevant variables, such as baseline affect and history of affective disorder, would be related to the postcessation withdrawal parameters. Second, because symptom scores are also influenced by pharmacologic factors, we expected nicotine dependence, smoking history variables, and pharmacotherapy to be related to the withdrawal parameters.

Method

Parent Trial Participants and Design

Data were drawn from a four-center, double-blind, fully factorial clinical trial evaluating the 21-mg nicotine patch and bupropion for smoking cessation (Jorenby et al., 1999). A total of 893 smokers met inclusion and exclusion criteria (see Piasecki et al., 2003a) and were randomly assigned to one of four treatment groups, with preferential assignment to treatments involving active medication: placebo patch plus placebo pill (n = 160), nicotine patch plus placebo pill (n = 244), bupropion plus placebo patch (n = 244), bupropion plus nicotine patch (n = 245). Fifty-two percent (n = 467) of the participants were female. Smokers enrolled in the trial were similar to those of other clinical trial samples (Hughes, Giovino, Klevens, & Fiore, 1997).

The trial consisted of three phases: a 1-week baseline phase, a 9-week treatment phase, and a follow-up phase that extended to 1 year after the initiation of therapy. Participants were screened, completed a battery of self-report measures, and received brief individual counseling during the baseline phase. Participants began taking assigned pills during the 1st week of the treatment phase and continued to take them for the remainder of the 9 weeks. For smokers assigned to active bupropion, this translated into 3 days of 150 mg bupropion per day, followed by 8.5 weeks of bupropion at 150 mg b.i.d. Placebo bupropion participants took the same number of equivalent-appearing tablets. Placebo or active patch therapy began for all participants on the 8th day of the treatment phase (Day 8 served as the quit date for all participants) and continued for the remainder of the treatment phase. Nicotine patch therapy was tapered; active patch participants wore 21-mg patches for the first 6 weeks of the quit attempt, stepped down to 14-mg patches for 1 week, and stepped down again to 7-mg patches for an additional week before discontinuing patch use. Participants reported to the study center once per week during the treatment phase to complete assessments and received brief individual counseling (<10 min).

Measures

Withdrawal. Smoking withdrawal symptoms were assessed with a daily diary that contained a modification of the Minnesota Nicotine Withdrawal Scale (MNWS; Hughes & Hatsukami, 1986). Participants rated nine symptoms on a scale ranging from 0 to 4 (0 = *absent*, 1 = *slight*, 2 = *mild*, 3 = *moderate*, 4 = *severe*): craving for cigarettes, depressed mood, difficulty falling asleep, awakening at night, irritability/frustration/anger, anxiety, difficulty concentrating, restlessness, and increased appetite. Participants were instructed to rate their withdrawal symptoms just before going to bed each night so that they could reflect on the entire day's experience when providing their responses. Participants completed the diaries daily during the baseline and treatment phases.

Daily data from the first 8 postcessation weeks (the period in which active pharmacotherapy was provided) were used as dependent variables in growth models. In all growth models, the raw sum of item scores was used as the repeated dependent measure. We modeled total withdrawal scores rather than individual symptoms because the MNWS uses a single item to measure each symptom domain; modeling each symptom separately would require relying on outcome variables of questionable reliability (Nunnally & Bernstein, 1994). Although some individual symptoms might have unique correlates, analysis of the total score was deemed reasonable because a series of correlational analyses consistently revealed positive correlation manifolds among all symptoms when either ratings from a single day or rates of linear change in each symptom over time were considered. Coefficient alpha for the MNWS was found to be greater than .80 in the postcessation ratings, and there was no item that consistently improved alpha when deleted from the scale. Thus, the data suggested that the MNWS total score taps a reasonably syndromal latent construct.

Postcessation smoking. Participants were also asked to record in the daily diaries the number of cigarettes smoked each day during the study (or to enter a zero on days they were abstinent).

Lapser–abstainer split. Some analyses required splitting the sample according to the occurrence of postcessation smoking. This split was determined using data from daily cigarette tallies from the first 8 weeks of the cessation attempt. Eight hundred ninety-three participants attended a screening session and were randomized to a treatment group. Four of these individuals did not return for additional sessions. Of the remaining 889 participants, 418 provided a complete series of cigarette tallies, with 194 of these reporting complete abstinence and 224 reporting at least one smoking event. Of the 471 participants with one or more missing

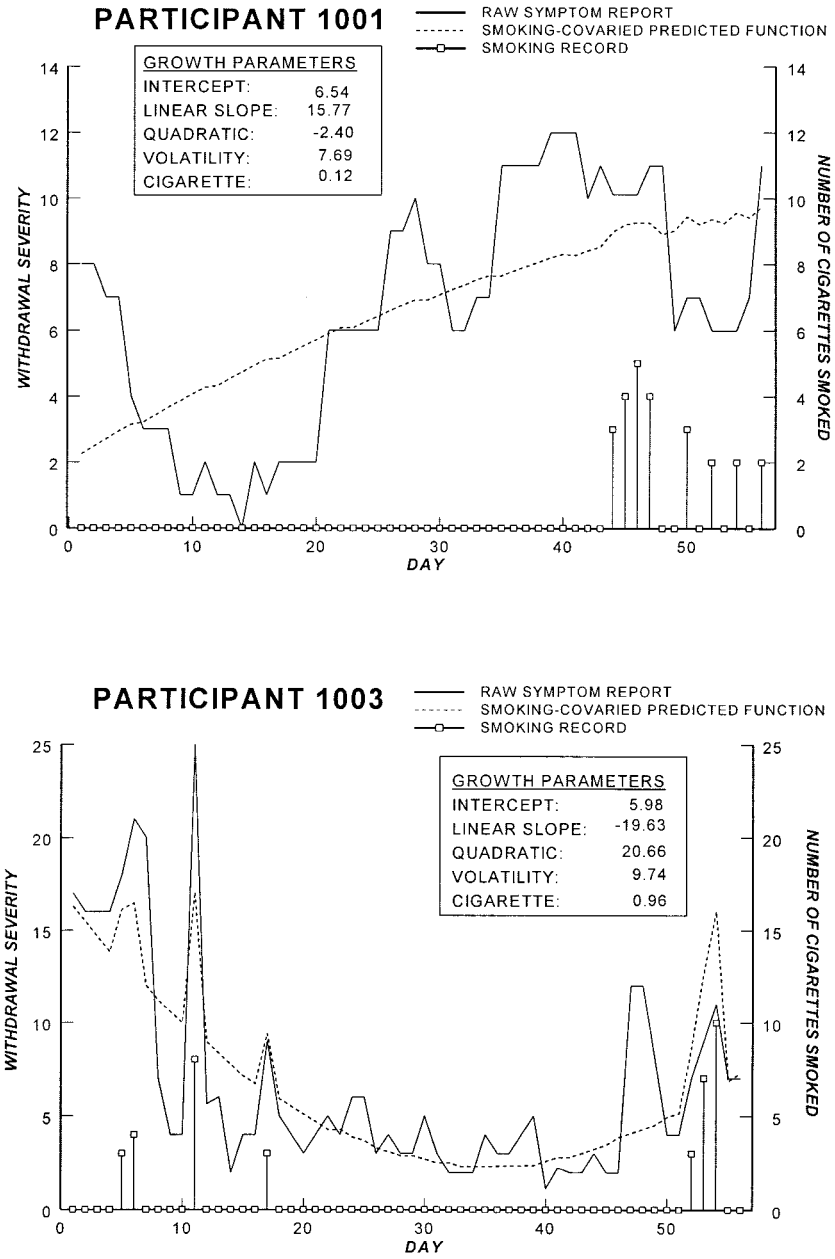

Figure 1. Predicted withdrawal growth functions, raw withdrawal score profiles, smoking tallies, and estimated withdrawal parameters over 8 weeks of the quit attempt for 2 participants in the parent trial. *Intercept* refers to the mean level of symptoms across the 8-week postquit period. *Linear slope* captures the direction and magnitude of linear change across the postquit period, with negative slopes indicating that symptoms decrease over time and positive slopes indicating that symptoms tend to grow worse over time. *Quadratic* indexes the extent to which the participant's withdrawal profile is U-shaped, with positive scores indicating a concave profile and negative scores indicating a convex profile. *Volatility* indexes the scatter of observed scores around the fitted prediction function and thus captures any day-to-day variability in symptoms not accounted for by the smoking-covaried regression curve. Volatility is computed as the average squared deviation of observed scores from predicted scores. Higher values indicate more unexplained symptom variability. *Cigarette coefficient* indexes the direction and magnitude of any deflections from the prediction function associated with lapse events. Positive cigarette coefficients indicate symptoms are acutely higher on days when lapses occur, and negative coefficients indicate symptoms are lower when lapses occur. The interested reader may wish to consult Piasecki et al. (2003a) for greater detail on the computation of the growth parameters.

cigarette tally in their diaries, 318 reported smoking at least one postcessation cigarette in the completed ratings. This left 153 participants with an incomplete series of cigarette reports, with all completed reports indicating zero smoking. The frequency distribution for missing cigarette reports in this subgroup was bimodal, showing peaks at 1 missing report (n = 41) and at 56 missing

reports ($n = 52$). We assumed that participants with only a few missing reports were likely to have been abstinent, whereas those with many missing reports were likely to have lapsed and dropped out of the trial. Thus, we allocated participants to lapsed or abstinent groups based on the extensity of missing data; those with three or fewer missing ratings were counted as abstainers, whereas those with four or more missing values were assigned to the smoking group. According to this rule, 63 participants were assigned to the abstinent group, and 90 were assigned to the lapsed group. In sum, 257 (29%) participants were counted as continuously abstinent, whereas 632 (71%) participants were counted as lapsers.

Predictors of withdrawal variability. Four classes of variables were tested as predictors of withdrawal parameters: demographic variables, affect/psychopathology variables, dependence/smoking history variables, and treatment assignment. We standardized continuously scaled variables before entering them into prediction models. Treatment of categorical variables was as described below.

Demographic variables. Four demographic variables were tested: age, sex, study site, and presence of other smokers in the household. A demography screen administered at baseline collected information about participants' age, and sex. Sex was represented by a 0–1 dichotomous variable, with men assigned a score of 1. The parent trial was a multicenter study, with data collected from participants in four states. Three 0–1 dichotomous variables (one each for the Arizona, California, and Nebraska sites) were used to represent site differences; these variables were always entered as a set, with the Wisconsin site (with a score of 0 on all variables) as the reference category. We did not expect site effects to be theoretically informative but included these variables to control for any moderating effect of site differences on prediction from other variables. Participants were asked in a smoking history questionnaire whether they resided in a household with one or more other smokers (yes = 1, no = 0).

Affect/psychopathology. Three affect/psychopathology variables were tested: baseline negative affect, history of major depressive disorder, and history of dysthymia. Participants completed a past-week version of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) at baseline. Because negative affectivity is conceptually and empirically linked to withdrawal (Baker et al., 1987; Piasecki et al., 1998, 2000), scores on the Negative subscale of the PANAS (i.e., the NPANAS) were used as predictors. The mood disorder module of the Structured Clinical Interview for the *DSM–IV* (Spitzer, Williams, Gibbon, & First, 1994) was administered to all participants at screening, and lifetime histories of major depressive disorder and dysthymia were recorded. Dichotomous 0–1 variables, with a score of 1 indicating a positive history of disorder, were computed for depression and dysthymia.

Dependence/smoking history. Eight dependence/smoking history variables were tested. Participants were queried in a smoking history questionnaire administered at baseline about the age at which they began regular smoking, the average number of cigarettes smoked per day, the number of serious prior quit attempts, and their confidence in the ability to quit smoking permanently as a result of the upcoming quit attempt. Confidence was rated using a single item with a Likert-type scale ranging from 1 (*extremely sure*) to 4 (*not too sure*). The Fagerström Tolerance Questionnaire (FTQ; Fagerström, 1978) was also administered at baseline. Two biochemical indices of smoking heaviness were also collected: breath carbon monoxide (measured in parts per million) and blood cotinine concentrations (measured in nanograms per milliliter).

Treatment assignments. A set of three 0–1 dummy codes was used to test the impact of pharmacotherapy assignment. Dummy codes were constructed such that the patch-only, bupropion-only, and patch plus bupropion groups assigned values of 1, and the placebo condition was the reference category (score of 0 on all three variables). These variables were entered together as a block in all models in which treatment was tested.

Analyses Predicting Withdrawal Parameters

Rationale for separate full-sample and lapser-only models. In a companion article (Piasecki et al., 2003a), data from participants who lapsed and participants who maintained continuous abstinence over the 8 weeks immediately following the quit date were combined in a single growth model. Lapse status was included as a predictor in this model, and results revealed robust differences between the groups on all parameters. Relative to continuous abstainers, lapsers showed more severe withdrawal (higher intercepts), less rapid improvement over time (differential linear slope), and a more U-shaped quadratic trend. Planned comparisons also demonstrated that lapsers showed more day-to-day symptom variability (higher volatility) than did abstainers. In this article, we expand these models to include additional predictors of withdrawal while still accounting for the moderating impact of lapse status. Using data from the whole sample permitted the most informative estimates of the relations between predictor variables and all withdrawal parameters except the cigarette coefficient (see below). Controlling for lapse status permitted interpretation of predictive relations without the potential confounding influence of known differences between lapsers and abstainers. Controlling for lapse status corrects estimates of predictive relations for differences between lapsers and abstainers on both the baseline predictor variables (see below, Table 1) and postcessation symptom dynamics (some of which might be attributable to postcessation smoking per se; Piasecki et al., 2003a).

In an additional, parallel set of prediction models we used data only from the lapser subgroup. Separate lapser-only models were necessary because one withdrawal parameter, the cigarette coefficient, can only be estimated in lapsers (complete abstainers have no variance on cigarette tallies). Data from the subgroup of lapsers also permitted us to test the correlates of volatility statistics from which the known effects of smoking (e.g., the spikes in the predicted withdrawal functions in Figure 1) were removed. A separate analysis limited to abstainers was not performed because, as noted above, we believed the most informative estimates of predictor–withdrawal relations for all withdrawal parameters computable for abstainers (i.e., intercept, linear slope, quadratic trend, volatility) were obtained from the full-sample model that controlled for lapse status.

Because the subgroups of lapsers and abstainers were self-selected and could differ in important ways, we compared the two groups on each of the predictors for descriptive purposes (see Table 1). It is not surprising that pharmacotherapy assignment was related to lapse status, $\chi^2(3, N = 889) = 32.3$, $p < .01$, with lapsers being more likely than abstainers to have been assigned placebo. Abstainers and lapsers were differentially distributed across study sites status, $\chi^2(3, N = 889) = 8.9$, $p < .05$. The lapser group contained a significantly higher proportion of women than did the abstainer group, $\chi^2(1, N = 889) = 3.8$, $p < .05$. Lapsers also tended to be younger, $t(887) = 3.7$, $p < .01$; to have started smoking at a younger age, $t(886) = 2.0$, $p < .05$; to have higher cotinine levels, $t(877) = -2.8$, $p < .01$; higher FTQ scores, $t(886) = -4.1$, $p < .01$; and higher NPANAS scores, $t(885) = -3.3$, $p < .01$, at baseline; and to be less confident in their ability to quit smoking, $t(886) = -2.6$, $p < .01$. Note that when lapse

Table 1
Baseline and Demographic Characteristics of Abstainer and Lapser Subgroups

	Abstainers (n = 257)		Lapsers (n = 632)	
Categorical measure	%	*n*	%	*n*
Treatment group*				
Placebo	8.6	22	21.5	136
Bupropion	33.9	87	24.7	156
Patch	23.0	59	29.3	185
Combination	34.6	89	24.5	155
Study site*				
Wisconsin	25.7	66	25.3	160
Arizona	18.7	48	26.7	169
California	25.7	66	25.3	160
Nebraska	30.0	77	22.6	153
Sex (female)*	47.1	121	54.3	343
Other smokers in household	25.4	65	30.4	192
History of depression	16.3	42	18.8	119
History of dysthymia	1.6	4	0.9	6
Continuous measure	*M*	*SD*	*M*	*SD*
Age*	45.4	10.4	42.5	10.8
Age began smoking*	17.7	3.8	17.1	4.2
Cigarettes per day	25.8	9.3	26.9	9.5
CO (ppm)	27.8	10.7	29.2	11.1
Cotinine (ng/ml)*	336.2	154.4	372.7	183.9
FTQ*	7.0	1.7	7.5	1.7
NPANAS*	1.40	0.4	1.53	0.6
Baseline intercept	4.8	10.6	4.7	15.0
Baseline linear slope	1.3	4.6	1.0	5.5
Baseline quadratic	0.4	3.9	0.2	4.4
Baseline cigarette coefficient	−0.01	0.5	−0.003	0.7
Baseline volatility	12.1	21.8	15.8	34.9
Confidence*	1.28	0.5	1.38	0.5
Prior quits	3.1	4.0	2.7	3.0

Note. An asterisk indicates that groups differ at $p < .05$; see text. CO = expired air carbon monoxide; FTQ = Fagerström Tolerance Questionnaire; NPANAS = Negative subscale of the Positive and Negative Affect Schedule.

status is entered in multivariate withdrawal prediction models, it controls for group differences on these baseline variables.

Conceptual overview of HLM growth models. Growth models were conducted using HLM (see Bryk & Raudenbush, 1992, for a detailed mathematical–statistical treatment of HLM). The ultimate aim of the present models was to understand how baseline variables and pharmacotherapy assignment relate to diverse aspects of withdrawal symptom growth or change across the postquit period. At the simplest conceptual level, one can construe this task as involving two nested regression problems.

First, one needs a way of quantifying each participant's pattern of symptoms over time. This can be accomplished by specifying a regression equation in which an individual participant's set of withdrawal scores is predicted from polynomials representing time-related trends and important events (lapses to smoking in this case) that occur at known times. When applied to data from a single participant, this kind of regression equation, often referred to as a *Level 1* model, provides quantitative indices of independent aspects of that participant's symptom "growth"—in the form of regression coefficients for the terms in the equation. Figure 1 presents the regression functions for 2 participants in this trial and illustrates how differences between participants in withdrawal patterning can be indexed by comparing their Level 1 regression coefficients (i.e., growth parameters).

Understanding how baseline variables and pharmacotherapy relate to symptom parameters requires an additional step that can be thought of as involving a "regression equation predicting regression coefficients." Once each individual's withdrawal data are reduced by Level 1 regressions to a small set of quantitative indexes, these growth parameters can be regressed onto baseline variables to determine whether growth-parameter differences can be predicted from baseline variables. To take a simple case, one might write a conventional regression equation in which the dependent variable is withdrawal intercept (i.e., mean severity of withdrawal computed and compiled from hundreds of Level 1 models) and the predictors are sex and pharmacotherapy. This kind of model, in which differences among the coefficients estimated in the Level 1 equations are predicted from individual-difference variables, is conventionally termed a *Level 2* model. Note that the Level 1 model is computed within individuals, and the Level 2 model is computed across individuals. This arrangement of regression equations (linear models) is hierarchical because the statistical outputs from Level 1 become the dependent variables at Level 2.

Of course, this description oversimplifies the process used by statistical packages such as HLM (Bryk, Raudenbush, & Congdon, 1996). For instance, in actual practice, Level 2 models are algebraically substituted into the Level 1 model, and this combined model is solved in a single step by a maximum-likelihood estimation procedure. Nonetheless, the analogy of "regression of regressions" is sufficient for conveying the essential logic of the procedure. Below, we describe the form of the Level 1 models used to index individual differences in symptom growth and the form of the Level 2 models used to predict variations in the Level 1 growth estimates from baseline variables and pharmacotherapy assignment.

Full-sample Level 1 growth model. We assessed connections between predictors and variability in all withdrawal parameters except volatility by elaborating Level 2 models of the growth analyses presented in the companion article (Piasecki et al., 2003a). A quadratic model was applied to the full sample and yielded estimates of intercept, linear slope, and quadratic trend for each participant. Because orthogonal polynomials were used to represent linear and quadratic trends, the intercept term represents mean elevation for the entire 8-week period modeled (i.e., not a quit-date score). HLMs can tolerate missing data (Bryk & Raudenbush, 1992), but in some extreme cases (e.g., a participant who only provided 1 or 2 days of withdrawal ratings), there are insufficient data to fit the Level 1 model. In these instances, participants are automatically ejected from the estimation procedure. Of the 889 participants who reported for at least one study visit, 836 provided enough data to be included in the full-sample Level 1 growth model.

Lapser-only Level 1 growth model. A second model was estimated only for the subsample of smokers who reported lapses during the first 8 weeks of the quit attempt. The Level 1 model in this analysis was similar to that used in the full-sample analyses but was elaborated to include cigarette tallies as a time-varying covariate. This model was a necessary supplement to the full-sample analysis because acute symptomatic changes associated with lapse events (i.e., cigarette coefficients) could only be computed for participants who lapsed. Of the 632 participants counted as lapsers, 578 provided enough data to contribute to the estimation of the overall model (539 participants provided enough nonzero cigarette tallies for their Level 1 models to be estimated).

Level 2 models. We computed predictive relations with variance in computed Level 1 parameters (intercept and linear slope and quadratic trend in the abstainer model; intercept, slope, quadratic, and cigarette coefficients in the lapser model) using the

HLM software (Bryk et al., 1996), entering predictors into Level 2 equations for each parameter described by the Level 1 model. The family of Level 2 models was substituted into the appropriate Level 1 model to form a combined model, and all varying parameters were estimated simultaneously by means of restricted maximum likelihood (Bryk et al., 1996). In these analyses, *t* ratios associated with each fixed effect tested the significance of individual variables against the null hypothesis that the obtained Level 2 coefficient was equal to zero.

Regression analyses predicting volatility. In a companion article (Piasecki et al., 2003a), we introduced a volatility statistic to complement the growth parameters derived from multilevel modeling. The volatility statistic is a measure of day-to-day symptom variability and is defined as the average squared deviation of raw symptom scores from the corresponding predicted values. Volatility estimates for the full sample and lapser subsamples were computed around their respective Level 1 prediction functions. Thus, lapser-only volatility estimates were computed around smoking-covaried Level 1 function, and the systematic effects of postcessation smoking were removed, whereas full-sample volatility measures did not control this source of variance. Volatility could not be modeled using HLM because the indices were computed separately—they require prior estimation of the growth function to be computed (Piasecki et al., 2003a). Relations between baseline variables and volatility were therefore assessed in conventional multiple regression analyses. Separate analyses were performed for the full sample and the lapser-only subsample.

Model-building strategy. Regression and HLM modeling each followed a general strategy outlined by Bryk and Thum (1989); predictors were split into conceptually integrated families of variables (see above), and each family was entered as a set into separate models to identify the significant predictors within each class. Significant predictors from each class were then entered into cross-class multivariate models (hereafter termed *final models*) to identify the strongest simultaneous predictors of each parameter. Because the goal of the analyses was to screen for useful predictors, not to build best-fitting models, and because of the large number of variables, interaction terms were not constructed or tested in the present analyses. To conserve space, we report only the findings from the final models. Each variable tested at Level 2 in a reported final model (see Tables 2–4) was found to be a significant predictor in one of the preliminary models.

This basic modeling strategy was extended in two ways to control important sources of withdrawal variance and aid interpretation of the findings. First, in the full-sample models, lapse status was always entered as a moderator–control variable in predicting each parameter. This was done because prior modeling showed robust differences between lapsers and abstainers on all withdrawal parameters (Piasecki et al., 2003a). Second, we controlled for any significant relations between corresponding pre- and postsymptom parameters before evaluating the predictive power of other variables (see *Baseline symptom dynamics* below). Controlling stable individual differences in symptom expression allowed assessment of how well the baseline variables predict cessation-contingent symptom change.

Baseline symptom dynamics. We used symptom ratings and cigarette tallies from the baseline phase to construct a prequit symptom growth model and then used growth parameters estimated in this model to represent individual differences in parameters of prequit symptomatic experience that corresponded to those modeled postquit. Because participants were actively smoking during the baseline period, the form of the prequit Level 1 growth model was identical to that used to describe lapsers' postquit withdrawal data (i.e., it included a cigarette coefficient). The Level 1 growth parameters (i.e., regression coefficients) from the baseline period were saved to a data file and used as predictors in the postcessation Level 2 models. Including baseline symptom growth parameters as predictors controlled for stable individual differences in symptomatology that were not contingent on cessation.

Results

Full-Sample Final Growth Model

Table 2 presents the final model in which all significant predictors from the preliminary models were combined at Level 2. In this simultaneous model, age, NPANAS scores, and FTQ scores were each positively related to postquit intercepts. All three active pharmacotherapies were associated with lower intercepts. Both control variables (baseline intercept and lapse group) were also positively related to postcessation intercepts. Site and lapse group differences were the only significant effects for linear trend. Age and lapse group were predictive of quadratic trend. Tests of the random effects revealed that significant residual variability was left unexplained by the predictors in the model.

Table 2
Summary of the Final Full-Sample Growth Model Incorporating Significant Predictors From Each Class at Level 2

Fixed effect	Coefficient	*SE*	*t*	*p*
Intercept	9.12	0.65	13.93	<.001
Age	0.65	0.19	3.45	.001
Sex	−0.56	0.35	−1.59	.112
Arizona site	0.14	0.52	0.28	.783
California site	0.07	0.49	0.14	.889
Nebraska site	−0.58	0.46	−1.28	.202
NPANAS	1.74	0.21	8.23	<.001
Depression	0.63	0.47	1.32	.188
FTQ	0.78	0.17	4.63	<.001
Bupropion only	−1.86	0.56	−3.32	.001
Patch only	−1.75	0.57	−3.10	.002
Bupropion + patch	−2.09	0.57	−3.67	<.001
Baseline intercept	0.58	0.21	2.75	.006
Lapse	2.44	0.38	6.46	<.001
Linear slope	−9.99	0.87	−11.42	<.001
Arizona site	2.10	1.00	2.08	.038
California site	0.98	1.02	0.95	.341
Nebraska site	0.91	0.99	0.92	.358
Lapse	3.57	0.75	4.79	<.001
Quadratic	4.31	0.50	8.57	<.001
Age	−0.92	0.26	−3.53	.001
Lapse	−1.74	0.61	−2.85	.005

Random effect	Variance	*df*	χ^2	*p*
Intercept	25.90	822	120808.18	<.001
Linear slope	102.31	831	9064.86	<.001
Quadratic	56.23	833	5255.67	<.001

Note. $N = 836$. Continuous predictor variables were standardized prior to growth modeling. Thus, model coefficients associated with these variables refer to the impact of a $1 - SD$ unit change. NPANAS = Negative subscale of the Positive and Negative Affect Schedule; FTQ = Fagerström Tolerance Questionnaire.

Lapser-Only Final Growth Model

Table 3 presents the final model for the lapser subgroup. In this model, age, NPANAS, and FTQ remained significantly and positively related to postquit intercepts. All three active treatments were associated with significantly lower postquit intercepts. Age was significantly related to postquit quadratic trend. Baseline intercept and baseline quadratic trend significantly predicted corresponding postcessation parameters. No variables were significantly related to postcessation linear slope or cigarette coefficients in the model. Significant residual variability was left unexplained by the predictors in the model.

Predictors of Volatility in the Full Sample

The top portion of Table 4 summarizes the final regression model of full-sample postquit volatility. Younger participants and men were predicted to have less volatility, whereas participants with a history of depression were predicted to have higher volatility. The control variables (lapse status and baseline volatility) were positively related to postquit volatility. Together, the variables in the model accounted for only 8% of the variance in postquit volatility.

Predictors of Cigarette-Adjusted Volatility in Lapsers

The bottom portion of Table 4 summarizes the final regression model predicting volatility in lapsers. Depression history and smoking rate were positively related to postcessation volatility in lapsers. Baseline volatility also predicted postquit symptom volatility. The model accounted for only about 5% of the total variance.

Table 3
Summary of the Final, Lapser-Only, Smoking-Covaried Growth Model Incorporating Significant Predictors From Each Class at Level 2

Fixed effect	Coefficient	*SE*	*t*	*p*
Intercept	11.55	0.51	22.77	<.001
Age	0.71	0.23	3.07	.003
Sex	−0.79	0.44	−1.79	.073
NPANAS	1.62	0.24	6.63	<.001
FTQ	0.86	0.21	4.08	<.001
Bupropion	−1.53	0.65	−2.34	.019
Nicotine patch	−1.36	0.62	−2.19	.029
Combination therapy	−2.10	0.66	−3.17	.002
Baseline intercept	0.63	0.27	2.38	.018
Linear slope	−7.14	0.87	−8.21	<.001
Arizona site	2.22	1.24	1.78	.074
California site	1.82	1.24	1.47	.141
Nebraska site	1.27	1.24	1.03	.305
Quadratic	2.56	0.33	7.79	<.001
Age	−0.95	0.29	−3.22	.002
Baseline quadratic	1.08	0.34	3.22	.002
Cigarette coefficient	0.22	0.03	6.94	<.001

Random effect	Variance	*df*	χ^2	*p*
Intercept	30.69	530	59525.49	<.001
Linear slope	113.67	535	5900.13	<.001
Quadratic	52.03	536	3121.91	<.001
Cigarette	0.32	538	4304.68	<.001

Note. $N = 578$. Continuous predictor variables were standardized prior to growth modeling. Thus, model coefficients associated with these variables refer to the impact of a $1 - SD$ unit change. NPANAS = Negative subscale of the Positive and Negative Affect Schedule; FTQ = Fagerström Tolerance Questionnaire.

Table 4
Summary of the Final Regression Analyses Predicting Symptom Volatility

Variable	*B*	*SE* (*B*)	β	*p*
Full sample[a]				
Lapse	4.91	1.19	0.15	<.001
Age	−1.35	0.55	−0.09	.014
Sex	−2.21	1.11	−0.07	.048
NPANAS	0.73	0.58	0.05	.207
Depression	3.99	1.46	0.10	.006
Baseline volatility	1.56	0.56	0.10	.006
Lapsers[b]				
Sex	−2.16	1.24	−0.08	.081
Depression	3.11	1.56	0.09	.048
Smoking rate	1.52	0.59	0.11	.011
Baseline volatility	1.61	0.53	0.13	.002

Note. Continuous predictor variables were standardized prior to growth modeling. Thus, model coefficients associated with these variables refer to the impact of a $1 - SD$ unit change. NPANAS = Negative subscale of the Positive and Negative Affect Schedule. [a] $F(6, 737) = 10.18$, $p < .001$, $R^2 = .077$. [b] $F(4, 532) = 6.31$, $p < .001$, $R^2 = .046$.

Discussion

The major aim of this article was to explore connections between a variety of baseline measures and individual differences in parameters indexing distinct aspects of the course of smoking withdrawal. This is important because, although certain individual-difference variables (e.g., sex, depression history, dependence) are often predictive of smoking cessation outcomes, the causal or mediational processes that might link these characteristics with cessation success or failure are not known. Identifying such links could enhance the ability to design effective treatments.

The results of this study demonstrated that the withdrawal parameters differed in terms of the number and nature of their correlates. Another clear finding was that the available baseline predictors only accounted for a small portion of the between-persons variation in withdrawal features. For instance, available measures accounted for less than 10% of the variance in the volatility measure. Given that volatility was defined in a manner that is conceptually similar to the definition of error in regression models (i.e., residuals from the prediction line), it may not be surprising that it was poorly predicted. However, it was surprising that other parameters could not be strongly predicted. Withdrawal intercepts were the best-predicted growth parameter. Supplementary analyses (comparing variance estimates from nested growth models) suggested that the predictors listed in Table 2 only accounted for about 23% of the intercept variance in the full sample (lapse status accounted for 5.5%, and the remaining variables accounted for 17.5%). In the

lapser-only subsample, the predictor set (see Table 3) accounted for approximately 15% of the intercept variance. There clearly must be additional determinants of withdrawal variation that were not measured in this trial.

A robust finding, consistent with those of a companion article (Piasecki et al., 2003a), was that lapsers were predicted to have symptom parameters that should be more aversive (e.g., higher intercepts, less negative or positive slopes, greater curvature, and greater volatility) than those reported by complete abstainers. The present data show that this lapse effect persists even when baseline variables known to differ between lapsers and abstainers (e.g., pharmacotherapy, dependence scores; see Table 1) are included in expanded Level 2 models. This finding has two potential theoretical interpretations. One interpretation is that lapse status is a predictor because extreme withdrawal parameters are direct artifacts of postcessation smoking. If smoking drives the symptom patterning, then lapse status should be a potent proximal instigator that displaces more distal baseline variables in prediction models. We believe this account is not completely accurate because we have demonstrated that lapsers and abstainers differ in their withdrawal patterning even before the first lapse to smoking has occurred (Piasecki et al., 2003b). A second possibility is that idiosyncratic postcessation situational variables, such as stressor occurrence (unmeasured in this trial), are strong determinants of withdrawal patterning, and this patterning then contributes to lapse likelihood. If this account is correct, then lapse status may serve as a proxy measure that integrates information about participant-specific instigators of distress. Of course, a blend of these two accounts is possible, such that extreme early withdrawal symptoms kindle lapses that then cause further disturbances in withdrawal dynamics.

Smoking withdrawal symptoms are presumed to arise after cessation, at least in part because of physical nicotine dependence (Benowitz, 1991; Shadel et al., 2000). Dependence level was clearly implicated as a determinant of the elevation of postquit withdrawal scores. The FTQ was related to higher withdrawal intercepts in both the full-sample and lapser-only growth models. This finding is consistent with the hypothesis that more highly dependent individuals should suffer more intense pharmacologic withdrawal (Etter, Vu Duc, & Perneger, 1999; West & Russell, 1985). However, with the exception of the finding that heavier smokers showed more day-to-day variability of symptoms in the lapser-only volatility analyses, none of the dependence-related variables predicted individual differences in withdrawal parameters other than the intercept.

All three active pharmacotherapy regimens were associated with diminished intercept values, indicating that they alleviated mean withdrawal severity. The finding that pharmacotherapy resulted in less severe mean symptomatology is consistent with prior clinical trial data showing that these treatments tend to ameliorate withdrawal symptoms (Hurt et al., 1997; Jorenby et al., 1996). However, no other withdrawal parameter was significantly predicted by treatment assignment in either lapsers or abstainers. It is notable that we have found that withdrawal parameters such as linear slope, volatility, and cigarette coefficients predict smoking relapse (Piasecki et al., 2003b). Our findings join with others (e.g., Tiffany, Cox, & Elash, 2000) in suggesting that measuring the dynamics of postcessation symptoms may reveal limitations of current treatments that are not apparent when only mean symptom levels are analyzed.

Clinically, the pharmacotherapy findings suggest that clinicians prescribing current smoking cessation pharmacotherapies may need to temper their patients' expectations about the kinds of symptom relief they will obtain. Available medications may weaken the overall intensity of withdrawal but may have little effect on the variability of symptoms over time, direction and rate of change in symptoms across the quit attempt, and so on. In short, smokers preparing for a quit attempt must understand that a "bumpy ride" is possible even with the aid of pharmacotherapy. They should be counseled against inferring that medications are not working or that they are somehow unusual or "doomed to fail" when they encounter variable or occasionally strong symptoms after quitting.

As expected, affect/psychopathology measures showed connections with various withdrawal parameters. Participants with higher baseline NPANAS scores reported higher withdrawal intercepts (indicating more severe mean withdrawal). This finding is consistent with an earlier study showing that NPANAS scores predicted withdrawal elevation across 4 weeks of cessation (Piasecki et al., 2000). Other studies have shown that NPANAS scores collected after cessation both track withdrawal (Piasecki et al., 1998) and are strong predictors of relapse (Kenford et al., 2002). It is possible that high negative affect prior to quitting may mark individuals who have more stressful lives at the time of the quit attempt, or that high baseline NPANAS scores reflect well-founded apprehension about the fate of the quit attempt.

Depression history has been linked to more frequent and severe withdrawal symptoms (e.g., Covey et al., 1990). Our data did not reveal particularly strong connections between depression history and withdrawal severity, slope, or cigarette coefficients. It was intriguing, however, that depression history was associated with elevated day-to-day symptom volatility. This suggests that depression-positive smokers are more reactive to (unmeasured) environmental events after cessation or that they simply are prone to experience more variable affective states.

There was some evidence that certain symptom parameters were stable from precessation monitoring to the postquit period. In particular, baseline intercepts and volatility were good predictors of the corresponding postquit parameters. These relations suggest that some features of postquit scores may reflect persistent individual differences in affective style or rhythm. Future research measuring symptoms during longer periods of ongoing smoking could help to better characterize both stability and cessation-contingent change in withdrawal-like symptoms.

In prior cluster-based studies of withdrawal dynamics, we found that women were overrepresented in withdrawal clusters characterized by unusual shapes (Piasecki et al., 1998). In the present study, women were overrepresented in the

lapse group. Sex effects on withdrawal measures were relatively weak, however, and only one effect remained significant in the final models: Women were predicted to experience more volatile day-to-day symptom variability in the full-sample analysis. This suggests that sex effects on withdrawal may have been mediated by other factors that did emerge as significant predictors. Future research should examine whether sex differences in volatility account for the tendency for women to relapse more often than men (Wetter et al., 1999).

One interesting and unexpected finding was that the withdrawal parameters differed widely in the number of significant predictors. In particular, linear slopes and cigarette coefficients were not predicted by any of the available, theoretically informative measures. It is possible that facets of withdrawal such as linear slope or cigarette coefficient may need to be explained with reference to dynamic postcessation events. Consistent with this notion, we found that lapsers with negative cigarette coefficients reported more lapse days after quitting and more cigarettes per lapse day (Piasecki et al., 2003a), suggesting that negative cigarette coefficients may capture acute negative reinforcement or self-medication effects. If withdrawal parameters such as linear slope or cigarette coefficients are highly dependent on idiosyncratic postcessation events, it would stand to reason that they would not be predicted well by stable individual-difference measures collected at a temporally distal baseline point. The fact that linear slopes and cigarette coefficients are related to relapse (Piasecki et al., 2003b) suggests that these dimensions have real clinical usefulness and that their correlates deserve to be explored further.

Limitations of the present study should be borne in mind. The analyses were clearly exploratory in nature. The emphasis was on deriving a first-pass sense of factors contributing to different facets of withdrawal rather than distilling best-fitting models for each parameter. Because of these goals and the large number of predictors, interaction terms were not included in the prediction analyses. In addition, to avoid premature closure, we did not perform post-hoc adjustments of statistical significance tests to manage the experimentwise error rate. The tests used were fairly stringent multivariate prediction models that required a variable to emerge as one of the best predictors in its class and then remain significant in the presence of the best predictors from other variable classes before being interpreted. Nonetheless, some of the predictors in this research still might have been found to be statistically significant because of chance alone. Isolating replicable predictors of withdrawal parameters is a long-run research problem that necessarily must involve the use of multiple independent samples. The present analyses may be viewed as a start to this process, perhaps identifying a handful of variables worthy of future scrutiny, but further research is necessary to evaluate their consistency and examine interactions among them. Additional limitations of this study include reliance on paper diary data; the lack of sensitive postcessation measures of affect-relevant constructs; the short duration of the baseline symptom assessments; and the use of a brief assessment of withdrawal symptoms that did not contain reliable, multi-item subscales, thus not permitting symptom-specific analyses.

Such limitations notwithstanding, the present findings highlight that withdrawal symptoms may be better understood through analyses that capture the information about the numerous ways in which withdrawal can vary across time and persons. Second, the findings suggest that current pharmacologic treatments are relatively impotent in affecting some motivationally vital (i.e., relapse-related) aspects of the withdrawal experience. Third, the data suggest that, although some withdrawal parameters such as volatility and symptom elevation may mediate the impact of stable risk factors for relapse (e.g., gender, affective disorder), other dimensions such as symptom slope may reflect the impact of transient or phasic events. This suggests diverse mediational relations in which both persons and environmental risk factors are posited to affect relapse via specific paths involving different withdrawal parameters. Thus, dimensional approaches to withdrawal assessment may ultimately reveal not only heterogeneity in withdrawal but also heterogeneity in links between risk factors, treatment, withdrawal, and relapse.

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