Smoking Withdrawal Dynamics in Unaided Quitters

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Considerable research shows that withdrawal severity is inconsistently related to smoking cessation outcomes. This may result from measurement problems or failure to scrutinize important dimensions of the withdrawal experience. Two recent studies demonstrated that withdrawal elevation and variations in the time course of withdrawal were related to relapse in smokers treated with the nicotine patch (T. M. Piasecki, M. C. Fiore, & T. B. Baker, 1998). This article reports a conceptual replication and extension of those findings in unaided quitters. Evidence for temporal heterogeneity was found across different types of withdrawal symptoms. Patterns or slopes of affect and urge reports over time predicted smoking status at follow-up, as did mean elevation in withdrawal symptoms. These results suggest that affect and urge withdrawal symptoms make independent contributions to relapse and that relapse is related to both symptom severity and trajectory.

The study of withdrawal is at a watershed. Traditional models of drug dependence and drug motivation have accorded withdrawal a central role in motivating addictive drug use and relapse (e.g., Isbell & White, 1953; Seevers, 1962; Wikler, 1973). In addition, for many years, the presence of withdrawal symptoms has constituted a central criterion for the diagnosis of a drug dependence disorder (American Psychiatric Association, 1994). However, recent models of dependence and drug motivation have almost universally de-emphasized withdrawal as a central explanatory construct (cf. Heyman, 1996; Marlatt, 1985; Robinson & Berridge, 1993; Vuchinich & Tucker, 1988). This has transpired largely

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Correspondence concerning this article should be addressed to Thomas M. Piasecki, Center for Tobacco Research and Intervention, 7275 Medical Sciences Center, 1300 University Avenue, Madison, Wisconsin 53706, or Raymond Niaura, The Miriam Hospital, Division of Behavioral and Preventive Medicine, 164 Summit Avenue, Providence, Rhode Island 02906. Electronic mail be sent to tmp@ctri.medicine.wisc.edu or Raymond_Niaura@brown.edu. because measures of withdrawal have not covaried consistently with relapse vulnerability and other purported indices of dependence. Theoreticians have offered a litany of observations that challenge withdrawal-based accounts. These include the fact that there is an inconsistent relation between the tendency of a drug to support habitual use and the severity of the drug's withdrawal syndrome (e.g., Jaffe, 1980). Also, drug-dependent persons frequently do not identify withdrawal as an instigator of relapse (Marlatt, 1985), and relapse often occurs long after the cessation of drug use, when withdrawal symptoms should have abated (Brandon, Tiffany, Obremski, & Baker, 1990). Finally, the extent to which treatments suppress withdrawal does not correspond well with their ability to produce abstinence (Jorenby et al., 1995; Transdermal Nicotine Study Group, 1991).

Various factors might account for the weak relations between withdrawal and relapse. For instance, it may be the case that smoking withdrawal symptoms have little impact on cessation and the maintenance of abstinence. Alternatively, inadequate conceptualization and assessment strategies may have caused researchers to overlook important dimensions of the withdrawal phenomenon (Gilbert et al., 1998; Piasecki, Kenford, Smith, Fiore, & Baker, 1997). Prior to abandoning withdrawal as a vital explanatory construct, it seems prudent to pursue novel approaches to the study of withdrawal that may yield additional information about its correlates and consequences (Meehl, 1978; Patten & Martin, 1996a). Indeed, investigators have begun to adopt new and innovative approaches to the study of withdrawal (e.g., Epping-Jordan, Watkins, Koob, & Markou, 1998; Gilbert et al., 1998; Keenan, Hatsukami, Pentel, Thompson, & Grillo, 1994; Shiffman et al., 1997; Swan, Ward, & Jack, 1996).

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When a mismatch between strong theory and empirical findings exists, measurement issues are a natural object of scrutiny (Serlin & Lapsley, 1985); investigators should be reluctant to discard venerable theories if there is a possibility that empirical discrepancies might stem from inadequate measures. Hence, it is not surprising that smoking researchers have begun to reexamine withdrawal instruments (Patten & Martin, 1996b; Welsch et al., 1999). Another tactic that can be used to reevaluate withdrawal is to start with withdrawal measures that are presumed valid and then analyze them in novel ways.

We have recently reported results from two studies examining variability in the time course of withdrawal symptoms that reflect this approach to measurement evaluation (Piasecki, Fiore, & Baker, 1998). In these studies, we clustered quitters according to the shapes of their withdrawal profiles and demonstrated that atypical withdrawal, characterized by late peaks or unremitting symptoms, was quite common. Such withdrawal profiles are termed *atypical* because they diverge from the prototype generated by traditional models of withdrawal (Hughes, Higgins, & Hatsukami, 1990). Atypical profiles were common, occurring in smokers who had lapsed and those who were abstinent and occurring in smokers who received diverse interventions. Data were drawn from two clinical trials of the nicotine patch, and the withdrawal scale and assessment schedules used in these trials conformed to common research practice.

Atypical profiles were not only shown to be fairly common, but they were also related to relapse; participants with atypical profiles were more likely to relapse than were other smokers. This relation could involve a variety of mechanisms. For instance, smokers with worsening symptoms might be more likely to return to smoking in order to manage their worsening symptoms. Smokers who lapse might experience an exacerbation in withdrawal due to the lapse, and this might motivate further smoking and relapse. Thus, although little is known about the mechanisms that link withdrawal profiles to relapse, there is evidence that profile shape carries motivational meaning.

Although preliminary research suggests that smoking withdrawal heterogeneity is real and consequential, vital questions remain about the phenomenon and its generality requires exploration. For instance, our previous research was conducted with participants who were undergoing intensive formal cessation treatments. Because smokers who volunteer for such treatments tend to differ from other smokers (Fiore et al., 1990; Lichtenstein & Hollis, 1992), it is important to establish the generalizability of the results with unaided quitters. In addition, it is vital to determine whether withdrawal profile type is significantly related to relapse when participants' postcessation smoking/lapse status is statistically controlled. If withdrawal profile is merely a proxy for lapse status, then profile information should contribute nothing to statistical models of relapse once lapse status is taken into account. Finally, the withdrawal measure used in the previous research was a brief eight-item scale laden with items tapping affect. A broader measure with multiple-item scales tapping distinct symptom domains may reveal different results across the different domains.

In this article, we examine symptom trajectories in a variety of withdrawal subdomains in a sample of unaided quitters. To characterize temporal variance in withdrawal reports, we explore connections between symptom trajectories and an array of affect and dependence indices, and we use a variety of strategies to begin teasing apart the connections between postcessation smoking and withdrawal patterns. We assess the clinical relevance of atypical withdrawal by examining the connections between categorical (cluster based) and continuous (simple slope) measures of withdrawal trajectory and relapse. Central theoretical principles guiding our research on withdrawal heterogeneity posit that the trajectory of withdrawal symptoms reflects the responsivity of affective processing systems to pharmacologic and nonpharmacologic stimulation (Piasecki et al., 1997, 1998) and that affects provide a readout of motivated behavior (e.g., Baker, Morse, & Sherman, 1987; Buck, 1993). These principles give rise to the prediction that the affective components of the withdrawal syndrome should show the strongest relations with relapse.

Method

Participants and Procedure

Data for this report were drawn from a study of unaided quitters conducted at The Miriam Hospital in Providence, Rhode Island (Shadel et al., 1998). To be eligible for the study, participants had to: (a) be between 18 and 75 years of age; (b) have no decrease in the number of cigarettes or nicotine content of their usual brand of cigarettes by more than half in the previous 3 months; (c) have no use of tobacco products other than cigarettes; (d) report motivation to quit as at least 5 on a 10-point scale; (e) not be enrolled in any other smoking cessation program or use any other quitting methods (e.g., patch, gum); (f) not attempt, if female, to become pregnant; (g) not be currently treated for ongoing psychiatric disorders; and (h) not be taking medication that might interfere with psychophysiological assessments (e.g., beta blockers).

Participants visited the laboratory a total of eight times during the study. Three sessions occurred prior to the quit date. At the first session, participants completed a variety of self-report measures and gave blood samples. At a second session, held the next day, participants underwent various experimental manipulations of smoking motivation (see Shadel et al., 1998). The third session, scheduled 3 days after the experimental session, was a brief check-in meeting where participants completed a withdrawal assessment and were given a copies of the American Lung Association's "Freedom from Smoking" self-help pamphlet. The quit day for all participants was scheduled for 4 days after the check-in meeting. Participants reported to the lab for brief assessments (including withdrawal assessments) on the quit date and on Days 2, 7, 14, and 30 postquit. Other than the self-help pamphlet, participants were offered no formal behavioral support or pharmacological treatment for smoking cessation. Participants received \$170 for completing the study.

The original sample included 183 individuals, however, the profile methods used in this research required that complete withdrawal data be available for all participants at each follow-up time point. This criterion eliminated 24 individuals, leaving 159 cases for analysis. The analyzed sample was 56% female and 95% Caucasian with a mean age of 42.9 years (SD = 12.1). The analyzed sample reported a mean Fagerström Tolerance Questionnaire (FTQ; Fagerström, 1978) score of 6.5 (SD = 1.8), a history of 3.3 serious quit attempts (SD = 2.3), and a typical smoking rate of 24.2 (SD = 9.5) cigarettes per day. A series of analyses comparing analyzed participants with excluded sample members did not reveal significant differences between the groups on any demographic or dependence variables.

The sample analyzed was not limited to only those individuals who maintained continuous abstinence throughout the follow-up period. Eliminating participants who slip or lapse may restrict the range of both withdrawal and relapse data, attenuating relations between them (e.g., Gilbert et al., 1998). Instead, we opted to conduct analyses to assess the impact of intratrial smoking on our findings (see following discussion).

Measures

Four types of measures were used in this research: measures of withdrawal, postcessation cigarette counts, end-of-study abstinence, and baseline smoker characteristics.

Withdrawal

The primary measure of withdrawal symptomatology in this research included items from the Hughes and Hatsukami (1986) and Shiffman and Jarvik (1976) withdrawal scales, along with additional items tapping urge/ craving. An exploratory factor analysis with principal components extraction and varimax rotation performed on the quit-day data suggested that three factors influenced the manifest item intercorrelations. The first factor largely comprised items related to negative affect (Affect), the second factor largely concerned urges/cravings (Urge), and the third factor included items concerning sleep disturbance/energy (Sleep/Energy). The withdrawal items and their loadings on the factors to which they were assigned are given in the Appendix. These symptom subdomains were considered separately, and dynamic patterns in each were examined using cluster analysis. To create symptom subscales that were conceptually pure and distinct, the factor score coefficient matrix derived from the quit-day data factor analysis was used to compute quasi-orthogonal subscales at all time points.1

Postcessation Cigarette Counts

At each postcessation study visit, participants were asked to recall retrospectively how many cigarettes they had smoked each day since the last study visit.

Abstinence

Participants were considered to be abstinent at the end of the study if they reported smoking zero cigarettes in the 7 days preceding the Day 30 assessment and if they provided a saliva sample containing less than 20 ng/ml cotinine.

Baseline Smoker Characteristics

At the first precessation visit, participants completed a series of paperand-pencil measures that tapped smoking history/nicotine dependence and mood. Smoking history/dependence measures included the FTQ, age of smoking initiation, regular smoking rate, number of serious quit attempts, and plasma nicotine and cotinine. Mood measures were the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988), the Center for Epidemiological Studies Depression Scale (CESD; Radloff, 1977), and the Perceived Stress Scale (PS; Cohen, Kamarck, & Mermelstein, 1983). A variety of other measures were collected but were not used for the purposes of this article.

Statistical Analyses

A three-phase analytic strategy was adopted for this article. The descriptive phase served to characterize temporal variability in symptom subdomains and to form participant subgroups that would become the bases for subsequent analyses. The second analytic phase involved screening groupings from the first phase for clinical relevance by using a series of logistic regression analyses. The final phase, external correlates, examined an array of affective, demographic, and dependence variables to see whether any of these foreshadowed postcessation symptom patterning.

Descriptive Analyses

Factor score evaluation. The withdrawal measure used in this research contained a mixture of items from different scales, and item scores were transformed to yield nonredundant symptom measures. Before forming subgroups of smokers based upon these factor score scales, we sought to determine whether the measures showed aggregate temporal patterns consistent with those shown in the descriptive literature. Mean profiles computed across the entire sample for each factor score scale were plotted. In essence, these plots assessed whether the variance unique to each symptom domain met a minimum validity standard as a measure of withdrawal.

Profile clustering. The basic profile methodology from our previous studies was used with these data. In brief, participants' withdrawal ratings for Days 1, 2, 7, 14, and 30 were considered to constitute their dynamic profile. Profiles were standardized casewise to remove elevation and scatter, leaving only shape information (i.e., the patterns of ups and downs; Cronbach & Gleser, 1953). Then, the 159 standardized profiles were submitted to a hierarchical agglomerative cluster analysis using Ward's (1963) minimum variance procedure and the squared Euclidian distance measure. In this procedure, each resulting cluster represents a group of individuals with similar temporal trajectories on the measure under consideration (e.g., Prochaska, Velicer, DiClimente, Gaudagnoli, & Rossi, 1991). This procedure was performed three times, once for profiles from each of the symptom domains. Thus, the entire analyzed sample was partitioned three separate times, and each participant was assigned to an affect scale cluster, an urge scale cluster, and a sleep/energy scale cluster.

Stopping rules for hierarchical cluster analysis remain the focus of considerable research and debate (e.g., Krolak-Schwerdt & Eckes, 1992; Overall & Magee, 1992; Schweizer, 1992), and no single quantitative or heuristic algorithm has clearly emerged as superior. Absent an empirically validated criterion, we opted to use a simple, practical heuristic; we retained the maximum number of clusters with no fewer than 20 participants in any cluster (Morral, Iguchi, Belding, & Lamb, 1997), in conjunction with visual inspection of the dendrogram and fusion coefficients. Such a method allowed for participant differentiation while restricting the retention of trivially small groups. This rule is somewhat arbitrary, but its validity is better evaluated by examination of its usefulness for organizing other aspects of the data than by its mathematical rigor or sophistication (Morral et al., 1997; Skinner, 1981).

Prototypic-atypical classification. For analyses in subsequent phases of this study, members of atypical clusters were combined. This was done for both theoretical and statistical reasons. First, whether or not an individual displays an atypical withdrawal trajectory may be more important than the specific form of that trajectory (Piasecki et al., 1997). Theory and prior research (Piasecki et al., 1998) suggest that if

¹ These factor scores were, of course, only quasi-orthogonal, because a factor score coefficient matrix derived at one time point was used to adjust scores at all time points. These coefficients do not yield truly orthogonal subscales at all time points, because they capitalize on indiosyncracies in the quit day data. Nonetheless, it seemed most reasonable to apply a consistent mathematical rule for adjusting item scores at all time points. On the quit day, the Affect and Sleep/Energy subscales were intercorrelated at .58, whereas their factor scores were correlated at -.09. Comparable diminution in subdomain intercorrelations was observed across subdomain pairs and time points.

smokers have withdrawal symptoms that either increase or remain elevated, they are at higher risk of relapse than smokers who have withdrawal symptoms that steadily abate. Collapsing across atypical profile types also increases statistical power. Although we collapse participants with atypical profiles for theoretically motivated statistical tests, we graphically depict symptom profiles on a group-by-group basis to convey maximal descriptive information.

Cluster analyses generated a family of profiles for each factor score scale that was analyzed. To designate a prototypic profile (with a steadily descending course) from each family of profiles in an objective manner, we first identified mean withdrawal ratings for the relevant time points from the prototypical group in a previous study (i.e., Study 1; Piasecki et al., 1998). S correlations with this historical prototype were then computed for all group profiles that were derived from the present data. For each scale type, the group with the highest S correlation (i.e., a product-moment correlation computed between group profiles across time points; Cattell, 1966) was considered the prototype. Dummy coding was used to contrast prototypic and atypical clusters in all subsequent analyses.

Assessing Clinical Relevance

A series of logistic regression analyses were performed using Day 30 point-prevalence relapse as the dependent measure. These were conducted to ascertain (a) withdrawal parameters that were associated with relapse; (b) the best combination of withdrawal parameters for modeling relapse; (c) the incremental improvement made to the final withdrawal model, if any, by nonwithdrawal variables; and (d) the influence of postcessation smoking on relations between withdrawal parameters and relapse. Multivariate logistic models were constructed following the strategy advocated by Hosmer and Lemeshow (1989). Four withdrawal parameters were considered as predictors in these analyses: the three dummy variables, representing the typical-atypical distinction derived from each cluster analysis, and an elevation variable, representing each participants' mean level of reported global distress (total scale score) across the assessment period. The elevation variable was included based on a priori rationales. First, it was a strong predictor of outcome in our previous studies (Piasecki et al., 1998). Second, it controls for the impact of symptom severity per se, which allowed us to isolate the predictive validity of profile shape.

Screening candidate predictors. Before building logistic models, we assessed the overlap of the prototypic-atypical groupings for each symptom measure using chi-square tests, because redundant classifications would influence the interpretability of multivariate models. Individual withdrawal variables were then screened in univariate logistic models before multivariate modeling. The main effects of each withdrawal parameter were assessed via separate logistic regression models. In each model, the dependent variable was Day 30 point-prevalence relapse rates, as defined earlier, and a withdrawal parameter was entered as the sole predictor.

Multivariate logistic model. Withdrawal variables displaying at least modest relations with the outcome measure (p < .25) were considered for inclusion in a multivariate withdrawal model. Once the pool of candidate predictors was determined, a multivariate model containing all of the retained variables (i.e., a saturated model) was fitted. The Wald statistic and other estimated coefficients were examined in the saturated model, and variables that did not contribute to the model were deleted. After a variable was deleted, the likelihood ratio statistic was computed to determine whether the deletion resulted in a decrement in overall model goodness of fit; if this occurred, the predictor was retained. This process was performed iteratively until a final model, from which no other variables could be deleted, was obtained. The overall goodness of fit of this final model was then assessed via the Hosmer-Lemeshow \hat{C} statistic.

Contribution of nonwithdrawal variables. After the multivariate withdrawal model was built, we sought to determine whether any of the smoker characteristics measured at baseline would improve the model. In separate logistic regression analyses, each of the smoker characteristic variables was added to the best-fitting withdrawal model at the last step. All continuous predictors were tested for linearity of the logit; where the assumption of linearity appeared to be violated, continuous variables were converted to polytomous form and were tested again. The model chi-square improvement statistic was examined in each analysis to determine whether any nonwithdrawal variable significantly augmented the withdrawal model.

Influence of postcessation cigarette use. We conducted several analyses to examine the extent to which the differing withdrawal patterns and their associations with relapse were attributable to participants' smoking during the follow-up period. The first strategy was to perform a series of repeated measures analyses of variance (ANOVAs) using cigarette counts as the dependent measure, comparing typical and atypical cluster members on the pattern and amount of cigarettes smoked during the trial. Only participants who reported smoking at least one cigarette during the cessation period were included in these analyses. Separate analyses were performed for each cluster solution.

Second, logistic regression models were built to assess whether any withdrawal parameter could improve prediction of relapse after a variable representing lapse occurrence (i.e., the participant reported smoking at least one cigarette from Day 1–21 of the quit attempt) during the trial was forced into the model at a prior step. Model building followed the same strategy that was used to construct the multivariate withdrawal model. At Step 1, lapse status was entered; at Step 2, all withdrawal parameters retained in the final withdrawal model were entered as a block to form a saturated model. Withdrawal variables that appeared to be weak predictors were then individually deleted, unless deletion worsened overall model fit. Because postcessation slips are strongly and consistently related to relapse at distant follow-up points (e.g., Gourlay, Forbes, Marriner, Pethica, & McNeil, 1994; Kenford et al., 1994), this represents a fairly conservative test of the hypothesis that withdrawal patterns contain unique motivational information.

A final, rather more descriptive approach to this problem involved splitting each cluster into subgroups based upon latency to first postcessation cigarette puff. As many as six groups could be formed for any cluster. One group represented individuals who reported continuous abstinence across the 30 days, and five groups represented various postcessation assessment visits at which the first report of smoking was given (first lapse reported at Day 1, Day 2, Day 7, Day 14, or Day 30). Once groups were formed, symptom profiles were plotted for each group to depict the degree of symptom pattern coherence within each cluster. This coherence was also quantified using coefficient alpha (treating subgroups as items and occasions as subjects) to provide a summary index of the extent to which symptoms jointly wax and wane across groups within a cluster. If postcessation smoking events precipitate atypical withdrawal, then we would expect to see that groups differing in the latency to the first lapse show systematically different patterns of withdrawal (e.g., time-shifted peaks), even though they happen to be in the same cluster, and that only a small family of lapse histories are represented in each cluster (for example, just participants lapsing early in the quit attempt).

Symptom slopes and relapse. We use cluster analyses in this research because it systematically replicates our prior findings and because clustering is a descriptively rich method of data reduction. Nonetheless, cluster analysis involves subjective judgments that raise concerns about the reliability of the procedure. In addition, clustering masks intracluster heterogeneity. We have argued in previous work that cluster membership predicts smoking outcomes because the clusters are formed on the basis of symptom trajectory and this has motivational meaning. This suggests that the use of simple symptom slopes across the postcessation period should replicate some of the cluster solution findings. Strong negative slopes would indicate symptomatic relief and should be associated with abstinence, whereas near-zero or positive slopes would suggest arrested or exacerbating withdrawal and should be associated with relapse. Finding a relation between slopes and outcomes would suggest that symptom-relapse connections are not dependent upon a fortuitous cluster solution.

To test this idea, we computed rough slope indices by taking a simple difference score for each participant, subtracting postcessation Day 2 scores in each symptom domain from Day 30 scores. These slope measures were then substituted for the prototypic-atypical group distinction in the three logistic regression models (multivariate withdrawal model, withdrawal model + nonwithdrawal predictors, lapse-adjusted model; derivation of these models was described earlier) to determine whether symptom trajectory remained associated with relapse when continuously scaled representations were used.

Slope measures and cluster distinctions clearly reflect somewhat different information about the symptom trajectory. For instance, slope measures would be expected to be relatively insensitive to transient increases in symptoms occurring in the middle of the cessation period, but this information would influence clustering. A final set of logistic models assessed whether prototypic-atypical cluster distinctions contributed to prediction of relapse after elevation and slope measures were forced into the model at an earlier step. These analyses were performed only for symptom classes that were related to relapse, and they address whether there is any unique value to clustering.

External Correlates

A series of analyses tested whether symptom variables that were related to clinical outcome in the multivariate withdrawal models were related to baseline smokers' characteristics. One set of comparisons focused on nicotine dependence measures, which were broadly conceived (Skinner, 1990). Variables considered in this set of comparisons were: plasma nicotine, plasma cotinine, FTQ, age of smoking initiation, regular smoking rate, and number of serious quit attempts. The second set of comparisons focused on affect-relevant baseline characteristics. Variables included in this set were both subscales of the PANAS, the CESD, and the PS. Associations between baseline measures and symptom slopes and elevation were tested using zero-order correlations, whereas while associations with prototypic-atypical cluster distinctions were assessed using t tests.

Results

Descriptive Analyses

Factor Score Evaluation

The time course, averaged across the entire sample, for each transformed symptom subscale, is depicted in Figure 1. In the plot, profiles are transformed into a standard z-score space to emphasize their shape and to foster comparability. The Affect and Urge subscales generally conformed to the the expected transient temporal pattern expected of classic withdrawal symtoms (Hughes & Hatsukami, 1986; Hughes et al., 1990), although Affect scores had not resolved to baseline levels by the end of follow-up. However, the unique variance in the Sleep/Energy items did not show the classic temporal signature of a withdrawal measure. Scores on this scale were highest at baseline and were lower than baseline



Figure 1. Aggregate time course of the individual symptom measures used in this article. Data points represent mean ratings from the whole sample for each factor score at the corresponding time point. Profiles are depicted in z-score space to eliminate differences in scaling. B1 = 8 days precessation; B2 = 4 days precessation; QD = quit day. Remaining axis labels represent the number of days postquit.

throughout the abstinence attempt.² Despite their unusual aggregate pattern, we chose to include Sleep/Energy factor scores in subsequent analyses to characterize temporal variability in this measure and to assess its clinical relevance.

Profile Clustering

The three panels of Figure 2 display in standardized form the group profiles derived by submitting each withdrawal subscale measure to cluster analysis.³ In all panels of Figure 2, the prototypical cluster is Cluster I and is depicted as a solid line. Four clusters emerged for Affect factor scores and Sleep/Energy factor scores, whereas two clusters were retained for Urge factor scores. Plotting the cluster means against time revealed substantial heterogeneity in patterns of withdrawal symptomatology. For all measures, notable proportions (25%–70%) of the sample reported withdrawal trajectories that differed markedly from the prototypical pattern seen in group-level studies.

Affect factor scores. The prototypical group (Cluster I) contained 50 (31%) participants and showed a rise in symptoms from the quit day to Day 2 and then a sharp diminution in symptomatology thereafter. Clusters II–IV comprised 36 (23%), 42 (26%), and 31 (19%) participants, respectively. As is seen in Figure 2, Cluster I showed the most consistent improvement in symptoms after Day 2.

Urge factor scores. Cluster I contained 118 (74%) participants, and Cluster II contained 41 (26%) participants. Again, Cluster I members were distinctive in that their Urge scores declined steadily, dropping below baseline Urge reports by Day 7 of the quit attempt.

Sleep/Energy factor scores. The prototype, Cluster I, included 48 (30%) participants, whereas Clusters II–IV contained 39 (25%), 44 (28%), and 28 (18%) participants, respectively. Figure 2 shows that only Cluster I had the characteristic transient pattern, with scores increasing slightly above baseline on the quit date and dropping below baseline levels at all subsequent assessments.

Assessing Clinical Relevance

Screening Candidate Predictors

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The affect classification was not redundant with groupings based on the remaining symptoms. Thirty-seven (74%) of the prototypic affect smokers were also assigned to the prototypic urge group, whereas 28 (26%) of the atypical affect smokers were counted in the atypical urge group, $\chi^2(1, N = 159) = 0.002$, ns. Seventeen (34%) of the prototypic affect smokers were counted as prototypic in the sleep/energy analysis, and 78 (72%) of atypical affect smokers were counted as atypical, $\chi^2(1, N = 159) = 0.50$, ns. However, there was some overlap between the urge and sleep/ energy classifications. Twenty-nine (25%) of the prototypic urge smokers were counted as prototypic in the sleep/energy analysis, whereas 22 (54%) of atypical urge smokers were classified as atypical, $\chi^2(1, N = 159) = 6.84$, p < .01. Despite this overlap, we decided to retain both groupings as potential independent variables for model building, because 108 (69%) of the total sample fell into off-diagonal cells of the contingency table.

The elevation variable, model $\chi^2(1, N = 159) = 12.55, p < .001$, affect classification, model $\chi^2(1, N = 159) = 5.82, p = .01$, and urge classification, model $\chi^2(1, N = 159) = 5.48, p = .01$, were all significantly associated with relapse in univariate logistic models. Sleep/energy classification was not associated with relapse, model $\chi^2(1, N = 159) = 0.06, ns$.

Seventeen (34%) of the participants assigned to the prototypic affect group were counted as abstinent at the end of the trial, compared to 18 (17%) of atypical affect group members, $\chi^2(1, N = 159) = 6.11$, p = .01. Thirty-one (26%) of the participants assigned to the prototypic urge group were counted as abstinent, compared to 4 (10%) of atypical urge group members, $\chi^2(1, N =$ 159) = 4.84, p < .05. Relapse rates for sleep/energy typical and atypical groups were very similar; they were 10 (21%) and 25 (22%), respectively, $\chi^2(1, N = 159) = 0.56$, ns.

Multivariate Withdrawal Model

Based on the univariate findings, all of the withdrawal parameters except the sleep/energy classification were considered as candidate predictors in the multivariate model. Elevation, affect classification, and urge classification were all significant predictors when entered simultaneously in a saturated logistic model, so no variable was deleted. The top portion of Table 1 summarizes the estimated logistic regression coefficients associated with this model. The odds ratios reported in Table 1 indicate that, when overall symptomatology and urge classification are controlled, atypical affect participants had over 4.2 times the odds of being counted as relapsed by Day 30. Likewise, atypical urge members had 3.9 times the odds of relapse. A 1-SD increase in overall symptom elevation was associated with a 2.7-fold increase in the odds of relapse. The Hosmer-Lemeshow fit statistic for this final model suggests that it fit the data adequately, $\hat{C}(8, N) =$ 159) = 11.87, p = .16.

³ The goal of our cluster analyses was to subdivide the total sample into smaller groups that were comparatively homogeneous with respect to the shape of their withdrawal profiles. To assess whether this was achieved, we examined the variability in withdrawal ratings before and after clustering. Across cluster solutions and time points, the standard deviations of standardized symptom measures were generally reduced between 20%-40% after clustering, suggesting that the analyses succeeded in grouping together individuals with similar withdrawal trajectories.

² The temporal pattern of the transformed sleep/energy scores might be interpreted as a simple offset effect (Hughes et al., 1990). However, inspection of the raw Sleep/Energy scale scores (a simple sum of the relevant item scores) did show transient increases in these symptoms after the quit date, a finding that is consistent with other research examining raw scores on these symptoms (e.g., Jorenby et al., 1996). We suspect that this discrepancy results from the specification of orthogonal factors. In other words, raw scores on the items dominating the Sleep/Energy factor may reflect general withdrawal malaise, but this effect is mitigated when factor scores are stripped of variance shared with affect and urge. To some extent, this finding may reflect component overextraction (Wood, Tataryn, & Gorsuch, 1996). However, there is some evidence that objective measures of sleep quality poorly correspond with self-report assessments (Wetter, Fiore, Baker, & Young, 1995), a finding consistent with the interpretation that raw scores on sleep/energy items may be driven to some degree by method variance and general discomfort.



Figure 2. Standardized group symptom profiles derived from cluster analyses. For each measure, the prototypic group is designated Cluster I and is depicted as a solid line with open circles. B1 = 8 days precessation; B2 = 4 days precessation; QD = quit day. Remaining axis labels represent the number of days postquit.

Contribution of Nonwithdrawal Variables

Of all baseline smoker characteristics (plasma nicotine, plasma cotinine, FTQ, age of smoking initiation, regular smoking rate, number of quit attempts, baseline PANAS, CESD, and PS), only plasma nicotine, model $\chi^2(1, N = 157) = 4.98, p < .05$, and plasma cotinine, model $\chi^2(1, N = 157) = 4.39, p < .05$, significantly improved the model when they were added separately. These measures were significantly correlated with one another (r = .67, p < .001). Therefore, in building the combined model, we opted to include only plasma nicotine, the slightly stronger predictor. The middle portion of Table 1 displays the results of this logistic model. A 1-SD increase in plasma nicotine at baseline was

associated with a 1.7-fold increase in the odds of relapse. With plasma nicotine in the model, the odds ratios for the classification variables were altered slightly, but all withdrawal parameters remained significantly associated with the abstinence criterion. The Hosmer-Lemeshow fit statistic for this final model suggests that the model fit the data well, $\hat{C}(8, N = 157) = 6.62, p = .58$.

Influence of Postcessation Cigarette Use

Repeated measures ANOVAs that compared typical and atypical clusters on the pattern and amount of cigarette use across the cessation period did not reveal significant main effects or Cluster \times Time interactions for any subscale grouping.

Table 1Logistic Regression Results Assessing Relations BetweenWithdrawal Variables and Relapse

Predictor	β	SE (β)	Wald	р	OR
	Multi	variate with	ndrawal m	odel	
Elevation	0.99	0.26	13.45	<.001	2.67 per SD
Affect group	1.45	0.46	9.86	.002	4.24
Urge group	1.37	0.62	4.94	.026	3.93
С	ontributi	on of nonw	ithdrawal	variables	
Elevation	0.99	0.27	13.21	<.001	2.70 per SD
Affect group	1.34	0.47	8.13	.004	3.85
Urge group	1.39	0.63	4.87	.027	4.03
Plasma nicotine	0.53	0.25	4.51	.033	1.70 per SD
	Ι	apse-adjus	ted model		
Lapse status	4.51	0.71	40.60	<.001	91.00
Elevation	0.99	0.37	7.31	.006	2.69 per SD
Affect group	1.84	0.68	7,44	.006	6.34

Note. The odds ratio (OR) for the elevation and plasma nicotine measures reflects the OR for a 1-SD increase on each of these continuous measures. Reference groups for categorical variables were the prototypic groups for the symptom measures and nonlapsers for lapse status. For all measures, higher values of the OR indicate a higher probability of being counted as relapsed at the Day 30 assessment. Parameter estimates reported for all three analyses reflect results with all listed variables simultaneously included in the model.

A logistic regression analysis was performed to determine whether any withdrawal variables could improve on a prediction model once intrastudy smoking lapses were included as a covariate. As in the multivariate model, all withdrawal parameters except sleep/energy clusters were considered as candidate predictors. Lapse status was entered along with the remaining withdrawal parameters to form a saturated model, and withdrawal variables were deleted as necessary. Urge classification, $\chi^2(1, N =$ 159) = 1.85, p > .05, was deleted without a significant decrement in model fit. Affect classification and symptom elevation could not be deleted without worsening overall model fit, so they were retained. The bottom portion of Table 1 displays the results of the final model. The Hosmer-Lemeshow fit statistic for this final lapse-adjusted model suggested that the model fit the data well, $\hat{C}(8, N = 159) = 7.48, p = .49$.

In a final set of assessments, clusters were divided into subgroups according to the latency at which the first lapse was recorded. This was done only for affect and urge cluster solutions, because these were the only symptom domains associated with relapse. In each cluster, at least five of the six possible lapse histories were represented; some continuously abstinent smokers were found in each cluster.⁴ Moreover, intracluster group profiles showed fairly tight correspondence across time. Coefficient alphas across groups within each cluster were as follows: Affect I, .89; Affect II, .95; Affect III, .87; Affect IV, .93; Urge I, .98; Urge II, .78. Figure 3 illustrates this correspondence, depicting the intracluster withdrawal profiles as a function of latency to first reported lapse for the affect clusters. Two interpretations stem from these descriptive findings. First, the tight coherence among within81

cluster subgroups suggests that the cluster analyses performed as they are purported to, that is, they isolated homogeneous subgroups of smokers who showed the same symptom shape regardless of when they started smoking. Second, the fact that smokers with diverse lapse histories were assigned to each cluster suggests that symptom shape does not simply parrot postcessation smoking experiences.

Symptom Slopes and Relapse

Table 2 presents the results of the foregoing logistic regression models, with simple slope measures substituted for cluster distinctions. In each model, slope measures remained significantly associated with relapse, suggesting that symptom-relapse associations did not depend on clustering per se or on a particular sample split. Some differences in other model coefficients emerged when slopes were used. Most notably, the odds ratio associated with the elevation variable was increased in all models, and plasma nicotine was no longer a significant predictor of relapse. As with the cluster classifications, Hosmer-Lemeshow goodness-of-fit statistics indicated that all three models fit the data adequately: multivariate withdrawal model, $\hat{C}(8, N = 159) = 10.74, p = .22$; nonwithdrawal variables model, $\hat{C}(8, N = 157) = 10.19, p = .25$; lapse-adjusted model, $\hat{C}(8, N = 159) = 9.82, p = .28$.

When elevation and affect slopes were forced into a logistic model of relapse, the prototypic-atypical cluster distinction improved the model when they were entered at a subsequent step, model $\chi^2(1, N = 159) = 3.68, p = .05$. This suggests that cluster distinctions may capture features of withdrawal profiles not reflected in slopes and that these features are related to relapse. The urge cluster distinction did not significantly improve a model incorporating slope and elevation information, model $\chi^2(1, N = 159) = 1.88, ns$. This makes sense when considering the two cluster profile shapes (see Figure 2); urge cluster group profiles differed chiefly in slope per se across time.

External Correlates

Elevation was related to FTQ scores (r = .17, p < .05), negative PANAS scores (r = .40, p < .01), CESD scores (r = .35, p < .01), and PS scores (r = .16, p < .05). No significant differences were found between prototypic and atypical cluster members in either the affect or urge analyses on the smoker characteristic measures (plasma nicotine, plasma cotinine, FTQ, age of smoking initiation, regular smoking rate, number of quit attempts, baseline PANAS,

⁴ Subgroup tallies for each cluster (listed as *n* continuously abstinent, *n* reporting first lapse on Day 1, *n* reporting on Day 2, *n* reporting on Day 7, *n* reporting on Day 14, and *n* reporting on Day 30, respectively) were as follows: Affect I, 13, 26, 4, 6, 1, 0; Affect II, 5, 25, 2, 2, 0, 2; Affect III, 6, 22, 3, 8, 2, 1; Affect IV, 5, 19, 2, 3, 1, 1; Urge I, 26, 69, 5, 13, 2, 3; Urge II, 3, 23, 6, 6, 2, 1. Clearly, lapse reports were not evenly distributed across the postcessation period within clusters, and clusters differences in subgroup size, at least in part, reflect the lapse history of the sample, with early lapse reports being quite common. Cross-cluster differences, especially in the number of continuously abstinent persons, are congruent with the results of the logistic regression models.



Figure 3. Affect score profiles for subgroups formed on the basis of latency to first reported cigarette in each affect cluster. Profiles are depicted in factor score units. QD = quit day. (Subgroup *ns* are given in Footnote 4.)

CESD, and PS). None of these measures was significantly correlated with affect or urge slopes. No connections between precessation variables and either cluster distinctions or slopes were found in models with elevation statistically controlled.

Discussion

One major aim of this research was to evaluate the robustness of two earlier findings—the existence of marked heterogeneity in postcessation symptom patterns, and the connection between temporal symptom parameters and relapse (Piasecki et al., 1998). A conceptual replication of these findings was deemed especially important given the historical inconsistency of withdrawal measures (Patten & Martin, 1996a). Because we sought a relatively "risky" test of these phenomena, we examined data from a study that was methodologically quite different from our previous studies. This sample comprised unaided quitters, used a different withdrawal measure, and assessed withdrawal relatively infrequently during a shorter postcessation window. Despite these differences, we obtained evidence corroborating earlier findings.

Examination of temporal profiles in three distinct symptom domains revealed that a substantial proportion of the sample reported atypical withdrawal patterns for each symptom class. After controlling for overall symptom severity (elevation), the prototypic-atypical distinction for the Affect and Urge subscales was found to be associated with smoking status at follow-up. After lapse status was statistically controlled, affect classification still significantly improved the logistic model of relapse. Temporal variability in Affect factor scores was more robustly related to clinical outcomes than was variability in other withdrawal subdomains. This is consistent with our speculation that affective processes play a central motivational role in the return to smoking. Withdrawal variables considered alone constituted an adequate fitting model of relapse likelihood, but measures of baseline plasma nicotine and cotinine yielded an orthogonal improvement of this model. This and other findings suggest that temporal variance in symptom measures does not simply mediate the influence of nicotine dependence. Finally, connections between symptom patterns and relapse were robust, regardless of whether they

Table 2	
Logistic Regression Results Assessing Relations Between	n
Withdrawal Variables and Relapse, Substituting Simple	
Slope Measures for Cluster Distinctions	

Predictor	β	SE (β)	Wald	р	OR
	Multi	variate with	ndrawal m	odel	
Elevation	1.21	0.29	17.82	<.001	3.37 per SD
Affect slope	0.89	0.26	11.64	<.001	2.44 per SD
Urge slope	0.70	0.27	7.08	.008	2.02 per SD
C	ontributi	on of nonw	ithdrawal	variables	
Elevation	1.18	0.29	16.62	<.001	3.27 per SD
Affect slope	0.80	0.26	9.34	.002	2.22 per SD
Urge slope	0.62	0.27	5.18	.023	1.85 per SD
Plasma nicotine	0.40	0.24	2.81	.093	1.49 per SD
	I	_apse-adjus	ted model		
Lapse status	4.53	0.72	39.82	<.001	92.79
Elevation	1.21	0.40	9.03	.003	3.35 per SD
Affect slope	1.04	0.38	7.37	.006	2.82 per SD

Note. The odds ratio (OR) for all predictors except lapse status reflects the OR for a 1-SD increase on each of these continuous measures. The reference group for lapse status is nonlapsers. For all measures, higher values of the OR indicate a higher probability of being counted as relapsed at the Day 30 assessment. Parameter estimates reported for all three analyses reflect results with all listed variables simultaneously included in the model.

were represented as cluster-based categorical variables or simple slope measures. This suggests that the findings are not an artifact associated with any particular data reduction technique. Thus, this research replicates our earlier work that showed that mean symptom elevation and the trajectory of symptoms are strongly associated with smokers' eventual relapse fates. These findings differ markedly from conclusions of many existing studies (Hughes et al., 1990; Patten & Martin, 1996a).

Earlier, we noted that applying unique analytic techniques to accepted measures may throw new light on the meaning of those measures. What is the core of the meaningful information in withdrawal measures? We constructed symptom subscales that were essentially orthogonal to one another in an attempt to isolate the core, clinically relevant sources of variance in withdrawal scales. Plotting these scales against time in the aggregate showed that only urge and affect ratings conformed to conventional expectations regarding the time course of withdrawal symptoms. Other analyses suggested that, across time, the unique variance in each of these measures was fairly independent. Very different profile shapes were obtained for the two symptom classes in cluster analyses. Of course, this may result in part from metric properties of the computed scales or from the imprecision inherent in cluster analysis. However, the finding that cluster groupings based on both urge and affect were associated with relapse when entered simultaneously into logistic regression models suggests that the two measures may reflect different processes. This conclusion is supported by the fact that membership in the atypical groups differed substantially across the two measures. Such findings suggest that factors that give rise to trajectories of urge and affect self-reports may exert somewhat independent influences on relapse. It is possible, for instance, that some smokers may be urge reactive whereas others are affect reactive. This conjecture must remain speculative in lieu of further research.

Descriptive research has shown that urges are often higher during continued smoking than during an abstinence attempt (e.g., Hughes, 1992). Hence, the pattern of increasing urge scores seen in the atypical urge group in this sample may simply reflect the reestablishment of typical smoking behavior across the study period. This might explain why, when the study controlled for lapses, the relation between urge cluster membership and relapse was attenuated. If smoking is associated with elevated urges, a measure of lapse occurrence would be statistically redundant with urge growth, causing urge classification to drop out of the prediction model. Of course, there is independent evidence that urge ratings acutely foreshadow first lapses (e.g., Shiffman, Paty, Gnys, Kassel, & Hickcox, 1996; Shiffman et al., 1997).

A great deal of evidence links affect to smoking motivation and demonstrates that affect may be modified by smoking (Brandon, 1994). Nonetheless, it seems likely that patterns of affect ratings after cessation reflect more than simple smoking status. For instance, affect patterns may reflect the cumulation of stressors, the mood-related consequences of progressive sleep deprivation, the loss of reinforcement, the emergence of a depressive episode, and so on. Postcessation lapses may acutely alleviate exacerbations in negative affect, but, in many cases, smoking would not be expected to remove the source of mounting dysphoria. Ex-smokers seeking more consistent relief from negative mood may turn to cigarettes more and more frequently, eventuating in relapse. This account is likewise consistent with the finding that affect classification contributed to a logistic model of relapse even after early lapses were controlled.

Another major aim of this research was to investigate precessation variables that might foretell which individuals are at risk for atypical symptom dynamics. Although an array of precessation measures was available to correlate with cluster membership and symptom slopes (Shadel et al., 1998), we restricted our precessation analyses to dependence and affect measures for theoretical reasons. These analyses revealed no significant relations between these measures and either cluster membership or symptom slopes. Symptom elevation was related to baseline FTQ scores and a variety of affect measures. This finding corresponds with results of a recent study by Gilbert and colleagues (1998) that show robust connections between trait measures of affect and the broad-span severity of abstinence symptomatology. The dearth of connections between precessation variables and various representations of symptom trajectory in this sample and two previous studies (Piasecki et al., 1998), coupled with the prediction of severity from baseline measures, suggests that postcessation events and stimuli may account for the dynamic features of abstinence symptomatology. This predictive dissociation and the unique contributions of elevation and trajectory to logistic models of relapse confirm the independence of these two facets of withdrawal.

Relations between smoking and symptom measures in this research were complex, and these connections are in need of further research. Symptom measures were robustly related to smoking status at follow-up, suggesting a strong connection between the two variables. Lapses might directly cause both relapse and atypical withdrawal patterns. A strong version of a lapse-based account might ascribe little or no causal role for withdrawal patterns in relapse. Ancillary analyses aimed at characterizing the relations between lapses, 30-day outcomes, and symptom patterns challenge such an account. Smokers classified in typical versus atypical groups did not differ in the pattern or rate of smoking after cessation. Moreover, including lapse status in logistic models as a covariate did not eliminate the relation between affect symptom patterning and smoking status at follow-up, suggesting that withdrawal dynamics contribute nonredundant information regarding relapse risk. Finally, continuously abstinent smokers and smokers with diverse latencies to the first lapse were represented in each cluster. This demonstrates that symptom pattern differences do not depend upon particular lapse histories.

One could legitimately question whether symptom reports gathered long after cessation truly reflect withdrawal. One response would be to equate concretely the construct with the measures and claim that the analyses must capture withdrawal if they are based on widely used and validated instruments. This argument is, of course, simplistic. A clear conceptual definition of withdrawal is required to address this question. For instance, if lapses trigger larger symptomatic increases in abstinent versus continuing smokers, would such symptomatic reactions to drug use constitute withdrawal responses? Moreover, if smoking is used to suppress the emergence of affective disorder, is the affective disorder a withdrawal symptom complex? At present, the field's datagathering and analytic capabilities may have outstripped the complexity and clarity of its definitions.

Symptom trajectory was related to follow-up smoking status in this research, regardless of whether it was represented by categorical cluster assignments or continuous slope measures. This naturally raises questions about the value of cluster analysis and other categorical data reduction methods in withdrawal heterogeneity research. Can cluster analysis and similar techniques contribute anything to the study of withdrawal that slopes cannot? The best statistical representation of trajectory variance will need to be scrutinized in future research. We suspect that continuous measures (simple slopes, correlations between individual profiles, and theoretical function forms, etc.) will be found to be useful tools for assessing relations among symptom trajectory, precessation variables, postcessation events, and clinical outcomes. Nonetheless, cluster analyses may continue to be valuable at initial stages of the data analytic process, providing useful descriptive information not captured by other representations of trajectory. Clustering isolates a family of common profiles present in a sample, and these may have many useful diagnostic applications. For instance, modal symptom profiles can provide a sense of the frequency with which particular patterns are observed, and, indeed, certain profiles might prove to have taxonic significance. Moreover, examining these profiles may help researchers locate particular time periods in which symptoms worsen, permitting hypothesis generation and the construction of sensitive continuous indicators of trajectory. In the present research, affect cluster distinctions were found to contribute information to a model of relapse that controlled for symptom elevation and affect slope. We attribute this finding to the sensitivity of clustering to transient but potentially important vicissitudes in withdrawal. This is consistent with the fact that profile shape may be too complex and rich to be captured by a simple change index.

Limitations of our study must be considered in evaluating this research. For instance, the follow-up window in this study was short, and the point-prevalence window for the abstinence criterion overlapped with the period in which withdrawal was being assessed. These aspects of the study prevent us from definitively assessing the temporal precedence of symptom change in the return to smoking. Other limitations of the study include the fairly small sample size, the fact that daily symptom data were not collected, the exclusive use of self-report measures, and the brief postcessation study period (i.e., only 30 days). The small sample size means that some of our findings may not generalize well to self-quitters. The sample comprised self-quitters, which we viewed as an important distinction from our past studies of nicotine patch users. Nonetheless, because they were willing to commit to timeconsuming data collection procedures, the present sample may not be highly representative of self-quitters in general. Moreover, the lack of daily data and the brief follow-up period certainly affected the shapes of the postcessation profiles obtained. This also means that the profile shapes cannot be easily compared with those generated in our previous research (Piasecki et al., 1998). In addition, some of the smokers labelled as abstinent at 30-days postcessation certainly went on to relapse at a later time (Gilpin, Pierce, & Farkas, 1997). The lack of objective (i.e., non-selfreport) data means that we know virtually nothing about the physiological and behavioral changes that smokers are undergoing late in the postcessation period. For instance, we do not know whether smokers are actually experiencing greater neural activity in systems that process negative affect; thus, the exacerbation in rated withdrawal symptoms reported by some participants may merely reflect changes in their perception or scaling of affective signals. Reactive effects associated with repeated self-reports of mood states may also have contributed to some observed symptomatic change (Gilbert et al., 1998). Future research should be designed to assess these concerns, incorporating precessation run-in assessments to minimize subsequent reactivity and including periodic objective assessments after the quit date.

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Appendix

Items Constituting Each Subscale and Their Loadings on Those Factors

Item	Loading	
Factor 1: Affect		
Irritability	.826	
Anxiety	.787	
Impatience	.779	
Restlessness	.752	
Difficulty concentrating	.684	
Sadness	.603	
Appetite increase	.476	
Factor 2: Urge		
Urge to smoke today	.760	
Miss a cigarette	.752	
Wanting/craving a cigarette	.749	
If could smoke freely, want cigarette now	.729	
Thinking of cigarettes more than usual	.715	
If permitted to smoke, refuse cigarette now ^a	.644	
Factor 3: Sleen/Energy		
Dismuted sleen	663	
Trouble falling asleep	.654	

^a Reverse scored in all analyses.

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