Published in final edited form as:

J Consult Clin Psychol. 2002 April ; 70(2): 356–361.

Patterns of Change in Depressive Symptoms During Smoking Cessation: Who's at Risk for Relapse?

Ellen S. Burgess, Richard A. Brown, Christopher W. Kahler, Raymond Niaura, David B. Abrams, Michael G. Goldstein, and Ivan W. Miller
Brown University School of Medicine

Abstract

The authors examined patterns of change in depressive symptoms during smoking cessation treatment in 163 smokers with past major depressive disorder (MDD). Cluster analysis of Beck Depression Inventory (A. T. Beck, C. H. Ward, M. Mendelson, J. Mock, & J. Erbaugh, 1961) scores identified 5 patterns of change. Although 40% of participants belonged to clusters characterized by increasing depressive symptoms during quitting (rapid increasers, $n = 31$, and delayed increasers, $n = 35$), almost 47% were in clusters characterized by decreasing symptoms (delayed decreasers, $n = 24$, and rapid decreasers, $n = 52$). Both rapid and delayed increasers had especially poor smoking cessation outcomes. Results suggest that among smokers with an MDD history there is substantial heterogeneity in patterns of depressive symptoms during quitting and that patterns involving increased symptoms are associated with low abstinence rates.

A disproportionately high percentage of smokers participating in smoking cessation studies are found to have a lifetime history of major depression disorder (MDD). The prevalence of a history of MDD in smokers entering treatment has ranged from 22% to 61% (Ginsberg, Hall, Reus, & Muñoz, 1995; Glassman et al., 1988; Hall, Muñoz, & Reus, 1994; Hall et al., 1996; Kinnunen, Doherty, Militello, & Garvey, 1996). This is considerably higher than the lifetime prevalence of MDD in the general population, which is approximately 17% (Kessler, 1994). Population and community studies have confirmed the importance of a history of MDD in relation to smoking status and nicotine dependence. In a catchment area survey (Glassman et al., 1990), an MDD history was more common in smokers than nonsmokers and was associated with greater frequency of regular smoking. Likewise, in a community survey, lifetime history of MDD was associated with increased smoking prevalence in both men and women (S. Cohen, Schwartz, Bromet, & Parkinson, 1991).

A history of MDD appears to impede efforts at smoking cessation. In treatment studies, Glassman and colleagues found positive MDD history predicted poorer smoking outcome at both 4-weeks (Glassman et al., 1988) and 10-weeks postquit date (Glassman et al., 1993). However, in treatment studies with longer follow-ups, MDD history failed to predict smoking status at 52-weeks postquit date (Ginsberg et al., 1995; Hall et al., 1994). Survey research also

Correspondence concerning this article should be addressed to Richard A. Brown, Brown University School of Medicine/Butler Hospital, 345 Blackstone Boulevard, Providence, Rhode Island 02906. E-mail: richard_brown@brown.edu.

Ellen S. Burgess, Richard A. Brown, and Christopher W. Kahler, Department of Psychiatry and Human Behavior, Brown University School of Medicine/Butler Hospital; Raymond Niaura, David B. Abrams, and Michael G. Goldstein, Department of Psychiatry and Human Behavior, Brown University School of Medicine/Miriam Hospital; Ivan W. Miller, Department of Psychiatry and Human Behavior, Brown University School of Medicine/Rhode Island Hospital.

Ellen S. Burgess is now at Massachusetts Mental Health Center, Harvard Medical School. Michael G. Goldstein is now at Bayer Institute of Health Care Communication, West Haven, Connecticut.

This study was partially supported by National Institute on Drug Abuse Grant DA08511 to Richard A. Brown. We gratefully acknowledge Suzanne Sales, Jessica Whitely, and Michelle Ricci for their assistance on this project.

indicates a deleterious effect of MDD history on quitting smoking; individuals with a history of MDD are less likely to have quit smoking than individuals without such a history (Glassman et al., 1990).

Why do smokers with a history of MDD have poor outcomes in smoking cessation? One theory suggests that smokers with a history of MDD self-medicate with nicotine and subsequently begin to experience an increase in depressive symptoms when they quit smoking (Hughes, 1988). This increase in depressive symptoms may then undermine their quit attempt because of decreased motivation or decreased self-efficacy. Indeed, smokers with an MDD history are more likely to report elevated depressed mood while quitting than are smokers without an MDD history (Breslau, Kilbey, & Andreski, 1992; Covey, Glassman, & Stetner, 1990; Ginsberg et al., 1995; Hall et al., 1994, 1996), and increases in depressed mood immediately after quitting predict relapse to smoking (Covey et al., 1990; Hall et al., 1996). Finally, Covey, Glassman, and Stetner (1997) found that a greater percentage of smokers with a history of recurrent MDD (30%) experienced a new major depressive episode following quitting as compared with smokers with a single past episode (17%) or no history of MDD (2%).

Given the relationship between depressive symptoms–depressed mood and smoking cessation in smokers with a history of MDD, several attempts have been made to increase smoking cessation success in history positive smokers by adding coping skills for depression–negative mood to standard smoking cessation treatments. Two studies have found significant effects of mood-management skills for smokers with an MDD history when the experimental treatment had greater therapist contact time than the control (Hall et al., 1994, 1998). However, in a study that equated for therapist contact between conditions, no significant differences were found between standard smoking cessation treatment and standard treatment with the addition of a mood-management component (Hall et al., 1996). Also, in each of the Hall et al. studies (Hall et al., 1994, 1996, 1998), mood management did not attenuate postcessation increases in depressive symptoms among smokers with an MDD history.

In a recent study of smokers with a history of MDD, we found that cognitive–behavioral therapy for depression (CBT-D), when incorporated into standard smoking cessation treatment (ST), led to better outcomes for heavier smokers and for smokers with a history of recurrent, but not of single-episode, MDD (Brown et al., 2001). CBT-D did not, however, decrease depressive symptoms prior to or after quitting. Also, contrary to expectations, depressive symptoms in the sample as a whole did not increase significantly following quit date. These findings led us to question how many smokers with an MDD history show a pattern of increasing depressive symptoms during quitting and what other patterns of change in depressive symptoms could be identified within this population. In this article, we use cluster analysis to classify patterns of change in depressive symptoms in smokers with a history of MDD. We then examine the relationship of these patterns of change to treatment outcome and to baseline characteristics.

Method

Participants

One hundred seventy-nine smokers with a history of MDD participated in a smoking-cessation-treatment study in which they were randomized to a standard smoking cessation intervention or to a standard smoking cessation intervention combined with coping skills for depression (CBT-D; see Brown et al., 2001, for a complete description). Both treatments consisted of eight 2-hr sessions over 6 weeks. Quit date began on awakening on the morning of the fifth session, 4 weeks after Session 1. Sessions occurred weekly, except for the sixth session, which took place 3 days after quit date. The standard smoking cessation treatment was a comprehensive, cognitive–behavioral program that included self-monitoring, self-management, nicotine fading, relapse prevention, and social support enhancement. The CBT-D condition consisted

of an integration of the standard, cognitive-behavioral smoking cessation skills and cognitive-behavioral coping skills for depression that included daily mood ratings, pleasant-event scheduling, cognitive restructuring, and assertiveness training. Neither condition provided pharmacotherapy.

Participants were included if they were between the ages of 18 and 70 years; had regularly smoked cigarettes for at least 1 year; were currently smoking at least 10 cigarettes per day; and had a past history of MDD according to the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; *DSM-III-R*; American Psychiatric Association, 1987), as determined by the Structured Clinical Interview for *DSM-III-R*—Non-patient edition (SCID-NP, Version 1.0; Spitzer, Williams, Gibbon & First, 1990). Exclusion criteria were (a) *DSM-III-R* diagnosis of current MDD, dysthymia, or other Axis I disorder; (b) *DSM-III-R* diagnosis of current psychoactive substance abuse or dependence within the past 6 months (other than nicotine); (c) current use of psychotropic medication; (d) current weekly psychotherapy; (e) current use of other tobacco products; and (f) intent to use pharmacological aid to cessation. The two treatment conditions are described in detail elsewhere (Brown et al., 2001).

Of the 179 participants randomized to treatment, 107 (59.8%) were women and 94 (52.5%) were married or living with a partner as if married. The mean age of the sample was 45.1 years ($SD = 9.3$), and the mean number of years of education completed was 14.5 ($SD = 2.5$). Almost all participants ($n = 174$, 97.2%) identified themselves as White. One participant was African American, and 4 were of other ethnic origins. Prior to treatment, participants reported smoking an average of 27.3 ($SD = 11.3$) cigarettes per day and had been smoking for an average of 27.1 years ($SD = 9.5$). The sample mean on the Fagerstrom Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) was 6.4 ($SD = 1.8$), and saliva cotinine levels averaged 383.7 ng/ml ($SD = 170.6$) at baseline. All but 7 participants (3.9%) had made a prior quit attempt. The sample mean on the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) administered immediately prior to Session 1 was 7.8 ($SD = 6.31$). Ninety-three participants were randomized to the ST condition, and 86 were randomized to CBT-D.

Measures

At the baseline assessment, participants provided demographic and background information such as age, gender, years of education, marital status, number of years of regular smoking, average number of cigarettes smoked per day, and number of previous smoking cessation attempts. Lifetime and current *DSM-III-R* Axis I diagnoses were determined with the SCID-NP. Severity of nicotine dependence was assessed with the FTND, and level of depressive symptoms was assessed with the BDI. At Session 1, negative mood was assessed with the Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1971), and depressive symptoms again were assessed with the BDI. The BDI was also completed at Session 5 (quit date; 4 weeks after Session 1), Session 7 (1 week after quit date), and Session 8 (end of treatment; 2 weeks after quit date). Participants rated their level of depressive symptoms during the previous week.

Self-reports of smoking status were collected at each treatment session from quit date to the end of treatment and by telephone at 1-, 6-, and 12-month follow-up. Outcome analyses were based on 7-day point-prevalence abstinence (i.e., reported abstinence of at least 7 days prior to the assessment day). Participants' reports of abstinence were verified biochemically with alveolar carbon monoxide (CO) using a CMD/CO Carbon Monoxide Monitor (Model 3110; Spirometrics, Inc., Auburn, ME) and with a 2-ml saliva sample assayed for cotinine by the American Health Foundation, Valhalla, New York. Abstinence was confirmed by a combination of $CO \leq 10$ ppm and cotinine ≤ 46 ng/ml (Cummings & Richard, 1988). In those

few cases where biochemical verification could not be obtained (6.5%), self-reported abstinence was verified through interviews with significant others.

Results

Brown et al. (2001) found no significant baseline differences between treatment groups, and there was no significant main effect of treatment on 7-day point prevalence at any assessment point. Thus, we collapsed our data across treatment conditions. However, to be conservative, we used treatment condition as a covariate in predicting 7-day point prevalence.

Cluster Analysis

Cluster analysis is an exploratory analytic method used to identify natural groupings of observations according to their relative similarity to or distance from each other on a given set of measures (Johnson & Wichern, 1992). We used cluster analysis to group participants into clusters according to the shape of their depressive symptom profiles while quitting smoking. Prior to analysis, BDI scores at Sessions 1, 5 (quit date), 7 (1 week after quit date), and 8 (2 weeks after quit date) were standardized within each case to remove elevation and scatter (Cronbach & Gleser, 1953) from symptom profiles. Cluster analysis was then conducted on these standardized scores in SAS using PROC FASTCLUS (SAS Institute, 1990), which is similar to the *k*-means clustering algorithm. This method was chosen over hierarchical agglomerative clustering methods because we wanted to include participants with some missing data. In the FASTCLUS procedure, clustering is done based on Euclidean distances computed from the clustering variables. Observations are assigned to the same cluster if they are very close together and are assigned to different clusters if they are far apart.

Although PROC FASTCLUS would allow for inclusion of participants with only one completed BDI, we analyzed only participants who completed a BDI at Session 1 and at least one BDI on or after quit date (i.e., Sessions 5, 7, or 8) so that some estimate of change could be obtained. This inclusion criterion allowed us to utilize data from 163 of 179 participants. Chi-square analyses and *t* tests revealed that, of the baseline variables investigated in this study, included participants differed from those not included only in that they smoked fewer cigarettes per day prior to treatment, $t(177) = 2.45, p = .02$.

We examined two-, three-, four-, five-, six-, and seven-cluster solutions. The purpose of using cluster analysis was not to “test” how many unique clusters were in these data; rather, we wanted to capture as much variability in profiles of depressive symptoms over time as possible, while also providing sufficiently large cell sizes for meaningful statistical comparisons (20 subjects per cell was chosen as a cut-off so that we could detect mean differences between the smallest clusters of one standard deviation with a power of .80 using an alpha of .05; J. Cohen, 1988). A five-cluster solution appeared to meet these objectives and to fit the data well. Using the pseudo *F* statistic, a method of comparing the total amount of variance accounted for by a given cluster solution with the number of clusters and observations utilized (SAS Institute, 1990), we found relative peaks for the two-, five-, and seven-cluster solutions, suggesting that these cluster solutions were relatively optimal. We chose the five-cluster solution, which accounted for 60.9% of the variance in the clustering variables and had over 20 observations in each cluster. By contrast, the two-cluster solution accounted for only 30.4% of the total variance and the seven-cluster solution, which accounted for 70.7% of the total variance, yielded two clusters with less than 20 observations.

Profile Comparisons

We used the random-effects model for continuous responses (Laird & Ware, 1982) to examine whether the clusters differed in the shape of their profiles during treatment. This model accounts

for correlation between outcomes measured on the same person. It also allows regression parameters to be estimated even though assessments may be missing for some participants. Analyses were conducted in SAS using PROC MIXED (SAS Institute, 1997). Time was dummy-coded with Session 1 as the reference period so that significant BDI changes could be detected. BDI scores were square-root transformed to correct positive skewness.

As expected, a significant Cluster $\times$ Time interaction indicated that the clusters differed in the shape of their BDI profiles during treatment, $F(12, 409) = 37.75, p < .01$. Transformed BDI scores during treatment are shown for each cluster in Figure 1. Two of the five clusters were characterized by an increasing pattern of depressive symptoms. Cluster 1 (rapid increasers) included 31 participants. This group was characterized by marked and sustained elevations in BDI scores at Session 5 ($B = 1.33, SE = 0.15, p < .01$), Session 7 ($B = 1.23, SE = 0.16, p < .01$), and Session 8 ($B = 1.32, SE = 0.14, p < .01$) compared with Session 1. Cluster 2 (delayed increasers, $n = 35$), on the other hand, was characterized by significantly lower BDI scores at Session 5 ($B = -0.37, SE = 0.14, p < .01$), a tendency toward higher BDI scores at Session 7 ($B = 0.25, SE = 0.15, p = .10$), and significantly higher BDI scores at Session 8 ($B = 0.54, SE = 0.13, p < .01$).

Cluster 3 (brief reactors, $n = 21$) comprised 21 participants. This group demonstrated a relatively large increase in BDI scores at Session 5 ($B = 1.20, SE = 0.17, p < .01$), followed by a rapid decrease in BDI scores. Depressive symptoms at Session 7 ($B = 0.29, SE = 0.19, p = 0.14$) and Session 8 ($B = -0.17, SE = 0.17, p = 0.32$) were not significantly different from Session 1 levels.

Two clusters were characterized by a decreasing pattern of BDI scores. Cluster 4 (delayed decreaseers, $n = 24$) showed a nonsignificant decrease in BDI scores at Session 5 ($B = -0.22, SE = 0.16, p = .18$), a significant increase at Session 7 ($B = 0.40, SE = 0.17, p = .02$), and a significant and relatively large decrease at Session 8 ($B = -1.23, SE = 0.16, p < .01$). Finally, Cluster 5 (rapid decreaseers, $n = 52$) showed a more consistent and rapid decrease in depressive symptoms. Within this cluster, BDI scores were significantly lower at Session 5 ($B = -0.95, SE = 0.11, p < .01$), Session 7 ($B = -1.29, SE = 0.12, p < .01$), and Session 8 ($B = -0.57, SE = 0.11$) compared with Session 1.

An analysis of variance (ANOVA) of Session 1 BDI scores (square-root transformed) was conducted to determine whether clusters differed in initial level of depressive symptoms in addition to their previously demonstrated differences in shape. This analysis indicated that depressive symptoms differed significantly by cluster at the start of treatment, $F(4, 168) = 5.12, p < .01$. Pairwise comparisons were made with Tukey's procedure with alpha set at .05. Results indicated that brief reactors reported lower BDI scores at Session 1 than rapid decreaseers, delayed reactors, and delayed increasers. Also, rapid decreaseers reported significantly more depressive symptoms than rapid increasers at Session 1.

Cluster Membership and Smoking Outcome

Observed rates of abstinence within each cluster at posttreatment, 1-month, 6-month, and 12-month follow-ups are depicted in Figure 2. The effect of cluster membership on smoking outcomes was analyzed using generalized estimating equations (GEE; Liang & Zeger, 1986; Zeger & Liang, 1986). Analyses were conducted in SAS using PROC GENMOD with an unstructured covariance matrix specified. Session 1 BDI was included as a covariate in the GEE analysis so that the cluster effect could be assessed while controlling for differences in initial levels of depressive symptoms. Treatment condition and time of assessment were also used as covariates. Cluster membership was dummy-coded with rapid increasers as the reference group. This cluster was chosen as the reference group because it best represents the

pattern of increasing depressive symptoms that is hypothesized to occur among many smokers with a depression history following smoking cessation.

Results of the GEE analysis indicated that the odds of abstinence in the sample as a whole tended to decrease over time, odds ratio (OR) = 0.88, $p = .08$. Elevations in Session 1 BDI scores (square-root transformed) were also associated with lower odds of abstinence, OR = 0.69, $p < .01$. Treatment condition, however, did not predict outcome, $p = .42$.

The effect of cluster membership on outcome was significant. Across assessments, rapid decreaseers and brief reactors were significantly more likely to abstain than rapid increaseers, OR = 5.95, $p < .01$, and OR = 2.92, $p = .03$, respectively. Follow-up analyses indicated that rapid decreaseers also were more likely to abstain than delayed increaseers, OR = 3.55, $p < .01$, and delayed decreaseers, OR = 2.69, $p = .03$. However, there was a significant interaction between cluster and time. The effect of time among delayed increaseers and brief reactors was significantly different from the effect of time among rapid increaseers, $ps < .03$. The likelihood of abstinence among delayed increaseers (OR = 0.66, $p = .02$) and brief reactors (OR = 0.58, $p < .01$) decreased over time, whereas the effect of time was nonsignificant for other clusters, $ps > .35$. There were no significant interactions between treatment and cluster membership in predicting smoking cessation outcome.

Baseline Predictors of Cluster Membership

We performed a stepwise discriminant-function analysis (with alpha set at .15 for both inclusion and removal) to determine which baseline variables most strongly predicted cluster membership. Variables were chosen from three domains of interest: demographics (age, gender, years of education, and marital status), smoking-related variables (FTND score, mean number of cigarettes smoked per day pretreatment, baseline saliva cotinine, number of years of regular smoking, and number of previous quit attempts), and depression-related variables (Session 1 BDI score, history of recurrent major depression, age at onset of first major depression, time since last major depression, and total mood disturbance score from the POMS at Session 1). We also included treatment condition to test whether treatment condition affected depressive symptoms differentially during treatment.

The following variables were selected in the given order: Session 1 BDI score, history of recurrent major depression, and age at onset of first major depression. Mean values on these variables by cluster are presented in Table 1. Differences between clusters on Session 1 BDI were reported above and are not repeated here (see Table 1). An ANOVA showed that clusters differed significantly on age of onset of MDD, $F(4, 152) = 4.34$, $p < .01$ (valid data were not available on this variable for 7 participants). Tukey's procedure for pairwise comparisons revealed that rapid increaseers had a significantly earlier age of onset than rapid decreaseers. Also, a chi-square test indicated that clusters varied significantly in frequency of recurrent depression, $\chi^2(4, N = 161) = 10.56$, $p = .03$ (valid data were not available on this variable for 2 participants). Follow-up pairwise comparisons showed that delayed increaseers were less likely to have a history of recurrent major depression than were rapid increaseers, $\chi^2(1, N = 66) = 9.10$, $p < .01$, and delayed decreaseers, $\chi^2(1, N = 58) = 4.38$, $p = .04$. Rapid increaseers also had a tendency to have higher rates of recurrent depression history than rapid decreaseers, $\chi^2(1, N = 82) = 3.05$, $p = .08$, and brief reactors, $\chi^2(1, N = 52) = 3.81$, $p = .05$.

Discussion

Although it often has been assumed that smoking cessation in individuals with an MDD history will be associated with increases in depressive symptoms, we found that among this population, there is substantial heterogeneity in patterns of depressive symptoms during quitting. Although about 40% of our sample exhibited a profile involving increased depressive symptoms, almost

47% exhibited a profile of decreasing depressive symptoms from baseline to 2-weeks postquit date. Of importance, depressive symptom profiles predicted smoking status during the 1 year after quit date, with abstinence rates generally lower in increasers compared with decreasers. This finding is consistent with other studies that have shown that an increase in depressive symptoms while quitting increases the risk for relapse (Covey et al., 1990; Hall et al., 1996).

Baseline depressive symptoms, age at onset of first MDD, and history of recurrent depression may be useful in predicting which smokers with a history of MDD are at risk for increasing depressive symptoms while quitting. Of particular importance, those participants who showed a rapid and sustained elevation in depressive symptoms following quitting also reported high rates of recurrent depression history and younger age at onset of first MDD. These smokers appear to have a more chronic course of major depression and appear to fail early in the cessation process. By contrast, smokers who showed delayed increases in depressive symptoms had moderately positive outcomes 2 weeks after quitting but showed continued decreases in abstinence over the next year. A gradual increase in depressive symptoms may have impeded their coping efforts or reduced their motivation to remain abstinent.

The brief reactors cluster began treatment low in depressive symptoms and showed a marked but very brief increase in depressive symptoms at quit date. These smokers showed high rates of abstinence in the first 6 weeks after quitting but began to relapse later in that year. This pattern was different from that of smokers whose depressive symptoms significantly lessened in the 2 weeks after quitting. Both rapid and delayed decreasers generally sustained the levels of abstinence they achieved at posttreatment, although overall outcomes for rapid decreasers were significantly better than for delayed decreasers. It may be that smokers who experience a significant decrease in depressive symptoms after quitting feel rewarded for their efforts and are motivated to remain abstinent.

Although our results confirm that an increase in depressive symptoms is related to poor smoking cessation outcome in smokers with a history of MDD, it was interesting to find that many participants did not exhibit a profile of increasing depressive symptoms. In the past, research has focused on smokers with a history of MDD as a group, finding that, on average, they report more depressive symptoms while quitting (Breslau, Kilbey, & Andreski, 1992; Covey et al., 1990; Ginsberg et al., 1995; Hall et al., 1994, 1996). However, this phenomenon may be due to only a minority of participants who are inflating the means. This may explain why treatments focused on teaching mood-management skills to smokers with a history of MDD are generally not more successful than traditional smoking cessation programs. Mood-management skills may only apply to a subset of smokers with a history of MDD.

We found that CBT-D did not affect the course of depressive symptoms during smoking cessation. That is, it did not predict cluster membership. As we have previously suggested (Brown et al., 2001), it may not be feasible to affect depressive symptoms with CBT-D while concurrently having smokers cut down on and then quit smoking altogether. Also, although we might expect that CBT-D would help smokers experiencing elevations in depressive symptoms to cope with these symptoms without smoking, we found that CBT-D did not differentially benefit any particular profile of depressive symptom change. Thus, there is little evidence that CBT-D, when delivered concurrent to smoking cessation treatment, can improve smoking cessation outcomes by moderating depressive reactions.

Several limitations of this study deserve mention. First, cluster analysis always involves some ambiguities when determining how many clusters to examine. A larger sample size may have allowed us to define additional clusters and patterns of change. However, the five-cluster solution appeared to fit the data well and to capture much of the variability in depressive symptom profiles. Second, it has been demonstrated that a decline in negative mood scores

often occurs with repeated testing (e.g., Sharpe & Gilbert, 1998), which could result in an artificially high baseline assessment. Although this effect should have been lessened somewhat by our using the second administration of the BDI (i.e., at Session 1) as the baseline measure, a longer period of baseline assessments with more BDI administrations would have provided a more stable baseline estimate that might have been lower than the one we used. If this were the case, more participants might have been classified as depressive symptom increasers.

Third, only participants who attended some treatment sessions after quitting were included in the cluster solution. Analyses revealed that those excluded were particularly heavy smokers who may be especially prone to increases in depressive symptoms while quitting. Furthermore, if participants experiencing an increase in depressive symptoms were more likely to drop out of treatment, this would lead us to underestimate the percentage of smokers with a history of MDD who experience an increase in depressive symptoms while quitting. Finally, given that our participants were predominantly Caucasian and fairly well educated, the generalizability of our findings may be limited.

In sum, although a pattern of increasing depressive symptoms was related to poor outcome in this sample of smokers with an MDD history, many participants did not report an increase in depressive symptoms while quitting, contrary to what might be expected in this population. In fact, almost half of the participants showed a decrease in depressive symptoms from the beginning to the end of treatment. These results suggest that researchers might need to look beyond history of MDD when identifying potential moderating variables in smoking cessation treatment studies. Nonetheless, that depressive symptom profiles were related to both short- and long-term smoking cessation outcome suggests that changes in depressive symptoms following quitting are important clinical phenomena that continue to deserve attention. Further work is needed to identify more completely those smokers at heightened risk for affective disturbances following smoking cessation.

References

- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3. Washington, DC: Author; 1987.
- Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Archives of General Psychiatry* 1961;4:561–571. [PubMed: 13688369]
- Breslau N, Kilbey NM, Andreski P. Nicotine dependence, major depression, and anxiety in young adults. *Archives of General Psychiatry* 1991;48:1069–1074. [PubMed: 1845224]
- Breslau N, Kilbey NM, Andreski P. Nicotine withdrawal symptoms and psychiatric disorder: Findings from an epidemiological study of young adults. *American Journal of Psychiatry* 1992;149:464–469. [PubMed: 1554030]
- Brown RA, Kahler CW, Niaura R, Abrams DB, Sales SD, Ramsey SE, et al. Cognitive-behavioral treatment for depression in smoking cessation. *Journal of Consulting and Clinical Psychology* 2001;69:471–480. [PubMed: 11495176]
- Cohen, J. Statistical power analysis for the behavioral sciences. 2. Hillsdale, NJ: Erlbaum; 1988.
- Cohen S, Schwartz JE, Bromet EJ, Parkinson DK. Mental health, stress, and poor health behaviors in two community samples. *Preventive Medicine* 1991;20:306–315. [PubMed: 2057476]
- Covey LS, Glassman AH, Stetner F. Depression and depressive symptoms in smoking cessation. *Comprehensive Psychiatry* 1990;31:350–354. [PubMed: 2387147]
- Covey LS, Glassman AH, Stetner F. Major depression following smoking cessation. *American Journal of Psychiatry* 1997;154:263–265. [PubMed: 9016279]
- Cronbach LJ, Gleser GC. Assessing similarity between profiles. *Psychological Bulletin* 1953;50:456–473. [PubMed: 13112334]
- Cummings SR, Richard RJ. Optimum cutoff points for biochemical validation of smoking status. *American Journal of Public Health* 1988;78:574–575. [PubMed: 3354745]

- Ginsberg D, Hall SM, Reus VI, Muñoz RF. Mood and depression diagnosis in smoking cessation. *Experimental and Clinical Psychopharmacology* 1995;3:389–395.
- Glassman AH, Covey LS, Dalack GW, Stetner F, Rivelli SK, Fleiss JF, Cooper TB. Smoking cessation, clonidine, and vulnerability to nicotine among dependent smokers. *Clinical Pharmacology and Therapeutics* 1993;54:670–679. [PubMed: 8275622]
- Glassman AH, Helzer JE, Covey LS, Cottler LB, Stetner F, Tipp JE, Johnson J. Smoking, smoking cessation, and major depression. *Journal of the American Medical Association* 1990;264:1546–1549. [PubMed: 2395194]
- Glassman AH, Stetner F, Walsh BT, Raizman PS, Fleiss JL, Cooper TB, Covey LS. Heavy smokers, smoking cessation, and clonidine. *Journal of the American Medical Association* 1988;259:2863–2866. [PubMed: 3367452]
- Hall SM, Muñoz RF, Reus VI. Cognitive-behavioral intervention increases abstinence rates for depressive-history smokers. *Journal of Consulting and Clinical Psychology* 1994;62:141–146. [PubMed: 8034816]
- Hall SM, Muñoz RF, Reus VI, Sees KL, Duncan C, Humfleet GL, Hartz DT. Mood management and nicotine gum in smoking treatment: A therapeutic contact and placebo-controlled study. *Journal of Consulting and Clinical Psychology* 1996;64:1003–1009. [PubMed: 8916629]
- Hall SM, Reus VI, Muñoz RF, Sees KL, Humfleet G, Hartz DT, et al. Nortriptyline and cognitive-behavioral therapy in the treatment of cigarette smoking. *Archives of General Psychiatry* 1998;55:683–690. [PubMed: 9707377]
- Heatherton TF, Kozlowski LT, Frecker RC, Fagerstrom KO. The Fagerstrom test for nicotine dependence: A revision of the Fagerstrom Tolerance Questionnaire. *British Journal of Addiction* 1991;86:1119–1127. [PubMed: 1932883]
- Hughes JR. Clonidine, depression, and smoking cessation. *Journal of the American Medical Association* 1988;259:2901–2902. [PubMed: 3367460]
- Johnson, RA.; Wichern, DW. *Applied multivariate statistical analysis*. 3. Englewood Cliffs, NJ: Prentice-Hall; 1992.
- Kessler RC. The National Comorbidity Survey of the United States. *International Review of Psychiatry* 1994;6:365–376.
- Kinnunen T, Doherty K, Militello FS, Garvey AJ. Depression and smoking cessation: Characteristics of depressed smokers and effects of nicotine dependence. *Journal of Consulting and Clinical Psychology* 1996;64:791–798. [PubMed: 8803370]
- Laird NM, Ware JH. Random-effects models for longitudinal data. *Biometrics* 1982;38:963–974. [PubMed: 7168798]
- Liang KY, Zeger SL. Longitudinal data analysis using generalized linear models. *Biometrika* 1986;73:13–22.
- McNair, DM.; Lorr, M.; Droppleman, LF. *EITS manual for the Profile of Mood States*. San Diego, CA: Educational and Industrial Testing Service; 1971.
- SAS Institute. *SAS/STAT user's guide*. 4. 1. Cary, NC: Author; 1990. Version 6
- SAS Institute. *SAS/STAT software: Changes and enhancements through Release 6.12*. Cary, NC: Author; 1997.
- Sharpe JP, Gilbert DG. Effects of repeated administration of the Beck Depression Inventory and other measures of negative mood states. *Personality and Individual Differences* 1998;24:457–463.
- Spitzer, RL.; Williams, JB.; Gibbon, M.; First, MB. *Structured Clinical Interview for DSM-III-R—Non-patient edition (SCID-NP, Version 1.0)*. Washington, DC: American Psychiatric Press; 1990.
- Zeger SL, Liang KY. Longitudinal data analysis for discrete and continuous outcomes. *Biometrics* 1986;42:121–130. [PubMed: 3719049]

Figure 1.
Square-root transformed Beck Depression Inventory (BDI) scores by cluster at Sessions 1, 5 (quit day), 7, and 8.

Figure 2. Observed point-prevalence abstinence rates by cluster at post-treatment, 1-month, 6-month, and 12-month follow-up.

Table 1

Variables Discriminating Between Clusters

Variable	Cluster 1 rapid increasers	Cluster 2 delayed increasers	Cluster 3 brief reactors	Cluster 4 delayed decreaseers	Cluster 5 rapid decreaseers
<i>M (and SD)</i>					
Baseline BDI ^a	6.03 _{a,b} (5.40)	7.60 _{a,c} (5.08)	4.57 _b (5.69)	7.79 _{a,c} (4.81)	9.48 _c (6.48)
Age at first major depressive episode, years	24.40 _a (10.59)	32.11 _{a,b} (12.08)	28.62 _{a,b} (12.06)	28.18 _{a,b} (13.69)	35.64 _b (13.41)
History of recurrent depression (%)	74.2 _a	37.1 _b	47.6 _{a,b}	65.2 _a	54.9 _{a,b}

Note. Cluster 1, $n = 31$; Cluster 2, $n = 35$; Cluster 3, $n = 21$; Cluster 4, $n = 24$; Cluster 5, $n = 52$. Means or percentages with different subscripts differ significantly at $p < .05$ in Tukey's tests for pairwise comparisons or in pairwise chi-square tests. BDI = Beck Depression Inventory.

^a Analyses were conducted on square-root transformed variables to correct for positive skewness.