Depression predicts smoking early but not late in a quit attempt

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Depression predicts smoking early but not late in a quit attempt

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This study examined the relationship between depression history and smoking after a quit attempt. A total of 677 smokers participating in a randomized smoking cessation trial (Smith et al., 2001) provided data on current depression, depression history, and depression-related measures and smoking at 1 week and 6 months after a quit date. Depression history predicted smoking at 1 week postquit but not at 6 months postquit. Smoking during the first week was not predictive of smoking at 6 months in those with a history of depression but was predictive among those with no history of depression. Prediction models including depression history and depression-related measures (e.g., negative affect, negative cognitive style) showed that depression history was a powerful predictor of smoking early in the quit attempt.

Introduction

Individuals with depression are more likely to be smokers than are nondepressed individuals (Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998; Fergusson, Goodwin, & Horwood, 2003; Hughes, Hatsuksami, Mitchell, & Dahlgren, 1986). In addition, smokers are more likely to have had a major depressive episode than are nonsmokers (Breslau, Novak, & Kessler, 2004; Breslau et al., 1998; Lasser et al., 2000; Grant, Hasin, & Chou, 2004). One longitudinal study revealed that over a 40-year period, individuals who became depressed during the 40-year study period were more likely to initiate smoking and less likely to quit during those 40 years than were nondepressed individuals (Murphy et al., 2003). These associations between depression and smoking raise the possibility that depression may interfere with smoking cessation.

At present, the data are mixed as to whether depression is associated with difficulty in quitting smoking. Some studies have shown that a history of depression is associated with more difficulty quitting (Breslau, Kilbey, & Andreski, 1992; Covey, Glassman, & Stetner, 1990; Glassman et al., 1993; Glassman et al., 1990; Smith et al., 2003). Other studies have failed to replicate this finding (Ginsberg, Hall, Reus, & Muñoz, 1995; Hall, Muñoz, & Reus, 1994; Hall et al., 1996; Hayford et al., 1999; Keuthen et al., 2000; Killen et al, 2000). Hitsman, Borrelli, McChargue, Spring, and Niaura (2003) conducted a meta-analysis of 15 smoking cessation studies and concluded that depression history was not a risk factor for relapse. However, questions have been raised about the methodology used in the Hitsman et al. study.

Covey (2004) pointed out that many of the studies that Hitsman et al. (2003) included in their analysis were clinical trials in which an antidepressant medication was tested as a smoking cessation aid. These medications may have neutralized the effect of depression history on cessation failure because they may have mitigated depressed mood. Hitsman, Spring, Borrelli, McChargue, and Niaura (2004) have since reanalyzed the data using only the
nonmedicated control conditions and again failed to
detect a relationship between depression history and
relapse. Hall (2004) suggested that failure to find a
depression-cessation relationship may be due to
analyzing depression as a dichotomous variable and
called for the use of continuous measures of
depression in future research.

The Hitsman et al. (2003) analysis used only 3- and
6-month outcomes. If no 3-month outcome was
available, they used the time point in the study
closest to 3 months. This method did not capture
potential immediate differences in abstinence levels
between the two groups that either had a history of
depression or did not. This finding is significant
because some evidence shows that smokers with
current depressive symptoms fail in a cessation
attempt more quickly than those without depressive
symptoms (Burgess et al., 2002; Gilbert, Crauthers,
Mooney, McClernon, & Jensen, 1999; Niaura et al.,
2001). Therefore, depression may manifest as less
success early in a quit attempt.

Although the Hitsman et al. (2003) meta-analysis
revealed little association between history of depression
and smoking relapse, other research has revealed a strong association between current (pre-
quit) symptoms of depression and relapse. For example, Niaura et al. (2001) demonstrated a strong
association across several studies, with even mild depressive symptoms predicting relapse. It is hard to
reconcile these data with the Hitsman et al. (2003)
results, given that a history of depression predicts
current depression well, and vice versa (Coyne,
Thompson, & Racioppo, 2001). Finally, many
cessation studies rigorously exclude individuals with
current depression and those who use antidepressant
drugs. This may have weakened the relationship
between depression history and cessation outcomes
in the Hitsman et al. (2003) meta-analysis. The
inconsistencies in the relationship between depression
and relapse vulnerability make it important to
establish with greater certainty the relationships
among depression history, current depression, and
the process of smoking cessation.

The present study is a secondary analysis of data
from a large clinical trial on step-up treatments for
smoking cessation (Smith et al., 2001). The clinical
trial did not use an antidepressant medication, and it
did not exclude individuals who used such medi-
cations. Data on DSM-type diagnoses of depression (a
dichotomous measure) as well as several continuous
measures of constructs related to depression (e.g.,
affective style and positive and negative affect) were
collected. Finally, the dataset contained both
immediate and long-term measures of smoking
outcomes.

The present study had three goals. First, we
evaluated our a priori prediction that participants
with either a history of depression or current
depression would have greater cessation failure than
would participants without a history of depression.
Our second hypothesis was that the relationship
between depression and smoking outcomes would be
supported by collateral analyses in which depression-
related measures (such as positive and negative affect
and negative cognitive style) also would predict
smoking outcomes. Such analyses might augment
confidence regarding the relationship between
depression and cessation failure, and suggest possible
causal factors. Our final goal was to construct best-
fitting models of abstinence outcomes from which we
hoped to discover the extent to which depression-
related constructs (measuring particular features or
correlates of depression) account for obtained
relationships with depression history.

Method
Participants

Participants were 677 smoking volunteers who
received free smoking cessation treatment (counsel-
ing and nicotine patches) in exchange for participa-
tion in the study. They were not paid for their
participation. Table 1 presents demographic infor-
mation. This study was approved by the Health Sciences
Human Subjects Committee of the University of
Wisconsin at Madison.

Design and procedure

Recruitment. Interested individuals, recruited via
mass media, called a central telephone number to
inquire about the study. Calls were returned by a
staff member who informed the individuals about
the study and screened interested individuals for elig-
ibility. All smokers had to be at least 18 years of age
and had to report smoking at least 10 cigarettes/day
for the past year. Exclusion criteria included recent
(within past 3 months) cardiac arrhythmia, heart
attack, stroke, cardiac surgery, or balloon angio-
plasty; schizophrenia or bipolar disorder; pregnancy,
breastfeeding, or being likely to become pregnant in
the next 3 months; current use of nicotine patches or
nicotine gum; use of exclusionary medications (e.g.,
antipsychotics, lithium); and serious contact allergies
to skin adhesives or serious skin sensitivity.

Procedure. All participants attended a group session
during which they provided written informed con-
sent. All participants returned for a prequit session
10 days prior to their target quit day. At this time,
we further assessed participants for eligibility
requirements including reassessing the requirements in the phone screen and assessing for exclusion criteria that could not be assessed over the phone (e.g., blood pressure). Five participants were excluded during the prequit session. The remaining participants received brief counseling and nicotine patches during the prequit visit.

On the target quit day, participants came to the clinic and were again given brief counseling and a carbon monoxide test. At this time, participants received daily diary forms, which they completed each day for the first week of their quit attempt. Participants returned for a visit 1 week postquit, at which time they were randomized into treatment groups (Figure 1). The three treatment conditions included a brief intervention condition and two step-up treatments—cognitive behavior/skills training group treatment and motivational interviewing/supportive group treatment (see Smith et al., 2001). A total of 122 participants dropped out between the prequit session and randomization. These participants were significantly more likely to have a history of depression and current depression than was true of the general study population: 36% of the dropouts had current depression or a history of depression (current depression=20%, history of depression=36%) versus 24% of those who did not drop out (current depression=10%, history of depression=24%; \( \chi^2 \) [for history of or current depression; \( df = 1 \] = 5.51, \( p = .02 \)).

### Table 1. Baseline sample characteristics by depression history.

<table>
<thead>
<tr>
<th>Variable</th>
<th>No history of depression ((n=514))</th>
<th>Past depression ((n=92))</th>
<th>Current depression ((n=71))</th>
<th>Total ((N=677))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (per cent female)</td>
<td>55.1</td>
<td>64.1</td>
<td>64.8</td>
<td>57.3</td>
</tr>
<tr>
<td>Race (per cent White)</td>
<td>94.5</td>
<td>97.8</td>
<td>95.8</td>
<td>95.1</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>42.76</td>
<td>40.1</td>
<td>41.25</td>
<td>42.23</td>
</tr>
<tr>
<td>SD</td>
<td>11.84</td>
<td>8.69</td>
<td>11.55</td>
<td>11.46</td>
</tr>
<tr>
<td>Cigarettes/day**</td>
<td>M</td>
<td>24.79**</td>
<td>24.91‡</td>
<td>28.75</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>9.96</td>
<td>9.76</td>
<td>10.74</td>
</tr>
<tr>
<td>Number of years smoking</td>
<td>M</td>
<td>24.80</td>
<td>23.10</td>
<td>23.99</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>11.48</td>
<td>8.26</td>
<td>12.02</td>
</tr>
<tr>
<td>Carbon monoxide (ppm)</td>
<td>M</td>
<td>24.42</td>
<td>25.17</td>
<td>23.32</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>9.91</td>
<td>10.94</td>
<td>10.49</td>
</tr>
<tr>
<td>FTND score**</td>
<td>M</td>
<td>4.45**</td>
<td>4.63</td>
<td>5.11</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>1.63</td>
<td>1.66</td>
<td>1.58</td>
</tr>
<tr>
<td>Positive PANAS**</td>
<td>M</td>
<td>3.52**</td>
<td>3.35†</td>
<td>3.11</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>0.68</td>
<td>0.67</td>
<td>0.87</td>
</tr>
<tr>
<td>Negative PANAS**</td>
<td>M</td>
<td>1.94**</td>
<td>2.18**‡</td>
<td>2.71</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>0.71</td>
<td>0.75</td>
<td>0.82</td>
</tr>
</tbody>
</table>

Note. \( M = \) mean; \( SD = \) standard deviation; FTND=Fagerström Test for Nicotine Dependence; PANAS=Positive and Negative Affect Scale. ** Overall \( F \) significant at level \( p < .01 \). * Contrast between no history and current groups significant at \( p < .05 \). ‡ Contrast between no history and current groups significant at \( p < .01 \). † Contrast between past and current groups significant at \( p < .01 \). ‡‡ Contrast between past and current groups significant at \( p < .05 \).

### Treatments

**Pharmacotherapy.** All participants received 8 weeks of nicotine patches (6 weeks of 22-mg/24-hr and 2 weeks of 11-mg/24-hr).

**Brief individual counselling.** All participants received brief individual counseling at the prequit, quit-day, and 1 week postquit visits. During the first counseling session, all participants received the Clearing the Air booklet (National Cancer Institute, 1988), containing tips to help individuals quit smoking.

**Step-up treatments.** In addition to the three individual counseling sessions, participants in the two step-up treatments attended a total of six 90-min sessions during weeks 2 through 5 of the quit attempt. Two sessions occurred per week for the first 2 weeks and one each week for the final 2 weeks. The two counseling treatments were a cognitive-behavioral therapy (CBT) treatment modeled after a treatment by Hall et al. (1994) and a motivational interviewing (MI) treatment based on the principles articulated by Miller and Rollnick (1991). For more information on the treatments, see Smith et al. (2001).

### Measures

At all clinic visits, participants completed an expired-air carbon monoxide (CO) test, and the Positive and
Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). For a full report of all assessments, see Smith et al. (2001). At the prequit visit, participants completed the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, 1991), the Affective Information Processing Questionnaire (AIPQ; Wetter, Brandon, & Baker, 1992), the Depression Proneness Inventory (DPI; L. Alloy, L. Y. Abramson, G. I. Metalsky, & S. Harlages, unpublished manuscript, 1990), and the Primary Care Evaluation of Mental Disorders psychiatric diagnostic system (PRIME-MD; Spitzer et al., 1994).

Smoking status. We used a 7-day point-prevalence measure of smoking outcomes. Because continuous data on daily smoking were not available, we were unable to determine which individuals did or did not establish initial abstinence in the quit attempt. Daily diary forms were handed out, but completion rates were low, especially by subjects with a history of depression (33%). Biochemical verification of abstinence, using an expired-air CO test, was collected at all time points except the 6-week and 3-month follow-ups. A CO value of less than 10 ppm was considered verification of abstinence. Participants without CO verification, or who did not respond to follow-up contacts, were considered to be smokers.

FTND. The FTND is a six-item self-report measure of nicotine dependence (Heatherton et al., 1991). The scale yields scores ranging from 0 to 10, with higher numbers indicating stronger dependence on nicotine.

PANAS. The PANAS measures positive and negative affect (Watson et al., 1988). It consists of 20 feeling words (e.g., interested, distressed, excited), which the participant rates on a five-point Likert scale to indicate the extent to which he or she feels that feeling.

DPI. The DPI is a 10-item self-report measure of proneness or vulnerability to depression (Alloy et al., 1990). Items (e.g., “Are you the type of person who easily becomes sad, blue or down in the dumps?”) are rated on a seven-point Likert scale and yield a summed score.

AIPQ. The AIPQ is a revised version of an affective style questionnaire that measures cognitive style and
responses to negative events (Wetter et al., 1992). The present version of the questionnaire comprises five negative situations (e.g., “You are not getting along with your family”). Using a seven-point Likert scale, participants are asked to rate the strength of their emotional response, the extent to which they could change their mood by doing something other than smoking, the extent to which they could change their mood by smoking, causal attribution for the situation (internal, global, stable), and whether the situation happening would mean that the participant is flawed in some way.

**PRIME-MD.** The PRIME-MD (Spitzer et al., 1994) is an assessment system designed to be used by primary care physicians to screen for psychological disorders using criteria provided in the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition, DSM-IV-TR; American Psychiatric Association, 2000). This instrument comprises five modules (mood, anxiety, alcohol, eating, and somatoform disorders). The present study used only the first four modules and used two versions of the PRIME-MD, one to assess the presence of current psychopathology (identical to the original PRIME-MD) and one to assess any episode of psychopathology in the past 5 years (adapted from the original). Overall agreement between diagnoses made by primary care physicians using the PRIME-MD and those made by mental health professionals using the Structured Clinical Interview for DSM (SCID) was 88%. The PRIME-MD tended to underdiagnose in the cases in which disagreement was found (Spitzer et al., 1994). With respect to diagnoses of mood disorders, the PRIME-MD diagnoses agreed with the mental health professionals’ SCID diagnoses 78% of the time, again underdiagnosing relative to the SCID (Spitzer et al., 1994). The PRIME-MD screens for four depressive disorders: major depression, minor depression, major depression in partial remission, and dysthymia. For our analyses, we only used diagnoses of major depression.

**Antidepressant use.** To assess antidepressant use, we examined participants’ reports of the medications they were currently taking. Participants were classified as using an antidepressant if they were taking any of the following: a tricyclic antidepressant, a monoamine oxidase inhibitor, a selective serotonin reuptake inhibitor, or an atypical antidepressant (e.g., bupropion) regardless of indication.

**Data analyses**

Logistic regression was used for most analyses because the main outcome variable was dichotomous (point-prevalence smoking). Depression history variables were dummy coded to make three groups: no history of depression (NHD), past depression but no current depression (past depression only; PDO), and current depression within last 2 weeks (CD).

Model-building procedures determined the relative influence of variables associated with history of depression and a depression diagnosis on smoking outcomes. We performed model building instead of mediational analyses because temporal priority was not established. As such, it was unclear whether high levels on depression-related variables were a cause, or a result, of history of depression (Cole & Maxwell, 2003). Thus the causal framework implied by mediational analyses seemed inappropriate. We followed the Hosmer and Lemeshow (2003) guidelines for model building. The models included dependence (FTND total score) and gender as control variables. The initial variable set comprised variables from the prequit measures that predicted (p<.25) the outcome (smoking status) in a regression equation in the presence of control variables.

We performed both forward and backward model-building. All variables were included in the model and then removed one at a time (those with the largest p values were removed first) until all the predictors in the model were significant. In addition, each variable was added to the model one by one, and then the model was retested. Only variables that remained significant were allowed to remain in the model. The final models presented here include predictors that were significant in both the forward and backward approaches.

**Results**

Of the 677 participants in the study, 163 (24.1%) had a history of major depression in the past 5 years, according to the PRIME-MD. Of those 163 participants, 71 (43.6%) met criteria for current major depression (CD group), 92 had a history of depression but no current depression (PDO group). A total of 91 participants in the sample reported using antidepressant medications: 29 (31.5%) for PDO individuals, 16 (22.5 %) for CD individuals, and 46 (8.9 %) for NHD individuals. When those in the NHD group who were currently taking antidepressant medications were excluded from the analyses, the results were unchanged. Thus the analyses here allowed individuals to be in the NHD group even if they were currently using antidepressant medications. Of those individuals with any past or current depression, only 63 of the 163 total (38.7%) did not have current depression and were not currently using antidepressant medications.

The sample comprised individuals with several DSM-IV-TR diagnoses, other than major depression, within the past 5 years. Most notably, in the
past 5 years, 3.5% of participants met criteria for panic disorder, 11.7% met criteria for generalized anxiety disorder, 10.7% met criteria for minor depression, and 20.8% met criteria for alcohol abuse or dependence. In the past month, 5.9% met criteria for dysthymia. Because of the prevalence rates of such other DSM-IV-TR diagnoses in the sample, all analyses were completed both with and without controlling for other DSM-IV-TR diagnoses.

History of depression

Logistic regression analyses revealed that, compared with NHD individuals, CD and PDO individuals were significantly more likely to be smoking at 1 week postquit (PDO: OR=5.62, 95% CI=3.36–9.38; CD: OR=40.60, 95% CI=12.60–130.87) but not at 6 weeks, 3 months, or 6 months (Table 2). When controlling for other psychiatric diagnoses (alcohol abuse or dependence, generalized anxiety disorder, panic disorder, and eating disorders), age, gender, dependence (FTND), antidepressant use, and treatment condition, we found that the effect remained significant at 1 week (PDO: OR=3.73, 95% CI=1.58–8.81; CD: OR=25.37, 95% CI=6.52–98.74) and nonsignificant at all other time points. At 1 week postquit, individuals with CD showed significantly higher smoking rates (95.8%) than those with PDO (75%; χ²=12.90, p<.001). No significant interactions were found between either current or past depression and treatment, gender, or dependence (FTND score) in predicting smoking rate. It is possible that CD or PDO individuals were merely mistaken in their self-report of smoking during the first week, or that they smoked only on their quit day but not thereafter. These hypotheses are countered by the fact that those in the CD and PDO groups had somewhat higher CO values (6.6) at the 1 week visit than did the NHD individuals (5.4), a statistically significant difference, F(1, 668)=4.90, p<.03.

Table 2 shows that the above pattern of findings was obtained because a very high proportion of CD and PDO smokers were smoking at week 1, but a smaller number of these individuals were smoking at later follow-up time points. As Table 2 makes clear, smoking rates decreased among CD and PDO individuals after week 1, whereas they increased markedly (doubled) among NHD individuals. Thus logistic regression analyses showed that among individuals smoking at week 1, PDO and CD individuals were more likely to be abstinent at 6 months than were NHD individuals (PDO: OR=0.42, 95% CI=0.19–0.95; CD: OR=0.31, 95% CI=0.14–0.68). The same pattern held for the 6-week and 3-month outcome data. These relationships were not related to treatment group assignment.

Excluding those with current depression and current antidepressant use

Previous studies often excluded those with current depression and those taking antidepressant medication. These policies may have masked or reduced associations between depression status and abstinence outcomes. Therefore, we examined the effect of excluding versus including individuals with current depression and current antidepressant use. First, for comparison purposes, we conducted an analysis of a history of depression (HD) group (including CD and PDO individuals) versus a NHD group (antidepressant medication users were included in the analysis but remained in whichever group their PRIME-MD

Table 2. Number and percentage of subjects smoking at each follow-up point, by depression history.

<table>
<thead>
<tr>
<th>Follow-up</th>
<th>Depression group</th>
<th>Number smoking (per cent)</th>
<th>Odds ratio a</th>
<th>95 % Confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 week</td>
<td>CD</td>
<td>68 (95.8%)</td>
<td>40.60</td>
<td>12.60–130.81</td>
</tr>
<tr>
<td></td>
<td>PDO</td>
<td>69 (75.0%)</td>
<td>5.62</td>
<td>3.36–9.38</td>
</tr>
<tr>
<td></td>
<td>NHD</td>
<td>184 (35.8%)</td>
<td>321 (47.4%)</td>
<td></td>
</tr>
<tr>
<td>6 weeks</td>
<td>CD</td>
<td>45 (63.4%)</td>
<td>1.02</td>
<td>0.61–1.70</td>
</tr>
<tr>
<td></td>
<td>PDO</td>
<td>57 (62.0%)</td>
<td>1.35</td>
<td>0.83–2.20</td>
</tr>
<tr>
<td></td>
<td>NHD</td>
<td>324 (63.0%)</td>
<td>324 (63.0%)</td>
<td></td>
</tr>
<tr>
<td>3 months</td>
<td>CD</td>
<td>49 (60.0%)</td>
<td>1.20</td>
<td>0.70–2.05</td>
</tr>
<tr>
<td></td>
<td>PDO</td>
<td>66 (71.7%)</td>
<td>1.35</td>
<td>0.83–2.20</td>
</tr>
<tr>
<td></td>
<td>NHD</td>
<td>335 (65.2%)</td>
<td>335 (65.2%)</td>
<td></td>
</tr>
<tr>
<td>6 months</td>
<td>CD</td>
<td>56 (78.9%)</td>
<td>0.93</td>
<td>0.50–1.71</td>
</tr>
<tr>
<td></td>
<td>PDO</td>
<td>75 (81.5%)</td>
<td>1.08</td>
<td>0.61–1.91</td>
</tr>
<tr>
<td></td>
<td>NHD</td>
<td>412 (80.2%)</td>
<td>412 (80.2%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>450 (66.5%)</td>
<td>450 (66.5%)</td>
<td></td>
</tr>
</tbody>
</table>

Note. CD=current depression; PDO=past depression only; NHD=no history of depression. aRelative to NHD.
score indicated). The results of this analysis showed that history of depression predicted smoking at 1 week ($OR=9.82$, $95\%\ CI=6.18$–$15.60$), compared with the NHD group. History of depression was not predictive of smoking at any later follow-up points.

Next we assessed the effect of excluding these individuals. Thus we conducted a second analysis of history of depression predicting smoking in which individuals with current depression and individuals currently using antidepressant medications were excluded from the analyses (to be consistent with past literature). The odds ratio for history of depression predicting smoking at 1 week postquit decreased but remained statistically significant ($OR=4.10$, $95\%\ CI=2.32$–$7.25$). History of depression still did not predict smoking at any of the other follow-up points.

**Depression-related measures**

All depression-related measures (PANAS, AIPQ, DPI) showed a pattern that was similar to history of depression in predicting smoking. When we controlled for gender and tobacco dependence (FTND), all measures predicted smoking at 1 week postquit but not at any other time point (Table 3).

**Model building**

We conducted both backward and forward model-building for smoking during the first week of the quit attempt. The candidates for the model included the negative and positive subscales of the PANAS, the globality and stability scales of the AIPQ, and the DPI. The control variables included gender and FTND score. The best-fitting model contained past depression only ($OR=4.80$, $95\%\ CI=2.79$–$8.24$), current depression ($OR=30.60$, $95\%\ CI=9.31$–$100.64$), DPI ($OR=1.02$, $95\%\ CI=1.00$–$1.04$), and FTND score ($OR=1.18$, $95\%\ CI=1.06$–$1.31$). Similar results for depression-related measures were obtained when we did not control for gender and FTND score.

**Table 3.** Results of individual logistic regressions of depression-related measures predicting smoking at 1 week postquit, controlling for FTND scores and gender.

<table>
<thead>
<tr>
<th>Measure</th>
<th>$B$</th>
<th>SE</th>
<th>Wald</th>
<th>Degrees of freedom</th>
<th>$p$ value</th>
<th>Odds ratio</th>
<th>95% confidence interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>pPANAS$^a$</td>
<td>-0.25</td>
<td>0.11</td>
<td>5.03</td>
<td>1</td>
<td>.025</td>
<td>0.78</td>
<td>0.62–0.97</td>
</tr>
<tr>
<td>nPANAS$^b$</td>
<td>0.35</td>
<td>0.11</td>
<td>10.76</td>
<td>1</td>
<td>&lt;.001</td>
<td>1.42</td>
<td>1.15–1.74</td>
</tr>
<tr>
<td>DPI</td>
<td>0.05</td>
<td>0.01</td>
<td>39.00</td>
<td>1</td>
<td>&lt;.001</td>
<td>1.05</td>
<td>1.04–1.07</td>
</tr>
<tr>
<td>AIPQ globality$^c$</td>
<td>0.21</td>
<td>0.07</td>
<td>10.11</td>
<td>1</td>
<td>.001</td>
<td>1.24</td>
<td>1.09–1.41</td>
</tr>
<tr>
<td>AIPQ stability$^d$</td>
<td>0.15</td>
<td>0.07</td>
<td>4.51</td>
<td>1</td>
<td>.03</td>
<td>1.16</td>
<td>1.01–1.33</td>
</tr>
</tbody>
</table>

Note. AIPQ=Affective Information Processing Questionnaire; DPI=Depression Proneness Index; FTND=Fagerström Test for Nicotine Dependence; PANAS=Positive and Negative Affect Scale. $a$ Positive affect scale of the PANAS (Watson et al., 1988) administered at orientation. $b$ Negative affect scale of the PANAS (Watson et al., 1988) administered at orientation. $c$ Globality question: “Think about the cause. Does the cause lead to other problems in your life—or does the cause pretty much only lead to the above situation?” $d$ Stability question: “Think about the cause. Do you think the cause will produce similar problems for you in the future?”

**Discussion**

An important finding of this research is that our results agree with those of Hitsman et al. (2003). That is, we found little or no relationship between depression status and smoking status at later follow-up time points (e.g., 3 and 6 months). This finding is notable given that our sample was relatively large, depression symptoms were assessed with a variety of diagnostic and continuous measures, and the sample was not restricted in range (i.e., it comprised those with current depression and those taking antidepressants). Moreover, the pharmacotherapy evaluated in this trial was a nicotine replacement pharmacotherapy, not an antidepressant.

Results also showed that depression history predicted higher smoking rates early in the quit attempt (1 week). Moreover, those with current depression had much higher smoking rates at 1 week (96%) than those with only a past history of depression (75%). These predictive relationships were characterized by very large effect sizes (odds ratios). These were differences of about 40% and 60%, respectively, between past and current depression history groups, on the one hand, and those with no depression history, on the other hand.

Our results suggest that the breadth of inclusion criteria affects the relationship between depression and 1-week abstinence. First, excluding individuals with current depression and individuals currently taking antidepressant medications decreased our depression sample by 61%. This not only decreased power but also might have biased tests. In support of this possibility, excluding these individuals markedly reduced the strength of the association between depression and smoking during the first week postquit.

Our model-building efforts revealed that the dichotomous depression measure was superior to the continuous depression-related measures in predicting 1-week smoking status. In fact, depression history displaced all such variables except vulnerability to become depressed in the best-fitting model.
Thus, based on our results, it is unlikely that the use of dichotomous diagnostic measures (as opposed to continuous) results in insensitive tests of the depression-abstinence relationship (cf. Hall, 2004). It is noteworthy, however, that both past and current depression remained in the model. This suggests that both conditions may act somewhat independently in terms of their influence on smoking during the start of a quit attempt. It is interesting to speculate why having had a past major depressive episode, in the absence of a current episode, affects smoking outcome. It could be that individuals with a history of depression who are currently in remission still have some subsyndromal levels of depressive symptomatology that are affecting their ability to stay quit. Or some other related factor that makes certain individuals susceptible to depression might also influence their ability to refrain from smoking early in the quit attempt (e.g., distress intolerance, skill deficits).

The present study also showed that current depression tended to show stronger relationships with outcome than did previous depression per se. This finding is consistent with Niaura et al.’s (2001) finding that measures of current depression predicted cessation outcomes. However, the present research contrasts with other research in that current depression predicted only 1-week outcomes (cf. Niaura et al., 2001).

A surprising finding was that smoking during the first week postquit was less predictive of ultimate cessation failure for those with a history of depression than it was for other smokers. This finding has a variety of possible explanations. Nondepressed participants might marshal all their forces to quit completely on the quit day. For these individuals, failure to achieve initial cessation could be motivationally draining and sap their self-efficacy. If participants with a history of depression do not expect to quit completely and immediately, early smoking may be less of a sign of failure and less discouraging for these individuals, or it may be that reductions in smoking (vs. complete cessation) may be more reinforcing for individuals with a history of depression. This partial success may bolster their self-efficacy, or reduced smoking could reduce stress symptoms associated with smoking (Parrott, 2004). This finding suggests that different populations of smokers (e.g., depressed vs. nondepressed) may show different dynamic patterns of smoking following the quit attempt. Thus smokers may show not only different patterns of withdrawal symptoms postquit (Piasecki, Jorenby, Smith, Fiore, & Baker, 2003) but also different patterns of smoking leading to ultimate success or failure.

The different patterns of cessation found in this research may be important for two reasons. First, these patterns suggest that continuous or survival measures of outcome may penalize depression-history smokers and create the impression that such smokers have higher relapse rates than other individuals (i.e., continuous measures would not reflect the ultimate quitting of the former individuals). These results also support the suggestion that investigators use a “grace period” in their follow-up analyses (during which very early smoking is ignored; Hughes et al., 2003). Second, this finding implicates different quitting processes for depression-history versus other smokers. Perhaps treatments need to be tailored for the former individuals, for example, with less emphasis on abrupt cessation and more use of fading treatments or sustained treatments.

These results must be evaluated with certain limitations in mind, for example, the lack of continuous measures of abstinence and the use of the PRIME-MD rather than a better validated diagnostic instrument (e.g., the SCID). The use of the PRIME-MD is of particular concern because it tends to underdiagnose (Spitzer et al., 1994). This underdiagnosis may be due in part to the fact that the PRIME-MD assesses episodes of mental illness that occur only within the past 5 years. In addition, individuals with depression were significantly more likely to drop out before randomization. Had these individuals remained in the study through randomization, and been counted as smokers, our results might have been different.

In conclusion, we found that (a) smokers with positive depression histories did not differ from other smokers in terms of long-term abstinence status following a quit attempt, replicating the Hitsman et al. (2003) findings, (b) individuals with past and current depression were more likely to show higher rates of cessation failure early in a quit attempt, (c) those with current depression were particularly likely to smoke in the first week postquit, compared with those with past depression or those with no history of depression, (d) smoking during the first week was less predictive of future smoking behavior for individuals with past or current depression than it was for nondepressed smokers (an observation that contrasts sharply with evidence among general populations of smokers that smoking early in a quit attempt is highly predictive of ultimate failure; Kenford et al., 1994; Westman, Behm, Simel, & Rose, 1997), and (e) model-building procedures showed that a diagnostic measure of depression was a better predictor of week 1 smoking status than were continuous measures of depression-related variables, suggesting that there is something special about the whole, rather than the sum of its parts.

The results of the present study demonstrate a need to more closely examine smoking patterns postquit, focusing particularly on early outcomes.
References


Acknowledgments

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