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Depression Vulnerability Moderates the Effects of Cognitive Behavior Therapy in a Randomized Controlled Trial for Smoking Cessation

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Several clinical trials have tested the hypothesis that smoking cessation treatments with a mood management component derived from cognitive behavior therapy (CBT) for depression would be specifically effective for depression-vulnerable smokers, with mixed results. This trial addressed methodological concerns with some of the previous studies to clarify whether depression vulnerability does in fact moderate CBT smoking cessation outcome. The study compared 8-session group CBT with a time-matched comparison group condition in a sample of 100 cigarette smokers randomized to treatment condition. Each treatment group was led by one of 7 American University clinical psychology graduate students; therapists were crossed with treatment conditions. Outcome (7-day point prevalence abstinence) was evaluated 1 month and 3 months after quit date. Baseline self-reported depression vulnerability (sample median split on the Depression Proneness Inventory) moderated treatment response, such that more depression-prone smokers fared better in CBT whereas less depression-prone smokers fared better in the comparison condition. These results may have implications for determining when to use CBT components in smoking cessation programs.

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Tobacco use continues to be the leading cause of preventable death, disability, and illness in the United States (Centers for Disease Control [CDC], 2002), yet an estimated 20% of the United States adult population are current smokers, and of these smokers 78% smoke cigarettes daily (CDC, 2008). Between 1997 and 2001, smoking-related illnesses accounted for an estimated 438,000 premature deaths per year (CDC, 2005) and, additionally, they produced about $157 billion in annual health-related economic costs (CDC, 2002).

A commonly reported motive for cigarette smoking is negative affect (Kassel, Stroud, & Paronis, 2003). Episodic negative affect also poses a high risk of relapse for those who have recently quit smoking (e.g., Shiffman, Paty, Gnys, Kassel, & Hickcox, 1996). Finally, chronic negative affect additionally plays a significant role in smoking. For example, depressed people are overrepresented among current smokers (e.g., Acierno, Kilpatrick, Resnick, Saunders, & Best, 1996), especially smokers high in nicotine dependence (e.g., Breslau, Kilbey, & Andreski, 1991). In a longitudinal epidemiological study of young adults, those with a history of major depression at baseline were substantially more likely than those without such a history to progress to daily smoking (Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998). Moreover, depressed people appear to have a harder time quitting smoking than do nondepressed smokers (Glassman, 1993); this applies even to individuals with low, subclinical levels of depressive symptoms (Niaura et al., 2001), to populations with depressed mood (Cinciripini et al., 2003), and to those with a lifetime history of at least one period of depressed mood or anhedonia lasting at least 2 weeks (Ziedonis et al.). A recent review

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concluded that there may well be bidirectional linkages between smoking and depression, such that smoking can lead to depression or vice versa (Ziedonis et al., 2008).

Based on such studies linking depression and smoking, it has been hypothesized that adaptations of psychotherapies for depression could be effective in helping smokers learn alternate (nonsmoking) means of coping with negative mood states and thereby enhance the probability of successful abstinence. Given that extensive research supports the efficacy of cognitive behavior therapy (CBT; Beck, Rush, Shaw, & Emery, 1979) in the treatment of depression (e.g., Chambless et al., 1998), several treatment programs that draw upon CBT have been applied to the smoking cessation context (Brown, 2003). CBT for smokers in general has been shown to be significantly more effective than minimal cessation advice alone through 12-month follow-up (e.g., Marks & Sykes, 2002).

Although few studies have been conducted of psychosocial treatment for smokers *currently* experiencing major depression (for an exception, see Hall et al., 2006), several investigators have tested the hypothesis that CBT would be specifically effective for smokers who are vulnerable to experiencing depression. The premise is that such smokers would especially benefit from learning healthier means of managing negative mood states as a way of maintaining abstinence. A history of major depression at baseline is not a significant independent predictor of failing to benefit from smoking cessation treatment (Covey, Bomback, & Yan, 2006; Hitsman, Borrelli, McChargue, Spring, & Niaura, 2003). Nevertheless, history of major depression predicts depression in the wake of smoking cessation treatment (Covey, Glassman, & Stetner, 1997), and increases in depressive symptoms in response to quitting smoking predict relapse (Burgess et al., 2002), so it is plausible that smokers vulnerable to depression could particularly benefit from mood management skills addressed in CBT.

Clinical trials testing this moderator hypothesis have yielded mixed results (Haaga, Hall, & Haas, 2006). In a sample of smokers with a history of alcohol dependence, baseline depressive symptoms interacted with treatment condition such that CBT mood management techniques were helpful only for smokers high in depressive symptoms (Patten, Drews, Myers, Martin, & Wolter, 2002). Given that baseline depressive symptom level is a significant predictor of later incidence of major depression (Lewinsohn, Solomon, Seeley, & Zeiss, 2000), this finding can be seen as consistent with the view that CBT would be especially helpful for those vulnerable to depression.

Most studies have instead operationalized vulnerability to depression as the presence of a history of major depression. Hall, Muñoz, and Reus (1994) found that a CBT group treatment added to a standard health-education-based program significantly outperformed the health education program alone only for depression-vulnerable smokers, operationalized in this study as having a history of major depression. This result was replicated by Hall et al. (1998).

However, a third clinical trial by the same research group equated the two conditions for therapy contact time and failed to replicate the interaction of depression vulnerability and treatment condition (Hall et al., 1996). In a study of smokers with a history of alcohol dependence, CBT significantly enhanced the efficacy of a behavioral treatment based on nicotine fading and self-monitoring, even with therapy contact time controlled (Patten, Martin, Myers, Calfas, & Williams, 1998). However, all participants were positive for a history of depression, so there is no way to determine whether the beneficial impact of CBT was specific to this group.

Finally, Brown and colleagues (2001) obtained a specific effect for CBT with smokers with a history of depression, but only if they had a history of recurrent depression, not just a single previous episode, suggesting that the method of measuring depression vulnerability may influence results. This effect was replicated in a secondary analysis of the three Hall et al. (1994, 1996, 1998) clinical trials cited earlier—CBT was more effective than a health education comparison condition only for participants who had experienced at least two prior major depressive episodes, not zero or one (Haas, Muñoz, Humfleet, Reus, & Hall, 2004).

The Brown et al. (2001) and Haas et al. (2004) results suggest that CBT may provide benefit specifically for depression-vulnerable smokers and that this effect might be found only at fairly high levels of depression vulnerability. These results would seem to bring welcome clarity to what has been a confusing literature, but we believe additional research is needed. History of recurrent major depression has itself proven inconsistent as a moderator of CBT effects on smoking cessation. A subsequent trial (Brown et al., 2007) did not find CBT mood management treatment (relative to standard CBT lacking the mood management component, and crossed with either buproprion or placebo) to be differentially effective among those with a history of recurrent major depression, though this nonreplication could have resulted from limited statistical power. Of the 524 patients randomized, only 16 had experienced multiple

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prior depressive episodes. By the same token, this paucity of participants with multiple previous major depressive episodes is not just a statistical issue. It suggests that operationally defining depression vulnerability in this manner limits the vulnerable subgroup substantially in a typical smoking cessation clinic and sets constraints on the practical utility of the findings for clinicians in such settings. Most importantly, it is not clear that this substantial winnowing of the population of smokers seeking to quit actually defines the depression-vulnerable subgroup in the most valid way possible.

Depression history (whether recurrent or not) may be an imprecise assessment of current vulnerability to depression for a couple of reasons (Just, Abramson, & Alloy, 2001). There might be individuals who have yet to experience a major depressive episode because no suitably major stressor has occurred, even though they are actually high in depression vulnerability. Their depression vulnerability therefore would be underestimated if assessment is based only on the past occurrence of depressive episodes. Conversely, some smokers with histories of depression might no longer be highly vulnerable to depression as a result of enduring effects of interventions used in helping them recover in the first place.

To address the ambiguities associated with depression history as a measure of vulnerability in the research reported in this article, we measured current depression vulnerability with the Depression Proneness Inventory (DPI; Alloy, Hartlage, Metalsky, & Abramson, 1987). To our knowledge, only two previous studies of cognitive-behavioral interventions for cigarette smokers have used the DPI as a predictor. A comparison of CBT with an intervention based upon motivational interviewing found no specific benefit of CBT for depression-vulnerable (high-DPI) smokers (Smith et al., 2001). However, this study differed from earlier CBT studies in that CBT and motivational interviewing were implemented as "step-up" treatments after an initial brief intervention and cessation attempt. It is not known whether results would be similar were these treatments implemented from the outset of the smoking cessation attempt. Conversely, Brandon et al. (1997) *did* report a selective effect of CBT for those high in depression proneness.

In view of the Brown et al. (2001) and Haas et al. (2004) findings indicating that a high level of depression vulnerability is necessary to show a selective benefit of CBT for smoking cessation, we did not predict that the DPI as a continuous variable in a sample unselected for depression vulnerability would moderate treatment response. Instead, we expected that high levels of depression vulnerability would be necessary. Taxometric research conducted in a large sample of treatment-seeking smokers suggested that the DPI validly measures a taxonic construct of depression proneness (Strong, Brown, Kahler, Lloyd-Richardson, & Niaura, 2004). In the absence of precise guidance from the literature on what DPI score would be high enough to suggest probable membership in the "depression-prone" taxon,[1] we used our sample median split to select high and low depression-prone groups.

In summary, several studies have obtained interactive effects such that CBT mood management therapy is specifically effective for depression-vulnerable smokers, but findings have been inconsistent, perhaps as a function of methods of measuring depression vulnerability. We therefore conducted a randomized clinical trial of CBT and a time-matched comparison treatment. We hypothesized that self-rated current depression proneness would interact with type of treatment in predicting abstinence outcomes through 3 months after quit date. CBT was expected to be more effective than the comparison condition for those above the sample median in depression proneness, but not for those below the median.

Method

PARTICIPANTS

Cigarette smokers were recruited from the Washington, DC, metropolitan area via newspaper advertisements, community fliers, public service announcements, advocacy organizations (e.g., American Lung Association), online postings (e.g., www.craigslist.org), and community and university health centers and hospitals. Advertisements solicited "smokers who want to quit" and indicated that help would be provided in the form of "group therapy sessions" or "group counseling"; there was no mention of mood management, cognitive behavior therapy, or depression proneness in the

[1] Strong et al. (2004) obtained an estimated base rate of 19% for the depression-prone taxon, which would imply that our characterization of those participants above the DPI sample median as highly depression-prone is overly liberal. However, (a) their taxometric analyses were based on a subset of DPI items, so it is not possible to reconstruct an exact total DPI score optimally separating the taxon members from nonmembers; (b) there was variability in the base rates estimated from different taxometric analyses, suggesting that more research is needed to pin this figure down more precisely; and (c) most importantly, their sample appears to have been less depression-prone than ours. Their sample obtained total DPI scores averaging 23.18 (SD = 8.12), whereas ours obtained a mean of 31.71 (SD = 11.32). As such, our above-the-median subsample (32 and higher) were all at least one standard deviation above the mean of the Strong et al. sample and therefore likely candidates for the depression-prone taxon even with only 19% of their sample qualifying as such.

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ads. Cigarette smokers were enrolled in the program if they smoked at least 1 cigarette per day for the past 4 weeks, wanted to quit smoking, were fluent in English, were willing to be treated in a group setting, and were at least 18 years old. We set a low minimum smoking rate for eligibility (relative to some other trials that require, for instance, $\geq$10 cigarettes/day) because even very light smoking (1 to 4 cigarettes/day) has been linked in longitudinal epidemiological research with death from heart disease and with all-cause mortality (Bjartveit & Tverdal, 2005). As such, practice guidelines (USDHHS, 2008) recommend helping all tobacco users to quit.

Prospective participants were excluded and referred elsewhere if they were actively suicidal, on the premise that smoking cessation can be stressful and could exacerbate suicidal ideation.

One hundred participants (49 male, 51 female) both enrolled in the program and were randomized to a treatment condition. Four participants enrolled in the program but dropped out prior to randomization; therefore, these participants were excluded from all remaining analyses. The sample size was determined by the number of eligible participants we were able to enroll and treat within the project funding period. The moderator effect of depression vulnerability in CBT smoking cessation studies has been erratic (see Introduction), and we did not have a confident a priori estimate of its effect size for sample size planning purposes. There were no interim analyses conducted during the study. Figure 1 summarizes the flow of participants from assessment to follow-up and analysis.

Participants ranged in age from 20 to 68 years (M=42.85, SD=12.80) and reported 9 to 21 years of education (M=15.84, SD=2.46). Participants were full-time employed (56%), part-time employed (14%), had a leave of absence or were unemployed (11%), were full-time students (8%), or retired (7%). Their annual household incomes ranged from less than $10,000 to over $200,000 with the most common range (17%) being between $50,000 to $75,000.

A majority of participants were Caucasian (65%), whereas about one-quarter were African American (29%), with the remaining participants being Asian American (2%) or other races (3%). About one-tenth of the participants (9%) were of Hispanic ethnicity.

Pretreatment daily smoking rates varied widely, from 4 to 60 cigarettes, with an average just under a pack a day (M=17.76, SD=8.34). All participants reported having smoked for at least 1 year (mean years smoked=23.49, SD=13.33). Participants estimated that they tried to quit up to 50 times before (median=3; 25th percentile=1; 75th percentile=5). Their longest previous quit attempts ranged from less than 1 day to 6,120 days (median=90; 25th percentile=21; 75th percentile=270). The participants reported moderate nicotine dependence on the Fagerström Test for Nicotine Dependence (M=4.66, SD=2.34).

MEASURES

Suicidality was assessed with the Beck Scale for Suicide Ideation (BSI; Beck, Steer, & Ranieri, 1988). The interviewer determined if significant suicidal ideation was present by following up on any positive responses on this questionnaire. If so, the participant was excluded from the study and referred elsewhere so that suicidal ideation could be addressed first.

Sample demographics and smoking history were assessed using brief, face valid questionnaires concerning age, gender, socioeconomic status, number of cigarettes smoked per day, number of past quit attempts, age at which the first cigarette was smoked, and the number of years that the participant smoked daily.

Nicotine dependence was measured with the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991). This 6-item self-report has moderate internal consistency (alpha=.64), satisfactory retest reliability over 2 to 3 weeks (r=.88), and positive correlations with cotinine levels (r=.39), with self-reports of "addiction" as a reason to smoke (r=.53), and with the number of years as a smoker (r=.52; Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994). Depression proneness was measured with the Depression Proneness Inventory (DPI; Alloy et al., 1987). The DPI is a 10-item self-report measure of vulnerability to depressive reactions to stress. The DPI is face valid, as the questions ask about proneness to depression (e.g., "Would your friends who know you best rate you as a person who easily becomes very depressed, sad, blue, or down in the dumps?"). Each item is rated on a 1 to 7 Likert-type scale, and the total DPI score is the sum of the item scores (i.e., 10 to 70). The DPI is highly internally consistent (alpha=.90 in nonclinical samples) and stable (1-month retest reliability r=.88; Alloy et al.). The DPI has correlated positively with current depressive symptoms and with number of past episodes of major or minor depressive disorder, but not with past episodes of anxiety disorders, mania, or drug and alcohol abuse (Alloy et al., 1987), supporting its specificity to depression proneness. A prospective study in an undergraduate sample supported its predictive validity in that DPI scores

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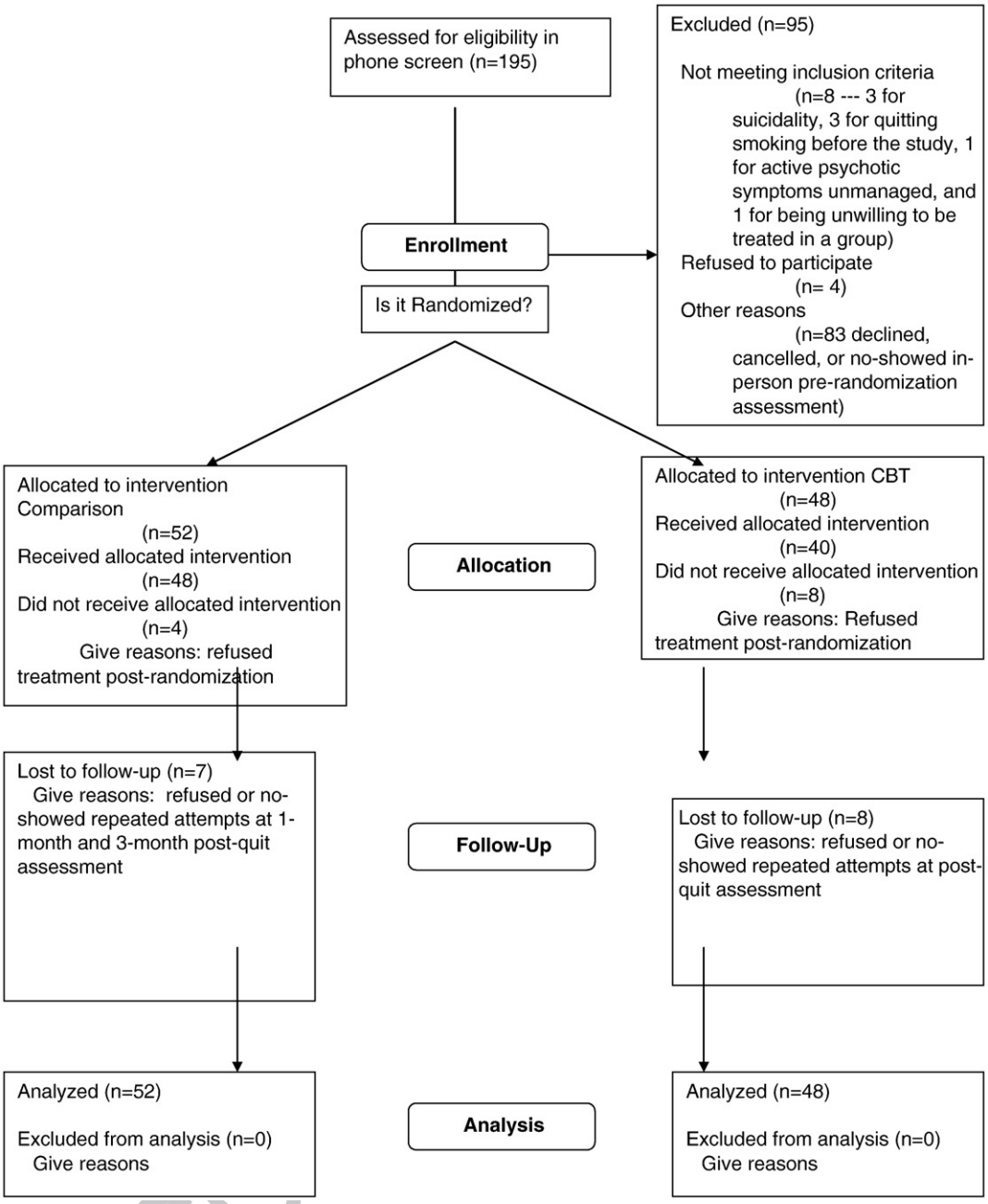

FIGURE 1 The CONSORT E-Flowchart.

from the beginning of an introductory psychology course predicted increased depressive symptoms in the wake of a poor performance on a midterm examination above and beyond what could be predicted on the basis of Time 1 depression scores (Alloy et al.). In a clinical trial of smoking cessation methods, smokers who lapsed even once during the first week after a quit attempt had scored higher on the DPI at baseline than did those who maintained abstinence during the first week (Smith et al., 2001). In descriptive studies of smokers, DPI scores have been positively correlated with interview-derived diagnoses of past major depression (Haaga et al., 2004) and with self-reported motivation to smoke in order to reduce negative mood (Brody et al., 2005). The association of DPI scores with past major depression was significant even after controlling for age, gender, and current depressive symptoms (Strong et al., 2004).

Smoking status was measured by self-report. When participants self-reported abstinence, expired air carbon monoxide (CO) was measured for verification purposes. Self-reports were collected in person at each treatment session after quit date

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(sessions 5 through 8), at a posttreatment assessment 1 month after quit date, and by phone at 3 months after quit date. CO measurement always took place in person. If a participant reported abstinence by phone at the 3-month follow-up, an appointment was made for the participant to have their CO level measured in person. Our outcome measure was 7-day point prevalence abstinence, which entailed self-report of no use of tobacco products in the prior 7 days, as well as an expired air carbon monoxide (CO) reading of ≤ 8 parts per million (SRNT Subcommittee on Biochemical Verification, 2002). Seven-day point prevalence abstinence is the metric used in compiling results for the U.S. Department of Health and Human Services practice guideline (USDHHS, 2008). At each follow-up (1 month and 3 months post target quit date) there was one participant whose self-reported abstinence was disconfirmed by the CO reading, resulting in reclassification as a smoker.

Therapist adherence was measured by audiotaping each group treatment session. Masked raters who were familiar with the manuals developed for each condition subsequently rated a random sample of session tapes with respect to which therapy condition was being conducted, as a measure of the differentiability of the treatment conditions. Independently, additional raters aware of what condition was being conducted and of the session number rated a random sample of session tapes with regard to whether each of the topics or activities highlighted in the manual was actually addressed in the session.

PROCEDURE

Design Overview, Research Setting, and Therapists

We randomized participants to one of two types of group smoking cessation treatment: (a) comparison condition: scheduled reduced smoking plus health education and (b) CBT condition: scheduled reduced smoking plus health education plus cognitive behavior therapy mood management procedures. Each condition consisted of eight sessions of 90 minutes each. Treatment length was held constant so that any differences in outcome between the two conditions could not be attributed to extra treatment time (Haaga & Stiles, 2000). Each group consisted of approximately three to five participants with one of the seven graduate student therapists trained and then supervised weekly throughout the study. The supervisor (David Haaga, Ph.D.) is a licensed clinical psychologist with extensive training and experience in CBT and in training and supervising student therapists using these same treatments in a pilot study for this project (Thorndike, Friedman-Wheeler, & Haaga, 2006). To avoid confounding general therapist skill with treatment condition, therapists were crossed with condition. All assessments were conducted in the Department of Psychology at American University. Treatment group sessions were held in the psychotherapy training clinic housed within the same department.

Assessment Sequence

Smokers who called in response to study advertisements were screened over the phone. Those appearing likely to be eligible were scheduled for an in-person pretreatment assessment. Upon completion of the 8-session intervention, each participant was asked to complete an individual posttreatment assessment session approximately 1 week after the treatment's conclusion (1 month after quit date) as well as a 3-month posttreatment follow-up appointment.

Pretreatment assessment. All assessments were conducted individually. Along with an appointment reminder letter, participants received a self-monitoring form that requested the participant to monitor baseline levels of daily smoking and time spent asleep (information required for planning the details of scheduled reduced smoking). At the beginning of the pretreatment assessment, a trained master's or doctoral student completed written informed consent with the participant. The study was conducted in accordance with APA ethical standards and was approved by the American University IRB.

Participants were asked then to complete the Beck Suicidality Index (BSI). If any ideation was endorsed, the study staff conducted a clinical interview, provided hotline and referral information, and discussed the clinical management of the participant with the principal investigator. If the risk of suicide was none to minimal, the assessment session proceeded.

Participants were asked to provide a $40.00 deposit at the pretreatment assessment; $20 was returned upon completion of the posttreatment assessment, and the remaining $20 was returned upon the completion of the 3-month follow-up assessment.

In addition to smoking history, nicotine dependence, demographic, and depression vulnerability measures (as described in the Measures subsection), participants completed several questionnaires and computerized behavioral assessment tasks not relevant to this report (Schloss & Haaga, in press).

After individual pretreatment assessments were conducted with enough eligible participants to form a new group, and the group had been scheduled with a therapist, the project director would so

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inform the principal investigator. The PI then used a random number table to assign the group to a treatment condition (CBT or comparison) and informed the project director and therapist of this assignment. No subject variables were used to stratify random assignment. During pretreatment assessment, therefore, both assessors and participants were masked to treatment condition. During posttreatment and follow-up assessments such masking was not possible, but both participants and assessors remained masked to pretreatment depression proneness scores throughout the study, and smoking status reports were subject to biochemical corroboration and therefore should not be biased by knowledge of the treatment condition assignment.

Posttreatment assessment. Approximately 1 week after completion of the final treatment session for both the comparison and CBT conditions (i.e., 1 month after quit date), participants were scheduled for an individual posttreatment assessment session. Similar to the pretreatment assessment, participants were interviewed about their smoking status and then completed the same measures provided at the pretreatment assessment (excluding demographics and smoking history).

Three-month follow-up. Three months after the scheduled quit date, the study staff called group participants to inquire about their smoking status. If a participant indicated that she or he was abstinent, then that participant was scheduled to visit American University to have this report corroborated by an expired CO reading.

TREATMENTS

Treatment: Common Components

Each condition was guided by a treatment manual (available from the corresponding author) and incorporated an education component, as well as scheduled reduced smoking with a target quit date for all participants between the fourth and fifth therapy sessions. In each condition, all sessions were audiotaped for use in evaluating therapist adherence (see Results section).

Education

The psychoeducation component addressed nicotine dependence and withdrawal symptoms. Participants were encouraged to analyze how the negative consequences of smoking (e.g., health risks, financial costs) applied to them in particular, along with what benefits they might obtain from smoking cessation. In the first session, participants' smoking histories were discussed, along with any previous quit attempts and where they might have gone awry. The education component also emphasized the value of physical exercise, social support for nonsmoking, and self-reinforcement. Practical strategies for handling common temptation situations were discussed in each group, including very concrete strategies for the target quit date such as discarding all tobacco products from one's home and reminding one's friends and family of the participant's commitment to nonsmoking. Each group addressed concerns about weight gain following cessation, identifying for instance low-calorie snacks that could be used when a participant wants something in her or his mouth instead of a cigarette and exercise plans feasible for each participant's lifestyle and current fitness. Finally, each condition included the option of using nicotine replacement, and participants in all groups received information about the nicotine patch. Nicotine replacement was monitored by therapists but was neither provided nor required as part of the study treatment. As part of the consent process, participants had agreed not to participate in any other form of counseling for smoking cessation during the study, but nicotine replacement or medication treatment was allowed.

Scheduled Reduced Smoking

Participants in each treatment condition prepared for quit date using scheduled reduced smoking (Cinciripini, Wetter, & McClure, 1997). This method directs smokers to smoke only at designated times, on a predetermined schedule. The schedule gradually increases the amount of time between cigarettes and reduces the number of cigarettes smoked daily. In principle, adherence to such a schedule should make cessation easier because (a) gradual reduction of nicotine leads to diminished withdrawal symptoms after quit date, and (b) smoking at predetermined times should help break associations between the act of smoking and specific environmental or internal cues. Protocol instructions for this component of treatment were adapted from a manual by Cinciripini, Baile, and Blalock (undated). Previous research showed increased 1-year abstinence in a CBT smoking cessation program among those who had been assigned to scheduled reduced smoking prior to quit date, compared to scheduled, nonreduced smoking, nonscheduled/nonreduced smoking (i.e., abrupt cessation), or nonscheduled reduced smoking (i.e., number fading; Cinciripini et al., 1995).

CBT Condition: The Unique Component

The CBT mood management component of the program was based on Muñoz, Organista, and Hall

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(1993), a manual tested in Hall et al. (1994) and Hall et al. (1996), as well as a protocol for "negative affect reduction counseling" by Brandon and colleagues (Herzog et al., 2002). Participants in CBT groups were taught to identify and evaluate negative cognitions and their impact on mood. They were asked to keep a record of their negative automatic thoughts and to evaluate the evidence bearing on these thoughts. Therapists taught participants to identify more adaptive, alternative thoughts when minimal evidence for the automatic thought existed. Participants were encouraged to intervene and cope with negative thoughts through cognitive restructuring instead of smoking. Toward the end of treatment, participants discussed with the help of the other group members how they would cope with their individual high-risk situations in the future (similar plans were made in the comparison condition, but not in relation to the use of mood management techniques).

Data Analysis

The hypothesized interaction of Treatment Condition X Depression Proneness was tested using both 1-month and 3-month point prevalence abstinence data within the Generalized Estimating Equations (GEE) framework, as recommended by Hall et al. (2001). GEE was implemented using SPSS 17.0, with robust covariance estimator, the Logit link function, and unstructured correlation matrix specified. The within-subject effect was time (1 month and 3 months after target quit date), and the dependent variable was abstinence. Predictor variables in the model were depression proneness (DPI sample median split: ≥ 32 vs. ≤ 31), treatment condition (CBT vs. comparison condition), and the interaction of depression proneness and treatment condition.

Results

BASELINE COMPARISONS

Demographics, depression proneness, and cigarette smoking variables from the pretreatment assessment are reported separately by treatment condition in Table 1. Our sample scored about one-third to one-half a standard deviation higher in depression proneness than a sample of smokers not seeking treatment ($M = 26.00$, $SD = 9.69$; Haaga et al., 2004) and a large adult sample consisting of a mix of current smokers, former smokers, and never-smokers ($M = 28.56$, $SD = 11.50$; Brody, Hamer, & Haaga, 2005). Demographics and smoking variables from pretreatment are reported separately by level of depression proneness in Table 2. Differences were nonsignificant, with two exceptions. First, the highly depression prone were more likely to be Caucasian, and the less depression prone were more likely to be African American.[2] Second, as might be expected, the highly depression prone were more likely to have ever taken antidepressant medication. However, it should be noted that they did not exceed their low-depression-proneness counterparts in taking antidepressant medication as part of the current smoking cessation attempt, which was uncommon in our sample (6% of the high-DPI subsample, 10% of the low-DPI subsample).

PARTICIPANT FLOW AND ATTENDANCE AT ASSESSMENT AND THERAPY SESSIONS

Enrollment of participants in the study occurred from January 2005 through January 2007. Seventy-one percent of participants completed the 1-month post-quit-date assessment, and 82% completed the 3-month assessment. Eighty-five percent of the participants provided at least some follow-up data on smoking status.

Participants on average attended a little over one half of the 8 scheduled sessions. Comparison condition participants ($M = 4.60$, $SD = 2.81$) did not differ significantly from CBT participants ($M = 4.35$, $SD = 2.86$) in session attendance, $t(98) = 0.43$, $p > .6$. About one eighth (12%) of participants refused treatment altogether, attending zero sessions. In some cases, these were people who had been kept waiting for a group to form, and by the time it started they had quit smoking, sought help elsewhere, or had their schedules change in such a way that they could not attend. With treatment refusers excluded, average attendance still did not differ significantly between the Comparison condition ($M = 4.98$, $SD = 2.57$) and CBT condition ($M = 5.23$, $SD = 2.27$), $t(86) = 0.47$, $p > .6$.

IMPLEMENTATION OF INTERVENTIONS

All treatment sessions were audiotaped to facilitate clinical supervision as well as to assess the differentiability of the interventions and therapist adherence to the manualized interventions. With respect to differentiability, 15% of the session

[2] Despite this baseline difference in race as a function of depression proneness, race was not included as a covariate in our main analyses because (a) it was not prespecified as a covariate to include in planning the clinical trial, and adjusting for unplanned covariates because of baseline differences between groups may bias estimates of treatment effects (Altman, 1998; Raab, Day, & Sales, 2000); and (b) it was not predictive of outcome (focusing only on African Americans and Caucasians, the subgroups for whom we had enough participants to conduct an analysis, there was no significant relation between race and 3-month point prevalence abstinence, X^2 (1) = 1.16, p = .28.

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t1.1 Table 1

t1.2 Pretreatment Characteristics of Comparison Condition and CBT Participants

t1.3		Comparison (n=52)	CBT (n=48)
t1.4	Demographics		
t1.5	Mean (SD) Years of Age	42.73 (12.88)	42.98 (12.85)
t1.6	% female	48	54
t1.7	Race: % Caucasian	60	71
t1.8	% African American or Black	32	25
t1.9	% Asian American	2	2
t1.10	% other or declined to answer	6	2
t1.11	Ethnicity: % Hispanic	12	6
t1.12	Employment: % employed fulltime	52	60
t1.13	Smoking: Current		
t1.14	Mean (SD) cigarettes per day	17.48 (9.88)	18.06 (6.35)
t1.15	Nicotine dependence (FTND Mean (SD))	4.67 (2.38)	4.65 (2.32)
t1.16	Smoking and Quitting History		
t1.17	Mean (SD) years of smoking	23.09 (13.21)	23.92 (13.58)
t1.18	Median (25%ile, 75%ile) prior quit attempts	3 (1.5, 5)	2 (1, 5)
t1.19	Median (25%ile, 75%ile) days longest prior quit	60 (18, 240)	105 (21, 292)
t1.20	Depression Proneness: Mean (SD) DPI total	31.54 (11.98)	31.90 (10.71)
t1.21	Ever Taken Antidepressant medication (%)	50	48

t1.22 *Note.* CBT=Cognitive Behavior Therapy; FTND=Fagerstrom Test for Nicotine Dependence; DPI=Depression Proneness Inventory.

audiotapes were selected at random for evaluation by one of two graduate student raters. The raters were familiar with the treatment manuals but were masked to what condition was intended for each session tape. They correctly identified the session as either CBT or comparison 100% of the time (30 of 30 tapes).

A separate random sample of tapes (32 sessions) was selected for use in rating therapist adherence by one of two graduate student raters. For this task, the raters were made aware of the treatment condition and session number and were familiar with the manuals. They completed a checklist of the topics to be addressed in each session (typically 6 or 7 per session). Raters indicated that 100% of the intended topics were covered in the comparison condition sessions (and no CBT mood management content was detected in these sessions), with 99% of the intended topics covered in CBT sessions. All told, it appeared that raters could tell the conditions apart, and therapists were implementing essentially all of the methods called for by the protocol.

t2.1 Table 2

t2.2 Pretreatment Characteristics of High- and Low-Depression Prone Participants

t2.3		Low DPI (n=48)	High DPI (n=50)	t (96) (X^2) [U]	p
t2.4	Demographics				
t2.5	Mean (SD) Years of Age	43.48 (12.60)	41.82 (13.00)	0.64	.52
t2.6	% female	54	48	0.37	.54
t2.7	Race: % Caucasian	54	76	(5.91)	.02
t2.8	% African American	40	18		
t2.9	% Asian American	0	4		
t2.10	% other	6	2		
t2.11	Ethnicity: % Hispanic	6	12	(0.97)	.32
t2.12	Employment: % employed fulltime	56	54	(0.05)	.82
t2.13	Smoking: Current				
t2.14	Mean (SD) cigarettes per day	17.77 (6.07)	17.80 (10.23)	0.02	.99
t2.15	Nicotine dependence (FTND Mean (SD))	4.67 (2.14)	4.62 (2.56)	0.10	.92
t2.16	Smoking and Quitting History				
t2.17	Mean (SD) years of smoking	24.80 (13.19)	22.12 (13.57)	0.99	.32
t2.18	Median (25%ile, 75%ile) prior quit attempts	2 (1, 5)	3 (2, 5)	[883.5]	.13
t2.19	Median (25%ile, 75%ile) days longest prior quit	30 (21, 210)	112 (30, 364)	[811.5]	.16
t2.20	Ever taken antidepressant medication (%)	35	62	(6.92)	.008

t2.21 *Note.* FTND=Fagerstrom Test for Nicotine Dependence; Low DPI=Depression Proneness Inventory ≤31 at pretreatment; High DPI=Depression Proneness Inventory ≥32 at pretreatment; U=Mann-Whitney U test statistic.

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ADVERSE EVENTS

The Beck Depression Inventory (BDI) was administered at each assessment and treatment session with the aim of tracking any increases in depressive symptoms during treatment. An increase (at any point) of 8 points or more on the BDI relative to the pretreatment assessment was flagged as an adverse event. This value falls within the range (e.g., 6.64 in McGlinchey, Atkins, & Jacobson, 2002; 11 in Persons, Bostrom, & Bertagnolli, 1999) of estimates of the magnitude of BDI change signifying statistically reliable deterioration. By this definition, nine participants in CBT and five in the comparison condition experienced increased depressive symptoms, which was not a significant difference across conditions, X^2 ($df = 1$, $N = 100$) = 1.05, $p = .30$. Also, one participant in each condition experienced an increase from pretreatment to posttreatment in daily smoking rate.

MODERATOR EFFECT OF DEPRESSION PRONENESS ON EFFICACY OF CBT

To test the hypothesized interaction of depression proneness and treatment condition, we conducted a GEE analysis as described in the Method section. The main effect of treatment condition was not significant, Wald chi-square ($df = 1$) = 0.82, $p > .3$. Likewise, the main effect of depression proneness was not significant, Wald chi-square ($df = 1$) = 0.45, $p > .5$. However, the interaction of treatment condition and depression proneness was a significant predictor of abstinence, Wald chi-square (df = 1) = 4.04, $p < .05$, B = -2.01 (95% confidence interval = -3.97 to -.05).

The interaction effect was in the predicted direction. To illustrate it, Table 3 and Figure 2 show the 7-day point prevalence abstinence rates at each follow-up. For example, at 3 months post-quit date, among those high in baseline depression proneness abstinence rates were higher in CBT (35% to 22%), whereas among those low in depression proneness abstinence rates were higher in the comparison condition (33% to 10%).

Table 3
Seven-Day Point Prevalence Abstinence Percentages in Each Treatment Condition for High and Low Depression-Prone Smokers at Each Follow-up

		CBT	Comparison	OR
One Month After Quit Date				
Depression Proneness	High	41	29	1.68
	Low	16	50	0.19
Three Months After Quit Date				
Depression Proneness	High	35	22	1.94
	Low	10	33	0.31

Note. CBT = Cognitive Behavior Therapy; OR = odds ratio for efficacy of CBT within each level of depression proneness (High = Depression Proneness Inventory ≥32; Low = Depression Proneness Inventory ≤31).

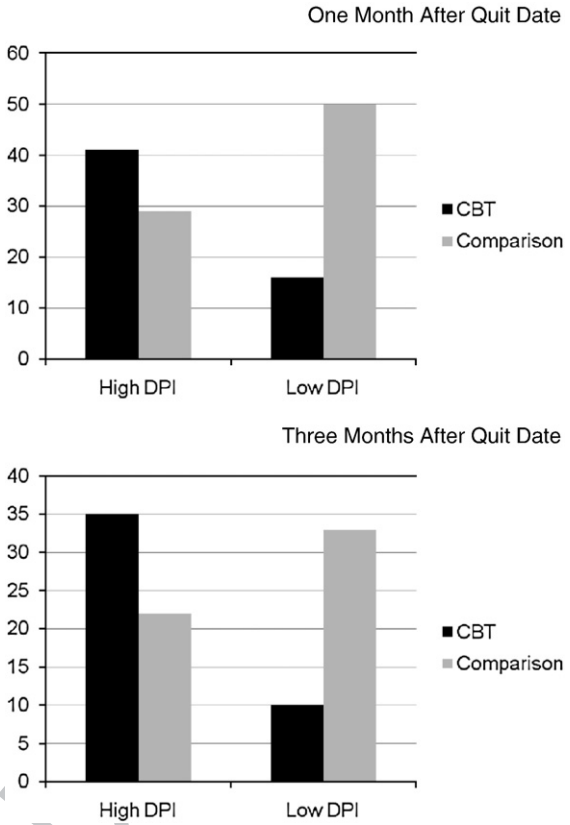

FIGURE 2 Seven-Day Point Prevalence Abstinence Percentages in Each Treatment Condition for High and Low Depression-Prone Smokers at Each Follow-up. *Note.* CBT = Cognitive Behavior Therapy; High DPI = Depression Proneness Inventory ≥ 32; Low DPI = Depression Proneness Inventory ≤ 31.

SECONDARY ANALYSES OF PROCESS VARIABLES

Collapsing across treatment condition, we examined in exploratory analyses a couple of potential process predictors of 3-month abstinence.

Session Attendance

In the pilot study for this project, we had found that participants who attended every treatment session were significantly more likely to become abstainers than were those who did not. This relation held in the current study as well. Of the 16 participants attending all 8 treatment sessions and providing 3-month follow-up data, 50% ($n = 8$) were 3-month abstainers, compared to 18% (12 of 66) of those who missed at least one session, chi-squared ($df = 1$, $N = 82$) = 7.07, $p < .01$, phi = .29, OR = 4.5 (95% CI = 1.41 to 14.39). This correlational finding does not establish a causal effect of session attendance. It could instead stem from reverse causality (e.g., those who are getting more out of treatment are potentially more likely to keep attending) or the effect of a

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third variable (e.g., high motivation to quit smoking could lead to both perfect session attendance and successful abstinence).

Adjunctive Use of Nicotine Replacement

About one-third of participants (34%) reported at any point having used nicotine replacement products. There was no difference between treatment conditions, X^2 ($df=1$, $N=96$) $=0.63$, $p>.4$, or between groups defined by median split on the DPI, X^2 ($df=1$, $N=94$) $=0.05$, $p>.8$, in the frequency of using nicotine replacement. Approximately one-third (10 of 31, 32%) of participants who used nicotine replacement were abstinent at the 3-month follow-up, a proportion that did not differ significantly from the abstinence rate (10 of 50, 20%) among those who chose not to use nicotine replacement, X^2 ($df=1$, $N=80$) $=1.55$, $p>.2$.

Discussion

In a randomized controlled trial of small-group smoking cessation interventions, self-rated depression proneness moderated response to CBT. In particular, abstinence was more likely among the highly depression-prone if they were assigned to a treatment condition incorporating the use of cognitive restructuring as a mood management method, whereas less depression-prone smokers fared better if assigned to a time-matched comparison condition omitting the cognitive restructuring component and mood management emphasis. Both conditions involved scheduled reduced smoking prior to quit date, health education, an emphasis on social support seeking inside and outside the group, planning for challenges in the early days after quitting, and other standard psychosocial methods.

It seems likely that CBT mood management treatment helps depression-vulnerable smokers by giving them other means, aside from smoking, to respond to the negative mood states that they often experience and that prompt relapse for some recent quitters. An issue for future empirical research is to pin down the nature of this mediating mechanism of the effects of CBT for depression-vulnerable smokers. Descriptive research has implicated poor coping skills as a correlate of depression vulnerability among smokers (Haaga et al., 2004; Kahler, Brown, Lloyd-Richardson, & Niaura, 2003; Rabois & Haaga, 1997), but to date there is no evidence that CBT has a specific effect in improving these coping skills (Thorndike et al., 2006). This possibility, and other candidate mechanisms, should be evaluated in samples large enough to support powerful analyses of mediation effects for treatments exerting specific benefits only for a subgroup (e.g., the more depression-prone) of participants, in other words "mediated moderation" (Muller, Judd, & Yzerbyt, 2005).

Conversely, for less depression-vulnerable smokers, inclusion of mood management techniques derived from CBT for depression might be something of a waste of time, addressing a concern that does not really apply to them. In this regard, it is important to note that the treatment conditions in this study were time-matched, so it is possible that the common components (health education, social support, weight management, self-reinforcement, etc.) could have received shorter shrift in the CBT condition, to the detriment of the low-depression-vulnerable smokers. No topics or techniques were eliminated altogether from the CBT condition, but a given topic (e.g., brainstorming strategies for rewarding oneself for achieving abstinence goals) might have been addressed at greater length in groups in the comparison condition given that they did not need to incorporate cognitive restructuring practice/instruction in sessions. This concern is particularly salient in our study given that (a) participants who began treatment averaged approximately 5 sessions attended, and (b) perfect attendance (8 of 8 sessions) was associated with better outcomes. Thus, it is possible that more treatment time is better and that the treatment dose for many of our participants was not high, so any time spent on a skill or topic a given participant does not need (e.g., mood management for those not prone to depression) is potentially problematic.

This issue poses a methodological challenge for any study employing a dismantling design to try to isolate the impact of a subset of treatment techniques. If treatment time is held constant, as in this study and in, for one example, a well-known dismantling investigation of cognitive therapy of depression (Jacobson et al., 1996), then the common treatment component(s) may be weaker in the experimental condition(s) incorporating extra components. On the other hand, if that problem is prevented by letting the combination treatment run longer, as in Hall et al. (1994), then additional treatment time per se becomes a viable rival hypothesis for the effects of the isolated treatment component.

Our findings are consistent with several previous demonstrations of an interaction of depression vulnerability with treatment condition in the study of CBT for smokers (e.g., Brandon et al., 1997; Brown et al., 2001; Haas et al., 2004; Hall et al., 1994; Hall et al., 1998; Patten et al., 2002) but are inconsistent with other reports of failures to replicate the effect (e.g., Brown et al., 2007; Hall et al., 1996). As described in the Introduction, we

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believe that measurement issues may be relevant in determining these inconsistencies and believe that our reliance on self-reported current depression proneness rather than history of depression is a methodological strength of this study. Future research could evaluate the role of measurement method more definitively either by (a) quantitatively reviewing the full set of studies of depression vulnerability as a moderator of CBT effects for smokers and determining whether effects are significantly heterogeneous and, if so, whether partitioning the studies by type of depression vulnerability measure reduces that heterogeneity, or (b) conducting a large prospective study incorporating multiple measures of depression vulnerability. A prospective-study methodology for resolving measurement issues in this area would have the advantage of determining whether our results are replicable and whether the DPI score (≥ 32) range selected in our sample on the basis of a median split is optimal as a marker of high vulnerability.

Methodological issues

The results reported in this manuscript should be interpreted in light of the strengths and limitations of the study. On the positive side, participants were randomly assigned to conditions, and self-reported abstinence was corroborated by expired air CO levels. Treatment conditions were differentiable by coders unaware of the intended condition, and therapist adherence ratings were high.

Methodological limitations include a modest sample size for studying moderator effects, making replication especially important. Interactions between patient variables and treatment conditions are potentially important both theoretically and practically (e.g., Latimer, Katulak, Mowad, & Salovey, 2005) but are often small effects and therefore somewhat erratic in individual studies (Noar, Benac, & Harris, 2007). Also, while differentiability of treatments was assured, and therapist adherence measured, there was no measure of therapist competence, leaving open the question of whether the CBT and comparison conditions were equally well executed.

Finally, the follow-up duration of 3 months after quit date was relatively brief. Longer term follow-ups may well have yielded lower 7-day point prevalence abstinence rates. For example, in both CBT conditions (one combined with buproprion, the other with placebo) in Brown et al. (2007), abstinence rates at 12 months were 18%. At the 2-month follow-up, the CBT abstinence rates were 25% and 26%, quite similar to the rate in this study at 3 months (see Table 3). Although a longer duration of follow-up would likely have lowered our absolute abstinence rates, we do not have a conceptual basis for predicting that longer follow-up would have eliminated the moderator effect we observed.

Conclusion

Thus, numerous questions remain for future research, such as the mediating mechanisms for, and durability at longer follow-ups of, the moderator effect of depression vulnerability on the efficacy of CBT for smokers. However, if future studies corroborate our findings, the results have straightforward clinical implications.

Most importantly, practitioners may be able to enhance smoking cessation outcomes by measuring depression proneness at baseline and incorporating CBT mood management interventions *only* for the highly depression-vulnerable. If our findings prove replicable, the practical effects of such a strategy would be important. Considering 3-month point prevalence data (Table 3), a clinician matching interventions to depression proneness (CBT for highly depression-prone, comparison for low depression-prone) could anticipate success with 34% of smokers, whereas a mismatching strategy would yield 16% successes, and a random strategy (use CBT or comparison without regard to depression proneness) 25% successes. Deliberate mismatching is unlikely as a real-world scenario, but matching relative to random allocation would result in important gains given the large population of smokers. The number-needed-to-treat for this difference (34% vs. 25%) is 11, meaning that for every 11 smokers treated, there would be one additional favorable result (abstinence in this case).

Also, clinicians could highlight for cigarette smokers that, while causal inferences are not warranted on the basis of our correlational findings, high engagement in the treatment (operationalized in our study as perfect attendance at 8 sessions of treatment) is at least associated with a substantially greater likelihood of successful abstinence.

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