CHANGES IN AFFECT FOLLOWING SMOKING CESSATION IN DEPRESSED SMOKERS

A Dissertation

Presented to

The Faculty of the Department

of Psychology

University of Houston

In Partial Fulfillment

Of the Requirements for the Degree of

Doctor of Philosophy

By

Amanda R. Mathew

May, 2012

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ABSTRACT

Smoking cessation for individuals with Major Depressive Disorder (MDD) represents an important clinical issue. It often has been hypothesized that withdrawing from cigarettes exacerbates affective disturbances in this population. However, studies testing the impact of smoking cessation on changes in affect in smokers with MDD are limited and equivocal. The current study examined affective processes in smokers with MDD undergoing a 12-week smoking cessation intervention (N = 49). The Positive and Negative Affect Scale (PANAS) was used to measure participants' positive affect (PA) and negative affect (NA) trajectories over the course of a quit attempt. Prolonged smoking abstinence was examined as a predictor of affective changes over time. Models also were run to examine pre-quit affective treatment response and the interaction of pre-quit affective response and abstinence status in predicting post-quit affect. Those who were prolonged abstainers at the 3-month follow-up showed significant increases in PA over the course of a quit attempt, as compared to nonabstainers. No significant differences in NA were found between prolonged abstainers and nonabstainers. Prequit affective trajectories significantly predicted post-quit affect, for measures of both PA and NA. Lastly, the interaction of abstinence status and early affective response was significant in predicting affect over time for NA, but not for PA. This suggests that, for those with less pre-quit improvement in NA, being abstinent at any given timpoint following the quit date is associated with lower values of NA than being nonabstinent. The current study highlights important differences in affect between prolonged abstainers and nonabstainers. Study results are discussed with regard to etiological models of smoking-MDD as well as implications for tailoring interventions to this at-risk group of smokers.

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Changes in Affect Following Smoking Cessation in Depressed Smokers

Cigarette smoking is the main preventable cause of cancer-related death worldwide (see Sasco, Secretan, & Straif, 2004 for review) and is responsible for 31% of all cancer-related deaths in the United States (Jemal et al., 2007). Despite clear links to mortality and morbidity, over 40 million adults in the U.S. currently smoke (U.S. Department of Health and Human Services, 2004). Quitting smoking decreases health risk and increases survival time among those who have developed medical problems (Samet, 1991). Approximately 70% of adult smokers would like to quit (Centers for Disease Control, 1995), but 90-95% of smokers who try to quit on their own (Garvey, Heinold, & Rosner, 1989), and 60-80% who attend treatment programs (Brown & Emmons, 1991; Schwartz, 1987), fail to quit smoking.

Selection Hypothesis

The selection hypothesis of smoking prevalence posits that smokers who fail to quit likely possess risk factors or vulnerabilities that make quitting more difficult (Coambs, Kozlowski, & Ferrence, 1989; Hughes, 1993; Hughes & Brandon, 2003). Those who continue to smoke despite extensive public health warnings are likely to be nicotine-dependent smokers with a history of unsuccessful quit attempts (Benowitz, 1998; Karan & Rosecrans, 2000). This nicotine-dependent group is more likely to be of a lower socioeconomic status and older age and is more likely to have smoking-related health problems such as cardiovascular disease, cerebrovascular disease, and emphysema (Fiore, 2000; Karan & Rosecrans, 2000).

In addition to these risk factors, psychiatric disorders have been implicated as an important risk factor for smoking. Cigarette smoking rates are about twice as high in those

with a mental disorder than those without (Lasser et al, 2000), and nicotine dependence is the most prevalent substance abuse disorder among individuals with mental illness (American Psychiatric Association, 1994). There appears to be a relationship between increased levels of psychological distress and smoking prevalence and average number of cigarettes smoked per day (Lawrence, Mitrou, & Zubrick, 2009). Individuals with mental illness comprise 44-46% of the U.S. cigarette market (Grant, Hasin, Chou, Stinson, & Dawson, 2004; Lasser et al., 2000), totaling 175 billion cigarettes per year or $39 billion in annual sales (Federal Trade Commission, 2005).

In particular, major depressive disorder (MDD) has been established as a critical risk factor for smoking behavior. Those with MDD have significantly higher rates of cigarette smoking than those in the general population (see Ziedonis et al., 2008, for review). Over 30% of patients with current MDD are daily smokers (Grant et al., 2004; Waxmonsky et al., 2005). Those with a history of MDD are more likely to smoke (Kandel & Davies, 1986), to smoke more heavily, and to be more nicotine dependent (Breslau, Kilbey, & Andreski, 1993, 1994; Breslau, 1995).

Conversely, smokers also have significantly higher rates of lifetime MDD than nonsmokers (Breslau, Kilbey, & Andreski, 1991). Current smokers were shown to have nearly twice the risk of an episode of major depression in the past month compared to non-smokers (Anda et al., 1990). Current smokers also report more depressive symptoms than non-smokers (Anda et al., 1990), more frequent and more severe episodes of MDD (Glassman, 1993), increased frequency of suicidal ideation when depressed, and higher rates of suicide (Glassman, 1993; Tanskanen, Viinamaki, Hintikka, Koivumaa-Hnkanen, & Lehtonen, 1998; Angst & Clayton, 1998; Malone, Waternaux, Haas, Cooper, Li, & Mann,

2003). The relationship between MDD and smoking is strongest in those with heavy smoking and nicotine dependence (Dierker, Avenevoli, Stolar, & Merikangas, 2002). Some suggest this may represent a dose-response relationship; that is, the heavier the smoking, the greater the risk of MDD (Bolam et al., 2011; Hemenway, Solnick, & Colditz, 1993; Tanskanen et al., 1998).

Combined MDD-Smoking Health Risk

Understanding the combined health risk of co-occurring mental illness and cigarette smoking is important from a public health perspective, as those with a psychiatric disorder who smoke represent a population at critical health risk. Those with chronic mental illness have been found to die, on average, 25 years earlier than the general population (Colton & Manderschied, 2006). Smoking makes a significant contribution to the excess mortality associated with mental illness (Ziedonis et al., 2008), by exacerbating major causes of death such as cardiovascular disease, lung disease, and diabetes mellitus (Colton & Manderschied, 2006; Mauer, 2006). In this population, smoking has also been found to relate to increased medical comorbidity (Brown, Inskip, & Barraclough, 2000; Colton & Manderscheid, 2006; Dixon, Postrado, Delahanty, Fischer, & Lehman, 1999; Joukamaa et al., 2001; Osby, Correia, Brandt, Ekbom, & Sparen, 2000), more psychiatric symptoms and increased rate of hospitalization (Dalack & Glassman, 1992; Desai, Seabolt, & Jann, 2001; Goff, Henderson, & Amico, 1992; Williams & Ziedonis, 2004; Ziedonis, Kosten, Glazer, & Frances, 1994), and increased risk of future suicidal behavior (Oquendo et al., 2004), relative to the general population. Additionally, cigarette smoking can reduce therapeutic blood levels of many psychotropic medications (Zevin & Benowitz, 1999), lessening their effectiveness in the management of psychiatric disorders.

A growing body of research suggests that the cancer risk to individuals who both smoke and have MDD is greater than the considerable risk of either condition alone. In a 12-year longitudinal study, Linkins and Comstock (1990) found that depressed mood and smoking interacted to predict a marked increase in relative cancer risk compared to nondepressed smokers. Findings from Knekt and colleagues (1996) provide further evidence for the combined health risk of depression and smoking by establishing that depression moderates the effect of smoking on lung cancer occurrence. Finally, depression and smoking also have been found to have synergistic effects on reducing NK cell lysis (Jung & Irwin, 1999), providing information on potential mechanisms through which MDD and smoking might combine to increase cancer risk.

Mental Health Care System & MDD-Smoking

Although smokers with MDD are at increased cancer risk relative to the general population, the mental health care system has been reluctant to address tobacco dependence in smokers with MDD (Himelhoch & Daumit, 2003; Hughes, 1998). In fact, a culture of reinforcing or "normalizing" cigarette smoking often is found in mental health facilities (see Hall & Prochaska, 2009 for review). Smoking is perceived as a way to fit in or cope with boredom when other options are limited (Morris, Waxmonsky, May, & Giese, 2009). Psychiatric patients received smoking cessation counseling during only 38% of their visits with a primary care physician and 12% of their visits with a psychiatrist (Thorndike et al., 2001). One study showed 59% of state psychiatric hospitals in the U.S. permitted patient smoking on their premises as recently as 2005 (Monihan et al., 2006). Additionally, the tobacco industry has marketed cigarettes to individuals with mental illness, provided tax-free

cigarettes to psychiatric facilities, and funded research promoting the "self-medication" hypothesis of smoking and mental illness (Prochaska et al., 2008).

This reluctance of the mental health care system to address nicotine dependence may be explained in part by mental health practitioners' beliefs about smoking cessation in this population. Though clinicians commonly believe mental health issues cannot be effectively treated until substance use is controlled (Riggs & Foa, 2008), this belief appears not to apply to cigarette smoking. Commonly reported barriers to offering nicotine dependence treatment include prioritization of mental health treatment, lack of appreciation for the negative health consequences of smoking, and the belief that individuals with mental illness are not willing or able to quit (Hall & Prochaska, 2009). These barriers suggest that smokers with mental illness may not be motivated to quit smoking secondary to increased stress and lack of stability. However, research data largely do not support this assertion. Studies of patients from both outpatient and inpatient mental health settings suggest that smokers with psychiatric disorders are about as likely to report interest in quitting smoking as the general population (Acton et al., 2005; Prochaska et al., 2004, 2006).

Additionally, another important barrier to treatment in this population is mental health practitioners' fear of exacerbating depressive symptoms with a quit attempt (Hughes, 1998; Covey, Glassman, & Stetner, 1997). Clinical observation suggests that smoking may assuage painful feelings, aid in regulating mood, and provide psychological relief to those with MDD (Glassman, 1990). Following from this, mental health practitioners have significant concerns about the emotional aftermath that results from smoking cessation, during both acute nicotine withdrawal syndrome and in the extended postcessation time period (Covey et al., 1997). Glassman and colleagues (1990) report several severe cases in which depressed smokers who

quit smoking experienced such marked mood disturbance that they were considered "psychiatric casualties" and advised to resume smoking. Clearly, the relationship between smoking abstinence and deterioration in psychological status represents an area of concern for mental health practitioners. However, few studies have explored whether smoking cessation truly causes the worsening of depressive symptoms in this high-risk population.

In sum, those with MDD are more likely to smoke than the general population and at critical cancer risk, but prevailing beliefs among mental health practitioners make it more difficult for them to obtain effective smoking cessation services. An important aim for researchers is to better understand affective processes in depressed smokers during quit attempts that will assist in development of interventions tailored to this high-risk population.

Etiological Models

In considering affective processes during quit attempts in smokers with MDD, it is important to understand etiological models proposed to explain co-occurring smoking and MDD. The *common factor model* emphasizes the role of a third environmental or genetic factor that underlies both smoking and MDD. Common genetic factors have been supported in the association between lifetime MDD, lifetime daily cigarette consumption and lifetime nicotine dependence (Kendler et al., 1993; Lyons et al., 2008). Additionally, Dierker and colleagues (2002) found evidence to support shared familial vulnerability to dysthymia and heavy smoking, but not MDD and heavy smoking. Researchers also have examined the role of behavioral and environmental factors common to both smoking and MDD, particularly in adolescence. Shared social and environmental predictors may be easy access and availability of cigarettes, poverty, and stressful environments (Morisano et al., 2009). Comorbid psychiatric disorders also are commonly related to smoking and MDD (Kalman et al., 2005).

Brown and colleagues (1996) found that smoking status and MDD among adolescents was not correlated after controlling for the presence of other psychiatric disorders.

It also has been hypothesized that smoking and MDD are related in a causal manner. The *primary depression model*, also known as the self-medication model, proposes an etiological link from MDD to cigarette smoking. According to this model, depressed individuals are more likely to initiate smoking and progress to nicotine dependence in part because nicotine serves to manage negative affect or aid in coping with distress related to depressive symptom development. In line with this model, young adults with a history of MDD were found to be three times more likely to progress to daily smoking than those without a history of MDD (Breslau et al., 1998) and almost twice as likely to be nicotine dependent (Breslau et al., 1993). Nicotine has been shown to produce an elevation in mood and subjective improvements in well-being (Kendler et al., 1993). It may be that depressive symptoms trigger cravings for nicotine because they previously have been alleviated by nicotine use (Carmody, 1989; Pomerleau & Pomerleau, 1984).

The primary depression model focuses on chemical properties of nicotine and suggests smoking may reduce dysphoria (Glassman et al., 1990; Khantzian, 1997) and that the reinforcing effects of nicotine's mood altering characteristics are especially powerful in depressed smokers. Nicotine is thought to have antidepressant properties (Balfour & Ridley, 2000; Tizabi et al., 1999), as it is considered to increase activity of the serotonergic system (Vazquez-Palacios et al., 2005). Nicotine also promotes the release of other neurotransmitters relevant to mood regulation, including dopamine, acetylcholine, GABA, and norepinephrine (Picciotto, 2003). Smoking a cigarette without nicotine has been found to produce less

dopamine release and less of a self-reported increase in mood than a cigarette containing nicotine (Brody et al., 2004; Brody et al., 2009).

Conversely, the *primary smoking model* proposes a causal relationship from smoking to MDD. This model posits that smoking increases risk of developing MDD due to alterations in neurotransmitter pathways following prolonged exposure to nicotine (Hughes, 1999; Markou & Kenny, 2002; Markou, Kosten, & Koob, 1998). The primary smoking model stresses the role of nicotine not as an antidepressant, but as a psychologically damaging drug of addiction (Parrott, 2000). Although smokers may believe smoking reduces their negative affect, negative affect ultimately is elevated due to repetitive episodes of acute nicotine withdrawal. In support of this model, researchers have found that nicotine causes changes in neurotransmitters that may relate to the etiology of MDD (Haustein, Haffner & Woodcock, 2002; Kendler et al., 1993; Picciotto et al., 2000).

The *bidirectional model* of MDD-smoking states that the ongoing, interactional effect between smoking and MDD may account for their high rate of comorbidity. It has been suggested that smoking could trigger MDD in biologically vulnerable individuals, which is subsequently maintained by continued smoking due to negative reinforcement processes (Mueser, Drake, & Wallach, 1998). Another proposed explanation in line with the bidirectional model is that acute or infrequent tobacco use may reduce negative affect, while chronic use may exacerbate negative affect (Munafo & Araya, 2010). Alternatively, smoking may promote development of MDD in some individuals but relieve depressive symptoms in others (Rodriguez et al., 2005). Accounting for heterogeneity in depression (e.g., subthreshold symptoms, single-episode, or recurrent; Burgess et al., 2002; Rodriguez et al.,

2005) as well as level of smoking may help elucidate bidirectional relationships between MDD and smoking.

Taken together, findings provide mixed results regarding etiological models of MDD-smoking. Traditionally, the primary depression or self-medication model has been accepted by researchers and clinicians alike. However, several recent studies have failed to support a key premise of the model – that smoking abstinence worsens depressive symptoms, even in those with a history of MDD (Berlin et al., 2010; Kahler et al., 2011; Torres et al., 2010). Additionally, the self-medication model has drawn concern from researchers as it may be used to "normalize" smoking in those with MDD (Prochaska et al., 2008). While some studies have supported the role of common factors, others have found evidence for a smoking-MDD association that cannot be accounted for by a third factor (see Boden, Fergusson, & Horwood, 2010 for review). Further studies are needed to examine questions that might further shed light on the etiology of the MDD-smoking relationship, particularly in a currently depressed sample.

The current study takes one step in examining a question that could shed light on etiological models of MDD-smoking. A key premise of two leading models of co-occurring smoking and MDD is examined: the primary smoking and primary depression models. If affective trajectories are shown to worsen over time following abstinence from cigarettes, this would support the primary depression model's premise that smoking cessation exacerbates mood problems in currently depressed smokers. However, if prolonged abstainers show improved affective trajectories following abstinence, this would lend support to the primary smoking model and the notion that smoking behavior itself worsens affect.

Affect and Smoking Cessation

Changes in affect are typically observed in smokers undergoing a quit attempt. Negative affect refers to a general dimension of subjective distress including anger, guilt, fear, anxiety, and depression; positive affect refers to a dimension of pleasurable engagement characterized by enthusiasm, high energy, and alertness (Watson, Clark, & Tellegen 1988). Generally, smokers' affective disturbance increases after quitting and decreases gradually over the next several weeks (Piasecki et al., 2003a), although many smokers do not follow this typical pattern (Burgess et al., 2002; Kahler et al., 2002; McCarthy, Piasecki, Fiore, & Baker, 2006; Piasecki & Baker, 2000; Piasecki, Fiore, & Baker, 1998; Piasecki, Jorenby, Smith, Fiore, & Baker, 2003b). Common symptoms of negative affect that co-occur with cessation are depressed mood, anxiety, nervousness, restlessness, and irritability (Hughes, Hatsukami, Mitchell, & Dahlgren, 1986). The smoking cessation-affect link is important to understand given its relevance to predicting smoking relapse (Kenford et al., 2002; Shiffman et al., 2007).

Changes in affect are thought to be particularly relevant for smokers with MDD during a quit attempt, although few studies have examined this question in a currently depressed sample. MDD is characterized by patterns of high negative affect and low positive affect (Clark & Watson, 1991). The notion based on clinical experience that cessation can provoke the worsening of major depression for smokers with MDD (e.g., Glassman et al., 1990) is prevalent in mental health settings (see Hall & Prochaska, 2009 for review). However, the majority of reports are based on case studies (e.g., Borrelli et al., 1996; Covey et al., 1990, 1997). Two studies that set out to specifically test this question in depression-vulnerable smokers reached different conclusions. Tsoh et al. (2000) found no relationship between abstinence status and the occurrence of an MDD episode over a one-year period. By

contrast, Glassman et al. (2001) found that a significantly higher percentage of smokers with a history of MDD who had successfully quit experienced a recurrence of depression, compared to current smokers (31% vs. 6%). However, some researchers have speculated that differences in outcome may be due to markedly different dropout rates between the two study groups (see Hall & Prochaska, 2009). In sum, the few studies examining depression following smoking cessation have produced equivocal and conflicting results, suggesting that further study is needed.

Positive affect is less frequently studied but also may be an important factor in smoking cessation, especially among smokers with MDD. In a previous study of depressed smokers, prolonged abstinence from smoking was found to predict increased positive affect over time (Blalock, Robinson, Wetter, Schreindorfer, & Cinciripini, 2008). Additionally, Strong and colleagues (2009) found decreases in positive affect prior to quitting and lower levels of positive affect each related to higher risk of smoking lapse. Further study is needed to elucidate the relationship between positive affect and smoking abstinence over time.

Finally, pre-quit affect also is expected to be an important predictor of post-quit affect. Previous studies of depressed individuals undergoing intensive cognitive therapy for MDD (Kelly, Roberts, & Ciesla, 2005; Tang & DeRubeis, 1999) have found that those whose affect improves most in early sessions of treatment maintain these gains and show higher rates of recovery than those without early gains. Additionally, Blalock and colleagues (2008) showed depressed smokers who became prolonged abstainers also began to improve early in treatment. In this study, pre-cessation changes in affect in smokers with current MDD were important in the trajectory of change in affect and critical to distinguishing prolonged abstainers from nonabstainers. Thus, in the proposed project, it is expected that

those who display early improvement in affect will be most likely to show further improvements in affective trajectories in the post-quit period.

Affect Following Smoking Cessation in Smokers with MDD

The few intervention studies conducted with currently depressed smokers have all found that quitting smoking does not result in a worsening of affect. In fact, preliminary data suggest that abstinence from smoking may improve affect over time. To date, four published intervention studies have targeted smokers with current MDD (Blalock et al., 2008; Hall et al., 2006; Munoz, Marin, Posner, & Perez-Stable, 1997; Thorsteinsson et al., 2001).

Hall and colleagues (2006) examined a staged care intervention strategy, including computerized feedback and smoking cessation treatment, for 322 smokers receiving outpatient treatment for depression. Participants all met criteria for current unipolar depression and reported smoking 1 or more cigarettes per day during the week before study recruitment. Abstinence rates for those in the treatment and control conditions were 24.6% and 19.1%, respectively, at the 18-month follow-up assessment. In this study, abstinence status was found to be unrelated to changes in mental health functioning as measured by the Short Form-36, days of hospitalization, or changes in depression severity (Hall & Prochaska, 2009).

Munoz and colleagues (1997) examined the efficacy of a self-administered mood management intervention for Latino smokers with both a history of and current MDD (n = 136). Those who received a smoking cessation guide plus mood management reported a 23% abstinence rate at 3 months, compared to an 11% abstinence rate for those with the smoking cessation guide alone. Though participants' overall depressive symptom scores improved

over the course of the study, the relationship between smoking abstinence and affect was not specifically examined.

Thorsteinsson and colleagues (2001) examined the effect of transdermal nicotine patches for smoking cessation among 38 currently depressed smokers. The sample was comprised of heavy smokers, as participants were required to have smoked at least one pack of cigarettes per day for at least a year. Although Thorsteinsson and colleagues had hypothesized an exacerbation of depressive symptoms following a quit attempt, results actually showed a slight improvement in mood ratings among those who remained abstinent over the 29 day study period (n = 24).

Thus, while smoking cessation is thought to exacerbate depression in MDD-positive smokers, research studies have not provided support for this assertion. In fact, Blalock and colleagues (2008) provided preliminary evidence that smokers with MDD may experience an improvement in affect post-quit. In this study, 21 smokers with current threshold and subthreshold depressive disorders participated in a pilot study of intensive smoking cessation interventions. Prolonged abstainers were found to have a decrease in depressive symptoms and craving and an increase in positive affect over time, as compared to nonabstainers. Notably, at the 3-month follow-up, 44% of prolonged abstainers were in complete remission of their baseline depressive disorders, as compared to 0% remission among nonabstainers.

In addition to these four intervention studies, newly emerging research supports the role of abstinence in improving affect over time in depression-vulnerable smokers. In a sample of heavy drinking smokers, Kahler and colleagues (2011) found prolonged abstinence to associate with a reduction in depressive symptoms over time. In a sample of smokers with past MDD, Berlin and colleagues (2010) found that abstainers showed significant

improvement on measures of depression, anxiety, and suicidality, while nonabstainers experienced significant mood deterioration. Finally, in a large, international sample of participants (n = 3056), Torres and colleagues (2010) found that smoking abstinence was not associated with increased risk of a major depressive episode, even for smokers with a history of MDD. Instead, continued smoking was shown to predict a major depressive episode shortly following a quit attempt.

Current Study

Smokers with Major Depressive Disorder (MDD) represent a critical population to target in smoking cessation efforts, as individuals with MDD are over-represented among smokers, face additional barriers to treatment, and are at greater health risk than smokers without MDD. It often has been hypothesized that smoking cessation exacerbates negative affect as part of the withdrawal process, particularly among smokers with MDD. However, the studies examining the impact of smoking cessation on negative affect in depressed smokers are limited and equivocal. The current study replicates and builds on previous research by addressing the critical question of how prolonged abstinence relates to affect in smokers with MDD. Findings from this study are expected to inform effective intervention efforts and inform theory, which may ultimately decrease smoking rates and reduce cancer risk.

Specific Aims and Hypotheses

The current study had three primary aims. The first aim examined whether smoking abstainers have different patterns of pre- and post-quit affective changers than relapsers. It was hypothesized that those who were prolonged abstainers at the 3-month follow-up would report decreased negative affect and increased positive affect on the Positive and Negative

Affect Scales (PANAS), both pre- and post-quit, relative to nonabstainers. The second aim investigated whether pre-quit affective response to treatment in the first 6 pre-quit treatment sessions was related to post-quit affective response. It was hypothesized that those who showed significant improvement in their affective trajectories over the first 6 pre-quit treatment sessions would report decreased negative post-quit affect and increased post-quit positive affect, compared to those who do not show improvement in affect during the pre-quit treatment sessions. The third aim examined the interaction of early affective treatment response and abstinence at any given timepoint following the quit date in predicting post-quit affect. It was hypothesized that those who showed the most improvement in pre-quit affect and who were abstinent at any given timepoint following the quit date would show the greatest improvement in post-quit affect.

Method

Overview

Data were drawn from a randomized, controlled clinical trial of a mood-focused intervention for smoking cessation in depressed smokers. The primary aim of the parent project was to compare the Cognitive Behavioral Analysis System of Psychotherapy in combination with standard smoking cessation treatment (CBASP/ST) to Health Education plus standard smoking cessation treatment (HE/ST) in smokers with current chronic depressive disorders (dysthymia or MDD). CBASP is an intervention that has been shown to be efficacious in the treatment of chronic MDD. The proposed study extended the project to examine the separate question of affective trajectories in the post-quit period, controlling for treatment condition.

Participants

Forty-nine participants comprised the current study sample. Participants were recruited from Houston and the surrounding metropolitan area by means of a multi-dimensional, targeted, and strategically planned outreach program. All recruitment messages stated that the study would recruit individuals who wished to quit smoking and are also having problems with depression, to evaluate a smoking cessation treatment specifically designed to help this group of smokers. Advertising included newspaper ads, radio and TV advertisements, public service announcements, flyers, posters, displays, and brochures. The study description and recruitment messages also reached internet users through the MD Anderson webpage under smoking cessation resources.

In order to be eligible, participants were required to smoke 5 or more cigarettes per day at baseline and meet criteria for a chronic form of a depressive disorder (recurrent MDD, major depressive episode with a duration of 2 years or more, or dythymic disorder). Participants must have been experiencing or in partial remission of a major depressive episode at baseline. In addition, participants were required to score ≥ 8 on the Patient Health Questionnaire (PHQ), indicating at least moderate depressive symptoms, at the baseline session. Mood diagnoses were based on interview, at baseline, with the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1994). The SCID is a widely used clinician-administered interview that is used to assess mood disorders according to DSM-IV criteria. Project staff members were trained to administer the SCID and periodic checks of interrater reliability were conducted.

Exclusion criteria included a history of psychotic or bipolar disorder or current principal Axis I disorder other than unipolar depression or nicotine dependence. Participants who reported current psychotherapy or current use of an antidepressant also were ineligible

for the study. Participants who endorsed more than moderate levels of suicidal ideation, severe levels of depressive symptoms, vegetative symptoms, and/or severe symptoms of secondary psychiatric disorders were considered in need of immediate treatment for their disorder. They were excluded from the study and provided with appropriate referrals. Lastly, those with medical contraindications for use of the nicotine patch were excluded from the current study.

Participants found eligible for the study were fully consented regarding its risks and benefits. Approval was obtained from the University of Texas MD Anderson Cancer Center Institutional Review Board.

Procedures

Participants were assigned to treatment conditions using a form of adaptive randomization called minimization that balanced groups with respect to patient characteristics, including gender, baseline rate of smoking, and baseline depressive symptom severity. A total of 12 treatment sessions were provided by clinical psychologists in the Department of Behavioral Science at the University of Texas MD Anderson Cancer Center. Participants were instructed to set a quit date following week 6 of treatment. Nicotine replacement therapy (NRT) was also provided to participants in both groups. Participants were provided with a total of 8 weeks of NRT, beginning on the scheduled quit date and tapering from patches with 21 mg nicotine dosages to 14 mg and 7 mg patches. Data were collected from participants at 12 weekly treatment sessions, as well as follow-up sessions at 3 and 6 months.

Measures

Participants were assessed on a variety of interview, self-report, and biochemical measures at baseline and each treatment session, and at 3- and 6-months after the targeted quit date. Relevant measures to the current study are discussed below.

Positive and Negative Affect Scale. The Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988) was administered at each treatment session and at the 3- and 6-month follow-up visits. The PANAS is a widely-used self-report measure of the experience of positive and negative affect within the past week. The measure consists of two 10-item mood scales, one for positive affect and one for negative affect. Participants are asked to rate various feelings and emotions on a scale of 1 (very slightly to not at all) to 5 (extremely). The PANAS is commonly used as a measure of affective change in patients undergoing smoking cessation treatment. Cronbach's alpha reliabilities for the measure were shown to be acceptably high, ranging from .86 to .90 for PA and from .84 to .87 for NA (Watson et al., 1988).

Beck Depression Inventory-II (BDI-II). Baseline severity of depressive symptoms was measured with the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a widely used 21-item self-report measure developed to assess depressive symptoms in both normal and clinical populations.

Fagerström Test for Nicotine Dependence. Baseline severity of nicotine dependence was measured with the Fagerström Test for Nicotine Dependence (FTND, Heatherton, Kozlowski, Frecker, & Fagerström, 1991), a questionnaire that assesses various components of smoking behavior such as daily intake, difficulty in refraining from smoking, and other information related to patterns of intake.

Timeline Follow Back. Participant report of daily smoking behavior was collected with the Timeline Follow Back procedure (TLFB; Brown, Burgess, Sales, Evans, & Miller, 1998). A computerized program was used to provide the interviewer with a calendar on which to record the amount of cigarettes smoked on each day since last contact, highlighting the days between contacts for easy reference.

Biochemical Verification of Smoking Status. Self-reported smoking status was verified with breath samples providing biochemical verification of abstinence. Participants' expired CO was measured at each treatment session and follow-up visit using a Bedfont EC50 Micro III Smokerlizer (Bedfont Scientific, Meford, NJ).

Abstinence Group Categorization. Due to the difficulties likely experienced by currently, chronically depressed smokers in quitting smoking, prolonged abstinence status definitions were somewhat relaxed from the standard guidelines (Hughes et al., 2003). Prolonged abstinence was defined as a self-report of sustained abstinence beginning at end of treatment (EOT) and a CO value of <10 parts per million at the 3-month follow-up. Beginning at EOT, relapse was defined by either 7 consecutive days of smoking or smoking at least one cigarette over 2 consecutive weeks within the period of interest (Hughes et al., 2003). Participants who met criteria for prolonged abstinence were categorized as prolonged abstainers; those who did not meet these criteria were categorized as nonabstainers. Seven-day point prevalence abstinence was defined as self-report of no smoking, not even a puff, in the 7 days prior to the selected time point of interest.

Analytic Strategy

Mixed-effects models (SAS Proc Mixed) were used to examine proposed study aims. Generalized mixed model regression is a method of repeated measures analyses that allows

for inclusion of both categorical and continuous independent variables and for appropriate modeling of covariance structures where observations are correlated across time. The mixed-model approach provides a generalization to the classic linear regression model, using likelihood functions to estimate effects in place of least squares (McCullagh & Nedler, 1989). Mixed-model analyses are well suited for repeated measures designs as they allow for estimates of the correlation structure of the residuals and can efficiently handle unbalanced designs and missing data without excluding participants or imputing values (Gibbons, Hedeker, & Waternaux, 1988; Gibbons et al., 1993).

All analyses were run covarying for treatment group. Subjects were included as a random effect. Residual error variances over time were modeled as a heteroscedastic random effect, using an autoregressive function, but were not shown to improve model fit over the homoscedastic model (Snijders & Berkhof, 2008). Missing data on questionnaire measures was evaluated to determine whether missing data were distributed randomly. Missing abstinence data was addressed by using appropriate missing value imputation techniques for variables in the data analysis, including pattern mixture models.

For Aim 1.1, models were run to examine the interaction of abstinence status (prolonged abstinence or nonabstinence at the 3-month follow-up) as a between-subjects factor and time (pre-quit treatment sessions 1-6) as a within-subjects factor on pre-quit affect. For Aim 1.2, models examined the interaction of abstinence status (prolonged abstinence at the 3-month follow-up) as a between-subjects factor and time (post-quit treatment sessions 7-12 and 3- and 6-month follow-ups) as a within-subjects factor on post-quit affect. As PA and NA represent orthogonal constructs (Watson, Clark, & Tellegen, 1988), separate models

were run to examine trajectories for PA and NA. Higher order effects for time were modeled but were not shown to be significant, so only linear effects of time were reported in results.

To address Aim 2, pre-quit slope (Bayes estimate) were used as a parameter for pre-quit affective trajectory. PA and NA were modeled separately. Models were run examining pre-quit slope (at pre-quit treatment sessions 1-6) for both PA and NA as a between-subjects factor in predicting post-quit PA and NA trajectories, respectively.

For Aim 3, the association of smoking abstinence and affect was tested at concurrent assessments following the quit date. Abstinence status was defined as 7-day point prevalence at each post-quit treatment session. Models were run examining the interaction of pre-quit affective response to treatment (high- or low-responders) as a between-subject factor and abstinence status (abstainers or nonabstainers at treatment sessions 8-12) as a within-subject factor in predicting post-quit affect over time.

Results

Demographic, Smoking, and Depression-Related Characteristics

The study sample was comprised of 49 participants (61% female) with chronic depression. Participants ranged in age from 21 to 61 (M = 41.9; SD =11.5). The sample was ethnically diverse, with 69% of the sample being Caucasian, 25% African American, 4% Hispanic/Latino, and 2% Asian American. Participants reported smoking an average of 18.5 cigarettes per day at baseline (SD = 8.6). At visit 1, participants reported an average score on the Beck Depression Inventory (BDI-II) of 26.3, which falls within the moderately depressed range. The correlation between NA and PA scores at baseline was -.14, suggesting that the scales share approximately 1.4% of their variance in the current sample. Demographic, smoking, and depression-related characteristics of the sample by abstinence groups are

presented in Table 1. One-way analyses of variance were used to evaluate the abstinence group differences on continuous measures, whereas chi-square tests were used to evaluate abstinence group differences on categorical measures. In order to accommodate a zero cell in the marital status variable, Fisher's exact test was performed in place of chi-square test, and no significant differences were found (p = .17). There was no main effect of abstinence group on any of the smoking and depression-related variables. Prolonged abstainers were more likely to be women than men, $F(1, 49) = 5.37$, p = .021.

Aim 1

Analyses were conducted to examine whether prolonged smoking abstinence was a predictor of affective changes over time, both pre- and post-quit. First, models were run to examine the interaction of abstinence status (prolonged abstinence or nonabstinence at the 3-month follow-up) as a between-subjects factor and time (pre-quit treatment sessions 1-6) as a within-subjects factor on affect. Days from quit date were used as a proxy for time to best account for missing or makeup visits. All models were run covarying for treatment group. The interaction of abstinence group with days to quit date was significant for pre-quit PA, $F(1, 196) = 4.98$, p = .027. Slopes indicated that prolonged abstainers reported increased PA over time (PE = .105, SE = .028), relative to nonabstainers (PE = .026, SE = .021). The interaction of abstinence group with days to quit date was not significant for pre-quit NA, $F(1, 196) = 0.37$, p = .544.

Second, models were run examining the interaction of abstinence status (prolonged abstinence at the 3-month follow-up) as a between-subjects factor and time (post-quit treatment sessions 7-12 and 3- and 6-month follow-ups) as a within-subjects factor on affect.

Models were run covarying for treatment group. The interactions of abstinence group with days to quit date was not significant for the post-quit sessions for either PA or NA.

Alternatively, models were also run examining changes in affect over the entire course of the quit attempt (visits 1-12 and 3- and 6-month follow-ups). For the measure of PA, the interaction of abstinence groups with days to quit date was significant, $F(1,449) = 6.69$, $p = .010$. As seen in Figure 1, slopes indicate that prolonged abstainers report increased PA over time ($PE = .046$, $SE = .007$), relative to nonabstainers ($PE = .024$, $SE = .005$). For the measure of NA, the interaction of abstinence group with days to quit date was not significant, $F(1, 449) = 2.58$, $p = .109$, indicating that prolonged abstainers and nonabstainers did not significantly differ in NA (see Figure 2).

Aim 2

Analyses were conducted to test whether pre-quit affective response to treatment in the first 6 pre-quit treatment sessions predicted post-quit affective response. Models were run using Bayes estimates of pre-quit slope, time, and their interaction as predictors, covarying for treatment group and abstinence status, with post-quit scores as dependent variables. Days from quit date was used as a proxy for time to best account for missing or makeup visits. There was a significant main effect for pre-quit PA slope, $F(1,36) = 11.43$, $p = .002$, indicating that pre-quit increases in PA slope were positively associated with post-quit PA scores ($PE = 36.09$, $SE = 10.67$). There was also a significant main effect for pre-quit NA slope, $F(1,36) = 25.77$, $p < .0001$, indicating that pre-quit decreases in NA slope were positively associated with post-quit NA scores ($PE = 49.76$, $SE = 9.80$).

For PA, a significant main effect was found for days from quit date, $F = 11.43$, $p = .002$, indicating that post-quit PA scores were positively associated with days from quit date

(*PE* = .010, *SE* = .005). For NA, no significant main effect for days from quit date was found, $F(1,216) = 1.71$, $p = .19$.

Interaction effects between pre-quit affect and time were then examined. For PA, the interaction of pre-quit slope and days from quit date was not significant, $F(1, 215) = 2.04$, $p = .155$. For NA, the interaction of pre-quit slope and days from quit date was not significant, $F(1, 215) = .37$, $p = .542$.

Aim 3

Analyses were conducted to examine the interaction of early affective treatment response and abstinence at any given post-quit timepoint on post-quit affect. For Aim 3, abstinence status was defined as 7-day point prevalence at each post-quit treatment session (sessions 8-12), in order to identify dynamic, week-by-week effects of abstinence on affect. Models were run examining the interaction of pre-quit affective response to treatment as a between-subject factor and abstinence status as a within-subject factor in predicting post-quit affect over time. Models were graphed according to procedures in Aiken & West (1991). For PA, the interaction of pre-quit affective response and abstinence status was not significant, $F(1, 115) = 2.03$, $p = .157$ (see Figure 3). For NA, the interaction of pre-quit affective response and abstinence status was significant, $F(1,115) = 8.23$, $p = .005$). As seen in Figure 4, slopes indicate that those who showed the most improvement in pre-quit NA and were abstinent following the quit date showed the greatest improvement in post-quit NA (PE = 50.05, SE = 17.45). Models were also run examining the three-way interaction of pre-quit affective response to treatment, abstinence status, and time in predicting post-quit affect. Three-way interaction models were not significant for either PA, $F(1, 112) = 0.65$, $p = .422$, or NA, $F(1, 112) = 0.59$, $p = .445$.

Discussion

In summary, those who were prolonged abstainers at the 3-month follow-up showed significant increases in PA over the course of a quit attempt, as compared to nonabstainers. No significant differences in NA were found between prolonged abstainers and nonabstainers. Prequit affective trajectories significantly predicted post-quit affect, for measures of both PA and NA. Lastly, the interaction of abstinence status and early affective response was significant in predicting affect over time for NA, but not for PA. This suggests that, for those with less pre-quit improvement in NA, being abstinent at any given timpoint following the quit date is associated with lower values of NA than being nonabstinent.

Results of the current study highlight important differences between prolonged abstainers and nonabstainers that emerge early in treatment and persist over the course of a quit attempt. Several factors may explain the differences in affect observed between the two abstinence groups. It is possible these affective changes between prolonged abstainers and nonabstainers are consistent with neurobiological or physical processes. Motivational systems have been shown to distinguish groups of drug users, with differences in how individuals process drug relative to nondrug rewards (Buhler et al., 2010). It may be that differences observed in PA in the current study suggest that prolonged abstainers were more able to respond to non-cigarette rewards in the environment than nonabstainers. Additionally, biological mechanisms such as improved physical function or elimination of repeated nicotine withdrawal may distinguish prolonged abstainers from nonabstainers.

An alternative explanation is that the differences observed between abstinence groups represent differences in psychological variables and response to intensive intervention. It may be that those who respond to intensive intervention with increased perceptions of self-

efficacy and self-esteem and decreased perceptions of stress are more likely to succeed in a quit attempt (Cohen & Lichtenstein, 1990). Abstinence self-efficacy was recently shown to be a robust mediator of treatment effects among those in an intensive smoking cessation intervention (Hendricks et al., 2010). Further study is needed to elucidate the nature of the affective differences observed between the two abstinence groups.

The current study also highlights the important role of early affective response to treatment as a predictor of overall changes in affect. Replicating past research (Blalock et al., 2008), results show that pre-cessation changes in affect in smokers with current MDD were important in distinguishing prolonged abstainers from nonabstainers. While early gains have been shown to be predictive of positive treatment outcome in the depression literature (e.g., Kelly, Roberts, & Ciesla, 2005; Tang & DeRubeis, 1999), limited research has explored treatment processes in the context of smoking cessation. It may be that those whose affective trajectories improve early in treatment are better able to maintain these gains and reduce risk of relapse to smoking. Results underscore the importance of early affective changes that may serve as a useful target for smoking cessation intervention in this population.

Although extant research has focused on negative affect and depressed mood as barriers to smoking cessation, the current study adds to emerging research supporting the critical role of low positive affect to smoking cessation efforts (Leventhal et al., 2008; McCarthy et al., 2008). It may be that nonabstainers represent a subset of depressed smokers with significant deficits in PA that call for tailored intervention. Accordingly, current findings may provide additional rationale for interventions designed to enhance positive affect among smokers, such as behavioral activation and positive psychology. Indeed, a preliminary trial has found behavioral activation to be a promising treatment for smoking

cessation among smokers with elevated depression symptoms (e.g., MacPherson et al., 2010). It may be that interventions addressing not only management of negative mood, but fostering of positive affect, can improve smoking cessation rates for depressed smokers.

Results of the current study provide partial support for the primary smoking model of smoking-depression co-occurrence, which posits that smoking increases risk of developing MDD due to alterations in neurotransmitter pathways following prolonged exposure to nicotine (Hughes, 1999; Markou & Kenny, 2002; Markou, Kosten, & Koob, 1998). Although the hypothesis that prolonged abstainers would experience a decrease in NA was not supported, prolonged abstinence was not associated with a worsening of NA, as would be consistent with the primary depression or self-medication model. Thus, prolonged abstinence status did not predict an exacerbation of NA, and in fact predicted improvement in levels of PA. This finding supports a key premise of the primary smoking model; namely, that abstinence from cigarettes supports improvements in psychological functioning over time.

The current study has several limitations. First, the study design does not allow definitive conclusions to be drawn regarding the directionality of relationships between abstinence status and affect. A causal role cannot be established when participants are not randomized to abstinence. It is possible that changes in affect precipitated changes in abstinence from cigarettes, rather than the other way around. Additionally, several other relevant factors must be considered. Participants in the current study received time-intensive counseling as well as nicotine replacement therapy. To address questions of generalizability, it will be important for future research to examine the effect of prolonged abstinence on affect in different types of treatment, including brief interventions.

Second, the relatively small sample size may have limited power to test some study aims. Sample size also prevented testing time-varying definitions of abstinence status following the quit attempt. However, balancing the limitation of small sample size, one strength of the current study was the use of an especially high-risk group of currently, chronically depressed smokers that have not often been included in other research studies. Though the generalizability of these findings to less severe populations of smokers may be limited, the current sample afforded the opportunity to examine affective processes in an especially at-risk group of smokers.

Lastly, the scope of the current study included examining affective changes but not other components of the withdrawal process from nicotine. Although NA is thought to be an important component of the withdrawal process, it is also important to examine other aspects of withdrawal, such as craving.

Taken together with other recent findings (e.g., Berlin et al., 2010; Blalock et al., 2008; Kahler et al., 2011; Torres et al., 2010), results of the current study show that significant improvements in psychological functioning can be observed among those who successfully quit smoking even in the most severe psychiatric group when assessed in the context of intensive treatment. These results have implications for future smoking cessation efforts among high-risk psychiatric populations of smokers. Although smokers in this group are not often encouraged to quit, it may be that smokers who attempt to quit are likely to feel more rather than less psychologically healthy. Those who are able to achieve prolonged abstinence may experience significant improvement in affect over time. This finding adds to burgeoning research that supports the role of abstinence in improving positive affect over

time in depression-vulnerable smokers. This study contributes unique findings from a currently, chronically depressed sample of smokers.

Future directions include the identification of characteristics that distinguish prolonged abstainers from nonabstainers and the measurement of these variables as potential mediators of treatment. Further examining these differences between abstinence groups may clarify the nature of co-occurring smoking and depression. Ultimately, findings may provide valuable information on tailoring interventions to psychiatric groups of smokers.

Table 1

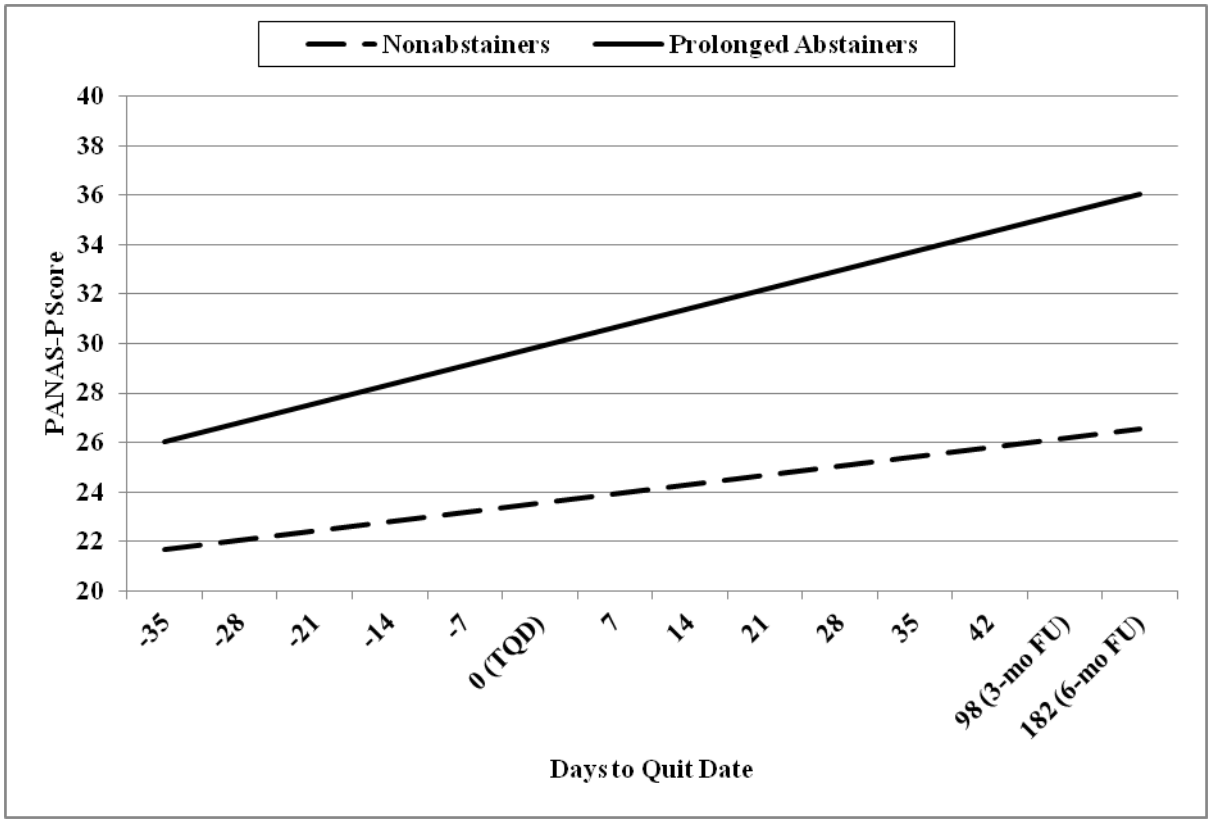
Baseline Demographic, Smoking-Related, and Depression-Related Characteristics of the Sample

	Abstinence group		
Characteristic	Nonabstainers	Prolonged abstainers	Total
n	35	14	49
Female (*%*)	10 (28.57)	9 (64.29)	19 (38.78)*
Age Mean years (*SD*)	41.46 (12.05)	43.00 (10.43)	41.90 (11.53)
Married (*%*)	7 (20)	0 (0)	7 (14.29)*
White (*%*)	26 (74.29)	8 (57.14)	34 (69.39)
Some college/bachelor's degree (*%*)	23 (65.71)	11 (78.57)	34 (69.39)
Mean expired carbon monoxide (*SD*)	13.31 (8.23)	14.14 (11.26)	13.55 (9.09)
# years smoking (*SD*)	21.14 (13.02)	25.79 (10.47)	22.47 (12.42)
Age started smoking (*SD*)	17.91 (4.97)	16.00 (4.96)	17.37 (4.99)
Mean FTND score (*SD*)	5.27 (1.82)	5.38 (2.60)	5.30 (2.04)
Mean BDI score (*SD*)	25.54 (8.64)	28.21 (7.53)	26.31 (8.35)
Primary Diagnosis: MDD (*%*)	31 (88.57)	14 (100)	45 (91.84)
Primary Diagnosis: Dysthymia (*%*)	4 (11.43)	0 (0)	4 (8.16)

Note. MDD = Major Depressive Disorder; FTND = Fagerstrom Test for Nicotine Dependence; BDI = Beck Depression Inventory; *$p < .05$.

Figure 1

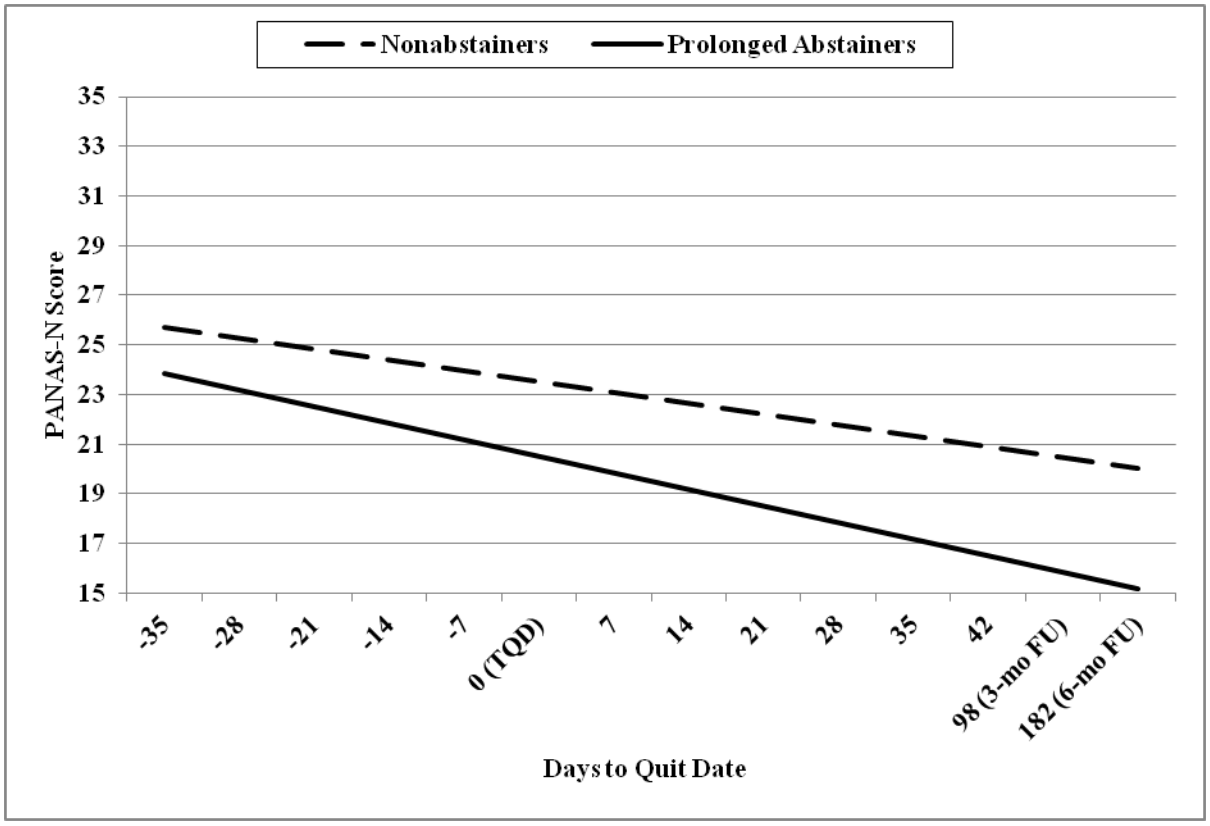
Positive and Negative Affect Scale-Positive Affect (PANAS-P) Scores in Prolonged Abstainers versus Nonabstainers



Note. TQD = target quit date.

Figure 2

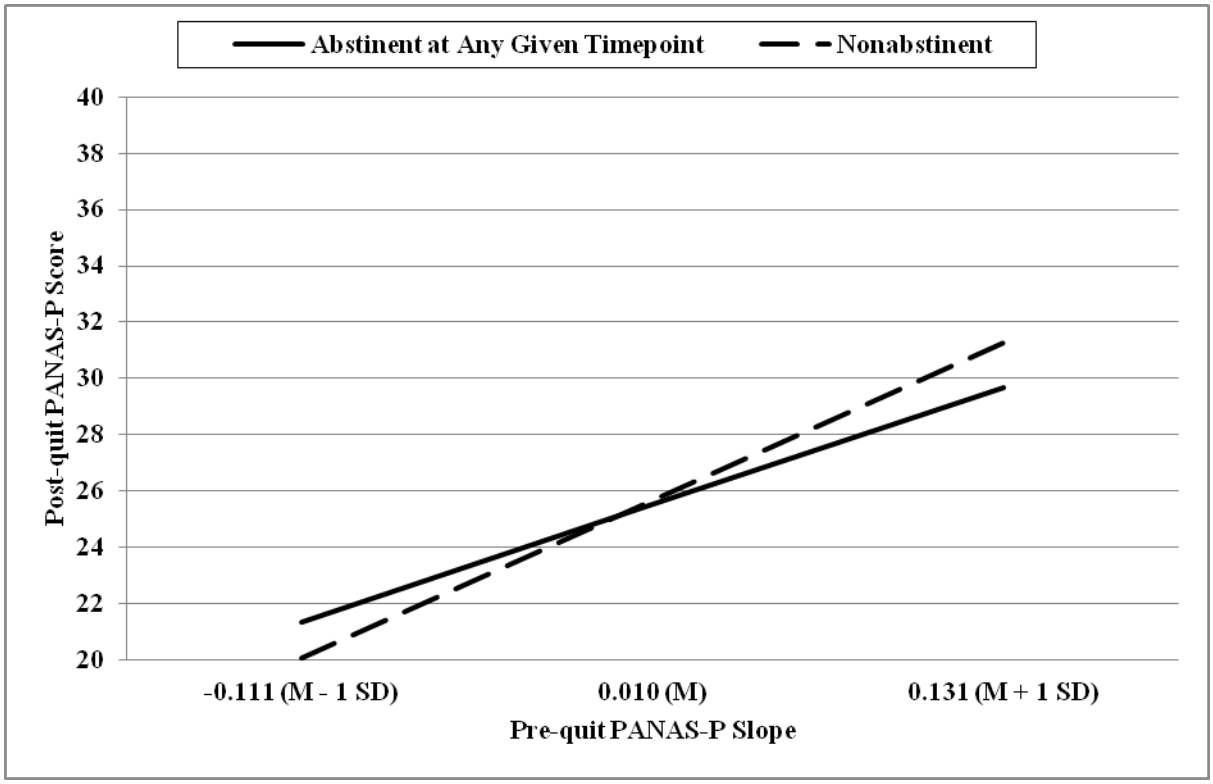
Positive and Negative Affect Scale-Negative Affect (PANAS-N) Scores in Prolonged Abstainers versus Nonabstainers



Note. TQD = target quit date.

Figure 3

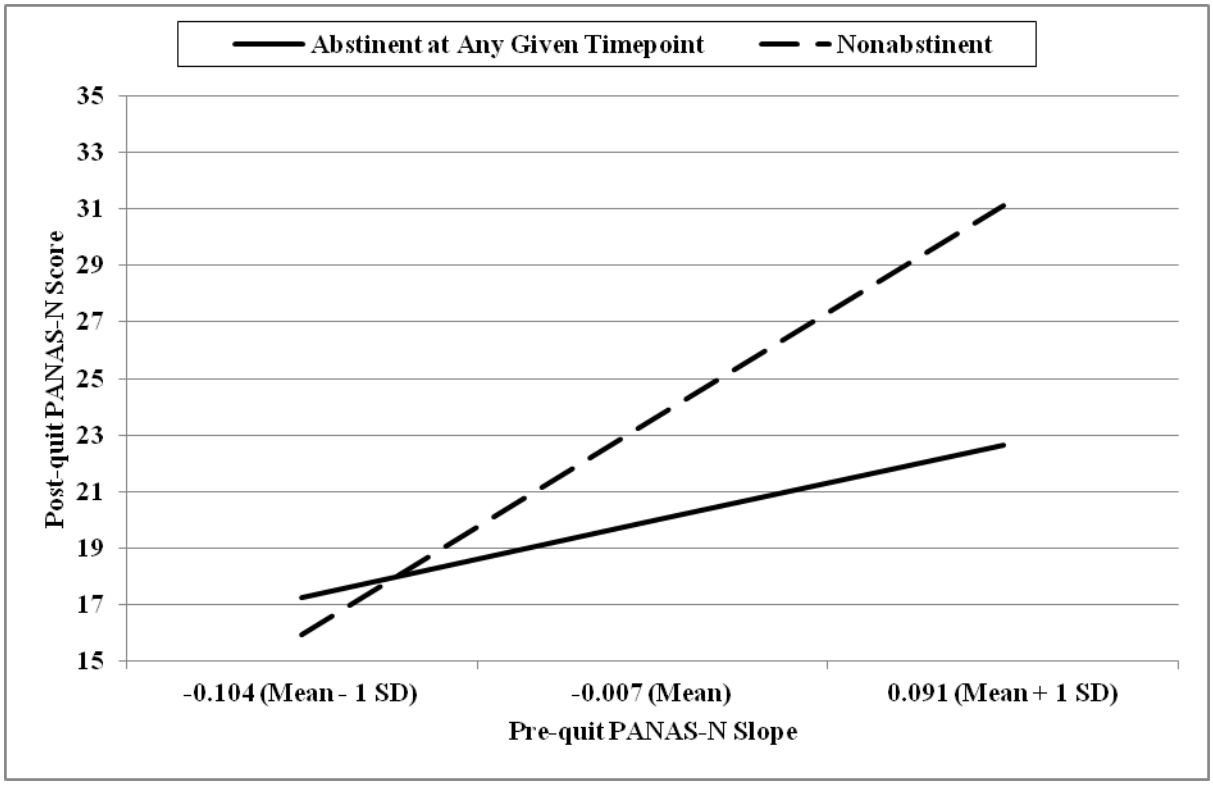
The Nonsignificant Interaction between Pre-quit PANAS-P Slope and 7-day Point Prevalence Abstinence Status on Post-quit PANAS-P Scores



Note. SD = standard deviation.

Figure 4

The Significant Interaction between Pre-quit PANAS-N Slope and 7-day Point Prevalence Abstinence Status on Post-quit PANAS-N Scores



Note. SD = standard deviation.

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