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Does successful smoking cessation reduce anxious arousal among treatment-seeking smokers?

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ABSTRACT

Introduction: There is limited work that has examined the effect of quitting smoking on anxious arousal, an underlying dimension of anxiety symptoms and psychopathology.
Method: Smokers ($n = 185$, 54.1% female) enrolled in a smoking cessation treatment trial were monitored post-cessation in terms of abstinence status (biochemically verified; at Weeks 1, 2, and Month 1 post-quit) and severity of panic-relevant symptoms (self-reported; at Month 1 and 3 post-quit). Structural equation models were conducted, adjusting for participant sex, age, treatment condition, and pre-cessation nicotine dependence, presence of depressive/anxiety disorders, anxious arousal, and anxiety sensitivity.
Results: After adjusting for covariates, participants who remained abstinent for one month ($n = 80$; 43.2%) relative to those who did not ($n = 105$; 56.8%) demonstrated significant reductions in anxious arousal at Month 1 ($\beta = -.26$, $p = .04$) and Month 3 post-quit ($\beta = -.36$, $p = .006$); abstinence status had a non-significant effect on anxious arousal severity at Month 3 after controlling for Month 1 anxious arousal ($\beta = -.18$, $p = .09$).
Discussion: Findings align with theoretical models of smoking-anxiety interplay and suggest that smoking cessation can result in reductions in anxious arousal.

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1. Introduction

Smokers with comorbid psychiatric disorders show less success in being able to quit smoking, which contribute to the stagnation of smoking cessation rates well-documented over recent years (Goodwin, Zvolensky, Keyes, & Hasin, 2012; Ziedonis et al., 2008). Anxiety symptoms and disorders, in particular, serve to maintain and even promote tobacco use and dependence (Morissette, Tull, Gulliver, Kamholz, & Zimering, 2007) and impair quit success (Piper, Cook, Schlam, Jorenby, & Baker, 2011). Indeed, there are bidirectional associations between smoking and anxiety symptoms/disorders (Amering et al., 1999; Cosci, Knuts, Abrams, Griez, & Schruers, 2010; Zvolensky & Bernstein, 2005; Zvolensky, Feldner, Leen-Feldner, & McLeish, 2005). For example, the initiation of cigarette smoking typically precedes the initial onset of anxiety psychopathology (e.g., Bernstein, Zvolensky, Schmidt, & Sachs-Ericcson, 2007) and smoking increases the later risk for anxiety psychopathology (Breslau, Novak, & Kessler, 2004; Isensee, Wittchen, Stein, Hofler, & Lieb, 2003; Johnson et al., 2000; Jamal, Does, Penninx, & Cuijpers, 2011). Additionally, successful smoking cessation is associated with reductions in anxiety symptoms and decreased likelihood of anxiety disorders among those with pre-existing disorders (Cavazos-Rehg et al., 2014; McDermott, Marteau, Hollands, Hankins, & Aveyard, 2013; Shahab, Andrew, & West, 2014; Taylor et al., 2014).

Given the phenotypic heterogeneity in the expression of symptoms across anxiety disorders (Watson, 2005), transdiagnostic models of emotional disorders (anxiety and depressive conditions) have suggested underlying dimensional constructs may serve to explain between-individual variability in symptom presentation (i.e., tripartite model; Clark & Watson, 1991; Watson & Clark et al., 1995; Watson & Weber et al., 1995). Anxious arousal, reflecting the extent to which one experiences somatic arousal and tension (e.g., shortness of breath, dizziness, lightheadedness, trembling, shacking), is a core, cross-cutting feature of many anxiety disorders (Watson & Clark et al., 1995). Anxious arousal also is related to

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smoking (see review; Ameringer & Leventhal, 2010). For example, anxious arousal is associated with higher levels of nicotine dependence (Zvolensky, Stewart, Vujanovic, Gavric, & Steeves, 2009) and a history of a greater number of unsuccessful smoking cessation attempts (Zvolensky, Johnson, Leyro, Hogan, & Tursi, 2009). Further, poorer perceptions of physical health are associated with higher levels of anxious arousal among daily smokers (McLeish, Zvolensky, Bonn-Miller, & Bernstein, 2006). Evidence also suggests that smoking heaviness and anxious arousal may be bi-directionally linked by affect-regulatory smoking motives (Johnson, Stewart, Zvolensky, & Steeves, 2009). Moreover, anxious arousal is associated with experiencing greater increases in abstinence-induced depression and fatigue (Leventhal, Ameringer, Osborn, Zvolensky, & Langdon, 2013), which was not seen in other tripartite aspects of anxiety and depression (e.g., anhedonic depression). Yet, there is no empirical data on the nature of anxious arousal after a smoking cessation attempt. This gap in knowledge is clinically important to address in order to better understand anxiety phenomena broadly during the process of quitting, which may have cross-cutting implications across specific forms of anxiety psychopathology. That is, based on complex heterogeneity across and within different anxiety disorders, examining anxious arousal, a more parsimonious transdiagnostic construct, would provide more precise and informative information regarding the patterning of (anxiety) symptoms after a quit attempt.

The majority of empirical evidence suggests that smoking cessation yields on reductions of anxiety symptoms (Becoña, Vázquez, & Míguez, 2002; Dawkins, Powell, Pickering, Powell, & West, 2009; McDermott et al., 2013; Solomon et al., 2006), although there is large heterogeneity across studies (see meta-analysis; Taylor et al., 2014). It is possible that variability in study findings is, in part, a function of motivation to quit smoking (Taylor et al., 2014), but also may be due to broad types of measurement of anxiety symptoms utilized in existing studies that do not permit an examination of 'pure' anxious arousal (i.e, they cannot distinguish between anxiety symptoms that do and do not overlap with depression; Taylor et al., 2014). For this reason, it is important to examine the nature of anxious arousal as a function of smoking cessation. Reductions in anxious arousal after smoking cessation would be expected based on the understanding that nicotine has anxiogenic properties over time and can actually promote greater levels of anxiety over time (Kassel, Stroud, & Paronis, 2003; Leventhal & Zvolensky, 2015; Zvolensky & Bernstein, 2005), despite the (perceived or actual) affect regulation and modulation properties of smoking (nicotine) in the immediate context of use—likely due to narrowing of attentional focus to most immediate stimuli in environment and away from subjective distressing thoughts, feelings or sensations (Kassel & Unrod, 2000). Thus, although abstinence from smoking may induce symptoms in the short-term (Vessichhio, Termine, & George, 2002; Zvolensky, Lejuez, Kahler, & Brown, 2004), perhaps due to heightened cognitive–affective reactivity to interoceptive perturbation (Zvolensky & Bernstein, 2005), quitting smoking should theoretically lessen the severity of anxious arousal during periods of sustained abstinence. To address this question, the present study tested the hypothesis that abstainers, relative to non-abstainers, would report less severe anxious arousal at one- and three-months post cessation. These effects were expected to be significant after adjusting for participant gender, age, study treatment condition, and severity of pre-quit levels of nicotine dependence and anxious arousal. The effects were also expected to be significant after adjusting for the presence of depressive/anxiety disorders pre-cessation. Additionally, unlike past work (Taylor et al., 2014), to strengthen the test of these effects, pre-quit levels of anxiety sensitivity (i.e., tendency to misinterpret the meaning of anxious arousal or fear of anxious arousal) was adjusted for in analyses.

2. Material and methods

2.1. Participants

Participants ($n = 185$; 54.1% female; $M_{age} = 39.41$, SD = 13.76) were adult daily smokers recruited as part of a smoking cessation and panic disorder prevention trial (clinicaltrials.gov #NCT01753141). Eligibility criteria for the parent study included: smoking ≥8 cigarettes per day for at least the past year, and motivation to quit rated at least 5 or higher on a 10-point scale. Exclusionary criteria included current use of smoking cessation products or treatment, regular use of other tobacco products, unstable psychotropic medication (had to be stable ≥3 months), history of panic disorder (defined by the DSM-IV-TR), past-month suicidality, a history of psychotic-spectrum disorders, current pregnancy or nursing and inability to provide informed consent. Participants were included in the current study if smoking cessation data were available for at least two of three post-quit follow-up appointments (i.e., Week 1, Week 2, and/or Month 1).

The majority of participants identified race as white (85.4%) and completed at least some college (80.6%). The average daily smoking rate was of this sample was 16.8 (SD = 8.39) cigarettes per day and on average participants reported daily smoking for 21.0 years (SD = 13.86). Moderate levels of nicotine dependence were reported among the sample per the Fagerström for Nicotine Dependence ($M = 5.1$, SD = 2.27) and baseline expired carbon monoxide (CO) averaged 20.6 ppm (SD = 11.99). Current (past-month) Axis I primary diagnoses were as follows: depressive/mood disorder (7.0%), alcohol use disorder (3.8%), substance use disorder (2.7%), social anxiety disorder (9.2%), specific phobia (3.8%), obsessive-compulsive disorder (1.6%), posttraumatic stress disorder (2.2%), and generalized anxiety disorder (5.4%).

2.2. Procedure

Participants were recruited through community-based advertisements at two treatment sites (University of Vermont, Burlington VT and Florida State University, Tallahassee, FL). Potentially-eligible participants were scheduled for a baseline assessment during which participants were assessed using a structured clinical diagnostic assessment, provided a CO analysis to verify smoking status and completed a computerized battery of self-report questionnaires. Eligible participants were randomly assigned to one of two smoking cessation treatment programs and scheduled for treatment initiation approximately 1–2 weeks after the baseline assessment. Smoking cessation treatment consisted of either (1) a standard smoking cessation program (Fiore et al., 2008), or (2) anxiety-focused smoking cessation treatment (Zvolensky, Yartz, Gregor, Gonzalez, & Bernstein, 2008), both included use of nicotine replacement therapy via the transdermal nicotine patch, which was initiated at treatment session four (quit day). Treatment consisted of four 60-min weekly individual sessions conducted by a trained doctoral-level graduate student. Quit day occurred during the last treatment session (Session 4). All treatment was supervised by study authors (MJZ and NBS) and checked for treatment fidelity by independent reviewers. Follow-up assessments occurred at Week 1, Week 2, Month 1, Month 3 post-quit attempt. All participants provided informed consent prior to participation and the study protocol was approved by the Institutional Review Board at both institutions, where the study was conducted.

2.3. Measures

2.3.1. Descriptive measures

The *Smoking History Questionnaire* (SHQ; Brown, Lejuez, Kahler, & Strong, 2002) is a self-report questionnaire used to assess smok-

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