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The indirect effect of panic disorder on smoking cognitions via difficulties in emotion regulation

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Abstract

Panic disorder (PD) and cigarette smoking are highly comorbid and associated with worse panic and smoking outcomes. Smoking may become an overlearned automatized response to relieve panic-like withdrawal distress, leading to corresponding smoking cognitions, which contribute to its reinforcing properties and difficultly abstaining. Difficulties in emotion regulation (ER) may underlie this relation such that in the absence of adaptive emotion regulatory strategies, smokers with PD may more readily rely upon smoking to manage affective distress. In the current study, the indirect relation between PD status and smoking cognitions through ER difficulties was examined among daily smokers ($N = 74$). We found evidence for an indirect relation between PD status and negative affect, addictive and habitual smoking motives, and anticipating smoking will result in negative reinforcement and personal harm, through self-reported difficulties with ER. Our findings are aligned with theoretical models on anxiety and smoking, and suggest that reports of greater smoking cognitions may be due to ER difficulties.

1. Introduction

Cigarette smoking remains the leading preventable cause of disability, disease (e.g., cardiovascular disease), and death in the United States (US Department of Health and Human Services, 2014). Despite a decrease in smoking prevalence over the past several decades, prevalence among individuals with mental illness is disproportionately high as compared to the general US adult population (McClave, McKnight-Eily, Davis, & Dube, 2010). Smoking prevalence among individuals with a diagnosis of panic disorder (PD), specifically, is disproportionately high (e.g., 35–60%; Kalman, Morissette, & George, 2005; Lasser et al., 2000) as compared to the general US adult population (e.g., 13.5%; Glasheen, Hedden, Forman-Hoffman, & Colpe, 2014; McClave et al., 2010). This observation is of even greater significance given the prevalence of PD and panic psychopathology (e.g., unexpected panic attacks), in the general population. Previous research has indicated that smoking increases the risk of developing and maintaining panic attacks (Bakhshaie et al., n.d.) and panic disorder (PD) (Breslau and Klein, 1999). Furthermore, PD and smoking comorbidity is associated with worse smoking (e.g., less self-efficacy in quitting, Zvolensky et al., 2005) and panic relevant interference (e.g., greater severity in panic symptoms, Zvolensky, Schmidt, & McCreary, 2003).

Whereas smoking appears to precipitate the onset of panic attacks and PD, the maintenance of this comorbidity, and subsequent difficulties abstaining appears to be more complex. One theory is that smokers with PD may conflate affective distress associated with panic and nicotine withdrawal, and via repeated learning trials, learn that smoking is effective in relieving both sets of symptoms (Zvolensky and Bernstein, 2005; Zvolensky and Schmidt, 2003; Zvolensky et al., 2005). Over time, these individuals may develop problematic reasons for smoking (i.e., smoking motives, Ikard, Green, & Horn, 1969) and expectancies about the effects of smoking (i.e., smoking expectancies, Brandon and Baker, 1991), thereby increasing the risk for continued smoking and difficulty quitting (e.g., heavy smoking, relapse, Piper et al., 2004).

Research in this domain has found that, as compared to smokers without PD, smokers with PD are more motivated to smoke in order to relieve negative affect, likely to report anxiety symptoms as cessation barrier, and report less self-efficacy in staying abstinent under emotionally distressing states (Zvolensky et al., 2005). Also, in daily smokers, the combination of PD and more intense nicotine withdrawal

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symptoms, compared to either individuals with PD with less withdrawal symptoms, smokers without PD or nonsmokers with PD, is also associated with greater subjective and physiological reactivity and slower recovery from a biological challenge (Leyro and Zvolensky, 2013, 2014; Zvolensky et al., 2004). Among individuals with PD, there is a significant association between vigilance to bodily sensations and health related concerns (Schmidt, Joiner Jr, Staab, & Williams, 2003; Starcevic et al., 2009), which may in part explain why smokers with PD are more sensitive to nicotine withdrawal symptoms (Jarvik et al., 2000; Shadel, Niaura, Brown, Hutchison, & Abrams, 2001). In smokers with PD, this hypervigilance may increase the expectation that their smoking is negatively affecting health. Whereas concern about the negative consequences of smoking may promote cessation (Copeland, Brandon, & Quinn, 1995; Rose, Chassin, Presson, & Sherman, 1996), generally, among smokers with PD who are characterized by behavioral avoidance and difficulty managing negative affect, it may paradoxically contribute to reliance on smoking. Together smokers with PD are characterized by a problematic set of beliefs and behaviors that likely impede their ability to successfully refrain from smoking. However, little research has examined malleable affective vulnerabilities that may indirectly play a role in the relation between PD and smoking cognitions (Piper, Cook, Schlam, Jorenby, & Baker, 2011; Zvolensky et al., 2004).

Emotion regulation (ER), a multidimensional construct that includes awareness and acceptance of one's emotions and the ability to modulate emotional *responding* in accord with one's personal goals (Gratz and Roemer, 2004), is a transdiagnostic vulnerability that has not been well examined in the context of smoking. Difficulty in ER may characterize smokers with PD who perceive themselves as being unable to adaptively respond to and cope with distress, thereby exacerbating affective and behavioral responding to nicotine withdrawal. Through associative learning, smokers with PD may be more likely to smoke to regulate negative affect in an automatized manner (Zvolensky and Bernstein, 2005). Anticipation of the immediate anxiolytic effect of smoking (Leventhal and Zvolensky, 2015) may hinder these individuals from considering alternate, more adaptive, regulatory strategies, which, in turn, may promote the belief that they are ineffective in regulating negative emotional states. Indeed, research has found that among smokers, lower ER is related to greater motivation to smoke for stimulation, habitual, sensorimotor (Gonzalez, Zvolensky, Vujanovic, Leyro, & Marshall, 2008; Short, Oglesby, Raines, Zvolensky, & Schmidt, 2015), addictive, and negative affect reduction smoking motives (Short et al., 2015), which may develop over the course of smoking. Similar findings have been found for other types of substance use, including alcohol (Paulus et al., 2017). Moreover, individuals with PD, compared to those without PD, report greater ER difficulties in general (Tull, Stipelman, Salters-Pedneault, & Gratz, 2009). One investigation found that anxiety sensitivity, a well-known risk factor of PD, is associated with greater negative affect smoking motives, outcome expectances, and perceived cessation barriers, through its relation to difficulty in ER, among treatment-seeking daily smokers (Johnson, Farris, Schmidt, & Zvolensky, 2012). Other work has found similar effects for alcohol (Paulus et al., 2016). In addition, whereas previous research on smokers has found that ER may be modified via brief cognitive interventions (e.g., Szasz, Szentagotai, & Hofmann, 2012), its role has not been examined in a clinical population or examined as a treatment adjunct.

The current study extends previous research on ER difficulties to a highly prevalent clinical sample of smokers, to better understand how interventions targeting this transdiagnostic vulnerability may be packaged into targeted smoking cessation interventions. Specifically, the current study examined the indirect relation between PD status and smoking cognitions (i.e., motives and outcome expectancies, see Fig. 1) through ER difficulties. It was hypothesized that diagnosis of PD (i.e., PD status) will display indirect effect on criterion variables (i.e., smoking cognitions) via difficulty in ER. These predictions are expected to be above and beyond the variance accounted for by age, sex, and nicotine dependence.

2. Method

2.1. Participants

The current study is a secondary data analysis of non-treatment seeking daily smokers who were recruited to participate in a study investigating the interaction of PD and nicotine withdrawal in the prediction of fearful responding to bodily sensations under carbon dioxide (CO_2)-enriched air biological challenge (Leyro and Zvolensky, 2013). The present analyses are novel and have not been previously published. Inclusion criteria for the parent study were: (a) being a daily smoker for at least the past year (cigarettes per day ≥ 7[1]); (b) having not decreased the number of cigarettes smoked per day by more than half in the past 6 months; (c) being 18 to 65 years old; and (d) reporting a willingness to abstain from smoking for a 12-hr period. Exclusion criteria of the parent study were: (a) having a current medical condition that contraindicated CO_2 administration (cardiovascular, endocrine, pulmonary, respiratory [including severe asthma], or gastrointestinal illness); (b) meeting criteria for a past diagnosis of PD, (c) limited mental competency (not oriented to person, place, or time) and the inability to give informed, voluntary, written consent to participate; (d) pregnancy or the possibility of being pregnant (by self-report); (e) current use of nicotine replacement therapy; (f) current or past history of psychotic-spectrum symptoms or disorders; (g) current substance dependence; (h) prior experience with CO_2 challenge; (i) suicidality; and (j) any current use of psychotropic medication which could impact the effectiveness of the laboratory challenge (e.g., regular use of benzodiazepines and beta-blockers, which directly affects the autonomic nervous system, and hence, has impact on their response to the laboratory stressor[2]).

2.2. Measures

2.2.1. Structured Clinical Interview-Non-Patient Version for DSM-IV, non-patient edition (SCID-IV-N/P; First, Spitzer, Gibbon, & Williams, 1994)

The SCID-IV-N/P was administered to determine diagnostic inclusion and exclusion. Reliability has been proved to be adequate (First et al., 1994). The principal investigator of the parent study or trained senior graduate students administered the SCID-N/P. Interviews were audio-recorded and 20% were cross-checked by senior level graduate clinical psychology students, indicating 98% agreement. Regarding PD diagnosis, there was no disagreement.

2.2.2. Smoking History Questionnaire (SHQ; Brown, Lejuez, Kahler, & Strong, 2002)

The SHQ is a well-established questionnaire that assesses smoking history and pattern of use. The items pertain to smoking rate, age of onset of initiation, and years of regular smoking. In the current study, the SHQ was used as a descriptive measure of smoking history.

2.2.3. Fagerström Test for Cigarette Dependence (FTCD; Fagerström, 2012; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991)

The FTCD is a 6-item self-report scale assessing continuous levels of tobacco dependence. The FTCD is a revised tool from the Fagerström Tolerance Questionnaire (FTQ; (Fagerström, 1978), which original name was Fagerström Test for Cigarette Dependence (Heatherton

[1] The inclusion criteria in the parent study for cigarettes per day was equal to or > 10 cig/day. However, in order to increase recruitment, the number was decreased to 7 cig/day. In this sample, 2.7% of the sample ($n = 2$) reported < 10 cig/day.

[2] Participants prescribed benzodiazepines or beta-blockers were included if they reported taking them infrequently (e.g., < 1/month) and expressed a willingness to abstain from use during the study.