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The Indirect Effect of Panic Disorder on Smoking Cognitions via Difficulties in Emotion Regulation

Min-Jeong Yang^a, Michael J. Zvolensky^{b,c}, and Teresa M. Leyro^{a,*}

^aRutgers, The State University of New Jersey, Department of Psychology, Tillett Hall, 53 Avenue E., Piscataway, NJ 08854, USA

^bUniversity of Houston, Department of Psychology, 126 Fred J. Heyne Building, Suite 104, Houston, TX 77204, USA

^cThe University of Texas MD Anderson Cancer Center, Department of Behavioral Science, 1155 Pressler Street, Houston, TX 77030, USA

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 difficulty 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.

Keywords

emotion regulation; panic disorder; smoking; smoking motive; smoking outcome expectancy; anxiety

*Corresponding author. Phone: +1 848 445 2090, Fax: +1 732 445 0036, teresa.leyro@rutgers.edu.

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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, McKnight-Eily, Davis, & Dube, 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, Zvolensky, & Goodwin, In press) and panic disorder (PD) (Breslau & 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 & Bernstein, 2005; Zvolensky & 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 & 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 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 & 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 & 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 & Bernstein, 2005). Anticipation of the immediate anxiolytic effect of smoking (Leventhal & 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., In press). 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 expectancies, 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 Figure 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 (CO₂)-enriched air biological challenge (Leyro & 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 CO₂ 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 CO₂ 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 stressor²).

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-

¹The inclusion criteria in the parent study for cigarettes per day was equal to or more than 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 less than 10 cig/day.

²Participants 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.

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 et al., 1991). Despite low internal consistency ($\alpha = .61$; Heatherton et al., 1991), its test-retest reliability is high (Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994) and it is positively correlated with key smoking variables such as saliva cotinine (Heatherton et al., 1991; Payne, Smith, McCracken, McSherry, & Antony, 1994). In the current investigation, the FTCD was employed to index nicotine dependence (Cronbach's $\alpha = .44$ in the present sample).

2.2.4. Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004).—The DERS is a 36-item self-report scale assessing emotion dysregulation on a five-point Likert-style scale (1 = *almost never* to 5 = *almost always*). There are six subscales: non-acceptance (e.g., “when I’m upset, I feel guilty for feeling that way.”), goals (e.g., “when I’m upset, I have difficulty getting work done.”), impulse (e.g., “when I’m upset I have difficulty controlling my behaviors.”), awareness (e.g., “I pay attention to how I feel.”), strategies (e.g., “when I’m upset any emotions feel overwhelming.”), and lack of clarity (e.g., “I have no idea how I am feeling.”). In the current study, the total score of DERS was used as an index of general difficulties in emotion regulation such that higher scores suggest greater emotion dysregulation (Cronbach's $\alpha = .95$ in the present sample).

2.2.5. Reason for Smoking (RFS; Ikard et al., 1969).—The RFS is a validated 23-item self-report scale used to assess smoking motives on a five-point Likert-style scale (1 = *never* to 5 = *always*) (Shiffman, 1993). In the current study, the Habitual (RFS-HA; e.g., “I’ve found a cigarette in my mouth and didn’t remember putting it there”), Addictive (RFS-AD; e.g., “Between cigarettes, I get a craving only a cigarette can satisfy”), and Negative Affect Reduction (RFS-NA; e.g., “When I feel uncomfortable or upset about something, I light up a cigarette”) subscales were utilized to measure theoretically relevant smoking motives (Cronbach's α for total scale and each subscale of interest was as follows; RFS = .90, RFS-HA = .66, RFS-AD = .80, and RFS-NA = .87 in the present sample).

2.2.6. Smoking Consequences Questionnaire (SCQ; Brandon & Baker, 1991).—The SCQ is a validated 50-item self-report scale assessing smoking expectancies on a 10-point Likert-style scale (0 = *completely unlikely* to 9 = *completely likely*) (Brandon & Baker, 1991; Buckley et al., 2005; Downey & Kilbey, 1995). In the current study, the Negative Consequences (SCQ-NC; e.g., “the more I smoke, the more I risk my health”) and Negative Reinforcement (SCQ-NR; e.g., “smoking helps me calm down when I feel nervous”) subscales of the SCQ were utilized to measure theoretically relevant outcome expectancies for smoking (Cronbach α for total scale and each subscale of interest was as follows; SCQ = .94, SCQ-NC = .88, and SCQ-NR = .94 in the present sample).

2.3. Procedure

Data for the current investigation was gathered during the baseline session of the parent study. During this visit, participants were assessed for current (past 12 month) Axis I psychopathology using the SCID-IV-N/P (First et al., 1994)), to assess study eligibility and determine PD status. Smoking history and cigarette dependence were measured with the

SHQ and FTCD, respectively. As part of the baseline assessment, a self-report battery was completed including RFS, SCQ, and DERS. For additional details of the parent study, see Leyro and Zvolensky (2013).

2.4. Data Analysis

All analyses were conducted in PASW Statistics 22.0 (IBM SPSS Statistics). First, zero order correlations among predictor (current PD status), proposed mediator (DERS total), criterion variables (RFS-HA, RFS-AD, RFS-NA, SCQ-NC, and SCQ-NR), and covariates (age, sex, and FTCD) were conducted. Next, a series of mediation models were conducted to examine the indirect effect of PD status on the outcome variables via DERS. Five mediation models were tested with each of five criterion variables. Age, sex, and cigarette dependence (FTCD) were included in the models as covariates, due to previously observed relations with outcomes of interest and theoretical relevance. The mediation analyses were carried out utilizing PROCESS, a conditional process modeling program that utilizes an ordinary least squares based path analytical framework to test for both direct (path c') and indirect effects ($a*b$) (Hayes, 2013). Based on Kenny and Judd (2013) and Hayes' (2009) discussion on statistical issues in Baron and Kenny's (1986) causal steps approach, the conditional process modeling was chosen in order to quantify indirect effect ($a*b$) without merely relying upon the significance of each path (i.e., path a , b , c , and c'). Here, path c refers to total effect of the predictor on the outcome variable without accounting for the mediator, while path c' refers to the direct effect, controlling for the mediator (Hayes & Rockwood, 2016). Regarding the determination of statistical significance of indirect and direct effect, we utilized 95-percentile bias-corrected confidence intervals (CI), estimated with bootstrap analyses of 10,000 samples (as recommended by (Hayes, 2009; Hayes & Scharkow, 2013; Preacher & Hayes, 2004, 2008). The bias-corrected bootstrap CI has been reported to be a relatively better index of statistical significance for indirect effect (i.e., highest power and the best Type I error control, (Hayes, 2009)) as compared to other methods such as test of joint significance or Monte Carlo CI (Hayes & Scharkow, 2013).

3. Results

3.1. Participants

Participants included 74 daily smokers (52.7% male, $M_{\text{age}}=29.9$, $SD=12.6$) who reported smoking on average 19.9 cigarettes/day ($SD=7.8$), for an average of 12.7 years ($SD=11.8$). Sample characteristics are shown in Table 1. The sample met for low to moderate levels of cigarette dependence ($M=3.9$, $SD=1.7$; Fagerström, 2012). Approximately 39.2% ($n=29$) met DSM-IV criteria for current PD with and without agoraphobia. An additional 20.3% ($n=15$) met criteria for another current mood or anxiety disorder, and 40.5% ($n=30$) were diagnosis free. Of the participants, 93.2% were identified as Caucasian, 2.7% African American, 1.4% Asian, and 2.7% "other." There was no significant difference between PD and non-PD groups in study covariates which included age ($t(72) = -.36$, $p = .72$), gender ($X^2 = .67$, $p = .41$), and levels of cigarette dependence ($t(71) = .31$, $p = .76$).

3.2. Zero-order correlations

PD status was significantly correlated to difficulty in ER (DERS total) and negative personal consequence smoking outcome expectancies (SCQ-NC) (all p 's < .01), but not other smoking cognitions. The mediator, DERS total, showed significant positive associations with each criterion variable (all p 's < .01); correlations were moderate in strength. Examination of theoretically relevant covariates revealed female gender was significantly correlated with addictive reasons for smoking (p < .01). Cigarette dependence was positively associated with difficulty in ER (p < .05), habitual, addictive, and negative affect reduction reasons for smoking (all p 's < .01), and negative reinforcement smoking outcome expectancies (p < .05). However, age did not show any significant association with difficulty in ER or smoking cognitions.

3.3. Mediation analyses

Five path models were planned for the examination of the indirect effect of PD status on each criterion variable, through difficulty in emotion regulation. The regression coefficients for paths a, b, c, and c' are shown in Table 3. The paths are for each of the five models. Table 3 also presents the estimates of the indirect effects, which were the paths tested for mediation.

In the first path model examining habitual motives (Y1=RFS-HA), both the total effect model ($R^2_{y1,x} = .227$, $df = 4, 68$, $F = 5.218$, $p < .01$; path c) and the full model with the mediator ($R^2_{y1,x} = .316$, $df = 5, 67$, $F = 5.690$, $p < .0001$) were significant. In the full model, higher nicotine dependence was significantly associated with higher scores on the RFS-HA ($b = .153$, $t = 3.045$, $p < .01$). Both the total effect (path c) of PD status and the direct effect (path c') of PD status on RFS-HA, after controlling for the mediator, were nonsignificant. Regarding the test of the indirect effect, PD status was related to greater habitual smoking reason through greater levels of difficulty in emotion regulation (effect a*b).

In the second path model examining addictive motives (Y2=RFS-AD), both the total effect model ($R^2_{y1,x} = .307$, $df = 4, 68$, $F = 10.980$, $p < .0001$) and the full model with the mediator ($R^2_{y1,x} = .405$, $df = 5, 67$, $F = 14.066$, $p < .0001$) were significant. In the full model, gender ($b = .516$, $t = 3.159$, $p < .01$) and nicotine dependence ($b = .156$, $t = 2.700$, $p < .01$) were significantly related to higher scores on the RFSAD. Both the total effect of PD status and the direct effect of PD status on RFS-AD, after controlling for the mediator, were nonsignificant. Regarding the test of the indirect effect, PD status was associated with greater addictive smoking reason indirectly through greater levels of difficulty in emotion regulation (effect a*b).

With regard to negative affect reduction motives, (Y3=RFS-NA), both the total effects model ($R^2_{y1,x} = .213$, $df = 4, 68$, $F = 6.221$, $p < .0001$) and the full model with the mediator ($R^2_{y1,x} = .290$, $df = 5, 67$, $F = 6.028$, $p < .0001$) were significant. In the full model, gender ($b = .374$, $t = 2.067$, $p < .05$) and nicotine dependence ($b = .146$, $t = 2.727$, $p < .01$) were significantly associated with higher scores on the RFS-NA. The direct effect of PD status on RFS-NA, after controlling for the mediator, was nonsignificant. Regarding the test of the indirect

effect, PD status was related to greater negative affect reduction smoking reason indirectly through greater levels of difficulty in emotion regulation (effect $a*b$).

With regard to expecting smoking to result in negative personal consequences ($Y4=SCQ-NC$), both the total effects model ($R^2_{y1,x} = .177$, $df = 4, 68$, $F = 2.996$, $p < .01$) and the full model with the mediator ($R^2_{y1,x} = .250$, $df = 5, 67$, $F = 3.028$, $p < .05$) were significant. In the full model, no covariates were significant. After controlling the mediator, the direct effect of PD status on SCQ-NC was nonsignificant. Regarding the test of the indirect effect, PD status was associated with greater negative consequence smoking expectancy indirectly through greater levels of difficulty in emotion regulation (effect $a*b$).

Lastly, with regarding to negative reinforcement outcome expectancies ($Y5=SCQ-NR$), both the total effects model ($R^2_{y1,x} = .176$, $df = 4, 68$, $F = 6.040$, $p < .0001$) and the full model with the mediator ($R^2_{y1,x} = .278$, $df = 5, 67$, $F = 5.510$, $p < .0001$) were significant. In the full model, nicotine dependence ($b = .270$, $t = 2.118$, $p < .05$) was significantly associated with higher scores on the SCQ-NR. After controlling the mediator, the direct effect of PD status on SCQ-NR was nonsignificant. Regarding the test of the indirect effect, PD status was related to greater negative reinforcement smoking expectancy indirectly through greater levels of difficulty in emotion regulation (effect $a*b$).

4. Discussion

Smokers with panic psychopathology are vulnerable to a set of beliefs and expectancies about smoking that may place them at greater risk for continued use (Johnson, Farris, Schmidt, Smits, & Zvolensky, 2013; Zvolensky et al., 2005). In order to identify targetable mechanisms that may explain these relations, the current study sought to examine the indirect effect of PD status on smoking cognitions via ER difficulties in a group of smokers with and without a current diagnosis of PD. Consistent with study hypotheses, as compared to smokers without a PD diagnosis, smokers with PD reported greater habitual, addictive, and negative affect reduction smoking motives as well as negative personal consequences and negative reinforcement smoking outcome expectancies, through difficulties in ER.

Our finding that PD is associated with habitual, addictive, and negative affect reduction smoking motives through ER difficulties extends a previously observed indirect effect of anxiety sensitivity on negative affect reduction smoking motives via ER difficulties (Johnson et al., 2012), to a clinical population of smokers. Moreover, while there was significant total effect in the relation between PD status, and negative affect reduction smoking motives, no total effect was observed with habitual and addictive smoking motives. These findings are consistent with previously observed nonsignificant associations between PD status and habitual and addictive smoking motives among smokers (Zvolensky et al., 2005). However, in the current study, this significant relation was observed indirectly through ER difficulties. This novel finding suggests that smokers with PD who perceive themselves experiencing ER difficulties may have elevated habitual and addictive smoking motives, which highlights the role of ER in problematic motives for use in smokers with PD. Collectively, the current findings suggest that ER may be an important construct in the maintenance of smoking motives. Specific cognitive-behavioral intervention strategies targeting ER in smokers with

PD, such as reappraisal (Szasz et al., 2012), may be a means by which to reduce these motives, and subsequently reduce smoking in the context of heightened craving, automatized use, and affective distress.

Our finding that ER difficulties explains the relation between PD status in smokers and negative reinforcement and negative personal consequence smoking outcome expectancies extends previously observed relations among smokers high in anxiety sensitivity (Johnson et al., 2012). Individuals with PD who report greater hypervigilance to bodily sensations, fear of anxious arousal (Schmidt et al., 2003), and worry about health (Starcevic et al., 2009), as compared to those who report fewer concerns, may be more sensitive to nicotine withdrawal symptoms (Jarvik et al., 2000; Shadel et al., 2001). In the context of poorer regulation strategies, and perhaps exacerbation of symptoms, their awareness of their smoking status may increase expectations about negative health consequences (Zvolensky & Bernstein, 2005; Zvolensky & Schmidt, 2003). Research indicates smokers who expect smoking to be harmful to their health are more motivated to plan a quit attempt (Copeland et al., 1995; Rose et al., 1996). However, observed elevations in negative reinforcement smoking motives and expectancies through ER difficulties in this group may paradoxically confound the ability to successfully abstain. For example, reliance on smoking to relieve distress may overpower or obstruct motivation to quit due to elevated health concerns (Wetter et al., 1994). Instead, it is possible that the combination of negative reinforcement related smoking cognitions and expectancies of negative consequences of smoking may lead to additional affective distress, thereby providing further opportunities to promote negative reinforcement smoking (Zvolensky & Schmidt, 2003).

Several limitations to the current investigation should be noted. First, our sample consisted of community-recruited, non-treatment-seeking daily cigarette smokers who were primarily white and low to moderate levels of cigarette dependence. Future studies would benefit from replicating our findings in a more ethnically/racially diverse sample of smokers with higher levels of cigarette dependence. Second, due to the limited sample size in the current study, our findings may not be generalizable to all smokers with PD; hence, replication in a larger sample is required. Regardless of PD diagnosis, the PD group evidenced significantly more current diagnoses of psychopathology than the non-PD group. This may in part be explained by epidemiological findings suggesting that comorbid conditions are found in 83.1% to 100% of individuals with a diagnosis of PD with or without agoraphobia (Kessler et al., 2006), which suggests that they may be more psychiatrically unhealthy. Whereas our flexible inclusion criteria may be viewed as a strength, increasing generalizability to current smokers, employment of more rigid inclusion/exclusion criteria may strengthen confidence in the specificity of our findings to smokers with PD. In addition, given emerging work suggesting that ER difficulties are a transdiagnostic risk factor for psychopathology (e.g., Aldao, Nolen-Hoeksema, & Schweizer, 2010), additional research may benefit from examining whether ER difficulties mediate the relation between psychopathology, more broadly, and smoking motives. Fourth, longitudinal investigations are necessary to examine the prospective and transactive relations between ER difficulties, panic psychopathology, and smoking beliefs and expectancies. Lastly, this study relied on a self-report index of general ER strategies. Future studies would benefit from a multi-method ER assessment and clarification of how specific facets of ER relate to various smoking processes.

The current study is the first study to examine the indirect effect of PD status on smoking cognitions via ER difficulties. Collectively, our findings suggest that emotion dysregulation may help explain how panic psychopathology results in cognitive-based smoking processes. Specifically, among smokers with PD, reports of greater motives to smoke and expectancies about smoking may be due to difficulties in ER, which may impede their motivation to plan a quit, confidence, and success (Zvolensky et al., 2005). Therefore, our findings may be used to inform later research addressing the role of ER difficulties, for example, prior to individual's quit day, to promote a more successful quit attempt.

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Highlights

- 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.
- Difficulties in emotion regulation (ER) may underlie the relation between PD and smoking cognitions in daily smokers.
- We found the indirect effect of PD on negative affect, addictive and habitual motives, and expectancies that smoking will result in negative reinforcement and personal harm through difficulties in ER.
- Developing ER skills in smokers with PD may reduce smoking cognitions and has implications for reducing smoking in this group.

Figure 1.
Study Model

Table 1.

Descriptive statistics of sample characteristics

Descriptive summary	Total N=74	PD n=29	Non-PD n=45	Test for Group Difference
Race ³ n (%)				
White	69 (93.2%)	29 (100.0%)	40 (88.9%)	$\chi^2 = 3.46, p = .33$
Black	2 (2.7%)	0 (0%)	2 (4.4%)	
Asian	1 (1.4%)	0 (0%)	1 (2.2%)	
Other	2 (2.7%)	0 (0%)	2 (4.4%)	
Marital status n (%)				
Never married	51 (68.9%)	16 (55.2%)	35 (77.8%)	$\chi^2 = 7.99, p = .09$
Married/cohabitating	9 (12.2%)	6 (20.7%)	3 (6.7%)	
Divorced/annulled	8 (10.8%)	4 (13.8%)	4 (8.9%)	
Separated	4 (5.4%)	3 (10.3%)	1 (2.2%)	
Widowed	2 (2.7%)	0 (0%)	2 (4.4%)	
Education n (%) ⁴				
At least high school	31 (41.9%)	7 (24.1%)	24 (53.3%)	$\chi^2 = 5.67, p = .02$
At least part college	42 (56.8%)	21 (72.4%)	21 (46.7%)	
Smoking history M (SD)				
Cigarettes per day	19.9 (7.8)	18.2 (6.3)	21.0 (8.5)	$t(72) = 1.50, p = .14$
Age of first use	14.2 (3.7)	13.6 (2.1)	14.6 (4.4)	$t(71) = 1.10, p = .28$
Years of use	12.7 (11.8)	13.7 (12.2)	12.1 (11.6)	$t(71) = -.57, p = .57$
Psychiatric disorders, current n (%) ⁵				
No disorder	30 (40.5%)	0 (0%)	30 (66.7%)	$\chi^2 = 32.52, p = .00$
Any disorder	44 (59.5%)	29 (100.0%)	15 (33.3%)	
Psychiatric disorders, past n (%)				
No disorder	12 (16.2%)	3 (10.3%)	9 (20.0%)	$\chi^2 = 1.21, p = .27$
Any disorder	62 (83.8%)	26 (89.7%)	36 (80.0%)	

Note. M=Mean; SD=Standard deviation

³All of participants were non-hispanic/latino.⁴Missing one data point⁵Any current or past PD diagnosis was excluded from the Non-PD group. Note. M=Mean;

Table 2.

Zero-order correlations for study variables

	1	2	3	4	5	6	7	8	9	10	M (SD)
1. Age	1	.021	.435 **	.042	.110	.212	.155	.076	.204	-.055	29.9 (12.6)
2. Sex ^a		1	-.022	-.095	.081	.196	.323 **	.201	.141	.170	47.3% ^b
3. FTND			1	-.037	.238 *	.433 **	.383 **	.316 **	.092	.234 *	3.9 (1.7)
4. PD Status ^a				1	.463 **	.062	.119	.188	.310 **	.220	39.2%
5. DERS total					1	.414 **	.479 **	.437 **	.420 **	.449 **	81.7 (23.3)
6. RFS-HA						1	.572 **	.583 **	.277 *	.356 **	2.4 (.8)
7. RFS-AD							1	.673 **	.315 **	.495 **	3.3 (.8)
8. RFS-NA								1	.412 **	.738 **	3.6 (.8)
9. SCQ-NC									1	.373 **	6.0 (1.4)
10. SCQ-NR										1	5.8 (1.9)

Note.

*
 $p < .05$,**
 $p < .01$, two-tailed;^aSpearman correlation coefficients;^b females;

M=Mean; SD=Standard deviation; Sex was coded 1 for males and 2 for females; FTCD=Fagerström test for cigarette dependence; PD=Panic disorder (1=yes, 0=no); DERS=Difficulties in emotion regulation scale; RFS=Reasons for Smoking; HA=Habitual subscale; AD=Addictive subscale; NA=Negative affect reduction subscale; SCQ=Smoking consequences questionnaire; NC=Negative consequences subscale; NR=Negative reinforcement subscale

Table 3.

Regression results for the mediation models

Y	Model	B	SE	t	P	CI (lower)	CI (upper)
1	PD→DERS (a)	23.336	4.883	4.779	0.000	13.593	33.08
	DERS→RFS-HA (b)	0.012	0.004	2.965	0.004	0.004	0.019
	PD→RFS-HA (c')	-0.136	0.184	-0.737	0.464	-0.504	0.232
	PD→RFS-HA (c)	0.133	0.166	0.798	0.427	-0.199	0.464
	PD→DERS→RFS-HA (a*b)	0.268	0.097			0.114	0.512
2	PD→DERS (a)	23.336	4.883	4.779	0.000	13.593	33.08
	DERS→RFS-AD (b)	0.013	0.005	2.574	0.012	0.003	0.023
	PD→RFS-AD (c')	0.016	0.181	0.089	0.929	-0.345	0.377
	PD→RFS-AD (c)	0.317	0.164	1.930	0.058	-0.011	0.645
	PD→DERS→RFS-AD (a*b)	0.301	0.125			0.098	0.597
3	PD→DERS (a)	23.336	4.883	4.779	0.000	13.593	33.08
	DERS→RFS-NA (b)	0.012	0.005	2.182	0.033	0.001	0.023
	PD→RFS-NA (c')	0.101	0.224	0.448	0.655	-0.347	0.549
	PD→RFS-NA (c)	0.379	0.188	2.017	0.048	0.004	0.755
	PD→DERS→RFS-NA (a*b)	0.279	0.143			0.044	0.608
4	PD→DERS (a)	23.336	4.883	4.779	0.000	13.593	33.08
	DERS→SCQ-NC (b)	0.019	0.007	2.791	0.007	0.005	0.033
	PD→SCQ-NC (c')	0.496	0.311	1.593	0.116	-0.126	1.118
	PD→SCQ-NC (c)	0.941	0.331	2.846	0.006	0.281	1.601
	PD→DERS→SCQ-NC (a*b)	0.445	0.206			0.123	0.938
5	PD→DERS (a)	23.336	4.883	4.779	0.000	13.593	33.08
	DERS→SCQ-NR (b)	0.031	0.01	3.023	0.004	0.011	0.052
	PD→SCQ-NR (c')	0.169	0.469	0.360	0.720	-0.767	1.104
	PD→SCQ-NR (c)	0.896	0.446	2.008	0.049	0.005	1.787
	PD→DERS→SCQ-NR (a*b)	0.728	0.304			0.264	1.476

Note. FTND=Fagerström test for nicotine dependence; PD=Panic disorder; DERS=Difficulties in emotion regulation scale; RFS=Reasons for Smoking; HA=Habitual subscale; AD=Addictive subscale; NA=Negative affect reduction subscale; SCQ=Smoking consequences questionnaire; NC=Negative consequences subscale; NR=Negative reinforcement subscale