

PAIN-RELATED ANXIETY AND SMOKING OUTCOMES: THE EXPLANATORY  
ROLE OF DYSPHORIA

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A Senior Honors Thesis

Presented to

the Faculty of the Department of Psychology

University of Houston

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In Partial Fulfillment

of the Requirements for the Degree

Bachelor of Science

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By

Tanya Smit

May, 2018

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## ABSTRACT

Scientific evidence suggests that pain-related anxiety may contribute to the maintenance of tobacco addiction among smokers with varying levels of pain. Yet, no work has investigated the relation between pain-related anxiety and cognitive-based smoking processes within a mechanistic model. Dysphoria may explain the relation between pain-related anxiety and smoking outcomes, as it is a construct that relates to pain and smoking outcomes. Thus, the current study examined the explanatory role of dysphoria in the relation between pain-related anxiety and three clinically significant smoking outcomes: perceived barriers to cessation, negative affect reduction motives, and negative mood abstinence expectancies. Participants included 101 ( $M_{\text{age}} = 32.74$  years,  $SD = 13.60$ ; 35.6% female) adult smokers. Results indicated that pain-related anxiety had an indirect effect on all dependent variables through dysphoria. The current findings provide evidence that dysphoria may serve to maintain maladaptive smoking processes in smokers with pain-related anxiety.

**Keywords:** Pain, Pain-Related Anxiety, Dysphoria, Smoking, Tobacco

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## **Pain-related Anxiety and Smoking Outcomes: The Explanatory Role of Dysphoria**

Cigarette smoking is the leading cause of preventable death and disability in the United States, contributing to over 480,000 deaths each year (Health and Human Services [HHS], 2010, 2014; Mack & Centers for Disease Control and Prevention [CDC], 2013). Smoking kills more people than obesity, substance abuse, infectious diseases, firearms, and traffic accidents, and increases the risk for coronary heart disease, stroke, lung cancer, and respiratory and cardiovascular disease combined (HHS, 2014). Despite widespread recognition of the health consequences of smoking (HHS, 2014), roughly 36.5 million Americans still smoke (Jamal, 2016), and thousands more become daily smokers each year (HHS, 2014). Extensive work has been devoted to understanding socioeconomic and psychological factors, including psychiatric disorders and symptoms, that contribute to the onset and maintenance of tobacco smoking (Grant, Hasin, Chou, Stinson, & Dawson, 2004; Leventhal & Zvolensky, 2015; Mathew, Hogarth, Leventhal, Cook, & Hitsman, 2017). Yet, within this body of literature, pain, a potentially clinically important risk factor for smoking, has received comparatively less scholarly attention.

Pain and pain-related processes frequently co-occur with and contribute to maladaptive smoking (Ditre, Brandon, Zale, & Meagher, 2011). Indeed, smoking is significantly more prevalent among individuals with chronic pain relative to the general population (Zvolensky, McMillan, Gonzalez, & Asmundson, 2009), and such differences may be a consequence of increased motivation to smoke cigarettes for pain coping (Ditre & Brandon, 2008; Patterson et al., 2012). Consequently, individuals who experience pain are at greater risk for smoking (Shi, Weingarten, Mantilla, Hooten, & Warner, 2010) and difficulty quitting (Ditre, Kosiba, Zale, Zvolensky, & Maisto, 2016; Zale & Ditre, 2014).

In addition to pain being linked to more maladaptive smoking patterns and cognitions, tobacco smoking has been identified as a causal agent in the development of numerous painful chronic health conditions, including chronic low back pain and rheumatoid arthritis (Shiri, Karppinen, Leino-Arjas, Solovieva, & Viikari-Juntura, 2010; Sugiyama et al., 2010). Thus, there are likely bidirectional pathways by which pain and smoking contribute to and maintain each other (Zale, Maisto, & Ditre, 2016). These pathways carry several public health implications, including increased negative health consequences and greater financial burden placed on individuals and health care systems (Ditre et al., 2011; Zale et al., 2016). The clinical importance and public health relevance of this work supports further scientific inquiry into the association between pain and tobacco cigarette smoking.

To more clearly elucidate the pain and smoking relation, researchers have begun to examine cognitive-affective process that relate to both conditions (Gonzalez, Hogan, McLeish, & Zvolensky, 2010; LaRowe, Langdon, Zvolensky, Zale, & Ditre, 2017). One such cognitive-affective pain process of interest is pain-related anxiety. Pain-related anxiety is the tendency for an individual to respond to actual or potential experiences of pain with anxiety or fear (McCracken, Zayfert, & Gross, 1992). Pain-related anxiety has been cross-sectionally and prospectively implicated as a unique predictor of smoking outcome expectancies, perceived barriers to quitting, tobacco dependence, and early lapse and relapse (Ditre, Langdon, Kosiba, Zale, & Zvolensky, 2015; Ditre, Zale, Kosiba, & Zvolensky, 2013; Gonzalez et al., 2010). Importantly, these relations have been observed even in the absence of co-occurring *chronic* pain (Abrams, Carleton, & Asmundson 2007; LaRowe et al., 2017). Thus, pain-related anxiety may contribute to the maintenance



of tobacco addiction among smokers who experience varying levels of pain. Although pain-related anxiety appears to be a clinically important construct in relation to smoking, this work is in its infancy. Indeed, while the relation between pain-related anxiety and smoking is increasingly well established, no research has identified underlying mechanisms that may explain these associations.

One promising transdiagnostic factor that may contribute to the pain-related anxiety-smoking relation is dysphoria. Dysphoria is a core symptom cluster of depression characterized by anhedonia, sadness, psychomotor disturbance, loss of self-esteem, cognitive difficulty, and worry (Leventhal, Zvolensky, & Schmidt, 2011). Dysphoria is associated with multiple aspects of smoking behavior, including higher levels of tobacco dependence, greater perceived barriers to cessation, and more severe withdrawal symptoms (Bakhshaie et al., 2017; Buckner et al., 2015). Among depressive symptoms, dysphoria holds the strongest association to smoking outcomes and is theorized to play a central role in the development and maintenance of maladaptive smoking (Leventhal & Zvolensky, 2015; Leventhal et al., 2011). Additionally, while not dysphoric symptoms per se, research on depressive symptoms, more broadly, supports a robust association between pain and depressive symptoms, such that smokers who experience chronic pain endorse greater levels of depressive symptoms (Linton, 2013; Shi et al., 2010; Zale et al., 2016). Indeed, depressive symptoms have been found to mediate the relation between pain and smoking (Goesling, Brummett, & Hassett, 2012). Despite the evidence for the association between pain constructs, depressive symptoms, and smoking, no research has examined the role of a specific symptom cluster of depression, such as dysphoria, in the association between pain-related anxiety and smoking processes. Thus, a clinically

important extension of extant empirical work would be to evaluate dysphoric symptoms in the relation between pain-related anxiety and smoking.

Theoretically, smokers who experience greater pain-related anxiety may avoid activities that may cause them pain, leading to self-isolation and, consequently, increased symptoms of dysphoria. Thus, as a by-product of fear or anxiety related to anticipated or experienced pain, these individuals may experience greater levels of dysphoric symptoms. Subsequently, consistent with negative reinforcement models of addiction (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004), these smokers may use smoking as a method to reduce or escape dysphoric states. The negative reinforcement cycle experienced by smokers with elevated pain-related anxiety, and subsequently increased dysphoria, may lead to more strongly held maladaptive beliefs about smoking, including greater perceived barriers to quitting (Macnee & Talsma, 1995), motivation to smoke to relieve negative affect symptoms (Piper et al., 2004), and negative mood-related abstinence expectancies (Abrams, Zvolensky, Dorman, Gonzalez, & Mayer, 2011). These outcomes are of particular interest because, when conceptualizing quitting as a dynamic process (Shiffman, 2005; Swan & Denk, 1987), they represent a continuum of processes that maintain tobacco use and therefore impede smoking cessation (Brandon, 1994; Copeland & Brandon, 2000; Zvolensky et al., 2007). Indeed, these processes tap into focused attention on the immediate positive effects of smoking (i.e., negative affect reduction), the potential challenges and difficulties that would arise if one attempted to quit, and beliefs that quitting will have negative consequences. To date, however, no work has examined the explanatory pathway from pain-related anxiety through dysphoric symptoms to these clinically-relevant smoking processes. Elucidating these relations

would provide a more thorough conceptual understanding for how pain-related constructs and dysphoria uniquely relate to barriers to quitting, motives for use, and negative expectations about quitting.

Together, the current study explored the explanatory role of dysphoria in the relation between pain-related anxiety and smoking outcomes (i.e. perceived barriers to cessation, negative reinforcement smoking motives, and smoking abstinence expectancies related to negative mood). Specifically, we hypothesized that smokers with elevated pain-related anxiety would experience greater barriers to cessation, endorse greater motivation to smoke to relieve negative internal states, and expect higher levels of negative mood during periods of smoking abstinence through dysphoria. Consistent with previous research, all effects were examined adjusting for gender, presence of a psychiatric disorder, cigarette dependence, and severity of recently experienced pain (Ditre et al., 2011; Ditre et al., 2015; Gonzalez et al., 2010).

## **METHOD**

### **Participants**

Participants included 101 adult daily smokers ( $M_{\text{age}} = 32.74$  years,  $SD = 13.60$ ; 35.6% female) recruited from communities in Burlington, Vermont and Houston, Texas. Inclusion criteria for the current study were: (1) being between 18 and 65 years of age; (2) being interested in making a serious, unaided, quit attempt; and (3) smoking a minimum of 5 cigarettes per day. Participants were excluded based on the following criteria: (1) pregnancy or the possibility of being pregnant (by self-report); (2) current use of nicotine replacement therapy and/or smoking cessation counseling; (3) current or past history of psychotic-spectrum symptoms or disorders; or (4) current suicidality. Most

participants (54 %) met criteria for a current mental health diagnosis based upon a structured clinical interview. The most commonly endorsed diagnoses were social phobia (14%), specific phobia (12%), panic disorder with or without agoraphobia (12%), and major depressive disorder (7%). The racial/ethnic distribution of this sample was 84.2% White/Caucasian; 7.9% Black/Non-Hispanic; 2.0% Hispanic; 1.0% Asian; 1.0% American Indian/Alaska Native; 3.0% Multi-racial; and 3.0% 'Others'. On average, participants reported smoking 16.7 cigarettes per day ( $SD = 10.46$ ), smoking their first cigarette at 17.3 years of age ( $SD = 3.37$ ), and reported being a daily smoker for an average of 14.8 years ( $SD = 12.94$ ).

## Measures

*Pain Anxiety Symptoms Scale (PASS)*. The PASS (McCracken et al., 1992) is comprised of four 10-item subscales that measure distinct dimensions of pain-related anxiety including cognitive anxiety/interference, fearful appraisals of pain, escape and avoidance behavior, and psychological symptoms related to pain. Of the 40 items rated on a 6-point scale from 0 (never) to 5 (always), five items (i.e. items 2, 8, 16, 31, and 40) are reverse scored (Asmundson, Collimore, Bernstein, Zvolensky, & Hadjistavropoulos, 2007; Ditre et al., 2015). The current study utilized total the PASS score (Cronbach's  $\alpha = .94$ ).

*Inventory of Anxiety and Depression Symptoms (IDAS)*. The IDAS (Watson et al., 2007) is a 64-item self-report instrument that assesses distinct affect symptom dimensions within the past two weeks. Items are answered on a 5-point Likert scale ranging from "not at all" to "extremely." The IDAS subscales show strong internal consistency, convergent and discriminant validity with psychiatric diagnoses and self-

report measures; and short-term retest reliability ( $r = 0.79$ ) with both community, and psychiatric patient samples (Watson et al., 2007). The current study used the dysphoria subscale (e.g. “I felt depressed;” 10 items), which demonstrated excellent internal consistency (Cronbach’s  $\alpha = .90$ ).

*Barriers to Cessation Scale (BCS)*. The BCS (Macnee & Talsma, 1995) is a 19-item self-report assessment of perceived barriers to or stressors resulting from smoking cessation (e.g., “Feeling less in control of your moods”). Responses are provided on a 4-point Likert scale ranging from not a barrier (0) to large barrier (3). The BCS has three subscales as originally developed: Addictive Barriers, External Barriers, and Internal Barriers. The BCS has demonstrated strong psychometric properties in a sample of treatment seeking smokers (Bakhshaie et al., 2017; Garey et al., 2017). The BCS total score was utilized in the present study (Cronbach’s  $\alpha = .87$ ).

*Wisconsin Inventory of Smoking Dependence Motives (WISDM)*. The WISDM (Piper et al., 2004) is a 68-item measure that reflects mechanisms underlying smoking dependence. All subscales of the WISDM are present across different levels of smoking heaviness and are related to symptoms of dependence and relapse (Piper et al., 2004). The WISDM is a reliable and valid measure of tobacco dependence (Ditre et al., 2015). The current study utilized the negative reinforcement subscale of the WISDM (Cronbach’s  $\alpha = .89$ ).

*Smoking Abstinence Expectancies Questionnaire (SAEQ)*. The SAEQ (Abrams et al., 2011) is a 28-item self-report measure that assesses consequences to be expected because of acute smoking abstinence (i.e., stop smoking cigarettes/using nicotine for one day). Items are rated in terms of expected likelihood on a scale from 0 (very unlikely) to

6 (very likely). The SAEQ yields four subscales: somatic symptoms (e.g., “I would feel short of breath”), harmful consequences (e.g., “I would feel like I’m dying”), negative mood consequences (e.g., “I would feel tense”), and positive consequences (e.g., “I would find it easy to concentrate” (Abrams et. al., 2011). The negative mood subscale was utilized in the current study (Cronbach’s  $\alpha = .88$ ).

*Fagerström Test for Cigarette Dependence (FTCD).* The FTCD (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) is a six-item assessment of an individual smoker’s tobacco dependence. Total scale scores range from 0 to 10, with higher scores reflecting high levels of physiological tobacco dependence on nicotine. In the current study, the FTCD total score was used to describe the smoking severity of the sample and included as a covariate. Although the internal consistency for the FTCD was low in the present sample (Cronbach’s  $\alpha = .57$ ), the alpha was similar to that reported in other work (Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994).

*Recent Bodily Pain.* The Short Form Health Survey – 20 (SF-20; Stewart, Hays, & Ware, 1988) is a 20 item self-report measure of general mental and physical health. Consistent with previous research, a single item was used to assess the presence of past-month bodily pain (i.e., “How much bodily pain have you had during the past four weeks”; (Ditre et al., 2015; LaRowe et al., 2017; Zvolensky et al., 2009). Response options ranged from 1 (*None*) to 5 (*Severe*).

## **Procedure**

Adult daily smokers were recruited from the community through a variety of methods (e.g., flyers, newspaper ads, radio announcements) to participate in a self-guided quit study examining barriers to successful smoking cessation (Langdon, Farris, Hogan,

Grover, & Zvolensky 2016; Langdon, Farris, Øverup, & Zvolensky 2015). Interested participants were scheduled for an in-person baseline assessment to determine study eligibility. Following written informed consent, participants were interviewed using the SCID-I/NP and completed a computerized self-report battery of questionnaires. Participants were compensated \$20 for participating in the baseline assessment, regardless of study eligibility. The current study is based on baseline (pre-cessation) data for a sub-set of the sample, which was selected on the basis of available data on all studied variables. The study protocol was approved by the University of Vermont and University of Houston Institutional Review Board.

### **Analytic Strategy**

Sample descriptive statistics and zero-order correlations among study variables were examined. Regression analyses were conducted using bootstrapping techniques through PROCESS, a conditional modeling program that utilizes an ordinary least squares-based path analytical framework to test for both direct and indirect effects (Hayes, 2013). Bootstrapping is the recommended approach when data distribution is non-normal or unknown (Kelley, 2005; Kirby & Gerlanc, 2013). An indirect effect is the product of path *a* and path *b* and is assumed to be significant if the confidence intervals (CIs) around their product do not include zero (Preacher & Hayes, 2008; Zhao, Lynch, & Chen, 2010). Effect size in mediation analysis was assessed with the completely standardized indirect effect size (*ES*), represented as the indirect effect of a one-unit change in the standardized predictor (1 unit=1 standard deviation) on the standardized outcome. *ES* is interpreted as small (0.01), medium (0.09), and large (0.25) (Preacher & Kelley, 2011).

Three models were conducted with (1) barriers to cessation (as measured by the BCS), (2) negative reinforcement smoking dependence motives (as measured by the WISDM-NR), and (3) negative mood abstinence expectancies (as measured by the SAEQ-NM) as criterion variables. Pain-related anxiety (as measured by the PASS) served as the predictor, and dysphoria (as measured by the IDAS-DYS) served as the explanatory variable in all models. Based on prior research (LaRowe, Langdon, Zvolensky, Zale, & Ditre, 2017; Ditre, Langdon, Koshiba, Zale, & Zvolensky, 2014; Zvolensky, McMillan, Gonzalez, & Asmundson, 2009), covariates included gender (0 = male, 1 = female), presence of an axis I mental health diagnosis (0 = absent, 1 = present), severity of bodily pain (as measured by the SF-20) and cigarette dependence (as measured by the FTCD); see Figure 1. All models were subjected to 10,000 bootstrap resamplings and 95-percent CIs were estimated (Hayes, 2009; as recommended by Kristopher J. Preacher & Hayes, 2004; Preacher & Hayes, 2008).

## **RESULTS**

### **Descriptive Analyses**

Of the 105 current, daily smokers potentially eligible to be included in analyses, 4 participants had incomplete data and were excluded from further analyses; no multivariate outliers were identified. The final sample included in analyses consisted of 101 participants. Zero-order correlations among all study variables are presented in Table 1. Pain-related anxiety and dysphoria were positively correlated ( $r = .48, p < .001$ ). Dysphoria was positively related to all criterion measures ( $r$ 's = .34 - .38,  $p < .001$ ). Pain-related anxiety was also positively correlated to all criterion measures ( $r$ 's = .22 - .35,  $p$ 's  $< .05$ ).



## Indirect Effect Analyses

Regression results are presented in Figure 1. For BCS, including covariates and PASS in a regression model accounted for significant variance ( $R^2 = .19$ ,  $F[5, 95] = 4.59$ ,  $p = .001$ ). The addition of IDAS-DYS to the regression model accounted for greater variance in BCS ( $R^2 = .24$ ,  $F[6, 94] = 4.99$ ,  $p < .001$ ). The independent indirect effect of PASS on BCS through IDAS-DYS was significant ( $ab = .03$ ,  $SE = .01$ ,  $CI_{95\%} [.007, .061]$ ). Specifically, greater pain-related anxiety was significantly associated with increased dysphoric symptoms, which was subsequently associated with a greater number of perceived barriers to cessation. The effect size for this pathway was in the small to medium range ( $ES = .08$ ,  $CI_{95\%} [.021, .180]$ ).

Specific to WISDM-NR, including covariates and PASS in a regression model accounted for significant variance ( $R^2 = .17$ ,  $F[5, 95] = 3.90$ ,  $p = .003$ ). The addition of IDAS-DYS to the regression model accounted for greater variance in WISDM-NR ( $R^2 = .26$ ,  $F[6, 94] = 5.44$ ,  $p < .001$ ). The independent indirect effect of PASS on WISDM-NR through IDAS-DYS was significant ( $ab = .03$ ,  $SE = .01$ ,  $CI_{95\%} [.009, .063]$ ). Specifically, greater pain-related anxiety was significantly associated with increased dysphoric symptoms, which was subsequently associated with greater motivation to smoke to relieve negative internal states. The effect size for this pathway was in the medium to large range ( $ES = .12$ ,  $CI_{95\%} [.033, .230]$ ).

Regarding SAEQ-NM, including covariates and PASS in a regression model accounted for significant variance ( $R^2 = .11$ ,  $F[5, 95] = 2.39$ ,  $p = .04$ ). The addition of IDAS-DYS to the regression model accounted for greater variance in SAEQ-NM ( $R^2 = .23$ ,  $F[6, 94] = 4.65$ ,  $p < .001$ ). The independent indirect effect of PASS on SAEQ-NM

through IDAS-DYS was significant ( $ab = .04$ ,  $SE = .02$ ,  $CI_{95\%} [.016, .081]$ ). Specifically, greater pain-related anxiety was significantly associated with increased dysphoric symptoms, which was subsequently associated with expecting higher levels of negative mood during periods of smoking abstinence. The effect size for this pathway was in the medium to large range ( $ES = .12$ ,  $CI_{95\%} [.046, .230]$ ).

### **Specificity Analyses**

To further strengthen interpretation of results, pain-related anxiety and dysphoria variables were reversed for each of the three models tested (Preacher & Hayes, 2004); specifically, IDAS-DYS was entered as the predictor, PASS was the explanatory variable, and all criterion variables remained the same. Tests of the indirect effects in these models were estimated based on 10,000 bootstrap re-samples. All results of the reversed models were non-significant (BCS:  $ab = .08$ ,  $SE = .07$ ,  $CI_{95\%} [-.0004, .257]$ ; WISDM-NR:  $ab = .04$ ,  $SE = .04$ ,  $CI_{95\%} [-.032, .142]$ ; and SAEQ-NM:  $ab = -.03$ ,  $SE = .05$ ,  $CI_{95\%} [-.039, .154]$ ), suggesting that dysphoria uniquely serves as a mediator among the observed relations.

## **DISCUSSION**

The current study tested the indirect effect of pain-related anxiety, through dysphoria, in relation to a number of clinically relevant smoking cognitions, including perceived barriers to cessation, motivation to smoke to relieve negative internal states, and expectations of higher levels of negative mood during periods of smoking abstinence. As hypothesized, results indicated that pain-related anxiety had an indirect effect on all criterion variables through dysphoria. Further, all effects were observed after controlling for gender, presence of a psychiatric disorder, cigarette dependence, and severity of

experienced pain. Additionally, to further strengthen findings, alternative models were tested and these yielded no significant indirect effects. Thus, there was empirical evidence that effects were specific to the hypothesized pathways.

Extending prior work on pain and smoking (Gonzalez et al., 2010; LaRowe et al., 2017), the present findings indicate that pain-related anxiety may be related to an array of clinically-relevant smoking processes via dysphoric symptoms. The present findings provide support for a conceptual model wherein smokers with elevated pain-related anxiety endorse more severe dysphoria. Indeed, these smokers may use smoking as a method to regulate negative affect symptoms related to increased pain-related anxiety and dysphoria, which, in turn, contributes to maladaptive thought patterns regarding smoking, including more perceived barriers to cessation, greater motivation to smoke to relieve negative internal states, and greater expectancies for negative mood experiences during periods of smoking abstinence. To our knowledge, this is the first study to investigate the relation between pain-related anxiety and smoking processes within a mechanistic model.

Based on the present findings, dysphoria serves as an important third variable that, prior to the current report, has been overlooked when examining relations between pain-related anxiety and smoking processes. More specifically, while pain-related anxiety significantly (or marginally) was related to all criterion variables, these relations were fully explained by the relation dysphoric symptoms demonstrated with both the predictor and criterion variables. Indeed, the tested models evinced a substantial impact on the dependent variables, as indicated by the medium to large effect sizes across two of the three criterion variables. Considering that both pain-related anxiety and dysphoric symptoms have been independently implicated as predictors of maladaptive smoking

processes (Buckner et al., 2015; Ditre et al., 2015; Gonzalez et al., 2010; LaRowe et al., 2017), the present study broadened this work by providing a more holistic understanding for how these constructs relate to and contribute to smoking cognitive processes.

Although not primary aims of the present report, two observations warrant comment. First, pain-related anxiety and dysphoria shared approximately 23% of their variance. Thus, although the two constructs were significantly related, this observation provides empirical data for the uniqueness as evinced by having 77% unique variance that differentiates them. Thus, pain-related anxiety and dysphoria are related, but distinct constructs. Second, it is also notable that although we did not target chronic pain patients, PASS scores were elevated (mean = 65) in our sample, with means comparable to those reported by chronic pain patients (60.7 to 68.4; (Crombez, Vlaeyen, Heuts, & Lysens, 1999; Roelofs, Peters, McCracken, & Vlaeyen, 2003). This finding further strengthens theoretical and empirical data that purport smokers may be inherently more vulnerable to experiencing pain and therefore report higher rates of pain-related anxiety.

Clinically, these findings provide empirical evidence that smoking interventions for those with elevated of pain-related anxiety may benefit from the addition of therapeutic tactics aimed at reducing dysphoric symptoms (e.g., behavioral activation; mindfulness- and acceptance-based approaches; (Cuijpers, Van Straten, & Warmerdam, 2007; Zettle, Rains, & Hayes, 2011). Considering that research has identified mindfulness- and acceptance-based therapy as successful for both smoking cessation treatment (Bowen & Marlatt, 2009; Gifford et al., 2004) and treatment for pain-related anxiety (McCracken & Keogh, 2009; McCracken, Spertus, Janeck, Sinclair, & Wetzel,

1999), integrating such treatments with standard smoking cessation treatment may be especially beneficial.

The implications of this study should be considered in the context of its limitations. First, participants were primarily White/Caucasian smokers. To generalize the observed pathways, replication in a sample of ethically/racially diverse smokers is needed. Second, this study employed self-report measures. Future work would benefit from the utilization of multi-method assessment approaches, such as interviewing or implicit testing. Third, only three clinically-relevant smoking processes were examined in this study. Although these processes are highly relevant to smoking behavior (Copeland & Brandon, 2000; Ditre et al., 2015; Garey et al., 2017), the current findings cannot determine how the observed pathways relate to other smoking processes or smoking behavior. Thus, while the present study provides the necessary foundation for explanatory pathways from pain-related anxiety to smoking outcomes through dysphoria, this work would benefit from testing these models with additional outcomes, such as relapse. Another possible extension of this research would be to consider potential moderators of the unique effects of dysphoria on smoking outcomes, such as age, gender, executive functioning, or other related constructs (e.g., pain-smoking expectancies; (Ditre, Zale, Heckman, & Hendricks, 2017)). Lastly, although this study examined dysphoria, a construct highly related to smoking behavior, it is possible that other psycho-social constructs may explain the observed effect of pain-related anxiety in the studied outcomes. To increase theoretical and empirical understanding of the relation between pain-related anxiety and tobacco smoking, additional explanatory variables should be tested.

Overall, findings provide a unique perspective for the role of dysphoria in the relation between pain-related anxiety and cognitive-based smoking processes as well as the strength of its contribution. This study furthers work on the pain-smoking relation by providing evidence for a conceptual model in which smokers with elevated pain-related anxiety endorse greater dysphoric symptoms and use smoking to reduce or escape symptoms of their pain-related anxiety and dysphoria, thus contributing to more maladaptive smoking patterns. Future work is needed to explore the extent to which dysphoria accounts for relations between pain-related anxiety and other smoking processes (e.g. withdrawal, cessation outcomes) to further inform evolving theoretical models of pain and tobacco dependence.

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Table 1.  
Descriptive Statistics and Correlations among Variables

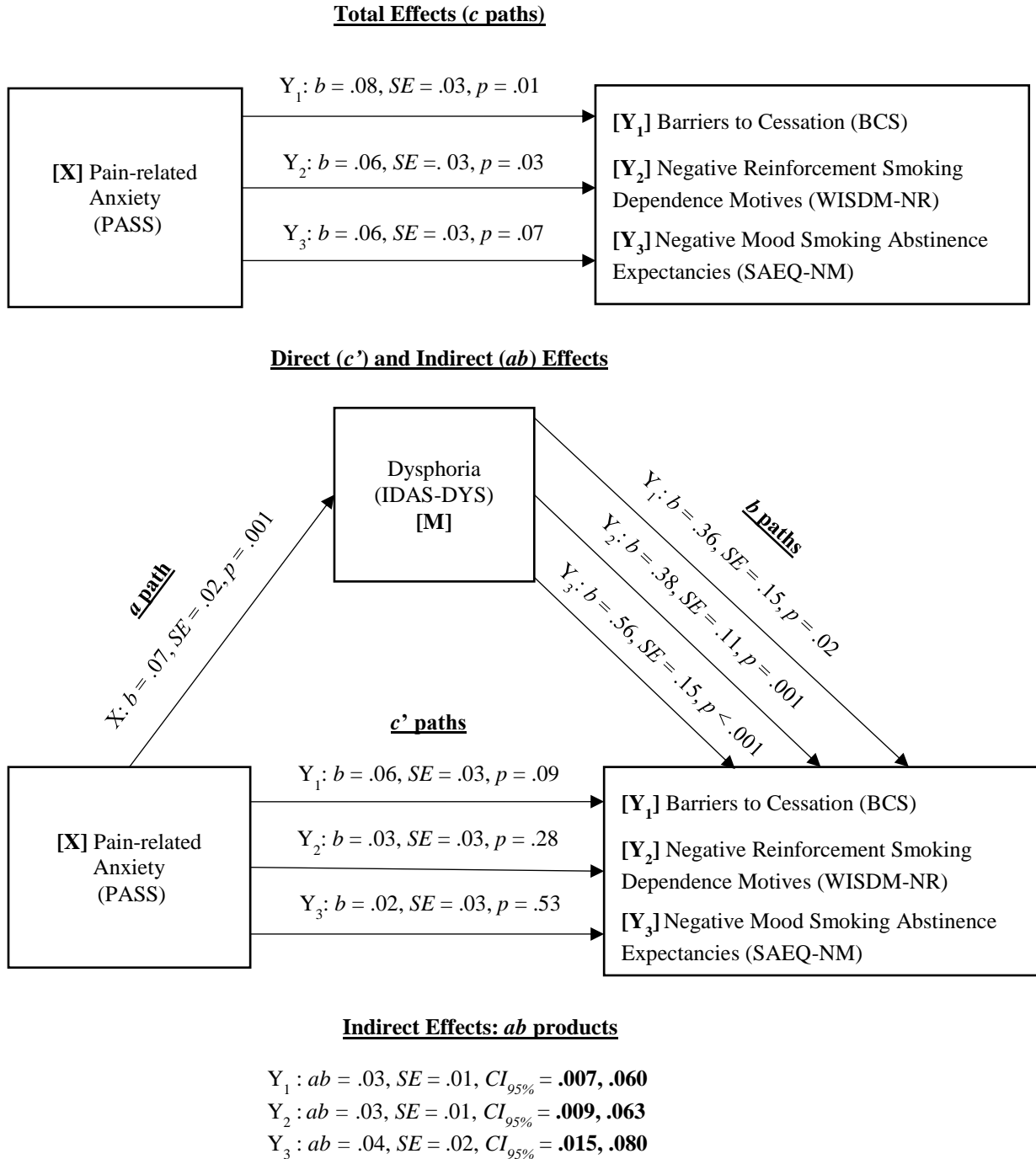
	Mean/ <i>n</i>	<i>SD</i> / %	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Sex (% female)	36	35.6	--								
2. Mental HD (% present)	54	53.5	.07	--							
3. Recent Bodily Pain	2.43	1.13	.14	.16	--						
4. FTCD	3.54	1.94	.07	-.17	.15	--					
5. PASS	65.07	33.19	.27**	.37***	.32**	-.2	--				
6. IDAS-DYS	20.12	7.76	.12	.47***	.29**	-.02	.48***	--			
7. BCS	26.78	10.32	.26**	.20*	.15	.18	.35***	.38***	--		
8. WISDM-NR	27.74	8.04	.18	.02	.08	.32**	.23*	.34**	.40***	--	
9. SAEQ-NM	24.63	10.21	.27**	-.002	.03	.10	.22*	.35***	.40***	.46***	--

*Note.* N = 101; \*\*\* p < .001, \*\* p < .01, \* p < .05. Gender: 0 = Male, 1 = Female; Mental HD (Health Diagnosis): 0= Absent, 1 = Present; FTCD = Fagerström Test for Cigarette Dependence (Heatherton et al., 1991), Recent Bodily Pain = Short Form Health Survey: pain item (Ware et al., 1992)), PASS = Pain Anxiety Symptoms Scale (McCracken et. al., 1992), IDAS-DYS = Inventory of Anxiety and Depression Symptoms-Dysphoria Subscale (Watson et. al., 2007), BCS = Barriers to Cessation Scale (Macnee & Talsma, 1995), WISDM = Wisconsin Inventory of smoking Dependence Motives (Piper et. al., 2004), SAEQ-NM = Smoking Abstinence Expectancies Questionnaire-Negative Mood Subscale (Abrams, Zvolensky, Dorman, Gonzalez, & Mayer, 2011).



Figure 1.

Conceptual model of the indirect effect ( $ab$ ) of pain anxiety on barriers to cessation, negative reinforcement smoking dependence motives, and smoking abstinence negative mood expectancies.



Note:  $N = 101$ ; \*  $p < .05$ . a path = Effect of X on M; b paths = Effect of M on  $Y_i$ ; c paths = Total effect of X on  $Y_i$ ; c' paths = Direct effect of X on  $Y_i$  controlling for M. Three separate paths were conducted ( $Y_{1-3}$ ) with the predictor (X). Covariates included in the establishment of paths included: Gender, Mental Health Diagnosis, FTCD = Fagerström Test for Cigarette Dependence (Heatherton et al., 1991), SFHS = Short Form Health Survey: Pain Item (Ware, Sherbourne, & Davies, 1992).