Experimental and Clinical Psychopharmacology

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Online First Publication, June 8, 2017. http://dx.doi.org/10.1037/pha0000127

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1064-1297/17/$12.00 http://dx.doi.org/10.1037/pha0000127

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Although emerging research suggests that pain-related anxiety may play a role in the maintenance of tobacco dependence, no previous work has examined pain-related anxiety as a predictor of smoking cessation outcomes. The current study aimed to test the hypothesis that pain-related anxiety would predict early lapse and relapse to cigarette smoking. These data were collected in the context of a primary study examining the role of emotional vulnerabilities in smoking cessation. The current analyses were conducted among 55 daily cigarette smokers who attempted to quit without psychosocial or pharmacological cessation aids. Pain-related anxiety was assessed at baseline using the Pain Anxiety Symptom Scale-20 (PASS-20). Early lapse and relapse were assessed using timeline follow-back procedures. Cox regression analyses indicated that pain-related anxiety was a significant predictor of both early smoking lapse and relapse such that for every 1-point increase on the PASS-20, the risk of early lapse increased by 3.7% and the risk of early relapse increased by 3.6%. These effects were evident above and beyond the variance accounted for by tobacco dependence, past 4-week pain severity, anxiety sensitivity, and the presence of current Axis I psychopathology. Kaplan-Meier survival analyses further revealed that among early lapsers, greater pain-related anxiety predicted a more rapid trajectory to lapse. Pain-related anxiety was also a significant predictor of early lapse when the sample was limited to smokers with past 4-week pain. These findings lend empirical support to the notion that pain-related anxiety may contribute to the maintenance of tobacco dependence among smokers who experience varying levels of pain intensity.

Public Health Significance
Pain-related anxiety was shown to predict early lapse and relapse to cigarette smoking. This study provides additional evidence that pain-related anxiety may contribute to the maintenance of tobacco dependence among smokers who experience varying levels of pain intensity.

Keywords: smoking, smoking cessation, pain, pain-related anxiety

Cigarette smoking remains the leading cause of preventable death worldwide (World Health Organization, 2008). Despite known health risks and an annual economic burden in excess of $300 billion in the United States, approximately 42 million Americans continue to smoke tobacco cigarettes (U.S. Department of Health & Human Services, 2014). Although approximately half of all smokers attempt to quit each year, nearly 70% do not utilize recommended pharmacological or behavioral cessation aids (e.g., nicotine replacement, cognitive–behavioral counseling; Centers for Disease Control and Prevention, 2011), and the vast majority (72–85%) who engage in an unaided quit attempt relapse within the first month (Hughes, Keely, & Naud, 2004). Whereas substantial progress has been made in the identification of reliable predictors of smoking cessation (e.g., cigarette dependence, withdrawal symptoms, self-efficacy for quitting; Ditre, Zale, & Brandon, 2015), additional work is needed to identify factors that can be addressed in the context of tailored interventions.

There is increasing empirical and clinical interest in the role of pain and related factors in the maintenance of tobacco dependence (e.g., Ditre, Heckman, Butts, & Brandon, 2010; Ditre, Langdon, Kosiba, Zale, & Zvolensky, 2015). Pain has been defined as "an unpleasant sensory and emotional experience associated with ac-

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This work was supported in part by National Research Service Award Grant F31-DA026634 awarded to Kirsten J. Langdon and an endowment awarded to Michael J. Zvolensky. All authors contributed in a significant way to the manuscript, and all authors have read and approved the final manuscript. There has been no prior dissemination of the ideas and data appearing in this article.

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tual or potential tissue damage or defined in terms of such damage" (International Association for the Study of Pain, 1994), and pain complaints account for up to 80% of all annual U.S. physician visits (Mayo Clinic, 2001). Although approximately 17% of U.S. adults smoke cigarettes (Jamal et al., 2015), an estimated 30–42% of persons with pain concurrently smoke tobacco cigarettes (Zvolensky, McMillan, Gonzalez, & Asmundson, 2009), and the prevalence of smoking among clinical pain patients may be as high as 68% (Michna et al., 2004).

An evolving reciprocal model suggests that pain and smoking interact in the manner of a positive feedback loop, resulting in greater pain and the maintenance of tobacco dependence (Ditre, Brandon, Zale, & Meagher, 2011; Zale, Maisto, & Ditre, 2016). Consistent with this perspective, regular tobacco smoking has been identified as a unique risk factor in the onset and progression of several chronically painful conditions, including low back pain (odds ratio [*OR*] = 1.79; Shiri, Karppinen, Leino-Arjas, Solovieva, & Viikari-Juntura, 2010) and rheumatoid arthritis (*OR* = 1.87 and 1.31, for men and women, respectively; Sugiyama et al., 2010), situational pain has been shown to motivate smoking urge and behavior (e.g., Ditre & Brandon, 2008), and pain patients have reliably endorsed smoking cigarettes to cope with pain (e.g., Patterson et al., 2012).

To better inform the development of tailored interventions, researchers have recently turned their attention to the identification of anxiety-relevant transdiagnostic factors in the etiology, progression, and maintenance of both pain and smoking (Zale et al., 2016). Indeed, various facets of anxiety (e.g., anxiety sensitivity) have been associated with greater pain and the maintenance of tobacco dependence (e.g., McCracken & Keogh, 2009). One factor that is of increasing clinical and empirical interest is the cognitive-affective construct termed *pain-related anxiety* (e.g., Ditre, Langdon, et al., 2015). Pain-related anxiety reflects the tendency to respond to pain or pain-related events with anxiety or fear (McCracken, Zayfert, & Gross, 1992), and it may be important to understanding the maintenance and exacerbation of tobacco dependence due to its specificity to pain-related phenomena and incorporation of pain-relevant behavioral responses (i.e., responding with escape or avoidance). Pain-related anxiety has been identified as a risk factor in the transition from acute to chronic pain (e.g., Boersma & Linton, 2006), and greater pain-related anxiety has been related to greater pain intensity, maladaptive approaches to pain coping, and increased somatic reactivity in anticipation of pain-eliciting physical activity (McCracken, Gross, Sorg, & Edmands, 1993). A growing body of evidence further suggests that pain-related anxiety can be experienced even in the absence of cooccurring clinical pain (e.g., Abrams, Carleton, & Asmundson, 2007).

Pain-related anxiety has also been implicated in the maintenance of substance use in general (e.g., Hogan, Gonzalez, Howell, Bonn-Miller, & Zvolensky, 2010) and tobacco smoking in particular (e.g., Ditre, Langdon, et al., 2015). Among individuals with chronic pain, higher levels of pain-related anxiety have been associated with current smoking (vs. nonsmoking; Hooten et al., 2009; Hsu, Harden, & Houle, 2002) and the use of tobacco to cope with pain (Patterson et al., 2012). Pain-related anxiety has also been positively associated with both primary (i.e., central features of tobacco dependence, such as compulsion to smoke) and secondary (i.e., situational motivators of smoking, such as mood regulation) smoking-dependence motives among smokers with chronic pain (Ditre, Zale, Kosiba, & Zvolensky, 2013) and those recruited from the local community (Ditre, Langdon, et al., 2015). Among community samples, pain-related anxiety has been positively associated with self-reported barriers to smoking cessation and expectations that smoking can alleviate negative mood (Ditre, Langdon, et al., 2015; Gonzalez, Hogan, McLeish, & Zvolensky, 2010). Collectively, these data suggest that pain-related anxiety should be considered in the maintenance of tobacco dependence among smokers with and without cooccurring pain.

The primary goal of the current study was to conduct a series of secondary analyses for the purpose of testing whether pain-related anxiety would predict early cessation outcomes among a sample of daily tobacco smokers who participated in an unaided smoking cessation attempt (i.e., they were asked to quit smoking without using any psychosocial or pharmacological cessation aids). Given evidence that pain-related anxiety may be particularly important among smokers with cooccurring pain (e.g., Ditre et al., 2013; Hooten et al., 2009), a secondary goal of this study was to examine the relationship between pain-related anxiety and cessation outcomes among a subsample of participants who endorsed past 4-week pain. Specifically, we hypothesized that higher levels of pain-related anxiety would be associated with (a) an increased risk of early lapse (i.e., a lapse within 14 days postquit) and early relapse (i.e., a relapse beginning within 28 days postquit) to smoking and (b) a more rapid trajectory to lapse among early lapsers and to relapse among early relapsers. Early abstinence outcomes were of particular interest given that participants engaged in an unaided quit attempt and that duration of abstinence before the first lapse/relapse (e.g., <1 month) is highly predictive of longer-term cessation outcomes (e.g., 6–12 months; Brown et al., 2001).

Method

Participants

Participants were recruited from the local community at two sites (Houston, TX and Burlington, VT) to participate in a primary study of emotional vulnerabilities in smoking cessation that consisted of a 90-day unaided quit attempt (Langdon, Farris, Øverup, & Zvolensky, 2016). Participants were screened for the following inclusion criteria: between 18 and 65 years of age, smoke at least eight cigarettes per day for at least 1 year (verified via expired carbon monoxide [CO] breath analysis ≥8 ppm), have not decreased the number of daily cigarettes smoked by more than half in the past 6 months, and willing to engage in an unaided quit attempt. Participants were also screened for the following exclusion criteria: current use of nicotine replacement, other tobacco products, or pharmacological smoking cessation aids (e.g., varenicline); current substance dependence (excluding nicotine dependence); current or past history of psychotic spectrum symptoms or disorders; and current use of psychotropic medication.

A total of 122 participants attended a baseline session, and 83 were deemed eligible to participate in the self-guided quit attempt (those who were ineligible endorsed current use of psychotropic medications, current substance dependence, expired CO < 8 ppm, and/or a reduction in cigarettes smoked per day >50% in the last 6 months; $n = 39$). Three participants were excluded because they

did not complete the measure of pain-related anxiety. Of the remaining 80 participants, 55 (69%) attended their quit day appointment and were included in the current analyses. It is important to note that modest retention rates are expected in studies that ask daily tobacco smokers to engage in an unaided, nonincentivized quit attempt. For example, in a previous study that required an unaided quit attempt, approximately 23% of the sample withdrew before the quit day (Zvolensky et al., 2008). In the current sample, those who attended their quit day appointment smoked fewer cigarettes per day ($M = 15.564$, $SD = 0.809$) than those who did not attend their quit day appointment ($M = 18.600$, $SD = 1.200$), $F(1, 78) = 4.399$, $p = .039$. No differences in any other baseline sociodemographic, smoking, or pain variable were observed as a function of quit day attendance (all ps $> .05$).

Measures

Pain-related anxiety. Pain-related anxiety was assessed using the Pain Anxiety Symptom Scale-20 (PASS-20; McCracken & Dhingra, 2002) item. The PASS-20 (range: 0–100) uses a 6-point Likert scale ranging from 0 (*never*) to 5 (*always*) to assess how often participants engage in various thoughts (e.g., "When I feel pain, I am afraid that something terrible will happen") and behaviors (e.g., "I avoid important activities when I hurt"). The PASS-20 demonstrated excellent internal consistency in the current sample ($\alpha = .91$).

Past 4-week pain severity. The Short Form Health Survey-12 (SFHS; Ware, Kosinski, & Keller, 1996) is a widely used 12-item self-report measure of mental and physical health. Consistent with previous research (e.g., Ditre, Langdon, et al., 2015), a single item was used to assess the presence of past 4-week bodily pain at the baseline session (i.e., "How much bodily pain have you had during the past four weeks?"; Ware et al., 1996). Response options consisted of *none*, *very mild*, *mild*, *moderate*, and *severe.* Given demonstrated associations between the presence of pain and numerous smoking-related factors and outcomes (e.g., Ditre et al., 2011; Zvolensky, McMillan, et al., 2009), past 4-week bodily pain severity was selected as an a priori covariate for all statistical analyses.

Tobacco use and dependence. Historical and current tobacco use (e.g., number of cigarettes smoked per day) were assessed via self-report. Tobacco dependence was assessed using the Heaviness of Smoking Index (HSI; Heatherton, Kozlowski, Frecker, Rickert, & Robinson, 1989), which is composed of two items (i.e., "How soon after you wake up do you smoke your first cigarette?" and "How many cigarettes per day do you smoke?"; Heatherton et al., 1989). HSI scores (range: 0–6) were identified as an a priori covariate given that they have been shown to predict smoking abstinence outcomes (Courvoisier & Etter, 2010), especially during the early stages (i.e., 1 week to 1 month) of a cessation attempt (Yong et al., 2014).

Current Axis I psychopathology. All participants were administered the Structured Clinical Interview for *DSM–IV–TR* Axis I Disorders, Non-Patient Edition (SCID-IV-N/P), by a trained clinician to assess whether participants met criteria for past-month Axis I psychopathology. Interviews were audiotaped, the reliability of a random selection of 10% of interviews was checked for accuracy, and there were no diagnostic disagreements between the SCID-IV-N/P interviewer and outsider raters. Consistent with previous work, a single dichotomous variable was created to reflect the presence (1) or absence (0) of current Axis I psychopathology (e.g., Johnson, Farris, Schmidt, Smits, & Zvolensky, 2013). Given established associations between the presence of Axis I psychopathology and greater difficulty quitting (e.g., Lasser et al., 2000), this variable was identified as an a priori covariate.

Anxiety sensitivity. The Anxiety Sensitivity Index-III (ASI-3; Taylor et al., 2007) is a well-established, 18-item measure of the fear that arousal-related sensations may result in adverse consequences such as death, insanity, or social rejection. The ASI-3 (range: 0–72) uses a 5-point Likert scale ranging from *very little* (0) to *very much* (5) to assess how much participants agree with various statements (e.g., "It scares me when my heart beats rapidly"). Given that anxiety sensitivity has been positively associated with affect-regulatory smoking motives (Farris, Leventhal, Schmidt, & Zvolensky, 2015), more severe nicotine withdrawal (e.g., Johnson, Stewart, Rosenfield, Steeves, & Zvolensky, 2012), and increased risk for lapse and relapse (Assayag, Bernstein, Zvolensky, Steeves, & Stewart, 2012; Zvolensky, Stewart, Vujanovic, Gavric, & Steeves, 2009), ASI-3 scores were selected as an a priori covariate. The ASI-3 demonstrated excellent internal consistency in the current sample ($\alpha = .93$).

Time to lapse/relapse. Time to lapse and relapse were assessed via timeline follow-back procedures. The timeline follow-back is a widely used interview-style assessment that incorporates calendar-guided recall and anchoring of dates to significant events (Sobell & Sobell, 1992). Participants were asked to recall and record the total number of cigarettes smoked each day before the follow-up session. Timeline follow-back procedures for cigarette smoking have demonstrated high reliability and validity (as measured by correlations with daily monitored smoking, reports from significant others, and saliva cotinine levels; Brown et al., 1998). Consistent with previous research, a lapse (i.e., any instance of smoking after a quit attempt) during the first 14 days postquit was classified as an early lapse (e.g., Brown et al., 2008; Holt, Litt, & Cooney, 2012; Zvolensky, Stewart, et al., 2009), and a relapse (i.e., 7 consecutive days of smoking following a quit attempt) beginning during the first 28 days postquit was classified as an early relapse (e.g., al'Absi, Hatsukami, & Davis, 2005; Nakajima & al'Absi, 2011). These classifications were also used because they tend to capture most early lapses/relapses and have demonstrated utility in predicting longer-term abstinence rates (e.g., Brown et al., 2001, 2009).

Biochemical verification of smoking status. Expired CO was measured at all in-person visits using a Vitalograph Breathco CO monitor (Vitalograph Inc. Lenexa, Kansas). Expired CO is measured in parts per million and provides an indirect, noninvasive measure of blood carboxyhemoglobin (Bittoun, 2008) that is most sensitive to recent smoking (e.g., within 24 h; Bittoun, 2008). Recent smoking can be biochemically verified via expired CO $\geq$ 8 ppm (Benowitz et al., 2002).

Procedure

All procedures for this study, titled "Anxiety Vulnerability and Smoking Cessation," were approved by the University of Vermont Institutional Review Board (IRB #CHRBS B09-058) and the University of Houston Institutional Review Board (IRB #12389–02-[7513]). Participants were recruited to take part in a study exam-

ining barriers to successful smoking cessation (Langdon et al., 2016). Upon arrival to the baseline session, smoking status was biochemically verified by expired CO, the SCID-IV/NP was administered, and self-report questionnaires were completed. Participants were compensated $20 for completion of the baseline session. Eligible respondents were then invited to participate in an unaided smoking cessation attempt. Each participant selected his or her own quit date, which typically occurred within 2 weeks of the baseline assessment (M = 12.8 days, SD = 5.7). Participants were instructed to quit smoking on their own, without any assistance (i.e., pharmacological or psychosocial treatment; Langdon et al., 2016). In-person follow-up appointments were scheduled for the quit day and Days 3, 7, 14, 28, and 90 postquit. Each follow-up visit consisted of self-report assessments, timeline follow-back procedures, and biochemical verification of smoking status via expired CO. Participants were compensated $10 for completing each follow-up assessment and could earn an additional $20 for completing all of them. Participants were not incentivized to remain abstinent.

Data Analytic Plan

All analyses were conducted using SPSS Statistics 21 (IBM Corporation, 2012). First, we ran a series of bivariate correlations to test zero-order associations among PASS-20 total scores (pain-related anxiety), number of days to lapse and relapse, HSI scores (tobacco dependence), severity of past 4-week bodily pain, presence of current Axis I psychopathology, ASI-3 scores (anxiety sensitivity), and sociodemographic factors.

Next, we used the Cox proportional hazards model to estimate the risk of both early lapse and early relapse as a function of pain-related anxiety. The Cox model is a well-established statistical procedure that has frequently been used to examine predictors of lapse/relapse to cigarette smoking (e.g., al'Absi, Nakajima, Allen, Lemieux, & Hatsukami, 2015; Zvolensky et al., 2008). This semiparametric model estimates hazard ratios (HRs) by examining the pattern of covariation of predictor variables with the event of interest (Cox & Oakes, 1984). Consistent with previous research, individuals who maintained abstinence during the given time period (i.e., did not report an early lapse or early relapse) or withdrew from the study before having lapsed/relapsed were censored (e.g., al'Absi et al., 2015). Established procedures for the Cox proportional hazards model indicate that a minimum of five events should be included per predictor variable to increase confidence interval coverage and decrease relative bias and Type I error (Vittinghoff & McCulloch, 2007). After ensuring that our models were consistent with this recommendation (our models included five predictor variables, and we observed 44 events for early lapse and 34 events for early relapse), covariates (i.e., ASI-3 total scores, past 4-week pain severity, presence of current Axis I psychopathology, and HSI scores) were entered into the first step of each model and continuous PASS-20 scores were entered at the second step.

We used Kaplan-Meier survival curves to compare the trajectories to early lapse and relapse between participants with high versus low levels of pain-related anxiety. Consistent with previous research, PASS-20 scores were dichotomized via median split (e.g., Evans, Seidman, Lung, Zeltzer, & Tsao, 2013). The Kaplan-Meier survival curve represents the probability of maintaining smoking abstinence for a given length of time while considering time in many small intervals (Kaplan & Meier, 1958). Two survival curves can be compared statistically using a log-rank test to challenge the null hypothesis that the survival curves do not differ by group (Goel, Khanna, & Kishore, 2010). If a significant log-rank result is observed ($p < .05$), then it can be concluded that the trajectory to lapse/relapse differs based on group status. These procedures are well-established and have been used in numerous studies examining differences in time to lapse/relapse to cigarette smoking (e.g., Wong, Chan, & Lam, 2016).

Results

Participant Characteristics

Participants included 55 current daily tobacco smokers (66% male; M_{age} = 34.8 years, SD = 14.6) who reported smoking approximately 16 cigarettes per day (SD = 5.5) for an average of 16 years (SD = 14.0). Mean expired CO at baseline was 18.93 ppm (SD = 9.30), and the mean HSI score was 2.6 (SD = 1.4), indicating a moderate level of tobacco dependence (e.g., Chaiton, Cohen, McDonald, & Bondy, 2007). The sample was predominantly White (87%) and fairly well educated (27% completed 4 years of college). Structured clinical interviews (SCID-IV-N/P) revealed that 38% of the sample met criteria for current (past-month) Axis I psychopathology. PASS-20 total scores ranged from 0 to 68 (M = 28.4, SD = 16.5).

Most participants (n = 44; 80%) endorsed an early lapse to smoking, and approximately 62% of the sample (n = 34) endorsed an early relapse to smoking. Participants who reported continued abstinence provided a lower CO reading at both the Day 14 follow-up appointment (M = 2.78 ppm, SD = 3.96) and the Day 28 follow-up appointment (M = 2.43, SD = 3.05) compared with participants who reported having smoked (M = 8.00 ppm, SD = 7.35 and M = 11.66, SD = 7.94, respectively; ps $< .05$). Although expired CO could not be used to verify prolonged abstinence (expired CO is most sensitive to recent smoking; Bittoun, 2008), readings at the in-person follow-up appointments were consistent with self-reported smoking status. We observed a moderate correlation (r = .543) between early lapse and early relapse, which indicates that although there was some overlap between early lapsers and early relapsers, these outcomes were not redundant (e.g., 25% of participants who endorsed an early lapse to smoking did not subsequently endorse an early relapse).

Approximately 73% (n = 40) of the sample endorsed at least very mild past 4-week pain, and smokers who endorsed past 4-week pain reported higher levels of pain-related anxiety (M = 31.68, SD = 16.06) than those who did not endorse past 4-week pain (M = 19.60, SD = 14.68; $F(1, 53) = 6.449$, p = .014). Among smokers who reported past 4-week pain, 82.5% (n = 33) endorsed an early lapse to smoking, and 65% (n = 26) endorsed an early relapse. Sociodemographic and clinical data are presented in Table 1.

Bivariate Correlations

PASS-20 total scores were positively correlated with past 4-week pain severity, r = .37, $p < .01$, ASI-3 total scores, r = .70,

Table 1
Sociodemographic, Smoking, and Pain Characteristics

Demographic/characteristic	Pain-related anxiety		Past 4-week pain status		Total sample
	Low *n* (%)	High *n* (%)	No pain *n* (%)	Pain *n* (%)	
Gender					
Male	22 (78.6)	14 (51.9)	9 (60.0)	27 (67.5)	36 (65.5)
Race					
White	25 (89.3)	23 (85.2)	14 (93.3)	34 (85.0)	48 (87.3)
Black or African American	0 (0.0)	3 (11.1)	0 (0.0)	3 (7.5)	3 (5.5)
Other	3 (10.7)	1 (3.7)	1 (6.7)	3 (7.5)	4 (7.3)
Marital status					
Single	13 (46.4)	17 (63.0)	9 (60.0)	21 (52.5)	30 (54.5)
Married/living with someone	10 (35.7)	5 (18.5)	3 (20.0)	12 (30.0)	15 (27.3)
Separated/divorced/annulled	5 (17.9)	5 (18.5)	3 (20.0)	7 (17.5)	10 (18.2)
Education, years					
12	2 (7.1)	4 (14.8)	2 (13.3)	4 (10.0)	6 (10.9)
12–15	20 (71.4)	14 (51.8)	9 (60.0)	25 (62.5)	33 (61.9)
≥16	6 (21.4)	9 (33.3)	4 (26.7)	11 (27.5)	15 (27.3)
Current Axis I psychopathology[a]	8 (28.6)	13 (48.1)	5 (33.3)	16 (40.0)	21 (38.2)
Past 4-week pain severity[b]					
None	11 (39.3)	4 (14.8)	15 (100)	0 (0.0)	15 (27.3)
Very mild	10 (35.7)	12 (44.4)	0 (0.0)	22 (55.0)	22 (40.0)
Mild	4 (14.3)	5 (18.5)	0 (0.0)	9 (22.5)	9 (16.4)
Moderate	1 (3.6)	5 (18.5)	0 (0.0)	6 (15.0)	6 (10.9)
Severe	2 (7.1)	1 (3.7)	0 (0.0)	3 (7.5)	3 (5.5)
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Age	34.46 (15.58)	35.11 (13.68)	29.87 (12.64)	36.63 (14.93)	34.78 (14.55)
Cigarettes per day	16.32 (6.40)	14.78 (4.45)	16.27 (6.87)	15.30 (5.01)	15.56 (5.53)
Years of smoking	15.75 (14.72)	16.78 (13.44)	12.80 (13.68)	17.55 (14.04)	16.25 (13.98)
HSI tobacco dependence	2.75 (1.48)	2.37 (1.31)	2.73 (1.44)	2.50 (1.40)	2.56 (1.40)
Anxiety sensitivity[c*]	8.61 (6.15)	21.26 (14.05)	11.47 (8.53)	16.08 (13.50)	14.82 (12.44)
Pain-related anxiety[d**]	15.71 (8.16)	41.52 (11.95)	19.60 (14.68)	31.68 (16.06)	28.38 (16.48)
Days to first lapse	18.48 (31.86)	6.92 (18.42)	17.14 (29.04)	11.37 (25.98)	12.92 (26.67)
Days to first relapse	28.18 (35.91)	15.66 (25.69)	27.55 (33.49)	19.80 (30.84)	21.65 (31.29)

Note. Assessed using the [a] SCID-IV-N/P, [b] a single item from the SFHS, [c] the ASI-3, and [d] the PASS-20.
[*] Significant ($p < .05$) difference as a function of pain-related anxiety. [**] Significant ($p < .05$) difference as a function of past 4-week pain status.

$p < .01$, and the presence of current Axis I psychopathology, $r = .30$, $p < .05$. The presence of current Axis I psychopathology was negatively associated with number of days to first smoking lapse ($r = -.29$; $p < .05$). No additional covariates were identified via bivariate analyses.

Pain-Related Anxiety and Early Lapse and Early Relapse to Smoking

As hypothesized, Cox regression analysis revealed that pain-related anxiety was a significant predictor of early smoking lapse (HR = 1.037, $p = .013$; see Table 2). Examination of the HR revealed that for every 1-point increase on the PASS-20, the risk of early lapse increased by 3.7%. These effects were evident above and beyond the variance accounted for by tobacco dependence scores, past 4-week pain severity, anxiety sensitivity, and the presence of current Axis I psychopathology. Also as hypothesized, Cox regression analysis revealed that pain-related anxiety was a significant predictor of early smoking relapse after accounting for the same covariates, such that for every 1-point increase on the PASS-20, the risk of early relapse increased by 3.6% (HR = 1.036, $p = .027$; see Table 2).

Pain-Related Anxiety and Trajectories to Early Lapse and Early Relapse to Smoking

Kaplan-Meier survival analysis further revealed that, among early lapsers, greater pain-related anxiety predicted a more rapid trajectory to lapse ($p = .037$; Figure 1). All participants (100%) high in pain-related anxiety lapsed by Day 7, compared with just 77.3% of those low in pain-related anxiety. Among early relapsers, 84.2% of participants who reported high pain-related anxiety relapsed by Day 7, compared with only 66.7% of those low in pain-related anxiety. However, no significant differences in relapse trajectories were observed as a function of pain-related anxiety ($p = .322$; Figure 1).

Pain-Related Anxiety and Early Lapse/Relapse Among Smokers With Pain

To further explore relations between pain-related anxiety and early lapse/relapse, we replicated our analyses among the subsample of participants who endorsed past 4-week pain. Similar to the primary findings, Cox regression analysis revealed that pain-related anxiety was a significant predictor of early smoking lapse among smokers who endorsed past 4-week pain (HR = 1.050, $p =$

Table 2
Cox Proportional Hazards Regressions

Variable	Unadjusted HR (95% confidence interval)	*p*	Adjusted HR (95% confidence interval)	*p*
Early lapse among entire sample[a]				
Past 4-week pain severity[c]	1.160 (0.915–1.469)	.219	1.172 (0.901–1.525)	.238
Anxiety sensitivity[d]	0.999 (0.976–1.022)	.938	0.940 (0.899−0.982)	.005**
Current Axis I psychopathology[e]	2.232 (1.183–4.210)	.013*	3.567 (1.641–7.755)	.001**
Tobacco dependence[f]	0.988 (0.815–1.197)	.900	1.129 (0.897–1.421)	.302
Pain-related anxiety[g]	1.011 (0.995–1.028)	.174	1.037 (1.008–1.068)	.013*
Early relapse among entire sample[b]				
Past 4-week pain severity	1.066 (0.804–1.412)	.658	1.041 (0.748–1.449)	.812
Anxiety sensitivity	1.007 (0.981–1.033)	.597	0.958 (0.913–1.004)	.071
Current Axis I psychopathology	1.895 (0.947–3.789)	.071	3.033 (1.276–7.207)	.012*
Tobacco dependence	1.175 (0.926–1.492)	.185	1.379 (1.032–1.842)	.030*
Pain-related anxiety	1.014 (0.995–1.033)	.154	1.036 (1.004–1.069)	.027*
Early lapse among participants with pain				
Past 4-week pain severity	1.043 (0.748–1.453)	.804	0.993 (0.687–1.434)	.968
Anxiety sensitivity	1.007 (0.986–1.029)	.517	0.944 (0.898−0.992)	.023*
Current Axis I psychopathology	1.863 (0.896–3.876)	.096	2.767 (0.977–7.833)	.055
Tobacco dependence	0.965 (0.772–1.205)	.752	1.055 (0.820–1.357)	.677
Pain-related anxiety	1.019 (1.000–1.039)	.046*	1.050 (1.012–1.089)	.009**
Early relapse among participants with pain				
Past 4-week pain severity[b]	0.884 (0.583–1.340)	.562	0.862 (0.549–1.352)	.517
Anxiety sensitivity[c]	1.010 (0.985–1.036)	.443	0.966 (0.914–1.021)	.216
Current Axis I psychopathology[d]	1.816 (0.825–3.995)	.138	2.443 (0.818–7.292)	.109
Tobacco dependence[e]	1.141 (0.866–1.503)	.350	1.234 (0.910–1.673)	.176
Pain-related anxiety[f]	1.017 (0.995–1.040)	.122	1.036 (0.996–1.078)	.075

Note. [a] First lapse within 14 days and [b] relapse within 28 days. Assessed using a single item from the [c] SFHS and the [d] ASI-3, [e] SCID-IV-N/P, [f] HSI, and [g] PASS-20.
* $p < .05$. ** $p < .01$.

.009; shown in Table 2), such that every 1-point increase on the PASS-20 was associated with a 5.0% increase in the risk of early lapse. Although 78.3% of smokers with high levels of pain-related anxiety endorsed an early relapse to smoking (vs. 47.1% of smokers with low levels of pain-related anxiety), pain-related anxiety was not found to be a significant predictor of early relapse among smokers with past 4-week pain (HR = 1.036, $p = .075$; see Table 2). Kaplan-Meier survival analyses revealed no differences in trajectory to lapse among early lapsers ($p = .098$) or to relapse among early relapsers ($p = .958$) as a function of pain-related anxiety among participants with cooccurring pain.

Discussion

The current study is the first to examine pain-related anxiety as a predictor of lapse and relapse to smoking. As hypothesized,

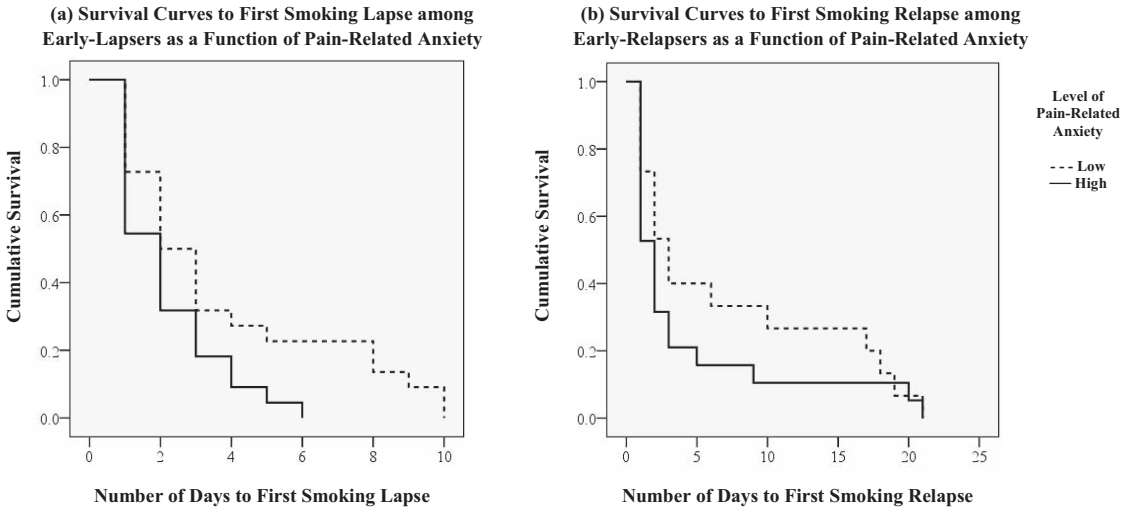

Figure 1. Kaplan-Meier survival curves.

pain-related anxiety was observed to be a significant predictor of both early lapse and early relapse among a sample of smokers who were recruited from the local community to participate in an unaided quit attempt. It is important to note that these effects were evident above and beyond the variance accounted for by tobacco dependence, past 4-week pain severity, anxiety sensitivity, and the presence of current Axis I psychopathology. Among early lapsers, greater pain-related anxiety was also associated with a more rapid trajectory to lapse. No differences in trajectory to early relapse were observed as a function of pain-related anxiety. Pain and anxiety have both been shown to motivate smoking behavior, and it is possible that these factors may be more likely to interfere with quit attempts among smokers who endorse higher levels of pain-related anxiety (e.g., Ditre & Brandon, 2008; Ditre et al., 2010; Kimbrel, Morissette, Gulliver, Langdon, & Zvolensky, 2014). Only recently have researchers begun to evaluate pain and anxiety-related cognitive-affective processes as potential mechanisms in the maintenance of tobacco dependence (e.g., Ditre et al., 2011; Hooten, Shi, Gazelka, & Warner, 2011), and these findings are consistent with demonstrated positive associations among pain-related anxiety, smoking-dependence motives (Ditre, Langdon, et al., 2015; Ditre et al., 2013), and perceived barriers to cessation (Ditre, Langdon, et al., 2015).

Analyses conducted among the subsample of participants who endorsed past 4-week pain largely corroborated the primary findings and suggest that pain-related anxiety may be associated with an even greater risk of early lapse among smokers with cooccurring pain (every 1-point increase on the PASS-20 was associated with a 5% increased risk in early lapse among smokers with cooccurring pain compared with a 3.7% increased risk among the total sample). Pain-related anxiety was not associated with risk of early relapse when analyses were limited to smokers who endorsed past 4-week pain, although this may have been a function of reduced statistical power ($n = 40$ smokers endorsed past 4-week pain). It is interesting to note that we observed a wide range of PASS-20 scores among the subsample of smokers who reported no past 4-week pain ($M = 19.60$, $SD = 14.68$, range = 55), demonstrating that smokers may endorse varying levels of pain-related anxiety even in the absence of cooccurring pain. In addition, we noted that anxiety sensitivity was associated with a decreased likelihood of early lapse among both the entire sample and the subsample of smokers with cooccurring pain ($ps < .05$). In contrast, previous work has documented an increased risk of early lapse among smokers with greater anxiety sensitivity (e.g., Assayag et al., 2012; Zvolensky, Stewart, et al., 2009); however, these studies did not account for the influence of pain-related anxiety. Future work should attempt to explicate the relative contributions of pain-related anxiety and anxiety sensitivity to smoking cessation outcomes.

Several study limitations should be noted, including the modest sample size. It is important that future work replicate these findings among a larger and more diverse sample of tobacco smokers (although it can be beneficial to study smaller samples during the early stages of hypothesis testing, and we took steps to ensure that the Cox proportional hazards models were adequately powered). It is also important to consider that participants volunteered to engage in an unaided quit attempt. Although an unaided quit approach confers several benefits (e.g., provides knowledge about self-quitters, limits interpretive problems related to delivering an intervention, provides a natural history of smoking cessation that can yield normative data against which treatment outcomes can be compared; e.g., Marlatt, Curry, & Gordon, 1988; Zvolensky et al., 2008), the extent to which these results may generalize to smokers receiving cessation treatment remains unclear. In addition, we did not assess chronic pain status or use of pain medications. Future work would benefit from examining these factors in relation to pain-related anxiety and smoking cessation trajectories. It is also possible that the 4-week timeframe may have been too brief to observe a relationship between pain-related anxiety and trajectory to relapse. Indeed, previous work has suggested that anxiety-related constructs (e.g., anxiety sensitivity) may place smokers at increased risk for relapse in the longer term because of repeated lapses over time (Zvolensky, Stewart, et al., 2009). Future research should examine the effects of pain-related anxiety on trajectory to relapse beyond the first 4 weeks of a quit attempt. Finally, although duration of abstinence before first lapse and relapse has been shown to predict longer-term cessation outcomes (e.g., Gilpin, Pierce, & Farkas, 1997), early lapse and relapse cannot adequately capture the dynamic process of quitting (e.g., Velicer, Prochaska, Rossi, & Snow, 1992), and future research should examine pain-related anxiety as a predictor of both prolonged and point-prevalence cessation outcomes to better account for smokers who regain abstinence after an early lapse or relapse.

Clinical research has implicated pain-related anxiety in the onset and exacerbation of pain (e.g., Boersma & Linton, 2006), and these data contribute to an emerging literature that suggests that pain-related anxiety may also play a role in the maintenance of cigarette smoking. Indeed, smokers with high pain-related anxiety may face unique cessation challenges that warrant tailored intervention, and pain-related anxiety may function as a transdiagnostic factor in the cooccurrence of pain and tobacco addiction. Treatments that incorporate psychoeducation, cognitive restructuring, and interoceptive exposure have been shown to decrease pain-related anxiety among persons with chronic pain (e.g., Watt, Stewart, Lefaivre, & Uman, 2006; Wetherell et al., 2011) and improve cessation outcomes among tobacco smokers (Fiore et al., 2008; Gifford et al., 2004). Future research should examine the efficacy of tailored treatments for smokers who present with high pain-related anxiety. Such treatments could provide psychoeducation regarding pain–smoking interrelations to aid in the development of discrepancy between continued tobacco smoking and stated goals for reducing pain and pain-related anxiety. Tailored precessation coping skills training could also help smokers to better manage their pain and pain-related anxiety during the early stages of quitting. Finally, future research would benefit from testing the utility of sequential versus integrated treatments (e.g., Ries, 1996) for pain-related anxiety and smoking cessation among smokers with cooccurring pain.

In summary, results of the current study represent an initial yet important step toward better understanding the role of pain-related anxiety in the maintenance of tobacco dependence. No previous research has examined associations between pain-related anxiety and attempts to abstain from smoking, and these findings provide the first evidence that high levels of pain-related anxiety may be associated with early lapse and relapse to smoking tobacco cigarettes. This and future work has the potential to inform the development of tailored cessation interventions for smokers who experience varying levels of pain intensity and pain-related anxiety.

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Received January 9, 2017
Revision received March 23, 2017
Accepted March 24, 2017 ■