Post-Traumatic Stress Disorder Symptoms, Underlying Affective Vulnerabilities, and Smoking for Affect Regulation

Amanda R. Mathew, PhD,1 Jessica W. Cook, PhD,2 Sandra J. Japuntich, PhD,3 Adam M. Leventhal, PhD4

1Departments of Neurosciences and Psychiatry and Behavioral Sciences, Medical University of South Carolina, Charleston, South Carolina
2University of Wisconsin-Madison School of Medicine and Public Health, Madison, Wisconsin
3National Center for PTSD at VA Boston Healthcare System, Boston, Massachusetts
4Departments of Preventive Medicine and Psychology, University of Southern California Keck School of Medicine, Los Angeles, California

Background and Objectives: Post-traumatic stress disorder (PTSD) is overrepresented among cigarette smokers. It has been hypothesized that those with PTSD smoke to alleviate negative affect and counteract deficient positive affect commonly associated with the disorder; however, limited research has examined associations between PTSD symptoms, smoking motives, and affective vulnerability factors. In the current study, we examined (1) whether PTSD symptoms were associated with positive reinforcement and negative reinforcement smoking motives; and (2) whether two affective vulnerability factors implicated in PTSD—anxiety sensitivity and anhedonia—mediated relationships between PTSD symptoms and smoking motives.

Methods: Data were drawn from a community sample of non-treatment-seeking smokers recruited without regard for trauma history (N = 342; 10+ cig/day). We used the Posttraumatic Stress Disorder Checklist-Civilian Version (PCL-C) to assess overall PTSD symptom severity as well as individual PTSD subfactors.

Results: Overall, PTSD symptom severity was significantly associated with negative reinforcement, but not positive reinforcement, smoking motives. Variation in anxiety sensitivity significantly mediated the relation between PTSD symptom severity and negative reinforcement smoking motives, whereas anhedonia did not. Regarding PTSD subfactors, emotional numbing was the only PTSD subfactor associated with smoking rate, while re-experiencing symptoms were uniquely associated with both positive reinforcement and negative reinforcement smoking motives.

Conclusions and Scientific Significance: Findings suggest that anxiety sensitivity may be an important feature associated with PTSD that enhances motivation to smoke for negative reinforcement purposes. Smoking cessation interventions that alleviate anxiety sensitivity and enhance coping with negative affect may be useful for smokers with elevated PTSD symptoms. (Am J Addict 2015;24:39–46)

INTRODUCTION

A burgeoning body of research has highlighted the strong relationship between Post-Traumatic Stress Disorder (PTSD) and cigarette smoking.1,2 Relative to those without the condition, individuals with PTSD are more likely to smoke, smoke more heavily, and have increased prevalence of nicotine dependence.1 Because smoking contributes to the significantly poorer physical health and higher use of medical care services for those with PTSD,3–5 tobacco dependence among individuals with PTSD represents an important clinical problem. However, individuals with PTSD have close to the worst quit rates in comparison with patients with other psychiatric conditions.6 Thus, it is important to examine the mechanisms that maintain smoking behavior among those with elevated PTSD symptoms in order to guide the development of more effective interventions.

Converging findings suggest that PTSD is best understood as a dimensional construct rather than as a categorical disorder.7 First, PTSD symptoms below diagnostic threshold are associated with significant functional impairment8–11 and subthreshold PTSD symptoms are associated with increased likelihood of smoking.12,13 Second, although many investigations of the PTSD-smoking relationship regard PTSD as a homogenous construct, PTSD symptoms are phenomenologically heterogeneous and may be psychometrically distinct.14–16

PTSD symptoms are characterized by extreme and often sustained psychological and biochemical stress response to
trauma exposure. In addition to experiencing PTSD symptom clusters (re-experiencing, effortful avoidance, emotional numbing, and hyperarousal), those with PTSD report high negative affect (NA) and low positive affect (PA) relative to those without the condition. Regular self-administration of nicotine among those with PTSD may serve as one strategy for regulating these affective deficits. Extant studies examining associations between PTSD symptomatology and motives for smoking primarily focus on negative reinforcement mechanisms. These results suggest that those with PTSD smoke to reduce NA to a greater extent and have higher expectations that smoking reduces NA, relative to smokers without PTSD.

Regarding the positive reinforcing effects of smoking, one study illustrated that individuals with versus without PTSD were more likely to report smoking expectancies associated with stimulation/performance enhancement, enjoyment of taste/sensorimotor manipulation aspects of smoking, and social facilitation. While these motives are relevant to positive reinforcement, they do not solely reflect affect-mediated motivation to smoke for enhancement of PA, and other studies in this population have found no relationship between smoking and PA enhancement. Hence, it remains unclear whether elevated PTSD symptoms are associated with stronger motives to smoke for PA enhancement. Clarifying this relationship is key to theoretical conceptualization, as leading models of PTSD-smoking comorbidity highlight affect-mediated positive reinforcement as a putative mechanism of PTSD-smoking co-occurrence.

Extant research has not yet addressed underlying vulnerabilities that may explain the relation between PTSD symptoms and negative reinforcement smoking motives. Additionally, further work is needed to explore an empirical link between PTSD symptoms and smoking for PA enhancement, as well as potential malleable affective vulnerabilities that may explain this relation. Herein, we explore two candidate affective vulnerability factors that may underlie PTSD-smoking relations: anxiety sensitivity and anhedonia. Anxiety sensitivity (AS; fear that anxiety-related sensations have harmful consequences) is an affective vulnerability factor that is shown to be elevated in those with PTSD. Smokers high on AS have been shown to smoke more often to manage negative moods and be less able to tolerate early withdrawal symptoms, relative to smokers low on AS. Hence, AS may underlie relations between PTSD symptoms and negative reinforcement smoking motives. Anhedonia (diminished capacity to experience pleasure) may also serve as an affective vulnerability factor. PTSD symptoms and anhedonia frequently co-occur, particularly among those with elevated levels of emotional numbing and emotional expression. Anhedonia has been associated with smoking initiation and progression, relapse following a quit attempt, and stronger urge to smoke for PA enhancement during smoking abstinence. Accordingly, if those with elevated PTSD symptoms smoke to mitigate losses in PA, anhedonia may underpin the relation between PTSD symptoms and positive reinforcement smoking. Identifying the affective vulnerabilities that may account for relations between PTSD and smoking motives may point towards psychological targets for offsetting the affective drive to smoke among individuals with elevated PTSD symptoms.

In the current cross-sectional correlational study of community-dwelling non-treatment-seeking smokers recruited without regard to trauma exposure, we sought to explore the relationship between PTSD symptom variation and smoking characteristics. Although there was no assessment of traumatic event history (Criterion A) in the current study, PTSD symptoms were assessed with the Posttraumatic Stress Disorder Checklist-Civilian Version (PCL-C). The PCL-C has been validated as a diagnostic tool to identify PTSD symptoms in research studies among those without established trauma exposure. By controlling for other emotional symptoms (ie, anxious arousal and general distress/depression), we sought to examine the unique contribution of PTSD symptoms to smoking motives. First, we sought to clarify the relationship between PTSD symptoms and smoking characteristics. As PTSD symptom dimensions may have distinct relations with smoking motives and implications for treatment, we examined each of four underlying PTSD symptom dimensions (re-experiencing, effortful avoidance, emotional numbing, and hyperarousal) as well as total PTSD symptom severity, in association with smoking-related variables. Second, we examined if PTSD symptoms were associated with smoking for positive reinforcement/pleasure enhancement and negative reinforcement/distress reduction. We expected to replicate prior work suggesting overall PTSD severity to be positively correlated with both positive reinforcement and negative reinforcement smoking. As individual PTSD symptom clusters have been examined less frequently in relation to smoking motives, these analyses were exploratory. Third, we sought to extend this work by examining whether two affective vulnerability factors implicated in PTSD—anxiety sensitivity and anhedonia—accounted for the relation between PTSD symptoms and affect-based smoking motives.

**MATERIALS AND METHODS**

**Participants**

Data for the current study were drawn from a larger lab study of acute tobacco deprivation. The sample comprised 342 non-treatment-seeking smokers with data available on all measures of interest. Inclusion criteria were: (1) >18 years old; (2) regular cigarette smoking for 2+ years; (3) currently smoking 10+ cig/day; (4) normal or corrected-to-normal vision; and (5) fluent in English. Exclusion criteria were: (1) current DSM-IV non-nicotine substance dependence; (2) current DSM-IV mood disorder or psychotic symptoms to minimize cognition-impairing effects of acute psychiatric dysfunction; (3) breath carbon monoxide (CO) levels
<10 ppm at intake; (4) use of non-cigarette forms of tobacco or nicotine products; (5) use of psychiatric medications; and (6) currently pregnant. Participants were compensated $200 for completing the study. The University of Southern California Internal Review Board approved the protocol.

**Procedure**

Data for the current study are drawn only from the baseline session at which PTSD symptoms, affective vulnerability, and smoking motives were assessed. Following a telephone screen, participants attended an in-person baseline session involving informed consent, breath CO analysis, psychiatric interview (to determine eligibility), and other measures of smoking and affect-related variables. The substance use disorders, affective disorders, and psychotic screen sections of the Structured Clinical Interview for DSM-IV Non Patient Edition (SCID-I/NP) was used to determine lifetime prevalence of key Axis I diagnoses (substance abuse/dependence, major depression, and psychotic symptoms) to determine eligibility.

**Measures**

**Fagerström Test for Nicotine Dependence (FTND)**

The FTND is the most widely used measure of nicotine dependence. The measure (possible range 0–10) consists of six items assessing smoking rate and characteristics, with higher scores indicating more severe levels of nicotine dependence.

**Posttraumatic Stress Disorder Checklist-Civilian Version (PCL-C)**

PTSD symptomatology was assessed with the PCL-C. The PCL-C is a 17-item self-report instrument that parallels diagnostic criteria for PTSD. The instructions are worded to assess symptoms associated with general depressive experiences (eg, “Felt cheerful”) and smoking to alleviate depressive symptoms. The SHAPS was found to be highly reliable in terms of both internal consistency and test-retest stability.

**Snaith Hamilton Pleasure Scale (SHAPS)**

Anhedonia was assessed with the SHAPS, a 14-item questionnaire on which participants are asked the extent to which they would hypothetically enjoy various interest/pastimes, social activities, and sensory experiences that are typically pleasant, and respond with: *Strongly Disagree, Disagree, Agree, or Strongly Agree.* Example items include, “I would enjoy being with my family or close friends.” Either of the “Disagree” responses is scored 1 point and either of the “Agree” responses is scored 0 points. Thus, the score range is 0–14, with higher scores indicating higher levels of trait anhedonia. The SHAPS was found to be highly reliable in terms of both internal consistency and test-retest stability.

**Michigan Nicotine Reinforcement Questionnaire (MRNQ)**

Smoking motives were assessed with Positive Reinforcement (PR) and Negative Reinforcement (NR) subscales of the Michigan Nicotine Reinforcement Questionnaire (MRNQ). Items are summed to create each subscale. The MRNQ-PR subscale (5 items) is associated with higher scores on novelty seeking, reward dependence, and pleasurable sensations during early experimentation with smoking. Example items include “I smoke to get a sense of euphoria or pleasure.” The MRNQ-NR subscale (8 items) is associated with craving, symptoms of nicotine withdrawal (eg, anxiety, irritability, difficulty concentrating, restlessness, depressed mood, trouble sleeping, and increased appetite), and smoking to alleviate these symptoms. Example items include “I crave a cigarette to provide relief from withdrawal.” Factor analyses of the MRNQ support distinct PR and NR factors in smoking motivation.

**Mood and Anxiety Symptom Questionnaire (MASQ)**

The MASQ is a 62-item measure assessing symptoms that commonly occur in the mood and anxiety disorders. Two subscales were used as covariates in the current analyses. The Anxious Arousal subscale comprises 17 items assessing symptoms of somatic tension and hyperarousal (eg, “Startled easily,” “Was trembling or shaking”). The General Distress/Depression subscale consists of 12 items assessing symptoms associated with general depressive symptoms (eg, “Felt like nothing was very enjoyable”) and reverse-keyed items assessing positive emotional experiences (eg, “Felt cheerful”). Each item is rated on a 1 (not at all) to 5 (extremely) Likert-type scale.

**Analytic Strategy**

All analyses were run in SAS version 9.3. Variables were tested for normality and skewed distributions were corrected with a log transformation. Models were run with standardized variables. Demographic variables of gender, race/ethnicity,
age, and level of education, and cigarettes per day were tested as potential covariates. Only age was significantly associated with PCL-C score, with older individuals reporting significantly lower PCL-C scores, \(r(339) = -0.12, p = 0.03\), so age was retained as a covariate in all regression models reported below, along with cigarettes per day, MASQ-General Distress: Depression subscale and MASQ-Anxious Arousal subscale. First, we assessed the relationship between PTSD symptoms and smoking characteristics. Separate models were run with both overall PTSD severity score and each of 4 PTSD symptom clusters as independent variables. Second, we examined PTSD symptoms as a predictor of affective smoking motives. A series of linear regression models were run to examine the following predictors: (1) PTSD severity score; (2) each of the four PTSD symptom clusters individually; and (3) all four PTSD symptom clusters simultaneously in a combined model. Separate models were run for each outcome variable: (1) positive reinforcement (PR); and (2) negative reinforcement (NR) smoking motives outcomes. Thus, a total of 12 linear regression models were specified. Results are reported as standardized regression weights (\(\beta\)). Third, we examined whether anxiety sensitivity and anhedonia mediated significant relationships between PTSD symptoms and smoking motives. Separate models were run to test each proposed mediator using a products of coefficients approach. Results were considered statistically significant if confidence intervals for the meditational effect did not include zero.

Results

Sample Characteristics

The sample comprised 342 participants (110 women) with average age of 43.7 (SD = 10.8) years. Participants were 51.2% Black, 30.1% White, and 18.7% Multiracial/Other. Descriptives and intercorrelations between study variables are presented in Table 1. PCL-C scores ranged from 17 to 71 with a mean of 25.8 (SD = 10.6). Roughly one-fifth of participants endorsed the minimum PCL-C score of 17 (\(n = 69\); 20.2%), indicating absence of PTSD symptoms. Using the suggested PTSD clinical cut-off score for a community, non-military sample (PCL-C \(> 44\)), 6.7% of the sample (23/342) met criteria for PTSD. Intercorrelations between PCL-C subfactors illustrated that, as expected, re-experiencing, effortful avoidance, emotional numbing, and hyperarousal were all positively correlated (r ranged from .58 to .72, \(p < .001\)).

PTSD Symptoms and Smoking Characteristics

PCL-C total score was not significantly associated with smoking variables including cigarettes per day (\(\beta = .07, p = .25\)), FTND score (\(\beta = .06, p = .32\)), and time to first cigarette (\(\beta = -.01, p = .98\)). PCL-C emotional numbing subfactor was positively associated with cigarettes per day (\(\beta = .13, p = .02\)). Associations between other PCL-C subfactors and smoking variables were not significant.

### Table 1. Means, standard deviations, correlations, and internal consistencies (Cronbach’s alphas, in parentheses) of study variables

<table>
<thead>
<tr>
<th>M (SD)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCL-C-total</td>
<td>25.84 (10.55)</td>
<td>.62 (62)</td>
<td>.69** (69)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCL-C-re-experiencing</td>
<td>7.40 (3.32)</td>
<td>.79 (79)</td>
<td>.61** (61)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCL-C-avoidance</td>
<td>3.52 (2.02)</td>
<td>.77** (77)</td>
<td>.06 (06)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCL-C-Emot. numbing</td>
<td>7.16 (3.40)</td>
<td>.75 (75)</td>
<td>.58** (58)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCL-C-hyperarousal</td>
<td>7.75 (3.47)</td>
<td>.67** (67)</td>
<td>.40 (40)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASI</td>
<td>20.52 (13.03)</td>
<td>.66** (66)</td>
<td>.28** (28)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SHAPS</td>
<td>95 (13.72)</td>
<td>.72** (72)</td>
<td>.48** (48)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MASQ-AAA</td>
<td>2.54 (1.94)</td>
<td>.90** (90)</td>
<td>.14** (14)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MESQ-GD</td>
<td>17.64 (7.10)</td>
<td>.09 (09)</td>
<td>.07 (07)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FTND</td>
<td>7.94 (3.22)</td>
<td>.03 (03)</td>
<td>.01 (01)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cigarettes per day</td>
<td>6.06 (1.94)</td>
<td>.01 (01)</td>
<td>.08 (08)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MNRQ-PR</td>
<td>7.94 (3.22)</td>
<td>.03 (03)</td>
<td>.14 (14)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MNRQ-NR</td>
<td>9.80 (4.76)</td>
<td>.29 (29)</td>
<td>.32 (32)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Columns and rows, indicated by 1–13 show the Pearson correlation between items in those rows and columns. M = means; SD = standard deviation; PCL-C = posttraumatic stress disorder checklist-civilian version; ASI = anxiety sensitivity index; SHAPS = Snaith Hamilton pleasure scale; MASQ = mood and anxiety symptom questionnaire-anxious arousal (AA) and general distress: depression (GD) subscales; FTND = Fagerström test for nicotine dependence; MNRQ = michigan nicotine reinforcement questionnaire-positive reinforcement (PR) and negative reinforcement (NR) subscales.

* \(p < .05\). ** \(p < .01\).
PTSD Symptoms on Smoking Motives

After adjusting for covariates, PCL-C total score was significantly associated with negative reinforcement smoking motives, but not positive reinforcement smoking motives (See Table 2). We next examined associations between PCL-C subfactors and smoking motives. Only PCL-C re-experiencing subfactor was associated with positive reinforcement smoking motives ($\beta = .29, p = .001$). In single predictor analyses, each PCL-C subfactor was significantly associated with negative reinforcement smoking motives ($\beta$ ranged from .19 to .30, $p < .001$). With all PCL-C subfactors included in the model, only the re-experiencing subfactor remained significantly associated with negative reinforcement smoking motives ($\beta = .26, p = .003$).

Mediating Role of Anhedonia and Anxiety Sensitivity

Next, we tested for a possible mediating effect of anxiety sensitivity on significant relationships between PCL-C total score, PCL-C re-experiencing subfactor, and smoking motives, respectively. First, PCL-C total score was significantly associated with ASI ($\beta = .60, p < .0001$). ASI was significantly associated with negative reinforcement smoking motives ($\beta = .20, p < .001$). Variation in anxiety sensitivity significantly mediated the relation between PCL-C total score and negative reinforcement smoking motives ($\mu = .12; 95\% \text{ CI} .04-.20$), such that higher levels of AS were associated with greater endorsement of negative reinforcement motives. Second, PCL-C re-experiencing subfactor was significantly associated with ASI ($\beta = .39, p < .0001$). Variation in anxiety sensitivity significantly mediated the relation between PCL-C re-experiencing subfactor and negative reinforcement smoking motives ($\mu = .08; 95\% \text{ CI} .03-.13$), such that higher levels of AS were associated with greater endorsement of negative reinforcement motives. Lastly, the ASI was not associated with positive reinforcement smoking motives, and was not shown to significantly mediate the relation between PCL-C re-experiencing subfactor and positive reinforcement smoking motives.

The SHAPS was significantly associated with both PCL-C total score ($\beta = .20, p < .001$) and PCL-C re-experiencing subfactor ($\beta = .20, p < .001$). However, SHAPS was uncorrelated with either positive reinforcement or negative reinforcement smoking motives, and was not a significant mediator of the relation between PTSD symptoms and smoking motives.

**DISCUSSION**

Results of the current study are consistent with previous research on PTSD symptoms and smoking motives, and suggest several unique factors that may help account for this relationship. First, PTSD symptom severity was significantly associated with negative reinforcement smoking motives. Second, this relationship was significantly mediated by levels of anxiety sensitivity, but not anhedonia. Third, interesting findings emerged with regard to the relationship between PTSD subfactors and smoking. While emotional numbing was the only PTSD subfactor associated with smoking rate, re-experiencing symptoms were uniquely associated with both positive reinforcement and negative reinforcement smoking motives. Results were robust even after controlling for other emotional symptoms (ie, anxious arousal and general distress/depression), suggesting a unique contribution of PTSD symptoms.

The current study identifies one candidate mechanism, anxiety sensitivity, which may be a shared vulnerability factor between PTSD symptoms and smoking. Anxiety sensitivity has been robustly related to motivation to smoke for negative reinforcement purposes.\(^{28,56,57}\) Anxiety sensitivity may be particularly relevant to smokers with PTSD, who endorse elevated smoking withdrawal symptoms in the early stage of abstinence.\(^{58-60}\) As such, withdrawal symptoms among those with PTSD may be interpreted as especially catastrophic, motivating smoking as means of escaping withdrawal.

Re-experiencing symptoms were shown to be uniquely related to both positive reinforcement and negative reinforcement smoking motives in the current study, even after

**TABLE 2.** Associations of PCL-C total score and subfactors to smoking motives

<table>
<thead>
<tr>
<th></th>
<th>PCL-C total score</th>
<th>Re-experiencing</th>
<th>Emotional numbing</th>
<th>Avoidance</th>
<th>Hyperarousal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Indiv. model(^a)</td>
<td>Indiv. model(^a)</td>
<td>Comb. model(^b)</td>
<td>Comb. model(^b)</td>
<td>Indiv. model(^a)</td>
</tr>
<tr>
<td>NR motives ($\beta$)</td>
<td>.28**</td>
<td>.29**</td>
<td>.26**</td>
<td>.06</td>
<td>-.12</td>
</tr>
<tr>
<td>PR motives ($\beta$)</td>
<td>.10</td>
<td>.21**</td>
<td>.29**</td>
<td>-.03</td>
<td>-.12</td>
</tr>
</tbody>
</table>

\(\beta = \text{standardized } \beta\)-weights; PCL-C = posttraumatic stress disorder checklist-civilian version; NR and PR motives = michigan nicotine reinforcement questionnaire-negative reinforcement and positive reinforcement subscales.

\(^a\)Individual model includes the respective PTSD variable as the sole predictor.

\(^b\)Combined model includes all four PTSD subfactors as simultaneous predictors.

\(^c\)Models include primary predictor(s) after adjusting for age, cigarettes per day, MASQ-Anxious Arousal subscale, and MASQ-General Distress: Depression subscale.

\(*p < .05\).

\(**p < .01\).

Mathew et al. January 2015 43
covarying for general depressive and anxiety symptoms. Further, the relationship between re-experiencing symptoms and NR motives was significantly mediated by anxiety sensitivity. Though PCL-C subscales in the current sample were highly correlated, the re-experiencing symptom subscale evidenced a unique relationship with smoking motives, perhaps due to specific content of the items (ie, intrusive thoughts and nightmares about a traumatic event) that best characterized impairment consistent with PTSD. Those elevated on this symptom cluster may have strong motivation to smoke in the context of cues associated with intrusive recollections of trauma. This is consistent with previous research, which found that exposure to trauma-related stimuli is associated with intensified negative affect and cigarette craving among both PTSD and non-PTSD smokers and may serve as a cue for smoking behavior. Additionally, findings suggest that re-experiencing symptoms operate in part through elevated anxiety sensitivity in conferring specific risk for smoking. Though previous research has linked re-experiencing symptoms and AS, particularly the somatic concerns dimension, other studies have failed to support this association, highlighting the need for further exploration in clinical samples of smokers with PTSD.

Given established relationships between PTSD symptoms, low PA, and smoking for PA enhancement, findings were unexpected with regard to positive reinforcement smoking motives and anhedonia. First, positive reinforcement smoking motives were associated only with re-experiencing symptoms, but not with overall PTSD symptoms or other symptom clusters. The specificity of this finding serves to further highlight the unique relations between re-experiencing symptoms and affective smoking motives. Second, anhedonia was not shown to significantly mediate the relationship between PTSD symptoms and smoking. Although symptoms of anhedonia and PTSD are conceptually similar, anhedonia is thought to reflect a broad dimension of diminished pleasure response to rewards, while PTSD symptoms consist of loss of interest and blunted PA, but not necessarily diminished pleasure response. Thus, anhedonia may represent a distinct construct that relates to smoking through a separate pathway from PTSD symptoms.

Additionally, it was unexpected that no relationship between overall PTSD symptom severity and smoking variables was detected, as this has been found in previous research. This may reflect discrepancies in study methodology, including lower overall PTSD symptom severity. Although research suggests that most individuals in the general population encounter at least one traumatic event in their lives, participants in the current study were recruited without regard for history of trauma exposure. However, current findings replicated the unique association between emotional numbing symptoms and smoking rate observed in previous studies. As emotional numbing symptoms were not associated with affective smoking motives, it may be that this symptom cluster relates to smoking behavior through alternate motives that were not assessed in the current study, such as automaticity, environmental influences, or improved cognition.

Findings of the current study have several implications for tailoring effective smoking cessation treatment to those with elevated PTSD symptoms. Smoking cessation interventions that alleviate anxiety sensitivity and enhance coping may be useful for smokers with elevated PTSD symptoms, particularly those with significant interference due to re-experiencing symptoms. Brief interventions that incorporate psychoeducation and interoceptive exposure to arousal-related bodily sensations are shown to result in substantial reductions in anxiety sensitivity as well as PTSD symptoms. In particular, these skills may be critical early in the course of a quit attempt, as smokers with PTSD are shown to lapse earlier than those without PTSD. Importantly, smokers with PTSD endorsed negative affect and trauma reminders as lapse causes, highlighting both anxiety sensitivity and re-experiencing symptoms as potential targets for intervention among this population. The current study was limited by use of a community sample, cross-sectional design, and reliance on a single method of assessment. Participants were recruited without regard to trauma history; thus, we were unable to select participants based on trauma exposure, and about one-fifth of participants indicated absence of any symptoms consistent with PTSD. Measurements of PTSD and other symptoms were based on self-report instruments rather than clinician-administered structured interviews. One strength of this approach is a focus on behavioral domains relevant to PTSD psychopathology as opposed to clinical diagnosis, consistent with similar decisions in the NIMH’s Research Domain Criteria (RDoC) initiative. However, the current assessment strategy is unable to establish whether reported PTSD symptoms truly resulted from a traumatic or stressful event. Thus, it will be important for future research to cross-validate findings in clinical populations of smokers with PTSD and supplement self-report measures with clinician interviews, behavioral tasks, or other methods of assessment. Additionally, the cross-sectional design employed in the present study precludes our ability to identify causal or temporal relationships, limiting our interpretation to the observation that PTSD symptoms, anxiety sensitivity, and negative reinforcement smoking motives tend to co-occur and are not better accounted for by general depressive or anxiety symptoms. Although anxiety sensitivity and anhedonia were considered as potential mediators in the current study, these constructs are not an exhaustive list of candidates. Other potential mechanisms relevant to both PTSD and smoking that may be considered in future research include attention, distress tolerance, and self-efficacy. Additionally, as it is not yet known if these mechanisms predict quit rates in smokers with PTSD, studies are needed to test the mediating role of anxiety sensitivity on the relationship between PTSD and cessation success.

In sum, PTSD symptoms were associated with negative reinforcement smoking motives, and this relationship was
significantly mediated by levels of anxiety sensitivity. Unique findings emerged with regard to emotional numbing symptoms and re-experiencing symptoms in accounting for co-occurrence of PTSD symptoms and smoking. Findings add to research literature on PTSD symptomatology and smoking by highlighting the important role of anxiety sensitivity in accounting for PTSD-smoking comorbidity.

This work was supported by grants R01-DA026831 and K08-DA025041 to Dr. Leventhal from the National Institute on Drug Abuse, Bethesda, MD. Dr. Mathew is supported by National Institute on Drug Abuse grant T32 DA007288. This work was supported in part by Career Development Award (Japuntich) # 1IK2CX000918-01AI from the United States Department of Veterans Affairs Clinical Sciences Research and Development Service. The contents do not represent the views of the Department of Veterans Affairs or the United States Government.

REFERENCES


