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Anxiety sensitivity mediates the association between post-traumatic stress symptom severity and interoceptive threat-related smoking abstinence expectancies among World Trade Center disaster-exposed smokers

Samantha G. Farris [a,*], Daniel J. Paulus [a], Adam Gonzalez [b], Brittain L. Mahaffey [b], Evelyn J. Bromet [b], Benjamin J. Luft [c], Roman Kotov [b], Michael J. Zvolensky [a,d]

[a] University of Houston, Department of Psychology, Houston, TX 77024, United States
[b] Stony Brook University, Department of Psychiatry, Stony Brook, NY 11794, United States
[c] Stony Brook University, Department of Medicine, Stony Brook, NY 11794, United States
[d] The University of Texas MD Anderson Cancer Center, Department of Behavioral Science, Houston, TX 77030, United States

HIGHLIGHTS

- PTSD symptom severity is related to difficulties with smoking cessation.
- Anxiety sensitivity is a cognitive-affective risk variable that may link PTSD and smoking.
- PTSD symptom severity was associated with threat-related smoking abstinence expectancies.
- Anxiety sensitivity was indirectly associated with the PTSD-smoking abstinence expectancies link.

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ABSTRACT

Introduction: Anxiety sensitivity (fear of internal anxiety-relevant bodily sensations) is an individual difference variable that is associated with the development and maintenance of posttraumatic stress disorder (PTSD) and is also involved in the maintenance/relapse of smoking. Abstinence expectancies are crucial to smoking maintenance, yet, past work has not explored how PTSD symptom severity and anxiety sensitivity contribute to them.
Method: Participants were 122 treatment-seeking daily smokers (36.1% female; $M_{age} = 49.2$, $SD = 9.7$; cigarettes per day: $M = 18.3$, $SD = 15.2$) who were exposed to the World Trade Center disaster on September 11, 2001 and responded to an advertisement for a clinical smoking cessation trial. The indirect effect of anxiety sensitivity was tested in terms of the effect of PTSD symptom severity on smoking abstinence expectancies (*i.e.*, anxiety sensitivity as a statistical mediator).
Results: PTSD symptom severity was positively associated with interoceptive threat-related smoking abstinence expectancies: expecting harmful consequences ($\beta = .33$, $p < .001$) and somatic symptoms ($\beta = .26$, $p = .007$). PTSD symptom severity was also significantly associated with anxiety sensitivity ($\beta = .27$, $p = .003$). Anxiety sensitivity mediated the association between PTSD symptom severity and expectancies about the harmful consequences ($\beta = .09$, $CI_{95\%} = .02–.21$; $\Delta R^2 = .076$) and somatic symptoms ($\beta = .11$, $CI_{95\%} = .02–.24$; $\Delta R^2 = .123$) from smoking abstinence, with medium effect sizes ($K^2 = .08$ and .10, respectively).
Conclusions: These data document the role of PTSD symptoms in threat-based expectancies about smoking abstinence and suggest anxiety sensitivity may underlie the associations between PTSD symptom severity and abstinence expectancies.

1. Introduction

Exposure to potentially traumatic events, with or without the development of posttraumatic stress disorder [PTSD] is associated with the maintenance of cigarette smoking (see reviews by Feldner, Babson, Zvolensky, Vujanovic, et al., 2007; Fu et al., 2007). For example, rates

* Corresponding author at: The University of Houston, Department of Psychology, 126 Fred J. Heyne Building, Suite 104, Houston, Texas 77204-5502, United States.
E-mail address: sgfarris@uh.edu (S.G. Farris).

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of smoking are significantly higher among trauma-exposed individuals and those with elevated PTSD symptoms (with and without clinical levels of PTSD) relative to those without psychopathology or other anxiety disorders (*e.g.*, Koenen et al., 2005; Pietrzak, Goldstein, Southwick, & Grant, 2011). Trauma-exposure and elevated PTSD symptoms are also associated with heavier smoking (Hapke et al., 2005), less success in quitting smoking (Beckham, Calhoun, Dennis, Wilson, & Dedert, 2013; Zvolensky, Yartz, Gregor, Gonzalez, & Bernstein, 2008), as well as smoking in response to negative affect, PTSD cues, smoking craving, or restlessness (Beckham et al., 2005). Thus, smoking in trauma-exposed individuals is thought to be motivated at least in part by avoidance-based affect regulatory smoking motives (Farris, Zvolensky, Beckham, Vujanovic, & Schmidt, 2014) and expectancies about the negative-affect reduction properties of cigarettes (Calhoun, Levin, Dedert, Johnson, & Beckham, 2011; Carmody et al., 2012). These cognitive processes may help to explain reliance on cigarettes, low self-efficacy for being abstinent, and perceived (or actual) cessation difficulties (Carmody et al., 2012; Farris, Langdon, DiBello, & Zvolensky, 2015).

One cognitive vulnerability process that may underlie the association between PTSD symptom severity and smoking is anxiety sensitivity, defined as the tendency to catastrophize and misinterpret the meaning of anxiety-relevant interoceptive sensations (Reiss, Peterson, Gursky, & McNally, 1986). Anxiety sensitivity is a relatively stable, yet malleable, multidimensional construct that consists of concerns about the physical, social, and cognitive consequences of anxiety-related physiological sensations (Taylor et al., 2007). Anxiety sensitivity is theoretically implicated and empirically associated with the development and maintenance of anxiety symptoms and disorders, including PTSD (see meta analyses by Naragon-Gainey, 2010; Olatunji & Wolitzky-Taylor, 2009).

Trauma-exposed individuals report higher levels of anxiety sensitivity relative to those not exposed to trauma (*e.g.*, Lang, Kennedy, & Stein, 2002), which is related to greater PTSD symptom severity (G. N. Marshall, Miles, & Stewart, 2010). Anxiety sensitivity is also associated with various aspects of cigarette smoking, including cognitive-based processes (*e.g.*, affect-regulatory smoking motives, smoking outcomes expectancies, and perceived barriers to smoking cessation; Farris, Leventhal, Schmidt, & Zvolensky, 2015; Johnson, Farris, Schmidt, Smits, & Zvolensky, 2013; Zvolensky et al., 2007; Zvolensky, Farris, Schmidt, & Smits, 2014). Initial scholarly work has explored the nature of anxiety sensitivity in terms of the interplay between PTSD symptoms and smoking, primarily in trauma-exposed community smokers (Farris, Vujanovic, Hogan, Schmidt, & Zvolensky, 2014; Feldner, Vujanovic, Gibson, & Zvolensky, 2008; Marshall et al., 2008; Vujanovic, Marshall, Gibson, & Zvolensky, 2010) and veteran smokers with combat-related PTSD (Van Voorhees et al., 2013), generally suggesting that among smokers, anxiety sensitivity is related to the amplification of PTSD symptom severity (*e.g.*, Farris, Vujanovic, et al., 2014; Feldner, Vujanovic, et al., 2008), and smoking to reduce negative affect (Cook, McFall, Calhoun, & Beckham, 2007).

Past work has not yet explored the nature of the associations between PTSD symptom severity and expectancies about *smoking abstinence* in general, or the possible role of anxiety sensitivity in these relations. In contrast to expectancies about the outcome effects of smoking, smoking abstinence expectancies are beliefs about the acute effects of nicotine abstinence (*i.e.*, short-term psychological and physiological consequences from abstaining from smoking; Abrams, Zvolensky, Dorman, Gonzalez, & Mayer, 2011). Past work suggests negative smoking abstinence expectancies are associated with greater nicotine dependence and subjective withdrawal symptoms (Abrams et al., 2011; Hendricks & Leventhal, 2013). Moreover, expecting harmful consequences from acute abstinence is associated with lower likelihood of quitting for at least a 24 h period (Abrams et al., 2011). Anxiety sensitivity is related to 'interoceptive threat' smoking abstinence expectancies (*i.e.*, expectancies about somatic or harmful consequences from smoking abstinence; Abrams et al., 2011; Farris, Langdon, et al., 2015) and importantly, has been conceptualized as a cognitively-based construct specific to interpretations of anxious states, which is distinct from smoking abstinence expectancies, a cognitive construct composed of specific beliefs about the expected reactions to smoking deprivation. Thus, while these two constructs have expectancy processes in common, they are related, but distinct, in their origins (Cox, Klinger, & Fadardi, 2015).

Given fallible threat perception and development of erroneous cognitions/attributions are both contributing factors in the onset and maintenance of PTSD symptoms (*e.g.*, "The world is a dangerous place"; Ehlers & Clark, 2000; Foa & Kozak, 1986), smokers with PTSD symptoms may be apt to hold catastrophic expectancies about the consequences of acute smoking abstinence (*e.g.*, "I would feel dizzy or tense if I could not smoke for 24 h"). Additionally, it is possible that anxiety sensitivity, as a cognitive process related to both PTSD symptoms and smoking abstinence expectancies, may indirectly explain the associations. That is, among trauma-exposed smokers, more severe PTSD symptoms may be related to catastrophic interpretation about anxiety-relevant interoceptive sensations (Marshall et al., 2010), which in turn may be related to interoceptive threat smoking abstinence expectancies (Farris, Langdon, et al., 2015).

The current study aimed to examine these associations among a sample of treatment-seeking smokers who were exposed to the terrorist attack on the World Trade Center (WTC) on September 11, 2001 (9/11). A range of physical and mental health conditions have been documented among those exposed to the disaster — with particular focus on PTSD (Brackbill et al., 2009; Liu, Tarigan, Bromet, & Kim, 2014). It was hypothesized that greater PTSD symptom severity would be directly associated with expecting interoceptive threat from smoking abstinence (somatic symptoms and harmful consequences), which would be explained (mediated) by anxiety sensitivity. These associations were expected to be evident after adjusting for gender, level of nicotine dependence, and neuroticism.

2. Material and methods

2.1. Participants

Participants were adult daily smokers who were screened for possible inclusion in a smoking cessation treatment program for individuals exposed to the 9/11 WTC disaster. Potential participants were recruited from the WTC Health Program, the New York City Department of Health WTC Health Registry, as well as local newspapers and Craigslist—New York. Inclusion criteria included (1) smoking at least five cigarettes per day, (2) interest in smoking cessation treatment, (3) direct exposure to the WTC disaster (*e.g.*, responding to the event or witnessing the event in person), and (4) scoring at least in the intermediate range (30 or greater; Andrykowski, Cordova, Studts, & Miller, 1998) on the Post-traumatic Stress Disorder Checklist (PCL-S; Weathers, Litz, Herman, Huska, & Keane, 1993). Participants were ineligible for the study if they were currently engaged in other smoking cessation treatment or were suffering from serious psychological disorders (*e.g.*, psychosis, mania) or alcohol/substance dependence. For the current analyses, 25 individuals were excluded due to incomplete data on the measures of interest, yielding 122 smokers for the current study. The sample was mostly (63.9%) male, with an average age of 49.2 (SD = 9.7) years. Regarding race, 56.6% identified as White, 31.1% Black/African American, 0.8% American Indian/Alaskan Native, 0.8% Asian, 3.3% selected "other/multi-racial" and 7.4% did not disclose race; 15.6% identified ethnicity as Hispanic. Of note, 41.0% of the sample met diagnostic criteria for current PTSD.

2.2. Measures

2.2.1. Structured Clinical Interview for DSM-IV Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 2007)

The SCID-I is a clinician-administered diagnostic assessment used to assess the presence of psychopathology. The SCID-I was administered

by a trained clinical psychologist. All assessments were reviewed for rater agreement; no cases of disagreement were noted. A dichotomous variable was created to reflect those who met criteria for 9/11-related PTSD in the past 12-months (0 = absence of PTSD; 1 = presence of PTSD).

2.2.2. Posttraumatic Stress Disorder Checklist, Specific Version (PCL-S; Weathers et al., 1993)

The PCL-S is a self-report measure of PTSD symptom severity. It consists of 17 items, experienced within the past month, which correspond to DSM-IV-TR (American Psychiatric Association, 2000) PTSD criteria. Each item is rated from 1 (*not at all*) to 5 (*extremely*) and summed to create a total score. For the current study, instructions were tailored to capture symptoms "in relation to 9/11." The PCL-S is a reliable measure that correlates well with clinician-administered PTSD interview (Keen, Kutter, Niles, & Krinsley, 2008). For the current sample, internal consistency of total PCL-S scores was excellent ($\alpha = .92$).

2.2.3. Anxiety Sensitivity Index-3 (ASI-3; Taylor et al., 2007)

The ASI-3 is an 18-item self-report measure of anxiety sensitivity, a relatively stable tendency to fear sensations associated with anxiety (Reiss & McNally, 1985). Items are rated on a scale from 0 (*very little*) to 4 (*very much*); items can be summed to derive a total score or can be scored to compute three sub-factors (physical concerns, cognitive concerns, social concerns; Taylor et al., 2007). The ASI-3 measure has well-documented psychometric properties (Taylor et al., 2007) and has been validated in treatment-seeking smokers (Farris et al., 2015). Due to clerical error, item 14 of the ASI-3 was not administered. Average scores for each participant on the cognitive scale were used in place of this item. In the current sample, internal consistency for the full scale was excellent ($\alpha = .92$) and was good for each of the sub-factors: ASI-3 physical ($\alpha = .88$), ASI-3 cognitive ($\alpha = .89$), and ASI-3 social ($\alpha = .81$).

2.2.4. Smoking Abstinence Expectancies Questionnaire (SAEQ; Abrams et al., 2011)

The SAEQ is a 28-item self-report measure of expected consequences as a result of 24-h of smoking abstinence. Items are rated in terms of expected likelihood on a scale from 0 (*very unlikely*) to 6 (*very likely*). The SAEQ yields a psychometrically sound full-scale score in addition to four internally consistent subscales that index abstinence expectancies related to harmful consequences, somatic symptoms, negative mood, and positive consequences. Items are summed for each subscale, which can also be summed together for a total score (with the positive consequences subscale reverse-scored). In the current sample, internal consistency was good for the full scale ($\alpha = .88$) and acceptable to good for all subscale items; the harmful consequences (*e.g.*, I would feel like I'm dying; $\alpha = .84$) and somatic symptoms (*e.g.*, My throat would feel dry; $\alpha = .76$) subscales were used in the current study.

2.2.5. Fagerström Test for Nicotine Dependence (FTND; Fagerström, 1978)

The FTND is a 6-item self-report measure of nicotine dependence severity yielding a total score between 0–10. It has shown strong relations with other smoking variables (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991). The internal consistency of the FTND items in the current sample was relatively low ($\alpha = .45$), which is an issue often apparent with this measure (Korte, Capron, Zvolensky, & Schmidt, 2013).

2.2.6. Big Five Inventory (BFI; John, Donahue, & Kentle, 1991)

The BFI is a 44-item self-report measure of five personality dimensions: openness to experience, conscientiousness, extraversion, agreeableness, and neuroticism. Here, the neuroticism subscale (BFI-N) was used, which consists of 8 statements (*e.g.*, "I see myself as someone who can be tense") rated from 1 (*disagree strongly*) to 5 (*agree strongly*), summed (three items are reversed-scored) together. All BFI scales have been shown to have excellent reliability as well as convergent and discriminant validity (Benet-Martínez & John, 1998; John & Srivastava, 1999). BFI-N items had acceptable internal consistency in the current sample ($\alpha = .73$).

2.3. Procedures

Respondents completed a telephone prescreen to determine potential study eligibility. Potentially eligible participants were invited to an in-person baseline screening assessment that included a clinician-administered diagnostic assessment of psychopathology (per the SCID-I), self-report assessments (including the PCL-S, ASI-3, and SAEQ), and biochemical verification of smoking *via* expired carbon monoxide breath samples. All participants provided written informed consent prior to initiation of any study procedures. All participants, whether eligible for the treatment trial or not, were compensated $50 for completing baseline procedures. The study was approved by the Stony Brook University Institutional Review Board.

2.4. Data analytic strategy

Preliminary descriptive analyses were conducted to examine the bivariate associations (zero order Pearson's correlation) between study variables. Primary analyses included two regression-based path models to examine the mediating role of anxiety sensitivity in the association between PTSD symptom severity and two types of interoceptive threat-related smoking abstinence expectancies: harmful consequences and somatic symptoms. See Fig. 1 for conceptual model. In both models, covariates included gender, neuroticism, and level of nicotine dependence. Additional models were constructed to test the unique mediating role of the anxiety sensitivity sub-factors. The statistical strategy utilized (Hayes, 2009; Preacher & Hayes, 2004) allows for estimation and significance testing of the total and specific indirect (mediation) effects through bootstrapping. Bootstrapping generates an empirical representation of the sampling distribution of the indirect effect, from which a confidence interval can be generated (Hayes, 2009). The current analytic approach allows for examination of multiple explanatory variables (e.g., ASI-3 sub-factors), in a causal or sequential fashion, while simultaneously testing the indirect effects of each independently (Hayes, 2013). That is, the model estimates each specific indirect effect (*e.g.*, path $a_1 * b_1$, path $a_2 * b_2$, path $a_3 * b_3$) and tests for statistical

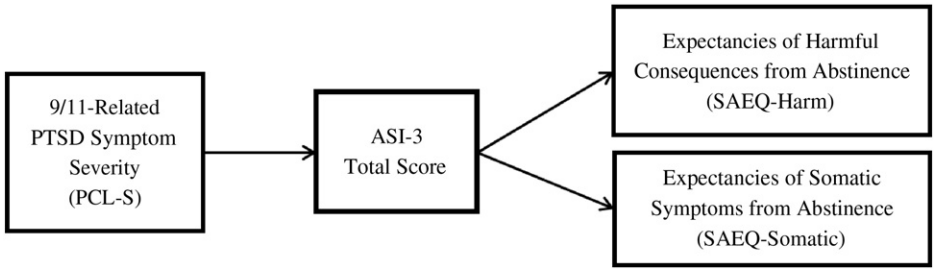

Fig. 1. Conceptual model. Note: Each criterion outcome was tested in a separate model. All models adjusted for gender, level of nicotine dependence, and neuroticism.

significance, while also estimating the total indirect effect and statistical significance.

Analyses were conducted using PROCESS, a conditional process modeling program that utilizes an ordinary least squares-based path analytical framework to test for both direct and indirect effects (Hayes, 2013). Model 4 was specified. The 95-percentile confidence intervals (CI) for beta indices were obtained analytically; the CIs for the specific and conditional indirect effects were estimated with bootstrap analyses (10,000 resamples) as recommended (Hayes, 2009; Preacher & Hayes, 2004, 2008). Effect sizes (K^2) were calculated for each indirect effect as per recommendations of Preacher and Kelley (2011) as the squared ratio of the obtained indirect effect to the largest possible indirect effect that could have been obtained. This value is interpreted in a similar regard as squared correlation coefficients, as outlined by Cohen (1988).

Additionally, two post-hoc models were conducted to examine if the presence/absence of PTSD conditionally impacted the mediating effect of anxiety sensitivity on the association between PTSD symptom severity and abstinence expectancies. PROCESS model 7 was specified, which allows for the test of the conditional (moderating) effect of a variable (w) on path a ($x \rightarrow m$), and the conditional mediating (indirect) effect (Hayes, 2015). That is, this model tested whether mediating role of anxiety sensitivity (linking PTSD symptom severity to abstinence expectancies) was impacted by the presence/absence of PTSD.

3. Results

Descriptive characteristics of the sample are presented in Table 1. Average PCL-S scores were 46.8 ($SD = 14.5$), indicative of severe PTSD symptoms. In terms of anxiety sensitivity, average scores on the ASI-3 were 22.8 ($SD = 15.8$), which is consistent with scores reported in clinical samples of smokers with PTSD (Asnaani, Farris, Carpenter, Zandberg, & Foa, 2015). Bivariate associations between variables are presented in Table 2. PTSD symptom severity was positively associated with neuroticism, anxiety sensitivity, level of nicotine dependence, and both abstinence outcome expectancies. Anxiety sensitivity was also positively associated with neuroticism and both abstinence outcome expectancies, but not significantly associated with level of nicotine dependence. The abstinence outcome expectancies were significantly inter-correlated.

3.1. Test of the mediating role of anxiety sensitivity

Two separate analyses were conducted (see Table 3 for standardized regression coefficients and indirect effects) in order to test anxiety sensitivity as a mediator of the effect of PTSD symptom severity on harmful consequences and somatic symptom expectancies of smoking abstinence. First, PTSD symptom severity was significantly and positively associated with harmful consequence and somatic symptom expectancies of smoking abstinence (p's < .01; path c effects). PTSD symptom severity was significantly and positively associated with anxiety sensitivity ($p = .004$; path a). Anxiety sensitivity was also significantly positively associated with both harmful consequence and somatic symptom expectancies of smoking abstinence (p's < .001; path b); anxiety sensitivity accounted for an additional 7.6% and 12.3% variance in outcomes (harmful consequences and somatic symptom expectancies, respectively). In the test of the indirect effects, anxiety sensitivity emerged as a significant mediator of the association between PTSD symptom severity and harmful consequence and somatic symptom expectancies. The effect size for the indirect effect was medium for both models ($K^2 = .08$ and .10, respectively).

Table 2
Bivariate correlations (n = 122).

Variable	2.	3.	4.	5.	6.	7.
1. Gender (female)	.08	−.07	.07	−.02	.07	.06
2. Neuroticism	–	.07	.37**	.44**	.04	.25**
3. Nicotine dependence		–	.20*	.13	.15	.28**
4. PTSD symptom severity			–	.39**	.26**	.40**
5. Anxiety sensitivity				–	.39**	.44**
6. Harmful consequences expectancies					–	.72**
7. Somatic symptom expectancies						–

Note: Neuroticism (per BFI-Neuroticism scale); Nicotine dependence (per FTND); PTSD symptom severity (9/11-related PTSD symptoms per the PCL-S); Anxiety sensitivity (per ASI-3 total score); harmful consequences expectancies (per SAEQ-Harmful consequences subscale); somatic symptom expectancies (SAEQ — Somatic subscale). Numbers across header correspond with variables numbered 2–7.

* p < .05.
** p < .01.

3.2. Alternative test of mediation

To increase specificity in meditational effect, all three sub-factors of anxiety sensitivity (ASI-3 Physical Concerns, Cognitive Concerns, and Social Concerns) were entered as simultaneous mediators into the model presented above. Results indicated that none of the associations were significant between the anxiety sensitivity sub-factors and smoking abstinence expectancies about harmful consequences or somatic symptoms (*i.e.*, lack of path b effects). However, ASI-3 Physical concerns emerged as a unique mediator (indirect predictor) of the association between PTSD symptom severity and expectancies about

Table 1
Participant characteristics.

Variable	Full sample (n = 122)	Smokers with PTSD (n = 50)	Smokers w/o PTSD (n = 72)	t/x^2
	M/n (SD/%)	M/n (SD/%)	M/n (SD/%)	
Gender (female)	44 (36.1%)	21 (42.0%)	23 (31.9%)	1.29
Age	49.2 (9.7)	49.5 (9.8)	48.7 (9.7)	−0.47
Age initiation smoking	14.7 (3.3)	14.3 (2.7)	15.0 (3.7)	1.21
Cigarettes/day (past-week)	18.3 (15.2)	19.8 (14.3)	17.3 (15.8)	−0.88
Nicotine dependence (FTND)	5.4 (1.9)	5.6 (1.9)	5.1 (1.9)	−1.58
Expired CO breath sampled (ppm)	15.8 (9.5)	17.2 (10.2)	14.8 (8.9)	−1.30
# prior quit attempts	4.5 (5.4)	5.1 (7.9)	4.1 (2.5)	−0.82
PTSD symptom severity (PCL-S)	46.8 (14.5)	57.8 (9.5)	39.1 (12.3)	−9.41***
Neuroticism (BFI-N)	24.2 (5.8)	26.8 (4.9)	22.5 (5.7)	−4.31***
Anxiety sensitivity (ASI-3)	22.8 (15.8)	27.0 (15.2)	19.8 (15.6)	−2.52*
Harmful consequences expectancies (SAEQ-Harm)	13.7 (8.6)	16.3 (8.5)	11.9 (8.3)	−2.85**
Somatic symptom expectancies (SAEQ-Somatic)	13.4 (8.2)	13.4 (8.1)	13.3 (8.2)	−0.05

* p < .05
** p < .01.
*** p < .001.

Table 3
Standardized regression results for the tests of indirect effects.

Y	Model	R^2	β	SE	t	p	CI (l)	CI (u)
1	PCL-S → ASI-3 (a)	.258	.26	.09	2.92	.004	.08	.43
	ASI-3 → Harm (b)	.293	.31	.09	3.53	< .001	.14	.49
	PCL-S → Harm (c′)		.23	.09	2.67	.009	.06	.41
	PCL-S → Harm (c)	.217	.32	.09	3.55	< .001	.14	.49
	PCL-S → ASI-3 → Harm (a ∗ b)		.08	.04			.02	.20
2	ASI-3 → Somatic (b)	.209	.40	.09	4.26	< .001	.22	.59
	PCL-S → Somatic (c′)		.15	.09	1.64	.10	-.03	.34
	PCL-S → Somatic (c)	.086	.26	.10	2.66	.009	.07	.45
	PCL-S → ASI-3 → Somatic (a ∗ b)		.10	.05			.02	.24

Note. Path a_i is equal in models Y_{1-2}; therefore, it presented only in the models with Y_1 to avoid redundancies. N for analyses is 122 cases. The standard error and 95% CI for the indirect effects are obtained through bootstrapping with 10,000 re-samples. PCL-S (9/11-related PTSD symptoms) is the independent variable (X) in all models. ASI3-total (Anxiety Sensitivity Index-3, Total score; M); SAEQ Harm (Expecting harmful consequences from acute smoking abstinence; Y_1) and Somatic (Expecting somatic symptoms from acute smoking abstinence; Y_2), are the outcome variables. Gender, FTND, and BFI-Neuroticism were covariates in all models. CI (l) = lower bound of a 95% confidence interval; CI (u) = upper bound; → = affects.

somatic symptoms from smoking abstinence ($\beta = .06$, bootstrapped $CI_{95\%} = .01–.16$) after accounting for other sub-factors; a small to medium sized effect ($K^2 = .06$).

3.3. Post-hoc test of conditional mediation

Based on the high rate of the sample that met criteria for PTSD (41.0%), participant characteristics were examined stratified by those who met criteria for PTSD and those who did not. Smokers who met criteria for PTSD relative to those who did not (sub-clinical symptoms) reported significantly higher PTSD symptom severity, anxiety sensitivity, neuroticism, and expectancies that smoking abstinence would yield harmful consequences (Table 1).

In terms of regression models, results indicated that PTSD diagnosis did not moderate the effect of PTSD symptom severity on anxiety sensitivity ($\beta = .22$, $t = 0.99$, $p = .322$). Inferential tests of the equality of the mediating effect in smokers with and without PTSD revealed that the conditional indirect effect was non-significant for expectancies of harmful consequences ($\beta = .07$, SE = .09, bootstrapped $CI_{95\%} = -.05–.31$) or somatic symptoms ($\beta = .09$, SE = .11, bootstrapped $CI_{95\%} = -.08–.36$); thus, the hypothesis of moderated-mediation was rejected.

4. Discussion

The current study examined (a) the role of PTSD symptom severity in terms of interoceptive threat-related smoking abstinence expectancies and (b) the mediating role of anxiety sensitivity in this association, among a sample of treatment-seeking smokers exposed to the WTC disaster. First, consistent with *a priori* prediction, PTSD symptom severity was directly associated with expectancies of harmful consequences and somatic symptoms from 24-h of smoking abstinence. Specifically, more severe PTSD symptoms accounted for significant variance in expectations that acute smoking abstinence would produce harmful outcomes and uncomfortable/unmanageable somatic symptoms. Consistent with theoretical models of PTSD (*e.g.*, Foa & Kozak, 1986), heightened trauma-specific threat/harm perception and distorted cognitions are thought to underlie the maintenance of PTSD, which may also generalize to smoking-specific cognitions of harm and promote avoidance (continued smoking). These findings suggest that, among treatment-seeking smokers, greater PTSD symptom severity may contribute to stronger beliefs that acute abstinence may be harmful or result in somatic distress. Based on the observation that smokers often increase the average number of cigarettes consumed per day after disaster exposure (Biggs et al., 2010), continued reliance on cigarettes may be, in part, due to expectancies that smoking abstinence would be harmful and physically distressing. Accordingly, it may be important to assess for and intervene to modify such expectancies among trauma-exposed treatment-seeking smokers in efforts to reduce smoking rate or facilitate quit success (Feldner, Smith, Monson, & Zvolensky, 2013), especially smokers presenting with elevated PTSD severity.

Second, as hypothesized, anxiety sensitivity mediated the association between PTSD symptom severity and threat-related abstinence expectancies. Consistent with prior findings, PTSD symptom severity appears to be associated with the tendency to catastrophically interpret the meaning of anxiety-relevant sensations (G. N. Marshall et al., 2010), which in turn, is associated with increased threat-related smoking abstinence expectancies (Abrams et al., 2011; Farris, Langdon, et al., 2015). Here, findings underscore the relevance of not only clinically assessing and empirically understanding the way in which PTSD symptom *severity* is related to smoking processes, but additionally explicating the role of one's *interpretation* of such symptoms (*i.e.*, as indicative of harmful or catastrophic outcomes; McNally, 2002; Reiss et al., 1986). Notably, the inclusion of anxiety sensitivity accounted for unique variance abstinence expectancies (between 7.6%–12.3%; medium size effects). Moreover, the mediating role of anxiety sensitivity was significant after adjusting for neuroticism, which documents the explanatory specificity of these findings beyond the general propensity to experience negative affective states.

Some evidence for anxiety sensitivity sub-factor specificity emerged. Specifically, anxiety sensitivity physical concerns sub-factor (but not social or cognitive concerns) was a significant mediator of the association between PTSD symptom severity and expectancies of uncomfortable somatic symptoms from smoking abstinence (but not harmful consequences). These findings offer increased conceptual specificity to how cognitive reactivity to somatic distress (*e.g.*, "When I feel dizzy, I worry I might faint") among disaster-exposed smokers may impact cognitions about the somatic consequences of smoking abstinence. Although the size of this effect was small in size, these data are conceptually relevant given the physiological nature of some nicotine withdrawal symptoms (*e.g.*, restlessness, sleep disturbances, coughing, dizziness; Hughes, 2007) and physical health consequences of smoking. Indeed, previous work has also found specificity for the physical concerns sub-factor of anxiety sensitivity in terms of predicting smoking heaviness among treatment-seeking smokers (Farris et al., 2015) and more severe PTSD symptoms (Feldner, Babson, Zvolensky, Monson, et al., 2007; Feldner, Leen-Feldner, Trainor, Blanchard, & Monson, 2008).

A post-hoc mediation model was conducted to further explicate the nature of the associations between PTSD symptom severity, anxiety sensitivity, and threat-based abstinence expectancies. Specifically, given the sample comprised of smokers with both clinical and sub-clinical levels of PTSD, the presence/absence of a PTSD diagnosis was examined as a conditional variable impacting the meditational effects. Here, data indicated that the mediating effect of anxiety sensitivity smokers was not conditional upon PTSD diagnostic status, suggesting that these mechanistic associations may be relevant for both clinical and non-clinical trauma-exposed smokers (Zlotnick, Franklin, & Zimmerman, 2002). This finding is meaningful given the bulk of existing studies that have examined the interplay between PTSD symptom severity and anxiety sensitivity have utilized samples of smokers with sub-clinical levels of PTSD (*e.g.*, for exception, see Asnaani et al., 2015), and none to our knowledge have empirically examined the conditional role of presence/absence of PTSD. It is important to understand differences between smokers with and without PTSD, as such inquiry may facilitate understanding of individual differences factors that may maintain PTSD and smoking behavior.

There are a number of study limitations. First, the analyses presented here utilized cross-sectional data. Future work will benefit from longitudinal tests of these associations to understand how PTSD severity may impact actual smoking cessation outcomes as a function of anxiety

sensitivity. Second, although participants in the current sample were excluded for having a current alcohol/substance use disorder, the extent of alcohol use and other substance use is an important contextual factor that should be considered in understanding the nature of PTSD symptoms, expectancies about smoking abstinence, and anxiety sensitivity (*e.g.*, Simpson, Jakupcak, & Luterek, 2006). Lastly, while the PCL-S is the most widely used self-report measure of PTSD symptoms (McDonald & Calhoun, 2010), future work should consider use of clinician-administered severity assessment of PTSD symptoms (*e.g.*, Blake et al., 1995).

The current findings suggest that anxiety sensitivity may underlie the association between PTSD symptom expression and threat-related smoking abstinence expectancies among WTC disaster-exposed smokers. Accordingly, there may be a need to clinically address anxiety sensitivity to facilitate change in abstinence expectancies among trauma-exposed responders. For example, the current data suggest that it may be advisable to understand and clinically address anxiety sensitivity to enhance psychological flexibility related to smoking abstinence beliefs in order to address unhelpful smoking cognitions and facilitate change in smoking behavior. Overall, the present study serves as an initial investigation into the nature of the association between PTSD symptom severity, anxiety sensitivity, and abstinence expectancies among smokers exposed to the WTC disaster. Future work is needed to explore the extent to which anxiety sensitivity accounts for relations between PTSD symptoms and other smoking processes (*e.g.*, withdrawal, cessation outcome) to further clarify theoretical models of trauma-related emotional vulnerability and smoking.

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Contributors

Drs. Kotov, Bromet, and Luft designed the parent study and wrote the protocol. Ms. Farris and Dr. Zvolensky conducted literature searches and provided summaries of previous research studies for the current paper. Ms. Farris and Mr. Paulus conducted the statistical analysis and wrote the first draft of the manuscript. All authors edited the manuscript, and have approved the final manuscript.

Conflict of interest

Ms. Farris, Mr. Paulus, Dr. Gonzalez, Dr. Mahaffey, Dr. Bromet, Dr. Luft, Dr. Kotov and Dr. Zvolensky, declare that they have no conflict of interest.

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