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Anxiety sensitivity as an amplifier of subjective and behavioral tobacco abstinence effects

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ABSTRACT

Background: Anxiety sensitivity, a transdiagnostic cognitive vulnerability factor described as an amplifier of negative emotional states, is implicated in the maintenance of cigarette smoking and cessation difficulties. The current study aimed to examine the role of anxiety sensitivity in predicting abstinence-induced changes in nicotine withdrawal, smoking urges and smoking behavior during an experimental relapse analogue task (RAT).

Method: Participants were 258 non-treatment seeking smokers (*M* [*SD*] age = 44.0 [10.73]; 69.8% male). Participants attended two counterbalanced experimental sessions including smoking deprivation (16 h of smoking abstinence) and smoking as usual. The Minnesota Nicotine Withdrawal Scale (MNWS) and Brief Questionnaire of Smoking Urges (QSU) were completed at each session in addition to the RAT. Hierarchical regressions were conducted to examine the predictive impact of anxiety sensitivity on withdrawal and urges during smoking deprivation. Follow-up mediational analyses were conducted to examine whether abstinence-induced withdrawal and urges mediated responding during the RAT.

Results: Anxiety sensitivity amplified the effects of experimentally manipulated acute abstinence on subjective nicotine withdrawal symptoms and smoking urges. Additionally, higher levels of anxiety sensitivity indirectly predicted shorter latency to smoking initiation after deprivation during the RAT through the effects of greater abstinence-induced nicotine withdrawal and smoking urges. Anxiety sensitivity was unrelated to increased smoking during the RAT, although this may be partially attributed to the type of laboratory assessment employed.

Conclusions: Elevated anxiety sensitivity appears to impact initiation of smoking after nicotine deprivation through the effects of abstinence-induced withdrawal and smoking urges.

1. Introduction

Smokers with comorbid psychiatric conditions often struggle to quit smoking, contributing to a stagnation of smoking base rates in the United States (Hughes, 2011). Smokers with anxiety psychopathology represent one of the most common of these 'high risk' groups (Williams et al., 2013). Indeed, nearly one-fourth of individuals with nicotine dependence suffer from at least one comorbid anxiety disorder (Grant et al., 2004). Moreover, elevated anxiety symptoms and disorder status increases the risk of smoking experimentation (Leventhal et al., 2011; Patton et al., 1998), progression to daily smoking (Audrain-McGovern et al., 2011), and development of nicotine dependence (McKenzie et al., 2010). Among current smokers, anxiety symptoms and disorders often increase risk of smoking cessation failure (Hall et al., 1994), heighten severity of tobacco withdrawal (Langdon et al., 2013), and contribute to maladaptive cognitive beliefs and cognitive–emotional reactions to tobacco (Peasley-Miklus et al., 2012).

Various types of anxiety symptoms and disorders are associated with smoking variables, including PTSD (Feldner et al., 2007), social anxiety disorder (McCabe et al., 2004), panic attacks and disorder (Zvolensky et al., 2003c), and generalized anxiety disorder (Goodwin et al., 2012). Hence, one promising means of elucidating the role of anxiety in cigarette use is to investigate the influence of transdiagnostic psychological vulnerability factors that

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underlie multiple anxiety-related conditions on smoking. Anxiety sensitivity is one such transdiagnostic factor. Anxiety sensitivity is a relatively stable, but malleable, psychological individual difference factor related to sensitivity to (or anticipation and fear of the consequences of) aversive internal states of anxiety (Reiss et al., 1986). Over two decades of research has indicated that anxiety sensitivity is concurrently and prospectively associated with anxiety symptoms and with the onset of various types of anxiety disorders (Marshall et al., 2010; Schmidt et al., 2006) and incrementally predicts clinical anxiety outcomes over and above trait or state anxiety symptoms and other negative affect states (e.g., depression; Rapee and Medoro, 1994; Zvolensky et al., 2003a).

Importantly, anxiety sensitivity also is consistently implicated in the maintenance of smoking behavior. Indeed, smokers higher relative to lower in anxiety sensitivity perceive quitting as more difficult (Johnson et al., 2013) and have less success when making quit attempts (Assayag et al., 2012; Zvolensky et al., 2009), even when statistically controlling for variation in anxiety symptoms (Zvolensky et al., 2009). Laboratory studies suggest anxiety sensitivity may be involved in affective reactivity to stress during smoking and non-smoking contexts. For example, in one laboratory study, reward and affect relief were greater during a stress-inducing speech preparation task among smokers high in anxiety sensitivity (Perkins et al., 2010a). Based upon these findings, it is important to elucidate the mechanisms by which anxiety sensitivity potentially maintains smoking behavior, particularly upon abstinence.

Nicotine withdrawal symptoms experienced as a result of smoking reduction are key mechanisms of tobacco dependence. They are reliably associated with increased risk of smoking behavior (Nakajima and al'Absi, 2012; Patterson et al., 2008; Piper et al., 2011a), presumably because withdrawal provokes negative reinforcement processes that motivate the resumption of smoking upon abstinence to quell withdrawal distress (Baker et al., 2004). Available work provides a reason to predict that anxiety sensitivity may enhance acute nicotine withdrawal, possibly due to greater cognitive-affective reactivity to interoceptive and other internal affective stimuli experienced in withdrawal (Zvolensky and Bernstein, 2005). The vast majority of work on this topic has primarily been drawn from cessation studies, which illustrate that higher levels of anxiety sensitivity are indeed related to more intense nicotine withdrawal symptoms during the early phases of quitting (Johnson et al., 2012; Langdon et al., 2013; Marshall et al., 2009). However, these investigations were limited in that not all smokers successfully maintained abstinence at the time of withdrawal measurement, potentially leaving open the confounding factor of variation in smoking deprivation level (i.e., smokers who relapsed are protected against abstinence-induced exacerbation in withdrawal symptoms). Laboratory based designs involving experimental manipulation of acute (overnight) tobacco abstinence in smokers not wishing to quit allow for the control and standardization of degree of smoking deprivation, thereby preventing such confounds that can occur in naturalistic quit smoking studies. Though there are limits to external validity, tobacco withdrawal severity during experimentally-induced abstinence predicts withdrawal in a subsequent naturalistic self-initiated quit attempt (al'Absi et al., 2005), suggesting that experimentally-manipulated abstinence research may generalize outside the laboratory. Furthermore, these acute abstinence effects are relevant to the withdrawal experiences of smokers not attempting to quit that may ultimately maintain daily smoking behavior (e.g., symptoms experienced before the first cigarette of the day). This type of process may be especially pertinent to anxiety sensitive smokers who report greater worry about quitting (Zvolensky et al., 2009), tend to lapse faster (Brown et al., 2001), and endorse negative reinforcement smoking expectancy effects and motives (Johnson et al., 2013; Leyro et al., 2008).

In addition to limits in study design, the assessment of withdrawal phenomena in anxiety sensitivity research has been narrow. For instance, no past work has explored psychological motivation (Piasecki et al., 2010), one of the most robust phenotypic expressions of the tobacco withdrawal syndrome (Leventhal et al., 2010) and a key predictor of smoking relapse (Piper et al., 2011b). Further, anxiety sensitivity research has yet to examine important behavioral reactions to acute tobacco abstinence effects. A key behavioral consequence related to withdrawal is abstinence-provoked increases in motivation smoke. In the laboratory, researchers have assessed this process using analogue models of lapse behavior in which participants are monetarily rewarded to: (1) delay the opportunity to initiate smoking and (2) smoke fewer cigarettes when given the opportunity to smoke (McKee et al., 2006). This behavioral consequence of tobacco abstinence is particularly important given research illustrating that smokers with current anxiety disorders are more likely to lapse on their planned quit date or avoid making a quit attempt altogether (Leventhal et al., 2012). Establishing if anxiety sensitivity effects on lapse-like behavior during tobacco abstinence are mediated by abstinence-related provocations of subjective withdrawal symptoms may shed important light on the mechanisms through which anxiety sensitivity helps maintain smoking behavior.

To address gaps in existing knowledge, the current laboratory study examined the extent to which (trait) anxiety sensitivity amplifies the influence of experimentally-manipulated acute tobacco abstinence on subjective withdrawal symptoms and smoking urge as well as on an analogue objective measure of lapse-like behavior. First, it was hypothesized that higher levels of anxiety sensitivity would predict greater abstinence-induced increases in nicotine withdrawal symptoms and smoking urges. Second, it was hypothesized that anxiety sensitivity would be associated with abstinence-induced lapse behavior (i.e., reductions in ability to delay smoking for money and increases in cigarettes purchased following delay) indirectly through greater abstinence-induced subjective withdrawal and urge.

2. Method

2.1. Participants

Participants were non-treatment seeking smokers recruited in the Los Angeles, California area to participate in a study of individual differences in tobacco withdrawal (Leventhal et al., 2014). The sample in the current report included 258 smokers (*M* [*SD*] age = 44.0 [10.73]; 69.8% male) who were administered measures of anxiety sensitivity. Inclusion criteria for the study included being 18 years of age or older, smoking at a daily rate of ≥10 cigs/day (biochemically verified by ≥10 ppm carbon monoxide expired breath sample at baseline), being a regular smoker for at least the past two years, and fluency in English. Exclusion criteria included current *DSM–IV* non-nicotine substance dependence, current *DSM–IV* mood disorder or psychotic symptoms, regular use of other tobacco/nicotine products, current use of psychotropic medications, current pregnancy, and intentions to quit or substantially cut down smoking in the next 30 days. The original sample included 343 participants; 57 did not complete the study after enrolling and 28 participants completed the study prior to the introduction of anxiety sensitivity and other measures analyzed in this report, which were introduced into the study midstream through recruitment.

Participants primarily identified as African American (51.6%) and White (33.7%); 14.3% identified their ethnicity as Hispanic. The average daily smoking rate of this sample was 16.6 (*SD* = 7.01), and

severity of nicotine dependence was moderate (Fagerström Test of Nicotine Dependence: $M = 5.2$, $SD = 1.95$).

2.2. Procedure

Interested persons completed a preliminary telephone assessment to determine likely eligibility, after which they were scheduled for an in-person baseline session. The baseline appointment included informed consent, biochemical verification of positive smoking status, an assessment of lifetime psychosis and current (past month) mood and substance use disorders, and a series of self-report questionnaire assessments. If eligible at the baseline session, participants were scheduled to attend two counterbalanced experimental sessions: smoking deprivation (16 h of smoking abstinence) and smoking as usual (non-abstinent). Each experimental session started at noon and were typically scheduled a minimum of 2 days apart and no more than 14 days apart. Participants completed a breath alcohol analysis and CO assessment at the start of each experimental session. Participants with a positive breath alcohol analysis or with a breath CO reading >9 ppm at their abstinent session were considered non-abstinent and rescheduled. To control for any abstinence that may have occurred prior to arriving for the non-abstinent session, participants smoked a cigarette of their preferred brand in the lab at the beginning of that session. Participants were compensated approximately $200 for completing the study. The University of Southern California Institutional Review Board approved the protocol.

2.3. Measures

Baseline assessments: Diagnostic assessments of current (past month) Axis I psychopathology were conducted using the Structured Clinical Interview Non-Patient Version for DSM-IV Disorders (SCID-I/NP; First et al., 2007) to assess study eligibility. The Fagerström Test of Nicotine Dependence (FTND; Heatherton et al., 1991) was used as a measure of nicotine dependence severity and a covariate in the current analyses. The Center for Epidemiologic Studies Depression Scale (CES-D; Shafer, 2006) assessed baseline depressive symptoms; the 7-item negative affect subscale (CES-D-NA) was used as a covariate in analyses.

The Anxiety Sensitivity Index (ASI; Reiss et al., 1986) is a 16-item self-report questionnaire that measures the extent to which individuals are sensitive to emotional or physical sensations that they may experience (e.g., "It scares me when I feel faint"). Participants are asked to indicate the extent to which they agree with each statement, rated on a Likert-type scale that ranges from 0 (*very little*) to 4 (*very much*). Earlier studies have shown that the ASI has adequate test–retest reliability and good internal consistency (Reiss et al., 1986). The total score was used as the predictor in these analyses.

Experimental session assessments: Upon arrival to the experimental sessions, participants completed the Minnesota Nicotine Withdrawal Scale (MNWS; Hughes and Hatsukami, 1986), which was used to assess withdrawal symptoms experienced "so far today". The 11-item version of this measure was used. Items are rated on a 6-point Likert scale with higher scores reflecting greater subjective reporting of withdrawal. A total score was derived from a composite index based on mean response per item. The 10-item Brief Questionnaire of Smoking Urges (QSU; Cox et al., 2001) was used to assess smoking urges experienced "right now." Items are rated on 6-point Likert scale (higher scores indicate stronger urges); a composite total index based on mean response per item is yielded. Secondary analyses focused on the two subscales of desire/urges and negative affect relief.

Next, participants completed an experimental analogue task (RAT; McKee et al., 2006). During this task, participants received a tray containing eight cigarettes (their usual brand), a lighter, and an ashtray. At the outset of the delay period, participants were instructed they could begin smoking at any point during the next 50 min, but they would earn $.20 for each 5 min they delay smoking, for a total of $2 maximum for delaying smoking. Thus, the delay period could span 0–50 min. Once participants indicated that they wished to initiate smoking or following 50 min (whichever occurred first), the participants were informed that they could smoke as much or little as they wanted during the next 60 min. Participants were told they had a $1.60 credit, and for each cigarette lit, it would cost $.20. Two criterion variables were computed–change in time delay (delay [in minutes] during abstinent–delay during non-abstinent session; possible range −50 to +50) and change in number of cigarettes smoked (0 = no change from abstinent/non-abstinent RAT and 1 = increase in one or more number of cigarettes smoked in abstinent RAT relative to non-abstinent because the numerical value of number of cigarettes was not normally distributed). Prior research illustrates that tobacco deprivation, stress, and cessation medications modulate outcomes on the RAT in expected directions, supporting the validity of the RAT (Leeman et al., 2010; McKee et al., 2011, 2012).

2.4. Data analytic strategy

First, descriptive statistics and zero-order correlations among study variables were examined. To address Aim 1, initial analyses included two hierarchical regression models to examine the impact of anxiety sensitivity on abstinence-induced effects (score at abstinent session) in (a) nicotine withdrawal (MNWS) and (b) smoking urges (QSU). In each regression model, the corresponding non-abstinent outcome measure was included as a covariate in Step 1 of the model to control for non-abstinent ratings. Additionally, gender, nicotine dependence (FTND), and negative affect (CESD-NA) were included as planned covariates in Step 1. Gender was selected as a covariate because of prior associations with abstinence-induced nicotine withdrawal (Leventhal et al., 2007). CESD-NA and FTND were selected as covariates to examine whether any relation between anxiety sensitivity and withdrawal was not solely explained by individual differences in general negative affective symptomatology or heavier more compulsive smoking behavior, respectively. At step 2 of each model, ASI was entered to examine incremental prediction over and above the covariates. Next, to address Aim 2, four regression-based mediation models were conducted to examine abstinence-induced changes in MNWS (M_1) and QSU (M_2) as potential mediators of the effect of AS (X) on abstinence-induced performance during the RAT: decrease in time delay of smoking during abstinent RAT (Y_1) and increase in number of cigarettes smoked during abstinent RAT (yes = 1; no = 0) (Y_2). Models of Y_2 were logistic regression models. Gender, CESD-NA, FTND, and the corresponding non-abstinent RAT outcome measure and non-abstinent mediator score were included as covariates in the models of M and Y. The mediation analyses were conducted using PROCESS, a conditional modeling program that utilizes an ordinary least squares-based path analytical framework to test for both direct and indirect effects (Hayes, 2013). All indirect effects were subjected to follow-up bootstrap analyses with 10,000 samples and 95-percentile confidence intervals (CI) were estimated (as recommended by Hayes, 2009; Preacher and Hayes, 2004, 2008).

3. Results

3.1. Descriptive overview

Anxiety sensitivity, as indexed by the ASI-total score, averaged 20.2 ($SD = 12.79$), which is consistent with other

Table 1
Descriptive statistics and correlations ($N = 258$).

Variable	2	3	4	5	6	7	8	9	10	11	12	Mean (or n)	SD (or %)
1. Gender (F)	−.019	−.030	−.063	−.065	−.129*	.024	−.059	−.057	.058	−.040	−.069	78	30.2
2. FTND	1	.029	.130*	.159*	.257**	.300**	.384**	.008	−.151*	−.039	.061	5.2	1.95
3. CES-D		1	.391**	.312**	.209**	.084	.053	−.082	.032	−.009	−.063	0.4	.51
4. ASI-Total			1	.263**	.287**	.077	.222**	−.053	−.056	−.052	−.027	20.2	12.79
5. MNWS-Non-Abs				1	.471**	.316**	.260**	−.197**	−.197**	.107	.153*	1.8	1.10
6. MNWS-Abs					1	.144*	.575**	−.076	−.240**	.129*	.111	1.0	.91
7. QSU-Non-Abs						1	.367**	−.119	−.110	.077	.118	1.0	1.12
8. QSU-Abs							1	−.030	−.250**	.018	.154*	3.3	1.06
9. Time delay-Non-Abs								1	.358**	−.306**	−.450**	23.6	22.84
10. Time delay-Abs									1	−.511**	−.356**	39.5	17.52
11. Cig Smoke-Non-Abs										1	.480**	1.3	.93
12. Cig Smoke-Abs											1	1.5	.94

Note: * $p < .05$; ** $p < .01$; gender = % listed are females; 0 = female; 1 = male; FTND = Fagerström Test for Nicotine Dependence—total score; CES-D = Center for Epidemiologic Studies Depression Scale; ASI-Total = Anxiety Sensitivity Index-Total Score; MNWS-Non-Abs = Minnesota Nicotine Withdrawal Scale at non-abstinent experimental session; MNWS-Abs = Minnesota Nicotine Withdrawal Scale at abstinence/deprivation experimental session; QSU-Non-Abs = Brief Questionnaire of Smoking Urges at non-abstinent experimental session; QSU-Abs = Brief Questionnaire of Smoking Urges at abstinence/deprivation experimental session; Delay-Non-Abs = Delay in seconds to relapse analogue task during non-abstinent experimental session; Delay-Abs = delay in seconds to relapse analogue task during abstinence/deprivation experimental session; Cig Smoke-Non-Abs = number of cigarettes smoked after non-abstinent RAT; Cig Smoked-Abs = number of cigarette smoked after abstinent RAT. For regression analyses, a "Cig Increased" variable was coded by 0 = no change in smoking from abstinent to non-abstinent sessions; 1 = any increase in smoking during abstinent session relative to non-abstinent, due to the non-normal distribution of this variable; 37.6% of participants ($n = 97$) demonstrated increased in smoking behavior during deprived trial, relative to satiated. Columns numbers 2–12 correspond to the variables numbers in the far left column.

community-recruited smoking samples (Zvolensky et al., 2007). The ASI was significantly and positively associated with nicotine dependence, negative affect, abstinent and non-abstinent nicotine withdrawal symptoms, and abstinent smoking urges (see Table 1). Notably, abstinence-induced changes in withdrawal and urges were significantly correlated with each other (moderate in strength). Additionally, female gender was associated with greater subjective reporting of abstinence-induced withdrawal.

3.2. Test of main effects

Next, the incremental effect of anxiety sensitivity on abstinence-induced nicotine withdrawal (MNWS) and smoking urges (QSU) was evaluated (see Table 2). The model for MNWS accounted for 28.8% of the overall variance [$F(5,252) = 20.389$, $p < .0001$]. Step 1 covariates accounted for 27.0% of variance. Step 2 accounted for an additional 1.8% of variance; higher anxiety sensitivity significantly predicted greater abstinence-induced increases in composite nicotine withdrawal symptom severity.

The model of QSU accounted for 24.8% of the overall variance [$F(5,252) = 16.663$, $p < .0001$]. Step 1 covariates accounted for 22.1% of variance in abstinence-induced changes in urges. Step 2 accounted for an additional 2.7% of variance. Again, anxiety sensitivity was significantly predictive of greater abstinence-induced increases in smoking urges.

Post-hoc analyses were conducted to test the main effect of anxiety sensitivity on abstinence-induced changes in the two subscales of the QSU: (1) desire/urges and (2) negative affect relief. Results revealed a non-significant effect of anxiety sensitivity on QSU-Desire/Urges subscale ($B = .009$, $t = 1.652$, $p = .100$); however, the effect of anxiety sensitivity on QSU-negative affect relief subscale was significant ($B = .020$, $t = 3.436$, $p = .001$). A total of 33.1% of variance was accounted by the full model; 3.1% was accounted by Step 2 (the addition of anxiety sensitivity; $F(5,252) = 24.921$, $p < .0001$). As expected, higher levels of anxiety sensitivity predicted greater self-reported negative affect relief smoking urges during abstinence.

3.3. Mediation analyses

Meditation analyses were conducted next (please see Table 3) to examine the effect of anxiety sensitivity as a predictor of abstinence-induced change in time delay to smoking [Y_1] and cigarettes smoked [Y_2] during RAT, through the proposed mediators (MNWS [M_1]; QSU [M_2]). Thus, a total of four meditational models were tested.

With regard to the model of Y_1,M_1, the total effects model including anxiety sensitivity and all covariates was significant ($R^2_{y1,x} = .179$, $df = 6, 251$, $F = 9.098$, $p < .0001$), which was largely driven by some of the covariates; the total effect of ASI on abstinence-induced time delay to smoking was non-significant. The full model with the mediator was significant ($R^2_{M1,x} = .201$, $df = 7, 250$, $F = 8.972$, $p < .0001$), with abstinence-induced withdrawal significantly predicting time delay (path b; $p = .009$). The direct effect of ASI on time delay, after controlling for the mediator, was non-significant. Regarding the test of the indirect (mediational) effect, the effects of path a (effect of anxiety sensitivity on abstinence-induced withdrawal) × path b (effect of abstinence-induce withdrawal on time delay to smoking) was significant; higher levels of anxiety sensitivity were predictive of shorter time delay to smoking indirectly through greater subjective levels of nicotine withdrawal.

In terms of Y_1,M_2, the total effects model was significant ($R^2_{y1,x} = .165$, $df = 6, 251$, $F = 9.098$, $p < .0001$), which again was largely driven by covariates; the total effect of ASI on abstinence-induced changes in time delay was non-significant. The full model with the mediator was significant ($R^2_{M2,x} = .201$, $df = 7, 250$, $F = 8.960$, $p < .0001$), with abstinence-induced smoking urges significantly predicting time delay (path b; $p = .0009$). After controlling for the mediator, the direct effect of ASI on time delay to smoking was non-significant. The indirect effect of path a × path b was significant, with higher levels of anxiety sensitivity predicting shorter time delay to smoking indirectly through greater subjective smoking urges. Post-hoc analyses indicated that this indirect effect was only significant for the QSU-negative affect relief subscale ($b = -.080$, $se = .033$, $CI = -.162, -.080$).

In terms of Y_2,M_1, the total effects model (Nagelkrk $R^2_{y2,x} = .027$, $x^2(5) = 5.071$) and full model with the mediator (Nagelkrk $R^2_{M1,x} = .031$, $x^2(6) = 6.015$) predicted a non-significant amount variance in likelihood of smoking more cigarettes during abstinence RAT. The indirect effect was estimated; results were non-significant. Similarly in the model of Y_2,M_2, neither the total effects model (Nagelkrk $R^2_{y2,x} = .029$, $x^2(5) = 5.507$) nor the full model with the mediator (Nagelkrk $R^2_{M2,x} = .041$, $x^2(6) = 7.958$) predicted a significant amount variance in the likelihood of smoking

Table 2
Regression models for main effects of anxiety sensitivity on abstinence-induced changes in subjective nicotine withdrawal symptoms and smoking urges.

DV		ΔR^2	Predictors	*B*	SE	*t*	*p*
MNWS-Abs	1	.270	Gender	−.232	.129	−1.801	.073
			FTND	.106	.031	3.455	.001
			CES-D	.156	.121	1.282	.201
			MNWS-Non Abs	.498	.069	7.195	.000
	2	.018	ASI-Total	.013	.005	2.500	.013
QSU-Abs	1	.221	Gender	−.136	.128	−1.066	.288
			FTND	.163	.032	5.149	.000
			CES-D	.040	.115	.346	.730
			QSU-Non Abs	.262	.055	4.745	.000
	2	.027	ASI-Total	.015	.005	3.022	.003

Note: Covariates: gender = coded 0 = female; 1 = male; FTND = Fagerström Test for Nicotine Dependence–total score; CES-D = Center for Epidemiologic Studies Depression Scale; MNWS-Non-Abs = Minnesota Nicotine Withdrawal Scale at non-abstinent experimental session; QSU-Non-Abs = Brief Questionnaire of Smoking Urges at non-abstinent experimental session. Predictor: ASI-Total = Anxiety Sensitivity Index-Total Score; Criterion Outcome: MNWS-Abs = Minnesota Nicotine Withdrawal Scale at abstinence/deprivation experimental session; QSU-Abs = Brief Questionnaire of Smoking Urges at abstinence/deprivation experimental session.

Table 3
Regression results for mediation of anxiety sensitivity on analogue lapse behavior outcomes by nicotine withdrawal and smoking urges.

Model	Path	*b*	*SE*	*t*	*p*	CI (*l*)	CI (*u*)
Y_1, M_1	ASI → MNWS (a)	.013	.005	2.495	.013	.003	.023
	MNWS → DELAY (b)	−3.661	1.391	−2.632	.009	−6.401	−.922
	ASI → DELAY (c′)	−.002	.113	−.019	.985	−.225	.221
	ASI → DELAY (c)	−.049	.113	−.429	.669	−.272	.174
	ASI → MNWS → DELAY ($a \times b$)	−.046	.027			−.126	−.006
Y_1, M_2	ASI → QSU-T (a)	.015	.005	3.016	.003	.005	.025
	QSU-T → DELAY (b)	−4.715	1.404	−3.357	.001	−7.481	−1.949
	ASI → DELAY (c′)	−.011	.113	−.095	.924	−.233	.212
	ASI → DELAY (c)	−.082	.113	−.721	.471	−.304	.141
	ASI → QSU-T → DELAY ($a \times b$)	−.071	.032			−.150	−.022
Y_2, M_1	MNWS → CIG (b)	.135	.139	.970	.332	−.138	.408
	ASI → CIG (c')	.002	.011	.152	.880	−.021	.024
	ASI → CIG (c)	.003	.011	.304	.761	−.019	.025
	ASI → MNWS → CIG ($a \times b$)	.002	.002			−.001	.008
Y_2, M_2	QSU-T → CIG (b)	.227	.146	1.55	.122	−.060	.514
	ASI → CIG (c′)	.001	.011	.004	.999	−.022	.022
	ASI → CIG (c)	.004	.011	.314	.754	−.018	.025
	ASI → QSU-T → CIG ($a \times b$)	.003	.003			−.001	.011

Note. $N = 258$ in all models. In a simple mediation model, the impact of X on Y is considered a total effect (path c), interpreted as the expected amount by which two cases that differ by one unit on X are expected to differ on Y, which may occur directly or indirectly. The direct effect of X (path c′) is interpreted as the part of the effect of X on Y that is independent of the pathway through M. The indirect effect (product of path a and b) is interpreted as the amount by which two cases who differ by one unit on X are expected to differ on Y through X's effect on M, which in turn affects Y. This is the test of mediation (the effect of X on Y through M) or the difference between the total and direct effects ($a \times b = c - c'$). The statistical strategy utilized here (as recommended by Hayes, 2009; Preacher and Hayes, 2004) allows for estimation and significance testing of the indirect effect, through bootstrapping, which generates an empirical representation of the sampling distribution of the indirect effect, from which a confidence interval can be generated. Please see Hayes (2009) for a more comprehensive overview. The standard error and 95% CI for $a \times b$ are obtained by bootstrapping with 10,000 re-samples. ASI (anxiety sensitivity) is the independent variable (X); MNWS (abstinence-induced nicotine withdrawal; M_1) and QSU-T (abstinence-induced smoking urges, total score; M_2) are the mediators; and DELAY (abstinence-induced time delay to RAT; Y_1) and CIG (change in cigarettes smoked: 0 = no change in cigarettes smoked from non-abstinent to abstinent RAT; 1 = increase in smoking during abstinent RAT relative to non-abstinent; Y_2) are the outcomes. Covariates in all models included gender, FTND, CES-D, and the non-abstinent measure of the mediator (either MNWS or QSU-T) and non-abstinent outcome measure (time delay to smoking). CI (*l*) = lower bound of a 95% confidence interval; CI (*u*) = upper bound; → = predicts.

more cigarettes during abstinence RAT. The indirect effect was non-significant.

4. Discussion

In line with predictions, anxiety sensitivity amplified the effects of experimentally manipulated acute tobacco abstinence on subjective nicotine withdrawal symptoms and smoking urges. These effects were relatively small in absolute effect size, but they were evident above and beyond the variance accounted for by the corresponding non-abstinent outcome measure as well as by gender, nicotine dependence, and depressive symptoms. Post hoc analyses for urges suggested that anxiety sensitivity predicted greater negative affect relief smoking urges during abstinence. Overall, these results suggest that anxiety sensitivity is not merely a proxy for other factors implicated in tobacco withdrawal or baseline (i.e., sated/non-deprived) withdrawal-like symptoms. Such laboratory findings are consistent with past cessation studies that have reported anxiety sensitivity is related to more intense nicotine withdrawal symptoms during the early phases of quitting (Johnson et al., 2012; Langdon et al., 2013; Marshall et al., 2009) and provide novel data extending prior work to: (1) a well-controlled laboratory setting that minimized the potential confound of tobacco deprivation variation; (2) urge to smoke—a theoretically and clinically meaningful subjective manifestation of tobacco withdrawal (Piper et al., 2011b). Anxiety sensitivity is by definition a fear of and hyper-reaction to aversive internal states (e.g., "unusual body sensations scare me" is an ASI item), and this fear may actually prolong and enhance aversive internal states (Taylor, 1999). We, therefore, speculate that hypersensitivity and fear in response to the sensations provoked by disruptions of neurobiological homeostasis produced by nicotine withdrawal along with the psychological stress of coping without smoking in anxiety sensitive smokers may amplify the standard subjective manifestations of tobacco withdrawal (Zvolensky and Bernstein, 2005).

A secondary finding from the present investigation found that higher levels of anxiety sensitivity indirectly predicted shorter latency to smoking initiation after deprivation (relative to

satiation) during the behavioral task through the effects of greater abstinence-induced nicotine withdrawal and smoking urges. These results are relevant to smoking treatment studies that have found anxiety sensitivity is related to increased risk of smoking lapse during the early phase (one week) of quitting (Brown et al., 2001; Zvolensky et al., 2009) as they identify a putative mechanism – subjective withdrawal – that may channel anxiety sensitivity effects on lapse behavior. However, there was no evidence of a total effect of anxiety sensitivity on abstinence-induced changes in ability to delay smoking, although these findings may have been influenced by the payment offered (McKee et al., 2011). Additionally, as the RAT task was conducted as part of a laboratory assessment; thus, could be an insensitive or less ecologically valid measure of ad libitum smoking after delay. In general, these findings suggest the possibility that other (unmeasured) factors may perhaps protect against abstinence-related provocation of smoking behavior for anxiety sensitive smokers, which in combination with anxiety sensitivity's amplifying effect on subjective withdrawal may result in a net effect on lapse behavior that is minimal. Identification of such factors that may be protective is warranted, given prior evidence that anxiety sensitive smokers tend to expect greater smoking-related health consequences and report greater motivation to quit (Zvolensky et al., 2007). There was no evidence of an anxiety sensitivity effect for likelihood of increasing smoking rate after deprivation (relative to satiation) or mediation via subjective withdrawal. Given that there was no evidence of any covariates we specifically tested, including nicotine dependence, in predicting changes in the number of cigarettes smoked, it is possible that this may not be an ideal outcome (McKee et al., 2011). Future work may be warranted to explore how high anxiety sensitive smokers actually smoke (i.e., puff style/topography), especially a single cigarette post-deprivation. This type of examination may yield micro-level information regarding the smoking motivation among anxiety sensitive smokers.

There are a number of interpretive caveats to the present study that warrant further consideration. First, our sample consisted of community-recruited, daily cigarette smokers with moderate levels of nicotine dependence. Future studies may benefit by sampling from lighter and heavier smoking populations to ensure the generalizability of the results to the general smoking population. Second, the observed significant effects, while generally consistent with the a priori theoretical model, were modest in absolute effect size. These effects are also generally consistent with prior laboratory-based smoking work (e.g., Perkins et al., 2010b). Yet, given withdrawal and craving mediated the effect of anxiety sensitivity on time to smoking initiation, in conjunction with extant literature suggesting the importance of cognitive-affective vulnerabilities for smoking behavior and relapse risk (e.g., Cameron et al., 2013; Correa-Fernandez et al., 2012; Gwaltney et al., 2005; Shiffman, 2005), it is likely that the effects demonstrated in this study, although small, may be likely to impact smoking behavior and thus could be clinically relevant. It is notable that there was *no* direct effect of elevated anxiety sensitivity on latency to smoking re-initiation, rather only an indirect effect through deprivation-induced withdrawal and urges. Notably, mediation is fully possible without a significant total effect when there are other mediators not accounted for in the model (Hayes, 2013). Thus, more comprehensive models of AS and cessation-related processes warrant further exploration.

Overall, the present study provides novel, methodologically rigorous multi-method evidence of anxiety sensitivity as an amplifier of tobacco abstinence effects. Collectively, the current findings are potentially important because they open the possibility that anxiety sensitivity may serve to amplify important contributing factors to the re-initiation of smoking following brief periods of abstinence (e.g., overnight, temporary restrictions due to work) and to earlier lapse during an actual quit attempt—subjective withdrawal symptoms and urge to smoke. These findings provide evidence for continued efforts to target reductions in anxiety sensitivity in integrated smoking protocols to improve cessation outcomes. In fact, extant work, while still emerging, suggests anxiety sensitivity reduction programs for smoking achieved via psychoeducation, cognitive restructuring, and interoceptive exposure are related to reductions in cigarettes smoked using various methodological designs, including case study, case series, open clinical trial, and randomized clinical trial (Feldner et al., 2013; Zvolensky et al., 2014, 2003b, 2008). We believe that further triangulation of laboratory, naturalistic, and clinical research of anxiety sensitivity and smoking is warranted to ultimately elucidate the clinical significance of this work and its role in offsetting the public health burden of comorbid anxiety syndromes and tobacco addiction.

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Contributors

All authors were involved in the design of the study. Dr. Zvolensky, Ms. Farris, and Dr. Leventhal managed the literature searches and summaries of previous related work and completed statistical analyses. Drs. Leventhal and Guillot edited and assisted with data collection and management. All authors contributed to and have approved the final manuscript.

Conflict of interest statement

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this paper.

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