Examination of Smoking Inflexibility as a Mechanism Linking Anxiety Sensitivity and Severity of Smoking Behavior

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Background and Objectives: Anxiety sensitivity is associated with smoking processes and poorer clinical outcomes. Yet, the specific mechanisms underlying this association are unclear. Smoking-specific avoidance and inflexibility (AIS) is a construct implicated in multiple manifestations of mood regulation that may underlie smoking severity. The current study examined whether AIS accounted for (ie, statistically mediated) the relationship between anxiety sensitivity and multiple indices of smoking severity.
Methods: Baseline self-report data were collected among treatment-seeking smokers ($N = 396$; 48% female; $M_{age} = 37.8$ years) taking part in a larger intervention study. Gender, smoking-related medical history, Axis I diagnoses, hazardous alcohol use, substance abuse/dependence, and negative affectivity were statistically controlled in analyses.
Results: Anxiety sensitivity was indirectly related to all smoking severity variables, with the exception of nicotine dependence, through its relation with AIS.
Discussion and Conclusions: These findings provide initial evidence suggesting AIS may be an important construct in better understanding anxiety sensitivity-smoking relations.
Scientific Significance: Results suggest the importance of AIS as a malleable target for smoking cessation intervention. (Am J Addict 2015;24:374–381)

INTRODUCTION

Anxiety symptoms and disorders are associated with smoking.[1] One promising means of elucidating the role of anxiety in cigarette use is to investigate the influence of

transdiagnostic psychological vulnerability factors that underlie multiple anxiety-related conditions on smoking. Anxiety sensitivity is one such transdiagnostic factor. Anxiety sensitivity is a relatively stable, but malleable, cognitive-based individual difference factor related to the anticipation and fear of the consequences of aversive internal (ie, interoceptive) states.[2] Anxiety sensitivity is distinguishable empirically and theoretically from anxiety *symptoms* and other negative affect states like depressive symptoms.[3]

Anxiety sensitivity also is related to smoking behavior. For example, anxiety sensitivity is positively correlated with smoking motives to reduce negative affect[4] and beliefs that smoking will reduce negative affect.[5] Higher levels of anxiety sensitivity are predictive of increases in positive affect after cigarette smoking,[6] and smoking has been found to reduce anxiety in high anxiety sensitive smokers who smoked during a stressful situation, but not in a no-stress situation.[7] Smokers higher in anxiety sensitivity, relative to those lower, perceive quitting as more difficult[8] and experience more intense nicotine withdrawal during early phases in quitting[9] and in the laboratory.[10] Moreover, higher levels of anxiety sensitivity are related to greater odds of early smoking lapse[11] and relapse during quit attempts.[12] Importantly, the observed anxiety sensitivity-smoking effects do not appear to be explained by smoking rate, gender, other concurrent substance use (eg, alcohol, cannabis), panic attack history, or trait-like negative mood propensity.[5,6] Yet, little research has explored the underlying mechanisms that connect anxiety sensitivity and smoking behavior. Increasingly, attention has been given to experiential avoidance in the development and maintenance of both anxiety disorders[13,14] and substance use disorders[15] as a dysfunctional means of coping with internal distress. Experiential avoidance is a cognitive–affective regulatory process wherein individuals are unwilling to experience or remain in contact with aversive internal experiences

(ie, avoidance) and attempt to control the frequency or form of the experiences and the contexts in which they occur (ie, psychological inflexibility). Recent non-smoking research distinguishing experiential avoidance from anxiety sensitivity suggests that experiential avoidance may account for the relation of anxiety sensitivity-related beliefs with avoidant behavior and disabling outcomes.[13] Indeed, experiential avoidance may be an affect regulatory mechanism by which individuals with high anxiety sensitivity develop disorders involving affect regulatory difficulties. However, it is unclear whether experiential avoidance explains the relation between anxiety sensitivity and smoking.

One's tendency to respond to smoking-related urges, negative affect, or interoceptive states with experiential avoidance (ie, avoidance/inflexibility to smoking; AIS) may contribute to reliance on cigarettes and cessation difficulties via more severe cessation sequelae (eg, withdrawal, craving, negative affect).[16] There is some limited evidence that inhibiting smoking-related thoughts is, in fact, related to a greater number of failed cessation attempts.[17] Moreover, when smokers are provided cognitive-behavioral smoking cessation treatment specifically aimed at promoting psychological flexibility in the context of smoking-related distress (eg, acceptance and commitment-based treatments),[18,19] decreases in AIS are associated with increased likelihood of smoking abstinence after treatment.[19,20]

Despite past work, it is presently unknown if and how AIS impacts actual smoking behavior reflective of smoking severity (eg, latency to first cigarette). Moreover, while anxiety sensitivity predicts smoking behavior[5,6] and is associated with experiential avoidance,[13] no studies to date have examined whether AIS may explain the relation between anxiety sensitivity and smoking severity. One study found anxiety sensitivity was indirectly related to greater barriers to cessation, greater number of prior quit attempts and greater mood-management smoking expectancies through AIS.[21] These findings invite further empirical exploration of the role of AIS in anxiety sensitivity–smoking relations. It may be that, as high anxiety sensitive smokers perceive anxiety-related sensations as a sign of imminent harm, they are likely to respond to interoceptive peturbation with avoidance via smoking, which, in turn, may increase smoking severity.

The primary aim of the present research was to examine whether AIS explains the relation of anxiety sensitivity and smoking severity among treatment-seeking smokers. It was hypothesized that after controlling for gender, smoking-related medical history, current Axis I disorders, hazardous alcohol use, substance abuse/dependence, and negative affectivity, anxiety sensitivity would have a significant indirect effect on smoking severity through its relation with AIS. Smoking severity was assessed via current (ie, past week) smoking rate (ie, average number of cigarettes per day), smoking rate during the heaviest smoking period (henceforth called "heaviest smoking rate"), latency to first cigarette of the day (henceforth called "latency to first cigarette"), and nicotine dependence.

METHOD

Participants

The sample consisted of 396 treatment-seeking adult smokers (48% female; $M_{age} = 37.8$; SD = 13.2; age range: 18–65 years) who reported smoking at least eight cigarettes per day for the past year. Recruitment occurred via advertisements, media releases, community postings, and medical referrals. Breath carbon monoxide level (cutoff >6 ppm) was used to verify smoking status ($M_{breath\ CO} = 21$ ppm, SD = 11.4).[22] Exclusion criteria were suicidality and psychosis; based on these criteria, five people were excluded. The ethnic distribution of this sample was as follows: 86.8% White/Caucasian; 7.1% Black/Non-Hispanic; 0.4% Black/Hispanic; 2.7% Hispanic; 0.9% Asian; and 1.8% "Other." Within the sample, 42.3% met criteria for at least one current (past year) Axis I diagnosis; the most common diagnoses were social anxiety disorder (9.9%), current major depressive episode (4.2%), posttraumatic stress disorder (2.9%), and generalized anxiety disorder (4.7%). Participants reported an average smoking rate of 17.5 (SD = 9.7), smoking their first cigarette at 14.7 years of age (SD = 3.7), and smoking regularly at 17.3 years of age (SD = 3.4). The average level of nicotine dependence was 5.3 (SD = 2.2) on the 10-point Fagerström Test for Nicotine Dependence, indicating moderate levels of nicotine dependence.

Measures

Demographics Questionnaire

Demographic information collected included gender, age, and race.

Structured Clinical Interview-Non-Patient Version for DSM-IV (SCID-I/NP)

Diagnostic assessments of past year Axis I disorder were conducted using the SCID-I/NP,[23] which was administered by doctoral level staff or trained research assistants and supervised by independent doctoral-level professionals. The reliability of a random selection of 12.5% of interviews was evaluated (MJZ) for accuracy; there were no cases of diagnostic disagreement. The present study created separate variables for the presence of a non-substance-related Axis I disorder (yes = 1; no = 0) and the presence of a non-alcohol-related substance use disorder (yes = 1; no = 0) that served as covariates.

Smoking History Questionnaire (SHQ)

The SHQ is a self-report questionnaire used to assess smoking history (eg, onset of daily smoking) and pattern (eg, smoking rate).[22] In the present study, the SHQ was used to describe the sample on smoking history and patterns of use. In addition, we used the following items as outcome variables: "Think about your smoking in the last week, how many cigarettes did you smoke in an average day?" (smoking rate); and "When smoking the heaviest, how many cigarettes did you smoke per day?" (heaviest smoking rate).

Fagerström Test for Nicotine Dependence (FTND)

The FTND is a six-item scale that assesses individual smoker's "nicotine dependence."[24] Total scale scores range from 0 to 10, with higher scores reflecting high levels of physiological dependence on nicotine. The FTND has shown adequate internal consistency ($\alpha = .61$), positive relations with key smoking variables (eg, saliva cotinine), and high test–retest reliability.[24] In the present sample, the FTND total score internal consistency was Cronbach's $\alpha = .57$. For the current study, we used the FTND total score and the single item assessing latency to first cigarette ("How many minutes after you wake do you smoke your first cigarette?") as separate outcome variables.

Carbon Monoxide

Biochemical verification of smoking status was completed by carbon monoxide (CO) analysis of breath samples. Expired air CO levels were assessed using a CMD/CO Carbon Monoxide Monitor (Model 3110; Spirometrics, Inc., Auburn, ME).

Medical History Form

Current and lifetime medical illnesses and current use of prescribed medication were assessed using a medical history checklist. For current and lifetime medical illnesses, a composite variable was computed for the present study as an index of tobacco-related medical illnesses, which was used as a covariate in all models. Items in which participants indicated having ever been diagnosed (respiratory disease, asthma, heart problems, and hypertension, all coded $0 = $ no, $1 = $ yes) were summed to create a total score (observed range from 0 to 4), with greater scores reflecting the presence of multiple markers of tobacco-related illnesses.

Alcohol Use Disorders Identification Test (AUDIT)

The AUDIT is a 10-item self-report measure developed to identify individuals with problematic drinking.[25] Its scores range from 0 to 30, with higher scores reflecting more problematic drinking. The AUDIT's psychometric properties are well documented. In the current investigation, the AUDIT total score internal consistency was good (Cronbach's $\alpha = .88$).

Positive and Negative Affect Scale (PANAS)

The PANAS is a self-report measure asking participants to rate the extent to which they experience each of 20 different feelings and emotions (eg, interested, nervous) based on a Likert scale that ranges from 1 ("very slightly or not at all") to 5 ("extremely").[26] The measure yields two factors, positive affect (PA) and negative affect (NA), and has shown good internal reliability (PA: $\alpha = .86$; NA: $\alpha = .87$) and criterion validity.[26] The negative affectivity subscale (PANAS-NA) internal consistency was good in the present sample (Cronbach's $\alpha = .89$).

Anxiety Sensitivity Index-3 (ASI-3)

The ASI-3 is an 18-item measure derived from the Anxiety Sensitivity Index questionnaire.[27] Respondents indicate the extent to which they are concerned about possible negative consequences of anxiety-related symptoms (eg, "It scares me when my heart beats rapidly"). Responses are rated on a 5-point Likert scale ranging from 0 ("very little") to 4 ("very much") and summed to create a total score. In the current study, internal consistency was excellent (Cronbach's $\alpha = .91$).

Avoidance and Inflexibility Scale (AIS)

The AIS is a 13-item self-report measured that assesses the link between internal (affective) triggers and smoking (smoking-related inflexibility/avoidance).[19] Respondents are asked how they respond to different feelings that encourage smoking (eg, stress, fatigue), difficult thoughts that encourage smoking (eg, "I need a cigarette"), and bodily sensations that encourage smoking (eg, "physical cravings or withdrawal symptoms"). Sample items include the following: "How important is getting rid of [thoughts/feelings/sensations]?"; "How likely is it you will smoke in response to [thoughts/feelings/sensations]?"; and "To what degree must you reduce how often you have these [thoughts/feelings/sensations] in order not to smoke?" Items are rated on a 5-point Likert scale (1 = "not at all" to 5 = "very much"), with higher scores reflecting more inflexibility/avoidance when facing difficult smoking-related feelings, thoughts, and sensations. The AIS has displayed good reliability and validity in past work.[19] In the present study, the AIS demonstrated excellent internal reliability (Cronbach's $\alpha = .93$). The AIS total score was used as the proposed mediator.
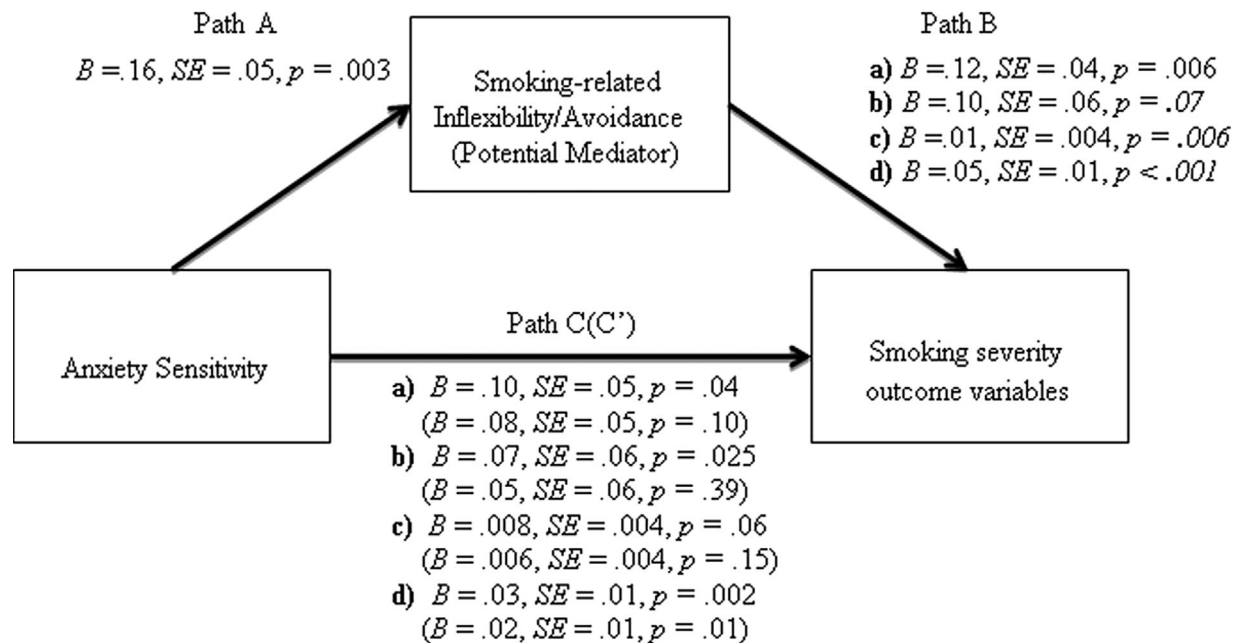
Procedure

The present study was part of a large, multi-site randomized controlled clinical trial examining the efficacy of two smoking cessation interventions.[28] Following written informed consent, participants completed an in-person, baseline assessment to evaluate study eligibility, including a diagnostic interview (SCID-I/NP) and a computerized self-report battery. The study protocol was approved by the Institutional Review Boards at the University of Vermont and Florida State University. The current study is based on secondary analyses of baseline (pre-treatment) data for a subset of the sample, which was selected on the basis of complete data for all studied variables.

Data Analytic Strategy

The proposed mediation analysis was conducted using bootstrapping techniques through Indirect Macro (Version 4.2, Andrew F. Hayes, Columbus, Ohio, USA),[29] a computational tool for observed variable mediation analysis. As a non-parametric method, bootstrapping estimates the sampling distribution of an estimator based on resampling with replacement. The indirect effect (mediation pathway) was computed for each of the samples, resulting in an empirically generated sampling distribution.[30] We conducted separate analyses for each outcome variable, with anxiety sensitivity as the predictor and AIS as the proposed mediator in each analysis (see Fig. 1). Covariates included gender, tobacco-related medical illness, current non-substance-related Axis I diagnosis,

Proposed model: Smoking-related inflexibility/avoidance as a potential mediator of the association between anxiety Sensitivity and smoking behaviors.

Path A

$B = .16, SE = .05, p = .003$

Smoking-related Inflexibility/Avoidance (Potential Mediator)

Path B

a) $B = .12, SE = .04, p = .006$
b) $B = .10, SE = .06, p = .07$
c) $B = .01, SE = .004, p = .006$
d) $B = .05, SE = .01, p < .001$

Anxiety Sensitivity

Path C(C')

a) $B = .10, SE = .05, p = .04$
   $(B = .08, SE = .05, p = .10)$
b) $B = .07, SE = .06, p = .025$
   $(B = .05, SE = .06, p = .39)$
c) $B = .008, SE = .004, p = .06$
   $(B = .006, SE = .004, p = .15)$
d) $B = .03, SE = .01, p = .002$
   $(B = .02, SE = .01, p = .01)$

Smoking severity outcome variables

Note: Smoking severity outcome variables examined included: a) Smoking rate; b) Heaviest smoking rate; c) Latency to first cigarette; and d) Nicotine dependence.

FIGURE 1. Proposed model: Smoking-related inflexibility/avoidance as a potential mediator of the association between anxiety sensitivity and smoking behaviors.

hazardous alcohol use, current substance abuse/dependence diagnosis, and negative affectivity. Coefficient of determination (R^2) was utilized to index the fit of each model. R^2 (the square of the coefficient of multiple correlation) measures how well the regression line correlates with the real data (ie, model fit).[31] We calculated the semipartial correlations to depict the correlation between the predictor and the outcome variable after removing common variance with other predictors (residualized predictor). This can be interpreted as the proportion of the variance in outcome variable contributed uniquely by the predictor.[31] Ten thousand bootstrap resamplings were conducted to detect the indirect effects of the proposed predictor on outcome variables through the proposed mediator (ie, the product of the beta coefficients of path A and path B; see Fig. 1).

Based on recommendations for reducing type I error,[32] percentile-based (PB) confidence intervals were used. A bootstrap-confidence interval that does not include zero provides evidence of a significant indirect effect.[29] Completely standardized indirect effects for each significant indirect pathway were also presented.[33] Finally, the theoretical models for each outcome variable were compared to two alternative models. In the first alternative model, the proposed predictor and mediator variables were reversed; in the second

alternative model, the proposed outcome and mediator variables were reversed. Comparing alternative models with alternative variable sequences is suggested in cross-sectional studies examining statistical mediation effects as an additional test of the hypothesized order of influence among the study variables in the absence of a prospective study design.[29,30]

RESULTS

Descriptive data and correlations of the all variables included in the models are presented in Table 1. To examine the theoretical models using the Indirect Macro,[29] the indirect effects of anxiety sensitivity on the outcome variables through AIS were estimated as the product of the beta coefficients predicting AIS from anxiety sensitivity (path A in Fig. 1) and each of the outcome variables from AIS (path B in Fig. 1). Table 2 presents the results of these analyses.

Regarding smoking rate, bootstrap analysis (10,000 resamples) revealed a significant positive indirect effect in the proposed model (point estimate = .024, PB 95% CI: .0057–.0510; total effect of AS controlling for AIS = .08, SE = .05, p = .10; completely standardized indirect effect = 0.03). Moreover, results of bootstrap analysis (10,000 resamples) of the alternative

TABLE 1. Zero-order correlations among theoretically relevant variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Gender (% female)[a]	—	.132**	−.132**	.022	.143**	−.007	−.088	−.074	.002	.000	.074	.202**
2. PANAS-NA[a]		—	.237**	.010	.344**	.124*	.024	.023	−.034	.044	.608**	.240**
3. AUDIT[a]			—	−.102*	.187**	.178**	−.019	−.055	−.110*	−.064	.208**	.035
4. Medical problems[a]				—	.033	−.068	.067	.069	−.006	−.011	.011	.061
5. Axis I disorder[a]					—	.339**	.067	.013	.064	.113*	.337**	144**
6. Substance use[a]						—	.021	−.067	−.064	−.033	.130**	.022
7. Smoking rate[d]							—	.576**	.388**	.596**	.080	.179**
8. Heaviest smoking rate[d]								—	.287**	.470**	.020	.118*
9. Latency to 1st cigarette[d]									—	.790**	.040	.156**
10. Nicotine dependence[d]										—	.122*	.266**
11. ASI-3[b]											—	242**
12. AIS[c]												—
Mean (or n)	190	18.90	5.76	0.36	167	31	17.49	25.97	1.97	5.33	14.92	45.35
SD (or %)	48.00	6.94	5.83	0.61	42.30	7.80	9.71	12.56	0.89	2.23	11.92	10.84

Gender = % listed are females (coded 0 = male; 1 = female); PANAS-NA = Positive and Negative Affect Scale—Negative Affect subscale; AUDIT = Alcohol Use Disorders Identification Test—total score; Medical Problems = Tobacco-related medical problems per the Medical Screening Questionnaire; Axis I Disorder = Current Axis I disorder per the Structured Clinical Interview—Non-Patient Version for DSM-IV; Substance Use = Current non-alcohol substance abuse/dependence diagnosis per the Structured Clinical Interview—Non-Patient Version for DSM-IV; Smoking Rate = Number of cigarettes per day during past week per the Smoking History Questionnaire; Heaviest smoking rate = Number of cigarettes per day during the heaviest smoking period per the Smoking History Questionnaire; Latency to 1st Cigarette = Time between waking and first cigarette of the day per the Fagerström Test for Nicotine Dependence; Nicotine Dependence = Fagerström Test for Nicotine Dependence—total score; ASI-3 = Anxiety Sensitivity Index-3—total score; AIS = Acceptance and Inflexibility Scale —total score.
[a]Covariates; [b]Predictor; [c]Mediator; [d]Outcome variables; *$p < .05$; **$p < .00$.

models excluded the possibility of mediation in these directions (point estimate =.0044, PB 95% CI: −.0073 to .0183; and point estimate =.013, PB 95% CI: −.0066 to .0379, for alternative models 1 and 2 respectively).

Regarding heaviest smoking rate, bootstrap analysis (10,000 resamples) revealed a significant positive indirect effect in the proposed model (point estimates =.021, PB 95% CI: .0046–.0495; total effect of AS controlling for AIS =.05, SE =.06, p =.39; completely standardized indirect effect = 0.021). Moreover, results of bootstrap analysis (10,000 resamples) of the alternative models excluded the possibility of mediation in these directions (point estimate = −.025, PB 95% CI: −.0192 to .0121; and point estimate = −.004, PB 95% CI: −.0163 to .014, for alternative models 1 and 2 respectively).

Regarding the latency to first cigarette, bootstrap analysis (10,000 resamples) revealed a significant positive indirect effect in the proposed model (point estimate =.0021, PB 95% CI: .0006–.0045; total effect of AS controlling for AIS =.006, SE =.004, p =.15; completely standardized indirect effect = 0.028). Moreover, results of bootstrap analysis (10,000 resamples) of the alternative models excluded the possibility of mediation in these directions (point estimate = −.0006, PB 95% CI: −.0004 to .0019; and point estimate =.0132, PB 95% CI: −.0008 to .0369, for alternative models 1 and 2 respectively).

Regarding nicotine dependence, bootstrap analysis (10,000 resamples) revealed a significant positive indirect effect in the proposed model (point estimates =.0084, PB 95% CI: .0031–.0159; total effect of AS controlling for AIS =.02, SE =.01, p =.01; completely standardized indirect effect = 0.05; see Table 2). Moreover, results of bootstrap analysis (10,000 resamples) of the alternative models excluded the possibility of mediation for the first alternative model (point estimate =.002, PB 95% CI: −.0008 to .0054) but not the second model (point estimate =.030, PB 95% CI: .0041–.0650).

DISCUSSION

The present study explored whether AIS explains, in part, the relation between anxiety sensitivity and markers of smoking severity. As hypothesized, anxiety sensitivity exerted a significant indirect effect through AIS upon smoking rate, heaviest smoking rate, and latency to first cigarette. However, the hypothesized indirect effect of anxiety sensitivity via AIS upon nicotine dependence was not supported. These results suggest that increased sensitivity to aversive smoking-specific cognitions and sensations (ie, anxiety sensitivity) may contribute to efforts to control such internal cues (ie, AIS), which, in turn, leads to greater smoking rate and smoking severity, but not necessarily nicotine dependence. These results are broadly in line with past research that has indicated AIS contributes to difficulties with quitting smoking[19–21] and uniquely extend it to numerous indicators of smoking severity.

TABLE 2. Model fit indices for all examined outcome variables

Outcome variable	ΔR^2 (p-value)	Predictors	β	t	Sr^2
		Constant		4.003	
		Gender	−.142**	−2.747	−.136
		NA	−.044	−0.674	−.033
		AUDIT	−.052	−0.993	−.049
Smoking rate	.4 (p = .003)	Health	.048	0.967	.048
		Axis I	.061	1.077	.053
		Sub. Use	.008	0.152	.008
		ASI-3	.049	0.760	.038
		AIS	.195**	3.742	.185
		Constant		6.150	
		Gender	−.118*	6.157	−.112
		NA	−.118	−2.249	.025
		AUDIT	.032	0.492	−.064
Heaviest smoking rate	.015 (p = .08)	Health	−.069	−1.280	.046
		Axis I	.046	0.918	.033
		Sub. Use	.038	0.656	−.060
		ASI-3	−.064	−1.193	−.018
		AIS	−.023*	−0.355	.125
		Constant		6.237	
		Gender	−.046	−0.897	−.044
		NA	−.106	−1.634	−.081
		AUDIT	−.118*	−2.235	−.111
Latency to 1st cigarette	.04 (p = .004)	Health	−.038	−0.753	−.037
		Axis I	.109	1.906	.094
		Sub. Use	−.080	−1.514	−.075
		ASI-3	.067	1.037	.051
		AIS	.165**	3.158	.156
		Constant		5.190	
		Gender	−.075	−1.485	−.072
		NA	−.070	−1.102	−.053
		AUDIT	−.099	−1.923	−.093
Nicotine dependence	.08 (p = .0001)	Health	−.045	−0.931	−.045
		Axis I	.120*	2.146	.104
		Sub. Use	−.066	−1.266	−.061
		ASI-3	.088	1.394	.067
		AIS	.266**	5.209	.252

Gender = % listed are females (coded 0 = male; 1 = female); PANAS-NA = Positive and Negative Affect Scale—Negative Affect subscale; AUDIT = Alcohol Use Disorders Identification Test—total score; Medical Problems = Tobacco-related medical problems per the Medical Screening Questionnaire; Axis I Disorder = Current Axis I disorder per the Structured Clinical Interview—Non-Patient Version for DSM-IV; Sub. Use = Current non-alcohol substance abuse/ dependence diagnosis per the Structured Clinical Interview—Non-Patient Version for DSM-IV; Smoking Rate = Number of cigarettes per day during past week per the Smoking History Questionnaire; Heaviest smoking rate = Number of cigarettes per day during the heaviest smoking period per the Smoking History Questionnaire; Latency to 1st Cigarette = Time between waking and first cigarette of the day per the Fagerström Test for Nicotine Dependence; Nicotine Dependence = Fagerström Test for Nicotine Dependence—total score; ASI-3 = Anxiety Sensitivity Index-3—total score; AIS = Acceptance and Inflexibility Scale—total score.
*$p < .05$; **$p < .00$.

Namely, they suggest the effect of anxiety sensitivity on smoking severity was indirect and dependent upon the degree to which a person responded to aversive internal cues with smoking-specific inflexibility and avoidance.

The observed mediation effects were evident after adjusting for the influence of factors known to correlate with the severity of smoking behavior, including gender, history of smoking-related illnesses, current Axis I diagnoses, hazardous alcohol use, substance abuse/dependence, and negative affectivity. Therefore, AIS was shown to have a unique effect over and above that of other theoretically relevant factors. Moreover, the two other models tested, with either anxiety sensitivity or each outcome variable serving as the proposed mediator, showed no significant indirect effects (with one

exception), suggesting that the statistical mediation effect was specific to AIS. Notably, the alternative model testing nicotine dependence as a mediator between the relation between anxiety sensitivity and AIS showed a significant indirect effect. Given that the theoretical model for nicotine dependence also showed a significant indirect effect, smoking-related experiential avoidance and nicotine dependence may exert reciprocal effects. Among individuals with greater anxiety sensitivity, greater nicotine dependence may increase the frequency and salience of aversive smoking-related cognitions and sensations, which, in turn, increases efforts to control or avoid such internal cues. Such efforts to control may then involve smoking, further reinforcing nicotine dependence. To more fully explore nature of the relation among these variables over time, future prospective modeling of the temporal ordering of anxiety sensitivity and AIS in relation to smoking severity is warranted.

The findings from the investigation may serve to conceptually inform the development of specialized intervention strategies for smokers with elevated anxiety sensitivity. Existing anxiety sensitivity reduction programs for smoking cessation, albeit still in developmental phases, have provided evidence of the feasibility and merit of incorporating tailored cognitive-behavioral skills that specifically address affective vulnerabilities (eg, interoceptive exposure, psychoeducation) into smoking cessation programs.[10] The current data suggest that it may be advisable to understand and clinically address anxiety sensitivity to enhance psychological flexibility related to smoking in order to address maladaptive smoking cognitions and facilitate change in smoking behavior. Acceptance-based techniques (eg, experiential awareness, openness, willingness, mindfulness, cognitive diffusion) have been shown to reliably reduce AIS.[18] Thus, such skills may be important to integrate into existing cognitive-behavioral anxiety sensitivity-reduction smoking cessation programs or other psychosocial intervention programs for anxiety/mood disordered smokers.

There are a number of interpretive caveats to the present study that warrant further consideration. First, given the cross-sectional nature of these data, it is unknown whether anxiety sensitivity is causally related to greater AIS or to the smoking severity outcomes. The present tests were solely based on a theoretical framework and did not allow for testing of temporal sequencing. Based upon the present results, future prospective studies are necessary to determine the directional effects of these relations. Second, our sample consisted of community-recruited, treatment-seeking 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. It also is noteworthy that the FTND internal consistency was relatively low, an issue often apparent with this measure.[34] Yet, Cronbach's α values are fairly sensitive to the number of items in each scale and it is not uncommon to find lower Cronbach values with shorter scales (eg, scales with less than 10 items, such as the 6-item FTND).[35] Third, the sample was largely comprised of a relatively homogenous group of treatment-seeking smokers. To rule out a selection bias and increase the generalizability of these findings, it will be important for future studies to recruit a more ethnically/racially diverse sample of smokers.

Overall, the present study serves as an initial investigation into the nature of the association between anxiety sensitivity, AIS and smoking behavior. Future work is needed to explore the extent to which AIS accounts for relations between anxiety sensitivity and other smoking processes (eg, withdrawal, cessation outcome) to further clarify theoretical models of emotional vulnerability and smoking.

Declaration of Interest

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

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