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Anxiety Sensitivity Explains Associations between Anxious Arousal Symptoms and Smoking Abstinence Expectancies, Perceived Barriers to Cessation, and Problems Experienced during Past Quit Attempts among Low-Income Smokers

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Abstract

Disproportionately more smokers report low-income and mental health problems relative to nonsmokers. Low-income smokers may use smoking to alleviate negative emotional states resulting from exposure to multiple stressors. Yet, little work has been devoted to elucidating mechanisms that may explain the association between negative emotional states and smoking-related processes among low-income smokers. The present study sought to address this gap by examining anxiety sensitivity, a transdiagnostic factor related to both anxiety and smoking, as a potential mediator for the influence of anxiety symptoms on smoking-related processes, including threat-related smoking abstinence expectancies (somatic symptoms and harmful consequences), perceived barriers for cessation, and problems experienced during past quit attempts. Participants included treatmentseeking daily cigarette smokers (n = 101; 68.3% male; $M_{age} = 47.1$; SD = 10.2). Results indicated that anxiety symptoms exerted a significant indirect effect through anxiety sensitivity for threatrelated smoking abstinence expectancies (somatic symptoms and harmful consequences), perceived barriers for cessation, and problems experienced during past quit attempts. The present results provide empirical support that anxiety sensitivity may be an underlying mechanism that partially explains the relation between anxiety symptoms and smoking processes among lowincome treatment-seeking smokers. Findings broaden current theoretical understanding of pathways through which anxiety symptoms contribute to maladaptive smoking processes and cognitions among socioeconomically disadvantaged smokers.

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More than 40 million adults in the United States (U.S.) are current smokers (Thorne, Malarcher, Maurice, & Caraballo, 2008). Smoking remains the leading cause of death and disability in the U.S. Between 2005 and 2009, smoking was responsible for over 480,000 premature deaths a year (Green, Beckham, Youssef, & Elbogen, 2014). Smoking is the leading preventable cause of death in the U.S., and is causally related to 17 different types of cancer (IARC, 2012). Moreover, smoking causes 30% of all cancer deaths, with 90% of all lung cancer deaths causally attributable to smoking (U.S. Department of Health and Human Services (USDHHS), 2014). Although the prevalence of smoking has declined over the past 50 years, large disparities in tobacco use remain across a number of groups (Green et al., 2014), particularly disadvantaged populations. Perhaps most notably, the initiation and maintenance of smoking is twice as likely for individuals living at or below the poverty level than for those living above the poverty level (Thorne et al., 2008). Comparisons of smoking prevalence using National Health and Nutrition Examination Survey (NHANES) data for the past 25 years reveal a worsening gap between those at the lowest and highest income levels (Kanjilal et al., 2006). Similarly, the prevalence of smoking among individuals living below poverty is 27.9% compared to 17% among those at or above poverty level (Morbidity and Mortality Weekly Report 2014, 2014). Low income smokers therefore represent a major health disparity population in the US and available data indicate that their unique healthcare needs are not being met (Cuevas et al., 2014).

A major contributing factor to smoking among low SES persons is the increased exposure to multiple stressors associated with low SES environments (e.g., high urban stress), which in turn, contribute to disruptions in emotional processes (Reitzel et al., 2013), including emotion regulatory processes (Cuevas et al., 2014; Doan & Evans, 2011; Evans & Fuller-Rowell, 2013; Reitzel et al., 2013a,b). Low SES individuals report more economic, employment-related, interpersonal, and transportation-related stressors than their higher SES counterparts (Businelle, Cuate, Kesh, Poonawalla, & Kendzor, 2013; Gallo & Matthews, 2003; Lantz, House, Mero, & Williams, 2005; Matthews, Flory, Muldoon, & Manuck, 2000; McLeod & Kessler, 1990). The neighborhoods in which low SES individuals live also contribute to greater relative exposure to stressful stimuli (Everson-Rose et al., 2011; L. Reitzel et al., 2013) and lower SES individuals often disproportionately suffer from psychological distress due to experienced stressors (Chen & Matthews, 2001; S. Cohen, Doyle, & Baum, 2006; Collins et al., 1998). Smokers living in neighborhoods with low social capital (D. A. Cohen, Finch, Bower, & Sastry, 2006), collective efficacy, (Bandura, 2000) and high economic disadvantage report greater tobacco dependence (Reitzel et al., 2012) and experience lower odds of cessation (Kendzor et al., 2012; Reitzel et al., 2013; L. R. Reitzel et al., 2013), potentially due to associated distress (Kendzor et al., 2010; Reitzel et al., 2013; Reitzel et al., 2011; Reitzel et al., 2010; Whembolua et al., 2012). Even when low SES individuals report fewer stressors, findings suggest that the stressors reported are more severe in nature (Grzywacz, Almeida, Neupert, & Ettner, 2004). Low SES has also been associated with greater engagement in maladaptive health behaviors to regulate stress (Jackson, Knight, & Rafferty, 2010), (Businelle et al., 2010; Kendzor et al., 2009). Consequently, given the many stressors faced by low SES persons, smoking may function as a readily-available way to a regulate stress/negative affect. Additionally, this population is exposed to significantly more highly stressful environments, which may contribute to,

exacerbate, or maintain chronically elevated negative affective disturbances (Haushofer & Fehr, 2014). Together, available data illustrate the close interconnection between low income smoking and negative affect as well as chronic stress.

Notably, mental health problems are disproportionately common among cigarette smokers in general, and a link between poor mental health and smoking persistence/inability to quit and remain abstinent is well-established (Cinciripini et al., 2010). As such, mental health problems are one possible factor that may be related to the stagnated rates of smoking prevalence in the United States (U.S.). For instance, among adults (at least 18 years old), smoking is more prevalent among those with, compared to those without, psychological disorders [39.1% versus 25.0%; (Substance Abuse and Mental Health Services Administration (SAMHSA), 2014)]. Findings from population-based studies indicate disproportionate rates of smoking among adults with psychological distress, whether measured dimensionally (Gfroerer et al., 2013; Kiviniemi, Orom, & Giovino, 2011; Lawrence, Hafekost, Hull, Mitrou, & Zubrick, 2013; Lawrence, Mitrou, & Zubrick, 2011; Leung, Gartner, Dobson, Lucke, & Hall, 2011; A. E. Taylor et al., 2014) or categorically (McClave, McKnight-Eily, Davis, & Dube, 2010; Substance Abuse and Mental Health Services Administration (SAMHSA), 2014). Additionally, smokers with psychological distress smoke more heavily than those without psychological distress (Kiviniemi et al., 2011; Leung et al., 2011; McClave et al., 2010) and smoking prevalence increases with the number of comorbid psychological disorders (McClave et al., 2010), which presumably contribute to greater distress and lead to increases in smoking rates. Finally, numerous population-based studies have reported that adults with psychological distress exhibit less success in quitting and remaining abstinent than those without psychological distress (Carter, van der Deen, Wilson, & Blakely, 2014; Gfroerer et al., 2013; Kiviniemi et al., 2011; Lawrence et al., 2013; Lawrence et al., 2011; Leung et al., 2011; McClave et al., 2010; Substance Abuse and Mental Health Services Administration (SAMHSA), 2014; A. E. Taylor et al., 2014).

There has been increased recognition that anxiety and its disorders covary with smoking. Anxiety symptoms and psychopathology co-occurs with smoking at rates that exceed those found in non-psychiatric populations (Ziedonis et al., 2008). Lasser et al. 2000) found that current smoking rates for respondents with an anxiety disorder (past month or lifetime) were significantly greater than smoking rates among respondents with no mental illness. Reported rates of smoking were highest among individuals with panic-related problems and other anxiety disorders where panic attacks are common (e.g., social phobia, posttraumatic stress disorder [PTSD], generalized anxiety disorder; Lasser et al., 2000). Similar findings have been found by others (Hapke et al., 2005; Sonntag, Wittchen, Hofler, Kessler, & Stein, 2000). Moreover, the observed association between smoking and anxiety psychopathology does not appear to be due to sociodemographic characteristics, other psychiatric comorbidities, or symptom overlap in diagnostic criteria for anxiety disorders and nicotine dependence (Zvolensky & Schmidt, 2003). Numerous studies indicate anxiety disorders significantly impair cessation success (Hapke et al., 2005; Lasser et al., 2000; Zvolensky et al., 2008). For example, Piper and colleagues (Piper et al., 2010) examined the relation of psychiatric disorders to tobacco dependence and cessation outcomes on 1,504 people making an aided smoking cessation attempt as part of a clinical trial. Six months after

quitting, those ever diagnosed with an anxiety disorder had the lowest abstinence rates. These findings provide evidence that anxiety and its disorders are important in reducing the odds of successful quitting.

Despite the documented association between poverty and smoking, and anxiety and smoking-related processes, respectively, there is little understanding of anxiety processes among low-income smokers. Additionally, no work has examined possible mediators of the relations between anxiety states and smoking processes among low-income smokers. Anxiety sensitivity, defined as the extent to which individuals believe anxiety and anxietyrelated sensations have harmful consequences (McNally, 2002), is one promising transdiagnostic candidate that may explain how anxiety contributes to smoking maintenance among low-income smokers. Anxiety sensitivity is a relatively stable individual difference variable that predisposes individuals to the development of anxiety/depressive problems (S. Taylor, 1999). Moreover, anxiety sensitivity significantly relates to avoidance-based response styles for coping with aversive events (Zvolensky & Forsyth, 2002) and is reliably correlated with smoking and other types of drug/alcohol use to reduce negative affect (Richard A Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001; Novak, Burgess, Clark, Zvolensky, & Brown, 2003). Specifically, a wide range of studies have documented the role of anxiety sensitivity in aspects of smoking maintenance and relapse processes. For example, anxiety sensitivity is associated with the tendency to perceive quitting as more difficult (Zvolensky et al., 2007) and to expect periods of smoking deprivation or abstinence to be more stressful and personally threatening (Guillot, Leventhal, Raines, Zvolensky, & Schmidt, 2016; Langdon, Farris, Hogan, Grover, & Zvolensky, 2016). In fact, anxiety sensitivity is related to greater negative affect, craving, and nicotine withdrawal symptoms, shorter time to lapse and relapse, and lower overall abstinence during quit attempts (Johnson, Stewart, Rosenfield, Steeves, & Zvolensky, 2012; Langdon et al., 2013). Other work has found that anxiety sensitivity mediates the relation between emotional disorders and severity of smoking behavior (Zvolensky, Farris, Leventhal, & Schmidt, 2014) as well relations between posttraumatic stress symptoms and threat-related smoking abstinence expectancies among trauma exposed smokers (Farris et al., 2015). Overall, extant data suggest anxiety sensitivity represents an explanatory mechanism between anxiety-related symptoms and smoking maintenance and relapse. However, no past work has focused on low-income smokers, leaving unclear the role of anxiety sensitivity in the interconnection between anxiety states and smoking among socioeconomically disadvantaged adults.

The current study sought to build on past research and theory to test the hypothesis that, among low-income smokers, anxiety symptoms would be significantly related to threat-related smoking abstinence expectancies (somatic symptoms and harmful consequences), perceived barriers for cessation, and problem experienced during past quit attempts. It was further hypothesized that these relations would be explained by anxiety sensitivity, such that anxiety symptoms would be indirectly associated with the studied smoking processes (see Figure 1). All effects were expected to be evident above and beyond the variance accounted for by a range of theoretically-relevant covariates, including cigarettes smoked per day (CPD), participant sex, racial/ethnic minority status, marital status, education attainment, and employment status.

Method

Participants

Data from 101 adult daily smokers were included in the current study ($M_{age} = 47.1$ years [SD = 10.2]; 68.3% male). On average, the sample reported 4.9 (SD = 7.7) serious quit attempts, lifetime. Age of onset for regular smoking was 18.9 (SD = 6.9). The sample was racially diverse with 67.3% identifying as Black or African American, 28.7% as White, and 3.0% as "other", and 1.0% as Asian. Additionally, 5.9% identified their ethnicity as Hispanic or Latino(a). Most (72.0%) reported living in an urban area with 16.0% in suburban areas and 12.0% rural. Overall, the sample reported low-income: 44.6% reported earning less than \$5,000 per year with 14.9% reporting \$5,000-\$9,999, 18.8% reporting \$10,000-\$14,999, 12.9% reporting \$15,000–\$24,999, and 8.9% reporting \$25,000–\$34,999. Most the sample was single (59.4%), with 13.9% reporting divorce, 9.9% married, 8.9% living with a partner, 5.0% separated from a partner, and 3.0% widowed. Regarding education, 1.0% reported less than 7 years of formal education with 2.0% indicating junior high school as the highest level achieved, 9.9% reporting partial high school education, 34.7% reporting completing high school, 32.7% partial college, 8.9% completing college, and 10.9% completing graduate school. Only 14.9% of the sample reported full-time employment with 25.7% reporting parttime employment, 35.7% indicating public or private assistance, 5.0% reporting dependence on a spouse or student status and 19.8% reporting unemployment.

The prevalence rates for current psychological disorders were as follows: alcohol use disorder (22.8%), major depressive disorder (21.8%), substance use disorder (17.8%), posttraumatic stress disorder (15.8%), generalized anxiety disorder (14.9%), social anxiety disorder (13.9%), panic disorder (10.9%), bipolar I/II (9.0%), specific phobia (8.9%), agoraphobia (5.9%), dysthymia (5.9%), anxiety not otherwise specified (4.0%), eating disorder (3.0%), and obsessive-compulsive disorder (3.0%).

Procedure

Participants were recruited from Houston, Texas and participated between 2013–2016. Following written informed consent, participants completed a diagnostic interview and a computerized battery of self-report questionnaires. The current investigation is based on secondary analyses of baseline (pre-treatment) data obtained from a larger ongoing smoking cessation trial. Inclusionary criteria for the current study included smoking a minimum of 6 CPD (for at least 1 year), reporting motivation to quit smoking (i.e., scoring 5 or greater on a 0–10 Likert scale), and having elevated anxiety symptoms (Mood Anxiety Symptom Questionnaire [MASQ; Watson & Clark, 1991, see below] 17). The cutoff for elevated anxiety symptoms was determined based on inclusionary criteria for the larger trial. The study protocol was approved by the Institutional Review Board at the University of Houston. Each participant was paid \$20 upon completion of the baseline portion of the study.

Measures

Demographics Questionnaire—Demographic information collected included gender, age, race/ethnicity, income, marital status, education, and employment status. Information collected via the demographics questionnaire was used to describe the sample.

Structured Clinical Interview for DSM-IV Disorders—(SCID-I; First, Spitzer, Robert, Gibbon, & Williams, 2002). The SCID-I is a clinician-administered diagnostic assessment used to assess the presence of psychopathology. In the current study, the SCID-I was administered by trained doctoral students and research assistants under the supervision of a clinical psychologist. It was used to index current (past month) Axis I diagnoses. The SCID-I has demonstrated good reliability (Zanarini et al., 2000) and good to excellent validity (Basco et al., 2000) in past work. All assessments were reviewed for rater agreement; no cases of disagreement were noted.

Mood Anxiety Symptom Questionnaire Short Form—(MASQ; D. Watson & Clark, 1991). The MASQ is a 62-item self-report measure of affective symptoms. Participants rate each item (e.g., "felt dizzy") on a Likert scale from 1 (*not at all*) to 5 (*extremely*). The MASQ yields three subscales: general distress (MASQ-GD), anhedonic depression (MASQ-AD), and anxious arousal (MASQ-AA). The MASQ has demonstrated convergent validity with other measures of anxiety and depression (David Watson et al., 1995). The MASQ-AA was used in the current study as an index of symptoms unique to anxiety (i.e. non-overlapping with depression; Watson et al., 1995). Internal consistency for the MASQ-AA was excellent in the current sample ($\alpha = .94$).

Anxiety Sensitivity Index-3—(ASI-3; S. Taylor et al., 2007). The ASI-3 is a self-report measure of anxiety sensitivity derived, in part, from the original Anxiety Sensitivity Index (Reiss, Peterson, Gursky, & McNally, 1986). Each of 18 items (e.g., "It scares me when my heart beats rapidly") is rated on a scale from 0 (*very little*) to 4 (*very much*) and summed to a total score. The ASI-3 has strong psychometric properties (Taylor et al., 2007), which have been replicated among those with anxiety disorders (Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012) and among treatment-seeking smokers (Farris et al., 2015). In the current study, internal consistency was excellent for the total score ($\alpha = .95$).

Smoking Abstinence Expectances Questionnaire—(SAEQ; Abrams, Zvolensky, Dorman, Gonzalez, & Mayer, 2011). The SAEQ is a 28-item self-report measure that assesses consequences to be expected because of acute smoking abstinence (i.e., stop smoking cigarettes/using nicotine for one day). Items are rated in terms of expected likelihood on a scale from 0 (*very unlikely*) to 6 (*very likely*). The SAEQ yields four subscales: somatic symptoms (e.g., "I would feel short of breath"), harmful consequences (e.g., "I would feel like I'm dying"), negative mood consequences (e.g., "I would feel tense"), and positive consequences (e.g., "I would find it easy to concentrate"; Abrams et al., 2011). In the current study, the somatic symptoms ($\alpha = .84$) and harmful consequences ($\alpha = .87$) subscales were used as dependent measures, consistent with past work (Farris, Paulus, et al., 2015).

The Barriers to Cessation Scale—(BCS; Macnee & Talsma, 1995). The BCS is a 19-item self-report measure assessing the severity of perceived barriers to successfully quitting smoking. Each item (e.g., "No encouragement or help from friends") is rated on a Likert scale from 0 (*not a barrier/not applicable*) to 3 (*large barrier*) and summed to a total score with greater scores indicating greater/more severe perceived barriers to smoking cessation.

Past work has found good content validity, predictive validity, and internal consistency for the BCS (Garey et al., in press; Macnee & Talsma, 1995). In the current study, internal consistency was good ($\alpha = .89$).

Smoking History Questionnaire—(SHQ; Richard A. Brown, Lejuez, Kahler, & Strong, 2002). The SHQ is a self-report questionnaire used to assess history of smoking (e.g., smoking quantity, age of smoking onset) and problems experienced during previous quit attempts (e.g., weight gain, irritability). As in past work (Buckner et al., 2015), the SHQ was used to describe the sample and to derive a composite score of problems experienced during quit attempts. Each of 16 potential problems was rated on a Likert scale from 1 (*not at all*) to 5 (*extremely*) regarding the severity during quit attempts. The average rating was used to index problems experienced during quit attempts. In the current sample, internal consistency was excellent for the 16 items indexing problems during quit attempts ($\alpha = .93$).

Data Analytic Plan

The explanatory role of anxiety sensitivity (ASI-3), in the relation between anxiety symptoms (MASQ-AA) and (1) threat related smoking abstinence expectancies (SAEQ; somatic and harmful consequences subscales), (2) perceived barriers to cessation (BCS), and (3) severity of problems experienced during past quit attempts (SHQ) was tested using ordinary least squares regression analyses to estimate the indirect association of anxiety symptoms via anxiety sensitivity on the outcome measures (Andrew F Hayes, 2009; Andrew F. Hayes, 2013). To determine significance of the indirect coefficient, 10,000 bootstrapped samples were used to generate a sampling distribution and a 95% confidence interval (CI) around the indirect coefficient; statistical significance of the indirect effect is determined by the absence of zero from the confidence interval. Effect sizes (κ^2) were calculated for the indirect associations (Preacher & Kelley, 2011). In all models, covariates included CPD, participant sex, racial/ethnic minority status (coded as 1 for those identifying as a racial/ ethnic minority and 0 for those who did not), marital status (coded as 1 for those who were married and 0 for those who were not), education attainment (coded as 1 for those who attained more than a high school degree and 0 for those with a high school degree or less), and employment status (coded as 1 for those employed and 0 for those not employed). Last, comparative models were run to evaluate specificity of hypothesized models. For each outcome, an alternative model was run examining indirect associations of anxiety sensitivity via anxiety symptoms (in contrast to the hypothesized model of anxiety symptoms via anxiety sensitivity).

Descriptive Statistics

Bivariate correlations are presented in Table 1. Anxiety symptoms were positively correlated with anxiety sensitivity as well as harmful consequences, somatic symptoms, barriers to cessation, and problems during past quit attempts. Anxiety sensitivity was positively associated with harmful consequences, somatic symptoms, barriers to cessation, and problems during past quit attempts (r= .55, p< .001).

Mediation Analyses

Smoking Abstinence Expectancies—In terms of somatic symptom expectancies, there was a significant total association of anxiety symptoms (B = .38, SE = .06, p < .001). The indirect association of anxiety symptoms via anxiety sensitivity was statistically significant (B = .10, SE = .04, CI [.04, .18], κ^2 = .15). After accounting for anxiety sensitivity, the direct association of anxiety symptoms with somatic symptom expectancies remained significant (B = .28, SE = .06, p < .001).

Regarding harmful consequences, there was a significant total association of anxiety symptoms in relation to harmful consequences expectancies (B=.37, SE=.07, p<.001). The indirect association of anxiety symptoms via anxiety sensitivity was statistically significant (B=.10, SE=.05, CI[.03, .24, κ^2 =.14]). After accounting for anxiety sensitivity, the direct association of anxiety symptoms with harmful consequences expectancies was significant (B=.26, SE=.07, p<.001).

Perceived Barriers to Cessation—For perceived barriers to cessation, there was a significant total association of anxiety symptoms (B = .23, SE = .09, p = .014). The indirect association of anxiety symptoms via anxiety sensitivity was also significant (B = .17, SE = .07, CI[.05, .31], $\kappa^2 = .16$). However, there was not a statistically significant direct association of anxiety symptoms with perceived barriers after accounting for anxiety sensitivity (B = .06, SE = .10, p = .054).

Problems Experienced During Past Quit Attempts—Regarding harmful consequences, there was a significant total association of anxiety during past quit attempts, there was a significant total (B = .03, SE = .01, p = <.001) association of anxiety symptoms. The indirect association of anxiety symptoms via anxiety sensitivity was also significant (B=.01, SE=.01, CI[.01, .02], $\kappa^2 = .12$). The direct association of anxiety symptoms with problems experienced during past quit problems was also significant (B=.02, SE=.01, p=.004).

Specificity Tests

To further strengthen interpretation of results, anxiety symptoms and anxiety sensitivity variables were reversed for each model with a significant indirect association. For somatic symptom expectancies, there was a significant indirect association of anxiety sensitivity via anxiety symptoms (B=.11, SE=.04, CI[.05, .20], $\kappa^2=.22$). In terms of harmful consequence expectancies, the indirect association of anxiety sensitivity via anxiety symptoms was significant (B=.10, SE=.04, CI[.04, .20], $\kappa^2=.18$). For barriers to cessation the indirect association of anxiety sensitivity via anxiety symptoms was not significant (B=.02, SE=.04, CI[-.06, .11], $\kappa^2=.03$). Finally, in terms of problems experienced during past quit attempts, the indirect association of anxiety sensitivity via anxiety symptoms was significant (B=.01, SE=.01, SE=.01, SE=.01, SE=.01.

Discussion

The present study examined whether anxiety sensitivity explains, in part, the relation between anxiety symptoms and an array of clinically-relevant smoking processes among low-income smokers. Results indicated that anxiety exerted a significant indirect effect through anxiety sensitivity for threat-related smoking abstinence expectancies (somatic symptoms and harmful consequences), perceived barriers for cessation, and problems experienced during past quit attempts. Notably, the indirect effects were of medium size for all four outcomes tested (κ^2 = .12–.16). These results are consistent with the hypothesis that anxiety symptoms are related to anxiety sensitivity (the 'a' path) which is related to a variety of smoking maintenance processes (the 'b' path). Notably, the observed indirect effects were evident after adjusting for the influence of factors known to correlate with the severity of smoking behavior and anxiety states, including CPD, participant sex, racial/ethnic minority status, marital status, education attainment, and employment status.

Comparative models were run to evaluate the specificity of hypothesized models. For barriers to cessation, the alternative model was rejected, adding support to the hypothesized pathway. However, these findings revealed that a bi-directional explanatory process is likely to be involved in relation to threat-related smoking abstinence expectancies and problems experienced during past quit attempts. To more fully explore the nature of relations among these variables over time, future prospective modeling of the temporal ordering of anxiety symptoms, anxiety sensitivity, and smoking processes is warranted. Specifically, anxiety symptoms, particularly anxious arousal, may impact anxiety sensitivity over time, increasing catastrophic interpretations of arousal sensations. This process may result in hypervigilance for physiological sensations experienced because of anxiety and/or smoking cessation and may result in increased threat-related expectancies of smoking cessation, more perceived barriers to quitting, and greater problems successfully quitting.

The findings from the present investigation may serve to conceptually inform the development of specialized intervention strategies for low-income smokers. Specifically, among low-income smokers, it may be advisable to understand and clinically address anxiety states and anxiety sensitivity to enhance psychological flexibility related to maladaptive smoking cognitions (e.g., "I need a cigarette to cope with my (stressful) life") and facilitate change in smoking behavior. For example, anxiety sensitivity reduction techniques, based upon cognitive behavioral interventions (Smits, Berry, Tart, & Powers, 2008), have shown promise in reducing anxiety sensitivity among those with and without psychopathology (Keough & Schmidt, 2012) and promoting abstinence among smokers (Zvolensky, Bogiaizian, Salazar, Farris, & Bakhshaie, 2014). Recent work also has demonstrated that it is possible to reduce anxiety sensitivity among smokers via brief theoretically-driven intervention tactics (Schmidt, Raines, Allan, & Zvolensky, 2016). It is possible that tailoring these treatments to low-income smokers would offer a novel and more personalized approach to improve mental health and change smoking behavior.

There are several interpretive caveats to the present study. First, given the cross-sectional nature of these data, causal relations cannot be explicated. The present tests were based on a theoretical framework supported by extant empirical data, but 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 low-income 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. Third, as the key variables were assessed via self-report, there is the possibility that the observed relations were in part a function of shared method variance. Future research would benefit by employing a multi-method assessment approach to cross-index the nature of the relations observed in the current report.

Overall, the present study serves as an initial investigation into the nature of the associations between anxiety symptoms, anxiety sensitivity, and smoking processes among low-income smokers. Future work is needed to explore the extent to which anxiety sensitivity accounts for relations between anxiety symptoms and other smoking processes (e.g., withdrawal, cessation outcome) to further clarify theoretical health disparity models involving these comorbid conditions.

on M; b = Effect of M on Y_i ; c' = Direct effect of X on Y_i controlling for M.

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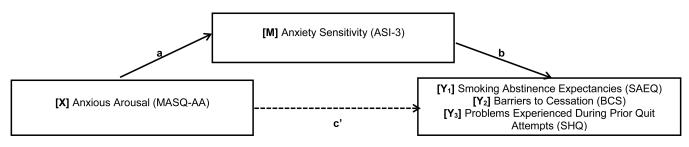


Figure 1. Proposed model *Note*: a = Effect of X

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Table 1

Bivariate correlations among study variables

Variable	M(SD)/N[%]	1	2	3	4	5	9	7	8	9	10	11
1. MASQ-AA	29.8 (13.7)	-										
2. ASI-3	23.1 (18.6)	.51 **										
3. SAEQ-Somatic	11.5 (9.4)	.58**	.51 **	1								
4. SAEQ-Harmful	12.7 (10.3)	.50**	.46 **	.76**	ı							
5. BCS	28.3 (12.5)	.25*	.38**	.36**	.43 **	1						
6. Quit Problems	2.3 (0.9)	** 54.	.41 **	.40 **	.43 **	.41 **						
7. CPD	15.6 (9.0)	.14	.05	.22*	.07	02	.03	,				
8. Sex (% female)	32 [31.7]	.05	.03	.01	.17	.14	.20*	02	-			
9. Minority status (% racial/ethnic minority)	74 [73.3]	.05	.07	.13	.11	90.	.04	12	02	1		
10. Marital status (% married)	10 [9.9]	.01	02	.17	.16	.02	10	.04	90.	.05	-	
11. Education (% greater than high school education)	53 [52.5]	.02	17	16	12	01	80.	04	.01	13	22*	-
12. Employment (% employed)	41 [40.6]	07	04	10	13	60.	11	05	13	60.	90°	.02
							1					l

Expectancies Questionnaire; Somatic = Somatic Expectancies Subscale; Harmful Consequences Subscale; BCS = Barriers to Cessation Scale; Quit Problems = Problems Experienced During Note: Note: M = Mean, SD = Standard Deviation; MASQ-AA = Mood Anxiety Symptom Questionnaire, Anxious Arousal subscale; ASI-3 = Anxiety Sensitivity Index-3; SAEQ = Smoking Abstinence Past Quit Attempts; Sex, coded as Male = 0 and Female = 1, with descriptive statistics for number and percentage female; Minority Status, coded as Non-Racial/Ethnic Minority = 0 and Racial/Ethnic Minority = 1; Marital Status, coded as unmarried = 0 and married = 1; Education, coded as high school degree or less = 0 and more than high school= 1; Employment, coded as unemployed = 0 and employed = 1.

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

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Table 2

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Unstandardized Coefficients

Y	Model	R^2	В	SE	t	p	CI(I)	CI(u)	
1	$MASQ-AA \rightarrow ASI-3 (a)$.30	.70	.12	.782	<.001	.46	.94	
	$ASI-3 \rightarrow SAEQ-SOM (b)$.47	.14	50.	3.01	.003	50.	.23	
	$MASQ-AA \rightarrow SAEQ-SOM (c)$.38	90°	06'9	<.001	72.	.49	
	$MASQ-AA \rightarrow SAEQ-SOM (c')$.28	90°	4.58	<.001	.16	.41	
	$MASQ-AA \rightarrow ASI-3 \rightarrow SAEQ-SOM (ab)$.10	.04			.04	.18	
2	$ASI-3 \rightarrow SAEQ-HAR$ (b)	.37	.15	.05	2.72	800°	.04	.26	
	MASQ-AA →SAEQ-HAR (c)		.37	.07	5.59	<.001	.24	.50	
	$MASQ-AA \rightarrow SAEQ-HAR (c')$.26	.07	3.53	<.001	.11	.41	
	$MASQ\text{-}AA \rightarrow ASI\text{-}3 \rightarrow SAEQ\text{-}HAR \text{ (ab)}$.10	.05			.03	.24	
3	$ASI-3 \rightarrow BCS (b)$.19	.24	80.	3.20	.011	60.	.39	
	$MASQ-AA \rightarrow BCS (c)$.23	60:	2.51	.014	.05	.41	
	$MASQ-AA \rightarrow BCS (c')$.06	.10	.593	.554	14	.15	
	$MASQ-AA \rightarrow ASI-3 \rightarrow BCS$ (ab)		.17	.07			.05	.31	
4	ASI-3 \rightarrow Problems (b)	.30	.01	.01	2.48	.014	.01	.02	
	$MASQ-AA \rightarrow Problems (c)$.03	.01	4.82	<.001	.02	.04	
	$MASQ-AA \rightarrow Problems (c^*)$.02	.01	2.95	.004	.01	.03	
	MASQ-AA \rightarrow ASI-3 \rightarrow Problems (ab)		.01	.01			.01	.02	

through mediator. Path a is equal in all models, therefore, it presented only in model 1. The standard error and 95% CI for ab are obtained by bootstrap with 10,000 re-samples. MASQ-AA (Anxious Arousal) is the predictor in all models. ASI-3 (Anxiety Sensitivity) is the mediator in all models. SAEQ-SOM (Somatic Expectancies), SAEQ-HAR (Harmful Expectancies), BCS (Barriers to Cessation), Note, a = Association of X with M; b = association of M with Y₁; c = Total association of X with Y₁; c = Direct association of X with Y₁ controlling for M; ab = Indirect effect of predictor on outcome Problems (during quit) are the outcome variable in models 1–4, respectively. CI (lower) = lower bound of a 95% confidence interval; CI (upper) = upper bound; → = association