

ORIGINAL ARTICLE

Anxiety sensitivity as a mediator of the association between asthma and smoking

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*Department of Psychology, University of Cincinnati, Cincinnati, OH, USA***Abstract**

Objective: Despite its negative effects, smoking is more common among individuals with asthma compared to those without. Anxiety sensitivity (fear of arousal-related sensations) is associated with both smoking and asthma; however, no research, to date, has examined the interplay between these three factors. Thus, the purpose of the current study was to evaluate the mediating role of anxiety sensitivity in the association between asthma diagnosis and smoking status. **Methods:** The current study was a secondary analysis of data from three existing datasets of non-smokers and smokers with and without asthma ($n=433$; 56.3% female, $M_{\text{age}}=34.01$ years, $SD=13.9$). Participants provided information on their asthma diagnosis status and smoking status and completed self-report measures. **Results:** As hypothesized, after controlling for gender, race and age, there was a significant indirect effect of asthma diagnosis on smoking status through anxiety sensitivity (95% CI = 0.07–0.48). **Conclusions:** These results indicate that the association between asthma diagnosis and smoking status appears to be driven, in part, by anxiety sensitivity and suggest that anxiety sensitivity may serve as an important target for prevention and intervention efforts for smokers with asthma.

Keywords

Anxiety sensitivity, asthma, cigarette smoking, statistical mediation, tobacco

History

Received 23 August 2014

Revised 13 October 2014

Accepted 1 November 2014

Published online 24 November 2014

Introduction

One in 12 adults in the US currently suffer from asthma, a reversible obstructive airway disease that consists of chronic airway inflammation and episodes of exacerbations in response to certain stimuli [1]. Despite the known negative effects of smoking on lung function and health, it has been well established that smoking is more common among individuals with asthma compared to those without [2–5]. Indeed, compared to individuals without asthma, individuals with asthma are 1.36 times more likely to be a smoker and nearly three times more likely to be nicotine dependent [4,6]. Overall, it is estimated that 21% of asthma patients in the US are current smokers, compared to a smoking rate of 18% in the general US population [7,8]. In addition, individuals with asthma are more likely to be regular smokers and heavier smokers than those without asthma [9]. Not surprisingly, research has consistently found that smoking results in poorer asthma control and more frequent and severe asthma exacerbations [4,10,11]. Furthermore, compared to non-smokers with asthma, smokers with asthma experience greater

symptom severity, increased levels of daily asthma symptoms and greater asthma-related interference with daily activities [12–14]. Smoking has also been found to significantly decrease the effectiveness of inhaled corticosteroids [15].

Although smoking is more common among individuals with asthma, relatively little is known about factors that influence the asthma–smoking association. In terms of smoking behavior, extant research indicates that there are no differences between smokers with and without asthma in terms of daily smoking rate, level of nicotine dependence, stage of change in terms of readiness to quit smoking and smoking topography [16–18]. Avallone and colleagues, however, did find that among smokers, asthma diagnosis was a significant predictor of younger age of smoking onset and a greater number of quit attempts [16]. Similarly, there are relatively few differences between smokers with and without asthma in terms of smoking-related cognitive processes. The only differences appear to be that smokers with asthma are more likely to report believing that their current and future health will be negatively impacted by smoking [17] and motives for quitting related to self-control, but not health concerns [16]. Given these similarities, factors other than those related to smoking may hold greater promise for helping to explain the smoking–asthma association.

As asthma and smoking both commonly co-occur with panic psychopathology (i.e. panic attacks, panic disorder and agoraphobia) [19–21], examination of the role of panic-related cognitive risk factors is an important next step in

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elucidating the nature of the asthma–smoking association. One such risk factor is anxiety sensitivity, defined as the fear of arousal-related physical and psychological sensations [22,23]. Anxiety sensitivity reflects a relatively stable, albeit malleable, cognitive pre-disposition that is theoretically and empirically distinct from the tendency to experience negative emotional states [22]. When anxious, individuals high in anxiety sensitivity become acutely fearful due to beliefs that these anxiety-related interoceptive sensations have harmful physical, social or psychological consequences [24]. In line with this theory, anxiety sensitivity is concurrently and prospectively associated with an increased risk of anxiety symptoms and with the onset of certain anxiety disorders (e.g. panic attacks, panic disorder and post-traumatic stress disorder) [25–27].

Extant research on anxiety sensitivity and smoking has demonstrated that anxiety sensitivity plays an important role in both smoking behavior and cognitive-based smoking processes. For example, anxiety sensitivity is primarily associated with smoking to reduce negative affect [28] and expectancies that smoking will result in negative affect reduction [29]. In terms of smoking cessation, anxiety sensitivity is associated with increased motivation to quit [30] and a greater number of quit attempts [31] as well as decreased self-efficacy in refraining from smoking when emotionally distressed [32] and greater perceived barriers to cessation [29,33]. Further, anxiety sensitivity has been found to predict greater intensity of nicotine withdrawal symptoms and earlier lapse and relapse during a quit attempt [34,35].

There is a growing body of research that indicates anxiety sensitivity also plays an important role in asthma. Anxiety sensitivity has been found to predict panic in response to asthma symptoms [36] as well as increased activity limitations due to asthma symptoms and poorer asthma-related physical and emotional health [37]. In an undergraduate sample of individuals with self-reported, physician-diagnosed asthma, McLeish, Zvolensky and Luberto found that the physical concerns facet of anxiety sensitivity, but not the cognitive or social concerns facets, significantly predicted poorer levels of asthma control independent of the effects of gender, daily cigarette smoking and negative affectivity [38]. Similarly, Avallone, McLeish, Luberto and Bernstein found that the physical concerns facet of anxiety sensitivity significantly predicted poorer asthma control and asthma-related quality of life above and beyond the effects of gender, age, negative affectivity and number of comorbid medical problems in a community sample of individuals with a physician-verified asthma diagnosis [39].

As research has consistently found that anxiety sensitivity is associated with both smoking and asthma, anxiety sensitivity may help to explain the association between smoking and asthma. That is, smoking may be associated with asthma due to its association with anxiety sensitivity and the association between anxiety sensitivity and asthma. Despite evidence of these associations, no research, to date, has examined the interplay between these anxiety sensitivity, smoking and asthma. Thus, the overarching purpose of this investigation was to evaluate the mediating role of anxiety sensitivity in the association between asthma diagnosis and smoking status. It was hypothesized that after controlling for

relevant demographic covariates, anxiety sensitivity would mediate the relationship between asthma diagnosis and smoking status.

Methods

Participants and procedure

The current study was a secondary analysis of data combined from three existing datasets. Four distinct groups were created based on asthma status and smoking status: (1) non-smokers without asthma; (2) non-smokers with asthma; (3) smokers with asthma; and (4) smokers without asthma. Demographic information and study procedures for each group are described separately below. All participants completed the same self-report measures, with the exception of smoking-specific and asthma-specific measures. The Institutional Review Board approved all study procedures and materials prior to data collection.

Non-smokers without asthma

Participants in this group were 113 non-smoking undergraduate students without a current or past history of asthma (74.1% female, $M_{\text{age}} = 20.32$ years, $SD = 3.7$). The racial composition of the sample was 85% Caucasian, 8.8% African American, 5.3% Asian and 0.9% other.

Undergraduate students in Introductory Psychology courses at a large, urban university who were over the age of 18 were invited to participate in a study investigating the relationship between exercise-induced bronchoconstriction and psychological factors (e.g. depression, anxiety). Students interested in participating in the study were first screened for eligibility via email. Individuals who reported never having received a diagnosis of asthma or exercise-induced asthma and denied current cigarette smoking were deemed eligible for this study. Eligible participants were then given a link and password to complete measures online. Participants were then able to complete the questionnaires at their convenience and submit them anonymously via Survey Monkey. Such web-based techniques can provide a valid and reliable approach to data collection, comparable to the in-person administration of self-report measures [40]. After completing the study, participants contacted study personnel to receive course credit for their participation. The use of an undergraduate sample as a control group is consistent with previous studies of anxiety sensitivity [23,41,42]. Further, scores on the Anxiety Sensitivity Index-3 (ASI-3) (see “Measures” section below for more information on this measure) for this sample ($M = 13.3$, $SD = 12.1$) did not differ significantly from non-clinical population norms ($M = 12.8$, $SD = 10.6$) [23].

Non-smokers with asthma

Participants in this group were 121 patients (75% female, $M_{\text{age}} = 43.64$ years, $SD = 12.33$) recruited from a community allergy and asthma office who had been seen for a physician follow-up visit between 1 January 2009 and 1 January 2010 for a study examining the relationship between psychological factors and asthma. The racial composition of this group was 80.8% Caucasian, 9.2% African American, 1.7% Asian and

8.3% other. In terms of asthma control, participants reported a mean Asthma Control Test (ACT) [43] score of 19.88 ($SD = 4.30$), indicating well-controlled asthma.

To be included in the non-smokers with asthma group, participants had to (1) have a diagnosis of asthma; (2) be between the ages of 18 and 65; and (3) be a non-smoker. Using these criteria, a total of 368 potential participants were identified. Study materials were then mailed out to these individuals. Interested participants provided written informed consent, completed self-report measures, provided their contact information to receive compensation and then mailed all materials back to the asthma and allergy office in a self-addressed stamped envelope that was provided for them. Participants were then mailed a \$25 gift card as compensation for their time and effort. A total of 127 questionnaire packets were returned (34.5% response rate).

Smokers with and without asthma

In total, 209 regular daily smokers between the ages of 18 and 65 were recruited from the community to participate in a study on smoking and asthma: 83 smokers with self-reported, physician-diagnosed asthma and 126 smokers without a current or past history of asthma. Participants were recruited via newspaper ads (online and print) and flyers posted throughout the community. Eligibility criteria for the study were: (1) daily smoking rate greater than 10 cigarettes per day; (2) expired carbon monoxide levels greater than 10 parts per million (ppm); and (3) regular, daily smoking for at least 1 year. Additional eligibility criteria for the smokers with asthma were: (1) asthma diagnosis received prior to smoking initiation; (2) current prescription for asthma medication; and (3) the presence of asthma symptoms in the last year. After providing informed, written consent, participants' smoking status was biochemically verified via expired CO analysis. Then, participants completed a battery of self-report measures. Participants received \$30 as compensation for their time and effort.

The smokers without asthma group was 29.6% female and, on average, 36.5 ($SD = 13.05$) years old. The racial composition of the smokers without asthma group was 69.8% Caucasian, 26.2% African-American and 4% other. Smokers without asthma smoked, on average, 18.52 cigarettes per day ($SD = 8.7$) and had been regular smokers for an average of 18.31 ($SD = 12.7$) years. The smokers with asthma group was 45.1% female and, on average, 34.2 ($SD = 11.5$) years old. The racial composition of the smokers with asthma group was 37% Caucasian, 60.5% African-American, 1.2% Asian and 1.2% other. Smokers with asthma smoked on average 20.55 cigarettes per day ($SD = 11.9$) and had been regular smokers for an average of 20.5 ($SD = 11.8$) years. Smokers with asthma reported a mean ACT [43] score of 15.61 ($SD = 4.2$), indicating difficulties with asthma control.

Measures

Expired carbon monoxide

For the smokers with and without asthma, biochemical verification of smoking status was completed by carbon monoxide (CO) analysis of breath samples assessed using a

Bedfont Micro 4 Smokerlyzer CO Monitor (Model EC50; Bedfont Scientific USA, Williamsburg, VA). Research indicates that 10 ppm is an optimal cutoff score for reliably discriminating smoking status [44]. Values obtained above this cutoff were considered indicative of smoking.

Asthma control test

The asthma control test (ACT) [43] is a five-item self-report measure that assesses asthma control. The ACT measures frequency of symptoms (e.g. How often have you had shortness of breath?) and functional impairment due to symptoms (e.g. How much of the time did your asthma keep you from getting as much done at work or at home?) within the past 4 weeks. Scores range from 5 to 25 with higher scores indicating better control. Scores at or above 19 indicate that an individual's asthma is well controlled. The ACT shows good reliability and is able to discriminate between groups of patients with different levels of asthma control [43]. The ACT was used for descriptive purposes in the smokers with asthma and non-smokers with asthma groups only and was not employed in the analyses.

Smoking history questionnaire

Smoking history and pattern was assessed with the smoking history questionnaire (SHQ) [45], which includes items pertaining to smoking status, smoking rate, age of onset at initiation and years of being a daily smoker. The SHQ has been successfully used in previous studies and has been identified as a psychometrically sound descriptive measure of smoking history [46]. The SHQ was used for descriptive purposes in the smokers with and without asthma groups only and was not employed in the analyses.

Anxiety sensitivity index-3

The Anxiety sensitivity index-3 (ASI-3) [23] was administered to assess anxiety sensitivity. The ASI-3 is an 18-item measure that asks respondents to rate on a five-point Likert scale (0 = very little to 4 = very much), the degree to which they fear negative consequences stemming from anxiety symptoms. The ASI-3 is comprised of one higher-order anxiety sensitivity factor and three specific lower-order factors [23]. The three lower-order factors consist of physical (e.g. It scares me when my heart beats rapidly), social (e.g. I worry that other people will notice my anxiety) and cognitive (e.g. When my thoughts seem to speed up, I worry that I might be going crazy) concerns. The ASI-3 has demonstrated that the strongest psychometric properties of any current measure of anxiety sensitivity [23]. Internal consistency for the ASI-3 in the current study was good (range across groups: 0.90–0.95).

Analytic approach

One-way analysis of variance (ANOVA) or Chi-square tests as appropriate were used to determine whether there were any significant group differences on demographic variables (i.e. gender, race and age) that needed to be accounted for in subsequent analyses. There were significant differences between groups with regard to gender [$\chi^2(3) = 71.86$,

$p < 0.001$] and race [$\chi^2(9) = 105.69$, $p < 0.001$], with more males in the smokers with and without asthma groups and more African-Americans in the smokers with asthma group. There were also significant group differences in terms of age [$F(3,435) = 90.50$, $p < 0.001$]. More specifically, non-smokers with asthma were older and non-smokers without asthma were younger than all other groups. Thus, gender, race and age were added as covariates in all subsequent analyses. Zero-order correlations were then computed to assess the associations between the independent variable (asthma diagnosis), covariates, the proposed mediator (anxiety sensitivity) and the primary dependent variable (smoking status). The mediation analyses for this study followed recent recommendations, which propose directly testing the indirect effect and do not require the presence of a significant direct effect to determine the presence of mediation [47–49].

Results

Descriptive data and zero-order associations

Refer Table 1 for descriptive data and sample characteristics. The associations among covariates, the proposed mediator, the independent variable and the dependent variable are presented in Table 2. Both asthma diagnosis and smoking status were each significantly correlated with gender, age and anxiety sensitivity. Anxiety sensitivity was not significantly associated with age or gender.

Mediation analyses

Using PROCESS [50] in SPSS, OLS regressions (or logistic regressions as appropriate) were conducted to estimate the following models to obtain the total, direct and indirect effects of asthma diagnosis on smoking status through anxiety sensitivity. Covariates for these analyses included gender, race and age and these were entered in the models simultaneously with predictor variables.

Model 1 represents the total effect of asthma diagnosis on smoking status after controlling for gender, race and age. Results indicate that the log odds of being a smoker were lower among individuals with asthma compared to those without asthma (Table 3, Model 1 and Figure 1 path c). Model 2 represents the effect of asthma diagnosis on anxiety sensitivity after controlling for gender, race and age; having an asthma diagnosis significantly predicted higher levels of anxiety sensitivity (Table 3, Model 2 and Figure 1 path a).

Model 3 represents the direct effect of asthma diagnosis on smoking status after accounting for the influence of anxiety sensitivity, gender, race and age. Anxiety sensitivity was positively associated with the log odds of being a smoker (Table 3, Model 3 and Figure 1 path b), and the direct effect of asthma diagnosis (Table 3, Model 3 and Figure 1 path c') was negatively associated with smoking status after accounting for the effects of anxiety sensitivity, gender, race and age.

The indirect effect was calculated by multiplying the path *a* coefficient obtained from the OLS regression analysis for asthma diagnosis predicting anxiety sensitivity by the path *b* coefficient obtained from the logistic regression analysis for anxiety sensitivity predicting smoking status: $ab = 4.82 \times 0.05 = 0.24$. Using 10 000 bias-corrected bootstrapped

Table 2. Intercorrelations among covariates, proposed mediator, independent, and dependent variables.

	1	2	3	4	5
(1) Gender ^a	–	–0.02	0.02	0.12 ^d	–0.39 ^d
(2) Age	–	–	–0.02	0.39 ^d	–0.11 ^e
(3) AS	–	–	–	0.15 ^d	0.27 ^d
(4) Asthma diagnosis ^b	–	–	–	–	–0.12 ^e
(5) Smoking status ^c	–	–	–	–	–

^aGender: 1 = male, 2 = female.

^bAsthma diagnosis: 0 = no asthma diagnosis, 1 = asthma diagnosis.

^cSmoking status: 0 = non-smoker, 1 = smoker.

^d $p < 0.01$.

^e $p < 0.05$.

Table 3. Logistic regression and ordinary least squares regression model coefficients.

Outcome	Model 1 Smoking status	Model 2 AS ^a	Model 3 Smoking status
Constant	1.46 ^e	16.74 ^c	0.80
Asthma diagnosis	–0.90 ^e	4.82 ^c	–1.18 ^c
AS			0.05 ^c
Gender	–1.69 ^c	–0.30	–1.83 ^c
Race	0.46 ^c	2.80 ^c	0.34 ^d
Age	0.02 ^c	–0.11 ^d	0.03 ^c
R ²	0.26	0.05	0.36

Models 1 and 3 contain unstandardized logistic regression coefficients with Nagelkerke R^2 and Model 2 contains unstandardized ordinary least squares (OLS) regression coefficients with OLS R^2 .

^aAS, anxiety sensitivity.

^c $p < 0.01$.

^d $p < 0.05$.

Table 1. Demographic characteristics of study sample.

	Total sample	Non-smokers without asthma	Non-smokers with asthma	Smokers with asthma	Smokers without asthma	Test statistic (df)
Age (years)	34.01 (13.88)	20.32 (3.72)	43.64 (12.33)	34.16 (11.54)	36.51 (13.05)	$F(3, 435) = 90.50^a$
Gender (female)	192 (56%)	29 (74%)	30 (75%)	45 (45%)	88 (30%)	$\chi^2(3) = 71.86^a$
Race						$\chi^2(9) = 105.69^a$
Caucasian	311 (71%)	96 (85%)	97 (81%)	30 (37%)	88 (70%)	
Afr. Am.	103 (23%)	10 (9%)	11 (9%)	49 (61%)	33 (26%)	
Asian	9 (2%)	6 (5%)	2 (2%)	1 (1%)	0 (0%)	
Other	17 (4%)	1 (1%)	10 (8%)	1 (1%)	5 (4%)	
AS	18.74 (14.67)	13.27 (12.11)	16.63 (13.47)	28.29 (17.31)	19.62 (13.07)	

Values in the table reflect M (SD) or n (%), and p values are based on results from one-way ANOVA or Pearson chi-square tests. Afr. Am., African-American; AS, anxiety sensitivity.

^a $p < 0.01$

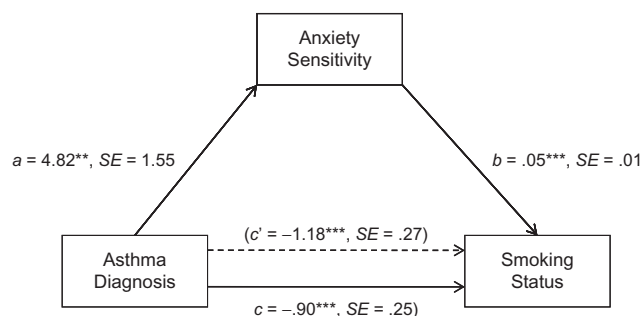


Figure 1. Path coefficients for simple mediation analysis with asthma diagnosis as the independent variable, anxiety sensitivity as the proposed mediator and smoking status as the dependent variable. Dotted line denotes the effect of asthma diagnosis on smoking status when anxiety sensitivity is included as a mediator. a is an unstandardized OLS regression coefficient, b , c , and c' are unstandardized logistic regression coefficients. Gender, race and age were included as covariates but are not represented here. ** $p < 0.01$, *** $p < 0.001$.

samples calculated with PROCESS, the resulting confidence interval (95% CI = 0.07–0.48) did not include zero, suggesting a significant indirect effect of asthma diagnosis on smoking status through anxiety sensitivity. That is, anxiety sensitivity serves as a mediator of the association between asthma diagnosis and smoking status.

Discussion

There has been a growing level of interest in recent years in better understanding the unexpected association between asthma and smoking. Research efforts focused on examining smokers with and without asthma, however, have found few differences in smoking behavior and smoking-related cognitive processes. Thus, there is a need to identify other factors, or so-called “third variables”, that may influence this association. Understanding the role of these third variables is critically important in creating a theoretical model of these associations, which currently does not exist. Such an examination would also assist in identifying specific targets for prevention and intervention efforts among individuals with asthma.

Given the individual associations between anxiety sensitivity and both asthma and smoking, this study aimed to evaluate the mediating role of anxiety sensitivity in the asthma diagnosis-smoking status association. Results of the current study indicate that, as hypothesized, there was a significant indirect effect of asthma diagnosis on smoking status through anxiety sensitivity. In other words, the association between asthma diagnosis and smoking status appears to be driven, in part, by anxiety sensitivity. Asthma diagnosis was a significant predictor of anxiety sensitivity, which, in turn, significantly predicted an increased likelihood of being a smoker. It should be noted that these significant effects were found even after controlling for the effects of gender, race and age. These results suggest that because individuals with asthma tend to be more fearful of anxiety-related sensations (i.e. high anxiety sensitivity), they may find asthma-related physical sensations (e.g. shortness of breath) particularly aversive, resulting in greater vigilance to and catastrophizing about such symptoms ultimately resulting in greater overall anxiety. These anxiety symptoms are also

experienced as aversive, thus individuals with asthma may turn to smoking as an affect regulation strategy despite the negative impact on lung function. Unfortunately, nicotine withdrawal produces both aversive physical and affective symptoms, making smoking cessation difficult as well. While the current study provides preliminary support for this theorizing, the full model of these associations is still in need of empirical support.

Interestingly, the association between asthma diagnosis and smoking status, although significant, was not in the expected direction; that is, individuals with asthma were less likely to smoke compared to those without asthma. This finding is in contrast to a large body of literature documenting a greater likelihood of smoking among individuals with asthma [2–6]. One possible explanation for this unexpected finding may be due to the sampling procedures used in this study, which utilized data from individuals recruited specifically based on the presence or absence of smoking and asthma, and therefore, do not accurately reflect the prevalence rates of smoking and asthma found in the general population. However, these potential sampling issues do not negate the current findings, as the statistical approach to testing these indirect effects does not require there to even be a significant direct effect. In fact, one could argue that in attempting to understand these indirect effects, the nature of the direct effect is not important. Clearly, the asthma–smoking relationship is a complex one, and examination of indirect pathways is critical in better understanding the nature of these associations.

While this study contributes to and extends previous work on asthma and smoking, there are a number of interpretive caveats that warrant consideration. First, the sample consisted of participants drawn from several different studies using different recruitment methods and study procedures. Although this study fills a gap in the existing literature and is the first to examine the interrelationships between asthma, smoking and anxiety sensitivity, these findings need to be replicated in a single sample that includes smokers and non-smokers with and without asthma. Along these same lines, the non-smokers without asthma group consisted of undergraduate students while all other groups consisted of participants recruited from the community. This method is consistent with previous research on anxiety sensitivity that has used undergraduate control groups [24,41,42]. Further, anxiety sensitivity levels in our undergraduate sample did not significantly differ from community norms, lending greater confidence to the results of the current study. Nevertheless, future work in this area should aim to recruit participants from the community to decrease demographic differences and increase the generalizability of the findings.

Second, for the smokers with asthma group, asthma diagnosis was assessed via self-report and was not verified by objective measures of lung function (e.g. spirometry). While efforts were made to verify asthma diagnosis with proxy indicators (e.g. experiencing symptoms in last year, current prescription medication), it is possible that some of the individuals within that group did not truly have asthma. Thus, future studies should utilize objective measures of lung function and/or physician exam to verify asthma diagnosis. Third, while biochemical verification of smoking status was

used to verify smoking status, it was not utilized to verify non-smoking status in the non-smokers with and without asthma groups. In general, non-smokers tend to accurately report their smoking status [51,52]; however, future work should biochemically verify both smoking and non-smoking to minimize misclassification of smoking status.

Fourth, current medications were not comprehensively assessed in this study. Such medications (e.g. psychiatric and sympathomimetics) could impact one's emotional state; thus, it will be important for future studies to take such medications into consideration. An additional limitation of this study is that it was cross-sectional in nature and, as a result, no conclusions regarding causation or temporal pattern can be made. Similarly, this study did not assess asthma, smoking and anxiety sensitivity at multiple time points, which limits our ability to understand how these variables may change or impact one another over time. The use of ecological momentary assessment procedures, longitudinal designs or prospective studies would allow for the examination of how these variables relate to one another and develop over time. Finally, while this study found that anxiety sensitivity mediates the relationship between asthma diagnosis and smoking status, it is likely that there are other mediators in this association as well. Examining other possible key variables (e.g. distress tolerance, emotion regulation) in this association would improve our understanding of this association and would help further develop the model of these associations described above.

Despite these limitations, there are a number of clinical implications for both prevention and intervention efforts that can be drawn from the findings of this study. For example, physicians could assess anxiety sensitivity during asthma-related office visits to identify asthma patients at risk for smoking onset due to high levels of anxiety sensitivity. This may be particularly helpful for children and adolescents to intervene prior to smoking initiation. Similarly, assessing anxiety sensitivity in individuals with asthma who already smoke could identify smokers in need of additional support during smoking cessation or specialized cessation interventions. Indeed, research has shown that traditional cognitive-behavioral treatments for panic disorder, particularly interoceptive exposure techniques, are effective in reducing anxiety sensitivity [53], and exposure-based techniques have also been successfully used to improve smoking cessation outcomes among high anxiety sensitive individuals [54]. It will, however, be important for future work to identify ways in which these interventions need to be tailored for asthma patients. For example, it may be necessary to incorporate an asthma psychoeducation component into treatment to address asthma-related concerns and to help these individuals recognize differences between asthma and anxiety symptoms.

Conclusions

In conclusion, the present findings suggest a significant indirect effect of asthma diagnosis on smoking status through anxiety sensitivity. The primary implication of these findings is that anxiety sensitivity may serve as an explanatory mechanism underlying the asthma–smoking association.

The identification of such mechanisms is clinically important as it helps to refine our understanding of complex associations between chronic illness, addictive behaviors and psychopathology.

Declaration of interest

The authors do not have any conflicts of interest to disclose. The authors alone are responsible for the content and writing of the paper.

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