I, Kimberly M. Avallone, hereby submit this original work as part of the requirements for the degree of Doctor of Philosophy in Psychology.

It is entitled:
Anxiety Sensitivity as a Mediator of the Association between Asthma and Smoking

Student’s name: Kimberly M. Avallone

This work and its defense approved by:

Committee chair: Alison Mcleish, Ph.D.

Committee member: Christine Hovanitz, Ph.D.

Committee member: Farrah Jacquez, Ph.D.
Anxiety Sensitivity as a Mediator of the Association between Asthma and Smoking

A dissertation submitted to the
Division of Graduate Education and Research
of the University of Cincinnati

in partial fulfillment of the
requirements for the degree of

Doctorate of Philosophy

in the Department of Psychology
of the College of Arts and Sciences
March 2013
by
Kimberly M. Avallone
B.S., University of Florida, 2008
M.A., University of Cincinnati 2011

Committee: Alison C. McLeish, Ph.D. (chair)
Christine A. Hovanitz, Ph.D.
Farrah M. Jacquez, Ph.D.
Abstract

Despite the known compromising effects of smoking on lung function and health, smoking is more common among individuals with asthma compared to those without, resulting in increased asthma symptom severity and poorer asthma control. Overall, few differences exist between smokers with and without asthma in terms of smoking behavior and smoking-related cognitive processes, thus, it is likely that there are other key factors that may help explain the association between smoking and asthma. One such factor that has received increasing empirical attention in relation to both smoking and asthma is anxiety sensitivity (AS), defined as the fear of arousal-related physical and psychological sensations. AS has been found to be associated with negative affect reduction smoking motives and greater difficulties with smoking cessation as well as poorer asthma control, and asthma-related quality of life. As research has consistently found that AS is associated with both smoking and asthma, AS may help to explain the association between smoking and asthma. Thus, the purpose of the current study was to evaluate the mediating role of AS in the association between asthma diagnosis and smoking status. The study sample consisted of four distinct groups based on asthma status and smoking status created with data from three existing datasets (N = 485): (1) 113 non-smokers without asthma (74.1% female, \(M_{age} = 20.3\) years, \(SD = 3.7\)); (2) 121 non-smokers with asthma (75% female, \(M_{age} = 43.6\) years, \(SD = 12.3\)); (3) 125 smokers with asthma (54% male; \(M_{age} = 37.7\) years, \(SD = 12.1\)); and (4) 126 smokers without asthma (70.4% male; \(M_{age} = 36.5\) years, \(SD = 13.1\)). After controlling for gender, race, and age, having an asthma diagnosis significantly predicted higher levels of AS. AS was positively associated with the log odds of being a smoker, and the direct effect of asthma diagnosis was negatively associated with smoking status. Bias-corrected bootstrapping (\(k = 10,000\) samples) was used to generate a 95% confidence interval to test the significance of the
indirect effect. Results indicated a significant indirect effect of asthma diagnosis on smoking status through AS (95% CI = .09 to .46), suggesting that AS mediates the association between asthma diagnosis and smoking status. The primary implication of these findings is that AS may serve as an important target for prevention and intervention efforts in this population.

**Keywords:** asthma, cigarette smoking, anxiety sensitivity, statistical mediation
# Table of Contents

Abstract ..................................................................................................................................................... ii

Table of Contents ..................................................................................................................................... v

List of Tables ........................................................................................................................................ vi

List of Figures ........................................................................................................................................ vii

Introduction .............................................................................................................................................. 1
  Asthma ........................................................................................................................................................ 1
  Cigarette Smoking .................................................................................................................................... 3
  Asthma and Smoking ............................................................................................................................... 4
  Asthma, Smoking Behavior, and Smoking-Related Cognitive Processes .............................................. 5
  Smoking and Anxiety ............................................................................................................................... 7
  Smoking and Anxiety Sensitivity ........................................................................................................... 8
  Asthma and Anxiety ............................................................................................................................... 10
  Asthma and Anxiety Sensitivity ............................................................................................................. 11
  Present Study ......................................................................................................................................... 12

Method ..................................................................................................................................................... 13
  Participants and Procedures .................................................................................................................. 13
  Measures ................................................................................................................................................. 16
  Analytic Approach ................................................................................................................................. 18

Results ..................................................................................................................................................... 22
  Identification of Covariates ................................................................................................................... 22
  Bivariate Correlations ........................................................................................................................... 24
  Mediation Analyses ............................................................................................................................... 24

Discussion .............................................................................................................................................. 26
  Limitations and Future Directions ........................................................................................................ 28
  Clinical Implications ............................................................................................................................. 30
  Conclusions .......................................................................................................................................... 30

References ............................................................................................................................................... 32
List of Tables

Table 1. Asthma Severity Classification .................................................................2
Table 2. Demographic Characteristics of Study Sample ........................................23
Table 3. Intercorrelations among Covariates, Proposed Mediator, Independent, and Dependent Variables .................................................................24
Table 4. Logistic Regression and Ordinary Least Squares Regression Model Coefficients .................................................................25
List of Figures

Figure 1. Diagram of simple mediation model……………………………………………….20
Figure 2. Path coefficients for simple mediation analysis on smoking status………………26
Introduction

Asthma

Asthma is a reversible obstructive airway disease consisting of chronic airway inflammation and episodes of exacerbation in response to certain stimuli (American Lung Association [ALA], 2010a). These airway exacerbations are commonly referred to as asthma attacks and consist of increased swelling of the airway lining, tightening of the muscles around the airways, and increased mucus production, resulting in symptoms of wheezing, coughing, chest tightness, and shortness of breath (ALA, 2010b). Common triggers for asthma attacks include changes in weather (e.g., increased humidity, cold temperatures), mold, pets, exercise, environmental tobacco smoke, and allergens and irritants (e.g., dust mites, cockroaches; ALA, 2008). Asthma affects approximately 20 million individuals in the United States (U.S.; ALA, 2010a), and over half of the individuals diagnosed with asthma experience at least one asthma attack each year (Centers for Disease Control and Prevention [CDC], 2007). Among U.S. adults, the lifetime prevalence rate of asthma is 13.3%, with slightly higher rates among females compared to males (13.9% and 12.5%, respectively; ALA, 2010c). Females also have consistently higher rates of asthma attacks each year compared to males (ALA, 2010c).

Asthma is generally diagnosed by physician determination of the presence of recurrent episodes of airflow obstruction (usually at least partially reversible) or airway hyperresponsiveness (National Heart, Lung, and Blood Institute [NHLBI], 2007). The classification of asthma severity is typically completed through physical exam to assess an individual’s lung functioning, frequency of symptoms, and frequency of rescue inhaler use. Lung functioning is assessed via spirometry, which measures peak expiratory flow rates and forced expiratory volume in one second (FEV\textsubscript{1}; Sims, 2006). In both cases, lower levels correspond
with more severe impairment. Based on the findings from this assessment, patients can be placed into one of the following categories of severity: intermittent, mild persistent, moderate persistent, or severe persistent (NHLBI, 2007). Please see Table 1 for a more detailed description of asthma severity classification for individuals over 12 years of age.

Table 1
*Asthma Severity Classification* (NHLBI, 2007, p. 43)

<table>
<thead>
<tr>
<th>Symptom Frequency</th>
<th>Intermittent</th>
<th>Mild Persistent</th>
<th>Moderate Persistent</th>
<th>Severe Persistent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>≤ 2 days/week</td>
<td>&gt; 2 days/week, &lt; daily</td>
<td>Daily</td>
<td>Throughout the day</td>
</tr>
<tr>
<td>Interference</td>
<td>none</td>
<td>minor limitations</td>
<td>some limitations</td>
<td>extreme limitations</td>
</tr>
<tr>
<td>with normal activity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nighttime</td>
<td>≤ 2x/month</td>
<td>3-4x/month</td>
<td>&gt; 1x/week, &lt; nightly</td>
<td>Nightly</td>
</tr>
<tr>
<td>awakenings</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Short-acting</td>
<td>≤ 2 days/week</td>
<td>&gt; 2 days/week, &lt; daily, not &gt;1x/day</td>
<td>Daily</td>
<td>Several times/day</td>
</tr>
<tr>
<td>beta&lt;sub&gt;2&lt;/sub&gt;-agonist</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>use</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- **Lung functioning**
  - Normal FEV<sub>1</sub> between attacks
  - FEV<sub>1</sub> > 80% predicted
  - FEV<sub>1</sub>/FVC normal
  - FEV<sub>1</sub> > 80%, but < 80% predicted
  - FEV<sub>1</sub>/FVC reduced 5%
  - FEV<sub>1</sub> < 60% predicted
  - FEV<sub>1</sub>/FVC reduced > 5%

*Note*: FEV<sub>1</sub> = forced expiratory volume in one second; FVC = forced vital capacity; Normal FEV<sub>1</sub>/FVC for ages 8-19 yrs = 85%, 20-39 yrs = 80%, 40-59 yrs = 75%, 60-80 yrs = 70%

Due to information gaps regarding the etiology of asthma, clinical focus is currently aimed at disease management rather than prevention. Treatment consists primarily of decreasing exposure to asthma triggers and medication to control inflammation (CDC, 2007). There are two classes of medications typically used to control asthma: controller medications (anti-
inflammatory medications, typically inhaled corticosteroids) and reliever medications (bronchodilators, such as short-acting beta-agonists). In terms of reducing exposure to environmental triggers, environmental tobacco smoke has been the primary target of research and prevention/intervention efforts. Substantially less research, however, has examined active smoking among individuals with asthma. Surprisingly, despite the negative impact of smoking on already compromised lung functioning, a substantial proportion of individuals with asthma are active cigarette smokers.

**Cigarette Smoking**

Despite increased efforts to prevent the onset or maintenance of cigarette smoking, smoking remains a significant public health concern (Pleis, Lucas, & Ward, 2009). Currently, approximately 19.3% of U.S. adults are regular cigarette smokers (CDC, 2011), with higher rates among men compared to women (23.1% and 18.3%, respectively; CDC, 2009). Rates of smoking had been steadily decreasing until 2004; however, this rate of decline seems to have stalled in recent years (CDC, 2010). In the U.S., each day, approximately 1,000 children under the age of 18 and 1,800 adults become regular daily smokers (CDC, 2010). Individuals typically become daily smokers during adolescence, particularly between the ages of 15 and 20; daily smoking rarely begins after 25 years of age (Breslau, Johnson, Hiripi, & Kessler, 2001). On average, regular smokers smoke 13 cigarettes per day, with men smoking slightly more cigarettes per day than women (Substance Abuse and Mental Health Services Administration [SAMHSA], 2003).

Smoking is the leading preventable cause of death and disability in the U.S., accounting for 1 in 5 deaths each year (CDC, 2011; U.S. Department of Health and Human Services [USDHHS], 2004). Smoking harms nearly every organ in the body, resulting in approximately
8.6 million individuals in the U.S. who suffer from one or more serious illnesses associated with cigarette smoking (e.g., chronic obstructive lung disease [COPD], lung cancer; ALA, 2010d). Other conditions associated with smoking include various types of non-lung-related cancers (e.g., kidney, bladder, cervical), coronary heart disease, stroke, pneumonia, preterm deliveries, and asthma (ALA, 2010d). Compared to nonsmokers, smokers are more likely to be absent from work, have longer-lasting illnesses, incur more medical costs, have more frequent physician office visits, and be admitted to the hospital more often and for longer periods of time (USDHHS, 2004). Recent reports estimate that the total costs of cigarette smoking in the U.S. are approximately $193 billion per year (CDC, 2011).

**Asthma and Smoking**

Despite the known compromising effects of smoking on lung function and health, smoking is more common among individuals with asthma compared to those without. Indeed, the odds of being an active smoker are higher than of being a non-smoker among individuals with both a current and lifetime asthma diagnosis (OR = 1.36 and 1.31, respectively; Gwynn, 2004). Moreover, young adults with current asthma are more likely to be regular smokers (> 4 days per week) and heavier smokers (> 10 cigarettes per day) than those without asthma (Avila, Soto-Martinez, Soto-Quiros, & Celedon, 2005). With regard to temporal patterning, the majority of studies have found that smoking predicts later asthma onset (Genuneit et al., 2006; Plashke et al., 2000). However, two studies have also found the reverse to be true (Oechsli, Selzer, & van den Berg, 1987; Van de Ven, Engels, Kerstjens, & Van den Eijnden, 2007). Thus, there appear to be significant bidirectional associations, or multiple pathways, leading to asthma-smoking co-occurrence.
Not surprisingly, smoking negatively affects asthma symptom severity and symptom management. In terms of asthma control, research has consistently found that smoking results in poorer asthma control and more frequent and severe asthma exacerbations (McCoy et al., 2006; McLeish & Zvolensky, 2010; Schatz, Zeiger, Vollmer, Mosen, & Cook, 2006). Among individuals with not-well-controlled asthma, smoking status appears to have the greatest influence on whether or not patients achieve asthma control (Pedersen, Bateman, Bousquet, Busse, Yoxall, & Clark, 2007). Moreover, 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 (Althuis, Sexton, & Prybylski, 1999; Beeh, Micke, Ksoll, & Buhl, 2001; Boulet, FitzGerald, McIvor, Zimmerman, & Chapman, 2008). Further, smokers diagnosed with asthma are 2.4 times more likely to experience daily asthma attacks and 1.86 times more likely to have an asthma-related hospitalization compared to non-smokers with asthma (Eisner & Iribarren, 2007; Siroux, Pin, Oryszczyn, Le Moual, & Kauffman, 2000). In addition to increasing asthma severity and decreasing asthma control, smoking also decreases the effectiveness of asthma treatment. Indeed, corticosteroid use results in improved lung functioning and asthma control and decreased asthma symptoms in non-smokers, but not in current smokers (Chaudhuri, Livingston, McMahon, Thomson, Borland, & Thomson, 2003).

Asthma, Smoking Behavior, and Smoking-Related Cognitive Processes

In order to better understand the smoking-asthma association, researchers have begun investigating patterns of smoking behavior as well as smoking-related cognitive processes among smokers with and without asthma. In terms of smoking behavior, there appear to be no differences between smokers with and without asthma in terms of daily smoking rate, likelihood
of making a quit attempt, and stage of change in terms of readiness to quit smoking (Wakefield, Roberts, Ruffin, Wilson, & Campbell, 1995). Among adolescent smokers, Zimmerman and colleagues (2004) found no differences between adolescents with and without asthma in terms of puff volume, puff duration, interpuff interval, puff velocity, age at first cigarette, age at daily smoking, time to treatment, or number of quit attempts. More recently, however, Avallone and colleagues (in press) found that, compared to smokers without asthma, adult smokers with asthma reported a younger age of smoking onset and a greater number of quit attempts. Similar to previous research, no differences were found in terms of smoking rate and level of nicotine dependence in this study.

With regard to smoking motives, adolescent smokers with asthma, compared to adolescent smokers without asthma, are more likely to report smoking as a means of weight control and less likely to smoke for social reasons (Precht, Keiding, Nielsen, & Madsen, 2006). In terms of other smoking-related cognitive processes, Van de Ven, Van den Eijnden, and Engels (2006) found that the only difference between adolescent smokers with and without asthma was that smokers with asthma reported a higher perceived risk of having severe lung problems after smoking for several years. Similarly, adult smokers with asthma are twice as likely as smokers without asthma to report that they believe their health is worse due to smoking and that their health in the future will be worse due to smoking (Wakefield et al., 1995). Despite these beliefs, recent research has found that adult smokers with asthma do not appear to be more likely than adult smokers without asthma to report motives for quitting related to health concerns, but are more likely to report motives for quitting related to self-control (e.g., wanting to show oneself or others that one can quit successfully; Avallone et al., in press).
Overall, it appears that there are few differences between smokers with and without asthma in terms of smoking behavior and smoking-related cognitive processes. Therefore, it is likely that there are other key factors involved in the association between smoking and asthma. One such factor may be anxiety psychopathology, which has been linked to both smoking and asthma (Feldner, Babson, & Zvolensky, 2007; Goodwin, Jacobi, & Thefeld, 2003; Hasler, et al., 2005; McCabe et al., 2004).

Smoking and Anxiety

Approximately half of all daily smokers have a lifetime history of a psychiatric disorder, and these individuals consume a disproportionate amount of cigarettes (Lasser et al., 2000). A number of efforts have been made to understand the associations between smoking and specific types of mental illness. The vast majority of this research has focused on schizophrenia and depressive-related disorders (e.g., Ginsberg, Hall, Reus, & Muñoz, 1995; Kinnunen, Doherty, Militello, & Garvey, 1996), whereas comparatively less attention has been devoted to the link between smoking and anxiety disorders. This lack of attention is unfortunate, as anxiety disorders are among the most common forms of psychopathology (Kessler, Chiu, Demler, & Walters, 2005) and co-occur with smoking at rates that exceed those found in the general non-psychiatric population and in many other psychiatric conditions (Feldner et al., 2007; McCabe et al., 2004; Morissette, Tull, Gulliver, Kamholz, & Zimering, 2007). Some of the strongest associations between smoking and anxiety have been found for panic psychopathology (i.e., panic attacks, panic disorder, and agoraphobia; Baker-Morissette, Gulliver, Wiegel, & Barlow, 2004; Lasser et al., 2000; McCabe et al., 2004; Zvolensky, Feldner, Leen-Feldner, & McLeish, 2005).
Extant research indicates that daily cigarette smoking, especially at higher rates, is concurrently and prospectively related to an increased risk of panic-related symptoms, panic attacks, panic disorder, and agoraphobia (Breslau & Klein, 1999; Breslau, Novak, & Kessler, 2004; Isensee, Wittchen, Stein, Höfler, & Lieb, 2003; Johnson et al., 2000; Zvolensky, Leen-Feldner, et al., 2004). Furthermore, among individuals with an anxiety disorder diagnosis, heavier amounts of smoking are associated with more severe anxiety symptoms (McCabe et al., 2004; Zvolensky, Leen-Feldner, et al., 2004). Lastly, anxiety-related risk factors (e.g., history of panic psychopathology) are associated with increased risk of smoking problems, including greater likelihood for nicotine dependence (Farrell et al., 2001), more severe nicotine withdrawal symptoms (Zvolensky, Lejuez, Kahler, & Brown, 2004), and the tendency to smoke primarily to reduce negative affective states (Zvolensky, Schmidt, et al., 2005).

**Smoking and Anxiety Sensitivity**

One risk factor for anxiety psychopathology that has received increasing empirical attention in the smoking literature is anxiety sensitivity (AS), defined as the fear of arousal-related physical and psychological sensations (McNally, 2002; Reiss & McNally, 1985). AS reflects a relatively stable, albeit malleable, cognitive predisposition that is theoretically and empirically distinct from the tendency to experience negative emotional states (McNally, 2002). AS encompasses three lower-order dimensions: physical, cognitive, and social concerns that load onto a single higher-order factor (Deacon & Valentiner, 2001; Taylor, 1999; Zinbarg, Barlow, & Brown, 1997). When anxious, individuals high in AS become acutely fearful due to beliefs that these anxiety-related interoceptive sensations have harmful physical, social, or psychological consequences (Taylor et al., 2007). In line with this theory, AS 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, posttraumatic stress disorder; Feldner, Zvolensky, Schmidt, & Smith, 2008; Hayward, Killen, Kraemer, & Taylor, 2000; Li & Zimbarg, 2007; Maller & Reiss, 1992; Marshall, Miles, & Stewart, 2010; Schmidt, Lerew, & Jackson, 1997, 1999; Schmidt, Zvolensky, & Maner, 2006).

Extant research on AS and smoking has demonstrated that AS plays an important role in both smoking behavior and cognitive-based smoking processes. For example, AS is primarily associated with smoking to reduce negative affect (Battista et al., 2008; Gonzalez, Zvolensky, Vujanovic, Leyro, & Marshall, 2008; Gregor, Zvolensky, Bernstein, Marshall, & Yartz, 2007; Leyro, Zvolensky, Vujanovic, & Bernstein, 2008; Novak, Burgess, Clark, Zvolensky, & Brown, 2003; Zvolensky, Bonn-Miller, et al., 2006). AS is also associated with expectancies that smoking will result in negative affect reduction (Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001; Gregor, Zvolensky, McLeish, Bernstein, & Morissette, 2008). In terms of smoking cessation, AS is associated with increased motivation to quit (Zvolensky, Baker, et al., 2004; Zvolensky, Vujanovic et al., 2007) as well as a greater number of quit attempts (Zvolensky, Johnson, Leyro, Hogan, & Tursi, 2009). However, higher levels of AS are also associated with decreased self-efficacy in refraining from smoking when emotionally distressed (Zvolensky, Bonn-Miller, et al., 2006) and greater perceived barriers to cessation (Gonzalez et al., 2008; Gregor et al., 2008). Further, AS has been found to predict greater intensity of nicotine withdrawal symptoms during a quit attempt (Zvolensky, Baker, et al., 2004; Zvolensky, Feldner et al., 2005) and earlier lapses and relapses to smoking during a smoking cessation attempt, particularly in the first two weeks of the attempt (Brown, Kahler, Zvolensky, Lejuez, & Ramsey, 2001; Zvolensky, Bernstein, Cardenas, Colotla, Marshall, & Feldner, 2007; Zvolensky, Bonn-Miller, Bernstein, & Marshall, 2006; Zvolensky, Stewart, Vujanovic, Gavric, & Steeves, 2009).
Asthma and Anxiety

Similar to the patterns seen among smokers, psychiatric diagnoses, particularly anxiety disorders, are also more common among individuals with asthma compared to those without. On average, the prevalence rate of any anxiety disorder among adults with asthma is 34% (Weiser, 2007), with some studies finding rates as high as 70% (Fernandez et al., 2010). Although extant research has clearly documented an association between asthma and anxiety disorders, there is evidence for a relatively specific association between asthma and panic psychopathology. Indeed, rates of panic attacks and panic disorder are consistently higher among individuals with asthma compared to the general population (Goodwin, Jacobi, & Thefeld, 2003; Goodwin, Pine, & Hoven, 2003). Moreover, there appears to be a dose-response relationship, such that with every additional panic symptom the likelihood of asthma increases (Goodwin, Pine, & Hoven, 2003; Hasler et al., 2005). A recent 20-year longitudinal study found a significant bidirectional relationship between the two; asthma was a significant predictor of future panic disorder (OR = 4.5), and panic disorder predicted later asthma activity (OR = 6.3; Hasler et al., 2005).

Not surprisingly, having a comorbid anxiety disorder diagnosis adversely affects asthma. Research has consistently found that individuals with asthma and a comorbid anxiety disorder, despite showing no differences in objective indices of lung function, report poorer asthma control and quality of life (Feldman, Siddique, Morales, Kaminski, Lu, & Lehrer, 2005; Fernandes et al., 2010; Lavoie et al., 2005; Strine et al., 2008) as well as increased impairment in daily functioning and greater frequency of asthma symptoms (McCauley, Katon, Russo, Richardson, & Lozano, 2007; Strine, Mokdad, Balluz, Berry, & Gonzalez, 2008; ten Brinke, Ouwerkerk, Zwinderman, Spinhoven, & Bel, 2001). Furthermore, comorbid anxiety has been
linked to greater asthma severity (Fernandes et al., 2010; Rietveld, Van Beest, & Prins, 2005) and health care utilization (Fernandes et al., 2010). Specifically, comorbid anxiety and asthma are associated with more frequent visits to the emergency room, primary care physicians, outpatient providers, and increased prescription fills (Fernandes et al., 2010; Richardson, Russo, Lozano, McCauley, & Katon, 2008; ten Brinke et al., 2001).

**Asthma and Anxiety Sensitivity**

In order to better understand the mechanisms underlying the anxiety-asthma association, recent research has focused on the role of anxiety-related cognitive risk factors, most notably AS. This research, although limited, is promising. For example, independent of asthma severity, AS has been found to predict panic in response to asthma symptoms (Carr, Lehrer, & Hochron, 1995) as well as increased activity limitations due to asthma symptoms and poorer asthma-related physical and emotional health (McCauley et al., 2007). Further, individuals with atopic (i.e., allergic or extrinsic) asthma have higher levels of AS than those with non-atopic (i.e., intrinsic) asthma (Barone, Bacon, Campbell, Labrecque, Ditto, & Lavoie, 2008). In an undergraduate sample of individuals with self-reported, physician-diagnosed asthma, McLeish, Zvolensky, and Luberto (2011), found that the physical concerns facet of AS, but not the cognitive or social concerns facets, significantly predicted poorer levels of asthma control independent of gender, daily cigarette smoking, and negative affectivity. Similarly, Avallone, McLeish, Luberto, and Bernstein (2012) found that the physical concerns facet of AS significantly predicted 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.
Taken together, empirical evidence suggests that: (1) smoking is more prevalent among individuals with asthma compared to those without; (2) few differences appear to exist between smokers with and without asthma in terms of smoking behavior and smoking-related cognitive processes; and (3) anxiety, particularly AS, is associated with both smoking and asthma. However, the current body of literature has yet to examine the interplay between anxiety, smoking, and asthma. That is, no research to date has examined the role of AS as a mediator in the smoking-asthma association. A mediator is a third variable that accounts for some of the effect of the independent variable on the dependent variable; that is, it helps to explain how or why the effect occurs (Baron & Kenny, 1986). As research has consistently found that AS is associated with both smoking and asthma, AS may help to explain the association between smoking and asthma. That is, smoking may be associated with asthma because of its association with AS and the association between AS and asthma. Examining the mediating effect of AS on the relationship between smoking and asthma would not only help to better understand this association, but would provide a specific target for prevention and intervention efforts in this population.

**Present Study**

The overarching purpose of the present investigation was to evaluate the mediating role of AS in the association between asthma diagnosis and smoking status. It was hypothesized that after controlling for relevant covariates (e.g., gender, race, age), AS would mediate the relationship between asthma diagnosis and smoking status.
Method

Participants and Procedures

The study sample consisted of four distinct groups based on asthma status and smoking status created with data from three existing datasets ($N = 485$): (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.

Non-Smokers without Asthma. Participants in this group were 113 non-smoking undergraduate students without asthma (74.1% female, $M_{age} = 20.32$ years, $SD = 3.72$). 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 nonsmokers, and did not have a history of asthma were invited to participate in the study. Students interested in participating in the study were first screened for eligibility via email. Eligible participants were then given a link and password to complete the measures online. Participants were then able to complete the questionnaires at their convenience and submit them anonymously via Survey Monkey. Information regarding participants’ IP addresses was not collected to ensure anonymity. After completing the study, participants contacted study personnel to receive course credit for their participation. The Institutional Review Board approved all study procedures and materials prior to data collection. Consistent with previous studies of AS (e.g., Osman, Gutierrez, Smith, Fang, Lozano, & Devine, 2010; Taylor et al., 2007; Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012), an undergraduate sample was used as the control group in the present study. Further, scores on the Anxiety Sensitivity Index-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 ($M = 12.8, SD = 10.6$; Taylor et al., 2007).

**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 January 1, 2009 and January 1, 2010. To be eligible for the study 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. Specifically, individuals received: (1) a letter that invited them to participate in the study and explained the study details; (2) a questionnaire packet; (3) two copies of the informed consent document (one to keep and one to return); and (4) a form on which to provide their mailing address in order to receive compensation. Interested participants provided written informed consent, completed the packet of questionnaires, provided their contact information, 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 $25 gift card as compensation for their time and effort. A total of 127 questionnaire packets were returned (34.5% response rate). Six individuals reported current regular smoking and were excluded from the study, resulting in a total of 121 participants. The racial composition of this group was 80.8% Caucasian, 9.2% African American, 1.7% Asian, and 8.3% other. In terms of asthma control, participants reported a mean Asthma Control Test (Nathan et al., 2004) score of 19.88 ($SD = 4.30$), indicating well-controlled asthma.

**Smokers with Asthma.** Participants in this group were 125 regular daily smokers with self-reported, physician-diagnosed asthma (54% male; $M_{\text{age}} = 37.66$ years, $SD = 12.12$) between
the ages of 18 and 65 who were recruited from the greater Cincinnati, OH community. The racial composition of the group was 41.5% Caucasian, 54.5% African American, 1.6% Asian, and 2.4% other. Smokers with asthma smoked on average 20.78 cigarettes per day \((SD = 12.84)\) and reported a mean Asthma Control Test (Nathan et al., 2004) score of 15.48 \((SD = 4.21)\), indicating difficulties with asthma control. Participants were recruited via newspaper ads (online and print) and flyers posted throughout the community. Eligibility criteria for the study were: (1) self-report of a physician diagnosis of asthma; (2) current prescription for asthma medication; (3) presence of asthma symptoms within the past year; (4) daily smoking rate greater than 10 cigarettes per day; (5) expired carbon monoxide levels greater than 10 parts per million (ppm; please see Measures section for details); and (6) regular, daily smoking for at least one year. After providing informed, written consent, participants’ smoking status was biochemically verified via expired CO analysis. Then, participants were administered a demographic interview to obtain information on key demographic variables, asthma history, and general medical history. They then completed a battery of self-report measures. Participants received $30 as compensation for their time and effort.

**Smokers without Asthma.** This group consisted of 126 regular, daily smokers with no history of asthma (70.4% male; \(M_{age} = 36.51\) years, \(SD = 13.05\)). The racial composition of the group was 69.8% Caucasian, 26.2% African American, and 4% Other. Smokers without asthma smoked, on average, 18.52 cigarettes per day \((SD = 8.69)\). 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; please see Measures section for details); and (3) regular, daily smoking for at least one year. Exclusion criterion was any endorsement of
current or past diagnosis of asthma. After providing informed, written consent, participants’ smoking status was biochemically verified via expired CO analysis. Then, participants were administered a demographic interview to obtain information on key demographic variables and medical history. They then completed a battery of self-report measures. Participants received $30 as compensation for their time and effort.

**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 (Corcores, 1993). Obtained values above this cutoff were considered indicative of smoking.

**Asthma History Questionnaire.** Participants reporting a diagnosis of asthma completed the Asthma History Questionnaire, which asked about their history of asthma as well as current medication use and previous hospitalizations. Those who endorsed an asthma diagnosis and who reported a current prescription for an asthma-related medication and asthma-related symptoms within the past 12 months were considered to have 'current asthma.' This strategy has been successfully employed in previous research in our laboratory (e.g., McLeish, Zvolensky, & Luberto, 2011). Information was also gathered regarding age at diagnosis, number of hospitalizations, and recent symptoms and medication use.

**Asthma Control Test (ACT).** The ACT (Nathan et al., 2004) 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 5-25 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 (Nathan et al., 2004). 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 (SHQ).** Smoking history and pattern was assessed with the SHQ (Brown, Lejuez, Kahler, & Strong, 2002), 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 (Zvolensky, Schmidt, et al., 2005). 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 (ASI-3).** The ASI-3 (Taylor et al., 2007) was administered to assess AS. The ASI-3 is an 18-item measure that asks respondents to rate on a 5-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 AS factor and three specific lower-order factors (Taylor et al., 2007). 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 the
strongest psychometric properties of any current measure of AS (Taylor et al., 2007). Internal consistency for the ASI-3 in the current study was good (range: .90 to .95).

**Analytic Approach**

**Identification of covariates.** Independent samples t-tests (or Chi-square tests as appropriate) were used to determine whether there were any significant group differences on demographic variables (i.e., gender, race, age) that needed to be accounted for in subsequent analyses.

**Correlation analyses.** Zero-order correlations were computed to assess the associations between the independent variable (asthma diagnosis), covariates, the proposed mediator (AS), and the primary dependent variable (smoking status).

**Mediation analysis.** In order to test the hypothesis that AS mediates the relationship between asthma diagnosis and smoking status, a simple mediation analysis was conducted using PROCESS in SPSS (Hayes, 2012). Asthma diagnosis was identified as the independent variable and smoking status was identified as the dependent variable. Although it would also be plausible for smoking status to be the independent variable and asthma diagnosis the dependent variable, it is more likely that physiological changes related to smoking, rather than AS, would lead to asthma onset. Thus, it is not surprising that smoking would lead to asthma given the negative impact of smoking on the lungs. What is less clear, however, is what would explain, or partially explain, why an individual with asthma would initiate smoking, which was the focus of the current study.

Recently, there has been debate regarding the appropriate methodological approach for mediation analyses. In the traditional causal steps mediation approach proposed by Baron & Kenny (1986), the following conditions must be met in order for mediation to exist: (1) a
significant relationship between the independent variable and the dependent variable; (2) a significant association between the independent variable and the proposed mediator; (3) a significant relationship between the proposed mediator and the dependent variable; and (4) the association between the independent and dependent variable must be reduced when the proposed mediator is controlled for or added to the model. If these criteria are met, the Sobel test is typically used to test whether the indirect effect is significantly different from zero (Sobel, 1982). This approach has been criticized, because it only infers that the indirect effect exists, rather than directly quantifying the effect (Hayes, 2009; Preacher & Hayes, 2004; Rucker, Preacher, Tormala, & Petty, 2011). Further limitations to the causal steps approach include the possibility of missing mediation effects due to the requirement that there first be a significant relationship between the independent and dependent variables and low statistical power (particularly in small samples; Hayes, 2009; Preacher & Hayes, 2004). The Sobel test is also limited by the assumption of normality of the indirect effect (i.e., if the distributions of the direct effects are normally distributed, their product will be normally distributed); typically the distribution of the indirect effect is not normally distributed (Preacher & Hayes, 2004).

The analyses for this study followed the recent recommendations for mediation analyses, which propose directly testing the indirect effect (Hayes, 2009; MacKinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2004). In line with these recommendations, ordinary least squares (OLS) or logistic regressions, as appropriate, were conducted in order to estimate the total, direct, and indirect effects of asthma diagnosis on smoking status through AS using PROCESS in SPSS (Hayes, 2012). Figure 1 illustrates the framework of the simple mediation model that was used for this study.
Figure 1. Diagram of simple mediation model with asthma diagnosis as the independent variable, anxiety sensitivity (AS) as the proposed mediator, and smoking status as the dependent variable.

The top part of the figure represents the total effect of asthma diagnosis on smoking status, while the bottom part of the figure depicts the model with AS introduced as the mediator. In this diagram, $c$ represents the total effect of asthma on smoking without taking the mediator into consideration, while $c'$ represents the direct effect of asthma diagnosis on smoking status in the mediation model. The effect of asthma diagnosis on AS is represented by $a$, and $b$ represents the effect of AS on smoking status after controlling for asthma diagnosis. The indirect effect is the product of $a$ and $b$ and represents the effect of asthma diagnosis on smoking status through AS.

In order to estimate the total effect, $c$, a logistic regression was conducted with asthma diagnosis (0 = no asthma diagnosis, 1 = asthma diagnosis) entered as the predictor variable, smoking status (0 = non-smoker, 1 = smoker) entered as the outcome variable, and demographic variables for which the groups differed entered as covariates. The direct effect, $c'$, was estimated
via a logistic regression with asthma diagnosis entered as the predictor variable (0 = no asthma diagnosis, 1 = asthma diagnosis) and smoking status entered as the outcome variable (0 = non-smoker, 1 = smoker), controlling for AS and the covariates. The effect of asthma diagnosis on AS, $a$, was estimated via an OLS regression with the covariates entered at step one and asthma diagnosis entered at step two. In order to estimate the effect of AS on smoking status, $b$, a logistic regression was conducted with AS entered as the predictor variable and smoking status (0 = non-smoker, 1 = smoker) entered as the outcome variable, controlling for asthma diagnosis and the covariates. The indirect effect ($ab$) was calculated by multiplying the coefficient $a$ by the coefficient $b$. In order to determine whether the indirect effect was significant (i.e., whether AS mediated the relationship between asthma diagnosis and smoking status), bootstrapping, an alternative to the Sobel test that also directly tests the indirect effect yet does not assume normality of the sampling distribution, was conducted. Bootstrapping involves generating a number of samples ($k$) from the original sample in order to estimate an effect from each of the $k$ samples. These estimated effects are then used to generate confidence intervals, which are then used to test whether the indirect effect is significantly different from zero. Given the nature of bootstrapping, there is the potential for skew or bias in the distribution of the estimated effects and resulting confidence intervals compared to that of the original sample. One way to reduce this potential bias is to conduct bias-corrected bootstrapping, a type of bootstrapping that accounts for the bias in the bootstrapped samples (Steck & Jaakkola, 2003). Extant research indicates that, in addition to not assuming normality, bootstrapping is a more powerful and valid way of testing mediating effects compared to other popular methods (e.g., Sobel test; MacKinnon et al., 2004). A bias-corrected bootstrap-confidence interval (CI) for the product of these paths that does not include zero provides evidence of a significant indirect effect of asthma.
diagnosis on smoking status through AS (Preacher & Hayes, 2008; Hayes, 2009). For the present study, bias-corrected bootstrapping \((k = 10,000)\) was conducted in order to generate a 95% confidence interval (CI) to test the significance of the indirect effect of AS in the relationship between asthma diagnosis and smoking status.

**Results**

**Identification of Covariates**

Descriptive data and sample characteristics are presented in Table 2. There were significant differences between groups with regard to gender \(\chi^2 (3) = 72.69, p < .001\) and race \(\chi^2 (9) = 104.30, p < .001\), 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 \(F (3,447) = 90.41, p < .001\). More specifically, nonsmokers with asthma were older and nonsmokers without asthma were younger than all other groups. Thus, gender, race, and age were added as covariates in all subsequent analyses.
Table 2
Demographic Characteristics of Study Sample

<table>
<thead>
<tr>
<th></th>
<th>Total Sample</th>
<th>Nonsmokers without Asthma</th>
<th>Nonsmokers with Asthma</th>
<th>Smokers with Asthma</th>
<th>Smokers without Asthma</th>
<th>Test Statistic (df)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>34.93 (13.91)</td>
<td>20.32 (3.72)</td>
<td>43.64 (12.33)</td>
<td>37.66 (12.12)</td>
<td>36.51 (13.05)</td>
<td>$F (3, 477) = 90.41^*$</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>214 (45%)</td>
<td>29 (26%)</td>
<td>30 (25%)</td>
<td>67 (54%)</td>
<td>88 (70%)</td>
<td>$\chi^2 (3) = 72.69^*$</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>$\chi^2 (9) = 104.30^*$</td>
</tr>
<tr>
<td>Caucasian</td>
<td>332 (69%)</td>
<td>96 (85%)</td>
<td>97 (81%)</td>
<td>51 (42%)</td>
<td>88 (70%)</td>
<td></td>
</tr>
<tr>
<td>Af. Am.</td>
<td>121 (25%)</td>
<td>10 (9%)</td>
<td>11 (9%)</td>
<td>67 (55%)</td>
<td>33 (26%)</td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>10 (2%)</td>
<td>6 (5%)</td>
<td>2 (2%)</td>
<td>2 (2%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>19 (4%)</td>
<td>1 (1%)</td>
<td>10 (8%)</td>
<td>3 (2%)</td>
<td>5 (4%)</td>
<td></td>
</tr>
</tbody>
</table>

Note: Values in the table reflect $M (SD)$ or no. (%), and p-values are based on results from one-way ANOVA or Pearson chi-square tests. * indicates p-value significant at $p < .001$. Af. Am. = African American.
**Bivariate Correlations**

The associations among covariates, the proposed mediator, the independent variable, and the dependent variable are presented in Table 3.

### Table 3

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>-</td>
<td>-.03</td>
<td>.02</td>
<td>.10*</td>
<td>-.37**</td>
</tr>
<tr>
<td>Age</td>
<td>-</td>
<td>-</td>
<td>-.02</td>
<td>.42**</td>
<td>.16**</td>
</tr>
<tr>
<td>AS</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>.17**</td>
<td>.26**</td>
</tr>
<tr>
<td>Asthma Diagnosis</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-.02</td>
</tr>
<tr>
<td>Smoking Status</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

*Note.* A single asterisk indicates correlation is significant at .05 level; a double asterisk indicates correlation is significant at .01 level; Gender: 1 = male, 2 = female; AS: Anxiety Sensitivity; Asthma Diagnosis: 0 = no asthma diagnosis, 1 = asthma diagnosis; Smoking Status: 0 = non-smoker, 1 = smoker.

Both asthma diagnosis and smoking status were each significantly correlated with gender, age, and AS. AS was not significantly associated with age or gender.

**Mediation Analyses**

Using PROCESS (Hayes, 2012) in SPSS, OLS or logistic regressions (as appropriate) were conducted in order to estimate the following models to obtain the total, direct, and indirect effects of asthma diagnosis on smoking status through AS:

1. \[
    \text{Log odds (smoking status)} = \text{constant} + c(\text{asthma diagnosis}) + \text{covariates} + \text{error}
    \]

2. \[
    \text{AS} = \text{constant} + a(\text{asthma diagnosis}) + \text{covariates} + \text{error}
    \]

3. \[
    \text{Log odds (smoking status)} = \text{constant} + c'(\text{asthma diagnosis}) + b(\text{AS}) + \text{covariates} + \text{error}
    \]

Covariates for these analyses included gender, race, and age and these were entered in the models simultaneously with predictor variables. The coefficients \(c\), \(c'\), and \(b\) in equations 1 and 3
were estimated in PROCESS using logistic regression due to the dichotomous outcome variable, while the coefficient, $a$, in equation 2 was estimated via OLS regression analyses.

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 4, Model 1, Figure 2 path $c$). Model 2 represents the effect of asthma diagnosis on AS after controlling for gender, race, and age; having an asthma diagnosis significantly predicted higher levels of AS (Table 4, Model 2, Figure 2 path $a$). Model 3 represents the direct effect of asthma diagnosis on smoking status after accounting for the influence of AS, gender, race, and age. AS was positively associated with the log odds of being a smoker (Table 4, Model 3, Figure 2 path $b$), and the direct effect of asthma diagnosis (Table 4, Model 3; Figure 2 path $c'$) was negatively associated with smoking status after accounting for the effects of AS, gender, race, and age.

### Table 4

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking Status</td>
<td>1.28</td>
<td>17.18**</td>
<td>.60</td>
</tr>
<tr>
<td>AS</td>
<td>.05**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>-1.65**</td>
<td>-.04</td>
<td>-1.80**</td>
</tr>
<tr>
<td>Race</td>
<td>.48**</td>
<td>2.31*</td>
<td>.38*</td>
</tr>
<tr>
<td>Age</td>
<td>.03**</td>
<td>-.12*</td>
<td>.04**</td>
</tr>
<tr>
<td>$R^2$</td>
<td>.24</td>
<td>.04</td>
<td>.33</td>
</tr>
</tbody>
</table>

*Note.* 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 $R^2$. * $p < .05$, ** $p < .01$. 
The indirect effect was calculated by multiplying the path $a$ coefficient obtained from the OLS regression analysis for asthma diagnosis predicting AS by the path $b$ coefficient obtained from the logistic regression analysis for AS predicting smoking status: $ab = 5.17 \times .05 = .24$. A bias-corrected bootstrapped confidence interval that does not include zero suggests a significant indirect effect of asthma diagnosis on smoking status through AS. Using 10,000 bias-corrected bootstrapped samples calculated with PROCESS, the resulting confidence interval (95% CI = .09 to .46) did not include zero, suggesting a significant indirect effect of asthma diagnosis on smoking status through AS. That is, AS does indeed serve 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 (Avallone et al., in press; Wakefield et al., 1995). 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. Given the individual associations between AS and both asthma and smoking, the present study aimed to evaluate the mediating role of AS 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 AS. In other words, the association between asthma diagnosis and smoking status appears to be driven, in part, by AS. Asthma diagnosis was a significant predictor of AS, 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 AS), 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 (McLeish & Zvolensky, 2010). One possible explanation for this unexpected finding may be due to the sampling procedures used in the present study, which utilized data from four separate samples recruited specifically based on the presence or absence of smoking and/or asthma. As these groups were relatively equal in size, they likely do not accurately reflect the prevalence rates of smoking and asthma found in the general population (19.3 and 13.3%, respectively; ALA, 2010c; CDC, 2011). Despite potential sampling issues, results of the current study indicate that the asthma-smoking relationship is a complex one, and examination of mediating variables is critical in better understanding the nature of these associations.

**Limitations and Future Directions**

While the present 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. Thus, these findings need to be replicated in a sample that includes smokers and non-smokers with and without asthma. Along these same lines, the nonsmokers 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 AS that has used undergraduates control groups (Osman et al., 2010; Taylor et al., 2007; Wheaton et al., 2012), and AS 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 in order to decrease demographic differences.

Second, for the smokers with asthma group, asthma diagnosis was assessed via self-report and was not verified by objective measures of lung functioning (e.g., spirometry). While efforts were made to verify asthma diagnosis with proxy indicators (e.g., experiencing symptoms in past 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, this study was cross-sectional in nature and, as a result, no conclusions regarding causation or temporal pattern can be made. It is, therefore, still unclear whether individuals with asthma develop higher levels of AS as a result of their asthma diagnosis or whether higher levels of AS actually put them at greater risk for developing asthma. Similarly, the present study did not assess asthma, smoking, and AS 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.

Lastly, while the present study found that AS 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, discomfort intolerance) in this association would improve our understanding of this association and would help further develop the model of these associations described above. Indeed, the findings of the present study lend support to the formation of a tentative theory for the asthma-smoking association, mainly that these individuals smoke to help regulate AS, and potentially more
generally, negative affect. Examining the role of other cognitive-vulnerability factors (e.g., distress tolerance, emotion regulation) would help to better understand whether this association is solely maintained by anxiety or if it is more generally maintained by negative affect and enable the formulation of a more concrete, complete theory.

Clinical Implications

Despite these limitations, there are a number of clinical implications for both prevention and intervention efforts that can be drawn from the findings of the present study. For example, physicians could assess AS during asthma-related office visits to identify asthma patients at risk for smoking onset due to high levels of AS. Similarly, assessing AS in individuals with asthma who already smoke could identify smokers in need of specialized cessation interventions. Indeed, research has shown that traditional cognitive-behavioral treatments for panic disorder, particularly interoceptive exposure techniques, are effective in reducing AS (Telch et al., 1993), and exposure-based techniques have also been successfully used to improve smoking cessation outcomes among high AS individuals (Zvolensky, Yartz, Gregor, Gonzalez, & Bernstein, 2008). 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

The present findings suggest AS mediates the association between asthma diagnosis and smoking status. The primary implication of these findings is that AS 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.
References


in asthma patients? *Respiratory Medicine, 99*, 1249-1257. doi: 10.1016/j.rmed.2005.03.003


Substance Abuse and Mental Health Services Administration, Office of Applied Studies


Psychopathology in patients with severe asthma is associated with increased health care utilization. *American Journal of Respiratory and Critical Care Medicine, 163*, 1093-1096.


Therapy, 33, 114-125. doi: 10.1080/16506070310016969


