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Does higher body mass index contribute to worse asthma control in an urban population?

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Abstract

Background—Epidemiologic findings support a positive association between asthma and obesity.

Objective—Determine whether obesity or increasing level of body mass index (BMI) are associated with worse asthma control in an ethnically diverse urban population.

Methods—Cross sectional assessment of asthma control was done in asthmatics recruited from primary care offices using four different validated asthma control questionnaires: the Asthma Control and Communication Instrument (ACCI), the Asthma Control Test (ACT), the Asthma Control Questionnaire (ACQ) and the Asthma Therapy Assessment Questionnaire (ATAQ). Multiple linear regression analysis was performed to evaluate the association between obesity and increasing BMI level and asthma control.

Results—Of 292 subjects mean age of 47 years, the majority were women (82%) and African American (67%). There was a high prevalence of obesity with 63%, with only 15% being normal weight. The mean score from all four questionnaires showed an average sub-optimal asthma control (mean score/maximum possible score): ACCI (8.3/19), ACT (15.4/ 25), ACQ (2.1/ 6), and ATAQ (1.3/ 4). Regression analysis showed no association between obesity or increasing BMI level and asthma control using all four questionnaires. This finding persisted even after adjusting for FEV₁, smoking status, race, gender, selected co-morbid illnesses, and long-term asthma controller use.

Conclusion—Using four validated asthma control questionnaires, we failed to find an association between obesity and asthma control in an urban population with asthma. Weight loss may not be an appropriate strategy to improve asthma control in this population.

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Clinical implications

Although weight loss has health benefits for those obese, it may not improve asthma control. Studies are needed to understand the effect of obesity on asthma control in different populations.

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Capsule Summary—Using four different validated asthma control measures, there was no association between obesity or increasing body mass index and asthma control in a largely obese urban outpatient minority population.

Keywords

asthma; asthma Control; obesity; overweight; body mass index; inner city; asthma communication control instrument; ACCI; African-American

Introduction

Over the past 20 years, the prevalence of asthma and obesity in the United States have increased significantly (1,2). According to the latest National Health and Nutrition Examination Survey (NHANES), more than 10 million (5.2%) US adults reports having a current asthma diagnosis (3) and approximately 30% of the US population meets the criteria for obesity, based on a body mass index (BMI) ≥ 30 kg/m² (4). The prevalence of asthma and obesity has been most notable among ethnic minorities, a group disproportionately affected by both disorders (5,6). In addition, African Americans have been shown to have higher asthma-related morbidity including hospital outpatient visits (14.2% vs. 5.5%) and emergency department (ED) visits (21.0% vs. 7.0) compared to Whites (7).

Epidemiologic studies looking at the relationship between obesity and asthma have found increasing BMI to be associated with increased asthma incidence (8). Whether this association is coincidental or due to a true physiologic link remains unclear. To date, studies looking at the association of obesity and cardinal features of asthma pathophysiology, such as hyper-responsiveness (9) and airflow limitation (10,11) have yielded conflicting results. Although weight loss has been shown to lead to improved symptoms in patients with asthma, studies have failed to show any effect of weight loss on pathophysiologic features of asthma (12). Obesity is associated with changes in lung volumes and gastroesophageal symptoms (i.e. gastroesophageal reflux disease), which may mimic asthma and contribute to inaccurate diagnosis of asthma in the morbidly obese (13). Furthermore, obesity and asthma may share common risk factors such as behavioral, environmental, and genetic factors that may account for their epidemiology link (14). Given the lack of consistency regarding the association between obesity and asthma pathophysiology, it is also debatable whether prior reports of a positive association between obesity and worse asthma severity (15-17) is in part due to publication bias, with failure of the literature to report negative studies.

Asthma control questionnaires have been used extensively in research to assess disease activity and/or evaluate treatment effectiveness (18,19). Moreover, clinical studies have shown inadequately controlled asthma, assessed using asthma control questionnaires, to be associated with worse asthma outcomes (19,20). According to the 2007 National Asthma Education and Prevention Program (NAEPP) guidelines, asthma control assessed using patient-reported validated asthma symptom questionnaires should be used rather than asthma severity in the long-term management of patients with asthma (21). Given that poor asthma control is associated with increased risk of hospitalization and acute health care use ((20,22), we sought to determine whether obesity contributes to worse asthma control in a urban community-based sample of people with asthma and a high prevalence of obesity. We hypothesized that subjects with higher BMI would have worse asthma control.

Methods

The data for this study were collected as part of a clinical trial conducted by the Howard-Hopkins Center to Reduce Asthma Disparities. The primary aim of that study was to test the

clinical utility of the Asthma Control and Communication Instrument (ACCI), an asthma health status questionnaire specifically designed to be culturally appropriate for ethnically diverse populations (23).

Study Population

Adults (≥ 17 yrs of age) from 5 community-based outpatient primary care practices in Baltimore, MD, and Washington, DC, were enrolled if they: 1) had doctor-diagnosed asthma, 2) were presenting for an already scheduled appointment and 3) had evidence of active asthma based on recent symptoms and/or reliever medication use. Participants were excluded if they: 1) were unable to speak and read English, 2) had previous participation in the study, or 3) had co-morbidities that would interfere with the study. Primary care clinics were selected based on demographic data indicating that they serve populations with a high proportion of African-Americans. Subjects provided informed consent and received a small financial incentive of \$30.00 for participation. Participants were not aware that the association between obesity and asthma control was being assessed. This study was approved by the Western Institutional Review Board (Spokane, Washington).

Following enrollment, participants completed a comprehensive survey regarding demographics, general health information, and asthma history (i.e. medications and health care use). Medications were classified as relievers (short acting beta agonists) or long-term controllers, with the latter being comprised of inhaled corticosteroids, long acting beta agonists, leukotriene modifiers, xanthines, IgE-blocker, as well as mast cells stabilizers.

Asthma Control

We assessed asthma control using four different survey tools: the Asthma Control and Communication Instrument (ACCI), the Asthma Control Test (ACT), the Asthma Control Questionnaire (ACQ) and the Asthma Therapy Assessment Questionnaire (ATAQ).

The ACCI is a 12-item self-administered survey that contains questions structured around five conceptual domains of asthma: Acute care, Bother from asthma, Control, Direction of disease activity and Adherence to long-term control medications. The control domain measures frequency of daytime symptoms, nocturnal symptoms, rescue medication use, asthma attacks and activity limitation due to asthma. The ACCI has been found to have face and content validity (23). Asthma control was defined two ways: 1) a sum score of the 5 control items which could range from 0 (better control) to 19 (worse control) (24).

The ACT is a validated patient-completed questionnaire consisting of five items aimed at assessing asthma symptoms (daytime and nocturnal), use of rescue medications, and the effect of asthma on daily functioning. Each item includes five response options. The score ranges from 5 (poor control of asthma) to 25 (complete control of asthma). An ACT score of 19 or less provides optimum balance of sensitivity and specificity for detecting uncontrolled asthma (25).

The ACQ is a validated 7-item questionnaire that asks patients to recall their experiences during the previous week and respond to each question on a 7-point scale, which ranges from 0 (well controlled) to 6 (extremely poor controlled) (18). Values are displayed as mean score ranging from 0-6. A score above 1.5 indicates poorly controlled asthma. We used the shortened version of the ACQ, which excludes pulmonary function parameters in the calculation of the overall score, due to possible effects of obesity on lung function. Prior studies have shown that exclusion of the pulmonary function parameters has no influence on the validity of the ACQ (26).

Lastly, ATAQ, a self-administered 4-item questionnaire, was used to generate a five-level measure of asthma control (0=no control problems to 4=four control problems) (27). The scoring system reflects the level of asthma control in the past 4 weeks and identifies problems in disease management (19,20). A score greater than zero indicates sub-optimal controlled asthma.

Spirometry

Subjects underwent spirometric testing performed by trained personnel. All sites used the same model spirometer (KoKo Spirometer; Pulmonary Data Services; Lewisville, CO). Spirometer calibration was checked using a 3-liter syringe each day of testing. Spirometry techniques were carried out according to American Thoracic Society recommendations (27). Maneuvers were done without the administration of albuterol. Percentage of predicted FEV₁ was calculated according to Hankinson's reference values adjusted for race/ethnicity (28).

Body Mass Index

Due to missing data, weight and height were based on self-report. Out of 292 subjects, 199 and 45 had measured weight and height documented, respectively. Self reported height and weight was validated using measured height and weight obtained from medical charts. The Pearson coefficients for height (n=45), weight (n=199), and BMI (n=45) was 0.97, 0.97, 0.94 respectively (all $p < 0.01$), with a mean difference of 1.06 kilograms between measured and self-reported weights. This observation is consistent with prior findings which show self-reported height and weight to be highly correlated with directly measured values (30,31). As such, self-reported height and weight were used in the final analysis to optimize our analytical power.

Body Mass Index was defined as the weight in kilograms divided by the square of height in meters. The international standard definition of obesity, as determined by the National Heart, Lung, and Blood Institute (NHLBI) was used (29). BMI was classified as normal ($18.5 \leq \text{BMI} \leq 24.9 \text{ kg/m}^2$), overweight ($25 \leq \text{BMI} \leq 29.9 \text{ kg/m}^2$), non-obese ($\text{BMI} \leq 30 \text{ kg/m}^2$) or obese ($\text{BMI} \geq 30 \text{ kg/m}^2$). Obesity was further subdivided into 3 classes according to the NHLBI obesity classification: class I ($30 \leq \text{BMI} \leq 34.9 \text{ kg/m}^2$), class II ($35 \leq \text{BMI} \leq 39.9 \text{ kg/m}^2$), and class III ($\text{BMI} \geq 40 \text{ kg/m}^2$) (29).

Statistical Analysis

Subjects with BMI less than 18.5 kg/m^2 were excluded, since very low BMI can be associated with cachexia and advanced chronic illnesses. The association between BMI and asthma control was assessed using Pearson's correlation. Pearson's chi-square and ANOVA were used to assess the effects of obesity on categorical and continuous variables respectively. Using the available sample size of 292 subjects, we have 80% power to detect a mean between-group difference of 0.26 with the ACQ based on a 2-sided alpha 0.05. Univariate analysis was done to evaluate the association between 1) obesity and asthma control, and 2) increasing BMI level and asthma control. Multivariate regression models were used to adjust for potential confounders, such as age, race, gender, education, insurance and smoking status (Model 1). Another model (Model 2) was used to adjust for additional confounders, which were hypothesized to possibly affect asthma control, including FEV₁, FVC, and selected co-morbidities: GERD, rhinitis, chronic bronchitis, and sinusitis and use of asthma controllers. A two-sided p-value of less than 0.05 was used to determine statistical significance for all analyses. Computations were performed using STATA version 9.2 (College Station, TX).

Results

Patient Characteristics (Table 1)

Of the 298 subjects who agreed to participate in the study, 6 participants were excluded from the analysis based on missing BMI information (N=3), and BMI < 18.5 kg/m², leaving 292 subjects for the final analysis. The majority of the participants were African-American (63%) and women (82%) with a mean age of 47 years (SD 15). Almost one third of the cohort reported having a less than a high school education (27%) and half had public health insurance (52%). There was a high prevalence of smoking with almost two-thirds of participants (63%) having a positive smoking history (36% current smoker, 27% former smoker). The median FEV₁/FVC ratio was 76% (IQR 68, 81) with a median FEV₁% predicted of 71% (IQR 59, 83), and FVC % predicted of 79% (IQR 66, 89). There was no consistent trend with respect to adherence to controller medications observed based on BMI category.

There was a high prevalence of obesity (average BMI was 34.3 kg/m²; range: 18.6-74.1), with only 15% of participants meeting criteria for normal weight, compared to 22% and 63% for overweight and obesity respectively. Of those obese, 21% were classified as obese class I (30 ≤ BMI ≤ 34.9 kg/m²), 17% obese class II (35 ≤ BMI ≤ 39.9 kg/m²), and 24% obese class III (BMI ≥ 40 kg/m²). Analysis by BMI categories showed those obese to more likely be non-smokers, have private insurance, higher level of education (p-value < 0.05). Although the gradient of FEV₁/FVC ratio was not statistically different across BMI categories, increasing BMI level was associated with a lower median FEV₁ % predicted (p= 0.04), and FVC% predicted (p<0.01).

Effect of Obesity on Asthma Control- Table 2

Mean scores from all four asthma control questionnaires, ACCI (8.3), ACT (15.4), ACQ (2.1), and ATAQ (1.3) demonstrated sub-optimal asthma control on average, with 96% of the cohort meeting criteria for sub-optimal control on at least one of the questionnaires. There was no association between BMI and asthma control using any of the four control questionnaires (p > 0.05). This finding persisted when the analysis was repeated using BMI as a categorical variable (Figure 1), or a dichotomous variable comparing obese (BMI ≥ 30 kg/m²) to non-obese (BMI < 30 kg/m²), or obese to normal weight subjects (BMI < 25 kg/m²) p-values > 0.05 (Data Not Shown).

Multivariate analyses adjusted for age, gender, race, insurance status, smoking status, with and without FEV₁ percent predicted and selected co-morbidities showed no association between obesity and mean level of asthma control using all four control instruments (Table 3).

Acute health care utilization and prescribed asthma medications- Table 4

A substantial percentage of participants reported a history of hospitalization (13%) or emergency room visits (35%) for asthma-related complaints in the year preceding enrollment. The majority of subjects were actively being treated for asthma with a reliever (98%) or a long-term controller medication (63%). There was no difference in asthma-related acute health care utilization or prescribed asthma medication by BMI categories. There was a trend for obese subjects to be more likely to report using a long-term controller medication compared to those who were normal weight (67% vs. 57%, p=0.09).

Discussion

In the present study, conducted in an urban population cared for in a primary care setting, obesity was not associated with worse asthma control. Obese patients had asthma control that was similar to that of the non-obese, and even among those who were obese, there was no

tendency toward worse control with greater degrees of obesity. Although there are many health benefits associated with weight loss, findings from the present study do not suggest that weight loss would result in improved asthma control.

Our results add to published medical literature, in which there is evidence both for and against a link between obesity and asthma morbidity. Reports of the effects of obesity on asthma “severity” have been inconsistent, with some showing a positive association (15-17) while others do not (31-33). Recent studies that examined the effects of obesity on asthma “control” have been more consistently positive (17,31). However, differentiation between asthma control and asthma severity may be important when examining the effects of obesity. Even though asthma control and asthma severity are often used interchangeably, they are two distinct concepts, and thus may be affected differently by obesity. According to the latest asthma guidelines, asthma severity pertains to “the intrinsic intensity of the disease process” and should be used to initiate treatment, while asthma control refers to the “degree to which the clinical manifestations are minimized and the goals of therapy are met” and should be used to adjust therapy (21). As such, obesity related factors such as reduction in forced vital capacity and tidal volume, along with increased risk of gastrointestinal symptoms in those obese may contribute to worse asthma control by increasing symptom reporting or seeming to decrease response to therapy without any effects on the intrinsic disease process. This concept is supported by a lack of objective evidence linking obesity to worse asthma pathophysiology including airflow obstruction or airway inflammation (11,34-36).

The distinction between assessment of “severity” and “control” is most striking in the study by Lavoie *et al.* which found obesity to be associated with worse asthma control and not asthma severity, when the latter was assessed according to the 2002 GINA guidelines. Using a validated “control” instrument (the ACQ), obesity was found to be independently associated with worse asthma control (31). In addition, sub-optimal asthma control has been associated with several risk factors including demographics (i.e. African American race, low socioeconomic status) (37), psychosocial factors (i.e. depression, medication adherence) (38, 39), and environment (urban vs. rural setting) (40). The contribution of these individual factors to asthma control and how they are affected by obesity is unknown. It is therefore unclear whether the high prevalence of some of these risk factors in our cohort, compared to previous studies showing a positive association between obesity and asthma control, accounts for our contradictory results by masking any effects of obesity on asthma control.

It is also important to consider that obesity and asthma are two highly prevalent clinical conditions which likely share environmental, behavioral and genetic antecedents. For example, a diet high in calories (including certain fats and carbohydrates) may contribute to obesity, while the same diet may be lacking in foods (whole unprocessed fruits and vegetables for example) with certain antioxidants, which could predispose to worse inflammation and oxidative stress. Sedentary lifestyles may contribute to obesity, but people with less active lifestyles may also spend a greater proportion of time in environments with factors that worsen asthma (e.g., in a home with high allergen concentrations). Thus, previous studies that have found an association of obesity with asthma morbidity may have simply found the coincidence of illness severity that emanates from common underlying risk factors. Although the current study does not assess for these potential confounders, our findings highlights the potential complexity of the obesity and asthma relationship as well as underscores the need for studies that can adequately account for the distribution of suspected risk factors for both conditions.

Our findings are strengthened by the use of four different asthma control measures, which assures that the absence of associations is unlikely to be attributable to misclassification of asthma control by a single survey. There was general agreement between the questionnaires regarding the degree of asthma control for the overall group. In addition, the use of the ACCI,

an asthma control questionnaire specifically designed to be culturally sensitive, makes it unlikely that our findings are due to potential limitations of the other questionnaires to adequately assess level of asthma control in this ethnically diverse population (41). However, certain limitations of the present study design should be taken into account when interpreting our findings. Our findings may not generalize beyond our chosen study population. The current study population is representative of an outpatient urban primary care practice population well-represented with African American and women patients, two groups with high asthma-related morbidity and obesity prevalence (42). Since we selected patients who were seeking care in a clinical setting, our findings may not reflect patients at the well controlled end of the spectrum of control. And if non-obese patients were less likely to attend the clinics, we may have underrepresented the impact of non-obese patients. Nevertheless, the range of BMI observed in our study included normal weight people and a remarkable distribution of obesity, making it unlikely that a spectrum bias played an important role in our findings. In addition, while it is possible that our findings are reflective of unmeasured confounders, we tried to account for common co-morbid conditions that may be associated with both obesity status and worst asthma control, such as active smoking, gastro-esophageal reflux disease (GERD) (51%), rhinitis (40%), sinusitis (54%) and chronic bronchitis (33%). However, even after consideration of these factors we failed to detect any effects of BMI on asthma control in our regression analysis.

In conclusion, in our study of adults with asthma in an urban primary care setting, we did not find an association between obesity and asthma control putting in question prior reports of a link between obesity and asthma control. The most recent NAEPP guidelines recommend that obese patients with asthma “may be advised that weight loss, in addition to improving overall health, might also improve asthma control”(21). This statement, while cautious, may be premature. At this point, evidence is needed from future clinical trials aimed at evaluating the effects of weight loss on asthma control. Until such trials are conducted, weight loss should of course be recommended for people with obesity for other health reasons, rather than for the sake of asthma control. Clinicians should continue to focus their attention on proven treatments including avoidance of environmental triggers and proper use of medications.

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Abbreviations used

ACCI, Asthma Control and Communication Instrument; ACQ, Asthma Control Questionnaire; ACT, Asthma Control Test; ATAQ, Asthma Therapy Assessment Questionnaire; BMI, Body Mass Index; NAEPP, National Asthma Education and Prevention Program; IgE, Immunoglobulin E.

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Figure 1.

Mean asthma control score by BMI categories using ACT, ACQ, ATAQ and ACCI. There was no statistical difference in asthma control among BMI categories. Normal ($18.5 \leq \text{BMI} < 25 \text{ kg/m}^2$), overweight ($25 \leq \text{BMI} < 30 \text{ kg/m}^2$), obesity class I ($30 \leq \text{BMI} < 35 \text{ kg/m}^2$), obesity class II ($35 \leq \text{BMI} < 40 \text{ kg/m}^2$), and obesity class III ($\text{BMI} \geq 40 \text{ kg/m}^2$).

Table 1

Patient demographics by BMI category

	Overall (n=292)	Normal Weight 18.5-24.9 (n=44)	Overweight 25-29.9 (n=65)	Obese I 30-34.9 (n=62)	Obese II 35-39.9 (n=50)	Obese III ≥40 (n=71)	p value
Age, mean (sd)	47 (15)	42 (16)	48 (15)	46 (13)	46 (11)	50 (18)	0.07
Female, N (%)	239 (82)	30 (68)	55 (85)	50 (81)	41 (82)	63 (89)	0.09
Race, N (%)							0.65
African-American	184 (63)	25 (57)	37 (57)	41 (66)	36 (72)	45 (63)	
Caucasian	89 (30)	16 (36)	22 (34)	19 (31)	10 (20)	22 (31)	
Other	19 (7)	3 (7)	6 (9)	2 (3)	4 (8)	4 (6)	
Insurance status, N (%)							0.052
Private	136 (47)	12 (27)	34 (52)	38 (61)	22 (44)	30 (42)	
Public	151 (52)	32 (73)	28 (43)	24 (39)	28 (56)	39 (55)	
Self-Pay	3 (1)	0 (0)	2 (3)	0 (0)	0 (0)	1 (1)	
Other	2 (1)	0 (0)	1 (2)	0 (0)	0 (0)	1 (1)	0.03
Education, N (%)							
Less than high school	78 (27)	17 (39)	16 (25)	12 (19)	13 (26)	20 (28)	
High school or equivalent	96 (33)	14 (32)	12 (18)	25 (40)	17 (34)	28 (39)	
More than high school	118 (40)	13 (30)	37 (57)	25 (40)	20 (40)	23 (32)	
Smoking status, N (%)							0.01
Current	103 (36)	25 (57)	27 (42)	19 (31)	17 (34)	15 (22)	
Former	79 (27)	6 (14)	14 (22)	17 (27)	18 (36)	24 (35)	
Never	107 (37)	13 (30)	23 (36)	26 (42)	15 (30)	30 (43)	
Comorbidities, N (%)							
GERD (n=290)	152 (52)	18 (42)	35 (54)	34 (56)	28 (56)	37 (52)	0.64
Rhinitis (n=286)	165 (58)	29 (69)	40 (63)	32 (52)	29 (59)	35 (50)	0.26
Sinusitis (n=276)	152 (55)	16 (39)	29 (48)	35 (60)	28 (60)	44 (63)	0.09
Chronic bronchitis (n=284)	97 (34)	12 (28)	25 (39)	18 (31)	15 (31)	27 (39)	0.59
Controller Medication Adherence	125 (68)	15 (71)	25 (63)	28 (70)	19 (59)	38 (76)	0.51
Lung function, Median (IQR)							

	Overall (n=292)	Normal Weight 18.5-24.9 (n=44)	Overweight 25-29.9 (n=65)	Obese I 30-34.9 (n=62)	Obese II 35-39.9 (n=50)	Obese III ≥40 (n=71)	p value
FVC % predicted, (n=252)	79 (66,89)	80 (66,89)	86 (78,92)	81 (68,91)	75 (65,85)	71 (59,81)	<0.01
FEV1/FVC (n=236)	76 (68,81)	75 (68,82)	75 (67,81)	74 (67,79)	78 (68,81)	78 (73,85)	0.06
FEV1% predicted (n=235)	71 (59 ,83)	72 (55,86)	79 (70,85)	70 (61,81)	68 (58,81)	68 (56,80)	0.047

Table 2

Mean Asthma Control Scores by BMI Category

	Overall (n=292)	Normal Weight 18.5-24.9 (n=44)	Overweight 25-29.9 (n=65)	Obese I 30-34.9 (n=62)	Obese II 35-39.9 (n=50)	Obese III ≥40 (n=71)	p value [*]
ACT, Mean (sd)	15.4 (4.1)	15.2 (3.9)	15.8 (4.1)	15.6 (4.3)	15.4 (4.4)	14.8 (3.8)	0.71
ATAQ, Mean (sd)	1.3 (1.1)	1.3 (1.0)	1.4 (1.3)	1.3 (1.2)	1.2 (1.2)	1.1 (1.0)	0.5
ACQ, Mean (sd)	2.1 (1.1)	2.2 (1.1)	2.0 (1.1)	2.1 (1.1)	1.9 (1.1)	2.2 (1.0)	0.6
ACCI, Mean (sd)	8.3 (4.2)	8.3 (4.1)	8.2 (4.1)	8.5 (4.8)	7.8 (3.9)	8.6 (4.1)	0.86

ACT -Asthma control test; ATAQ- Asthma therapy assessment questionnaire; ACQ- Asthma control questionnaire; ACCI- Asthma control communication instrument.

^{*} p values determined by ANOVA

Table 3
Linear Regression Analysis of the effect of obesity on asthma control

	Regression Coefficient [†]			
	ACT	ATAQ	ACQ	ACCI
* Model 1	-0.18 (-0.52,0.17)	-0.06 (-0.16,0.04)	-0.03 (-0.57, 0.52)	0.09 (-0.27, 0.44)
** Model 2, n=226	-0.15 (-0.54,0.24)	-0.02 (-0.14,0.10)	-0.23 (-0.84,0.37)	-0.09 (-0.49,0.31)

predicted, GERD, rhinitis, sinusitis, and chronic bronchitis.

[†] All p-values > 0.05.

* **Model 1** adjusted for adjusted for age, gender, race, insurance status, smoking status.

** **Model 2** includes all predictors from **Model 1** as well as FEV₁ percent predicted, FVC percent

Table 4
Self-reported health care use & prescribed asthma medication by BMI category

	Overall (n=292)	Normal Weight 18.5-24.9 (n=44)	Overweight 25-29.9 (n=65)	Obese I 30-34.9 (n=62)	Obese II 35-39.9 (n=50)	Obese III ≥40 (n=71)	p value
Acute Care, N (%)							
Hospitalized within the past year	39 (13)	5 (11)	10 (15)	5 (8)	7 (14)	12 (17)	0.61
ED visit within the past year	102 (35)	16 (36)	28 (43)	18 (29)	21 (42)	19 (27)	0.19
Prescribed Inhalers N (%)							
Short-acting beta-agonists	285 (98)	42 (95)	65 (100)	60 (97)	48 (96)	70 (99)	0.48
Long-term controllers	184 (63)	22 (50)	40 (62)	40 (65)	32 (64)	50 (70)	0.29