

Impact of obesity in asthma: evidence from a large prospective disease management study

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Background: Asthma and obesity continue to have a significant effect on public health. It is widely accepted that obesity may be an independent risk factor for asthma and affect asthma severity and quality of life (QOL).

Objective: To examine the relationship between body mass index (BMI [calculated as weight in kilograms divided by height in meters squared]) and asthma severity, spirometry findings, health care utilization (HCU), and QOL.

Methods: This 12-month prospective randomized controlled trial comparing disease management with traditional care enrolled 902 patients (473 pediatric and 429 adults) representing an underserved population. Data collected at baseline and at 6-month intervals included demographics, asthma severity, medication use, spirometry findings, and HCU. The QOL was assessed using the pediatric and adult versions of the Asthma Quality of Life Questionnaire and the 36-Item Short Form Health Survey. All HCU was determined by means of patient interview and extensive medical record review. Data were analyzed using negative binomial regression and analysis of variance.

Results: In children, 45% were overweight/obese (17% with BMIs >85th percentile; 28% with BMIs ≥95th percentile). In adults, 58% were obese (BMIs ≥30). There was no relationship in children between BMI and severity of asthma, spirometry findings, QOL, or HCU. In adults, there was no relationship between BMI and asthma severity or HCU. Higher BMI was associated with a significant reduction in QOL ($P < .001$). The BMI had an inverse relationship with forced vital capacity but with no other spirometric values.

Conclusions: Obesity was not associated with worse asthma severity, spirometry findings, QOL, or HCU in children. In adults with asthma, obesity was associated with lower forced vital capacity and QOL but not with severity or HCU.

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INTRODUCTION

Asthma and obesity are important public health issues, and their prevalence rates continue to rise. Asthma affects approximately 7% of Americans, and related costs are estimated to be more than \$12 billion per year. Between 1980 and 2008, the prevalence of asthma increased in children from 3.5% to 9.4% and in adults from 2.9% to 7.3%.¹ Between 2001 and 2003, an average of 11.6 million people experienced an asthma attack, and asthma accounted for 61.2 unscheduled office visits per 100 persons with asthma, 1.8 million emergency department visits, 504,000 hospitalizations, and approximately 4,000 deaths.² In addition to the rising prevalence of asthma, the prevalence of obesity in the US population is rising in all age groups. Data from the 2003–2004 National Health and Nutrition Examination Survey indicate that an estimated 66% of US adults are overweight or obese. Two National Health and Nutrition Examination Surveys

(1976–1980 and 2003–2004) show similar findings in children: for ages 2 to 5 years, an increase in obesity from 5.0% to 13.9%; for ages 6 to 11 years, an increase from 6.5% to 18.8%; and for ages 12 to 19 years, an increase from 5.0% to 17.4%.³

Obesity can have negative consequences on basic respiratory physiologic factors, such as restrictive volumes, increased work of breathing, obstructive sleep apnea, and impaired gas exchange. A growing body of literature describes the relationship between asthma and obesity, and cross-sectional studies indicate an increased prevalence of asthma in obese individuals. Several mechanisms are implicated in this causal relationship, with most of them revolving around the principle that obesity leads to a pro-inflammatory state that worsens airway inflammation and that subsequently leads to airway hyperreactivity (AHR).⁴

Although cross-sectional studies^{5–8} in adults have demonstrated an increased prevalence of asthma in obese individuals, many of these studies relied on self-reported weight and height. Another potential limitation of these studies is that most relied on a self-reported diagnosis of asthma rather than on physician records, physiologic evaluation, or response to inhaled therapy. This raises the possibility that the respiratory symptoms were related to the physiologic impairment from obesity rather than to asthma. There is more heterogeneity in the pediatric epidemiologic literature relating to the obesity–asthma relationship. There are a limited number of prospective studies in the adult and pediatric populations that specifically look at obesity as it relates to asthma severity, quality

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of life (QOL), spirometric values, and health care utilization (HCU). The purpose of this study was to evaluate the relationship of body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared) in adults and children with asthma to asthma control/severity, spirometry findings, QOL, and HCU using data obtained from a large prospective disease management trial.

METHODS

The South Texas Asthma Management Project was a single-center, prospective, randomized controlled trial conducted by the University of Texas Health Science Center at San Antonio and approved by its institutional review board. Written consent was obtained from each patient before randomization. Nine hundred two patients (aged 5–64 years; 429 adults and 473 pediatric) with asthma were randomly assigned to 1 of 3 study groups after consenting to participate in the study. Patients included in this study had a documented physician diagnosis of asthma, access to a telephone, and 1 or more of the following: 1 hospitalization or emergency department visit, 4 or more office visits with a diagnosis of asthma in the previous 12 months, or a physician diagnosis of moderate to severe persistent asthma supported by results of pulmonary function testing. Patients were excluded if they had any other lung disease or any diagnosis that required long-term use of systemic corticosteroids. Details of the study have previously been published.⁹

Severity of asthma, spirometry findings, and QOL were analyzed using baseline data. Data on HCU were collected prospectively for each treatment group. Because there were no significant differences between any of the groups, HCU is presented by combining the treatment groups into pediatric patients (aged 5–17 years) and adult patients (aged 18–64 years). Asthma severity was categorized using the National Heart, Lung, and Blood Institute asthma severity classification and the Global Initiative for Asthma guidelines.^{10–12} Adults were administered the 36-Item Short Form Health Survey (SF-36) and the Asthma Quality of Life Questionnaire (AQLQ) during each visit.¹³ For children, the Pediatric AQLQ and the Pediatric Asthma Caregivers Quality of Life Questionnaire were used.¹⁴

All the participants were telephonically polled regarding HCU every 2 months throughout the trial. Using this self-

report data, study staff obtained authorization from participants for release of medical records and made requests of all known providers, facilities, and pharmacies for records covering the enrollment period. Medical record abstraction was performed by study staff. The abstraction process was monitored by audits of randomly selected medical records. Utilization outcome analysis was based on events verified by medical record review.

Continuous variables were analyzed as mean and standard deviation, and intergroup differences were analyzed using the *t* test and 1-way analysis of variance. Group differences in categorical variables were analyzed by means of χ^2 tests. Differences in rates of utilization were analyzed using negative binomial regression. A $P \leq .05$ was considered significant.

RESULTS

Patient characteristics are given in Table 1. Of the 902 patients, 429 were adults and 473 were children. In the adult population, the mean age was 42.7 years, with most adults being women (78%) and Hispanic (51%). The prevalence of obesity was 58% in the adult population and 28% in the pediatric population. The mean age of the pediatric population was 9.5 years, with most children being boys (59%) and Hispanic (68%). There were no differences in mean age, sex, or race/ethnicity between obese and nonobese pediatric patients. Adult obese patients were significantly more likely to be older ($P = .007$), female ($P = .01$), and Hispanic ($P = .001$) and less likely to be non-Hispanic white ($P < .001$).

Asthma severity as measured by the National Asthma Education and Prevention Program guidelines is given in Table 2. Two-thirds of the pediatric population had moderate or severe persistent asthma, with most of the remainder having mild persistent asthma. At baseline, there were no significant differences in asthma severity by BMI ($P > .60$ for all comparisons). Most adult participants (>75%) had moderate to severe persistent asthma. Similar to the analysis in children, baseline severity of asthma in adults did not differ by BMI ($P > .40$ for all comparisons).

Baseline spirometry values are reported in Table 3. At baseline, there were no differences in percentage of predicted forced expiratory volume in 1 second (FEV₁), FEV₁ to forced vital capacity (FVC), FVC, or bronchodilator response between

Table 1. Demographic Characteristics of the 902 Study Patients

	Adult population				Pediatric population			
	Total (N = 429)	BMI <30 (n = 179)	BMI >30 (n = 250)	P value	Total (N = 473)	<85th percentile (n = 260)	>95th percentile (n = 132)	P value
Age, mean (SD), y	42.7 (12.3)	40.8 (12.9)	44.1 (11.7)	.007	9.5 (3.3)	9.4 (3.3)	9.9 (3.5)	.28
Female sex, %	78	72	82	.01	40.6	42	36.4	.50
Race/ethnicity, %								
Hispanic	51	42	58	.001	67.8	65.0	72.7	.29
White	31	39	25	<.001	15.4	17.3	12.1	.40
Black/other	18	19	17	.58	16.7	17.6	15.1	.76

Abbreviation: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared).

Table 2. Asthma Severity as Defined by National Asthma Education and Prevention Program Guidelines

	Pediatric population ^a				Adult population ^b		
	Total, No. (N = 414)	<85th percentile, % (n = 227)	85th to <95th percentile, % (n = 69)	≥95th percentile, % (n = 118)	Total, No. (N = 372)	BMI <30, % (n = 153)	BMI ≥30, % (n = 219)
Mild intermittent	28	7.1	13.0	2.5	12	3.3	3.2
Mild persistent	119	26.4	24.6	35.6	72	20.3	18.7
Moderate persistent	139	36.0	36.2	27.0	117	30.0	32.4
Severe persistent	128	30.4	26.1	34.8	171	46.4	45.6

Abbreviation: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared).

^a For all comparisons, $P > .60$

^b For all comparisons, $P > .40$.

obese and nonobese children with asthma ($P > .5$). In adults, there were no significant differences in the percentage of predicted FEV₁ across all groups ($P = .96$); however, obesity was associated with a worse prebronchodilator FVC. Forty-seven percent of patients with a BMI of at least 30 (vs 35% with a BMI <30, $P = .01$) had a pre-BD FVC less than 80% of predicted. Because obesity may be associated with a disproportionate decline in FVC, obstruction as measured using the FEV₁:FVC ratio was most likely underrepresented in the obese population. There was no significant difference in bronchodilator response between BMI groups in either adults or children.

At enrollment, QOL was measured in children using the Pediatric AQLQ score. There was no significant difference in the mean scores of the obese and nonobese groups (4.8 vs 5.0, $P = .16$). In adults, higher BMI was associated with lower QOL as assessed using the SF-36 physical and mental component

scores, and the AQLQ score (Table 4). The mean AQLQ scores for obese and nonobese patients were 3.9 and 4.2, respectively ($P = .006$). This mean difference was even greater when the very obese (BMI ≥40) were compared with the nonobese (3.7 vs 4.2, $P < .001$). A difference of 0.5 denotes clinical significance according to the AQLQ scoring definitions.¹⁵

In children, there was no relationship between BMI and hospital admissions, ED visits, unscheduled office visits, or overall HCU ($P > .19$ -.79). Likewise, in adults, there were no significant differences between any BMI group and HCU ($P > .09$ -.78).

DISCUSSION

The concurrent rise in asthma and obesity has prompted researchers to investigate associations between these 2 disorders. To date, 13 prospective studies have examined the

Table 3. Baseline Spirometry Findings

	Pediatric population					Adult population			
	Total (N = 473)	<85th percentile (n = 260)	85th-94th percentile (n = 81)	≥95th percentile (n = 132)	P value ^a	Total (N = 429)	BMI <30 (n = 179)	BMI ≥30 (n = 250)	P value ^b
FEV ₁ , mean (SD), % of predicted	98.0 (19)	98.5 (19.6)	97.0 (18)	98.5 (19)	.83	77 (19)	77 (21)	77 (17)	.96
FVC, mean (SD), % of predicted	103.9 (18.7)	104.2 (21.8)	103.6 (15.1)	103.6 (16.4)	.96	81 (17)	84 (18)	79 (17)	.006
FVC <80% of predicted pre-BD, % ^c	5.7 (14)	4.5 (5)	4.2 (2)	8.0 (7)	.52	42 (170)	35 (59)	47 (111)	.01
FVC <80% of predicted post-BD, % ^c	NA	NA	NA	NA	NA	34 (124)	27 (40)	39 (84)	.01
FEV ₁ :FVC <75%, %	19.7 (48)	17.4 (19)	21.2 (10)	21.8 (19)	.71	37 (153)	46 (78)	32 (75)	.004
Positive (12% and 200 mL) response to BD, % ^d	21.1 (65)	21.2 (33)	22.6 (12)	20.2 (20)	.94	26 (94)	30 (45)	23 (49)	.14

Abbreviations: BD, bronchodilator; BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity; NA, not applicable.

^a Comparison between less than the 85th percentile and the 95th percentile or greater. $P > .05$ for all group comparisons.

^b Comparison between BMI less than 30 vs BMI of 30 or greater.

^c For adults, findings did not change when the lower limit of normal was analyzed.

^d According to the 2005 American Thoracic Society/European Respiratory Society guidelines.

Table 4. Quality of Life Scores for Adults as Measured Using the SF-36 and the AQLQ

	BMI <30	BMI ≥30	P value ^a	BMI 30–39.9	BMI ≥40
AQLQ score, mean (SD)	4.2 (1.2)	3.9 (1.3)	.006	4.0 (1.3)	3.7 (1.2) ^b
SF-36 MCS, mean (SD)	51.4 (10.0)	48.9 (10.0)	.01	NA	NA
SF-36 PCS, mean (SD)	52.4 (9.1)	48.3 (8.7)	<.001	NA	NA

Abbreviations: AQLQ, Asthma Quality of Life Questionnaire; BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); MCS, mental component score; NA, not available; PCS, physical component score; SF-36, 36-Item Short Form Health Survey.

^a Quality of life was significantly lower for the AQLQ and for the MCS and PCS of the SF-36.

^b The AQLQ score reaches “minimal” clinical significance only in comparing BMI less than 30 with BMI of at least 40 (very obese).

relationship between obesity and the onset of asthma: 8 in adults^{16–23} and 5 in children.^{24–28} Each study enrolled several thousand subjects with follow-up periods up to 20 years. All but 1 study²³ revealed an increased incidence of asthma in obese or overweight individuals. Although it is possible that reduced exercise or use of oral corticosteroids in patients with asthma could result in obesity, each of these longitudinal studies showed that obesity preceded asthma. Although several mechanisms have been implicated in this causal relationship, most animal research and human data suggest either that obesity leads to a pro-inflammatory state that worsens airflow limitation or that the 2 disorders have a shared genetic basis.²⁹ Obesity may also be more common in individuals with asthma than in the general population. Thomson et al³⁰ noted that 75% of patients presenting to the emergency department with asthma were either overweight or obese, a percentage significantly greater than the prevalence of these conditions in the general population. The study also found a high incidence of obesity; 58% of adults and 28% of children in this study met the criteria for obesity. This finding was even more striking in adult women of Hispanic descent.

Conflicting findings regarding the observed association between obesity and asthma prevalence or asthma severity may be due to differences in the definition of asthma.^{31,32} In contrast to many previous studies, the present study used stringent criteria for the diagnosis of asthma. We enrolled only patients with a documented physician diagnosis of asthma supported by clinical or spirometric data. Approximately 40% of these patients demonstrated obstruction on baseline spirometry, and more than 25% demonstrated a positive bronchodilator response despite being maintained on 1 or more controller medications. Therefore, it seems unlikely that a significant portion of this study population represented individuals with pulmonary symptoms related to obesity rather than true asthma.

The present study did not find an association between obesity and asthma severity in either the adult or pediatric population; however, the adult population demonstrated a worse QOL. We examined the relationship between BMI and asthma severity in adults and children. Adults with a BMI greater than 30 did not have more severe asthma based on spirometry findings, nocturnal waking, or rate of exacerbations. When use of controller medications was included in the analysis, using the Global Initiative for Asthma 2002 severity guidelines, we still did not detect a significant difference in severity by BMI group. Simi-

larly, we found no differences in severity in children by BMI (<85th percentile, 85th–94th percentile, or ≥95th percentile). These findings may be partially explained by a study by Schachter et al³³ that found that severe obesity was a significant risk factor for wheezing, “recent asthma,” and use of inhaled medication; however, the authors found no association of obesity with pulmonary function findings (peak expiratory flow rate, FEV₁:FVC, or forced expiratory flow between 25% and 75%) or with airway hyperresponsiveness, as measured by means of histamine provocation. Therefore, although symptoms and medication use were more common in obese patients, the authors did not find objective evidence to support that asthma was more severe in this group.³³

Obesity causes a variety of physiologic changes, including a reduction in airway compliance, lung volumes, and peripheral airway diameter.³⁴ In the present study, spirometry and flow volume loops were obtained by trained primary care physicians and were reviewed by an adult or pediatric pulmonologist. All suboptimal studies were excluded from analysis. In adults, we found no significant differences in the percentage of predicted FEV₁ or in the response to bronchodilators comparing obese with nonobese patients. Obesity was associated with a significant decrease in prebronchodilator FVC, which resulted in a significant increase in the FEV₁ ratio (FEV₁:FVC). Unlike nonobese asthmatic patients, the reductions in FEV₁ and FVC in obese individuals are typically symmetrical and result in a preserved FEV₁:FVC ratio.³⁴ This symmetrical fall in FEV₁ and FVC could tend to obscure obstruction and lead to the diagnosis of restrictive lung disease. Although we did not investigate sex differences in this study, several studies agree that the effect of weight gain on lung function is greater in men than in women, probably due to sex-related differences in fat distribution.³⁵ In central obesity, a pattern most often seen in males, the fat deposits in subcutaneous and visceral tissues of the abdomen, resulting in an increased mechanical effect on the diaphragm.³⁶ In addition, central adipose tissue and visceral fat are more metabolically active and have been linked to the overproduction of adipokines and cytokines that may affect AHR.³⁵ In 1 previous study,³⁷ abdominal obesity was the strongest predictor of lung function impairment. Finally, obese adults with asthma may differ from nonobese adults in their response to methacholine therapy, demonstrating a greater decrease in inspiratory capacity and a greater increase in functional residual capacity. These differences may contribute to reported

differences in perceived impairment and asthma symptoms between obese and nonobese adults.³⁸

In children, there were no significant differences in FEV₁, FVC, or FEV₁:FVC ratio between obese and nonobese patients. In addition, the response to bronchodilators was similar in pediatric patients who were normal weight, overweight, or obese. These findings are not surprising because the literature for airflow obstruction and AHR are conflicting.³⁴ One study³⁹ of almost 6,000 children showed that obesity was associated with symptoms and use of rescue medication but not with AHR. In contrast to the reported decline in FVC with obesity in adults, investigators have reported either no change in FVC or even an increased FVC with increased BMI in children.^{27,40}

The present study found no difference in QOL between obese and nonobese children. One previous study⁴¹ in children showed significantly lower QOL in obese children with asthma compared with nonobese children with asthma. However, in that study,⁴¹ only 32% of children were taking inhaled corticosteroids. In adults, there was a difference in QOL based on SF-36 and AQLQ scores. Obese patients had a statistically significantly lower score (worse QOL) on the physical and mental outcome scores of the SF-36. There was also a statistically significantly lower mean score on the AQLQ. In a subgroup analysis of patients with severe obesity (BMI ≥40), reduced QOL reached statistical and clinical significance.

The present study results in adults are similar to those of Lavoie.⁴² The study by Lavoie evaluated almost 250 overweight or obese adults with asthma and found the total AQLQ score to be statistically significantly different from that of patient with a normal BMI. In contrast, 2 other studies^{43,44} found no significant associations between BMI and QOL.

A recent study⁴⁵ evaluated the role of weight reduction in obese patients with asthma. This small randomized study demonstrated that weight reduction was associated with improvement in pulmonary function, symptoms, and asthma-related QOL. It is possible that obese patients falsely attribute weight-related respiratory symptoms to asthma rather than to mechanical limitations. Future studies examining the effects of weight loss on asthma should include measures of asthma control and AHR to better understand whether weight loss intervention actually affects the underlying pathogenesis of asthma.

Two large prospective studies suggest that obesity may have adverse long-term effects on asthma. In an international cohort of adults with asthma, increasing BMI across time was negatively associated with asthma remission.⁴⁶ Similarly, in the Tucson Longitudinal Study,⁴⁷ obesity was a significant independent predictor of unremitting asthma in children.

Few studies actually evaluate the impact of obesity on asthma-related HCU. The present study prospectively examined HCU not only through patient interviews but also by review of medical records. We did not detect a significant relationship between BMI and hospital admissions, ED visits, or unscheduled office visits in either children or adults. One previous study⁴⁸ suggested that increased BMI was associated with a delay in and avoidance of seeking health care. Al-

though delay in seeking care may have affected these results, the duration of this study (12 months of observation) makes this confounder less likely to have affected the results. Other studies have shown a positive association between obesity and self-reported HCU for asthma. In the cross-sectional National Asthma Study, obesity was associated with increased self-reported asthma severity and HCU; however, less than one-third of respondents were taking inhaled corticosteroids.⁴⁹ In the National Cooperative Inner-City Asthma Study of children, a higher baseline BMI was associated with more subsequent daytime asthma symptoms and emergency department visits but not with night symptoms, slowed play, or hospitalizations.⁵⁰

There are some limitations to this study. The patient population was predominantly an indigent, Hispanic population, which may limit the generalizability of the results to other patient populations. In addition, we sampled patients with more severe asthma, and the results may not apply to patients with less severe disease. Also, some patients may have used health care facilities that we were unable to capture during data gathering. We tried to minimize this possibility via periodic telephone communications, face-to-face interviews, and review of all medical records. Finally, these patients had a higher incidence of inhaled corticosteroid use and compliance as assessed by pharmacy records compared with that reported in the literature (69% vs 20%–30%).⁹ Thus, it is possible that high utilization of inhaled corticosteroids may overcome the detrimental effects of obesity in asthma.

In conclusion, this study did not demonstrate that obesity had a significant effect on severity, spirometry findings, QOL, or HCU in children with asthma. Obese adults with asthma had a lower FVC that may have masked an obstructive defect on spirometry. Obese adults with asthma, compared with nonobese patients, had a lower QOL; however, there was no difference in asthma severity or HCU.

REFERENCES

1. National Center for Health Statistics. Faststats: asthma. Centers for Disease Control and Prevention Web site. <http://www.cdc.gov/nchs/fastats/asthma.htm>. Accessed September 5, 2010.
2. Moorman JE, Rudd RA, Johnson CA, et al; Centers for Disease Control and Prevention (CDC). National surveillance for asthma—United States, 1980–2004. *MMWR Surveill Summ*. 2007;56:1–54.
3. National Center for Health Statistics. Prevalence of overweight and obesity among adults: United States, 2003–2004. http://www.cdc.gov/nchs/products/pubs/pubd/hestats/overweight/overweight_adult_03.
4. Shore SA, Johnston RA. Obesity and asthma. *Pharmacol Ther*. 2006; 110:83–1025.
5. Beckett WS, Jacobs DR Jr, Yu X, et al. Asthma is associated with weight gain in females but not males, independent of physical activity. *Am J Respir Crit Care Med*. 2001;164:2045–2050.
6. Chen Y, Dales R, Krewski D, et al. Increased effects of smoking and obesity on asthma among female Canadians: the National Population Health Survey, 1994–1995. *Am J Epidemiol*. 1999;150:255–262.
7. Guerra S, Sherrill DL, Bobadilla A, et al. The relation of body mass index to asthma, chronic bronchitis, and emphysema. *Chest*. 2002;122: 1256–1263.
8. Shaheen SO, Sterne JA, Montgomery SM, et al. Birth weight, body mass index and asthma in young adults. *Thorax*. 1999;54:396–402.

9. Galbreath AD, Smith B, Wood PR, et al. Assessing the value of disease management: impact of two disease management strategies in an under-served asthma population. *Ann Allergy Asthma Immunol.* 2008;101: 599–607.
10. Pellegrino R, Viegi G, Brusasco V, et al. Interpretive strategies for lung function tests: ATS guidelines. *Eur Respir J.* 2005;26:948–968.
11. Expert Panel Report: guidelines for the diagnosis and management of asthma: update on selected topics 2002. National Heart, Lung, and Blood Institute Web site. http://www.nhlbi.nih.gov/guidelines/archives/epr-2_upd/asthmafullrpt_archive.pdf.
12. Masoli M, Fabian D, Holt S, et al; Global Initiative for Asthma (GINA) Program. The global burden of asthma: executive summary of the GINA. *Allergy.* 2004;59:469–478.
13. Juniper EF, Guyatt GH, Ferrie PJ, et al. Measuring quality of life in asthma. *Am Rev Respir Dis.* 1993;147:832–838.
14. Juniper EF, Guyatt GH, Feeny DH, et al. Measuring quality of life in the parents of children with asthma. *Qual Life Res.* 1996;5:27–46.
15. Juniper EF, Guyatt GH, William A, et al. Determining the minimal important change in a disease-specific quality of life questionnaire. *J Clin Epidemiol.* 1994;47:81–87.
16. Camargo CA Jr, Weiss ST, Zhang S, et al. Prospective study of body mass index, weight change, and risk of adult-onset asthma in women. *Arch Intern Med.* 1999;159:2582–2588.
17. Beckett WS, Jacobs DR Jr, Yu X, et al. Asthma is associated with weight gain in females but not males, independent of physical activity. *Am J Respir Crit Care Med.* 2001;164:2045–2050.
18. Chen Y, Dales R, Tang M, et al. Obesity may increase the incidence of asthma in women but not men: longitudinal observations from the Canadian National Population Health Surveys. *Am J Epidemiol.* 2002; 155:191–197.
19. Guerra S, Sherrill DL, Bobadilla A, et al. The relation of body mass index to asthma, chronic bronchitis, and emphysema. *Chest.* 2002;122: 1256–1263.
20. Huovinen E, Kaprio J, Koskenvuo M. Factors associated to lifestyle and risk of adult onset asthma. *Respir Med.* 2003;97:273–280.
21. Ford ES, Mannino DM, Redd SC, et al. Body mass index and asthma incidence among USA adults. *Eur Respir J.* 2004;24:740–744.
22. Nystad W, Meyer HE, Nafstad P, et al. Body mass index in relation to adult asthma among 135,000 Norwegian men and women. *Am J Epidemiol.* 2004;160:969–976.
23. Stanley AH, Demissie K, Rhoads GG. Asthma development with obesity exposure: observations from the cohort of the National Health and Nutrition Evaluation Survey Epidemiologic Follow-up Study (NHEFS). *J Asthma.* 2005;42:97–99.
24. Castro-Rodriguez JA, Holberg CJ, Morgan WJ, et al. Increased incidence of asthma like symptoms in girls who become overweight or obese during the school years. *Am J Respir Crit Care Med.* 2001;163: 1344–1349.
25. Chinn S, Rona RJ. Can the increase in body mass index explain the rising trend in asthma in children? *Thorax.* 2001;56:845–850.
26. Gilliland FD, Berhane K, Islam T, et al. Obesity and the risk of newly diagnosed asthma in school-age children. *Am J Epidemiol.* 2003;158: 406–415.
27. Gold DR, Damokosh AI, Dockery DW, et al. Body mass index as a predictor of incident asthma in a prospective cohort of children. *Pediatr Pulmonol.* 2003;36:514–521.
28. Guerra S, Wright AL, Morgan WJ, et al. Persistence of asthma symptoms during adolescence: role of obesity and age at onset of puberty. *Am J Respir Crit Care Med.* 2004;170:78–85.
29. Beuther DA, Sutherland ER. Overweight, obesity, and incident asthma: a meta-analysis of prospective epidemiologic studies. *Am J Respir Crit Care Med.* 2007;175:661–666.
30. Thomson CC, Clark S, Camargo CA, et al. Body mass index and asthma severity among adults presenting to the emergency department. *Chest.* 2003;124:795–802.
31. Jarvis D, Chinn S, Potts J, et al. Association of body mass index with respiratory symptoms and atopy: results from the European Community Respiratory Health Survey. *Clin Exp Allergy.* 2002;32:831–837.
32. Sin DD, Jones RL, Man SFP. Obesity is a risk factor for dyspnea but not for airflow obstruction. *Arch Intern Med.* 2002;162:1477–1481.
33. Schachter LM, Salome CM, Peat JK, et al. Obesity is a risk for asthma and wheeze but not airway hyperresponsiveness. *Thorax.* 2001;56:4–8.
34. Beuther DA, Weiss ST, Sutherland ER. Obesity and Asthma. *Am J Respir Crit Care Med.* 2006;174:112–119.
35. McClean KM, Kee F, Young IS, et al. Obesity and the lung. *Thorax.* 2008;63:649–654.
36. Carey IM, Cook DG, Strachan DP. The effects of adiposity and weight change on forced expiratory volume decline in a longitudinal study of adults. *Int J Obes Relat Metab Disord.* 1999;23:979–985.
37. Leone N, Courbon D, Thomas F, et al. Lung function impairment and metabolic syndrome: the critical role of abdominal obesity. *Am J Respir Crit Care Med.* 2009;179:509–516.
38. Sutherland TJT, Cowan JO, Taylor DR. Dynamic hyperinflation with bronchoconstriction. *Am J Respir Crit Care Med.* 2008;177:970–975.
39. Bibi H, Shoseyov D, Feigenbaum D, et al. The relationship between asthma and obesity in children: is it real or a case of over diagnosis? *J Asthma.* 2004;41:403–410.
40. Tantisira KG, Litonjua AA, Weiss ST, et al. Association of body mass with pulmonary function in the Childhood Asthma Management Program. *Thorax.* 2003;58:1036–1041.
41. Van Gent R, van der Ent CK, Rovers MM, et al. Excessive body weight is associated with additional loss of quality of life in children with asthma. *J Allergy Clin Immunol.* 2007;119:591–596.
42. Lavoie KL, Bacon SL, Labrecque M, et al. Higher BMI is associated with worse asthma control and quality of life but not asthma severity. *Respir Med.* 2006;100:648–657.
43. Ackerman MJ, Calacanis CM, Madsen MK. Relationship between asthma severity and obesity. *J Asthma.* 2004;41:521–526.
44. Varraso R, Siroux V, Maccario J, et al. Asthma severity is associated with body mass index and early menarche in women. *Am J Respir Crit Care Med.* 2005;171:334–339.
45. Stenius-Aarniala B, Poussa T, Kvarnstrom J, et al. Immediate and long term effects of weight reduction in obese people with asthma: randomised controlled study. *BMJ.* 2000;320:827–832.
46. De Marco R, Marcon A, Jarvis D, et al. Prognostic factors of asthma severity: a 9-year international prospective cohort study. *J Allergy Clin Immunol.* 2006;117:1249–1256.
47. Guerra S, Wright AL, Morgan WJ, Sherrill DL, Holberg CJ, Martinez FD. Persistence of asthma symptoms during adolescence: role of obesity and age at the onset of puberty. *Am J Respir Crit Care Med.* 2004;170:78–85.
48. Figueroa-Munoz JI, Chinn S, Rona RJ. Association between obesity and asthma in 4–11 year old children in the UK. *Thorax.* 2001;56:133–137.
49. Taylor B, Mannino D, Brown C, et al. Body mass index and asthma severity in the National Asthma Survey. *Thorax.* 2008;63:14–20.
50. Belamarich PF, Luder E, Kattan M, et al. Do obese inner-city children with asthma have more symptoms than non-obese children with asthma? *Pediatrics.* 2000;106:1436–1441.

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