The Association Between Obesity and Asthma Is Stronger in Nonallergic Than Allergic Adults*

Yue Chen, PhD; Robert Dales, MD; and Ying Jiang, MSc

Study objective: To determine the modifying effects of sex and allergy history on the association between body mass index (BMI) and asthma prevalence.

Design: Cross-sectional study of 86,144 Canadians who were 20 to 64 years of age in 2000–2001.

Setting: A national survey.

Measurements and analysis: Self-reported asthma, allergy history, height, and weight. Logistic regression analysis was used to detect effect modification and to adjust for covariates. Population weight and design effects associated with complex survey design were taken into consideration.

Results: The adjusted odds ratios (ORs) for obesity associated with asthma was 1.85 (95% confidence interval [CI], 1.65 to 2.07) for women and 1.21 (95% CI, 1.05 to 1.40) for men. One unit of increased BMI was associated with an approximate 6% increase in asthma risk in women, and 3% in men. A stronger association between obesity and asthma was observed in nonallergic women than in allergic women, with the adjusted ORs being 2.53 (95% CI, 2.11 to 3.04) and 1.57 (95% CI, 1.36 to 1.82), respectively. For men, the corresponding ORs were 1.30 (95% CI, 1.05 to 1.62) and 1.18 (95% CI, 0.98 to 1.53), respectively.

Conclusions: Obesity is likely to have a larger effect on nonallergic asthma. The greater prevalence of nonallergic asthma in women may explain the stronger obesity-asthma association seen in women compared with men and children who have a greater prevalence of allergic asthma.

Key words: allergy; asthma; body mass index; obesity; sex

Abbreviations: BMI = body mass index; CCHS = Canadian Community Health Survey; CI = confidence interval; OR = odds ratio

Both asthma and obesity are important health issues in Canada and other developed countries. There is an increasing body of evidence that obesity is an important determinant of asthma, particularly for adults.1–16 The association between obesity and asthma appears to be modified by sex. Studies of women have demonstrated an increased risk of asthma in obese persons.1–4 When stratified by sex, several studies have found that the obesity-asthma association either is only significant in women but not in men7–10 or is stronger in women than in men.11 However, the sex-related difference was not obvious in a Norwegian cohort study13 with a long period of observation from 1963 and 2002, with the relative risks of asthma associated with obesity being 1.78 in men and 1.99 in women. The sex modifica-

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tion of the association between obesity and asthma needs to be explored further.

The mechanisms of the obesity-asthma association are not known, and there are a number of possibilities. The European Community Respiratory Health Survey found that obesity was associated with an increased risk of wheeze with shortness of breath and other asthma-like symptoms. However, body mass index (BMI) was not associated with hay fever or nasal allergies, specific IgE levels for house dust mite, grass, or cat dander, or with total IgE, suggesting that atopy may not be involved in the obesity-asthma association. Another study found that severe obesity was associated with asthma, but not with atopy and airway hyperresponsiveness. Based on these observations, we speculate that obesity may have different effects on patients with allergic asthma than on those with nonallergic asthma.

To further investigate the association between obesity and asthma, and its possible mechanisms, we examined the modifying effects of sex and history of allergy on the association between BMI and asthma based on data from a national survey that was conducted in Canada. We hypothesized that the relationship of obesity and asthma would be stronger in nonallergic persons than in allergic persons. A large sample size provided an opportunity to examine the association stratified by sex and history of allergy with good precision.

**Materials and Methods**

This analysis was based on the 2000–2001 Canadian Community Health Survey (CCHS) that was conducted by Statistics Canada (Ottawa, ON, Canada). The target population of the survey was household residents aged ≥12 years in all of the 10 provinces and 3 territories in Canada. Persons living on Indian reserves or Crown lands, clientele of institutions, full-time members of the Canadian Armed Forces, and residents of certain remote regions were excluded from this survey.

A multistage stratified sampling design was used in the survey with dwelling to be the final sampling unit. A representative sample of 136,937 households was selected for the CCHS, with a national combined response rate of 84.7%. In all selected dwellings, a knowledgeable household member was asked to supply basic demographic information on all residents of the dwelling. Depending on the composition of the household, one or two members were then selected for a more in-depth interview. The survey included questions related to health status, health-care utilization, and health determinants.

The present study was based on data from 86,144 subjects who were 20 to 64 years of age (41,742 men and 44,402 women), who responded to questions about asthma and provided information on height and weight. The survey asked about “long-term conditions” that had lasted or were expected to last ≥6 months and that had been diagnosed by a health professional. Respondents who answered the following questions affirmatively were considered to have asthma: “Do you have asthma diagnosed by a health professional?”

BMI was calculated as follows: 
$$\text{BMI} = \frac{\text{weight (in kilograms)}}{\text{height (in meters)}^2}.$$ 
Subjects were grouped into the following four BMI categories: underweight (<20.0 kg/m²); normal weight (20.0 to 24.9 kg/m²); overweight (25.0 to 29.9 kg/m²); and obesity (≥30.0 kg/m²). A positive history of allergy was indicated if an affirmative response was given to either of the following questions: “Do you have any food allergies diagnosed by a health professional?”; or “Do you have other allergies diagnosed by a health professional?”

Current smokers were respondents who reported smoking cigarettes every day at the time of the survey. Former smokers were those who reported smoking cigarettes daily in the past but were not smoking at the time of the survey. Otherwise, subjects were classified as nonsmokers. Subjects were classified into low-income, middle-income, or high-income groups based on the total household income adjusted for the number of household members. Subjects were grouped into two education categories. Subjects not proceeding beyond secondary school were classified into the lower education group. The higher education group included those who had been admitted to college or university and those with a post-secondary school certificate or diploma. Other variables included in the analysis were age, marital status (ie, married or common law or partner, single, separated, or divorced or widowed), immigrant status (yes or no), alcohol drinking (current drinker, former drinker, drink fewer than one time per week, and drink one or more times per week), and regular exercise (yes or no).

We calculated the prevalence of asthma according to BMI, age, and history of allergy in men and women separately. Logistic regression analysis was used to examine the association between BMI and asthma stratified by sex and history of allergy, before and after adjustment for covariates. Model parameters were estimated by using the method of maximum likelihood and were tested for significance by using the Wald statistic. The effect modifications of sex and allergy history on a multiplicative scale were assessed by including interaction terms between BMI and sex or allergy history in logistic regression models. The CCHS used a complex survey design. All of the point estimates were weighted to the Canadian population, and the average design effect was taken into consideration in variance estimation in both simple and multivariate analyses. All the statistical analyses were conducted using a statistical software package (SAS, version 8.2; SAS Institute, Cary, NC).

**Results**

The prevalence of physician-diagnosed asthma was higher in women than in men (9.9% vs 6.0%, respectively). In both men and women, the prevalence of asthma was higher in the group of persons 20 to 29 years of age and did not show a marked difference in other age groups (Table 1). Allergic men and women had a fourfold to fivefold higher risk of asthma compared with their nonallergic counterparts. Both obese and overweight women had an increased risk of asthma compared with those of normal weight. Among men, overweight people and people of normal weight showed a similar risk of asthma, while obese people had an increased risk (Table 1).

A multiple logistic regression model was used to assess the BMI associated with the prevalence of asthma. After controlling for covariates, obese
women had an 85% increase in the risk of asthma compared with those of normal weight (Table 2). Obese men had an increase of approximately 20% compared with those of a normal weight (Table 2). The multiplicative interaction of obesity with sex was statistically significant \( p = 0.012 \), indicating that the association between obesity and asthma prevalence was stronger in women than in men.

Table 3 shows the prevalence of asthma according to BMI stratified by sex and history of allergy. In both men and women who were allergic or not, the prevalence of asthma was higher in the obese groups than in the normal-weight groups. The difference was most striking for nonallergic women, with an asthma risk of 9.5% for obese women compared to 3.1% for women of normal weight.

After adjustment for covariates in Table 4, the odds ratio (OR) for asthma associated with obesity was 2.53 (95% confidence interval [CI], 2.11 to 3.04) for nonallergic women and 1.57 (95% CI, 1.36 to 1.82) for allergic women. For men, the corresponding ORs were 1.30 (95% CI, 1.05 to 1.62) and 1.18 (95% CI, 0.98 to 1.53), respectively. The obesity-
BMI (1 kg/m²) was associated with an approximately 6% increase in asthma risk in women, and 3% in men. The increased risk of asthma associated with BMI was more pronounced in nonallergic people than in allergic people, especially among women (Table 5).

**Discussion**

In this study of > 80,000 Canadians, obesity showed a stronger association with asthma risk in women than in men, which is consistent with previous observations in Canada, and the modifying effect of sex was statistically significant. The point estimate for the obesity-asthma association in men was similar compared with those of two previous studies, but it was significant in this study because of its large sample size. We estimated that one unit of increased BMI was associated with an approximate 6% increase in asthma risk in women, and 3% in men.

Cross-sectional, case-control, and cohort studies of other populations have shown a similar sex specificity for the association of obesity and asthma. In a British study of 8,960 adults who were 26 years of age, the prevalence of asthma increased with increasing BMI, and the association was stronger in women. In a nested case-control study from the longitudinal cohort study in Tucson, AZ, obesity (ie, BMI, ≥ 28 kg/m²) increased the risk of asthma (OR, 2.10; 95% CI, 1.31 to 3.36), and the association between elevated BMI and new asthma was significant only among women. The adjusted ORs for overweight and obese women vs those with a BMI of < 25 kg/m² were 1.51 (95% CI, 1.11 to 2.06) and 1.84 (95% CI, 1.19 to 2.84), respectively. In men, the corresponding ORs were 1.13 (95% CI, 0.82 to 1.56) and 1.43 (95% CI, 0.82 to 2.50), respectively. A US follow-up study of 4,547 young adults demonstrated that a gain in BMI was associated with new asthma diagnosis, and when stratified by sex the association was only significant in women. The sex specificity for the obesity-asthma association was observed when waist circumference was used as an indicator of obesity. In a recent study of 135 Hispanic men and 398 women, Del-Rio-Navarro et al found that asthma symptoms were associated with higher levels of both waist circumference and BMI in women. In men, none of the anthropometric measures were related to asthma symptoms.

However, the gender specificity was not observed in a recent Norwegian cohort study. Compared with persons with a BMI of < 25 kg/m², overweight men and women had relative risks of asthma of 1.27 (95% CI, 1.13 to 1.43) and 1.30 (95% CI, 1.17 to 1.45), respectively, and obese men and women had relative risks of 1.78 (95% CI, 1.35 to 2.34) and 1.99 (95% CI, 1.67 to 2.37), respectively. The reasons for this discrepancy concerning the sex specificity are not known. The authors pointed out that it was not a usual cohort design but rather combined a number of national surveys that were conducted during the period between 1963 and 1999. These surveys included the information on height and weight, and BMI was calculated at baseline. Health surveys conducted between 1994 and 2002 provided information on asthma, and the analysis was based on data from 135,000 adults who had information on both baseline BMI and asthma. This heterogeneous cohort had a baseline BMI measured between the ages of 14 and 59 years, and during different time periods, with the participants being followed up over a wide age span from 0 to 39 years. The mean time interval from the measurement of BMI to the onset of asthma was 15 years with no information on changes in BMI. BMI is sex-dependent and age-dependent, and equivalent values represent different levels of adiposity in different sex and age groups.

The present study further demonstrated that the association between increased BMI and asthma was stronger among nonallergy adults compared with allergy adults. If one assumes that those without a history of allergy are less likely to have allergic asthma, our observations suggest that obesity has a stronger relation with nonallergic than allergic asthma. The former type of asthma is more likely to be adult-onset, which is more common in women, is more severe, and yields negative allergy skin-prick

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**Table 5—Unadjusted and Adjusted ORs and 95% CIs for Asthma Associated With 1 Unit Change in BMI by History of Allergy in the CCHS 2000–2001**

<table>
<thead>
<tr>
<th>BMI</th>
<th>Unadjusted OR (95% CI)</th>
<th>Adjusted* OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allergy (−)</td>
<td>1.034 (1.016–1.052)</td>
<td>1.035 (1.017–1.053)</td>
</tr>
<tr>
<td>Allergy (+)</td>
<td>1.023 (1.008–1.039)</td>
<td>1.024 (1.009–1.040)</td>
</tr>
<tr>
<td>Total</td>
<td>1.028 (1.016–1.039)</td>
<td>1.027 (1.015–1.039)</td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allergy (−)</td>
<td>1.071 (1.058–1.084)</td>
<td>1.071 (1.058–1.085)</td>
</tr>
<tr>
<td>Allergy (+)</td>
<td>1.038 (1.028–1.048)</td>
<td>1.040 (1.030–1.050)</td>
</tr>
<tr>
<td>Total</td>
<td>1.053 (1.045–1.060)</td>
<td>1.063 (1.037–1.089)</td>
</tr>
</tbody>
</table>

*− = no allergy; + = allergy.
†Adjusted for age, income, educational level, immigrant status, marital status, smoking status, alcohol use and exercise.
test results. In addition, this association of nonallergic asthma and obesity was stronger in women than in men. One unit of increased BMI was associated with a 7.1% increase in asthma risk in nonallergic women and a 4.0% increase in allergic women. The increases were 3.4% in nonallergic men and 2.4% in allergic men. These findings are consistent with and help to explain several previous observations. First, nonallergic asthma is more common in women, and there is a stronger relationship between obesity and asthma in women. Second, asthma in children is usually allergic, and the evidence for a positive association between obesity and asthma is much weaker and less consistent in children. Finally, there is a lack of association between obesity and atopy in adults.

Are there possible or plausible biological explanations for an enhanced effect of obesity on nonallergic vs allergic asthma? Truncal obesity reduces chest wall compliance, loads the inspiratory muscles, increases the work of breathing, and predisposes the individual to breathlessness that may increase the probability of asthma being diagnosed. Weight loss is associated with improved symptoms and ventilatory function but not with obvious improvement in airways reactivity. If there were a subgroup of symptomatic individuals who were overweight and did not have asthma but who had erroneously received a diagnosis of asthma, they would be less likely to have “allergic asthma” and therefore could show a stronger association of BMI and asthma than those persons with true asthma and allergic mechanisms. However, this mechanism would not explain the gender modification of the obesity and asthma association. Compared to nonobese mice, ovalbumin-specific IgE levels were lower, but there was a greater tendency to antigen-induced T-cell responses and γ-interferon production, suggesting a less pronounced allergic reaction but an increased T-cell-mediated mechanism of asthma. Perhaps there exists a human parallel with obesity and asthma that is less mediated by allergic mechanisms. In support of this, Mai et al have provided evidence that γ-interferon may be a mediator in “leptin-induced inflammation,” which has been implicated in asthma among overweight children.

The effect of obesity on female hormone levels may also be related to our findings. Obesity may reduce progesterone, which would reduce β₂-receptor function, which may reduce bronchial smooth muscle relaxation and worsen asthma control. Weight loss increases progesterone and adrenoreceptor density. If this mechanism of asthma has more influence than allergic mechanisms in a subgroup of obese women, then weight reduction would be expected to have a stronger effect in this group than one in which allergic mechanisms dominate. A female hormonal influence would also be consistent with the greater effect of BMI in women with asthma.

There are several limitations in this study. Because it is a cross-sectional study, the data did not provide direct information on whether obesity preceded the development of asthma, which has been discussed in a previous study. However, several longitudinal studies, including one Canadian study, have already indicated that this is the case. Bias in the reporting of asthma is always a concern in large-scale epidemiologic studies and is likely to be different between men and women. An analysis of 16,171 American men and women combined found that the highest BMI quintile had the greatest risk of self-reported asthma, bronchodilator use, and dyspnea with exertion, but had the lowest risk for significant airflow obstruction, and the authors suggested that there could be an overreporting of asthma in obese subjects. However, there is no strong evidence that obesity leads to a markedly greater overdiagnosis of asthma in women than in men. A measurement of airway responsiveness is desirable but not practical in studies like ours. The modifying effect of allergy does not support self-reporting bias as an explanation for the association of obesity and asthma. We may expect a larger reporting bias of asthma (if it exists) associated with obesity in allergic women than in nonallergic women, but not vice versa. We did not have objective measures for allergic status; however, a possible misclassification is likely to be nondifferential with regard to obesity. BMI assessed on the basis of objectively measured height and weight and waist circumference have shown a similar sex difference in the association between obesity and asthma, suggesting that self-reporting bias does not explain the sex specificity of the obesity effect, and the association was robust to the anthropometric measures.

In summary, our study demonstrated that obesity associated with asthma was modified by sex and history of allergy. A stronger association was found in women than in men, and in those with no allergy history than in those with allergy history. Our study suggested that obesity may have a greater effect on nonallergic asthma than on allergic asthma. These findings are consistent with and may help to explain the observed differences in associations of obesity and asthma between children and adults, and between men and women.

References

1. 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; 159:2552–2558
16 Gunnbjornsdottir MI, Omenaaas E, Gislason T, et al. Obesity and nocturnal gastro-oesophageal reflux are related to onset of asthma and respiratory symptoms. Eur Respir J 2004; 24:116–121
17 Weiss ST, Shore S. Obesity and asthma: directions for research. Am J Respir Crit Care Med 2004; 169:963–968
23 Chen Y. Obesity and asthma in children. J Pediatr 2004; 144:146–147
29 Mai XM, Bottcher MF, Leijon I. Leptin and asthma in overweight children at 12 years of age. Pediat Allergy Immunol 2004; 15:523–530
31 Wilson MM, Irwin RS. The association of asthma and obesity: is it real or a matter of definition, Presbyterian minister’s salaries, and earlobe creases? Arch Intern Med 1999; 159:2513–2514
33 Sin DD, Jones RL, Man SF. Obesity is a risk factor for dyspnea but not for airflow obstruction. Arch Intern Med 2002; 162:1477–1481
34 Chen Y, Rennie D, Cormier Y, et al. Sex specificity of asthma and wheeze but not airway hyperresponsiveness. Thorax 2001; 56:4–8
38 Chen Y. Obesity and asthma in children. J Pediatr 2004; 144:146–147
44 Mai XM, Bottcher MF, Leijon I. Leptin and asthma in overweight children at 12 years of age. Pediat Allergy Immunol 2004; 15:523–530
46 Wilson MM, Irwin RS. The association of asthma and obesity: is it real or a matter of definition, Presbyterian minister’s salaries, and earlobe creases? Arch Intern Med 1999; 159:2513–2514