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Body mass index and asthma incidence in the Black Women's Health Study

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Abstract

Background—Evidence from prospective studies consistently links obesity to asthma onset in white women, although there is controversy as to whether the association is causal. There are few data on this topic in black women, among whom the prevalence of obesity and asthma is high.

Objective—We prospectively assessed the relation of body mass index (BMI) to asthma incidence in the Black Women's Health Study.

Methods—We followed 46,435 women from 1995 through 2005 with biennial mailed questionnaires. Cox regression models were used to estimate incidence rate ratios and 95% CIs.

Results—During 403,394 person-years of follow-up, 1068 participants reported physician-diagnosed asthma and concurrent use of asthma medication. Compared with women with BMIs of 20 to 24, the multivariate incidence rate ratios for higher categories of BMI increased from 1.26 (95% CI, 1.05–1.51) for BMIs of 25 to 29 to 2.85 (95% CI, 2.19–3.72) for BMIs of 40 or greater, with a significant trend. The association of BMI with asthma risk was consistent across strata of smoking status, age, presence of sleep apnea, parental history of asthma, BMI at age 18 years, and energy expenditure and intake.

Conclusion—In this large cohort of African American women, there was a positive association between BMI and asthma risk that was similar in magnitude to those observed in longitudinal studies of white women. (*J Allergy Clin Immunol* 2009;123:89–95.)

Keywords

Asthma; body mass index; cohort studies; African Americans; incidence; longitudinal studies

Asthma has reached epidemic proportions in the United States. According to National Health Interview Survey data from 2001–2004, African American subjects fared worse than white subjects on most measures of asthma, including a higher prevalence of current asthma (9.2% in African American subjects and 6.9% in white subjects), more asthma-related emergency department visits (195.0 emergency department visits/10,000 population in African American subjects compared with 43.6 visits/10,000 in white subjects), more hospitalizations (33.5 hospitalizations/10,000 population in African American subjects compared with 10.0 hospitalizations/10,000 in white subjects), and more deaths from asthma (3.4 deaths/10,000

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population in African American subjects compared with 1.9/10,000 in white subjects).¹ Women fared worse than men on these measures.

Although studies have implicated current smoking, socioeconomic status, household allergens, and childhood or prenatal exposures as risk factors for adult-onset asthma,^{2,3} the evidence is most consistent for obesity. In 8 prospective studies obesity was associated with increased risk of adult-onset asthma in women, with relative risks ranging from 1.6 to 3.0.^{4–11} Previous studies have included relatively few black women, and only 1 study reported separately on black women.¹² In that study, which was cross-sectional, obesity was not associated with prevalent asthma.¹² There is still controversy as to whether obesity causes asthma or whether obesity and asthma are common consequences of factors, such as genetics, *in utero* exposures, diet, or sedentariness.^{13,14}

We present here a prospective analysis of obesity and asthma incidence in the Black Women's Health Study (BWHS), a large cohort study of African American women who have been followed for more than 10 years. Our aim was to test the hypothesis that body mass index (BMI) was positively associated with the incidence of adult-onset asthma, as has been observed in white women. It is important to assess this hypothesis among African American women because they have high prevalences of obesity and severe obesity.¹⁵ A causal connection between obesity and asthma in this population would have important public health consequences.

METHODS

Study population

The BWHS is a prospective cohort study established in 1995, when approximately 59,000 African American women aged 21 through 69 years were recruited mainly from subscribers to *Essence* magazine.¹⁶ The baseline questionnaire elicited information on demographic and lifestyle factors, reproductive history, height and weight, dietary intake, and medical conditions. The cohort is followed biennially by using mailed questionnaires. All the women in the cohort that we followed self-identified as "black," and 95.1% were born in the United States; 1.8% reported Hispanic ancestry. Follow-up has averaged more than 80% of the original cohort through 5 questionnaire cycles. The study protocol was approved by the Institutional Review Board of Boston University, and participants indicated their consent by completing and returning the questionnaire or questionnaires. At baseline, the median age was 38 years. Participants were from across the US: Northeast, 28%; South, 30%; Midwest, 23%, and West, 19%. Ninety-seven percent of the cohort completed high school or a higher level of education, with 36% having some college, and 44% having completed college.

This analysis uses data from the baseline questionnaire and 5 subsequent follow-up cycles covering the time period 1995 through 2005. Of 58,605 women who completed the baseline questionnaire and returned at least 1 follow-up questionnaire, we excluded women who reported asthma diagnosed before 1996 ($n = 5621$), reported bariatric surgery ($n = 145$), were pregnant at baseline ($n = 912$), had lung cancer at baseline ($n = 31$), had cancer of any kind over the follow-up period ($n = 1570$), or were missing information on BMI or smoking status at baseline ($n = 1765$).

Diagnosis of asthma

Asthma diagnosis was based on questions about physician-diagnosed asthma that were asked on all questionnaires from 1997 through 2005. In the 1997 questionnaire, respondents were asked whether a physician had told them that they had asthma and whether the asthma was diagnosed before the baseline questionnaire in 1995 or in the 2 years since then. On

questionnaires from 1999 through 2003 participants were asked whether a physician had told them they had asthma in the previous 2-year interval and, if yes, the year in which they had first been diagnosed. In 2005, they were asked whether they had ever been given a diagnosis of asthma and, if so, in what year. If a participant reported different years of diagnosis on 2 separate questionnaires, we used the year of diagnosis given on the first questionnaire on which asthma was reported. On questionnaires from 1997 through 2005, respondents were asked whether they used “inhalers or pills” for asthma at least 3 days per week. Cases were defined as women who reported an initial physician’s diagnosis of asthma after 1995 through 2005 and who reported concurrent use of asthma medication. We excluded women who reported physician-diagnosed asthma but did not use asthma medication ($n = 1036$) or reported asthma medication use before their year of diagnosis ($n = 104$) and women who did not report asthma diagnosis but did report use of asthma medication ($n = 986$). After exclusions, 46,435 women remained, of whom 1068 met the case criteria.

Assessment of BMI and covariates

Data on weight and height were obtained in 1995, and weight was updated on each subsequent follow-up questionnaire. In a validation study conducted among 110 participants, the mean self-reported weight was 176 pounds and the mean technician-measured weight was 181 pounds. For height, the self-reported and technician-measured values were 64.4 inches and 64.0 inches, respectively. The Spearman correlation coefficients between self-reported and measured weight and height at baseline were, respectively, 0.97 ($P < .001$) and 0.93 ($P < .001$). Weight was updated on all subsequent questionnaires, and BMI was calculated for each questionnaire cycle as weight in kilograms/height in square meters. We set the value of BMI to “missing” for women who were pregnant at the time of completion of follow-up questionnaires. We imputed missing BMI by carrying the previous value forward. The proportions of subjects with missing BMI (caused by pregnancy or failure to answer the weight question) on follow-up questionnaires were as follows: 1997, 13%; 1999, 11%; 2001, 17%; and 2003, 12%.

Data on smoking history were first obtained in 1995. Ever smokers were asked about the age they had started smoking, the number of cigarettes smoked, and the total number of years they had smoked. Former smokers were asked how many years ago they had quit. Smoking status and amount smoked were updated on each follow-up questionnaire. Pack-years of smoking were calculated by multiplying the duration of smoking in years by the average number of packs of cigarettes smoked per day. We imputed missing number of cigarettes smoked per day by carrying the previous value forward. In 1997, respondents were asked about exposure to secondhand tobacco smoke at ages 0 to 10, 11 to 20, 21 to 30, and 31 to 40 years and current exposure at home and the office (for ages ≥ 21 years), and exposure was defined as being “in the same room with a smoker for at least an hour a day for 12 consecutive months or more.”

Weight at age 18 years, marital status, household size, and years of education were queried in 1995. Residential location in 1995 was geocoded, and the median housing value for the block group of residence was obtained from the US Census as a measure of neighborhood socioeconomic status. In 1997, women were asked whether their mother or father had ever had asthma, whether they had a regular physician and health insurance, and whether they lived in an urban, suburban, rural, or other setting until age 18 years. In 2001, participants were asked whether they had ever been given a diagnosis of sleep apnea.

Alcohol consumption, menopausal status, and use of hormone replacement therapy were ascertained in 1995 and updated every questionnaire cycle. Physical activity was ascertained in 1995, and subsequent cycles were ascertained by asking about hours per week in the past year spent walking and in vigorous exercise. We calculated energy expenditure in terms of average metabolic equivalents (METs) expended per hour as follows:

$$\begin{aligned} & (\text{Vigorous activity in hours per week} \times 7 \text{ METs}) \\ & + (\text{Walking in hours per week} \times 3.5 \text{ METs}). \end{aligned}$$

Energy expenditure was updated every questionnaire cycle.

The 1995 questionnaire included a 68-item modification of the short Block–National Cancer Institute food frequency questionnaire that assessed the consumption of specified foods during the previous year.¹⁷ This questionnaire was validated in 400 cohort members against 3 nonconsecutive 24-hour recalls and a 3-day food diary.¹⁸

Statistical analysis

Each participant contributed person-time from baseline in 1995 until the diagnosis of asthma, death, loss to follow-up, or end of follow-up, whichever came first. We used Cox proportional hazards models to estimate incidence rate ratios (IRRs) and 95% CIs for incident asthma in various categories of BMI compared with BMIs of 20 to 24. We calculated IRRs for variables representing change in weight from age 18 years and change in weight from baseline in 1995. To control for age and calendar time, we adjusted all analyses by age in 1-year intervals at the start of follow-up and the calendar year of the questionnaire cycle. The Anderson-Gill data structure was used to update BMI, change in BMI since 1995, change in BMI since age 18 years, and other time-varying covariates.¹⁹ In the Andersen-Gill data structure, a new data record is created for every questionnaire cycle in which a participant was at risk, with covariates set to their values at the time the questionnaire was completed. For exposures that vary over time (eg, BMI), cases and person-time are reassigned every 2 years according to the value reported on the biennial questionnaires completed before the questionnaire on which the outcome was reported. Thus BMI represented BMIs calculated from weight reported on the questionnaire before the questionnaire cycle during which the asthma occurred or the questionnaire before the last follow-up (for noncases).

We present results from a basic model that accounted only for age and calendar time and a multivariate model that also included variables that we *a priori* believed might influence an association between BMI and asthma (parental history of asthma, current smoking status, pack-years of smoking, and secondhand smoke exposure at various ages) and variables that were associated with both asthma risk and BMI in our data (menopausal status, marital status, income, energy expenditure in MET-hours per week, median housing value in the census block of the participant residence, use of hormone replacement therapy, energy intake, and BMI at age 18 years). Missing values were modeled as indicator variables. The addition of the following variables did not change the final multivariate results: alcohol consumption; region of the country of residence; household size; median age of housing in the census block group of the participant's residence; education; living primarily in an urban, rural, or suburban setting until age 18 years; presence of sleep apnea; and having a regular physician and health insurance.

We tested for trend by including BMI, change in weight since 1995, and change in weight since age 18 years as continuous variables in multivariate models. We assessed interaction with BMI by using the likelihood ratio test to compare models with and without cross-product terms for BMI and age, smoking status, parental history of asthma, presence of sleep apnea, energy expenditure, energy intake, and BMI at age 18 years. We stratified by these variables to assess confounding and effect modification. Two-sided *P* values are reported. We conducted several sensitivity analyses: we excluded asthma cases diagnosed in the first 4 years of follow-up (1996–1999) in case subclinical asthma preceded obesity onset in these early cases. In an effort to identify more severe cases of asthma, we restricted the analysis to the 60% of cases (*n* = 642) who reported use of a specific preventive asthma medication (eg, fluticasone) in addition

to regular use of “pills or inhalers for asthma.” To evaluate the effect of imputing missing BMI, we conducted an analysis confined to women with complete BMI data. Analyses were conducted with SAS statistical software version 9.1 (SAS Institute, Inc, Cary, NC).

RESULTS

The cohort included in this analysis comprised 46,435 women who were followed up for an average of 9.3 years, for a total of 403,394 person-years of follow-up. At baseline, 31.8% of the women were overweight (BMI, 25–29), 23.2% were obese (BMI, 30–39), and 5.2% were severely obese (BMI, ≥ 40 ; Table I). BMI was positively associated with age, BMI at age 18 years, energy intake, postmenopausal status, sleep apnea, parental history of asthma, being a past smoker, and exposure to secondhand smoke and inversely associated with energy expenditure, current smoking, income, median housing value of the neighborhood of residence, and being married. BMI was not associated with having a regular physician (Table I).

As shown in Table II, BMI was associated with asthma risk in multivariate analyses in which we adjusted for age, time period, smoking, secondhand exposure to smoking, parental history of asthma, menopausal status, marital status, income, physical activity, median housing value of the census block, menopausal female hormone use, BMI at age 18 years, and energy intake. Relative to BMIs of 20 to 24, the multivariate IRR increased from 1.26 (95% CI, 1.05–1.51) for overweight women (BMI, 25–29) to 2.85 (95% CI, 2.19–3.72) for severely obese women (BMI, ≥ 40 ; P value for trend $< .0001$; Table II). The IRR was lower among never smokers compared with ever or current smokers (IRRs for BMI ≥ 40 , 2.70, 3.19, and 3.44, respectively), but there was no statistically significant interaction by smoking status (Table II). The trends in IRRs across BMI increments were significant in smoking subgroups ($P < .0001$ for all groups).

The association of BMI with asthma was present within strata of age (< 40 years, ≥ 40 years), presence of sleep apnea, parental history of asthma, energy expenditure (less than median, median or greater), energy intake (less than median, median or greater), and BMI at age 18 years (BMI, < 25 , BMI, ≥ 25 ; Table III). There was no statistically significant interaction between BMI and any of the variables.

Change in weight since age 18 years was associated with asthma, with IRRs of 1.29 (95% CI, 1.02–1.63), 1.35 (95% CI, 1.07–1.71), and 1.88 (95% CI, 1.54–2.28) for weight gain of 15 to 19 kg, 20 to 24 kg, and 25 kg or more, respectively, with a significant trend ($P < .0001$, Table IV). Change in weight since baseline in 1995 was significantly associated with asthma only for women who gained at least 15 kg (IRR, 1.51; 95% CI, 1.12–2.05; $P < .0001$; Table IV).

We estimated IRRs, excluding cases diagnosed in the first 4 years of follow-up, and the IRRs were similar to the models including all subjects (eg, IRR for BMI ≥ 40 , 2.94; 95% CI, 2.06–4.20). When we used a less stringent case definition and included all patients who reported physician-diagnosed asthma regardless of medication use, the IRRs were slightly attenuated for each BMI category: IRRs for BMIs of 25 to 29, 1.12 (95% CI, 0.99–1.27); 30 to 35, 1.39 (95% CI, 1.21–1.60); 35–39, 1.69 (95% CI, 1.44–2.00), and 40 or greater, 2.41 (95% CI, 2.04–2.84). When we used a more strict case definition and confined the analysis to those patients who reported use of a specific asthma preventative (eg, fluticasone), the IRRs for the various categories of obesity (IRR for BMI ≥ 40 , 2.77; 95% CI, 1.97–3.90) were similar to those from the main analysis that included all patients who reported any asthma medication use. When the analysis was confined to participants with complete data on BMI, the IRRs were similar to those from the analysis including women with imputed BMI: IRRs for BMIs of 25 to 29, 1.26 (95% CI, 1.04–1.52); 30 to 35, 1.54 (95% CI, 1.25–1.91); 35 to 39, 2.01 (95% CI, 1.56–2.58); and 40 or greater, 2.50 (95% CI, 1.90–3.29).

DISCUSSION

This is the first study to examine the relation of obesity to asthma incidence in a large cohort of black women. The risk of asthma increased as the level of BMI increased, with severely obese women estimated to have an incidence almost 3-fold greater than that of women with BMIs of 20 to 24. The slight but nonsignificant lower risk among very thin women (BMI, <20) is compatible with the idea that there is a dose-response relation between BMI and asthma with no threshold. Change in weight since age 18 years was positively associated with asthma risk, with significant increases seen for weight gains of 15 kg or more. Gains of 15 kg or more since 1995 were also associated with asthma risk. The association of BMI and asthma was seen among all strata of smoking, age, energy expenditure and intake, presence of sleep apnea or parental history of asthma, and BMI at age 18 years. The IRRs for BMI categories were little changed when we restricted cases to those diagnosed at least 4 years after the start of follow-up and those patients who used a preventive medication for asthma, suggesting that preclinical asthma did not predate obesity and that overdiagnosis of less severe asthma in the obese does not explain the increased risk.

Most previous studies have included few black participants. The Coronary Artery Risk Development in Young Adults (CARDIA) study included 1286 black women⁴ but did not report on them separately. The cross-sectional Behavioral Risk Factor Surveillance System Study (BRFSS) is the only study that reported separately on black women.¹² There was no association between obesity and prevalent asthma among the 2598 black women studied, whereas there was a 2-fold increase in risk among obese white and Hispanic women. BRFSS participants were younger (mean age, 25.6 years) than BWHS participants, but this does not explain the lack of association because an obesity-asthma association was observed in the BWHS among both the younger and older women. The small number of black women included in the BRFSS might have limited the power of the study to detect an association.

Our results are consistent with most previous prospective studies of obesity and adult asthma, 4–11,20 8 of which found a positive association in women.^{4–11} The studies identified cases using self-reported physician diagnosis alone^{6,7,9–11} or a combination of self-reported physician diagnosis and asthma medication use.^{4,5} Four studies used self-reported height and weight,^{4–6,11} and 3 used measured height and weight.^{8–10} Follow-up time ranged from 3 years¹¹ to 20 years.^{9,10} A 2-fold increase in asthma risk has been consistently reported in previous cohorts^{4–11}; estimates range from 1.6 in the highest quintile of BMI in the CARDIA study⁴ to 3.0 in Nurses' Health Study participants with BMIs of 30 or greater compared with women with BMIs of 20 to 22.4.⁵ A meta-analysis of prospective studies reported a summary odds of incident asthma of 1.92 for obese compared with normal-weight subjects.²¹

Our study relied on self-reported weight and height. A validation study indicated that BWHS participants report height and weight with a high degree of accuracy. Our study also relied on self-report of asthma. To increase specificity, we included use of an asthma medication in the case definition. There is no gold standard for defining asthma.^{22,23} Testing of airway hyperresponsiveness has good specificity but low sensitivity,²² and clinical diagnosis can be inconsistent.¹³ In 2 population-based studies wherein self-report of physician-diagnosed asthma was compared with airway hyperresponsiveness test results, the sensitivities were 47% and 52%, and the specificities were 91% and 92%.²³ As noted, most prospective studies have used self-reported physician-diagnosed asthma to identify cases because in large national studies it is not feasible to clinically examine all patients to verify that they have asthma and impossible to examine all nonpatients to identify undiagnosed asthma. Self-reported physician-diagnosed asthma has been recommended as the preferred outcome assessment for use in large population-based studies.²⁴

There is the possibility that chronic obstructive pulmonary disease might have been misdiagnosed by the physician or misreported by the participant as asthma. This is not likely to have occurred among never smokers or younger women, in whom the prevalence of chronic obstructive pulmonary disease is low.²⁵ In our data the association between obesity and asthma was apparent both in never smokers and younger women.

Detection bias is of concern because breathlessness and dyspnea, symptoms similar to asthma, occur more often in obese than nonobese persons. A study that assessed the prevalence of undiagnosed asthma by conducting lung function tests among Australian adults with and without self-reported physician-diagnosed asthma found that the prevalence of obesity among persons with diagnosed asthma was 34.7% compared with 26.8% among persons with undiagnosed asthma.²⁶ Although these results suggest that there might be detection bias, the relevance of these results to BWHS participants is questionable. The Australian sample was from an area of high social disadvantage and chronic disease. In contrast, BWHS participants have a relatively high level of education and high use of medical care.²⁷ In the Nurses' Health Study the clinical characteristics of asthma were similar among thin and obese patients,⁵ which might suggest lack of detection bias. In our data, when we confined the cases to women who reported use of a preventive asthma medication, which might indicate more severe asthma, the relation between BMI and asthma risk was unchanged. Furthermore, the IRR was increased even for the overweight category (BMI, 25–29). However, we acknowledge that we cannot directly assess whether detection bias played a role in our results.

The BWHS cohort is not a probability sample. The participants come from all regions of the country and represent a wide range of occupations. However, they are more highly educated than the population of all US black women. For example, according to the 2000 US Census, 17% of US black women of the same ages as the BWHS cohort have not completed high school compared with 2% of BWHS participants.²⁸ Thus our results might not be generalizable to black women who have not finished high school.

Both direct and indirect mechanisms have been suggested to explain the association of obesity with asthma risk. Obese persons have reduced functional residual capacity²⁹ and tidal volume,³⁰ which might reduce airway caliber,¹⁴ and there is evidence that some of the proteins synthesized by adipose tissue (eg, TNF- α and leptin) might adversely affect airway function.¹⁴ In addition, in some studies, when asthmatic subjects lose weight, their symptoms improve.^{7,13,31} Alternately, it has been suggested that aspects of physical activity or diet that contribute to obesity might be the true causes of asthma in obese persons. Physical activity did not have an independent effect on asthma risk in our data, nor was it related to asthma onset in the Nurses' Health Study⁵ or in the CARDIA study.⁴ In our data energy intake did have an independent association with asthma risk. However, inclusion of energy intake and expenditure in the model did not alter the association between BMI and asthma. Thus these 2 variables, as far as we could measure them, did not confound the association between obesity and asthma, nor did they modify the association because IRRs were similar among strata of energy intake and expenditure.

An increased prevalence of both sleep apnea^{14,32} and gastroesophageal reflux disease (GERD) has been observed among asthmatic subjects^{14,31,32}; some have suggested that the conditions might confound the association between asthma and obesity, and others suggest that they might be steps in the causal chain.¹³ Sleep apnea and GERD can exacerbate asthma,³³ but they are not established as causes of asthma.^{33,34} It is equally plausible that sleep apnea, GERD, and asthma are common consequences of obesity, and although the 3 might interact to some degree, their relationship is not causal. Sleep apnea did not confound or modify the association that we observed between obesity and asthma. We lacked data on GERD.

In conclusion, our data demonstrate a positive association between BMI and asthma risk in black women that is similar in its magnitude to that observed in prior longitudinal studies of white women. The association has great public health importance given the high and increasing prevalence of obesity and severe obesity in African American women.

Key messages

- In this large cohort of African American women, there was a positive association between BMI and the risk of adult-onset asthma.
- The association between BMI and asthma risk was observed in all strata of age, smoking status, energy expenditure and intake, presence of sleep apnea, and parental history of asthma.

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Abbreviations

BMI	Body mass index
BRFSS	Behavioral Risk Factor Surveillance System Study
CARDIA	Coronary Artery Risk Development in Young Adults
BWHS	Black Women's Health Study
GERD	Gastroesophageal reflux disease
IRR	Incidence rate ratio
MET	Metabolic equivalent

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TABLE 1
 Characteristics of 46,435 BWHHS participants according to BMI in 1995*

	BMI					
	<20	20–24	25–29	30–34	35–39	≥40
No. (%) of women	2715 (5.8)	15,747 (33.9)	14,767 (31.8)	7483 (16.1)	3304 (7.1)	2419 (5.2)
Sociodemographic characteristics						
Age (y), mean	32.3	36.5	40.3	40.9	40.0	39.1
Income						
≤\$25,000	11.2%	7.5%	8.5%	11.2%	12.3%	14.6%
\$25,001–50,000	22.3%	21.1%	24.0%	26.2%	27.0%	26.6%
\$50,001–100,000	26.2%	30.4%	29.4%	26.4%	26.2%	24.2%
≥\$100,000	13.2%	15.8%	12.0%	9.2%	7.2%	5.6%
Missing	27.1%	25.3%	26.0%	27.0%	27.4%	28.7%
Median housing value of census block group of residence						
<\$72,383	15.3%	15.3%	18.4%	22.1%	22.0%	24.8%
\$72,384–100,850	16.3%	16.7%	19.4%	19.4%	19.6%	19.9%
\$100,851–140,804	16.9%	18.7%	18.8%	17.5%	18.2%	20.0%
\$140,805–190,016	20.8%	19.5%	18.1%	17.0%	17.4%	15.8%
≥\$190,017	22.6%	21.6%	17.2%	15.5%	14.2%	13.3%
Missing	8.1%	8.3%	8.1%	8.5%	8.5%	9.2%
Marital status						
Married/living as married	34.4%	39.4%	41.8%	39.0%	36.0%	32.8%
Divorced/widowed/separated	23.9%	24.9%	24.9%	25.2%	24.0%	25.2%
Never married	40.9%	34.6%	32.4%	35.1%	39.1%	40.8%
Smoking-related characteristics						
Pack-years of smoking, mean	12.2	11.4	11.9	12.3	11.8	12.0
Current smoker	19.3%	13.7%	14.8%	14.5%	13.8%	11.2%
Past smoker	14.0%	17.8%	20.0%	20.8%	21.1%	22.5%
Never smoker	66.7%	68.5%	65.2%	64.7%	65.1%	66.2%
Exposure to secondhand smoke						
Age 0–20 y	49.8%	50.8%	53.3%	53.2%	54.3%	56.4%
Age 21–30 y	41.8%	43.0%	47.3%	48.9%	49.7%	49.4%

	BMI					
	<20	20–24	25–29	30–34	35–39	≥40
Age 31–40 y						
Currently	27.6%	27.6%	31.2%	31.7%	32.3%	33.0%
Medical history						
BMI at age 18 y, mean	17.9	19.7	21.2	23.1	25.2	28.5
Postmenopausal	23.2%	23.2%	25.2%	26.1%	26.4%	25.2%
Presence of sleep apnea	0.2%	0.7%	1.6%	2.4%	3.5%	5.8%
Parental history of asthma	5.6%	5.9%	5.4%	6.4%	6.6%	6.0%
Had regular physician [†]	77.3%	78.5%	78.9%	78.8%	78.8%	77.5%
Female hormone use	20.6%	21.3%	22.3%	22.4%	21.8	20.8%
Energy-related characteristics						
Energy intake (kcal/d), mean	1539	1474	1554	1659	1723	1849
Energy expenditure (MET·h/wk), mean	20.5	24.2	21.7	18.3	16.0	13.7

* All characteristics, with the exception of age, are age adjusted in 5-year intervals.

[†] Queried in 1997 about the previous 2-year period.

TABLE II
Incidence of asthma in relation to BMI among all subjects and by smoking status,
BWHs 1995–2005

BMI	Person-years	Cases	Age-adjusted IRR (95% CI)	Multivariate IRR* (95% CI)
All subjects				
<20	16,195	27	0.92 (0.61–1.37)	0.84 (0.56–1.26)
20–24	117,495	212	1.0	1.0
25–29	133,160	312	1.26 (1.06–1.50)	1.26 (1.05–1.51)
30–34	75,338	228	1.64 (1.36–1.98)	1.62 (1.32–1.98)
35–39	34,388	143	2.26 (1.82–2.79)	2.24 (1.76–2.84)
≥40	26,098	146	3.09 (2.50–3.82)	2.85 (2.19–3.72)
	Test for trend			<i>P</i> < .0001
Never smokers				
<20	12,429	16	0.79 (0.47–1.33)	0.74 (0.44–1.26)
20–24	83,481	140	1.0	1.0
25–29	85,751	186	1.27 (1.02–1.59)	1.26 (1.01–1.58)
30–34	47,867	125	1.54 (1.21–1.96)	1.49 (1.14–1.94)
35–39	22,008	92	2.46 (1.89–3.21)	2.33 (1.72–3.16)
≥40	17,146	88	3.08 (2.36–4.03)	2.70 (1.92–3.80)
	Test for trend			<i>P</i> < .0001
Ever smokers				
<20	4,486	11	1.19 (0.63–2.26)	1.03 (0.54–1.97)
20–24	34,015	72	1.0	1.0
25–29	47,403	126	1.26 (0.94–1.68)	1.27 (0.94–1.70)
30–34	27,471	103	1.80 (1.33–2.44)	1.85 (1.34–2.56)
35–39	12,381	51	1.93 (1.34–2.78)	2.09 (1.41–3.10)
≥40	8,952	58	3.20 (2.26–4.54)	3.19 (2.09–4.88)
	Test for trend			<i>P</i> < .0001
Current smokers				
<20	2,375	9	1.55 (0.74–3.25)	1.35 (0.63–2.88)
20–24	16,104	34	1.0	1.0
25–29	19,468	56	1.39 (0.90–2.13)	1.53 (0.98–2.37)
30–34	10,542	38	1.82 (1.14–2.90)	2.15 (1.30–3.55)
35–39	4,592	21	2.28 (1.32–3.94)	2.71 (1.46–5.01)
≥40	2,873	19	3.29 (1.86–5.82)	3.44 (1.72–6.89)
	Test for trend			<i>P</i> < .0001

* Adjusted for age (single years); time period; current smoker (yes, no); pack-years of smoking (0, 1–4, 5–9, 10–14, 15–24, 25–39, ≥40); secondhand smoke exposure at ages 0 to 20 years (never, age 0–10 years only, age 11–20 years only, age 0–20 years, missing), 21 to 30 years, 31 to 40 years, and currently (no exposure, at home only, at work only, at home and work, missing); parental history of asthma (no, yes, unknown); menopausal status (pre, peri, post, unknown); marital status (married, separated/widowed, divorced, single); income (<\$15,000, \$15,001–25,000, \$25,001–35,000, \$35,001–50,000, \$50,001–100,000, ≥\$100,001, missing); METs expended in vigorous activity or walking in MET-hours per week (none, 0–4, 5–9, 10–19, 20–39, ≥40, missing); median housing value of residential census block in quintiles; menopausal female hormone use (ever, never); BMI at age 18 years (<18.5, 18.5–19.9, 20.0–22.9, 23.0–25.9, 26.0–28.9, ≥29); and energy intake (kcal/day) in quintiles.

TABLE III
Multivariate IRRs (95% CIs)* for asthma among strata of BWHS participants, 1995–2005

Characteristics	BMI					
	<20	20–24	25–29	30–34	35–39	≥40
Age (y)						
<40						
Cases/person-years	17/11,792	107/62,311	105/50,988	68/26,628	51/13,065	45/11,010
IRR	0.73 (0.43–1.23)	1.0	1.26 (0.95–1.67)	1.61 (1.15–2.28)	2.54 (1.70–3.79)	2.55 (1.59–4.06)
≥40						
Cases/person-years	10/5,123	105/55,184	207/82,172	160/48,710	92/21,323	101/15,088
IRR	0.94 (0.49–1.80)	1.0	1.30 (1.02–1.65)	1.69 (1.30–2.19)	2.17 (1.60–2.95)	3.13 (2.26–4.35)
Sleep apnea						
Yes						
Cases/person-years	0/60	3/728	16/1,803	12/1,767	18/1,216	25/1,601
IRR	—	1.0	2.51 (0.65–9.70)	1.56 (0.38–6.44)	3.56 (0.88–14.5)	3.36 (0.77–14.6)
No						
Cases/person-years	22/13,275	178/94,252	245/106,539	172/59,422	97/27,765	94/19,013
IRR	0.82 (0.52–1.28)	1.0	1.20 (0.99–1.47)	1.54 (1.23–1.94)	1.96 (1.48–2.58)	2.62 (1.92–3.57)
Parental history of asthma						
Yes						
Cases/person-years	3/1,024	33/7,173	43/7,568	26/4,755	16/2,259	20/1,667
IRR	0.66 (0.20–2.21)	1.0	1.10 (0.68–1.78)	1.08 (0.61–1.92)	1.40 (0.70–2.78)	2.03 (0.98–4.20)
No						
Cases/person-years	23/13,984	169/98,627	249/113,947	184/64,279	117/29,312	119/22,375
IRR	0.91 (0.58–1.42)	1.0	1.25 (1.02–1.52)	1.65 (1.32–2.07)	2.33 (1.78–3.03)	2.97 (2.22–3.99)
Energy expenditure						
< Median						
Cases/person-years	12/8,094	83/45,343	144/57,480	127/38,243	94/19,637	100/16,623
IRR	0.74 (0.40–1.37)	1.0	1.32 (1.03–1.68)	1.89 (1.40–2.55)	2.76 (2.00–3.86)	3.54 (2.47–5.07)
≥ Median						
Cases/person-years	13/8,359	120/69,124	163/72,025	93/34,980	47/13,917	44/8,816
IRR	0.89 (0.50–1.60)	1.0	1.23 (0.96–1.57)	1.38 (1.03–1.85)	1.74 (1.19–2.52)	2.37 (1.56–3.60)

Characteristics	BMI					
	<20	20–24	25–29	30–34	35–39	≥40
Energy intake						
< Median						
Cases/person-years	10/7,803	112/60,136	147/64,143	89/32,762	49/13,212	37/8,379
IRR	0.69 (0.37–1.29)	1.0	1.32 (1.05–1.68)	1.55 (1.16–2.08)	2.22 (1.55–3.19)	2.81 (1.85–4.25)
≥ Median						
Cases/person-years	15/7,679	92/49,157	138/59,478	126/36,809	80/18,001	93/15,153
IRR	1.00 (0.58–1.71)	1.0	1.21 (0.93–1.58)	1.70 (1.27–2.26)	2.29 (1.65–3.17)	2.86 (2.01–4.07)
BMI at age 18 y						
<25						
Cases/person-years	23/14,480	196/109,276	276/120,928	183/60,589	100/21,132	55/10,023
IRR	0.87 (0.57–1.35)	1.0	1.18 (0.98–1.42)	1.47 (1.20–1.82)	2.22 (1.73–2.85)	2.49 (1.82–3.39)
≥25						
Cases/person-years	0/56	5/2,871	20/8,280	40/13,318	43/12,802	91/15,791
IRR	—	1.0	1.61 (0.60–4.33)	1.91 (0.75–4.91)	2.21 (0.86–5.68)	3.70 (1.47–9.31)

* Adjusted for age (single years); time period; current smoker (yes, no); pack-years of smoking (0, 1–4, 5–9, 10–14, 15–24, 25–39, ≥40); secondhand smoke exposure at ages 0 to 20 years (never, age 0–10 years only, age 11–20 years only, age 21–30 years, missing), 31 to 40 years, and currently (no exposure, at home only, at work only, at home and work, missing); parental history of asthma (no, yes, unknown, except in strata of parental history); menopausal status (pre, peri, post, unknown); marital status (married, separated/widowed, divorced, single); income (<\$15,000, \$15,001–25,000, \$25,001–35,000, \$35,001–50,000, \$50,001–100,000, ≥\$100,001, missing); METs expended in vigorous activity or walking in MET-hours per week (none, 0–4, 5–9, 10–19, 20–39, ≥40, missing, except in strata of energy expenditure); median housing value of residential census block in quintiles; menopausal female hormone use (ever, never); BMI at age 18 years (<18.5, 18.5–19.9, 20.0–22.9, 23.0–25.9, 26.0–28.9, ≥29, except in strata of BMI at age 18 years); and energy intake (kcal/day) in quintiles (except in strata of energy intake).

TABLE IV

Incidence of asthma in relation to change in weight since 1995 (baseline) and change in weight since age 18 years, BWHS 1995–2005

	Person-years	Cases	Multivariate IRR* (95% CI)
Change in weight since age 18 y (kg) [†]			
<0	21,809	47	1.01 (0.72–1.41)
0–9	86,286	149	1.0
10–14	60,079	116	1.07 (0.84–1.37)
15–19	57,573	139	1.29 (1.02–1.63)
20–24	57,575	150	1.35 (1.07–1.71)
≥25	116,910	460	1.88 (1.54–2.28)
	Test for trend		<i>P</i> < .0001
Change in weight since 1995 (kg)			
<0	73,362	188	0.82 (0.65–1.05)
0	131,180	319	1.0
>0–4	98,301	246	0.87 (0.69–1.09)
5–9	56,488	151	0.97 (0.75–1.25)
10–14	25,157	82	1.20 (0.89–1.62)
≥15	18,906	82	1.51 (1.12–2.05)
	Test for trend		<i>P</i> < .0001

* Adjusted for age (single years); time period; current smoker (yes, no); pack-years of smoking (0, 1–4, 5–9, 10–14, 15–24, 25–39, ≥40); secondhand smoke exposure at ages 0 to 20 years (never, age 0–10 years only, age 11–20 years only, age 0–20 years, missing), 21 to 30 years, 31 to 40 years, and currently (no exposure, at home only, at work only, at home and work, missing); parental history of asthma (no, yes, unknown); menopausal status (pre, peri, post, unknown); marital status (married, separated/widowed, divorced, single); income (<\$15,000, \$15,001–25,000, \$25,001–35,000, \$35,001–50,000, \$50,001–100,000, ≥\$100,001, missing); METs expended invigorous activity or walking in MET-hours per week (none, 0–4, 5–9, 10–19, 20–39, ≥40, missing); median housing value of residential census block in quintiles; menopausal female hormone use (ever, never); BMI at age 18 years (<18.5, 18.5–19.9, 20.0–22.9, 23.0–25.9, 26.0–28.9, ≥29); and energy intake (kcal/day) in quintiles.

[†] One thousand five hundred eighty-two participants, including 7 cases, were excluded because of missing value for BMI at age 18 years.