Body mass index and asthma incidence among USA adults

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Body mass index and asthma incidence among USA adults. E.S. Ford, D.M. Mannino, S.C. Redd, A.H. Mokdad, J.A. Mott. ©ERS Journals Ltd 2004.

ABSTRACT: The aim of this study was to examine the association between body mass index (BMI) and asthma incidence.

Data from the baseline examination conducted during 1971–1975, and the first follow-up conducted during 1982–1984, of the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study (a cohort study) was used. Asthma was self-reported or reported by proxies. BMI was calculated from measured height and weight obtained during the baseline examination.

Among 9,456 participants aged 25–74 yrs who were free of asthma at baseline, 317 participants reported a diagnosis of asthma during the follow-up interview. Compared with participants with a BMI of $18.5 < 25.0 \text{ kg}\cdot\text{m}^{-2}$, the odds ratio (OR) for those with a BMI of $\geq 35 \text{ kg}\cdot\text{m}^{-2}$ was 1.87 (95% confidence interval (CI) 1.12–3.13). ORs were similar for males and females. However, only 125 of the 298 participants who recalled a date of onset reported a diagnosis that occurred after their baseline examination. Among this group of participants, BMI was not significantly associated with asthma incidence (OR 1.52, 95% CI 0.62–3.77).

In conclusion, although obese people reported more "incident" asthma during follow-up, it remains unclear whether this represents reactivation of previously diagnosed asthma or the onset of new cases, and whether these new cases actually represent true asthma or respiratory symptoms misdiagnosed as asthma.

Eur Respir J 2004; 24: 740–744.

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Keywords: Asthma, body mass index, cohort studies, obesity

Received: July 29 2003
Accepted after revision: June 2 2004

During 1980–1996, the prevalence of self- or proxy-reported asthma has increased by ~74% [1]. The reasons for this increase are not clear, but the coincident rise in obesity prevalence in the USA may have contributed to the increase in asthma prevalence [2]. As obesity is a potentially modifiable risk factor, its relationship to asthma incidence needs to be clarified. Several lines of evidence suggest that obesity may be causally related to asthma incidence. Numerous cross-sectional studies and case-control studies have reported associations between obesity and asthma. Several prospective studies in adults have shown significant associations between obesity and asthma incidence primarily among females [3–5], as did a prospective study among children [6]. In addition, weight gain resulted in a greater likelihood of developing new asthma symptoms in young females [7]. In a recent nested case-control study, males with a body mass index (BMI) > 29.4 had an increased risk of developing airway hyperresponsiveness [8]. Furthermore, major decreases in the prevalence of asthma occurred in patients who underwent bariatric surgery [9–12]. Finally, weight-loss studies in asthmatic patients showed improvements in lung function, symptoms, morbidity and health status [13, 14]. Nevertheless, the relationship between obesity and asthma remains controversial [15].

As few prospective studies have addressed the relationship between obesity and asthma incidence, this association was examined using prospective data from the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study (NHEFS).

Methods

The study design was a cohort study. Participants aged 25–74 yrs from the first National Health and Nutrition Examination Survey (NHANES I), conducted from 1971–1975, were followed through 1992 or 1993 ($n=14,407$). The original sample was selected using a complex sampling design to ensure that the results would be representative of the noninstitutionalised civilian population. Details of the NHANES I and the NHEFS have been published elsewhere [16–19].

During the baseline interview, participants were asked "Has a doctor ever told you that you have any of the following conditions (asthma) and, if so, do you still have it?". Participants who responded that they still had asthma were considered to be prevalent cases of asthma and were excluded from the analyses. At the first follow-up interview during 1982–1984, participants were asked "Has a doctor ever told you that you had any of the following conditions?" and "In what year were you first told that you had any of these conditions?". Participants who gave an affirmative response to the first question were considered to have "incident" asthma.

The exposure of interest was BMI ($\text{kg}\cdot\text{m}^{-2}$), which was calculated from measured height and weight. BMI was divided into the following categories: $18.5 < 25 \text{ kg}\cdot\text{m}^{-2}$, $25 < 30 \text{ kg}\cdot\text{m}^{-2}$, $30 < 35 \text{ kg}\cdot\text{m}^{-2}$ and $\geq 35 \text{ kg}\cdot\text{m}^{-2}$. For some analyses, the two top categories were combined into $\geq 30 \text{ kg}\cdot\text{m}^{-2}$.

Baseline covariates included: age; race or ethnicity (non-White, White); education (number of yrs); cigarette smoking (never, former, current); recreational exercise (much, moderate, little or no exercise); and nonrecreational exercise (very active, moderately active, quite inactive). These variables were derived from questionnaire responses. For smoking, the current authors used a variable constructed, in part, from responses obtained during the baseline interview and, in part, from the first follow-up interview [20, 21].

The analyses were limited to participants who were alive at the time of the first follow-up during 1982–1984. Participants who reported at baseline ever having been told that they had asthma were considered as prevalent cases and were excluded from the analyses. Pregnant females were excluded. In addition, participants with BMI <18.5 kg·m⁻² were excluded. Two-sample comparisons of categorical and continuous variables were conducted using unpaired t-tests. To examine the independent association between BMI and asthma incidence, multiple logistic regression analysis was used because of the uncertainty associated with the date of onset of asthma. The complex sampling design information of the survey was used to calculate standard errors, and sampling weights were used in the analyses.

Results

Among the 14,407 participants who were followed-up, 11,296 were alive at the time of the first follow-up. Among these participants, 392 (weighted percentage 3.5%) had asthma at the time of the baseline interview, 210 (weighted percentage 2.0%) previously had the condition but did not have it at the time of interview, 26 (weighted percentage 0.3%) had the condition but were unsure whether they still had it at the time of the interview, and 10,663 had never had asthma (five provided no answer). Among the 10,663 participants without baseline asthma, 9,895 provided a response to the follow-up asthma questions, of whom 337 answered whether they had ever been told that they had asthma, and 9,558 did not report having been told that they had asthma or they did not have a hospitalisation for asthma. After excluding pregnant females, participants with BMI <18.5 kg·m⁻² and participants who had missing data for any of the covariates, 317 participants with incident asthma and 9,139 participants who had never had asthma were included in the analyses. Six proxies completed the questionnaire for the 317 participants with "incident" asthma. Of the 298 participants with new asthma who provided a year of onset ranging from 1906–1983, 125 had a year of onset after the year in which they received their baseline examination, and 173 reported a year of onset that either antedated their year of examination or that occurred in the same year as their examination.

None of the baseline characteristics shown in table 1 differed significantly between participants who developed asthma during the follow-up period and those who remained free of asthma. However, three characteristics were of borderline significance. Compared with participants who did not develop asthma, those who developed asthma were more likely to be female, to be a current smoker and to have a larger BMI.

In multiple logistic regression analyses, participants with a baseline BMI of ≥ 35 kg·m⁻² were more likely to develop asthma than those with BMI 18.5–<25 kg·m⁻² (table 2). BMI, when used as a continuous variable in a fully adjusted model, was significantly associated with asthma (odds ratio (OR) per kg·m⁻² 1.03, 95% confidence interval (CI) 1.00–1.05, $p=0.039$). ORs for males and females were similar ($p=0.435$ for the interaction term between sex and BMI). The current authors also examined whether the age composition of the analytic sample affected the results. For the age-specific analyses, three categories of BMI were created: 18.5–<25 kg·m⁻², 25–<30 kg·m⁻² and ≥ 30 kg·m⁻². Among 3,190 participants aged ≥ 55 yrs (112 developed asthma), those with BMI ≥ 30 kg·m⁻² were more likely to develop asthma than those with BMI 18.5–<25 kg·m⁻² (OR 2.86, 95% CI 1.45–5.64, $p=0.003$). In contrast, among 6,266 participants aged <55 yrs (205 developed asthma), those with BMI ≥ 30 kg·m⁻² were no more likely to develop asthma than those with BMI 18.5–<25 kg·m⁻² (OR 1.17, 95% CI 0.73–1.87, $p=0.521$). The interaction term for age and BMI was of borderline significance (Wald Chi-squared test, $p=0.089$).

Separate logistic regression analyses were also performed for those participants who reported an onset of their asthma after the year of their baseline examination and those participants who reported an onset of their asthma in or before the year of their examination ("reactivators"). The two groups differed only in age and educational status (table 3). Compared to their counterparts with BMI 18.5–<25 kg·m⁻², participants with BMI ≥ 35 kg·m⁻² who reported an onset of their asthma after the year of their baseline examination were more likely than participants whose onset was in or before the year of their baseline examination to report at their follow-up interview having ever been told that they had asthma (table 4). However, the CI of the ORs for the two groups showed considerable overlap.

Discussion

In this large prospective study of a representative sample of the USA population, increased BMI (especially ≥ 35 kg·m⁻²) was significantly associated with the risk of incident asthma over a period of ~10 yrs. In contrast to previous cohort

Table 1.—Selected baseline characteristics by incident asthma status, National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study, 1971–1975 to 1982–1984

Baseline characteristics	Incident asthma [#]	No incident asthma [†]	p-value
Age yrs	46.3 (0.9)	45.5 (0.2)	0.924
Males %	41.3 (3.1)	47.4 (0.8)	0.072
Whites %	90.1 (2.2)	90.6 (0.7)	0.838
Education yrs	11.7 (0.3)	11.7 (0.1)	0.881
Current smoker %	44.6 (3.2)	38.5 (0.8)	0.073
Active, recreational activity %	18.2 (2.5)	21.2 (0.7)	0.210
Active, nonrecreational activity %	41.1 (3.6)	46.4 (0.9)	0.132
BMI kg·m ⁻²	26.5 (0.4)	25.8 (0.1)	0.066

Data are presented as mean (SE) and % (SE), unless otherwise stated. BMI: body mass index. [#]: n=317; [†]: n=9,139.

Table 2.—Associations between body mass index (BMI) class and incidence of self-reported asthma, National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study, 1971–1975 to 1982–1984

	BMI class				Wald Chi-squared p-value
	18.5–<25 kg·m ⁻²	25–<30 kg·m ⁻²	30–<35 kg·m ⁻²	≥ 35 kg·m ⁻²	
Total [#]					
Incidence n [†]	145/4672 (3.1)	102/3202 (2.8)	45/1121 (3.7)	25/461 (6.0)	
Unadjusted	1.00	0.91 (0.67–1.26)	1.22 (0.79–1.87)	2.02 (1.22–3.33)	0.013
Multiple adjusted ⁺	1.00	0.95 (0.68–1.33)	1.28 (0.83–1.96)	1.87 (1.12–3.13)	0.033
Males [§]					
Incidence	41/1531 (2.8)	42/1618 (2.3)	14/383 (4.2)	5/89 (5.9)	
Unadjusted	1.00	0.80 (0.47–1.33)	1.52 (0.82–2.84)	2.15 (0.71–6.55)	0.070
Multiple adjusted ⁺	1.00	0.79 (0.47–1.34)	1.52 (0.82–2.82)	1.98 (0.60–6.54)	0.096
Females [‡]					
Incidence	104/3141 (3.3)	60/1584 (3.7)	31/738 (3.3)	20/372 (6.1)	
Unadjusted	1.00	1.14 (0.77–1.68)	1.02 (0.59–1.75)	1.93 (1.08–3.43)	0.149
Multiple adjusted ⁺	1.00	1.13 (0.73–1.75)	1.10 (0.64–1.89)	1.87 (1.05–3.33)	0.187

Data are presented as n/n (%) and odds ratios (95% confidence interval), unless otherwise stated. [#]: 317/9,456; [†]: calculated using sampling weights; ⁺: adjusted for age, sex (except sex-specific models), race or ethnicity, education, smoking status, recreational physical activity and nonrecreational activity; [§]: 102/3,621; [‡]: 215/5,835.

Table 3.—Selected characteristics by asthma status, National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study, 1971–1975 to 1982–1984

	Reactivators [#]	Incident asthma [†]	p-value
Age yrs	44.5 (1.1)	48.4 (1.3)	0.020
Males %	46.1 (5.1)	34.7 (5.9)	0.198
Whites %	90.3 (2.5)	89.3 (3.1)	0.747
Education yrs	12.2 (0.3)	11.0 (0.4)	0.012
Current smoker %	43.0 (4.7)	44.0 (5.5)	0.899
Active, recreational activity %	21.6 (3.3)	13.5 (4.0)	0.096
Active, nonrecreational activity %	37.8 (4.7)	47.5 (6.2)	0.236
BMI kg·m ⁻²	26.6 (0.6)	26.4 (0.6)	0.810

Data are presented as mean (SE) or % (SE), unless otherwise stated. BMI: body mass index. [#]: n=173; [†]: n=125.

Table 4.—Associations between body mass index (BMI) class and incidence of self-reported asthma, National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study, 1971–1975 to 1982–1984

	BMI class				Wald Chi-squared p-value
	18.5–<25 kg·m ⁻²	25–<30 kg·m ⁻²	30–<35 kg·m ⁻²	≥ 35 kg·m ⁻²	
Incidence [#]					
Unadjusted	1.00	1.05 (0.61–1.81)	1.23 (0.61–2.48)	1.89 (0.78–4.57)	0.487
Multiple-adjusted	1.00	1.10 (0.60–2.00)	1.19 (0.58–2.43)	1.52 (0.62–3.77)	0.803
Reactivators [†]					
Unadjusted	1.00	0.86 (0.58–1.27)	1.07 (0.62–1.83)	2.07 (1.04–4.10)	0.062
Multiple-adjusted	1.00	0.88 (0.58–1.33)	1.17 (0.68–2.01)	2.08 (0.99–4.37)	0.111

Data are presented as odds ratios (95% confidence interval), unless otherwise stated. [#]: 125/9,264; [†]: 173/9,312.

studies, the strength of the association between BMI and asthma incidence was similar in males and females.

Whether obesity causes asthma remains controversial [15]. Some studies have reported that obesity is associated with respiratory symptoms, such as wheezing and dyspnoea, but not with bronchial hyperresponsiveness or airflow obstruction, suggesting that asthma may be overdiagnosed in obese people [22, 23]. However, among 11,277 participants of the European Community Respiratory Health Survey, bronchial hyperresponsiveness was significantly associated with BMI [24]. Nevertheless, whether obesity is a primary risk factor for

asthma, whether it exacerbates asthma, or causes wheezing and dyspnoea, obesity remains an important source of respiratory morbidity.

Several cohort studies have examined the association of BMI with asthma incidence in adults. Out of 85,911 participants (aged 26–46 yrs) of the Nurses' Health Study who were followed for 4 yrs, 1,596 developed asthma [3]. A continuous relationship was shown between BMI and asthma incidence. In the Coronary Artery Risk Development in Young Adults study, 4,547 males and females aged 18–30 yrs were followed for 10 yrs, and 416 of them developed asthma

[4]. BMI was associated with asthma among females only. During a 2-yr follow-up period of 9,149 participants aged 20–64 yrs of the National Population Health Survey in Canada, baseline BMI was associated with self-reported asthma incidence among females, but not males [5]. In that study, 49 males and 127 females developed asthma.

The NHEFS differs from the previous prospective studies in several aspects. First, the study was conducted earlier than the other studies, before the prevalence of obesity became a national concern. CHINN and RONA [6] concluded that the association between obesity and asthma was of recent origin. During the baseline examination of the NHEFS, the prevalence of asthma in the USA was lower than that during later decades [1]. The baseline prevalence of asthma in the NHEFS cohort was ~3.5% and the lowest of several cohort studies. The baseline prevalence of asthma was ~6% in the Nurses' Health Study [3], ranged from 7.0–12.6% in the four sites of the Coronary Artery Risk Development in Young Adults Study [4], and was ~10% among adolescents and 5% among adults in the Canadian National Population Health Surveys [5]. Secondly, the cumulative incidence was the lowest of the four studies. Thirdly, the study population was somewhat older than those of the other studies. These differences and other methodological differences may account for differences among studies.

A number of mechanisms have been proposed to explain a possible association between obesity and asthma incidence [3]. Summarising a number of studies, CAMARGO *et al.* [3] suggested that obesity causes histological changes in the lungs of obese rats, reduction in airway calibre and increased bronchial hyperreactivity. Furthermore, they suggested that obese people may be more sedentary; thus, they spend more time indoors with continued exposure to potential allergens, they are more likely to have gastro-oesophageal reflux that has been associated with asthma, and they may have diets that are more likely to lead to asthma. Finally, they speculate that effects of obesity on hormonal patterns could be related to the risk of developing asthma. Obesity affects immune function [25, 26]. As the immune system is critically involved in the pathophysiology of asthma, some of these immune alterations may be linked to asthma. In addition, obesity may be associated with a state of low-grade inflammation [27] that may contribute to airway inflammation [28].

It is unclear why the majority of participants who developed "incident" asthma reported a year of onset that occurred in or before the year of their examination. The diagnosis of asthma rests upon the clinical impression of a physician, along with demonstration of airway reversibility. Furthermore, unlike many other conditions, asthma may wax and wane and, thus, people may not experience an asthma exacerbation for a considerable time, if not exposed to triggers. Participants who experienced an asthma episode during the follow-up period may have recalled episodes during previous times in their lives that they had forgotten. Another possibility is that physicians, who the participants consulted during the follow-up period for asthmatic symptoms, may have pinpointed a time in their patients' lives when, perhaps, they first had asthma after taking a medical history. Finally, participants may have had trouble recalling accurately the date of onset of their asthma. In a recent study from Norway, estimates of asthma incidence, (calculated separately from a prospective and a retrospective study, conducted in the same location and time period among participants born in the same years) differed greatly, suggesting that participants recalled poorly the age of onset [29].

The current study has several positive attributes: measured height and weight to calculate BMI, a long follow-up period, and a sample representative of the USA noninstitutionalised

civilian population. The use of self-reported asthma was the most significant limitation of this study. Previous studies have suggested, however, that self-reported asthma has acceptable validity and reliability [30–32]. Nevertheless, the current data could be interpreted as suggesting that people have some trouble in accurately reporting facts about asthma. In addition, questions to determine smoking status were administered to approximately half of the sample at baseline. However, the use of data from the follow-up interview to augment that from the baseline interview, in order to establish smoking status, has been shown to work well. The possibility that people who are obese are seen more often by healthcare providers, and, thus, have more opportunities to have asthma diagnosed, cannot be ruled out.

The current authors' prospective study provides modest support for the proposition that obesity may predict the development of asthma. Nevertheless, a clear understanding of the role of excess weight in the pathophysiology of asthma remains elusive. As excess weight is highly prevalent in the USA and increasing in other parts of the world, and because it is potentially preventable, it is imperative to bring clarity into this area. If excess weight were indeed causally related to asthma, this would have important ramifications for the prevention and treatment of asthma.

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