

Obesity and asthma in 11–12 year old New Zealand children in 1989 and 2000

K Wickens, D Barry, A Friezema, R Rhodius, N Bone, G Purdie, J Crane

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See end of article for authors' affiliations

Correspondence to: K Wickens, Wellington Asthma Research Group, Wellington School of Medicine and Health Sciences, P O Box 7343, Wellington South, New Zealand; kwickens@wnmeds.ac.nz

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Background: There has been a concurrent increase in the prevalence of obesity and asthma in recent years in New Zealand and other countries.

Methods: Two cross sectional surveys performed in 1989 and 2000 were used to test this association in children of mean age 11.7 years. Body mass index (BMI) was calculated as weight/height² (kg/m²) and obesity and overweight defined according to an international standard. Standard questions were used to measure the prevalence of asthma symptoms.

Results: Significant increases in the prevalence of reported symptoms and disease between 1989 and 2000 were not explained by a concurrent increase in the prevalence of obesity. In 2000, multivariate analysis showed that increasing BMI standard deviation score was significantly associated with current wheeze ($p=0.002$), inhaled steroid use ($p=0.004$), and the use of any medication ($p=0.001$). None of the associations was significantly different for boys or girls.

Conclusion: There is some evidence for an association of obesity with asthma symptoms and treatment but this does not explain the increasing prevalence of this disease.

There is convincing evidence that body mass index (BMI) has increased in the last 10–30 years in developed countries,¹ including New Zealand.^{2,3} A rise in the prevalence of asthma in these countries seems to have occurred over the same time period.^{4,5} BMI has been associated with asthma symptoms and severity in a large number of studies of adults^{6–9} and children,^{10–14} although in some populations the association is limited to girls^{11,13,14} and women.^{6,7,15} Although sex interaction terms were seldom reported in these studies, the possibility of a sex specific effect needs further investigation. Evidence that the obesity/asthma association may be causal is provided by two small randomised controlled trials of obese asthmatic adults where weight reduction was accompanied by an improvement in lung function, symptom scores, and peak flow variability.^{16,17} Thus, while BMI and asthma symptom reporting have both increased over time and occur together more often than would be expected by chance, few studies¹⁸ have been able to show how much of the increase in asthma symptoms over time is due to increases in BMI during the same time period. The present study investigates changing asthma prevalence and its association with obesity among 11–12 year old boys and girls between 1989 and 2000 in a New Zealand population.

METHODS

Cross sectional prevalence studies of children attending schools in the City of Hastings and in the borough of Havelock North were conducted in 1989 and 2000. All children in the 1989 survey turned 12 in the survey year (mean age 11.7 years). In 2000 the study population comprised a broader age range of children (mean age 11.4 years) but, for analysis comparing study years this data set was restricted to children in their 12th year (mean age 11.7). The principals of schools qualifying for the study distributed a parental/caregiver questionnaire and consent form. The questionnaires collected information on the child's ethnicity (Maori, European, Pacific Island group, other) and gender. In 2000, standard International Study of Asthma and Allergies in Childhood (ISAAC) questions were used to

collect information on the prevalence of asthma symptoms.¹⁹ At times these differed slightly from the questions used in 1989. In 1989, having a history of wheeze was defined as a positive response to the question "Has a wheeze—that is, a whistling noise (high or low pitched)—ever been heard coming from the child's chest?" The equivalent question in 2000 was "Has your child ever had wheezing or whistling in the chest at any time in the past?" In 1989 current wheeze was defined as a response of once or more to "How many times has it occurred in the last 12 months?" In 2000 current wheeze was defined as a positive response to the question "Has your child had wheezing or whistling in the chest in the last 12 months?" In 1989 respondents were asked "Has your child ever suffered from any of the following?" with a positive response to "asthma" defining this condition. The equivalent question in 2000 was "Has your child ever had asthma?" The same questions were used in 1989 and 2000 to define wheezing on exercise, hospital admissions, and medication use.

Following parental consent, eligible children underwent an exercise challenge indoors at school, as previously outlined,⁴ to test for airway hyperresponsiveness. Identical methods and the same equipment were used for measuring weight and height of children in each year. Height was measured to the nearest 0.1 cm using a portable field instrument that has been shown to be accurate by comparison with a standard rule. The child stands on a flat base, heels and back against an upright pylon. The movable part of the pylon surrounds the fixed part on three sides, has a fixed head piece and a pointer at its lowest border. Children were weighed in light clothing on calibrated step scales and weight was recorded to the nearest kilogram. Each child ran briskly for 6 minutes, radial pulse rate was recorded at rest and immediately after the run. Five peak flow rate (PEFR) measurements were made before and 5 minutes after exercise using the same standard (clockface) Wright PEFR meters in both studies. The change in peak flow after exercise was calculated as the mean of the highest three post-exercise peak flow measurements as a percentage of the mean of the highest three pre-exercise peak flow measurements. Exercise induced

bronchospasm was defined as a fall in peak flow of more than 15%. If children had received short acting β_2 agonists within 6 hours of the exercise challenge they were exercised, and if their PEFR decreased by less than 15% from the baseline they were re-exercised on another occasion without prior β_2 agonist exposure.

To ensure comparability between survey years, the study commenced in February in each year, the exercise challenge procedure was undertaken by the same nurse (NB) in both years, and the PEFR measurements were supervised by the same investigator (DB). Temperature and humidity were measured using a whirling hygrometer just before each set of exercise challenges.

The only difference between years was that skin prick testing to common environmental allergens was performed before the exercise challenge in 2000. This was undertaken as part of the ISAAC phase II protocol using ALK allergens (ALK Allergologisk Laboratorium A/s, Horsholm, Denmark) (*Dermatophagoides pteronyssinus*, *Dermatophagoides farinae*, *Felis domesticus*, *Alternaria*, mixed grasses, mixed trees) and positive and negative controls. A positive reaction was defined as a mean wheal diameter of 3 mm or greater to an allergen with atopy defined as any positive reaction.

Ethical approval was obtained from the Hawkes Bay ethics committee.

Statistical analysis

Data analysis was conducted using SAS version 8 (SAS Institute Inc, Cary, NC, USA). Only observations with complete height and weight data were included in the analysis. Missing data were treated identically in both survey years. A small number (<6) of non-responders to the health outcome questions were classified with those responding negatively, as previously in ISAAC. For all other variables, respondents with missing values were excluded from the analysis. Inhaled steroid and cromoglycate use in the last 12 months were combined as one variable. BMI was calculated as weight/height² (kg/m²). The definition of obesity and overweight in this population of children was based on a standard international definition which used pooled international BMI data and is linked to the widely

used adult overweight (25 kg/m²) and obesity (30 kg/m²) definitions.²⁰ There is no similar agreed international definition of underweight. Using New Zealand BMI curves,²¹ we extrapolated from a BMI of 18.5 at age 18 to determine a BMI cut off of 15.2 in boys and 15.1 in girls, below which children in this study were considered underweight.

The standard deviation score (SDS) for BMI was calculated adjusting for age for boys and girls separately using the combined years. Logistic regression analysis was used to determine the effects of study year on the prevalence of the dependent variables (prevalence and history of wheeze, asthma, hospital admissions, bronchial hyperresponsiveness (BHR) and asthma medication use) before and after adjustment for BMI (obese, overweight, underweight versus normal weight), ethnicity and sex. Within year associations of obesity, overweight and underweight with the outcome variables were also examined using logistic regression analysis. For year 2000 the full dataset (n = 1287) was used to adjust for the potential confounding effects of family history of allergic disease, family size, birth weight, current smoking in the home, father's years of education, frequency of exercise and hamburger consumption, as well as ethnicity, sex and year of birth. After the exclusion of respondents with missing data for family size, birth weight, ethnicity, hamburger consumption, and exercise frequency there were 1237 respondents included in these models. p values are reported for the null hypothesis of no trend with BMI SDS. Differences in the association of BMI SDS with the outcome variables between years and between the sexes were tested using interaction terms. The criteria for concluding significance were adjusted to take into account multiple outcome variables using the Holm's method.²² Unadjusted p values are reported. Odds ratios and confidence intervals for comparisons between the normal BMI and other categories are given.

RESULTS

In 1989, 12 of the 13 school principals in the study area agreed to their school participating in the research and, in 2000, all school principals agreed to participate. The prevalence questionnaire response rate was 94% (n = 873)

Table 1 Prevalence % (n) of study characteristics by year of survey

	1989 (N = 871)	2000* (N = 894)	2000† (N = 1287)
Body mass index			
Obese	2.6 (23)	8.7 (78)	8.1 (104)
Overweight	11.9 (104)	22.5 (201)	21.0 (270)
Normal weight	76.3 (665)	66.6 (595)	68.1 (877)
Underweight	9.1 (79)	2.2 (20)	2.8 (36)
Male sex	52.1 (454)	49.7 (444)	50.2 (645)
Mean (range) age (years)	11.7 (11.1–12.2)	11.7 (11.1–12.3)	11.5 (10.1–12.6)
Ethnicity			
Maori	25.8 (225)	30.8 (275)	30.6 (394)
European	66.8 (582)	62.8 (561)	62.3 (802)
Pacific Island	3.1 (27)	4.5 (40)	4.8 (62)
Other	1.0 (9)	1.5 (13)	1.6 (20)
Missing	3.2 (28)	0.5 (5)	0.7 (9)
Wheeze ever	26.2 (228)	44.7 (400)	44.1 (567)
Wheeze in last 12 months	17.7 (154)	23.3 (208)	22.0 (283)
Wheeze with exercise	14.8 (129)	23.8 (213)	22.8 (293)
Asthma ever	16.9 (147)	37.0 (331)	35.7 (460)
Hospital admission ever	8.5 (74)	13.4 (120)	14.0 (180)
Inhaled steroid or cromoglycate in last 12 months	5.5 (48)	16.1 (144)	15.5 (200)
Any medication in last 12 months	12.7 (111)	25.2 (225)	24.3 (313)
BHR to exercise	12.3 (107/868)	9.0 (80/892)	8.4 (108/1284)
Atopy	–	37.5 (334/891)	34.7 (444/1281)

BHR, bronchial hyperresponsiveness.

*2000 data restricted to children in their 12th year.

†Unrestricted 2000 dataset.

Table 2 Unadjusted and adjusted OR (95% CI) showing associations for study year and underweight, overweight, and obesity versus normal weight in the combined dataset

	Unadjusted	Adjusted†	Body mass index				p value for trend with BMI SDS
	Year	Year	Underweight	Normal	Overweight	Obese	
	2000† v 1989	2000† v 1989					
Wheeze ever	2.28 (1.87 to 2.79)**	2.29 (1.85 to 2.83)**	1.01 (0.63 to 1.62)	1.00	1.25 (0.96 to 1.64)	1.26 (0.82 to 1.93)	0.03
Wheeze last 12 months	1.41 (1.12 to 1.78)**	1.33 (1.04 to 1.70)	0.77 (0.42 to 1.38)	1.00	1.36 (1.00 to 1.84)	1.69 (1.06 to 2.69)	0.002*
Wheeze with exercise	1.80 (1.41 to 2.29)**	1.72 (1.34 to 2.22)**	0.81 (0.44 to 1.50)	1.00	1.22 (0.89 to 1.67)	1.76 (1.11 to 2.80)	0.003*
Asthma ever	2.90 (2.32 to 3.62)**	2.88 (2.28 to 3.64)**	1.17 (0.69 to 1.96)	1.00	1.08 (0.81 to 1.44)	1.39 (0.90 to 2.17)	0.08
Hospital admission ever	1.66 (1.22 to 2.25)**	1.61 (1.17 to 2.23)	1.08 (0.50 to 2.31)	1.00	1.08 (0.72 to 1.60)	1.18 (0.66 to 2.12)	0.98
Inhaled steroid or cromoglycate in last 12 months	3.29 (2.34 to 4.63)**	3.20 (2.24 to 4.68)**	1.33 (0.64 to 2.78)	1.00	1.23 (0.83 to 1.83)	2.33 (1.38 to 3.94)	0.002*
Any medication in last 12 months	2.30 (1.79 to 2.96)**	2.20 (1.70 to 2.85)**	0.95 (0.52 to 1.72)	1.00	1.08 (0.79 to 1.50)	1.82 (1.15 to 2.89)	0.005*
BHR to exercise	0.70 (0.52 to 0.95)*	0.71 (0.51 to 0.97)	1.28 (0.69 to 2.39)	1.00	1.21 (0.81 to 1.83)	0.93 (0.45 to 1.93)	0.90

*Meets the Holm's criterion for significance (adjusting for eight comparisons) at the 5% level.²²

**Meets the Holm's criterion for significance (adjusting for eight comparisons) at the 1% level.²²

†2000 data restricted to children in their 12th year.

‡Adjusted for body mass index (obese, overweight, underweight v normal), ethnicity (Maori, Polynesian, other v European) and sex.

in 1989 and 84% in the full (n = 1321) 2000 dataset. Among the children who returned questionnaires, height and weight were measured for 99.8% (n = 871) in 1989, 97.4% in the full 2000 dataset (n = 1287), and 97.3% in the 2000 dataset restricted to children in their 12th year (n = 894). Of these, 99.7% (n = 868) in 1989, 99.8% in the full (n = 1284) and restricted (n = 892) 2000 datasets completed the exercise challenge. Skin prick tests were completed on 99.5% (n = 1281) of the full and 99.7% (n = 891) of the restricted 2000 datasets.

Table 1 shows that, between 1989 and 2000, the prevalence of overweight children had doubled, the prevalence of obese children had tripled, and the prevalence of underweight children was a quarter of the 1989 prevalence. Corresponding to this, between 1989 and 2000 there was a doubling in the prevalence of a history of wheeze, asthma and any asthma medication use in the past 12 months, three times the use of inhaled steroid or cromoglycate in the last 12 months, and smaller but substantial increases in current wheeze and hospital admissions. In contrast, there was a decline in the prevalence of a positive exercise challenge, with boys accounting for most of this decline (data not shown).

The odds of having a history of diagnosed asthma or symptoms or using medication in 2000 compared with 1989 were significantly increased and were not explained by changes in the prevalence of obesity, ethnicity and sex over this time period (table 2). In this dataset, where both years are combined, there were significant trends in BMI SDS for the prevalence of symptoms and medication use.

In 1989 there was little evidence that BMI SDS was associated with any outcome (table 3). By 2000 there were significant trends in BMI SDS for all symptom and disease outcomes, inhaled steroid (including cromoglycate) use, and any medication use. Subgroup analyses according to sex showed that these trends remained significant for girls only. However, after adjustment for multiple comparisons, none of the associations with BMI SDS was significantly different for boys or girls in either year. There were also no significant between year differences in associations of BMI SDS with any outcome.

Table 4 shows the year 2000 associations of BMI groups with each outcome adjusted for potential confounders (mostly available for year 2000 only). The strength of the BMI group associations and significance of BMI SDS trend with current wheeze, inhaled steroid (including cromoglycate) use, and any medication use were similar to the univariate associations. However, after adjustment for confounders, the significance of increasing BMI SDS for having a

history of wheeze or asthma and wheeze with exercise reduced. The associations were not significantly different for boys or girls. Nevertheless, there were significant trends in increasing BMI SDS with increases in asthma disease and symptom reporting and the use of inhaled steroids and other medication in girls only.

DISCUSSION

The results show clearly that there have been large increases in the prevalence of reported asthma symptoms between 1989 and 2000 in New Zealand, reflected also in increased medication use for asthma. There was a parallel increase in BMI during the same time period. However, since there was little change in the associations between survey year and outcomes after adjusting for BMI, we conclude that increases in BMI are unlikely to explain the increase in asthma symptoms. A similar conclusion was reached in another study investigating whether an increase in BMI contributed to the rise in asthma in British children between 1982 and 1994.¹⁸

Although the associations of BMI with asthma symptoms in 2000 were stronger than in 1989, these differences were not significant. Thus, this study does not provide evidence of a change in these associations over time. In contrast, a recent review by Chinn *et al*²³ concludes that, in children at least, the association between obesity and asthma is recent. In support of this they cite earlier studies where obesity has not been shown to be associated with asthma. For example, in a large cohort of British children born in 1958 Peckham *et al*²⁴ showed that, at age 11, children with frequent current asthma attacks and/or wheezy bronchitis had lower mean relative weight. A later analysis of this cohort,²⁵ which distinguished between asthmatic and wheezy bronchitic children, showed that although there was no significant association of BMI with asthma at age 7, wheezy bronchitic children had a higher BMI than non-wheezy bronchitic children at age 7 and than asthmatics at age 11. In 1977 a second study²⁶ in England and Scotland in children aged 5–11 showed a higher prevalence of the symptom "chest ever wheezy" among overweight children. The authors of the 1958 cohort study²⁵ report that definitions of asthma changed as the cohort aged. The definition of asthma has continued to evolve and, although it is no longer fashionable to collect data on "wheezy bronchitis", "wheeze" is a central characteristic of current definitions. Thus, part of the problem in understanding the association between obesity and asthma is due to poor and changing definitions of the disease, making it

Table 3 Univariate OR (95% CI) showing associations for underweight, overweight, and obesity versus normal weight in 1989 and 2000 among all children, girls and boys†

Outcome variables	All		Girls		Boys	
	1989	2000†	1989	2000†	1989	2000†
	Total (n=871)	Total (n=894)	Total (n=417)	Total (n=449)	Total (n=454)	Total (n=444)
Wheeze ever						
Underweight	1.00 (0.58 to 1.70)	0.71 (0.28 to 1.81)	1.01 (0.45 to 2.31)	0.33 (0.07 to 1.53)	1.04 (0.51 to 2.13)	1.80 (0.42 to 7.67)
Normal	1.00	1.00	1.00	1.00	1.00	1.00
Overweight	1.30 (0.83 to 2.05)	1.26 (0.91 to 1.74)	1.36 (0.69 to 2.70)	1.50 (0.96 to 2.35)	1.34 (0.73 to 2.46)	1.08 (0.68 to 1.72)
Obese	1.28 (0.52 to 3.17)	1.32 (0.82 to 2.12)	1.40 (0.37 to 5.31)	1.81 (0.95 to 3.42)	1.28 (0.37 to 4.48)	0.96 (0.47 to 1.95)
p value‡	0.30	0.006*	0.67	0.0005**	0.34	0.87
Wheeze last 12 months						
Underweight	0.96 (0.51 to 1.80)	0.19 (0.03 to 1.45)	1.16 (0.46 to 2.94)	0.34 (0.04 to 2.71)	0.87 (0.37 to 2.05)	§
Normal	1.00	1.00	1.00	1.00	1.00	1.00
Overweight	1.47 (0.89 to 2.42)	1.31 (0.90 to 1.89)	1.77 (0.85 to 3.73)	1.21 (0.72 to 2.05)	1.32 (0.67 to 2.60)	1.42 (0.84 to 2.40)
Obese	1.03 (0.34 to 3.08)	1.82 (1.09 to 3.03)	1.36 (0.29 to 6.41)	1.58 (0.78 to 3.21)	0.86 (0.18 to 4.05)	2.19 (1.04 to 4.59)
p value‡	0.22	0.002*	0.44	0.013*	0.36	0.08
Wheeze with exercise						
Underweight	1.05 (0.55 to 2.01)	0.19 (0.02 to 1.39)	0.69 (0.24 to 2.04)	§	1.45 (0.63 to 3.23)	0.49 (0.06 to 4.02)
Normal	1.00	1.00	1.00	1.00	1.00	1.00
Overweight	0.99 (0.55 to 1.78)	1.29 (0.89 to 1.86)	1.49 (0.70 to 3.19)	1.28 (0.77 to 2.14)	0.59 (0.23 to 1.55)	1.30 (0.77 to 2.20)
Obese	1.63 (0.59 to 4.48)	1.75 (1.05 to 2.92)	1.28 (0.27 to 6.05)	1.69 (0.84 to 3.38)	2.04 (0.52 to 7.93)	1.86 (0.87 to 3.94)
p value‡	0.89	0.0006**	0.47	0.004*	0.61	0.05
Asthma ever						
Underweight	1.03 (0.56 to 1.90)	1.01 (0.40 to 2.57)	0.82 (0.31 to 2.20)	0.49 (0.11 to 2.28)	1.26 (0.57 to 2.78)	2.47 (0.58 to 10.54)
Normal	1.00	1.00	1.00	1.00	1.00	1.00
Overweight	0.74 (0.41 to 1.35)	1.27 (0.91 to 1.76)	0.90 (0.38 to 2.12)	1.52 (0.95 to 2.42)	0.65 (0.28 to 1.49)	1.10 (0.69 to 1.76)
Obese	1.01 (0.34 to 3.02)	1.61 (1.00 to 2.58)	1.18 (0.25 to 5.57)	2.45 (1.29 to 4.66)	0.90 (0.19 to 4.27)	1.04 (0.51 to 2.13)
p value‡	0.73	0.004*	0.82	0.0001**	0.50	1.00
Hospital admission ever						
Underweight	0.40 (0.12 to 1.33)	2.50 (0.88 to 7.09)	0.31 (0.04 to 2.37)	4.11 (1.17 to 14.46)	0.49 (0.11 to 2.11)	0.99 (0.12 to 8.23)
Normal	1.00	1.00	1.00	1.00	1.00	1.00
Overweight	0.73 (0.32 to 1.63)	1.42 (0.90 to 2.23)	1.02 (0.34 to 3.06)	1.04 (0.51 to 2.11)	0.53 (0.16 to 1.79)	1.86 (1.03 to 3.39)
Obese	2.11 (0.69 to 6.40)	1.50 (0.79 to 2.86)	1.13 (0.14 to 9.15)	1.83 (0.78 to 4.29)	3.20 (0.81 to 12.57)	1.19 (0.44 to 3.25)
p value‡	0.32	0.27	0.42	0.43	0.54	0.43
Inhaled steroid or cromoglycate in last 12 months						
Underweight	1.86 (0.79 to 4.36)	0.66 (0.15 to 2.89)	2.38 (0.63 to 9.02)	0.54 (0.07 to 4.32)	1.69 (0.55 to 5.18)	0.83 (0.10 to 6.95)
Normal	1.00	1.00	1.00	1.00	1.00	1.00
Overweight	1.38 (0.59 to 3.21)	1.21 (0.78 to 1.86)	1.18 (0.25 to 5.54)	0.97 (0.51 to 1.84)	1.56 (0.57 to 4.32)	1.48 (0.82 to 2.68)
Obese	0.87 (0.11 to 6.66)	2.33 (1.35 to 4.00)	2.73 (0.32 to 23.30)	3.09 (1.53 to 6.26)	††	1.52 (0.62 to 3.69)
p value‡	0.80	0.002*	0.89	0.0008**	0.85	0.42
Medication in last 12 months						
Underweight	1.08 (0.55 to 2.11)	0.58 (0.17 to 2.02)	0.67 (0.19 to 2.27)	0.67 (0.14 to 3.12)	1.48 (0.65 to 3.40)	0.47 (0.06 to 3.87)
Normal	1.00	1.00	1.00	1.00	1.00	1.00
Overweight	0.77 (0.36 to 1.41)	1.25 (0.87 to 1.80)	0.69 (0.23 to 2.03)	1.18 (0.71 to 1.97)	0.74 (0.30 to 1.82)	1.32 (0.79 to 2.22)
Obese	1.00 (0.29 to 3.42)	1.96 (1.19 to 3.22)	1.69 (0.35 to 8.02)	2.31 (1.19 to 4.45)	0.56 (0.07 to 4.43)	1.57 (0.73 to 3.37)
p value‡	0.62	0.0007**	0.85	0.001**	0.40	0.18
BHR to exercise						
Underweight	1.47 (0.76 to 2.85)	0.53 (0.07 to 4.02)	1.64 (0.64 to 4.23)	0.75 (0.09 to 5.99)	1.35 (0.53 to 3.42)	§
Normal	1.00	1.00	1.00	1.00	1.00	1.00
Overweight	1.67 (0.95 to 2.93)	1.05 (0.60 to 1.81)	2.10 (0.96 to 4.59)	0.94 (0.46 to 1.95)	1.34 (0.59 to 3.04)	1.15 (0.50 to 2.67)
Obese	1.20 (0.35 to 4.12)	0.87 (0.36 to 2.10)	0.82 (0.10 to 6.58)	0.87 (0.29 to 2.59)	1.60 (0.34 to 7.66)	0.77 (0.17 to 3.44)
p value‡	0.85	0.87	0.92	0.78	0.88	0.90

BHR, bronchial hyperresponsiveness.

*Meets the Holm's criterion for significance (adjusting for eight comparisons) at the 5% level.²²**Meets the Holm's criterion for significance (adjusting for eight comparisons) at the 1% level.²²

†2000 data restricted to children in their 12th year.

‡p value for trend with BMI SDS.

§One observation is missing information on sex in year 2000.

¶No underweight subjects with this outcome.

††No obese subjects with this outcome.

difficult to conclude that associations with obesity in children are recent.

Reporting bias may explain associations of asthma with obesity if dyspnoea in obese children is interpreted as a symptom of asthma by clinicians. In support of this, we found that BHR to exercise (an objective marker of asthma) was no more common among obese children. We also found no significant association between BMI and atopy. These findings confirm those of Schachter *et al*⁸ in Australian adults although, in a later report, these authors found that high BMI was associated with a higher prevalence of atopy in girls.²⁷ In Taiwan, Huang *et al*¹⁴ found that BMI was associated with an increased prevalence of BHR and atopy among girls, but for

BHR this was due to a reduced prevalence in the lowest quintile only.

Although many studies have reported sex differences in the effect of obesity on asthma, few studies have reported the significance of the interaction term.¹³ We found no significant interaction with sex for any of the outcomes. However, these tests have low power and, in general, as in other studies, the size of the effect was larger for obese girls than for obese boys. After adjustment for potential confounders, increasing BMI SDS in girls in year 2000 showed significant trends with increases in asthma disease and symptom reporting and asthma medication use. These findings are independent of frequency of hamburger consumption and exercise. We

Table 4 Adjusted OR (95% CI) showing associations for underweight, overweight, and obesity versus normal weight for year 2000 among all children, girls and boys (using the total dataset for 2000)

Outcome variables	All† (N= 1237)	Girls‡ (N= 617)	Boys‡ (N= 620)
Wheeze ever			
Underweight	0.84 (0.41 to 1.72)	0.42 (0.13 to 1.33)	1.58 (0.58 to 4.30)
Normal	1.00	1.00	1.00
Overweight	1.02 (0.76 to 1.36)	1.40 (0.92 to 2.13)	0.77 (0.50 to 1.18)
Obese	1.41 (0.90 to 2.20)	2.23 (1.18 to 4.22)	0.88 (0.46 to 1.70)
p value¶	0.051	0.00003**	0.36
Wheeze in last 12 months			
Underweight	0.49 (0.17 to 1.42)	0.47 (0.10 to 2.12)	0.52 (0.11 to 2.35)
Normal	1.00	1.00	1.00
Overweight	1.15 (0.81 to 1.62)	1.23 (0.74 to 2.03)	1.09 (0.67 to 1.78)
Obese	1.95 (1.20 to 3.16)	1.85 (0.92 to 3.75)	2.09 (1.06 to 4.15)
p value¶	0.002*	0.002*	0.17
Wheeze with exercise			
Underweight	0.35 (0.10 to 1.17)	0.21 (0.03 to 1.59)	0.48 (0.10 to 2.21)
Normal	1.00	1.00	1.00
Overweight	1.15 (0.82 to 1.62)	1.38 (0.85 to 2.24)	1.01 (0.61 to 1.65)
Obese	1.63 (1.01 to 2.65)	1.62 (0.80 to 3.27)	1.82 (0.91 to 3.65)
p value¶	0.0157	0.02	0.17
Asthma ever			
Underweight	1.14 (0.55 to 2.37)	0.93 (0.31 to 2.75)	1.44 (0.51 to 4.05)
Normal	1.00	1.00	1.00
Overweight	1.07 (0.79 to 1.46)	1.44 (0.93 to 2.24)	0.86 (0.56 to 1.34)
Obese	1.71 (1.09 to 2.69)	3.10 (1.63 to 5.93)	1.00 (0.52 to 1.93)
p value¶	0.04	0.0002**	0.48
Hospital admission ever			
Underweight	2.35 (1.01 to 5.47)	2.23 (0.65 to 7.64)	2.54 (0.75 to 8.56)
Normal	1.00	1.00	1.00
Overweight	1.38 (0.92 to 2.07)	0.92 (0.48 to 1.78)	1.98 (1.15 to 3.39)
Obese	1.63 (0.93 to 2.87)	2.05 (0.93 to 4.53)	1.03 (0.42 to 2.49)
p value¶	0.33	0.37	0.80
Inhaled steroid or cromoglycates in last 12 months			
Underweight	0.55 (0.16 to 1.84)	0.36 (0.05 to 2.80)	0.81 (0.18 to 3.72)
Normal	1.00	1.00	1.00
Overweight	1.16 (0.78 to 1.73)	1.08 (0.60 to 1.94)	1.30 (0.75 to 2.25)
Obese	2.33 (1.39 to 3.91)	2.97 (1.43 to 6.18)	1.98 (0.92 to 4.26)
p value¶	0.004*	0.003*	0.22
Medication in last 12 months			
Underweight	0.88 (0.38 to 2.08)	1.01 (0.31 to 3.24)	0.78 (0.21 to 2.83)
Normal	1.00	1.00	1.00
Overweight	1.22 (0.87 to 1.70)	1.34 (0.83 to 2.18)	1.19 (0.74 to 1.91)
Obese	2.07 (1.30 to 3.31)	2.71 (1.39 to 5.31)	1.73 (0.88 to 3.42)
p value¶	0.001*	0.00006**	0.16
BHR to exercise			
Underweight	0.36 (0.05 to 2.70)	0.65 (0.08 to 5.20)	§
Normal	1.00	1.00	1.00
Overweight	0.94 (0.56 to 1.58)	0.89 (0.44 to 1.81)	1.04 (0.47 to 2.29)
Obese	0.94 (0.44 to 1.98)	0.91 (0.33 to 2.47)	0.96 (0.29 to 3.16)
p value¶	0.91	0.96	0.96
Atopy			
Underweight	0.51 (0.22 to 1.21)	0.91 (0.30 to 2.71)	0.25 (0.06 to 1.13)
Normal	1.00	1.00	1.00
Overweight	1.05 (0.78 to 1.43)	0.83 (0.53 to 1.30)	1.31 (0.86 to 2.01)
Obese	1.44 (0.91 to 2.28)	1.47 (0.76 to 2.84)	1.45 (0.76 to 2.78)
p value¶	0.016	0.32	0.012

*Meets the Holm's criterion for significance (adjusting for nine comparisons) at the 5% level.²²**Meets the Holm's criterion for significance (adjusting for nine comparisons) at the 1% level.²²

†Adjusted for family history of allergic disease, family size (as a continuous variable), birth weight (< or ≥2500 g), current smoking in the home, father's years of post primary education (< or ≥5.5 years), frequency of hamburger consumption (1+ times a week, less than once a week, or never) and exercise (once a week or less, 2–3 times a week, 4–6 times a week, or everyday), sex, ethnicity (Maori/Polynesian or European/other), and year born (1988 or 1989).

‡Adjusted as previously but after removal of sex as a variable.

¶p value for trend with BMI SDS.

§No underweight boys with this outcome.

acknowledge the limitations in defining food consumption and exercise frequency according to questionnaire based reports. However, few other studies⁶ have been able to adjust for these potential confounders, both of which have previously shown associations with asthma.^{28–31}

As an explanation for the predominance of asthma in obese women and girls,^{7 11 12 15} in a recent review Tantisira *et al*³² discuss the possibility that obesity amplifies the pro-inflammatory effects of leptin and oestrogens in women.

Furthermore, obese girls reach puberty earlier than non-obese girls.³³ Although some girls in the current study would have reached puberty, we did not collect this information. However, studies of prepubescent children^{11 13} showing a greater risk of asthma among obese girls would tend to refute this hypothesis, unless prepubescent children are exposed to sources of environmental oestrogens, such as in food, which become concentrated in adipose tissue.

Obesity may also develop as a consequence of asthma if the disease leads to a sedentary lifestyle. However, reverse causation is an unlikely explanation for the association since prospective studies have shown that obesity precedes the onset of asthma^{12 15} and interventions that reduce obesity in asthmatics are followed by improvements in airways obstruction, peak flow variability, and symptoms.^{16 17}

There is some evidence for an association of BMI with asthma but this does not explain the increasing prevalence of asthma. Despite indications in our data and others^{11 13 14} that this association is stronger in girls, we failed to show significant interactions with sex. The exact nature of the effect of obesity on asthma needs to be further explored prospectively in large cohort studies.

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Authors' affiliations

K Wickens, J Crane, Wellington Asthma Research Group, Wellington School of Medicine and Health Sciences, New Zealand

D Barry, N Bone, Healthcare Hawkes Bay, Hastings, New Zealand

A Friezema, R Rhodius, Faculty of Medicine, University of Groningen, The Netherlands

G Purdie, Department of Public Health, Wellington School of Medicine and Health Sciences, New Zealand

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