Association of Body Mass Index and Obesity Measured in Early Childhood With Risk of Coronary Heart Disease and Stroke in Middle Age

Findings From the Aberdeen Children of the 1950s Prospective Cohort Study

Debbie A. Lawlor, PhD, MSc, MFPHM; David A. Leon, PhD, MSc

Background—There is concern that the childhood epidemic of obesity will result in increases in the risk of cardiovascular disease in the future; however, there is currently little direct evidence on this issue.

Methods and Results—We assessed the association of body mass index, measured when subjects were a mean age of 4.9 years old, with the future risk of coronary heart disease (CHD) and stroke in a large Scottish birth cohort (born in the 1950s) who have been linked to hospital admissions and mortality data. At the start of the follow-up period (1981), there were 11 106 (91%) members of the cohort alive and believed to be resident in Scotland. Over the follow-up period, they contributed 245 000 person-years of risk. Among these subjects, there were 302 (53 fatal) cases of CHD, 109 (4 fatal) cases of stroke, and 397 (57 fatal) cases of either a CHD or stroke. There was no association between childhood body mass index and CHD risk. There was no linear association between childhood body mass index and stroke risk, but those who were obese in childhood (top 2.5% of the body mass index distribution) compared with all others had an increased risk of stroke; the adjusted (for gender, father’s occupational social class at birth, number of siblings, and birth weight) hazards ratio was 2.41 (95% CI 1.00 to 5.86).

Conclusions—Body mass index in early childhood does not appear to be associated with increased CHD risk in later life. (Circulation. 2005;111:1891-1896.)

Key Words: obesity ■ pediatrics ■ risk factors ■ cardiovascular diseases

The population prevalence of childhood obesity has increased by ≥3-fold in most industrialized countries over the last 20 years.1 These trends are likely to have major public health consequences.1 In particular, there is concern that the epidemic of childhood obesity will result in increased cardiovascular disease risk in the future; however, the evidence in support of this is unclear.2,3 Many,4–8 although not all,2 studies have found that overweight and obese children and adolescents have adverse lipid, blood pressure, and insulin levels and are more likely to become obese adults than are nonoverweight children. In one recent cross-sectional study, severely obese children (>95th percentile) had increased arterial stiffness compared with healthy-weight children, but there was no difference in carotid intima-media thickness between the 2 groups.9 In a second similar study, but with larger numbers, both arterial stiffness and carotid intima-media thickness were greater in severely obese 6- to 14-year-olds than in healthy-weight children, with this effect attenuating toward the null with adjustment for insulin resistance.10 One historical cohort study found that body mass index measured at either age 9 or age 13 was not associated with intima-media thickness measured at 50 years of age2; however, there was a large loss to follow-up in that study, with both childhood and adulthood measurements for only approximately one third of the original participants. Obesity in childhood or adolescence has also been related to increased all-cause mortality.11–13

To the best of our knowledge, only 3 previous studies have examined the association between childhood or adolescent measures of obesity and cardiovascular disease outcomes. In a nested case-control study, the odds of coronary heart disease (CHD) mortality was 2.5 times greater in those whose body mass index was ≥25 kg/m² at age 18 years than in those whose body mass index at that age was in the range 19.00 to 19.99 kg/m².14 In one small study (n=508), CHD mortality among men whose body mass index was greater than the 75th percentile at 13 to 18 years of age was double that of those whose body mass index was between the 25th and 50th

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percentile after a median of 58 years of follow-up. Similar results were found in both sexes in a second study (n=1234 females and 1165 males) in whom body mass index was assessed when the participants were aged between 2 and 14 years. Finally, among a large cohort of women and men born in Helsinki, Finland, between 1934 and 1944, CHD risk was greatest in those with low birth weight, who then showed downward centile crossing in the first year of life but subsequently showed marked growth acceleration so that by 12 years, their body mass index was identical to the rest of the cohort. A direct assessment of the association between childhood body mass index and future CHD risk was not presented in that study. To the best of our knowledge, no previous study has examined the association between body mass index in childhood and stroke risk.

The aim of the present study was to assess the association between body mass index measured at primary school entry (mean age 4.9 years) and future CHD and stroke risk among a large cohort of individuals born in Aberdeen, Scotland in the 1950s.

Methods
Data from the Aberdeen Children of the 1950s cohort study were used. Described in detail elsewhere, the cohort is based on participants in the Aberdeen Child Development Survey, which collected data on the parental and childhood characteristics of 14,938 children who were in Aberdeen primary schools in 1962. For the 12,150 of these children who were born in Aberdeen, comprehensive information was abstracted from the Aberdeen Maternity and Neonatal Databank (AMND) about the course of their mother’s pregnancy and the children’s physical characteristics at birth. These 12,150 individuals born in Aberdeen between 1950 and 1956 form the index members of the Aberdeen Children of the 1950s cohort.

For the present study, all participants in the Aberdeen Children of the 1950s study who were alive and resident in Scotland at the start of the follow-up period (1981) were included in the analysis (n=11,106).

The Scottish multicenter research ethics committee, local research ethics committees, and the Scottish Privacy Advisory Committee approved the revitalization of the Children of the 1950s cohort. All record linkage was undertaken by the Information and Statistics Division, who provided us with an anonymous data set for analysis.

Data were analyzed with Cox proportional hazards regression models, with participant’s age as the time axis. Because the SMR01 records of hospital admissions only begin in 1981, the follow-up period began on January 1, 1981. Participants were omitted from the analyses if they died (n=116), emigrated to anywhere outside Scotland (n=927), or experienced a nonfatal stroke or CHD (n=1) before January 1, 1981. For the main analyses, childhood gender- and age-standardized body mass index z scores were entered into the model in the 4 percentile categories. Linear trends with childhood body mass index were assessed by entering the body mass index z score as a continuous variable into the models, and nonlinear associations were assessed by adding a quadratic term (z score and the square of the z score) to the model. Potential confounding factors were entered as follows: father’s occupational social class at birth (6-level categorical variable); number of siblings (6-level categorical variable); and birth weight z score (continuous).

Contributions to risk were censored at the earlier of (1) emigration date (this includes emigration to England or Wales); (2) death of a cause other than the outcome of interest; or (3) December 31, 2003. For the emigration date of those moving to England or Wales, we used their first posting date (the date that they first appear on health authority lists as being registered with a general practitioner) with a general practitioner from England or Wales. These are likely to overestimate the time at risk, because most individuals do not register with a new general practitioner immediately on moving. To determine the impact of this on the results of the present study, we undertook sensitivity analyses in which we repeated the Cox proportional hazards models with the date of censoring for those who had moved to England or Wales moved back in time by 6 months, 1 year, and 5 years. Proportionality assumptions were assessed by inspection of cumulative incidence plots and by testing for evidence of a statistical interaction with the time scale of the models. There was no evidence of any violation of the proportionality assumption in any models.

To determine whether the effect of childhood body mass index on cardiovascular disease outcomes varied by gender stratum, specific hazard ratios were examined and likelihood ratio tests of interaction computed. In the Cox proportional models, robust standard errors, taking account of nonindependence between siblings within the sample, were used to estimate all standard errors and probability values. All analyses were conducted using Stata version 8.0 (Stata Corp, 2002).

Socioeconomic position in childhood (known to be associated with adult body mass index and cardiovascular disease) and birth weight (known to be associated with body mass index in childhood and cardiovascular disease risk) were considered to be potential confounding factors in any associations. Two indicators of childhood socioeconomic position were assessed: the occupational social class of the participant’s father at the time of their birth, obtained from the AMND obstetric records (6 categories: I, professional; II, managerial; III, nonmanual/skilled nonmanual; III, manual/skilled manual; IV semi-skilled; and V, unskilled manual; these categories can be collapsed into 2 categories: nonmanual [I-IIInm] and manual [IIIm to V]), and the number of siblings, obtained from the subjects at the time of the 1962 survey (6 categories: 0, 1, 2, 3, 4, ≥5). Birth weight for gender and gestational age z score were derived from data in the AMND.

In 1999, we began tracing study members through the National Health Service Central Register (NHSCR), and 97% have been traced successfully. Traced participants have been linked to the Scottish Morbidity Register (SMR01), which provides information for all admissions to hospitals in Scotland, including International Classification of Diseases (ICD) coded diagnoses. Hospital admissions to hospitals in England and Wales cannot be obtained, which means that individuals who migrated to England and Wales could no longer be considered at risk for the purpose of our survival analyses. We were notified by the NHSCR of the dates and causes of deaths that occurred in Scotland or England and Wales. The codes used to define CHD were 410 to 414 and 429.2 (ICD-9) and I20 to I25 and I51.6 (ICD-10), and those used to define stroke were 430 to 438 (ICD-9) and I60 to I69 and G45 (ICD-10).

Statistical Methods
Data were analyzed with Cox proportional hazards regression models, with participant’s age as the time axis. Because the SMR01 records of hospital admissions only begin in 1981, the follow-up period began on January 1, 1981. Participants were omitted from the analyses if they died (n=116), emigrated to anywhere outside Scotland (n=927), or experienced a nonfatal stroke or CHD (n=1) before January 1, 1981. For the main analyses, childhood gender- and age-standardized body mass index z scores were entered into the model in the 4 percentile categories. Linear trends with childhood body mass index were assessed by entering the body mass index z score as a continuous variable into the models, and nonlinear associations were assessed by adding a quadratic term (z score and the square of the z score) to the model. Potential confounding factors were entered as follows: father’s occupational social class at birth (6-level categorical variable); number of siblings (6-level categorical variable); and birth weight z score (continuous).

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To determine whether the effect of childhood body mass index on cardiovascular disease outcomes varied by gender stratum, specific hazard ratios were examined and likelihood ratio tests of interaction computed. In the Cox proportional models, robust standard errors, taking account of nonindependence between siblings within the sample, were used to estimate all standard errors and probability values. All analyses were conducted using Stata version 8.0 (Stata Corp, 2002).
TABLE 1. Rates of CHD and Stroke up to December 2004 Among 11 106 Women and Men Born in Scotland in the 1950s

<table>
<thead>
<tr>
<th></th>
<th>Women Rate per 10 000 Women-Years</th>
<th>Men Rate per 10 000 Men-Years</th>
<th>Total Rate per 10 000 Person-Years</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD</td>
<td>n 69 (5.7 (4.5–7.3))</td>
<td>233 18.7 (16.5–21.3)</td>
<td>302 12.3 (11.0–13.8)</td>
</tr>
<tr>
<td>Stroke</td>
<td>51 4.2 (3.2–5.6)</td>
<td>58 4.6 (3.6–6.0)</td>
<td>109 4.4 (3.7–5.4)</td>
</tr>
<tr>
<td>Either CHD or stroke</td>
<td>117 9.8 (8.1–11.7)</td>
<td>280 22.5 (20.1–25.3)</td>
<td>397 16.3 (14.7–17.9)</td>
</tr>
</tbody>
</table>

Rates are for hospital admissions and mortality.

Results
The mean (SD) age of the children at the time of their school medical examination (when their height and weight were assessed) was 4.9 (0.7) years. Twenty-two percent of the children were aged 4 years when they were measured, 74.9% were aged 5 years, and 2.8% were aged 6 years or older. When those were aged 6 years or older at assessment were excluded from the analyses, results did not differ from those presented here for the whole cohort. The body mass index range for boys was 10.3 to 32.3 kg/m², with a mean (SD) of 16.6 (1.8) kg/m² and a median of 16.4 kg/m². For girls, the range was 9.6 to 35.5 kg/m², with a mean (SD) of 16.3 (2.0) kg/m² and a median of 16.0 kg/m². With the adult equivalent categories, 17.9% of the boys were overweight (equivalent to an adult body mass index 25 to 29.99 kg/m²), and an additional 2.1% were obese (equivalent to an adult body mass index ≥30 kg/m² or more); 17.7% of the girls were overweight, and an additional 3.0% were obese.

A total of 671 participants emigrated during the follow-up period (either to England and Wales or other countries). These were censored at the date that they emigrated. Childhood body mass index was not related to the likelihood of emigration, with 25% of those in the lowest body mass index z-score quarter, 26% in the middle quarter, 25% in the third quarter, and 24% in the highest quarter emigrating (P = 0.7). Over the follow-up period, a total of 420 participants died of any cause (these deaths include deaths that occurred in Scotland, England, and Wales). All-cause mortality was not related to emigration (P = 0.4), but there was a linear association between childhood body mass index z score and all-cause mortality (hazard ratio for an increase in 1 unit of body mass index z score: 1.06 [95% CI 0.98 to 1.13], P = 0.10), which did not reach levels of statistical significance. For the remaining analyses, deaths of cardiovascular disease that occurred in Scotland only are considered, because we did not have information on nonfatal events in England and Wales and therefore censored all of those who moved to England and Wales during the follow-up period.

At the start of the follow-up period (1981), there were 11 106 members of the cohort alive and believed to be resident in Scotland. Over the follow-up period, they contributed 245 000 person-years of risk. Among these subjects, there were 302 cases of CHD (53 fatal), 109 cases of stroke (4 fatal), and 397 cases of either a CHD or stroke (57 fatal). Table 1 shows rates of CHD and stroke for women and men. The associations of childhood body mass index with both CHD and stroke were the same in both sexes, and therefore all results are presented for women and men combined (all probability values for interactions with gender > 0.4). For example, the hazard ratio for CHD for a 1-kg/m² increase in boys was 0.99 (95% CI 0.92 to 1.07), and for girls, it was 0.97 (95% CI 0.84 to 1.11); the hazard ratio for stroke was 1.07 (0.96, 1.19) in boys and 1.06 (95% CI 0.95 to 1.18) in girls; for either CHD or stroke, it was 1.02 (95% CI 0.96 to 1.08) in boys and 1.01 (95% CI 0.92 to 1.10) in girls.

Table 2 shows the rates of CHD and stroke and other characteristics by categories of the childhood body mass index distribution. Subjects from more adverse socioeconomic positions in childhood (fathers in manual occupations and having 5 or more siblings) had higher body mass indices in childhood, and there was a strong positive association between birth weight and childhood body mass index. These associations are in the same direction and of similar magnitudes to what would be expected from the existing literature. Rates of CHD increased with each category of body mass index from the lowest to third-highest quarter, but then decreased in those above the 75th percentile. Rates of stroke were similar in those in the 50th to 74th percentile category and those in the above-75th percentile category and were higher in these 2 categories than stroke rates in those with lower body mass indices; however, there was no strong statistical evidence of a linear or nonlinear (quadratic) association between childhood body mass index and either CHD or stroke.

Table 3 shows the multivariable associations of childhood body mass index with CHD and stroke outcomes. There was no association between childhood body mass index and either outcome in unadjusted or adjusted models. When these analyses were repeated with the thresholds for defining overweight and obesity based on growth trajectory models that give childhood values that equate to the thresholds used to define adult overweight and obesity, there was no association between childhood overweight or obesity status and risk of CHD and no association between being overweight and stroke risk (Table 4). Those in the obese category (n = 282, 2.5% of the total) were at increased risk of stroke compared with all others (Table 4).

Discussion
Among this cohort of women and men born in Scotland in the 1950s, we found no clear evidence of an association between early childhood body mass index (measured at a mean age of
4.9 years) and future CHD risk. Although there was no linear association across the early childhood body mass index distribution with stroke risk, there was evidence that children who were defined as obese in childhood (with the use of adult-equivalent categories and who represented 2.5% of the subjects) were at increased risk of stroke in later life; however, because of the small numbers of stroke cases, our estimate of the effect of childhood obesity on stroke risk was imprecise and needs confirmation in larger prospective cohort studies.

Most,4–8,10 although not all,2 previous studies have found body mass index in childhood to be related to cardiovascular disease risk factors, including dyslipidemia and insulin resistance, as well as indicators of atherosclerosis, including arterial stiffness and intima media thickness, measured in childhood; however, there are few prospective studies of the relationship between childhood body mass index and disease end points. To date, this is the largest study to assess the association of body mass index measured in childhood with CHD and the only one to assess the association with stroke outcomes. Unlike 2 previous studies, we did not find that those with early childhood body mass indices above the 75th percentile had higher CHD risk than those in the 25th to 49th percentile category.15,16 A previous nested case-control study (n=648 cases) also found increased risk of CHD mortality in those with a body mass index above 25 kg/m² compared with those in a “normal” body mass index category at age 18 years.14 Again, the results for our adult-equivalent body mass index categories are not consistent with these findings. Several factors may explain these differences. The results in the earlier studies may have been chance positive findings. Other studies may have found null associations, as in the present study, but these may not have been published (publication bias).

**TABLE 2. Rates of CHD and Stroke, Social Class at Birth, Number of Siblings, and Birth Weight by Early Childhood BMI z Score**

<table>
<thead>
<tr>
<th>BMI z Score Category at Primary School Entry (Mean Age 4.9 y)</th>
<th>&lt;25th Percentile</th>
<th>25–49th Percentile</th>
<th>50–74th Percentile</th>
<th>≥75th Percentile</th>
<th>P Linear*</th>
<th>P Nonlinear†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SD) absolute BMI, kg/m² [n for males]</td>
<td>15.0 (0.7) [1404]</td>
<td>15.9 (0.4) [1445]</td>
<td>16.9 (1.4) [1499]</td>
<td>18.3 (2.1) [1337]</td>
<td>0.7</td>
<td>0.3</td>
</tr>
<tr>
<td>Mean (SD) absolute BMI, kg/m² [n for females]</td>
<td>14.7 (0.7) [1425]</td>
<td>15.8 (0.4) [1308]</td>
<td>16.5 (0.4) [1345]</td>
<td>18.2 (2.3) [1343]</td>
<td>0.5</td>
<td>0.4</td>
</tr>
<tr>
<td>CHD rate per 10 000 person-years (95% CI)</td>
<td>10.2 (8.0, 13.2)</td>
<td>13.1 (10.5, 16.6)</td>
<td>15.5 (12.6, 19.2)</td>
<td>10.6 (8.4, 13.7)</td>
<td>0.7</td>
<td>0.3</td>
</tr>
<tr>
<td>Stroke rate per 10 000 person-years (95% CI)</td>
<td>4.2 (2.9, 6.4)</td>
<td>3.6 (2.4, 5.6)</td>
<td>5.0 (3.6, 7.3)</td>
<td>4.9 (3.4, 7.2)</td>
<td>0.2</td>
<td>0.5</td>
</tr>
<tr>
<td>Manual social class at birth, % (95% CI)</td>
<td>77.3 (75.5, 78.9)</td>
<td>79.3 (77.6, 80.9)</td>
<td>79.8 (78.1, 81.4)</td>
<td>80.1 (78.4, 81.7)</td>
<td>0.05</td>
<td>0.5</td>
</tr>
<tr>
<td>≥5 Siblings, % (95% CI)</td>
<td>15.8 (14.3, 17.4)</td>
<td>17.4 (15.9, 19.1)</td>
<td>18.8 (17.2, 20.5)</td>
<td>20.8 (19.1, 22.7)</td>
<td>&lt;0.001</td>
<td>0.3</td>
</tr>
<tr>
<td>Birth weight z score, mean (SD)</td>
<td>−0.21 (1.00)</td>
<td>−0.08 (0.97)</td>
<td>0.08 (0.97)</td>
<td>0.22 (0.99)</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Assessment of a linear increase per 1 z score.
†With inclusion of a quadratic term in the model.

**TABLE 3. Adjusted Hazard Ratios of CHD and Stroke Risk by Early Childhood Body Mass Index Quarters**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>BMI z Score Category at Primary School Entry (Mean Age 4.9 y)</th>
<th>&lt;25th Percentile</th>
<th>25–49th Percentile</th>
<th>50–74th Percentile</th>
<th>≥75th Percentile</th>
<th>P Linear*</th>
<th>P Nonlinear†</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD</td>
<td>Gender adjusted</td>
<td>0.82 (0.57–1.18)</td>
<td>1.00</td>
<td>1.33 (0.96–1.85)</td>
<td>0.79 (0.54–1.14)</td>
<td>0.6</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td>Fully adjusted‡</td>
<td>0.82 (0.57–1.18)</td>
<td>1.00</td>
<td>1.35 (0.97–1.88)</td>
<td>0.81 (0.55–1.17)</td>
<td>0.7</td>
<td>0.4</td>
</tr>
<tr>
<td>Stroke</td>
<td>Gender adjusted</td>
<td>1.04 (0.56–1.93)</td>
<td>1.00</td>
<td>1.33 (0.75–2.38)</td>
<td>1.27 (0.70–2.27)</td>
<td>0.2</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td>Fully adjusted‡</td>
<td>1.03 (0.56–1.90)</td>
<td>1.00</td>
<td>1.37 (0.75–2.38)</td>
<td>1.34 (0.74–2.46)</td>
<td>0.08</td>
<td>0.3</td>
</tr>
<tr>
<td>Either CHD or stroke</td>
<td>Gender adjusted</td>
<td>0.86 (0.62–1.18)</td>
<td>1.00</td>
<td>1.33 (1.00–1.79)</td>
<td>0.93 (0.68–1.28)</td>
<td>0.6</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td>Fully adjusted‡</td>
<td>0.85 (0.62–1.18)</td>
<td>1.00</td>
<td>1.36 (1.01–1.82)</td>
<td>0.97 (0.70–1.33)</td>
<td>0.5</td>
<td>0.3</td>
</tr>
</tbody>
</table>

Values in parentheses are 95% CIs.
*Assessment of a linear increase per 1 z score.
†With inclusion of a quadratic term in the model.
‡Adjusted for gender, father’s social class at birth, No. of siblings, and birth weight for gestational age z score.
Body mass index measured in early childhood, as in the present study, may not be associated with increased CHD in later life, but greater body mass index in later childhood or adolescence may be associated with increased risk. Correlations between childhood and adult body mass index increase with increasing age at the childhood measurement. Participants in the Harvard Alumni Study had body mass index measured when they were aged 13 to 18 years, and in the Dutch nested case-control study, body mass index was measured at age 18. In the Boyd Orr cohort, the ages ranged from 2 to 14 years, and in that study, there was some evidence that the linear association between body mass index was restricted to those aged 8 years and older. For example, the hazards ratio of CHD comparing those in the 50th to 75th percentile category with those in the 25th to 49th percentile was 2.4 (95% CI 1.3 to 4.5) in those aged 8 years or more and 1.3 (95% CI 0.5 to 3.3) in those aged less than 8 years. However, as the authors pointed out, there was no strong statistical evidence for a difference in the effect by age group, and such subgroup analyses should be treated with caution. Adiposity has also been associated with coronary atherosclerosis in adolescence and early adulthood in autopsy studies. In the Pathological Determinants of Atherosclerosis in Youth (PDAY) study, autopsy results from 3000 individuals aged 15 to 34 years who had died of external causes were examined. Among males in that study, body mass index measured at autopsy was positively associated with fatty streaks and atherosclerotic lesions in both coronary arteries; in females, body mass index was not related to atherosclerosis. Thick panniculus adiposus was associated with a greater number of coronary artery lesions in both genders. It is possible, therefore, that it is obesity in adolescence and young adulthood that is more critical for future cardiovascular disease risk than measures of adiposity in early childhood, as in the present study.

These earlier studies have examined the association with CHD mortality, whereas in the present study, we have examined the association with fatal and nonfatal outcomes, with the majority being nonfatal. It is possible that childhood obesity is associated with factors that affect survival from CHD but not its primary occurrence; however, the mechanisms that are believed to link childhood obesity with future cardiovascular disease risk (ie, the associations in childhood with abnormal lipids, higher blood pressure, arterial stiffness, and intima media thickness) would all be expected to increase incidence as well as survival. The incident cases used in the present study are based on hospital diagnoses and were not ascertained using strict research criteria; however, these SMR01 data have been used extensively in research, with other studies demonstrating associations of established adult cardiovascular disease risk factors with CHD and stroke outcomes using these data that are of the magnitude and direction that one would expect.

All studies to date have used body mass index as their proxy measure of childhood adiposity, and we used category definitions for our main analyses that were similar to previous studies. Therefore, differences in type of measurement cannot explain our different results. Although it has been widely used in clinical practice and research, is easy to measure, is more reproducible than skinfold thickness measurements, and is acceptable to most patients and participants in research, body mass index may not be the best indicator of adiposity in childhood. It would be valuable to have further studies that examine the associations of direct measures of fat mass, and assessments of visceral fat, in childhood with future cardiovascular disease risk.

The findings of the present study are likely to be relevant to contemporary populations, because the body mass index distribution of this sample is almost identical to those of contemporary populations in Britain. For example, mean body mass index among both boys and girls aged 5 years was 16.2 kg/m² in the 1995 to 1997 Health Survey for England, and in the 1998 Scottish Health Survey, it was 16.6 kg/m² for boys and 16.4 kg/m² for girls. Using the same criteria as used in the present study, in 1994, 2.1% of Scottish boys and 3.2% of Scottish girls were overweight. Rates of obesity among children in the United States, particularly among black children, are greater, and in these groups, it is possible that an association with future cardiovascular disease may be found.

In conclusion, we did not find an association between body mass index measured at a mean age of 5 years and risk of future CHD events. There is some evidence from previous studies that body mass index measured in later childhood and in adolescence is associated with increased risk of atherosclerosis; however, ours is the largest cohort study to look at the association of childhood body mass index in young children with CHD events in adulthood. Further research in large prospective studies with repeated measures of childhood anthropometry from early childhood through adolescence are required to determine whether any association between overweight or obesity and CHD risk only becomes apparent in later childhood.

### TABLE 4. Adjusted Hazard Ratios of CHD and Stroke Risk by Overweight and Obese Status in Early Childhood* (Mean Age 4.9 y)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Normal* (n=8830)</th>
<th>Overweight* (n=1994)</th>
<th>Obese* (n=282)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Gender adjusted 1</td>
<td>0.97 (0.72–1.35)</td>
<td>0.71 (0.36–1.39)</td>
</tr>
<tr>
<td></td>
<td>Fully adjusted† 1</td>
<td>0.98 (0.72–1.36)</td>
<td>0.78 (0.39–1.52)</td>
</tr>
<tr>
<td>Stroke</td>
<td>Gender adjusted 1</td>
<td>0.80 (0.46–1.42)</td>
<td>2.38 (0.98–5.78)</td>
</tr>
<tr>
<td></td>
<td>Fully adjusted† 1</td>
<td>0.82 (0.47–1.44)</td>
<td>2.41 (1.00–5.86)</td>
</tr>
<tr>
<td>Either CHD or stroke</td>
<td>Gender adjusted 1</td>
<td>0.96 (0.73–1.27)</td>
<td>1.08 (0.56–2.08)</td>
</tr>
<tr>
<td></td>
<td>Fully adjusted† 1</td>
<td>0.97 (0.73–1.29)</td>
<td>1.25 (0.65–2.40)</td>
</tr>
</tbody>
</table>

*Categories of overweight and obesity were derived on the basis of age- and gender-specific thresholds for children that correspond to a body mass index of 24 kg/m² or greater (for overweight) and 30 kg/m² or over (for obesity) in adulthood. See Cole et al.*

†Adjusted for gender, father’s social class at birth, No. of siblings, and birth weight for gestational age z score.
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References