

Childhood obesity and adult cardiovascular mortality: a 57-y follow-up study based on the Boyd Orr cohort¹⁻³

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ABSTRACT Few studies have examined associations between childhood overweight and adult disease. We examined the relation between BMI measured in childhood and adult all-cause and cardiovascular mortality in a 57-y follow-up of a cohort study based on the Carnegie (Boyd Orr) Survey of Family Diet and Health in prewar Britain (1937–1939). Complete baseline and follow-up data were available for 1165 males and 1234 females who were aged between 2 y and 14 y 9 mo when they were examined. All-cause and cardiovascular mortality were associated with higher childhood BMIs. Compared with those with BMIs between the 25th and 49th centiles, the hazard ratio (95% CI) for all-cause mortality in those above the 75th BMI centile for their age and sex was 1.5 (1.1, 2.2) and for ischemic heart disease it was 2.0 (1.0, 3.9). There was also a suggestion of a nonlinear association with overall mortality; those in the 25–49th centile of the BMI distribution had the lowest mortality rates. The linear associations may be due in part to the tracking of BMI between childhood and adulthood. High BMI in adults is known to be associated with raised blood pressure and abnormal lipid profiles. The relative contributions of adult and childhood overweight to the observed mortality patterns are uncertain. From the public health perspective, strategies aimed at reducing weight in childhood are important but may only affect adult health if such weight reduction persists into adulthood. *Am J Clin Nutr* 1998;67:1111–8.

KEY WORDS Body mass index, childhood, overweight, risk factors, cardiovascular mortality, overall mortality, all-cause mortality, obesity, United Kingdom, Britain, Boyd Orr Study, Carnegie Survey of Family Diet and Health, ischemic heart disease

INTRODUCTION

There is growing concern that the prevalence of obesity is increasing in the developed world (1). Overweight in adulthood is associated with several adverse health outcomes and cardiovascular disease in particular (2). Some cohort studies also show an increased mortality risk in the underweight (3–5). This apparent increased risk is thought to be because the underweight group contains a relatively higher proportion of people with occult disease and because smokers are on average lighter than nonsmokers (2, 6). If both smokers and those dying in the first few years of follow-up are excluded from body mass index

(BMI)-mortality analyses the relation becomes linear, with mortality increasing with increases in BMI (6, 7).

The relation between overweight in childhood and adult disease has been the subject of little research. The available evidence suggests that overweight in childhood is associated with increased all-cause (8–10) and cardiovascular (8) mortality but associations with cancer vary depending on the site of the cancer (8, 11). Obese children tend to become obese adults (12) and for this reason the prevention of overweight in childhood has been suggested as one means of preventing adult diseases associated with obesity (13).

We analyzed the relation between childhood BMI and adult all-cause and cardiovascular disease mortality in a 57-y follow-up of a cohort of 1165 men and 1234 women whose heights and weights were recorded in the Carnegie Survey of Family Diet and Health in prewar Britain (14, 15).

SUBJECTS AND METHODS

The Boyd Orr cohort

The methods used in the Boyd Orr cohort, the tracing of the original survey members, and the representativeness of those traced and flagged on the National Health Service Central Register have been described previously (15). Ethical approval for this analysis was provided by the Office for National Statistics in the United Kingdom. In short, the material used for this analysis is drawn from the original records of the Carnegie Survey of Family Diet and Health in prewar Britain (14). One thousand, three hundred fifty-two families living in 16 areas of England and Scotland were surveyed over a 2-y period between 1937 and 1939. Families were generally identified from the more deprived districts in these areas through contacts made by local health

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workers. Some families from more affluent backgrounds were recruited separately by the survey team. Most of the families who were approached consented to participate and most of them completed the 1-wk household dietary diary. The occupation of the head of the household was recorded, as were details of family composition and total family expenditure on food.

In all but two of the survey areas the children from the families underwent detailed physical examination. This included measures of height and weight. Children <11 y of age were weighed naked, those aged ≥ 11 y "wore only trousers or knickers for which standard reductions were made" (14). Weight was measured by using a W&T Avery standard model calibrated level balance (now known as Avery Berkel, Smethick, United Kingdom) and recorded to the nearest ounce (28.4 g). Standing height was measured to the nearest millimeter with a portable measuring stand (14). The children wore no shoes or socks for these measurements.

Sample used in this analysis

Only survey members aged 2 y to 14 y 9 mo when examined ($n = 2990$) are included in these analyses. Anthropometry in children aged <2 y is unreliable and less complete; 47% of these children had missing values for height or weight. The number of children aged >14 y 9 mo is small ($n = 159$). The National Health Service Central Register has been used to trace survey members and all of the traced survey members who were alive and resident in Britain on January 1, 1948, are included in the mortality analyses, which are based on deaths occurring up to July 31, 1995.

Anthropometry

BMI (or Quetelet index; weight/height²) was used as the measure of adiposity in all analyses. Although BMI varies with height in a curvilinear fashion, it is the simplest and most stable measure of adiposity or malnutrition (16). BMI and height vary with age and sex in children; therefore, to allow comparison between children of different ages and sexes, each child's measurement was converted to SD scores (z scores). These express a child's measurement as the number of SDs from the mean for his or her age and sex. The z score thus provides a means of comparison across groups that is independent of age and sex. z Scores for children's

BMI were calculated by using two methods. First, they were calculated on the basis of 1990 British reference values for BMI (17). Second, because the distribution of BMI in cohort members differed from that of the reference standard (Figure 1) and no acceptable cross-sectional reference standards exist for children in the 1930s, we also used internally derived standards and repeated the main analyses using these measures. Younger cohort members tended to have higher BMIs than expected by modern-day standards, whereas the opposite was the case for older children (Figure 1). These distributional differences may have been due to changes in growth patterns, diet, or child-rearing practices.

For the internally derived z scores the data were transformed by using the reciprocal transformation because the values for BMI within 6-mo age bands were generally positively skewed (17). The transformed values for BMI approximated a normal distribution. Cubic polynomial regression models were then used to estimate expected values for the anthropometric indexes for males and females separately based on the complete data set. Polynomial regression models were also used to smooth the age-specific SDs. SD was modeled on age by dividing the cohort into 6-mo age bands and calculating the SDs for the anthropometric variable within each of these bands.

Statistical methods

The main analyses were performed by using the z scores for BMI derived from the 1990 UK age- and sex-specific reference values (17). Cohort members were categorized into four groups (<25th centile, 25–49th, 50–5th, and >75th centile) on the basis of their BMI in relation to the external reference. Hazard ratios are presented for these four categories with children in the 25–49th centile being used as the reference category. The hazard ratios for those with BMIs >90th centile were compared with the rest of the cohort.

Age-standardized mortality rates were calculated by using person-years at risk. All rates were standardized for age at entry to the study by the direct method by using the study population as the standard. The data fulfilled the assumptions for Cox's proportional hazards models and these were used to adjust for the effects of age and variables relating to childhood social circumstances and adult socioeconomic status in the SAS PHREG procedure (18).

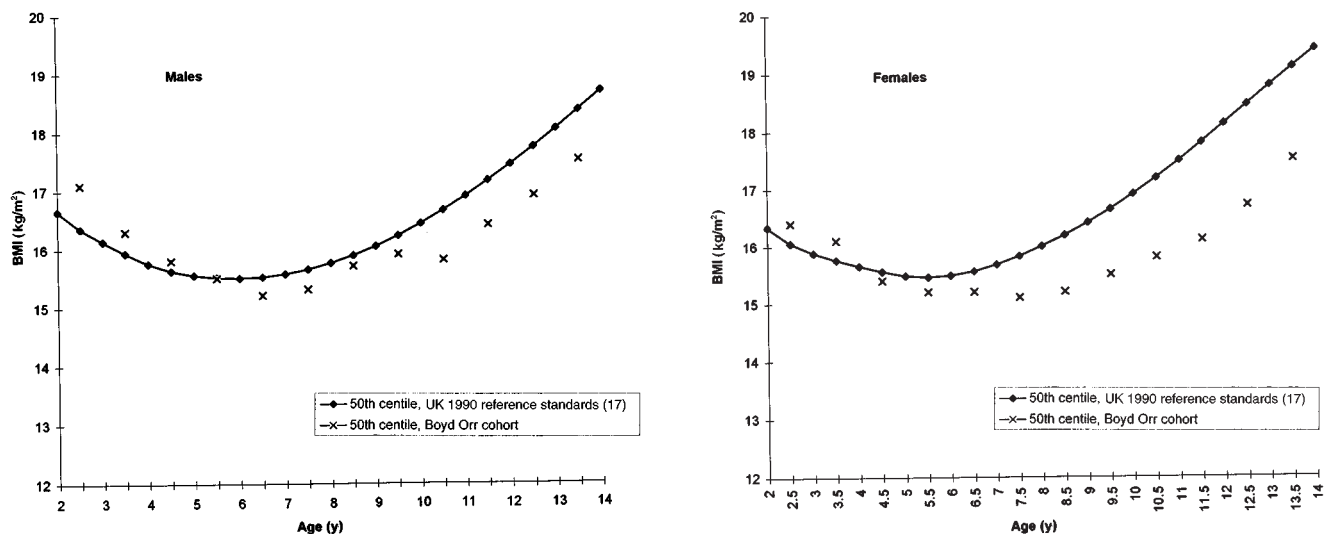


FIGURE 1. 50th centiles for BMI in male and female members of the Boyd Orr cohort compared with 1990 British reference standards (17).

All models were stratified by survey center to account for possible unmeasured differences between centers and differences in measuring techniques as the survey progressed. Because BMI is not independent of height, a term for z score for height was included in all models examining relations between childhood BMI and mortality. The effects of the following variables were examined in proportional hazards models in males and females: 1) the social class of head of household according to the Registrar General's 1931 classification; 2) the total number of children in the household; 3) a measure of current socioeconomic deprivation—the Townsend score—based on levels of car ownership, house ownership, overcrowding, and unemployment in the Health Authority area of residence at the time of the subject's migration, death, or at the end of the follow-up period; and 4) a measure of family food expenditure weighted according to the age and sex of household members, which used weightings modified from a 1933 nutritional report (19). In this latter schema household members were weighted according to the estimated cost of their weekly food requirements relative to those of an adult male. Thus, for example, an adult male was given a weighting of 1.00, an adult female 0.83, and a 2-y-old 0.54. Weighted family food expenditure was then calculated by dividing total food expenditure by the sum of the weighted values for each family member.

Age adjustments were made by using the survey member's age in years and months at the time of the survey. A second variable was also fitted to account for the fact that the survey was carried out over a 2-y period, so those aged 2 y when examined in 1937 were 2 y older than those aged 2 y in 1939.

Separate models were fitted to examine the following: 1) the age-adjusted relation between BMI z score, and all-cause mortality, cardiovascular disease [International Classification of Disease (ICD) codes 390–459] (20), ischemic heart disease (IHD) (ICD 410–414), and stroke (ICD 431–438); and 2) the relation between the above variables and all-cause and disease-specific mortality adjusted for age and the four socioeconomic variables listed above. Tests for nonlinear trends between z score for BMI and mortality were also undertaken. To examine age-specific effects, the analyses were repeated separately for those aged <8 y ($n = 1243$) and ≥ 8 y at the time of the survey ($n = 1156$). This age was chosen so that there were approximately equal numbers of survey members in both age groups. Because there were few deaths of the younger cohort members, this analysis combined males and females and is stratified by sex.

The sampling unit in this survey was the family. Possible clustering effects may have arisen because several cohort members belonged to the same families and therefore shared childhood conditions and possible genetic influences on mortality. Separate analyses have therefore been performed to examine the effects on the main results of taking account of the hierarchical nature of the data set. The STATA software package's cluster option was used to estimate SEs while making allowance for possible nonindependence between study members within a given family (21).

RESULTS

Characteristics of traced cohort members

Complete information on childhood BMI, social circumstances, and adult socioeconomic status was available for 1165 males and 1234 females who contributed 107 488 person-years of risk over

the follow-up period between 1948 and 1995. Over this time, 364 study members died and 67 emigrated. One hundred seven (29%) of the deaths were from IHD (80 males and 27 females) and 24 (6.6%) were from stroke (18 males and 6 females). Cause of death was not known for one (male) survey member.

Compared with the complete data set, those included in the mortality analyses were more likely to be male (83% males compared with 78% females, $P < 0.01$) and were on average 6 mo younger than those who were not included in these analyses ($P < 0.01$). The main reason for these differences is that males and younger study members are more likely to have been traced. There were no significant differences in the BMI z scores, number of children in the household, household food expenditure, or adult socioeconomic status of those included in the mortality analyses compared with the rest of the cohort, although they were shorter by 0.1 SD ($P = 0.03$) and tended to be of slightly higher childhood social class group ($P = 0.009$).

The distribution of BMI by age and sex for the study members included in the main analyses is shown in **Table 1**. It is clear from Table 1 and Figure 1 that the distribution of BMI by age differed from that of the reference standard. Younger children in the study cohort tended to have higher than expected BMIs and older children generally had lower BMIs. On the whole, children in this cohort had lower BMIs than their present-day counterparts. Altogether, 92 (3.8%) of the children were above the 90th centile and 321 (13.4%) were above the 75th centile for BMI according to modern standards. The distributions of the socioeconomic variables used in the main analyses are shown in **Table 2**.

Relation between anthropometry, childhood socioeconomic variables, and adult socioeconomic status

The partial correlations (adjusted for height) between the socioeconomic variables and z scores for childhood BMI for all survey members included in the mortality analyses are shown in **Table 3**. The correlations are all small and these measures account for <1% of the variability in childhood BMI. The significant correlations suggest that in males higher childhood BMI was associated with coming from a larger family and in females higher childhood BMI was associated with socioeconomic deprivation in adulthood. The correlations between the z scores for height and BMI were 0.11 in males and 0.13 in females. Thus, in both sexes, children with higher BMIs tended to be taller.

Age-standardized mortality rates in relation to BMI

The age-standardized mortality rates for males, females, and both sexes combined by quartile of BMI z score are given in **Table 4**. The overall mortality rate was higher in males (4.3/1000 person-years) than in females (2.7/1000 person-years). No clear pattern was seen, although in both sexes combined overall mortality rates were higher in those who were above the 50th centile for BMI. In males and both sexes combined there was a suggestion of a J-shaped relation between BMI and all-cause mortality.

Cox's proportional hazards analyses

The age-adjusted and fully adjusted hazard ratios for all-cause and cause-specific mortality in relation to BMI category are shown in **Table 5**. Those whose BMI z score was between the 25th and 49th centile by modern-day standards were used as the reference group. All analyses were adjusted by z score for height. Tests for trend were based on the continuous variable for BMI z score. Because of the small number of stroke deaths, hazard

ratios across categories are presented here only for males and females combined. Analyses for both sexes together were stratified by sex because no significant interactions were seen between sex and BMI in their relation with either cardiovascular or all-cause mortality. Because inspection of the age-adjusted mortality rate suggested a possible nonlinear relation between BMI and mortality, additional models including a quadratic as well as a linear term for BMI z score were fitted. In both males and females the hazard ratios within categories are consistent with a linear increased risk of IHD mortality with increasing BMI; however, the small number of events within some categories means that the CIs are wide. In addition, in males and both sexes combined there was a significant nonlinear relation between BMI and all-cause mortality. In analyses based on males and both sexes combined, for all outcomes except stroke, those with higher BMIs were at greater risk than those in the lighter categories, although there is a suggestion of a J-shaped relation with some increased risk in the lighter children. These relations were affected little by adjustment for adult and childhood socioeconomic circumstances.

Few children (3.8% of the whole cohort) were above the 90th centile for BMI. Over the follow-up period, 13 of these individuals died, 7 from IHD. The hazard ratios (95% CI) for

all-cause and IHD mortality in these children compared with the rest of the cohort were 1.31 (0.74, 2.31) and 2.82 (1.26, 6.33), respectively.

The analyses were repeated for those aged <8 y ($n = 1243$) and ≥ 8 y ($n = 1156$) separately (Table 6). Because there were few deaths in the younger children, this analysis combined males and females and was stratified by sex as well as district. In those cohort members whose BMI was recorded when they were younger, there was a strong nonlinear relation with overall mortality and a weaker one with cardiovascular deaths. In the older children, there was a significant linear increase in both IHD and all-cardiovascular mortality with increasing BMI. Terms to test for interactions between age and z score for BMI were fitted to models examining the relations between BMI and overall, cardiovascular, IHD, and stroke mortality. No significant interactions were found.

The main analyses were repeated taking account of the hierarchical nature of the data set. All the significant hazard ratios remained significant in these analyses. In addition, the analyses were repeated by using the internally derived z scores for BMI, thus reducing possible bias associated with secular changes in the distribution of BMI with age. In these analyses the hazard ratios associated with IHD mortality were reduced slightly but

TABLE 1
BMI distribution of members of the Boyd Orr cohort included in mortality analyses

Age group ¹	Median BMI kg/m ²	Number over 50th centile of 1990 standards ²	Number over 75th centile of 1990 standards	Number over 90th centile of 1990 standards
Males				
2 ($n = 78$)	17.1	55	28	14
3 ($n = 83$)	16.3	54	33	12
4 ($n = 95$)	15.8	54	25	7
5 ($n = 129$)	15.5	66	35	8
6 ($n = 111$)	15.2	42	13	3
7 ($n = 110$)	15.3	45	16	2
8 ($n = 99$)	15.7	42	10	2
9 ($n = 113$)	15.9	43	9	2
10 ($n = 88$)	15.8	26	4	0
11 ($n = 99$)	16.4	28	5	0
12 ($n = 61$)	16.9	20	7	3
13 ($n = 69$)	17.5	22	1	1
14 ($n = 30$)	17.8	7	3	0
Total ($n = 1165$)		504	189	54
Females				
2 ($n = 92$)	16.4	51	28	11
3 ($n = 100$)	16.1	62	24	9
4 ($n = 104$)	15.4	49	21	6
5 ($n = 120$)	15.2	49	14	3
6 ($n = 122$)	15.2	47	13	2
7 ($n = 99$)	15.1	27	6	2
8 ($n = 120$)	15.2	33	4	0
9 ($n = 102$)	15.5	22	9	4
10 ($n = 105$)	15.8	23	3	0
11 ($n = 99$)	16.1	14	2	0
12 ($n = 79$)	16.7	9	6	1
13 ($n = 70$)	17.5	10	1	0
14 ($n = 22$)	18.1	5	1	0
Total ($n = 1234$)		401	132	38

¹ The cohort was aged between 2 y and 14 y 9 mo when they were examined.

² 1990 UK reference standards (17).



TABLE 2

Distribution of childhood and adult socioeconomic variables used in the main analyses

Variable	Value
Number of children in household	4.3 ± 1.8 (1–9) ¹
Weighted household food expenditure (new pence/wk)	33 ± 12.0 (7–89)
Townsend score ²	0.44 ± 4.6 (–5.1 to 14.7)
Social class of head of household ³	
I/II	166 (6.9) ⁴
III	619 (25.8)
IV	410 (17.1)
V	319 (13.3)
Unemployed	693 (28.9)
Unclassifiable	192 (8.0)

¹ \bar{x} ± SD; range in parentheses.² Based on levels of car ownership, house ownership, overcrowding, and unemployment in geographic areas; high scores indicate greater deprivation.³ Based on Registrar General's 1931 classification of occupations from Social Class I (professional) to Social Class V (unskilled manual).⁴ Number with classification; percentage in parentheses.

remained significant at conventional levels.

DISCUSSION

Two main relations were observed between childhood BMI and adult mortality. First, there was a linear relation between childhood BMI and IHD in analyses based on males and females

TABLE 4

Age-adjusted mortality rates per 1000 person-years and number of deaths by centile category of BMI for males and females

BMI category ¹	Males (n = 1165)			Females (n = 1234)			All subjects (n = 2399)		
	Rate	SE	Number of deaths	Rate	SE	Number of deaths	Rate	SE	Number of deaths
All causes									
< 25th centile	4.3	0.6	55	2.8	0.4	60	3.4	0.4	115
25–49th centile	3.1	0.5	62	2.6	0.4	42	3.1	0.3	104
50–75th centile	3.4	0.6	65	3.5	0.9	31	4.1	0.4	96
> 75th centile	5.1	1.1	33	2.5	0.7	16	4.0	0.7	49
Total	4.3	0.3	215	2.7	0.2	149	3.5	0.2	364
Cardiovascular disease ²									
< 25th centile	2.2	0.4	30	0.9	0.2	19	1.4	0.2	49
25–49th centile	1.7	0.3	27	1.1	0.3	17	1.3	0.2	44
50–75th centile	2.7	0.5	36	1.8	0.8	11	2.2	0.3	47
> 75th centile	2.3	0.8	16	0.5	0.3	3	1.6	0.5	19
Total	2.2	0.2	109	0.9	0.1	50	1.5	0.1	159
Ischemic heart disease ³									
< 25th centile	1.5	0.3	21	0.4	0.1	11	0.9	0.2	32
25–49th centile	0.9	0.2	15	0.4	0.1	11	0.8	0.2	26
50–75th centile	2.3	0.4	31	0.3	0.2	4	1.6	0.3	35
> 75th centile	1.8	0.6	13	0.2	0.2	1	1.1	0.4	14
Total	1.6	0.2	80	0.5	0.1	27	1.0	0.1	107
Stroke ⁴									
< 25th centile	—	—	—	—	—	—	0.2	0.1	8
25–49th centile	—	—	—	—	—	—	0.3	0.1	10
50–75th centile	—	—	—	—	—	—	0.2	0.1	3
> 75th centile	—	—	—	—	—	—	0.4	0.3	3
Total	—	—	—	—	—	—	0.2	0.05	24

¹ Based on UK 1990 reference standards (17); categories based on an individual's BMI in relation to the external standard.² International Classification of Disease death codes 390–459 (20).³ International Classification of Disease death codes 410–414 (20).⁴ International Classification of Disease death codes 431–438 (20). Data not presented for males and females because of small numbers.**TABLE 3**

Pearson's partial correlation coefficients between z score for BMI and both childhood and adult socioeconomic variables adjusted for z score for height

Socioeconomic index	Males (n = 1165)	Females (n = 1234)
Number of children in household	0.08 ¹	0.01
Weighted per capita food expenditure	–0.04	–0.03
Social Class of head of household ²	0.04	–0.01
Adult Townsend score ³	–0.02	0.08 ¹

¹ $P < 0.05$.² Spearman's rank correlation coefficient used, Social Class I (affluent, professional) to Social Class VI (unemployed) (based on Registrar General's 1931 classification of occupations) excludes cohort members with unclassifiable occupations for head of household. Thus, in females BMI decreased as childhood socioeconomic status decreased.³ High Townsend scores indicate greater deprivation, thus, in females, those with higher BMIs in childhood were more likely to experience adult socioeconomic deprivation.

combined. Study members who as children were above the 75th centile for BMI by modern-day standards had about twice the risk of IHD death compared with those whose BMI was between the 25th and 49th centiles. Second, there was a suggestion that those who were underweight in childhood were at increased risk of all-cause mortality compared with those of average weight.

In subgroup analyses based on those aged < or ≥ 8 y when examined, the significant linear relation with IHD was only seen in older children and the nonlinear relation with all-cause



TABLE 5

Overall and cause-specific age- and height-adjusted hazard ratios and 95% CIs for males and females separately in relation to centile category for BMI z score¹

Cause of death ²	Number of deaths	Hazard ratio for BMI centile category				<i>P</i> value for linear term ³	<i>P</i> value for quadratic term ⁴
		<25th	25–49th	50–75th	>75th		
Males (<i>n</i> = 1165)							
All causes	215	1.1 (0.8, 1.6) [293] ⁵	1.0 [368]	1.4 (1.0, 2.0) [315]	1.5 (1.0, 2.4) [189]	0.27	< 0.01
All cardiovascular deaths	109	1.3 (0.8, 2.2)	1.0	1.8 (1.1, 3.0)	1.8 (0.9, 3.6)	0.23	0.14
Ischemic heart disease	80	1.6 (0.8, 3.1)	1.0	2.8 (1.5, 5.3)	2.6 (1.2, 5.8)	0.06	0.10
Females (<i>n</i> = 1234)							
All causes	149	1.0 (0.7, 1.5) [444]	1.0 [389]	1.3 (0.8, 2.1) [269]	1.6 (0.9, 2.9) [132]	0.09	0.19
All cardiovascular deaths	50	0.7 (0.4, 1.4)	1.0	1.2 (0.6, 2.6)	0.9 (0.2, 3.2)	0.12	0.84
Ischemic heart disease	27	0.5 (0.2, 1.3)	1.0	0.7 (0.3, 2.3)	0.5 (0.1, 4.6)	0.16	0.69
All subjects (<i>n</i> = 2399)							
All causes	364	1.1 (0.8, 1.4) [737]	1.0 [757]	1.4 (1.1, 1.8) [584]	1.5 (1.1, 2.2) [321]	0.15	< 0.01
All cardiovascular deaths	159	1.1 (0.7, 1.6)	1.0	1.6 (1.0, 2.4)	1.5 (0.9, 2.6)	0.07	0.17
Ischemic heart disease	107	1.2 (0.7, 2.0)	1.0	2.0 (1.2, 3.4)	2.0 (1.0, 3.9)	0.02	0.16
Stroke	24	0.9 (0.3, 2.3)	1.0	0.4 (0.1, 1.6)	1.2 (0.3, 4.5)	0.30	0.52
Adjusted for age and childhood and adult socioeconomic factors							
Males (<i>n</i> = 1165)							
All causes	215	1.1 (0.7, 1.5) [293]	1.0 [368]	1.5 (1.0, 2.1) [315]	1.6 (1.0, 2.5) [189]	0.16	< 0.001
All cardiovascular deaths	109	1.3 (0.8, 2.2)	1.0	1.9 (1.2, 3.2)	1.9 (1.0, 3.6)	0.15	0.11
Ischemic heart disease	80	1.6 (0.8, 3.1)	1.0	3.1 (1.6, 5.7)	2.7 (1.2, 6.0)	0.04	0.09
Females (<i>n</i> = 1234)							
All causes	149	1.0 (0.7, 1.5) [444]	1.0 [389]	1.3 (0.8, 2.1) [269]	1.5 (0.8, 2.8) [132]	0.36	0.20
All cardiovascular deaths	50	0.7 (0.4, 1.4)	1.0	1.2 (0.6, 2.6)	0.9 (0.3, 3.2)	0.16	0.75
Ischemic heart disease	27	0.6 (0.2, 1.4)	1.0	0.7 (0.2, 2.3)	0.5 (0.1, 4.5)	0.22	0.69
All subjects (<i>n</i> = 2399)							
All causes	364	1.1 (0.8, 1.4) [737]	1.0 [757]	1.4 (1.1, 1.9) [584]	1.6 (1.1, 2.3) [321]	0.11	< 0.001
All cardiovascular deaths	159	1.1 (0.7, 1.6)	1.0	1.6 (1.1, 2.5)	1.6 (0.9, 2.7)	0.05	0.12
Ischemic heart disease	107	1.2 (0.7, 2.0)	1.0	2.1 (1.3, 3.6)	2.0 (1.0, 3.9)	0.02	0.14
Stroke	24	0.9 (0.3, 2.4)	1.0	0.5 (0.1, 1.7)	1.3 (0.3, 5.0)	0.35	0.41

¹ BMI z score based on UK 1990 reference standards (17); categories based on an individual's BMI in relation to the external standard. Children in the 25–49th centile used as reference category.

² International Classification of Disease (20) death codes 390–459 for cardiovascular disease, 410–414 for ischemic heart disease, and 431–438 for stroke.

³ The *P* value for the linear term was derived from the model without the quadratic term; based on continuous variables for BMI z score.

⁴ The *P* value for the quadratic term was obtained by adding the relevant variable (BMI z score squared) to the model including the linear term; based on continuous variables for BMI z score.

⁵ 95% CI in parentheses; *n* for grouping in brackets.

mortality was only significant in younger children. Reasons for the differences in the shape of the relation in the younger and older members of the cohort are uncertain. Correlations between childhood and adult obesity increase with age and it is possible that the stronger relation seen with overweight in older children is because overweight at this age is more strongly predictive of adult overweight (12, 22). However, the results of these subgroup analyses must be viewed with caution because there was no significant interaction with age in the full models.

There are four main limitations to the analyses presented here. First, we currently have no information on the adult BMIs of cohort members, and the extent to which BMI-mortality relations are due to persisting overweight in adulthood cannot be determined. In those studies that do have information on both childhood and adult weight, mortality relations with adult weight are often either stronger than those with weight in childhood (23), are seen only in obese children who become obese adults (10), or diminish when adult weight is adjusted for (8). Second, we have no information currently on adult disease risk factors such as smoking, blood pressure, diet, and exercise habits. There

was, however, no significant relation between BMI and noncardiovascular smoking-related mortality (data not shown). Third, the analyses presented here are based on mortality alone; nonfatal cardiovascular events are not currently recorded for study members. Last, members of this cohort were relatively lean by modern-day standards. This was due in part to both recent increases in BMI and to the fact that some cohort members came from families with extremely poor backgrounds. Thus, the study has limited power to investigate adverse health effects of more extreme overweight in childhood.

Childhood BMI and adult mortality

Both of the population-based studies that have examined the relation between overall mortality and childhood overweight reported significant increased risk associated with higher childhood weight (8, 9). Nieto et al (9) found a linear relation between relative weight and overall mortality in boys and girls who were examined in Hagerstown, MD; they did not, however, examine specific causes of mortality and they found no great differences in these relations in pre- and postpubertal children (9). In a cohort based on the Third Harvard Longitudinal Growth Survey,



TABLE 6

Overall and cause-specific hazard ratios and 95% CIs for persons aged <8 years and ≥8 separately in relation to centile category for BMI adjusted for height¹

Cause of death ²	Number of deaths	Hazard ratio for BMI centile category				P value for linear term ³	P value for quadratic term ⁴
		<25th	25–49th	50–75th	>75th		
Aged <8 y (n = 1243)							
All causes	142	1.6 (1.0, 2.6) [262] ⁵	1.0 [380]	1.3 (0.8, 2.1) [345]	2.0 (1.2, 3.3) [256]	0.72	0.001
Cardiovascular disease	59	1.6 (0.7, 3.3)	1.0	1.2 (0.6, 2.4)	1.6 (0.8, 3.6)	0.83	0.03
Ischemic heart disease	38	1.5 (0.6, 4.1)	1.0	1.3 (0.5, 3.3)	1.9 (0.7, 5.0)	0.50	0.03
Stroke	8	0.4 (0.0, 4.1)	1.0	0.3 (0.0, 3.1)	0.9 (0.1, 6.1)	0.56	0.38
Aged ≥8 y (n = 1156)							
All causes	222	0.9 (0.6, 1.2) [475]	1.0 [377]	1.4 (1.0, 2.1) [239]	1.0 (0.5, 1.9) [65]	0.10	0.03
Cardiovascular disease	100	0.9 (0.5, 1.4)	1.0	1.8 (1.1, 3.0)	1.1 (0.4, 3.1)	0.03	0.31
Ischemic heart disease	69	1.0 (0.5, 1.8)	1.0	2.4 (1.3, 4.5)	1.7 (0.6, 5.3)	0.01	0.31
Stroke	16	1.0 (0.3, 3.1)	1.0	0.5 (0.1, 2.4)	1.0 (0.1, 9.8)	0.37	0.39

¹ BMI based on UK 1990 reference standards (17); categories based on an individual's BMI in relation to the external standard; original sample was stratified by sex and district.

² International Classification of Disease (20) death codes 390–459 for cardiovascular disease, 410–414 for ischemic heart disease, and 431–438 for stroke.

³ The P value for the linear term was derived from the model without the quadratic term; based on continuous variables for BMI z score.

⁴ The P value for the quadratic term was obtained by adding the relevant variable (BMI z score squared) to the model including the linear term; based on continuous variables for BMI z score.

⁵ 95% CI in parentheses; n for grouping in brackets.

males who were overweight in childhood (defined as being above the 75th centile for BMI twice between the ages of 13 and 18 y) were at increased mortality risk; this risk remained significant after adjustment for adult BMI. The increased risk was mainly due to IHD mortality. No relation between childhood overweight and mortality was found in females (8).

We are aware of two cohorts in which the relation between BMI recorded in early adulthood and later all-cause mortality was examined (24–26). For both of these, J-shaped relations between BMI and overall mortality were reported, with significantly greater risk in those overweight [BMI ≥26 (24) and ≥27 (26)]. Both studies were based on male national service examination data and the subjects were followed up to middle age (age: 40–50 y). Thus, the distribution of main causes of death in these cohorts of young men differs from those in the Boyd Orr cohort. However, in both of these cohorts there were greater numbers of cardiovascular deaths in the low-weight categories, supporting the J-shaped relation between BMI and overall mortality found in the Boyd Orr cohort.

BMI and cardiovascular mortality

The linear relation between childhood BMI and adult IHD mortality seen in this study is in keeping with the relations reported in adult cohorts (2, 6, 7, 27, 28). The most likely explanation for this relation in adults is the strong association between being overweight and hypertension, hyperlipidemia, and low levels of exercise. In this study the greatest risks were experienced by those with childhood BMIs above the 90th centile and older children. The stronger associations in older members of the cohort are possibly because correlations between childhood and adult BMI increase with age—overweight 8-y-olds are more likely to become overweight adults than are overweight 2-y-olds (29). Against this interpretation is the observation that none of the interactions between age at measurement and BMI in mortality relations were significant. Childhood overweight is a precursor of adult overweight and the significant relations in the analyses reported here underline the potential importance of overweight in childhood.


Possible explanations for nonlinear mortality relations

The nonlinear (J-shaped) relations between BMI and mortality in this analysis are in keeping with the findings of similar studies (24–26). Two different pathways may underlie this phenomenon. First, those with the lowest BMI in childhood may be wasted as a result of undernutrition and in the long run this may lead to stunting, which in turn may paradoxically predispose them to obesity (30). Such adverse health effects of undernutrition in childhood were also suggested in two studies that reported an association between impaired glucose tolerance or diabetes and low weight in infancy (31) and childhood (32). In other studies, low socioeconomic status in childhood, a marker for possible undernutrition, is associated with adult overweight (33). In the Boyd Orr cohort, childhood BMI was weakly negatively associated with childhood socioeconomic status (as measured by household food expenditure) but positively associated with adult socioeconomic status in women (Table 3). Second, as already mentioned, overweight children tend to become overweight adults and, thus, the increased risk of those with higher childhood BMI may reflect a continuation of their relative overweight status into adulthood. Thus, underweight in childhood is a marker for undernourishment and low socioeconomic status, both of which predispose to overall and cardiovascular mortality. Overweight in childhood predisposes to overweight in adulthood, which has increased mortality risk as well.

Conclusion

This study supports the view that childhood overweight is associated with increased mortality risk in later life. This is likely to be in part because overweight in childhood predisposes to adult overweight, which itself is related to IHD risk. The significant associations between childhood weight and adult mortality many years later have public health significance. In the literature, the greatest risk is seen in overweight children who become overweight adults or underweight children who become overweight adults. The increased risk we observed in the underweight children in this study may be due to other factors associated with their low socioeconomic status in childhood or their



predisposition to obesity and metabolic disturbances in adulthood. From the public health perspective, strategies aimed at reducing weight in childhood may only affect IHD morbidity and mortality if such weight reduction persists into adulthood. Further information on the adult BMI of study members is required to elucidate the effects of childhood overweight considered alongside adult BMI. 

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