Original Contribution

Childhood Body Weight in Relation to Morbidity From Cardiovascular Disease and Cancer in Older Adulthood: 67-Year Follow-up of Participants in the 1947 Scottish Mental Survey

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Although it has been well documented that elevated body weight in middle- and older-aged populations is associated with multiple morbidities, the influence of childhood body weight on health endpoints other than coronary heart disease is not well understood. Accordingly, using a subsample of 4,620 participants (2,288 women) from the Scottish Mental Survey of 1947, we examined the association between body mass index measured at 11 years of age and future risk of 9 independent health endpoints as ascertained from national hospital admissions and cancer registers until 2014 (up to age 77 years). Although there was some evidence of a relationship between elevated childhood body mass index and higher rates of peripheral vascular disease (per each 1 standard deviation increase in body mass index, hazard ratio = 1.21, 95% confidence interval: 1.07, 1.37) and smoking-related cancers (per each 1 standard deviation increase in body mass index, hazard ratio = 1.09, 95% confidence interval: 1.01, 1.17), there was no apparent association with coronary heart disease, stroke (including ischemic stroke), heart failure, or carcinomas of the colorectum, stomach, lung, prostate, or breast. In conclusion, a relationship between childhood body weight and later morbidity was largely lacking in the present study.

body mass index; body weight; cancer; cardiovascular disease; cohort study; life course; morbidity

Abbreviations: BMI, body mass index; CVD, cardiovascular disease.

It has been well documented that obesity in middle- and older-aged populations is associated with multiple morbidities, including coronary heart disease (1, 2), stroke (1, 2) and selected cancers (1, 3). The rapid secular increases in the prevalence of obesity in childhood and early adulthood over the past 3 decades have now brought into sharp focus the potential deleterious effects on health of higher body weight earlier in life.

There is a strong prima facie case for weight in childhood having a long-term influence on adult health. First, body mass index (BMI) “tracks” across the life course, such that overweight and obese children have a higher likelihood of becoming overweight and obese adults (4). Given the established relationship between adult BMI and chronic disease, it is plausible that childhood overweight might, either directly or indirectly, exert a similar impact. Second, in cross-sectional studies of children, obesity has been shown to be associated with blood pressure and cholesterol level (5), both of which predict certain health endpoints, such as cardiovascular disease (CVD), in later life (6, 7). Despite this biological plausibility, the evidence base for a link between childhood overweight and adult health is comparatively modest, and findings are often discordant across studies (8). Thus, although higher levels of pre-adult BMI tend to be related to a higher risk of total mortality and coronary heart disease, findings for all strokes combined are less clear. There are currently too few studies of other common presentations of CVD (e.g., stroke subtypes, heart failure, peripheral vascular disease) and cancer (e.g., colorectal cancer, stomach cancer) to draw any clear conclusions about their links with pre-adult body weight.

Methodological shortcomings also complicate the interpretation of some existing findings. These shortcomings include small sample sizes that result in endpoint rarity, a lack of studies of women, and a reliance by some investigators...
on the distant recall of early-life weight by middle- and older-aged study members (9, 10) rather than direct measurement in early life. Thus, in a systematic review of childhood weight and the rate of prostate cancer, studies in which directly measured BMI was used revealed stronger associations than did those that relied on self-recall (11). It is also the case that most studies include a single endpoint as the outcome rather than exploring the influence of BMI across a range of chronic diseases, thereby limiting insights into the specificity of the association, which is key in establishing causality when using observational data (12). Using data from the Scottish Mental Survey of 1947, we addressed this paucity of data and these methodological concerns by relating direct measurements of BMI when the study members were 11 years of age to an array of chronic diseases up to 67 years later in a well-characterized, nationally representative cohort of men and women.

METHODS

On June 4, 1947, investigators from the Scottish Mental Survey of 1947 attempted to measure the intelligence of every 11-year-old child who was attending school on that day in Scotland; 88% of children responded (n = 70,805) (13, 14). A subgroup of study members—the “36-Day Sample,” comprising children born on the first, second, and third days of each month of 1936—were selected by the Scottish Council for Research in Education for further research participation. This subsample was representative of the full sample from the Scottish Mental Survey of 1947 in terms of sex, geographical location, family size, and cognitive score (15). In present analyses of 5,083 children (2,561 girls) from that period, we excluded those born on the first day of the even-numbered months of 1936 who went on to take part in a more intensive longitudinal survey (the “6-Day Sample”). Our revitalization of the Scottish Mental Survey of 1947 as a cohort study was approved by the Scotland-A Research Ethics Committee, the National Health Service Scotland Privacy Advisory Committee, and the Confidentiality Advisory Group of the Health Research Authority (16).

For each study member, a head teacher answered a questionnaire (known as the “sociological survey”) pertaining to each pupil’s physical attributes and socioeconomic circumstances, including physical disability, father’s occupational level, number of people in the home, and the number of rooms in the home. Physical disability was denoted by a history of chorea, congenital paralysis, defective vision, deafness, encephalitis, epilepsy, or meningitis. Room occupancy was computed by dividing the number of people living in the dwelling by the number of rooms. Father’s or main guardian’s occupation was coded into 1 of 5 social class categorizations (17), ranging from professional (highest prestige) to unskilled. Height (inches) and weight (stone/pounds) were directly measured. Conversion to metric units allowed us to compute BMI using the standard formula (weight (kg)/height (m)²).

Morbidity ascertainment

We electronically traced study members who resided in Scotland using the National Health Service Central Register and located those who had migrated to England and Wales using the Medical Research Information Service Integrated Database and Administration System. For those persons who were not automatically matched, a manual search was undertaken. Morbidity was ascertained through linkage to 2 sources: hospital admissions records (Scotland: 1980–2014; England and Wales: 1997–2013) and cancer registrations (Scotland: 1980–2014; England and Wales: 1984–2013). Irrespective of database, we used the first diagnosis of disease in our analyses, categorizing them according to the International Classification of Diseases, Ninth or Tenth Revisions (see Tables 1 and 2 for codes). We also grouped malignancies into those known to have an association with cigarette smoking (18, 19) and, by inference, those with no such relationship. In so doing, we attempted to circumvent the problem of an absence of data on smoking status, a potential confounding variable in the present study.

Statistical analyses

Of the initial 5,083 study participants, we were able to trace 4,826 (95%). Comparing the traced and untraced groups, we found essentially no difference in BMI (16.8 vs. 16.9, respectively; P value for difference = 0.39) or other baseline characteristics. Exclusion of people with missing data for BMI or other covariates resulted in an analytical sample of 4,620 (2,288 women). In the main analyses, we divided the participants into quartiles by BMI and used those in the lowest quartile as the reference group; we also computed morbidity risk for each 1-standard deviation increase in BMI value. We used Cox proportional hazards regression analyses (20) with age in years as the time scale to compute hazard ratios with accompanying 95% confidence intervals in order to estimate the relationship between childhood BMI and later risk of morbidity. Study members were censored at age at hospitalization or age at the end of follow-up period, whichever occurred first. In preliminary analyses conducted separately for men and women, there was no evidence that sex modified the link between BMI and the major causes of morbidity. These data were therefore pooled. Hazard ratios were adjusted first for sex and then for the additional covariates of fathers’ occupation, room occupancy, height, and physical disability. All analyses were undertaken using the SPSS for Windows, version 21.0 (IBM Corp., Armonk, New York).

RESULTS

The 4,620 study members were followed-up for a maximum of 67 years, resulting in 1,778 (38.6%) CVD-related hospital admissions and 981 (21.3%) cancer diagnoses. In Table 1, we present the associations of childhood BMI with different CVD presentations. Childhood weight was essentially unrelated to the risks of total CVD, coronary heart disease, myocardial infarction, stroke (including ischemic stroke), and heart failure. The risk of peripheral vascular disease was higher in the highest BMI category (for a 1-standard deviation higher BMI, fully adjusted hazard ratio = 1.21, 95% confidence interval: 1.07, 1.37).

The results of the analyses of the association between childhood BMI and subsequent risk of cancer are depicted in Table 2. There were no apparent associations of weight with...
malignancy, which included all cancers combined and colorectal, breast (women only), prostate, stomach, and non–smoking-related cancers. However, BMI was positively related to the risk of smoking-related cancers in selected analyses (hazard ratio = 1.09, 95% confidence interval: 1.01, 1.17).

**DISCUSSION**

Taking the results from the present study together, we found that there was little evidence of a clear relationship between BMI measured at 11 years of age and 9 independent morbidities assessed up to 67 years later. The occasional positive associations that were apparent—with smoking-related cancers and peripheral vascular disease—might have been generated by chance alone, given the large number of models necessarily conducted in the course of our analyses.

**Comparison with other studies**

Our results agree with some existing findings. In a systematic review in which the authors stratified studies into age at BMI assessment (<7, 7–17, or 18–30 years), investigators found that the positive relation with coronary heart disease was strongest in the older group (7 studies) and null in the youngest (3 studies), whereas the aggregated result in the intermediate age group (7 studies) for a 1-standard deviation increase in BMI (hazard ratio = 1.09, 95% confidence interval: 1.00, 1.07) somewhat resembled our own (hazard ratio = 1.00, 1.07).
Table 2. Hazard Ratios for the Associations of Childhood Body Mass Index With Selected Cancer Diagnoses in the 1947 Scottish Mental Survey, 1947–2014

<table>
<thead>
<tr>
<th>Cancer Diagnosis and Model</th>
<th>BMI Quartile&lt;sup&gt;a&lt;/sup&gt;</th>
<th>P for Trend</th>
<th>HR per Each 1-SD Increase in BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 (n = 1,162)</td>
<td>2 (n = 1,152)</td>
<td>3 (n = 1,162)</td>
</tr>
<tr>
<td>All malignant neoplasms&lt;sup&gt;b&lt;/sup&gt;&lt;sup&gt;c&lt;/sup&gt; (n = 981)</td>
<td>20.5 HR 95% CI: 20.4, 22.5</td>
<td>21.5 HR 95% CI: 20.4, 22.5</td>
<td>21.5 HR 95% CI: 20.4, 22.5</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>1.00 Referent</td>
<td>1.00 Referent</td>
<td>1.07 Referent</td>
</tr>
<tr>
<td>Adjusted for multiple covariates&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.00 Referent</td>
<td>1.00 Referent</td>
<td>1.07 Referent</td>
</tr>
<tr>
<td>Breast cancer&lt;sup&gt;e&lt;/sup&gt;&lt;sup&gt;f&lt;/sup&gt; (in women; n = 150)</td>
<td>7.1 HR 95% CI: 6.4, 4.1</td>
<td>6.5 HR 95% CI: 6.4, 4.1</td>
<td>6.5 HR 95% CI: 6.4, 4.1</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>1.00 Referent</td>
<td>0.90 Referent</td>
<td>0.93 Referent</td>
</tr>
<tr>
<td>Adjusted for multiple covariates&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.00 Referent</td>
<td>0.88 Referent</td>
<td>0.92 Referent</td>
</tr>
<tr>
<td>Prostate cancer&lt;sup&gt;c&lt;/sup&gt;&lt;sup&gt;i&lt;/sup&gt; (n = 109)</td>
<td>4.0 HR 95% CI: 4.4, 6.0</td>
<td>3.2 HR 95% CI: 4.4, 6.0</td>
<td>3.2 HR 95% CI: 4.4, 6.0</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>1.00 Referent</td>
<td>1.09 Referent</td>
<td>0.90 Referent</td>
</tr>
<tr>
<td>Adjusted for multiple covariates&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.00 Referent</td>
<td>1.10 Referent</td>
<td>0.89 Referent</td>
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<tr>
<td>Colorectal cancer&lt;sup&gt;c&lt;/sup&gt;&lt;sup&gt;i&lt;/sup&gt; (n = 127)</td>
<td>2.7 HR 95% CI: 3.0, 2.5</td>
<td>2.8 HR 95% CI: 3.0, 2.5</td>
<td>2.8 HR 95% CI: 3.0, 2.5</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>1.00 Referent</td>
<td>1.14 Referent</td>
<td>0.94 Referent</td>
</tr>
<tr>
<td>Adjusted for multiple covariates&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.00 Referent</td>
<td>1.15 Referent</td>
<td>0.92 Referent</td>
</tr>
<tr>
<td>Stomach cancer&lt;sup&gt;c&lt;/sup&gt;&lt;sup&gt;i&lt;/sup&gt; (n = 49)</td>
<td>1.2 HR 95% CI: 1.3, 1.1</td>
<td>0.6 HR 95% CI: 1.3, 1.1</td>
<td>0.6 HR 95% CI: 1.3, 1.1</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>1.00 Referent</td>
<td>1.08 Referent</td>
<td>0.93 Referent</td>
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<tr>
<td>Adjusted for multiple covariates&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.00 Referent</td>
<td>1.09 Referent</td>
<td>0.96 Referent</td>
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<tr>
<td>Lung cancer&lt;sup&gt;c&lt;/sup&gt;&lt;sup&gt;i&lt;/sup&gt; (n = 192)</td>
<td>3.5 HR 95% CI: 4.0, 4.9</td>
<td>4.2 HR 95% CI: 4.0, 4.9</td>
<td>4.2 HR 95% CI: 4.0, 4.9</td>
</tr>
<tr>
<td>Smoking-related cancers&lt;sup&gt;e&lt;/sup&gt; (n = 633)</td>
<td>12.4 HR 95% CI: 13.2, 15.0</td>
<td>14.2 HR 95% CI: 13.2, 15.0</td>
<td>14.2 HR 95% CI: 13.2, 15.0</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>1.00 Referent</td>
<td>1.07 Referent</td>
<td>0.99 Referent</td>
</tr>
<tr>
<td>Adjusted for multiple covariates&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.00 Referent</td>
<td>1.07 Referent</td>
<td>0.97 Referent</td>
</tr>
<tr>
<td>Non–smoking-related cancers&lt;sup&gt;e&lt;/sup&gt; (n = 384)</td>
<td>8.8 HR 95% CI: 8.2, 8.3</td>
<td>7.9 HR 95% CI: 8.2, 8.3</td>
<td>7.9 HR 95% CI: 8.2, 8.3</td>
</tr>
<tr>
<td>Unadjusted</td>
<td>1.00 Referent</td>
<td>0.94 Referent</td>
<td>0.95 Referent</td>
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<tr>
<td>Adjusted for multiple covariates&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.00 Referent</td>
<td>0.94 Referent</td>
<td>0.94 Referent</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index; CI, confidence interval; HR, hazard ratio; SD, standard deviation.

<sup>a</sup> For men, the thresholds for quartiles of BMI (measured as weight in kilograms divided by height in meters squared) were <15.85, 15.86–16.70, 16.71–17.60, and ≥17.61. For women, they were <15.46, 15.46–16.38, 16.39–17.51, and ≥17.52.

<sup>b</sup> International Classification of Diseases, Tenth Revision, codes C000–C439 and C450–C497.

<sup>c</sup> Values are expressed as percentages.

<sup>d</sup> Adjusted for sex, room occupancy, father’s socioeconomic status, height, and physical disability.

<sup>e</sup> International Classification of Diseases, Tenth Revision, codes C500–C599.

<sup>f</sup> International Classification of Diseases, Tenth Revision, codes C500–C509.

<sup>i</sup> International Classification of Diseases, Tenth Revision, codes C000–C169.

95% confidence interval: 0.94, 1.07) (21). In a separate review of 8 studies that featured stroke as the outcome of interest (8), 3 studies had null results, which is in keeping with the finding herein. To the best of our knowledge, ours is the first examination of the link between childhood BMI and other presentations of CVD, particularly heart failure and peripheral vascular disease. Results from a few studies have suggested a positive association between childhood BMI and colorectal cancers (22, 23), but null results have also been reported (24), as they have for lung cancer (23). The mean and standard deviations for BMI in the present study approximate those from other studies in similar era and with similar ages at BMI assessment (25), including samples drawn from the United Kingdom (26, 27). These studies also revealed positive associations with coronary heart disease; therefore, it is perhaps unlikely to be the narrow distribution of BMI in our cohort members, one that is very lean by contemporary standards, that is responsible for the negative results.

Strengths and limitations

Although the present study has a series of strengths, including the unusually comprehensive range of health endpoints,
the use of direct measurements of childhood BMI rather than adult recall, the high proportion of the original study members traced, and the utilization of a nationally representative sample, it is of course not without its weaknesses. The previously described tracking of BMI between childhood and adulthood means that the apparently increased rates of peripheral vascular disease in study members with higher weight in early life may instead be due to adult overweight. We had no repeated measurement of BMI with which to test this hypothesis. Although we were able to analyze the relationships of BMI with an array of health outcomes, there were too few cancer events to facilitate analyses for selected malignancies, such as head and neck cancer and pancreatic cancer. A further potential explanation for our generally negative results is that, unlike in other published analyses, we used nonfatal endpoints only. However, there is evidence that, at least for BMI measured in middle-aged adults, similar findings are apparent for both CVD (28) and cancer (29) irrespective of whether the outcome is incidence (nonfatal events) or mortality. Consistent with our various ethical agreements, the morbidity and mortality data have each been merged separately with the early-life data, creating 2 distinct data sets. Because of this, it was not possible for us to ascertain whether there was a different relationship of pre-adult BMI with the onset of a first event via hospitalization (etiology) or survival from it (prognosis). It is also the case that if an obese adult who was also obese in childhood died suddenly without any recorded morbidities, he or she might contribute to the finding of no association between childhood obesity and later morbidity risk. Again, however, the absence of linked morbidity and mortality data does not facilitate scrutiny of this hypothesis.

In conclusion, a link between pre-adult body weight and later morbidity was largely lacking in the present study. The general paucity of studies in this field should be addressed.

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G.D.B. and C.M.C. contributed equally to this work.

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REFERENCES


