Associations between parental and offspring adiposity up to midlife: the contribution of adult lifestyle factors in the 1958 British Birth Cohort Study

Rachel Cooper, Elina Hyppönen, Diane Berry, and Chris Power

ABSTRACT

Background: Parent-offspring associations in adiposity are well known, but the extent to which they are explained by modifiable environmental and lifestyle factors remains to be elucidated.

Objectives: The objectives were to assess whether 1) parent-offspring associations in body mass index (BMI; in kg/m²) persist from childhood to midadulthood, 2) parental BMI is associated with the offspring’s adult lifestyle, and 3) parent-offspring BMI associations in midadulthood are explained by lifestyle factors.

Design: Participants in the 1958 British Birth Cohort Study and their parents (n = 9346) were examined. Parental BMI was assessed in 1969; offspring (ie, cohort members) BMI was ascertained prospectively at 11 and 44–45 y. Lifestyle factors of the offspring, including diet, physical activity, alcohol consumption, and smoking, were assessed prospectively in adulthood.

Results: Maternal and paternal BMI were positively associated with offspring BMI in both childhood and midadulthood, and the strength of the association did not diminish with offspring age. Maternal BMI was associated with several offspring lifestyle factors across adulthood; fewer associations were observed for paternal BMI. Parent-offspring BMI associations in adulthood were largely maintained after adjustment for multiple lifestyle and socioeconomic factors at different life stages: if parental BMI was 1 unit higher, offspring BMI at 44–45 y was higher by between 0.21 and 0.29 units in adjusted models.

Conclusions: Strong parent-offspring BMI associations are maintained into midlife. These associations are largely unaffected by adjustment for a wide range of lifestyle factors. Offspring of obese parents are an important target for interventions aimed at reducing population levels of overweight and obesity.


INTRODUCTION

Familial risk of overweight and obesity is widely recognized (1–3), as evidenced by consistent findings of a positive association between parental and offspring adiposity across many studies (4). Most studies that have examined these parental-offspring associations have focused on the adiposity of offspring in childhood and/or adolescence (5–11), but studies have also shown that this association persists into adulthood (12–18). Persons whose parents were overweight or obese during their childhood are more likely to be overweight or obese themselves than are those whose parents were of normal weight, and the familial risk of obesity increases as the level of obesity increases.

Genetic predisposition, prenatal programming, and shared environmental factors all play a role in explaining parental-offspring adiposity associations. Genome-wide association studies recently identified variants at several loci, including FTO and MC4R, which are associated with body mass index (19–23). These findings along with other evidence that adiposity is highly heritable (24–28) indicate an important role for genetic factors. However, the speed of the rise in the prevalence of obesity over the past few decades suggests that environmental and behavioral factors are also important determinants of adiposity. Such factors would be expected to contribute to the parent-offspring adiposity association (18) because parents and their offspring share similar environments and lifestyles, largely through the influence of the parent’s lifestyles on that of their offspring (5, 29, 30). Parent-offspring similarities would be expected even when offspring reach adulthood and are living independently, because lifestyle factors and health behaviors are thought to become established early in life (5) and, like adiposity, have been shown to track across life (5, 31–35). However, it is unclear to what extent associations between parental and offspring adiposity are explained by shared environmental and lifestyle factors, given existing evidence—notably from adoption studies (25–28). These studies have shown that the body mass index (BMI) of adopted offspring is more strongly associated with the BMI of their biological parents than with that of their adoptive parents, even

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when adopted offspring have shared the environment of their adoptive family from very early in life (25–28).

As the prevalence of overweight and obesity continue to rise across the globe (36), a growing number of children will be born to obese parents and will thus be at increased familial risk of obesity. Understanding the explanations for the associations between parental and offspring adiposity is therefore increasingly important, especially with regard to modifiable factors, because such knowledge could inform policy strategies designed to reverse current trends in obesity.

The objectives of this study, with the use of data from the 1958 British birth cohort, were to 1) assess whether an association between parental and offspring adiposity persists to midlife and establish whether the magnitude of this association differs from that seen when offspring were children, 2) examine whether parental BMI is associated with lifestyle factors of offspring in early and midadulthood, and 3) most importantly, examine whether associations between parental and offspring BMI in midlife are explained by lifestyle factors of offspring.

SUBJECTS AND METHODS

The 1958 British birth cohort consists of 17,638 males and females followed up since their enrollment in the Perinatal Mortality Survey at the time of their births during 1 wk in March 1958 across England, Scotland, and Wales (37). Immigrants with the same birth dates (n = 920) were recruited into the study up to age 16 y. The cohort has been followed up across life to age 44–45 y, when a target sample of 11,971 cohort members were invited to participate in a biomedical survey, of whom 9377 (78%) responded. Information on the parents of the cohort members was obtained at time points throughout their offspring’s childhood, with these analyses using information collected in 1969 when their offspring, the 1958 cohort members, were aged 11 y. Ethical approval was obtained for this study from the South East Multi-center Research Ethics Committee (ref: 01/1/44), and the study participants provided informed consent.

BMI

BMI of the cohort members at age 11 y was calculated by using measured heights and weights recorded by trained medical personnel using a standardized protocol. BMI at age 44–45 y was calculated by using measured height and weights taken during physical examinations performed by nurses at the study participants’ homes using standardized protocols. Height and weight were measured by using Leicester portable stadiometers and Tanita solar scales, respectively, while the participants were lightly clothed and unshod. Self-reported weights (n = 99) and heights (n = 71) were used if measurements were inaccurate, as assessed by the nurse taking the measurements or if consent for measurement was not provided.

Parents’ heights and weights were reported in 1969 when cohort members were aged 11 y. Heights were reported in feet and inches to the nearest inch. Weights were reported in pre-classified groups ranging from 6 stone 4 pounds (39.9 kg) to 19 stone 10 pounds (125.2 kg) in increments of 3 pounds (2.7 kg). To calculate BMI, heights were converted into meters and a weight (in kg) was assigned that was equivalent to the midpoint of the recorded weight category. For the specified analyses, maternal and paternal BMI (in kg/m²) were categorized into 4 standard groups: <20 (underweight), 20–25 (normal), >25–30 (overweight), and >30 (obese).

Lifestyle factors in early and midadulthood

The lifestyles of cohort members were assessed during the course of the study, with these analyses using the most detailed measures available from early and midadulthood. As described in detail elsewhere (34), at ages 33 and 42 y, cohort members reported their frequency of consumption of different food groups. Variables were created to identify the amount of fried food and the amount of fruit consumed at ages 33 and 42 y, and these 2 measures were used as an indicator in these analyses of overall diet. Fried food consumption was categorized as follows: never, <1 d, 1–2 d/wk, 3–6 d/wk, or ≥1 time/d. Fruit consumption was categorized as follows: >1 time/d, 1 time/d, 3–6 d/wk, 1–2 d/wk, or <1 d/wk.

We used information on physical activity levels recorded at ages 23 and 42 y (34). At age 23 y, the participants were asked a single question about frequency of sports participation in the past 4 wk, which was categorized as follows: >3 times/wk, 1–2 times/wk, 1–3 times in the past 4 wk, or none. At age 42 y, cohort members were asked a single question about participation in regular physical activity, which was categorized into 4 groups: 4–7 times/wk, 2–3 times/wk, 1 time/wk, or ≤2–3 times/mono.

Frequency of television viewing was reported at ages 23 and 44–45 y (38). At age 23 y, cohort members reported how frequently they had watched television in the past 4 wk, which was categorized into 4 groups: <1 time/wk, 1–2 times/wk, 3–4 times/wk, and ≥5 times/wk. At age 44–45 y, hours spent watching television each day was reported and categorized into 5 groups from <1 h/d to >4 h/d.

Smoking status self-reported at ages 23 and 42 y (or at 33 y if missing; n = 168), was categorized as never, ex-smoker, or current smoker. Alcohol consumption at age 23 y was assessed by using self-reports of alcoholic beverages consumed within the previous week, from which the total number of units consumed was estimated. This was then categorized into 3 groups by sex: <14/21 units/wk, ≥15/22 units/wk, or nondrinker. A quantity-frequency index of alcohol use at age 44–45 y was derived from the Alcohol Use Disorders Identification Test questionnaire (39) and categorized into 5 groups based on the average number of standard drinks consumed: nondrinker, 1–7 drinks/wk, 7–14 drinks/wk, 14–21 drinks/wk, or ≥21 drinks/wk.

Covariates

Father’s occupational class at birth (or at 7 y if missing) was categorized into 4 groups by using the Registrar General’s Social Classification: I (professional) or II; IIINM; III; and IV, V (unskilled), or single mother. The classification is a standard method of categorizing occupations in the United Kingdom, coded from professional (I) to unskilled manual (V) (40). Own occupational class of cohort members at age 42 y (or at 33 y if missing) was categorized into the same 4 groups. Educational level attained was recorded at age 42 y and categorized as follows: degree or higher, advanced secondary, ordinary secondary, below secondary, or no qualifications.
Analysis

First, the unadjusted associations between parental and offspring BMI at ages 11 and 44–45 y were tested by using linear regression models. Models included maternal and paternal BMI as continuous terms and, to allow a comparison of the magnitude of associations between ages, sex-specific standardized measures of offspring BMI, at ages 11 and 44–45 y. Correlation coefficients between parental and offspring BMI at ages 11 and 44–45 y were also calculated. To ensure the comparability of results, these associations were assessed among the sample with complete data on both offspring BMI measures. For the purposes of presentation, the unadjusted associations of maternal and paternal BMI, categorized as described above, with offspring BMI at ages 11 and 44–45 y were also tested by using analysis of variance.

Because the distribution of the cohort member’s BMI at both 11 and 44–45 y differed by sex (t tests of sex differences, \( P < 0.001 \)) and there was evidence of sex interactions in some models of the associations between paternal and offspring BMI, all analyses in which offspring BMI was the outcome, including those described above, were performed on sex-specific models.

Second, the unadjusted associations of maternal and paternal BMI with each of the lifestyle factors were tested by using chi-square tests. Associations were then adjusted for sex, education, and occupational class. In these analyses both parental BMI and lifestyle factors were included as categorical terms, and, because there was no evidence of sex interactions in these models, the analyses were run on men and women combined.

Finally, the sex-specific associations of maternal and paternal BMI with offspring BMI in midlife, adjusted for covariates, were tested by using multiple linear regression. Offspring BMI was included as a continuous term, and, in the first sets of models, paternal and maternal BMI were included as categorical variables. Tests of deviation from linearity were performed. When there was no evidence of deviation, parental BMI was included in a second set of models as a continuous term. These analyses were adjusted first for parental age, second for parental age and all lifestyle factors described, third for parental age and markers of lifetime socioeconomic position, and, finally, for all factors.

All analyses, with the exception of those comparing models including offspring BMI at 11 y with BMI at 44–45 y, were run on a sample of 9346 cohort members who had participated in the biometric survey at age 44–45 y, had a valid BMI measure at this age, and were not pregnant at the time of this survey (\( n = 2 \)). To minimize the loss in numbers and the level of possible bias due to missing information, missing values in the covariates were imputed by using the multiple imputation chained equations (MICE) implemented in Stata version 10 (41). The multiple linear regression analyses described were run across 10 multiply imputed data sets. Sensitivity analyses were also performed with each model run on the sample with complete data on all covariates and also with adjustment only for lifestyle factors from midadulthood. Findings from these analyses were similar. Results based on models using multiple imputation are presented.

Results

More than 50% of the cohort member’s mothers and fathers had a BMI in 1969 within the normal range (BMI: 20–25) (Table 1): the mean (\( \pm \)SD) BMI of the mothers was 24.04 \( \pm \) 3.91 and of the fathers was 24.70 \( \pm \) 3.05. Male cohort members had a mean (\( \pm \)SD) BMI of 27.84 \( \pm \) 4.37 at age 44–45 y, and females had a mean (\( \pm \)SD) BMI of 27.59 \( \pm \) 6.47 at 11 y and of 27.00 \( \pm \) 5.64 at age 44–45 y.

In unadjusted analyses, both maternal and paternal BMI were positively associated with offspring BMI at ages 11 and 44–45 y in both sexes (Table 1). The estimated increases in offspring BMI SD scores per 1-unit increase in parental BMI were similar at ages 11 and 44–45 y: 0.06 (95% CI: 0.05, 0.07) at both ages and in both sexes in association with maternal BMI; for paternal BMI,
estimates ranged between 0.05 (0.03, 0.06) and 0.07 (0.06, 0.08). The correlation coefficients between maternal and offspring BMI at ages 11 and 44–45 y were also similar for males and females and at ages 11 and 44–45 y ranging, between 0.21 and 0.23 (Table 2). The correlation coefficients between paternal and male offspring BMI were 0.19 and 0.21 for ages 11 and 44–45 y, respectively, and for females were 0.17 and 0.14, respectively.

Maternal BMI was found to be associated with most offspring lifestyle factors in adulthood in unadjusted analyses (Table 3). A higher proportion of the cohort members whose mothers were obese in 1969, than of cohort members whose mothers had been of normal weight in 1969, were consuming fried food at least once a day at ages 33 and 42 y, were watching television for the most amount of time at ages 23 and 44–45 y, were participating in the least amount of physical activity, and were smoking at ages 23 and 42 y. Many associations were J-shaped, whereby those cohort members whose mothers were normal weight were the least likely to follow an “unhealthy” lifestyle in adulthood. Cohort members whose mothers had been obese were less likely to be consuming the highest levels of alcohol at ages 23 and 44–45 y than were other cohort members. After adjustment for sex and socioeconomic factors, many of the associations found between maternal BMI and offspring lifestyle factors were maintained, including the associations with fried food consumption, television viewing, physical activity, and alcohol consumption at age 42 y. Little evidence suggested that paternal BMI was associated with offspring lifestyle factors (Table 3). Only smoking status at 42 y and television viewing at 44–45 y were associated with paternal BMI at conventional levels of statistical significance in unadjusted analyses, with cohort members whose fathers were overweight or obese in 1969 being more likely to watch television for >4 h/d at age 44–45 y and smoke at age 42 y than cohort members whose fathers were of normal weight in 1969. However, neither of these associations was maintained after adjustment. Offspring BMI at age 44–45 y was strongly associated with most of the lifestyle factors examined (Table 3).

Results from multiple linear regression analyses of parental and offspring BMI demonstrate strong associations in both sexes; if parental BMI was 1-unit higher, offspring BMI at 44–45 y was higher by between 0.24 and 0.35 (Table 4). Adjustment for lifestyle and socioeconomic factors attenuated associations slightly, but all associations were maintained in fully adjusted models (Table 4).

## DISCUSSION

Both maternal and paternal BMI were found to be strongly associated with offspring BMI in childhood and midlife, with no evidence to suggest that the strength of this association had diminished with increasing offspring age. Furthermore, the strong positive associations between parental and offspring BMI in adulthood were largely maintained after adjustment for a range of lifestyle and socioeconomic factors at different life stages. This was despite the fact that maternal BMI was associated with many lifestyle factors of offspring across adulthood, whereby it may have been expected that these may explain the parental-offspring BMI associations to some extent.

The finding that the magnitude of associations found between parental and adult offspring adiposity was not more strongly affected by adjustments for lifestyle and socioeconomic factors suggests that there may be alternative explanations of these associations. There is a growing body of literature that highlights the important role of genetic factors, and recent studies have identified several new common obesity variants (19–21, 42, 43). Heritability of obesity has been estimated to vary between 40% and 70% (43); however, even if the influence of all genes identified to date is combined only a relatively small proportion of the total variation is explained (43). Obesity is clearly a multifactorial trait with a complex genetic background, but our findings suggest that the effect of environmental factors is also not simple. Because neither genetics nor environmental factors alone should be expected to explain the associations, future research should examine gene-environment interactions.

## Comparisons with previous work

Our study builds on previous work on the 1958 birth cohort, which established that parental BMI and offspring BMI up to age 33 y were positively correlated (12); there were associations between cohort members’ BMI trajectories between 7 and 33 y and their children’s BMIs (11), and lifestyle factors, including physical activity and television viewing, were associated with subsequent BMI (38, 44, 45).

Some previous studies have examined the association between parental and offspring adiposity in adulthood (12–18); however, our study provides some of the first evidence that the association persists beyond early adulthood and into midlife. Furthermore, we found no evidence to suggest that these associations diminish with age despite the increased length of time over which influences other than those relating to parents could play a role. Of the

## TABLE 2

Correlation coefficients between offspring BMI at ages 11 and 44–45 y and parental BMI in 1969

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
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<tbody>
<tr>
<td></td>
<td>Maternal BMI</td>
<td>Paternal BMI</td>
</tr>
<tr>
<td></td>
<td>(n = 3537)</td>
<td>(n = 3470)</td>
</tr>
<tr>
<td>Maternal BMI</td>
<td>1.00</td>
<td></td>
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<tr>
<td>Paternal BMI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI at 11 y</td>
<td>0.23</td>
<td>0.19</td>
</tr>
<tr>
<td>BMI at 44–45 y</td>
<td>0.21</td>
<td>0.21</td>
</tr>
</tbody>
</table>

1 The sample includes those with a valid measure of own BMI at ages 11 and 44–45 y (total n = 7392). Total n = 7392 because of missing data on BMI at 11 y for 1954 persons with data on BMI at 44–45 y; n values for each column vary because of missing data on maternal and paternal BMI.

2 n = 3417 for men and 3453 for women.
other studies that have examined offspring BMI in adulthood, one investigated and found, similarly to us, that the parental-offspring association was maintained after adjustment for offspring lifestyle factors in adulthood, despite parental BMI being associated with offspring lifestyle factors (14). However, our study has the strength of being able to examine the role of a wider range of lifestyle factors across multiple time points in adulthood, including earlier in adulthood, closer to the time of direct parental influence. Our findings are consistent with evidence from adoption studies (25–28), which showed stronger associations of adopted offspring BMI with the BMI of their biological parents than with adoptive parents, even when adopted offspring have shared their environment with their adoptive family from very early in life.

Methodologic considerations

We included an extensive range of offspring lifestyle factors covering several stages of life, all of which were ascertained prospectively; this was a major strength of our study. However, it is possible that despite adjustment for this wide range of offspring lifestyle factors, we still failed to capture those that are the most important. Furthermore, despite tracking in the patterns of behavior, it may be that, by examining offspring lifestyle only at specific time points, we did not adequately capture lifestyle when it is most influential. Also, we did not examine parental lifestyle influences on adiposity, and, in sensitivity analyses, it is possible that despite adjustment for this wide range of offspring lifestyle factors, we still failed to capture those that are the most important.

The consumption of fruit and fat was included in the analyses as an indicator of overall diet and to reduce the number of highly correlated dietary variables included in the models. Although it can be argued that consumption of these 2 food groups does not correlate dietary variables included in the models. Although it can be argued that consumption of these 2 food groups does not

### TABLE 3

Distribution of offspring lifestyle factors in early and midadulthood by parental BMI in 1969 and own BMI at age 44–45 y

<table>
<thead>
<tr>
<th>Lifestyle factorsa</th>
<th>33 y</th>
<th>42 y</th>
<th>33 y</th>
<th>42 y</th>
<th>23 y</th>
<th>44–45 y</th>
<th>23 y</th>
<th>42 y</th>
<th>23 y</th>
<th>44–45 y</th>
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<tbody>
<tr>
<td>Maternal BMI</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20 kg/m²</td>
<td>4.3</td>
<td>5.6</td>
<td>12.1</td>
<td>20.2</td>
<td>68.8</td>
<td>9.1</td>
<td>50.4</td>
<td>32.6</td>
<td>34.2</td>
<td>22.2</td>
<td>26.4</td>
<td>10.3</td>
</tr>
<tr>
<td>20–25 kg/m²</td>
<td>3.1</td>
<td>5.2</td>
<td>11.4</td>
<td>18.2</td>
<td>65.1</td>
<td>8.3</td>
<td>48.0</td>
<td>32.5</td>
<td>34.5</td>
<td>22.9</td>
<td>25.2</td>
<td>9.3</td>
</tr>
<tr>
<td>&gt;25–30 kg/m²</td>
<td>4.2</td>
<td>6.1</td>
<td>13.4</td>
<td>19.8</td>
<td>69.5</td>
<td>11.8</td>
<td>52.3</td>
<td>36.6</td>
<td>37.2</td>
<td>26.0</td>
<td>26.3</td>
<td>9.1</td>
</tr>
<tr>
<td>&gt;30 kg/m²</td>
<td>5.6</td>
<td>6.9</td>
<td>15.0</td>
<td>20.8</td>
<td>73.3</td>
<td>14.5</td>
<td>54.6</td>
<td>38.2</td>
<td>42.7</td>
<td>30.2</td>
<td>20.9</td>
<td>7.8</td>
</tr>
<tr>
<td>Unknown</td>
<td>4.2</td>
<td>6.8</td>
<td>11.8</td>
<td>17.7</td>
<td>66.8</td>
<td>9.4</td>
<td>53.3</td>
<td>34.3</td>
<td>41.6</td>
<td>27.3</td>
<td>25.1</td>
<td>8.5</td>
</tr>
<tr>
<td>P value</td>
<td>0.02</td>
<td>0.01</td>
<td>0.14</td>
<td>0.23</td>
<td>0.001</td>
<td>&lt;0.001</td>
<td>0.03</td>
<td>0.002</td>
<td>0.01</td>
<td>0.001</td>
<td>0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>P value</td>
<td>0.48</td>
<td>0.004</td>
<td>0.51</td>
<td>0.04</td>
<td>0.12</td>
<td>0.004</td>
<td>0.26</td>
<td>0.04</td>
<td>0.61</td>
<td>0.45</td>
<td>0.02</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

| Paternal BMI       |      |      |      |      |      |         |      |      |      |         |      |         |
| <20 kg/m²          | 5.5  | 5.0  | 9.3  | 23.8 | 68.8 | 13.4    | 51.4 | 34.8 | 35.0 | 27.7    | 25.9 | 7.6     |
| 20–25 kg/m²        | 3.7  | 5.4  | 11.8 | 18.3 | 66.3 | 8.7     | 49.2 | 34.0 | 34.7 | 23.4    | 25.1 | 9.7     |
| >25–30 kg/m²       | 4.0  | 6.1  | 12.9 | 19.0 | 67.5 | 9.9     | 49.6 | 33.0 | 36.8 | 23.5    | 25.8 | 9.2     |
| >30 kg/m²          | 3.3  | 5.1  | 14.2 | 18.3 | 69.5 | 12.4    | 57.3 | 36.4 | 38.1 | 26.7    | 22.2 | 6.5     |
| Unknown             | 3.6  | 6.6  | 12.1 | 19.2 | 68.4 | 10.4    | 53.2 | 35.1 | 41.7 | 28.8    | 25.4 | 8.3     |
| P value            | 0.07 | 0.12 | 0.67 | 0.48 | 0.79 | 0.009   | 0.12 | 0.56 | 0.64 | 0.03    | 0.67 | 0.19    |
| P value           | 0.08 | 0.38 | 0.65 | 0.20 | 0.90 | 0.51    | 0.36 | 0.46 | 0.84 | 0.10    | 0.91 | 0.23    |

| Own BMI            |      |      |      |      |      |         |      |      |      |         |      |         |
| <20 kg/m²          | 3.1  | 10.0 | 8.9  | 19.6 | 55.1 | 9.3     | 57.7 | 36.9 | 42.3 | 40.2    | 16.3 | 7.2     |
| 20–25 kg/m²        | 3.4  | 6.5  | 11.2 | 18.2 | 64.5 | 7.8     | 51.1 | 31.1 | 38.4 | 26.6    | 24.2 | 8.2     |
| >25–30 kg/m²       | 4.0  | 5.4  | 12.7 | 18.6 | 66.9 | 8.7     | 46.3 | 32.3 | 34.6 | 22.3    | 28.0 | 9.9     |
| >30 kg/m²          | 4.0  | 5.1  | 13.0 | 19.9 | 72.8 | 13.9    | 55.7 | 40.3 | 37.5 | 24.4    | 23.1 | 9.1     |
| P value            | <0.001 | <0.001 | 0.01 | 0.04 | <0.001 | <0.001 | <0.001 | <0.001 | 0.03 | <0.001   | <0.001 | <0.001 |
| P value           | <0.19 | <0.001 | 0.13 | 0.03 | <0.001 | <0.001 | 0.04 | 0.002 | <0.001 | <0.001   | 0.01 | <0.001 |

a1 The sample includes those with a valid measure of own BMI at age 44–45 y (total n = 9346). Total n varies between 9287 and 8029 because of missing data on lifestyle factors.

b High fried food consumption, ≥1 portion/d; low fruit consumption, <1 d/wk; high frequency of television viewing, ≥5 times/wk at 23 y and >4 h/d at 44–45 y; physical inactivity, none at 23 y and ≤2–3 times/mo at 42 y; smoking, current smoker; high alcohol consumption, ≥15/22 units/wk at 23 y and ≥21 drinks/wk at 44–45 y.

c P values from chi-square tests of association between parental or own BMI (excluding unknown category) and each of the lifestyle factors (by the categorizations described in Subjects and Methods); only the “unhealthiest” category is shown in the table for ease of presentation.

d P values from multinominal logistic regression models of the association between parental or own BMI (excluding unknown category) and each of the lifestyle factors (by the categorizations described in Subjects and Methods) with adjustment for sex, own occupational class, and educational level.
TABLE 4
Differences in mean offspring BMI (in kg/m²) at age 44–45 y by parental BMI in 1969 determined by using multiple linear regression models on 10 multiply imputed data sets with the sample with complete data on BMI at age 44–45 y (n = 9346; 4651 men and 4695 women)

<table>
<thead>
<tr>
<th>Maternal BMI</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Difference in mean offspring BMI at age 44–45 y (95% CI)</td>
<td>P value</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
</tr>
<tr>
<td>&lt;20 kg/m²</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>20–25 kg/m²</td>
<td>0.60 (−1.32, 0.12)</td>
<td>0.69 (−1.42, 0.04)</td>
<td>0.67 (−1.38, 0.04)</td>
<td>0.72 (−1.44, 0.001)</td>
</tr>
<tr>
<td>&gt;25–30 kg/m²</td>
<td>1.08 (0.80, 1.37)</td>
<td>1.07 (0.79, 1.35)</td>
<td>1.03 (0.75, 1.32)</td>
<td>1.03 (0.75, 1.31)</td>
</tr>
<tr>
<td>&gt;30 kg/m²</td>
<td>2.87 (2.27, 3.48)</td>
<td>2.75 (2.17, 3.34)</td>
<td>2.69 (2.08, 3.29)</td>
<td>2.63 (2.03, 3.22)</td>
</tr>
<tr>
<td>Per 1-kg/m² increase</td>
<td>0.28 (0.24, 0.33)</td>
<td>0.28 (0.23, 0.32)</td>
<td>0.27 (0.23, 0.32)</td>
<td>0.27 (0.23, 0.31)</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Paternal BMI</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
</tr>
<tr>
<td>&lt;20 kg/m²</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>20–25 kg/m²</td>
<td>0.24 (−1.12, 0.64)</td>
<td>0.15 (−1.01, 0.71)</td>
<td>0.30 (−1.17, 0.57)</td>
<td>0.14 (−1.00, 0.72)</td>
</tr>
<tr>
<td>&gt;25–30 kg/m²</td>
<td>1.22 (0.84, 1.60)</td>
<td>1.08 (0.70, 1.46)</td>
<td>1.10 (0.72, 1.48)</td>
<td>1.02 (0.64, 1.41)</td>
</tr>
<tr>
<td>&gt;30 kg/m²</td>
<td>2.50 (1.56, 3.45)</td>
<td>2.19 (1.32, 3.06)</td>
<td>2.16 (1.21, 3.11)</td>
<td>2.03 (1.15, 2.90)</td>
</tr>
<tr>
<td>Per 1-kg/m² increase</td>
<td>0.27 (0.21, 0.33)</td>
<td>0.24 (0.18, 0.29)</td>
<td>0.24 (0.18, 0.30)</td>
<td>0.22 (0.17, 0.28)</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

1 Adjusted for parental age.
2 Adjusted as for model 1 plus all lifestyle factors (fried food and fruit consumption at 33 and 42 y, television viewing at 23 and 44–45 y, physical activity at 23 and 42 y, smoking status at 23 and 42 y, and alcohol consumption at 23 and 44–45 y).
3 Adjusted as for model 1 plus markers of lifetime socioeconomic position (father’s and own occupational class and educational level).
4 Adjusted as for model 1 plus all lifestyle factors and markers of socioeconomic position.

Parental heights and weights were self-reported, and weight was reported in categories. Furthermore, paternal heights and weights were usually reported by mothers, which, along with our inability to assess levels of nonpaternity, led us to recommend that comparisons of the effects of maternal BMI with the effects of paternal BMI should not be made. However, these were prospective measurements, and offspring adiposity—which was also ascertained prospectively—was assessed at both time points on the basis of measures taken by nurses using a standardized protocol. We are also confident that results from analyses of offspring BMI at 11 y are not affected by puberty, because similar results were obtained for offspring BMI at 7 y (data not shown).

It has been shown that parents of the 1958 birth cohort had lower rates of mortality than the general population, which suggests that they may be a select healthy group (46). However, this difference in mortality may be explained by the lower mortality among parents than among nulliparous people, and the parents of the 1958 cohort members are likely to be representative of all parents in this generation (46). Attrition over time has reduced the original 1958 birth cohort sample and may have introduced bias. However, evidence suggests that the cohort followed up to age 44–45 y remains largely representative of the original sample (47). Furthermore, we were able to limit the effect of loss of individuals from analyses because of missing data on covariates using multiple imputation methods.

The potential selection biases affecting both parents and cohort members could influence the generalizability of findings, as could the fact that the prevalence of obesity was lower in parents in 1958 than it is in current generations of parents. However, we would still expect to find similar associations in more recent populations of parents and their offspring and may even expect that the effect of lifestyle factors would be greater in these populations exposed to environments, which are more obesogenic.

Conclusions

Associations between parental and offspring adiposity are strong and are maintained in midlife. These associations were not
greatly affected by adjustment for a wide range of lifestyle factors. Further work is needed to examine gene-environment interactions, which may help to elucidate an explanation for our findings. Whatever complex pathway underlies the associations found, it is clear that offspring of obese parents are an important target for interventions aimed at reducing population levels of overweight and obesity. Furthermore, these interventions need to be introduced at an early age, because once the associations between parental and offspring adiposity are established, they are likely to persist.

The authors’ responsibilities were as follows—RC, EH, and CP: conceived the idea for the study and developed the study objectives; RC and DB: analyzed the data; RC: drafted the manuscript; and EH, DB, and CP: commented on the draft of the manuscript and contributed to the final version. No conflicts of interest were reported.

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35. Lake AA, Mathers JC, Rugg-Gunn AJ, Adamson AJ. Longitudinal change in food habits between adolescence (11-12 years) and adulthood (32-33 years): the ASH30 study. J Public Health (Oxf) 2006;28:10–6.