Birth Weight and Lipids in a National Birth Cohort Study

Paula M.L. Skidmore, Rebecca J. Hardy, Diana J. Kuh, Claudia Langenberg, Michael E.J. Wadsworth

Objective—To investigate the association between birth weight and lipid levels in a 53-year-old birth cohort from England, Scotland, and Wales.

Methods and Results—Lipid levels were obtained from nonfasting blood samples, collected at the most recent follow-up of the MRC National Survey of Health and Development, for 2559 men and women. Regression models indicated that in men, a 1-kg increase in birth weight was associated with a 0.13-mmol/L decrease (95% CI: −0.23, −0.01) in total cholesterol at age 53 years (P=0.03), compared with a 0.02-mmol/L (95% CI: −0.11, 0.15) increase in women and a 0.06-mmol/L (95% CI: −0.15, 0.02) decrease in men and women combined. Adjustment for current height and body mass index (BMI) in men reduced the size of the relationship, with height being responsible for the reduction. Adult height and height at 2 and 4 years were significantly associated with total cholesterol in men and in men and women combined. The negative association between total cholesterol and birth weight was strongest among men with high BMI at age 53 years (P=0.03 for test for interaction between birth weight and BMI). There was no significant association between birth weight and LDL or HDL cholesterol in men or women before adjustment, but there was a positive association with HDL in women. When both sexes were analyzed together, an association was seen after adjustment for current body size. No confounding of these findings with social class was observed in this study.

Conclusions—Our results suggest that the small effect of birth weight on lipid levels at age 53 years has a limited public health impact. The findings suggest that childhood height growth may be more important than prenatal growth. (Arterioscler Thromb Vasc Biol. 2004;24:588-594.)

Key Words: birth weight • height • body mass index • cholesterol • epidemiology

High blood cholesterol is an important risk factor for cardiovascular disease and may be programmed in early life. According to Barker’s fetal origins hypothesis, impaired fetal growth that leads to changes in lipid metabolism is one explanation for the observed inverse associations between birth weight and other measures of size at birth and adult cardiovascular disease.

Results from studies relating markers of fetal growth to adult lipid levels have, however, been inconsistent and a recent meta-analysis estimated a decrease of only 0.05 mmol/L per 1 kg increase in birth weight. To our knowledge, all except one study of European adults have fewer than 800 subjects and may therefore have lacked the power to test for sex differences. Sex differences in the association between fetal growth and later coronary heart disease risk might be anticipated if fetal nutrition is the underlying mechanism because male fetuses grow at a faster rate, on average, than female fetuses. However, discussion of sex differences based on separate analyses for males and females, without statistical tests of interaction, may result in an overemphasis of chance differences. Adjustment of the birth weight effect by current body size is controversial, and correct interpretation relies on adjusted and unadjusted associations. Although most previous studies have adjusted for current weight, the majority have not adjusted for current height. Increased height has been consistently related to more favorable blood lipid levels, both in children and in adults. Findings have also rarely been adjusted for either childhood or adult social circumstances, which are inversely associated with cardiovascular disease. Forsdahl hypothesized that childhood nutritional deprivation followed by a more abundant diet in adulthood led to elevated blood cholesterol levels and thus raised cardiovascular risk. Results of studies relating childhood socio-economic conditions to adult lipid levels have been inconsistent. Whereas a consistent association between high lipid levels and lower socio-economic conditions has been found in women, the relationship in men has been less consistent.

Using data from the MRC National Survey of Health and Development (NSHD), we study the association between birth weight and lipids in a sample of 53-year-old British men and women followed-up since their birth in 1946. The sample is large enough to allow us to formally test for sex differences in the relationship between birth weight and lipid levels. We...
also adjust for the potential confounding factors of childhood and adult social class and test whether the effect of birth weight is confounded by current BMI and height.

Methods

Subjects and Study Design
The Medical Research Council’s National Survey of Health and Development (NSHD) is a prospective birth cohort study, consisting of a class-stratified sample (5362 births; 2547 women, 2815 men) of all births that occurred in the first week of March 1946 in England, Scotland, and Wales. Follow-up has included 21 contacts with the whole cohort from birth to the most recent, when survey members were age 53 years. At this most recent contact, 3035 participants (1472 men, 1563 women) provided information, and the majority (n=2989) were interviewed and examined in their own homes by research nurses. When it was inconvenient for the study member to have a home visit, participants completed a postal questionnaire (n=46). This corresponds to a participation rate of 70.5% among those residents in England, Wales, or Scotland and 89.6% for whom contact was attempted. Contact was not attempted for individuals who had previously refused to take part (n=648), were living abroad (n=583), were untraced since last contact at 43 years (n=266), or had already died (n=476). The responding sample at age 53 is in most respects representative of the national population of a similar age.30

Birth weights of cohort members, to the nearest quarter of a pound, were extracted from medical records within a few weeks of delivery and converted into kilograms. Heights and weights were measured at the 53-year interviews by research nurses according to a standardized protocol used at previous visits.31 Heights and weights were also measured during childhood. Body mass index (BMI), defined as weight/height,2 was calculated from these measurements. During these visits, nonfasting venous blood samples were taken and blood pressure was measured. Social class (categorized into nonmanual and manual) was defined in childhood using father’s occupation when the cohort member was age 4 years, and in adulthood based on cohort member’s occupation at age 53. The interview nurses recorded any information on participants’ current medication. This information was coded according to the British National Formula (BNF) Number 40 (2000). In this analysis, we used information on current use of lipid-regulating medication (using drug versus not using any drug from the BNF section 2.12). Because menopause status might influence lipid levels,32–34 women were divided into three categories (premenopausal, perimenopausal, and postmenopausal). These groups were based on the criteria used in the Massachusetts Women’s Health Study35 using data on menstrual characteristics collected annually in postal questionnaires to the women in the NSHD.36 Women who reported no menstrual bleeding in the past 12 months were classified as menopausal, those with no menstrual bleeding for 3 to 12 months or with less regular bleeding were classified as perimenopausal. Women who were using hormone replacement therapy or who had had a hysterectomy could not be allocated to any of these groups.

<table>
<thead>
<tr>
<th>TABLE 1. Characteristics of the Study Population</th>
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<tr>
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<tr>
<td>Men</td>
</tr>
<tr>
<td>Birth weight (kg) 1278</td>
</tr>
<tr>
<td>BMI (kg/m²) 1268</td>
</tr>
<tr>
<td>Total cholesterol (mmol/l) 1278</td>
</tr>
<tr>
<td>HDL (mmol/l) 1143</td>
</tr>
<tr>
<td>LDL (mmol/l) 1138</td>
</tr>
<tr>
<td>Women</td>
</tr>
<tr>
<td>Birth weight (kg) 1281</td>
</tr>
<tr>
<td>BMI (kg/m²) 1264</td>
</tr>
<tr>
<td>Total cholesterol (mmol/l) 1281</td>
</tr>
<tr>
<td>HDL (mmol/l) 1235</td>
</tr>
<tr>
<td>LDL (mmol/l) 1232</td>
</tr>
</tbody>
</table>

Laboratory Methods
Total cholesterol was measured by enzymatic CHOD–PAP. Precipitation for measurement of high-density lipoprotein (HDL) cholesterol was performed using phosphotungstic Mg²⁺; triglycerides were measured using a glycerol/kinase POD–linked reaction of glycerol liberated enzymatically from triglycerides. All of these measurements were made with a Bayer DAX-72. Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald formula: LDL cholesterol (mmol/L) = total cholesterol – HDL cholesterol – 0.45 × triglycerides.

Statistical Methods
Total cholesterol, HDL cholesterol, and LDL cholesterol were all normally distributed. Multiple linear regression models were used, first testing the unadjusted association between birth weight and each outcome measure, having tested for deviations from linearity. BMI at 53 years and a birth weight by BMI interaction term, to test whether the effect of birth weight on each lipid measure varied by current BMI, were then added to each model. These analyses were repeated with current height replacing BMI, and then with both BMI and height included. The possible influence of childhood growth was investigated as appropriate. We tested whether the effect of birth weight on lipids was confounded by social class at 4 years or at 53 years. Men and women were analyzed separately, as well as together. In models including both men and women, adjustment was made for sex and tests for interaction were performed to formally assess differences in the birth weight effect between sexes. Assessment of the association between birth weight and lipids among women, dependent on menopausal status, was also performed. Finally, all analyses were repeated excluding survey members who were using cholesterol-lowering drugs. All statistical analyses were performed using SPSS for Unix version 10 (SPSS Inc. Chicago, Ill).

Results
At age 53, 3035 subjects were followed-up (57% of the original cohort and 83% of the target sample). Birth weight and total cholesterol measures were available for 2559 subjects (1278 men and 1281 women) or 84% for whom contact was attempted. Mean birth weight of these 2559 was higher (3.40 kg) than for those not included (3.36 kg, P=0.03). Mean total cholesterol and HDL cholesterol were higher in women than in men, and there was little difference between the sexes for LDL cholesterol (Table 1).

Results from the unadjusted regression analysis indicated that a 1-kg increase in birth weight in men was associated with a decrease of 0.13 mmol/L (95% CI: −0.23, −0.01) in total cholesterol (P=0.03) (Table 2). There was no significant association between birth weight and LDL or HDL cholesterol. In women, there were no significant relationships between birth weight and any of the cholesterol measures. All relationships were linear. There were no significant associations when men and women were included in the same model (Table 2). The interaction term for birth weight and sex is of
Mean cholesterol levels for men as predicted from the regression model including birth weight (as a continuous variable), BMI (as a continuous variable), and the interaction between birth weight and BMI. The predicted relationship between birth weight and total cholesterol is presented for BMI values of 20, 25, and 30 m/kg².

borderline statistical significance ($P=0.08$) for total cholesterol but is not statistically significant for the other lipid measures.

There were only slight changes in the effect of birth weight in each case after adjustment for BMI only at age 53 years. In men, a 1-kg increase in birth weight was associated with a slightly greater decrease in total cholesterol after adjustment ($-0.15$ mmol/L per 1-kg birth weight; CI: $-0.26$, $-0.03$; $P=0.01$). An interaction between birth weight and current BMI was observed for total cholesterol among men ($P=0.03$). The negative association between birth weight and total cholesterol was strongest in men with high BMI at age 53 years (Figure 1). Adjustment of birth weight for height at 53 halved the size of the association between birth weight and total cholesterol in men ($-0.07$ mmol/L per 1 kg birth weight; CI: $-0.19$, $0.05$; $P=0.24$). There was no significant interaction between height at 53 years and birth weight. Adjustment for both BMI and height led to a 1-kg increase in birth weight being associated with a 0.10-mmol/L decrease in total cholesterol ($P=0.11$) (Table 2). In this model, a 1-cm increase in height was associated with a 0.01-mmol/L decrease in total cholesterol (CI: $-0.02$, $-0.01$; $P=0.003$), and a 1-kg/m² increase in BMI was associated with a 0.05-mmol/L increase (CI: $0.03$, $0.06$; $P<0.001$). The birth weight by BMI interaction remained significant. The addition of BMI and height to the model partly reduces the small birth weight effect in men and women combined and has little impact on the association in women (Table 2).

Adult height may capture the effects of childhood growth; hence, we investigated the influence of childhood height measures on total cholesterol and on the relationship between birth weight and total cholesterol (Table 3). Childhood height at 2 and 4 years were associated with total cholesterol for men and for men and women combined with increasing height being associated with decreasing total cholesterol. These height measures also weakened the effect of birth weight on total cholesterol in a similar way to that of adult height (Table 3).

Addition of height and BMI resulted in a slight strengthening of the positive effect of birth weight on HDL among men and women combined ($P=0.02$) and women only ($P=0.04$) (Table 2). The effect was largely caused by the BMI adjustment. In men and women combined, a 1-kg/m² increase in BMI was associated with a 0.03-mmol/L decrease in HDL (CI: $-0.04$, $-0.03$; $P=0.001$), and a 1-cm increase in height was associated with a 0.01-mmol/L decrease in HDL (CI: $-0.01$, $0.00$; $P=0.04$). There was a weak but significant ($P=0.04$) birth weight by BMI interaction in this model, in which the birth weight effect was strongest in those had low BMI at 53 years.

In all cases, there was no evidence of confounding of the birth weight effect with social class at 4 years and 53 years. Adult social class was not associated with any cholesterol measure. Social class in childhood was only associated with HDL in women, because those from a manual class had lower HDL cholesterol than did those from a nonmanual class.

### TABLE 2. Regression Analyses of Birth Weight and Lipids at 53 Years

<table>
<thead>
<tr>
<th>Regression Coefficient</th>
<th>95% CI</th>
<th>$P$</th>
<th>Regression Coefficient</th>
<th>95% CI</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td><strong>Adjusted for BMI and Height</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>$-0.13$</td>
<td>$-0.23$, $-0.01$</td>
<td>0.03</td>
<td>$-0.10$</td>
<td>$-0.21$, $0.02$</td>
</tr>
<tr>
<td>HDL</td>
<td>0.00</td>
<td>$-0.05$, 0.05</td>
<td>0.99</td>
<td>0.03</td>
<td>$-0.01$, 0.08</td>
</tr>
<tr>
<td>LDL</td>
<td>$-0.07$</td>
<td>$-0.18$, 0.03</td>
<td>0.17</td>
<td>$-0.07$</td>
<td>$-0.18$, 0.04</td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td><strong>Birth Weight</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>0.02</td>
<td>$-0.11$, 0.15</td>
<td>0.78</td>
<td>0.04</td>
<td>$-0.09$, 0.17</td>
</tr>
<tr>
<td>HDL</td>
<td>0.04</td>
<td>$-0.01$, 0.11</td>
<td>0.10</td>
<td>0.06</td>
<td>0.01, 0.11</td>
</tr>
<tr>
<td>LDL</td>
<td>0.04</td>
<td>$-0.08$, 0.16</td>
<td>0.50</td>
<td>0.05</td>
<td>$-0.07$, 0.17</td>
</tr>
<tr>
<td><strong>All Subjects</strong>*</td>
<td></td>
<td></td>
<td><strong>Interaction $P$ Value</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>$-0.06$</td>
<td>$-0.15$, 0.02</td>
<td>0.16</td>
<td>$-0.03$</td>
<td>$-0.11$, 0.05</td>
</tr>
<tr>
<td>HDL</td>
<td>0.02</td>
<td>$-0.01$, 0.06</td>
<td>0.23</td>
<td>0.04</td>
<td>0.01, 0.08</td>
</tr>
<tr>
<td>LDL</td>
<td>$-0.02$</td>
<td>$-0.10$, 0.06</td>
<td>0.61</td>
<td>$-0.01$</td>
<td>$-0.09$, 0.06</td>
</tr>
</tbody>
</table>

Unadjusted and adjusted for BMI (kg/m²) and Height (cm) at 53 years.

*Adjusted for sex.
TABLE 3. Regression Analyses of Birth Weight (BW) per Kilogram, Height (HT) per Centimeter, and Total Cholesterol at 53 Years

<table>
<thead>
<tr>
<th>n</th>
<th>Regression Coefficient</th>
<th>95% CI</th>
<th>P</th>
<th>Regression Coefficient</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td>Unadjusted</td>
<td>Adjusted</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1062 BW</td>
<td>−0.13</td>
<td>−0.30, 0.01</td>
<td>0.03</td>
<td>−0.12</td>
<td>−0.24, 0.01</td>
<td>0.06</td>
</tr>
<tr>
<td>1131 HT at 2</td>
<td>−0.01</td>
<td>−0.03, 0.00</td>
<td>0.03</td>
<td>−0.01</td>
<td>−0.02, 0.01</td>
<td>0.07</td>
</tr>
<tr>
<td>1268 BW</td>
<td>−0.13</td>
<td>−0.23, 0.01</td>
<td>0.03</td>
<td>−0.07</td>
<td>−0.19, 0.05</td>
<td>0.24</td>
</tr>
<tr>
<td>1268 HT at 53</td>
<td>−0.01</td>
<td>−0.03, 0.01</td>
<td>&lt;0.001</td>
<td>−0.02</td>
<td>−0.03, 0.01</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Women</td>
<td>Unadjusted</td>
<td>Adjusted</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1013 BW</td>
<td>−0.01</td>
<td>−0.15, 0.12</td>
<td>0.86</td>
<td>−0.003</td>
<td>−0.14, 0.13</td>
<td>0.96</td>
</tr>
<tr>
<td>1115 HT at 2</td>
<td>−0.01</td>
<td>−0.02, 0.01</td>
<td>0.25</td>
<td>−0.009</td>
<td>−0.02, 0.01</td>
<td>0.25</td>
</tr>
<tr>
<td>1263 BW</td>
<td>0.04</td>
<td>−0.10, 0.18</td>
<td>0.57</td>
<td>0.06</td>
<td>−0.08, 0.12</td>
<td>0.41</td>
</tr>
<tr>
<td>1263 HT at 53</td>
<td>−0.01</td>
<td>−0.02, 0.01</td>
<td>0.21</td>
<td>−0.01</td>
<td>−0.02, 0.01</td>
<td>0.16</td>
</tr>
<tr>
<td>All subjects</td>
<td>Unadjusted</td>
<td>Adjusted</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2075 BW</td>
<td>−0.09</td>
<td>−0.18, 0.00</td>
<td>0.05</td>
<td>−0.08</td>
<td>−0.17, 0.02</td>
<td>0.11</td>
</tr>
<tr>
<td>2246 HT at 2</td>
<td>−0.01</td>
<td>−0.02, 0.01</td>
<td>0.01</td>
<td>−0.01</td>
<td>−0.02, 0.01</td>
<td>0.02</td>
</tr>
<tr>
<td>2532 BW</td>
<td>−0.07</td>
<td>−0.16, 0.02</td>
<td>0.14</td>
<td>−0.04</td>
<td>−0.13, 0.05</td>
<td>0.41</td>
</tr>
<tr>
<td>2532 HT at 53</td>
<td>−0.01</td>
<td>−0.02, 0.01</td>
<td>0.004</td>
<td>−0.01</td>
<td>−0.02, 0.01</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Three adjusted models (the first containing birth weight and height at 2 years, the second containing birth weight and height at 4 years, and the third containing birth weight and adult height) are compared with the unadjusted regression coefficients based on the same sample.

There were no significant relationships between birth weight and lipid levels among women in premenopausal, perimenopausal, or postmenopausal groups. However, the relationship between birth weight and total cholesterol in postmenopausal women was negative, whereas the relationship in premenopausal and perimenopausal women was positive.

There were 49 (3.8%) men and 22 (1.7%) women using cholesterol-lowering drugs. When analyses were repeated, excluding those using cholesterol-lowering drugs, the findings remained essentially unchanged.

**Discussion**

We found a statistically significant inverse relationship between birth weight and total cholesterol levels in men, both before and after adjustment for current BMI, which was reduced after the addition of adult height. This association was strongest in those with high current BMI. We found no such association in women, and there was some evidence of a sex difference in the size of the birth weight effect \( (P=0.08\) test for interaction\). No unadjusted associations between birth weight and HDL or LDL cholesterol were observed. A marginally significant positive relationship with HDL cholesterol emerged in the analysis considering men and women combined and women on their own after adjustment for BMI and height, although the size of these associations were only slightly larger than the unadjusted effects.

Interpretation of the birth weight effect after adjustment for current body size is controversial,\(^{57,38}\) and results may be indicative that postnatal change in size, rather than fetal growth, is important.\(^{18}\) The unadjusted association better represents the total effect of birth weight on the outcome of interest. Our findings would therefore suggest that only total cholesterol might be influenced by fetal growth. The potential underlying mechanism linking small birth size with adverse lipid levels in adult life remains unclear. Barker et al\(^{13}\) observed that small abdominal circumference (a measure not recorded in the NSHD), rather than low birth weight, was associated with higher levels of total and LDL cholesterol. They suggested that small abdominal circumference represented small liver size, and as the liver regulates lipid metabolism, impaired in utero growth of the liver may program more adverse levels of total and LDL cholesterol. Others have postulated that genetic factors, particularly those influencing insulin resistance, may explain the association between birth weight and cardiovascular risk.\(^{39}\) Low levels of HDL and high levels of triglycerides are components of the insulin-resistance syndrome.\(^{40}\) It is therefore possible that the mechanism underlying the association of birth weight with HDL cholesterol and triglycerides may be different than that underlying the association with total and LDL cholesterol.

The reduction of the effect of birth weight on total cholesterol after adjustment for adult and childhood height
and the effect of the heights themselves suggest that poor childhood growth in height may be more important in determining cholesterol than poor prenatal growth. The interaction between birth weight and BMI suggests the importance of accelerated postnatal BMI change. Nevertheless, it remains unclear whether this accelerated growth at a sensitive period of the life course or at any stage is detrimental to those born small. The detrimental impact of greater gain in weight relative to height during the life-course is also implicated in the findings for HDL, with the strengthening of the positive association after adjustment for current BMI.

Relationships between total cholesterol and birth weight have previously been found in subjects with nonfasting levels, whereas significant relationships with other lipids were generally only seen in studies using fasting lipid levels. The use of nonfasting measures in the NSHD may explain our lack of association of birth weight with LDL and HDL cholesterol. Nonfasting lipid measures were used because of the large number of subjects and the time constraints, which meant that blood samples were taken at varying times of the day. Some samples were taken in the late afternoon, meaning that fasting was not feasible. Nonfasting triglyceride, but not total cholesterol levels, have been found to vary significantly throughout the day. Nonfasting levels of LDL cholesterol have been found to be lower, and nonfasting levels of triglycerides are higher than fasting levels, but there were no differences in total cholesterol levels or HDL cholesterol. As triglyceride levels vary throughout the day, we have not included them in the analysis. Because LDL cholesterol was calculated using the Friedewald formula, which includes triglyceride, we must view the results for this measure with caution. The mean value for total cholesterol in our sample was similar to fasting mean levels in the Dietary and Nutritional Survey of British Adults and those in the Caerphilly study.

This is the first large birth cohort study to our knowledge to consider the association between birth weight and lipids in older adults. Our unadjusted estimate combined for both sexes is similar to that found in a recent meta-analysis of the association between birth weight and total cholesterol in which an increase in 1 kg of birth weight was associated with a 0.05-mmol/L reduction in total cholesterol. Some of the previous studies in smaller European samples of a similar age have reported a significant negative association between birth weight and total cholesterol, consistent with our unadjusted findings in men. Others report no association, and one study showed a significant negative association after adjustment for age, smoking, alcohol, and BMI. None of these studies adjusted for adult height. No study found an association with LDL cholesterol and some have found an association with HDL and HDL cholesterol, although some studies of adults found significant relationships when men and women were analyzed together, when males and females were analyzed separately in these same studies significant relationships were only seen in men. These agree with our findings of a possible sex difference in effect in total cholesterol, and studies that have analyzed men and women together may have obscured a relationship in men. However, evidence in our study for a sex difference was relatively weak and may represent a chance finding, while the small size of the effect, even among the men, means that it is likely to be of limited public health importance. A recent meta-analysis found no evidence of a sex difference.

Only a few studies have reported testing for an interaction between birth weight and current body size. None found evidence of any such effect modification, although all but one probably lacked the necessary power. Our finding that the effect of birth weight on total cholesterol in men was strongest in those who subsequently became large in BMI is consistent with results in relation to other cardiovascular risk factors, such as blood pressure. In contrast, the effect of birth weight on HDL cholesterol was strongest in those of low BMI, but this interaction was weaker. The difference may be a result of different underlying mechanisms.

Lipoprotein levels have been found to be more adverse in postmenopausal than in premenopausal women, with total cholesterol levels increasing at menopause, whether it occurs naturally or because of hysterectomy, with or without bilateral oophorectomy. We found that the relationship between total cholesterol and birth weight was negative in postmenopausal women and positive in premenopausal and perimenopausal women. Comparisons among subgroups must be performed with extreme caution, and although these analyses lacked statistical power and the results were nonsignificant, the findings could suggest that the pattern of the relationship between birth weight and lipids may become more like that in men as women age. The NSHD will be able to test this with future follow-ups. The only study to date in postmenopausal women included 297 women aged 60 to 71 years and found a positive association between birth weight and HDL cholesterol, but no association with total or LDL cholesterol or triglycerides. We see a small positive association with HDL in the total sample of women consistent with this study.

In conclusion, birth weight was inversely related to total cholesterol levels in men, but not women, independently of social class in either childhood or adulthood. The small size of the association, however, means that it may have little importance in public health terms. Adjustment for current and childhood heights resulted in a weakening of this association, and adjustment for current BMI strengthened the positive association between birth weight and HDL when both sexes were analyzed together. There was also evidence that the negative association between birth weight and total cholesterol in men was stronger in those with higher BMI. These findings, together with the stronger association of lipid outcomes with childhood and adult heights and adult BMI, suggest that postnatal growth may be more important than poor prenatal growth.

Acknowledgments

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References


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