Maternal and Social Origins of Hypertension

David J.P. Barker, Clive Osmond, Tom J. Forsen, Eero Kajantie, Johan G. Eriksson

Abstract—We previously reported that in 2003 people from the Helsinki birth cohort whose blood pressures were measured, 2 different paths of growth preceded the development of hypertension. People already diagnosed with hypertension were small at birth but of average body size at age 11 years. People newly diagnosed with hypertension grew slowly in utero and through childhood. We have now examined how the mother’s body size, placental size, and living conditions after birth, 3 influences that affect growth, affect hypertension. Diagnosed hypertension was associated with low placental weight and poor living conditions after birth. The odds ratios were 1.6 (95% CI, 1.1 to 2.3) in people with placental weights <550 g, compared with those with weights >750 g, and 2.2 (95% CI, 1.5 to 3.3) in people whose fathers were laborers compared with those in upper middle-class families. Newly diagnosed hypertension was associated with a small anteroposterior diameter of the mother’s bony pelvis, a known consequence of rickets or lesser degrees of malnutrition in infancy. The odds ratio was 2.2 (95% CI, 1.4 to 3.5) in people whose mothers’ pelvic external conjugate diameters were <18 cm when compared with people whose mothers’ diameters were ≥19 cm. We conclude that one path of growth that leads to hypertension is initiated by fetal undernutrition, which may make a baby vulnerable to postnatal stress, whereas the other originates in a functional incapacity in the mother’s metabolism, possibly protein metabolism, which she acquired through undernutrition during her infancy. (Hypertension. 2007;50:565-571.)

Key Words: hypertension ■ fetal programming ■ early growth ■ placenta ■ protein metabolism

We reported how prenatal and postnatal growth patterns are linked to the later occurrence of hypertension in a clinical study of 2003 people age 62 years from the Helsinki birth cohort. We found that 2 different paths of growth preceded the development of hypertension. Subjects previously diagnosed with hypertension had small body size at birth and low weight gain from birth to 2 years but grew rapidly after the age of 2. At age 11 years, their body size was about average. As adults, they tended to be obese and insulin resistant. A second group of people had not been previously diagnosed, but their blood pressures were classified as hypertensive under current definitions. They were short at birth, had low weight gain from birth to 2 years, and remained small after the age of 2. At age 11 years, they were short and thin. As adults, they tended to have atherogenic lipid profiles. We suggested that the 2 paths of growth led to hypertension through different biologic mechanisms. In this cohort, the first path of growth was associated with coronary heart disease, whereas the second was associated with stroke.

All residents in Finland have access to publicly funded primary health care. The state may reimburse the costs of medication for hypertension, but this is subject to the approval of a physician and is intended for patients with persisting, severe hypertension or those with complications of the disease or with other disorders, such as type 2 diabetes. These people tend to be obese. Consistent with this, the subjects in our study who had already been diagnosed as hypertensive had blood pressure levels similar to those who were newly diagnosed, although the former were receiving medication. This suggests that they had more severe hypertension. Thirty-nine percent of them were obese, with a body mass index >30 kg/m², compared with 24% of those with newly diagnosed hypertension and 11% of normotensive subjects.

We have now examined how the mother’s body size, placental size, and living conditions after birth, 3 influences known to affect early growth, affect the later development of hypertension. We have previously shown that, in this cohort, coronary heart disease was associated with low placental weight and with poor living conditions after birth, as indicated by low paternal social class. Stroke was associated with small maternal body size, specifically with a small external conjugate diameter of the bony pelvis. This diameter was routinely measured in pregnant women in the past. A diameter <18 cm defined a “flat” pelvis, the result of rickets or lesser degrees of malnutrition in early childhood.

Our hypotheses were that the path of growth that leads to diagnosed hypertension is associated with low placental weight and poor living conditions after birth, whereas the path that leads to newly diagnosed hypertension is associated with a small maternal external conjugate diameter.
TABLE 1. Mean and SD of Maternal Characteristics and Childhood Living Conditions in Normotensive and Hypertensive Men and Women

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Normotension (n=557)</th>
<th>Newly Diagnosed Hypertension (n=802)</th>
<th>Previously Diagnosed Hypertension (n=644)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height, cm</td>
<td>1779</td>
<td>160.2 5.9</td>
<td>159.1 5.9</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>1762</td>
<td>68.5 8.1</td>
<td>67.0 8.0</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>1759</td>
<td>26.7 2.8</td>
<td>26.5 2.9</td>
</tr>
<tr>
<td>Pelvic external conjugate diameter, cm</td>
<td>1089</td>
<td>19.6 1.0</td>
<td>19.3 1.1</td>
</tr>
<tr>
<td>Percentage external conjugate diameter &lt;18 cm</td>
<td>1089</td>
<td>16.8 ...</td>
<td>28.2 ... 0.0009</td>
</tr>
<tr>
<td>Intercristal diameter, cm</td>
<td>1087</td>
<td>28.5 1.5</td>
<td>28.3 1.5</td>
</tr>
<tr>
<td>Interspinous diameter, cm</td>
<td>1089</td>
<td>26.1 1.5</td>
<td>25.8 1.5</td>
</tr>
<tr>
<td>Age, y</td>
<td>2003</td>
<td>29.0 5.4</td>
<td>28.7 5.5</td>
</tr>
<tr>
<td>Parity</td>
<td>2003</td>
<td>2.0 1.3</td>
<td>1.9 1.3</td>
</tr>
<tr>
<td>Placenta</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight, g</td>
<td>2001</td>
<td>653 123</td>
<td>655 123 0.9</td>
</tr>
<tr>
<td>Percentage of fathers as laborers</td>
<td>1918</td>
<td>66 ... 0.3</td>
<td>68 ... 0.3 0.002</td>
</tr>
<tr>
<td>No. of rooms in home</td>
<td>1463</td>
<td>1.8 0.9</td>
<td>1.9 0.8 0.6</td>
</tr>
<tr>
<td>No. of people in home</td>
<td>1302</td>
<td>3.9 1.2</td>
<td>3.9 1.1 0.5</td>
</tr>
</tbody>
</table>

$P_1$, $P$ for comparison of newly diagnosed hypertensives against normotensives, adjusted for age and sex; $P_2$, $P$ for previously diagnosed hypertensives against normotensives; $P_3$, $P$ for previously diagnosed against newly diagnosed hypertensives.

Subjects and Methods

The cohort comprised men and women who were born in Helsinki University Central Hospital from 1934 to 1944 and attended child welfare clinics in the city. Most of them also went to school in the city. Details of the birth records, child welfare clinic records, and school health records have been previously described.1,2 The birth records included weight of the placenta. We identified 8760 men and women who were living in Finland in 1971, when a unique identification number was allocated to each member of the Finnish population. We used random-number tables to select a subset of 2003 people within the cohort who were still alive and living in Helsinki.2 They attended a clinic at the National Public Health Institute in Helsinki.2 The procedures used at the clinic have been described.1,2 Six hundred forty-four of the men and women had been measured.6 Mothers whose pelvises were measured tended to be younger than the mothers of the normotensives, and a higher percentage of them had diameters less than 18 cm (7 in.) was used as a marker of a flat pelvis, which increased the risk of obstructed labor.6 Mothers whose pelvises were measured tended to be younger and of lower social class, and more of them were primiparous. The external conjugate diameter is the distance between the spine of the fifth lumbar vertebra and the front of the pubic bone.6 A diameter of less than 18 cm (7 in.) was used as a marker of a flat pelvis, which increased the risk of obstructed labor.6 The intercristal distance is the maximal distance between the iliac crests, whereas the interspinous distance is the distance between the anterosuperior iliac spines. The intercristal distance is usually 2.5 cm (1 in.) longer than the interspinous distance, but in a flat pelvis the 2 diameters may be similar.

Birth records included data on the fathers’ occupations, which were grouped into upper and lower middle-class and laborers, based on classification from Statistics Finland. The child welfare clinic record included data on the numbers of rooms and of people in the home. The men’s and women’s own occupations, recorded at the 1980 census, were obtained through Statistics Finland, which grouped them into 4 classes: higher official, lower official, self-employed, and laborer.5 Written, informed consent was obtained from each subject before any procedures were carried out. The ethics committee at the National Public Health Institute, Finland, approved the study.

Statistical Analysis

We used multiple linear and logistic regression analyses, adjusted for age and sex, to examine the effects of maternal body size, placental size, and paternal social class on hypertension.

Results

Subject Data

The mean age of the subjects was 62 years (range 57 to 70 years). They were grouped as normotensive, newly diagnosed hypertensive, and previously diagnosed hypertensive.1

Maternal Body Size

Table 1 shows the characteristics of the mothers of the subjects and indices of living conditions in childhood. The mothers of both groups of hypertensives were shorter and lighter than the mothers of the normotensives, although their mean body mass indices were similar. The mothers of the newly diagnosed hypertensives had lower mean pelvic external conjugate, intercristal, and interspinous diameters than the normotensives, and a higher percentage of them had an external conjugate diameter <18 cm. There were no similar associations with previously diagnosed hypertension. Comparing the 2 groups of hypertensive subjects, those who were newly diagnosed had significantly lower mean external conjugate diameters, and a higher percentage had diameters less...
circumference in adult life, \(1\) we present probability values
strong association between hypertension and a high waist
height and pelvic external conjugate diameter. Because of the
the 3 groups.

Table 2 shows the trends in hypertension with maternal
height and pelvic external conjugate diameter. Because of the
strong association between hypertension and a high waist
circumference. The odds ratios (ORs) for both previously and newly
diagnosed hypertension fell with increasing maternal height. Newly
diagnosed hypertension, however, was also associated with the external
conjugate diameter, the ORs falling as the diameter increased. In a simultaneous
regression of the effects of height and external conjugate on newly
diagnosed hypertension, only the trend with external conjugate diameter
remained statistically significant, the probability values being
0.09 and 0.009, respectively. The trend with external conjugate diameter was matched by similar, though weaker, trends with the other 2 pelvic diameters,

Table 2 shows the trends in hypertension according to maternal
height and pelvic external conjugate diameter. Because of the
the mothers’ pelvic exter-

We used data from the entire cohort of 8760 people to
examine the associations between the mothers’ pelvic external
conjugate diameters and the growth of their children. A small
diameter was associated with reduced linear growth before and after birth. The children of mothers who had an external conjugate diameter \(<18\) cm were 0.5 cm (95% CI, 0.4 to 0.6) shorter at birth and 2.3 cm (95% CI, 1.9 to 2.7)
shorter at age 11 years. In a simultaneous regression with
maternal height, the effects of external conjugate diameter on the child’s height remained statistically significant (\(P<0.0001\)
for height at 11 years of age). Within the 2003 subjects, we
examined the simultaneous effect of the mother’s external
conjugate diameter and the child’s body size at 2 years. With
inclusion of the external conjugate diameter in the regression,
neither height nor weight at 2 years was related to newly
diagnosed hypertension.

Because in the past rickets was a seasonal disorder, we
used data from the entire cohort to examine associations between
the mother’s external conjugate diameter and her month of birth. We found that among mothers born in
December or January, the mean diameter was 0.3 cm less than
that among those born in the remaining months. There was little variation among the diameters of mothers born in the other months. Thirty-two percent of mothers born during
December and January had external conjugate diameters <18
cm compared with 20% among mothers born in the remaining
months (\(P=0.0005\)). There were no similar trends in the
mother’s height with month of birth.

Placental Size
Table 1 shows that the mean placental weight of people with
previously diagnosed hypertension was lower than that of normotensives and newly diagnosed hypertensives. Table 3 shows that the ORs for previously diagnosed hypertension fell with increasing placental weight. In a simultaneous
regression, both short maternal height and low placental
weight were associated with previously diagnosed hypertension (\(P=0.02\) and 0.002, respectively).

Childhood Living Conditions
Table 1 shows that more of the previously diagnosed hyper-
tensives than the normotensives or newly diagnosed hypertensives were born into families of laborers. The number of
rooms in the home and the number of people were similar in the
hypertensive and normotensive groups. Table 4 shows the
trends in hypertension according to paternal social class. The ORs for previously diagnosed hypertension rose between
people born into the upper middle class and those born into

than 18 cm (Table 1). Maternal age and parity were similar in
the 3 groups.

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strong association between hypertension and a high waist
circumference in adult life, \(1\) we present probability values
both adjusted and unadjusted for waist circumference. The

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### Table 2: ORs (95% CIs) for Hypertension According to Maternal Height and Pelvic External Conjugate Diameter

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Newly Diagnosed Hypertension (n=602)</th>
<th>Previously Diagnosed Hypertension (n=644)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal height, cm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-155</td>
<td>0.97 (0.95 to 0.99), P=0.002</td>
<td>0.97 (0.95 to 1.00), P=0.02</td>
</tr>
<tr>
<td>-158</td>
<td>0.97 (0.95 to 0.99), P=0.0007</td>
<td>0.97 (0.95 to 0.99), P=0.008</td>
</tr>
<tr>
<td>-165</td>
<td>0.97 (0.95 to 0.99), P=0.0007</td>
<td>0.97 (0.95 to 0.99), P=0.008</td>
</tr>
<tr>
<td>Maternal pelvic external conjugate diameter, cm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-18</td>
<td>0.79 (0.68 to 0.91), P=0.0001</td>
<td>0.94 (0.80 to 1.10), P=0.4</td>
</tr>
<tr>
<td>-19</td>
<td>0.76 (0.66 to 0.89), P=0.0004</td>
<td>0.89 (0.75 to 1.05), P=0.2</td>
</tr>
</tbody>
</table>

*Adjusted for age and sex.
†Adjusted for age, sex, and adult waist circumference.
the families of laborers. There was no similar trend in newly
diagnosed hypertension. After allowing for paternal social
class, the subject’s social class in adult life had no effect on
previously diagnosed hypertension.

We examined the combined effects of low social class and
low placental weight on previously diagnosed hypertension.
In a simultaneous regression, both had statistically significant
effects (P = 0.0002 and 0.006, respectively), which are shown
in Table 5. The effects of paternal social class were confined
to people whose placental weight was ≥ 650 g, whereas
placental weight only had an effect among people whose
fathers were laborers (P for interaction = 0.02). There was no
similar interaction with birth weight (P = 0.2).

We used data from the entire cohort to examine associa-
tions between paternal social class and offspring growth. Low
social class was not associated with any measure of body size
at birth or with placental weight; but it was associated with
reduced height during infancy. At 2 years of age, the mean
height of the children of laborers was 85.9 cm compared with
86.5 and 86.2 cm in children from upper and lower middle-
class families. In a simultaneous regression within the 2003
subjects in our study, both low paternal social class and low
weight at 2 years increased the risk of previously diagnosed
hypertension, P = 0.0004 and 0.0002, respectively.

Discussion
In a clinical study of men and women age ≈ 62 years, we have
shown that 2 different patterns of growth preceded the
development of hypertension. People already diagnosed with
hypertension were small at birth but of average body size at
age 11 years. People newly diagnosed with hypertension
grew slowly in utero and through childhood. We have now
examined how maternal body size, placental size, and child-
hood living conditions, 3 influences that affect growth, are
related to later hypertension.

Maternal Body Size
The mothers of people in either group of hypertensives
tended to be short in stature. ORs for hypertension fell with
increasing maternal height. The mothers of people with newly
diagnosed hypertension also had small pelvic bones, indi-
cated by a reduction in 3 standard diameters, measured
externally. In the past these measurements were used in
obstetrics to assess the likelihood of obstructed labor.6 Small
pelvic bones are a persisting consequence of undernutrition
during infancy, in particular, lack of vitamin D.7–11 This
softens the bones and reduces all pelvic dimensions, but its
greatest effect is on the external conjugate diameter. When an
undernourished infant with soft bones begins to stand, the
forces on the bony pelvis tend to flatten it from front to back.
The external conjugate diameter, measured as the distance
between the front of the pubic bone and the spine of the fifth
lumbar vertebra, is reduced, as is seen in rickets.7 This
reduced diameter, in relation to other diameters, persists into
adult life and increases the risk of obstructed labor,6 because
the anteroposterior shape of the lower hip is not remodelled
during childhood and adolescent growth. A reduced external
conjugate diameter is the principal marker of a flat pelvis. A

<table>
<thead>
<tr>
<th>Placental weight, g</th>
<th>Newly Diagnosed Hypertension (n=802)</th>
<th>Previously Diagnosed Hypertension (n=644)</th>
</tr>
</thead>
<tbody>
<tr>
<td>−550</td>
<td>475</td>
<td>1.0 (0.7 to 1.4)</td>
</tr>
<tr>
<td>−650</td>
<td>656</td>
<td>1.2 (0.8 to 1.6)</td>
</tr>
<tr>
<td>−750</td>
<td>524</td>
<td>1.3 (0.9 to 1.8)</td>
</tr>
<tr>
<td>&gt;750</td>
<td>346</td>
<td>1.0 (Baseline)</td>
</tr>
</tbody>
</table>

OR (95% CI)* per 100 g:

<table>
<thead>
<tr>
<th></th>
<th>Newly Diagnosed Hypertension (n=802)</th>
<th>Previously Diagnosed Hypertension (n=644)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TABLE 3. ORs (95% CIs) for Hypertension According to Placental Weight

<table>
<thead>
<tr>
<th>Father’s social class</th>
<th>Newly Diagnosed Hypertension (n=802)</th>
<th>Previously Diagnosed Hypertension (n=644)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper middle</td>
<td>213</td>
<td>1.0 (Baseline)</td>
</tr>
<tr>
<td>Lower middle</td>
<td>375</td>
<td>1.4 (1.0 to 2.1)</td>
</tr>
<tr>
<td>Laborer</td>
<td>1299</td>
<td>1.3 (0.9 to 1.8)</td>
</tr>
</tbody>
</table>

OR (95% CI)* per class:

<table>
<thead>
<tr>
<th></th>
<th>Newly Diagnosed Hypertension (n=802)</th>
<th>Previously Diagnosed Hypertension (n=644)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TABLE 4. ORs (95% CIs) for Hypertension According to Paternal Social Class

*Adjusted for age and sex.
†Adjusted for age, sex, and adult waist circumference.
diameter of less than 18 cm (7 in.) is diagnostic. We found that the external conjugate diameter was more strongly associated with newly diagnosed hypertension than the other 2 diameters, whose relative size changes through childhood. After allowing for the external conjugate diameter, neither short maternal stature nor the other 2 diameters had an effect on newly diagnosed hypertension. These findings point to an effect of undernutrition and poor growth during the mothers’ infancy rather than later in childhood.

In the early years of the last century, when vitamin D deficiency and rickets were widespread across Europe and the United States, its peak incidence was in the winter months. This was more apparent in northern countries such as Finland, where active rickets was common in winter but rare in the summer months. Consistent with this, we found that a higher percentage of mothers born in December and January had small external conjugate diameters. Although bone deformities may not become apparent until around 6 months of age, the disturbances in calcium metabolism that lead to rickets begin soon after birth.

Lesser levels of infant malnutrition than those that lead to rickets can lead to flattening of the pelvis. Radiologic surveys of young women, carried out on both side of the Atlantic during the 1930s and 1940s, showed that minor degrees of anteroposterior flattening of the pelvic inlet were common. The pelvic inlets of women born into poor socioeconomic conditions were generally flatter than those of women born into better circumstances, which tended to be round. This was attributed to their worse nutrition during infancy. Three studies, one in this cohort, one in an older Helsinki cohort, and the other in Sheffield, UK, have shown a link between flat maternal pelvis and increased stroke risk in their offspring. These observations led to the hypothesis that stroke originatates through malnutrition in young girls, which leads to impaired development of their offspring. One suggested mechanism is impaired vascular development, especially in the brain during its period of rapid growth before and after birth. Two studies support this concept. In Holland, men and women whose mothers had small external conjugate diameters had higher systolic pressures and narrower common carotid arteries. In South India, men and women whose mothers had small external conjugate diameters had stiffer, less compliant arteries in the arms and legs.

In our study, children whose mothers had a small external conjugate diameter grew slowly in utero and through childhood. This pattern of growth led to newly diagnosed hypertension and to stroke in this cohort. It was not caused by poor living conditions after birth, as measured by low paternal social class, which was unrelated to newly diagnosed hypertension. After allowing for maternal external conjugate diameter, the child’s body size at 2 years was no longer related to later hypertension. One possible explanation is that the children had acquired, in utero, a reduced capacity for growth as a result of their mothers’ metabolic incapacity. The babies of mothers who have low rates of protein synthesis in pregnancy are short at birth. A hypothesis to explain our findings is that malnutrition during infancy, including a lack of vitamin D, leads to persisting changes in protein metabolism that prejudice the early nutrition and growth of the next generation and lead, in adult life, to an atherogenic lipid profile, hypertension, and stroke.

### Placental Size

In contrast to newly diagnosed hypertension, previously diagnosed hypertension was related to low placental weight. This is consistent with the association between low placental weight and coronary heart disease in this cohort. Associations between low placental weight and raised blood pressure have been reported in other studies. In a study of an older birth cohort in Helsinki, which was based on the records of people being reimbursed for the costs of medication rather than on clinical observations, those receiving medication for both hypertension and diabetes tended to have low placental weights. These findings suggest that the form of hypertension that is associated with insulin resistance and coronary heart disease may originate in fetal undernutrition as a consequence of impaired placental development.

### Childhood Living Conditions

Previously diagnosed hypertension was associated with poor living conditions in childhood, as indicated by the father working as a laborer. After allowing for paternal social class, the subject’s own social class in adult life had no effect on previously diagnosed hypertension. We found that the effect of poor living conditions on the risk of hypertension was confined to people with placental weights ≤650 g (Table 5). Conversely, low placental weight increased the risk of hyper-

<table>
<thead>
<tr>
<th>Placental weight, g</th>
<th>Prevalence No.</th>
<th>Prevalence No.</th>
<th>Prevalence No.</th>
<th>P for Trend*</th>
</tr>
</thead>
<tbody>
<tr>
<td>−550</td>
<td>19</td>
<td>69</td>
<td>34</td>
<td>43</td>
</tr>
<tr>
<td>−650</td>
<td>20</td>
<td>69</td>
<td>28</td>
<td>117</td>
</tr>
<tr>
<td>−750</td>
<td>29</td>
<td>55</td>
<td>31</td>
<td>102</td>
</tr>
<tr>
<td>&gt;750</td>
<td>24</td>
<td>42</td>
<td>30</td>
<td>61</td>
</tr>
</tbody>
</table>

*Adjusted for age and sex.
tension only in people who had poor childhood living conditions. There was no similar interaction between the effects of low birth weight and poor childhood living conditions. Poor living conditions slow infant growth through poor nutrition and recurrent infective illnesses.\textsuperscript{23} We found, however, that low social class increased the risk of previously diagnosed hypertension, independently of its adverse effects on infant growth. One possible explanation for our findings is that fetal undernutrition makes a baby vulnerable to the postnatal stress that accompanies poor living conditions. Raised blood pressure may be a persisting consequence of vulnerability to stress in prenatal, perinatal, or postnatal life.\textsuperscript{24,25}

Limitations of the Study
We previously discussed possible limitations of our study.\textsuperscript{1,2} It was restricted to people who had attended child welfare clinics. Although the majority of children attended these clinics, which were free, attendance was voluntary. The people in our study may not be representative of all people now living in Helsinki, although at birth their social class distribution was similar to that in the city as a whole. Only about half of the mothers had measurements of the bony pelvis, and these mothers tended to be younger, less parous, and of lower social class than other mothers. None of these variables was related to newly diagnosed hypertension. The path of growth that led to previously diagnosed hypertension led to coronary heart disease but not stroke. This could reflect selective benefits of antihypertensive medication on stroke. Such an argument would not explain why the path of growth that led to newly diagnosed hypertension was associated with stroke but not coronary heart disease.

Public Health Implications
The findings in this cohort have implications for the prevention of cardiovascular disease. Hypertension and insulin resistance and coronary heart disease are associated with low placental weight and poor living conditions in childhood. Primary prevention of these disorders may depend on improving fetal nutrition and protecting the nutrition and growth of infants. Hypertension and an atherogenic lipid profile, and stroke, are associated with a small external conjugate diameter in the mothers. Prevention of these disorders may ultimately depend on improving the nutrition and growth of infant girls. Policies based on the prevention of hypertension by improving the nutrition of girls and young women are preferable to policies based on treatment only. Improved nutrition not only will benefit the present population but also may reduce disease in future generations.

Perspectives
In a clinical study within the Helsinki birth cohort, we previously described 2 different patterns of growth that preceded the development of hypertension. In one, which was associated with more severe hypertension in people who tended to be obese and insulin resistant, small body size at birth and during infancy were followed by rapid growth. This path of growth also led to coronary heart disease. We now report that it is associated with low placental weight and may therefore be initiated by fetal undernutrition. Low placental weight only increased the risk of later hypertension if it was followed by poor living conditions in childhood. This could be because fetal undernutrition makes a baby vulnerable to postnatal stress. In the other path of growth, which was associated with less severe hypertension in people who had atherogenic lipid profiles, slow linear growth in utero and during infancy was followed by persisting small body size. This path of growth also led to stroke. We now report that it is associated with a reduced anteroposterior diameter of the mother’s bony pelvis, which is a consequence of rickets or lesser degrees of malnutrition in infancy. Malnutrition of infant girls could lead to persisting changes in protein metabolism that prejudice the early nutrition and growth of the next generation. Prevention of hypertension may ultimately depend on improving fetal nutrition and protecting the nutrition and growth of infants.

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Disclosures
None.

References


