Blood Pressure Is Lower in Children and Adolescents With a Low-Saturated-Fat Diet Since Infancy

The Special Turku Coronary Risk Factor Intervention Project

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Abstract—Blood pressure was measured in the prospective randomized Special Turku Coronary Risk Factor Intervention Project Study with an oscillometric method every year from 7 months to 15 years of age in 540 children receiving a low-saturated-fat, low-cholesterol diet and in 522 control children. Dietary intakes, family history of parental hypertension, and grandparental vascular disease were recorded. Systolic and diastolic blood pressures were 1.0 mm Hg lower (95% CI for systolic: −1.7 to −0.2 mm Hg; 95% CI for diastolic: −1.5 to −0.4 mm Hg) in children receiving low-saturated-fat counseling through childhood than in control children. Intakes of saturated fat were lower (P<0.001), those of polyunsaturated fat higher (P<0.001), and intakes of potassium slightly higher (P=0.002) in the intervention group, but sodium intakes were not influenced by the intervention (P=0.76). Children whose parents were hypertensive had 4- to 6-mm Hg higher systolic and 3- to 4-mm Hg higher diastolic blood pressures than children of normotensive parents (P<0.001). Diastolic blood pressure of children with grandparental vascular disease, ie, early cardiovascular or cerebrovascular disease, tended to be higher than that of children with no grandparental disease (P=0.051). We conclude that restriction of saturated fat from infancy until 15 years of age decreases childhood and adolescent blood pressure with a meaningful population-attributable amount. The importance of childhood lifestyle counseling and primary prevention of hypertension should be emphasized, especially in those children with a family history of hypertension or atherosclerotic vascular disease. (Hypertension. 2009;53:918-924.)

Key Words: atherosclerosis prevention ■ child nutrition sciences ■ cholesterol ■ diet ■ hypertension

Long-term health risks of hypertension in children and adolescents are substantial.1 Genes, environment, and their interaction influence the rise of blood pressure (BP) in childhood. The BP of children of hypertensive parents rises more rapidly than that of children of normotensive parents2–4; their vascular endothelium is also less elastic5 and left ventricular mass greater6 than those of children with no family history of hypertension. High childhood BP predisposes to adult hypertension,7 and adult vascular endothelial characteristics, eg, intima-media thickness of the common carotid artery, correlate significantly with systolic BP measured over the 2 previous decades at 12 to 18 years of age.8

Dietary sodium intake is regarded as the dietary culprit in BP.9 Thus, attempts to control its environmental risk factors in childhood should be directed to the entire population,10 including children. However, no controlled studies have been published on the effects of a long-term, low-saturated-fat diet on BP in childhood and adolescence.

Stop Hypertension,14 in which 459 adults were randomly assigned to receive one of the following: (1) a diet rich in fruits and vegetables; (2) a “combination” diet rich in fruits, vegetables, and high intake of low-fat dairy; or (3) a control diet. The combination diet, ie, the diet with high intake of fruits and vegetables and high intake of low-fat dairy, lowered BP significantly more than the fruit-and-vegetable diet only, and the effect was most marked in hypertensive subjects.

These observations support the concept that genes steer the age-dependent and environmentally modifiable increase in BP and hypertension-induced complications, and the published data suggest that nutrition during the very first year(s) of life may have a central role in programming of the future BP.9 Thus, attempts to control its environmental risk factors should be directed to the entire population,15 including children. However, no controlled studies have been published on the effects of a long-term, low-saturated-fat diet on BP in childhood and adolescence.

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This trial has been registered at www.clinicaltrials.gov (identifier NCT00223600).
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In the randomized, prospective Special Turku Coronary Risk Factor Intervention Project (STRIP) Study, children were at 7 months randomly allocated into 2 groups: those receiving saturated-fat–oriented counseling or a control group. We recently published the 14-year follow-up of the trial and showed that this intervention led to decreased saturated fat intake and serum cholesterol concentrations without effects on growth or pubertal development. In the present study we reported BP values of the intervention and control children until 15 years of age, as well as intakes of BP-related nutrients, and studied how parental hypertension and family history of vascular disease associated with childhood BP.

**Methods**

**Study Design and Counseling of the Families**

In the prospective, randomized STRIP Study, healthy infants were randomly assigned either to receive individualized dietary and lifestyle counseling aiming at modification of the environmental coronary heart disease risk factors (n=540) or to a control group (n=522) at 7 months of age. The intervention was individualized and aimed at the child’s fat intake of 30% to 35% of daily energy (E%), saturated:monounsaturated plus polyunsaturated fatty acid ratio of 1:2, and cholesterol intake <200 mg/d (for details see Reference 16). Breastfeeding or formula was advised during the first year of life. After 12 months of age, 0.5 to 0.6 L of skim milk were recommended daily. To maintain adequate fat intake, the parents were taught to add daily 2 or 3 teaspoonfuls (10 g) of soft margarine or vegetable oil, mainly low-erucic-acid grapeseed oil, to the child’s food from 12 to 24 months of age.

The counseling given in STRIP mainly focused on intakes of fat and saturated fat. Consequently, intakes of salt and sodium were not lower in the intervention children during the first years of life. Later, guidance toward lower sodium intakes was started when the child reached age 8 years, but this was never the main subject of the intervention. Ample use of vegetables, fruits, berries, and whole grain products was encouraged. The control children received the basic health education routinely given at Finnish well-baby clinics and school health care. At the age of 12 months, cow’s milk with 1.9% (1.5% after May 1995) fat was recommended for daily use. No suggestions on the use of fats were given to the control families, and dietary issues were discussed only superficially.

**Food Records and Anthropometric Measurements**

Food consumption data were obtained using annual 4-day food records (3-day food records before 2 years of age) as described, and the nutrient intakes were calculated using Micro Nutrica software. The energy and energy nutrient intakes of the STRIP children ≤14 years of age have been published previously. All of the children were weighed and measured annually as described, and body mass index (BMI) was calculated as kilograms per meter squared.

**Ethics**

The joint commission on ethics of the Turku University and the Turku University Central Hospital approved the STRIP Study.

**Measurement of BP**

Sitting BP of intervention and control children was measured annually from 7 months to 15 years of age using an oscillometric noninvasive BP monitor (Criticon Dinamap 1846 SX until 2001, thereafter Criticon Dinamap Compact T) after an appropriate rest of ≥15 minutes. BP of both parents was also measured annually. The accuracy of the device was regularly checked against a mercury manometer. Proper cuff size according to the size of the child’s right arm was used. Until 7 years of age, BP was measured once; at 8 to 9 years of age, it was measured 2 to 4 times; and after 9 years of age it was measured twice at each visit. BPs of the children and parents were measured annually at times of day suitable for the family. Both study groups were seen by the study team at the same time of year in the same environment (similar ambient temperature, etc), and both study groups were seen multiple times throughout the study, thereby minimizing tension and stress in both groups.

**Statistical Analyses**

The energy and micronutrient and macronutrient intakes (fat, saturated fat, protein, sodium, potassium, calcium, and magnesium) of the intervention and control children at ages 8 months to 15 years were compared with repeated-measures ANOVA.

In BP analyses, all of the BP values of children between 7 months and 15 years of age were used (maximum number of observations: 16). Before 8 years of age, BP was measured only once at each visit, but the mean values of 2 to 4 BP values measured at each visit at 8 to 15 years of age were used in the statistical analyses. First, repeated-measures ANOVA was performed to study BP of the intervention and control children across the age points from 7 months to 15 years. Second, BPs, weights, heights, and BMIs of intervention and control children at the study end point (ie, at 15 years of age) were compared by t test. Third, to evaluate the effect of parental hypertension on children’s BP in infancy and childhood, the children were divided into 3 groups on the basis of whether their parents were normotensive or if they had 1 or 2 hypertensive parents (ie, adult with mean BP during the study ≥140/≥90 mm Hg or medication for hypertension). All of the parents with ≥3 BP measurements during the study were included in these analyses. In these analyses, the study group (intervention/control) was used as a covariant. Effect of parental borderline hypertension (BP: 130 to 139/85 to 89 mm Hg) was studied alike. Fourth, the children were divided in 2 groups on the basis of history of early (men <60 years and women <70 years) coronary heart disease or cerebrovascular disease in their grandparents; BPs of these groups of children were compared with repeated-measures ANOVA.

Results were considered statistically significant at a value of P<0.05. Statistical analyses were performed using the SAS Software for Windows, release 9.1 (SAS Institute).

**Results**

**Macronutrient and Micronutrient Intakes of the Study Children Up to Age 15 Years**

As reported before, intakes of fat and saturated fat were lower in children of the intervention group than in those of the control group through the study (P<0.001; Table 1). Intakes of monounsaturated fat (as E%) did not differ between the study groups (P=0.12), but children of the intervention group had higher polynsaturated fat intakes than the controls (P<0.001). Dietary intakes of sodium (P=0.76) and calcium (P=0.08) did not differ between the study groups, but intakes of potassium (P=0.002) and magnesium (P<0.001) were slightly but significantly higher in children of the intervention group than in the controls (Table 1).

**Impact of Age and Sex on BP**

The systolic BP increased gradually from 92 mm Hg at 7 months to 110 mm Hg (mean) at 13 years in both sexes (Figure 1). Diastolic BP was at the lowest at 5 to 8 years of age and increased thereafter from 56 to 58 mm Hg to 60 to 62 mm Hg at 15 years of age (Figure 2). Boys tended to have higher systolic BP than girls (P=0.058), and the mean (95% CI) difference between the systolic BP of boys and girls was 0.8 mm Hg (95% CI: −0.0 to 1.6 mm Hg). There was a significant gender*age interaction in BP values (P<0.001);
this interaction was significant at ages 14 and 15 years. Thus, after 13 years of age, the systolic BP was clearly higher in boys than in girls. The overall diastolic BP did not differ between sexes (P = 0.95), but there was a significant gender × age interaction (P = 0.01) so that the boys had higher diastolic BP than girls at 7 months of age.

Effect of Dietary Counseling on BP

The children of the intervention group had lower systolic BP than controls (P = 0.018). The mean (95% CI) difference between the systolic BP of the intervention and control children was −1.0 mm Hg (95% CI: −1.7 to −0.2 mm Hg; Figure 1). The children of the intervention group also had lower diastolic BP than controls (P < 0.001; Figure 2), and the mean (95% CI) difference between diastolic BP of the intervention and control children was −1.0 mm Hg (95% CI: −1.5 to −0.4 mm Hg). The mean (SD) BPs, weights, heights, and BMIs of the intervention and control children at 15 years of age are presented in Table 2.

Effect of Parental Hypertension on Children’s BP

Of the studied children, 608 had normotensive parents, 281 had 1 hypertensive parent, and 36 had 2 hypertensive parents. The systolic BP differed significantly among these 3 groups of children (P < 0.001; Figure 3). At 7 months of age, the systolic BP of children with normotensive parents was 91.8 mm Hg (±13.2 mm Hg), whereas that of children with 2

Table 1. Mean (SD) Dietary Intakes of Intervention and Control Children From 13 Months to 15 Years of Age

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Intervention</th>
<th>Control</th>
<th>Intervention</th>
<th>Control</th>
<th>Intervention</th>
<th>Control</th>
<th>Intervention</th>
<th>Control</th>
<th>Intervention</th>
<th>Control</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td>510</td>
<td>481</td>
<td>429</td>
<td>414</td>
<td>363</td>
<td>358</td>
<td>295</td>
<td>309</td>
<td>266</td>
<td>287</td>
<td>248</td>
</tr>
<tr>
<td>Energy, kcal†</td>
<td>955 (179)</td>
<td>981 (185)</td>
<td>1202 (228)</td>
<td>1490 (248)</td>
<td>1497 (258)</td>
<td>1645 (305)</td>
<td>1828 (365)</td>
<td>1847 (413)</td>
<td>1958 (504)</td>
<td>2022 (558)</td>
<td>-0.001</td>
</tr>
<tr>
<td>Fat E%‡</td>
<td>26.3 (5.8)</td>
<td>27.8 (4.7)</td>
<td>30.3 (4.7)</td>
<td>32.7 (4.4)</td>
<td>29.6 (4.3)</td>
<td>32.4 (4.3)</td>
<td>28.7 (4.7)</td>
<td>32.1 (5.1)</td>
<td>30.5 (4.8)</td>
<td>31.4 (4.9)</td>
<td>-0.001</td>
</tr>
<tr>
<td>SAFA, E%</td>
<td>9.5 (2.9)</td>
<td>12.8 (2.7)</td>
<td>11.8 (2.4)</td>
<td>14.6 (2.6)</td>
<td>11.5 (2.2)</td>
<td>13.9 (2.4)</td>
<td>11.1 (2.5)</td>
<td>13.7 (2.8)</td>
<td>11.4 (2.7)</td>
<td>12.9 (2.7)</td>
<td>-0.001</td>
</tr>
<tr>
<td>MUFA, E%</td>
<td>9.4 (2.6)</td>
<td>8.8 (1.9)</td>
<td>10.2 (2.0)</td>
<td>10.5 (1.8)</td>
<td>10.6 (1.8)</td>
<td>11.0 (1.7)</td>
<td>10.9 (2.0)</td>
<td>11.0 (2.0)</td>
<td>11.1 (2.1)</td>
<td>10.7 (2.1)</td>
<td>10.9 (2.4)</td>
</tr>
<tr>
<td>PUFA, E%</td>
<td>5.2 (2.1)</td>
<td>3.9 (1.3)</td>
<td>5.5 (1.4)</td>
<td>4.8 (1.2)</td>
<td>5.3 (1.2)</td>
<td>5.0 (1.3)</td>
<td>5.7 (1.3)</td>
<td>5.2 (1.3)</td>
<td>5.8 (1.4)</td>
<td>5.3 (1.4)</td>
<td>5.9 (1.6)</td>
</tr>
<tr>
<td>Protein, E%</td>
<td>17.8 (2.8)</td>
<td>17.4 (2.6)</td>
<td>16.3 (2.2)</td>
<td>15.6 (2.1)</td>
<td>16.4 (2.2)</td>
<td>15.8 (2.1)</td>
<td>16.4 (2.7)</td>
<td>16.1 (2.4)</td>
<td>16.9 (2.9)</td>
<td>16.6 (2.6)</td>
<td>17.8 (3.0)</td>
</tr>
<tr>
<td>Sodium, mg</td>
<td>1533 (463)</td>
<td>1525 (512)</td>
<td>1897 (476)</td>
<td>1838 (484)</td>
<td>2290 (496)</td>
<td>2250 (535)</td>
<td>2196 (534)</td>
<td>2332 (595)</td>
<td>2739 (681)</td>
<td>2768 (685)</td>
<td>2973 (863)</td>
</tr>
<tr>
<td>Potassium, mg</td>
<td>2193 (520)</td>
<td>2161 (467)</td>
<td>2312 (477)</td>
<td>2200 (503)</td>
<td>2755 (574)</td>
<td>2596 (532)</td>
<td>2952 (613)</td>
<td>2884 (591)</td>
<td>3095 (728)</td>
<td>3063 (734)</td>
<td>3485 (971)</td>
</tr>
<tr>
<td>Calcium, mg</td>
<td>779 (230)</td>
<td>807 (240)</td>
<td>822 (221)</td>
<td>836 (248)</td>
<td>1035 (267)</td>
<td>1007 (260)</td>
<td>1070 (306)</td>
<td>1127 (320)</td>
<td>1154 (368)</td>
<td>1181 (380)</td>
<td>1228 (426)</td>
</tr>
<tr>
<td>Magnesium, mg</td>
<td>186 (45)</td>
<td>184 (41)</td>
<td>199 (41)</td>
<td>188 (42)</td>
<td>241 (49)</td>
<td>225 (45)</td>
<td>262 (54)</td>
<td>253 (52)</td>
<td>277 (66)</td>
<td>273 (64)</td>
<td>308 (84)</td>
</tr>
</tbody>
</table>

Intakes at ages 8 and 18 months and 2, 4, 5, 7, 8, 10, 11, 13, and 14 years are not shown.

*Difference between groups, by repeated-measures ANOVA.

Figure 1. Mean (SE) systolic BP of intervention and control girls and boys from 7 months to 15 years of age (P = 0.018 between intervention and control groups).

Figure 2. Mean (SE) diastolic BP of intervention and control girls and boys from 7 months to 15 years of age (P < 0.001 between intervention and control groups).
hypertensive parents was 94.9 mm Hg (±10.5 mm Hg). At 15 years, the respective BPs were 116.7 mm Hg (±12.3 mm Hg) and 121.6 mm Hg (±12.8 mm Hg; Table 2). The mean diastolic BP of children with normotensive parents was ~3 mm Hg lower than that of children with 2 hypertensive parents (P<0.001; Figure 4). The BP of the children with 1 hypertensive parent was between the BP of the children with normotensive parents and that of children with 2 hypertensive parents. BMIs of 15-year-old children with hypertensive parents were slightly higher than BMIs of children with normotensive parents (Table 2). Although the BPs of children with 2 parents with borderline high (130 to 139/85 to 89 mm Hg) BPs tended to be higher than those of children with normotensive parents this difference did not reach statistical significance (P=0.35 for systolic and P=0.13 for diastolic BPs; data not shown). The study group (intervention or control) did not influence these results, ie, parental hypertension affected childhood BP similarly in both study groups.

### Table 2. Mean (SD) BPs, Weights, Heights, and BMIs of 15-Year-Old Intervention and Control Children and Children With Either 2 Normotensive Parents or 1 or 2 Hypertensive Parents

<table>
<thead>
<tr>
<th>Study Subjects</th>
<th>No. of Children</th>
<th>Systolic BP, Mean (SD), mm Hg</th>
<th>Diastolic BP, Mean (SD), mm Hg</th>
<th>Height, Mean (SD), cm</th>
<th>Weight, Mean (SD), kg</th>
<th>BMI, Mean (SD), kg/m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intervention girls</td>
<td>115</td>
<td>113.6 (10.2)</td>
<td>60.2 (6.0)</td>
<td>166.6 (6.0)</td>
<td>57.1 (8.0)</td>
<td>20.6 (2.5)</td>
</tr>
<tr>
<td>Control girls</td>
<td>137</td>
<td>114.8 (11.9)</td>
<td>61.9 (6.9)</td>
<td>165.7 (5.9)</td>
<td>57.1 (10.6)</td>
<td>20.7 (3.6)</td>
</tr>
<tr>
<td>P</td>
<td>0.38</td>
<td>0.048†</td>
<td>0.28</td>
<td>0.74</td>
<td>0.86†</td>
<td></td>
</tr>
<tr>
<td>Intervention boys</td>
<td>133</td>
<td>120.8 (13.2)</td>
<td>60.8 (7.2)</td>
<td>173.9 (8.2)</td>
<td>61.2 (11.8)</td>
<td>20.1 (3.2)</td>
</tr>
<tr>
<td>Control boys</td>
<td>139</td>
<td>122.0 (12.3)</td>
<td>62.7 (7.5)</td>
<td>174.2 (7.0)</td>
<td>62.7 (11.7)</td>
<td>20.6 (3.5)</td>
</tr>
<tr>
<td>P</td>
<td>0.42</td>
<td>0.045†</td>
<td>0.78</td>
<td>0.24</td>
<td>0.23</td>
<td></td>
</tr>
<tr>
<td>Children with</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 normotensive parents</td>
<td>608 (296 F, 312 M)</td>
<td>116.7 (12.3)</td>
<td>60.8 (7.1)</td>
<td>170.8 (8.0)</td>
<td>59.2 (10.9)</td>
<td>20.2 (3.1)</td>
</tr>
<tr>
<td>1 hypertensive parent</td>
<td>281 (138 F, 143 M)</td>
<td>119.9 (12.5)</td>
<td>62.4 (6.8)</td>
<td>168.9 (7.6)</td>
<td>60.0 (11.0)</td>
<td>21.0 (3.4)</td>
</tr>
<tr>
<td>2 hypertensive parents</td>
<td>36 (19 F, 17 M)</td>
<td>121.6 (12.8)</td>
<td>63.2 (5.8)</td>
<td>171.1 (7.6)</td>
<td>61.2 (11.7)</td>
<td>20.9 (4.0)</td>
</tr>
<tr>
<td>P</td>
<td>0.008</td>
<td>0.023</td>
<td>0.035</td>
<td>0.53</td>
<td>0.032</td>
<td></td>
</tr>
</tbody>
</table>

F indicates female; M, male.

*Data were equated by t test.
†Logarithmic transformation was used to normalize the distribution.
‡Data were equated by ANOVA.

### Figure 3. Mean (SE) systolic BP from 7 months to 15 years of age in children with normotensive parents, 1 hypertensive parent, or 2 hypertensive parents (P<0.001 between the groups).

### Figure 4. Mean (SE) diastolic BP from 7 months to 15 years of age in children with normotensive parents, 1 hypertensive parent, or 2 hypertensive parents (P<0.001 between the groups).

Grandparents’ Vascular Disease and Children’s BP

Of the studied children, 292 had ≥1 grandparent with either early coronary heart disease or cerebrovascular disease, whereas grandparents of 763 children had no early vascular disease. The diastolic BP of the children with 1 hypertensive grandparent tended to be higher in children with early grandparental vascular disease (P=0.051).
Discussion

We recently published the 14-year follow-up results of the STRIP Study and showed that saturated fat–oriented counseling resulted in lower serum cholesterol concentrations, slightly decreased intake of total fat, and markedly decreased intake of saturated fat (11 to 12 E% versus 13 to 14 E% in control children). We have now shown that saturated fat–reduced diet since infancy decreases the mean systolic and diastolic BP values of both girls and boys by ≈1 mm Hg.

The diastolic BPs were higher until 3 years of age in the present study when compared with age- and height-adjusted mean values presented in a recent working group report. BP of >120/80 mm Hg in adolescence was considered “prehypertensive” by the working group report. In late puberty, the systolic BPs of the STRIP children were higher than recommended, because at 15 years of age, systolic BP was >120 mm Hg in 51% of the boys and 29% of the girls. On the other hand, the diastolic BP was >80 mm Hg (also considered prehypertensive) in only ≈1% of the adolescents.

Salt restriction during the first year of life significantly delays the BP rise in childhood. STRIP intervention, not being focused on salt or sodium intakes during the first years of life, did not lead to lower salt consumption in children of the intervention group than in controls up to age 5 years. In the present study we have shown that the intakes of sodium and calcium were not significantly influenced by the intervention. A minor increase in the intakes of potassium (~75 mg/d) and magnesium (~8 mg/d) was observed in children of the intervention group, but impact on BP of such small changes may not be detectable.

Quantity and quality of dietary fats influence BPs of laboratory animals as well as of humans, even without sodium restriction. A diet rich in monounsaturated fat resulted in reductions in systolic (~2.2%) and especially diastolic (~3.8%) BPs, but, interestingly, the favorable effects on BP disappeared at a total fat intake level >37 E%. Exercise alone has been associated with 1% to 3% reductions in both systolic and diastolic BPs, but the effects of the Dietary Approaches to Stop Hypertension diet and weight loss of several kilograms have been somewhat larger than those with exercise alone.

Fiber and protein intakes might also influence children’s BP. In the STRIP Study, protein intakes were constantly slightly higher in the intervention group than in the controls. The intakes of fat and saturated fat of the intervention children were lower, intakes of monounsaturated fat similar, and intakes of polyunsaturated fat higher in children of the intervention group than in controls. It is difficult to know whether the lower BP values in children of the intervention group were because of a lower intake of saturated fat, a higher intake of polyunsaturated fat, a higher intake of protein, or a combination of all of these.

Other than sodium consumption, obesity may be the most important modifiable factor influencing the increase of BP during childhood. Both obese children and adults have higher BPs that their lean counterparts, and weight loss associates with a decrease in BP. Central obesity is the most important factor of insulin sensitivity, but it is also influenced by dietary fat quality. The STRIP diet improved insulin sensitivity without differences in weights, heights, and BMIs between the children in the intervention and control groups. Thus, the 1-mm Hg difference in the mean BPs between the intervention and control groups might be attributed to improved insulin sensitivity rather than decreased sodium intake.

The BPs of boys and girls are closely similar until midpuberty, at which point BPs of boys tend to be higher than those of girls. This age coincides with the age at which also other cardiovascular risk factors (especially high-density lipoprotein cholesterol) deteriorate in boys when compared with girls. Although the BPs of the intervention boys were significantly lower than those of the controls, the pubertal BP increase could not be blunted by the fat-oriented intervention. Studies in traditional, nonindustrialized countries have shown that BPs do not always increase with age in adulthood. In the STRIP Study, BP increased with age along with increasing sodium intake and reached the adult BP levels of low-sodium cultures already at the age of 8 to 10 years.

The Fels Longitudinal Study showed that the risk of developing hypertension at the fourth decade of life was 3.8 to 4.5 times more common in those 5- to 7-year-old boys and girls who had BP at a single examination above that of children who remained free of metabolic syndrome as adults. A single routine BP measurement in adolescence predicts young adult hypertension (girls) and 10-year cardiovascular risk (boys). The impact of early BP on vascular health is immense, because childhood systolic BP measured at ages 12 to 18 years independently associates with adult carotid artery intima-media thickness 21 years later.

BP is higher in children of hypertensive parents, and van Hooft showed that no clear differences existed in BPs between 8-year-old children of parents with relatively high BP compared with those of children with no family history of high BP, but at 20 years of age, a 7-mm Hg difference in both systolic and diastolic BPs was found. In the STRIP Study, we have now shown that BPs in children of hypertensive parents are higher than in those of normotensive parents through childhood, and at late childhood, their BMIs are also slightly higher.

Relative risk for cardiovascular disease is 1.23 for a 10-mm Hg increase in systolic BP. This relationship is positive starting at the systolic BP level of 115 mm Hg and even more so above 120 mm Hg. In hypertensive patients, a 5- to 6-mm Hg decrease in diastolic BP decreases the risk of stroke and myocardial infarction by 38% and 16%, respectively. Therefore, a 1-mm Hg lower BP in children of the intervention group, if maintained through lifetime or even increased with age, might have an immense effect on future vascular health.

There are some potential weaknesses in our study. The reporting of grandparental vascular disease has possibly not been accurate in all of the families. On the other hand, this may even dilute the estimation of the effect of grandparental vascular disease on children’s BP, because some true cases of grandparental vascular disease may have been missed. Another possible weakness is the measurement of BP, which
was done only once at each visit until 7 years of age. Moreover, the oscillometric BP monitor is not completely accurate in estimating systolic or diastolic BPs.\textsuperscript{39} Clear strengths of the present study include a very long follow-up period, serial BP measurements, and accurate estimation of dietary intakes.

**Perspectives**

The STRIP data show that supervised dietary counseling to restrict saturated fat intake and to increase unsaturated fat, vegetable, and fruit intake from infancy until 15 years of age significantly decreases childhood systolic and diastolic BPs. Children of hypertensive parents are already prone to higher BPs in early childhood. These observations strongly emphasize the importance of primary prevention of hypertension in the general child population and especially in those with family history of vascular disease.

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**Disclosures**

None.

**References**


Blood Pressure Is Lower in Children and Adolescents With a Low-Saturated-Fat Diet Since Infancy: The Special Turku Coronary Risk Factor Intervention Project
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