Effect of 7-Year Infancy-Onset Dietary Intervention on Serum Lipoproteins and Lipoprotein Subclasses in Healthy Children in the Prospective, Randomized Special Turku coronary Risk factor Intervention Project for children (STRIP) Study

Tuuli Kaitosaari, MD; Tapani Rönnemaa, MD, PhD; Olli Raitakari, MD, PhD; Sanna Talvia, MSc; Katariina Kallio, MD; Iina Volanen, MD; Aila Leino, PhD; Eero Jokinen, MD, PhD; Iikka Välimäki, MD, MSc; Jorma Viikari, MD, PhD; Olli Simell, MD, PhD

Background—We previously showed that low-saturated-fat dietary intervention from infancy until 5 years of age safely and effectively reduced serum cholesterol concentration. We now report how such intervention influenced serum lipids, LDL particle size, and HDL subfractions in children when they reached the age of 7 years.

Methods and Results—Healthy 7-month-old infants (n=1062) were randomized to the intervention (n=540) and control (n=522) groups. Each year, two individualized counseling sessions were organized to the intervention families. Serum lipid values were measured annually. The intervention boys had 0.20 to 0.39 mmol/L lower serum cholesterol values than the control boys throughout the follow-up (always \( P<0.05 \)), but the values of the intervention and control girls did not differ. The LDL particle sizes and HDL subfractions were determined in a random subgroup of 96 intervention and 101 control children at the age of 7 years. The mean particle diameter of major LDL peak was 262.6 Å in the intervention boys and 258.5 Å in the control boys (\( P=0.05 \)), and 259.2 Å in the intervention girls and 261.3 Å in the control girls (\( P=0.30 \)). HDL\(_2\) and HDL\(_3\) cholesterol concentrations did not differ between the intervention and control children or between the two genders.

Conclusions—The 7-year intervention favorably influenced not only the serum total and LDL cholesterol concentrations but also the LDL particle size in boys. LDL particle size remained unchanged in girls, as did HDL\(_2\) and HDL\(_3\) concentrations in both genders. (Circulation. 2003;108:672-677.)

Key Words: atherosclerosis ■ LDL subclasses ■ lipoproteins ■ pediatrics ■ prevention

High LDL cholesterol concentration is an important risk factor for coronary heart disease (CHD).\(^1\) The size of LDL particles may determine their atherogenicity. The predominance of small, dense LDL (sdLDL) or LDL subclass pattern B (diameter of most LDL particles <255 Å) may be particularly atherogenic.\(^2\)\(^,\)\(^3\) This may be due to their low binding affinity to the LDL receptor, low resistance to oxidative stress, and prolonged plasma half-life.\(^3\)\(^,\)\(^4\) Small particle size may promote penetration into the intima and LDL cholesterol accumulation in the arterial wall, accelerating the development of atherosclerosis.\(^3\)\(^,\)\(^4\) Both genetic and environmental factors can influence LDL particle size.\(^5\)\(^,\)\(^7\)

HDL particles can be divided into HDL\(_2\) and HDL\(_3\) fractions according to their density. The lipid-rich HDL\(_2\) may be more important in the protection from atherosclerosis.\(^8\)

We recently showed that individualized, repeatedly given dietary counseling aiming at a low–saturated-fat, low-cholesterol diet from the age of 7 months markedly lowered total and non-HDL cholesterol and apolipoprotein B concentrations up to the age of 5 years.\(^9\)\(^,\)\(^10\) We have extended the study to 7 years of age, and report now, in addition to the follow-up data, for the first time the effects of the intervention on serum LDL and HDL fractions.

Methods

Study Design and Subjects

The study design of the on-going prospective, randomized STRIP study (Special Turku Coronary Risk Factor Intervention Project for Children), which began in 1990, has been published.\(^9\) Briefly, 1062 healthy infants were recruited and randomized to an intervention group (n=540) or a control group (n=522).

The intervention group received twice a year dietary and lifestyle counseling given by a team consisting of physicians and dietitians. The families recorded child’s food consumption for four days twice

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From the Research Centre of Applied and Preventive Cardiovascular Medicine (T.K., S.T., K.K., J.V.), Departments of Medicine (T.R., J.V.) and Pediatrics (I.V., O.S.), University of Turku, Finland; Clinical Physiology/PET-Centre (O.R.), Turku, Finland; Research and Development Unit of Social Insurance Institution (A.L.), Turku, Finland; Department of Clinical Chemistry (A.L.), Turku, Finland; and Hospital for Children and Adolescents (E.J.), Helsinki, Finland.

Correspondence to Tuuli Kaitosaari, MD, Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku, Kiïnämyllynkatu 10, FIN-20520 Turku, Finland. E-mail tuuli.kaitosaari@utu.fi

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Laboratory Methods

Serum total and HDL cholesterol, apolipoprotein A-I, apoB, and triglyceride values were measured as described. Nonfasting blood samples were drawn when children’s exposure to known environmental atherosclerosis risk factors. A dietitian checked the food records and suggested appropriate changes to diet. The diet was designed to meet the Nordic Dietary Recommendations. Since the age of 3 years, the recommended intakes comprised protein 10 to 15 E% (percentage of energy), fat 30 E% (saturated fat ≤10 E%), and carbohydrate 55 to 60 E%. The control children have received the basic health education given at Finnish well-baby clinics. During their regular STRIP visits, they received no detailed dietary counseling. Data used in the longitudinal analyses of serum lipid values comprised all those children whose blood samples had been successfully obtained at the ages of 7 months (baseline) and 7 years (n=511). The HDL and LDL subfractions were determined in a random subsample of 197 consecutive children at their 7-year STRIP visit (later called the lipoprotein subgroup). Of them, 51 girls and 45 boys were from the intervention group and 50 girls and 51 boys from the control group. LDL particle determination was successfully completed in 176 samples.

The study was approved by the Joint Commission on Ethics of the Turku University and the Turku University Central Hospital. Informed consent was obtained from all parents.

### Results

Serum Lipids and Lipoproteins During the 7-Year Intervention

The baseline-adjusted means of serum total and non-HDL cholesterol and apoB concentrations were lower in the intervention boys than in the control boys throughout the 7-year period (Table 1). HDL cholesterol and apoA-I concentrations were lower in the intervention boys until 36 months, but the difference then disappeared during the follow-up.
TABLE 2. Serum Lipid, Lipoprotein, and Apolipoprotein Concentrations in the Intervention and Control Girls at 7 to 84 Months of Age

<table>
<thead>
<tr>
<th></th>
<th>n*</th>
<th>7 mo</th>
<th>13 mo</th>
<th>36 mo</th>
<th>60 mo</th>
<th>84 mo</th>
<th>Δ 7–84 mo†</th>
<th>Overall intervention Effect (95% CI, mmol/L)</th>
<th>Difference in Change at a Single Age Point (95% CI, mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol, mmol/L</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Intervention girls</td>
<td>112</td>
<td>4.15±0.64</td>
<td>4.19±0.73</td>
<td>3.49±0.72</td>
<td>4.44±0.67</td>
<td>4.55±0.69</td>
<td>0.40±0.87</td>
<td>−0.090 (−0.213 to 0.032)</td>
<td></td>
</tr>
<tr>
<td>Control girls</td>
<td>125</td>
<td>4.13±0.63</td>
<td>4.31±0.78</td>
<td>4.46±0.69</td>
<td>4.54±0.75</td>
<td>4.56±0.65</td>
<td>0.43±0.78</td>
<td>P=0.15</td>
<td></td>
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<tr>
<td>Non-HDL cholesterol, mmol/L</td>
<td></td>
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<tr>
<td>Intervention girls</td>
<td>111</td>
<td>3.26±0.81</td>
<td>3.36±0.69</td>
<td>3.37±0.68</td>
<td>3.27±0.62</td>
<td>3.27±0.63</td>
<td>0.01±0.80</td>
<td>−0.061 (−0.177 to 0.055)</td>
<td></td>
</tr>
<tr>
<td>Control girls</td>
<td>125</td>
<td>3.24±0.80</td>
<td>3.41±0.76</td>
<td>3.39±0.66</td>
<td>3.34±0.70</td>
<td>3.29±0.61</td>
<td>0.05±0.69</td>
<td>P=0.30</td>
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<tr>
<td>HDL cholesterol, mmol/L</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>−0.049 (−0.082 to −0.016)§</td>
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<tr>
<td>Intervention girls</td>
<td>111</td>
<td>0.90±0.18</td>
<td>0.85±0.19</td>
<td>1.03±0.17</td>
<td>1.18±0.20</td>
<td>1.29±0.18</td>
<td>0.39±0.21</td>
<td>−0.017 (−0.047 to 0.013)§</td>
<td></td>
</tr>
<tr>
<td>Control girls</td>
<td>125</td>
<td>0.89±0.17</td>
<td>0.89±0.17</td>
<td>1.07±0.20</td>
<td>1.20±0.22</td>
<td>1.28±0.21</td>
<td>0.38±0.21</td>
<td>0.016 (−0.030 to 0.062)†</td>
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<tr>
<td>HDL/total cholesterol</td>
<td></td>
<td></td>
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<tr>
<td>Intervention girls</td>
<td>111</td>
<td>0.22±0.06</td>
<td>0.21±0.05</td>
<td>0.24±0.05</td>
<td>0.27±0.05</td>
<td>0.29±0.04</td>
<td>0.06±0.05</td>
<td>−0.003 (−0.011 to 0.005)</td>
<td></td>
</tr>
<tr>
<td>Control girls</td>
<td>125</td>
<td>0.22±0.05</td>
<td>0.21±0.05</td>
<td>0.24±0.05</td>
<td>0.27±0.05</td>
<td>0.28±0.05</td>
<td>0.06±0.05</td>
<td>P=0.49</td>
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<tr>
<td>ApoA-I, g/L</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention girls</td>
<td>96</td>
<td>1.11±0.12</td>
<td>1.05±0.15</td>
<td>1.11±0.13</td>
<td>1.24±0.18</td>
<td>1.31±0.16</td>
<td>0.19±0.17</td>
<td>−0.023 (−0.048 to 0.002)§</td>
<td></td>
</tr>
<tr>
<td>Control girls</td>
<td>107</td>
<td>1.11±0.14</td>
<td>1.08±0.15</td>
<td>1.16±0.14</td>
<td>1.27±0.18</td>
<td>1.30±0.18</td>
<td>0.19±0.17</td>
<td>0.004 (−0.037 to 0.045)†</td>
<td></td>
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<tr>
<td>ApoB, g/L</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention girls</td>
<td>96</td>
<td>0.80±0.19</td>
<td>0.86±0.18</td>
<td>0.82±0.16</td>
<td>0.79±0.15</td>
<td>0.74±0.15</td>
<td>−0.06±0.19</td>
<td>−0.019 (−0.049 to 0.012)</td>
<td></td>
</tr>
<tr>
<td>Control girls</td>
<td>107</td>
<td>0.80±0.19</td>
<td>0.87±0.18</td>
<td>0.84±0.16</td>
<td>0.81±0.17</td>
<td>0.76±0.15</td>
<td>−0.05±0.18</td>
<td>P=0.24</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean±SD. Data from 24 and 48 months not shown.

*Children in whom at least the baseline and the 84-month lipid measurement were obtained.

†Mean of SD of individual changes between 7 and 84 months.

§In the case of significant interaction (groups by time interaction) the differences (control vs intervention) of least square mean±SD are given at the ages of 13a, 48b, and 84c months instead of the P value.

The intervention and control girls did not differ in the baseline-adjusted means of serum cholesterol, non-HDL cholesterol, HDL/total cholesterol ratio, or apoB concentrations during the 7-year intervention (Table 2). The control girls had higher adjusted means of HDL cholesterol concentration than the intervention girls at the age of 13 months (P=0.004). ApoA-I concentrations were lower in the intervention girls up to 3 years (P=0.008).

The fasting LDL cholesterol and triglyceride concentrations at the ages of 5 and 7 are presented in Figure 1. From 5 to 7 years, triglycerides tended to decrease in the intervention group compared with the control group (difference in change between groups, P=0.042). As expected, the LDL cholesterol values were lower in the intervention boys than in the control boys (P<0.0001, 95% CI −0.333 to −0.150 mmol/L). In the intervention girls and control girls, the triglyceride and LDL cholesterol values were similar (P=0.75 and P=0.18, respectively), and the values also remained rather stable from 5 to 7 years of age.

**Energy Nutrient Intakes and Serum Lipid Values at 7 Years of Age**

At the age of 7 years, the mean daily intake of energy, total fat, and carbohydrates were 6818 kJ, 30.7 E%, and 52.8 E% respectively.

**Figure 1.** Fasting serum LDL cholesterol and triglyceride concentrations in the intervention children (filled bars) and control children (open bars) at the ages of 5 and 7 years according to gender. Mean and SD are shown (number of children in parentheses).
TABLE 3. Serum Lipid, Lipoprotein, and Apolipoprotein Concentrations in 7-Year-Old Children

<table>
<thead>
<tr>
<th></th>
<th>Interveneion Boys (n=170)</th>
<th>Control Boys (n=170)</th>
<th>P</th>
<th>95% CI†</th>
<th>Interveneion Girls (n=145)</th>
<th>Control Girls (n=155)</th>
<th>P</th>
<th>95% CI†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol, mmol/L</td>
<td>4.20±0.64</td>
<td>4.41±0.66</td>
<td>0.003</td>
<td>-0.348 to -0.071</td>
<td>4.55±0.66</td>
<td>4.57±0.72</td>
<td>0.78</td>
<td>-0.181 to 0.135</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>0.64±0.26</td>
<td>0.68±0.30</td>
<td>0.30</td>
<td></td>
<td>0.71±0.28</td>
<td>0.68±0.25</td>
<td>0.48</td>
<td></td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td>2.63±0.55</td>
<td>2.82±0.58</td>
<td>0.002</td>
<td>-0.312 to -0.071</td>
<td>2.94±0.60</td>
<td>2.98±0.65</td>
<td>0.59</td>
<td>-0.181 to 0.104</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.29±0.24</td>
<td>1.29±0.25</td>
<td>1.00</td>
<td></td>
<td>1.28±0.20</td>
<td>1.27±0.22</td>
<td>0.82</td>
<td>-0.042 to 0.052</td>
</tr>
<tr>
<td>HDL/total cholesterol, %</td>
<td>31.0±5.9</td>
<td>29.5±5.8</td>
<td>0.02</td>
<td></td>
<td>0.003 to 0.028</td>
<td>28.6±4.9</td>
<td>0.74</td>
<td>-0.010 to 0.014</td>
</tr>
<tr>
<td>ApoA-I, g/L</td>
<td>1.30±0.20†</td>
<td>1.31±0.21†</td>
<td>0.71</td>
<td>-0.053 to 0.036</td>
<td>1.30±0.16†</td>
<td>1.31±0.19†</td>
<td>0.79</td>
<td>-0.047 to 0.036</td>
</tr>
<tr>
<td>ApoB, g/L</td>
<td>0.67±0.15†</td>
<td>0.73±0.16†</td>
<td>0.002</td>
<td>-0.088 to -0.021</td>
<td>0.76±0.17†</td>
<td>0.76±0.17†</td>
<td>0.88</td>
<td>-0.036 to 0.041</td>
</tr>
</tbody>
</table>

Values are mean±SD. †Two-sample t test for comparison of the group means; 95% CI of the difference in the mean between the intervention and control children.

in the intervention boys and 6955kJ, 31.6 E% and 52.8 E% in the control boys, respectively (always P>0.20). The intake of saturated fat was 11.5 E% in the intervention boys and 13.6 E% in the control boys (P<0.001). The (polyunsaturated+monounsaturated fat)/saturated fat ([P+M]/S) ratios in the two groups of boys were 1.5 and 1.2 (P=0.002), respectively.

The mean daily intakes of energy, total fat, and carbohydrates were 6256 kJ, 30.4 E%, and 53.2 E% in the intervention girls and 6340 kJ, 31.8 E%, and 52.2 E% in the control girls, respectively (for the differences, P=0.47, P=0.005, and P=0.06). The intake of saturated fat in the intervention girls was 11.8 E% and the control girls 13.7 E% (P<0.001). The ([P+M]/S) ratios in the two groups of girls were 1.4 and 1.2 (P<0.001), respectively.

In 7-year-olds, the mean total and LDL cholesterol concentrations were 5% and 7% lower in the intervention boys than in the control boys, respectively (Table 3). The HDL/total cholesterol ratio was 5% higher in the intervention boys than in the control boys. Serum apoA-I concentration showed no difference, but apoB concentration was 9% lower in the intervention boys than in the control boys.

The 7-year-old intervention and control girls showed no differences in any of the measured lipid, lipoprotein, or apolipoprotein values.

The dietary (P+M)/S ratio correlated inversely with total and LDL cholesterol and apoB concentrations in boys (r=-0.14, r=-0.16, and r=-0.18, respectively; always P<0.02), but not in girls. There was no correlation between carbohydrate intake and any of the lipid variables in either gender.

The heights or weights at the age of 7 years of the intervention and control children and of the two genders were not different (data not shown). No correlation was found between parameters of adiposity (weight, relative weight, and body mass index) and lipid or lipoprotein concentrations except a weak positive correlation between adiposity variables and serum triglycerides in boys (r=0.15, r=0.12, and r=0.15, respectively; always P<0.025).

LDL and HDL Subclasses in 7-Year-Old Children (the Lipoprotein Subgroup)

The mean average LDL particle diameters were 260.3 Å and 260.2 Å in all boys (n=81) and in all girls (n=95), respectively. The intervention boys but not girls had larger major LDL particle diameter than their controls (Table 4). The median of major LDL particle size was 263 Å in intervention boys and 257 Å in control boys, and the distribution curves differed significantly between the groups (P=0.048; Figure 2).

To examine the possible relationship between serum LDL concentration and particle size, we divided the children into two groups using their median LDL cholesterol (2.79 mmol/L) as the cut-off point. The intervention boys...

TABLE 4. Serum LDL, HDL, HDL2, and HDL3 Cholesterol and Triglyceride Concentrations and LDL Particle Sizes in the Lipoprotein Subgroup of 7-Year-Old Children

<table>
<thead>
<tr>
<th></th>
<th>Intervention Boys (n=45)</th>
<th>Control Boys (n=51)</th>
<th>P</th>
<th>95% CI†</th>
<th>Intervention Girls (n=50)</th>
<th>Control Girls (n=50)</th>
<th>P</th>
<th>95% CI†</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td>2.67±0.54</td>
<td>2.75±0.55</td>
<td>0.49</td>
<td>-0.300 to 0.145</td>
<td>2.85±0.54</td>
<td>2.96±0.57</td>
<td>0.33</td>
<td>-0.325 to 0.111</td>
</tr>
<tr>
<td>LDL₅₀ particle size (Å)</td>
<td>262.6±9.0a</td>
<td>258.5±10.1a</td>
<td>0.05</td>
<td>-0.079 to 8.405</td>
<td>259.2±9.1i</td>
<td>261.3±10.3d</td>
<td>0.30</td>
<td>-6.017 to 1.860</td>
</tr>
<tr>
<td>LDL₄₀ particle size (Å)</td>
<td>263.2±10.3i</td>
<td>259.5±9.9g</td>
<td>0.10</td>
<td>-0.766 to 8.169</td>
<td>260.6±10.0d</td>
<td>262.1±10.4i</td>
<td>0.48</td>
<td>-5.571 to 2.643</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>0.58±0.22</td>
<td>0.72±0.29</td>
<td>0.007</td>
<td></td>
<td>0.72±0.25</td>
<td>0.69±0.22</td>
<td>0.48</td>
<td></td>
</tr>
<tr>
<td>HDL₅₀ cholesterol, mmol/L</td>
<td>1.49±0.27</td>
<td>1.47±0.35</td>
<td>0.78</td>
<td>-0.110 to 0.146</td>
<td>1.39±0.22</td>
<td>1.46±0.30</td>
<td>0.21</td>
<td>-0.170 to 0.037</td>
</tr>
<tr>
<td>HDL₄₀, mmol/L</td>
<td>0.32±0.11</td>
<td>0.31±0.11</td>
<td>0.53</td>
<td>-0.031 to 0.059</td>
<td>0.30±0.09</td>
<td>0.32±0.10</td>
<td>0.21</td>
<td>-0.061 to 0.014</td>
</tr>
<tr>
<td>HDL₃ₐ, mmol/L</td>
<td>1.17±0.18</td>
<td>1.16±0.26</td>
<td>0.91</td>
<td>-0.088 to 0.098</td>
<td>1.10±0.16</td>
<td>1.14±0.21</td>
<td>0.26</td>
<td>-0.117 to 0.032</td>
</tr>
</tbody>
</table>

Values are mean±SD. †Two-sample t test for comparison of the group means; 95% CI of the difference in the mean between the intervention and control children.

LDL₅₀ indicates major peak of LDL particle diameter; LDL₄₀, average LDL particle diameter; HDL₅₀, HDL concentration measured with heparin-manganese.
with low LDL concentration had large LDL particles, but the control boys with low LDL concentration had small LDL particles (average particle sizes, 264.4 Å and 255.9 Å; \( P = 0.006 \)). The intervention and control boys with LDL cholesterol concentration above the median had closely similar LDL particle sizes (average particle sizes, 261.4 Å and 263.6 Å; \( P = 0.51 \)). In girls, the LDL concentrations and LDL particle sizes were randomly distributed in both the intervention and control groups. Serum triglyceride concentration and LDL particle size correlated poorly in all groups of children (always \( r < 0.20; \) \( P = \) NS).

HDL\(_2\) and HDL\(_3\) cholesterol concentrations were similar between the intervention and control boys and the intervention and control girls (Table 4).

Discussion

This study confirms our former findings that dietary counseling given repeatedly starting at the age of 7 months aiming at permanently low intake of saturated fat decreases lipid risk factors of CHD in the children.9,10 Our study demonstrates that serum total cholesterol, HDL and non-HDL cholesterol, apoA-I, and apoB concentrations are persistently lower in the intervention boys than in the control boys when they have reached the age of 7 years. At 5 to 7 years old, children usually go through great changes in their daily life when daycare, preschool, and school usually begin. Despite this, the intervention effect remains essentially similar as in younger children. We also show that LDL particles in 7-year-old intervention boys are larger in size than those in control boys, indicating that the intervention has a favorable effect on the LDL particle composition.

Fasting serum LDL cholesterol and triglycerides were for the first time determined when the children were 5 years old.10 Now, we present these values for the 7-year-old children. LDL cholesterol was lower in the intervention boys than in the control boys at both time points. Serum triglycerides tended to decrease in the intervention boys and to increase in the control boys.

The LDL particles were larger in the intervention boys than in the control boys. This difference was confined to those who had LDL cholesterol values below the median, suggesting that the dietary intervention had a favorable influence on both serum LDL cholesterol concentration and composition of the LDL particles.

In adults, LDL particle size is inversely associated with serum triglycerides.2,17 We did not find such a correlation and nor was it found in a previous study on children under 10 years of age.18 In the Bogalusa heart study, the correlation between triglyceride concentration and LDL particle size in 10- to 17-year-old children was weak \( (r = -0.21) \), and many of these subjects were already in puberty.19 Arisaka et al3 observed that triglycerides were higher in 7- to 13-year-old children with LDL pattern B than those with pattern A, but correlation was not reported. Therefore, it is likely that the correlation between triglyceride concentration and LDL particle size strengthens with age.

Changing from high-fat to low-fat, high-carbohydrate diet may produce decreases in LDL cholesterol and HDL cholesterol concentrations and increases in triglycerides.20 LDL subclass pattern contributes to lipoprotein response to change in diet. Dreon et al21 in their study of the effects of short-term diet on LDL subclasses in normolipidemic men found that men with LDL pattern B achieved greater reductions in LDL cholesterol and apoB concentrations and number of small LDL particles than men with LDL pattern A on a low-fat, high-carbohydrate diet. Thus, pattern B subjects seem to benefit the most from a low-fat diet. In a study on younger subjects (mean age 14 years, \( n = 50 \)), a 10-day extremely low-fat (10%), high-carbohydrate diet converted LDL pattern A to pattern B in 6 genetically predisposed children.22 However, the findings of these studies were not explained by changes in serum triglyceride concentration.21,22 Thus, heredity and genetic variation in LDL subclass patterns strongly contribute to LDL particle’s responses to diet. Our study is the first to examine the effect of a long-term dietary intervention on LDL particle distribution in solely prepubertal children. As we measured LDL particle size only once after 7 years of intervention, we are unable to judge whether the effect of intervention depended on the LDL particle size at the beginning of the intervention.

Despite the fact that the intervention reduced saturated fat intake as much in the intervention girls than in the intervention boys, it had no effect on any lipid or lipoprotein variable in the girls at the age of 7 years. In agreement, LDL cholesterol response to sitostanol ester margarine use is greater in boys than in girls.23 Possible causes for inefficient intervention impact on serum lipid values in girls include gender differences in body composition, serum sex hormone concentrations, and exercise habits.

We conclude that the 7-year low–saturated-fat dietary intervention had a favorable influence on serum total and LDL cholesterol concentration, as well as LDL particle size in boys. LDL concentration and particle size remained unchanged in girls, as did the HDL\(_2\) and HDL\(_3\) concentrations in both genders.
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Tuuli Kaitosaari, Tapani Rönnemaa, Olli Raitakari, Sanna Talvia, Katariina Kallio, Iina Volanen, Aila Leino, Eero Jokinen, Ilkka Välimäki, Jorma Viikari and Olli Simell

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