Impact of Dietary Intervention, Sex, and Apolipoprotein E Phenotype on Tracking of Serum Lipids and Apolipoproteins in 1- to 5-Year-Old Children

The Special Turku Coronary Risk Factor Intervention Project (STRIP)

Leena Rask-Nissilä, Eero Jokinen, Jorma Viikari, Anne Tammi, Tapani Rönnemaa, Jukka Marniemi, Pia Salo, Taina Routi, Hans Helenius, Ilkka Välimäki, Olli Simell

Abstract—The effects of dietary intervention, sex, and apolipoprotein E phenotype on tracking of serum lipid values in young children have remained poorly characterized. We investigated these associations in 1062 infants who were randomized into control and intervention groups (n=522 and n=540, respectively) at age 7 months; the intervention group received counseling aimed at maintaining a low–saturated fat, low-cholesterol diet. In 519 children in the control (n=254) and intervention (n=265) groups, serum lipid values were studied annually between 13 months and 5 years of age. In all children, tracking was strongest for the ratio of high density lipoprotein (HDL) cholesterol to total cholesterol; when a 13-month-old child belonged to the lowest quartile of the distribution, the odds ratio for belonging to the same quartile at older ages was 39.0 (95% CI 23.1 to 66.0). Dietary intervention did not influence the tracking of serum lipids. Tracking of HDL cholesterol was stronger in the boys than in the girls (P=0.018). Tracking of non-HDL cholesterol and apolipoprotein B values in early childhood, whereas dietary intervention had no effect on tracking of any of the lipids. A child’s sex influenced tracking only of HDL cholesterol, with boys showing stronger tracking. (Arterioscler Thromb Vasc Biol. 2002;22:492-498.)

Key Words: children ■ diet ■ cholesterol ■ apolipoprotein E ■ tracking

Although tracking of serum cholesterol concentrations in childhood is well documented,1–9 the effects of exogenous factors like dietary modification on the tracking of serum lipid and lipoprotein concentrations have remained poorly characterized. Tracking of serum cholesterol concentrations is stronger in infants during exclusive breast feeding and weaker in infants receiving formula and solid foods,2 indicating that dietary factors may influence the tracking phenomenon.

ApoE plays a key role in lipoprotein metabolism and in atherogenesis.10,11 Three common codominant alleles at a single gene locus, ε2, ε3, and ε4, determine the 6 apoE phenotypes, E2/2, E2/3, E2/4, E3/3, E3/4, and E4/4. In the same order, the mean concentrations of serum cholesterol increase with the apoE phenotype.12,13 During childhood, the apoE polymorphisms influence not only serum lipid and lipoprotein patterns13–15 but also the tracking of serum lipids and lipoproteins.9,16 The apoE alleles are apparently also able to modulate the linkage between obesity and other lifestyle factors and serum lipoprotein concentrations.9

The main target of the Special Turku Coronary Risk Factor Intervention Project (STRIP) is reduction of the exposure of the intervention group to known environmental atherosclerosis risk factors.17,18 We have recently shown that serum cholesterol concentration increased only slightly with age in the children in the intervention group, who were counseled to consume a low–saturated fat, low-cholesterol diet since infancy, whereas the values increased more in the control children during the first 5 years of life.18,19 We have also demonstrated that the apoE phenotype already has an effect on serum cholesterol concentrations in infancy,14 but that the dietary intervention influences the values in the 13-month-old children independently of their apoE phenotypes.14 We have now examined how strongly repeated dietary counseling, sex, and the apoE phenotype influence the tracking of serum lipids and lipoproteins during the first 5 years of life in this prospective study project.

Study Design
As described previously in detail,17,18 1062 seven-month-old infants were randomized into a control group (n=522) or an intervention group to known environmental atherosclerosis risk factors.17,18

Received August 22, 2001; revision accepted November 28, 2001.

From the Research Centre of Applied and Preventive Cardiovascular Medicine (L.R.-N., A.T., P.S., T.R.) and the Departments of Pediatrics (L.R.-N., A.T., P.S., T.R., I.V., O.S.), Medicine (J.V., T.R.), and Biostatistics (H.H.), University of Turku, Turku, Finland; the Department of Pediatrics (E.J.), University of Helsinki, Helsinki, Finland; and the Research and Development Centre of Social Insurance Institution (J.M.), Turku, Finland.

Correspondence to Leena Rask-Nissilä, MD, Research Centre of Applied and Preventive Cardiovascular Medicine, University of Turku, Kiinamyllynkatu 10, FIN-20520 Turku, Finland. E-mail leena.rask-nissila@utu.fi

© 2002 American Heart Association, Inc.

Arterioscler Thromb Vasc Biol. is available at http://www.atvbaha.org DOI: 10.1161/hq0302.104516
group (n = 540). The intervention group received individualized dietary counseling (see below). The families in the intervention and control groups met a pediatrician and a diettian at 1- to 3-month and 4- to 6-month intervals, respectively. After the child reached 2 years of age, the visits in both groups occurred at 6-month intervals. The parents and the personnel of the day care centers recorded the child’s food consumption in a 3-day food record when the child was aged 8, 13, and 18 months; thereafter, a 4-day food record, always including at least 1 weekend day, was provided twice a year. The records were analyzed as described. Blood samples for serum lipid measurements were drawn yearly. 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.

Counseling

The details of the dietary counseling have been described. Briefly, the aims of the counseling of the intervention group were as follows: a fat intake of 30% to 35% of daily energy, a saturated/monounsaturated/polyunsaturated fatty acid ratio of 1:1:1, and a cholesterol intake < 200 mg/d. The families were advised to continue breast feeding or to use formula until the age of 12 months and to use skim milk (0.5 to 0.6 L/d) thereafter. The parents were taught to add 2 to 3 teaspoonfuls of soft margarine or vegetable oil, mainly low–erucic acid rapeseed oil, to the food of the 12- to 24-month-old children. The control families received the routine health education given to all Finnish families at the well-baby clinics. The mothers were advised to continue breast feeding or to use formula until the child had reached the age of 12 months, but cow’s milk containing at least 1.9% fat (1.5% fat after May 1995) was recommended thereafter. In both groups, at least partial breast feeding continued for 5-24 months (mean = SD). All children were fully weaned by 13 months of age.

Serum Lipids

Before the child had reached 5 years of age, nonfasting blood samples were drawn for measurement of serum cholesterol, HDL cholesterol, and apoA-I and apoB. Therefore, non-HDL cholesterol (total cholesterol – HDL cholesterol) values were used instead of LDL cholesterol values. ApoE phenotypes were determined by using isoelectric focusing. All analyses were performed in the laboratory of the Research and Development Center of the Social Insurance Institution in Turku, Finland, which regularly cross-checked lipid determinations with the World Health Organization reference laboratories in Prague, Czech Republic, and in Helsinki, Finland (Labquality).

Statistical Analysis

At 5 years of age, 764 children continued in the study. Because breast feeding leads to high serum cholesterol concentrations, the starting age of the tracking analysis was 13 months, when all children had been weaned. Thus, the children whose blood samples were successfully obtained at all of the ages (13 months and 2, 3, 4, and 5 years) were included in the analysis. Thus, serum cholesterol, HDL cholesterol, and apoE analyses included complete data on 519 children, which were evenly distributed between the intervention (n = 265, 143 boys) and control (n = 254, 131 boys) groups. Because of insufficient sample size, apoA-I and apoB analyses contained data on 508 children.

The children were divided into 8 groups according to intervention and apoE phenotypes: apoE4-negative (E2/3 or E3/3, n = 345) boys and girls of the intervention and control groups and apoE4-positive (E3/4 or E4/4, n = 174) boys and girls of the intervention and control groups. Because of the small number of children with phenotypes E2/2 (n = 1) and E2/4 (n = 12), they were excluded from the analyses. The distributions of the phenotypes and the sexes were similar in the intervention and control groups (P = 0.10 and P = 0.53, respectively). A greater proportion of the boys than of the girls were apoE4 negative (196 [72%] and 149 [61%], respectively; P = 0.010).

Separately, at every measurement age, a “risk group” of children was defined as those having serum cholesterol, non-HDL cholesterol, or apoB concentrations in the highest quartile of the distribution of the values. Correspondingly, for HDL cholesterol, apoA-I, and the ratios of HDL to total cholesterol and of apoA1 to apoB, the lowest quartile was defined as the risk group. Because of the age-related changes in the concentrations of serum lipids, the risk quartiles were defined separately for boys and for girls at every age point of measurement.

The differences between the intervention and control groups at different ages were calculated by using ANOVA for repeated measurements. The strength of tracking was studied by using the following methods: In all of these methods, differences between children at 13 months of age are taken into account by modeling (in first approach) or by including the observation at 13 months of age into the calculations (in the second and third approach).

First, the odds ratio (OR) was calculated for belonging to a risk group at any of the ages (2, 3, 4, and 5 years) if the child was or was not in the risk group at the age of 13 months. This calculation was performed by fitting a logistic regression model, in which belonging to the risk group at age 13 months was a predictor variable for belonging into the same risk group at the older ages also. Predicting whether a child will belong to a certain risk group at later ages when he or she belongs to a certain risk group at the age of 13 months means that we work with correlated binary observations because the follow-up data are from the same subjects. We must take this into account in the estimation of parameters in the logistic models; otherwise, the standard errors will be too small (which would further lead to a too-small probability value and too-narrow CIs). The generalized estimation method has been developed to solve the problem. The dependence of the classifications at the 4 follow-up ages was taken into account by applying the generalized estimation method for logistic regression analysis of binary repeated measurements. Thus, the OR describes the tracking corresponding to the odds of staying at the risk group, if the child initially belonged to the risk group, compared with the situation when the child initially did not belong to the risk group. The differences between the groups in tracking were analyzed by testing interactions in the logistic models.

Second, a separate quantification of tracking for the 3 groups (the lowest, highest, and the 2 intermediate quartiles) was made by calculating the proportion of agreement for each group. The proportion of agreement was estimated by determining the proportion of subjects who belong to the group at every age point from all who belong to that same group at least at 1 other age point. The proportion describes the stability of the phenomenon.

Third, intraclass correlation coefficient (ICC) for the variables was calculated on continuous measurements that were standardized according to the mean and standard deviation specific for the age. The ICC summarizes how large a fraction the between-children variation is of the total variation of measurements made in all children at all time points (ie, the variation between subjects plus the variation within subjects). The value of the coefficient varies between 0 and 1. If the variation of measurements within subjects is small compared with the variation between subjects, the coefficient is near 1.0. The estimate for variation between subjects and within subjects was computed by using ANOVA.

The results are shown as mean ± SD. The differences were considered significant at P < 0.05. The SAS (release 6.12) program package (SAS Institute) was used.

Results

Serum Lipid and Lipoprotein Levels

The children in the intervention group had lower serum cholesterol concentrations than did the children in the control group (Figure 1). The girls had higher serum non-HDL cholesterol concentrations and lower apoA1 to apoB ratios than did the boys (Figure 1). ApoE phenotype influenced all serum lipid concentrations studied, except the HDL cholesterol concentration (Figure 2). ApoE4-positive children had higher concentrations of serum cholesterol, non-HDL cholesterol, and apoB and a lower concentration of serum apoA1. The ratios of HDL cholesterol to total cholesterol and of
apoA-I to apoB for apoE4-positive children were lower than those for apoE4-negative children.

Tracking of Serum Lipid and Lipoprotein Levels
Tracking of all serum lipids and lipoproteins studied was of the same magnitude in the intervention and control groups. However, as a result of the dietary intervention, the concentrations of serum cholesterol for the children in the intervention group tracked on a lower level than did those for the children in the control group. Sex influenced only the tracking of HDL cholesterol ($P=0.018$), inasmuch as the tracking in boys was stronger than the tracking in girls. The apoE phenotype had an effect on tracking of non-HDL cholesterol and apoB, the ORs were greater in the apoE4-negative children than in the apoE4-positive children (for non-HDL cholesterol, ORs 30.7 [95% CI 15.3 to 61.5] and 13.2 [95% CI 6.1 to 28.6], respectively; and for apoB, ORs 23.0 [95% CI 11.8 to 44.8] and 7.3 [95% CI 3.6 to 14.8], respectively).

Strength of Tracking

Odds Ratios
ORs were calculated to quantify the associations between the study group, sex, apoE phenotype, and tracking (Table 1). When a 13-month-old child belonged to a risk group, the OR for belonging to the risk group at older ages was greatest when the ratio of HDL cholesterol to total cholesterol was studied. Tracking of non-HDL cholesterol was also strong. For HDL cholesterol, the boys showed stronger tracking (ORs for the boys and girls, 10.9 [95% CI 6.1 to 19.6] and 4.1 [95% CI 2.3 to 7.7], respectively). Similarly, for non-HDL cholesterol and apoB, the ORs were greater in the apoE4-negative children than in the apoE4-positive children (for non-HDL cholesterol, ORs 30.7 [95% CI 15.3 to 61.5] and 13.2 [95% CI 6.1 to 28.6], respectively; and for apoB, ORs 23.0 [95% CI 11.8 to 44.8] and 7.3 [95% CI 3.6 to 14.8], respectively).

Proportions of Agreement
To assess the impact of the apoE phenotype on tracking, the proportions of agreement were determined by calculating the proportions of those children who belonged to the specific subgroups (the lowest quartile, the highest quartile, or the 2 intermediate quartiles) of non-HDL cholesterol or apoB distributions at all age points to all children who at least at 1 age point belonged to the same subgroups (Table 2). Thus, the proportions describe the stability of the values in children with different apoE phenotypes. In the lowest quartiles of non-HDL cholesterol and apoB, tracking was significantly stronger in the apoE4-negative children than in the apoE4-positive children, whereas in the highest quartiles, tracking was stronger in the apoE4-positive children than in the apoE4-negative children, or tracking was of the same magnitude in the 2 groups. Similarly, a greater proportion of the apoE4-negative children than of the apoE4-positive children, who belonged to the lowest quartiles at age 13 months, always belonged to the very same quartiles (Figure 3). In the highest quartiles, the difference was less apparent. In the intervention group, the proportions of agreement of the apoE4-positive boys were greater than those of the apoE4-negative boys in the lowest quartiles of non-HDL cholesterol and apoB.

Intraclass Correlation Coefficients
ICCs were calculated to compare the within-children variation with the between-children variation in serum lipid values. The stronger the tracking, the greater was the coefficient. For HDL cholesterol, the ICCs of the boys were greater than those of the girls (Table 3). Similarly, for non-HDL cholesterol and apoB, the ICCs of the apoE4-negative children were in general greater than those of the apoE4-positive children. The only exception, again, were the boys of the intervention group, because the ICCs of the apoE4-positive boys were greater than those of the apoE4-negative boys.

Discussion
This sample of 519 children in the randomized, ongoing, long-term STRIP study shows that the tracking phenomenon of serum lipid and lipoprotein concentrations is already strong between 13 months and 5 years of age. Tracking of serum non-HDL cholesterol and apoB concentrations varies according to the apoE phenotype, and the sex of the child has an effect on tracking of HDL cholesterol concentrations. However, counseling aimed at reducing the intake of saturated fat and cholesterol does not influence the tracking phenomenon.

Our findings are consistent with previous reports concerning the tracking of serum lipids and lipoproteins. Tracking...
was strongest for the ratio of HDL cholesterol to total cholesterol and for non-HDL cholesterol. Similarly, tracking was strongest for LDL cholesterol in the Bogalusa Heart Study, in which a newborn cohort was followed without any interventions from birth to 7 years of age, and in a recent Finnish study, in which the children were followed from birth to the age of 11 years. In the Cardiovascular Risk in Young Finns Study, in which 883 subjects aged 3 to 18 years at the onset of the study were followed up without interventions for 12 years, tracking was strongest for the ratio of LDL cholesterol to HDL cholesterol.

The dietary intervention as exercised in the STRIP project had no effect on the tracking phenomenon. Tracking of all serum lipids and lipoproteins measured was of the same magnitude in the intervention and control groups. However, because of the successful dietary intervention, the concentrations of serum cholesterol of the intervention group tracked at a lower level than did those of the control group. The few earlier studies investigating the associations between diet and tracking of serum lipid concentrations in children have approached the question only for data collected during infancy. Serum cholesterol concentrations in infants receiving a relatively constant diet, eg, during exclusive breast feeding, track better than do the values in infants weaned to formula and mixed solid foods. However, after weaning, children’s relative serum cholesterol values soon stabilize, and the

### Table 1. OR for Belonging to the Risk Group at Older Ages When a Child Belonged to the Risk Group at 13 Months of Age

<table>
<thead>
<tr>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>15.6*</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>6.8*</td>
</tr>
<tr>
<td>HDL cholesterol/total cholesterol</td>
<td>23.1–66.0</td>
</tr>
<tr>
<td>Non-HDL cholesterol</td>
<td>24.2*</td>
</tr>
<tr>
<td>ApoA-I</td>
<td>5.7*</td>
</tr>
<tr>
<td>ApoB</td>
<td>16.3*</td>
</tr>
<tr>
<td>ApoA-I/apoB</td>
<td>18.8*</td>
</tr>
</tbody>
</table>

A child belongs to the risk group when the values of serum cholesterol, non-HDL cholesterol, or apoB are in the highest quartile or when the values of serum HDL cholesterol, HDL/total cholesterol, apoA-I, or ApoA-I/apoB are in the lowest quartile. The odds are the odds of belonging to the risk group vs not belonging to the risk group after the age of 13 months. The odds ratio is the ratio of the odds in the risk group vs the non-risk group at the age of 13 months.

*P<0.0001.
tracking of serum cholesterol concentration becomes of the same magnitude as in older children and adolescents.

Little has previously been known of the impact of the sex of the child on tracking of serum lipids and lipoproteins during childhood. In the present study, the HDL cholesterol values showed better tracking in the boys than in the girls. In the Cardiovascular Risk in Young Finns Study, in which samples were collected at 3-year intervals, males showed better 12-year tracking than did females. Although the sex difference in tracking was most marked for cholesterol, the difference in tracking was most marked for cholesterol, the tracking of all lipid variables differed between the sexes. In contrast to that study, females in the Amsterdam Growth and Health Study showed better 12-year tracking than did females. Although the sex difference in tracking was most marked for cholesterol, the tracking of all lipid variables differed between the sexes. In contrast to that study, females in the Amsterdam Growth and Health Study showed better tracking coefficients for HDL cholesterol than did males over a 15-year follow-up period. However, because the age of the subjects in that study was 13 years at the onset of follow-up, the sex differences in pubertal development are strong confounders, because of the puberty-induced marked changes in serum lipid values.6

According to several studies, the apoE phenotype clearly influences serum lipid and lipoprotein patterns as early as childhood.13–15 In Finnish newborns, serum cholesterol levels are low and do not differ between apoE phenotypes, but in 3-year-old children, the concentration of serum cholesterol increases with apoE phenotype in the order of E3/2, E3/3, E4/3, and E4/4.28 Furthermore, the apoE phenotype modulates tracking of serum lipids and lipoproteins. The apoE phenotype strongly influenced the correlation between the LDL cholesterol values measured in children during the first 11 years of life,16 because the children with phenotypes E2/3 or E2/4 maintained their relative cholesterol levels throughout childhood compared with the children with phenotype E3/3, and even more variation was seen in children with phenotypes E3/4 or E4/4. However, the data were based on only 2 measurements after the age of 1 year, made at the ages of 5 and 11 years.18 In the Bogalusa Heart Study, the apoE phenotype had an apparent effect on the persistence in ranks of the LDL cholesterol over the 16-year follow-up of the children and adolescents.9 Especially at the lowest quartile of LDL cholesterol distribution, the persistence in ranks over time was higher in children with E2/3 and E2/2 phenotypes compared with that in the children who had phenotypes E3/3, E3/4, or E4/4. On the contrary, at the highest quartile the apoE2 group showed a somewhat lower, although nonsignificant, persistence in ranks over time than children in the other phenotype groups. Furthermore, apoE phenotype modulates the change in serum cholesterol concentrations during adolescence, because E2/3 girls aged 8 to 14 years at the onset of follow-up showed a different pattern of change in serum cholesterol during the 4 years of follow-up than did E3/3 and E3/4 girls.79

Our data for 1- to 5-year-old children also show that the tracking of serum non-HDL cholesterol and apoB concentrations of the apoE4-negative children was stronger than that of the apoE4-positive children. Furthermore, tracking of serum non-HDL cholesterol and apoB concentrations of the apoE4-negative children was stronger, especially in the lowest quartiles of non-HDL cholesterol and apoB distributions, whereas tracking in the highest quartiles was weaker in the apoE4-negative children than in the apoE4-positive children. This finding may at least partly be explained by the greater intestinal cholesterol absorption by subjects with phenotypes E4/3 and E4/4 than by subjects with other phenotypes; thus,

<table>
<thead>
<tr>
<th></th>
<th>Intervention</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>E4−</td>
<td>E4+</td>
</tr>
<tr>
<td>Girls, n</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-HDL cholesterol</td>
<td>73</td>
<td>49</td>
</tr>
<tr>
<td>Highest quartile</td>
<td>0.40</td>
<td>0.33–0.46</td>
</tr>
<tr>
<td>Intermediate quartiles</td>
<td>0.45</td>
<td>0.41–0.49</td>
</tr>
<tr>
<td>Lowest quartile</td>
<td>0.35</td>
<td>0.30–0.41</td>
</tr>
<tr>
<td>Boys, n</td>
<td>112</td>
<td>31</td>
</tr>
<tr>
<td>Non-HDL cholesterol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest quartile</td>
<td>0.29</td>
<td>0.22–0.35</td>
</tr>
<tr>
<td>Intermediate quartiles</td>
<td>0.45</td>
<td>0.42–0.49</td>
</tr>
<tr>
<td>Lowest quartile</td>
<td>0.46</td>
<td>0.42–0.50</td>
</tr>
<tr>
<td>ApoB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest quartile</td>
<td>0.38</td>
<td>0.31–0.44</td>
</tr>
<tr>
<td>Intermediate quartiles</td>
<td>0.44</td>
<td>0.41–0.48</td>
</tr>
<tr>
<td>Lowest quartile</td>
<td>0.36</td>
<td>0.32–0.40</td>
</tr>
</tbody>
</table>

*E4− indicates phenotypes E2/3 or E3/3; E4+, phenotypes E3/4 or E4/4.*
subjects with the e4 allele are more sensitive to variations in the diet. Furthermore, the cholesterol-lowering effect of the e2 allele is 2 to 3 times stronger than the cholesterol-raising effect of the e4 allele.10 The greater proportions of agreement and intraclass correlation coefficients of the apoE4-positive boys in the intervention group than those of the apoE4-negative boys in the intervention group in the present study (a finding that differs from the general rule) suggest that the interrelationships between diet, apoE phenotype, and tracking of serum non-HDL cholesterol and apoB concentrations may differ in males and females at least in early childhood. In a population-based sample of 9- to 24-year-old free-living subjects, the influence of the apoE phenotype on serum lipid concentrations varied according the consumption of saturated fatty acids and cholesterol,31 but the effect was similar in both sexes. However, the links between these factors and tracking of serum lipids in early childhood have previously remained unexplored.

We conclude that the predictability of serum lipid values at the age of 13 months is to some extent determined by the apoE polymorphism during the 4-year follow-up. However, the dietary intervention aimed at decreasing the intake of saturated fat and cholesterol had no effect on the tracking of serum lipid values. Sex influenced only the tracking of HDL cholesterol, inasmuch as the boys showed better tracking than did the girls.

**Figure 3.** The apoE4-negative (E4−, stippled bars) and apoE4-positive (E4+, solid bars) children who initially belonged to the lowest or highest quartiles of non-HDL cholesterol or apoB at the age of 13 months. The lowest quartile of non-HDL cholesterol consists of 103 E4− children and 26 E4+ children, and the highest quartile consists of 67 apoE4− and 63 apoE4+ children. The lowest quartile of apoB consists of 94 E4− children and 23 E4+ children, and the highest quartile consists of 69 E4− and 59 E4+ children. Proportions of those children who later also belonged to the same quintile are shown.

**TABLE 3.** ICCs of the E4− and E4+ Girls and Boys of the Intervention and Control Groups

<table>
<thead>
<tr>
<th></th>
<th>Intervention</th>
<th>Control</th>
<th>Boys</th>
<th>Children</th>
<th>Control</th>
<th>Boys</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Girls</td>
<td></td>
<td></td>
<td>Girls</td>
<td></td>
<td></td>
<td>Girls</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>E4− (n=73)</td>
<td>E4+ (n=49)</td>
<td>E4− (n=76)</td>
<td>E4+ (n=47)</td>
<td>E4− (n=112)</td>
<td>E4+ (n=31)</td>
<td>E4− (n=84)</td>
</tr>
<tr>
<td></td>
<td>0.61</td>
<td>0.48</td>
<td>0.65</td>
<td>0.56</td>
<td>0.58</td>
<td>0.67</td>
<td>0.61</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>0.46</td>
<td>0.43</td>
<td>0.54</td>
<td>0.53</td>
<td>0.63</td>
<td>0.55</td>
<td>0.56</td>
</tr>
<tr>
<td>HDL/total cholesterol</td>
<td>0.63</td>
<td>0.54</td>
<td>0.69</td>
<td>0.61</td>
<td>0.65</td>
<td>0.81</td>
<td>0.78</td>
</tr>
<tr>
<td>Non-HDL cholesterol</td>
<td>0.67</td>
<td>0.52</td>
<td>0.71</td>
<td>0.61</td>
<td>0.61</td>
<td>0.70</td>
<td>0.68</td>
</tr>
<tr>
<td>ApoA-I</td>
<td>0.41</td>
<td>0.40</td>
<td>0.48</td>
<td>0.51</td>
<td>0.55</td>
<td>0.46</td>
<td>0.51</td>
</tr>
<tr>
<td>ApoB</td>
<td>0.57</td>
<td>0.45</td>
<td>0.66</td>
<td>0.53</td>
<td>0.55</td>
<td>0.65</td>
<td>0.62</td>
</tr>
<tr>
<td>ApoA-I/apoB</td>
<td>0.59</td>
<td>0.54</td>
<td>0.64</td>
<td>0.57</td>
<td>0.67</td>
<td>0.78</td>
<td>0.78</td>
</tr>
</tbody>
</table>

The total variation of age-adjusted measurements in a follow-up study comes from 2 origins: variation between subjects (BS) and variation within subjects (WS): ICC = BS/(BS + WS). A value of ICC near 1.0 suggests strong tracking.
Acknowledgments

This study was supported by grants from the Mannerheim League for Child Welfare, the Finnish Cardiac Research Foundation, the Foundation for Pediatric Research, Finland, the Academy of Finland, the Yrjö Jahnsson Foundation, the Sigrid Juselius Foundation, the Turku University Foundation, the Juho Vainio Foundation, the Finnish Cultural Foundation and its Regional Fund of Varisnius-Suomi, the Signe and Ane Gyllenberg Foundation, the City of Turku, the Raisio Group Research Foundation, and Van den Bergh Foods Co.

References

Impact of Dietary Intervention, Sex, and Apolipoprotein E Phenotype on Tracking of Serum Lipids and Apolipoproteins in 1- to 5-Year-Old Children: The Special Turku Coronary Risk Factor Intervention Project (STRIP)

Leena Rask-Nissilä, Eero Jokinen, Jorma Viikari, Anne Tammi, Tapani Rönönen, Jukka Marniemi, Pia Salo, Taina Routi, Hans Helenius, Ilkka Välimäki and Olli Simell

doi: 10.1161/hq0302.104516

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://atvb.ahajournals.org/content/22/3/492

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Arteriosclerosis, Thrombosis, and Vascular Biology can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Arteriosclerosis, Thrombosis, and Vascular Biology is online at:
http://atvb.ahajournals.org//subscriptions/