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Serum lipid and apolipoprotein levels in four-year-old children are associated with parental levels and track over time

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Running title Serum lipid levels in children

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ABSTRACT

Background/Objectives: There are only a few studies linking dietary fat intake to serum lipid

30 levels in young children. Our objective was to prospectively evaluate serum lipid levels from infancy to early childhood, and to explore their possible association with dietary, growth, and parental factors.

Methods: Children ($n = 127$) followed from early infancy were examined for serum lipid levels, anthropometry, and dietary intake at 4 y of age. We also studied possible associations with

35 parental anthropometric and blood biochemistry data collected from 122 mothers' and 118

fathers' when children were 4 y of age. **Results:** Serum concentrations of total cholesterol (TC), high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL) and the apolipoprotein B/apolipoprotein A-1 ratio (apo B/apo A-1) showed significant tracking from

infancy to 4 y. Furthermore, children's TC levels correlated with paternal TC level from 6 mo to

40 4 y, but with maternal only at 4 y. In girls, both LDL and HDL correlated with parental LDL and HDL. In all children, intake of saturated fatty acids (SFA) was higher than recommended, and in 90% of the children, polyunsaturated fatty acid (PUFA) intake was lower than recommended.

Conclusions: Serum lipid levels values tracked from infancy to 4 y and were associated with

45 parental values. Higher serum lipid levels at 4 y compared to 6–18 mo of age may result from changes in the quality of dietary fat. We therefore suggest that intake of dietary fat in 4 y old children should be more focused on quality. Furthermore, as there were strong associations between the child and parental serum lipid levels this supports the view that family-based rather than individual intervention is preferable.

50 **Keywords:** Serum lipids, children, parental, dietary fat, tracking

INTRODUCTION

The relationships between childhood dietary intake, serum lipid levels, obesity, and long-term risk for cardiovascular disease (CVD) are complex (Berenson *et al.*, 1998; Burke *et al.*, 2001;

Burke *et al.*, 2005). Overweight children have lower levels of high-density lipoprotein cholesterol (HDL) and higher levels of serum triglycerides (TG) compared to normal-weight children (Freedman *et al.*, 1999; Burke *et al.*, 2005).

Serum lipid levels differ between girls and boys already in early childhood, and dietary fat intake seems to affect girls and boys differently (Rogers *et al.*, 2001; Niinikoski *et al.*, 2007; Öhlund *et*

al., 2008). Total dietary fat intake, particularly higher intake of saturated fatty acids (SAFA), are positively associated with total serum cholesterol (TC) levels (Cowin *et al.*, 2001; Rogers and

Emmett 2001; Niinikoski *et al.*, 2007). Studies in Sweden (Enghardt *et al.*, 2006; Garemo *et al.*, 2006), as well as other countries (Agostoni *et al.*, 2000; Cowin and Emmett 2001), report that

intakes of SAFA are higher than the recommended $\leq 10\%$ of total energy intake (10 E%), while

intakes of polyunsaturated fatty acids (PUFA) are close to or below the recommended minimum intake of 5 E%. Both tracking (associations over time) from early infancy and heredity may be

additional important predictors of serum lipid levels later in life. In a longitudinal study, we

followed healthy Swedish infants from 6 mo of age and found that a higher intake of PUFA was associated with lower total serum cholesterol (TC) and low-density lipoprotein cholesterol

(LDL) levels at 12 mo (Öhlund *et al.*, 2008). In this longitudinally followed study group, we investigated whether these associations remained later in life and whether there was any

association with parental serum lipid levels.

The main objectives in the present follow-up study were to evaluate serum lipid levels from infancy to early childhood, i.e., 4 y of age; determine possible associations with dietary, growth,

75 and parental factors; and examine possible tracking of serum lipid levels from infancy to childhood.

METHODS

Study cohort

80 This was a follow-up of 127 healthy children (63 girls and 64 boys) at 4 y of age, who were prospectively followed from early infancy. Full details are published elsewhere (Lind *et al.*, 2003; Öhlund *et al.*, 2008).

Of the original 300 children 234 children (80%) completed monthly dietary and anthropometrical registrations from 6 to 18 mo of age. These children and their parents were invited to participate
 85 in the present follow-up study at 4 y of age starting in 2001. The parents of 127 children (54%) consented to renewed dietary, anthropometric, and biochemical measurements of the children. Of the parents, 240 (122 mothers and 118 fathers) consented to anthropometric and biochemical measurements when their child was 4 y. Mean maternal and paternal age was 33.7 y (4.5) and 35.3 y (5.5), respectively. The participating 4-y-olds were not different from the non-participating
 90 children with respect to weight and length at birth, 12 mo, and 18 mo; duration of breastfeeding; or with respect to dietary fat intake and serum lipid levels at 12 and 18 mo. However, a higher proportion of parents had university educations among the families who participated in the 4-y follow-up compared to those who did not (33% vs. 25%, $p < 0.001$). Of the 107 non-participating children, 12 were sick at the follow-up, 16 had moved from the area; of the remainder, the
 95 parents declined to participate without explanation.

The study was approved by the local Ethics Committee of the Faculty of medicine, Umeå University, Sweden.

Anthropometric data and biochemical analyses

100 Between 6–18 mo, the participating children were measured monthly. They were weighed to the nearest 0.01 kg using a Seca 835 digital baby scale, and recumbent length was measured to the nearest millimeter using a Harpenden infantometer (CMS Weighing Equipment, London, UK). At 4 y, weight was measured to the nearest 0.1 kg using a Seca 835 digital adult scale (Seca, Hamburg, Germany) and height to the nearest millimeter was measured using a CMS stadiometer (CMS Weighing Equipment). Mid upper-arm circumference was measured using a plastic measuring tape. Subcutaneous fat was measured by subscapular and triceps skin-fold thickness using a Harpenden skinfold caliper (Baty International, West Sussex, UK). Body mass index (BMI) was calculated and cut-off levels for overweight/obesity in children were set separately for girls and boys using the definition suggested by the International Obesity Task Force (Cole *et al.*, 2000). These cut-off points for overweight and obesity (iso-BMI ≥ 25 and ≥ 30 , respectively) were adjusted for age and gender and corresponded to the well-known thresholds of BMI ≥ 25 kg/m² and BMI ≥ 30 kg/m² for overweight and obesity, respectively, among adults.

115 Venous blood samples were taken at least 2 h after a meal from the children at 6, 12, and 18 mo, and at 4 y they were taken while the children and their parents were in a fasting state. Sera were stored at -20 °C until analysis. Serum lipids were analysed for TC, HDLC, apolipoprotein A-1 (apo A-1), apolipoprotein B (apo B), and TG by the Roche/Hitachi method, which is the routine analysis method used at the department of Clinical Chemistry at Umeå University hospital. 120 LDLC was calculated using the Friedewald formula (Friedewald Levy and Fredrickson 1972).

Dietary assessment

We assessed dietary intake in the children using monthly 5-d food records from 6 to 18 mo and then once at the 4 y follow-up. Product names were used; for example, the names of spreads, ice creams, candies, yogurts, and meals at fast-food restaurants. The parents used a booklet with photos of serving sizes of meals, snacks, etc., but also used standard household measures for quantities as described earlier (Lind *et al.*, 2003). If the participating child attended day care, the dietitian visited the centre to inform the staff of how to record dietary intake. Data regarding breastfeeding were recorded prospectively, except for children who had stopped breastfeeding before inclusion in the study at 6 mo of age. Each intake of breast milk was recorded either as a “meal” or a “snack” estimated as 134 ml per meal up to 8 mo of age, then 102 g per meal beyond 8 mo, and as 25 g per snack at all ages (Lind *et al.*, 2003). Nutrient intakes from breast milk were calculated in accordance with Jensen (1995) and Tsang *et al.* (1997). Breastfeeding at 6 mo was recorded as exclusive if no other foods were introduced, and partial if foods or formula had been introduced. Breastfeeding duration was used for establishing associations between breastfeeding and serum lipid levels. Energy and nutrient intake were calculated with the MATs S406 software program (Rudans Lättdata, Västerås, Sweden), which uses the food composition database of the Swedish National Food Administration, as well as special products for children and other products not included in the original database. Dietary fat intakes were described as percent of total energy (E%) and PUFA, mono saturated fatty acids (MUFA), and SAFA were described as proportions (%) of total fat. To evaluate the accuracy of the reported energy intake, we calculated the ratios of energy intakes (EI) to basal metabolic rate (BMR) in girls and boys (Livingstone *et al.*, 2003).

Calculations and statistical analyses

Statistical analyses were performed using SPSS 15.0 (SPSS, Chicago, IL, USA). Mean and standard deviation (SD) were used to describe serum lipid and apolipoprotein levels and dietary fat intakes. Gender difference and changes in dietary fat intake over time were analysed by Student's *t*-test. The skinfold measurements were skewed and were therefore log transformed before analysis. Pearson's correlation was used to analyse associations of serum lipids over time (tracking) and their association with parental levels. Paired sample *t*-test was used for changes in variables over time. Data reduction of dietary fat intakes were used and presented as mean daily intakes during the 6–9 mo, 10–14 mo, 15–18 mo and 4 y periods. The relationships between serum lipids and apolipoprotein levels at 4 y and dietary fat intake from 6 m to 4 y, duration of breastfeeding, the child's BMI at 4 y, parental serum lipid levels, and parental BMI were investigated using univariate regression analysis. Factors significantly associated ($p \leq 0.05$) with the children's serum lipids and ApoB/apoA-1 levels in the univariate analysis were included in a stepwise multivariate regression analysis.

RESULTS

Serum lipids and anthropometrics

Mean TC levels were stable during infancy. From 18 mo to 4 y, serum TC, LDLC, HDLC, ApoA-1, and ApoB levels increased ($p \leq 0.001$) while TG levels decreased ($p \leq 0.001$) (**Table 1**). The TC, HDLC, LDLC, Apo A-1 and Apo B levels as well as the TC/HDLC and Apo B/Apo A-1 ratios showed significant tracking over time. Furthermore, the children's TC levels were correlated with their father's TC level from 6 mo–4 y, but with their mother's only at 4 y. At 4 y, girls' but not boys' HDLC was positively correlated with maternal HDLC. Furthermore girls', but not boys' LDLC levels were positively correlated with parental LDLC. (**Table 2**)

At 4 y, 16% of the children were overweight (iso-BMI ≥ 25) with no gender difference. Among parents, overweight was more common among fathers compared to mothers (47% vs 28%, $p < 0.05$). BMI in children was positively correlated with mid-upper arm circumference ($r = 0.80$, $p < 0.001$), subscapularis skinfold ($r = 0.58$, $p < 0.001$), and triceps skinfold ($r = 0.66$, $p < 0.001$). No correlations were found between the children's serum lipid levels and growth rate, any of the other anthropometric parameters, or parental BMI (data not shown).

At 4 y, TC levels >5.2 mmol/l were observed in 22% (seven boys and 17 girls) of the children, in 49% of the fathers, and in 32% of the mothers. LDLC >3.34 mmol/l was observed in 24% of the children. The children with higher TC (>5.2 mmol/l) had significantly higher LDLC, HDLC, and apo B levels and apo B/apo A1-ratios, compared to children with TC <5.2 mmol/l (data not shown).

Dietary intakes

At 6 mo, five infants were exclusively breastfed, 97 were partially breastfed, and 24 no longer breastfed. The mean (SD) duration of breastfeeding was 8.7 (3.4) mo, and two children had not been breastfed at all. Mean (SD) daily energy intake at 4 y was 1462 (240) and 1382 (228) kcal per day in boys and girls, respectively; the calculated ratio of mean EI:BMR was 1.61 in both boys and girls.

Mean dietary fat intake decreased from 42 E% at 6 mo to 33 E% at 4 y (**Table 3**). However, as dietary fat (E%) decreased, the proportion of SAFA increased significantly ($p \leq 0.001$), whereas the proportions of MUFA and PUFA decreased ($p \leq 0.001$ and $p \leq 0.001$) (**Figure 1**). The intake of SAFA was higher than recommended in all children and the intake of PUFA was lower in most of the children (NNR 2004). At 4 y, none of the children had an intake of SAFA below the

recommended upper level of 10 E% whereas only 10% of the children had a total PUFA intake above the recommended lower level of 5 E%. For most children, the intake of MUFA was within the recommended limits (NNR 2004) .

Associations between children's serum lipid levels and dietary fat intake, and parental serum lipids.

At 6 mo, children who were still partially or exclusively breastfed had higher TC levels compared to those who were not breastfed ($p < 0.001$), but the difference did not remain at 12 mo, 18 mo, or 4 y. Factors associated with children's serum lipid levels were analysed using regression analysis in two models (**Table 4 and 5**). The serum levels of TC, LDLC, and HDLC, and the apo B/apo A-1 ratio were used as dependent variables. Independent variables with ($p < 0.05$) in the univariate model were included in a multivariate model. The child's serum lipid levels at 4 y were associated with both parents' serum lipid levels in the univariate model and in the final multivariate model the child's TC and LDLC were associated with paternal TC and LDLC levels and the child's HDLC with maternal HDLC levels. Duration of breastfeeding was negatively associated with HDLC and positively associated with the apoB/apoA-1 ratio in the univariate model but this did not remain in the multivariate analysis. Higher intake of SAFA before 18 mo was associated with a higher apoB/apoA-1 ratio. In opposite, a higher intake of unsaturated fatty acids (MUFA at 10–14 mo and 15–18 mo, and PUFA at 10–14 mo) was associated with a lower apoB/apoA-1 ratio in the univariate model. In the final analysis, higher intake of SAFA 10-14 mo remained positively associated with the child's apoB/apoA-1 ratio.

DISCUSSION

The main finding in the present study was the correlation between child and parental serum lipid levels. In the study group, which comprised children with a high intake of SAFA and a low intake of PUFA compared to recommended guidelines, TC levels above the cut-offs for abnormal TC (>5.2 mmol/l) (Daniels *et al.*, 2008) were present in 22% of the children, 49% of the fathers, and 32% of the mothers. Furthermore, 24% of the children had LDLC > 3.35 mmol/l, defined as a risk factor for adult dyslipidemia and associated cardiovascular disease (CVD) (Nicklas *et al.*, 2002; Jolliffe and Janssen 2006). This calculated risk factor in the present study group was 4-fold higher compared to the risk factor calculated for the Bogalusa Heart Study (Nicklas, von Duvillard and Berenson 2002). Over time, serum TC, LDLC, HDLC, ApoA-1, and ApoB levels increased significantly. In a previous paper, we reported that a higher intake of PUFA was associated with lower TC and LDLC levels at 12 mo (Öhlund *et al.*, 2008). However, three years later, at 4 y of age, the associations between dietary fat intake and serum lipids and apolipoproteins were weak. Compared with Finnish children on a restricted, low-fat diet (Niinikoski *et al.*, 2007), these children had more favourable lipid levels at 12 mo (Öhlund *et al.*, 2008), but this did not remain until 4 y of age. The reason may be that during the period 6 mo to 4 y, there was a deterioration in the quality of dietary fat intake. Although total dietary fat intake decreased from 6 mo to 4 y, the proportion of SAFA (%) increased whereas the proportions of both MUFA and PUFA (%) decreased with decreasing dietary fat (E%) resulting in lower fat of quality by 4 y of age. Thus there was an inverse relationship, between decreases in the intakes of PUFA and MUFA and increases in the TC and LDLC levels. Moreover, quality of dietary fat intake at 10–18 mo was associated with the apo B/apoA-1 ratio. A higher intake of SAFA at 10–14 mo and 15–18 mo was associated with higher apo B/apoA-1 ratios, and a higher intake of

MUFA and PUFA was associated with lower apo B/apoA-1 ratios at 4 y of age. To our

240 knowledge, few studies have examined associations between dietary patterns and apo B/apoA-1 ratios (Kurvinen *et al.*, 2005); more surveys should be performed.

Another finding of our study was tracking of serum lipids from infancy to early childhood. TC and HDLC levels and apo B/apo A-1 ratios tracked from 6 mo to 4 y. Tracking of the apo B/apo A-1 ratio was strongest, and was stronger in boys than in girls. Apo B and apo A-1 levels and

245 their ratio are possibly superior to TC, HDLC, and LDLC levels in terms of predicting CVD (Estonius *et al.*, 2005; Kallner *et al.*, 2006). Although there are limited data regarding the association between childhood apo B/apo A-1 ratios and long-term effects on CVD risk, they are suggested to have high predictive value in paediatric risk assessments (Frontini *et al.*, 2008; Juonala *et al.*, 2008). Tracking of serum lipids in Swedish children has not been reported
250 previously, but our results are in agreement with the Finnish STRIP study (Rask-Nissila *et al.*, 2002). Further, tracking of serum lipids from childhood into adulthood was demonstrated by the Bogalusa Heart Study (Nicklas, von Duvillard and Berenson 2002). This supports the importance of early interventions in families with known raised serum lipid levels and known CVD risk (Magnussen *et al.*, 2008). However, the cut-off levels for risk assessments in children are not yet
255 clear, although guidelines have been developed by the United States National Heart, Lung and Blood Institute's Pediatric Cardiovascular Risk Reduction Initiative (Kwiterovich 2008).

There were no correlations between the children's serum lipid levels, growth rate, BMI, or parental BMI. This is in accord with a British study in which central obesity, but not BMI, was
260 negatively associated with HDLC levels in boys and positively associated with LDLC levels in girls (Cowin *et al.*, 2000).

A strength of our study is that it was based on prospective longitudinal data for anthropometric and biochemical variables and carefully repeated registrations of dietary intake of the children. A

265 further strength of the study is the anthropometric and biochemical data collected from the parents when the child was 4 y. However, some limitations of this study should be noted. We have no data regarding the physical activity of the children and parents, and no data regarding the parents' diet and could not analyse whether the associations between the child's and their parents are based on diet or genetic factors . Furthermore, we were unable to include more than 127 out
270 of the original 234 children in the follow-up study. The by far most common reason for not participating in the follow-up study was lack of time/willingness to do another 5-day period of diet recording, which may lead to a selection bias. However there were no differences, except for the parents' education level, between the participating and non-participating children when compared at 12 and 18 mo. In addition, assessment of dental health at 4 y compared with
275 population data suggested that there was no significant selection bias affecting the present follow-up (Öhlund *et al.*, 2007). However, we cannot exclude that parents accepting to participate were more health concerned or had higher health awareness than those who did not. .

In conclusion, serum lipid and apolipoprotein levels tracked from infancy to 4 y, and the strongest tracking was for the apo B/apo A-1 ratio. Higher serum lipid levels at 4 y compared to
280 6–18 mo of age may, at least partly, result from changes in the quality of dietary fat. We therefore suggest that intake of dietary fat in 4 y old children should be more focused on quality.

Furthermore, as there were strong associations between the child and parental serum lipid levels this supports the view that family-based rather than individual intervention is preferable.

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Conflicts of Interests

290 The authors declare no conflict of interest.

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TABLES 1- 4

Table 1. Mean serum lipid and apolipoprotein levels in children from 6 mo to 4 y and their parents. Data provided as mean (SD).

	6 mo		12 mo		18 mo		4 y***		Mothers' ¹	Fathers' ¹
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls		
	(n = 58)	(n = 61)	(n = 55)	(n = 57)	(n = 58)	(n = 61)	(n=50)	(n=58)		
TC (mmol/l)	3.36 (0.65)	3.50 (0.76)	3.32 (0.67)	3.46 (0.63)	3.46 (0.70)	3.59 (0.60)	4.48 (0.94)	4.68 (0.69)	4.82 (0.90)	5.23 (0.89)
LDLC (mmol/l)	1.52 (0.73)	1.69 (0.76)	1.74 (0.52)	1.76 (0.62)	1.84 (0.58)	2.00 (0.58)	2.77 (0.60)	2.92 (0.61)	2.77 (0.72)	3.32 (0.75)
HDLC (mmol/l)	0.94 (0.27)	0.86 (0.21)	0.90 (0.32)	0.90 (0.19)	0.96 (0.30)	0.89 (0.23)	1.33 (0.23)	1.39 (0.22)	1.56 (0.4)	1.30 (0.32)
apoA-1 (g/l)	1.02 (0.20)	0.97 (0.14)	0.93 (0.22)	0.95 (0.12)	0.95 (0.21)	0.91 (0.14)	1.2 (0.20)	1.2 (0.19)	n/a	n/a
apoB (g/l)	0.67 (0.18)*	0.75 (0.21)*	0.68 (0.15)	0.72 (0.16)	0.72 (0.15)	0.77 (0.15)	0.79 (0.15)	0.83 (0.16)	n/a	n/a
apoB/apoA1	0.71 (0.44)	0.79 (0.26)	0.85 (0.82)	0.87 (0.24)	0.91 (1.14)	0.87 (0.24)	0.69 (0.25)	0.69 (0.16)	n/a	n/a
TG (mmol/l)	1.96 (0.88)	2.12 (0.97)	1.49 (0.72)	1.77 (0.86)	1.47 (0.87)	1.54 (0.57)	0.85 (0.50)	0.83 (0.31)	1.06 (0.54)	1.33 (0.62)

Abbreviations: TC, total cholesterol; HDLC, high-density lipoprotein cholesterol; LDLC, low-density lipoprotein cholesterol; apo A-1, apolipoprotein A-1 and apo B, apolipoprotein B

¹Parental serum lipids assessed when child was 4 y old, maternal mean age was 33.7 (4.5) y and paternal 35.3 (5.5) y. Apolipoprotein A-1 (apoA-1) and apolipoproteinB (apoB) not available (n/a) for mothers' and fathers'. No significant differences in apoB/apoA1 between 4 y old boys and younger age groups.* $p < 0.05$ between gender at 6 mo. No significant differences between gender at 12 m, 18 m, and 4 y. ***All differences in serum lipids between 4 y of age and the younger ages are significant at $p \leq 0.001$ unless otherwise stated.

Table 2. Tracking of children's serum lipid and apolipoprotein levels and associations with parental values, measured when children were 4 y old.

	6 mo¹		12 mo¹		18 mo¹		4 years¹	
	Boys (n = 61)	Girls (n = 62)	Boys (n = 52)	Girls (n = 56)	Boys (n = 58)	Girls (n = 60)	Boys (n = 47)	Girls (n = 57)
<u>TC</u>								
6 mo			0.41**	0.57***	0.32*	0.41**	0.32*	0.43*
12 mo					0.62***	0.55***	0.50***	0.57***
18 mo							0.72***	0.56***
Mothers'	ns	ns	ns	ns	ns	Ns	ns	0.32**
Fathers'	0.49***	0.28*	0.29*	ns	0.48***	Ns	0.35**	0.32**
<u>HDLC</u>								
6 mo			0.68**	0.50***	0.57***	0.45**	0.54***	0.38**
12 mo					0.75***	0.49**	0.51***	0.6***
18 mo							0.57***	0.51***
Mothers'	ns	ns	ns	ns	0.29*	Ns	ns	0.44**
Fathers'	0.39**	0.40**	0.42**	ns	0.32*	0.37**	ns	ns
<u>LDLC</u>								
6 mo				0.49***	ns	0.36**	ns	0.39**
12 mo					0.37**	0.51***	0.30*	0.57***
18 mo							0.48**	0.51***
Mothers'	ns	ns	ns	0.35*	0.31*	Ns	ns	0.37**
Fathers'	ns	ns	ns	ns	0.33*	0.27*	ns	0.28*
<u>TC/HDLC</u>								
6mo			0.92***	0.69***	0.91***	0.55***	0.58***	0.54***
12 mo					0.93***	0.62***	0.85***	0.71***
18 mo							0.86***	0.51***
Mothers'	ns	ns	ns	0.30*	ns	0.31*	ns	0.49***
Fathers'	0.26*	0.39**	ns	ns	ns	0.31*	ns	ns
<u>ApoA</u>								
6 mo			0.65***	0.34*	0.62***	0.31**	0.61***	0.38*
12 mo					0.78***	0.38**	0.65***	0.41**
18 mo							0.68***	0.43**
<u>ApoB</u>								
6 mo			0.41**	0.64***	0.35**	0.55***	0.33*	0.58***
12 mo					0.57***	0.69***	0.51***	0.68***
18 mo							0.56***	0.65***
<u>Apo B/apoA-1²</u>								
6 mo			0.93***	0.61***	0.91***	0.56***	0.83***	0.56***
12 mo					0.96***	0.65***	0.86***	0.66***
18 mo							0.84***	0.64***

Abbreviations: TC, total cholesterol; HDLC, high-density lipoprotein cholesterol; LDLC, low-density lipoprotein cholesterol; apo A-1, apolipoprotein A-1 and apo B, apolipoprotein B

¹Pearson correlation coefficients, * $p \leq 0.05$, ** $p \leq 0.01$, and *** $p \leq 0.001$, ns = not significant.

²Maternal and paternal apo B and apoA-1 not available.

Table 3. Changes in dietary fat intake as percent of energy (E%) and as proportion of total fat (%) from 6–9 mo to 4 y in Swedish children.

	6–9 mo	10–14 mo	15–18 mo	4 y
	(<i>n</i> = 126)	(<i>n</i> = 124)	(<i>n</i> = 107)	(<i>n</i> = 99)
Total dietary fat (E%)	38.6 (5.4)	29.4 (3.6)	34.6 (3.7)	33.2 (4.0)
SAFA (E%)	16.1 (3.1)	14.5 (2.2)	14.4 (2.0)	14.8 (2.2)
(%)	41.3 (2.8)	40.9 (3.4)	41.6 (3.6)	44.6 (3.7)
MUFA (E%)	15.7 (2.3)	14.0 (1.5)	13.6 (1.4)	12.1 (1.7)
(%)	40.7 (1.8)	40.0 (2.4)	39.2 (2.2)	36.5 (2.5)
PUFA (E%)	6.3 (1.2)	5.7 (1.1)	5.2 (1.6)	4.1 (1.0)
(%)	16.5 (2.9)	16.2 (2.5)	14.9 (2.9)	12.3 (2.5)
n-6 fatty acids (E%)	na ¹	na ¹	na ¹	3.3 (0.92)
n-3 fatty acids (E%)	na ¹	na ¹	na ¹	0.86 (0.78)

Abbreviations: SAFA, saturated fatty acids; PUFA, polyunsaturated fatty acid; MUFA, monounsaturated fatty acid.

5 ¹Data not available.

Data provided as mean (SD).

Table 4. Factors significantly associated with the children's serum lipid and the ratio of apolipoprotein levels measured when the child was 4 y old, adjusted for gender.

Model 1		Univariate analysis		10
Dependent	Independent	R ²	B (s.e.)	
TC	Paternal TC	0.14	0.28 (0.08)***	15
	Maternal TC	0.07	0.17 (0.07)*	
LDLC	Paternal LDLC	0.09	0.22 (0.09)**	
	Maternal LDLC	0.09	0.24 (0.08)**	
HDLC	Duration of breastfeeding	0.08	-0.02 (0.01)*	20
	Fat E% 6 mo	0.07	-0.01 (0.003)**	25
	MUFA E % 6 mo	0.06	-0.02 (0.01)**	
	MUFA E% 15-18 mo	0.13	0.05 (0.02)***	
	Maternal HDLC	0.12	0.19 (0.06)***	
	Paternal HDLC	0.05	0.13 (0.07)*	
ApoB/apoA-1¹	SAFA (%) 10–14 mo	0.08	0.02 (0.01)**	30
	MUFA (%) 10–14mo	0.06	-0.02 (0.01)*	
	PUFA (%) 10–14 mo	0.06	-0.03 (0.01)*	

Abbreviations: TC, total cholesterol; LDLC, low-density lipoprotein cholesterol; HDLC, high-density lipoprotein cholesterol; apo A-1, apolipoprotein A-1 and apo B, apolipoprotein B; SAFA, saturated fatty acids; PUFA, polyunsaturated fatty acid; MUFA, monounsaturated fatty acid. Independent variables included in the univariate regression model were dietary fat intake (E%); SAFA, MUFA, and PUFA as E% and as proportion (%); duration of breastfeeding (mo); and parental serum lipids (TC, LDLC; and HDLC). ¹Parental apo B and apoA-1 not available.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 5. Final multivariate analysis of factors associated with serum lipids and ratio of apolipoprotein levels in four-year-old children, adjusted for gender.

Model 2		Multivariate regression analysis		
Dependent	Independent	R ²	B(s.e)	45
TC	Paternal TC	0.11	0.28 (0.08)***	
LDLC	Paternal LDLC	0.07	0.23(0.09)**	
HDLC	Maternal HDLC	0.08	0.17 (0.62)**	
Apo B/apoA-1¹	SAFA 10-14 mo	0.92	0.02 (0.01)**	

Abbreviations: TC, total cholesterol; LDLC, low-density lipoprotein cholesterol; HDLC, high-density lipoprotein cholesterol; apo A-1, apolipoprotein A-1 and apo B, apolipoprotein B; SAFA, saturated fatty acids; PUFA, polyunsaturated fatty acid; MUFA, monounsaturated fatty acid. Independent variables entered into the analyses were variables with $P < 0.05$ in the univariate regression analysis; dietary fat intake (E%); SAFA, MUFA, and PUFA as E% and as proportion (%); duration of breastfeeding (mo); and parental serum lipids (TC, LDLC; and HDLC), ¹parental apo B and apoA-1 not available. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

FIGURES

Figure 1 Changes in dietary fat intake as a proportion of total fatty acids (TFA) from 6 mo to 4 y of age. The proportion of SAFA increased (mean 3.4%, $p \leq 0.001$) as the proportions of PUFA and MUFA decreased (mean 4.3%, $p \leq 0.001$ and mean 5.0%, $p \leq 0.001$), respectively.

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