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Longitudinal child-oriented dietary intervention: Association with parental diet and cardio-metabolic risk factors. The Special Turku Coronary Risk Factor Intervention Project

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Abstract

Background: The child-oriented dietary intervention given in the prospective Special Turku Coronary Risk Factor Intervention Project (STRIP) has decreased the intake of saturated fat and lowered serum cholesterol concentration in children from infancy until early adulthood. In this study, we investigated whether the uniquely long-term child-oriented intervention has affected also secondarily parental diet and cardio-metabolic risk factors.

Methods: The STRIP study is a longitudinal, randomized infancy-onset atherosclerosis prevention trial continued from the child's age of 8 months to 20 years. The main aim was to modify the child's diet towards reduced intake of saturated fat. Parental dietary intake assessed by a one-day food record and cardio-metabolic risk factors were analysed between the child's ages of 9–19 years.

Results: Saturated fat intake of parents in the intervention group was lower [mothers: 12.0 versus 13.9 daily energy (E%), $p < 0.0001$; fathers: 12.5 versus 13.9 E%, $p < 0.0001$] and polyunsaturated fat intake was higher (mothers: 6.1 versus 5.4 E%, $p < 0.0001$; fathers: 6.3 versus 5.9 E%, $p = 0.0003$) compared with the control parents. Maternal total and low-density lipoprotein cholesterol concentrations were lower in the intervention compared with the control group (mean $\pm$ SE 5.02 ± 0.04 versus 5.14 ± 0.04 mmol/l, $p = 0.04$ and 3.19 ± 0.04 versus 3.30 ± 0.03 mmol/l, $p = 0.03$, respectively). Paternal cholesterol values did not differ between the intervention and control groups. Other cardio-metabolic risk factors were similar in the study groups.

Conclusions: Child-oriented dietary intervention shifted the dietary fat intakes of parents closer to the recommendations and tended to decrease total and low-density lipoprotein cholesterol in the intervention mothers. Dietary intervention directed to children benefits also parents.

Keywords

Dietary counselling, parental, diet, cardio-metabolic risk factors

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Introduction

It has been demonstrated that diets rich in unsaturated fatty acids and low in saturated fatty acids associate with lower risk of cardiovascular events and mortality in adults.[1] Moreover, inclusion of diet improves the ability of a cardiovascular risk score in predicting future events.[2] Uniquely, the longitudinal randomized Special Turku Coronary Risk Factor Intervention Project (STRIP), initiated in 1989, aimed to prevent the development of adverse cardio-metabolic risk factor levels already in childhood, beginning since infancy. The intervention was executed by dietary counselling towards a heart-healthy diet with the main aim on modifying the intervention children's dietary fat quality by recommending foods rich in unsaturated fatty acids instead of foods rich in saturated fatty acids. The at-least-biannually repeated counselling continued from the child's age of 8 months to the age of 20 years. The counselling was naturally first given only to the parents, and after the child's age of 7 years, the children were also met alone. In addition to the careful monitoring of diet and cardio-metabolic risk factors in the children, parental dietary intake and cardio-metabolic risk factors have been repeatedly followed until the children were 19 years of age.

The repeated dietary counselling given in the STRIP study has been effective in decreasing saturated fat intake in the intervention children, and has led to lower serum low-density lipoprotein cholesterol concentration from infancy until 19 years of age.[3] Dietary changes during childhood have additionally been associated with improved insulin sensitivity,[4] lower blood pressure[5] and improved cardiovascular health as determined by the American Heart Association.[6] These results are in line with previous intervention studies in children with similar objectives to influence diet to improve cardio-metabolic health. Both the CATCH[7] (the Child and Adolescent Trial for Cardiovascular Health) and the DISC[8] (the Dietary Intervention Study in Children) studies were able to reduce the intake of saturated fat in the intervention children compared with the controls. In the parents of the STRIP intervention children, we have previously observed decreased saturated fat intake during the first 6 years of the children's life.[9,10] In line, the parents of the intervention children had lower serum total cholesterol concentrations than the parents of the control children already after 1 year since the beginning of the intervention.[11] The difference in the total cholesterol concentration was maintained in the mothers until the children were 5 years old.[9]

Previous studies that have targeted parents in order to modify the home food environment for their children have shown that such interventions have been associated with changes in maternal dietary intake in the short-term (7–18 months of follow up).[12–14] Observed dietary changes have included increased intakes of fruit and vegetables[12,14] and reduced intakes of high-energy and high-fat foods.[13] The evidence of the association of dietary intervention with paternal dietary intake is scarce. In addition, the long-term effects of child-oriented dietary intervention on parental cardio-metabolic health are not known. Therefore, we examined in this study whether the long-term dietary intervention focused on children associates with both parental dietary intake and cardio-metabolic risk factors over two decades of follow up.

Methods

Study design and participants

The prospective, randomized STRIP study originally comprised 1107 infants and their parents recruited at the well-baby clinics in Turku, Finland, between 1989–1992.[15] All participants were White. The families were randomly assigned to an intervention group ($n = 562$ children) or to a control group ($n = 545$ children). Study visits took place at 1–3-month intervals until the age of 2 years. Thereafter, the intervention children and their families were seen biannually. The control group families were seen biannually until the child was 7 years old and once a year thereafter. Repeated dietary intervention continued from the child's age of 8 months until the age of 20 years. Additionally, children were encouraged to have a physically active lifestyle, but it was not a structured, continuous part of the intervention. The primary prevention of smoking was introduced at the age of 8 years. Socioeconomic status was similar between the intervention and control parents.[16]

The Joint Commission on Ethics of the Turku University and the Turku University Central Hospital, Finland, approved the study. Written informed consent was received from the parents in the beginning of the trial and from the children at the age of 15 and 18 years.

Dietary intervention

The individualized dietary counselling was aimed to decrease the development of adverse cardio-metabolic risk factor levels in the intervention child. During the first years of the trial, the intervention provided by the nutritionist was given to the parents. The intervention was begun at child's age of 8 months and lasted until the age of 20 years. From the age of 7 years onwards counselling was also given to the intervention children, who were met alone without the parents. Parents were carefully informed about the contents of the sessions and encouraged to discuss the same food-related topics

with their child at home. The main focus of the dietary intervention was to reduce the intake of saturated fat and concomitantly increase the unsaturated fat intake of the child. Additionally, the children were encouraged to consume more vegetables, fruits, berries, and wholegrain products, and to lower salt intake. At the beginning of the trial the intervention aimed at achieving a fat intake of 30–35% of daily energy (E%), with a ratio of saturated to monounsaturated plus polyunsaturated fatty acid of 1:2 and cholesterol intake <200 mg/d.

Parental dietary intake

Parental dietary intake was assessed by a 1-day food record biennially between child's ages 9 and 19 years. Prior to this, a 24 h dietary recall was used until the child's age of 7 years. Due to the different assessment method and previously reported results related to the 24 h recall[9–11] we used in this study parental dietary data assessed by the food record beginning of the child's age of 9 years. Completeness and accuracy of the food record was reviewed by a nutritionist at each study visit. Nutrient intakes were analysed with a Micro Nutrica program developed at the Research and Development Centre of the Social Insurance Institution, Turku, Finland. The program calculates 66 nutrients of commonly used foods and dishes in Finland. The data bank of the program has been continuously updated.

Using the dietary data, a diet score was produced to describe the healthiness of the diet according to recent dietary guidelines. The diet score consisted of foods that were classified as 'healthy' (7 food groups) and 'unhealthy' (4 food groups). Wholegrain products, fruit and berries, vegetables, fish, nuts, vegetable fats and low-fat, unsweetened dairy products were classified as healthy, while red meat, sweetened beverages, snacks and sweet desserts were classified as unhealthy. Intake of food in the respective food groups was then divided into quartiles, varying from low to high intake. The intake quartiles were scored from 0–3 points, with higher points indicating a healthier diet. The healthy food groups were scored as follow: the highest quartile describing the highest intake as grams got 3 points, 3rd quartile 2 points, 2nd quartile 1 point and the lowest quartile 0 points. Unhealthy food groups were scored inversely: the highest quartile got 0 points, 3rd quartile 1 point, 2nd quartile 2 points and the lowest quartile 3 points. Finally, the diet score was calculated as the sum of points from food groups with a maximum score being 33.

Baseline characteristics and clinical measures

Weight, height and blood pressure of the parents were measured beginning from the child's age of 7 months until 20 years as shown in Table 1. Weight was measured using an electronic scale (Soehnle S10, Soehnle, Murrhardt, Germany) and height using Harpenden Stadiometer (Holtain, Crymych, Great Britain). Body mass index was calculated as weight (kg) divided by the square of height (m). Blood pressure was measured after >5 min rest twice with an oscillometric device (Criticon Dinamap 1846 SX until 2001, thereafter Criticon Dinamap Compact T) using appropriate cuff width. The mean of the two measurements was used in the statistical analyses.

Laboratory analyses

The serum lipids, glucose and insulin of the parents were measured repeatedly since the child's age of 7

Table 1. Parental clinical measures and blood samples collected beginning from the child's age of 7 months until the age of 20 years.

	Months		Years																		
	7[a]	13[a]	2[a]	3[a]	4[a]	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Body mass index, kg/m^2	X	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x
Blood pressure, mmHg	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x
Total cholesterol, mmol/l	x	x	x	x		x		x		x		x		x		x		x		x	
HDL-C, mmol/l	x	x	x	x		x		x		x		x		x		x		x		x	
LDL-C, mmol/l						x		x		x		x		x		x		x		x	
Triglycerides, mmol/l						x		x		x		x		x		x		x		x	
Apo A-1, g/l	x													x		x					
Apo B, g/l	x							x		x				x		x					
Glucose, mmol/l																x		x		x	
Insulin, μU/l																x					

[a]Non-fasting samples

Apo: apolipoprotein; HDL: high-density lipoprotein; LDL: low-density lipoprotein

months (Table 1). Blood samples were non-fasting before the 5 year follow-up visit and fasting samples thereafter. All serum analyses were performed at the laboratory of the National Public Health Institute in Turku, Finland. Serum cholesterol concentration was measured with a fully enzymatic cholesterol oxidase p-aminophenazone method (Merck, Darmstadt, Germany) and serum triglyceride concentration with the colorimetricglycerol-3-phospateoxidase p-aminophenazone method (Merck) as described previously.[3] Serum high-density lipoprotein cholesterol was analysed after precipitation of apolipoprotein (Apo) B-containing lipoprotein particles, including low-density lipoprotein cholesterol by dextran sulfate 500 000.[3] The Friedewald formula was used to estimate serum low-density lipoprotein cholesterol concentration.[17] Apo A-1 and Apo B were determined immunoturbidimetrically with Apo A-1 and B kits (OrionDiagnostica, Helsinki, Finland).[3] Blood samples used for the insulin and glucose concentration analyses were centrifuged immediately. Details of the insulin and glucose analyses have been reported previously.[4]

Related to the available food record data between the child's ages of 9–19 years, the cardio-metabolic risk factor data were also used during this period. Additionally, for total and low-density lipoprotein cholesterol the entire available data beginning from the child's age of 7 months or 5 years, respectively, were used.

Statistical analysis

Differences in the baseline characteristics including age, body mass index, blood pressure, and serum lipids between the intervention and control parents were studied by Student's *t*-test. The associations of dietary intervention with repeated measures of diet (from the child's age of 9–19 years) and cardio-metabolic risk factors (from the child's age of 7 months or 9 years to 19 years) were tested using repeated linear regression analysis. In all analyses, mothers and fathers were analysed separately. Since there was a difference in maternal Apo B cholesterol at the baseline it was used as a covariate when the difference in total and low-density lipoprotein cholesterol between intervention and control mothers was analysed. All variables were corrected for skewness when necessary. The study population comprised eight twin pairs; the twins' parents were considered in the analysis once. Pregnant women's data were excluded from the analysis at the time of pregnancy ($n = 417$). Statistical analyses were performed with SAS version 9.4 (SAS Institute Inc., Cary, NC, USA). It was considered that two-tailed p-values ≤ 0.05 were statistically significant.

Results

Baseline characteristics of the study population

The mean age of the STRIP study children's mothers was 30.2 [standard deviation (SD) 4.7] years and of the fathers, 32.3 (SD 5.7) years. The mean age of mothers and fathers, respectively, in the intervention and control groups was similar (Table 2.). Body mass index, blood pressure and serum cholesterol values were also similar between the intervention and control mothers and fathers, respectively, at baseline except for maternal Apo B, which was 0.98 (SD 0.16) in the intervention group compared with 1.00 (SD 0.17) in the control group ($p = 0.03$). Thus, the maternal baseline Apo B was considered as a covariate.

Effect of intervention on parental dietary intake

The child-oriented dietary intervention associated with the parental dietary intake between the child's ages of 9–19 years especially regarding the quality of dietary fat. Saturated fat intake was lower (for mothers 12.0 versus 13.8 E%, $p < 0.0001$; for fathers 12.3 versus 13.9 E%, $p < 0.0001$), polyunsaturated fat intake higher (for mothers 6.1 versus 5.4 E%, $p < 0.0001$; for fathers 6.4 versus 5.8 E%, $p = 0.0001$) and monounsaturated fat intake higher (for mothers 12.2. versus 11.6, $p = 0.01$; for fathers 12.8 versus 12.2, $p = 0.04$) in the intervention parents compared with controls (Table 3.). Figure 1 highlights that the difference in the intake of saturated fatty acids and polyunsaturated fatty acids persisted during the follow up both in mothers and fathers. Additionally, in the intervention fathers, and protein intake was higher compared with the control fathers (18.1 versus 17.5 E%, $p = 0.02$). Sucrose intake was similar in the intervention and control group both in the mothers and fathers. When the whole diet was taken into account by using the diet score, the intervention was associated with healthier diet, indicated by a higher diet score, both in the intervention mothers and in fathers (diet score; intervention versus control: 17.4 versus 16.2, $p < 0.0001$ in mothers and 17.2 versus 15.9, $p < 0.0001$ in fathers).

Effects of intervention on parental cardio-metabolic risk factors

The child-oriented dietary intervention associated with maternal serum cholesterol concentrations but not with paternal lipids (Table 4.). Maternal total and low-density lipoprotein cholesterol concentration was lower in the intervention group compared with the control group (5.01 versus 5.14 mmol/l, $p = 0.03$ and 3.18 versus 3.30 mmol/l, $p = 0.02$, respectively) between the child's age of 9–19 years. When the baseline Apo B was

Table 2. Mean values (standard deviation) or percent of the mothers and fathers at the baseline in the STRIP study. The Student's *t*-test or Chi-square test for education was used to study for the difference of the study groups.

	Mother's			Father's		
	intervention	Control	*p*-value	intervention	Control	*p*-value
Age, years	30.1 (4.6)	30.2 (4.9)	0.80	32.5 (5.9)	31.9 (5.4)	0.18
n	560	538		541	522	
Body mass index, kg/m^2	23.1 (3.6)	23.1 (3.7)	0.70	24.9 (3.2)	24.7 (3.2)	0.33
n	545	514		521	496	
Blood pressure						
Systolic, mmHg	119.2 (15.5)	120.0 (16.4)	0.46	135.0 (17.6)	133.6 (17.0)	0.18
Diastolic, mmHg	71.2 (9.5)	71.4 (9.6)	0.69	74.7 (10.4)	74.0 (10.5)	0.30
n	542	510		491	475	
Total cholesterol, mmol/l	5.00 (0.87)	5.10 (0.97)	0.11	5.35 (1.03)	5.34 (1.04)	0.96
HDL-C[1], mmol/l	1.34 (0.26)	1.32 (0.28)	0.33	1.12 (0.26)	1.13 (0.26)	0.68
n	547	516		520	497	
Apo A-1, g/l	1.65 (0.19)	1.64 (0.20)	0.23	1.54 (0.17)	1.54 (0.18)	0.77
Apo B, g/l	**0.98 (0.16)**	**1.00 (0.17)**	**0.03**	1.13 (0.21)	1.12 (0.20)	0.73
n	513	485		490	469	

Apo: apolipoprotein; HDL: high-density lipoprotein; the significant *p* values as bold ($p < 0.05$).

Table 3. Associations of dietary intervention with dietary intake in the intervention and control group mothers and fathers during the follow up between the child's ages of 9 to 19 years. Data are presented as least squares means (standard error).

	Mother's ($n = 608$)			Father's ($n = 487$)		
	intervention	Control	*p*-value	intervention	Control	*p*-value
n	286	322		225	262	
Energy (kcal)	1696 (22.0)	1695 (20.2)	0.94	2119 (34.9)	2104 (31.2)	0.74
Protein (E%)	17.0 (0.16)	17.1 (0.15)	0.74	**18.2 (0.22)**	**17.6 (0.19)**	**0.02**
Fat (E%)	33.0 (0.34)	33.8 (0.31)	0.08	34.2 (0.41)	34.9 (0.36)	0.19
Saturated fat	**12.0 (0.18)**	**13.8 (0.16)**	**<0.0001**	**12.3 (0.20)**	**13.9 (0.18)**	**<0.0001**
Monounsaturated fat	**12.2 (0.15)**	**11.6 (0.14)**	**0.01**	**12.8 (0.19)**	**12.2 (0.16)**	**0.04**
Polyunsaturated fat	**6.1 (0.08)**	**5.4 (0.07)**	**<0.0001**	**6.4 (0.10)**	**5.8 (0.09)**	**0.0001**
P/S ratio	**0.56 (0.01)**	**0.44 (0.01)**	**<0.0001**	**0.56 (0.01)**	**0.47 (0.01)**	**<0.0001**
Carbohydrates (E%)	48.4 (0.36)	47.8 (0.33)	0.23	44.8 (0.48)	44.5 (0.43)	0.69
Sucrose (E%)	7.1 (0.18)	7.3 (0.16)	0.86	6.1 (0.22)	6.5 (0.20)	0.23
Fiber (g)	18.9 (0.35)	18.4 (0.32)	0.25	20.8 (0.49)	20.1 (0.44)	0.35
Diet score (range 0–33)	**17.4 (0.18)**	**16.4 (0.17)**	**<0.0001**	**17.1 (0.21)**	**15.7 (0.19)**	**<0.0001**

E%: percent of daily energy; P/S: polyunsaturated/saturated fatty acids ratio; the significant *p* values as bold ($p < 0.05$)

taken into account the difference in maternal total and low-density lipoprotein cholesterol was not statistically significant (mean $\pm$ SE 5.04 ± 0.04 versus 5.12 ± 0.04 mmol/l, $p = 0.09$ and 3.21 ± 0.03 versus 3.29 ± 0.03, $p = 0.06$; respectively). There was no statistical difference in the paternal cholesterol concentrations between the intervention and control groups during the follow up (5.50 versus 5.58 mmol/l, $p = 0.33$ for total cholesterol and 3.57 versus 3.66 mmol/l, $p = 0.17$ for low-density lipoprotein cholesterol). There were no differences in the other cardiometabolic risk factors, such as high-density lipoprotein cholesterol, Apo A-1 and B, triglyceride, glucose and insulin, systolic and diastolic blood pressure and body mass index levels of parents of the intervention and control children.

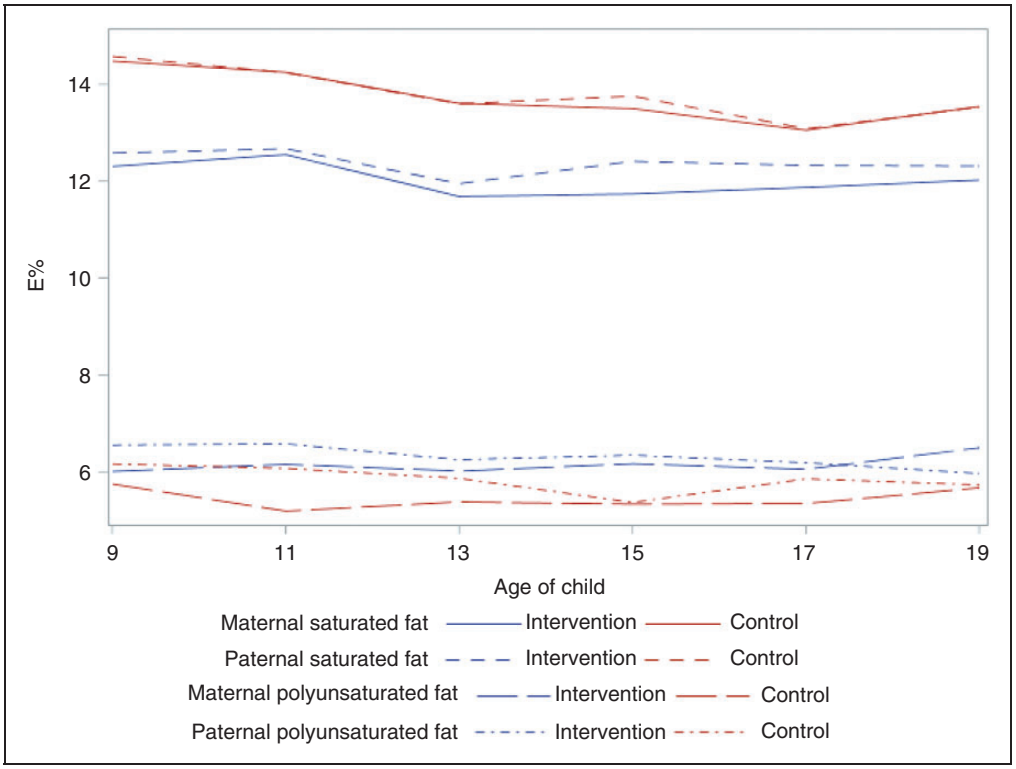

Figure 1. Mean saturated and polyunsaturated fat intake (percent of daily energy, E%) in mothers and fathers in the intervention and control groups from the child's age of 9 until 19 years. Parents in the intervention group (blue lines) have lower saturated and higher polyunsaturated fat intake compared with control group (red line).
E%: percent of daily energy.

Additional analyses for total and low-density lipoprotein cholesterol from the child's age of 7 months or 5 years (non-fasted samples) until the child's age of 19 years showed that the results were essentially similar to the data obtained between ages 9–19 years (Figure 2). The intervention mothers had lower total cholesterol (4.89 mmol/l) compared with controls (5.02 mmol/l, $p = 0.003$) and lower low-density lipoprotein cholesterol concentration (3.11 mmol/l for intervention versus 3.24 mmol/l for control group, $p = 0.003$) during the follow up while no statistical differences were observed in the fathers (5.35 versus 5.39 mmol/l, $p = 0.52$ for total cholesterol and 3.55 versus 3.62 mmol/l, $p = 0.23$ for low-density lipoprotein cholesterol).

Discussion

This study showed that child-oriented dietary intervention associated with a healthier diet, including decreased intake of saturated fat and the increased intake of polyunsaturated fat in the intervention children's parents compared with control children's parents. The dietary intervention also tended to associate with lower serum total cholesterol and low-density lipoprotein cholesterol concentrations in mothers. In fathers, the difference in low-density lipoprotein cholesterol between study groups was marginally seen but it was not statistically significant. These findings importantly add to, and are in line with, our previous findings showing that the child-oriented dietary intervention associated with the quality of dietary fat in both parents but associated with an improved serum lipid profile only in mothers during the first 5 years of the STRIP trial.[9]

The finding that the dietary intervention associated with dietary intake both in the mothers and fathers is in line with previously reported data from a short-term intervention targeted for children resulting in lowered intake of high-energy and high-fat foods in intervention children's mothers.[13] In the study by Lioret et al, the number of fathers was, however, marginal prohibiting conclusions on the intervention effects among them.[13] Evidence for the effect of dietary intervention targeted for children on paternal dietary intake is in all nearly non-existent. Reasons for the intervention effect on the parents' diet likely relate to the intervention, although child-oriented, being first given to the parents, the shared family foods/dishes and the very long-term intervention period. Taken together, our study showed the first

Table 4. Mean body mass index, blood pressure, fasting serum lipids, glucose and insulin in intervention and control group mothers and fathers during the follow up between the child's ages of 9–19 years. Data are presented as least squares means (standard error).

	Mother's			Father's		
	intervention	Control	*p*-value	intervention	Control	*p*-value
Body mass index, kg/m^2	24.9 (0.24)	25.2 (0.23)	0.78	26.6 (0.23)	26.7 (0.22)	0.97
n	*316*	*332*		*267*	*288*	
Blood pressure						
Systolic, mmHg	122.0 (0.83)	123.0 (0.80)	0.47	132.6 (0.84)	131.7 (0.80)	0.44
Diastolic, mmHg	72.8 (0.51)	73.1 (0.49)	0.66	78.0 (0.50)	77.3 (0.48)	0.36
n	*317*	*333*		*267*	*287*	
Total cholesterol, mmol/l	**5.01 (0.04)**	**5.14 (0.04)**	**0.03**	5.50 (0.06)	5.58 (0.05)	0.33
HDL-C, mmol/l	1.36 (0.02)	1.38 (0.02)	0.37	1.18 (0.02)	1.20 (0.02)	0.78
n	*308*	*329*		*252*	*277*	
Apo A-1, g/l	1.53 (0.01)	1.55 (0.01)	0.45	1.46 (0.01)	1.47 (0.01)	0.71
n	*249*	*286*		*213*	*236*	
Apo B, g/l	0.90 (0.01)	0.92 (0.01)	0.16	1.13 (0.02)	1.13 (0.02)	0.98
n	*300*	*323*		*242*	*260*	
LDL-C, mmol/l	**3.18 (0.04)**	**3.30 (0.03)**	**0.02**	3.57 (0.05)	3.66 (0.05)	0.17
n	*306*	*328*		*251*	*277*	
Triglycerides, mmol/l	1.05 (0.02)	1.01 (0.02)	0.83	1.69 (0.06)	1.31 (0.06)	0.35
n	*306*	*325*		*251*	*277*	
Glucose, mmol/l	5.08 (0.03)	5.15 (0.03)	0.09	5.46 (0.05)	5.45 (0.04)	0.63
n	*236*	*276*		*203*	*241*	
Insulin[a], μU/l	6.84 (0.28)	7.22 (0.32)	0.55	8.15 (0.44)	7.76 (0.43)	0.38
n	*217*	*264*		*187*	*228*	

[a]Measurement available at the age of child of 15 years, analysed by Student's *t*-test

Apo: apolipoprotein; HDL: high-density lipoprotein; LDL: low-density lipoprotein; the significant *p* values as bold ($p < 0.05$).

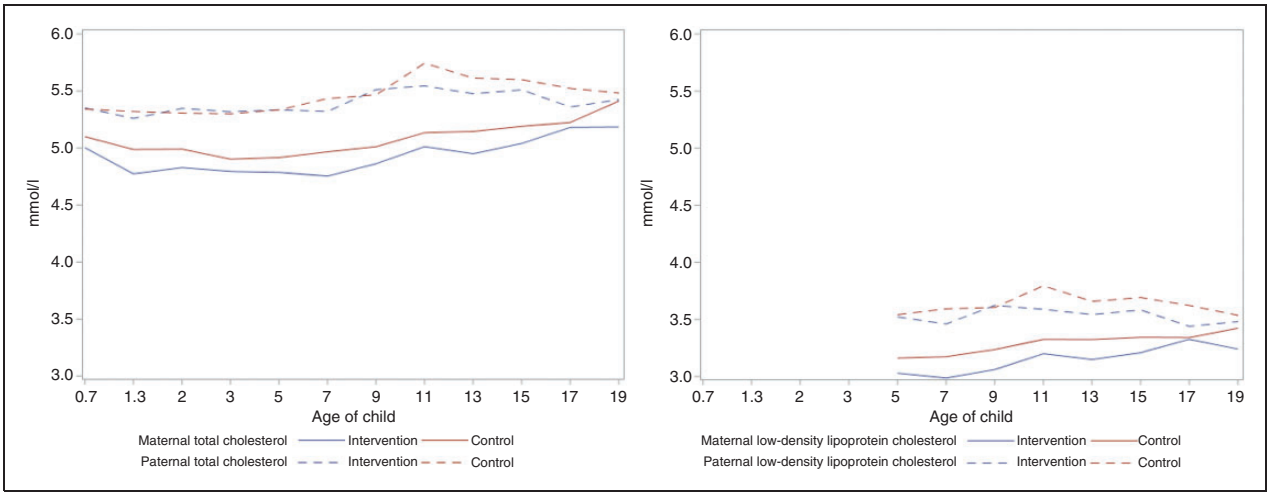

Figure 2. Trajectories of parental mean serum total and low-density lipoprotein (LDL) cholesterol (mmol/l) during the whole study period (from the child's age of 7 months or 5 years until the age of 19 years) in the mother and fathers of the intervention and control children ($n = 560$, $n = 537$, $n = 528$, $n = 516$ for total cholesterol and $n = 385$, $n = 381$, $n = 326$, $n = 344$ for LDL cholesterol, respectively). Maternal mean serum total and LDL cholesterol were lower in the intervention group (blue lines) compared with control group (red lines) (mean ± SE 4.89 ± 0.03 versus 5.02 ± 0.03, $p = 0.003$ for total and 3.11 ± 0.03 versus 3.24 ± 0.03, $p = 0.003$ for LDL cholesterol concentration). No differences were observed in the fathers (5.35 ± 0.04 versus 5.39 ± 0.04, $p = 0.52$ for total and 3.55 ± 0.04 versus 3.62 ± 0.04, $p = 0.23$ for LDL cholesterol concentration).

LDL: low-density lipoprotein; SE: standard error

time that child-oriented dietary counselling also benefits the parents.

There are several potential reasons for observing a significant lipid lowering effect in the intervention mothers and only marginally in fathers, although the effect on dietary intake was found in both parents. First, the finding may be partly mediated by the maternal baseline Apo B which was lower in the intervention mothers compared with the control mothers; whereas in fathers, all studied lipids were similar between the study groups at baseline. Second, the assessment of diet may be biased due to the following reasons. Mothers may have been more active to participate in the study compared with fathers and complied better with the diet which was guided for the children.[18] It is also possible that the mother has, at least partly, participated in filling the father's food record resulting in biased dietary intake estimates particularly towards healthier choices in the intervention group. Previously it has also been proposed that underreporting may be more common among men than women.[19,20] In our study, intervention fathers may also have misreported their dietary intake towards counselled diet aiming at socially desirable responses independently of actual behaviour changes.[21] Thus, underreporting of particular foods perceived as unhealthy may be higher in the intervention group compared with the control group[21] and partly explain our findings where dietary intervention was associated with paternal dietary intake but not with paternal serum lipids. Third, a reason for a lack of association between the intervention and paternal serum lipids may be that the differences in diet, such as the quality of dietary fat, between the study groups in fathers were not strong enough to cause a statistically significant difference in serum lipids as the effect of dietary intervention on maternal dietary intake was slightly greater than on paternal diet. The parents of STRIP intervention children received 1.6–1.8 E% less saturated fat and 0.6–0.7 E% more polyunsaturated fat than the parents of control children, in line with the difference observed between the intervention and control children.[3,16] According to the Keyes equation,[22] this difference in dietary fat quality would result in approximately 0.2 mmol/l lower total cholesterol concentration, as we found in the intervention boys.[3] In the parents of the intervention children the change in total cholesterol was thus less than predicted. In all, a marked increase in the mean body mass index was observed during the study period similar to a national FINRISK survey.[23]

A potential limitation of this longitudinal study extending 20 years is that participants discontinue participation which may cause selection in the study population. We have previously assessed the reasons for discontinuance, the most common including moving away from the community, having recurrent infections, or reluctance to have blood sampled.[15] We have no evidence indicating selective discontinuation within families, but such bias would be difficult to measure directly. Presumably, a selection bias would be caused by increased likelihood of more health conscious and motivated families to remain in the study. Thus, the differences in dietary intakes between the study groups may be weakened. A further limitation of the study is the assessment of dietary intake by 1-day food record, which represents an overview of the whole diet and we did not have data on the body composition of the parents. Particularly related to the parents, the child-oriented counselling was focused on the diet, not comprising all aspects of a heart-healthy lifestyle. The strength of this study is that fathers are extensively included in study population (44–49% of the participants) contrary to previous studies, in which study populations mainly consisted of mothers (95% of the participants).[12,14] Socioeconomic status has also been similar in intervention and control families.[16] Moreover, the major strength of the study is the unique intervention and long-term follow up with repeated measurements since infancy.

Conclusion

The present study shows for the first time that a child-oriented dietary counselling also contributes advantageously to the parental diet in long-term and tended to reflect lipid concentrations, particularly in mothers. The results emphasize that the longitudinal dietary counselling directed at children may be an efficient way to also improve the diet of parents.

Author contribution

All authors contributed to the conception or design of the work. JJ, KP and OR contributed to the acquisition analysis, or interpretation of data for the work. JJ drafted the initial manuscript. All authors critically reviewed and revised the manuscript and approved the final manuscript as submitted and agree to be accountable for all aspects of the work ensuring integrity and accuracy.

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