Effect of α-Linolenic Acid–Rich *Camelina sativa* Oil on Serum Fatty Acid Composition and Serum Lipids in Hypercholesterolemic Subjects

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***Camelina sativa*–derived oil (camelina oil) is a good source of α-linolenic acid. The proportion of α-linolenic acid in serum fatty acids is associated with the risk of cardiovascular diseases. We studied the effects of camelina oil on serum lipids and on the fatty acid composition of total lipids in comparison to rapeseed and olive oils in a parallel, double-blind setting. Sixty-eight hypercholesterolemic subjects aged 28 to 65 years were randomly assigned after a 2-week pretrial period to 1 of 3 oil groups: camelina oil, olive oil, and rapeseed oil. Subjects consumed daily 30 g (actual intake, ~33 mL) of test oils for 6 weeks. In the camelina group, the proportion of α-linolenic acid in fatty acids of serum lipids was significantly higher ($P < .001$) compared to the 2 other oil groups at the end of the study: 2.5 times higher compared to the rapeseed oil group and 4 times higher compared to the olive oil group. Respectively the proportions of 2 metabolites of α-linolenic acid (eicosapentaenoic and docosapentaenoic acids) increased and differed significantly in the camelina group from those in other groups. During the intervention, the serum low-density lipoprotein (LDL) cholesterol concentration decreased significantly by 12.2% in the camelina oil group, 5.4% in the rapeseed oil group, and 7.7% in the olive oil group. In conclusion, camelina oil significantly elevated the proportions of α-linolenic acid and its metabolites in serum of mildly or moderately hypercholesterolemic subjects. Camelina oil's serum cholesterol-lowering effect was comparable to that of rapeseed and olive oils.**

SERUM FATTY ACID composition is a good biomarker of the fatty acid composition of the diet.[1-4] Serum fatty acid analysis gives valuable information on the results of a specific diet and on the success of dietary advice for the treatment of, eg, hypercholesterolemia or non–insulin-dependent diabetes mellitus.[5] On the other hand, the proportion of some specific fatty acids of dietary origin in serum is associated with cardiovascular risk.[6,7]

Alpha-linolenic acid (18:3, n-3) can affect several cardiovascular risk factors, including serum lipids, blood pressure, and hemostatic factors, and thus reduce the risk for coronary heart disease.[8,9] According to some estimations, a 1% increment in the proportion of α-linolenic acid in adipose tissue corresponds to a 5-mm Hg lowering of systolic blood pressure.[10] Previous studies showed the effect of α-linolenic acid, an n-3 fatty acid, on serum lipoprotein metabolism to be equivalent to that of n-6 fatty acids.[11-14] Polyunsaturated fatty acids decrease serum cholesterol concentrations when exchanged for saturated fatty acids; in addition, n-6 fatty acids are considered to have an independent cholesterol-lowering property.[15]

There is also epidemiologic evidence of the cardioprotective effects of α-linolenic acid. In the European Community Multicenter Study on Antioxidants, Myocardial Infarction, and Breast Cancer (EURAMIC) study, the relative risk for myocardial infarction was highest in subjects with the lowest α-linolenic acid concentration in adipose tissue.[9] In the Lyon Diet Heart Study, the plasma concentration of α-linolenic acid tended to be inversely associated with cardiac death and nonfatal myocardial infarction in a prospective follow-up.[6] In addition, n-3 fatty acids are essential for normal growth and may play a role in the prevention and treatment of diabetes and some inflammatory and autoimmune disorders.[16]

Cold-pressed (produced by mechanical pressing of clean seed material in delicate and temperature-controlled conditions) camelina oil (*Camelina sativa*–derived oil) is a good source of α-linolenic acid compared to other edible oils. Altogether 36% to 40% of its fatty acid content consists of α-linolenic acid, an n-3 fatty acid of plant origin. However, compared to flaxseed oil (linseed oil), camelina oil not only has a lower content of α-linolenic acid but also a lower content of saturated fatty acids. Furthermore, compared to flaxseed, *Camelina sativa* seed has a higher content of tocopherols (~700 mg/kg), which gives the oil oxidative stability.[17,18]

In the present study, we examined the effect of *Camelina sativa*–derived oil on serum fatty acid composition and serum total and lipoprotein lipids. To our knowledge, this is the first controlled study on these effects of *Camelina sativa*–derived oil.

MATERIALS AND METHODS

Subjects

Inclusion criteria for the subjects were serum total cholesterol concentration between 5.0 and 8.0 mmol/L; serum total triacylglycerol less than 3.0 mmol/L; age 28 to 65 years; normal liver, kidney, and thyroid function; no lipid-lowering medication; no coronary artery disease events (myocardial infarction, coronary artery bypass graft [CABG], or percutaneous transluminal coronary angioplasty [PTCA] within the previous 6 months); diabetes; transient ischemic attack; or malignant diseases. Subjects reporting alcohol use more than 4 portions per day in the interview were also excluded, as were those with irregular eating habits or other difficulties in exchanging dietary fat to the test oil.

Subjects were recruited from the register of volunteers of Foodfiles Ltd in the Kuopio area of Eastern Finland. Ninety subjects with mild to moderate hypercholesterolemia were recruited. The purpose of the study was explained to the participants, and all provided written consent to participate. After the pretrial period, 17 of the 90 subjects

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Table 1. Pretrial Characteristics of the Study Subjects (N = 68)

	Camelina Oil	Rapeseed Oil	Olive Oil	*P* Value*
Men/women	10/13	5/17	12/11	NS†
Age (yr)	51 (11)	53 (9)	50 (9)	NS‡
Body mass index (kg/m^2)	24.8 (3.0)	26.9 (3.9)	25.3 (3.8)	NS
Serum total cholesterol (mmol/L)	6.2 (0.9)	6.2 (0.8)	6.3 (0.7)	NS
Serum LDL cholesterol (mmol/L)	4.2 (0.7)	4.1 (0.7)	4.3 (0.6)	NS
Serum HDL cholesterol (mmol/L)	1.4 (0.3)	1.6 (0.3)	1.5 (0.4)	NS
Serum total triacylglycerols (mmol/L)	1.4 (0.8)	1.1 (0.5)	1.2 (0.6)	NS
Systolic blood pressure (mm Hg)	131 (19)	133 (15)	132 (18)	NS
Diastolic blood pressure (mm Hg)	82 (8)	84 (7)	86 (7)	NS‡

NOTE. Values are means (SD). *P* values $< .05$ are considered significant.
Abbreviation: NS, not significant.
*Indicates the significance of differences among the groups analyzed with 1-way ANOVA.
†Analyzed with chi-square test.
‡Analyzed with the Kruskal-Wallis test.

recruited were not eligible for the study. Of the 73 eligible subjects, 68 (93%) completed the trial.

During the intervention, 5 subjects discontinued the study for the following reasons: 1 refused to start the use of oil due to unexpected change in health status; 2 dropped out due to sickness not related to the use of oil; and 2 discontinued the use of oil due to abdominal discomfort (diarrhea and nausea). There were 2 drop-outs in the camelina oil group, 2 in the rapeseed oil group, and 1 in the olive oil group.

Pretrial characteristics of the study subjects are listed in Table 1. A structured interview on previous and present diseases and routine hematology was performed at the beginning of the trial to ensure the eligibility of the subjects.

According to the structured interview, 40% of subjects used vitamin or mineral supplements, 21% used natural or herbal foods or organic remedies, and 6% consumed functional foods before the study. Lipid-lowering agents and therapies, including fish oil and other oil supplements, were discontinued several months before the intervention. The use of serum lipid-affecting functional foods (plant stanol/sterol esters) was also finished before the trial.

During the intervention, 11 subjects received medical treatment for hypertension, 17 used postmenopausal hormone therapy or hormonal contraceptives, and 13 received other medications. At the beginning of the study, 67% of the subjects exercised regularly, 16% smoked, and 84% used alcohol. Subjects were requested to keep their alcohol consumption, smoking habits, physical activity, and use of vitamins and other nutrient supplements constant during the study. A structured interview on alcohol and tobacco consumption and physical activity was repeated at the end of the trial. During the study period, subjects made daily records of any changes in their health status and life style. Possible adverse effects of the test oils were also recorded.

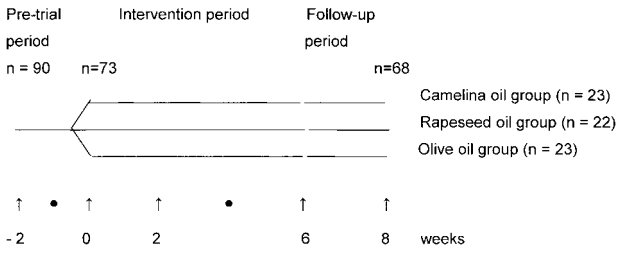

Fig 1. Study design. ↑, Blood samples and blood pressure; ●, Food record (4 days).

Methods

Study design and diets. The trial followed a randomized, double-blind, parallel design with a 2-week pretrial period followed by 6 weeks of intervention and 2 weeks of follow-up (Fig 1). The study design and written consent forms were approved by the Ethics Committee of the Kuopio University Hospital and University of Kuopio.

During the pretrial period, subjects followed their stable habitual diet. After the run-in period, all eligible subjects were randomized into 1 of the 3 groups. One group was instructed to consume 30 g of cold-pressed camelina oil per day, another group 30 g of cold-pressed rapeseed (canola) oil per day, and the third group 30 g of cold-pressed olive oil per day.

The test oils were delivered to the subjects in 500-mL bottles. Subjects were instructed by nutritionist to substitute 30 g of their current dietary fat and/or respective calorie amount of carbohydrates with 35 mL (30 g) of test oil per day. The diets were planned to have the same energy content. Study subjects portioned out the test oil with a graduated medicine cup and recorded the use of the test oils daily. The nutritionist checked the records at each visit. The composition of the test diet paralleled the composition of the study subjects' habitual diet, except for fat modification due to the use of test oils. The energy and nutrient compositions of the test oils are presented in Table 2. During the follow-up period, subjects followed their habitual diet.

The composition of the test diet was monitored by 4 day food records. During the intervention period, subjects recorded their dietary intake in food records for 4 consecutive days, including 1 weekend day or the person's day off from work. Serving sizes were estimated with a portion size picture booklet.[19,20] Oral instructions on recording food intake, accompanied by written instructions, were given to all subjects. At the study visits, food records were checked for completion by the nutritionist, who filled in the data that were lacking. Nutrients were

Table 2. Nutrient Composition of Test Oils (/100 g)

	Camelina Oil	Rapeseed Oil	Olive Oil
Energy (kJ/kcal)	3,753/897	3,800/900	3,730/891
Saturated fatty acids (g)	8	5	13
Monounsaturated fatty acids (g)	32	60	69
Polyunsaturated fatty acids (g)	56	31	9
α-linolenic acid (g)	38	10	1
Linoleic acid (g)	15	21	8
Vitamin E (mg)	7,6	30	20

calculated using Micro-Nutrica dietary analysis software (The Social Insurance Institution, Turku, Finland), which is based mainly on Finnish nutrient composition data.

Laboratory measurements. All blood samples were drawn after an overnight (12-hour) fast. Serum total and high-density lipoprotein (HDL) cholesterol and total triacylglycerol were analyzed using an enzymatic photometric method (Kone Pro Analysator, Kone Instruments, Espoo, Finland) using commercial kits (CHOL CHOD-PAP, Peridochrom triacylglycerol GPO-PAP, Boehringer Mannheim, Mannheim, Germany). During the study, the intra-assay coefficients of variation were 0.7% to 2.2% for serum total cholesterol, 0.2% to 0.8% for serum HDL cholesterol, and 1.0% to 4.0% for serum total triacylglycerol; the interassay coefficients of variation were 1.7% to 2.0% for total cholesterol, 0.7% to 1.9% for HDL cholesterol, and 1.9% to 3.3% for total triacylglycerol. Serum low-density lipoprotein (LDL) cholesterol concentration was calculated by the Friedewald formula.

Serum samples for analyses of fatty acid composition were kept deep-frozen (−70 °C) until analyzed at the end of the study. Serum fat was extracted with dichloromethane-methanol (2:1) (ie, slight modification to the method by Folch et al[21]) and fat was transesterified to fatty acid methyl esters with acidic methanol (1% H_2SO_4). The percentage composition of total fatty acid methylesters was determined by Hewlett Packard 6890 (Delaware) gas chromatograph with a 25-m long NB-351 fused silica capillary column (d = 0.32 mm; phase layer, 0.20 mm; HNU Nordion Instruments, Helsinki, Finland) and split injection system and hydrogen as carrier gas using temperature programed from 180°C to 230°C. Fatty acid methyl esters were separated from 14:0 to 22:6 with a Chemstation version A06.01 (Delaware). The fatty acids were identified with Nu-Chek-Prep (Elysian, USA) gas chromatography reference standards. Between-series variation was 0.5% to 1.4% for large gas chromatography peaks (>5%), 1% to 7% for small peaks (<5%), 4.4% for α-linolenic acid, and 15% for docosahexanoic acid.[22]

Blood pressure was measured from the right arm after 10 minutes of rest in a sitting position with a mercury sphygmomanometer (Mercuro 300, Speidel & Keller, Jungingen, Germany) at every visit. The disappearance of Korotkoff's sounds (phase V) was used to determine diastolic blood pressure. Three measurements were obtained and the mean of the last 2 was used in the analyses. Weight with light clothing was measured twice (mean was used in analyses) at every visit with a digital scale (Scale Seca 707, Vogel & Halke, Hamburg, Germany). Height to the closest 0.5 cm was measured at the sixth visit (Seca telescoping measuring rod 221, Vogel & Halke).

Statistical Methods

The data were analyzed using the SPSS 10.05 for Windows statistical program (SPSS Inc, Chicago, IL). All data are presented as means ± SD. P values < .05 were considered significant. Before further analyses, normal distribution of variables was verified with the Shapiro-Wilk test. To compare the means of body weight, body mass index, serum fatty acid composition, serum lipids, and blood pressure among the test groups, the general linear model (GLM) repeated-measures analysis was performed. One-way analysis of variance (ANOVA) was used to compare the means of serum fatty acid proportion when GLM indicated an interaction effect within the test group and time. Further, post hoc comparisons of means were done with Tukey's honestly significant difference test. Dietary variables were analyzed by 1-way ANOVA and post hoc comparisons of means were done with Tukey's honestly significant difference test. The variables (some pretrial laboratory measurements, consumption of test oil, proportion of some fatty acids in serum, and some dietary variables) that were not normally distributed even after logarithmic or other mathematical transformations were tested with the Kruskal-Wallis test.

RESULTS

The mean body weight remained stable during the study (data not shown). The exercising habits, smoking habits, and alcohol consumption of the study subjects remained stable during the study. The amount of everyday activity increased slightly.

Compliance with the use of test products was good and there were no significant differences in compliance among the test groups. The average total amount of test oil used per study subject was 33.4 ± 1.9 mL/d in the camelina group, 33.1 ± 3.4 mL/d in the rapeseed oil group, and 33.5 ± 2.9 mL/d in the olive oil group. During the study, the test oils replaced most of the visible fat (spreads and salad oil), ordinary shortening in baking, and fats from animal origin in the subjects' habitual diet. Subjects with low-fat intake also replaced carbohydrate-rich foods by the test oil to keep the energy level unchanged. During the pretrial period, 65% of the study subjects consumed a low-fat diet and during the intervention fat intake increased to a moderate level. According to food records, fat intake was significantly higher ($P < .001$, paired-sample t test) during the intervention period than during the pretrial period (change in grams, 7.8 ± 17.5). Total fat intake did not differ among the test groups.

Dietary intake of energy and nutrients during the pretrial and intervention periods are presented in Table 3. There were no differences in the diet among the test groups during the pretrial period. As predicted, the intake of monounsaturated fatty acids ($P < .001$) was significantly higher in the rapeseed oil and olive oil groups compared to the camelina group. Respectively, the intake of polyunsaturated fatty acids ($P < .001$) was significantly higher in the camelina group compared to the rapeseed oil and the olive oil groups. The differences in the dietary intake among the groups were in accordance with the fatty acid composition of the test oils. Otherwise, nutrient intake did not differ among the 3 groups.

At the beginning of the intervention, the fatty acid composition of serum total lipids did not differ among the test groups. After, there were significant differences in the fatty acid profile among the different study groups (Table 4 and Fig 2). The proportion of α-linolenic acid (C18:3, n-3) was significantly higher in the camelina oil group compared to the other 2 oil groups at the end of the study period. Respectively, the proportion of serum eicosapentaenoic (C20:5, n-3) and docosapentaenoic (C22:5, n-3) fatty acids increased in the camelina oil group, resulting in significantly higher proportion compared to the other groups at the end of the study. The proportion of serum oleic acid decreased in the camelina oil group, whereas it increased in the olive oil group, the difference being significant at the end of the intervention period. The proportion of linoleic acid increased in the rapeseed oil group without a change in the proportion of oleic acid. The proportion of long-chain saturated fatty acids (C14:0, C15:0, and C16:0) decreased in all study groups.

As to lipid changes, serum total and LDL cholesterol concentrations were significantly lower in the 3 oil groups after the intervention period than at the beginning of the intervention (total cholesterol, $P < .001$; LDL cholesterol $P = .003$). As a consequence, the mean concentrations of serum total or LDL

Table 3. Nutrient Composition of the Actual Diets Based on 4-Day Food Records

	Pretrial Period (n = 68)	Camelina Oil (n = 23)	Rapeseed Oil (n = 22)	Olive Oil (n = 23)	*P* Value*
Energy (MJ/d)	7.7 (1.8)	7.3 (1.5)	7.4 (2.0)	8.0 (1.6)	NS
Energy (kcal/d)	1,848 (432)	1,735 (357)	1,781 (461)	1,923 (392)	NS
Fat (E %)	31.0 (5.4)	35.0 (6.1)	35.5 (4.5)	36.9 (5.3)	NS
Saturated fatty acids (E %)	12.3 (2.7)	8.9 (2.8)	8.3 (2.2)‡	10.1 (2.1)	.041
Monounsaturated fatty acids (E%)	10.3 (2.4)	11.4 (2.1)	16.1 (2.4)§	18.2 (3.1)§	<.001†
Polyunsaturated fatty acids (E%)	5.0 (1.5)	12.3 (2.3)	8.5 (1.5)§	5.5 (1.3)§	<.001†
Protein (E %)	16.9 (2.6)	16.3 (2.5)	16.2 (3.1)	15.3 (2.5)	NS
Carbohydrates (E%)	47.9 (6.3)	46.3 (5.9)	44.7 (6.7)	44.0 (5.3)	NS
Alcohol (E%)	2.8 (4.0)	1.0 (2.2)	2.3 (3.2)	2.5 (3.6)‖	0.043†
Fiber (g/d)	23 (9)	22 (9)	22 (9)	23 (9)	NS
Fiber (g/MJ)	2.9 (0.8)	3.0 (0.9)	2.9 (0.8)	2.8 (0.8)	NS
Insoluble polysaccharides (g/d)	10.1 (4.7)	9.8 (4.1)	9.8 (5.0)	10.3 (4.7)	NS
Soluble polysaccharides (g/d)	5.2 (1.8)	4.9 (1.9)	4.7 (1.9)	5.1 (1.7)	NS
Cholesterol (mg/d)	228 (73)	165 (65)	182 (68)	176 (64)	NS†
Cholesterol (mg/MJ)	30 (9)	23 (7)	25 (8)	22 (6)	NS

NOTE. Values are means (SD). *P* values < .05 are considered significant.

*Indicates significance of the differences among the oil groups during the intervention period analyzed with 1-way ANOVA.

†Indicates significance of the differences among the oil groups during the intervention period analyzed with the Kruskal-Wallis test

‡$P < .05$ denotes significance of differences compared to the olive oil group analyzed with Tukey's test.

§$P < .001$ denotes significance of differences compared to the camelina oil group analyzed with the Mann-Whitney test.

‖$P < .05$ denotes significance of differences compared to the camelina oil group analyzed with the Mann-Whitney test.

cholesterol did not differ among the 3 tested oil groups at the end of the study. During the intervention, the serum total cholesterol concentration decreased from a mean (SD) of 5.9 (1.0) mmol/L to 5.6 (0.8) mmol/L in the camelina oil group, from 6.1 (0.9) mmol/L to 5.7 (0.8) mmol/L in the rapeseed oil group, and from 6.4 (0.8) mmol/L to 6.0 (0.8) mmol/L in the olive oil group. The absolute and percentage changes in total and and LDL cholesterol during the intervention are shown in Fig 3. There were equal numbers of of individuals in each group ($n = 4$ or 5) who did not reduce or increase their serum cholesterol concentration during the intervention.

There were no significant changes in serum triacylglycerol or HDL cholesterol concentrations within the test groups and no significant differences were found in the mean concentrations among the groups. Interestingly, the LDL/HDL ratio was improved significantly ($P = .024$) only in the camelina oil group.

At the beginning of the intervention, the average systolic blood pressure of all the study subjects was 130 ± 15 mm Hg and that of diastolic blood pressure 84 ± 7 mm Hg. There were no significant changes in systolic or diastolic blood pressure during the study.

DISCUSSION

The aim of the present study was to investigate the effect of cold-pressed camelina (*Camelina sativa*) oil on serum fatty acid composition and serum lipids in reference to active control oils—cold-pressed rapeseed oil and cold-pressed olive oil—in subjects with mildly to moderately elevated serum cholesterol concentrations. At baseline, the proportion of α-linolenic acid in serum in this Finnish cohort paralleled the level of α-linolenic acid reported in the recent EURAMIC study.[9] In the latter study, fatty acid composition of adipose tissue was used as a biomarker and therefore the proportion of docosahexaenoic acid was higher in this cohort in our study than that reported in the EUREMIC cohort.

The serum fatty acid composition reflected well the changes in dietary fatty acid composition induced by the 3 different oils. The proportion of α-linolenic acid in serum lipids was increased more than 3-fold during the camelina oil diet and to a lesser extent during the rapeseed oil diet. Due to elongation of α-linolenic acid, eicosapentaenoic acid (C20:5, n-3) and docosapentaenoic acid (C22:5, n-3) were also increased, but no effect on docosahexaenoic acid (C22:6, n-3) was observed. This finding, together with previous studies,[23] indicates that last step in the metabolism of α-linolenic acid from docosapentanoic acid to docosahexanoic acid via elongation, desaturation, and beta-oxidation (C22:5→C24:5→C24:6→C22:6) is not affected by dietary intake of α-linolenic acid. This route is probably so complex and less dominant in humans that it cannot be accelerated by ingestion of α-linolenic acid.

The reduced proportions of the linoleic acid metabolites, dihomo-γ-linolenic (C18:3, n-6) and arachidonic (C20:4, n-6) acid, probably reflect competition between α-linolenic acid and linoleic acid for the delta-6- and delta-5-desaturase and elongase activities. Reduced proportions of saturated fatty acids (C14:0, C15:0, and C16:0) reflect substitution of the test oils for dairy fat in the background diet, and the increase in oleic acid (C18:1, n-9) during the olive oil diet is in line with the high content of oleic acid in olive oil. All of these findings indicate good adherence to the prescribed diets.

An earlier 4-week study of flaxseed oil[8] demonstrated a significant increase in plasma lipid α-linolenic acid, eicosapentaenoic acid, and docosapentaenoic acid concentrations, but no change in the docosahexaenoic acid concentration. In the study of Kelley et al,[24] 20 g of dietary α-linolenic acid resulted in an increase of α-linolenic acid in tissue fatty acids from 0.2% to

Table 4. Fatty Acid Composition of Serum1 Lipids (% of total g) Before (0 wk) and After the Intervention (+6 wk)

	Camelina Oil (n = 23)	Rapeseed Oil (n = 22)	Olive Oil (n = 23)	*P* Value[a]
14:0				NS
0 wk	1.08 (0.43)	1.05 (0.33)	1.08 (0.29)	
+ 6 wk	0.88 (0.25)	0.82 (0.25)	0.84 (0.33)	
P value[b] (<.001)	NS	<.001	.003	
15:0				NS
0 wk	0.22 (0.05)	0.22 (0.05)	0.21 (0.05)	
+ 6 wk	0.19 (0.04)	0.19 (0.04)	0.19 (0.05)	
P value[b] (<.001)	<.001	<.001	<.001	
16:0				
0 wk	22.15 (1.97)	22.28 (2.12)	22.28 (1.63)	NS[c]
+ 6 wk	21.29 (2.01)	20.89 (1.78)[d]	21.33 (1.82)[d]	NS[c]
P value	.003[f]	.002[f]	.012[f]	
16:1(n-7)				NS
0 wk	2.82 (0.76)	2.90 (0.76)	2.68 (0.93)	
+ 6 wk	2.44 (0.88)	2.47 (0.59)	2.30 (1.01)	
P value[b] (<.001)	.003	<.001	.003	
18:0				NS
0 wk	6.82 (0.67)	6.86 (0.62)	6.86 (0.58)	
+ 6 wk	7.09 (0.73)	6.97 (0.56)	6.82 (0.74)	
P value[b] (NS)				
18:1(n-9)				
0 wk	22.05 (2.40)	22.26 (2.09)	22.89 (2.66)	NS[c]
+ 6 wk	20.83 (2.64)	22.62 (1.44)[e]	25.95 (3.22)[d]	<.001[c]
P value	.016[f]	NS[f]	<.001[f]	
18:1(n-7)				NS
0 wk	2.10 (0.29)	2.11 (0.23)	2.11 (0.28)	
+ 6 wk	1.91 (0.28)	2.15 (0.18)	2.10 (0.28)	
P value (.011)	.039	NS	NS	
18:2(n-6)				NS
0 wk	28.19 (3.38)	28.04 (3.51)	28.33 (3.66)	
+ 6 wk	29.16 (3.72)	29.57 (2.85)	27.35 (4.24)	
P value[g] (<.005)	NS	.015	NS	
18:3(n-6)				NS
0 wk	0.41 (0.15)	0.41 (0.18)	0.42 (0.19)	
+ 6 wk	0.33 (0.12)	0.35 (0.12)	0.40 (0.18)	
P value[g] (.001)	.006	NS	NS	
18:3(n-3)				
0 wk	1.00 (0.27)	1.01 (0.24)	1.03 (0.27)	NS[c]
+ 6 wk	3.49 (1.87)	1.41 (0.40)[d]	0.86 (0.22)[d]	<.001[c]
P value	<.001[f]	<.001[f]	.003[f]	
20:3(n-6)				NS
0 wk	1.53 (0.32)	1.57 (0.33)	1.49 (0.25)	
+ 6 wk	1.18 (0.19)	1.44 (0.33)	1.52 (0.28)	
P value[7] (<.001)	<.001	NS	NS	
20:4(n-6)				NS
0 wk	5.95 (1.03)	5.77 (1.29)	5.90 (1.33)	
+ 6 wk	5.23 (1.03)	5.61 (1.22)	5.66 (1.18)	
P value[b] (.001)	.009	NS	NS	
20:5(n-3)				
0 wk	1.71 (1.15)	1.52 (0.72)	1.24 (0.46)	NS[h]
+ 6 wk	2.08 (0.81)	1.50 (0.43)[i]	1.20 (0.53)[j]	<.001[h]
P value[g] (.011)	0.012	NS	NS	
22:5(n-3)				
0 wk	0.64 (0.15)	0.62 (0.12)	0.60 (0.10)	NS[h]
+ 6 wk	0.73 (0.15)	0.64 (0.10)[i]	0.56 (0.10)[j]	<0.001[h]
P value[g] (.001)	.018	NS	.039	
22:6(n-3)				NS
0 wk	3.34 (1.28)	3.39 (0.90)	2.86 (0.83)	
+ 6 wk	3.16 (0.93)	3.38 (0.67)	2.92 (0.84)	
P value[b] (NS)				

NOTE. Values are mean (SD). *P* values < .05 are considered as significant.

[a]Indicates the significance of difference among the groups analyzed with the GLM for repeated measures.

[b]Indicates the significance of difference within the groups analyzed with the GLM for repeated measures.

[c]Indicates the significance of difference among the groups analyzed with Kruskall-Wallis test.

[d,e]Denotes significance of differences compared to the camelina oil group analyzed with the Mann-Whitney test, [d]$P < .001$, [e]$P < .05$.

[f]Indicates the significance of differences within the test periods analyzed with the Wilcoxon signed-rank test.

[g]Indicates the significance of the interaction effect with-in the test groups and time analyzed with the GLM for repeated measures.

[h]Indicates the significance of the differences among the test groups analyzed with 1-way ANOVA.

[i,j]Denotes significance of differences, compared to the camelina oil group analyzed with the Tukey's HSD test, [i]$P < .05$, [j]$P < .001$.

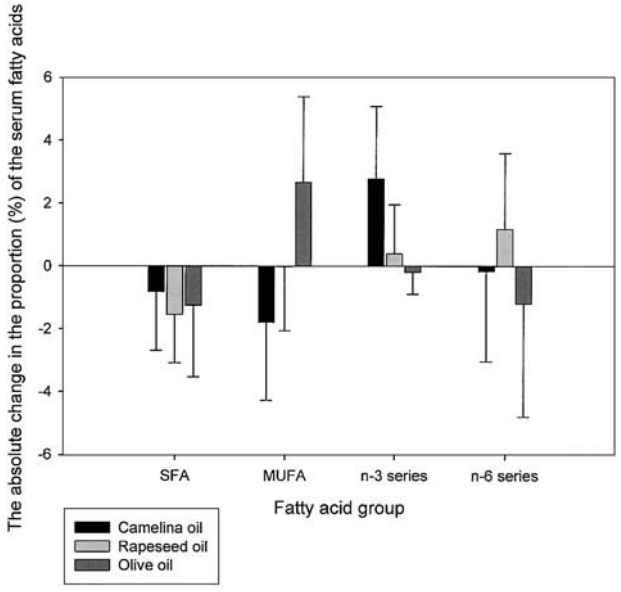

Fig 2. The absolute changes in the proportion (%) of saturated (SFA), monounsaturated (MUFA), and polyunsaturated n-3 series and n-6 series fatty acids in serum lipids.

3.2% in 56 days. The lack of effect on any longer chain metabolites of the n-3 series might alternatively be due to poor detection of long-chain fatty acids in the analysis. In the study of Abbey et al,[25] the proportions of α-linolenic acid increased from 0.6% to 2.2% and of eicosapentaenoic acid from 0.7% to 1.5% in plasma fatty acids when the diet was enriched with 9 g of α-linolenic from flaxseed oil for 6 weeks.

In all 3 intervention groups there was a significant decrease in serum total and LDL cholesterol concentrations during the oil consumption period. The investigated oils did not statistically differ in their cholesterol-lowering ability, although the decrease in the camelina group was 3 to 6 percentage units greater than in the other oil groups. Like in all dietary interventions, in all groups there was a small number of subjects who did not reduce or even increased their serum cholesterol concentration, indicating that effect of these dietary fat modifications was not completely consistent. In this study, none of the test oils significantly affected serum HDL cholesterol or triacylglycerol concentrations. The cholesterol-lowering abilities of the tested oils are in agreement with previous studies, where subjects were asked to reduce their fat intake and add vegetable oils to their normal diet. In the studies of Seppänen-Laakso et al[26] and Sarkkinen et al[27] in free-living subjects, total cholesterol declined by 4% to 9% after consumption of rapeseed oil.

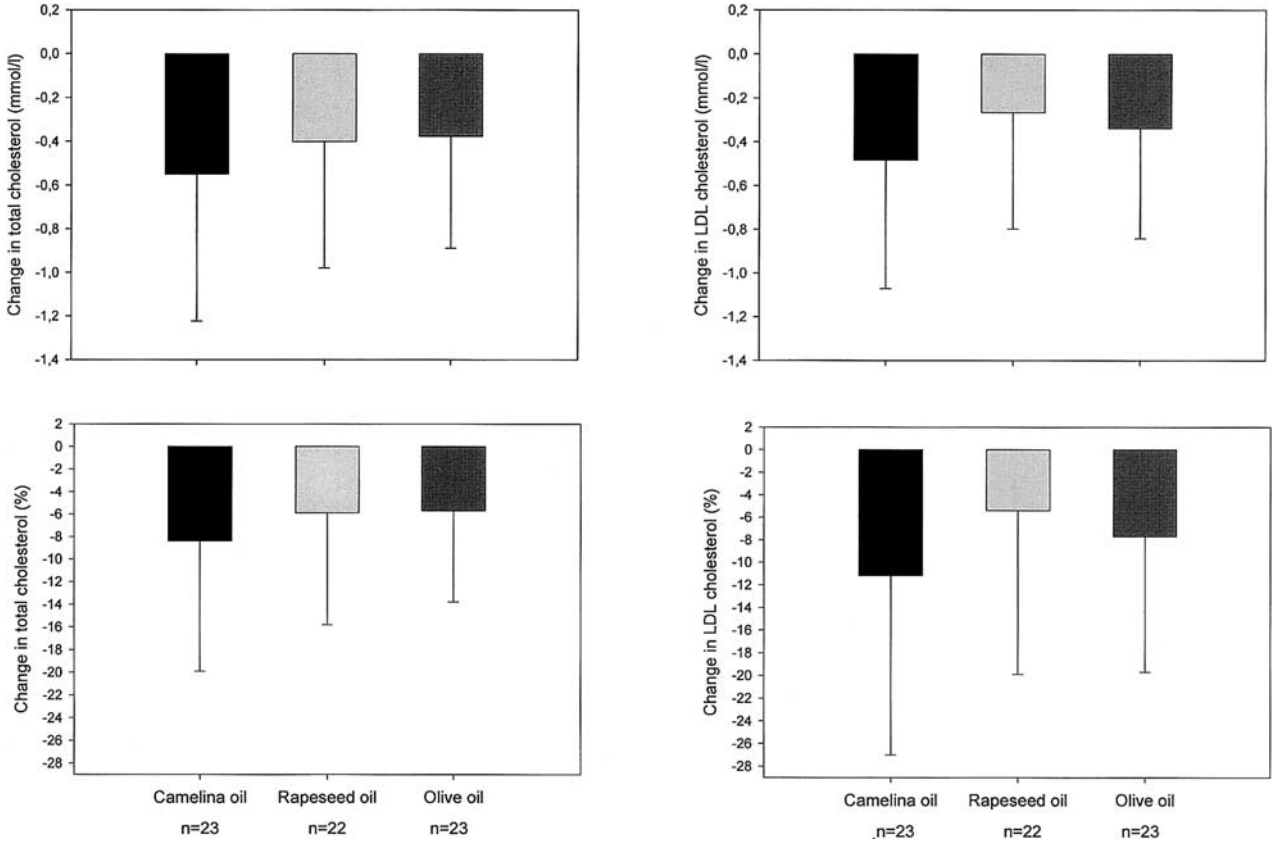

Fig 3. Mean (SD) of absolute and percentage changes in serum total and LDL cholesterol concentrations during the intervention. There was a significant reduction in total and LDL cholesterol concentrations in all test groups when analyzed by GLM for repeated measures. There were no significant differences among the oil groups when analyzed by Kruskal-Wallis test.

In the present study, systolic and diastolic blood pressure remained unchanged during the trial. Our results are similar to those of Mutanen et al,[28] who found that in normotensive subjects blood pressure was not altered by modifying fatty acid composition of the diet with rapeseed or sunflower oil. Fat modification is known to have a more marked effect on blood pressure in hypertensive than in normotensive subjects.[29] In the present study, subjects were mainly normotensive. Our findings are in a slight disagreement with those of Berry et al,[10] who found that a 1% increment in the proportion of α-linolenic acid in adipose fat tissue corresponded to a 5-mm Hg lowering of systolic blood pressure. Furthermore, Singer et al[30] found that large amounts of flaxseed oil reduced systolic blood pressure in a psychophysiological stress test from 175 $\pm$ 21 mm Hg to 161 $\pm$ 23 mm Hg. However, the daily amount of oil in their study was 60 g, which is double the amount used in the present study.

We used 30 g of oil because this amount can be consumed in everyday life. Although the amount of oil was greater than the study subjects were accustomed to consuming, compliance and palatability were good.

During the intervention, the proportion of fat as a source of energy increased slightly over the average habitual diet. This is because some of the study subjects, especially women with low energy expenditure, did not succeed in decreasing their fat intake by 30 g from their habitual diet. In these women, the fat of the test oils replaced carbohydrates to keep their energy intake constant and body weight unchanged. More important is that there were no significant differences in the adherence of the diets across the 3 study groups during the pretrial or intervention periods. The olive oil group had a higher intake of saturated fatty acids than the other oil groups. This was due to the higher content of saturated fatty acids in olive oil.

The National Cholesterol Education Program[31] recommends up to 10% of total calories be gained from polyunsaturated fat. In the present study, the energy intake from polyunsaturated fat was slightly over the recommendation in the camelina oil group. Nestel et al,[8] in a study with flaxseed oil, found that arterial compliance in obese subjects was improved with α-linolenic acid enrichment of the diet, despite the increased LDL oxidizability. Whether enriching the diet Camelina oil increases oxidative stress remains to be studied.

Recent epidemiologic studies have associated higher serum and adipose tissue α-linolenic acid concentrations with a lower risk for cardiac death and myocardial infarction.[6,9] Hu et al[32] found a lower risk of fatal ischemic heart disease in women with higher intake of α-linolenic acid, and Simon et al[33] determined that a 13% higher proportion of α-linolenic acid in cholesterol esters was independently associated with a lower risk of stroke in middle-aged men. In our study, there was a 3.5-fold increase in the proportion of α-linolenic acid in serum total lipids after six weeks consumption of camelina oil. Assuming that α-linolenic acid has direct effect on cardiovascular risk, the observed increase in the proportion of α-linolenic acid in serum lipids could play a role in the prevention of cardiovascular disease.[34]

In conclusion, we have shown that cold-pressed camelina oil can be used in household consumption to elevate the proportions of α-linolenic acid, eicosapentaenoic acid, and docosapentaenoic acid in serum of mildly to moderately hypercholesterolemic subjects. Camelina oil's serum cholesterol-lowering effect was comparable to that of rapeseed and olive oils.

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