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ORIGINAL CONTRIBUTION

K.-H. Wagner
R. Tomasch
I. Elmadfa

Impact of diets containing corn oil or olive/sunflower oil mixture on the human plasma and lipoprotein lipid metabolism

Summary *Background* The effects of monounsaturated fatty acids (MUFA) rich diets compared to those that are rich in polyunsaturated fatty acids (PUFA) as well as the effects of an intake of single oils compared to oil mixtures are controversially discussed and results are contradictory. *Aim of this study* To evaluate the effects of a plant oil-mixture (olive/sunflower oil; saturated/monounsaturated/polyunsaturated (S/M/P) = 14:69:17) high in oleic acid but also showing a moderate content of polyunsaturated fatty acids (PUFA) in comparison with a single, PUFA rich corn oil (S/M/P = 13:33:54) used in a normal, balanced diet on human plasma and lipoprotein metabolism. *Methods* The double-blind designed study comprised 28 healthy, non-smoking young men aged between 19 and 31 years. After two weeks of adjustment (mixed, balanced diet: 11.6 MJ average, average fat intake ~105 g/d), the design included a two week test period in which a diet with 80 g corn oil/d vs a mixture of 68 g olive- and 12 g sunflower oil/d (total 80 g) as the main fat source was given, followed by a crossover after two weeks. Compliance and ingestion of diets were monitored by assessing the fatty acid pattern in LDL and by determination of α- and γ-tocopherol in plasma and LDL. *Results* Diets were well incorporated due to the significant changes in plasma- and LDL-tocopherol levels and the significant different average ratio of oleic acid to linoleic acid in LDL. The PUFA-rich corn oil diet was able to reduce low density lipoprotein (LDL) cholesterol from adjustment to T2 significantly ($p < 0.01$), which was also confirmed by a trend after cross over ($p=0.15$). Total cholesterol (only after cross over at T3), total triglycerides (TG) and very low density lipoprotein (VLDL)-TG were significantly lower at T2 after the corn oil diet than after the mixed oil diet. Total high density lipoproteins (HDL) and HDL cholesterol remained unchanged by both diets. *Conclusions* The results show that during the intervention of two weeks for each diet and the following cross over the corn oil diet had more influence on lipoprotein metabolism than the MUFA-rich diet. The hypocholesteremic effect of the PUFA-rich diet must also be connected with the high amount of unsaponifiable substances, mainly phytosterols in the corn oil.

Key words Corn oil – Sunflower oil – Olive oil – Cholesterol – Triglyceride – Lipoproteins

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K.-H. Wagner (✉) · R. Tomasch · I. Elmadfa
Institute of Nutritional Sciences
University of Vienna
Althanstrasse 14
1090 Vienna, Austria
Tel.: +43-1/42 77-5 49 30
Fax: +43-1/42 77-95 49
E-Mail: karl-heinz.wagner@univie.ac.at

Introduction

During the last decade, clinical and epidemiological studies were the basis for postulating a relationship between the intake of plant oils and circulating plasma lipid levels. Commercially available edible oils are different in their fatty acid composition and unsaponifiable contents and reveal different impact on the lipid metabolism. Especially the highly monounsaturated "Mediterranean Diet", based on olive oil as the main dietary fat, is known to be associated with low plasma LDL cholesterol (LDL-C) and low triglyceride levels [1, 2]. Furthermore advantages of olive oil concerning cardiovascular diseases [3] and breast cancer [4] have been reported. In contrast to this, however, other data concern-

ing controlled human experiments indicate that, if olive oil is the major part of the dietary fat, total and LDL-C are somewhat higher than if the same amount of fat is canola oil or high oleic sunflower oil, which are both predominantly monounsaturated, but contain higher amounts of polyunsaturates than olive oil [5]. In several human studies monounsaturated fatty acids (MUFA) showed fewer plasma-cholesterol lowering effects than polyunsaturated fatty acids (PUFA) did [6, 7]. High density lipoprotein cholesterol (HDL-C) levels are found to be increased [8] or not affected [9] by diets rich in MUFA or PUFA. Edible plant oils with modified fatty acid composition such as the low erucic rapeseed oil that is rich in MUFA, highly oleic sunflower oil or oil mixtures, thus do not have the same effects on lipoprotein metabolism as conventional counterparts or olive oil itself [10, 11].

Considering the fact that oil mixtures combine positive effects of single oils, in this study the impact of a MUFA-rich oil mixture of olive oil/sunflower oil (85:15) diet on plasma and lipoprotein lipid concentrations in healthy young men was compared with the impact of a PUFA-rich corn oil diet. Additionally the change of the fatty acid composition in the LDC- and HDL-fractions and the bioavailability of oil borne tocopherols was assessed.

Material and methods

Study subjects

Twenty-eight male subjects, aged between 19 and 31, mean age 23.7 years, living in the area of Vienna, Austria, participated in this randomized study. They were informed about the purpose, nature and potential risks of the study; they gave consent in writing. The study protocol was approved by the Ethic Committee of the Medical Faculty, University of Vienna. All study participants were in good health as determined by a medical history questionnaire and results of clinical laboratory tests, normolipemic, non-smokers, free from acute or chronic illness, within normal range of body mass index (20.3 ± 2.3), and not taking any medication, mineral or vitamin supplements 4 weeks before the start and during the study. The baseline characteristics of the volunteers are shown in Table 1.

Table 1 Baseline characteristics of the volunteers

Parameters	28 Healthy men (n=28)	
	Mean ± SD	Range
Age (years)	23.7 ± 6	19–31
Body mass index (kg/m^2)	20.3 ± 2.3	18–22.3
Plasma cholesterol (mmol/l)	4.3 ± 0.8	3.7–5.3
LDL cholesterol (mmol/l)	3.2 ± 0.4	2.1–3.6
HDL cholesterol (mmol/l)	0.86 ± 0.2	0.5–1.4
Plasma triglycerides (mmol/l)	0.95 ± 0.27	0.72–1.23

LDL low density lipoproteins; *HDL* high density lipoproteins

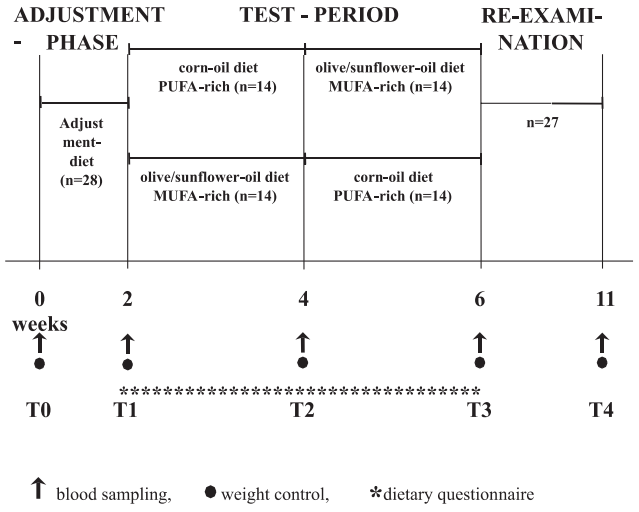

Fig. 1 Study design

Investigation trail and diets

The experiment lasted 42 days with a 35 day follow-up period. The first two weeks of baseline diet (T1) for adjustment on exactly the same diets with the same oil source (olive/sunflower/butter mixture, 11.6 MJ average, average fat intake ~105 g/d, 12 mg α-TE/d) was followed by two diet periods of two weeks each (Fig. 1). For the intervention trial (T2), the volunteers were randomized evenly into two groups, 14 men in each group (CO and MO). Both groups received similar isoenergenic diets, except for their fat intake – the CO group supplied their fat needs with 80 g corn oil/d (saturated/monounsaturated/polyunsaturated (S/M/P) diet = 28/33/39, 20 mg α-tocopherol/d and 100 mg γ-tocopherol/d), the MO group with a mixture of 68 g olive- and 12 g sunflower-oil/d (S/M/P diet = 28/49/23, 24 mg α-tocopherol/d and 2.4 mg γ-tocopherol/d) as the main fat source (fat specifications, see Table 2). At week five, a crossover was carried out and the intervention finished after week six (T3). Five weeks after T3 the last blood samples were taken (T4), to assess the development of plasma parameters after intervention. In this last period (T3-T4), the volunteers were allowed to have their usual diet; no intervention and no control of food intake was done.

Olive oil as the main fat for the MO group contained 73 % MUFA, whereas the CO had a PUFA content of 54 %.

The daily food intake during the respective study periods was prepared in accordance with the dietary guidelines of the DACH reference values, consisting of 50–55 % carbohydrates, 15 % protein and 30–35 % fat of

Table 2 Fatty acid pattern (% of total fatty acids) and content of tocopherols (mg/100 g) in plant oils and butter used for the study

	Fatty acids (% of total fatty acids)							Tocopherols (T) (mg/100 g)	
	C16:0	C16:1	C18:0	C18:1	C18:2	C18:3	C20:0	α-T	γ-T
Corn oil	10.0	0.5	2.4	31.1	50.0	0.9	0.5	24.6	126.2
Sunflower oil	6.2	0.5	4.8	21.9	60.2	0.5	0.5	85.3	8.8
Olive oil	10.8	1.5	2.4	71.7	8.0	0.9	0.5	20	1.7
Butter	21.0	1.8	9.7	20.1	1.8	1.2	0.1	2.1	n. d.*

* not detectable

total calories. All food for consumption at breakfast, lunch and dinner, about 90 % of total food, was prepared at the Institute of Nutritional Science, University of Vienna. Lunch was consumed every day at the institute in the presence of one of the investigators during all intervention periods; dinner and breakfast were taken home by the participants. The oil was incorporated into the meals differently: a defined amount for each subject was given with soups, pastries, rice or different kinds of pastas. A total amount of oil for all volunteers was used to prepare the main meals like lasagna or vegetables, meat and sweets in ovenproof dishes; however, each subject received the same portion size. The exact nutrient intake was checked by weighed dietary records during the entire study. Meals for lunch were changed daily over a 3-week period; weekly rotating menus were used for breakfast and lunch. In addition to the food supplied, the subjects were allowed to freely choose about 10 % of their energy with tocopherol-free foodstuffs. The additionally consumed food was assessed by weighed dietary records during the entire study. Total food consumption was converted into total energy and nutrient intakes by using the calculation program EWP 3.2 ("Ernährungswissenschaftliches Programm", Dato-Denkwerkzeuge, Vienna, Austria) based on the national German/Austrian food composition database BLS 2.1 [12]. The total daily intake of fat during the test-period was equal for each subject (110 g/d). The test oils used were the main fat source (80 g of 110 g/d) of the applied diets.

Plasma samples

Plasma and LDL concentrations of α-tocopherol, γ-tocopherol plasma total cholesterol (TC), LDL-C, VLDL-C and HDL-C as well as plasma and lipoprotein TG were observed in all volunteers every two weeks at T0–T3 and finally at T4.

Excretion of tocopherols was considered by determination of α- and γ-tocopherol in collected 24 h-stools using HPLC at T1–T4.

Blood samples were obtained from the overnight fasted volunteers by venipuncture into heparin-containing vacuum tubes (Vacuette; Greiner, Vienna, Austria). Platelet-poor plasma for tocopherols, TC and TG determination was separated by centrifugation at 3000 rpm for 20 min and frozen in aliquots at –80 °C until used. Plasma and lipoprotein concentrations of α- and γ-tocopherol were determined by reversed phase HPLC according to the method of Jakob and Elmadfa [13]. TC and HDL-C (after precipitation of ß-lipoproteins with dextran sulfate and magnesium chloride) in plasma were measured enzymatically (Cholesterol C-system CHOD-PAP using a test kit from Boehringer-Mannheim, Mannheim, Germany). The triglyceride concentration was determined by using the enzymatic method of Wahlefeld [14].

Isolation of lipoproteins

Lipoproteins were separated according to procedures described by Foreman et al. and Nelson [15, 16]. The density gradients were examined by NaCl solutions (d=1.0063 to separate VLDL and d=1.1816 to separate LDL). The sample was spun with a Sorvall UZ ULTRA 80 ultracentrifuge with a fixed angle T865 rotor for VLDL isolation and a TV865 vertical rotor for LDL separation. The lipoprotein fractions were removed after centrifugation by the standardized tube-slicing technique.

Analysis of fatty acids

The fatty acid pattern of the freshly prepared HDL and LDL fractions was determined by gas chromatography within the same day, while lipoprotein lipids (TC, TG) were analyzed, after storage at 4 °C, within the next day by using the boron triflouride method as previously described [17]. Briefly, fatty acid methyl esters (FAME) were obtained using boron trifluoride (BF3), by transesterification with methanolic sodium hydroxide at 100 °C for 5 min. The BF_3-methanol reagent was added and boiled. FAME were extracted into hexane twice and vaporized to dryness and re-dissolved in hexane for gas chromatography analysis. Peaks were identified with comparison to standards. Total amount of each fatty acid was given in FAME as percentage of total FAME in the sample. CV (coefficient of variation) was 5.3.

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Statistical analysis

Statistical differences between the two groups were determined by two tailed t-Student's test for dependent samples for normally distributed groups. For groups that were not normally distributed the Mann-Whitney U test was used. The area under the curve (AUC) of TAC in plasma and LDL was used to perform statistical calculations between groups. Pearson correlation coefficients and multiple regression analysis were conducted using SPSS 9.0 for Windows. Values were expressed as mean ± SE; differences were considered to be significant at a value of $p < 0.05$.

Results

All volunteers completed the study successfully and reported no side effects due to ingestion of the diet with corn oil or olive/sunflower oil diet within the study period. All subjects maintained their weight throughout the study, changes were less than 0.6 kg. The mean daily energy intake and the composition of the different diets, as determined by food records, are shown in Table 3. The ratio polyunsaturated to saturated fat (P/S ratio) of the CO diet was 1.39, of the MO diet 0.82, during the adjustment period 0.53. The total cholesterol content of diets was constant within the study period (191–335 mg/d). Dietary compliance was monitored by fatty acid analysis of the lipoprotein fractions LDL and HDL. Each fatty acid is calculated as % of total fatty acids; the total fatty acid pattern is structured into SFA, MUFA and PUFA (Table 4). The average ratio of oleic acid (C 18:1n9) to linoleic acid (C 18:2n6) in LDL was 0.46±0.08 in the adjustment diet. CO diet had a significantly lower ratio (0.32±0.02) than the MO diet (0.62±0.06) ($p < 0.001$). After the crossover, a significantly inverse development of the LDL fatty acid pattern, corresponding to the changed diets, was observed ($p < 0.001$). This was dependent on the different influences of the linoleic acid-rich CO diet and the oleic acid-rich MO diets on the LDL lipids. Only the PUFA-rich corn oil diet was able to significantly reduce low density lipoprotein (LDL) cholesterol from the adjustment period at T2 ($p < 0.01$) (Table 5). After cross-over at T3 a non-significant (p=0.15) reduction of cholesterol was observed for the CO diet in LDL (T2 after MO diet: 3.88±0.74 vs. T3 after CO diet: 3.34±1.01; –13.9 %). The MO diet did not significantly alter LDL-C (T2 after CO diet: 3.06±0.88 vs. T3 after MO diet: 3.25±0.91). After the crossover at T3 the CO diet reduced plasma-C significantly, compared with the MO diet. VLDL-C was significantly lower with the CO diet than with the MO diet at T2 ($p < 0.01$). No significant change was observed for HDL-C, neither between groups nor between test periods. Plasma and VLDL TG concentrations at T2 were significantly reduced with the CO diet compared with the MO diet ($p < 0.05$; $p < 0.01$) and the baseline diet ($p < 0.01$). Except plasma-C no significant differences between groups in cholesterol and TG levels were found at T3.

The lipid profile at T4, five weeks after finishing the intervention, showed similar lipid levels as for the blood sampling period at T0, which is contributed to the subjects returning to their usual diets.

The LDL-MUFA content after the MO diet increased by 25 % ($p < 0.001$) compared with the baseline diet (T1), whereas PUFA concentrations were reduced compared with T1. In contrast the CO diet increased the LDL-PUFA content between T1 and T2 by 11 % ($p < 0.001$), which is due to the high content of linoleic acid in the corn oil (see Table 2). PUFA in the HDL fraction

Table 3 Mean daily intake of nutrients in diets

	Adjustment	Corn oil group	Mixed oil group
Energy (MJ)	11.8±1.3	12.4±1.1	12.2±1.3
Protein (% of energy)	13	14	14
Carbohydrates (% of energy)	52	55	55
Fat (% of energy)	35	31	31
Fatty acids (% of total energy)			
Saturated (SFA)	13.2	8.8	8.7
Monounsaturated (MUFA)	14.7	10.2	15.1
Oleic acid (18:1)	13.0	9.6	13.6
Polyunsaturated (PUFA)	7.1	12.0	7.2
Linoleic acid (C18:2 n-6)	5.1	11.3	5.7
Cholesterol (mg/d)	291±39	278±51	284±48
Dietary fiber (g/d)	41	42	42
S/M/P ratio	38/42/20	28/33/39	28/49/23

Table 4 Mean LDL-fatty acid pattern (% of total fatty acids) baseline (T0), after adjustment diet (T1), corn oil (CO) diet and mixed oil (MO) diet (T2)

	% of total fatty acids		
	T1	T2	
	(after adjustment)	CO group (corn oil)	MO group (mixed oil)
C14:0	1.04±0.3	0.85±0.3	1.05±0.3
C16:0	18.95±2.1	16.99±2.4	18.75±2.1
C16:1n7	1.57±0.5	0.88±0.5	1.38±0.4
C18:0	7.28±0.7	6.99±0.8	7.12±0.9
C18:1n9	17.71±1.8	15.01±2.5	22.63±2.9
C18:2n6	38.49±2.1	47.41±3.4	36.43±2.9
C18:3n3	0.95±0.2	0.49±0.1	0.43±0.1
C20:3n6	1.47±0.3	1.56±0.3	1.97±0.3
C20:4n6	7.27±1.2	6.03±1.4	6.45±1.4
C20:5n3	2.48±0.2	2.01±0.3	2.02±0.2
C22:4n6	2.04±0.3	1.32±0.4	1.26±0.4
C22:6n3	0.76±0.3	0.63±0.2	0.67±0.2
Total	100±0.0	100±0.0	100±0.0
Mean oleic/ linoleic acid ratio	0.46	0.32	0.62
SFA/MUFA/PUFA	27/20/53	25/16/59	27/25/48

Table 5 Plasma (Pl) and LDL levels of tested tocopherols, cholesterol and triglycerides at baseline (T0), after adjustment diet (T1), corn oil (CO) diet, mixed oil (MO) diet (T2), crossover (T3) and 5 weeks after the intervention (T4)

	T0	T1	T2		T3		T4	p for differences between groups at	
		Adjustment phase	CO group (corn oil)	MO group (mixed oil)	CO group (corn oil)	MO group (mixed oil)		T2	T3
Pl cholesterol (mmol/l)	4.3 ± 0.9	4.9 ± 0.9	4.3 ± 0.7	4.5 ± 0.7	3.9 ± 0.8	4.6 ± 0.7	4.4 ± 0.9	n. s.	$p < 0.01$
Pl triglycerides (mmol/l)	1.2 ± 0.4	1.1 ± 0.4	0.8 ± 0.3	1.1 ± 0.4	0.8 ± 0.3	0.9 ± 0.4	1.1 ± 0.4	$p < 0.05$	n. s.
VLDL cholesterol (mmol/l)	0.89 ± 0.13	0.83± 0.08	0.71 ± 0.08	0.86 ± 0.19	0.77 ± 0.14	0.79 ± 0.12	0.89 ± 0.13	$p < 0.01$	n. s.
VLDL triglycerides (mmol/l)	0.40 ± 0.12	0.35 ± 0.08	0.25 ± 0.09	0.35 ± 0.09	0.31 ± 0.12	0.27 ± 0.06	0.40 ± 0.12	$p < 0.01$	n. s.
LDL cholesterol (mmol/l)	3.66 ± 1.1	3.91 ± 0.87	3.06 ± 0.88	3.88 ± 0.74	3.34 ± 1.01	3.25 ± 0.91	3.71 ± 1.08	$p < 0.01$	n. s.
HDL cholesterol (mmol/l)	0.86 ± 0.20	0.89 ± 0.19	0.92 ± 0.13	0.94 ± 0.16	0.98 ± 0.14	0.95 ± 0.13	0.93 ± 0.15	n. s.	n. s.
LDL α-tocopherol (μmol/mmol cholesterol)	3.46 ± 0.53	2.51 ± 0.78	2.56 ± 0.74	3.32 ± 0.81	2.78 ± 0.78	3.91 ± 0.35	3.41 ± 0.55	$p < 0.001$	$p < 0.001$
LDL γ-tocopherol (μmol/mmol cholesterol)	0.21 ± 0.07	0.14 ± 0.06	0.51 ± 0.11	0.13 ± 0.04	0.55 ± 0.13	0.16 ± 0.04	0.19 ± 0.07	$p < 0.001$	$p < 0.001$
Pl α-tocopherol (μmol/l)	21.1 ± 3.8	20.4 ± 2.4	18.9 ± 3.7	23.2 ± 4.3	18.5 ± 4.1	23.1 ± 3.9	21.2 ± 3.8	$p < 0.005$	$p < 0.001$
Pl γ-tocopherol (μmol/l)	1.48 ± 0.51	1.28 ± 0.35	4.01 ± 0.92	1.09 ± 0.42	2.89 ± 0.77	1.04 ± 0.36	1.55 ± 0.43	$p < 0.001$	$p < 0.001$

* *n. s.* not significant

was also increased significantly ($p < 0.05$) in the CO group, whereas no change in fatty acid pattern was analyzed in the MO group. A significant correlation between LDL-C and increased levels of LDL-PUFA was determined in the CO group ($r=0.69$, $p < 0.01$).

After the two week adjustment (T1) no significant differences between the two test groups regarding age, body mass index and contents of tocopherols, in plasma and lipoproteins were determined. After two weeks of the test diet with CO, significant differences between plasma α-, and γ-tocopherol content compared with the MO were found (α-tocopherol: CO: 18.9 ± 3.7 μmol/L vs MO: 23.2 ± 4.3 μmol/L, $p < 0.005$). As expected, the plasma γ-tocopherol content of the CO group (4.0 ± 0.9 μmol/L) significantly increased ($p < 0.001$) in contrast to the mixed oil group (1.1 ± 0.4 μmol/L) (Table 5). This was, similar to the fatty acid pattern in LDL, due to the control of the diets. After crossover at T3 a corresponding significant change in plasma- and LDL-tocopherol levels due to the change of diets was observed (Table 5). The daily intake of γ-tocopherol from the CO was nearly 50-fold higher than the intake with the MO. The main carriers for tocopherol transport in blood were LDLs (41–49 % for γ-tocopherol; 51–59 % for α-tocopherol). A significant increase in LDL-α-tocopherol content was measured within the test period only with the MO but not with the CO diet. The CO predominated diet increased LDL transported γ-tocopherol relatively (41 % T1 vs 49 % T2) and absolutely ($p < 0.001$) to the 4-fold amount compared with the adjustment-phase (T1). A highly significant correlation ($r^2=0.73$; $p < 0.001$) was found between plasma and LDL γ-tocopherol concentrations in the CO group, but there was no correlation between the plasma and LDL α-tocopherol concentration. In the MO group only a significant correlation between plasma and LDL levels was observed for α-tocopherol ($r^2=0.71$; $p < 0.05$).

Discussion

This study was carried out in order to evaluate the effects of diets containing monounsaturated versus polyunsaturated fatty acids on the lipid metabolism of healthy, non-smoking male subjects with regard to the different tocopherol contents of the test oils. The two weeks of intervention following two weeks after crossover were chosen on the basis of tocopherol incorporation. This time period was shown to be long enough to assess significant changes in tocopherol plasma and lipoprotein levels (refer to Table 5). Even if the significant changes in the LDL fatty acid pattern could show that the different diets were incorporated (refer to Table 4), a longer treatment time of three weeks or more would give more detailed information on changes, especially of cholesterol, due to a complete substitution of the fatty acids. In this study the main source of MUFA was olive oil; the source of PUFA was the corn oil. The differences between the two diets were fatty acid pattern, percentage of MUFA and PUFA, contents in phytosterols and γ-tocopherol and the calculated α-TE/DE ratio.

Previous human studies which focused on the impact of diets with different fatty acid patterns have found different results especially regarding plasma cholesterol and triglyceride levels [3, 5, 8, 9]. Many studies, based on the impact of high oleic acid – or linoleic acid intake, on total cholesterol were carried out with hypercholesteremic individuals. We have made efforts to study the effect of the oils, incorporated in a nutritionally optimally balanced diet, without altering the intake of the other nutrients. Except for the food intake the subjects maintained their usual daily activities. Compliance of subjects and ingestion of diets were monitored by assessing the change of fatty acids in LDLs and HDLs. The

LDL fatty acid pattern of both groups was influenced by the diets consumed. The S/M/P-LDL ratio changed significantly from 27:20:53 in the adjustment period to 25:16:59 for the CO diet and to 27:25:48 with the MUFA-rich MO diet (see Table 4).

A significant plasma hypocholesteremic effect in this study was only shown with the PUFA-rich diet. The plasma-C level was slightly but not significantly lower for both test diets at T2, but after crossover at T3, the CO diet significantly decreased plasma-C compared with the MO group ($p < 0.01$). In LDL and VLDL, the TC-lowering effects of CO were observed earlier at T2, indicating that these fractions are the main carriers of cholesterol and therefore changes caused by different diets can be seen earlier. The lowering effect in and LDL-C and VLDL-C with the PUFA-rich CO diet compared with the MUFA-rich MO at T2 was significant ($p < 0.01$). All significant differences between groups were measured at T2, with the exception of plasma-C. At T3, subjects receiving the CO diet showed only a trend for lower LDL- and VLDL-C as well as plasma- and VLDL-TG compared to T2 (refer to Table 5). The reason for minor changes might be the lack of a wash out period and the immediate switch to the other diet. The results confirm earlier findings with PUFA-rich diets [18]. Dreon et al. observed a significant correlation of LDL particles regarding size and composition between diets rich in PUFAs and MUFAs [19]. In a similar study with MUFA-rich rapeseed oil and PUFA-rich sunflower oil, Valstra et al. found cholesterol lowering effects for both diets but with significantly more efficacy for the sunflower oil [9]. The hypocholesteremic effect in the present study, on the one hand, is based on the high PUFA content of CO (p/s-ratio=4.2) but, on the other hand, it might also be due to the high content of unsaponifiable substances, like quinones, carotenes and especially phytosterols of CO [20]. In fact, the ability of phytosterols to lower LDL-cholesterol levels has already been known for several decades [21]. In a recently published study, Howell et al. found a hypocholesteremic effect due to a supplementation with phytosterols to olive oil when compared to olive oil without phytosterol enrichment [22]. Corn oil is one of the best sources of phytosterols in plant oils with a total amount of more than 800 mg/100 g oil [22]. Long-term intervention studies with plant sterol-enriched margarines have shown the high potential of phytosterols in lowering serum cholesterol [23, 24].

The reduction of LDL-C by PUFA was also postulated by Mensink and Katan [25], who found for SFA an increased, for PUFA a decreased and for MUFA an unchanged LDL-C level. The HDL-C was unaffected by the diets; neither the MUFA nor the PUFA rich diet was able to influence HDL-C significantly. Our results support previous findings suggesting that diets rich in PUFA do not lower HDL-C levels when the intake of linoleic acid is moderate (< 10–13 % of total energy) [9, 22]. In the presented study the average daily intake of linoleic acid in the CO group was 12.2 % of total daily energy.

Plasma and VLDL TG levels were significantly lower with the CO diet compared with the MO diet and the adjustment diet. Similar results with PUFA-rich oils were found in some [9, 25] but not all [26] previous studies, whereas MUFA-rich oils either lowered the plasma TG or showed no effect [3, 27]. TG lowering effects are beneficial since high serum TG is recognized to be one risk factor of coronary heart disease [28].

At an equal daily intake of 80 g CO and MO and a similar intake of α-tocopherol (20 mg/d with the CO diet; 24 mg/d with the MO diet), the group with the CO diet had a significantly lower α-tocopherol concentration in plasma and LDL than the MO group. This result may be due to the high content of PUFA in the CO (P/S ratio = 4.2). Increased uptake of PUFAs may also affect the vitamin E status, by either impairing the absorption of the antioxidants [29] or by causing increased vitamin E consumption in plasma and tissues due to enhanced lipid peroxidation [30]. As assumed, due to the additional intake of γ-tocopherol (100 mg/d) in the corn-oil group, γ-tocopherol plasma and LDL values increased significantly.

In conclusion, our results showed a hypocholesteremic effect in plasma only for the PUFA-rich CO. The CO diet also reduced LDL-C and VLDL-C as well as plasma and VLDL TG levels, compared with the MO diet. These results are presumably caused by a combined effect of the high P/S ratio and the high contents of unsaponifiable substances in the CO, especially phytosterols. Generally our results show that a CO-rich diet has more influence on the lipoprotein metabolism than a MUFA rich diet when considering the intervention time of only two weeks for each diet. Subjects of the CO group showed decreased LDL-C and VLDL-C but not HDL-C.

In this way, PUFA-rich diets may also be considered in reducing the risk factors hypercholesterinemia and triglyceridemia and subsequently might lower the risk of coronary heart diseases.

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