Effects of coconut oil, butter, and safflower oil on lipids and lipoproteins in persons with moderately elevated cholesterol levels

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Abstract The physiological effects of coconut oil, butter, and safflower oil on lipids and lipoproteins have been compared in moderately hypercholesterolemic individuals. Twenty eight participants (13 men, 15 women) followed three 6-week experimental diets of similar macronutrient distribution with the different test fats providing 50% total dietary fat. Total cholesterol and low density lipoprotein cholesterol were significantly higher ($P < 0.001$) on the diet containing butter [6.8 ± 0.9, 4.5 ± 0.8 mmol/l] (mean ± SD), respectively than on the coconut oil diet (6.4 ± 0.8; 4.2 ± 0.7 mmol/l) when levels were significantly higher ($P < 0.01$) than on the safflower diet (6.1 ± 0.8; 3.9 ± 0.7 mmol/l). Findings with regard to the other measures of lipids and lipoproteins were less consistent. Apolipoprotein A-I was significantly higher on coconut oil (157 ± 17 mg/dl) and on butter (141 ± 23 mg/dl) than on safflower oil (132 ± 22 mg/dl). Apolipoprotein B was also higher on butter (86 ± 20 mg/dl) and coconut oil (91 ± 32 mg/dl) than on safflower oil (77 ± 19 mg/dl). However gender differences were apparent. In the group as a whole, high density lipoprotein did not differ significantly on the three diets whereas levels in women on the butter and coconut oil diet were significantly higher than on the safflower oil diet. Triacylglycerol was higher on the butter diet than on the safflower and coconut oil diets but the difference only reached statistical significance in women. Cholesteryl ester transfer activity was significantly higher on butter than safflower oil in the group as a whole and in women. Thus, despite similar total saturated fatty acid composition and a higher total percentage of the cholesterol-elevating fatty acids (lauric, myristic and palmitic acids) in the coconut oil diet compared with the butter diet, levels of total and low density lipoprotein cholesterol were lower on the coconut oil diet The data provide confirmation that butter, a source of fat rich in palmitic acid, has a greater hypercholesterolemic effect than coconut oil that is rich in lauric acid.—**Cox, C., J. Mann, W. Sutherland, A. Chisholm, and M. Skeaff.** Effects of coconut oil, butter, and safflower oil on lipids and lipoproteins in persons with moderately elevated cholesterol levels. *J. Lipid Res.* 1995. **36:** 1787–1795.

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Dietary recommendations aimed at reducing coronary heart disease (CHD) suggest an appreciable reduction in saturated fatty acids (SFA) from present levels of intake in most western countries (1, 2). A major justification for this recommendation is the association between SFA and total and low density lipoprotein (LDL) cholesterol. Despite the clear demonstration that stearic acid (C18:0) has a negligible effect on total and LDL-cholesterol (3–7), recommendations have not attempted to distinguish between individual SFA. In view of potentially undesirable effects of high intakes of n-6 polyunsaturated fatty acids (PUFA) (8–10), which are commonly used to replace SFA, it seems important to establish more precisely the effects of individual SFA on lipids and lipoproteins and also to compare the effects of widely used fats with varying proportions of saturated fatty acids. The early and apparently contradictory results of Keys, Anderson, and Grande (4) and Hegsted et al. (5) regarding the hypercholesterolemic effect of various saturated fatty acids have only been pursued in depth relatively recently (11, 12). It appears that of the three cholesterol-raising saturated fatty acids, the effect of myristic acid is most marked and that of palmitic acid greater than that of lauric acid. Coconut oil raises cholesterol levels when compared with other fats (4, 13, 14), but there is limited information regarding direct com-

Abbreviations: CHD, coronary heart disease; SFA, saturated fatty acid; VLDL, very low density lipoprotein; LDL, low density lipoprotein; HDL, high density lipoprotein; PUFA, polyunsaturated fatty acid; TAG, triacylglycerol; CETA, cholesteryl ester transfer activity.

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parisons with other major sources of saturated fatty acids (14). One study in humans (14) and two studies in experimental animals (15, 16) suggest that an increase in high density lipoprotein (HDL) cholesterol might account for at least some of the increase in total cholesterol though LDL levels are also higher on coconut oil than on beef fat (14). We have compared the effects of coconut oil, butter, and safflower oil on lipids and lipoproteins of moderately hypercholesterolemic subjects in order to clarify further the physiological effects of these common fat sources on lipids and lipoproteins and to facilitate more precise recommendations regarding dietary fat intake.

SUBJECTS AND METHODS

Subjects

Twenty eight subjects (13 men, 15 women, **Table 1**) aged 29–67 years with a plasma total cholesterol between 5.5 and 7.9 mmol/l and plasma triacylglycerols (TAG) less than 3 mmol/l completed the complex 6-month protocol. They were recruited by newspaper advertisement and from those who had participated in other dietary studies. Individuals believed to have familial hypercholesterolemia, familial combined or secondary hyperlipidemia, or who were on drugs known to influence lipid metabolism were excluded; thus the majority had polygenic hyperlipidemia. All subjects gave informed consent on the understanding they could withdraw at any time from the study which was approved by the Ethical Committee of the Otago Area Health Board.

TABLE 1. Characteristics of subjects on entry

	Male (n = 3)	Female (n = 15)
Age (yr)	55 ± 8	52 ± 10
Body weight (kg)	85 ± 10	64 ± 6
Body-mass index (kg/m^2)	26 ± 3	24 ± 2
Cholesteryl ester transfer activity (nmol/ml/h)	27.3 ± 8.3	17.8 ± 3.7
Fasting glucose (mmol/L)	5.3 ± 0.6	4.9 ± 0.4
Plasma lipids (mmol/L)		
Total cholesterol	6.5 ± 0.6	6.2 ± 0.8
HDL cholesterol	1.2 ± 0.2	1.8 ± 0.3
LDL cholesterol	4.3 ± 0.6	4.0 ± 0.9
Triacylglycerols	2.3 ± 1.0	1.4 ± 0.4
ApoE phenotype		
2/2	0	1
3/2	3	1
3/3	8	10
4/3	2	3

Values are means ± SD, except for apoE phenotype for which the frequency is given.

Experimental design

During a 6-week run-in period, participants completed a 5-day food record, in order to assess usual food and nutrient intakes, as well as questionnaires concerning their past, present, and family medical history and use of medications. The 28 subjects were randomized to one of three dietary sequences based on a Latin-square design (**Fig. 1**). Dietary compliance during each intervention period was assessed by 5-day diet records and by measurement of plasma triacylglycerol fatty acid composition at week 4 of each experimental diet. Nutrient intake was calculated using the computer program "Diet Entry and Storage/Diet Cruncher" (17) and data from the New Zealand Food Composition database (18). Prior to randomization and at weeks 4 and 6 of each experimental diet, body weight was recorded and a fasting venous blood sample was collected into vacutainer tubes, one containing anticoagulant (EDTA). Blood specimens were separated by centrifugation at 1100 *g* at 4°C. Lipids and lipoproteins were measured at each sampling time. Apolipoproteins A-I and B and cholesteryl ester transfer activity (CETA) were measured prior to randomization (baseline) and at week 4 of each diet period.

Diets

The three experimental diets were individually prescribed and based on the energy intake calculated from the self-selected diets reported during the run-in period. They were designed so that protein, carbohydrate, and fat provided approximately 17%, 47%, and 36% total energy, respectively (**Table 2**). The coconut oil (C) and butter (B) diets were intended to be high in SFA (approximately 20% total energy) whereas in the safflower oil diet (S), PUFA and SFA were intended to each provide about 10% total energy. The diets were further designed so that the predominant fatty acid of each fat source (C12:0, C16:0 and C18:2n–6 for coconut oil, butter, and safflower oil, respectively) were roughly equivalent in each of the experimental diets. This was achieved by rigidly prescribing the dietary fat from three sources: fat from food (lean meat, fish, chicken, dairy products), "test" fats (coconut oil, butter, safflower oil) and "exchange" fats (which could be used in place of "test" fats). During each diet the appropriate test fats and several appropriate replacement foods containing "exchange" fats were provided free of charge to the participants. For example, for an 8.4 MJ/day diet, total dietary fat requirement was calculated to be 84 g. For diet C, 39 g of total fat was from coconut oil (providing 17.2 g C12:0) and the remaining 36 g from "fat from food". For diet B, 39 g of the total fat was from butter (providing 17.6 g C16:0), 20 g from "fat from food" and one butter "exchange", which for an 8.4 MJ/day diet

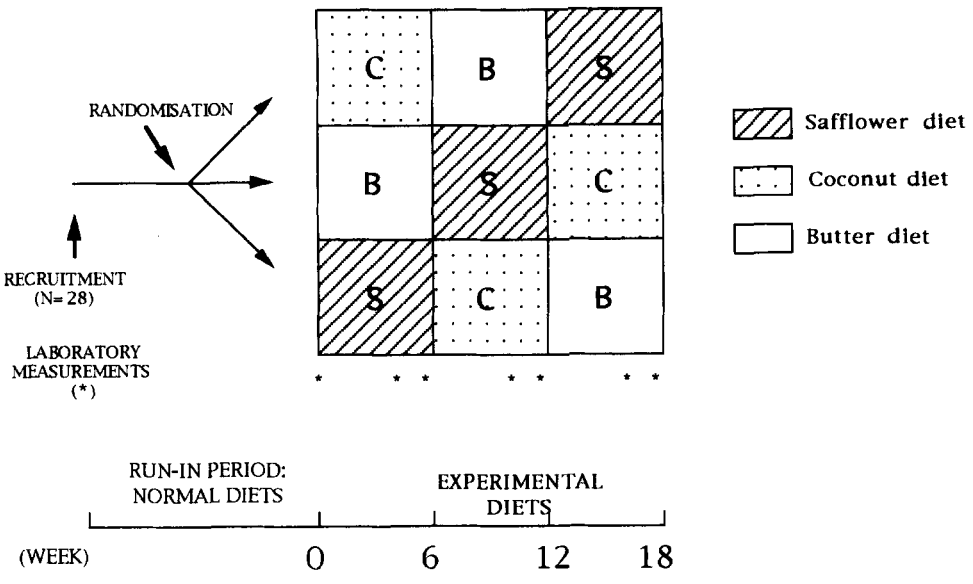

Fig. 1. Experimental design.

could include 32 g of cheddar cheese, 30 ml of cream or portions of various cakes and/or muesli bars made with butter. For diet S, 24 g of the total fat was from safflower oil (providing 17.1 g C18:2n-6), and the remainder from "fat from food". The diets were designed to contain roughly similar amounts of cholesterol (300–350 mg/day) which necessitated the addition of egg yolk to diets S and C. Detailed dietary instructions, menu suggestions, and recipes were provided and reinforced during personal interviews and telephone calls at regular intervals. Participants were encouraged to continue their usual pattern of physical activity and all other aspects of their lifestyle throughout the study.

TABLE 2. Recommended daily nutrient composition in a sample diet

	Butter	Coconut	Safflower
Energy (MJ)	8.6	8.8	8.7
Protein (% total energy)	16	18	20
Carbohydrate (% total energy)	48	45	46
Cholesterol (mg)	315	341	375
Fiber (g)	21	30	27
Total fat (% total energy)	36	37	35
SFA[a] (% total energy)	21	21	11
PUFA[a] (% total energy)	2	5	11
MUFA[a] (% total energy)	9	8	9
Fatty acids (g)			
C12:0	1.9	18.0	0.1
C14:0	6.7	6.5	2.1
C16:0	18.8	12.7	12.9
C18:0	7.6	3.6	3.6
C18:1	20.0	18.2	17.6
C18:2n-6	0.81	8.0	19.3

Quantities of cholesterol, fiber, and individual fatty acids depend upon the total energy requirements of participants.

[a]SFA, PUFA and MUFA refer to saturated, polyunsaturated, and monounsaturated fatty acids respectively.

Laboratory methods

Cholesterol and triglyceride concentrations in plasma and plasma fractions were measured by enzymatic methods using kits and calibrators from Boehringer-Mannheim and Roche Diagnostics. LDL cholesterol was obtained by difference at week 4 and by calculation using the Friedewald formula (19) at week 6. Very low density lipoproteins (VLDL) were separated by ultracentrifugating EDTA plasma according to the methods described by the Lipid Research Clinic protocol (20). HDL cholesterol was measured in the supernatant after precipitation of apoB-containing lipoproteins with phosphotungstate/magnesium chloride solution (21). Measurements were made on a Cobas Fara analyzer (Roche Diagnostics). The coefficient of variation for measurements during the study period was 1.6% for cholesterol and 3.4% for triglycerides in the Royal Australasian College of Pathologists Quality Assurance Programme. An isotopic assay, which uses endogenous lipoproteins, was used to determine plasma CETA as the rate of transfer of newly synthesized cholesteryl ester from HDL to VLDL and LDL (22). The coefficient of variation for this assay is 10% and values correlate closely with cholesteryl ester mass transfer measured by chemical methods (23). ApoA-I and apoB were measured by immunoturbimetry (24) using Boehringer kits (CV 2.6 and 6%, respectively). ApoE phenotype was

determined by isoelectric focussing of VLDL apolipoproteins by modification of a published method (25). Plasma lipids were extracted according to the procedure of Bligh and Dyer (26). Triacylglycerols were separated from other lipids by thin-layer chromatography of the lipid extract on silica gel G using a solvent system containing hexane–diethylether–acetic acid 85:15:1 (by volume). Triacylglycerol fatty acids were methylated for 2 h at 80°C with 6% H_2SO_4 in methanol. Fatty acid methyl esters were separated using a 25 m DB-225 MegaBore column (J&W Scientific) installed in a HP5890 Hewlett-Packard gas–liquid chromatograph (GLC) equipped with flame ionization detector. The following conditions were maintained during GLC operation: oven temperature, 210°C; detector and injector temperatures, 250°C; helium carrier gas flow, 6.5 ml/min; split ratio 4:1. Fatty acid methyl esters were identified by matching retention times with 99% pure commercial standards (Nu-Chek Prep). The precision of the fatty acid analysis was determined by repeated analysis of a pooled plasma sample. The coefficient of variation for all fatty acids reported in this paper ranged from 1 to 4%.

Statistical analysis

Preliminary analyses included examination of the diet sequence effect within an ANOVA to test whether the diet sequence significantly influenced the relative treatment response. If the sequence effect was nonsignificant then the three diets were compared by ANOVA. When a significant effect of diet was identified by ANOVA, differences between individual diet pairs (i.e., B vs. C, B vs. S, and C vs. S) were compared by paired Student's *t*-tests. As there were no significant differences between the values for any of the lipid and lipoprotein measurements at 4 and 6 weeks, the mean of the two values was taken as each individual's value for that dietary period. Furthermore, as diet sequence appeared to have no effect on the results, this was disregarded in subsequent analyses.

RESULTS

Results of the laboratory measurements are presented in Tables 3 and 4. Mean body weight (85 ± 10 kg for men, 64 ± 6 kg for women) remained unchanged throughout the study.

Total and low density lipoprotein cholesterol (Table 3)

Total cholesterol and LDL-cholesterol levels were highest on diet B and lowest on diet S, with intermediate levels on diet C; all differences among the diet periods were highly statistically significant ($P < 0.001$). Similar trends were apparent in men and women.

High density and very low density lipoprotein cholesterol and triacylglycerol (Table 3)

HDL-cholesterol was consistently lower and VLDL-cholesterol and TAG were consistently higher in men than women. In the total group HDL cholesterol did not differ significantly on the three diets. However, levels in women on the butter and coconut oil diets were significantly higher than on the safflower oil diet. Triacylglycerol was higher on the butter diet than on safflower and coconut oil, the differences only reaching statistical significance in women.

Apolipoproteins A-I and B and cholesteryl ester transfer activity (Table 4)

In the total group, levels of apoA-I were higher on diets C and B than on S, the differences reaching statistical significance only in men. Levels were generally higher in women than men except during diet C when observed levels were highest and similar in both men and women. ApoB was lowest on diet S in the total group and differences were less marked when data for women and men were examined separately. CETA was consistently higher in men than in women. Activity was significantly higher on butter than safflower oil in the group as a whole but when examining the subjects separately, the difference was only significant in women. Levels tended to be lower on coconut oil than on butter but none of the differences achieved statistical significance.

Compliance with dietary instructions (Tables 5 and 6)

The analysis of the 5-day dietary records during the three experimental periods is shown in **Table 5.** Total energy intake remained constant as did average percentage of energy derived for macronutrients, monounsaturated fatty acids, alcohol, and average daily intake of dietary fiber and cholesterol. Percentage energy values from SFA and PUFA were identical on diets B and C and strikingly different from S when the P:S ratio was five times greater than that calculated for B and C. On all three diets average total fat intake was 84 g/day. On diets B and C, saturated fatty acids derived principally from butter and coconut oil contributed about 44 g. On diet S, PUFA derived principally from safflower oil provided 24 g of fat and approximately 11% of total daily energy.

Plasma triacylglycerol fatty acid composition measured after 4 weeks of each experimental diet is shown in **Table 6.** The significant changes in triacylglycerol fatty acids parallelled changes in dietary fatty acid intake with significantly higher levels of linoleic acid during the

TABLE 3. Plasma lipids and lipoproteins during coconut, butter, and safflower diets.

Diet	TC	LDL-C	HDL-C	VLDL-C	TAG
Butter					
Total group	6.8 ± 0.9 (263 ± 33)	4.5 ± 0.8 (175 ± 30)	1.4 ± 0.4 (56 ± 14)	0.65 ± 0.65 (24 ± 25)	2.0 ± 1.3 (177 ± 115)
Male	7.0 ± 1.0 (269 ± 38)	4.7 ± 0.9 (181 ± 35)	1.2 ± 0.2[c] (45 ± 8)	1.0 ± 0.8[b] (38 ± 31)	2.6 ± 1.6[a] (230 ± 142)
Female	6.7 ± 0.7 (258 ± 28)	4.4 ± 0.7 (170 ± 26)	1.7 ± 0.3 (66 ± 10)	0.3 ± 0.2 (13 ± 8)	1.5 ± 0.4 (133 ± 35)
Coconut					
Total group	6.4 ± 0.8 (249 ± 29)	4.2 ± 0.8 (163 ± 29)	1.5 ± 0.4 (57 ± 15)	0.54 ± 0.51 (21 ± 19)	1.8 ± 1.0 (159 ± 89)
Male	6.6 ± 0.9 (255 ± 34)	4.4 ± 0.9 (171 ± 33)	1.2 ± 0.1[c] (45 ± 6)	0.8 ± 0.6[b] (32 ± 22)	2.4 ± 1.1[c] (231 ± 97)
Female	6.3 ± 0.6 (243 ± 24)	4.0 ± 0.6 (156 ± 25)	1.8 ± 0.3 (68 ± 11)	0.3 ± 0.2 (10 ± 6)	1.3 ± 0.3 (115 ± 27)
Safflower					
Total group	6.1 ± 0.8 (233 ± 29)	3.9 ± 0.7 (151 ± 28)	1.4 ± 0.3 (54 ± 13)	0.53 ± 0.54 (20 ± 20)	1.7 ± 1.0 (151 ± 89)
Male	6.2 ± 0.8 (239 ± 30)	4.0 ± 0.6 (155 ± 21)	1.2 ± 0.3[c] (46 ± 10)	0.8 ± 0.6[b] (31 ± 25)	2.3 ± 1.2[b] (204 ± 106)
Female	5.9 ± 0.7 (228 ± 28)	3.8 ± 0.9 (148 ± 33)	1.6 ± 0.3 (62 ± 10)	0.3 ± 0.2 (10 ± 7)	1.3 ± 0.3 (115 ± 27)
Total group comparisons					
Butter v Coconut	$P = 0.001$	$P = 0.001$	$P = 0.17$	$P = 0.13$	$P = 0.01$
Butter v Safflower	$P = 0.001$	$P = 0.001$	$P = 0.25$	$P = 0.02$	$P = 0.02$
Coconut v Safflower	$P = 0.001$	$P = 0.004$	$P = 0.08$	$P = 0.75$	$P = 0.48$
Female comparisons					
Butter v Coconut	$P = 0.003$	$P = 0.005$	$P = 0.15$	$P = 0.09$	$P = 0.01$
Butter v Safflower	$P = 0.001$	$P = 0.001$	$P = 0.02$	$P = 0.06$	$P = 0.005$
Coconut v Safflower	$P = 0.01$	$P = 0.14$	$P = 0.01$	$P = 0.88$	$P = 0.78$
Male comparisons					
Butter v Coconut	$P = 0.01$	$P = 0.07$	$P = 0.83$	$P = 0.33$	$P = 0.44$
Butter v Safflower	$P = 0.001$	$P = 0.006$	$P = 0.51$	$P = 0.13$	$P = 0.16$
Coconut v Safflower	$P = 0.004$	$P = 0.01$	$P = 0.57$	$P = 0.69$	$P = 0.52$

Values are mean ± SD in mmol/l and mg/dl in brackets. Statistics by paired Student's *t*-tests.
[a]Significantly different from females ($P < 0.05$).
[b]Significantly different from females ($P < 0.01$).
[c]Significantly different from females ($P < 0.001$).

safflower diet, lauric acid during the coconut diet, and myristoleic acid during the butter diet.

DISCUSSION

We have compared the effects of coconut oil, butter, and safflower oil on lipids and lipoproteins in a group of moderately hypercholesterolemic subjects. The study was undertaken amongst free-living individuals and it could be argued that reduced compliance might have influenced the results. The participants were all highly motivated individuals who were given detailed and specific dietary advice as well as free supplies of the test fats. The experimental design reduced the possibility that declining enthusiasm over the duration of the study might have accounted for reduced compliance with any particular experimental diet. Five-day dietary records meticulously kept throughout the study as well as fatty acid composition of plasma triacylglycerol confirmed a high level of compliance with each test fat. Thus, we would argue that our approach was appropriate for studying the physiological effects of the various test fats as well as having practical application, having been carried out in an outpatient setting rather than a metabolic ward.

The findings concerning total and LDL cholesterol were unequivocal. The coconut oil and butter diets resulted in increased total cholesterol and LDL cholesterol relative to the safflower oil diet, however, the levels of both these lipid measurements were significantly lower in the coconut oil than on the butter diet. There is no doubt that coconut oil is associated with increased levels of total cholesterol and LDL cholesterol when

TABLE 4. Apolipoproteins and cholesteryl ester transfer activity (CETA) during coconut, butter, and safflower diets

Diet	CETA	ApoA-I	ApoB
	nmol/ml/h	*mg/dL*	*mg/dL*
Butter			
Total Group	24.2 ± 9.4	141 ± 23	86 ± 20
Male	28.7 ± 11.0[a]	127 ± 13[c]	93 ± 19
Female	20.3 ± 5.6	154 ± 23	80 ± 18
Coconut			
Total Group	22.8 ± 8.1	157 ± 17	91 ± 32
Male	27.4 ± 8.5[b]	158 ± 23	109 ± 37[b]
Female	18.9 ± 5.4	155 ± 19	76 ± 16
Safflower			
Total Group	21.8 ± 9.9	132 ± 22	77 ± 19
Male	26.6 ± 11.4[a]	118 ± 17[c]	83 ± 21
Female	17.5 ± 6.1	146 ± 17	72 ± 17
Total group comparisons			
Butter v Coconut	$P = 0.24$	$P = 0.06$	$P = 0.34$
Butter v Safflower	$P = 0.02$	$P = 0.04$	$P = 0.02$
Coconut v Safflower	$P = 0.36$	$P = 0.01$	$P = 0.04$
Female group comparisons			
Butter v Coconut	$P = 0.25$	$P = 0.85$	$P = 0.28$
Butter v Safflower	$P = 0.01$	$P = 0.25$	$P = 0.02$
Coconut v Safflower	$P = 0.36$	$P = 0.19$	$P = 0.24$
Male group comparisons			
Butter v Coconut	$P = 0.55$	$P = 0.05$	$P = 0.17$
Butter v Safflower	$P = 0.31$	$P = 0.09$	$P = 0.17$
Coconut v Safflower	$P = 0.70$	$P = 0.01$	$P = 0.08$

Values are mean ± SD. Statistics by paired Student's *t*-tests.
[a]Significantly different from females ($P < 0.05$).
[b]Significantly different from females ($P < 0.01$).
[c]Significantly different from females ($P < 0.001$)

compared with oils containing predominantly unsaturated fatty acids (13, 14). This observation is not surprising as coconut oil contains a high proportion of the three saturated fatty acids (45% lauric acid C12:0; 17% myristic acid, C14:0, 8% palmitic acid, C16:0) which as a group have long been known to be associated with increased levels of total cholesterol and LDL cholesterol (4, 5, 27). However, there has been less certainty with regard to the effects of coconut oil when compared with other fats that are also rich in saturated fatty acids. Reiser et al. (14) compared diets rich in beef fat, coconut oil, and safflower oil. They found total and LDL cholesterol to be higher on coconut oil than on beef fat, a finding that might have been predicted in view of the fact that the former has a much higher proportion of total cholesterol-elevating saturated fatty acids. In beef fat, C12:0 and C14:0 together contributed less than 30% total fatty acids whereas in coconut oil they contributed almost 62% of total fatty acids. Hegsted and colleagues (5) included both coconut oil and butter in their classical studies in the 1960s and found a similar effect of the two fats on changes in cholesterol levels. However, their studies did not involve direct comparisons of the two fats and the overall fatty acid composition of their experimental diets differed appreciably from our own. Furthermore, other early studies comparing coconut oil with other sources of saturated fatty acids, reviewed by Reiser in 1973 (13), also do not contribute a great deal to a discussion concerning the relative effects of fats differing in saturated fatty acid composition because of the relatively unsophisticated experimental designs. There has, however, been a great deal of interest in the effects of the component saturated fatty acids ever since the apparently contradictory findings of Keys et al. (4) and Hegsted et al. (5).

The results of the Keys group (4) suggested that the cholesterol-raising potential of lauric, myristic, and palmitic acids was similar, whereas Hegsted and colleagues (5) found that lauric acid had a cholesterol-raising action that was one third that of palmitic acid and one quarter that of myristic acid. In a more recent carefully controlled metabolic ward study, Denke and Grundy (11) compared liquid formula diets rich in lauric, palmitic, and oleic acids. Both lauric and palmitic acids were associated with higher levels of total and LDL cholesterol when compared with oleic acid but the rise in LDL cholesterol on the high lauric acid diet was about two thirds of that on the high palmitic acid diet. Myristic

TABLE 5. Daily nutrient intake calculated from 5-day food diaries obtained at baseline and during the butter, coconut, and safflower diets

	Baseline	Butter	Coconut	Safflower
Energy (MJ)	8.3 ± 2.0	8.5 ± 2.1	8.3 ± 2.1	8.1 ± 2.2
Protein (% energy)	17 ± 3	16 ± 3	16 ± 3	16 ± 3
Carbohydrate (% energy)	48 ± 7	46 ± 7	46 ± 5	46 ± 5
Dietary fiber (g)	23 ± 5	24 ± 7	23 ± 7	26 ± 8
Total fat (% energy)	34 ± 7	37 ± 7	38 ± 5	37 ± 4
SFA (% energy)	14 ± 4	20 ± 4[a]	20 ± 4[a]	11 ± 3[b]
PUFA (% energy)	5 ± 2	4 ± 2[a]	4 ± 1[a]	11 ± 3[b]
MUFA (% energy)	11 ± 2	10 ± 2	10 ± 3	11 ± 2
P:S ratio	0.4 ± 0.3	0.2 ± 0.1[a]	0.2 ± 0.1[a]	1.1 ± 0.5[b]
Cholesterol (mg)	273 ± 91	279 ± 109	270 ± 93	248 ± 95

Values are mean ± SD. SFA, PUFA, and MUFA refer to saturated, polyunsaturated, and monounsaturated fatty acids respectively. Numbers not sharing a common superscript are significantly different; $P \leq 0.0001$ (paired Student's *t*-test).

acid appears to have an even more marked elevating effect on total and LDL cholesterol (5, 12). Multiple regression analyses (5) and a meta-analysis (28) based on many experiments suggested that its cholesterol-raising potential might be as great as four to six times that of palmitic acid, but a recent controlled study found a 1.5-fold greater effect when comparing the cholesterol-raising potential of myristic and palmitic acids (12). These findings are compatible with those of Sundram, Hayes, and Siru (29) who compared palmitic acid with a lauric plus myristic acid mixture while holding all other fatty acids constant (29). They found that palmitic acid was associated with lower levels of total and LDL cholesterol, an observation that could be explained by the marked effect of myristic acid on these lipid fractions.

Because of the paucity of data concerning direct comparisons of naturally occurring fat sources, especially those fats rich in different saturated fatty acids, Katan, Zock, and Mensink (30) developed a formula to predict the effects of changing the sources of dietary fat in a typical Dutch diet. Using their equation based on studies examining the effects of individual fatty acids, palm kernel oil and coconut oil appeared to have the most unfavorable effect on total and LDL cholesterol, with butter and palm oil having less than half this effect when compared with the present average Dutch diet. This observation is explained by the equal weighting given in their formula to the three cholesterol-raising fatty acids (C12:0, C14:0, C16:0). Our study, which involved a direct comparison of coconut oil, confirms the limitations of this approach for comparing naturally occurring fat sources. The data presented here do not permit a direct comparison of individual saturated fatty acids as the mix of fatty acids inevitably present in an experiment comparing naturally occurring fat sources could have influenced the outcome. Nevertheless, it is of interest to note (Table 2) that the total quantity of cholesterol-raising fatty acids on the coconut oil diet (37.2 g) was appreciably greater than that on the butter diet (27.4 g), that the percentage of myristic acid was identical on the two diets, and that percentages of the predominant fatty acids of the two test fats (palmitic in butter and lauric in coconut) were similar. We found that the coconut oil diet has approximately half the total- and LDL-cholesterol-raising potential of the butter diet when compared with the coconut oil diet. These findings are in between those of Hegsted et al. (5) and Denke and Grundy (11) when comparing lauric and palmitic acid and therefore provide further confirmation of their conclusions. ApoB is the major apolipoprotein of LDL and changes in response to diet might be expected to follow those observed in LDL-cholesterol. We observed higher levels on both the butter and coconut diets compared with safflower oil, but differences on the two diets rich in saturated fatty acids were not statistically significant.

TABLE 6. Fatty acid composition of plasma triacylglycerol

Fatty Acid	Butter (n = 15)	Coconut (n = 15)	Safflower (n = 14)
		mol %	
C12:0	0.36 ± 0.32[a]	1.21 ± 0.96[b]	0.18 ± 0.16[a]
C14:0	4.36 ± 1.37[a]	4.68 ± 1.71[a]	2.59 ± 1.09[b]
C14:1	0.55 ± 0.22[a]	0.35 ± 0.20[b]	0.28 ± 0.21[b]
C16:0	33.25 ± 3.20[a]	31.86 ± 4.58[a]	29.34 ± 4.95[b]
C16:1	5.96 ± 0.66	5.96 ± 2.07	5.38 ± 0.83
C18:0	4.51 ± 1.56[a]	3.3 ± 1.00[b]	3.18 ± 1.09[b]
C18:1n-9	34.7 ± 3.71	36.19 ± 4.71	37.11 ± 5.50
C18:2n-6	8.33 ± 2.96[a]	9.93 ± 3.61[a]	15.18 ± 4.8[b]
C18:3n-6	0.22 ± 0.11[a]	0.27 ± 0.19[a,b]	0.34 ± 0.16[b]
C18:3n-3	0.68 ± 0.22	0.59 ± 0.27	0.63 ± 0.36
C20:4n-6	0.53 ± 0.16	0.64 ± 0.32	0.66 ± 0.35
C20:5n-3	0.11 ± 0.08	0.12 ± 0.07	0.09 ± 0.07
C22:6n-3	0.18 ± 0.18	0.21 ± 0.20	0.18 ± 0.19

Values are mean ± SD. Numbers not sharing a common superscript are significantly different; $P < 0.01$ except for C16:0 (Coconut vs Safflower Oil) and C18:3n-6 (butter vs safflower oil) where $P \leq 0.05$.

ApoA-I, the major apolipoprotein of HDL, was higher on the coconut and butter diets than on safflower oil. Differences only reached statistical significance in men. There is a suggestion of higher levels on coconut oil than butter but the difference is not statistically significant. HDL-cholesterol on the other hand did not differ significantly on the three diets in the total group but in women levels were higher on butter and coconut diets than on safflower oil. Previous studies have generally found HDL-cholesterol to be lower when diets rich in polyunsaturated fatty acids were compared with those high in saturated fatty acids (27). However, there is much less information concerning the effects of various saturated fatty acids on HDL-cholesterol. Reiser et al. (14) found significantly higher levels of HDL cholesterol on coconut oil compared with beef fat and safflower oil, predictable results in view of the appreciably higher content of cholesterol-raising fatty acids in the coconut oil than in the other two experimental diets. Denke and Grundy (11) found no differences in HDL cholesterol when comparing palmitic and lauric acid. Zock, de Vries, and Katan (12) found that myristic acid was associated with slightly higher HDL-cholesterol and apoA-I levels than were observed on palmitic acid rich diets. Unfortunately, few other data are available for comparison but it seems possible that myristic acid, the fatty acid that has the greatest effect in raising total and LDL-cholesterol, also has an elevating effect on HDL-cholesterol and apoA-I, though the proportional increase is not so great. However, our data do not exclude the possibility of differential effects of palmitic and lauric acids on HDL-cholesterol and apoA-I. Our study and that of Denke and Grundy (11) may have lacked the precision necessary to detect the relatively small changes in HDL-cholesterol that tend to occur with dietary changes.

Previous in vitro studies have shown that transfer of cholesteryl esters from synthetic HDL with cholesteryl esters rich in laurate or myristate is slow compared with rates when they are replaced with other fatty acids (31). Furthermore, Groener and colleagues (32) have documented a tendency towards lower cholesteryl ester transfer protein activity in subjects consuming a diet rich in polyunsaturated fatty acids compared with the activity when they switched to a diet rich in saturated fats. Thus, in our data we might have expected lowest CETA during the safflower oil diet, highest on the butter diet, with intermediate levels of activity on the coconut diet. Such trends were observed, though only the butter/safflower oil difference reached statistical significance. There is evidence that transfer of cholesteryl esters into apoB-containing lipoproteins may influence plasma LDL cholesterol concentration (33). Thus it is conceivable that differences in plasma CETA may contribute to the corresponding differences in LDL cholesterol concentration between diet groups in the current study. On the other hand, these differences in CETA may be due to altered levels of apoB-containing lipoproteins that are acceptors of cholesteryl ester transferred from HDL. We cannot exclude this possibility particularly as the CETA assay we used is dependent on levels of endogenous lipoproteins in plasma.

Higher plasma CETA in men than women is consistent with their higher plasma triglyceride levels which have been previously associated with accelerated cholesteryl ester transfer (34). Other well-documented gender differences in lipids and lipoproteins were also apparent in our data. Triglycerides and VLDL cholesterol, and to a lesser extent total and LDL-cholesterol and apoB, were all higher in men whereas HDL-cholesterol and apoA-I were higher in women. Some groups have reported more variable responses to changes in dietary fat in women (12) and others have reported gender-specific effects of dietary fats on HDL-cholesterol (35, 36). While our data do provide some evidence for gender-specific effects, namely a more striking effect of dietary changes on HDL and triglyceride in women, it is conceivable that lack of statistical power may account for failure to detect these and other apparent gender-specific changes in the other sex. It is further important to emphasize that the most striking differences in response to changes in dietary fat, the changes in total and LDL cholesterol, were virtually identical in men and women.

In summary, our data provide convincing evidence that coconut oil rich in lauric acid has a lesser effect than butter, which is high in palmitic acid, on total and LDL cholesterol in hypercholesterolemic men and women. The findings suggest that, in certain circumstances, coconut oil might be a useful alternative to butter and hydrogenated vegetable fats. However, it should be noted that in individuals and populations with a tendency to obesity, all fat sources should be restricted and that depending upon the requirements of individuals, fats high in stearic acid or *cis*-monounsaturated fatty acids may be preferable to coconut oil.

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