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# Original Communication

# The effect of replacing dietary saturated fat with polyunsaturated or monounsaturated fat on plasma lipids in free-living young adults

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**Objective:** To examine, in free-living adults eating self-selected diets, the effects on plasma cholesterol of substituting saturated fat rich foods with either n-6 polyunsaturated or monounsaturated fat rich foods while at the same time adhering to a total fat intake of 30–33% of dietary energy.
**Design:** Two randomised crossover trials.
**Setting:** General community.
**Subjects:** Volunteer sample of healthy free-living nutrition students at the University of Otago. Trial I, $n = 29$; and trial II, $n = 42$.
**Interventions:** In trials I and II participants were asked to follow for $2\frac{1}{2}$ weeks a diet high in saturated fat yet with a total fat content that conformed to nutrition recommendations (30–33% energy). During the 2 1/2 week comparison diet, saturated fat rich foods were replaced with foods rich in n-6 polyunsaturated fats (trial I) whereas in trial II the replacement foods were rich in monounsaturated fats. Participants were asked to maintain a total fat intake of 30–33% of energy on all diets.
**Main outcome measures:** Energy and nutrient intakes, plasma triglyceride fatty acids, and plasma cholesterol.
**Results:** When replacing saturated fat with either n-6 polyunsaturated fat or monounsaturated fat, total fat intakes decreased by 2.9% energy and 5.1% energy, respectively. Replacing saturated fat with n-6 polyunsaturated fat (trial I) lowered plasma total cholesterol by 19% [from 4.87 (0.88) to 3.94 (0.92) mmol/l, mean (s.d.)], low density lipoprotein cholesterol by 22% [from 2.87 (0.75) to 2.24 (0.67) mmol/l], and high density lipoprotein cholesterol by 14% [from 1.39 (0.36) to 1.19 (0.34) mmol/l], whereas replacing saturated fat with monounsaturated fat (trial II) decreased total cholesterol by 12%, low density lipoprotein cholesterol by 15%, and high density lipoprotein cholesterol by 4%, respectively. The change in the ratio of total to high density lipoprotein cholesterol was similar during trial I and trial II.
**Conclusions:** Young adults are very responsive to dietary-induced changes in plasma cholesterol even when an isocaloric replacement of saturated fat with n-6 polyunsaturated or monounsaturated fat is not achieved. Replacing saturated fat with either n-6 polyunsaturated or monounsaturated fat is equally efficacious at reducing the total to high density lipoprotein cholesterol ratio.
**Sponsorship:** University of Otago, Meadow Lea Ltd.
**Descriptors:** young adults; plasma triglyceride fatty acids; saturated fat; monounsaturated fat; n-6 polyunsaturated fat; plasma cholesterol
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## Introduction

There is widespread agreement that decreasing saturated fat intake is the cornerstone of dietary recommendations to reduce blood cholesterol-mediated risk of cardiovascular disease (Grundy & Denke, 1990; NCEP, 1991). However, there is considerably less agreement on whether carbohydrate, monounsaturated fat or n-6 and n-3 polyunsaturated fats should replace saturated fat in the diet (Connor & Connor, 1997; Katan *et al*, 1997; NCEP, 1993).

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Many argue in favour of replacing saturated fat with monounsaturated and polyunsaturated fat rather than carbohydrate because low-fat high-carbohydrate diets — despite causing a favourable decrease in plasma low density lipoprotein cholesterol — often lead to decreased plasma high density lipoprotein cholesterol and increased triglyceride concentrations (Katan *et al*, 1997).

Whether monounsaturated or polyunsaturated fat is the best substitute for saturated fat remains undecided. Replacing saturated fat with polyunsaturated fat produces a slightly larger decrease in total cholesterol than with monounsaturated fat; however, the effect on high density lipoprotein cholesterol is uncertain (Becker *et al*, 1983; Gustafsson *et al*, 1992; Howard *et al*, 1995; Mattson & Grundy, 1985; Mensink & Katan, 1989; Vega *et al*, 1982; Wardlaw & Snook, 1990). Results from two carefully controlled studies using liquid formula diets indicate that polyunsaturated fat decreases high density lipoprotein cholesterol concentrations (Mattson & Grundy, 1985; Vega *et al*, 1982). This is in contrast to studies that have noted no change in high density lipoprotein cholesterol concentrations when feeding either liquid formula or solid foods rich in polyunsaturated fat (Becker *et al*, 1983; Howard *et al*, 1995; Mensink & Katan, 1989).

The bulk of studies that have compared the cholesterolaemic effects of substituting monounsaturated or polyunsaturated fat for saturated fat have done so while keeping the total fat content of the diets constant; usually at levels near or above 35% of dietary energy (Howard *et al*, 1995; Mata *et al*, 1992; Mensink & Katan, 1989; Noakes & Clifton, 1998). It is unclear how the results of these controlled intervention studies translate to dietary recommendations in most Westernised countries that advocate a reduction in total fat intake to roughly 30% of energy; in other words, can people who freely select their own foods according to recommended dietary guidelines isocalorically replace saturated fat with monounsaturated or polyunsaturated fat?

The purpose of the present study was to examine, in free-living young adults eating self-selected diets, the effect on plasma cholesterol of replacing saturated fat rich foods with foods rich in n-6 polyunsaturated fat (from sunflower and safflower oils) or monounsaturated fat (from canola oil) while keeping total fat content of the diet at 30–33% of dietary energy.

Furthermore, very few studies have investigated how changing the composition of dietary fat affects the plasma cholesterol levels of young adults. It is important to assess the effectiveness of dietary strategies to reduce plasma cholesterol levels in young adults as plasma cholesterol in this age group is associated with cardiovascular disease risk in later life (Klag *et al*, 1993; Myers *et al*, 1995).

## Methods

Participants were recruited voluntarily from students enrolled in a Human Nutrition course at the University of Otago. The Human Ethics Committee of the University of Otago approved the study and all participants gave informed consent. The participants were predominately Caucasian. Participants were excluded if they were on a special diet, affected by a metabolic disorder, on medication known to alter plasma lipids or had a total cholesterol greater than 6.5 mmol/l. Participants were asked not to change their smoking habits and to maintain their regular lifestyles, including physical activity, for the duration of the study.

The study was conducted in two phases: a high saturated fat diet was compared with a n-6 polyunsaturated fat-rich diet in trial I and a monounsaturated fat-rich diet in trial II. Each participant took part in either trial I or trial II but not in both. Thirty-two participants were recruited into trial I and 49 into trial II. The age range of participants was 20–41 y. A randomised crossover design was used in each trial. In trial I the participants were randomly assigned to start eating the saturated fat diet or the n-6 polyunsaturated fat diet. After $2\frac{1}{2}$ weeks of the initial diet they crossed over, without washout, to the alternate diet for $2\frac{1}{2}$ weeks. The same crossover design was used for trial II except that the saturated fat diet was compared with a diet that included monounsaturated-rich fats and oils (canola). The period of $2\frac{1}{2}$ weeks was chosen as it has previously been shown that plasma lipid levels stabilise within 2–3 weeks after initiating a change in dietary fat intake (Bonanome & Grundy, 1988; Mensink & Katan, 1987; O'Dea *et al*, 1990).

The total fat content of the high saturated fat diet used during trials I and II was designed to contribute 30–33% of energy with saturated fat content accounting for 15% of energy. The latter is typical of the New Zealand diet (Russell *et al*, 1999). Participants were instructed to self-select a background diet low in fat that would be consumed throughout the 5 week study period. Typical foods for the background diet included lean meat and poultry, low-fat fish, rice, pasta, legumes, beans, fruit and vegetables, cereals and breads. On all diets the fat was to be trimmed from the meat and the skin removed from the chicken. Deep-fried foods or foods cooked in or prepared with fats of unknown origin were to be avoided. To this background diet participants were asked to select foods containing the type of fats dictated by the diet they were following. During the saturated fat diet volunteers were asked to use butter as a spreading fat and in baking and cooking, and to consume high-fat dairy foods and avoid eating vegetable oils and fats and foods containing these fats. Dairy products were chosen as the main source of saturated fat because a high proportion of fat in the typical New Zealand diet comes from these foods (Russell *et al*, 1999). While on the n-6 polyunsaturated fat (trial I) and monounsaturated fat (trial II) diets participants were asked to avoid using butter, to use low-fat dairy foods and to minimise intake of foods high in saturated fat. For convenience, on the n-6 polyunsaturated fat diet participants were provided with safflower oil and sunflower-based spread to replace butter as a spreading fat and in baking and cooking. In trial II participants were provided with a canola spread and oil

and were asked to refrain from eating nuts and other foods rich in n-6 polyunsaturated fats. In both trials I and II participants were not provided with any additional foods other than the specific spreads and oils. Participants were provided with a small book of suggested recipes and general instructions which addressed the issues of eating to satiety in order to maintain a steady weight, and not making drastic changes in meal frequency. Other than these general guidelines the diets were not prescribed and participants self-selected and prepared all foods. While following each diet the participants completed a 3 day diet record. All foods and beverages consumed on two representative weekdays and one weekend day were weighed or estimated and recorded into food diaries. The recorded information was entered onto a computer and the nutrient composition of the diets calculated using 'Diet Entry and Storage' and 'Diet Cruncher' (Marshall, 1996), which uses food composition data from the New Zealand Institute for Crop and Food Research Ltd (Burlingham *et al*, 1993). *Trans* fatty acids in the canola spread were entered into the food composition database as saturated fatty acids.

Participants were weighed in light clothing at baseline and then on the last day of the respective $2\frac{1}{2}$ week diet period. Fasting blood samples were taken before initiating the respective dietary phases (ie baseline) and then on the last day of each $2\frac{1}{2}$ week diet period. After an overnight fast of at least 12 h, venous blood was collected into vacutainers containing disodium EDTA. Plasma was separated by centrifugation at 2000 g for 10 min, and aliquots were stored in plastic tubes at $-20^{\circ}$C. Cholesterol and triglyceride measurements were completed within 5 weeks of blood collection.

Cholesterol and triglyceride concentrations in plasma were measured by enzymatic kits from Boehringer-Mannheim and Roche Diagnostics on a Cobas Fara Analyser (Roche Diagnostics). High density lipoprotein cholesterol was measured in the supernatant following precipitation of apoB-containing lipoproteins with phosphotungstate-magnesium (Assmann *et al*, 1983). Low density lipoprotein cholesterol was calculated using the Friedewald formula (Friedewald *et al*, 1972). All assays were validated according to the Royal Australasian College of Pathologists Quality Assurance Program. The coefficient of variation (CV) for total cholesterol, high density lipoprotein cholesterol, calculated low density lipoprotein cholesterol and plasma triglycerides was 1.6, 1.7, 2.1 and 2.2%, respectively.

Plasma lipids were extracted according to the method of Bligh and Dyer (1959). An internal triglyceride standard was added before extraction so that moles of fatty acids could be measured. Triglycerides were separated by spotting lipid extracts onto silica gel 60 (Merck) thin-layer chromatography plates and running in a solvent system of hexane:diethyl ether:acetic acid (85:15:1). Lipid bands were visualised under UV light after spraying with 0.1% ANS (8-anilino-1-naphthalene sulphonic acid), and identification verified by using commercial standards. Triglyceride bands were scraped into glass tubes and methylated at 80°C with 6% $H_2SO_4$ in methanol for 2 h, then eluted into hexane and stored at $-20^{\circ}$C. Separation and quantitation of the fatty acid methyl esters from triglycerides was achieved using a DB-225 megabore column (25 m × 0.53 mm internal diameter; film thickness 0.25 μm; J & W Scientific) installed on an HP-6890 Series Gas Chromatograph with flame ionisation detection. Blanks were extracted, analysed and peak areas subtracted from corresponding areas in the sample runs. Precision of the fatty acid analysis was established by extracting 20 pooled plasma samples — approximately one sample of pooled plasma for every 10 experimental samples. The CV (%) for the fatty acids 16:0, 18:0, 18:1, 18:2 and 18:3n-3 were 2.1, 6.1, 1.0, 6.4 and 6.3, respectively.

All participants completed the study and provided blood samples in which plasma cholesterol was measured. However, a complete set of results including energy and nutrient intake as well as fatty acid composition of plasma triglycerides was available for 29 out of 32 participants in trial I and 42 out of 49 participants in trial II. Analysing the data from all participants or excluding those for whom complete data was not available made no appreciable difference to the final results. For the sake of simplicity the results are reported for participants having a complete set of results. Differences between measurements made on the two experimental diets within each phase were compared by repeated measures analysis of variance. A Wilcoxon signed rank test was used to assess differences in alcohol intake. As part of the analysis the effect of diet sequence was tested. Results were considered statistically significant when *P*-values were less than 0.05. The average difference (95% CI) between the change in plasma cholesterol in trial 1 minus the change in trial II was calculated using linear regression after adjusting for sex.

## Results

Participants in trial I were of similar age, weight and body mass index (BMI) to those in trial II (Table 1). However, there was a marginally lower proportion of females in trial I (77%) than in trial II (84%). Within trials I and II weight was unchanged by the respective dietary interventions.

**Table 1** Characteristics of participants

| | *Trial I* | | *Trial II* | |
|---|---|---|---|---|
| | *SAFA (n = 29)* | *n-6 PUFA (n = 29)* | *SAFA (n = 42)* | *MUFA (n = 42)* |
| Age (y) | 22.2 (2.9) | | 23.0 (4.2) | |
| Female/male | 22/7 | | 35/7 | |
| Weight (kg)[a] | 65.7 (8.2) | 65.5 (8.2) | 67.4 (11.3) | 67.2 (11.1) |
| BMI (kg/m$^2$) | 23.0 (2.2) | 22.9 (2.2) | 23.7 (3.2) | 23.7 (3.1) |

Values reported as means (s.d.).
[a]Weight measured on the last day of each diet.
SAFA, saturated fatty acids; MUFA, monounsaturated fatty acids; n-6 PUFA, polyunsaturated fatty acids; BMI, body mass index.

**Table 2** Fatty acid composition of spreads and oils (g/100 g fatty acids)

| | *n-6 PUFA* | | *MUFA* | |
|---|---|---|---|---|
| *Fatty acid* | *Safflower oil* [a] | *Sunflower spread* [a] | *Canola spread* [b] | *Canola oil* [b] |
| C16:0 | 9.1 | 11.0 | 7.9 | 5.0 |
| C18:0 | 2.3 | 5.9 | 4.3 | 1.8 |
| C18:1 *trans* [c] | — | < 1 | 8.6 | — |
| C18:1 | 19.9 | 37.8 | 54.4 | 58.2 |
| C18:2n-6 | 66.3 | 42.8 | 15.0 | 20.8 |
| C18:3n-3 | 1.2 | 1.0 | 5.3 | 10.1 |

Fatty acids less than 1% of total are not reported.
[a]New Zealand Food Composition Database.
[b]Manufacturer's analysis.
[c]*Trans* fatty acids included as saturated fatty acids in food composition database when calculating nutrient composition of diets.

The fatty acid composition of the spreads and oils that were provided to the participants during each phase of the study are presented in Table 2.

During trial I participants reportedly ate 29 g/day less saturated fat when following the n-6 polyunsaturated fat diet than when on the saturated fat diet; as a percentage of energy this equated to a difference of 9% energy. Participants in trial II achieved a similar difference in saturated fat intake of 28 g/day (or 9.3% energy) between the monounsaturated fat and saturated fat diet (Table 3). In both trials the bulk (85–90%) of the difference in saturated fat intake was accounted for by a change in dairy fat consumption.

The substantial decrease in dairy fat intake during both the n-6 polyunsaturated (by 41 g) and monounsaturated (by 43 g) diets was not entirely replaced with fat from the plant oils and spreads provided. Margarine and oil provided 28 g of total fat during the n-6 polyunsaturated diet and 29 g during the monounsaturated diet. Furthermore, energy and fat intake from fast food and confectionery products were lower on the n-6 polyunsaturated and monounsaturated fat diets. Alcohol consumption decreased by 2% energy when going from a saturated fat to a n-6 polyunsaturated fat diet. Consequently, total energy intake was lower by 1.6 MJ (358 kcal) on the n-6 polyunsaturated fat diet and 1.7 MJ (406 kcal) on the monounsaturated fat diet.

Plasma triglyceride fatty acids were analysed as an objective and alternate measurement of dietary compliance (Table 4). In general plasma triglyceride fatty acids reflected the changes in fat intake. The 5.6 mol% increase in linoleic acid content of plasma triglyceride when participants were on the n-6 polyunsaturated fat diet was significantly greater than the 2.9 mol% increase in linoleic acid when participants were on the monounsaturated fat diet. The magnitude of the changes in saturated fatty acids in plasma triglycerides was similar in trials I and II. Oleic acid increased on the monounsaturated fat diet but did not on the n-6 polyunsaturated fat diet. Use of canola-based spread and canola oil during the monounsaturated fat diet led to small increases ($P < 0.001$) in the levels of n-3 polyunsaturated fatty acids (C18:3n-3, C20:5n-3 and C22:6n-3) in plasma triglycerides.

In comparison to the corresponding saturated fat diet, plasma total cholesterol concentrations decreased by 19% ($P < 0.001$) on the n-6 polyunsaturated fat diet and by 12% ($P < 0.001$) on the monounsaturated fat diet (Table 5). The decrease was significantly greater on the n-6 polyunsaturated fat diet.

Plasma low density lipoprotein cholesterol decreased by 22% ($P < 0.001$) on the n-6 polyunsaturated fat diet and by 15% ($P < 0.001$) on the monounsaturated fat diet. The

**Table 3** Energy and nutrient composition of diets

| | *Trial I* | | *Trial II* | | |
|---|---|---|---|---|---|
| *Nutrient* | *SAFA (n = 29)* | *n-6 PUFA (n = 29)* | *SAFA (n = 42)* | *MUFA (n = 42)* | *Trial I change minus trial II change mean (95% CI)* |
| Energy (MJ) | 10.4 (3.8) | 8.8 (3.3)[a] | 9.9 (3.7) | 8.2 (2.2)[c] | −0.2 (−1.7, 1.2) |
| Protein (%kJ) | 14.6 (3.1) | 15.3 (4.0) | 15.3 (4.5) | 16.0 (3.2) | 0.0 (−2.1, 2.0) |
| Carbohydrate (%kJ) | 46.7 (7.8) | 51.2 (7.8)[b] | 48.7 (7.9) | 53.8 (7.3)[c] | 0.6 (−3.6, 4.8) |
| Total fat (%kJ) | 33.3 (7.3) | 30.4 (7.1) | 34.0 (5.8) | 28.9 (7.0)[c] | −2.2 (−6.4, 2.0) |
| SAFA (%kJ) | 17.5 (4.2) | 8.5 (2.8)[c] | 17.7 (4.2) | 8.4 (2.8)[c] | −0.3 (−2.7, 2.2) |
| MUFA (%kJ) | 9.6 (2.9) | 9.5 (2.7) | 9.7 (2.2) | 11.6 (3.4)[b] | 2.0 (0.3, 3.7) |
| PUFA (%kJ) | 2.7 (0.9) | 9.1 (4.2)[c] | 3.0 (1.2) | 6.1 (2.1)[c] | −3.4 (−5.0, −1.8) |
| P/S ratio | 0.2 (0.1) | 1.2 (0.8)[c] | 0.2 (0.1) | 0.8 (0.4)[c] | −0.5 (−0.7, −0.2) |
| P + M/S ratio | 0.7 (0.2) | 2.4 (1.0)[c] | 0.8 (0.3) | 2.3 (0.8)[c] | −0.2 (−0.6, 0.3) |
| Cholesterol (mg) | 303 (128) | 174 (94)[c] | 305 (189) | 168 (129)[c] | −8 (−73, 58) |
| Butter (g) | 22 (15) | 0 (0)[c] | 25 (17) | 0 (2)[c] | −3 (−11, 5) |
| Margarine (g) | 0 (0) | 27 (20)[c] | 4 (8) | 23 (17)[c] | −7 (−17, 2) |
| Oil (g) | 0 (0) | 8 (11)[c] | 0 (1) | 12 (15)[c] | 4 (−3, 10) |
| Fibre (g) | 30 (35) | 31 (26) | 26 (12) | 26 (9) | −1 (−14, 12) |
| Alcohol (%kJ) | 4.0 (6.8) | 1.7 (3.0) | 2.3 (4.2) | 1.9 (4.6) | 1.9 (−0.8, 5.1) |

Values reported as means (s.d.).
[a]$P < 0.05$; [b]$P < 0.01$; [c]$P < 0.001$ between diets within trials.
P/S ratio, polyunsaturated fat intake to saturated fat intake (by weight); P + M/S ratio, polyunsaturated and monounsaturated fat intake to saturated fat intake (by weight).

**Table 4** Fatty acid composition of plasma triglyceride (mol%)

| | *Trial I* | | *Trial II* | | |
|---|---|---|---|---|---|
| *Fatty acid* | *SAFA (n = 29)* | *n-6 PUFA (n = 29)* | *SAFA (n = 42)* | *MUFA (n = 42)* | *Trial I change minus trial II change mean (95% CI)* |
| C14:0 | 3.5 (1.7) | 2.7 (0.9)[b] | 3.3 (1.4) | 2.6 (1.2)[b] | 0.1 (−0.6, 0.9) |
| C14:1 | 0.4 (0.3) | 0.3 (0.2)[a] | 0.5 (0.3) | 0.3 (0.2)[b] | 0.0 (−0.2, 0.1) |
| C15:0 | 0.5 (0.2) | 0.4 (0.1)[b] | 0.4 (0.1) | 0.3 (0.1)[c] | 0.0 (−0.1, 0.1) |
| C16:0 | 30.4 (5.4) | 27.3 (3.7)[b] | 29.7 (4.5) | 26.0 (5.1)[c] | −0.6 (−3.0, 1.8) |
| C16:1 | 6.7 (2.1) | 6.6 (1.7) | 7.7 (1.7) | 7.2 (1.8) | −0.5 (−1.2, 0.3) |
| C18:0 | 3.7 (1.3) | 3.1 (0.8)[a] | 3.5 (1.1) | 2.8 (0.8)[c] | −0.1 (−0.8, 0.5) |
| C18:1n-9 | 36.6 (7.8) | 35.8 (4.3) | 37.3 (4.3) | 39.5 (4.1)[b] | 3.1 (0.3, 5.9) |
| C18:2n-6 | 11.9 (4.0) | 17.5 (6.0)[c] | 11.5 (3.9) | 14.4 (4.9)[c] | −2.6 (−4.8, −0.4) |
| C18:3n-6 | 0.3 (0.1) | 0.4 (0.3)[a] | 0.4 (0.2) | 0.5 (0.2)[a] | −0.1 (−0.2, 0.0) |
| C18:3n-3 | 1.2 (0.4) | 1.1 (0.4) | 0.9 (0.3) | 1.3 (0.6)[c] | 0.5 (0.3, 0.8) |
| C20:4n-6 | 0.8 (0.2) | 0.9 (0.3)[b] | 0.7 (0.3) | 0.9 (0.3)[c] | 0.1 (−0.1, 0.2) |
| C20:5n-3 | 0.2 (0.1) | 0.2 (0.1) | 0.1 (0.1) | 0.2 (0.1)[c] | 0.1 (0.0, 0.1) |
| C22:6n-3 | 0.7 (0.4) | 0.7 (0.3) | 0.5 (0.3) | 0.7 (0.3)[c] | 0.3 (0.1, 0.4) |

Values reported as means (s.d.).
[a]$P < 0.05$; [b]$P < 0.01$; [c]$P < 0.001$ between diets within trials.

change in low density lipoprotein cholesterol was larger on the n-6 polyunsaturated fat diet by 0.21 mmol/l but the 95% confidence interval slightly overlapped with zero (−0.05, 0.48).

On the n-6 polyunsaturated fat diet the 14% ($P < 0.001$) decline in high density lipoprotein cholesterol concentration was significantly greater than the 4% ($P < 0.05$) drop on the monounsaturated fat diet.

The reduction in the total to high density lipoprotein cholesterol ratio was similar on the n-6 polyunsaturated fat diet (6%, $P < 0.05$) to that on the monounsaturated fat diet (8%, $P < 0.001$).

Plasma triglyceride concentration decreased by 15% on both the n-6 polyunsaturated fat diet and the monounsaturated fat diet but did not reach statistical significance on the former because of the smaller sample size in trial I.

## Discussion

In the present study the replacement of saturated fat rich foods with foods rich in either n-6 polyunsaturated or monounsaturated fat lowered plasma total cholesterol by 19 and 12%, respectively, in young healthy adults eating self-selected diets. The decrease in total cholesterol and high density lipoprotein cholesterol was significantly larger when n-6 polyunsaturated rather than monounsaturated fat was substituted for saturated fat. The differences in energy and cholesterol intake, percentage energy from saturated fat, and percentage energy from carbohydrate between the two diets in trial I (saturated fat minus n-6 polyunsaturated fat diet) were virtually the same as the differences between the two diets in trial II (saturated fat minus monounsaturated fat). Thus, when comparing the changes in plasma cholesterol between trials I and II the differences must be attributable largely to the distinct effects of n-6 polyunsaturated or monounsaturated fat when replacing saturated fat in the diet.

In summarising the results of diet and cholesterol trials conducted in metabolic wards or using highly controlled diets, Clarke *et al*, (1997) and Mensink and Katan (1992) have reported that n-6 polyunsaturated fatty acids lower total and low density lipoprotein cholesterol more than monounsaturated fatty acids when isocalorically substituted

**Table 5** Plasma cholesterol and triglyceride concentrations

| | *Trial I* | | *Trial II* | | |
|---|---|---|---|---|---|
| *Lipid* | *SAFA (n = 29)* | *n-6 PUFA (n = 29)* | *SAFA (n = 42)* | *MUFA (n = 42)* | *Trial I change minus trial II change mean (95% CI)* |
| Tot-chol (mmol/l) | 4.87 (0.88) | 3.94 (0.92)[c] | 4.98 (0.78) | 4.40 (0.75)[c] | 0.35 (0.01, 0.68) |
| LDL-chol (mmol/l) | 2.87 (0.75) | 2.24 (0.67)[c] | 2.83 (0.65) | 2.41 (0.65)[c] | 0.21 (−0.05, 0.48) |
| HDL-chol (mmol/l) | 1.39 (0.36) | 1.19 (0.34)[c] | 1.47 (0.38) | 1.41 (0.35)[a] | 0.14 (0.03, 0.25) |
| Tot:HDL ratio (mmol/l) | 3.67 (0.96) | 3.46 (0.87)[a] | 3.56 (0.95) | 3.26 (0.87)[c] | −0.08 (−0.33, 0.17) |
| TG (mmol/l) | 1.31 (0.66) | 1.11 (0.49) | 1.47 (0.61) | 1.25 (0.63)[c] | −0.02 (−0.24, 0.20) |

Values reported as means (s.d.).
[a]$P < 0.05$; [b]$P < 0.01$; [c]$P < 0.001$ between diets within trials.
Tot-chol, plasma total cholesterol; LDL-chol, low density lipoprotein cholesterol; HDL-chol, high density lipoprotein cholesterol; Tot: HDL-chol, total to high density lipoprotein cholesterol ratio; TG, total triglyceride.

for saturated fatty acids in the diet. The results reported herein indicate that this differential effect remains even when the replacement of saturated fat is not isocaloric.

This is in contrast to two previous studies investigating the effect on plasma cholesterol of replacing saturated fat with either polyunsaturated fat or monounsaturated fat while at the same time reducing total fat intake. Both types of fat were equally effective at lowering plasma cholesterol when replacing saturated fat (Lichtenstein *et al*, 1993; Wahrburg *et al*, 1992). In both of these studies highly controlled diets were used and participants were provided with all foods and beverages. Lichtenstein *et al*, (1993) compared a diet high in total and saturated fat (35 and 13% total energy) to diets lower in total and saturated fat (30 and 7% total energy) and enriched with either polyunsaturated (11% total energy) or monounsaturated (15% total energy) fat. On the polyunsaturated and monounsaturated fat rich diets the reductions in total and low density lipoprotein cholesterol of 12 and 17%, respectively, were identical. Wahrburg *et al* (1992) compared a very high total and saturated fat (41 and 20% total energy) diet against a lower total fat (33% total energy) diet rich in either monounsaturated (16% total energy) or polyunsaturated (10% total energy) fat. On the lower fat diet enriched with either monounsaturated fatty acids or polyunsaturated fatty acids the decrease in total and low density lipoprotein cholesterol was the same, 13 and 14%, respectively. The reasons for the discrepancy between the results of the present study and those of Lichtenstein *et al* (1993) and Wahrburg *et al* (1992) are not readily apparent. Although in the study by Lichtenstein *et al* (1993) the male and female participants, were older (60 y) and had higher initial plasma total cholesterol (6.3 mmol/l), the characteristics of those in the study by Wahrburg *et al*, (1992) were very similar to the present study. Lack of compliance with the diets in the present study is not an explanation because the close agreement between plasma triglyceride fatty acids and the reported fat composition of the diets is objective evidence, in so far as fat intake is concerned, that participants actually ate what they said they did.

Participants—all nutrition students—had good knowledge of the fat content of foods and were asked specifically to keep total fat intake the same; nevertheless, they still found it difficult to replace the dairy fat consumed during the saturated fat phase with an equivalent amount of plant fats during either the n-6 polyunsaturated (trial I) or monounsaturated (trial II) phases. An explanation for this inability to isocalorically substitute fat probably lies in the fact that the population from which the participants were drawn are accustomed to and enjoy eating high-fat dairy foods such as butter, cheese, cream, ice cream and whole milk. These foods are readily available, convenient and require little preparation whereas the use of plant fats and oils in the diet, other than as a spread or for simple cooking, requires considerable change in food choice and preparation practices. Thus, the general public, who are likely to have less nutrition knowledge about the fat content of foods, will probably reduce their total fat intake when trying to replace saturated fat in their diet with n-6 polyunsaturated and monounsaturated fat rich foods.

Reported energy intakes decreased by 1.6 and 1.7 MJ/day when changing from the saturated fat rich diet to either the n-6 polyunsaturated (trial I) or monounsaturated fat (trial II) diets, respectively, yet body weights remained unchanged. The predicted weight change for a 1.7 MJ/day decrease in energy intake would be approximately 350 g/week or 875 g in 2.5 week (American Dietetic Association, 1981). Leibel *et al* (1995) reported that when the body is faced with an energy deficit or excess it initially compensates to resist weight change from its usual weight by altering total energy expenditure. It is possible that this phenomenon explains the lack of weight change in each trial. Alternatively, participants could have changed their exercise habits to compensate for the changes in energy intakes. Systematic under-reporting of energy intake during the n-6 polyunsaturated fat and monounsaturated fat diets and over-reporting during the saturated fat diets seems unlikely.

The present study indicates that even in young adults with little evidence of hypercholesterolaemia plasma cholesterol concentrations are highly responsive to changes in dietary fat within the range of normal intakes. The decrease in total cholesterol of 0.93 mmol/l when participants increased n-6 polyunsaturated fat and decreased total and saturated fat intakes was equal to the 0.95 mmol/l change predicted by Keys *et al* (1965) but larger than the 0.74 mmol/l change predicted by Hegsted *et al* (1993). When participants decreased total and saturated fat and increased monounsaturated fat intakes, total cholesterol decreased by 0.60 mmol/l, which is similar to the 0.67 mmol/l decrease predicted by Hegsted *et al* (1993) but slightly lower than the 0.83 mmol/l change predicted by Keys *et al* (1965).

In 1985 Mattson and Grundy, using a series of liquid formula diets, demonstrated that a high polyunsaturated fat diet in comparison to a high saturated fat diet decreased plasma high density lipoprotein cholesterol concentrations. More recently studies using whole foods have demonstrated similar effects of polyunsaturated-rich diets (Mensink & Katan, 1989; Wardlaw & Snook, 1990; Howard *et al* 1995; Noakes & Clifton, 1998; Wahrburg *et al* 1992; Lichtenstein *et al* 1993). There is increasing evidence of the importance of plasma high density lipoprotein cholesterol as a risk factor that is inversely and independently associated with cardiovascular disease (Castelli *et al* 1986; Jacobs *et al* 1990; Miller, 1975). The present study shows that if total and saturated fat intakes are reduced and there is an appreciable increase in n-6 polyunsaturated fat (6% of total energy), plasma high density lipoprotein cholesterol is lowered. In contrast, if monounsaturated fat intake is increased and total and saturated fat intakes decreased the reduction in high density lipoprotein cholesterol is minimised.

There is good evidence that the ratio of total to high density lipoprotein cholesterol is a better predictor of cardiovascular disease risk than either total cholesterol or

low density lipoprotein cholesterol alone (Castelli *et al* 1983; Kinosian *et al* 1994). The results of the present study show that the ratio of total to high density lipoprotein cholesterol is lowered equally when either n-6 polyunsaturated or monounsaturated fat replace saturated fat in the diet. However, if only using total cholesterol to predict risk of cardiovascular disease the present results show that replacing saturated fat with n-6 polyunsaturated fat, rather than monounsaturated fat, is more efficacious at lowering the predicted risk of cardiovascular disease due to a larger decline in plasma total cholesterol levels.

It is universally accepted that elevated plasma cholesterol is a major risk factor for cardiovascular disease (Mann *et al* 1993; NCEP, 1993; Rosa *et al* 1990). Although most of the conclusive evidence for this comes from research on middle-aged and older men and women, several studies confirm that high plasma cholesterol in young adults is a predictor of cardiovascular disease risk in later life (Klag *et al* 1993; Myers *et al* 1995). It is important that the dietary strategies to lower plasma cholesterol, largely elucidated through research on older adults, are found to be effective in young adults. In this regard, the present study makes an important contribution in demonstrating that young adults are very responsive to dietary-induced changes in plasma cholesterol. In the context of a free-living situation reducing blood cholesterol by replacing saturated fat with n-6 polyunsaturated fat or monounsaturated fat may result in a diet lower in total fat. In this context n-6 polyunsaturated or monounsaturated fat appears to be equally efficacious at lowering the total to high density lipoprotein cholesterol ratio.

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