The Serum LDL/HDL Cholesterol Ratio Is Influenced More Favorably by Exchanging Saturated with Unsaturated Fat Than by Reducing Saturated Fat in the Diet of Women

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ABSTRACT We compared the effects of a high fat diet [38.4% of energy (E%) from fat; HSAFA diet, polyunsaturated/saturated fatty acid (P/S) ratio = 0.14], a low fat diet (19.7 E% from fat; LSAFA diet, P/S = 0.17), both based on coconut oil, and a diet with a high content of mono- and polyunsaturated fatty acids (PUFA; 38.2 E% from fat; HUFA diet, P/S = 1.9) on serum lipoproteins. The 25 women studied consumed each diet for 3-wk periods in a crossover design. The two high fat diets were identical except for the quality of the test fat. The LSAFA diet was identical to the HSAFA diet except that half the fat was replaced by carbohydrates. Serum total cholesterol, LDL cholesterol and apoB concentrations did not differ between the HSAFA and the LSAFA diet periods. Total cholesterol, LDL cholesterol and apoB were lower when women consumed the HUFA diet than when they consumed the other two diets. HDL cholesterol and apoA-I were 15 and 11%, respectively, higher when women consumed the HSAFA diet than when they consumed the LSAFA diet; HDL cholesterol and apoA-I were lower when women consumed the HUFA diet than when they consumed the HSAFA diet, but not the LSAFA diet. The LDL cholesterol/HDL cholesterol and apoB/apoA-I ratios were higher when women consumed the LSAFA diet than when they consumed the HUFA diet. Triacylglycerol and VLDL cholesterol were higher when women consumed the LSAFA diet than when they consumed the HUFA diet, whereas apoB/apoA-I was higher when women consumed the LSAFA diet than when they consumed the HUFA diet. Triacylglycerol and VLDL cholesterol were higher when women consumed the HSAFA diet than when they consumed the HUFA diet or the HUFA diet. We conclude that, to influence the LDL/HDL cholesterol ratio, changing the proportions of dietary fatty acids may be more important than restricting the percentage of total or saturated fat energy, at least when derived mainly from lauric and myristic acids, both of which increase HDL cholesterol. J. Nutr. 133: 78–83, 2003.

KEY WORDS: • apolipoprotein A-I • apolipoprotein B • coconut oil • lauric acid • myristic acid

Epidemiological (1,2) and experimental data (3,4) indicate that a diet high in saturated fatty acids is associated with high levels of serum total cholesterol, which in turn, are related to a high incidence of coronary heart disease. In the Seven Countries study, Keys et al. (1) found an association between the percentage of total energy from saturated fatty acids and serum cholesterol. They also reported that the intake of saturated fatty acids, as a percentage of energy, was strongly correlated with coronary death rates (1). Keys and Kimura (5) compared the connection of plasma cholesterol with total fat and saturated fatty acid intake among three populations, Crete, Tanushimaru and Zutphen. Their results indicated that the level of saturated fatty acids in the diet rather than the amount of total fat was the main factor that explained the lower plasma cholesterol levels in the population in Crete compared with Zutphen. Population studies show an association between low fat diets and low incidence of coronary heart disease (6,7), although the desirability of low fat diets and the effects of such diets on the proportion of serum cholesterol in LDL and HDL fractions are not well established (8,9). There is also no agreement as to the desirable intake of monounsaturated fatty acids relative to carbohydrate (8). Low fat, high carbohydrate diets decrease HDL cholesterol and increase triacylglycerol (9,10) and are less effective than diets high in polyunsaturated fatty acids to decrease serum total cholesterol (3,4). Hu et al. (2) found an inverse association between the dietary intake of polyunsaturated fatty acids (PUFA)3 and the incidence of coronary disease. This is in line with several controlled studies demonstrating the cholesterol-decreasing effect of PUFA when exchanged for saturated fatty acids in the diet (11–13).

Hegsted et al. (14,15) suggested that the proportions of

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fatty acids in the dietary fat rather than the percentage of energy they supply is of primary importance for the level of serum cholesterol. Studies on the effects of high fat vs. lowfat diets on serum lipoprotein levels have varied widely in fatty acid composition; that is, the polyunsaturated/saturated fatty acid (P/S) ratio has varied (12,16,17). In such studies it is not easy to evaluate whether the effect on serum lipids is caused by change in total quantity of saturated fat and/or fatty acid composition.

In this study we compared the effects on serum lipid levels of quantity of dietary fat vs. fatty acid composition in a controlled study. The aim was to compare the effects on serum lipoproteins and in particular, on the LDL/HDL cholesterol ratio, of a high fat and a low fat coconut oil–based diet with identical P/S ratios. A high fat diet with a high content of mono- and polyunsaturated fatty acids, but otherwise identical to the high fat coconut oil diet, was also included for comparison.

MATERIALS AND METHODS

Participants and their baseline characteristics. Thirty-one female students in home economics, all in good health, were invited to participate in this strictly controlled dietary study. Of these, 25 completed the study. Their age (mean ± SD) was 30.5 ± 9.8 years, and except for oral contraceptives, none was taking any medication known to affect serum lipids. We had no screening criteria about smoking habits, age or physical activity. Those with body mass index (BMI) ≥ 32 kg/m² were excluded. The weight of the participants was 67.4 ± 12.1 kg and their BMI was 24.5 ± 3.2 kg/m². Seven women were taking oral contraceptives and nine were smokers. All participants were requested to maintain their regular lifestyles and usual level of physical activity throughout the study. They were asked to abstain from alcohol consumption and to report in a diary any deviation from their usual behavior. The protocol and the objective of the study were explained in detail to the participants and they gave their written consent before entry into the study. They received free food but no payment and were treated according to the principles of the Helsinki declaration. The study protocol was approved by the Regional Committee for Ethics in Biomedical Research of Norway.

Habitual diet. The participants filled in a validated quantitative food frequency questionnaire, meant to give the usual food intake during the past year (18) and each questionnaire was thoroughly checked. The calculated amounts of total fat, protein and carbohydrate in the habitual diet (mean ± SD) were 30 ± 5.9, 15 ± 2.0 and 53.9 ± 5.1 percent of energy (E%) (3), respectively. The mean daily intake of saturated, monounsaturated and PUFA was 11, 11 and 6 E%, respectively. The calculated mean intake of dietary cholesterol was 271 mg/day.

Study design. The study, from September to December 1998, was divided into periods of 22 d for the first and second periods and 20 d for the third period. The study was designed as an intraindividual crossover comparison of the effects on serum lipids of three diets. The participants were randomized in three groups and each group received the three diets in a sequence determined by a Latin-square design. In this way, variation attributed to residual effects of the previous diet or to drift of variables over time could be minimized. After the end of each test period, the participants began the next diet with a washout period of 1 wk, during which they returned to their normal eating habits. Body weight was monitored before lunch twice a week with light clothes on a digital balance, and read to the nearest 0.1 kg. Body height was measured without shoes and read to the nearest 0.1 cm. BMI was calculated as weight (kg)/height (m)².

Test margarines and experimental test diets. Two different test margarines were used in the study. A saturated fatty acid–rich margarine (SAFA margarine) that contained 80% coconut oil, 10% soybean oil and 10% rapeseed oil was used in two of the diets. One of these was designed to contain 22 E% fat; low saturated fatty acid diet (LSAFA diet) and the other, 42 E% fat; high saturated fatty acid diet (HSASA diet). A commercial, soft highly unsaturated margarine (HUFA margarine), consisting of coconut oil, palm oil, refined sunflower oil and refined rapeseed oil, was used in the third diet designed to contain 42 E% fat; high mono- and polyunsaturated fatty acid diet (HUFA diet). The fatty acid composition of the diets is presented below in Results. The margarines were produced by the addition of water, vitamin D₃, N-acetylcarcinic acid and β-carotene (color) and emulsifier. The SAFA margarine contained 16.3% water and the HUFA margarine, 16.1% water. Total tocopherol was 16.4 mg/100 g in SAFA margarine and 35.5 mg/100 g in the HUFA margarine.

The diets were based on a 7-d menu. They were prepared using a computer-based, nutrient-calculation program and were designed to have the same nutrient composition except for the fatty acid composition and fat content. The fat from the background diets was calculated to supply a minimal amount of 7.8 E% fat, whereas the test fat was planned to provide 34.2 E% in the high fat diets and 14.2 E% in the low fat diet. The menu for the experimental diets contained the same basic food items. The HSASA and the HUFA diets were identical except for the test fat. In the LSAFA diet, about half the fat energy was replaced by carbohydrates from fruits, orange juice and sugar candies.

The fat from the background diet came from meat, fish, bread, dairy products and cereals. The test fats were incorporated into the menus in several foods including bread, buns, porridge and sauces for dinner. All foods, including weekend meals, were prepared at the college. Dinner was served under supervision in a dining room every day except during weekends. The evening meal and breakfast for the next day were taken home by the participants. Weekend meals were packaged for home consumption. All perishable foods were provided frozen. During the controlled feeding periods no foods other than those in the menu were allowed. If the participants temporarily increased activity or lost weight, they were allowed to eat buns with the same fat composition as the rest of the diet. The participants were allowed to drink coffee, tea and mineral water with artificial sweeteners. All foodstuffs were weighed for individual participants, who were supplied with food to meet 100% of their mean daily energy requirements. The HSASA and HUFA diets were calculated to contain 109 g fat/10 MJ, of which the test margarines provided 89 g, and the LSAFA diet calculated to contain 57 g fat/10 MJ of which the test margarine provided 37 g.

Compliance with the diets was judged by direct observation of consumption of weekday dinners, by close personal follow-up and by evaluation of food diaries.

Chemical analysis. Duplicate portions were taken of the three diets, corresponding to a daily energy intake of 8.2 MJ. The duplicate portions were kept frozen at −20°C. After homogenization the homogenates were freeze-dried and the homogenates from 7 d were pooled into one portion for each diet. These portions were analyzed for nitrogen, total fat, metabolizable energy, cholesterol and fatty acid composition as previously described (19).

Blood sampling and analyses. Blood samples were taken after an overnight fast, before breakfast at the start of the first period and at the end of each period. Serum was obtained by low speed centrifugation within 1 h of venipuncture and stored at −70°C until analyzed.

Serum cholesterol and serum triacylglycerol were measured by enzymatic methods (20,21) using Cobas® Integra enzyme kits and an automated analyzer (Cobas Integra 700; Hoffman-La Roche, Basel, Switzerland). LDL cholesterol was calculated using the Friedewald equation (22). Serum HDL cholesterol was measured by a similar enzymatic technique (22) after complexing the LDL, VLDL and chylomicron fractions by polyanions (Cobas Integra HDL cholesterol Direct) and using equipment as above. Serum apolipoprotein A-I (apoA-I) and apolipoprotein B (apoB) were quantified immunoturbidimetrically using the Cobas Integra Apolipoprotein A-I and Apolipoprotein B kits and an automated enzyme analyzer (Cobas Integra 700) essentially according to the manufacturer's instructions. The CV were: total cholesterol, 2%; HDL cholesterol, 5%; triacylglycerol, 3%; apoA-I, 6.3%; apoB, 5.5%.

All lipid analyses were performed at the Clinical Chemistry Department and Clinical Research Unit, Ullevaal University Hospital, Oslo.

Statistical methods. Data were analyzed by repeated-measures ANOVA for a crossover trial (general linear models). When the
analysis indicated a significant effect of diet ($P < 0.05$), the Bonferroni method was used for a pairwise comparison between the three diet groups and for calculation of $95\%$ confidence limits for the differences between the diets. Values of $P / H 0.05$ were considered significant. The Bonferroni method encompasses a downward adjustment of significance limits for the differences between the diets. All $P$-values were two-tailed. Values in the text are means $\pm$ SD.

**RESULTS**

Five persons left the study after the first period and one left after the second period; thus 25 of the 31 volunteers completed the study. Dietary compliance was very good and no significant deviations from the diets were noticed.

Body weights of fasting subjects at the end of the first, second and third periods were $67.1 \pm 12.0$, $67.0 \pm 11.9$ and $67.1 \pm 12.0$ kg, respectively, and were not significantly different.

The energy contents of the diets were identical but slightly higher than planned (Table 1). The two high fat diets (HSAFA and HUFA diets) had the same proportions of fat, $38.4$ and $38.2$ E%, respectively, somewhat lower than the planned $42$ E%. The low fat diet (LSAFA diet) had about half the fat replaced by carbohydrates and contained $19.7$ E% as fat. The HSAFA diet contained $100.9$ g fat/10 MJ; the LSAFA diet, $51.7$ g fat; and the HUFA diet, $100.5$ g fat/10 MJ. The protein content was $15\%$ in the HSAFA diet and the HUFA diet and was slightly higher (i.e., $16.5\%$) in the LSAFA diet. All three diets were low in cholesterol (Table 1).

The HSAFA and the LSAFA diets had the same relative fatty acid compositions, with almost the same P/S ratio, $0.14$ and $0.17$, respectively (Table 2). The energy contents of the cholesterol-increasing fatty acids, $12:0$, $14:0$ and $16:0$, however, were about twice as high in the HSAFA as in the LSAFA diet.

The main differences in fatty acid composition between the HSAFA and the HUFA diets were the higher amounts of oleic ($18:1$) and linoleic acids ($18:2$) in the HUFA diet and the higher amount of saturated fatty acids in the HSAFA diet (Table 2).

Concentrations of total, LDL and HDL cholesterol, apoB, apoA-I and triacylglycerol at baseline and after completion of the three different test diets are shown in Table 3. Total cholesterol tended to be higher ($P = 0.09$) after consumption of the HSAFA than after the LSAFA. Total cholesterol, LDL cholesterol and apoB were significantly lower after consumption of the HUFA than after consumption of the two other diets. LDL cholesterol and apoB scarcely differed after intake of the HSAFA and LSAFA diets. HDL cholesterol and apoA-I

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**TABLE 1**

*Energy and nutrient composition of duplicate portions of the three test diets*¹

<table>
<thead>
<tr>
<th></th>
<th>HSAFA diet²</th>
<th>LSAFA diet³</th>
<th>HUFA diet⁴</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy, MJ</td>
<td>8.72</td>
<td>8.66</td>
<td>8.85</td>
</tr>
<tr>
<td>Protein, % of energy</td>
<td>14.9</td>
<td>16.5</td>
<td>15.0</td>
</tr>
<tr>
<td>Fat, % of energy</td>
<td>38.4</td>
<td>19.7</td>
<td>38.2</td>
</tr>
<tr>
<td>Carbohydrate, % of energy</td>
<td>46.7</td>
<td>63.8</td>
<td>46.8</td>
</tr>
<tr>
<td>Cholesterol, mg/d</td>
<td>51.1</td>
<td>49.3</td>
<td>56.9</td>
</tr>
</tbody>
</table>

¹ For fatty acid composition see Table 2.
² Diet high in saturated fatty acids.
³ Diet low in saturated fatty acids.
⁴ Diet high in poly- and monounsaturated fatty acids.

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**TABLE 2**

*Fatty acid composition of test margarines and corresponding diets*

<table>
<thead>
<tr>
<th>Fatty acid</th>
<th>SAFA margarine¹</th>
<th>HSAFA diet²</th>
<th>LSAFA diet³</th>
<th>HUFA margarine⁴</th>
<th>HUFA diet⁵</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mol/100 mol total fatty acids</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6:0</td>
<td>0.6</td>
<td>0.6</td>
<td>0.7</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>8:0</td>
<td>6.6</td>
<td>6.4</td>
<td>5.2</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>10:0</td>
<td>4.7</td>
<td>4.7</td>
<td>3.9</td>
<td>1.6</td>
<td>2.0</td>
</tr>
<tr>
<td>12:0</td>
<td>36</td>
<td>34.3</td>
<td>27.4</td>
<td>0.8</td>
<td>0.7</td>
</tr>
<tr>
<td>14:0</td>
<td>14.5</td>
<td>13.9</td>
<td>13.4</td>
<td>10.1</td>
<td>9.4</td>
</tr>
<tr>
<td>16:0</td>
<td>8.9</td>
<td>10.8</td>
<td>13.4</td>
<td>10.1</td>
<td>9.4</td>
</tr>
<tr>
<td>16:1c</td>
<td>&lt;0.1</td>
<td>0.25</td>
<td>0.7</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>18:0</td>
<td>2.9</td>
<td>3.6</td>
<td>4.9</td>
<td>8.4</td>
<td>8.0</td>
</tr>
<tr>
<td>18:1t</td>
<td>0.4</td>
<td>0.5</td>
<td>0.7</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>18:1c</td>
<td>13.8</td>
<td>14</td>
<td>17</td>
<td>36.5</td>
<td>36.7</td>
</tr>
<tr>
<td>18:2t</td>
<td>0.3</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>18:2c</td>
<td>9.1</td>
<td>8.6</td>
<td>10.2</td>
<td>36.1</td>
<td>36.2</td>
</tr>
<tr>
<td>18:3c</td>
<td>1.8</td>
<td>1.6</td>
<td>1.7</td>
<td>4.6</td>
<td>4.6</td>
</tr>
<tr>
<td>20:0</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>20:1t</td>
<td>0.1</td>
<td>0.2</td>
<td>0.2</td>
<td>0.7</td>
<td>0.6</td>
</tr>
<tr>
<td>20:1c</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
<td>0.3</td>
<td>0.2</td>
</tr>
<tr>
<td>22:0</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
<td>22.7</td>
<td>10.5</td>
</tr>
<tr>
<td>% of energy from 12:0, 14:0, 16:0</td>
<td>22.7</td>
<td>10.5</td>
<td>0.3</td>
<td>2.4</td>
<td>2.4</td>
</tr>
<tr>
<td>% of energy from cis MUFA</td>
<td>5.5</td>
<td>3.5</td>
<td>14.1</td>
<td>14.1</td>
<td>14.1</td>
</tr>
<tr>
<td>% of energy from cis PUFA</td>
<td>3.9</td>
<td>2.3</td>
<td>15.6</td>
<td>15.6</td>
<td>15.6</td>
</tr>
</tbody>
</table>

¹ A saturated fatty acid–rich margarine that contained $80\%$ coconut oil, $10\%$ soybean oil and $10\%$ rapeseed oil.
² Diet with a high content of SAFA margarine.
³ Diet with a low content of SAFA margarine.
⁴ A commercial highly unsaturated margarine consisting of coconut oil, palm oil, sunflower oil and rapeseed oil.
⁵ Diet with a high content of HUFA margarine.
were significantly higher after the HSAFA diet than after either the LSAFA diet or the HUFA diet. HDL cholesterol and apoA-I were not different after the HUFA and SAFA diets. The ratio of LDL cholesterol to HDL cholesterol was similar to the LDL/HDL cholesterol ratio, although it did not differ after the HSAFA and HUFA diet periods. Triacylglycerol and VLDL cholesterol were significantly higher after intake of the LSAFA diet than after the HSAFA and HUFA diets, although there was no significant difference between the effects of the HSAFA and HUFA diets (Table 3).

## DISCUSSION

The most important finding of this study was that lowering total saturated fat in the form of coconut oil, from 22.7 to 10.5 E% without change in the P/S ratio, did not lower total or LDL cholesterol, but significantly reduced HDL cholesterol. Thus, less favorable LDL/HDL cholesterol and apoB/apoA-I ratios occurred after intake of the LSAFA than after the HSAFA diet. After intake of the HUFA diet, on the other hand, there was a greater reduction in LDL cholesterol and lower LDL/HDL cholesterol and apoB/apoA-I ratios than after the other two diets. Our results show that replacing saturated fat from coconut oil by carbohydrates is less efficient than replacing by unsaturated fat if the aim is to improve the LDL/HDL cholesterol ratio, in accordance with results of other studies (23). In the literature there are conflicting reports as to the advantage of a low fat diet where saturated fatty acids are exchanged for carbohydrates compared to a higher fat diet where saturated fatty acids are replaced by unsaturated fatty acids, the argument of which is that the higher fat diet not only reduces LDL cholesterol but also has a more favorable influence on HDL cholesterol (10).

When we compared our results with those from other studies, where high fat and low fat diets were compared as to their effects on serum lipoproteins, we observed greater effects on LDL/HDL cholesterol and apoB/apoA-I ratios than in any other study. In the study of Nelson et al. (17), which was also similar to our study, where the effects of a low fat diet (22 E%) and a high fat diet (39 E%) both with P/S 1.0 were compared, no significant differences in plasma total or LDL cholesterol levels between the diets were observed. No difference was found in HDL cholesterol in that study, and thus no difference in the LDL/HDL cholesterol ratio (17). In the study of Barr et al. (24), 48 healthy men consumed an average American diet (AAD), with 37% of the energy from fat and 16% from saturated fatty acids for 3 wk. During the next 7 wk, one third of the participants continued to consume the AAD, one third switched to a 30% fat diet with 9% saturated fatty acids (Step 1 diet) and one third switched to a 30% fat diet with 14% saturated fatty acids (Sat diet). There was no significant difference in LDL and total cholesterol between those who consumed the AAD diet and those who consumed the Sat diet. Although LDL and HDL cholesterol were reduced on the Step 1 diet, no difference in LDL/HDL cholesterol or in apoB/apoA-I was observed (24). These findings support our observations that reducing total dietary fat without reducing the proportion of saturated fatty acids does not significantly lower LDL and total serum cholesterol concentrations in normal individuals. In another study comparing a high fat baseline diet (40 E%) with a very low fat diet (total fat 5–10 g), both with a P/S ratio of 0.1–0.3, significant decreases in LDL cholesterol and HDL cholesterol were observed, but again with no change in the LDL/HDL cholesterol ratio (25). Further, Ginsberg et al. (12) observed that compared with those who consumed an AAD (34.3 E% total fat, 15 E% SAFA, 6.5 E% PUFA), total and LDL cholesterol were lower in those who consumed a low saturated fat diet (28.6 E% total fat, 9 E% SAFA, 6.7 E% PUFA). HDL cholesterol was also lower and thus the LDL cholesterol/HDL cholesterol was unchanged (12).

We found that HDL cholesterol was significantly higher after intake of the HSAFA than after the HUFA diet. This is in accordance with previous findings that myristic acid increases HDL cholesterol (26). Data from two meta-analyses have shown that the saturated fatty acids 12:0–16:0 (13), and in particular 12:0 and 14:0 (27), are far more HDL cholesterol increasing than mono- or polyunsaturated fatty acids. Because coconut oil is very rich in lauric and myristic acids, this may explain the marked decrease in HDL cholesterol when changing from the HSAFA diet to the LSAFA or the HUFA diet. Why we observed a greater decrease in HDL cholesterol after intake of the LSAFA and HUFA diets, and thus a greater effect on the LDL/HDL cholesterol ratio than would be expected from similar previously published studies, may thus be

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Serum lipids and lipoprotein compositions in women at baseline consuming the three test diets&lt;sup&gt;1,2&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total cholesterol, mmol/L</td>
</tr>
<tr>
<td></td>
<td>Baseline</td>
</tr>
<tr>
<td>4.95 ± 0.71</td>
<td>5.38 ± 0.89&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>0.53 ± 0.24</td>
<td>0.49 ± 0.20&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>0.97 ± 0.20</td>
<td>0.97 ± 0.20&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>1</sup> Values are means ± sd, n = 25. Means with common superscripts differ: a and b, P < 0.01, c, P < 0.02 and d, P < 0.04.
<sup>2</sup> For fatty acid composition see Table 2.
<sup>3</sup> Diet high in saturated fatty acids.
<sup>4</sup> Diet low in saturated fatty acids.
<sup>5</sup> Diet high in poly- and monounsaturated fatty acids.
correlated to the use of coconut oil and additionally to the high content of carbohydrates in the LSAFA diet.

Several studies have shown that diets low in fat and high in carbohydrates decrease HDL cholesterol concentration (3,12,16,25,28) and this appears to be independent of the content of polyunsaturated fatty acids (29). It should be noted that in contrast to our results, Weisweiler et al. (30) found no significant difference in HDL cholesterol during intakes of 42 and 32 E% fat with a P/S ratio of 1.0. That study cannot be directly compared to ours, however, because the saturated fatty acids were derived mainly from animal sources.

The type of carbohydrate in the low fat diet may possibly be important both for the reductions in HDL cholesterol and the increase in plasma triacylglycerol after intake of the LSAFA diet. In healthy free-living men, Turley et al. (31) found that replacement of saturated fat with carbohydrates from grains, vegetables, legumes and fruit reduced total and LDL cholesterol, with only a minor effect on HDL cholesterol and triacylglycerol. Frost et al. (32) suggested that there is a connection between the glycemic index of the carbohydrates and the level of HDL cholesterol. Thus, intake of carbohydrates with a low glycemic index results in smaller HDL cholesterol reduction than after intake of carbohydrates with a higher index (32). In the LSAFA diet most of the eliminated fat was replaced by sucrose and fructose and thus of high glycemic index, which may have contributed to the depression of HDL cholesterol. On the other hand, the HDL-decreasing effect has been observed independent of the content of fiber in the carbohydrate diet (33).

There were significant (16 and 24%) increases both in triacylglycerol and VLDL concentrations after intake of the LSAFA diet than after intake of either of the HSAFA diet or the HUFA diet, which can be explained by the increase in the content of carbohydrate from 46.7 E% in the high fat diets to 63.8 E% in the LSAFA diet (12,28). Diets low in fat and high in carbohydrates increase triacylglycerol concentration through greater VLDL production and secretion by the liver (34). High carbohydrate/low fat intake is also associated with low serum HDL cholesterol and high triacylglycerol levels (35,36).

The effects of high carbohydrate/low fat diets on triacylglycerol and HDL cholesterol levels have been suggested to be transient (37,38), particularly after intake of ad libitum low fat diets (39). However, after changing dietary fat, others have shown that changes in HDL cholesterol and triacylglycerol are sustained for periods of several months (16) and up to 1 y (40).

The 25 participants in our study were all young women, who entered the study at different phases in their menstrual cycle, and to whom the diets were given in random sequence. This means that possible effects of the menstrual cycle on serum lipids (41,42) were minimized. Several studies reported similar effects of fatty acids on serum lipids in both men and women (12,13,43). There is thus no reason to believe that the results obtained in this study are valid only for females.

In this study we used coconut oil to vary the content of saturated fatty acids in the HSAFA and LSAFA diets. This oil should be particularly suitable because of its high content of the cholesterol-increasing fatty acids, 12:0, 14:0 and 16:0, and low content of stearic acid. Coconut oil has been shown to increase plasma cholesterol to about the same degree as that of butter fat (14). However, the effects of varying amounts of coconut oil in the diet on serum cholesterol do not necessarily follow different predictive equations. Thus, Keys et al. (3) found that their predictive equation appreciably overestimated the effect of hydrogenated coconut oil on serum total cholesterol. The Keys’ equation (4) predicts that changing from the HSAFA to the LSAFA diet would reduce total cholesterol 0.8 mmol/L, whereas our previously published equation (44) predicts 0.47 mmol/L, both considerably higher than the observed 0.25 mmol/L. The observed change from 0.97 to 0.99 mmol/L, from the HSAFA to the HUFA diet, respectively, was as predicted by our equation. Presumably, the predictability of such equations is lower when the change in fat energy percentage is very large. Thus, our total and LDL cholesterol predictive equations are based on studies with comparisons of the same fat energy percentage (44).

Reduction of dietary saturated (and trans) fatty acids is of primary importance in reducing the risk of cardiovascular diseases. There is considerable disagreement, however, as to what should replace saturated fat, carbohydrates or unsaturated fat (8,10). Replacing saturated fat by carbohydrates may increase serum triacylglycerol and VLDL cholesterol and reduce HDL cholesterol, as shown here. On the other hand, dietary changes with increased complex, fiber-rich carbohydrates are beneficial in efforts to maintain body weight control (45). Replacing saturated by unsaturated fatty acids will reduce LDL cholesterol and to some extent also reduce HDL cholesterol but less so than with carbohydrates, and thus result in a more favorable LDL/HDL cholesterol ratio. Evidence indicates that both LDL cholesterol/HDL cholesterol and apoB/apoA-I ratios are strong risk markers for coronary heart disease (46). Only long-term prospective studies will show which of these options is to be preferred. In the meantime the results of recently published prospective studies favor the view that replacing saturated and trans fatty acids by unsaturated fat may most effectively reduce the risk of cardiovascular diseases (2). This is also in accord with metabolic studies (11,14) and with the results of this study.

In conclusion, we have shown that the HDL cholesterol-increasing effect of lauric and myristic acids strongly influences the LDL/HDL cholesterol ratio. The results support the contention that reduction of dietary saturated fat high in these fatty acids, without altering the amount of unsaturated fat, does not appreciably alter serum LDL cholesterol, increases VLDL cholesterol and increases the LDL/HDL cholesterol ratio. Thus, the proportions of dietary fatty acids rather than restriction of the percentage of saturated fat energy may be of primary importance if the aim is to reduce the LDL/HDL cholesterol ratio.

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LITERATURE CITED


