Effect of a stearic acid–rich, structured triacylglycerol on plasma lipid concentrations

Paul J Nestel, Sylvia Pomeroy, Sally Kay, Takayuki Sasahara, and Takeshi Yamashita

ABSTRACT
Background: Structured lipids are being incorporated into foods to reduce their energy value. One such lipid is rich in stearic acid.

Objective: The objective of this study was to compare the effects on plasma lipids of a stearic acid–rich triacylglycerol and a fat rich in palmitic acid in hypercholesterolemic subjects.

Design: Fifteen subjects with an average plasma cholesterol concentration of 6.13 ± 0.80 mmol/L initially ate a low-fat diet for 2 wk (run-in period), followed in random order and blinded fashion by 2 high-fat diets (for 5 wk each) containing foods derived from margarines rich either in palmitic acid or in the structured, stearic acid–rich triacylglycerol.

Results: Plasma cholesterol concentrations with the low-fat, the stearic acid–rich, and the palmitic acid–rich diets were not significantly different (5.35 ± 0.83, 5.41 ± 0.78, and 5.52 ± 0.68 mmol/L, respectively) but were significantly lower (P < 0.001) than those measured during the habitual diet period (ie, 2 wk before the study began). Neither HDL cholesterol nor plasma triacylglycerol differed significantly among the 3 study diets.

Conclusion: A similar increase in the intake of stearic and palmitic acids (differing by <5% of total energy) to ensure a high fat intake resulted in plasma total and LDL-cholesterol concentrations that did not differ significantly from concentrations measured during a period of low-fat intake.


KEY WORDS Plasma lipids, palmitic acid, stearic acid–rich structured lipid, adults, humans

INTRODUCTION
The demand for low-fat and energy-reduced foods is being met through a variety of products, including structured lipids. Of these, a triacylglycerol containing stearic acid and short-chain fatty acids is currently being incorporated into confectionery and cookies. Although short-chain fatty acids contribute importantly to the body’s energy supply (1), their energy density is much less than that of long-chain fatty acids within a triacylglycerol molecule. Searic acid furthermore may be less completely absorbed than other long-chain fatty acids (2). It has therefore been postulated that a structured lipid containing stearic acid and short-chain fatty acids may have only ≈60% of the energy value of most edible triacylglycerols (3).

A further characteristic of stearic acid–containing triacylglycerols is their neutrality with respect to plasma cholesterol concentrations. Studies using sheanut oil (4, 5) and cocoa butter (6, 7) showed that their high stearic acid content prevented the increase in LDL cholesterol that generally occurs with other saturated fatty acids. However, the structured lipid incorporates stearic acid randomly into the middle and outside positions of the triacylglycerol molecule, whereas stearic acid is normally almost exclusively in the outside positions. Furthermore, a second structured lipid that contains 45% by weight of the poorly absorbed 22-carbon saturated fatty acid behenic acid (Caprenin; Proctor & Gamble, United Kingdom) was unexpectedly found to raise plasma cholesterol (8).

We therefore investigated the effect of the stearic acid–containing structured lipid on plasma lipids in hypercholesterolemic subjects. The average target intake of the lipid, which was incorporated into foods, was 30 g/d because this amount represents the anticipated upper level of consumption should this synthetic glyceride become included in a wide range of commercially available products (9). We compared the effects of stearic and palmitic acids, which differed by ≈5% of total energy, in 2 test margarines that were used as the test fats.

SUBJECTS AND METHODS
Subjects
Twenty middle-aged subjects (12 men and 8 women) were invited to participate in the study on the grounds that they were shown to have a high plasma cholesterol concentration during the preceding year. Potential subjects were excluded if they were aged >65 y, had a body mass index (in kg/m²) >32, drank >4 (men) or 2 (women) standard alcoholic drinks daily, had a disease (other than overweight) that could influence plasma lipid concentrations, had treatable hypertension, were smokers, or

1 From Baker Medical Research Institute, Melbourne.
2 Supported in part by Cultor Food Sciences USA, who also supplied Salatrim, and by Meadow Lea Foods (Australia), who also produced and provided the margarines and other test foods.
3 Address reprint requests to PJ Nestel, Baker Medical Research Institute, PO Box 6492, Melbourne 8008 Australia.
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were taking any medication that might influence lipid metabolism, including hormone replacement therapy. The trial was approved by the Alfred Hospital’s Human Ethics Committee and written, informed consent was obtained from the volunteers. The subjects had a mean (±SD) age of 51 ± 7 y, weight body of 77.8 ± 14.1 kg, and body mass index of 26.2 ± 3.9.

**Experimental design**

There were 3 dietary periods: an initial 2-wk run-in period during which a low-fat diet was consumed followed by randomization to the first of 2 high-fat diets containing the test fats for 5 wk each. Daily dietary records were obtained by the dietitian, who instructed the subjects and reviewed their progress by examining food records. The 2 test fats, 1 enriched in palmitic acid by use of palm oil and the other enriched in stearic acid by use of the structured triacylglycerol Salatrim (Cultor Food Sciences USA, Ardsley, NY), were each eaten for 5 wk in a crossover design. The subjects were unaware of the order in which they received the test fats but the investigators became aware of the order because of the different consistencies of the margarine enriched with palmitic acid (firm) and that enriched with stearic acid (soft).

Laboratory analyses were therefore carried out in a blinded fashion. All food products used were made with the test margarines (40–55 g/d), of which 20–35 g was eaten as margarine itself (80% fat content) and the remainder as 3–6 cookies (5.5 g fat in each) and 1–2 muffins (15.5 g fat in each).

The fatty acid composition of the 2 margarines is shown in **Table 1**. The analysis did not include the short-chain fatty acids acetic and propionic acids, which contributed <10% to the stearic acid–containing margarine. If acetic and propionic acids had been included, the amount of stearic acid would have been reduced by 3%. Assuming that about half of these volatile fatty acids (acetic and propionic acids) might be lost from the body, a small adjustment was made to the composition of the stearic acid–containing margarine so that both margarines contained equal amounts of fat-derived energy per unit mass.

To ensure that total saturates, monounsaturates, and polyunsaturates were approximately the same in both test margarines, mixtures of oils were added to each as necessary. The palmitic acid–rich margarine contained palm olein, palm stearin, and an oleic acid–rich oil—90% oleic acid sunflower (Sunola; Quality Food Oil Inc, Brooklyn, NY). The stearic acid–rich margarine contained 43% of the structured lipid (Table 1) plus sunflower oil and palm stearine. The oils that were added to the margarines were calculated to provide a fatty acid mix that was generally considered to be “cholesterol neutral.”

**Diet**

The screening procedure involved individual interviews and the completion of a questionnaire concerning habitual diet and exercise habits. Subjects and their spouses then met in small groups with the dietitian and received instructions on following a low-fat diet; recognizing, quantifying, and recording fats in food; and weighing food with digital electronic scales. The importance of maintaining a stable weight was emphasized. Two subjects who felt unable to follow these instructions dropped out. Subjects then commenced the 12-wk study, which was divided into 3 periods: a 2-wk run-in (low-fat) period followed by two 5-wk test-fat periods. Total fat was recorded daily by using simplified food tables. During each period, subjects weighed and recorded food and beverages for 3 d (2 weekdays and 1 weekend day). This information was checked during an interview with each subject and entered into a computer software program.

During the run-in period, subjects were instructed to limit their fat intake from sources other than the test fats (background fat) to <15% of energy (not including oils and spreads) so that their total fat-derived energy intake would be <25%. These food sources consisted of self-selected foods such as breads, cereals, vegetables, fruit, pasta, and rice. During the 2 test-fat diets, subjects were instructed to replace oils and spreads with the supplementary fats. Energy intake was determined for each subject during an interview, with information from the questionnaire, from the 24-h dietary recall, and usual exercise level taken into account.

Food records during each period included 3-d periods when the food eaten was weighed on electronic scales. The type and estimated amounts of fat consumed were calculated from food tables (10) and the subjects were asked to record such daily, which enabled them to focus on the need to maintain consistent eating patterns. Progress was evaluated at 2.5-wk intervals, which led to the early identification of 5 individuals whose record keeping was sporadic and presumably unreliable and whose adherence to eating the test fats as prescribed was in doubt (this was later confirmed by fatty acid analyses). Although these subjects were allowed to complete the study, their data were excluded from the analyses; therefore, complete and fully analyzed records were available for only 15 subjects. Reasons for faulty adherence included unforeseen business and entertainment commitments or an inability to consume the amount of added fat. Because the 2 test-fat diet periods resulted in almost identical plasma lipid concentrations in the 15 subjects who remained in the study, it is highly unlikely that the exclusion of the other 5 subjects confounded the outcome. The subjects maintained their normal activities.

**Laboratory measurements**

Blood was collected on 2 of the last 3 d in each period and plasma was stored at −70°C to be analyzed in one batch. Plasma cholesterol and triacylglycerol concentrations were measured with enzymatic kits with a Cobas-B10 automated analyzer (Roche, Basel, Switzerland). HDL cholesterol was separated...

**Table 1**

<table>
<thead>
<tr>
<th>Fatty acid</th>
<th>Palmitic acid–rich margarine</th>
<th>Stearic acid–rich margarine</th>
<th>Structured triacylglycerol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetic</td>
<td>NM</td>
<td>NM</td>
<td>22</td>
</tr>
<tr>
<td>Propionic</td>
<td>NM</td>
<td>NM</td>
<td>3</td>
</tr>
<tr>
<td>Lauric</td>
<td>0.1</td>
<td>&lt;0.1</td>
<td>—</td>
</tr>
<tr>
<td>Myristic</td>
<td>0.8</td>
<td>0.2</td>
<td>—</td>
</tr>
<tr>
<td>Palmitic</td>
<td>32</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Palmitoleic</td>
<td>0.3</td>
<td>0.1</td>
<td>—</td>
</tr>
<tr>
<td>Stearic</td>
<td>6</td>
<td>34</td>
<td>66</td>
</tr>
<tr>
<td>Oleic</td>
<td>52</td>
<td>48</td>
<td>—</td>
</tr>
<tr>
<td>Linoleic</td>
<td>7</td>
<td>7</td>
<td>—</td>
</tr>
<tr>
<td>Others</td>
<td>2</td>
<td>2</td>
<td>—</td>
</tr>
</tbody>
</table>

1NM, not measured. Estimated at 10% by wt from the structured lipid.

2Salatrim; Cultor Food Sciences USA, Ardsley, NY. This species of glyceride is not representative of all molecular species. Manufactured through interesterifying short-chain fatty acids with fully hydrogenated soybean oil.
from plasma by selective precipitation (11) and the LDL-cholesterol concentration was calculated (12). Fatty and methyl esters in plasma were determined by gas chromatography (13).

Statistical analyses

Repeated-measures analysis of variance (ANOVA) with Bonferroni adjustment was used to examine differences between mean plasma lipid concentrations during the 3 test periods and during the habitual diet. When significant differences were found, further analyses of the effects of the 2 test diets were carried out by Student's paired t test (SAS Institute Inc, Cary, NC).

RESULTS

Dietary fat consumption

Fifteen subjects provided satisfactory food records and direct information to the dietician to permit calculation of the group’s fat and cholesterol intakes during the 2 test-fat periods. Fat intake during the run-in period (=15% of energy) plus the fat intake provided by the test fats (=25% of energy) resulted in fat providing =40% of energy (Table 2). The average fat intake during the run-in period was 54 g, or 21% of the estimated energy intake. However, because body weights did not change during the study, fat intake values during the run-in period were approximate, mainly because the subjects were less able to provide consistent data during the short duration of the run-in period than during the longer test-fat periods. Subjects reduced their fat intake mainly by avoiding dairy fats, meat fats, and high-fat manufactured foods. Calculations of fat intake from the food diaries is likely to have been accurate because subjects had been instructed in how to accurately identify and quantify the fat in the food they ate. Body weights changed little during the study, averaging 77.4 ± 14.6, 78.2 ± 14.8, and 77.8 ± 14.4 kg at the end of the run-in, palmitic acid–rich, and stearic acid–rich diets, respectively. The average prestudy weight was 77.8 ± 14.1 kg. None of the subjects experienced adverse symptoms, such as bowel irritability.

Plasma lipids

Average total cholesterol, LDL-cholesterol, HDL-cholesterol, and triacylglycerol concentrations at the end of the run-in, palmitic acid–rich, and stearic acid–rich diets are shown in Table 3. Also shown are cholesterol and triacylglycerol concentrations at recruitment, which were measured =2 wk before the low-fat (run-in) diet began. These recruitment concentrations represent the subjects’ habitual lipid concentrations because they were similar to mean concentrations measured =6 mo before the study began (6.27 ± 0.80 mmol/L) and between 3 and 5 mo after the study ended (6.18 ± 0.76) in 13 of 15 subjects retested.

The low-fat intake during the 2-wk run-in period (ie, half that during the test-fat periods) resulted in a total cholesterol concentration that was significantly lower than that measured during the habitual diet, the difference being 0.78 mmol/L [P = 0.0018 (ANOVA across the periods) and P = 0.0004 for habitual compared with run-in, P = 0.0018 for habitual compared with palmitic, and P = 0.0069 for habitual compared with stearic, all after ANOVA with Bonferroni adjustments]. However, there were no subsequent significant changes; ie, neither the palmitic acid nor the stearic acid test fat raised plasma lipid concentrations back toward habitual values, despite the restoration of fat intakes toward habitual intakes. Nor were there significant differences in lipid concentrations between the 2 test-fat periods. The plasma triacylglycerol concentration also decreased significantly during the 2-wk run-in period relative to the habitual concentration, from 1.63 ± 0.73 to 1.38 ± 0.70 mmol/L (P < 0.05), and did not increase toward the habitual value after consumption of the 2 test fats: 1.58 ± 0.90 and 1.49 ± 0.66 mmol/L at the end of the palmitic acid–rich and stearic acid–rich diets, respectively.

### Table 2

<table>
<thead>
<tr>
<th>Diet</th>
<th>Run-in (low-fat) diet</th>
<th>Palmitic acid–rich diet</th>
<th>Stearic acid–rich diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total fat (g)</td>
<td>54 ± 30</td>
<td>117 ± 61</td>
<td>118 ± 38</td>
</tr>
<tr>
<td>(% of energy)</td>
<td>21</td>
<td>41</td>
<td>42</td>
</tr>
<tr>
<td>Saturated fat (g)</td>
<td>22 ± 14</td>
<td>51 ± 18</td>
<td>55 ± 15</td>
</tr>
<tr>
<td>(% of energy)</td>
<td>8.6 ± 5.4</td>
<td>17.9 ± 6.3</td>
<td>19.5 ± 5.3</td>
</tr>
<tr>
<td>Polyunsaturated fat (g)</td>
<td>8 ± 4</td>
<td>14 ± 14</td>
<td>14 ± 6</td>
</tr>
<tr>
<td>(% of energy)</td>
<td>3.1 ± 1.5</td>
<td>4.9 ± 4.9</td>
<td>5.0 ± 2.1</td>
</tr>
<tr>
<td>Monounsaturated fat (g)</td>
<td>19 ± 13</td>
<td>52 ± 27</td>
<td>53 ± 15</td>
</tr>
<tr>
<td>(% of energy)</td>
<td>7.4 ± 5.0</td>
<td>18.2 ± 9.4</td>
<td>17.1 ± 4.8</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>218 ± 134</td>
<td>284 ± 178</td>
<td>283 ± 160</td>
</tr>
<tr>
<td>Energy (MJ)</td>
<td>9852</td>
<td>11741 ± 4188</td>
<td>11549 ± 3084</td>
</tr>
</tbody>
</table>

1 x ± SD; n = 15. Values based on food records, focusing on fat and on direct discussion with a dietitian at 2.5-wk intervals.

2 The shorter duration (2 wk) of the run-in diet than of the test-fat diets (5 wk each) and the focus on identifying and reducing fat led to less reliable data on other macronutrients and energy; the percentage of energy derived from fat (21%) was thus maximal.

### Table 3

<table>
<thead>
<tr>
<th>Lipid</th>
<th>Habitual diet</th>
<th>Run-in (low-fat) diet</th>
<th>Palmitic acid–rich diet</th>
<th>Stearic acid–rich diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma cholesterol</td>
<td>6.13 ± 0.80</td>
<td>5.35 ± 0.83</td>
<td>5.52 ± 0.68</td>
<td>5.41 ± 0.78</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>—</td>
<td>3.63 ± 0.71</td>
<td>3.71 ± 0.66</td>
<td>3.65 ± 0.71</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>—</td>
<td>1.08 ± 0.34</td>
<td>1.09 ± 0.34</td>
<td>1.08 ± 0.33</td>
</tr>
<tr>
<td>Plasma triacylglycerol</td>
<td>1.63 ± 0.73</td>
<td>1.38 ± 0.70</td>
<td>1.58 ± 0.90</td>
<td>1.49 ± 0.66</td>
</tr>
</tbody>
</table>

1 x ± SD; n = 5.

2 Habitual plasma cholesterol and triacylglycerol concentrations were measured at the time of recruitment, 2 wk before the trial began.

3 Significantly different from the 3 other diets, P = 0.0018 (ANOVA).

4 Significantly different from the run-in diet, P < 0.05 (Student’s paired t test).
The present data can best be compared with data from 3 recent comparisons of stearic acid–rich diets with similar diets rich in other long-chain saturates: palmitic, myristic, and lauric acids. In addition, a recent meta-analysis of many well-controlled studies of the effects of dietary fatty acids on plasma lipids provided a reference for stearic acid. Although the overall conclusion is consistent with stearic acid having the least plasma cholesterol-raising effect of the 12–18-carbon saturated fatty acids, the magnitude and duration of the effects varied.

In 1994, Tholstrup et al (5) reported the greatest difference between the 12–16-carbon fatty acids and stearic acid. The source of stearic acid was shea butter, in which stearic acid is almost entirely in the outside position in the triacylglycerol molecule. Test fats contributed almost all of the fat (36% shea butter providing 42% of energy as fat), the duration of each test period was 3 wk (the first blood sample was taken at 2 wk), and the subjects were normocholesterolemic. Average LDL-cholesterol concentrations during the diets rich with 12–14-carbon, 16-carbon, and 18-carbon fatty acids were 3.07 ± 0.14, 2.96 ± 0.14, and 2.18 ± 0.12 mmol/L, respectively; concentrations were lowest during the stearic acid–rich diet.

The second study, reported in 1995 by Dougherty et al (4), also tested sheanut oil as the source of stearic acid and compared its effects with those of a diet high in palmitic acid. The study resembled ours in that the difference in energy provided by the 16- and 18-carbon fatty acids between the 2 test periods was slightly >5%. Note that the duration of the study by Dougherty et al (40 d) was similar to the duration of our study (35 d). This was important because the differences in LDL cholesterol between the high–stearic acid and high–palmitic acid diet periods were much higher at 20 d than at 40 d: LDL cholesterol fell initially with the high–stearic acid diet but then rose by 10% during the final 20 d. LDL-cholesterol concentrations in the 10 normolipidemic men were 3.1 ± 0.2 and 3.3 ± 0.2 mmol/L, respectively, after 40 d of the high–stearic acid and high–palmitic acid diets, respectively.

This small difference was similar to that in the present study.

Bonanome and Grundy (14) had previously found a bigger difference between the effects of stearic and palmitic acids on LDL cholesterol. Like Tholstrup et al (5), they provided nearly all the fat as one of the test fats; furthermore, they formulated a series of synthetic fats that through interesterification would have resulted in about one-third of the stearic acid occupying the midposition in the triacylglycerol molecule as in the present formulation. Stearic and palmitic acids provided 17% in one or more of the test fats; the respective LDL-cholesterol concentrations were 2.84 and 3.62 mmol/L.

The results of previous studies in which the test diets were based on cocoa butter (6, 7), which, although also indicated stearic acid as being “cholesterol neutral,” are more difficult to interpret because of the high–palmitic acid content of cocoa butter. The results of the 3 studies summarized above, together with the results of our present study, suggest a clear difference between the effects of stearic and palmitic acids (in favor of the former) on LDL cholesterol when these fatty acids provide most of the dietary fat, but possibly only for a short time— 3 wk. At lower intakes of stearic acid and over longer periods, its advantage over palmitic acid diminishes. However, as suggested in our study, LDL cholesterol is not raised above that seen with a low-fat diet.

We were limited in the amount of the structured lipid we could incorporate into the diets because the objective of the study was to test the amount that might be eaten if the structured lipid was

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**TABLE 4**

Percentages of the major fatty acids during the habitual and palmitic acid– and stearic acid–rich diet periods

<table>
<thead>
<tr>
<th>Fatty acid</th>
<th>Habitual diet</th>
<th>Palmitic acid–rich diet</th>
<th>Stearic acid–rich diet</th>
<th>% of total fatty acids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Palmitic</td>
<td>22.5 ± 1.2</td>
<td>24.8 ± 2.1</td>
<td>21.7 ± 2.3*</td>
<td></td>
</tr>
<tr>
<td>Stearic</td>
<td>6.9 ± 1.1</td>
<td>6.9 ± 1.0</td>
<td>8.2 ± 1.0*</td>
<td></td>
</tr>
<tr>
<td>Oleic</td>
<td>26.2 ± 4.4</td>
<td>27.3 ± 4.1</td>
<td>27.9 ± 3.3</td>
<td></td>
</tr>
<tr>
<td>Linoleic</td>
<td>28.9 ± 5.1</td>
<td>27.2 ± 4.1</td>
<td>27.5 ± 4.0</td>
<td></td>
</tr>
</tbody>
</table>

1SD; n = 15.
2Significantly different from palmitic acid–rich diet (Student’s t test):

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**Plasma fatty acids**

Plasma palmitic, stearic, oleic, and linoleic acids as a percentage of total fatty acids during the habitual, palmitic acid–rich, and stearic acid–rich diets are shown in Table 4. In the 15 subjects who adhered to the 2 test-fat diets, palmitic acid was significantly higher (P < 0.0001) during the palmitic acid–rich diet (24.8 ± 2.1%) than during the stearic acid–rich diet (21.7 ± 2.3%) and stearic acid was significantly higher (P < 0.0001) higher during the stearic acid–rich diet (8.2 ± 1.0%) than during the palmitic acid–rich diet (6.9 ± 1.0%). Oleic and linoleic acids did not differ significantly between the 2 test-fat periods. Palmitic acid was significantly lower (P < 0.05) during the habitual diet (22.5 ± 2.1%) than during the palmitic acid–rich diet (24.8 ± 2.1%).

**DISCUSSION**

The present study had 2 objectives. First, to use the unique characteristic of the structured triacylglycerol to test the effects of stearic acid on plasma cholesterol because the triacylglycerol contains no other long-chain fatty acids, other than a minimal amount of palmitate. Second, to determine whether structured glycerides may have an unusual effect on plasma lipids because another structured lipid, a completely different synthetic glyceride that contained the poorly absorbed behenic acid, was unexpectedly found to raise LDL cholesterol (8). Preliminary data on the structured lipid used in this study, the safety of which was established for human consumption, did not show an effect on plasma cholesterol in healthy volunteers (9).

In the present investigation of 15 men and women known to have been hypercholesterolemic on ≥2 occasions in the preceding 6 mo, the daily consumption of 30 g of the structured glyceride for 5 wk did not raise LDL cholesterol significantly, despite the fact that concentrations had decreased initially during the run-in period (low-fat diet) (Table 3), nor did HDL cholesterol decrease significantly, a finding observed in a previous study in which high amounts of stearic acid were tested (5). A second test diet, which contained 8% more energy as palmitic acid and 5% less energy as stearic acid, resulted in an average LDL-cholesterol concentration that was not significantly different from that during the stearic acid–rich diet or the low-fat diet.

The inclusion of 30 g of the structured triacylglycerol in the diet daily thus did not raise LDL cholesterol in hypercholesterolemic subjects over the 5-wk study period. This amount has been estimated to represent the daily 90th percentile intake of the structured lipid likely to be eaten in commercially available foods containing the lipid (9).
widely used as a fat substitute in appropriate foods (30 g). Thirty-three percent of the lipid incorporated into the test foods was stearic acid; however, the amount was less when the amount of fat in the background diet was taken into account. The test diets provided 20% of total energy, or ~55% of total fat. Assuming that the remaining 45% of fat (14% of energy) in the background diet did not differ significantly during the 2 test periods (on the basis of food records), the difference in stearic and palmitic acid contents between the 2 dietary periods was ~5% of total energy for each. On this basis, we calculated the respective effects of the 2 fatty acids on changes in LDL cholesterol using equations published recently by Yu et al (15). These equations were derived from a meta-analysis of 18 studies that contained sufficient detail to allow evaluation of the individual contributions from stearic and palmitic acids to plasma cholesterol concentrations. From their equations 4 and 5, both fatty acids predicted an increase in LDL cholesterol, which was only moderately greater for palmitic acid. The larger influence of palmitic acid on total cholesterol included a rise in HDL cholesterol, which decreased with stearic acid. Given the respective amounts of the 2 test fatty acids in the present study, the average difference in LDL cholesterol in Yu et al’s study was calculated to be <0.2 mmol/L, which is consistent with that observed in the present study. For such a small difference to achieve statistical significance, a large number of subjects need to be studied.

It might be asked why palmitic acid did not raise LDL cholesterol significantly above that observed after the 2-wk period of low fat intake (run-in period). This short intervention was sufficient to lower total cholesterol significantly by 13% (from an average of 6.13 ± 0.8 mmol/L to 5.35 ± 0.83 mmol/L, P < 0.001). With use of the equation of Yu et al (15), an increase in palmitic acid of 5% of energy would have led to the observed difference. Although this may appear less than that reported by many researchers, including ourselves (16–21), a reanalysis of our published data (19–22) suggests that when the effects of palmitic acid (from palm olein) were compared with those of a low-fat, high-carbohydrate diet, the average difference in LDL cholesterol was <0.2 mmol/L. However, the average difference was twice that when similar increments in palmitic acid were compared with diets rich in polyunsaturates or monounsaturates, which also lowered LDL cholesterol compared with low-fat diets.

Since the original analyses by Keys et al (16) and Hegsted et al (23), the degree to which palmitic acid raises plasma cholesterol has been disputed, with concurrence that the LDL-cholesterol concentration is higher in hypercholesterolemic subjects and that palmitic acid raises LDL cholesterol to a greater degree when compared with unsaturated fatty acids (16–23). In the present study, ~20 g stearic or palmitic acid was eaten as supplements (one-third of an average 60 g/d) together with other saturates as well as oleic and linoleic acids (mostly oleic), which were calculated to be “cholesterol neutral” and would not have been expected to prevent any cholesterol-raising potential of stearic acid. Cholesterol consumption during the 2 test diets was low (<290 mg/d), and this might also have modified the plasma cholesterol-raising potential of palmitic acid (24).

The reasons for the minimal cholesterol-raising effects of stearic acid have not been adequately explained. Earlier studies, mainly based on tristearin, showed poor digestibility and absorption (2). However more recently, absorption of stearic acid from cocoa butter (6) and from a synthetic, interesterified triacylglycerol (14) was reported to be >90%. Confounding variables include the degree of emulsification in the intestine (25) (tristearin being poorly emulsified) and the load of dietary stearic acid (4). Absorption of stearic acid may also be influenced by its position in the triacylglycerol molecule; the middle or β position, which gives rise to monoacylglycerol is thus absorbed more readily than are fatty acids from the outside positions of the molecule. In the structured lipid used in the present study, 33% of the stearic acid would theoretically have been in that position. Published studies of its absorption in rodents show that is incompletely absorbed (3). The synthetic lipid contains at least one short-chain fatty acid molecule and one stearic acid molecule per triacylglycerol molecule, but because stearic acid was the predominant fatty acid in the triacylglycerol used in the present study, we assumed that there was a substantial contribution from a second stearic acid molecule per triacylglycerol molecule. In our study there was no evidence of fat malabsorption; none of the subjects complained of diarrhea and average body weights remained stable compared with the weights during the run-in period.

It has been postulated that the positional specificity of stearic acid, the α position of the triacylglycerol, reduces its effect on plasma cholesterol, but recent studies with interesterified triacylglycerols have shown that this process does not affect the cholesterol-raising or -lowering properties of fatty acids (21). Plasma triacylglycerol concentrations did not differ significantly between the 2 test-fat diets, but decreased during the run-in period (low-fat diet). The triacylglycerol-raising potential of a low-fat diet is modified by the energy content of the diet and the nature of the carbohydrates and fats (26). In the present study, carbohydrates were rich in starch and fiber, and fat reduction was achieved largely by reducing the amount of saturated fat, both of which likely limited a rise in plasma triacylglycerol. A small reduction in energy intake could not, however, be excluded.

A similar increase in the intake of stearic and palmitic acids (differing by ~5% of total energy) to ensure a high fat intake resulted in plasma total and LDL-cholesterol concentrations that did not differ significantly from concentrations measured during a period of low-fat intake.

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REFERENCES
25. Emken EA. Metabolism of dietary stearic acid relative to other fatty acids in human subjects. Am J Clin Nutr 1994;60(suppl):1023S–8S.