The effect of palmitic acid (C16:0) on serum lipoprotein cholesterol levels is debatable. If C16:0 is hypercholesterolemic, then it may increase the endogenous cholesterol or decrease plasma cholesterol levels. Therefore, the current study was conducted to determine if C16:0 would decrease plasma cholesterol levels in a 24-week randomized, double-blind, placebo-controlled, crossover study.}

**Introduction**

Specific dietary saturated fatty acids may raise total cholesterol levels, and specifically LDL-cholesterol levels. However, the effects of individual saturated fatty acids are complicated by evidence showing that all saturated fats do not affect lipoprotein profiles equally. Stearic acid has little effect on plasma lipid levels, exerting a neutral effect similar to that of monounsaturated, while lauric and myristic acid-rich fats have potent cholesterol-raising effects.

The primary structural saturated fatty acid is palmitic acid. Palmitic acid is the major saturate in animal fats, occurring in large proportions in both meat and dairy products, as well as constituting a substantial component of fats in plants.

The earlier studies that involved palmitic acid identified this saturate as exerting a significant hypercholesterolemic effect. However, these findings have recently been questioned by a number of researchers. Thus the hypercholesterolemic effect of palmitic acid is debatable.

**Methods**

Studied which have identified palmitic acid as a cholesterol-raising fatty acid have focused on the effect of palmitic acid on the LDL-cholesterol fraction. It has been suggested that palmitic acid may suppress expression of LDL receptors, or accelerate VLDL cholesterol production, or increase adipose tissue synthesis.

Evidence also suggests that palmitic acid may enhance HDL-cholesterol production. Lindsey et al. examined the qualitative effects of specific fatty acids on plasma lipoprotein metabolism by feeding mice six, low-fat, cholesterol-free diets for four weeks each. The fat blends differed only in their source of fat: coconut oil, palm oil, sunflower oil, corn oil, and canola oil. In three of the diets, the PUFAs/MUFAs/SFA ratio was held constant while the lauric, myristic and palmitic acid levels varied. Replacing lauric acid with medium-chain triglycerides from coconut oil with palm oil induced a significant increase in HDL subclasses and a decrease in TG.

Prior to being assigned to each diet, it was confirmed that if palmitic acid raises serum cholesterol it may exert this effect by increasing the concentration of lipoprotein fractions known to favourably impact atherogenesis, i.e., HDL.

The controversy of dietary palmitic acid is further complicated by other researchers who have failed to demonstrate elevated plasma cholesterol following palmitic acid consumption. Ng et al. compared the effects of palmitic acid and oleic acid in normocholesterolemic subjects. Based on the observation that there is evidence suggesting that if palmitic acid increases serum cholesterol it may exert this effect by increasing the concentration of lipoprotein fractions known to favourably impact atherogenesis, i.e., HDL.

The controversy of dietary palmitic acid is further complicated by other researchers who have failed to demonstrate elevated plasma cholesterol following palmitic acid consumption. Ng et al. compared the effects of palmitic acid and oleic acid in normocholesterolemic subjects. Based on the observation that there is evidence suggesting that if palmitic acid raises serum cholesterol it may exert this effect by increasing the concentration of lipoprotein fractions known to favourably impact atherogenesis, i.e., HDL.

**Results**

The study concluded that the metabolic effect of palmitic acid is a matter of change observed in serum cholesterol levels. This observation has been supported by Hoyes, who also noted a neutral fatty acid.

In a study which examined 5% of energy from 12.0% for 16:0 in a healthy young man consuming a low-cholesterol diet, the dietary composition of the diet was analyzed. At the conclusion of the study, 14.7% energy from 12.0% for 16:0 was found to be associated with a significant increase in serum cholesterol levels by about 16.7%.

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**Table 1: Nutritional composition of diets**

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Low C16:0</th>
<th>Low C16:0</th>
<th>Low C16:0</th>
<th>High C16:0</th>
<th>High C16:0</th>
<th>High C16:0</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Calories</strong></td>
<td>2957.7 kcal</td>
<td>2957.7 kcal</td>
<td>2957.7 kcal</td>
<td>2957.7 kcal</td>
<td>2957.7 kcal</td>
<td>2957.7 kcal</td>
</tr>
<tr>
<td><strong>Protein (g)</strong></td>
<td>73.6 (4.9)</td>
<td>73.6 (4.9)</td>
<td>73.6 (4.9)</td>
<td>73.6 (4.9)</td>
<td>73.6 (4.9)</td>
<td>73.6 (4.9)</td>
</tr>
<tr>
<td><strong>Carbohydrate (g)</strong></td>
<td>273.2 (17.0)</td>
<td>273.2 (17.0)</td>
<td>273.2 (17.0)</td>
<td>273.2 (17.0)</td>
<td>273.2 (17.0)</td>
<td>273.2 (17.0)</td>
</tr>
<tr>
<td><strong>Fat (g)</strong></td>
<td>73.6 (4.9)</td>
<td>73.6 (4.9)</td>
<td>73.6 (4.9)</td>
<td>73.6 (4.9)</td>
<td>73.6 (4.9)</td>
<td>73.6 (4.9)</td>
</tr>
<tr>
<td><strong>Saturated Fat (g)</strong></td>
<td>4.8 (0.7)</td>
<td>4.8 (0.7)</td>
<td>4.8 (0.7)</td>
<td>4.8 (0.7)</td>
<td>4.8 (0.7)</td>
<td>4.8 (0.7)</td>
</tr>
<tr>
<td><strong>MUFAs (g)</strong></td>
<td>18.8 (0.9)</td>
<td>18.8 (0.9)</td>
<td>18.8 (0.9)</td>
<td>18.8 (0.9)</td>
<td>18.8 (0.9)</td>
<td>18.8 (0.9)</td>
</tr>
<tr>
<td><strong>EPA (g)</strong></td>
<td>0.1 (0.0)</td>
<td>0.1 (0.0)</td>
<td>0.1 (0.0)</td>
<td>0.1 (0.0)</td>
<td>0.1 (0.0)</td>
<td>0.1 (0.0)</td>
</tr>
<tr>
<td><strong>DHA (g)</strong></td>
<td>0.1 (0.0)</td>
<td>0.1 (0.0)</td>
<td>0.1 (0.0)</td>
<td>0.1 (0.0)</td>
<td>0.1 (0.0)</td>
<td>0.1 (0.0)</td>
</tr>
</tbody>
</table>

**Conclusion**

The study concluded that the metabolic effect of palmitic acid is a matter of change observed in serum cholesterol levels. This observation has been supported by Hoyes, who also noted a neutral fatty acid. In a study which examined 5% of energy from 12.0% for 16:0 in a healthy young man consuming a low-cholesterol diet, the dietary composition of the diet was analyzed. At the conclusion of the study, 14.7% energy from 12.0% for 16:0 was found to be associated with a significant increase in serum cholesterol levels by about 16.7%.
The effect of palmic acid (C16:0) on serum lipoprotein levels is debatable. If C16:0 is hypercholesterolemic, then it may increase the endogenous synthesis and decrease clearance of cholesterol. Foods enriched in C16:0 were formulated to provide combinations of two levels of C16:0 in two ratios of LDL/VLDL. Healthy male subjects received the four diet treatments for 21 days, followed by washout periods of 21 days. After 21 days of each diet treatment, a fasting blood sample was drawn for lipoprotein determination and to provide a measure of the background level of serum. A repeating dose of diet was administered at a second- and a second-12-hour interval after the first sample. Triglyceride mass spectrometry was used to determine the incorporation of diet into serum lipoprotein fractions and the fractional synthetic rate of cholesterol. Serum total cholesterol and LDL-cholesterol levels were significantly affected by the high level of C16:0 when diets also contained high saturated fat. There was no effect of C16:0 on HDL-cholesterol at either the high or low levels of intake. The fractional synthetic rates of cholesterol were not significantly different from one another, suggesting no relationship between the endogenous synthesis of cholesterol and diet C16:0 content. These results indicate that C16:0 had no effect on serum lipoprotein profiles in the presence of recommended intakes for PUFAs, nor did it increase rates of cholesterol synthesis in healthy males.

Key words: Lipidemia, fat intake, palmitic acid

Introduction

Specific dietary saturated fatty acids may raise total cholesterol levels, and specifically LDL-cholesterol levels. However, the effects of individual saturated fatty acids are complicated by evidence showing that all saturated fats do not affect lipoprotein profiles equally. Stearic acid has little effect on plasma lipid levels, exerting a neutral effect similar to that of some monounsaturated, while lauric and myristic acid-rich fats have potent cholesterol-raising effects. The principal dietary saturated fatty acid is palmitic acid. Palmitic acid is the major saturated in animal fats, occurring in large proportions in both meat and dairy products, as well as coconut and palm kernel oils. The earlier studies that involved palmitic acid identified this saturated as exerting a significant hypercholesterolemic effect. However, these findings have recently been questioned by a number of researchers. Thus the hypercholesterolemic effect of palmitic acid is debatable.

A controlled metabolic feeding study Tholstrup et al investigated the effects of three diets differing in the total fatty acid supplied (12.0, 14.0, and 16.0 g/d). According to original Keys hypothesis, palmitic acid should increase blood cholesterol levels. However, these commonly cited equations failed to separate the effects of these fatty acids (12.0, 14.0, and 16.0 g/d), defining these as equally hypercholesterolemic. In fact, the equation modified to treat palmitic acid as neutral (similar to stearic acid) provides a better description of the changes observed in serum cholesterol levels. This observation has been supported by Hoye, who described the effects of saturated fatty acid(C).

In a study which exchanged 5% of energy from 12.0 to 14.0 g/d for 16:0 in healthy young men consuming a low cholesterol diet, the dietary combination of 14.0 g/d produced significantly higher serum cholesterol levels than did 12.0 g/d.

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Table 1: Nutrient composition of diets

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Low C16:0</th>
<th>Low C16:0</th>
<th>High C16:0</th>
<th>High C16:0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calories</td>
<td>2030 ± 1</td>
<td>2030 ± 1</td>
<td>2030 ± 1</td>
<td>2030 ± 1</td>
</tr>
<tr>
<td>Protein</td>
<td>157 ± 3.3</td>
<td>157 ± 3.3</td>
<td>157 ± 3.3</td>
<td>157 ± 3.3</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>56.5 ± 4.8</td>
<td>57.0 ± 3.3</td>
<td>57.8 ± 0.8</td>
<td>57.0 ± 3.3</td>
</tr>
<tr>
<td>Fat</td>
<td>27.1 ± 1.7</td>
<td>27.1 ± 1.7</td>
<td>27.1 ± 1.7</td>
<td>27.1 ± 1.7</td>
</tr>
<tr>
<td>Saturated fat</td>
<td>4.8 ± 0.8</td>
<td>4.7 ± 0.3</td>
<td>3.9 ± 0.6</td>
<td>3.9 ± 0.6</td>
</tr>
<tr>
<td>MUFAs</td>
<td>18.8 ± 0.9</td>
<td>18.7 ± 0.9</td>
<td>18.7 ± 0.9</td>
<td>18.7 ± 0.9</td>
</tr>
<tr>
<td>SFA</td>
<td>3.2 ± 0.2</td>
<td>3.1 ± 0.2</td>
<td>3.1 ± 0.2</td>
<td>3.1 ± 0.2</td>
</tr>
<tr>
<td>n-3 PUFAs</td>
<td>0.5 ± 0.1</td>
<td>0.5 ± 0.1</td>
<td>0.5 ± 0.1</td>
<td>0.5 ± 0.1</td>
</tr>
<tr>
<td>choline</td>
<td>27.6 ± 2.0</td>
<td>27.6 ± 2.0</td>
<td>27.6 ± 2.0</td>
<td>27.6 ± 2.0</td>
</tr>
</tbody>
</table>

1 Values represent means ± SD for each diet treatment. Diet averages are calculated from the mean nutrient values for each diet. All values are derived from Food Processor III data and are based on selected food composition tables.
Between 0715 and 0830 on day 21 of each diet phase, a fasting blood sample was obtained by venipuncture (30 ml) and subjects consumed a pruning dose of 0.25 D2O/kg estimated total body water (29.8 kg). Subjects (n = 8; Montreal, Canada) prior to breakfast. Total body water was estimated as 60% of body weight (taken as the average over each of the 21 day feeding period). A 2.0 litre bottle of water containing 1.0g D2O/kg estimated total body water was provided for consumption over the next 24 hour period to maintain plasma deuterium enrichment at plasma deuterium enrichment values obtained in the diet at 24 hours after the last sample, a second fasting blood sample (25 ml) was obtained. Plasma was obtained by centrifugation at 3000 rpm and frozen at -20°C. Plasma (5 ml) was sent to the University of Alberta Hospitals for total cholesterol, LDL-C, HDL-C, and creatinine determination. Total cholesterol content was determined enzymatically. 33,35 HDL cholesterol was determined following precipitation of other lipoproteins using dextran sulphate magnesium as described. 32,34 LDL cholesterol levels were calculated by subtraction from the cholesterol level of 1.06 g/mL infranatant fraction. Serum creatinine levels were determined by the kinetic Jaffe reaction.33,34

Fat analysis of diets. Duplicate freeze-dried preparations of each complete meal cycle for each diet phase were homogenised in a polytron, aliquoted (10 g samples) and stored at -20°C until analysed for total fat and fatty acid content. Fat extraction was carried out prior to saponification and transesterification with KOH and methanol as described.33 Free acid methyl esters were analysed by gas liquid chromatography (Vista 6010 GLC and Vista 402 data system; Varian Instruments, Georgetown, Ontario). Fatty acid methyl esters were identified by comparison of retention times with that of authentic standards and quantified by peak area comparison with internal standards.

Determination of deuterium enrichment. Deuterium enrichment was measured in plasma water, plasma cholesterol and plasma cholesterol ester. To extract the free and esterified cholesterol, 2 ml of plasma at each time point was combined with 4 ml of methanol and heated at 60°C for 15 min. 12 ml of a 4:1 hexane:chloroform solution (v/v) was added and shaken mechanically for 10 min. 1 ml of water was added to inhibit the mixture shaken after. Centrifugation at 1500 x g for 1 min. was followed by removal of the upper hexane:chloroform phase. This process was repeated, upper phases combined, and solvent removed under nitrogen. Extracts were redissolved in 200 μL chloroform and spotted onto thin-layer silica gel plates (Analtech Inc., Newark, DE). Plates were developed using petroleum ether-dichloromethane-ethyl acetate (80:20:1, v/v/v), and air-dried. Lipid fractions were identified by comparison with a standard (Supelco, Bellefonte, PA). Free and esterified cholesterol bands were scraped from the plate and placed into 10 mm tubes containing 2.0 M HCl in methanol for 2 hour in a sand bath and the resultant free cholesterol was purified by thin layer chromatography as before. The saponification step was repeated once more with the cholesterol esterified band and the free cholesterol pooled with the previous lot. After two saponifications, no further cholesterol ester could be converted to free cholesterol. The dried cholesterol sample was transferred to a 1.5 ml Pyrex tube, using three washes of chloroform. Cupric oxide water (0.5 g) and a 2.5 cm-length 1 mm silver foil was added to the tube. The tube was placed inside a 15 x 9 x 5 mm Pyrex (Corning Glass Works, Corning, NY) sealed at one end. Combustion tubes were evacuated to less than 50 mtorr before being sealed with a hot flame. Tubes were placed in a 50°C for 4 hours to combustion the cholesterol to carbon dioxide and water. After cooling the furnace overnight, the tubes were attached to a vacuum manifold by means of flexible tubing between Cajon fittings (Swagelok Canada Ltd, Niagara Falls, Ontario). The seal was broken by flexing the tube. The combustion product water was transferred by vacuum distillation into a second Pyrex tube containing 60 μg of zinc mercaptide. The water samples from plasma water and combustion of cholesterol were reduced by zinc to hydrogen gas by placing the reaction tubes in a heating block at 470°C for 30 min. The reaction tubes could be attached directly to the mass spectrometer without further purification. The deuterium enrichment was measured by use of a Finnigan MAT 5110 Isotope Ratio Mass Spectrometer (Bremen, Germany) against hydrogen prepared from a water standard. The mass three abundance was corrected for H2 contribution. Mass abundance of hydrogen was corrected. A laboratory standard demonstrated the analytical precision (coefficient of variation) of this instrument at <1%. All samples were analysed in duplicate.

Cholesterol fractional synthesis rates (FSR) were determined from the initial incorporation rate of deuterium labelled cholesterol into the rapidly exchangeable cholesterol pool, relative to the initial precursor enrichment as determined using the body water deuterium enrichment. Maximum attainable body water enrichment was calculated as the body water pool enrichment corrected for the fraction of protons in de novo synthesised cholesterol that derive from water, as described previously.36 Fatty acid methyl esters were identified by comparison of retention times with that of authentic standards and quantified by peak area comparison with internal standards.

Results and discussion

Subjects. Demographic data of study participants is shown in Table 1. By observation of subjects in the metabolic unit, self reports by subjects and the lack of meals returned unfinished indicated that the level of subject compliance in completing meals was high. Weight body weight fluctuations by each subject over each diet feeding period was negligible (ranging from 0.2 to 0.6 kg). Body weight for individuals over the entire study period varied somewhat. The group mean weight change was small (+2.9%); the majority of weight change occurring during the washout periods. Mean creatinine levels were not significantly different between background and test diets for each subject on each diet treatment providing a measure of similarity status for each test period in (no significant change).

Diet. Composition of the diets consumed is shown in Table 2. The contribution of energy from protein, carbohydrate and fat was within 3.4% for each of the macronutrients between diet treatment groups. There were no differences in these treatments in regard to dietary fibre (29.9 ± 2.8 g), omega-3 fatty acid (1.54 ± 0.16mg) and cholesterol (183.4 ± 26.6 mg) content. The arachidonic acid, eicosapentaenoic acid and docosahexaenoic acid was negligible in each of the diet treatment (0.016 ± 0.006%, 0.022 ± 0.012%, and 0.030 ± 0.011% of fatty acids respectively). In diets in which the C16:0 and C18:2n-6 content did not provide more of the required dietary fat, the remainder of the fat was provided by monounsaturated fatty acids. The analysed fat content of meals in each diet treatment was close to the formulated values, and was consistent within each diet treatment.

Table 3. Lipoprotein cholesterol levels for subjects studied

| Main effects of diet treatment | Total cholesterol | LDL-cholesterol | HDL-cholesterol |
|------------------------------|-------------------|----------------|-----------------
| Low C16:0                     | 3.19 ± 0.05       | 1.79 ± 0.04    | 1.11 ± 0.02    |
| High C16:0                    | 3.69 ± 0.05       | 2.22 ± 0.04    | 1.19 ± 0.02    |
| Low C18:2                     | 3.53 ± 0.05       | 2.08 ± 0.04    | 1.15 ± 0.02    |
| High C18:2                    | 3.35 ± 0.05       | 1.94 ± 0.04    | 1.15 ± 0.02    |

Values are adjusted means ± pooled SEM.

The effect of dietary C18:2 was significant, with diets high in C18:2 producing a lower total cholesterol response (3.31 mmol/L) than diets low in C18:2 (3.59 mmol/L).

LDL cholesterol. The normal range of LDL-cholesterol is between 1.70 and 3.00 mmol/L. Throughout the study period normalised HDL-cholesterol values ranged from 1.76 to 2.48 mmol/L, within the low range of normal. Plasma LDL-cholesterol levels were higher (p < 0.0001) when subjects consumed the higher levels of C16:0 (2.18 mmol/L) at the low C18:2 intake level. LDL-cholesterol levels decreased (p > 0.01) at the high levels of C18:2 consumption (1.92 mmol/L) as compared to the lower levels of C18:2 (2.12 mmol/L). Similar to the total cholesterol response, when the diet was high in C18:2 raising the level of C16:0 did not have a significant effect on total plasma LDL-cholesterol levels.

HDL cholesterol. The normal levels of HDL-cholesterol is between 0.90 and 1.60 mmol/L. Throughout the study period, normalised HDL-cholesterol values varied only minimally, ranging from 1.08 to 1.24 mmol/L. Although no significant main effects of diet on HDL-cholesterol levels were observed, at levels of C18:2, high C16:0 raised HDL-cholesterol levels (1.09 mmol/L, vs. 1.24 mmol/L, p < 0.0001). Conversely, at high levels of C18:2, C18:2 levels were raised in HDL-cholesterol levels (1.21 mmol/L vs. 1.08 mmol/L, p < 0.0003).

Figure 1a. Total cholesterol for subjects. Values represent mean ± SEM for all subjects. NS = non-significant (p > 0.05).

Figure 1b. LDL-cholesterol for subjects. Values represent mean ± SEM for all subjects. NS = non-significant (p > 0.05).
The fat was provided by monounsaturated fatty acids. The analysed fat content of meals in each diet treatment was close to the formulated values, and was consistent within each diet treatment.

### Table 3. Lipoprotein cholesterol levels for subjects studied

<table>
<thead>
<tr>
<th>Main effects of diet treatment</th>
<th>Total-cholesterol</th>
<th>LDL-cholesterol</th>
<th>HDL-cholesterol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low C16:0</td>
<td>3.19 ± 0.05</td>
<td>1.99 ± 0.04</td>
<td>1.11 ± 0.02</td>
</tr>
<tr>
<td>High C16:0</td>
<td>3.69 ± 0.05</td>
<td>2.22 ± 0.04</td>
<td>1.19 ± 0.02</td>
</tr>
<tr>
<td>Low C18:2</td>
<td>3.53 ± 0.05</td>
<td>2.08 ± 0.04</td>
<td>1.15 ± 0.02</td>
</tr>
<tr>
<td>High C18:2</td>
<td>3.35 ± 0.05</td>
<td>1.94 ± 0.04</td>
<td>1.15 ± 0.02</td>
</tr>
</tbody>
</table>

Values are means ± SEM. No significant differences were found between treatments. However, a trend for lower total cholesterol values in response to high C16:0 was observed.

### LDL cholesterol

The normal range of LDL-cholesterol is between 1.00 and 1.60 mmol/L. Throughout the study period normalised LDL-cholesterol values varied between 1.76 and 2.48 mmol/L, within the low range of normal. Plasma LDL-cholesterol levels were higher (p<0.001) when subjects consumed the higher level of C16:0 (2.18 mmol/L) at the low C18:2 intake level. LDL-cholesterol levels decreased (p<0.01) at the high levels of C18:2 consumption (1.92 mmol/L) as compared to the lower levels of C18:2 (2.12 mmol/L). Similar to the total cholesterol response, when the diet was high in C18:2 raising the level of C16:0 did not have a significant effect on total plasma LDL-cholesterol levels.

### HDL cholesterol

The normal levels of HDL-cholesterol is between 0.90 and 1.60 mmol/L. Throughout the study period, normalised HDL-cholesterol values varied only minimally, ranging from 1.08 to 1.24 mmol/L. Although no significant main effects of diet on HDL-cholesterol levels were observed, at low levels of C18:2, high C16:0 raised HDL-cholesterol levels (1.09 mmol/L, vs. 1.24 mmol/L (p<0.001). Conversely, at high levels of C18:2 HDL-cholesterol levels were lower in HDL-cholesterol levels (1.21 mmol/L vs. 1.08 mmol/L (p<0.003).

### Fig. 1a. Total cholesterol for subjects. Values represent mean ± SEM for all subjects. NS = non-significant (p>0.05).

### Fig. 1b. LDL-cholesterol for subjects. Values represent mean ± SEM for all subjects. NS = non-significant (p>0.05).
The effect of palm oil on lipid profiles in humans.


Figure 1c. HDL cholesterol for subjects. Values represent mean ± SEM for all subjects. NS = non-significant (p<0.05).

Figure 2. Fractional synthesis rate (FSR) for total cholesterol in subjects consuming the following diets: Low C16:0, Low PUFAs; Low C16:0, High PUFAs; High C16:0, Low PUFAs; High C16:0, High PUFAs. Diet C16:0 diet significantly decreased VLDL cholesterol.

3.17 mmol/L. This data suggests that in the presence of adequate C12:0 palmitic acid has minimal effect on lipoprotein levels.

Fractional synthesis rate (FSR) of diet on cholesterol FSR values is shown in Fig. 2. Values for FSR were not significantly affected by either the C16:0 or C12:0 content of the diet. This data suggests that palmitic acid does not influence endogenous cholesterol synthesis for subjects with normal lipoprotein values. While no significant changes occur in the FSR for cholesterol, it appears that larger FSR values occur in the individual subjects who exhibit a change in serum cholesterol levels in response to diet treatment. This relationship, unlike that of the lipoprotein response, appears to be independent of the dietary linoleic acid level.

Current dietary recommendations suggest no more than 30% of total calories derived from fat. Of this, at least one-third, or 10% of calories, is suggested to be obtained from PUFA. The diets in this study utilized high levels of high medium chain fatty acids. This data suggests that the cholesterol raising potential of palmitic acid is dependent on the linoleic acid level in the diet. This finding may have important applications to the food industry in that the favourable properties of palmitolein in combination with a high saturated fat content can be exploited without expecting adverse effects on serum cholesterol levels.

Acknowledgements. The authors gratefully acknowledge the cooperation of study subjects and acknowledge the excellent technical assistance of I. Polga-Palikai, A. Jakab, and V. Vighoczky. The study was supported by grants from the AARI, INSEEC and the Palm Oil Research Institute of Malaysia.

References.