Original Research

The Chain Length of Dietary Saturated Fatty Acids Affects Human Postprandial Lipemia

Tilakavati Karupaiah, PhD, APD, AN, Choon H. Tan, BSc, Karuthan Chinna, PhD, Kalyana Sundram, PhD

National University of Malaysia, Kuala Lumpur, MALAYSIA (T.K., C.H.T.), University of Malaya, Kuala Lumpur, MALAYSIA (K.C.), Malaysian Palm Oil Council, Kelana Jaya, Selangor, MALAYSIA (K.S.)

Key words: postprandial lipemia, dietary fat, saturated fatty acids, lipoproteins, TAG

Objective: Saturated fats increase total cholesterol (TC) and low density lipoprotein-cholesterol (LDL-C) and are linked to coronary artery disease risk. The effect of variance in chain length of saturated fatty acids (SFA) on coronary artery disease in human postprandial lipemia is not well elucidated.

Methods: A total of 20 healthy volunteers were challenged with 3 test meals, similar in fat content (~31%) but varying in saturated SFA content and polyunsaturated/saturated fatty acid ratios (P/S). The 3 meals were lauric + myristic acid-rich (LM), P/S 0.19; palmitic acid-rich (POL), P/S 0.31; and stearic acid-rich (STE), P/S 0.22. Blood was sampled at fasted baseline and 2, 4, 5, 6, and 8 hours. Plasma lipids (triaclylglycerol (TAG) and lipoproteins (TC, LDL-C) high density lipoprotein-cholesterol (HDL-C) were evaluated.

Results: Varying SFA in the test meal significantly impacted postprandial TAG response (p < 0.05). Plasma TAG peaked at 5 hours for STE, 4 hours for POL, and 2 hours for LM test meals. Area-under-the-curve (AUC) for plasma TAG was increased significantly after STE treatment (STE > LM by 32.2%, p = 0.003; STE > POL by 27.9%, p = 0.023) but was not significantly different between POL and LM (POL > LM by 6.9%, p > 0.05). At 2 hours, plasma HDL-C increased significantly after the LM and POL test meals compared with STE (p < 0.05). In comparison to the STE test meal, HDL-C AUC was elevated 14.0% (p = 0.005) and 7.6% (p = 0.023) by the LM and POL test meals, respectively. The TC response was also increased significantly by LM compared with both POL and STE test meals (p < 0.05).

Conclusions: Chain length of saturates clearly mediated postmeal plasma TAG and HDL-C changes.

INTRODUCTION

A lower fat consumption with reduction in saturated fatty acid (SFA) content of the total diet has been an important strategy for the past 5 decades of cardiovascular disease (CVD) risk management associated with hypercholesterolemia [1-3]. Dietary fatty acid type is well established to affect total cholesterol (TC) with parallel changes in low density lipoprotein-cholesterol (LDL-C) [4,5] and is in agreement with predictive coefficients [4,6,7]. A meta-analysis of the National Cholesterol Education Program (NCEP) Step II diet studies incorporating <7% SFA calories have reported an achievable 16% decrease in LDL-C level [8].

A counterpoint to SFA reduction is an effect causing a reduction in high density lipoprotein-cholesterol (HDL-C) concentration. Three randomized controlled trials in humans have shown that diets providing 6%–7% SFA calories produced a 9%–11% reduction in LDL-C accompanied by a 7%–11% reduction in HDL-C [9-11]. Of these, a 9% increase in plasma triacylglycerol (TAG) was observed in one study [10]. Consuming ~4% SFA calories reported by one randomized controlled trial achieved an 8.6% reduction in LDL-C and a 10% reduction in HDL-C [12]. The Delta, OmniHeart Randomized Trial, Dietary Alternatives, and beFIT studies have demonstrated that limiting SFA calories by...
Dietary Saturate Type Affects Postprandial Lipemia

exchanging with carbohydrates increased plasma TAG and decreased HDL-C [11,13-15].

Atherogenic potential of SFA is not uniform as at a high fat energy intake, myristic acid (C14:0) is the most hypercholes-
terolemic, with a suggested potency 3-6 times greater than
lauryc (C12:0) or palmitic (C16:0) acids, whereas stearic acid
(C18:0) has been suggested to be neutral [16,17]. The
TC:HDL-C ratio has been shown in a meta-analysis by
Mensink et al. [7] to decrease with increasing saturation from
C12:0- to C18:0-rich diets. A recent systematic review
indicated that when C18:0 was substituted for other SFA,
LDL-C decreased but remained unchanged when substituted
for carbohydrate [18]. A proposed recommendation of the 2010
DGAC [19] was to support C18:0-rich foods, such as cocoa
butter (dark chocolate) or shea nut oil, as "not problematic"
compared with other types of SFA-rich vegetable oils.
However, the 2010 DGAC report [19] also concluded that
the impact of substituting C18:0 for other energy sources was
variable regarding LDL-C, and the potential impact of changes
in C18:0 intake on CVD risk remained unclear.

Since 2000 the NCEP Adult Treatment Panel III guidelines
through the Therapeutic Lifestyle Changes have promoted
global risk management for CVD to target hypertriglyceride-
emia, thrombogenesis, and inflammation while achieving low
TC and LDL-C [20,21]. Hypertriglyceridemia and low HDL-C
are now identified as new risks for coronary artery disease in
association with "low metabolic capacity." The dyslipidemic
environment is prone to prolonged postprandial lipemia, which
is associated with unfavorable hemostatic dynamics. Impaired
reverse cholesterol transport is now identified as a source of
inflammatory and thrombogenic triacylglycerol-rich lipopro-
teins (TRL) rising from absorption of dietary lipids [22,23].
Studies that evaluate the contributory role of fat to postmeal
hypertriglyceridemia are limited [24,25]. One study by Kralova
et al. [25] questioned whether reduced HDL-C on a high
polyunsaturated fatty acids (PUFA)/low SFA diet affected
reverse cholesterol transport negatively but did not find any
difference in cholesterol efflux. Given this new era of global
risk management in CVD, we undertook to compare the effects
of different SFAs on postprandial lipemia in normocholes-
terolemic subjects.

MATERIALS AND METHODS

Subjects

A total of 20 normolipemic healthy subjects, 10 men and 10
women aged between 22 to 38 years without a history of
atherosclerotic disease or hypertension were recruited into the
study. Female subjects were nonpregnant and nonlactating, and
none of the subjects were on any prescribed medication.
Subjects were nonsmokers, consumed no alcohol, and did not
take any nutritional supplements or participate in weight-loss
programs immediately prior to or during the study. Addition-
ally, female subjects ceased use of oral contraceptives. Subjects
were thoroughly briefed on the study protocol and gave their
written informed consent for participation in the study. They
had the freedom to drop out from the study at any time.
Subjects participated in an initial screening and their baseline
characteristics were as follows: age, 30.1 ± 8.03 years; body
mass index (BMI), 22.5 ± 4.40 kg/m²; TC, 4.97 ± 0.58 mmol/L;
TAG, 0.94 ± 0.32 mmol/L; LDL-C, 2.96 ± 0.54 mmol/L;
and HDL-C, 1.58 ± 0.31 mmol/L.

Study Design

The study was designed to evaluate postprandially the effect
of varying chain length saturation of dietary fats on lipids and
lipoproteins. Blending coconut oil with corn oil provided a
lauryc + myristic-rich (LM) fat with a polyunsaturated to
saturated fatty acid ratio (P/S) of 0.19, whereas a stearate-rich
fat (STE, P/S = 0.22) was obtained by blending cocoa butter
with corn oil. In contrast, palm olein on its own provided a
predominantly palmitic acid-rich fat (POL, P/S = 0.31).
Formulated test fats were rotated in a randomized crossover
design, with all subjects consuming experimental diets
containing a test fat for 7 days prior to a postprandial challenge
on the morning of the eighth day. A 7-day washout separated
test fat rotations. In each test-fat rotation, subjects were
assigned to 2 groups of 10 each to optimize the proper and
correct handling of blood specimens within the time constraints
imposed by the study design. Female subjects were assigned
between menses to the experimental groups. Subjects were
instructed to eat according to their usual individual food intake
patterns and were blinded to the test fats used for each rotation.
Body weight measurements were recorded before each
postprandial challenge to ensure weight fluctuations were
minimized between the test rotations. This study protocol was
approved by the institutional ethics committee.

Test Meals

A standard 7-day menu plan was adopted for all test fat
rotations. The test meals were eucaloric and differed only in the
composition of the incorporated test fats. This was confirmed
by actual fatty acid composition (FAC) of the test fats and
duplicated portions of test meals as consumed by the volunteers
(Table 1). All meals were eucaloric in their macronutrient
composition but differed in their FAC. The major SFA in the
test meals reflected the test fats, as evidenced by the content
(%) of C16:0 in the POL diet, C12:0 and C14:0 in the LM diet,
and C18:0 in the STE diet. The percentage linoleic acid (C18:2)
in terms of energy content across all test fats (~11% en) and
test meals (~3% en) was adjusted to be similar and to yield
overall low P/S ratios (0.19-0.31). The daily menu during the
7-day run-in period provided approximately 50 g of the test fat
in the diet, equivalent to ~26% en. Thus total daily fat content
Table 1. Fatty Acid Composition of the Test Fats and Test Meals*

<table>
<thead>
<tr>
<th>Fatty Acids</th>
<th>Test Fats (% Total Fat)</th>
<th>Test Meals (% Total en)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Test Fats</td>
<td>POL</td>
<td>LM</td>
</tr>
<tr>
<td>Total SFA</td>
<td>44.05</td>
<td>74.74</td>
</tr>
<tr>
<td>C8:0</td>
<td>nd</td>
<td>4.71</td>
</tr>
<tr>
<td>C10:0</td>
<td>nd</td>
<td>3.96</td>
</tr>
<tr>
<td>C12:0</td>
<td>0.23</td>
<td>40.83</td>
</tr>
<tr>
<td>C14:0</td>
<td>0.9</td>
<td>14.15</td>
</tr>
<tr>
<td>C16:0</td>
<td>38.82</td>
<td>8.91</td>
</tr>
<tr>
<td>C18:0</td>
<td>4.1</td>
<td>2.18</td>
</tr>
<tr>
<td>MUFA</td>
<td>44.45</td>
<td>20.93</td>
</tr>
<tr>
<td>C18:1</td>
<td>43.58</td>
<td>20.09</td>
</tr>
<tr>
<td>PUF A</td>
<td>11.58</td>
<td>11.67</td>
</tr>
<tr>
<td>C18:3</td>
<td>11.23</td>
<td>11.44</td>
</tr>
<tr>
<td>P/S ratio</td>
<td>0.35</td>
<td>0.23</td>
</tr>
<tr>
<td>Test Meals</td>
<td>0.26</td>
<td>0.16</td>
</tr>
</tbody>
</table>

*POL = palm olein only, LM = lauric + myristic acid-rich oil obtained from blending coconut and corn oil, STE = stearic acid oil obtained by blending cocoa butter with corn oil, SFA = saturated fatty acid, MUFA = monounsaturated fatty acid, PUF A = polyunsaturated fatty acid, nd = not detectable, P/S ratio = polyunsaturated / saturated fatty acid ratio.

1 % total en = percentage energy of total fat in the meal.

of test meals provided during each of these periods was maintained at 31% en with the remainder (~5% en) coming from dietary sources of invisible fats.

The menu used typical Malaysian recipes and was constructed according to the following meal plan: (1) for breakfast, a cereal dish and a snack item cooked with the test fat was served with either coffee or tea, (2) lunch included fish or chicken and two vegetables cooked with the test fat and accompanied by rice and fruits, and (3) for high tea, a snack item with the test fat incorporated was served with either tea or coffee. All meals were prepared by a trained caterer and standardized to recipes and portion sizes. Procedures for incorporating the test fats were supervised during meal preparations by a diettian. To maximize compliance, subjects were provided with the test fats for preparation of dinner and for weekend meals at home. On the eighth morning, subjects were challenged with the postprandial test meals.

Postprandial Challenge

Subjects were advised to refrain from strenuous physical activity for 24 hours preceding each postprandial event. They reported to the laboratory at 0800 hours on the day assigned for the postprandial fat challenge after an overnight minimum fast of 10 hours. A 12-ml fasted venous blood sample was obtained from each subject. Subjects then consumed the postprandial meal challenge containing the test fat within 15 minutes of drawing the fasted blood sample. The postprandial meal challenge consisted of fried rice and a breaded snack using the test fat specific to the test rotation and as described in Table 1. This meal consisted of 275 g of fried rice with 2 portions of breaded snack that provided approximately 960 kcal, 50 g fat (47% en), 98 g carbohydrate (41% en), and 29 g protein (12% en). Plain tea or coffee was allowed with the test meal. Chemical analyses of randomized samples of postprandial test meal challenges confirmed a mean fat content of 47.38 ± 11.63 g. Only the consumption of mineral water was allowed ad libitum for the ensuing 8 hours of the postprandial period. On completion of the postprandial sessions, subjects were provided with a cooked meal. Subjects remained in a rested state during the 8 hours of the postprandial challenge.

Twelve-ml blood samples were drawn by venous puncture from fasting subjects at 0 hours (i.e., before the meal challenge) and subsequently at 2, 4, 5, 6, and 8 hours after the consumption of the test-fat challenges. The same sequence of blood sampling for all timed events was followed during each postprandial challenge. Blood sampling at each time point was completed for all subjects within 15 minutes of beginning the session.

Blood Sampling and Biochemical Determination

Blood was collected into Vacutainer* tubes (Becton Dickinson Vacutainer, Franklin Lakes, NJ) containing EDTA (0.117 ml of 15% EDTA) and immediately centrifuged at 3000 g for 20 minutes at 4°C (Sigma 3K12, B. Braun, Tuttingen, Germany) to separate the plasma from red blood cells. Fresh plasma was reserved for ultracentrifugation, whereas the remaining plasma was aliquoted and snap-frozen in liquid nitrogen and stored at ~80°C for subsequent analyses.

Isolation of TRLs

Ultracentrifugation of fresh EDTA plasma to extract TRL-rich chylomicrons was carried out in sealed Beckman Quick-Seal® polylamellar tubes (Beckman Instruments Inc., Palo Alto, CA) as per procedures of the Lipid Research Clinics Programs of the National Institutes of Health/National Heart and Lung Institute in the United States [26]. Each Quick-Seal® tube was set upright in a holder and a long-tipped Pasteur pipette was introduced through the narrow neck of the tube. The Pasteur pipette served as the conduit for addition of 0.9% sodium chloride (NaCl) (specific density, d = 1.006) and plasma. About 3.5 ml of NaCl was delivered into the Quick-Seal® tube without introducing air bubbles. Then, 3 ml of EDTA plasma was layered under NaCl, without introducing air bubbles. The Pasteur pipette was carefully withdrawn without disturbing the plasma and NaCl phases. The final volume in the Quick-Seal® tube was topped with NaCl using a fine-bore 1.0-ml syringe before sealing the neck of the tube with a tube sealer (Beckman Instruments).

Sealed and labeled Quick-Seal® tubes were ultracentrifuged for 18 hours at 47,000g at 12°C with a type 50.3 Ti fixed angle
Dietary Saturate Type Affects Postprandial Lipemia

rotor (Beckman Instruments). At the end of ultracentrifugation, Quick-Seal® tubes were sliced at the point of sealing, and aliquots were removed in sequence. First, a precise 2.0-ml aliquot from the top fraction yielding TRL (d < 1.006 g/ml) was transferred by aspiration with a Pasteur pipette into a clean test tube for storage at −4°C. This fraction was reserved for further analysis. The remaining bottom fraction was recovered separately and made up to a final volume of 3.0 ml with NaCl solution (d > 1.006 g/ml). This fraction was subsequently used to determine HDL-C concentration (d = 1.063 g/ml) as well as to characterize plasma cholesterol ester (CE) FAC.

Lipids and Lipoproteins

The TC in plasma and cholesterol content of TRL and bottom fractions of ultracentrifuged plasma were determined by enzymatic procedures [27]. Plasma TAG was also similarly determined [28]. A precise 35-μl volume of dextran sulfate-Mg2+ precipitating agent was added to 350 μl of the bottom fraction, the mixture vortexed and the reaction timed for 10 minutes to precipitate LDL [29,30]. The clear supernatant, obtained through centrifugation for 10 minutes at 7000g (Speedufge HSC10K, Savant Instruments Inc., Farmingdale, NY), was used to determine HDL-C content. All assays were performed using a Ciba-Corning 550 Express Autoanalyzer (Ciba-Corning Diagnostics Corp., Oberlin, OH). Reagents, calibrators, and controls were supplied by Bayer Corp. (Tarrytown, NY), and the HDL-C precipitant was supplied by Chiron Diagnostics Corp. (Emeryville, CA). Plasma LDL-C was calculated by the differences between cholesteryl content of the bottom fraction of ultracentrifuged plasma and HDL-C [26,31].

Fatty Acid Composition

Two milliliters of the upper fraction of ultracentrifuged plasma containing TRL were used for lipids extraction by the Folch method [32]. The CE were similarly extracted from the bottom fraction of ultracentrifuged plasma samples [32]. Extracted lipids were then subjected to thin-layer chromatographic separation with a mixed solvent phase of hexane, diethyl ether, and acetic acid (80:20:2). Lipids from double-portioned meals were obtained by Soxhlet extraction [33]. Extracted lipids from TRL, CE, and test meals were converted into fatty acid methyl esters, dried under nitrogen, and reconstituted with hexane before injection into the gas chromatographer (Perkin-Elmer Autosystem, Perkin-Elmer, Norwalk, CT). The FAC of TRL, CE, and test meals were expressed as percentage composition (by weight), calculated by reference to authentic standards [33]. The FAC of the meals consumed by the subjects served to check whether the test meals achieved targeted FAC, whereas the FAC of subjects’ plasma served to check compliance to the study protocol.

Statistical Analyses

The crossover design enabled every subject to serve as his or her own control; all 20 subjects completed the 3 test-meal rotations. The Statistical Package for Social Sciences, SPSS® for Windows™ application (Version 15.0, SPSS Inc., Chicago, IL.) was used for the required statistical analyses. Differences between outcomes from the various postprandial time intervals and baseline values (0 hour) were interpreted as true measures of change resulting from dietary treatment. Multivariate analyses for repeated measures (MANOVA), using the general linear model, was performed for all time × test meal values for each measurement parameter. Univariate analysis was used to compare the area-under-the-curve (AUC) derived for the 8-hour duration of the postprandial period calculated by the trapezoidal rule [34]. The Levene test was used to examine equality of variances across treatment groups. Bonferroni adjustment for multiple paired comparisons was used to test mean differences between groups. Significance was set at p < 0.05 for all evaluated measures.

RESULTS

Subject Demographics at Baseline

All 20 subjects successfully completed the 3 test rotations. A nonsignificant increase in body weight of 0.62 ± 0.78 kg from baseline to study completion was evident.

Lipemic Response and Test Fat Clearance

Changes in plasma TAG (mean ± SD) occurring over timed intervals in response to 3 postprandial test fat challenges are presented in Fig. 1a,b. Dietary saturates caused highly significant (p = 0.002) time × meal treatment changes to plasma TAG concentrations during the 8-hour postprandial period (Fig. 1a). The magnitude and extent of plasma TAG response was greatest after feeding the C18:0-rich STE fat challenge and attained significance in comparison to the C16:0-rich POL (p = 0.023) and C12:0+C14:0-rich LM (p = 0.003) meals in pairwise comparisons of the estimated marginal means. AUC confirmed that the variable effect of the dietary saturates was significantly different (p = 0.016) and greatest for the STE treatment (STE > LM by 32.2%, STE > POL by 27.9%), versus the comparison between the POL and LM test meals (POL > LM by 6.0%) (Fig. 1b). The effect was significant from 2 hours onward (p < 0.05), and differences in lipemic clearance was evident, with plasma TAG peaking at 5 hours for the C18:0 meal, 4 hours for the C16:0 meal, and 2 hours for C12:0+C14:0 meal, indicating fat clearance was in the order of C12:0+C14:0 > C16:0 > C18:0. It was further observed that lipemic response generated by STE meal did not resume postabsorptive levels by 8 hours, unlike responses after the POL and LM treatments.
Dietary Saturate Type Affects Postprandial Lipemia

Plasma TC, LDL-C, and HDL-C

Changes in plasma lipoprotein concentrations occurring over time in response to the 3 postprandial test fat challenges (time × diet interactions) are presented in Figs. 2a,b; 3a,b; and 4a,b. Postprandial TC responses were not significantly affected by the type of saturates tested (Fig. 2a) except for a time effect from 5 to 8 hours (p = 0.026) for all meals, with a significantly higher increase after the LM challenge compared with the POL and STE treatments (p = 0.033). TC response after POL and STE treatments was not significantly different. Overall, AUC for TC did not result in any significance between different saturates (Fig. 2b).

Initial HDL-C concentrations decreased at baseline for all meals, but after 2 hours an upward trend was observed for the LM and POL test meals but not for the STE meal (Fig. 3a). Treatment effect became significant (p = 0.003) after correcting for baseline values as well as for pairwise comparisons of estimated marginal means between the STE and POL treatments (p = 0.023) and for the STE and LM treatments (p = 0.005). Differences between the LM and POL treatments were not significant. AUC was not significantly different from baseline (Fig. 3b). Percentage increase in AUC plasma HDL-C was 14.0% greater for the LM compared with the STE treatments (p = 0.005) and 7.6% between the POL and STE treatments (p = 0.023).

LDL-C concentrations for all saturates were elevated toward the end of the measured postprandial event, but this elevation was not significantly different between treatments (Fig. 4a). AUC for LDL-C response also did not achieve significance between the dietary saturates tested (Fig. 4b).

FAC Patterns of TRL and CE

The effect of time was not significant after correction for the baseline values for individual fatty acids in both TRL and CE. Fig. 5a-d indicates the distribution of individual fatty acids (mean ± SE) in TRL, expressed as a percentage of the FAC. A

Fig. 1. (a) Plasma TAG (mean ± SEM, n = 20) concentrations over 8 hours in response to test meals containing POL, LM, or STE oil type. Repeated measures MANOVA analyzed for time × test meal interactions was significant (p = 0.002). The lipemic trend generated by the C18:0-rich STE meal was greater than that of the C16:0-rich POL (p = 0.023) or LM (p = 0.003) test meals. (b) Univariate analyses on AUC for plasma TAG was significantly higher for the STE test meal (p = 0.016) compared with the LM (>32.2%) and POL (>27.9%) test meals, whereas the difference between the POL and LM test meals (6.0%) was not significant. POL = palm olein only (●), LM = lauric + myristic acid-rich oil obtained from blending coconut and corn oils (■), STE = stearic acid-rich oil obtained by blending cocoa butter with corn oil (▲).

Fig. 2. (a) Plasma TC response (mean ± SEM, n = 20) over 8 hours following a standardized fat load with the POL, LM, or STE test meals. Repeated measures MANOVA analysis for time × test meal interactions was not significant (p < 0.05). An effect of time was significant from 5 to 8 hours (p = 0.026) with significantly greater plasma TC response generated by the LM compared with the POL and STE test meals (p = 0.033). (b) Univariate analyses for AUC of plasma TC was not significantly different. POL = palm olein only (●), LM = lauric + myristic acid-rich oil obtained from blending coconut and corn oils (■), STE = stearic acid-rich oil obtained by blending cocoa butter with corn oil (▲).
Dietary Saturated Type Affects Postprandial Lipemia

Fig. 3. (a) Plasma HDL-C (mean ± SEM, n = 20) concentrations over 8 hours in response to test meals containing POL, LM, or STE test fats. Repeated measures MANOVA analyzed for time × test meal interactions was significant (p = 0.003) and comparisons between meals was significant between the STE and POL (p = 0.023) meals and the STE and LM (p = 0.005) meals but not between the LM and POL meals. (b) AUC for plasma HDL-C (mean ± SEM, n = 20) as analyzed by univariate analysis was not significantly different from baseline except at 2 hours (p = 0.055), for which the STE meal was 14.0% greater compared with the LM (p = 0.005) and 7.6% greater compared with the POL (p = 0.023) meals. POL = palm olein only (●), LM = lauric + myristic acid-rich oil obtained from blending coconut and corn oils (■), STE = stearic acid-rich oil obtained by blending cocoa butter with corn oil (▲).

Fig. 4. (a) Plasma LDL-C (mean ± SEM, n = 20) concentrations over 8 hours in response to test meals containing POL, LM, or STE test fats. Repeated measures MANOVA analyzed for time × test meal interactions were not significant (p > 0.05). (b) No significant difference for plasma LDL-C AUC (mean ± SEM, n = 20) was noted between test meals (p > 0.05). POL = palm olein only (●), LM = lauric + myristic acid-rich oil obtained from blending coconut and corn oils (■), STE = stearic acid-rich oil obtained by blending cocoa butter with corn oil (▲).

significant effect of dietary treatment (p < 0.05) was evident for lauric + myristic (C12:0+C14:0), palmitic (C16:0), stearic (C18:0), and linoleic (C18:2) acids in the composition of TRL. C12:0+C14:0 in TRL was significantly higher after LM treatment (p < 0.001) compared with the POL and STE treatments, an effect attributed to their dietary availability (LM = 40.0%; POL = 2.9%; STE = 2.4%). Incorporation of C16:0 into TRL was greatest after the POL test meal compared with the LM and STE meals (p < 0.001) as expected due to its greater dietary availability (POL = 34.5%; LM = 14.8%; STE = 25.3%), but surprisingly, despite the lowest content of C16:0 in the LM meal, incorporation into TRL was not significantly different between the STE and LM meals. It was noted that percentage C16:1n7 was not significantly different (p > 0.05) between diets (data not shown). Percentage C18:0 in TRL was significantly (p < 0.001) related to dietary availability (POL = 4.9%; LM = 4.3%; STE = 23.9%) with the greatest incorporation after the STE treatment and minimal after the POL and LM meals. C18:1 in TRL (p < 0.001) reflected dietary source (POL = 43.6%; LM = 20.1%; STE = 33.6%), and incorporation appeared to be dose-dependent with POL > LM (p < 0.001), POL > STE (p < 0.001), and STE > LM (p = 0.024). Presence of C18:2 in TRL was significantly higher after the LM treatment compared with the POL (p < 0.001) and STE (p < 0.001) treatments, and this was independent of the C18:2 content of the meals (POL = 12.5%; LM = 12.6%; STE = 10.7%). Though data are not shown here, percentage C18:1 was significantly affected by test meal treatment (p < 0.05) in the order of POL > STE > LM.

Individual fatty acid concentrations in CE as percentage FAC, in response to the various SFA test meals are presented in Fig. 6a-d. Significant differences in the CE fatty acids after correction for baseline values were evident for C12:0+C14:0 (p < 0.001), C16:0 (p = 0.001), C18:0 (p < 0.001), and C18:1 (p < 0.001). The appearance of these fatty acids in CE reflected meal source but were independent of test meal × time interactions.
Dietary Saturated Type Affects Postprandial Lipemia

A potential novelty of this study is that plasma fatty acids of TRL and CE fractions isolated from chylomicrons and plasma, respectively, have been reported for a postprandial study. Their fatty acid patterns clearly are traced back to the FAC of the dietary fats used in the postprandial challenges. These, therefore, may assist in enhancing our understanding of the dynamics of different dietary fatty acids during fat metabolism. Differences arising from postprandial response to the various fats tested in this study are directly attributed to the chain length of dietary saturates tested. The FAC of test fats and their incorporation into the respective test meals reflected the highest contents of C16:0 (34.5%) in POL, C12:0+C14:0 (40.0%) in LM, and C18:0 (24.0%) in STE meals, respectively. This was anticipated because palm olein as the POL test fat is a rich source of C16:0 and coconut oil provided the higher C12:0+C14:0 in the LM test fat, whereas the higher C18:0 content of the STE test fat was provided by cocoa butter. Corn oil was added to the LM and STE test meals to equalize the PUFA content among all meals. Only the MUFA content of the test meals differed.

The postprandial response of plasma TAG to dietary saturates was affected by the predominant SFA in the test meals and indicated a trend based on the carbon chain length of the fatty acids tested. Magnitude of lipemic response was greatly enhanced by the C18:0-rich STE meal and in the following order, C18:0 > C16:0 > C12:0+C14:0. Lipemia associated with the C18:0-rich STE meal was enhanced and prolonged for the postprandial duration of 8 hours compared with the C12:0+C14:0-rich LM meal and the C16:0-rich POL meal. Preferential fat clearance has been noted between fat classes such as PUFAs, MUFAs, and SFAs but not within a SFA class. Both Zampelas et al. [35] and Demacker et al. [36] attributed a slower return to the postabsorptive state to long-chain SFAs compared with PUFAs. Thomsen et al. [37] found TAG in the chylomicron-rich fraction of plasma to rise 2.5-fold to fivefold on a butter-rich meal compared with a corn-oil-rich meal. In contrast Mekki et al. [38] found a lower lipemia and chylomicron accumulation on a butter-rich meal compared with

(Fig. 5. Continued.)

(p < 0.05), whereas comparisons between LM and STE were not significantly different (p > 0.05). (c) Percentage C18:0 increased significantly after the STE meal compared with the POL and LM meals (p < 0.05), whereas comparisons between POL and LM were not significantly different (p > 0.05). (d) Percentage C18:1 increased significantly after the POL meal compared with the STE and LM meals (p < 0.05), whereas comparisons between STE and LM were not significantly different (p > 0.05). POL = palm olein only (●), LM = lauric + myristic acid-rich oil obtained from blending coconut and corn oils (●), STE = stearic acid-rich oil obtained by blending cocoa butter with corn oil (▲).
Dietary Saturate Type Affects Postprandial Lipemia

Fig. 6. Fatty acid composition (FAC) of plasma cholesteryl esters (CE) (mean % ± SEM, n = 20) in response to test meals containing POL, LM, or STE test fats. Statistical analyses were corrected for baseline values and significance reported at p < 0.05 for time × test meal interactions. (a) Percentage C12:0+C14:0 increased significantly after the LM meal compared with the POL and STE meals (p < 0.05), whereas comparisons between POL and STE were not significantly different (p > 0.05). (b) Percentage C16:0 increased significantly after the POL meal compared with the LM and STE meals (p < 0.05), whereas comparisons between LM and STE were not significantly different (p > 0.05). (c) Percentage C18:0 increased significantly after the STE meal compared with the POL and LM meals (p < 0.05), whereas comparisons between POL and LM were not significantly different (p > 0.05). (d) Percentage C18:1 was significantly greater after the POL and STE meals compared with the LM meal (p < 0.05), whereas comparisons between POL and STE were not significantly different (p > 0.05). POL = palm olein only (●), LM = lauric + myristic acid-rich oil obtained from blending coconut and corn oils (○), STE = stearic acid-rich oil obtained by blending cocoa butter with corn oil (▲).

olive-oil-rich and sunflower-oil-rich meals. Asakura et al. [39] also found a lower lipemia associated with medium-chain SFA-rich compared with an n-6 PUFArich fat challenges.

Plasma TAG clearance appeared to be affected by chain length of the dietary saturates tested. Peaking time was earlier with the LM meal at 2 hours, slowest with the STE meal at 5 hours, and 4 hours with the POL meal. The particle size of TRL-rich chylomicrons is hypothesized to affect postprandial fat clearance. Unsaturated fatty acids reportedly increase particle size compared with SFAs [40,41]. Circulating chylomicrons were smaller after a butter-rich meal compared with PUFA- and MUFA-rich meals [38]. It is, therefore, possible that the carbon-chain length of the different saturates tested in this study affected particle size and number of chylomicrons during lipemia. This may perhaps explain the association of the slowest clearance after the C18:0-rich meal and the rapid clearance after the C12:0+C14:0-rich LM meal. Preferential hydrolysis by lipoprotein lipase may also be linked to the differences in fat clearance seen for the different saturates tested [42,43].

A prolonged and higher lipemia is conducive to a prothrombotic state due to the exaggerated and prolonged presence of TAG-rich TRL in the circulation [23,24]. Postprandial lipemia has been shown to increase levels of factor VIIa (FVIIa) and plasminogen activator inhibitor 1 (PAI-1) [44,45]. Activation of factor VII (FVII) has been observed in healthy adults and in those with congenital deficiency of either factors XI or XII after a high-fat meal [46]. However the contributory effects of fat composition to thrombogenicity is unclear at present, given that mixed findings have been reported. Feeding olive oil lowered FVIIa response compared with either rapeseed or sunflower oils, but associated lipemia was significantly higher for olive oil. Sanders et al. [47] compared randomized and unrandomized cocoa butter in healthy subjects and found that unrandomized cocoa butter, rich with C18:1 in the sn-2 position, was absorbed more rapidly than randomized cocoa butter, which predominated in C18:0 in the sn-2 position. This, they concluded, led to increased activation of FVII associated with the unrandomized cocoa butter. A randomized control trial (n = 96) found FVIIa response to a fat-rich meal was independent of its fatty acid
composition, and not influenced by the different degree of lipemia of the C18:2/18:3 rich meal compared with C16:0 and C18:0 [48].

HDL-C response in this study was also influenced by chain-length of the dietary saturates, which increased with decreasing SFA chain length in the order of C12:0+C14:0 > C16:0 > C18:0. The postprandial HDL-C response saw an increase after the LM and POL diets but remained depressed for the STE meal. The significant change in HDL-C concentration that was observed for the LM and POL meals occurred 2 hours after the postprandial meal challenge. The postprandial trend observed for HDL-C may be linked to the differences in fat clearance that occurred as a result of the different saturates. Only Thomsen et al. [37] have reported a difference in HDL-C response when comparing olive oil and butter. In their study a higher HDL-C response associated with the olive oil meal was in tandem with a faster fat clearance. It remains to be explored whether differences seen in postprandial HDL-C response attributed to different saturates are linked to fat clearance and whether this is mediated through reverse cholesterol transport.

CONCLUSION

Individual dietary saturates, as this study demonstrated, variably affected postprandial plasma TAG and lipoproteins. The nature of dietary saturates clearly influenced fat digestion and absorption in the subjects. The magnitude of lipemic response was greatest for a stearic acid-rich (C18:0) meal and least for a lauric + myristic-rich (C12:0+C14:0) meal. Similarly the HDL-C response was affected by the chain length of saturates, albeit inversely. HDL-C concentrations were highest with the C12:0+C14:0-rich meal and decreased with increasing carbon-chain length of the fatty acid tested. In contrast, the responses elicited by the palmitic acid-rich (C16:0) meal, on lipemia and HDL-C, were moderate compared with LM and STE.

ACKNOWLEDGMENTS

The contribution of each author is as follows: T.K., an accredited practicing dietitian, was responsible for data acquisition and drafting of this manuscript; C.H.T. was a graduate student participating in the implementation of the study; K.C. was responsible for statistical analyses and interpretation of data; K.S. made substantial contributions to conception and design of the study and finalization of the manuscript. None of the authors have a conflict of interest to declare. This research protocol was approved by the Ethical Approval Committee of the National University of Malaysia with the registration number FF-165-2007.

REFERENCES

Dietary Saturate Type Affects Postprandial Lipemia


Dietary Saturate Type Affects Postprandial Lipemia


Received December 6, 2010; revision accepted October 6, 2011.