Butter Differs from Olive Oil and Sunflower Oil in Its Effects on Postprandial Lipemia and Triacylglycerol-Rich Lipoproteins after Single Mixed Meals in Healthy Young Men

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ABSTRACT Accumulation of postprandial triacylglycerol-rich lipoproteins is generated by assimilation of ingested dietary fat and has been increasingly related to atherogenic risk. Nevertheless, the influence of different kinds of dietary fatty acids on postprandial lipid metabolism is not well established, except for (n-3) polyunsaturated long-chain fatty acids. Our goal was to evaluate the effects of test meals containing a common edible fat source of saturated (butter), monounsaturated (olive oil) or (n-6) polyunsaturated (sunflower oil) fatty acids on postprandial lipid and triacylglycerol-rich lipoprotein responses. After a 12-h fast, 10 healthy young men ingested mixed meals containing 0 g (control) or 40 g fat, provided as butter, olive oil or sunflower oil in a random order. Fasting and postmeal blood samples were collected for 7 h. The no-fat test meal did not elicit any change over baseline except for plasma phospholipids, insulin and nonesterified fatty acids. Conversely, the three fat-containing meals elicited bell-shaped postprandial changes (P < 0.05) in serum triacylglycerols, free and esterified cholesterol, and nonesterified fatty acids. The butter meal induced a lower postprandial rise of triacylglycerols in serum and chylomicrons (incremental AUC, mmol·h/L: 0.72) than the two unsaturated oils (olive oil: 1.6, sunflower oil: 1.8), which did not differ. Circulating chylomicrons were smaller after the butter meal than after the two vegetable oil meals. The in vitro susceptibility of circulating chylomicrons to hydrolysis by postheparin plasma was higher after sunflower oil than after butter or olive oil. We conclude that butter results in lower postprandial lipemia and chylomicron accumulation in the circulation of young men than olive or sunflower oils after consumption of a single mixed meal. J. Nutr. 132: 3642-3649, 2002.

KEY WORDS: dietary fat • monounsaturated fatty acids • polyunsaturated fatty acids • saturated fatty acids • chylomicrons

Given usual dietary intake patterns, most individuals are in a postprandial hypertriglyceridemic state for the greatest part of every 24-h period (1,2). This results from dietary fat intake, efficient emulsification and hydrolysis of fat in the gut, intestinal absorption, intracellular lipid resynthesis and packaging with apoproteins, and finally, resecretion of lipid moieties in the circulation in the form of triacylglycerol-rich lipoproteins (TRL), primarily CM (CM). The hypertriglyceridemic state observed for hours postprandially is, thus, due to the accumulation of TRL particles in the circulation (3) from endogenous (hepatic VLDL) or exogenous (CM) origin, which are competing for hydrolysis by endovascular lipases and subsequent removal by peripheral tissues and the liver. In addition to other factors (sex, gene polymorphisms, dyslipoproteinemia, obesity, diabetes, and coronary heart disease), postprandial lipemia has been shown to be influenced by dietary variables such as the amount of dietary fat (1), cholesterol (4) or carbohydrate (5).

The adverse or beneficial effects of chronic intake of particular fatty acids on the cholesterol profile of food-deprived subjects (6) and risk for coronary heart disease (7) have been fully documented and serve as a basis for modern dietary recommendations. More recently, evidence has been obtained that hypertriglyceridemia due to TRL accumulation is an independent risk for coronary heart disease (8). This gives more weight to the possibility that atherosclerosis could be, at least partly, a postprandial phenomenon (9,10).

That the nature of ingested dietary fat can modulate the postprandial lipid and lipoprotein responses has been investi-
DIETARY FAT AND POSTPRANDIAL LIPEMIA

3643

gated for a decade, although a limited number of studies in humans and laboratory animals have been published as reviewed (2). Most studies with (n-3) PUFA-rich fats showed a reduced postprandial lipemia when ingested in the daily diet or as a single meal (2,11) compared with other fat sources. Comparisons of the effects of (n-6) (PUFA)-rich oils with olive oil (n-9) monounsaturated fatty acids or MUFA showed lower (12) or comparable (13,14) magnitudes of postprandial lipemia. Published studies dealing with saturated fatty acids (SFA) need careful evaluation because of the use of either dairy fat in the form of cream, milks, shakes or butter, other animal fat (beef tallow) or saturated vegetable fat (coconut oil, cocoa butter and palm oil). Most studies in which dairy fat or mixed SFA-rich vegetable oils were used did not generally show increased postprandial responses compared with PUFA-rich oils (2,14) after single meals. Conversely, a recent study showed an exacerbated chylomicron response after a butter meal compared with an olive oil one (15).

On the whole, from the studies available, it was not clear whether (n-6) PUFA oil, (n-9) MUFA and various types of SFA-rich fats elicited different postprandial responses. This study was, thus performed in healthy humans to carefully compare the effects of mixed meals containing a reasonable amount of fat (40 g) in the form of common fat sources such as olive oil (n-9) MUFA source, sunflower oil (n-6) PUFA source or butter (n-SFA source) on postprandial lipemia, CM, apoB48- and apoB100-TRL and nonesterified fatty acids (NEFA). A no-fat test meal was used as a control.

SUBJECTS AND METHODS

Subjects. Ten adult men (20–29 y old) participated in the study after giving written informed consent to a protocol approved by the local Medical Ethics Committee (Comité Consultatif pour la Protection des Personnes se prêtant à des Recherches Biomédicales, Mar- seille). None of the subjects suffered from any digestive or metabolic disease as checked by medical history and fasting blood chemistry. No subject was obese [body mass index (BMI): 22.1 ± 0.80 kg/m²] and body weights did not vary noticeably during the experiment. The subjects had not taken medications that interfered with lipid metabolism or vitamin A supplements for months. They were instructed not to exercise, given their reported disease as checked by medical history and fasting blood chemistry. No subject was obese [body mass index (BMI): 22.1 ± 0.80 kg/m²] and body weights did not vary noticeably during the experiment. The subjects had not taken medications that interfered with lipid metabolism or vitamin A supplements for months. They were instructed not to exercise, given their reported disease as checked by medical history and fasting blood chemistry. No subject was obese [body mass index (BMI): 22.1 ± 0.80 kg/m²] and body weights did not vary noticeably during the experiment. The subjects had not taken medications that interfered with lipid metabolism or vitamin A supplements for months. They were instructed not to exercise, given their reported disease as checked by medical history and fasting blood chemistry. No subject was obese [body mass index (BMI): 22.1 ± 0.80 kg/m²] and body weights did not vary noticeably during the experiment. The subjects had not taken medications that interfered with lipid metabolism or vitamin A supplements for months. They were instructed not to exercise, given their reported disease as checked by medical history and fasting blood chemistry. No subject was obese [body mass index (BMI): 22.1 ± 0.80 kg/m²] and body weights did not vary noticeably during the experiment. The subjects had not taken medications that interfered with lipid metabolism or vitamin A supplements for months. They were instructed not to exercise, given their reported disease as checked by medical history and fasting blood chemistry. No subject was obese [body mass index (BMI): 22.1 ± 0.80 kg/m²] and body weights did not vary noticeably during the experiment. The subjects had not taken medications that interfered with lipid metabolism or vitamin A supplements for months. They were instructed not to exercise, given their reported disease as checked by medical history and fasting blood chemistry.

Subject characteristics. The men consumed a typical French diet, with a moderate energy consumption (mean: 11,616.3 ± 491.9 kJ/d) with protein, carbohydrate and fat accounting for 13.9 ± 0.7%, 40.0 ± 2.3% and 45.5 ± 2.7% total energy intake, respectively. Daily intakes of saturated, monounsaturated and polyunsaturated lipids were 66.4 ± 5.7, 58.0 ± 5.2 and 16.8 ± 1.5 g, respectively. Alcohol intake was negligible. Daily intakes of cholesterol, fiber and retinol were 522.2 ± 71.6 mg, 19.7 ± 1.6 g and 440 ± 84 μg, respectively. Serum metabolites in the food-deprived men were comparable to one another and not different at the time of the four tested meals and were in the normal range (triaclyglycerols: 0.72–0.88 mmol/L; total cholesterol: 4.69–4.73 mmol/L; insulin: 76.6–111.20 pmol/L; glucose: 5.12–5.26 mmol/L). Eight subjects exhibited an apoE 3/3 genotype, one an apoE 2/2 and one an apoE3/4 genotype.

Test meals and sampling. The four experimental test meals were consumed by each subject in a random order. The interval between the two test meals was 5–7 d. As done previously (1) to prevent possible effect of the previous meal, the subjects were asked to have a light dinner before 2000 h the evening before each experiment.

Three mixed meals containing the same amount of dietary fat (40 g) but from different sources of fatty acid [i.e., butter (BF), olive oil (OO) or sunflower oil (SO)] and a meal without any fat (which served as control) were tested. Butter mainly provided saturated fat (53.7 g/100 g) in the form of 4.0–12.0 (14.1 g/100 g), 14:0 (11.0), 16:0 (30.0), 18:0 (11.3) and 18:1 (25.0). Olive oil mainly provided 16:0 (11.0 g/100 g), 18:1 (75.5) and 18:2 (8.5). Sunflower oil mainly provided 18:1 (21.2 g/100 g) and 18:2 (67.0). The meals consisted of commercially available food and contained two slices of French bread, 150 g cooked pasta, 50 g tomato sauce, one no fat yogurt (125 g) and the tested fat (40 g) or no fat. The vegetable oils were incorporated into the tomato sauce and butter spread on bread. The so-called no-fat test meal provided a negligible amount of fat (~1.3 g) and virtually no cholesterol. The OO and SO test meals also did not contain cholesterol while the butter test meal (BF) provided 100 mg of cholesterol. Retinyl palmitate (Rhône Poulenc-Rorer, Paris, France) was added at the level of 15 mg of retinol per meal. During the 7-h postprandial period, participants were allowed to drink 200 mL water and decaffeinated coffee (100 mL).

After an overnight fast, an antecubital vein was catheterized and intravenous cannulae equipped with disposable obturators (Jelco-Critikon, Chatenay-Malabry, France). A baseline (0 h) blood sample was collected. Then, the subjects ingested the test meal within 20 min. Blood samples (15–20 mL) were obtained every hour for 7 h as done previously (1,4).

Blood samples collected in tubes with or without EDTA, plasma and serum, respectively, were separated from whole blood by centrifugation (910 × g, 4°C, 10 min).

Analytical measurements

CM and triacylglycerol-rich lipoprotein separations. CM were isolated as described previously (1) from 1 mL plasma layered under 2 mL NaCl (9.0 g/L) by ultracentrifugation at 10°C for 20 min at 25,000 × g in a Beckmann TL100 ultracentrifuge and 100.3 rotor (Palo Alto, CA).

The TRL fraction (TRL: VLDL + large and small chylomicron remnants) was separated from whole plasma at a density of 1.019 kg/L and 16°C, at 540 960 g for 3 h 30 min in a 100.3 rotor with a Beckmann TL100 ultracentrifuge (1,16). We used plasma samples obtained at fasting, 2–4 h (pooled), and 7 h postprandially. Subsequently, apoB100-containing TRL particles were separated from apoB48-containing TRL (from the 2- to 4-h pooled plasma samples only) by affinity chromatography using a 2G8 monoclonal antibody (Mona, Moscow, Russia), which does not cross-react with apoB48 according to a procedure described by Cohn et al. (3) and as previously used (16). In normolipemic subjects, the apoB48 fraction obtained contains 4–11% of contaminating apoB100-containing particles (16).

TRL subfractions using flotation rate ranges in Svedberg units (Sf) >400 (diameter > 75 nm), 60<Sf<400 (diameter: 34–75 nm) and 20<Sf<60 (diameter: 20–34 nm) were isolated as described previously (15). Briefly, 1 mL NaCl solution (1.1 kg/L) was added to 3 mL plasma adjusted with NaCl to density 1.1 kg/L, to obtain a final volume of 4 mL at 1.1 kg/L. Then, a density gradient (1.065 kg/L, 1.020 kg/L and 1.006 kg/L NaCl) was made by successive addition of 3 mL NaCl solutions of each density. Successive ultracentrifugations at 200,000 g and 15°C using a SW 40 Ti rotor (Beckman) were performed to float particles of Sf > 400 (32 min), 60<Sf<400 (3 h 28 min) and 20<Sf<60 (17 h). Aliquots of 0.5 mL were aspirated from the tubes and the equivalent volume of 1.06 kg/L NaCl was added at the end of each centrifugation. Lipids in the different fractions were measured.

Biochemical assays. Serum and lipoprotein total cholesterol was measured (17) by an enzymatic method with a cholestero-Situm RTU kit from Biomérieux (Marcy, l’Etoile, France). Free cholesterol was determined enzymatically using a kit CHOD-POD TRINDER provided by Biotrol diagnostic (Lagnes, France). Triaclyglycerols (18) and phospholipids (19) were measured by enzymatic methods using PAP 150 kits provided by Biomérieux. Glucose levels (20) were measured by a glucose RTU kit from Biomérieux. Total insulin was assayed by an immuno-enzymatic method (21) with commercial kits (Boehringer Mannheim, Mannheim, Germany). Serum NEFA were measured by using the NEFA ACS-ACOD method (22) with a kit provided by Wako Chemicals GmbH (Neuss, Germany). Retinyl palmitate was assayed in the chylomicron fraction using a

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HPLC method described previously (4,16). Total plasma lipids (fasting and 2- to 4-h pooled samples) were extracted according to the method of Folch et al. (23). FAME with an added mixture of reference standards [14:0 to 22:6 (n-3); Interchim, Mont-Lucon, France] were prepared (24). The esters were separated by gas chromatography (Autosystem XL; Perkin Elmer, Montigny-Le-Bretonneux, France) using a 30-m capillary column (inner diameter: 0.22 mm, solid phase: BPX 70, film thickness: 0.25 μm) and equipped with a FID detector. The oven temperature was programmed from 160°C to 190°C at a rate of 1°C/min and held at the final temperature for 2 min. Peak areas were integrated and the results expressed as a proportion of total fatty acid mass.

Chylomicron sizes were measured at 20°C on suspended, freshly prepared samples by Photon Correlation Spectroscopy using a particle-size analyser (SEMATech, Nice, France) (16). ApoE genotyping was performed after DNA extraction from blood samples of food-deprived subjects collected on EDTA according to Botham et al. (27) as follows: concentrated CM (2.5 mg protein/mL) were adjusted to 20 μg/mL and lysed with 2-β-mercaptoethanol (20 μL). Lysate was diluted with 200 μL of sodium carbonate buffer (450 mM) and 100 μL glycglycine buffer (50 mM, pH 9.7) was added, so that the final volume was 1.6 mL. The absorbance at 550 nm was measured using a spectrophotometer (Beckman, Fullerton, CA).

In vitro lipolysis of human CM. Postheparin plasma samples (2–3 mL) were obtained 15 min after intravenous administration of 50 μg/kg of heparin to human volunteers. Lipoprotein lipase and hepatic lipase activities were measured according to the method of Krauss et al. (26). CM were collected as described above 2 to 4-h pooled samples) were extracted according to the method of Folch et al. (23). FAME with an added mixture of standards were prepared (24). The esters were separated by gas chromatography (Autosystem XL; Perkin Elmer, Montigny-Le-Bretonneux, France) using a 30-m capillary column (inner diameter: 0.22 mm, solid phase: BPX 70, film thickness: 0.25 μm) and equipped with a FID detector. The oven temperature was programmed from 160°C to 190°C at a rate of 1°C/min and held at the final temperature for 2 min. Peak areas were integrated and the results expressed as a proportion of total fatty acid mass.

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RESULTS

After the no-fat meal, the proportions of 16:0 (±7%) and 18:0 (±3%) fatty acids in the plasma were greater than at baseline (P < 0.05; Table 1). 14:0 significantly increased (±4.1%) 2–4 h after butter meal (BF) ingestion only. 16:0 and 18:0 were greater than baseline after the butter meal (±8% and ±1.5%) and were below the baseline level after olive (±7% and ±2%) and sunflower (±10% and ±2%) oil meals. As expected, the plasma 18:1 level was significantly greater than baseline after the olive oil meal (O0; ±21%), and 18:2 increased (±25%) after the sunflower oil meal (SO).

TABLE 1

<table>
<thead>
<tr>
<th>Fatty acid</th>
<th>Fasting</th>
<th>No-fat meal</th>
<th>Butter meal</th>
<th>Olive oil meal</th>
<th>Sunflower oil meal</th>
</tr>
</thead>
<tbody>
<tr>
<td>14:0</td>
<td>3.0 ± 0.8b</td>
<td>2.5 ± 0.8b</td>
<td>7.1 ± 0.9a</td>
<td>1.3 ± 0.4b</td>
<td>1.5 ± 0.6b</td>
</tr>
<tr>
<td>16:0</td>
<td>26.7 ± 4.4b</td>
<td>34.1 ± 2.0a</td>
<td>35.3 ± 1.5a</td>
<td>19.6 ± 1.6c</td>
<td>17.1 ± 1.2c</td>
</tr>
<tr>
<td>16:1</td>
<td>1.4 ± 0.3a</td>
<td>1.8 ± 0.6a</td>
<td>1.8 ± 0.5a</td>
<td>0.7 ± 0.1b</td>
<td>0.6 ± 0.1b</td>
</tr>
<tr>
<td>18:0</td>
<td>8.8 ± 1.4b</td>
<td>11.6 ± 1.6b</td>
<td>10.3 ± 0.3ab</td>
<td>6.4 ± 0.4b</td>
<td>6.5 ± 0.3b</td>
</tr>
<tr>
<td>18:1</td>
<td>25.2 ± 3.6b</td>
<td>22.7 ± 1.4b</td>
<td>24.9 ± 1.7b</td>
<td>53.4 ± 2.4a</td>
<td>27.5 ± 2.6b</td>
</tr>
<tr>
<td>18:2</td>
<td>19.0 ± 2.2b</td>
<td>20.2 ± 3.6b</td>
<td>16.4 ± 0.9b</td>
<td>16.7 ± 0.8b</td>
<td>45.4 ± 3.3a</td>
</tr>
<tr>
<td>20:4</td>
<td>3.8 ± 0.5a</td>
<td>4.5 ± 0.8a</td>
<td>3.1 ± 0.5a</td>
<td>2.5 ± 0.3ab</td>
<td>2.0 ± 0.4b</td>
</tr>
<tr>
<td>SFA3</td>
<td>44.9 ± 6.6a</td>
<td>47.6 ± 2.9a</td>
<td>53.7 ± 1.6a</td>
<td>24.6 ± 0.9b</td>
<td>25.5 ± 2.0b</td>
</tr>
<tr>
<td>MUFA</td>
<td>31.3 ± 5.6b</td>
<td>27.1 ± 4.2b</td>
<td>26.8 ± 1.7b</td>
<td>51.9 ± 3.1a</td>
<td>27.7 ± 2.4b</td>
</tr>
<tr>
<td>PUFA</td>
<td>23.2 ± 2.5b</td>
<td>24.4 ± 3.8b</td>
<td>19.5 ± 0.9b</td>
<td>21.0 ± 2.3b</td>
<td>48.2 ± 2.9a</td>
</tr>
</tbody>
</table>

1 Values are means ± SEM, n = 10. Those in a row without a common letter differ, P < 0.05.
2 Postprandial samples collected at 2, 3 and 4 h after ingestion of meals were pooled.
3 SFA, saturated fatty acids (14:0 + 16:0 + 18:0); MUFA, monounsaturated fatty acids (16:1 + 18:1); PUFA, polyunsaturated fatty acids (18:2 + 20:4).
showed comparable 1- to 7-h drops after intake of the three fat-containing test meals, while the no-fat meal tended ($P < 0.085$) to induce less of a change (Fig. 2B).

**Postprandial chylomicron and TRL lipid responses.** As expected, no CM triacylglycerols appeared when no fat was

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**FIGURE 1** Effects of four test meals providing no fat or butter or olive oil or sunflower oil on serum during the postprandial state (0–7 h), triacylglycerols (A) and phospholipids (B) in healthy young men. Values are means ± SEM, n = 10. The incremental areas under the curves (AUC) are shown in inserts. For a given test meal, a filled symbol indicates that the corresponding value differs ($P < 0.05$) from the fasting (0 h) value. Different letters indicate a difference ($P < 0.05$) between meals at a given time.

**FIGURE 2** Effects of four test meals providing no fat or butter or olive oil or sunflower oil on serum during the postprandial state (0–7 h), non esterified cholesterol (A) and esterified cholesterol (B) in healthy young men. Values are means ± SEM, n = 10. The incremental areas under the curves (AUC) are shown in inserts. For a given test meal, a filled symbol indicates that the corresponding value differs ($P < 0.05$) from the fasting (0 h) value. Different letters indicate a difference ($P < 0.05$) between meals at a given time.
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present in the meal (Fig. 3). Conversely, when fat was consumed, CM triacylglycerol concentrations markedly increased and peaked 2–4 h postprandially. However, the BF meal induced significantly lower CM triacylglycerol responses (2, 3 and 4 h) than the OO and SO meals (Fig. 3, insert).

Residual fasting CM had a small mean particle size (91-nm diameter) after intake of all three fat-containing meals (Fig. 4). During the 1st h postprandially, the mean CM size sharply increased; a maximum diameter was observed after 2–4 h (range: 148–163 nm after OO and SO meals), and then decreased until 6 h. CM diameters were not different after OO and SO meals. After the BF meal, the size of CM were lower after intake of OO and SO after 2, 3 and 4 h (134–127 nm). At 5 and 6 h postprandially, CM were smaller after all of the three test meals.

Chylomicron retinyl esters, a marker of intestinally derived particles, exhibited bell-shaped curves with maximum values 2–4 h postprandially and returned to baseline 6–7 h after all test meals (data not shown). CM retinyl ester 0–7 h AUC (mmol·h·L⁻¹) were higher (P < 0.05) after OO (2972 ± 299) and SO (2789 ± 288) meals compared with the BF (1777 ± 327) and no-fat (633 ± 314) meals.

Differences in the distribution of triacylglycerols were observed at 2–4 h after meal intake (Table 2). As expected, almost all TRL triacylglycerols were carried by VLDL in fasting subjects (range: 72.6–79.9%) or postprandially when no fat was added to the meal. Ingestion of the butter meal induced higher VLDL triacylglycerols than the OO or SO oil meals. Conversely, CM contained more triacylglycerols 2–4 h after OO and SO oil meals than after butter intake.

This observation was confirmed by fractionationing total TRL by size (data not shown). Less triacylglycerol was found in large Sf > 400 particles and more in small 20<Sf<60, 2–4 h after the BF meal than after OO and SO meals.

There were no differences 2–4 h after intake of fat-containing meals of TRL phospholipids, nonesterified cholesterol or esterified cholesterol concentrations. However, after BF meal intake, 60 ± 2% total TRL esterified cholesterol was present in the 20<Sf<60 fraction while 49 ± 2 and 52 ± 1%, respectively, were in corresponding TRL particles after OO and SO meals.

Total 2- to 4-h postprandial TRL particles were subfractionated in intestinally derived, apoB48-containing TRL and hepatically derived, apoB100-containing TRL particles. With no fat present in the meal, moderate levels of TRL triacylglycerols were found, with a vast majority (84.6%) in the form of apoB100 particles (Table 3). In contrast, the three fat-containing meals elicited significantly higher total TRL triacylglycerol levels, with apoB48-TRL accounting for ~50% of the total TRL. Nevertheless, BF and SO meals elicited the lowest, and comparable, apoB48-TRL triacylglycerol concentrations and the OO meal resulted in a significantly higher total TRL level than did the BF meal.

**In vitro chylomicron lipolysis.** After 20 min incubation, released free fatty acids (µmol·min·L⁻¹) tended to be higher (P = 0.076) in the CM obtained after the men consumed the SO meal. After 2 h incubation, significantly (P < 0.05) more NEFA were released from CM derived from SO meals (0.24 ± 0.07) than with those collected after the BF meal (0.19 ± 0.002) or OO meal (0.16 ± 0.002).

**DISCUSSION**

This study compared, in healthy young men, the postprandial responses to single mixed test meals containing 40 g of butter, olive oil or sunflower oil, and a no-fat meal. Mixed test meals of TRL phospholipids, nonesterified cholesterol or esterified cholesterol concentrations. However, after BF meal intake, 60 ± 2% total TRL esterified cholesterol was present in the 20<Sf<60 fraction while 49 ± 2 and 52 ± 1%, respectively, were in corresponding TRL particles after OO and SO meals.

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**FIGURE 3** Effects of four test meals providing no fat or butter or olive oil or sunflower oil during the postprandial state (0–7 h) on chylomicron triacylglycerols in healthy young men. Values are means ± SEM, n = 10. The incremental areas under the curves (AUC) are shown in insert. For a given test meal, a filled symbol indicates that the corresponding value differs (P < 0.05) from the fasting (0 h) value. Different letters indicate a difference (P < 0.05) between meals at a given time.

**FIGURE 4** Effect of three test meals providing butter or olive oil or sunflower oil on chylomicron sizes (nm diameters) during the postprandial state (0–6 h) in healthy young men. Values are means ± SEM, n = 10. For a given test meal, a filled symbol indicates that the corresponding value is significantly different (P < 0.05) from the fasting (0 h) value. Different letters indicate a difference (P < 0.05) between meals at a given time.
Dietary Fat and Postprandial Lipemia

TABLE 2
Triacylglycerol-rich lipoproteins (TRL), chylomicrons (CM) and VLDL triacylglycerol concentrations in men while fasting and 2–4 h and 7 h after ingestion of four test meals

<table>
<thead>
<tr>
<th></th>
<th>Fasting</th>
<th>2–4 h postprandially</th>
<th>7 h postprandially</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mmol/L</td>
<td>% of total</td>
<td>mmol/L</td>
</tr>
<tr>
<td>No fat</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total TRL</td>
<td>0.39 ± 0.06</td>
<td>—</td>
<td>0.45 ± 0.07</td>
</tr>
<tr>
<td>VLDL</td>
<td>0.30 ± 0.003</td>
<td>79.7 ± 4.0</td>
<td>0.39 ± 0.06</td>
</tr>
<tr>
<td>CM</td>
<td>0.09 ± 0.03</td>
<td>20.3 ± 4.0</td>
<td>0.06 ± 0.01†</td>
</tr>
<tr>
<td>Butter</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total TRL</td>
<td>0.36 ± 0.03$</td>
<td>—</td>
<td>0.64 ± 0.06</td>
</tr>
<tr>
<td>VLDL</td>
<td>0.27 ± 0.03a</td>
<td>75.7 ± 4.3x</td>
<td>0.36 ± 0.03a</td>
</tr>
<tr>
<td>CM</td>
<td>0.08 ± 0.02b</td>
<td>24.3 ± 4.3y</td>
<td>0.27 ± 0.04a</td>
</tr>
<tr>
<td>Olive oil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total TRL</td>
<td>0.45 ± 0.03b</td>
<td>—</td>
<td>0.89 ± 0.07a</td>
</tr>
<tr>
<td>VLDL</td>
<td>0.35 ± 0.03</td>
<td>78.5 ± 2.5x</td>
<td>0.38 ± 0.03</td>
</tr>
<tr>
<td>CM</td>
<td>0.09 ± 0.01b</td>
<td>21.5 ± 2.5y</td>
<td>0.50 ± 0.07a†</td>
</tr>
<tr>
<td>Sunflower oil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total TRL</td>
<td>0.40 ± 0.04b</td>
<td>—</td>
<td>0.83 ± 0.07a</td>
</tr>
<tr>
<td>VLDL</td>
<td>0.29 ± 0.04</td>
<td>72.6 ± 5.9x</td>
<td>0.39 ± 0.08</td>
</tr>
<tr>
<td>CM</td>
<td>0.10 ± 0.02b</td>
<td>28.8 ± 7.0y</td>
<td>0.48 ± 0.06a</td>
</tr>
</tbody>
</table>

1 Values are means ± SEM, n = 10. Those in a row with different superscript letters (a, b for the concentration) and (x, y for the percentage) differ (P < 0.05) between fasting, 2–4 h and 7 h time-points for a given meal and for the particular fraction. Percentage refers, for a given meal, to the fraction of VLDL or chylomicrons in total TRL. Different symbols (*, †, §) indicate a difference (P < 0.05) between meals at a given time point for a particular fraction (total TRL, VLDL or chylomicrons).

Equivalent ingested amounts of olive oil [essentially a source of 18:1 (n-9) oleic acid] and sunflower oil [a major source of 18:2 (n-6) linoleic acid] did not induce different postprandial responses for serum and chylomicron triacylglycerols, suggesting a comparable handling of both oils in the digestive tract and blood compartment (28,29). These data agree with some studies (13,14) but not another study (12) in which an olive oil emulsion generated a somewhat higher serum triacylglycerol response compared with a PUFA-rich fat emulsion of soybean oil. This apparent discrepancy could result from the use of lipid emulsions in place of mixed meals as used in this study. The lack of difference found in this study in the sizes of circulating CM and concentrations of intestinally derived apo48-TRL confirms that the acute handling of these two sources of unsaturated fat might be overall comparable. Previous reports showed comparable (30) or somewhat different (31) chylomicron sizes after MUFA or PUFA intakes. Although the same amount of the three types of fat were ingested by the subjects, the BF meal elicited significantly lower overall serum and chylomicron triacylglycerol postprandial increases (~38% and ~54% for 0–7 h AUC, respectively) than the two other fat-containing meals. This observation agrees with data obtained by others using dairy fats or unesterified saturated fat (14). In contrast, other studies showed higher postprandial responses with butter-containing meals (15), or no difference after meals providing dairy cream or saturated fat compared with unsaturated fats (32,33). Differences in the amount and nature of fat, composition of test meals and subjects involved could partly explain such discrepancies.

Several mechanisms could be involved in the reduced post-

TABLE 3
Triacylglycerol concentrations in total triacylglycerol-rich lipoproteins (TRL), apoB48- and apoB100-containing TRL isolated 2–4 h after ingestion of the test meals in serum of healthy young men

<table>
<thead>
<tr>
<th></th>
<th>Total TRL apoB48-TRL apoB100-TRL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>triacylglycerols</td>
</tr>
<tr>
<td></td>
<td>mmol/L</td>
</tr>
<tr>
<td>No-fat meal</td>
<td>0.45 ± 0.07$</td>
</tr>
<tr>
<td>Butter meal</td>
<td>0.64 ± 0.06a</td>
</tr>
<tr>
<td>Olive oil meal</td>
<td>0.89 ± 0.07a</td>
</tr>
<tr>
<td>Sunflower meal</td>
<td>0.83 ± 0.08a</td>
</tr>
</tbody>
</table>

1 Values are means ± SEM, n = 10. Those in a column with different superscript letters differ, P < 0.05.

2 TRL were obtained by ultracentrifugation of pooled 2, 3 and 4 h serum samples at d < 1.019 kg/L.
prandial response elicited by butter as observed in this study when considering the physicochemical state of butter (a water-in-oil emulsion) (34) and its fatty acid composition [42%, 17.5% and 7.6% of long-, medium- and short-chain fatty acids (SCFA)].

Given the process of fat emulsification occurring in the stomach that generates oil-in-water emulsified lipid droplets with 10- to 100-μm diameters in the aqueous medium (35), it is likely that ingested butter (a water-in-oil emulsion) is not a suitable substrate for gut lipases, which are acting at the aqueous interface of the lipid droplet surface. The coarsely emulsified vegetable oils used in this study are expected to provide triacylglycerols in a more suitable physicochemical state (35). Indeed, it has been reported that unemulsified long-chain saturated fat can have markedly reduced postprandial appearance (31). Thus, a slow and poor emulsification of butter fat associated with a possible delayed gastric emptying could explain some differences between the effects of butter and vegetable oils.

A second step possibly involved in the differential postprandial responses is the intestinal uptake of released free fatty acids and monoglycerides. A lower availability of long-chain SFA at the sn-1 position due to formation of insoluble calcium-soap complexes in the intestinal lumen has been reported (35,36). For this reason, some of these fatty acids present in butter could be slowly available and, thus, would somewhat delay or lower the overall rate of absorption and the subsequent rise in the circulation, as found in this study.

The resorption of fatty acids from the enterocytes into the circulation is likely to be another important step involved in the differences between dietary fats. Data obtained with cultured enterocytes of the human Caco2 cell line indicate that the basolateral secretion of triacylglycerol-rich lipoproteins is 2-fold stimulated in the presence of absorbed oleic and linoleic acids compared with long-chain SFA (29), with a higher triacylglycerol/apoB ratio with oleic acid. Thus, this study suggests that intestinal resorption of TRL long-chain fatty acids in the circulation may differ in the following decreasing order: oleic acid, linoleic acid, palmitic and stearic acids.

The vast majority of long-chain fatty acids (either saturated or unsaturated) are incorporated into triacylglycerols and phospholipids packaged with apoB48 as chylomicron particles (28). Conversely, short-chain and medium-chain fatty acids are readily transported from the enterocyte basolateral membrane as complexes with albumin via the portal route (37). Given that ~20% butter fatty acids are of short and medium chains, one might expect a reduction in this range in the peripheral plasma postprandially. Indeed, a 15% reduced triacylglycerol secretion in the lymph has been reported in rats at 8 h after butter oil infusion compared with after infusion of corn oil (30). In humans, the replacement in butter of naturally present medium-chain fatty acids by long-chain oleic and stearic acids increased postprandial chylomicron responses to some extent (38). In fact, the overall reduction observed herein in intestinally derived chylomicron triacylglycerols is much more dramatic (~54.8%), implicating the various mechanisms discussed above.

As measured using a laser-equipped particle sizer or after subfractionation according to flotation behavior, the size of circulating CM resulting from assimilation of the butter meal were consistently lower than those obtained after the two vegetable oil meals. The small amount (100 mg) of cholesterol provided by butter fat is not expected to influence chylomicron size (4). These differences can be attributed either to a reduced size of CM when secreted into the lymph or to a faster rate of endovascular lipolysis of butter fatty acid-containing CM. In fact, data obtained in this study in humans are in line with others obtained in rats (39,40) and cultured Caco2 cells (29). CM with different fatty acid compositions and sizes are expected to have different fates in the circulation. Indeed, ex vivo lipolysis of human CM performed in this study shows that CM derived from the butter meal exhibited a slower rate of lipolysis as catalyzed by postheparin plasma lipases than those derived from the SO meal, in agreement with previous data (27). This supports the concept of a reduced susceptibility to endovascular lipolysis of CM derived from butter fat.

Finally, no delay in the clearance of butter fatty acid-containing CM was observed in this study in healthy young men in line with another study that showed that plasma clearance of CM is not dependent on butter fat saturation (46). This has also been observed after chronic feeding of animals (39,44) or humans (2,47,48). Nevertheless, the removal of these CM with reduced size from the circulation is expected to be slowed given the lower rate of clearance of smaller particles as reported (41,42). Other acute experiments performed in rats (43–45) and rabbits (33) have also shown that the clearance of liver uptake of SFA-rich CM can be somewhat delay or lower the overall rate of absorption and the subsequent rise in the circulation, as found in this study.

In conclusion, this study shows that single mixed meals containing butter reduce postprandial lipemia and chylomicron accumulation in the circulation compared with OO- or SO-containing meals in healthy young men. This does not necessarily apply to other categories of subjects or patients. Thus, the overall well established detrimental effects of chronic intake of saturated fat on fasting plasma and LDL cholesterol could not be related directly to a detrimental influence on postprandial lipid metabolism in the case of butter.

LITERATURE CITED


