Modulating absorption and postprandial handling of dietary fatty acids by structuring fat in the meal: a randomized crossover clinical trial

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ABSTRACT

Background: Prolonged postprandial hypertriglyceridemia is a potential risk factor for cardiovascular diseases. In the context of obesity, this is associated with a chronic imbalance of lipid partitioning oriented toward storage and not toward β-oxidation.

Objective: We tested the hypothesis that the physical structure of fat in a meal can modify the absorption, chylomicron transport, and further handling of dietary fatty acids.

Design: Nine normal-weight and 9 obese subjects were fed 40 g milk fat (+[13C]triacylglycerols), either emulsified or nonemulsified, in breakfasts of identical composition. We measured the postprandial triacylglycerol content and size of the chylomicron-rich fraction, plasma kinetics of [13C]fatty acids, exogenous lipid oxidation with breath-test/indirect calorimetry, and fecal excretion.

Results: The emulsified fat resulted in earlier (>1 h) and sharper chylomicron and [13C]fatty acid peaks in plasma than in spread fat in both groups (P < 0.0001). After 2 h, the emulsified fat resulted in greater apolipoprotein B-48 concentrations (9.7 ± 0.7 compared with 7.1 ± 0.9 mg/L; P < 0.05) in the normal-weight subjects than did the spread fat. In the obese subjects, emulsified fat resulted in a 3-fold greater chylomicron size (218 ± 24 nm) compared with the spread fat (P < 0.05). The emulsified fat induced higher dietary fatty acid spillover in plasma and a sharper [13CO2] appearance, which provoked increased exogenous lipid oxidation with breath-test/indirect calorimetry, and fecal excretion.

Conclusion: This study supports a new concept of “slow vs fast fat,” whereby intestinal absorption can be modulated by structuring dietary fat to modulate postprandial lipemia and lipid β-oxidation in humans with different BMIs. This trial was registered at clinical-trials.gov as NCT01249378. Am J Clin Nutr doi: 10.3945/ajcn.112.043976.

INTRODUCTION

The metabolic importance of intestinal absorption and transport of nutrients in the postprandial period is recognized as important in the context of metabolic diseases such as obesity and type 2 diabetes (1). Regarding lipid metabolism, plasma kinetics, timing of peak of lipemia, and chylomicron size are recognized as factors determining metabolic complications that are still an open field of research (2–4). For this reason, control of intestinal lipid absorption, the resulting chylomicron transport dynamics, and the ultimate fate of dietary lipids may be an effective tool in the management of metabolic diseases. Recent studies have shown differential effects of oral sensory stimulation with high compared with low amounts of dietary fat on intestinal lipid absorption (5). The possible effects of the fatty acid (FA) profile of an oral fat load on chylomicron size have been suggested (6–10). Whereas both fat load and composition can affect postprandial lipid absorption, few studies have investigated the effects of fat structure on the postprandial metabolism of an identical lipid load. In diabetes, the concept of “slow/low glycemic index carbohydrates and fast/high glycemic index carbohydrates” is well established and has facilitated the development of specific foods and/or cooking methods to control postprandial glycemia (11). We thus raised the question of whether a similar concept may be applicable to dietary fat according to the way it is structured in the meal.

Dietary lipids are incorporated in food products with different physicochemical structures, eg, in dispersed lipid droplets in oil-in-water emulsions such as ice cream or in a continuous lipid phase in

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4 Abbreviations used: AP, atom percent; APE, atom percent excess; CMRF, chylomicron-rich fraction; FA, fatty acid; FAME, fatty acid methyl ester; iAUC, incremental AUC; NEFA, nonesterified fatty acid; NW, normal-weight.

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butter and margarine. Emulsions are the most widespread fat structures, in processed foods, and enteral formulas and are therefore of interest regarding their role in lipid digestion and absorption (12). Indeed, we previously showed in rodents the importance of the lipid-emulsified structure on FA absorption and \( \beta \)-oxidation (13–15). However, the effect of fat structure on the kinetics of lipid absorption and dietary FA handling in humans remains to be elucidated. Such effects also deserve to be elucidated in obese subjects who present altered storage function of dietary FA in the postprandial state (16).

We therefore hypothesized that the physicochemical structure of the fat in a meal could modulate postprandial lipemia and fat partitioning (storage compared with oxidation) and that the effect would be more pronounced in obese subjects. We investigated the metabolic response to fat-containing meals (40 g) differing only in the structuring of fat, emulsified or not, in healthy, young normal-weight (NW) and obese men. Measurements included chylomicron number and size, FA \( \beta \)-oxidation, and FA excretion in feces. The aim of this study was to define the contribution of fat structure and subject BMI on the postprandial lipemia and metabolism of dietary FA.

**SUBJECTS AND METHODS**

*Study design*

This study was an open-label trial with a crossover randomized controlled design involving 2 d of metabolic testing separated by ≥3 wk (see Supplemental Figure 1 under “Supplemental data,” in the online issue). It was conducted at the Human Nutrition Research Center Rhône-Alpes (Lyon, France) according to the Second Declaration of Helsinki and the French Huriet-Serusclat law. The LIPides INFlammation OXydation study was approved by the Scientific Ethics Committee of Lyon Sud-Est-II and Agence Francaise de Sécurité Sanitaire des Produits de Santé. Volunteers received written and oral information, and their medical history was reviewed. In addition, they underwent a physical examination and fasting clinical analysis before enrollment. Informed written consent was obtained from all subjects. The subjects performed the trial from April 2010 to July 2011. During the protocol, all subjects were asked to continue their regular diet and activity except for the week before and the 3-d period after each test day. The subjects were told to avoid foods naturally rich in \(^{13}\text{C}\) and were given a list of such foods. For 48 h before testing, the subjects were asked to refrain from consuming alcohol and to avoid exercise. In addition, the subjects were provided with a standardized dinner on the evening before testing. Compliance was checked through dietary records, 5 d before and 3 d after each test day.

After fasting overnight, the subjects ingested 1 of the 2 test breakfasts. The primary outcome measured was the effect of fat structure on postprandial lipemia. The secondary outcomes measured were the effect of fat structure and BMI on postprandial lipid metabolism. Previous studies on lipemia (17) and lipid oxidation (18) were used for the power analysis: a minimum sample size of 8 subjects per BMI group was calculated to be necessary to detect significant changes in these variables. The treatments were randomized according to a random allocation sequence performed by a Human Nutrition Research Center Rhône-Alpes biostatistician by using Stat v.11; 2 randomization lists were generated and stratified over BMI. Subjects were anonymized by using a number corresponding to the randomization sequence order.

*Subjects*

Twenty-two healthy men were recruited, 11 NW and 11 obese, and 20 completed the study (see Flow Diagram under “Supplemental data” in the online issue). One subject in each group was not included in the data analyses because of abnormal postprandial lipid metabolism; therefore, 18 healthy subjects with comparable mean ages were divided into 2 groups (9 NW and 9 obese) and were tested for the primary outcome (Table 1). The subjects were required to be nonsmokers, sedentary or having <4 h/wk of physical activity, and nonclaustrophobic. We excluded persons taking medication known to interfere with lipid metabolism, with a psychological illness, or with eating/metabolic disorders. In addition, the subjects were required to have had stable weight, to be free of diabetes, and to have not made a blood donation for 3 mo before the start of the study. Data were collected at Human Nutrition Research Center Rhône-Alpes.

*Test meals*

The test breakfasts were isenergetic and equal in nutrient composition (Table 2), and both consisted of bread (50 g), skim milk (160 mL), and anhydrous milk fat (40 g) containing 600 mg tracers—either spread on bread or emulsified in skim milk. Both meals had the same composition, and no additional emulsifier was added because milk proteins are sufficient to provide a submicronic milk fat emulsion. Before the test day, a mixture of labeled triacylglycerols (99 atom% \(^{13}\text{C}\); Eurisotop) proportionally representing each FA type present in test fat was first incorporated into melted milk fat: 300 mg \([1,1,1-{^{13}\text{C}}_3]\)tripalmitin for long-chain SFAs, 210 mg \([1,1,1-{^{13}\text{C}}_3]\)triolein for unsaturated SFAs, and 90 mg \([1,1,1-{^{13}\text{C}}_3]\)trioctanoin for short- and medium-chain FAs. For the emulsion test, melted labeled milk fat was coarsely premixed in skim milk (ProScientific Inc) and further finely emulsified (4 \( \times \) 1 min, Vibra-cell Ultrasonic Processor; Sonics) (see Supplemental Figure 2 under “Supplemental data” in the online issue). The test products were then kept at 4°C overnight.

A second meal was served 5 h after breakfast. The meal contained pasta (200 g), turkey (100 g), butter (10 g), olive oil (10 g), bread (50 g), and stewed fruit (100 g), which provided 713 kcal (2985 kJ) with 29%, 51%, and 20% of energy as lipids.

**TABLE 1**

Anthropometric and fasting metabolic variables

<table>
<thead>
<tr>
<th></th>
<th>Normal weight (n = 9)</th>
<th>Obese (n = 9)</th>
<th>( P ) value(^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Anthropometric variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (y)</td>
<td>28.3 ± 1.4</td>
<td>30.2 ± 2.2</td>
<td>NS</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>72.0 ± 2.1</td>
<td>101.2 ± 1.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>BMI (kg/m(^2))</td>
<td>22.5 ± 0.5</td>
<td>31.7 ± 0.3</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>83.3 ± 1.6</td>
<td>105.9 ± 0.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Fasting metabolic variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>4.94 ± 0.16</td>
<td>5.19 ± 0.15</td>
<td>NS</td>
</tr>
<tr>
<td>Insulin (mIU/L)</td>
<td>3.75 ± 0.59</td>
<td>7.14 ± 0.95</td>
<td>0.008</td>
</tr>
<tr>
<td>HOMA</td>
<td>0.85 ± 0.14</td>
<td>1.69 ± 0.25</td>
<td>0.009</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.85 ± 0.22</td>
<td>4.89 ± 0.24</td>
<td>NS</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.51 ± 0.10</td>
<td>1.09 ± 0.06</td>
<td>0.004</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>3.03 ± 0.27</td>
<td>3.11 ± 0.21</td>
<td>NS</td>
</tr>
<tr>
<td>Triacylglycerols (mmol/L)</td>
<td>0.85 ± 0.06</td>
<td>1.39 ± 0.18</td>
<td>0.017</td>
</tr>
</tbody>
</table>

\(^a\)All values are means ± SEMs.

\(^b\)Groups were compared by using an unpaired Student’s \( t \) test.
TABLE 2
Nutritional composition of the test breakfasts containing either spread or emulsified fat enriched with [13C]triacylglycerol tracers

<table>
<thead>
<tr>
<th>Breakfast composition</th>
<th>Food</th>
<th>Carbohydrates</th>
<th>Proteins</th>
<th>Lipids</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>g or mL</td>
<td>g</td>
<td>g</td>
<td>g</td>
</tr>
<tr>
<td>Anhydrous milk fat</td>
<td>40</td>
<td>—</td>
<td>—</td>
<td>40</td>
</tr>
<tr>
<td>Skim milk</td>
<td>160</td>
<td>7.5</td>
<td>5.3</td>
<td>0.3</td>
</tr>
<tr>
<td>Bread</td>
<td>50</td>
<td>28</td>
<td>4</td>
<td>0.5</td>
</tr>
<tr>
<td>[1,1,1-13C3]Tripalmitin</td>
<td>0.09</td>
<td>—</td>
<td>—</td>
<td>0.09</td>
</tr>
<tr>
<td>Trioctanoin [1,1,1-13C3]</td>
<td>0.30</td>
<td>—</td>
<td>—</td>
<td>0.30</td>
</tr>
<tr>
<td>[1,1,1-13C3]Cholesterol</td>
<td>0.21</td>
<td>—</td>
<td>—</td>
<td>0.21</td>
</tr>
<tr>
<td>Total (g)</td>
<td>250.6</td>
<td>35.5</td>
<td>9.3</td>
<td>41.4</td>
</tr>
<tr>
<td>Energy intake (%)</td>
<td>26</td>
<td>7</td>
<td>67</td>
<td></td>
</tr>
</tbody>
</table>

1 Identical nutrient composition for both spread and emulsion breakfasts.
2 Fatty acid profile of triacylglycerols includes 68.6% SFAs, 28.1% MUFAs, and 3.3% PUFAs.
3 Naturally vanilla flavored.

(22.7 g), carbohydrates (91.5 g), and proteins (35.7 g), respectively. All subjects were given 10 min to eat breakfast and 30 min to eat lunch. During the test, the participants were allowed to drink 200 mL water.

Test fat characterization

Emulsion droplet size was measured by Dynamic Light Scattering (Zetasizer Nano S). The specific surface area of emulsion droplets was calculated by using Laser Light Scattering (Mastersizer 2000). The melting temperature and crystalline state of the fat was characterized by Differential Scanning Calorimetry with a Q1000 DSC (TA Instruments) and by powder X-ray Diffraction on an AU 2700 Beckman Coulter (O’Cllagan’s mils) and expressed as differences in concentrations over baseline. The hydrodynamic diameter of CMRF particles was measured by dynamic light scattering at 25°C with a ZetaSizer using 1.0658 cP and 1.33 as viscosity and refractive index of the aqueous phase, respectively. The gravity-separated fraction containing chylomicrons and their large remnants are reported to range in size from 70 to 450 nm (20–22), and the postprandial increase in cholesterol content of such a fraction is about <0.2 mmol/L (23). Our fraction collected by ultracentrifugation contained particles with a mean diameter up to 200–300 nm and with a postprandial increase in cholesterol of <0.15 mmol/L in NW and <0.25 mmol/L in obese subjects. Our CMRF is thus typical of fractions that are rich in chylomicrons and that also contain large remnants.

Hunger assessment

Subjective assessment of hunger was measured on a 10-cm visual analog scale 2 min before breakfast and 2 min before lunch. The specific question used to assess hunger was “How hungry do you feel?”.

Metabolic explorations

Blood samples were obtained at baseline and at regular intervals after the meal from an antecubital arm vein through a catheter and collected into evacuated sterile tubes (with EDTA when necessary). Plasma was separated by centrifugation (1500 × g, 10 min, 4°C) and stored at −20°C until analyzed or at 4°C for separation of the chylomicron-rich fraction (CMRF).

Metabolic tests were divided into postprandial phases, including a first period of 5 h (0–300 min) after breakfast in the morning, a second period of 3 h after lunch (300–480 min), and the entire exploration day (0–480 min).

Indirect calorimetry was performed during metabolic testing by using a Deltatrac II calorimeter (Senormedics). Respiratory exchanges (production rates of expired oxygen and carbon dioxide) were recorded for 30 or 60 min during the 8-h test period. Substrate oxidations were calculated by using Ferrannini’s equations (19).

Plasma metabolite and hormone measurements

Nonesterified FA (NEFA) concentrations were measured with an enzymatic method (Wako). The concentration of apolipoprotein B-48 was measured by ELISA. Insulin concentrations were measured by radioimmunoassay (CISBIO Bi insulin IRMA).

Isolation and analysis of chylomicron-rich fractions

To collect the CMRF, containing chylomicrons and their large remnants, 250 μL plasma was deposited below a layer of 850 µL distilled/deionized water and centrifuged at 80,000 rpm for 30 min with a Sorvall Kendro ultracentrifuge. The floating layer was collected and stored at −80°C. Triacylglycerol and cholesterol concentrations of CMRF were measured with a lipase glycerokinase and a cholesterol esterase/oxidase method, respectively, on an AU 2700 Beckman Coulter (O’Cllagan’s mils) and expressed as differences in concentrations over baseline. The hydrodynamic diameter of CMRF particles was measured by dynamic light scattering at 25°C with a ZetaSizer using 1.0658 cP and 1.33 as viscosity and refractive index of the aqueous phase, respectively. The gravity-separated fraction containing chylomicrons and their large remnants are reported to range in size from 70 to 450 nm (20–22), and the postprandial increase in cholesterol content of such a fraction is about <0.2 mmol/L (23). Our fraction collected by ultracentrifugation contained particles with a mean diameter up to 200–300 nm and with a postprandial increase in cholesterol of <0.15 mmol/L in NW and <0.25 mmol/L in obese subjects. Our CMRF is thus typical of fractions that are rich in chylomicrons and that also contain large remnants.

[13C]Fatty acids in plasma lipids, NEFAs, CMRFs, and stools

Sample preparation

Internal standards were added according to the fraction analyzed (heptadecanoic acid or glycerol triheptadecanoate).

Plasma processing

Plasma samples were submitted to direct methylation as described previously (24).

NEFA processing

Total lipids were extracted from plasma aliquots at 120 min after breakfast consumption (700 μL) with 3 mL of a mixture of chloroform/methanol (2:1, vol:vol) according to the Folch method (25). NEFA fractions were obtained by thin-layer chromatography on silica-gel plates with a mobile phase of hexane: diethyl ether:acetic acid (80:20:1, vol:vol:vol). NEFAs were derivatized to FA methyl esters (FAMEs) (24).

CMRF processing

Lipids were extracted from CMRF at 120 min after breakfast according to the Folch method (25). Triacylglycerol fractions were
then processed, as mentioned above for the NEFA fractions, to obtain the FAMEs from CMRF.

**Stool processing**

Fecal samples were weighed and homogenized, and a precisely weighed aliquot was collected. Total lipids were extracted according to a modification of the Folch method and derivatized to obtain FAMEs (24).

**Sample analysis**

The amounts of FAs in stools, plasma, and NEFAs were assessed by gas chromatography–mass spectrometry with a quadrupole mass spectrometer connected to a gas chromatograph (MS 5975 and GC6890; Agilent Technologies). The isotopic enrichment of palmitic and oleic acids was determined by using gas chromatography–combustion isotope ratio mass spectrometry (Isoprime; GV Instruments) (24). The $^{13}$C enrichments were expressed as atom percent excess (APE). The plasma concentrations of nonesterified labeled palmitic and oleic acids ($[^{13}$C-NEFA]) and nonesterified unlabeled palmitic and oleic acids ($[^{13}$C-NEFA]) were also obtained from these analyses.

**Calculations associated with apparent dietary fatty acid “spillover”**

NEFA analysis 120 min after breakfast was used to calculate $^{13}$C enrichment in plasma NEFAs as follows: $[^{13}$C-NEFA]/($[^{13}$C-NEFA] + $[^{12}$C-NEFA]) (expressed as % enrichment). The proportion of exogenous FAs in plasma NEFAs, as expressed as %, was estimated as the ratio of $^{13}$C enrichment in plasma NEFAs to $^{13}$C enrichment of corresponding FAs in the ingested milk fat. The proportion of exogenous FAs in plasma that was present in the nonesterified form in the sum of pools NEFA + CMRF was calculated as $[^{13}$C-NEFA]/plasma/($[^{13}$C-NEFA]plasma + $[^{13}$C-FA]CMRF]plasma), where $[^{13}$C-FA]CMRF]plasma is the plasma concentration of $^{13}$C-FA esterified in CMRF-triacylglycerols = $[^{13}$C-FA]CMRF/($[^{13}$C-FA]CMRF + $[^{12}$C-FA]CMRF) × 3 × [CMRF-triacylglycerols]plasma.

**Calculations of exogenous lipid oxidation from indirect calorimetry and breath tests**

Exogenous lipid oxidation was calculated according to Binnert et al (18) from indirect calorimetry and breath test data. Here, the formula was adapted to use 3 labeled triacylglycerols as follows:

Exogenous lipid oxidation (% of ingested fat) = $\frac{[\text{APCO}_2(t) + \text{APCO}_2(t-30)]/2 - \text{APCO}_2(t_0)/100] \times \text{VCO}_2 \times 100}{(A) + (B) + (C)} \times 22.4 \times dARF \times 100$ (1)

With: $A = [\text{AP}^{13}$TG $C8 : 0]/100 \times [0.09/473.66] \times 27$ (2)

$B = [\text{AP}^{13}$TG $C16 : 0]/100 \times [0.30/810.30] \times 51$ (3)

$C = [\text{AP}^{13}$TG $C18 : 1]/100 \times [0.21/888.40] \times 57$ (4)

where atom percent (AP) carbon dioxide ($t$) is the AP value of the expired carbon dioxide at time $t$, AP carbon dioxide ($t_0$) is the AP value of the expired carbon dioxide at time $t_0$. CO tracers are the calculated AP values of the labeled mixture of triacylglycerols (so-called AP $^{13}$TG C8:0, AP $^{13}$TG C16:0, and AP $^{13}$TG C18:1 in Equations 2, 3, and 4), and $\text{VCO}_2$ is the production rate of expired CO$_2$ (indirect calorimetry). Mean molecular weights of trioctanoin, tripalmitin, and triolein are 473.66, 810.30, and 888.40 g/mol, respectively. The mean number of carbons in trioctanoin, tripalmitin, and triolein are 27, 51, and 57, respectively. The dietary acetate recovery factor (dARF) is the correction factor for incomplete recovery of $[^{13}$C]bicarbonate (0.505 for NW; 0.453 for obese (26)), and 22.4 is the molar volume (L) of carbon dioxide.

**Kinetic parameters**

We calculated the incremental AUC (iAUC); maximum postprandial concentration, delta, and diameter ($C_{\text{max}}, A_{\text{max}}$, and $d_{\text{max}}$, respectively); time for appearance of these maximum parameters ($t_{\text{max}}$), and appearance/enlargement percentages between 0 and 60 min.

**Statistical analysis**

Each subject served as his own control. All data are presented as means ± SEMS (n = 9 per group) and were analyzed with Statview 5.0 software (Abacus Concept). Postprandial data were compared by ANOVA for repeated measures followed by a post hoc test (Fisher’s protective least-significant difference) for statistical effects of $I$ time alone (P-time) over the first postprandial period (0–300 min), 2) meal alone (P-meal) independently of the time in the postprandial period, and 3) interaction of both factors, time and meal (P-time×meal). Kinetic parameters were compared by 2-factor ANOVA followed by Fisher’s protective least-significant difference test according to meal and BMI (P-meal, P-BMI, P-meal × BMI) and time period before and after lunch (P-meal × BMI × time). Multiple comparisons regarding tracer excretion in feces were performed by using ANOVA followed by a Bonferroni post hoc test. Comparisons between meals within subject groups were performed by using a paired Student’s $t$ test and comparisons between subject groups within meals with an unpaired Student’s $t$ test. Differences were considered significant at the $P < 0.05$ level.

**RESULTS**

**Properties of emulsion compared with spread fat**

The emulsion droplet size (Table 3; see Supplemental Figure 3 under “Supplemental data” in the online issue) indicates that the homogenization was effective at producing the emulsions. The emulsion had a surface area ~70,000-fold greater than that of the spread fat. To control for the possibility that the different metabolic effects could be attributed to the fat-melting properties, we measured melting profiles and crystalline structures in all conditions (with or without tracers, emulsified or not; see Supplemental Figure 4 under “Supplemental data” in the online issue). According to these analyses, the test fats differed only by their structure (Table 3).

**Hunger feeling**

At the end of the first postprandial period (0–300 min) just before lunch, NW subjects felt similarly hungry regardless of breakfast type (see Supplemental Figure 5 under “Supplemental
data" in the online issue). In contrast, obese subjects felt hungrier after emulsion than spread fat consumption ($P < 0.05$). Of note, before breakfast, all subjects felt equally hungry (data not shown).

**Postprandial concentration profile and size of CMRF**

As shown in Figure 1A and B, CMRF-triacylglycerols rapidly increased (60 min) in both groups after ingestion of emulsified fat and peaked at 3–4 h ($t_{\text{max}}$ in Table 4). The emulsion induced a significantly earlier and sharper increase in CMRF-triacylglycerols than did the spread fat (Table 4: $t_{\text{max}}$ and appearance-rate at 60 min; $P < 0.001$). These differences were dramatically marked in the obese subjects, with a significant delay in absorption of the spread fat from 0 to 300 min compared with NW subjects ($P < 0.01$; Table 4). At the end of the test, CMRF-triacylglycerols of NW subjects returned to lower values regardless of fat structure. The obese subjects showed different profiles, and CMRF-triacylglycerols remaining elevated above fasting baseline concentrations at the end of the spread fat test: eg, at 480 min, 0.61 ± 0.15 mmol/L for the spread compared with 0.27 ± 0.06 mmol/L for the emulsion ($P < 0.05$). These differences in profiles before and after lunch, according to obese state and meal type, are supported by different BMI × meal × time interactions for the $D_{\text{max}}$ and iAUC of CMRF-triacylglycerols (Table 4).

Mean CMRF particle size sharply increased in both groups from the first hour after the emulsion (Figure 1, C and D; $P$-meal $< 0.05$ for enlargement rate at 60 min; Table 4). For NW subjects, CMRF diameters were similar after the spread fat and emulsion throughout the test. In obese subjects, CMRF diameters became equal for both meals at 300 min. We note that, from 0 to 240 min, CMRF diameter in obese subjects was higher after emulsion than equal for both meals at 300 min. We note that, from 0 to 240 min, CMRF diameter in obese subjects was higher after emulsion than after spread fat consumption ($P$-meal $< 0.05$, $P$-time $< 0.001$). Overall, obese subjects had larger CMRF particles than did the NW subjects ($P < 0.01$ for $d_{\text{max}}$ 0–480 min; Table 4), and the large CMRF particles persisted after the second meal for the spread fat.

**Plasma concentration profile of apolipoprotein B-48**

As shown in Figure 1 (E and F), plasma apolipoprotein B-48 changed over time in both groups after both breakfasts ($P$-time $<$ 0.0001) and differently according to the type of breakfast for NW subjects ($P$-time×meal $= 0.001$). At 120 min, NW subjects accumulated more ($P < 0.05$) apolipoprotein B-48 after consumption of emulsion (9.73 ± 0.69 mg/L) than after consumption of spread fat (7.08 ± 0.86 mg/L) and more than obese subjects after consumption of emulsion (7.47 ± 0.78 mg/L).

**Plasma concentration profile of $[^{13}\text{C}]$fatty acid tracers and fecal loss**

A change in plasma $[^{13}\text{C}]$palmitic acid and $[^{13}\text{C}]$oleic acid over time was found in both groups after both breakfasts ($P$-time $< 0.0001$; Figure 2, A–D). In obese subjects, $[^{13}\text{C}]$palmitic acid appeared earlier and sharper in plasma when it was an emulsion ($P$-meal $= 0.007$), and plasma concentrations of $[^{13}\text{C}]$oleic acid were higher during 5 h of emulsion digestion ($P$-meal $= 0.018$ and $P$-time×meal $= 0.0002$). For both tracers, a second peak was observed 360 min after ingestion of the second meal.

During the first 300 min for obese subjects, the iAUC for plasma $[^{13}\text{C}]$FAs were significantly higher after consumption of emulsion compared with spread fat ($P < 0.05$; Figure 2, B and D). The iAUC after spread fat consumption was lower in the obese than in the NW subjects ($P < 0.05$; Figure 2, A–D). Fecal excretion of $[^{13}\text{C}]$palmitic acid was higher than that of $[^{13}\text{C}]$oleic acid (Figure 2E). No effect of breakfast type on fecal excretion of $[^{13}\text{C}]$palmitic acid or $[^{13}\text{C}]$oleic acid was found in the 2 groups.

**Plasma concentration profile of insulin and NEFAs and apparent dietary fatty acid spillover**

A significant change was shown in plasma insulin and NEFAs over time after the 2 breakfasts in both groups ($P$-time $< 0.0001$; Figure 3, A–D). Over the first 300 min, the NEFA profile indicated a meal type × time interaction in both groups, and the decrease in plasma NEFAs at 120 min was lower after emulsion than after spread consumption (Figure 3, C and D). Therefore, we measured $^{13}$C enrichment in plasma NEFAs at 120 min (Figure 3E) to estimate whether this would result from the contribution of exogenous FAs—the so-called apparent FA spillover. We observed higher apparent “spillover” during the postprandial phase of the emulsion than of the spread fat in both groups ($P < 0.05$ for NW and $P < 0.01$ for obese subjects; Figure 3E). The contribution of exogenous FAs to total NEFAs in NW subjects was 42% for the spread compared with 79% for the emulsion and was lower in the obese: 4% for spread compared with 50% for emulsion (meal effect, $P < 0.01$; BMI effect, $P < 0.01$; no meal × BMI interaction). Moreover, the proportion of exogenous FAs in nonesterified form in plasma at 120 min was 10.6% for the spread compared with 18.7% for the emulsion (ie, 1.8-fold increase) in the NW subjects and 10.1% for the spread compared with 15.0% for the emulsion (ie, 1.5-fold increase) in the obese subjects ($P < 0.05$ for spread compared with emulsion; no significant effect of BMI nor meal × BMI interaction). In the same time, emulsification increased total plasma NEFAs by 2.2-fold in the NW subjects and by 1.6-fold in the obese subjects (Figure 3, C and D). Overall, this means that >80% of the increase in plasma NEFAs due to emulsification may be explained by an increased amount of exogenous FAs being released in nonesterified form in the plasma (spillover).

---

**TABLE 3**

<table>
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<tr>
<th>Structure (type of breakfast)</th>
<th>Droplet size $^3$</th>
<th>$d_{32}^4$</th>
<th>Melting temperature $^5$</th>
</tr>
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<tr>
<td></td>
<td>$\mu m$</td>
<td>$\mu m$</td>
<td>$m^3$</td>
</tr>
<tr>
<td>Spread</td>
<td>1.04</td>
<td>0.63</td>
<td>0.006</td>
</tr>
<tr>
<td>Emulsion</td>
<td>1.04</td>
<td>0.63</td>
<td>0.006</td>
</tr>
</tbody>
</table>

$^1$Mixture of milk + $[^{13}\text{C}]$triacylglycerol tracers.

$^2$Diameter of the peak of maximum intensity measured by dynamic light scattering.

$^3$Surface averaged diameter measured by laser-light scattering.

$^4$Spread fat: calculated as the surface of an equivalent sphere of 40 g. Emulsion: calculated from the specific surface area ($m^2/g$ fat) calculated by the software, further multiplied by fat content in the meal.

$^5$Temperature at which the entire fat amount is in liquid form.
FIGURE 1. Postprandial profile after the consumption of spread fat or emulsion: CMRF-triacylglycerols in NW (A) and Ob (B) subjects and corresponding iAUC and CMRF particle size in NW (C) and Ob (D) subjects, and ApoB48 in NW (E) and Ob (F) subjects. Data are means ± SEMs; n = 9 per group; P-time, P-meal, and P-timexmeal for the postprandial period from 0 to 300 min (repeated-measures ANOVA followed by post hoc Fisher’s protective least-squares difference). B: **P < 0.01 for emulsion compared with spread fat (paired Student’s t-test); *P < 0.05 for emulsion compared with spread fat at time 480 min and for iAUC0–300 (paired Student’s t-test); *P < 0.05 for Ob compared with NW regarding spread fat iAUC300–480 (unpaired Student’s t-test). D: *P < 0.05 for time 120 min emulsion compared with spread fat (paired Student’s t-test). C and D: **P < 0.01 for time 120 min Ob compared with NW subjects (unpaired Student’s t-test); E and F: *P < 0.05 for time 120 min emulsion compared with spread fat (paired Student’s t-test), **P < 0.01 for time 120 min Ob compared with NW subjects (unpaired Student’s t-test). ApoB48, apolipoprotein B-48; CMRF, chylomicron-rich fraction; iAUC, incremental AUC; NW, normal-weight; Ob, obese; TAG, triacylglycerol; Δ, change.
<table>
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<tr>
<th>Variable</th>
<th>Normal weight</th>
<th>Obese</th>
<th>Normal weight</th>
<th>Obese</th>
<th>P-value</th>
<th>P-meal(^2)</th>
<th>P-BMI(^2)</th>
<th>P-meal(^2) × BMI(^2) × time(^2)</th>
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<tr>
<td>0–480 min</td>
<td>ΔCmax (mmol/L)</td>
<td>0.63 ± 0.13</td>
<td>0.80 ± 0.12</td>
<td>0.75 ± 0.09</td>
<td>0.94 ± 0.25</td>
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<tr>
<td></td>
<td>iAUC (mmol • min/L)</td>
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<td>t(_{\text{max}}) (min)</td>
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<td>207 ± 25</td>
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<td>NS</td>
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<td>Appearance rate(_{0–60\text{min}}) (μmol • L(^{-1}) • min(^{-1}))</td>
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<td>0–300 min</td>
<td>ΔCmax (mmol/L)</td>
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<td>0.53 ± 0.09</td>
<td>0.74 ± 0.09</td>
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<td>iAUC (mmol • min/L)</td>
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<td>300–480 min</td>
<td>ΔCmax (mmol/L)</td>
<td>0.54 ± 0.13</td>
<td>0.80 ± 0.12</td>
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<td>t(_{\text{max}}) (min)</td>
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<td>&lt;0.1</td>
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<td>CMRF particle size</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>0–480 min</td>
<td>d(_{\text{max}}) (nm)</td>
<td>253 ± 34</td>
<td>494 ± 93</td>
<td>262 ± 20</td>
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<td>NS</td>
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<td>t(_{\text{max}}) (min)</td>
<td>243 ± 25</td>
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<td>&lt;0.1</td>
<td>0.1</td>
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<td></td>
<td>Enlargement rate(_{0–60\text{min}}) (μmol • L(^{-1}) • min(^{-1}))</td>
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<td>-0.02 ± 0.11</td>
<td>0.65 ± 0.11</td>
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<td>t(_{\text{max}}) (min)</td>
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<tr>
<td>300–480 min</td>
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<td>207 ± 28</td>
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<td>t(_{\text{max}}) (min)</td>
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<td>(^{13})CO(_2) enrichment</td>
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<td>C(_{\text{max}}) (%)</td>
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<td>0.013 ± 0.001</td>
<td>0.019 ± 0.001</td>
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<td>AUC (± • min)</td>
<td>6.9 ± 0.5</td>
<td>4.8 ± 0.8</td>
<td>7.7 ± 0.2</td>
<td>6.4 ± 0.5</td>
<td>&lt;0.01</td>
<td>&lt;0.001</td>
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<tr>
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<td>t(_{\text{max}}) (min)</td>
<td>310 ± 21</td>
<td>347 ± 23</td>
<td>267 ± 26</td>
<td>267 ± 17</td>
<td>&lt;0.01</td>
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<td>NS</td>
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<tr>
<td>Appearance rate(_{0–60\text{min}}) (%/min)</td>
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<td>2.0 ± 10(^{-3}) ± 0.5 × 10(^{-5})</td>
<td>13.6 ± 10(^{-3}) ± 1.9 × 10(^{-5})</td>
<td>9.8 ± 10(^{-3}) ± 0.8 × 10(^{-5})</td>
<td>&lt;0.0001</td>
<td>&lt;0.01</td>
<td>NS</td>
<td></td>
</tr>
</tbody>
</table>

(Continued)
Postprandial appearance of label in expired carbon dioxide

The appearance of $^{13}$C in expired carbon dioxide represents the final product of FA $\beta$-oxidation. A significant change in APE occurred over time in both groups ($P$-time < 0.0001; Figure 4, A and B). In both groups, the APE was higher after consumption of emulsion than after spread fat over the first 300 min, which indicated an improvement of dietary fat $\beta$-oxidation after consumption of the emulsified form. The APE returned to baseline after 720 min. An effect of BMI was also observed, with a higher appearance rate0–60min in the NW than in the obese subjects ($P < 0.01$; Table 4).

As shown in the insets in Figure 4 (A and B), the AUC of expired $^{13}$CO$_2$ after 300 min was significantly higher with the emulsion than with the spread fat in both groups ($P < 0.01$ for NW subjects and $P < 0.001$ for obese subjects). Besides, over 0–720 min, obese subjects had a higher AUC of expired $^{13}$CO$_2$ after consumption of emulsion than after consumption of spread fat ($P < 0.05$; Figure 4B, inset). Overall, the structure of fat in the meal significantly affected the kinetic parameters of $^{13}$CO$_2$ air enrichment regardless of BMI, whereas BMI affected AUC and appearance rate0–60min (Table 4).

Exogenous lipid fate

We studied the metabolic handling of exogenous lipids by evaluating the fractions of ingested lipids that had been either oxidized or lost in feces, and so estimated the remaining fraction stored in body pools. For the same quantity and composition of ingested fat, all subjects $\beta$-oxidized FA better when fat was emulsified (Figure 4C). In turn, the calculated fraction of ingested lipids oriented toward storage in body pools was lower after consumption of fat ($P < 0.05$; Figure 4B, inset). After the portion of exogenous lipids lost in feces (Figure 4C) was accounted for, the percentage of exogenous lipid oxidation according to the fraction that was intestinally absorbed was higher for NW and obese subjects after consumption of spread fat than after spread consumption. The appearance of $^{13}$C in expired carbon dioxide represents the final product of $\beta$-oxidation of FA, all subjects $\beta$-oxidized FA better when fat was emulsified (Figure 4C). In turn, the calculated fraction of ingested lipids oriented toward storage in body pools was lower after consumption of fat ($P < 0.05$; Figure 4B, inset). Overall, the structure of fat in the meal significantly affected the kinetic parameters of $^{13}$CO$_2$ air enrichment regardless of BMI, whereas BMI affected AUC and appearance rate0–60min (Table 4).

DISCUSSION

Postprandial triglyceridemia is the first step in the metabolism of dietary lipids. Ingested FAs are first present in plasma triglycerides in the form of intestinally secreted chylomicrons, which further leads to large remnants after hydrolysis by lipoprotein lipase (10, 22). The next step concerns trafficking of FAs toward $\beta$-oxidation or storage, which is of utmost importance regarding the metabolic effect of these dietary FAs. Therefore investigated whether structuring fat in the meal could modify postprandial lipid metabolism, from the amount and size of chylomicrons to $\beta$-oxidation, including fecal loss. To this aim, labeled breakfasts containing either spread or emulsifed fat were fed to NW and obese subjects. The test meals were designed to be of equal composition. Thus, factors such as FA composition or protein content were not
FIGURE 2. Postprandial concentration profile and iAUC of total plasma lipids of [13C]palmitic acid and [13C]oleic acid in NW (A and C, respectively) and Ob (B and D, respectively) subjects who consumed spread fat or emulsion. E: Fecal excretion of [13C]palmitic acid and [13C]oleic acid in NW and Ob subjects who consumed spread fat or emulsion. Data are means ± SEMs; n = 9 per group. A–D: P-time, P-meal, and P-time×meal for postprandial period from 0 to 300 min (repeated-measures ANOVA followed by post hoc Fisher’s protective least-squares difference). A and B: *P < 0.05 for Ob iAUC 0–300 min emulsion compared with spread fat (paired Student’s t test); 1P < 0.05 for spread fat iAUC 0–300 min Ob compared with NW subjects (unpaired Student’s t test); 2P < 0.1 for Ob iAUC 0–480 min emulsion compared with spread fat (paired Student’s t test). C and D: *P < 0.05 for Ob iAUC 0–300 min emulsion compared with spread fat (paired Student’s t test); 1P < 0.05 for spread fat iAUC 0–300 min Ob compared with NW subjects (unpaired Student’s t test). E: different lowercase letters indicate a statistically significant difference, P < 0.001 (ANOVA followed by post hoc Bonferroni test). iAUC, incremental AUC; NW, normal-weight; Ob, obese.
involved in the currently observed differences in lipid metabolism, which can be uniquely attributed to the physicochemical structure of fat in the meal. The postprandial chylomicron triacylglycerol profile after emulsion consumption differed from that of the spread fat, with the peak being more rapidly achieved, more pronounced, and more quickly cleared, especially in obese subjects. This is consistent with reports of enhanced FA absorption when a simple bolus of vegetable oil was emulsified in humans (27) and rodents (14, 15, 28). One explanation was that the surface area of our emulsion was $70,000$ times greater for lipases than for spread fat, which is reported to enhance lipolysis and absorption (29). Enteral emulsions of different droplet sizes, $\sim 1$ compared with $\sim 10 \mu m$ (14.5-fold difference in fat surface area), were shown to result in small differences only in postprandial

**FIGURE 3.** Postprandial concentration profile of insulin and NEFAs in NW (A and C, respectively) and Ob (B and D, respectively) subjects who consumed spread fat or emulsion. E: $^{13}C$ enrichment of plasma NEFAs at 120 min, estimating so-called apparent fatty acid spillover in NW and Ob subjects who consumed spread fat or emulsion. Data are means ± SEMs; $n = 9$ per group. P-time, P-meal, and P-time×meal for postprandial period from 0 to 300 min (repeated-measures ANOVA followed by post hoc Fisher’s protective least-squares difference). C: $^{*}P < 0.01$ for NW subjects at 120 min emulsion compared with spread fat (paired Student’s t test). D: $^{*}P < 0.05$ for Ob subjects at 120 min emulsion compared with spread fat (paired Student’s t test). E: $^{*}P < 0.05$ for NW subjects at 120 min emulsion compared with spread fat (paired Student’s t test); $^{**}P < 0.01$ for Ob subjects at 120 min emulsion compared with spread fat (paired Student’s t test); ANOVA showed a meal effect ($P < 0.01$) and BMI effect ($P < 0.01$) but no significant meal × BMI interaction. NEFAs, nonesterified fatty acids; NW, normal-weight; Ob, obese.
FIGURE 4. Postprandial profile of $^{13}$C appearance in breath in NW (A) and Ob (B) subjects who consumed spread fat or emulsion. C: Exogenous lipid fate—oxidized, lost in feces, or stored [calculated as total – (lost + oxidized)]—in NW and Ob subjects over 480 min. D: Oxidation of intestinally absorbed lipids over 480 min in NW and Ob subjects who consumed spread fat or emulsion. E: Total lipid oxidation (total bar) and fraction of cumulative exogenous lipid over 480 min after the consumption of test breakfasts in NW compared with Ob subjects. Data are means ± SEMs; $n = 9$ per group. C–E: *$P < 0.05$ for NW subjects and **$P < 0.01$ for Ob subjects emulsion compared with spread fat (paired Student’s t test). ANOVA showed no significant meal × BMI interaction ($P = 0.087$). NW, normal-weight; Ob, obese.
lipemia in humans (17). The dramatic differences observed in the current study were due to the greatest differences in fat structure. Of note, postprandial lipid metabolism was previously found to be faster with unemulsified than with emulsified milk fat in rats (14). Differences in the current results can be explained by 1) rodent physiology of bile flow, which is different from that of humans (30), and 2) unemulsified melted milk fat being force-fed intragastrically before the proteinaceous phase. This could have favored lipid emptying in the upper intestine and a rapid rise of plasma triacylglycerols in rats. Our study also provides a proof of concept that the effects of fat structure in the meal can occur in a real mixed meal, whereas previous studies used an oil or emulsion bolus fed orally or intragastrically (17, 29). Regarding emulsifier type, our fat was emulsified by the proteins naturally present in skin milk. Emulsions stabilized with caseins and monoacylglycerols were recently reported to result in lower postprandial plasma triacylglycerols than those formulated with lecithin in nonobese humans (31), which can be explained by lower in vitro digestive lipolysis (32). Because obese men were the most affected by emulsification, further work should test the effect of emulsifiers on postprandial lipid metabolism. The importance of sensory exposure to lipids on postprandial metabolism in humans was also recently shown (5). Therefore, we cannot exclude a contribution of oral fat perception in our results.

Elevation of triacylglycerol-rich lipoproteins throughout the postprandial phase is an independent cardiovascular disease risk factor (3, 4), which can be mechanistically related to the atherogenic potential of small chylomicron remnants (21, 22). In this study, we collected CMRF fractions containing chylomicrons and their large remnants. The latter do not contribute significantly to the formation of small atherogenic remnants because of direct hepatic clearance (33). However, the role of triacylglycerol-rich particles is still debatable, which indicates that this issue still needs further study (1, 2). In this context, our study showed for the first time in NW and obese men that the postprandial lipemia profile can be modulated by structuring the fat in a mixed meal. Therefore, the study of atherogenic small particles in the postprandial phase after consumption of differently structured lipids in the meal should now be performed.

Obese men presented a delayed increase of CMRF-triacylglycerols after spread fat. Overweight men were also reported to have delayed triacylglycerol-rich lipoprotein metabolism after a high-fat load (34). Our observed differences between the 2 breakfasts in obese men could be explained by their lower pancreatic secretion and lower levels of gallbladder emptying compared with lean men (35). Therefore, obese subjects can better hydrolyze fat when it is preemulsified. Moreover, fine stable emulsions were reported to be emptied faster and to cause greater release of cholecystokinin than those that broke and layered in the stomach (36, 37). We thus suggest that the delay in fat absorption observed with the spread fat was due to layering in the stomach and thus to delayed emptying. Of note, immediately after lunch, a peak of [13C]FAs appeared in plasma. This so-called second-meal effect is known as the contribution of lipids from a meal to lipemia after the next meal (38). For obese men, the marked delay in lipemia appearance after spread fat cumulated with the second meal effect, causing high lipemia until the test ended. In contrast, obese subjects (without fasting hyperlipidemia herein) did not have difficulties in absorbing the emulsion, with a final return of lipemia to baseline.

In NW subjects, higher CMRF-triacylglycerols after the emulsion corresponded transiently to more particles, as shown by the similar CMRF particle size with an increased apolipoprotein B-48 concentration at 120 min. In obese subjects, however, apolipoprotein B-48 concentrations remained similar, i.e., the increase in lipemia after emulsion was due to increased CMRF particle size. High particle numbers estimated by apolipoprotein B-48 concentrations are reported to lead to increased chylomicron remnant numbers and hence a potentially increased atherosclerotic risk (10, 21). It would now be useful to explore the chronic metabolic effect of fat structure, especially regarding apolipoprotein B-48–containing particles.

The few reports about metabolic effects of emulsions have solely studied lipemia or plasma FA concentrations as endpoints. For the first time to our knowledge, our study shows that fat emulsification further affects the metabolic handling of exogenous FAs, including β-oxidation. Early appearance of [13C]FA was due to the rapid β-oxidation of short-chain FAs, which are directly absorbed in the portal vein and oxidized by the liver (39). Obesity is associated with a defect in the β-oxidation of dietary FAs (18, 40–42). Hodson et al (43) recently challenged this idea by showing greater FA β-oxidation in obese men and attributed this to specific FA partitioning. We highlight that exogenous FA oxidation can be enhanced in obese men by emulsifying fat. Disparities between reports can thus be explained by the current “fast vs slow lipid” notion. Indeed, lower β-oxidation in obese than in lean subjects was observed by using a single oil bolus (18), whereas higher β-oxidation in the obese subjects was observed when the tracer was dispersed into an emulsion (43). This aspect had not been taken into account by previous authors. Moreover, emulsification is now advised to enhance the intestinal absorption of essential FAs (27). However, our results highlight the risk that such essential FAs that are quickly absorbed can be lost in the β-oxidation process rather than being bioavailable for cell membrane turnover. Therefore, further studies on the structuring of oils rich in essential PUFAs should now investigate their final postprandial metabolic fate.

The effect of emulsification on exogenous lipid oxidation cannot be due to differences in intestinal absorption because of similar fecal excretion. Total lipid oxidation during the test day was unchanged by fat structuring, as well as total energy expenditure, and diet-induced thermogenesis. However, the source of β-oxidized FAs was different: with the emulsion, exogenous FAs ingested at breakfast were shunted toward β-oxidation pathways. With the spread fat, more endogenous FAs and/or exogenous FAs ingested at lunch were oxidized so that exogenous FAs ingested at breakfast were more oriented toward storage. Therefore, regardless of energy balance, FA metabolism is changed by lipid structure. This finding is consistent with the greater FA spill-over after emulsion consumption. This can be explained by the faster intestinal absorption, which resulted in enhanced lipolysis of chylomicrons that generated exogenous NEFAs (44). Their early influx can serve as fuel for tissues and explain their higher contribution to total FA oxidation with emulsion. However, high NEFAs can also constitute a risk of ectopic fat accumulation (44). Another aspect in obesity research concerns energy balance and satiety regulation (45, 46). Just before lunch, our obese subjects felt hungrier after emulsion than after spread fat consumption. Further trials could test the effect of fat structuring at breakfast on satiety regulation at lunch and energy balance.
In summary, we showed that the postprandial metabolic handling of dietary FAs can be significantly modified by emulsifying the fat in the meal, especially in obese subjects. The clinical perspectives of this first study should thus not be underrated. This study supports the further exploration of a possible dietary concept of “fast vs slow fat” for the nutritional management of metabolic diseases through food formulation. Our results in the postprandial phase raise the questions of whether 1) daily ingestion of “fast vs slow fat” would result in different lipid metabolism, adiposity, and/or cardiovascular disease risk markers in the long term, and 2) the composition and structuring of dietary lipids could be optimized to this aim.

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