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# Gut emptying affects dietary fat contribution to postprandial lipemia following sequential meals in healthy subjects

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**Abstract**

**Objective:** The present study examined the kinetic of plasma triacylglycerol (TAG) and gut emptying after sequential ingestion of breakfast and lunch, and the contribution of dietary fat ingested at breakfast to subsequent TAG after lunch.

**Methods:** Nine subjects ingested a breakfast (0730 h) and a lunch (1200 h) containing 25 and 44 g of fat, respectively. [1-$^{13}$C] palmitate was added in breakfast only. Plasma TAG and chylomicron-TAG (CM-TAG) concentrations and [1-$^{13}$C] palmitate enrichment were sequentially measured. On a consecutive day, an identical breakfast labeled with $^{123}$I-Lipiodol was ingested, followed by a lunch for three controls. $^{123}$I-Lipiodol dynamics was followed in vivo by scintigraphic imaging focused on the stomach, small bowel, and thoracic duct arch.

**Results:** An early rise in plasma and CM-TAG was observed after lunch ingestion. After breakfast, [1-$^{13}$C] palmitate enrichment was maximal 150 and 210 min in plasma TAG and CM-TAG, respectively, decreased thereafter, and increased rapidly (50 min for plasma TAG and 30 min for CM-TAG) after lunch ingestion. Scintigraphic imaging appeared to show that fat ingested at breakfast was retained in part within the gut at lunch time. For the three subjects who ingested a lunch, a decrease of activity in the stomach and small bowel and a tendency for increased activity in the thoracic arch were observed.

**Conclusion:** Contribution of fat ingested at breakfast to lipemia after lunch is confirmed. Fat ingested at breakfast was partly retained within the gut and was mobilized after lunch ingestion, as assessed by acceleration of gut emptying and thoracic duct flow after lunch. 

*Keywords:* Postprandial lipemia; Chylomicron; Gastric emptying; [1-$^{13}$C] palmitate; Triacylglycerol

## Introduction

Prolonged and exaggerated postprandial lipemic response, expressed as plasma triacylglycerol (TAG) changes after meal ingestion, has been recognized as a risk factor for coronary artery disease [1–4] and carotid atherosclerosis [5,6]. Postprandial TAG-rich lipoproteins and their remnants are atherogenic, directly and indirectly by influencing the metabolism of other lipoproteins [7,8]. Thus, understanding the factors that influence postprandial TAG-rich lipoprotein metabolism is crucial.

Many studies on postprandial changes of plasma TAG concentrations after fat ingestion have been designed using

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a single test meal. They usually demonstrated a TAG peak occurring 3–4 h after meal ingestion, with a slow return to baseline value afterward. Nevertheless, free-living Western individuals consume several meals per day, and they are on a postprandial state for at least 17 h of a 24-h period [9]. Thus, the postprandial state, rather than the fasting state, should be recognized as the usual metabolic state [10], and data from studies with a single meal do not reflect the metabolic events after sequential meals. Therefore, data on the origin of plasma TAG peaks in response to a high-fat meal cannot be extrapolated to those obtained in response to sequential meals.

When sequential meals were initially tested (pretest + test meals), a biphasic pattern of plasma TAG response to fat ingestion was observed [11,12]. Further investigations showed that fat from a breakfast contributed to TAG response to a lunch eaten 4–6 h later [13,14], in the form of an early postprandial plasma TAG peak. We previously reported that this "second meal effect" could be still observed when the interval between meals was prolonged up to 7 h [15], even though an early peak after the second meal was not observed. Overall these data support the hypothesis that the second meal provokes the release into the circulation of chylomicrons (CMs) containing fatty acids derived from the previous meal and this has been observed after carbohydrate [16], low-fat [14], or high-fat [13,17] meal ingestion. It has been proposed that these CMs derived from stored lipids within the enterocyte and/or intestinal lymphatics [17]. In addition, the contribution of dietary fat to postprandial TAG response during sequential meals might also be due to incomplete fat digestion and/or gastric and/or intestinal emptying delay between meals but such a possibility has not been challenged previously.

In the present study, we tested the hypothesis that part of the fat ingested at breakfast was still present in the gut at the time lunch was ingested and was released into the circulation after lunch. For this purpose, the rate of gastric/small bowel emptying was measured after the ingestion of a test meal containing 25 g of fat labeled with an iodized oil composed of ethyl esters of poppy seed oil labeled with $^{123}I$ ($^{123}I$-Lipiodol). The contribution of dietary fat ingested at breakfast to subsequent lipemia after lunch was also investigated using [1-$^{13}C$] palmitate as a marker of ingested fat at breakfast [15,18].

## Materials and methods

### *Subjects*

Nine healthy non-smoking male volunteers 19 to 25 y of age (median age 24 y) with body mass indexes of 19.7–24.9 kg/m$^2$ (median 22.3) were studied. Their good health was assessed from a detailed medical history, a thorough physical examination, an electrocardiogram, a routine biochemical screening of blood samples (fasting plasma glucose, TAG, and total cholesterol), and an oral glucose tolerance test (75 g). All subjects were non-smokers. Usual dietary habits were estimated from a 7-d dietary recall. They were asked to keep their usual diet before the test and to avoid intensive physical activity for 3 d before tests. All subjects signed an informed consent and were paid for their participation in the study. The human investigations ethics committee of our institution approved the experimental protocol according to current French regulations. Subjects were admitted 2 d consecutively for the two parts of the study.

### *Phase 1 (metabolic study)*

After a 12-h overnight fast, subjects were admitted in our metabolic unit at 0630 h. Then, they were placed at rest in a bed and an indwelling intravenous catheter cannula was inserted in a forearm vein. A 9‰ saline infusion was used throughout the study to keep the canula patent. Blood samples were taken at 30, 15, and 1 min before breakfast, which was given at 0730 h. Breakfast contained 18 g of protein, 25 g of fat (sunflower oil), and 62 g of carbohydrates. A half gram of [1-$^{13}C$] palmitate (99 mol% excess; EURISOTOP, Saint-Aubin, France) was added to this breakfast. Blood samples were taken at 30 min and every hour between 0800 and 1200 h. The test meal (lunch), containing 35 g of protein, 48 g of fat including 44 g of olive oil, 127 g of carbohydrate, and 250 mL of water, was given at 1200 h (270 min after breakfast) except for three randomized subjects who did not ingest lunch (controls). No [1-$^{13}C$] palmitate was ingested at lunch. The composition of meals is listed in Table 1. After lunch, blood samples were taken at 30 min, then every 20 min for 2 h, and every hour during the last 5 h of the test. Breakfast and lunch were consumed each within 15 min. All subjects stayed at rest in a bed during the study and were not allowed to sleep.

Table 1
Nutrient composition of meals*

| Meals and food | Carbohydrate (g) | Fat (g) | Protein (g) | Energy (kJ) |
|---|---|---|---|---|
| Breakfast | | | | |
| Bread 80 g | 44 | 0 | 6 | 924 |
| Orange juice 150 mL | 13 | 0 | 1 | 255 |
| Soft white cheese 0% fat 150 g | 5 | 0 | 5 | 294 |
| Sunflower oil 25 g | 0 | 25 | 0 | 940 |
| Total | 62 | 25 | 12 | 2413 |
| Lunch | | | | |
| Egg white raw 60 g | 0 | 0 | 6 | 100 |
| Egg yolk raw 12 g | 0 | 4 | 2 | 184 |
| Pasta uncooked 150 g | 110 | 0 | 18 | 2140 |
| Tomato sauce 400 g | 12 | 0 | 4 | 268 |
| Yogurt 0% fat 125 g | 5 | 0 | 5 | 167 |
| Olive oil 44 g | 0 | 44 | 0 | 1655 |
| Total | 127 | 48 | 35 | 4514 |

* Determined from food composition tables [29]. [1-$^{13}C$] palmitate was mixed with soft cheese and oil and ingested at lunch (0730 h).