Comparison of the metabolic fate of an oral long-chain triglyceride (TG) (LCT) load and a medium-chain TG (MCT) load in healthy humans. C Binnert [1], C Pachiaudi [1], M Beylot [1], P Chantre [2], J Goudable [1], JP Riou [1], M Laville [1] *([1] CRNH and Inserm 449, Lyon; [2] Laboratoires Arkopharma, Nice, France)*

Since carnitine plamitoyl transferase I (CPT I) is not required for the oxidation of MCT, MCT are preferentially oxidized rather than stored. Thus, MCT could be potentially interesting for the diets of obese patients, especially if they enhance total lipid oxidation. We compared the metabolic fate of 30 g of olive oil (LCT) with a mixed load of 15 g of olive oil + 15 g of MCT oil (80% of trioctanoate) in ten healthy women (23 ± 2 years, 21 ± 2 kg/m^2). In order to calculate the fraction of the load oxidized, we added 200 mg [1-1-1-$^{13}C_3$]triolein to the LCT load or 150 mg [1-1-1-$^{13}C_3$]trioctanoate to the MCT-LCT load.

Each protocol lasted 630 min. At T_{360} min the subjects were given a nonlabelled mixed meal in order to avoid any starvation effects. Indirect calorimetry measurements were performed throughout the test. Blood samples were collected every 30 min to measure metabolite concentrations. Expired gas samples were collected every 30 min for ^{13}C enrichment of CO_2 measurements ($^{13}CO_2$) in order to calculate the fraction of ingested TG having been oxidized.

After the ingestion of LCT, plasma TG levels increased with a peak at 180 min (1.4 ± 0.2 mM vs 0.8 ± 0.1 mM) but not after the MCT-LCT load (T_{240}: 0.9 ± 0.1 mM). NEFA concentrations increased more rapidly with the LCT load than with the MCT-LCT load (T_{120}: 0.63 ± 0.06 mM vs 0.39 ± 0.03, respectively, $P < 0.05$). On the contrary, ketone body concentrations increased as early as 30 min with the MCT-LCT load (T_{30}: 0.13 ± 0.02 mM). Insulinemia moderately but significantly increased during both protocols (T_{90}: 11 ± 1 mUI/L vs 8 ± 1 mUI/L at T_0, $P < 0.05$). The kinetics of the appearance of $^{13}CO_2$ was more rapid with the MCT-LCT load, and the amount oxidized was greater: 82 ± 4% of the MCT load was oxidized within 630 min but only 38 ± 3% fo the LCT load ($P < 0.01$). Total lipid oxidation was moderately and significantly increased during the MCT-LCT load: 21.3 ± 1.1 g vs 17.8 ± 1.4, $P < 0.01$.

Our results showed that a fraction of the MCT-LCT load was preferentially oxidized (80% of the 15 g ingested) and the total lipid oxidation was greater for the MCT-LCT load compared with the LCT load. Thus, MCT seems to be interesting for use in the diets of obese patients, if our results are confirmed in this pathology and during a mixed load.

Influence of meal time on postprandial lipemia. J Dallongeville [2], C Le Fur [1], P Lebel 2, JL Edme [3], JC Fruchart [2], M Romon [1] *([1] Service de nutrition, CHRU-Lille; [2] Serlia, Institut Pasteur de Lille; [3] Cereste, CHRU-Lille, LIlle, France)*

The goal of our study was to assess the influence of the actual time of a meal on postprandial lipemia. Nine healthy subjects aged 19 to 32 years were given a meal at 1:00 pm or 1:00 am in a random order. The sessions were given at 1 to 3 week intervals. The meal contained 40% of daily energy expenditure (15% protein, 40% lipid, 45% carbohydrate). The experimental meal was given 4 h after a standardized meal. Blood samples were drawn at baseline and hourly for 8 h (T_0 to T_8). Plasma total cholesterol (C), very low density lipoproteins (VLDL)-C, low density lipoproteins (LDL)-C, high density lipoproteins (HDL)-C, triglycerides (TG), VLDL-TG, and the LpE:B and LpC-III:B particles were measured postprandially. A two-way analysis of variance ('time of meal' and 'postprandial time') with two repeated measures was used for sta-

tistical analysis. The increase in TG ($P < 0.001$), VLDL-TG ($P < 0.001$) and VLDL-C ($P < 0.001$) was greater for the pm than the am meal. VLDL-TG was greater ($P < 0.02$) at T_1 (+49%) and T_2 (+23%) after the pm meal than after the am meal. The LpE:B levels (markers of TRL remnants) were higher ($P > 0.05$) at T_1 (+30%) and T_2 (+23%) after the pm meal than after the am meal. Conversely, LDL-C levels were lower ($P < 0.05$) following the pm meal at T_5 (–13%), T_6 (–17%), T_7 (–22%) and T_8 (–17%) than after the am meal. There were no statistically significant differences between am and pm for HDL-C and LpB:C-III. The results of the present study showed clear differences in postprandial lipemia between an am and pm meal. These observations support the concept of a slower TRL metabolism during the night as compared within during the day.

Measurement of the overall digestibility of carbon from carbohydrates following the ingestion of ^{13}C-carbohydrates in patients with a malabsorption syndrome.

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Dietary carbohydrates (CHO) that are not absorbed in the small intestine are fermented in the colon, producing gases (CO_2, H_2), short-chain fatty acids and bacterial mass. Currently, there is no available method to determine precisely the amount of unabsorbed dietary CHO and their fermentation products excreted in stools. Since carbon is present in CHO and a majority of fermentation metabolites (CO_2, short-chain fatty acids, bacterial mass, etc), its labelling and measurement could be an interesting method to use in the investigation and follow-up of patients with a malabsorption syndrome. For this purpose, patients with a malabsorption syndrome and normal subjects were given orally CHO naturally enriched with ^{13}C and we measured the overall excretion of ^{13}C in the stools and breath ($^{13}CO_2$). Ten patients with the short bowel syndrome (SBS) (length 73 ± 40 cm, mean ± SD) with ($n = 8$) or without ($n = 2$) the colon remaining in continuity and eight healthy volunteers (HV) ingested a test meal after an overnight fast (3 318 kJ, protein-fat-CHO, 17:26:57% energy) containing naturally ^{13}C enriched CHO in the form of 50 g corn starch (–12.22 8‰), 50 g sugar cane (–11.10 8 ‰) and carmine red as a faecal recovery marker. Patients consumed a low-^{13}C diet and their stools were collected 1 day before and 3 days after the test meal. Breath samples were collected half-hourly and CO_2 production was measured using indirect calorimetry hourly for 6 h before and after the test meal ingestion. In the breath, in the premeal and red postmeal stools, ^{13}C enrichment was measured by an isotope ratio mass spectrometer (GC/CN-IRMS, Europa Scientific). Faecal and breath ^{13}C enrichment was corrected using the basal premeal value.

The excess faecal output of ^{13}C was 142 ± 85 µmol (range 14–299) and 6 ± 7 µmol (range 0–16) in SBS and HV, respectively. The percentage of ingested dose recovered in the stools was 23 ± 18% (range 3–61) and 1 ± 1% (range 0–3) in SBS and HV, respectively. The percentage of ingested dose exhaled in breath for 6 h was 31 ± 9% (range 16–42) and 25 ± 2% (range 20–29) in SBS and HV, respectively. In SBS, the percentage of ^{13}C excreted in stools was inversely related to that excreted in breath for 6 h (Spearman correlation Rho = –0.85, $P < 0.02$).

It was concluded that the faecal measurement of ^{13}C after the ingestion of a breakfast containing CHO naturally enriched with ^{13}C is a useful tool for assessing both the fat of CHO and their carbon containing fermentation products after their passage