values from the non-steady state equation to the steady state at plateaus. The acetate endogenous fluxes did not change during cold acetate infusions of 7 and 14 $\mu mol.kg^{-1}.min^{-1}$ (6 ± 2 et 5 ± 2 $\mu mol.kg^{-1}.min^{-1}$). The *P* values converged to 0.7. There were no differences between turnover calculated with the Steele equation and the steady state. The endogenous flux fell down to 1 $\mu mol.kg^{-1}.min^{-1}$ when the total flux of acetate was higher than 21 $\mu mol.kg^{-1}.min^{-1}$.

We concluded that the acetate fluxes calculated from the Steele equation were not different from the steady state values, when the endogenous acetate flux was stable and the acetate concentration lower than ≈ 600 $\mu mol.L^{-1}$.

**Evidence for a defect of the oxidation of an oral long-chain triglycerides (LCT) load in obese subjects: interest of medium-chain triglycerides (MCT).** C Binnert [1], C Pachiaudi [1], M Beylot [1], D Hans [1], P Chantre [2], JP Riou [1], M Laville [1] *([1] CRNH and Inserm 449, Faculté de médecine Laënnec, 69008 Lyon; [2] Laboratoires Arkopharma, Nice, France).*

Obesity is characterized by an excess of adipose tissue, coming for a large part from dietary triglycerides (TG). We studied in eight control subjects and eight obese subjects (BMI = 21.2 ± 0.9 and 32.9 ± 2.5 $kg/m^2$) the metabolism of 30 g of LCT labeled with 200 mg of [1-1-1-$^{13}$C]triolein for 6 h. Another study with a mixed MCT-LCT load (50%) with 150 mg of [1-1-1-$^{13}$C]trioctanoin was also realized. Indirect calorimetry measurements were performed throughout the test, and blood sampling every 30 min in order to measure the isotopic enrichment in $^{13}$C in the TG fraction of chylomicrons (CM-TG) and in the non-esterified fatty acid (NEFA) fraction. 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 LCT load the amount of lipids oxidized was negatively correlated with fat mass, (measured by dual X-ray absorptiometry), $r = -0.75$, $P < 0.01$. The oxidation of the load was correlated with the appearance of exogenous NEFA in the plasma: correlation between the area under the curve of $^{13}$C-NEFA concentrations and the oxidation of the load: $r = -0.84$, $P < 0.01$, and ranged from 1.7 g to 8.5 g. On the contrary, the oxidation of the MCT moiety of the MCT-LCT load was not correlated with fat mass ($r = 0.22$). The MCT load was more oxidized than the LCT one (59.4 ± 2.5% vs 16.2 ± 1.7%, $P < 0.01$).

In conclusion, our results showed that: i) obesity is associated with a deficit in LCT oxidation but not with MCT; ii) this deficit was probably due to a deficit of appearance of NEFA coming from ingested TG probably due to an excessive uptake of NEFA by the adipose tissue.

**Influence of obesity and body fat distribution on postprandial lipemia in obese and lean women.** N Mekki [1], MA Cristofillis [2], C Atlan-Gepner [2], M Charbonnier [1], C Juhel [1], H Portugal [3], AM Pauli [3], B Vialettes [2], D Lairon [1] *([1] Unité 130, Inserm, 18, av Mozart; [2] Hôpital Ste Marguerite, service de nutrition; [3] Hôpital Ste Marguerite, laboratoire central d'analyses, av Viton, 13009 Marseille, France).*

It has already been shown that accumulation of upper body (abdominal) fat is associated with metabolic complications [Després (1994) Baillière's Clinical Endocrinology and Metabolism 3, 629] but it is not known how body fat repartition would influence postprandial lipemia in obese adults. Thus, this study explored the effect of waist-to-hip conference ratio