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Many studies have shown the hypocholesterolemic effect of legumes due to their high fiber content, which produces an increased fecal cholesterol excretion. On the other hand, it has been proposed that dietary proteins can modify plasma cholesterol concentration.

The present work was designed in order to compare the effects on this parameter of the incorporation of a legume, faba bean (*Vicia faba*) or its protein concentrate.

For this purpose a model of dietary hypercholesterolemy was created by using a high-saturated fat diet (36% of total energy) which provided 10 g/kg diet of cholesterol and 5 g/kg diet of cholic acid. Hypercholesterolemic rats were divided into three groups (n = 10): rats fed a hypercholesterolemic diet with casein as protein source (A), rats fed a hypercholesterolemic diet prepared by changing casein by *Vicia faba* (B) and rats fed a hypercholesterolemic diet prepared by changing casein by *Vicia faba* protein concentrate (C). At the end of the experimental period (15 days), animals were sacrificed and blood was collected. ANOVA test was used for statistical analysis.

	A	*B*	*C*
		mg/dL	
Cholest.	135 ± 11^a	85 ± 6^b	98 ± 9^b
HDL-c	38 ± 7^a	37 ± 2^a	32 ± 4^a
LDL-c	86 ± 12^a	32 ± 4^b	53 ± 7^b
TG	45 ± 4^a	46 ± 4^a	47 ± 6^a

Values not sharing the same letter are significantly different, $P < 0.05$

A reduction of plasma cholesterol was observed in rats fed the diet which provided faba bean protein concentrate. This effect was not attributable to the low sulphur aminoacid concentrations of legume proteins because diets were supplemented with 0.1% of methionine. This suggests that other factors related to the aminoacid profile can be involved.

When the legume seeds were used to prepare the diets, a stronger plasma cholesterol reduction was observed probably because the effect of soluble fibers was added to those of proteins.

Supported by UPV/EHU (101, 123-EA140/94).

Non steady state metabolism of acetate in humans: preliminary study. E Pouteau, K Vahedi, B Rakotoambimina, B Messing, B Flourie, M Krempf *(Centre de recherche en nutrition humaine, Hôpital Laënnec, 44035 Nantes; Inserm 290, Hôpital St Lazare, 75010 Paris, France).*

In order to measure acetate turnover with stable isotope in non steady state, the Steele equation was investigated. The active pool (p) fraction of acetate was therefore determined. Five volunteers (men, 31 ± 4 years, 21.7 ± 0.7 $kg.m^{-2}$) were submitted to a low-fiber diet (< 5 $g.day^{-1}$) for 3 days (H_2 < 15 ppm), and were infused with (1-^{13}C] acetate at 1 $\mu mol.kg^{-1}.min^{-1}$ for 225 min. At t = 90 min, exogenous [^{12}C] acetate were infused intravenously at increasing rates of 7, 14 and 21 $\mu mol.kg^{-1}.min^{-1}$ for 45 min each time. Arterialized blood was collected. Isotopic enrichments were performed and concentrations of acetate were determined by gas chromatography/mass spectrometry. Acetate turnover at the steady state was 7 ± 2 $\mu mol.kg^{-1}.min^{-1}$. At the non steady state (from 90 to 225 min), the acetate turnover increased to 12 ± 1, 18 ± 2 et 26 ± 2 $\mu mol.kg^{-1}.min^{-1}$ ($P < 0.05$). Simultaneously, acetate concentrations increased from 178 ± 24 to 318 ± 23, 555 ± 48 and 795 ± 103 $\mu mol.L^{-1}$ ($P < 0.05$). The parameter P was evaluated by steps, comparing turnover

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