of total particle ($P < 0.05$). In contrast, we observed a decreasing FC content of HDL_3: 2.6 ± 0.2 vs $4.4 \pm 0.5\%$ of total particle ($P < 0.05$). Thus, in healthy men fed a cholesterol-poor diet, soy proteins did not act directly on cholesterolemia via LDL as shown in animals and hypercholesterolemic subjects (1-4). However, soy proteins seemed to induce modifications in LDL and HDL_3 composition.

References:

1 Carroll KK (1991) Review of clinical studies on cholesterol-lowering response to soy protein. *J Am Diet Assoc* 91, 820-827

2 Chait A, Brunzell JD, Denke MA et al (1993) Rationale of the diet-heart statement of the American Heart Association: report of the Nutrition Committee. *Circulation* 88, 3008-3029

3 Andersdon JW, Johnstone BM, Cook-Newell ME (1995) Meta-analysis of the effects of soy protein intake on serum lipids. *N Engl J Med* 333, 276-282

4 Sirtori CR, Lovati MR, Mannoti C, Monetti M, Pazzucconi F, Gatti E (1995) Soy and cholesterol reduction: clinical experience. *J Nutr* 125, 598S-605S

Use of soy proteins in cholelithiasis prevention. I Catala [1,2], C Juste [1], K Benfiguig [3], A Ruskone-Fourmestraux [3], B Guy-Grand [4], F Bornet [2], T Corring [1] *([1] LEPSD, Inra, 78352 Jouy-en-Josas cedex, France; [2] Nutrition and Health Service, Eridania Béghin-Say, Vilvoorde Research Center, Vilvoorde, Belgium; [3] Service d'hépato-gastroentérologie, Hôtel-Dieu; [4] Service de nutrition, Hôtel-Dieu, 75004 Paris, France).*

The pathogenesis of cholesterol gallstones (or cholelithiasis) is related to the crystallization of biliary cholesterol. Diet is thought to be one of the factors involved in gallstone formation. Our study represents the first attempt in demonstrating a close relationship between the origin of dietary proteins and cholesterol crystallization from bile in healthy volunteers.

For this purpose, 12 healthy young men aged 29.1 ± 1.6 years, BMI = 22.7 ± 0.9 kg/m^2, who had no gallstones as shown by ultrasonography participated to a cross-over design protocol. The subjects were fed an isocaloric diet where proteins were either mainly from animal origin or mainly from soya origin for two 2-week periods separated by a 2-week interval on their usual diets. At the end of each dietary period, body weight was measured. After an overnight fast, samples of duodenal bile and blood were taken in order to evaluate whether the origin of dietary proteins could have influenced the propensity of bile to crystallize biliary cholesterol and biliary factors implicated in this process. No significant changes in the subjects' body weight and caloric intakes were observed during the 6 weeks of experimental protocol. Total biliary lipids and biliary cholesterol saturation were not influenced by the protein origin, but cholesterol crystallization was retarded ($\approx +4$ days) and decreased (≈ -100 µg crystallized cholesterol/mL bile at equilibrium) with soy proteins compared to animal proteins. Among the intrinsic factors of bile which are possibly responsible for preventing cholesterol precipitation (biliary proteins, molecular species of biliary lecithins and bile acids), the proportion of ursodeoxycholic acid (a bile acid currently used for gallstone dissolution) was shown to be doubled with the soy protein diet. This could partly explain the delay in biliary cholesterol crystallization observed with the soy protein diet.

Hypocholesterolemic effect of a *Vicia faba* protein concentrate in hypercholesterolaemic rats. MA De Diego, MP Portillo, R Cantoral, MT Macarulla *(Department of Nutrition, Faculty of Pharmacy, University*

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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