

Replacing standard dairy produce by low saturated fat equivalents is one of the usual advices reduce plasma cholesterol. Nevertheless, this kind of recommendations results most often in an increase in the dairy protein intake. To test the influence of dairy protein on cholesterol metabolism, we submitted seven healthy volunteers alternatively to two 7-day-diet sequences (cross-over study). One of the diet sequences was devoid of dairy produce and in the other one, half of the proteins came from dairy produce. Calories, glucides, proteins, cholesterol, total lipids, saturated, mono unsaturated and poly unsaturated fatty acids intake were kept constant between the two sequences and were fixed at the level of usual intake. Mean protein intake was  $93 \pm 5$  g/day including  $14 \pm 1$  g/day, none and  $43 \pm 4$  g/day coming from dairy produce in the usual diet, devoided of dairy produce and enriched in dairy produce diets respectively. Each of the diet sequences was separated from the following by a wash-out period when the individuals were going back to their usual diet. At the beginning (d0) and at the end (d7) of each diet sequence, the following parameters were determined in the plasma: total cholesterol, triglycerides, HDL cholesterol, Apo A1 and B lipoproteins, and lathosterol. This latter parameter is considered as an excellent index of in vivo cholesterol synthesis. No significant differences were observed for the plasma lipids between d0 and d7. The sequence enriched in dairy produce did not have any effect on lathosterol level ( $7.9 \pm 1.4$  at d0 vs  $7.8 \pm 1.5$   $\mu\text{mol/L}$  at d7, NS) as the sequence without dairy produce induced a decrease in plasma lathosterol ( $8.6 \pm 1.3$  at d0 vs  $7.0 \pm 1.3$   $\mu\text{mol/L}$  at d7,  $P < 0.02$ ).

These results show that: (i) proteins from dairy produce per se have an effect on cholesterol synthesis; (ii) their suppression induces a reduction in endogenous cholesterol synthesis; (iii) their increase over a given level (14 g/day) does not increase cholesterol synthesis.

**Effects of soy proteins on plasma lipoproteins in healthy men.** S Auboiron<sup>1,3</sup>, I Catala<sup>2,3</sup>, C Juste<sup>2</sup>, FRJ Borne<sup>3</sup>, T Corring<sup>2</sup>, B Guy-Grand<sup>1</sup> (<sup>1</sup> *Nutrition Department, Hôtel-Dieu, 75004 Paris*; <sup>2</sup> *LEPSD, Inra, 78352 Jouy-en-Josas cedex, France*; <sup>3</sup> *Nutrition & Health Service, Eridania Béghin-Say, 1800 Vilvoorde, Belgium*).

The hypocholesterolemic effect of soy proteins has been demonstrated in a variety of animal models, but not so obviously in humans. We have compared the effect of animal versus vegetable proteins on plasma lipoproteins in healthy men. For this purpose, 12 healthy young men ( $29.0 \pm 1.6$  years, BMI =  $22.7 \pm 0.9$   $\text{kg/m}^2$ ) participated in a cross-over design protocol. The subjects were fed an isocaloric diet ( $12\,870 \pm 330$  kJ/day, 15% of total energy of proteins, 51% of carbohydrates, 34% of fat and 300 mg/day of cholesterol) 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 diet.

At the end of each dietary period, after an overnight fasting, blood samples were collected to measure plasma lipids by enzymatic procedures and lipoprotein classes (VLDL, IDL, LDL, HDL<sub>2</sub>, HDL<sub>3</sub>) after separation by density gradient ultracentrifugation.

Body weight and food intake remained constant throughout the study. No difference between animal and soy protein containing diets was observed for plasma lipids (triglycerides (TG): 0.59 mM – 0.58 mM, free cholesterol (FC): 1.29 mM – 1.21 mM, esterified cholesterol (EC): 4.29 mM – 4.39 mM, phospholipids (PL): 2.05 mM – 1.90 mM for animal and soy diets respectively). No variation was noted in the plasma concentration of different lipoprotein classes. But soy proteins induced a decreasing TG content of LDL:  $3.7 \pm 0.4$  vs  $6.2 \pm 0.8\%$  of total particle ( $P < 0.05$ ), while the CE content increased:  $43.0 \pm 0.9$  vs  $38.0 \pm 1.2\%$

of total particle ( $P < 0.05$ ). In contrast, we observed a decreasing FC content of HDL<sub>3</sub>:  $2.6 \pm 0.2$  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<sub>3</sub> composition.

#### References:

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**Use of soy proteins in cholelithiasis prevention.** I Catala<sup>1,2</sup>, C Juste<sup>1</sup>, K Benfiguig<sup>3</sup>, A Ruskone-Fourmestraux<sup>3</sup>, B Guy-Grand<sup>4</sup>, F Bornet<sup>2</sup>, T Corring<sup>1</sup> (<sup>1</sup> *LEPSD, Inra, 78352 Jouy-en-Josas cedex, France*; <sup>2</sup> *Nutrition and Health Service, Eridania Béghin-Say, Vilvoorde Research Center, Vilvoorde, Belgium*; <sup>3</sup> *Service d'hépatogastroentérologie, Hôtel-Dieu*; <sup>4</sup> *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 relation-

ship between the origin of dietary proteins and cholesterol crystallization from bile in healthy volunteers.

For this purpose, 12 healthy young men aged  $29.1 \pm 1.6$  years, BMI =  $22.7 \pm 0.9$  kg/m<sup>2</sup>, 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 ( $\approx - 100$   $\mu$ g 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*