ascribed to intestinal fermentations and their consequences on liver lipid metabolism. When large intestine fermentation levels are low, elevating the rate of fecal steroid excretion seems to have limited effects on plasma lipid concentrations. In various pathophysiological situations, the intake of plant foods (rich in fibers or resistant starch) appears promising as it promotes cholesterol elimination from the body pool.

Comparison of metabolic responses to digestible and partly undigestible starches in healthy humans. L Achour, B Flourié, F Briet, C Franchisseur, F Bornet, JC Rambaud, B Messing *(Inserm U 290, hôpital Saint-Lazare, 75010 and Éridania Béghin-Say, 75008 Paris, France)*

Starch is the main energetic fuel in the human diet. Most starches are extensively digested in the human small intestine. It is now technologically possible to modify starch in order to slow down its digestion in the small intestine. The digestion of technologically modified starches will start in the small intestine and continue in the colon, where its fermentation releases short chain fatty acids (mainly acetate) and gases (H_2, CO_2). The metabolic consequences of this shift in starch digestion is unknown in healthy humans.

In this study, we measured certain metabolic indexes in healthy humans consuming a highly digestible corn starch (Dig S) in the small intestine and the same corn starch after retrogradation (Ret S).

Eight healthy volunteers were studied during two periods separated by 1 week. In each period, fasting volunteers consumed at 8:00 am a 425 kcal test meal in addition to 50 g of either the Dig S or Ret S. Blood and breath were sampled in the absorptive period hourly for 8 h. The same meal was given again the same day at 10:00 pm. At 8:00 am on the next morning, ie, 10 h after the ingestion of the test meal, blood and breath were sampled in the fasting subjects hourly for 3 h ie, in the postabsorptive period.

In the absorptive period, after the ingestion of Dig S, the glycemic index and area under the insulin curve were higher, and blood glycerol concentrations were lower ($P < 0.05$) than after the ingestion of Ret S. In the postabsorptive period, after the ingestion of Dig S the respiratory quotient, $^{13}CO_2$ and H_2 excretion in breath, blood acetate concentrations and satiety index were significantly lower, whereas blood glycerol concentrations were higher ($P < 0.05$) than after the ingestion of Ret S.

In healthy humans, the digestion of Ret S is slow in the small intestine and its colonic fermentation continues 10 to 13 h after its ingestion. Compared to the highly Dig S, the shift in starch digestion induced by retrogradation leads to changes in metabolic responses: Ret S reduces the glycemic and insulinic responses in the absorptive period, and lipolysis in the postabsorptive period. This last effect may be related to an inhibitory action on the lipolysis of short chain fatty acids produced during the colonic fermentation of unabsorbed starch.

Chronic ingestion of a high fat cholesterol diet increased postprandial lipemia and atheroma deposition in New Zealand white (NZW) rabbits. C Juhel, C Dubois, M Senft, D Lairon *(Unité 130-Inserm, National Institute of Health and Medical Research, 18, avenue Mozart, 13009 Marseille, France)*

Several recent human studies have shown the existence of some links between altered patterns of postprandial lipemia and the risk of atherosclerosis. Nevertheless, the mechanisms involved are still unknown. We therefore performed the present study in the NZW rabbit, given its capacity to

develop atheroma plaque within a few months, with the aim of evaluating the relationships between chronic fat intake, postprandial lipemia and atheroma plaque deposition.

Thirteen NZW male rabbits, 12–13 weeks old, weighing 2.20–2.40 kg were fed two different diets: a low fat (3% lipids), no cholesterol (LF, $n = 6$) or a high fat diet (HF, $n = 7$) with 0.17% cholesterol and 5.20% lard. On days 0 and 42 for LF rabbits and on days 14, 28, 42, 63, 84 for HF rabbits, the animals received a test meal (25 g bolus) containing 6 g peanut oil and 200 mg cholesterol with ^{3}H-cholesterol and ^{14}C-triolein. Blood samples were collected after a 24 h fast and 10, 24, 34 and 48 h after the test meal intake. Lipoproteins (chylomicrons + very low density lipoproteins [VLDL] or triglyceride-rich particles, low density [LDL] and high density lipoproteins [HDL]) were isolated by ultracentrifugation. Triglycerides, free and esterified cholesterol and phospholipids were assayed. On day 105, lipoprotein lipase (LPL) and hepatic lipase (HL) activities were measured on postheparin plasma. On day 119, the rabbits were killed and the atheroma plaques were quantified in the aorta.

Fasting lipids and lipoproteins as well as postprandial lipemia after the test meal intake were not modified in the LF rabbits and the extent of atheroma plaque was 0%. The HF diet induced hypercholesterolemia and hyperphospholipemia after a few weeks and led to 30.87 ± 3.76% ($P < 0.05$) of the rabbit aorta being covered by atheroma plaque. In HF rabbits, postprandial plasma triglycerides peaked after 24–36 h and the 0–48 h AUCs triglycerides steadily increased as rabbits became more hypercholesterolemic and were significantly higher than in LFs. Plasma ^{14}C-triolein and ^{3}H-cholesterol concentrations were higher in HF rabbits than in LFs during the postprandial period and were proportional to fasting cholesterolemia. LPL and HL activities were two-fold higher in HF rabbits than in LFs on day 105.

The present study shows that i) as compared to a LF diet, ingestion of a HF cholesterol diet led to stepwise increases in fasting plasma cholesterol, postprandial lipemia and postprandial occurrence of dietary lipids in the circulation; ii) these changes are related to a dramatic increase of atheroma deposition; iii) these changes could originate from the inability of hepatocytes to remove intestinal lipoproteins even when the lipase activity levels are doubled.

Sequential hydrolysis of bovine caseins in the preparation of a low-phenylalanine fraction for the treatment of phenylketonuria. E Perrin, L Miclo, A Driou, JL Gaillard, G Linden *(Laboratory of Food Bio-Sciences associated with Inra, Faculty of Sciences, University H-Poincaré Nancy-I, BP 239, 54506 Vandœuvre-lès-Nancy cedex, France)*

Children with phenylketonuria – an autosomal recessive disorder, concerning about one birth per 15 000, caused by reduction or absence of activity of hepatic phenylalanine hydroxylase (EC 1.14.16.1) – are constrained to a strictly controlled phenylalanine diet to prevent irreversible damage to the nervous system [Følling (1934) Hoppe-Seylers *Z Physiol Chem* 227, 169-176].

Proteic intake is made of an equilibrated amino acid mixture without phenylalanine. This bad tasting mixture has to be taken in rather high quantities. The rest of the meal is composed of food with low protein content (mainly vegetables and fruits) in amounts corresponding to the minimal needs of phenylalanine for maintenance or growth.

This study evaluates a preparation of a low-phenylalanine hydrolysate made from bovine casein. Proteolysis was performed with two sets of proteases chosen for their ability to cleave proteins at the phenylala-