lipids 30%, proteins 4%), a high isocaloric (634 Kcal) protein diet (glucids 50%, lipids 30%, proteins 20%) or a free diet at breakfast for a period of 4 weeks. At the end of each week and 3 h after breakfast, their cognitive capacities (Zazzo test) and blood glucose levels (LG) were evaluated. The intake of each nutriment was carefully assessed for each subject. Mean values were compared between groups with the low or high protein diet. The relationship between the Zazzo test, LG and caloric intake from the different nutriments was evaluated in all subjects.

There was no significant difference between the groups having the low and high protein diet (Student's *t*-test). Cognitive capacities, however, were significantly correlated with LG ($P = 0.006$), total caloric intake ($P = 0.011$), glucidic ($P = 0.009$) and lipidic calories ($P = 0.002$), but not with protein intake.

In conclusion, the total intake of nutriments at breakfast and especially the combination of glucids and lipids seemed able to increase late morning blood glucose levels and to improve cognitive capacities.

PROTEIN METABOLISM

Complementary modulation of intestinal and liver glutamine metabolism by nutritional conditions. C Rémésy, C Moundras, C Morand, C Demigné *(Laboratoire des maladies métaboliques, Inra-Clermont-Ferrand/Theix, 63122 Saint-Genès-Champanelle, France)*

Glutamine homeostasis is largely dependent on its metabolism by the splanchnic tissues. Glutamine is a major substrate in the intestine. The liver is capable of producing or utilizing this amino acid, depending on its digestive availability and on the acid/base status in the blood.

The aim of this work was to further investigate the relationship between the liver production of glutamine and changes in its intestinal metabolism. For this purpose, rats were adapted to different dietary protein levels (10 or 20% casein). The food was available for 8 h, blood and tissue sampling was performed during the postabsorptive or postprandial period. Digestive absorption and/or metabolism and liver metabolism were estimated by measurement of ateriovenous differences (portal vein-artery or hepatic vein-afferent differences). Whatever the dietary protein level, during the postabsorptive phase, liver glutamine release (about 2 µmol/min) matched intestinal utilization (1.5 to 1.75 µmol/min). In rats adapted to the low dietary protein level, during the postabsorptive period, the intestinal glutamine balance was negative (1.4 µmol/min), similar to what occurs during starvation. The liver production of glutamine remained relatively high during the postprandial period (1.7 µmol/min), as well as during the postabsorptive period. In rats fed the 20% casein diet, the digestive balance of glutamine remained slightly negative (0.6 µmol/min) despite a large supply of dietary glutamine. Under such conditions, the liver production tended to diminish, to about 1 µmol/min, which limited the supply available for the peripheral tissues. There was generally a noticeable absorption of glutamate during fed conditions (0.2–0.4 µmol/min) together with an additional release by the liver, in the range of 0.6 µmol/min. The liver release of glutamine during starvation was accompanied by a glutamate release (about 1 µmol/min), but the latter was not reutilized by the intestine. Thus, splanchnic tissues are liable to provide significant quantities of glutamate to the peripheral tissues.

Under normal conditions of acid/base balance, the liver metabolism of glutamine is closely related to intestine metabolism as a function of dietary protein levels. To

exceed the capacities of intestinal glutaminolysis, the dietary protein level has to be high. However, even with a large supply of protein, the splanchnic tissues continue to play a minor role in producing glutamine for muscles. In conclusion, under most usual nutritional conditions, there is an enterohepatic cycling of glutamine; the hepatic release of glutamine (and glutamate) may be interpreted as a nitrogen-salvaging process, independent of the acid/base status that becomes operative when the urea cycle activity is low.

Kinetic study of apoB-100 metabolism in non-insulin-dependent diabetics. C Maugeais, P Mahot, K Ouguerram, M Krempf, T Magot *(Centre de recherche en nutrition humaine, hôpital Laënnec, 44035 Nantes cedex 01, France)*

Non-insulin-dependent diabetic (NIDDM) subjects exhibit abnormalities in their plasma lipid and lipoprotein profiles that increased their risk of cardiovascular diseases. This study was designed to examine the metabolic behavior of apoB-100 using stable isotopes.

The five patients (37–66 years old) with NIDDM (HbA1C 6.7–10.3%) had hypertriglyceridaemia (TG 2.9 ± 0.9 g/L). They received a constant infusion (10^{-6} mol/kg/h) of deuterated leucine during 14 h. Patients were in fasting state during the experiment. Lipoproteins were isolated by ultracentrifugation and apolipoproteins by electrophoresis (SDS-PAGE). ApoB-100 was hydrolyzed and the tracer-to-tracee ratio in leucine was determined by mass spectrometry. Kinetic analysis of the tracer-to-tracee curves was performed by multicompartimental modeling.

Compared to control subjects, very low density lipoprotein (VLDL)-apoB production was higher (41.5 vs 24.8 mg/kg/day). In the experimental diabetic subjects, fractional catabolic rates (FCR) of VLDL-, and intermediate density lipoprotein (IDL)-apoB decreased (0.16 ± 0.05 vs 0.48 ± 0.05 h^{-1}, 0.15 ± 0.04 vs 0.62 ± 0.08 h^{-1}, respectively), while FCR of low density lipoprotein (LDL)-apoB slightly increased (0.030 ± 0.006 h^{-1} vs 0.022 ± 0.002 h^{-1}).

The main perturbations of lipoprotein metabolism in NIDDM were upstream of LDL as an increase in VLDL synthesis and as a decrease in VLDL and IDL catabolism.

Isotopic enrichment kinetics of the nitrogenous milk fractions in goats receiving a single intravenous injection of L-(U-^{14}C)-leucine. X Rubert-Aleman, G Rychen, C Claudon, F Laurent *(Ensaia-Inra, BP 172, 54505 Vandœuvre-les-Nancy, France)*

The aim of this experiment was to study the ^{14}C-labelling kinetics of goat milk nitrogenous fractions (casein fraction [CN], whey protein [WP] and the nonprotein fraction [NPN]) after a single intravenous injection of L-(U-^{14}C)-leucine, and to measure the apparition times of CN and WP labelling peaks to identify the labelled goat milk proteins which could be used for metabolic studies in monogastric animals (rat or pig).

L-(U-^{14}C)-leucine (Amersham, UK) was used as an adapted marker for three main reasons: its metabolic pathways are well defined (transamination of leucine to α-ketoisocaproate [KIC], decarboxylation or reamination of KIC to leucine), leucine is particularly abundant in milk proteins and its extraction rate from the plasma to the mammary gland is very high [Mepham and Linzell (1966) *Biochem J* 101, 76-83].

Three lactating Alpine goats (3 L milk/day) were each fitted with two temporary intravenous catheters placed respectively in each jugular vein. Isotope injection of L-(U-^{14}C)-leucine (3.6 x 10^6 Bq/goat diluted in 5 mL 0.9 % NaCl solution) was