of HGP was restored by metformin; iii) the mechanisms of control of the Glc–Glc6P cycle were impaired in insulin-resistant HF rats and were restored by metformin.

Role of the abundance of messenger RNA in the expression of hepatic and renal glucose-6 phosphatase in fasted and diabetic rats. G Mithieux, H Vidal, C Zitoun, C Minassian, N Daniele *(Faculté de médecine René-Laënnec, Inserm U 449, 69372 Lyon cedex 08, France)*

In this study we cloned and sequenced a rat glucose-6 phosphatase (Glc6Pase) complementary DNA (cDNA). The complete cDNA (1 071 bp) was amplified by RT-PCR using total liver RNA and oligonucleotide primers derived from murine species. The rat enzyme, deduced from the cDNA sequence, is a 95% homologue with the murine enzyme and 90% with the human enzyme. This cDNA was used to probe the abundance of Glc6Pase mRNA by Northern blot in vivo in fasted and diabetic rats (streptozotocin-induced).

In the liver, as compared with the fed rat, the Glc6Pase mRNA level increased 3.6 ± 0.4 and 3.6 ± 0.4 fold (arbitrary densitometric unit) after 24 and 48 h of fasting. It then returned to the fed rat level after 72 and 96 h of fasting (1.0 ± 0.3 and 1.4 ± 0.6 fold, mean ± SEM, $n = 5$). This agreed with the increase in Glc6Pase activity after 24 and 48 h of fasting and its later decrease at 72 and 96 h. In the kidney, the Glc6Pase mRNA level increased 2.7 ± 1.0 and 5 ± 1.2 fold after 24 and 48 h of fasting, it then plateaued at 72 and 96 h (4.5 ± 1.0 and 4.3 ± 1.0 fold). This is in agreement with the gradual increase in renal Glc6Pase activity throughout fasting. The liver Glc6Pase mRNA level in fasted rats rapidly decreased (92%) after refeeding for 90 min, 95% after 180 min and 97% after 420 min ($n = 3$ by time). The Glc6Pase mRNA abundance decreased less rapidly in the kidney. A significant 50% decrease was observed only after 3 h refeeding. However, Glc6Pase activity did not return to normal levels in either the liver or in the kidney during the 7 h refeeding period.

Glc6Pase mRNA abundance increased 4.5 ± 0.4 and 4.3 ± 0.1 fold in the liver and kidney of streptozotocin-diabetic rat, respectively. This correlated with similar increases in the Glc6Pase activity in both tissues. Glc6Pase mRNA level was partially compensated for by 12 h insulin treatment (2.4 ± 0.4 and 1.6 ± 0.2 fold in liver and kidney, respectively). Glc6Pase activity did not return to normal levels during the 12 h insulin treatment, in either of the tissues.

In conclusion, the Glc6Pase mRNA abundance was closely controlled at the pretranslational level in both the liver and kidney during nutritional transitions and diabetes. Our data suggested that insulin might play an important role in this control, and that additional mechanisms take place in the liver during the course of fasting.

The relationship between breakfast caloric intake and late morning blood glucose levels and cognitive capacities in a population of students. L Meynard-Rouard [1], C Jaffiol [2], JC Manderscheid [3], A Borderies [2] *([1] Lycée Victor-Hugo, Carpentras; [2] Service d'endocrinologie et Semhap; [3] Hôpital Lapeyronie, Montpellier, France)*

Several authors have stressed the problem of low breakfast caloric intake in France with its possible deleterous effects on scholastic results. However, discrepancies exist concerning the real effect of proteins and other nutriments on cognitive capacities and behaviour.

Two groups of 50 randomly selected young women (aged 15–21 years) received either a high carbohydrate (glucids 66%,

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