

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 \pm 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-Rouard¹, C Jaffiol², JC Manderscheid³, A Borderies² (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%,