

plasma leptin determination. Five of the 11 patients were studied by a hyperinsulinic euglycemic clamp with [6,6-2H₂]glucose used as tracer, and substrate oxydation measured by indirect calorimetry (IC).

The Wilcoxon test was used to compare results (mean \pm SEM) before and 12 months after GH treatment. These results showed that in spite of a significant IGF1 increase (158 ± 17 versus 77 ± 9 ng/ml), neither the body weight (70 ± 6 versus 71 ± 6 kg), nor the fat mass percentage (30 ± 2 versus 31 ± 2 %) nor the leptinemia (12 ± 3 versus 13 ± 3 μ g/L) were modified. Post-prandial endogenous glucose production (10.9 ± 0.6 versus 11.3 ± 0.4 mg/kg/min) and insulin stimulated glucose disappearance (40 ± 3 versus 47 ± 3 mg/kg/min) were not significantly modified by GH treatment. The IC measures showed a more pronounced ($P < 0.05$) protide oxydation inhibition under insulin perfusion (0.7 ± 0.1 versus 1.0 ± 0.1 mg/kg/min). Glucose and lipid oxydation were not significantly changed.

In conclusion, a complete GH substitution of GH deficient patients had no effect on body composition and did not seem to have any deleterous effect on glucose metabolism and insulinosensitivity.

Control of glucose-6-phosphatase in the small intestine of fasted and diabetic rats. N. Bruni, F. Rajas, S. Tarpin, C. Zitoun, G. Mithieux (Inserm U. 449, Faculté de Médecine RTH Laënnec, 69372 Lyon cedex 08, France).

Glucose-6-phosphatase (Glc6Pase) is the last enzyme of gluconeogenesis and glycogenolysis. In agreement with the concept that only the liver and kidney express Glc6Pase, it has been recently reported, using a RT-PCR approach, that Glc6Pase mRNA was not detectable in nine other tissues. Using the same approach, we demonstrated the presence of Glc6Pase mRNA in the duodenum (DUO) and

jejunum (JEJ) of adult rats (10–12 weeks), while Glc6Pase mRNA was not detected in the stomach, ileum, colon, brown adipose tissue, adrenals and bladder. We studied the effect of fasting and diabetes on Glc6Pase mRNA (by northern blot) and specific Glc6Pase activity in the small intestine in rats. The amount of Glc6Pase mRNA increased by eight times and six times ($P < 0.01$) in DUO and JEJ in streptozotocin diabetic rats. It returned to normal levels after insulin treatment for 10 h. Glc6Pase activity was increased by 300 % in DUO (2.6 ± 0.8 versus 0.83 ± 0.09 U/g wet tissue, mean \pm S.E.M., $n = 4$, $P < 0.05$) and JEJ (1.9 ± 0.3 versus 0.63 ± 0.03 U/g, $P < 0.01$) in diabetic rats as compared to normal rats. The amount of Glc6Pase mRNA increased by eight and seven times in DUO and JEJ of 48-h-fasted rats. It returned to normal levels in both tissues after refeeding for 7 h. Glc6Pase activity was significantly increased in fasted rats (2.5 ± 0.6 and 1.3 ± 0.2 U/g in DUO and JEJ, means \pm S.E.M., $n = 5$, $P < 0.05$ versus normal fed rats). In addition, Glc6Pase mRNA and activity were also expressed in ileum during fasting (1.3 ± 0.4 U/g). Glc6Pase mRNA and activity were also demonstrated in the human small intestine. In the human colon carcinoma cell line Caco-2, Glc6Pase is only expressed 15 days after the confluence of cells, in agreement with the state of differentiation of Caco-2 cells in the enterocytes for glucose metabolism. Thus, the small intestine might possess the capacity to produce endogenous glucose in portal blood. This should have important implications in various metabolic situations.

Assessment of insulin sensitivity from plasma insulin and glucose in the fasting or post oral glucose-load state. A. Avignon, A. Radauceanu, L. Monnier (Metabolic disease department, Lapeyronie hospital, 34295 Montpellier cedex 5, France).