

bition of Glc6Pase and may participate in the inhibition of hepatic glucose production occurring in this situation.

**Regulation of glucokinase expression by dietary carbohydrates in trout and carp livers.** S. Panserat<sup>a</sup>, C. Blin<sup>b</sup>, J. Breque<sup>a</sup>, C. Vachot<sup>a</sup>, F. Médale<sup>a</sup>, R. Krishnamoorthy<sup>b</sup>, S. Kaushik<sup>a</sup> (<sup>a</sup>Fish Nutrition Laboratory, Inra, 64310 St-Pée-sur-Nivelle, <sup>b</sup>Inserm U458, Paris, France).

Most teleosts are not able to utilize high levels of dietary carbohydrates efficiently. Previous studies suggested that this 'diabetic' phenotype may be related to the lack of an inducible glucokinase (hexokinase IV) enzyme in fish livers. Our objective was to obtain partial molecular probes for the glucokinases of trout and carp in order to study the regulation of hepatic glucokinase expression by dietary carbohydrates. Based on the hypothesis that all hexokinases are members of a gene family, we prepared degenerated primers corresponding to the highly conserved sequences of this family. Using these primers, we performed a RT-PCR (reverse transcription-polymerase chain reaction) with RNA extracted from livers of fishes fed a high carbohydrate diet. Hepatic glucokinase activities were measured. We obtained glucokinase-like molecular probes for trout (229 bp) and carp (232 bp). The sequences of the partial probes corresponded to an open reading frame that exhibited more than 80 % homology with the mammalian glucokinase sequence. Our data show that glucokinase activities in the liver increased with the dietary level of carbohydrates. Moreover, the results from northern blots and RT-PCR suggest an enhancement of glucokinase gene transcription in trout fed with high levels carbohydrates compared to those fed without carbohydrate. The regulation of glucokinase expression by

dietary carbohydrates in fish livers seems to be the same as that in mammals.

**A defect of suppression of endogenous glucose production contributes to lipid-induced glucose intolerance.** V. Rigalleau, M. Beylot, C. Pacciaudi, C. Guillot, G. Deleris, H. Gin (Service de diabétologie nutrition, USN hôpital Haut-Lévêque, 33600 Pessac, France).

An experimental lipid infusion results in an increased availability of lipid substrates. It impairs glucose tolerance, because it inhibits glucose oxidation. But the influence of lipids on endogenous glucose production (EGP) has not been examined during an OGTT. In eight normal subjects (age  $23 \pm 2$  years, BMI  $21.5 \pm 0.4$ ) we performed doubly labelled OGTT ( $1 \text{ g.kg}^{-1}$  maize glucose, naturally enriched in  $^{13}\text{C}$ , to measure exogenous glucose appearance RaE), with a primed-continuous di-deuterated glucose infusion to measure total glucose appearance RaT. EGP was calculated as  $\text{RaT}-\text{RaE}$ . Each subject underwent two OGTTs, first during a saline (Sa), second during an 'Ivélip 20%' (Iv) infusion ( $0.015 \text{ mL.kg}^{-1}.\text{min}^{-1}$ , started 90 min before an oral glucose charge). Post-absorptive EGP was not modified by the lipid infusion (Sa:  $2.32 \pm 0.11 \text{ mg.kg}^{-1}.\text{min}^{-1}$ , Iv:  $2.32 \pm 0.05$ ; NS). EGP was suppressed during the OGTTs (Nadir at +120 min). The lipid infusion produced a slight increase in glucose ( $P < 0.05$  from +120 to +180 min) and insulin ( $P < 0.05$  from +180 to +240 min) levels, EGP was less suppressed at time 90 and 120 min, and the 330 min cumulation of EGP was higher (Sa:  $317 \pm 57 \text{ mg.kg}^{-1}$ , Iv:  $395 \pm 58$ ;  $P < 0.05$ ). Despite identical oral charges, RaE was higher under 'Ivélip' (330 min cumulation: Sa:  $864 \pm 38 \text{ mg.kg}^{-1}$ , Iv:  $993 \pm 67$ ;  $P < 0.05$ ), suggesting an increased recycling of  $^{13}\text{C}$ . This suggests that increased gluconeogenesis may be the cause of the impaired sup-

pression of EGP under lipid infusion, and this impairment might have been underestimated the way we measured it.

**Effects of purified soybean proteins and dietary cholesterol on unsaturated fatty acid biosynthesis.** J. Bellenger, S. Madani, M. Narce, J. Prost, J.P. Poisson, J. Belleville (Unit of Cellular and Metabolic Nutrition, UPRES Lipids and Nutrition, Faculty of Sciences Mirande, University of Burgundy, BP 400, 21011 Dijon, France).

The aim of the present study was to investigate, in isolated rat hepatocytes, the effects of normoproteic (20 %) diets containing either casein or purified soybean proteins, with or without added cholesterol, on  $\Delta 6n-6$ ,  $\Delta 6n-3$ ,  $\Delta 5n-6$  and  $\Delta 9$  desaturase activities and total lipid fatty acid composition.

Twenty male Wistar rats (5 weeks old) were randomly divided into four groups. For 2 months they were fed, a diet containing 20 % of either casein (CAS) or highly purified (97 %) soybean proteins (SP), with or without 0.1 % added cholesterol. Isolated hepatocytes were incubated with  $1-^{14}C$  18:2n-6,  $1-^{14}C$  18:3n-3,  $1-^{14}C$  20:3n-6 or  $1-^{14}C$  18:0, precursors of  $\Delta 6n-6$ ,  $\Delta 6n-3$ ,  $\Delta 5n-6$  and  $\Delta 9$  desaturation steps, respectively. Desaturation rates were then determined after HPLC partition. The fatty acid composition of the isolated hepatocytes was measured by GLC. The significant differences of the results were assessed by the DUNCAN test.

$\Delta 6n-6$ ,  $\Delta 6n-3$ ,  $\Delta 5n-6$  and  $\Delta 9$  desaturase activities were significantly lower in the SP group versus CAS group (-43, -44, -45 and -33 %).  $\Delta 6n-3$ ,  $\Delta 5n-6$  and  $\Delta 9$  desaturase activities were decreased, when cholesterol was added to the CAS diet, by 47, 62 and 48 %, respectively, while the  $\Delta 6n-6$  desaturase activity was not significantly modified.  $\Delta 6n-6$  desaturase activity was increased by 36 % and  $\Delta 9$  desatu-

ration decreased by 30 % when cholesterol was added to the SP diet. Only the addition of cholesterol to the diets modified the fatty acid composition of the isolated hepatocytes, whatever the changes in the desaturase activities.

The decreased desaturase activities with the SP diet could be explained by the lower lysine/arginine ratio in SP than in CAS, which would have an activating effect on glucagon synthesis, as glucagon is known for its inhibitory effect on desaturase activities.

**Effects of dietary lipid source and energy restriction on the liver fatty acid profile in zucker rats (*fa/fa*).** R. Cantoral<sup>a</sup>, M.T. Macarulla<sup>a</sup>, M.I. Torres<sup>b</sup>, M.P. Portillo<sup>a</sup> (<sup>a</sup>Department of Nutrition, Faculty of Pharmacy, University of País Vasco; <sup>b</sup>Department of Public Health of the City Council, Vitoria, Spain).

Genetic obesity induces disturbances in the hepatic lipid metabolism such as an increase in triglycerides, cholesterol and phospholipid concentrations and changes in the fatty acid profile. The aim of this work was to study the effects of the dietary lipid source and energy restriction on the hepatic fatty acid content.

Twenty-eight obese male Zucker rats (*fa/fa*) were divided into four groups: rats fed ad libitum (group A), rats fed a 25 % energy restricted diet which provided a standard amount of fat (group B), rats fed a 25 % energy restricted diet which provided a high amount of olive oil (group C) and rats fed a 25 % energy restricted diet which provided a high amount of coconut oil (group D). After 4 weeks of dietary treatment, animals were killed by decapitation and the livers were dissected. Liver fatty acids were measured by gas chromatography. ANOVA test was used for statistical comparisons.