SPC (% dietary proteins)	0	50	75	100	100	100	RSD*
DL-methionine (% MS)		—	—	—	—	0.8	1.0
GDH (U/g proteins)	138^{c}	150^{c}	179^{b}	173^{b}	212^{a}	180^{b}	20
AAT (U/g proteins)	229^{b}	228^{b}	243^{b}	273^{ab}	496^{a}	201^{b}	164

Means of eight measurements. *Residual standard deviation of the analysis of variance, model: y = mean + dietary effects + residual; a, b, c, means with different superscript are significantly different ($P < 0.05$).

with the fact that SPC based diets induce an increase in nitrogen excretion [Médale et al., III International Symposium on Nutritional Strategies and Management of Aquaculture Waste, Vila Real, Portugal, 1997, 1-3/10/97]. This study highlights the influence of the origin of the dietary protein source on the activities of key enzymes of the nitrogen metabolism, influence which may then lead to modifications of protein accretion.

Role of phosphatidylinositol-3 kinase (PI3K) in the inhibition of glucose-6 phosphatase in the postprandial situation. N. Danièle[a], B. Payrastre[b], C. Zitoun[a], G. Mithieux[a] ([a]Inserm U. 449, Faculté de médecine, RTH Laënnec, rue Guillaume Paradin, 69372 Lyon cedex 98; [b]Inserm U. 326, Toulouse, France).

We have studied the molecular mechanism of glucose-6 phosphatase (Glc6Pase) inhibition, the last enzyme involved in hepatic glucose production, during the postprandial period in rats. By utilizing a rapid procedure of isolation of the microsomes, we showed that Glc6Pase activity was lower after refeeding for 360 min in rats previously unfed for 48 h: 65 ± 2 versus 96.5 ± 3 (in the presence of 1 mM Glc6P) and 225 ± 6 versus 306 ± 9 (20 mM Glc6P) nmol/min/mg prot., means ± S.E.M., $n = 12$, $P < 0.001$. The amount of immunoreactive Glc6Pase protein, detected by western blot, was not lower in microsomes from refed rats as compared to fasted rats. The amount of immunoreactive p85 (the regulatory subunit of phosphoinositide 3-kinase (PI3K)) and the PI3K catalytic activity, and the amount of IRS1 (insulin receptor substrate 1), were higher by a factor of 2.6, 2.4 and 2.6, respectively ($P < 0.01$), in the microsomes from the refed rats. After fractionation of microsomal membranes in sucrose gradients, p85 was immunodetected in all subfractions, either enriched in the plasma membranes or in the endoplasmic reticulum. We performed reconstitution experiments of microsomes from fasted rats with the two main lipid products of PI3K activity. Glc6Pase activity was inhibited in the presence of phosphatidylinositol 3,4 bisphosphate (competitive mechanism, Ki = 5.0 ± 0.1 μM, mean ± S.E.M., $n = 3$) and phosphatidylinositol 3,4,5 trisphosphate (Ki = 1.7 ± 0.7 μM). It was not inhibited in the presence of numerous other phospholipids. These results strongly suggested that an IRS1-triggered mechanism of PI3K translocation onto endoplasmic reticulum occurs in the liver of rats in the course of refeeding. This process, by means of the lipid products of PI3K activity, may account for the inhi-

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[a], C. Blin[b], J. Breque[a], C. Vachot[a], F. Médale[a], R. Krishnamoorthy[b], S. Kaushik[a] ([a]Fish Nutrition Laboratory, Inra, 64310 St-Pée-sur-Nivelle, [b]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 diabétologue nutrition, USN hôpital Haut-Lévêque, 33600 Pessac, France).

An experimental lipid infusion results in an increased availibility 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 ± 2 years, BMI 21.5 ± 0.4) we performed doubly labelled OGTT (1 $g.kg^{-1}$ maïze glucose, naturally enriched in ^{13}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 RaT-RaE. Each subject underwent two OGTTs, first during a saline (Sa), second during an 'Ivélip 20%' (Iv) infusion (0.015 $mL.kg^{-1}.min^{-1}$, started 90 min before an oral glucose charge). Post-absorptive EGP was not modified by the lipid infusion (Sa: 2.32 ± 0.11 $mg.kg^{-1}.min^{-1}$, Iv: 2.32 ± 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 ± 57 $mg.kg^{-1}$, Iv: 395 ± 58; $P < 0.05$). Despite identical oral charges, RaE was higher under 'Ivélip' (330 min cumulation: Sa: 864 ± 38 $mg.kg^{-1}$, Iv: 993 ± 67; $P < 0.05$), suggesting an increased recycling of ^{13}C. This suggests that increased gluconeogenesis may be the cause of the impaired sup-