

tion with riboflavin deficiency. Therefore, EMA might be used as a parameter for following acyl-CoA DH activities through AN refeeding.

METABOLIC EFFECTS OF UNSATURATED FATTY ACIDS

Effects of a high protein diet on food intake and some aspects of gut and liver nitrogen metabolism. C. Jean, G. Fromentin, J.-F. Huneau, V. Mathe, D. Tome (Unité Inra de nutrition humaine et de physiologie intestinale, Institut national agronomique de Paris-Grignon, 16, rue Claude Bernard, 75231 Paris cedex 05, France).

The consequences of feeding a high protein diet for several weeks have not been studied much. This study was designed to characterize the mechanisms of adaptation to a high protein diet in the intestine and the liver (transport and intracellular metabolism).

Materials and methods: Two groups of male Wistar rats were fed two protein diets (20 and 50 % casein) for 3 weeks. Liver cells were isolated using collagenase dissociation and amino acid transport was measured after adherence to plastic dishes. Brush-border membrane vesicles were prepared to measure the amino-acid transport rate in the small intestine.

Results: Feeding a 50 % casein diet resulted in a significant reduction in both food intake (-7 %) and growth rate (-20 %). Amino acid transport rate through system B⁰ and X_{A,G-} in the gut and system ASC in the liver were unaffected by the diet. In contrast, system A and X_{A,G-} activities were increased in the liver of rats fed the high protein diet. An increase in liver alanine aminotransferase, arginase, serine-threonine dehydratase activities was also observed in rats fed the

50 % casein diet, indicating that transaminations, ureogenesis and gluconeogenesis were increased by the high protein diet.

Conclusion: A high protein diet induces amino acid transport and metabolism adaptations in the liver. However, these changes appear to be insufficient to restore normal food intake and growth rate over the study period.

The effects of including soy protein concentrate in diets fed to rainbow trout on the activities of trans-deaminating enzymes. M. Mambrini^a, C. Vachot^b, S.J. Kaushik^b (^aLaboratoire de génétique des poissons, Inra, 78352 Jouy-en-Josas Cedex; ^bLaboratoire de nutrition des poissons, Inra, 64 310 St-Pée-sur-Nivelle, France).

In fish the importance of amino acid oxidation for energetic purposes – mainly due to trans-deamination reactions – explains their large dependence on dietary proteins. As part of a programme undertaken to measure the consequences of including soy protein concentrate (SPC) in diets fed to rainbow trout, we measured the activities of alanine amino transferase (AAT) and glutamate dehydrogenase (GDH) in the liver. Fish were fed for 3 months (mean final body weight 368 g), with six isonitrogenous diets where fish meal was progressively replaced by SPC, supplemented or not with DL-methionine. The liver was then sampled after fish were fasted for 48 h for the enzymatic assays.

GDH and AAT activities increased with the incorporation level of SPC, and those variations were not explained by any modification of glutamate intake. A negative linear relationship existed between GDH activity and whole body protein retention for the diets which were not deficient in DL-methionine (R = -0.995). These results are in agreement

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: $y = \text{mean} + \text{dietary effects} + \text{residual}$; 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. Payrastré^b, C. Zitoun^a, G. Mithieux^a (^aInserm U. 449, Faculté de médecine, RTH Laënnec, rue Guillaume Paradin, 69372 Lyon cedex 98; ^bInserm 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 \pm 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, $K_i = 5.0 \pm 0.1 \mu\text{M}$, mean \pm S.E.M., $n = 3$) and phosphatidylinositol 3,4,5 trisphosphate ($K_i = 1.7 \pm 0.7 \mu\text{M}$). 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-