Nutritional recovery after fasting. Morphological study of the intestinal mucosa restoration. S. Dunel-Erb, Cl. Chevalier, F. Decrock, A. Heitz, Y. Le Maho, A.C. Bach (Centre d'écologie et physiologie énergétiques, CNRS, 67087 Strasbourg cedex 2, France).

The kinetics of intestinal mucosa restoration was studied in refeeding rats after two different periods of fasting. Rats were fasted for 5 days (phase II, P2) or 8–16 days (phase III, P3), then rats were refed ad libitum (standard pellets) for 1, 3, 7 days or until recovering of the initial body mass (6–8 days after phase II, 10–13 days after phase III). Samples of jejunum were processed for microscopic observations and morphometry. The values are reported as means ± 1 SEM. ANOVA and Bonferroni tests were used for comparison ($P < 0.05$).

Compared to the control, starvation induced a significant decrease of the total intestine mass (P2, 35 %; P3, 40 %) and of the mucosa mass (P2, 54 %; P3, 60 %). The size of the villi also decreased (Control, 635 ± 14; P2, 400 ± 16; P3, 292 ± 13 µm). In addition, in P3 the villi density seemed to be reduced either because some villi were missing and/or because the size of the villi was reduced.

As early as 16 h after refeeding, villi were partly restored; but, after P3, enterocytes of the villi apex accumulated large lipid droplets. After 3 days of refeeding the morphometric studies showed values not significantly different from control: the villi were 560 ± 19 µm height in P2 and 566 ± 14 µm in P3. However, even after total body mass recovery, the structural homogeneity of the mucosa was not completely restored. Zones with damaged tissues were still visible.

Utilization of ethylmalonic acid as an indicator of acyl-coA dehydrogenase activities in anorexia nervosa patients refeeding. C.D. Capo-chichi[a], J.L. Guéant[a], E. Lefebvre[a], M. Vidailhet[a,b] ([a]Laboratoire de pathologie cellulaire et moléculaire en nutrition EP-CNRS 616, Faculté de médecine de Nancy, [b]Service de Pédiatrie, CHU Nancy, France).

Some studies have demonstrated that acyl-CoA dehydrogenases 'acyl-CoA DH' are affected by malnutrition. With the aim of evaluating acyl-CoA DH activities in anorexia nervosa 'AN', urinary organic acids 'OA' were analysed. The efficacity of refeeding was evaluated by amino acid 'AA' analyses and anthropomotric data. Twenty-two girls with AN (11–23 years old) were studied before and after refeeding. OA and AA were analysed respectively, by gas chromatography-mass spectrometry and by ion exchange chromatography. Wilcoxon paired test and Mann-Whitney test were used for statistical comparison between AN data before and after refeeding, and between AN and the controls, respectively. The values obtained after refeeding compared to the initial values showed a decrease in the 'non-indispensable AA/dispensable AA' ratio (1.83 ± 0.45 versus 2.6 ± 0.69; $P < 0.01$), an increase in body mass index 'BMI' (17.45 ± 1.55 versus 14.9 ± 2.12 kg/m^2; $P < 0.01$), of lean mass (34.12 ± 5.2 versus 29.16 ± 5.62 kg; $P < 0.05$), of fat mass (11.15 ± 2.31 versus 7.85 ± 2.12 kg; $P < 0.01$) and of ethylmalonic acid 'EMA' excretion (9.1 ± 4.9 versus 6.06 ± 3.82 µmol/mmol creatinine; $P < 0.05$). Despite weight recovery (44.53 ± 6.35 versus 38.01 ± 6.96 kg; $P < 0.01$), the persistence of elevated EMA excretion in AN compared to the controls (9.1 ± 4.9 versus 1.3 ± 2.83 µmol/mmol creatinine; $P < 0.01$) suggested that butyryl-CoA DH activity was affected and that normalization was delayed. This enzyme was one of the most affected in malnutri-

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 B^0 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 effets 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] ([a]Laboratoire de génétique des poissons, Inra, 78352 Jouy-en-Josas Cedex; [b]Laboratoire 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