
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 refeed 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-chichia, J.L. Guéanta, E. Lefebvre, M. Vidailhet (Laboratoire de pathologie cellulaire et moléculaire en nutrition EP-CNRS 616, Faculté de médecine de Nancy, 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 efficacy 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/m²; 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-