

protein were dramatically reduced in the colonic mucosa of GF vs CV rats (respectively  $-55 \pm 6\%$ , and  $-66 \pm 2\%$ ,  $P < 0.05$ ). Moreover, these changes were reversible, as mRNA and protein concentrations were restored in the colonic mucosa of IN rats.

In conclusion, there is a site-specific expression of mitochondrial HMG-CoA synthase in the intestinal mucosa of the adult rat. As opposed to what happens in the liver, its expression is not greatly affected by starvation vs feeding. In contrast, the large intestinal flora, through mechanisms which deserve further investigation, is a major factor controlling the enzyme.

**Influence of dietary proteins on colonic cell proliferation and luminal polyamine metabolism.** K Meziani, R Benamouzig, D Tomé (*Inra, UNHPI, 75006 Paris, France*).

Polyamines play an important role in DNA, RNA and protein synthesis. The current study was designed to investigate the influence of the quality of dietary proteins on luminal intestinal polyamine concentrations and their possible role in colonic cell proliferation.

Three groups of eight male Wistar rats were fed high protein diets (50% of either casein, zein or soy protein). After 4 weeks of feeding, both intestinal contents and colonic mucosa were recovered. Polyamines were assayed by HPLC and ornithine decarboxylase (ODC) activity was measured by the release of  $^{14}\text{CO}_2$  from 14C-L-ornithine.

Rats fed zein showed a 3-fold increase in mucosal colonic ODC activity compared to the other groups ( $31 \pm 11$ ,  $11 \pm 4$  and  $10 \pm 2 \mu\text{mol/h/mg}$  proteins for zein, casein and soy protein groups, respectively  $P < 0.05$ ). Luminal colonic putrescine and cadaverine levels were also higher in the group fed zein (putrescine:  $14 \pm 6$ ,  $3 \pm 1$  and  $0.6 \pm 0.3 \mu\text{mol/g DM}$ ; cadaverine:  $23 \pm 11$ ,  $0.6 \pm 0.1$  and  $0.2 \pm 0.09 \mu\text{mol/g DM}$  for zein,

casein and soy protein groups, respectively  $P < 0.05$ ). Furthermore, a high amount of polyamines were found in the jejunum and the ileum of rats receiving a soy protein diet, probably due to the higher level of polyamines in this diet. The difference of the true digestibility of dietary arginine (95% for soy protein, 93% for casein and 42% for zein), a precursor of polyamines, could explain these observations.

In conclusion, mucosal colonic ODC activity is influenced by dietary protein quality. Zein is associated with a higher colonic ODC activity. This modulation could be influenced by modifications in luminal polyamine concentrations.

**Uptake of  $\alpha$ -linolenic acid in human enterocyte-like Caco-2 cells.** T Tranchant<sup>1</sup>, P Besson<sup>2</sup>, C Hoinard<sup>2</sup>, J Delarue<sup>1</sup>, C Couet<sup>1</sup>, J Goré<sup>2</sup> (<sup>1</sup> *Laboratoire de nutrition, Faculté de médecine*; <sup>2</sup> *Laboratoire de physiologie et biophysique cellulaire, Faculté de pharmacie, Université de Tours, 37000 Tours, France*).

The uptake kinetics of  $\alpha$ -linolenic acid were investigated in the human intestinal cell line Caco-2. Four clones (PD10, PF11, PD7 and TC7) from the parental Caco-2 cells population were used. The TC7 clone was selected for the study of  $\alpha$ -linolenic acid uptake. [ $1\text{-}^{14}\text{C}$ ]linolenic acid dissolved in taurocholate was presented to the microvillus plasma membrane of differentiated TC7 cells. The initial rate of uptake is not a linear function of the  $\alpha$ -linolenic monomer concentration in the incubation medium. In the monomer concentration range studied (0.2 to  $36 \mu\text{M}$ ) apical uptake was saturable and followed Michaelis-Menten kinetics ( $V_m = 15.4 \pm 0.6 \text{ nmol/mg protein/min}$ ,  $K_m = 14.3 \pm 1.3 \mu\text{M}$ ). In addition, it was temperature and energy-dependent but was apparently unaffected by the sodium gradient. Excess of unlabeled long chain fatty acids led to a 27–68% reduction of [ $1\text{-}^{14}\text{C}$ ]linolenic acid

uptake. These facts argue in favour of the existence in these human intestinal cells of a carrier-mediated transport system for  $\alpha$ -linolenic acid and probably other long chain fatty acids as well.

**Chronic ingestion of acetate increases LDL in healthy subjects.** S Auboiron, C Alamowitch, G Slama, B Guy-Grand, FRJ Bornet (*Nutrition and Diabetes Departments-Inserm U 341, Hôtel-Dieu Hospital, 75004 Paris, France*).

Some authors have hypothesized that the hypocholesterolemic effect of dietary fibers is partly due to their end-products of colonic fermentation, the short chain fatty acids (SCFA), the main anion produced being acetate. To test this hypothesis, a cross-over design versus placebo experiment was drawn. Six healthy men ( $25.5 \pm 0.8$  years, BMI =  $21.8 \pm 0.4$  kg/m<sup>2</sup>) took during 4 weeks 100 mmol/day of acetate as capsules or a placebo. At the end of the treatment, plasma lipoproteins (chylomicron, VLDL, IDL, LDL, HDL<sub>2</sub> and HDL<sub>3</sub>) were isolated just before (T0), 2 (T2) and 4 h (T4) after a test meal (1 200 kcal, 48% lipids, 44% glucids, 8% proteins). Each time, plasma lipids and lipid content of lipoprotein particles were measured. Weight, food intake, blood glucose and insulin remained constant throughout the study. No difference between acetate and placebo treatment was observed for plasma triglycerides (TG) and phospholipides (PL). This was related to the lack of change in both level and content of TG-rich lipoproteins (Chylomicrons, VLDL, IDL). On the other hand, before test meal, we observed an increase of plasma cholesterol esters (CE), T0:  $207 \pm 16$  vs  $179 \pm 14$  mg/dL ( $P < 0.003$ ). This was still found after test meal, T2:  $198 \pm 14$  vs  $179 \pm 14$  mg/dL (NS), T4:  $200 \pm 14$  vs  $171 \pm 14$  mg/dL ( $P < 0.02$ ) without any change in free cholesterol (FC). The higher plasma CE levels were due to an increase in LDL

particles but not of HDL<sub>2</sub> and HDL<sub>3</sub>. The test meal emphasized the increase in LDL particles, T0:  $162 \pm 9$  vs  $141 \pm 11$  mg/dL (NS), T2:  $166 \pm 10$  vs  $145 \pm 10$  mg/dL ( $P < 0.03$ ) and T4:  $168 \pm 9$  vs  $148 \pm 8$  mg/dL ( $P < 0.005$ ). Acetate seemed to act on a number of LDL particles and also on the increase in their CE content,  $45 \pm 1$  vs  $41 \pm 3\%$  of the total mass (NS).

In conclusion, we observed an hypercholesterolemic effect of acetate; thus the effect of soluble fibers does not seem to be due to their SCFA production but could be the result of an increase in cholesterol fecal excretion.

**Regional metabolism of acetate in dogs.** E Pouteau, L Martin, H Dumon, M Champ, P Nguyen, M Krempf (*Centre de recherche en nutrition humaine; Laboratoire de nutrition et alimentation, École nationale vétérinaire de Nantes, 44000 Nantes, France*).

Acetate is mainly produced from colonic fermentation of non digestible substrats, but its endogenous origin and site of utilization remain unclear. Acetate metabolism was studied in peripheral and splanchnic tissues with stable isotope. Dogs were fasted 24 h, after 3 days of meat diet, and no expired hydrogen showed any bacterial fermentation. *Protocol 1*: five dogs were infused intravenously with [ $1-^{13}C$ ] acetate at  $2 \mu\text{mol.kg}^{-1}.\text{min}^{-1}$  for 200 min. Blood from the carotid artery and from the radial vein were collected. *Protocol 2*: five dogs were infused with [ $1-^{13}C$ ] acetate at  $1 \mu\text{mol.kg}^{-1}.\text{min}^{-1}$  for 120 min. Blood from the carotid artery, from a radial and the portal veins were sampled. Isotopic enrichments and concentrations of acetate were measured using a gas chromatography / mass spectrometry. The acetate turnover was calculated according to the steady state equation of the isotopic dilution method. *Protocol 1*: concentrations did not differ between arterial and venous plasma ( $167 \pm$