the mobilisation of hepatic TG stored, or from the reesterification of fatty acids coming from the hepatic captation of degraded lipoproteins.

Effect of butyrate on the expression of cyclin-dependent kinases and cyclin D by HT-29 epithelial cells. S Siavoshian [1], HM Blottière [2], C Cherbut [2], B Kaeffer [2], JP Galmiche [1] *([1] Centre de recherche en nutrition humaine de Nantes, Hôpital Laënnec; [2] Inra-LTAN, 44000 Nantes, France).*

Butyrate, a four-carbon fatty acid produced by dietary fibre fermentation, can modulate colonic epithelial cell proliferation in vitro. In a variety of cell systems, butyrate has been found to block cells in G_1 phase. D-type cyclins are synthesized in early G_1, and activate cdk4 and cdk6 as cells leave the quiescent phase. Cyclin E-cdk2 and cyclin A-cdk2 complexes form later in G_1 as cells prepare to DNA synthesis. The aim of our study was to investigate the mechanisms by which butyrate inhibits cell cycle progression toward S phase.

HT-29 cells were synchronized by exposing the culture to fetal calf serum-deprived DMEM medium. Cells were then exposed to complete medium (10% fetal calf serum) in the presence or absence of increasing concentration of sodium butyrate (from 2 to 8 mM) for 4 days. Proteins were extracted, and cyclin D, cdk2, cdk4 and cdk6 expression were studied by Western blotting.

After exposition to 2 mM sodium butyrate, a strong stimulation of cyclin D expression was observed, on HT-29 cells and it was optimal at 8 mM. This stimulation is associated with a decreased expression of cdk2. The level of cdk4 and cdk6 remained inchanged.

The increased level of cyclin D may be a marker of the blockade in G_1 phase, because in continuously proliferating cell populations, the level of D type cyclins is low. However, the decreased expression of cdk2 which leads to the inhibition of events essential for DNA replication, may participate directly to the mechanism of action of butyrate on cell proliferation.

Bacterial status is a key-factor controlling expression of the mitochondrial HMG-CoA synthase gene in the rat intestinal mucosa. C Cherbuy [1], B Darcy-Vrillon [1], JP Pégorier [2], C Andrieux [1], B Azzout [2], PH Duée [1] *([1] UEPSD, Inra, 78352 Jouy-en-Josas cedex; [2] Ceremod, CNRS, 92190 Meudon Bellevue, France).*

Butyrate from bacterial origin is a major energy substrate for colonic epithelial cells. In these cells, ketone body production from butyrate proceeds via the mitochondrial 3-hydroxy-3-methylglutaryl-CoA (HMG-CoA) synthase. We investigated the effect of the intestinal site, as well as the nutritional and bacterial status on this enzyme expression in the rat intestinal mucosa.

Thirty-six male rats (240–310 g) differing by their bacterial status were used at the age of 3 months: conventional (CV) rats, germ-free (GF) rats and germ-free rats inoculated (IN) at 2 months with the intestinal flora of conventional rats, by gastric administration of freshly obtained and diluted feces. All rats were allowed free access to water and diet, except for a group of CV rats which were fasted for 48 h prior to sacrifice. HMG-CoA synthase expression was quantified by northern and western blot analysis of jejunal or colonic mucosal scrapings.

The mRNA encoding HMG-CoA synthase were detected in the colonic mucosa of CV rats, and their concentration was only modestly decreased after a 48 h starvation ($-26 \pm 11\%$; $P < 0.10$). In contrast, HMG-CoA synthase was not expressed in the small intestinal mucosa, whatever the nutritional status of the animals. The concentrations of mRNA and immunoreactive