personal autoimmunity, we introduced an immunosuppressive therapy (azathioprine + corticosteroids). A consistent long term and stable remission was induced with only two relapses which occurred when the treatment was reduced and was put under control by reintroduction of the immunosuppressors.

Postheparin plasma LPL activity was decreased in the patient and was normalized by the treatment (1.9 vs 5.9 μmol/h/mL controls: 7.2 ± 2.2 μmol/h/mL). Hepatic lipase activity (HL) remained normal (6.1 vs 6.8 μmol/h/mL; controls: 6.9 ± 2 μmol/h/mL). To demonstrate a direct interaction between an autoantibody and LPL, we studied by Western blot, the binding of the patient's immunoglobulins to nitrocellulose-bound human LPL. A specific binding of IgG to LPL was observed only with the chylomicrons of the patient. No binding of IgG was found in other patients lipoproteins. No signal was obtained either with patient's chylomicrons obtained in the postprandial state while she was under immunosuppressive therapy or with those from diabetic patients with type V hyperchylomicronemia. A competitive inhibition of the IgG-LPL binding was obtained with porcine pancreatic lipase, a protein that shares a partial homology with human LPL. In vitro, proteins extracted from patients chylomicrons induced a dose-dependent specific inhibition of control postheparin plasma LPL activity.

These data demonstrate that an autoantibody can specifically inhibit LPL activity and induce hyperchylomicronemia. Additionally, they suggest that in vivo, LPL is bound on the surface of chylomicrons. However, it is likely that the inhibition of LPL activity is dependent upon a direct interaction of the antibody with LPL bound at the arterial wall.

Contribution of hepatic lipogenesis and plasma free fatty acids reesterification to hepatic triglycerides secretion in healthy subjects. F Diraison, C Pachiaudi, M Beylot *(Laboratoire de physiopathologie métabolique et rénale, Faculté de médecine René-Laënnec, 69373 Lyon cedex 08 et CRNH de Lyon, Hôpital Edouard-Herriot, 69472 Lyon cedex, France).*

Triglycerides (TG) secreted by liver can be synthesized with fatty acids derived from hepatic lipogenesis or from plasma free fatty acids (FFA) uptake (reesterification). The contribution of these two sources of fatty acids was measured in four healthy subjects (TG = 0.86 ± 0.02 mmol/L, mean ± SEM) in the post-absorptive period. Lipogenesis was calculated using the measurement of deuterium incorporation in TG-palmitate molecules from deuterated water (loading dose of 3 g/kg of estimated total body water). FFA reesterification was calculated using the kinetic of the incorporation in TG of intravenously infused [1–^{13}C]palmitate during 4 h. The fractional (k) and absolute (R_t) turnover rate and the half-life ($t_{1/2}$) of TG were calculated using the kinetic of decay (6 h) of ^{13}C in the palmitate of TG after the end of [1–^{13}C]palmitate infusion. The enrichments were measured using isotopic ratio mass spectrometry (deuterium in plasma water, ^{13}C in FFA-palmitate and TG-palmitate) or organic mass spectrometry (deuterium in TG-palmitate). Results were 2.91 ± 0.34 h, 0.226 ± 0.017 h^{-1} and 0.16 ± 0.01 mmol/kg/min respectively for $t_{1/2}$, k and R_t. These values agreed with those of other studies. The contribution of FFA reesterification to this TG plasma pool was 0.1 ± 0.01 h^{-1}, that is to say 44 ± 3% of TG R_t. The lipogenesis contribution to this R_t was only 3.5 ± 0.5%. These results confirm that hepatic lipogenesis, contrary to reesterification, is a minor pathway in healthy subjects. However, these two metabolic pathways represent only 50% of secreted TG. The remaining 50% could come from either

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