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

Chronic ingestion of acetate increases LDL in healty 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 ± 0.8 years, BMI = 21.8 ± 0.4 kg/m^2) 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_2 and HDL_3) 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 ± 16 vs 179 ± 14 mg/dL ($P < 0.003$). This was still found after test meal, T2: 198 ± 14 vs 179 ± 14 mg/dL (NS), T4: 200 ± 14 vs 171 ± 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_2 and HDL_3. The test meal emphasized the increase in LDL particles, T0: 162 ± 9 vs 141 ± 1 1 mg/dL (NS), T2: 166 ± 10 vs 145 ± 10 mg/dL ($P < 0.03$) and T4: 168 ± 9 vs 148 ± 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 ± 1 vs 41 ± 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 μmol.kg^{-1}.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 μmol.kg^{-1}.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 ±