values were normalized when expressed per kg LBM. Postprandial oxidation was reduced in elderly men relating to an increase in splanchnic extraction. These results suggested that a limited amino acid disposal to muscle in the elderly is a consequence of a higher splanchnic extraction that could, in turn, lead to a lower stimulation of muscle protein synthesis during feeding.

Plasma albumin concentration is a main factor in the control of lipoprotein metabolism: a kinetic study of two cases of analbuminemia. C Maugeais, K Ouguerram, P Maugeais, P Mahot, T Magot, M Krempf *(Centre de recherches en nutrition humaine, hôpital Laënnec, 44035 Nantes cedex 01, France)*

Hyperlipidaemia is commonly present in nephrotic syndrome. From previous studies, the physiopathology of these lipid disturbances could be related to a low albumin plasma level or to urinary loss of factors controlling the lipoprotein metabolism. Human analbuminemia is an inherited disease characterised by a low plasma albumin concentration and a dyslipidaemia but no proteinuria. This rare disease represents a good model for better understanding the mechanism of dyslipidaemia in nephrotic syndrome.

We studied the kinetic aspects of the apoB-containing lipoproteins metabolism of two sisters (26 and 30 years old) with analbuminemia using a constant infusion of leucine labeled with stable isotopes for 14 h.

Compared to control subjects (male, 21–25 years old), very low density lipoprotein (VLDL)-apoB production was higher (41.5 vs 24.8 mg/kg/day) as well as the production of intermediate density lipoprotein (IDL) and low density lipoprotein (LDL)-, apoB (30 vs 16 and 16 vs 10 mg/kg/day, respectively). The fractional catabolic rate of all apoB-containing lipoproteins was decreased (0.30 vs 0.48, 0.28 vs 0.62, 0.011 vs 0.022 h^{-1}, respectively).

These abnormalities were similar to the kinetic disturbances previously reported for nephrotic syndrome. The results suggested that a low albumin plasma concentration is probably the main factor controlling the lipid abnormalities in this kidney disease.

LIPID METABOLISM

Hepatic steatosis in the goose: influence of genotype on lipid metabolism. E Fournier [1], G Guy [2], R Peresson [1], D Hermier [1] *([1] Inra, 37380 Nouzilly; [2] Inra-Artiguères, 40280 Benquet, France)*

Susceptibility to liver steatosis in the goose is at least partly under genetic control. The Landes goose (L) exhibits a typical fatty liver in response to overfeeding, whereas the Poland goose (P) is partly resistant. Plasma and liver lipids were therefore analyzed in 14-week-old male geese weighing 5.2 kg at the start of the experimental period (16 L and 13 P). Plasma lipoproteins were isolated by density gradient ultracentrifugation [Hermier et al (1988) *J Lipid Res* 29, 893-907] and their concentration and chemical composition were determined before and after 14 days of overfeeding with boiled maize. Liver composition was determined after the completion of the overfeeding period.

Before overfeeding, the plasma lipoprotein profile of the geese was typical of birds, having a low amount of very low density lipoproteins (VLDL), and a predominance of high density lipoproteins (HDL). In both breeds, VLDL were abnormally poor in triglycerides (TG $\approx$ 30%), but HDL concen-

tration was higher in the P geese (6.44 vs 4.97 g/L). In response to overfeeding, fatty liver resulted from the accumulation of triglycerides (≈ 95% of liver lipid content), but also of phospholipids and free and esterified cholesterol. Hepatic storage was greater in the L geese for all lipids except for phospholipids, and resulted in a two-fold heavier fatty liver in this breed (1 005 vs 485 g). Consequently, lipid losses during sterilization, due to cell membrane disruption, were three-fold higher in the L geese (26.3 vs 7.5%). A parallel increase in plasma VLDL and HDL concentration (two- to three-fold) and in their TG content was noted, especially in the P geese in whom the effect of overfeeding was more pronounced.

In the P goose, the channelling of TG towards secretion, rather than intrahepatic storage, may have been responsible for the lower susceptibility of this breed to liver steatosis. Moreover, in both breeds, overfeeding seemed to induce a relative defect in phospholipid synthesis together with an increase in their secretion, especially as HDL. Consequently, the lack of intrahepatic phospholipids i) may have contributed to prevent cellular hypertrophia and therefore the development of hepatic steatosis and ii) accounted for membrane fragility which was responsible for considerable lipid losses, especially in the L geese.

Measurement of hepatic lipogenesis and cholesterol synthesis with deuterated water (D_2O). F Diraison, C Pachiaudi, M Beylot *(Faculté de médecine René-Laënnec, Inserm Unité 197 and CRNH de Lyon, 69008 Lyon, France)*

Hydrogen atoms are incorporated from body water during fatty acid and cholesterol synthesis. Measurement of deuterium appearance in these molecules during D_2O administration allows a determination of their rate of synthesis. After validation of this method in the rat, four healthy subjects (normal weight, total triglycerides 0.53 mM) drank a loading dose of deuterium oxide (3 g D_2O/kg body water) in two sessions, at 20 and 22 h, then deuterium water enriched at 0.44% APE (6 g/kg drinking water) was consumed over the following 60 h of the study. Deuterium enrichment in plasma water was determined by isotope ratio mass spectrometry (IRMS). Deuterium enrichments in plasma-free cholesterol, palmitate of total triglycerides (TG) and TG of very low density lipoproteins (VLDL) were measured by organic mass spectrometry.

A plateau level in plasma water enrichment was reached at 0.27 ± 0.01% APE. Using this value and the deuterium enrichment mr asured (at 8 h, the third day of the study) in palmitate of TG (0.8 ± 0.25 MPE), in TG-VLDL (0.67 ± 0.24 MPE), and in cholesterol (0.78 ± 0.18 MPE), the calculated fractional synthetic rates of these molecules were, respectively, 13.22 ± 4.7 and 10.87 ± 4.25% for plasma palmitate-TG and palmitate-TG-VLDL and 10.3 ± 1.9% for plasma-free cholesterol.

These values agree with those reported by Leicht and Jones [(1991) *Biol Mass Spectrom* 20, 392], who used a delicate technique which involved i) the combustion of TG and cholesterol molecules, and ii) the reduction of water produced for isotopic mass spectrometry analysis. Hellerstein et al [(1991) *J Clin Invest* 87, 1841], using ^{13}C-acetate, obtained lower results; however, there is evidence that labeling heterogeneity of cytosolic acetyl-CoA occurs, the precursor pool in this case [Zhang et al (1994) *J Biol Chem* 269, 11025].

In conclusion, deuterated water was determined to be a safe and convenient method for measuring the fractional synthetic rates of fatty acids and cholesterol in human beings.