Mirande, BP 138, 21004 Dijon Cedex, France)

Fatty liver and the similarity between skin lesions observed in kwashiorkor and those described in experimental essential fatty-acid (EFA) deficiency have led to the hypothesis that the effects of chronic malnutrition may result from combined protein and EFA deficiencies. The relationships of serum VLDL to hepatic lipid composition were studied after 28 d of protein depletion to determine the interactions between dietary protein levels and dietary EFA availability. Rats weighing 80 ± 5 g, were fed purified diets containing 20% or 2% casein and 5% fat as either salmon oil, rich in eicosapentaenoic (EPA) and docosahexaenoic (DHA) acids, or hydrogenated coconut oil, poor in EFA. All diets were isoenergetic (cornstarch replaced lacking casein). Animals were divided into 4 groups, SAC (20% casein + 5% salmon oil), SAd (2% casein + 5% salmon oil), COC (20% casein + 5% hydrogenated coconut oil), and COd (2% casein + 5% hydrogenated coconut oil). The procedures for chemical analysis utilized were similar to those described in detail in previous studies [Bouziane *et al* (1992) *J Nutr* 122, 2037-2046].

After 28 d the protein-deficient rats presented a fatty liver (mainly due to triacylglycerols) and reduced liver and VLDL phospholipid contents ($P < 0.001$). The VLDL apolipoprotein (apo) contents were diminished with protein deficiency and this decrease was mainly due to the reduced syntheses of apo B_{48} + apo B_{100}, whereas apo AI content was not affected. Triacylglycerol and phospholipid fatty acid compositions in both liver and VLDL, showed a depressed unsaturated to saturated fatty-acid ratio with protein deficiency, but more when hydrogenated coconut oil was associated. Furthermore, protein deficiency decreased linoleic, α-linolenic and arachidonic acids in VLDL phospholipids. Thus, feeding protein-deficient diets might enhance EFA requirements. Besides, in the phospholipid fractions with protein deficiency, EPA was diminished (SAd *vs* SAC, 2.7 *vs* 16.6 and 9.1 *vs* 13.0% of total fatty acids, $P < 0.001$, for liver and VLDL, respectively), and DHA was increased (SAd *vs* SAC, 16.8 *vs* 12.0 and 15.8 *vs* 11.2% of total fatty acids, $P < 0.001$, for liver and VLDL, respectively). Protein deficiency seems to accelerate the conversion of EPA to DHA with salmon oil diets. A similar effect has been observed in a previous study on phospholipid microsomes [Ulman *et al* (1992) *J Nutr Biochem* 3, 188-193]. Conversely, when protein-deficient diets were poor in both EFA and long-chain n–3 fatty acids such as hydrogenated coconut oil diets, there was more 22:5 (n–6) and less 20:4 (n–6) in VLDL lipids. Both eicosapentaenoic and arachidonic acids are the precursors of eicosanoids and their diminution may be related to some clinical symptoms observed in infants suffering from kwashiorkor.

Postprandial changes in the hepatic metabolism of lipoproteins in the preruminant calf, *Bos* spp.

D Bauchart, D Durand (*INRA-Theix, Laboratoire Croissance et Métabolisme des Herbivores, UR Métabolismes Énergétique et Lipidique, 63122 Saint-Genès-Champanelle, France*)

In spite of a low hepatic lipogenesis, the uptake, recycling and secretion of lipids (as lipoproteins) are major metabolic activities of the liver in the growing or lactating cattle [Bauchart (1993) *J Dairy Sci* 76, 3864-3882]. The hepatic metabolism of lipoproteins has been mainly studied in the high-producing dairy cow developing intense lipid infiltration of the liver in early lactation in situations of energy deficit [Mazur *et al* (1992) *Diabète et Métabolisme*, 18, 145-149]. Less information is available in non-pathological situations, except for the starving calf [Bauchart *et al* (1989) *J Lipid Res* 30, 1499-1514].

This work examines the postprandial changes in the hepatic metabolism of lipoproteins in four 3-week-old preruminant male calves (body weight: 46.0 ± 2.0 kg). Animals were given a conventional milk replacer containing tallow (23% diet DM) in 2 equal meals per day (9 h and 16 h). They were equipped with catheters and electromagnetic flow probes for estimation of hepatic lipoprotein fluxes.

Blood samples were collected 2 (T2), 7 (T7) and 15 h (T 15) after the morning meal. Plasma lipoproteins were isolated by density (d) gradient ultracentrifugation at 39,000 rpm for 46 h at 15°C and analyzed as described previously [Bauchart *et al* (1989) *op cit*].

Compared to the mean values determined during a 24 h period, plasma concentrations amounted to 250, 80 and 60% for insulin and 25, 180 and 160% for triglycerides (TG) at 2 (T2), 7 (T7) and 15 h (T15) after the meal, respectively. Net hepatic uptake (mg/min/kg BW) of very low density lipoproteins was noted at T2 (–0.20; NS)

T7 (–1.86; $P < 0.01$) and T15 (–1.42; $P < 0.01$) which confirmed the low capacity of the liver to secrete TG-rich lipoproteins [Bauchart *et al* (1989), *op cit*]. Hepatic production of low density lipoproteins ($1.026 < d < 1.060$ g/ml) was observed, but was not significant at T2 (0.29), T7 (1.78) and T15 (3.43).

Hepatic metabolism of high density lipoproteins (HDL) varied according to the size and density of the particles. With the light HDL (HDLl, $1.060 < d < 1.090$ g/ml), a net uptake was noted at T2 (–2.33; $P < 0.05$), but a net production was measured at T7 (+ 1.99; NS) and T15 (+8.02; $P < 0.01$). Conversely, a net production of heavy HDL (HDLh, $1.091 < d < 1.180$ g/ml) was noted at T2 (1.25; NS) and a net uptake was observed at T7 (–4.60; $P < 0.01$) and T15 (–7.0; $P < 0.01$). These results clearly indicate that the liver converts HDLh to HDLl during high triglyceridemia (T7 and T15) either by lipid transfer from TG-rich lipoproteins and/or by the increased lecithin/cholesterol acyl transferase (LCAT) activity associated with hypertriglyceridemia.

In vivo hepatic secretion of very low density lipoproteins in the growing turkey and chicken treated with an anti-lipoprotein lipase serum. M Kouba [1], D Hermier [2] ([1] *INRA-ENSA, Laboratoire de Biochimie, 65, rue de Saint-Brieuc, 35000 Rennes,* [2] *INRA-SRA Nouzilly, 37380 Monnaie, France*)

Previous results from our laboratory showed that the very low adiposity in turkeys compared to chickens may be related to a much lower hepatic lipogenesis in turkeys [Kouba *et al* (1992), *Br Poult Sci* 33, 1003-1014; Kouba *et al* (1993) *Comp Biochem Physiol* 105A, 359-362]. Since the liver is the main site of lipogenesis in birds, our results suggest that triglyceride (TG) production by the liver is lower in turkeys than in chickens. Therefore, the *in vivo* hepatic secretion of very low density lipoproteins (VLDL) and TG were measured in birds with a maximum growth rate (8-week-old chickens, 11-week-old turkeys). Both species were reared together and fed *ad libitum* the same diets: a starter diet (12.3 MJ/kg ME, 26% protein and 6.8% lipid) for 4 weeks and a grower diet (12.7 MJ/kg ME, 22.4% protein and 6.8% lipid) from 4 to 11 weeks.

Animals received an injection of an anti-lipoprotein lipase (LPL) serum (1 ml/kg body weight) in order to prevent the hydrolysis of the VLDL-TG and their uptake by peripherial tissues. VLDL were characterized after ultracentrifugation by adding their lipid component concentrations. The *in vivo* inhibition of the LPL activity caused a rapid rise in plasma VLDL concentration (11-fold in both species) and TG concentration (3.5-fold in turkeys, and 6-fold in chickens), and an alteration in VLDL composition. The free cholesterol (FC) and TG contents were higher after blockade (5.4 *vs* 2.7% FC and 69.7 *vs* 62.8% TG in turkeys; 4.2 *vs* 2.4% FC and 78.3 *vs* 68.2% TG in chickens). The cholesteryl ester (CE) and phospholipid (PL) percentages were lower (10.2 *vs* 18.4% CE and 14.7 *vs* 16.3% PL in turkeys; 2.9 *vs* 7.1% CE and 14.6 *vs* 22.3% PL in chickens). In both control and injected groups, plasma concentrations of VLDL, VLDL-TG and total TG were generally lower in turkeys than in chickens. The use of an anti-LPL serum and the determination of the plasma volume by the Evans' blue method made it possible to measure the hepatic secretion rate of VLDL, VLDL-TG and total TG (expressed as mg/h/kg body weight). They were higher ($P < 0.01$ or less) for all parameters in chickens than in turkeys ($m \pm SD$; 54.6 ± 9.6 *vs* 32.9 ± 11.2 for VLDL, 41.9 ± 7.3 *vs* 23.7 ± 9.3 for VLDL-TG and 61.7 ± 6.5 *vs* 31.8 ± 10.5 for total TG).

This study has provided evidence of a positive relationship between hepatic lipogenesis, VLDL secretion and fattening in turkeys and chickens.

Effects of dietary cholesterol and saturated to polyunsaturated fatty-acid ratio on the hererogeneity of LDL and HDL particles in the 1.040–1.090 g/ml interval in the preruminant calf, _Bos_ spp. L Leplaix-Charlat [1], D Durand [1], PM Laplaud [2], D Bauchart [1] ([1] *INRA-Theix, Laboratoire Croissance et Métabolismes des Herbivores, UR Métabolismes Énergétique et Lipidique, 63122 Saint-Genès-Champanelle;* [2] *Laboratoire de Biochimie, Faculté de Médecine et de Pharmacie, 2, rue du Dr-Marcland, 87025 Limoges cedex, France*)

Cattle are considered to be high density lipoprotein (HDL) mammals because they possess HDL