

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 (HDLI,  $1.060 < d < 1.090$  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 HDLI 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<sup>1</sup>, D Hermier<sup>2</sup> (<sup>1</sup> INRA-ENSA, Laboratoire de Biochimie, 65, rue de Saint-Brieuc, 35000 Rennes, <sup>2</sup> 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 peripheral 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 ( $m \pm SD$ ;  $54.6 \pm 9.6$  vs  $32.9 \pm 11.2$  for VLDL,  $41.9 \pm 7.3$  vs  $23.7 \pm 9.3$  for VLDL-TG and  $61.7 \pm 6.5$  vs  $31.8 \pm 10.5$  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 heterogeneity of LDL and HDL particles in the 1.040–1.090 g/ml interval in the preruminant calf, *Bos spp.*** L Leplaix-Charlat<sup>1</sup>, D Durand<sup>1</sup>, PM Laplaud<sup>2</sup>, D Bauchart<sup>1</sup> (<sup>1</sup> INRA-Theix, Laboratoire Croissance et Métabolismes des Herbivores, UR Métabolismes Énergétique et Lipidique, 63122 Saint-Genès-Champanelle; <sup>2</sup> 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