

as the predominant lipoprotein class ($\geq 80\%$ of total lipoproteins) [Bauchart *et al* (1989) *J Lipid Res* 30, 1499-1513]. Some of the low density lipoproteins (LDL) and HDL present similar densities in the 1.040–1.090 g/ml range. The proportion of LDL and HDL might be subject to marked changes with nutritional state and post-natal development in the preruminant calf [Bauchart (1993) *J Dairy Sci* 76, 3864–3882].

In the present study, the effects of dietary cholesterol and the source of dietary fatty acids (tallow vs soya bean oil) on the LDL and HDL distribution in the calf plasma were determined.

Twenty-two 4-week-old male calves (65 ± 5 kg body weight) were fed for 17 d: 1) a conventional milk replacer containing triglycerides (23% diet DM) either from tallow (T; 4% of n-6 polyunsaturated fatty acids, PUFA, n = 6) or from soya bean oil (S, 58% of n-6 PUFA, n = 5); or 2) the same diets supplemented with cholesterol (1% diet DM) (TC, n = 6 ; SC, n = 5). Blood samples were collected at peak lipid absorption (7 h after the morning meal) in the jugular vein. LDL and HDL particles in the 1.040–1.090 g/ml interval were isolated by ultracentrifugal flotation and the heterogeneity of these particles was resolved by heparin–sepharose affinity chromatography. Lipoprotein fractions were analyzed as described previously [Bauchart *et al* (1989) *op cit*].

Mean plasma concentrations of lipoproteins in the 1.040–1.090 g/ml interval were 230 (T), 295 (TC), 368 (S) and 539 (SC) mg/dl. These particles presented similar elution characteristics from the heparin–sepharose column exhibiting 3 distinct fractions: fractions I and II corresponding to HDL particles, fraction III to LDL particles. In this density range, the HDL to LDL ratio increased from 6 (T) to 16 and 17 with diets S and TC because of higher amounts of HDL particles, and to 10 with diet SC because of higher amounts of both LDL and HDL particles. Higher amounts of HDL in diets rich in PUFA and/or cholesterol were mainly associated with higher proportions of cholesteryl esters (CE) in the particles: 24% (T), 27% (TC), 28% (S) and 39% (SC). Similar CE enrichment was observed in LDL particles, especially with diet SC. These results emphasize the important role of PUFA: 1) in the incorporation of cholesterol in the LDL and HDL particles via the lecithin/cholesterol acyltransferase (LCAT) activity in cattle; and 2) in the stimulation of HDL synthesis since the accumulation of CE-rich HDL (1.040–1.090 g/ml) paralleled the accumulation of total HDL.

Relationship between plasma lipid and lipoprotein profile during pre-overfeeding, energy value of the diet, and differential responsiveness to overfeeding in two strains of geese. D Hermier¹, G Guy², R Peresson¹ (¹INRA, Station de Recherches Avicoles, 37380 Nouzilly; ²INRA, Station Expérimentale des Palmipèdes à Foie Gras, Artiguères, 40280 Benquet, France)

In the overfed goose, plasma very low density lipoproteins (VLDL) are poor in triglycerides (TG 30%) which remain stored in the hepatocyte and are partly responsible for the resulting fatty liver [Hermier *et al* (1991) *Lipids* 26, 331-339]. The phase of pre-overfeeding, during which the birds are allowed to feed *ad libitum* a diet containing 2 900 kcal/kg, 20.5% protein and 2.9% fat after a period of food restriction, is probably of major importance for the metabolic changes leading to steatosis. Plasma lipoproteins, prepared by density gradient ultracentrifugation, were compared before and after pre-overfeeding between the Landes strain, which is remarkably susceptible to liver steatosis, and the Rhine strain which is partly resistant (13 22-week-old male birds from each strain). Before pre-overfeeding, plasma TG concentration was lower in the Landes geese (0.69 vs 0.89 g/l), in accordance with the hypothesis that hepatic secretion of TG is impaired in this strain. However, plasma lipoprotein profile (concentration and composition) was typical of the goose and similar in both strains as concerns VLDL, IDL + LDL (intermediate- and low-density lipoproteins, considered as a whole, d 1.013–1.040) and HDL (high-density lipoproteins). After pre-overfeeding, plasma VLDL concentration increased by 35% in both strains, but, as in after overfeeding, they were unusually low in TG, especially in the Landes geese (21% TG in VLDL) compared with the Rhine geese (33%). At the same time, HDL (d 1.052–1.130) concentration decreased by 15% (7 and 6 g/l before and after pre-overfeeding respectively), whereas IDL + LDL did not change significantly (1.20–1.50 g/l), whatever the strain. A parallel experiment has been conducted to measure metabolizable energy of maize, which is the quasi-exclusive dietary component during overfeeding, under conditions of mild overfeeding (3 times the *ad libitum* food intake) in 10 birds from each strain. It showed that both strains have a similar and very efficient utilisation of dietary energy ($\approx 3\ 500$ kcal/kg

maize). It may be concluded that the difference in susceptibility to liver steatosis between the 2 strains does not result from a bad dietary efficiency in the Rhine strain, but rather from a defect in TG assembly to nascent VLDL, which is more pronounced in the Landes strain. This phenomenon occurs even when the geese overfeed spontaneously, and is not related specifically to artificial feeding.

LPL and HL activities in golden hamster during the suckling period. Influence of the maternal diet. R Sicart, R Sablé-Amplis, V Millet (CNRS, Université P-Sabatier, rue F- Magendie, 31400 Toulouse, France)

Hepatic lipoprotein lipase (HL) and lipoprotein lipase (LPL) are central enzymes in lipid metabolism. The development of their activities has not been studied in the hamster, a suitable model for studies of lipid metabolism. Therefore, we used hamsters (*Mesocricetus auratus*) to study the changes in the activities of HL and of LPL in inguinal adipose tissue and heart from birth to weaning (21 d). We also examined whether the enzyme activities were influenced by the composition of the maternal diet.

Newborns were obtained from mothers fed *ad libitum* either a standard diet or apples in addition to the same diet. LPL and HL activities [Nilsen-Ehle and Ekman (1977) *Artery* 3, 194–209] were expressed as mU/g fresh tissue (1 mU = 1 nmol of free fatty acid min^{-1}). In parallel, plasma insulin (IRI) was measured by radioimmunoassay and carcass lipid was evaluated gravimetrically. Values are given as means \pm SEM of at least 6 determinations.

Inguinal adipose tissue was not detectable at birth, but appeared at 1 d of age. The weight of tissue increased moderately during the early suckling period (10 d) then rose dramatically until weaning. The percentage of carcass lipid was 2% body weight at birth and reached 18% at 21d. LPL activity emerged with the development of the tissue and exhibited 2 peaks: one at 4 d ($2\,709 \pm 435$ mU/g fresh tissue), the other at 21 d ($1\,593 \pm 123$ mU/g fresh tissue). The lowest activity (406 ± 12) was noted at 10 d after birth. LPL activity in cardiac tissue was doubled from birth (401 ± 10) to 10 d (933 ± 79) then returned to the initial values at weaning (21 d). HL activity was low at birth (40 ± 2) and gradually rose until

weaning (303 ± 28). During the first suckling period, changes in LPL activity in adipose tissue were highly correlated ($r = 0.8$, $p < 0.01$) with plasma IRI levels (14.4 ± 2.2 $\mu\text{U/mL}$ at birth, 40.4 ± 3.1 at 4 d, 25.9 ± 3.1 at 10 d). All the examined parameters, except plasma IRI, were significantly lower during the first days of life in hamsters born to mothers fed the apple-enriched diet (LPL: -40% in adipose tissue and -20% in heart; HL: -20% , carcass lipid: -30%).

In conclusion, hamsters are characterized by the absence of white adipose tissue and by relatively high level of plasma IRI at birth. Moreover, LPL and HL activities increased during early life period and are lowered when the mother is fed the apple-enriched diet.

Reduction of turkey plasma cholesterol by dietary copper supplementation. GM Pesti, RI Bakalli, WL Ragland (Departments of Poultry Science and Avian Medicine, University of Georgia, Athens, GA 30602-2772, USA)

It is well established that rats fed diets deficient in copper exhibit hypercholesterolemia and elevated liver copper levels [Klevy (1976) *Am J Clin Nutr* 26, 1060–1068; Petering *et al* (1987) *J Agric Food Chem* 25, 1105–1109; Murthy and Petering (1976) *J Agric Food Chem* 24, 808–811]. Liver Cu is believed to regulate cholesterol biosynthesis by reducing hepatic glutathione levels [Kim *et al* (1992) *FASEB J* 6,2467-2471]. Glutathione stimulates the enzyme 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase in rats,

Table I. (GM Pesti *et al*)

Cu (mg/kg)	Gain* (g)	FCR** (g/g)	Plasma	
			Cu (ppm)	Cholesterol (mg/dL)
0	376	1.39	22.5	107
63	384	1.30	24.0	109
125	344	1.36	30.5	87
250	375	1.32	37.7	82

* Body weight gain, ** FCR = feed conversion ratio (g intake/g gained).