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The hepatic synthesis and secretion of lipoproteins such as VLDL and HDL is particularly important in lipoprotein homeostasis in bovine species (Bauchart *et al*, 1989). On the one hand, VLDL-mediated hepatic triglyceride export is low and can be limiting during lipomobilization, while on the other hand, HDL, which accounts for more than 75% of total plasma lipoproteins, plays a major role in reverse cholesterol transport in these species. In order to stimulate the hepatic production of lipoproteins, estrogen was administered to three 3-week-old male Friesian calves (55 ± 3 kg body weight) equipped with catheters and electromagnetic flow probes for estimation of hepatic lipoprotein fluxes. Animals were fed a conventional milk replacer in which lipid and carbohydrate content was reduced to 30% in order to induce lipomobilization. 17 β-Estradiol (E) was then administered by infusion through the portal route (20 µg per kg BW) for 1 h after the morning meal and subsequently by intramuscular injection (200 µg/d per kg BW) over the following 4 d. Blood samples were collected 7 h after the morning meal. Total plasma lipoproteins (d < 1.180 g/ml) were separated by density-gradient ultracentrifugation into 22 subfractions and their respective chemical composition was determined by enzymatic and immunological methods.

Estrogen treatment did not significantly modify the physicochemical properties and density distribution of bovine plasma lipoproteins. In spite of restrictive energy supply, low levels of hepatic VLDL production were observed under control conditions (0.09 mg/min per kg BW). Hepatic VLDL production was stimulated by estrogen perfusion (EP) (0.32 mg/min per kg BW) but strongly reduced by the long-term treatment of estrogen administered by intramuscular injection (EI) (– 0.62 mg/min per kg BW); these findings emphasize the importance of the route of administration and the hormonal dose under our dietary conditions. Estrogen treatment stimulated hepatic production of light HDL (1.060 – 1.091 g/ml) (– 2.71 *vs* 1.17 and 2.16 mg/min per kg BW in control, EP and EI treatment respectively) and hepatic uptake of heavy HDL (1.091 – 1.180 g/ml) (2.35 *vs* – 0.52 and 0.13 mg/min per kg BW). Modification in the hepatic metabolism of HDL by estrogen may involve a decrease in hepatic lipase activity (Hazzard *et al*, 1984) and cholesterol esterification by LCAT.

Bauchart D, Durand D, Laplaud PM, Forgez P, Goulinet S, Chapman MJ (1989) *J Lipid Res* 30, 1499-1513

Hazzard WR, Haffner SM, Kushwaha RS, Applebaum-Bowden D, Foster DM (1984) *Metabolism* 33, 779-784