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Effects of dietary cholesterol on hepatic metabolism of triglyceride-rich lipoproteins in the preruminant calf, Bos spp. L Leplaix ¹, D Bauchart ¹, D Durand ¹, PM Laplaud ^{2,3}, MJ Chapman ³ (¹ INRA, UR Métabolismes Énergétique et Lipidique, Theix, 63122 St-Genès Champanelle; ² Faculté de Médecine et de Pharmacie, Laboratoire de Biochimie Médicale, 87025 Limoges Cédex; ³ INSERM U321, Hôpital de la Pitié, Unité de Recherche sur les Lipoprotéines et l'Athéro-génèse, 75651 Paris Cédex, France)

In preruminant calves fed a high fat milk replacer or in high-production dairy cows during the fat mobilization period, hepatic disorders of fatty acid metabolism may occur and lead to steatosis. Steatosis could result from a limited hepatic secretion of VLDL-triglyceride (VLDL-TG).

Among the lipotrope agents, dietary cholesterol induces an increase in VLDL-TG hepatic secretion in hamster hepatocytes, and an increase in plasmatic apoprotein (apoB) VLDL in rabbit and bovine species (Beynen et al, 1983). Consequently, in order to stimulate hepatic production of VLDL, cholesterol was given to 5 male Friesian preruminant calves (1 month old) equipped with catheters implanted in the hepatic vessels to determine the *in vivo* net production of TG-rich lipoproteins by the liver (Bauchart et al, 1989). Animals were fed a conventional milk replacer with or without cholesterol in which lipids (tallow) constituted 22.1% dry matter (DM) and proteins

23.7% DM. Cholesterol mixed with milk at a 1% DM level was given for 21 d. Blood samples were collected 7 h after the morning meal. Plasma lipoproteins were separated by isopycnic density gradient ultracentrifugation and their respective chemical composition determined by enzymatic (lipids) and RID (apoB) methods.

Cholesterol treatment induced a large increase in plasma IDL (31.4 vs 4.4 mg/dl) and LDL (114.3 vs 41.4 mg/dl) and, to a lesser extent, an increase in VLDL (4.8 vs 2.9 mg/dl). An increased proportion of EC at the expense of TG was observed in VLDL, IDL and LDL. No apoE was detected in VLDL and IDL particles under both experimental conditions.

A net increase in VLDL secretion by the liver was observed in 2 calves (0.4 vs –0.5 mg/min/kg BW) but no significant effects were found in 3 others. In contrast, net increases in hepatic uptake of IDL (–0.4 vs 1.4 mg/min/kg BW) and LDL (–1.0 vs –0.7 mg/mn/kg BW) were noted in all calves.

The results suggest that cholesterol may not be a limiting factor for hepatic VLDL-TG secretion. However, further investigations are needed to characterize the effects of cholesterol on TG-rich lipoprotein metabolism in the liver regarding the nature of dietary fatty acids which are known to control hepatic lipid and apoprotein synthesis.

References

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