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Effects of dietary cholesterol on hepatic metabolism of triglyceride-rich lipoproteins in the preruminant calf, *Bos* spp.

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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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