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P-123 - VITAMIN B3 IMPAIRS CHOLESTEROL METABOLISM IN HYPERCHOLESTEROLEMIC MICE

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Resumen

Objectives: To study the effect of vitamin B3 administration over HDL function in a mouse model of hypercholesterolemia.

Methods: Apolipoprotein E-deficient (KOE) mice were challenged with a high-fat diet for 4 weeks. Groups of five KOE mice were treated with different doses of vitamin B3 (i.e., 0.25% and 1%) via tap water.

Results: The administration of vitamin B3 to KOE mice produced an elevation (~1.5-fold; $p < 0.05$) in the plasma levels of cholesterol, which was mainly accounted for the non-HDL fraction. Compared with untreated mice, the administration of vitamin B3 to KOE mice produced a [3H]-cholesterol plasma accumulation (~1.5-fold; $p < 0.05$) in the m-RCT. As revealed by kinetic analysis, the latter was mainly explained by an impaired clearance of non-HDL lipoproteins (~0.8-fold; $p < 0.05$) in high-dose, vitamin B3-treated KOE mice. Consistently, the relative content of [3H]-tracer was lowered in the livers (~0.6-fold; $p < 0.05$) and feces (> 0.5-fold; $p < 0.05$) of vitamin B3-treated mice. Nevertheless, the relative gene expression of several targets controlling hepatobiliary removal of cholesterol to feces, which include Abcg5 and Abcg8, showed a trend to be or were up-regulated in the liver (Abcg5: 2.9-fold; $p < 0.05$; Abcg8: 2.4-fold; $p = 0.06$) and small intestine (Abcg5: 2.1-fold; $p = 0.15$; Abcg8: 1.9-fold; $p < 0.05$) of high-dose, vitamin B3-treated mice.

Conclusions: Our data show that the administration of vitamin B3 to KOE mice impaired m-RCT in vivo. This finding was in part due to a defective clearance and reduced hepatic uptake of plasma non-HDL lipoproteins.