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LDL RECEPTOR DEFECTIVE MICE PRESENT ELEVATED LIVER LIPOGENESIS AND PERIGONADAL ADIPOSE TISSUE MASS

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LDL receptor knockout (K/O) mice are models of familiar hypercholesterolemia, a disease characterized by high LDL-cholesterol plasma levels and early atherosclerosis manifestation. However, the whole body cholesterol (chol) and triglycerides (TG) homeostasis are not completely established in these mice. In this work we determined the plasma lipoprotein profile, aortic atherosclerosis lesion, perigonadal adipose tissue mass and liver lipogenesis and steroidogenesis in K/O and wild type (WT) mice after 2 weeks on a low (chow) or high fat and high cholesterol (western) diets. K/O mice presented 10-fold and 33-fold increase in LDL-chol when fed a chow and western diet, respectively, as compared to WT mice. Two weeks of western diet induced aortic root atherosclerotic lesions in the K/O mice (22 x $10^3 \mu m^2$) but not in the WT mice. K/O mice presented lower body weight (-17%, $p<0.05$) but higher relative weight of perigonadal adipose tissue (+ 35%, $p<0.05$) than the age matched WT mice, for both type of diets. Total liver lipogenesis and steroidogenesis were 33 and 50% higher, respectively, in K/O than in WT mice under chow diet. As expected, after the western diet, these de novo biosynthetic processes were markedly inhibited and no differences were observed between K/O and WT mice. These results suggest that increased liver lipogenesis contributes to higher adipose depot formation in K/O mice and predispose these mice to develop obesity.

Cholesterol - Adipose tissue mass - Lipogenesis