Bile Acid Supplementation Improves Established Liver Steatosis in Obese Mice Independently of Glucagon-Like peptide-1 Secretion

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Abstract

Bile acids or its derivatives may influence non-alcoholic fatty liver disease development through multiple mechanisms. Intestinal L-cells secrete glucagon-like peptide-1 (GLP-1) and can be activated by bile acids (BA) influencing insulin resistance and hepatic steatosis development and progression. The aim of the present study was to assess the effects of cholic acid (CA) or ursodeoxycholic acid (UDCA) administration on portal and systemic levels of GLP-1 in genetically obese mice with established hepatic steatosis. Eight-week-old ob/ob mice were fed CA or UDCA during 4 weeks. Systemic and portal GLP-1 levels were measured as well as glucose tolerance test, serum and biliary parameters, hepatic triglyceride content, liver histology, and hepatic gene expression of relevant genes related to bile secretion. Eight-week-old ob/ob mice exhibited marked obesity, hyperinsulinemia, and fasting hyperglycemia. Administration of both CA and UDCA was associated to decreased hepatic triglyceride content and complete reversion of histological steatosis. BA-fed animals did not exhibit significant differences in glucose tolerance. In addition, neither CA nor UDCA administration significantly influenced portal or systemic GLP-1 levels. CA and UDCA strongly ameliorated established fatty liver in ob/ob mice independently of the GLP-1 incretin pathway. Thus, the anti-steatotic action of these bile acids is likely related to direct hepatic effects.

Keywords: Bile, Steatosis, Incretins, Insulin resistance.
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