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Basic Study

Ursodeoxycholic acid ameliorates hepatic lipid metabolism in LO2 cells by regulating the AKT/mTOR/SREBP-1 signaling pathway

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Abstract

BACKGROUND

Nonalcoholic fatty liver disease (NAFLD), the most common chronic liver disease, can progress into nonalcoholic steatohepatitis (NASH), cirrhosis, and even hepatocellular carcinoma. Bile acids such as ursodeoxycholic acid (UDCA) plays an essential role in the pathogenesis of NAFLD by regulating the level of sterol regulatory element-binding protein (SREBP) 1c, but the underlying regulation mechanism remains elusive. Increased evidence indicates that the AKT/mTOR/SREBP-1 signaling pathway is a key pathway to regulate hepatic cellular lipid metabolism. UDCA may regulate the AKT/mTOR/SREBP-1 signaling pathway to ameliorate hepatic lipid metabolism.

AIM

To investigate the functional mechanism of UDCA in an oleic acid

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