Involvement of fatty acid-CoA ligase in hepatocellular carcinoma growth: Roles of cyclic AMP and p38 mitogen-activated protein kinase.

朱娟秀

Liang YC;Wu CH;Chu JS;Wang CK;Hung LF;Wang YJ;Ho YS;Chang JG;Lin SY

摘要

Abstract

AIM: Fatty acid-CoA ligase 4 (FACL4) is an arachidonatepreferring enzyme which has been shown to be up-regulated in human colon cancer tissues and implicated in the colon tumorigenesis. The purpose of this study was to investigatethe role of FACL4 in the human hepatocellular carcinoma (HCC) tumorigenesis and the specific signal pathways involved in this process.METHODS: We investigated the expression and regulation of FACL4 in HCC, adjacent non-tumorous liver tissues, and cell lines.RESULTS: In HCC patients, we demonstrated that FACL4 gene expression was markedly elevated in the cancerous tissues than in the adjacent non-cancerous liver tissues.In addition, several human hepatoma cell lines, including Hep3B and HepG2, expressed high levels of FACL4. Stable overex-pression of FACL4 knockdown plasmids (small interfering RNA, siRNA) to Hep3B cells significantly decreased FACL4 expression and subsequently limited the cell proliferation. Treatment of Hep3B cells with 8bromo-cAMP and SB203508 (p38 MAPK inhibitor) significantly suppressed the FACL4 expression. CONCLUSION: FACL4 is involved in the HCC tumorigenesis and both cAMP and p38 MAPK pathways are associated with the regulation of FACL4 in HCC.