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ERK regulates liver cirrhosis and hepatocellular carcinoma

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Patients with liver cirrhosis have highest risk of developing hepatocellular carcinoma, a worldwide leading malignancy. Targeting liver injury-dependent signaling pathways could help to alleviate liver cirrhosis and prevent hepatocellular carcinoma development. MAPK signaling pathway plays an important role in cell proliferation, differentiation and regeneration. In this study, we investigate the role of Erk2 in chronic liver injury in terms of hepatocytes as well as immune responses. Both WT and Erk deficient mice were compared under choline deficient ethioine-supplemented diet (CDE diet), which leads to chronic liver injury and eventually lead to HCC development. Liver damage occurred in WT and $Erk2^{-/-}$ livers upon CDE diet treatment. $Erk2^{-/-}$ livers appear to have less degree of liver cirrhosis than WT livers. In addition, Erk2-/- cells also have lower expression in cirrhosis-related genes alpha-SMA and Col1al in comparison with WT. However, more activated T cells were found in $Erk2^{-/-}$ mice than WT mice upon liver injury. Besides, Erk2 could positively regulate the proliferation and survival in T cells. In HCC cell line, inhibition of Erk could induce apoptosis but did not alter cancer stem cell marker of CD133. In conclusion, ERK signaling play a role in regulation of liver cirrhosis and hepatocellular carcinoma.

Biography

Chiung-Fang Chang is currently a Researcher at Far Eastern Memorial Hospital, Taiwan. She graduated from National Taiwan University. She received her PhD degree in the Division of Biological Sciences at University of California, San Diego, USA. Her expertise includes Immunology, Cell Biology and Molecular Biology. Her current research projects focus on the cancer stem cells and immune responses in hepatocellular carcinoma.

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