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Summary

CFIM25 OVEREXPRESSION INHIBITS HEPATIC STELLATE CELL ACTIVATION AND LIVER FIBROSIS THROUGH KLF14/ PPARΓ SIGNALING PATHWAY

Yong Xie

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CFIm25 (NUDT21) overexpression inhibits hepatic stellate cell activation and reverses CCl4-induced liver fibrosis through the KLF14/PPARγ signaling pathway in mouse models and patient samples.

KEY POINTS

- CFIm25 expression was drastically decreased in activated murine hepatic stellate cells and fibrotic liver tissues from cirrhosis patients.
- CFIm25 overexpression downregulated fibrosis-related genes and inhibited hepatic stellate cell activation, migration, and proliferation.
- The antifibrotic effects of CFIm25 resulted from direct activation of the KLF14/PPARγ signaling axis, as demonstrated by the finding that KLF14 inhibition abrogated CFIm25-mediated antifibrotic effects.
- The study used adeno-associated viruses and adenoviruses to alter hepatic CFIm25 expression in vivo and in vitro, with mechanisms explored through RNA-seq and co-IP assays.
- The authors suggest CFIm25 might provide a novel therapeutic approach for liver fibrosis treatment.

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