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Summary

LIVER SINOSOIDAL ENDOTHELIAL CELL CONTRIBUTES TO LEUKOCYTE RECRUITMENT IN LIVER FIBROSIS THROUGH A CYCLOOXYGENASE-2-DEPENDENT MECHANISM

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COX-2 expression in liver sinusoidal endothelial cells promotes fibrosis through NF-κB-mediated leukocyte recruitment, and selective COX-2 inhibitors attenuate liver injury in mouse models.

KEY POINTS

- Ptgs2 deletion in mouse models reduced collagen deposition, fibrosis-related proteins (collagen I and α-SMA), inflammatory cell infiltration, and serum transaminase levels in TAA- and DDC-induced fibrosis
- COX-2 expression was elevated in LSECs in injured liver tissues, confirmed by immunofluorescence co-staining with LYVE1 and RNA-seq data from murine LSECs in various fibrotic models
- The COX-2-PGE2-EP2 axis increases chemokines (CXCL1 and CCL2) and adhesion molecules (ICAM1 and VCAM1) through NF-κB pathway activation, promoting leukocyte recruitment and hepatic inflammation
- Selective COX-2 inhibitors Celecoxib and Etoricoxib decreased collagen deposition, fibrosis-related protein expression, inflammatory cell infiltration, and serum transaminase levels in TAA and DDC models
- The authors suggest COX-2 as a potential therapeutic target in liver fibrosis treatment based on these findings in mouse models and human hepatic sinusoidal endothelial cells

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