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Summary

LSECTIN-CRISPR IMMORTALIZED LIVER SINUSOIDAL ENDOTHELIAL CELLS HAVE AND AUGMENTED PROINFLAMMATORY SECRETOME UPON STIMULATION WITH LSECTIN AND TOLL-LIKE RECEPTOR LIGANDS

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Hepatobiliary

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An in vitro study using LSECtin-knockout mouse liver sinusoidal endothelial cells demonstrates that LSECtin downregulation increases proinflammatory cytokine production while reducing IL-10 secretion, suggesting a role in cirrhosis-associated inflammation.

KEY POINTS

- LSECtin-Crispr liver sinusoidal endothelial cells showed significantly increased secretion of proinflammatory cytokines (IL-6, TNFalpha) and chemoattractants (CCL5, CXCL10) compared to wild type cells when stimulated with LSECtin ligands and Toll-like receptor ligands.
- IL-10 production was lower in LSECtin-Crispr cells than in wild type cells upon stimulation, indicating LSECtin normally favors anti-inflammatory cytokine secretion.
- Upon LPS stimulation, CXCL10 increased from 359.78 ng/mL in wild type to 852.42 ng/mL in Crispr cells, while IL-10 decreased from 2.21 ng/mL to 1.52 ng/mL.
- The findings suggest LSECtin loss, which occurs during chronic liver disease, may contribute to the inflammatory microenvironment in cirrhosis.
- This research is relevant to those studying the immunological mechanisms underlying chronic liver disease and cirrhosis.

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