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Summary

KLF4 ORCHESTRATES MYELOID-ENDOTHELIAL CROSSTALK VIA CXCL2/CXCL3-CXCR2 AXIS TO DRIVE NEUTROPHIL EXTRACELLULAR TRAPS AND INFLAMMATORY CASCADES IN ACETAMINOPHEN-INDUCED LIVER INJURY

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Myeloid KLF4 deletion drives acetaminophen-induced liver injury through a monocyte-neutrophil-endothelial inflammatory cascade involving CXCL2/CXCL3-CXCR2 signaling and neutrophil extracellular traps formation.

KEY POINTS

- Myeloid-specific KLF4 deletion increased CXCL2+CXCL3+CD24+ monocytes that activated NF-κB signaling to secrete CXCL2/CXCL3 chemokines, which bound CXCR2 on neutrophils and drove neutrophil infiltration and neutrophil extracellular traps formation.
- Neutrophil-derived NETs promoted endothelial cells toward pro-inflammatory polarization, while endothelial-derived CXCL1 further recruited neutrophils, establishing a monocyte-neutrophil-endothelial interaction that perpetuated microenvironmental inflammation.
- Myeloid KLF4 knockout induced enrichment of CXCL1+IL17RA+ endothelial cells and amplified the TNF-α signaling pathway during acetaminophen-induced liver injury progression.
- The study proposes therapeutic strategies targeting NETs formation or the CXCL2/CXCL3-CXCR2 axis as novel targets for precise intervention in acetaminophen-induced liver injury.
- This work used myeloid-specific Klf4 knockout mice with multidimensional omics analysis including RNA sequencing, single-cell transcriptomics, flow cytometry, and in vivo liver imaging to dissect microenvironmental remodeling.

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