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Summary

UPAR-DEPENDENT MACROPHAGE EFFEROCYTOSIS ORCHESTRATES HEPATIC INFLAMMATION RESOLUTION AND TISSUE REPAIR

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Study identifies uPAR/STAT3/IRF7/AXL signaling pathway as critical regulator of macrophage efferocytosis and hepatic homeostasis in acetaminophen-induced liver injury.

KEY POINTS

- uPAR expression was significantly elevated in liver tissue from both mouse DILI models and DILI patients
- Systemic and myeloid deficiency of uPAR alleviated liver inflammation in APAP-challenged mice by enhancing macrophage efferocytosis through upregulation of AXL
- Transcription factor IRF7 directly binds to the AXL promoter in macrophages, and uPAR regulates AXL-mediated efferocytosis through the STAT3/IRF7 pathway
- Macrophage-specific IRF7 overexpression enhanced efferocytosis and reduced liver inflammation in both DILI and ischemia-reperfusion injury models, while AXL-/- mice showed exacerbated injury and GAS-6 protein alleviated injury
- Findings were derived using multiplex immunohistochemistry, flow cytometry, spatial transcriptomics, single-cell RNA sequencing, intravital 3D imaging, and various knockout mouse models

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