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Summary

ALCOHOL-INDUCED IGE SECRETION IN THE GUT AGGRAVATES STEATOHEPATITIS VIA FCERIA-MEDIATED HISTAMINE RELEASE

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Hepatobiliary

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Experimental and clinical evidence demonstrates that elevated IgE levels in alcohol-associated liver disease are driven by intestinal Th2 cytokine shifts and contribute to liver injury through FcεRIα-histamine signaling.

KEY POINTS

- Alcohol intake increased circulating IgE in both mice and humans with ALD, partly directed against intestinal food antigens, and IgE levels in ALD patients associated with increased liver disease severity and correlated to plasma histamine levels
- IgE elevation was prevented by CD4+ T cell depletion and IL4Rα-deficiency in ethanol-fed mice, and IL4 and IL13 transcription was increased in the small intestines of mice and in human duodenal samples during ALD
- IgE-deficient and FcεRIα-deficient mice showed less severe ethanol-induced steatohepatitis compared to wildtype littermates, which associated with lower hepatic histamine levels
- Blocking free systemic IgE effectively prevented liver injury and inflammation in murine ALD, and IgE levels declined with alcohol abstinence in patients
- Elevated serum IgE is a specific hallmark of alcohol-related liver injury compared to patients with other liver diseases

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