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Summary

INVOLVEMENT OF P38 GAMMA/DELTA KINASES IN THE DEVELOPMENT OF ALCOHOLIC STEATOHEPATITIS

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Knockout of p38 γ/δ kinases alleviates alcoholic liver disease in mice by reducing steatosis and inflammation in a chronic-binge ethanol model.

KEY POINTS

- In a murine NIAAA model of alcoholic steatohepatitis, p38 γ/δ knockout mice treated with ethanol showed smaller liver size, decreased steatosis, and lower inflammatory cytokine expression compared to wild-type mice.
- The chronic-binge ethanol diet caused oxidative stress and mTOR pathway activation in both wild-type and knockout mice.
- Ethanol-treated wild-type mice exhibited p62 phosphorylation at Thr269 and Ser272, which was reduced in knockout mice, suggesting partial restoration of autophagic pathway inhibition.
- This experimental study may be relevant to researchers investigating molecular mechanisms of alcoholic liver disease and potential therapeutic targets involving p38 kinase pathways.

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