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Summary

ACTIVATED NOTCH SIGNALING PATHWAY PROTECTS THE INTESTINAL BARRIER IN DSS-INDUCED COLITIS BY REGULATING TIGHT JUNCTIONS

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Activation of the Jag1/Notch1/Hes1 signaling pathway protects intestinal mucosal barrier integrity during inflammatory injury by preventing MLCK-dependent tight junction dysfunction.

KEY POINTS

- In DSS-induced colitis mice, inhibition of Notch signaling with LY411,575 caused severe colitis exacerbation accompanied by tight junction deterioration and MLCK pathway activation.
- In vitro experiments confirmed that Notch signaling regulates tight junction function, and the MLCK inhibitor ML-7 effectively mitigated tight junction dysfunction induced by Notch inhibition.
- TNF-α-treated Caco-2 cells showed significantly increased expression of Jag1, Notch1, and Hes1, and silencing Jag1 with siRNA reduced NICD1 and Hes1 expression.
- The authors suggest that therapeutic Notch activation may represent a novel strategy for mucosal protection in IBD pathogenesis.

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