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Summary

HELICOBACTER PYLORI-INDUCED UP-REGULATED EXPRESSION OF IGF2BP1 PROMOTES GASTRIC TUMORIGENESIS VIA INHIBITING FERROPTOSIS

Jing Ning

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Stomach & H. Pylori

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IGF2BP1, an RNA-binding protein upregulated by Helicobacter pylori infection, promotes gastric cancer development by inhibiting ferroptosis through stabilization of SLC7A11 mRNA and represents a potential therapeutic target.

KEY POINTS

- IGF2BP1 expression was significantly upregulated in H. pylori-infected cell lines, mouse models, and patient samples
- IGF2BP1 promoted cell proliferation and inhibited ROS production by acting as an m6A methylation reader that binds and stabilizes SLC7A11 mRNA, thereby inhibiting ferroptosis
- Intratumoral injection of BTYNB, a selective inhibitor of IGF2BP1 binding to c-Myc mRNA, significantly inhibited tumor proliferation in xenograft models with high IGF2BP1 expression
- The study used multiple experimental approaches including in vitro cell culture, patient-derived organoids, in vivo mouse models, transcriptome sequencing, and RNA immunoprecipitation to characterize mechanisms
- The findings identify IGF2BP1 as a potential therapeutic target in gastric mucosal diseases associated with H. pylori infection

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