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Summary

CHIA DEFICIENCY IN THE CHIEF CELLS IS EFFICIENT TO INDUCE SPEM VIA IL-6/STAT3 SINGLING PATHWAY

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CHIA deficiency in chief cells induces spasmolytic polypeptide-expressing metaplasia independently of parietal cell loss via IL-6/STAT3 signaling in mice, with progressive CHIA loss observed across human gastric tissue from normal epithelium through SPEM to adenocarcinoma.

KEY POINTS

- CHIA deficient mice exhibited severe gastric pre-neoplastic phenotype with advanced age, including chief cell loss followed by chronic inflammation, SPEM, and high-grade intraepithelial neoplasia, while slight parietal cell loss occurred only at the HGIN stage.
- Deletion of CHIA in chief cells resulted in upregulation of SPEM markers TFF2 and MUC6, progressive reduction of gastric stem cell markers Lrg5 and Mist1 from 8 days to 15 months after birth, and spontaneous inflammation with upregulation of IL-6, IL33, IL25, IL-13 and IL17.
- Absence of CHIA caused epithelial cell hyperplasia with increased proliferation detected by Ki67 and suppressed apoptosis including BCL-2, BCL-xl, and CyclinD1 via activation of IL6/STAT3 signaling pathway.
- Human gastric adenocarcinoma tissues displayed significantly decreased CHIA mRNA and protein expression compared with healthy controls, with immunohistochemical analysis of human tissues showing progressive decrease in CHIA expression from normal gastric epithelium through SPEM to gastric cancer.
- This research is relevant to investigators studying gastric metaplasia pathways and the role of chief cell alterations in gastric carcinogenesis.

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