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Summary

ACIDIC MAMMALIAN CHITINASE DEFICIENCY RESULTS IN CHIEF CELLS PYROPTOSIS AND GASTRIC REGIONAL IMMUNITY REMODELING IS THE NEW CELLULAR ORIGIN OF SPEM

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Chief cell-specific CHIA deficiency induces SPEM through endoplasmic reticulum stress-mediated pyroptosis and IL-33/ST2-M2 macrophage axis activation, independent of parietal cell loss.

KEY POINTS

- In CHIA knockout mice, chief cell loss with M2 macrophage infiltration and SPEM formation occurred while parietal cells remained, demonstrated by histology and single-cell sequencing.
- CHIA deficiency caused endoplasmic reticulum dilation, mitochondrial membrane disruption, accumulation of ER stress markers (PERK, XBP-1, eIF2α/P-eIF2α, GRP78), and upregulation of pyroptosis markers (NLRP3, ACS, GSDMD, NEK7, Caspase1, Caspase11, IL-1β, IL-18).
- CHIA deficiency activated the IL-33/IL-13 circuit and induced M2 macrophage polarization (F4/80 and CD163+ colocalized with NLRP3) at the gastric mucosal base.
- CHIA over-expression in rescue mice reversed the pyroptosis, ER stress, immune remodeling, and SPEM development compared with controls.
- Chief cell initial loss is sufficient to induce SPEM through the IL-33/ST2-M2 macrophage polarization axis, independent of parietal cell loss.

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