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Summary

PARIETAL CELL-SELECTIVE SLC26A9 DEFICIENCY IN MICE INDUCE SPEM BY TRIGGERING M2 MACROPHAGE POLARIZATION VIA IL-33 SIGNALING

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Parietal cell-specific deletion of Slc26a9 in mice leads to development of spasmolytic polypeptide-expressing metaplasia (SPEM) through IL-33-mediated M2 macrophage polarization.

KEY POINTS

- Slc26a9fl/fl/Atp4b-Cre mice with parietal cell-specific Slc26a9 knockout exhibited autoimmune gastritis at 1 month with loss of parietal cells, oxyntic atrophy, and lymphocyte infiltration, followed by SPEM and hypergastrinemia at 6 months.
- Slc26a9fl/fl/Atp4b-Cre mice showed significantly increased M2 macrophage polarization markers (Arg1, Klf4, Fizz1, Ym1, Mrc1, CCL24, CCL22) and accumulation of F4/80+CD163+ cells compared to control mice.
- Gastric alarmin IL-33 was released with upregulation of SPEM markers TFF2 and MUC6 in knockout mice, and there was a corresponding increase in M2 macrophages capable of secreting IL-33.
- Overexpression of Slc26a9 in an IL-33-induced chronic SPEM model reduced SPEM formation and suppressed IL-33 downstream immune signaling as well as M2 macrophage polarization.
- This preclinical study is relevant to researchers investigating mechanisms of gastric metaplasia and the role of parietal cells in gastric mucosal immunity.

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