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Summary

SLC26A9 DEFICIENCY LEADS TO PARIETAL CELLS PYROPTOSIS AND ACTIVATION OF IL-25-ILC2-M2 MACROPHAGE AXIS, WHICH IS THE KEY EVENT TO INDUCE SPONTANEOUS AIG AND PROGRESS TO SPEM

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Oesophagus

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Parietal cell-specific deletion of Slc26a9 in mice causes autoimmune gastritis and progression to precancerous metaplasia through pyroptosis and immune remodeling.

KEY POINTS

- Slc26a9-deficient mice exhibited parietal cell loss, oxyntic atrophy, lymphocyte infiltration, and mitochondrial damage with swollen mitochondria and disrupted cristae
- Slc26a9 deletion led to accumulation of pyroptosis factors including NLRP3, Caspase-1, ASC, Gasdermin D, IL-1β and IL-18, with activated Gasdermin D binding mitochondrial cardiolipin and releasing mitochondrial mediators
- CD4+ T cells expressing H+/K+-ATPase-specific TCR infiltrated the stomach with upregulation of IFN-γ, IL-2, IL-11 and IL-17
- Hypergastrinemia induced Tuft cell migration and IL-25 release, with upregulation of ILC2 marker GATA3 and M2 macrophage markers F4/80 and CD163
- The IL-25-ILC2-M2 macrophage axis activation following Gasdermin D-mediated mitochondrial damage represents a key mechanism for autoimmune gastritis development and progression to SPEM

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