NON-ENZYMATIC ACTIVITY OF CHIA TRIGGERS CHIEF CELL INITIAL LOSS AND REGIONAL IMMUNE REMODELING, WHICH IS THE KEY EVENT TO INDUCE SPEM DEVELOPMENT

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UEG Week Barcelona 2026 | Presentation

Digestive Oncology | Stomach & H. Pylori | Mechanisms & Personalised Medicine | Immunology | Oesophagus

October 18, 2026

Chief cell-specific CHIA deficiency is sufficient to induce chief cell loss and SPEM formation independent of parietal cell loss, through ER stress-mediated pyroptosis and immune remodeling.

KEY POINTS

- CHIA knockout mice (CHIA^rox/rox-GIF-Dre) showed chief cell loss and SPEM formation while parietal cells remained intact, demonstrating that chief cell initial loss is the first and key step to induce SPEM.
- CHIA deficiency caused ER stress with accumulation of PERK, eIF2α, and GRP78, upregulation of pyroptosis markers NLRP3, GSDMD, and IL-1β, and triggered ER stress-mediated chief cell pyroptosis.
- CHIA deficiency activated the IL-33/IL-13 pathway and caused IL-33 upregulation colocalized with M2 macrophage markers F4/80 and CD163+ with NLRP3, indicating gastric regional immune remodeling.
- Overexpression of CHIA rescued enzymatic activity, suppressed ER stress-mediated chief cell pyroptosis and aberrant immune remodeling, and alleviated SPEM development despite identical chitin deposition, indicating the effect is independent of enzymatic activity.
- CHIA plays an important regulatory role in tissue and organ remodeling through non-enzymatic mechanisms, challenging the traditional view that parietal cell loss is the initial trigger of SPEM.

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