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Summary

SERPINB3 PROTECTS AGAINST REFLUX-INDUCED ESOPHAGEAL ADENOCARCINOMA: EVIDENCE FROM MURINE MODELS AND HUMAN TISSUES

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

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SerpinB3 deficiency accelerates reflux-induced esophageal carcinogenesis in a murine model, with loss associated with higher adenocarcinoma incidence at 9 months (p=0.022) and suppressed epithelial stress response and immune function.

KEY POINTS

- At 9 months, SerpinB3-knockout mice and wild-type mice treated with PAR2 inhibitor (which downregulates SerpinB3) showed significantly higher incidence of adenocarcinoma (p=0.022).
- At 6 months, SerpinB3-knockout mice exhibited increased lesion extension (p=0.036) and a trend toward more advanced histological stage (p=0.056).
- Transcriptomic analysis revealed coordinated downregulation of apoptosis, autophagy, antigen presentation, Notch and Hedgehog signalling in knockout mice, with strongest reductions in metabolic stress, MAPK and PI3K-Akt pathways.
- Immune cell profiling suggested a trend toward reduced T- and NK-cell infiltration in knockout tissues.
- In human esophageal adenocarcinoma samples, low SerpinB3 expression was associated with decreased CD8+ T-cell infiltration and reduced cytotoxic activity (CD107a+).
- Findings suggest a stage-dependent role for SerpinB3, with protective effects during early reflux-induced injury contrasting with previously reported tumor-promoting functions in established cancers, and reduced expression may serve as a biomarker for increased progression risk.

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