FUNCTIONAL INACTIVATION OF THE EXTRACELLULAR MATRIX PROTEIN EMILIN-1 PROMOTES GASTRIC PRE-NEOPLASTIC LESIONS: EVIDENCE FROM KNOCKOUT, MUTANT, AND PHARMACOLOGICAL MOUSE MODELS

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EMILIN-1 suppresses gastric pre-neoplastic progression, and its inactivation by neutrophil elastase is a critical pathogenic event in early gastric carcinogenesis.

KEY POINTS

- In mouse models, tamoxifen treatment induced SPEM in all genotypes, with greater lesion severity in EMILIN-1 knockout and E955A mutant mice compared to wild-type mice, although not statistically significant.
- DMP-777 treatment led to more pronounced pre-neoplastic alterations in EMILIN-1-deficient and mutant mice, whereas wild-type mice displayed milder changes.
- EMILIN-1 exerts a protective function in the gastric environment through its gC1q domain, which modulates epithelial-stromal signaling and immune homeostasis by binding to α4β1/α9β1 integrins.
- The gC1q domain is specifically targeted and cleaved by neutrophil elastase, resulting in functional inactivation of EMILIN-1.
- Pharmacological targeting of this axis with selective inhibitors may represent a novel preventive strategy in early gastric carcinogenesis.

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