

AN EMILIN-1-ALTERED MICROENVIRONMENT PROMOTES TUMOR CELL PROLIFERATION AND LYMPH-NODE INVASION IN MOUSE MODELS OF GASTRIC CANCER

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Poster

Oesophagus

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Loss of the extracellular matrix protein EMILIN-1 promotes gastric cancer onset, progression, and lymphatic metastasis in mouse models through $\alpha4/\alpha9\beta1$ integrin pathways.

KEY POINTS

- EMILIN-1 knockout and integrin-binding-deficient mutant mice developed larger, earlier gastric tumors with statistically significant increases in intratumoral lymphangiogenesis compared to wild-type mice when injected with syngeneic YTN16 gastric cancer cells.
- Enhanced lymph node metastasis and higher histopathologic invasion scores were observed in EMILIN-1 mutant animals in models of tumor transplantation and peritoneal carcinomatosis.
- In a chemically-induced gastric carcinogenesis model using N-methyl-N-nitrosourea, EMILIN-1 knockout mice had higher tumor incidence, bigger adenomas, and lower survival after 30 weeks.
- The integrin-binding-deficient E933A knock-in model produced similar results to the knockout, indicating EMILIN-1's oncosuppressive effects are mediated through $\alpha4/\alpha9\beta1$ integrin interaction.
- This work presents a novel animal model targeting a gastric tumor microenvironment component and may be relevant to researchers investigating extracellular matrix contributions to gastric cancer biology.

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