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Summary

TSPAN8 FACILITATES CANCER-ASSOCIATED FIBROBLAST-INDUCED GASTRIC CANCER PROGRESSION VIA WNT SIGNALING

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TSPAN8 is identified as a central epithelial hub gene in gastric cancer that mediates tumor cell responsiveness to cancer-associated fibroblasts through a Wnt/β-catenin signaling axis and is associated with poor overall survival.

KEY POINTS

- Co-expression network analysis of single-cell RNA-seq data identified TSPAN8 as a central hub gene localized to epithelial clusters, significantly upregulated in gastric cancer and associated with poor overall survival.
- TSPAN8 depletion suppressed gastric cancer cell proliferation and migration, whereas TSPAN8 overexpression enhanced these malignant phenotypes in functional assays.
- Cancer-associated fibroblast-derived cues promoted proliferation and motility, but these effects were attenuated by TSPAN8 knockdown and amplified by TSPAN8 overexpression, establishing TSPAN8 as a critical mediator of CAF-driven tumor-promoting activity.
- Ligand-receptor analysis suggested preferential CAF-to-epithelium signaling toward TSPAN8-high cells with enrichment of Wnt-related interactions, and CAF co-culture increased β-catenin levels in gastric cancer cells, further strengthened by TSPAN8 overexpression.
- The findings reveal a TSPAN8-CAF-Wnt/β-catenin axis that amplifies CAF-driven progression and suggest targeting TSPAN8-mediated stromal-epithelial communication may represent a therapeutic opportunity for fibroblast-rich gastric cancers.

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