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Summary

DKK3 IN PANCREATIC CANCER – ELUCIDATING THE ROLES OF A DOUBLE-EDGED SWORD

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Pancreas

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Loss of DKK3 accelerates pancreatic ductal adenocarcinoma progression in a KRAS-driven mouse model, promoting early metaplastic lesions, increased invasiveness, and fibrosis.

KEY POINTS

- DKK3-deficient mice showed significantly more acinar-to-ductal metaplasia and precursor lesions from 10 weeks onward, translating into substantial invasiveness at 36 weeks and reduced survival
- Loss of DKK3 positively correlated with higher fibrotic content and increased myofibroblast formation
- DKK3-null acinar cells demonstrated significantly increased capacity to form ductal structures in ex vivo assays
- Tumor cells lacking DKK3 exhibited higher mesenchymal marker expression (Vimentin, N-cadherin) and increased invasion and migration compared to controls
- The findings suggest DKK3 has tumor-restraining capability within the epithelial compartment and could potentially serve as a stage-specific biomarker for early PDAC detection

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