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Summary

THERAPEUTIC POTENTIAL OF TARGETING PROTEIN HYPER-SUMOYLATION IN CHOLANGIOCARCINOMA

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Aberrant SUMOylation machinery is overexpressed in cholangiocarcinoma and correlates with poor prognosis, while genetic and pharmacological SUMOylation inhibition halts tumor growth in preclinical models.

KEY POINTS

- The SUMOylation machinery was overexpressed and overactivated in human cholangiocarcinoma cells and tumors, correlating with poor prognosis
- Genetic inhibition using CRISPR/Cas9 against UBE2I and pharmacological inhibition with S-adenosylmethionine or ML792 reduced cholangiocarcinoma cell proliferation, impeded colony formation in vitro, and induced apoptotic cell death
- SUMOylation depletion halted tumorigenesis in subcutaneous cholangiocarcinoma models in vivo
- SUMOylation deficiency in cholangiocarcinoma cells reduced cancer-associated fibroblast and endothelial cell proliferation and impaired macrophage polarization towards an anti-inflammatory M2-like phenotype
- The findings suggest that impaired SUMOylation may represent a potential new therapeutic strategy for patients with cholangiocarcinoma

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