

KLF5 UPREGULATION IS A COMMON EVENT IN CHOLANGIOCARCINOMA, ACTING AS AN ONCOGENE AND CONSTITUTING A BAD PROGNOSTIC FACTOR

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KLF5 is upregulated in cholangiocarcinoma across multiple patient cohorts, correlates with poor prognosis, and its inhibition with ML264 blocks tumor growth and enhances chemotherapy sensitivity in preclinical models.

KEY POINTS

- KLF5 expression was upregulated in human CCA tissues from five different patient cohorts compared to normal liver tissue and bile ducts, independent of tumor mutational background
- High KLF5 levels correlated with lymph node invasion, worse overall survival, and served as an important prognostic factor
- Genetic ablation of KLF5 in CCA cells decreased proliferation, colony formation, and migration while promoting cell cycle arrest at G1/S and apoptosis in vitro, with no tumor development observed in xenograft models
- Pharmacological inhibition of KLF5 with ML264 blocked tumor growth in vivo and the combination of ML264 with standard chemotherapy (gemcitabine plus cisplatin) completely halted CCA tumor growth in mice
- Both genetic and pharmacological KLF5 inhibition sensitized CCA cells to chemotherapy-induced apoptosis, with altered expression of chemoresistance-related proteins that significantly correlated with KLF5 levels in human CCA tissues

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