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Kruppel-Like Factor 2 in Cholangiocarcinoma

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Background: Kruppel-like factor 2 (KLF2) is a transcription factor with tumor suppressive functions that has dysregulated expression in several cancers. The potential role of KLF2 in cholangiocarcinoma is as of yet unknown.

Objective: We sought to investigate functional effects of KLF2 in cholangiocarcinoma and hypothesized that KLF2 regulates proliferation, migration and apoptosis in cholangiocarcinoma cells.

Methods: Cholangiocarcinoma cell line KMCH was employed as well as Hek293T. Cells were stably transfected with KLF2 plasmid containing puromycin selection vector. Proliferation was measured using MTT assay. Transwell and scratch assays were used to evaluate migration. Apoptosis was induced using either TRAIL or staurosporine and cell death was measured by caspase 3/7 activity or nuclear morphology changes.

Results: Expression of KLF2 led to reduced growth by 24 hours in KMCH cells compared to parental cells and by 48 hours in Hek293T. KMCH cells with overexpression of KLF2 exhibited significantly reduced migration over 72 hours compared to parental cells. Induction of cell death in KLF2-expressing KMCH cells led to less apoptosis compared to parental cells. No difference was observed in apoptosis resistance between KLF2 transfected Hek293T cells compared to parental.

Conclusion: We found that KLF2 expression leads to reduced proliferation and migration as well as increased resistance to apoptosis, reflecting an overall more quiescent phenotype. Experiments are underway to evaluate KLF2 in an *in vivo* cholangiocarcinoma model.