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J Cancer Sci Ther 2019, Volume 11
DOI: 10.4172/1948-5956-C1-160

ZFX promotes proliferation and metastasis of pancreatic cancer cells via the MAPK pathway

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Background & Aims: The role of ZFX in tumourigenesis is unclear. We aimed to study ZFX expression, regulation, function and the clinical implications of this protein in human pancreatic cancer (PCa).

Methods: 122 patients with histologically confirmed PCa who underwent surgery were recruited for this study. Tumor samples and PCa cell lines were used to examine ZFX. Various cell functions related to tumourigenesis were assessed. *In vivo* mouse tumor xenografts were used to confirm the *in vitro* results.

Results: Patients with ZFX-positive tumors had worse overall survival than patients with ZFX-negative tumors. The depletion of ZFX using lentiviral shRNAs significantly inhibited cell proliferation by inducing cell cycle arrest in G0/G1 phase and resulted in increased cell apoptosis and invasive repression. *In vivo* studies confirmed that ZFX promoted tumor growth. Mechanistically, MAPK pathway activation was involved in the oncogenic functions of ZFX.

Conclusion: ZFX acts as a putative oncogene in PCa and could be a novel therapeutic target for this disease.

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