J Cancer Sci Ther 2018, Volume 10
DOI: 10.4172/1948-5956-C11-154

Abrogation of glutathione peroxidase-1 drives emt and chemoresistance in pancreatic cancer by activating ros-mediated akt/gsk3β/snail signaling

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Purpose: Pancreatic ductal adenocarcinoma (PDAC) stays one of the deadliest malignancies around the world, mostly because of tumor chemoresistance.Numerous studies have shown that glutathione peroxidase-1 (GPx1) play various roles in the development and progression of multiple tumors. However, its role in pancreatic cancer remains unclear. In this study, we sought to elucidate the function of GPx1 in pancreatic cancer malignancy and gemcitabine (GEM) resistance. Experimental Design: PDAC tissue microarrays were used to evaluate the correlation between GPx1 expression and clinicopathological features. Cytobiology, sub-atomic science measures and mouse models were performed to research the point by point instruments. At long last, RNA-sequencing was performed in the scramble-shRNA and GPx1-shRNA MiaPaCa-2 cells to distinguish core signaling pathways.

Results: The level of GPx1 expression was negatively associated with overall survival (OS) in patients with PDAC. Silencing of GPx1 resulted in an epithelial-mesenchymal transition (EMT) phenotype and increased chemoresistance to GEM in vitro and *in vivo* Moreover, actuation of Akt/GSK3β/Snail signaling was exhibited to be engaged with this procedure.

Conclusions: Our outcomes uncover that GPx1 could restrain EMT and chemoresistance by directing Akt/GSK3β/Snail pivot in PDAC.

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