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S100A14 promotes progression and gemcitabine resistance in pancreatic cancer

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Background & Aims: S100 calcium binding protein A14 (S100A14) is a member of the S100 protein family that plays an important role in the progression of several types of cancer. However, its expression and role in pancreatic ductal adenocarcinoma (PDAC) have not yet been clarified. Here, we demonstrated the important roles ofS100A14 in the progression and chemoresistance of PDAC.

Methods: Gene expression microarray analysis was conducted in four pairs of human PDAC tumor and corresponding non-tumor tissues. Quantitative real-time PCR (qRT-PCR) was used to determine S100A14 mRNA levels in PDAC tissue samples and cell lines. S100A14 protein levels in PDAC tissue microarray were examined by Immunohistochemical staining (IHC). The functions of S100A14 in cell proliferation and transformation were examined by colony formation assay and soft agar assay respectively. The roles of S100A14 in tumorigenicity were examined in PDAC xenografted mice model. The effect of S100A14 on chemo-resistance of gemcitabine was tested in vitro.

Results: S100A14 mRNA levels were dramatically upregulated in human PDAC samples compared with adjacent non-tumor tissues. IHC staining showed that S100A14 protein level was higher in PDAC tissues compared to non-tumor tissues. Moreover, the abundance of S100A14 was positively correlated to the increase of cancer stages. S100A14 overexpression promoted PDAC cell proliferation, transformation, migration, and invasion, whereas S100A14 knockdown inhibited these properties. Knock-down of S100A14 significantly inhibited SW1990 xenografted tumor growth. In addition, S100A14 knockdown enhanced the chemosensitivity of CAPAN2 cells to gemcitabine in vitro.

Conclusion: S100A14 was overexpressed in human pancreatic cancer tissues and played important roles in cell proliferation, transformation, tumorigenicity and chemo-sensitivity of gemcitabine.

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