Introduction
Pancreatic ductal adenocarcinoma (PDAC) is a deadly disease that is predicted to be the second leading cause of cancer death in USA in 2030. The overall 5-year survival rate for pancreatic cancer in USA is only 11%. Figure 1 shows the multistep progression of pancreatic cancer from normal duct to infiltrating cancer. This progression occurs through a series of histologically defined precursors called Pancreatic Intraepithelial Neoplasia (PanIN), which are precursors to pancreatic cancer (PDAC).

Methods
In our laboratory we use in-vitro and in-vivo model systems to study KDM4C in PDAC. The in-vitro models are (i) human and mouse tissues for assessment of KDM4C expression, and (ii) human and mouse pancreatic cancer cell lines with CRISPR/Cas9 edited knockout of endogenous KDM4C. For in-vivo analyses, we have athymic mice that are injected with PDAC cells bearing knockout of KDM4C versus control lines, as well as novel genetically engineered mouse models of PDAC with deregulated KDM4C expression.

Results

Kdm4C is overexpressed pancreatic cancer tissues. The downregulation of Kdm4C leads to reduction of anchorage independent growth in the mouse pancreatic cancer cell line MT4. The down regulation of MAP kinase signaling pathway by Western blot suggests that Kdm4c may be involved in direct or indirect regulation of a key effector of the Ras signaling pathway. All these data might be helpful in future studies to investigate the role of Kdm4C in regulating H3K9 methylation and expression of oncogenic transcripts involved in pancreatic cancer.

References

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