AB106. P080. Interferon gamma-inducible protein 10 in pancreatic cancer progression

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Abstract: CXCL10 or the Interferon gamma-inducible Protein 10 (IP10) belongs to the CXC subfamily of chemokines. Classically, chemokines are known to modulate leukocyte trafficking of cells expressing corresponding receptors. CXCL10 expression in pancreatic ductal adenocarcinoma (PDA) patient samples has been correlated with CXCR3 (receptor for CXCL10) positive regulatory T cell (T_{reg}) infiltration contributing to an immuno-suppressed tumor microenvironment. However, other studies suggest a more direct and non-classical role of CXCL10 in promoting acinar cell injury and apoptosis. Chronic pancreatitis patients have also been shown to express high levels of CXCL10 which is suggestive of its role in the pathogenesis of the disease. Clinically, there is a significant overlap between chronic pancreatitis and pancreatic cancer, but the role of CXCL10 specifically in the context of genetic mutations causing pancreatic cancer has not been fully explored. Our preliminary in situ hybridization data show that the cells in PanIN lesions of $p48^{Cre}$;LSL-$Kras^{G12D}$ mice express CXCL10 in contrast to no expression seen in normal adjacent acinar cells, whereas the receptor CXCR3 is highly expressed in the normal adjacent acinar cells as compared to that in the precursor lesion cells. Induction of CXCL10 expression in the PanIN cells and expression of CXCR3 in normal adjacent acinar cells suggests an important role of this chemokine in exacerbating the inflammatory pancreatic environment during the development of pancreatic cancer. This study aims to (I) delineate the mechanism of induction of CXCL10 expression in areas with precursor lesion; (II) determine the effect of CXCL10 on normal acinar cells; and (III) test if CXCL10 neutralization in the pancreas has an effect on the progression of the disease.

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