Identification of a novel IL-5 signaling pathway in chronic pancreatitis and tumor cells

Sarah Beth Gitto
Jordan M Beardsley
Sai Preethi Nakkina
Jeremiah L Oyer
Kathryn A Cline
Sally A Litherland
Alicja J Copik
Amr S. Khaled
Na'im Fanaian
J. Pablo Arnoletti
Deborah A Altomare

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Abstract

Pancreatic ductal adenocarcinoma is one of the most lethal cancers, with a five-year survival rate of only 9%. Inflammation-mediated tissue damage plays a role in pancreatic cancer, and chronic pancreatitis is a risk factor for cancer development. Unfortunately, exactly how inflammation drives cancer initiation remains unclear. A recent study identified a novel signaling pathway connecting inflammation to pancreatic cancer. In a mouse model of chronic pancreatitis, repeatedly inducing pancreatic inflammation accelerated tumor development. Immune cells including M2 macrophages and eosinophils were recruited to fibrotic regions of the pancreas, and expression of the cytokine IL-5, which can recruit these inflammatory cells, increased in response to inflammation. Pancreatic cells upregulated the receptor for IL-5 upon transformation to tumor cells, which increased tumor cell motility. Although more research is needed to fully understand the regulation of IL-5-mediated pathways in the pancreas, these results suggest that IL-5 signaling may be an ideal target for novel molecular therapeutics to block tumor spreading in pancreatic cancer.