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Pancreatic Cancer and the Tumor Microenvironment

Matthew Kovari*

Karmanos Cancer Insitute, Department of Biochemistry and Molecular Biology, Wayne State University School of Medicine, Detroit, MI, USA

Description

With a 5-year survival rate of fewer than 5%, pancreatic cancer is the fourth greatest cause of cancer-related deaths in both men and women in the United States (after lung, prostate/breast, and colon rectum). Despite fast breakthroughs in diagnostic and surgical techniques, patient survival has remained relatively unchanged over the last decade. The main causes of poor clinical outcomes are chemoresistance, early metastases, and late clinical presentation. This depressing reality strongly emphasises that new research avenues and alternative/complementary techniques are urgently needed to enhance pancreatic cancer clinical outcomes. Pancreatic stellate cells were first discovered as fat-storing cells in the pancreas, with functional similarities to hepatic stellate cells, the liver's counterpart cells [1-3].

PSCs were discovered in the pancreas in 1998 and are mostly seen in the peri-acinar, peri-ductal, and perivascular areas. The presence of intracellular fat droplets and the absence of -smooth muscle actin (-SMA) were used to identify the PSCs in a quiescent state when they were first isolated. Primary PSCs get activated in pancreatic injury or during cultivation, and their phenotype changes to that of a myofibroblast, with the production of -SMA and extracellular matrix (ECM) proteins and the removal of intracellular fat droplets. PSCs have several key signalling pathways that are involved in their functions. MAPK/ERKs have been proposed as important signal molecules regulating PSC functions, as they are activated by growth factors and ethanol. The activation of CXC-chemokine receptors is one of the MAPK/ERKs signalling pathways upstream signalling events [2,3].

Overexpression of CXCchemokine receptors (such as CXCR2 and CXCR4) and their associated ligands in pancreatic adenocarcinoma has been linked to a higher tumour grade and stage. Furthermore, a growing body of evidence suggests that inhibiting CXC-chemokine receptor signalling can diminish tumour proliferation, invasion, and tumor-induced angiogenesis both in vitro and in vivo. PDZ (PSD-95/DlgA/ZO-1) domain-containing proteins (also known as PDZ scaffolding proteins) have been shown to nucleate the formation of compartmentalised multi-protein complexes, which are essential for efficient and selective cell signalling. These membrane-bound PDZ scaffolding proteins interact with membrane proteins (such as receptors and channels) and their downstream effectors. Characterizing the PDZ domain-mediated chemokine receptor macromolecular signalling complex in PSCs could be a novel way to learn more about the molecular underpinnings of tumor-stroma interactions in pancreatic cancer, as well as a strategy to help patients transition to more effective chemotherapies [4,5].

The strong desmoplastic response, a pronounced increase in connective tissue around the tumour parts, is a major histological hallmark of pancreatic cancer, and the stroma can make up to 90% of the tumour. Desmoplastic of the pancreatic tumour has also been linked to its biological and clinical aggressiveness. As a result, research into the tumour microenvironment, which includes fibroblasts, peritumor nerves, endothelial cells, and macrophages, has progressively increased, with pancreatic stellate cell (PSC) becoming a rising star in pancreatic cancer research. In conclusion, patient survival in pancreatic cancer has improved only slightly in the recent decade, indicating that new treatments other than focusing on cancer cells alone are urgently needed.PSCs are the primary mediators of pancreatic cancer's substantial desmoplastic responses, which represent a distinct hallmark. Tumor progression is aided by the interaction between pancreatic cancer cells and PSCs [1,2].

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Conflict of Interest

The author reported no potential conflict of interest.

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***Address for Correspondence:** Matthew Kovari, Karmanos Cancer Insitute, Department of Biochemistry and Molecular Biology, Wayne State University School of Medicine, Detroit, MI, USA; E-mail: mat.kovari@up.ac.za*

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