SHORT COMMUNICATION

# Pancreatic Cancer Remains a Deadly Disease. Why?

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**ABSTRACT**

In comparison to many other malignancies, pancreatic cancer has an extremely low cumulative five-year survival rate-the percentage of all patients who are alive for five years after diagnosis-of about 5 to 10%. This is because considerably more people are diagnosed with stage IV disease after it has spread. Pancreatic cancer is the most lethal of all common malignancies and delays in detection and treatment can be fatal. When pancreatic cancer is detected early enough, it is possible to remove the pancreas, which is the sole cure for the condition. Only 10% of people with pancreatic cancer are discovered in time for life-saving surgery.

Although pancreatic cancer is uncommon, it is one of the worst types of cancer. This is because symptoms usually do not appear until the malignancy is advanced, making treatment difficult.

## INTRODUCTION

Significant progress has been achieved in our understanding of the biology of pancreatic cancer, as well as breakthroughs in patient management. Screening first-degree relatives of persons with multiple family members affected by pancreatic cancer may reveal non-invasive precursors of this malignant disease, according to preliminary evidence. The incidence and number of fatalities caused by pancreatic tumours have continuously increased, while the incidence and mortality of other prevalent cancers have decreased. Those with pancreatic cancer have a better chance of survival because surgical excision is now the only option for cure. Unfortunately, 80-85% of patients have severe, incurable illness. Furthermore, most chemotherapy drugs have a poor response to pancreatic cancer. As a result, we must comprehend the molecular pathways that contribute to the genesis and progression of pancreatic tumours. The most frequent and lethal form of pancreatic cancer, pancreatic ductal adenocarcinoma [1].

Pancreatic cancer is a deadly malignancy that primarily affects men of advanced age (40-85 years) and has an aggressive course. Its frequency has gradually increased in recent years. It is responsible for 2% of all cancers and 5% of all cancer-related deaths. Pancreatic cancer is the most common asymptomatic cancer. Adenocarcinomas account for 90% of all cases. 10% of the patients have a genetic predisposition. This disease has no early warning signals and spreads swiftly to surrounding organs, is one of the most lethal types of cancer. Pancreatic cancer can be caused by genetic germline or somatic acquired mutations in cancer-related genes and these mutations also contribute to cancer growth and metastasis [2].

Pancreatic cancer is a lethal illness with a high fatality rate due to challenges in early detection and spread. The tumour microenvironment created by interactions between pancreatic epithelial/cancer cells and stromal cells is crucial for pancreatic cancer growth and has been linked to chemotherapy, radiation treatment, and immunotherapy failure. Interactions between pancreatic cancer cells and stromal cells are required for microenvironment development. Components of the pancreatic cancer microenvironment that contribute to desmoplasia and immunosuppression are linked to a poor prognosis in patients. These components can promote metastasis by increasing angiogenesis/lymphangiogenesis, epithelial-mesenchymal transition, invasion/migration, and pre-metastatic niche development in primary and metastatic locales. Some chemicals are involved in both the creation of microenvironments and the spread of cancer. In this review, we focus on the mechanisms of pancreatic cancer microenvironment formation and discuss how the pancreatic cancer microenvironment participates in metastasis, suggesting that the pancreatic cancer microenvironment could be a potential target for combination therapy to improve overall survival [3].

Pancreatic cancer is one of the deadliest cancers due to its late diagnosis and poor treatment response. Tractable

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approaches for identifying and interrogating pancreatic tumorigenesis pathways are desperately needed. Organoid models were created using normal and cancerous murine and human pancreas tissues. Pancreatic organoids may be created quickly from resected tumours and biopsies, can be cryopreserved, and have ductal and disease-stage specific features. Orthotopically transplanted neoplastic organoids mimic the entire tumour formation process, starting with low-grade neoplasms and progressing to locally invasive and metastatic carcinomas. Organoids provide a platform for investigating genetic cooperation because of their potential to be genetically altered. Comprehensive transcriptional and proteomic investigations of murine pancreas organoids revealed changed genes and pathways as the disease progressed. Many of these protein alterations have been confirmed in real tissues, demonstrating that organoids provide a simple model system for discovering hallmarks of this lethal cancer [4].

Pancreatic cancer is still a fatal disease, with only an 8% 5-year survival rate. Even after surgical resection, the majority of patients have cancer recurrence. Over the previous decade, advances in chemotherapy regimens have resulted in a doubling of median overall survival. In this article, we discuss the management of advanced pancreatic cancer and highlight vaccine therapy as a potential treatment option [5].

## CONCLUSION

This article examines current approaches to diagnosing and treating resectable and advanced pancreas cancer. Pancreas cancer is a leading cause of cancer death; consequently, developing early detection measures and efficient treatment is critical. Imaging technology advancements, as well as the use of biomarkers such as CA 19-9, are transforming the way pancreas cancer is identified and staged. Although progress in pancreas cancer treatment has been slow, research into combination therapies incorporating both chemotherapeutic and biologic drugs is ongoing.

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