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Opinion Article

Biological and Immunological Characteristics of Pancreatic Ductal Adenocarcinoma

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DESCRIPTION

Pancreatic Ductal Adenocarcinoma (PDAC) is the most common pancreatic neoplastic disease, accounting for more than 90% of all pancreatic malignancies. PDAC is currently the fourth leading cause of cancer-related deaths worldwide, with a 5-year overall survival rate of less than 8%. PDAC is expected to become more common in the future, with projections indicating a more than two-fold increase in the number of cases over the next ten years, both in terms of new diagnoses and PDAC-related deaths in the United States and Europe.

Apart from the general ageing of our society, one particular reason for this condition is the obvious role of obesity and type 2 diabetes. Two emerging public health challenges in PDAC aetiology are Alcohol and tobacco abuse, which are well-known to increase the risk of several other types of cancer, including lung cancer and squamous cell carcinomas of the head and neck region. Finally, genetic predispositions, such as germline mutations in the Breast Cancer 1 (*BRCA1*) and Breast Cancer 2 (*BRCA2*) genes, MutL homolog 1(*MLH1*), Tumor protein p53 (*TP53*), or Cyclin-Dependent Kinase Inhibitor 2A (*CDKN2A*), represent additional risk factors for a subset of approximately 5%-6% of all PDAC patients.

PDAC's biological and immunological characteristics

Plasticity and heterogeneity in tumours: The pancreas contains exocrine (Acinar), epithelial (Ductal), and endocrine cells, with acinar cells being well known for their high degree of plasticity. This plasticity is thought to intiate pancreas homeostasis and regeneration because, unlike other gastrointestinal organs, the pancreas appears to lack a defined stem cell compartment. When exposed to certain macro- and micro environmental stimuli, such as tissue damage, inflammatory or stress conditions, acinar cells trans-differentiate to more epithelial (Ductal-like) phenotypes, a process known as acinar-to-ductal metaplasia (ADM). Acinar cells develop 'progenitor cell-like' characteristics during ADM, making them more vulnerable to pro-oncogenic such as activating mutations in the proto-oncogene Kirsten rat sarcoma viral oncogene homolog (*KRAS*), eventually transforming them into Pancreatic Intra-Epithelial Neoplasias (PanINs). This transformation is widely regarded as the first step in the development of PDAC, followed by a series of genetic in several tumour suppressor genes. A number of next generation sequencing approaches have been initiated in recent years to investigate the mutational and transcriptional of PDAC. These findings suggest that the gene encoding of proto-oncogenic GTPase *KRAS*, as well as several tumour suppressor genes, such as tumour suppressor protein 53 (TP53), Cyclin-Dependent Kinase Inhibitor 2A (*CDKN2A*), and Against Decapentaplegic Homologue 4 (*SMAD4*), are the most frequently altered and/or mutated in PDAC. *KRAS*, for example, was not only found to be mutated in the majority of PDAC tumours (>90%), but its mutant alleles were also amplified in a subset of samples, resulting in an acceleration of their tumor-promoting potential. Furthermore, the RAC-beta serine/threonine-protein kinase (*AKT2*) is frequently overexpressed in PDAC, and the activity of its upstream regulator, phosphoinositide 3-kinase (PI3K), is frequently increased, leading to increased tumour cell survival. Aside from these key mutations, several more uncommon alterations have been discovered in certain subsets of patients, such as germline mutations in DNA damage repair genes (e.g., breast cancer early onset genes 1/2 (*BRCA1/2*), partner and localizer of *BRCA2* (PALB2), and ataxia telangiectasia mutated protein serine/threonine kinase or somatic mutations in DNA mismatch repair regulator genes leading to increased microsa. It is worth noting that the transcriptomic landscape of PDAC is not entirely controlled by genetic changes.

CONCLUSION

PDAC is a cancer entity with a high malignancy, a particularly poor prognosis, and an ever-increasing patient population. Because of its aggressive biology and the fact that the majority of patients present in advanced or disseminated stages of disease, developing novel PDAC treatment strategies is one of the most difficult challenges in current oncological research. The findings of the last two decades have resulted in the development of a

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detailed multi-step model of PDAC development and progression. Although these findings have improved our understanding of PDAC as a disease, but none of them have yet been successfully developed into a therapeutic breakthrough.