

Special Issue

The Study of Molecular Pathogenesis and Therapeutic Strategies of Pancreatic Cancer

Message from the Guest Editor

I am pleased to invite you to contribute to the Special Issue entitled “The Study of Molecular Pathogenesis and Therapeutic Strategies of Pancreatic Cancer”.

Pancreatic cancer is an aggressive form of cancer characterised by poor prognosis and survival rate. Despite rapid progress in this field, and the fact that the core genetic mutations associated with pancreatic cancer are well known, their role in pancreatic cancer pathogenesis and progression is yet to be explored. Henceforth, delineating the heterogeneous molecular pathology of this cancer will facilitate the identification of new molecular targets for personalised therapeutic options. This Special Issue aims to discuss recent advancements in our understanding of new molecular targets, multifocal neoplasia associated with precancerous lesions, dysregulation/upregulation of cell signalling networks, pathophysiology of the complex tumour microenvironment and innovative therapeutic intervention in pancreatic cancer. In this Special Issue, original research articles and reviews are welcome. Research areas may include (but are not limited to) the following: original research articles and reviews.

Guest Editor

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Deadline for manuscript submissions

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About the Journal

Message from the Editor-in-Chief

Cancers is an international online journal addressing both clinical and basic science issues related to cancer research. The journal is publishing in Open Access format, which will certainly evolve to ensure that the journal takes full advantage of the rapidly changing world of information and knowledge dissemination. It publishes high-quality clinical, translational, and basic science research on cancer prevention, initiation, progression, and treatment, as well as other related topics, particularly to capture the most seminal studies in the rapidly growing area of immunology, immunotherapy, and tumor microenvironment.

Editor-in-Chief

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