

Invitation to the seminar on

Medical Radiation Physics and Ion-Beam Therapy

by

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Radiation-induced immunogenicity in pancreatic cancer

Pancreatic ductal adenocarcinoma (PDAC) remains a clinical challenge characterized by an alarmingly low survival rate. Despite surgical advances and new chemotherapy combinations, currently available treatment fails to improve the overall survival of PDAC patients largely due to an immunosuppressive tumor microenvironment. Radiation can induce cell death and reprogramme the tumor microenvironment. The cGAS-STING and RIG-I-MAVS pathways are part of the innate immune system and initiate type I interferon production. This study aims to exploit the radiation-induced DNA damage and inhibition of DNA repair or cell cycle checkpoints, to activate these pathways and enhance the immunogenic signalling in PDAC. Two different PDAC cell lines were used to test two different radiation modalities (X-rays and carbon ions) and regimens (single and hypofractionated dose) in combination with ATR and CHK1 inhibitors as well as the STING agonist diABZI. Cell survival, immunogenic cell death, accumulation of cytosolic dsDNA and micronuclei, gene expression profiles, and STING- and NF- κ B-dependent immune signaling were assessed. Here we demonstrate that ATR inhibition (ATRi) sensitizes BxPC-3 cells to the cytotoxic effects of radiation and potentiates the immunogenic effects of carbon ions and hypofractionated X-rays (3x8 Gy). Increased accumulation of cytosolic dsDNA and micronuclei was coupled with STING-dependent IFNB1 secretion and genome-wide induction of inflammatory gene expression programs, ultimately resulting in the activation of monocytes. This was not the case for PANC-1 cells, where radiation alone exerted immunosuppressive effects on monocytes. Our results support further evaluation of ATR inhibition in combination with radiotherapy in KRAS wild-type pancreatic cancer.

Tuesday, 30. June 2026, 09:00 am

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