Seminar

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Institute for Plasma Research

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Title: Dynamic Cellular Plasticity in Cancer: EMT Transitions and Stem Cell Differentiation Strategies

Speaker: Dr. Ankit Mathur
Bharathiar University, Coimbatore

Date: 23rd August 2024 (Friday)

Time: 10.30 AM

Venue: Online: https://meet.google.com/nkj-wmhk-bub

Abstract

In my upcoming talk, I will discuss my research journey, covering both my PhD and postdoctoral experiences. I have focused on understanding the cellular and molecular mechanisms underlying cancer progression and therapeutic resistance. During my PhD at the Institute of Nuclear Medicine and Allied Sciences, DRDO, Delhi, India, my thesis titled "An In Vitro Study of Low-Dose Radiation Sensitivity and Cellular Adhesion Protein Alterations During Progressive Stages of Neoplastic Transformation" explored the dynamics of cell adhesion molecules during neoplastic progression, particularly focusing on epithelial-mesenchymal transition (EMT) and mesenchymal-epithelial transition (MET). The study revealed that the low-dose hyper-radiosensitivity (HRS) response might be an inherent feature of progressively transforming cells, and that alterations in certain signaling proteins, such as connexin-43 (Cx43), are associated with this altered cellular behavior. Specifically, radiation-induced overexpression and mitochondrial translocation of Cx43 were highlighted as critical factors. The findings suggest that targeting low-dose radiation-induced signaling could offer new therapeutic strategies to counter tumor progression and systemic spread of micrometastases.

In my postdoctoral work at Dr. B.R. Ambedkar Center for Biomedical Research, University of Delhi, I extended my research to cancer stem cells (CSCs) and differentiation therapies. My research investigated the role of the natural compound esculetin in reversing epithelial-mesenchymal transition (EMT) and CSC differentiation in colorectal cancer and as a potential differentiating agent in Acute Myeloid Leukemia (AML). Using Kasumi-1 cells with AML-ETO translocation, we found that esculetin induced terminal differentiation of leukemic blast cells and downregulated the canonical Wnt signaling axis. Similarly, esculetin showed promise in reducing the aggressiveness of colon cancer cells by reversing EMT and CSC phenotypes.

In summary, my research contributions have provided novel insights into the mechanisms of EMT, CSC differentiation, and the potential therapeutic applications of targeting these processes, particularly through the use of natural compounds like esculetin.