Radiation Genome Systems Biology

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Research topics

What is the intracellular environment of cancer cells who was exposed to genotoxic stresses that provides an epigenetic landscape permissive for cell survival in vivo? How do cancer cells with such genome systems acquire genetic and epigenetic alterations that drive cancer spreading? To address these challenges, we are focusing on the following critical signaling proteins involved in mitotic events, which would become potential targets for the development of new therapeutic agents for cancer.

Outline of Research

A brief overview of our research

Working in the broad areas of cancer cell biology including cell transformation, DNA repair, cell cycle checkpoints, and invasion and metastasis, we have made several seminal discoveries; these include the discovery and characterization of mammalian proteins for driving tumor aneuploidy, such as the chromosomal passenger protein AIM-1 (Aurora-B) and its related proteins, and also the discovery and characterization of RhoGDIβ as metastasis-related gene. More recently, we characterized a new substrate of AIM-1/Aurora-B, which we named tRNA (cytosine- 5-)-methyltransferase SAKI. This protein is now also known as MISU and NSUN2. SAKI/MISU/NSUN2 is overexpressed in human cancer cells with an increased gene copy number, and is involved in carcinoma progression and plays a role in chemo-sensitivity.

Our research goal

We are interested in understanding how cancer cells survive and escape the cytotoxic effects of radiations and chemotherapeutic agents, eventually increasing invasive and metastatic properties. To this end, my group is now studying the role of several important proteins that are involved in both of mitotic events and apoptotic signaling. One of their goals is to understand the mechanisms that drive cancer spreading in the tumor microenvironment. Another goal is to provide a novel way to improve conventional radiotherapy and chemotherapy outcomes and/or a novel molecularly targeted therapy for cancer treatment.

Reference

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