



Department of Biotechnology  
Ministry of Science and Technology  
Government of India  
**DBT**



National Institute of  
Advanced Industrial Science  
and Technology  
**AIST**

# DBT - AIST International Laboratory for Advanced Biomedicine

  
**DAILAB**

**Classroom for Advanced & Frontier Education  
CAFE**

  
**DAILAB-CAFE**

## Series - 09

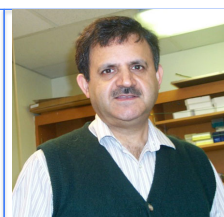
Date and Time – May 29, 2015 (16:30~17:15)

Venue - Central 4-1 (2F) Meeting Room 1

Speaker – Tej K. Pandita

Affiliation – The Houston Methodist Research Institute, Houston, TX 77030

E-mail: [tpandita@houstonmethodist.org](mailto:tpandita@houstonmethodist.org)



### Title : Chromatin status and DNA DSB Repair

Although we know a great deal about the pathways of DNA DSB repair, very little is known about how DSB repair occurs in its natural context in the cell, that is, within chromatin, which is an impediment for proteins accessing the DNA, yet the repair machinery is somehow able to navigate through the chromatin and successfully repair damaged DNA. It is becoming apparent that chromatin structure changes in multiple ways during DSB repair to regulate the efficiency and accuracy of DSB repair, as well as transducing the cell's response to DNA damage via the DNA damage response. However, our understanding of how the preexisting chromatin environment and its dynamic changes regulate DSB repair is in its infancy. We also know very little about how DSB repair occurs within regions of the genome that are simultaneously being transcribed. DSBs within transcribed regions presumably must be repaired especially rapidly and accurately to prevent the production of aberrant transcripts, but such mechanisms are currently unknown. Our mammalian cell lines have been engineered using CRISPR technology to introduce meganuclease I-SceI cleavage sites at defined unique locations within a highly transcribed gene, or within heterochromatin versus euchromatin. The cutting and repair efficiency of these systems is unparalleled in mammalian cells, with most of the cells having a single DSB at the same time in the population. These systems enabled us for simultaneous assessment of DSB repair kinetics and transcription efficiency while also allowing us to measure factor recruitment, protein post-translational modifications and chromatin structure at the DNA break by Chromatin ImmunoPrecipitation analysis. We will about our new data that could greatly enhance our understanding of DNA DSB repair in the native environment.

# DAI LAB-CAFE

## Series - 10

Date and Time – May 29, 2015 (17:20~18:00)

Venue - Central 4-1 (2F) Meeting Room 1

Speaker – Naduparambil K. Jacob PhD

Affiliation –Department of Radiation Oncology

The Ohio State University Comprehensive Cancer Center, Columbus, USA

E-mail: jacob.86@osu.edu



### **Title –Circulating microRNAs as Biomarkers of Radiation Response and Biodosimeters**

Approximately 50% of cancer patients receive radiation during their treatment course. Ionizing radiation is used to target cancers; however normal tissue toxicity is inevitable, which is manifested as acute radiation syndromes (ARS) in patients and as delayed or late effects such as pneumonitis and fibrosis in survivors. Optimization of an amplification-free hybridization based nCounter assay for evaluation of changes in circulating exosomal miRNAs detectable in body fluids such as serum, plasma and urine enabled us developing a panel of miRNA biomarkers, exhibiting dose and time dependent changes in rodents, non-human primates as well as in human patients. In the presentation, I plan to discuss our recent studies developing miRNAs as biomarkers of radiation injury in three major affected radiosensitive organs—bone marrow, gut and lung—with the manifestation of varying dose-time responses, combined effects and interactions. Significance of these findings in clinical radiation biodosimetry, triage in radiation accidents, early prediction of delayed/late effects and preclinical testing of radiation countermeasures will be discussed. Mechanisms by which miRNAs modulate DNA damage repair, inflammatory responses and molecular processes such as epithelial to mesenchymal transition driving progressive diseases will also be discussed.





**Dear Dr Pandita**

**Thank you !**

**for being CAFÉ 09 International Mentor**

**We all enjoyed your talk and appreciate your efforts !**

**DAILAB-CAFE**







**Dear Dr Jacob  
Thank you !**

**for being CAFÉ 10 International Mentor**

**We all enjoyed your talk and appreciate your efforts !**

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