Tata Institute of Fundamental Research
Survey No. 36/P, Gopanpally Village, Serilingampally, Ranga Reddy Dist., Hyderabad - 500107

Students' Annual Seminar

Connecting DNA Damage Responses (DDR) to the Cell Cycle and Genome Organization

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DNA damage can arise anytime in a cell from both endogenous sources like reactive oxygen species as byproducts of cellular metabolism, replication errors, or modification of bases or exogenous sources like radiation or environmental mutagens [1]. Specific checkpoints in the cell cycle and genome surveillance mechanisms regulate the fidelity of the replication by halting the cell cycle to repair such damages — which is why cellular responses to such stresses depend not only on the type of damage but also on the cell cycle stage of the cell [2]. In the past year I developed and improvised on a non-invasive microscopy-based technique to study cell cycle dependence of DDR and associated gene expression. In this presentation I will describe how P53—an important tumor suppressor gene [3]—is regulated during DNA damage across the cell cycle and discuss my initial endeavors to investigate the possible role that genome organization and nuclear architecture [4] may have in the observed cell-cycle dependent regulation of DDR associated gene expression.

References:

[1]. Marnett LJ. Oxyradicals and DNA damage, Carcinogenesis, 2000.
[2]. M. Shrivastava. Regulation of DNA double-strand break repair pathway choice, Cell Research, 2007.
[3]. Lane. p53, guardian of the genome, Nature, 1992.
[4]. Lemaıˆtre et al. Nuclear Position Dictates DNA Repair Pathway Choice, Genes and Development, 2014

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05:15 PM (Tea/Coffee at 04:45 PM)

Seminar Hall, TIFR-H