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Research Area: Chromatin Biology and Genome Stability

Research Interest: Genomic instability is a hallmark of cancer cells, and the cause of various genetic and developmental disorders. Such instabilities can result from S-phase replication errors, DNA damage caused by endogenous/exogenous agents, and/or M-phase chromosome segregation defects. Cellular surveillance systems, also known as 'checkpoints' work at different stages of the cell-cycle to promote damage repair and ensure genome integrity. Apart from checkpoint signaling proteins/kinases, hundreds of different proteins work collaboratively at S-phase and M-phase to execute cell cycle functions in a coordinated and timely fashion. In their absence, cells experience fatal consequences or enter aberrant developmental programs. Broadly, our research interest is directed towards understanding the intricacies of cellular security mechanisms and the coordinated protein network involved in genome surveillance and protection. We utilize genetic, biochemical and cell biological approaches to investigate (i) DNA Damage Response (DDR) following a DNA damage or replication stress to gain mechanistic insights on genomic instability (GIN), (ii) mitotic and meiotic chromosome segregation to learn the molecular mechanisms involved in chromosome instability (CIN) and (iii) the crosstalk between DNA replication and chromosome segregation to study how coordination between S- and M-phases helps to maintain genomic integrity. We take advantage of unicellular eukaryote budding yeast (*Saccharomyces cerevisiae*) with its unique genetic, biochemical and cell biological attributes, as model organisms for the majority of these studies. To understand the evolution of genomic surveillance systems across ascomycetes, we are extending the GIN- and CIN-related studies in fission yeast (*Schizosaccharomyces pombe*) – another evolutionarily distinct (from budding yeast) unicellular eukaryotic model organism. Moreover, we intend to start working with pathogenic yeasts to understand the molecular mechanism of different fungal diseases as well as to study fungi-caused multidrug resistance.

Current Research Projects and Funding:

1. DBT-Ramalingaswami Re-entry Fellowship project titled "A molecular approach to study genome and episome stability factors." (2018-2023)
2. DST-SERB Core Research Grant titled "A crosstalk between DNA replication and chromosome segregation machineries." (2021-2024)

Joining The SAU Lab: Research minded students interested to work on chromatin biology & genome stability field (as research scholars / voluntary research trainees / master's students for MSc thesis) can send their CVs (with a cover letter) at **soumitrasau16@gmail.com.** Students with their own fellowships (BET-JRF / NET-JRF) will be directly enrolled into Amity University Kolkata PhD program.

Selected recent publications: [complete list: https://pubmed.ncbi.nlm.nih.gov/?term=soumitra+sau]

1. *Access to PCNA by Srs2 and Elg1 Controls the Choice between Alternative Repair Pathways in* Saccharomyces cerevisiae. Arbel M, Bronstein A, **Sau S et al. mBio.** 2020 May 5;11(3).
2. *The Yeast PCNA Unloader Elg1 RFC-Like Complex Plays a Role in Eliciting the DNA Damage Checkpoint.* **Sau S et al. mBio.** 2019 Jun 11;10(3). [ASM Press, USA]
3. *A structure-function analysis of the yeast Elg1 protein reveals the importance of PCNA unloading in genome stability maintenance.* Shemesh K, Sebesta M, Pacesa M, **Sau S et al. Nucleic Acids Research.** 2017 Apr 7;45(6):3189-3203. [Oxford Press, UK]
4. *A selfish DNA element engages a meiosis-specific motor and telomeres for germ-line propagation*. **Sau S et al. Journal of Cell Biology**. 2014 Jun 9;205(5):643-61. [Rockefeller University Press, USA]