Prof. Das's research has made significant strides in cancer therapy by identifying PRMT5 as a critical therapeutic target for repairing DNA breaks associated with Top1-DNA trapped complexes. His work established PRMT5 as a major resistance factor in cancer chemotherapy, showing that its knockdown enhances cancer cell sensitivity to camptothecin. This groundbreaking discovery, published in *Nucleic Acids Research* (2018) and highlighted in *Nature Reviews Molecular Cell Biology* (2019), positions PRMT5 as a promising druggable candidate. In a separate study, Prof. Das's lab at IACS developed an innovative assay system to analyze human Top1 subnuclear dynamics using live-cell imaging of EGFP-tagged Top1. This assay, combined with fluorescence recovery after photobleaching and kinetic modeling, allowed for the quantification of CPT-induced Top1cc formation across the nuclear genome. The research further revealed the critical roles of Trp205 and Asn722 in Top1 subnuclear dynamics, with the N722S Top1 mutant showing restricted nucleolar localization in the presence of CPT due to its deficiency in forming Top1cc. These robust assays have paved the way for the development of new Top1 inhibitors using live-cell microscopy, underscoring the lethal impact of unrepaired DSBs on dividing malignant cells (*Das SK.*, *Nucleic Acids Research*, 2016).

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