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Investigating the nexus between DNA repair pathways and genomic instability in cancer



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DNA double-strand breaks are one of the most lethal lesions to a cell that can be repaired by one of the two cellular pathways; non-homologous end joining or homologous recombination. Homologous recombination genes are particularly attractive targets for precision cancer therapy because these genes have altered expression patterns in cancer cells when compared with normal cells and these genetic abnormalities can be targeted for selectively killing cancer cells while leaving normal cells unscathed. Synthetic lethality is thought to be the new frontier of cancer therapeutics because it overcomes the limitation of chemotherapy, which is unable to discriminate between cancer cells and normal cells. Two genes are synthetically lethal when simultaneous disruptions of both genes gives rise to a lethal phenotype, while the disruption of either gene alone is viable. Many homologous recombination genes have synthetic lethal relationships with oncogenes and tumor suppressor genes, which can be targeted for the development of cancer therapy- an approach referred to as combination therapy. In my presentation, I will summarize recent progress in understanding both the functioning and the regulation of the DNA repair machinery and elaborate on the clinical applications of these proteins in cancer therapy.

Biography

Sonali Bhattacharjee did her B.Sc in Biotechnology from Bangalore University in 2006 and M.Sc in Applied Genetics from Bangalore University in 2008. She then moved to England to pursue her DPhil (Phd) in Biochemistry from Oxford University where she studied the role of Fml1 and its partner proteins Mhf1 and Mhf2 in promoting genome stability. She was awarded her DPhil in 2012. During her time at Oxford, she was also a tutor at Greene's College, Oxford. In 2013, she moved to Cold Spring Harbor Laboratory, New York. At CSHL, her work has focused on understanding the epigenetic regulation of DNA repair. She is also an academic tutor at the Watson School of Biological studies, the school for graduate studies at CSHL.

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