J Biotechnol Biomater 2018, Volume 8
DOI: 10.4172/2155-952X-C6-104



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

**Sonali Bhattacharjee**
Cold Spring Harbor Laboratory, USA

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 the presentation, author 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.

bhattacharjee@cshl.edu

**Notes:**