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A novel role of 53BP1 in alternative non-homologous end joining

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DNA double-strand breaks (DSBs) are one of the most cytotoxic forms of DNA damage that cause cell death or genomic instability if unrepaired or misrepaired. Homologous recombination and non-homologous end joining are two major pathways required for repairing DNA DSBs. A third modality of repair, alternative NHEJ (Alt-NHEJ), has come to be appreciated recently. This lecture will address a novel role of 53BP1 in Alt-NHEJ via promoting single strand DNA (ssDNA) resection/deletion, perhaps in the G1 and early S phase of cell cycle. The role of 53BP1 in Alt-NHEJ explained the previous observation that 53BP1 specifically promotes DNA DSBs repair in the G1 phase cells and contributes to G1-specific cell survival in response to ionizing radiation. In addition, this lecture will discuss how 53BP1 regulates Alt-NHEJ in the absence to BRCA1, a critical homologous recombination repair protein encoded by a tumor suppressor gene frequently mutated in familial breast and ovarian cancers. This presentation will provide the supporting evidence that Alt-NHEJ is a highly regulated DNA DSBs repair pathway which is differentially regulated by 53BP1 in the context of presence or absence of BRCA1. In both situations, 53BP1 is essential for maintenance of genome integrity.

Biography

Junran Zhang has extensive experience with DNA double strand breaks repair pathways and has more than 10 years of experience in this field, having published 20 high quality papers on this topic in journals including *Nature Structure Molecular Biology, Molecular Cell Biology, Cancer Research and Nucleic* Acids Research et al. He is a leader of the DNA Repair focus group in the Case Comprehensive Cancer Center.

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