**ROLE OF CHECKPOINT PROTEINS IN THE SUCCESS OF BIR**

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Break-induced replication (BIR) is an important homologous recombination (HR) pathway employed to repair DNA lesions and has been implicated in various chromosomal instabilities, including loss of heterozygosity, translocations, and alternative telomere lengthening. Here, we study the role of checkpoint proteins in DNA repair in yeast *Saccharomyces cerevisiae*. Cell cycle checkpoints are required for the proper progression of the cell cycle. These checkpoint proteins sense problems during the cell cycle and halt progression to allow mistakes to be corrected and the loss of checkpoint controls leads to major defects. RAD9 and RAD24, two important checkpoint proteins play a vital role in arresting the cell cycle upon DNA damage and are also responsible for bringing together the DNA repair machinery. We observed that mutations made in the genes encoding *RAD9* and *RAD24* resulted in the formation of multiple sectors in individual colonies where, every individual sector repaired differently. We analyze the frequency of different repair outcomes associated with BIR in these multi-sectored events. We also report that defective BIR in these checkpoint mutants lead to formation of half-crossovers similar to NRTs reported in mammals, which are implicated in the initiation of cascades of genomic instability characteristic of human cancer cells.

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The work is supported by a grant from NIGMS: 1R01GM084242-01