



DNA Repair Mechanisms in Radiation Induced Genetic Instability

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DESCRIPTION

DNA repair mechanisms are essential for maintaining genomic stability in cells exposed to radiation, as they counteract the diverse forms of damage induced by ionizing and non-ionizing radiation. Radiation exposure leads to a wide array of DNA lesions, including single-strand breaks, double-strand breaks, base modifications, and DNA cross-links. Among these, double-strand breaks are the most deleterious, as they involve the simultaneous disruption of both strands of the DNA helix and pose a significant challenge to accurate repair. The efficiency and fidelity of DNA repair systems play a crucial role in determining whether a cell survives with intact genetic information or accumulates mutations that contribute to carcinogenesis.

Cells utilize several highly coordinated DNA repair pathways to address radiation-induced damage. Homologous recombination is considered a high-fidelity repair mechanism that uses a sister chromatid as a template to accurately restore the original DNA sequence. This process is mainly active during the S and G2 phases of the cell cycle when a sister chromatid is available. In contrast, non-homologous end joining is a faster but error-prone pathway that directly ligates broken DNA ends without the need for a template. Although efficient, this mechanism often results in insertions, deletions, or chromosomal rearrangements, thereby contributing to genomic instability.

In addition to these pathways, base excision repair plays a critical role in correcting small base lesions caused by oxidative stress, which is a common consequence of radiation exposure. This pathway involves the recognition and removal of damaged bases followed by DNA synthesis and ligation. Nucleotide excision repair, on the other hand, is responsible for removing bulky DNA adducts and helix-distorting lesions. The coordinated action of these repair systems is essential for preserving DNA integrity and preventing the accumulation of mutations.

Deficiencies or mutations in genes encoding DNA repair proteins can significantly impair the cell's ability to respond to radiation-induced damage. Such defects are associated with increased sensitivity to radiation and a higher risk of cancer development. Individuals with inherited disorders affecting DNA repair pathways often exhibit elevated levels of genomic instability, highlighting the protective role of these mechanisms. Moreover, acquired defects in repair pathways are frequently observed in cancer cells, further contributing to their genetic heterogeneity.

Radiation exposure can also modulate the expression and activity of DNA repair proteins. Depending on the context, this may lead to adaptive responses that enhance repair capacity or, conversely, to impaired repair due to overwhelming damage. The balance between these outcomes determines the extent of genetic instability and influences the likelihood of malignant transformation. Persistent DNA damage and defective repair can result in chromosomal aberrations, mutations, and altered gene expression patterns.

In the context of cancer therapy, DNA repair mechanisms present both challenges and opportunities. While they protect normal cells from radiation-induced damage, they can also enable tumor cells to survive and develop resistance to radiotherapy. As a result, targeting DNA repair pathways has emerged as a promising strategy to enhance treatment efficacy. Inhibitors of specific repair proteins can sensitize cancer cells to radiation, increasing the extent of DNA damage and promoting cell death.

In conclusion, DNA repair mechanisms are fundamental to the cellular response to radiation-induced damage and play a critical role in maintaining genomic stability. Their proper functioning is essential for preventing mutagenesis and cancer, while their manipulation offers promising avenues for improving cancer therapy and patient outcomes.

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