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Commentary

The Role of Deoxy Ribo Nucleic Acid Repair Pathways in Cancer Development and their Implications for Targeted Therapy

Anthony Glover*

Department of Genetics, University of Edinburgh, Edinburgh, UK

DESCRIPTION

Deoxy Ribo Nucleic Acid (DNA) repair mechanisms are important for maintaining genomic stability, ensuring the integrity of genetic material across cell divisions. When these repair pathways are disrupted, it can lead to mutations, chromosomal aberrations and ultimately cancer. The relationship between DNA repair pathways and cancer development is complex, as both defects in repair mechanisms and enhanced repair abilities can contribute to tumorigenesis. This article explain the role of DNA repair pathways in cancer development and discusses the potential for targeted therapies that exploit these mechanisms.

DNA repair pathways are essential for the correction of various forms of DNA damage, such as those induced by radiation, chemicals and normal cellular processes. There are several key repair mechanisms, including Base Excision Repair (BER), Nucleotide Excision Repair (NER), Mismatch Repair (MMR), Homologous Recombination (HR) and Non-Homologous End Joining (NHEJ). These pathways work together to repair single-strand breaks, double-strand breaks and other forms of genetic damage, ensuring the stability of the genome.

HR and NHEJ are particularly important in the repair of double-strand breaks, which are highly deleterious to cells if left unrepaired. HR is an error-free repair process that uses a sister chromatid as a template, while NHEJ is a more error-prone process that directly ligates broken DNA ends. Both mechanisms are important

Cancer cells are often characterized by genetic instability, which arises when DNA repair pathways fail or become dysregulated. Defects in DNA repair mechanisms lead to an accumulation of mutations, chromosomal aberrations and genomic instability all of which are hallmarks of cancer. For instance, mutations in the Breast Cancer Gene 1(*BRCA1*) and Breast Cancer Gene 1 (*BRCA2*) genes, which are important for homologous recombination, increase the risk of breast, ovarian and other cancers. When these repair pathways are defective, cells are more likely to accumulate mutations, which may drive tumorigenesis.

On the other hand, some cancers have developed mechanisms to enhance DNA repair, allowing them to resist therapies that induce DNA damage. For example, many tumors have upregulated DNA repair proteins, such as Poly (ADP-Ribose) Polymerase (PARP), which enables them to repair single-strand DNA breaks and survive treatments like chemotherapy and radiation. These enhanced repair capabilities can lead to therapy resistance, making it more challenging to treat these cancers effectively.

One of the most well-known examples of this approach is the use of PARP inhibitors in cancers with defective *BRCA1/2* genes. PARP inhibitors block the repair of single-strand breaks in DNA and in cells that already have compromised homologous recombination repair due to BRCA mutations, this results in the accumulation of DNA damage and cell death. Clinical trials have demonstrated the effectiveness of PARP inhibitors, such as olaparib, in treating ovarian, breast and prostate cancers with BRCA mutations.

In addition to targeting PARP, other therapeutic strategies aim to exploit defects in specific DNA repair pathways. For example, inhibitors of DNA-Dependent Protein Kinase (DNA-PK), which is involved in Non-homologous end joining (NHEJ), are being explored as potential treatments for cancers that exhibit defects in HR. By targeting the repair pathways that are significant for the survival of cancer cells, researchers hope to develop therapies that are both highly selective and effective. Cancer cells often acquire resistance to treatments that induce DNA damage, such as chemotherapy and radiation, by upregulating DNA repair mechanisms. Inhibiting these repair pathways can explain the effectiveness of these treatments.

CONCLUSION

DNA repair pathways are essential for maintaining genomic stability and their dysfunction plays an important role in cancer

Correspondence to: Anthony Glover, Department of Genetics, University of Edinburgh, Edinburgh, UK, E-mail: anthony@glover.uk

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development. Understanding how these pathways contribute to tumorigenesis has created the procedure for targeted therapies that exploit DNA repair defects in cancer cells. From PARP inhibitors to novel DNA repair pathway inhibitors, these therapies offer the potential to provide more effective and personalized treatments for cancer patients. As research into DNA repair mechanisms continues, new therapeutic strategies are likely to emerge, offering hope for better outcomes in cancer treatment. For instance, combining DNA repair inhibitors with traditional chemotherapy or radiation therapy may prevent cancer cells from repairing the damage caused by these treatments, increasing the likelihood of tumor cell death.