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The Role of DNA Repair Genes in Thyroid Cancer

Jim Kau*

Department of Biochemistry, Sejong University, South Korea

INTRODUCTION

Thyroid cancer, the most common endocrine malignancy, has been rising in incidence over recent decades. While the prognosis for most thyroid cancers is generally favorable, understanding the molecular underpinnings of the disease remains crucial for improving diagnosis, treatment, and prevention strategies. Among the various genetic factors involved, DNA repair genes play a significant role in the development and progression of thyroid cancer. These genes are essential for maintaining genomic stability by correcting DNA damage, and their dysfunction can lead to mutations that contribute to carcinogenesis. This article explores the impact of DNA repair genes on thyroid cancer, highlighting key pathways and their clinical implications.

DESCRIPTION

XRCC1 and XRCC3, genes are critical for the repair of single-strand and double-strand breaks, respectively. XRCC1 interacts with DNA ligase III, poly (ADP-ribose) polymerase, and DNA polymerase beta to facilitate BER. XRCC3, a member of the RAD51 family, is involved in HR. Polymorphisms in these genes have been linked to an increased susceptibility to thyroid cancer.

MLH1 and MSH2, MMR genes are crucial for correcting base-base mismatches and insertion-deletion loops that occur during DNA replication. Mutations or reduced expression of MLH1 and MSH2 have been observed in various cancers, including thyroid cancer. Defective MMR leads to microsatellite instability, a condition characterized by widespread mutations throughout the genome. MSI has been reported in a subset of thyroid cancers, indicating the involvement of MMR deficiencies in thyroid tumorigenesis. BRCA1 and BRCA2, well-known genes, primarily associated with breast and ovarian cancers, also play roles in HR repair of double-strand breaks. Mutations in BRCA1 and BRCA2 can impair HR, leading to genomic instability and increased cancer risk. The TP53 gene, encoding the tumor suppressor protein p53, is integral to multiple DNA repair processes. P53 can activate the transcription of genes involved in BER, NER, and HR. Mutations in TP53 are relatively rare in thyroid cancer compared to other malignancies, but when present, they are often associated with aggressive tumor behavior and poor prognosis. P53 mutations disrupt its role in DNA repair and cell cycle regulation, facilitating thyroid cancer progression.

Understanding the role of DNA repair genes in thyroid cancer has significant clinical implications. First, genetic testing for polymorphisms and mutations in DNA repair genes can enhance risk assessment and early detection strategies. Individuals with inherited mutations in key repair genes may benefit from more rigorous screening protocols. Second, targeting DNA repair pathways offers promising therapeutic avenues. PARP inhibitors, which have shown efficacy in BRCA-mutated breast and ovarian cancers, are being explored for their potential in treating thyroid cancers with similar DNA repair deficiencies. By inhibiting PARP, these drugs prevent the repair of single-strand breaks, leading to the accumulation of double-strand breaks and cancer cell death, particularly in cells already compromised in HR repair. Additionally, understanding DNA repair gene status can inform the use of radiation therapy. Radiation induces DNA damage, and tumors deficient in DNA repair may be more susceptible to radiation-induced cell death. Therefore, assessing the DNA repair capacity of thyroid tumors could help tailor radiation therapy to maximize efficacy while minimizing adverse effects.

CONCLUSION

Continued research into the role of DNA repair genes in thyroid cancer is essential for advancing our understanding and improving patient outcomes. Large-scale genomic studies and next-generation sequencing technologies are uncovering novel mutations and polymorphisms in DNA repair genes, providing new insights into thyroid cancer biology. DNA repair genes play a crucial role in maintaining genomic stability and preventing cancer. In thyroid cancer, mutations and polymorphisms in these genes contribute to carcinogenesis and influence disease progression and treatment response. Advances in our understanding of DNA repair mechanisms offer promising avenues for improving risk assessment, early detection, and therapeutic strategies in thyroid cancer. By continuing to unravel the complexities of DNA repair in thyroid cancer, we move closer to personalized and effective treatments that can enhance patient outcomes and quality of life.

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Corresponding author Jim Kau, Department of Biochemistry, Sejong University, South Korea, E-mail: jim@la.re.kr

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