OPEN ACCESS Freely available online

ISSN: 2161-1041
Hereditary Genetics: Current Research

Opinion Article

Targeting DNA Damage Pathways as Therapeutic Strategies in Rheumatoid Arthritis

William Becker*

Department of Molecular and Cellular Biology, Harvard University, Cambridge, Massachusetts, United States of America

DESCRIPTION

Rheumatoid Arthritis (RA) is a chronic autoimmune illness that causes inflammation and joint destruction. DNA damage refers to any alteration in the DNA sequence that disrupts its normal structure and function. It can result from various sources, including environmental factors, oxidative stress, and the body's own immune response. Fortunately, cells possess intricate DNA repair mechanisms that can recognize and correct these damaged areas. However, in conditions like rheumatoid arthritis, the balance between DNA damage and repair is disrupted, leading to the accumulation of DNA lesions. Multiple mechanisms contribute to DNA damage in rheumatoid arthritis. Firstly, chronic inflammation in the synovial tissue releases various pro-inflammatory mediators, such as cytokines and Reactive Oxygen Species (ROS). These inflammatory molecules can directly induce DNA damage by causing oxidative stress and DNA strand breaks. Additionally, immune cells, particularly neutrophils, produce high levels of ROS and release DNA-binding proteins, forming extracellular traps that can damage surrounding tissues and trigger an autoimmune response. DNA damage in rheumatoid arthritis has significant clinical implications. Firstly, it can lead to the production of autoantibodies, such as Rheumatoid Factor (RF) and Anti-Citrullinated Protein Antibodies (ACPA), which are diagnostic markers of RA. These autoantibodies recognize and target specific proteins that have undergone post-translational modifications due to DNA damage. Furthermore, DNA damage can activate immune cells, leading to a perpetuation of inflammation and joint destruction in RA. Accumulating evidence suggests that DNA damage contributes to joint destruction in rheumatoid arthritis. DNA lesions within the synovial tissue can activate inflammatory pathways, leading to the production of pro-inflammatory cytokines and Matrix Metalloproteinases (MMPs). These enzymes degrade the extracellular matrix and promote the destruction of cartilage and bone, ultimately causing irreversible damage to the affected joints.

Understanding the role of DNA damage in rheumatoid arthritis opens up potential therapeutic strategies. Firstly, targeting the underlying mechanisms of DNA damage, such as oxidative stress and inflammation, may help mitigate the disease process.

Antioxidant agents, including vitamins C and E, have shown potential results in reducing DNA damage and inflammation in experimental models of RA. Moreover, modulating the activity of DNA repair enzymes, such as Poly (ADP-ribose) Polymerase (PARP), may enhance DNA repair capacity and protect against joint damage in RA.

In recent years, personalized medicine approaches have gained attention in rheumatoid arthritis management. Evaluating DNA damage biomarkers, such as levels of DNA strand breaks, oxidative DNA damage markers, and DNA repair gene polymorphisms, may help identify individuals at higher risk for severe disease progression. This knowledge can guide treatment decisions, allowing for customized interventions and closer monitoring of disease activity and joint damage.

CONCLUSION

DNA damage plays a significant role in the pathogenesis of rheumatoid arthritis, contributing to inflammation, autoimmunity, and joint destruction. Understanding the underlying mechanisms of DNA damage and impaired repair mechanisms provides insights into potential therapeutic targets and personalized medicine approaches. Further research is needed to solve the complex interplay between DNA damage and rheumatoid arthritis and develop innovative strategies to halt disease progression, preserve joint function, and improve the quality of life for individuals living with RA.

Correspondence to: William Becker, Department of Molecular and Cellular Biology, Harvard University, Cambridge, Massachusetts, United States of America, E-mail: wlambeck@icloud.com

Received: 02-Jun-2023, Manuscript No. HGCR-23-22087; **Editor assigned:** 05-Jun-2023, PreQC No. HGCR-23-22087 (PQ); **Reviewed:** 19-Jun-2023, QC No. HGCR-23-22087; **Revised:** 26-Jun-2023, Manuscript No. HGCR-23-22087 (R); **Published:** 03-Jul-2023, DOI: 10.35248/2161-1041.23.12.242

Citation: Becker W (2023) Targeting DNA Damage Pathways as Therapeutic Strategies in Rheumatoid Arthritis. Hereditary Genet. 12:242.