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Epigenetic Alterations in Autoimmune Disease Pathogenesis.

Sarah L. Martinez*

Department of Immunology, Biomedical Research Institute, Biomedical University of Health Sciences, United States

***Correspondence to:** Sarah L. Martinez *, Department of Immunology, Biomedical Research Institute, Biomedical University of Health Sciences, United States,. Email: sarah.martinez@biomeduni.edu

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Introduction

Autoimmune diseases arise from an aberrant immune response in which the body's immune system attacks its own tissues. Traditionally, genetic susceptibility and environmental triggers were considered the primary drivers of autoimmune pathogenesis. However, emerging evidence indicates that epigenetic alterations—heritable changes in gene expression that do not involve modifications to the DNA sequence—play a pivotal role in disease onset and progression [1, 2, 3, 4, 5].

Epigenetic mechanisms, including DNA methylation, histone modifications, and non-coding RNAs, modulate immune cell differentiation, cytokine production, and tolerance to self-antigens. Disruption in these processes can lead to aberrant activation of autoreactive T and B cells, perpetuating chronic inflammation. Environmental factors such as diet, infections, and exposure to chemicals can influence these epigenetic marks, thereby bridging genetic predisposition and clinical disease.

Several autoimmune conditions, including systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and multiple sclerosis (MS), display distinctive epigenetic signatures. For example, hypomethylation of genes encoding pro-inflammatory cytokines has been linked to heightened immune responses in SLE. Similarly, histone acetylation changes can alter chromatin accessibility, facilitating pathogenic gene transcription in RA. Understanding these alterations not only sheds light on disease pathogenesis but also opens avenues for targeted epigenetic therapies such as histone deacetylase inhibitors and DNA methyltransferase modulators.

Conclusion

Epigenetic alterations serve as a critical interface between genetic susceptibility and environmental influences in autoimmune disease pathogenesis. These modifications can act as both triggers and perpetuators of autoimmunity by reshaping immune cell function and inflammatory pathways. As research advances, mapping disease-specific epigenetic patterns will enhance diagnostic precision and pave the way for personalized therapeutic interventions. Targeting epigenetic regulators holds promise for reversing pathogenic immune responses without broadly suppressing immunity, offering hope for more effective and safer treatments for autoimmune disorders.

References

1. Ballestar, E. (2017). Epigenetics lessons from twins: Prospects for autoimmune disease. *Clinical Reviews in Allergy & Immunology, 52*(1), 42–56.
2. Kolarz, B., & Rudnicka, W. (2020). Epigenetic mechanisms in rheumatoid arthritis. *Reumatologia, 58*(1), 1–8.
3. Sawalha, A. H., & Jeffries, M. (2019). Defective DNA methylation and CD70 overexpression in lupus T cells. *Journal of Immunology, 182*(9), 5676–5683.
4. Zhang, P., & Su, Y. (2021). Histone modifications in autoimmune diseases: Friend or foe? *Frontiers in Immunology, 12*, 667951.
5. Hedrich, C. M., & Tsokos, G. C. (2011). Epigenetic mechanisms in systemic lupus erythematosus and other autoimmune diseases. *Trends in Molecular Medicine, 17*(12), 714–724.

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