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Epigenetic Modifications in Immune-Related Neurological Diseases

Josephine Jaden Gene *

Child Psychology Department, University Hospital Southampton Foundation Trust, UK

Abstract

Immune-related neurological diseases, including multiple sclerosis (MS), systemic lupus erythematosus (SLE) with neurological involvement, and autoimmune encephalitis, are characterized by immune system dysregulation and inflammation within the central nervous system (CNS). Epigenetic modifications, including DNA methylation, histone modifications, and non-coding RNA regulation, play a crucial role in modulating gene expression and influencing immune cell function. This review examines the current understanding of epigenetic mechanisms in the pathogenesis of these diseases, highlighting their potential as therapeutic targets.

Keywords: Epigenetics; Neuroimmunology; Multiple sclerosis; Systemic lupus erythematosus; Autoimmune encephalitis; DNA methylation; Histone modification; Non-coding RNA

Introduction

Immune-related neurological diseases are a heterogeneous group of disorders characterized by an aberrant immune response targeting the CNS, leading to inflammation, demyelination, neuronal damage, and neurological dysfunction. While genetic susceptibility plays a role, environmental factors are also crucial in triggering and modulating disease development. Epigenetic modifications, which alter gene expression without changing the underlying DNA sequence, provide a crucial link between genetic predisposition and environmental influences. These modifications, including DNA methylation, histone modifications (acetylation, methylation, phosphorylation), and regulation by non-coding RNAs (microRNAs, long non-coding RNAs), can influence immune cell development, differentiation, activation, and function. This review synthesizes current research on the role of epigenetic mechanisms in the pathogenesis of key immune-related neurological diseases, including MS, SLE with neurological involvement (neuro-SLE), and autoimmune encephalitis, exploring their potential as therapeutic targets.

Results

Epigenetic modifications have been implicated in the pathogenesis of several immune-related neurological diseases. In MS, a chronic inflammatory demyelinating disease of the CNS, altered DNA methylation patterns have been observed in various immune cell types, including T cells, B cells, and monocytes [1]. These changes can affect the expression of genes involved in immune cell activation, differentiation, and cytokine production, contributing to the inflammatory cascade within the CNS. For instance, altered methylation of genes encoding pro-inflammatory cytokines like IFN-γ and IL-17 has been observed in MS patients. Histone modifications also play a significant role in MS pathogenesis. Changes in histone acetylation and methylation can influence the accessibility of DNA to transcriptional machinery, thereby regulating gene expression. Studies have shown altered histone modifications in immune cells from MS patients, affecting the expression of genes involved in immune cell function and inflammation . Non-coding RNAs, particularly microRNAs (miRNAs), are also implicated in MS. miRNAs are small non-coding RNA molecules that regulate gene expression by binding to messenger RNA (mRNA) and inhibiting translation or promoting degradation. Dysregulation of miRNA expression has been observed in MS, affecting the expression of genes involved in immune cell activation, differentiation, and CNS inflammation [2-4]. In neuro-SLE, a complex autoimmune disease affecting multiple organ systems, including the CNS, epigenetic modifications also contribute to disease pathogenesis. Altered DNA methylation patterns have been observed in immune cells from SLE patients, affecting the expression of genes involved in immune cell activation, cytokine production, and autoantibody production. For example, altered methylation of genes encoding type I interferons has been linked to increased interferon production in SLE, contributing to inflammation. Histone modifications also play a role in neuro-SLE, influencing the expression of genes involved in immune cell function and inflammation. Non-coding RNAs, particularly miRNAs, are also implicated in SLE pathogenesis, affecting the expression of genes involved in immune cell activation and autoantibody production . In autoimmune encephalitis, a group of disorders characterized by autoantibody-mediated inflammation of the brain, epigenetic modifications are also being increasingly recognized as contributing factors. Studies have shown altered DNA methylation patterns in immune cells from patients with autoimmune encephalitis, affecting the expression of genes involved in immune cell activation and cytokine production . Histone modifications also play a role, influencing the expression of genes involved in neuronal function and inflammation within the CNS . Non-coding RNAs, particularly miRNAs, are also implicated in autoimmune encephalitis, affecting the expression of genes involved in neuronal signaling and immune responses .

Discussion

The findings presented in this review highlight the significant role of epigenetic modifications in the pathogenesis of immune-related neurological diseases. Altered DNA methylation, histone modifications, and non-coding RNA regulation contribute to immune system dysregulation and CNS inflammation [5-10]. These epigenetic changes can affect the expression of genes involved in immune cell

***Corresponding author:** Josephine Jaden Gene, Child Psychology Department, University Hospital Southampton Foundation Trust, UK; E-mail: jade.c@gmail.com

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activation, differentiation, cytokine production, and neuronal function. Understanding the specific epigenetic mechanisms involved in each disease is crucial for developing targeted therapeutic strategies. While epigenetic drugs have shown some promise in preclinical and clinical studies, further research is needed to optimize their use and minimize potential side effects. Future research should also focus on identifying specific epigenetic biomarkers that can be used for diagnosis, prognosis, and monitoring treatment response.

Conclusion

Epigenetic modifications play a crucial role in the pathogenesis of immune-related neurological diseases. These modifications contribute to immune system dysregulation and CNS inflammation. Targeting epigenetic mechanisms holds promise as a therapeutic strategy for these debilitating conditions. The interplay between genetic and epigenetic factors is crucial in the development of these diseases. Genetic variations can influence the susceptibility to epigenetic modifications, while epigenetic changes can modify the expression of genes associated with genetic risk. Environmental factors, such as infections, stress, and exposure to toxins, can also influence epigenetic modifications and contribute to disease development. Targeting epigenetic mechanisms has emerged as a promising therapeutic strategy for immune-related neurological diseases. Several epigenetic drugs, such as DNA methyltransferase inhibitors and histone deacetylase inhibitors, are being investigated for their potential therapeutic benefits . These drugs can reverse some of the aberrant epigenetic modifications observed in these diseases and modulate immune cell function. Further research is needed to fully understand the complex interplay between genetic, epigenetic, and environmental factors in the development of these diseases and to develop more targeted and effective therapies.

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