Perspective

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Epigenetic Modifications and Their Role in Neurodegenerative Disorders: A Genome-Wide Perspective

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Introduction

Neurodegenerative disorders such as Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), and amyotrophic lateral sclerosis (ALS) are characterized by the progressive loss of neuronal structure and function. Despite extensive research, the precise molecular mechanisms underlying these disorders remain only partially understood. Recent advances in epigenomics have revealed that epigenetic modifications heritable changes in gene expression that do not involve alterations in the DNA sequence play a pivotal role in the onset and progression of neurodegeneration. Epigenetic mechanisms, including DNA methylation, histone modification, and non-coding RNA regulation, dynamically influence gene activity and neural plasticity throughout life. Environmental factors such as diet, stress, and exposure to toxins can reshape the epigenetic landscape, leading to aberrant gene expression patterns implicated in neuronal dysfunction [1].

Description

DNA methylation, one of the most extensively studied epigenetic marks, involves the addition of a methyl group to cytosine residues, typically leading to gene silencing. In neurodegenerative disorders, genome-wide methylation studies have revealed altered methylation patterns in genes related to synaptic signaling, inflammation, and protein degradation. For example, hypermethylation of the ANK1 gene and hypomethylation of APP have been associated with Alzheimer's disease, suggesting their potential roles in disease progression. Similarly, in Parkinson's disease, aberrant methylation in the SNCA gene, which encodes α-synuclein, contributes to abnormal protein aggregation and neuronal death [2].

Histone modifications, including acetylation, methylation, an phosphorylation, also play crucial roles in chromatin remodeling and gene expression. Dysregulation of histone-modifying enzymes, such as Histone Deacetylases (HDACs), has been observed in multiple neurodegenerative diseases. HDAC inhibitors, which promote histone acetylation and enhance gene expression, have shown neuroprotective effects in Experimental models, highlighting the therapeutic potential of targeting epigenetic mechanisms. Beyond DNA and histone modifications, non-coding RNAs (ncRNAs) particularly microRNAs (miRNAs) and long non-coding RNAs (lncRNAs) serve as critical regulators of gene expression in neural tissues. Genome-wide transcriptomic and epigenomic analyses have identified dysregulated miRNAs that control key pathways such as apoptosis, oxidative stress response, and mitochondrial function [3].

Similarly, aberrant expression of lncRNAs has been linked to disrupted neuronal differentiation, synaptic plasticity, and neuroinflammatory responses. These ncRNAs often interact with chromatin-modifying complexes, amplifying their impact on epigenetic regulation.

For instance, altered expression of miR-34a and miR-132 has been linked to neuronal apoptosis in Alzheimer's disease, while miR-155 is associated with neuroinflammation in Parkinson's disease. Integrating multi-omics data, including DNA methylation, histone modifications, and RNA expression, provides a comprehensive view of the molecular architecture underlying neurodegeneration [4,5].

Conclusion

In conclusion, epigenetic modifications represent a dynamic interface between the genome and the environment, profoundly influencing neuronal health and disease. A genome-wide perspective on these modifications has illuminated novel pathways and molecular interactions contributing to neurodegeneration. The reversible nature of epigenetic changes makes them promising therapeutic targets for drug development, offering hope for interventions that can restore normal gene expression and halt disease progression. However, translating epigenetic discoveries into clinical applications requires further validation through large-scale studies and an ethical framework for genome-based therapies.

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None

Conflict of Interest

None

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