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# Epigenetic Modifications in Essential Hypertension: Emerging Insights and Clinical Implications

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## Introduction

Essential hypertension, accounting for approximately 90–95% of all hypertension cases, is traditionally viewed as a multifactorial condition influenced by genetic predisposition, environmental exposures and lifestyle behaviors. However, the genetic heritability of blood pressure only partially explains individual variability, leading to growing interest in epigenetic modifications as a missing link. Epigenetics refers to heritable changes in gene expression that do not involve alterations in DNA sequence and it encompasses mechanisms such as DNA methylation, histone modifications and non-coding RNA activity. These modifications are influenced by diet, stress, physical activity, toxins and aging—factors well known to contribute to hypertension. In recent years, extensive research has identified epigenetic patterns associated with hypertension pathophysiology, offering fresh insights into its origins and progression. This article explores the emerging understanding of epigenetic influences in essential hypertension and discusses their potential for improving risk prediction, therapeutic strategies and long-term clinical outcomes [1].

## Description

Among epigenetic mechanisms, DNA methylation has been the most extensively studied in the context of hypertension. This process typically involves the addition of a methyl group to the cytosine residue in CpG dinucleotides, often leading to transcriptional silencing of genes. Several studies have demonstrated that altered methylation patterns in genes involved in vascular tone, sodium balance, inflammation and hormonal regulation are associated with elevated blood pressure. For instance, hypermethylation of the NOS3 gene, encoding endothelial nitric oxide synthase, has been linked to impaired vasodilation and increased vascular resistance. Similarly, aberrant methylation of the ACE, AGTR1 and ADD1 genes has been shown to modulate components of the renin-angiotensin-aldosterone system (RAAS), promoting sodium retention and vasoconstriction. These methylation signatures are not only biomarkers of disease presence but also indicators of cumulative environmental exposure, offering a dynamic framework for understanding hypertension beyond static genomic risk. As such, DNA methylation profiles may serve as early predictors of hypertension, enabling timely preventive measures [2-3].

In addition to DNA methylation, histone modifications play a pivotal role in epigenetic regulation of gene expression in hypertension. Histones are proteins around which DNA is wound and chemical modifications such as acetylation, methylation and phosphorylation can influence chromatin structure and accessibility. Acetylation of histone H3 lysine 9 (H3K9ac), for example, is associated with relaxed chromatin and active gene transcription. In hypertensive models, reduced acetylation levels of anti-inflammatory genes have been reported, leading to vascular dysfunction and increased blood pressure. Conversely, histone methylation, depending on the residue and context, can either activate or repress gene transcription. Abnormal histone methylation has been linked to oxidative stress and inflammation—two central components of hypertension pathology. Recent evidence also indicates that environmental stressors, such as high-salt diets or psychosocial stress, can induce persistent histone modifications in key cardiovascular genes. These findings underscore the plasticity of the epigenome and its responsiveness to environmental triggers, supporting the development of histone-targeting therapeutics for blood pressure control [4].

A growing body of evidence highlights the involvement of non-coding RNAs, particularly microRNAs (miRNAs) and long non-coding RNAs (lncRNAs), in the regulation of gene expression relevant to hypertension. MicroRNAs are short, ~22-nucleotide RNA sequences that suppress gene translation by binding to target mRNA. Several miRNAs—such as miR-155, miR-21 and miR-126—have been implicated in vascular remodeling, endothelial dysfunction and immune activation, all of which contribute to elevated blood pressure. For instance, miR-155 downregulates the angiotensin II type 1 receptor, acting as a negative regulator of vasoconstriction. On the other hand, aberrant overexpression of miR-21 has been associated with fibrosis and vascular stiffness in hypertensive patients. LncRNAs, though less studied, are emerging as crucial regulators of transcription and chromatin organization. Certain lncRNAs such as GAS5 and H19 have shown involvement in smooth muscle cell proliferation and cardiac hypertrophy. The utility of circulating miRNAs and lncRNAs as non-invasive biomarkers for hypertension diagnosis and prognosis is also gaining traction. Collectively, non-coding RNAs present new targets for therapeutic intervention and potential tools for personalized hypertension care [5].

## Conclusion

Despite substantial progress, many questions remain regarding the causal versus correlative nature of epigenetic changes in hypertension. Longitudinal studies and epigenome-wide association studies (EWAS) across diverse populations are needed to validate candidate markers and elucidate temporal relationships between exposures, epigenetic modifications and blood pressure changes. Further, integrating epigenomic data with other 'omics' technologies—such as genomics, transcriptomics, proteomics and metabolomics—will provide a systems-level understanding of hypertension pathogenesis. There is also an urgent need for standardization of methodologies in epigenetic research to ensure reproducibility and clinical applicability.

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## Conflict of Interest

None.

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