Article type: Short Communication

Home Page URL: https://www.alliedacademies.org/journal-pharmacology-therapeutic-research/

Epigenetic modifications in aging: Exploring DNA methylation and histone acetylation as therapeutic targets.

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Received: 1-Jan-2025, Manuscript No. aajptr-25-168702; **Editor assigned:** 4-Jan-2025, PreQC No. aajptr-25-168702 (PQ); **Reviewed:** 18-Jan-2025, QC No. aajptr-25-168702; **Revised:** 25-Jan-2025, Manuscript No. aajptr-25-168702 (R); **Published:** 30-Jan-2025, DOI: 10.35841/aajptr-9.1.187

Introduction

Aging is a complex biological process influenced by genetic, environmental, and epigenetic factors. Among these, epigenetic modifications—heritable yet reversible changes in gene expression that do not alter the DNA sequence—play a significant role. Two of the most studied epigenetic mechanisms in the context of aging are DNA methylation and histone acetylation, both of which modulate chromatin structure and gene activity. Understanding these processes not only sheds light on the biology of aging but also opens avenues for novel therapeutic strategies [1].

DNA methylation typically involves the addition of a methyl group to the 5th carbon of cytosine residues, usually in the context of CpG dinucleotides. In young, healthy individuals, DNA methylation patterns are tightly regulated and maintain genomic stability. However, with age, these patterns become dysregulated—characterized by global hypomethylation and localized hypermethylation at gene promoters. Global hypomethylation can lead to chromosomal instability and aberrant gene activation, while hypermethylation of tumor suppressor or DNA repair genes contributes to age-related pathologies, including cancer [2].

The concept of an "epigenetic clock" has emerged from studies analyzing DNA methylation changes at specific loci to estimate biological age. Horvath's epigenetic clock, for example, uses DNA methylation data from multiple tissues to predict chronological age with remarkable accuracy. Deviations from expected methylation patterns may indicate accelerated aging and increased disease risk, making the epigenetic clock a potential biomarker for healthspan and lifespan [3].

Histone acetylation, on the other hand, is associated with transcriptional activation. Acetylation of histone tails, mediated by histone acetyltransferases (HATs), reduces the positive charge of histones, weakening their interaction with negatively charged DNA and allowing transcription factors easier access. Conversely, histone deacetylases (HDACs) remove these acetyl groups, resulting in tighter chromatin and gene repression. Age-related decline in histone acetylation has been linked to reduced expression of genes involved in DNA repair, antioxidant defense, and cellular homeostasis [4].

One of the key findings in aging research is that changes in histone acetylation are not uniform. Certain genes may become hyperacetylated and abnormally active, while others may lose acetylation and become silenced. This imbalance contributes to cellular senescence and the loss of tissue function. Notably, caloric restriction—one of the few interventions shown to extend lifespan across species—has been associated with preserved histone acetylation patterns, particularly through the activation of sirtuins, a family of NAD+-dependent HDACs [5].

Conclusion

In conclusion, epigenetic modifications—particularly DNA methylation and histone acetylation—are central to the aging process and serve as valuable biomarkers and therapeutic targets. Their dynamic and reversible nature provides hope for interventions aimed at extending healthspan and mitigating age-related diseases. As our understanding deepens, the manipulation of the epigenome may become a cornerstone in the fight against aging and its associated conditions.

Citation: Ricci K. Epigenetic modifications in aging: Exploring DNA methylation and histone acetylation as therapeutic targets. J Pharmacol Ther Res. 2025;9(1):187.

References

1. Cha Y, Kim HP, Lim Y, et al. FGFR2 amplification is predictive of sensitivity to regorafenib in gastric and colorectal cancers in vitro. Mol Oncol. 2018;12:993-1003.
2. Diehl F, Schmidt K, Choti MA, et al. Circulating mutant DNA to assess tumor dynamics. Nat Med. 2008;14(9):985-90.
3. Ding Y, Li W, Wang K, et al. Perspectives of the application of liquid biopsy in colorectal cancer. Biomed Res Int. 2020;6843180.
4. Draht MXG, Goudkade D, Koch A, et al. Prognostic DNA methylation markers for sporadic colorectal cancer: A systematic review. Clin Epigenetics. 2018;10:35.
5. Drula R, Ott LF, Berindan-Neagoe I, et al. MicroRNAs from liquid biopsy derived extracellular vesicles: Recent advances in detection and characterization methods. Cancer. 2020;12(8):2009.

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