



Histone Modifications are used in the Gene Functions through Rheumatoid Arthritis

Lucy Aikin*

Department of Clinical Epigenetics, University of Bath, United Kingdom

INTRODUCTION

Lysine acetylation is a widespread and versatile post-translational modification of proteins. Lysine acetyltransferase and lysine deacetylase catalyze the addition and removal of acetyl groups, respectively, on both histone and non-histone targets. This review describes some features of acetylation and deacetylation. This includes target diversity, rapid turnover, excellent sensitivity to the concentrations of the cofactors acetyl-CoA, acyl-CoA, and NAD⁺, and close interactions with metabolism. Histone acetylation and non-histone protein acetylation affect a variety of cellular and physiological processes, including transcription, phase separation, autophagy, mitosis, differentiation and neuronal function. In turn, lysine acetyltransferase and lysine deacetylase activities are regulated by metabolic state, diet, and certain small molecules. Recently, it has also been shown that histone acetylation mediates cellular memory. These functions allow acetylation to integrate cellular state with transcriptional output and cell fate decisions.

DESCRIPTION

Closely related and mutually regulated metabolic rewiring and epigenetic remodeling is one of the well-known hallmarks of cancer. Recent evidence suggests that many metabolites function as substrates or cofactors for chromatin-modifying enzymes as a result of translocation or spatial localization of the enzyme or metabolite. Various metabolic alterations and epigenetic modifications have also been reported to promote immune escape or impede immune surveillance in certain situations, and play important roles in tumor progression. This review focuses on how metabolic reprogramming of tumor and immune cells remodels epigenetic changes, particularly histone protein and DNA acetylation and methylation. We also describe other prominent metabolic modifications such as succinylation, hydroxybutyrylation and lactylation, updating current progress in therapeutic prospects based on metabolic and

epigenetic modifications in cancer.

Epigenetic changes associated with histone modifications play important roles in the development and maintenance of phenotypes in various types of cancer. In contrast to direct mutations of major DNA sequences, these changes are reversible, making the development of inhibitors of post-translational histone-modifying enzymes one of the most promising strategies for anticancer drug development. To date, various histone modifications have been found to play important roles in regulating chromatin state, gene expression, and other nuclear events. This review examines key features of the most common and studied epigenetic histone modifications that have proven roles in the pathogenesis of a wide range of malignant neoplasms. HDACs and the HMT/HDMT family are associated with the development of oncological pathologies. Data on the association of histone modifications with specific types of cancer are presented and discussed. Particular attention is paid to examining different strategies for the development of epigenetic inhibitors.

CONCLUSION

Rheumatoid arthritis (RA) is an autoimmune disease. The etiology of RA remains undetermined and the etiology is complex. Ideal therapeutic agents and treatment strategies are still lacking. Epigenetic modifications have a profound impact on the vital state of the body by influencing and regulating gene function and properties through mechanisms such as DNA methylation, histone modifications, chromosomal remodeling, and RNAi. Recently, the phenomenon of epigenetic alterations in RA has attracted increasing research interest. The application of epigenetically modified methods is at the forefront of research on RA pathogenesis. This review article focuses on research on the pathogenesis of RA based on epigenetic modifications over the past five years, thereby proposing new methods and strategies for the diagnosis and treatment of RA.

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Corresponding author Lucy Aikin, Department of Clinical Epigenetics, University of Bath, United Kingdom, E-mail: lucyaikin231@gmail.com

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