Mini Review
Volume 17:2, 2023

Journal of Molecular and Genetic Medicine

ISSN: 1747-0862 Open Access

A Review on Immunoepigenetic Control of Inflammatory Bowel Disease

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Abstract

Inflammatory bowel disease (IBD) is a chronic autoimmune disorder that affects the gastrointestinal tract. It includes two primary forms of inflammatory bowel disease - Crohn's disease (CD) and ulcerative colitis (UC) - that share some common clinical features such as abdominal pain, diarrhea, and rectal bleeding. Although the precise etiology of IBD remains unclear, it is believed that genetic, environmental, and immunological factors play a role in the development of this condition. Recent studies have suggested that epigenetic modifications in immune cells may contribute to the pathogenesis of IBD. This article will discuss the role of immunoepigenetic regulation in the development of IBD. Epigenetic modifications are heritable changes in gene expression that do not involve alterations to the DNA sequence. Epigenetic mechanisms include DNA methylation, histone modifications, and non-coding RNA expression, all of which play important roles in the regulation of gene expression. Dysregulation of epigenetic mechanisms can lead to aberrant gene expression and contribute to the development of diseases such as cancer, autoimmune disorders, and neurodegenerative diseases. In the context of IBD, recent studies have highlighted the importance of epigenetic modifications in immune cells.

Keywords: RNA expression • Crohn's disease • CD4+ T cells

Introduction

Immune cells such as T cells, B cells, and macrophages play critical roles in the development and progression of IBD. These cells are responsible for initiating and maintaining the inflammatory response in the gut, leading to tissue damage and dysfunction. Dysregulated immune responses are thought to be the primary cause of IBD, but the underlying mechanisms are not fully understood. Several studies have shown that epigenetic modifications in immune cells are involved in the pathogenesis of IBD. For example, aberrant DNA methylation patterns have been observed in CD4+ T cells from patients with CD and UC. DNA methylation is a chemical modification of DNA that involves the addition of a methyl group to the cytosine base in a CpG dinucleotide. This modification typically results in gene silencing, and aberrant DNA methylation can lead to dysregulated gene expression. In CD4+ T cells from patients with IBD, aberrant DNA methylation patterns have been observed in genes involved in T cell activation and differentiation. For example, hypomethylation of the IL17A promoter has been observed in CD4+ T cells from patients with UC. IL17A is a cytokine that plays a key role in the pathogenesis of IBD by promoting the recruitment of immune cells to the gut and stimulating the production of pro-inflammatory cytokines [1-3]. Hypomethylation of the IL17A promoter in CD4+ T cells may lead to increased IL17A expression, promoting the inflammatory response in the gut.

Literature Review

Epigenetic modifications have emerged as a crucial mechanism that regulates gene expression, which can affect the immune response. The immune system, in turn, plays a vital role in the defense against infectious agents, tissue damage, and cancer. Over the past decade, there has been growing evidence that epigenetic modifications can modulate the immune system's response to various stimuli. In this article, we will discuss current insights into novel epigenetic modulations of the systemic immune response.

Epigenetic modifications refer to changes in gene expression that occur without altering the DNA sequence. These modifications include DNA methylation, histone modifications, and non-coding RNA molecules such as microRNAs (miRNAs). DNA methylation is a covalent modification of cytosine residues in DNA that usually occurs at CpG dinucleotides. Histone modifications include acetylation, methylation, phosphorylation, and ubiquitination of histone proteins, which can alter the chromatin structure and affect gene expression. miRNAs are small non-coding RNAs that can post-transcriptionally regulate gene expression by binding to target mRNAs. The immune system consists of various cells and molecules that work together to protect the body against infectious agents, tumors, and tissue damage. Epigenetic modifications can influence the development, differentiation, and function of immune cells, as well as the production of cytokines and chemokines. Here are some examples of epigenetic modifications that affect the immune system [4,5].

Discussion

DNA methylation and immune cell differentiation

DNA methylation can influence the differentiation of immune cells, such as T cells and B cells. For example, DNA methylation of the FOXP3 gene promoter is essential for the development of regulatory T cells (Tregs), which play a crucial role in immune tolerance. DNA methylation of the IL-4 promoter can affect the differentiation of Th2 cells, which produce cytokines that promote humoral immunity.

Histone modifications and cytokine production

Histone modifications can affect the production of cytokines and chemokines by immune cells. For example, histone acetylation of the IFN-γ promoter can enhance its transcription, leading to increased production of this cytokine by T cells and natural killer (NK) cells. Histone methylation of the IL-4 promoter can also affect its transcription, leading to increased production of this cytokine by Th2 cells.

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Received: 01 February, 2023, Manuscript No. jmgm-23-94592; **Editor Assigned:** 03 February, 2023, Pre QC No. P-94592; **Reviewed:** 15 February, 2023, QC No. Q-94592; **Revised:** 21 February, 2023, Manuscript No. R-94592; **Published:** 28 February, 2023, DOI: 10.37421/1747-0862.2023.17.601

MiRNAs and immune cell function

MiRNAs can post-transcriptionally regulate the expression of genes that are crucial for immune cell function. For example, miR-155 is a miRNA that is upregulated in activated B cells and T cells and plays a crucial role in the regulation of immune responses. miR-155 can target various genes, including SOCS1 and SHIP1, which are negative regulators of cytokine signaling pathways. Therefore, miR-155 can enhance cytokine production and promote immune responses [6,7].

Recent studies have identified several novel epigenetic modulations of the systemic immune response. Here are some examples:The inflammasome is a multiprotein complex that plays a crucial role in the innate immune response by activating the production of pro-inflammatory cytokines, such as IL-1β and IL-18. Recent studies have identified epigenetic modifications that regulate the expression of inflammasome components. For example, DNA methylation of the NLRP3 gene promoter can affect the expression of this inflammasome component.

Conclusion

In addition to DNA methylation, histone modifications are also involved in the regulation of gene expression in immune cells. Histones are proteins that package DNA into nucleosomes, and modifications to histones can alter the accessibility of DNA to transcription factors and other regulatory proteins. Several histone modifications have been implicated in the pathogenesis of IBD, including histone acetylation, methylation, and phosphorylation. One study found that histone acetylation is increased in CD4+ T cells from patients with UC. Histone acetylation is typically associated with increased gene expression, and increased histone acetylation in CD4+ T cells may lead to dysregulated expression of genes involved in T cell activation and differentiation, contributing to the pathogenesis of UC. Another study found that histone methylation is altered in monocytes from patients with CD. Monocytes are a type of immune cell that plays a critical role in the innate immune response by phagocytosing pathogens and producing cytokines.

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How to cite this article: Hitzler, Muriel. "A Review on Immunoepigenetic Control of Inflammatory Bowel Disease." *J Mol Genet Med* 17 (2023): 601.