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Role of Long Non-coding RNAs (lncRNAs) in NF-κB-Mediated Macrophage Inflammation

Ana Bratic*

Department of Genome Editing, University of Galway, University Rd, Galway, Ireland

Introduction

Macrophage inflammation plays a pivotal role in the body's immune response, orchestrating defense against pathogens and maintaining tissue homeostasis. The NF-κB signaling pathway serves as a central regulator of macrophage inflammation, controlling the expression of numerous pro-inflammatory genes. Emerging evidence suggests that long non-coding RNAs exert significant influence on NF-κB-mediated macrophage inflammation, acting as regulators at various levels of gene expression. This article explores the roles of lncRNAs in modulating NF-κB signaling in macrophages, highlighting their potential as therapeutic targets for inflammatory diseases.

Macrophages are key players in the immune system, responsible for detecting and eliminating pathogens as well as maintaining tissue homeostasis. In response to various stimuli such as infection, injury, or inflammatory signals, macrophages undergo activation, leading to the production of inflammatory mediators. The NF-κB signaling pathway is a central regulator of this inflammatory response, controlling the expression of genes encoding cytokines, chemokines, and other pro-inflammatory molecules. Dysregulation of NF-κB signaling in macrophages is implicated in the pathogenesis of numerous inflammatory diseases, including arthritis, atherosclerosis, and inflammatory bowel disease. In recent years, there has been growing interest in understanding the regulatory mechanisms that modulate NF-κB-mediated macrophage inflammation, with a particular focus on long non-coding RNAs [1-3].

Description

Long non-coding RNAs are a diverse class of RNA molecules that lack protein-coding potential but play crucial roles in the regulation of gene expression. Through various mechanisms, including chromatin remodeling, transcriptional regulation, and post-transcriptional processing, lncRNAs can influence the expression of target genes. Recent studies have identified numerous lncRNAs that are differentially expressed in response to inflammatory stimuli in macrophages, suggesting their involvement in the modulation of NF-κB signaling and macrophage inflammation.

Several lncRNAs have been implicated in the regulation of NF-κB signaling in macrophages through diverse mechanisms. For example, lncRNA MALAT1 has been shown to promote NF-κB activation by enhancing the expression of NF-κB target genes in response to inflammatory stimuli. Similarly, lncRNA NEAT1 acts as a scaffold for the assembly of NF-κB signaling components, facilitating the activation of the pathway. Conversely, some lncRNAs exert inhibitory effects on NF-κB signaling, such as lncRNA GAS5, which sequesters NF-κB in the cytoplasm, preventing its translocation to the nucleus and subsequent activation of target genes. These findings highlight the complexity of lncRNA-mediated regulation of NF-κB signaling and macrophage inflammation.

Beyond their effects on NF-κB signaling, lncRNAs also play functional roles in macrophage inflammation. For example, lncRNA HOTAIR has been implicated in the regulation of macrophage polarization, promoting the M1 pro-inflammatory phenotype while inhibiting the M2 anti-inflammatory phenotype. Similarly, lncRNA MEG3 has been shown to modulate macrophage activation and cytokine production in response to inflammatory stimuli. These findings suggest that lncRNAs may serve as critical regulators of macrophage phenotype and function during inflammation [4,5].

The dysregulation of lncRNAs in macrophages has been linked to various inflammatory diseases, highlighting their potential as therapeutic targets. Strategies aimed at modulating the expression or activity of specific lncRNAs could offer novel therapeutic approaches for the treatment of inflammatory disorders. For example, small molecules or antisense oligonucleotides targeting pro-inflammatory lncRNAs could be developed as anti-inflammatory agents. Furthermore, the delivery of exogenous lncRNAs or lncRNA mimics could be explored as a means to modulate macrophage phenotype and function in inflammatory diseases.

Conclusion

In conclusion, lncRNAs play diverse and intricate roles in the regulation of NF-κB-mediated macrophage inflammation. By influencing various aspects of NF-κB signaling and macrophage function, lncRNAs emerge as critical regulators of the inflammatory response. Further research into the mechanisms underlying lncRNA-mediated regulation of macrophage inflammation may uncover new therapeutic targets for inflammatory diseases.

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***Address for Correspondence:** Ana Bratic, Department of Genome Editing, University of Galway, University Rd, Galway, Ireland, E-mail: ana.bratic8@gmail.com*

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