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The Multifaceted Roles of Long Non-coding RNAs in NF-κB-mediated Macrophage Inflammation

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Description

Long non-coding RNAs have emerged as pivotal regulators in various cellular processes, including inflammation. In the context of macrophage inflammation, the NF-κB signaling pathway plays a central role. This article elucidates the multifaceted roles of lncRNAs in modulating NF-κB-mediated macrophage inflammation. It explores the regulatory mechanisms through which lncRNAs influence NF-κB activation, cytokine production, and macrophage polarization, providing insights into their therapeutic potential in inflammatory diseases [1].

Macrophage inflammation is a complex process orchestrated by various signaling pathways, among which NF-κB stands out as a master regulator. NF-κB activation induces the expression of pro-inflammatory cytokines and mediates macrophage polarization, thereby shaping the immune response. Long non-coding RNAs (lncRNAs) have recently emerged as crucial players in fine-tuning gene expression at transcriptional and post-transcriptional levels. This article delves into the intricate interplay between lncRNAs and NF-κB signaling in macrophage inflammation, shedding light on their potential as therapeutic targets in inflammatory disorders.

NF-κB is a family of transcription factors that regulate the expression of genes involved in immune and inflammatory responses. In macrophages, NF-κB activation occurs in response to various stimuli, including pathogen-associated molecular patterns and cytokines. Upon activation, NF-κB translocates to the nucleus and initiates the transcription of target genes encoding pro-inflammatory cytokines such as Tumor Necrosis Factor alpha (TNF-α), Interleukin-1β (IL-1β), and Interleukin-6 (IL-6). Additionally, NF-κB signaling influences macrophage polarization, with classical (M1) activation promoting pro-inflammatory responses and alternative (M2) activation associated with tissue repair and immunoregulation [2].

Several lncRNAs modulate NF-κB signaling by regulating key components of the pathway. For instance, lncRNA NEAT1 enhances NF-κB activation by promoting the phosphorylation and degradation of IκBα, an inhibitor of NF-κB. Conversely, lncRNA GAS5 acts as a negative regulator of NF-κB by sequestering NF-κB subunits and preventing their nuclear translocation. Other lncRNAs, such as MALAT1 and HOTAIR, exert context-dependent effects on NF-κB activation, highlighting the complexity of their regulatory functions. LncRNAs play a crucial role in modulating the expression of pro-inflammatory cytokines downstream of NF-κB. For example, lncRNA THRIL promotes TNF-α expression by facilitating the recruitment of hnRNPL to the TNF-α promoter region. Similarly, lncRNA NKILA inhibits NF-κB-dependent transcription of pro-inflammatory genes, including IL-6 and IL-8, by interacting with the NF-κB/IκB complex. Emerging evidence suggests that lncRNAs contribute to macrophage polarization by modulating NF-κB signaling. LncRNA Mirt2 promotes M2 polarization by inhibiting NF-κB activation and enhancing STAT6 signaling, leading to increased expression of M2 markers such as Arg1 and Fizz1. Conversely, lncRNA THRIL promotes M1 polarization by enhancing NF-κB-dependent transcription of pro-inflammatory genes [3].

Understanding the intricate crosstalk between lncRNAs and NF-κB signaling is crucial for developing novel therapeutic strategies for inflammatory diseases. Targeting specific lncRNAs involved in NF-κB-mediated macrophage inflammation holds promise for modulating immune responses and attenuating excessive inflammation associated with various pathological conditions. Further research is warranted to elucidate the precise mechanisms underlying the regulatory functions of lncRNAs in macrophage inflammation and to explore their therapeutic potential in preclinical and clinical settings [4]. lncRNAs play diverse and dynamic roles in NF-κB-mediated macrophage inflammation, influencing NF-κB activation, cytokine production, and macrophage polarization. Elucidating the regulatory mechanisms of lncRNAs in this context provides valuable insights into the pathogenesis of inflammatory diseases and opens new avenues for therapeutic intervention [5].

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Conflict of Interest

None.

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