Commentary

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The Hidden Role of Non-Coding RNAs in COPD Pathogenesis and Progression

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Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory condition characterized by persistent airflow limitation, chronic inflammation, and structural remodeling of the lungs. Despite decades of research, COPD remains a leading cause of morbidity and mortality worldwide, largely due to the complexity of its molecular underpinnings and the lack of disease-modifying therapies. While environmental exposures such as cigarette smoke and biomass fuel combustion are well-established risk factors, growing evidence suggests that genetic and epigenetic mechanisms play equally crucial roles in disease susceptibility, progression, and heterogeneity. One of the most intriguing discoveries in recent molecular biology has been the emergence of non-coding RNAs (ncRNAs) as key regulators of gene expression and cellular homeostasis. Unlike messenger RNAs (mRNAs), ncRNAs do not encode proteins but exert regulatory effects at transcriptional, post-transcriptional, and epigenetic levels [1].

Description

The role of ncRNAs in cancer, cardiovascular disease, and neurological disorders is now well established; however, their role in chronic lung diseases such as COPD has only recently come into sharper focus. A deeper understanding of how ncRNAs orchestrate molecular networks in the lungs offers not only novel insights into disease biology but also potential therapeutic and diagnostic opportunities. This article explores the hidden role of ncRNAs in COPD pathogenesis and progression, highlighting their involvement in inflammation, cellular senescence, oxidative stress, immune dysregulation, and tissue remodeling, as well as their promise as biomarkers and therapeutic targets [2].

The role of microRNAs in COPD has been particularly well-documented. These short RNA molecules, typically 18–25 nucleotides in length, regulate gene expression post-transcriptionally by binding to complementary sequences in messenger RNAs and promoting degradation or inhibiting translation. In COPD, cigarette smoke exposure and chronic inflammation alter microRNA expression patterns in epithelial cells, alveolar macrophages, circulating immune cells, downregulation of miR-146a, an anti-inflammatory microRNA, has been associated with hyperactivation of NF-κB signaling, leading to exaggerated production of pro-inflammatory cytokines such as TNF-α and IL-6. Similarly, miR-21, a microRNA involved in fibrotic processes, is upregulated in COPD lungs, promoting tissue remodeling and fibrosis by modulating TGF-β signaling pathways. These findings suggest that miRNAs act as molecular switches that determine the balance between protective immune responses and chronic pathological inflammation. Moreover, circulating miRNAs have been proposed as potential non-invasive biomarkers for COPD diagnosis, prognosis, and monitoring of therapeutic responses. Their stability in body fluids, including blood and sputum, makes them attractive candidates for clinical translation [3,4].

Beyond microRNAs, long non-coding RNAs have garnered increasing attention in COPD research. LncRNAs are transcripts longer than 200 nucleotides that lack protein-coding potential but exert regulatory effects on chromatin remodeling, transcription, and post-transcriptional processing. Several lncRNAs have been found to modulate inflammation, apoptosis, and oxidative stress responses in lung tissues. lncRNA MALAT1, which is elevated in COPD patients, has been implicated in promoting inflammatory gene expression and exacerbating epithelial cell injury in response to cigarette smoke extract. Similarly, lncRNA HOTAIR, known for its role in chromatin remodeling, contributes to abnormal epithelial repair and airway remodeling in COPD. LncRNAs can also act as competing endogenous RNAs (ceRNAs), sequestering microRNAs and thereby indirectly regulating their target mRNAs. By acting as molecular sponges, lncRNAs can fine-tune the activity of multiple microRNAs simultaneously, amplifying or dampening pathological pathway [5].

Conclusion

The emerging field of non-coding RNA biology has revealed a hidden regulatory layer in the pathogenesis and progression of COPD. MicroRNAs, long non-coding RNAs, and circular RNAs intricately modulate inflammatory signaling, oxidative stress responses, immune cell dynamics, and tissue remodeling in the diseased lung. Their dysregulation contributes to the initiation and perpetuation of COPD, linking environmental insults such as cigarette smoke to molecular and cellular dysfunction. Beyond pathophysiological insights, ncRNAs represent a promising frontier for biomarker discovery and therapeutic innovation. While significant challenges remain in translating these findings into clinical practice, the integration of ncRNA research into COPD management has the potential to revolutionize diagnosis, prognosis, and treatment. As our understanding deepens, the hidden world of non-coding RNAs may ultimately illuminate new pathways toward precision medicine in COPD, offering hope for millions of patients burdened by this debilitating disease.

Acknowledgment

None.

Conflict of Interest

None.

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