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Short Notes on DNA Methylation and Importance of Precision Medicine for Idiopathic Pulmonary Fibrosis

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Abstract

Idiopathic pulmonary fibrosis (IPF) is a chronic and progressive lung disease with unknown etiology that leads to the formation of scar tissue in the lungs. Although the molecular mechanisms underlying IPF remain unclear, epigenetic changes have been implicated in the disease pathogenesis. Epigenetics refers to modifications to DNA and histones that alter gene expression without changing the DNA sequence itself. These modifications include DNA methylation, histone modifications, and non-coding RNA expression, all of which are influenced by environmental factors. Epigenetic changes have been observed in IPF, particularly alterations in DNA methylation and histone modifications. Studies have identified DNA methylation changes in genes related to extracellular matrix remodeling, inflammation, and oxidative stress, which are all pathways that are dysregulated in IPF.In addition, histone modifications, such as histone acetylation and methylation, have been shown to regulate the expression of genes involved in fibrosis and inflammation. Given the potential role of epigenetic changes in IPF, precision medicine approaches targeting epigenetic modifications have been proposed as a potential therapeutic strategy. One approach is the use of drugs that target specific epigenetic enzymes, such as histone deacetylases (HDACs) and DNA methyltransferases (DNMTs), which are involved in histone and DNA modifications, respectively. For example, HDAC inhibitors have been shown to reduce fibrosis and improve lung function in animal models of IPF. However, the use of these drugs in human clinical trials has been limited due to their potential side effects and lack of specificity.

Keywords: Histone deacetylases • Methylation • DNA methyltransferase

Introduction

DNA methylation is a biochemical process that involves the addition of a methyl group to a DNA molecule, usually to a cytosine base in a CpG dinucleotide context. This process is important for regulating gene expression and plays a critical role in various biological processes, including embryonic development, imprinting, X-chromosome inactivation, and carcinogenesis. Methylation occurs when a DNA methyltransferase enzyme transfers a methyl group from S-adenosyl methionine to the carbon-5 position of the cytosine ring. The result is 5-methylcytosine (5mC), which can be further modified to other forms of methylated cytosines, such as 5-hydroxymethylcytosine (5hmC), 5-formylcytosine (5fC), and 5-carboxylcytosine (5caC).The presence of methyl groups on DNA can affect gene expression by altering the accessibility of the DNA to transcription factors and other proteins. Methylated DNA is generally associated with gene silencing, while unmethylated DNA is associated with active gene expression. The patterns of DNA methylation are often heritable and can be passed down from one generation to the next [1-3]. In summary, DNA methylation is an important epigenetic mechanism that plays a critical role in regulating gene expression and various biological processes.

Another approach is the use of non-coding RNA molecules, such as microRNAs (miRNAs) and long non-coding RNAs (lncRNAs), which regulate gene expression at the post-transcriptional level. Dysregulation of miRNAs has been implicated in IPF, and studies have shown that restoring miRNA expression can reduce fibrosis and improve lung function in animal models. Similarly, lncRNAs have been shown to play a role in IPF pathogenesis, and targeting specific lncRNAs has been proposed as a potential therapeutic strategy. In conclusion, epigenetic changes play a role in the pathogenesis of IPF, and precision medicine approaches targeting these changes have the potential to be effective therapies for this disease. While more research is needed to understand the specific epigenetic changes involved in IPF and to develop targeted therapies with minimal side effects, these approaches represent an exciting new avenue for the treatment of this debilitating disease.

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Literature Review

The epigenetics process, rather than DNA, is used to transmit genetic information. Epigenetics is the study of molecular missing link pathways that may bridge the genetic and environmental risk factors that contribute to the pathogenesis of pulmonary fibrosis. Endophenotypes underlying the development of idiopathic pulmonary fibrosis are affected by specific epigenetic patterns, particularly DNA methylation, histone modifications, long non-coding RNA (lncRNA), and microRNA (miRNA). (IPF). DNA methylation modifications have been the most extensively studied epigenetic marks in IPF. This review summarises current knowledge about DNA methylation changes in pulmonary fibrosis and shows a promising novel epigenetics-based precision medicine approach [4,5].

IPF is a chronic, debilitating, and irreversible lung disease characterised by microinjury-induced alveolar epithelial cell stress, pathogenic myofibroblast differentiation, imbalanced macrophage polarization, and extensive extracellular matrix deposition. (ECM). IPF progression is associated with decreased lung function, progressive respiratory failure, high mortality, recurrent acute exacerbations, and an overall poor prognosis. On histopathological and/or radiological examination, IPF is distinguished by heterogeneous areas of normal-appearing lung intermixed with collagenized fibrosis in sub-pleural and paraseptal, a honeycombing pattern, and ECM-producing myofibroblasts referred to as fibroblast foci.

Discussion

According to a recent hypothesis, recurrent injuries cause epithelial cells to

transdifferentiate into fibroblast epithelial-mesenchymal transition (EMT), which may induce fibrosis independently of inflammatory events. Despite the lack of an explicit mechanism, several pieces of evidence suggest that alveolar epithelial injury caused by environmental triggers results in lung fibrosis. Recurrent microenvironment injury to senescent epithelial cells in genetically susceptible individuals results in abnormal fibroblast activation, accumulating ECM, and fibrosis. The pathogenic mechanisms underlying the onset, development, and progression of IPF are unknown. Many studies, however, have shown that in older people, dynamic interactions of genetic susceptibility, environmental factors, and host risk factors contribute to epigenetic pro-fibrotic reprogramming, resulting in the development of IPF. Hey et al. discovered a strong link between microenvironment-driven epigenetic changes and macrophage inflammation and polarization [6,7].

Omics-based approaches, which include high-throughput technologies that provide snapshots of a holistic view of the molecules that make up a cell, tissue, or organism, include: genomics, which measures deoxyribonucleic acid (DNA) sequence variation; epigenomics, which focuses on the genome-wide characterization of reversible modifications of DNA or DNA alterations; transcriptomics, which evaluates the standard of ribonucleic acid (RNA) expression; and (4) Pulmonary disease omics research has primarily focused on tissue- and cell-specific omics data and has identified several fundamental mechanisms underlying pulmonary biological processes, disease endotypes, and appropriate novel therapeutics for specific individuals.

Conclusion

Epigenomics is the study of gene expression via epigenetic mechanisms such as DNA methylation, RNA, and histone modification. Because these components interact and stabilise one another, disrupting epigenetic nucleosomes can result in their inappropriate expression, resulting in epigenetic disorders. Epigenetics and epigenomics can help us understand how our environment influences our phenotype. Hi-C, a comprehensive technique developed to capture chromosome conformation, and another tool for whole genome methylation profiling: MBD-isolated genome sequencing are commonly used in epigenomics studies. (MiGS). These authors thoroughly examine the technical and experimental parameters that must be considered when designing epigenomic experiments.

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Conflict of Interest

Authors declare no conflict of interest.

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