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Development and Factors Involved in Epigenetic Asthma

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DESCRIPTION

Asthma is one of the most common respiratory illnesses affecting both children and adults around the world, and the various phenotypes and underlying etiological mechanisms are not well understood. As the technology of genome sequencing has advanced, scientific efforts have been made to explain and predict the complexity and heterogeneity of asthma, and Genome Wide Association Studies (GWAS) have rapidly become the preferred research method. Over the last few decades, several genetic markers and loci associated with asthma susceptibility, atopic and childhood-onset asthma have been identified.

Asthma is a mysterious disease with heterogeneity in etiology, and clinical phenotype. On-going studies have provided a better understanding of asthma, but in many respect its natural history, progression, etiology, diverse phenotypes, and even the exact posterior association between childhood asthma and adult/senile asthma. It remains elusive. Although heritability of asthma has been demonstrated by genetic studies, genetics is not the only factor affecting asthma. The increased incidence and some open questions suggest that there may be other factors related to the inheritance of asthma. Epigenetic mechanisms link genetic and environmental factors to the developmental trajectory of asthma.

Epigenetic mechanisms represent potential molecular pathways that can bridge the gap between the genetic background and the environmental risk factors that contribute to the pathogenesis of lung disease. In patients with COPD, asthma, and Pulmonary Arterial Hypertension (PAH), aberrant epigenetic marks that directly mediate DNA reversible modifications, primarily DNA methylation and histone modifications, without affecting genomic sequences. Evidence is increasing. Post-translational events and microRNAs are also epigenetic and may be involved in the etiology of the disease. Therefore, new etiological mechanisms and presumed biomarkers can be detected in peripheral blood, sputum, nasal and cheek swabs or lung tissue. In addition, DNA methylation plays an important role in the early stages of fetal development, can be affected by environmental factors, and ultimately affects susceptibility to later COPD, asthma, and PAH.

CONCLUSION

Idiopathic Pulmonary Fibrosis (IPF) is a fatal lung disease of unknown origin that can lead to other dangerous diseases such as lung cancer. Environmental and genetic predispositions are two major causes or risk factors involved in the pathology of IPF. Among the environmental risk factors, smoking is one of the leading causes of the development of IPF. Acquired signaling pathways such as nucleosome remodeling, DNA methylation, histone modification, and miRNA-mediated genes play important roles in the development of IPF. Genetic mutations target epigenetic factors as important drug targets for IPF. Changes in transcription due to environmental factors are also associated with the progression of IPF.

Exposure to particulate matter in the air increases the risk of developing human illness. Epigenetic mechanisms are associated with environmental stress and human illness. This review focuses on currently available studies demonstrating the relationship between epigenetic marks, exposure to air pollution, and human health. Air pollutants involved in epigenetic changes are associated with a variety of specific mechanisms (DNA methylation, post-translation histone modifications, and non-coding RNA transcripts), which are described in a separate section. Several studies explain how these epigenetic mechanisms are affected by environmental factors, including air pollution. This interaction between PM and epigenetic factors modifies the profile of these marks, both globally and site-specific.

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CONFLICTS OF INTERESTS

The authors declare that they have no conflict of interest.

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