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Commentary

Age-Related Genes with Variable Expressed Methylation Sites in Asthma

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DESCRIPTION

In addition to abnormal immunological reactions to allergens, reversible airflow restriction, Airway Hyper-Responsiveness (AHR), and other environmental insulators, asthma is a complicated pulmonary inflammatory illness. Approximately 5%-10% of asthma patients are still steroid-refractory, which always has lower lung function and higher mortality. Despite the fact that bronchodilators and inhaled/systemic corticosteroids are extremely effective in the majority of asthma patients, these patients still have lower lung function. Traditional theories like "allergic constitution" or "airway inflammation" fall short when it comes to describing how and why asthma develops. As a result, an increasing number of researches are looking for new internal aetiology of asthma and identifying new potential treatment targets.

It's interesting to note that asthma is frequently more severe and has fewer chances of going into remission in older people (age over 65). Idiopathic Pulmonary Fibrosis (IPF) and chronic obstructive pulmonary disorders are examples of chronic lung diseases that have been linked to ageing in accumulating studies Chronic Obstructive Pulmonary Disease (COPD). It is well established that the pathophysiological changes in asthma, such as airway remodelling, chronic inflammation, and reduced lung function, are similar to those in COPD and IPF. It is conceivable to hypothesise that ageing may have a role in the emergence of asthma. Indeed, a number of solid pieces of data suggest that getting older poses a serious risk for the development of asthma. Asthma sufferers' immunological and structural cells have both undergone aging-related alterations. What is noteworthy is that telomere attrition, epigenetic changes, proteostasis loss, and altered intercellular communication have all been seen in asthma patients. Additionally, ageing can affect the diagnosis, management, and appearance of asthma, which is important for the treatment of asthma. Aging of various targeted cells can also contribute to asthma pathobiology, including airway remodelling, inflammation, and impaired lung function. Furthermore, it has been demonstrated that anti-aging techniques can help asthma patients with pathological processes such airway inflammation and airway remodelling.

Despite the fact that there is mounting scientific evidence linking ageing with asthma, the exact mechanisms of ageing and how it contributes to the emergence of asthma are still unknown. Numerous recent studies have shown that epigenetic mechanisms have a role in the control of the expression of genes relevant to ageing. Without changing the nucleotide sequence, epigenetic mechanisms involving DNA, microRNA expression, and histone changes can control the transcriptional activity of target genes. The epigenetic regulation of DNAm, which has been shown to be particularly important in the regulation of genes associated to ageing, is the one that has been investigated the most thoroughly. It has been demonstrated, in particular, that cytosine methylation at the CpG site altered a number of regulatory mechanisms of aging-related genes during transcription and further contributed to aging-related diseases as asthma and COPD. On the DNAm changes of aging-related genes in asthma patients, however, there is yet no conclusive literature.

It has been discovered that in the peripheral venous blood of COPD patients, DNAm controls the expression of 9 genes associated to ageing. Additionally, the prevalence and severity of COPD were directly correlated with the degree of methylation at a few unique CpG sites. We aim to investigate the involvement of DNAm of aging-related genes in asthma patients in order to further investigate the potential involvement of these nine previously screened aging-related genes in the parthenogenesis of asthma. In this work, we first looked at the expression and DNAm level of the 9 aging-related genes in HCs and asthmatic patients' peripheral venous blood. The relationship between DMSs and clinical markers in patients with asthma was then examined. Finally, we evaluated the viability of using the methylation levels or methylation change rates of particular DMSs as biomarkers to differentiate HCs from asthma.

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