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Perspective

Identification of Chronic Inflammatory Asthma and Bioinformatics Analysis

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DESCRIPTION

Chronic, heterogeneous, inflammatory asthma has a wide range of clinical symptoms and complex pathological processes. One of the phenotypes is severe asthma, which is defined as uncontrolled asthma despite adherence to the most effective therapy and asthma that gets worse when the high-dose medication is reduced. With a 3.1 fold higher risk of osteoporotic fracture and a 2.7 fold higher risk of pneumonia, patients with severe asthma try to maintain control and prevent life-threatening exacerbations with large doses of inhaled corticosteroids or even oral corticosteroids. Additionally, severe asthma patients frequently develop corticosteroid resistance, which reduces the efficacy of corticosteroid therapy. Novel immune-related medicines have been created while taking the drawbacks and adverse effects of conventional medications into account. Nonetheless, early attempts at immunosuppressive treatments have failed, underscoring a thorough knowledge

Patients with severe asthma account for between 50 and 60 percent of asthma costs and are linked to low life expectancy, high mortality, and high morbidity. It has been difficult for clinicians to treat this subtype of asthma due to the uncertain molecular basis and refractory response to conventional asthmatic medications reported in these patients. In this study, we used the Differentially Expressed Genes (DEGs) between severe and mild asthma samples for the first time to build a co-expression network by Weighted Gene Coexpression Network Analysis (WGCNA) and performed a thorough analysis of key genes and pathological processes associated with asthma severity in the hopes that the results will provide ideas for the comprehension and future treatment of severe asthma.

The Toll-Like Receptor (TLR) family of receptors serves as the body's initial line of defence against invasive microorganisms. They trigger Th17 reactions, increasing IL-8 and IL-17 production, which can alter the structure of the airways and contribute to the reduced FEV1, remodeling, and airway blockage seen in those with severe neutrophilic asthma. This study found that TLR2 expression was rising in patients with severe asthma. The increase of TLR2 in non-eosinophilic asthmatic sputum T cells and TLR2 pathway in severe asthma are consistent with our findings. TLR2 may lower Th17 cytokines, however, by blocking a Th17 phenotype in Treg cells, according to a recent study. It suggests that TLR2 may cause an asthmatic remission. A second mouse study found that the TLR2/4 pathway may assist to avoid asthma later in life if it is stimulated appropriately.

Heavy neutrophil chemotactic factor FPR1, associated with chronic inflammatory disorders. Although FPR1 was said to respond to cigarette smoke and participate in glucocorticoid's anti-inflammatory actions, little was known regarding its impact on the development of asthma.

Immune-related genes include FCGR3B, FCGR2A, and ITGAM. Many immune-mediated conditions, including systemic lupus erythematosus and severe nephropathy, are linked to FCGR2B and FCGR3A. ITGAM is also referred to as the systemic lupus erythematosus biomarker. Platelets, monocytes, macrophages, and lymphocytes are the primary protein kinase C substrates for PLEK. It is unknown how exactly these genes affect asthma patients.

To find processes and hub genes in severe asthma, co-expression modules were initially constructed using WGCNA and DEGs of mild-to-severe asthmatics from bronchial epithelial brushings. Our study does have certain limitations. Secondly, the study's sample sizes are limited, mostly because there aren't many associated gene expression profiles with diverse clinical characteristics. Second, future research is required to substantiate our research findings, which are based on data from open web databases. Third, additional experiments are necessary to explain the specific processes of identified hub genes.

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