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Opinion Article

# Effects of Bronchial Asthma on Children's Growth

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## DESCRIPTION

Previous studies have placed a high importance on bronchial asthma because of its recurrent nature, its effects on children's growth and development, and the reduction in quality of life it causes. Bronchial asthma is also thought to be a significant financial burden. In China, there are around 30 million asthmatic patients, with children accounting for nearly a third of the total number of cases. The pathogenesis of asthma is complex, the most important immunological mechanism implicated in the pathophysiology of eosinophilic asthma is Th1/Th2 imbalance, which is regarded the dominating factor in Th2 imbalance.

The most frequent non-infectious disease in children is bronchial asthma. There are over 300 million asthmatic patients worldwide. In recent years, the prevalence of this disease has risen significantly in middle- income and low-income countries. The increase of eosinophils is thought to be one of the characteristics of asthmatic airway inflammation. However, in recent years, some patients did not have an increased sputum eosinophil number, and more than 50% of asthmatic patients had neutrophil infiltration of airway inflammation. The number of neutrophils was also higher during exacerbations and increased duration of asthma. The importance of neutrophil infiltration in the treatment of severe asthma, fatal asthma, chronic persistent asthma, and refractory asthma is increasingly emphasized. It is of particular significance to study and understand the role of neutrophil infiltration in the pathogenesis of asthma

The chronic airway inflammation that causes asthma by epithelial cells, fibroblasts, dendritic cells, neutrophils, eosinophils, mast cells, T-lymphocytes, and other cells. Th2-related cytokines, as well as a number of pathogenic processes triggered by mucus secretion, cell proliferation, and eosinophil infiltration, are hypothesised to have a role in the development and maintenance of allergic asthma, with the severity of symptoms correlating positively. In contrast to these findings, it has been demonstrated that IFN-secretion by Th1 cells inhibits Th2 proliferation and possesses anti-inflammatory properties. The Th1/Th2 imbalance is defined by relative suppression of Th1 activity and relative hyperfunction of Th2, resulting in increased IgE production and the release of cytokines such as IL-4, IL-5, and IL-13.

CD4+T cell subsets, B cells, and mast cells all produce IL-4 it is a pleiotropic cytokine. Th2 cells release this cytokine, which has a strong chemical chemotactic effect on eosinophils, neutrophils, and other inflammatory cells. It can cause exacerbations of acute asthma by increasing the concentration and activation of inflammatory cells in the airways. IFN- is a cytokine that has recently been researched and has been shown to decrease B lymphocyte proliferation as well as IgG1 and IgE release. It can also stop B cells from expressing low affinity IgE receptors. IFN- may reduce allergic reactions and asthma symptoms while also boosting immune function and acting as a non-specific anti-infection agent.

Furthermore, it has the ability to improve phagocytic function while inhibiting plasma cell survival, which is protective in the aetiology of asthma. In recent years, it has been demonstrated that neutrophil infiltration in bronchial biopsies and generated sputum is responsible for asthmatic patients' severe or acute exacerbation. Neutrophils primarily produce NE, MMPs (most notably MMP-9) and IL-17. Basement membrane elastin is mostly degraded by NE, which is linked to lung tissue injury. Increased NE levels in neutrophils can cause ARDS and severe acute lung damage. It was discovered that NE levels were significantly higher in BALF samples from patients with acute exacerbation of severe asthma and in patients with severe asthmatic respiratory tract submucosal inflammation, regardless of the presence of eosinophils, indicating that NE promotes neutrophil recruitment and persists in the inflammatory response at accumulation sites. MMP9 and NE concentrations were found to be positively linked. Previous research has shown that Th17/IL-17 activation can exacerbate asthmatic airway hyper responsiveness and inflammation, with the degree of IL-17 elevation being strongly linked to asthma severity.

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