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Commentary

Role of Innate Lymphoid Cells in Airway Remodeling and Persistent Asthmatic Inflammation

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DESCRIPTION

Asthma is a chronic respiratory condition characterized by airway inflammation, bronchial hyperresponsiveness, and structural remodeling of the airways. While adaptive immune responses have long been considered central to disease development, increasing attention has shifted toward innate immune populations that operate at mucosal surfaces. Innate lymphoid cells are a recently characterized group of immune cells that lack antigen-specific receptors yet contribute significantly to mucosal immunity and tissue regulation. Their involvement in airway inflammation and remodeling has expanded understanding of asthma pathophysiology. This article examines the contribution of innate lymphoid cells to persistent asthmatic inflammation and airway structural changes.

The respiratory tract is continuously exposed to environmental particles, allergens, microorganisms, and pollutants. To maintain airway integrity, the immune system employs both rapid and adaptive defense mechanisms. Epithelial cells form the first barrier and actively participate in immune signaling by releasing cytokines and chemokines in response to injury or irritation. These signals activate resident immune populations, including innate lymphoid cells, which respond rapidly without prior antigen exposure.

Innate lymphoid cells are classified into several subsets based on cytokine production and transcription factor expression. Type 2 innate lymphoid cells are particularly relevant in allergic airway diseases. These cells produce cytokines such as interleukin-5 and interleukin-13, which contribute to eosinophilic inflammation, mucus production, and airway hyperreactivity. Their activation is driven by epithelial-derived mediators released during allergen exposure or viral infection.

In asthmatic airways, epithelial injury leads to increased production of cytokines such as interleukin-25, interleukin-33, and thymic stromal lymphopoietin. These mediators stimulate innate lymphoid cells and enhance their cytokine output. The resulting inflammatory environment promotes recruitment of eosinophils and other immune cells that contribute to airway inflammation. Eosinophils play a major role in asthma-related tissue damage. These cells release cytotoxic granule proteins, lipid mediators, and reactive oxygen species that affect airway epithelial integrity. Interaction between innate lymphoid cells and eosinophils amplifies inflammatory responses and contributes to persistent airway dysfunction.

Airway remodeling represents a long-term consequence of chronic inflammation. Structural changes include smooth muscle hypertrophy, goblet cell hyperplasia, subepithelial fibrosis, and increased extracellular matrix deposition. Cytokines produced by innate lymphoid cells influence these processes by stimulating fibroblast activation and mucus production.

Interleukin-13 is particularly important in driving mucus hypersecretion and epithelial remodeling. Elevated levels of this cytokine promote goblet cell differentiation and increased mucus viscosity, which can obstruct airflow. Continuous production of interleukin-13 contributes to chronic symptoms and reduced lung function. Interleukin-5 produced by innate lymphoid cells supports eosinophil growth, survival, and recruitment. Sustained eosinophilic inflammation is associated with disease severity and frequent exacerbations. The persistence of these cells in airway tissues contributes to ongoing inflammation and structural damage.

The interaction between innate lymphoid cells and adaptive immune responses is complex. Although innate lymphoid cells act independently of antigen recognition, they influence T helper cell differentiation and cytokine balance. This interaction contributes to the overall inflammatory profile observed in asthmatic individuals. Type 2 T helper cells and innate lymphoid cells share similar cytokine profiles, leading to overlapping functions in allergic inflammation. However, innate lymphoid cells respond more rapidly to environmental stimuli, making them important in early disease activation and acute exacerbations.

Airway epithelial cells also interact directly with innate lymphoid cells. Damage to epithelial barriers exposes underlying tissues to environmental irritants, further stimulating immune activation. This epithelial-immune interaction forms a feedback loop that maintains inflammation.Recent studies using single-cell analysis

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have revealed significant diversity among innate lymphoid cell populations in the lungs. Distinct subsets exhibit different functional profiles and localization patterns within airway tissues. This heterogeneity may explain variability in disease presentation among patients.

Airway microbiota composition may also influence immune activation. Microbial communities present in the respiratory tract interact with epithelial cells and immune populations, affecting cytokine production and inflammatory signaling. Alterations in microbial balance may contribute to disease exacerbation. Therapeutic approaches targeting cytokines produced by innate lymphoid cells have shown clinical benefits in reducing asthma severity. Biological agents that inhibit interleukin-5 and interleukin-13 signaling pathways have demonstrated effectiveness in controlling eosinophilic inflammation.

CONCLUSION

Innate lymphoid cells play a significant role in the development and persistence of airway inflammation and remodeling in asthma. Through rapid cytokine production and interaction with epithelial and immune cells, they contribute to both acute symptoms and long-term structural changes in the airways. Continued research into their biological functions may support improved strategies for managing chronic respiratory inflammation.