Mediators of Inflammation

Hindawi

Special Issue on

Airway Tissue Remodeling in Asthma and Chronic Obstructive Pulmonary Disease: The Role of Mediators of Inflammation

CALL FOR PAPERS

Asthma and chronic pulmonary disease (COPD) are the most prominent obstructive airway diseases and affect millions of people with increasing incidence. The functional consequence of asthma and COPD is airflow limitation and chronic inflammation, conditions associated with structural changes of the lung parenchyma, referred to as remodeling, goblet cell hyperplasia, mucus gland hypertrophy, and the luminal obstruction caused by inflammatory exudates and mucous. Some recent evidences support the idea that a significant part of the phenotypic overlap between asthma and COPD can be ascribed to the airway remodeling that takes place in these diseases. Airway remodeling occurs in both asthma and COPD albeit with important differences in the structures, anatomic sites, and cell types involved in the two diseases. The immune response represents an important feature that contributes to the differentiation of asthma and COPD.

The airways of asthmatic individuals are characterized by a T-helper cell 2 profile of inflammation that orchestrates the release of cytokines and chemokines involved in the recruitment and activation of the primary effector cells of the allergic response, namely, mast cells and eosinophils. Consequently, such inflammatory cells release mediators that trigger bronchoconstriction, mucous secretion, and, possibly, remodeling. Conversely, COPD is characterized by the accumulation, in the airway and in the lung parenchyma, of neutrophils, macrophages, and CD8 T-lymphocytes that release chemokines involved in pulmonary remodeling, including excessive proliferation of endothelial cells, smooth muscle cells, and fibroblasts.

In the last decade, research has elucidated that chronic inflammation of the airways is the most important determinant of tissue injury and airway remodeling. Nonetheless, the complex mechanism underlying this process and the specific elements of airway remodeling which contribute to the symptoms or progression of asthma and COPD remain poorly understood and asthma and COPD are still underdiagnosed (or misdiagnosed) and undertreated. Hence, the understanding of molecular basis of tissue remodeling in these two diseases at an early stage is fundamental for selection of the correct therapy to reduce symptoms, morbidity, and mortality.

In this special issue, we are interested in reviews as well as original articles that can give an overview about the current research on the role of immune, epithelial, vascular endothelial, and smooth muscle cells in mediating the airway remodeling in asthma and COPD and new advances in the therapy.

Potential topics include, but are not limited to:

- How inflammatory cells and cytokines and chemokines orchestrate airway remodeling in asthma and COPD
- Role of intracellular signaling pathways in fibrosis and tissue destruction
- Significance of the vascular component on the airway remodeling
- Role of epithelium in airway remodeling
- New advances in the understanding of the molecular mechanisms underlying the asthma and COPD overlap syndrome (ACOS)
- Cell-based therapy in asthma and COPD

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/mi/aria/.

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