Hindawi

Mediators of Inflammation

Special Issue on

Mediators of Inflammation in Asthma

CALL FOR PAPERS

Multiple factors contribute to asthma severity as clinical presentations, physiological characteristics, and immune responses which are exceedingly diverse. To date, two major clinical forms of asthma have been defined, allergic and nonallergic (intrinsic). Although allergic asthma was long considered the hallmark T helper type 2 (TH2) disorders of the lungs, it is now known that group 2 innate lymphoid (ILC2) cells also promote eosinophilic asthmatic inflammation and airway hyperresponsiveness. Functionally, TH2 and ILC2 cells share multiple overlapping characteristics in the lung, including differential TH2-associated cytokine production and expression of chemokine receptors. In addition to TH2 and ILC2-associated airway inflammation, emerging evidence implicates a distinct role of other immune cell subtypes such as T helper 17 (TH17) cells, regulatory T (Treg) cells, and dendritic cells in asthma subpopulations. While increased leukocyte populations drive inflammation in distinct asthma subsets, abnormal or disrupted airway epithelial cell barrier and increased airway smooth muscle (ASM) are also associated with asthma severity and progression. Extensive support now shows that aberrant and dynamic effects (paracrine/autocrine) of structural cell-derived chemokines/cytokines, matrix proteases, and cell adhesion molecules mitigate leukocyte homeostasis, airway inflammation, immune cell dysfunction, and airway remodeling in asthma.

We invite investigators to contribute original research articles as well as review articles focusing on the inflammatory mediators in asthma pathogenesis. We are particularly interested in research articles illustrating the cell-cell communication in asthmatic airways. Moreover, papers examining the crosstalk between epithelial, ASM, and immune cells that contribute to the initiation of airway disease would be of great interest.

Potential topics include, but are not limited to:

- Inflammatory mediators in asthma pathogenesis
- Regulation of TH2 and ILC2 cell populations in asthma
- Regulation of TH17 and ILC3 cell populations in asthma
- Regulation of Treg cells in asthma
- Asthma exacerbations and respiratory infections
- Epithelial cell-derived mediators in asthma
- Role of thymic stromal lymphopoietin (TSLP) in asthma
- ASM-derived mediators in asthma
- Regulation of toll-like receptors (TLR) in structural and immune cells of the airway
- Identification of novel asthma biomarkers
- Inflammatory mediators and the extracellular matrix in the asthmatic airway

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

Lead Guest Editor
Sathish Venakatachalem, Mayo Clinic, Rochester, USA
venkatachalem.sathish@mayo.edu

Guest Editors
Patrick Geraghty, SUNY Downstate Medical Center, Brooklyn, USA
patrick.geraghty@downstate.edu

Gautam Damera, Medimmune LLC, Gaithersburg, USA
damerag@medimmune.com

Sinead Weldon, Queen's University Belfast, Belfast, UK
s.weldon@qub.ac.uk

Manuscript Due
Friday, 19 August 2016

First Round of Reviews
Friday, 11 November 2016

Publication Date
Friday, 6 January 2017