Mediators of Inflammation

Hindawi

Special Issue on

Airway Inflammatory/Immune Responses in COPD and Cystic Fibrosis

CALL FOR PAPERS

Cystic fibrosis (CF) is a genetic disease caused by mutations in the cystic fibrosis transmembrane conductance regulator (CFTR) gene and remains one of the most common fatal hereditary disorders worldwide. Although CF is a complex multiorgan disease, morbidity and mortality are mainly determined by progressive chronic obstructive lung disease.

COPD is a major and increasing global health problem. The disease is caused by both genetic and environmental factors, and cigarette smoking is the main risk factor.

Although the pathogenesis and the pathophysiology of these diseases are different, they share key phenotypical features, including reduced mucociliary clearance, small airways mucus obstruction, goblet cell metaplasia and mucus hypersecretion, chronic neutrophilic airway inflammation, exacerbated innate and adaptive immune responses in airways, and chronic/recurrent bacterial infections. In both diseases, a persistent high-intensity inflammation, driven by continuous neutrophil recruitment, leads to permanent structural damage of the airways and impaired lung function. Furthermore, both patients with CF and COPD also suffer from systemic inflammation.

Several defective inflammatory responses have been linked to CFTR deficiency including innate and acquired immunity dysregulation, cell membrane lipid abnormalities, and various transcription factor signaling defects. Interestingly, cigarette smoke has also been implicated in reduced CFTR expression and function, suggesting that common mechanisms may contribute to the chronic nonresolving inflammation in both CF and COPD. These mechanisms however are still poorly defined. An improved understanding of the common mechanisms and/or pathways that can be affected by both CFTR mutations and environmental insults involved in COPD should provide a road map for potential therapeutic interventions in future COPD therapies.

This special issue will focus on airway inflammation, structural abnormalities, and immune responses in CF and COPD. We invite investigators to contribute original research articles as well as review articles focusing on similarities and differences between inflammatory/immune responses in CF and COPD, potential mechanistic links, and common therapeutic targets.

Potential topics include but are not limited to the following:

- Investigations into evidence of common inflammatory mechanisms in CF and COPD
- Effect of CFTR dysfunction on airway inflammatory/immune responses
- Role of protease and redox imbalance in CF and COPD inflammatory responses
- Possible biomarkers of the airway inflammatory process in both diseases
- Infection/microbiome and inflammatory/immune response in the CF or COPD in lung
- Potential common therapeutic targets in CF and COPD airway inflammation
- Therapeutic potential of ion transport modulators in CF and COPD inflammatory process

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

Papers are published upon acceptance, regardless of the Special Issue publication date.

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