European Respiratory Society
Annual Congress 2012

Abstract Number: 4848

Publication Number: P1433

Abstract Group: 3.2. Airway Cell Biology and Immunopathology

Keyword 1: COPD - mechanism **Keyword 2:** Animal models **Keyword 3:** Immunology

Title: The role of IL -17 and lymphoid follicles in the pathogenesis of COPD

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Body: Chronic Obstructive Pulmonary Disease (COPD) is one of the most prevalent respiratory diseases in the world. There is no definitive treatment to arrest the progressive loss of lung function characteristic of COPD, partly due to a lack of understanding of the underlying cellular pathophysiological mechanisms. In individuals with severe COPD, there is an accumulation of adaptive immune cells as well as an increase in the frequency of lymphoid follicles in the lung; however, the role of lymphoid follicles in driving the disease and the factors that promote their formation are largely unknown. In addition, individuals with COPD exhibit elevated levels of IL-17, a cytokine that is associated with autoimmunity and was recently shown to promote lymphoid neogenesis. We have modeled different severities of COPD by incremental instillation of LPS and elastase in mice. We observed an increase in the levels of IL-17A following repeated challenges, which coincided with the progressive drop in lung function as well as the appearance of lymphoid follicles. IL-17A production was in part triggered by the engagement of Toll Like Receptor 3 implicating a role for an endogenous danger signal in COPD. Neutralization of IL-17A at specific times after the initiation of disease ameliorated the impaired lung function and affected B Cell and macrophage activation states. Our data indicates that IL-17A is involved in COPD progression by induction of lymphoid follicles and regulation of both innate and adaptive immunity.