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## New Insights into the Pathological Features of Asthma/COPD and Pulmonary Arterial Hypertension

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Chronic inflammatory airway diseases such as bronchial asthma and chronic obstructive pulmonary disease (COPD) are major contributors to the global burden of disease [1,2]. Pulmonary arterial hypertension (PAH) is a rare but life-threatening disease characterized by increasing pulmonary vascular resistance and progressive pulmonary hypertension that leads to right ventricular failure and death [3]. There is mounting evidence that asthma, COPD and PAH share important pathological features, including inflammation, smooth muscle contraction and remodeling [4, 5]. A better understanding of the pathobiochemical mechanisms that causally involved in these multifactorial diseases can contribute to developing effective therapeutic strategies that target multiple genes and signal transduction pathways.

Asthma is a worldwide problem with an estimated 300 million affected individuals. The World Health Organization (WHO) has estimated that 15 million disability-adjusted life years are lost annually due to asthma, representing 1% of the total global disease burden [6]. The key clinical features of asthma are airflow obstruction and airway hyper responsiveness that caused by airway inflammation [2]. Many of the inflammatory events in asthma are thought to be mediated by Th2 cells. It also involves mast cells, eosinophils, neutrophils and mesenchymal cells such as epithelial cells, fibroblasts, smooth muscle cells and endothelial cells. The inflammatory mediators, including cytokines, chemokines, adhesion molecules, proteinases and growth factors released by these cells participant in this process at various stages and interact to maintain and amplify the inflammatory response [6]. Two categories of drugs are currently used in asthma therapies: bronchodilators and anti-inflammatory drugs. Despite the availability of these medications, the asthma epidemic continues to increase. The key clinical feature of COPD is airflow limitation results from airway constriction and irreversible reduction in the caliber of the small airways of the lung. Cigarette smoking is an important risk factor of COPD. The airflow limitation or obstruction that happens in COPD is caused by a mixture of small airway disease, parenchymal destruction (emphysema) and in many cases, increased airway responsiveness (asthma) [1]. Currently available medications that are helpful in treating COPD include bronchodilators and corticosteroids. Antibiotics are useful in treating exacerbations caused by bacterial infections. No medications have been found to cure the disease or reverse the loss of lung function caused by smoking [1]. The main pathological features of PAH in the pulmonary vasculature are perivascular inflammation, thrombosis, abnormal growth of vascular smooth muscle cells and extracellular matrix accumulation, leading to remodeling of the pulmonary vessel wall, obstruct pulmonary blood flow and ultimately cause right heart failure. Current treatment of PAH, which includes the use of prostacyclins, endothelin receptor antagonists, and phosphodiesterase type 5 inhibitors, either alone or in combination, have only limited efficacy in the improvement of clinical symptoms, hemodynamics, and long-term survival [7-9].

Studies have shown that there is a large overlap of up to 30% between people who have a clinical diagnosis of COPD and asthma [10]. There is also a high incidence of mild to moderate PAH prevalence, reaching

to 50% in advanced chronic obstructive COPD [11]. As Said suggested that PAH/asthma/COPD share important pathological features, including inflammation, smooth muscle contraction and remodeling [12]. Inflammation has long been acknowledged as a key feature of the asthma and COPD [1,2,6,10,11]. Perivascular inflammation has also been increasingly recognized as a significant component of clinical and experimental PAH phenotypes [13]. In these diseases there is increased resistance in, and narrowing of, airways and pulmonary arteries, respectively, due to airway and pulmonary vasoconstriction, smooth muscle constriction, and thickening of the walls caused by smooth muscle and other cell proliferation known as remodeling [12]. Muscularisation and remodeling of smaller pulmonary arteries are essential pathological lesions in PAH [14]. Airway remodeling caused by airway inflammation includes an increase in airway wall thickness, fibrosis, smooth muscle mass and vascularity, as well as abnormalities in extracellular matrix composition [11,15]. These shared pathological features suggest possible common underlying mechanism among PAH/asthma/COPD.

Thus, a further improvement in the treatment of asthma, COPD and PAH can be directed at identification of the pathobiochemical mechanisms that causally involved in the disease progression, and developing additional novel therapeutic approaches that target the various components of these multifactorial diseases.

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