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Editorial

The Role of Oxidative Imbalance on Pulmonary Diseases

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In the uncontrolled inflammatory response, reactive oxygen species (ROS) overwhelm the cellular antioxidant defense system, resulting in direct or indirect ROS-mediated damage on nucleic acids, proteins, and lipids. In the acute inflammatory process, high intracellular levels of ROS have been implicated in an impairment of resolution of inflammation and cellular injury, resulting in chronic inflammation. The imbalance due to excess oxidants reduces the ability of the cell to mount an effective antioxidant response and is implicated in inflammatory diseases from the airway and lung. In this special issue, we gathered diverse studies expanding our knowledge about the effects of ROS imbalance and/or signaling on the development or aggravation of pulmonary diseases as well as the effects of compounds with antioxidant properties on acute or chronic lung diseases.

Mitochondria are dynamic organelles responsible for energy metabolism and have a crucial role in maintaining the functions of eukaryotic cells. Furthermore, mitochondria are one of the key components involved in the production of ROS. Mitochondrial dysfunction causes increase in ROS production, inflammatory response, and induction of cellular senescence (inflammaging) in several lung inflammatory diseases such as acute lung injury, chronic obstructive pulmonary disease (COPD), pulmonary fibrosis, and bronchopulmonary dysplasia. M. A. Antunes et al. summarized that cigarette smoke induces the reduction of mitochondrial quality control and cellular stress resistance that lead to an impact on oxidative stress during COPD development and progression. C. Hou et al. described the overexpression of fatty acid synthase (FAS) in the lung tissue of mice with hypoxia-induced pulmonary arterial hypertension (PAH) and also demonstrated that the inhibition of FAS reversed hypoxia-induced lung mitochondrial dysfunction and oxidative stress. Furthermore, D. Yang et al. showed that the peptide SS-31 was able to alleviated cigarette smoke- (CS-) induced airway inflammation and ROS production through reversion of mitochondrial dysfunction.

It is known that the overproduction of ROS promotes the deterioration of the inflammatory response and causes the destruction of pulmonary microvascular cells and epithelial cells in acute lung injury (ALI)/acute respiratory distress syndrome (ARDS). H. Zhang et al. showed that the production of soluble receptor for advanced glycation end products (sRAGE) mediated by matrix metalloproteinase- (MMP-) 9 may serve as a self-limiting mechanism to control and resolve excessive inflammation and oxidative stress in sepsis-induced ALI. In addition, Y.-F. Chen et al. reported that microRNA-23a-5p (miR-23a5p) is a valuable therapeutic candidate for the treatment of ALI once it inhibits lipopolysaccharide- (LPS-) induced inflammation and oxidative stress by both the activation of apoptosis signal-regulating kinase 1 (ASK1) and heat shock protein 20 (HSP20) pathways. On the other hand, W. Jiang et al. demonstrated that endogenous miR-31-5p is a key factor for LPS-induced inflammation and oxidative damage in the lungs, through calcium-binding protein 39- (Cab-39-)

dependent inhibition of AMP-activated protein kinase α (AMPKα). Y. Liu et al. summarized that the effect of hypoxia-inducible factor-1 (HIF-1), an oxygen-dependent conversion activator, is related to ROS activity in the pathogenesis of ALI. They also propose that HIF inhibitors can be applied for the treatment of ALI. Among the several noncardiogenic factors that cause ALI is infection by the new SARS-CoV-2 virus. Here, I. G. Fernandes et al. reviewed how the ROS can affect the pathogenesis of SARS-CoV-2 and other viral infections.

Chronic lung diseases involve the activation and accumulation of inflammatory cells in the airways and lung parenchyma. The inflammatory response induces overproduction of ROS in the lungs and consequent damage to the basement membrane, evolving to the development of airway remodeling or pulmonary fibrosis. J. Ma et al. found an elevated expression of key components of the Wnt/β-catenin pathway and NOX-4 in the lungs of silicotic mice. They also showed that silicon dioxide induced the activation of the Wnt/β-catenin pathway and NOX-4, culminating in the epithelial-mesenchymal transition by lung epithelial cells. Likewise, B. Hao et al. described an increase in the expression of NOX-4 in the epithelial cells and airway smooth muscle (ASM) cells of lungs from COPD patients and in the lungs of the CS-induced emphysema mouse model. Furthermore, they showed that NOX-4-mediated ROS production participates in the TGF-β-induced differentiation of human bronchial smooth muscle cells (HBSMCs) and consequent increase in the synthesis of type I collagen by a mechanism related to activation of the p38MAPK/Akt signaling pathway in a Smad-dependent manner. Q. Wu et al. summarized the association between p53 protein and pulmonary fibrosis, providing innovative ideas to improve the prognosis, clinical diagnosis, and treatment. Finally, T. Victoni et al. and L. H. C. Vasconcelos et al. reviewed the role of oxidative imbalance in inflammatory lung diseases and bronchial asthma, respectively. They also proposed that antioxidants, alone or combined with anti-inflammatory drugs, are promising therapeutic strategies for the treatment of these diseases.

In the last decades, our understanding of the effect of oxidative imbalance on pulmonary diseases has remarkably increased. However, extensive challenges remain to provide a more comprehensive picture. The articles presented in this special issue show the complex circuitry affecting the oxidative imbalance in different pulmonary diseases and indicate future directions for the new therapeutic strategies.

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Conflicts of Interest

The editors declare that they have no conflicts of interest regarding the publication of this special issue.