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Airway Inflammation Induced by Oxidative Stress

Guest Editor:

Dr. Samuel Santos Valença

Instituto de Ciências Biomédicas,
Universidade Federal do Rio
Janeiro, Rio de Janeiro 21941-
590, RJ, Brazil

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Message from the Guest Editor

Oxidative inactivation of antiproteinases, airspace epithelial injury, increased sequestration of leukocytes in the pulmonary microvasculature, and gene expression of proinflammatory mediators are important events in the pathogenesis of lung diseases. Epithelial cells in the lungs are uniquely vulnerable to oxidative damage due to their potential for exposure to both endogenous and exogenous oxidants. Lung cells use several nonenzymatic and enzymatic antioxidant mechanisms to protect against oxidative insult through redox-sensitive transcription factors such as Nrf2, NF- κ B and AP-1, which regulate genes for proinflammatory mediators and protective mechanisms. Although antioxidant drugs could play a useful role in the therapy of inflammatory lung diseases, their clinical impact is relatively modest at present.

This Special Issue aims to publish manuscripts that will explore all aspects of airway inflammation induced by oxidative stress by focusing on the pathophysiology mechanism of several stimuli and lung diseases. Original papers describing recent discoveries in the field of lung redox biology and inflammation are welcome, as well as reviews from experts in this field.



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Special Issue



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Editor-in-Chief

**Prof. Dr. Alessandra
Napolitano**

Department of Chemical
Sciences, University of Naples
"Federico II", Via Cintia 4, I-80126
Naples, Italy

Message from the Editor-in-Chief

It has been recognized in medical sciences that in order to prevent adverse effects of "oxidative stress" a balance exists between prooxidants and antioxidants in living systems. Imbalances are found in a variety of diseases and chronic health situations. Our journal *Antioxidants* serves as an authoritative source of information on current topics of research in the area of oxidative stress and antioxidant defense systems. The future is bright for antioxidant research and since 2012, *Antioxidants* has become a key forum for researchers to bring their findings to the forefront.

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Antioxidants Editorial Office
MDPI, Grosspeteranlage 5
4052 Basel, Switzerland

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