Special Issue

Airway Inflammation Induced by Oxidative Stress

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 redoxsensitive transcription factors such as Nrf2, NF-kB 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.

Guest Editor

Dr. Samuel Santos Valença

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

Deadline for manuscript submissions

closed (10 January 2023)



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Antioxidants
Editorial Office
MDPI, Grosspeteranlage 5
4052 Basel, Switzerland
Tel: +41 61 683 77 34
antioxidants@mdpi.com

mdpi.com/journal/ antioxidants





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About the Journal

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.

Editor-in-Chief

Prof. Dr. Alessandra Napolitano

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

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