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Oxidative Stress in Chronic Obstructive Pulmonary Disease

Guest Editor:

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Message from the Guest Editor

Chronic obstructive pulmonary disease (COPD), caused by exposure to noxious irritants including cigarette smoke (CS), is a progressive inflammatory disease characterized by persistent respiratory symptoms and airflow limitation. Under normal conditions in the respiratory tract, it is now appreciated that ROS play a variety of physiological roles in signal transduction, injury-repair response, ECM remodeling, and pathogen clearance. In a pathologic conditions, ROS causes cell injury, mitochondrial dysfunction, formation, and release of toxic metabolites and even DNA damage. To protect themselves from ROS, cells contain antioxidant defenses including superoxide dismutase, catalase, and glutathione peroxidase to neutralize ROS. When ROS production exceeds antioxidant capacity, proteins, lipids, and nucleic acids become oxidized and often trigger an adaptive stress response. Moreover, crosstalk among oxidative stress, protease, and inflammation, which is called pathogenic triad, has been recently suggested to potentiate their deleterious effects. However, there are still many issues which need to be clarified in the pathogenic mechanism of oxidative stress in COPD.

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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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