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Oxidative Stress and Antioxidants in Hypoxia and Human Pathophysiology Settings: Novel Pharmacological Targets

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Message from the Guest Editor

Hypoxic (HI) injury is defined as the worsening of organ/cellular dysfunction and cell death following reduction in blood flow due to organ targeting or increases in oxygen consumption in a related tissue. Reperfusion itself causes reversible and irreversible changes in tissue viability and organ function, the basic pathophysiology of ischemia-reperfusion (IR) injury, especially oxidative stress, and cell death mechanism. When the blood supply is re-established, local inflammation and oxidative stress production increase, leading to secondary injury. This Special Issue is focused on the following topics:

Current concepts of pathophysiology and therapies in cardiac HI and pharmacological preconditioning;

Mechanisms of liver preconditioning in animal and clinical models of HI and IR injury;

Current concepts of pathophysiology and therapies in cerebral HI and IR injury;

Ex vivo models to reduce HI injury in organs for transplantation: role of antioxidants;

Role of hypoxia in cardiovascular programing: mechanisms and potential therapeutic target with antioxidants;

Role of microRNAs in the regulation of cardiac HI injury: animals and clinical settings.

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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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