Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

# Oxidative Stress and Vascular Dysfunction in Myocardial Ischemia

CALL FOR PAPERS

Oxidative stress has been rigorously studied for many years and represents a very interesting and important area, crucial for both physiological and pathophysiological and clinical aspects. It has come to light that reactive oxygen species play major roles in normal physiological functions (contributing, for instance, to cellular and molecular signaling pathways), as well as in disorders and therapeutic strategies. Oxidative stress is one of the crucial factors and mechanisms regulating vascular function and dysfunction and contributes to a wide range of changes of vascular function and structure. The complex and convoluted mechanism linking oxidative stress and vascular (dys)function from a molecular and cellular level to tissue and organ levels, with pathophysiological and functional consequences, represents a major challenge. The understanding of these interactions is, however, crucial if we want to devise effective therapeutic strategies for some of the most widespread disorders of vascular structure and function.

Myocardial ischemia occurs when blood flow to myocardial cells is reduced, preventing them from receiving enough oxygen to meet their metabolic and functional demands. It represents one of the most relevant health problems in the modern world. Although there have been many therapeutic and diagnostic advances in recent decades, the understanding of the pathophysiology of myocardial ischemia, as well as the therapeutic results, is still not satisfactory and faces multiple challenges.

The proposed special issue focuses on oxidative stress as a physiological and pathogenetic factor in vascular regulation and vascular dysfunction in myocardial ischemia. This is a topic of high current interest, since it is becoming more and more evident that oxidative stress plays important roles and takes part in multiple pathogenetic mechanisms in vascular dysfunction during myocardial ischemia. It may therefore be a focus of modern therapeutic strategies. The proposed issue would cover the most recent research findings on endothelial dysfunction, changes in conducted vasomotor response, microvascular reactivity, and other functional in vivo and in vitro studies of myocardial ischemia, as well as molecular and genetic cutting-edge research (in humans and animal models) elucidating the role of oxidative stress in vascular dysfunction as a cause and perpetuating factor of myocardial ischemia. Clinical, preclinical, and translational studies are welcome, as well as pertinent reviews with a critical synthesis of relevant published data. The issue also encourages submissions of investigations of oxidative stress as a therapeutic target in myocardial ischemia (especially with a focus on vascular dysfunction).

Potential topics include but are not limited to the following:

- Pathophysiology of oxidative stress as a cause and perpetuating factor of vascular dysfunction in myocardial ischemia
- Genetic and molecular basis and mechanisms of oxidative stress in myocardial ischemia
- Oxidative stress as a therapeutic target in myocardial ischemia
- Association of reactive oxygen species with endothelial dysfunction, microvascular reactivity changes, and changes in conducted vasomotor responses in animal experimental models in vitro and in vivo
- Oxidative stress in endothelial macrovascular dysfunction and microvascular reactivity changes in clinical studies, including human patients with myocardial ischemia
- Role of oxidative stress in molecular and morphological changes with potential implications for vascular function
- Translational studies of the role of oxidative stress in vascular dysfunction during myocardial ischemia
- Other relevant and cutting-edge researches of oxidative stress connected with vascular function changes in acute and chronic myocardial ischemia

Authors can submit their manuscripts through the Manuscript Tracking System at https://mts.hindawi.com/submit/journals/omcl/trosvd/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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