Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Interplay of Oxidative Stress, Calcium Mishandling, and Mitochondrial Bioenergetics in the Cardiac Function

CALL FOR PAPERS

The prevalence and the incidence of cardiac diseases are rising worldwide and remain the leading cause of death. Growing lines of evidence identify Ca^{2+} mishandling, redox imbalance, and impaired mitochondrial bioenergetic as key factors in the development of cardiac diseases. In the heart, cytosolic Ca^{2+} oscillations initiate cardiac contraction during excitation-contraction coupling. The extrusion of Ca^{2+} out of the cytoplasm by the Na^{+}/Ca^{2+} exchanger and recapturing into the sarcoplasmic reticulum by a Ca^{2+} ATPase participate in the cardiomyocyte relaxation. A fraction of this Ca^{2+} is also taken up by mitochondria, which produce 95% of the energy in form of ATP of the cell. In addition, the mitochondria represent the primary source of reactive molecules involved in redox signaling, known to induce Ca^{2+} mishandling. Therefore, mitochondrial dysfunction can compromise the energy supply for the contractile machinery and accelerate the development of cardiac dysfunction by production of reactive species. Thus, mechanisms that improve mitochondrial function are essential for cellular homeostasis and will help restore the cardiac function of cardiomyopathies.

While, in the last years, the interest in understanding the role of myocardial energy metabolism in health and disease has grown, the regulation of mitochondrial signaling and metabolism during excitation-contraction coupling still remains elusive. In this issue, we invite investigators to contribute original research articles as well as review articles to increase our understanding of the mitochondrial bioenergetics, function, and excitation-contraction mechanisms in health and disease. We encourage submissions of basic, translational, and clinical studies describing signaling mechanisms and novel approaches to treat cardiac dysfunction.

Potential topics include but are not limited to the following:

- Redox regulation of the cardiac excitation-contraction coupling during physiological and pathological conditions
- Cardiac redox signaling and oxidative stress in heart failure, ischemia reperfusion, diabetic cardiomyopathy, and hormone regulation
- Cardiac mitochondrial dynamics: biogenesis, fusion, and fission in pathophysiology
- Mitochondrial bioenergetics in cardiomyopathy (heart failure, hypertrophy, and diabetes)
- New therapeutic strategies targeting mitochondria and oxidative stress

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

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