Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Mitochondria at the Crossroads of Survival and Demise

CALL FOR PAPERS

Mitochondria are multifunctional organelles and their structural and functional integrity is fundamental to cell life. In addition to their critical role in the production of ATP via oxidative phosphorylation and biosynthetic intermediates, mitochondria are also a major hub for cellular Ca2+ signaling. They decode Ca2+ signals received from the endoplasmic reticulum into specific inputs to regulate essential functions including metabolism and energy production. However, mitochondria can actively or passively drive cellular dysfunction or demise. They can become the major source of reactive oxygen species in pathological processes and reversely they are highly vulnerable to damage. Thereby, mitochondria represent a point of convergence for a variety of upstream cell death stimuli and undergo structural and functional remodeling with subsequent transmission of signals to downstream executioner proteins. The intrinsic pathway includes death stimuli like lack of dioxygen, metabolic perturbation, deprivation of survival factors, oxidative stress, Ca2+ overload, DNA damage, proteotoxic stress, and oncogene activation. In response to this diversity of stimuli mitochondria are central to several different modes of regulated cell death like apoptosis and necrosis. Initial events in mitochondria leading to apoptosis are the permeability transition in the inner membrane leading to outer membrane rupture and the permeabilization of the outer membrane permitting the release of apoptogenic factors from the mitochondrial intermembrane space. Necrosis is characterized by mitochondrial membrane depolarization, decreased ATP levels, cellular and organellar swelling, and loss of plasma membrane integrity. These signaling pathways are highly orchestrated as well as integrated at the molecular level and overlap substantially with respect to disease. Based on their pathophysiological importance, mitochondria have attracted broad scientific and clinical interest as an important target for intervention in disease.

We invite investigators to contribute original research articles as well as review articles that will advance our understanding of the mitochondria behavior and function in response to oxidative stress in disease/disorders such as cardiovascular and neurodegenerative diseases, which could be important for the development of new mechanism-based pharmacotherapeutic strategies to treat many human diseases.

Potential topics include but are not limited to the following:

- Regulation of mitochondrial energy production in health and disease progression
- Involvement of mitochondrial perturbation in regulated cell death
- Mitochondrial outer and inner membrane permeabilization: structure and assembly of the pores
- Calcium overload and mitochondrial damage
- Endoplasmic reticulum-mitochondria tethering: the role of MAMs (mitochondria-associated membranes) in health and disease progression
- Physical composition of mitochondria and their physical location within the cell in health and disease progression
- Role of mitochondrial reverse electron transport in ROS signaling in health and disease progression

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

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

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