Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Dietary Compounds and Mitochondria-Related Redox Signaling

CALL FOR PAPERS

Mitochondria are double-membrane organelles responsible for the production of ATP through the oxidative phosphorylation system in mammalian cells. Furthermore, mitochondria are a source of reactive species physiologically. Electron leakage from the mitochondrial electron transfer chain (METC) plays a role in the generation of reactive oxygen species (ROS). Moreover, reduced levels of mitochondria-located antioxidant defenses favor oxidative damage in mitochondrial components, as, for instance, membrane lipids and enzymes involved in the maintenance of mitochondrial functions. In some pathological conditions, the production of reactive species is enhanced in mitochondria, leading to oxidative and nitrosative damage in mammalian tissues. In this regard, mitochondrial medicine becomes an interesting strategy in order to achieve mitochondrial protection against impairments resulting from both endogenous and exogenous sources.

Natural compounds play an important role in modulating transcription of enzymes involved in the maintenance of the mitochondria-related redox environment and also regulate mitochondria-associated signaling, for example, by inducing or repressing the intrinsic apoptotic pathway, which is dependent on mitochondria to occur. Additionally, natural compounds may trigger mitochondrial biogenesis, improving cellular bioenergetics and counteracting dysfunctions induced by several agents in mammalian cells. Therefore, mitochondrial function, dynamics, and redox-related signaling may be modulated by natural compounds, causing cell survival and enhancing longevity. In this context, synthetic molecules and nanotechnology-related strategies have been also developed focusing on mitochondria, but further research is necessary to better understand whether and how such alternative agents impact mitochondria and cellular viability. In addition, mitochondrial toxicity needs special attention due to the impacts resulting from disturbances in the organelle function.

In this special issue, we are interested in manuscripts dealing with investigations involving the consequences of mammalian cells exposure to natural compounds regarding mitochondria function, dynamics, and signaling pathways associated with the organelle. Both *in vitro* and *in vivo* experimental models are welcome. Original papers, perspectives, and review manuscripts may be submitted.

Potential topics include, but are not limited to:

- Mitochondrial function and related signaling pathways
- Mitochondrial dynamics: mitochondrial biogenesis, fusion, and fission
- Mitochondria-related redox environment
- Interactions of mitochondria with other organelles
- Mitochondrial dysfunction
- Methods to access mitochondrial function and dynamics
- Future strategies to treat mitochondrial dysfunction

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

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