



# Oxidative Medicine and Cellular Longevity

## Special Issue on **Mitochondrial Bioenergetics and Quality Control Mechanisms in Health and Disease**

# CALL FOR PAPERS

Since the first endosymbiotic event occurred, where a proteobacterium was engulfed by larger cells, evolutionary pressure was imposed into mitochondria in order to facilitate the higher energy output required for the evolution of complexity. Therefore, mitochondria became the powerhouse of most eukaryotic cells and the main source of reactive molecules involved in both redox signaling (physiological events) and oxidative stress (pathological events). Despite their central role in ensuring that tissue energy demands are met by energy supply, mitochondria have been evolved in a wide range of intercellular and intracellular processes such as controlling nuclear gene expression, ions homeostasis, and apoptosis. Thus, mechanisms of surveillance and quality control capable of maintaining mitochondrial integrity and functionality (not only as an intracellular power plant) are critical for cellular homeostasis. In general, mitochondrial quality is controlled by a myriad of interconnected systems including: (a) enzymatic and nonenzymatic elements capable of fighting oxygen-mediated mitochondrial toxicity; (b) mitochondrial proteases and chaperones responsible for the maintenance of mitochondrial proteostasis; and (c) a multilayer network of proteins involved in the control of mitochondrial morphology, location, and number. Disruption of mitochondrial quality control mechanisms in general results in adverse effects that contribute to the establishment and progression of several diseases. Therefore, development of pharmacological and nonpharmacological approaches capable of optimizing mitochondrial surveillance and quality control mechanisms are promising tools to treat diseases.

In this special issue, we invite investigators to contribute their original research as well as review article to broaden our understanding of the regulatory processes involved in mitochondrial bioenergetics, surveillance, and quality control mechanisms in health and disease. We encourage submission of basic, translational, and clinical studies describing signaling mechanisms and novel approaches for diagnostics and therapeutics according to the proposed topic.

Potential topics include, but are not limited to:

- ▶ Redox signaling and oxidative stress
- ▶ Mitochondrial proteostasis
- ▶ Mitochondria-nucleus communication (mitochondrial retrograde signaling)
- ▶ Mitochondrial dynamics: mitochondrial biogenesis, fusion, and fission
- ▶ Mitochondrial clearance (mitophagy)
- ▶ Mitochondrial bioenergetics

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

### **Lead Guest Editor**

Julio C. B. Ferreira, University of São Paulo, São Paulo, Brazil  
[jcesarbf@usp.br](mailto:jcesarbf@usp.br)

### **Guest Editors**

Xin Qi, Case Western Reserve University, Cleveland, USA  
[xxq38@case.edu](mailto:xxq38@case.edu)

Suresh S. Palaniyandi, Henry Ford Health System, Detroit, USA  
[spalani2@hfhs.org](mailto:spalani2@hfhs.org)

### **Manuscript Due**

Friday, 14 October 2016

### **First Round of Reviews**

Friday, 6 January 2017

### **Publication Date**

Friday, 3 March 2017