

Special Issue on **Mitochondrial Dysfunction and Production of Reactive Oxygen Species in Obesity-Associated Diabetes and Hypertension**

CALL FOR PAPERS

Mitochondria are unique and highly specialized organelles providing cellular adenosine triphosphate (ATP) through oxidative phosphorylation. Mitochondria can undergo dramatic changes in morphology in metabolic inputs by adapting the balance between mitochondrial fusion and fission. Despite being the major ATP producer in mammalian cells, mitochondria are a physiological source for the generation of reactive oxygen species, such as superoxide anion radical ($O_2^{\cdot-}$) and hydrogen peroxide (H_2O_2), through the components of the respiratory chain and a number of redox enzymes. This production is counterbalanced by potent antioxidative defense systems. Mitochondria are central to all basic and advanced cellular functions by regulating the energy metabolism and by regulating ROS production; they also act as signalling hubs, essential for cells survival. Metabolic pathologies such as obesity, diabetes, and metabolic syndrome are closely connected with changes in mitochondrial dynamics and function. Indeed, mitochondrial dysfunction is implicated in all common diseases ranging from cardiovascular and neuromuscular diseases to obesity-related diabetes and cancer.

In this special issue, we proudly invite researchers to submit original research as well as review manuscripts exploring the current knowledge on the role of mitochondria function during obesity-associated type 2 diabetes, hypertension, and metabolic syndrome. Studies may include organs such as, but not limited to, the adipose tissue, heart, liver, kidney, brain, and muscle.

Potential topics include but are not limited to the following:

- ▶ Role of obesity-related inflammation on mitochondrial structure, function, and/or dynamics
- ▶ Role of obesity-related type 2 diabetes on mitochondria structure, function, and/or dynamics
- ▶ Regulation of energy metabolism and the production of reactive oxygen species
- ▶ Tissue-specific mitochondrial dysfunction across various organs in obesity-related diabetes and hypertension
- ▶ Mitochondrial dysfunction resulting from obesity-related diabetes and hypertension
- ▶ Mitochondrial dysfunction in metabolic syndrome
- ▶ The role of mitophagy in obesity-related type 2 diabetes
- ▶ Mitochondrial redox biology and bioenergetics in obesity and type 2 diabetes
- ▶ Mitochondria-related signalling pathways in obesity and type 2 diabetes
- ▶ Cell survival versus cell death: role of mitochondrial function
- ▶ Methodologies to monitor and evaluation of mitochondrial reactive oxygen species production

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

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

Lead Guest Editor

Aurelie N. D. Cat, Cordeliers Research Centre, Paris, France
cattuong.ndc@gmail.com

Guest Editors

Rita Tostes, University of Sao Paulo, Ribeirao Preto, Brazil
rtostes@usp.br

Victoria Cachofeiro, Universidad Complutense de Madrid, Madrid, Spain
vcara@ucm.es

Malou Friederich-Persson, Uppsala University, Uppsala, Sweden
malou.friederich@mcb.uu.se

Submission Deadline

Friday, 27 October 2017

Publication Date

March 2018