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Mitochondrial Dysfunction in Neurological Disorders: Molecular Mechanisms and Potential Points for Intervention

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Message from the Guest Editors

Dear Colleagues,

Mitochondria play a major role in the regulation of cellular bioenergetics as well as various non-energetic processes. like cell death mechanisms, generation of free radicals and antioxidative defense, immune response, and regulation of calcium homeostasis. Therefore, abnormal mitochondria signaling may be an important link between various disease-associated factors common to, and critical in, different central nervous system (CNS) diseases: from neurodevelopmental disorders, that occur early in life and persist until adulthood, to age-related neurodegenerative diseases. Understanding the exact position mitochondrial dysfunction in the deleterious feed-forward loop critical for the development and progression of CNS diseases may help design successful therapeutic strategies for several fatal syndromes.

In this Special Issue, we cordially invite you to contribute, either in the form of original research articles, or reviews on all aspects related to the topic, describing mechanistic, functional, cellular, biochemical, or general aspects of mitochondrial dysfunction in neurological disorders.













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Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. Cells encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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