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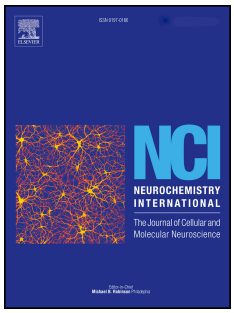

Mitochondria in the nervous system: From Health to disease, part II

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Mitochondria in the Nervous System: From Health to Disease, Part II

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Abstract

In Part II of this Special Issue on "Mitochondria in the Nervous System: From Health to Disease", the editors bring together more reviews and original articles from researchers in the field of mitochondrial metabolism in the healthy and diseased nervous system. Subjects span from basic mitochondrial physiology to papers on mitochondrial dynamics and to those altered states of the nervous system that can be considered “mitopathologies”. Finally, a few papers approach aspects of mitochondrial biology linked to the feasibility and validity of a mitochondrial therapy.

1. Introduction

Because of their physical and functional association with crucial pathways in energy metabolism, mitochondria are classically considered the “powerhouse of the cell”. Following studies on a number of mitochondria-linked human diseases, our modern view of these organelles has evolved and expanded, assigning them a plethora of other functions regulating the life and death of a cell, be it in differentiation and development or in ageing and disease. This second volume of the Special Issue on “Mitochondria in the Nervous System: From Health to Disease” brings together 17 articles, with some covering aspects of basic mitochondrial function and dynamics, and others focusing on the role played by alterations of these organelles in neurological diseases. Such alterations might constitute a target for therapy, provided that we first succeed in fully elucidating how the complex mitochondrial molecular network is regulated.

2. Physiological mitochondrial functions in the CNS

Christos Chinopoulos (Chinopoulos 2017) opens this issue with a critical review on what we know - or thought we knew - on the structure of the mitochondrial permeability transition (mPT) pore. The mPT pore is a megachannel of the inner mitochondrial membrane allowing a non-selective flux of metabolites with a molecular weight of up to 1.5 kDa (Crompton, 1999). Its prolonged opening leads to severe mitochondrial impairment causing cell death in a final pathway that is common to major disease conditions. The c-subunit rings of the ATP synthase complex (Alavian et al., 2014) or the interface between ATP synthase dimers (Giorgio et al., 2013) have been proposed