Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Oxidative Stress and Mitochondrial Damage in Neurodegenerative Diseases: From Molecular Mechanisms to Targeted Therapies

CALL FOR PAPERS

Oxidative stress has been implicated in the pathogenesis of several neurodegenerative diseases, including Alzheimer's disease (AD), Parkinson's disease (PD), and amyotrophic lateral sclerosis (ALS). These neurodegenerative disorders are normally characterized by progressive loss of neuronal cells and compromised motor or cognitive functions. It has been shown that neuronal cells are particularly vulnerable to oxidative damage due to their high polyunsaturated fatty acid content in membranes, high oxygen consumption, and weak antioxidant defense. Alteration (or negative regulation) of intracellular redox state leads to the overproduction of reactive oxygen species (ROS), which can induce mitochondrial DNA mutations, damage the mitochondrial respiratory chain complexes, alter membrane permeability, influence Ca^{2+} homeostasis, and challenge mitochondrial quality control and mitochondrial defense systems. All these changes are implicated in the development of neurodegenerative diseases, mediating or amplifying neuronal dysfunction and triggering neurodegeneration. However, the exact molecular pathogenesis of neurodegeneration related to the disruption of redox balance remains unclear. The progress in the development of neuroprotective therapies has been hindered, because it is difficult to define the right targets for treatment and what should be considered as an efficient neuroprotective agent (or medicament). Therefore, in the past years, the attention was focused on researches about pharmacological targets that could protect neurons against oxidative stress. Novel antioxidants have shown great potential in mediating disease phenotypes and several potential drugs have been screened in in vitro and in vivo models of neurodegenerative diseases. Although the outcomes from these studies are not yet translated in benefit for the patients and disappointing results were obtained from numerous clinical trials, the area of targeting oxidative stress in neurodegenerative diseases could be of great interest for further research.

We invite investigators to contribute to this special issue with original research articles as well as reviews addressing the role of oxidative stress in neurodegeneration. We encourage submissions of manuscripts aimed at elucidating the molecular and cellular mechanisms linking the disruption of intracellular redox status with the development of neurodegenerative diseases. We are particularly interested in articles covering the identification of new relevant targets regulated by redox signaling as well as new potential antioxidant therapy or alternative ways to prevent or control free radicals generation.

Potential topics include but are not limited to the following:

- Updates about the role of mitochondrial dysfunction, ER stress, and oxidative stress in the onset and propagation of neurodegenerative diseases
- Mitochondrial dysfunction in aging-related neurodegenerative diseases: causes or consequences
- Recent advances in antioxidant prevention in pharmacology and treatments against mitochondrial failure of neurodegenerative diseases
- Oxidative stress and its impact on neurons and glia
- Experimental models to study the role of oxidative stress and mitochondrial dysfunction in neurodegeneration and neurodegenerative diseases
- Potential role of mitochondrial damage in the pathogenesis of neurodegenerative disorders
- Oxidative stress and mitochondrial failure in neurodegeneration

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

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

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Submission Deadline
Friday, 5 April 2019

Publication Date
August 2019