Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Oxidative Stress: Neuropathy, Excitability, and Neurodegeneration

CALL FOR PAPERS

Energy production by aerobic cellular metabolism generates free radicals. Imbalance between the production of these radicals and their elimination via antioxidant defenses lead to oxidative stress. The toxicity of free radicals contributes to proteins and DNA injury, inflammation, tissue damage, and subsequent cellular apoptosis. Due to their very high demands in energy, large numbers of mitochondria, reduced capability of limiting glucose uptake, weak antioxidant defense, and weak bioavailability to antioxidant treatment and molecules, neurons are highly susceptible to reactive oxygen species- (ROS-) mediated damage. Alongside environmental and/or genetic factors, oxidative stress (OS) leads to a range of disorders such as Amyotrophic Lateral Sclerosis, Alzheimer's disease, and Parkinson's disease as well as chemotherapy- and diabetic-induced neuropathy. Thus, reduced antioxidant defense, overproduction, or incorporation of free radicals from the environment into the system leads to neurodegeneration. For example, diabetes-induced hyperglycemia enhanced neuronal ROS levels, leading to damaged proteins, decreased biological activity, reduced energy, impaired signaling, and protein trafficking which ultimately lead to neuronal death characteristic of diabetic neuropathy.

We invite investigators to contribute to this special issue with original research articles as well as reviews addressing the physiology, the mechanisms, and the impact of oxidative stress in neuron excitability, neurodegeneration, and neuropathy. The potential of antioxidant therapy or alternative ways to recess or control free radicals generation is also of interest. While this topic has been extensively studied, recent advances linking excitability, oxidative stress, and degeneration emerged, and such cascade appears as a major driver in CNS pathology. A particular attention will therefore be given on manuscript exploring or discussing the mechanism of neuronal excitability and oxidative stress.

Potential topics include but are not limited to the following:

- Mechanism leading to oxidative stress; physiology; and pathology
  - Mitochondrial dysfunction
  - Hyperglycemia
  - ER-stress
  - Chemotherapy
  - Nerve injury
- Consequences of increased oxidative stress on neuron function
  - Diabetes-related neurological implications
  - Hyperexcitability
  - Small fiber Neuropathy
  - Neurodegeneration
- Potential treatment
  - Antioxidant

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

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

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Submission Deadline
Friday, 30 March 2018

Publication Date
August 2018