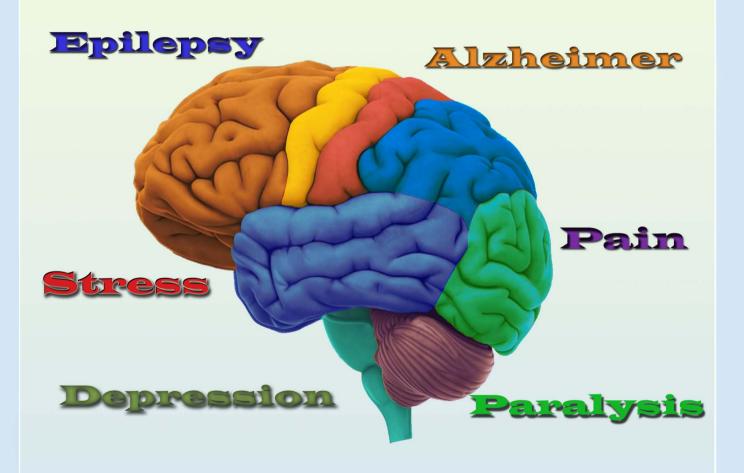
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AIM AND SCOPES

Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of physiological and pharmacological biophysical, processes that regulate cellular function, and the control or alteration of these processes by the action of receptors, neurotransmitters, second messengers, cation, anions, drugs or disease.

Areas of particular interest are four topics. They are;

A- Ion Channels (Na⁺- K⁺ Channels, Cl⁻ channels, Ca²⁺ channels, ADP-Ribose and metabolism of NAD, Patch-Clamp applications)

B- Oxidative Stress (Antioxidant vitamins, antioxidant enzymes, metabolism of nitric oxide, oxidative stress, biophysics, biochemistry and physiology of free oxygen radicals)

C- Interaction Between Oxidative Stress and Ion Channels in Neuroscience

(Effects of the oxidative stress on the activation of the voltage sensitive cation channels, effect of ADP-Ribose and NAD+ on activation of the cation channels which are sensitive to voltage, effect of the oxidative stress on activation of the TRP channels in neurodegenerative diseases such Parkinson's and Alzheimer's diseases)

D- Gene and Oxidative Stress

(Gene abnormalities. Interaction between gene and free radicals. Gene anomalies and iron. Role of radiation and cancer on gene polymorphism)

READERSHIP

Biophysics Biochemistry

Biology **Biomedical Engineering** PhysiologyGenetics Pharmacology

Cardiology Neurology Oncology Psychiatry

Neuroscience Neuropharmacology

Keywords

Ion channels, cell biochemistry, biophysics, calcium signaling, cellular function, cellular physiology, metabolism, apoptosis, lipid peroxidation, nitric oxide, ageing, antioxidants, neuropathy, traumatic brain injury, pain, spinal cord injury, Alzheimer's Disease, Parkinson's Disease.

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Oral Presentations



Oral Presentation 10

Chemotherapeutic agents increase mitochondrial oxidative stress and apoptosis in optic nerve

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Chemotherapeutic agents such as cisplatin and 5fluorouracil are very effective and commonly used chemotherapeutic agents in treatment of several cancers including breast, testicular, ovarian and lung cancers. However, they have adverse effects and apoptosis in normal cells and neurons including optic nerve (Cardellicchio et al. 2014). Oxidative stress occurs during the several physiological functions such as mitochondria and phagocytosis. If the products of oxidative stress such as superoxide radical and hydrogen peroxide will be controlled by antioxidants the cell injury in normal tissue will not be occur. Results of recent reports indicated that optic nerve injury was induced through excessive production of reactive oxygen species (ROS) in rats by chemotherapeutic agents, although ROS were scavenged by antioxidants such as pycnogenol and rutin (Icel et al. 2018; Taşlı et al. 2018). It seems that the chemotherapeutic agentsinduced excessive ROS production results in increased levels of lipid peroxidation as malondialdehyde and inflammation markers such as TNF-α and NF-κB levels, but decrease of glutathione and total antioxidant levels. Apoptosis in the optic nerve was induced in ARPE19 eye cells by activation of intrinsic apoptosis pathway and death receptor signaling (Güçlü et al. 2018). In the presentation, we discussed novel effects of oxidative stress and apoptosis on the optic nerve injury in rodents

and human.

The results of current data suggest that oxidative stress has a main role in chemotherapeutic agents-induced optic nerve injury in rodents, although the injury was attenuated by the antioxidant treatment.

Keywords: Antioxidants; Apoptosis; Inflammation; Optic nerve injury; Oxidative stress.

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