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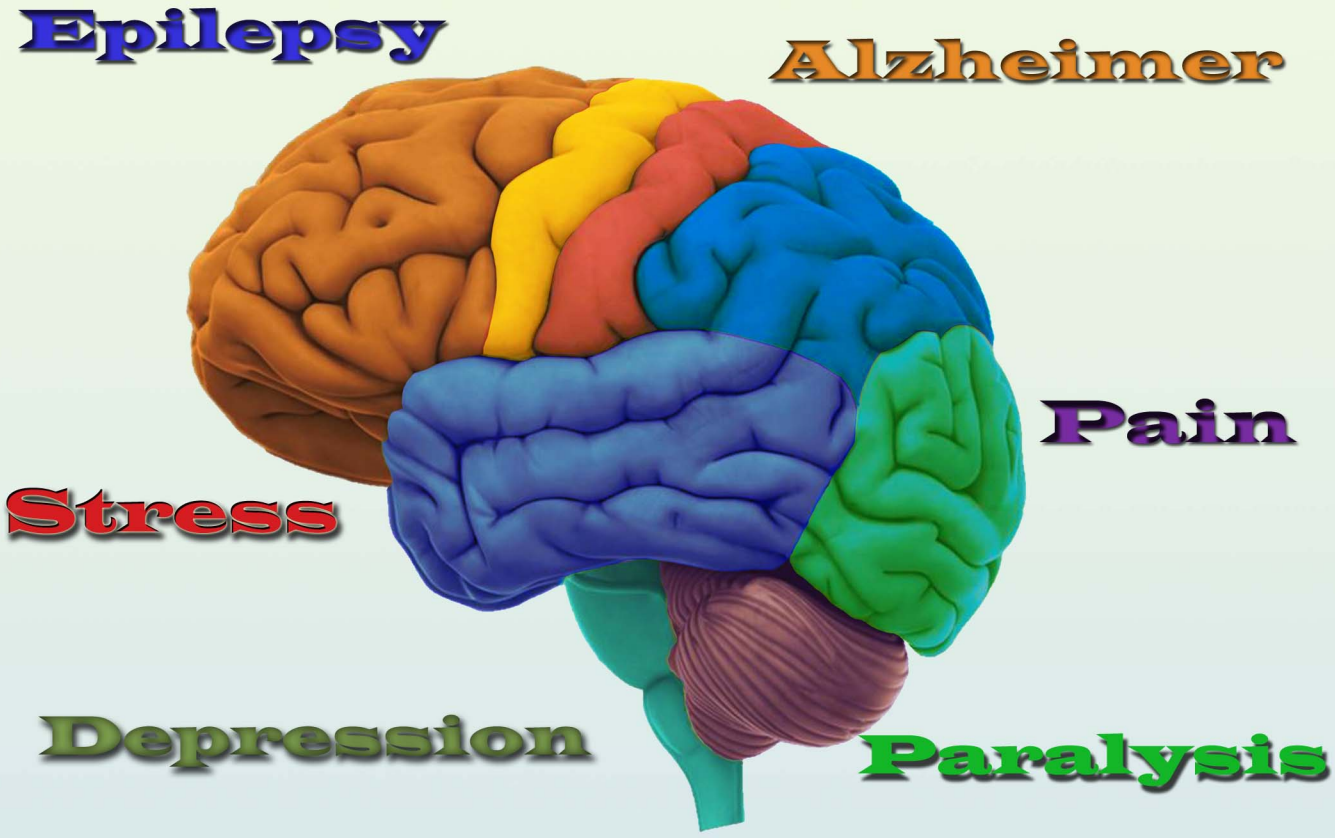

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AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate 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, Ca^{2+} 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
Pharmacology	PhysiologyGenetics
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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Isparta, Turkey

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BSN Health Analyses, Innovation, Consultancy, Organization, Industry and Trade Limited Company

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Oral Presentations

Oral Presentations

Oral Presentation 7

Potential therapeutic role of melatonin in traumatic brain injury: A literature review

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Oxidative stress induces excessive production of reactive oxygen species (ROS). ROS are including several free oxygen radicals such as singlet oxygen and superoxide radical. Excessive ROS production induces injuries of lipids, nucleic acids and proteins in several cells. Brain and neurons have a high amount of polyunsaturated fatty acids (PUFAs) and consumption of oxygen, but they have low level of antioxidant. Oxidative stress is controlled by several enzymatic and non-enzymatic antioxidants. One of the main non-enzymatic antioxidant is melatonin. Melatonin is secreted from the pineal gland by physiological circadian cycles. It has several physiological functions such as mediator of circannual reproductive rhythms (Tamtaji et al. 2019). However, it has also a regulatory role in the pathophysiological pathways of traumatic brain injury (TBI) in human and rodents (Barlow et al. 2019). TBI is one of the most common causes of the mortalities. Secondary events occur after primary events like shearing of nerve cells and blood vessels, cause posttraumatic neurodegenerations with an increase in ROS and ROS-mediated lipid peroxidation. It was reported that TBI-induced oxidative stress in experimental TBI was inhibited by the melatonin treatment (Senol and Nazıroğlu, 2014). Results of a recent study indicated protective role of melatonin through inhibition of Nrf2 signaling pathway, inflammation and oxidative stress in TBI-induced mice (Wang et al. 2019). In human studies, behavioral outcomes of TBI were modulated by the melatonin treatment (Barlow et al. 2019). In the oral presentation, I will review recent studies on TBI in human and experimental animals.

In conclusion, there are pre-clinical and clinical evidences that melatonin treatment after TBI significantly improves both behavior-cognition outcomes and pathophysiological outcomes such as oxidative stress and inflammation. It seems that the certain interaction between melatonin and TBI still remain to be determined.

Keywords; Behavior; Melatonin; Traumatic brain injury; Oxidative stress; Inflammation.

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