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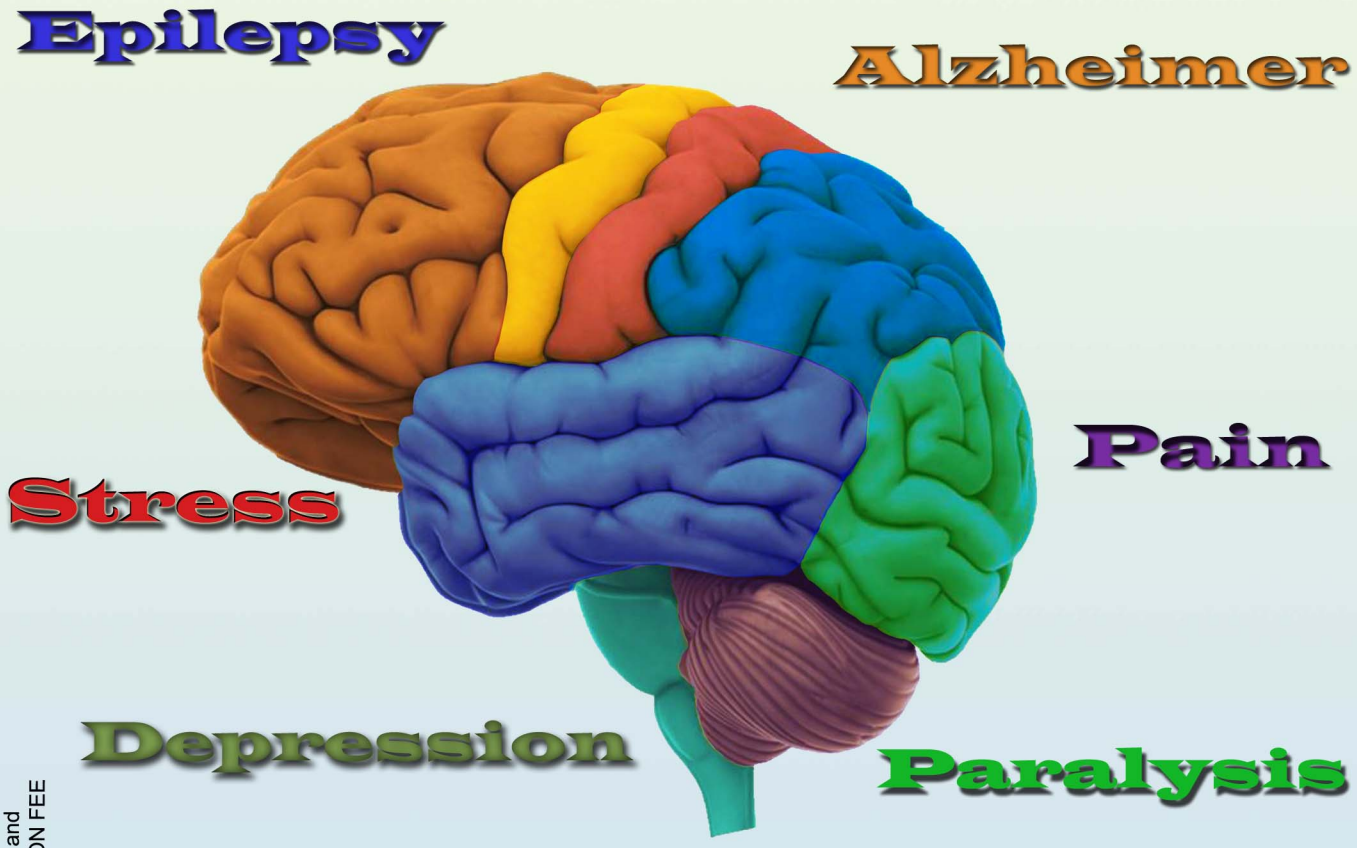

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AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate 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)

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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 Presentation 12

Role of melatonin on oxidative stress in traumatic brain injury

Yener AKYUVA
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Oxidative stress occurs in the several physiological processes such as phagocytic activity and mitochondrial membrane functions. Oxidative stress is controlled by several enzymatic and non-enzymatic antioxidants. Traumatic brain injury 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. Melatonin is a member of non-enzymatic antioxidant group. The protective effects of melatonin on traumatic brain injury have been shown in vivo and in vitro studies (Barlow et al. 2018). Also melatonin has been shown to counteract oxidative stress-induced pathophysiologic conditions like ischemia/reperfusion injury, neuronal excitotoxicity and chronic inflammation. Recently, it was reported that TBI-induced oxidative stress in experimental TBI was inhibited by the melatonin treatment (Senol and Nazıroğlu, 2014). In the oral presentation, I will review recent studies on traumatic brain injury in human and rodents.

I concluded that the oxidative stress causes changes through activation of second messengers, which may lead to the pathology of TBI, although melatonin has protective effects on the pathology. It seems to that the exact relationship between melatonin and TBI still remain to be determined.

Key words; Melatonin; Hipocampus; Traumatic brain injury; Oxidative stress; Antioxidants.

References

Barlow K, Esser MMJ, Veidt M, Boyd R. 2018. Melatonin as a treatment after Traumatic Brain Injury: A systematic review and meta-analysis of the pre-clinical and clinical literature. J Neurotrauma. doi: 10.1089/neu.2018.5752.

Senol N, Nazıroğlu M. 2014. Melatonin reduces traumatic brain injury-induced oxidative stress in the cerebral cortex and blood of rats. Neural Regen Res. 9:1112-6.