E-ISSN: 2149-7222 (Online)

Journal Cellular Neuroscience and Oxidative Stress

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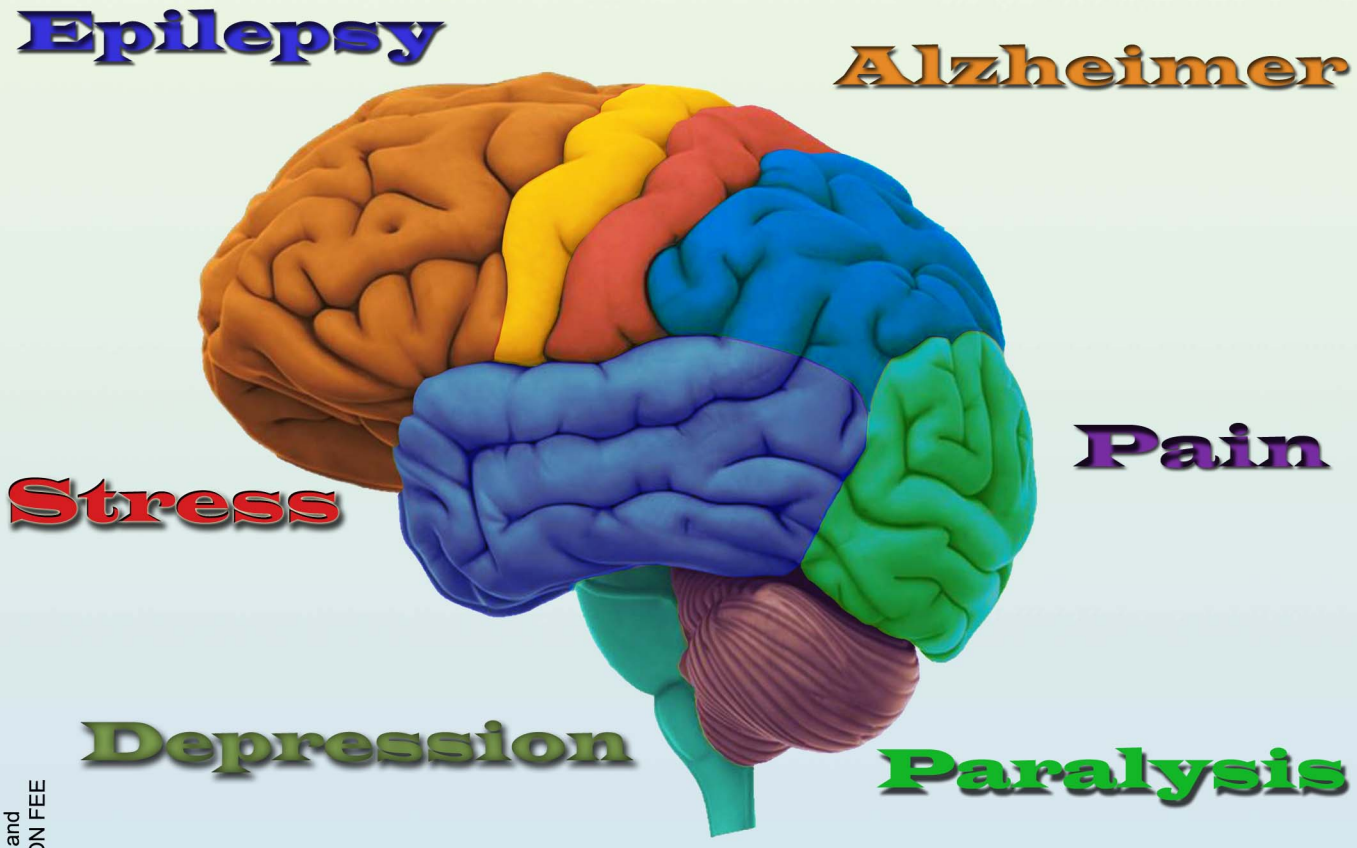

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Brain Research School

Editor in Chief
Prof.Dr. Mustafa NAZIROĞLU

Volume 10, Number 3, 2018

Journal of Cellular Neuroscience and Oxidative Stress

http://dergipark.gov.tr/jcnos

An Official Journal of the Cellular Neuroscience and Oxidative Stress Society

http://hsord.org.tr/en/

Formerly known as:

Cell Membranes and Free Radical Research (2008 - 2014)

Volume 10, Number 3, 2018

3rd International Brain Research School

25 June – 1 July 2018 Isparta /TURKEY

2018.brs.org.tr

Volume 10, Number 3, 2018
E-ISSN Number: 2149-7222 (Online)
Indexing: Google Scholar, Index Copernicus, Chemical Abstracts, Scopus (Elsevier),
EBSCOhost Research Database, Citation Index Database,

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 Presentations

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Oral Presentation 1

Traumatic brain injury models in rats

Kemal ERTİLAV
Department of Neurosurgery, Faculty of Medicine, Suleyman Demirel University, Isparta, Turkey

Traumatic brain injury (TBI) is induced in the brain by external forces such as traffic accidents and heat trauma. Death and disability are induced by the TBI. Indeed, worldwide, about 10 million people are annually deaths or hospitalizations annually by the TBI exposures. In addition, about 57 million exposed to brain injury after TBI annually (Xiong et al. 2013). There is no direct treatment method for the TBI. After the TBI, different pathological processes such as oxidative stress, inflammation and apoptosis are induced by the brain injury. Hence, investigations of new treatment methods in rodent models have important role for inhibition of the pathological processes of human.

Marmarou method has been used to make a diffuse head trauma (Marmarou et al. 1994) and it is popular for induction of TBI in rats. Before induction of TBI, the animals should anesthetized by anesthetics such as ketamine and xylazine combination. The animals are placed in prone position on the trauma table under the anesthesia. After skin incision, a steel disc (10 mm X 3 mm) is placed midline between coronal and lambdoid sutures on the animal's skull, and a 250-300 g weight is freely dropped through a cylindrical tube, with 19 mm inner diameter, from 2 m height onto the head of the animal (Marmarou et al. 1994). In the presentation, a selection of the principal models is described and the model was compared with the other models (Xiong et al. 2013).

Key words; Traumatic brain injury; Apoptosis; Rodents; Inflammation.

References

Marmarou A, Foda MA, Brink WVB, Campbell J, Kita H, Demetriadou K. 1994. A new model of diffuse brain injury in rats. J Neurosurg 80:291-300.

Xiong Y, Mahmood A, Chopp M. 2013. Animal models of traumatic brain injury. Nat Rev Neurosci. 14:128-142.