E-ISSN: 2149-7222 (Online)

Journal Cellular Neuroscience and Oxidative Stress

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Former name; Cell Membranes and Free Radical Research

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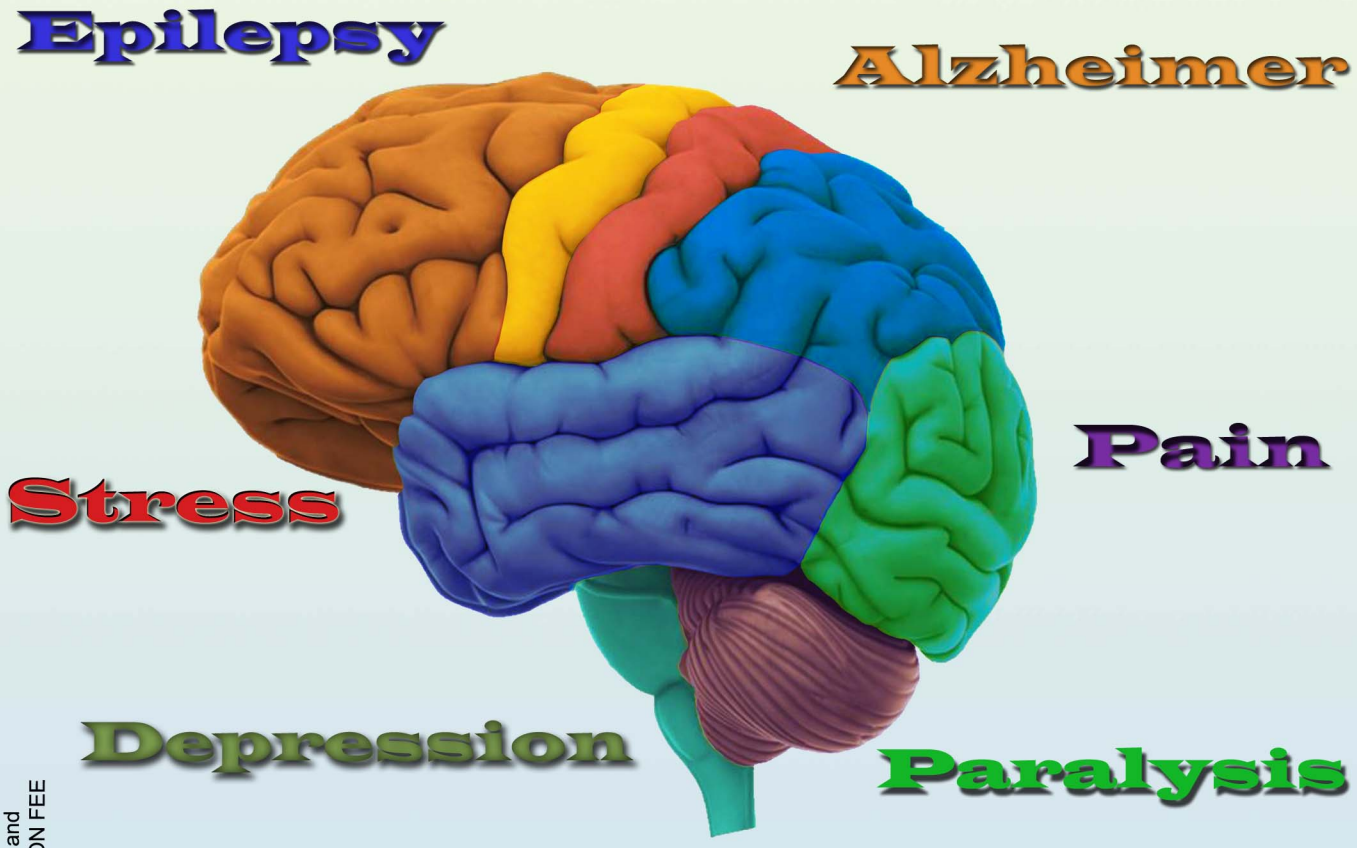

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Brain Research School

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

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 Presentation 6

TRPV1 channel is a potential drug discovery channel for epilepsy

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Epilepsy is one of the most frequent and heterogeneous neurological disorders and it is characterized by several disabilities. Epilepsy is affecting about 3% of people worldwide. Current anti-epileptic drugs are only effective in 60% of individuals and many drugs can induce several unwanted side effects in patients. Etiology of epilepsy has not been clarified fully. However, increased intracellular calcium ion (Ca^{2+}) concentration has main role in etiology of epilepsy. Ca^{2+} passes the cell membrane through different cell membrane channels. One of the channels is TRP superfamily. The family is containing six subfamilies. TRPV1 channel is a member of TRPV subfamily. Capsaicin is a component of hot chili pepper. The TRPV1 channels is activated by different stimuli such as acidic pH, high temperature (≥ 42° C) and capsaicin, causing pain, inflammation and hyperalgesia in peripheral nervous system (Caterina et al. 1997). Is has been well known that hippocampus is main area in the brain for induction of epilepsy. Expression levels of TRPV1 channels in different areas of hippocampus are high (Gonzalez-Reyes et al. 2013). Results of recent studies indicated involvement of TRPV1 channels in epilepsy (Nazıroğlu and Övey, 2015; Cho et al. 2018). In the oral presentation, I discussed novel roles of TRPV1 on the epilepsy induction by the capsaicin.

Results of a recent study indicated increased levels of intracellular Ca^{2+} concentration in hippocampus of epilepsy induced rats (Nazıroğlu and Övey, 2015). They also observed increased levels of intracellular mitochondrial oxidative stress and apoptosis levels in the neurons by the capsaicin stimulation. However, their levels were decreased by inhibition of TRPV1 channel blocker, capsazepine.

I concluded that the results of recent studies suggest that TRPV1 stimulation through capsaicin causes oxidative stress and intracellular Ca^{2+} signaling in epileptic rats. It seems to that the certain role of TRPV1 channel activation in in the epilepsy still remains to be determined.

Key words: Epilepsy; TRPV1 channels; Capsaicin; Oxidative stress.

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