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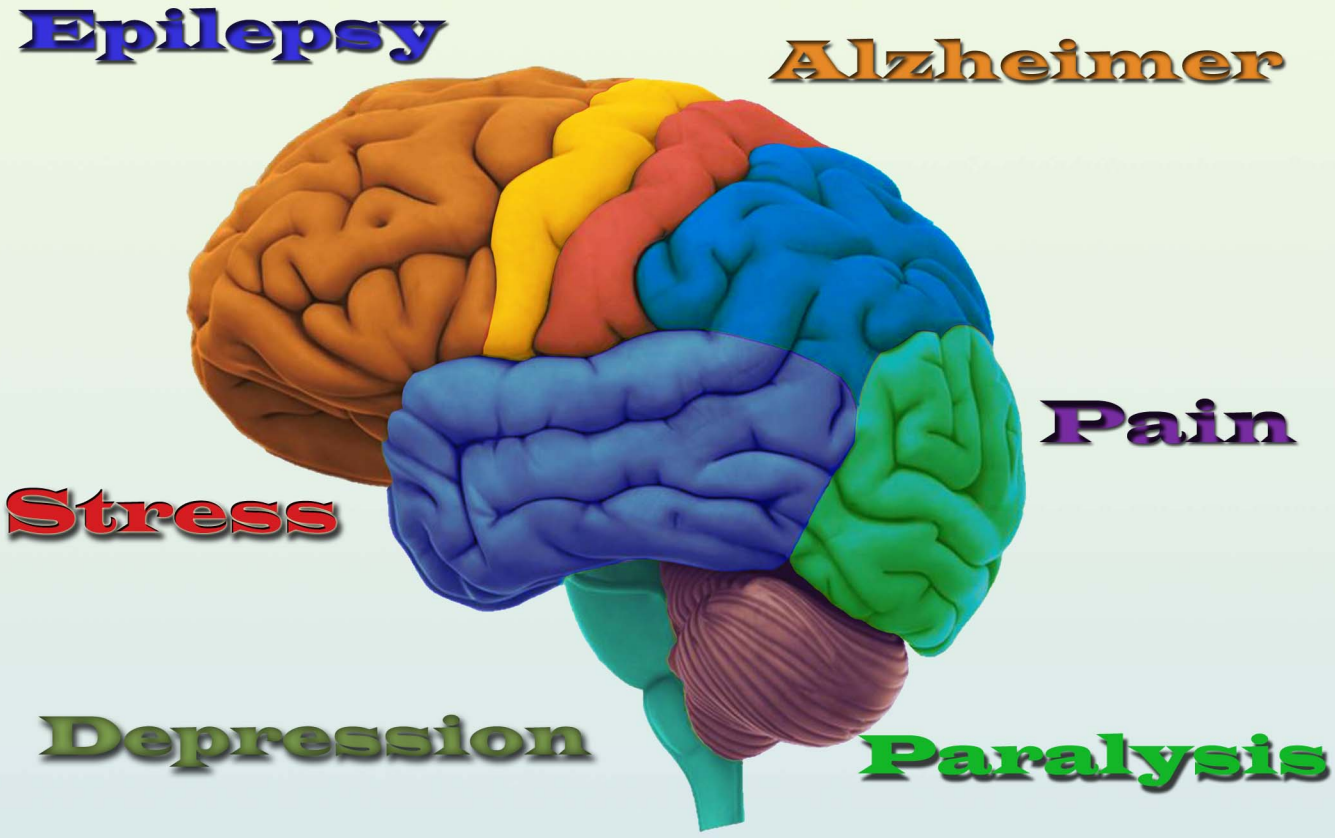

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AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate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

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Isparta, Turkey

with collaboration of

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

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Oral Presentations

Oral Presentations

Oral Presentation 3

Involvement of oxidative stress and TRP channels in cerebral ischemia

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Abnormalities of intracellular free Ca^{+2} concentration is caused through activation of mitochondrial membrane depolarization by excessive levels of reactive oxygen species (ROS). In etiology of cerebral ischemia, the abnormalities of intracellular free Ca^{+2} concentration and excessive productions of ROS play an important role in the pathophysiology of cerebral ischemia (Chinopoulos and Adam-Vizi, 2006). Ca^{2+} influx occurs through activation of different cation channels. Well-known cations channels in cell membrane are chemical and voltage gated channels. Apart from the well-known cation channels, there is transient receptor potential (TRP) superfamily. The TRP superfamily is containing 28 members in 7 subfamilies in mammalian. Activation and inhibition mechanisms of the TRP channels are very different from the well-known calcium channels. TRPM2 channel is activated by ADP-ribose NAD^{+}. Another member of TRP superfamily is TRPV1 channel and it is activated several stimuli, including capsaicin, heat (≥43 °C) and acidic pH (≤ 6) (Chinopoulos and Adam-Vizi, 2006; Toda et al, 2019). Both channels are also activated by oxidative stress. Recent data indicated protective roles of some drugs on cerebral ischemia in rodents. One of the drug is duloxetine (DULOX) and it reduced the effects of Ca^{2+} entry and ROS through inhibition of TRPM2 channel (Toda et al. 2019). Another drug is dexmedetomidine (DEX) and it is an important drug for long-term sedation in intensive care patients, because it induces a rapid response. In addition to the intensive care patients, it has been started to use for sedation and analgesia in emergency medicine patients (McMorrow and Abramo, 2012). Recently, the protective role of DEX through inhibition of TRPM2 and TRPV1 channels on experimental cerebral ischemia in rats was reported (Akpınar et al. 2016). In the oral presentation, I discussed novel effects of TRPM2, TRPV1 and oxidative stress on the cerebral ischemia in rodents and human.

I concluded that the results of current data suggest that antioxidant drugs such as DEX and DULOX treatments reduce cerebral ischemia-induced oxidative stress and intracellular Ca^{2+} signaling through inhibition of TRPM2 and TRPV1 channels. It seems to that the exact relationship between TRP channel activation and the drugs in cerebral ischemia still remains to be determined.

Keywords: Dexmedetomidine; Duloxetine; Cerebral Ischemia; Oxidative stress; TRPM2 and TRPV1 channel.

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