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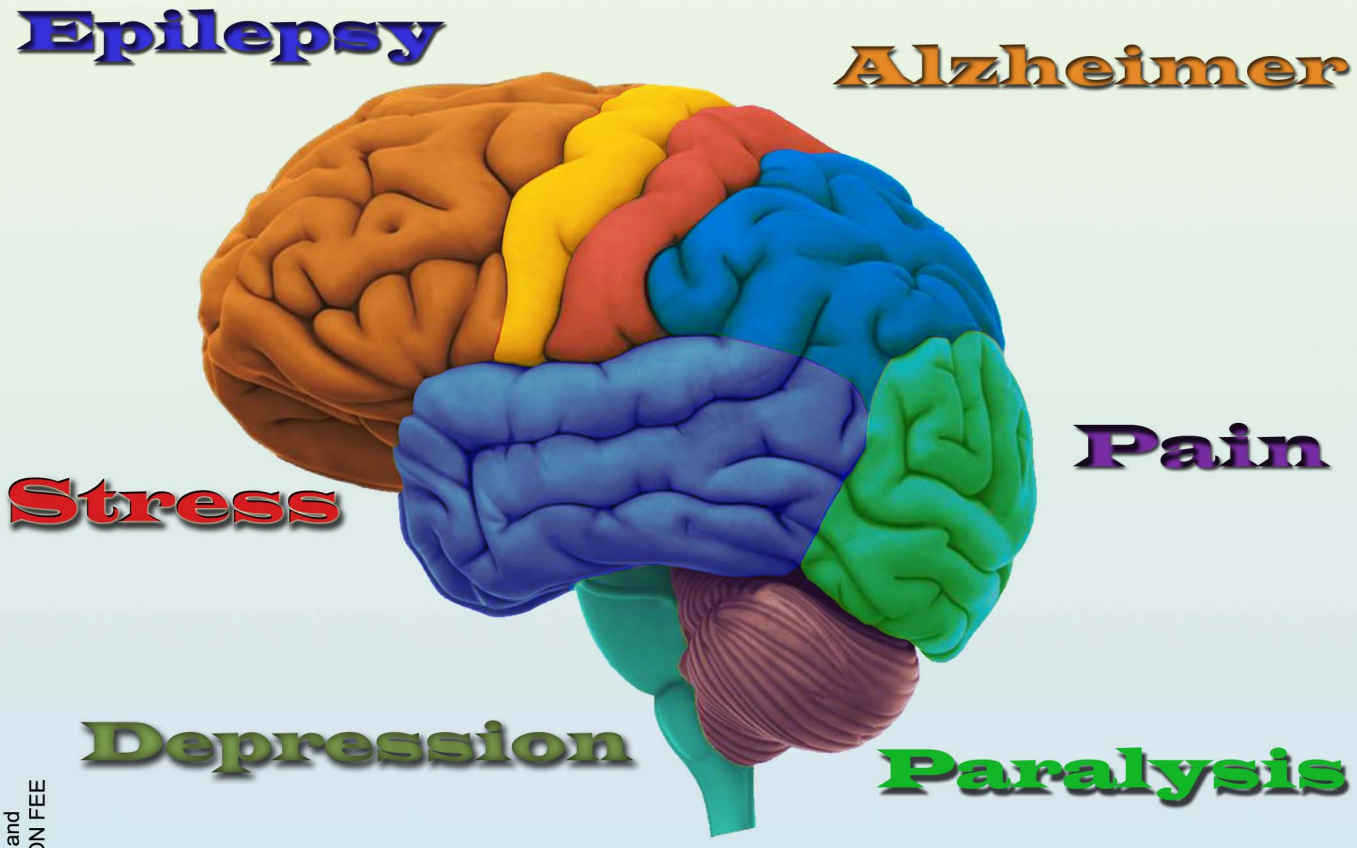

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AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate 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 9

Involvement of Thermo TRP channels on chemothrepeutic agents-induced peripheral pain

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Accumulating evidences have indicated that disturbances in intracellular free calcium ($[Ca^{2+}]i$) concentration play an important role in the pathophysiology of peripheral pain. Ca^{2+} passes cell membrane via different channels such as chemical and voltage gated channels. Apart from the well-known cation channels, there is recently discovered channels namely transient receptor potential (TRP) family. At least, 11 TRP channels in mammalian cells have been identified as thermosensitive TRP (thermo-TRP) channels (Uchida et al. 2017). Two TRP channels (TRPV1 and TRPV2) are activated by high temperatures. Five TRP channels (TRPV1-4 and TRPM2) are activated by different heat temperatures, although two of TRP channels (TRPA1 and TRPM8) are activated by cold and cool temperatures, respectively (Nazıroğlu and Braidy, 2017). It is well known that increase of $[Ca^{2+}]_i$ concentration but decrease of intracellular Mg^{2+} levels induces activation of nitric oxide synthase (NOS) enzyme. By catalytic activity of NOS, nitric oxide synthetizes in neurons. In turn, it induces pain through production of excitatory amino acids and substance P (Medvedeva et al. 2008). Results of recent studies indicated involvement of chemothrepeutic agents (i.e. cisplatin, oxaliplatin and paclitaxel)-induced mitochondrial oxidative stress through activation of Thermo TRP channels such as TRPA1, TRPV1 and TRPM8, although antioxidants induced protective action on the pain induction through inhibition of the TRP channels in the experimental animals (Materazzi et al. 2012). In the oral presentation, I discussed novel effects of chemotherapeutic agents on the peripheral pain by the regulation of TRP channels.

I concluded that the chemotherapeutic agents cause TRP channel activation and oxidative stress, which may lead to the pathology of peripheral pain. It seems to that the exact relationship between TRP channel activation and chemotherapeutic agents still remain to be determined.

Key words: TRP channel: Pain; Calcium ion; Oxaliplatin; Oxidative stress.

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