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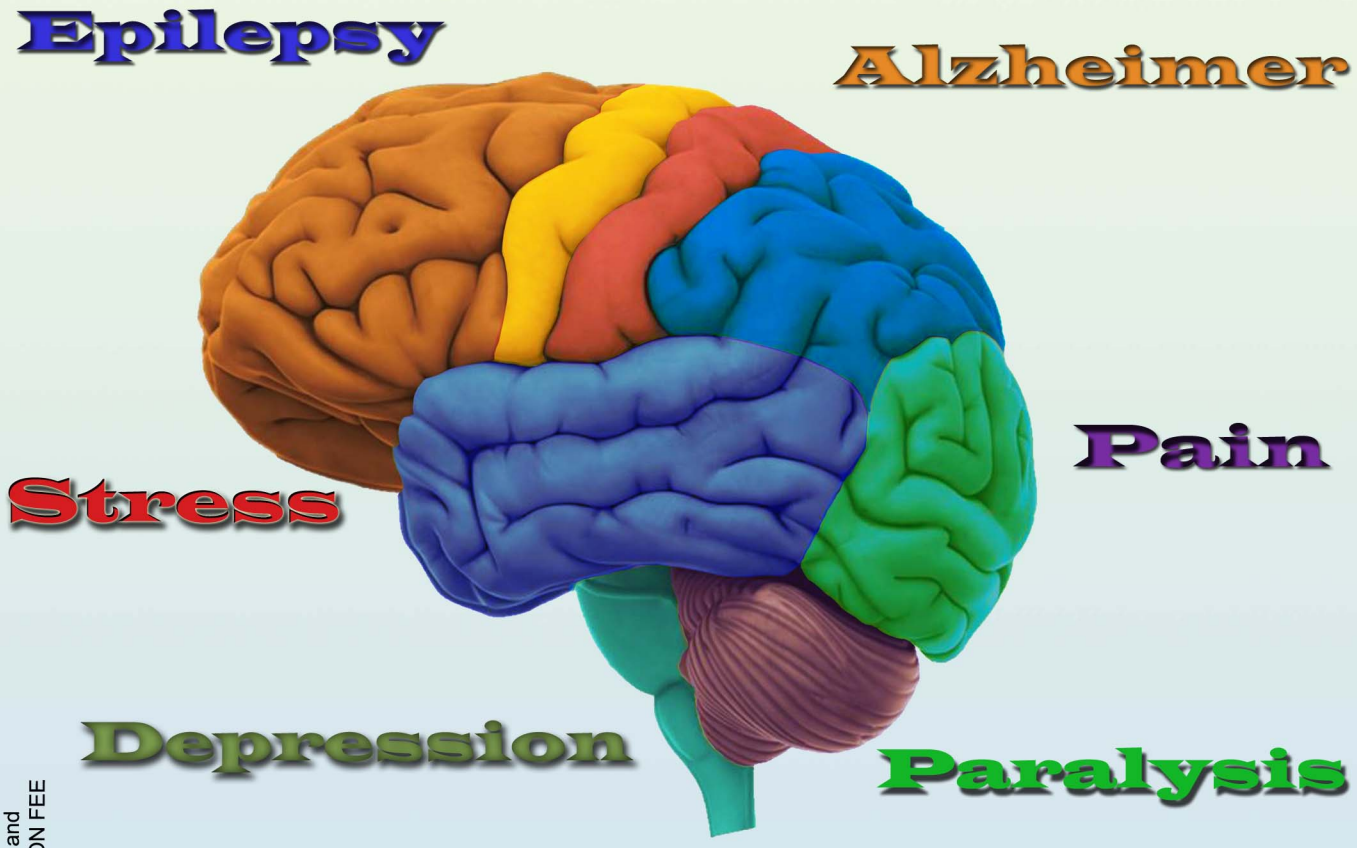

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AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate 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 4

Roles of dexmedetomidine and calcium signaling in cerebral ischemia: Focus TRP channels

Haci Ömer OSMANLIOĞLU
Department of Anesthesiology and Reanimation, Faculty of Medicine, Suleyman Demirel University, Isparta, Turkey

An accumulating body of evidence indicates that abnormalities of intracellular free calcium ($[Ca^{2+}]i$) concentration is caused by excessive levels of reactive oxygen species (ROS) in rats with cerebral ischemia in play an important role in the pathophysiology of cerebral ischemia (Miyanohara et al. 2015; Belrose and Jackson, 2018). 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. The TRP superfamily is containing 7 subfamilies with 28 members in mammalian. Activation and inhibition mechanisms of the TRP channels are very different from the voltage gated calcium channels. For example, TRPM2 channel is activated by ADP-ribose and oxidative stress, but TRPV1 channel is activated several stimuli, including capsaicin and oxidative stress (Belrose and Jackson, 2018). Dexmedetomidine (DEX) is an important drug for long-term sedation in intensive care patients because it induces a rapid response and is easily controllable. There is some modulator role of DEX on the $[Ca^{2+}]i$ concentration in several neurons (Akpınar et al. 2016). Results of a recent study indicated that DEX induced modulator role on cerebral ischemia-induced ROS, TRPM2 and TRPV1 channel activation in hippocampus of rats.

I concluded that the results of recent studies suggest that DEX treatment reduces cerebral ischemia-induced oxidative stress and intracellular Ca^{2+} signaling through inhibition of TRP channels. It seems to that the exact relationship between TRP channel activation and DEX in cerebral ischemia still remains to be determined.

Key words: Dexmedetomidine; Cerebral Ischemia; Calcium ion; Oxidative stress.

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