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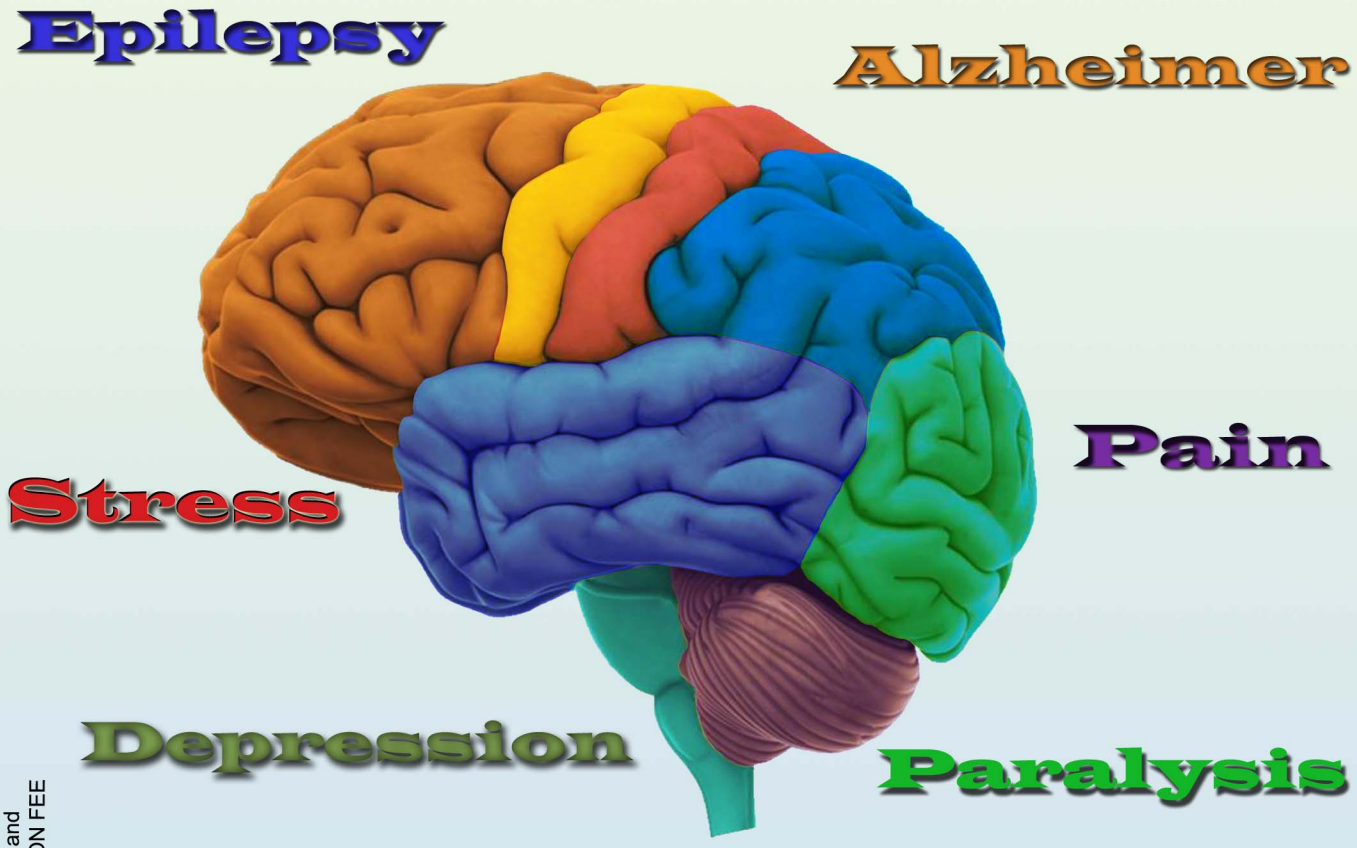

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AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate 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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Poster No. 4

Effects of alpha lipoic acid on TRPV1 cation channel in dorsal root ganglion of diabetes-induced rats

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Diabetes is a common chronic metabolic disease worldwide. It causes diabetes, tissue degenerations, cell death and associated functional disability. Diabetes also causes the development of common diseases such as cardiovascular and nervous diseases. The most important pathophysiological mechanisms that are accepted in describing diabetic degenerative processes are oxidative stress damage (El-Refaei et al. 2014). Neuropathic pain is induced by several factors including inflammation, excessive Ca^{2+} influx, oxidative stress and tissue degeneration. Inflammatory reactions in destroying neurons lead to the activation of the pain molecular pathway. In addition, destruction cell membranes induce excessive Ca^{2+} entry in the neurons, because the Ca^{2+} concentration is about 10.000 times higher in out of the neurons than in the inside of the neuron. Overload Ca^{2+} influx into cytosol and it leads to excessive production of ROS in the neurons. Alpha lipoic acid (ALA) is an important member of the thiol cycle because it is containing the sulfur groups. It is powerful antioxidant substance and it can prevent the complications of diabetes (Ghibu et al. 2009). Transient receptor potential (TRP) channels have six subfamilies and 28 members in human. Most of these channels are responsible in dorsal root ganglia (DRG) neurons for the Ca^{2+} permeation especially in neuronal cells. Expression level of the TRPV1 channels is high in the DRG neurons and they show oxidative stress dependent activation. The TRPV1 channel expression levels in the DRG increased in different types of pain.

Forty female rats were divided into four groups: First group was used as control. Second group used as diabetic. Third and fourth groups received ALA and STZ+ALA, respectively. Diabetes was induced using a single dose of intraperitoneal STZ. On 14th day of DRG samples were freshly taken from all animals. In plate reader analyses, we observed modulator role of ALA on apoptosis, caspase 3, caspase 9, mitochondrial depolarization and cytosolic ROS production values on TRPV1 channel in the DRG neurons. In addition, we observed modulator role of ALA on intracellular Ca^{2+} concentration through inhibition of TRPV1 in neuropathic pain-induced rats.

In conclusion, in our diabetes experimental model, oxidative stress are involved in the Ca^{2+} entry-induced neuronal death, and modulation of this channel activity by ALA pretreatment may account for their neuroprotective activity against apoptosis.

Keywords: Apoptosis; Oxidative stress; Alpha lipoic acid; Diabetes.

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