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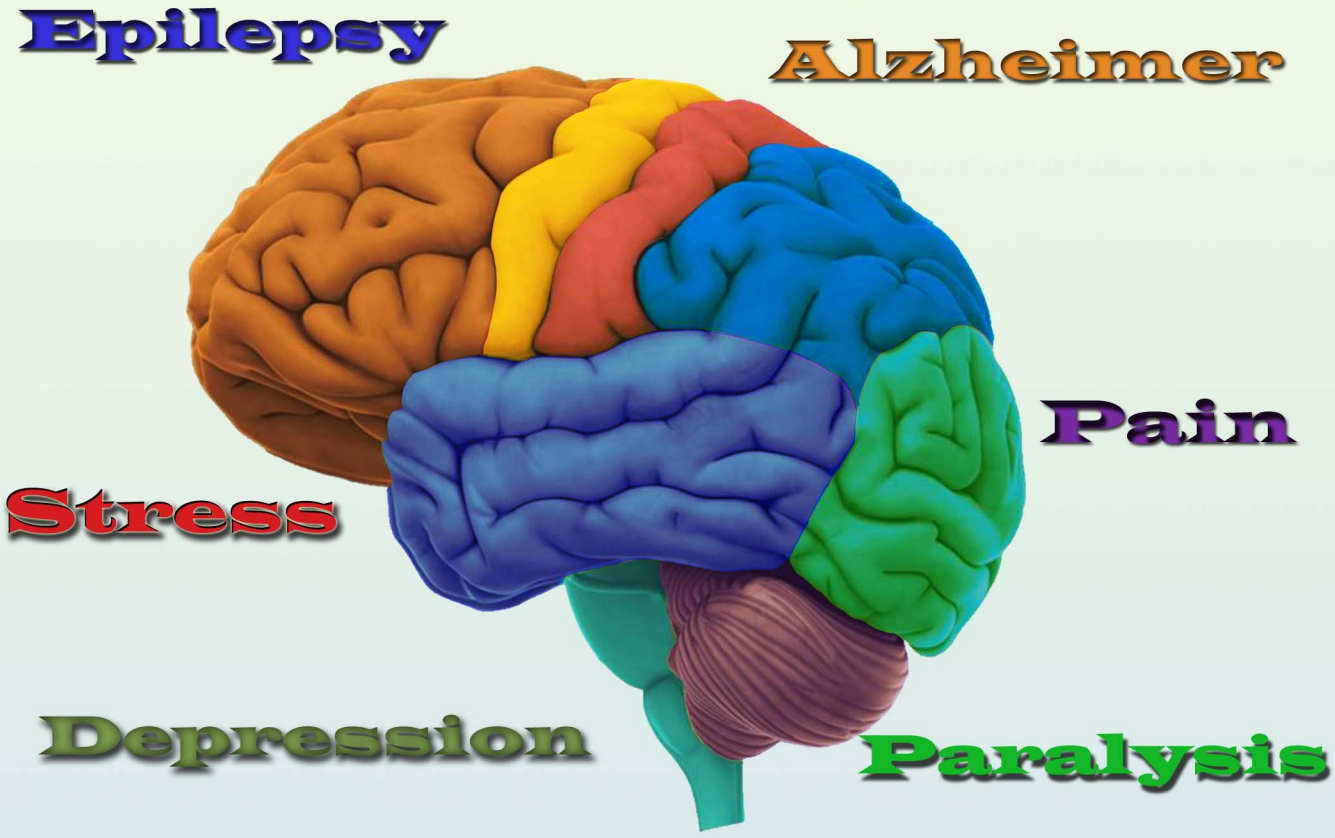

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AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate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

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Poster Presentations

Poster Presentations

Poster No. 1

Signalling mechanisms for ROS-induced TRPM2-mediated microglial cell activation

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Microglial cell is a highly plastic cell in which it retracts its branched processes upon activation by structurally diverse molecules. Elevation of these molecules in the brain has been implicated in a diversity of diseases conditions in the CNS, where these molecules promote production of toxicity mediators, such as ROS. Microglial cell activation in response to ROS has been of particular interest. Emerging evidence supports a role for the TRPM2 channel in ROS-induced neuroinflammation. Thus, the current study aims to examine the role of the TRPM2 channel in mediating H_2O_2-induced microglial activation. A multidisciplinary approach was adopted, including primary microglial isolation, single cell calcium imaging, immunocytochemistry, confocal microscopy and computer-aided analysis of cell morphology.

H_2O_2-induced microglial activation were observed in WT microglial cells but were ablated by genetic or pharmacological inhibition of the TRPM2 channel. Exposure to H_2O_2 raised the $[Ca^{2+}]_i$ via promoting Ca^{2+} influx, which was prevented by TRPM2-KO. H_2O_2 induced ROS production and PARP-1 activation. H_2O_2-induced ROS production and PARP-1 activation as well as an increase in the $[Ca^{2+}]_i$ and microglial activation, were suppressed by inhibiting PKC and NOX. Furthermore, H_2O_2-induced PARP-1 activation, increase in the $[Ca^{2+}]_i$ and microglial activation were attenuated by inhibiting the Ca^{2+}-sensitive PYK2 and downstream MEK/ERK kinases.

The findings provide strong evidence to support that the TRPM2 channel is functionally expressed and plays a major role in ROS-induced Ca^{2+} signalling as well as cell activation in microglia. Such information is useful for a better understanding of microglial cells in oxidative stress-related pathologies.

Keywords: TRPM2 channel; Reactive oxygen species; H_2O_2; Ca^{2+} signalling; Microglial activation; Oxidative stress