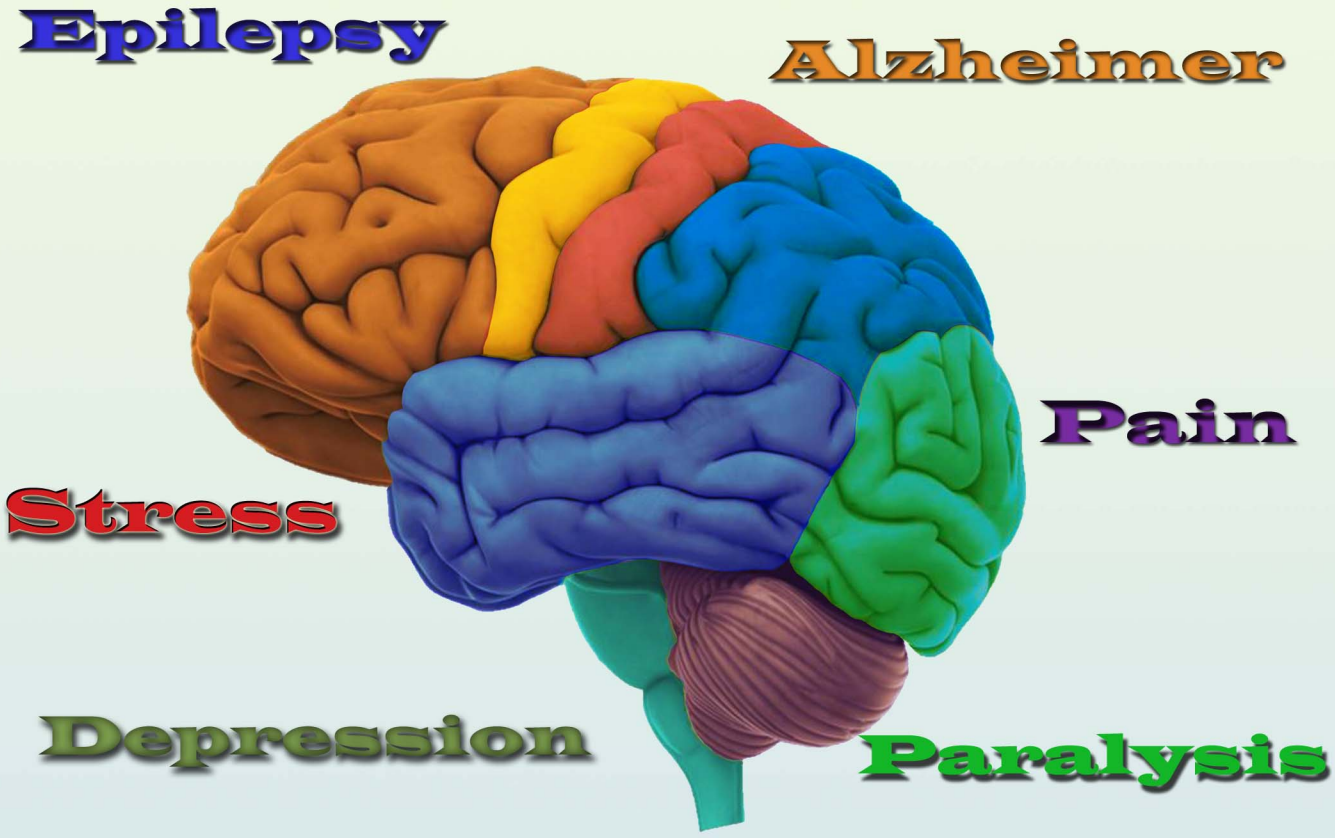
E-ISSN: 2149-7222 (Online)

Journal Cellular Neuroscience and Oxidative Stress

http://dergipark.gov.tr/jcnos

Former name; Cell Membranes and Free Radical Research



Brain Research School

OPEN ACCESS and NO PUBLICATION FEE

Supp 1 Volume, 2019

4th International Brain Research School

24-30 June 2019 Isparta /TURKEY

2019.brs.org.tr

Journal of Cellular Neuroscience and Oxidative Stress

http://dergipark.gov.tr/jcnos

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

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Formerly known as:

Cell Membranes and Free Radical Research (2008 - 2014)

Supp 1 Volume, 2019

Supp 1 Volume, 2019
E-ISSN Number: 2149-7222 (Online)
Indexing: Google Scholar, Index Copernicus, Chemical Abstracts, Scopus (Elsevier),
EBSCOhost Research Database, Citation Index Database,

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.

4th International Brain Research School

Abstract Book

of

4th International Brain Research School

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Isparta, Turkey

with collaboration of

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

& Neuroscience Research Center, Süleyman Demirel University

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Speakers

SPEAKERS

Speak No. 5

Intracellular zinc mobilization is required for nNOS (+) neuron loss. Role of zinc in the excitotoxic cascade

Alberto GRANZOTTO and Stefano L. SENSI
CeSI-MeT, Center for Excellence on Aging and Translational Medicine, University "G. d'Annunzio" Chieti-Pescara, Chieti, Italy

NMDA receptor (NMDAR) overstimulation by glutamate promotes massive calcium (Ca^{2+}) entry and initiates a cascade of events leading to the overproduction of Reactive Oxygen Species (ROS), mitochondrial dysfunction, intraneuronal zinc (Zn^{2+}) mobilization, and, ultimately, neuronal demise (Choi 1992). This glutamate-driven form of neuronal death has been described as excitotoxicity (Olney 1969). NADPH-diaphorase neurons [nNOS (+) neurons] are a subpopulation of nitric-oxide synthase-overexpressing interneurons that is spared from the NMDAR-mediated neuronal death (Koh and Choi, 1988). The mechanisms underlying the reduced vulnerability of nNOS (+) neurons to NMDAR-driven neuronal death are still largely unexplored. In the talk, we will discuss the mechanisms that are involved in the reduced vulnerability of nNOS (+) neurons. Differences between nNOS (+) and nNOS (-) neurons as far as changes in intracellular Ca^{2+} levels, mitochondrial functioning, ROS production as well as the intraneuronal accumulation of Zn^{2+} were investigated. We found that nNOS (+) neurons differ from nNOS (-) cells by lacking the production of a significant amount of ROS in response to NMDAR activation. The absence of NMDA-driven oxidative stress shown by the nNOS (+) neurons abolished the neurotoxic accumulation of Zn^{2+}. Exposure of nNOS (-) neurons to NMDA in the presence of TPEN (a Zn^{2+} chelator) mimicked the behavior of the nNOS (+) subpopulation and preserved the nNOS (-) population from the excitotoxic damage. These results indicate that Zn^{2+} mobilization is the mandatory step of the excitotoxic cascade. These findings identify the intraneuronal accumulation of Zn^{2+} as a therapeutic target for the treatment of excitotoxic prone neurological conditions.

Keywords: NMDA receptor; nNOS

References

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Olney JW. 1969. Brain lesions, obesity, and other disturbances in mice treated with monosodium glutamate. Science (80-.). 164:719–721.

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