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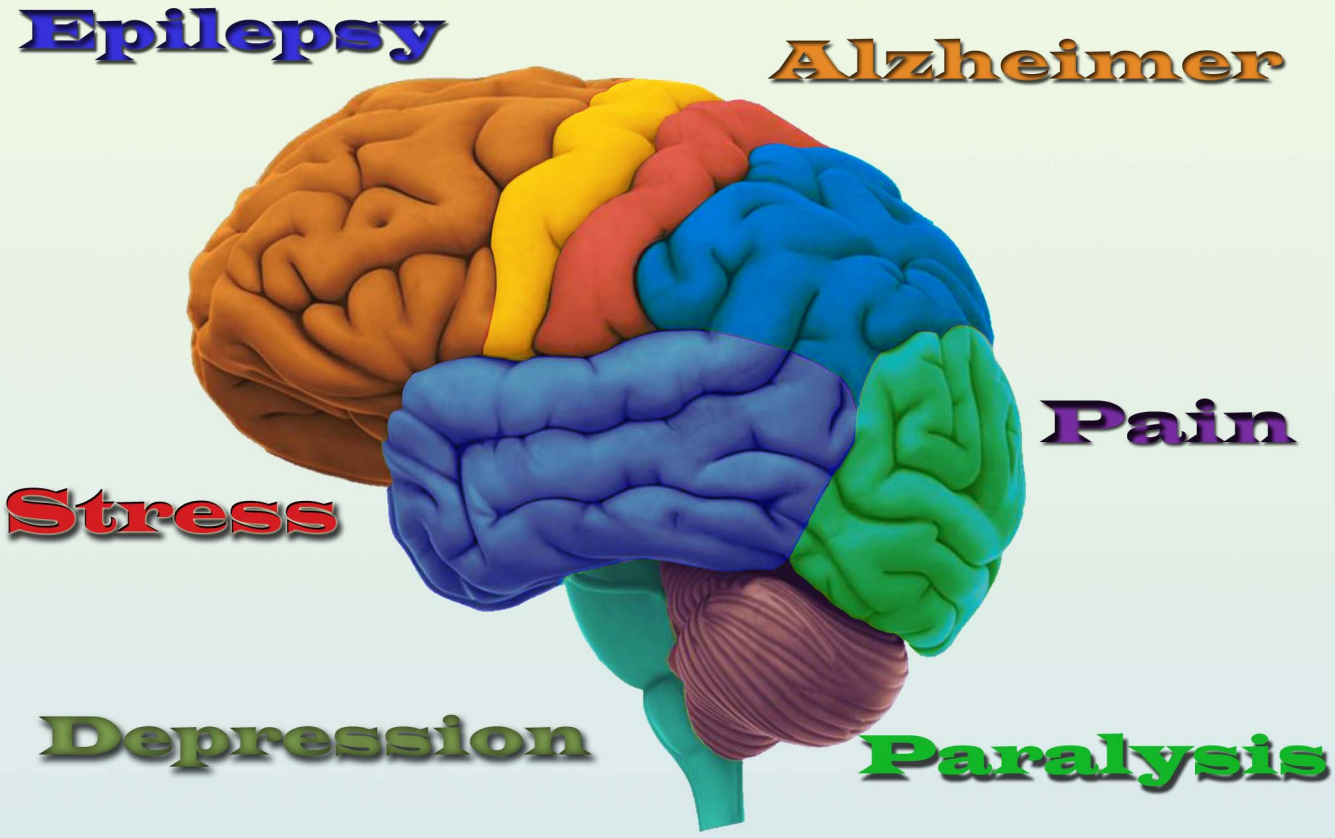

Brain Research School

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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

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

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Oral Presentations

Oral Presentations

Oral Presentation 11

Psychological and oxidative stress induce apoptosis through TRPV1 channel activation in granulosa cells of oocyte during in vitro fertilization

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Several physiological and pathophysiological functions such as mitochondria and phagocytosis induce oxidative stress. Oxidative stress results in excessive production of reactive oxygen species (ROS). There is a high amount of psychologically and chemically stress in in vitro fertilization (IVF), because of presence stressful permanent infertility and treatment procedures (An et al. 2013). Oocytes are surrounded by granulosa cells. It is well-known that there is a direct relationship between oxidative stress contents of granulosa cells and oocyte quality (Tola et al 2013). Excessive Ca^{2+} influx induces excessive mitochondrial ROS production and apoptosis through activation of caspase activations. Involvement of voltage gated Ca^{2+} channels on oocyte quality and apoptosis in the granulosa cells has been clarified by results of several studies (Platano et al. 2013; Tola et al 2013). Transient receptor potential vanilloid 1 (TRPV1) channel is a calcium permeable and non-selective cation channel. The similar effects of voltage gated calcium channels may present between oxidative stress and TRPV1 channel activation in the oocyte, because the TRPV1 channel is activated by excessive production of ROS. The importance of TRPV1 channel on the oocyte maturation was recently reported (Cecconi et al. 2019). In the oral presentation, I will review recent studies on apoptosis through TRPV1 channel activation in granulosa cells of oocyte during IVF.

In conclusion, current literature data indicated that psychological and oxidative stress-induced ROS, apoptosis and Ca^{2+} contents of oocyte and granulosa cells have very important roles on the oocyte maturation in patients with infertility during the IVF. There are some involvement clues of TRPV1 channels on the oocyte maturation and apoptosis, but the subject needs future studies.

Keywords; Apoptosis; Oocyte granulosa cells; Oxidative stress; TRPV1 channel.

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