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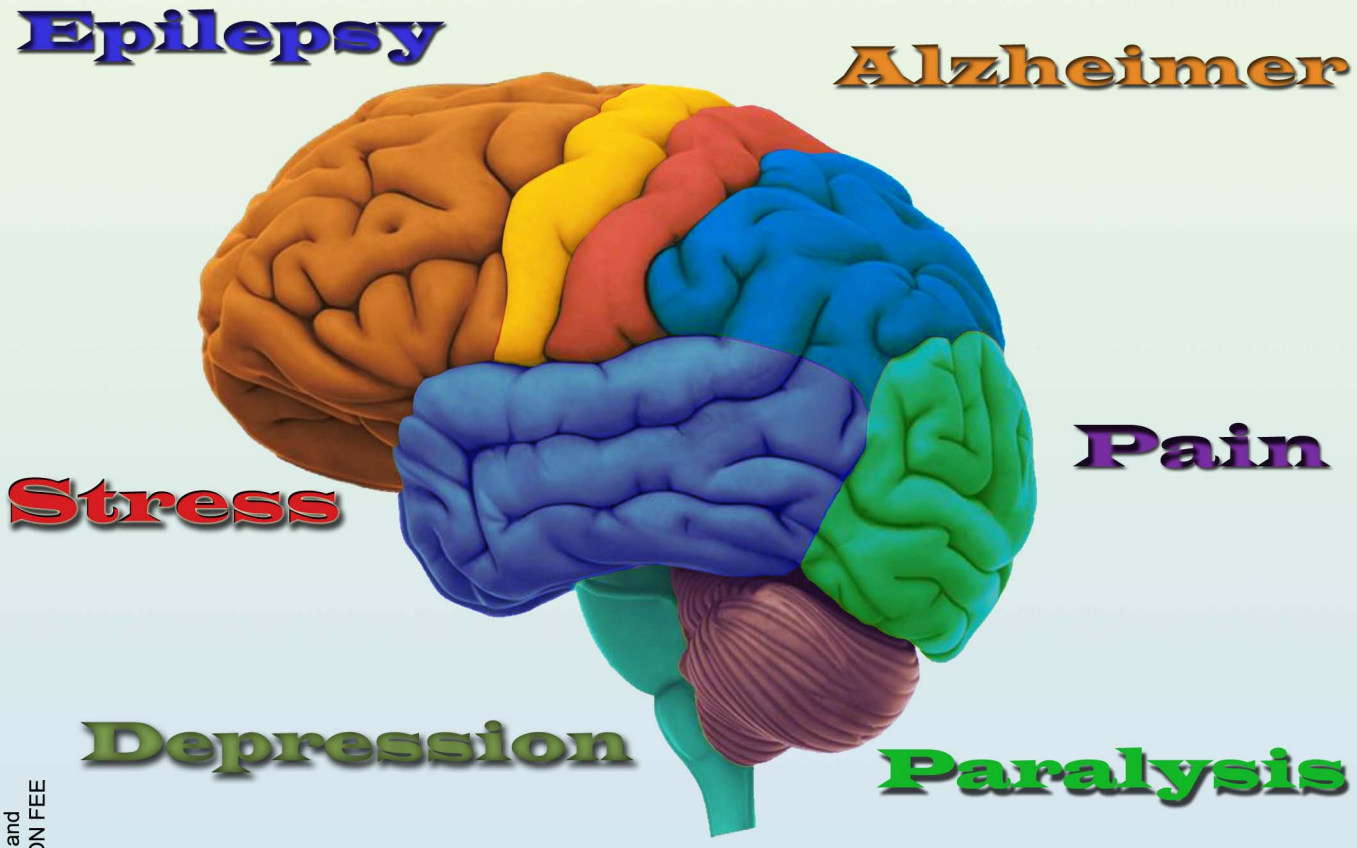

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AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate 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. 1

Dysbiosis of gut microbiota and Alzheimer's Disease

Orhan AKPINAR
Suleyman Demirel University Health Sciences Institute, Departmant of Medical Microbiology, Isparta,Turkey

Alzheimer's Disease (AD) is a degenerative, chronic, progressive disease of CNS. Pathological changes that develop in the course of the disease lead to memory loss, alteration of thought, and deterioration of other brain functions. The disease progresses slowly, resulting in cell death and brain damage (Jiang 2017; Knopman 2016) .

Increased permeability of the intestinal and blood brain barrier due to microbial dysbosis plays a role in the pathogenesis of AD and other neurodegenerative disorders associated with aging. In addition, intestinal microbiota bacterial populations secrete amyloids and lipopolysaccharides in large quantities, which may contribute to the modulation of signaling pathways and the production of proinflammatory cytokines associated with the pathogenesis of AD (Jiang 2017). Amyloid precursor protein (APP) , which constitutes Aβ plaques and is normally secreted by intestinal bacteria, is expressed by the enteric nervous system. However, the accumulation corrupts the CNS functions. Escherichia Coli and Salmonella Enterica are some of the many bacterial strains that express and secrete APP and play a role in the pathogenesis of AD (Tse 2017).

Production and clearance of Aβ in CNS is a dynamic change and some bacteria and fungi are amyloid secretions, which disrupt the dynamic balance of Aβ protein in CNS and increase the amyloid levels. This causes Aβ protein accumulation in the brain and a high risk of AD (Hill 2015).It is very important for cognitive function in serotonin, 95% of serotonin is synthesized in intestines and intestinal microorganisms play an important role in the synthesis of serotonin. There is evidence that serotonin may reduce the formation of Aβ plaques and thus reduce AD risk (Hill 2015; Jiang 2017).

Key words; Microbiota; Dysbiosis; Alzheımer's Dısease.

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