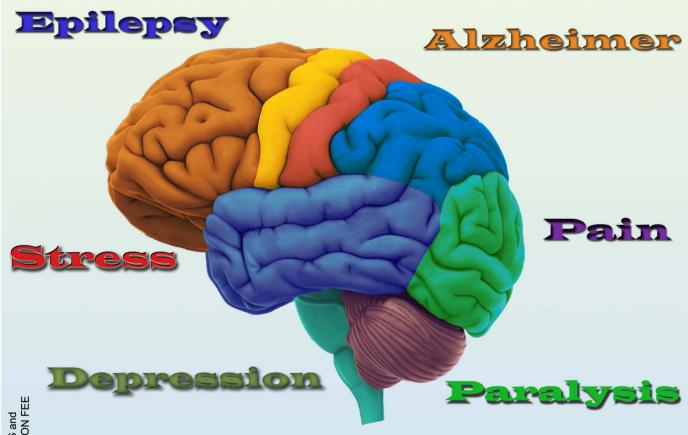
# Journal Cellular Neuroscience and Oxidative Stress

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Editor in Chief Prof.Dr. Mustafa NAZIROĞLU

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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<sup>+</sup>- K<sup>+</sup> Channels, Cl<sup>-</sup> channels, Ca<sup>2+</sup> channels, ADP-Ribose and metabolism of NAD<sup>+</sup>, 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<sup>+</sup> 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

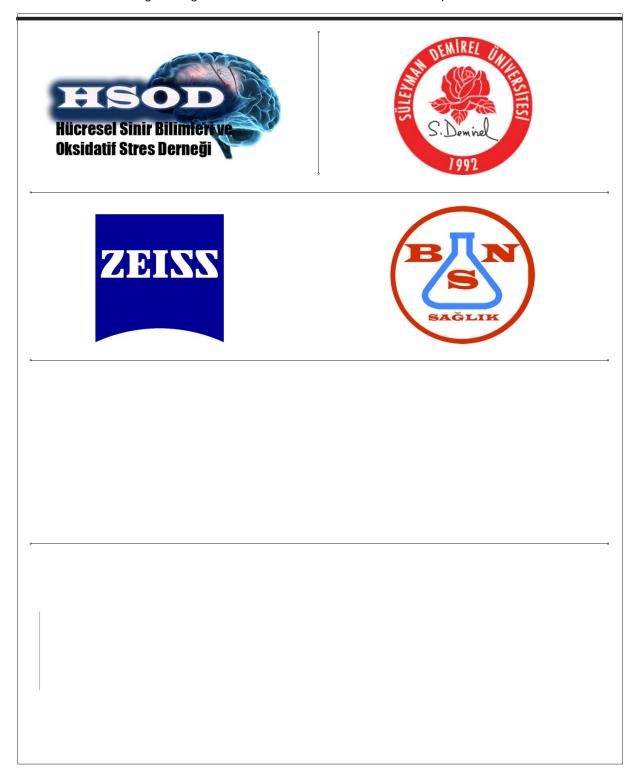
Neuroscience Neuropharmacology

#### **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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#### Neurodegenerative disease and microbiota

#### Mustafa GÜZEL<sup>1</sup>, Doğan AKDOĞAN<sup>2</sup>, Orhan AKPINAR<sup>3</sup>

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Evidence suggests that intestinal microbiota, especially in the case of dysbiosis, may affect the progression of neurological diseases and may even lead to the formation of the disease. It has been realized that decreasing diversity in aging gut of the microbiota may be an important factor in the development of neurodegeneration. Neuroinflammation is one of the major mechanisms that associate microbiota with agerelated diseases. Intestinal microbiota; plays a key role in the activation of microglia and it is suggested that manipulation of intestinal microbiota, especially with short chain fatty acid producing bacteria, may modulate neuroimmun activation (Westfall et al. 2017).

On the clinical and scientific level, most neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis. Disease related pathology may spread across the nervous system in a self-propagative fashion. Importantly, there is a strong bidirectional interaction between gut microbiota and the central nervous system, a connection recently termed the "microbiota-gut-brainaxis" (Jiang et al. 2017; Houser and Tansey, 2017).

While the effects of the autonomic nervous system on gut physiology have been known for a long time, we are just beginning to understand that gut microbiota has strong effects on CNS physiology as well. The vast number of ways through which gut microbiota affects the host shows intriguing overlaps with pathways previously implicated in neurodegeneration. Although evidence for involvement of microbiota neurodegenerative diseases is still very preliminary, initial findings are extremely promising (Zhu et al. 2017).

This presentation will give an overview of recent findings regarding the connections between gutmicrobiota and neurodegenerative disorders and how this may reshape our understanding of these diseases.

Kev words: Microbiota; Neurodegeneration; Alzheimer's disease: Parkinson's disease.

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