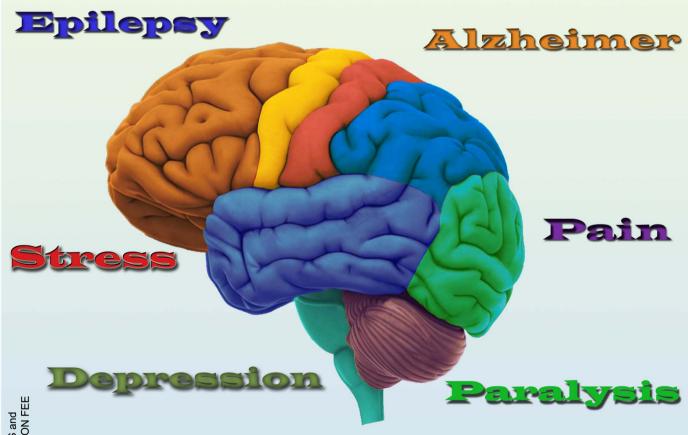
# Journal Cellular Neuroscience and Oxidative Stress

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Former name; Cell Membranes and Free Radical Research





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Phone: +90 246 211 36 41, Fax:+90 246 237 11 65

E-mail: mustafanaziroglu@sdu.edu.tr

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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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#### Human gut microbiota and Parkinson Disease

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

<sup>1</sup>Department of Medical Microbiology, Maltepe Medical Center, Istanbul, Turkey

<sup>2</sup>Department of Medical Microbiology, Health Sciences Institute, Suleyman Demirel University Isparta, Turkey

Human gut microbiota (GM) has now been accepted as a potential modulator ofhuman biology. Although new to the world of science, GM's impaction brain and behavior has drawn great attention around the globe. Studies have now proven that GM can directly or indirectly modify brain neurochemistry via various mechanisms like neural, immune and endocrine. The intestinal microbiota influence neurodevelopment, modulate behavior, and contribute to neurological disorders. This presentation is an overview of recent findings regarding the GM -brain axis in PD (Braniste et al. 2014; Sampson et al. 2016)

Parkinson disease (PD) is the second-most common neurodegenerative disorder. PD patients show alpha-synuclein deposits and neurodegeneration in the enteric nervous system as well as breakdown of the mucosal barrier, bacterial invasion, and mucosal inflammation in the colon. Alterations in GM and increased gut permeability may influence PD pathophysiology via epigenetic processes that alter αSyn regulation (Matsumoto et al. 2010).

Sampson et al. (2016) suggest that GM are required for the hallmark motor and GI dysfunction in a mouse model of PD, via postnatal gut-brain signaling by microbial molecules that impact neuroinflammation and  $\alpha$ Syn aggregation. They propose that GM regulate movement disorders and suggest that alterations in the human microbiome represent a risk factor for PD. GM do not only affect gut physiology, but there is also an intense bidirectional interaction with the brain influencing neuronal activity, behavior, as well as levels of neurotransmitter receptors, neurotrophic factors, and inflammation. Recently, gut microbiome alterations in PD subjects and a connection between GM and motoras well as non-motor symptoms have been described

(Sampson et al. 2016; Parashar and Udayabanu 2017)

**Key words;** Parkinson disease; Gut microbiota; gutbrain signaling.

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