

# Journal Cellular Neuroscience and Oxidative Stress

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Former name; Cell Membranes and Free Radical Research

**Epilepsy**

**Alzheimer**



**Pain**

**Stress**

**Depression**

**Paralysis**

**Brain Research School**

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Editor in Chief  
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Supp 1 Volume, 2019

# 4<sup>th</sup> International Brain Research School

24-30 June 2019 Isparta /TURKEY  
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# Journal of Cellular Neuroscience and Oxidative Stress

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Supp 1 Volume, 2019

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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.

# 4<sup>th</sup> International Brain Research School

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## Abstract Book

of

4<sup>th</sup> International Brain  
Research School

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with collaboration of  
BSN Health Analyses, Innovation,  
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& Neuroscience Research Center,  
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# SPEAKERS

## ▶ Speak No. 7

### **Voltage gated sodium channels and epilepsy**

#### **Simon HEBEISEN**

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Epilepsy is the fourth most common neurological disorder and affects people of all ages. Medication for epilepsy is often life-long and has a major impact on the quality of life - mostly being related to substantial adverse effects. Therefore, over 30% of people with epilepsy do not achieve sufficient seizure control whilst effective medication being available.

Ion channels are often primary targets of anticonvulsant drugs. They can either act as blockers for voltage gated sodium and calcium channels or as activators for potassium or chloride channels. Additionally, modulators of ligand gated ion channels (GABA or Glutamate receptors) are frequently used to treat epilepsy.

Employing a panel of functional electrophysiological assays using fluorescence based methods and patch-clamping on a broad range of voltage and ligand gated ion channels, we were able to successfully screen for drugs with a beneficial action profile. In successful leads we found drugs that selectively interacted with TTX sensitive, neuronal voltage gated sodium channels. Activation and fast inactivation were unchanged, while an increased affinity in the slow inactivated state was observed. This profile is in contrast to traditional anticonvulsant drugs which show their major effects on the fast inactivated state of voltage gated sodium channels. One drug showed substantial shifts of the voltage dependence of the slow inactivation only for Na<sub>v</sub>1.2 and 1.6. This favours this drug for treating patients with diseases with compromised Na<sub>v</sub>1.1 function in interneurons, such as

Alzheimer's disease.

**Keywords:** Epilepsy, voltage gated sodium channels, state dependent inactivation, patch-clamp technique