

# 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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# 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

## Abstract Book

of

4<sup>th</sup> International Brain  
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with collaboration of  
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# Oral Presentations

## ▶ Oral Presentation 8

### The anticonvulsant effects of salmon calcitonin on pentylentetrazole-kindled rats

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Epilepsy is a disorder of the brain, characterized by an enduring predisposition for the generation of epileptic seizures because of hyperexcitability and hypersynchrony of cortical neurons (Devinsky et al. 2014). Salmon calcitonin is a type of calcitonin with 32 amino acids. It is more potency than human calcitonin due to differences in its amino acid sequence (Masi et al. 2007). In the current study, we investigated the effects of salmon calcitonin on pentylentetrazole-induced seizures in kindled rats.

In our study, 48 (240-260 g) male Wistar Albino rats were used. Rats were kindled by injections of a subconvulsant dose of pentylentetrazole (35 mg/kg) once every other day for 15 times. Epileptic behaviors were observed for a period of 30 min. Seizure activity was scored, using the revised Racine's scale. Rats that had seizure stages of 4 or 5 after three consecutive injections of PTZ were defined as fully kindled. The kindled rats were divided into six groups (n=8 for each group) as saline (1 ml/kg saline), salmon calcitonin (25, 50 and 100 µg/kg), ethosuximide (100 mg/kg) and ethosuximide + salmon calcitonin. Electrodes were placed to animals' skulls under stereotaxy to receive electroencephalography (EEG). After thirty minutes of administration of drugs, 35 mg/kg PTZ was given to induce seizures. EEG and video recordings of animals were taken simultaneously for thirty minutes. In the evaluation of the video and EEG recordings, the seizure stages of animals, the first myoclonic jerk time and the number of epileptic seizure spikes were calculated.

Salmon calcitonin reduced seizures stage, epileptic seizure spikes and also prolonged first myoclonic jerk time compared to saline group. In addition, salmon calcitonin and ethosuximide combination decreased epileptic seizure spikes and increased the first myoclonic jerk time compare to ethosuximide group.

In conclusion, salmon calcitonin decreased epileptic seizures and improved anticonvulsant effect of ethosuximide in the pentylentetrazole-kindled rat.

**Keywords:** Epilepsy, Salmon Calcitonin, Ethosuximide, Pentylentetrazole, Rats

### References

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