

# 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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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

## Abstract Book

of

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

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with collaboration of  
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# Oral Presentations

## ▶ Oral Presentation 2

### The effects of quercetin on antioxidant and cytokine levels in rat hippocampus exposed to acute cadmium toxicity

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The aim of this study was to determine cadmium neurotoxicity (Branca et al. 2018) and beneficial effect of quercetin (QE) (Kanter et al. 2016) against neuronal damage in hippocampus exposed with acute cadmium (Cd).

Adult male Wistar-Albino rats (n = 30) were used and divided into four groups as Control (C, n = 6), Cadmium (Cd, n = 8), Quercetin (Q, n = 8) and Cadmium + Quercetin (Cd + Q, n = 8). Cadmium (CdCl<sub>2</sub>, 4 mg kg<sup>-1</sup> daily, s.c) were administrated to Cd and Cd+Q groups, and Quercetin (Q, 50 mg kg<sup>-1</sup> daily, i.p) were administrated to Q and Cd + Q groups for 3 days, respectively. At 4<sup>th</sup> day after the treatments, hippocampal samples were taken from the four groups.

Cadmium decreased superoxide dismutase (SOD) and reduced glutathione (GSH) levels and the SOD activity and GSH level were markedly (p< 0.05) lower in Cd group than in the Q and C groups. Lipid peroxidation (MDA) levels were higher in Cd group when compared to the control, Q and Cd+Q groups. IL-1 levels were found statistically higher in Cd group than in the control, Q and Cd+Q groups. IL-6 and TNF-alfa levels were significantly (p< 0.05) higher in Cd and

Cd+Q groups than the Q and C groups. In addition, IL-10 levels were detected the lowest in Cd group when compared to other groups.

In conclusion, our results show that quercetin can be beneficial against to neurotoxic effects of acute cadmium toxicity in the rat hippocampus through upregulation of antioxidant system but down regulation of cytokine levels.

**Keywords:** Cadmium; Cytokines; Hippocampus; Oxidative stress; Quercetin

### References

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