

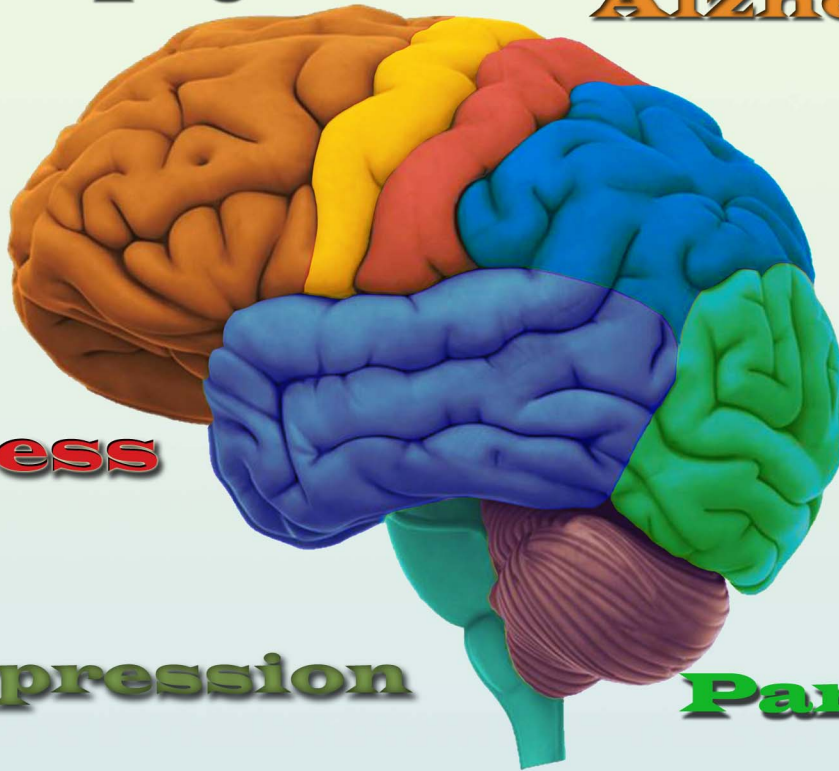
# Journal Cellular Neuroscience and Oxidative Stress

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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

<http://dergipark.gov.tr/jcnos>

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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

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with collaboration of  
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# SPEAKERS

## ▶ Speak No. 3

### **In vivo and ex vivo imaging of nociceptor expression and activity**

**Marie MULIER, Joris VRIENS, Thomas VOETS**

KULeuven University, Laboratory of ion channel research Leuven, Belgium

Our ability to perceive nociceptive pain is crucial to respond to harmful stimuli: it warns us about noxiously hot or cold objects. Sensory nerve endings that respond to pain (nociceptors) initiate an electrical signal in the periphery and transport it to the dorsal horn and eventually to higher brain centers. The detection of noxious stimuli involves the expression of nociceptive ion channels in sensory nerve endings, such as transient receptor potential (TRP) channels. Recently we found that the acute response to noxious heat relies on the functional expression of TRPV1, TRPM3 and TRPA1 in sensory nerve endings. Calcium imaging on isolated dorsal root ganglia (DRG) is a widely accepted model to study the involvement of TRP channels in acute pain responses. However, DRGs are clusters of nerve cell bodies, present in the dorsal root of spinal nerves, far away from the skin where the physiological stimulus detection take place.

We use two different optical measurement protocols to study ion channel activity in sensory neurons: an *in vivo*-protocol to image DRGs and an *in tissue*-protocol to visualize intact skin measurements. A cre-dependent GCaMP3 mouse line is used. These mice express the genetically encoded calcium indicator GCaMP3 in specific somatosensory neurons. Using this technique, we study the role of TRP channels in different pain models, such as inflammation.

**Keywords:** Nociceptor expressions; TRP channels; Pain.