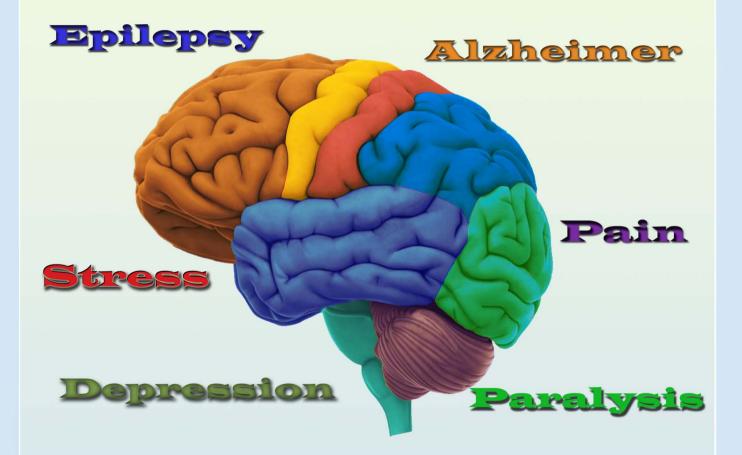
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Prof. Dr. Mustafa Nazıroğlu, Department of Biophysics and Neurosciences, Medical Faculty, Suleyman Demirel University, Isparta, Turkey. Phone: +90 246 211 36 41, Fax:+90 246 237 11 65 E-mail: mustafanaziroglu@sdu.edu.tr

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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⁺- K⁺ Channels, Cl⁻ channels, Ca²⁺ channels, ADP-Ribose and metabolism of NAD⁺, 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^+ 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)

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Biophysics	Biochemistry
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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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Interactions between chemotherapy-induced neuropathic pain and TRPV1 channel

Haci Ömer OSMANLIOĞLU

Department of Anesthesiology and Reanimation, Faculty of Medicine, Suleyman Demirel University, Isparta, Turkey.

As a complex problem, pain activates several conditions, symptoms, and molecular pathways. After stimulation of a nociceptors, action potentials are generated and then propagated to the brain, resulting in a sensation of pain is induces through production and propagation of action potential. Most efficient way to treat chronic pain is with opioids, however the drugs of opioid system induce several adverse effects such as addictive behavior and desensitization. Chemotherapeutic agent (such as oxaliplatin, cisplatin, paclitaxel)-based anticancer drugs cause neurotoxicity through excessive calcium ion (Ca²⁺) influx. Peripheral neuropathies are a common side effect of treatment of various chemotherapeutics. Today, targeting the cation channels and excessive Ca²⁺ influx that contribute to the detection of stimuli may be an effective approach in chemotherapeutic treating agents-induced pain syndromes. Several physiological and pathophysiological functions are induced by excessive Ca²⁺ influx. The Ca²⁺ passes the cell membrane through several channels such as voltage gated calcium channels (VGCC) and chemical (ligand) channels. In addition to the well-known VGCC and ligand channel, new channels namely transient receptor potential (TRP) channels were discovered within last decades. The TRP superfamily is including 28 members in mammalian and a member of the TRP superfamily is TRP vanilloid 1 (TRPV1) channels. The TRPV1 channel is activated by several stimuli including hot chili pepper component

(capsaicin), heat, acidic pH and oxidative stress (Caterina et al. 1997). Expression levels of TRPV1 channel is high in dorsal root ganglion (DRG) and it is mainly responsible from neuropathic pain (Nazıroğlu and Braidy, 2017; Muller et al. 2019). Therefore, TRPV1 channel has great importance in the chemotherapy-induced neuropathic pain induction. In the current study, I will summarize present reports on the TRPV1 channel in literature. as novel target for treating chemotherapy-induced peripheral pain. In addition, I will summarize future directions of the novel targets.

Keywords: Chemotherapeutics; Neuropathic pain; Calcium ion; Oxidative stress.

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