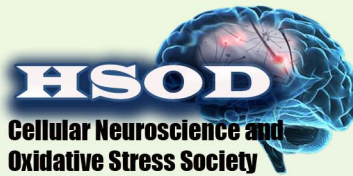


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Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate cellular 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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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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Role of desflurane on oxidative stress in neuroscience

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Oxidative stress in a neuron is induced by several physiological and pathological processes. Within the pathophysiological processes, ischemia-reperfusion injury has major role in the neurons and brain, because the neurons and brain are very sensitive to oxidative stress as compared to other tissues due to their high oxygen consumption rate and rich poly unsaturated fatty acid content but low antioxidant levels. Results of rodent studies indicated that exposure to volatile anesthetics as a result of ischemia-reperfusion injury can activate leukocytes or alveolar macrophages, which, in turn, release inflammatory mediators and reactive oxygen species (ROS). This release of inflammatory mediators, ischemia/reperfusion injury, and ROS has been clearly demonstrated in generalized inflammatory reactions involving the production of phagocytic cells such as leucocytes and microglia. A common volatile general anesthetic is desflurane and results of several recent papers indicated that it can increase oxidative stress but can decrease antioxidant defense mechanisms through ischemia/reperfusion injury mechanisms.

The excessive production of ROS is scavenged by enzymatic and non-enzymatic antioxidants. Major enzymatic antioxidants are vitamin A, vitamin C, vitamin E, glutathione, alpha lipoic acid and melatonin. Major non enzymatic antioxidants are glutathione peroxidase (GSH-Px), superoxide dismutase (SOD) and catalase (CAT). Superoxide radical is converted to hydrogen peroxide by SOD enzyme and then the hydrogen peroxide is converted to water by CAT and GSH-Px enzymes. Results of papers indicated that the CAT, GSH-Px, SOD, vitamin A, vitamin E and vitamin C values were decreased in plasma and erythrocytes of human and animals by desflurane anesthesia, but

oxidative stress levels were increased by desflurane anesthesia (Allaouchiche et al. 2001; Ceylan et al. 2011; Yalcin et al. 2013). In the oral presentation, I will summarize the results of recent papers on oxidative stress and antioxidants in human and rodents.

In conclusion, it seems that desflurane anesthesia has oxidant effects through down-regulating the enzymatic and non-enzymatic antioxidants but up-regulating of lipid peroxidation.

Key words: Antioxidants; Anesthesia; Desflurane; Oxidative stress.

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