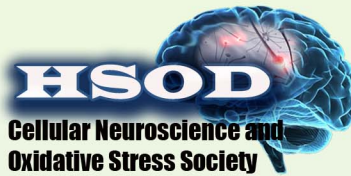


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Stress

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Depression

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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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Experimental Parkinson's disease models**Eda Duygu IPEK, Hulki BASALOGLU**

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Parkinson's disease (PD) is a neurodegenerative disease that develops slowly; however, there is no efficient method of early diagnosis, nor is there a cure. It is characterized by the relatively selective loss of dopaminergic neuronal cells in the substantia nigra pars compacta and the presence of alpha-synuclein aggregation named as Lewy bodies and Lewy neurites in surviving affected neurons. Nigrostriatal dopaminergic neurodegeneration is shared with other parkinsonian disorders, including some genetic forms of parkinsonism, but many of these disorders do not have Lewy bodies. An ideal animal model for PD, therefore, should exhibit age-dependent and progressive dopaminergic neurodegeneration, motor and non-motor dysfunction, and abnormal alpha-synuclein pathology.

A wide range of neurotoxic agents are used to induce PD, alterations that are similar with dose observed in human PD. These agents are classified mainly by administration route and the species involved. The toxins that are mainly used in present 6-hydroxydopamine, 1-Methyl-4-phenyl-1,2,3,6-tetrahydropyridine, rotenone, paraquat, reserpine, methamphetamine, 3-nitrotyrosine and isoquinoline derivatives (Tieu, 2011; McDowell and Chesselet, 2012; Bezard et al. 2013). In addition, viral mediated expression of human α -synuclein, as well as the inoculation of pathogenic α -synuclein species from Lewy bodies of PD patients, for accurately modelling progressive self-propagating neurodegeneration and genetic LRRK2 models (PARK8 gene mutation) has been used (Jiang and Dickson, 2018).

In conclusion, these models are only approximations, each possibly holding a certain degree of relevance. Thus, researchers should select models whose characteristics are most suitable for addressing the experimental question.

Keywords: Experimental Parkinson's disease; Neurotoxic agents.

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