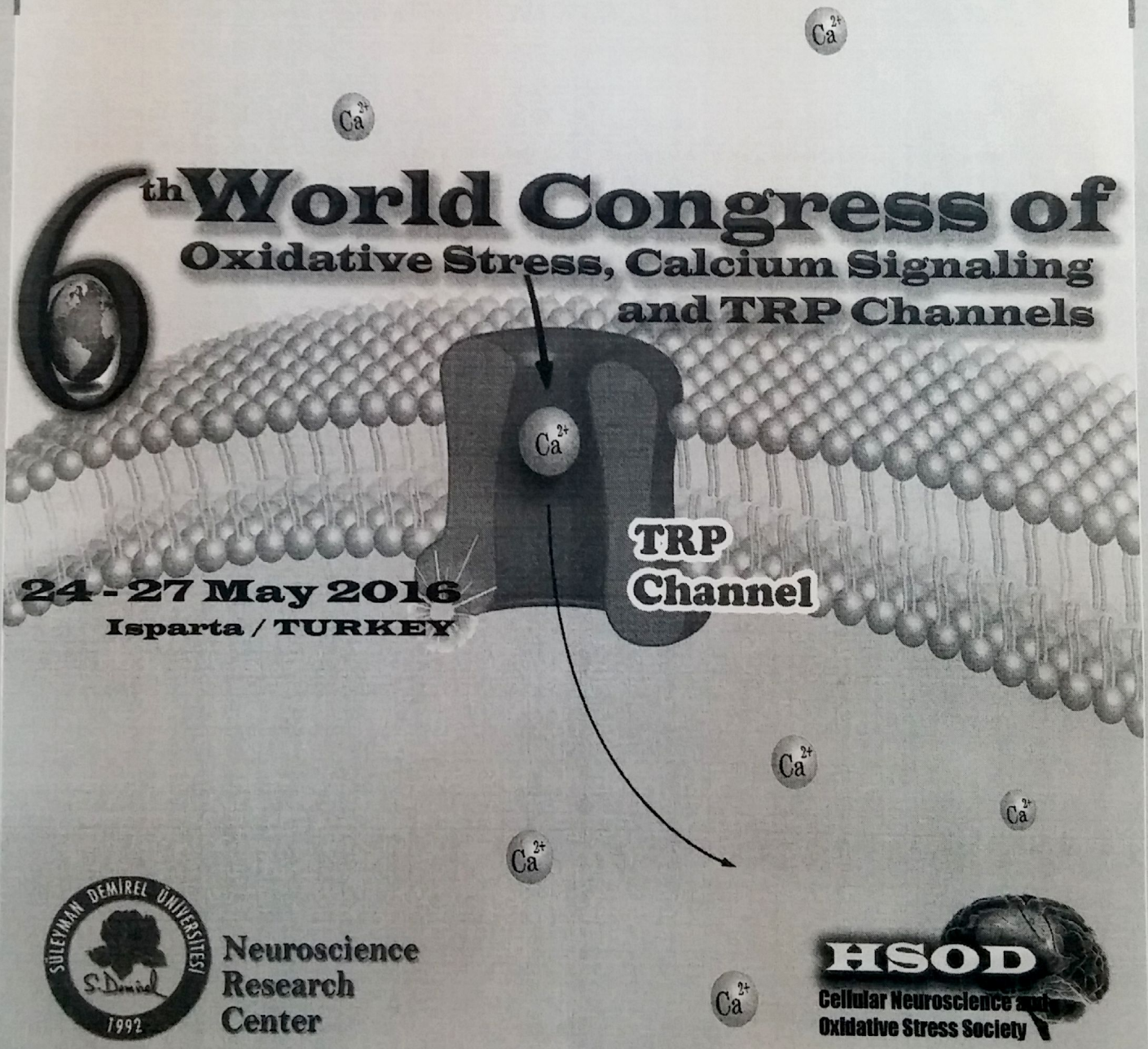
E-ISSN: 2149-7222 (Online)

Journal of Cellular Neuroscience and Oxidative Stress

Former name: Cell Membranes and Free Radical Research



6th World Congress of Oxidative Stress, Calcium Signaling and TRP Channels

24 - 27 May 2016
Isparta / TURKEY

Süleyman Demirel Üniversitesi 1992

Neuroscience Research Center

HSOD
Cellular Neuroscience and Oxidative Stress Society

Volume 8, Number 1, 2016

(feeling of guilt) subscale score. *SAP* level did significantly correlated with the HDRS-8 (retardation). *NP2* level did significantly correlate with the HDRS-14 (genital symptoms).

The main results of this study NP1 and NP2 levels were lower in MD. These molecules were found to be involved for neurogenesis (neuromodulation, synaptogenesis and synaptic plasticity) and neurotoxicity (Alzheimer's disease, in hypoxic-ischemic brain damage) in preclinical and clinic studies (3,4). Low levels of NP1 and NP2 levels may indicate insufficient neurogenesis in MD. The establishment of inflammatory abnormalities and oxidative stress in the etiopathogenesis of MD might also provide the rationale for studies of therapies directed at the modulation of this process.

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Poster No. 35

Oxidative effect of thiacloprid and d-tubocurarine on *Rana ridibunda* gastrocnemius muscle

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Reactive oxygen species (ROS) and reactive nitrogen species (RNS) increase in vertebrates as a result of in vivo exposure to different pesticides (1). Such mediator reactives are: hydrogen peroxide ($H_2O_2^-$) formed by partial reduction of the oxygen; superoxide anion (O_2^-); hydroxyl (OH) radicals and nitric oxide (NO). RNS and ROS can cause damage to biological systems by reacting with, for example, DNA, proteins and cell membranes (2). In this study, the effect of neonicotinoid insecticide thiacloprid and its antagonist d-tubocurarine on the amount of thiobarbituric acid reactive substances and their effects on catalase enzyme activity was investigated in frog gastrocnemius muscle. The isolated gastrocnemius muscle was subjected to four different doses of thiacloprid (1×10^{-3} 1×10^{-4}, 1×10^{-5} and 1×10^{-6} M) for 120 minutes. The muscles were also treated with a $1x10^{-5}$ M thiacloprid and $1x10^{-4}$ M d-tubocurarine mixture, with $1x10^{-6}$ M thiacloprid and $1x10^{-5}$ M d-tubocurarine mixture. The muscle tissues in the control group were maintained in Ringer's solution for 120 minutes. Each dose group was studied with an equal number of preparations (n = 5). Based on the research results, it was determined that $1x10^{-3}$, $1x10^{-4}$, $1x10^{-5}$, $1x10^{-6}$ M thiacloprid and the mixture of $1x10^{-6}$ M thiacloprid and 1x10-5 M d-tubocurarine decreased the amount of thiobarbituric acid reactive substances of the striated muscle tissue compared to the control group ($P < 0.05$). $1x10^{-3}$ ($P < 0.001$) and $1x10^{-4}$ M thiacloprid ($P < 0.05$) decreased the catalase enzyme activity in muscle tissue. This study provides important data for the potential of thiacloprid and d-tubocurarine creating oxidative stress in skeletal muscle, for assessing the possible effects on non-target organisms and for assessing their environmental risks.

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Poster No. 36

Oxidative effect of acetamiprid and d-tubocurarine on frog nerve tissue

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Target region of neurotoxic insecticides are the enzymes in the insect nervous system, ion channels or ion receptors (1). Insecticides have different effects on the target areas and they exhibit considerable diversity in terms of enzyme inhibition, agonist or antagonist effect on receptor and ion channel modulation (2). The aim of this study is to investigate the toxic effects of acetamiprid and d-tubocurarine, on the sciatic nerve of *Rana ridibunda* by using biochemical methods. Frog sciatic nerves were isolated after making them spinal. Four different concentrations of acetamiprid solution (1×10^{-3}, 1×10^{-4}, 1×10^{-5} and 1×10^{-6} M) were applied on sciatic nerves for 120 minutes. In addition, the sciatic nerve tissues were maintained for 120 minutes in the mixture of $1x10^{-3}$ M acetamiprid and $1x10^{-2}$ M d-tubocurarine, the mixture of $1x10^{-5}$ M acetamiprid and $1x10^{-4}$ M d-tubocurarine, the mixture of $1x10^{-6}$ M acetamiprid and $1x10^{-5}$ M d-tubocurarine. Nerve tissues in the control group were maintained in Ringer's solution for 120 minutes. The agonist and antagonist effects were studied in the experimental group an equal number of subjects (n = 5). The results of the colorimetric analysis revealed that application of $1x10^{-3}$ M acetamiprid on sciatic nerve significantly reduced the catalase activity and the level of acetylcholinesterase compared to that of control group. In contrast, it was determined that, the same dose of the insecticide significantly increased the level of malondialdehyde on nerve tissue compared to that of control group. The significant reduction of catalase and acetylcholinesterase in the sciatic nerve tissue due to acetamiprid while causing significant increase of malondialdehyde indicates that this insecticide, depending upon the oxidative stress, causes damage on peripheral nerves in high doses.

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Poster No. 37

Selenium attenuates apoptosis, inflammation, and oxidative stress in the blood, and brains of aged rats with scopolamine-induced dementia

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Decreased acetyl choline concentrations, increased inflammation, apoptosis, and oxidative stress are implicated in the etiology of Alzheimer's disease (AD). A potent antioxidant, selenium might modulate dementia-induced progression of brain and blood oxidative and apoptotic injuries. The present study explores whether selenium protects against experimental dementia-induced brain, and blood oxidative stress, apoptosis levels, and cytokine production in rats with scopolamine (SCOP)-induced dementia.

Thirty-two rats were equally divided into four groups. The first group was used as an untreated control. The second group was treated with SCOP to induce dementia. The third and fourth groups received 1.5 mg/kg selenium (sodium selenite) and SCOP+selenium, respectively. Dementia was induced in the second and forth groups by intraperitoneal SCOP (1 mg/kg) administration.

Brain, plasma, and erythrocyte lipid peroxidation levels as well as plasma TNF-α, interleukin (IL)-1β, and IL-4 levels were high in the SCOP group though they were low in selenium treatments. Selenium and selenium+SCOP treatments increased the lowered glutathione peroxidase activity and reduced glutathione, β-carotene, vitamins A and E concentrations in the brains, erythrocytes and plasmas of the SCOP group. SCOP increased the apoptotic values as active caspase-