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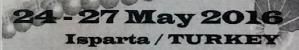
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Oxidative Stress, Calcium Signaling and TRP Channels







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Ca2+

Cellular Neuroscience and Oxidative Stress Society

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Ca²⁺

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(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) 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 dtubocurarine 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 (H2O2) formed by partial reduction of the oxygen; superoxide anion (O2); 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 $(1\times10^{-3}\ 1\times10^{-4},\ 1\times10^{-5}\ and\ 1\times10^{-6}$ 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 dtubocurarine 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 dtubocurarine on frog nerve tissue

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