Brahim Gargouri et al., J Neurol Disord 2017, 5:5(Suppl)
DOI: 10.4172/2329-6895-C1-023

Pyrethroid bifenthrin induces neuronal damage, cognitive impairment associated with oxidative damage in rat's hippocampus: Possible involvement of Nurr1/Nrf2 and NFkb pathways

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Substantial evidence has shown that exposure to pyrethroid pesticides may cause adverse neurodevelopmental outcomes and cognitive impairment, but the underlying neurobiological mechanism is poorly understood so far. In this study, we investigated the alterations of neuronal damage, glial activation oxidative stress and cholinergic dysfunction, and their causal relationship with the cognitive deficits induced by bifenthrin. Our results revealed that exposure to bifenthrin for 8 weeks at doses 6 and 21 mg/kgbw leads to reduction in the levels of acetyl-cholinesterase, Na^+/K^+, $Ca2^+$, $Mg2^+$ ATPases, enzymatic and non-enzymatic antioxidants activities in the hippocampus region. Further, in hippocampus tissue, bifenthrin significantly enhance the mRNA gene expression of nuclear receptor related 1 protein (nurr1), nuclear factor erythroid 2 (nrf2) and nuclear factorkB pathway (NFkB). Oxidative/nitrosative stress was evident in bifenthrin-treated groups by increased malondialdehyde (MDA), protein carbonyls (PCO), and nitrite concentration (NO) in hippocampus. Further, we found that treated rats with bifenthrin exhibited spatial learning and memory impairments and working memory dysfunction compared with control rats. This is also supported by histopathological findings of hippocampus region of rats. Correlational analyses revealed that spatial learning and memory impairments and working memory dysfunction were significantly correlated with the measures of neuronal damage, cholinergic dysfunction and oxidative damage in the hippocampus of treated rats. Moreover, the measures of neuronal damage and central cholinergic dysfunction were significantly correlated with the indexes of oxidative damage in treated rats. The results of the present study suggest that neuronal damage, cholinergic dysfunction and oxidative damage in the hippocampus following bifenthrin exposure could be involved in cognitive deficits.

Biography

Brahim Gargouri is currently working in the Laboratory of Toxicology-Microbiology and Environmental Health at University of Sfax, Tunisia. He has published several original research papers in the reputed and peer reviewed journals.

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