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Poster Presentation

Neurotoxicants and Mechanisms Neurodegenerative in Acrylamide

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Abstract

Many chemicals with broad industrial, pharmaceutical and agricultural application produce a neurotoxic syndrome in humans and experimental animals involving weight loss, skeletal muscle weakness and ataxia. Neurotoxicity is defined as a structural change or a functional alteration of the nervous system resulting from exposure to a chemical, biological or physical agent. Neurotoxicity including Neuronopathia, Axonopathia and myelopathies. The causes of Neuropathies Are Doxorubicin, Methyl Mercury. Axonopathies causes of Gamma-Diketones, β′-Iminodipropionitrile, Acrylamide and Myelopathies causes of Hexachlorophene Tellurium, Lead. Acrylamide (ACR) as a chemical industry is a poison in foods prepared at high temperatures and is the most important neurotoxic agent. Humans that workers in factories are more susceptible for peripheral neuropathies of these toxic agent at high doses. The first symptoms are observed in Pacinian corpuscles, muscle spindles and the nerve terminal. These side effects is result from additions of neurofilaments at the nerve terminal. Developing of Para nodal swellings, cause myelin withdrawal. In additional, Acrylamide lead to sensory axonopathy, Axonal degeneration and peripheral neuropathy. In study mention that, mild ataxia, typical ataxia and hind limb weakness were appeared with 10 mg/kg and 20 mg/kg respectively. The GAP-43 protein marker, is tested for assess of neuronal function that is related to hippocampal neuronal growth, promoting axonal elongation and retaining axonal morphology. Also GAP-43 protein may modulate the transmission of neural signals because of extensively distribution at the axonal terminate. ACR exposure cause inhibition of expression of GAP-43, so may result in disturbance in axonal growth, synaptic terminal vesicles, mitochondria, synaptic inhibition and eventually, terminate the retrograde and anterograde axonal transport. In more recent studies were suggested that ACR, even in its low-dose, can lead to neurological symptoms and nerve terminal degeneration like axonopathy.

Keywords: Acrylamide, Neurotoxicity, Axonopathy

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