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Original Article

PYRAMIDAL LAYER THINNING, SHRUNKEN NEURONS AND DEEP VACUOLATION IN HIPPOCAMPUS DUE TO THE ORGANIC LEAD INDUCED TOXICITY

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ABSTRACT

Lead, a known heavy metal exerts its toxicity on different organ systems of which the neurotoxicity is a considered to a significant consequence. The organic lead exposure on the hippocampus, that plays a significant role in the formation of short and long term memory, navigation and also participates in the limbic system in the brain, was studied. The resulting effects were the thinning of neuron layers, vacuolation and reduction in overall cell population of neurons, thereby showing the primary effect on the pyramidal layers of the hippocampus.

KEYWORDS: Lead acetate, organic lead, neurotoxicity, Charles foster strain, rats, pyramidal layer, hippocampus.

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INTRODUCTION

Heavy metals in different forms in the environment substantiate a major hazard to the different life forms. Among them, the toxicity of lead has been a major cause that has attracted several writings. The clinical effect of lead toxicity called as lumbism, which has been known since ages. Exposure of lead present in the environment can be through ingestion, inhalation or absorption through the skin. The exposure through different routes also has an age related variation, as compared to the human adults were the absorption is 20% in case of ingestion absorption has been found to be 50% in children. The largest proportion of lead absorbed is sequestered by bone followed by liver and kidney.

At cellular level the toxicity is thought to be due to the affinity of lead for cell membrane & mitochondria followed by affecting the mitochondrial oxidative phosphorylation as well as Ca, K & Na ATPases. Further, it also impairs the activity of Ca dependent intracellular messengers and of the brain protein kinase C. additionally, the lead can also alter the gene expression by the translocation in the nucleus by the formation of inclusion bodies.

The most significant result of lead toxicity is its effect on the central nervous system. Several researchers have worked with the effects of lead; the work related to the histological changes is being presented here. In a study by Markov & Dimova (1974) [1] of chronic lead poisoning on Wister rats they observed hypertrophied microglial cells and vascular pericytes in parietal cortex with all other elements appearing intact. Boulding and Krigman (1975) [2] demonstrated the primary toxic effect at neuronal level without