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THE AMELIORATING ROLE OF "α-LIPOIC ACID" SUPPLMENTATION ON ADVERSE EFFECTS INDUCED BY LEAD ACETATE IN BOTH THE HIPPOCAMPUS AND THE CEREBRAL CORTEX OF THE MALE ALBINO RATS

Eman M. Abd-Ella*; Suzan F. El-Sisy, Heba A. El-Dash***
*** Zoology Department, Faculty of Science, Fayoum University, Fayoum, Egypt**
****Branch of Zoology, National Organization of Drugs Control and Research, Giza, Egypt**

Aim of the work: The present study focused on the two brain areas the hippocampus and the cerebral cortex. It was designed to investigate the two possible mechanisms involved in Lead acetate neurotoxicity and the potential protective effects of α-Lipoic acid (α-LA), as an antioxidant against lead-induced neurotoxicity. **Methods:** 40 adult male albino rats *Rattus rattus* (each weighing 130 ± 10 g) were divided into four groups (n=8): **1- Normal control group; C:** Normal male rats fed *ad libitum* and allowed a free access of water, and were kept without any treatments.**2- Initiation group (Lead-Acetate control group; A):** Normal male rats received Lead acetate in an interperitoneal dose of 20 mg/kg.b.wt. daily for two weeks. **3- Treatment group (α-Lipoic acid control group; B):** Normal male rats were intraperitoneally injected with α-Lipoic acid in a dose of 20 mg/kg.b.wt. daily for three weeks. **4- Pre-initiation treatment group (prophylactic group; D):** Normal male rats were intraperitoneally injected with α-Lipoic acid in a dose of 20 mg/kg.b.wt. daily for one week and then received the above-mentioned dose of the Lead acetate for other two successive weeks together with the same dose of α-Lipoic acid.**5- Post-initiation treatment group (therapeutic group; E):** Normal male rats were injected (*i.p.*) once daily with lead acetate (20 mg/kg.b.wt.) for two weeks then were intraperitoneally injected with α-Lipoic acid in a dose of 20 mg/kg.b.wt, daily for successive three weeks. The levels of lipid peroxidation as malondialdehyde (MDA), protein carbonyl content (PCC), reduced glutathione (GSH), nitric oxide level (NO), and serotonin (5- HT) were estimated in the selected brain regions, the hippocampus and the cerebral cortex, of adult male albino rats in the different groups. These biochemical variables were supported by histopathological examinations by using H&E and toluidine blue staining. **Results** showed that administration of Lead acetate led to enhancement of lipid peroxidation MDA, PCC with a concomitant reduction in GSH levels and NO production. In addition, the levels of 5- HT decreased in the hippocampus and the cerebral cortex. However, the administration of α-LA showed a protective effect against Lead acetate-induced neurotoxicity. This rats treated with α-LA showed a corresponding decrease in lipid peroxidation, MDA and PCC levels. Furthermore, there was an increase in brain GSH level, NO production and level of 5- HT in both brain regions the hippocampus and the cerebral cortex. Histological studies showed that Lead acetate caused different degrees of neuronal degeneration, focal gliosis, pyknotic neurons, oedema in meninges, cerebral neuropil vacuolization and degenerations. The rats treated with α-Lipoic acid revealed an improvement in the histopathological alterations induced by Lead acetate. Finally, the data showed that α-LA has an effect when administered as a therapeutic drug more than as a prophylactic one.