EFFECTS OF VITAMIN C SUPPLEMENTATION ON LEAD-INDUCED APOPTOSIS IN ADULT RAT HIPPOCAMPUS

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Lead has caused widespread pollution in the environment. One principle target for lead in the human body is the central nervous system. However, research has demonstrated that neurotoxic effects of lead can be ameliorated by antioxidant agents, such as vitamin C. To investigate the protective effects of vitamin C supplementation against lead-induced apoptosis in the adult rat hippocampus, as well as changes in pro-apoptotic protein Bax expression.

Design, time and setting: A randomized, controlled, animal study was performed at the College of Medicine, Iran University of Medical Sciences from December 2007 to April 2009.

Lead acetate and vitamin C were purchased from Sigma, USA.

Thirty male rats were randomly assigned to three groups, with 10 rats in each group: control, lead, and lead + vitamin C. Rats from the lead group received intraperitoneal administration of lead 20 mg/kg per day for 7 days. Rats from the lead + vitamin C group received ascorbic acid 500 mg/kg Per day in addition to lead acetate (as per the lead group).

Main outcome measures: After 7 days of vitamin C administration, hippocampal cell apoptosis was observed using uranyl acetate and lead citrate stainings, hippocampal Bax protein expression was detected by Western blot analysis, and blood lead levels were measured by atomic absorption spectrometry.

Vitamin C supplementation significantly reduced lead-induced hippocampal cell apoptosis and decreased hippocampal Bax protein expression. However, vitamin C treatment did not significantly decrease blood lead levels. Vitamin C significantly decreased Bax expression and reduced lead-induced hippocampal cell apoptosis.