LEAD-INDUCED OXIDATIVE STRESS IN HUMAN LIVER CARCINOMA (HEPG2) CELLS

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Abstract: Lead is a ubiquitous metal that has been used by humans for more than 3 millennia. Its toxic effects on humans are well documented in history. Most research on lead has focused on its effects on organ systems such as the nervous system, the red blood cells, and the kidneys which are considered to be the primary targets of lead toxicity. However, its molecular mechanisms of toxicity are still largely unknown. In this research, we used HepG2 cells as a model to study the cytotoxicity and oxidative stress associated with exposure to lead nitrate. We hypothesized that oxidative stress plays a key role in lead nitrate induced cytotoxicity. To test this hypothesis, we performed both MTT [3-(4,5-dimethylthiazol-2-yl)-2, 5-diphenyltetrazolium bromide] assay and trypan blue exclusion test for cell viability and the thiobarbituric acid test for lipid peroxidation. Data obtained from the MTT assay indicated that lead nitrate significantly reduced the viability of HepG2 cells, showing a LD50 value of about 28 μg/mL upon 48 hours of exposure, indicating a dose-dependent response. Similar trend was obtained with the trypan blue exclusion test using the hemacytometer to count the cell manually. Data generated from the thiobarbituric acid test showed a significant (p ≤ 0.05) increase in MDA levels in lead nitrate-treated HepG2 cells compared to control cells. Lead nitrate treatment significantly increased cellular content of reactive oxygen species (ROS), as evidenced by the increase in lipid peroxidation by-products. Taken together, these results indicate that lead nitrate is highly cytotoxic to HepG2 cells. This cytotoxicity is found to be mediated by oxidative stress, a biomarker of cellular injury.

Keywords: Lead nitrate, lipid peroxidation, MDA, cytotoxicity, and HepG2 cells

Acknowledgements: This research was financially supported in part by a grant from the National Institutes of Health (Grant No. 1G12RR13459), through the RCMI Center for Environmental Health, and in part by a grant from the U.S. Department of the Army (Cooperative Agreement No. W912H2-04-2-0002) through the CMCM Program at Jackson State University. The authors thank Dr. Abdul Mohamed: Dean Emeritus, and Dr. Mark Hardy: Interim Dean of the College of Science, Engineering, and Technology for their technical support in this research.