A STUDY OF THE APOPTOTIC MECHANISMS OF LEAD TOXICITY IN HUMAN LEUKEMIA (HL-60) CELLS

Jessica N. Milner1, Clement G. Yedjou2 and Paul B. Tchounwou2

1University of Mississippi Medical Center, 2500 North State Street, Jackson, MS 39216, USA
2Cellomics and Toxicogenomics Research Laboratory, NIH-Center for Environmental Health, College of Science, Engineering and Technology, Jackson State University, Jackson, MS 39217, USA

Abstract: Lead exposure represents a medical and public health emergency, especially in children consuming high amounts of lead-contaminated flake paints. It may also cause hematological effects to people of all ages. Recent studies in our laboratory have indicated that apoptosis may be associated with the lead-induced oxidative stress and DNA damage. However, the mechanisms underlying its effect on lymphocytes are still largely unknown. Therefore, the aim of the present study was to investigate the apoptotic mechanisms of lead nitrate \( [\text{Pb(NO}_3\text{)}_2] \) using HL-60 cells as a test model. HL-60 cells were treated with different concentrations of \( \text{Pb(NO}_3\text{)}_2 \) for 24 h prior to cell viability assay and flow cytometry assessment. The results obtained from the trypan blue exclusion test indicated that at very low concentration, \( \text{Pb(NO}_3\text{)}_2 \) has no effect on the viability of HL-60 cells. A significant (\( p < 0.05 \)) decrease in cell viability was observed when exposed to high level of \( \text{Pb(NO}_3\text{)}_2 \). Data generated from the flow cytometric assessment indicated that \( \text{Pb(NO}_3\text{)}_2 \) exposure significantly (\( p < 0.05 \)) increased the proportion of annexin V positive cells (apoptotic cells) compared to the control. \( \text{Pb(NO}_3\text{)}_2 \) induced apoptosis of HL-60 cells was associated with the activation of caspase-3. In summary, these studies demonstrated that \( \text{Pb(NO}_3\text{)}_2 \) represents an apoptosis-inducing agent in HL-60 promyelocytic leukemia cells and its apoptotic mechanism functions, at least in part via, induction of phosphatidylserine externalization and caspase-3 activation.

Key words: Lead nitrate, HL-60 cells, toxicity, annexin V, caspace-3

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