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Effects of depleted uranium on thymocytes

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Depleted uranium (DU) is a by-product of the uranium-235 radionuclide enrichment process for nuclear reactors or nuclear weapons. Recent combat applications of DU alloy resulted in acute exposure of humans to DU dust, vapor, or aerosol. Also, between 1958 and 1984 National Lead Industries made uranium products in Colonie, NY, contaminating the surrounding residential area. Although there has been minimal scientific evaluation, exposure to DU in war sites was implicated in increased cases of birth defects and cancer among exposed people. In this study, we have examined the effects of uranyl acetate on acutely dissociated thymocytes of mice, using flow cytometry and fluorescence spectrophotometry. Fluorescent probes Fluo-3-AM, H2DCFDA (2',7'-dichlorofluoresceine diacetate), and DiBAC4(3) (bis-(1,3-dibutylbarbituric acid)-trimethine oxonol) were used to estimate intracellular calcium concentration, reactive oxygen species (ROS), and membrane potential, respectively. Membrane fluidity was assessed by measuring steady-state fluorescence polarization of diphenylhexatriene. Uranyl acetate induced death of thymocytes, as monitored by use of the DNA-binding dye, propidium iodide, in a time- and dose-dependent fashion. Exposure to uranyl acetate was accompanied by a decrease in membrane potential and an increase in ROS and intracellular calcium levels. Our results demonstrate the presence of toxic effects of DU in acutely dissociated thymocytes of mice. In future studies we intend to explore further the mechanisms of DU toxicity.