Oxidative damage by bisphenol A induced lipid peroxidation and apoptosis

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It is very important to understand the ROS generation of estrogen-like chemicals. Of such chemicals, we were especially interested in bisphenol A for its wide spreadness in environmental and structual similiarity with aneuploidogenic stilbene estrogen diethylstilbestrol. The purpose of the this study was to evaluate the lipid peroxidation and DNA fragmentation by bisphenol A in the presence of a rat liver S9 mix containing cytochrome P 450 enzymes and Cu(II) in HaCaT cell lines. The specific content of malondialdehyde, an end product of lipid peroxidation, was also found to increase with concentration. The fragmentation of intact DNA, a parameter of apoptotic cell death, was evaulated qualitatively by agarose gel electrophoresis analysis and quantitatively by diphenylamine reation method. BPA induced apoptotic cell death in a dose-dependent manner. When HaCaT cells were exposed to 50uM BPA for 48h, the DNA fragmentaion was significantly increased to 54%. The effect of radical scavenger on the apoptotic cell death induced by BPA was investigated. The DNA fragmentation induced by BPA was significantly inhibited by addition of ROS scavenger to the culture medium. Also we examined the enzyme activities of Cu,Zn-SOD, Mn-SOD, catalase, and GPx in the cells. The activities of Cu,Zn-SOD, glutathione peroxidase, Catalase were found to decrease with concentration. However, the activity of Mn-SOD were unchanged. This indicated that elevated oxidative stress caused by an imbalance between the production and removal of ROS and free radicals occured in cells.