Different cell death mechanisms and gene expression in human cells induced by pentachlorophenol and its major metabolite, tetrachlorohydroquinone

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Abstract

Pentachlorophenol (PCP) and its salt are used extensively as biocide and wood preservative. Due to improper disposal, PCP has become an environmental pollutant and is now considered to be ubiquitos. Metabolic studies carried out in rodents or human liver homogenate have indicated that PCP undergoes oxidative dechlorination to form tetrachlorohydroquinone (TCHQ). The cytotoxicity, cell death mechanisms and gene expression of PCP and TCHQ are investigated in human liver and bladder cells and show that TCHQ induces apoptosis and DNA genomic fragmentation in bladder cells but not liver cells. No apoptotic features could be induced by treatment of PCP in both cell lines. The concentrations of PCP required to cause 50% cell death in T-24 and Chang liver cells were 5-10-fold greater than the concentrations of TCHQ. Several gene products are important in controlling the apoptotic and necrotic processes. Of these, hsp 70, CAS, bcl-2 and bax were studied. The expression of the hsp70 gene increased significantly (2-3-fold) in cells treated with TCHQ. However, no significant change was found in the cells treated with PCP. The expression of CAS gene decreased significantly in T-24 cells treated with both TCHQ and PCP. Whereas, no significant change was found in Chang liver cells with the same treatment. In addition, the expression of the bcl-2/bax protein decreased significantly in these two cell lines treated with TCHQ but not PCP.