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Organochlorine pesticides and polychlorinated biphenyls(PCBs) are ubiquitously distributed in the environment and belong to a group of contaminants whose occurrence in the environment is of a serious concern to environmental chemists and toxicologists. This is due to their resistance to degradation in the environment as well as their potential toxicity. The occurrence of organochlorine pesticides and PCBs in the environment and subsequently in parts of the food chain, resulting in the intake of these compounds by man and animal. The measure of the levels of organochlorine pesticides or polychlorinated biphenyls (PCBs) in tissues or blood of human populations are good markers in determining the extent of exposure and in the evaluating the hazards. So, most countries have conducted initial monitoring programs to determine orgahochlorine pesticides and PCBs in human tissues. But few report has been presented in Korea. In this study, organochlorine pesticides(α-BHC, β-BHC, ɣ-BHC, δ-BHC, p,p'-DDT, p,p'- DDD, p,p'-DDE, endrin, dieldein, aldrin) and marker PCBs(PCB nos. 28, 52, 101, 118, 138, 153, 180) were determined in human blood, adipose tissue and liver tissues collected at autopsy of 10 men and 10 women, by using GC/ECD. From the results, the significant differences in the levels of organochlorine pesticides or PCBs between sexes, districts where they had lived and ages were also investigated.

[PA3-14] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Effects of bisphenol A on T cell and B cell population and cytokine production of splenocytes.

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Bisphenol A is a monomer used in the manufacturing epoxy resins and polycarbonates, and may be released into the environment through its use and handling. This study was designed to investigate the effects of BPA on the T cell population and B cell , and production of IL-4 and IFN-r. Female ICR mice were administered to various concentrations(100, 500, 1000 mg/kg/day) of BPA for 30 days. After 2 days expose, mice were sacrificed.
Helper T cell population in spleen from exposed to BPA was decreased with increase in B cell population. The cytokines production of Con A-stimulated spleen cell from the BPA exposed mice was decreased. When normal splenocytes were activated with Con A in the presence (1, 10, 25, 50, and 100 uM) or absence of BPA, BPA suppressed cytokines production at 50, 100 uM. These results revealed immunotoxicity of BPA.

[PA3-15] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Bisphenol A-metabolites induces Oxidative DNA damage and reduced cell proliferation

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Bisphenol A, a monomer of polycarbonate and epoxy resins, has been detected in canned food and human saliva. BPA stimulate cell proliferation and induces expression of estrogen-response genes in vitro. This report considers the hypothesis that BPA is converted in vivo to hydroxylated metabolites with enhanced estrogenicity and cytotoxicity. The purpose of the this study was to evaluate the cytotoxicity

and cell proliferation of bisphenol A in the presence of a rat liver S9 mix containing cytochrome P 450 enzymes and Cu(II). In the present study, we found that BPA in combination with Cu(II) exhibited a enhancement in cytotoxicity, which was inhibited by reacitve oxygen species scavenger. For cell proliferation assay MCF-7 cells were seeded on a 96-well multi-well-plate at 1.5 x 10(3) cells per well. After 24hr cultivation, the S9 mix and Cu(II) was added to the wells as an S9 mix group (+S9), and medium was added to the other wells as a none-S9 mix group (-S9), then 5 different concentrations of various BPA were added to each well. After 5 days, a sulforhodamine B (SRB) assay was conducted to measure cell proliferation. +S9 mix group enhanced the proliferation of MCF-7 cells at much lower concentrations than -S9 mix group which was inhibited by the ROS scavenger. These results suggest that reacitve oxygen species reacts with Cu(I) leading oxidative stress. Also the formation of reacitve oxygen species induced by BPA was dose-dependently by inhibited by tamoxifen, which suggests that the effect of BPA was estrogenic action via estrogen receptors.

[PA3-16] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Toxicity Identification Evaluation of Water Pollution using in vitro bioassay

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So far, investigation of environmental pollution has been achieved in field study. This remains the most exhaustive approach, current dimensions of environmental researches and their inherent complexity require that relatively inexpensive and simple laboratory procedures are developed to make possible the screening of large numbers of sites and samples. At this point, micro-bioassay has been highlighted. The purpose of this study is to evaluate the water pollution using micro-bioassay. Micro-bioassay methods were optimized and validated for the sensitive and quantitative determination of total toxic effects of the water samples. EROD bioassay was focused to detect PAHs, PCBs and dioxinlike components in the water. The EROD bioassay was executed in rat hepatoma cell line, H4 II E cell lines. 50L of river water was adsorbed using XAD-2 resin column. Pollutants adsorbed to the XAD-2 resin were extracted by elution with methanol (sample I), and with ethyl acetate (sample II). Toxic effects of extracts were determined by micro-bioassay methods.

[PA3-17] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

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