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Estrogens are potent mitogens in a number of target tissue including the mammary gland where they play a pivotal role in the development and progression of mammary carcinoma. Many endocrine disruptors (EDs) show the estrogenic effect. As the effects of EDs are reported to be main causes of hormone-related cancers such as breast cancer among women, we studied the effects of EDs using mouse mammary gland organ culture (MMOC) model. Also, the expression of estrogen receptor and p53 in the preneoplastic lesion was measured by using the flow cytometry. The research on more parameters related to the breast cancer will help in proving the mechanism of preneoplastic lesion by EDs. Moreover, it will help develop the antiestrogenic agent to inhibit the EDs activity.

[PC1-40] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

Effects of PCBs on human mast cell line HMC-1.

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Polychlorinated biphenyls (PCBs) are widely spread environmental contaminants consisting of chemical mixtures containing many of the 209 possible congeners. The potential immunomodulatory properties of PCBs have been the subject of extensive experimental investigations. The available evidence indicates that the immune system is a target for PCBs and is perhaps one of the most sensitive indicators for adverse PCB induced health effects.
Mast cells are the primary effector cells of immediate hypersensitivity reactions in humans and their numbers are increased in a broad spectrum of pathologic conditions.
We have examined effects of PCBs on human mast cell line HMC-1. In this study, expressions of xenobiotic responsive genes were analyzed to examine their molecular mechanisms in 2,2',4,4',5,5'-hexachlorobiphenyl (2,2',4,4',5,5'-hexaCB)-treated HMC-1. Reverse transcriptase-polymerase chain reaction (RT-PCR) and immunoblot analysis were performed to detect altered expressions of genes associated with 2,2',4,4',5,5'-hexaCB responses. The RT-PCR analysis showed that interleukin-6 (IL-6) and cyclooxygenase-2 (COX-2) genes were well expressed. Whereas interleukin-1β (IL-1β) and interleukin-4 (IL-4) did not expressed. In case of tumor necrosis factor-α (TNF-α) and aromatic hydrocarbon receptor (AhR), gene expressions were decreased by dose dose- and time-dependent manner. However transcription levels of AhR nuclear translocator (ARNT) were not changed.

[PC1-41] [10/20/2000 (Fri) 15:30 - 16:30 / [Hall B]]

Molecular Mechanism of Dioxin-induced Endocrine Disruption through Induction of Oxidative Estrogen Metabolism

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2,3,7,8-Tetrachlorodibenzo-para-dioxin (TCDD; dioxin), the prototype agonist of the aromatic hydrocarbon (Ah) receptor, has a marked effect on estrogen metabolism in MCF10A cells by induction of human cytochrome P1A1(CYP1A1) and P450 1B1 (CYP1B1), which are responsible for hydroxylation of 17beta-estradiol (E_2) at C-2 and C-4 positions, respectively. The resulting catechol estragens, 2-hydroxyestradiol ($2OHE_2$) and 4-hydroxyestradiol ($4OHE_2$) have been