

Recent industrial society has human widely exposed to PAHs that are coming from the incomplete combustion of organic material as widespread environmental contaminants. Biological activities of PAHs are not known although PAHs are considered as carcinogens. The mechanism of action of PAHs has been studied extensively, however it is not clear how PAHs turn on CYP1A1 in human breast cancer. Our laboratory have been studied the effect of PAHs in the human breast cancer cell MCF-7. In this study, we examined the ZR-75-1 human breast cancer cells as a new system to evaluate bioactivity of PAHs and to compare the PAH action with that of MCF-7 cells. ZR-75-1 human breast cancer cell line is response to estrogen and progesteron. We have been able to establish long term culture system of this cells then used for the study to the effect of 13 different PAHs and environmental samples. We demonstrate that PAHs induced the CYP1A1 promoter and 7-ethoxyresolufin O-deethylase(EROD) activity in a concentration-dependent manner. RT-PCR analysis indicated that PAHs significantly up-regulate the level of CYP1A1 mRNA. Some of PAHs showed stronger stimulatory effect on CYP1 gene expression than TCDD. Apparently, ZR-75-1 cells have Aryl hydrocarbon receptors, therefore it would be good experimental tool to study the cross-talk between PAHs and steroid actions.

[PA4-32] [04/17/2003 (Thr) 14:00 - 17:00 / Hall P]

Environmental endocrine disruptors and endometriosis

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Endometriosis is classically defined as the growth of endometrial glands and stroma at extra-uterine sites. Although it is a common gynecological problem accompanied by chronic pelvic pain, infertility, and adhesion formation, the etiology of this disease is unknown. Endometriosis pathogenesis may involve endocrine and immune dysregulation since uterine endometrial growth is regulated by sex hormone in concert with bioactive mediators produced by uterine immune and endocrine cells. thus, exposure to environmental toxicants disrupting endocrine and immune responses potentially affect the development and progression of endometriosis.

In this study, we attempted to identify the possible association between dioxin like compounds (such as TCDD, PCDDs, PCDFs, and PCBs) and the occurrence and severity of endometriosis using CALUX (Chemically Activated Luciferase eXpression) bioassay method. We analyzed the serum levels of dioxin like compounds in the endometriosis patients (n=46) and control patients with similar symptoms (n=14). Among them, adipose tissues of 10 cases were analyzed by high resolution GC/MS for validation of CALUX bioassay. The CALUX TEQs significantly correlated with the total TEQs determine by GC/MS ($r^2 = 0.96$). So we demonstrated that CALUX bioassay is a rapid, sensitive and quantitative assay for biomonitoring of dioxin like compounds from small volume of blood. This study showed statistically significant association between exposure to dioxin like compounds and the occurrence of endometriosis ($p < 0.003$). The mean TEQ of control patient was 0.144 $\mu\text{g TEQ/L}$ and the mean TEQ of endometriosis patient was 0.321 $\mu\text{g TEQ/L}$. After adjusting confounding factor, we found that the higher stage of the endometriosis, the higher level of CALUX TEQ. The TEQs of endometriosis I, II, III, and IV was 0.213 $\mu\text{g TEQ/L}$, 0.284 $\mu\text{g TEQ/L}$, 0.352 $\mu\text{g TEQ/L}$, and 0.450 $\mu\text{g TEQ/L}$, respectively.

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The Inhibitory Effect of Zinc on the Cadmium-Induced Apoptosis in Human Breast