

3-MCPD induced spermatotoxic effect, which caused a antifertility on male.

[PA4-9] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Effects of zinc and resveratrol on cadmium-induced apoptosis and cell arrest in MCF-7 and MDA-MB-231 cells

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Cadmium, a human carcinogen, can induce apoptosis in various cell lines. Despite extensive research, the mechanisms of cadmium-induced apoptosis are poorly understood, and its toxicity and estrogenic potential in human are not clear. This study was performed to investigate the apoptotic activities of cadmium on two human breast cancer cell lines: MCF-7 cells, an estrogen receptor (ER) positive cell line, and MDA-MB-231 cells, an ER negative cell line. Both cells were treated with CdCl₂ 100µM for 12hrs, and the apoptosis was determined by DNA fragmentation, DAPI staining, and expression of caspase-9. Flow cytometric analysis showed that MCF-7 cell was arrested in S phase and induced expression of p21 and p27 after treated CdCl₂. The expression of ER-α, ER-β, pS2 and activities of antioxidative enzymes such as superoxide dismutase, catalase, glutathion reductase were determined also. Cadmium induced apoptotic cells dose-dependently, increased S phase cell population and decreased antioxidative enzyme activities in both cells. The expressions of ER-α, ER-β and pS2 were increased in MCF-7 cells, and the expression of ER-β and pS2 were increased in MDA-MB-231 cells. Co-treatment of zinc (100µM, 12hrs) or preincubation with resveratrol (25µM, 12hrs) decreased the cadmium-induced apoptotic cell numbers and recovered the antioxidative enzyme activities in both cells. Our data showed that the cadmium induced apoptosis and cell cycle arrest in human breast cancer cells by oxidative stress and antioxidants such as zinc and resveratrol inhibited cadmium-induced apoptosis. and cell arrest.

[PA4-10] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Analysis of 3-monochloro-1,2-propanediol(3-MCPD)in soy sauce products in Korea

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3-Monochloro-1,2-propanediol(3-MCPD) was analyzed in soy sauce products commercially available in Korea. A total of 24 samples were collected and 3-MCPD was determined by GC/MS. Sources of 24 samples were classified by manufacturing methods as naturally brewed(NB), acid hydrolyzed(AH) and mixed(M=NB+AH) soy sauces. 3-MCPD was not detected in NB soy sauce products (< 0.01 ppm, mg/kg) whereas AH and M soy sauce products showed a wide range of 3-MCPD contamination(0.01 ~ 2.038ppm). The contaminated levels of 3-MCPD in soy sauce products were higher than the permissible or tentative permissible level of 3-MCPD in both European Community(0.02 ppm) and Korea(0.3 ppm). These data suggest that 3-MCPD levels contaminated in soy sauce products in Korea were shown to be too high and should be reduced to as low a level technologically feasible to protect Koreans from the exposure to toxic chemical, 3-MCPD.

[PA4-11] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Effects of Saururus chinensis Baill on Atherosclerosis and Lipidperoxidation in 2,3,7,8-tetrachlorodibenzo-p-dioxin Treated Rats.

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Saururus chinensis Baill (Saururaceae) is a perennial plant that has been used in the treatment of edema, jaundice and gonorrhea in Korean folk medicine. This study was carried out to investigate the inhibitive effects of

Saururus chinensis Baill (SCB) on lipid metabolism in Sprague-Dawley rat (SD-rat) acutely exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). After 7 days from TCDD (1 µg/kg) injection, SCB (200 mg/kg) was administered into rats intraperitoneally for 4 weeks. We examined the lipid parameters by measuring the levels of Total Cholesterol, Triglyceride (TG), HDL-Cholesterol, and LDL-Cholesterol in serum and Malondialdehyde (MDA) in liver tissue of rats. Cholesterol was significantly elevated in TCDD-treated abnormal group (TTA). The higher level of HDL-Cholesterol was found in Saururus chinensis Baill and TCDD administered (STT) group, which showed the lower levels of Total-Cholesterol and LDL-Cholesterol. TG content in the STT group was inhibited compared to TTA group by 18.90%. MDA content in the STT group was inhibited compared to TTA group by 17.14%. These findings indicate that Saururus chinensis Baill may have a protective effect against TCDD-treated lipid peroxidation in rats.

[PA4-12] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

PC12 and cortical neuron cell death by Bisphenol A through ERK signal pathway : role of estrogen-receptor β

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Bisphenol A (BPA) mimics estrogen and its activity is one third to one quarter that of estradiol. BPA, an ubiquitous environmental contaminant has been shown to cause development reproductive toxicity and carcinogenic effect. BPA may do physiological action through ER α and ER β which are expressed in central nerve system. We previously found that exposure of BPA to immature mice resulted in behavioral alternation, suggesting that overexposure of BPA could be neurotoxic. In this study, to further investigate molecular mechanisms by which BPA induced behavioral alternation, we examined whether BPA may interfere differentiation of undifferentiated neuronal cells, thereby modify the behavioral development. BPA concentration dependently increased vulnerability (increased cell viability and decreased differentiation) of undifferentiated PC12 cells and undifferentiated neurocortical cells isolated postnatal (Day 1) rat brain. These effects were prevented in the presence of estrogen receptor-beta antagonists, ICI 182, 780 and Tamoxifen. The greater increase of cell vulnerability was also found in the PC12 cells overexpressing ER- β . The increased vulnerability by BPA were mediated by phosphorylation of ERK. Activation of ERK signaling was further augmented in the PC12 cells overexpressing ER- β . The present data show that BPA dose dependently increased neuronal cell vulnerability through activation of ERK signals, and this effect was associated with ER- β receptor. This study demonstrated that exposure of certain level of BPA may interfere normal neuronal cell differentiation, and thereby alter behavioral development.

[PA4-13] [2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function]

Aspirin Inhibits Dimethylnitrosamine-Induced Liver Damage in Rats

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Aspirin and aspirin-like nonsteroidal antiinflammatory drug have been the mainstay of therapy for rheumatoid arthritis. In this study, we investigated the hepatoprotective effect of aspirin on the dimethylnitrosamine (DMN)-induced liver damage in rats. Oral administration of aspirin (7.5, 15 mg/kg daily for 4 weeks) into the DMN-treated rats remarkably prevented the elevation of serum alanine transaminase, aspartate transaminase and alkaline phosphatase, and bilirubin levels. Aspirin also increased serum protein level and reduced the hepatic level of malondialdehyde in DMN-treated rats. Furthermore, DMN-induced elevation of hydroxyproline content was reduced by the treatment of aspirin and which result was consistent with a histochemical analysis of liver tissue stained with Sirius red. In conclusion, these results demonstrate that the in vivo hepatoprotective effect of aspirin against DMN-induced liver injury, and suggest that aspirin may be useful in the prevention of liver damage.