

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.

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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.