female (po; control, 0.1, 0.4, and 0.8 ㎎/㎏ TBF for 2 days) and male (po; control, 0.1, 0.5, and 1.0 ㎎/㎏ TBF for 3 days) rats were sacrificed 24 hr after administration. In *experiment II*, 48-days-old female and male rats (po; 0.5 ㎎/㎏ TBF for 2 days) were sacrificed 0, 6, 12, 24 and 72 hr after the last dose. In *experiment III*, 48-days-old female and male rats (po; control or 0.5 ㎎/㎏ TBF for 2 days) were sacrificed 12 hr after last dose. *Result*: In *experiment I*, mortality was 25% in 1.0 ㎎/㎏ TBF group of male and 50% in 0.4 and 0.8 ㎎/㎏ TBF groups of female rats. AchE was significantly decreased only in the frontal and entorhinal cortexes of female rats receiving 0.4 or 0.8 ㎎/㎏ TBF. In *experiment II*, no death was observed in female or male rats. The maximal inhibition in the brain regions or plasma was 2 or 3-fold higher in female, which occurred 6 or 12 hr after last dose. In *experiment III*, mortality was 20% and 0% in female and male rats, respectively. AchE activity in the frontal cortex was significantly inhibited by 60-65% in female and 10-15% in male rats treated with TBF. These results show that female is more sensitive to the inhibition of AchE or mortality than male rats, indicating that TBF causes sexual dimorphic effects on AchE inhibition or mortality in age-matched rats.

[PA4-28] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

Changes of serum immunoglobulin in the subacute oral administration of bisphenol A

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Bisphenol A(BPA), a monomer used in the manufacturing epoxy resins and polycarbonates, has been reported to induce estrogenic activity, it has been considered as an environmental endocrine disruptor. But the immunomodulatory effects of BPA exposure have not been systemically evaluated. We investigated whether BPA effects on the ability of immunoglobulin(Ig) production of mice. To initiate investigation of BPA-induced alterations of the immune system, BPA at dose of 100, 500, 1000 mg/kg b.w./day with or without OVA-antigen for 30 days were orally administered to female ICR mice. Mice were sacrificed and serum was collected on day 2 following administration of BPA for 30days. Total IgG1, total IgG2a, total IgE, OVA-specific IgG1, OVA-specific IgG2a, and OVA-specific IgE in serum were determined and compared with those of non-treated mice.
In the groups of BPA with OVA antigen, total IgG1, total IgG2a, total IgE,
OVA-specific IgG1 and OVA-specific IgG2a were significantly decreased at dose of 500mg/kg/day and 1000mg/kg/day. However, in mice treated with BPA alone, total IgG1,and IgG2a were not much altered and total IgE was significantly increased at dose of 1000mg/kg/day. These results demonstrated the BPA modulates the production of immunoglobulin.

[PA4-29] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

In utero exposure to 2, 3', 4, 4', 5- Pentachlorobiphenyl (PCB 118) alters postnatal reproductive development in female rat

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Our previous study demonstrated that 2, 3', 4, 4', 5- Pentachlorobiphenyl (PCB 118) showed an antiestrogenic activity in vitro and in vivo. In the present study, we examined the effect of PCB 118 on postnatal reproductive development in female rats. PCB 118 (0.001, 0.01 or 0.1 ㎎/㎏/day) was administered to pregnant female SD rats from gestation day (GD) 6 to 18 via subcutaneous injection, and developmental parameters such as vaginal opening were determined. PCB 118 significantly delayed vaginal opening of female offsprings at dose of 0.1 ㎍/㎏/day, whereas had no effects on body weights. In addition, in utero treatment of PCB 118 caused significant decreases in serum levels of E2, T3 and T4 in female offsprings at certain doses on postnatal day (PND) 22. Our data of results indicate that in utero exposure to PCB 118 may alter postnatal reproductive development in female rat through its antiestrogenic activity.

[PA4-30] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]