Induction of CYP1A1 in the liver of rat offspring by in utero and lactational exposure to 3,3',4,4',5-pentachlorobiphenyl (PCB 126)

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In this study, we investigated the induction of hepatic CYP1A1 in rat offspring by in utero and lactational exposure to 3,3',4,4',5-pentachlorobiphenyl (PCB 126), a congener of coplanar PCBs, using immunohistochemistry during the period between postnatal day (PND) 1 and PND 24. In addition, mRNAs encoding CYP1A1, aryl-hydrocarbon receptor (AhR) and aryl-hydrocarbon receptor nuclear translocator (Arnt) were semi-quantified using RT-PCR. For this study, adult female Sprague-Dawley rats copulated and conceived after daily oral administration for 2 weeks of either corn oil or 1 and 3 µg/kg/day of PCB 126. Daily administration was continued until 20 days after delivery through the gestational period. In this procedure, cumulative maternal exposure to PCB 126 until weaning was estimated to be 57-68 µg/kg in the 1 µg/kg/day-exposed group and 171-186 µg/kg/day in the 3 µg/kg/day-exposed group.

By immunohistochemistry, CYP1A1 was not detected in the liver of control rats. However, it diffusely distributed in the hepatic lobules on PNDs 1, 5 and 15, and then localized in the centrilobular hepatocytes on PND 24 in the rats of both exposed groups.

The RT-PCR study showed that CYP1A1 mRNA was present as early as on PND 1 in the rats of both exposed groups and consistently expressed until PND 24 examined, and there were no significant differences in the levels of the mRNA on PNDs 1, 5, 15 and 24 in both exposed groups. In the control rats, CYP1A1 mRNA was not detected. The mRNAs of AhR and Arnt were expressed in the liver of both exposed and control rats. There were no differences in the levels of these mRNAs increased with age. These findings suggest that maternal exposure to PCB 126 may consistently affect the progeny during the prenatal and postnatal periods.

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