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Effect of Polychlorinated Biphenyls (Aroclor 1254) on Rhythmic Pituitary-Adrenal Function

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Numerous studies have documented the general toxicity of the polychlorinated biphenyls (PCBs) and a sizeable literature has accumulated concerning the toxic effects of PCBs on a variety of homeostatic systems (FISHBEIN, 1974). However, current information regarding the effects of PCBs on rhythmic endocrine function is quite limited. Thus, the present study was undertaken to determine whether circadian periodicity in pituitary-adrenal function is affected by exposure to PCBs.

MATERIALS AND METHODS

Twenty-four adult male Sprague-Dawley rats (Charles River) housed 3/cage and acclimated to controlled lighting (fluorescent illumination from 0400 to 1800) and temperature (26°C ± 2°C) for two weeks were divided according to a randomized block design into three groups: control, vehicle-injected, and PCB-injected (PCB). PCB rats were injected intraperitoneally (i.p.) with Aroclor 1254 (1000 mg/kg, B.W.) in 0.5 ml corn oil. Vehicle-injected rats received 0.5 ml corn oil and C rats served as non-treated controls. After PCB and vehicle injections, rats were housed one/cage in a quarantined biohazard animal facility and maintained on 14 hours light, 10 hours dark as described above. In all cases food and water were available ad libitum.

Three weeks after injections jugular blood samples were obtained from all animals for assessing rhythmic fluctuations in pituitary-adrenal activity. Beginning at 0600, 1800, or 0200 each rat was removed from its home cage to an adjoining room where it was quickly anesthetized with methoxyfluorane (Penthrane). The jugular vein was then exposed and 0.5 ml of blood was withdrawn into a heparinized syringe within 3 min from the time of cage opening. Blood samples were centrifuged and plasma collected for determination of corticosterone concentration.

Six weeks after injections the rats were sacrificed via rapid decapitation and morning non-stress blood samples were obtained for corticosterone analysis. Additionally, the adrenal glands were removed, cleaned of surrounding tissue, weighed to the nearest 0.2 mg and frozen for subsequent corticosteroid determination. Adrenal and plasma concentrations were determined using the fluorometic assay of GLICK et al. (1964). Statistical probabilities for plasma corticosterone concentrations were determined by analysis of variance for repeated measures. All other data were evaluated by one-way analysis of variance.

RESULTS AND DISCUSSION

Aroclor 1254 did not alter the periodicity in plasma corticosterone concentration three weeks after injection (Figure 1). All groups showed significant diurnal variation in plasma corticosterone levels and corticosterone levels of PCB rats were not different from control or vehicle groups at any sampling period.

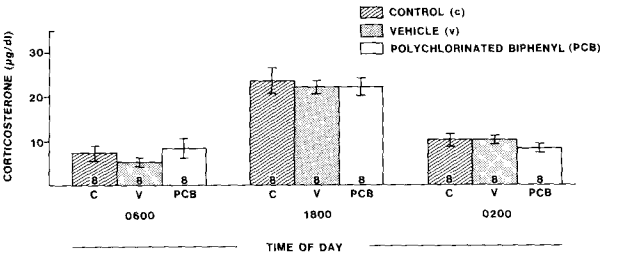

Fig. 1 Effect of Aroclor 1254 on diurnal variation in plasma corticosterone levels. The number of animals per treatment group is indicated at the base of the columns. Vertical lines indicate the S.E.

Six weeks after PCB treatment morning plasma corticosterone levels did not differ among the three groups ($p > 0.05$) and the levels were not different from the 0600 levels shown in Fig. 1. Consistent with the plasma corticosterone levels, the relative adrenal weights and the adrenal corticosterone concentration of PCB rats were not different from control or vehicle groups ($P > 0.05$) (Table I).

Table 1. Effects of PCBs (Aroclor 1254) on Plasma and Adrenal Corticosterone and Relative Adrenal Gland Weights Six Weeks After Injection

Groups(N*)	Plasma Corticosterone µg/100 ml Plasma	Adrenal Corticosterone µg/g	Adrenal Weight Mg/100g B.W.
Control (7)	8.0 ± 1.9	10.29 ± 2.76	5.4 ± 0.4
Vehicle (7)	7.7 ± 1.2	13.16 ± 2.06	4.7 ± 0.2
PCB-treated (7)	11.5 ± 3.4	17.34 ± 3.44	5.1 ± 0.2

*N is the number of animals.

Our finding that exposure to Aroclor 1254 did not alter the 24-hour periodicity in plasma corticosterone is consistent with previous studies which have shown that polybrominated biphenyl (PBB) compounds do not adversely affect plasma corticosterone levels. CASTRACANE et al. (1982) found a normal diurnal rhythmicity in plasma corticosterone 26 days after chronic exposure to PBB and GARTKOFF et al. (1977) reported no change in plasma corticosterone levels after 3 weeks of PBB feeding. In contrast, BYRNE et al. (1980) reported that exposure to both PCBs and PBBs can result in decreased plasma corticosterone levels but only after 3-5 months of treatment.

The possibility that i.p. administration of Aroclor 1254 in the present study was an ineffective means of exposure to PCBs does not seem to be a viable explanation for our data. Although not reported herein, other tissues were evaluated and, importantly, liver tissue from PCB rats showed the inclusions and lesions characteristic of PCB-treated animals (KIMBOUGH et al., 1972). Since both PCB (COLLINS et al., 1977) and PBB (ALLEN-ROWLANDS et al., 1981) compounds have been shown to have a marked effect on thyroid function, it is possible that the long-term effect of thyroid hypofunction would result in lowered plasma corticosterone levels similar to that reported by BYRNE et al. (1980). Whether chronic exposure over an extended period of time would ablate the 24-hr periodicity characteristic of pituitary-adrenal activity is not known. However, the data presented herein clearly show that a single, relatively large dose of PCBs does not abolish adrenocortical rhythmicity.

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