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Effects of Chronic Polychlorinated Biphenyls Exposure on Reproductive Success of White-Footed Mice (*Peromyscus leucopus*)

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Abstract. Reproductive success was impaired in white-footed mice (*Peromyscus leucopus*) chronically exposed to food treated at a rate of 10 ppm (μg/g) polychlorinated biphenyls (PCBs) (Aroclor® 1254). Mice in which PCBs exposure was initiated at adulthood (wild-caught and subsequently paired) and laboratory-raised mice paired and first exposed to PCBs-treated food at 16 weeks of age weaned significantly smaller numbers of young. Other parameters of reproductive success (interval between births, litter size at birth) were unaffected.

Laboratory-raised white-footed mice paired and first exposed to PCBs-treated food at 12 weeks of age exhibited longer intervals between births, smaller litter sizes at birth, and smaller litter sizes at weaning.

These results suggest that exposure to PCBs-contaminated natural foods can contribute to declines in natural populations of white-footed mice by reducing the number of young mice entering the breeding population.

Polychlorinated biphenyls (PCBs) are industrial chemicals that have become ubiquitous environmental contaminants (Risebrough *et al.* 1968; Fleming *et al.* 1983). Exposure to PCBs may affect reproductive success in mammals by a variety of mechanisms, although the underlying cause appears to be that PCBs are powerful inducers of hepatic microsomal enzyme systems. Although enzyme induction has been noted following exposures to dietary dosages as low as 0.5 ppm (μg/g), the degree of response is positively dose-related (Chen and DuBois 1973; Litterst *et al.* 1972; Sanders and Kirkpatrick 1975, 1977; Shull *et al.* 1982). These enzymes have the ability to metabolize steroid hormones, including those needed to regulate reproductive processes (Conney and Burns 1972; Peakall 1970; Sanders and Kirkpatrick 1975, 1977; Welch *et al.* 1971). In females, PCBs exposure (150 ppm, μg/g, in diet) has been shown to reduce plasma concentrations of estradiol and progesterone (Jonsson *et al.* 1976). Disruptions in normal levels of these hormones would be expected to lengthen the estrous cycle (Fishbein 1974), hinder implantation of embryos (Finn and Martin 1972; Orberg and Kihlstrom 1973; Smith 1968), and impair maintenance of the placenta (Choudary and Greenwald 1969). In males, lowered corticosteroid levels following PCBs ingestion (200 and 400 ppm, μg/g) have been correlated with lower than normal sperm counts and reduced accessory gland weights (Sanders and Kirkpatrick 1975; Sanders *et al.* 1977).

There are few data that can be used directly to assess quantitative effects on wild mammals at the population level. Several studies on mink (*Mustela vison*) indicate that reproduction in this species is drastically reduced or eliminated by chronic exposure to 2 ppm (μg/g) or less in the diet (Platonow and Karstad 1973; Aulerich and Ringer 1977). In contrast, chronic exposure of cottontail rabbits (*Sylvilagus floridanus*) to PCBs in the diet (10 ppm, 12 weeks) had no significant effect on breeding performance (Zepp and Kirkpatrick 1976). Studies on the white-footed mouse (*Peromyscus leucopus*) have revealed a variety of reproductive effects, with severity depending upon dosage level and duration of exposure. Sanders and Kirkpatrick (1975, 1977) found that PCBs (Aroclor® 1254) fed at levels up to 100 ppm (μg/g) for short periods (2–3 weeks) had no effect on reproductive organ weights. At

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higher levels (400 ppm), plasma corticoid levels, seminal vesicle weights, and number of testicular spermatozoa were reduced. During a longer term study (60 days) of reproductive effects, Merson and Kirkpatrick (1976) compared breeding success of control pairs with those ingesting food treated with 200 ppm PCBs (Aroclor® 1254). Among these mice, which ranged in age from 44 to 222 days at the time of pairing, fewer PCBs-treated pairs reproduced (84.6% control vs. 29.6% PCBs), but litter size did not differ significantly.

The objective of the present study was to assess the effect of chronic dietary exposure to a low concentration of PCBs (10 ppm, μg/g) on reproductive performance of three different age classes of white-footed mice (*Peromyscus leucopus*). This species was chosen for both practical and theoretical reasons. White-footed mice are easily maintained and breed readily in the laboratory, thus providing an ideal subject for studies attempting to model the effects of toxicants on wild animal populations. Also, white-footed mice are important components of nearly all terrestrial ecosystems, where they provide a major food source for a variety of carnivorous species.

Materials and Methods

White-footed mice (*Peromyscus leucopus*) were either wild caught or were offspring of mice that were wild caught in rural western Pennsylvania. The mice were maintained under a 14L:10D light regime and were allowed *ad libitum* access to food and water. Litters were separated from parents at 28 days of age unless earlier removal was mandated by birth of a subsequent litter.

Rodent blocks (Wayne Rodent Lab-Blox) were hand ground by a heavy duty food grinder and the resulting powder was treated with PCBs at a rate of 10 ppm (10 mg PCBs/kg food). The PCBs formulation was Aroclor® 1254 (Foxboro Co., North Haven, CT). Ethanol (95%) was used to prepare the stock solution of PCBs and also as the carrier to disperse the chemical in the ground food. After thoroughly mixing the food with the ethanol-PCBs solution, the food slurry was dried on a hot plate (approximately 40°C) for two days with occasional stirring. The control diet consisted of ground food mixed with ethanol and similarly dried.

Before the effects of PCBs treatment on reproduction could be evaluated, it was necessary to determine if white-footed mice consumed the treated diet as readily as the control diet. To test palatability, two groups of three females and two males were tested with control food and PCBs-treated food. Food consumption and body weight were monitored every second day for 28 days. Mice eating the control diet consumed an average of 0.142 g/g body weight/day, which was not significantly different from the 0.127 g/g body weight/day consumed by mice eating PCBs-treated food (Mann-Whitney U test, $p = 0.2360$). Although mice initially lost an average of 0.9 g (control) and 0.7 g (PCBs-treated) (probably due to abrupt switch from block to ground food), pre-experimental weights were reattained by the 8th day in the PCBs-treated mice and by the 20th day in the control mice (Table 1). A comparison of weights at 4-day intervals with weights at the beginning of the test (two-way analysis of variance, significance level $p < 0.05$) revealed only one significant difference, *i.e.* control mice at day 4 weighed significantly less ($p = 0.02$).

Table 1. Body weights (g) of white-footed mice fed control and PCBs-treated diets. n = 5 per group.

Day	Control	PCBs-treated
0	20.9	23.1
4	20.0	22.4
8	20.4	23.2
12	19.9	22.8
16	20.4	23.5
20	20.9	23.5
24	21.4	23.7
28	21.1	23.4

Three separate experiments were carried out simultaneously. The first involved white-footed mice that were field caught as adults in December 1984 and maintained in the laboratory for three to five weeks prior to pairing. The exact ages and breeding histories of these mice is unknown. The second and third experiments involved laboratory-raised offspring of field-caught mice that were paired and placed on control or PCBs-treated diets at 12 and 16 weeks of age.

Statistical analysis of reproductive data employed one-way analysis of variance (ANOVA) or Mann-Whitney U (M-W U) tests, depending upon whether or not variates were normally distributed. Effects of treatment were considered significant at $p < 0.05$.

Results

Wild-caught Pairs

At initiation, this experiment involved 10 control pairs and 10 PCBs-treated pairs. White-footed mice from the wild-caught laboratory colony were chosen randomly (alternating males and females), paired, and assigned alternately to control and PCBs-treated groups within a two day period. Data were recorded over 18 months. Data for four females that died before termination of the experiment were retained in the analysis because they lived long enough to have reproduced frequently. For example, one PCBs-treated female produced a single litter within a month following pairing, but did not breed again before her death 11 months later. Similarly, three female control mice that died had been paired for eight to ten months without reproducing. At the close of the study, three control and five PCBs-treated pairs were still reproductively active.

Table 2. Reproductive data for wild-caught *Peromyscus leucopus* fed control and PCBs-treated diets. Mean values ± standard errors are followed by sample sizes in parentheses. Statistically significant differences indicated by asterisk.

	Control	PCBs-treated
% Pairs breeding	60	70
No litters/breeding female	8.8 ± 1.20 (6)	9.9 ± 2.27 (7)
Birth interval (days)	33.3 ± 1.80 (46)	32.3 ± 1.45 (62)
No young/litter at birth	4.4 ± 0.22 (50)	4.0 ± 0.12 (69)
No young/litter at 28 days*	3.6 ± 0.30 (50)	2.6 ± 0.22 (68)

Table 3. Reproductive data for laboratory-raised *Peromyscus leucopus* paired at 12 weeks of age. Mean values ± standard errors are followed by sample sizes in parentheses. Statistically significant differences indicated by asterisk.

	Control	PCBs-treated
% Pairs breeding	53	28
No litters/breeding female	6.5 ± 1.64 (8)	3.8 ± 0.97 (5)
Birth interval (days)*	33.0 ± 1.06 (44)	44.7 ± 6.20 (14)
No young/litter at birth*	4.8 ± 0.15 (52)	3.6 ± 0.23 (19)
No young/litter at 28 days*	4.7 ± 0.18 (50)	2.0 ± 0.38 (18)

Reproductive histories are summarized in Table 2. Six control pairs produced 53 litters during the experiment, while seven PCBs-treated pairs produced 69 litters. The number of litters produced per breeding female ranged from one to sixteen and was more variable among PCBs-treated (1–16) than among control (4–11) pairs, but the mean differences were not statistically significant (ANOVA, $p = 0.36$). Although the interval between births was shorter and the number of young per litter fewer for PCBs-treated pairs, these differences also were not significant (M-W U, $p = 0.11$; ANOVA, $p = 0.08$; respectively). However, significantly fewer young of PCBs-treated pairs survived to 28 days of age (M-W U, $p = 0.002$).

PCBs exposure did not have visible toxic effects on appearance or behavior of treated white-footed mice. However, autopsies of individuals that either died during the experiment or were sacrificed at the end of the study revealed that liver aberrations were common among PCBs treated mice. For example, of nine females examined, two had abnormally pale (yellowish) organs and the liver was markedly enlarged in four others. In nine males, abnormal coloration occurred in two animals and two others exhibited liver enlargement. It is possible that the yellowish color is due to lipid accumulation, as dietary PCBs have been reported to cause increased total liver lipid content (Saito *et al.* 1983). No other organs were visibly affected. In contrast, the livers of 8 female and 9 male control mice were normal in appearance and size.

Laboratory-raised White-footed Mice Paired at 12 Weeks of Age

This experiment involved 15 control and 18 PCBs-treated pairs. Pairing of white-footed mice for this experiment occurred as the laboratory colony reached 12 weeks of age (*i.e.* initiation was not simultaneous). Thus, not all pairs were together for the same amount of time. Precautions to assure comparability included approximately equal assignment of mice from each laboratory "cohort" to treatment type, as well as partitioning litter members of each sex to a different treatment group. Duration of the experiment was a maximum of 15 months and a minimum of slightly more than nine months for the various pairs. One control pair died during the experiment; there were no mortalities among PCBs-treated pairs.

Reproductive data for these pairs are presented in Table 3. The percentage of PCBs-treated pairs breeding was lower than that of control pairs. A total of 52 litters was produced by eight control pairs, while 19 litters were born to five PCBs-treated pairs. Although PCBs-treated pairs produced fewer litters per breeding female than did control pairs, the difference was not significant (M-W U, $p = 0.19$). The interval between births of litters was significantly longer for PCBs-treated pairs and showed much greater variability (25–61 days, controls; 18–104 days, PCBs) (M-W U, $p = 0.03$). Finally, PCBs-treated pairs produced significantly smaller litters (averaging one less individual) (M-W U, $p = 0.0002$) and survival to 28 days of age was significantly reduced (control—98% survival, PCBs—56% survival) (M-W U, $p = 0.0001$).

Although breeding performance of white-footed mice paired at 12 weeks was poorer than that of wild-caught pairs, they exhibited no outward indication of PCBs contamination. However, of 14 PCBs-treated pairs autopsied at the close of the study, 10 females and seven males had yellowish livers (due to lipid accumulation?). Liver enlargement occurred rarely (one PCBs-treated male) and other internal organs appeared normal. Abnormal liver coloration was not observed in any of the autopsied controls (14 pairs).

Table 4. Reproductive data for laboratory-raised *Peromyscus leucopus* paired at 16 weeks of age. Mean values ± standard errors are followed by sample sizes in parentheses. Statistically significant differences indicated by asterisk.

	Control	PCBs-treated
% Pairs breeding	68	40
No litters/breeding female	3.8 ± 0.76 (13)	3.6 ± 1.02 (8)
Birth interval (days)	33.6 ± 2.70 (37)	34.1 ± 5.81 (21)
No young/litter at birth	4.6 ± 0.20 (49)	4.6 ± 0.28 (28)
No young/litter at 28 days*	2.8 ± 0.30 (45)	0.5 ± 0.21 (24)

Laboratory-raised White-footed Mice Paired at 16 Weeks of Age

The method for pairing and strategies for ensuring comparability of data were the same for this experiment as for white-footed mice paired at 12 weeks of age. Nineteen control and 20 PCBs-treated pairs received experimental diets for between seven and one-half and nine months. Two control females and one PCBs-treated female died during the experiment.

Reproductive data for these pairs are presented in Table 4. Eight PCBs-treated females produced 29 litters, while 13 control females produced 50 litters. The number of litters per breeding female did not differ (M-W U, $p = 0.5$). Moreover, the interval between births did not differ significantly (M-W U, $p = 0.07$), although the PCBs-treated pairs were slightly more variable (21–144 days, PCBs; 22–120 days, control). Although the average numbers of young per litter were similar (ANOVA, $p = 0.90$), survival of young from PCBs-treated litters was poor (control—61% survival, PCBs—11% survival) (M-W U, $p = 0.0001$).

PCBs treatment had no visible toxic effects on this group, although abnormal liver coloration was prevalent. In 31 PCBs-treated pairs, five males and six females exhibited pale, yellowish organs (due to lipid accumulation?). Liver enlargement occurred in one PCBs-treated female. No abnormalities were noted in 12 autopsied control pairs. Other internal organs of both groups appeared normal.

Discussion

Experimental results suggest that breeding success of PCBs-treated white-footed mice depends upon the age at which exposure is initiated. In the adult group, more PCBs-treated pairs reproduced than did the controls. However, among mice paired [illegible] and 16 weeks of age, 25% and 28% fewer PCBs-treated pairs reproduced, respectively. Although these differences appear to be substantial, sample size does not allow statistical testing on equality of percentages (Sokal and Rohlf 1969). Lengthening of the interval between births (suggesting a lengthened or interrupted estrous cycle) occurred among 12 week pairs, but not among 16-week pairs or adults. If PCBs affect reproduction by altering levels of steroid hormones, as has been suggested by several researchers (Conney and Burns 1972; Jonsson et al. 1976; Peakall 1970; Sanders and Kirkpatrick 1969; 1977), their presence may disrupt the establishment of regular estrous cycles in younger females and/or more severely affect sperm production in younger males, thus reducing breeding success. However, since the average number of litters produced by a breeding female did not differ in any of the three groups, apparently once breeding cycles are established, they are not easily disrupted.

Since PCBs are transferred to young at much higher rates through milk than through the placenta (Ando 1978; Hori *et al.* 1981), nursing young may be more susceptible to PCBs toxicity than fetal young (assuming that embryos have successfully implanted and that a functional placenta has been established). This suggestion is supported by the present data in that survival of young to 28 days of age was consistently poor in all PCBs-treated groups, but reduction in litter size at birth occurred only in the 12 week pairs. Furthermore, reduction of litter size between birth and 28 days of age was greater in PCBs litters born to younger females (adults—1.4 fewer young at 28 days, 12 weeks—1.6 fewer at 28 days; 16 weeks—4.1 fewer at 28 days). Overall, the final number of young per litter that survived to the age of weaning (= recruitment into a population) was highest for the adult group and lowest for the 16 weeks group.

The possibility that juvenile mortality resulted from parental attacks rather than from direct PCBs toxicity cannot be discounted. Although many young mice had no obvious injuries, others had been bitten in several places and/or were partially eaten. It does not seem likely that PCBs-treated parents were using the young as a secondary food source. Although food consumption by experimental pairs was not measured, body weights of adult mice in the wild-caught experiment were monitored during the initial five months and the data reveal no drastic weight changes among males and non-pregnant females in either group (initial and final weights of 12 mice—PCBs-treated: 21.8 g, 21.9 g; control: 23.1 g, 22.6 g). An alternative

possibility is that PCBs-related behavioral aberrations among parents (attacks or neglect) may have resulted in high juvenile mortality. Neurological effects of PCBs toxicity have been reported (Chen *et al.* 1983, 1985; Seegal *et al.* 1985) and there are data supporting a link between exposure to PCBs and behavior (Bowman *et al.* 1978; Storm *et al.* 1981; Tori and Peterle 1983).

It is difficult to make critical comparisons between the results of this study and previous research on PCBs effects on reproduction in white-footed mice because of differences in duration of the study and ages of individuals at initiation of the study. In general, the results of this study infer that white-footed mice may be susceptible to reproductive effects at a lower dosage level than previously suspected. For example, no effects on male reproductive organ weights and plasma corticoid levels were observed after short-term exposures to less than 400 ppm PCBs in the diet (Sanders and Kirkpatrick 1975). Merson and Kilpatrick (1976) reported that longer term exposure to 200 ppm PCBs reduced the incidence of breeding among females that were between six and 33 weeks at pairing by about 50%.

Previous laboratory studies of PCBs toxicity have been largely confined to laboratory species, especially white mice and rats. However, a comparison of data from the present study with some of those results (Aroclor® 1254, dietary exposure route) indicates that laboratory species are generally less sensitive to effects of PCBs on reproduction and thus are not appropriate models to evaluate effects on wild populations. For example, exposure of male white mice for up to 30 days to food with PCBs added at the rate of 50 to 1,000 ppm (μg/g) had no effect on testes or accessory gland weights, although sperm counts were reduced in comparison with controls (Sanders *et al.* 1974, 1977). Furthermore, Keplinger *et al.* (1971) failed to find reproductive effects (No. breeding, survival of young) at treatment rates of one or 10 ppm. In white rats exposed to 20 ppm over two generations, the first generation was unaffected in regard to the number of females breeding, litter size or survival to weaning (Linder *et al.* 1974).

Although it is difficult to extend the findings of a laboratory study to a field situation, these results indicate a potential negative effect on populations (lowered juvenile recruitment) exposed to PCBs in the diet. Although 10 ppm is a low exposure level compared with other laboratory studies on rodents, it is undoubtedly considerably higher than would be encountered in the natural diets of most white-footed mice. However, it seems possible that observable effects on reproductive success occur at considerably lower (and perhaps common in certain environments) chronic exposure levels.

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