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EFFECT OF DIETARY POLYCHLORINATED BIPHENYLS ON GROWTH AND REPRODUCTION OF MINK

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The Great Lakes have played a prominent role in the development of the mink industry in nearby states as these lakes furnished the rancher with an inexpensive and abundant supply of fish deemed desirable for mink feed. During the middle 1960s, reports from mink ranchers stating that reduced reproduction was a consequence of feeding Great Lakes fish were called to the attention of university researchers (Hartsough, 1965). Coho salmon taken from the tributaries of Lake Michigan during the spawning run in 1967 were made available to commercial ranchers as food for mink and resulted in a precipitous increase in newborn mink (kit) mortality. The extent of mortality appeared as a time-dosage response to coho salmon in the diet. Adult breeders were reported to be unaffected. Rancidity, chlorinated hydrocarbon pesticide contamination and mercury poisoning were each prominent suspects as the causative agent responsible for the high kit mortality. Because of the disastrous effect of feeding coho salmon, mink ranchers discontinued use of Great Lakes fish in mink diets.

In 1969 our laboratory confirmed the mink industry's observation that Lake Michigan coho salmon, when fed to breeder mink, resulted in either total cessation of mink reproduction (embryo toxicity) or kit mortality, the degree of complication being dependent upon the percentage coho salmon fed and the duration of feeding (Aulerich _et al_., 1971). Coho salmon _per se_ were not implicated since west coast coho salmon yielded totally acceptable reproductive parameters. Subsequent reproduction studies yielded data indicating that the causative agent(s) was not confined solely to the salmon from Lake Michigan, but was apparently present, although to a lesser extent, in other fish from the Great Lakes (Aulerich _et al_., 1971).

Rancidity and mercury contamination data for the various fish did not correlate with mink toxicity data and each was discounted as a contributor to the problem; however, hydrocarbon pesticide contamination (total DDT and isomers or dieldrin) of the fish fed was inversely proportional to number of mink offspring born per female. This relationship appears spurious and may reflect, in part, the fat content of the fish. Feeding either DDT and isomers or dieldrin in levels far in excess of that found analytically in the fish failed to simulate the effect on mink of feeding diets containing coho salmon (Aulerich and Ringer, 1970; Aulerich _et al_., 1971).

This study was undertaken to investigate the effects of polychlorinated biphenyls, known to contaminate Lake Michigan and its fish, on growth and reproduction of ranch mink.

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MATERIALS AND METHODS

This study consisted of three feeding trials encompassing polychlorinated biphenyls (PCB) supplementation to mink. PCB supplementation was by incorporating the Aroclor® into a cereal grain premix and then into the diet. The same basal (control) diet, except as specified, was fed in all experiments. The diet composition was cereal, 15%; tripe, 20%; beef liver, 5%; horsemeat, 15%; chicken, 15%; and fish, 30%. Standard ranch procedures of animal care and breeding were used throughout the course of the experiments.

RESULTS

Experiment I

A total of 45 mink were divided into 3 groups (12 females and 3 males per dietary treatment group). The treatments were a basal diet containing ocean fish, substitution of coho salmon for the ocean fish, and the basal diet supplemented with 10 ppm each of Aroclors 1242, 1248, and 1254.

No female mink receiving dietary coho salmon bore young (whelped) nor did those receiving 30 ppm PCB supplementation (Table 1). All adult mink fed supplemental PCB's died by the end of the normal whelping period. Those females, on PCB's, that survived sufficient time to produce offspring showed no signs of whelping. Six mink fed coho salmon died during the experimental period of January 1 to June 30. No adult control mink died.

Table 1

Effect of Dietary PCB Supplementation(1) on Reproduction of Ranch Mink (January 1 to June 30)

	Females mated	Females whelped	Kits born Alive	Kits born Dead	Kits alive (4 wks.)
Basal (control)	12	11	35	19	29
30% coho salmon	12	0	0	0	0
Basal + 30 ppm PCB's	11(2)	0	0	0	0

(1) 10 ppm each of Aroclors 1242, 1248, and 1254.
(2) One female died prior to the mating season.

Tissue analysis for PCB residues from several mink that died while on experimentation are shown in Table 2. Control mink were sacrificed for tissue analysis. The similarity in the tissue residues of the PCB

Aroclor® is the trade name of polychlorinated biphenyls by Monsanto Company, St. Louis, Mo.

treatment and the coho treatment group is striking. Organ weights expressed as a percentage of brain weight from those mink that died and from sacrificed controls were not significantly different ($P > 0.05$).

Table 2

PCB Tissue Residues (in ppm) of Mink that Died While Receiving Dietary Coho Salmon or Supplemental PCB's[1] and from Sacrificed Controls (Mean ± S.E.)

	Basal (control)	30% coho salmon	Basal + 30 ppm PCB's
No. mink	4	3	12
Brain	< 0.01	11.07 ± 0.78	11.00 ± 1.43
Liver	< 0.01	5.21 ± 1.66	4.18 ± 0.58
Kidney	< 0.01	6.37 ± 0.25	4.47 ± 0.42
Spleen	< 0.01	6.19 ± 0.09	4.79 ± 0.39
Lung	< 0.01	5.15 ± 0.22	4.78 ± 0.53
Muscle	< 0.01	4.73 ± 1.80	4.88 ± 0.54
Heart	< 0.01	2.84 ± 1.21	3.26 ± 0.49

(1) 10 ppm each of Aroclors 1242, 1248, and 1254.

Experiment II

Thirty-five mink were divided into five groups of six females and one male per group. Dietary treatments were initiated on August 25 to measure growth rate of young, growing mink and will continue until June 30 when the reproductive phase is complete. Treatments consisted of a basal diet with ocean fish, basal plus 5 ppm Aroclor 1254, basal plus 10 ppm Aroclor 1254, basal plus 10 ppm Aroclor 1254 and 10 ppm DDT, and basal plus 10 ppm Aroclor 1254 and 0.5 ppm dieldrin.

Results of body weight gains for the growth phase from August 25 to December 22 are shown in Table 3. Results for the reproduction phase are included in Table 4.

Table 3

Effect of Aroclor 1254, Alone or in Combination with Pesticides, on Body Weight Gains of Female Mink

Dietary treatment	No. Mink	Initial body wt. (gm) (Aug. 25)	Body weight gain (gm) ± S.E. from initial wt. 1 mo. (Sept. 24)	2 mo. (Oct. 25)	3 mo. (Nov. 25)	4 mo. (Dec. 22)
Basal diet (30% ocean fish mix; control)	6	1020	43 ± 17.4	203 ± 25.4	187 ± 46.2	210 ± 35.1
Basal diet plus 5 ppm PCB(1)	6	940	62 ± 22.4	153 ± 44.2	138 ± 42.7	128 ± 43.7
Basal diet plus 10 ppm PCB	6	925	43 ± 20.4	148 ± 32.0	132 ± 26.3	92 ± 35.1*
Basal diet plus 10 ppm PCB and 10 ppm DDT	6	1052	43 ± 14.5	57 ± 14.6**	28 ± 21.5**	-15 ± 35.4**
Basal diet plus 10 ppm PCB and 0.5 ppm dieldrin	6	940	108 ± 25.1	133 ± 26.7	115 ± 18.9	57 ± 24.4**

(1) Aroclor 1254.
*Significantly different ($P < 0.05$) from controls.
**Significantly different ($P < 0.01$) from controls.

Table 4

Effect of Aroclor 1254, Fed Alone or in Combination with Pesticides, on Mortality and Reproduction of Mink (Aug. 25 to May 10) (6 females and 1 male/group)

	% mortality	Females alive	Females whelped	Kits born Alive	Kits born Dead	Kits/female alive
Control	14	5	3	17	8	5.0
5 ppm PCB(1)	29	4	0	0	0	0
10 ppm PCB	71	1	0	0	0	0
10 ppm PCB + 10 ppm DDT	43	4	0	0	0	0
10 ppm PCB + 0.5 ppm dieldrin	100	0	0	0	0	0

(1) Aroclor 1254.

Data from this experiment demonstrate that 10 ppm Aroclor 1254 depressed growth gains, when fed continuously to growing mink. A dose-response curve for data on 0, 5, and 10 ppm Aroclor 1254 at 4 months treatment yielded a regression equation of $\hat{Y} = 207.5 - 107.4X$ where $X = \ln(\frac{ppm}{5} + 1)$. This regression equation gives a significant dose-response ($P < .05$).

All mink fed PCB supplemented diets failed to produce offspring. Those breeders that died during the gestation period exhibited various degrees of embryo toxicity when the uterus was opened for examination.

Experiment III

One hundred twenty-eight mink were divided into eight groups of twelve females and four males per group to evaluate the effect of Aroclor 1254 on reproduction. The dietary treatments were initiated on January 1 and will continue until June 30. Included in the treatments (Table 5) was a group given 5 ppm Aroclor 1254 plus 6 ppm DDT and 0.2 ppm dieldrin. These levels approximate the residue values found in diets where 30 percent raw Lake Michigan coho salmon constituted the fish portion of the diet. Cooked coho salmon (120° C at 1.05 kg/sq cm for 30 min) was included to test for the heat stability of the factor(s) causing the reproductive problem.

Table 5.

Effect of Aroclor 1254 Fed Singly and in Combination with Hydrocarbon on Reproductive Parameters (Jan. 1 to May 10) (12 females and 4 males/group)

	% mortality	Females alive	Females whelped	Kits born Alive	Kits born Dead	Kits/female alive
Control (ocean fish)	6	11	11	56	10	6.0
Raw coho salmon	19	11	0	0	0	0
Cooked coho salmon	6	12	0	0	0	0
1 ppm PCB(1)	6*	10	8	35	8	4.3
5 ppm PCB	0	12	3	3	6	0.8
15 ppm PCB	31	8	0	0	0	0
5 ppm PCB + 6 ppm DDT	0	12	4	5	6	0.5
5 ppm PCB + 6 ppm DDT + 0.2 ppm dieldrin	19	10	5	15	7	2.2

(1) Aroclor 1254.
* One female escaped.

Data in Table 5 indicate that 5 ppm Aroclor 1254 markedly reduced reproduction when fed only 4 months, 1 ppm Aroclor 1254 caused only slight reduction and 15 ppm PCB totally inhibited reproduction. The factor(s) present in Lake Michigan coho salmon was heat stable and duplication, in part, of several factors known to be present in the salmon did not totally reproduce the coho salmon effect.

The results of these experiments clearly point out the sensitivity of mink to polychlorinated biphenyls and cast suspicion on PCB's as a possible factor involved in the poor reproduction following Lake Michigan coho salmon feeding.

LITERATURE CITED

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