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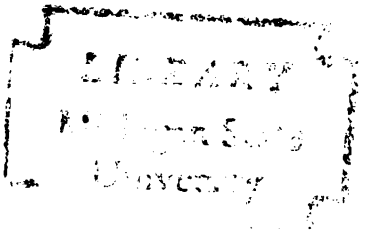

This is to certify that the

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of Great Lakes Fish for Animal Feed

presented by

Thomas C. Hornshaw

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RENEWED USE OF UNDERUTILIZED SPECIES OF GREAT LAKES FISH FOR ANIMAL FEED

By

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ABSTRACT

RENEWED USE OF UNDERUTILIZED SPECIES
OF GREAT LAKES FISH FOR ANIMAL FEED

By

Thomas C. Hornshaw

Great Lakes fish stocks, once an abundant and inexpensive feed ingredient for Mid-western mink ranchers, have been unsuitable for mink feed since the mid-1960's due to toxic contaminants, primarily polychlorinated biphenyls (PCBs). Since PCB residues in Great Lakes fish have declined steadily since the mid-1970's, this study was initiated to determine if they might once again be safely fed to mink. The results indicate that growth and furring were normal for all species tested. However, mink fed carp failed to reproduce and reproductive performance of mink fed perch, whitefish, and sucker was suboptimum. Reproductive performance of mink fed alewife fish meal was equivalent to controls. PCB residues (as Aroclor 1254) accumulated in subcutaneous body fat as much as 38 times, while some congeners accumulated up to 200 times. It was estimated that at least 100 days were required for elimination of 50% of the body fat PCB burden.

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I wish to express my deep appreciation to my wife Elena for her love, understanding, and support throughout my course of study.

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Renewed Use of Underutilized Species of Great Lakes Fish for Animal Feed

For many years Great Lakes fish stocks provided an abundant and inexpensive supply of fish and fish products for the mink ranching industry of the Midwest. Since the late 1960's, however, these fish stocks have proven unsuitable for feeding most animals due to toxic contaminants, forcing the mink industry to rely on more costly marine fish and fish products to meet its feed demands.

Laboratory analyses and mink feeding experiments have shown polychlorinated biphenyls (PCBs) to be the toxic factor present in Great Lakes fish (Aulerich et al., 1971, 1973; Ringer et al., 1972) and in Baltic herring (Jensen et al., 1977). Subsequent studies (Platonow and Karstad, 1973; Aulerich and Ringer, 1977; Bleavins et al., 1980) have shown that mink are extremely sensitive to certain PCBs.

Since PCB use has been greatly curtailed in the U.S., residue levels in most Great Lakes fish species have declined steadily, falling below the 5 ppm limit currently deemed safe for human consumption in most species and below the 2 ppm level for certain species (Michigan Department of Natural Resources, personal communication). Noting this decline, mink ranchers have expressed an interest in once again utilizing some of these species if they can be shown to be safe for animal consumption.

Although monitoring PCB concentrations in fish can provide a good indication of general pollution trends, it is much

more difficult to attempt to predict the degree of toxicity associated with such concentrations due to variations in the chemical and biological properties of the many PCB congeners*. Most animal feeding studies have involved the addition of specific commercial grade PCBs (which contain a specific amount of chlorine by weight and a relatively limited number of congeners) to the diet, which does not take into account the much wider array of congeners normally present in environmentally contaminated fish, the other potentially toxic pollutants often found in conjunction with PCBs, nor the toxicity of the metabolized or selectively retained PCBs in the fish. Some studies have shown marked differences in the toxicity of the various commercial PCBs when fed to mink (Aulerich and Ringer, 1977; Bleavins et al., 1980) and to other species, for example rabbits (Villeneuve et al., 1971a; Koller and Zinkl, 1973), leghorn chickens (Lillie et al., 1974), and various aquatic organisms (Meyer et al., 1972). Research results also indicate that once PCBs have been exposed to biological processes the resultant changes in the ratio of the congeners may result in substantially altered toxicity (Platonow and Karstad, 1973; Biocca et al., 1976; McKinney, 1976). Therefore, feeding trials were deemed necessary to determine the safety of Great Lakes fish and fish products for animal consumption. Experiments were designed to evaluate the growth, furring, and reproductive performance of mink when fed

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* 209 different congeners are possible, containing 1-10 chlorine atoms. Of these, over 100 have been identified in various commercial PCB mixtures (Sissons and Welti, 1971).

various species of Great Lakes fish, in order to assess the suitability of these fish species for use by commercial mink ranchers. The experiments were initiated at the Michigan State University Fur Animal Project on August 15, 1979.

Methods and Materials

During the first year of the study, 96 sub-adult mink were randomly assigned to one of six dietary groups, with the exception that litter-mates were not assigned to the same group. Each diet group consisted of 4 males and 12 females (2 males and 6 females each of the natural dark and pastel color varieties) which were fed *ad libitum* a basal diet (Table 1) differing only in the type of fish or fish product which comprised 30% of the diet. Five fish or fish products were selected for trial, including whole carp (*Cyprinus carpio*), whole white sucker (*Catastomus commersoni*) (both species taken from Saginaw Bay, Lake Huron), yellow perch (*Perca flavescens*) scraps (taken from northern Lake Erie), lake whitefish (*Coregonus clupeaformis*) racks (taken from Big Bay de Noc, Lake Michigan), and alewife (*Alosa pseudoharengus*) fish meal (taken from Green Bay, Lake Michigan). The fish portion of the control diet consisted of ocean fish scrap mix, which included cod, haddock, and flounder trimmings.

Each fish or fish product was assayed for fat content, and since the fat contents varied widely, corn oil was added to all fish or fish products except carp to approximately equalize the energy contents of the various diets (Table 2). In addition, the carp, sucker, and whitefish were cooked by

Table 1. Composition of Basal Diet.

Ingredient	Wgt (kg)	Percentage
Commercial Mink Cereal*	13.3	13.3
Whole Chicken	16.0	16.0
Fish/Fish Products	30.0	30.0
Beef Tripe	5.3	5.3
Beef Liver	2.7	2.7
Beef Lungs	2.7	2.7
Beef Trimmings	2.7	2.7
Cooked Eggs	2.7	2.7
Added Water	24.7	24.7
Total	100.1	100.1

* XK-40 Grower, XK Mink Foods, Thiensville, WI.

Table 2. Fat Content of Fish or Fish Products and Amount of Added Corn Oil.

Diet Group	% Fat	Kg Oil/Fish Portion of Diet	Added Corn Oil (kg)
Carp	21.2	6.4	0
Whitefish	16.6	5.0	1.4
Perch Scraps	8.8	2.6	3.7
Sucker	2.7	0.8	5.5
Alewife Fish Meal	10.6	0.5	5.9
Control	3.8	1.1	5.2

placing them in sealed containers in 95°C water for approximately 2 hours to inactivate thiaminase. (Thiaminise is an antimetabolite of thiamine present in certain fish species which, if not denatured prior to feeding to mink and foxes, can cause a muscular paralysis known as Chastek's paralysis).

During the second year of the study, beginning July 2, 1980, 28 sub-adult (kit) female mink were assigned to either a Great Lakes fish diet group or a control (marine fish) diet group. The assignments were made as follows: 5 pastel and 2 natural dark kits raised by dams fed the perch, sucker, or whitefish diet during the first year of the study (all that remained after mortalities and sacrifices) plus 5 pastel and 2 natural dark kits selected at random from ranch stock were assigned to the Great Lakes fish diet group, while 5 pastel and 2 natural dark (randomly selected) kits raised by dams fed the control or alewife fish meal diet during the first year plus 5 pastel and 2 natural dark kits selected at random from ranch stock were assigned to the control group. The Great Lakes fish diet group was initially fed the perch and sucker (uncooked) diets, as in the first year of the study but without added corn oil, on alternate days. However, after 4 months several mink exhibited signs of Chastek's paralysis, so the sucker diet was fed only every third day after November 6, 1980. The control group received the basal diet, but with the fish portion comprising only 12.5% of the diet instead of 30% as in the first year, and also without added corn oil.

Routine ranch procedures were followed in the feeding, care, and breeding of the animals. All mink were vaccinated as kits against botulism, virus enteritis, and canine distemper. Body weights were recorded at the beginning of each year's trial and monthly thereafter until the breeding season (in March).

During the first year of the study, the females were mated to males within the same diet group, while during the second year the females were mated to males from ranch stock. All matings were verified by the presence of normal, motile spermatozoa in a vaginal smear taken after copulation.

Mated females were checked daily for kits during the whelping period. Kits were counted and weighed on the day of birth and at 4 weeks of age the first year and at 3 weeks the second year (due to a change in ranch record-keeping).

All fish and fish products were analyzed for PCB content (Food and Drug Administration, 1975). In addition, 4 females from each of the first year Great Lakes fish diet groups and 8 females from the control group were randomly selected and anesthetized with Tilazol®*, an incision was made in the inguinal area, and a fat sample (approximately 1 g) was taken for PCB analysis (Food and Drug Administration, 1975). Fat samples were taken in November (at the time of greatest fat deposition) and in February (at the time of least fat deposition). A 500 mg sample of milk was collected (Jones et al.,

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* Parke Davis Co., Ann Arbor, MI.

1981) for PCB analysis from the females previously fat-sampled and lactating in May. These milk samples were extracted by a modification of the method of the Food and Drug Administration (1975), because the milk curdled upon addition of the extracting solvent acetonitrile and had to be ground in a mortar and pestle to break up the curds. Additionally, in an attempt to determine the rate of PCB elimination from the body fat stores of mink, 4 males that had been fed the carp diet (equivalent to 1.5 ppm as Aroclor 1254) since August 15, 1979 were placed on the control ration (equivalent to 0.04 ppm as Aroclor 1254) on July 8, 1980. Fat biopsies from the inguinal area of these males were taken for PCB analysis at the time of the dietary change, and at 2 week intervals thereafter for the next 16 weeks.

Extracts from all samples were injected into a MicroTek Model MT-220 gas chromatograph equipped with a ^{63}Ni high temperature electron capture detector and a 6 foot glass column packed with 3% SE-30 on 60-80 mesh Gas-Chrom Q. The following conditions were maintained for all injections:

Column temperature = 180
Inlet temperature = 210
Detector temperature = 300
Flow rate = 40 ml N_2/minute
Attenuation = 10 x 128

The approximate limit of detection for this analytical system was 0.1 ppm Aroclor 1254. Quantitation was based on Aroclor 1254, using the area of peaks A (retention time = 2.0 minutes), D (retention time = 3.0 minutes), and I (retention time = 6.6 minutes) as shown in Figure 1. Recoveries for the various

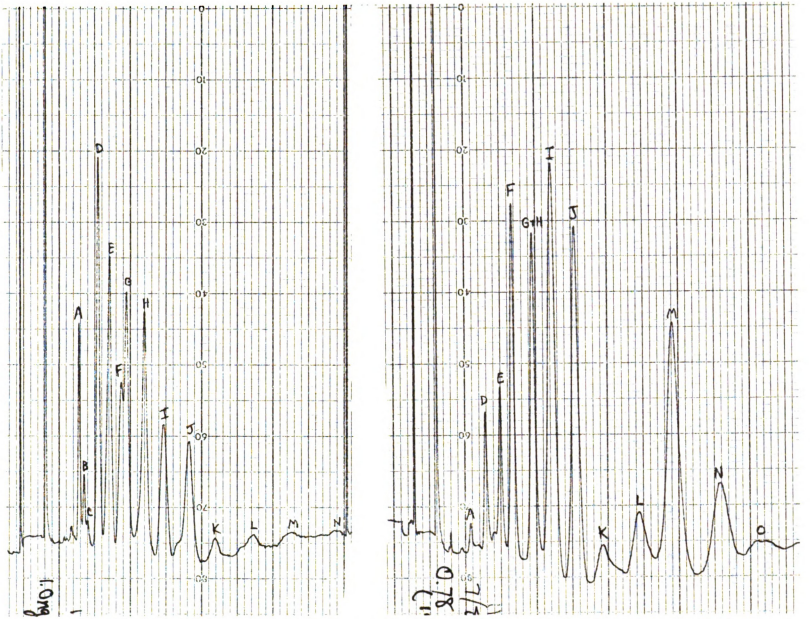

Figure 1. Gas Chromatograph Tracings of Aroclor 1254 Standard (Left) and Mink Fat From a Male Fed 30% Carp (Right).

samples ranged from 72% to 95%, and all reported values are unadjusted. All values are reported as ppm Aroclor 1254. The contributions of peaks A, D, and I to the total calculated PCB residue for the various samples are included for comparison.

Following weaning of the kits in early July, all females previously biopsied plus one of their kits (if any were still surviving) were sacrificed by cervical dislocation. The weights of brain, kidneys, liver, and spleen were recorded.

The data were analyzed by Dunnett's "t"-test for the difference between means of the control and treatment group.

Results

Growth (Table 3) and furring of mink on all diets were normal in both years. Average weight gains by mink fed all Great Lakes fish diets were not significantly different from the control diet. There were, however, mortalities due to Chastek's paralysis in mink fed the carp and sucker diets during the first year (due to insufficiently cooked fish) and in mink fed the alternated perch and sucker diet in the second year. During the first year, 3 females and 1 male (natural dark) were added to the carp and sucker diet groups on January 8, 1980 to replace mortalities. Previous research (Leonard, 1966) had indicated that feeding a diet containing thiaminase with a diet lacking thiaminase on alternate days resulted in no harm to mink, but we found this not to be the case, at least with the sucker diet during the second year feeding trial.

Table 3. Growth of Mink.

Diet Group	Average Weight Gain (g) August 15, 1979-March 15, 1980
(First Year)	
Control	321
Carp	225
Sucker	234
Perch Scraps	360
Whitefish Racks	299
Alewife Fish Meal	265
(Second Year)	July 6, 1980-February 6, 1981
Control	429
Perch & Sucker	440

The PCB residue levels in the Great Lakes fish or fish products and in the total diets for the first and second years, shown in Table 4, range from 1.6-5.0 ppm and 0.2-1.5 ppm, respectively, for the first year and are 2.2 and 0.7 ppm, respectively, for the second year. The PCB content of the total diets for the first year was calculated on the basis of 30% fish in the ration for the control, carp, sucker, whitefish, and perch diets, and on the basis of 6.5% for the alewife fish meal diet (1 kg of fish meal is equivalent to 6.2 kg of whole fish on a dry weight basis). For the second year, the PCB content of the perch and sucker diet was calculated by averaging the PCB contents of the 30% perch and sucker portions, while the PCB content of the control diet was calculated on the basis of 12.5% fish in the diet.

The PCB residue levels in the mink fat samples taken in November, 1979 and February, 1980, shown in Table 5, indicate that PCBs are more concentrated in fat at the end of winter than at the beginning. A bioaccumulation factor (residue level in the fat divided by the residue level in the total diet) has been calculated for these samples, which also shows an increase from November to February.

The reproductive performance of females in the various groups is summarized in Table 6 for both years. No live offspring were produced by females fed the carp diet in the first year, and no kits whelped by females fed the perch and sucker diet in the second year survived to 3 weeks. Table 7 shows

Table 4. PCB Residue Levels in Fish and Diets.

Fish	PCB Residue Level[1] (ppm) In Fish Total	In Fish (Peak A:Peak D:Peak I)	In Diet[2] Total	In Diet[2] (Peak A:Peak D:Peak I
(First Year)				
Control	0.3	(0.09:0.18:0.03)	0.09	(0.03:0.05:0.01)
Carp	5.0	(1.92:2.63:0.48)	1.50	(0.58:0.79:0.14)
Sucker	2.1	(0.27:1.12:0.71)	0.63	(0.08:0.34:0.21)
Perch Scraps	2.3	(0.20:1.02:1.09)	0.69	(0.06:0.31:0.33)
Whitefish Racks	1.6	(0.19:1.25:0.13)	0.48	(0.06:0.38:0.04)
Alewife Fish Meal	3.3	(0.84:1.93:0.52)	0.21	(0.05:0.13:0.03)
(Second Year)				
Control	0.3	(0.09:0.18:0.03)	0.04	(0.01:0.02:0.01)
Perch & Sucker	2.2	(0.24:10.7:0.90)	0.66	(0.07:0.32:0.27)

[1] Based on Aroclor 1254.

[2] Calculated values.

Table 5. PCB Residue Levels in Mink Fat.

Diet Group	n	Mean PCB Residue Level[1] (ppm) Total	Mean PCB Residue Level[1] (ppm) (Peak A:Peak D:Peak I)	Bioaccumulation Factor[2] Total	Bioaccumulation Factor[2] (Peak A:Peak D:Peak I)
(November, 1979)					
Control	7[3]	1.7	(0.13:0.64:0.98)	19.3	(4.8:11.9:108.9)
Carp	4	24.8	(3.53:8.58:12.73)	16.6	(6.1:10.9:88.4)
Sucker	4	13.5	(0.36:1.70:11.46)	21.4	(4.4:5.1:53.8)
Perch Scraps	4	7.6	(0.42:2.71:4.42)	10.9	(7.0:8.9:13.5)
Whitefish Racks	4	6.0	(0.36:3.22:2.47)	12.4	(6.3:8.6:63.3)
Alewife Fish Meal	4	4.0	(0.49:2.04:1.47)	19.1	(9.0:16.3:43.5)
(February, 1980)					
Control	8	2.9	(0.02:1.14:1.76)	32.6	(0.7:21.1:195.6)
Carp	2	42.8	(3.70:9.84:29.32)	28.6	(6.4:12.5:203.6)
Carp	4[4]	36.8	(2.96:9.98:23.88)	24.6	(5.1:12.6:165.8)
Sucker	2	10.8	(1.23:1.70:7.88)	17.2	(15.2:5.1:37.0)
Sucker	4[4]	9.5	(1.20:1.81:6.39)	15.1	(16.2:5.4:30.0)
Perch Scraps	4	13.3	(0.45:3.12:9.77)	19.3	(7.5:10.2:29.9)
Whitefish Racks	4	13.3	(0.50:4.60:8.15)	27.6	(8.8:12.3:209.0)
Alewife Fish Meal	4	8.1	(0.29:2.62:5.15)	38.4	(5.3:20.9:152.4)

[1] Based on Aroclor 1254.

[2] Residue Level in Fat/Residue Level in Total Diet.

[3] 1 Sample Contaminated.

[4] Includes 2 Mink Added in January, 1980 to Replace Mortalities.

Table 6. Reproductive Performance of Female Mink Fed Diets that Contained Various Fish or Fish Products.

Diet Group	No. Females Whelped/ Mated	No. Kits Whelped		Average No. Live Kits/Female		% Kit Survival (Birth to 4 weeks)
		Alive	Dead	Whelped	Mated	
(First Year)						
Control	9/10	38	11	4.2	3.8	55
Carp	3/11	0[1]	9	0[1]	0[1]	0[1]
Sucker	9/10	25	14	2.8	2.5	40
Perch Scraps	9/11	33	12	3.7	3.0	36
Whitefish Racks	8/10	32	7	4.0	3.2	28
Alewife Fish Meal	10/12	53	9	5.3	4.2	51
(Second Year)						(Birth to 3 weeks)
Control	12/14	62	3	5.2	4.4	65
Perch & Sucker	4/8	4[2]	4	1.0[2]	0.5[2]	0[1]

[1] Significantly Less Than Control ($p \leq .001$).

[2] Significantly Less Than Control ($p \leq .01$).

Table 7. Average Body Weights and Biomass of Kits at Birth and at 4 Weeks (First Year) or 3 Weeks (Second Year) of Age.

Diet Group	Birth Avg. Wgt. (g)	Birth Biomass[1] (g)	4 Weeks Avg. Wgt. (g)	4 Weeks Biomass[2] (g)
(First Year)				
Control	8.4	29.7	122.8	368.5
Carp	0.0	0.0	0.0	0.0
Sucker	8.7	24.0	111.1	185.2
Perch Scraps	8.1	29.7	98.3	294.9
Whitefish Racks	8.5	34.1	107.1	321.3
Alewife Fish Meal	8.4	44.5	124.4	419.8
(Second Year)	Birth		3 Weeks	
Control	9.0	46.6	92.4	369.8
Perch & Sucker	7.7	7.7	0.0	0.0

[1] Biomass = Total Weight of all Live Kits/Number of Females That Whelped.

[2] Biomass = Total Weight of all Live Kits/Number of Lactating Females.

the average kit body weights and biomass (total weight of all live kits per female that whelped) at birth and at 4 and 3 weeks for the first and second years, respectively.

The PCB residue levels in the mink milk samples, shown in Table 8, are in the range of 0.4-1.0 ppm for all Great Lakes fish diet groups. Due to the small number of females still lactating on the various diets and the relatively wide range of numbers between groups it was deemed inappropriate to analyze the milk data statistically.

Gross examination of the organs taken at the time of sacrifice revealed no abnormalities, with the exception of some hemorrhages found on the spleens of one of the control females and her kit. Liver weights, expressed as average liver weights, as well as % of brain weight and % of body weight, are shown in Table 9. Livers from mink fed the carp and whitefish diets were significantly larger than the controls, although there were only 2 individuals sacrificed from the whitefish diet. No significant differences were found when the weights of the other organs taken were compared with controls.

The elimination of PCBs from male mink is illustrated in Figure 2 (total PCBs) and Figure 3 (peaks A, D, and I). Over the 16 weeks of observation 60.3% of the total PCB burden in the subcutaneous body fat was eliminated. Of the individual peaks, 87.2% of peak A, 88.9% of peak D, and 55.4% of peak I was eliminated.

Table 8. PCB Residue Levels in Mink Milk.

Diet Group	n	PCB Residue Level[1] (ppm) Total	(Peak A:Peak D:Peak I)	Range (ppm)
Control	5	0.22	(0.05:0.05:0.11)	0.11-0.28
Sucker	3	0.81	(0.10:0.18:0.54)	0.72-0.93
Perch Scraps	2	0.79	(0.12:0.24:0.43)	0.58-1.01
Whitefish Racks	1	0.96	(0.14:0.36:0.46)	---
Alewife Fish Meal	4	0.84	(0.12:0.32:0.40)	0.43-1.07

[1] Based on Aroclor 1254.

Table 9. Liver Weights of Mink Fed Diets that Contained Various Fish or Fish Products.

Diet Group	n	Average Liver Wgt. (g)	% Brain Wgt.	% Body Wgt.
Control	7	21.54	281.3	2.73
Carp	4	30.54[1]	395.3[2]	4.01[2]
Sucker	4	25.99	334.3	3.11
Perch Scraps	4	25.76	308.7	3.17
Whitefish Racks	2	37.09[1]	486.8[1]	4.54[2]
Alewife Fish Meal	4	24.27	322.9	2.70

[1] Significantly Greater Than Control ($p \leq .01$).

[2] Significantly Greater Than Control ($p \leq .05$).

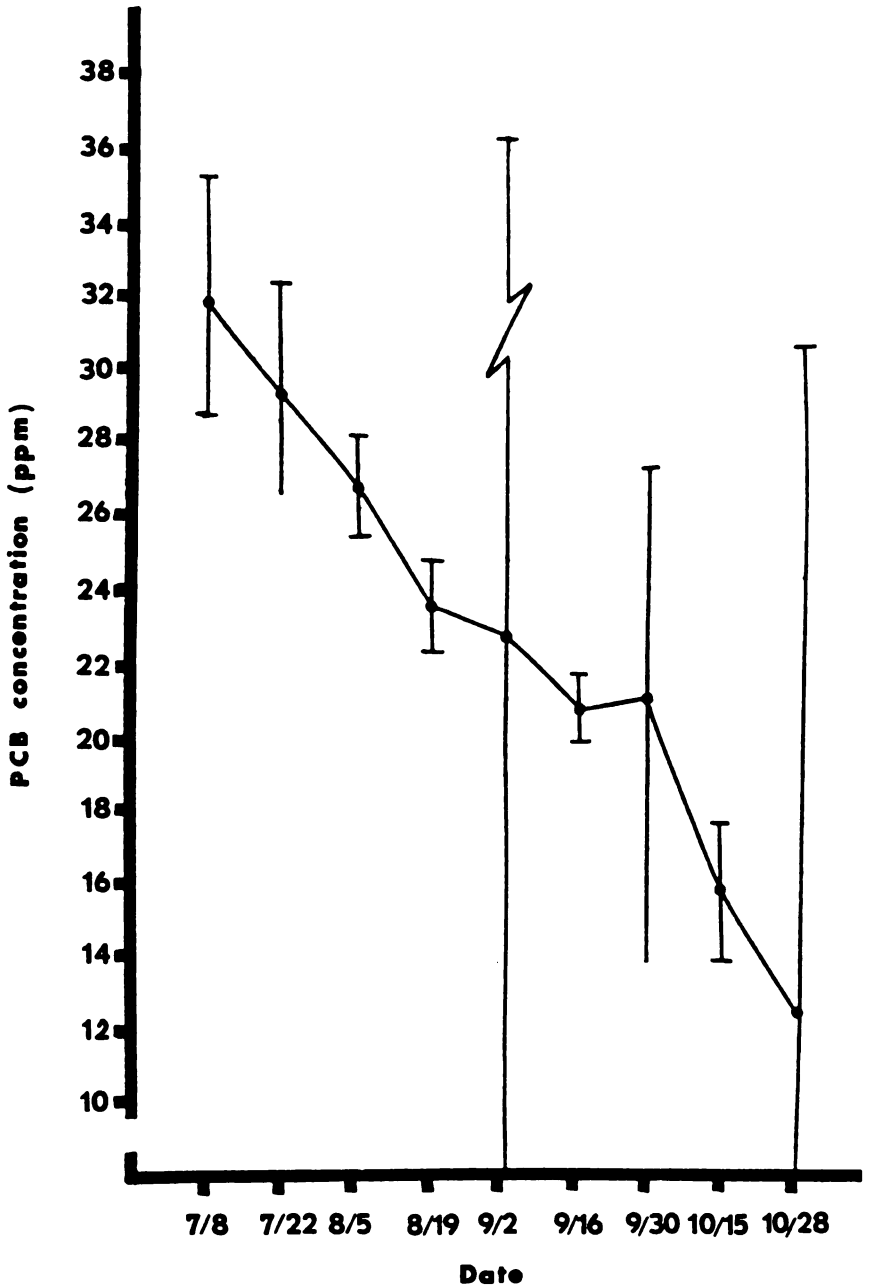

Figure 2. Elimination of Total PCBs From Mink Fat Over a 16 Week Withdrawal Period.

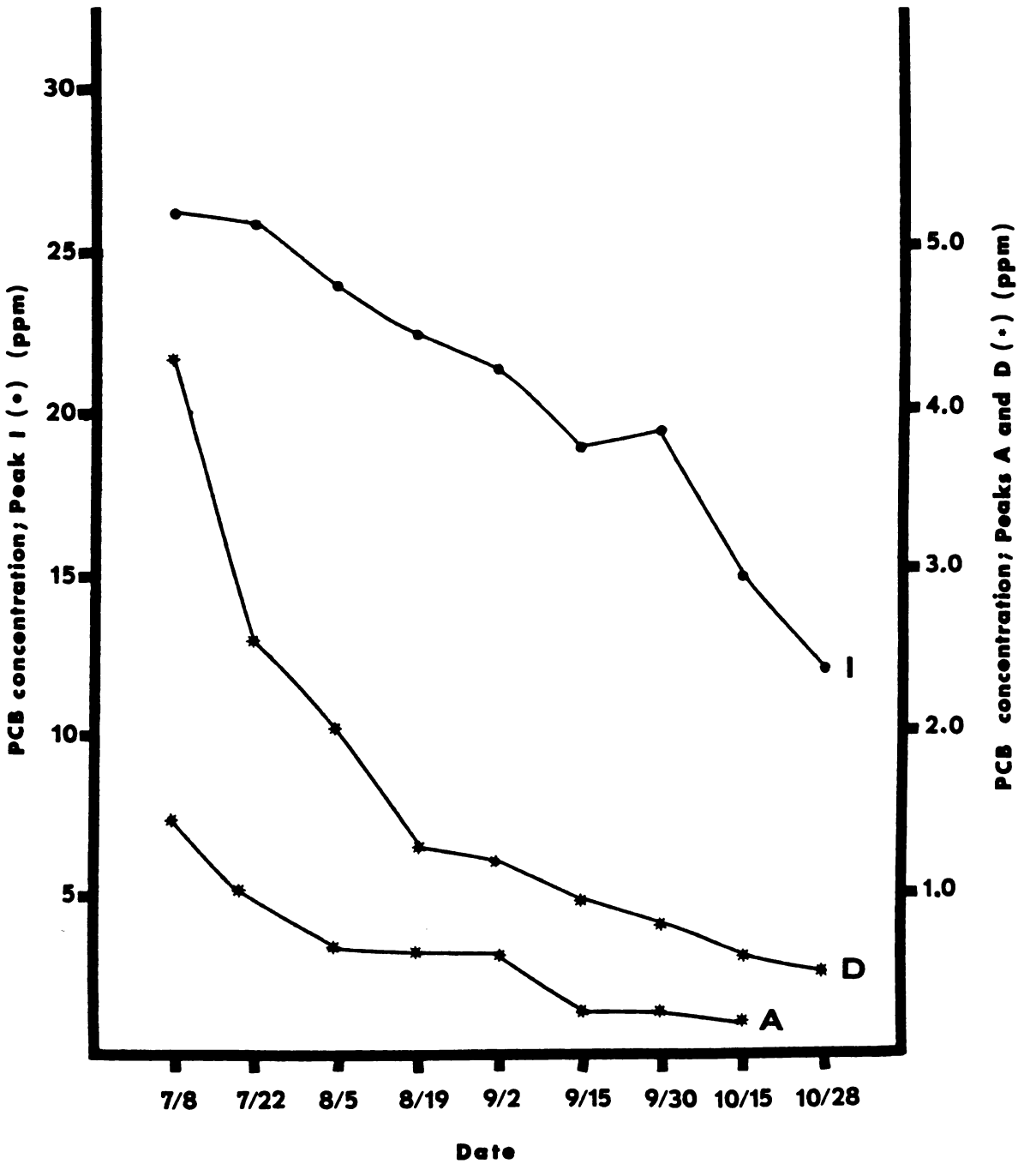

Figure 3. Elimination of Peaks A, D, and I From Mink Fat Over a 16 Week Withdrawal Period.

Discussion

The primary objective of this study was to assess the suitability of Great Lakes fish and fish products for mink feed. With this in mind, an attempt was made, where possible, to simulate the "worst possible" set of circumstances a commercial mink ranch might encounter using Great Lakes fish or fish products. Thus, the relatively high level of 30% fish or fish products was used in the diets, since some commercial mink ranches routinely utilize this level of fish in their diets. Also, whenever possible the fish or fish products were procured from relatively polluted environments such as Saginaw Bay, Green Bay, and the western basin of Lake Erie. Finally, in the second year of the study as many offspring of first year dams as possible were used in the Great Lakes fish diet group to provide maximum exposure to the PCBs derived from Great Lakes fish or fish products.

Before discussing the results of the study, the reader should be aware that compounds other than PCBs, most notably chlorinated dibenzofurans (CDFs) and chlorinated dibenzodioxins (CDDs), have been found to have many of the same effects on laboratory animals as PCBs, and usually at much lower levels (Vos, 1972, Gupta et al., 1973; Moore et al., 1976). These compounds have been identified as contaminants of some commercial PCB mixtures (Vos et al., 1970; Porter and Burke, 1971; Bowes et al., 1975), and, in addition, may be present in certain environments, especially Saginaw Bay, as a result of industrial activities (unpublished research,

Pesticide Research Center, Michigan State University). Thus, it is possible that effects attributed to PCBs in this study may be due to the presence of CDFs and/or CDDs in the fish or fish products used. However, since the effects of these compounds on mink have not been studied, and since these compounds were not analyzed in the fish or fish products, it will be assumed that the effects noted are due to PCBs.

The reader should also be aware that problems exist with the handling of environmental PCB samples. Various methods are recommended for the extraction, clean-up, chromatography, and confirmation of environmental PCB samples, as well as for methods of quantitation*. In this study the 3 peaks used for quantitation were chosen for the lack of known interfering compounds at the respective retention times of these peaks and for the relative prominence of these peaks in the fish, fat, and milk samples. A list of some of the chlorobiphenyls known to elute at or near the retention times of peaks A, D, and I is shown in Table 10.

PCBs in Fish, Mink Fat, and Mink Milk

PCBs are readily stored in mink adipose tissue from dietary exposure. Previous research (Iwamoto, 1973) has shown that PCB residues reach levels approximately 25 times those in the diet, then plateau after 8 weeks. Examination of the data in Table 5 shows that the total PCB residue levels in

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* See Duinker et al., 1980 for a discussion of some of the problems involved with quantitation of environmental PCB samples.

Table 10. Chlorobiphenyls Eluting at or Near Retention Times of Peaks A, D, and I.

Peak	Major Component	Minor Components
A	2,5,2',5' Tetra-CB[1]	2,4,2',5' Tetra-CB[1]
D	2,3,6,2',5' Penta-CB[1]	2,5,3',4' Tetra-CB[1,2] 2,4,3',4' Tetra-CB[1]
I	2,4,5,2',4',5' Hexa-CB[1]	2,3,4,3',4' Penta-CB[2] 2,3,4,2',3',6' Hexa-CB[2]

[1] From Webb and McCall, 1972.

[2] From Sissons and Welti, 1971.

mink fat in February were approximately double the levels in November for all diet groups except sucker (which unexplainable dropped from the November level), and in the range of 17-38 times the dietary level, indicating that, as fat stores are mobilized during cold weather, PCB residues are concentrated in the fat depots. This is consistent with results obtained with pigeons which, after reaching plateau levels of Aroclor 1254 in adipose tissue, approximately doubled the concentraton of PCB in adipose tissue after exposure to 1 week of cold temperature and starvation (DeFreitas and Norstrom, 1974).

Of particular interest are the bioaccumulation factors for peaks A, D, and I. Comparison of these values for November and February shows that peaks A and D accumulated to approximately the same levels at both sampling times, while peak I accumulated in February to approximately twice the November level (again, the sucker diet group is the exception). This indicates that there is net movement out of the fat depot for some (or all) of the chlorobiphenyls comprising peaks A and D, since the bioaccumulation factors would be expected to rise in response to a shrinking of the fat depot. This net movement could be due to relocation of the chlorobiphenyls to other tissues, metabolism and elimination of the chlorobiphenyls, or, perhaps, a combination of both processes. Similar results have been found in robins (Sodergren and Ulfstrand, 1972), which relocate PCBs (primarily to brain and breast muscle), and in pigeons (DeFreitas and Norstrom, 1974), which relocate the extensively metabolize PCBs.

The bioaccumulation factors for peak I also illustrate the relatively high concentrations that can be attained by the more highly chlorinated PCB congeners in mink fat. In fact, the gas chromatographic patterns of the adipose tissue extracts more closely resembled the pattern of Aroclor 1260 than Aroclor 1254. This PCB mixture was not, however, used for quantitation, since the few peaks in the fish extracts with retention times that corresponded to major peaks in the Aroclor 1260 mixture were too small for reliable quantitation. Nevertheless, these "1260" peaks were very prominent in all adipose tissue samples (Figure 4).

The total PCB residue levels in adipose tissue are consistent with published data (Odsjo, 1973) which show levels of 45 ppm Aroclor 1254 in adipose tissue of mink whose diets included Baltic Sea fish. PCB bioaccumulation factors for several other animal species are listed in Table 11 for comparison.

No trends were evident when the concentration of PCBs in the milk samples were compared with either the dietary or adipose tissue PCB levels, although the small numbers of milk samples make generalizations based on these values unreliable. The most notable observation from these values was the relatively high PCB concentration in the milk samples taken from females in the alewife fish meal diet group. These values were equivalent to those of the other diet groups (in fact, the highest individual PCB concentration in milk was found in

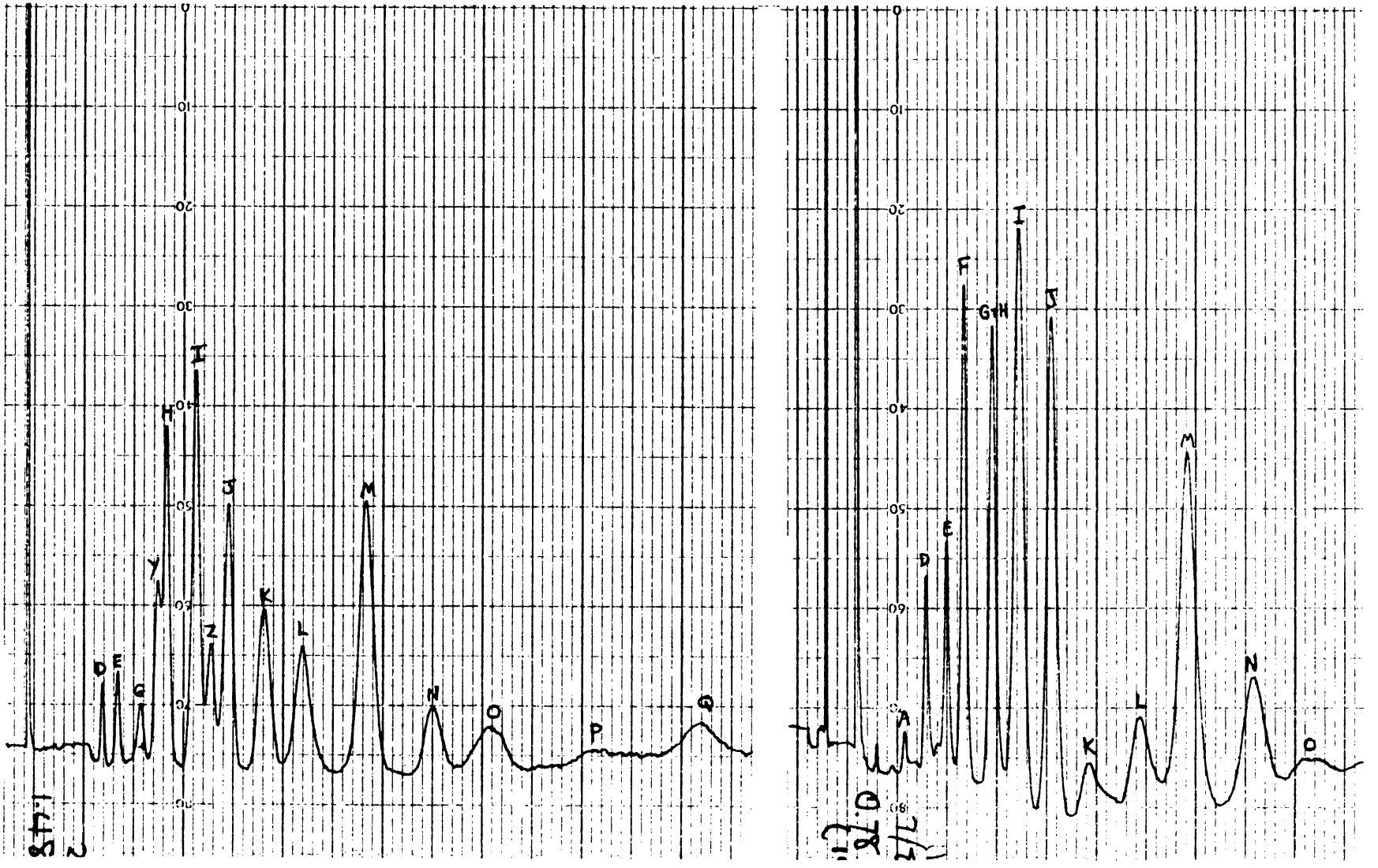

Figure 4. Gas Chromatograph Tracings of Aroclor 1260 Standard (Left) and Mink Fat From a Male Fed 30% Carp (Right).

Table 11. PCB Bioaccumulation Factors for Various Species.

Species	Bioaccumulation Factor[1] (Aroclor Exposure)	Time Until Plateau (Weeks)	Source
Monkey (Rhesus)	5* (1248)	8	Allen, 1975
Rat (Male, Sprague-Dawley)	10* (1254)	39	Allen et al., 1976
	30* (1262)	39	
Swine	0.5* (1242)	13	Hansen, 1979
	1.9* (1254)	13	
Sheep	0.8* (1242)	16	Hansen, 1979
	1.5* (1254)	16	
Chicken (NS)[2]	2.8* (1242)	8	Hansen, 1979
	3.0* (1254)	8	
Chicken (Male Broiler)	1.0-2.5* (1942)	3	Hansen et al., 1976
	4.4-6.9* (1254)	3	
Chicken (Layer)	11* (1254)	NS[2]	Teske et al., 1974
Channel Catfish	5.9* (1242)	36	Hansen, 1979
Fathead Minnow	1.2×10^5 # (1248)	NS[2]	Defoe et al., 1978
	2.7×10^5 # (1260)	NS[2]	
Crayfish (Orconectes sp.)	0.8×10^3 # (1254)	3	Meyer et al., 1972
Amphipod (Gammarus sp.)	6.3×10^3 # (1254)	2	Meyer et al., 1972
Daphnia Magna	3.8×10^3 # (1254)	0.5	Meyer et al., 1972
Glass Shrimp (Paleomontes sp.)	2.5×10^3 # (1254)	3	Meyer et al., 1972
Mosquito (Culex sp.)	3.5×10^3 # (1254)	1	Meyer et al., 1972
Midge (Chaoborus sp.)	2.7×10^3 # (1254)	2	Meyer et al., 1972

[1] * = Residue Level in Fat/Concentration in Total Diet.
= Residue Level in Whole Body/Concentration in Water.

[2] NS = Not Specified.

this group), even though the dietary and adipose tissue PCB concentrations of the alewife fish meal diet group much more closely approximated those of the control group. Also notable is the fact that the concentraton of PCBs in the mink milk was comparable to the concentration of PCBs in the diet, whereas in cows the milk concentration was approximately 4 times the dietary level (Fries et al., 1973).

Reproduction

The detrimental effects of PCBs on mink reproduction are well documented. Aulerich and Ringer (1977) have shown that diets supplemented with as little as 2 ppm of Aroclor 1254 result in near total reproductive failure. Platonow and Karstad (1973) have shown that "metabolized" PCBs are even more detrimental to mink reproduction, since beef products from cows which had been dosed with Aroclor 1254 and fed to mink at levels as low as 0.64 ppm (as Aroclor 1254) had severe effects on mink reproduction. The list of animal species found to suffer reproductive problems upon exposure to PCBS is extensive; see, for example, the reviews of Stendell (1976) and Kimbrough (1978).

The results of this study further illustrate the deleterious effects of PCBs on mink reproduction, especially PCBs which have been exposed to biological processes before being fed to mink. During the first year of the study, females fed the carp diet, with the equivalent of 1.5 ppm Aroclor 1254,

produced no offspring which survived to 24 hours, while those fed the perch, sucker, and whitefish diets, with the equivalent of 0.69, 0.63, and 0.48 ppm Aroclor 1254, respectively, showed reduced reproduction, as measured by average litter size and kit survival to 4 weeks (Table 6). However, the kit survival of all groups, including the control group, was below that which would be deemed acceptable on a commercial mink ranch. (Perhaps the addition of the corn oil to the first year diets had a negative effect on kit survival. Thus, the elimination of added corn oil from the second year diets). During the second year of the study, females fed the alternated perch and sucker diets, with the equivalent of 0.66 ppm Aroclor 1254, showed significantly reduced reproduction and 100% mortality at 3 weeks. The second year results are nearly identical to the results of Platonow and Karstad (1973), including dietary PCB level, average litter size, and kit survival.

Assuming that adult female mink consume an average of 150 g of feed per day (Schaible, 1970; Bleavins and Aulerich, 1981), the total amount of PCBs consumed by the females to the beginning of whelping can be calculated. These values, based on 250 days exposure in the first year and 290 days in the second, are shown in Table 12. Comparison of these results with the results of Aulerich and Ringer (1977) further illustrate the difference between adding technical PCB mixtures to a mink diet and adding environmentally derived PCBs to a mink diet. At a level of 2 ppm Aroclor 1254 in the

Table 12. Total Dietary PCB Intake of Female Mink Fed Diets that Contained Various Fish or Fish Products[1].

Diet Group	Intake (mg)
(First Year)	
Control	3.4
Carp	56.2
Sucker	23.6
Perch Scraps	25.9
Whitefish Racks	18.0
Alewife Fish Meal	7.9
(Second Year)	
Control	1.7
Perch & Sucker	28.7

[1] Based on an Assumed Feed Consumption of 150 g/Day.

diet for 9 months (the lowest level which Aulerich and Ringer found to have a significant effect on reproduction) the females consumed approximately 61 mg of PCB. In the second year of this study, similar results were obtained from females which consumed approximately 29 mg of PCB, while in the Platonow and Karstad (1973) study (0.64 ppm PCB for 160 days) similar results were obtained after consumption of about 15 mg of PCB.

Enhanced toxicity of biologically modified PCBs can be explained by an increased toxicity of metabolized forms of PCBs, by selective retention of the more toxic (usually the more highly chlorinated) congeners of a PCB mixture within an animal, by interactions of PCBs with other compounds present within an animal, or by combinations of these factors. Whatever the causative factor(s) may be, it would appear that biologically modified PCBs are more toxic to mink than corresponding technical mixtures.

In contrast to the poor results obtained with whole fish and fish by-products, the results from the first year of this study (and those of a commercial mink ranch) with alewife fish meal are encouraging. In this study reproduction and survival of kits were comparable to that of controls (although, as noted above, kit survival was sub-optimum for both groups). Furthermore, a large commercial mink ranch in Illinois which used the same alewife fish meal fed in this study in some of their diets reports satisfactory mink growth and reproduction on these diets (personal communication,

Northwood Fur Farms, Inc.). The results of this study also suggest the possibility of using other underutilized Great Lakes fish stocks for making fish meal.

Comparison of the results of the first and second years of this study (Tables 6 and 7) reveal an interesting possibility. Since the Great Lakes fish diets for both years differ only in the amount of oil in the diets, yet the reproduction, biomass, and kit survival are dissimilar, it is possible that the addition of corn oil to the first year diets may have lessened the effects of the PCBs on the mink. Similarly, Hansen et al. (1976) have reported a small positive effect on PCB elimination in a high fat diet fed to broiler cockerels.

Liver Weights

Increased liver weight and/or fatty degeneration of the liver has been associated with PCB exposure in practically all species studied. Rats (Grant et al., 1971; Kimbrough et al., 1972; Allen and Abrahamson, 1973; Cecil et al., 1973; Grant and Phillips, 1974; Allen et al., 1976), mice (Orberg and Lundberg, 1974), rabbits (Villeneuve et al., 1971b; Koller and Zinkl, 1973), swine (Hansen et al., 1975), monkeys (Allen et al., 1974), pigeons (Bailey and Bunyan, 1972), quail (Bailey and Bunyan, 1972; Cecil et al., 1973), pheasants (Dahlgren et al., 1972), and chickens (Iturri, 1974) have all shown liver enlargement due to exposure to various PCB mixtures. Mink are no exception, showing increased liver

weight when fed Aroclor 1254 at 5 ppm for 9 months (Aulerich and Ringer, 1977). In this study, female mink fed the carp and whitefish diets for 9 months also exhibited significantly enlarged livers, at levels equivalent to 1.5 and 0.5 ppm Aroclor 1254, respectively (Table 9). Livers of females fed the other Great Lakes fish diets were also slightly enlarged, but not significantly different from the controls. These results again point to increased toxicity of biologically modified PCBs to mink.

Elimination

Elimination of PCBs from fat stores in mink has been studied (Iwamoto, 1973), although the rate of elimination was based on relatively few samples. In an effort to provide more information on the rate of PCB elimination from mink, 4 males that had been on the carp diet for 11 months were fat biopsied biweekly and the samples analyzed for PCB concentration. The results (Figure 2) indicate that first order decay occurred over the length of the study (16 weeks; the study was terminated after the 16th week). Regression analysis of the data gave an estimate of 199 days for elimination of PCBs from adipose tissue and 98.4 days for 50% removal ($r = -0.985$). These results are underestimates, however, since the study was terminated before second order elimination could occur. Nevertheless, the estimate of 98 days for 50% elimination may provide a gauge by which mink ranchers can judge whether mink which have been accidentally or unintentionally exposed to PCBs should be pelted or kept for breeding.

The results (Figure 3) also indicate that rates of elimination for individual PCB congeners can vary widely. Peak A exhibited second order decay throughout the study, peak D exhibited first order decay for the first week after removal of the carp diet followed by second order decay to termination, and peak I exhibited first order decay throughout the study. (Since peak I dominated the composition of the total PCB residue, the rate of elimination for total PCBs was first order also).

The relatively fast elimination of peak I (primarily 2,4,5,2',4',5'-hexachlorobiphenyl) indicates the high level of activity of mink mixed-function oxidase enzyme systems. This PCB congener, which has an apparent half-life of infinity in the rat (Hutzinger et al., 1972; Matthews and Anderson, 1975), pigeon and brook trout (Hutzinger et al., 1972), was undoubtedly among the congeners eliminated during the course of the elimination of peak I. The rate of elimination of PCBs from various species is shown in Table 13 for comparison.

Other Observations

Certain observations in this study suggest that natural dark and pastel mink are not completely equivalent metabolically. First, 9 of 10 deaths during the study due to Chastek's paralysis occurred to natural dark mink. Re-examination of the monthly weight records for December and January of the first year of the study, when the first year deaths occurred, further illustrates the different susceptibility of natural

Table 13. Rate of Elimination of PCBs from Adipose Tissue of Various Species.

Species	Half-Life (Days)	PCB Tested	Source
Swine	283.5	Aroclor 1254	Hansen, 1979
	>350	245-HCB[1]	Hansen, 1979
Rat		245-HCB[1]	Matthews and Anderson, 1975
		245-HCB[1]	Hutzinger et al., 1972
Brook Trout		245-HCB[1]	Hutzinger et al., 1972
Pigeon		245-HCB[1]	Hutzinger et al., 1972
	125	Aroclor 1242	Bailey and Bunyan, 1972
Chicken (Layer)	20.5	Aroclor 1254	Teske et al., 1974
	85.4	Aroclor 1254	Hansen, 1979
	30.8	245-HCB[1]	Hansen, 1979

[1] 2,4,5,2',4',5'-Hexachlorobiphenyl.

dark and pastel mink to thiaminase. The records for the carp, sucker, and whitefish diet groups, which are summarized in Table 14, indicate that weight loss, which results from reduced food intake due to the reduced mobility associated with muscular paralysis, is greater for natural dark mink than for pastels in all groups except the December 14 whitefish group. The PCB gas chromatographic patterns of natural dark and pastel mink show several differences between the two color varieties. Figure 5 shows representative gas chromatographic tracings of 2 male mink fat samples, one of the natural dark and one of the pastel variety, taken at the initiation of the elimination study. If the tracings are superimposed so that peaks L, M, N, and O coincide, it will be apparent that peaks A, E, F, G & H, J, and K differ in their relative heights. Note especially the difference between peaks A, E, and G & H in the 2 tracings, as well as the difference between peak K in relation to peak L. These differences were most pronounced in the elimination study (so pronounced, in fact, that it was possible to identify the color variety of a sample without it being labelled, by inspecting the gas chromatographic trace), but they were also evident in several of the February biopsy samples.

Conclusions

Based on the results of this study, the following conclusions were made:

Table 14. Body Weight Changes of Mink Fed the Carp, Sucker, and Whitefish Diets, December 14, 1979 to January 15, 1980.

Diet Group	Color Variety	Average Wgt. Change (g) December 14, 1979 January 15, 1980	
Carp	Dark	- 96	-137
	Pastel	- 29	- 30
Sucker	Dark	-113	-210
	Pastel	- 48	- 74
Whitefish Racks	Dark	- 51	- 48
	Pastel	- 64	+ 1

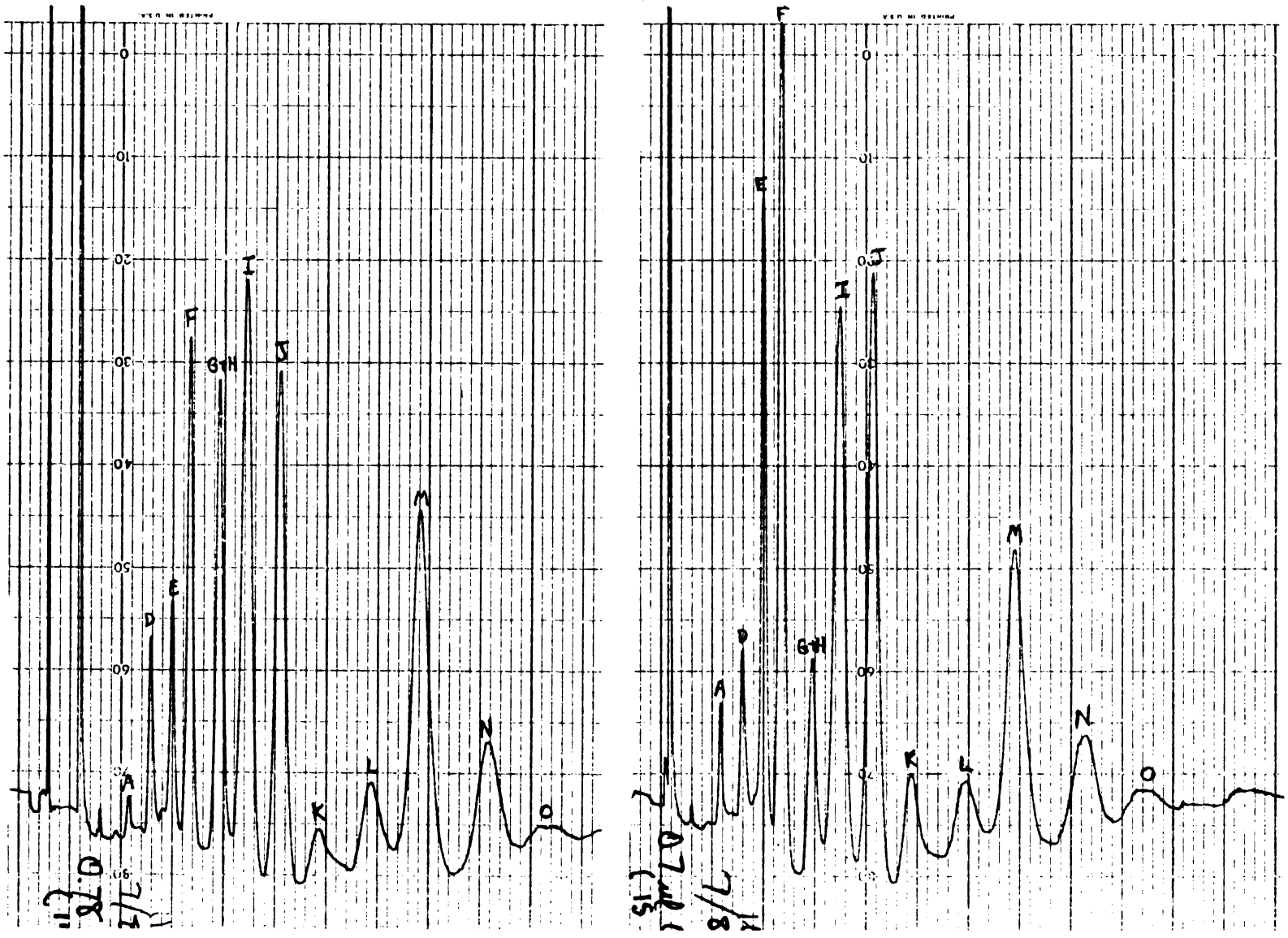

Figure 5. Gas Chromatograph Tracings of Natural Dark (Left) and Pastel Mink (Right) Fed 30% Carp.

(1) Whole Great Lakes fish or fish products are currently unsafe for use as mink feed when fed at a level of 30% of the diet.

(2) Alewife fish meal may be safe for use in mink diets at levels up to the equivalent of 30% whole fish. Further study would be helpful in determining its safety.

(3) Mink can accumulate PCBs as much as 38 times the dietary level, and can accumulate individual congeners as much as 200 times the dietary level.

(4) It is estimated that at least 100 days are required for mink to eliminate 50% of the adipose tissue burden of PCBs.

(5) Environmentally derived PCBs appear to be more toxic to mink than corresponding technical mixtures.

REFERENCES

Allen, J.R., 1975. Response of the non-human primate to polychlorinated biphenyl exposure. Fed. Proc. 34:1675.

Allen, J.R. and L.J. Abrahamson, 1973. Morphological and biochemical changes in the liver of rats fed polychlorinated biphenyls. Arch. Environ. Contam. Toxicol. 1:265.

Allen, J.R., L.A. Carstens, and L.J. Abrahamson, 1976. Responses of rats exposed to polychlorinated biphenyls for fifty-two weeks. I. Comparison of tissue levels of PCB and biological changes. Arch. Environ. Contam. Toxicol. 4:404.

Allen, J.R., D.H. Norback, and I.C. Hsu, 1974. Tissue modifications in monkeys as related to absorption, distribution, and excretion of polychlorinated biphenyls. Arch. Environ. Contam. Toxicol. 2:86.

Aulerich, R.J. and R.K. Ringer, 1977. Current status of PCB toxicity to mink, and effect on their reproduction. Arch. Environ. Contam. Toxicol. 6:279.

Aulerich, R.J., R.K. Ringer, and S. Iwamoto, 1973. Reproductive failure and mortality in mink fed on Great Lakes fish. J. Reprod. Fert. Suppl. 19:365.

Aulerich, R.J., R.K. Ringer, H.L. Seagran, and W.G. Youatt, 1971. Effect of feeding coho salmon and other Great Lakes fish on mink reproduction. Can. J. Zool. 49:611.

Bailey, S. and P.J. Bunyan, 1972. Interpretation of persistence and effects of polychlorinated biphenyls in birds. Nature 236:34.

Biocca, M., J.A. Moore, B.N. Gupta, and J.D. McKinney, 1976. Toxicology of selected symmetrical hexachlorobiphenyl isomers. I. Biological responses in chicks and mice. Proc. Natl. Conf. on Polychlorinated Biphenyls. Nov. 19-21, 1975, Chicago, IL pp. 67-72.

Bleavins, M.R. and R.J. Aulerich, 1981. Feed consumption and food passage time in mink (Mustela vison) and European ferrets (Mustela putorius furo). Lab. Animal Sci. 31:268.

Bleavins, M.R., R.J. Aulerich, and R.K. Ringer, 1980. Polychlorinated biphenyls (Aroclors 1016 and 1242): Effects on survival and reproduction in mink and ferrets. Arch. Environ. Contam. Toxicol. 9:627.

Bowes, G., M. Mulvihill, B.R.T. Simoneit, A.L. Burlingame, and R.W. Risebrough, 1975. Identification of chlorinated dibenzofurans in American polychlorinated biphenyls. Nature 256:305.

Cecil, H.C., S.J. Harris, J. Bitman, and G.F. Fries, 1973. Polychlorinated biphenyl-induced decrease in liver vitamin A in Japanese quail and rats. Bull. Environ. Contam. Toxicol. 9:179.

Dahlgren, R.B., R.J. Bury, R.L. Linder, and R.F. Reidinger, Jr., 1972. Residue levels and histopathology in pheasants given polychlorinated biphenyls. J. Wildlife Mangt. 36:524.

DeFoe, D.L., G.D. Veith, and R.W. Carlson, 1980. Effects of Aroclor 1248 and 1260 on the fathead minnow (Pimephales promelas). J. Fish. Res. Board Can. 35:997.

DeFreitas, A.S. and R.J. Norstrom, 1974. Turnover and metabolism of PCBs in relation to their chemical structure and the movement of lipids in the pigeon. Can. J. Physiol. 52:1080.

Duinker, J.D., M.T.J. Hillebrand, K.H. Palmork, and S. Wilhelmsen, 1980. An evaluation of existing methods for quantitation of polychlorinated biphenyls in environmental samples and suggestions for an improved method based on measurement of individual components. Bull. Environ. Contam. Toxicol. 25:956.

Environmental Protection Agency, 1976. Proceedings of National Conference on Polychlorinated Biphenyls, November 19-21, 1975, Chicago, IL. Report No. EPA-560/6-75-004. Washington, D.C.

Food and Drug Administration, 1975. FDA Pesticide Analytical Manual, Vol. I. 1968 (rev. 1975).

Fries, G.F., G.S. Marrow, and C.H. Gordon, 1973. Long-term studies of residues retention and excretion by cows fed a polychlorinated biphenyl (Aroclor 1254). J. Agric. Food Chem. 21:117.

Grant, D.L. and W.E.J. Phillips, 1974. The effect of age and sex on the toxicity of Aroclor 1254, a polychlorinated biphenyl, in the rat. Bull. Environ. Contam. Toxicol. 12:145.

Grant, D.L., W.E.J. Phillips, and D.C. Villeneuve, 1971. Metabolism of a polychlorinated biphenyl (Aroclor 1254) mixture in the rat. Bull. Environ. Contam. Toxicol. 6:102.

Gupta, B.N., J.G. Vos, J.A. Moore, J.G. Zinkl, and B.C. Bullock, 1973. Pathological effects of 2,3,7,8,-tetrachloro-p-dioxin in laboratory animals. Environ. Health Perspect. 5:125.

Hansen, L.G., 1979. Selective accumulation and depletion of polychlorinated biphenyl components: Food animal implications. Ann. N.Y. Acad. Sci. 320:238.

Hansen, L.G., C.S. Byerly, R.L. Metcalf, and R.F. Bevill, 1975. Effect of polychlorinated biphenyl mixture on swine reproduction and tissue residues. Am. J. Vet. Res. 36:23.

Hansen, L.G., D.W. Wilson, R.L. Metcalf, and M.E. Welborn, 1976. Residues of polychlorinated biphenyl components in broiler cockerels receiving two Aroclors in three dietary variations. J. Agric. Food Chem. 24:256.

Hutzinger, O., D.M. Nash, S. Safe, A.S.W. DeFreitas, R.J. Norstrom, D.J. Wildish, and V. Zitko, 1972. Polychlorinated biphenyls: metabolic behavior of pure isomers in pigeons, rats, and brook trout. Sci. 178:312.

Iturri, S.J., 1974. The effects of various chlorinated hydrocarbons on the cardiovascular physiology and hematology of the domestic fowl. Ph.D. Thesis, Department of Poultry Science, Michigan State University, East Lansing, MI 144 pp.

Iwamoto, S., 1973. The effects of polychlorinated biphenyls and coho salmon on mink. M.S. Thesis, Department of Poultry Science, Michigan State University, East Lansing, MI 73 pp.

Jensen, S., J.E. Kihlstrom, M. Olsson, C. Lundberg, and J. Orberg, 1977. Effects of PCB and DDT on mink (Mustela vison) during the reproductive season. Ambio 6:239.

Jones, R.E., R.K. Ringer, and R.J. Aulerich, 1980. A simple method for milking mink. Fur Rancher 60:4.

Kimbrough, R., J. Buckley, L. Fishbein, G. Flamm, L. Kasza, W. Marcus, S. Shibko, and R. Teske, 1978. Animal toxicology. Environ. Health Perspect. 24:173.

Kimbrough, R.D., R.E. Linder, and T.B. Gaines, 1972. Morphological changes in livers of rats fed polychlorinated biphenyls. Light microscopy and ultrastructure. Arch. Environ. Health 25:354.

Koller, L.D. and J.G. Zinkl, 1973. Pathology of polychlorinated biphenyls in rabbits. Am. J. Pathol. 70:363.

Leonard, A., 1966. Modern Mink Management. Ralston Purina Co., St. Louis, MO 206 pp.

Lillie, R.J., H.C. Cecil, J. Bitman, and G.F. Fries, 1974. Differences in response of caged White Leghorn layers to various polychlorinated biphenyls (PCBs) in the diet. Poultry Sci. 53:726.

Matthews, H.G. and M.W. Anderson, 1975. Effect of chlorination on the distribution and excretion of polychlorinated biphenyls. Drug Metab. Disp. 3:371.

McKinney, J.D., 1976. Toxicology of selected symmetrical hexachlorobiphenyl isomers: II. Correlating biological effects with chemical structure. Proc. Natl. Conf. on Polychlorinated Biphenyls, Nov. 19-21, 1975, Chicago, IL pp. 73-76.

Meyer, F.L., P.M. Mehrle, and H.O. Sanders, 1972. Residue dynamics and biological effects of polychlorinated biphenyls in aquatic organisms. Proc. 164th Natl. Meeting Amer. Chem. Soc., New York, N.Y., 1972.

Moore, J.A., B.N. Gupta, and J.G. Vos, 1976. Toxicity of 2,3,7,8-tetrachlorodibenzofuran - preliminary results. Proc. Natl. Conf. on Polychlorinated Biphenyls, Nov. 19-21, 1975, Chicago, IL pp. 77-80.

Odsjo, R., 1973. PCB in some Swedish terrestrial organisms. In PCB Conference II Nat. Swedish Environ. Protection Board Publications: 4E. pp. 45-58.

Orberg, J. and C. Lundberg, 1974. Some effects of DDT and PCB on the hormonal system in the male mouse. Environ. Physiol. Biochem. 4:116.

Platonow, N.S. and L.H. Karstad, 1973. Dietary effects of polychlorinated biphenyls on mink. Canad. J. Comp. Med. 37:391.

Porter, M.S. and J.A. Burke, 1971. Separation of three chlorodibenzo-p-dioxins from some polychlorinated biphenyls by chromatography on an aluminum oxide column. J. Assoc. Offic. Anal. Chem. 54:1426.

Ringer, R.K., R.J. Aulerich, and M. Zabik, 1972. Effect of dietary polychlorinated biphenyls on growth and reproduction in mink. 164th Natl. Meeting, Amer. Chem. Soc. 12:149.

Schaible, P.J., 1970. Nutrition and Feeding. Sect. III. In: The Blue Book of Fur Farming. Milwaukee, WI, Editorial Service Co.

Sissons, D. and D. Welti, 1971. Structural identification of polychlorinated biphenyls in commercial mixtures by gas-liquid chromatography, nuclear magnetic resonance, and mass spectrometry. J. Chromatogr. 60:15.

Sodergren, A. and P. Ulfstrand, 1972. DDT and PCB relocate when caged robins use fat reserves. Ambio 1:36.

Stendell, R.C., 1976. Summary of recent information regarding effects of PCBs on birds and mammals. Proc. Natl. Conf. on Polychlorinated Biphenyls, Nov. 19-21, Chicago, IL pp. 262-267.

Teske, R.H., B.H. Armbrecht, R.J. Condon, and H.J. Paulin, 1974. Residues of polychlorinated biphenyl in products from poultry fed Aroclor 1254. J. Agric. Food Chem. 22:900.

Villeneuve, D.C., D.L. Grant, K.Khera, D.J. Clegg, H. Baer, and W.E.J. Phillips, 1971a. The fetotoxicity of a polychlorinated biphenyl mixture (Aroclor 1254) in the rabbit and in the rat. Environ. Physiol. 1:67.

Villeneuve, D.C., D.L. Grant, W.E.J. Phillips, M.L. Clark, and D.J. Clegg, 1971b. Effects of PCB administration on microsomal enzyme activity in pregnant rabbits. Bull. Environ. Contam. Toxicol. 6:120.

Vos, J.G., 1972. Toxicology of PCBs for mammals and for birds. Environ. Health Perspect. 1:105.

Vos, J.G., J.H. Koeman, H.L. Van Der Maas, M.C. Ten Noever de Brauw, and R.H. DeVos, 1970. Identification and toxicological evaluation of chlorinated dibenzofuran and chlorinated naphthalene in two commercial polychlorinated biphenyls. Food Cosmet. Toxicol. 8:625.

Webb, R.G. and A.C. McCall, 1972. Identities of polychlorinated biphenyl isomers in Aroclors. J. Assoc. Offic. Anal. Chem. 55:746.