Bull. Environ. Contam. Toxicol. (1987) 39:939–944

Bulletin of Environmental Contamination and Toxicology

Organochlorine and PCB Residues in Lake Erie Mink Populations

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PCB poisoning has been found in mink (Mustela vison) fed on Great Lakes fish (Aulerich et al. 1973) but is poorly known for wild mink populations (O'Shea et al. 1981). The objective of this study was to determine whether mink from the Lake Erie basin were accummulating levels of PCB and organochlorine residues high enough to cause health effects.

MATERIALS AND METHODS

The skinned carcasses of 55 wild mink caught in November and December 1978 and 1979, and from January to March 1979, were obtained from trappers through the Ontario Ministry of Natural Resources (Fig. 1). Carcasses were wrapped in aluminium foil and stored at -40°C. Weights of the skinned carcasses were corrected by assuming the skin to be 17% of the body weight (Sherburne and Dimond 1969). The head, lower legs, tail and stomach contents were discarded and carcasses were homogenized in a Hobart chopper for 5 to 20 minutes. Homogenates were analyzed by the Ontario Research Foundation for DDT, DDD, DDE, dieldrin, heptachlor epoxide, HCH, chlorobenzenes, chlordane, mirex, and PCB. Procedures are described in Reynolds and Cooper (1975) and Norstrom et al. (1980). PCBs were quantified by capillary gas chromatography against a 1:1 standard mixture of Aroclor 1254:1260. Geometric means were calculated for all residue data as a measure of central tendency because of the logarithmic distribution of the data. The logarithmic means of the residues were compared to each other in analyses of variances and t-tests (Dixon and Massey 1969).

RESULTS AND DISCUSSION

Levels of PCB 1254/1260 detected in mink body homogenates varied from 0.06 to 7.37 ppm, on a wet weight basis, and from 0.8 to

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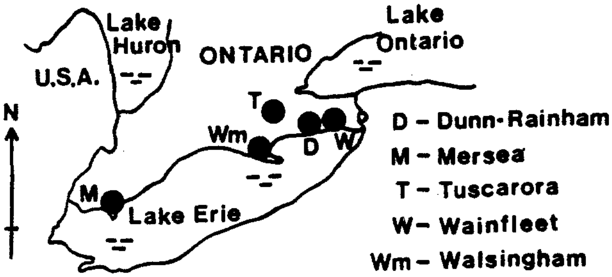

Figure 1. Location of study areas on the north shore of Lake Erie.

117.7 ppm, on a lipid weight basis (Tables 1 and 2). Analyses of variance showed a significant difference between the logarithmic means of the mink samples on a wet weight basis ($F_{4, 50}$ = 17.467, p <0.005) and on a lipid weight basis ($F_{4, 48}$ = 13.743, p <0.005). Both sets of data indicated that mink samples from Mersea and Dunn-Rainham Twps. had very high mean PCB levels, similar (p >0.05) to each other but significantly (p <0.005) greater than those of other regions (Tables 1 and 2).

Mink body homogenates had DDE residue levels ranging from 0.02 to 0.91 ppm, on a wet weight basis, and from 0.26 to 33.7 ppm, on a lipid weight basis (Tables 1 and 2). Analyses of variance showed a significant difference between the logarithmic means of the mink samples on a wet weight basis ($F_{4, 50}$ = 8.217, p <0.005) and on a lipid weight basis ($F_{4, 48}$, 7.375, p <0.005). The highest mean DDE levels were calculated from Walsingham and Mersea Twps. samples (Tables 1 and 2). The Tuscarora Twp. sample mean DDE level was not different (p >0.05) from other means because of the large variance (values ranging from 0.02 to 0.66 ppm on a wet weight basis). The lower PCB/DDE ratio (1.11:1) was calculated for the Tuscarora Twp. sample, and the highest one (12.56:1), for the Dunn-Reinham Twp. sample (Table 2).

HCB residues were detected in all samples, but at low (<0.01 ppm) levels (Table 1). Oxychlordane residues were detected in most samples at levels below 0.1 ppm. Levels of dieldrin and heptachlor epoxide were detected in most samples at uniformly low levels. Residues of mirex were not detectable. Beta-HCH concentrations were low (Table 1). The detection of uniform levels of these trace organochlorines in all samples indicates the ubiquitous spread of these contaminants in the environment.

Platonow and Karstad (1973) found a significant reduction in the number of surviving kits per female that had average concentrations of 0.23 to 0.39 ppm PCB in brain, kidney, liver, muscle and heart tissues. With average concentrations of 0.87

Table 1. Organochlorine and PCB residues (ppm wet weight) in mink body homogenates from 5 regions of Lake Erie.

	Mersea Twp.	Dunn-Rainham Twp.	Walsingham Twp.	Wainfleet Twp.	Tuscarora Twp.
PCBs	1.32 (17)[a]	1.71 (9)	0.51 (11)	0.29 (13)	0.08 (5)
	0.23-4.59[b]	0.45-7.37	0.16-1.39	0.11-1.54	0.06-0.15
	0.85-2.07[c]	0.78-3.73	0.33-0.78	0.17-0.49	0.05-0.13
DDE	0.22 (17)	0.14 (9)	0.26 (11)	0.05 (13)	0.07 (5)
	0.08-0.62	0.06-0.60	0.04-0.91	0.02-0.13	0.02-0.66
	0.11-0.46	0.08-0.24	0.13-0.53	0.04-0.08	0.01-0.44
HCB	<0.01 (16)	<0.01 (7)	<0.01 (11)	<0.01 (13)	<0.01 (5)
	-	-	-	-	-
Oxychlordane	0.02 (14)	0.02 (6)	0.01 (7)	0.01 (5)	0.01 (1)
	0.01-0.107	0.01-0.04	-	0.01-0.02	-
B-HCH	0.01 (5)	0.03 (6)	0.02 (5)	0.01 (4)	-
	0.01-0.03	0.01-0.05	0.01-0.02	-	-
pp'-DDT	0.02 (1)	-	0.02 (1)	-	-
	-	-	-	-	-
pp'-DDD	0.03 (9)	0.01 (2)	0.02 (1)	0.01 (3)	0.02 (1)
	0.01-0.08	-	-	-	-
Dieldrin	0.03 (7)	0.02 (7)	0.02 (9)	0.01 (7)	0.02 (2)
	0.01-0.10	0.01-0.03	0.01-0.04	-	0.01-0.03
Mirex	ND	ND	ND	ND	ND

[a]Geometric mean (number of carcasses with detectable levels)
[b]Range of individual values
[c]Confidence interval estimates of the logarithmic mean (calculated for PCBs and DDE)
ND: not detectable

Table 2. PCB and DDE residues (ppm lipid weight) in mink body homogenates from 5 regions of Lake Erie.

	Mersea Twp.	Dunn-Rainham Twp.	Walsingham Twp.	Wainfleet Twp.	Tuscarora Twp.
PCB	29.17 (15)[a]	26.12 (9)	10.74 (11)	6.08 (13)	1.81 (5)
	4.1-102.5[b]	7.8- 80.1	2.3-35.9	1.4-23.0	0.8 -2.9
	17.22-49.42[c]	13.36-51.05	6.0-19.22	3.82-9.67	0.65-5.06
DDE	4.83 (15)	2.08 (9)	5.48 (11)	1.16 (13)	1.62 (5)
	1.67- 13.08	1.29-6.52	0.80-33.7	0.26-4.64	0.27-12.7
	3.39- 6.88	1.35-3.23	2.59-11.62	0.73-1.85	0.29-8.89
Ratio PCB/DDE	6.00	12.56	1.96	5.24	1.11

[a]Geometric mean (number of carcasses with detectable levels)
[b]Range of individual values
[c]Confidence interval limits of the logarithmic mean

to 1.33 ppm PCB in liver tissues and 0.62 to 0.97 ppm in muscle tissues, only 1 out of 12 females produced a litter, all kits died during their first day of life, and two other females died during the study (Platonow and Karstad 1973). Hornshaw *et al.* (1983) found that females with PCB concentrations in their adipose tissues ranging from approximately 10 ppm to 43 ppm showed significantly reduced reproduction and 100% kit mortality at 3 weeks.

The mean PCB level detected in Mersea and Dunn-Rainham mink body homogenates were markedly higher than the average concentrations detected in organs of mink fed experimental diets (Platonow and Karstad 1973). The analysis of the Lake Erie mink samples was not conducted on adipose tissues. However, because PCB residues accumulate mostly in mink subcutaneous fat (Hornshaw et al. 1983), and assuming an equal distribution of PCB residues in the adipose tissues of all organs, PCB levels calculated on a lipid weight basis for Lake Erie body homogenates are believed to be indicative of the levels of accumulation of PCB residues in adipose tissues. Mean PCB levels similar or higher than those reported by Hornshaw et al. (1983) for adipose tissues, were recorded in mink samples from Walsingham (10.7 ppm), Dunn-Rainham (26.1 ppm) and Mersea (29.2 ppm) Twps. Knowing that metabolized PCBs are more toxic to mink than corresponding technical mixtures (Platonow and Karstad 1973, Hornshaw et al. 1983), one can conclude that these PCB levels were probably great enough to cause reduced reproductive success and PCB poisoning (Ringer et al. 1972, Platonow and Karstad 1973) in these Lake Erie wild mink populations.

Mink are less sensitive to DDT, DDD and DDE than PCBs (Aulerich and Ringer 1970, Jensen et al. 1977) and laboratory studies showed no effect on reproduction among mink fed 100 ppm p,p'DDT plus 50 ppm p,p'-DDD (Aulerich and Ringer 1970) or 100 ppm p,p'-DDT (Duby 1970). The mean DDE levels reported in this present study were markedly lower than Frank et al.'s (1979) average of 0.54 ppm DDE for Ontario mink muscle tissues and similar to those found by Sherburne and Dimond (1969) in mink from an unsprayed forest area in Maine. It is unlikely that the levels recorded in this study are toxic to mink (Aulerich et al. 1972).

It is known that HCB has adverse effects on the survival of kits, even at levels of exposure as low as 1 ppm (Bleavins et al. 1984). However, the low levels (<0.01 ppm) recorded in our study would probably have little effect on the development or survival of kits. The significance for mink of other organochlorines detected in this study has not been determined experimentally (O'Shea et al. 1981), but it is doubtful that individually they would have any significant effect on the mink populations because of their small concentrations. However, future study on the synergistic effect on mink reproduction of all these residues is necessary to understand the full impact of insecticide residues on wild mink (Franson et al. 1974).

The high PCB levels found in some of these mink samples, and the presence of organochlorine residues, may become significant in winter. PCB's, DDE and other organochlorine contaminants accumulate primarily in the fatty tissue (Aulerich and Ringer 1970, Albros and Fishbein 1972, Hornshaw et al. 1983). Because of a low food availability in winter, fat reserves would be mobilized, thereby releasing accumulated organochlorines and polychlorinated biphenyls that could be toxic.

Organochlorine residues in mink will be largely a function of uptake through diet and elimination by metabolic processes. Also, using DDE as a reference compound (Norstrom et al. 1978), this study showed that the PCB accumulation in mink carcasses varied considerably from one region to another. This implies that these mink either had different diets from one region to another, or had similar diets with different levels of organochlorine contamination. This initial investigation invites further studies on wild mink populations in order to better assess the effect of PCBs and organochlorines on reproduction, to define the possible origins of residues through mink scat analyses, and to better manage mink populations and habitats.

Acknowledgments. We thank all trappers who collected carcasses and Mr. Peter Courtney who ground up the carcasses. We also thank Mr. John Struger, Canadian Wildlife Service, and Dr. James D. Somers, Alberta Environmental Centre, for review of the original manuscript.

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Received August 11, 1986; accepted September 12, 1986.