

7-29-2009

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Haynes, James M.; Pagano, James J.; and Wellman, Sara Tucker, "Total PCBs, Dioxin-Furan TEQs and Total Mercury Concentrations in Mink In and Out of the Rochester Embayment Area of Concern Near and Inland from the Shore of Lake Ontario" (2009).

Environmental Science and Ecology Faculty Publications. 106.

https://digitalcommons.brockport.edu/env_facpub/106

Citation/Publisher Attribution:

Haynes, J.M., Pagano, J.J. & Wellman, S.T. Total PCBs, Dioxin-Furan TEQs and Total Mercury Concentrations in Mink In and Out of the Rochester Embayment Area of Concern Near and Inland from the Shore of Lake Ontario. *Arch Environ Contam Toxicol* (2009) 57: 794.

<https://doi.org/10.1007/s00244-009-9365-0>

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**Total PCBs, Dioxin-Furan TEQs and Total Mercury Concentrations in Mink
In and Out of the Rochester Embayment Area of Concern
Near and Inland from the Shore of Lake Ontario**

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Published article available at:

<http://www.springerlink.com/openurl.asp?genre=article&id=doi:10.1007/s00244-009-9365-0>

Abstract

In terms of reproductive and other adverse outcomes after exposure to polychlorinated biphenyls (PCBs), dioxins and furans, the mink (*Mustela vison*) is one of the most sensitive mammals. Our objective was to determine if there are differences in the concentrations of total mercury (Hg), total PCBs, and dioxin-furan toxic equivalents (TEQs) between mink living in and out of the Rochester Embayment of Lake Ontario (RELO) Area of Concern (AOC) and between mink living near the shore of Lake Ontario and inland. Concentrations of total Hg in brain, total PCB and dioxin-furan TEQ in adipose, and total PCB in liver were significantly higher for mink living near the shore of Lake Ontario than inland. For mink living in and out of the AOC, differences in total PCB and dioxin-furan TEQ in adipose and liver were substantial but not significant. Correlations between concentrations of total Hg, total PCB, and dioxin-furan TEQ in mink were high. Our results suggest that contamination of mink living near the southern shore of Lake Ontario primarily comes from contact with the Lake Ontario food web, not from sources in the RELO AOC.

Introduction

In the 1980s the binational (Canada, U. S.) International Joint Commission (IJC) began the process of creating and implementing remedial action plans (RAP) in 43 contaminated areas of concern (AOC) throughout the Great Lakes Basin. The IJC established 14 “use impairments” that could cause a local area to be “listed” as an AOC, including “degradation of fish and wildlife populations” and “bird or animal deformities or reproductive problems.” In the Rochester Embayment of Lake Ontario (RELO) AOC, both uses were listed as impaired because “very few” mink (*Mustela vison*), one of the most sensitive mammals to PCBs, dioxins and furans (Basu et al. 2007), were then being trapped or observed within 3 km of the Lake Ontario shoreline (RAP 1993, 1997).

The RELO AOC includes the Rochester Embayment, a 94 km² portion of Lake Ontario south of a line between Bogus Point in the town of Parma and Nine Mile Point in the town of Webster (both in Monroe County, New York), adjacent wetlands and bays, and the 10 km reach of the Genesee River from the Lower Falls to Lake Ontario (Figure 1).

We report on concentrations of total PCB, dioxin-furan TEQ, and total mercury in lakeshore and inland populations of mink in and out of the AOC. While total PCB and dioxin-furan TEQ have limitations as estimators of toxicity to wildlife (Giesy and Kannan 2008, Blankenship et al. 2008), we wanted to develop a simple biomonitoring tool to answer the question of whether mink in the RELO AOC are potentially suffering from the “bird or animal deformities or reproductive problems” or “degradation of fish and wildlife populations” use impairments, and whether these impairments can be “delisted” by the IJC. For detailed examination of potential toxicity to mink, congeners of PCBs, dioxins and furans also were analyzed (Pagano and Haynes, unpublished data).

Methods

Specimen Collection, Processing and Handling

Collection. Forty mink carcasses were collected by trappers (90% during climatic winter in western New York, November through March) during 2004 and 2005 in five areas which we divided into four regions (Figure 1): Inland/AOC, Lakeshore/AOC, Inland/Out of AOC, and Lakeshore/Out of AOC. Lakeshore/AOC was the Braddock Bay Wildlife Management Area (BBWMA—a large wetland complex), and Lakeshore/Out of AOC was along streams in uplands and small wetlands beginning 15 km west of the BBWMA. Inland/AOC included animals taken in the AOC watershed more than 5 km from the lakeshore (primarily from near the Bergen Swamp, 34 km south of the BBWMA), and Inland/Out of AOC included animals taken ~180 km east of the BBWMA in the Tug Hill Plateau beyond the eastern end of Lake Ontario and from the Iroquois National Wildlife Refuge 50 km southwest of the BBWMA (we used five mink from each of these two inland regions). Skinned carcasses were put in labeled plastic bags and frozen by the trappers who also completed log sheets indicating the date and location of capture for each animal. Chemical concentrations were determined for ten mink from each region.

Processing. We thawed the frozen mink carcasses before recording tail-less and pelt-less body weight and length and the weight of each tissue sample. We placed carcasses in

hexane-rinsed aluminum containers for resection, and all utensils used were rinsed with hexane between uses. Tissues collected for analyses were adipose and liver (total PCB, dioxin-furan TEQ) and brain (total mercury). Adipose was scarce (<6 g) on most (29/40) carcasses, so it was collected wherever it was found. Teeth were collected and aged but aging proved unreliable, similar to the results of Tansy et al. (2003).

Analytical Procedures

Dioxins, Furans and Mercury. Liver and adipose were shipped frozen on dry ice to Columbia Analytical Services (CAS), Inc.'s laboratory in Houston, TX for dioxin/furan analyses (Method 8290A, USEPA 1998), and to the Environmental Research Center (ERC) at SUNY Oswego for PCB analysis. Brains for total mercury analysis (Method 7471A, USEPA 1994) were shipped to CAS's laboratory in Kelso, WA. Methyl mercury accounts for >90% of total mercury in mink brain (Evans et al. 2000). Depending on the sample and congener, method detection limits (MDL) for dioxins/furans and mercury were 1.0-5.0 ng/kg and 0.001-0.019 mg/kg, respectively. Toxic equivalency factors (TEF) used to calculate toxic equivalents (TEQ) came from USEPA (1989).

Total PCB Sample Extraction and Clean-up. All tissue samples were extracted for gas chromatographic analysis after methods developed at The College at Oswego ERC (Pagano et al. 1999, Stewart et al. 2008). Each sample was extracted three times with 50 mL hexane using a Brinkman Polytron homogenizer. Lipid analysis was conducted by gravimetric procedures utilizing a subsample of the extracted sample. Sample cleanup utilized automated Gel Permeation Chromatography (USEPA 1997, Method 3640A) followed by a silica gel for separation of PCBs from other interferences. In general, silica gel adsorption column cleanup utilized 5.5 grams of 4% deactivated silica gel (100-200 mesh) placed in a 10.5 x 250 mm chromatography column with an upper layer of anhydrous sodium sulfate. The sample extract was added to the silica gel column and sequentially eluted with hexane which was concentrated to 1 mL with a Kuderna-Danish apparatus using a three ball Snyder Column on a steam bath before gas chromatographic analysis.

Total PCB Chemical Analysis. Dual-column congener-specific PCB analyses were based on capillary column procedures developed by Frame et al. (1996) and described previously (Pagano et al. 1999, Chiarenzelli et al. 2001, Stewart et al. 2008). Briefly, analytical instruments were recalibrated every five samples, with a system blank, instrument blank, and mid-level calibration check solution analyzed during each analytical run. A Hewlett-Packard (HP) Model 5890II GC with an electron capture detector (ECD - Ni⁶³) and autosampler was used for primary data acquisition. The capillary column utilized was a HP Ultra II, 25 meter with 0.22 mm id and 0.33 μ m film thickness. The calibration standard was a 1:1:1:1 mixture of Aroclors 1221, 1016, 1254 and 1260 each at 200 pg/uL, hexachlorobenzene (HCB) at 5 pg/uL, and p-p' DDE and Mirex each at 10 pg/uL (Custom Mix - AccuStandard, Inc., New Haven, CT), which allowed for the analysis of 99 chromatographic zones of 132 congeners/co-eluters.

PCB analyses were confirmed with a HP Model 5890 II gas chromatograph with an electron capture detector (Ni⁶³) and autosampler using a 60 meter DB-XLB capillary column with 0.25 mm id and 0.25 μ m film thickness. The calibration standard was a 1:1:1:1:1

mixture of congener mixtures C-CSQ-SETS 1-5 at 10pg/uL per individual congener (C-CSQ-SET; AccuStandard, Inc.) based on the work of Frame et al. (1996), allowing for the analysis of 122 chromatographic zones of 155 congeners/co-eluters. Total PCBs were determined by summation of all congeners/co-eluters. Method detection limits for total PCBs (by summation of congeners) were determined for adipose (87.7 ng/g, N=7) and liver (5.4 ng/g, N=6) by using the average method blank (MB) + STDEV of (MB) * student's t-statistic ($t_{0.99, n-1}$ DF).

Total PCB Quality Documentation. Laboratory quality assurance at The College at Oswego ERC is based on a quality management program developed from USEPA protocols (USEPA 1997, Pagano 2005, Holsen et al. 2008). The program consists of measurement quality objective acceptance criteria for replicate analyses, surrogate analyte recoveries, matrix spikes/matrix spike duplicates, and method, reagent and system blanks. Surrogate recoveries (SR, mean \pm std. dev.) for the various mink tissues analyzed in this project averaged: SR PCB_014 = 86.9 \pm 14.3%, SR PCB_065 = 86.4 \pm 17.6%, SR PCB_166 = 90.1 \pm 21.6% and SR PCT_003 = 83.1 \pm 20.4% (polychlorinated triphenyl). During the project, general laboratory quality assurance and silica gel method validation were determined by analysis (N=16) of National Institute of Standards and Technology (NIST) Standard Reference Material 1946 (Lake Superior Fish Tissue). Average recoveries \pm standard deviations of certified concentration values for PCBs were 92.9 \pm 5.9%.

Data Analysis

We used Microsoft® Excel 2000 for data management and non-statistical calculations and SPSS® 16.0 (SPSS Inc., Chicago, IL) for statistical analyses. We used General Linear Models (ANCOVA with two fixed factors: Area—AOC in vs. AOC out and Location—near lakeshore vs. inland, using the covariates sex, body weight, and percent lipid in adipose and liver, as appropriate) to analyze relationships between chemical concentrations and capture areas and locations in the study area. Bonferroni corrections for $\alpha = 0.05$ associated with multiple F-tests were employed. To meet the assumptions of ANOVA regarding normality and homoscedasticity, extreme but valid outliers in the chemical data (mink 17, 22 and 49 in Appendix 1) were reduced in magnitude compared to the rest of the data in their area-location groups (Tabachnick and Fidell 2007) and all chemical data were $\log_{10}(n+1)$ -transformed. Covariates percent lipid in adipose and liver were arcsine-transformed while body mass and sex were not transformed. After transformation, data for each of the five chemicals and three of the covariates (body mass and percent lipid in adipose and liver; sex has a binomial distribution) met the assumption of homogeneous variance (Levene's test, $P = 0.138-0.996$), and 30/32 chemical-area-location combinations, as well as the covariates of body mass and percent lipids in adipose and liver, met the assumption of normality (Shapiro-Wilk test; the two combinations that were not normally distributed were acceptably close; $P \geq 0.035$, Tabachnick and Fidell 2007). Zero values for chemical results below detection limits (BDL) were excluded to avoid skewing general trends low (BDL \neq no chemical present). Correlation analysis was used to evaluate relationships between concentrations of total PCB, dioxin-furan TEQ, and total Hg.

Results

Excluding one mink not analyzed and three BDL, 36 of 40 mink had mercury in brain (Appendix 1). Total Hg concentrations did not differ in and out of the AOC but concentrations in mink from near the Lake Ontario shore were significantly higher than those of inland mink ($P = 0.026$, Table 1). The sex and mass covariates were not significant.

Thirty-eight of 40 mink had sufficient adipose for total PCB analysis and 40 livers were analyzed for total PCB. Total PCB concentrations in adipose and liver did not differ in and out of the AOC but there were significant differences for total PCB between lakeshore and inland mink for adipose ($P = 0.001$) and liver ($P = 0.031$). Among the covariates used to analyze total PCB in adipose—sex, mass, and percent lipid in adipose—none were significant. Among the covariates used to analyze total PCB in liver—sex, mass, and percent lipid in liver—percent lipid ($P < 0.001$) was significant and sex ($P = 0.070$) and mass ($P = 0.081$) approached significance (Table 1).

Thirty-two and 18 of 40 mink had total dioxin-furan TEQ above detection limits in adipose and liver, respectively. Dioxin-furan TEQ in adipose and liver did not differ in and out of the AOC. Between lakeshore and inland mink there was a significant ($P = 0.016$) difference for dioxin-furan TEQ in adipose but not for liver ($P = 0.171$). Among the covariates used to analyze dioxin-furan TEQ in adipose and liver—sex, mass, and percent lipid in the respective tissue—none were significant (Table 1).

Correlations between concentrations of total Hg, total PCB, and dioxin-furan TEQ in brain, adipose and liver were high ($r = 0.591$ - 0.878) and significant ($P < 0.0001$ - 0.016 , Table 2). Furthermore, within laboratory correlations for adipose and liver were very high and significant for total PCB (ERC, $r = 0.982$, $P < 0.0001$) and dioxin-furan TEQ (CAS, $r = 0.748$, $P = 0.0009$).

Discussion

Except for dioxin-furan TEQ in liver (lowest concentrations and sample sizes), total Hg in brain, total PCB in adipose and liver, and dioxin-furan TEQ in adipose were significantly higher in mink captured near the shore of Lake Ontario than in those captured inland. There were no significant differences in total PCB, dioxin-furan TEQ, and total Hg concentrations in and out of the Rochester Embayment AOC, although mean concentrations for total PCB and dioxin-furan TEQ were higher in the AOC than out of the AOC by factors of 2-4 (Table 1). High variations in concentrations among animals resulted in low statistical power to distinguish quite different mean concentrations despite sample sizes of 10-20 mink per chemical both in and out of the AOC (Table 1). Total Hg concentrations differed little in and out of the AOC.

Concentrations of total PCB and dioxin-furan TEQ in liver in our study ($n = 40$ mink) ranged from 7-5,871 ng/g-ww (mean = 327) and BDL-47.6 ng/kg-ww (mean = 6.4), respectively (Appendix 1). For South Carolina ($n = 6$) and Louisiana ($n = 6$) mink, Tansy et al. (2003) reported total PCB of 133-413 (mean = 263) and 65-238 (mean = 139) ng/g-ww, respectively. Mean dioxin-furan TEQ in South Carolina and Louisiana were 21.0 and 13.8 ng/kg-ww (reported as pg/g-ww by Tansy et al. 2003), respectively. In our study two mink had very high total PCB and three had very high dioxin-furan TEQ in liver (see below). Excluding these data, total PCB and dioxin-furan TEQ in our study ranged from 7-755 ng/g-

ww (mean = 127) and BDL-4.2 ng/kg (mean = 1.0), respectively. Overall, excluding the mink with exceptionally high concentrations, total PCB concentrations in mink in our study area are similar to or lower than those in Louisiana and South Carolina, respectively, while dioxin-furan TEQ in our study was far below that reported by Tansy et al. (2003).

Due to expense, no co-planar PCB data were collected for this project; therefore, the dioxin-furan TEQ values reported here are lower than total TEQ. Co-planar PCBs accounted for 20-25% of total TEQ in wild adult male mink in Louisiana and South Carolina, respectively (Tansy et al. 2003), and for 43% in kits and juveniles in a laboratory study (Martin et al. 2006). Therefore, multiplying the dioxin-furan TEQ values we report by factors of 1.25 and 2.0 gives the likely range of total TEQ in mink in our study.

The highest mean dioxin-furan TEQ concentration in mink liver was 14.8 ng/kg-ww near the lakeshore in the AOC, and the lowest mean concentration was 0.3 ng/kg-ww inland out of the AOC (Table 1). Multiplying these lowest and highest mean dioxin-furan TEQ concentrations by factors of 1.25 and 2.0, respectively, predicts the range of mean total TEQ in mink liver across the entire study area: 0.4-29.6 ng/kg-ww. The lowest LOAEL (lowest observable adverse effect levels) for total TEQ in liver known for mink (cancerous jaw lesions) is 40.2 ng/kg-ww (Bursian et al. 2006 a-c). Therefore, most mink in the entire study area are unlikely to be adversely affected by total TEQ.

Two mink (17, 22), captured in the AOC near the shore of Lake Ontario, had exceptionally high concentrations of total PCB in liver: 5,871 and 2,389 ng/g-ww, respectively (Appendix 1). The lowest LOAEL (lowest observable adverse effect level) for total PCB in liver known for mink (cancerous jaw lesions) is 1,698 ng/g-ww (Bursian et al. 2006 a-c). The same two mink also had high dioxin-furan TEQ in liver: 21.3 and 47.6 ng/kg-ww, respectively. Mink 49, caught near the lakeshore out of the AOC, also had high dioxin-furan TEQ in liver (31.8 ng/kg-ww). Again using the factors 1.25 and 2.0, low and high total TEQ in liver for these mink likely ranged from 26.6-95.2 ng/kg-ww. Therefore, the most highly exposed mink along the lakeshore in the study area potentially may suffer from cancerous jaw lesions from either total PCB or total TEQ (see Haynes et al. 2009).

Several factors may account for the high levels of total PCB and dioxin-furan TEQ in mink 17 and 22. First, these mink were caught in the Braddock Bay Wildlife Management Area (Figure 1) that has a broad connection to Lake Ontario water and its food web, including the carcasses of migrating salmonines with high contaminant concentrations (O'Toole et al. 2006, Carlson and Swackhamer 2006) each fall. Second, there may be small toxic hot spots in the BBWMA to which mink are exposed through their food web. Haynes et al. (2004) reported that one sediment sample from a creek near where mink 17 was captured had a concentration of 1.5 ppm total PCB. Mink 22 was captured near another area of the BBWMA that was a munitions factory during and after World War II. In contrast, lakeshore mink out of the AOC were captured in a much smaller watershed that has fewer migrating salmonines and no suspected toxic hotspots. Third, stable isotope analysis (see Wellman et al. 2009) showed that mink 17 ($\delta N = 16.9$) fed one trophic level higher than other lakeshore mink ($\delta N = 12.7$) but mink 22 ($\delta N = 11.09$) and 49 ($\delta N = 11.63$) fed below the average trophic level of lakeshore mink, again suggesting that fish migrating from Lake Ontario may have comprised a high proportion of the diet of mink 17.

Conclusion

Mink captured near the shore of Lake Ontario (i.e., potentially in contact with the lake's food web) had significantly higher concentrations of total Hg in brain, total PCB and dioxin-furan TEQ in adipose, and total PCB in liver than inland mink. Concentrations of dioxin-furan TEQ in liver did not differ between mink caught near the lakeshore and inland. Although concentrations in mink in the inland portion of the Rochester Embayment AOC were generally higher than those in inland mink outside of the AOC, highest concentrations did not exceed LOAEL concentrations for total PCB and estimated total TEQ (Bursian et al. 2006 a-c). It appears that sources of PCB and TEQ originating in the AOC, if any, are not contributing to the “degradation of fish and wildlife populations” and “bird or animal deformities or reproductive problems” use impairments identified in the RAP (1993, 1997). The major source of these chemicals appears to be contact with the Lake Ontario food web.

Correlations among concentrations of total PCB and dioxin-furan TEQ in adipose and liver and total Hg in brain were high and highly significant. This finding suggests that fewer tissues and, possibly, fewer chemicals need to be monitored in the future in the Rochester Embayment of Lake Ontario to assess the status of chemicals of concern in mink and other sentinel species. Liver is the best tissue to sample for PCB and TEQ because detectable levels of these chemicals concentrate there and concentrations in liver associated with a variety of adverse impacts are known (Bursian et al. 2006 a-c). However, chemicals of emerging concern (e.g., polybrominated diethyl ethers, PBDE) should not be ignored in future sampling to evaluate chemical contamination in the AOC.

For biomonitoring and remediation purposes in the RELO AOC and other locations with mink, before engaging in costly, detailed, congener-specific analyses, we recommend a four-step approach assess the potential for toxicity to mink. 1) Use the latest USEPA data to determine the concentrations of PCB and TEQ in mink water supplies and use the modeling approach described in Wellman et al. (2009) to determine if estimated concentrations in mink are likely to cause jaw lesions. 2) If concentrations sufficient to cause jaw lesions are predicted by modeling, capture mink and evaluate the prevalence of jaw lesions (Haynes et al. 2009). 3) If jaw lesions are found, find and remediate sources of PCB and TEQ contaminating water in the area of concern. 4) Repeat steps 1-3 every 5-10 years until no evidence of jaw lesions is found, at which time the “bird or animal deformities or reproductive problems” or “degradation of fish and wildlife populations” use impairments can be “delisted” by the IJC.

Acknowledgments

Randall Baase, Matt Lochner, and other trappers contributed carcasses; Scott Wells and Ross Abbett dissected the mink. “Doc” Fink, Dick Sands, Mel Reber, and Al Burkhart allowed us to collect mink on their property, as did James Slusarczyk of the New York State Office of Parks, Recreation, and Historic Preservation. Gregory M. Sumner prepared samples for gas chromatographic analysis. Carol Zollweg created Figure 1. Two anonymous reviewers greatly improved the quality of the manuscript. This project was funded by grant C302399 from the New York State Great Lakes Protection Fund.

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Table 1. Concentrations of total mercury, total PCBs, and dioxin-furan TEQs in mink from the four study areas: In AOC/Lakeshore, In AOC/Inland, Out of AOC/Lakeshore, and Out of AOC/Inland. Means and standard deviations are actual values but statistics were calculated using log₁₀-transformed data for all chemicals and arcsine-transformed percent lipid in adipose and liver. N/A = not applicable.

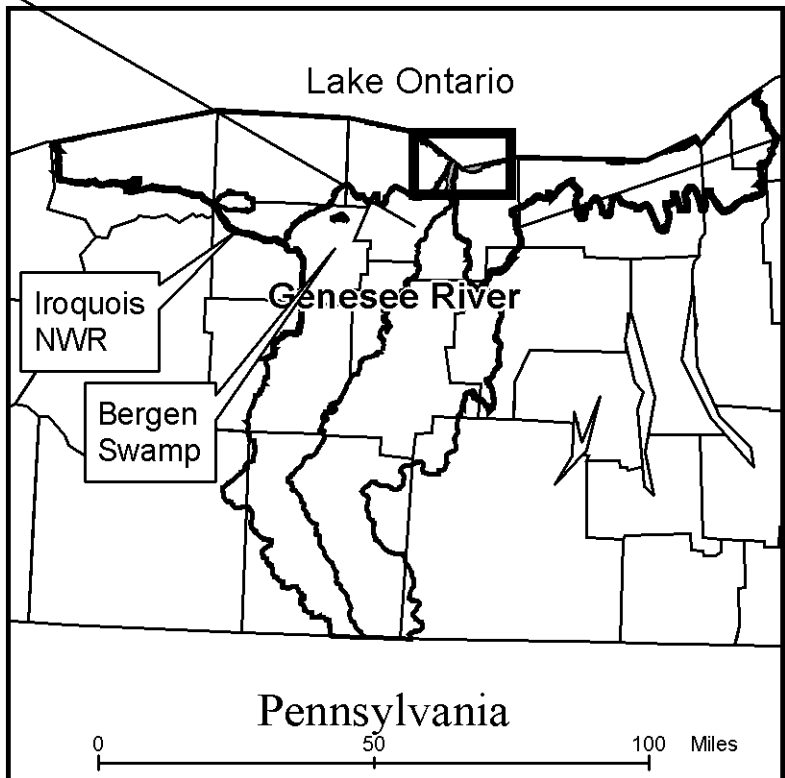
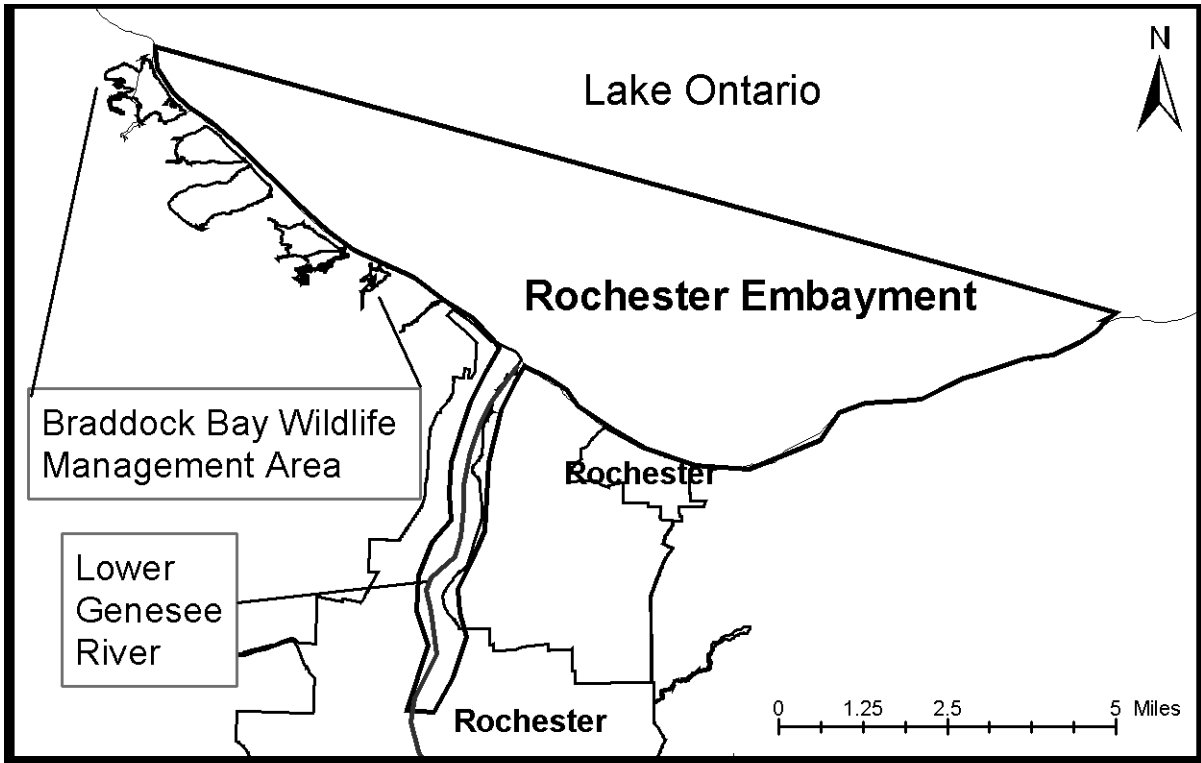
Chemical	Area/Location	N	Mean (SD)	Lakeshore/ Inland P	In AOC/ Out AOC P	Interaction P	Covariate P	Power
Mercury (brain) (mg/kg-wet wt)	In AOC/Lakeshore	9	0.42 (0.44)					
	In AOC/Inland	9	0.16 (0.16)					
	Out AOC/Lakeshore	9	0.30 (0.16)	0.026				0.620
	Out AOC/Inland	9	0.19 (0.15)		0.853			0.054
	Interaction					0.920		0.051
	Sex covariate						0.838	0.055
	Mass covariate						0.661	0.071
% lipid covariate						N/A	N/A	
Total PCB (adipose) (ng/g-wet wt)	In AOC/Lakeshore	10	13610 (31129)					
	In AOC/Inland	10	1680 (2502)					
	Out AOC/Lakeshore	10	3970 (3281)	0.001				0.954
	Out AOC/Inland	8	387 (226)		0.492			0.103
	Interaction					0.316		0.167
	Sex covariate						0.345	0.154
	Mass covariate						0.210	0.237
% lipid covariate						0.208	0.238	
Total PCB (liver) (ng/g-wet wt)	In AOC/Lakeshore	10	957 (1873)					
	In AOC/Inland	10	101 (175)					
	Out AOC/Lakeshore	10	231 (227)	0.017				0.688
	Out AOC/Inland	10	30 (13)		0.429			0.122

	Interaction					0.035		0.571
	Sex covariate						0.070	0.442
	Mass covariate						0.081	0.416
	% lipid covariate						<0.001	0.998
TEQ (adipose) (ng/kg-wet wt)	In AOC/Lakeshore	8	51.4 (116.4)					
	In AOC/Inland	9	4.8 (4.4)					
	Out AOC/Lakeshore	9	15.4 (12.2)	0.016				0.698
	Out AOC/Inland	6	5.0 (5.3)		0.471			0.108
	Interaction					0.957		0.050
	Sex covariate						0.319	0.164
	Mass covariate						0.523	0.095
	% lipid covariate						0.269	0.192
TEQ (liver) (ng/kg-wet wt)	In AOC/Lakeshore	5	14.8 (20.3)					
	In AOC/Inland	5	1.0 (1.8)					
	Out AOC/Lakeshore	5	7.2 (13.8)	0.171				0.268
	Out AOC/Inland	3	0.3 (0.4)		0.642			0.072
	Interaction					0.931		0.051
	Sex covariate						0.287	0.176
	Mass covariate						0.556	0.086
	% lipid covariate						0.220	0.221

Table 2. Correlations of concentrations of total PCB and dioxin-furan TEQ in adipose and liver and total mercury (Hg) in the brain of mink.

	Adipose Dioxin-furan TEQ	Total PCB	Liver Dioxin-furan TEQ	Total PCB
Total Hg				
r =	0.796	0.733	0.591	0.713
P =	0.0002	0.0012	0.0160	0.0019
n =	31	34	32	35
Total PCB (adipose)				
r =	0.878	----	0.855	0.959
P =	<0.0001	----	<0.0001	<0.0001
n =	30	----	31	38

Figure 1. Map showing the four regions of mink capture. AOC/Lakeshore is the Braddock Bay Wildlife Management Area (BBWMA), AOC/Inland is mostly near the Bergen Swamp ~34 km south of the BBWMA, and Out of AOC/Lakeshore began 15 km west of the BBWMA. Out of AOC/Inland is the Iroquois National Wildlife Refuge ~50 km southwest of the BBWMA and beyond the eastern end of Lake Ontario ~180 km east of the BBWMA (not shown on the map).



Appendix 1. Geographical, biological and chemical data for the 40 mink in this study.

Specimen	Area	Location	Capture Season	Age ^a (years)	Gender	Weight ^b (g)	% Lipid ^c Adipose	% Lipid Liver	TPCB	TPCB	TEQ	TEQ	Mercury
									Adipose (ng/g-ww)	Liver (ng/g-ww)	Adipose ^{d,e} (ng/kg-ww)	Liver (ng/kg-ww)	Brain ^f (mg/kg-ww)
1	In AOC	Inland	Winter	0.7	M	830	81.5	0.6	197	9	0.03	0.1	0.07
3	Out AOC	Inland	Winter	0.7	M	862	72.8	1.5	212	12	4.8	BDL	0.04
5	Out AOC	Inland	Winter	0.7	M	846	48.1	0.7	224	10	BDL	BDL	0.12
10	Out AOC	Inland	Winter	ND	M	652	53.2	0.9	233	8	0.4	0.01	0.40
11	Out AOC	Inland	Winter	0.7	M	879	63.0	1.1	652	28	BDL	BDL	0.07
14	Out AOC	Inland	Winter	0.7	M	642	75.0	1.4	104	7	AE	0.03	0.01
17	In AOC	Lakeshore	Winter	ND	M	815	52.5	4.4	100838	5871	339	21.3	1.55
20	In AOC	Lakeshore	Winter	ND	M	996	83.2	2.8	864	86	BDL	BDL	BDL
21	In AOC	Lakeshore	Winter	2.7	F	453	41.8	5.5	2918	682	AE	3.5	0.37
22	In AOC	Lakeshore	Winter	0.7	M	967	76.3	2.5	18659	2389	22.4	47.6	0.37
23	In AOC	Inland	Summer	ND	M	673	68.1	1.1	508	18	BDL	BDL	BDL
24	In AOC	Inland	Winter	0.6	M	734	53.5	1.6	300	27	3.0	0.6	0.05
30	Out AOC	Inland	Winter	ND	M	587	IT	1.6	IT	45	IT	BDL	NA
31	Out AOC	Inland	Winter	1.6	M	642	76.9	2.5	543	31	2.7	BDL	0.27
32	Out AOC	Inland	Winter	3.6	M	575	68.8	0.9	705	14	5.8	BDL	0.19
33	Out AOC	Inland	Winter	0.6	F	351	IT	0.6	IT	19	1.6	BDL	0.25
34	Out AOC	Inland	Winter	0.6	F	485	69.9	1.3	424	32	14.9	0.8	0.39
38	In AOC	Lakeshore	Winter	1.7	F	635	79.7	1.3	4593	214	10.8	0.3	0.25
39	In AOC	Lakeshore	Winter	ND	M	681	68.8	2.2	515	35	1.2	1.2	0.09
41	In AOC	Lakeshore	Winter	ND	M	578	32.6	2.7	378	32	3.6	BDL	0.11
43	In AOC	Inland	Winter	3.7	M	930	79.8	1.5	469	30	1.6	0.1	0.04
44	In AOC	Inland	Winter	3.7	M	879	51.8	1.0	415	13	0.3	0.01	0.03
45	In AOC	Inland	Winter	3.7	M	555	48.8	2.0	505	29	3.2	BDL	0.30
46	Out AOC	Lakeshore	Spring	4.0	M	694	68.1	2.8	6071	230	19.6	2.1	0.50

47	Out AOC	Lakeshore	Winter	2.7	M	742	84.6	1.2	1238	43	9.4	1.0	0.12
48	Out AOC	Lakeshore	Winter	ND	M	651	42.9	2.2	2360	185	BDL	BDL	BDL
49	Out AOC	Lakeshore	Winter	ND	M	704	40.0	1.2	11653	755	38.3	31.8	0.30
50	Out AOC	Lakeshore	Winter	ND	M	1013	60.0	1.3	4373	171	9.2	0.2	0.13
51	Out AOC	Lakeshore	Winter	3.8	M	1111	68.6	1.4	4778	411	30.1	BDL	0.29
52	Out AOC	Lakeshore	Winter	1.7	M	916	70.2	1.4	1419	69	5.4	BDL	0.28
53	Out AOC	Lakeshore	Winter	0.6	M	619	56.6	0.9	232	14	5.3	0.9	0.12
54	Out AOC	Lakeshore	Spring	3.0	M	995	77.1	0.7	2783	67	3.5	BDL	0.50
55	Out AOC	Lakeshore	Winter	ND	M	1039	71.6	2.0	4797	360	18.0	BDL	0.43
56	In AOC	Lakeshore	Winter	1.0	F	456	69.6	0.8	525	15	7.7	BDL	0.21
57	In AOC	Lakeshore	Winter	2.7	F	361	64.3	1.7	4642	153	10.2	BDL	0.35
58	In AOC	Lakeshore	Winter	0.9	M	766	79.5	2.3	2170	96	16.1	BDL	0.50
59	In AOC	Inland	Winter	3.7	M	983	60.5	0.5	316	11	3.4	BDL	0.06
61	In AOC	Inland	Winter	4.8	M	695	64.3	2.5	1615	64	9.0	BDL	0.13
62	In AOC	Inland	Spring	ND	M	560	53.1	1.7	5010	250	12.6	4.2	0.46
63	In AOC	Inland	Spring	1.0	M	863	38.2	2.7	7465	554	9.1	BDL	0.34

^a ND = no data

^b body weight without tail and skin which add an average of 18% to body weight

^c IT = insufficient adipose tissue for analysis

^d BDL = below detection limit

^e AE = analytical error

^f NA = not analyzed