Appendix A-2: Data Record Sheets

Figure A-2a. Sample System Log**.** During each field service session, we recorded the date of service, the ID numbers of the videotape cassettes and batteries inserted, and any comments such as mink tracks observed, operational failures, or other pertinent information.

MustelaVision

System Log C

Location (Creek/Road)	Tape #	Battery #	2003 Start Date	End Date	Tracks/Comments	# Mink Sights
RPC	5	1	6/14/03	6/21		0
	10	10	6/21	6/26		0
	20	2	6/26	7/2	mink tracks	1
	20	10	7/2	7/8		1
	39	23	7/8	7/14	battery down: VCR won't record	nil on tape 39
	14	3	7/14	7/20	did record (tape full of veg/wind)	0
	9	21	7/20	7/2[illegible]		
	39	2	7/28	8/4	tracks: mink + weasel	2
	3	23	8/4	8/11		1
	15	12	8/11	8/18		0
	17	26	8/18	8/25		
	15	4	8/25	9/1		0
	18	26	9/1	9/8		1
	13	[illegible]	9/8	9/16		0
	3	12	9/16	9/23	weasel + small mink	1
	6	26	9/23	10/1		0
	20	13	10/1	10/7		0
	12	14	10/7	10/14		1
	37	11	10/14	10/21		0
	15	13	10/21	10/27	pulled system	0
2004			2004			
Sour Springs Rd	1	22	6/25	7/1		0
	8	28	7/1	7/8		1
	7	39	7/8	7/15		0
	39	1	7/15	7/22		0
	38	36	7/22	7/29		0
	16	47	7/29	8/5	Fuse blew	0
	18	37	8/5	8/12	Replaced fuse	?
	25	39	8/12	8/19		0
	19	66	8/19	8/26		0

System Log C

Figure A-2b. Sample Tape Log. The start and end dates for each session generated the Max Trap Nights (TN) for that session, and the day (D) and night (N) periods, recorded as they occurred in the video, yielded the Min TN for that session.

MustelaVision
Tape Log 36

		2003 Date		Tape Counter		Trigger		
Location (Creek/Road)	System	Start	End	Session	Cue	Day	Night	Animal
INWR Feeder Rd	H	7/7	7/13	19:		1		Heron
		DD	N					
Bogus C./LOSP	A	7/28	8/4	3:16:55		IIII		Ducks
		DD	NN			II		Heron
						1		Spider
							1	Raccoons
					5:11:14	1		Heron from woods (20+ min)
Tonawanda Rt 77	E	8/14	8/21	1:42: ★	21:31	1	✓	Mink Mink_Day_Tonawanda_Rt77_08_14_03_A
		DDDDDD	NNNNN	★	22:27	1	✓	Mink " " " _B
				★	26:26		1 ✓	"Mink_Night_Tonawanda_Rt77_08-14-03
				★	27:57		1 ✓	2 Minks 2 Minks_Night_Ton_Rt77_08-14-03
				★	59:32	1	✓	Mink Mink_Day_Tonawanda_Rt77_08-15-03
INWR Sour Springs	G	8/28	9/4	1:08:		IIIIIII+		Ducks
		DDDD	NNN	★	7:40	1	✓	Mink Mink_INWR_Sour_Springs_08_26_03
						1		Rail?
					54:04	1	✓	Mink Mink_Tail_Night_INRW_Sour Springs_08_28_03
					55:07	1	✗	" Could't capture
					57:26		✓	Ducks_under_attack_INWR_Sour_Springs_08_28_03 Ducks being attacked
					1:05:22		✓	Mink? (fur in LR corner) 0_INWR_S_08_28_03 Muskrat?
Tonawanda Rt 77	E	9/25	10/2	29:			IIV	Muskrats
		DDDDD	NNNNN	★	12:26	9/26	✗	~~Weasel or Mink~~ Mouse
				★	13:31	9/27	✓	Mink_Tonawanda_Rt77_day_09_27_03
				★	20:05	9/29	✓	Weasel! " _09_29_03
						1		Heron

Tape Log 36

Appendix B: Passage Rate Analyses (Delta PRs)

Table B-1. Mean Delta PRs

One-Sample T: Dmin, Dmax

```
Test of mean Delta PR = 0 vs mean Delta PR not = 0

Variable          N      Mean     StDev   SE Mean
Dmin              9     0.159     0.693     0.231
Dmax              9    -0.109     0.392     0.131

Variable             95.0% CI            T      P
Dmin          (  -0.374,   0.691)     0.69  0.511
Dmax          (  -0.410,   0.192)    -0.83  0.428
```

Table B-2. Delta PRs: AOC: In (trapped for this study) vs. Out (trapped historically)

Two-Sample T-Test and CI: Dmax**, Area**

```
Area        N      Mean     StDev   SE Mean
AOC         5   -0.0970    0.0919     0.041
Out         4    -0.124     0.630      0.32

Difference = mu (AOC) - mu (Out)
Estimate for difference:  0.027
95% CI for difference: (-0.984, 1.038)
T-Test of difference = 0 (vs not =): T-Value = 0.08  P-Value = 0.938  DF = 3
```

Two-Sample T-Test and CI: Dmin**, Area**

```
Area        N      Mean     StDev   SE Mean
AOC         5   -0.0018    0.0497     0.022
Out         4      0.36      1.09      0.54

Difference = mu (AOC) - mu (Out)
Estimate for difference:  -0.361
95% CI for difference: (-2.092, 1.369)
T-Test of difference = 0 (vs not =): T-Value = -0.66  P-Value = 0.554  DF = 3
```

Appendix C: Regional Descriptor Analyses

Appendix C-1. Descriptive Statistics: Regional Descriptors

Table C-2a. Descriptive Statistics: AOC: In vs. Out

Variable	AOC:In vs. Out	N	N*	Mean	SE Mean	StDev	Minimum	Q1
Max	In	16	0	0.0768	0.0238	0.0950	0.00000	0.00281
	Out	13	0	0.1675	0.0611	0.2203	0.00000	0.00000
Min	In	16	0	0.0399	0.0130	0.0522	0.00000	0.00210
	Out	13	0	0.0859	0.0339	0.1222	0.00000	0.00000

Variable	AOC	Median	Q3	Maximum
Max	In	0.0340	0.0960	0.3077
	Out	0.0471	0.4249	0.5714
Min	In	0.0147	0.0536	0.1552
	Out	0.0317	0.1671	0.3902

Table C-2b. Descriptive Statistics: Inland vs. Lakeshore

Variable	Lakeshore vs.Inland	N	N*	Mean	SE Mean	StDev	Minimum	Q1
Max	Inland	17	0	0.1424	0.0479	0.1977	0.00000	0.0000
	Lakeshore	12	0	0.0821	0.0309	0.1069	0.00000	0.0116
Min	Inland	17	0	0.0720	0.0266	0.1097	0.00000	0.0000
	Lakeshore	12	0	0.0443	0.0168	0.0583	0.00000	0.00774

Variable	Lakeshore vs.Inland	Median	Q3	Maximum
Max	Inland	0.0755	0.2449	0.5714
	Lakeshore	0.0251	0.1751	0.3077
Min	Inland	0.0290	0.0827	0.3902
	Lakeshore	0.0119	0.0834	0.1552

Table C-2c. Descriptive Statistics: Landscape: Wetlands vs. Mix

Variable	Landscape	N	N*	Mean	SE Mean	StDev	Minimum	Q1
Max	Mix	12	0	0.0352	0.0106	0.0368	0.00000	0.00000
	Wetlands	17	0	0.1755	0.0479	0.1974	0.00000	0.00562
Min	Mix	12	0	0.01802	0.00557	0.01930	0.00000	0.00000
	Wetlands	17	0	0.0905	0.0267	0.1102	0.00000	0.00420

Variable	Landscape	Median	Q3	Maximum
Max	Mix	0.0240	0.0744	0.0968
	Wetlands	0.0938	0.3462	0.5714
Min	Mix	0.01220	0.03106	0.05556
	Wetlands	0.0494	0.1551	0.3902

Appendix C-2: Main Effects Plots

Figure C-1a. Main effects plot showing effects of Regional Descriptors on Max PR.

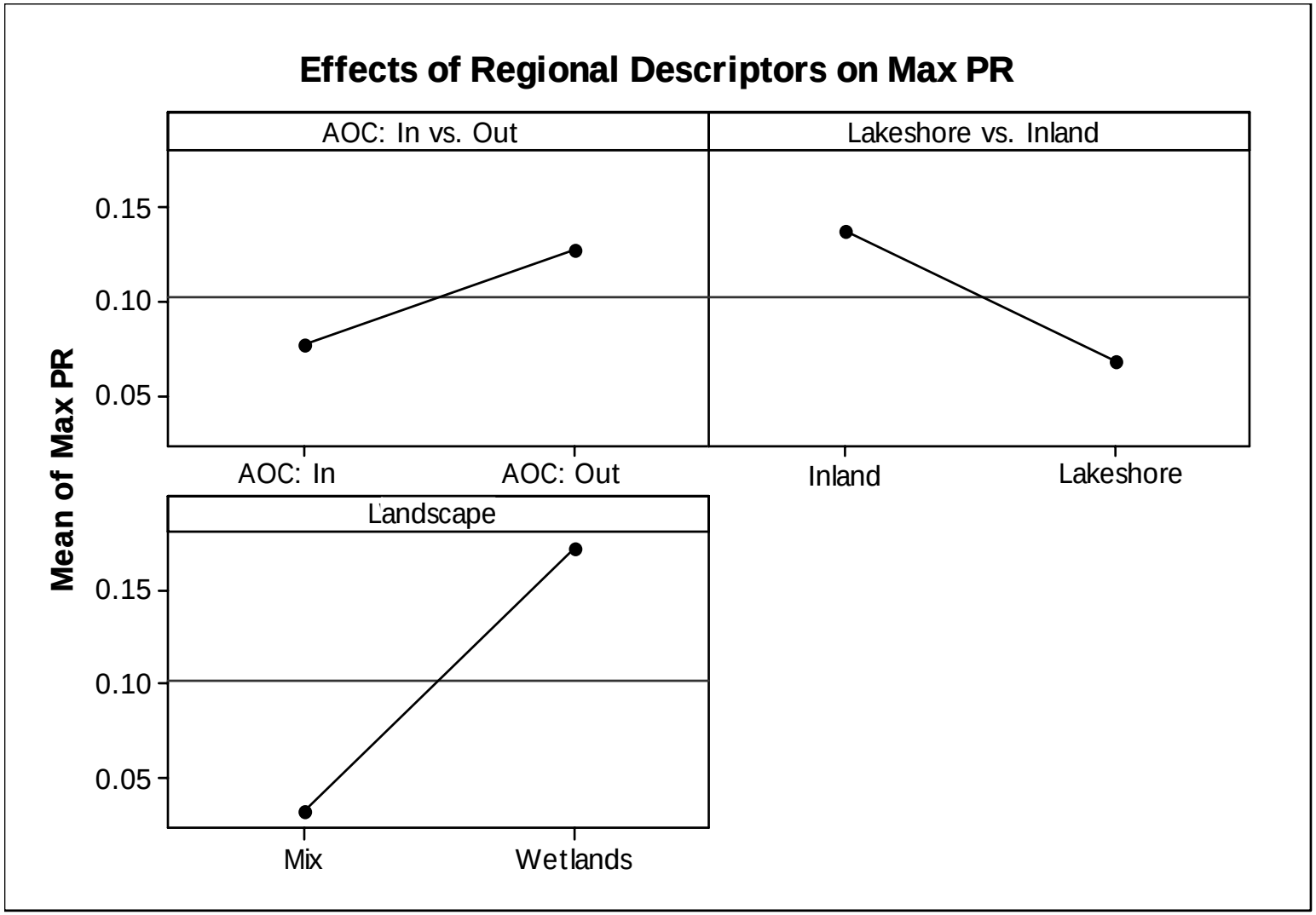
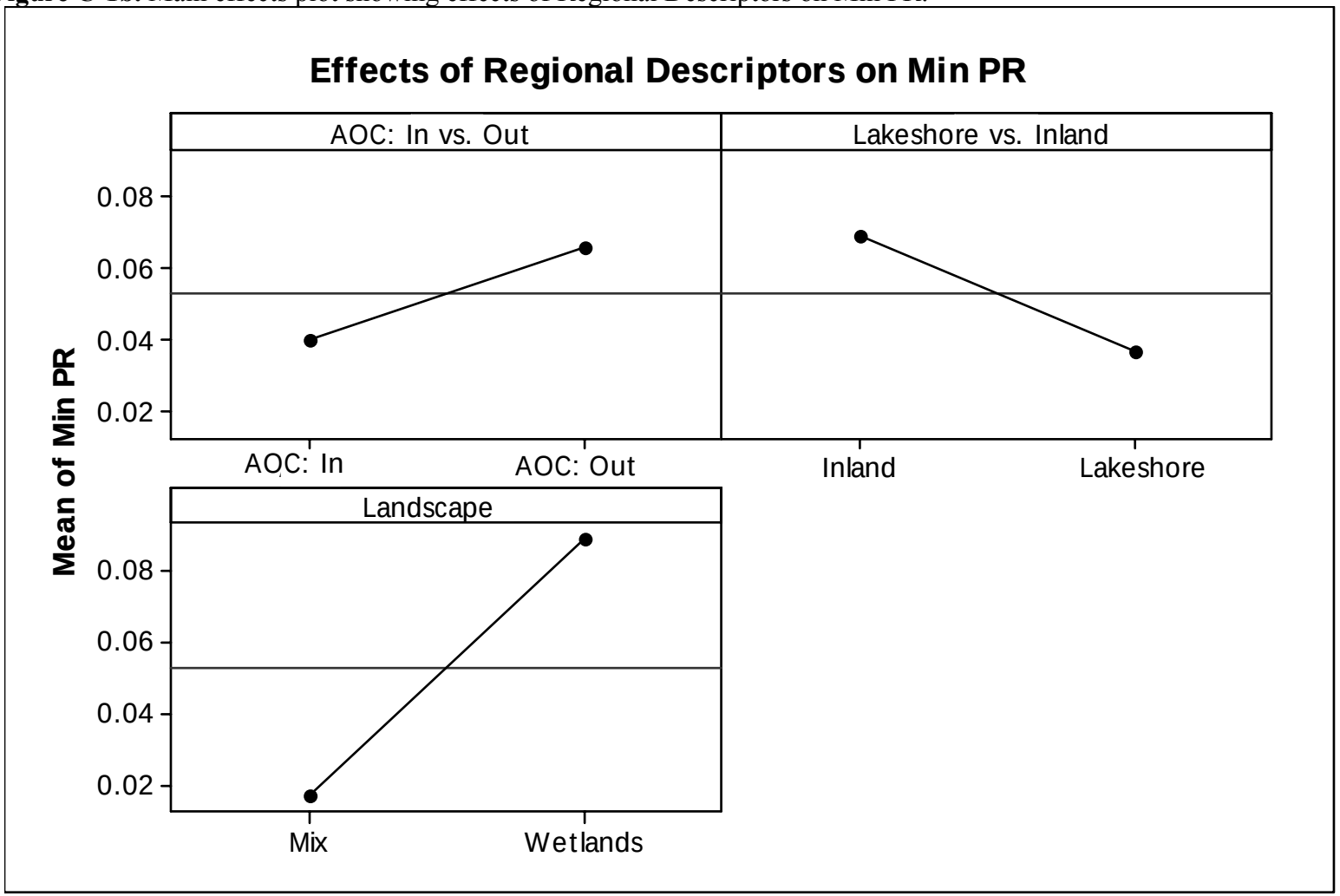


Figure C-1b. Main effects plot showing effects of Regional Descriptors on Min PR.



Appendix C-3. General Linear Model: Regional Descriptors

Table C-3. General Linear Model: Regional Descriptors

Factor	Type	Levels	Values
AOC	fixed	2	AOC, Out
Shore	fixed	2	Inland, Shore
Wetlands	fixed	2	Mix, Wetlands

Analysis of Variance for Max, using Adjusted SS for Tests

Source	DF	Seq SS	Adj SS	Adj MS	F	P
AOC	1	0.05893	0.01658	0.01658	0.72	0.404
Lakeshore	1	0.01323	0.03176	0.03176	1.38	0.251
Wetlands	1	0.12819	0.12819	0.12819	5.56	**0.026**
Error	25	0.57615	0.57615	0.02305		
Total	28	0.77651				

S = 0.151810 R-Sq = 25.80% R-Sq(adj) = 16.90%

Unusual Observations for Max

Obs	Max	Fit	SE Fit	Residual	St Resid
24	0.571429	0.231957	0.050603	0.339472	2.37 R

R denotes an observation with a large standardized residual.

Analysis of Variance for Min, using Adjusted SS for Tests

Source	DF	Seq SS	Adj SS	Adj MS	F	P
AOC	1	0.015166	0.004399	0.004399	0.60	0.446
Shore	1	0.002547	0.006896	0.006896	0.94	0.342
Wetlands	1	0.033783	0.033783	0.033783	4.59	**0.042**
Error	25	0.183830	0.183830	0.007353		
Total	28	0.235325				

S = 0.0857507 R-Sq = 21.88% R-Sq(adj) = 12.51%

Unusual Observations for Min

Obs	Min	Fit	SE Fit	Residual	St Resid
24	0.390244	0.117964	0.028584	0.272280	3.37 R

R denotes an observation with a large standardized residual.

Appendix D: Site Descriptor Analyses

Appendix D-1. Descriptive Statistics

Table D-1a. Descriptive Statistics: Cover

Variable	Cover	N	N*	Mean	SE Mean	StDev	Minimum	Q1
Max	Brush	8	0	0.0942	0.0387	0.1095	0.000000000	0.00313
	Cattails	12	0	0.1843	0.0643	0.2227	0.000000000	0.000000000
	Forest	9	0	0.0490	0.0255	0.0765	0.000000000	0.00562
Min	Brush	8	0	0.0566	0.0227	0.0643	0.000000000	0.00188
	Cattails	12	0	0.0935	0.0360	0.1248	0.000000000	0.000000000
	Forest	9	0	0.0200	0.0100	0.0300	0.000000000	0.00420

Variable	Cover	Median	Q3	Maximum
Max	Brush	0.0673	0.1759	0.3077
	Cattails	0.0846	0.4450	0.5714
	Forest	0.0196	0.0655	0.2407
Min	Brush	0.0397	0.1302	0.1552
	Cattails	0.0471	0.1969	0.3902
	Forest	0.00847	0.0247	0.0963

Table D-1b. Descriptive Statistics: Habitat

Variable	Habitat	N	N*	Mean	SE Mean	StDev	Minimum	Q1
Max	Mix	13	0	0.1125	0.0459	0.1655	0.000000000	0.00562
	Upland	8	0	0.0512	0.0289	0.0819	0.000000000	0.000000000
	Wetland	8	0	0.1918	0.0757	0.2142	0.000000000	0.00746
Min	Mix	13	0	0.0706	0.0305	0.1101	0.000000000	0.00376
	Upland	8	0	0.0204	0.0115	0.0324	0.000000000	0.000000000
	Wetland	8	0	0.0842	0.0337	0.0954	0.000000000	0.00385

Variable	Habitat	Median	Q3	Maximum
Max	Mix	0.0471	0.1538	0.5714
	Upland	0.0189	0.0744	0.2407
	Wetland	0.0846	0.4450	0.4857
Min	Mix	0.0317	0.1065	0.3902
	Upland	0.00871	0.0268	0.0963
	Wetland	0.0471	0.1969	0.2299

Table D-1c. Descriptive Statistics: Ledge

Variable	Ledge	N	N*	Mean	SE Mean	StDev	Minimum	Q1
Max	No	18	0	0.1214	0.0421	0.1788	0.000000000	0.000000000
	Yes	11	0	0.1109	0.0459	0.1524	0.000000000	0.000000000
Min	No	18	0	0.0610	0.0235	0.0999	0.000000000	0.000000000
	Yes	11	0	0.0597	0.0244	0.0810	0.000000000	0.000000000

Variable	Ledge	Median	Q3	Maximum
Max	No	0.0385	0.1391	0.5714
	Yes	0.0476	0.2022	0.4651
Min	No	0.0236	0.0676	0.3902
	Yes	0.0204	0.1550	0.2299

Table D-1d. Descriptive Statistics: Tunnel

Variable	Tunnel	N	N*	Mean	SE Mean	StDev	Minimum	Q1
Max	No	19	0	0.0557	0.0227	0.0991	0.000000000	0.000000000
	Yes	10	0	0.2348	0.0658	0.2081	0.0112	0.0641
Min	No	19	0	0.02219	0.00753	0.03281	0.000000000	0.000000000
	Yes	10	0	0.1333	0.0387	0.1225	0.00847	0.0374

Variable	Tunnel	Median	Q3	Maximum
Max	No	0.0182	0.0833	0.3846
	Yes	0.1538	0.4703	0.5714
Min	No	0.00840	0.03175	0.10753
	Yes	0.1065	0.2275	0.3902

Appendix D-2. One-Way ANOVAs: Site Descriptors

Table D-2a. Cover

Factor	Type	Levels	Values
Cover	fixed	3	Brush, Cattails, Forest

One-way ANOVA: Max versus Cover

Source	DF	SS	MS	F	P
Cover	2	0.1000	0.0500	1.92	0.166
Error	26	0.6765	0.0260		
Total	28	0.7765			

S = 0.1613 R-Sq = 12.88% R-Sq(adj) = 6.18%

One-way ANOVA: Min versus Cover

Source	DF	SS	MS	F	P
Cover	2	0.02794	0.01397	1.75	0.193
Error	26	0.20739	0.00798		
Total	28	0.23533			

S = 0.08931 R-Sq = 11.87% R-Sq(adj) = 5.09%

Table D-2b. Habitat

Factor	Type	Levels	Values
Habitat	fixed	3	Mix, Upland, Wetland

One-way ANOVA: Max versus Habitat

Source	DF	SS	MS	F	P
Habitat	2	0.0797	0.0398	1.49	0.245
Error	26	0.6968	0.0268		
Total	28	0.7765			

S = 0.1637 R-Sq = 10.26% R-Sq(adj) = 3.36%

One-way ANOVA: Min versus Habitat

Source	DF	SS	MS	F	P
Habitat	2	0.01869	0.00935	1.12	0.341
Error	26	0.21663	0.00833		
Total	28	0.23533			

S = 0.09128 R-Sq = 7.94% R-Sq(adj) = 0.86%

Table D-2c. Ledge

Factor	Type	Levels	Values
Ledge	fixed	2	No, Yes

One-way ANOVA: Max versus Shelf

Source	DF	SS	MS	F	P
Shelf	1	0.0008	0.0008	0.03	0.872
Error	27	0.7758	0.0287		
Total	28	0.7765			

S = 0.1695 R-Sq = 0.10% R-Sq(adj) = 0.00%

One-way ANOVA: Min versus Shelf

Source	DF	SS	MS	F	P
Shelf	1	0.00001	0.00001	0.00	0.970
Error	27	0.23531	0.00872		
Total	28	0.23533			

S = 0.09336 R-Sq = 0.01% R-Sq(adj) = 0.00%

Table D-2d. Tunnel

Factor	Type	Levels	Values
Tunnel	fixed	2	No, Yes

One-way ANOVA: Max versus Tunnel

Source	DF	SS	MS	F	P
Tunnel	1	0.2101	0.2101	10.02	**0.004**
Error	27	0.5664	0.0210		
Total	28	0.7765			

S = 0.1448 R-Sq = 27.06% R-Sq(adj) = 24.36%

One-way ANOVA: Min versus Tunnel

Source	DF	SS	MS	F	P
Tunnel	1	0.08089	0.08089	14.14	**0.001**
Error	27	0.15444	0.00572		
Total	28	0.23533			

S = 0.07563 R-Sq = 34.37% R-Sq(adj) = 31.94%

Appendix 2

Age, Size, and Stable Isotope Data of Mink Populations, and a Predictive Model of Bioaccumulation of Chemicals of Concern in the Rochester Embayment of Lake Ontario

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June 2006

OVERVIEW

This report is the second of four resulting from project C302399 funded by the New York Great Lakes Protection Fund in 2004 to address use impairments related to water quality identified in the Remedial Action Plan for the Rochester Embayment of Lake Ontario (RELO RAP). It deals with ages, sizes and trophic positions (stable isotope analysis) of mink (*Mustela vison*) in the study area, and provides a predictive model for exposure levels of mink to bioaccumulative chemicals of concern (BCCs). The previous report (Wellman and Haynes 2005) addressed the development and use of videocapture (MustelaVision) systems that established the presence and reproduction of mink in and out of the RELO RAP Area of Concern (AOC). Two more reports will be written in 2006: (1) levels of BCCs in mink tissues (Pagano and Haynes, in preparation), and (2) a literature review of the effects of BCCs on mink (Wellman, in preparation). Because mink are the most sensitive known species to BCCs, the results of this four-part project will determine whether delisting the fish and wildlife reproduction impairment for the RELO AOC can be recommended.

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Age, Size, and Stable Isotope Data of Mink Populations, and a Predictive Model of Bioaccumulation of Chemicals of Concern in the Rochester Embayment of Lake Ontario

INTRODUCTION

In the 1980s, the binational (Canada, U.S.) International Joint Commission (IJC) began the process of creating and implementing remedial action plans (RAPs) in 43 contaminated areas of concern (AOCs) 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 AOC, both uses were defined as impaired because "very few" mink were then being trapped or observed within 2 miles of the lake (RAP 1993, 1997). This study was part of a project (Haynes *et al.* 2002) to determine if populations of mink on the shore of the Rochester Embayment of Lake Ontario (RELO) are negatively impacted by bioaccumulative chemicals of concern (BCCs) and, if so, whether the BCCs are originating in the embayment watershed or elsewhere.

The RELO AOC includes the Embayment, a 35 square mile 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 six mile reach of the Genesee River, from the Lower Falls to the mouth at Lake Ontario. The RAP also includes the subwatersheds of Salmon Creek, the Genesee River and Irondequoit Creek (RAP 1993, 1997; **Figure 1**).

The initial questions addressed by this portion of the study were: 1) Can stable isotope analysis be used to evaluate mink diets, at lakeshore and inland areas in and out of the AOC, in terms of trophic levels and terrestrial and aquatic food sources? 2) Can stable isotope results be used to construct a food web/bioaccumulation model for mink in the Rochester AOC to predict body burdens of BCCs in mink in relation to their diets?

Stable isotopes (SIs) of carbon and nitrogen are often used to evaluate trophic webs of ecosystems to give lifetime, integrated estimates of both trophic level and dietary sources for organisms. Both ^{12}C and ^{14}N have stable, heavier isotopes (^{13}C and ^{15}N) which occur naturally, and the heavier and lighter isotopes are differentially absorbed and metabolized by organisms. Usually the lighter isotopes are excreted preferentially, leading to a relative enrichment of the heavier isotopes in organisms relative to their environment or diet. These enrichments are measurable through mass spectrometry, and are reported in parts per thousand (δ‰) relative to a standard:

$$\delta X = [(R_{sample} - R_{standard}) - 1] \times 10^3$$

where X is ^{13}C or ^{15}N and R is the corresponding ratio $^{13}C/^{12}C$ or $^{15}N/^{14}N$. The standard for carbon is PeeDee Belemnite (PDB) limestone, and the standard for nitrogen is atmospheric nitrogen (Fry 1991).

Selective excretion of ^{14}N over ^{15}N by animals results in an increase of approximately 3.4‰ in the $\delta^{15}N$ at each trophic level; thus, ^{15}N analysis can determine the trophic level at which an animal feeds (Peterson and Fry 1987, Cabana and Rasmussen 1994). Carbon is also enriched between trophic levels, but at a much lower rate, between 0 and 1‰. Because freshwater algae have a much less negative $\delta^{13}C$ than terrestrial plants (e.g., terrestrial leaves $\delta^{13}C$ = -27 to -31‰ versus algae > -17‰; Collier and Lyon 1991), ^{13}C analysis can differentiate

between these as original sources of carbon in a diet, indicating whether the diet is primarily of aquatic or terrestrial origin.

Once trophic level and percent aquatic diet are known, the exposure level for each BCC can be calculated using a model adapted from Sample *et al.* (1996). The model takes into account the concentration of the BCC in the water, daily food and water ingestion rates, proportion of the diet originating from aquatic carbon sources, body weight of the animal, and bioaccumulation factors (BAF) for each BCC. The BAF is dependent upon the trophic level and the octanol-water partition coefficient of the compound (Sample *et al.* 1996).

Our approach was to conduct stable isotope analysis for ^{13}C and ^{15}N on tissues from the same mink collected for BCC analyses (Pagano and Haynes, in preparation). We tested the null hypotheses that there are no differences in stable isotope ratios ($\delta^{13}C$ and $\delta^{15}N$) among regions (AOC: In vs. Out, Lakeshore vs. Inland). We used our results for trophic level of prey in mink diets to model the bioaccumulation of selected BCCs in mink in the AOC for later comparison with the results of Pagano's and Haynes' (in preparation) study.

Because it was desirable to know the ages of the mink for the Pagano and Haynes study (in preparation), we had the minks' teeth aged and used those results to answer further questions, such as: How do body length and weight relate to ages of trapped mink? Do the ages of mink trapped vary in and out of the AOC and between lakeshore and inland areas? Do stable isotope values in mink vary with age?

MATERIALS AND METHODS

Specimen collection, processing and handling

Collection

Mink carcasses were collected from trappers (after skinning) in five areas. We divided the study area into four Regions: Inland/AOC, Lakeshore/AOC, Inland/Out of AOC, and Lakeshore/Out of AOC. Both Lakeshore regions were identical to those defined for the MustelaVision videocapture study (Wellman and Haynes 2005) — Lakeshore/AOC (Wetlands) was the Braddock Bay Wildlife Management Area (BBWMA), and Lakeshore/Out of AOC (Mixed) was the Lake Ontario State Parkway west of Route 19 (LOSPW). However, for this study Inland/AOC (Mixed) included any animals taken in the AOC watershed more than 5 km from the lakeshore, and Inland/Out of AOC included animals taken from the Tug Hill Plateau, as well as from the Iroquois National Wildlife Refuge area, to provide two presumably "clean" control areas. Thus, Inland/Out of AOC was the only region that included both Wetlands and Mixed habitats. For the purpose of analysis, we described each collection area using the Regional Descriptors AOC: In versus Out; Lakeshore versus Inland; and Landscape: Wetlands (large wetlands complex) versus Mixed Habitat.

Carcasses were put in plastic bags and frozen by the trappers as soon as possible. The trappers completed log sheets indicating the date and location of capture for each animal, as well as the trapper's name and contact information. Carcasses were assigned specimen numbers in the order in which they were collected, and the specimen number, date and location of capture were written on the plastic bags with a permanent marker.

Processing

We thawed the frozen mink carcasses overnight in a refrigerator before processing them. Because some trappers removed the tails when skinning the carcasses, we removed all other tails before weighing and measuring to obtain comparable measures of body weight and length. We

placed carcasses in hexane-rinsed aluminum pans or aluminum foil for resection, and all utensils used were rinsed with hexane before each use. We placed muscle tissue from the left thigh of each carcass into a hexane-rinsed glass specimen bottle, labeled with the specimen number and tissue type, and froze it. We extracted two canine teeth from each mink and placed them in similarly labeled specimen envelopes. We recorded the body weight, tail-less body length, and weight of each tissue sample (except teeth) on a separate sheet for each mink, along with its specimen number and collection record.

Handling & Shipping

We shipped the muscle tissue samples, frozen and packaged with dry ice, to Cornell University's Stable Isotope Laboratory (COIL).We let the teeth dry in their paper envelopes and shipped them to Matson's Laboratory (Milltown, MT) for aging.

Isotope Analysis

At COIL, stable isotope analyses for ^{13}C and ^{15}N were conducted using a continuous flow Elemental Analyzer (NC2500, CE Elantech, New Jersey) interfaced with an Isotope Ratio Mass Spectrometer (IRMS) (Delta Plus, Thermo Electron Corp., Germany). Strict quality control procedures included standards to: (1) test for instrument linearity and define instrument response for the determination of elemental composition, and (2) measure stability of precision and accuracy over the length of a run (Arthur Kasson, COIL, personal communication).

Aging

Matson's Laboratory aged the mink teeth using a standardized species- and tooth-specific cementum analysis. Mammalian teeth, like trees, show seasonal growth rings when properly stained (Matson 1981). Matson's assumed a birth date of April 1 for all mink, and reported ages in whole years only. We then calculated the additional partial year for each mink between April 1 and its capture date, and added that to Matson's result to obtain the age of each mink.

Data Analysis

We used Microsoft ® Excel 2000 for data management and non-statistical calculations. For statistical analyses, we used Minitab™ Statistical Software Release 14.13 (2005). We conducted regression analyses to evaluate the relationships between age and both body length and weight. We computed descriptive statistics for age versus the Regional Descriptors (AOC: In vs. Out, Lakeshore vs. Inland, and Landscape: Wetland vs. Mixed Habitat) and then used Minitab's General Linear Model (GLM, a 2-way ANOVA with unbalanced cells, Tukey's pair-wise comparisons) to analyze the relationships between age and the Regional Descriptors. We also estimated historical trapping pressure in each area based on our conversations with DEC employees and trappers, and assigned Trapping Pressure values as a covariate in the GLM. Trapping Pressures were: 1 = mink not previously targeted by trappers (Lakeshore/Out of AOC and Inland/AOC), 2 = mink trapped historically (Lakeshore/AOC and Inland/Out of AOC). We then conducted regression analyses for each isotope ($\delta^{13}C$ and δN) versus age, and finally computed descriptive statistics and GLMs for each isotope versus the Regional Descriptors.

We estimated the power of the GLMs using Minitab's 2-Level Factorial power calculator (Factors = 3, Corners = 4, Replicates = minimum number for any level of the factor of concern (Regional Descriptor or Trapping Pressure), Effects = the smallest differences between the means for each factor). Although this calculator is not designed for unbalanced cells, using the minimum number of replicates among treatments produced a conservatively low estimate of the actual power (Minitab support staff 2006, personal communication).

Modeling

Trophic level is calculated by dividing the $\delta^{15}N$ value of an organism by the change in $\delta^{15}N$ per trophic level, usually 3.4‰ (Minigawa and Wada 1984, Vander Zanden and Rasmussen 1999, Doucett 1999). Calculating percent aquatic diet using $\delta^{13}C$ requires 1) determining the $\delta^{13}C$ value in tissue, 2) estimating the difference between the $\delta^{13}C$ values in the tissue and in the diet, and 3) calculating the relative contributions of aquatic and terrestrial sources required to yield the estimated $\delta^{13}C$ of the diet (DeNiro and Epstein 1978). COIL's analysis provided the data for step 1. Literature review provided appropriate estimated values for step 2. The equation for step 3, calculating the proportion of a diet ($\%_A$) originating from one of two dietary sources of carbon with different $\delta^{13}C$ values, is

$$\%_A = \frac{\delta^{13}C_{animal} - \delta^{13}C_B - f \cdot x}{\delta^{13}C_A - \delta^{13}C_B} \times 100,$$

where $\delta^{13}C_{animal}$ is the stable-isotope ratio in the animal, $\delta^{13}C_A$ and $\delta^{13}C_B$ are the stable-isotope ratios of the two carbon sources, *f* is the trophic fractionation between the animal and its diet, and *x* is the trophic position of the animal (adapted from Doucett 1999).

Once the trophic level and aquatic portion of an animal's diet are known, the animal's exposure to a BCC can be modeled knowing the concentration of the compound in ambient water. The equation to predict the daily exposure level of an animal to a BCC in water is

$$Exp = \frac{C_w[W + (F \times P_{aq} \times BAF)]}{bw},$$

where Exp is the exposure from both food and water, C_w is the concentration of the BCC in the water, W and F are the daily water and food consumption rates in L/day and g/day, respectively, P_{aq} is the aquatic proportion of the diet, BAF is the bioaccumulation factor of the chemical of concern (based on the trophic level of the animal and the octanol-water coefficient k_{ow}, a measure of hydrophobicity of the compound), and bw is the body weight of the animal in grams (adapted from Sample *et al.* 1996).

RESULTS AND DISCUSSION

Mink Age, Length and Weight

Although we used 41 mink in this study, 12 were not aged due to damaged or lost teeth. The ages of the mink ranged from 0.60 to 4.75 years; 41% (12/29) of those aged were less than one year old, while only 2 mink (7%) were over 4 years old (Figures 2, 3). Eagle and Whitman (1997) stated that wild mink rarely live longer than 3 years, and cited a study (Adams and Chapman 1981) in which only one of 169 trapped mink had reached the age of four years. Mitchell (1961) reported almost complete turnover of a population in Montana within three years. In contrast, Aulerich *et al.* (1999) reported life spans of 7-11 years in ranch mink.

Neither body length ($r = 0.007$, $P = 0.655$) nor body weight ($r = 0.019$, $P = 0.476$) were correlated with the age of the mink trapped (Appendix A: Figures A1, 2), a result explained by earlier studies. Mitchell (1961) reported that juvenile females attained their adult weight by mid-August, and males sometime during their first winter. Dunstone (1993) reported that mink reach adult body size by about 10 months (their first breeding season) although males may continue to gain weight after the first year (and body weight may cycle with seasons). Aulerich *et al.* (1999) reported that mink reach 95% or more of their adult body length by 16 weeks of age (September).

Trapping season in the study area does not open until late November, except for the Tug Hill Plateau, where it opens in October (our contracted trapper for the Rochester Embayment also started in October with a special collector's permit). Thus, the mink should have been close to their adult weight by their first trapping season, and a correlation between weight and age would not be expected.

Females in our study averaged 32.6 (± 1.0) cm body length and 456.8 (± 42.0) g body weight, while mean male body length was 37.4 (± 2.7) cm and mean body weight 781.5 (± 26.8) g. These means are somewhat smaller than reported by Mitchell (1961) in Montana, where males averaged 1150 g and non-pregnant females 600 g. The average male body length and weight in our study were 15% and 71% greater, respectively, than the female means. Dunstone (1993) reported that males are typically about 75% heavier than females, and Aulerich *et al*. (1999) reported male body weights 68% and 85% higher than females of the same age.

Age vs. Regional Descriptors

None of the Regional Descriptors (or their interactions) had any effect on the age of mink trapped (P-values ranged from 0.304 to 0.404; Table 1, also Appendix B: Table B2). However, in examining the ages of the mink from each area (Figure 3), and the descriptive statistics (Appendix B, Table B1), we noticed that the largest difference between the mean ages occurred for the Landscape descriptor and that none of the mink from the Iroquois National Wildlife Refuge (INWR, Inland/Out of AOC) area had reached one year of age (Appendix A, Figure A2). Also, seven of the eight mink older than three years were trapped in the Inland/AOC and Lakeshore/Out of AOC areas where, to our knowledge, mink had not previously been trapped. Despite the small sample size from INWR (only four of those five mink could be aged), we hypothesized that trapping pressure might have an effect on the ages of the mink trapped, and assigned the Trapping Pressure levels to each area, as described above, to enable further investigation.

The descriptive statistics (Appendix B, Table B1) indicated that Trapping Pressure did have an effect on the ages of mink trapped. In previously non-trapped areas (Lakeshore/Out of AOC and Inland/AOC) the mean age (and standard error) was 2.6 (0.37), the median 3.0, and the maximum 4.8 years. In trapped areas (Lakeshore/AOC, Inland/Out of AOC) the mean age was 1.4 (0.27), the median 0.8, and the maximum 3.6.

Having observed these differences, we used Trapping Pressure as a covariate in the GLM (Appendix B, Table B3), (which forced us to drop the Landscape Descriptor and the AOC X Lakeshore Interaction due to empty cells). The results were that the P-value for Trapping Pressure was 0.017, while the P-values for the Regional Descriptors rose to 0.634 or higher (Table 1). The low power levels (Table 1) calculated for AOC: In vs. Out and Lakeshore vs. Inland are due to the small differences between the means for each factor (0.4 and 0.3 years, respectively), which can be attributed to the length of the trapping season (0.2 years in western NY to 0.5 years on the Tug Hill Plateau and for our contracted trapper with his special permit). In contrast, the difference in ages between historically trapped and non-trapped mink was 1.3 years, significant especially when compared to the short (3-4 yr) life spans of wild mink.

Our conclusion that Trapping Pressure is a biologically significant factor in the ages of the mink trapped is supported by Eagle and Whitman (1997). They reported higher proportions of juveniles in heavily trapped populations than in untrapped populations and hypothesized that reproduction or juvenile survival may be suppressed in untrapped areas that may reach their carrying capacity for mink. Mitchell (1961) also found juvenile to adult female ratios higher in a

commercially trapped area than in a historically non-trapped area. Unfortunately, we have no way of knowing whether the trapped mink in heavily trapped areas truly represented the population structure, or whether older mink are more trap-wary and less likely to be caught and counted. In historically non-trapped areas, trap-wariness should not be a factor and the trapped mink might better represent the population structure.

The presence of mink less than one year old in each area implies reproduction in all areas. It is possible that the young of the year trapped in an area were recently dispersed newcomers, as Gerell (1970) reported one dispersing mink traveling 45 km and another averaging 800 m per day over 27 days. However, the MustelaVision study (Wellman and Haynes 2005) recorded family units in all areas except Lakeshore/Out of AOC, confirming reproduction in the AOC.

Isotopes

Isotope Data vs. Age

Although the P-value of 0.043 indicated a significant correlation between age and $\delta^{15}N$, the R^2 of 0.144 is small enough to conclude that there is no real effect (Appendix C1). For example, Mink #58 (AOC: In/Inland/Mixed) had a high $\delta^{15}N$ (14.85) but was only 0.87 years (10.4 months) old. Minigawa and Wada (1984) found no relationship between age and $\delta^{15}N$ in marine mussels or in tilapia. Age also had no effect on $\delta^{13}C$ ($R^2 = 0.008$; P = 0.641; Appendix C2). Our results were similar to, but the reverse of, Kiriluk *et al.*'s (1995) findings that the correlation between $\delta^{15}N$ and age in lake trout was not significant, but $\delta^{13}C$ and age were weakly correlated ($r^2 = 0.22$, P = 0.0001).

Isotope Data vs. Regional Descriptors

Two Regional Descriptors, AOC: In vs. Out and Lakeshore vs. Inland, had significant effects on $\delta^{15}N$ values of the mink (Table 2; also Appendix D2). Mink in the AOC had higher $\delta^{15}N$ values than mink out of the AOC (P = 0.025), and Lakeshore mink had higher $\delta^{15}N$ values than Inland mink (P = 0.002). Landscape had no effect (P = 0.613, power = 0.711). The highest mean $\delta^{15}N$ of the four regions was found in the AOC (Lakeshore/Wetlands), where the mean $\delta^{15}N$ = 13.2 ‰ (SE = 0.5). The highest individual $\delta^{15}N$ value (16.9 ‰, Mink #17) was also found in the Lakeshore/AOC area, while the lowest was found in the AOC/Inland area (9.2 ‰, Mink #24) (Appendix D1).

None of the Regional Descriptors had significant effect on the $\delta^{13}C$ values of the mink studied (Table 3 and Appendix D2). The highest (most positive) $\delta^{13}C$ value (–28.29 ‰) was found in Mink #17 (Lakeshore/AOC) while the lowest (–19.89 ‰) was Mink #5 (Inland/Out of AOC) (Appendix D2). The low power levels are due to the small differences between the means for each factor (Table 3).

Construction of Bioaccumulation Model

Calculation of Trophic Level

Using the $\delta^{15}N$ value of 11.9 (grand mean of 41 mink in our study) and the commonly accepted value of 3.4‰ $\delta^{15}N$ per trophic level, the average trophic level of mink in our study was 3.50. If we use 3.5‰ $\delta^{15}N$ per trophic level, as reported by Cabana and Rasmussen (1994) for the Lake Ontario food web, the trophic level of our mink averaged 3.40. The higher mean $\delta^{13}N$ of 13.2 for mink in Lakeshore/AOC resulted in higher values for the trophic level of those mink (3.87 or 3.76, using 3.4‰ or 3.5‰ $\delta^{14}N$ per trophic level, respectively). All of these values agree well with estimates found in the literature; USEPA (1995a) reported estimates for mink prey levels ranging from 2.5 to 2.9, which would imply the minks' trophic level to be 3.5 to 3.9.

For the purpose of the model, we chose 3.8 as the trophic level of mink, for several reasons. As the ultimate purpose of the project is to protect mink populations in the AOC, we wanted to represent the mink in the AOC at greatest risk, those along the lakeshore. We chose an intermediate value between those based on the two estimates of the change in δN per trophic level, because, although Cabana and Rasmussen (1994) studied Lake Ontario, they analyzed only the pelagic food web. Therefore, their estimate is not fully appropriate for the diet of mink that feed in the littoral zone of the lake, associated wetlands, or in streams.

The mean $\delta^{15}N$ in mink from the Lakeshore was 1.5‰ higher than the mean from Inland areas. This represents almost one-half of a trophic level difference between Lakeshore and Inland mink. The mean $\delta^{15}N$ for mink in the AOC was 1.1‰ higher than the mean out of the AOC, about one-third of a trophic level. If the Lakeshore minks' diet includes a higher proportion of aquatic-based prey, then inferring a higher trophic level for Lakeshore than Inland mink may be confounded by the fact that aquatic primary producers typically have $\delta^{15}N$ values 1-3‰ higher than terrestrial values (Figure 4). However, the hypothesis that Lakeshore mink feed at a slightly higher trophic level than Inland mink is supported by BCC analysis of the mink tissues (James Pagano, SUNY Oswego, personal communication).

Calculation of Aquatic Portion of Diet

In reference to the three steps involved in calculating the aquatic portion of the diet of an animal using $\delta^{13}C$, the calculated mean $\delta^{13}C$ in mink muscle tissue from the Lakeshore/AOC was –25 (Table 3). For step 2 of the calculation, since the mink tissue used was thigh muscle, we relied on DeNiro and Epstein's (1978) report that the $\delta^{13}C$ of thigh muscle from mice fed two different diets was depleted (more negative) by 1.9 ± 0.5 ‰ from the $\delta^{13}C$ value of the diets. Adding this value (+1.9‰) to the $\delta^{13}C$ value of the mink tissue (–25) yielded a value of –23.1‰ $\delta^{13}C$ for the diet of mink in BBWMA.

DeNiro and Epstein (1978) examined insects, nematodes, snails and mice, and found that $\delta^{13}C$ values varied significantly between tissues of the same animal such that no single tissue truly represents the $\delta^{13}C$ value of the whole animal. They also reported that $\delta^{13}C$ values differ among conspecifics raised on different diets, but that differences between an animal and its diet are similar within a species regardless of diet. Thus, mouse $\delta^{13}C$ values would not be applicable to mink, but since the ^{13}C fractionation is due to metabolic processes that are similar in all mammals, herbivores and carnivores, our estimate for the ^{13}C depletion from diet to thigh muscle should be satisfactory. Focken and Becker (1998) cautioned that the lipid content of tissue in their study had such a strong influence on $\delta^{13}C$ ratios that the trophic shift was not constant among, or even within, species, and that within-species differences were sometimes higher than levels commonly assumed for trophic level shift. However, the mouse data was the best estimate we found.

We had several difficulties with step 3 of the calculation of the aquatic portion of the diet of mink. The formula,

$$\%_A = \frac{\delta^{13}C_{animal} - \delta^{13}C_B - f \cdot x}{\delta^{13}C_A - \delta^{13}C_B} \times 100,$$

works quite well if there are only two dietary sources of carbon with $\delta^{13}C$ values separated by 5‰ to 10‰ (with sample sizes of 50 to 15, respectively; Doucett 1999). So, if the $\delta^{13}C$ values of terrestrial and aquatic carbon sources in our study differed by at least 6‰, we should have had no problem in calculating the aquatic portion of the diet. For example, Balasse *et al.* (2005) were able to use the difference between terrestrial vegetation δC values (mean of –27 ‰) and seaweed

(ranging from –18.5 to –13.1‰) to determine that seaweed made a significant contribution to the diet of coastal sheep in Scotland.

The first problem was that wetlands have more than two sources of ^{13}C, including phytoplankton, C_3 vascular plants (terrestrial, emergent, floating-leaved, submersed), and epiphytic and filamentous algae. The second problem was variation within, and overlap among, $\delta^{13}C$ values in these sources. Figure 4 shows $\delta^{13}C$ and $\delta^{15}N$ values of vegetation and algae in a wetland associated with Lake Superior, along with values for phytoplankton from Lake Superior (Keough *et al.* 1996). Fry (1991) reported that $\delta^{13}C$ values for terrestrial plants range from –35 to –25‰, while algae range from –34 to –18‰. Other researchers (Peterson *et al.* 1985, France 1995, Albuquerque *et al.* 1997, Doucett 1999, Cloern *et al.* 2002, Choi *et al.* 2005) each reported different ranges of values for the sources mentioned above, with many of the ranges overlapping even in the same study. Peterson and Fry (1987) explained that the ^{13}C content of freshwater organisms depends on the source of the dissolved CO_2 in the water and upon the ^{13}C fractionation by those organisms. Heaton (1999) reported that plant $\delta^{13}C$ values were affected up to 2‰ by factors such as growing conditions (e.g., light, temperature, water and nutrient availability, air flow), variation between parts of the same plant, variation between individuals of the same species in the same location (genetic diversity, microhabitat, age), and seasonal or annual variations. DeNiro and Epstein (1978) noted that the $\delta^{13}C$ values of a single plant species can vary 5‰ or more in a growing season, and Kiriluk *et al*. (1995) found that Lake Ontario net plankton $\delta^{13}C$ values ranged from –33.10 in May to –21.92 in September 1992. Cloern *et al.* (2002) concluded that isotope studies to determine primary producers are confounded by overlap of the isotopic ratios of the primary producers and changes in the isotopic composition of plant matter as it degrades. They warned of the danger in applying isotopic data from one ecosystem to another, even in congeneric species. For these reasons, we reluctantly concluded that we could not use $\delta^{13}C$ values of our mink to determine the aquatic portion of their diet without having also analyzed samples of the vegetation (and the minks' known prey species) in the AOC.

Modeling Exposure of Mink in the AOC to BCCs

Although we were unable to calculate the proportion of aquatic foods in the diet of the mink in our study, we found several estimates in the literature. Although most diet studies only report frequencies of occurrence of diet items in scats, digestive tracts, or dens (e.g., USEPA 1993 summarizes the results of 19 such studies), USEPA (1995a) points out that this is not a good representation of biomass assimilated by the mink. However, USEPA (1995b) cited a study by Alexander (1977) reporting that the aquatic portion of minks' diets was 75% to 90%, based on wet weight of stomach contents year-round. Sample and Suter (1999) averaged the results of five studies to conclude that the aquatic portion of minks' diet is 54.6%. (The standard deviation for that average was reported as ± 0.21%, which seems in error, as the average included Alexander's 1977 study; it is much more likely that the standard deviation was 21%). USEPA (1995b) used both 90% and 50% to calculate Wildlife Values for DDT, Hg, 2,3,7,8-TCDD, and PCBs; therefore, we chose the same bounds on the aquatic portion of the diet of mink.

Other values needed for the model are the body weight of the animal (g), daily consumption rates of food (g/day) and water (L/day), the bioaccumulation factor (BAF) of the chemical of concern (which also requires knowing the k_{ow} of the compound), and the concentration of the BCC in the water. The mean body weight of females in our study was 456.8 (± 42.0) g, while males averaged 781.5 (± 26.8) g. Because we had six females and 35 males, we averaged the male and female means for a representative average body weight of 620 g. We then had to correct for the absence of tails and pelts on the mink, since we presumed that the body

weight in the model would have included these. The tails that we removed from mink averaged 1% of the body weight of the mink, and Aulerich *et al*. (1999) gives the weight of a mink skin as about 17% of the body weight. Those body weights from July through pelting would have included skin and tail, so taking the inverse of 0.82 gave us the multiplying factor of 1.22 to get from our tail-less, skinned carcasses to a whole body weight of about 760g.

Several sources give daily food and water consumption rates along with body weights of mink. Sample and Suter (1999) cited Bleavin and Aulerich's (1981) value of 137 g of food per day and estimated daily intakes of 0.099 L of water, using a model by Calder and Brown (1983), for mink averaging 970 g body weight. USEPA (1995b) estimated intakes of 177 g of food (using an allometric model by Nagy 1987) and 0.081 L water per day (using Calder and Brown's 1983 model) for mink with a body weight of 800 g. For captive adult males averaging 2200 g, Aulerich *et al*. (1999) reported that they drank 0.127 L/day and daily food consumption ranged from 147 g to 275 g, depending upon the caloric content of the food and the season. Since our largest mink weighed only 1111 g, and we wanted to make our model conservative (protective of the AOC mink) but not unrealistic, we discounted Aulerich's consumption rates as too high, and chose the larger of the remaining two values for daily food and water intakes. Thus, for our model, the daily food and water consumption rates were 177 g and 0.1 L (= 100g), respectively.

To demonstrate the model, we used several BCCs for which we could find literature values for the concentrations in Lake Ontario. We chose 2,3,7,8-TCDD (dioxin), with a relatively high k_{ow} of 6.53, and lindane, with a moderately low k_{ow} of 3.73, reasoning that compounds with k_{ow}s lower than lindane would have low potential for biomagnification. We also modeled benzo(a)pyrene, dieldrin, and mercury, for which Booty *et al*. (2005) reported k_{ow} values and Lake Ontario water concentrations; they also modeled the Lake Ontario concentration of TCDD. The concentration of lindane in Lake Ontario was 0.24 ng/L in 1992 (Williams *et al*. 2001, cited by Marvin *et al*. 2004). The k_{ow}s and BAFs were taken from Sample *et al*. (1996), who assumed that all fish consumed by mink are trophic level 3 (small fish). However, Melquist *et al*. 1981 reported mink feeding on kokanee (land-locked *Oncorhyncus nerka*) after their spawning, and we have reason to believe that mink feed on piscivorous salmonids in the AOC when they are available (Haynes and Pagano, in preparation). Still, the average trophic level of 3.8 for mink in BBWMA indicates that if salmonids (trophic level 4) do contribute a significant portion of the minks' diet, they are balanced by a comparable portion of level 2 aquatic prey. Thus, we used the BAF factors provided by Sample *et al*. (1996) for prey of trophic level 3, which is slightly higher (and thus more protective than) the prey from trophic level of 2.8 implied by our results.

When our results are incorporated into the equation for exposure, the equation becomes

$$Exp = \frac{C_w[100\,g + (177\,g \times P_{aq} \times BAF)]}{760\,g}.$$

Given C_w (the concentration of the BCC in the water), P_{aq} (the aquatic proportion of the diet), and BAF (the bioaccumulation factor of the chemical of concern at trophic level 3), the results of this model are the levels of BCCs to which mink in the AOC would be exposed daily. For example, if the concentration of dieldrin in water is 1.55E-9 ng/L, and a mink's diet consists of 50% aquatic prey, the mink will be exposed to 1.02E-5 ng/g body weight per day. A 760 g mink would thus be exposed to 7.8E-3 ng, or 7.8 picograms, of dieldrin per day. In contrast, if the dieldrin concentration in water is 3.4E-09 ng/L and the mink's diet is 90% aquatic, the same mink would ingest 3.1E-2 ng, or 31 picograms, of dieldrin per day (Table 4).

Confirmation of the model awaits the results of the Pagano's and Haynes' (in preparation) analyses of BCC concentrations in mink, and will require knowing BCC concentrations in Lake Ontario water. If the sample results (Pagano and Haynes, in preparation) and model results are comparable, the model can be expanded to apply to any BCC if the concentration in the ambient water is known, along with the k_{ow} and BAF of the compound (many of which are in Sample *et al*. 1996). Comparing the results of a validated model to NOAELs (No Observed Adverse Effect Levels) or LOAELs (Lowest Observed Adverse Effect Levels) for specific compounds in mink (Wellman, in preparation) is a preferable method to assess the risk to mink exposed to Lake Ontario or other waters in the AOC because it will require only measuring BCC concentrations in water, not sacrificing mink.

SUMMARY

The first question addressed by this study was: Can stable isotope analysis be used to evaluate mink diets, at lakeshore and inland areas in and out of the AOC, in terms of trophic levels and terrestrial and aquatic food sources? Analysis of $\delta^{15}N$ allowed us to determine that mink in the study area feed on prey at trophic level 2.5 (slightly higher along the lakeshore and in the AOC than elsewhere), with the highest level (2.8) in the Lakeshore/AOC area. We were unable to use $\delta^{13}C$ values to determine % aquatic diet because we had no $\delta^{13}C$ values for carbon sources in the AOC wetlands.

The second question addressed by this study was: Can stable isotope results be used to construct a food web/bioaccumulation model for mink in the Rochester AOC that can predict body burdens of BCCs in mink in relation to their diets? Using our trophic level calculation and literature values of 50% and 90% aquatic diet, we were able to create a food web bioaccumulation model to predict the exposure of mink in the AOC to BCCs, once the BCCs' concentrations in ambient water such as Lake Ontario or Braddock Bay are known. Validation of the model awaits the results of the analyses of mink tissues for BCCs (Pagano and Haynes, in preparation).

In addition, we found that the ages of mink trapped had no effect on either body weight or body length, as they were all near or at their adult size when they were trapped. The Regional Descriptors (AOC: In vs. Out, Lakeshore vs. Inland, Wetland vs. Mixed habitat) also had no effect on the ages of mink trapped. Mean ages of trapped mink were lower in historically trapped areas. Mink less than one year old were trapped in all areas, implying reproduction in all areas including the AOC. However, indications of reproduction of mink in the AOC are not sufficient to justify delisting the wildlife reproduction impairment for the Rochester Embayment. That determination will require the comparison of BCCs in mink tissues (Pagano and Haynes, in preparation) to the NOAELs and LOAELs of the BCCs in question (Wellman, in preparation).

ACKNOWLEDGMENTS

We are very grateful to Randy Baase who for two winters spent many hours trapping mink in the worst weather, and who taught us much about mink. Thanks are also due to other trappers who contributed carcasses and taught us about mink: Matt Lochner, Dan Carroll, Dan Frey, Michael Ingham, Don Newcombe, Tom Raduns, William Schwartz, Jr., Fred Sinclair, and Chuck Tirrano; and to the landowners near the Bergen Swamp who allowed us to collect mink on their property: "Doc" Fink, Dick Sands, Mel Reber and Al Burkhart. We are grateful to Scott

Wells and Ross Abbett for dissecting the mink, and to Albert Fulton for creating Figure 1. We thank Art Kasson at COIL for doing the isotope analyses, and Matson's Lab for aging the mink teeth. Bob Gilliam of the InterLibrary Loan office of Drake Library was very helpful in acquiring requested literature. We are very grateful for Dr. Richard Aulerich's generous donation of a copy of the Handbook of Biological Data for Mink. The government personnel who graciously allowed us to work in their territories include NYS DEC: Dan Carroll, Dave Woodruff and Heidi Bogner Kennedy; NYS OPRHP: James Slusarczyk, and USFWS: Bob Lamoy and Paul Hess. Finally, we gratefully acknowledge grant C302399 from the New York State Great Lakes Protection Fund that made this study possible.

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TABLES

Table 1. Table of Age versus Regional Descriptors. The General Linear Model (GLM) was run with and without Trapping Pressure (TP) as a covariate. With TP as a covariate, the Landscape Regional Descriptor was dropped due to empty cells. Significant effects are in bold. The power estimates are conservative, since Minitab's power calculator is not built to deal with GLMs (see Methods). *Power is not relevant since effect is significant (Chittenden 2002).

Regional Descriptor	**N**	**Mean Age (SE)**	**P-value w/o TP**	**P-value w/ TP**	**Power**
AOC: In vs. Out	29		0.404	0.660	0.171
AOC: In	15	2.2 (0.37)			
AOC: Out	14	1.8 (0.36)			
Lakeshore vs. Inland			0.386	0.634	0.074
Inland	17	1.9 (0.37)			
Lakeshore	12	2.2 (0.35)			
Landscape: Wetlands vs. Mixed			0.304	N/A	0.806
Mixed Habitat	19	2.4 (0.33)			
Wetlands Complex	10	1.2 (0.27)			
Trapping Pressure			N/A	**0.017**	*
None Previous	15	2.6 (0.37)			
Historically Trapped	14	1.4 (0.26)			

Table 2. Table of $\delta^{15}N$ versus Regional Descriptors showing selected descriptive statistics, and the results of the GLM with estimated power. Significant effects are in bold. The power estimates are conservative, since Minitab's power calculator is not built to deal with GLMs (see Methods). *Power is not relevant since the effect is significant (Chittenden 2002).

Area	**N**	**Mean $\delta^{15}N$ (SE) (‰)**	**Min δN (‰)**	**Max δN (‰)**
BBWMA	10	13.2 (0.54)	11.09	16.88
AOC : In/Inland	11	11.8 (0.48)	9.20	14.55
INWR	5	11.2 (0.18)	10.49	11.56
LOSPW	10	12.2 (0.42)	10.45	14.28
TUGHL	5	9.8 (0.12)	9.40	10.14
Entire Study	41	11.9 (0.23)	9.20	16.88
Regional Descriptor	**N**	**Mean $\delta^{15}N$ (SE) (‰)**	**P-value**	**Power**
AOC: In vs. Out	41		**0.025**	*
AOC: In	21	12.4 (0.4)		
AOC: Out	20	11.3 (0.3)		
Lakeshore vs. Inland			**0.002**	*
Inland	21	11.2 (0.3)		
Lakeshore	20	12.7 (0.3)		
Landscape: Wetlands vs. Mixed			0.613	0.711
Mixed Habitat	26	11.6 (0.3)		
Wetlands Complex	15	12.5 (0.4)		

Table 3. Table of $\delta^{13}C$ versus Regional Descriptors showing selected descriptive statistics, and the results of the GLM with estimated power. The power estimates are conservative, since Minitab's power calculator is not built to deal with GLMs (see Methods).

Area	**N**	**Mean $\delta^{13}C$ (SE) (‰)**	**Min δC (‰)**	**Max δC (‰)**
BBWMA	10	–25.0 (0.7)	–28.10	–19.98
AOC : In/Inland	11	–25.3 (0.2)	–26.56	–24.36
INWR	5	–25.7 (1.0)	–28.29	–23.14
LOSPW	10	–25.3 (0.4)	–27.03	–23.15
TUGHL	5	–26.2 (0.3)	–26.95	–25.31
Entire Study	41	–25.4 (1.5)	–28.29	–19.98
Regional Descriptor	**N**	**Mean $\delta^{13}C$ (SE) (‰)**	**P-value**	**Power**
AOC: In vs. Out	41		0.333	0.292
AOC: In	21	–25.1 (0.3)		
AOC: Out	20	–25.6 (0.3)		
Lakeshore vs. Inland			0.314	0.231
Inland	21	–25.6 (0.3)		
Lakeshore	20	–25.1 (0.4)		
Landscape: Wetlands vs. Mixed			0.963	0.091
Mixed Habitat	26	–25.5 (0.2)		
Wetlands Complex	15	–25.2 (0.6)		

Table 4. Predicted daily exposure levels of mink in the AOC, based on literature values for BCC concentrations in Lake Ontario. Observed values indicated by (o); estimated values by (e). BAF values are from Sample *et al*. (1996). Constants used: water intake = 0.1 L/day, food intake = 177 g/day, body weight of mink = 760 g. The concentration of 2,3,7,8-TCDD was estimated by Booty *et al*. (2005), as the compound was not detectable in their study. The concentrations for dieldrin and mercury are the minimum and maximum values observed by Booty *et al*. (2005), while the concentration for B(a)P is the maximum they observed (minimum was zero). The concentration of lindane was reported by Williams *et al*. (2001), cited by Marvin *et al*. (2004).

				Daily Exposure (ng/g bw)	
Compound	**K(ow)**	**BAF: Prey Trophic Level 3**	**Concentration Cw (ng/L)**	**Diet 50% Aquatic**	**Diet 90% Aquatic**
2,3,7,8-TCDD (e)	6.53	172100	1.8E-7	3.61E-03	6.49E-03
Benzo(a)pyrene (o)	6.11	293831	1.48E-09	5.06E-05	9.12E-05
Dieldrin (o)	5.37	56523	1.55E-09	1.02E-05	1.84E-05
		56523	3.40E-09	2.24E-05	4.03E-05
Lindane (o)	3.73	454	2.4E-10	1.27E-08	2.28E-08
Mercury (o)	N/A	27900	2.60E-09	8.45E-06	1.52E-05
		27900	2.50E-08	8.12E-05	1.46E-04

FIGURES

Figure 1. Map showing the four regions referred to in the study. AOC/Lakeshore is Braddock Bay WMA, AOC/Inland is at least 3 km from Lake Ontario, Out of AOC/Lakeshore is the Lake Ontario State Parkway west of Rte.19, and Out of AOC/Inland is Iroquois NWR and the Tug Hill Plateau (not shown). RELO is the Rochester Embayment of Lake Ontario. (Map by Albert Fulton 2005.)

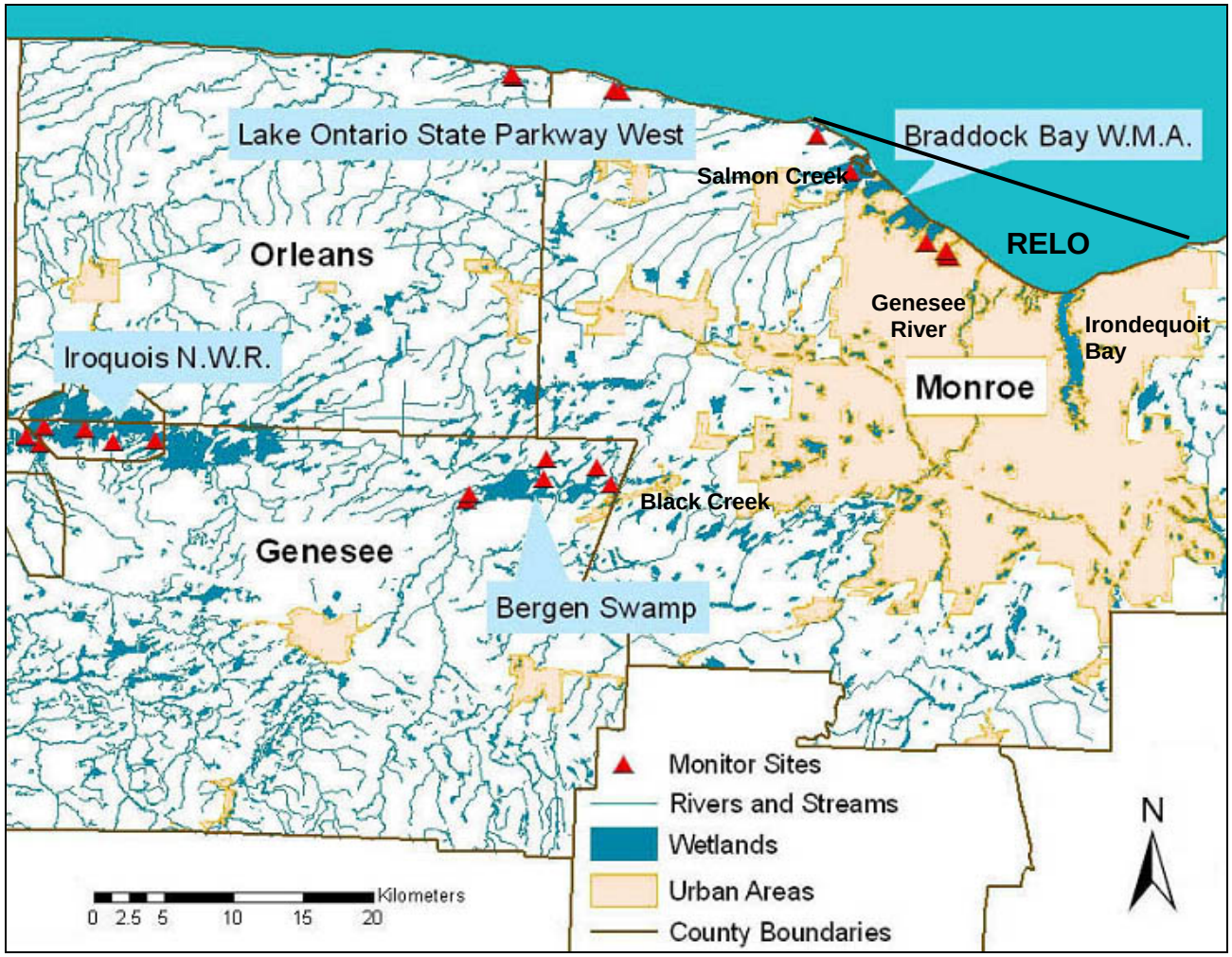


Figure 2. Ages of mink trapped.

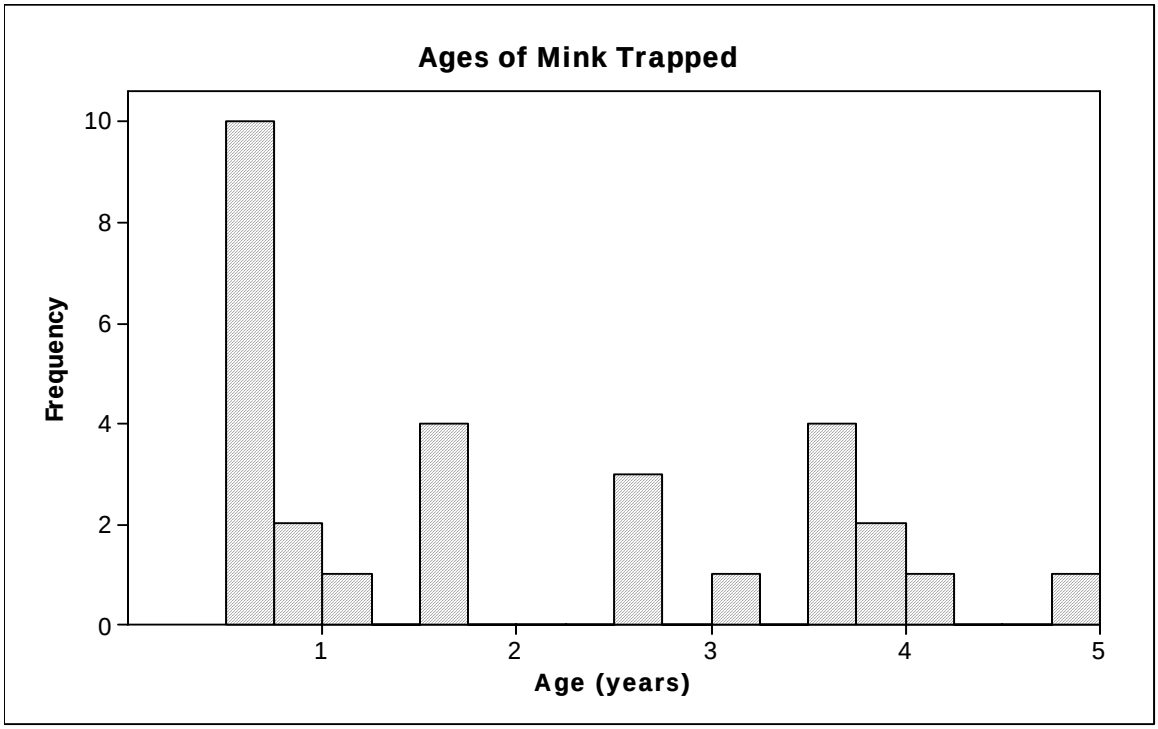


Figure 3. Plot of individual mink ages in each region.

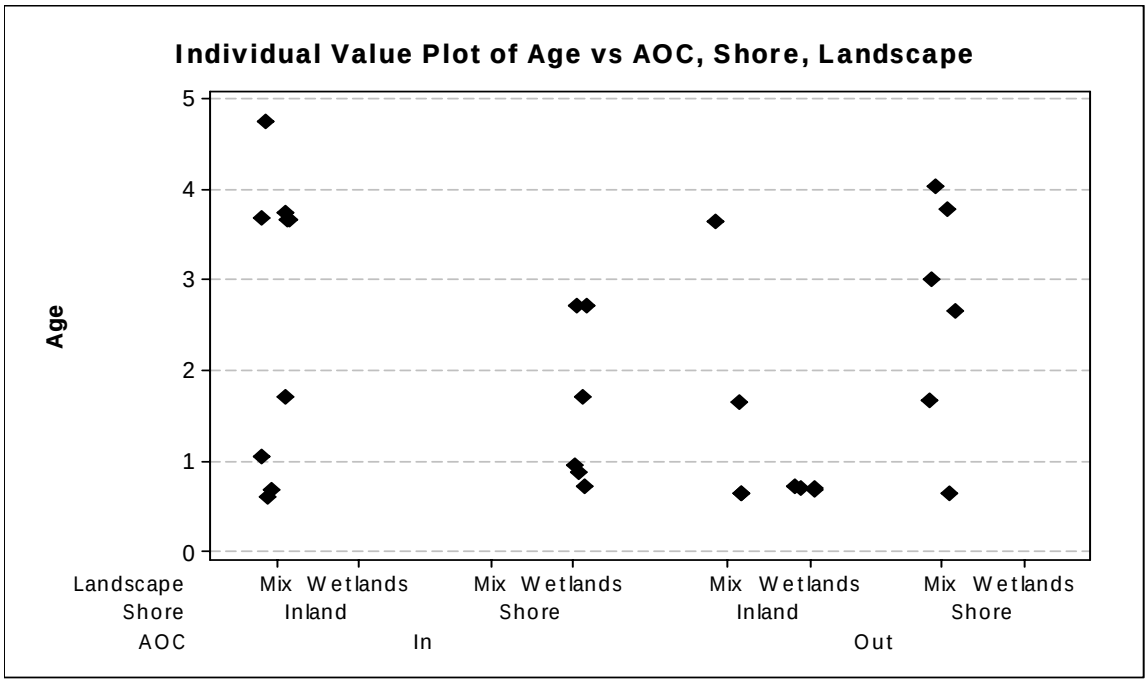
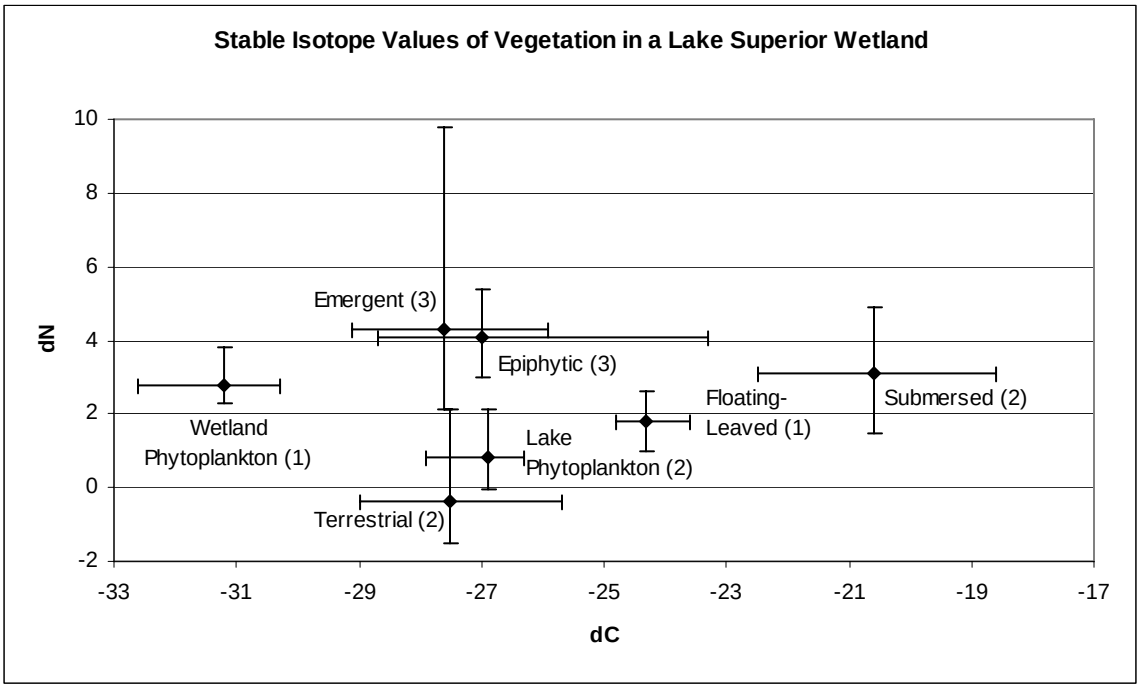


Figure 4. Stable isotope values of carbon sources in a Lake Superior wetland. (Constructed using values from Keough *et al.* 1996; numbers in parentheses are the number of species or types in each category).



APPENDICES

Appendix A: Mink age, length and weight relationships.

Figure A1: Body length versus age of mink, showing no correlation ($R^2 = 0.007$, $P = 0.655$). Tails removed for consistency (see Methods).

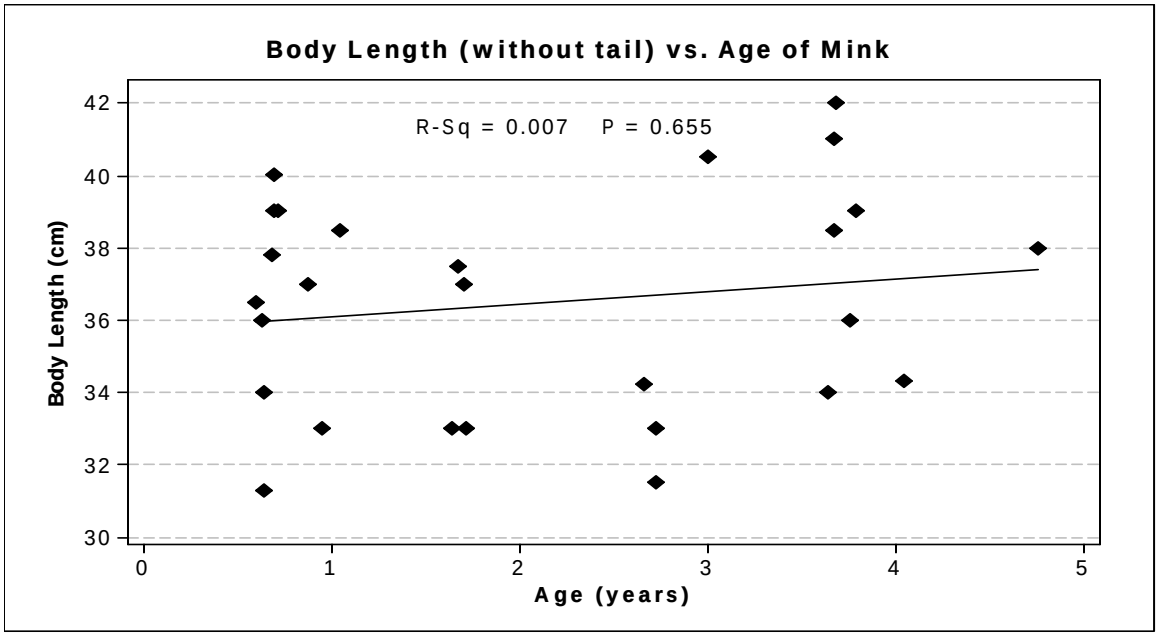
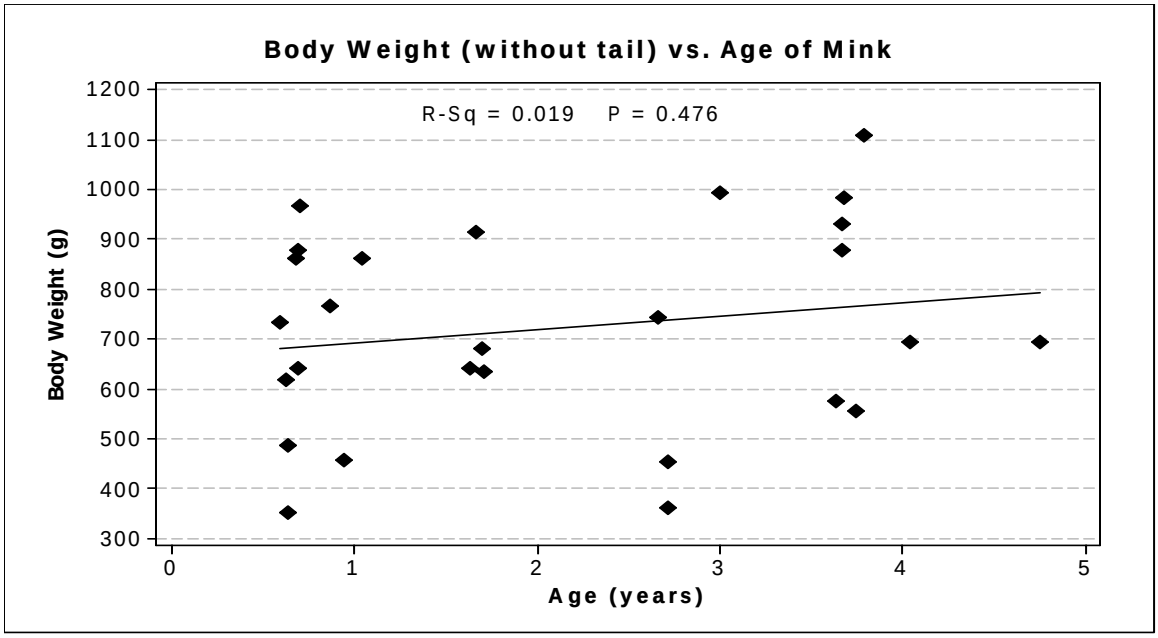


Figure A2. Body weight versus age of mink, showing no correlation ($R^2 = 0.019$, $P = 0.476$). Tails removed for consistency (see Methods).



Appendix B: Age versus Regional Descriptors

Table B1: Descriptive statistics for Age versus Regional Descriptors

Descriptive Statistics: AOC: In vs. Out, One-way ANOVA P = 0.442

Variable	AOC	N	N*	Mean	SE Mean	StDev	Minimum	Q1	Median
Age	In	15	6	2.214	0.367	1.420	0.600	0.870	1.710
	Out	14	6	1.794	0.360	1.346	0.630	0.670	1.175

Variable	AOC	Q3	Maximum
Age	In	3.670	4.750
	Out	3.160	4.040

Descriptive Statistics: Inland vs. Lakeshore, One-way ANOVA P = 0.722

Variable	Shore	N	N*	Mean	SE Mean	StDev	Minimum	Q1	Median
Age	Inland	17	4	1.933	0.369	1.522	0.600	0.675	1.040
	Shore	12	8	2.123	0.346	1.197	0.630	0.890	2.185

Variable	Shore	Q3	Maximum
Age	Inland	3.670	4.750
	Shore	2.930	4.040

Descriptive Statistics: Landscape: Wetlands vs. Mix, One-way ANOVA P = 0.027

Variable	Landscape	N	N*	Mean	SE Mean	StDev	Minimum	Q1
Age	Mix	19	7	2.415	0.332	1.449	0.600	0.670
	Wetlands	10	5	1.245	0.265	0.837	0.680	0.690

Variable	Landscape	Median	Q3	Maximum
Age	Mix	2.660	3.680	4.750
	Wetlands	0.790	1.963	2.720

Descriptive Statistics: Trapping History, One-way ANOVA P = 0.018

Variable	Trap Pressure	N	N*	Mean	SE Mean	StDev	Minimum	Q1
Age	0	15	6	2.621	0.370	1.433	0.600	1.040
	1	14	6	1.358	0.265	0.990	0.640	0.688

Variable	Trap Pressure	Median	Q3	Maximum
Age	0	3.000	3.750	4.750
	1	0.790	1.963	3.640

Table B2: General Linear Model: Age versus AOC, Shore, Landscape (without Trapping Pressure as a covariate)

Factor	Type	Levels	Values
AOC	fixed	2	AOC: In, AOC: Out
Lakeshore	fixed	2	Inland, Lakeshore
Landscape	fixed	2	Mix, Wetlands

Analysis of Variance for Age, using adjusted SS for Tests

Source	DF	Seq SS	Adj SS	Adj MS	F	P
AOC	1	1.276	1.176	1.176	0.72	0.404
Lakeshore	1	0.287	1.267	1.267	0.78	0.386
AOC*Lakeshore	1	10.684	0.645	0.645	0.40	0.535
Landscape	1	1.796	1.796	1.796	1.10	0.304
Error	24	39.029	39.029	1.626		
Total	28	53.071				

S = 1.27522 R-Sq = 26.46% R-Sq(adj) = 14.20%

Figure B2a: Main effects plot.

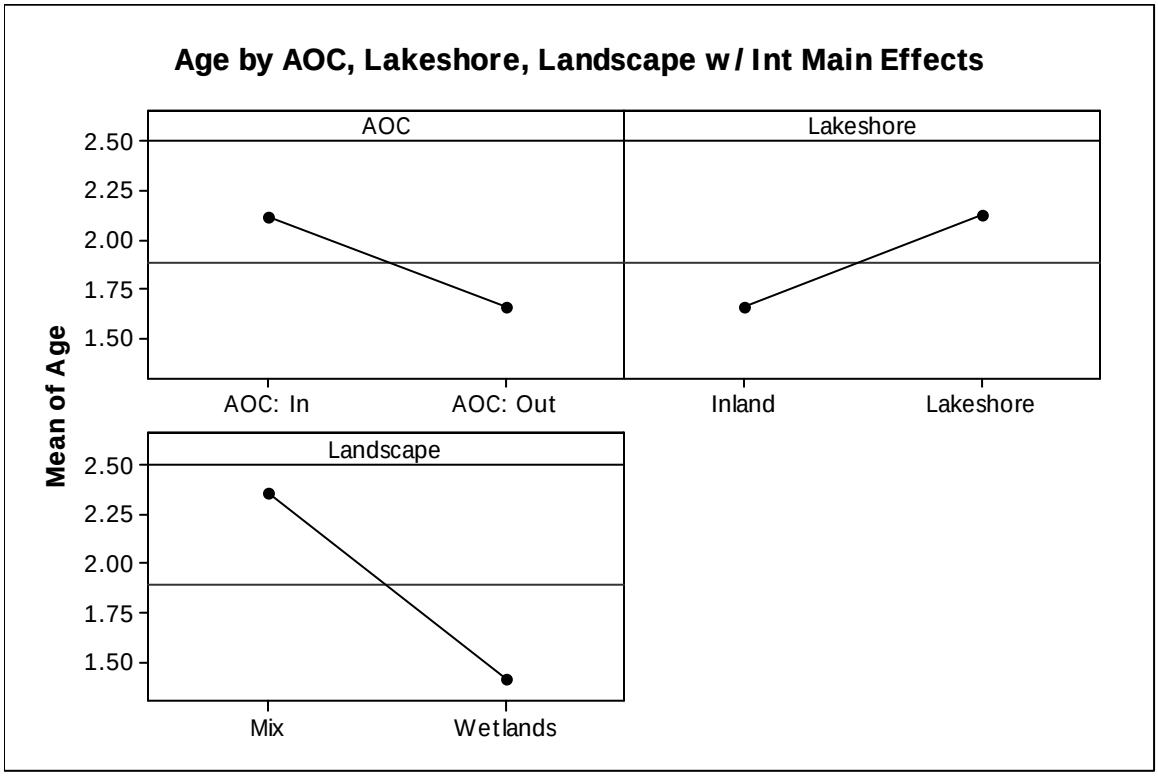


Figure B2b: Interactions plot.

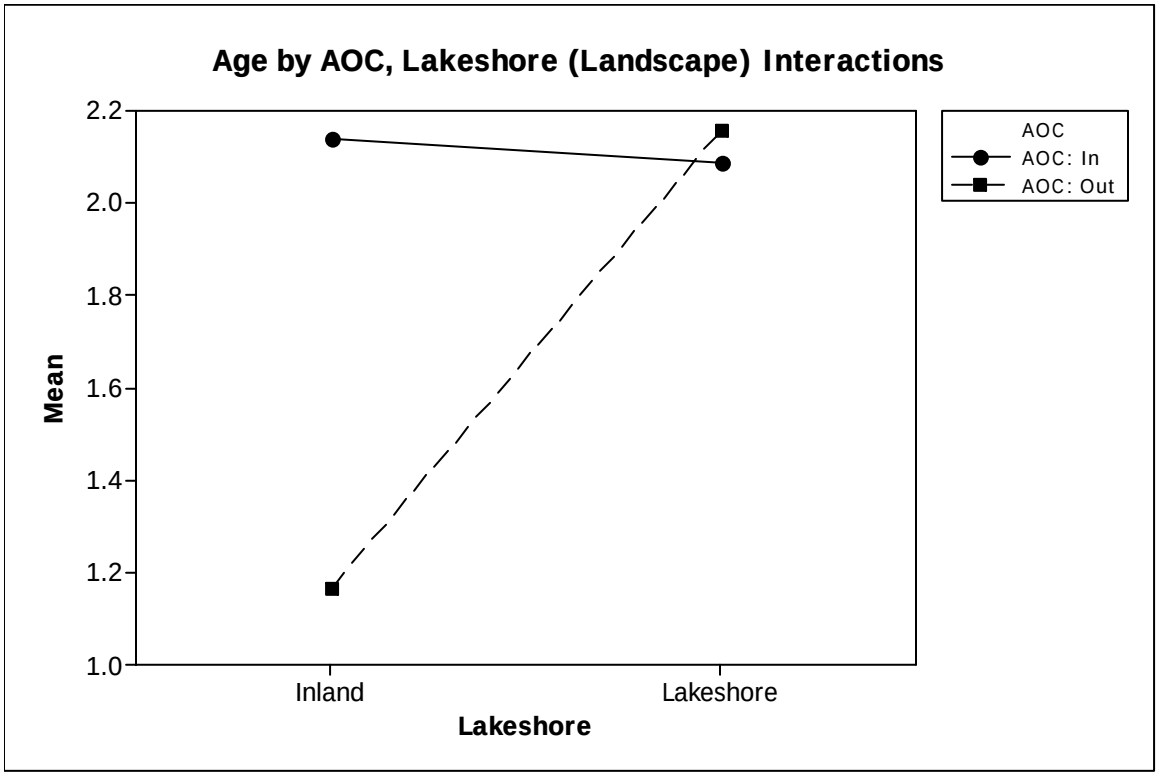


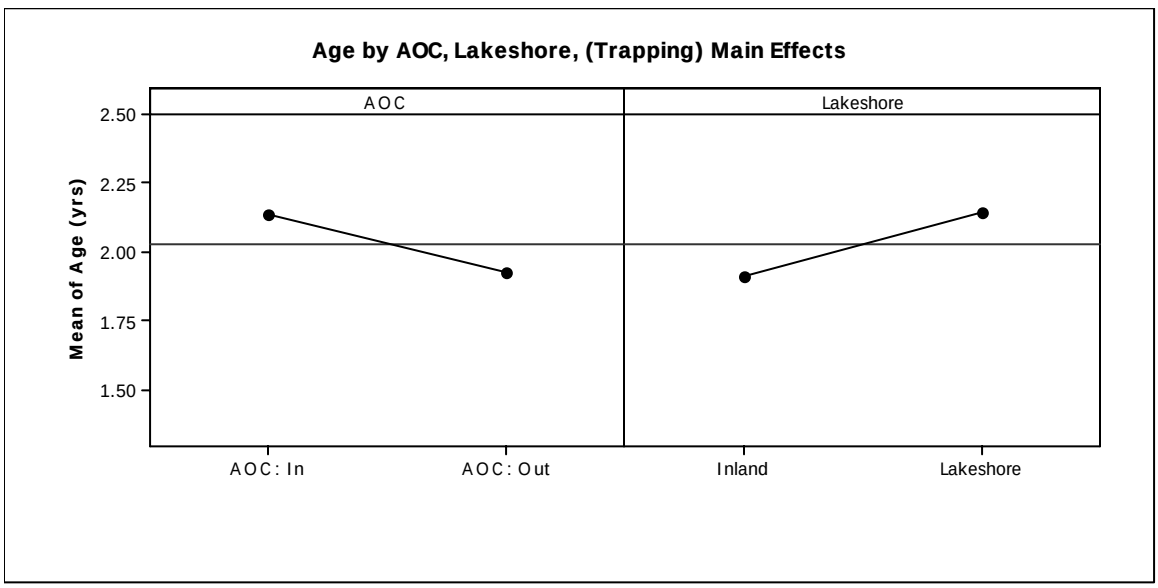
Table B3: General Linear Model: Age versus AOC, Lakeshore with Trapping Pressure as covariate.

```
Factor     Type   Levels  Values
AOC        fixed       2  AOC: In, AOC: Out
Lakeshore  fixed       2  Inland, Lakeshore
```

Analysis of Variance for Age, using Adjusted SS for Tests

```
Source     DF  Seq SS  Adj SS  Adj MS     F      P
Trapping    1  11.560  10.684  10.684  6.54  0.017
AOC         1   0.308   0.324   0.324  0.20  0.660
Lakeshore   1   0.379   0.379   0.379  0.23  0.634
Error      25  40.824  40.824   1.633
Total      28  53.071

S = 1.27788   R-Sq = 23.08%   R-Sq(adj) = 13.84%

Term         Coef  SE Coef      T      P
Constant   3.8563   0.7568   5.10  0.000
Trapping  -1.2333   0.4822  -2.56  0.017

Unusual Observations for Age

Obs      Age      Fit   SE Fit  Residual  St Resid
 15  3.64000  1.16625  0.45180   2.47375      2.07 R

R denotes an observation with a large standardized residual.
```

Figure B3a: Main effects plot.



Appendix C: Isotope data versus Age.

Appendix C1: $\delta^{15}N$ versus Age.

Figure C1. Scatterplot of $\delta^{15}N$ versus Age showing very weak correlation ($R^2 = 0.144$, $P = 0.043$).

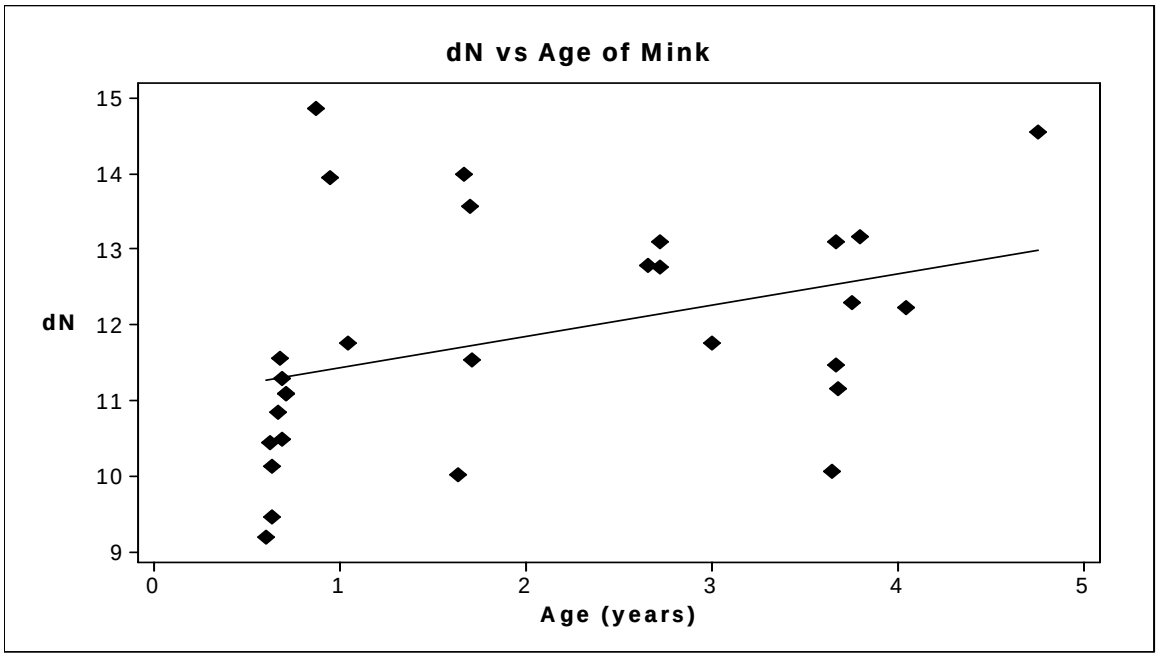


Table C1. Regression analysis: $\delta^{15}N$ versus Age (29 mink)

```
The regression equation is
δ15N = 11.0 + 0.417 Age

29 cases used, 12 cases contain missing values

Predictor      Coef  SE Coef      T      P
Constant    11.0110   0.4752  23.17  0.000
Age          0.4173   0.1960   2.13  0.043

S = 1.42811   R-Sq = 14.4%   R-Sq(adj) = 11.2%
```

Analysis of Variance

```
Source          DF      SS     MS     F      P
Regression       1   9.241  9.241  4.53  0.043
Residual Error  27  55.066  2.039
Total           28  64.307

Unusual Observations
Obs   Age      δN     Fit  SE Fit  Residual  St Resid
 36  0.87  14.850  11.374   0.347     3.476     2.51R
```

Mink #58, Bergen

```
R denotes an observation with a large standardized residual.
```

Appendix C2: $\delta^{13}C$ versus Age

Figure C2. Scatterplot of $\delta^{13}C$ versus Age showing no correlation ($R^2 = 0.008$, $P = 0.641$).

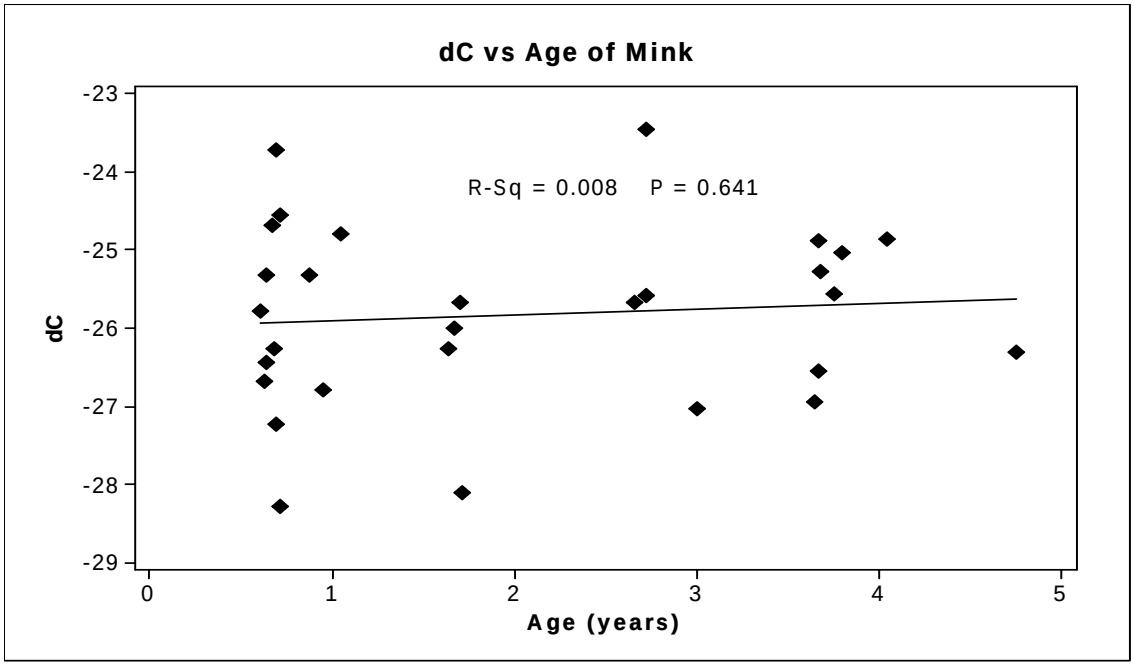


Table C2. Regression Analysis: $\delta^{13}C$ versus Age (29 mink)

The regression equation is
$\delta^{13}C = - 26.0 + 0.075$ Age

29 cases used, 12 cases contain missing values

Predictor	Coef	SE Coef	T	P
Constant	-25.9799	0.3862	-67.27	0.000
Age	0.0752	0.1593	0.47	0.641

S = 1.16071 R-Sq = 0.8% R-Sq(adj) = 0.0%

Analysis of Variance

Source	DF	SS	MS	F	P
Regression	1	0.300	0.300	0.22	0.641
Residual Error	27	36.376	1.347		
Total	28	36.676			

Unusual Observations

Obs	Age	dC	Fit	SE Fit	Residual	St Resid
3	0.71	-28.290	-25.927	0.299	-2.363	-2.11R
9	2.72	-23.450	-25.775	0.243	2.325	2.05R

R denotes an observation with a large standardized residual.

Appendix D: Isotope Data versus Regional Descriptors

Appendix D1: $\delta^{15}N$ vs. Regional Descriptors

Table D1a. Descriptive statistics: $\delta^{15}N$ vs. Regional Factors

Descriptive Statistics: AOC: In vs. Out, One-way ANOVA P = 0.035

Variable	AOC	N	N*	Mean	SE Mean	StDev	Minimum	Q1	Median	Q3
$\delta^{15}N$	In	21	0	12.429	0.382	1.753	9.200	11.305	12.420	13.325
	Out	20	0	11.349	0.310	1.385	9.400	10.218	11.310	12.110

Variable	AOC	Maximum
$\delta^{15}N$	In	16.880
	Out	14.280

Descriptive Statistics: Inland vs. Lakeshore, One-way ANOVA P = 0.002

Variable	Shore	N	N*	Mean	SE Mean	StDev	Minimum	Q1	Median
$\delta^{15}N$	Inland	21	0	11.158	0.307	1.409	9.200	10.045	11.150
	Shore	20	0	12.684	0.349	1.561	10.450	11.563	12.490

Variable	Shore	Q3	Maximum
$\delta^{15}N$	Inland	11.835	14.550
	Shore	13.755	16.880

Descriptive Statistics: Landscape: Wetlands vs. Mix, One-way ANOVA P = 0.085

Variable	Landscape	N	N*	Mean	SE Mean	StDev	Minimum	Q1	Median
$\delta^{15}N$	Mix	26	0	11.563	0.308	1.573	9.200	10.120	11.545
	Wetlands	15	0	12.489	0.436	1.688	10.490	11.280	12.420

Variable	Landscape	Q3	Maximum
$\delta^{15}N$	Mix	12.858	14.550
	Wetlands	13.090	16.880

Descriptive Statistics: BBWMA (AOC: In, Lakeshore, Wetlands)

Variable	Landscape	N	N*	Mean	SE Mean	StDev	Minimum	Q1	Median
$\delta^{15}N$	Wetlands	10	0	13.157	0.536	1.694	11.090	12.200	12.650

Variable	Landscape	Q3	Maximum
$\delta^{15}N$	Wetlands	14.175	16.880

Table D1b. General Linear Model: $\delta^{15}N$ versus AOC, Shore, Landscape

Factor	Type	Levels	Values
AOC	fixed	2	In, Out
Shore	fixed	2	Inland, Shore
Landscape	fixed	2	Mix, Wetlands

Analysis of Variance for δ15N, using Adjusted SS for Tests

Source	DF	Seq SS	Adj SS	Adj MS	F	P	
AOC	1	11.950	10.764	10.764	5.48	**0.025**	Significant effect.
Shore	1	24.692	20.780	20.780	10.57	**0.002**	Significant effect.
Landscape	1	0.510	0.510	0.510	0.26	0.613	No effect.
Error	37	72.714	72.714	1.965			
Total	40	109.866					

S = 1.40187 R-Sq = 33.82% R-Sq(adj) = 28.45%

Unusual Observations for dN

Obs	dN	Fit	SE Fit	Residual	St Resid		
7	16.8800	13.3379	0.4258	3.5421	2.65	R	(Mink #17, BBWMA)
39	14.5500	11.6028	0.4075	2.9472	2.20	R	(Mink #61, AOC: In/Inland)

R denotes an observation with a large standardized residual.

Appendix D2: $\delta^{13}C$ vs. Regional Factors

Table D2a. Descriptive statistics: $\delta^{13}C$ vs. Regional Factors

Descriptive Statistics: AOC: In vs. Out, One-way ANOVA P = 0.330

Variable	AOC	N	N*	Mean	SE Mean	StDev	Minimum	Q1	Median
$\delta^{13}C$	In	21	0	-25.155	0.344	1.578	-28.100	-25.990	-25.320
	Out	20	0	-25.617	0.315	1.409	-28.290	-26.615	-25.810

Variable	AOC	Q3	Maximum
$\delta^{13}C$	In	-24.555	-19.890
	Out	-24.903	-23.140

Descriptive Statistics: Inland vs. Lakeshore, One-way ANOVA P = 0.304

Variable	Shore	N	N*	Mean	SE Mean	StDev	Minimum	Q1
$\delta^{13}C$	Inland	21	0	-25.618	0.264	1.211	-28.290	-26.375
	Shore	20	0	-25.131	0.391	1.747	-28.100	-26.143

Variable	Shore	Median	Q3	Maximum
$\delta^{13}C$	Inland	-25.670	-24.740	-23.140
	Shore	-25.430	-24.455	-19.890

Descriptive Statistics: Landscape: Wetlands vs. Mix, One-way ANOVA P = 0.635

Variable	Landscape	N	N*	Mean	SE Mean	StDev	Minimum	Q1
$\delta^{13}C$	Mix	26	0	-25.466	0.190	0.971	-27.030	-26.278
	Wetlands	15	0	-25.231	0.559	2.166	-28.290	-26.780

Variable	Landscape	Median	Q3	Maximum
$\delta^{13}C$	Mix	-25.610	-24.843	-23.150
	Wetlands	-25.540	-23.710	-19.890

Descriptive Statistics: In AOC, Lakeshore (BBWMA)

Variable	Landscape	N	N*	Mean	SE Mean	StDev	Minimum	Q1
$\delta^{13}C$	Wetlands	10	0	-24.984	0.699	2.211	-28.100	-26.338

Variable	Landscape	Median	Q3	Maximum
$\delta^{13}C$	Wetlands	-25.430	-24.178	-19.890

Table D2a. General Linear Model: $\delta^{13}C$ versus AOC, Shore, Landscape

Factor	Type	Levels	Values
AOC	fixed	2	In, Out
Shore	fixed	2	Inland, Shore
Landscape	fixed	2	Mix, Wetlands

Analysis of Variance for δ13C, using Adjusted SS for Tests

Source	DF	Seq SS	Adj SS	Adj MS	F	P
AOC	1	2.189	2.208	2.208	0.96	0.333
Shore	1	2.538	2.393	2.393	1.04	0.314
Landscape	1	0.005	0.005	0.005	0.00	0.963
Error	37	85.013	85.013	2.298		
Total	40	89.744				

S = 1.51580 R-Sq = 5.27% R-Sq(adj) = 0.00%

Unusual Observations for dC

Obs	$\delta^{13}C$	Fit	SE Fit	Residual	St Resid		
7	-19.8900	-24.9033	0.4604	5.0133	3.47	R	Mink #17, BBWMA
18	-28.1000	-24.9033	0.4604	-3.1967	-2.21	R	Mink #38, BBWMA

R denotes an observation with a large standardized residual.

Appendix 3

Levels of Bioaccumulative Chemicals of Concern in Mink In and Out of the Rochester Embayment Area of Concern and Along and Inland from the Shore of Lake Ontario

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March 2007

OVERVIEW

This report is the fourth of four resulting from project C302399 funded by the New York Great Lakes Protection Fund in 2004 to address use impairments related to water quality identified in the Remedial Action Plan for the Rochester Embayment of Lake Ontario (RELO RAP). It deals with the concentrations of bioaccumulative chemicals of concern (BCCs) in mink (*Mustela vison*) in and out of the Rochester Embayment Area of Concern (AOC) and along and inland from the shore of Lake Ontario. The previous reports addressed 1) development and use of videocapture (MustelaVision) systems that established the presence and reproduction of mink in and out of the AOC (Wellman and Haynes 2006a); 2) ages, sizes and trophic positions (stable isotope analysis) of mink in the study areas and a predictive model for bioaccumulation of BCCs by mink (Wellman and Haynes 2006b); and 3) literature review of the effects of BCCs on mink and testing of the bioaccumulation model (Wellman and Haynes 2007) against actual tissue concentrations found in this part of the study. Because mink are the most sensitive species to BCCs known, the results of this four-part project (to be integrated and summarized in a final report in early 2007) will determine whether or not delisting can be recommended for the fish and wildlife population, reproduction and deformities use impairments identified in the RELO AOC.

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Levels of Bioaccumulative Chemicals of Concern in Mink In and Out of the Rochester Embayment Area of Concern and Along and Inland from the Shore of Lake Ontario

INTRODUCTION

In the 1980s the binational (Canada, U.S.) International Joint Commission (IJC) began the process of creating and implementing remedial action plans (RAPs) in 43 contaminated areas of concern (AOCs) 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 AOC, both uses were defined as impaired because "very few" mink were then being trapped or observed within 2 miles of the lake (RAP 1993, 1997). This study was part of a project (Haynes et al. 2002) to determine if populations of mink on the shore of the Rochester Embayment of Lake Ontario (RELO) are negatively impacted by bioaccumulative chemicals of concern (BCCs) and, if so, whether the BCCs are originating in the embayment watershed or elsewhere.

The RELO AOC includes the Embayment, a 35 square mile 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 six mile reach of the Genesee River, from the Lower Falls to the mouth at Lake Ontario. The RAP also includes the subwatersheds of Salmon Creek, the Genesee River and Irondequoit Creek (RAP 1993, 1997; **Figure 1**).

The question addressed by this portion of the study was: What are the current levels of BCCs in lakeshore and inland populations of mink in and out of the AOC, and how do the levels compare between the four regions? These data are needed for comparison to levels of BCCs known to affect mink reproduction (Wellman and Haynes 2007) in order to determine if mink in the RELO AOC are potentially suffering from the "degradation of fish and wildlife populations" and "bird or animal deformities or reproductive problems" listed as use impairments in the RAP (1993, 1997).

MATERIALS AND METHODS

Specimen Collection, Processing and Handling

Collection

Mink carcasses were collected from trappers (after skinning) in five areas. We divided the study area into four Regions: Inland/AOC, Lakeshore/AOC, Inland/Out of AOC, and Lakeshore/Out of AOC. Both Lakeshore regions were identical to those defined by Wellman and Haynes (2006a, 2006b)—Lakeshore/AOC was the Braddock Bay Wildlife Management Area (BBWMA), and Lakeshore/Out of AOC was along the Lake Ontario State Parkway west of Route 19 (LOSPW). Inland/AOC included any animals taken in the AOC watershed more than 5 km from the lakeshore(primarily from areas near the Bergen Swamp), and Inland/Out of AOC included animals taken from the Tug Hill Plateau and the Iroquois National Wildlife Refuge (to provide two presumably "clean" control areas). For the purpose of analysis, we described each collection area using the Regional Descriptors AOC: In vs. Out and Lakeshore vs. Inland.

Carcasses were put in plastic bags and frozen by the trappers as soon as possible. The trappers completed log sheets indicating the date and location of capture for each animal, as well as the trapper's name and contact information. Carcasses were assigned specimen numbers in the

order in which they were collected, and the specimen number, date and location of capture were written on the plastic bags with a permanent marker.

Processing

We thawed the frozen mink carcasses overnight in a refrigerator before processing them. Because some trappers removed the tails when skinning the carcasses, we removed all other tails to obtain comparable measures of body weight and length. We recorded the body weight, tail-less body length, and weight of each tissue sample on a separate sheet for each mink, along with its specimen number and collection record. We placed carcasses in hexane-rinsed aluminum pans or aluminum foil for resection, and all utensils used were rinsed with hexane before each use. Tissues collected for analyses were adipose, liver, brain, testis, kidney and thigh muscle.

Handling and Shipping

Liver and adipose samples were divided into halves or thirds and shipped frozen in dry ice to Columbia Analytical Services (CAS), Inc.'s laboratory in Houston, TX for dioxin/furan analyses, and to the Environmental Research Center (ERC) at SUNY Oswego (co-PI Pagano's lab) for PCB-pesticide-PBDE analyses. If liver or adipose tissue was sufficiently large to divide into thirds, the third sample was frozen and kept at SUNY Brockport. Brain tissues for total mercury analyses were shipped to CAS's laboratory in Kelso, WA. Other tissues (testes, kidney and thigh muscle) were transferred frozen to the ERC for PCB-pesticide-PBDE analyses.

Analytical Procedures: PCBs, Organochlorine Pesticides and PBDEs

Sample Extraction and Clean-Up

The chemicals examined in this study are listed in Appendix A. All tissue samples were extracted for gas chromatographic analysis after methods developed at the SUNY Oswego ERC (Pagano et al. 1999). Pre-cleaned anhydrous sodium sulfate (approximately 10 times sample weight) was added, and the sample extracted three times each with 50 mL hexane using a Brinkman Polytron homogenizer (Model PT 10/35) with small generator (PTA-10S). After each extraction, the hexane extracts were transferred into a volumetric flask and brought to volume. Lipid analysis was conducted by gravimetric procedures utilizing an aliquot (subsample) of the extracted sample. The remaining sample was used for congener-specific PCB, OC pesticide and PBDE analyses. Sample cleanup for OC pesticides followed USEPA Method 3640A (1997) using a Waters Gel Permeation Chromatography (GPC) system (binary pump, Envirogel column, UV detector and fraction collector) followed by specialized silica gel column for separation of PCBs/OCs/PBDEs from other interferences. The analytical methods used to separate PCBs, OCs and PBDEs were based on methods and standard operating procedures (SOPs) developed at the SUNY Oswego ERC and adapted from Method 3630C-USEPA (1997), Basu (1995a,b), and Harlin and Surratt (1995). In general, silica gel adsorption column cleanup utilized 5.5 grams of 4% deactivated silica gel (100-200 mesh) placed in a 10.5x250 mm chromatography column (VWR-Labglass Wilmad) with an upper layer (0.5 g) of anhydrous sodium sulfate. The sample extract was added to the silica gel column and sequentially eluted with hexane and DCM into PCB (F1) and OC/PBDE (F2) fractions, which were concentrated to 1 mL with a Kuderna-Danish (KD) apparatus using a three ball Snyder Column on a steam bath for gas chromatographic analysis.

Chemical Analysis

Congener-specific PCB, hexachlorobenzene, p-p' DDE, and mirex analyses were conducted based on capillary column procedures previously described (Pagano et al. 1995,

Pagano et al. 1998, Pagano et al. 1999, Chiarenzelli et al. 2001). 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 - Ni^{63}) 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 *u*m film thickness. The calibration standard used 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 (dichlorodiphenyldichloroethylene) and Mirex each at 10 pg/uL (Custom Mixed Fraction #3, AccuStandard, Inc.), which allowed for the analysis of 99 chromatographic zones of 132 congeners/co-eluters (Table 1). PCB analyses were confirmed with a HP Model 5890 II gas chromatograph with an electron capture detector (Ni^{63}) and autosampler using a 60 meter DB-XLB capillary column with 0.25 mm id and 0.25 *u*m film thickness. The calibration standard used was a 1:1:1:1:1 mixture of congener mixture sets (C-CSQ-SET 1-5; 10 pg/uL per individual congener, AccuStandard, Inc., New Haven, CT) based on the work of George Frame and co-workers (1996). This analytical setup allowed for analysis of 122 chromatographic zones of 155 congeners/co-eluters (Table 2).

PCB congener nomenclature is based on the arrangement and number of chlorines (1-10) substituted per biphenyl molecule. Congener determination, assignments and accuracy of quantitation were verified for both GC-ECD analytical systems utilizing nine PCB congener mixtures (C-CSQ-SET; AccuStandard, Inc., New Haven, CT) (Frame et al. 1996). Chromatographic data were collected and processed by use of the HP ChemStation software and Microsoft Excel spreadsheet procedures developed at the SUNY Oswego Environmental Research Center (Pagano et al. 1995). The HP software system generated the identity and amount of each PCB congener, confirmed by operator reprocessing of each chromatographic run.

The complex, congener-specific PCB patterns (data) found in mink adipose and liver samples were further processed to a single number representing the average overall chlorination of all congeners in the sample based either on PCB mass or moles of PCBs measured. The unit used was average number of chlorines per biphenyl (Avg Cl/BP). The manipulation of congener-specific data allows for a direct comparison of different types of tissue samples or widely different concentration levels. In addition, congener-specific data provided by other researchers can be transformed to provide a direct comparison of PCB chlorination (qualitative assessment) and concentration (quantitative assessment) between this and previous studies.

Selected organochlorine (OC) pesticides were measured based on Methods 8081A (USEPA 1996). Single instrument/column detection was used for quantitation (DB-XLB, see conditions above). The calibration standard was a 100 pg composite mixture (Single-Column Analytes Mix, M-8081-SC, AccuStandard, Inc.) of USEPA 8081A standard analytes (Table 3). Polybrominated diethyl ethers (PBDEs) were co-analyzed with the OCs on the DB-XLB column setup. The PBDE calibration standard used was an 800 pg/uL (total PBDE - 12 components) solution using the original Great Lakes Chemical Corporation (Great Lakes DE-71, CAS 32534-81-9) technical formulation. The DE-71 technical formulation mass fractions and congener identifications were confirmed with pure PBDE congener standards (BDE-MXE) purchased from Wellington Laboratories (Guelph, ON, Canada) and by mass spectrometric confirmation.

As needed, confirmation of PCBs, OCs, PBDEs, and any co-eluting contaminants were determined utilizing a HP Model 5890II GC with a Model 5971 mass selective detector (GC/EI-MS, electron impact mode) and autosampler. The GC/MS system was set-up to complement the

GC-ECD system, utilizing the same column and temperature programming. Helium was used as the carrier gas at 55 kPa. The injection port and mass selective detector interface were maintained at 270 ^{o}C and 300 ^{o}C, respectively. Selective ion monitoring (SIM) for PCB (SIM-PCB, Cl homologs 1-10) included ions (m/z) 152, 186, 188, 190, 220, 222, 224, 254, 256, 258, 290, 292, 324, 326, 360, 362, 394, 396, 428, 430, 432, 462 and 464 (Pagano et al. 1998).

Laboratory Quality Assurance/Quality Control

The QA/QC program at the SUNY Oswego ERC is based on a program developed from USEPA protocols (USEPA 1997). The program consists of replicate analyses, surrogate analyte recoveries (IUPAC 14, 30 IS (F1+F2), 65, 166, and PCT3-F2), matrix spikes/matrix spike duplicates, and method, reagent and system blanks at prescribed intervals. Surrogate recoveries and surrogate spike checks for the various mink tissues analyzed for this project are reported in Table 4. Instrument detection limits (IDL) and detector linearity were established at the start of the project by replicate analyses (N=7) of progressively smaller serial dilutions from the quantitation standards utilized for each analytical system (acceptance criteria > 10% Relative Standard Deviation, RSD). Analytical detection limits (IDL) and practical detection limits (PDL) are provided in the PDL+IDL worksheet of Appendix B (a spreadsheet on a CD that includes all chemical data collected for this project).

Limitations associated with the accurate and bias-free measurement of low-level PCBs, OCs and PBDEs are generally not due to IDL, but are attributed to the ubiquitous background contamination found in the analytical manipulations necessary to prepare and extract samples. The qualitative nature and quantitative amount of the analytical background is of critical importance when analyzing environmental samples at part per billion levels for individual congeners (Stewart et al. 2000). The SUNY Oswego ERC has developed a methodology to determine practical detection limits (PDL) based on the assessment of the extent and congener-specific distribution of background contamination using method (procedural) blanks analyzed with every sample batch. Method blanks are used to document contamination resulting from the analytical process. Method blanks encompass all Standard Operating Procedure (SOP) sample preparation and analytical manipulations within an analyte-free matrix. PDLs are calculated by multiplying the standard deviation of a series of method blanks by the associated Student t variate (usually N=7, df=6, t=3.134) to provide a known confidence interval ($t_{.99}$) for the PDL estimate. PDLs are reported in the PDL+IDL worksheet of Appendix B.

During the project, general laboratory quality assurance and silica gel method validation was determined by analysis (N=16) of National Institute of Standards and Technology (NIST) Standard Reference Material 1946 (Lake Superior Fish Tissue). Results are provided in the NIST SRM 1946 worksheet of Appendix B. Average recoveries of certified concentration values for PCBs were 92.9% and average recoveries of various organochlorine pesticides was 77.6%.

Analytical Procedures: Dioxins/Furans and Mercury

Dioxin-furan analyses were done using Method 8290A (USEPA 1998) for extractable organics in solid and chemical materials by Columbia Analytical Services, Inc., Houston, TX. Because of analytical uncertainties, because virtually all mercury in biota is methyl mercury (Me-Hg), and because the highest mercury concentrations are found in brain tissue (J. Freemyer, CAS, Houston, TX, pers. comm.), CAS (Kelso, WA) used Method 7471A (USEPA 1994) for total mercury to estimate the Me-Hg concentrations in mink brains. CAS is NELAC-certified (E87611, FAC Rule 64E-1 regulations) by the state of Florida.

Data Analysis

We used Microsoft ® Excel 2000 for data management and non-statistical calculations. For statistical analyses, we used SPSS ® 13.0 for Windows (SPSS Inc., Chicago, IL). We conducted regression analyses to evaluate the relationships between BCC concentrations. We computed descriptive statistics for selected chemicals in selected tissues: 1) total mercury in brain, and 2) total PCBs and dioxin-furan TEQs; average number of chlorine atoms per biphenyl; dieldrin; PBDEs, mirex and DDE in liver and adipose. We used General Linear Models (GLM, a 2-way ANOVA followed by Tukey's pair-wise comparisons and estimates of statistical power) to analyze the relationships between BCC concentrations and the Regional Descriptors—AOC: In vs. Out and Lakeshore vs. Inland. Results for a number of chemicals, detected infrequently or in minute quantities in mink tissues, are not presented here, but all analytical results and calculations are presented in the NYS GLPF MINK worksheet of Appendix B.

Excluding a statistical outlier (mink 17) and results below detection limits, 36 of 40 mink had mercury in brain tissue. Excluding a statistical outlier (mink 17), one animal with insufficient adipose tissue (mink 30), and a procedural error by CAS (addition of the wrong chemical during the preparation of two samples), 33 of 40 mink had total dioxin-furan TEFs above detection limits in adipose tissue. In liver tissue, excluding statistical outliers (17, 22), 17 of 40 mink had total dioxin-furan TEQs above detection limits. For total PCBs, average chlorine number per biphenyl, mirex, dieldrin and DDE, 37 of 41 mink were used in the statistical analyses of liver. Two mink were statistical outliers for total PCBs (17, 22), one was an outlier for mirex (49), and one had insufficient adipose tissue (29). Outliers (> ±3 SE beyond the mean) and values below detection limits (BDL) were excluded from the analyses to avoid skewing general trends high (outliers) or low (BDL ≠ no chemical present in a tissue), respectively. Relationships of the highest and lowest levels of BCCs found in the tissues of mink to their lowest observed adverse effect levels (LOAEL) are addressed by Wellman and Haynes (2006a).

RESULTS

Relationships of BCC Concentrations

Correlations between chemical concentrations were high in adipose and liver tissue. Among the 28 comparisons of seven chemicals and average chlorine per biphenyl in adipose, 25 were highly significant ($P < 0.01$; $r = 0.441$-0.905), two were significant ($P < 0.05$; $r = 0.367$-0.424) and one was suggestive of significance ($P = 0.088$, dieldrin vs. average chlorine per biphenyl; $r = 0.284$) (Table 5). Among the 28 comparisons of seven chemicals and average chlorine per biphenyl in liver, 19 were highly significant ($P < 0.01$; $r = 0.430$-0.899), four were significant ($P < 0.05$; $r = 0.358$-0.408) and two were suggestive of significance ($P < 0.100$; $r = 0.274$-0.278) (Table 6).

BCC Concentrations vs. Regional Descriptors

Total Mercury in Brain Tissue

Total mercury concentrations (Table 7) did not differ (excluding outlier #17) in and out of the AOC ($P = 0.609$; power = 0.079) but concentrations in mink from the Lake Ontario shore were higher than those of inland mink ($P = 0.032$; power = 0.587). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland ($P = 0.835$; power = 0.055).

BCC Concentrations in Adipose Tissue

Total PCB concentrations (Table 8a) did not differ (excluding outlier #17) in and out of the AOC ($P = 0.632$; power = 0.076) but concentrations in mink captured near Lake Ontario

were higher than those of inland mink (P = 0.014; power = 0.708). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.601; power = 0.081).

The average number of chlorine atoms per biphenyl molecule (Table 8b) did not differ (excluding outlier #17) in and out of the AOC (P = 0.475; power = 0.108) but chlorination in mink captured near the Lake Ontario shore was higher than that of inland mink (P = 0.006; power = 0.817). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.375; power = 0.141).

Total dioxin-furan TEQs (Table 8c) did not differ (excluding outlier #17) in and out of the AOC (P = 0.354; power = 0.149) but concentrations in mink captured near Lake Ontario were higher than those of inland mink (P = 0.010; power = 0.763). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.405; power = 0.129).

DDE concentrations (Table 8d) did not differ (excluding outlier #17) in and out of the AOC (P = 0.357; power = 0.148) but concentrations in mink captured near Lake Ontario were higher than those of inland mink (P = 0.002; power = 0.916). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.339; power = 0.156).

Dieldrin concentrations (Table 8e) did not differ (excluding outlier #17) in and out of the AOC (P = 0.241; power = 0.212) but concentrations in mink captured near Lake Ontario were higher than those of inland mink (P = 0.007; power = 0.800). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.614; power = 0.079).

Mirex concentrations (Table 8f) did not differ (excluding outlier #17) in and out of the AOC (P = 0.259; power = 0.200). The data suggested that concentrations in mink captured near Lake Ontario were higher than those of inland mink (P = 0.073; power = 0.536). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.136; power = 0.317).

Polybrominated diphenyl ether concentrations (Table 8g) did not differ (excluding outlier #17) in and out of the AOC (P = 0.937; power = 0.051), but concentrations in mink captured near Lake Ontario were higher than those of inland mink (P = 0.005; power = 0.838). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.266; power = 0.196).

BCC Concentrations in Liver Tissue

Total PCB concentrations (Table 9a) did not differ (excluding outliers #17, 22) in and out of the AOC (P = 0.960; power = 0.050) but concentrations in mink captured near Lake Ontario were higher than those of inland mink (P = 0.018; power = 0.673). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.232; power = 0.219).

The average number of chlorine atoms per biphenyl molecule (Table 9b) did not differ (excluding outliers #17, 22) in and out of the AOC (P = 0.449; power = 0.116) but chlorination in mink captured near Lake Ontario was higher than that of inland mink (P = 0.026; power = 0.618). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.145; power = 0.306).

Total dioxin-furan TEQs (Table 9c) did not differ (excluding outliers #17, 22) in and out of the AOC (P = 0.547; power = 0.089) or between the Lake Ontario shoreline and inland (P = 0.337; power = 0.152), nor was there an interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.423; power = 0.120).

DDE concentrations (Table 9d) did not differ (excluding outliers #17, 22) in and out of the AOC (P = 0.193; power = 0.252) but concentrations in mink captured near Lake Ontario

were higher than those of inland mink (P = 0.005; power = 0.836). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.667; power = 0.071).

Dieldrin concentrations (Table 9e) did not differ (excluding outliers #17, 22) in and out of the AOC (P = 0.363; power = 0.146) but concentrations in mink captured near Lake Ontario were higher than those of inland mink (P = 0.037; power = 0.559). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.260; power = 0.200).

Mirex concentrations (Table 9e) did not differ (excluding outliers #17, 22) in and out of the AOC (P = 0.153; power = 0.295). The data suggested differences between the Lake Ontario shore and inland (P = 0.066; power = 0.455) and an interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.068; power = 0.449).

Polybrominated diethyl ether concentrations (Table 9g) did not differ (excluding outliers #17, 22) in and out of the AOC (P = 0.811; power = 0.056) but concentrations in mink captured near Lake Ontario were higher than those of inland mink (P = 0.002; power = 0.899). There was no interaction between the factors AOC: In vs. Out and Lakeshore vs. Inland (P = 0.143; power = 0.308).

DISCUSSION

The question addressed by this study was: What are the current levels of BCCs in lakeshore and inland populations of mink in and out of the AOC, and how do levels compare between the four regions? Highly consistent patterns were observed across tissues and chemicals.

- Correlations among concentrations of the seven most notable chemicals analyzed were mostly high and significant in adipose and liver tissue.
- There were no significant differences in BCC concentrations in and out of the Rochester Embayment AOC, although mean values were almost always higher (mostly by factors > 3) in the AOC.
- BCC concentrations in mink captured near the Lake Ontario shore were almost always significantly (P < 0.05) or suggestively (0.05 < P < 0.1) greater than concentrations in mink captured inland.
- After removing statistical outliers, there were no significant statistical interactions between the two factors analyzed—AOC: In vs. Out and Lakeshore vs. Inland.

The clear signal in these chemical data are that mink captured near Lake Ontario, and presumably eating organisms exposed to Lake Ontario water and its food web, have significantly higher BCC concentrations in their tissues than mink captured inland. Therefore, it is unlikely that BCC sources in the AOC are 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). Whether the data collected in the four parts of this study (Wellman and Haynes 2006a, 2006b, 2007; this report) support delisting of these two use impairments will be addressed in detail in the final report for this project (Haynes et al., in prep.).

Statistical and Other Issues

Outliers

Mink #17 was excluded from statistical analyses of adipose and liver tissue, and mink #22 was excluded from analysis of liver tissue. The BCC concentrations reported for these two lakeshore-AOC mink are accurate but very high compared to the other lakeshore mink. When

statistical analyses were run including these animals, the resulting significant interaction effects made the results difficult to interpret. Excluding the data for these animals, there were no interaction effects between the treatments AOC: In vs. Out and Lakeshore vs. Inland.

Several factors may account for the high levels of BCCs in minks #17 and #22. Haynes et al. 2004) reported that one sediment sample from Salmon Creek near where mink #17 was captured had a concentration of 1.5 ppm total PCBs. Mink #17 was caught in the large Braddock Bay wetlands complex with broad access to Lake Ontario water and its food web, particularly the carcasses of migrating salmonines. The stable isotope analysis indicated that it fed on organisms about one-half trophic level higher than other mink captured in the Braddock Bay area (Wellman and Haynes 2006b), suggesting that salmonines may have been in its diet. In contrast, lakeshore mink out of the AOC were captured in the upland portion of the Yanty Creek basin south of the Lake Ontario State Parkway, an area with less direct contact to Lake Ontario.

Mink #22 was captured where Round Pond Creek crosses under the Lake Ontario State Parkway (Figure 1), upstream from an area anecdotally reported to have been a munitions factory long ago. Although not previously suspected to exist (RAP 1993, 1997), it is possible that small toxic hotspots exist in the Braddock Bay area to which mink are exposed. Alternatively, it may be that some mink store more BCCs than others due to individual physiological differences in uptake, biotransformation and excretion rates. For example, before beginning the analyses of the 40 mink discussed here, tissues of six mink from the Tug Hill Plateau (presumably a "clean" area) were analyzed to test analytical procedures. For no apparent reason, mink #28 had very high levels of BCCs (Appendix B).

Statistical Power

In each analysis in this report showing no significant difference between the AOC: In vs. Out treatments and their interactions with the Lakeshore vs. Inland treatments, statistical power was very low despite sample sizes of 8-11 mink per treatment. This result was a consequence of high variation in BCC concentrations among animals, even after eliminating statistical outliers (see above). It is notable that the non-significant differences in tissue concentrations reported for inland mink are always greater in than out of the AOC (e.g., TPCB: 1552 vs. 387 ng/g wet, Table 8a; PBDE: 128 vs. 8 ng/g wet, Table 9g). Thus, it is possible that there are differences in the concentrations of BCCs in the tissues of mink in and out of the AOC not detected in our study, but a large number of additional mink would have to be captured and their tissues analyzed to test this hypothesis. Given the large differences in BCC concentrations between lakeshore and inland mink, even if enough additional mink were analyzed it is unlikely that any differences that might be found in concentrations in and out of the AOC would be biologically meaningful in comparison. Therefore, despite low power for the AOC: In vs. Out treatments, the most reasonable conclusion without much greater expense is that there are no biologically meaningful differences in the BCC concentrations of mink in and out of the AOC.

Lack of Co-planar PCB Concentration Data

Due to expense, no co-planar PCB data were collected for this project; therefore, total TEQ values reported here are low. However, it is well established that co-planar PCBs account for 50-90% of dioxin-furan TEQs in tissues (Wellman and Haynes, 2006b), and this issue was addressed in their modeling predictions for total TEQ levels in Rochester Embayment mink.

ACKNOWLEDGMENTS

We are very grateful to Randy Baase who for two winters spent many hours trapping mink in the worst weather, and who taught us much about mink. Thanks are also due to other trappers who contributed carcasses and taught us about mink: Matt Lochner, Dan Carroll, Dan Frey, Michael Ingham, Don Newcombe, Tom Raduns, William Schwartz, Jr., Fred Sinclair and Chuck Tirrano; and to the landowners near the Bergen Swamp who allowed us to collect mink on their property: "Doc" Fink, Dick Sands, Mel Reber and Al Burkhart. We are grateful to Scott Wells and Ross Abbett for dissecting the mink, and to Albert Fulton for creating Figure 1. The government personnel who graciously allowed us to work in their territories include NYS DEC: Dan Carroll, Dave Woodruff and Heidi Bogner Kennedy; NYS OPRHP: James Slusarczyk; and USFWS: Bob Lamoy and Paul Hess. Sara T. Wellman critically reviewed an earlier version of this document. Finally, we gratefully acknowledge grant C302399 from the New York State Great Lakes Protection Fund that made this study possible.

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TABLES

Table 1. Primary column: PCB congeners analyzed with Agilent 25m UltraII.

Peak #	# Cl	IUPAC #		Peak #	# Cl	IUPAC #		Peak #	# Cl	IUPAC #
1	1	1		34	4+4+4	41+64+71		67	7	179
2	1	3		35	5	96		68	6	137
3	2+2	4+10		36	4	40		69	6+7	130+176
4	2+2	7+9		37	4	67		70	6+6+6	138+163+164
5	2	6		38	4	63		71	6	158
6	2+2	5+8		39	4	74		72	6+7	129+178
7		HCB		40	4	70		73	6	166
8	2	14		41	4+5	66+95		74	7+7	182+187
9	3	19		42	5	91		75	7	183
10	3	30		43	4+4+5	56+60+92		76	6+6	128+167
11	2+2	12+13		44	5	84		77	7	185
12	3	18		45	5+5	89+101		78	7	174
13	2+3	15+17		46	5	99		79	7	177
14	3+3	24+27		47	5	119		80	6+7+8	156+171+202
15	3+3	16+32		48	5	83		81	6+7+8	157+173+201
16	3	34		49	5	97		82	7	172
17	3+4	29+54		50	5+5+5	87+115+117		83	8	197
18	3	26		51		p-p'- DDE		84	7	180
19	3	25		52	5	85		85	7	193
20	3	31		53	6	136		86	7	191
21	3	28		54	4+5	77+110+154		87	8	200
22	3+3+4	20+33+53		55	5+6	82+151		88		mirex
23	4	51		56	5+6+6	124+135+144		89	7+7	170+190
24	3	22		57	5+6	109+147		90	8	198
25	4	45		58	5+6	123+149		91	8	199
26	4	46		59	5	118		92	8+8	196+203
27	4	52		60	6	134		93	7	189
28	4+4	43+49		61	5+6	114+133		94	8+9	195+208
29	4+4+4	47+48+75		62	5+6	122+131		95	9	207
30	4	65		63	6	146		96	8	194
31	3	35		64	6	153		97	8	205
32	4	44		65	5+6	105+132		98	9	206
33	3+4+4	37+42+59		66	6	141		99	10	209

Table 2. Confirmation column: PCB congeners analyzed with Agilent 60m DB-XLB.

Peak #	# Cl	IUPAC #		Peak #	# Cl	IUPAC #		Peak #	# Cl	IUPAC #
1	1	1		42	4	42		83	7	179
2	1	2		43	3	35		84	5+6	105+141
3	1	3		44	4	71		85	7	176
4	2+2	4+10		45	3+4	37+41		86	6	137
5	2	9		46	4	64		87	6	130
6	2	7		47	4+5	40+103		88	6	164
7	2	6		48	5	100		89	6	138
8	2	5		49	4	67		90	6	163
		HCB		50	4+5	63+93		91	6+7	129+178
9	2	8		51	5	95		92	6	158
10	3	19		52	4	74		93	7	175
11	2	14		53	4	70		94	7	187
12	3	30		54	4+5	66+91		95	7	183
13	3	18		55	5	92		96	6+7	128+185
14	3	17		56	4+5	56+84		97	7	174
15	2	12		57	5+5	90+101		98	6	167
16	2+3	13+27		58	4	60		99	8	202
17	3	24		59	5	99		100	7	177
18	3	16		60	5+5	83+119		101	7+8	171+201
19	2	15		61	5	97		102	7	173
20	3	32		62	5	87		103	8	197
21	3+4	34+54				p-p' DDE		104	6	156
22	3	29		63	5+6	117+136		105	7	172
23	3	26		64	5+5+6	85+115+154		106	6	157
24	3	25		65	5	110		107	7	180
25	3	31		66	4	81		108	7	193
26	4	53		67	6	151		109	8	200
27	3	28		68	5	82		110	7	191
28	3+3	20+33		69	6	135		111	7	170
29	4	51		70	4+6	77+144		112	8	199
30	4	45		71	6	147		113	7	190
31	3	22		72	6	149				Mirex
32	4	46		73	5	124		114	8	196
33	4	73		74	5+5	109+123		115	8	203
34	4	69		75	6	134		116	9	208
35	4	52		76	5	118		117	7	189
36	4	48		77	6	131		118	8+9	195+207
37	4	49		78	5+6	122+165		119	8	194
38	4+5	47+104		79	6	146		120	8	205
39	4	75		80	5	114		121	9	206
40	4	44		81	6	153		122	10	209
41	4	59		82	6	132				

Table 3. Primary analytical column: organochlorine pesticide components and polybrominated diphenyl ether congeners analyzed with 60m DB-XLB.

Organochlorine Pesticides	**PBDEs**
cis-chlordane	BDE-17
trans-chlordane	BDE-28
alpha-BHC (HCH)	BDE-47
beta-BHC	BDE-66
delta-BHC	BDE-85
gamma-BHC (Lindane)	BDE-99
dieldrin	BDE-100
endosulfan I	BDE-119
endosulfan II	BDE-138
endosulfan sulfate	BDE-153
endrin	BDE-154
hexachlorobenzene (HCB)	
heptachlor epoxide	**Polychlorinated biphenyls (PCBs)**
mirex	**(Dual column confirmational analysis)**
p-p'-DDT	
p-p'-DDD	HP UltraII (25m) 100 zones of 132 congeners/co-eluters
p-p'-DDE	DB-XLB (60m) 122 zones of 155 congeners/co-eluters

Table 4. GLPF mink project surrogate recoveries (SR) and surrogate spike checks for various sample matrices. F1 and F2 denote silica separation fractions.

		SR14-F1	**IS30-F1**	**IS30-F2**	**SR65-F1**	**SR166-F1**	**SRPCT3-F2**
Adipose	**N=42**	90.4%	111.6%	110.1%	89.6%	97.9%	94.2%
	STDEV	17.2%	6.4%	8.7%	20.8%	16.1%	15.8%
Kidney	**N=26**	78.9%	115.5%	97.8%	80.7%	80.8%	67.0%
	STDEV	11.0%	11.1%	6.5%	20.5%	12.3%	10.9%
Testes	**N=33**	86.6%	119.6%	110.5%	85.2%	99.2%	89.6%
	STDEV	14.5%	9.4%	6.0%	13.8%	20.0%	14.1%
Liver	**N=45**	92.0%	118.6%	106.3%	90.2%	82.4%	81.8%
	STDEV	10.4%	7.5%	8.6%	13.8%	26.6%	26.5%
Surrogate	**N=33**	107.9%	114.2%	98.4%	93.2%	97.8%	95.1%
Checks	**STDEV**	6.0%	7.1%	3.2%	6.4%	5.8%	3.3%

Table 5. Correlations of concentrations of selected bioaccumulative chemicals of concern (BCC) in adipose and brain (total Hg only) tissue of mink. PCB = polychlorinated biphenyl; DDE = dichlorodiphenyldichloroethylene; TEQ = 2,3,7,8-tetrachlorodibenzo-p-dioxin toxic equivalents; BDE = polybrominated diethyl ether. *P < 0.05, **P < 0.01; 2-tailed significance level.

BCC	Total PCB	Avg. Cl/ Biphenyl	Dieldrin	DDE	Total TEQ	Mirex	Total BDE
Total Hg							
r =	0.484**	0.424*	0.484**	0.530**	0.536**	0.554**	0.561**
P =	0.004	0.012	0.004	0.001	0.002	0.001	0.001
n =	34	34	34	34	31	34	34
Total PCB							
r =		0.468**	0.460**	0.507**	0.654**	0.809**	0.894**
P =		0.0003	0.004	0.001	<0.001	<0.001	<0.001
n =		37	37	37	30	37	37
Avg. Cl/ Biphenyl							
r =			0.284	0.447**	0.367*	0.464**	0.509**
P =			0.088	0.006	0.046	0.004	0.001
n =			37	37	30	37	37
Dieldrin							
r =				0.905**	0.529**	0.441**	0.501**
P =				<0.001	0.003	0.006	0.002
n =				37	30	37	37
DDE							
r =					0.607**	0.541**	0.525**
P =					<0.001	0.001	0.001
n =					30	37	37
Total TEQ							
r =						0.764**	0.737**
P =						<0.001	<0.001
n =						30	30
Mirex							
r =							0.825**
P =							<0.001
n =							37

Table 6. Correlations of concentrations of selected bioaccumulative chemicals of concern (BCC) in liver and brain (Total Hg only) tissue of mink. PCB = polychlorinated biphenyl; DDE = dichlorodiphenyldichloroethylene; TEQ = 2,3,7,8-tetrachlorodibenzo-p-dioxin toxic equivalents; BDE = polybrominated diethyl ether. *P < 0.05, **P < 0.01; 2-tailed significance level.

BCC	Total PCB	Avg. Cl/ Biphenyl	Dieldrin	DDE	Total TEQ	Mirex	Total BDE
Total Hg							
r =	0.474**	0.430**	0.480**	0.533**	0.473**	0.248	0.467**
P =	0.004	0.010	0.004	0.001	0.006	0.150	0.005
n =	35	35	35	35	32	35	35
Total PCB							
r =							
P =		0.479**	0.408*	0.496**	0.722**	0.621**	0.833**
n =		0.002	0.011	0.002	<0.001	<0.001	<0.001
		38	38	33	31	38	38
Avg. Cl/ Biphenyl							
r =			0.278	0.453**	0.379*	0.274	0.468**
P =			0.091	0.004	0.036	0.096	0.003
n =			38	38	31	38	38
Dieldrin							
r =				0.899**	0.399*	0.113	0.358*
P =				<0.001	0.026	0.499	0.027
n =				38	31	38	38
DDE							
r =					0.532**	0.251	0.450**
P =					0.002	0.129	0.005
n =					31	38	38
Total TEQ							
r =							
P =						0.759**	0.841**
n =						<0.001	<0.001
						31	31
Mirex							
r =							0.822**
P =							<0.001
n =							38

Table 7. Total mercury concentrations (ng/g) in the brains of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	8	0.281	0.143	0.609	0.079
Inland	10	0.158	0.154		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	9	0.296	0.155	0.032	0.587
Inland	9	0.194	0.145		
				Interaction	
				0.835	0.055

Table 8a. Total polychlorinated biphenyl (TPCB) concentrations (ng/g wet) in the adipose tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	9	3918.3	5780.1	0.632	0.076
Inland	11	1552.4	2410.9		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	3970.4	3280.8	0.014	0.708
Inland	8	387.3	226.2		
				Interaction	
				0.601	0.081

Table 8b. Average number of chlorine atoms per biphenyl in the adipose tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	9	6.35	0.28	0.475	0.108
Inland	11	6.16	0.24		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	6.37	0.27	0.006	0.817
Inland	8	6.10	0.37		
				Interaction	
				0.808	0.141

Table 8c. Total TEQ values for dioxins and furans (ng/Kg) in the adipose tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	7	10.281	7.229	0.354	0.149
Inland	10	4.723	4.143		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	9	15.436	12.159	0.010	0.763
Inland	6	5.005	5.252		
				Interaction	
				0.405	0.129

Table 8d. Dichlorodiphenyldichloroethylene (DDE) concentrations (ng/g wet) in the adipose tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	9	5612.2	6118.9	0.357	0.148
Inland	11	2600.5	2967.3		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	5656.1	3081.7	0.002	0.916
Inland	8	276.9	452.8		
				Interaction	
				0.339	0.156

Table 8e. Dieldrin concentrations (ng/g wet) in the adipose tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	9	40.1	51.4	0.241	0.212
Inland	11	18.6	15.0		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	33.9	17.1	0.007	0.800
Inland	8	3.2	1.7		
				Interaction	
				0.614	0.079

Table 8f. Mirex concentrations (ng/g wet) in the adipose tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	9	16.2	22.1	0.259	0.200
Inland	11	9.3	13.8		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	72.6	123.0	0.073	0.436
Inland	8	1.3	1.2		
				Interaction	
				0.136	0.317

Table 8g. Polybrominated diphenyl ether (PBDE) concentrations (ng/g wet) in the adipose tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	9	177.9	167.3	0.937	0.051
Inland	11	87.6	99.0		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	227.7	208.5	0.005	0.838
Inland	8	30.4	11.9		
				Interaction	
				0.266	0.196

Table 9a. Total polychlorinated biphenyl (PCB) concentrations (ng/g wet) in the liver tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	8	164.3	219.7	0.960	0.050
Inland	11	92.6	168.3		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	230.5	227.3	0.018	0.673
Inland	10	20.6	12.7		
				Interaction	
				0.232	0.219

Table 9b. Average number of chlorine atoms per biphenyl in the liver tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	8	6.14	0.31	0.449	0.116
Inland	11	6.05	0.36		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	6.37	0.20	0.026	0.618
Inland	10	5.97	0.37		
				Interaction	
				0.145	0.306

Table 9c. Total TEQ values for dioxins and furans (ng/Kg) in the liver tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	3	1.655	1.656	0.547	0.089
Inland	5	1.009	1.778		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	5	7.201	13.772	0.337	0.152
Inland	4	0.206	0.390		
				Interaction	
				0.423	0.120

Table 9d. Dichlorodiphenyldichloroethylene (DDE) concentrations (ng/g wet) in the liver tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	8	305.0	354.0	0.193	0.252
Inland	11	128.2	216.4		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	244.0	157.1	0.005	0.836
Inland	10	8.0	11.0		
				Interaction	
				0.667	0.071

Table 9e. Dieldrin concentrations (ng/g wet) in the liver tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	9	10.7	11.6	0.363	0.146
Inland	11	7.2	14.4		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	11.4	9.3	0.037	0.559
Inland	8	0.3	0.2		
				Interaction	
				0.260	0.200

Table 9f. Mirex concentrations (ng/g wet) in the liver tissue of mink.

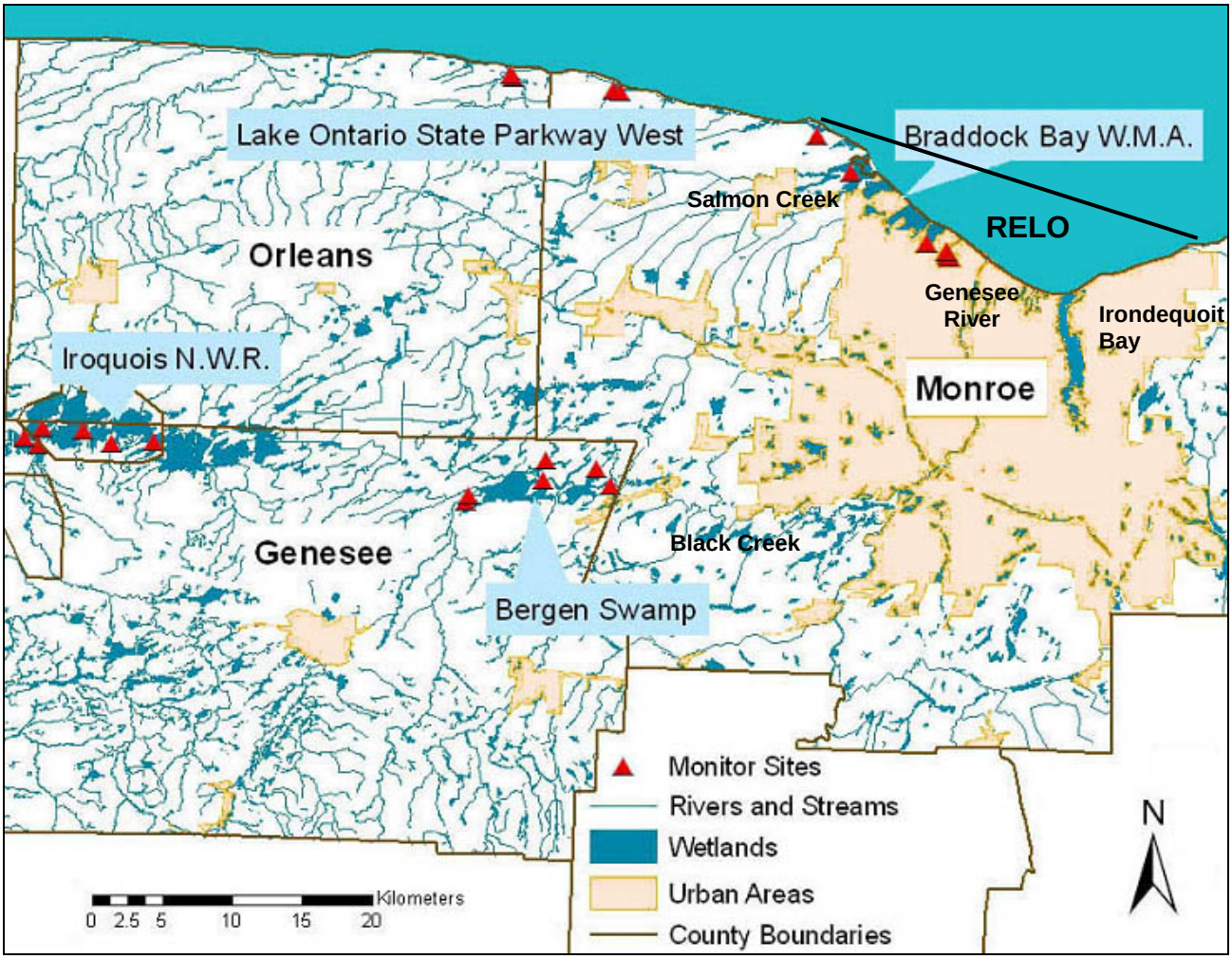
Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	8	3.2	5.4	0.153	0.295
Inland	11	3.1	6.4		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	25.9	40.7	0.066	0.455
Inland	10	0.2	0.2		
				Interaction	
				0.068	0.449

Table 9g. Polybrominated diphenyl ether (PBDE) concentrations (ng/g wet) in the liver tissue of mink.

Location	N	Mean	Std. Dev.	P-value	Power
AOC: In				AOC: In vs. Out	
Lakeshore	8	6.1	7.5	0.811	0.056
Inland	11	3.0	3.7		
AOC: Out				Lakeshore vs. Inland	
Lakeshore	10	9.1	7.1	0.002	0.899
Inland	10	0.8	0.6		
				Interaction	
				0.143	0.308

FIGURES

Figure 4. Map showing the four regions referred to in the study. AOC/Lakeshore is Braddock Bay WMA, AOC/Inland is at least 3 km from Lake Ontario, Out of AOC/Lakeshore is the Lake Ontario State Parkway west of Rte.19, and Out of AOC/Inland is Iroquois NWR and the Tug Hill Plateau (not shown). RELO is the Rochester Embayment of Lake Ontario. (Map by Albert Fulton 2005.)



APPENDICES

Appendix A. Chemicals detected in this study. Names in bold were analyzed statistically and are discussed in the text. Raw data for all chemical are in Appendix B (CD).

Chemical	Abbreviation
Total Polychlorinated Biphenyls[1,2]	**TPCB**
Cl-1	
Cl-2	
Cl-3	
Cl-4	
Cl-5	
Cl-6	
Cl-7	
Cl-8	
Cl-9	
Cl-10	
Pesticides	
(cis) alpha-chlordane	ACHLOR
(trans) gamma-chlordane	GCHLOR
Aldrin	ALDRIN
alpha-BHC (HCH)	ABHC
beta-BHC	BBHC
delta-BHC	DBHC
Dieldrin	**DIELDRIN**
endosulfan I	ENDO1
endosulfan II	ENDO2
endosulfan sulfate	ENDOSUL
Endrin	ENDRIN
endrin aldehyde	ENDA
endrin ketone	ENDK
gamma-BHC	GBHC
Heptachlor	HEP
heptachlor epoxide	HEPEPO
Hexachlorobenzene	HCB
Methoxychlor	METH
Mirex	**MIREX**
p-p'-DDD	DDD
p-p'-DDE	**DDE**
p-p'-DDT	DDT
Total Brominated Diethyl Ethers	**TBDE**
BDE-17	BDE17
BDE-28	BDE28
BDE-47	BDE47

BDE-66	BDE66
BDE-85	BDE85
BDE-99	BDE99
BDE-100	BDE100
BDE-119	BDE119
BDE-138	BDE138
BDE-153	BDE153
BDE-154	BDE154
Dibenzo-p-dioxins	
2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)	TCDD
1,2,3,7,8-Pentachlorodibenzo-p-dioxin (PeCDD)	PeCDD
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	HxCDD
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin (HxCDD)	HxCDD2
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin (HxCDD)	HxCDD3
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin (HpCDD)	HpCDD
Octachlorodibenzo-p-dioxin (OCDD)	OCDD
Tetrachlorodibenzofurans	
2,3,7,8-Tetrachlorodibenzofuran (TCDF)	TCDF
1,2,3,7,8-Pentachlorodibenzofuran (PeCDF)	PeCDF
2,3,4,7,8-Pentachlorodibenzofuran (PeCDF)	PeCDF2
1,2,3,4,7,8-Hexachlorodibenzofuran (HxCDF)	HxCDF
1,2,3,6,7,8-Hexachlorodibenzofuran (HxCDF)	HxCDF2
1,2,3,7,8,9-Hexachlorodibenzofuran (HxCDF)	HxCDF3
2,3,4,6,7,8-Hexachlorodibenzofuran (HxCDF)	HxCDF4
1,2,3,4,6,7,8-Heptachlorodibenzofuran (HpCDF)	HpCDF
1,2,3,4,7,8,9-Heptachlorodibenzofuran (HpCDF)	HpCDF2
Octachlorodibenzofuran (OCDF)	OCDF
Total Toxic Equivalents[1] (TEQ)	**Total TEQ**
Total Mercury	**Total Hg**

[1]Excluding co-planar PCBs. See Discussion for explanation.

[2]PCB homologues were determined by spreadsheet manipulations (see NYS GLPF MINK worksheet in Appendix B) based on congener-specific PCB measurements. PCB data were further processed such that the mole percent (congener specific and homologue) and average chlorine/biphenyl (Cl/BP) values were generated. Coeluting congeners were assumed to be in equal proportions for all spreadsheet calculations (Pagano et al., 1995).

Appendix B. Separate spreadsheet on a CD with all BCC data collected in this study.

Appendix 4

Bioaccumulative Chemicals of Concern in Mink: Adverse Effects Levels and Results of a Predictive Model for the Rochester Embayment of Lake Ontario

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March 2007

OVERVIEW

This report is the fourth of four resulting from project C302399 funded by the New York Great Lakes Protection Fund in 2004 to address use impairments related to water quality identified in the Remedial Action Plan for the Rochester Embayment of Lake Ontario (RELO RAP). It gives a brief review of pertinent literature and reports the results of a model for bioaccumulation of selected chemicals in mink in the Rochester Embayment. Previous reports addressed the 1) development and use of videocapture (Mustelavision) systems that established the presence and reproduction of mink in and out of the RELO RAP Area of Concern (AOC); ages, sizes and trophic positions (stable isotope analysis) of mink (*Mustela vison*) in the study area; and 3) levels of BCCs in mink tissues. Because mink are the most sensitive known species to BCCs, the results of this four-part project will determine whether delisting the fish and wildlife reproduction impairment for the RELO AOC can be recommended.

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INTRODUCTION

In the 1980s the binational (Canada, U.S.) International Joint Commission (IJC) began the process of creating and implementing remedial action plans (RAPs) in 43 contaminated areas of concern (AOCs) 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 AOC, both uses were defined as impaired because "very few" mink (*Mustela vison*) were then being trapped or observed within 2 miles of the lake (RAP 1993, 1997). This study was part of a project (Haynes *et al.* 2002) to determine if populations of mink on the shore of the Rochester Embayment of Lake Ontario (RELO) are negatively impacted by bioaccumulative chemicals of concern (BCCs) and, if so, whether the BCCs originate in the embayment watershed or elsewhere.

The RELO AOC includes the Embayment, a 35 square mile 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 six mile reach of the Genesee River from the Lower Falls to the mouth at Lake Ontario. The RAP also includes the subwatersheds of Salmon Creek, the Genesee River and Irondequoit Creek (RAP 1993, 1997).

The initial questions addressed by this portion of the study were: 1) Which BCCs, and at what levels, are known to cause adverse effects on populations or reproduction, or to cause deformities, in mink? 2) How do predicted levels of BCCs in mink tissues (based on concentrations in Lake Ontario water) compare with measured tissue residues in our lakeshore mink specimens?

Our approach to the first question was to do a literature search, looking for reports on the levels of BCCs in mink tissues corresponding to adverse effects. This gave us values to compare to the tissue residues we found in mink (Pagano and Haynes, in preparation). The results of the comparisons will be described in the final report for this project (Haynes *et al.,* in preparation), along with an assessment of risk to AOC and Lake Ontario shoreline mink.

To answer the second question, our approach was to provide a predictive model for the bioaccumulation and biomagnification of BCCs in AOC mink tissues based on concentrations of BCCs in the waters of Lake Ontario (Wellman and Haynes 2006). If model predictions are well correlated with actual tissue concentrations in mink, by knowing tissue residue levels that cause adverse effects we will have created a risk assessment tool for mink without the expense of tissue analyses.

ADVERSE EFFECTS LEVELS

Method

Basu *et al.* (2006) wrote an extensive review of the literature on the toxic effects of BCCs in mink, covering field and lab studies and the use of mink in hazard assessments. Leonards *et al.* (1995) and Kannan *et al.* (2000) reviewed the available literature on the toxicity of PCBs to mink. Rather than duplicate those efforts, we focused on studies which linked dietary levels of BCCs to tissue residues as well as to reproductive or other effects. Such studies allow direct comparisons of our tissue residue results (Pagano and Haynes, in preparation) to levels associated with adverse effects. We also concentrated on reports of

chronic exposures, as they would best represent the exposures of mink to BCCs in the environment. Search engines used included Academic Search Premier, BioOne, BasicBIOSIS, InfoTrac OneFile, JSTOR, and ScienceDirect.

Results and Discussion

We found studies linking dietary concentrations to tissue residues for dioxins and furans, PCBs, and mercury. No studies of this type were found for any of the organo-chlorine (OC) pesticides except hexachlorobenzene (HCB). Rush *et al.* (1983) reported increased kit mortality at 1 ppm HCB, resulting in adipose residues of 95 ppb and undetectable liver residues. According to Giesy *et al.* (1994), studies in the 1970s and 1980s determined that OC pesticides were not the cause of effects seen in mink that ate Great Lakes fish. Because OC pesticide levels have decreased in the environment since then, they would be even less significant today, which probably accounts for the lack of studies regarding them.

Dioxins and furans

The toxicity of dioxins and furans to mink is well-established (Basu *et al.* 2006, Hochstein *et al.* 1998, 2001, Render *et al.* 2000, 2001), and LOAELs (Lowest Observable Adverse Effect Levels) are frequently reported as 2,3,7,8-TCDD equivalents (TEQs) in ppb (dietary) or pg/g (tissue residue) wet weight.

Many studies evaluated toxic end points in terms of dietary concentrations but not as tissue residues in the mink. Render *et al.* (2000) reported that a dietary concentration of 5 ppb 2,3,7,8-TCDD fed to adult females for six months caused proliferation of squamous epithelial cells in bone adjacent to teeth, and Render *et al.* (2001) found the same effect in 6- and 12-week-old kits fed 2.4 ppb 2,3,7,8-TCDD for as little as 14 days. Hochstein *et al.* (1998) reported that 1 ppb caused 62.5% mortality in adult female mink fed 2,3,7,8-TCDD for 125 days. Hochstein *et al.* (2001) found that when mink dams were fed 0.053 ppb 2,3,7,8-TCDD, kit survival was reduced to 47% vs. 83% in the control group (0.0006 ppb TCDD). Unfortunately, these studies do not provide data that is directly comparable to our tissue analyses (Pagano and Haynes, in preparation).

Tissue residues as well as dietary concentrations were reported by several researchers feeding wild-caught fish to mink. Since the fish used as the source of BCCs contained PCBs as well as dioxins and furans, their total TEQ values also included contributions from the coplanar PCBs in the fish (Table 1 and Appendix A). Heaton *et al.* (1995) and Tillit *et al.* (1996) reported reduced 3- and 6-week-old kit survival at a maternal dietary concentration of 22.4 pg TEQ/g (0.72 μg/g TPCBs), which resulted in maternal liver residues of 208.3 pg TEQ/g (2.19 μg/g TPCBs). Bursian *et al.* (2006a, b) reported increased mortality in 3- to 6-week-old mink kits whose dams had been fed 68.5 pg TEQ/g (3.7 μg/g TPCBs), resulting in maternal liver residue levels of 218.4 pgTEQ/g (3.133 μg/g TPCBs). Bursian *et al.* (2006a, b, c) also found jaw lesions in 27- and 31-week-old juveniles fed 47 pg TEQ/g (1.1 μg/g TPCBs) and 9.2 pg TEQ/g (0.96 μg/g TPCBs), respectively, with corresponding juvenile liver residues of 75 pg TEQ/g (16 μg/g TPCBs) and 40.2 pg TEQ/g (1.7 μg/g TPCBs).

Polychlorinated biphenyls

Bursian *et al.* (2006a) found that the dietary LC_{10} and LC_{20} for total PCBs were 0.231 and 0.984 μg/g TPCBs, and estimated a threshold dietary concentration of 33.2 pg/g TPCBs. Restum *et al*. (1998) reported reduced whelping in dams fed 0.25 ppm PCBs, resulting in a liver concentration of 860 ng/g. Halbrook *et al.* (1999) found a trend (P = 0.069) for reduced

litter size in dams fed 1360 ppb Aroclor 1260 equivalent (EQ) PCBs in the diet, resulting in a maternal liver concentration of 7.25 ppm Aroclor 1260 EQ and a maternal adipose concentration of 129 ppm Aroclor 1260 EQ. However, these results may have been confounded by the presence of mercury in the fish used in the diet, resulting in 0.22 ppm Hg in the diet and 3.67 ppm in the maternal livers.

Mercury

In 1974, Aulerich *et al.* reported that all mink fed a diet including 5 ppm mercury died after 29 days of treatment, with brain residue levels of approximately 20 ppm. Wobeser *et al.* (1976) reported that 1.1 ppm dietary MeHg caused "classic mercury intoxication," including neurotoxicity; the corresponding brain concentration was 8.2 ppm. Wren *et al.* (1987a, b) reported that 1.0 μg/g dietary MeHg caused adult mortality as well as a reduction in litter size, with a brain residue of 15.3 μg/g. However, this dietary level is misleading because the 1 ppm chow was used only every other day after unexpected mortalities within less than three months.

Dansereau *et al.* (1999) reported a reduction in the proportion of females whelping at dietary levels of 0.5 ppm Hg, resulting in liver residues of 80.4 μg/g. Halbrook *et al.* (1997) found reduced litter size at 0.22 ppm dietary Hg (in fish), resulting in liver residues of 3.67 ppm. Using the average of brain:liver residue ratios from Evans *et al.* (2000), Wobeser *et al.* (1976) and Wren *et al.* (1987a, b), we estimated the brain residues for the reduced whelping (Dansereau *et al.* 1999) as 23.2 μg/g, and for reduced litter size (Halbrook *et al.* 1997) as 1.06 μg/g. While these estimations are not strictly accurate, because the brain:liver ratio varies within and among studies, they should be close enough to allow comparison with our brain residue results (Pagano and Haynes, in preparation).

Overlaps of PCB and dioxin/furan toxicities

Unfortunately, miscommunication between PIs Pagano and Haynes and with Columbia Analytical Services resulted in no analyses for co-planar PCBs in our study. However, several studies show distinct relationships between total TEQ, PCB TEQ and dioxin-furan TEQ. Analyses of fish from the Housatonic River used in mink diets by Bursian *et al.* (2006a) showed that approximately 91% of the dietary TEQ value was contributed by PCBs, and only about one-tenth of the TEQ value by dioxins and furans. According to Bursian (2006c), Heaton *et al.* (1995) and Tillitt *et al.* (1996) reported that PCBs accounted for 73% of the TEQ in fish from Saginaw Bay, Michigan. In contrast, PCBs made up less than 44% of the TEQ in the Saginaw Bay fish examined by Bursian *et al.* (2006c), with about half the TEQ value contributed by dioxins and furans. Thus, an estimate of the total environmental TEQ exposure, based on analysis of only dioxins and furans, would have to multiply the dioxin/furan TEQ by a correction factor between two and ten, and this is what we did.

Conclusion

In our literature review, jaw lesions had the lowest LOAELs (Table 1). Table 2 shows total TEQ values (low, average, high) for lakeshore and inland mink in the AOC, based on dioxin/furan analyses, and calculated estimates including co-planar PCBs. The highest measured TEQ value for AOC lakeshore mink in our study was 47.62 pg TEQ/g wet weight (Pagano and Haynes, in preparation), which is higher than the LOAEL of 40 pg TEQ/g liver at which jaw lesions were seen (Bursian *et al.* 2006a, b). The lowest measured TEQ value in lakeshore mink, 0.22 pg TEQ/g, even when multiplied by ten is still an order of magnitude smaller than the LOAEL, indicating no risk. However, the average (excluding high and low) of 7.8 pg TEQ/g for our lakeshore mink (Pagano and Haynes, in preparation), if multiplied by a correction factor of five, approaches the LOAEL for jaw lesions. This would indicate that some

lakeshore mink are at risk of developing jaw lesions, which have been shown to lead to jaw deformities, osteolysis, and tooth loss (Render *et al.* 2001). Histological examinations of our minks' jaws for this lesion could help to determine whether to delist the RELO AOC for deformities. Further comparisons of the levels of BCCs in our mink tissues to LOAELs will be presented in our final report (Haynes *et al.* in preparation).

The highest measured TEQ value for inland mink was 4.16 pg TEQ/g. When multiplied by the correction factor of ten, the result is approximately equal to the LOAEL of 40 pg TEQ/g, indicating that the most exposed of the inland mink may be at risk for developing jaw lesions. However, the lowest value of 0.00 pg TEQ/g and the average TEQ value of 0.25 pg TEQ/g, even when multiplied by a factor of ten, indicate that the majority of inland mink are not at risk.

BIOACCUMULATION MODEL

Method

For modeling the bioaccumulation of chemicals in mink, we started with Equation 28 from Sample *et al.* (1996), adding the units for clarity:

$$C_w\left(\frac{mg}{L}\right)=\frac{NOAEL\left(\frac{mg}{kg\cdot d}\right)\times bw(kg)}{W\left(\frac{L}{d}\right)+\left[F\left(\frac{kg}{d}\right)\times BAF\right]}, \qquad (1)$$

where C_w is the concentration of the BCC in the water, *NOAEL* is the No Observed Adverse Effects Level; *W* and *F* are the daily water and food consumption rates in L/day and kg/day, respectively; *BAF* is the bioaccumulation factor for the chemical of concern (based on the trophic level of the animal and the octanol-water partition coefficient, k_{ow}, a measure of hydrophobicity of the compound); and *bw* is the body weight of the animal in kilograms (Sample *et al.* 1996).

We solved for *NOAEL*, and taking into account the aquatic portion of the animal's diet (P_{aq}), got an equation to predict the exposure level of an animal to a BCC in water:

$$NOAEL\left(\frac{mg}{kg\ (bw)\cdot d}\right)=\frac{C_w\left(\frac{mg}{kg}\right)[W(kg)+(F(kg)\times P_{aq}\times BAF)]}{bw(kg)}. \qquad (2)$$

According to Sample *et al.* (1996), the dietary concentration C_f (mg/kg) equivalent to the NOAEL is

$$C_f\left(\frac{mg}{kg}\right)=\frac{NOAEL\left(\frac{mg}{kg\cdot d}\right)\times bw(kg)}{F\left(\frac{kg}{d}\right)}. \qquad (3)$$

Substituting equation (2) for *NOAEL* into equation (3) for C_f, the *bw* terms in the numerator and denominator cancel out and give a dietary concentration equivalent to the exposure level based on the BCC concentration in water, the food and water consumption rates of mink (177 g and 0.1 L as reported by Wellman and Haynes 2006), the percent aquatic diet

(between 50 and 90% as in USEPA 1995), and the bioaccumulation factor (Sample *et al.* 1996):

$$C_f\left(\frac{mg}{kg}\right)=\frac{C_w\left(\frac{mg}{kg}\right)\left[W\left(\frac{kg}{d}\right)+F\left(\frac{kg}{d}\right)\times P_{aq}\times BAF\right]}{F\left(\frac{kg}{d}\right)}. \qquad (4)$$

This dietary concentration equivalent can be directly compared to dietary concentrations of BCCs known to cause adverse effects in mink.

Using the highest and lowest values for diet-to-tissue biomagnification factors (BMF_t) calculated from the literature (see Appendix A), we predicted levels of selected BCCs in mink tissue with the equation

$$C_t\left(\frac{\mu g}{g}\right)=C_f\left(\frac{\mu g}{g}\right)\times BMF_t. \qquad (5)$$

Results and Discussion

Table 3 compares the predicted low and high values of total PCBs and TEQs from dioxins and furans to the lowest and highest tissue levels found in the livers, and the values of MeHg in the brains, of lakeshore mink. The low predicted values were calculated using the lowest C_w found in either Luckey and Litton (2005) or in Environment Canada's 2004 survey of Lake Ontario (J. Vincent, personal communication), and assuming 50% aquatic diet and the lowest diet-to-tissue BMF calculated from the literature (Hg: Wobeser *et al.* 1976; TEQs: Heaton *et al.* 1995, Tillitt *et al.* 1996; PCBs: Bursian *et al*. 2006a, b). The high predicted values were calculated using the highest C_w, 90% aquatic diet, and highest BMF (Hg: Wobeser *et al.* 1976; TEQs: Heaton *et al.* 1995, Tillitt *et al.* 1996; PCBs: Halbrook *et al*. 1999). The measured values were provided by J. Pagano (personal communication) and will be detailed in a forthcoming report (Pagano and Haynes, in preparation).

The model worked well for dioxin/furan TEQs and for PCBs. In both cases, the predicted low and high values bounded our measured values, except for the low estimate for PCBs, which was very close to the lowest measured value in lakeshore mink. This is to be expected, as the AOC is neither the most polluted nor the cleanest portion of Lake Ontario (Luckey and Litton 2005; J. Vincent, personal communication). The model did not predict tissue levels of mercury well; the measured values were up to three orders of magnitude higher than predicted values. The reason for this discrepancy is not known. One possibility is the fact that the model is based on the octanol-water coefficient, a concept which applies only to lipophilic compounds, which mercury is not. However, Sample *et al.* (1996) apparently intended the model to be used with mercury, as they provided BAF factors for it (as well as several other heavy metals). Another possibility is be that the model predicts mercury concentrations in tissue based only on aquatic exposures, while the mink in our study might have had exposure to mercury through terrestrial sources unaccounted for by the model. Further investigation and development of the model will be required if it is deemed necessary to predict mercury levels in mink of the Rochester Embayment.

SUMMARY

The first question addressed by this study was: Which BCCs, and at what levels, are known to cause adverse effects on populations or reproduction, or to cause deformities, in mink? We found that the most sensitive endpoint for PCBs and TEQs was a squamous epithelial cell lesion in mink jawbones, corresponding to 40.2 pg TEQ/g wet weight in mink livers (Bursian *et al.* 2006a, b). We compared this level to tissue residues in Lake Ontario shoreline mink, and concluded that lakeshore mink are at risk for developing jaw lesions. We have the jaws of mink used in this study. A small amount of additional funding would permit analysis of lesions and final determination of whether mink in the Rochester Embayment AOC are adversely impacted by BCCs.

The second question addressed by this study was: How do predicted levels of BCCs in mink tissues (based on concentrations in Lake Ontario water) compare with measured tissue residues in our lakeshore mink specimens? We found that for PCBs and dioxins/furans, the model worked well, but it was less successful in predicting levels of mercury in lakeshore mink.

ACKNOWLEDGMENTS

We are grateful to Steve Bursian, Bob Gilliam, Kurunthachalam Kannan, Fred Luckey, Gary Neuderfer, Don Tillit, Jennifer Vincent, and John Waud for providing articles or data required for this study.

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TABLES

Table 1. Selected endpoints and effects levels reported for mercury, PCBs, and TEQs in mink diets and tissues. (Values in italics were estimated by the authors of this report, using the average brain:liver ratios from Evans *et al.* 2000, Wobeser *et al.* 1976, and Wren *et al*. 1987a, b.) CDD = chlorinated dibenzo dioxins, CDF = chlorinated dibenzo furans, HCB = hexachlorobenzene.

Impairment	Endpoint	Toxin	Effect Level	Conc. (ppm or ug/g) Diet	Tissue	Reference
					Brain	
Population	Adult mortality	Hg	LC100	5 ppm	19.9 ppm	Aulerich et al. 1974
Reproduction	Whelping reduced	Hg in fish	LOAEL	0.5 ppm	*23.2 ug/g*	Dansereau et al. 1999
Reproduction	Litter size reduced	Hg in fish	LOAEL	0.22 ppm	*1.06 ppm*	Halbrook et al. 1997
Population	Hg intoxication	MeHg	LOAEL	1.1 ppm	8.2 ppm	Wobeser et al. 1976
Reproduction	Litter size reduced	MeHg	LOAEL	1.0 ug/g	2.0 ug/g	Wren et al. 1987a,b
					Liver	
Reproduction	Kit survival 3 & 6 wks	PCBs	LOAEL	720 pg/g	2190 pg/g	Heaton et al. 1995, Tillit et al. 1996
		CDDs	LOAEL	60 pg/g	2626 pg/g	
		CDFs	LOAEL	13 pg/g	335 pg/g	
		TEQs	LOAEL	22.4 pg/g	208.3 pg/g	
Deformities	Jaw lesion in 31-wk kits	PCBs	LOAEL	0.96 ug/g	1.698 ug/g	Bursian et al. 2006a, b
		TEQs	LOAEL	9.2 pg/g	40.2 pg/g	
Deformities	Jaw lesion in 27-wk kits	PCBs	LOAEL	1.1 ug/g	16 ug/g	Bursian et al. 2006c
		TEQs	LOAEL	47 pg/g	75 pg/g	
Reproduction	Litter size	PCBs	LOAEL	1360 ppb	7250 ppb	Halbrook et al. 1999
Reproduction	P-1 Whelping reduced	PCBs	LOAEL	0.25 ppm	860 ng/g	Restum et al. 1998
	F-2 Kit mortality	PCBs	LOAEL	0.5 ppm	464 ng/g	
					Adipose	
Reproduction	Kit mortality	HCB	LOAEL	1 ppm	95 ppb	Rush et al. 1983

Table 2. TEQ values from dioxins and furans for Lakeshore and Inland mink livers, showing high, low and average (excluding high and low) values for each category.

Location	Value	TEQ	TEQ*2	TEQ*10
Lakeshore	Low	0.22	0.44	2.2
	Average (8)	7.75	15.50	77.5
	High	47.62	95.24	476.2
Inland	Low	0.00	0.00	0.00
	Average (8)	0.25	0.50	2.50
	High	4.16	8.32	41.6

Table 3. Predicted versus measured values for tissue residues of dioxin/furans (TEQs), methylmercury, and PCBs, based on water concentrations in Lake Ontario as reported by J. Vincent (2006, personal communication) and Luckey and Litton (2005).

			Tissue Level	
		Water Conc.	**Predicted**	**Measured**
BCC	**Value**	**pg/kg**	**ng/g**	**ng/g**
TEQs (liver)	Low	6.00E-05	5.52E-05	2.20E-04
	High	2.40E-02	6.21E-02	2.13E-02
MeHg (brain)	Low	0.00E+00	0.00E+00	9.00E+01
	High	1.80E+01	4.70E+00	1.55E+03
PCBs (liver)	Low	2.60E+01	1.92E+01	1.36E+01
	High	9.15E+02	1.60E+05	5.87E+03

Appendix A: Adverse Effects Levels for Selected BCCs

Notes

NOAEL = No Observed Adverse Effects Level
LOAEL = Lowest Observed Adverse Effects Level
ETD = Estimated Threshold Dose; geometric mean of NOAEL and LOAEL =(SqRt(NOAEL X LOAEL))
LC10 = Concentration Lethal to 10% of population
TEQs = 2,3,7,8-Tetrachloro-dibenzi-P-dioxin equivalent units
Tissue/Diet BMF is diet-to-tissue Biomagnification Factor
In rare cases, we have made an estimation based on reported data. Our estimations are shown in italics.
Unless noted as "estimated by STW", all calculations are done by the authors.
NA = Not Available or Not Addressed in report

LOAELs for each report are highlighted in yellow

Estimated threshold values are highlighted in gold

A-1. Adverse Effects Levels of Dioxin

		Effect	Concentration			
Endpoint	**Toxin**	**Level**	**Diet**	**Tissue**	**References**	**Notes**
Adult Mortality	TCDD	NOAEL	0.1 ppb		**Hochstein et al. 1998**	
		LOAEL	1.0 ppb			
Adult Mortality	TCDD	NOAEL	0.18 ppb	N/A	**Hochstein et al. 2001**	
		LOAEL	1.40 ppb			
Kit survival @ 3 wks		NOAEL	0.0006 ppb			
		LOAEL	0.053 ppb			
Toenail deformities		NOAEL	0.18 ppb			
		LOAEL	1.40 ppb			
Jaw lesions	TCDD	NOAEL	0 ppb	N/A	**Render et al. 2000**	
		LOAEL	5 ppb			
Jaw lesions	TCDD	NOAEL	0 ppb	N/A	**Render et al. 2001**	Jaw lesions caused deformities and displacement of teeth
		LOAEL	2.4 ppb			
Kit mortality		NOAEL	0 ppb	N/A		
		LOAEL	24 ppb			

A-2. Adverse Effects Levels of Polychlorinated Biphenyls								
			Effect	Concentration				Tissue/Diet
Impairment	**Endpoint**	**Toxin**	**Level**	**Diet**	**Tissue**	**References**	**Notes**	**BMF**
					Liver	**Bursian et al. 2006i, 2006ii**		
Reproduction	Kit survival reduced	PCBs	NOAEL	1.6 ug/g	3.083 ug/g			**1.9**
		PCBs	LOAEL	3.7 ug/g	3.133 ug/g			0.8
		PCBs	ETD	2.4 ug/g			calc. by STW from report data	
		PCBs	LC10	0.231 ug/g			estimated by regression analysis	
		PCBs	LC20	0.984 ug/g			estimated by regression analysis	
		TEQs	NOAEL	16.1 pg/g	55.9 pg/g			3.5
		TEQs	LOAEL	68.5 pg/g	218.4 pg/g			3.2
		TEQs	ETD	33.2 pg/g				
Deformities	Jaw lesion in 31-wk kits	PCBs	LOAEL	0.96 ug/g	1.698 ug/g			1.8
		TEQs	LOAEL	9.2 pg/g	40.2 pg/g			4.4
					Liver	**Bursian et al. 2006iii**	Reproduction effects measured included breeding success, whelping success, gestation length, and litter size.	
Reproduction	Reproduction and kit survivability	PCBs	NOAEL	1.7 ug/g	NA			
		TEQs	NOAEL	73 pg/g	NA			
Deformities	Jaw lesion in 27-wk kits	PCBs	NOAEL	0.83 ug/g	8.1 ug/g		Jaw lesions can lead to displaced and loose teeth (see Render et al 2000, 2001)	9.8
		PCBs	LOAEL	1.1 ug/g	16 ug/g			14.5
		TEQs	NOAEL	28 pg/g	28 pg/g			1.0
		TEQs	LOAEL	47 pg/g	75 pg/g			
					Liver	**Halbrook et al. 1999**	PCBs = Aroclor 1260 equivalents, in fish	
Reproduction	Litter size	PCBs	NOAEL	1009 ppb	<5 ppb			
			LOAEL	1360 ppb	7250 ppb		Results may have been confounded by presence of Hg in fish (Halbrook et al 1997)	5.3
					Adipose			
			NOAEL	1009 ppb	105860 ppb			105.0
			LOAEL	1360 ppb	128630 ppb			95.0

					Liver	**Martin et al. 2006**		
Metabolic	Plasma T4 increase in 6-wk kits	TPCBs	NOAEL	0.03 mg/kg	0.19 ug/kg			6.3
			LOAEL	0.83 mg/kg	4.38 ug/kg			5.3
		TEQs	NOAEL	3.4 ng/kg	3.1 pg/kg			0.9
			LOAEL	27.9 ng/kg	72 pg/kg			2.6
	Plasma T3 decrease in 27-wk juveniles	TPCBs	NOAEL	1.05 mg/kg	16.25 ug/kg		Many of our mink were only a month or two older than these juveniles	15.5
			LOAEL	1.69 mg/kg	17.79 ug/kg			10.5
		TEQs	NOAEL	47.6 ng/kg	74.6 pg/kg			1.6
			LOAEL	73.2 ng/kg	105.6 pg/kg			1.4
	Plasma retinol and retinyl esters decreased in 6-wk kits and 27-wk juveniles	TPCBs	NOAEL	1.05 mg/kg	Kits 10.54 ug/kg juvs 16.25 ug/kg			
			LOAEL	1.69 mg/kg	kits 18.8 ug/kg juvs 17.79 ug/kg			
		TEQs	NOAEL	47.6 ng/kg	Kits 152.4 pg/kg juvs 74.6 pg/kg			
			LOAEL	73.2 ng/kg	Kits 306.6 pg/kg juvs 105.6 pg/kg			
	Kidney retinyl esters reduced in 6-wk kits and 27-wk juveniles	TPCBs	NOAEL	0.03 mg/kg	kits 0.19 ug/kg juvs 2.57 ug/kg			
			LOAEL	0.83 mg/kg	kits 4.38 ug/kg juvs 8.14 ug/kg			

		TEQs	NOAEL	3.4 ng/kg	kits 3.1 pg/kg juvs 7.6 pg/kg			
			LOAEL	27.9 ng/kg	kits 71.2 pg/kg juvs 26.9 pg/kg			
		PCBs			**Liver**	**Restum et al. 1998**	Liver concentrations here are weighted averages of male and female values given in report	
Reproduction	Delayed estrus P-1 and F-1		NOAEL	0.0 ppm	P-1 70.7 ng/g F-1 83.2 ng/g			
			LOAEL	0.25 ppm	P-1 860 ng/g F-1 635 ng/g			P-1 =3.4 F-1 = 2.5
	Reduced mating P-1 females		NOAEL	0.5 ppm	923 ng/g			1.8
			LOAEL	1.0 ppm	1580 ng/g			1.6
	Reduced whelping P-1		NOAEL	0.0 ppm	70.7 ng/g			
			LOAEL	0.25 ppm	860 ng/g			3.4
	F-1 Kit mortality		NOAEL	0.25 ppm	635 ng/g			2.5
			LOAEL	0.5 ppm	967 ng/g			1.9
	F-1 Kit body weight		NOAEL	0.0 ppm	83.2 ng/g			
			LOAEL	0.25 ppm	635 ng/g			2.5
	F-2 Kit mortality		NOAEL	0.25 ppm	275 ng/g		No F-2 males at 0.5 ppb or above, or females at 1.0 ppb, survived to 3 weeks	1.1
			LOAEL	0.5 ppm	464 ng/g			0.9
	F-2 Kit body weight		NOAEL	0.25 ppm	275 ng/g			
			LOAEL	0.5 ppm	464 ng/g			
Deformity	Jaw lesions	PCBs	NOAEL	0 ppb	N/A	**Render et al. 2001**	Jaw lesions caused deformities and displacement of teeth	
			LOAEL	24 ppb				
Population	Kit mortality		NOAEL	0 ppb	N/A			
			LOAEL	24 ppb				

A-3. Adverse Effects Levels of Mercury								
			Effect	**Concentration (ppm or ug/g)**				**Tissue/Diet**
Impairment	**Endpoint**	**Toxin**	**Level**	**Diet**	**Tissue**	**References**	**Notes**	**BMF**
		Hg				**Aulerich et al. 1974**		
Population					**Brain**			**Brain**
	Adult mortality		LC100	5 ppm	19.9 ± 4.55 ppm		All mink died after 30-37 days after treatment started; treatment ended on 29th day.	4.0
		tHg in fish			**Liver**	**Dansereau et al. 1999**		**Liver**
Population	Adult mortality		LC60	1.0 ppm	96.6 ppm		In 1st generation females	96.6
Reproduction	Whelping reduced		NOAEL	0.1	28.2 ppm		In 1st and 2nd generation females	282.0
			LOAEL	0.5 ppm	80.4 ppm			168.0
					Brain			**Brain**
			LC60	1.0 ppm	*26.0 ug/g*		*Estimated by STW using average of Liver:Brain levels in other studies = 3.7*	
			NOAEL	0.1	*7.6 ug/g*			
			LOAEL	0.5 ppm	*21.6 ug/g*			
		tHg in env.		N/A	**Brain**	**Evans et al. 2000**		
					0.34 ± 0.24 ug/g		*Liver:Brain Ratio =*	4.5
					Liver			
					1.53 ± 1.24 ug/g			
					Fur			
					17.26 ug/g			
		MeHg in env.			**Brain**			
					0.26 ± 0.19 ug/g		*Liver:Brain Ratio =*	4.7
					Liver			
					1.21 ± 0.85 ug/g			
					Fur			
					11.25 ug/g			

Reproduction	Reduced litter Size	Hg in fish			**Liver**	**Halbrook et al. 1997**		Liver
				0.02 ppm	0.41 ppm			20.5
				0.05 ppm	0.61 ppm			12.2
				0.09 ppm	1.06 ppm			11.8
			NOAEL	0.15 ppm	1.93 ppm			12.9
			LOAEL	0.22 ppm	3.67 ppm			16.7
					Fur			
				0.02 ppm	3.79 ppm			
				0.05 ppm	7.43 ppm			
				0.09 ppm	7.71 ppm			
			NOAEL	0.15 ppm	13.44 ppm			
			LOAEL	0.22 ppm	19.03 ppm			
					Brain			
				0.02 ppm	*0.11 ppm*		*Estimated by STW using average of Liver:Brain levels in other studies = 3.4*	
				0.05 ppm	*0.18 ppm*			
				0.09 ppm	*0.31 ppm*			
			NOAEL	0.15 ppm	*0.56 ppm*			
			LOAEL	0.22 ppm	*1.06 ppm*			
Population	Adult mortality	MeHg			**Liver**	**Wobeser et al. 1976**	Report does not specify whether total or methyl Hg in tissues, but Hg in feed was MeHg.	**Liver**
			NOAEL	1.1 ppm	25.4 ppm			25.4
			LOAEL	1.8 ppm	21.3ppm			21.3
					Brain		Tissue levels measured for only two animals in each treatment group.	**Brain**
			NOAEL	1.1 ppm	8.2 ppm			7.5
			LOAEL	1.8 ppm	8.2 ppm			10.4
			LC40	1.8 ppm	8.2 ppm		*Liver:Brain Ratio (above)* =	3.1, 2.6
			Est. Threshold		10 ppm		*Liver:Brain Ratio (below)* =	4.5
	Sugg. Diag. Crit.				5ppm		Suggested diagnostic criterion	

	Classic Hg intoxication (neuro-toxicity, etc.)				**Liver**			**Liver**
			NOAEL	0.1 ppm	0.45 ppm			4.5
			LOAEL	1.1 ppm	25.4 ppm			23.1
					Brain			**Brain**
			NOAEL	0.1 ppm	0.1 ppm			1.0
			LOAEL	1.1 ppm	8.2 ppm			7.5
Population	Adult mortality	MeHg			**Brain**	**Wren et al. 1987i, ii**		**Brain**
			NOAEL	0.5 ug/g	N/A			
			LOAEL	1.0 ug/g	15.3 ug/g		Dietary levels are somewhat misleading because after unexpected mortalities within less than 3 months, the 1.0 ppm chow was used only every other day.	15.3
					Liver			**Liver**
			NOAEL	0.5 ug/g	N/A			
			LOAEL	1.0 ug/g	44.1 ug/g			44.1
			LC56	1.0 ug/g			9 of 16 in treatment group died	
			LC19	1.0 ug/g			3 of 16 in treatment group died	
Reproduction	Kits/female mated		NOAEL	1.0 ug/g	0.84 ug/g		Addition of PCBs decreased effect on #kits/female mated	
			LOAEL	1.0 ug/g	2.0 ug/g			
	Kit growth		NOAEL	1.0 ug/g	2.0 ug/g		Hg without PCBs had no effect	
			NOAEL	0.5 ug/g	1.32 ug/g			
			LOAEL	1.0 ug/g	0.84 ug/g		*Liver:Brain Ratio (above)* =	2.9

A-4. Adverse Effects Levels of Organochlorine Pesticides							
			Effect	Concentration			
Impairment	**Endpoint**	**Toxin**	**Level**	**Diet**	**Tissue**	**References**	**Notes**
Population	Adult Mortality	Heptachlor	NOAEL	50 mg/kg		**Aulerich et al. 1990**	
			LOAEL	100 mg/kg			
Reproduction	Reduced number of second matings	Lindane	LOAEL	1 mg/kg bw/d		**Beard et al. 1997, Beard and Rawlings 1998**	NOAEL for all is zero, as only one concentration was tested.
	Reduced whelping (incl. embryo loss)		LOAEL	1 mg/kg bw/d			
	Increase duration of pregnancy		LOAEL	1 mg/kg bw/d			
	Reduced litter size (2nd generation)		LOAEL	1 mg/kg bw/d			Effect "far greater" in 2nd gen. than in 1st gen.
	Reduced testis size		LOAEL	1 mg/kg bw/d			
Reproduction	Reduced number of second matings	PCP	LOAEL	1 mg/kg bw/d			NOAEL for all is zero, as only one concentration was tested.
	Reduced whelping (incl. embryo loss)		LOAEL	1 mg/kg bw/d			

	Increase duration of pregnancy		LOAEL	1 mg/kg bw/d			
	Increased prostate hyperplasia		LOAEL	1 mg/kg bw/d			
	Increased serum pro-gesterone	Carbofuran	LOAEL	0.05 mg/kg bw/d			NOAEL for all is zero, as only one concentration was tested.
Population	Adult Mortality	HCB	NOAEL	31 ppm		**Bleavins et al. 1984**	
			LOAEL	106 ppm			
Reproduction	Litter Size (total)		NOAEL	2 ppm,			
			LOAEL	31 ppm			
	Litter Size (live)		NOAEL	2 ppm,			
			LOAEL	31 ppm			
	Kit weight (birth)		NOAEL	0 ppm			
			LOAEL	1 ppm			
	Kit weight (3 & 6 wks)		NOAEL	1 ppm			
			LOAEL	2 ppm,			
	Kit mortality (3 & 6 wks)		NOAEL	0 ppm			
			LOAEL	1 ppm			
	Adult Mortality Female	Heptachlor	NOAEL	0 ppm		**Crum et al. 1993**	
			LOAEL	5 ppm			
			LC50	10.5 ppm			
	Adult Mortality Male		NOAEL	5 ppm			
			LOAEL	12 ppm			
	% Kits stillborn		NOAEL	5 ppm			
			LOAEL	12 ppm			
	Kit weight (birth)		NOAEL	5 ppm			
			LOAEL	12 ppm			
	Kit weight (3 & 6 wks)		NOAEL	0 ppm			
			LOAEL	5 ppm			
			NOAEL	5 ppm			

	Kit mortality (3 wks)		LOAEL	12 ppm			
	Kit mortality (17 wks)	HCB			**Adipose**	**Rush et al. 1983**	Tissue levels not given here were all ND (not detected)
			NOAEL	0 ppm	36 ppb		
			LOAEL	1 ppm	95 ppb		
				5 ppm	626 ppb		
					Brain		
				5 ppm	36 ppb		
					Liver		
				1 ppm	*5.6 ppb*		*Estimated by STW, based on Liver:Adipose at 5 ppb dietary level.*
				5 ppm	37 ppb		
					Kidney		
				1 ppm	4 ppb		
				5 ppm	15 ppb		
					Muscle		
				1 ppm	1 ppb		
				5 ppm	8 ppb		

A-5. Adverse Effects Levels of Multiple Polyhalogenated Hydrocarbons								
			Effect	Concentration				Tissue/Diet
Impairment	Endpoint	Toxin	Level	Diet	Tissue	References	Notes	BMF
					Liver			
Reproduction	Gestation length	PCBs	NOAEL	0.015 ug/g	90 pg/g	**Heaton et al. 1995, Tillit et al. 1996**	Mink were fed fish from Saginaw Bay which contained PCBs, dioxins (CCD), and furans (CDF) (and other BCCs).	6
		CDDs		42 pg/g	617 pg/g			14.7
		CDFs		1 pg/g	6 pg/g			6
		PCBs	LOAEL	.72 ug/g	2190 pg/g			3
		CDDs		60 pg/g	2626 pg/g		Values for PCBs, CDDs, and CDFs calculated by STW from Table 1 of Tillet et al (1996).	43.8
		CDFs		13 pg/g	335 pg/g			25.8
		TEQs	NOAEL	0.9 pg/g	17.5 pg/g			10.7
		TEQs	LOAEL	22.4 pg/g	208.3 pg/g			16.7
	Ave. litter size	PCBs	NOAEL	1.53 ug/g	3050 pg/g		Values for TEQs calculated by STW from Table 8 of Tillet et al (1996) using WHO TEQs from van den Berg et al (1998), per D. Tillitt's suggestion (2006, pers. comm.).	2
		CDDs		46 pg/g	2329 pg/g			50.6
		CDFs		20 pg/g	551 pg/g			27.6
		PCBs	LOAEL	2.56 ug/g	6270 pg/g			2.4
		CDDs		84 pg/g	3002 pg/g			35.7
		CDFs		43 pg/g	914 pg/g			21.2
		TEQs	NOAEL	43.4 pg/g	321.6 pg/g			14.5
		TEQs	LOAEL	86.8 pg/g	560.1 pg/g			11.1
	No. live kits	PCBs	NOAEL	1.53 ug/g	3050 pg/g			
		CDDs		46 pg/g	2329 pg/g			
		CDFs		20 pg/g	551 pg/g			
		PCBs	LOAEL	2.56 ug/g	6270 pg/g			
		CDDs		84 pg/g	3002 pg/g			

		CDFs		43 pg/g	914 pg/g			
		TEQs	NOAEL	43.4pg/g	321.6 pg/g			
		TEQs	LOAEL	86.8pg/g	89560 pg/g			
	Kit body wt birth	PCBs	NOAEL	.72 ug/g	2190 pg/g			
		CDDs		60 pg/g	2626 pg/g			
		CDFs		13 pg/g	335 pg/g			
		PCBs	LOAEL	1.53 ug/g	3050 pg/g			
		CDDs		46 pg/g	2329 pg/g			
		CDFs		20 pg/g	551 pg/g			
		TEQs	NOAEL	22.4 pg/g	208.3 pg/g			
		TEQs	LOAEL	43.4 pg/g	321.6 pg/g			
	Kit body wt 3 & 6 wks	PCBs	NOAEL	.015 ug/g	90 pg/g			
		CDDs		42 pg/g	617 pg/g			
		CDFs		1 pg/g	6 pg/g			
		PCBs	LOAEL	.72 ug/g	2190 pg/g			
		CDDs		60 pg/g	2626 pg/g			
		CDFs		13 pg/g	335 pg/g			
		TEQs	NOAEL	0.9 pg/g	17.5 pg/g			
		TEQs	LOAEL	22.4 pg/g	208.3 pg/g			
	Kit survival 3 & 6 wks	PCBs	NOAEL	.015 ug/g	90 pg/g			
		CDDs		42 pg/g	617 pg/g			
		CDFs		1 pg/g	6 pg/g			
		PCBs	LOAEL	.72 ug/g	2190 pg/g			
		CDDs		60 pg/g	2626 pg/g			
		CDFs		13 pg/g	335 pg/g			
		TEQs	NOAEL	0.9 pg/g	17.5 pg/g			
		TEQs	LOAEL	22.4 pg/g	208.3 pg/g			

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To avoid confusion, we have used i, ii, iii, here when referring to multiple papers in the same year, as the report may have referred to them in a different order as a, b, c.

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Appendix 5. Mink jaw deformities in relation to total PCBs and TEQs in their tissues. Mink 17 had multiple proliferative squamous cysts from incisor to molar on the mandible and both maxillae. na = not analyzed, nd = not detected, us = unsuitable sample.

AOC-Lakeshore				**Deformities**		
	Total PCB Liver	**Adipose TEQ**	**Liver TEQ**		**Left**	**Right**
Mink	ng/g ww	pg/g ww	pg/g ww	**Mandible**	**Maxilla**	**Maxilla**
17	5870.8	338.98	21.26	Yes	Yes	Yes
20	86.4	na	Na			
21	682.0	nd	3.50	us	No	No
22	2388.8	22.38	47.62			
38	213.6	10.79	0.30			
39	35.3	1.24	1.17			
41	32.2	3.59	Nd			
56	14.7	7.66	nd	us	No	No
57	153.4	10.23	nd			
58	96.4	16.07	nd			
AOC-Inland						
1	8.5	0.03	0.10	No	No	No
23	18.1	na	na			
24	27.3	3.99	0.64			
43	29.7	1.63	nd			
44	13.4	0.31	nd			
45	28.8	3.23	nd			
59	10.6	3.38	nd			
60	12.8	3.99	nd			
61	64.2	8.96	nd	No	No	Us
62	250.5	12.57	4.16			
63	554.4	9.13	nd	No	No	No
Out of AOC-Lakeshore						
46	229.8	19.63	2.09	No	No	No
47	43.4	9.35	nd			
48	184.8	na	na			
49	755.0	38.29	38.31	No	No	No
50	171.2	9.22	nd			
51	411.3	30.14	nd			
52	69.1	5.43	nd			
53	13.6	5.30	0.92	No	No	No
54	66.7	3.54	nd			
55	360.0	18.03	nd			

Out of AOC-Inland

3	11.7	4.78	0.00			
5	10.0	na	na			
10	8.0	0.35	0.01			
11	27.8	na	na			
14	7.0	nd	0.03	us	No	No
30	45.0	na	na	No	No	No
31	31.4	na	na			
32	14.5	na	na			
33	19.3	na	na	us	No	No
34	31.7	0.79	nd			