CONCENTRATIONS AND PROFILES OF POLYCHLORINATED BIPHENYLS, -DIBENZO-*p*-DIOXINS AND -DIBENZOFURANS IN LIVERS OF MINK FROM SOUTH CAROLINA AND LOUISIANA, U.S.A.

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Abstract. In South Carolina, U.S.A., mink have been reintroduced from two apparently healthy populations to areas where populations have existed in the past but have been extirpated. High mortality was observed during transport of mink from the source populations. In order to elucidate the potential effects of dioxin-like compounds on the survival and reproduction of mink, concentrations of total polychlorinated biphenyls (PCBs), *p,p′*-DDE, dioxin-like PCBs, polychlorinated dibenzo-*p*-dioxins (PCDDs), and dibenzofurans (PCDFs) were measured in livers of mink collected from the source populations in South Carolina and Louisiana. Concentrations of total 2,3,7,8-tetrachlorodibenzo-*p*-dioxin equivalents (TEQs) for the South Carolina and Louisiana mink were 21 and 14 pg g^{-1}, wet wt., respectively. PCB and TEQ concentrations were close to the threshold values that can, under laboratory conditions, elicit toxic effects in ranch mink. Therefore, any additional exposures of these populations to TEQs might adversely affect their populations.

Keywords: aquatic mammal, Mink, PCBs, PCDDs, PCDFs, TEQs

1. Introduction

In recent years, mink (*Mustela vison*) populations in South Carolina, U.S.A., have been declining in certain areas of the state, particularly along the coastal plain. Flood tide surveys, conducted by the South Carolina Department of Natural Resources, indicated an absence of mink in areas of the northern coastal plain where habitat appears suitable and available (Baker, 2000). Trapper observations, although anecdotal, suggest that mink populations were once abundant in areas north of Charleston, where populations are no longer observed (Baker, 2000). Concern for this valuable wildlife species initiated a reintroduction effort to attempt to re-establish the population in the northern coastal plain. The source populations for the reintroduction effort were selected from areas of the southeastern United States where apparently healthy populations exist. Mink were translocated from

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the southern coastal marshes of South Carolina and Acadia Parish in Louisiana, and introduced at Cape Romain National Wildlife Refuge in South Carolina. Initial attempts to transport the mink resulted in extremely high mortality and led to concerns regarding the health of these mink. Because mink are known to be sensitive to the toxic effects of dioxin-like compounds, a survey of these compounds and their possible effects on survival and reproduction were conducted.

Mink are semi-aquatic, opportunistic carnivores that prey on various invertebrates, fish, amphibians, birds, and mammals. Because of their position as a top predator, mink readily accumulate lipophilic contaminants, including polychlorinated biphenyls (PCBs), polychlorinated dibenzo-*p*-dioxins (PCDDs), and polychlorinated dibenzofurans (PCDFs). Several laboratory studies have shown mink to be extremely sensitive to planar halogenated hydrocarbons (PHHs) (Aulerich *et al*., 1973; Platonow and Karstad, 1973; Aulerich and Ringer, 1977; Ringer, 1981; Hochstein *et al*., 1988; Heaton *et al*., 1995; Tillitt *et al*., 1996; Restum *et al*., 1998). PCBs have been shown to have numerous deleterious effects in mink, including impaired reproductive success. Of particular concern are the dioxin-like PCBs, which include the non-*ortho* and mono-*ortho* substituted congeners (Aulerich *et al*., 1985; Kihlstrom *et al*., 1992). These dioxin-like PCBs are co-planar, as are PCDDs and PCDFs, and can therefore elicit a response via the aryl hydrocarbon (Ah) receptor (Giesy and Kannan, 1998).

Environmental exposure to PCBs has previously been explored as a potential cause for mink population declines in South Carolina, North Carolina, and Georgia (Osowski *et al*., 1995). However, concentrations of PCDDs and PCDFs in wild mink are largely unknown. In this study, the concentrations of *p,p′*-DDE, PCBs, PCDDs, and PCDFs are measured in livers of mink collected from the source populations for the reintroduction effort in South Carolina. Body burdens of PCBs, PCDDs, and PCDFs in mink may provide insight for these recent management attempts to re-establish natural mink populations. The objective of this study was to characterize exposure to dioxin-like toxicity in mink from the South Carolina and Louisiana source populations in an effort to determine if these contaminants may be affecting the health of these animals, and thereby contributing to the difficulties with the reintroduction effort.

2. Materials and Methods

2.1. Sample collection

In December 1999, 14 wild mink were captured from Acadia Parish, Louisiana (Figure 1a). In May 2000, 10 mink were collected from the tidal brackish marshes of the Ashepoo and Edisto Rivers of South Carolina's ACE Basin (Figure 1b). Animals were euthanized and necropsied in the field. Tissues were wrapped in aluminum foil and frozen in plastic bags until contaminant analysis. A subset of

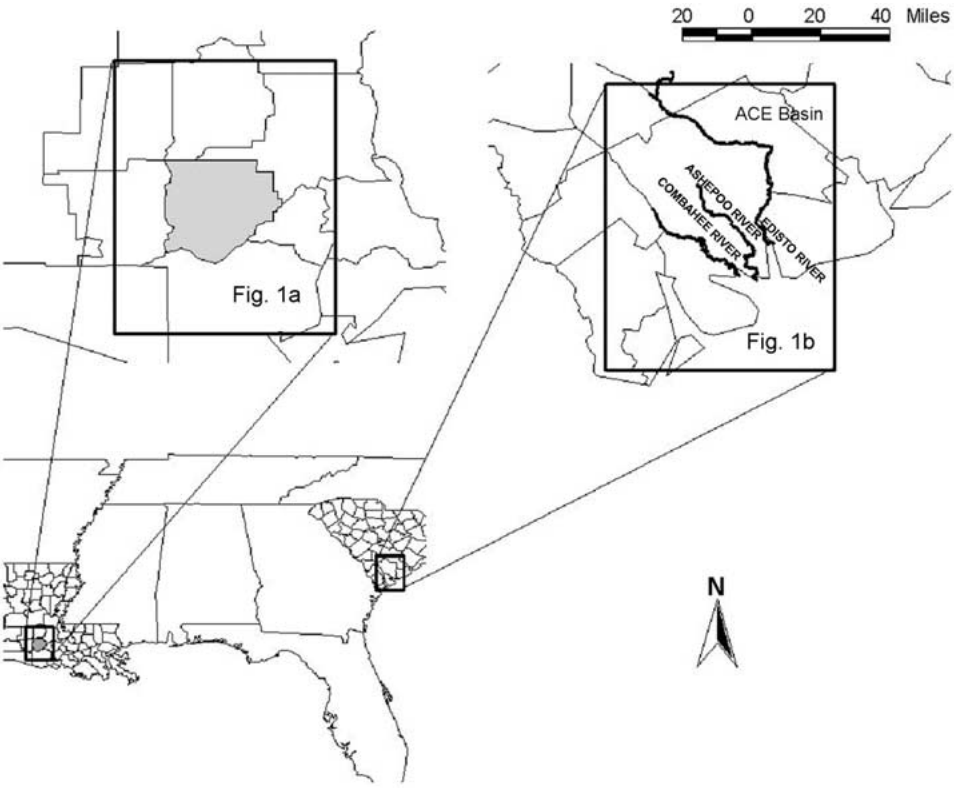

Figure 1. Map of collection sites. (a) Acadia Parish, Louisiana; (b) South Carolina's ACE Basin (Ashepoo, Combahee, Edisto River Basin).

mink samples was selected for analysis for PCDD/DFs and dioxin-like PCB congeners. Six mink livers were chosen from each population for analysis. Of the 12 samples selected, all were male except for one pregnant female (South Carolina). Canine teeth were extracted from each animal for cementum analysis to determine age. The results indicated that all animals were juveniles. It has been suggested that the mild climate of the southern United States may inhibit annual cementum deposition, causing all mink to be classified as juvenile (O. E. Baker, pers. comm.). Thus, the exact ages of the mink are unknown.

2.2. Analytical Methods

Polychlorinated biphenyls and 2,3,7,8-substituted congeners of PCDDs and PCDFs were analyzed following methods described elsewhere (Kannan *et al.*, 2000b; Senthilkumar *et al.*, 2001). Liver samples were freeze-dried and moisture content determined. Samples were extracted using a Soxhlet apparatus for 10–15 hr in dichloromethane. Details of the analytical procedure have been reported previously. Briefly, after the extraction, samples were concentrated to 10 mL using a Kuderna-

Danish (K-D) concentrator and the solvent transferred to n-hexane. Lipid content was determined gravimetrically from an aliquot of the extract. Seventeen ^{13}C-labeled 2,3,7,8-substituted tetra-, penta-, hexa-, hepta-, and octa-CDD and CDF congeners and dioxin-like PCBs (IUPAC Nos. 81, 77, 126, 169, 105, 114, 118, 123, 156, 157, 167, 189) were spiked into hexane extracts prior to sulfuric acid treatment. The hexane layer was rinsed twice with hexane-washed water, and dried by passing through anhydrous sodium sulfate in a glass funnel. The solution was concentrated to 2 mL and sequentially subjected to silica gel, alumina and silica gel impregnated activated carbon column chromatography. Extracts were passed through activated silica gel (activated at 130 °C for $3\frac{1}{2}$ hr) packed in a glass column (Wakogel, silica gel 60; 2 g) and eluted with 130 mL of hexane, which contained PCDD/DFs and PCBs. The hexane extract was K-D concentrated and passed through activated alumina (190 °C for 3 hr) column (Merck-Alumina oxide, activity grade 1; 5 g) and eluted with 30 mL of 2% dichloromethane in hexane as the first fraction, which contained several *ortho*-substituted PCBs. This fraction was saved for the analysis of total PCBs and DDE. The second fraction eluted with 30 mL of 50% dichloromethane in hexane contained PCDD/DFs and dioxin-like PCBs, which was purged under a gentle stream of nitrogen to dryness and passed through silica gel impregnated activated carbon column (0.5 g) to further separate mono- and di-*ortho* PCBs from non-*ortho* PCBs and PCDD/DFs. The first fraction eluted with 25 mL of 25% dichloromethane in hexane contained mono- and di-*ortho* PCBs. The second fraction eluted with 250 mL toluene contained non-*ortho* PCBs and PCDD/DFs. For the analysis of PCDDs, PCDFs and dioxin-like PCBs, sample extracts were analyzed by a high-resolution gas chromatograph interfaced with a high-resolution mass spectrometer (HRGC-HRMS). Procedural blanks were analyzed to check for interferences. Octachlorodibenzo-*p*-dioxin (OCDD) was found at approximately 1 pg g^{-1} in procedural blanks. The values obtained for OCDD were not corrected for blank concentrations, but the detection limit for OCDD was set at 3 pg g^{-1}, wet wt. We have participated in an interlaboratory comparison study of dioxins in environmental samples and our results are within the acceptable limits of certified values.

2.3. Identification and quantification

Identification and quantification of 2,3,7,8-substituted congeners of PCDD/DFs and dioxin-like PCBs were performed using a HRGC (Hewlett Packard 6890 Series) coupled with a HRMS (Micromass Autospec - Ultima). The HRMS was operated in an electron impact, selected ion monitoring mode at a resolution R > 10 000 (10% valley). Separation was achieved using a DB-5 (J&W Scientific; 0.25 mm i.d. × 60 m length) and a DB-17 column (J&W Scientific; 0.25 mm i.d. × 60 m length). Details of the oven temperature program are given elsewhere (Senthilkumar *et al.*, 2001). Prior to injection, ^{13}C-labelled 1,2,3,4-TeCDD and 1,2,3,7,8,9-HxCDD were added as injection recovery standard. Mean (range) recoveries of

spiked internal standard through the entire analytical procedure were 74% (60–95%). Concentrations of dioxin-like PCBs refer to the sum of 14 non-, mono- and di-ortho substituted PCB congeners. PCB congeners are represented by the IUPAC numbers.

Total PCB concentrations were determined in extracts from silica gel columns by using a gas chromatograph (Perkin Elmer series 600) equipped with ^{63}Ni electron capture detector (GC-ECD). A fused silica capillary column coated with DB-5MS [(5%-phenyl)-methylpolysiloxane, 30 m × 0.25 mm i.d.; J&W Scientific, Folsom, CA, U.S.A.], having a film thickness of 0.25 μm, was used. PCB congeners were identified against a standard mixture containing 100 congeners of known composition and content. Further details of PCB analysis are reported elsewhere (Kannan *et al*., 2000b).

2.4. Determination of toxic equivalents (TEQS)

PCBs, PCDDs, and PCDFs are found in the environment as complex mixtures. The individual isomers examined in this study are thought to elicit their toxic response in mink through the same initial mode of action-binding to the Ah receptor. The relative toxicity of each compound is related to the most toxic compound, the 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). Relative potencies of the individual congeners are normalized to the potency of TCDD using toxic equivalency factors (TEFs), calculated from numerous *in vivo* and *in vitro* studies (Van den Berg *et al*., 1998). In this study, TEQs were determined in order to estimate the relative toxicity of each class of contaminants. Concentrations and TEQs are reported as wet weight unless otherwise indicated.

3. Results

3.1. Concentrations

Concentrations of total PCBs in livers of mink from South Carolina and Louisiana ranged from 133–413 (mean, 263) and 65–238 (mean, 139) ng g^{-1}, wet weight, respectively (Table I). Concentrations of total PCBs in livers of mink from South Carolina were significantly greater than those from Louisiana ($p = 0.03$, two-tailed *t*-test).

Livers of mink from South Carolina contained greater mean concentrations of non-*ortho* and mono-*ortho* PCBs than did those from Louisiana (Table II). The mean concentrations of the non-*ortho* congeners 77, 81, 126 and 169 totaled 53.7 pg g^{-1}, wet wt., in the South Carolina population, with a range of 31.8–111 pg g^{-1}. The lower end of the range is the concentration in the liver of the pregnant female (the only female examined in this study). The mean concentration of non-*ortho* congeners in the Louisiana mink ranged from 18.9–57.9 pg g^{-1}

TABLE I

Concentrations (ng g^{-1}, wet wt.) of total PCBs and *p,p′*-DDE in livers of mink from South Carolina and Louisiana

	Louisiana (n = 6)		South Carolina (n = 6)	
	Mean±SD	Range	Mean±SD	Range
Lipid (%)	3.8±0.45	3–4.4	4.97±0.89	3.6–6.4
Moisture (%)	68.2±1.07	67–70	67.80±2.27	63.0–70
PCBs	139.0±52.2	65–238	263.00±99	133.0–413
DDE	78.7±33.8	32–128	16.20±13.5	1.2–16

(mean, 33.2 pg g^{-1}, wet wt.). PCB 126 was the predominant non-*ortho* PCB congener, with mean concentrations of 43.3 pg g^{-1} in the South Carolina mink and 25.5 pg g^{-1} in the Louisiana mink. In general, the concentrations of non-*ortho* PCB congeners in mink were in the order: 126 » 169 > 77 > 81. Hexa-, hepta and octa-chlorobiphenyls were the predominant congeners found in mink livers from both the locations.

Among mono-*ortho* PCBs, congeners 118 (2,3′,4,4′,5-pentaCB), 156 (2,3,3′,4,4′,5-hexaCB) and 105 (2,3,3′,4,4′-pentaCB) were the predominant congeners in mink livers. While concentrations of PCB congener 156 were less than those of 118 and 105 in mink from Louisiana, this congener accounted for a major portion of mono-*ortho* PCBs in South Carolina mink.

Mean concentrations of total PCDDs in livers were comparable between the South Carolina and Louisiana minks (Table II). There was a similar pattern in the composition of PCDD congeners in both mink populations, with the more highly chlorinated congeners reaching greater concentrations. 1,2,3,4,6,7,8,9-OCDD was the predominant congener in all of the South Carolina mink livers and almost all of the Louisiana mink livers, with concentrations ranging from 73–519 pg g^{-1} (mean, 323) and 18–733 pg g^{-1} (mean, 326), respectively. 2,3,7,8-TCDD was found in all the mink livers analyzed. Concentrations of PCDFs were 16 and 42 fold less than that of the PCDDs in Louisiana and South Carolina minks, respectively. The Louisiana mink had higher mean concentrations of all examined PCDFs isomers compared to that of the South Carolina samples. 2,3,4,7,8-pentachlorodibenzo-*p*-furan (PnCDF) and 2,3,4,6,7,8-hexachlorodibenzofuran (H × CDF) were the predominant congeners in both populations.

p,p′-DDE was also found in all the mink livers analyzed (Table I). Concentrations of *p,p′*-DDE in livers of mink from South Carolina were five-fold less than those from Louisiana. Concentrations of *p,p′*-DDE in mink livers from South Carolina were approximately five-fold less than those reported in 1989–1991 (Osowski *et al.*, 1995).

TABLE II

Concentrations of dioxin-like PCBs, PCDDs, PCDFs and their corresponding toxic equivalencies (pg g^{-1}, wet weight)

	TEF	South Carolina (n = 6)				Louisiana (n = 6)			
		Conc.[a]	(Range)	TEQ	(Range)	Conc.[a]	(Range)	TEQ	(Range)
Non-ortho PCBs									
81	0.0001	0.90	(0.61–1.34)	<0.001	(<0.001)	1.31	(0.32–3.93)	<0.001	(<0.001)
77	0.0001	3.46	(1.62–5.18)	<0.001	(<0.001–0.001)	3.75	(1.55–8.42)	<0.001	(<0.001)
126	0.1	43.28	(26 –86)	4.33	(2.63 –8.61)	25.5	(15 –45)	2.55	(1.53 –4.5)
169	0.01	6.03	(1.52–18.5)	0.06	(0.02 –0.18)	2.68	(0.9 –4.7)	0.03	(0.01 –0.05)
Sum of non-ortho PCBs		**53.67**		**4.39**		**33.24**		**2.58**	
Mono-ortho PCBs									
105	0.0001	962	(343 –2730)	0.10	(0.03 –0.27)	770	(255 –2080)	0.08	(0.03 –0.21)
114	0.0005	5.38	(0.34–29)	0.002	(<0.001–0.01)	45.2	(6.1 –170)	0.02	(0.005 –0.09)
118	0.0001	1965	(808 –5750)	0.20	(0.08 –0.57)	1357	(533 –1910)	0.14	(0.05 –0.19)
123	0.0001	<10	<10	<0.001	<0.001	<10	<10	<0.001	<0.001
156	0.0005	2498	(344 –6910)	1.25	(0.17 –3.45)	667	(166 –1180)	0.33	(0.08 –0.59)
157	0.0005	660	(105 –2000)	0.33	(0.05 –1)	206	(44 –307)	0.10	(0.02 –0.15)
167	0.00001	512	(103 –1620)	0.005	(<0.001–0.02)	264	(67 –603)	0.002	(<0.001–0.01)
189	0.0001	225	(11.5–642)	0.023	(<0.001–0.06)	<10	<10	<0.001	<0.001
Sum of mono-ortho PCBs		**6827.38**		**1.91**		**3309.2**		**0.672**	

[a] Conc. = concentration.

TABLE II
(continued)

	TEF	South Carolina (n = 6)				Louisiana (n = 6)			
		Conc.[a]	(Range)	TEQ	(Range)	Conc.[a]	(Range)	TEQ	(Range)
PCDD isomers									
2,3,7,8-D	1.0000	1.29	(0.85–1.68)	1.291	(0.85 –1.68)	1.03	(0.4 –1.7)	1.03	(0.42 –1.69)
1,2,3,7,8-D	1.0000	5.52	(2.2 –8.9)	5.518	(2.21 –8.93)	1.37	(0.33–2.1)	1.37	(0.33 –2.05)
1,2,3,4,7,8-D	0.1000	9.65	(6.8 –13)	0.965	(0.68 –1.35)	3.03	(0.52–6.7)	0.30	(0.05 –0.67)
1,2,3,6,7,8-D	0.1000	34.57	(21 –44)	3.457	(2.11 –4.45)	25.6	(2.1 –87)	2.56	(0.21 –8.73)
1,2,3,7,8,9-D	0.1000	2.74	(2.1 –3.7)	0.274	(0.21 –0.37)	0.72	(0.16–1.1)	0.07	(0.02 –0.10)
1,2,3,4,6,7,8-D	0.0100	88.77	(52 –116)	0.888	(0.52 –1.16)	90.8	(5.67–216)	0.91	(0.06 –2.16)
OCDD	0.0001	323.11	(73 –519)	0.032	(0.01 –0.05)	326	(18 –733)	0.03	(<0.001–0.07)
Sum of PCDD isomers		**465.7**		**12.4**		**448.8**		**6.28**	
PCDF isomers									
2,3,7,8-F	0.1000	0.78	(0.18–1.7)	0.078	(0.02 –0.17)	0.63	(0.08–1.6)	0.06	(0.01 –0.16)
1,2,3,7,8-F	0.0500	0.16	(0.06–0.31)	0.008	(<0.001–0.02)	0.19	(0.03–0.36)	0.01	(<0.001–0.02)
2,3,4,7,8-F	0.5000	3.21	(1.7 –4.3)	1.606	(0.86 –2.13)	5.06	(0.83–14)	2.53	(0.42 –6.8)
1,2,3,4,7,8-F	0.1000	1.05	(0.65–1.32)	0.105	(0.06 –0.13)	3.66	(0.27–14)	0.37	(0.03 –1.38)
1,2,3,6,7,8-F	0.1000	1.71	(1.12–2.1)	0.171	(0.11 –0.21)	4.98	(0.48–20)	0.50	(0.05 –2)
2,3,4,6,7,8-F	0.1000	2.62	(2.0 –3.7)	0.262	(0.2 –0.37)	7.82	(0.87–31)	0.78	(0.09 –3.1)
1,2,3,7,8,9-F	0.1000	0.15	(0.03–0.44)	0.015	(<0.001–0.04)	0.29	(0.05–0.75)	0.03	(0.01 –0.08)
1,2,3,4,6,7,8-F	0.0100	0.74	(0.56–1.0)	0.007	0.001 –0.01	2.64	(0.28–10)	0.03	(<0.001–0.1)
1,2,3,4,7,8,9-F	0.0100	0.17	(0.07–0.4)	0.002	0.001 –0.004	0.64	(0.07–2.5)	0.01	(<0.001–0.03)
OCDF	0.0001	0.51	(0.23–0.94)	<0.001	<0.001	2.12	(0.17–6.83)	<0.001	<0.001
Sum of PCDF isomers		**11.10**		**2.25**		**28.01**		**4.31**	
PCDD/DF congeners		**477**		**15**		**477**		**11**	
Total TEQs				**21.0**				**13.8**	

[a] Conc. = concentration.

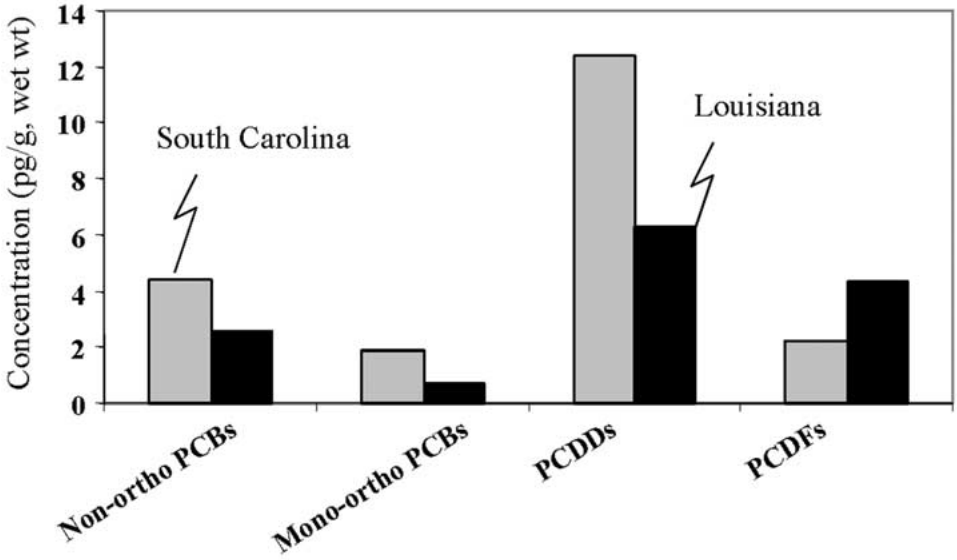

Figure 2. Comparison of TEQ concentrations contributed by dioxin-like PCBs, PCDDs, and PCDFs in mink livers.

3.2. Concentrations of TEQs

The concentrations of TEQs estimated for PCDDs, PCDFs and dioxin-like PCBs in mink livers from South Carolina and Louisiana were 21 and 14 pg g^{-1}, wet wt., respectively (Table II). PCDDs were the major contributors to total TEQs in mink livers accounting for 45% of the TEQs in Louisiana mink and 59% in South Carolina mink (Figure 2). Contribution by individual PCDD congeners to TEQ concentrations varied between the locations. 1,2,3,6,7,8-HxCDD was the major contributor to TEQs in mink from Louisiana, whereas 1,2,3,7,8-PnCDD was the major contributor to TEQs in South Carolina mink (Figure 3). Contribution of PCDFs to TEQs is great in Louisiana mink accounting for 31% of the total TEQs (Figure 2). Among PCDFs, congener 2,3,4,7,8-PnCDF accounted for 59–71% of the PCDF-TEQs (Figure 4).

Non-*ortho* coplanar PCBs accounted for 20% of the total TEQs. PCB 126 accounted for 99% of the TEQs contributed by non-*ortho* congeners. Mono-*ortho* PCBs contributed 5–10% of the total TEQs.

4. Discussion

The sensitivity of mink to PCBs was first shown in 1973, and further research confirmed that mink are one of the most sensitive mammals to the toxic effects of PCBs and other PHHs. PHH contamination, particularly from PCBs, has been implicated in the decline of wild mink and otter populations in both North America

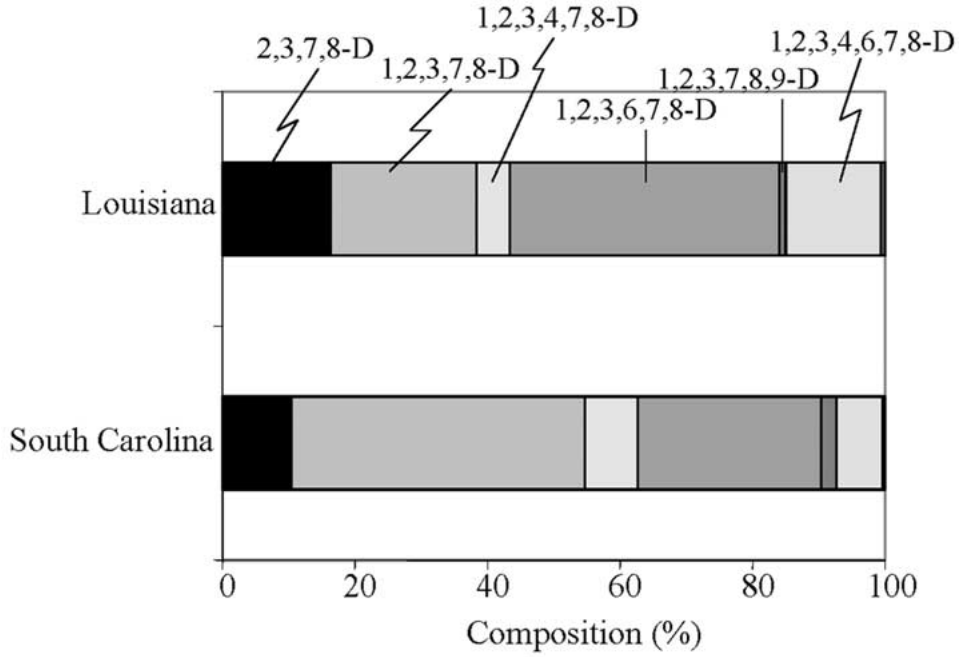

Figure 3. Relative contributions of PCDD isomers to TEQs in mink livers.

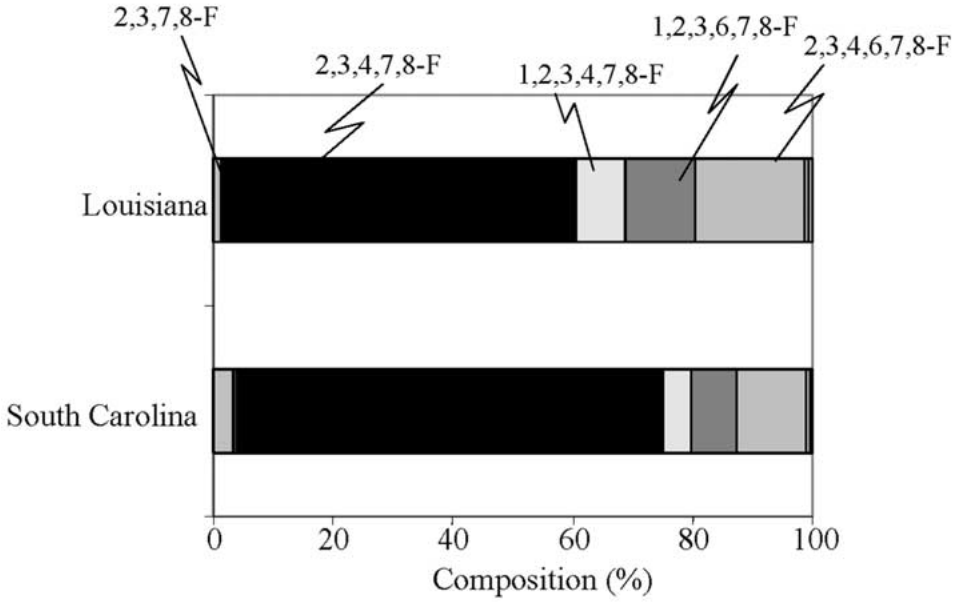

Figure 4. Relative contribution of PCDF isomers to TEQs in mink livers.

and Europe. As such, it is important to determine if PCB/PHH contamination could be limiting mink populations in South Carolina and Louisiana.

4.1. Comparison to laboratory studies

Mean concentrations of total PCBs in liver of South Carolina and Louisiana mink, 263 and 139 ng g^{-1}, respectively, are generally less than concentrations associated with effects in laboratory studies. Total PCB concentrations of 4 μg g^{-1} in mink livers have been associated with lethality in ranch mink fed PCB contaminated fish (Aulerich *et al*., 1973). Captive mink fed meat contaminated with high levels of Aroclor 1254 (3.57 ppm) resulted in lethality, with average PCB residues in liver of 11.99 μg g^{-1} (Platonow and Karstad, 1973). Mink fed a lower dose, 0.64 ppm, accumulated PCB residues in the liver with concentrations that ranged from 0.87 to 1.3 μg g^{-1}, which corresponded with reproductive failure. Studies using environmentally weathered compounds have also been conducted. Mink fed diets that included 40% Saginaw Bay carp resulted in mean PCB residues in liver of 6.27 μg g^{-1} (Tillitt *et al*., 1996). Kits born to females in that treatment group were stillborn or died within 24 hr (Heaton *et al*., 1995).

Based on the analysis of data from several laboratory and semi-field exposure studies of aquatic mammals, threshold concentration for total PCBs in livers of aquatic mammals to elicit physiological effects has been estimated to be 8.7 μg g^{-1}, lipid wt. (Kannan *et al*., 2000a). On a lipid normalized basis, concentrations of total PCBs in mink livers from South Carolina and Louisiana were 2.4–11.5 and 1.6–6 μg g^{-1}, lipid wt., respectively. In terms of TEQs, a threshold concentration for PHHs in livers of mink that were reported to cause reproductive effects in mink was 60 TEQ pg g^{-1}, wet wt. (Kannan *et al*., 2000a). When factoring in uncertainty factors, a more conservative estimate of 32 TEQ pg g^{-1} has been suggested (Kannan *et al*., 2000a). The measured concentrations of TEQs in the livers of the mink from the South Carolina and Louisiana populations were close to or above the threshold values determined to elicit physiological effects.

4.2. Comparison to wild mink populations

Although there have been a great number of laboratory studies conducted examining the effects of PHHs on laboratory mink, comparatively few data exist on the levels of PHHs and associated impacts on wild mink populations. Furthermore, it is difficult to make a direct comparison of PCB levels in this study to those reported in other studies because of differences in analytical methods and in quantification techniques. In this discussion, the method of quantifying total PCBs is given to remind the reader that direct comparisons are not necessarily appropriate when different quantification methods are employed.

The concentrations of PCBs in the South Carolina and Louisiana mink were greater than those reported for two areas of northwestern North America (Table III).

TABLE III

The range in mean concentrations of total PCBs (μg g^{-1} wet weight) in mink livers reported in North America

Location	Σ PCBs	Year(s) of mink collection	References
South Carolina	0.13 –0.33[a]	2000	This study
Louisiana	0.07 –0.24	1999	This study
St. Maurice River,	1.46 –2.39[b]	1991–1992	Champoux, 1996
LaTuque, Quebec		1994–1995	
South Carolina	0.042–1.46	1989–1001	Osowski *et al*,.1995
Georgia	ND –1.433		
North Carolina	ND –12.78		
British Columbia	<0.001–0.024	1990–1992	Elliott *et al*., 1999
Washington	0.067–0.38		
Oregon	0.039–0.36		
New York	0.1 –0.6[c]	1982–1984	Foley *et al*., 1988
Southern Ontario, Great Lakes	0.034–1.8	1988–1989	Haffner *et al*., 1998
Oregon	0.52 –3.5	1978–1979	Henny *et al*., 1981
Rural western Maryland	0.62 –2.4[d]	1978–1979	O'Shea *et al*., 1981
Northwest Territories	0.007–0.073	1991–1993	Poole *et al*., 1998
British Columbia	0.07 –0.08[e]	1994–1996	Harding *et al*., 1999

[a] Calculated as the sum of congeners.
[b] Both liver and muscle were analyzed.
[c] Calculated as Aroclor 1254 and 1260.
[d] Calculated as Aroclor 1254.
[e] Calculated as Aroclor 1260.

In the western Northwest Territories, the maximum community mean PCB concentration in mink livers was 73.07 ng g^{-1}, where total PCBs were calculated as the sum of 43 congeners (Poole *et al*., 1998). The mean PCB concentration, calculated as the sum of 28 congeners, in mink livers from the Columbia and Fraser River drainage basins was 102 ng g^{-1} (Elliott *et al*., 1999).

In other areas of North American, there have been declines in mink populations in areas associated with elevated PCB levels, indicating contaminants may be potentially affecting mink fitness in these areas. In general, these levels are greater than reported here. Concentrations of PCBs in mink livers collected from the Lower Columbia River in Oregon ranged from 0.5 to 3.5 μg g^{-1} (Henny *et al*., 1981); the method of quantifying total PCBs is not mentioned in this study. In Maryland, mean PCB concentrations (calculated as Aroclor 1254) in female and male mink livers were 1.5 and 1.4 μg g^{-1}, respectively (O'Shea *et al*., 1981). Mean PCB concentrations in Mersea Township, bordering Lake Erie, Ontario, were reported

at 1.8 μg g^{-1} (calculated as 1254 and 1260) (Haffner *et al*., 1998). Total PCB concentrations reported in this study are similar to those previously reported for the coastal plain of North Carolina, South Carolina, and Georgia, with respective mean values of 219, 216, and 154 ng g^{-1}, reported as the sum of 9 congeners.

The profile of the non-*ortho* congeners in this study is different from those observed in dolphins (Kannan *et al*., 1993; Senthilkumar *et al*., 1999; Watanabe *et al*., 2000), fishes (Kannan *et al*., 2000b) as well as in technical PCB preparations, which contain PCB congener 77 at greater concentrations than PCB 126 (Kannan *et al*., 1993). Similarly, greater concentrations of PCB 126 compared to that of PCB 77 has been reported for mink from northwestern North America (Tillitt *et al*., 1996; Harding *et al*., 1999). These results indicate that mink have great ability to metabolize and excrete PCB congener 77.

Harding *et al*. (1999) demonstrated a significant negative correlation between total PCB concentration and baculum length in juvenile mink from British Columbia, even at relatively low total PCB concentrations (approximately 60 ng g^{-1}, wet wt.), although the ultimate effect on reproductive success of these mink is unknown. Hepatic retinoids have been shown to be negatively correlated with total PCB concentrations in wild and captive European otter (*Lutra lutra*) (Murk *et al*., 1998). The reduction of hepatic retinoids associated from 42 pg TEQ g^{-1} (EC_1 or the NOAEL) to 84 pg TEQ g^{-1} (EC_{90}) coincided with a higher incidence of infectious disease (Murk *et al*., 1998).

Generally low levels of PCDDs and PCDFs have been reported in wild mink (Poole *et al*., 1998; Elliott *et al*., 1999; Harding *et al*., 1999). The mean concentration of 2,3,7,8-TCDD observed in mink livers in this study is greater than those reported for mink from the St. Maurice River, Quebec, and the Kootenay River and Lower Fraser Valley, British Columbia (Table IV) (Champoux, 1996; Harding *et al*., 1999). TEQ concentrations for PCDD/DFs in mink from upstream, near downstream, and far downstream were 2, 3, and 33 pg g^{-1} (Champoux, 1996). A similar pattern of the more highly chlorinated isomers reaching higher concentrations in mink liver has also been observed in mink from British Columbia, Quebec, and the Columbia and Fraser Rivers (Elliott *et al*., 1999; Champoux, 1996; Harding *et al*., 1999).

4.3. HAZARD/RISK ASSESSMENT

Hazard quotients are a simple approach commonly used to determine risk and are often used in conducting Tier I risk assessments. A hazard quotient is simply the ratio of the concentration in the tissue divided by an appropriate reference dose. Values of the HQ can range from below 0.1 to infinity, with values of less than one considered a level of acceptable risk. An HQ of one or greater indicates that concentrations may elicit a statistically significant response and therefore is an unacceptable risk. However, population level impacts are frequently not observed until HQ values are above 10 (Giesy and Kannan, 1998).

TABLE IV

Ranges of concentrations of PCDDs and PCDFs (pg g^{-1}, wet weight) in mink livers reported in North America

	St. Maurice River, Quebec[a]	Kootenay River and Lower Fraser Valley, British Columbia[b]	Northwest Territories[c]	Columbia and Fraser Rivers[d]
2,3,7,8-D	ND –0.9	<1.9–<3.6		<0.4–2.1
1,2,3,6,7,8-D	4.0 –20.55	<4.0–<7.0		<0.4–21.5
1,2,3,4,6,7,8-D	1.5 –35.5			<1.2–193
OCDD	<7.0 –50.48	<13 –<30		<15 –842
2,3,7,8-F	ND –0.38	<1.9–<2.6		
1,2,3,7,8-F	ND –0.27			
2,3,4,7,8-F	3.48–44.15	<1.7–<3.0	0.34	<0.2–6.0
1,2,3,6,7,8-F	<4.0 –21.65			<0.3–2.3
2,3,4,6,7,8-F			0.36–0.41	
OCDF	ND –0.60			<0.7–38.71

[a] Champoux 1996.
[b] Harding *et al*., 1999.
[c] Poole *et al*., 1998.
[d] Elliott *et al*., 1999.

TABLE V

Hazard quotients calculated from total PCB ranges and TEQ levels found in mink from South Carolina and Louisiana. All calculations are based on wet wts. unless otherwise noted. Reference doses used for calculations are included

	RfD (Kannan *et al*., 2000)		South Carolina		Louisiana	
	PCB (μg g^{-1})	TEQ (pg g^{-1})	PCB	TEQ	PCB	TEQ
Otter EC_1	0.17	42	0.78–**2.43**	0.5	0.38–**1.40**	0.33
Otter EC_{90}	0.46	84	0.29–0.90	0.25	0.14–0.52	0.16
Mink NOAEL	2.03 lipid wt.	11	**1.18–5.67**	**1.91**	0.79–**2.96**	**1.25**
Mink LOAEL	44.4 lipid wt.	324	0.05–0.26	0.06	0.04–0.14	0.04
Mink MATC	–	60	–	0.35	–	0.23

The reference doses (RfD) chosen for this assessment (Table V) are based on field and semi-field studies with mustelids (Murk *et al*., 1998; Heaton *et al*., 1995; Tillitt *et al*., 1996; as cited in Kannan *et al*., 2000a). The RfDs from these studies allow for the comparison of risk, from environmentally weathered compounds, of immunosuppressive and/or reproductive effects in mink.

The resulting HQs calculated using both PCB and TEQ based RfDs are generally below 1. Tissue concentrations indicate that the South Carolina mink population is being exposed to additional and/or greater sources of PHHs than the Louisiana source population. Although concentrations of PCBs from South Carolina mink were significantly higher (statistically) than those from Louisiana, there does not seem to be a difference in regards to risk. As the HQs calculated using the mink maximum acceptable toxicant concentration (MATC) are below one, it is unlikely that PHH contamination is causing reproductive failure (or other PHH-related reproductive effects) on a population level. Because contaminant levels are at or slightly above threshold levels (HQ > 1), there is some cause for concern.

The results of this preliminary study suggest that the contaminant levels are not responsible for the high mortality levels observed during transport of the mink during reintroduction effort. Some individuals within the South Carolina and Louisiana populations may be experiencing immunological and/or reproductive impairments due to PCB/PHH contamination. However, because of the small sample size, additional research is needed. Future studies should examine the contaminant concentrations in prey items to complement this initial assessment of PHH levels in mink.

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