Appendix C

TABLES AND FIGURES FOR CHAPTER 4: ASSESSMENT OF RISK TO PISCIVORES ON THE OAK RIDGE RESERVATION

Table C.1. Life history parameters for mink

Parameter	Value	Comments	Reference
Body Weight	1.0 kg (mean ♂+♀)		EPA 1993b
Food Consumption Rate	0.137 kg/d (mean ♂+♀)		Bleavins and Aulerich 1981
Water Consumption Rate	0.099 L/d	estimated using allometric equation[a] assuming 1.0 kg bw	After Calder and Braun 1983
Diet Composition	Diverse diet includes: mammals, fish, aquatic invertebrates, amphibians, and birds		Hamilton 1940, Sealander 1943, Korschgen 1958, Burgess and Bider 1980 Alexander 1977
	Proportion of aquatic prey (fish, amphibians, inverts, etc.) = 0.546±0.21	Proportion represents means of values from five studies	
	fish sizes: 0-10 cm=72% 11-20 cm=28%		
Home Range	2.63 km (♂) 1.85 km (♀)	stream - Sweden	Gerell 1970
	770 ha (♂)	prairie potholes, Manitoba	Arnold and Fritzell 1987
		range size and shape depends on habitat - linear along streams, circular in marshes	EPA 1993a.
Habitat Requirements	aquatic habitats - streams, lakes, marshes;		Burt and Grossenheider 1976
Population Density	0.03 - 0.085 /ha	river - Montana	Mitchell 1961
	0.6/km	river - Michigan	EPA 1993a
Behavior	nocturnal active year-round, does not hibernate		EPA 1993a

Table C.2. Life history parameters for river otter

Parameter	Value	Comments	Reference
Body Weight	**8.0** kg (mean ♂+♀)		EPA 1993b
Food Consumption Rate	**0.9** kg/d (mean ♂+♀)		EPA 1993b
Water Consumption Rate	**0.64** L/d		EPA 1993b
Diet Composition	Almost exclusively fish 2-50 cm in size; most ≥30 cm.		Melquist and Hornocker 1983
	50% large and 50% small fish		EPA 1993b
Home Range	10-78 km	river-Idaho	Melquist and Hornocker 1983
		range size and shape depends on habitat - linear along streams, circular in marshes	EPA 1993b
Habitat Requirements	aquatic habitats - streams, lakes, marshes;		EPA 1993b
Population Density	0.17 - 0.37 /km	river-Idaho	Melquist and Hornocker 1983
	0.0094-0.014/ha		EPA 1993b
Behavior	Generally most active morning and evening, but may be active at any time in day.		Melquist and Hornocker 1983
	active year-round, does not hibernate		EPA 1993b

Table C.3. Life history parameters for belted kingfisher

Parameter	Value	Comments	Reference
Body Weight	**0.148** kg		Dunning 1984
Food Consumption Rate	50% bw		Alexander 1977
	0.075 kg/d	assuming 0.148 kg bw	
Water Consumption Rate	**0.016** L/d	estimated using allometric equation[a] assuming 0.148 kg bw	
Soil Consumption Rate	as a piscivore, assumed to be negligible		
Diet Composition	Cyprinids - 76.4% other fish - 10.2% crayfish - 13.3%	Ohio - creek	Davis 1982
	lizards, small snakes, frogs, salamanders, and insects may be consumed if fish are unavailable		Landrum et al. 1993
Home Range	1.03 km (breeding) 0.39 km (non-breeding)	Ohio - creek	Davis 1982
	2.19 km (breeding)	Pennsylvania - stream summer	Brooks and Davis 1987
Habitat Requirements	uses a diverse aquatic habitats (stream, river, lake, marsh, coastline)		Brooks and Davis 1987
	require high vertical banks composed of >75% sand and <7% clay for nest construction		
	prefer relatively clear waters free of thick vegetation		Bent 1940.
Population Density	0.11 - 0.19 pairs/km shore	Pennsylvania - stream summer	Brooks and Davis 1987
Behavior	while most migrate from northern parts of range, some may stay in areas where water remains ice-free		Bent 1940.

Table C.4. Life history parameters for great blue heron

Parameter	Value	Comments	Reference
Body Weight	2.576 kg (♂) 2.204 kg (♀) **2.39** kg (mean♂+♀)		Dunning 1984
Food Consumption Rate	**0.42** kg/d	estimated using allometric equation[a] specific for herons and egrets assuming 2.39 kg bw	Kushlan 1978
Water Consumption Rate	**0.1058** L/d	estimated using allometric equation[b] assuming 2.39 kg bw	After Calder and Braun 1983
Diet Composition	diet predominantly fish but may include crustaceans, insects, snails, amphibians, reptiles, birds, and mammals		Kushlan 1978 Callazo 1985 Hoffman 1978
	fish sizes: 0-10 cm=39.2% 11-20 cm=47.1% 21-30 cm=13.7%		Alexander 1977
Home Range (foraging distance from colony)	3.1 km	up to 24.2 km - S. Dakota - river	EPA 1993a.
	7 - 8 km	N. Carolina - coastal	Short and Cooper 1985
Habitat Requirements	use both coastal and inland water-associated habitats		Short and Cooper 1985
	Foraging: shallow shores of ponds, lakes, streams, wet meadows, wooded swamps, bays, and marshes		DeGraaf et al. 1981
	breeding: trees for rookery sites. In absence of trees will use rock ledges, cliffs, and artificial structures		Short and Cooper 1985

Table C.4 (continued)

Parameter	Value	Comments	Reference
Population Density	nest colonially, therefore population density depends on availability of nest habitat and suitable foraging habitat		EPA 1993a
	2.3 - 3.6 /km	North Dakota rivers and streams	
Behavior	may or may not defend a feeding territory depending on local population size and food availability		Kushlan 1978
	Migrates in northern U.S. and southern Canada; year round resident from WV, PA south.		National Geographic Society 1987.

Table C.5. Life history parameters for osprey

Parameter	Value	Comments	Reference
Body Weight	1.5 kg (♂+♀)		EPA 1993c
Food Consumption Rate	0.3 kg/d	fresh weight	EPA 1993c
Water Consumption Rate	0.077 L/d		EPA 1993c
Diet Composition	almost 100% fish	all parts of fish consumed except large bones	EPA 1993c
	fish sizes: 0-10 cm=3.3% 11-20 cm=42.1% 21-30 cm=46.7% 31-40 cm=6.6% >41 cm=1.3%		Van Daele and Van Daele 1982
Home Range (foraging distance from nest site)	10-15 km		VanDaele and VanDaele 1982
Habitat Requirements	Coastal areas plus large rivers and lakes		EPA 1993b
	Nesting habitat requires open, shallow water nearby plus abundant fish.		
	Nests atop isolated (often dead) trees and man-made structures		
Population Density	0.005-0.1 nests/ha		EPA 1993b
Behavior	year-round resident in southern part of range (i.e. Florida)		EPA 1993b
	Migratory in Tennessee		

Table C.6. Summary statistics for fish data from the ORR

						Contaminant concentrations in fish (mg/kg)					
Analyte	Location	Watershed	Size	Obs	Det	Mean	Standard error	95% UCL	Maximum	Lognormal mean	Lognormal standard deviation
Mercury	BCK 0.6	Bear Creek	small	24	24	0.269	0.020	0.304	0.549	0.269	0.091
Mercury	BCK 3.3	Bear Creek	small	8	8	0.060	0.006	0.071	0.086	0.060	0.016
Mercury	BCK 4.5	Bear Creek	small	11	11	0.194	0.021	0.233	0.339	0.195	0.067
Mercury	BCK 9.4	Bear Creek	small	8	8	0.079	0.006	0.090	0.110	0.079	0.015
Mercury	BCK 12.4	Bear Creek	small	8	8	0.120	0.010	0.139	0.170	0.120	0.026
Mercury	EFK 2.1	East Fork	small	28	28	0.281	0.012	0.301	0.436	0.281	0.065
Mercury	EFK 6.3	East Fork	small	24	24	0.420	0.016	0.447	0.549	0.420	0.082
Mercury	EFK 13.8	East Fork	small	24	24	0.425	0.022	0.462	0.612	0.426	0.112
Mercury	EFK 18.2	East Fork	small	25	25	0.430	0.014	0.454	0.549	0.430	0.069
Mercury	EFK 24.0	East Fork	small	24	24	0.411	0.028	0.459	0.761	0.410	0.125
Mercury	EFK 23.4	East Fork	small	24	24	0.420	0.028	0.467	1.009	0.418	0.099
Mercury	EFK 24.5	East Fork	small	24	24	0.762	0.031	0.815	0.956	0.766	0.187
Mercury	EFK 6.3	East Fork	large	12	12	0.339	0.036	0.403	0.580	0.341	0.128
Mercury	EFK 13.8	East Fork	large	12	12	0.307	0.023	0.348	0.418	0.310	0.096
Mercury	EFK 18.2	East Fork	large	12	12	0.300	0.037	0.366	0.580	0.301	0.126
Mercury	EFK 24.0	East Fork	large	4	4	0.400	0.029	0.467	0.483	0.401	0.055
Mercury	K-901	K-25	small	8	8	0.035	0.004	0.042	0.050	0.035	0.012
Mercury	K-710	K-25	small	8	8	0.089	0.004	0.097	0.106	0.089	0.013
Mercury	K-1007b P1	K-25	small	8	8	0.070	0.003	0.075	0.078	0.070	0.009
Mercury	K-1007b P5	K-25	small	8	8	0.047	0.005	0.055	0.072	0.047	0.013
Mercury	CRK 15	K-25	small	29	29	0.112	0.008	0.126	0.245	0.113	0.046
Mercury	MIK 0.2	K-25	small	8	8	0.219	0.030	0.276	0.354	0.221	0.090
Mercury	PCK 1.6	K-25	small	28	27	0.179	0.014	0.202	0.383	0.179	0.075

Table C.6 (continued)

Analyte	Location	Watershed	Size	Obs	Det	Contaminant concentrations in fish (mg/kg)					
						Mean	Standard error	95% UCL	Maximum	Lognormal mean	Lognormal standard deviation
Mercury	PCK 2.3	K-25	small	11	11	0.183	0.016	0.211	0.247	0.187	0.075
Mercury	PCK 6.9	K-25	small	8	8	0.203	0.009	0.220	0.237	0.203	0.026
Mercury	PCK 7.2	K-25	small	12	12	0.278	0.025	0.323	0.361	0.288	0.142
Mercury	PCK 7.4	K-25	small	12	12	0.212	0.019	0.246	0.394	0.212	0.056
Mercury	PCK 8.2	K-25	small	16	16	0.251	0.017	0.281	0.443	0.252	0.063
Mercury	PCK 8.5	K-25	small	9	7	0.233	0.021	0.272	0.335	0.223	0.096
Mercury	PCK 8.9	K-25	small	9	9	0.290	0.030	0.346	0.408	0.294	0.111
Mercury	PCK 9.7	K-25	small	10	10	0.108	0.032	0.167	0.311	0.105	0.086
Mercury	PCK 18.2	K-25	small	8	8	0.052	0.003	0.058	0.066	0.052	0.009
Mercury	K-901	K-25	large	18	18	0.174	0.025	0.223	0.483	0.171	0.084
Mercury	CRK 15	K-25	large	20	15	0.146	0.018	0.178	0.343	0.149	0.092
Mercury	PCK 1.6	K-25	large	20	20	0.284	0.031	0.337	0.645	0.282	0.121
Mercury	PCK 2.3	K-25	large	2	2	0.269	0.019	0.392	0.288	0.269	0.028
Mercury	PCK 7.2	K-25	large	1	1	0.321	0.321				
Mercury	PCK 7.4	K-25	large	5	5	0.376	0.078	0.543	0.602	0.387	0.203
Mercury	PCK 8.5	K-25	large	2	2	0.531	0.192	1.744	0.723	0.571	0.330
Mercury	MEK 0.2	White Oak	small	24	24	0.090	0.006	0.099	0.161	0.090	0.025
Mercury	NTK 0.2	White Oak	small	8	8	0.123	0.008	0.139	0.157	0.124	0.026
Mercury	WCK 0.9	White Oak	small	16	16	0.103	0.006	0.114	0.161	0.103	0.024
Mercury	WCK 1.5	White Oak	small	16	16	0.096	0.009	0.111	0.166	0.096	0.035
Mercury	WCK 2.3	White Oak	small	8	8	0.154	0.020	0.191	0.261	0.155	0.052
Mercury	WCK 2.9	White Oak	small	8	8	0.176	0.016	0.207	0.245	0.177	0.046
Mercury	WCK 3.5	White Oak	small	16	16	0.111	0.007	0.124	0.166	0.112	0.033
Mercury	WCK 1.5	White Oak	large	16	16	0.154	0.018	0.185	0.301	0.155	0.082

Table C.6 (continued)

Analyte	Location	Watershed	Size	Obs	Det	Contaminant concentrations in fish (mg/kg)					
						Mean	Standard error	95% UCL	Maximum	Lognormal mean	Lognormal standard deviation
PCBs	BCK 0.6	Bear Creek	small	24	24	0.718	0.083	0.860	1.703	0.727	0.464
PCBs	BCK 3.3	Bear Creek	small	8	8	0.978	0.150	1.272	1.750	0.978	0.417
PCBs	BCK 4.5	Bear Creek	small	11	11	1.951	0.368	2.618	3.766	2.342	3.011
PCBs	BCK 9.4	Bear Creek	small	8	8	2.855	0.494	3.823	4.500	2.961	2.004
PCBs	BCK 12.4	Bear Creek	small	8	8	0.275	0.066	0.403	0.610	0.285	0.250
PCBs	EFK 2.1	East Fork	small	28	28	0.613	0.063	0.720	1.564	0.644	0.489
PCBs	EFK 6.3	East Fork	small	24	24	0.663	0.086	0.810	2.166	0.658	0.363
PCBs	EFK 13.8	East Fork	small	24	24	0.869	0.138	1.106	3.360	0.864	0.620
PCBs	EFK 18.2	East Fork	small	24	24	1.364	0.181	1.673	3.856	1.373	0.938
PCBs	EFK 24.0	East Fork	small	24	24	5.705	0.852	7.165	17.430	6.217	7.126
PCBs	EFK 23.4	East Fork	small	24	24	2.543	0.629	3.620	15.746	2.372	1.922
PCBs	EFK 24.5	East Fork	small	24	24	7.479	2.208	11.264	53.442	7.268	9.933
PCBs	EFK 6.3	East Fork	large	12	12	2.281	0.664	3.473	7.514	2.442	3.539
PCBs	EFK 13.8	East Fork	large	12	12	3.225	0.636	4.368	8.185	3.268	2.367
PCBs	EFK 18.2	East Fork	large	12	12	2.410	0.347	3.033	5.007	2.454	1.377
PCBs	EFK 24.0	East Fork	large	4	4	10.920	0.911	13.063	12.770	10.961	1.878
PCBs	K-901	K-25	small	8	8	6.338	1.470	9.218	15.000	6.346	4.186
PCBs	K-1007b P1	K-25	small	4	4	4.538	1.773	8.710	8.597	5.805	8.868
PCBs	K-1007b P5	K-25	small	4	4	0.123	0.018	0.165	0.164	0.124	0.040
PCBs	CRK 15	K-25	small	6	6	0.870	0.057	0.985	1.110	0.872	0.136
PCBs	MIK 0.2	K-25	small	3	3	3.099	0.262	3.864	3.464	3.112	0.477
PCBs	PCK 1.6	K-25	small	14	13	0.984	0.106	1.172	1.830	0.978	0.378
PCBs	PCK 7.4	K-25	small	2	2	2.460	0.459	5.358	2.919	2.504	0.681
PCBs	K-901	K-25	large	9	9	1.013	0.321	1.641	2.884	0.972	0.834

Table C.6 (continued)

Analyte	Location	Watershed	Size	Obs	Det	Contaminant concentrations in fish (mg/kg) Mean	Standard error	95% UCL	Maximum	Lognormal mean	Lognormal standard deviation
PCBs	K-710	K-25	large	4	4	0.806	0.237	1.363	1.481	0.835	0.502
PCBs	K-1007b P1	K-25	large	14	14	29.964	3.346	35.890	58.212	30.166	13.015
PCBs	K-1007b P5	K-25	large	2	2	1.327	0.962	7.403	2.290	2.124	4.453
PCBs	CRK 15	K-25	large	38	38	2.509	0.134	2.734	4.036	2.528	0.965
PCBs	PCK 1.1	K-25	large	10	10	0.937	0.078	1.080	1.328	0.940	0.244
PCBs	PCK 1.6	K-25	large	40	39	3.242	0.143	3.482	5.281	3.245	1.019
PCBs	PCK 2.3	K-25	large	10	10	1.073	0.112	1.278	1.840	1.075	0.332
PCBs	PCK 6.9	K-25	large	8	8	1.988	0.178	2.326	2.715	1.996	0.513
PCBs	PCK 7.2	K-25	large	8	8	1.554	0.184	1.901	2.575	1.566	0.544
PCBs	PCK 7.4	K-25	large	8	8	2.789	0.254	3.271	3.808	2.807	0.793
PCBs	PCK 8.5	K-25	large	12	11	2.916	0.184	3.247	3.512	2.931	0.810
PCBs	PCK 8.9	K-25	large	8	8	0.842	0.097	1.025	1.128	0.859	0.361
PCBs	PCK 9.7	K-25	large	4	4	0.776	0.130	1.081	1.102	0.789	0.282
PCBs	MEK 0.2	White Oak	small	20	15	0.247	0.062	0.355	1.330	0.257	0.384
PCBs	NTK 0.2	White Oak	small	8	8	0.290	0.108	0.495	0.992	0.300	0.344
PCBs	WCK 0.9	White Oak	small	13	13	0.587	0.110	0.783	1.724	0.609	0.470
PCBs	WCK 1.5	White Oak	small	24	24	2.097	0.284	2.584	6.587	2.100	1.371
PCBs	WCK 2.3	White Oak	small	8	8	1.592	0.304	2.169	3.502	1.603	0.805
PCBs	WCK 2.9	White Oak	small	16	16	1.107	0.195	1.448	2.915	1.141	0.978
PCBs	WCK 3.5	White Oak	small	16	16	1.300	0.160	1.580	2.303	1.349	0.889
PCBs	WCK 0.9	White Oak	large	10	10	6.483	1.236	8.748	13.008	7.501	8.244
PCBs	WCK 1.5	White Oak	large	16	16	13.149	1.814	16.329	28.445	13.520	9.065
PCBs	WCK 0.3	White Oak	large	4	4	5.829	0.421	6.819	6.702	5.847	0.887

Table C.7. Estimated exposure of mink on the ORR to mercury and PCBs

Analyte	Drainage	Sampling station	Dietary exposure (mg/kg-d)	NOAEL HQ	LOAEL HQ
Mercury	East Fork	EFK 2.1	0.0225	1.50	0.90
Mercury	East Fork	EFK 6.3	0.0334	2.23	1.34
Mercury	East Fork	EFK 13.8	0.0346	2.31	1.38
Mercury	East Fork	EFK 18.2	0.0339	2.26	1.36
Mercury	East Fork	EFK 23.4	0.0349	2.33	1.40
Mercury	East Fork	EFK 24.0	0.0343	2.29	1.37
Mercury	East Fork	EFK 24.5	0.0609	4.06	2.44
Mercury	Bear Creek	BCK 0.6	0.0227	1.51	0.91
Mercury	Bear Creek	BCK 3.3	0.0053	0.35	0.21
Mercury	Bear Creek	BCK 4.5	0.0174	1.16	0.70
Mercury	Bear Creek	BCK 9.4	0.0067	0.45	0.27
Mercury	Bear Creek	BCK 12.4	0.0104	0.69	0.42
Mercury	K-25	CRK 15	0.0094	0.63	0.38
Mercury	K-25	K-901	0.0032	0.21	0.13
Mercury	K-25	K-710	0.0073	0.48	0.29
Mercury	K-25	PCK 1.6	0.0151	1.01	0.61
Mercury	K-25	K-1007b P1	0.0056	0.37	0.22
Mercury	K-25	K-1007b P5	0.0041	0.28	0.17
Mercury	K-25	PCK 2.3	0.0158	1.05	0.63
Mercury	K-25	PCK 6.9	0.0165	1.10	0.66
Mercury	K-25	PCK 7.2	0.0242	1.61	0.97
Mercury	K-25	PCK 7.4	0.0184	1.22	0.73
Mercury	K-25	MIK 0.2	0.0206	1.37	0.82
Mercury	K-25	PCK 8.2	0.0210	1.40	0.84
Mercury	K-25	PCK 8.5	0.0203	1.36	0.81
Mercury	K-25	PCK 8.9	0.0259	1.72	1.04
Mercury	K-25	PCK 9.7	0.0125	0.83	0.50
Mercury	K-25	PCK 18.2	0.0043	0.29	0.17
Mercury	White Oak	WCK 0.9	0.0085	0.57	0.34
Mercury	White Oak	WCK 1.5	0.0083	0.55	0.33
Mercury	White Oak	WCK 2.3	0.0143	0.95	0.57
Mercury	White Oak	MEK 0.2	0.0074	0.49	0.30
Mercury	White Oak	WCK 2.9	0.0155	1.03	0.62
Mercury	White Oak	WCK 3.5	0.0093	0.62	0.37
Mercury	White Oak	NTK 0.2	0.0104	0.69	0.42
PCBs	East Fork	EFK 2.1	0.0538	0.38	0.08
PCBs	East Fork	EFK 6.3	0.0606	0.43	0.09
PCBs	East Fork	EFK 13.8	0.0827	0.59	0.12
PCBs	East Fork	EFK 18.2	0.1251	0.89	0.18
PCBs	East Fork	EFK 23.4	0.2708	1.93	0.39

Table C.7 (continued)

Analyte	Drainage	Sampling station	Dietary exposure (mg/kg-d)	NOAEL HQ	LOAEL HQ
PCBs	East Fork	EFK 24.0	0.5360	3.83	0.78
PCBs	East Fork	EFK 24.5	0.8426	6.02	1.22
PCBs	Bear Creek	BCK 0.6	0.0644	0.46	0.09
PCBs	Bear Creek	BCK 3.3	0.0951	0.68	0.14
PCBs	Bear Creek	BCK 4.5	0.1959	1.40	0.28
PCBs	Bear Creek	BCK 9.4	0.2860	2.04	0.41
PCBs	Bear Creek	BCK 12.4	0.0301	0.22	0.04
PCBs	K-25	CRK 15	0.0736	0.53	0.11
PCBs	K-25	K-901	0.6895	4.93	1.00
PCBs	K-25	K-710	0.1020	0.73	0.15
PCBs	K-25	PCK 1.1	0.0808	0.58	0.12
PCBs	K-25	PCK 1.6	0.0876	0.63	0.13
PCBs	K-25	K-1007b P1	0.6516	4.65	0.94
PCBs	K-25	K-1007b P5	0.0123	0.09	0.02
PCBs	K-25	PCK 2.3	0.0956	0.68	0.14
PCBs	K-25	PCK 6.9	0.1740	1.24	0.25
PCBs	K-25	PCK 7.2	0.1422	1.02	0.21
PCBs	K-25	PCK 7.4	0.4008	2.86	0.58
PCBs	K-25	MIK 0.2	0.2891	2.06	0.42
PCBs	K-25	PCK 8.5	0.2429	1.73	0.35
PCBs	K-25	PCK 8.9	0.0767	0.55	0.11
PCBs	K-25	PCK 9.7	0.0809	0.58	0.12
PCBs	White Oak	WCK 0.3	0.5101	3.64	0.74
PCBs	White Oak	WCK 0.9	0.0586	0.42	0.08
PCBs	White Oak	WCK 1.5	0.1933	1.38	0.28
PCBs	White Oak	WCK 2.3	0.1622	1.16	0.24
PCBs	White Oak	MEK 0.2	0.0265	0.19	0.04
PCBs	White Oak	WCK 2.9	0.1083	0.77	0.16
PCBs	White Oak	WCK 3.5	0.1182	0.84	0.17
PCBs	White Oak	NTK 0.2	0.0370	0.26	0.05

Table C.8. Estimated exposure of river otter on the ORR to mercury and PCBs

Analyte	Drainage	Sampling station	Dietary exposure (mg/kg-d)	NOAEL HQ	LOAEL HQ
Mercury	EFK 2.1	East Fork	0.0339	3.77	2.26
Mercury	EFK 6.3	East Fork	0.0478	5.31	3.19
Mercury	EFK 13.8	East Fork	0.0456	5.07	3.04
Mercury	EFK 18.2	East Fork	0.0461	5.12	3.07
Mercury	EFK 23.4	East Fork	0.0526	5.84	3.50
Mercury	EFK 24.0	East Fork	0.0521	5.79	3.47
Mercury	EFK 24.5	East Fork	0.0917	10.18	6.11
Mercury	BCK 0.6	Bear Creek	0.0342	3.80	2.28
Mercury	BCK 3.3	Bear Creek	0.0080	0.89	0.53
Mercury	BCK 4.5	Bear Creek	0.0262	2.91	1.74
Mercury	BCK 9.4	Bear Creek	0.0101	1.13	0.68
Mercury	BCK 12.4	Bear Creek	0.0156	1.74	1.04
Mercury	CRK 15	K-25	0.0171	1.90	1.14
Mercury	K-901	K-25	0.0149	1.66	0.99
Mercury	K-710	K-25	0.0109	1.22	0.73
Mercury	PCK 1.6	K-25	0.0303	3.37	2.02
Mercury	K-1007b P1	K-25	0.0084	0.94	0.56
Mercury	K-1007b P5	K-25	0.0062	0.69	0.42
Mercury	PCK 2.3	K-25	0.0339	3.77	2.26
Mercury	PCK 6.9	K-25	0.0248	2.76	1.65
Mercury	PCK 7.2	K-25	0.0363	4.03	2.42
Mercury	PCK 7.4	K-25	0.0443	4.93	2.96
Mercury	MIK 0.2	K-25	0.0310	3.45	2.07
Mercury	PCK 8.2	K-25	0.0316	3.51	2.11
Mercury	PCK 8.5	K-25	0.1134	12.60	7.56
Mercury	PCK 8.9	K-25	0.0389	4.32	2.59
Mercury	PCK 9.7	K-25	0.0188	2.09	1.25
Mercury	PCK 18.2	K-25	0.0065	0.73	0.44
Mercury	WCK 0.9	White Oak	0.0128	1.43	0.86
Mercury	WCK 1.5	White Oak	0.0166	1.85	1.11
Mercury	WCK 2.3	White Oak	0.0215	2.39	1.43
Mercury	MEK 0.2	White Oak	0.0111	1.24	0.74
Mercury	WCK 2.9	White Oak	0.0233	2.59	1.55
Mercury	WCK 3.5	White Oak	0.0140	1.55	0.93
Mercury	NTK 0.2	White Oak	0.0157	1.74	1.04
PCBs	BCK 0.6	Bear Creek	0.0968	1.17	0.24
PCBs	BCK 3.3	Bear Creek	0.1431	1.72	0.35
PCBs	BCK 4.5	Bear Creek	0.2946	3.55	0.72
PCBs	BCK 9.4	Bear Creek	0.4301	5.18	1.05
PCBs	BCK 12.4	Bear Creek	0.0453	0.55	0.11

Table C.8 (continued)

Analyte	Drainage	Sampling station	Dietary exposure (mg/kg-d)	NOAEL HQ	LOAEL HQ
PCBs	EFK 2.1	East Fork	0.0810	0.98	0.20
PCBs	EFK 6.3	East Fork	0.2409	2.90	0.59
PCBs	EFK 13.8	East Fork	0.3079	3.71	0.75
PCBs	EFK 18.2	East Fork	0.2647	3.19	0.65
PCBs	EFK 23.4	East Fork	0.4073	4.91	0.99
PCBs	EFK 24.0	East Fork	1.1379	13.71	2.78
PCBs	EFK 24.5	East Fork	1.2672	15.27	3.09
PCBs	CRK 15	K-25	0.2092	2.52	0.51
PCBs	K-901	K-25	0.6108	7.36	1.49
PCBs	K-710	K-25	0.1533	1.85	0.37
PCBs	PCK 1.1	K-25	0.1215	1.46	0.30
PCBs	PCK 1.6	K-25	0.2618	3.15	0.64
PCBs	K-1007b P1	K-25	2.5088	30.23	6.12
PCBs	K-1007b P5	K-25	0.4257	5.13	1.04
PCBs	PCK 2.3	K-25	0.1438	1.73	0.35
PCBs	PCK 6.9	K-25	0.2616	3.15	0.64
PCBs	PCK 7.2	K-25	0.2139	2.58	0.52
PCBs	PCK 7.4	K-25	0.4853	5.85	1.18
PCBs	MIK 0.2	K-25	0.4347	5.24	1.06
PCBs	PCK 8.5	K-25	0.3653	4.40	0.89
PCBs	PCK 8.9	K-25	0.1154	1.39	0.28
PCBs	PCK 9.7	K-25	0.1216	1.47	0.30
PCBs	WCK 0.3	White Oak	0.7671	9.24	1.87
PCBs	WCK 0.9	White Oak	0.5362	6.46	1.31
PCBs	WCK 1.5	White Oak	1.0638	12.82	2.59
PCBs	WCK 2.3	White Oak	0.2440	2.94	0.60
PCBs	MEK 0.2	White Oak	0.0399	0.48	0.10
PCBs	WCK 2.9	White Oak	0.1629	1.96	0.40
PCBs	WCK 3.5	White Oak	0.1777	2.14	0.43
PCBs	NTK 0.2	White Oak	0.0557	0.67	0.14

Table C.9. Estimated exposure of belted kingfisher on the ORR to mercury and PCBs

Analyte	Drainage	Sampling station	Dietary exposure (mg/kg-d)	NOAEL HQ	LOAEL HQ
Mercury	EFK 2.1	East Fork	0.1527	25.46	2.39
Mercury	EFK 6.3	East Fork	0.2264	37.73	3.54
Mercury	EFK 13.8	East Fork	0.2343	39.05	3.66
Mercury	EFK 18.2	East Fork	0.2299	38.32	3.59
Mercury	EFK 23.4	East Fork	0.2368	39.46	3.70
Mercury	EFK 24.0	East Fork	0.2327	38.78	3.64
Mercury	EFK 24.5	East Fork	0.4129	68.81	6.45
Mercury	BCK 0.6	Bear Creek	0.1539	25.64	2.40
Mercury	BCK 3.3	Bear Creek	0.0360	6.00	0.56
Mercury	BCK 4.5	Bear Creek	0.1178	19.64	1.84
Mercury	BCK 9.4	Bear Creek	0.0456	7.60	0.71
Mercury	BCK 12.4	Bear Creek	0.0704	11.74	1.10
Mercury	CRK 15	K-25	0.0638	10.63	1.00
Mercury	K-901	K-25	0.0215	3.58	0.34
Mercury	K-710	K-25	0.0493	8.21	0.77
Mercury	PCK 1.6	K-25	0.1025	17.08	1.60
Mercury	K-1007b P1	K-25	0.0381	6.34	0.59
Mercury	K-1007b P5	K-25	0.0281	4.68	0.44
Mercury	PCK 2.3	K-25	0.1070	17.83	1.67
Mercury	PCK 6.9	K-25	0.1117	18.61	1.75
Mercury	PCK 7.2	K-25	0.1639	27.31	2.56
Mercury	PCK 7.4	K-25	0.1244	20.73	1.94
Mercury	MIK 0.2	K-25	0.1397	23.28	2.18
Mercury	PCK 8.2	K-25	0.1423	23.72	2.22
Mercury	PCK 8.5	K-25	0.1377	22.96	2.15
Mercury	PCK 8.9	K-25	0.1753	29.21	2.74
Mercury	PCK 9.7	K-25	0.0846	14.10	1.32
Mercury	PCK 18.2	K-25	0.0294	4.90	0.46
Mercury	WCK 0.9	White Oak	0.0579	9.65	0.90
Mercury	WCK 1.5	White Oak	0.0560	9.34	0.88
Mercury	WCK 2.3	White Oak	0.0969	16.16	1.51
Mercury	MEK 0.2	White Oak	0.0502	8.37	0.78
Mercury	WCK 2.9	White Oak	0.1048	17.47	1.64
Mercury	WCK 3.5	White Oak	0.0630	10.50	0.98
Mercury	NTK 0.2	White Oak	0.0706	11.77	1.10
PCBs	BCK 0.6	Bear Creek	0.4360	2.42	0.24
PCBs	BCK 3.3	Bear Creek	0.6446	3.58	0.36
PCBs	BCK 4.5	Bear Creek	1.3269	7.37	0.74
PCBs	BCK 9.4	Bear Creek	1.9373	10.76	1.08
PCBs	BCK 12.4	Bear Creek	0.2042	1.13	0.11

Table C.9 (continued)

Analyte	Drainage	Sampling station	Dietary exposure (mg/kg-d)	NOAEL HQ	LOAEL HQ
PCBs	EFK 2.1	East Fork	0.3647	2.03	0.20
PCBs	EFK 6.3	East Fork	0.4105	2.28	0.23
PCBs	EFK 13.8	East Fork	0.5602	3.11	0.31
PCBs	EFK 18.2	East Fork	0.8478	4.71	0.47
PCBs	EFK 23.4	East Fork	1.8345	10.19	1.02
PCBs	EFK 24.0	East Fork	3.6311	20.17	2.02
PCBs	EFK 24.5	East Fork	5.7083	31.71	3.17
PCBs	CRK 15	K-25	0.4989	2.77	0.28
PCBs	K-901	K-25	4.6713	25.95	2.60
PCBs	K-710	K-25	0.6907	3.84	0.38
PCBs	PCK 1.1	K-25	0.5473	3.04	0.30
PCBs	PCK 1.6	K-25	0.5937	3.30	0.33
PCBs	K-1007b P1	K-25	4.4141	24.52	2.45
PCBs	K-1007b P5	K-25	0.0836	0.46	0.05
PCBs	PCK 2.3	K-25	0.6476	3.60	0.36
PCBs	PCK 6.9	K-25	1.1786	6.55	0.65
PCBs	PCK 7.2	K-25	0.9635	5.35	0.54
PCBs	PCK 7.4	K-25	2.7151	15.08	1.51
PCBs	MIK 0.2	K-25	1.9583	10.88	1.09
PCBs	PCK 8.5	K-25	1.6453	9.14	0.91
PCBs	PCK 8.9	K-25	0.5196	2.89	0.29
PCBs	PCK 9.7	K-25	0.5480	3.04	0.30
PCBs	WCK 0.3	White Oak	3.4556	19.20	1.92
PCBs	WCK 0.9	White Oak	0.3969	2.21	0.22
PCBs	WCK 1.5	White Oak	1.3093	7.27	0.73
PCBs	WCK 2.3	White Oak	1.0990	6.11	0.61
PCBs	MEK 0.2	White Oak	0.1797	1.00	0.10
PCBs	WCK 2.9	White Oak	0.7339	4.08	0.41
PCBs	WCK 3.5	White Oak	0.8005	4.45	0.44
PCBs	NTK 0.2	White Oak	0.2508	1.39	0.14

Table C.10. Estimated exposure of great blue heron on the ORR to mercury and PCBs

Analyte	Drainage	Sampling station	Dietary exposure (mg/kg-d)	NOAEL HQ	LOAEL HQ
Mercury	EFK 2.1	East Fork	0.0530	8.83	0.83
Mercury	EFK 6.3	East Fork	0.0785	13.08	1.23
Mercury	EFK 13.8	East Fork	0.0812	13.54	1.27
Mercury	EFK 18.2	East Fork	0.0797	13.29	1.25
Mercury	EFK 23.4	East Fork	0.0821	13.68	1.28
Mercury	EFK 24.0	East Fork	0.0807	13.45	1.26
Mercury	EFK 24.5	East Fork	0.1432	23.86	2.24
Mercury	BCK 0.6	Bear Creek	0.0534	8.89	0.83
Mercury	BCK 3.3	Bear Creek	0.0125	2.08	0.20
Mercury	BCK 4.5	Bear Creek	0.0409	6.81	0.64
Mercury	BCK 9.4	Bear Creek	0.0158	2.64	0.25
Mercury	BCK 12.4	Bear Creek	0.0244	4.07	0.38
Mercury	CRK 15	K-25	0.0221	3.68	0.35
Mercury	K-901	K-25	0.0075	1.24	0.12
Mercury	K-710	K-25	0.0171	2.85	0.27
Mercury	PCK 1.6	K-25	0.0355	5.92	0.56
Mercury	K-1007b P1	K-25	0.0132	2.20	0.21
Mercury	K-1007b P5	K-25	0.0097	1.62	0.15
Mercury	PCK 2.3	K-25	0.0371	6.18	0.58
Mercury	PCK 6.9	K-25	0.0387	6.46	0.61
Mercury	PCK 7.2	K-25	0.0568	9.47	0.89
Mercury	PCK 7.4	K-25	0.0431	7.19	0.67
Mercury	MIK 0.2	K-25	0.0484	8.07	0.76
Mercury	PCK 8.2	K-25	0.0493	8.22	0.77
Mercury	PCK 8.5	K-25	0.0478	7.96	0.75
Mercury	PCK 8.9	K-25	0.0608	10.13	0.95
Mercury	PCK 9.7	K-25	0.0293	4.89	0.46
Mercury	PCK 18.2	K-25	0.0102	1.70	0.16
Mercury	WCK 0.9	White Oak	0.0201	3.34	0.31
Mercury	WCK 1.5	White Oak	0.0194	3.24	0.30
Mercury	WCK 2.3	White Oak	0.0336	5.60	0.53
Mercury	MEK 0.2	White Oak	0.0174	2.90	0.27
Mercury	WCK 2.9	White Oak	0.0364	6.06	0.57
Mercury	WCK 3.5	White Oak	0.0218	3.64	0.34
Mercury	NTK 0.2	White Oak	0.0245	4.08	0.38
PCBs	EFK 2.1	East Fork	0.1265	0.70	0.07
PCBs	EFK 6.3	East Fork	0.1424	0.79	0.08
PCBs	EFK 13.8	East Fork	0.1943	1.08	0.11
PCBs	EFK 18.2	East Fork	0.2940	1.63	0.16
PCBs	EFK 23.4	East Fork	0.6362	3.53	0.35

Table C.10 (continued)

Analyte	Drainage	Sampling station	Dietary exposure (mg/kg-d)	NOAEL HQ	LOAEL HQ
PCBs	EFK 24.0	East Fork	1.2592	7.00	0.70
PCBs	EFK 24.5	East Fork	1.9795	11.00	1.10
PCBs	BCK 0.6	Bear Creek	0.1512	0.84	0.08
PCBs	BCK 3.3	Bear Creek	0.2235	1.24	0.12
PCBs	BCK 4.5	Bear Creek	0.4601	2.56	0.26
PCBs	BCK 9.4	Bear Creek	0.6718	3.73	0.37
PCBs	BCK 12.4	Bear Creek	0.0708	0.39	0.04
PCBs	CRK 15	K-25	0.1730	0.96	0.10
PCBs	K-901	K-25	1.6199	9.00	0.90
PCBs	K-710	K-25	0.2395	1.33	0.13
PCBs	PCK 1.1	K-25	0.1898	1.05	0.11
PCBs	PCK 1.6	K-25	0.2059	1.14	0.11
PCBs	K-1007b P1	K-25	1.5307	8.50	0.85
PCBs	K-1007b P5	K-25	0.0290	0.16	0.02
PCBs	PCK 2.3	K-25	0.2246	1.25	0.12
PCBs	PCK 6.9	K-25	0.4087	2.27	0.23
PCBs	PCK 7.2	K-25	0.3341	1.86	0.19
PCBs	PCK 7.4	K-25	0.9415	5.23	0.52
PCBs	MIK 0.2	K-25	0.6791	3.77	0.38
PCBs	PCK 8.5	K-25	0.5706	3.17	0.32
PCBs	PCK 8.9	K-25	0.1802	1.00	0.10
PCBs	PCK 9.7	K-25	0.1900	1.06	0.11
PCBs	WCK 0.3	White Oak	1.1983	6.66	0.67
PCBs	WCK 0.9	White Oak	0.1377	0.76	0.08
PCBs	WCK 1.5	White Oak	0.4540	2.52	0.25
PCBs	WCK 2.3	White Oak	0.3811	2.12	0.21
PCBs	MEK 0.2	White Oak	0.0623	0.35	0.03
PCBs	WCK 2.9	White Oak	0.2545	1.41	0.14
PCBs	WCK 3.5	White Oak	0.2776	1.54	0.15
PCBs	NTK 0.2	White Oak	0.0870	0.48	0.05

Table C.11. Estimated exposure of osprey on the ORR to mercury and PCBs

Analyte	Drainage	Sampling station	Dietary exposure (mg/kg-d)	NOAEL HQ	LOAEL HQ
Mercury	CRK 15	K-25	0.0260	4.33	0.41
Mercury	K-901	K-25	0.0113	1.89	0.18
Mercury	K-710	K-25	0.0194	3.24	0.30
Mercury	PCK 1.6	K-25	0.0426	7.10	0.67
Mercury	K-1007b P1	K-25	0.0150	2.50	0.23
Mercury	K-1007b P5	K-25	0.0111	1.85	0.17
Mercury	PCK 2.3	K-25	0.0451	7.51	0.70
Mercury	PCK 6.9	K-25	0.0441	7.35	0.69
Mercury	PCK 7.2	K-25	0.0646	10.77	1.01
Mercury	PCK 7.4	K-25	0.0538	8.97	0.84
Mercury	MIK 0.2	K-25	0.0551	9.19	0.86
Mercury	PCK 8.2	K-25	0.0562	9.36	0.88
Mercury	PCK 8.5	K-25	0.0776	12.94	1.21
Mercury	PCK 8.9	K-25	0.0692	11.53	1.08
Mercury	PCK 9.7	K-25	0.0334	5.56	0.52
Mercury	PCK 18.2	K-25	0.0116	1.93	0.18
Mercury	WCK 0.9	White Oak	0.0228	3.81	0.36
Mercury	WCK 1.5	White Oak	0.0233	3.88	0.36
PCBs	CRK 15	K-25	0.2245	1.25	0.12
PCBs	K-901	K-25	1.7239	9.58	0.96
PCBs	K-710	K-25	0.2726	1.51	0.15
PCBs	PCK 1.1	K-25	0.2160	1.20	0.12
PCBs	PCK 1.6	K-25	0.2708	1.50	0.15
PCBs	K-1007b P1	K-25	2.1715	12.06	1.21
PCBs	K-1007b P5	K-25	0.1474	0.82	0.08
PCBs	PCK 2.3	K-25	0.2556	1.42	0.14
PCBs	PCK 6.9	K-25	0.4651	2.58	0.26
PCBs	PCK 7.2	K-25	0.3803	2.11	0.21
PCBs	PCK 7.4	K-25	1.0386	5.77	0.58
PCBs	MIK 0.2	K-25	0.7729	4.29	0.43
PCBs	PCK 8.5	K-25	0.6493	3.61	0.36
PCBs	PCK 8.9	K-25	0.2051	1.14	0.11
PCBs	PCK 9.7	K-25	0.2163	1.20	0.12
PCBs	WCK 0.3	White Oak	1.3638	7.58	0.76
PCBs	WCK 0.9	White Oak	0.2825	1.57	0.16
PCBs	WCK 1.5	White Oak	0.7339	4.08	0.41

Table C.12. Biomass of fish observed at fish sampling locations in Bear Creek in 1993

Sample location		Fish biomass (g/m^2)			
Fish community	Bioaccumulation	Spring	Fall	Mean annual biomass	Proportion of total biomass at sampled locations
BCK 0.7	BCK 0.6	5.84	4.73	5.29	0.18
BCK 3.25	BCK 3.3	2.57	3.29	2.93	0.1
BCK 3.25	BCK 4.5	2.57	3.29	2.93	0.1
BCK 9.4	BCK 9.4	5.37	12.59	8.98	0.30
BCK 12.36	BCK 12.4	14.43	4.43	9.43	0.32

Source: Hinzman et al. 1995.

Table C.13. Density of fish observed at fish sampling locations in East Fork Poplar Creek

Sample location		Fish Density (no./m²)		Proportion of total density at sampled locations
Fish community	Bioaccumulation	Year	Mean annual density	
EFK 2.3 [a]	EFK 2.1	1991	16.4	0.25
EFK 7.3 [a]	EFK 6.3	1991	4.4	0.067
EFK 10.8 [a]	EFK 13.8	1991	16.6	0.253
EFK 17.6 [b]	EFK 18.2	1991	2.8	0.043
EFK 23.4 [b]	EFK 23.4	1993-1995	11.1	0.169
EFK 23.4+24.4 [b]	EFK 24.0	1993-1995	8.5	0.129
EFK 24.4 [b]	EFK 24.5	1993-1995	5.9	0.09

[a] Source:SAIC. 1994.
[b] Source: unpublished UEFPC BMAP data (to be in UEFPC RI workplan)

Table C.14. Total biomass of fish observed at fish sampling locations in White Oak Creek Watershed

Sample location		Fish biomass (g/m^2)				Proportion of total biomass at sampled locations	
Fish community	Bioaccumulation	Spring	Fall	Year	Mean annual biomass	data from all 8 locations	data from only 7 locations
WOL [a]	WCK 0.3	-	-	1987	53.66	0.248	
WOL [a]	WCK 0.9	-	-	1987	53.66	0.248	0.33
WOL [a]	WCK 1.5	-	-	1987	53.66	0.248	0.33
WCK 2.3 [b]	WCK 2.3	10.49	17.06	1993	13.78	0.064	0.085
MEK 0.6 [b]	MEK 0.2	10.52	9.6	1993	10.06	0.046	0.062
WCK 2.9 [b]	WCK 2.9	10.80	13.34	1993	12.07	0.056	0.075
WCK 3.4 [b]	WCK 3.5	17.16	14.30	1993	15.73	0.073	0.097
NTK 0.3 [b]	NTK 0.2	3.27	4.50	1993	3.89	0.018	0.024

[a] Source:Loar et al. 1992.
[b] Source:Ashwood et al. 1994.

Table C.15. Results of Monte Carlo simulation of exposure for piscivores on the ORR

Location	Analyte	Species	No. of sampling locations	Mean	Standard deviation	80th percentile	%> NOAEL	%> LOAEL
Bear Creek	Mercury	Mink	5	0.0102	0.0025	0.0121	5%	<5%
East Fork Poplar Creek	Mercury	Mink	7	0.0310	0.0060	0.0361	>95%	80%
K-25	Mercury	Mink	17	0.0119	0.0019	0.0135	5%	<5%
White Oak Creek	Mercury	Mink	7	0.0083	0.0019	0.0098	<5%	<5%
Bear Creek	Mercury	Otter	5	0.0153	0.0022	0.0170	>95%	50%
East Fork Poplar Creek	Mercury	Otter	7	0.0447	0.0034	0.0477	>95%	>95%
K-25	Mercury	Otter	17	0.0213	0.0020	0.0228	>95%	>95%
White Oak Creek	Mercury	Otter	7	0.0136	0.0018	0.0150	>95%	25%
Bear Creek	Mercury	Kingfisher	5	0.0691	0.0101	0.0769	>95%	65-70%
East Fork Poplar Creek	Mercury	Kingfisher	7	0.2118	0.0230	0.2308	>95%	>95%
K-25	Mercury	Kingfisher	17	0.0816	0.0091	0.0889	>95%	>95%
White Oak Creek	Mercury	Kingfisher	7	0.0564	0.0077	0.0625	>95%	15%
Bear Creek	Mercury	Heron	5	0.0238	0.0034	0.0263	>95%	<5%
East Fork Poplar Creek	Mercury	Heron	7	0.0792	0.0074	0.0851	>95%	>95%
K-25	Mercury	Heron	17	0.0282	0.0031	0.0306	>95%	<5%
White Oak Creek	Mercury	Heron	7	0.0198	0.0028	0.0220	>95%	<5%
K-25	Mercury	Osprey	17	0.0330	0.0033	0.0355	>95%	<5%
White Oak Creek	Mercury	Osprey	7	0.0202	0.0037	0.0229	>95%	<5%
Bear Creek	PCBs	Mink	5	0.1102	0.0610	0.1481	20-25%	<5%
East Fork Poplar Creek	PCBs	Mink	7	0.1735	0.1021	0.2301	50-55%	<5%
K-25	PCBs	Mink	17	0.1476	0.0406	0.1752	50%	<5%
White Oak Creek	PCBs	Mink	8	0.1785	0.0563	0.2194	75%	<5%

Table C.15 (continued)

Location	Analyte	Species	No. of sampling locations	Mean	Standard deviation	80th percentile	%> NOAEL	%> LOAEL
Bear Creek	PCBs	Otter	5	0.1627	0.0752	0.2067	90-95%	<5%
East Fork Poplar Creek	PCBs	Otter	7	0.3483	0.1038	0.4057	>95%	15-20%
K-25	PCBs	Otter	17	0.3222	0.0659	0.3638	>95%	5-10%
White Oak Creek	PCBs	Otter	8	0.5242	0.1784	0.6249	>95%	70-75%
Bear Creek	PCBs	Kingfisher	5	0.7212	0.3406	0.9271	>95%	<5%
East Fork Poplar Creek	PCBs	Kingfisher	7	1.1850	0.6537	1.5290	>95%	10-15%
K-25	PCBs	Kingfisher	17	1.0332	0.3337	1.1631	>95%	<5%
White Oak Creek	PCBs	Kingfisher	8	1.2136	0.2056	1.3590	>95%	<5%
Bear Creek	PCBs	Heron	5	0.2532	0.1169	0.3225	70-75%	<5%
East Fork Poplar Creek	PCBs	Heron	7	0.5017	0.3609	0.6358	>95%	<5%
K-25	PCBs	Heron	17	0.3573	0.1075	0.4051	>95%	<5%
White Oak Creek	PCBs	Heron	8	0.4202	0.0753	0.4721	>95%	<5%
K-25	PCBs	Osprey	17	0.4318	0.1073	0.4859	>95%	<5%
White Oak Creek	PCBs	Osprey	8	0.6605	0.1144	0.7489	>95%	<5%

Table C.16. Estimated NOAELs and LOAELs for mink and river otter

Contaminant	Experimental information						Estimated NOAEL (mg/kg/d)		Estimated LOAEL (mg/kg/d)	
	Form	Test species	NOAEL (mg/kg/d) and duration	LOAEL (mg/kg/d) and duration	Endpoint	Citation	mink	otter	mink	otter
Mercury	methyl	mink	0.015[2] 93 d	0.025[2] 93 d	mortality	Wobeser et al. 1976	0.015	0.009	0.025	0.015
PCB's	Aroclor 1254	mink	0.14 4.5 mo.	0.69 4.5 mo.	reproduction	Aulerich and Ringer 1987	0.14	0.083	0.69	0.41

[2] Estimated value: subchronic-chronic factor of 10 applied.

Table C.17. Estimated NOAELs and LOAELs for belted kingfisher, great blue heron, and osprey

Contaminant	Experimental information						Estimated Values (mg/kg/d)	
	Form	Test species	NOAEL (mg/kg/d) and duration	LOAEL (mg/kg/d) and duration	Endpoint	Citation	NOAEL	LOAEL
Mercury	methyl	mallard duck	0.006[2] 3 gen.	0.064 3 gen.	reproduction	Heinz 1979	0.006	0.064
PCB's	Aroclor 1254	Ring-necked Pheasant	0.18[2] 17 wk	1.8 17 wk	reproduction	Dahlgren et al. 1972	0.18	1.8

[2] Estimated NOAEL: LOAEL-NOAEL factor of 10 applied.

Table C.18 Contaminant concentrations (mg/kg) found in Kingfisher egg shells and feathers found on the ORR

Matrix	Burrow[a]	As	Cd	Se	Pb	Hg	^{60}Co (pCi/g)	^{137}Cs (pCi/g)
egg shell	CRD	0.135	< 0.0333[b]	1.58	2.0	< 0.020	< 7.45	< 9.09
egg shell	WOC	0.0536	0.0583	1.41	5.31	0.182	< 1.89	58.1 _ 1.7
feathers	CRU	0.074	0.0132	5.72	0.657	1.03	< 0.21	< 0.18
feathers	CRU	0.0449	< 0.0102	6.54	1.42	1.01	< 0.18	< 0.19
feathers	CRU	0.052	< 0.010	5.72	1.67	1.04	< 0.17	< 0.18
feathers	CRU	0.0755	0.0755	6.83	1.91	0.726	< 0.25	< 0.16

[a]CRD = Clinch River downstream of WOL Embayment; WOC = White Oak Creek downstream of WCK 3.5; CRU = Clinch River upstream of Oak Ridge Reservation.

[b]Less than values are below minimum detection limit.

Table C.19 Contaminant concentrations in tissues of the three kingfishers found on the ORR

Bird No.	Watershed and Location	Organ	^{137}Cs (pCi/g)	Cd (mg/kg)[a]	Pb (mg/kg)[a]	Se (mg/kg)	Hg (mg/kg)
1	East Fork Poplar Creek, Lake Reality	whole body	<2				
		feathers		ND	2.67	5.38	13.9
		kidney		4.04	ND	5.81	8.65
		liver		0.95	ND	2.71	3.69
		heart		ND	ND	1.25	1.1
		muscle		ND	ND	ND	0.572
2	East Fork Poplar Creek	feathers		7.21	1.86	5.63	4.55
		kidney		0.40	ND	3.14	1.46
		liver		0.23	ND	3.45	0.955
		heart		ND	ND	2.01	0.594
		muscle	3	ND	ND	1.04	0.805
3	White Oak Creek, Bldg. 4505	whole body	13,690				
		feathers		0.34	4.88	7.29	2.72
		kidney	69	1.53	0.42	6.01	26.8
		liver	76	0.90	0.40	7.5	17.6
		heart	81	ND	ND	2.2	9.52
		muscle	151	ND	0.58	1.84	6.34

[a]ND= Nondetect: As- <0.40 mg/kg, Cd- <0.20 mg/kg, Pb- <0.40 mg/kg, and Se-<0.40 mg/kg.

Table C.20. Summary of number of locations where HQs>1 were observed

Watershed	Endpoint	Analyte	No. locations where NOAEL-based HQ>1	No. locations where LOAEL-based HQ>1
Bear Creek	Mink	Hg	2	0
		PCBs	2	0
	River Otter	Hg	4	3
		PCBs	4	1
	Kingfisher	Hg	5	3
		PCBs	5	1
	Heron	Hg	5	0
		PCBs	3	0
East Fork Poplar Creek	Mink	Hg	7	6
		PCBs	3	1
	Otter	Hg	7	7
		PCBs	6	2
	Kingfisher	Hg	7	7
		PCBs	7	3
	Heron	Hg	7	6
		PCBs	5	1
K-25	Mink	Hg	9	1
		PCBs	7	0
	Otter	Hg	13	11
		PCBs	15	5
	Kingfisher	Hg	16	10
		PCBs	14	4
	Heron	Hg	16	0
		PCBs	13	0
	Osprey	Hg	16	3
		PCBs	14	1
White Oak Creek	Mink	Hg	1	0
		PCBs	3	0
	Otter	Hg	7	3

Table C.20 (continued)

Watershed	Endpoint	Analyte	No. locations where NOAEL-based HQ>1	No. locations where LOAEL-based HQ>1
		PCBs	6	3
	Kingfisher	Hg	7	3
		PCBs	7	1
	Heron	Hg	7	0
		PCBs	5	0
	Osprey	Hg	2	0
		PCBs	3	0

Table C.21. Summary of number of individuals of piscivore endpoint species estimated to be experiencing adverse effects by watershed and for the ORR

Location	Analyte	Species	%> LOAEL	Number in Watershed	Number Adversely Affected	Percent Adversely Affected
Bear Creek	Mercury	Mink	<5%	7	0	0%
East Fork Poplar Creek	Mercury	Mink	80%	15	12	80%
K-25	Mercury	Mink	<5%	14	0	0%
White Oak Creek	Mercury	Mink	<5%	4	0	0%
ORR-wide	Mercury	Mink		40	12	30%
Bear Creek	Mercury	Otter	50%	5	2	40%
East Fork Poplar Creek	Mercury	Otter	>95%	9	9	100%
K-25	Mercury	Otter	>95%	9	9	100%
White Oak Creek	Mercury	Otter	25%	2	0	0%
ORR-wide	Mercury	Otter		25	20	80%
Bear Creek	Mercury	Kingfisher	65-70%	5	3	60%
East Fork Poplar Creek	Mercury	Kingfisher	>95%	10	10	100%
K-25	Mercury	Kingfisher	>95%	9	9	100%
White Oak Creek	Mercury	Kingfisher	15%	3	0	0%
ORR-wide	Mercury	Kingfisher		27	22	81.5%
Bear Creek	Mercury	Heron	<5%	29	0	0%
East Fork Poplar Creek	Mercury	Heron	>95%	57	57	100%
K-25	Mercury	Heron	<5%	54	0	0%
White Oak Creek	Mercury	Heron	<5%	15	0	0%
ORR-wide	Mercury	Heron		155	57	36.8%
K-25	Mercury	Osprey	<5%		0	0%
White Oak Creek	Mercury	Osprey	<5%		0	0%
Bear Creek	PCBs	Mink	<5%	7	0	0%

Table C.21 (continued)

Location	Analyte	Species	%> LOAEL	Number in Watershed	Number Adversely Affected	Percent Adversely Affected
East Fork Poplar Creek	PCBs	Mink	<5%	15	0	0%
K-25	PCBs	Mink	<5%	14	0	0%
White Oak Creek	PCBs	Mink	<5%	4	0	0%
ORR-wide	PCBs	Mink		40	0	0%
Bear Creek	PCBs	Otter	<5%	5	0	0%
East Fork Poplar Creek	PCBs	Otter	15-20%	9	1	11%
K-25	PCBs	Otter	5-10%	9	0	0%
White Oak Creek	PCBs	Otter	70-75%	2	1	50%
ORR-wide	PCBs	Otter		25	2	8%
Bear Creek	PCBs	Kingfisher	<5%	5	0	0%
East Fork Poplar Creek	PCBs	Kingfisher	10-15%	10	0	0%
K-25	PCBs	Kingfisher	<5%	9	0	0%
White Oak Creek	PCBs	Kingfisher	<5%	3	0	0%
ORR-wide	PCBs	Kingfisher		27	0	0%
Bear Creek	PCBs	Heron	<5%	29	0	0%
East Fork Poplar Creek	PCBs	Heron	<5%	57	0	0%
K-25	PCBs	Heron	<5%	54	0	0%
White Oak Creek	PCBs	Heron	<5%	15	0	0%
ORR-wide	PCBs	Heron		155	0	0%
K-25	PCBs	Osprey	<5%		0	0%
White Oak Creek	PCBs	Osprey	<5%		0	0%

Table C.22. Simulation of exposure of mink to mercury and PCBs in toxicity test diets

Diet	Analyte	Concentration in diet				Distribution used in simulation	Modeled exposure (mg/kg-d)			% > NOAEL [a]	% > LOAEL [a]
		Mean	STD	Min	Max		Mean	STD	80th percentile		
A	Mercury	0.02	0	0.02	0.03	Triangular	0.0034	0.0009	0.0042	<5%	<5%
B	Mercury	0.05	0	0.04	0.06	Triangular	0.0074	0.0019	0.0088	<5%	<5%
C	Mercury	0.09	0	0.08	0.11	Triangular	0.0138	0.0035	0.016	30%	<5%
D	Mercury	0.15	0.01			Normal	0.022	0.0059	0.026	>95%	25%
E	Mercury	0.22	0.01			Normal	0.033	0.008	0.038	>95%	85%
A	PCB 1260	0.169	0.002			Normal	0.025	0.0063	0.029	<5%	<1%
B	PCB 1260	11.44	0.327			Normal	1.70	0.43	1.97	>95%	>95%
C	PCB 1260	4.697	0.174			Normal	0.698	0.18	0.82	>95%	40-45%
D	PCB 1260	10.41	0.25			Normal	1.54	0.39	1.79	>95%	>95%
E	PCB 1260	20.67	0.458			Normal	3.07	0.77	3.55	>95%	>95%

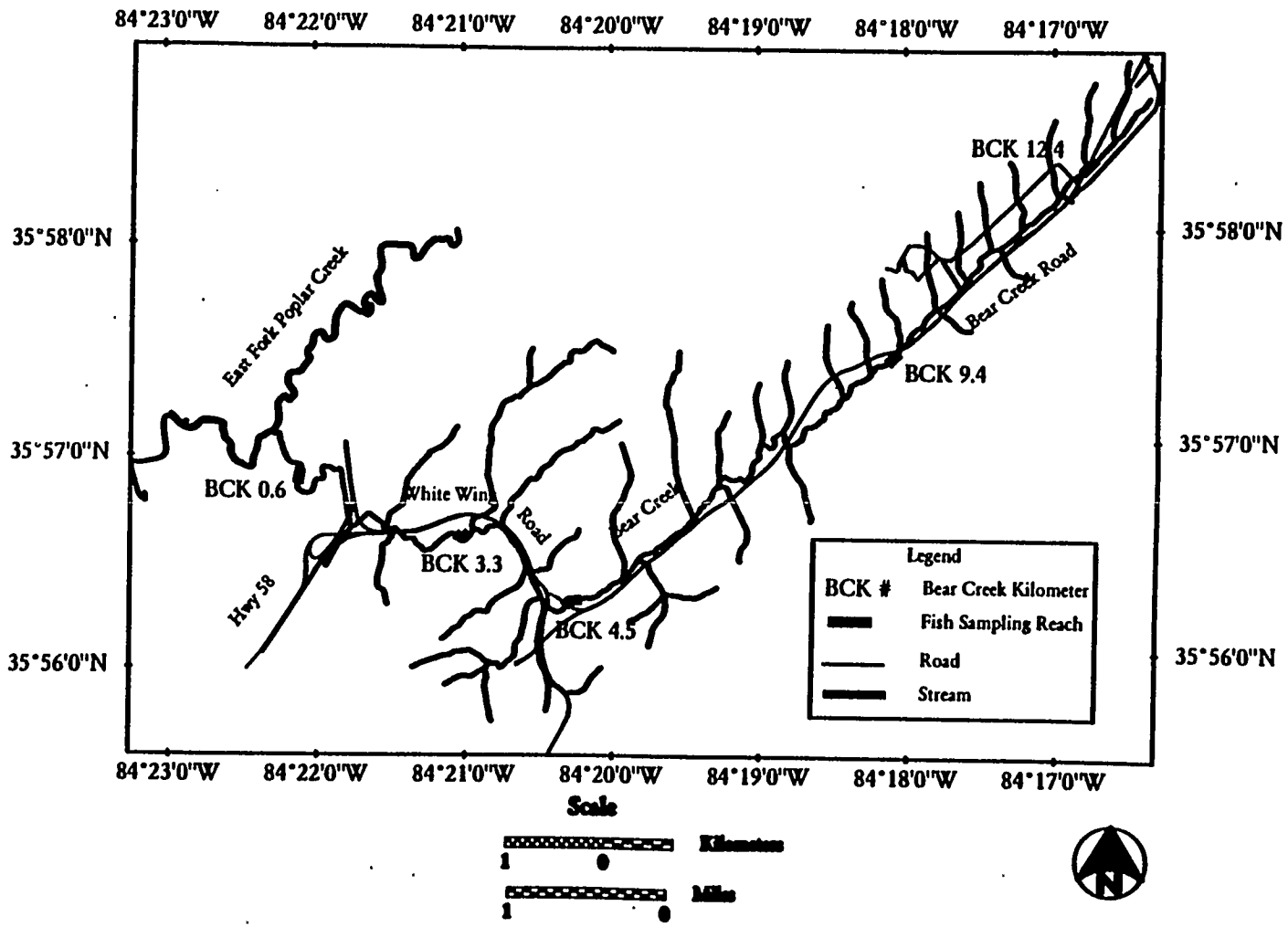

Fig. C.1. The Bear Creek fish sampling locations used in the ORR-wide ecological risk assessment. The map projection is Tennessee State Plane (TSP) meters, Zone 5301, and NAD 83. The study site location data is from BMAP and OREIS spatial database. The map was prepared by R.A. Washington-Allen, Environmental Sciences Division (ESD) ORNL on July 27, 1995.

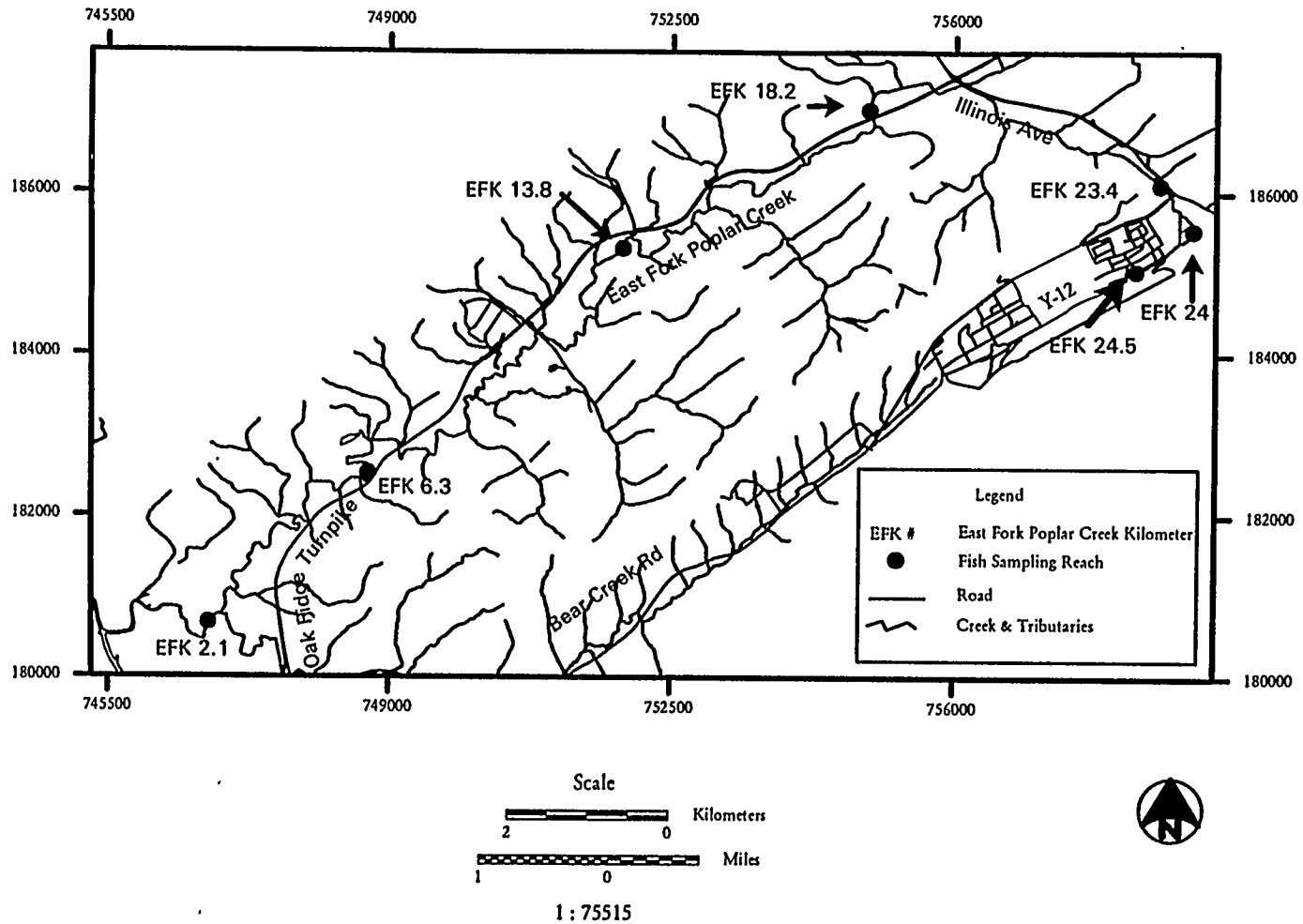

Fig. C.2. The East Fork Poplar Creek fish and ecological sampling locations (from EFPC RI) used in the ORR-wide ecological risk assessement. The map projection is Tennessee State Plane meters, Zone 5301, and NAD 83. The study site location data is from SAIC, BMAP, and OREIS spatial database. The map was prepared by R. A. Washington-Allen, Environmental Sciences Division, ORNL on September 11, 1996.

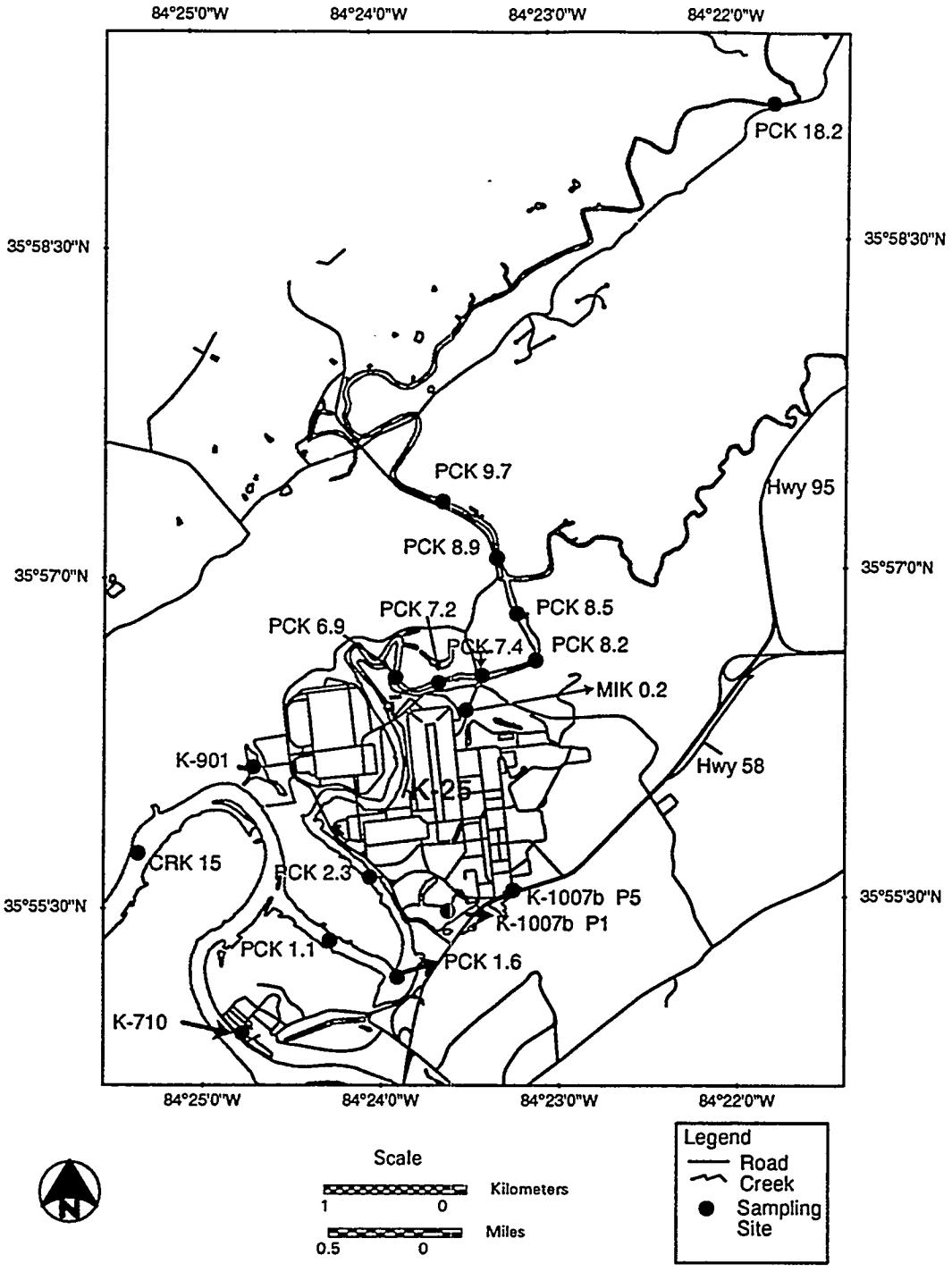

Fig. C.3. The Poplar Creek sampling locations and ponds used to evaluate risks in the K-25 vicinity for the ORR-wide ecological risk assessment. The map projection is Tennessee State Plane meters, Zone 5301, and NAD 83. The study site location data is from BMAP and OREIS spatial database. The map was prepared by R. A. Washington-Allen, Environmental Sciences Division, ORNL on September 11, 1996.

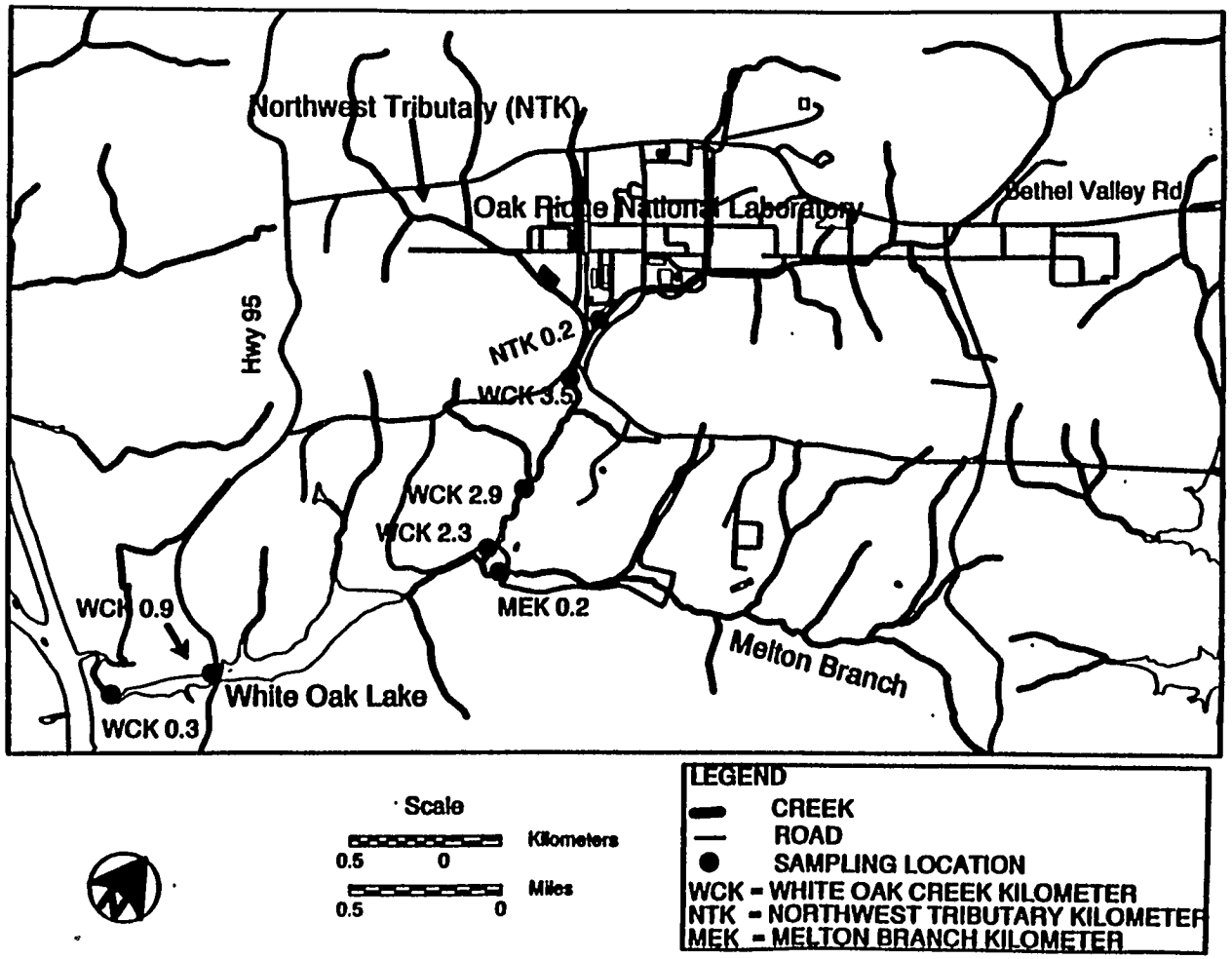

Fig. C.4. The White Oak Creek fish sampling locations used in the ORR-wide ecological risk assessment. The map projection is Tennessee State Plane (TSP) meters, Zone 5301, and NAD 83. The study site location data is from BMAP and OREIS spatial database. The map was prepared by R.A. Washington-Allen, Environmental Sciences Division (ESD) ORNL on July 27, 1995.

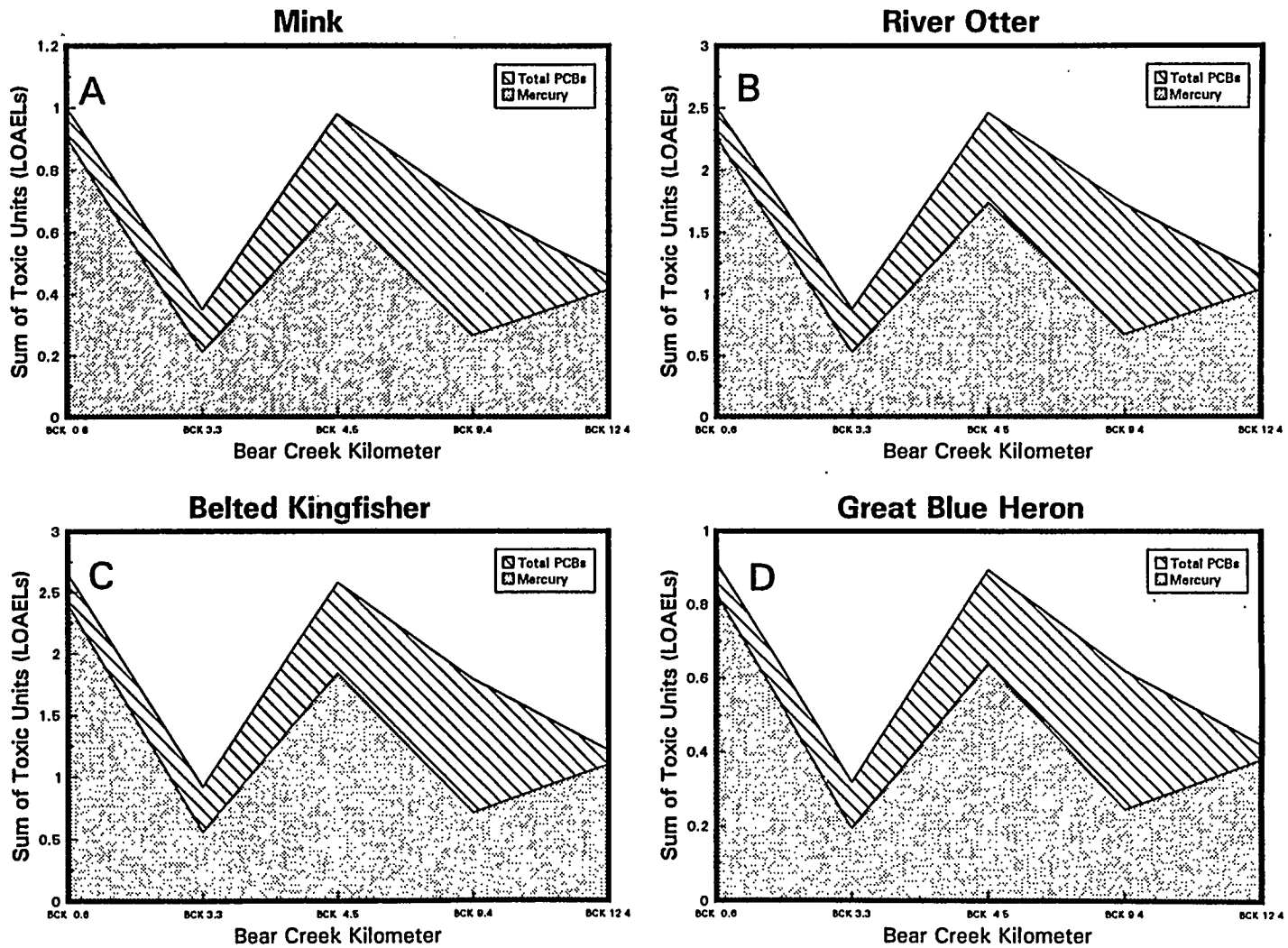

Fig. C.5. Sum of LOAEL-based toxic units for evaluation of risks to piscivores in the Bear Creek Watershed.

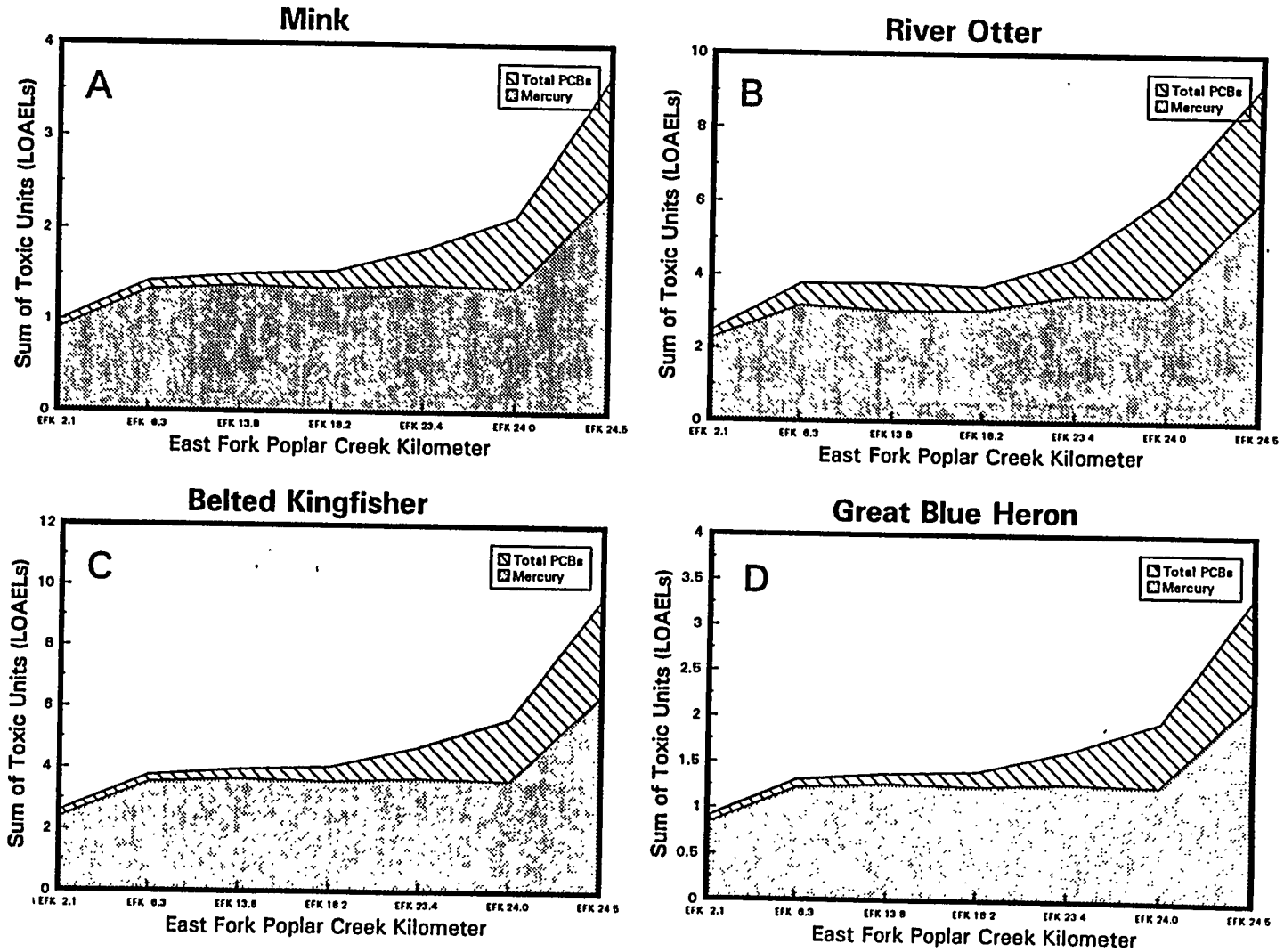

Fig. C.6. Sum of LOAEL-based toxic units for evaluation of risks to piscivores in the East Fork Poplar Creek Watershed.

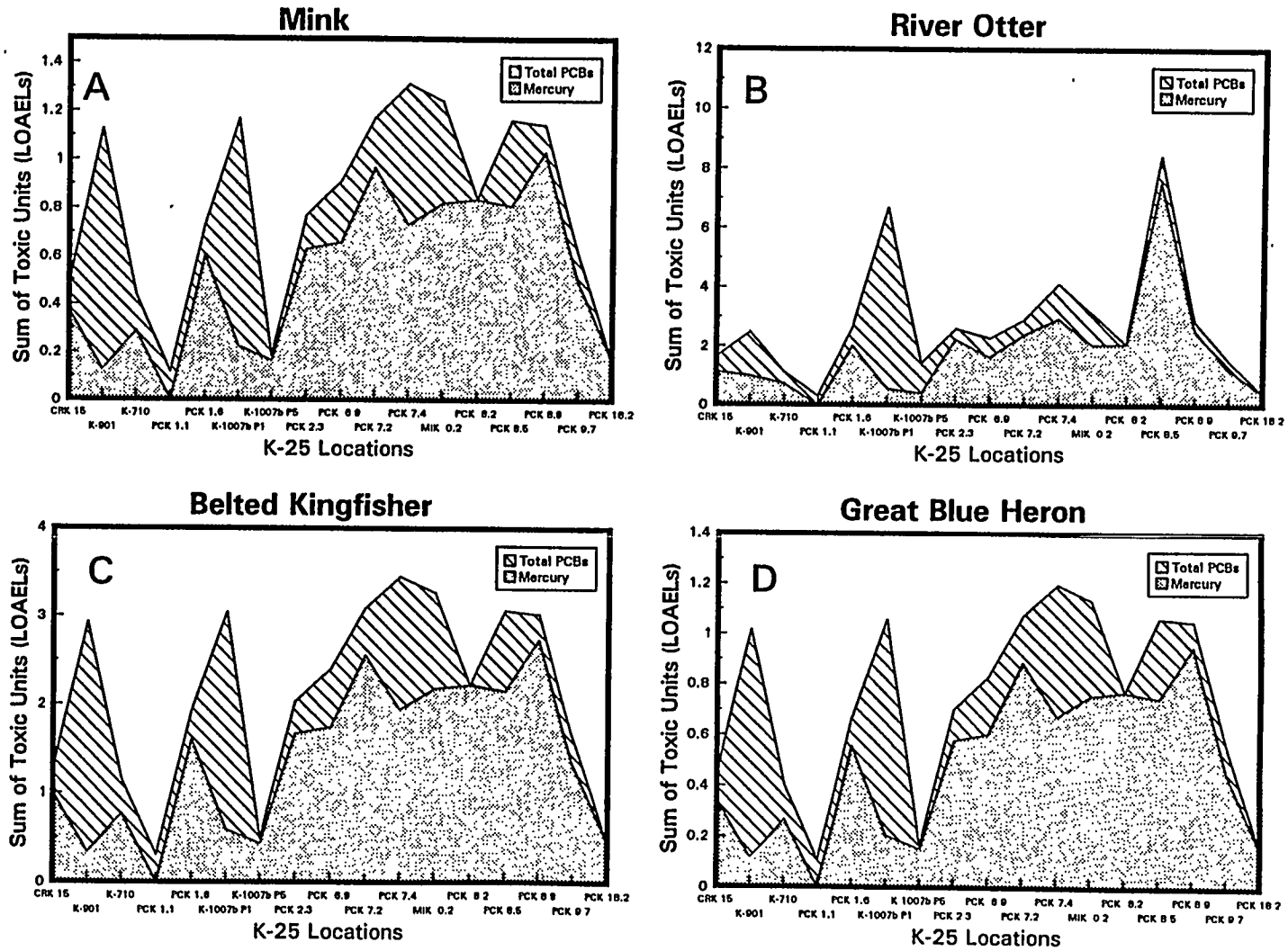

Fig. C.7. Sum of LOAEL-based toxic units for evaluation of risks to piscivores in the K-25 vicinity.

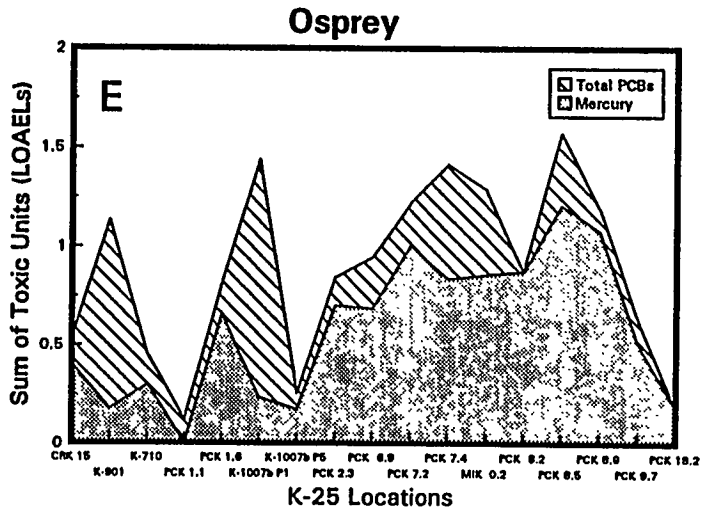

Fig. C.7 (continued)

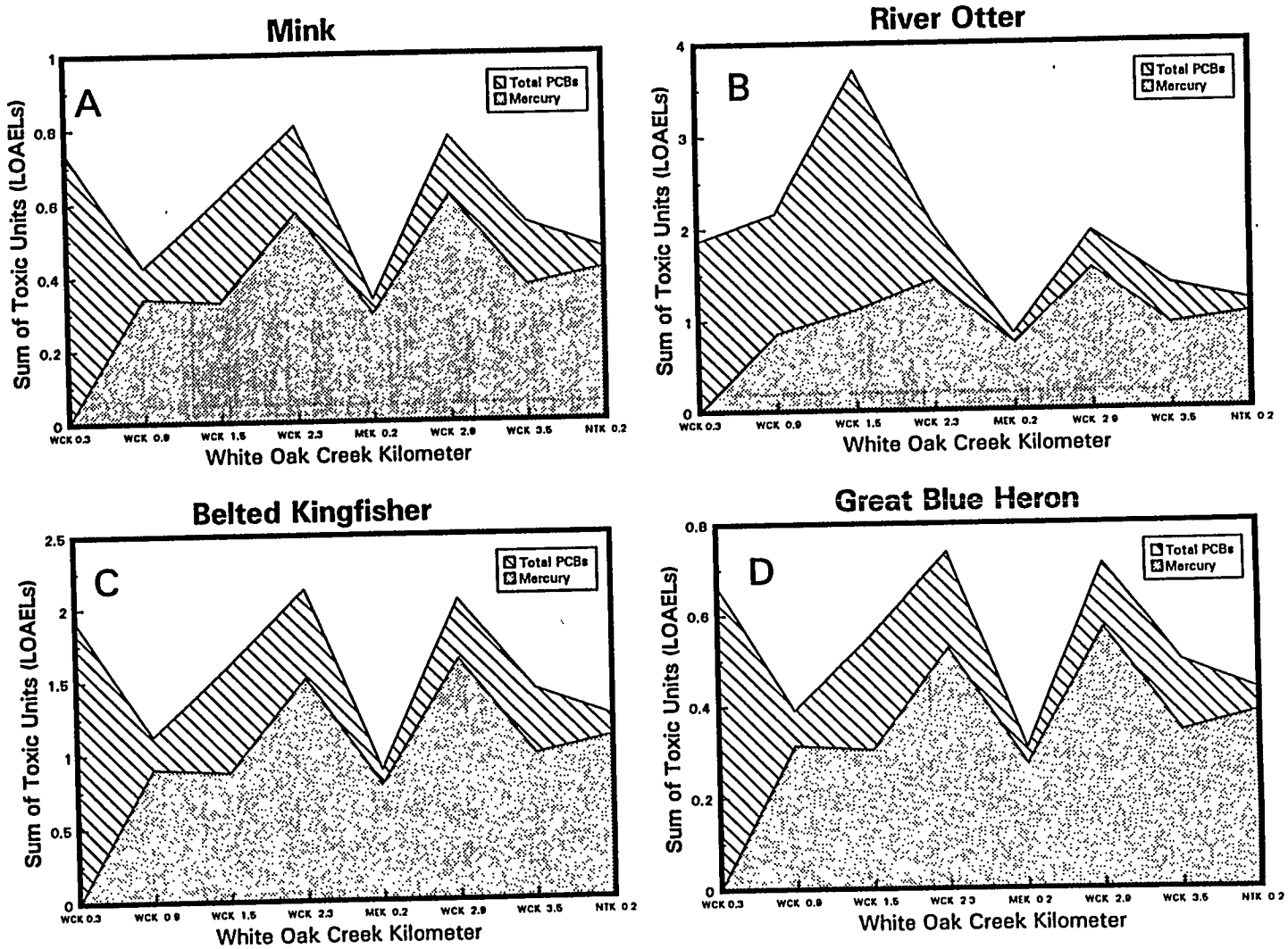

Fig. C.8. Sum of LOAEL-based toxic units for evaluation of risks to piscivores in the White Oak Creek Watershed.

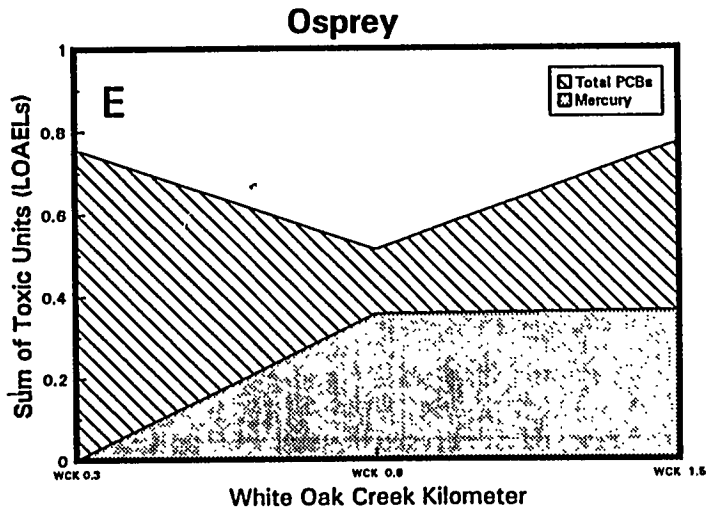

Fig. C.8 (continued)

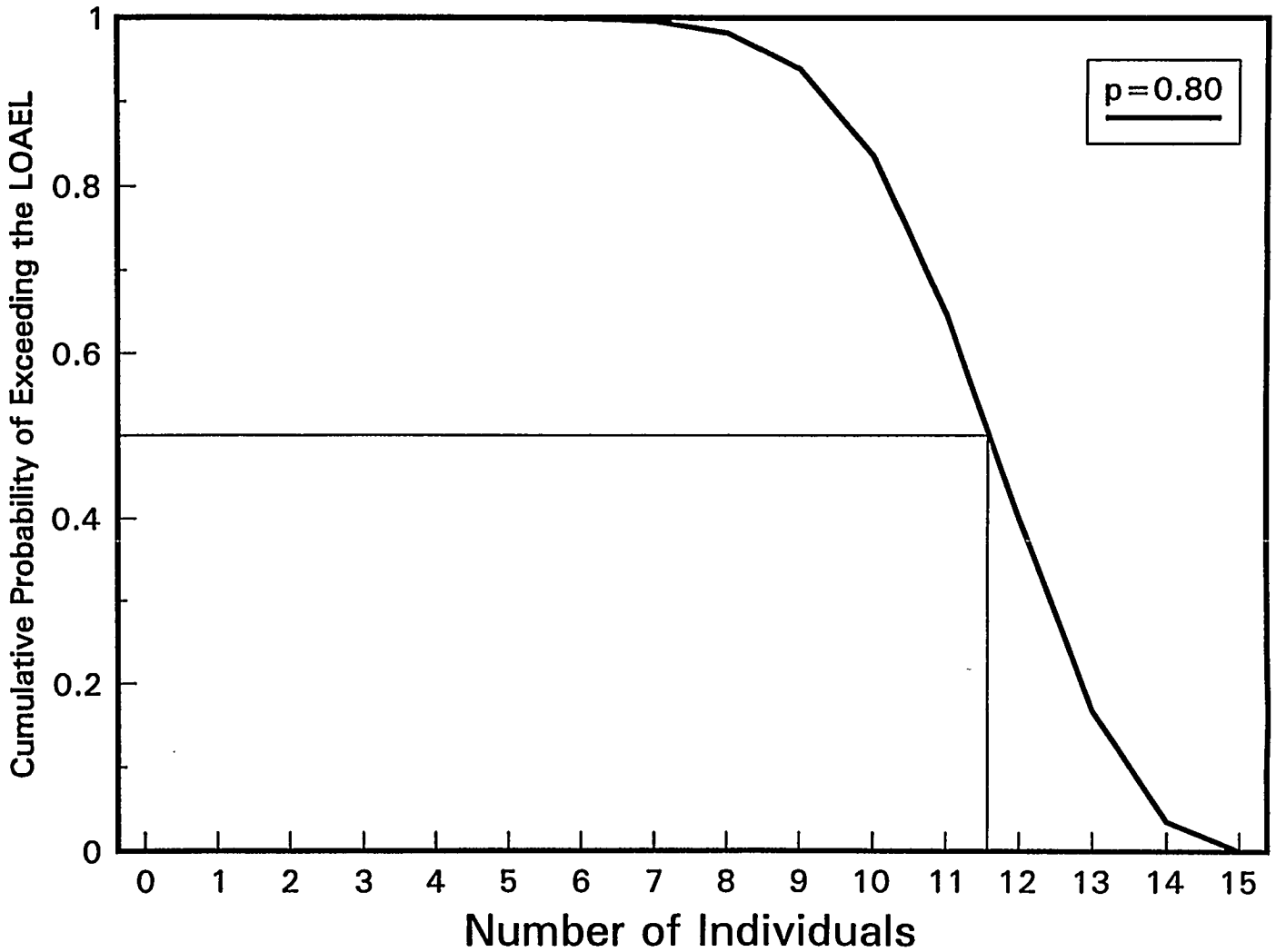

Fig. C.9. Cumulative binomial probability of mink experiencing exposure to mercury in East Fork Poplar Creek in excess of the LOAEL.

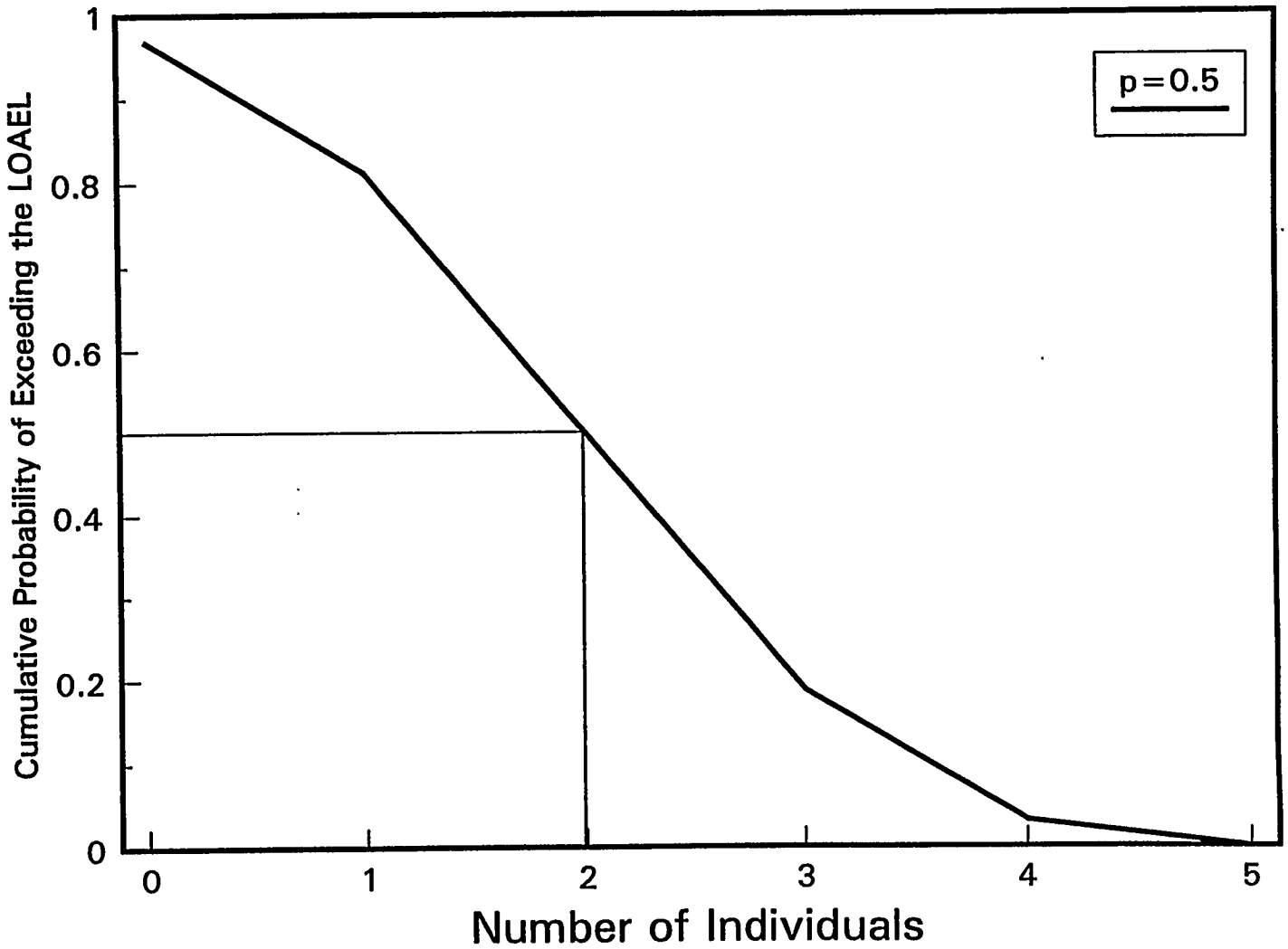

Fig. C.10. Cumulative binomial probability of river otter experiencing exposure to mercury in Bear Creek in excess of the LOAEL.

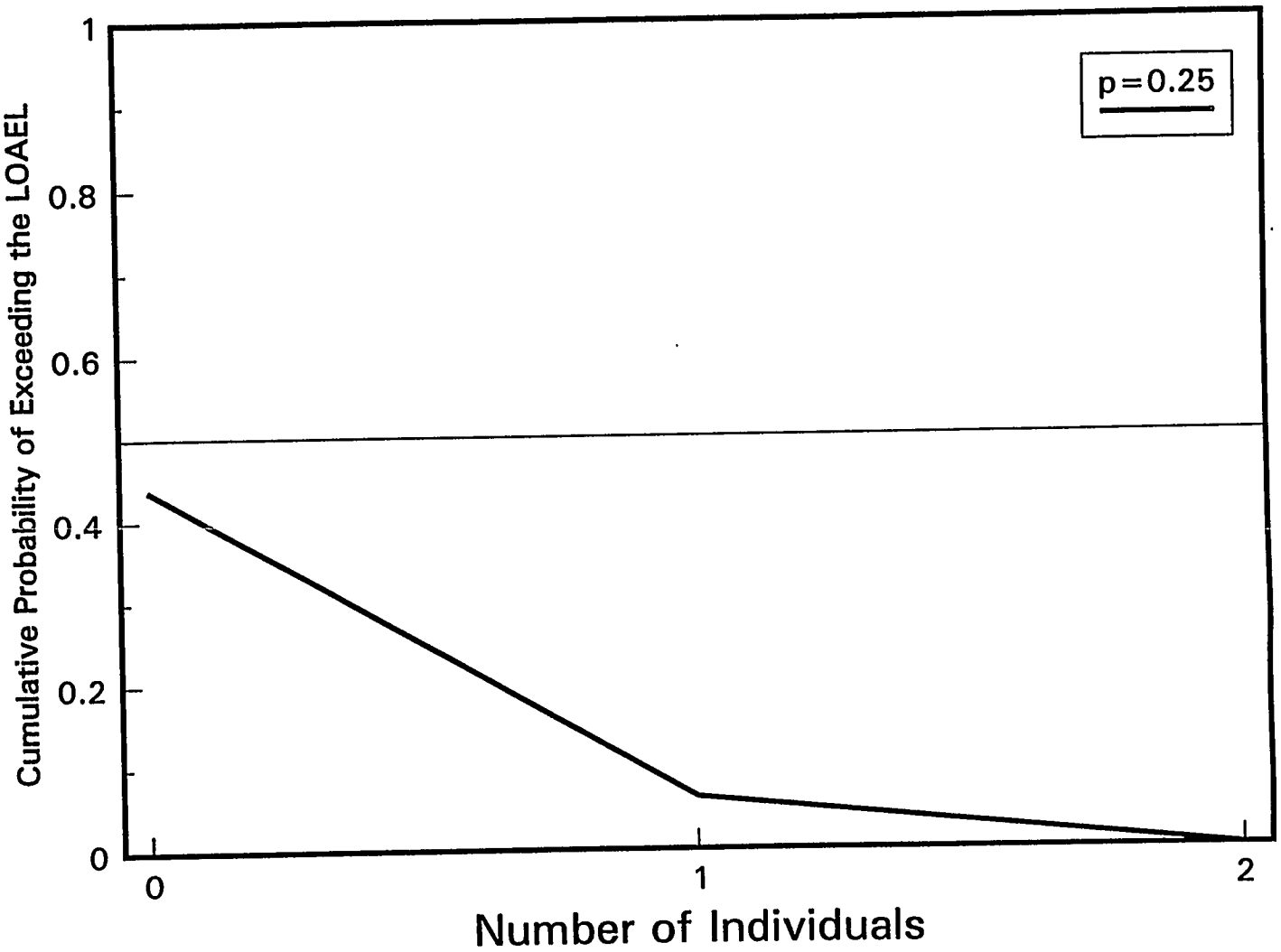

Fig. C.11. Cumulative binomial probability of river otter experiencing exposure to mercury in White Oak Creek in excess of the LOAEL.

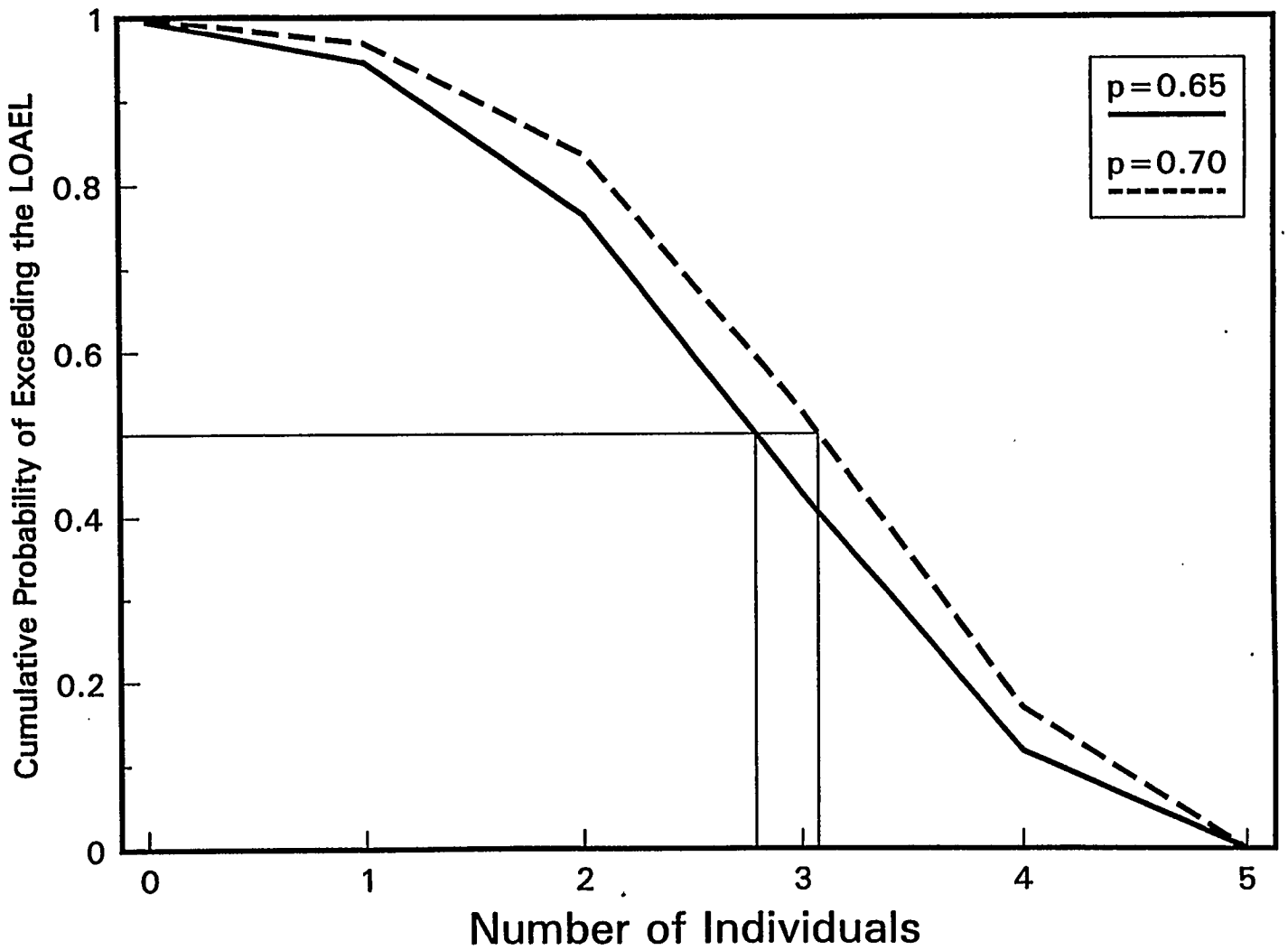

Fig. C.12. Cumulative binomial probability of belted kingfisher experiencing exposure to mercury in Bear Creek in excess of the LOAEL.

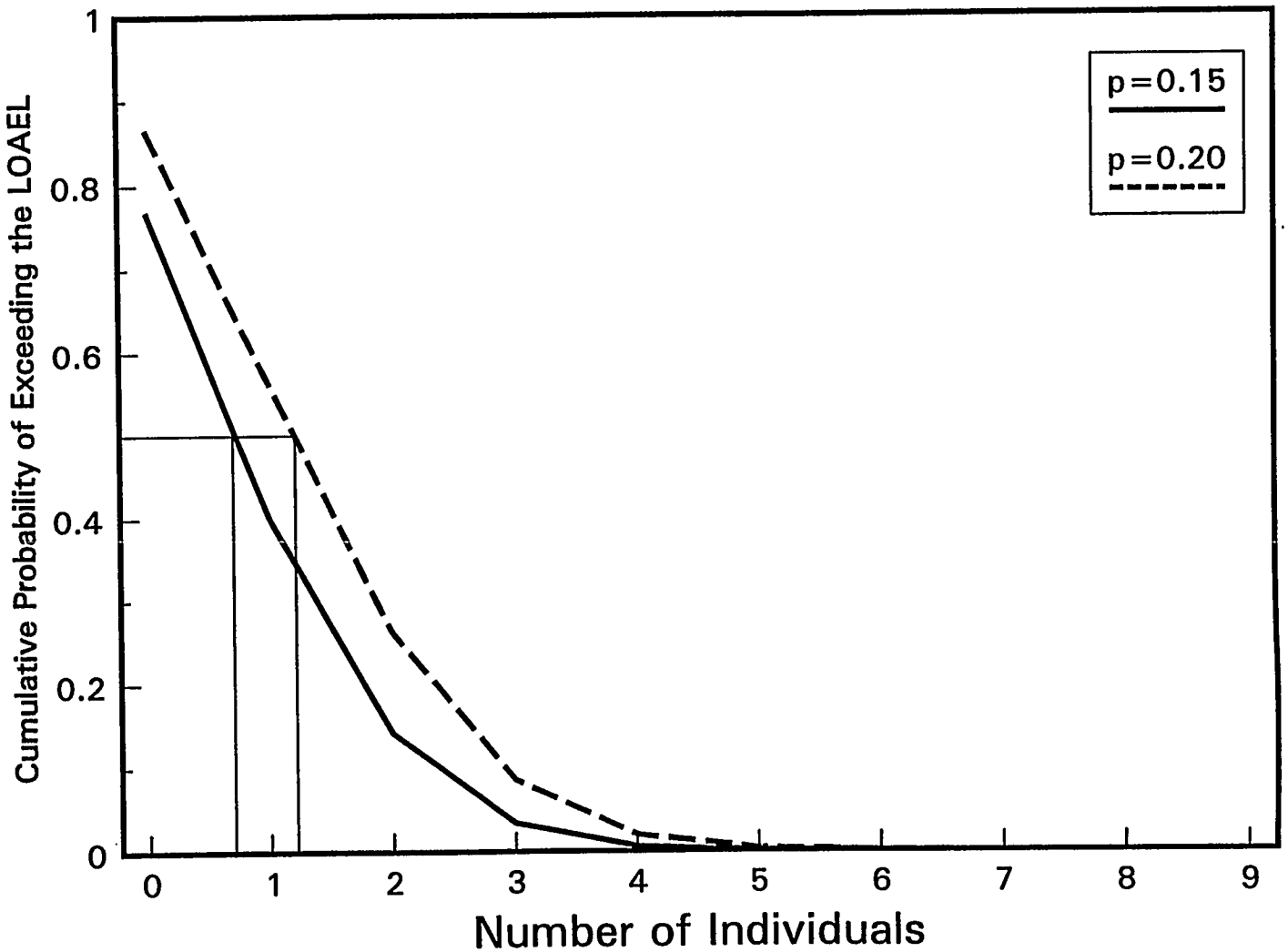

Fig. C.13. Cumulative binomial probability of river otter experiencing exposure to PCBs in East Fork Poplar Creek in excess of the LOAEL.

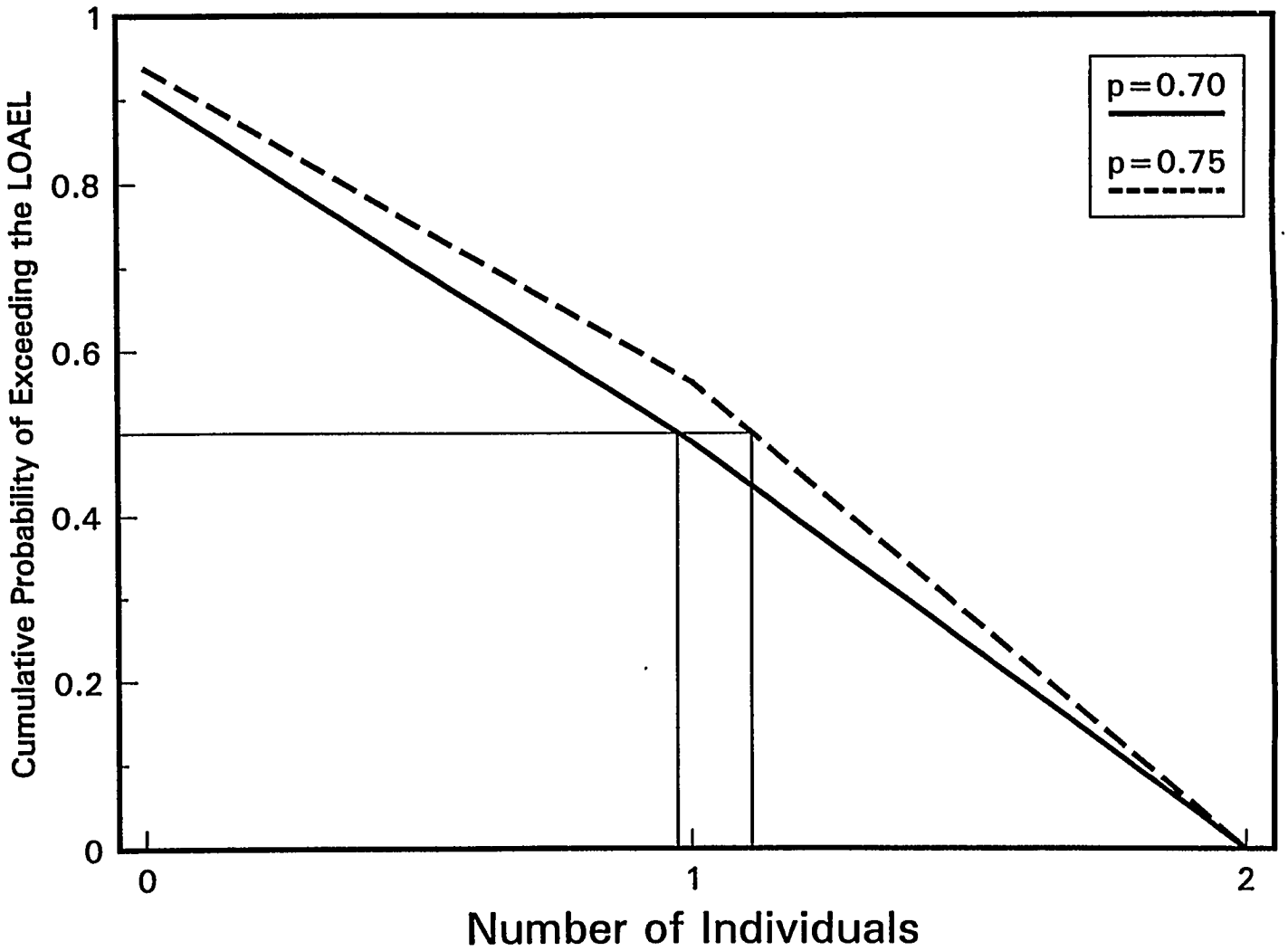

Fig. C.14. Cumulative binomial probability of river otter experiencing exposure to PCBs in White Oak Creek in excess of the LOAEL.

Appendix D

TOXICOLOGICAL PROFILES

TOXICOLOGICAL PROFILES

Aluminum. Aluminum is an ubiquitous metal, being the third most abundant element in the earth's crust (Krueger et al. 1984). Relative to other metals, the toxicity of aluminum is low (Sorensen et al. 1974). The oral LD_{50} for mice ranges from 770 to 980 mg aluminum/kg body weight (Ondreicka et al. 1966). The principal effect of aluminum is to interfere with phosphorous metabolism; in the alimentary canal, aluminum forms insoluble compounds with phosphorous resulting in an imbalance of calcium and phosphorous (Carrerie et al. 1986). Other effects of aluminum include neurotoxicity. Rats exposed to aluminum display behavioral abnormalities and have reduced acetylcholinesterase activity (Krueger et al. 1984). Mice consuming diets containing 500 to 1000 ppm aluminum displayed ataxia and paralysis of the hind limbs (Golub et al. 1987). In humans, aluminum has been associated with several degenerative diseases of the nervous system, including Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis (Ganrot 1986).

Ondreicka et al. (1966) evaluated the effects of aluminum on mammalian reproduction. Mice received 19.3 mg aluminum/kg bodyweight/day (as $AlCl_3$) in drinking water for three generations. While the number of litters and offspring per litter was not reduced, growth was significantly reduced among all offspring in the second and third generations. In a similar study, rats received daily intragastric doses of 0, 180, 360, or 720 mg aluminum/kg body weight/day (Domingo et al. 1987) for one generation. Growth and survival of young was reduced among the groups that received 360 and 720 mg aluminum/kg/day. Other studies also report that while aluminum does not appear to affect the number of litters or number of offspring/litter, growth and survival of offspring of aluminum exposed parents is reduced (Golub et al. 1987; Paternain et al. 1988).

Due to it's interference with phosphorous and calcium metabolism, it has been suggested that aluminum may impair eggshell formation by birds, resulting in eggshell thinning (Nyholm 1981). To test this hypothesis, Carriere et al. (1986) fed breeding ring doves (*Streptopelia risoria*) a diet containing 1000 ppm aluminum (and adequate but reduced calcium and phosphorous) and observed reproduction. While no reproductive effects or embryonic malformations were observed at this dosage level, significant reproductive effects resulted when birds were fed a diet deficient in calcium and phosphorous that contained 750 ppm aluminum. Therefore, among birds it appears that the manifestation of toxic effects of aluminum are dependent upon the nutritional quality of their diet.

Arsenic. Arsenic is present in the earth's crust at approximately 2 ppm, but tissues of animals generally contain an average of <0.5 ppm (Venugopal and Luckey 1978). Arsenic may be a required micronutrient; growth, survival, and reproduction of goats is poor if the diet contains <0.05 ppm As (NAS 1977).

Arsenic is a carcinogen and teratogen. Other effects include reduced growth, hearing/sight loss, liver/kidney damage, and death (Eisler 1988a). Inorganic arsenic is usually more toxic than organic arsenic compounds. Wildlife mortality and malformations have been observed for chronic doses of 1-10 mg As/kg bw and dietary concentrations of 5-50 ppm (Eisler 1988a). Acute LD_{50}s for mammals of 35–100 mg calcium arsenate/kg body weight and 10-50 mg lead arsenate/kg body weight have been reported (NRCC 1978).

Schroeder and Mitchner (1971) exposed mice to 5 ppm sodium arsenite in drinking water for three generations. While mice fed arsenic survived well, litter size decreased in subsequent generations. A dose of 0.38 mg arsenic/kg over a lifetime was sufficient to cause a slight decrease

in the median lifespan of laboratory mice (Schroeder and Balassa 1967), but it had no effect on growth. As little as 3 mg arsenic trioxide/kg body weight or 1 mg sodium arsenite/kg body weight can be lethal (NAS 1977).

Because metabolism of arsenic in rats is unlike that in other animals, results of toxicity studies using rats generally should not be extrapolated to other species (Eisler 1988a).

Among birds, LD_{50}s for arsenic compounds range from 17.4 to 3300 mg/kg bw (Eisler, 1988a). While no mortality was observed among mallard ducks fed a diet containing 100 ppm sodium arsenite for 128 days, 12% to 92% mortality was observed for ducks fed diets containing 250 to 1000 ppm arsenite (USFWS 1964). Camardese et al. (1990) and Whitworth et al. (1991) fed mallards diets containing 30, 100, or 300 ppm sodium arsenate. While no effects were observed on behavior, growth was reduced for male ducks consuming 300 ppm arsenic and for female ducks at all exposure levels.

Barium. The soluble salts of barium, an alkaline earth metal, are toxic in mammalian systems. At low doses, barium acts as a muscle stimulant and at higher doses affects the nervous system eventually leading to paralysis. The LD_{50} for rats is listed as 630 mg/kg for barium carbonate, 118 mg/kg for barium chloride, and 921 mg/kg for barium acetate (Lewis and Sweet 1984).

Schroeder and Mitchener (1975a, b) exposed rats and mice to 5 mg barium/L in drinking water for their lifetime. There was a slight but significant reduction in longevity of treated male mice when measured as the mean age at death of the last surviving 10% of animals. The overall average life span of the group, however, was about the same as the control group. In another study, Perry et al. (1983) exposed rats to 0, 1, 10, or 100 ppm barium for up to 16 months. A significant increase in average blood pressure was observed in the highest dose group; a slight but statistically significant increase was seen in the 10 ppm dose group. Information on developmental and reproductive toxicity of barium to mammals is not available.

The LD_{50} of barium to chickens is 623 mg/kg (Johnson et al. 1960). Johnson et al. (1960) report that while chickens will tolerate 1000 ppm barium in their diet without adverse effects, 2000 ppm reduces growth, 8000 ppm produces 50% mortality in 4 weeks, and diets containing 16,000 or 32,000 ppm barium are 100% lethal.

Cadmium. While there is little information to indicate that this relatively rare metal is biologically essential or beneficial, Cd has been suggested as the cause of various deleterious effects to wildlife (Eisler 1985a). Mammals and birds are comparatively resistant to the biocidal properties of Cd, which include growth retardation, anemia, and testicular damage. Cd tends to bioaccumulation the liver and kidney, eventually acting as a cumulative toxin. Cd residues of 2 ppm whole body fresh weight are evidence of Cd contamination, and residues >5 ppm whole animal fresh weight may be life-threatening (Eisler 1985a).

The lowest oral dose resulting in death for rats was 250 mg Cd/kg body weight (EPA 1980a). Weigel et al. (1987) fed rats 0.24, 0.85, or 2.25 mg/kg Cd in diet for 8 weeks. Concentrations ≥0.85 mg/kg resulted in reduced food intake, reduced body weights, and reduced enzyme activity, but no hematological effects were noted. Ma et al. (1991) determined that an average cadmium intake of 15 mg/kg/day corresponded with critical renal metal loads of 120 mg/kg, a level indicative of adverse health effects. Rats on a diet with 5 ppm Cd suffered shortened lifespans (Schroeder et al. 1965). Cd at 50 ppm in the diet depleted iron from rat livers (Whanger 1973). Rats eating diets with

7.15 ppm Cd (as CdO) exhibited growth reductions, but those consuming a diet with 2.80 ppm Cd did not (Weigel et al. 1987). In a 3 generation reproductive study, the population of mice exposed to 1 ppm $CdCl_2$ in their drinking water died out after the second generation (Schroeder and Mitchner 1971). Rats receiving >6 mg Cd/kg body weight daily during pregnancy gave birth to malformed fetuses (Ferm and Layton 1981).

No mortality was observed among adult mallard ducks fed diets containing 0, 2, 20, and 200 ppm Cd, however egg production was significantly reduced in the group consuming 200 ppm Cd (White and Finley 1978). In addition, the testes of males in the 200 ppm Cd group atrophied and the spermatogenic process was disrupted (White et al. 1978). Among mallard ducklings, 20 ppm Cd in the diet produces mild to severe kidney lesions, reduces packed cell volume and hemoglobin concentrations in the blood (Cain et al. 1983). Avoidance behavior of black ducklings is impaired by diets containing 40 ppm Cd (Heinz and Haseltine 1983).

Copper. Copper occurs naturally in elemental form and as a component of many minerals. It is an essential nutrient that is normally present in a wide variety of tissues (ATSDR 1990; EPA 1987). Because of its high electrical and thermal conductivity, it is widely used in the manufacture of electrical equipment. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH 1986). The largest anthropogenic releases of copper to the environment result from mining operations, agriculture, solid waste, and sludge from sewage treatment plants. Natural discharges to air and water, such as windblown dust and volcanic eruptions, may be significant (ATSDR 1990).

Copper is a component of a number of metalloenzymes such as catalase, peroxidases, and cytochrome oxidase and is essential for the utilization of iron (Goyer 1991; Stokinger 1981a). Although most copper salts occur in two valence states, as cuprous (Cu^{+}) or cupric (Cu^{2+}) ions, the biological availability and toxicity of copper is most likely associated with the divalent state (ATSDR 1990). Copper sulfate is the most common copper salt. Copper is soluble in nitric acid and hot sulfuric acid, very slightly soluble in hydrochloric acid and ammonia, and insoluble in water (Stokinger 1981a).

The metabolism of copper involves mainly its transfer to and from various organic ligands, most notably sulfhydryl and imidazole groups on amino acids and proteins (ATSDR 1990). The liver is one of the main organs involved in the storage and metabolism of copper. Absorption of ingested copper occurs primarily in the upper gastrointestinal tract (EPA 1987). Soluble copper compounds (oxides, hydroxides, citrates) are readily absorbed but water-insoluble compounds (sulfides) are poorly absorbed (Venugopal and Luckey 1978). Zinc, molybdenum, and other metals may decrease dietary copper absorption (USAF 1990).

In animal studies, oral exposure to copper caused hepatic and renal accumulation of copper, liver and kidney necrosis at doses of ≥100 mg/kg/day, and hematological effects at doses of 40 mg/kg/day (EPA 1986; Haywood 1985; Rana and Kumar 1978; Gopinath et al. 1974; Kline et al. 1971). Oral or intravenous administration of copper sulfate can increase fetal mortality and developmental abnormalities in experimental animals (Lecyk 1980; Ferm and Hanlon 1974). Rat oral LD_{50} values for various copper compounds are 140 mg/kg for copper chloride ($CuCl_2$); 470 mg/kg for copper oxide (Cu_2O); 940 mg/kg for copper nitrate ($Cu(NO_3)_2 \cdot 3H_2O$); and 960 mg/kg for copper sulfate ($CuSO_4 \cdot 5\ H_2O$) (Stokinger 1981a). Deaths in animals given lethal doses of copper have been attributed to extensive hepatic centrilobular necrosis (USAF 1990).

In a 90-day subchronic study with copper cyanide (CuCN), high mortality, attributed to hemolytic anemia, was seen in both male and female rats receiving 50 mg/kg/day by gavage, but not in those receiving ≤ 5 mg/kg/day (EPA 1986). In general, male rats appeared to be more sensitive to the effects of CuCN than female rats. Rats receiving 500 ppm copper in their diet (about 5 mg/day) appeared normal, while rats receiving 1000 ppm exhibited depressed growth, those at 2000 ppm hardly grew at all, and those on a 4000 ppm diet lost weight rapidly and died (Boyden et al. 1938). Salt licks containing 5–9% copper sulfate caused anorexia, hemolytic anemia, icterus, and hemoglobinuria, followed by death within 2 days in sheep using the licks (Gopinath et al. 1974). The estimated ingested dose was 40–49 g over a 25- to 86-day period. Lecyk (1980) observed reduced litter size, decreased fetal weights, and skeletal abnormalities in the offspring of mice fed diets supplemented with 3000 or 4000 ppm copper sulfate (155 or 207 mg copper/kg/day, respectively) for one month prior to gestation and on days 0–19 of gestation.

Aulerich et al. (1982) reported an increased mortality rate in the offspring of minks fed a diet supplemented with >3 mg copper/kg/day as copper sulfate for 50 weeks. Although kit mortality was greater and litter mass was reduced relative to controls, reproductive performance of mink fed diets supplemented with up to 200 ppm copper for 357 days was within the normal range for the species (Aulerich et al. 1982). Lifetime exposure to 42.4 mg copper/kg/day (as copper gluconate) in drinking water caused a 12.8% decrease in the maximal lifespan in mice (Massie and Aiello 1984).

Domestic chicks on diets ≥324 ppm copper grew slowly; mortality increased with dietary copper concentrations of 1270 ppm (Mayo et al. 1956). Arthur et al. (1958) observed no ill effects in chicks fed ≤500 ppm copper in diet up to 8 weeks of age. Dietary copper levels from 588–1176 ppm for 10 weeks exerted a toxic effect on chick growth; the minimum toxic level of copper appeared to be about 500 ppm (Mehring et al. 1960). Turkey poults tolerated 676 ppm copper in starter diets for 21 days with no deleterious effects, but copper was definitely toxic at levels >1620 ppm (Vohra and Kratzer 1968). Chickens given a daily dose of >70 mg/kg of $CuCO_3$ died while those receiving <60 mg/kg exhibited slight symptoms of copper poisoning but survived (Pullar 1940). No symptoms of copper poisoning were observed in domestic mallards ingesting ≤29 mg/kg/day of $CuCO_3$, but daily intakes ≥55 mg/kg/day were toxic (Pullar 1940).

Chromium. Chromium occurs as either chromium (III) or chromium (VI). Trivalent chromium is an essential metal in man and wildlife, playing an important role in insulin metabolism (Larngard and Norseth 1979). Hexavalent chromium is more toxic than chromium (III) because of its high oxidation potential and the ease with which it penetrates biological membranes (Steven et al. 1976; Taylor and Parr 1978). However, it is unlikely that all chromium in soil would be chromium (VI) because it is a highly oxidizing chemical species which is usually reduced by soil organic matter to chromium (III). Chromium (III) solubility decreases with increasing pH, and it is completely precipitated at pH above 5.5. In most soils, chromium is primarily present as precipitated chromium (III) and is not bioavailable. Most chromium in soil and sediments is unavailable to living organisms, and there is little evidence of chromium biomagnifying through food chains in its inorganic form (Eisler 1986a). Concentrations of total chromium >4.0 mg/kg dry weight should be viewed as presumptive evidence of chromium contamination (Eisler 1986a).

At high concentrations, chromium is a mutagen, teratogen and carcinogen (Eisler 1986a). The LD_{50} for chromium (III) in mice is 260 mg/kg bw and 5 mg/kg bw for chromium (VI) (Steven et al. 1976). Rats fed chromium (VI) reached a toxic threshold at 1000 ppm (Steven et al. 1976). Pregnant hamsters injected with 5 to 15 mg CrO_3 [chromium (VI)]/kg bw displayed a dose-dependent increase in the number of resorbed and malformed fetuses (Gale, 1978). Guinea pigs fed

50 ppm chromium (III) for 21 weeks showed no adverse effects (Preston et al. 1976). Similar results were observed among rats consuming water containing 25 ppm chromium (VI) for 1 year (Mackenzie et al. 1958).

Injection of 0.002 to 0.05 mg CrO_3 [chromium (VI)]/chicken egg produced a dose-dependent decrease in egg viability and increased frequency of malformed embryos (Gilani and Marano, 1979). In contrast, adult black ducks fed a diet containing 0, 10, or 50 ppm chromium (III) for 10 months displayed normal growth and reproduction (Haseltine et al., unpublished manuscript). While no malformations were observed among ducklings from treated birds, growth and survivorship was reduced. Heinz and Haseltine (1981) observed no effects on avoidance behavior of black ducklings fed a diet containing 20 or 200 ppm chromium (III).

DDT. DDT (1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane) is an organochlorine insecticide that was banned for use in the United States in 1972. DDT, and its metabolites, DDE and DDD, are highly persistent. The half-life of DDT in soil is reported to range from 2 to 15 years (ATSDR 1993). DDT and it's metabolites are also highly lipophilic and have a high bioaccumulation potential. A bioconcentration factor for rainbow trout is reported to be 12000 (ATSDR 1993). Braune and Norstrom (1989) found DDE concentrations in herring gills to be 85 times higher than fish in their diet.

Acute oral toxicity of DDT and its metabolites is relatively low. Mammalian oral LD_{50} values range from 87 mg/kg for rats to >5000 mg/kg for hamsters (EPA 1993). Avian oral LD_{50} values range from 595 mg/kg for California quail to >4000 mg/kg for rock doves (Hudson et al. 1984). Hill and Camardese (1986) report 5-day dietary LC_{50}s for DDT and DDE to be 416 mg/kg and 859 mg/kg, respectively.

Despite low acute toxicity, chronic exposure to low levels of DDT in food has adverse effects of reproduction in wildlife. The primary adverse effect among birds is eggshell thinning and decreased reproductive success (Ratcliffe 1967). Anderson et al. (1975) studied the reproductive success of pelicans from 1969 through 1974. During this time, DDT residues in anchovies, their primary food, declined from 4.27 ppm (wet weight) to 0.15 ppm (wet weight). While reproductive success improved from 1969 to 1974, in 1974 the fledgling rate was still 30% below that needed to maintain a stable population. Because this study was long-term and considered reproductive effects in a wildlife species, EPA (1993) judged this study to be the most appropriate to evaluate DDT effects to avian wildlife. Therefore the 0.15 ppm DDT value was considered to be a chronic LOAEL.

In a study of the effects of DDT on reproduction in mammals, Fitzhugh (1948) exposed rats to 10, 50, 100, or 600 ppm DDT in their diet for two years. While consumption of 50 ppm or more DDT in the diet reduced the number of young produced, no adverse effects were observed at the 10 ppm DDT dose level. Because the study considered exposure throughout 2 years and reproduction, the 10 and 50 ppm DDT doses were considered to be chronic NOAELs and LOAELs, respectively.

Lead. Lead is a comparatively rare metal, averaging 16 ppm in the earth's crust, that is neither essential nor beneficial in living organisms (Eisler 1988b). Lead has adverse effects on survival, growth, reproduction, development, behavior, learning, and metabolism. In general, organic lead compounds are more toxic than inorganic compounds, biomagnification of lead is minimal, and younger organisms are more susceptible to lead toxicity (Eisler 1988b).

Acute oral doses of 5–108 mg lead/kg bw reduced rat survival (Eisler 1988b), and rats fed diets with 5 ppm lead had shortened life spans (Schroeder et al. 1965). An acute LD_{50} based on a single oral dose of 12 mg tetraethyllead/kg body weight was reported by Branica and Konrad (1980). Rats fed 0.5, 5, 25, or 250 ppm inorganic lead in diets over two generations exhibited no substantial developmental effects (Kimmel et al. 1980). In another study, Azar et al. (1973) fed rats a diet containing 0, 10, 50, 100, 1000, or 2000 ppm lead acetate for three generations. While the number of litters and young/litter was not affected by any dose level, growth was reduced and kidney histopathologies were observed among offspring in the 1000 and 2000 ppm treatments. Frequency of pregnancy was reduced in mice ingesting 3 mg/kg body weight tetraethyllead daily, and daily ingestion of 1.5 mg/kg tetraethyllead chloride resulted in a reduction in the success of implanted ova (Clark 1979).

Anemia and other hematological effects were induced among pigeons orally dosed with 6.25 mg lead/kg bw/day (Anders et al. 1982). Kendall and Scanlon (1981) exposed ring doves to drinking water containing 0 or 100 ppm lead and observed no effects on time to produce eggs, egg production, or fertility. However, testes weight and sperm count was decreased among lead-exposed males. Grandjean (1976) correlated eggshell thickness and eggshell lead levels in European kestrels (*Falco tinnunculus*), suggesting that lead may cause eggshell thinning. Among American kestrels (*Falco sparvarius*) fed diets containing 0, 10, or 50 ppm lead, no adverse effects survival, egg-laying, initiation of incubation, egg fertility, or eggshell thickness were observed (Pattee 1984).

Mercury. Mercury has no known biological function and is potentially toxic to fish and wildlife. Mercury is a mutagen, teratogen, and carcinogen that adversely affects the central nervous, renal, and reproductive systems of wildlife (Eisler 1987). Inorganic mercury compounds in aquatic systems are readily converted to organomercury by microbial action (Berlin 1979), with organomercury compounds being more toxic than inorganic mercury compounds. Biota bioconcentrate mercury compounds which can be further biomagnified through food chains (Wren, 1986).

Daily doses of 0.1–0.5 mg/kg bw/day and dietary concentrations of 1.0–5.0 ppm are lethal to sensitive mammals (Eisler 1987). Central nervous system toxicity, weight loss, and mortality were observed among rats fed a diet containing 250 ppm methyl mercury (MeHg) for 2 weeks (Verschuuren et al. 1976a). Rats consuming 2.5 ppm MeHg in the diet for 2 years displayed reduced growth, increased kidney weight, and altered kidney histochemistry (Verschuuren et al. 1976b). To study effects on reproduction, Verschuuren et al. (1976c) fed rats a diet containing 0, 0.1, 0.5, and 2.5 ppm MeHg for three generations. While no effects were observed among rats fed 0.1 or 0.5 ppm MeHg, offspring viability was reduced among rats in the 2.5 ppm treatment. Among mink, 93-day consumption of diets containing 1.8 to 15.0 ppm MeHg produced mortality, ataxia, anorexia, and paralysis (Wobeser et al. 1976), with the highest exposures showing the greatest effects.

The LD_{50} for MeHg for *Coturnix* quail ranges from 14.4 to 33.7 mg/kg bw (Eisler 1987). Growth was decreased and mortality increased among leghorn cockerels fed diets containing 6 to 18 ppm MeHg (Fimreite 1970). Ring-necked pheasants fed diets of MeHg-treated grains displayed reduced egg production and hatchability and laid more shell-less eggs than controls (Fimreite 1971). Heinz (1979) fed mallard ducks a diet containing 0.5 ppm MeHg for three generations. While MeHg consumption did not affect adult weights or weight change during the reproductive season, MeHg-exposed females laid fewer eggs (with more eggs outside the nest box), produced fewer young, and displayed slightly thinner eggshells. Young of MeHg-treated adults were less responsive to maternal calls and hyper-responsive to fright stimuli.

Nickel. Nickel is a naturally occurring element that may exist in various mineral forms. It forms 0.008% of the earth's crust (NAS 1980). Soil and sediment are the primary receptacles for nickel, but mobilization may occur depending on physico-chemical characteristics of the soil (ATSDR 1988; USAF 1990). Nickel is used in a wide variety of applications including metallurgical processes and electrical components, such as batteries (ATSDR 1988; USAF 1990). There is some evidence that nickel may be an essential trace element for mammals.

Nickel occurs in nature in the nonionic and divalent states; other valence states occur very infrequently (Mastromatteo 1986).

The absorption of nickel is dependent on its physico-chemical form, with water soluble forms being more readily absorbed. Soluble nickel compounds tend to be more toxic than insoluble compounds (Goyer 1991). The metabolism of nickel involves conversion to various chemical forms and binding to various ligands (ATSDR 1988). Nickel is excreted in the urine and feces with relative amounts for each route being dependent on the route of exposure and chemical form. Most nickel enters the body via food and water consumption.

Oral LD_{50} values for rats range from 67 mg nickel/kg (nickel sulfate hexahydrate) to >9000 mg nickel/kg (nickel powder) (ATSDR 1988). The Food and Drug Research Laboratories, Inc. (1984) reported an acute oral LD_{50} of 175 mg/kg for female rats exposed to nickel dioxide hexahydrate; acute oral LD_{50}s for 11 other nickel compounds ranged from 275 to >9000 mg/kg. Toxic effects of oral exposure to nickel usually involve the kidneys with some evidence from animal studies showing a possible developmental/reproductive toxicity effect (ATSDR 1988; Goyer 1991).

Inorganic nickel compounds are well-tolerated when taken orally by rodents in doses up to 500 mg/kg (Mastromatteo 1986). Rats continually fed a 250 ppm nickel diet for 16 months suffered no deleterious effects and were considered in excellent condition (Phatak and Patwardhan 1952). Progressive accumulation of nickel was not observed in the tissues assayed. In a three-generation study of rats, Ambrose et al. (1976) reported a no-observed-adverse-effects level (NOAEL) and lowest-observed-adverse-effects level (LOAEL) of 5 mg/kg/day and 50 mg/kg/day, respectively. Doses of 24.15 mg/kg-day administered as nickel sulfate in the diet had no adverse effects on reproduction of the rats. Growth in dogs was depressed by dietary concentrations of 2500 ppm nickel sulfate hexahydrate; in the rats, growth was depressed at dietary concentrations >1000 ppm (Ambrose et al. 1976).

Weber and Reid (1968) fed a basal diet of up to 1300 ppm nickel sulfate or nickel acetate to domestic chicks for 4 weeks. Growth of chicks was significantly depressed at 700 ppm nickel and above. Doses of 21.4 mg/kg-day administered as nickel sulfate in the diet had no adverse effects on weight gain after 4 weeks. Mallard ducklings on diets with ≥800 ppm nickel would be adversely affected (Cain and Pafford 1981).

PCBs. Polychlorinated biphenyls (PCBs) are a family of man-made chemicals consisting of 209 individual compounds with varying toxicity (ATSDR 1989a). Aroclor is the trade name for PCBs made by Monsanto. Because of their insulating and nonflammable properties, PCBs were widely used in industrial applications such as coolants and lubricants in transformers, capacitors, and electrical equipment (ATSDR 1989a). The United States stopped manufacturing PCBs in 1977 due to evidence that they accumulate in the environment. PCBs have become widespread environmental contaminants.

Most exposures to PCBs are oral. Absorption of PCBs following oral exposure is often >90% (ATSDR 1989a). PCBs are preferentially stored in adipose tissues in animals. They may cross the placenta or be transferred to offspring through milk. PCBs with higher chlorine content (the last 2 digits of the Aroclor designation indicate the percent Cl content of the compound) tend to persist in the environment longer than those with lower Cl content, and PCBs are known to bioaccumulate and biomagnify to toxic concentrations in animals (Eisler 1986b; ATSDR 1989a). Chronic exposures are of particular concern. PCBs with high K_{ow} values and high numbers of chlorines in adjacent positions are generally the most toxic. Although relatively insoluble in water, PCBs are generally freely soluble in nonpolar organic solvents and in biological lipids (EPA 1980).

Sixty percent of mice fed diets containing 1,000 ppm Aroclor 1254 for 14 days died within 15 days, but none of the mice fed diets with only 250 ppm Aroclor for 14 days died (Sanders et al. 1974). These diets translate to doses of 130 and 32.5 mg/kg/day, respectively (ATSDR 1989a). White-footed mice fed 10 ppm Aroclor 1254 for 18 months had fewer offspring produced and a longer time between litters than control mice (Linzey 1987).

Feeding studies suggest a total intake of 500-2,000 mg/kg of Aroclor 1254 obtained through the diet over 1 to 7 weeks is lethal in rats (Hudson et al. 1984). Male rats consuming diets containing 0-100 ppm Aroclor 1254 for 104 weeks suffered dose-related reduced survival (NCI 1978); however, there was no effect on similarly treated female rats. Dietary concentrations of ≥20 ppm Aroclor 1254 reduced litter sizes in one- and two-generation reproduction studies with rats; concentrations <5 ppm had no effect (Linder et al. 1974).

Mink are one of the most susceptible mammals; dietary levels as low as 0.1 ppm fresh weight have caused death and reproductive toxicity (Eisler 1986b). Diets containing 20 ppm Aroclor 1242 were lethal to mink in a 247-day experiment. The LC_{50} for chronic exposures is 6.65 ppm Aroclor 1254 for mink over a 8 month period (Ringer et al. 1981). Diets containing 5 ppm Aroclor 1242 caused complete reproductive failure (Bleavins et al. 1980). Exposure for 160 days to 3.57 ppm Aroclor 1254 resulted in 100% mortality of adult mink (Platonow and Karstad 1973).

A chronic study was conducted over 4.5 months exposing mink to 1, 5 and 15 ppm Aroclor 1254 in the diet. There was a significant reduction in the number of offspring born alive at the 5 and 15 mg/kg exposures (Aulerich and Ringer 1977). Mink fed carp containing 1.5 ppm Aroclor 1254 for 6 months produced no offspring that survived to 24 hours (Hornshaw et al. 1983). No effects were observed in mink fed 0.64 ppm Aroclor 1254 for 160 days (Platonow and Karstad 1973). Exposure of mink for 6 months to 1 ppm Aroclor 1254 resulted in no significant difference from controls in number of offspring, or offspring mortality (Wren et al. 1987). Therefore, the 1 ppm dose was considered to be a chronic NOAEL.

A dietary dose of 25 ppm Aroclor 1254 fed for at lease a month before egg-laying in mallard ducks had no detrimental effect on reproductive success (Custer and Heinz 1980). Dietary exposure of 5 ppm Aroclor 1254 for 39 weeks to laying hens and roosters resulted in reduced egg production, although hatchability of fertile eggs was not affected (Platonow and Reinhart 1973). Screech owls fed 3 ppm Aroclor 1248 through two breeding seasons did not have significantly different reproductive success, relative to controls (McLane and Hughes 1980). Exposure of pheasants to 12.5 mg/bird/week (1.8 mg/kg/d) of Aroclor 1254 for 17 weeks resulted in significantly reduced egg hatchability (Dahlgren et al. 1972). Because this study considered exposure throughout a critical lifestage (reproduction), the 12.5 mg/bird/week dose was considered to be a chronic LOAEL.

Selenium. While selenium is an essential nutrient that interacts with Vitamin E and maintains muscle integrity, t has a very narrow tolerance range; in humans, while 0.04-0.1 ppm is required in diet, 4 ppm may produce toxic effects (Eisler 1985b). In mammals, chronic selenium poisoning is induced by diets containing 1–44 ppm selenium (Harr 1978). Symptoms include liver cirrhosis, lameness, loss of hair, emaciation, reduced conception, and increased fetal resorption. Plants convert inorganic selenium to organic selenium compounds, thereby increasing their biological availability (Lo and Sandi 1980).

To evaluate the effects of selenium on reproduction, Schroeder and Mitchner (1971) exposed mice to 3 ppm selenate in drinking water for three generations. This dosage level increased juvenile mortality, number of runts, and resulted in reproductive failure by the third generation. In another study, exposure to 3 ppm selenate or selenite in water for a lifetime had no effect on mouse longevity and no tumorigenicity was observed (Schroeder and Mitchner 1972).

Selenium is both embryotoxic and teratogenic to birds, with organic selenium (selenomethionine) being more toxic than inorganic selenium (Hoffman and Heinz 1988). Mallard ducks were fed diets containing 1, 5, 10, 25, or 100 ppm selenite (Heinz et al. 1987) or 1, 2, 4, 8, or 16 ppm selenomethionine (Heinz et al. 1989) for about 10 weeks. Exposure to 1, 5, or 10 ppm selenite or 1, 2, or 4 ppm selenomethionine in the diet had no effect on survival, growth, or reproductive success of adults. The diet containing 100 ppm selenite killed 11 of 12 adults. While only one adult receiving the 25 ppm diet died, time to laying, interval between eggs was increased, and duckling survivorship was reduced in this treatment (Heinz et al. 1987). Diets containing 8 and 16 ppm selenomethionine resulted in 6.8% and 67.9% malformed embryos, respectively. In addition, duckling survival was significantly reduced (Heinz et al. 1989).

The most visible incident of environmental selenium toxicity occurred at the Kesterson National Wildlife Refuge in California. Agricultural wastewater containing approximately 0.3 ppm selenium was used for marsh management at the refuge (Ohlendorf et al. 1986). Mean selenium concentrations in plants, invertebrates, and fish at the site were 22–175 ppm (dry weight). As a result, reproductive success among water birds was poor, and the incidence of embryo mortality and developmental abnormalities was dramatically increased. Raccoons on the refuge were found to bioaccumulate selenium (Clark et al. 1989). While peak births at the refuge was 2 months later than reported at other locations, no adverse effects on raccoon reproduction were observed.

Metabolism of selenium may be significantly modified through interactions with heavy metals, and selenium may provide some protection from adverse effects associated with various metals, including cadmium and mercury (Eisler 1985b). Arsenite inhibits methylation of selenium but increases fecal excretion of selenite (Venugopal and Luckey 1978).

Thallium. Thallium is a widely distributed metal, occurring at concentrations of approximately 1 ppm in the earths crust (Kazantzis 1979). Principal systems affected by Tl exposure include nervous and digestive; renal damage and hair loss have also been observed. Thallium sulfate, which has been widely used as a rodenticide, has an acute oral LD_{50} of 16 mg/kg (Ware 1978). In chronic studies, rats tolerated a dose of 10 mg Tl acetate/ kg, while 30 mg/kg was lethal to males by 15 weeks. All rats fed a daily dose of 0.45 mg Tl/kg died after 4 months (Kazantzis 1979). Rats exposed to 10 ppm Tl in drinking water for 2 mo accumulated Tl in testis and exhibited signs of testicular toxicity including reduced sperm motility (Formigli et al. 1986).

Bean and Hudson (1976) orally dosed 3 golden eagles with 60 and 120 mg Tl_2SO_4/kg bw; the bird receiving 60 mg Tl_2SO_4/kg survived while the two dosed with 120 mg Tl_2SO_4/kg died, suggesting an LD_{50} between the doses. Oral LD_{50}'s for quail, geese, and ducks are 12, 15, and 30 mg/kg respectively (Shaw 1933). No long-term studies of thallium toxicity to birds are currently available.

Uranium. Uranium is an element used as a nuclear fuel, in nuclear weapons production, and in its natural or depleted form as counterweights for airplanes and as shielding material (Burkart 1991). Its average concentration in the earth's crust is approximately 3–4 ppm (Merritt 1971).

Except for their radioactivity, metallic uranium and particles of insoluble uranium compounds are biologically inert. The chemical toxicity of uranium is exerted only by its aqueous ions (Durbin and Wrenn 1975). Aqueous ions have been identified for uranium (III), uranium (IV), uranium (V), and uranium (VI), but only uranium (IV) and uranium (VI) are stable in solution. In a solution of low acidity, uranium (IV) hydrolyzes to form insoluble hydroxides (Durbin and Wrenn 1975). Uranium (VI) is the most frequently encountered oxidation state in nuclear fuel cycles. Uranyl nitrate and uranyl fluoride are 1.4–2 times more toxic than UCl_5, UCl_4, UO_3, or $NO_2U_2O_7$ and 3 times more toxic than $(NH_4)_2U_2O_7$ (Durbin and Wrenn 1975). Uranium-235 is the most radioactive of the uranium isotopes. Other uranium isotopes including uranium-233, -234, and -238 have low specific activities, long half-lives, and have lower potential to cause radiation induced diseases (ATSDR 1990).

The absorption level of uranium compounds following oral exposure is generally considered to be quite low. In animals, once uranium has been absorbed following inhalation exposures, it leaves the blood very quickly for distribution to body tissues (ATSDR 1990). Some of the uranium reacts with the protein surface of the columnular cells lining the renal tubule and injures or kills these cells. With small or moderate doses, the distal portion of the proximal convoluted tubule receives the severest injury. If death ensues, it follows a typical uremia caused by kidney dysfunction. If the animal survives, cellular regeneration restores much of the kidney tissue and function (NAS 1980). Most of the absorbed uranium is excreted in the urine, and renal clearance of uranium (VI) in cats, dogs, humans, and rabbits is high (Durbin and Wrenn 1975). About 60% is excreted as a soluble bicarbonate complex, whereas the remainder is bound to plasma protein. Sixty percent is excreted in the urine within 24 hours. About 25% may be fixed in the bone (Chen et al. 1961).

The toxicity of uranium compounds depends on the degree of solubility, transport across cellular barriers, and absorption into blood. Toxicological effects from the ingestion of uranium are the result of the action of uranium as a metal and its radioactive properties. For humans and animals, uranium and its salts are highly toxic. Dermatitis, renal damage, and acute arterial lesions may occur. Acute intoxication may lead to irreversible damage and to death due to renal dysfunction (Burkart 1991). The primary toxic chemical effect of uranium is seen in kidney damage, but bone is considered the critical tissue for long-term radiation effects (ICRP 1959). Studies in rabbits, mice, and dogs showed kidney damage in a dose-related effect. Fetal skeletal abnormalities and fetal death were found in pregnant mice exposed to 6 mg/kg of uranyl acetate dihydrate (ATSDR 1990). Uranium toxicity is dependent upon and modified by many factors and most of the reported studies have been conducted with laboratory animals, primarily mice (NAS 1980). Herbivores may be highly sensitive because of the acidity of their urine (Dounce, 1951).

The acute LD_{50} for natural uranium injected in mice varies with strain and sex from 6–25 mg uranium/kg (Dounce et al. 1951; Tannenbaum and Silverstone 1951). It is somewhat lower for $^{233}UO_2(NO_3)_2$: 4.5 mg ^{233}U/kg (Durbin and Wrenn 1975). The toxicity benchmark for mammals is based on a 30-day study involving rabbits (Maynard and Hodge 1949). Doses of 2.8 mg/kg-day administered as uranium soluble salts in the diet had adverse effects on the kidneys of the rats. However, a dietary level of 400 ppm uranium appears to be safe for rats, even when the uranium is in a soluble form (NAS 1980).

Inhalation of uranium dioxide dust by rats, dogs, and monkeys at a concentration of 5 mg uranium/m^3 for up to 5 years produced accumulation in the lungs and tracheobronchial lymph nodes that accounted for 90% of the body burden. No evidence of toxicity was observed despite the long duration of observation (Leach et al. 1970). Doses up to 10 mg uranium/m^3 failed to cause excess mortality in dogs subjected to one year of continual inhalation (36 hrs/wk) (Durbin and Wrenn 1975). Following inhalation of the insoluble uranium salts, retention by the lungs is prolonged (Goyer 1991).

In a 6-week study of black ducks fed 0–1600 ppm powdered uranium in their feed, Haseltine and Sileo (1983) found no alterations in kidney or liver weights, no significant lesions, and no increase in mortality relative to controls. Doses of 86 mg/kg-day administered as depleted metallic uranium in the diet caused no adverse effects on the liver, kidney, or mortality rates of the ducks.

Vanadium. Vanadium is a metallic element that occurs in six oxidation states and numerous inorganic compounds. The toxicity of vanadium depends on its physico-chemical state, particularly on its valence state and solubility. Based on acute toxicity, pentavalent NH_4VO_3 has been reported to be more than twice as toxic as trivalent VCl_3 and more than 6 times as toxic as divalent VI_2. Pentavalent V_2O_5 has been reported to be more than 5 times as toxic as trivalent V_2O_3 (Roshchin 1967). In animals, acutely toxic oral doses cause vasoconstriction, diffuse desquamative enteritis, congestion and fatty degeneration of the liver, congestion and focal hemorrhages in the lungs and adrenal cortex (Gosselin et al. 1984). Minimal effects seen after subchronic oral exposures to animals include diarrhea, altered renal function, and decreases in erythrocyte counts, hemoglobin, and hematocrit (Domingo et al. 1985; Zaporowska and Wasilewski 1991).

A vanadyl sulfate concentration of 5 μg/mL in drinking water, plus a vanadium level of 3.2 μg/g in the diet (4.1 mg V/kg total) of mice, was reported to cause no adverse effects over a lifetime exposure period (Schroeder and Balassa 1967). In similar lifetime studies, rats and mice exhibited no adverse effects when exposed to 5 ppm vanadium (as vanadyl sulfate) in drinking water (Schroeder et al. 1970; Schroeder and Mitchner 1975b). The estimated dose levels were 0.7 mg V/kg/day for rats and 0.9 mg V/kg/day for mice. Vanadium pentoxide in the diet of rats at levels of 10 and 100 ppm for their entire lifetime resulted in no significant toxicological effects except for a reduction in hair cystine content (Stokinger 1981b).

White and Dieter (1978) observed no mortality among mallard ducks fed diets containing 1, 10, or 100 ppm vanadyl sulfate for 12 weeks. Altered lipid metabolism was observed among birds fed 100 ppm vanadium; no other effects were observed. Among chickens, 200 to 400 ppm $Ca_2(VO_4)_2$ in the diet produced 100% mortality; weight gain decreased among chicks fed 20 to 40 ppm $Ca_2(VO_4)_2$ (Romoser et al. 1961).

Zinc. Zinc makes up about 0.002% of the earth's crust (NAS 1980). Zinc is an essential trace element in all living organisms; it assures the stability of biological molecules and structures such

as DNA, membranes, and ribosomes (Eisler 1993). It is used commercially primarily in galvanized metals and metal alloys, but zinc compounds also have wide applications as chemical intermediates, catalysts, pigments, vulcanization activators and accelerators in the rubber industry, UV stabilizers, and supplements in animal feeds and fertilizers. Zinc compounds are also used in rayon manufacture, smoke bombs, soldering fluxes, mordants for printing and dyeing, wood preservatives, mildew inhibitors, deodorants, antiseptics, and astringents (Lloyd 1984; ATSDR 1989b). In addition, zinc phosphide is used as a rodenticide.

Zinc occurs in nature as a sulfide, oxide, or carbonate (Eisler 1993). It is divalent in solution. In freshwater with pH >4 and <7 it exists almost exclusively as the aquo ion $(Zn(H_2O)_6)^{2+}$ (Campbell and Stokes 1985). Zinc interacts with many chemicals, and it may diminish the toxic effects of cadmium and protects against lead toxicosis in terrestrial animals (Eisler 1993). Background concentrations seldom exceed 0.040 mg/L in water or 200 mg/kg in soil or sediment (Eisler 1993).

Although it is essential for normal growth and reproduction (Prasad 1979; Stahl et al. 1989) and important to central nervous system function (Eisler 1993), the primary toxic effect of zinc is on zinc-dependent enzymes that regulate RNA and DNA. It is most harmful to aquatic life in conditions of low pH, low alkalinity, low dissolved oxygen, and elevated temperature. Zinc is relatively nontoxic in mammals, but excessive intake can cause a variety of effects. It is not known to be carcinogenic by normal exposure routes (Eisler 1993).

Gastrointestinal absorption of zinc is variable (20–80%) and depends on the chemical compound as well as on zinc levels in the body and on dietary concentrations of other nutrients (EPA 1984). Information on pulmonary absorption is limited and complicated by the potential for gastrointestinal absorption due to mucociliary clearance from the respiratory tract and subsequent swallowing. Pulmonary inflammation and changes in lung function have been observed in inhalation studies on animals (Amur et al. 1982; Lam et al. 1985; Drinker and Drinker 1928). Zinc is present in all tissues with the highest concentrations in the prostate, kidney, liver, heart, and pancreas. Zinc is a vital component of many metalloenzymes such as carbonic anhydrase, which regulates CO_2 exchange (Stokinger 1981).

In animals, gastrointestinal and hepatic lesions (Allen et al. 1983; Brink et al. 1959), pancreatic lesions (Maita et al. 1981; Drinker et al. 1927), anemia (ATSDR 1989b; Fox and Jacobs 1986; Maita et al. 1981), and diffuse nephrosis (Maita et al. 1981; Allen et al. 1983) have been observed following subchronic oral exposures. Anemia and pancreatitis were the major adverse effects observed in chronic animal studies (Aughey et al. 1977; Drinker et al. 1927; Walters and Roe 1965; Sutton and Nelson 1937). Teratogenic effects have not been seen in animals exposed to zinc; however, high oral doses can affect reproduction and fetal growth (Ketcheson et al. 1969; Schlicker and Cox 1967, 1968; Sutton and Nelson 1937).

Livestock and small mammals are tolerant of extended dietary loadings >100 times the minimum recommended daily zinc requirement (Eisler 1993). No adverse effects on general health or reproduction were observed in dairy cows fed 1310 mg zinc/kg food (Miller et al., 1989). A diet of 4000–5000 mg zinc/kg food for 18 days resulted in fetotoxicity and poor reproduction in rats (NAS 1979). Acute oral LD_{50} doses of 350–800 mg zinc/kg body weight have been reported for rats (Eisler 1993). Wlostowski et al. (1988) recommended 30 mg zinc/kg in the diet of bank voles.

Dogs on diets with up to 1000 mg zinc/kg of food for up to one year showed no measurable signs of damage (NAS 1979). Horses ingesting >90 mg zinc/kg body weight daily in the vicinity

of a lead-zinc smelter exhibited decreased growth and death (NAS 1979). No effects were observed in mice fed <682 mg zinc/kg food (<109 mg zinc/kg body weight daily) for 13 weeks, but at 6820 mg zinc/kg food adverse effects on growth and survival were documented (Maita et al. 1981). In a 37-day study involving rats, doses of 97 mg/kg-day administered as zinc carbonate in the diet had no adverse effects on the reproductivity of rats (Kinnamon 1963). European ferrets (*Mustela putorius furo*) fed up to 500 mg zinc/kg for up to 197 days all survived with no significant histopathologies, but those fed 1500 or 3000 mg/kg diet died within 21 days (Straube et al. 1980; Reece et al. 1986). Reproduction ceased entirely in female rats ingesting a diet with 500 mg zinc/kg/day (Sutton and Nelson 1937), possibly a result of zinc-induced anemia.

Mallards (*Anas platyrhynchos*) fed diets containing >3000 mg zinc/kg for 30 or 60 days suffered leg paralysis, decreased food consumption, and high mortality (Gasaway and Buss 1972; NAS 1979). Egg production in Japanese quail (*Coturnix coturnix japonica*) hens fed 15,000 mg zinc (as ZnO)/kg feed for 7 days decreased to near zero within 3 days (Hussein et al. 1988). Seven percent of 14-day old quail fed 600 mg zinc (as zinc phosphide)/kg feed over 5 days died, 53% of those fed 990 mg/kg died, and 93% of those fed 1634 mg/kg died (Hill and Camardese, 1986). Domestic chicken pullets and hens on a diet with 20,000 mg zinc/kg feed for 5 days were lighter weight by day 5 and produced significantly fewer eggs for 4 weeks following treatment (Palafox and Ho-A 1980). Eggs collected 14–28 days post-treatment had reduced fertility and hatchability. However, normal growth, egg production, fertility, and hatchability was observed 4–12 weeks post-treatment. Acute oral LD_{50} values for zinc phosphide, a rodenticide, were between 16 and 47 mg/kg body weight in ring-necked pheasants (*Phasianus colchicus*), golden eagles (*Aquila chrysaetos*), mallards, and horned larks (*Eremophila alpestris*) (Hudson et al. 1984), but much of the biocidal action is attributed to the phosphide rather than the zinc (Eisler 1993).

Diets containing 28, 48, 228, or 2028 mg zinc/kg for 12–44 weeks had no effect on overall egg production by domestic chickens although zinc levels were elevated in hens on the highest zinc diet (Stahl et al. 1990). All day-old chicks fed diets containing 16,000 mg zinc/kg feed for 5 weeks and 80% of those fed 8000 mg/kg died; those on a 4000 mg zinc/kg diet showed no significant reductions in growth or survival (Oh et al. 1979). In a 60-day study, doses of 170 mg/kg-day administered as zinc carbonate in the diet caused increased mortality and altered blood chemistry in mallards (Gasaway and Buss 1972).

In chickens, adverse effects associated with zinc deficiency have been observed at <38 mg zinc/kg dry weight feed (Blamberg et al. 1960; Westmoreland and Hoekstra 1969; Stahl et al., 1989), but concentrations of 93–120 mg/kg are suggested as adequate in the diet (Blamberg et al. 1960; Westmoreland and Hoekstra 1969). Greater than 178 mg/kg dry weight feed is considered excessive (Stahl et al. 1989), and dietary concentrations >2000 mg/kg dry weight feed are considered toxic (NAS 1979; Oh et al. 1979; Stahl et al. 1990). Turkey poults tolerated zinc levels up to 2000 ppm in starter diets for 21 days with no deleterious effects, but levels ≥4000 ppm resulted in marked growth depression (Vohra and Kratzer 1968). No mortality was observed in poults on a diet containing 10,000 ppm zinc (Vohra and Kratzer, 1968), but increased mortality has been observed for chickens on diets with 3000 ppm zinc (Roberson and Schaible 1960).

Appendix E

REPRODUCTIVE PERFORMANCE OF MINK

REPRODUCTIVE PERFORMANCE OF MINK

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1. INTRODUCTION

Plant operations and waste disposal at the Oak Ridge National Laboratory (ORNL), the Gaseous Diffusion Plant (ORGDP), and the Weapons Plant (Y-12) have introduced an assortment of potentially harmful contaminants into the surrounding environment (Ashwood et al. 1986, Suter 1990). The potential for off-reservation transport of contaminants by streams on the Oak Ridge Reservation (ORR) that empty into the Clinch and Tennessee River systems is a concern.

Contaminants of special concern include mercury (Hg) and polychlorinated biphenyls (PCB's) (Suter 1990). Elevated concentrations of Hg and PCB's have been found in fish collected from East Fork Poplar Creek (EFPC) and Bear Creek, and elevated concentrations of PCB's also have been found in fish from White Oak Creek (Loar 1990). East Fork Poplar Creek and Bear Creek originate within the Y-12 Plant and flow into Poplar Creek north of the K-25 Plant and White Oak Creek flows through ORNL. Both creeks empty into the Clinch River on the upper reach of Watts Bar Lake. In the Screening Level Risk Assessment for the Off-Site Ecological Effects in Surface Waters Downstream from the U.S. Department of Energy Oak Ridge Reservation, Suter (1990) indicated that piscivorous wildlife along the Clinch River are at risk.

Mink (*Mustela vison*) have been shown to be among the most sensitive, if not the single most sensitive, mammalian species to polychlorinated biphenyl (PCB) toxicity (Aulerich and Ringer 1977). Feeding studies conducted by Aulerich *et al.* (1971, 1973), Hornshaw *et al.* (1983), and Heaton (1992) have demonstrated the extreme sensitivity of mink to chlorinated hydrocarbon contaminants, especially PCBs, contained in fish taken from the Great Lakes. Additional studies have shown this species also to be sensitive to other halogenated hydrocarbons, including polybrominated biphenyls (Aulerich and Ringer 1979), hexachlorobenzene (Bleavins *et al.* 1988) and mercury (Hg) (Aulerich et al. 1974, Wobeser and Swift 1976, Wobeser et al. 1976). Numerous other toxicological studies with mink have been reported in the literature (Scientifur 1987, Sundqvist 1989, Leonards et al. 1994) and summarized by Calabrese *et al.* (1992). In addition, mink have been recommended as an indicator species for the goal of virtual elimination of persistent toxic substances in the Great Lakes by the International Joint Commission Virtual Elimination Task Force Biomarker Workshop (personal communication from Glen Fox, Environment Canada, Ottawa, Ontario).

Because mink are sensitive to PCB and Hg toxicity and inhabit wetland areas, they are potentially good indicators of environmental effects of these chemicals in aquatic habitats. However, mink are secretive and population densities tend to be low (male home ranges average 2,600 m in stream length, Dunstone 1993), making assessment of environmental contaminant effects in natural populations difficult. Since it is known that fish inhabiting aquatic systems downstream from the Oak Ridge Reservation (ORR) contain elevated concentrations of PCBs and Hg (Sect. 3.5.1), and that fish are a major food item of mink, the objectives of this study were to compare biological accumulation of environmental contaminants and reproductive effects in mink fed fish collected on the ORR to accumulation and effects in mink fed fish collected from the Clinch River above the ORR, or from the ocean.

2. MATERIALS AND METHODS

2.1 Fish Collection and Diet Preparation

Fish used in mink diets were collected from the reach of Poplar Creek between East Fork Poplar Creek and the confluence of Poplar Creek with the Clinch River (on the ORR) and from the Clinch River above Melton Hill Dam near Bull Run Power Plant (above the ORR), or were ocean fish (mackerel) obtain from a commercial supplier. Fish collected on the ORR and above the ORR were identified, weighted, placed in labeled plastic bags, frozen and shipped on dry ice for overnight delivery to the Michigan State University (MSU) Experimental Fur Farm. Ocean fish were frozen and shipped directly to MSU by a commercial supplier. At MSU, fish collected from the same location were ground through a 3/8 inch plate and mixed in a paddle mixer. This process was repeated until all fish from the same location were ground and mixed together so that a homogeneous mixture was obtained. Ten aliquots of the homogeneous mixture were placed in whirlpac bags, labelled, and frozen for contaminant analyses. This process was repeated for fish collected from each source.

Five diets, each composed of 75% fish and 25% normal ranch mink chow, were prepared. Appropriate proportions of homogenized fish from prescribed locations were blended with components of normal mink diet (eggs, liver, vitamin and mineral premix, d-biotin, and cereal). The fish portion of 2 diets (A and B) contained 75% ocean fish and 75% fish collected above the ORR (Clinch River above Melton Hill Dam), respectively. These served as reference diets for this study. The fish portion of the remaining 3 diets (C, D, and E) contained 25, 50 and 75% fish collected on the ORR and 50, 25, and 0% ocean fish, respectively. All diets were formulated to meet the nutrient requirements of the mink (NRC, 1982). Diet proximate analyses were determined by MSU. Ten aliquots of each diet were placed in whirlpac bags, labelled, and frozen for contaminant analyses.

2.2 Mink Feeding Experiment

Fifty adult, natural dark mink from the MSU Experimental Fur Farm, Michigan State University (MSU), East Lansing, Michigan were uniquely identified and randomly divided into 5 groups of 2 males and 8 females per group. Mink were housed individually in wire cages (61 x 76 x 46 cm) with attached nest boxes (38 x 28 x 27 cm). Cages were suspended above the ground in open-sided sheds. Throughout the study, mink were provided food and drinking water *at libitum* and exposed to ambient temperature and photoperiod. Mink were immunized against canine distemper, virus enteritis, infectious pneumonia, and botulism, and provided thiamine daily to prevent thiamine deficiency resulting form thiaminase in fish. Mink were acclimated to the test facilities for at least one week prior to the definitive test.

Each mink group was fed one of the prepared diets from December 1, 1993 (approximately 3 months prior to breeding) through approximately June 30, 1993 (6 weeks postpartum). Mink were weighed at the beginning of the feeding trials and at monthly intervals thereafter (except during the gestation period). They were observed daily and any behavioral changes or clinical signs of toxicity recorded.

Mating began March 1, 1994 and was confined within the respective groups. Females were given an opportunity to mate every fourth day until a confirmed mating (presence of motile spermatozoa in vaginal aspirations) was obtained. The mated females were given an opportunity for a second mating the day following the initial mating or eight days later (a standard commercial

mink ranch practice). The bred females were checked daily during gestation for evidence of whelping. The gestation period of mink averages 51 days but is highly variable (42 to 65 days) due to delayed implantation. Whelped kits were counted, sexed, and weighed on the day of birth and at three and six weeks of age. Mink kits begin to consume solid feed at 21 to 24 days of age and are weaned at 6 weeks. Thus, kit body weight at 3 weeks of age provides a good indication of the lactational performance of the female. Reproductive indices measured included: number of females mated; number of females whelping; length of gestation; number of kits whelped (alive, dead); kit sex ratio; average kit body weight at birth, 3, and 6 weeks of age; and kit survival to 3 and 6 weeks of age.

Adult mink with > 30 % decrease in original body weight and all adults at the termination of the study were euthanized (CO_2). All adult mink were necropsied, organ (brain, liver, kidneys, heart, lungs, gonads, and adrenal glands) weights were recorded and samples of adipose, liver, kidney, and hair collected for residue analysis. At 6 weeks of age, 3 kits each from 3 randomly selected females from dietary groups A, B, C, and E were euthanized, liver, spleen, and kidneys weighed, and samples (liver, kidney, and remaining carcass) collected for residue analyses. Tissue samples (brain, liver, kidney, heart, lungs, and adrenal gland) also were collected from adult mink and preserved in 10% formalin for histopathologic examination.

Ten aliquots, each, of homogenized fish and mink diets, and collected mink tissues were shipped frozen to the Southwest Research Institute, San Antonio, TX for PCB (Aroclors), CB (congeners) and mercury concentration determination. Adult mink liver and fat tissues and kidney, liver, and hair tissues were analyzed for Aroclor and CB concentrations, and mercury concentration, respectively. Liver tissue also was analyzed for ethoxyresorufin-o-deethylase (EROD) activity. Kit whole carcass and liver tissue, and whole carcass and kidney tissues were analyzed for PCB and CB concentrations, and mercury concentration, respectively. Lipid concentration (% lipid) was determined for all samples analyzed for PCBs

PCB Aroclor and congener results were adjusted for lipid concentration (tissue concentrations divided by the % lipid) prior to statistical analysis. Results of contaminant analyses, physiological measurements, and reproductive parameters were statistically evaluated for differences among diet groups and between diet groups A or B and E using non-parametric (Wilcoxon or Kruskel-Wallis) and gausian (t-test or ANOVA) tests.

3. RESULTS

The species composition of fish collected from the ORR and the Clinch River above Melton Hill Dam were similar and consisted mostly of benthic species (Table E.1). Mean mercury concentrations were significantly different among fish collected from the ORR, Clinch River, or ocean (Table E.2). Mean mercury concentrations in mink diets increased progressively from diet A through diet E (Table E.2). Correspondingly, mercury concentrations in liver, kidney, and hair of adult female mink increased progressively in mink fed diets A through E (Table E.3). Mercury concentrations in kit kidney tissue and homogenized carcass were not significantly different in offspring of mink fed diets A, B, or C, but were significantly greater ($P < 0.05$) in offspring of mink fed diet E (Table E.3).

3.1 PCB and Congener Profiles in Fish and Mink Diets

Aroclor 1260 was the dominant Aroclor detected in aliquots of homogenized fish, mink diets, and tissues of adult mink and kits, although Aroclor 1254 also was quantified in several tissue samples. Aroclor 1260 was quantified in aliquots of all diets except for diet A (75% ocean fish), in fat tissue from mink in all diet groups except diet group C, and in liver tissue of mink from diet groups D and E. Twenty-three specific congeners, including 8 coplanar congeners (non-ortho or mono-ortho congeners), were evaluated in homogenized fish, diets, liver and fat tissues of adult mink, and liver tissue and carcass of kits. Ninety-six percent of congener concentrations were significantly different among ocean, ORR, and Clinch River homogenized fish, including all but one coplanar congeners (Table E.4). In all cases where significant differences existed in homogenized fish congener concentrations among collection sites, the greatest concentrations were in fish collected from the ORR. Aroclor 1260 and 87% of the congener evaluated also were quantified in mink diets (Table E.5). Mean Aroclor 1260 concentration was significantly greater ($P < 0.01$) in diet E compared to concentrations other diets. Coplanar CBs 126 and 189 were not detected in any diets, CB 156 was detected in low concentrations in diets B, C, and D, and CBs 77 and 81 were quantified in low concentrations in diets C, D, and E, while CB 167 was only quantified in diets D and E. Concentrations of coplanar CB 118 were significantly greater ($P < 0.01$) in aliquots of diet E compared to aliquots of the other diets. Mean concentrations of CB 123 progressively increased from diet C to D, however, quantified concentrations in diet E were surprisingly lower than concentrations in diet C. Low concentrations of this CB in diet E are thought to result from matrix interference in this diet. Similarly, lower than expected concentrations were quantified for CBs 99, 101, 156, and 171 in diet E. The remaining non-plainer congener concentrations were generally greatest in aliquots from diet E (Table E.5).

3.2 PCB and Congener Profile in Mink Tissues

Mean Aroclor 1260 concentration was significantly greater in liver tissue of female mink fed diet E (Table E.6). Coplanar CB 189 was quantified at low concentrations (<21 ppb) in liver tissue from all mink fed diet E, 50% of those fed diet D, and less than 10% of those fed diets A, B, or C. Liver concentrations of CB 126 were significantly greater ($P < 0.01$) in samples collected from female mink fed diet E. Low concentrations (<32 ppb) of CB 126 also were quantified in liver tissue from female mink fed diets B and D. Mean liver concentrations of coplanar CBs 77 and 81 were $\leq$ 6 ppb in all female mink regardless of diet. Mean concentrations of CBs 156 and 167 increased progressive in liver tissue from female mink fed diets A - E and were significantly greater ($P < 0.01$) in mink fed diet E compared to those fed diets A or B. Mean concentration of coplanar CBs 118 and 123 were significantly greater in liver tissue from female mink on diet E compared to concentrations in liver tissue from mink fed diets A or B. There were significant differences ($P < 0.05$) in all female mink liver tissue non-planar congener concentrations among diet groups. For all non-planar congener in female liver tissue, concentrations were significantly greater ($P < 0.05$) in tissues from diet E mink compared to diet A mink, except for concentrations of CBs 195 and 196 which were not significantly different. Similarly, mean female mink liver concentrations of all non-planar congeners were significantly greater in female mink liver from diet group E mink compared to diet group B mink, except for CBs 146, 153, 170, 180, 183, and 201 which were not significantly different between these diet groups.

Mean Aroclor 1260 concentration in fat tissue from female mink fed diet E were significantly greater than mean concentrations from female mink fed diets A or B (Table E.7). Mean concentrations of all coplanar CBs were significantly greater ($P < 0.05$) in fat tissue from female

mink fed diet E compared to mean concentrations in mink fed diets A or B, except that there was no difference in CB 126 fat concentration between female mink fed diets E and B. Non-planar CB mean concentrations were significantly greater ($P < 0.05$) in fat tissue from female mink fed diet E compared to mean concentrations in mink fed diets A or B, except CBs 170, 194, and 195. There was no difference between CB 170 or 194 mean fat concentrations between female mink fed diets B or E, and mean fat concentration of CB 195 in female mink was significantly greater ($P < 0.05$) in female mink fed diet B compared to diet E.

Concentrations of coplanar CBs 77, 81, 123, 156, and 189 were < 20 ppb in liver tissue from kits in all diet groups (Table E.8). Concentrations of coplanar CBs 118, 126, and 167 were significantly greater ($P < 0.05$) in liver from diet E kits compared to those from diets A, B, or C. Concentrations of non-planar CBs 101, 151, 171, and 195 were < 15 ppb in liver tissue from kits in all diet groups. Concentrations of the remaining non-planar CBs quantified were greater in liver tissue of diet E kits.

The pattern of concentrations of coplanar CBs in kit carcass homogenates from the various diets were similar to those observed in kit liver tissue (Table E.9). Except, the concentration of coplanar CB 126 was < 25 ppb in kit carcass homogenates and the concentration of CB 156 was significantly greater ($P < 0.05$) in kit carcass homogenates from diet group E compared to other diet groups. Concentration patterns of non-planar CBs in the various diet groups also were similar in carcass homogenates compared to concentrations in liver tissue. Except concentrations of CBs 151 and 196 were the only CBs with concentrations < 25 ppb. Concentrations of all other CBs quantified were greater in homogenized carcasses of kits from diet group E.

3.3 Physiological and Reproductive Effects

Two mink from diet group A, one male and one female, died during the experimental period. The male died on March 18, 1994 from hemorrhagic and necrotizing cystitis and the female died on April 24, 1994 due to complications during parturition. In addition to these adult mink, kits from one female in diet group B developed staph infections and all but one diet prior to 6 weeks of age.

One, 2, and 4 females did not whelp in diet groups A and B, D, and C, respectively (Table E.10) . Two females that did not whelp (1 from diet group A and 1 from diet group C) had cyst in the reproductive tracts that probably interfered with normal reproduction. Two females that did not whelp (1 from diet group C and 1 from diet group D) had no placental scars in the uterus and therefore probably were not pregnant. The reason the remaining 4 females did not whelp is unknown.

Mean whole body weights of female mink were not significantly different among diet groups at the beginning of the experimental period, however, mean weights of females in diet group E were significantly less ($P=0.03$) than mean weights of females in diet group A at the end of the experimental period (Table E.10). Mean female relative organ weights (organ weights/body weight) were not significantly different among diet groups. At 6 weeks of age, mean whole body weights were significantly lower ($P=0.004$) in male kits from diet group E compared to those from diet group A. A similar trend was observed in 6 week old female kits, although differences were not statistically significant. Mean relative kidney weights were significantly lower ($P=0.003$) in kits from diet group B ($\bar{x} = 1.0$ g) compared to those from diet group E ($\bar{x} = 1.3$ g). Kit mean relative liver and spleen weights were not significantly different among diet groups. No histological lesions were attributed to diets.

Mean litter size was significantly reduced (P=0.01) in diet group E compared to diet groups A, B, and C but not diet group D (Table E.10). Liver EROD activity was significantly increased in adult female mink from diet groups D and E compared to those from diet group A (Table E.10).

Although concentrations of mercury and PCBs were greater in fish collected from streams located on the ORR and these contaminants were higher in diets fed to mink with increasing percentage of ORR fish, reproductive effects were only noted in mink fed 75% ORR fish. Liver EROD activity, a sensitive biomarker of exposure to PCBs increased in mink fed diets containing 50% and 75% fish collected from the ORR.

Although fish are a major food item in the diet of wild mink, the proportion of fish in their diets normally does not exceed 40-60%. However, in addition to fish, concentrations of contaminants in other food items (crayfish, frogs, muskrat, ducks, and reodents) need to be evaluated in assessing effects of contaminants on mink living on the ORR.

Table E.1. Percent by weight of the most common fish species collected from the ORR and Clinch River above Melton Hill Dam

Common name	ORR %	Weight kg	Clinch River %	Weight kg
Sucker	17	280	3	12
Carp	20	330	24	97
Catfish	15	248	13	53
Shad	7	116	6	24
Buffalo	17	280	42	170
Other species	24	396	12	49
TOTALS	100	1,650	100	405

Table E.2. Mean mercury concentrations (ppm, wet wt) in homogenized fish[1] collected on the ORR[2], Clinch River above the ORR[3], and from the ocean[4] and diets fed to mink

	N[5]	MEAN[6]	SE	MIN	MAX
LOCATION					
On the ORR	10	0.35[a]	0.03	0.17	0.43
Clinch River	10	0.07[b]	0.00	0.05	0.09
Ocean	10	0.03[c]	0.00	0.02	0.04
DIET					
A[7]	10	0.02[a]	0.00	0.02	0.03
B[8]	10	0.05[b]	0.00	0.04	0.06
C[9]	10	0.09[c]	0.00	0.08	0.11
D[10]	10	0.15[d]	0.01	0.12	0.18
E[11]	10	0.22[e]	0.01	0.16	0.24

[1]Various fish species.
[2]Poplar Creek between East Fork Poplar Creek and confluence with the Clinch River.
[3]Above Melton Hill Dam near Bull Run Power Plant.
[4]Mackerel purchased from commercial supplier.
[5]Number of aliquots analyzed.
[6]Means followed by different letters are significantly different, $P<0.05$.
[7]75% ocean fish, 25% ranch mink diet.
[8]75% fish collected from the Clinch River above the Oak Ridge Reservation (ORR), 25% ranch mink diet.
[9]25% fish collected from Poplar Creek on the ORR, 50% ocean fish, 25% ranch mink diet.
[10]50% fish collected from Poplar Creek on the ORR, 25% ocean fish, 25% ranch mink diet.
[11]75% fish collected from Poplar Creek on the ORR, 25% ranch mink diet.

Table E.3. Mercury concentration (ppm, wet wt) in tissues from female mink fed (n=8/diet) various diets[1] and their 6-week-old kits

		Diet A	Diet B	Diet C	Diet D	Diet E
ADULT FEMALE MINK						
Liver Hg	Mean[2]	0.41[a]	0.61[a]	1.06[a]	1.93[b]	3.67[c]
	SE	0.07	0.06	0.10	0.15	0.32
	Range	0.17-0.8	0.47-0.87	0.74-1.49	1.26-2.47	2.52-5.20
Kidney	Mean	0.84[a]	1.25[ab]	2.22[bc]	3.47[cd]	4.35[d]
	SE	0.13	0.18	0.35	0.52	0.34
	Range	0.25-1.46	0.53-1.96	1.24-4.22	2.38-7.00	3.33-6.25
Hair Hg	Mean	3.79[a]	7.43[b]	7.71[b]	13.44[c]	19.03[d]
	SE	0.26	0.55	0.63	0.79	0.57
	Range	2.20-4.61	5.05-9.70	4.38-9.62	10.2-15.6	16.8-21.4
KITS						
Carcass	Mean	0.02[a]	0.03[a]	0.05[a]		0.20[b]
	SE	0.004	0.003	0.003		0.02
	Range	0.01-0.05	0.02-0.05	0.04-0.06		0.10-0.30
	N	9	9	8		9
Kidney	Mean	0.03[a]	0.03[a]	0.06[a]		0.19[b]
	SE	0.001	0.003	0.003		0.02
	Range	0.02-0.04	0.02-0.04	0.05-0.07		0.11-0.31
	N	9	9	8		9

[1]Diet A = 75% ocean fish, Diet B = 75% fish collected above the Oak Ridge Reservation, Diet C = 25% fish collected on the Oak Ridge Reservation and 50% ocean fish, Diet D = 50% fish collected on the Oak Ridge Reservation and 25% ocean fish, Diet E = 75% fish collected on the Oak Ridge Reservation.

[2]Means with different superscripts are significantly different, $P<0.05$.

Table E.4. Mean[1] ±SE lipid adjusted Aroclor 1260 and PCB congener concentrations (ppm, wet wt) in homogenized fish[2] collected on the ORR[3], Clinch River above the ORR[4], and from the ocean[5]

	Ocean	Clinch River	ORR
CB 77	0.022[a]±0.000 (10)	0.039[b]±0.001 (10)	0.190[c]±0.004 (10)
CB 81	0.053[a]±0.001 (10)	0.592[b]±0.062 (10)	1.419[c]±0.262 (9)
CB 99	0.022[a]±0.000 (10)	0.076[b]±0.011 (10)	0.894[c]±0.015 (10)
CB 101	0.022[a]±0.000 (10)	0.142[b]±0.022 (10)	0.609[c]±0.010 (10)
CB 118	0.022[a]±0.000 (10)	0.468[ab]±0.219 (10)	1.150[b]±0.030 (9)
CB 123	0.022[a]±0.000 (10)	0.039[ab]±0.001 (10)	0.051[b]±0.010 (10)
CB 126	0.022[a]±0.000 (10)	0.039[b]±0.001 (10)	0.041[b]±0.001 (10)
CB 128	0.022[a]±0.000 (10)	0.178[b]±0.003 (10)	0.636[c]±0.012 (10)
CB 138	0.022[a]±0.000 (10)	0.706[b]±0.014 (10)	1.611[c]±0.052 (9)
CB 146	0.022[a]±0.000 (10)	0.388[b]±0.007 (10)	0.822[c]±0.023 (10)
CB 151	0.039[a]±0.002 (10)	0.496[b]±0.008 (10)	1.731[c]±0.048 (10)
CB 153	0.074[a]±0.002 (10)	2.813[b]±0.082 (10)	4.733[c]±0.123 (9)
CB 156	0.022[a]±0.000 (10)	0.039[b]±0.001 (10)	0.078[b]±0.037 (10)
CB 167	0.022[a]±0.000 (10)	0.044[b]±0.003 (10)	0.193[c]±0.003 (10)
CB 170	0.022[a]±0.000 (10)	0.455[b]±0.010 (10)	1.315[c]±0.025 (10)
CB 171	0.022[a]±0.000 (10)	0.039[b]±0.001 (10)	0.041[b]±0.001 (10)
CB 180	0.022[a]±0.000 (10)	3.021[b]±0.099 (10)	3.592[c]±0.088 (10)
CB 183	0.022[a]±0.000 (10)	0.690[b]±0.012 (10)	1.000[c]±0.016 (10)
CB 189	0.022[a]±0.000 (10)	0.039[b]±0.001 (10)	0.041[b]±0.001 (10)
CB 194	0.022[a]±0.000 (10)	0.376[b]±0.009 (10)	0.430[c]±0.006 (10)
CB 195	0.022[a]±0.000 (10)	0.042[b]±0.002 (10)	0.043[b]±0.002 (10)
CB 196	0.022[a]±0.000 (10)	0.570[b]±0.014 (10)	1.660[c]±0.041 (10)
CB 201	0.022[a]±0.000 (10)	0.486[b]±0.009 (10)	0.605[c]±0.008 (10)
Aroclor 1260	0.379[a]±0.008 (10)	21.917[b]±0.681 (10)	28.997[c]±0.659 (10)

[1]Means with different superscripts are significantly different, $P<0.05$.
[2]Various fish species.
[3]Poplar Creek between East Fork Poplar Creek and confluence with the Clinch River.
[4]Above Melton Hill Dam near Bull Run Power Plant.
[5]Mackerel purchased from commercial supplier.

Table E.5. Mean ±SE lipid adjusted Aroclor 1260 and PCB congener concentrations (ppm, wet wt) in mink diets[1] (n=10/diet)

	Diet A	Diet B	Diet C	Diet D	Diet E
CB 77	0.025±0.000	0.037±0.001	0.046±0.003	0.102±0.002	0.116±0.005
CB 81	0.027±0.002	0.337±0.051	0.507±0.011	0.873±0.022	1.158±0.033
CB 99	0.025±0.000	0.037±0.001	0.276±0.011	0.531±0.014	0.520±0.013
CB 101	0.031±0.004	0.239±0.032	0.288±0.009	0.585±0.012	0.396±0.020
CB 118	0.025±0.000	0.117±0.010	0.192±0.007	0.405±0.007	0.668±0.021
CB 123	0.025±0.000	0.037±0.001	0.212±0.008	0.446±0.008	0.076±0.003
CB 126	0.025±0.000	0.037±0.001	0.027±0.001	0.038±0.003	0.033±0.001
CB 128	0.025±0.000	0.085±0.003	0.104±0.004	0.228±0.005	0.351±0.013
CB 138	0.080±0.002	0.791±0.087	0.331±0.011	0.690±0.009	1.048±0.020
CB 146	0.025±0.000	0.037±0.001	0.141±0.006	0.335±0.007	0.501±0.015
CB 151	0.025±0.000	0.314±0.010	0.254±0.009	0.555±0.009	1.058±0.039
CB 153	0.025±0.000	1.862±0.025	0.766±0.029	1.772±0.026	3.126±0.056
CB 156	0.025±0.000	0.139±0.013	0.065±0.002	0.094±0.015	0.033±0.001
CB 167	0.025±0.000	0.037±0.001	0.027±0.001	0.061±0.003	0.103±0.004
CB 170	0.025±0.000	1.847±0.964	0.154±0.007	0.346±0.008	0.798±0.016
CB 171	0.025±0.000	0.037±0.001	0.061±0.002	0.127±0.003	0.033±0.001
CB 180	0.025±0.000	1.011±0.266	0.507±0.019	1.091±0.018	2.292±0.040
CB 183	0.025±0.000	0.276±0.053	0.170±0.006	0.355±0.007	0.573±0.019
CB 189	0.025±0.000	0.037±0.001	0.027±0.001	0.031±0.000	0.033±0.001
CB 194	0.025±0.000	0.058±0.022	0.072±0.008	0.149±0.020	0.245±0.009
CB 195	0.025±0.000	0.039±0.003	0.027±0.001	0.033±0.002	0.033±0.001
CB 196	0.025±0.000	1.081±0.084	0.184±0.008	0.415±0.008	1.071±0.024
CB 201	0.025±0.000	0.230±0.034	0.060±0.002	0.131±0.003	0.338±0.010
Aroclor 1260	0.169±0.002	11.440±0.327	4.697±0.174	10.405±0.250	20.670±0.458

[1]Diet A = 75% ocean fish, Diet B = 75% fish collected above the Oak Ridge Reservation, Diet C = 25% fish collected on the Oak Ridge Reservation and 50% ocean fish, Diet D = 50% fish collected on the Oak Ridge Reservation and 25% ocean fish, Diet E = 75% fish collected on the Oak Ridge Reservation.

Table E.6. Mean ±SE lipid adjusted Aroclor 1260 and PCB congener concentrations (ppm, wet wt) in liver tissue from female mink (n=8/diet) fed various diets[1]

	Diet A	Diet B	Diet C	Diet D	Diet E
CB 77	0.048±0.016	0.081±0.010	0.033±0.004	0.095±0.040	0.028±0.005
CB 81	0.048±0.016	0.054±0.010	0.033±0.004	0.095±0.040	0.197±0.117
CB 99	0.048±0.016	0.117±0.014	0.420±0.061	0.962±0.123	0.823±0.215
CB 101	0.048±0.016	0.053±0.011	0.033±0.004	0.145±0.019	0.154±0.015
CB 118	0.141±0.008	0.590±0.040	1.066±0.153	2.157±0.406	1.656±0.332
CB 123	0.048±0.016	0.041±0.006	0.033±0.004	0.095±0.040	1.598±0.228
CB 126	0.048±0.016	0.224±0.023	0.033±0.004	0.095±0.040	1.703±0.251
CB 128	0.048±0.016	0.174±0.023	0.151±0.024	0.353±0.054	0.691±0.097
CB 138	0.136±0.023	2.063±0.479	2.083±0.224	4.816±0.915	5.649±0.465
CB 146	0.059±0.015	0.627±0.051	0.545±0.055	1.188±0.113	0.604±0.111
CB 153	0.206±0.020	5.075±0.445	3.858±0.365	8.063±1.195	7.242±1.658
CB 156	0.048±0.016	0.228±0.074	0.193±0.037	0.480±0.102	0.648±0.102
CB 167	0.048±0.016	0.125±0.013	0.166±0.020	0.244±0.077	0.776±0.100
CB 170	0.057±0.015	2.249±0.219	0.774±0.144	1.971±0.444	1.878±0.194
CB 171	0.048±0.016	0.041±0.006	0.093±0.013	0.217±0.027	0.774±0.065
CB 180	0.131±0.015	6.576±0.772	3.117±0.390	6.980±1.296	7.752±0.815
CB 183	0.048±0.016	0.412±0.032	0.229±0.024	0.479±0.097	0.527±0.086
CB 189	0.048±0.016	0.044±0.007	0.042±0.007	0.137±0.038	0.106±0.013
CB 194	0.050±0.016	1.887±0.208	0.803±0.086	1.795±0.202	1.218±0.174
CB 195	0.048±0.016	0.041±0.006	0.192±0.018	0.457±0.035	0.115±0.015
CB 196	0.068±0.015	2.998±0.302	0.033±0.004	0.095±0.040	1.863±0.985
CB 201	0.048±0.016	2.085±0.261	0.245±0.084	1.448±0.459	2.794±0.356
Aroclor 1260	0.344±0.132	0.270±0.039	0.230±0.021	0.723±0.329	79.486±8.112

[1]Diet A = 75% ocean fish, Diet B = 75% fish collected above the Oak Ridge Reservation, Diet C = 25% fish collected on the Oak Ridge Reservation and 50% ocean fish, Diet D = 50% fish collected on the Oak Ridge Reservation and 25% ocean fish, Diet E = 75% fish collected on the Oak Ridge Reservation.

Table E.7. Mean ±SE Aroclor 1260 and PCB congener concentrations (ppm, wet wt) in fat tissue from female mink (n=8/diet) fed various diets[1]

	Diet A	Diet B	Diet C	Diet D	Diet E
CB 77	0.011±0.005	0.035±0.006	0.046±0.006	0.083±0.019	0.138±0.011
CB 81	0.120±0.042	0.339±0.061	0.280±0.052	0.429±0.023	0.696±0.078
CB 99	0.050±0.018	0.169±0.029	0.786±0.168	1.447±0.265	1.708±0.272
CB 101	0.016±0.006	0.098±0.020	0.107±0.018	0.263±0.029	0.311±0.037
CB 118	0.163±0.066	0.464±0.089	1.461±0.276	2.943±0.388	3.175±0.299
CB 123	0.130±0.036	0.458±0.088	0.033±0.006	0.063±0.006	2.375±0.330
CB 126	0.064±0.029	1.171±0.212	0.196±0.032	0.481±0.035	1.511±0.160
CB 128	0.033±0.020	0.299±0.048	0.352±0.059	0.781±0.080	1.210±0.097
CB 138	0.267±0.135	2.800±0.609	3.464±0.638	9.543±1.036	9.925±0.833
CB 146	0.041±0.015	0.500±0.104	0.600±0.097	1.421±0.111	1.771±0.179
CB 153	0.405±0.230	5.538±1.069	5.195±0.988	15.714±1.686	15.375±1.349
CB 156	0.009±0.003	0.469±0.084	0.409±0.073	0.914±0.086	1.188±0.077
CB 167	0.040±0.019	0.350±0.055	0.621±0.109	1.347±0.126	1.500±0.125
CB 170	0.093±0.051	2.278±0.471	0.800±0.157	2.043±0.238	3.013±0.168
CB 171	0.035±0.017	0.521±0.093	0.488±0.086	1.083±0.100	1.393±0.084
CB 180	0.290±0.188	7.175±1.544	5.264±1.034	14.571±1.325	14.750±1.161
CB 183	0.019±0.012	0.559±0.102	0.368±0.069	0.899±0.073	1.114±0.143
CB 189	0.005±0.001	0.090±0.019	0.041±0.006	0.130±0.015	0.186±0.018
CB 194	0.060±0.035	2.103±0.475	0.918±0.174	2.386±0.201	2.688±0.351
CB 195	0.011±0.007	0.419±0.078	0.033±0.006	0.063±0.006	0.133±0.021
CB 196	0.021±0.014	0.778±0.163	0.426±0.091	1.064±0.105	1.645±0.295
CB 201	0.031±0.019	0.889±0.151	0.477±0.080	1.226±0.101	1.813±0.178
Aroclor 1260	3.169±1.849	61.250±12.560	0.261±0.044	105.86±11.26	128.63±7.73

[1]Diet A = 75% ocean fish, Diet B = 75% fish collected above the Oak Ridge Reservation, Diet C = 25% fish collected on the Oak Ridge Reservation and 50% ocean fish, Diet D = 50% fish collected on the Oak Ridge Reservation and 25% ocean fish, Diet E = 75% fish collected on the Oak Ridge Reservation.

Table E.8. Mean[1] ±SE Aroclor 1260 and PCB congener concentrations (ppm, wet wt) in liver tissue from 6-week-old mink kits from dams fed diets of fish collected from various sources[2]

	Diet A	Diet B	Diet C	Diet E
CB 77	0.003±0.000 (9)	0.006±0.001 (9)	0.007±0.001 (8)	0.006±0.001 (9)
CB 81	0.004±0.000 (9)	0.006±0.001 (9)	0.007±0.001 (8)	0.007±0.001 (9)
CB 99	0.003[a]±0.000 (9)	0.006[a]±0.001 (9)	0.016[a]±0.005 (8)	0.049[b]±0.012 (9)
CB 101	0.004±0.000 (9)	0.006±0.001 (9)	0.007±0.001 (8)	0.006±0.001 (9)
CB 118	0.012[a]±0.001 (9)	0.012[a]±0.002 (9)	0.068[b]±0.014 (8)	0.138[c]±0.025 (9)
CB 123	0.008±0.001 (9)	0.007±0.000 (9)	0.007±0.001 (8)	0.006±0.001 (9)
CB 126	0.003[a]±0.000 (9)	0.007[a]±0.001 (9)	0.007[a]±0.001 (8)	0.014[b]±0.003 (9)
CB 128	0.003[a]±0.000 (9)	0.006[a]±0.001 (9)	0.020[a]±0.005 (8)	0.092[b]±0.034 (9)
CB 138	0.015[a]±0.002 (9)	0.058[a]±0.011 (9)	0.141[a]±0.033 (8)	0.547[b]±0.200 (9)
CB 146	0.004[a]±0.000 (9)	0.011[a]±0.002 (9)	0.033[a]±0.006 (8)	0.143[b]±0.042 (9)
CB 151	0.004±0.000 (9)	0.006±0.001 (9)	0.007±0.001 (8)	0.006±0.001 (9)
CB 153	0.011[a]±0.002 (9)	0.065[a]±0.010 (9)	0.157[a]±0.039 (8)	0.509[b]±0.137 (9)
CB 156	0.003±0.000 (9)	0.008±0.001 (9)	0.009±0.001 (8)	0.006±0.001 (9)
CB 167	0.004[a]±0.000 (9)	0.007[a]±0.001 (9)	0.009[a]±0.002 (8)	0.028[b]±0.007 (9)
CB 170	0.004[a]±0.000 (9)	0.054[a]±0.009 (9)	0.078[b]±0.014 (8)	0.294[b]±0.118 (9)
CB 171	0.003±0.000 (9)	0.008±0.001 (9)	0.007±0.001 (8)	0.006±0.001 (9)
CB 180	0.006[a]±0.001 (9)	0.109[a]±0.018 (9)	0.194[b]±0.040 (8)	0.629[b]±0.235 (9)
CB 183	0.004[a]±0.000 (9)	0.007[a]±0.000 (9)	0.013[a]±0.004 (8)	0.035[b]±0.009 (9)
CB 189	0.003±0.000 (9)	0.006±0.001 (9)	0.007±0.001 (8)	0.008±0.002 (9)
CB 194	0.004[a]±0.000 (9)	0.019[a]±0.003 (9)	0.041[b]±0.008 (8)	0.146[b]±0.055 (9)
CB 195	0.003±0.000 (9)	0.006±0.001 (9)	0.007±0.001 (8)	0.006±0.001 (9)
CB 196	0.004[a]±0.000 (9)	0.052[a]±0.011 (9)	0.084[b]±0.015 (8)	0.319[b]±0.128 (9)
CB 201	0.003[a]±0.000 (9)	0.014[a]±0.002 (9)	0.027[a]±0.005 (8)	0.142[b]±0.032 (9)
Aroclor 1260	0.099±0.016 (9)	0.151±0.099 (9)	0.058±0.009 (8)	0.048±0.008 (9)

[1]Means with different superscripts are significantly different, $P \leq 0.05$.
[2]Diet A = 75% ocean fish, Diet B = 75% fish collected above the Oak Ridge Reservation, Diet C = 25% fish collected on the Oak Ridge Reservation and 50% ocean fish, Diet E = 75% fish collected on the Oak Ridge Reservation.

Table E.9. Mean[1] ±SE Aroclor 1260 and PCB congener concentrations (ppm, wet wt) in carcass[2] of 6-week-old mink kits from dams fed diets of fish collected from various sources[3]

	Diet A	Diet B	Diet C	Diet E
CB 77	0.003±0.000 (9)	0.003±0.000 (9)	0.003±0.000 (8)	0.003±0.000 (9)
CB 81	0.003±0.000 (9)	0.003±0.000 (9)	0.003±0.000 (8)	0.005±0.001 (9)
CB 99	0.003^{a}±0.000 (9)	0.009^{a}±0.003 (9)	0.017^{a}±0.003 (8)	0.096^{b}±0.023 (9)
CB 101	0.003^{a}±0.000 (9)	0.004^{a}±0.001 (9)	0.006^{a}±0.001 (8)	0.017^{b}±0.002 (9)
CB 118	0.008^{a}±0.001 (9)	0.030^{a}±0.009 (9)	0.071^{a}±0.018 (7)	0.245^{b}±0.036 (9)
CB 123	0.003±0.000 (9)	0.011±0.005 (9)	0.003±0.000 (8)	0.008±0.001 (9)
CB 126	0.003±0.000 (9)	0.011±0.001 (9)	0.005±0.001 (8)	0.003±0.000 (9)
CB 128	0.003^{a}±0.000 (9)	0.011^{a}±0.004 (9)	0.012^{a}±0.002 (8)	0.054^{b}±0.007 (9)
CB 138	0.008^{a}±0.001 (9)	0.112^{a}±0.016 (9)	0.106^{a}±0.021 (7)	0.634^{b}±0.110 (9)
CB 146	0.003^{a}±0.000 (9)	0.013^{a}±0.002 (9)	0.019^{a}±0.003 (8)	0.103^{b}±0.017 (9)
CB 151	0.003±0.000 (9)	0.003±0.000 (9)	0.003±0.000 (8)	0.005±0.001 (9)
CB 153	0.009^{a}±0.001 (9)	0.141^{a}±0.024 (9)	0.119^{a}±0.025 (7)	0.619^{b}±0.096 (9)
CB 156	0.003^{a}±0.000 (9)	0.017^{ab}±0.003 (9)	0.022^{b}±0.003 (8)	0.058^{c}±0.008 (9)
CB 167	0.003^{a}±0.000 (9)	0.014^{bc}±0.002 (9)	0.010^{ab}±0.002 (8)	0.022^{c}±0.003 (9)
CB 170	0.003^{a}±0.000 (9)	0.073^{a}±0.012 (9)	0.047^{a}±0.007 (8)	0.238^{b}±0.036 (9)
CB 171	0.003^{a}±0.000 (9)	0.021^{a}±0.003 (9)	0.008^{a}±0.004 (8)	0.017^{b}±0.002 (9)
CB 180	0.004^{a}±0.000 (9)	0.203^{a}±0.026 (9)	0.121^{a}±0.019 (7)	0.611^{b}±0.080 (9)
CB 183	0.003^{a}±0.000 (9)	0.011^{a}±0.001 (9)	0.007^{a}±0.002 (8)	0.031^{b}±0.005 (9)
CB 189	0.003±0.000 (9)	0.003±0.000 (9)	0.003±0.000 (8)	0.008±0.001 (9)
CB 194	0.003^{a}±0.000 (9)	0.025^{a}±0.003 (9)	0.019^{a}±0.004 (8)	0.089^{b}±0.010 (9)
CB 195	0.003^{a}±0.000 (9)	0.003^{a}±0.000 (9)	0.004^{a}±0.001 (8)	0.023^{b}±0.003 (9)
CB 196	0.003±0.000 (9)	0.014±0.002 (9)	0.005±0.002 (8)	0.005±0.001 (9)
CB 201	0.003^{a}±0.000 (9)	0.013^{a}±0.002 (9)	0.012^{a}±0.002 (8)	0.050^{b}±0.009 (9)
Aroclor 1260	0.082±0.005 (9)	1.791±0.282 (9)	0.353±0.245 (8)	0.049±0.006 (9)

[1]Means with different superscripts are significantly different, $P<0.05$. Diet groups were separated based on lipid adjusted concentrations.

[2]Whole body minus liver tissue.

[3]Diet A = 75% ocean fish, Diet B = 75% fish collected above the Oak Ridge Reservation, Diet C = 25% fish collected on the Oak Ridge Reservation and 50% ocean fish, Diet E = 75% fish collected on the Oak Ridge Reservation.

Table E.10. Reproductive performance of female mink fed diets of 75% fish from various sources[1]

	Diet A	Diet B	Diet C	Diet D	Diet E
Females whelping	6	7	4	6	8
Female weights					
December[2]	1269±64[3]	1245±63	1374±64	1258±64	1230±64
June[4]	1168±84[a]	1016±81	1134±81	1020±81	935±81[b]
Gestation (days)	44.6	46.4	44.3	47.5	44.9
Kit wts (6 wks)	328±14	311±10	333±20	307±12	295±11
Females	296±38				268±37
Males	376±42[a]				312±44[b]
Litter size[5]	6.9[a]	7.3[a]	7.8[a]	6.0[ab]	4.3[b]
EROD[6]	51±13[a]	134±26[ab]	124±32[ab]	276±33[b]	262.54±31[b]

[1]Diet A = 75% ocean fish, Diet B = 75% fish collected above the Oak Ridge Reservation, Diet C = 25% fish collected on the Oak Ridge Reservation and 50% ocean fish, Diet D = 50% fish collected on the Oak Ridge Reservation and 25% ocean fish, Diet E = 75% fish collected on the Oak Ridge Reservation.
[2]Beginning of study.
[3]Means followed by different superscripts are significantly different, $P<0.05$.
[4]End of study.
[5]Kits/female.
[6]Ethoxyresorufin-o-deethylase, pmoles/mg protein/min.