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Source: Journal of Wildlife Diseases, 23(2) : 279-291

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-23.2.279

Journal of Wildlife Diseases, 23(2), 1987, pp. 279–291

ENVIRONMENTAL POLLUTANT AND NECROPSY DATA FOR OSPREYS FROM THE EASTERN UNITED STATES, 1975–1982

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ABSTRACT: Twenty-three ospreys (*Pandion haliaetus*) found dead or moribund in the eastern United States during 1975–1982 were necropsied and selected tissues were analyzed for organochlorines and metals. Major causes or factors contributing to death were trauma, impact injuries, and emaciation. DDE was detected in 96% of the osprey carcasses, DDD in 65%, DDT and heptachlor epoxide in 13%, dieldrin, oxychlordane, and *cis*-nonachlor in 35%, *cis*-chlordane in 52%, *trans*-nonachlor in 45%, and PCB's in 83%. Carcasses of immature ospreys from the Chesapeake Bay had significantly lower concentrations of DDE, DDD + DDT, *cis*-chlordane, and PCB's than carcasses of adults from the same area. Concentrations of some organochlorines in ospreys from the Chesapeake Bay declined significantly from 1971–1973 to 1975–1982. Significant differences in concentrations of certain metals in the ospreys' livers were noted between time periods, and sex and age groups for birds from the Chesapeake Bay. During 1975–1982, adults had significantly lower concentrations of chromium, copper, and arsenic than immatures and nestlings, and adult males had higher mercury concentrations than adult females. Adult females had lower zinc concentrations in 1975–1982 than in 1971–1973. Immatures and nestlings had higher concentrations of chromium and lead in 1975–1982 than in 1971–1973. A slightly elevated concentration of chromium (1.7 ppm) or arsenic (3.2 ppm) was found in the livers of individual ospreys. Several ospreys had elevated concentrations of mercury in their livers; two ospreys had more than 20 ppm which may have contributed to their deaths.

Key words: Osprey, *Pandion haliaetus*, organochlorines, trace elements, mortality, environmental pollutants, eastern United States.

INTRODUCTION

Many avian species rely on the productivity of estuaries for their food source. A variety of pollutants may affect estuary bird populations in diverse ways (Blus et al., 1977b). Therefore, the quality of estuaries is important and must be monitored to detect factors that may cause adverse impacts. Ospreys (*Pandion haliaetus*) are relatively abundant in many estuarine areas along the East Coast of the United States. Ospreys feed almost exclusively on fish and are good biological indicators of persistent contaminants in these ecosystems. Organochlorines have been implicated as a causative factor in the decline of ospreys in the northeastern United States (Ames, 1966; Wiemeyer et al., 1975; Spitzer et al., 1978; Wiemeyer et al., 1978; Wiemeyer et al., 1980). Wiemeyer et al. (1975) reported on the causes of death of six ospreys from Connecticut and Virginia in 1967–1970 and provided data on organochlorines in tissues. Seven ospreys from Florida that died during 1969–1976 were analyzed for DDT and metabolites and dieldrin (Johnston, 1978), and DDT and metabolites, dieldrin, and PCB's were reported for four ospreys found dead in Florida in 1974–1975 (Sundlof et al., 1986). Causes of death of 33 ospreys from the eastern United States in 1964–1973 and environmental contaminants in 26 of these birds were previously reported (Wiemeyer et al., 1980). We report here on necropsy results and contaminant levels for 23 ospreys found dead or dying in the eastern United States during 1975–1982, a period

when populations were recovering (Spitzer et al., 1978; Spitzer and Poole, 1980).

MATERIALS AND METHODS

Collection

Ospreys from the eastern United States were obtained during 1975–1982. Most birds were found dead; a few died shortly after they were found sick or injured. All large nestlings were euthanized because major deformities or severe injuries precluded rehabilitation. Most birds were frozen before necropsy. Birds were aged on the basis of plumage or on banding information. Major emphasis was placed on obtaining birds during or shortly following the breeding season (April–August). Some migrant birds also were obtained.

Necropsy

Twenty ospreys were necropsied at the National Wildlife Health Center, Madison, Wisconsin. Three were necropsied at the Patuxent Wildlife Research Center, Laurel, Maryland. Most carcasses were weighed and subsequently examined externally for any gross abnormalities. The head was decapitated and the brain removed and saved for later chemical analysis. The liver, heart, kidneys, and alimentary canal were removed, and sex was determined by examination of the gonads. After examination, the heart was returned to the carcass and the carcass was wrapped in aluminum foil and frozen for later chemical studies. Entire brain, liver, and kidneys were placed separately in chemically clean jars and frozen for later analysis. The alimentary canal was opened and examined for parasitic helminths. When lesions were present, small pieces of liver, lung, spleen, or kidney were placed in 10% formalin, fixed, sectioned, and stained with hematoxylin and eosin for histopathological studies. For osprey numbers 5, 10–13, 17, 18, 21, and 23 (Table 1), cloacal and tracheal swabs were collected and placed in transport media consisting of Hank's balanced salt solution with 0.5% gelatin, 1,500 IU penicillin, 1,500 μg streptomycin, 100 μg gentamicin, and 100 IU mycostatin per ml. They were either submitted immediately after necropsy for virus isolation attempts or stored at −70 C in liquid nitrogen until they could be submitted. Portions of various tissues including lung, liver, airsacs, and heart blood were saved for bacteriological studies. All microbiological studies were conducted using established techniques for isolation of avian pathogens (Hitchner et al., 1980).

Chemical analysis

Tissue samples for organochlorine analyses were prepared, extracted, and separated as described by Cromartie et al. (1975) and Kaiser et al. (1980). The elution and quantitation of organochlorines in birds collected in 1975–1976 and in two birds (No. 7 and 14) in 1977 were the same as those described by Cromartie et al. (1975); the remainder were analyzed according to the methods of Kaiser et al. (1980). Methods used in kepone analysis were those described by Stafford et al. (1978). Lower limits of detection in ppm wet weight were 0.10 for insecticides and metabolites and 0.50 for PCB's.

Liver samples were analyzed for lead, copper, zinc, cadmium, chromium, and arsenic as described by Haseltine et al. (1981). Samples were analyzed for iron and nickel in the same manner as lead. Samples were prepared for mercury analysis as described by Monk (1961) and determinations were made using the method of Hatch and Ott (1968). The lower limits of detection for metals and trace elements in ppm wet weight for most birds were: 0.02 or 0.05 for mercury; 0.05 for chromium and arsenic; 0.10 for lead, copper, zinc, nickel, and cadmium; and 1.0 for iron. Because of a small amount of tissue, the lower limits of detection for one osprey (No. 18) were 0.20 ppm for chromium and 0.50 ppm for lead, copper, zinc, and cadmium.

All residues reported herein are on a wet weight basis, except for some values reported from the literature that are on a dry weight basis and are designated (dw). Dry weight values can be converted to approximate wet weight values by dividing the former by four.

Statistical analysis

Contaminant concentrations were converted to common logarithms primarily to equalize variances between groups, but also to correct for the skewed distribution of values. Equality of variances and skewness were evaluated for nontransformed and log-transformed data. The assumptions of the statistical analyses were more closely met by use of log-transformed data. Values below the lower limits of detection were set to one-half that limit. Comparisons between groups were made with t-tests. If more than one-half of the values for a given contaminant in a given comparison were below the detection limit, a statistical comparison was not made. Ninety-five percent confidence intervals (CI) are reported for geometric means. Concentrations of DDD and DDT were combined for statistical comparisons because DDD may be rapidly formed from DDT in some avian tissues during frozen storage (Walker and Jefferies, 1978). Some

comparisons used data from a previously published paper (Wiemeyer et al., 1980). Statistical significance was determined at $P \leq 0.05$.

RESULTS

Collection data and sex ratios

More ospreys were obtained from the Chesapeake Bay area than any other single area. These included one osprey (No. 3) from Matthews County, Virginia; three (No. 11, 12, and 14) from the lower Potomac River; four (No. 6, 7, 10, and 13) from Talbot County, Maryland; and three (No. 5, 8, and 9) from Anne Arundel County, Maryland (Table 1). All Maryland and Virginia ospreys, with the possible exception of No. 11, were obtained during or shortly after the breeding season. No. 11 was collected during the beginning of a migratory period. Seventy-five percent of the Maryland and Virginia adults had been banded as nestlings in the Chesapeake Bay area. All of the remaining ospreys with known collection dates, except for bird No. 15 from New Jersey and bird No. 2 from North Carolina, were collected during periods of migration (Henny and Van Velzen, 1972). Osprey No. 1 originally had been banded as a nestling in Massachusetts, but was found dead in the Savannah River Swamp in Jasper County, South Carolina. Newburyport, Massachusetts (osprey No. 21) is near the mouth of the Merrimack River.

Six of 14 adults were males and eight were females (Table 1). The sex ratio was not different ($P > 0.05$) from 1:1 or from the sex ratio of adult ospreys found dead during 1964–1973 (Wiemeyer et al., 1980). Five of nine immatures in our current sample were females, three were males, and the sex of one was not determined.

Necropsy data

Trauma, impact injuries and emaciation were major causes or factors contributing to death (Table 1). Virological tests for avian influenza and Newcastle disease were negative. *Staphylococcus aureus* was isolated from lung, liver, pericardial sac, and peritoneal fluid of osprey No. 17. Pathogenic bacteria were not isolated from other ospreys. Only one osprey (No. 22) possibly had been shot.

Osprey No. 4 had a 1 cm wide indentation of the muscles overlying the medial portion of the right tibiotarsus, the same width as a plastic band on that leg. The right foot and lower right calf were slightly edematous. The feathers were worn off in the area of the indentation and some small fly larvae were present. The muscle under the indentation was scarred and the muscle distal to the area was necrotic. Apparently, the band had been pushed onto that portion of the leg from its normal position on the tarsometatarsus, causing a reduction in circulation and necrosis, leading to emaciation and eventual death. Several hundred trematodes identified as *Ribeiroia ondatrae* were present in the bird's ventriculus. Osprey No. 3 also had a 1 cm wide area on its right calf with missing feathers and bruising of underlying tissues. However, this injury may not have contributed to the bird's death.

All three Maryland nestlings had leg injuries or deformities. One nestling (No. 12) had become entangled in monofilament line. The left foot had developed dry gangrene due to the line that had nearly severed the leg at mid-tibiotarsus. The left tibiotarsus showed signs of severe osteomyelitis. Osprey No. 13 had a marked lateral deviation of the left tibiotarsus, which was bowed. The proximal epiphysis was inflamed and hemorrhagic. The left leg had cubital ulcers over the tarsal-metatarsal and tibio-tarsal joints. The feet appeared to have been useless. Osprey No. 14 had suspected developmental joint abnormalities; the right and left tarsometatarsus were rotated 90 and 180 degrees medially.

Organochlorines

Organochlorines in carcasses of the ospreys varied widely from nondetectable

TABLE 1. Collection, necropsy, and body condition data on ospreys from the eastern United States, 1975–1982.

Bird number	Collection locality	Collection date	Age and sex[a]	Weight at necropsy (g)	% Lipid in carcass	Diagnosis and comments
1[b]	Tillman, SC	18 Oct 1976	A (9) M	965	0.8	Emaciation; probable stomach ulcer
2	Pea Island NWR, NC	7 Aug 1981	I F	1,270	7.1	Terminal trauma; foot abrasions
3[c]	Pine Hall, VA	13 Apr 1980	A (6) M	1,730	5.2	Trauma; injury to right leg
4[c]	Cape Charles, VA	1 Aug 1981	A (10) M	1,000	0.9	Emaciation; necrosis of right leg
5	Annapolis, MD	24 May 1976	A F	1,738	5.2	Impact—radio tower
6	Easton, MD	15 June 1977	A M	1,250	1.5	Emaciation and blunt trauma
7[d]	St. Michaels, MD	16 Jun 1977	A (7) F	1,690	3.9	Open[e]; spleen enlarged
8[f]	Churchton, MD	15 Aug 1981	A (9) F	1,900	11.0	Open
9[g]	Rhode River, MD	11 Apr 1982	A (6) F	2,000	5.6	Fractured radius from osprey attack; possible drowning
10[c]	Easton, MD	2 May 1982	A (5) F	1,650	5.9	Prolapsed oviduct; possible blunt trauma
11	Newburg, MD	26 Aug 1975	I U	1,487	9.3	Impact; possible mercury involvement
12	Rock Point, MD	19 Jul 1976	N M	1,200	2.5	Gangrene of foot; entangled in monofilament line; euthanized
13	St. Michaels, MD	19 Jul 1976	N F	698	2.0	Damage to epiphysis of leg bones; euthanized
14	Rock Point, MD	30 Jun 1977	N F	—[h]	6.6	Developmental joint abnormalities of legs; euthanized
15[i]	Brigantine NWR, NJ	11 Aug 1976	A (11) F	2,184	26.1	Open
16	Ocean City, NJ	9 Oct 1976	A F	1,561	18.5	Electrocution and impact
17	Spring Lake, NJ	26 Oct 1975	I M	1,392	12.9	Possible pneumonia or airsaculitis
18	Cape May, NJ	29 Oct 1975	I M	962	0.3	Open; emaciation and dehydration
19	MA	1976	A M	1,565	17.6	Impact
20	MA	25 Oct 1976	I F	—	0.2	Open; emaciation, possible anemia
21	Newburyport, MA	18 Oct 1979	I F	1,025	0.1	Emaciation; possible mercury poisoning
22	Janesville, WI	24 Sep 1975	A M	1,380	13.9	Possibly shot; fracture of wing
23	Missouri Valley, IA	1 Oct 1979	A F	1,150	0.5	Cloacal impaction; emaciation

[a] A, adult (values in parentheses indicate calendar year of life when known); I, immature, in first calendar year; N, nestling; M, male; F, female; U, unknown.
[b] Banded as a nestling in Massachusetts.
[c] Banded as a nestling near recovery site.
[d] Banded as a nestling on lower Potomac River, MD.
[e] Open, cause of death unknown.
[f] Banded as a nestling in Talbot Co., MD.
[g] Banded as a nestling in Matthews Co., VA.
[h] Not determined.
[i] Banded as a nestling in Cape May Co., NJ.

TABLE 2. Organochlorine residues in carcasses of ospreys from the eastern United States, 1975–1982.

Source and bird number	Residues (ppm wet weight)[a]									
	p,p'-DDE	p,p'-DDD	p,p'-DDT	Dieldrin	HE	OC	C-C	T-N	C-N	PCB's
South Carolina										
1[b]	8.8	0.30	—[c]	—	—	0.14	—	—	—	140
North Carolina										
2	0.30	—	—	—	—	—	—	—	—	0.83
Virginia										
3	4.8	2.0	1.30	—	—	—	—	—	—	8.8
4	17	1.6	—	0.16	0.11	0.13	0.24	0.16	0.16	25
Maryland										
5[d]	4.5	1.5	0.19	0.18	—	—	0.13	0.12	—	3.8
6[e]	48	3.8	—	2.2	0.38	0.35	0.68	na[f]	0.48	48
7	3.5	0.68	—	0.33	—	0.10	0.24	0.16	0.16	23
8	3.5	0.95	—	0.17	0.15	0.33	0.40	0.13	0.28	15
9	2.5	0.90	0.16	—	—	0.16	0.50	0.18	0.30	13
10	2.3	0.73	—	—	—	0.12	0.33	0.20	0.33	6.8
11	1.7	—	—	—	—	—	—	—	—	8.0
12[g]	0.63	0.30	—	—	—	—	0.10	0.10	—	3.0
13[g]	0.63	0.15	—	0.18	—	—	0.10	0.11	—	1.9
14	0.63	0.24	—	—	—	—	0.11	0.11	—	5.5
New Jersey										
15	31	13	—	0.30	—	0.17	0.48	0.28	0.28	24
16	—	—	—	—	—	—	—	—	—	—
17	0.11	—	—	—	—	—	—	—	—	—
18	0.60	—	—	—	—	—	—	—	—	1.1
Massachusetts										
19	0.28	—	—	—	—	—	—	—	—	0.95
20	0.63	0.21	—	—	—	—	—	—	—	1.2
21	0.38	—	—	—	—	—	—	—	—	—
Wisconsin										
22	1.5	0.58	—	0.20	—	—	0.25	—	0.11	0.70
Iowa										
23	0.20	—	—	—	—	—	—	—	—	—

[a] HE, heptachlor epoxide; OC, oxychlordane; C-C, *cis*-chlordane; T-N, *trans*-nonachlor; C-N, *cis*-nonachlor; PCB's, polychlorinated biphenyls.
[b] Carcass also contained 0.11 ppm mirex.
[c] —, none detected.
[d] Liver analyzed for kepone; 0.28 ppm present.
[e] Carcass also contained 0.32 ppm mirex.
[f] na, not analyzed.
[g] Liver analyzed for kepone; none detected.

concentrations to moderate and occasionally high concentrations (Table 2). DDE was detected in 96% of the osprey carcasses, DDD in 65%, DDT and heptachlor epoxide in 13%, dieldrin, oxychlordane, and *cis*-nonachlor in 35%, *cis*-chlordane in 52%, *trans*-nonachlor in 45% (not analyzed in one), and PCB's in 83%. PCB residues resembled Aroclor® 1260 in 16 of the 19 carcasses in which they were detected, and resembled Aroclor 1254 in the remaining three (No. 1, 7, and 14). Aroclor

is the commercial trade name for several PCB's containing varying percentages of chlorine as indicated by the last two digits in the 1200 series type number. Although brains were analyzed, the results are not reported because organochlorine concentrations in brain on a wet weight basis are highly dependent on concentrations in carcass on a lipid weight basis (Wiemeyer and Cromartie, 1981). Concentrations in brains were far below values associated with organochlorine induced mortality.

For the years 1975–1982, carcasses of four immature Chesapeake Bay ospreys contained lower concentrations of DDE, DDD + DDT, *cis*-chlordane, and PCB's than carcasses of eight adults (DDE: $\bar{x}$ = 0.81 ppm, CI 0.37–1.8, versus 5.8 ppm, CI 2.4–14, $P = 0.006$; DDD + DDT: $\bar{x}$ = 0.21 ppm, CI 0.09–0.51, versus 1.5 ppm, CI 0.87–2.5, $P = 0.0004$; *cis*-chlordane: $\bar{x}$ = 0.09, CI 0.05–0.15, versus 0.25, CI 0.13–0.51, $P = 0.035$; PCB's: $\bar{x}$ = 4.0 ppm, CI 1.4–11, versus 14 ppm, CI 7.0–27, P = 0.024). Concentrations of dieldrin and *trans*-nonachlor, and percent lipid of wet weight in carcasses were similar between age classes ($P > 0.15$). Five adult female ospreys from Maryland in 1976–1982 had lower concentrations of DDE, DDD + DDT, dieldrin, and heptachlor epoxide than five collected in 1971–1973 (DDE: $\bar{x}$ = 3.2 ppm, CI 2.2–4.4, versus 16 ppm, CI 7.7–35, $P = 0.001$; DDD + DDT: $\bar{x}$ = 1.0 ppm, CI 0.67–1.5, versus 4.2 ppm, CI 1.4–12, $P = 0.009$; dieldrin: $\bar{x}$ = 0.12 ppm, CI 0.04–0.34, versus 0.82 ppm, CI 0.62–1.1, $P = 0.001$; heptachlor epoxide: $\bar{x}$ = 0.06, CI 0.03–0.11, versus 0.28 ppm, CI 0.19–0.43, $P = 0.001$). Concentrations of oxychlordane, *cis*-nonachlor, and PCB's, and percent lipid of wet weight in carcasses were similar between periods ($P > 0.10$). Immatures from Maryland and Virginia in 1975–1977 ($n = 4$) had lower concentrations of DDD + DDT and dieldrin (DDD + DDT: $\bar{x}$ = 0.21 ppm, CI 0.09–0.51, versus 0.73 ppm, CI 0.59–0.90, P = 0.0005; dieldrin: $\bar{x}$ = 0.08 ppm, CI 0.03–0.20, versus 0.23 ppm, CI 0.15–0.36, P = 0.009), but not DDE or PCB's ($P > 0.15$), than those obtained in 1971–1973 ($n = 7$). Percent lipid of wet weight in carcasses was similar between periods ($P > 0.15$). Adult male ($n = 3$) ospreys collected during 1975–1982 had significantly higher concentrations of DDE and DDD + DDT than adult females ($n = 5$) collected during the same period (DDE: $\bar{x}$ = 16 ppm, CI 0.92–271, versus 3.2 ppm, CI 2.2–4.4, P = 0.021; DDD + DDT: $\bar{x}$ = 1.0 ppm, CI 0.67–1.5, versus 2.8 ppm, CI 0.91–8.4, P = 0.011). Concentrations of dieldrin, oxychlordane, *cis*-chlordane, *cis*-nonachlor, and PCB's, and percent lipid of wet weight in carcass were similar for both sexes ($P > 0.05$).

Metals

Wide ranges of metal concentrations were found in the livers of ospreys (Table 3). A few ospreys had clearly elevated concentrations of certain metals. Bird No. 18 from Cape May, New Jersey, had 1.7 ppm chromium in its liver which was more than four times the next highest concentration. Copper concentrations exceeded 10 ppm only in livers of immature and nestling ospreys. The 3.2 ppm arsenic in an adult (No. 15) from Brigantine National Wildlife Refuge, New Jersey, was more than six times the next highest value. The concentration of lead in the Massachusetts bird (No. 21) was considerably higher than that in other ospreys; this osprey also had the highest cadmium and mercury concentrations and was the only bird with a detectable concentration of nickel. Livers of only six ospreys were analyzed for nickel and iron.

Mercury concentrations were clearly elevated (>10 ppm) in four birds. Osprey No. 21, with the highest mercury concentration, was an emaciated female collected in Massachusetts. This osprey had hemorrhagic enteritis, marked retention of urates in both kidneys, and its cloaca was

TABLE 3. Trace element concentrations in livers of ospreys from the eastern United States, 1975–1982.

Source and bird number	Residues (ppm wet weight)								
	Cr	Cu	Zn	As	Cd	Hg	Pb	Ni	Fe
South Carolina									
1	0.40	7.3	73	0.17	0.23	3.8	0.69	na[a]	na
North Carolina									
2	—[b]	3.30	69	—	—	3.7	—	—	690
Virginia									
3	0.09	2.3	27	—	0.23	13	1.0	na	na
4	—	2.8	150	—	—	6.7	—	—	2,800
Maryland									
5	0.22	4.8	30	0.07	0.14	2.7	0.50	na	na
6	0.15	3.9	25	—	0.17	9.1	0.46	na	na
7	0.22	3.3	23	0.13	0.14	1.5	0.41	na	na
8	—	2.2	34	—	—	2.2	—	—	860
9	0.13	2.4	19	—	—	0.85	—	—	320
10	—	2.6	34	—	0.20	0.28	—	—	340
11	0.27	12	27	0.13	0.10	21	0.34	na	na
12	0.22	17	34	0.06	—	0.55	0.42	na	na
13	0.28	30	28	0.47	—	0.62	0.42	na	na
14	0.40	55	33	0.10	—	0.40	0.49	na	na
New Jersey									
15	0.27	3.2	74	3.20	0.13	1.1	1.4	na	na
16	0.21	1.8	73	0.08	0.17	2.9	0.39	na	na
17	0.20	1.5	63	0.15	—	3.4	0.39	na	na
18	1.70	11	120	—	0.21	8.5	1.7	na	na
Massachusetts									
21	0.22	8.1	89	—	0.74	35	4.6	0.28	920
Wisconsin									
22	0.35	5.5	59	0.09	0.22	11	0.73	na	na
Iowa									
23	0.35	5.3	98	na	0.18	4.3	1.5	na	na

[a] na, not analyzed.
[b] —, none detected.

filled with a thick white pasty accumulation of urates. The osprey (No. 11) with the second highest mercury concentration was in good flesh with good deposits of fat, and apparently had been struck by a vehicle in Maryland. Unfortunately, peripheral nervous tissue was not taken for histopathological evaluation, which may have shown damage indicative of methylmercury poisoning.

Significantly different concentrations of metals were noted between several groups of ospreys collected from the Chesapeake Bay. Some comparisons included data from an earlier report (Wiemeyer et al., 1980). During 1975–1982, eight adults had lower concentrations of chromium, copper, and arsenic than four immatures and nestlings (Cr: $\bar{x}$ = 0.08 ppm, CI 0.03–0.18, versus 0.29 ppm, CI 0.19–0.42, P = 0.029; Cu: $\bar{x}$ = 2.9 ppm, CI 2.3–3.7, versus 24 ppm, CI 8.3–70, P = 0.00002; As: $\bar{x}$ = 0.03 ppm, CI 0.02–0.06, versus 0.14 ppm, CI 0.03–0.56, P = 0.011). Concentrations of zinc,

cadmium, mercury, and lead were similar between age groups ($P > 0.10$). Adult male ospreys during 1975–1982 had higher ($P = 0.01$) mercury concentrations ($\bar{x} = 9.3$ ppm, CI 4.1–21, $n = 3$) than females ($\bar{x} = 1.2$ ppm, CI 0.38–3.6, $n = 5$) collected during the same period. Concentrations of chromium, copper, zinc, cadmium, and lead were similar between sexes ($P > 0.25$). Changes in metal concentrations also were noted between time periods (1971–1973 versus 1975–1982) for birds from the Chesapeake Bay. Adult females had lower ($P = 0.004$) zinc concentrations in the more recent period ($\bar{x} = 27$ ppm, CI 20–38, $n = 5$) than the earlier period ($\bar{x} = 61$ ppm, CI 39–95, $n = 3$). Chromium, copper, arsenic, cadmium, mercury, and lead concentrations were similar between periods for adult females ($P > 0.10$). The more recent collection of immatures and nestlings had higher ($P = 0.011$) concentrations of chromium ($\bar{x} = 0.29$ ppm, CI 0.19–0.42, $n = 4$) than those collected in the earlier period ($\bar{x} = 0.03$ ppm, CI 0.01–0.11, $n = 7$), as well as higher ($P = 0.0001$) concentrations of lead ($\bar{x} = 0.41$ ppm, CI 0.33–0.53, versus $\bar{x} = 0.06$ ppm, CI 0.04–0.10). No differences were found for copper, zinc, arsenic, cadmium, and mercury ($P > 0.05$).

DISCUSSION

Comparative data reported herein must be viewed with caution. First, samples of birds were not collected at random. Birds that died of some causes, such as impact with man-made structures, may have had a greater chance of being recovered than those that died of non-human related causes, such as disease. Collection biases could have changed between time periods. Secondly, cause of death may have been related to fat condition of ospreys that were analyzed. This could have influenced residue concentrations of organochlorines. However, the percent lipid of wet weight in carcass was not significantly different between groups for which organochlorine concentrations were compared. Also, liver samples for this study were analyzed at a different laboratory than those of Wiemeyer et al. (1980) and different analytical techniques were used by the two laboratories for some elements. It is not known if this may have affected the comparisons of element concentrations. Finally, small sample sizes were involved in the comparisons.

Necropsy data

Impact injuries, emaciation, shooting, pneumonia, and trauma were the major causes of death of 33 ospreys found dead or moribund in the eastern United States in 1964–1973 (Wiemeyer et al., 1980). A maximum of 18% of the ospreys in that study may have been shot, whereas only one osprey (4%) in the present study may have been shot, a slight but non-significant (χ^2; $P > 0.10$) decline. The frequency of occurrence of other causes of death between the two osprey studies did not change. The presence of leg injuries or deformities of nestlings from Maryland had been previously reported by Reese (1977). He suspected that dislocated or broken bones of nestlings had resulted from accidents shortly after hatching.

The leg injury of osprey No. 4 which appeared to have resulted from movement of a plastic leg band to an abnormal position emphasizes the need for great care in the selection of band materials, sizes, and proper application methods. Improper banding techniques may result in unnecessary suffering and an increased mortality rate.

Organochlorines

While concentrations of several organochlorines in the carcasses of Chesapeake Bay ospreys declined between 1971 and 1982, changes also occurred in concentrations in fish from Conowingo Dam on the Susquehanna River, a major source of water and chlorinated hydrocarbons to the upper

Chesapeake Bay (Munson et al., 1976). Schmitt et al. (1983) found that DDD concentrations declined significantly, whereas dieldrin concentrations increased in fish (lipid weight basis) from Conowingo Dam during the years 1974–1979; DDE, DDT and total PCB concentrations were unchanged. A significant decline in dieldrin concentrations (lipid weight basis) in fish from this site for the years 1976–1981 was detected later (Schmitt et al., 1985), whereas concentrations of most other organochlorines were stable. DDT concentrations in fish from the Potomac River, another major source of water to the Chesapeake Bay, and the Susquehanna River appeared to decline for the years 1967–1981 (Henderson et al., 1969; Schmitt et al., 1981, 1983, 1985). These changes in concentrations in freshwater fish indicated changes in contaminant input into Chesapeake Bay which eventually was reflected in concentrations of residues in ospreys in the area.

Ospreys from New York, New Jersey, and Maryland had the same general distribution on their common wintering area (Henny and Van Velzen, 1972). However, organochlorine concentrations in osprey eggs from these populations have been markedly different (Wiemeyer et al., 1975, 1978; Spitzer et al., 1978), an indication that organochlorine exposure occurred primarily in the breeding areas. Therefore, organochlorine concentrations in ospreys that were collected during or shortly following the breeding season should be representative primarily of exposure in the breeding area where the ospreys were collected, whereas concentrations in ospreys during periods of migration probably are not representative of the area of collection. For example, concentrations in osprey No. 1 may represent exposure in its presumed breeding area of Massachusetts rather than in South Carolina. Ospreys usually breed near the area where fledged, with males nearer the natal area than females (Spitzer, 1978).

The osprey from South Carolina had clearly elevated concentrations of PCB's in its carcass and brain; it is unlikely that PCB's contributed to its death. The elevated concentration in its brain (220 ppm) was probably the result of mobilization and transport of PCB's resulting from emaciation (Ecobichon and Saschenbrecker, 1969; Bogan and Newton, 1977). Stickel et al. (1984) reported that PCB concentrations in brain associated with death in birds started at 310 ppm, a concentration somewhat higher than that found in the brain of this osprey.

The higher concentrations of organochlorines in adults than in immatures found in this study and that of Wiemeyer et al. (1980), indicated that these contaminants were accumulated over an extended period. The bans and declines in usage of organochlorine insecticides may have resulted in the lower concentrations that were found in both adults and immatures from Chesapeake Bay in the more recent collection period. This may also explain the lower frequency of detection of many organochlorines in this study compared to that of the earlier period (Wiemeyer et al., 1980). Spitzer et al. (1978) reported significant declines in concentrations of DDE and dieldrin, but not PCB's, in osprey eggs from the Connecticut-New York area during 1969–1976.

Metals

The majority of chromium concentrations in osprey livers of this study were between 0.20 to 0.40 ppm whereas those in the previous collection period (Wiemeyer et al., 1980) did not exceed 0.20 ppm. A variety of aquatic birds from noncontaminated areas have had chromium concentrations in their livers similar to those found in this study (Anderlini et al., 1972; Connors et al., 1972; Blus et al., 1977a; White et al., 1979; Howarth et al., 1981; Custer and Mulhern, 1983). Therefore, the chromium concentrations, with the possible exception of the highest value,

were considered normal. The cause for the presence of higher chromium concentrations in immatures and nestlings than adults from Chesapeake Bay, and the presence of higher chromium residues in Chesapeake Bay immatures and nestlings in the recent period was not determined.

The finding of significantly higher concentrations of copper in Chesapeake Bay nestlings and immatures than adults was in agreement with that reported by Wiemeyer et al. (1980). Insufficient numbers of nestlings and immatures have been collected from other populations during the months of June through August to determine if they also have elevated concentrations of copper in their livers. The presence of consistently low concentrations of copper in livers of adults from Chesapeake Bay indicated copper contamination of the area was unlikely. The maximum copper concentration in an immature osprey in this study (55 ppm) was far below that (452 ppm) previously reported by Wiemeyer et al. (1980). The copper concentrations in nestlings and immatures in the present study were not considered toxic based on information presented by Wiemeyer et al. (1980). Custer and Mulhern (1983) found elevated concentrations ($\bar{x} > 85$ ppm dw) of copper in prefledgling black-crowned night-herons (*Nycticorax nycticorax*) from three Atlantic coast colonies; comparative data for adults were not given.

Zinc concentrations in osprey livers in this study (19–150 ppm wet weight) were similar to those reported by Wiemeyer et al. (1980) for 1971–1973. An explanation for the presence of lower zinc concentrations in Chesapeake Bay females in the current sample than that found earlier was not determined. The suggestion of higher concentrations in immatures than adults (Wiemeyer et al., 1980) was not supported by data from the current sample. Zinc concentrations of the magnitude found in these ospreys were considered normal (Wiemeyer et al., 1980).

Arsenic concentrations in livers of ospreys in this study were generally low; even the highest concentration appeared to be well below concentrations associated with mortality (Wiemeyer et al., 1980). The cause of higher concentrations in immatures and nestlings than adults from Chesapeake Bay in this study was not determined. The adult female (No. 15) from Brigantine National Wildlife Refuge, New Jersey, may have been exposed to arsenic on its breeding area which most likely was in New Jersey.

The cadmium concentrations found in this study were similar to those previously found in osprey livers (Wiemeyer et al., 1980), and with the possible exception of the highest value, were similar to concentrations found in livers of uncontaminated birds in other studies (White and Finley, 1978; Di Giulio and Scanlon, 1984; Stoewsand et al., 1984; see also discussion in Wiemeyer et al., 1980). Therefore, these ospreys appeared to have background concentrations of cadmium. A significant difference between age groups in cadmium concentrations like that reported by Wiemeyer et al. (1980) was not found in the present study.

Mercury residues in this study varied widely from 0.28 to 35 ppm. Five ospreys had <1.0 ppm, five had 1.0–2.9 ppm, seven had 3.0–10 ppm, and four had >10 ppm. Holt et al. (1979) found 0.60–18 ppm mercury (median 2.8 ppm) in livers of Norwegian ospreys. Finley et al. (1979) considered mercury residues in soft tissues in excess of 20 ppm as extremely hazardous. Two of the ospreys in this study had concentrations of this magnitude. Three red-tailed hawks (*Buteo jamaicensis*) that died on a mercury contaminated diet had 16.7 to 20 ppm mercury in their livers (Fimreite and Karstad, 1971). However, other birds of prey that died of experimental mercury poisoning had much higher concentrations (≥49 ppm) in their livers (Borg et al., 1970; Koeman et al., 1971). A diagnosis of mercury poisoning cannot be based on tissue concentrations

alone (Finley et al., 1979). At least four ospreys in this study and possibly as many as 11 had elevated mercury concentrations that may indicate exposure to this metal. The ospreys that contained clearly elevated concentrations (>10 ppm) were obtained near or during periods of migration. Therefore, the sources of mercury in the environment were unknown. Higher mercury concentrations were found in adult males than adult female ospreys from Chesapeake Bay. Heinz (1979) found significantly higher mercury residues in male than female mallards (*Anas platyrhynchos*) dosed with methylmercury; lower residues in females may have resulted from mercury elimination through continuous egg laying.

A few ospreys in this study had lead levels in their livers that appeared to have resulted from elevated environmental exposure, although the levels found were well below those associated with lead poisoning in other raptorial species (Pattee et al., 1981; Custer et al., 1984; Pattee, 1984). It is unlikely that ospreys are exposed to metallic lead because they do not feed on prey shot by man; therefore, lead poisoning should not be a problem. Low level chronic exposure to biologically incorporated lead in some areas may be possible. The cause of higher lead residues in livers of Chesapeake Bay nestlings and immatures in this study compared to that of Wiemeyer et al. (1980) was not determined.

Only one of six liver samples analyzed for nickel contained a detectable concentration (0.28 ppm). This was slightly above concentrations found in a number of other species of birds from non-contaminated areas or that were controls in experimental studies (Blus et al., 1977a; Eastin and O'Shea, 1981; Custer and Mulhern, 1983; Rose and Parker, 1983; Wiemeyer et al., 1986), indicating the possibility of minor exposure to environmental contamination. This concentration, however, was below those associated with nickel toxicity in young chickens and mallards (Ling and Leach, 1979; Cain and Pafford, 1981).

Iron concentrations found in osprey livers in this study appeared normal when compared with concentrations found in other species of birds (Howarth et al., 1981; Rose and Parker, 1983; Wiemeyer et al., 1986).

These data have aided only minimally in documenting declines in residues of organochlorine insecticides following their ban in the early 1970's and in providing knowledge on mortality factors. Much more comprehensive collections are required to provide definitive data in these areas. Data from this study have helped in the delineation of background concentrations of trace elements in livers of ospreys, which helps in the determination of areas of elevated exposure. Additional analyses of osprey tissue, with emphasis on trace elements, would be helpful in evaluating the continuing role of environmental contaminants in the health of osprey populations.

ACKNOWLEDGMENTS

We thank L. N. Locke for assistance in necropsies and interpretation of necropsy reports. S. M. Kerr necropsied several birds. L. Sileo necropsied and conducted histopathological studies on several birds. D. E. Docherty conducted virological studies and R. M. Duncan conducted bacteriological studies. Trematodes were identified by B. N. Tuggle. C. M. Bunck provided advice on statistical analyses and interpretation. The paper was improved by the reviews of M. L. Gay, C. E. Grue, and G. H. Heinz. M. G. Holmes typed the manuscript.

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Received for publication 24 July 1986.