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ORGANOCHLORINE POISONING OF RING-BILLED GULLS IN SOUTHERN ONTARIO[1]

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Abstract: Clinical, necropsy, bacteriologic, parasitologic, histopathologic, toxicologic and animal inoculation studies suggest that organochlorine (PCB, dieldrin and DDE) poisoning was an important factor in causing deaths of free-flying ring-billed gulls (*Larus delawarensis*) in southern Ontario in 1969 and 1973. The brains of gulls dying with clinical signs of neurologic involvement, and dead gulls with no other apparent cause of death, contained organochlorine residues of significantly greater levels than those found in healthy gulls shot for comparison.

INTRODUCTION

Organochlorine residues in tissues from wildlife carcasses present a difficult diagnostic problem for wildlife pathologists. Residue data are difficult to interpret for several reasons: many different organochlorines are detected in a wide variety of wildlife species, there is considerable variation in residue levels in different tissues associated with death, and experimentally determined lethal residue levels are not available for most wild species. Experimental studies of organochlorine poisoning in captive birds suggest several generalizations which aid in interpretation of residue data from free-flying birds. Residue levels of DDT, dieldrin, and PCB in brain correlate well with death and are of greater value to the diagnostician than levels in other tissues.[6,8,35,36,37,39] Preliminary evidence suggests that the toxic effects of some of the organochlorine residues may be additive, at least when two organochlorines are fed simultaneously or sequentially.[14,20,23] Mobilization of fat from systemic adipose depots results in release and systemic redistribution of the residues.[5,24,34,40] Individuals with higher susceptibility die with lower residue levels than resistant individuals.[4,36] In addition to being directly toxic, experimental evidence suggests that systemic PCB's and dieldrin residues may have a deleterious effect on resistance to infectious disease.[12,13]

Fatal dieldrin residue levels in brain tissue of experimentally poisoned birds are reported for coturnix (*Coturnix coturnix*),[35,39] pigeons (*Columba livia*),[35] pheasants (*Phasianus colchicus*),[22] tree ducks (*Dendrocygna bicolor*),[10] mallards (*Anas platyrhynchos*),[13] redwing blackbirds (*Agelaius phoeniceus*),[4] and song thrushes (*Turdus ericetorum*).[18] Dieldrin has been suspected or clearly implicated in the deaths of a wide variety of free-flying birds: osprey (*Pandion haliaetus*),[43] bald eagles (*Haliaetus leucocephalus*),[2,25,32] buzzards (*Buteo lagopus*),[30] peregrines (*Falco peregrinus*),[33] lanners (*Falco biarmicus*),[17] egrets (*Casmerodius*

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albus),[9] heron (*Ardea herodias*),[3] various waterfowl and shorebirds,[10] and various songbirds.[19,33]

Fatal DDE residue levels in brain tissue of experimentally poisoned birds are reported for kestrels (*Falco sparverius*),[29] pigeons,[1] and cowbirds (*Molothrus ater*).[38] High DDE residues were suggested as a contributing cause of death in a bald eagle[2] and a gannet (*Morus bassanus*).[27] We are not aware of published reports of uncomplicated DDE-caused deaths in wild birds.

Brain PCB levels associated with death in birds fed experimentally contaminated diets are reported for chickens (*Gallus domesticus*),[28,41] Bengalese finches (*Lonchura striata*),[31] coturnix,[19] pheasants,[5,6] ring doves (*Streptopelia risoria*)[21] and various passerine species.[31,37] Tremors, emaciation, black gastro-intestinal contents and hepatic necrosis and hemorrhage were reported for birds experimentally poisoned with PCB's.[7,28,37,41] We are not aware of reports clearly implicating PCB's in the deaths of free-flying birds. The bald eagle mentioned above in connection with DDE poisoning also had very high brain levels of PCB's.[2] PCB's were considered as a possible contributing cause of the extensive mortality of seabirds in the Irish Sea in 1969, but later workers considered this was not likely.[26] These last two cases show the quandry of the wildlife diagnostician. DDE, dieldrin and PCB's are toxic, are ubiquitous environmental contaminants, and are regularly present in wildlife found dead in the field, yet there are few guidelines for assessment of their pathological significance.

We present a summary of the results of post-mortem examination of 138 ring-billed gulls (including toxicologic analyses of 82) submitted for necropsy. Organochlorine poisoning is implicated by comparing brain residue levels found in gulls with known causes of death to levels found in gulls which died of undetermined causes. An attempt is made to evaluate the significance of the total organochlorine load in individual gulls by means of a computed organochlorine index.

MATERIALS AND METHODS

Dead or dying gulls found throughout southern Ontario were submitted during late July, August and early September of 1969 and 1973. Sex, age and body weight were recorded in most cases. Post-mortem decomposition precluded reliable examination in some cases. In 55 cases, selected tissues were prepared for culture on blood agar and MacConkey's agar and were incubated aerobically at 37.5 C for 24 h. Selected tissue specimens from 52 gulls were fixed in 10% buffered formalin, embedded in paraffin, sectioned at 5 μm and stained by the hematoxylin and eosin method for microscopic examination. Laboratory mice were inoculated with suspensions prepared from heart blood and liver of 36 gulls to test for botulinus toxin. Chicken embryos and 1 day old chicks were inoculated with pooled tissue suspensions from 19 gulls. In 26 gulls, parasites were identified and their significance assessed. A variety of tissues from 128 gulls were analyzed for organochlorines using chromatographic methods described by Frank *et al.*[11] Brain residue data are presented for 82 gulls. Necropsy records were placed in one of three groups according to the cause of death: infectious diseases, gunshot, or cause of death not determined. Differences in residue levels of dieldrin, DDE, and PCB's between the three groups in each year and between years were analyzed for their significance using a t-test for comparison of means of samples with unpaired observations and unequal variances. Residues were compared on a ppm wet weight basis, and all data are presented on this basis.

To evaluate the total combined load of all three organochlorine residues, an "organochlorine index" was computed for each gull. The literature suggests that wet weight brain residues of 5 ppm of dieldrin, 150 ppm of DDE, and 300 ppm of PCB are potentially toxic levels associated with death in some avian species.[1,5,6,7,21,27,28,29,31,34,38,41] The brain dieldrin residue for each gull was expressed as a ratio of 5, the brain DDE residue as a ratio of 150, and the brain PCB residue as a ratio of 300. The sum of

these three ratios was termed the brain "organochlorine index." An organochlorine index of one or greater suggests that the toxic effects of combined brain residues of dieldrin, DDE and PCB were potentially toxic levels.

RESULTS

Ninety-three gulls were examined in 1969 and 45 in 1973. Few ring-billed gulls were submitted in the intervening years. Eight of the gulls of various sexes and ages had extensive lesions indicating aspergillosis, salmonellosis, peritonitis, or amyloidosis as the cause of death. Another 21 healthy gulls were shot.

The cause of death was not apparent for the remaining 109 gulls (this number included a few decomposed or partial carcasses not suitable for complete necropsy). All post-fledgling sex and age categories were represented. Moribund gulls were weak, unable to stand, and had spasmodic muscle tremors when captured. Nearly all of the 109 carcasses were in poor body condition with depleted fat storage depots. The average body weight was 376 gm in 1969 and 332 gm in 1973. Gulls shot in 1969 and 1973 were significantly heavier than gulls dying of undetermined causes ($t=4.12$, $P<.001$ and $t=5.58$, $P<.01$ respectively) or gulls dying of infectious diseases (Tables 1 and 2). Proventriculi and gizzards generally were empty, with a thin layer of black muco-serous material on the mucosae. Bacteriologic cultures of liver, spleen, or intestine were sterile or yielded *Escherichia coli*, but no other potential pathogens. Suspensions of liver and blood were not toxic to mice. Results of chick embryo and chick inoculations were inconclusive. *Capillaria* sp., *Cosmocephalus* sp., *Tetrameres* sp., *Echinostoma* sp., *Diplostomum* sp., and unidentified cestodes and gizzard worms were present in moderate numbers in the digestive tract of many gulls. Post-mortem and freeze damage precluded reliable histopathologic examinations in many cases. Microscopic liver lesions noted consistently in fresh carcasses were non-zonal focal areas of fatty degeneration, necrosis, hemorrhage, hepatocellular degeneration and periportal aggregates of lymphocytes. Other microscopic lesions were noted, e.g. amyloid deposits, but were neither consistently found nor extensive and were not considered a cause of death. There was no microscopic evidence of splenic lymphoid atrophy.

TABLE 1. A summary of post-mortem findings and brain organochlorine levels for ring-billed gulls examined in southern Ontario in 1969.

Gull #	Cause of Death[1]	Body Weight (g)	DDE ppm	Dieldrin ppm	PCB ppm	Organochlorine Index[2]
1	ND	ND	71	5.9	320	2.72
2	ND	ND	68	2.0	705	3.20
3	ND	ND	2	4.1	510	2.53
4	ND	ND	77	4.5	455	2.93
5	ND	ND	67	7.1	595	3.85
6	ND	ND	52	4.3	535	2.99
7	ND	ND	63	4.1	770	3.81
8	ND	ND	106	10.0	1110	6.41
9	ND	ND	53	2.9	390	2.23
10	ND	ND	87	5.6	425	3.12

TABLE 1—continued

Gull #	Cause of Death[1]	Body Weight (g)	DDE ppm	Dieldrin ppm	PCB ppm	Organochlorine Index[2]
11	ND	ND	64	5.3	340	2.62
12	ND	ND	95	4.5	235	2.32
13	ND	ND	70	4.9	420	2.85
14	ND	ND	59	2.9	225	1.72
15	ND	289	101	2.1	330	2.19
16	ND	483	75	1.29	269	1.65
17	ND	246	83	5.31	232	2.39
18	ND	420	96	1.90	234	1.80
19	ND	380	159	0	344	2.21
20	ND	319	3.9	6.00	940	6.94
21	ND	405	77	5.10	187	2.16
22	ND	424	55	Trace	355	1.55
23	ND	322	108	2.53	428	2.65
24	ND	391	212	1.65	691	4.05
25	ND	319	60	3.85	186	1.79
26	ND	322	296	17.70	950	8.68
27	ND	344	75	0	289	1.46
28	ND	494	101	4.11	356	2.68
29	ND	428	232	3.54	716	4.64
30	D	293	8.9	0.28	25	.20
31	D	381	0.4	0.05	2	.02
32	D	343	14.5	0.13	68	.35
33	S	ND	5.5	0.06	30	.15
34	S	ND	0.6	0.03	3	.02
35	S	423	0.3	0.04	1	.01
36	S	426	7.7	0.77	48	.36
37	S	518	0.7	0.03	2	.02
38	S	462	0.3	0.02	1	.01
39	S	582	0.6	0.07	1	.02
40	S	424	0.8	0.05	3	.02
41	S	493	1.2	0.19	3	.06
42	S	474	1.0	0.08	4	.04
43	S	510	1.0	0.09	4	.04
44	S	456	0.5	0.07	2	.02
45	S	439	0.6	0.08	6	.04
46	S	478	1.3	0.10	5	.05
47	S	440	0.7	0.16	2	.04
48	S	434	0.5	0.12	2	.03
49	S	410	3.4	0.42	10	.14
50	S	448	2.9	0.23	9	.10

[1]ND = not determined; D = disease; S = gunshot

[2]Index = DDE/150 + Dieldrin/5 + PCB/300

TABLE 2. A summary of post-mortem findings and brain organochlorine levels for ring-billed gulls examined in southern Cntario in 1973.

Gull #	Cause of Death[1]	Body Weight (g)	DDE ppm	Dieldrin ppm	PCB ppm	Organochlorine Index[2]
1	ND	350	ND	ND	434	1.45
2	ND	340	ND	ND	363	1.21
3	ND	350	ND	ND	483	1.61
4	ND	330	ND	ND	448	1.49
5	ND	320	17.5	0	320	1.18
6	ND	330	26.7	0	370	1.41
7	ND	ND	47.5	.88	360	1.69
8	ND	ND	5.76	1.27	291	1.26
9	ND	ND	37.0	3.18	290	1.85
10	ND	530	27.6	.69	260	1.19
11	ND	270	24.2	.66	460	1.83
12	ND	380	31.3	1.16	310	1.47
13	ND	290	22.2	.19	550	2.02
14	ND	320	26.6	.71	350	1.49
15	ND	400	30.1	.83	250	1.20
16	ND	290	28.1	.59	140	.77
17	ND	220	13.8	0	190	.73
18	ND	310	16.8	0	190	.75
19	ND	330	11.0	0	200	.74
20	ND	370	24.0	.69	160	.83
21	ND	320	10.6	0	250	.90
22	ND	ND	28.2	0	270	1.09
23	ND	280	28.0	.64	250	1.15
24	ND	340	15.1	0	290	1.07
25	ND	350	ND	ND	295	.98
26	D	270	18.5	0	430	1.56
27	D	235	2.2	.06	100	.36
28	D	430	.10	.004	1	.00
29	D	410	.01	Trace	<1	.00
30	S	470	.48	.03	7	.03
31	S	500	.17	.02	3	.02
32	S	470	.10	.01	0	.00

[1]ND = not determined; D = disease; S = gunshot

[2]Index = DDE/150 + Dieldrin/5 + PCB/300

On the average, in both 1969 and 1973, there were very significantly higher brain residues of DDE ($t=5.79$, $P<.001$ and $t=10.57$, $P<.001$ respectively) and PCB's ($t=9.999$, $P<.001$ and $t=9.56$, $P<.001$ respectively) in birds dying of undetermined causes than in healthy birds purposely killed (Table 3). In 1969, dieldrin brain residues in gulls dying of undetermined causes were very significantly higher ($t=7.29$, $P<.001$) than in gulls purposely killed (Table 3). The data suggest that the same was true in 1973, but insufficient numbers of gulls were analyzed for dieldrin to afford a statistical comparison. Levels of DDE and PCB's in gulls for which aspergillosis and salmonellosis were considered the cause of death were variable and usually intermediate to levels observed in gulls dying of undetermined causes and gulls which were killed (Table 3).

The organochlorine indices between groups of gulls and years have the same relationship and magnitude of difference as the residue levels (Table 3). For gulls shot in 1969 the average organochlorine index was .06 while gulls with undetermined cause of death had an average index of 3.22. In 1973, the same relationship was present for gulls shot, the average index was .02, while for gulls with undetermined cause of death the index was 1.25.

TABLE 3. Average organochlorine residues and computed organochlorine indices for gulls examined in 1969 and 1973.

Cause of Death	1969 ppm/N DDE	Dieldrin	PCB	Average OC Index*	1973 ppm/N DDE	Dieldrin	PCB	Average OC Index*
Not apparent	105 / 29	4.73 / 27	467 / 29	3.22 / 29	24 / 20	.96 / 11	311 / 25	1.25 / 25
Diseased	8 / 3	0.15 / 3	32 / 3	0.19 / 3	6 / 4	.02 / 3	133 / 4	0.48 / 4
Shot	2 / 18	0.14 / 18	8 / 18	0.06 / 18	1 / 3	.02 / 3	5 / 3	0.02 / 3

*An organochlorine index greater than one suggests a potentially toxic level.

Many individual gulls had brain residues of a single organochlorine well above the potentially toxic level. But there are several cases where the level of one or more individual residues is on the borderline, e.g. gull #9 in 1973 (Table 2). The computed organochlorine index aids in evaluating the total organochlorine load in such cases. The index for gull #9 was 1.85, well over the potentially toxic level of 1.00.

Low levels of TDE were detected in brain tissue of seven gulls; DDE was the only other DDT metabolite detected. Low levels of HCB and heptachlor epoxide residues were detected in a few gulls.

DISCUSSION

Organochlorines were reported in fauna of the Great Lakes and in eggs of a variety of birds in southern Ontario, so residues in ring-billed gulls were not unexpected.[11,15] The parasites noted were not particularly pathogenic. They may have contributed to the moribund state of some gulls, but it is unlikely they were the cause of death.

Levels of DDE, dieldrin, and PCB averaged 30 to 90 times higher in brain tissue of moribund and dead gulls than in healthy gulls. A diagnosis of organochlorine poisoning as the cause of death of most of the gulls was suggested by

the clinical, necropsy, histologic and toxologic findings and the absence of any other apparent cause of death.

There was an inverse relationship between body weight and brain organochlorine indices in Tables 1 and 2. Shot gulls were in good flesh and had low brain indices while gulls with undetermined cause of death were usually emaciated and had high brain indices. The release of residues from adipose tissue during mobilization of fat reserves must account for some (or much) of the brain residues. Submission of specimens suggests that most mortality occurred in late summer and early autumn. We do not know what environmental stresses the gulls were subjected to during that period. Events such as the postnuptial or post-juvenal molt may have contributed to the emaciation. Normal utilization of stored body fat possibly influenced the timing of the mortality. Alternatively, there are numerous reports of anorexia and emaciation caused by experimental dietary organochlorine poisoning.[1,4,6,14,10,21,31,35] Our data suggest correlation, possibly a cause-effect relationship between emaciation and a high brain organochlorine index, but it is not possible to determine which is cause and which is effect from post-mortem data.

Although experimentally fed PCB and dieldrin residues decrease resistance to infectious disease in some species, there was no obvious association between the brain organochlorine index and the presence of infectious disease in these gulls.

Toxicologic data from the early part of 1969 is not presented in this report because the then undetected PCB's biased other residue determinations. Even in late 1969, when the PCB component was recognized, fractionation techniques were partial and the dieldrin and DDE components may have contained some PCB contaminants. This may explain why dieldrin and DDE levels and the organochlorine indices are higher in 1969 than in 1973; PCB contamination of the dieldrin and DDE component biased the levels and indices.

Analyses for polychlorinated dibenzofurans or dioxins were not done. Vos *et al.*[42] suggest that these residues were contaminants of some commercially produced PCB's and that hepatic lesions produced by experimental feeding of PCB's were due to the contaminants.[42] This may explain the presence of focal hepatitis in these gulls.

In conclusion, it seemed likely that many ring-billed gulls were carrying fatal burdens of organochlorine residues. PCB brain levels were very high even though sale and distribution of this chemical in North America was limited to "closed circuit" uses 2 years previously.[26] The brain organochlorine index seemed a simple but useful method to quantify the pathological significance of the total organochlorine load in an individual gull carcass. These data suggest the need for further studies of the cyclical mortality, the source of organochlorine residues, and the relationship between behaviour, cachexia and brain organochlorine residues in free-flying ring-billed gulls in southern Ontario.

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