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ORGANOCHLORINE POISONING OF HERONS

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Over a period of years interested individuals have submitted many dead or moribund herons of various species to our laboratory to learn whether the birds had been affected by diseases or organochlorine poisoning. Residue concentrations in carcasses of birds and mammals are considered the best measure of sublethal exposure, whereas residues in brains are best to use for diagnosing death by most organochlorine chemicals (see Heinz et al. 1979 for review).

Residue concentrations in brains that are diagnostic of organochlorine poisoning for birds have been determined in the laboratory for DDT and its metabolites (L. F. Stickel et al. 1966; W. H. Stickel et al. 1970; Stickel and Stickel 1970; W. H. Stickel, pers. comm.); dieldrin (W. H. Stickel et al. 1969; W. H. Stickel, pers. comm.); heptachlor, chlordane, and oxychlordane (L. F. Stickel et al. 1979; W. H. Stickel, pers. comm.); endrin (W. H. Stickel et al. 1979); Aroclor 1254 (W. H. Stickel 1975; W. H. Stickel, pers. comm.); and mirex (W. H. Stickel et al. 1973). This subject is discussed in further detail by Heinz et al. (1979).

The purpose of the present paper is to document the occurrence and concentration of organochlorine residues in the brains of herons from various areas in the United States. By comparing these residue concentrations with laboratory-determined diagnostic lethal levels, we conclude that some herons were killed by organochlorine poisoning; others were at least seriously endangered by the residues they carried.

Complete results of carcass analyses for these and other herons, as well as further details on residues in brains, will be reported elsewhere (H. M. Ohlendorf, D. M. Swineford, and L. N. Locke, unpublished manuscript). Overall, we analyzed carcasses or brains of more than 70 herons found dead or moribund and 36 others taken in planned collections. Residue levels in carcasses of many herons were not high enough to warrant analysis of brains. In the present paper we compare carcass and brain residues of dieldrin in 23 herons of which both carcass and brain were analyzed.

METHODS AND MATERIALS

We analyzed brains of 49 herons found dead or moribund. They represented 5 species and were collected in 12 States (Table 1). Some specimens were delivered or shipped to our laboratories while still fresh.

Table 1. Distribution of herons (by State and species) found dead or moribund for which brains were analyzed.

REGION	State	Species	Number analyzed
NORTHEASTERN			
	Maine	Great Blue Heron[a]	6
MID-ATLANTIC			
	Maryland	Great Blue Heron	3
	Virginia	Great Blue Heron	3
SOUTHEASTERN			
	Florida	Great Blue Heron	3
	North Carolina	Great Blue Heron	1
GREAT LAKES			
	Illinois	Great Blue Heron	1
	Minnesota	Great Blue Heron	14
		Great Egret[b]	4
	Wisconsin	Great Blue Heron	3
		Black-crowned Night Heron[c]	1
CENTRAL AND WESTERN			
	California	Great Egret	1
	Nebraska	Cattle Egret[d]	1
	Nevada	Black-crowned Night Heron	3
	New Mexico	Cattle Egret	3
		Snowy Egret[e]	1
		Black-crowned Night Heron	1
		TOTAL	49

[a] Ardea herodias
[b] Casmerodius albus
[c] Nycticorax nycticorax
[d] Bubulcus ibis
[e] Egretta thula

Most, however, were frozen, packed in dry ice, and shipped by air express to the laboratory. There they were stored at -25° C until processed for analysis.

Necropsies were performed on many of these herons to determine causes of death. Others were dissected without complete necropsy or histological examination. The brain was removed, placed in an acetone- and hexane-rinsed glass jar, and frozen until analyzed.

Specimens were analyzed for residues of DDE, DDD, DDT, dieldrin, heptachlor epoxide, oxychlordane, cis-chlordane, trans-nonachlor, cis-nonachlor, endrin, toxaphene, HCB, mirex, and PCBs. Samples were homogenized, extracted in a Soxhlet, and cleaned up with a Florisil column as previously described by Cromartie et al. (1975). Pesticide and PCBs were separated into four fractions on a Silicar column as follows:

Fraction 1 - 80 ml petroleum ether, which elutes HCB and mirex;
Fraction 2 - 320 ml petroleum ether, which elutes PCBs and DDE;
Fraction 3 - 275 ml of 15% methylene chloride in hexane, which elutes the remaining organochlorine compounds except endrin and dieldrin;
Fraction 4 - 200 ml of 1% acetonitrile, 19% hexane in methylene chloride, which elutes endrin and dieldrin.

The individual fractions were analyzed by a gas-liquid chromatograph equipped with an electron-capture detector, automatic sampler, digital processor, and a 1.5% OV-17/1.95% QF-1 column at 190° C. Toxaphene was quantitated as described by Prouty et al. (1977).

Residues in 10% of the samples were confirmed with an LKB 9000 or Finnigan 4000 series gas-liquid chromatograph/mass spectrometer. Operating parameters for the LKB are listed in Cromartie et al. (1975). The operating conditions for the Finnigan were: flow rate of 30 ml/min helium; 1.5% OV-17/1.95% QF-1 column, temperature-programmed at 2° C/min from 140 to 225° C; separator 235° C; and ion source 300° C.

Residue levels were not corrected for recovery, which ranged from 83 to 104%. Most tissues were analyzed at a lower limit of detection of 0.1 ppm for pesticides and 0.5 ppm for PCBs. Residue concentrations are expressed on wet-weight basis, unless otherwise indicated.

Soxhlet extracts were concentrated to dryness for lipid determinations.

RESULTS

The residues found most frequently were PCBs (44 of 49 brains analyzed). DDE was second (in 42 brains), and dieldrin was third (24). Endrin (11), mirex (8), and HCB (5) were found least often. The frequency of residue occurrence in brains was similar to that in the carcasses (H. M. Ohlendorf, D. M. Swineford, and L. N. Locke, unpublished manuscript).

Residue concentrations were usually higher in adult herons than in immatures. All birds with hazardous or lethal concentrations in their brains were adults (Table 2). Most of the herons that apparently died of organochlorine poisoning were Great Blue Herons (Ardea herodias). Dieldrin was the chemical most often responsible for death. Residue concentrations that were considered to be lethal or hazardous in the brains of selected herons are presented in Table 2.

Dieldrin residues in the brain (wet-weight basis) were a function of the concentrations in the carcass (lipid-weight basis) of 23 herons for which both the brain and carcass were analyzed:

$$\log \text{brain concentration} = \log a + b\ (\log \text{carcass concentration}).$$

Common logarithms were used in the calculations; R^2 was 0.89 (Fig. 1). Substituting the Y intercept (a) and slope of the regression (b) values

Table 2. Adult herons found dead or moribund with lethal or hazardous organochlorine residue concentrations in their brains. (For each bird, only the chemicals of principal concern are shown.)

Collection locality[a]	Date	Species[b]	Sex	Carcass lipid (%)	Residues in brains (ppm, wet weight)
D I E L D R I N					
Florida; Monroe, Everglades NP	28 Feb. 1973	GBH	M	0.8	6.6[c]
Illinois; McHenry, Harvard	17 June 1977	GBH	M	1.1	12.0
Minnesota; Benton, St. Cloud	14 May 1973	GBH	F	0.5	14.0
Minnesota; Pope, Lake Johanna	22 June 1975	GBH	M	0.9	9.2
Minnesota; Stearns, Cold Springs	9 May 1972	GBH	M	0.3	14.0
Wisconsin; Dane, Belleville	15 May 1979	GBH	M	ND[d]	6.0
Nebraska; Harlan, Alma	8 May 1978	CE	F	0.5	5.6
Virginia; Fairfax, Mason Neck NWR	31 May 1974	GBH	F	0.7	5.1[e]
Virginia; James City, Jamestown	1970	GBH	F	1.6	8.9[e]
Virginia; James City, Williamsburg	12 Apr. 1972	GBH	M	0.2	5.8[f]
E N D R I N					
Minnesota; Stearns, Sartell	16 Apr. 1972	GBH	M	0.3	0.86
Wisconsin; Kenosha, Brighton	1 May 1978	GBH	M	0.3	0.60

Table 2. (Continued).

Collection locality[a]	Date	Species[b]	Sex	Carcass lipid (%)	Residues in brains (ppm, wet weight)
D D T + M E T A B O L I T E S					
Nevada; Elko, Ruby Lake NWR	18 July 1975	BCNH	?	0.6	230 DDE 1.1 DDT
North Carolina; Hyde, Mattamuskeet NWR	4 Feb. 1976	GBH	?	ND	62 DDE 21 DDD 20 DDT
P C B s					
Wisconsin; Sheboygan, Sheboygan Falls	18 Dec. 1976	GBH	M	ND	220

[a] State; county, and nearest city or other identifying location.
[b] GBH = Great Blue Heron; CE = Cattle Egret; BCNH = Black-crowned Night Heron.
[c] Bird had apparently been wounded by shooting, and starvation may have contributed to its death.
[d] ND = not determined.
[e] Bird was also infected by nematodes (Eustrongylides sp.) that caused verminous peritonitis.
[f] Brain residue concentration estimated on the basis of dieldrin concentration in carcass (see text). This bird also had verminous peritonitis.

in the equation the relationship was:

log brain concentration = -1.2525 + 0.8549 (log carcass concentration).

This equation was used to estimate dieldrin concentrations in the brains of herons for which only the carcass was analyzed, but the brain level in only the one heron from Virginia (Table 2) was in the known lethal range.

DISCUSSION AND CONCLUSIONS

When birds are found dead or moribund in the field, it is sometimes possible to establish the cause of death by necropsy. Diseases, including verminous peritonitis, caused the deaths of many of the herons we examined (H. M. Ohlendorf, D. M. Swineford, and L. N. Locke, unpublished manuscript). For others, however, there was no apparent cause of death; it was these birds in particular that we analyzed to determine organochlorine residue levels in the brain.

Four Great Blue Herons from Illinois and Minnesota died with more than 9 ppm dieldrin in their brains, the level that indicates probable death by dieldrin poisoning (W. H. Stickel, pers. comm.). A Great Blue Heron from Wisconsin and a Cattle Egret (Bubulcus ibis) from Nebraska died with dieldrin residues at levels that caused mortality (5-9 ppm) in some individuals in the laboratory studies, but at which others survived.

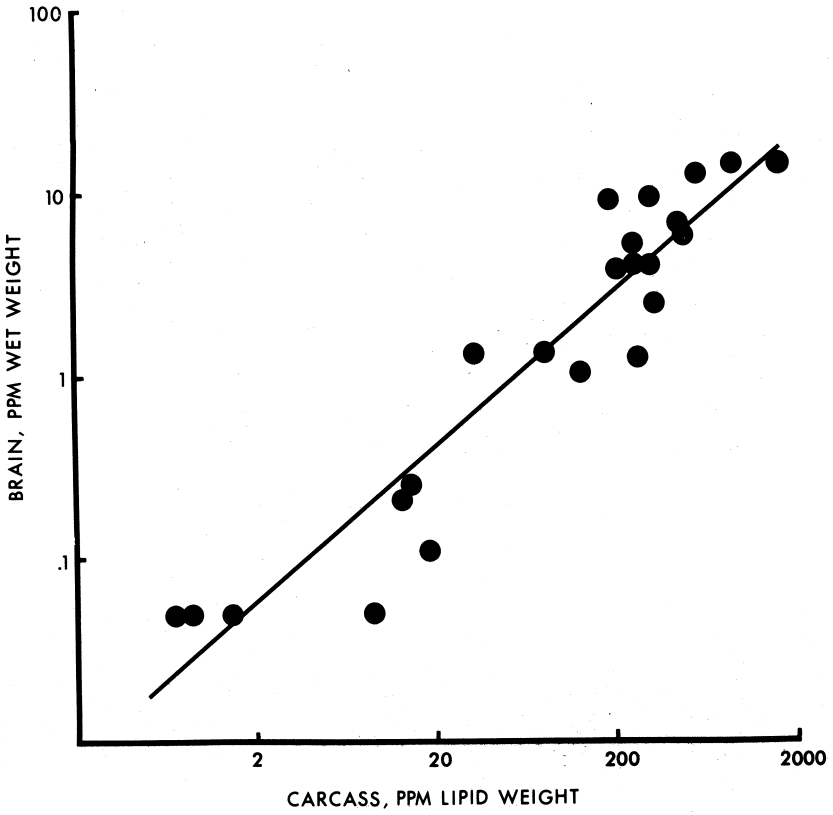

Figure 1. Regression of dieldrin concentration (ppm, wet weight) in the brains of 23 herons on that in the carcass (ppm, lipid weight). R^2 is 0.89; the regression equation is:
log brain concentration = -1.2525 + 0.8549 (log carcass concentration).

However, because complete necropsy of both these birds did not reveal other probable causes of death, it seems likely that they died of dieldrin poisoning.

Four other Great Blue Herons had levels of dieldrin that were clearly hazardous. However, dieldrin may not have been the sole cause of death in these birds and elevated brain concentrations may have been secondary to emaciation caused by other factors. The heron that died in Florida apparently

had been shot, but the injury was not immediately fatal. The bird may have been unable to feed because of a broken mandible and therefore starved. Three herons from Virginia had levels of dieldrin that may have been lethal, but they also had verminous peritonitis as a result of being infected by nematodes (Eustrongylides sp.). Although many herons apparently survive infections by nematodes of the type that caused peritonitis in these individuals (Wiese et al. 1977; J. L. Crites, pers. comm), others may die if certain organs are reached by the nematodes, which burrow through the ventriculus and proventriculus into the body cavity (Locke 1961).

Carcass lipid levels were low (usually less than 1%) in all of the herons that had high dieldrin levels in their brains; much of the dieldrin that had been stored in carcass lipids had been mobilized. Therefore, residue concentrations in the brain had become elevated, probably with lethal effects in some birds.

Experimental Brown-headed Cowbirds (Molothrus ater) that averaged 4.4 ppm dieldrin in their brains (some were as low as 1.5 ppm) ceased feeding while still in good flesh and fat (Heinz and Johnson, in press). After birds stopped feeding, they seldom ate again; that is, the starvation process was generally irreversible. When a bird stopped eating, it kept mobilizing additional dieldrin from fat. Some of the mobilized dieldrin deposited in the brain, further sickening the bird and continuing to keep it from feeding. Therefore, levels of dieldrin that are clearly sublethal may nevertheless present a serious hazard to birds. The same process is believed to occur with all truly persistent organochlorine insecticides, although the residue levels vary widely with the toxicity of the chemical.

Birds seldom survive with more than 0.8 ppm endrin in the brain (W. H. Stickel et al. 1979), so it appears likely that one Great Blue Heron from Minnesota died of endrin poisoning. The heron from Wisconsin with 0.60 ppm endrin falls into the zone of overlap (0.6 - 0.8 ppm) at which birds may die or survive.

The Black-crowned Night Heron (Nycticorax nycticorax) from Nevada had DDE residues in its brain that were clearly hazardous, but perhaps not lethal. The lower limit of the lethal range is about 250 ppm (W. H. Stickel, pers. comm.). However, the Great Blue Heron from North Carolina almost certainly died of DDT poisoning; no other cause of death was found at necropsy or in other subsequent studies of the specimen. The combined DDT and DDD residues exceeded 30 ppm, the practical separation point between dead birds and survivors in laboratory studies (L. F. Stickel et al. 1966; Stickel and Stickel 1970; W. H. Stickel, pers. comm.).

Organochlorine residues in herons have been reported by others. The death of a Great Blue Heron in South Dakota in 1975 was attributed to DDE poisoning (Call et al. 1976); this bird had 246 ppm DDE in its brain. Organochlorine residues also have been reported at relatively high levels in the livers of Gray Herons (Ardea cinerea) in Britain (Prestt 1970). However, residues in carcasses and brains were not reported, so direct comparisons with our findings are not possible. Four Great Egrets (Casmerodius albus) found dead or moribund in a California heronry had hazardous or lethal levels of dieldrin (4.4 - 6.8 ppm) in their brains (Faber et al. 1972). Faber et al. (1972) suggested death of those birds was caused by dieldrin poisoning because residue levels were elevated and some had convulsions before death.

The concentration of PCB residues (Aroclor 1254) in a Great Blue Heron from Wisconsin was high enough to be considered hazardous, though not lethal.

Most of the herons we analyzed were picked up during the birds' breeding

season. This peak may reflect increased mortality during that period of seasonal weight loss (Gish and Chura 1970; Van Velzen et al. 1972; W. H. Stickel, pers. comm.) or more persons may have been studying herons then and dead birds were more likely to be found in the spring and submitted for analysis.

For each heron that is found and analyzed, scores probably die; our specimens must represent a minor fraction of the heron mortality that occurred during the period of our study. We made no organized searches and obtained specimens only when other individuals found dead herons by chance. Therefore, it seems especially significant that we found lethal or hazardous organochlorine residue concentrations in more than 20% of the specimens analyzed.

Dieldrin was banned for use in the United States in 1975; DDT was banned in 1972 (Anonymous 1977). It is important to note that each of these chemicals caused mortality of herons years later.

SUMMARY

Herons found dead or moribund in the field since 1966 were analyzed for residues of organochlorine chemicals. Concentrations in brains were compared with those known to be diagnostic of poisoning in other species.

Most of the herons that apparently died of organochlorine poisoning were Great Blue Herons. Dieldrin was the chemical most often responsible for death. Three Great Blue Herons from Minnesota (9.2, 14.0, and 14.0 ppm, wet weight) and another from Illinois (12.0 ppm) died with enough dieldrin in their brains to account for death. A Great Blue Heron from Wisconsin (6.0 ppm) and a Cattle Egret from Nebraska (5.6 ppm) had marginal levels of dieldrin, but necropsy revealed no other cause of death. A Great Blue Heron from Florida and three others from Virginia had hazardous dieldrin levels (5.1-8.9 ppm) but they also had other health problems.

Dieldrin residues in the brain (wet-weight basis) were a function of the concentration in the carcass (lipid-weight basis) of herons for which both the brain and carcass were analyzed.

One Great Blue Heron from Minnesota probably died of endrin poisoning (0.86 ppm), and another from Wisconsin (0.60 ppm) may have died from endrin poisoning.

A Great Blue Heron from North Carolina apparently died as a result of DDT poisoning (20 ppm DDT, 21 ppm DDD, and 62 ppm DDE). A Black-crowned Night Heron from Nevada (230 ppm DDE plus 1.1 ppm DDT) may have died of DDE poisoning; it was at least seriously endangered.

Our observations indicate that herons died of DDT and dieldrin poisoning years after the chemicals were banned in the United States, and also that more than 20% of the herons analyzed had lethal or hazardous levels of organochlorines in their brains.

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