DDE Poisoning in Wild Great Blue Heron

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The impact of DDE on eggshell thinning and reproductive failures in many raptorial and piscivorous avian species has been well documented. However, little information is available regarding mortality in adult wild birds due to DDE poisoning. This case report is of special interest, as the poisoning occurred approximately three years after the ban on the commercial use of the parent compound, DDT, was imposed in the United States.

MATERIALS AND METHODS

An adult great blue heron (Ardea herodias) in moribund condition was collected in the field on May 12, 1975, in Marshall county of northeastern South Dakota. It was presented for examination on the following day. The bird was in tremors, and died a few hours after being received.

The heron was necropsied, and the liver and brain were analyzed for organochlorine insecticide and polychlorinated biphenyl (PCB) residues by gas chromatography (GREICHUS et al., 1973). Thin-layer chromatography and mass spectrometry were used for confirmation (GREICHUS et al., 1973; GREICHUS et al., 1974). The liver was screened for lead, mercury, arsenic, antimony, bismuth, silver, sulfur, selenium, and tellurium (BAMFORD, 1951).

RESULTS AND DISCUSSION

Gross examination revealed that the heron was dehydrated and its gastrointestinal tract was empty. The bird was emaciated with little body fat. One large ascarid was present in the gizzard. No lesions were observed in sections of the brain, heart, gizzard, kidney, intestine or liver when examined by light microscopy.

Residue levels for organochlorine insecticides and PCB's are presented in Table 1. No abnormal

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levels of any of the elements screened for were detected.

TABLE 1

Organochlorine residues in great blue heron (ppm, wet weight basis)

Compound	Brain	Liver
p,p'-DDE	246.	570.
p,p'-DDD	0.98	1.63
p,p'-DDT	0.60	0.64
Dieldrin	0.47	1.06
Lindane	<0.05	<0.05
Heptachlor epoxide	0.35	0.51
PCB's	1.00	2.50

The tremors noted prior to death, and the high brain and liver residues led us to conclude that DDE poisoning was an important factor in the death, with the associated starvation and dehydration contributory. None of the other observations were considered pathological.

Two American kestrels (Falco sparverius) that died from a low level dietary DDE treatment (2.8 ppm) had brain residues of 212.5 and 301.1 ppm DDE (PORTER and WIEMEYER, 1972). These levels are comparable to the 246 ppm DDE in the heron brain. A bald eagle (Haliaeetus leucocephalus) suspected of having died from organochlorine poisoning contained 385 ppm DDE in the brain in addition to 235 ppm PCB's which probably contributed to its death as well (BELISLE et al., 1972). A loon (Gavia immer) that presumably died from DDD poisoning 1 1/2 years after the DDT moratorium contained 200 ppm DDD and 130 ppm DDE in its brain (PROUTY et al., 1975).

Dietary toxicity data for birds show that DDT is more toxic than either DDD or DDE, and that the relative toxicities of DDD and DDE vary greatly with species (HEATH et al., 1972; STICKEL et al., 1970). Brain DDE residues have been largely excluded from mortality considerations (HILL et al., 1971; STICKEL and STICKEL, 1969; VAN VELZEN et al., 1972). However, DDE brain residues alone have not been reliable indicators of DDT toxicity (GREICHUS and HANNON, 1973; STICKEL and STICKEL, 1969). A system that considers all three components (DDT, DDD, and DDE) has been

proposed (STICKEL et al., 1970). It places relative toxicity values on brain DDT, DDD, and DDE residues, such that one DDT equivalent equals either 1 ppm DDT, 5 ppm DDD, or 15 ppm DDE. Application of the system would yield 16.4 DDT equivalents in the heron brain, based on DDE levels alone. The known lethal range begins at 10 equivalents (STICKEL et al., 1970).

DDE is a metabolite of DDT, and its 250-day biological half-life is approximately 10 times that of DDD or DDT (BAILEY et al., 1969 a, b). Stored DDT residues present a hazard to birds using stored fat during periods of stress (VAN VELZEN et al., 1972). Some wild adult cormorants (Phalacrocorax auritus) contained sufficient quantities of DDT and metabolites to produce intoxication had the residues shifted from carcass to brain (GREICHUS and HANNON, 1973). As the heron was collected on May 12, it is probable that it had recently migrated from its wintering grounds, and had utilized its fat reserves in migration. This may have been responsible for mobilization of the lipid-soluble DDE, which then accumulated in the brain.

Eggshell thickness was inversely correlated with DDE residue levels in great blue heron eggs from Alberta, Canada (VERMEER and REYNOLDS, 1970). In view of the long biological half-life of DDE, and the relative longevity of the larger fish-eating avian species, the evidence indicates that DDE may have a lingering effect upon both mortality and reproductive success in certain birds.

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LITERATURE CITED

BAILEY, S., P. J. BUNYAN, B. D. RENNISON, and A. TAYLOR. Toxicol. Appl. Pharmacol. 14, 13 (1969 a).

BAILEY, S., P. J. BUNYAN, B. D. RENNISON, and A. TAYLOR. Toxicol. Appl. Pharmacol. 14, 23 (1969 b).

BAMFORD, F. Poisons, their isolation and identification. Philadelphia: The Blakiston Co. (1951).

BELISLE, A. A., W. L. REICHEL, L. N. LOCKE, T. G. LAMONT, B. M. MULHERN, R. M. PROUTY, R. B. DEWOLF, and E. CROMARTIE. Pest. Monit. J. 6, 133 (1972).

GREICHUS, Y. A, A. GREICHUS, and R. J. EMERICK. Bull. Environ. Contamin. Toxicol. 9, 321 (1973).

GREICHUS, Y. A. and M. R. HANNON. Toxicol. Appl. Pharmacol. 26, 483 (1973).

GREICHUS, Y. A., J. J. WORMAN, M. A. PEARSON, and D. J. CALL. Bull. Environ. Contamin. Toxicol. 11, 113 (1974).

HEATH, R. G., J. W. SPANN, E. F. HILL, and J. F. KREITZER. Comparative dietary toxicities of pesticides to birds. Washington, D. C.: U. S. Gov't. Printing Office (1972).

HILL, E. F., W. E. DALE, and J. W. MILES. Toxicol. Appl. Pharmacol. 20, 502 (1971).

PORTER, R. D. and S. N. WIEMEYER. Bull. Environ. Contamin. Toxicol. 8, 193 (1972).

PROUTY, R. M., J. E. PETERSON, L. N. LOCKE, and B. M. MULHERN. Bull. Environ. Contamin. Toxicol. 14, 385 (1975).

STICKEL, L. and W. STICKEL. Indust. Med. and Surg. 38, 91 (1969).

STICKEL, W. H., L. F. STICKEL and F.B. COON. pp. 287-294 in "Pesticides Symposia." Miami: Halos and Associates (1970).

VAN VELZEN, A. C., W. B. STILES, and L. F. STICKEL. J. Wildl. Manage. 36, 733 (1972).

VERMEER, K. and L. M. REYNOLDS. Canadian Field-Nat. 84, 117 (1970).