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ORGANOPHOSPHATE INSECTICIDE POISONING OF CANADA GEESE IN THE TEXAS PANHANDLE

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The long-term subtle effects of persistent organochlorine pesticides, such as DDT and dieldrin, on avian populations have been well documented (Anderson and Hickey 1972, Blus et al. 1974, Longcore and Stendell 1977, King et al. 1980). The use of some of these chemicals in the United States has been severely restricted, or banned entirely, and others are under close scrutiny. In contrast, the newer organophosphate compounds (OPs) are favored for field application because they are quick acting and relatively short-lived, and do not accumulate in food webs (Stickel 1974). However, certain of the OPs are extremely toxic to wildlife for short periods after application and reports of mortality in exposed avian populations have increased in recent years (Mendelssohn and Paz 1977, Zinkl et al. 1978, Stone 1979, White et al. 1979, Zinkl et al. 1981). OPs kill primarily by inhibiting acetylcholinesterase in the nervous system, thereby disrupting synaptic transmission of nerve impulses; death usually occurs from asphyxiation because of failure of the respiratory center of the brain (Hill and Fleming 1982). A 20% reduction in brain cholinesterase (ChE) activity of birds indicates exposure to an OP and inhibition greater than 50% may be diagnostic of death (Ludke et al. 1975).

On 26 January 1981, we received word that a large number of dead and dying waterfowl were seen at a playa lake about 3 km southwest of Etter, Moore County, Texas. The following day, Wynn counted 1600 dead waterfowl on or near the playa. These included approximately 1480 Canada Geese (*Branta canadensis*), 20 White-fronted Geese (*Anser albifrons*), 75 Mallards (*Anas platyrhynchos*), and 25 Pintails (*Anas acuta*). Croplands adjacent to the playa were planted with winter wheat, as were many other fields in the surrounding countryside. Because of heavy greenbug (*Schizaphis graminum*) infestations, farmers were applying OPs to their wheat for insect control. In this paper we report the results of our investigation of the waterfowl die-off near Etter, Texas, and discuss the adverse impacts on waterfowl populations from toxic chemical use in this important wintering area.

METHODS

Fifteen dead Canada Geese were collected from the die-off site to determine the cause of mortality. In addition, 7 healthy Canada Geese were shot near Hereford, Texas, to serve as controls in the analyses. Five of the geese found dead were shipped unfrozen to the National Wildlife Health Laboratory, Madison, Wisconsin, for necropsy and

pathological examination. The remaining 10 specimens found dead and the controls were kept frozen until chemical analyses could be performed.

To determine brain ChE activities, brains of controls and geese found dead were homogenized in .05 Tris buffer (pH 8.0) at a ratio of 100 mg/ml. To 20 μl of each homogenate, 2.9 ml dithiobisnitrobenzene acid and .1 ml acetylthiocholine iodide were added, and then the solutions were analyzed simultaneously on a spectrophotometer for ChE activities (Hill and Fleming 1982).

The anterior portion of each gastrointestinal (GI) tract was opened, all contents were removed, placed in a flask, and covered with methylene chloride. The flask was shaken several times over a 4-h period. The methylene chloride extract was analyzed by a gas chromatograph equipped with a flame photometric detector and a 1.5% SP-2250/1.95% SP-2401 column at 192°C. The lower limit of reportable residues was .05 ppm on a wet weight basis. To facilitate analysis by gas chromatography-mass spectrometry (GC-MS), the extract of 1 specimen was cleaned up by gel permeation chromatography, as described by White et al. (1979), except that the first 250 ml of eluate was discarded and then 150 ml was collected. In this specimen, the presence of parathion and methyl parathion was confirmed by GC-MS.

RESULTS

There was no evidence of infectious disease or significant histopathology in the 5 geese examined at the National Wildlife Health Laboratory. However, brain ChE activity was significantly depressed ($P < .001$, $t = 11.4$, df = 15) in birds found dead compared to that in controls, indicating gross exposure to an anti-ChE agent. Inhibition was enough to account for death in all instances (Ludke et al. 1975), ranging from 54 to 86% and averaging 75% (Fig. 1).

The GI tracts of geese found dead were packed with winter wheat; whole wet weight of boluses averaged 23 g and ranged up to 34 g. Apparently, the geese ingested only the leaves and stems of the young wheat plants for we found no trace of root material in the samples. Parathion and methyl parathion were detected in GI tract contents of all the geese found dead that were analyzed (Table 1); both chemicals are highly toxic OPs that are recommended for use on certain grain crops in the Texas Panhandle (Texas Agricultural Extension Service, Pamphlet B-1251). Parathion was detected in the ingesta at a ratio of about 2:1 over methyl parathion (Table 1). No OPs were detected in GI tracts of controls.

DISCUSSION

We conclude that a mixture of parathion and methyl parathion applied to winter wheat was responsible for the deaths of 1600 waterfowl, mostly Canada Geese, found dead at a playa lake near Etter, Texas, on 26 January 1981. We base our conclusion on the premises that no dis-

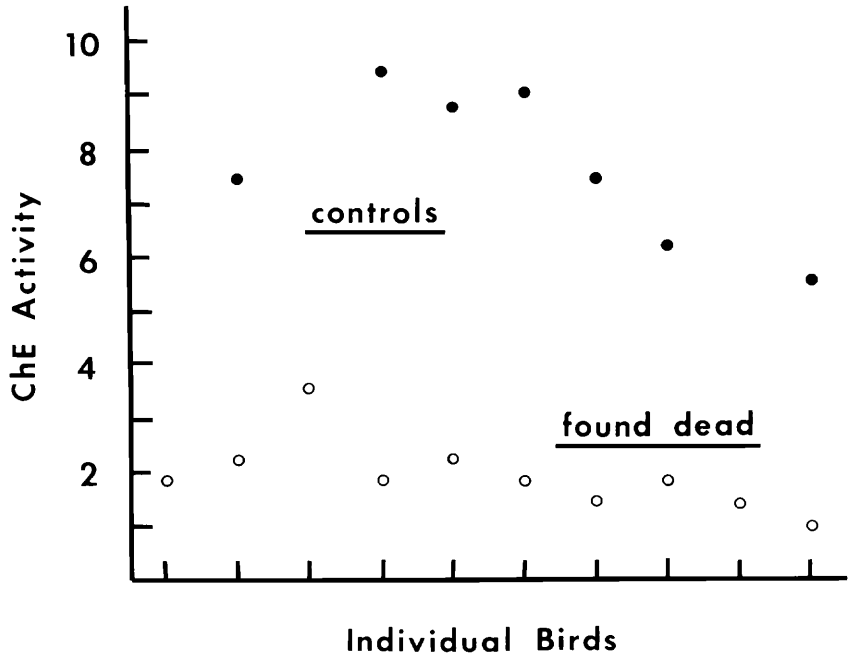

FIGURE 1. Brain cholinesterase (ChE) activity in Canada Geese found dead and in control specimens; data points were plotted randomly, therefore no trends are represented. ChE activity expressed as micromoles acetylthiocholine hydrolyzed per min per g brain tissue. Sample size is 7 controls and 10 specimens found dead.

ease was detected in a sample of the specimens, brain ChE levels in dead birds were 75% below normal, and residues of OPs were present in the ingesta of affected birds. On 24 January 1981, a wheat field situated about 100 m from the edge of the playa received an aerial application of parathion : methyl parathion at a rate of .85 kg per ha; the original formulation used in the mixture contained 2.73 kg parathion and 1.36 kg methyl parathion, or 2 parts parathion to 1 part methyl parathion (William Ray, pers. comm.). The average ratio of parathion to methyl parathion in ingesta of birds found dead was 2.1:1 (Table 1), almost identical to the formulation applied to the nearby wheat field. However, other wheat fields in the surrounding countryside may have been sprayed with similar mixtures, since greenbug infestations of wheat supposedly were widespread in the Texas Panhandle.

Parathion poisoning of waterfowl in wheat fields is not new. In April 1956, near Porter, Oklahoma, 50 Snow Geese (*Chen caerulescens*) apparently were killed from feeding in parathion-sprayed wheat and alfalfa fields (Patuxent Wildl. Res. Cent., Laurel, Md., unpubl. rep.). In March 1967, near Garber, Oklahoma, from 50 to 100 Snow Geese were found

TABLE 1. Organophosphate insecticide residues (ppm wet weight) in GI tract contents of Canada Geese found dead near Etter, Texas.

Bird no.	Parathion	Methyl parathion
1	4.0	1.6
2	4.7	1.6
3[1]	12.0	6.3
4	3.5	2.0
5	6.8	3.1
6	17.0	6.1
7	3.8	1.5
8	5.1	2.7
9	5.3	0.5
10	0.8	4.5

[1] Residues confirmed by mass spectrometry.

dead in and near parathion-treated wheat fields; the stomach contents of 4 specimens analyzed contained parathion residues (Patuxent Wildl. Res. Cent., Laurel, Md., unpubl. rep.). About 500 Canada Geese were found dead in mid-December 1980 at the same playa lake where waterfowl were obtained for this report; parathion and methyl parathion were detected in pooled ingesta of 4 specimens, indicating that the mid-December die-off also was caused by OP poisoning (Texas A&M Veterinary Medical Diagnostic Laboratory, Amarillo, Tex., unpubl. rep.). In late February 1981, 72 geese, mostly Canadas, died on the Hagerman National Wildlife Refuge near Sherman, Texas; brain ChE levels in a sample of the dead geese were inhibited by 78% and their stomachs were full of winter wheat containing parathion residues (D. H. White, unpubl. data). Undoubtedly, many other waterfowl deaths occur in this region from ingestion of treated grain crops but go undetected or unreported. Parathion and methyl parathion also have been responsible for adverse effects on other birds, for example, breeding Laughing Gulls (*Larus atricilla*) (White et al. 1979), songbirds (Edwards and Graber 1968), and Ring-necked Pheasants (*Phasianus colchicus*) (Wolfe et al. 1971).

The Southern High Plains region of the Texas Panhandle is one of the most intensively farmed areas in the United States (Bolen et al. 1979). Principal crops include corn, wheat, and grain sorghum. Several hundred thousand waterfowl spend the winter there and probably derive the major portion of their diet from grain crops (Bolen et al. 1979, Simpson et al. 1981). About 50,000 Canada Geese winter in this area, and because of their grazing habits, they often are associated with wheat and other grain or forage crops. In years when heavy greenbug infestations require frequent pesticide applications, wild geese would be expected to suffer adverse effects.

According to the Texas A&M Agricultural Extension Service (Pam-

phlet B-1251), 5 OPs are recommended for use in greenbug control. Listed in order of decreasing toxicity to Mallards in experimental studies (Hill et al. 1975, Tucker and Crabtree 1970) they are: phorate, parathion, disulfoton, dimethoate, and malathion. Parathion, by far, is the one used most extensively against greenbugs on winter wheat in the Texas Panhandle (William Ray, pers. comm.). Since parathion is a potent waterfowl killer, farmers should be urged to use less toxic materials, such as malathion, on their wheat when waterfowl are in the area. Although malathion may be somewhat more expensive than parathion, it was practically non-toxic to Mallards in experimental feeding studies (Hill et al. 1975).

SUMMARY

Sixteen hundred waterfowl, mostly Canada Geese, died near Etter, Texas, in late January 1981 from anticholinesterase poisoning. Winter wheat in the area of the die-off had been treated with organophosphate insecticides to control greenbugs. Cholinesterase (ChE) levels in brains of a sample of geese found dead were 75% below normal, enough to account for death (Ludke et al. 1975). The gastrointestinal (GI) tracts of geese found dead were packed with winter wheat; gas chromatography techniques identified parathion and methyl parathion in the GI tract contents. Residues of both chemicals were confirmed by mass spectrometry. We recommend that less toxic materials, such as malathion, be used on grain crops when waterfowl are in the vicinity of treatment.

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