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Source: Journal of Wildlife Diseases, 25(2) : 184-188

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-25.2.184>

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CASE HISTORIES OF WILD BIRDS KILLED INTENTIONALLY WITH FAMPHUR IN GEORGIA AND WEST VIRGINIA

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ABSTRACT: Five incidences of bird mortality in Georgia and West Virginia (USA) involving migratory waterfowl, cranes, raptors, corvids and songbirds were investigated during the first 6 mo of 1988. Gross and histopathologic examinations revealed no evidence of infectious or other diseases. However, severe depression of cholinesterase activity was evident in brains of birds found dead, suggesting gross exposure to an organophosphorus (OP) or carbamate pesticide. All of the gastrointestinal tract contents chemically analyzed contained famphur, an OP insecticide used as a pour-on treatment against lice and grubs on livestock, ranging from 5 to 1,480 ppm (wet weight). Grain scattered at two of the mortality sites contained 4,240 and 8,500 ppm famphur. Gastrointestinal tracts of most of the dead birds contained mainly corn and some wheat. This is the first report to document the use of famphur as an intentional means of killing wildlife thought to be depredating crops.

Key words: Organophosphorus poisoning, famphur, raptors, red-tailed hawk, migratory waterfowl, songbirds, sandhill cranes, case histories.

INTRODUCTION

The banning or restriction of certain persistent organochlorine pesticides, such as DDT and dieldrin, has led to increased use of organophosphorus (OP) compounds. Organophosphorus compounds are favored for field application because they are quick-acting, relatively short-lived, and do not accumulate in food webs (Stickel, 1974). However, certain of the OP's are extremely toxic to wildlife for short periods of time after application and reports of mortality in exposed avian populations have been well documented (Mendelssohn and Paz, 1977; Zinkl et al., 1978; Hill and Fleming, 1982; White et al., 1982; Henny et al., 1985). Most avian poisonings are probably inadvertent, but some are intentional (Stone, 1979; White et al., 1983; Flickinger et al., 1984; Stone et al., 1984). The primary action of OP's is inhibition of cholinesterase (ChE) in the nervous system, resulting in disruption of synaptic transmission of nerve impulses; death usually occurs from asphyxiation because of failure of the respiratory center of the brain

(Decandole et al., 1953). In general, a 20% reduction in brain ChE activity indicates exposure to an OP and inhibition of $\geq 50\%$ may be sufficient for diagnosing cause of death (Ludke et al., 1975).

Five separate incidences of bird mortality involving migratory waterfowl, cranes, raptors, corvids, and songbirds were investigated during the first 6 mo of 1988. In this report we present the results of our investigations and document for the first time the illegal use of famphur (O,O-dimethyl O-[p-(dimethylsulfamoyl)phenyl] phosphorothioate) as a means of poisoning depredating wildlife.

MATERIALS AND METHODS

Concerned citizens reported various bird mortalities to the Georgia Department of Natural Resources (DNR), in Georgia (205 Butler St., Atlanta, Georgia 30334, USA) and West Virginia (1800 Washington St., Charleston, West Virginia 25305, USA) during the period 14 January to 11 June 1988. Fresh specimens were collected from each locale by DNR personnel and sent on wet ice the same day to the Southeastern Cooperative Wildlife Disease Study (SCWDS; College of Veterinary Medicine, The

University of Georgia, Athens, Georgia 30602, USA) for necropsy; specimens were necropsied immediately upon arrival at SCWDS. Brains and upper gastrointestinal tracts of dead birds were saved for brain ChE assays and chemical analyses of gastrointestinal tract contents; these were stored frozen (-20 C) for <1 wk until analyses were performed. Brain ChE activities were determined by the method of Ellman et al. (1961) as described by Hill (1988). Healthy individuals of the same species (controls) were not obtainable for determining normal ChE activity, so we used Hill's (1988) control values in testing for ChE depression in birds found dead. Chemical analyses were conducted at the Cooperative Extension Service (College of Agriculture, The University of Georgia, Athens, Georgia 30602, USA).

Gastrointestinal tract contents were homogenized with 50 g sodium sulfate and 300 ml ethyl acetate for 5 to 10 min. Each homogenate was filtered, dried, brought to 10 ml with 3:1 ethyl acetate:toluene and the fat was removed by gel permeation chromatography (Johnson et al., 1976). Sample extracts were analyzed separately with a Tracor Model 565 gas chromatograph (GC; Tracor, Inc., 6500 Tracor Lane, Austin, Texas 78721, USA) equipped with a flame photometric detector operated in the phosphorus mode using a Supelco 3% OV-1 column (Supelco, Inc., Supelco Park, Bellefonte, Pennsylvania 16823, USA), or with a Tracor Model 565 GC equipped with a Ni electron capture detector using a Supelco 1.5% OV-201/1.95% OV-210 column. Famphur residues recovered from samples were confirmed with a Finnigan Model 4000 gas chromatograph-mass spectrometer (Finnigan, Inc., 355 River Oaks Parkway, San Jose, California 95134, USA).

CASE HISTORIES

Mortality in waterfowl and corvids from West Virginia

On 27 February 1988, a private citizen reported seeing sick birds in a field in Upshur County, West Virginia ($38^{\circ}82'\text{N}$, $80^{\circ}20'\text{W}$). On 28 February, DNR personnel found 37 dead birds at the site including 18 Canada geese (*Branta canadensis*), 12 mallards (*Anas platyrhynchos*), one American black duck (*Anas rubripes*), and six American crows (*Corvus brachyrhynchos*). Yellow corn was found scattered in the field to which the birds had access. Four Canada geese, seven mallards, three American crows and a sample of the corn were submitted to SCWDS for necropsy. The birds were in good condition with abundant fat deposits. Gross and histopathologic examinations revealed no evidence of infectious or other dis-

eases. Inhibition of brain ChE activity was a mean of 71 ± 2 (SE)% in the geese, $69 \pm 3\%$ in the mallards and $85 \pm 4\%$ in the crows (Table 1). All the upper gastrointestinal tracts contained large amounts of corn. The gastrointestinal tract contents analyzed ($n = 2$ for each species) contained famphur, averaging 1,250 ppm (wet weight) in geese, 869 ppm in mallards and 823 ppm in crows. The sample of corn collected from the field contained 8,500 ppm famphur.

Mortality in blackbirds from Georgia

In January 1988, a concerned landowner reported seeing numerous dead and dying blackbirds on his property in Whitfield County, Georgia ($34^{\circ}78'\text{N}$, $84^{\circ}92'\text{W}$). A DNR biologist found >100 dead or sick birds; he collected 15 common grackles (*Quiscalus quiscula*) and seven red-winged blackbirds (*Agelaius phoeniceus*) and submitted them to SCWDS for necropsy. The birds were in relatively good condition, and no significant external or internal gross lesions were noted. Brain ChE activities were depressed by averages of $45 \pm 6\%$ in the red-winged blackbirds and $50 \pm 4\%$ in the grackles (Table 1). The gizzards of most of the birds contained corn and a few also contained wheat. Pooled gizzard contents contained famphur at 14.5 and 14.9 ppm for grackles and red-winged blackbirds, respectively.

Mortality in sandhill cranes from Georgia

Two sandhill cranes (*Grus canadensis*) were found dead in a pasture adjacent to a farm in Gilmer County, Georgia ($34^{\circ}47'\text{N}$, $84^{\circ}33'\text{W}$) by DNR law enforcement personnel on 25 February 1988. One crane had been partially eaten and was not collected. The second crane had a radio transmitter on the left leg, and three colored plastic bands and a U.S. Fish and Wildlife Service band on the right leg. This bird was submitted to SCWDS for necropsy. It was an adult in good physical condition with no significant external or internal gross abnormalities. Analysis of brain tissue showed a 75% reduction in ChE activity (Table 1). The upper gastrointestinal tract contained green vegetation and ground corn. Stomach contents contained 69 ppm famphur.

Mortality in American crows and red-tailed hawks from Georgia

On 28 February 1988, a concerned citizen reported finding at least 20 crows, three hawks and several songbirds dead along a creek in Fannin County, Georgia ($34^{\circ}88'\text{N}$, $84^{\circ}27'\text{W}$). DNR personnel searched the area and found eight dead American crows and one red-tailed hawk (*Buteo jamaicensis*), although some carcasses

TABLE 1. Brain cholinesterase (ChE) activities ($\bar{x} \pm SE$) of birds found dead in Georgia (GA) and West Virginia (WV), 1988.

Location	Species (n)	ChE activity ^a	Normal ChE activity ^b
Upshur Co., West Virginia	Canada goose (4)	3.8 \pm 0.31	13 \pm 0.4
	Mallard (7)	3.7 \pm 0.52	12 \pm 0.4
	American crow (3)	3.1 \pm 0.61	20 \pm 1.2
Whitfield Co., Georgia	Common grackle (15)	10.0 \pm 0.82	20 \pm 0.7
	Red-winged blackbird (7)	11.6 \pm 1.21	21 \pm 0.8
Gilmer Co., Georgia	Sandhill crane (1)	4.2	17 \pm 0.5
Fannin Co., Georgia	Red-tailed hawk (1)	4.1	19 \pm 0.8
	American crow (8)	1.9 \pm 0.13	20 \pm 1.2
Dawson Co., Georgia	American crow (2)	2.2 \pm 0.20	20 \pm 1.2

^a ChE activity expressed as micromoles acetylthiocholine hydrolyzed/min/g brain tissue.

^b Normal ChE values for birds were taken from Hill (1988).

could have been removed by predators. Necropsy at SCWDS revealed that all the birds were in good body condition as evidenced by moderate to heavy subcutaneous and abdominal fat and well developed flight muscles. Significant external or internal lesions were not present. Brain ChE activity of the crows was inhibited by an average of $91 \pm 1\%$, and the red-tailed hawk's brain ChE activity was depressed by 78% (Table 1). The gizzards of all the crows contained yellow corn, unidentified small seeds and fine gravel. The crop and stomach of the red-tailed hawk contained crow feathers, body parts including entrails, and yellow corn; the latter was presumably eaten earlier by the crow(s) that the hawk had ingested. Pooled gizzard contents of the crows were found to contain 255 ppm famphur. Stomach contents of the red-tailed hawk contained 5 ppm famphur.

Mortality in American crows from Georgia

On 10 June 1988, a concerned citizen complained to the Georgia DNR that a corn field in Dawson County, Georgia (34°44'N, 84°12'W) had been baited with poisoned grain. A DNR visit on 11 June revealed two dead American crows and 4 to 6 gallons of scratch feed (primarily cracked corn and wheat) scattered over the field. The crows and a sample of the grain bait were submitted to SCWDS for necropsy and chemical analysis. One of the crows was in good physical condition and no significant lesions were observed; its gizzard was full of cracked corn and wheat. The other crow apparently had been crushed by a vehicle and could not be completely examined because of severe trauma to the carcass. Cholinesterase activity in brains of the dead birds was depressed by 88 and 90% (Table 1). Gizzard contents of the intact crow contained 816 ppm famphur.

The sample of grain bait collected from the field contained 4,240 ppm famphur.

DISCUSSION

Famphur is a systemic OP insecticide primarily used as a pour-on treatment on livestock against lice and grubs. It is not registered for use on crops or forests. It is moderately to highly toxic to mammals and extremely toxic to birds (Smith, 1987). Famphur, poured on the backs of cattle, killed black-billed magpies (*Pica pica*) for more than 3 mo, and red-tailed hawks died secondarily from feeding on the poisoned magpies (Henny et al., 1985). Secondary poisoning from famphur also has been documented in bald eagles (*Haliaeetus leucocephalus*) and great horned owls (*Bubo virginianus*) (Franson et al., 1985; Henny et al., 1987). To our knowledge, this is the first report of famphur being used illegally to kill supposedly depredated wildlife.

All five incidences discussed in this report clearly indicate OP poisoning; results of necropsies ruled out disease as a contributing factor. Inhibition of brain ChE activity in most birds found sick or dead exceeded the 50% threshold believed to be indicative of death from OP exposure. Famphur was detected in gastrointestinal tract contents of all the samples analyzed, ranging from 5 ppm (wet weight) in a red-tailed hawk to 1,480 ppm in a Canada

goose. In most instances, famphur residues found in gastrointestinal tract contents were far greater than the oral LD₅₀ values established for mallards (9.9 mg/kg) and songbirds (1.8 to 4.2 mg/kg) in experimental studies (Smith, 1987). The sources of famphur in two incidences were whole and cracked kernels of corn and wheat that had been soaked in the chemical and scattered in and around fields. Apparently, the intention was to kill wildlife thought to be depredating the crops. All birds died from direct ingestion of this poisoned bait, except the red-tailed hawk that had eaten one or more poisoned crows. Famphur-treated bait was found only at two sites, but we do not know how thoroughly the other areas were searched; DNR personnel may not have suspected intentional poisoning in all incidences.

This investigation further demonstrates the harm to migratory waterbirds and other protected species that highly toxic pesticides can cause in the hands of unscrupulous users. Famphur can be added to the list of other toxic OP's, such as monocrotophos, dicrotophos, and parathion (White et al., 1983; Flickinger et al., 1984; Stone et al., 1984), as a means of intentionally killing wildlife.

ACKNOWLEDGMENTS

We thank the Georgia and West Virginia DNR's for bringing the various mortalities to our attention and for the collection of specimens. L. J. Blus provided critical comments, and D. W. Holman and B. J. Fancher typed the manuscript.

LITERATURE CITED

- DECANDOLE, C. A., W. N. DOUGLAS, C. LOVATT-EVANS, R. HOLMES, K. E. V. SPENCER, R. W. TORRANCE, AND K. M. WILSON. 1953. The failure of respiration in death by anticholinesterase poisoning. *British Journal of Pharmacology and Chemotherapy* 8: 466-475.
- ELLMAN, G. L., K. D. COURTNEY, V. ANDRES, JR., AND R. M. FEATHERSTONE. 1961. A new and rapid colorimetric determination of acetylcholinesterase activity. *Biochemical Pharmacology* 7: 88-95.
- FLICKINGER, E. L., D. H. WHITE, C. A. MITCHELL, AND T. G. LAMONT. 1984. Monocrotophos and dicrotophos residues in birds as a result of misuse of organophosphates in Matagorda County, Texas. *Journal of the Association of Official Analytical Chemists* 67: 827-828.
- FRANSON, J. C., E. J. KOLBE, AND J. W. CARPENTER. 1985. Famphur toxicosis in a bald eagle. *Journal of Wildlife Diseases* 21: 318-320.
- HENNY, C. J., L. J. BLUS, E. J. KOLBE, AND R. E. FITZNER. 1985. Organophosphate insecticide (famphur) topically applied to cattle kills magpies and hawks. *The Journal of Wildlife Management* 49: 648-658.
- , E. J. KOLBE, E. F. HILL, AND L. J. BLUS. 1987. Case histories of bald eagles and other raptors killed by organophosphorus insecticides topically applied to cattle. *Journal of Wildlife Diseases* 23: 292-295.
- HILL, E. F. 1988. Brain cholinesterase activity of apparently normal wild birds. *Journal of Wildlife Diseases* 24: 51-61.
- , AND W. J. FLEMING. 1982. Anticholinesterase poisoning of birds: Field monitoring and diagnosis of acute poisoning. *Environmental Toxicology and Chemistry* 1: 27-38.
- JOHNSON, L. D., R. H. WALTZ, J. P. USSARY, AND F. E. KAISER. 1976. Automated gel permeation chromatographic cleanup of animal and plant extracts for pesticide residue determination. *Journal of the Association of Official Analytical Chemists* 59: 174-187.
- LUDKE, J. L., E. F. HILL, AND M. P. DIETER. 1975. Cholinesterase (ChE) response and related mortality among birds fed ChE inhibitors. *Archives of Environmental Contamination and Toxicology* 3: 1-21.
- MENDELSSOHN, H., AND U. PAZ. 1977. Mass mortality of birds of prey by azodrin, an organophosphate insecticide. *Biological Conservation* 11: 163-170.
- SMITH, G. J. 1987. Pesticide use and toxicology in relation to wildlife: Organophosphorus and carbamate compounds. Resource Publication No. 170, U.S. Fish and Wildlife Service, Washington, D.C., 171 pp.
- STICKEL, W. H. 1974. Effects on wildlife of newer pesticides and other pollutants. *Proceedings of the Annual Conference of the Western Association of State Game and Fish Commissions* 53: 484-491.
- STONE, W. B. 1979. Poisoning of wild birds by organophosphate and carbamate pesticides. *New York Fish and Game Journal* 26: 37-47.
- , S. R. OVERMANN, AND J. C. OKONIEWSKI. 1984. Intentional poisoning of birds with parathion. *The Condor* 86: 333-336.
- WHITE, D. H., C. A. MITCHELL, E. J. KOLBE, AND W. H. FERGUSON. 1983. Azodrin poisoning of waterfowl in rice fields in Louisiana. *Journal of Wildlife Diseases* 19: 373-375.

———, ———, L. D. WYNN, E. L. FLICKINGER, AND E. J. KOLBE. 1982. Organophosphate insecticide poisoning of Canada geese in the Texas Panhandle. *Journal of Field Ornithology* 53: 22–27.

ZINKL, J. G., J. RATHERT, AND R. R. HUDSON. 1978.

Diazinon poisoning in wild Canada geese. *The Journal of Wildlife Management* 42: 406–408.

Received for publication 1 September 1988.

Journal of Wildlife Diseases, 25(2), 1989, p. 188

BOOK REVIEW . . .

Zoonoses and Communicable Diseases Common to Man and Animals, Second Edition, Pedro N. Acha and Boris Szyfres. Pan American Health Organization, Scientific Publication 503, Washington, D.C. 20037, USA. 1987. 963 pp. \$20.00 U.S.

The second edition of this paperback book has been updated, expanded to include additional and newly recognized diseases, and broadened to provide worldwide coverage of the common zoonotic and communicable diseases of humans and domestic animals. This comprehensive book contains information on 176 diseases divided into five sections: bacterioses, mycoses, chlamydioses and rickettsioses, viroses, and parasitic zoonoses. The various diseases are not covered in equal detail because of the availability of information, the frequency of occurrence, the distribution of the disease, and possibly the interests of the authors. Many of the important diseases, such as brucellosis, rabies, Venezuelan equine encephalitis, trypanosomiasis, and schistosomiasis, are described in depth, but a few important diseases, such as Lyme disease, Rocky Mountain spotted fever, Colorado tick fever and Chikungunya fever, are not well described.

Diagrams of the modes of transmission are presented for many of the diseases, although some are oversimplified; distribution maps and disease frequency charts and tables are included where necessary. The description of each disease includes information on synonymy, etiology, geographic distribution, occurrence and manifestations in humans and animals, source of infection and mode of transmission, diagnosis, control, the epidemiologic role of animals, and a limited bibliography. Comprehensive summary tables are provided at the end of each main section, and the appendix includes a thorough summary table of food-borne diseases and a glossary of epidemiologic terms.

The primary emphasis of the book is on disease in humans and domestic animals and less on disease in wildlife species, although for a few diseases such as rabies and hydatidosis, the involvement of wildlife species is covered more thoroughly. In general, the species of wildlife involved as reservoirs and infected by the pathogens are listed, but information on the ecology of the diseases and on the role of the vertebrate hosts is limited. In the sections on diseases with which I am most familiar, I noted a few minor mistakes (e.g., wild fowl instead of wild passerines as the natural vertebrate hosts for eastern equine encephalitis virus and a tree squirrel instead of a ground squirrel or a chipmunk as the vertebrate host in the simple diagram of the virus cycle of Colorado tick fever virus). Some of these inaccuracies may result from having used secondary sources instead of original publications. The book contains only a few spelling and typing errors and a few misused terms; in general, the book is very well written.

This book, which brings together a vast amount of information on a large number and variety of diseases found throughout the world, will be a handy reference for researchers and public health workers concerned with this array of diseases. The book will be useful as a text in courses on zoonotic diseases and vector and vector-borne diseases and as a supplemental text in courses on wildlife diseases. This excellent summary of the current scientific and technical knowledge will help many countries study some of their significant zoonotic diseases and solve their public health and veterinary health problems.

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