

Brain Cholinesterase Inhibition in Songbirds from Pecan Groves Sprayed with Phosalone and Disulfoton

Authors: White, Donald H., and Seginak, John T.

Source: Journal of Wildlife Diseases, 26(1): 103-106

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-26.1.103

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at <u>www.bioone.org/terms-of-use</u>.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

Brain Cholinesterase Inhibition in Songbirds from Pecan Groves Sprayed with Phosalone and Disulfoton

Donald H. White and John T. Seginak, U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Southeast Research Station, School of Forest Resources, The University of Georgia, Athens, Georgia 30602, USA

ABSTRACT: Brain cholinesterase (ChE) activities of songbirds collected in pecan groves 6 to 7 hr after separate applications of the organophosphorus pesticides, phosalone and disulfoton, were compared to mean ChE activities of controls (normals) as a measure of insecticide exposure. In general, reduction of brain ChE activity ≥ 2 standard deviations below the control mean indicates exposure to an anticholinesterase compound. Phosalone had little effect on brain ChE activity of birds from treated groves; only slight to moderate (21 to 38%) ChE inhibition was detected in blue jays (Cyanocitta cristata) and red-bellied woodpeckers (Melanerpes carolinus). However, 11 of 15 blue jays from disulfoton-treated groves had moderate to severe ChE depression, ranging from 32 to 72%. Inhibition $\geq 50\%$ of normal may be diagnostic for cause of death. Direct mortality was not observed, but studies have shown that bird carcasses disappear rapidly from agricultural areas, many within 24 hr. We recommend additional field studies of the effects of disulfoton to wildlife, since large wheat-growing areas in the western United States are being considered for disulfoton treatment to control the Russian wheat aphid (Diuraphis noxia).

Key words: Cholinesterase inhibition, songbirds, disulfoton, phosalone, organophosphorus pesticides, insecticide, field study.

Pecan cultivation is a major industry in southern Georgia (USA). Thousands of acres of pecan groves are regularly sprayed during summer with large quantities of chemicals, mainly organophosphorus (OP) compounds. Many of the OP's are extremely toxic to wildlife, and reports of anticholinesterase poisoning in exposed avian populations have been well documented (Seabloom et al., 1973; Mendelssohn and Paz, 1977; Hill and Fleming, 1982; White et al., 1982). In general, reduction of brain cholinesterase (ChE) activity ≥ 2 standard deviations (SD) below the control mean is indicative of OP exposure, and inhibition $\geq 50\%$ of normal may be sufficient for diagnosing cause of death (Ludke et al., 1975). Some investigators have measured brain ChE activity in wild birds collected in areas after OP applications as a means of monitoring exposure (Zinkl et al., 1980; DeWeese et al., 1983; Niethammer and Baskett, 1983; Grue and Hunter, 1984).

Many species of songbirds nest in pecan groves in southern Georgia. As part of a study to determine reproductive effects of pesticides on birds nesting in pecan groves, we collected specimens before and after phosalone (phosphorodithioic acid S-[(6chloro-2-oxo-3(2H)-benzoxazolyl)methyl] 0,0-diethyl ester) and disulfoton (phosphorodithioic acid 0,0-diethyl S-[2-(ethylthio) ethyl] ester) applications to groves for brain ChE determinations. Here, we report the results of our brain ChE assays and discuss potential hazards to wildlife from the use of highly toxic OP's, such as disulfoton.

The study site was Wildmeade Plantation (31°30'N, 84°31'W) near Leary, Calhoun County, Georgia (USA); about 245 ha are in cultivated pecan groves. Blue jays (Cyanocitta cristata) and red-bellied woodpeckers (Melanerpes carolinus) were shot in pecan groves that were sprayed 6 to 7 hr earlier with phosalone on 8 July 1987 and with disulfoton on 26 August 1987. Both compounds (emulsifiable concentrates) were applied in separate groves by truck-mounted blower at the rate of 0.83 kg active ingredient/ha. Birds were tagged, placed in polyethylene bags on wet ice, and frozen (-20 C) at the end of the day. Control specimens for normal ChE determinations were collected in groves on 7 July 1987 before any pesticides were applied and handled in the same way. Partially thawed half-brains were excised and assayed for ChE activity following Ellman et al. (1961) as described by Hill and Flem-

TABLE 1. Brain cholinesterase (ChE) activities of controls collected in pecan groves before pesticide applications.

Species	n	ChE activity*		
		Mean	SD	Bounds ^b
Blue jay Red-bellied	5	34.5	3.7	27.1-41.9
woodpecker	5	35.9	2.1	31.7-40.1

• ChE activity expressed as micromoles acetylthiocholine hydrolyzed/min/g brain tissue, wet weight.

^b Normal bounds = mean ± 2 SD.

ing (1982). Cholinesterase activities for controls and experimentals were determined concurrently on a Spectronic 401 spectrophotometer (Milton Roy Co., Rochester, New York 14625, USA) at about 25 C.

Brain ChE activities of controls collected in pecan groves before pesticide applications are summarized in Table 1. The mean \pm standard error (SE) ChE activity (expressed as micromoles acetylthiocholine hydrolyzed/min/g brain tissue, wet weight) for blue jays was 34.5 ± 1.65 (range = 29.2 to 39.1) and that for red-bellied woodpeckers was 35.9 ± 0.94 (range = 32.9 to 38.4).

Cholinesterase activities of most birds collected from phosalone-treated groves 6 to 7 hr postspray were within the range of normal ChE activity (Fig. 1). Six blue jays had slight to moderate ChE inhibition when compared to the control mean, ranging up to 38%. Only two red-bellied woodpeckers showed slight ChE depression of 21 and 24%. Phosalone is a broad-spectrum nonsystemic OP insecticide and acaricide used primarily on fruit and nut trees; the major crop is pecans. Phosalone is considered only moderately toxic to laboratory animals, but its toxicity to most wildlife species has not been evaluated (Smith, 1987). Wildlife mortality or other adverse effects from phosalone applications have not been reported.

In contrast, blue jays collected from pecan groves 6 to 7 hr after disulfoton was applied had moderate to severe ChE

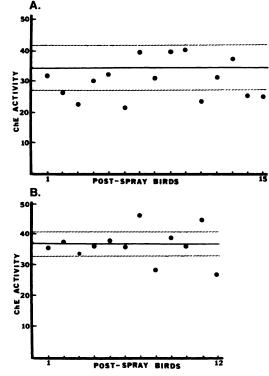


FIGURE 1. Brain cholinesterase (ChE) activities of 15 blue jays (A) and 12 red-bellied woodpeckers (B) from pecan groves 6 to 7 hr after a phosalone application. The black line in each graph is the control mean and wavy lines are normal bounds, defined as the control mean ± 2 SD.

depression. In 11 of 15 birds, ChE was inhibited by 32 to 72%; four birds had normal activity (Fig. 2). Only one red-bellied woodpecker was obtained, and its ChE activity was within normal bounds. Disulfoton is a systemic OP insecticide used as a side dressing, as a foliar spray or broadcast to control insects and mites. It is extremely toxic to mammals, birds, fish, aquatic organisms and bees (Thomson, 1982). In an eight to 10 county area where disulfoton was broadcast aerially, about 50 mammals and birds were reportedly killed (Environmental Protection Agency, 1981). Toxicity studies have documented very low acute oral LD₅₀'s for mammals and birds (Hudson et al., 1984), and acute dermal LD_{50} 's are also low, as disulfoton is rapidly absorbed through the skin (Hudson et al.,



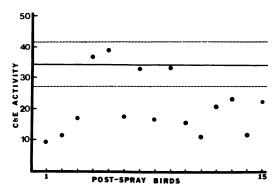


FIGURE 2. Brain cholinesterase (ChE) activities of 15 blue jays from pecan groves 6 to 7 hr after a disulfoton application. The black line is the control mean and wavy lines are normal bounds, defined as the control mean ± 2 SD.

1979; Gaines, 1969). Disulfoton residual insecticidal activity may last 6 to 8 wk after treatment (Chemagro Corporation, 1971).

The phosalone application probably had a minimal effect on blue jays and redbellied woodpeckers using the groves, as indicated by normal ChE activity for most specimens 6 to 7 hr postspray (Fig. 1). However, eight of 15 blue jays collected 6 to 7 hr postspray (Fig. 2) had ChE depression (50 to 72%) within the range diagnostic for cause of death from OP toxicosis (Ludke et al., 1975). While average brain ChE depression of birds killed by OP's in the field usually is $\geq 80\%$ (Hill, 1988), mortality may occur with as little as 39% ChE depression (White et al., 1989), dependent upon the chemical and the species affected. No dead birds were found during systematic searches along 200-m transects 24 hr after chemicals were applied, but mortality may have been undetected. Bird carcasses disappear rapidly from agricultural areas, many within 24 hr, presumably removed by predators or scavengers (Balcomb, 1986). In addition, sublethal exposure to an OP may alter nesting behavior (Grue et al., 1982; White et al., 1983) or render birds more susceptible to predation or food deprivation (White et al., 1979). Large numbers of songbirds nest in or adjacent to pecan groves in southern Georgia and studies are underway to evaluate the effects of OP's on reproduction in several species.

Presently, disulfoton is being considered for broad-scale use in wheat-growing areas of the western United States for control of the Russian wheat aphid (*Diuraphis noxia*). Few data exist of the environmental consequences of disulfoton use (Smith, 1987). Our limited results suggest that disulfoton may be hazardous to birds using treated areas; however, additional field studies incorporating a wider variety of species and collection regimens are warranted.

We thank H. A. Shepard, owner of Wildmeade Plantation, for access to his property. R. C. Simpson assisted in field collections, C. E. Grue and E. F. Hill reviewed the manuscript, and B. J. Fancher typed the manuscript.

LITERATURE CITED

- BALCOMB, R. 1986. Songbird carcasses disappear rapidly from agricultural fields. Auk 103: 817– 820.
- CHEMAGRO CORPORATION. 1971. Di-syston insecticide technical data sheet. Chemagro Corporation, Kansas City, Missouri, 4 pp.
- DEWEESE, L. R., L. C. MCEWEN, L. A. SETTIMI, AND R. D. DEBLINGER. 1983. Effects on birds of fenthion aerial application for mosquito control. Journal of Economic Entomology 76: 906–911.
- 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.
- ENVIRONMENTAL PROTECTION AGENCY. 1981. Pesticide incident monitoring system, summary reports 1978-81. U.S. Environmental Protection Agency, Washington, D.C., 116 pp.
- GAINES, T. B. 1969. Acute toxicity of pesticides. Toxicology and Applied Pharmacology 14: 515– 534.
- GRUE, C. E., AND C. C. HUNTER. 1984. Cholinesterase activity in fledgling starlings: Implications for monitoring exposure of songbirds to ChE inhibitors. Bulletin of Environmental Contamination and Toxicology 32: 282–289.
- —, G. V. N. POWELL, AND M. J. MCCHESNEY. 1982. Care of nestlings by wild female starlings exposed to an organophosphate pesticide. Journal of Applied Ecology 19: 327–335.
- 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.
- HUDSON, R. H., M. A. HAEGELE, AND R. K. TUCKER. 1979. Acute oral and percutaneous toxicity of pesticides to mallards: Correlations with mammal toxicity data. Toxicology and Applied Pharmacology 47: 451-460.
- , R. K. TUCKER, AND M. A. HAEGELE. 1984. Handbook of toxicity of pesticides to wildlife, 2nd ed. Resource Publication No. 153, U.S. Fish and Wildlife Service, Washington, D.C., 90 pp.
- 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.
- NIETHAMMER, K. R., AND T. S. BASKETT. 1983. Cholinesterase inhibition of birds inhabiting wheat fields treated with methyl parathion and toxaphene. Archives of Environmental Contamination and Toxicology 12: 471-475.
- SEABLOOM, R. W., G. L. PEARSON, L. W. ORING, AND J. R. REILLY. 1973. An incident of fenthion mosquito control and subsequent avian mortality. Journal of Wildlife Diseases 9: 18-20.

- 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.
- THOMSON, W. T. 1982. Agricultural chemicals, Vols. 1–4. Thomson Publishing, Fresno, California, 1,821 pp.
- WHITE, D. H., L. E. HAYES, AND P. B. BUSH. 1989. Case histories of wild birds killed intentionally with famphur in Georgia and West Virginia. Journal of Wildlife Diseases 25: 184-188.
- , K. A. KING, C. A. MITCHELL, E. F. HILL, AND T. G. LAMONT. 1979. Parathion causes secondary poisoning in a laughing gull breeding colony. Bulletin of Environmental Contamination and Toxicology 23: 281–284.
- , C. A. MITCHELL, AND E. F. HILL. 1983. Parathion alters incubation behavior of laughing gulls. Bulletin of Environmental Contamination and Toxicology 31: 93–97.
- ——, ——, 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., R. B. ROBERTS, C. J. HENNY, AND D. J. LENHART. 1980. Inhibition of brain cholinesterase activity in forest birds and squirrels exposed to aerially applied acephate. Bulletin of Environmental Contamination and Toxicology 24: 676-683.

Received for publication 17 April 1989.