

FENTHION POISONING OF WADING BIRDS

Authors: ZINKL, JOSEPH G., JESSUP, DAVID A., BISCHOFF, A.I., LEW, TOM E., and WHEELDONE, ERIC B.

Source: Journal of Wildlife Diseases, 17(1) : 117-119

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-17.1.117>

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 www.bioone.org/terms-of-use.

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.

FENTHION POISONING OF WADING BIRDS

JOSEPH G. ZINKL,¹ DAVID A. JESSUP,² A.I. BISCHOFF,² TOM E. LEW² and
ERIC B. WHEELDON²

Abstract: Low brain and serum cholinesterase activity were found in several species of wading birds. The area from which these birds were taken recently had been sprayed with the organophosphorous insecticide fenthion (*O*, *O*-dimethyl *O* - [4-(methylthio)-*m* - tolyl] phosphorothioate). Analysis of stomach contents and water samples revealed residues of fenthion. These findings suggest that fenthion caused lethal cholinesterase depression.

CASE REPORT

On 4 April 1979, seven dead snowy egrets (*Egretta thula*) and two dead great egrets (*Casmerodius albus*) were recovered from an active dredge spoil site and an adjacent slough near the city of Vallejo, California. In addition, a snowy egret that was unable to stand or fly and a great blue heron (*Ardea herodias*) that was convulsing and dyspneic were captured. Water samples also were taken from the dredge site and adjacent slough.

The great blue heron was sent to the Veterinary Medical Teaching Hospital (VMTH) of the School of Veterinary Medicine, University of California, Davis. The other birds and water samples were sent to the California Department of Fish and Game Wildlife Investigations Laboratory, Sacramento.

The live snowy egret was incoordinated and was unable to stand or fly. Tonic contracture of the flexors of the feet subsided in the first two days. Forced alimentation and rehydration and maintenance of body temperature were the only therapeutic measures taken. Over the next 7 days the bird recovered the use of its legs and wings. Blood samples were collected on 6 and 9 April,

and the serum was harvested to determine cholinesterase (ChE) activity. On 11 April the bird was released.

The great blue heron was prostrate, was dyspneic and was having clonic convulsions. It died shortly after reaching the VMTH. A blood sample was obtained shortly before death.

Two days prior to the collection of the dead birds, the area in which they were found had been sprayed with 0.11 kg fenthion (*O*, *O*-dimethyl *O* - [4-(methylthio) - *m* - tolyl] phosphorothioate) (Baytex®) per hectare (0.1 lb/acre), the recommended rate for mosquito control.

Laboratory Findings

Necropsies of the dead birds revealed no significant lesions. Neither the snowy egrets nor the great blue heron had any food in their intestinal tracts, but food items were present in the stomachs of both great egrets. These were collected and composited for insecticide residue analysis.

Brains of all the dead birds and of a great blue heron that had died in an unrelated incident, together with the serum samples of the great blue heron and the live snowy egret, were analyzed

¹ Department of Clinical Pathology, School of Veterinary Medicine, University of California, Davis, California 95616, USA.

² California Department of Fish and Game, 987 Jedamith Drive, Sacramento, California 95819, USA.

² Department of Pathology, School of Veterinary Medicine, University of California, Davis, California 95616, USA.

for ChE activity by a modification¹ of the Ellman² technique. Brain ChE activities were very low, very likely much lower than the minimum 50% inhibition indicative of lethal exposure.⁴ Serum ChE activity of the great blue heron and the first sample of the live snowy egret were also low. Subsequently, greater serum ChE activity was present in the second sample of the live snowy egret (Table 1).

Water samples were extracted with petroleum ether and analyzed on the Varian 3700 gas chromatograph equipped with a flame photometric detector in the phosphorus mode. The dredge spoil pond water did not show any insecticide. However, water from the adjacent slough had 16 $\mu\text{g}/\text{l}$ (16 ppb) fenthion. The stomach contents were extracted with acetone and analyzed in a manner similar to the water samples. The combined stomach contents of the great egrets contained 93 mg/kg (93 ppm) fenthion.

DISCUSSION

The extremely low serum and brain ChE activities suggest that the birds were poisoned by a ChE inhibiting insecticide. It is not possible to determine the magnitude (percent) of the brain ChE depression since control brains for the egrets could not be obtained. It is very likely that inhibition was much greater than 50%, however. A single frozen brain from a great blue heron that had died

previously in an unrelated incident had a very high activity compared to that of the heron in the present incident (Table 1). Comparison of these activities indicate that the brain ChE activity of the heron of the present study was about 95% inhibited. Additionally, the marked increase in the snowy egret's serum ChE activity from 6 to 9 April further indicates that a ChE inhibitor had been present earlier.

The detection of fenthion in water from the area where the birds had been found and in the stomach contents indicate that the ChE inhibitor was fenthion. Fenthion is very toxic to birds, having an acute LD_{50} of 5.9 mg/kg for Canada geese.⁶

The poisoning of these birds appears to have been accidental since the insecticide was used at its recommended rate. It is thought that because of inshore wind, the pesticide may have been sprayed into the wind in order to reach the far side of the slough. Perhaps subsequent wind action on the water surface concentrated the toxicant on the leeward side of the slough. The birds could have been exposed to a very high concentration since the residue concentration in the water was 16 $\mu\text{g}/\text{l}$ two days after the spray. It was calculated that 0.11 kg/ha, the highest recommended rate for mosquito control, would produce a concentration of 147 $\mu\text{g}/\text{l}$ in water 15 cm deep. Perhaps the concentration during the

TABLE 1. Brain and serum cholinesterase activities in birds dying after exposure to fenthion.

Species	n	Brain (mU/mg)	Serum (mU/ml)
Snowy Egret	6	0.3-3.4	
	1		295.8 (2 days after spray) 941.3 (5 days after spray)
Great Egret*	2	1.9-2.6	
Great Blue Heron	1	3.0**	138.3
Control			
Great Blue Heron	1	55.3	

*Stomach contents contained 93 mg/kg fenthion

**Approximately 95% depression

initial stages was much higher than this and exposure of birds at that time killed them. In addition, they may have been attracted to poisoned and distressed food items.³ Alternatively, birds of these species may be very susceptible to fenthion, and the recommended rate is lethal for them.

Incidents such as this suggest that unfavorable conditions can affect the safety of an applied insecticide. This poisoning is probably not an isolated incident since similar incidents of poisoning have occurred. Wilson's phalaropes (*Setganopus tricolor*), redwinged blackbirds (*Agelaius phoeniceus*) and other birds as well as a few mammals apparently were killed by fenthion in the Spring, 1978 and 1979 near Laramie, Wyoming, (Deweese, R.L. U.S. Fish and Wildlife Service, pers. commun.). Fenthion was responsible in January, 1976, for the death of cedar waxwings (*Bombycilla cedrarum*) in Tulare County, California, where the

pesticide had been used illegally on pyracantha plants. Gizzard contents from the dead birds contained 156 ppm fenthion while 11.5 ppm was detected in berries of the treated plants (Bischoff, unpubl.). In 1969, many birds, perhaps up to 25,000, were killed by fenthion in a mosquito control operation in North Dakota.⁵ In 1968, bird kills caused by fenthion occurred in Idaho, Ohio and California.³ As early as 1962, fenthion was thought responsible for the death of shore birds and wading birds in a mosquito control operation in Delaware.⁴ Several investigators suggest that the high kill rate of fenthion leaves many dead and dying insects that attract birds which die after eating the insecticide laden insects.⁴ Perhaps the improper application under adverse field conditions occurred and resulted in a high concentration of fenthion in the food of the birds. Increased susceptibility of the birds may have compounded the situation.

LITERATURE CITED

1. DIETER, M.P. and J.L. LUDKE. 1975. Studies on combined effect of organophosphates and heavy metal in birds. I. Plasma and brain cholinesterase in coturnix quail fed methyl mercury and orally dosed with parathion. Bull. Environ. Contamin. Toxicol. 13: 257-262.
2. ELLMAN, G.L., K.D. COURTNEY, V. ANDRES, Jr. and R.M. FEATHERSTONE. 1961. A new and rapid colormetric determination of acetylcholinesterase activity. Biochem. Pharmacol. 7: 88-95.
3. KEITH, J.O. and D.B. KNAPP. 1969. Some effects on birds of fenthion used as a mosquito larvicide. Proc. and Papers. 37th Ann. Conf. Calif. Mosquito Control Ass. 4 p.
4. LUDKE, J.L., E.F. HILL and M.P. DIETER. 1975. Cholinesterase (ChE) response and related mortality among birds fed ChE inhibitors. Arch. Environ. Contamin. Toxicol. 3: 1-21.
5. SEABLOOM, R.W., G.L. PEARSON, L.W. ORING and J.R. REILLY. 1973. An incident of fenthion mosquito control and subsequent avian mortality. J. Wildl. Dis. 9: 18-20.
6. TUCKER, R.K. and D.G. CRABTREE. 1970. Handbook of Toxicity of Pesticides to Wildlife. U.S. Department of Interior, Fish and Wildlife Service Resource Publication No. 84. 131 pp.

Received for publication 17 April 1980