Azodrin® Poisoning of Waterfowl in Rice Fields in Louisiana

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Although infestations of sarcoptic mange have sometimes occurred concomitantly with lice infestations (Gier et al., 1978, *In* Coyotes: Biology, Behavior, and Management, Bekoff (ed.), Academic Press, New York, 384 pp.; Pence and Custer, 1981, *In* Worldwide Furbearer Conf. Proc., Vol. II, Chapman and Pursely (eds.), Worldwide Furbearer Conf., Inc., Frostburg, Maryland, pp. 760–845), there was no evidence of mange mites on the four hide samples examined in our study.

Trichodectes canis has not been previously identified on wild canid populations in Alaska, although it is found occasionally on dogs according to a statewide survey of veterinarians. Although no additional morbidity or mortality was observed, it seems obvious that severely infested wolves have a higher probability of contracting other diseases associated with stress or possibly suffer increased mortality from exposure during severe winters.

In addition, the extreme hair loss associated with heavy lice infestations reduced the commercial value of pelts. Hides of most pup wolves and the coyote were worthless, while those of the adult wolves were worth one-third of their normal market value. If the prevalence of infestation on wolves from the Kenai Peninsula persists or increases, the commercial value of their hides and possibly those of coyotes will decrease significantly.

We thank S. Mersch who originally isolated and identified the louse; Dr. R. Zarnke for examining scalp samples for mange; T. Spraker, Game Biologist, for collecting lice specimens from sealed wolves; trappers J. Cook, W. Sather, C. Bierdaman, E. Jordan, and A. Horwath for providing samples of lice from harvested wolves; E. Bangs, Kenai National Wildlife Refuge Biologist, for data on physical condition of harvested wolves, B. Taylor for data on louse density and information from his statewide survey of veterinarians concerning *T. canis* occurrence on domestic dogs; and K. Schneider, A. Franzmann, W. Ballard, and S. Peterson for reviewing the manuscript.

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During the period 2–4 April 1981 about 100 birds, mostly ducks and geese, were found dead and dying in a rice field near Sweet Lake, Calcasieu Parish, Louisiana. Fresh specimens were collected to determine the cause of mortality. Birds were placed individually in polyethylene freezer bags, tagged, and frozen soon after collection. Four snow geese (*Chen caerulescens*), two blue-winged teal (*Anas discors*), one green-winged teal (*Anas crecca*), and one mottled duck (*Anas fulvigula*) were shipped to the National Wildlife Health Laboratory (NWHL), Madison, Wisconsin, for necropsy and pathological examination. Ten snow geese, 10 blue-winged teal, three green-winged teal, three great-tailed grackles (*Quiscalus mexicanus*), and eight red-winged blackbirds (*Agelaius phoeniceus*) were transported to the Gulf Coast Field Station, Victoria, Texas, for brain acetylcholinesterase (AChE) activity determinations and preparation for chemical residue analysis. Additionally, apparently healthy specimens of the affected species were collected near Lake Charles, Louisiana, and Victoria, Texas, to serve as controls in the analyses.

Brain AChE activities of birds found dead and of controls were determined by the Ellman et al. (1961, Biochem. Pharmacol. 7: 88–95) technique as described by Hill and Fleming (1982, Environ. Toxicol. Chem. 1: 27–38). Most of the proventriculi of the birds found dead

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TABLE 1. Brain acetylcholinesterase (AChE) activities of birds found dead and of apparently healthy specimens (controls).

Species	n	x̄ AChE activity[a]	x̄ % inhibition
Snow goose			
Died	10	1.5 (1.1–1.9)[b]	84
Controls	7	9.3 (4.8–13.8)	0
Blue-winged teal			
Died	10	2.1 (1.4–3.6)	85
Controls	10	13.6 (10.1–16.7)	0
Green-winged teal			
Died	3	1.7 (1.4–2.2)	82
Controls	3	9.6 (8.2–10.1)	0
Great-tailed grackle			
Died	3	1.8 (1.1–2.8)	89
Controls	5	15.8 (12.8–17.8)	0
Red-winged blackbird			
Died	8	3.1 (1.1–5.1)	86
Controls	5	21.6 (18.9–25.5)	0

[a] AChE activity expressed as micromoles acetylthiocholine hydrolyzed per min per g brain tissue.
[b] Extreme values.

were packed with rice seed. The proventricular contents of the five snow geese and five blue-winged teal and those of a control snow goose and a control blue-winged teal were removed and analyzed separately at the Patuxent Wildlife Research Center for organophosphate (OP) insecticide residues as were seven rice seed samples collected from the die-off site. Residues were determined on a gas chromatograph equipped with a flame photometric detector following White et al. (1982a, J. Field Ornithol. 53: 22–27).

There was no evidence of infectious disease or significant histopathology in the birds examined at the NWHL. However, brain AChE activity was greatly inhibited in birds found dead compared to that in controls (Table 1), indicating gross exposure to an anti-AChE agent. Inhibition far exceeded the 50% level considered sufficient for diagnosing cause of death (Ludke et al., 1975, Arch. Environ. Contam. Toxicol. 3: 1–21). Azodrin® (=monocrotophos; (E)-phosphoric acid dimethyl (1-methyl-3-(methylamino)-3-oxo-1-propenyl) ester) was detected in proventricular contents of all the snow geese and in three of the five blue-winged teal that were analyzed, ranging from 0.65 to 110 ppm, wet weight; residues in one specimen were confirmed by mass spectrometry (White et al., 1979, Bull. Environ. Contam. Toxicol. 23: 281–284). Also, five of the seven rice seed samples collected at the die-off site contained Azodrin, ranging from 160 to 720 ppm, wet weight; its presence was confirmed by mass spectrometry in one rice seed sample. No OP insecticides were detected in the proventricular contents of control birds.

Organophosphate compounds are favored for field application because they are quick-acting and there is little known bioaccumulation in food webs (Stickel, 1974, Annu. Conf. West. Assoc. State Game Fish Comm. 53: 484–491). Nevertheless, certain of the OP's 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, Biol. Conserv. 11: 163–170; Stone, 1979, N.Y. Fish Game J. 26: 37–47; White et al., 1982a, op. cit.; White et al., 1982b, J. Wildl. Dis. 18: 389–391). In controlled laboratory studies Azodrin was highly toxic to mallards (*Anas platyrhynchos*), having a single-dose LD_{50} of about 4.8 mg/kg (Tucker and Crabtree, 1970, U.S. Fish and Wildl. Serv., Resource Publ. No. 84, 131 pp.), and it also has been implicated in massive secondary poisoning of birds of prey in the field (Mendelssohn and Paz, 1977, op. cit.).

We conclude that anti-AChE poisoning due to Azodrin was the cause of death in about 100 birds, mostly waterfowl, found at a rice field near Sweet Lake, Louisiana. Our conclusion is based on the premise that no lesions or signs of disease were detected in a sample of the specimens, brain AChE activity in birds found dead averaged 82–89% below normal (Table 1), and Azodrin was detected in the ingesta of affected birds. Azodrin is a restricted-use pesticide that has not been registered for use on rice in any fashion (EPA reg. No. 201-157). In this incident, rice seed was soaked in Azodrin and broadcast from an airplane onto a field that had already been planted with rice several days earlier. In fact, the field had a stand of seedlings 5–10 cm high, so there was no reason to replant.

Thus, the Azodrin-soaked seed may have been broadcast with the sole intention of poisoning depredating waterfowl and blackbirds. Informants told us that the illegal practice had been going on for years and that large numbers of migratory waterfowl and other birds had been killed in the process. The practice is not restricted to Louisiana. In the spring of 1982, Azodrin-soaked rice seed was used to kill waterfowl and other birds in Texas rice fields (Flickinger and White, pers. obs.). This investigation demonstrates the harm to wildlife that potentially beneficial, but highly toxic, pesticides can cause in the hands of unscrupulous users.

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BOOK REVIEW . . .

CRC Handbook Series in Zoonoses—Section C: Parasitic Zoonoses (Volumes I–III), James H. Steele, editor-in-chief, CRC Press, Inc., 2000 Corporate Blvd., N.W., Boca Raton, Florida 33431, USA. 1982. Vol. I, 400 pp., $86.00 (US), outside USA $99.00 (US); Vol. II, 360 pp., $79.50 (US), outside USA $91.50 (US); Vol. III, 384 pp., $86.00 (US), outside USA $99.00 (US).

Section C of the Handbook Series in Zoonoses is a three-volume reference for over 70 protozoan, cestode, nematode, trematode, and arthropod parasitic infections affecting humans and the lower animals. The book has been recommended for "public health workers and the specialists, and to many others who share the view that man's well-being and health are directly related to that of animals." A notable feature of the series is the orderly and systematic presentation of each subject and the frequent inclusion of an extensive bibliography. Each disease is discussed with sections on common synonyms, etiologic agent, life cycle including primary and alternate hosts, geographic distribution, disease in animals and man, diagnosis, prevention and control, and references. The diseases are arranged in alphabetic order with an index for each volume. The contributing authors are recognized experts in parasitology and tropical medicine.

The Handbook Series in Parasitic Zoonoses has several shortcomings that limit its usefulness for wildlife specialists. There is no host species index and the general index contains few references to wildlife species. For example, in Volume II, Nematode Zoonoses, there is only one reference to the skunk and raccoon as they relate to gnathostomiasis, while there is no reference to the bear or fox as they pertain to trichinosis. In addition, some diseases are reviewed in depth with more than 300 up-to-date references (e.g., trichinosis) while others include no references beyond 1977 (e.g., toxoplasmosis). The inadvertent transposition of photographs from the sections on ascariasis and trichiuriasis may pose problems for readers unfamiliar with these parasites.

The broad scope and in-depth treatment of the parasitic zoonoses make this an ideal reference for public health professionals, especially those concerned with epidemiology and disease prevention. It is an excellent textbook for a graduate course in parasitic zoonoses. Despite the high cost, lack of a host species index, and disparity in detail from one disease to another, it would be a useful reference for wildlife specialists particularly concerned with parasites transmitted from wildlife to humans.

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