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SHORT COMMUNICATIONS

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Pesticide Poisoning Events in Wild Birds in Korea from 1998 to 2002

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ABSTRACT: We describe cases of pesticide poisoning of wild birds diagnosed at the National Veterinary Research and Quarantine Service (Kyunggi, Korea) from 1998 to 2002. Forty-one mortality events (759 birds) of 87 incidents (2,464 birds) investigated were associated with pesticide poisoning, and six organophosphates or carbamates were identified as being responsible for the poisoning. Phosphamidon was most frequently identified as the cause of poisoning, accounting for 23 mortality events. Other pesticides identified as poisons for birds were organophosphates monocrotophus, fenthion, parathion, EPN, and diazinon, and the carbamate carbofuran. Pesticide poisoning is a problem in wild birds in Korea.

Key words: Carbamate, Korea, organophosphates, pesticide, phosphamidon, poisoning, wild birds.

Organophosphate (OP) and carbamate pesticides are used to control pests in the agricultural industry, home gardening, and veterinary medicine. These toxic compounds are capable of disrupting the avian nervous system by inhibiting cholinesterase (ChE; Ludke et al., 1975; Hill and Fleming, 1982; Quick, 1982). This short communication reports the relative likelihood of poisoning by various pesticides and gives an indication of the wild birds most affected in Korea. This report is based on wild bird mortalities submitted to the Avian Disease Division, National Veterinary Research & Quarantine Service (Anyang, Kyunggi, Korea) from 1998 to 2002 and diagnosed as pesticide poisonings.

Although some birds were submitted alive with neurologic signs such as convulsions, lethargy, and paralysis, most birds were dead. At the time of necropsy, no specific gross lesions were observed in most birds. Ingested grains were commonly found in the upper digestive tracts of waterfowl and cranes, and feathers, flesh, or other animal parts were frequently observed in the esophagus and stomach of raptors and scavengers.

OP or carbamate poisoning was confirmed by identifying pesticide residues in stomach contents and tissues—including liv-

			No. of pes	ticide poisonir	ng events		Total no.
Compound	Class	1998	1999	2000	2001	2002	of cases
Carbofuran	Carbamate	_	_	1		_	1
Diazinon	OPa	1				1	2
EPN	OP	_		2		_	2
Fenthion	OP	_	4	_			4
Parathion	OP	3				_	3
Monocrotophus	OP	_		4	2		6
Phosphamidon	OP	4	9	4	3	3	23
Total		8 (19) ^b	13 (21)	11 (19)	5(15)	4(13)	41 (87)

TABLE 1. Summary of pesticide poisoning events investigated in birds from 1988 to 2002 in Korea.

^a OP = organophosphate.

^b Confirmed cases (mortality events).

		No. of birds poi	No. of birds poisoned (no. of mortality events)	rtality events)		Total no.	No. of	-LL
Avian species	1998	1999	2000	2001	2002	or pesuciae poisoning events	burds examined	poisoned (%)
Bean goose (Anser fabalis)		3(1)				1	14	3 (21)
White-fronted goose (Anser albifrons)			4(1)		3(1)	61	27	7(26)
Baikal teal (Anas formosa)			56(1)			1	1,250	56(22.4)
Mallard (Anas platyrhynchos)	10(10)	26(2)	93(2)	73 (3)		8	278	202 (72.6)
Mandrin duck (<i>Aix galericulata</i>)		21 (2)	24(2)	99(1)		ю	185	144(77.8)
Hooded crane (Grus monachus)	11(1)				4(1)	c1	31	15(48)
Red-crowned crane (Grus japonensis)	5(1)					Г	s	5(40)
White-naped crane (<i>Grus vipio</i>)	3(1)	1 (1)			5(1)	c:	13	(69) 6
Cattle egret (Bubulcus ibis)		5(1)				1	11	5(45)
Little egret (<i>Egretta garzetta</i>)		7 (1)				1	25	7 (28)
Intermediate egret								
(Mesophoyx intermedia)		25(1)				Т	164	25(15.2)
Kamchatka gull (<i>Larus canus</i>)		91 (1)				1	197	91 (46.1)
Hill pigeon (Columba rupestris)	151(3)	12(1)			5(1)	Ŋ	250	168 (81.9)
		E t				-	ŗ	
(Nycticorax nycticorax)		(T) /				Т	Τ/	(41)
Driental stork (<i>Ciconia boyciana</i>)	1 (1)					1	1	1(100)
Golden eagle (Aquila chrysaetos)		2(1)	6(3)	3(1)		20	29	11(38)
Tawny owl (Strix aluco)			3(2)			2	6	3(33)
Total	181 (8)	200(13)	186 (11)	175 (5)	17 (4)	41	2 464	759(30.8)

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TABLE 2. Summary of avian mortality events associated with pesticides between 1998 and 2002 in Korea.

er, kidney, fat, and brain—of wild birds using gas chromatography/mass spectrometry (Hewlett Packard, Palo Alto, California USA) or high-performance liquid chromatography (Pickering, Mountain View, California). The detection limits of pesticides by these analyzers were lower than 1 ng.

Among 87 mortality events investigated from 1998 to 2002, 41 were diagnosed as being attributable to pesticide poisoning (Table 1). Six OPs or a carbamate were identified as being responsible for poisoning the wild birds. The compound most frequently identified as the cause of poisoning was phosphamidon, which accounted for poisoning in 23 of 41 cases. Other compounds identified as poisons were the OPs monocrotophus, fenthion, parathion, EPN, and diazinon and the carbamate carbofuran. More than 10 to 1,000 times the minimum toxic dose of pesticide was commonly detected in the stomach contents of wild birds.

Among a total of 394 species of wild birds found in Korea (Woo and Yoon 1989), 759 birds of 17 species were submitted to our laboratory (Table 2). The number of birds involved in each case varied from a few birds to a substantial number closely related with flock size or treated areas. There were 17 incidents involving waterfowl, such as mallards (*Anas platyrhynchos*), mandrin duck (*Aix galericulata*), and white-fronted goose (*Anser albifrons*), which were most frequently affected by pesticide poisoning.

Because OP and carbamate pesticides are typically short-lived compounds in the environment, wild bird mortality is generally closely associated with methods of pesticide application (Prijono and Leighton, 1991). The most common route of exposure to pesticides is by ingestion of poisoned insects, carcasses, or grains intentionally treated with pesticides for bait (Hill and Fleming, 1982; Quick, 1982; Reece and Handson, 1982). Furthermore, OP pesticides, which are cheap, easy to obtain, and highly toxic to birds, seem to be preferred by those who poison birds in-

tentionally. Most of the poisoning incidents summarized in this report appear to have been caused by the deliberate abuse of pesticides. In particular, wild ducks have been traditionally used in Chinese medicine in Korea. Therefore, the most frequent occurrences of pesticide poisoning in waterfowl are probably due to the intentional activity of people hunting waterfowl for use in pharmaceutical treatments. Many incidents of pesticide poisoning are due to deliberate poisoning with food (mostly rice seed) intentionally baited with these compounds. Other species affected in Korea included tawny owl (Strix aluco), cranes (Grus monachus, Grus japonensis, and Grus vipio), hill pigeon (Columba repestris), and oriental stork (Ciconia boy*ciana*). These species are known to scavenge carcasses and may be secondarily poisoned by scavenging on ducks and other birds that have died from feeding in pesticide-treated fields (Hill and Mendenhall, 1980; Henny et al., 1987).

Although we were fortunate in identifying the causative pesticides in many cases, a diagnosis of pesticide poisoning in wild birds can be difficult, and not all cases are solved. The changes in birds affected due to OP and carbamate toxicity are mostly biochemical, and gross and microscopic lesions are not present. These compounds may be undetectable in dead birds because of the rapid breakdown of the chemicals. Alternately, analysis of brain ChE activity is widely used as a diagnostic technique. However, diagnostic interpretation of ChE data always requires knowledge of the normal value for each species (Blakley and Skelley, 1988), and it may be confounded as well by changes in ChE levels caused by post mortem decomposition. Therefore, findings reported by field investigators and complete histories for avian mortality events are very important in the diagnosis of pesticide poisoning.

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