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Avian Mortality Events in the United States Caused by Anticholinesterase Pesticides: A Retrospective Summary of National Wildlife Health Center Records from 1980 to 2000

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Abstract. We reviewed the U.S. Geological Survey National Wildlife Health Center (NWHC) mortality database from 1980 to 2000 to identify cases of poisoning caused by organophosphorus and carbamate pesticides. From the 35,022 cases from which one or more avian carcasses were submitted to the NWHC for necropsy, we identified 335 mortality events attributed to anticholinesterase poisoning, 119 of which have been included in earlier reports. Poisoning events were classified as confirmed ($n = 205$) when supported by findings of ≥50% inhibition of cholinesterase (ChE) activity in brain tissue and the detection of a specific pesticide in the gastrointestinal contents of one or more carcasses. Suspected poisonings ($n = 130$) were defined as cases where brain ChE activity was ≥50% inhibited or a specific pesticide was identified in gastrointestinal contents. The 335 avian mortality events occurred in 42 states. Washington, Virginia, and Ohio had the highest frequency of events, with 24 (7.2%), 21 (6.3%), and 20 (6.0%) events, respectively. A total of 8877 carcasses of 103 avian species in 12 orders was recovered. Because carcass counts underestimate total mortality, this represents the minimum actual mortality. Of 24 different pesticides identified, the most frequent were famphur ($n = 59$; 18%), carbofuran ($n = 52$; 15%), diazinon ($n = 40$; 12%), and fenthion ($n = 17$; 5.1%). Falconiformes were reported killed most frequently (49% of all die-offs) but Anseriformes were found dead in the greatest numbers (64% of 8877 found dead). The majority of birds reported killed by famphur were Passeriformes and Falconiformes, with the latter found dead in 90% of famphur-related poisoning events. Carbofuran and famphur were involved in mortality of the greatest variety of species (45 and 33, respectively). Most of the mortality events caused by diazinon involved waterfowl.

Anticholinesterase pesticides, which include organophosphorus (OP) and carbamate (CB) compounds, have largely replaced the more environmentally and biologically persistent organochlorine pesticides and have become widely used throughout the world (Osteen 1993; Hill 1995). They poison animals by inhibiting cholinesterase (ChE) enzymes that normally break down the neurotransmitter acetylcholine (Fairbrother 1996). Acetylcholine then accumulates at nerve synapses, resulting in uninterrupted stimulation and leading to paralysis of respiratory muscles and asphyxiation (Fairbrother 1996). Wild birds may be exposed to anticholinesterase pesticides via ingestion of contaminated water, seeds or foliage, poisoned invertebrates or vertebrates, and formulated granular products (Hill 1995) and via dermal contact and inhalation (Grue *et al.* 1983). Signs of intoxication include feather fluffing, incoordination, blindness, lacrimation, lethargy, hyperexcitability, rapid and difficult breathing, tremors, and convulsions (Grue *et al.* 1991). Analysis of anticholinesterase pesticides in avian tissues is not often useful because they are rapidly excreted (Hill and Fleming 1982; Hill 1995; Fairbrother 1996). Diagnosis of anticholinesterase poisoning in birds is based on history, postmortem examination, demonstration of ≥50% inhibition of brain ChE activity, and detection of OP or CB compounds in gastrointestinal contents (Hill and Fleming 1982; Smith *et al.* 1995; Fairbrother 1996).

In the United States (U.S.), individual incidents of anticholinesterase pesticide poisoning have been reported in a diversity of wild birds, including wild turkeys (*Meleagris gallopavo*) (Nettles 1976), raptors (Henny *et al.* 1987), waterfowl and gulls (Hill and Fleming 1982), and passerines (Augspurger *et al.* 1996). Others have described multiple mortality events caused by one or more anticholinesterase compound. For example, Stone and Gradoni (1985) reported 54 avian mortality events involving diazinon, and Littrell (1988) described 22 waterfowl incidents caused by carbofuran. Grue *et al.* (1983) listed 30 documented wildlife mortality incidents involving 12 different OPs in North America between 1965 and 1983 and Franson and Smith (1999) summarized results from selected avian incidents involving 9 OPs and CBs.

Wild bird mortality due to anticholinesterase toxicity also has been documented in other parts of the world. Grue *et al.* (1983) reported finding evidence of nearly 400 incidents outside of North America, and Mineau *et al.* (1999) reported 165 incidents in raptors from 1985 to 1995 in the United Kingdom

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and Canada and 255 in the U.S. We reviewed National Wildlife Health Center (NWHC) records to identify species affected, number of dead reported, compounds involved, and pesticide use in cases of pesticide-related avian mortality submitted to the NWHC for diagnostic evaluation from 1980 through 2000.

Materials and Methods

Source of Data

The mission of the NWHC includes conducting research on wildlife health issues and providing diagnostic services to natural resource managers throughout the U.S. and its territories. Field information is gathered on wildlife mortality events, as reported by biologists and natural resource managers. Submitted carcasses are examined at necropsy, and laboratory tests, based on case history and postmortem observations, may include analysis of brain ChE activity, other toxicological evaluations, testing for botulinum toxin, microbiological cultures, parasitological examinations, and histopathological evaluations. Field and necropsy data are maintained in an electronic database that, for the period 1980 through 2000, contains 35,022 cases from which one or more avian carcasses were necropsied at the NWHC. We screened the data set for avian mortality events involving anticholinesterase compounds, examined corresponding NWHC necropsy records, and surveyed the literature for any previously published reports of these events.

ChE and Pesticide Analysis

Brains were saved frozen at −20°C until ChE analysis was performed. Brain ChE activity was measured by colorimetric assay (Ellman *et al.* 1961; Hill and Fleming 1982) at the Patuxent Wildlife Research Center (PWRC), Laurel, MD, during 1980–1985 and at NWHC from 1986 to 2000. The ChE activity was compared to normal activity for the same species, using data from individuals that died of causes other than poisoning (Hill 1988; NWHC files). If the ChE activity of the sample was two standard deviations below the mean normal activity, the brain homogenate was incubated at 37°C for 18 h and reanalyzed to determine if enzyme reactivation occurred. Reactivation, or a return toward normal brain ChE activity, is characteristic of CBs but not OPs (Martin *et al.* 1981; Smith *et al.* 1995).

In 81% of cases in which gastrointestinal contents were checked for OP or CB pesticides, the analysis was done at PWRC (later Patuxent Analytical Control Facility [PACF]) by gas chromatography/mass spectrometry (GC/MS) according to White *et al.* (1982) from 1980 to 1988 and according to PACF standard operating procedure 0–25.00 from 1989 to 2000. Another 9% of the analyses on gastrointestinal contents were done at the National Fish and Wildlife Forensics Laboratory (NFWFL), Ashland, OR, using GC/MS for OPs and high-performance liquid chromatography and GC/MS for CBs (NFWFL standard operating procedures TOX-002 and TOX-003, respectively). The remaining 10% of chemical analyses were done at the Animal Health Diagnostic Laboratory (ADHL) at Michigan State University, Lansing (Braselton *et al.* 2000), Wisconsin Central Animal Health Laboratory, Madison (US FDA 1977), Laboratory Services Division of the Oregon Department of Agriculture, Salem (Luke *et al.* 1981), Virginia Department of General Services, Division of Consolidated Laboratory Services, Richmond (Stahr 1980), and Pesticide Residue Laboratory, Biochemistry Department of Virginia Polytechnic Institute and State University, Blacksburg (Bertuzzi *et al.* 1967; Shuttleworth 1973).

Pesticide use information was classified into one of three categories: (1) *use,* if the pesticide associated with the mortality event was reported to have been used in the area shortly before the die-off; (2) *misuse,* if the case history contained evidence of pesticide-tainted bait or negligent handling of pesticide; and (3) *undetermined,* if no pesticide-related history was available.

Criteria for Confirmed and Suspected Anticholinesterase Poisonings

Confirmed anticholinesterase-poisoning events were defined as those in which the brain ChE activity of one or more birds was inhibited by ≥50% and an OP or CB compound was detected in gastrointestinal contents (Hill and Fleming 1982; Fairbrother 1996). In all but four of the confirmed poisoning events, both of these criteria were met through analyses performed on the same bird or birds; in four events, measurement of brain ChE activity and chemical analysis of gut contents were performed on samples from different individuals of the same species. In 14 carcasses in 14 instances where carbofuran (n = 13) or aldicarb (n = 1) was identified in gut contents, the brain ChE activity was not inhibited by 50%, presumably because of spontaneous reactivation (Martin *et al.* 1981; Grue *et al.* 1991) or rapid death before significant levels of CB reached brain tissue (Grue *et al.* 1991). Suspected anticholinesterase poisoning events were those in which either the brain ChE activity was depressed by ≥50% or a specific OP or CB compound was identified in a gastrointestinal sample.

Results and Discussion

The NWHC records contained information on 335 mortality events (205 confirmed and 130 suspected) caused by anticholinesterase pesticides (Table 1), 119 of which have been reported previously (see footnotes to Table 2). Although many anticholinesterase-poisoned birds had concurrent infections, parasites, or traumatic lesions, these were considered secondary in all cases except those identified. Of the 130 suspected poisonings, 89 had decreased brain ChE activity, but no chemical analysis was done on gastrointestinal contents, and 3 had OPs in gastrointestinal contents, but brain ChE activity was not measured. In 22 other suspected poisoning events brain ChE activity was depressed, but gut contents were negative for anticholinesterase compounds. In the remaining 16 events, anticholinesterase compounds were found in gut contents, but brain ChE activity was not inhibited (12 events), brain tissue was too decomposed for interpretation of ChE activity (2 events), or the brain ChE activity and gastrointestinal analyses were performed in two different species (2 events). In one of the latter cases, 28 black-billed magpies (*Pica hudsonia*), 5 bald eagles (*Haliaeetus leucocephalus*), a golden eagle (*Aquila chrysaetos*), and a northern flicker (*Colaptes auratus*) were found dead within a 3-mi radius of rabbit and deer remains. Gastrointestinal contents of two bald eagles contained 6.1 and 15 ppm wet weight of famphur, but brains were too decomposed for ChE analysis. A black-billed magpie had brain ChE inhibition of 88% that persisted upon incubation but lacked an identifiable gastrointestinal tract due to maggot infestation. The other case involved two house finches (*Carpodacus mexicanus*), a mourning dove (*Zenaida macroura*), a house sparrow (*Passer domesticus*), and an unidentified passerine. Brain ChE activity was depressed in the house finches but not in the other three birds, two of which were decomposed. Although no OPs

Table 1. Confirmed (≥50% inhibition of brain cholinesterase [ChE] activity and identification of one or more anticholinesterase compounds in gastrointestinal contents) and suspected (≥50% inhibition of brain ChE activity or identification of one or more anticholinesterase compounds in gastrointestinal contents) avian mortality events caused by anticholinesterase pesticides

Pesticide(s) involved	No. events	States in which poisonings occurred (No. events)	Minimum combined mortality
Confirmed poisoning events			
Aldicarb[a]	5	ID (2), LA (1), UT (1), WA (1)	12
Carbofuran[b]	44	AR (1), CA (2), CO (2), DE (6), FL (4), IA (1), IL (1), MD (9), MO (1), MT (3), NC (1), NM (1), OR (1), SD (2), TN (2), TX (1), VA (4), WI (2)	1752
Chlorpyrifos[c]	3	LA (1), OH (1), OR (1)	43
Coumaphos[d]	1	WY (1)	1
Diazinon[e]	34	AZ (1), ID (1), IL (3), IN (3), KS (1), MA (1), MD (1), ME (1), MN (1), MO (1), MT (1), NM (1), OH (4), PA (1), RI (1), UT (1), VA (4), WA (7)	833
Dicrotophos[f]	2	TX (2)	244
Dimethoate[g]	1	CA (1)	6
Disulfoton[h]	3	AK (1), ID (1), ME (1)	43
Famphur[i]	56	CA (2), CO (5), IA (1), ID (4), LA (1), MN (1), MO (1), MT (8), ND (1), NE (7), NM (1), NV (1), OH (2), OR (5), SD (2), UT (4), WA (4), WV (1), WY (5)	597
Fensulfothion[j]	1	OH (1)	156
Fenthion[k]	16	IA (2), ID (1), IL (2), KS (1), MN (1), MO (2), MT (1), OH (2), OK (1), OR (1), VA (1), WI (1)	342
Fonofos[l]	1	IL (1)	17
Methamidophos[m]	1	WI (1)	19
Methiocarb[n]	1	OH (1)	22
Monocrotophos[o]	4	AL (1), LA (3)	126
Oxamyl[p]	1	TN (1)	146
Parathion[q]	8	AL (1), DE (1), FL (1), MA (1), MD (1), OK (1), TX (1), WA (1)	270
Phorate[r]	9	IA (1), ID (1), MN (1), NE (1), OR (1), SD (2), VA (1), WI (1)	655
Phosphamidon[s]	1	KS (1)	6
Terbufos[t]	6	IA (1), ID (1), NE (2), OR (1), TX (1)	49
Acephate[u] and methamidophos	1	AR (1)	15
Chlorpyrifos and diazinon	2	AR (2)	22

[a] 2-Methyl-2-(methylthio)propanal *O*-[(methylamino)carbonyl]oxime.
[b] 2,3-Dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate.
[c] *O,O*-Diethyl *O*-(3,5,6-trichloro-2-pyridinyl) phosphorothioate.
[d] *O*-(3-Chloro-4-methyl-2-oxo-2*H*-1-benzopyran-7-yl) *O,O*-diethyl phosphorothioate.
[e] *O,O*-Diethyl *O*-[6-methyl-2-(1-methylethyl)-4-pyrimidinyl] phosphorothioate.
[f] (*E*)-3-(Dimethylamino)-1-methyl-3-oxo-1-propenyl dimethyl phosphate.
[g] *O,O*-Dimethyl *S*-[2-(methylamino)-2-oxoethyl] phosphorodithioate.
[h] *O,O*-Diethyl *S*-[2-(ethylthio)ethyl] phosphorodithioate.
[i] *O*-[4-[(Dimethylamino)sulfonyl]phenyl] *O,O*-dimethyl phosphorothioate.
[j] *O,O*-Diethyl *O*-[4-(methylsulfinyl)phenyl] phosphorothioate.
[k] *O,O*-Dimethyl *O*-[3-methyl-4-(methylthio)phenyl] phosphorothioate.
[l] *O*-Ethyl *S*-phenyl ethylphosphonodithioate.
[m] *O,S*-Dimethyl phosphoramidothioate.
[n] 3,5-Dimethyl-4-(methylthio)phenyl methylcarbamate.
[o] Dimethyl (*E*)-1-methyl-3-(methylamino)-3-oxo-1-propenyl phosphate.
[p] Methyl 2-(dimethylamino)-*N*-[[(methylamino)carbonyl]oxy]-2-oxoethanimidothioate.
[q] *O,O*-Diethyl *O*-(4-nitrophenyl) phosphorothioate.
[r] *O,O*-Diethyl *S*-[(ethylthio)methyl] phosphorodithioate.
[s] 2-Chloro-3-(diethylamino)-1-methyl-3-oxo-1-propenyl dimethyl phosphate.
[t] *S*-[[(1,1-Dimethylethyl)thio]methyl] *O,O*-diethyl phosphorodithioate.
[u] *O,S*-Dimethyl acetylphosphoramidothioate.
[v] *O,O*-Dimethyl *O*-(4-nitrophenyl) phosphorothioate.
[w] 2,2-Dimethyl-1,3-benzodioxol-4-yl methylcarbamate.
[x] Methyl *N*-[[(methylamino)carbonyl]oxy]ethanimidothioate.
[y] Brain ChE activity returned to normal after 18 h of incubation at 37°C; no compound detected in gastrointestinal contents (5) or no chemical analysis done (9).
[z] Brain ChE activity remained severely inhibited after 18 h of incubation at 37°C; no compound detected in gastrointestinal contents (14) or no chemical analysis done (72).
[aa] Brain ChE activity increased in some samples after 18 h of incubation at 37°C, but not to normal levels; no compound detected in gastrointestinal contents (3) or no chemical analysis done (7).

Table 1. (*Continued*)

Pesticide(s) involved	No. events	States in which poisonings occurred (No. events)	Minimum combined mortality
Chlorpyrifos and fonofos	1	OH (1)	65
Monocrotophos and fenthion	1	AZ (1)	27
Parathion and methyl parathion[v]	2	TX (2)	1604
Total	205		7072
Suspected poisoning events			
Aldicarb	1	WY (1)	1
Bendiocarb[w]	1	CT (1)	5
Carbofuran	8	CA (1), DE (1), MD (4), SD (2)	24
Diazinon	4	TX (1), VA (1), WA (2)	126
Famphur	3	CO (1), WY (2)	37
Methomyl[x]	1	ID (1)	107
Parathion	1	MA (1)	22
Terbufos	1	IA (1)	1
Unknown CB[y]	14	AR (2), CA (1), FL (1), IA (1), LA (1), MD (2), MI (1), ND (1), OH (1), VA (2), WA (1)	387
Unknown OP[z]	86	AL (1), AZ (1), CA (4), CO (2), FL (1), ID (1), IL (7), LA (1), MD (2), MN (4), MO (7), MT (6), NC (1), NE (7), NJ (1), NM (1), NV (2), OH (6), OK (1), OR (5), TX (3), UT (2), VA (8), WA (7), WI (3), WY (2)	775
Unknown OP or CB[aa]	10	FL (1), ID (1), KS (1), LA (3), NC (1), OH (1), WA (1), WI (1)	320
Total	130		1805
Grand total	335		8877

Table 2. Frequency of mortality and minimum mortality of 12 avian orders involved in 335 mortality events attributed to anticholinesterase pesticides

Order	No. events (minimum mortality)					
	Famphur	Carbofuran	Diazinon	Fenthion	All others[a]	Total[b]
Anseriformes	1 (12)	11 (1481)	34 (846)	1 (252)	65 (3134)	112 (5725)[1]
Passeriformes	11 (433)	13 (153)	9 (133)	4 (57)	42 (1037)	79 (1813)[2]
Falconiformes	53 (90)	38 (94)	0 (0)	14 (42)	60 (97)	165 (323)[3]
Columbiformes	1 (4)	3 (6)	0 (0)	2 (11)	12 (281)	18 (302)
Charadriiformes	0 (0)	4 (35)	0 (0)	0 (0)	11 (225)	15 (260)[4]
Strigiformes	1 (1)	2 (2)	0 (0)	4 (6)	5 (8)	12 (17)[5]
Ciconiiformes	1 (6)	2 (5)	0 (0)	0 (0)	4 (110)	7 (121)[6]
Gruiformes	0 (0)	0 (0)	1 (1)	0 (0)	3 (23)	4 (24)
Other[c]	1 (1)	0 (0)	1 (1)	0 (0)	4 (62)	6 (64)[7]
Unidentified orders[d]	1 (87)	0 (0)	0 (0)	1 (1)	6 (140)	8 (228)
Total[e]	59 (634)	52 (1776)	40 (981)	17 (369)	167 (5117)	335 (8877)

[a] Other anticholinesterase compounds (Table 1).

[b] Information from National Wildlife Health Center records has previously been published for 119 of these events: [1]White *et al.* (1982, 1983), Stone and Gradoni (1985), Flickinger *et al.* (1991), Franson and Smith (1999); [2]White *et al.* (1983), Flickinger *et al.* (1984), Stone and Gradoni (1985), Wenneborg (1986), Augspurger *et al.* (1996); [3]Henny *et al.* (1987), Franson (1994), Franson *et al.* (1996), Mineau *et al.* (1999); [4]Flickinger *et al.* (1984); [5]Wenneborg (1986), Franson (1994), Blus (1996), Franson and Little (1996), Franson and Smith (1999), Mineau *et al.* (1999); [6]Mineau *et al.* (1999); [7]Stone and Gradoni (1985).

[c] Galliformes, Pelecaniformes, Apodiformes, and Piciformes.

[d] Birds not identified to order, or birds of multiple orders grouped together.

[e] The total number of events attributed to a pesticide may be less than the sum of events in the column because a single event frequently involved multiple orders.

or CBs were found in house finch gut contents, bendiocarb was present in gut contents of the latter three birds, at 24, 1.3, and 8.4 ppm wet weight.

We classified five die-offs as confirmed poisonings in the absence of brain ChE control data for the affected species. In one die-off, the brain ChE activities in three fulvous whistling-ducks (*Dendrocygna bicolor*) were 3.41, 2.02, and 1.82 μmol/min/g, all less than half of the lowest mean normal brain ChE activity available for species in the family Anatidae (Hill 1988; NWHC files). The brain ChE activities measured in a turkey vulture (*Cathartes aura*) and northern harrier (*Circus cyaneus*) (6.3 and 4.4 μmol/min/g, respectively) from a second die-off, in four dark-eyed juncos (*Junco hyemalis*) (2.28, 2.66, 5.40, and 6.53 μmol/min/g) from a third die-off, and in a common

raven (*Corvus corax*) (0.6 μmol/min/g) from a fourth die-off were less than half of the lowest mean normal brain ChE activity available for species in the family Carthartidae, subfamily Accipitrinae, family Emberizidae, and genus *Corvus,* respectively (Hill 1988; NWHC files). In the final mortality event, a great-tailed grackle (*Quiscalus mexicanus*) had a brain ChE activity of 17.4 μmol/min/g, increasing to 29.2 μmol/min/g after 18 h of incubation at 37°C. Because we found 9.48 ppm wet weight of carbofuran in the great-tailed grackle gastrointestinal contents, we concluded that brain ChE had likely experienced some degree of spontaneous reactivation prior to its initial measurement (Martin *et al.* 1981; Grue *et al.* 1991).

In eight events, eight birds had concurrent diagnoses that could have resulted in death. These were a snow goose (*Chen caerulescens*) with *Aspergillus* sp. in its air sacs; a bald eagle with avian tuberculosis; two bald eagles with lead concentrations in their livers of 27 and 11 ppm wet weight, levels compatible with death in Falconiformes (Franson 1996); two bald eagles with evidence of trauma and another which was electrocuted; and a sandhill crane (*Grus canadensis*) with botulism Type C, a condition not associated with decreased brain ChE activity (Rocke and Samuel 1991).

Epidemiological Factors in Mortality Events

The 335 anticholinesterase-related avian mortality events occurred in 42 states. The states with the highest frequencies of events were Washington (24; 7.2%), Virginia (21; 6.3%), and Ohio (20; 6.0%) (Table 1). Of 24 pesticides identified, famphur ($n = 59$; 18%), carbofuran ($n = 52$; 15%), diazinon ($n = 40$; 12%), and fenthion ($n = 17$; 5.1%) were involved in the most mortality events (Table 1). Simultaneous exposure to diazinon and chlorpyrifos occurred in two events, and exposure to fenthion and monocrotophos occurred in one event.

In total, 8877 dead birds were picked up or counted during the 335 mortality events (Table 1). Dead birds included 103 species in 12 orders (Tables 2 and 3). Falconiformes and Anseriformes were reported killed most frequently (in 49 and 33% of events, respectively). Waterfowl and passerines were found dead in the greatest numbers (5725 and 1813, respectively). When the species were not identified, carcasses were included in the "unidentified" category within the correct order (Table 3). When birds were not identified to order, or when birds in multiple orders were grouped together, they were included within "unidentified orders" in Tables 2 and 3. In a number of instances, a species was identified but the number dead was not reported. In addition, records of many of the die-offs contained descriptions of greater mortality than were quantified by carcass counts or recoveries. Thus, the number dead reported here represent the minimum mortality associated with these events.

Famphur, carbofuran, diazinon, and fenthion, each of which is highly toxic for birds (Mineau *et al.* 2001), were the pesticides most frequently associated with mortality in the die-offs we reviewed, accounting for 50% of the events. Famphur was responsible for the greatest number of events (59), involving the deaths of 634 individuals of 33 species in seven orders. Falconiformes were reported killed in 90% of the famphur-related events but passerines were found dead in the greatest numbers (68% of famphur deaths), followed by Falconiformes (14% of famphur deaths) (Table 2). In 12% of famphur-related die-offs there was evidence of famphur use, which occurred primarily in Western states in association with cattle. These cases included two famphur poisoning events, described by Henny *et al.* (1987), in which bald eagles were found dead near a cow carcass in an area where cattle had been topically treated with famphur. There was evidence of pesticide misuse by baiting in 7% of famphur poisoning events. For example, famphur was used in a known attempt to kill starlings, resulting in the deaths of at least 17 birds (four passerine and three raptor species). Pesticide use was undetermined in the remaining 81% of famphur poisoning events.

Carbofuran was identified in 52 events, resulting in the reported deaths of 1776 birds of 45 species in seven orders, primarily Anseriformes (9 species; 83% of carbofuran deaths), Passeriformes (19 species; 9% of carbofuran deaths), and Falconiformes (5 species; 5% of carbofuran deaths). Falconiformes were most frequently found dead (38 events; 73% of carbofuran-related events) (Table 2). Twenty-three percent of the carbofuran die-offs were attributed to pesticide use, 29% to misuse, and 48% were undetermined. Most reported uses of carbofuran occurred in the mid-Atlantic states on corn fields. In one instance, 35 snow geese, two mallards (*Anas platyrhynchos*), a blue-winged teal (*Anas discors*), and a ring-billed gull (*Larus delawarensis*) were found dead after granular carbofuran was applied in furrows during planting of no-till corn. Misuse of carbofuran often involved baiting, where carbofuran was applied to animal carcasses, similarly to that described by Allen *et al.* (1996). For example, three American crows (*Corvus brachyrhynchos*), three northern harriers, and two red-tailed hawks (*Buteo jamaicensis*) were found dead along with several mammal carcasses, with carbofuran-tainted poultry carcasses nearby.

Diazinon was involved in 40 mortality events involving 22 avian species in four orders. Waterfowl accounted for 86% of the 981 individuals reported killed by diazinon and were among the birds that died in 34 (85%) of the diazinon-related events (Table 2). Evidence of diazinon use was available for 25% ($n = 10$) of diazinon die-offs. While some uses occurred at parks and in agricultural settings, the majority occurred in residential areas near open water. In one case, granular diazinon was applied to a lawn near two lakes, resulting in the death of 47 mallards. In another case, nine mallards were found dead on a residential lakeshore after diazinon was applied to control crane fly larvae. One case (2.5%) involved misuse of diazinon, as it was applied to a turf farm in 1998, 10 years after its use was cancelled for that application (Extoxnet 1996). Pesticide use information was not available for 29 (72.5%) of diazinon-related die-offs.

In 17 mortality events, fenthion caused the reported deaths of 369 individuals of 21 species in 5 orders. Falconiformes were involved in 82% of the fenthion-associated die-offs, but Anseriformes comprised the greatest proportion of recorded deaths (68%) (Table 2). In 18% ($n = 3$) of fenthion poisoning events there was evidence of fenthion use in pest bird control operations. For example, a mortality event occurred involving 26 European starlings (*Sturnus vulgaris*), 3 great horned owls (*Bubo virginianus*), a bald eagle, and a rock dove (*Columba livia*) in association with the use of Rid-A-Bird perches at a power plant (see Wenneborg 1986; Mineau *et al.* 1999). Evi-

Table 3. Number of carcasses picked up or counted during 335 mortality events attributed to anticholinesterase pesticides

Common name	Scientific name	Minimum dead
Anseriformes		
Wood duck	*Aix sponsa*	9
Northern pintail	*Anas acuta*	1068
American wigeon	*Anas americana*	158
Green-winged teal	*Anas crecca*	212
Blue-winged teal	*Anas discors*	14
Mottled duck	*Anas fulvigula*	1
Pekin duck	*Anas platyrhynchos*	21
Mallard	*Anas platyrhynchos*	1230
Mallard hybrid	*Anas platyrhynchos*	50
Gadwall	*Anas strepera*	8
Greater white-fronted goose	*Anser albifrons*	21
Lesser scaup	*Aythya affinis*	122
Ring-necked duck	*Aythya collaris*	30
Canada goose	*Branta canadensis*	2160
Bufflehead	*Bucephala albeola*	1
Muscovy duck	*Cairina moschata*	8
Snow goose	*Chen caerulescens*	63
Ross's goose	*Chen rossii*	6
Black-bellied whistling-duck	*Dendrocygna autumnalis*	20
Fulvous whistling-duck	*Dendrocygna bicolor*	24
Unidentified Anseriformes		499
Passeriformes		
Red-winged blackbird	*Agelaius phoeniceus*	260
Cedar waxwing	*Bombycilla cedrorum*	160
Northern cardinal	*Cardinalis cardinalis*	40
Pine siskin	*Carduelis pinus*	16
American goldfinch	*Carduelis tristis*	107
House finch	*Carpodacus mexicanus*	43
American crow	*Corvus brachyrhynchos*	18
Common raven	*Corvus corax*	14
Blue jay	*Cyanocitta cristata*	11
Rusty blackbird	*Euphagus carolinus*	1
Brewer's blackbird	*Euphagus cyanocephalus*	11
Common yellowthroat	*Geothlypis trichas*	1
Barn swallow	*Hirundo rustica*	1
Dark-eyed junco	*Junco hyemalis*	26
Swamp sparrow	*Melospiza georgiana*	1
Song sparrow	*Melospiza melodia*	1
Brown-headed cowbird	*Molothrus ater*	74
House sparrow	*Passer domesticus*	57
Black-billed magpie	*Pica hudsonia*	83
Black-capped chickadee	*Poecile atricapilla*	1
Vesper sparrow	*Pooecetes gramineus*	2
Prothonotary warbler	*Protonotaria citrea*	1
Boat-tailed grackle	*Quiscalus major*	141
Great-tailed grackle	*Quiscalus mexicanus*	45
Common grackle	*Quiscalus quiscula*	214
Eastern bluebird	*Sialia sialis*	2
American tree sparrow	*Spizella arborea*	3
Field sparrow	*Spizella pusilla*	1
Eastern meadowlark	*Sturnella magna*	5
Western meadowlark	*Sturnella neglecta*	12
European starling	*Sturnus vulgaris*	318
Tree swallow	*Tachycineta bicolor*	2
Curve-billed thrasher	*Toxostoma curvirostre*	1
Brown thrasher	*Toxostoma rufum*	3
American robin	*Turdus migratorius*	43
Yellow-headed blackbird	*Xanthocephalus xanthocephalus*	1
White-throated sparrow	*Zonotrichia albicollis*	2
White-crowned sparrow	*Zonotrichia leucophrys*	2
Unidentified Passeriformes		89
Falconiformes		
Cooper's hawk	*Accipiter cooperii*	1
Sharp-shinned hawk	*Accipiter striatus*	1
Golden eagle	*Aquila chrysaetos*	26
Red-tailed hawk	*Buteo jamaicensis*	79
Rough-legged hawk	*Buteo lagopus*	1
Ferruginous hawk	*Buteo regalis*	1
Swainson's hawk	*Buteo swainsoni*	1
Northern harrier	*Circus cyaneus*	18
Merlin	*Falco columbarius*	1
Prairie falcon	*Falco mexicanus*	1
Peregrine falcon	*Falco peregrinus*	6
American kestrel	*Falco sparverius*	3
Bald eagle	*Haliaeetus leucocephalus*	158
Mississippi kite	*Ictinia mississippiensis*	14
Unidentified Falconiformes		12
Columbiformes		
Rock dove	*Columba livia*	4
Inca dove	*Columbina inca*	1
Mourning dove	*Zenaida macroura*	297
Charadriiformes		
Dunlin	*Calidris alpina*	10
Least sandpiper	*Calidris minutilla*	1
Semipalmated sandpiper	*Calidris pusilla*	1
Semipalmated plover	*Charadrius semipalmatus*	1
Killdeer	*Charadrius vociferus*	5
Black tern	*Chlidonias niger*	62
Common snipe	*Gallinago gallinago*	2
Herring gull	*Larus argentatus*	59
Laughing gull	*Larus atricilla*	16
Ring-billed gull	*Larus delawarensis*	29
Caspian tern	*Sterna caspia*	4
Forster's tern	*Sterna forsteri*	1
Unidentified Charadriiformes		69
Strigiformes		
Great horned owl	*Bubo virginianus*	13
Barred owl	*Strix varia*	2
Unidentified Strigiformes		2
Ciconiiformes		
Great egret	*Ardea alba*	38
Great blue heron	*Ardea herodias*	3
Cattle egret	*Bubulcus ibis*	6
Turkey vulture	*Cathartes aura*	4
Black vulture	*Coragyps atratus*	65
Snowy egret	*Egretta thula*	2
Glossy ibis	*Plegadis falcinellus*	2
Unidentified Ciconiiformes		1
Gruiformes		
American coot	*Fulica americana*	11
Sandhill crane	*Grus canadensis*	13
Galliformes		
Greater sage-grouse	*Centrocercus urophasianus*	50
Northern bobwhite	*Colinus virginianus*	2
Wild turkey	*Meleagris gallopavo*	1
Pelecaniformes		
Brown pelican	*Pelecanus occidentalis*	9
Apodiformes		
Unidentified Apodiformes		1
Piciformes		
Northern flicker	*Colaptes auratus*	1
Unidentified orders		228
Total		8877

dence of misuse by baiting was present in 12% ($n = 2$) of fenthion die-offs, and pesticide use was undetermined in 70% ($n = 12$) of the events. Of the 33 die-offs involving the next four most frequently diagnosed pesticides, parathion ($n = 11$), phorate ($n = 9$), terbufos ($n = 7$), and aldicarb ($n = 6$), evidence of pesticide use existed for 3 events (9%), each involving parathion, 5 events (15%) were categorized as misuse (aldicarb, $n = 2$; parathion, $n = 1$; and phorate, $n = 2$), and 25 events (76%) were undetermined.

Regulatory changes occurred for at least three chemicals that we found to be associated with avian mortality during 1980–2000. Beginning in 1991, most uses of granular carbofuran were phased out, and in 1988 the use of diazinon on golf courses and sod farms, and all uses of monocrotophos, were cancelled (Extoxnet 1996; US EPA 2000). We cannot use the frequency of poisonings in the NWHC database to critically evaluate the effect of these regulatory changes on avian mortality because: (1) we have little information on pesticide use (use information was unavailable for 48 and 72.5% of the carbofuran and diazinon cases, respectively); (2) sample sizes are small (*e.g.*, a total of only five cases of monocrotophos poisoning is present in the database); and (3) carcass submissions do not constitute a statistically representative sample of avian mortality because they are dependent on the level of use of NWHC's diagnostic services, which varies geographically and temporally according to current contaminant and disease issues. Thus, although the number of carbofuran cases before and after 1991 were similar, and there were more diazinon cases after 1988 than before, we cannot infer a relationship between these observations and the regulatory changes.

Limitations of Mortality Observations in Estimating Pesticide Effects

The true magnitude of avian mortality associated with the events we have reported here was probably considerably greater, because a variety of factors prevents complete and accurate counts of the total number dead in wildlife die-offs. Pesticide-related mortality events may go altogether undetected, particularly if few animals are involved, and carcass counts may be affected by the presence of scavengers that remove dead wildlife, the tendency of sick wildlife to seek cover, the intensity of searches, carcass morphology (large or colorful species are more likely to be noticed than small or less colorful species) and state of decomposition, and vegetative structure that camouflages carcasses or blocks the vision of searchers (Vyas 1999). Flocking species, such as waterfowl and blackbirds, may be more likely to be observed than more solitary species, such as raptors. Even when mortality is observed, it may not be reported to appropriate wildlife agencies, the cause of death may not be confirmed, or delays in reporting or response may prevent an accurate evaluation of the event (Vyas 1999). Furthermore, the level of brain ChE inhibition chosen as an indicator of poisoning will affect the number of birds reported as having died of anticholinesterase poisoning. An inhibition of 50% or greater is commonly accepted as the critical level (Fairbrother 1996), but free-living birds may die with less brain ChE inhibition because of stressors encountered in the wild (Grue *et al.* 1991).

A determination of the magnitude of the impact of anticholinesterase pesticides on wildlife is also complicated by the occurrence of pesticide-associated sublethal effects that may affect populations. Grue *et al.* (1997) reviewed the literature for evidence of neurophysiological and behavioral changes in thermoregulation, food consumption, and reproduction in wildlife exposed to OP and CB pesticides. They summarized studies indicating that pesticide-induced hypothermia is often associated with brain ChE depression of 50% or greater, with body temperature usually returning to normal within 24 h, and that pesticide toxicity is enhanced by exposure to cold in some instances. Physical signs, such as diarrhea, convulsions, and vomiting, are often seen in animals acutely exposed to OPs and CBs and later reductions in consumption of clean feed may be due to anorexia as a result of clinical signs or conditioned aversion, which may occur after toxin ingestion (Grue *et al.* 1997; Nicolaus and Lee 1999). Sublethal pesticide exposure may affect reproduction in a variety of ways, including interference with migration and sexual behavior, reduction in egg laying, and impacts on nest attentiveness and incubation behavior leading to decreased hatching success (Grue *et al.* 1997). Although conditioned taste aversion may decrease subsequent exposure to a pesticide, subsequent avoidance of untainted prey and food items disrupts foraging and endangers breeding efficiency (Nicolaus and Lee 1999). Sublethal exposure to anticholinesterase pesticides may also negatively affect avoidance behaviors and activity budgets. House sparrows exposed to fenthion were 12 times more likely to be captured by American kestrels (*Falco sparverius*) than unexposed sparrows, probably because of behavioral changes making the exposed birds more conspicuous to kestrels (Hunt *et al.* 1992). Acute doses of chlorfenvinphos in birds affected activity (such as flying and singing), posture (possibly indicating incoordination), and seed-handling efficiency (resulting in weight loss even with abundant food in laboratory conditions), and these effects were correlated with brain ChE activity (Hart 1993; Fryday *et al.* 1994).

Summary and Conclusions

From 1980 through 2000, avian carcasses from 335 anticholinesterase-pesticide poisoning events were submitted to the NWHC for necropsy. A total of 8877 avian carcasses of 103 species in 12 orders was recovered during these mortality events. Because avian carcass counts underestimate total mortality and sublethal effects of anticholinesterase pesticides may make birds more susceptible to death by other causes, the carcass numbers listed in our report represent the minimum actual avian mortality that occurred in these events. Falconiformes were found dead in more die-offs than any other order and were reported killed in the majority of cases associated with famphur, carbofuran, and fenthion. Of 24 pesticides identified, famphur, carbofuran, diazinon, and fenthion were the most frequently involved. Waterfowl were recovered in the greatest numbers and in the majority of die-offs caused by diazinon. A wider variety of bird species was reported killed by carbofuran and famphur than by diazinon. Overall, 14% of mortality events were associated with pesticide use, 10% with misuse, and 76% had undetermined use. Famphur poisoning

was frequently associated with pesticide use in cattle operations in Western states, carbofuran poisoning often occurred in association with its use on corn fields in mid-Atlantic states, and diazinon poisonings took place more frequently in residential areas near open water. Mortality events involving the use of fenthion occurred in association with bird control activities. Misuse by baiting occurred with famphur, carbofuran, and fenthion. A prohibited use of diazinon occurred in one event. Mortality events were associated with the use and misuse of parathion and the misuse of aldicarb and phorate. The retrospective nature of the study and the variability in the depth to which the cases were investigated over the years probably account for the high frequency of unknown pesticide use. The data presented here are the result of voluntary field reports, not random sampling, thus preventing us from drawing conclusions regarding geographic or year-to-year variations of mortality events.

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