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SECONDARY POISONING OF EAGLES FOLLOWING INTENTIONAL POISONING OF COYOTES WITH ANTICHOLINESTERASE PESTICIDES IN WESTERN CANADA

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ABSTRACT: Records of eagles, coyotes (*Canis latrans*), and red foxes (*Vulpes vulpes*) necropsied at the Western College of Veterinary Medicine, Saskatoon, Saskatchewan, Canada, between 1967 and 2002 were reviewed for cases suggestive of anticholinesterase poisoning. From 1993 to 2002, 54 putative poisoning incidents involving 70 bald eagles (*Haliaeetus leucocephalus*) and 10 golden eagles (*Aquila chrysaetus*) were identified. Of these, 50 incidents occurred in Saskatchewan, two were in Manitoba, and one occurred in each of Alberta and the Northwest Territories. The diagnosis was confirmed in eight instances by demonstration of pesticide in ingesta from eagles or known use of pesticide at the site together with brain cholinesterase (AChE) reduction of >50% in at least one animal. A presumptive diagnosis of poisoning was made in 33 incidents based on brain AChE reduction of >50% in at least one animal; 13 incidents were considered suspicious because of circumstantial evidence of the death of eagles in association with other species and limited AChE reduction. Other wild species were found dead in 85% of the incidents involving eagles. Coyotes, foxes, black-billed magpies (*Pica pica*), and striped skunks (*Mephitis mephitis*) were associated with 34, six, six, and three incidents, respectively. There were eight additional incidents that did not involve eagles in which poisoning was diagnosed in coyotes. Carbofuran was identified in nine incidents. Carbamate poisoning was indicated on the basis of reactivation of brain AChE activity in two additional incidents. Brain AChE activity was not reduced from normal in eagles in four of seven incidents in which carbofuran was identified. The organophosphorous insecticide terbufos was found together with carbofuran in one incident. Brain AChE activity was measured in wild canids and in eagles in 15 incidents; in all of these incidents, brain AChE was reduced by >50% in at least one mammal, whereas this level of reduction occurred in eagles in only four incidents. Use of anticholinesterase pesticides to poison coyotes is illegal, but the practice continues and secondary poisoning of eagles is a problem of unknown proportions in western North America.

Key words: *Aquila chrysaetus, Canis latrans,* carbofuran, *Haliaeetus leucocephalus,* pesticide poisoning, *Vulpes vulpes.*

INTRODUCTION

Historically, pesticide poisoning of raptors was associated with organochlorine compounds. As these were replaced by less persistent, but often more acutely toxic organophosphorous and carbamate compounds, secondary poisoning of raptors with anticholinesterase compounds has been reported with increasing frequency (Balcom, 1983; Elliot et al., 1996, 1997; Mineau et al., 1999; Hosea et al., 2001). Poisoning of eagles secondary to illegal use of carbofuran (2,3-dihydro-2,2-dimethy-7-benzo-furanyl methyl carbamate) to poison coyotes (*Canis latrans*) in Kansas in 1992 was reported by Allen et al. (1996). At about the same time, specimens with a history similar to those reported in Kansas began to be submitted for necropsy to the Canadian Cooperative Wildlife Health Centre in Saskatoon, Saskatchewan. This paper reviews poisoning of eagles and wild mammalian carnivores between 1967 and 2002 and documents what we believe to be a serious and ongoing cause of mortality for scavenging birds of prey.

TABLE 1. Reference brain cholinesterase activity, expressed in micromoles of acetylthiocholine iodide hydrolyzed per minute per gram of tissue (wet weight) at 25 C, for the species described in the text, together with various levels of reduction that have been proposed as indicative of exposure to or poisoning by anticholinesterase compounds.

Species	Normal reference value mean±SD (*n*)[a]	25% reduction[b]	Reduced >2 SD from mean[c]	50% reduction[d]
Bald eagle	17.21±3.18 (29)	12.0	10.9	8.6
Golden eagle	13.37±0.90 (4)	10.0	11.50	6.7
Black-billed magpie	19.5 (1)	14.8		9.8
Coyote	2.71±0.49 (6)	2.0	1.73	1.4
Red fox	9.63 (1)	7.2		4.8
Striped skunk	5.42±1.15 (4)	4.1	3.12	2.7

[a] From Blakley and Yole (2002).
[b] Indicative of exposure to a cholinesterase-inhibiting compound (Friend and Franson, 1999).
[c] Diagnostic threshold for cholinesterase-inhibiting compounds (Augsberger et al., 1996).
[d] Diagnostic of death from exposure to an anticholinesterase compound (Hill and Fleming, 1982; Fairbrother, 1996; Friend and Franson, 1999).

MATERIALS AND METHODS

Diagnostic files of the Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan, Canada, from 1967 through 1990 and the database of the Western and Northern Regional Centre of the Canadian Cooperative Wildlife Health Centre, Saskatoon, Saskatchewan, Canada, from 1991 through 2002 were searched for records of bald eagles (*Haliaeetus leucocephalus*), golden eagles (*Aquila chrysaetus*), coyotes, and foxes (*Vulpes vulpes*) examined at necropsy from western and northern Canada. Animals that met any of the following criteria were selected for further study: 1) eagles that had been observed feeding on a carnivore carcass or found in the immediate vicinity of dead carnivores (coyote, fox, or striped skunk [*Mephitis mephitis*]); 2) eagles with carnivore fur in their talons and/or carnivore tissue including fur in the upper digestive tract at the time of death; 3) animals found in the vicinity of bait believed to have been placed for carnivores or with putative bait in the upper digestive tract at the time of death; 4) animals found with dead avian scavengers (black-billed magpie [*Pica hudsonia*], common raven [*Corvus corax*], or gull [*Larus* sp.]) or with plumage of these birds in their digestive tract at the time of death; 5) and/or animals found dead with depressed brain cholinesterase (AChE) activity and with no alternate explanation for death.

Most suspected poisoning incidents occurred in late autumn, winter, or early spring, so many specimens were or had been frozen prior to submission. The interval between death and collection of animals was unknown in most cases. Animals were thawed at room temperature prior to necropsy. Because examinations were done over an extended period by 11 pathologists, methods varied somewhat; however, multiple tissues were collected and processed for routine bacteriology and histopathology from all eagles, except for those with advanced autolysis, and from most wild carnivores. Brain tissue removed from animals was frozen at −20 C prior to AChE analysis. A modification (Blakley and Yole, 2002) of the Ellman procedure (Ellman et al., 1961) was used to measure brain AChE activity. Results were expressed in micromoles of acetylthiocholine iodide hydrolyzed per minute per gram of tissue (wet weight [$\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue]) at 25 C. Activity was compared with normal animals of the same species previously examined in the laboratory (Table 1). Esophageal or stomach contents from eagles involved in six incidents in 1994 and 1995, and from a coyote involved in an incident in 1995 in which no eagles were detected, were examined by Zenon Corporation, Burnaby, British Columbia, Canada, under contract to the Canadian Wildlife Service using multi-residue pesticide analysis that examined for the potential presence of 124 organic compounds (pesticides and metabolites). The laboratory was accredited by the Canadian Association of Environmental Analytical Laboratories and used standard methodologies (Greenberg et al., 1992; US Environmental Protection Agency, 1992).

The history, diagnostic results, and conclusions by the pathologists were used to categorize incidents in one of three diagnostic classes. A diagnosis was considered "confirmed" when anticholinesterase pesticide was found in the alimentary tract content of animals or when it was known that such a compound had been

TABLE 2. Occurrence of incidents diagnosed as anticholinesterase poisoning of eagles between 1993 and 2002.

Year	Poisoning incidents (*n*)	Bald eagle (*n*)	Golden eagle (*n*)	Location[a]
1993	1	2		SK
1994	2	2		SK
1995	10	18		SK
1996	4	1	4	SK
1997	1	4		SK
1998	2	2		SK
1999	5	5		SK (4), NT (1)
2000	9	11	1	SK
2001	9	14	1	SK
2002	11	11	4	SK (8), MB (2), AB (1)
Total	54	70	10	

[a] SK = Saskatchewan; NT = Northwest Territories; MB = Manitoba; AB = Alberta.

placed in baits where animals were found dead, together with >50% reduction in brain AChE activity in at least one animal from the site. A diagnosis was considered "presumptive" when brain AChE activity was reduced by >50% in one or more animals from the site. The category "suspicious" was used for incidents in which eagles were found dead in association with dead mammalian carnivores or scavenging birds but in which brain AChE activity was not reduced by >50% in animals tested. We used reduction of brain AChE activity by >50% as the diagnostic threshold for anticholinesterase poisoning. This is the most conservative of several values proposed for diagnosis (Table 1).

In selected instances, AChE activity was measured after chemical reactivation using pyridine-2-aldoxime methochloride (2-PAM) and reactivation after gel filtration and dilution (Westlake et al., 1981). Stomach contents of a few birds were examined for the presence of strychnine following extraction with chloroform and dilute sulfuric acid using ultraviolet spectrophotometric techniques (Blakley, 1984). Lead content of the liver of several birds was determined by atomic absorption flame spectrophotometry at 219 nm following dry ashing of the tissue (Kim et al., 1990).

RESULTS

Only one instance of anticholinesterase poisoning was found prior to 1991. In 1987, a golden eagle was poisoned after consuming a black-billed magpie that died in association with the use of an organophosphorous compound to treat cattle for ectoparasites. The first indication that a "new" poison might have been used to kill wild carnivores occurred in 1991. On 13 May of that year, portions of a sheep carcass found with four dead coyotes near Outlook, Saskatchewan (51°30′N, 107° 03′W), were submitted to the laboratory. The sheep tissue tested negative for strychnine but was not examined for the presence of insecticides; the coyotes were not submitted to the laboratory. On 24 May 1991, two golden eagles were found dead within 1 m of a scavenged fox carcass near Killdeer, Saskatchewan (49°18′N, 106°13′W). The eagles' stomach content did not contain strychnine; brains were unsuitable for AChE testing because of desiccation. (These incidents were not included in subsequent analyses.) No incidents that fitted the criteria for inclusion were received in 1992. Specimens from 54 putative poisoning incidents involving 80 eagles were examined between 1993 and 2002 (Table 2). No explanation for death of the eagles or other species involved in these events was identified through necropsy, histopathology, or bacterial culture. Strychnine or lead were not identified in the cases tested for these poisons.

Incidents were located primarily in Saskatchewan and were widely distributed through the southern part of the province. There were peaks of occurrence of incidents in April and December (Fig. 1). Eagles were found on cropland, in pastures,

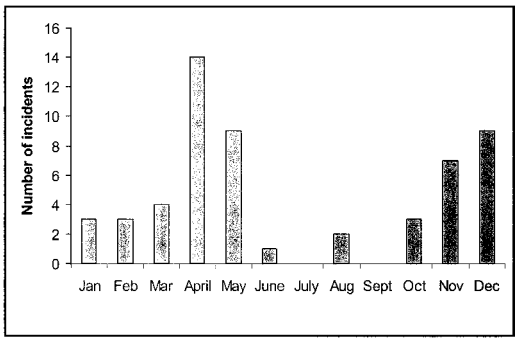

FIGURE 1. Seasonal distribution of anticholinesterase poisoning incidents involving eagles in western Canada between 1993 and 2002.

on a lake shore, near beehives, near a pulp mill, and along a road on 31, six, two, two, one, and one occasion(s), respectively, of 44 incidents in which habitat was described. Of 68 bald eagles for which data were available, 66% (45) had adult plumage (30 males, 14 females, one gender not recorded), 18 were after-hatch-year with subadult plumage (four males, 12 females, two gender not recorded), and five were hatch-year birds (two males, three females, one gender not recorded). Six of 10 golden eagles were adult (two males, four females) and four were of unknown age (one male, two females, one gender not recorded). Clinical signs in six eagles that were alive when discovered included weakness, inability to fly, outstretched wings, regurgitation or salivation, opisthotonus, and clenched feet. Postures in dead birds included sitting or lying with the wings spread or cupped, talons clenched and often containing fur or vegetation, and the head and neck in opisthotonus or tipped forward with the tail raised. Body condition was recorded for 76 eagles of which 94% (71) had substantial fat reserves, two were in moderate body condition, and three were in poor body condition. The esophagus contained food in 86% of eagles and was often distended with up to 400 g of undigested tissue.

Other wild species were associated with sick or dead eagles in 46 incidents. Coyotes occurred in 63% (34/54) of incidents. In 22 instances, eagles were found with dead coyotes; in 12 other incidents, coyote fur was found clenched in the talons of eagles and/or coyote fur and tissues were identified in the oral cavity or esophagus at necropsy. Dead foxes, magpies, skunks, and ravens were found together with eagles in six, six, three, and two events, respectively. A red-tailed hawk (*Buteo jamaicensis*), an unidentified hawk, and an unidentified gull were each associated with one event. At eight sites, more than one other wild species was found dead with eagles. At one site, a domestic dog died within 20 min after visiting the area near beehives where a bald eagle, a skunk, and a magpie were found dead. In eight incidents no association with other wild species was recorded; in six of these, eagles had material suggestive of bait in their esophagus. In three instances, the material was chicken flesh and feathers, in two it was fish, and in one it was unidentified mammalian flesh.

Poisoning of eagles by insecticides was confirmed by chemical analysis in eight incidents (Table 3). Carbofuran was identified in each case. The metabolite 3-hydroxycarbofuran was detected together with carbofuran in four incidents. The organothiophosphorus insecticide terbufos (S-tert-butylthiomethyl 0,0-diethyl phosphorodithioate) was found in addition to carbofuran in one incident. No other pesticide was detected. In three other confirmed poisonings, a person admitted to authorities that carbofuran was placed in bait at the site. In four of seven events in which only carbofuran was identified, brain AChE activity in the eagle(s) examined was not reduced from normal, although coyotes from three of these sites had >50% reduction in brain AChE.

A presumptive diagnosis of anticholinesterase poisoning, based on reduction of brain AChE by >50% in at least one animal, was made in 33 incidents. Brain AChE activity was measured in both eagles and coyotes in 13 incidents, and in two incidents brain AChE was measured

TABLE 3. Features of eight instances of confirmed anticholinesterase poisoning involving eagles in Saskatchewan.

Year	Location	Species[b] (*n*)	Brain AChE activity[a]		Pesticide[c]	Other
			Eagle(s)	Other species		
1994	49°20′N 102°37′W	BE (2)	2.47, 7.45		11 ppm carbofuran	Chicken flesh in eagle's crop
1995	49°35′N 102°45′W	BE, F	26.4		1.6 ppm carbofuran	
1995	52°58′N 108°02′W	BE, C	8.24		6.4 ppm carbofuran	
1995	52°07′N 106°38′W	BE (3), C, M	19.2, 20.3	1.29 (C)		Carbofuran placed in bait
1995	51°05′N 104°57′W	BE (5), C (2)	7.81, 6.21, 7.82, 7.74		5.1 ppm terbufos, 0.14 ppm carbofuran	
1995	51°36′N 107° 9°W	BE (2), C, F, S, M	18.4, 16.5	0.74 (C)	2.7 ppm carbofuran	
1995	51°25′N 107°20′W	BE (2), C (9), F (7), S (4)	19.2, 20.3	1.29 (C)		Carbofuran placed in pig carcass
2000	49°55′N 109°27′W	BE, S, M, D		0.85 (S), 0.79 (S), 19.5 (M)		Carbofuran placed in eggs near beehives

[a] Micromoles of acetylthiocholine iodide hydrolyzed per minute per gram of tissue (wet weight) at 25 C.
[b] AChE = cholinesterase; BE = bald eagle; C = coyote; F = fox; M = black-billed magpie; S = striped skunk; D = domestic dog.
[c] Detected in ingesta in upper alimentary tract of eagle.

in an eagle and a fox. In all 15 incidents, brain AChE activity was inhibited by >50% in one or more mammal, whereas brain activity was inhibited by >50% in eagles in only four incidents. Brain AChE was inhibited by >2 SD from the reference mean in eagles in two other incidents, and by >25% in one incident.

In six of 13 incidents considered suspicious for anticholinesterase poisoning, based on finding dead eagles together with other dead wild animals, brain AChE of eagles or coyotes was depressed by >2 SD but <50% of the reference mean.

In addition to the 54 incidents in which eagles were found dead, there were eight incidents in which coyotes were found dead with no mention of eagles in the record. Brain AChE activity in coyotes in these incidents ranged from 0.53 to 0.97 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue (approximately 60–80% reduction from normal; Table 1). Carbofuran (24 ppm) and 3-hydroxycarbofuran (0.07 ppm) were detected in the stomach content of a coyote found dead in 1995; brain AChE activity in this animal was 0.92 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue. A magpie found with a coyote at one site had brain AChE activity of 1.6 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue (about 8% of normal), and two ravens found with a coyote at another site had brain AChE activity of 1.78 and 1.74 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue, respectively. We have no reference value for ravens, but the activity in these birds was much less than reference values for other corvids (magpie [19.5 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue; Blakley and Yole, 2002]; American crow [*Corvus brachyrhynchos,* 20±3.4 $\mu mol \cdot min^{-1} \cdot g^{-1}$; Hill, 1988]).

The following examples illustrate two situations in which poisoning was recognized. Between 24 and 27 November 1997, 12 dead coyotes and one dead and one live bald eagle were found within 600 m of a cow carcass at Braddock Reservoir, Saskatchewan (50°05′N, 107°22′W). The live eagle's wings and tail were spread,

there was froth and meat in its beak, and its talons were clenched; it died shortly after discovery. The dead eagle was 25 m from a scavenged coyote. The eagles and 11 coyotes were examined. The eagles were adults (male, female) in excellent body condition, with large amounts of flesh and intestines in their esophagus. Brain AChE activity of the dead eagle was 4.32 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue. After 2-PAM and gel filtration reactivation, brain AChE activity was 8.3 and 11.0 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue, respectively. Substantial reactivation following gel filtration alone was suggestive for carbamate involvement. Brain AChE activity in the eagle found alive was 4.85 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue. Six coyotes had been consumed partially by scavengers, and five had identifiable stomach content (hide, rumen, omasum) compatible with having fed on the cow carcass. Brain AChE activity in the two coyotes tested was 1.19 and 0.94 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue, respectively. There was no appreciable reactivation after treatment of the first of these brains with 2-PAM, but the AChE activity was 8.92 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue after gel filtration. The results were interpreted as presumptive of poisoning with a carbamate pesticide (Westlake et al., 1981; Smith et al., 1995). The following spring, two bald eagles and a coyote were found on the melting ice of the reservoir. The eagles were adult females in good body condition with crops filled with decomposed mammalian viscera. Their brain AChE activity was 10.1 and 13.3 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue. The coyote found was in good condition and had brain AChE activity of 1.58 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue. A presumptive diagnosis of carbamate poisoning was made.

On 1 December 1999, an adult male bald eagle was found 3 m from a scavenged fox carcass near Fairy Glen, Saskatchewan (53°03′N, 104°33′W). The eagle was "hunched over" with fluid from its beak and fox fur clenched in its talons. It was in moderate body condition, with intestines, flesh, and fox fur in its esophagus. The fox was in moderate body condition; the abdominal and thoracic viscera were missing. Brain AChE activity of the eagle and fox were 3.33 and 0.89 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue, respectively. This was classed as a presumptive diagnosis of anticholinesterase poisoning.

DISCUSSION

Fairbrother (1996, p. 56) stated, "... anticholinesterase poisoning should be considered as part of the differential diagnosis whenever animals are found dead in otherwise good condition, i.e., significant amounts of body fat, ingesta present in the gastrointestinal tract, normal parasite load, and necropsy lesions are unremarkable." The eagles and mammals in the incidents reported here, except for three eagles in poor nutritional condition, fit this profile and no alternate explanation for their deaths was identified. Evidence, including the circumstances in which animals were found, brain AChE activity, and pesticide analysis, were used to arrive at diagnoses. We believe that the criteria for including incidents for consideration and for diagnosis are conservative, especially because a carbamate insecticide, carbofuran, was found to be responsible for most cases in which a pesticide was identified. Additional unexplained deaths of eagles and coyotes that fitted the general profile but lacked supporting evidence were excluded from the review.

Based on the common association between poisoned eagles and coyotes, we believe that most eagle deaths were secondary to intentional illegal poisoning of coyotes, as described by Allen et al. (1996), although eagles may have fed directly on poisoned baits in some situations. In two incidents associated with beehives, the "target" may have been black bears (*Ursus americanus*) or skunks, rather than coyotes. Secondary poisoning of raptors usually results from ingestion of unassimilated pesticides in the alimentary tract of the primary victim (Hill, 1995; Mineau et al.,

1999). In many incidents, abdominal viscera were missing from coyote and fox carcasses from which relatively little muscle had been removed by scavengers. Both carbofuran and terbufos are readily available insecticides in western Canada. Carbofuran, the most commonly identified compound, is very poisonous for mammals and "has one of the highest recorded toxicities to birds of any pesticide registered in Canada" (Mineau, 1993, p.1). Birds and mammals usually die within a few minutes of exposure or recover quickly with little evidence of toxicity (Hill, 1995). Secondary poisoning of raptors has been documented where carbofuran was used on crops (Mineau, 1993; Elliot et al., 1996), and the compound has been used for deliberate poisoning of raptors (Mineau, 1993; Mineau et al., 1999).

Brain AChE activity is used commonly to identify anticholinesterase poisoning, but interpretation is difficult for specimens collected in the field because chemicals have a variable effect on AChE, and the level of exposure, time period, and conditions between death and specimen analysis are highly variable. The interval and conditions between death and submission were unknown for most animals in this review; however, most specimens were collected during cool to cold weather, and many were frozen when they reached the laboratory. It is unlikely that brain AChE activity would have decreased as a result of decomposition in these animals, because brain AChE is very stable for up to 12 days of postmortem decomposition at temperatures up to 25 C (Burn and Leighton, 1996), but spontaneous reactivation may have occurred. Interpretation of brain AChE activity is particularly problematic when carbamates are involved because birds may die from high levels of exposure resulting in rapid death from neuromuscular blockage, which may prevent significant penetration of the central nervous system (Westlake et al., 1981), and because binding of carbamates to AChE can reverse spontaneously (Martin et al., 1981; Hill, 1989). Spontaneous reactivation of brain AChE postmortem has been documented in the laboratory (Hill, 1989) and in poisoning cases involving carbamates (Mineau and Tucker, 2002a,b). Birds poisoned by carbamates may have little or no reduction in brain AChE. American wigeon (*Anas americana*) collected "promptly after death" from a field sprayed with carbofuran had significantly lower brain AChE activity than wigeon found dead a day later, and only one of six wigeon examined had brain AChE activity reduced by >50% (Hill and Fleming, 1982). Only three of six red-winged blackbirds (*Agelaius phoenecius*) that died after exposure to carbofuran had any reduction in brain AChE; the maximum depression was 48% of normal, and the bird with the highest concentration of carbofuran in gut content had no AChE depression (Augsberger et al., 1996). This pattern was evident in eagles known to have ingested carbofuran (Table 3). Based on the 15 incidents in which both eagles and canids were examined from a site, brain AChE activity in coyotes and foxes appears to be a more reliable indicator of poisoning than AChE activity in secondary avian victims.

Under ideal conditions, normal animals of the same species and age that have been exposed to the same conditions between death and analysis should be used as controls for diagnostic cases; however, this was not possible. Reference values used were generated in the same laboratory using the methodology used for the diagnostic cases. The AChE activity in the reference eagles was similar to that reported by other laboratories using modifications of the same basic technique (Table 4). Since publication of the values by Blakley and Yole (2002), another magpie was found to have brain AChE activity of 23.6 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue. Westlake et al. (1983) reported a mean value of 4.92 $\mu mol \cdot min^{-1} \cdot g^{-1}$ of tissue for six foxes. We are not aware of published reference values for coyotes or skunks.

Seasonal occurrence of poisoning coin-

TABLE 4. Comparison of reference brain cholinesterase (AChE) activity (micromoles per minute per gram of tissue) in eagles used as control values in this study with those reported by other authors. Values are mean ± SD (*n*).

	This study[a]	Hill (1988)	Smith et al. (1995)	Friend and Franson (1999)
Bald eagle	17.21±3.18 (29)	16.0±1.7 (6)	16.1±2.1 (76)	16.1±2.6 (156)
Golden eagle	13.37±0.90 (4)	14±3.2 (16)	16.2±2.2 (29)	16.0±2.2 (57)

[a] From Blakley and Yole (2002).

cided with migration of eagles through the area. Paucity of cases in January and February is likely related to the presence of relatively few eagles during these months and because coyote and fox carcasses, as well as baits, may have been snow-covered and unavailable. Buried carcasses and bait would become available with snow melt in March and April as eagles were returning. We do not know how long the frozen animals had been dead prior to discovery or how long carbofuran persists in bait or carcasses under cold conditions. Carbofuran caused direct and secondary poisoning for at least 60 days after placement under autumn conditions in Kansas (Allen et al., 1996), and it may have persisted longer under colder conditions. Lack of poisoning incidents in June through September may have resulted from several factors, including less placement of poison at this time of year; rapid decomposition of carcasses; breakdown of pesticides in warm weather; and because most nesting eagles are located further north, in the boreal forest, at this time of year. Only three incidents involving eagles were detected during these four months. The case in June was discovered 1 June, at which time both the eagle and fox involved were desiccated and unsuitable for testing, indicating that the deaths had occurred weeks earlier. The two cases in August occurred in a distinctly different situation than all other cases. Both eagles were found dead along the shore of a northern lake—one in Saskatchewan, the other in the Northwest Territories. Their esophaguses contained recently ingested fish flesh, suggesting that they had consumed poison bait.

Almost all of the incidents occurred in Saskatchewan, which reflects the origin of submissions to the laboratory. Between 1991 and 2002, only one eagle was submitted from each of Alberta and the Northwest Territories. In addition to the eagles from Manitoba reported here, other bald eagles from that province that were classified as either suspicious or presumed to have been poisoned by anticholinesterase pesticides were excluded from analysis because the date and location of death were not available. Our findings—together with those of Allen et al. (1996) and unconfirmed reports of pesticides, especially carbofuran, being used to poison coyotes in other jurisdictions—suggest that secondary poisoning of eagles may be widespread in western North America. Mineau et al. (1999) concluded that bald and golden eagles figured prominently in cases of pesticide abuse in the USA between 1985 and 1995. The impact of anticholinesterase poisoning on regional eagle populations is unknown. We do not know what proportion of poisoning incidents are detected and submitted for examination, but we suspect that specimens from relatively few incidents reach a diagnostic laboratory. Mortality of adults, as occurred in >60% of the eagles examined, is of concern for long-lived birds that are late to mature. Experience in Europe where local population reduction or disappearance of various raptors occurred as a result of use of strychnine baits to control wolves (*Canis lupus*) and foxes in the past (Bijleveld, 1974) should be considered cautionary.

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