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POISONING OF WILDLIFE WITH ANTICOAGULANT RODENTICIDES IN NEW YORK

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ABSTRACT: From 1971 through 1997, we documented 51 cases (55 individual animals) of poisoning of non-target wildlife in New York (plus two cases in adjoining states) (USA) with anticoagulant rodenticides—all but two of these cases occurred in the last 8 yrs. Brodifacoum was implicated in 80% of the incidents. Diphacinone was identified in four cases, bromadiolone in three cases (once in combination with brodifacoum), and chlorophacinone and coumatetralyl were detected once each in the company of brodifacoum. Warfarin accounted for the three cases documented prior to 1989, and one case involving a bald eagle (*Haliaeetus leucocephalus*) in 1995. Secondary intoxication of raptors, principally great horned owls (*Bubo virginianus*) and red-tailed hawks (*Buteo jamaicensis*), comprised one-half of the cases. Gray squirrels (*Sciurus carolinensis*), raccoons (*Procyon lotor*) and white-tailed deer (*Odocoileus virginianus*) were the most frequently poisoned mammals. All of the deer originated from a rather unique situation on a barrier island off southern Long Island (New York). Restrictions on the use of brodifacoum appear warranted.

Key words: Anticoagulant rodenticide poisoning, brodifacoum, bromadiolone, *Bubo virginianus, Buteo jamaicensis,* diphacinone, great-horned owl, *Odocoileus virginianus,* raptor, red-tailed hawk, white-tailed deer, wildlife.

INTRODUCTION

Anticoagulant poisons interfere with the action of vitamin K in the production of clotting factors in the liver, and thereby kill by predisposing animals to fatal hemorrhage. They are presently the most commonly-used pesticides for the control rodent pests world-wide. The first anticoagulant synthesized for use as a pesticide was the coumarin-based compound warfarin, introduced in the 1940's (Osweiler et al., 1985). Other coumarin-based rodenticides were subsequently developed, as were indandione compounds like diphacinone which show similar anticoagulant activity. Emergence of rat populations with resistance to warfarin and some of the other early anticoagulants eventually led to the development of more potent compounds like brodifacoum and bromadiolone which, unlike the older compounds, will kill rodents after single feedings.

Toxicity data for a variety of domestic animals (Osweiler et al., 1985), suggest that anticoagulant rodenticides are no doubt a potential hazard to many wild mammals and birds. The degree of hazard would be expected to vary by compound, species, and type of application. Poisoning could occur by direct ingestion of bait (primary poisoning), or via consumption of poisoned rodents (secondary poisoning). Potential for the latter was demonstrated by Evans and Ward (1967) who fed anticoagulant-killed nutria (*Myocastor coypus*) to dogs and commercial mink, by Mendenhall and Pank (1980) who fed rats and mice killed with a variety of rodenticides to owls, and by Townsend et al. (1984) who fed warfarin-dosed mice to least weasels (*Mustela nivalis*).

In the United States, applications of brodifacoum in apple orchards resulted in the deaths of radio-marked screech owls (*Otus asio*) (Hegdal and Colvin, 1988). Littrel (1988) reported the diphacinone-caused deaths of a raccoon (*Procyon lotor*) and a mountain lion (*Felis concolor*) at a site in northern California under unknown circumstances. More recently, two barn owls were killed by brodifacoum near a poultry farm in Georgia (USA) in 1995 (C. F. Quist, pers. comm.).

In the UK, Shawyer (1987) reported a "mass mortality" involving tawny owl (*Strix aluco*), buzzard (*Buteo buteo*), magpie (*Pica pica*), and red fox (*Vulpes vulpes*) which followed baiting with brodifacoum at a Hampshire farm in 1981. Shawyer (1987) also reported suspected poisonings in barn owls (*Tyto alba*) associated with the use of brodifacoum (four cases), difenacoum (four cases) and bromadiolone (one case) between 1982 and 1985. Subsequently, about 10% of 145 barn owls found dead in Britain between 1983 and 1989 were found to contain detectable (>0.005 ppm) levels of brodifacoum, although only one owl was considered to have succumbed to poisoning (Newton et al., 1990). In France, a four-year study of possible anticoagulant poisonings of wildlife (Berny et al., 1997) yielded 59 confirmed diagnoses for bromadiolone and 41 for chlorophacinone. Twenty-eight animals, principally red foxes and buzzards, were recovered from a single area near the Swiss border where bromadiolone was applied to carrot baits for the control of voles (*Arvicola terrestris*). Similar use of bromadiolone in Switzerland itself was followed by a large kill of buzzards and kites (*Milvus milvus*). Several unspecified predatory mammals also were killed (Beguin, 1983 and Pedrolic, 1983, cited in Shawyer, 1987). In Malaysia, barn owls were reportedly decimated when brodifacoum and coumachlor replaced warfarin on a palm oil plantation (Duckett, 1984). In New Zealand, one magpie, one paradise duck (*Tadorna ferruginea*), two unidentified hawks, two unidentified gulls, one unidentified passerine, and one unidentified hare were found dead following experimental use of brodifacoum to control rabbits (*Oryctolagus cuniculus*) (Rammell et al., 1984). Other reports of avian mortality linked to brodifacoum use in New Zealand have been reviewed by Eason and Spurr (1995).

Given the experimental evidence, the magnitude of rodenticide use, some of the reports cited above, and our own data, we suspect that the poisoning of wildlife in the United States with anticoagulants is far more common than the published record suggests. The purpose of the present paper is to report both primary and secondary poisoning of non-target wildlife with anticoagulant rodenticides in New York (USA) from 1971 through 1997.

METHODS

Most of the cases included in this report were submitted for diagnosis directly or indirectly by the general public. Wildlife rehabilitators also contributed a significant number of specimens. When necropsies showed hemorrhage or anemia in the absence of traumatic injury or infectious or parasitic disease processes, the livers were collected, frozen, and shipped to an analytical laboratory. Analyses were completed at the New York State Police Laboratory (Albany, New York) prior to 1977, and subsequently at the WARF Institute and its successor, Raltech Scientific Services (Madison, Wisconsin, USA), through 1984. Since then, analyses were completed at the State of Illinois Animal Disease Laboratory (Centralia, Illinois, USA) using a high-performance liquid chromatography screening procedure. This method utilized a solid-phase cleanup to prepare small-sized samples (2g) for analysis (Chalermchaikit et al., 1993). Identification and quantitation of the 11 different anticoagulant rodenticides in the screen were achieved by reverse-phase separation using both UV and fluorescence detectors (Shimadzu models SPD-6AV and RF-535, Shimadzu Scientific Instruments, Inc., Columbia, Maryland, USA) in tandem to facilitate both the primary and confirmatory analysis (J. R. Stedelin, unpubl. data). When needed, particularly with indandione compounds, additional sensitivity and confirmation were attained with an ion-pairing method (Hunter, 1985). Detection limits at the Illinois Laboratory were 0.02 ppm for brodifacoum and bromadiolone; 0.03 ppm for difenacoum; 0.05 ppm for coumatetralyl; 0.1 ppm for warfarin, fumarin, and coumachlor; 0.02 ppm for diphacinone; 0.4 ppm for pindone and valone; and 0.5 for 4, 6, 7 and 8-OH warfarin.

RESULTS

Death from hemorrhage associated with anticoagulant rodenticides was confirmed in 51 cases (56 individual animals) over a 27 yr period (Table 1). Only three cases, all involving warfarin, were diagnosed pri-

TABLE 1. Anticoagulant rodenticide poisonings in wildlife in New York (USA) and adjoining states, 1971–1997.

Species[a]	Date (Mo/yr)	County[b]	Gross pathology[c]	Toxicant (ppm in liver)
Gray squirrel	10/71	Westchester	d	warfarin (not quantified)
Gray squirrel	9/81	Niagara	b, d, k	warfarin (0.228)
Peregrine falcon	10/86	Cape May	c, d, h	warfarin (1.48)
Great horned owl	3/89	Putnam	a, b	brodifacoum (0.01)
Great horned owl	10/89	Suffolk	a, b	brodifacoum (0.2)
Gray squirrel	6/90	Westchester	a, h	brodifacoum (0.7)
Gray squirrel	7/90	Monroe	b, l	brodifacoum (4.1)
Eastern chipmunk	6/92	Albany	a, b	brodifacoum (3.8)
Raccoon	6/92	Niagara	j	brodifacoum (1.8 in stomach contents)
Raccoons (3)	9/92	Nassau	c	brodifacoum (3.1, 5.3, 4.6)
Gray squirrel	8/93	Albany	a, d, h	brodifacoum (0.53), chlorofacinone (0.62)
Gray squirrel	9/93	Albany	a, d, j	brodifacoum (25.8 in colon contents)
Snowy owl	11/93	Dutchess	a, d	diphacinone (0.26)
Great horned owl	3/94	Niagara	b, h	brodifacoum (0.53)
Great horned owl	6/94	Albany	a, b, f, g	brodifacoum (0.64)
White-tailed deer	10/94	Suffolk	c, i	brodifacoum (0.38)
Great horned owl	10/94	Erie	a, b, f	brodifacoum (0.41)
Red-tailed hawk	11/94	Westchester	b, g, f	brodifacoum (0.41)
Great horned owl	11/94	Orleans	a, b, e, f, g	brodifacoum (0.73)
Great horned owl	12/94	Albany	a, b, d, f, g	brodifacoum (0.1)
Red-tailed hawk	12/94	Westchester	a, b, f	brodifacoum (0.23)
Red-tailed hawk	1/95	Richmond	b, h	brodifacoum (0.43)
Red-tailed hawk	3/95	Nassau	b, g	brodifacoum (0.76)
Bald eagle	4/95	Orleans	i	warfarin (1.4)
Great horned owl	8/95	Suffolk	a, d, h	brodifacoum (0.53), bromadiolone (0.14)
White-tailed deer	9/95	Suffolk	c, d, e	brodifacoum (0.37), coumatetralyl (0.5)
Red-tailed hawk	12/95	Suffolk	a, b, f, g	brodifacoum (1.6)
Great horned owl	2/96	Chenango	a, h, i	brodifacoum (0.36)
Raccoon	3/96	Suffolk	a, d, e, j	brodifacoum (1.0)
Red fox (2)			b, c, e	brodifacoum (1.32 and 4.01)
Skunks (3)	4/96	Westchester	a, c, e	bromadiolone (0.02, 0.28, 0.08)
Raven	4/96	Rensselaer	a, d, h	brodifacoum (1.04)
Golden eagle	4/96	Monroe	c	brodifacoum (0.03)
White-tailed deer	4/96	Suffolk	c, e	brodifacoum (0.12)
White-tailed deer	5/96	Suffolk	c, k	brodifacoum (0.41)
Red-tailed hawk	6/96	Onondaga	a, b, f, g	brodifacoum (0.65)
Great horned owl	6/96	Monroe	a, b, d, e, g	brodifacoum (0.35)
White-tailed deer	10/96	Suffolk	c, e	diphacinone (0.93)
Red-tailed hawk	10/96	Suffolk	b, i	brodifacoum (0.5)
Opossum	11/96	Albany	c	bromadiolone (0.8)
White-tailed deer	12/96	Suffolk	c	diphacinone (0.2)
Gray squirrel	12/96	Albany	a, b, d	brodifacoum (1.39)
Common crow	1/97	Fairfield	a, d, f, j	brodifacoum (1.34)
Screech owl	2/97	Suffolk	a, c	brodifacoum (0.34)
Great horned owl	2/97	Greene	a, b, f	brodifacoum (0.08)
Raccoon	3/97	Albany	a, d, f, l	brodifacoum (0.32)
Gray squirrel	4/97	Suffolk	a, e	diphacinone (2.0)
Opossum	4/97	Albany	e	brodifacoum (0.18)
Great horned owl	4/97	Niagara	i	brodifacoum (0.11)
Great horned owl	6/97	Dutchess	b, g	brodifacoum (0.22)
Screech owl	10/97	Erie	d	brodifacoum (0.80)
White-tailed deer	12/97	Suffolk	a, c, e	brodifacoum (0.16)

[a] See text for scientific names.

[b] All in New York except for Cape May (New Jersey) and Fairfield (Connecticut).

[c] a = subcutaneous hemorrhage; b = pallor of muscle and/or internal organs; c = hemorrhage in lungs; d = inter- and intramuscular hemorrhages; e = subcutaneous edema; f = low blood volume heart/major vessels; g = excessive hemorrhage from superficial wounds; h = free hemorrhage or bloody fluid in body cavity; i = hemorrhage into alimentary canal; j = dyed rodenticide bait in alimentary canal; k = hemorrhage and/or serum in pericardial sac; l = intrauterine hemorrhage.

or to 1989. Most of the poisonings originated in one of three regions in New York including (1) Long Island and the lower Hudson River Valley (40°30′ to 41°31′N and 72°50′ to 74°00′W), (2) within about 80km of our laboratory near Albany (42°35′N and 73°53′W), and (3) counties bordering the southwest shore of Lake Ontario and the Niagara River (42°25′ to 43°23′N and 77°37′ to 79°04′W).

Presumed secondary poisoning of raptors was documented in 26 cases, principally in great horned owls (13 cases) and red-tailed hawks (seven cases). Presumed primary intoxications were confirmed in 16 cases, primarily in gray squirrels and white-tailed deer. Rodenticide bait was visually identified in the alimentary canal in four of these cases: two raccoons, one squirrel, and one common crow (*Corvus brachyrhynchos*). The poisonings in the red foxes, skunks (*Mephitis mephitis*), opossums (*Didelphis virginiana*), and the other raccoons could have been primary or secondary in nature.

Brodifacoum was identified in the liver or alimentary canal in 41 cases (80%). Warfarin was identified in four cases, diaphacinone in four, and bromadiolone in three (once in combination with brodifacoum). Coumatetralyl and chlorophacinone were found (once each) in the company of brodifacoum.

Gross post-mortem findings (Table 2) typically included subcutaneous hemorrhage in both avian and mammalian specimens. Birds tended to show more noticeable overall pallor, frequently had a notably reduced volume of blood in the heart and associated large vessels, and about one-third showed excessive external hemorrhage from superficial wounds. Subcutaneous hemorrhage in raptors frequently included sites over the abdomen, including intense hemorrhage between the stomach and the abdominal wall in several specimens. Pulmonary hemorrhage was far more common in mammals. Also, mammals sometimes showed areas of subcutaneous edema. The edematous fluid was frequently blood-tinged and, in some cases, perhaps a sequel to earlier hemorrhage. In general, tentative diagnoses based on gross findings in birds were confirmed by analytical results in almost every case. However, the gross findings on mammals seem to be less pathognomonic, and confirmations were much less certain, particularily for carnivores and ungulates.

TABLE 2. Gross pathological findings in wildlife killed by anticoagulant rodenticides in New York.

Finding	Frequency[a] Birds (n = 25)	Mammals (n = 27)
Subcutaneous hemorrhage	+++	+++
Pallor	+++	++
Hemorrhage in lungs	+	+++
Inter/intra-muscular hemorrhage	++	++
Subcutaneous edema	+	++
Free hemorrhage in body cavity	++	+
Dyed bait in alimentary canal	+	++
Excessive bleeding from minor wounds	++	o
Low blood volume in heart/major vessels	++	o
Hemorrhage into alimentary canal	+	+
Hemorrhage/serum in pericardial sac	o	+
Intrauterine hemorrhage	NA	+

[a] Constructed from necropsy records, these frequencies should be considered conservative as records varied in degree of detail: +++ frequent (>50%), ++ occasional (10–50%), + infrequent (<10%), o not observed, NA not applicable.

We were able to link known anticoagulant use and raptor mortality to a particular site on only three occasions. In the first case, brodifacoum was employed in barns and sheds at a small farm outside of Albany (New York) where horses, goats, and llamas were kept. Availability of livestock food and an absence of barn cats combined to produce an enormous rat population. Despite the fact that a great horned owl had been repeatedly observed in the vicinity of the barnyard, brodifacoum-treated bait was used in the buildings. An

owl was eventually found near death at the site, having suffered near complete exsanguination from a small laceration on one toe.

The second case involved a red-tailed hawk in rehabilitation at our facility in Delmar (New York). The bird had been fed a rat found dead on the ground by an employee (counter to instructions never to utilize animals found dead for animal food) unaware that brodifacoum bait was being used at an adjoining residence. The hawk, kept in an outdoor flight cage not designed to exclude small animals, may have captured additional brodifacoum-exposed rodents on its own.

The third incident occurred at a lower Hudson Valley correctional facility where a rodent control program (using a tracking powder containing 0.2% diphacinone) coincided with presence of a snowy owl (*Nyctea scandiaca*) which had established a temporary residence at the site. The owl's stomach was full of rat remains.

The white-tailed deer poisonings with brodifacoum (4) and diphacinone (2) all occurred on Fire Island, a barrier island on the south shore of Long Island. No hunting is allowed and the deer population is large and food-limited. The deer are fed by some Fire Island residents and, as we observed on one field visit, have come to associate plastic bags with food opportunities. The remains of plastic bags or wrappers were occasionally found in the rumen of deer at necropsy. This situation is sometimes coupled with a heavy use of rodenticides in some of the small communities. Deer have been observed eating baits, even going to considerable effort to extract them from beneath buildings supported on low piers. Other deer we have examined in the past few years from this location may also have been poisoned by anticoagulants, but post-mortem scavenging and/or decay precluded or compromised confirming analyses. The most common gross finding in poisoned deer was extremely hemorrhagic lungs.

TABLE 3. Acute oral LD_{50} values[a] (mg/kg) for some common anticoagulant rodenticides in several species.

	LD^{50} (mg/kg)			
Species	Warfarin	Diphacinone	Bromadiolone	Brodifacoum
Rat	50–100	1.5	1.25	0.27
Mouse	374[b]	340	1.75	0.4
Rabbit	800[b]	35	1.0	0.29
Pig	3	—	—	0.5–2.0
Dog	50	3	11–15[c]	0.25–1.0
Cat	5–50	15	>25[c]	25
Chicken	1,000[b]	—	—	10–100

[a] As reported in Osweiler et al. (1985) unless otherwise indicated.
[b] Reported in Hagan and Radomski (1953).
[c] Reported in Felice and Murphy (1995).

DISCUSSION

Our data suggest that poisonings of nontarget wildlife with anticoagulant rodenticides may currently be widespread, largely due to the use of brodifacoum. More acutely toxic to most species tested than other commonly-used anticoagulants (Table 3), brodifacoum is also very persistent in the liver (Bachmann and Sullivan, 1983; Laas et al., 1985). For example, substantial amounts of brodifacoum were found in the livers of sublethally dosed possums (*Trichosurus vulpecula*) 8 mo after exposure, with little decline after the first week (Eason et al., 1996). Repeated sublethal exposures, even intermittent ones, may therefore be expected to eventually cause fatal hemorrhage. Not surprisingly, Mendenhall and Pank (1980) experimentally showed brodifacoum to be the most hazardous of six anticoagulants to barn owls fed poisoned rats.

Brodifacoum and other anticoagulants are not registered for field use in the USA. Some outdoor use may occur, however, as U.S. Environmental Protection Agency-approved label directions for commensal rodent control permit placement of bait "in and around" structures—the term "around" being unquantified. Extensive outdoor placement is probably rare in New York, however, as we have confirmed

relatively few primary anticoagulant poisonings in species that should be particularly vulnerable to outdoor baiting.

The toll on New York State wildlife is nonetheless impressive, particularly with respect to two raptor species. Since 1994, anticoagulant poisonings have comprised 17% (n = 59) of our diagnoses for great horned owls, and 6% (n = 114) of our diagnoses for red-tailed hawks. The actual rate of exposure of these raptors and other wildlife in New York to these anticoagulants is unknown as we have not screened the livers of animals found dead of other causes.

The poisoning of white-tailed deer on Fire Island raises some questions about human consumption of deer and other game. A common pattern of residential development in many areas of New York State at present is for houses to be erected on large (e.g., 0.4–2.0 ha) lots in prime wildlife habitat on the outskirts of cities and suburbs. White-tailed deer often continue to thrive in close proximity to these houses. Norway rats (*Rattus norwegicus*) also occasionally do well in some of these areas if birdseed or pet foods provide a food source. When the latter occurs, the use of rodenticides may be expected to increase, possibly exposing deer and other wildlife to poison bait. As long as sufficient natural foods persist, the likelihood of deer consuming bait is probably slight. However, as hunting is almost inevitably restricted to some extent in these areas, deer populations may become food limited (as on Fire Island) and bait consumption could become more common. Unlike Fire Island, however, some hunting, at least by archers, is likely to continue in or adjacent to these developments, potentially exposing hunters to contaminated flesh. It would be useful to survey deer livers for anticoagulants in some areas which currently approximate these circumstances. Surveys of livers of game species would also appear warranted in locations abroad where field applications of anticoagulants occur.

We recommend that physiologically persistent anticoagulants, particularly brodifacoum, should not be used if direct or indirect exposures to wildlife are likely to occur. Warfarin or a similarly non-persistent anticoagulant requiring multiple feedings should be considered in locations where warfarin-resistance has not been demonstrated. Rodenticides containing cholecalciferol, bromethalin or phosphide can be used if warfarin-resistance is known to be present, although these products can also present a serious direct threat to some wildlife species if placed outdoors. Appropriate regulatory agencies worldwide should consider additional restrictions on the use of brodifacoum.

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