EXPOSURE OF NON-TARGET WILDLIFE TO ANTICOAGULANT RODENTICIDES IN CALIFORNIA

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ABSTRACT: The California Department of Fish and Game collected and analyzed tissue samples from non-target birds and mammals for anticoagulant rodenticides from 1994 through 1999. Many of these animals were collected in recently urbanized areas adjacent to wildlands where they were either found dead or trapped and euthanized as vertebrate pests. The results of the analyses indicate a high frequency of exposure to the anticoagulant rodenticide brodifacoum. Fifty-eight percent of the animals examined had been exposed to brodifacoum, 19% to bromadiolone, 9% to diphacinone and 8% to chlorophacinone. All of the identified anticoagulants are registered for use to control commensal rodents found in and around structures and are available for sale "over-the-counter" for homeowner use. Brodifacoum and bromadiolone are registered exclusively for commensal rodent control. This paper assesses the frequency of anticoagulant rodenticide residues in tissues of non-target mammalian and avian wildlife and the possible impacts.

KEY WORDS: anticoagulant, brodifacoum, bromadiolone, chlorophacinone, diphacinone, wildlife, non-target, coyotes, golden eagle, raptor, carnivore, rodenticide

THIS PAPER HAS BEEN PEER REVIEWED.

Proc. 19th Vertebr. Pest Conf. (T.P. Salmon & A.C. Crabb, Eds.) Published at Univ. of Calif., Davis. 2000.

INTRODUCTION

Beginning in the 1940s with warfarin and dicoumarin, anticoagulant rodenticides (Hadler and Buckle 1992) have served as important tools in vertebrate pest control programs around the world (Brown and Singleton 1998; Kay et al. 1994; Clark 1978; Whisson 1996). Clark (1978) noted that agricultural commissioners in Santa Barbara and Ventura counties, California, were successfully using warfarin and pival treated grain baits for control of California ground squirrels (*Spermophilus beecheyi*) in agricultural settings by 1953.

The early or "first generation" anticoagulants included warfarin, pival, coumafuryl, coumachlor, coumatetralyl, diphacinone, and chlorophacinone. These compounds acted as chronic toxicants, requiring multiple exposures over a short period of time (days) to be effective (Hadler and Buckle 1992). The risk to non-target animals appeared to be low because the half life ($t_{½}$) for these compounds in the tissues of exposed animals was relatively short. For example, the $t_{½}$ for warfarin is between 5 and 28 hours (Hadler and Buckle 1992; Aiello 1998). A short retention time ensured that residues in a target animal, should it be captured prior to death or the carcass scavenged, were not very high. Unless several exposed prey were captured by the same predator in very quick succession, the probability of a lethal dose through secondary exposure was very low (Jackson and Ashton 1992). The low probability of toxic effects resulting from secondary exposure to first generation compounds was demonstrated by Townsend et al. (1981) investigating the effects of warfarin on Tawny Owls (*Strix aluco*).

By the 1960s, resistance to warfarin was reported in several commensal rodent species in the U.S. and the U.K. (Hadler and Buckle 1992). With the advent of resistance, came the development of new, more acutely toxic "second generation" anticoagulant compounds. These compounds included, worldwide: brodifacoum; bromadiolone; difethialone; difenacoum; and flocoumafen. These new compounds had a significantly higher toxicity; a lethal dose resulted from a single feeding as opposed to the multiple feedings needed for the earlier anticoagulants (Hadler and Buckle 1992).

The principle use of anticoagulants worldwide has been for control of commensal rodents, primarily black rats (*Rattus rattus*), Norway rats (*Rattus norvegicus*) and house mice (*Mus musculus*) (Whisson 1996). Over 200 products containing anticoagulant compounds are currently registered for control of commensal rodents in California (California Department of Pesticide Regulation registration data). Three second generation compounds (brodifacoum, bromadiolone, and difethialone) are currently registered for use in California for control of commensal rodents. There are 35 active registrations for products containing brodifacoum, 37 for bromadiolone, and 12 for difethialone (California Department of Pesticide Regulation registration data). Brodifacoum, in particular, because of its high toxicity has been used in situations where exposure may be limited to a single application. For example, eradication of rats and mice from islands has been accomplished (Howald 1997; Empson and Miskelly 1999; Stephenson et al. 1999; Ogilvie et al. 1997; Taylor and Thomas 1989; Newman 1994). Some large-scale field applications of brodifacoum have also been employed for control of pest species including: rabbits (*Oryctolagus cuniculus*), brush-tailed possums (*Trichosurus vulpecula*), house mice (*Mus musculus*), and experimentally in the U.S., voles (*Microtus* sp.) (Williams et al. 1986; Murphy et al. 1998; Rammell et al. 1984; Brown and Singleton 1998; Kay et al. 1994; Duckett 1984; Hegdal and Colvin 1988). Bromadiolone has also been extensively marketed as a single feeding commensal rodent control compound.

Laboratory tests have demonstrated that secondary poisoning of non-target wildlife is a possibility with anticoagulant compounds (Evans and Ward 1967; Mendenhall and Pank 1980; LaVoie 1990). In some of the laboratory tests, sub-lethal effects such as significantly lengthened clotting times were observed in

addition to mortality effects (Townsend et al. 1981; Mendenhall and Pank 1980). Even so, some believed that anticoagulants were much safer than the previously available acutely toxic rodenticides like Compound 1080 and strychnine (Hadler and Buckle 1992; Kaukeinen 1982). The availability of an antidote in Vitamin K_1 (Hadler and Buckle 1992; Miller 1984; Mackintosh et al. 1988) and the time lag between exposure and onset of symptoms provided an opportunity for administration of the antidote. Published reports of non-target wildlife losses, due both to primary and secondary exposure, have been associated with applications of anticoagulant baits for agricultural pest control, rat eradication programs on islands, and large-scale eradication and control programs for introduced species. In California, a raccoon (*Procyon lotor*), and a mountain lion (*Felis concolor*) were killed from primary exposure to diphacinone, resulting from a deliberate misuse of rodent baits (Littrell 1988). Wild or feral mammalian carnivore losses have been reported from New Zealand, England, France, and the United States (Stone et al. 1999; Alterio 1996; Alterio et al. 1997; Stephenson et al. 1999; Walton 1970 in Birks 1998; Berny et al. 1997).

Predatory and scavenging birds are also at risk from exposure. Howald (1997) reported Common Ravens (*Corvus corax*) removed and consumed bait blocks containing brodifacoum from bait stations on Langara Island during a rat eradication program. Blood plasma residues of brodifacoum were also documented for Bald Eagles (*Haliaeetus leucocephalus*) captured and tested after the initiation of the intensive baiting program (Howald et al. 1999). Both the Common Ravens and Bald Eagles could have been exposed through scavenging of rat carcasses containing residues of brodifacoum or through catching exposed rats. The eagles may also have been exposed as a result of scavenging raven carcasses (Howald 1997; Howald et al. 1999). Brodifacoum residues were detected in 13 raven carcasses collected from the island. Significant haemorrhages were identified in 12 of the 13 birds, the 13th bird being too autolytic for a complete necropsy (Howald et al. 1999). Brodifacoum residues were also detected in Northwestern Crows (*C. caurinus*) following a preliminary baiting test on an adjacent small island (Howald 1997; Howald et al. 1999). Hegdal and Colvin (1988) and Merson et al. (1984) reported Eastern Screech Owls (*Otus asio*) were exposed to brodifacoum, with some fatalities, following experimental broadcast applications of brodifacoum baits for control of voles in dormant apple orchards. Shawyer (1987); Newton et al. (1990); Rammel et al. (1984); Stephenson et al. (1999), and Ogilvie et al. (1997) all reported losses of raptors due to exposure to anticoagulant rodenticides (brodifacoum, difenacoum, and bromadiolone). The barn owl (*Tyto alba*) population in the oil palm plantations on the Malaysian peninsula dropped dramatically following the replacement of warfarin and coumachlor based rat baits with baits containing brodifacoum (Duckett 1984). Duckett (1984) further reported that several birds had been recovered dead from some of the plantations with evidence of bleeding from the nares. Hegdal and Blaskiewicz (1984) however, reported that the use of brodifacoum treated baits on farms in New Jersey did not result in adverse impacts to the resident Barn Owl population as the birds tended to feed in grasslands away from the buildings where the applications were being made.

Few reports of non-target wildlife losses from urban or structural use have been published (Stone et al. 1999; Godfrey 1985; Birks 1998). Godfrey (1985) reported birds in a zoo aviary died after secondary exposure to brodifacoum through consumption of insects which had fed on rodent baits in bait stations. In New York, mammals ranging from herbivores such as Eastern gray squirrels (*Sciurus carolinensis*) and white-tailed deer (*Odocoileus virginianus*) to carnivores like raccoons and red fox (*Vulpes vulpes*) have been recovered and determined to have died as a result of exposure to brodifacoum, chlorophacinone, coumatetralyl, diphacinone, bromadiolone, and warfarin (Stone et al. 1999). It should be noted that coumatetralyl is not registered for use in the United States. Stone et al. (1999) also reported losses of several avian predator and common scavenger species including Golden Eagles (*Aquila chrysaetos*), Great Horned Owls (*Bubo virginianus*), Red-Tailed Hawks (*Buteo jamaicensis*), Snowy Owls (*Nyctea scandia*), Ravens, Common Crows (*C. brachyrhynchos*), and Eastern Screech Owls. Many of these animals were recovered from areas with significant urban development (W. B. Stone, pers. comm.)

METHODS

The California Department of Fish and Game Pesticide Investigations Unit (PIU) is notified of wildlife losses with possible pesticide exposure by department enforcement personnel (wardens), department biologists, and staff of other State or local (county) agencies. Cooperating wildlife rehabilitation groups and private citizens also will notify the PIU of suspicious wildlife losses (Littrell 1990). Carcasses of recovered birds and mammals are delivered to the PIU where necropsies are performed and appropriate tissue samples are collected and submitted for analysis of pesticide residues. The wildlife discussed in this paper were recovered dead, in a moribund condition and subsequently euthanized, or trapped as vertebrate pests or public safety animals and euthanized. The animals in this latter category (eight coyotes and two raccoons) all reportedly appeared to be in good health at the time of capture.

The wildlife discussed in this paper were examined for possible exposure to, and symptomology of, anticoagulant rodenticides. Symptoms of anticoagulant exposure include: 1) the presence of large subcutaneous haematomas without accompanying signs of physical trauma; 2) significant haemorrhage into the thoracic or abdominal cavities; 3) haemorrhage into the gastrointestinal tract; 4) a lack of clotting of blood in the heart and major vessels if the carcass is fresh; 5) congestion in the liver or lungs with significant bleeding from any cut surfaces; 6) the presence of mesenteric or subcutaneous fat deposits colored blue as a result of consumption of marker dyes used on some anticoagulant treated rodent baits; or 7) a history or physical signs of bleeding from the nares, mouth, or anus without accompanying signs of physical trauma.

This paper has been peer reviewed.

The principle organ where anticoagulant compounds accumulate is the liver (Buck et al. 1976; Newton et al. 1990; Howald 1997). To avoid contamination of tissue samples by any anticoagulant residues present in the bile, the gall bladder was first excised from the animal before the liver was collected. Newton et al. (1990) reported that different anticoagulant compounds may be concentrated in different areas within the liver. To account for this possibility the entire liver was homogenized and analyzed for anticoagulant residues. In addition to liver tissue, free blood from the heart or major vessels was, on occasion, collected and submitted for analysis. Gut contents were also collected and analyzed if it was suspected that rodent baits had been consumed. In cases where fat deposits in the carcass appeared to be dyed blue, samples of mesenteric fat were collected and analyzed for the presence of the blue marker dye Dupont Oil Blue A™ or Keystone Oil Blue A™.

Laboratory Analytical Methods

Most tissue samples collected during 1994 and 1995 were analyzed for the presence of chlorophacinone or diphacinone at the Department of Fish and Game Water Pollution Control Laboratory (WPCL) using high performance liquid chromatography (HPLC) with a UV detector. Some tissue samples during this time were submitted to Michigan State University for analysis of nine anticoagulant compounds (brodifacoum, bromadiolone, warfarin, coumachlor, pindone, chlorophacinone, diphacinone, valone, and coumafuryl) using the methods described by Braselton et al. (1992). Beginning in September 1995, tissue samples were analyzed for nine anticoagulant compounds (coumafuryl, warfarin, bromadiolone, brodifacoum, coumachlor, diphacinone, chlorophacinone, pindone, and difethialone) at the California Veterinary Diagnostic Laboratory at the University of California, Davis (CVDL). CVDL samples were analyzed using HPLC with post column fluorescence detector (Palazoglu et al. 1998). The detection limits for the three laboratories varied (Table 1). Fat samples were submitted to the WPCL for a qualitative determination of the presence of blue dye using the methodology given in Littrell et al. (1987).

RESULTS

Since 1994, tissues from 74 animals have been collected and analyzed for residues of anticoagulant rodenticides. The animals represented 21 different species of birds and mammals. Residues of anticoagulant rodenticides were identified in 30 of 43 (70%) mammals examined (Table 2). Sixty-one percent of the mammals had been exposed to brodifacoum, 19% to bromodialone, and 12% to each chlorophacinone and diphacinone (Figure 1). Twelve (28%) mammals had been exposed to multiple anticoagulant compounds.

Residues of anticoagulant rodenticides were also identified in 21 of 31 (68%) birds examined (Table 3). Fifty-five percent of the birds had been exposed to brodifacoum, 19% to bromadiolone, and less than 10% to each diphacinone and chlorophacinone (Figure 1). Five of the birds (16%) had been exposed to multiple anticoagulant compounds. The two mammals most frequently exposed to anticoagulant rodenticides were coyotes (*Canis latrans*) (15) and bobcats (*Lynx rufus*) (4). The two birds most frequently exposed to anticoagulant rodenticides were Golden Eagles (6) and Barn Owls (4).

Table 1. Detection limits (parts per million, ppm) for analytical procedures to detect anticoagulant rodenticides in tissue samples.

Anticoagulant Compound	DFG, WPCL[a]	Michigan State, VDL[b]	UC Davis, CVDLS[c]
Warfarin	N.A.[d]	0.02	0.05
Pindone	N.A.	0.2	0.25
Valone	N.A.	0.2	N.A.
Coumachlor	N.A.	0.02	0.05
Brodifacoum	N.A.	0.002	0.01
Bromadiolone	N.A.	0.02	0.05
Difethialone	N.A.	N.A.	0.25
Coumafuryl	N.A.	1.0	0.1
Diphacinone	0.4	0.2	0.25
Chlorophacinone	0.2	0.2	0.25

[a]Department of Fish and Game Water Pollution Control Laboratory.
[b]Michigan State University Veterinary Diagnostic Laboratory (Braselton et al. 1992).
[c]University of California, Davis Veterinary Diagnostic Laboratory (Palazoglu et al. 1998).
[d]No analytical procedure.

This paper has been peer reviewed.

Table 2. Numbers of non-target mammals examined for residues of anticoagulant rodenticides.

Species	n	Non-Detect	Brodifacoum	Bromadiolone	Chloro-phacinone	Diphacinone	Brodifacoum, Bromadiolone[a]	Brodifacoum, Diphacinone	Brodifacoum, Chlorophacinone	Brodifacoum, Diphacinone, Chlorophacinone	Brodifacoum, Bromadiolone, Diphacinone
Coyote[b]	17	2	6		1	1	4	1	1	1	
Red Fox	3		3								
S.J. Kit Fox	4	1	1				1		1		
Gray Fox	2	1	1								
Raccoon[c]	2						1				1
Bobcat	9	5	2		1		1				
Mountain Lion	1		1								
Heermann's Kangaroo Rat	1					1					
Fox Squirrel	1	1									
Beaver	1	1									
Badger	1	1									
Black-tailed Deer	2	2									
Total	43	13	14	0	2	2	7	1	2	1	1

[a]Residues of multiple compounds identified in the same animal.

[b]Eight animals, appearing to be in good health, were trapped as vertebrate pest or public safety animals and euthanized.

[c]Two animals, appearing to be in good health, were trapped as vertebrate pests and euthanized.

Table 3. Numbers of non-target birds examined for residues of anticoagulant rodenticides.

Species	n	Non-Detect	Brodifacoum	Bromadiolone	Chloro-phacinone	Diphacinone	Brodifacoum, Bromadiolone[a]	Brodifacoum, Diphacinone	Brodifacoum, Chlorophacinone	Brodifacoum, Diphacinone, Chlorophacinone	Brodifacoum, Bromadiolone, Diphacinone
Golden Eagle	10	2	8								
Red-tailed Hawk	4	3	1								
Red-shouldered Hawk	2		1				1				
American Kestrel	1			1							
Barn Owl	6	2	1	1			2				
Great Horned Owl	4	1	1				1	1			
Turkey Vulture	2	1				1					
Turkey	1				1						
Oregon Junco	1	1									
Total	31	10	12	2	1	1	4	1	0	0	0

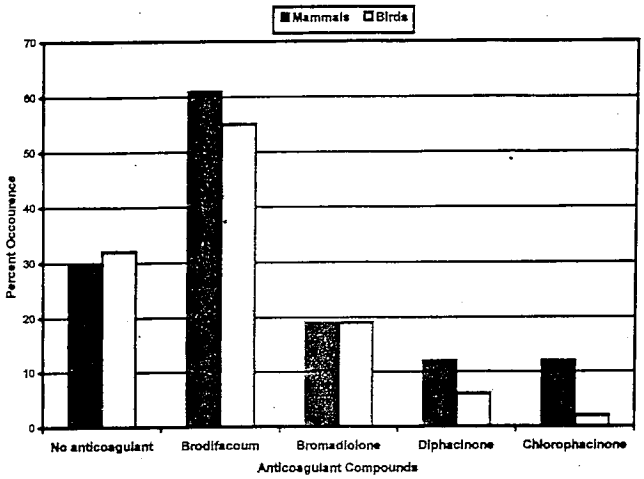

Figure 1. Frequency of anticoagulant residues in California wildlife.

In animals where multiple residues were identified, brodifacoum was always one of the compounds. The concentration of anticoagulants in liver tissue exhibited intraspecies variation of several orders of magnitude (Table 4). In mammals, bromadiolone was always found in association with one or more other anticoagulant compounds.

Clinical signs consistent with anticoagulant toxicosis were observed during necropsies in 43% of the animals with anticoagulant residues. These signs included subcutaneous haemorrhage, pulmonary haemorrhage, thoracic and coelomic haemorrhages, and the presence of quantities of unclotted blood in the heart and major blood vessels in fresh carcasses. Clinical signs consistent with anticoagulant toxicosis were also observed in two bobcats and a Golden Eagle, which did not contain residues of anticoagulants. However, tissues from these three animals were analyzed at WPCL, and only for diphacinone and chlorophacinone. Other anticoagulant compounds including brodifacoum may have been present but were not detected because of the analytical method.

Table 4. Range of anticoagulant rodenticide concentrations (ppm) identified in non-target wildlife.

Wildlife	n	Brodifacoum	Bromadiolone	Chlorophacinone	Diphacinone
Mammals					
Coyote	17	<0.01-0.5 (12)	0.07-0.46 (4)	<0.01-1.2 (3)	0.043-1.3 (3)
Red Fox	3	0.04-0.05 (2)	N.D.	N.D.	N.D.
S. J. Kit Fox	4	0.07-0.47 (3)	0.72 (1)	0.77 (1)	N.D
Gray Fox	2	0.03 (1)	N.D.	N.D.	N.D.
Racoon	2	0.08-0.41 (2)	0.011-1.1 (2)	N.D.	0.13 (1)
Bobcat	8	0.018-0.07 (3)	0.11 (1)	0.4 (1)	N.D.
Mountain Lion	1	0.52 (1)	N.D.	N.D.	N.D.
Heermann's Kangaroo Rat	1	N.D.	N.D.	N.D.	3.5 (1)
Mammalian LD_{50}[a]		0.27-25.0	1.125-25.0	2.1-50	0.88-340
Birds					
Golden Eagle	10	<0.01-0.13 (8)	N.D.	N.D.	N.D.
Red-tailed Hawk	4	0.01 (1)	N.D.	N.D.	N.D.
Red-shouldered Hawk	2	0.01-0.15 (2)	0.28 (1)	N.D.	N.D.
American Kestrel	1	N.D.	<0.01 (1)	N.D.	N.D.
Barn Owl	6	0.07-0.35 (3)	0.31-0.38 (3)	N.D.	N.D.
Great Horned Owl	4	0.015-0.35 (3)	0.065 (1)	N.D.	0.6 (1)
Turkey Vulture	2	N.D.	N.D.	N.D.	0.4 (1)
Turkey	1	N.D.	N.D.	5.5 (1)	N.D.
Avian LD_{50}[a]		2-100	16.93	100	3158

[a]Acute LD_{50} value ranges (Stone et al. 1999; PMEP 1999; Anonymous 2000; and California Department Pesticide Regulation data).

This paper has been peer reviewed.

The Heermann's kangaroo rat (*Dipodomys heermanni*) also exhibited blue dyed subcutaneous and mesenteric fat deposits. This is characteristic of primary consumption of grain baits dyed with the identifier dye, Dupont Oil Blue A™. Additional possible causes of death in these animals included physical trauma such as vehicle impacts, wind generator strikes, and bite wounds, lead toxicosis, and organophosphate toxicosis. In some cases no specific reason for the loss could be identified.

The majority of the animals in the current study were recovered from recently urbanized areas adjacent to wildlands. Some of the animals (eight coyotes and two racoons) were trapped in urban areas as vertebrate pests or public safety animals. Most of the animals recovered had been part of ongoing radio telemetry studies. The telemetry data on these animals indicated that their territories included both areas of urban development and wildlands.

DISCUSSION

Fifty-one of the 74 animals (69%) examined in the current study had been exposed to anticoagulant rodenticides. Over 95% of the exposed animals in the current study came from areas with significant urban development. These facts indicate that exposure of non-target wildlife through urban use of anticoagulant rodenticides may be important in California. The primary anticoagulant compound identified in both the current study and in New York (Stone et al. 1999) was brodifacoum. In this study, 43 of the animals (58%) examined had been exposed to brodifacoum, 61% of the mammals, and 55% of the birds. Nineteen percent of the animals (14) examined had been exposed to bromadiolone. In California, both of these compounds are only registered for use in, or adjacent to, structures. The observed frequency of non-target exposure to brodifacoum and bromadiolone may be due to the availability of these two compounds for homeowner use. Manufacturers' data for 1996 and 1997 indicated that more than 90% of the "over-the-counter" rodent control products available to the homeowner contained one of these two compounds (Dale Kaukeinen, pers. comm.). All four of the compounds (brodifacoum, bromadiolone, diphacinone, and chlorophacinone) identified during this study are registered for the control of commensal rodents.

It is not possible to estimate the time from exposure to death nor the amount of anticoagulants ingested by the animals. This is because of the variable delay between ingestion and when the animals were examined. This delay also makes, for some species, the determination of primary or secondary exposure, problematic. Some presumptions pertaining to primary or secondary exposure can be made based on the biology of the individual species. Birds of prey, will characteristically not consume pelletized or grain type foods. Therefore, it is highly likely that exposure to anticoagulant rodenticides in these species is secondary through consumption of exposed prey. Bobcats and mountain lions are primarily carnivores. Likewise, it would seem unlikely for these species to consume rodenticide baits directly. Raccoons and canid species, particularly coyotes, are omnivorous. They are known to be associated with urban areas and to feed on pet food, garden snails, fruits, as well as rodents and trash found around residences (Rex Baker, pers. comm.). Thus, it may not be possible to determine if exposure to the compounds was primary or secondary. With granivorous species such as the wild turkey (*Meleagris gallopavo*) or the kangaroo rat, exposure was most probably primary. In the case of the kangaroo rat this is supported by the observation of blue dyed fat during the necropsy.

Acute LD_{50} data indicate that brodifacoum has the highest toxicity of the four identified rodenticides for both birds and mammals (Table 4). In birds, the second most toxic compound is bromadiolone. Diphacinone appears to have a higher toxicity than bromadiolone for canids and felids, but it ranked below bromadiolone for other mammals (Anonymous 2000; Stone 1999). Chlorophacinone appears to be the least toxic of the four compounds for mammals while available data indicates that diphacinone is the least toxic compound in birds. The concentration of an anticoagulant compound in a tissue represents only a part of the total amount the animal ingested. Laboratory studies found that between 4% and 33% of anticoagulants ingested by owls was regurgitated, unabsorbed, in pellets of undigested bones and fur (Newton et al. 1990; Gray et al. 1994; Townsend et al. 1981). Laas et al. (1985) and Townsend et al. (1981) also demonstrated that between 6% and 33% of anticoagulant compounds may be passed directly through the digestive tract and excreted in the faeces of both birds and mammals. These findings indicate that a significant percentage of the ingested quantity of anticoagulant compound may be eliminated from the animal's body without being absorbed. Because of its higher toxicity, brodifacoum would be expected to have an effect on exposed animals at a lower concentration than the other compounds. In some cases where a sub-lethal exposure has occurred, tissue retention times are significant. Eason et al. (1996a, 1996b) have reported extended tissue retention times of more than 250 days for brush-tailed possums exposed to a single sub-lethal dose of brodifacoum. Rodents exposed to anticoagulants may continue to feed on treated baits, if available, until shortly before death and could possibly be taken by predators during that time. Howald (1997) reported that rats collected from Langara Island during an eradication program contained up to 1.8 mg of brodifacoum in the complete carcass. Predators exposed to a sub-lethal dose of an anticoagulant would be expected to continue to hunt and could consume additional exposed prey. This recurring exposure, coupled with a protracted tissue retention time, could result in accumulation of a lethal concentration of a compound. The presence of multiple residues in some of the animals in this study could be explained in this way. Berny et al. (1997) and Stone et al. (1999) also both reported residues of multiple compounds in non-target animals. Rodent control activities by individual homeowners using different rodent control products could result in multiple anticoagulant compounds being present in the prey base. This in turn could result in multiple exposures in non-target predators. The effects of exposure to multiple anticoagulant rodenticides do not appear to have been extensively studied.

This paper has been peer reviewed.

Most of the animals examined during this study came from areas with significant urban interface with wildlands or undeveloped areas which could provide habitat to support limited wildlife populations. Some of the animals like coyotes, kit foxes (*Vulpes macrotis mutica*), and barn owls seem to have adapted very well to living in this type of setting. Others, like Golden Eagles and mountain lions, have probably incidentally encompassed part of the urban landscape into relatively large territories. A similar phenomenon of non-target exposure has not been observed in areas where primarily agricultural activities interface with wildlands. The anticoagulant compounds identified, the percentages of their occurrence in recovered animals, and the proximity of urban development all support the theory that these animals have been exposed to anticoagulant rodenticides through commensal rodent control activities. However, deliberate misuse of the compounds to kill predatory animals or to control other rodent species cannot be ruled out.

In July 1999, based on the significant numbers of exposed non-target wildlife, the California Department of Fish and Game (DFG) requested that the California Department of Pesticide Regulation place all products containing the active ingredient brodifacoum into re-evaluation. As part of this re-evaluation request DFG identified areas where additional data were needed. These data needs included the extent of non-target wildlife exposure to brodifacoum in California, the impact of sub-lethal levels of brodifacoum on wildlife health and the effects of concurrent exposure to brodifacoum and other anticoagulant rodenticides in non-target wildlife.

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