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Anticoagulant Rodenticides in Three Owl Species from Western Canada, 1988–2003

Courtney A. Albert · Laurie K. Wilson · Pierre Mineau · Suzanne Trudeau · John E. Elliott

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Abstract Anticoagulant rodenticides are widely used to control rodent infestations. Previous studies have shown that nontarget organisms, such as birds, are at risk for both primary and secondary poisoning. This paper presents rodenticide residue information on the livers from 164 strigiformes which included barn owls (*Tyto alba*), barred owls (*Strix varia*), and great horned owls (*Bubo virginianus*), collected from 1988 to 2003 in the province of British Columbia and the Yukon Territory, Canada. Livers were analyzed for brodifacoum, bromadiolone, chlorophacinone, diphacinone, difethialone, and warfarin. Our results show that, of the 164 owl livers analyzed, 70% had residues of at least one rodenticide, and of these 41% had more than one rodenticide detected. Of the three species of owls examined, barred owls were most frequently exposed (92%, $n = 23$); brodifacoum and bromadiolone were most often detected, with liver concentrations ranging from 0.001 to 0.927 mg/kg brodifacoum, and 0.002 to 1.012 mg/kg bromadiolone. Six of the owls (three barred owls, two barn owls, and one great horned owl) were diagnosed as having died from anticoagulant poisoning; all six owls had brodifacoum residues in the liver.

C. A. Albert · J. E. Elliott (✉)
Science and Technology Branch, Environment Canada, Pacific and Yukon Region, Delta, BC V4K 3N2, Canada
e-mail: John.elliott@ec.gc.ca

C. A. Albert
e-mail: courtney.albert@ec.gc.ca

L. K. Wilson
Canadian Wildlife Service, Environment Canada, Pacific and Yukon Region, Delta, BC V4K 3N2, Canada
e-mail: Laurie.wilson@ec.gc.ca

P. Mineau · S. Trudeau
Science and Technology Branch, Environment Canada, National Wildlife Research Center, Ottawa, ON K1A 0H3, Canada
e-mail: Pierre.mineau@ec.gc.ca

S. Trudeau
e-mail: Suzanne.trudeau@ec.gc.ca

Rodent infestations continue to cause substantial property damage and pose health risks in both urban and rural farm land. One preferred method of controlling rodent populations worldwide is the use of anticoagulant rodenticides, which kill rodents by predisposing them to fatal hemorrhage (Stone et al. 1999). Commonly used rodenticides, such as brodifacoum, bromadiolone, and difethialone, are classified as second-generation anticoagulant rodenticides (SGARs). These types of rodenticides were introduced in the 1970s following widespread development of rodent resistance to first-generation anticoagulant rodenticides such as warfarin, chlorophacinone, and diphacinone (Buckle et al. 1994). SGARs are much more acutely toxic than the first-generation anticoagulant rodenticides, generally providing a lethal dose after a single feeding. They tend to be considerably more persistent in animal tissues (US EPA 2004) and have higher affinity for liver tissue (Parmar et al. 1987).

Surveys of pesticide sales in British Columbia (B.C.) determined that from 1991 to 2003 there was a 100% increase in the sales of brodifacoum active ingredient (a.i.) (0.21 kg in 1991 to 0.42 kg in 2003), and a 24% increase in the sales of bromadiolone a.i. (0.43 kg to 0.53 kg). Difethialone was registered in B.C. in 2000, and 0.081 kg of this compound was sold in B.C. in 2003. Chlorophacinone a.i. sales dropped 48% in this period (0.24 kg to 0.13 kg), and diphacinone a.i. sales went down 13% (0.17 kg to 0.15 kg) (Environment Canada 2005).

In British Columbia, meadow and montane voles (*Microtus pennsylvanicus*, *M. montanus*), northern pocket

gophers (*Thomomys talpoides*), Columbian ground squirrels (*Spermophilus columbianus*), Norway and roof rats (*Rattus norvegicus*, *R. rattus*), and house mice (*Mus musculus*) are considered common rodent pests, and for many years anticoagulant rodenticides have been primary tools in control programs (Anderson and Kluge 1986). Commercial anticoagulant rodenticide (AR) baits often used by farmers can be purchased in the form of pellets, loose meal, paraffin blocks or packet baits, and are available from various companies and in varying concentrations (Pest Management Regulatory Agency 2006). Tamper-proof bait stations, meant to reduce primary poisoning of nontarget animals, are available. Historically, the use of these covered, locking bait stations was only recommended but not mandatory. However, in 2006, labels on commercial SGAR bait were revised to include that either tamper-proof bait stations be used or bait be placed in locations not accessible to children, pets or livestock (Pest Management Regulatory Agency 2006). In general, second-generation anticoagulant rodenticides are largely restricted to indoor use, or against the outside walls of buildings. The B.C. Ministry of Agriculture, Food, and Fisheries (1996) recommends that farmers record the number, location, date placed, and date replaced of each bait station on their farm property. However, these recommendations are not enforceable. The extent to which commercial applicators are used and to which the general public comply with these use directions is unclear.

In an anticoagulant rodenticide baiting situation, rodents may not die for several days after consuming a lethal dose (Cox and Smith 1992), and may to continue to feed on the bait if it is available. Rodents exposed to anticoagulant rodenticides show altered behavior such as spending more time in open areas, staggering, and sitting motionless before death; all of these behaviors may increase susceptibility to predation (Cox and Smith 1992). Consequently, the use of anticoagulant rodenticides to control rodent pests has resulted in secondary exposure of various nontarget animals, including populations of birds of prey worldwide (Merson et al. 1984; Hegdal and Colvin 1988; Stone et al. 1999; Howald et al. 1999; Eason et al. 2002; Walker et al. 2008). Reports include sublethal exposure to anticoagulant rodenticides in liver tissue, as well as larger-scale events of anticoagulant rodenticide poisoning. Brodifacoum has been shown to have greater secondary toxicity to barn owls (*Tyto alba*) than bromadiolone (Mendenhall and Pank 1980) and difenacoum (Newton et al. 1990).

As part of a longer-term raptor monitoring study, several owls were believed to have been acutely poisoned by anticoagulant rodenticides, based on severe abdominal hemorrhaging. This was impetus for a broader assessment of exposure. In this paper we present anticoagulant rodenticide residue data on the livers from 164 owls collected throughout British Columbia and the Yukon from 1988 to 2003. The degree of exposure as well as spatial and temporal trends are also examined.

Methods

Sample Collection

Between 1988 and 2003, 164 dead owls were collected from British Columbia and the Yukon region of Western Canada (Fig. 1). Owl carcasses were received from the B.C. Ministry of Environment, the Yukon Ministry of Environment, Canadian Wildlife Service, Monika's Wildlife Shelter, Orphaned Wildlife Rehabilitation Society (OWL), Wildlife Rescue Association (WRA), Mountainaire Avian Rescue Society (MARS), Fur and Feather Taxidermy, South Okanagan Rehabilitation Center for Owls (SORCO), North Island Wildlife Recovery Association (NIWRA) as well as from the general public. Specimens consisted of 61 great horned owls (GHOW, *Bubo virginianus*), 25 barred owls (BDOW, *Strix varia*), and 78 barn owls (BNOW, *Tyto alba*). Owls were grouped into eight geographical areas as follows: Upper Fraser Valley (Chilliwack, Aggasiz, Hatzic, Mission, Abbotsford, Hope, Maple Ridge, Pitt Meadows), Fraser Delta–Urban (Vancouver, West and North Vancouver, Burnaby, New Westminster, Coquitlam), Fraser Delta–Agricultural (Delta, Richmond, Tsawwassen, WhiteRock, Point Roberts, Surrey), North Coast (Whistler, Pemberton, Prince Rupert), Vancouver Island (Courtenay, Nanaimo, Duncan, Galiano Island, Port Hardy), Okanagan (Kelowna, Oliver, Summerland), Interior (Kamloops), and the Yukon. Two owls were submitted to rehabilitation centers with no information regarding where they came from in B.C.

Most of the owls were dead upon arrival to the rehabilitation centers, while others died while in custody or were euthanized due to the severity of the injuries. Suspected final cause of death was determined by a veterinarian upon post mortem inspection, and body condition was ranked as follows: 1 (emaciated), 2 (thin), 3 (fair), 4 (good/very good), 5 (excellent). Livers were then extracted from carcasses and analyzed for anticoagulant rodenticide residues.

Exposure was based on whether an owl had detectable levels of anticoagulant rodenticides in the liver tissue. Poisoning was determined based on post mortem observations of severe abdominal hemorrhaging as well as confirmation by rodenticide residue analysis.

Chemical Analysis

Chemical analysis was conducted at the National Wildlife Research Center in Ottawa, Ontario, Canada. Liver (0.50 g) was ground in a mortar with about 5 g anhydrous

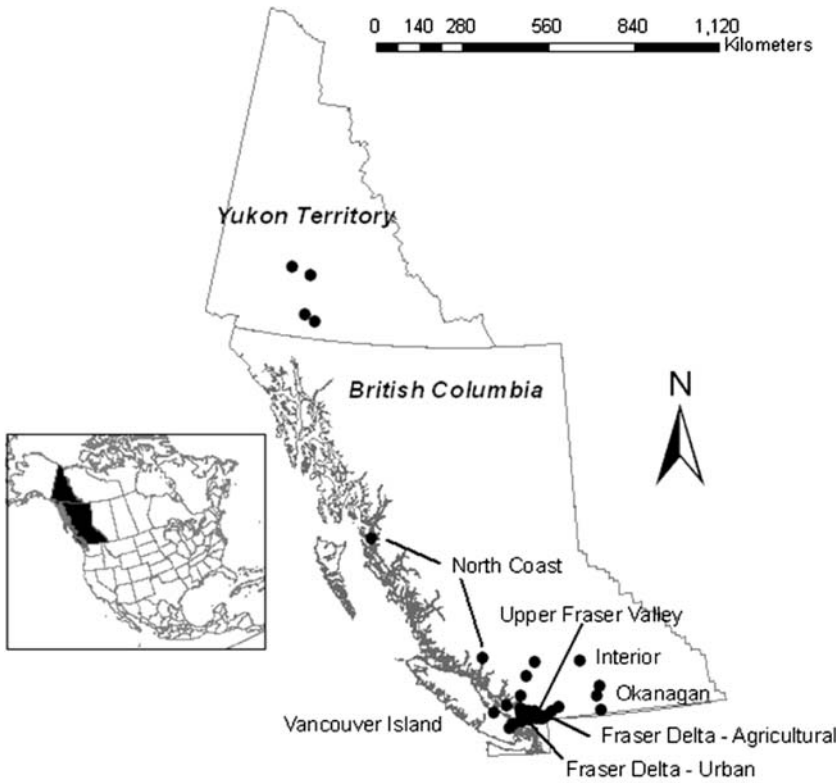

Fig. 1 Locations where owls were collected in British Columbia and the Yukon from 1988 to 2003

sodium sulfate (Fisher no. S420-3). The resulting mixture was transferred to a 25-mL amber glass septum bottle and extracted with acetonitrile (EMD Omnisolv, AX0142-1, HPLC grade; 1 × 7 mL and 2 × 5 mL). The extract was hand shaken vigorously for 2 min and then shaken mechanically for 15 min. After centrifugation at room temperature at about 1,000 rpm for 15 min, the supernatant was removed and transferred into a 40-mL graduated conical tube. The supernatant of the two subsequent extractions were combined with the first supernatant. The total supernatant was evaporated to 10 mL under a stream of nitrogen in a water bath maintained at about 40°C.

To clean up liver extract, a 2 mL portion (corresponding to 0.1 g liver) was transferred into a test tube and evaporated to dryness. The sample was reconstituted in 1 mL acetonitrile and cleaned by solid-phase extraction using one or two Sep-Pak plus tC18 conditioned cartridges (cat WAT036810). After the introduction of the sample into the SPE cartridge, the tube containing the sample was rinsed with 6 mL acetonitrile and that 6 mL was added to the SPE cartridges. The eluate (1 mL sample + 6 mL rinse) was evaporated to dryness, reconstituted in 1 mL MeOH, and filtered through an Acrodisc® syringe filter with a 0.45-μm polyvinylidene fluoride (PVDF) membrane. A dilution was made and internal standards were added before liquid chromatography-mass spectrometry (LC-MSMS) analysis. A volume of 10 μL of the diluted filtered extract was analyzed.

Samples were analyzed by LC-MSMS. The high-performance liquid chromatography (HPLC) system consisted of a Waters Alliance 2695 HPLC, equipped with an X-Terra® MS C_{18} 3.5 μm, 2.1 × 100 mm (Waters) analytical column, and the corresponding guard column X-Terra® MS C_{18} 3.5 μm, 2.1 × 20 mm (Waters). The column temperature was maintained at 40°C and the sample temperature at 22°C. The mobile phase consisted of A = ammonium acetate (Fisher) 10 mM, pH 6.8 and B = methanol. The flow rate was maintained at 0.250 ml/min with the following mobile phase gradient:

Mobile phase gradient

Time (min)	A %	B %
0.00	75	25
10.00	5	95
20.00	5	95
24.00	75	25
28.00	75	25

The rodenticides were detected with a triple quadrupole mass Quatro-Ultima (Waters) with negative electrospray ionization (ESI) in multiple reaction monitoring scanning mode (MRM). The following parameters were chosen:

MRM parameters

Compound	Parent ion (Da)	Daughter ion (Da)	Cone voltage (V)	Collision energy (eV)	Dwell time (s)
Pindone	229.0	116.0	52	35	0.30
Warfarin	307.1	161.0	50	20	0.30
Diphacinone	339.0	167.0	52	26	0.30
Chlorophacinone	373.0	201.0	78	24	0.30
Brodifacoum	521.0	135.0	104	42	0.30
Bromadiolone	525.0	250.0	96	35	0.30
Difethialone	536.9	150.9	54	45	0.30

Triple quadrupole settings

Electrospray source (negative mode) settings	Analyzer settings
Capillary voltage: 3 kV	LM 1 and HM 1 resolution: 13.0
Source temperature: 120°C	Ion energy 1: 1.0
Desolvation temperature: 350 °C	LM 2 and HM 2 resolution: 13.0
Cone gas flow: 105 L/h	Ion energy 2: 1.0
Desolvation gas flow: 694 L/h	Pirani pressure (collision): 2.79×10^{-3} mbar

The minimum detectable amount was 0.005 µg/g for diphacinone and difethialone, and 0.002 µg/g for warfarin, brodifacoum, chlorophacinone, and bromadiolone. The standards were all analytical grade (>98% purity). The calibration curve was built with five levels of concentrations (ranging from 2.5 to 80 pg) with $r^2 > 0.99$. The samples were diluted in order to fit within the limits of the calibration curve. Recoveries at low and high level were >70 % for all compounds. The addition of a known amount of coumatetralyl (5 pg/µL; transition 291.00 >140.90) and flocoumafen (1 pg/µL; transition 541.40 >382.00) to each sample prior to the injection allowed monitoring for ion suppression. A blank containing 100% methanol was injected between each sample to monitor for any possible contamination due to carry over.

Statistics

To determine if a difference existed in mean liver residue in birds which died from suspected rodenticide poisoning, trauma, disease or undetermined causes of death, a non-parametric analysis of variance (ANOVA) was applied. Where differences were found, Tukey's honestly significant difference (HSD) tests were used to identify which cause of death differed significantly in terms of mean liver residue. When comparing the magnitudes of liver residues over the sampling period a one-way ANOVA was applied.

Results

Post Mortem Results

Final diagnoses were categorized as trauma (motor vehicle collisions, window collisions, electrocution, attacks by other wildlife, caught in leg-traps, flight collision, falling out of the nest), poisoning by rodenticides, disease (aspergillosis, bumblefoot, pericarditis, chest infection, herpes virus, starvation) or undetermined (Table 1). Of the 164 owls examined, the most common diagnosis was trauma ($n = 113$) followed by undetermined causes ($n = 25$), disease ($n = 19$), and rodenticide poisoning ($n = 6$). Body condition of each owl was ranked by veterinarian upon post mortem examination (Table 2).

There was no relationship between presence or absence of residues and final diagnosis. There was no obvious

Table 1 Final cause of death of owls and anticoagulant rodenticide (AR) status

	Number	Number with one or more AR residues	Number with more than one AR residue
Trauma*	113	81	47
Disease**	19	15	9
AR poisoning	6	6	3
Undetermined	25	12	9
Total	164	114	68

* Trauma includes motor vehicle collisions, window collisions, electrocution, attacks by other wildlife, caught in leg-traps, flight collision, falling out of the nest

** Disease includes aspergillosis, bumblefoot, pericarditis, chest infection, herpes virus, starvation

Table 2 Body condition of each owl, ranked on a scale from 1 (emaciated) to 5 (excellent), and AR residue range in mg/kg wet wt

Body condition	GHOW	BNOW	BDOW	All	AR residue range
1	11	15	2	28	0–0.609
2	10	13	3	26	0–0.366
3	15	22	2	39	0–0.768
4	16	22	13	51	0–1.052
5	7	4	4	15	0–0.941
Undetermined	2	2	1	5	
Total	61	78	25	164	

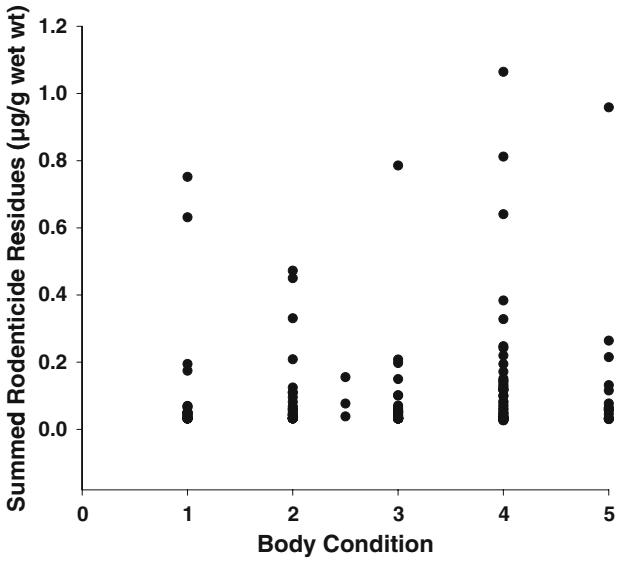

Fig. 2 Summed rodenticide residues (µg/g wet weight) in livers of three species of owls from British Columbia and the Yukon from 1988 to 2003, plotted against the numerical ranking for body condition on a scale from 1 (emaciated) to 5 (excellent)

relationship between body condition and rodenticide residues; owls with the highest levels of rodenticides and confirmed poisoning cases were not necessarily in poor condition, and some were ranked as being in excellent condition (Fig. 2).

Anticoagulant Rodenticide Residues

Of the 164 liver samples analyzed, 70% had detectable residues of at least one anticoagulant rodenticide, and 41% had more than one anticoagulant. Seventeen livers (10%) had detectable residues of three anticoagulant rodenticides; only one liver had residues of four different anticoagulant rodenticides. SGARs, especially brodifacoum and bromadiolone, were the most commonly detected anticoagulant rodenticides in all three species (Table 3); brodifacoum was detected in 51% of the livers analyzed, while bromadiolone was detected in 52% of all livers analyzed, with liver concentrations ranging from 0.001 to 0.927 mg/kg and 0.002 to 1.012 mg/kg, respectively. Difethialone, diphacinone, chlorophacinone, and warfarin residues were detected in 9%, 5%, 4%, and 3% of all 164 livers analyzed, respectively.

Owls that were suspected to have died from anticoagulant rodenticide poisoning had variable levels of rodenticides in liver tissue, with a range of 0.060 mg/kg up to 1.065 mg/kg summed rodenticide residues. However, when compared with other final diagnoses, concentrations of rodenticides were significantly higher on average in livers from owls that died from suspected rodenticide poisoning than from trauma and disease-related deaths and undetermined causes of death ($P > 0.005$; Fig. 3). Brodifacoum was present in all six cases where SGARs were the cause of death. Bromadiolone was present in four of these six cases.

Temporal Patterns

An examination of seasonal trends showed that the majority of each species were submitted in the winter months (December through February). Most of the collections were opportunistic and there were additional resources available to collect in the winter months. Additionally, that is a more stressful time for wild birds, which likely also increased collections during the winter months. When the magnitudes of liver residues over the sampling

Table 3 Number of owls (%) with detectable rodenticide residues out of total owls analyzed and mean (range) of AR residue concentrations; values presented as micrograms per gram wet weight

Species (sample size)	First-generation ARs			Second-generation ARs			Percentage exposed	Percentage poisoned**
	Warfarin	Diphacinone	Chlorophacinone	Brodifacoum	Bromadiolone	Difethialone		
GHOW (61)	5%	7%	5%	46%	56%	5%	70%	2%
	0.0037	0.011	0.0029	0.052	0.042	0.004		
	(0.0025–0.72)	(0.008*–0.012)	(0.0025–0.014)	(0.001–0.609)	(0.005–0.571)	(0.0025–0.03)		
BDOW (25)	4%	4%	16%	68%	76%	4%	92%	12%
	0.0026	0.010	0.0043	0.074	0.084	0.003		
	(0.0025–0.005)	(0.010–0.012)	(0.0025–0.015)	(0.001–0.927)	(0.002*–1.012)	(0.0025–0.017)		
BNOW (78)	1%	4%	0%	45%	40%	13%	62%	3%
	0.0025	0.010	N/A	0.027	0.020	0.019		
	(0.0025–0.008)	(0.010–0.02)		(0.01–0.47	(0.005–0.72)	(0.0025–0.72)		

* Lowest value less than half the detection limit, as found by chemist

** Final cause of death determined by a veterinarian

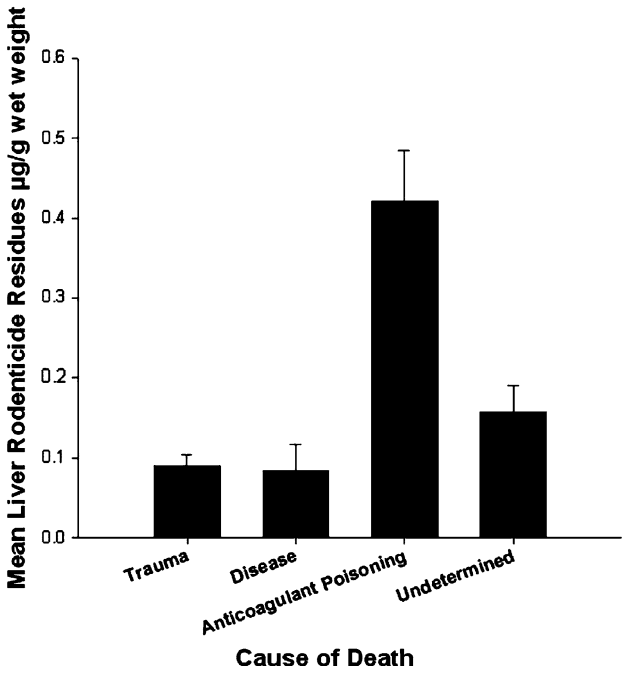

Fig. 3 Liver concentrations (µg/g wet weight) of anticoagulant rodenticides for each cause of death for all three owl species combined. Data are expressed as mean ± standard error

period were examined, there were no significant differences in any of the compounds over time (one-way ANOVA, $P > 0.05$ in all cases). The frequency with which each compound was detected was difficult to assess due to the varying numbers of owls collected each year. However, proportionately, there was evidence of an increase in frequency of detections from 1988 to 2003 (Fig. 4). Levels of magnitude of each compound did not increase over time within owl species ($P > 0.05$ in all cases) with the exception of brodifacoum, which significantly increased over time in barred owls (one-way ANOVA, $P < 0.05$).

Spatial Patterns

In general the overall distribution of positive cases reflected the geographic distribution pattern of owl submissions. The majority of the owls came from both the Upper Fraser Valley and the Fraser Delta–Agricultural areas. Forty-two percent of the cases with at least one anticoagulant rodenticide were from the Upper Fraser Valley, and 42% of all the positive brodifacoum cases came from this area. Similarly, 45% of the positive bromadiolone cases, and 42% of the positive difethialone cases, came from the Upper Fraser Valley area. Conversely, there were few chlorophacinone, diphacinone or warfarin detections in that area. Twenty-eight of the cases with at least one anticoagulant rodenticide were from the Fraser Delta–Agricultural area, and 31% of all the positive brodifacoum cases, 29% of all the positive bromadiolone cases, and 21% of all positive difethialone cases came from this area. In total, seven owls came from the Yukon, with two of these cases having chlorophacinone residues, and three of these cases having bromadiolone residues.

Species Patterns

Residues were detected in 62% of barn owls, 92% barred owls, and 70% of great horned owls (Table 3). A higher proportion of barred owls had detectable levels of brodifacoum, bromadiolone, and chlorophacinone, and at higher concentrations, than the other two owl species (Table 3). The proportion of owls with detectable liver residues of warfarin, diphacinone, and difethialone was similar for all species. Of the six confirmed rodenticide poisonings, three were barred owls, one was a great-horned owl, and two were barn owls.

Discussion

Anticoagulant rodenticides were detected in 70% of 164 owls, indicating a high rate of exposure to owls in British Columbia and the Yukon. Based on post mortem visual observations of hemorrhaging and residues, SGAR poisoning was confirmed to be the final diagnosis in six of the cases, and suspected as a possible mortality factor in several other cases based on liver residues. Three species of owls were analyzed and detections were especially frequent (92%, $n = 23$) in barred owls. There was no correlation between body condition and anticoagulant rodenticides residues in this study; owls with the highest levels of rodenticides, as well as those cases that were confirmed poisonings, were not necessarily in poor condition, and some were ranked as being in excellent condition (Fig. 4). Based on the high frequency of SGAR detections, this is likely due to the acute nature of the toxicity of SGARs and their ability to kill quickly, after a single dose.

Brodifacoum and bromadiolone were the most common residues in owl livers in this study. Between 1998 and 2001 in New York State, Stone et al. (2003) reported anticoagulant rodenticide residues for 265 raptors (including eagles, falcons, hawks, owls, and vultures) submitted by the general public. Anticoagulants were detected in livers of 49% of the 265 raptors. Of those detections, brodifacoum was the most frequently detected anticoagulant (84%), and was the suspected final cause of death in several of the cases. Brodifacoum levels were similar to those found in British Columbia and the Yukon, with an average of 0.18 µg/g (range 0.005–1.28 µg/g). In a similar study by Newton et al. (2000) in Great Britain, 54 barn owl livers were

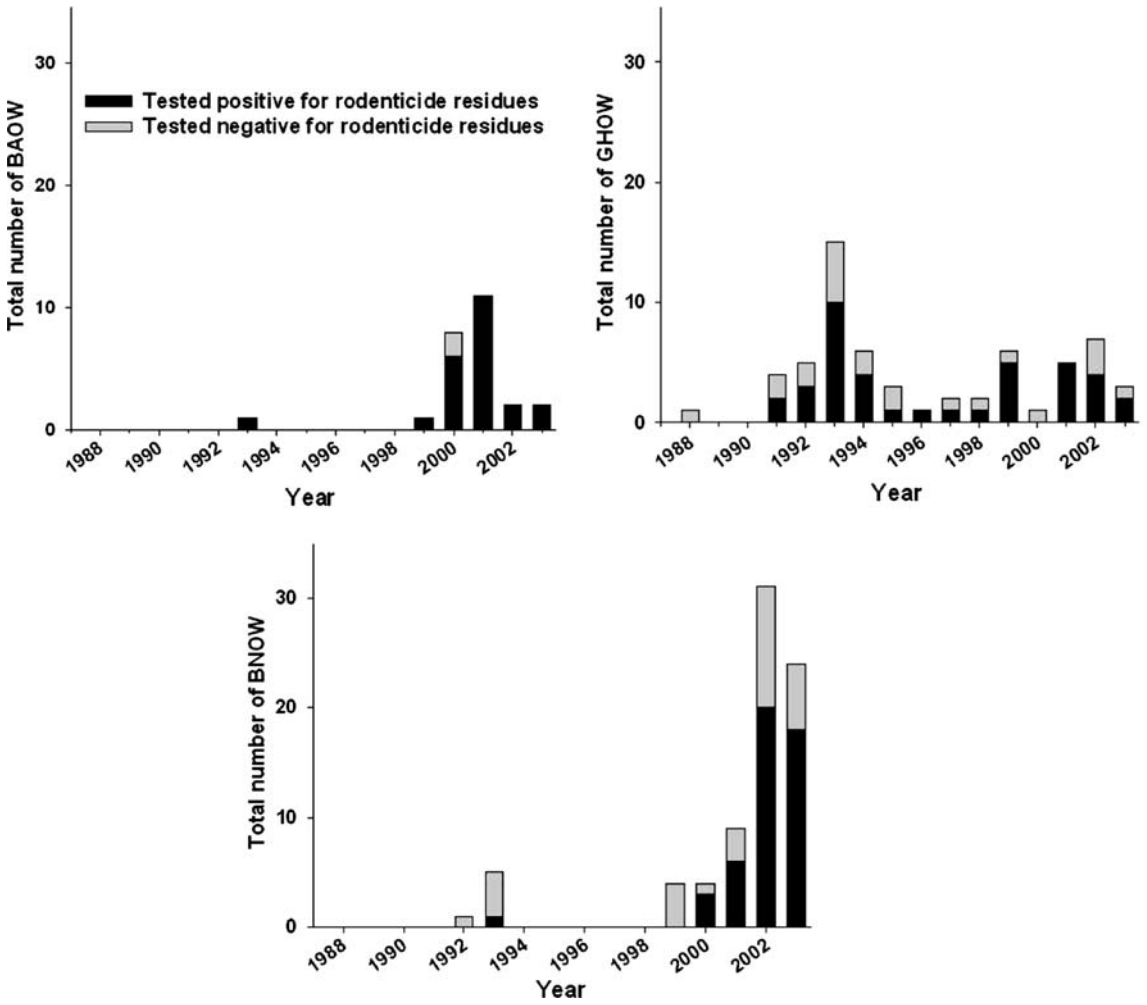

Fig. 4 Total number of barred owls (BAOW), great-horned owls (GHOW), and barn owls (BNOW) tested in each year and the proportion of those that tested positive

analyzed and 28 (52%) had detectable levels of rodenticide residues, and 24% had brodifacoum residues; six of those (11%) had levels which likely resulted in mortality. In a more recent survey of anticoagulant rodenticide exposure in tawny owls (*Strix aluco*) in Great Britain, 172 livers were analyzed using HPLC, which yields much higher detection levels than analysis with LC-MSMS as used in our study. Results from that British survey showed 19% had detectable residues of one or more AR, with bromadiolone being the most common (11.6%) followed by difenacoum (5.8%) and brodifacoum (4.7%) (Walker et al. 2008). SGARs are known to have longer half-lives in both the blood and tissues of animals than first-generation anticoagulant rodenticides, such as chlorophacinone, diphacinone, and warfarin, which are metabolized and excreted much more readily (Vandenbroucke et al. 2008). Several studies have shown considerable persistence of SGARs in animal tissues of a variety of species (Laas et al. 1985; Fisher et al. 2003). Based on the pharmacokinetics of brodifacoum and bromadiolone, Eason et al. (2002) predicted the likely persistence of residues after a sublethal dose in target and nontarget animals to be 24 months or longer (Eason et al. 2008). The increase in sales and use of SGARs and the decrease in sales and use of first-generation anticoagulant rodenticides may explain the high frequency of SGAR detections in our study compared with the more easily excreted first-generation anticoagulant rodenticides. Some owls may, therefore, have had low-level, sublethal body burdens of ARs for an extended period. However, the high frequency of SGAR detections in this study may not be completely indicative of actual exposure of owls to rodenticides in B.C. and the Yukon. Brodifacoum and bromadiolone were the two most detected compounds in our study; however, exposure to other anticoagulant rodenticides may be occurring more frequently than we are capable of detecting due to their faster metabolism and excretion from the body, thus we cannot rule out possible risks of exposure of owls to other anticoagulant

rodenticides compounds. Furthermore, as others have found (e.g., Stone et al. 2003) it is difficult to correlate mortality and liver residues in this study. Although the final diagnosis for most of the owls was determined to be other than SGAR poisoning, the high presence of residues in owl livers analyzed makes it difficult to rule out that the initial problem may have been behavioral changes such as lethargy due to SGAR poisoning, followed by a subsequent accident. Thus, we believe the proportion of deaths resulting from exposure to ARs in this study may be underestimated, and that rodenticides may have contributed to more owl deaths than we could confirm through post mortem observations and liver residues.

In our study, liver concentrations of brodifacoum and bromadiolone ranged from 0.002 to 0.97 μg/g and from 0.002 to 1.012 μg/g respectively. In a study by Newton et al. (1990), four out of six barn owls died after eating three brodifacoum-poisoned mice in 1 day, which was approximately the equivalent of a dose of 0.150–0.182 mg/kg owl weight. Deceased owls had liver concentrations of 0.63–1.25 μg/g. In later studies it was suggested that SGAR liver residues considered to be in the lethal range to barn owls is >0.1 μg/g wet weight (Newton et al. 1998) and then >0.2 μg/g wet weight (Newton et al. 1999). Similarly, in a study assessing rodenticide poisonings in New York, great horned owls experienced subcutaneous hemorrhaging with liver concentrations of brodifacoum as low as 0.01 μg/g (Stone et al. 1999). Of the owls in the present study with detectable anticoagulant rodenticides residues (summed compounds), 92% had residues >0.01 μg/g, 32% had residues >0.1 μg/g, 15% had residues >0.2 μg/g, and 6% had residues >0.63 μg/g. Therefore, since SGAR-exposed owls in our study are well within liver concentrations reported to cause mortality, we can assume that owls throughout British Columbia and the Yukon are at some risk of SGAR poisoning.

In general the distribution of anticoagulant rodenticides cases reflects the geographic distribution pattern of owl submissions. For example, the majority (43%) of owls submitted were from the Upper Fraser Valley, and the majority (42%) of the total positive cases came from that area. The highest brodifacoum liver residue was found in a barred owl from West Vancouver, part of the Fraser Delta–Urban area. The highest bromadiolone liver residue was found in a barred owl from Surrey, part of the Fraser Delta–Agricultural area, where mouse and rat infestations are common. SGARs are used in both urban and agricultural areas to control rodent problems, and can be purchased in Canada without a license. Brodifacoum is registered in Canada for use in and around industrial and commercial establishments, food service establishments, and garbage dumps, and bromadiolone is registered in Canada for use in commercial and residential buildings as well as around transportation facilities (LiphaTech Inc. 2007). Both brodifacoum and bromadiolone are acutely toxic, single-feed SGARs, and were the most commonly detected in our study. Additionally, the highest incidences of detectable residues were in owls from Abbotsford, an area of the Upper Fraser Valley, which has a heavy concentration of intensive agriculture, particularly poultry and dairy operations, as well as substantial urban and suburban areas. This finding could be due to one of two things: (1) sample submissions were more common, likely due to the additional help we had in this area with respect to searching for bird carcasses, (2) bromadiolone and brodifacoum are more commonly used for commensal rodent control than other anticoagulant rodenticides in urban and agricultural settings, in comparison with less developed areas. Therefore, there may be more rodents poisoned and subsequently more secondary exposure of owls in these areas, especially with SGARs. Of the three species of owls in this study, barred owls are the most likely to inhabit urban and suburban areas (Campbell et al. 1990), which may explain the high levels of SGARs found in barred owls.

There is limited population information on barred and great horned owls in British Columbia and the Yukon. However, in a study assessing how change in land use is affecting barn owls in the Fraser Valley of B.C., spring and summer surveys of potential barn owl nesting sites in Delta and Surrey areas in 2007 suggest that there is a decline in the percent of occupancy and breeding when compared with surveys from 1991 to 1993 (Hindmarch, in prep.). Despite the relatively large numbers of barn owls in B.C. compared with the rest of Canada, numbers may be declining (Campbell and Campbell 1984). Factors limiting barn owl populations in B.C are urban development, agricultural intensification, loss of nest sites, and pesticide use, which includes rodenticides (COSEWIC 2001).

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