

**Second generation anticoagulant rodenticides in tawny owls
(*Strix aluco*) from Great Britain**

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Abstract

Secondary exposure of vertebrate predators to second-generation anticoagulant rodenticides (SGARs) is widespread in Britain. Tawny owl (*Strix aluco*) populations in the UK have declined since the 1970s, when SGARs were first introduced, and these compounds may have contributed to the decline in owl numbers. Our aims were to conduct the first systematic survey of SGAR exposure in tawny owls and ascertain whether there had been a change in the proportion of exposed birds that was concurrent with the decline in the population. Liver difenacoum, bromadiolone, flocoumafen and brodifacoum concentrations in British tawny owls from two periods (1990-1993 and 2003-2005) were quantified. In total, some 20% of birds contained detectable residues of one or more SGAR. The extent of exposure (% of birds exposed, magnitude of residues) to different SGARs did not change consistently between time periods. Of the raptors analysed to date in Britain, tawny owls had the lowest proportion of individuals that contained detectable liver residues and so appear to be the least vulnerable to exposure and/or assimilation of SGARs. We found no clear evidence of an association between SGAR exposure and a decline in tawny owl numbers in Britain between 1990 and 2005.

Key words: anticoagulant rodenticide, tawny owl, UK, liver residues, population change

1. Introduction

Second-generation anticoagulant rodenticides (SGARs) were developed and used following the increasingly widespread development of resistance amongst rodents to older (first-generation) anticoagulant rodenticides, such as warfarin (Cowan et al., 1995). SGARs have been in use in Britain since the mid-1970s. Like warfarin, they are derived from coumarin but are approximately 100-1000 times more acutely toxic and have relatively long biological half-lives in tissues such as the liver (Parmar et al., 1987; Huckle et al., 1988; Huckle et al., 1989; Eason et al., 1995). Both characteristics enhance the potential for these compounds to cause secondary poisoning in predators. Rodents typically die 3-8 days after ingesting a lethal SGAR dose (Redfern et al., 1976) and so can be captured and eaten by a predator during this time; some predators may also scavenge the carcasses of poisoned animals.

There is now clear evidence that substantial proportions of the populations of various avian predators are unintentionally exposed to SGARs. Raptors in which relatively widespread exposure has been documented in Britain include the barn owl (*Tyto alba*), buzzard (*Buteo buteo*), red kite (*Milvus milvus*), and kestrel (*Falco tinnunculus*) (Newton et al., 1999; Shore et al., 2000; Shore et al., 2006). The liver concentrations in many individuals are relatively small and are thought to reflect sub-lethal exposures, but larger residues have been found in some individuals and SGARs diagnosed as the likely cause of death (eg., Barnett et al., 2005, 2006, 2007). The exposure pathways for different raptor species have not been clearly defined but red kites in Britain are known to scavenge common rats (*Rattus norvegicus*) and other carrion (Carter, 2001; Pain et al., 2007). Other raptors take few rats and are probably exposed by eating non-target small mammals such as wood mice (*Apodemus sylvaticus*), and bank voles (*Myodes glareolus* - former genus name *Clethrionomys*

(Wilson et al., 2005; Pavlinov, 2006). Brakes & Smith (2005) demonstrated that almost half of the local population of these non-target small mammals fed upon rodenticide baits used during rat control around farm buildings and pheasant feeders.

The tawny owl (*Strix aluco*) is a specialist nocturnal hunter of woodland rodents. In broadleaf woodland habitat, tawny owls feed predominantly on bank voles and *Apodemus* spp. although they also take greater numbers of *Microtus* spp in open and fragmented woodlands with grassland patches (Petty, 1999). This dietary overlap between tawny owls and other raptor species known to be exposed to SGARs means it is likely that tawny owls are likewise widely exposed to SGARs.

Long-term population monitoring has indicated that the tawny owl population in the UK has declined since the early 1970s and that this decline may have accelerated since the late 1990s (British Trust for Ornithology, 2007). This period coincides with the introduction and increasing use of SGARs in Britain and the associated exposure of predatory birds and mammals to these compounds (Shore et al., 2003a,b). It is therefore possible that SGARs may have contributed to the decline in owl numbers but, as far as we are aware, exposure of tawny owls to SGARs has not previously been systematically studied in Britain or elsewhere. Our aims in this study were to determine: (i) the level of exposure of tawny owls to SGARs; (ii) whether the severity of exposure has changed between 1990-3 and 2003-5, part of the period over which UK tawny owl populations declined most rapidly, and (iii) the extent of exposure of tawny owls compared to that of barn owls and kestrels, two other species known to be widely exposed to SGARs in Britain (Newton et al., 1999; Shore et al., 2005).

Methods

Tawny owl carcasses were collected through the Predatory Bird Monitoring Scheme (<http://pbms.ceh.ac.uk/>), a long term UK chemical monitoring programme. The livers from 172 tawny owls were selected for analysis. The samples were from birds that had died either in the years 1990-93 or 2003-05, inclusive. These periods spanned the time (1994-2002) when the UK tawny owl population underwent an estimated 35% reduction (British Trust for Ornithology, 2007). The birds were diagnosed as having died from a variety of causes, but mostly as a result of collisions with motor vehicles or other objects (55% of the sample), starvation (17%) or unknown trauma (10%).

McDonald *et al.* (2000) demonstrated that 84-89% of gamekeepers use rodenticides during the winter and spring (December-May inclusive) compared to 37-45% in summer and autumn (June-November inclusive). We therefore stratified the samples of owls that were analysed so as to eliminate any potential seasonal biases in exposure that might be associated with seasonal variation in use (Table 1). This stratification ensured that the proportion of owls found dead in the winter/spring and in the summer/autumn did not differ significantly between the 1990-93 and 2003-05 sampling periods (Fisher's Exact test, $P=0.352$). The age and sex of each tawny owl was determined by macroscopic observation based largely upon moult pattern and identification of gonads, respectively.

Liver concentrations of difenacoum (CAS No. 56073-07-5), bromadiolone (CAS No. 28772-56-7), brodifacoum (CAS No. 56073-10-0) and flocoumafen (CAS No. 90035-08-8) were determined by high-performance liquid chromatography (HPLC) following extraction methods described by (Hunter, 1985) and subsequently modified by Jones, (1996). For each liver sample, approximately 1 g of tissue was

weighed, ground to a homogenous paste with furnace-cleaned sand, and mixed with furnace-cleaned anhydrous sodium sulphate to form a free-flowing mixture. Approximately 20 ml of extract solvent (30% v/v HPLC grade acetone in dichloromethane; Rathburn Chemical Co. Ltd, Walkburn, Scotland) was mixed thoroughly with the sulphated mixture, left to stand for one hour, and then collected through glass wool and sodium sulphate into a measuring cylinder. The sulphated mass was repeatedly washed with further 20 ml aliquots of extract solvent which were added to the measuring cylinder until a total of 100 ml was collected. The extract was then mixed, left to stand for approximately 12 hours, and a sub-sample (25% or 50%) was reduced to zero volume; the lipid content was determined gravimetrically.

The reduced extract was re-dissolved in 2-3 ml of the extract solvent and cleaned up using a SPE LC-alumina-N column (Supelco UK, Poole, Dorset) that had been pre-conditioned with the extraction solvent. The re-dissolved extract was poured on to the column which was then washed with 2 ml of acetone:dichloromethane (75:25) and eluted with 3 ml of 5% acetic acid in methanol. The eluate was reduced to dryness by standing in a water bath under a stream of nitrogen, re-suspended in 0.5 ml methanol, and analysed by HPLC (HP Series 1100, Agilent Technologies, Bracknell, Berkshire) using a Hypersil ODS (C18) 250 mm x 4.6 mm 5 μ column (Alltech Associates Applied Science, Carnforth, Lancs) at 27°C. A 15 μ l aliquot was injected onto the column using 76:24 methanol:water (v/v), supplemented with 0.25% (v/v) acetic acid and 40 mM ammonium acetate, as the mobile phase pumped at 1.1 ml min⁻¹ isocratically.

Rodenticides were detected by fluorescence spectrometry (HP 1100 series fluorescence detector) using three excitation wavelengths (310, 320 and 350 nm) and a single emission wavelength (390 nm). The excitation wavelength of 310 nm gave

the greatest emission signal at 390 nm and was used for quantification. The ratio the emission response elicited by the 320 nm wavelength to that elicited by 310 nm and the ratio elicited by 350 nm to that elicited by 310 nm were both used to aid identification. Each rodenticide was separated into two isomeric peaks. As the ratios of the isomers in the standard material were not known, individual isomer concentrations could not be quantified. Therefore, the detector response for each isomer was measured and summed to give a total response for the compound. Quantification was by comparison of peak height of external standards and samples. A chromatographic peak was identified as a rodenticide if the absolute retention time of the peak fell within the retention time window of the calibration standards, and if the ratios of the signals for each excitation wavelength matched the ratios in the calibration standards.

Procedural blanks containing reagents only were analysed to detect possible contamination during the sample preparation. Duplicate liver samples were spiked with known concentrations of rodenticides and analysed to determine sample matrix interference and % recovery data; average recoveries were typically between 60 and 70%. For each compound, the Limit of Detection (LoD) was determined from the linear regression of the multilevel calibration using the equation $Y=Y_0 + 3S_{y/x}$ where Y is the LoD response, Y_0 is the Y intercept and $S_{y/x}$ is the standard error of the regression line; LoDs for the individual compounds were 0.045, 0.013, 0.002 and 0.050 $\mu\text{g/g}$ ww for bromadiolone, difenacoum, flocoumafen and brodifacoum, respectively. All concentration data are presented on a wet weight basis.

The frequency of occurrence and magnitude of liver SGAR in tawny owls from the present study was compared with that for barn owls and kestrels over similar time periods. Data for barn owls and kestrels were obtained from the long-term

monitoring data collected by the PBMS (Shore et al., 2005). When making comparisons between species, the LoDs for the tawny owls were applied to the barn owl and kestrel data so that small differences in the LoDs between studies did not introduce error into the statistical comparison of the datasets.

Results

Of the 172 tawny owl livers analysed, 33 (19.2%) contained detectable concentrations of one or more SGAR (Figure 1). The % occurrence of individual rodenticides was 11.6%, 5.8%, and 4.7% for bromadiolone, difenacoum, and brodifacoum, respectively. Flocoumafen was not detected in any tawny owls. Most livers contained detectable residues of only one SGAR, but five livers contained two rodenticides; none contained three or more compounds. There was no significant difference in the proportion of owls with detectable SGAR residues between summer/autumn and winter/spring in the early 1990s, the 2003-5 sample, or for both sampling periods combined (Fisher's Exact Test, $P > 0.05$ in all cases).

The percentage of tawny owls that had detectable liver residues of one or more SGARs was approximately 20% and did not differ significantly between the 1990-1993 and 2003-05 sampling periods (Table 2, Fishers Exact test, $P = 0.352$). When the occurrence of individual compounds was examined, there was again no clear evidence of changes over time. The frequency with which difenacoum was detected was almost identical in the two sampling periods. Although the frequency of occurrence of brodifacoum and bromadiolone in owls in the 1990s was higher and lower, respectively, than in birds from 2003-5 (Table 2), the differences were not statistically significant (Fishers Exact test, $P > 0.05$ in both cases).

In birds with detectable residues, there were no significant differences between

the two sampling periods in the magnitude of liver bromadiolone (student t test, $t_{(18)} = 1.117$; $P=0.279$), difenacoum ($t_{(8)}=0.477$; $P=0.646$) or sum SGAR ($t_{(21)} = 0.198$; $P=0.845$) concentrations. Only one liver from the 2003-05 tawny owl sample contained detectable concentrations of brodifacoum (0.103 $\mu\text{g/g}$ wet wt) and this was within the 95% confidence interval for the brodifacoum residues measured in livers from the early 1990s. Thus there was no evidence of any increase over time in the amount of any individual SGAR accumulated by birds that had been exposed. Overall, the geometric mean sum SGAR concentration in tawny owls was 0.125 $\mu\text{g/g}$ wet wt (Figure 2), equivalent on a (nano)molar basis to 0.247 nmoles/g wet weight.

Comparisons between species of the scale of exposure to SGARs were conducted for each sampling period separately. In the early 1990s, there were significant differences between tawny owls and barn owls in the proportion of birds that contained specific SGARs, but the differences were not consistent across compounds (Table 2). A higher proportion of tawny owls than barn owls had brodifacoum residues in their livers (Fisher's Exact test, $P<0.01$) whereas the reverse was true for difenacoum ($P=0.051$). The proportion of birds with liver bromadiolone residues was similar in both species ($P=0.538$). Overall, the proportion of birds with detectable residues of one or more SGAR did not differ significantly between tawny and barn owls ($P=0.188$). In birds that died between 2003 and 2005, difenacoum was still detected more frequently in barn owls than tawny owls ($P=0.002$), and there was again no significant difference between the species in the frequency with which bromadiolone was detected. Unlike in the 1990s, there was also no significant difference between tawny owls and barn owls in the proportion of birds that had detectable concentrations of brodifacoum. Overall, the proportion of barn owls that contained one or more SGAR rose by approximately half between the early 1990s and

2003-05 whereas there was no such increase for tawny owls.

Kestrels were monitored by the PBMS for SGARs during the period 2003 to 2005 but not in earlier years (Table 2). The proportion of kestrels that had liver residues of one or more SGAR was more approximately twice that in barn owls and more than three times greater than in tawny owls (Chi Squared test: $\chi^2_{(2)}=39.9$, $P<0.0001$). Brodifacoum, bromadiolone and difenacoum were all detected two-three times more frequently in kestrels compared with tawny owls.

Critical liver SGAR concentrations associated with adverse effects and mortality have not been defined for most raptor species. However, the residue considered to be in the potentially lethal range for barn owl has variously been described as $> 0.1 \mu\text{g/g}$ wet wt (Newton et al., 1998) and $> 0.2 \mu\text{g/g}$ wet wt (Newton et al., 1999). This has been classified on the basis of two sets of observations. These are that barn owls diagnosed at post-mortem of having died from rodenticide poisoning (because they had characteristic signs of haemorrhaging from such organs as the heart, lungs, liver, brain and/or subcutaneous areas) almost all had liver residues $>0.1 \mu\text{g/g}$ wet wt, and, secondly, that owls that had been experimentally poisoned had residues of the range $0.2\text{-}1.72 \mu\text{g/g}$ wet wt (Newton et al., 1999 for review). If the criteria for barn owls were applied to the data for the tawny owls, 21 birds (12.2% of the sample) had liver SGAR residues (different compounds summed) that were $>0.1 \mu\text{g/g}$ wet wt, and 10 of these birds had summed residues that were $> 0.2 \mu\text{g/g}$ wet wt. Of these 10, nine either did not show signs of haemorrhaging or the haemorrhaging that was apparent was related to physical trauma. However, one tawny owl had haemorrhaging, a brodifacoum liver concentration of $1.2 \mu\text{g/g}$ wet wt., and was found close to a site where brodifacoum had been used prior to the bird being found. Given these factors

and the lack of any other apparent cause of death, it is probable that this bird had been fatally poisoned by brodifacoum.

Discussion

This study is the first large scale survey of SGARs in tawny owls and indicates that, over the time periods that birds were collected, at least 19% of birds were exposed to SGARs. A small number of tawny owls (27) from Scotland that died between 1987 and 2005 have also been analysed as part of the Wildlife Incident Investigation Scheme (WIIS) monitoring in Scotland and five (18.5%) contained residues of SGARs in their livers (Hunter *unpub. data*). These findings are very similar to those of the current study, although the sampling period and analytical methods differed. Neither the proportion of tawny owls with = one liver SGAR residue, nor the liver SGAR concentrations in birds with detectable residues, changed significantly between 1990-1993 and 2003-05. This lack of change over time contrasts with that observed for barn owls, but is consistent with the lack of change in exposure in polecats *Mustela putorius* during the 1990s (Shore et al., 2003b).

The proportion of tawny owls exposed to SGARs appears to be lower than that observed in other avian predators of small mammals, such as the barn owl and kestrel, and in avian scavengers such as buzzards and red kites. The proportion of individuals with liver SGARs in these species varies between 40% and 70 % (Newton et al., 1999; Shore et al., 2000; Shore et al., 2006). However, the frequency of occurrence of SGARs in tawny owls was broadly similar to that of a mammalian predator, the weasel *Mustela nivalis* that, like tawny owls, feeds predominantly on *Apodemus spp.* and voles. McDonald et al. (1998) found that one out of 10 weasels analysed contained detectable liver SGAR concentrations, although three contained

coumatetralyl, a first generation anticoagulant rodenticide. Overall, the relatively low incidence of exposure of tawny owls to SGARs, lower than that of other species (such as the barn owl and buzzard) which have not declined in numbers despite a higher scale of exposure, suggests that SGARs are unlikely to have been a prime cause for the 35% reduction in tawny owl numbers that has occurred in UK between 1994 and 2002 (British Trust for Ornithology, 2007). This conclusion assumes that tawny owls are not more sensitive to SGARs than other species; laboratory mammals can vary markedly in their sensitivity to SGARs (World Health Organisation, 1995) but almost nothing is known about the relative sensitivity of different raptor species. However, the lack of any rise in exposure between 1990-93 and 2003-05, despite the apparent acceleration in population decline since the late 1990s (British Trust for Ornithology, 2007), also suggests that SGARs have not been a major factor driving the decline in tawny owl numbers in Britain since 1990.

Given that tawny owls predominantly hunt in woodlands (Petty, 1999), it might be assumed that their most likely route of exposure is from rodenticide use in and close to woodland, and so is associated with game-rearing. Thus, SGAR exposure in tawny owls may particularly reflect gamekeeper use of rodenticides. If so, the apparent lack of change in SGAR exposure over time in tawny owls has significant implications when interpreting exposure in other species, and specifically suggests that the increase in exposure in barn owls over time must be due to factors other than gamekeeper use. However, care is needed when assuming that contamination of tawny owls reflects SGAR use by gamekeepers. The relationship between usage patterns and occurrence of liver residues in predators is complex and potentially affected by physiological, ecological and anthropomorphic factors (Shore et al., 2003b; Shore et al., 2006). Furthermore, in the present study, there were no seasonal

differences in the frequency and magnitude of liver SGARs in tawny owls even though gamekeepers apparently vary their intensity of rodenticide use during the year (McDonald et al., 2000). Given the long half-lives of SGARs in the liver, a lack of detectable seasonal variation in contamination in tawny owls is not surprising, but may indicate that non-gamekeeping use of SGARs also contributes significantly to tawny owl exposure.

Conclusions

Our data indicate that at least one fifth of tawny owls in Britain have been exposed to SGARs, as indicated by the presence of liver residues. There is no evidence that the extent of exposure (in terms of proportion of birds exposed or magnitude of residues) has changed significantly over approximately the last 15 years. These data indicate that, of the raptors analysed to date in Britain, tawny owls appear to be the least vulnerable to exposure and/or assimilation of SGARs. However, the presence of high liver residues in a small proportion of tawny owls suggests that SGARs may kill some individuals. This is also evident from the diagnosis by the WIIS of SGAR poisoning in a few tawny owls in some years (Barnett et al., 2005, 2007). Overall, the apparent relatively low level exposure/assimilation of SGARs, the lack of any large-scale mortality as detected by the WIIS, and the lack of any increase in exposure concurrent with the accelerated decline in numbers post-1999, leads us to conclude that there is no clear evidence to implicate SGARs as a major factor affecting tawny owls numbers in Britain between 1990 and 2005.

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Table 1

Summary for each sampling period of age, sex and the season and year when found for tawny owls analysed for second generation anticoagulant rodenticides.

		2003-5			1990-1993		
		Winter/ Spring	Summer/ Autumn	Unknown	Winter/ Spring	Summer/ Autumn	Unknown
	All	32	50	1	41	46	2
Male	Adult	15	12	-	25	10	1
	Juvenile ^a	1	4	1	4	3	-
	Unknown	1	-	-	-	-	-
Female	Adult	10	15	-	10	11	1
	Juvenile ^a	5	13	-	2	19	-
	Unknown	-	1	-	-	-	-
Unknown	Adult	-	-	-	-	-	-
	Juvenile ^a	-	3	-	-	0	-
	Unknown	-	2	-	-	3	-

^a Owls were classed as juveniles if they hatched during the same or previous calendar year that they were found.

Table 2

The numbers of tawny owls, barn owls and common kestrels in 1990-1993 and 2003-2005 with detected and non-detectable liver residues of difenacoum (Difen), bromadiolone (Brom), flocoumafen (Floc) and brodifacoum (Brodif). Data for kestrels and barn owls are from the long-term PBMS monitoring as most recently summarised by Shore et al. (2005). Rodenticides were not quantified in the livers of kestrels during the early 1990s.

Species		1990-1993					2003-2005				
		Brom	Difen	Floc	Brodif	=one SGAR	Brom	Difen	Floc	Brodif	= one SGAR
Tawny Owl	Detected	7	5	0	7	16	13	5	0	1	17
	ND ^a	81	83	88	81	72	71	79	84	83	67
	% detected	8.0%	5.7%	0.0%	8.0%	18.2%	15.5%	6.0%	0.0%	1.2%	20.2%
Barn Owl	Detected	25	33	5	3	60	28	39	0	6	64
	ND	212	204	232	234	177	166	155	194	188	130
	% detected	10.5%	13.9%	2.1%	1.3%	25.3%	14.4%	20.1%	0.0%	3.1%	33.0%
Kestrel	Detected	-	-	-	-	-	22	37	0	8	49
	ND	-	-	-	-	-	51	36	73	65	24
	% detected	-	-	-	-	-	30.1%	50.7%	0.0%	11.0%	67.1%

^a ND indicates number of liver in which SGARs were not detected

Figure Legends

Figure 1. Percentage (%) of tawny owls (both sampling periods combined) with detectable liver concentrations of bromadiolone, difenacoum, flocoumafen, brodifacoum or one or more of these rodenticides (= one SGAR). No owls contained detectable liver concentrations of flocoumafen.

Figure 2. Geometric mean ($\pm 95\%$ CI) concentrations of difenacoum, bromadiolone, brodifacoum, and summed SGAR concentrations in tawny owl livers sampled in 1990-93, 2003-05 and for both periods combined. Flocoumafen was not detected in any tawny owl livers, while only one liver from owls that died in the 2003-05 period contained detectable residues of brodifacoum.

Figure 1

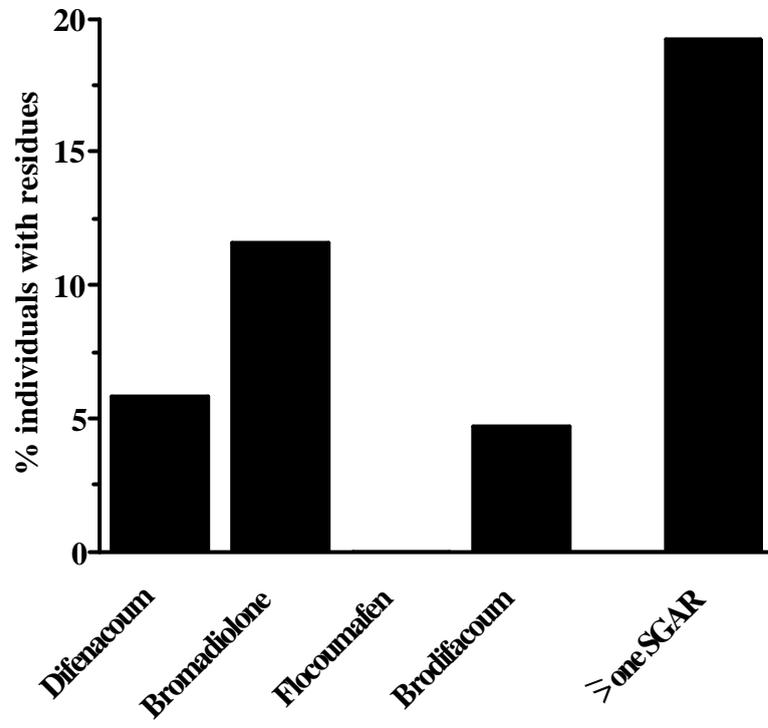


Figure 2

