Ecotoxicology (2012) 21:1325–1332
DOI 10.1007/s10646-012-0886-3

Rodenticide exposure in wood mouse and house mouse populations on farms and potential secondary risk to predators

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Accepted: 8 March 2012 / Published online: 24 March 2012

Abstract We compared capture rates and exposure to SGARs of wood mice (*Apodemus sylvaticus*) and house mice (*Mus domesticus*) in autumn/winter on farms that currently used, had previously used, and never used SGARs. 6–10 weeks after baiting programmes began, 15 % of 55 wood mice and 33 % of 12 house mice had detectable liver SGAR residues. Wood mice with residues occurred on farms not using rodenticides, reflecting the high mobility of these animals, and four had multiple liver residues, possibly due to cross-contamination of baits. The winter decline in wood mouse numbers was similar on farms that did and did not use SGARs, suggesting little long-term impact of SGARs on populations on farms. Our results indicate residual levels of rodenticides will be ever present in small mammal prey across the agricultural landscape unless all farms in a locality cease application. The implications for secondary exposure and poisoning of predators are discussed.

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Keywords Anticoagulant rodenticide · *Apodemus sylvaticus* · Population numbers · Secondary exposure · Secondary poisoning

Introduction

Anticoagulant rodenticides (ARs) are widely used to control commensal rodent populations. Two groups of ARs exist, First and Second-Generation Anticoagulant Rodenticides (FGARs and SGARs, respectively). In the United Kingdom (UK), where the level of AR use is high, SGARs are the most widely used group (Dawson et al. 2001; McDonald and Harris 2000; Tosh et al. 2011a). Although biochemically similar to FGARs, SGARS are used more widely because of their greater acute toxicity (Fisher et al. 2003; Parmar et al. 1987) and the lower degree of resistance to these compounds in UK rodent populations (MacNicoll 1986). However, the longer persistence and greater acute toxicity of SGARs has led to concern about the risk to predators from secondary exposure to SGARs (Shore et al. 2003a; 2006).

The exposure of a range of non-target predatory mammals and birds to SGARs is now well documented (Berny et al. 1997; Dowding et al. 2010; Fournier-Chambrillon et al. 2004; McDonald et al. 1998; Shore et al. 1999, 2003a, b; Stone et al. 2003; Thomas et al. 2011; Walker et al. 2008a, b, 2010). The main route of exposure for many predators is thought to be indirect and occur via predation of exposed prey. This secondary exposure can arise either from the predation of target species, which in the UK are the house mouse (*Mus domesticus*) and brown rat (*Rattus norvegicus*), or non-targets such as wood mice (*Apodemus sylvaticus*), and bank voles (*Myodes glareolus*) which can be inadvertently exposed to ARs during routine control of

commensal rodents (Brakes and Smith 2005; Harradine 1976; Townsend et al. 1995). Predators such as the barn owl (*Tyto alba*) and kestrel (*Falco tinnunculus*) eat commensal rodents only occasionally (Love et al. 2000; Yalden and Warburton 1979) and so are therefore thought to be exposed mainly through eating non-target small mammals. Prevalence of exposure varies amongst different non-target small mammal species and tends to be greater in wood mice than in vole populations (Brakes and Smith 2005; Harradine 1976; Townsend et al. 1995). Therefore, the risk of exposure through predation of non-target small mammals may be greatest for those species that mostly eat wood mice.

Assessing the risk of AR exposure to predators feeding on non-target small mammals is limited by a lack of knowledge of prey exposure. There have been laboratory and field studies on exposure and effects in commensal rodent populations (Greaves et al. 1982; Rowe et al. 1981, 1985) but their applicability, if any, to wild populations of non-target small mammals is unclear. While there have been a small number of studies that have investigated exposure of non-target small mammals through baits targeted at commensal rodents (Cox and Smith 1990; Brakes and Smith 2005; Harradine 1976; Townsend et al. 1995), only two (Cox and Smith 1990; Townsend et al. 1995) involved SGARs. Both found that non-target small mammal species could be exposed to SGARs but replication was very limited and neither study reported residues in animals likely to be eaten by predators, key information when assessing the risk to predators from secondary exposure.

To improve our understanding of the risk ARs may pose to predatory species, we investigated the exposure of small mammal populations to ARs on farms where patterns of AR usage were typical of farmers (Tosh et al. 2011a). The aims of the study were to: (1) investigate whether AR use has an effect on the relative abundance of small mammal populations around farms; (2) determine whether small mammals are exposed to ARs as a result of typical AR usage; (3) examine the spatial distribution of exposed small mammals in relation to AR application.

Methods

Study site

Small mammals were captured on 16 farms in County Down, Northern Ireland (NI). All the farms were predominantly pastoral and reared some combination of beef cattle, dairy cattle, sheep or horses. Eight farms used ARs during the study (current usage), four had previously used ARs but did not use them during this study (previous usage) and four did not use ARs (no usage). All current usage farms used bromadiolone ARs but current usage farms 2, 7 and 8 also used difenacoum and farms 1 and 3 also used flocumafen ARs. All previous usage farms (1–4) used bromadiolone baits but farm 3 also used a difenacoum based AR. On the no usage farms, farmers did not use ARs because they did not consider rodents to be a problem, did not like to use chemicals or preferred other means of control such as cats or traps. No usage farms were 150–448 m (mean = 288 m) from neighbouring farms that did use ARs. AR use on the other study farms involved application of baits during the autumn or winter months in response to colder weather or an increase in sightings of rodents. On each farm, AR usage was confined to application in and around (along the outside walls of buildings) up to three buildings containing grain, animal feed or livestock. No more than three bait points were located in and around each building. All baits used along the outside walls of buildings were contained within either pipes or manufacturers bait boxes. All bait used indoors was also contained within bait boxes apart from on one farm (current 3) that laid down unprotected (in sachets) within rooms.

Assessment of wood mouse abundance

The relative abundance of wood mice and house mice was assessed on each farm before and after baiting (November/December 2007 and January/February 2008). Trapping before and after baiting therefore took place 10 or 11 weeks apart on each farm. Trapping was conducted along two 260 m long trap lines, each with ten trap points and two Longworth traps per point. Each line ran from the edge of the farmyard to an adjacent hedgerow and then along the hedgerow. Trapping was focused along hedge boundaries as the grazed interiors of pasture fields are unsuitable for small mammals in NI (Montgomery and Dowie 1993a). The first point on each trap line was located inside a building, the second adjacent to the building, and the remaining eight points were spaced at 15, 30, 45, 60, 110, 160, 210 and 260 m along the trap line. Traps were provisioned with hay bedding and baited with a mixture of oats and carrots as food for mice and fly larvae in case of incidental capture of pygmy shrews (*Sorex minutus*). Traps were checked twice daily for three nights (120 trap nights) per session per farm. Captured animals were sexed and marked with a fur clip to identify whether they were new or recaptured animals.

As population estimates were only made over 3-day periods, minimum number alive (MNA) was used as the measure of abundance for this study; MNA estimates of abundance typically correlate closely with estimates of small mammal population size that are derived from more

complex statistical models (Hopkins and Kennedy 2004; Montgomery 1989, Slade and Blair 2000).

Assessment of mouse exposure and spatial distribution of contamination

Individuals caught on the final day of the second trapping session on each farm were humanely killed and analysed for rodenticide residues to determine their level of exposure to SGARs. These mice were weighed to the nearest 0.1 g, sexed, and body length (distance from nose to anus in millimetres) was recorded. Livers were excised from animals and stored at −20 °C in glass vials until analysis.

The liver of each mouse that was analysed was defrosted, homogenised and a subsample (~0.25 g) was weighed accurately. Rodenticide residues were extracted by grinding the subsample with 2.0 g of anhydrous sodium sulphate to form a free flowing powder. This was mixed with 5 ml of 1:1 chloroform–acetone, vortexed for 10 s and shaken for 30 min. Extracts were spun at 11,000 rpm for 5 min and the supernatant was removed to a 10 ml test-tube in which it was blown down to dryness at 40 °C under nitrogen. 1 ml of 1:1 chloroform–acetone was added to re-dissolve the lipid fraction, the tube was vortexed for 30 s, 4 ml of acetonitrile was then added and the tube was again vortexed for 30 s. Clean up used a 1 g/6 ml C-18 SPE column and a 200 mg/3 ml Phenomenex Stratta X-WA column in combination. Columns were conditioned with 6 ml of first methanol and then acetonitrile at 4–10 ml/min. The sample extract was added to the C-18 column (flow rate <4 ml/min), washed twice with acetonitrile, the C-18 column was then removed and 4 ml of ammoniacal methanol was added to the X-AW column. The eluant was collected, evaporated to dryness, re-suspended in 1 ml of LCMS-MS mobile phase (1:1 water–methanol both containing 10 mM ammonium acetate) and filtered (13 mm PTFE, 0.2 µm filter). A 5 µl aliquot was injected into a triple quadrupole LCMSMS (ThermoFisher Scientific, Quantum Ultra with Accela LC) and separated on a Hypersil Gold column (50 × 2.1, 1.9 µm, ThermoFisher Scientific) at 50 °C using a gradient from 70/30 water/methanol (both containing 10 mM ammonium acetate) to 100 % methanol over 15 min, at 300 µl/min. Determination was carried out using atmospheric pressure chemical ionisation in negative mode and compound quantification and confirmation used Multiple Reaction Monitoring. External calibration was with standards in 50/50 methanol/water in 10 mM ammonium. All liver rodenticide concentrations are expressed as µg/g wet weight (wet wt.).

The limits of detection (LoD) in µg/g wet wt. were calculated for each AR using the method described in Dowding et al. (2010) and were 0.002 for warfarin, coumatetralyl, chlorophacinone, difenacoum and flocoumafen, 0.004 for bromadiolone and brodifacoum, and 0.005 for diphacinone. To assess losses of rodenticide due to the method, six uncontaminated chicken liver samples were spiked with rodenticides, extracted, cleaned up and analysed. Mean (±SE) recoveries varied between compounds but were highly consistent for each compound; recoveries were 60.6 (±3.9), 70.0 (±2.0), 46.6 (±8.7), 43.6 (±6.2), 34.1 (±5.6) and 31.4 (±5.9) % for warfarin, coumatetralyl, bromadiolone, difenacoum, flocoumafen and brodifacoum respectively. Data were recovery corrected. This analytical method is not designed to quantify chlorophacinone and diphacinone and the analytical method was highly variable for these compounds (20.5 ± 10.3 and 35.1 ± 29.9 %, respectively). Therefore, they were simply reported here as present or absent.

Bait analysis

To determine whether the active ingredient in applied bait matched the residues found in mice on the same farm, seven whole grain (6 bromadiolone, 1 difenacoum), two cut wheat (difenacoum) and one pellet (bromadiolone) bait were analysed from current use farms. The protocol for extraction, clean up and analysis of baits was as for livers.

Statistical analysis

Variation in wood mouse captures in the three treatment groups was compared using Kruskal–Wallis tests. Comparison of wood mouse abundance before and after baiting on paired farms was tested with a Wilcoxon matched pairs test with each individual farm as the pairing factor.

Factors affecting the presence or absence of AR residues in individual mice was investigated using a logistic regression model; Presence/absence of ARs was the binary response, mouse weight (g) and distance of the capture point from the farm (m) were covariates while sex and treatment group (current usage, previous usage, no usage) were included as factors in the model. Model selection was made using Aikike's information criterion (AIC) values and *F* tests and fit was assessed using QQ plots and plots of fitted values and residuals. All analyses were conducted using the statistical programme R (R Development Core Team 2009).

Results

Abundance

In all, 363 (191 male, 172 female) wood mice were trapped across all farms before baiting began and 221 mice (119 male, 102 female) were trapped after baiting; 17 % (38) of

wood mice caught after baiting had previously been captured in the trapping session before baiting started. The overall decline in abundance after baiting was significant (Wilcoxon Matched Pairs: $V = 456.5$, $p < 0.001$) but did not differ markedly between treatments; median declines were 40, 35 and 50 % on no usage, previous usage and current usage farms respectively. There was no significant difference in wood mouse captures between the three groups of farms either before (KW = 4.5607, df = 2, $p = 0.102$) or after (KW = 4.9795, df = 2, $p = 0.082$) baiting (Fig. 1).

A total of 126 (77 male, 49 female) house mice were caught before baiting and 25 (17 male, 8 female) after baiting. The (median) numbers of house mice captured before baiting on no usage, previous usage and current usage farms were 6, 6 and 4 respectively and, after baiting, had declined by 77–89 % across all three groups of farms. In addition to the two species of mouse, five brown rats and two pygmy shrews were caught before baiting and three brown rats and one pygmy shrew after baiting.

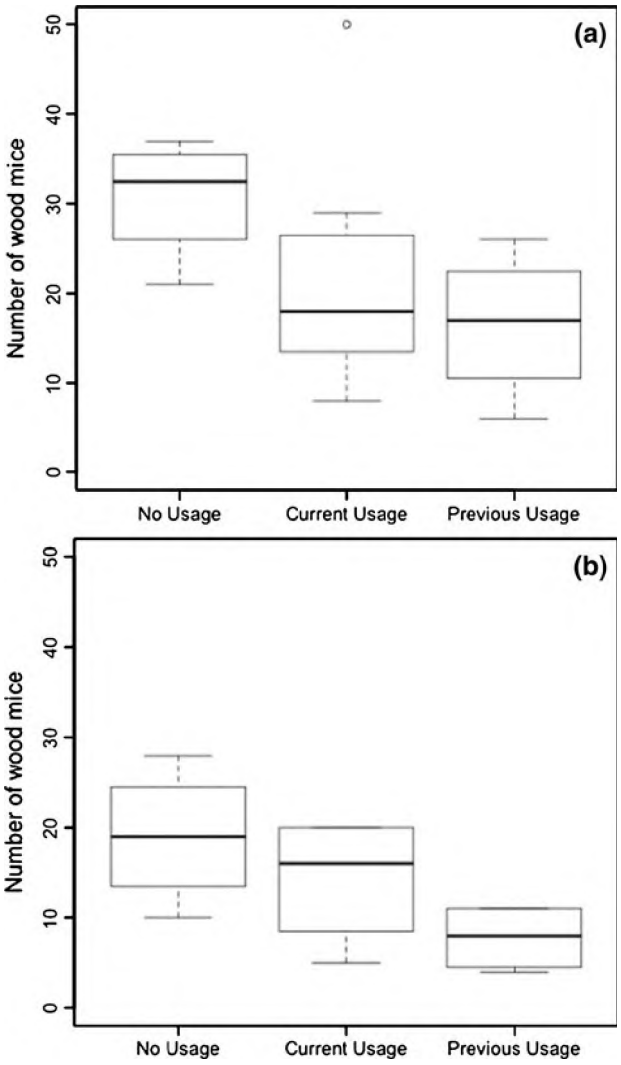

Fig. 1 Numbers of wood mouse on the three farm treatment groups before baiting (**a**) and after baiting (**b**). *Boxes* denote interquartile range, *bold lines* are medians, *whiskers* represent data range and *circles* are outliers

Table 1 Total number of mice analysed and found to contain detectable rodenticide residues

	No usage	Previous usage	Current usage	Overall
Wood mice				
n Tested	18	11	26	55
n Exposed	2	2	4	8
% Exposed	11.1	18.2	15.4	14.5
House mice				
n Tested	8	1	3	12
n Exposed	3	0	1	4
% Exposed	37.5	0	33.3	33.3

Exposure of wood mice and house mice to ARs

In all, 55 wood mice (28 male, 27 female) and 12 house mice (9 males, 3 females) were analysed for ARs (Table 1). Eight wood mice (14.5 %) and four house mice contained detectable liver AR residues (Table 2). Four of these wood mice and one house mice were from no usage or previous usage farms, despite no ARs being used that year on those holdings. All four SGARs and warfarin were detected in both mouse species and brodifacoum occurred most frequently (Table 2). Chlorophacinone, diphacinone and coumatetralyl were not detected in any mice. Multiple ARs were detected in three wood mice and one house mouse. Seven wood mice and three house mice had liver residues of ARs not used on the farms where they were captured (Table 2). Of these, two wood mice and one house mouse contained ARs that were used on neighbouring farms but the remainder contained residues that could not be attributed to adjacent AR use. This included five animals with brodifacoum residues, even though brodifacoum baits were not used on any of the farms where trapping was carried out. However, brodifacoum was detected as a contaminant in four of the ten baits tested and comprised 7.7–13.2 % (mean = 9.8 %) of the total AR detected in the bait. All contaminated baits were of the same brand and levels of brodifacoum contamination ranged from 0.063 to 0.197 μg/g (mean = 0.113 μg/g). No other bait contaminants were identified.

Spatial distribution of captures and contamination

Wood mouse captures increased with distance of the trap point from the farm (Fig. 2a) before ($r^2 = 0.86$, df = 8,

Table 2 Recovery corrected anticoagulant rodenticide residues in the livers of wood mice and house mice from farms in NI

Species	Sex	Farm[a]	AR used on farm	Warfarin	Bromadiolone	Difenacoum	Flocoumafen	Brodifacoum	Total
Wood mouse	F	No usage 2	None	0.009					0.009
	F	No usage 3	None		0.041			0.640	0.681
	M	Previous 3	Difenacoum, bromadiolone					0.016	0.016
	M	Previous 3	Difenacoum, bromadiolone					0.019	0.019
	F	Current 1	Bromadiolone, flocoumafen				0.615		0.615
	M	Current 1	Bromadiolone, flocoumafen			0.014	0.014		0.028
	F	Current 4	Bromadiolone	0.003					0.003
	M	Current 6	Bromadiolone		0.017			0.047	0.064
House mouse	M	No usage 2	None	0.006					0.006
	M	Current 1	Bromadiolone, flocoumafen					0.022	0.022
	M	Current 3	Bromadiolone, flocoumafen		0.006	0.027	0.008		0.041
	M	Current 3	Bromadiolone, flocoumafen			0.015			0.015

[a] Different numbers associated with farm type indicate different farms

$p < 0.001$) and after baiting ($r^2 = 0.96$, df = 8, $p < 0.001$) with ~10 % of wood mice caught inside and around buildings (Fig. 2a). In contrast, the commensal nature of house mice was reflected by the general reduction of captures with increasing distance from farms before ($r^2 = 0.60$, df = 8, $p = 0.005$) and after ($r^2 = 0.31$, df = 8, $p = 0.05$) baiting (Fig. 2b). Some 41–48 % of all house mice captured were caught in and around buildings.

Exposed house mice were typically found closer to farm buildings than exposed wood mice (Fig. 3). All exposed house mice occurred within 30 m of farm buildings whereas all wood mice with detectable AR residues were trapped at least 30 m away from farm buildings. The furthest distance that a contaminated wood mouse was captured away from buildings was 160 m on a no usage farm and 110 m on a current usage farm (Fig. 3). There was no apparent relationship between distance from farm buildings and residue magnitude although this was not surprising given the sample size was small and there was no baiting in the buildings on no usage and previous usage farms. The highest liver AR residues were detected in wood mice that were captured at 60 m (mean = 0.237 μg/g) and 110 m (mean = 0.317 μg/g) from the nearest farm building (Fig. 3). None of the explanatory variables (weight, sex, treatment, distance) in the logistic regression model were able to explain exposure to ARs in the mice tested.

Discussion

In common with previous studies, we have confirmed that non-target wood mice are exposed to ARs during periods of rodent control (Brakes and Smith 2005; Cox and Smith 1990; Harradine 1976; Townsend et al. 1995). In our study, 15 % of wood mice that we caught on farms had detectable liver AR residues. This is similar to the 20–30 % of wood mice found to be exposed to a chemical bait marker associated with AR usage (Townsend et al. 1995). Exposure of wood mice, determined by inspecting faeces for coloured bait marker, was higher (50–70 %) on two farms (Brakes and Smith 2005). However, direct comparison between studies is problematic because numerous factors

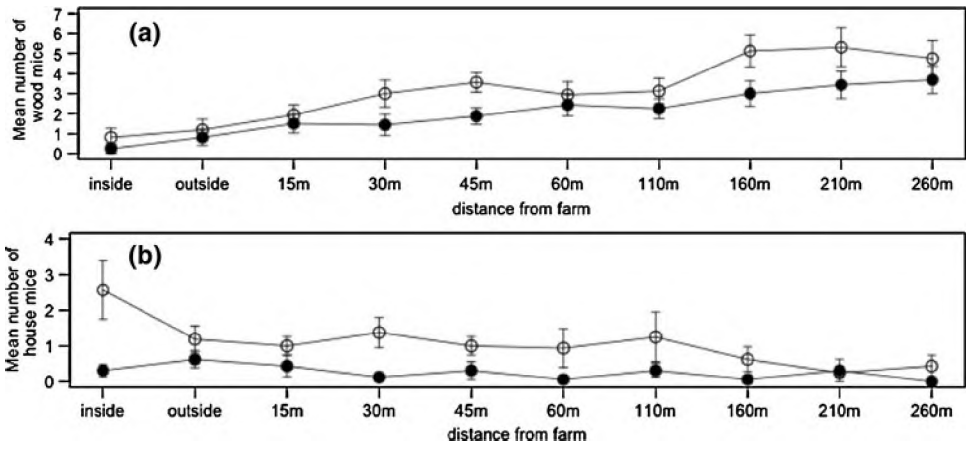

Fig. 2 Mean (and standard error) number of wood mice (*upper* graph) and house mice (*lower* graph) caught at each trapping point in session one (*open circle*) and session two (*filled circle*). Data shown is from all treatments combined

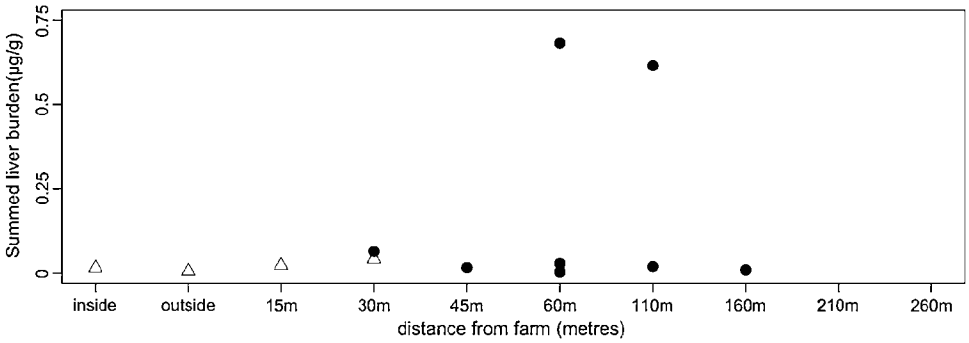

Fig. 3 The distance (m) from farms of exposed wood mice (*circle*) and house mice (*triangle*) were located during the study plotted against the summed liver AR concentrations (µg/g) detected in each exposed mouse. Each individual *symbol* represents one mouse

can potentially alter both the likelihood of, and ability to detect, exposure. These include season, pattern, intensity and duration of baiting, the toxicity of the compounds used, time when exposure is monitored relative to onset of baiting, spatial extent of trapping, population density of non-target species and the methodology used to quantify exposure. The relatively low prevalence of exposure detected in the present study may be partly because trapping extended further away (by at least 2–3fold) from the baited area than in other studies, and because exposure was monitored some 6–10 weeks after the start of baiting. Overall however, our results indicate that contamination persists in non-target species away from farm buildings for months after typical AR baiting. This is perhaps partly because only a third of farmers typically remove baits after infestations have been controlled (Tosh et al. 2011a).

The detection of AR residues in the livers of wood mice on farms demonstrates that restricting AR use to within and around buildings does not eliminate the risk of non-target exposure. Wood mice are highly mobile, especially in agricultural habitats, and have been recorded moving distances of over 1,300 m recorded in one night (Brown et al. 1994; Macdonald et al. 2000; Wolton and Flowerdew 1985). Therefore, farmyards are likely to be within the ranging activity of multiple mice occupying habitats around farm buildings. The home ranges of wood mice on some of our study farms were likely to have incorporated the premises of neighbouring farms. This may account for the detection in some wood mice of ARs that were not used on the study farm but were used on adjacent properties. However, mobility is not likely to be the sole reason for the detection in mice of compounds that were not used on the farms under study. ARs other than the stated active ingredient were found in baits and may also account for the detection of multiple residues in wood mice, especially when livers contained predominantly one compound and only traces of others. This may also partly account for the high frequency of detection of multiple residues in predatory birds and mammals that have been previously attributed to multiple exposure events (McDonald et al. 1998; Tosh et al. 2011b; Walker et al. 2008b, 2010). Our study suggests that the ranging behaviour of prey species and contamination of baits may both be explanatory factors for the presence of multiple compounds in prey and predators. Although it is also possible that contaminated mice were found on some no usage farms because those farms used rodenticides despite claiming not to, we believe this to be unlikely. Our access to the study farms was unrestricted and we observed no signs of bait use on no usage or previous usage farms.

The lack of difference between farms in changes in wood mouse abundance between trapping sessions suggests that declines in numbers were mainly due to natural seasonal changes. Any localised effect of rodenticides on wood mouse abundance may have been quickly offset by immigration from surrounding areas, as has been observed when wood mouse numbers were reduced by autumn applications of methiocarb on arable fields (Johnson et al. 1991). Wood mouse populations peak in late autumn and numbers typically decline to yearly lows in spring as a consequence of reduced food supplies and cold weather (Montgomery 1989; Montgomery and Dowie 1993b; Watts 1969). The way in which ARs were used on farms in this study was typical of agricultural end user behaviour (Tosh et al. 2011a) but was less intensive than that reported in prior studies investigating AR exposure in non-target small mammals (Brakes and Smith 2005; Cox and Smith 1990; Townsend et al. 1995). During field trials of ARs, exposure of target species can be reduced by lowering the rate of application (Merson et al. 1984) and intensity of AR use may have a similar impact on the exposure rate of non-target small mammals. Therefore, the rodent control methods adopted by the majority of farmers, and as employed by farmers in the present study, may have little persistent impact on wood mouse populations.

Studies on the toxicity of ARs to predatory birds and mammals are scant but chronic dietary concentrations (whole body residues in rodents fed to barn owls over several days) that caused some mortality in barn owls are 2.5–14 mg/kg for bromadiolone, 1.6–5.5 mg/kg for difenacoum, 0.78–1.3 mg/kg for flocoumafen and 0.1–0.2 mg/kg for brodifacoum (Luttik et al. 1999). The median liver

concentrations in those mice with detectable residues in the present study were some two–three orders of magnitude lower than the dietary concentrations of bromadiolone, difenacoum and flocoumafen that caused some mortality in owls and 5–10fold lower than the concentrations of brodifacoum that cause mortality. Whole body rodenticide concentrations in these mice would be expected to be lower as the liver tends to contain the bulk of the rodenticide that is sequestered in the body (Giraudoux et al. 2006; Sage et al. 2008) but constitutes only ~5 % of body mass (this study). Given this and that only 14 % of rodents contained detectable rodenticides, it would appear that the risk of secondary poisoning to predators of wood mice some 2–3 months after the onset of baiting is low.

This simple assessment of risk contrasts with the finding that 84 % of foxes from the same geographical area contained AR liver residues and that 16 % contained liver residues of bromadiolone greater than 0.8 μg/g wet weight (Tosh et al. 2011b), a residue level associated with mortality in foxes (Berny et al. 1997). Residues in any wood mice and house mice that were lethally poisoned would likely be higher than in animals captured in the present study and there may be selective predation of dead and moribund rodents. This would result in a greater secondary exposure and poisoning risk to predators, but we have no information on how the abundance and availability of such animals to predators may have changed following onset of baiting on our study farms. Sage et al. (2008) found that the greatest numbers of contaminated water voles (*Arvicola terrestris*), the target species, were found within 2–3 weeks of the onset of extensive baiting. It is possible that, similarly, there is a time-window of high risk of secondary exposure and poisoning on farms during the first couple of weeks after onset of baiting, but this may be localised to farm buildings and not persist. Any such exposure pattern would mean that the period and spatial extent of risk to predators may be relatively discrete and, as such, may lend itself to active mitigation measures.

In summary, this study has demonstrated exposure of non-target small mammals when ARs are used to control commensal species but there was no evidence that typical usage had any significant long-term impact on wood mouse abundance. Our results suggest that the risk of secondary poisoning in predators through consumption of non-target small mammals is relatively low some 6–10 weeks after onset of baiting, even though the majority of farmers state that they do not remove baits after infestations of commensal rodents abate (Tosh et al. 2011a). It is possible that the risk of secondary exposure and poisoning may be greater in the period soon after onset of baiting or following periods of more intensive AR use. It is also evident from our results that the likelihood of secondary exposure is not restricted to locations of AR use. The mobility of small mammal prey maintains the potential of secondary exposure in predators across the agricultural landscape, and it is probable that residual levels of AR will be ever present in small mammals unless all farms in a locality cease their application.

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