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Exposure of non-target small mammals to rodenticides: short-term effects, recovery and implications for secondary poisoning

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Summary

1. Monitoring of exposure to pesticides in many countries shows extensive exposure of predators to anticoagulant rodenticides, which are used to control rats. Many predators and scavengers are declining in numbers, and exposure to rodenticides might therefore be of importance in conservation biology.
2. Predators and scavengers of poisoned rats are at most risk of secondary poisoning. However, several predatory species of conservation concern rarely eat rats, implicating non-target small mammals as the major route of exposure. For the first time, this research investigated the importance of non-target small mammals as routes of exposure to rodenticide for predators and scavengers in the UK.
3. Exposure studies of non-target small mammals were carried out alongside routine rat control at five sites, around agricultural buildings (n = 2) and feed hoppers for game birds (n = 3).
4. Three non-target rodent species fed on rodenticide from bait boxes during routine rat control treatments. A large proportion (48·6%) of individuals in local populations ate the bait: woodmice *Apodemus sylvaticus* were most exposed, followed by bank voles *Clethrionomys glareolus* then field voles *Microtus agrestis*.
5. Local populations of non-target small mammals declined significantly following rodenticidal rat control but their relative proportions did not change significantly. Populations recovered partially after 3 months, depending on the time of the year relative to the breeding cycle.
6. *Synthesis and applications.* Our results clearly demonstrate that routine rat control reduced local populations of non-target small mammals. This may limit the food supply of some specialist predators. Most importantly, this demonstrates a significant route of exposure of predators and scavengers of small mammals to secondary poisoning. Rodenticides are applied on farms and game estates across the UK. Hence the results of this study are indicative of non-target rodenticide exposure nationally. Mitigation requires a shift from the current reliance on rodenticides to ecologically based rodent management, involving improvements in site management and the adoption of good farming practice.

Key-words: anticoagulant, coumatetralyl, farms, game feeders, predators, rat control, scavengers

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Introduction

Pesticides are integral to modern agriculture across the world but many pesticides have measurable, adverse effects on non-target wildlife. Regulators must balance acceptability of adverse effects against economic and health benefits to society as part of environmental risk assessment. Most rodenticides are anticoagulants and rely on a single mode of action, i.e. blocking the vitamin K cycle and preventing formation of blood-clotting factors. Anticoagulant rodenticides are categorized as either second-generation (1970–1980s) anticoagulants, for example difenacoum, bromadiolone, brodifacoum and flocoumafen, or their first-generation predecessors

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(1940–1960s), for example warfarin, pindone and coumatetralyl (Eason *et al*. 2002). Second-generation rodenticides are more potent than first-generation rodenticides, with greater affinity to binding sites in the liver and consequently greater accumulation and persistence (Parmar *et al*. 1987; Huckle, Hutson & Warburton 1988). Anticoagulants are toxic to all vertebrates.

Recent studies around the world have demonstrated extensive exposure of many non-target species to anticoagulants (Eason & Spurr 1995; Berny *et al*. 1997; Eason *et al*. 1999; Howald *et al*. 1999; Shore, Birks & Freestone 1999; Stone, Okoniewski & Stedelin 1999; Burn, Carter & Shore 2002). Use of rodenticides on farms in the UK. increased from 74% in 1992 to 89% in 2000 (Dawson, Bankes & Garthwaite 2003). Difenacoum is reported to be the most widely used rodenticide on arable farms (Thomas & Wild 1996) and game estates (McDonald & Harris 2000) in the UK. Exposure may be direct (primary), when non-target species eat bait, secondary, when predators eat contaminated prey, or even tertiary (Smith, Cox & Rampaud 1990). Second-generation rodenticides present the greatest secondary poisoning hazard to predators such as mustelids and raptors, with elimination half-lives > 100 days in the livers of rats (Parmar *et al*. 1987) and quail *Coturnix japonica* (Temminck & Schlegel) (Huckle *et al*. 1989).

The common rat *Rattus norvegicus* (Berkenhout), house mouse *Mus domesticus* (Schwartz & Schwartz) and grey squirrel *Sciurus carolinensis* (Gmelin) are the main targets of rodenticidal control in Britain, and their predators and scavengers are most at risk from secondary poisoning. Species that do not normally eat rats, however, are also affected. Surveys of rodenticide contamination in kestrel *Falco tinnunculus* (L.) (Shore *et al*. 2001), stoat *Mustela erminea* (L.) and weasel *Mustela nivalis* (L.) (McDonald *et al*. 1998) have all demonstrated significant rodenticide residues. Kestrels, stoats and weasels are specialist predators of non-target small mammals, a collective term used here to mean those species not targeted by rodenticidal control, including woodmouse *Apodemus sylvaticus* (L.), bank vole *Clethrionomys glareolus* (Schreber) and field vole *Microtus agrestis* (L.). This study aimed to determine whether small mammals could be an important route of exposure to rodenticide for predators and scavengers.

Small mammals are important in the diet of many predatory and scavenging species such as the weasel, kestrel, barn owl *Tyto alba* (Scopoli), long-eared owl *Asio otus* (L.), short-eared owl *Asio flammeus* (Pontoppidan) and tawny owl *Strix aluco* (L.). Townsend *et al*. (1984) reported secondary poisoning of weasels by warfarin, and mice dosed with the rodenticide coumatetralyl caused the death of 4/4 weasels over a period of 11–68 days (Anonymous 1981). Generalists, such as the fox *Vulpes vulpes* (L.), polecat *Mustela putorius* (L.), buzzard *Buteo buteo* (L.) and red kite *Milvus milvus* (L.), rely less on small mammals and alter their feeding habits depending on available prey. Non-target species may feed upon contaminated rodents around farms and other sites where rodent control is practised, for example feed hoppers used in rearing pheasant *Phasianus colchicus* (L.) on game estates.

Carcasses of 40 stoats and 10 weasels were collected from estate gamekeepers and analysed for six anticoagulant compounds in order to assess incidence of rodenticide exposure (McDonald *et al*. 1998). Residues were detected in 30% of weasels and 23% of stoats. A survey of 29 polecats revealed rodenticide residues in 31% (Shore *et al*. 1996). Birks (1998) highlighted heavy utilization of agricultural premises by polecats during winter, when rat populations are high and consequently bait application is likely to be at its highest. Analysis of polecat faeces confirmed rats as the principal prey item, although woodmice and voles were also taken.

The Centre for Ecology and Hydrology's (CEH; formerly the Institute for Terrestrial Ecology or ITE) predatory bird monitoring scheme and the Wildlife Incident Investigation Scheme (WIIS) revealed rodenticide exposure in kestrels, prompting analysis of kestrel livers for second-generation anticoagulants (Shore *et al*. 2001): 24/36 kestrels (67%) collected between 1997 and 2000 contained residues, indicating significant exposure through feeding. As a comparison, 187/717 barn owls (26%) analysed by CEH during 1983–96 contained detectable liver residues of second-generation rodenticides (Newton *et al*. 1999). Kestrels and barn owls rarely eat rats, suggesting that non-target small mammals may be the major route of exposure. Several studies have found small mammals to be attracted to rodenticide bait (Harradine 1976; Wood & Phillipson 1977; Cox 1991; Townsend, Entwhistle & Hart 1995).

The main aims of this study were to estimate proportions of small mammals exposed to rodenticide bait and to document population changes following exposure. Non-target exposure was studied alongside routine rat control programmes, to ensure that results were relevant to normal rat control on farms. Two scenarios were examined: around farm buildings, and around pheasant-feed hoppers on game estates. This study detailed the results of replicate trials where rat infestations were present on two farms and three pheasant-feeder sites on a large game estate.

The specific hypothesis tested was that non-target small mammals would eat bait and that small mammal populations at rat control sites would decline compared with populations at untreated reference sites.

Materials and methods

STUDY AREAS

Farm 1 was a mixed (arable and sheep) farm and game estate in Leicestershire, UK, and farm 2 an intensive pig farm in Northamptonshire in the east Midlands, UK. Farm 2 had a recent history of severe rat infestations, both within pig units and along field boundaries; rats were controlled using rodenticide in large-scale

operations, three to four times a year. Rat infestations were less severe on farm 1 and were controlled perhaps once or twice a year. Both areas were in a rural arable and pasture environment with variable topography interspersed with small copses, and adjacent to small villages. Pheasant-feeder sites were adjacent to arable fields on farm 1. Pheasant-feeder sites 1 and 2 were near streams bordered by thick established hedgerows interspersed with mature deciduous trees and scrub grassland. Pheasant-feeder site 3 was within a copse of mixed deciduous and coniferous trees. Each pheasant-feeder site contained several feeders. Grain spilled by pheasants was accessible to rats.

RAT BAITING

Sites were surveyed for rat activity in order to define infestation boundaries and determine best places for baiting. Bait points were set close to burrows and in areas of concentrated rat activity, revealed by rat runs and fresh droppings. Studies were conducted as follows (dimensions define areas over which rats were intensively active and small mammal populations were studied): farm 1 (190 × 100 m), February 2002; pheasant feeder 1 (10 × 150 m), March–April 2002; farm 2 (100 × 115 m), June 2002; pheasant feeder 2 (15 × 160 m), July–August 2002; pheasant feeder 3 (50 × 80 m), September–October 2002.

There were 15–30 bait points per site, with number and spacing dependent on extent and density of rat populations. Bait points were plastic bait trays inside a wooden box (40 × 15 × 15 cm) open at each end. Weighted rectangles of hardboard set against the ends of bait boxes at an angle prevented feeding by birds. The active ingredient in the rodenticide bait used in all trials was coumatetralyl (375 mg kg^{-1}; trade name Racumin; Bayer Environmental Science, Waltham Cross, Herts, UK), a first-generation, multiple-dose rodenticide with a half-life of 55 days in rat liver (Parmar *et al*. 1987) and relatively low toxicity to birds (Joermann 1998; Burn, Carter & Shore 2002). This was important because red kites (a protected species and the subject of a reintroduction programme in the UK) were present.

Bait points were pre-baited for 1 week to overcome neophobia (Barnett 1963) then baited with 100 g each. Bait points were checked daily; if all 100 g were consumed (complete take), the quantity of bait was doubled to 200 g. Where takes were partial, containers were topped up to 100 g or 200 g every 4 days to maintain a surplus. This surplus-baiting strategy is a standard approach (Buckle 1994) and is specified on rodenticide labels. Following the pre-bait week, rodenticide bait was applied for 10 days at each site, representing typical practice rather than best practice.

MAMMAL TRAPPING

Small mammals were live-trapped (Longworth traps; Penlon Ltd, Abingdon, UK), in order to monitor bait exposure and estimate small mammal populations. Traps were placed within and around the baited area independently of bait points. Fifty traps were placed in pairs in a grid system, located according to habitat (Gurnell & Flowerdew 1994) and marked with a numbered cane. Traps were filled with hay for bedding and warmth, and small handfuls of rolled oats for food. Fly castors were provided in case shrews (*Sorex* spp.) were accidentally captured. Traps were set at dusk and checked at dawn (times dependent on time of year) to cover active periods of all three study species (woodmice, bank voles and field voles). Animals were identified to species, sexed, weighed and marked by clipping guard hairs to reveal the undercoat of a different colour.

POPULATION ESTIMATION

Population estimates were required before (trap session 1) and after (session 2) rodenticidal treatment. The operational definition of a population in this study was 'number of animals that move and feed within the area enclosed by traps, or whose home range encompasses the location of the traps'. Following a trap pre-bait period of two to three nights, trapping was carried out for five nights and population size was estimated using mark–release–recapture (MRR) (Greenwood 1996). Identical trapping sessions were carried out 5 days after application of bait. The Cormack–Jolly–Seber (CJS) method of population size estimation was applied to MRR data as it is a fully stochastic model that allows for births, deaths, immigration and emigration (Greenwood 1996). Study areas were defined by extent of rat infestations and area of small mammal trapping; no site was isolated from surrounding small mammal habitat. Marking was batch-specific, with each mark on a different part of the body corresponding to a specific trap night. Populations could then be estimated with data on the number of animals with each possible capture history.

CJS estimates are imprecise when number of marked animals in each sample is below 10 (Greenwood 1996). Because rodenticide treatment sometimes left insufficient marked animals for a species-specific CJS estimate, estimates were made for the total small mammal community. Population estimation was repeated after 3 months in order to estimate population recovery at all sites, except at farm 2 where the farmer did not allow further access.

RODENTICIDE EXPOSURE

Small mammals invariably defecate within the trap tunnel before release. Rodenticide exposure was shown by the presence of a bait marker dye, pre-mixed with rodenticide bait, in faeces. Although the commercial bait was already dyed blue (as a deterrent to birds; Pank 1976), Chicago Sky Blue 6B dye (Sigma Aldrich Co. Ltd, Poole, Dorset, UK) was also added (700 mg kg^{-1}; Cox 1991) to ensure exposure was reliably identified.

The addition of dye to bait does not affect palatability to small mammals and rats (Cox 1991).

Daily inspections of rat bait boxes provided evidence of small mammal feeding: both grain remains and faeces differ between species (Cox 1991). Tracking tiles were also used to record footprints of animals entering bait boxes (Shepherd & Greaves 1984). Small mammal trapping continued through the first 5 days of rodenticide application, in order to record bait exposure. The mode of action of anticoagulant rodenticide is delayed, resulting in death 4–10 days after consumption of a lethal dose. Some exposed individuals would have died or been close to death after 5 days. During the second 5 days of rodenticide application, another, post-rodenticide, small mammal population estimation was carried out (trap session 2).

REFERENCE POPULATIONS WITHOUT RODENTICIDE

Small mammal population densities fluctuate widely and numbers are influenced by short-term climatic extremes. Reference populations were therefore monitored simultaneously in order to distinguish possible effects of rodenticide treatment from natural fluctuations. Reference sites were chosen to be as similar in habitat structure as possible to corresponding treatment sites.

Reference sites were 300–1000 m from treated sites. There was no history of rodenticide application in any reference site. A grain bait was provided at bait points in reference sites to allow for effects of supplementary feeding influencing population estimates through immigration from surrounding habitat. Fifty traps were placed at reference sites in the same manner as in treatment sites and trapping was performed in tandem.

STATISTICAL ANALYSIS

Statistical analyses used either a non-parametric sign test or a paired *t*-test. No transformation of data was necessary for the paired *t*-test.

Results

SMALL MAMMAL EXPOSURE

Woodmice and bank voles were trapped at all sites, field voles at 2/5 sites and house mice at farm 2 only. Small mammal visits to bait boxes were evident by the presence of footprints and tracks on tracking tiles. In every trial, both rat and small mammal footprints were found on individual plates, indicating commensal feeding at common sites, although they may have fed at different times. Small mammal faeces were found scattered both in bait boxes and within bait, showing that they fed while sitting in the bait as well as from the edge of the tray. These animals would be exposed both in feeding and through ingestion of bait powder and residue from grooming. Small mammal feeding was also evident from scrutiny of feeding remains.

The primary indicator of exposure was the presence of blue-coloured faeces in Longworth traps. Blue dye was generally obvious; when there was uncertainty, closer investigation (squashing or breaking open droppings) confirmed the presence of dye. Overall, an average of 48·6% (n = 938) of small mammals trapped had fed on rodenticide bait from bait boxes. Proportions ranged from 32% (n = 129) at pheasant feeder 1 to 67% (n = 363) at pheasant feeder 3 (Fig. 1). All four species that were trapped had been exposed, although apparently to different degrees. Woodmice were most attracted to bait, with an average of 57·4% (SD ± 14·5%) of animals that were trapped found to have eaten bait; exposure of bank voles was 30·6% (± 12·6%), exposure of field voles was 19·5% (± 2·1%) and of the house mice trapped at farm 2, 30% had dyed faeces (Fig. 2a–e).

CONSEQUENCES OF EXPOSURE

Following introduction of rodenticide bait (replacing pre-bait), small mammals began feeding immediately. A high proportion of traps contained dyed faeces the next day. The first signs of rodenticide poisoning in small mammals were observed 2–3 days later. Bleeding from orifices (nose, ears, anus and vagina) was noted in live woodmice and bank voles. Animals found dead in traps accompanied by dyed faeces were considered to be rodenticide victims.

Intoxicated animals showed changes in behaviour compared with unexposed, healthy animals (Cox 1991; Littin *et al*. 2002). Both woodmice and bank voles showed reduced escape responses, sometimes with unco-ordinated movement and a staggering gait.

POPULATION CHANGES

Small mammal populations declined at all treated sites following rat control treatments (Table 1). All but one of the corresponding reference populations increased over the same period. Only the reference population for farm 1 declined, probably because of poor weather conditions during February 2002, but this decline (44%) was less than at the corresponding rat treatment site (79%).

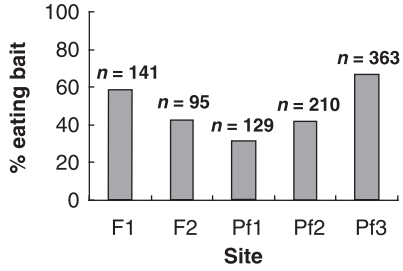

Fig. 1. The percentage of all small mammals trapped having eaten rodenticide bait, at each of five sites. F1, farm 1; F2, farm 2; Pf1, pheasant feeder 1; Pf2, pheasant feeder 2; Pf3, pheasant feeder 3.

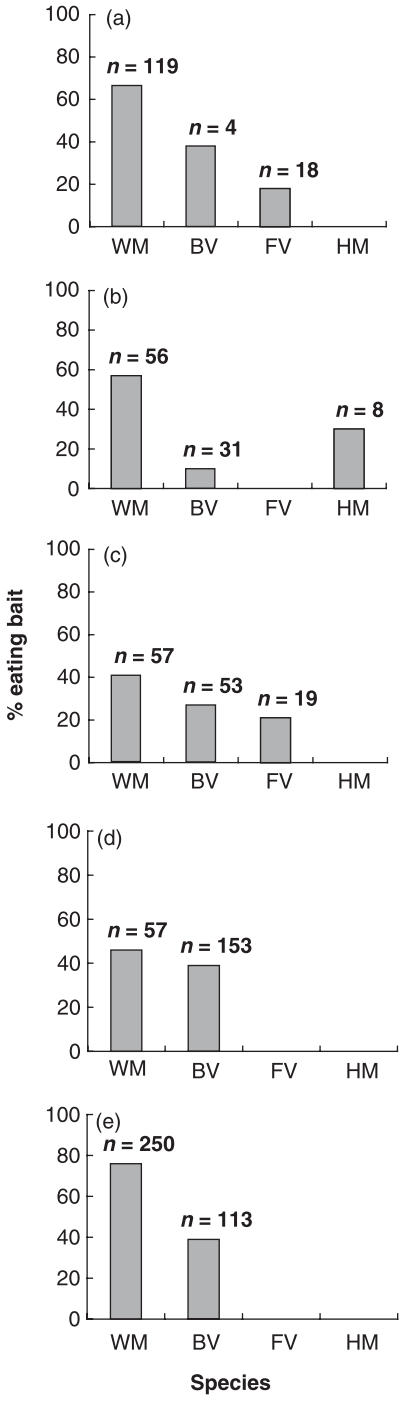

Fig. 2. The percentage of small mammal species trapped that were known to have eaten rodenticide bait in each study. (a) Farm 1; (b) farm 2; (c) pheasant feeder 1; (d) pheasant feeder 2; (e) pheasant feeder 3. WM, woodmouse; BV, bank vole; FV, field vole; HM, house mouse.

The specific hypothesis, that rat control reduced non-target small mammal populations, was tested by a one-tailed sign test. The one-tailed probability of a decline relative to reference populations at all five rat control sites occurring by chance was $P = 0{\cdot}5^5 = 0{\cdot}03125$, i.e. significant at the 5% level. Thus there was a significant decrease in small mammal populations, averaging about 60%, as a result of rodenticide poisoning.

POPULATION RECOVERY

Population estimates made 3 months after each trial indicated longer-term effects of rodenticide treatments (Table 1). At each pheasant-feeder site, small mammal populations had recovered or were at a level that may be expected for the time of year, suggesting density-dependent migration from adjacent populations. At farm 1 no small mammals were found after 3 months, possibly because woodmouse, bank vole and field vole populations are normally lowest in April–May.

Population recovery data are analysed in Table 2. The proportional rate of change is measured was N_3/N_0, the small mammal population size (CJS) after 3 months (N_3) compared with the initial population size (N_0) prior to rodenticide treatment. In all four cases, the rate of change was higher in the untreated reference populations than in the corresponding rodenticide treated populations, even when reference populations declined outside the breeding period. Effects of rat control were thus only partly offset by summer breeding, and outside the breeding period the two rat control sites (farm 1 and pheasant feeder 3) declined more than the untreated reference sites. A paired *t*-test showed that the effect of rat control on small mammal populations was statistically significant compared with reference sites ($P = 0{\cdot}047$).

Discussion

EXPOSURE OF SMALL MAMMALS TO RODENTICIDE

Differences in the proportions of each species feeding from bait boxes may reflect species' differences in foraging ecology and typical diets. The woodmouse is a generalist seed and insect eater, the bank vole a generalist herb, leaf and seed eater, and the field vole specializes in eating grass (Hansson 1985; Flowerdew 1993). It seems unlikely that behavioural interactions with rats at the bait boxes are responsible for differences in the proportions of each species that were exposed. Rats might defend a food source against small mammals but are normally most active nocturnally. Accordingly, the greatest degree of potential interaction would be with the nocturnal woodmouse (Montgomery & Gurnell 1985), yet the woodmouse had the highest level of exposure to rodenticide. Interaction at bait boxes may be largely avoided by non-overlapping periods of feeding during the night. Both vole species are active nocturnally and diurnally (Gipps 1985) and therefore were expected to have unhindered access to bait throughout the day. In monitoring poison hoppers used to control grey squirrels, Wood & Phillipson (1977) discovered a wide range of mammal and bird species attracted to and consuming warfarin bait. They estimated that 55–65% of bait was consumed by non-target animals, including woodmice, and most bait was removed at night, when squirrels do not feed.

Table 1. Small mammal population changes following rodenticide exposure at rodenticide-treated and reference sites using the Cormack–Jolly–Seber estimate. Small mammals included the following species: woodmice, bank voles, field voles and house mice. Not all species were present at every site

Site	Initial population	Population post-rodenticide exposure	Population change (%)	Population after 3 months
Farm 1	39	8	−79	0
Reference	41	23	−44	28
Farm 2	19	11	−42	No site access
Reference	25	25	0	No site access
Pheasant feeder 1	35	7	−80	34
Reference	17	25	+32	38
Pheasant feeder 2	28	25	−11	30
Reference	26	31	+16	45
Pheasant feeder 3	80	38	−53	25
Reference	73	96	+24	38
Total rodenticide-treated sites	201	89	−56	
Total reference sites	182	200	+9	

Table 2. Population recovery and proportional rate of change 3 months after rodenticide treatment. Data considered in relation to the time of year and small mammal breeding/non-breeding periods

Site	Time of year	Population after 3 months (N_3)	Initial population (N_0)	Proportional rate of change (N_3/N_0)
Farm 1	February–May	0	39	0
Reference		28	41	0·6829
Pheasant feeder 1	March–June	34	35	0·9714
Reference		38	17	2·2353
Pheasant feeder 2	July–October	30	28	1·0714
Reference		45	26	1·7308
Pheasant feeder 3	September–December	25	80	0·3125
Reference		38	73	0·5205
Breeding: rodenticide-treatment sites		64	63	1·0159
Breeding: reference sites		83	43	1·9302
Non-breeding: rodenticide-treatment sites		25	119	0·2101
Non-breeding: reference sites		66	114	0·5789
Total rodenticide-treatment sites		89	182	0·4890
Total reference sites		149	157	0·9490

There was clear evidence of caching in woodmice and bank voles through the presence of dyed faeces up to 3 days after removal of bait boxes from the site. Food stores and caches are common in burrows of woodmice and bank voles, particularly during autumn and winter (Montgomery & Gurnell 1985; Flowerdew 1993). Caching of bait would extend exposure time to an individual and might also lead to exposure of individuals that did not visit bait boxes, especially during winter when both species are known to share nests with conspecifics to conserve heat (Flowerdew 1993).

Shrews are insectivores and not expected to feed on grain-based bait, yet several were found dead with dyed faeces in traps. Residues of bait have been found in shrews in other studies (Colvin 1984; Townsend, Entwhistle & Hart 1995). Shrews may have taken grain bait opportunistically (Flowerdew 1993) or they may have consumed contaminated invertebrates. Insectivorous birds died from eating ants and cockroaches that had fed on brodifacoum baits (Godfrey 1985) and snails found near brodifacoum bait during rodent control were found to contain 0·91 mg kg^{-1} of brodifacoum (Howald *et al.* 1999). Residues of brodifacoum have been found in beetles feeding from bait stations (Eason & Spurr 1995). Indeed, both snails and beetles were found in bait trays in this study. Shrews are very susceptible to poisoning on low doses of warfarin and are reported to have a 28 times lower tolerance than woodmice (Churchfield 1990).

Assessing exposure using dye may underestimate the numbers of animals feeding on bait in some circumstances. At pheasant feeder 1, the incidence of dyed faeces seemed lower than suggested by the evidence of bait feeding and the reduction in estimated population size. A possible explanation for this may be the high rates of metabolism and digestion in small mammals. Small mammals that entered traps several hours after feeding on bait might already have excreted dyed faeces. Indeed, on investigation of faeces in traps, both dyed and light brown faeces could be found, resulting from bait feeding and feeding on the rolled oats provided in the trap, illustrating the high rate of digestion and elimination.

SMALL MAMMAL POPULATION CHANGES

The largest population declines were observed at farm 1 and pheasant feeder 1 during February and late March–early April, respectively. Woodmouse and bank vole populations are typically at their lowest at this time of year; the post-winter/pre-breeding season, and individuals that die are unlikely to be replaced. A large population decline was observed in the trial at farm 2, carried out in June, just after the breeding season, when numbers were expected to be increasing. The smallest population decline was observed at pheasant feeder 2, where trapping rates actually increased post-rodenticide exposure. This trial was carried out during late July–early August, when both woodmouse and bank vole populations are increasing following their respective breeding seasons. The trial at pheasant feeder 3 was carried out during late September–early October, when both woodmouse and bank vole populations are close to their peak. Population decline in the small mammal community here was mainly borne by the bank vole population. Trapping rates of woodmice remained high at pheasant feeder 3, despite a high proportion being exposed to bait. A possible explanation is that individuals that succumbed may have been replaced rapidly by dispersing woodmice from adjacent unaffected populations. Bank voles are not thought to disperse as rapidly as woodmice (Wolton & Flowerdew 1985).

It is clear that rat control treatments had a significant effect on local populations of the small mammal community. The magnitude of the effect, however, may have been influenced by time of year and the corresponding typical population densities. Thus population-level effects of rodenticide-induced mortalities in autumn may be tempered by high dispersal rates of juveniles following the breeding season (Table 2). In late winter, when small mammal numbers are at their lowest, rodenticide application may all but eliminate the remainder (Cox & Smith 1990; Cox 1991).

Immigration of new individuals following population losses as a result of rodenticide treatment will depend on both productivity of adjacent unaffected populations and quality of wildlife corridors. Certainly, all pheasant-feeder sites had a greater degree of habitat connectivity than farm 1, enabling populations to recover more quickly.

Relative proportions of each small mammal species did not change significantly following rodenticide treatment. The dominant species in each community prior to rodenticide application remained dominant post-treatment. Any changes in community structure between initial populations and recovered populations, 3 months after treatment, matched those in reference populations; such changes are clearly the effects of natural annual changes in specific species' abundances and not rodenticide-induced effects.

In view of the high proportions of small mammal populations exposed to bait and the effects on populations, duration of rodenticide treatment is likely to be critical for long-term population status and potential secondary exposure of predators and scavengers of small mammals. The objective of this study was to investigate small mammal rodenticide exposure during rat control treatments. Treatment periods lasted only 10 days, and population effects may have been less than would be expected in more extended rat control treatments, which often last 4–5 weeks, undoubtedly resulting in greater, longer term and probably wider negative effects on small mammal populations. Indeed, in studying non-target small mammal population effects of permanent warfarin-bait stations in Scottish shelterbelts, Harradine (1976) found that woodmouse and bank vole populations were reduced such that none of the breeding cohort remained to repopulate the site the following year. Analysis of bait markers in woodmice showed that individuals trapped as far as 80 m from a treatment area had fed from bait points (Townsend, Entwhistle & Hart 1995). Further, immigration rates of woodmice sometimes increase with provision of supplementary food (Flowerdew 1972). It appears likely that attraction to rodenticide bait, essentially a supplementary food source, could extend exposure beyond the treated site. Rodenticide-treated sites, especially where baiting is permanent, could act as local population sinks for small mammals, resulting in a continual supply of intoxicated prey and contaminated carcasses to predators and scavengers.

Populations can recover where rodenticide treatment is temporary. In studying resilience of small mammal populations, Sullivan (1986) found repopulation of sites, experimentally depopulated using poison, by five species of small mammal, showing that poison did not effectively suppress populations for long when baiting was carried out over limited areas. Table 2 demonstrates, however, that the proportional rate of recovery of rodenticide-treated sites was significantly less than in reference sites, i.e. the effects of rodenticide treatment persisted for at least 3 months.

EXPOSURE OF PREDATORS AND SCAVENGERS

Rodenticide-induced behavioural changes exhibited as symptoms of haemorrhage in rats, i.e. reduced escape response and staggering gait (Cox 1991), were also observed in contaminated woodmice and voles in this study. Internal haemorrhage greatly affects limb joints, which may account for decreased mobility (Wood & Phillipson 1977). Cox (1991) also suggests that rat foraging behaviour will shift from that which is perceived normal (i.e. thigmotaxis, maximal use of available cover) to a pre-lethal anticoagulant-toxicosis-induced behaviour (movement in the open and away from cover) that increases exposure and vulnerability to predation. Foraging behaviour of intoxicated small mammals will change similarly. Post-mortem examination of anticoagulated rats often reveals cerebral haemorrhages, which could account for aberrant behaviour (Cox 1991). The ability of predators to kill prey depends on ease of

capture and handling and therefore the issue of prey vulnerability becomes paramount (Cicero 1993). Certainly, a wide variety of observational and experimental evidence supports the generalization that prey moving slowly or abnormally are preferentially selected by predators (Popham 1943; Rudebeck 1950, 1951; Kenward 1978; Cicero 1993). For example, Hunt *et al.* (1992) found that the house sparrow *Passer domesticus* (L.) exposed to the contact avicide fenthion was selectively predated by the American kestrel *Falco sparverius* (L.). Results from this study show that a high proportion of the small mammal population living in areas where rat control is practised are exposed to rodenticide (as high as 67%). Development by a predator of a search image (Tinbergen 1960) for prey exhibiting rodenticide-intoxicated behaviours could increase the proportion of contaminated individuals in the diet and the likelihood of ingesting a harmful dose (Brakes 2003).

Generalist species, which do not normally contain a large proportion of small mammals in their diets, may also be at risk. The adaptive and opportunistic nature of generalist scavengers and predators may lead some individuals to capitalize on a large number of conspicuous and lethargic prey. Short-term rodenticide treatments resulting in a temporary glut of carcasses could lead to the almost certain death of species that exhibit caching behaviour, for example mustelids (King 1989) and foxes (Macdonald 1987).

Most species employ both predatory and scavenging modes of foraging. Thus, a species may be vulnerable to consumption of both rodent carcasses and living, but lethargic, rodents suffering sublethal anticoagulant toxicosis. The proportions of poisoned moribund animals that die out of sight or in the open are not known, yet this is an important element in quantifying potential availability of contaminated rodent carcasses to scavengers. It is generally thought that most poisoned rats retreat to their burrows or nests (Birks 1998; Newton *et al.* 1999) and this may apply to small mammals. As part of a rat control campaign on a seabird colony on Langara Island, British Columbia, Canada, a radio-tracking study of 19 rats found that 13/15 rats recovered had died underground in burrows (Howald *et al.* 1999). Routine carcass searches found only 35 individuals above ground, of the estimated pre-eradication rat population of 3000, representing 1·2% of the rat population (Howald *et al.* 1999). Other rat control studies report similar findings (Fenn, Tew & McDonald 1987; Taylor & Thomas 1993). Carcass searching is, however, notoriously unreliable (Stutzenbaker, Brown & Lobpries 1986). If poisoned moribund rodents retreat under cover, the secondary poisoning hazard to avian and other larger scavengers may be substantially reduced, although they may still be accessible to smaller mammalian scavengers that can access burrows and other rodent harbourage. This may increase the relative risk to weasels and stoats hunting in the burrows of small mammals. Rodenticide residues have been found to be more prevalent in female than male stoats (McDonald *et al.* 1998; Murphy *et al.* 1998), probably because female stoats eat more small mammals than male stoats (King 1989). Female weasels might also be exposed to poisoned moribund small mammals and their carcasses more frequently than males, because of their greater propensity for accessing small mammal tunnels and hunting underground (Erlinge 1975). Alternatively, differences in residue concentrations may reflect toxicokinetic differences between sexes, for example warfarin metabolism and rates of elimination are markedly different in male and female rats (Eason *et al.* 2002). Owls, hawks and larger predators occasionally predate stoats and weasels, leading to the possibility of tertiary poisoning.

EFFECTS OF REDUCED PREY ABUNDANCE

The distribution, density and reproduction of specialist predators are intimately linked to the population dynamics of their prey (Flowerdew 1993). Reductions in numbers of woodmice and voles have been shown to cause local declines and to affect reproductive success in weasels, stoats and tawny owls. In years of low numbers of small mammals, breeding failure is high and juvenile survival low in weasels and stoats (Tapper 1979; Erlinge 1981, 1983; King 1983). Studies of tawny owls and barn owls have shown that undernourished birds may not attain breeding fitness when prey availability is low. Even if reproduction is viable, clutch size, hatching success, hatchling survival and fledging success are all greatly affected by prey numbers (Southern 1970; Shawyer 1987). Field vole numbers affect population numbers and breeding of the kestrel, long-eared owl, short-eared owl and hen harrier *Circus cyaneus* (L.), where voles comprise a large proportion of the diet (Snow 1968; Galushin 1974; Glue 1977; Watson 1977; Village 1981; Flowerdew 1993). When field vole numbers are low, and where there are differences in hunting habitats, other small mammal species increase in importance or may even replace voles as the principal prey in the diet; even unpalatable shrews may become more important. All small mammal species studied here were exposed to differing degrees of rodenticide contamination, resulting in local population declines. It is possible that, in contrast with typical diets, easy capture of intoxicated animals could increase the proportion of a particular species in the diet. For example, predatory species for which the principal prey item is normally field voles might switch to woodmice if capture effort is lower because of rodenticidal intoxication.

SUBLETHAL EXPOSURE

In most cases of secondary poisoning, exposure is likely to be chronic with possible sublethal effects on behaviour and fitness. A reduction in mobility as a result of sublethal rodenticide toxicosis could increase likelihood of mortality from other causes. For example,

impairment of hazard awareness or speed of reaction might result in collision with traffic or power lines. Anticoagulants do not appear to have any obvious effects on laboratory animals at sublethal levels, although non-specific signs such as anorexia and depression have been observed shortly before clinical signs (Berny *et al*. 1997). Sublethal effects are very difficult to measure, especially in the wild. Sublethal doses of brodifacoum can cause abortions and reduced lambing rates in sheep (Godfrey 1985) and abortions in rats (WHO 1995). However, extensive brodifacoum poisoning operations on New Zealand islands produced no evidence of sublethal effects of low-level exposure in birds (Eason & Spurr 1995). In studying the incidence of rodenticide residues in stoats and weasels, McDonald *et al*. (1998) were unable to detect differences in body condition between contaminated and uncontaminated animals. Townsend *et al*. (1981) concluded that it was unlikely that tawny owls would obtain a lethal dose of warfarin from consumption of contaminated mice in treated woodlands, but expressed concern about sublethal effects of measured reductions in plasma prothrombin. A study of secondary poisoning of the golden eagle *Aquila chrysaetos* (L.) (Savarie *et al*. 1979) found that prothrombin-clotting time had significantly increased and, although clotting times eventually returned to normal (2 weeks later), eagles appeared weaker, with evidence of external bleeding. Such clotting disorders would be hazardous to any predator that was wounded or stressed.

EFFECTS ON PREDATOR AND SCAVENGER POPULATIONS

While there is no evidence that rodenticide-induced mortality in non-target predators and scavengers is causing populations to decline (Smith 1999), there is evidence of extensive exposure, with potential to cause additional mortality that may not be sustainable in populations already experiencing critical limitations. For example, kestrel numbers have declined by 29% in the UK over the period 1994–2000 (Noble, Raven & Baillie 2001), possibly linked with overall declines in farmland biodiversity in recent decades (Burn, Carter & Shore 2002). Many populations can withstand a certain amount of extra mortality (or reduced reproduction) without declining in the long term, because of density-dependent processes that enable remaining individuals to survive better or to reproduce more prolifically (Smith 1999), or through redistribution of individuals from more heavily populated areas (Newton 1998).

Small mammals have relatively short times to first breeding and high reproductive rates. They are therefore capable of recovering relatively rapidly from perturbations. It should be noted, however, that although rodenticide treatments typically continue for 3–5 weeks, rodenticide baits are sometimes available continuously, especially in areas with rodenticide resistance (Smith 1999). Predators and scavengers, in contrast, are larger, mature more slowly and have a lower reproductive rate. Predatory birds typically forage over large areas, encompassing several locations where rodenticide treatments may take place. History shows that recovery from perturbations may take many years [e.g. the sparrowhawk *Accipiter nisus* (L.); Newton 1988] even after the cause of population decline has been removed. Anticoagulant rodenticides have, of course, been in use for 50 years and second-generation rodenticides for more than 20 years. This has coincided with the decline of some farmland predators, although no direct link has been established. There are two reasons why we should be concerned now. One is the requirement of purchasers of farm produce for assurance schemes that include prophylactic rodent control. The other is the increasing importance of income from game rearing and shooting, which encourages feeding game birds in fields and controlling the concentrations of rats that inevitably follow. This study has shown that non-target small mammals provide a route of exposure to rodenticides that will increase in importance as the use of rodenticide away from farm buildings increases. It is clearly of concern to conservation biologists that predators and scavengers are exposed to rodenticide-contaminated animals through non-target as well as target species.

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