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EXPOSURE OF NON-TARGET VERTEBRATES TO SECOND-GENERATION RODENTICIDES IN BRITAIN, WITH PARTICULAR REFERENCE TO THE POLECAT *MUSTELA PUTORIUS*

Summary: In Britain, the use of "second-generation" rodenticides has become widespread on agricultural premises. The high toxicity and relatively long half-lives of these compounds has raised concerns over potential secondary exposure and poisoning of non-target predators. Over the last 15 years, exposure has been extensively documented in the barn owl *Tyto alba* but relatively little is known about mammalian terrestrial predators. This paper reviews recent studies and demonstrates that there is evidence of both secondary exposure and secondary poisoning in a variety of non-target, terrestrial mammals in Britain. It also presents new data on rodenticide levels in the polecat *Mustela putorius* which preys on farmyard rats in winter in Britain and is, therefore, considered to be highly vulnerable to exposure to rodenticides. The new data demonstrated that 26% of polecats in the sample contained difenacoum or bromadiolone and that exposure was geographically widespread and occurred in several years. The possible effects of secondary exposure on populations of polecats and other predators are discussed.

Keywords: Secondary poisoning; difenacoum; bromadiolone; mustelids; fox.

Introduction

Rodent infestations on agricultural premises are common in Britain (Meyer *et al*., 1995) and, as a result, use of rodenticides on farms is widespread. A 1992-1993 survey indicated that approximately three-quarters of farms growing grass, arable or fodder crops in Britain controlled rodents with poison (Olney and Garthwaite, 1994; Olney, Thomas and Garthwaite, 1994). Most rodenticides are applied in and around farm buildings to control common rats *(Rattus norvegicus* Berkenhout) and some are licensed only for use indoors. However, others are also used along hedgerows and in fields to protect crops and feed hoppers from various rodent species (McDonald *et al*., 1998).

Much of the control on agricultural premises now involves the so-called "second-generation" rodenticides, difenacoum, bromadiolone, brodifacoum and flocoumafen. They were introduced between 1975 and 1985 to replace warfarin, to which rats had become resistant (Cowan *et al*., 1995). The second-generation rodenticides are approximately 100-1000 times more toxic than warfarin and the other first generation compounds, a single meal of bait being sufficient to kill a rodent (Newton *et al*., in press). They also have long biological half-lives in tissues such as the liver, (Eason, Wright and Batcheler, 1996; Huckle *et al*., 1989; Parmar *et al*., 1987). Both attributes enhance their potential to cause secondary poisoning in predators. Rodents usually die several days after consuming bait containing second-generation compounds and so can be captured and eaten by a predator during this time. Some predators may also scavenge the carcasses of poisoned animals.

In Britain, there are a number of native predatory birds and mammals that feed on rodents in and around agricultural premises. Species about which there is particular concern with regard to secondary poisoning are the barn owl *(Tyto alba* Scopoli), and the polecat *(Mustela putorius* L.). Barn Owls have declined in numbers in recent decades (Shawyer, 1987) and the polecat is relatively rare because of persecution in the 19th Century (Langley and Yalden, 1977); until recently it has been restricted in its distribution to Wales (Blandford, 1987; Birks, 1993). Both species have partial or total legal protection in Britain. Other mammals that may feed on rats and other rodents in the vicinity of farms are the weasel *(Mustela nivalis* Erxleben), stoat *(M. erminea* L.), mink *(M. vison* Scheeber) and fox *(Vulpes vulpes* L.). Because of their foraging habits, all could be potentially exposed to rodenticides by eating contaminated prey. In this paper, we review the evidence that secondary exposure and poisoning

(used here as a term to indicate lethal poisoning) of rodent predators by rodenticides does occur in Britain. We also present new data on second-generation rodenticide residues in polecats, a species that, because of its ecology, may be especially vulnerable to secondary exposure and poisoning.

Evidence of secondary exposure and poisoning of wild birds and mammals by rodenticides in Britain

Secondary poisoning by rodenticides was considered to be a hazard for wild vertebrate predators in Britain as long ago as the 1960s. Walton (1970) suggested that poisoning of polecats by anticoagulants, largely warfarin at that time, was a common occurrence around farmyards. There was little field evidence to support this, possibly because polecats die out of sight and are rarely found (Birks, 1998). Subsequent experimental studies on weasels, however, did confirm that secondary poisoning by warfarin could occur in mustelids (Townsend *et al.,* 1984).

The issue of secondary exposure and poisoning by second-generation rodenticides was highlighted in Britain in the 1980s by a long-term study in which residues were detected in barn owl carcasses that had been collected from throughout the country as part of a national Predatory Bird Monitoring Scheme (Newton, Wyllie and Freestone, 1990). This scheme is still going on and, in a recent review of the data, it was calculated that the proportion of carcasses which contained detectable residues had increased from 5% in 1983-84 to 36% in 1995-96 and may be reaching steady-state (Newton *et a!.,* in press). Investigations in other countries in the 1980s also indicated the potential for secondary exposure and poisoning of raptors by second-generation rodenticides (Duckett, 1984; Hegdal and Blaskiewicz, 1984; Hegdal and Colvin, 1988; Mendenhall and Pank, 1980; Merson, Byers and Kaukeinen, 1984).

The demonstration of widespread exposure in barn owls prompted concern that free-living mammalian predators, in particular small mustelids, may be similarly exposed and at risk from secondary poisoning. There have been two recent British studies of the levels of rodenticides in mustelids. In the first, 29 polecat carcasses, mostly traffic casualties, were collected between 1992 and 1994 and 31 % contained detectable residues of second-generation rodenticides (Shore *et al.,* 1996). In the second survey, first and second generation rodenticides were detected in nine out of 40 stoats (23%) and three out of ten weasels (30%) which had been trapped or shot (McDonald *et al.,* 1998). Thus, both mustelid surveys and the barn owl study indicated that some 25-35% of the animals examined had been exposed to rodenticides.

Determining whether secondary poisoning occurs in free-living vertebrates is difficult because it requires the discovery of animals that died as a result of exposure. Using both post-mortem examination and quantification of liver residues, it was demonstrated that some barn owls collected for the Predatory Bird Monitoring Scheme were poisoned by second-generation rodenticides (Newton *et a!.,* in press). Secondary poisoning of free-living mustelids has been demonstrated in New Zealand (Alterio, 1996; Alterio, Brown and Moller, 1997; Brown, Alterio and Moller, 1998) but was a result of large-scale field applications of brodifacoum, a usage pattern not employed in Britain. As far as we are aware, the only documented evidence of secondary poisoning by second-generation rodenticides in mustelids in Britain is from a single polecat that died while being radio-tracked (Birks, 1998). It had a liver difenacoum residue of 1.4 $\mu g\ g^{-1}$. and was diagnosed as having died from difenacoum poisoning (Fletcher, Hunter and Barnett, 1994). The residue studies by Shore *et al.* (1996) and McDonald *et al.* (1998) had sampling methods that made unlikely and precluded respectively the collection of poisoned animals. Thus, they were not designed to determine if secondary poisoning occurred. They did show that some individuals survived exposure to rodenticides, at least until they were killed by other causes. However, five animals (two stoats, two polecats, one weasel) had bromadiolone concentrations of between 0.12 and 0.38 $\mu g\ g^{-1}$., similar to that (0.23 $\mu g\ g^{-1}$.) detected in an experimentally poisoned stoat (Grolleau, Lorgue and Nahas, 1989). The occurrence of such residues suggests that secondary poisoning of some mustelids in Britain is likely, although we have no means of knowing what percentage of exposures are fatal.

There is one further source of evidence that indicates secondary poisoning does occur in predatory mammals in Britain. The UK Ministry of Agriculture, Food and Fisheries runs a Wildlife Incident Investigation Scheme (WIIS), the aim of which is to investigate incidents of wildlife mortality and to determine whether pesticides are implicated. A post-mortem examination is carried out on animals submitted to the WIIS and, when appropriate, chemical analyses of specific body tissues are also conducted. Examination of the WIIS annual reports for the 1990s indicated that there have been incidents in which companion animals and a wide range of wild mammals have been poisoned by rodenticides (Table 1). Some of these incidents

Table 1: *List of species detected by the MAFF Wildlife Incident Investigation Scheme (WIIS) as being poisoned by rodenticides and statistical breakdown for fox incidents from 1990 to 1996. Data are taken from Fletcher et* al. *1991, 1992, Fletcher and Hunter 1993, Fletcher* et al. *1994, 1995, 1996, 1997.*

	Species poisoned by rodenticides detected by the WIIS scheme			
Year		Number of fox incidents	Incidents in which foxes were poisoned	poisoned by rodenticides
1990	fox, dog, cat	32	12	3
1991	fox, badger, rabbit, grey squirrel, dog, cat	43	8	2
1992	fox, badger, hedgehog, grey squirrel, dog, cat	43	9	9
1993	fox, polecat, grey squirrel, cat, dog, pig	53	11	5
1994	fox, badger, rabbit, dog, cat	57	10	3
1995	fox, grey squirrel, dog, cat	54	9	6
1996	fox, badger, rabbit, dog, cat, pig	41	14	6

involved misuse or abuse of pesticides and others may have been due to primary poisoning but some of the incidents in predators were thought to have been due to secondary poisoning.

Therefore, the WIIS provides evidence that secondary poisoning does occur in various predators. The almost complete absence of small mustelids from the data was probably because few individuals were submitted to the scheme and is not an indication that poisoning does not occur in these species. The most striking aspect of the species list in Table 1 is the consistency with which the fox appears (Table 1). A survey of animals found dead in France also indicated that foxes are poisoned by rodenticides (Berny *et al*., 1997). Of the fox incidents investigated each year by the WIIS, approximately 15-40% were pesticide-related and the number of pesticide poisonings that involved rodenticides varied from 25% in 1990 and 1991 to 100% in 1992 (Table 1). Overall, poisoning by rodenticides accounted for some 5-20% of all incidents investigated each year. These figures do not represent the proportion of the fox population that dies from rodenticide poisoning, because individuals suspected of dying from causes unrelated to pesticides are not submitted to the WIIS. However, they do suggest that secondary exposure to rodenticides is probably widespread in foxes and results in some secondary poisoning.

Evidence of secondary exposure of polecats to second-generation rodenticides

Polecats are strictly carnivorous (Blandford, 1987) and primary exposure to rodenticides, through direct take of bait, is improbable. Thus, exposure in this species is almost certain to be secondary and result mainly from consumption of poisoned rodents. Some exposure may also occur from eating lagomorphs, which can be exposed to rodenticides (see Table 1), and possibly through scavenging the carcasses of poisoned animals. Of all British mammals, polecats are arguably the most likely to be secondarily exposed to rodenticides because they actively hunt farmyard rodents in winter. Birks (1998) carried out a radio-tracking study on eleven polecats and found that ten of the animals visited agricultural premises during the period from September to March, the time when rats are most abundant on farms. Analysis of scats indicated that common rats made up 65% of the polecat diet at that time. It is because of their reliance on farmyard rats as winter food and because of their relative rarity in Britain that we have concentrated upon polecats in our studies of secondary exposure to rodenticides.

In the survey of polecat carcasses that we conducted previously (Shore *et al*., 1996), the livers of 24 animals were analysed for second-generation rodenticides. A second study, in which the livers of another 26 adult animals (19 males, 7 females) were analysed for the same compounds, has now been carried out. As in the first study, almost all of the animals in the second study were killed on the roads; the cause of death of one animal was unknown. The methods of carcass collection and chemical analysis of the liver were the same in the two surveys and are fully described by Shore *et at*. (1996). Residues were corrected for column recoveries (65%) in the second survey so as to make them compatible with data from the first survey. Limits of detection in the second survey were the same as those in the first (see Shore *et al*., 1996) except for a slight improvement for bromadiolone (0.013 μg in the whole sample). For the purposes of the present paper, data from the two surveys were combined to assess overall temporal and geographical trends in residue distribution.

The results of the surveys (Table 2) were that second-generation rodenticides were detected in 13 of the 50 livers analysed (26%). Difenacoum and bromadiolone were the predominant compounds, occurring in 16% and 14% of animals respectively, whereas brodifacoum was only found in one polecat and flocoumafen was not detected at all. More than one compound was present in two animals, suggesting that multiple exposure does occur. Males were more numerous in the sample but both sexes appeared equally likely to be exposed to rodenticides; 9 of the 34 males (26%) and 4 of the 16 females (25%) contained residues (Table 2). Examination of the geographical distribution of residues revealed that contaminated polecats came from various counties (Fig. 1), indicating that exposure was not a localised occurrence. A breakdown of the data by month (different years combined) showed that, although the sample contained polecats which had died in every month except December, residues were largely only found in animals killed between November and April (Fig. 2). When the year was divided on a calendar basis into the three 4-month periods, the ratio of contaminated to uncontaminated animals in January-April was 10: 12 compared with 1:8 in May-August and 2: 17 in September-December; these ratios were significantly different from each other ($X_{(2)}^2 = 7.73$, *P<0.05)*. When data were analysed on an annual basis. it was evident that exposure occurred in several years but not in all (Fig. 3). Most notably, residues were not found in animals killed in 1995. This may simply have been a result of random variation. It did not appear to be due to any bias in the time when carcasses were coIlected as the proportion of polecats from the winter and spring months, when residues were usually detected, was not particularly low in 1995 (Fig. 3).

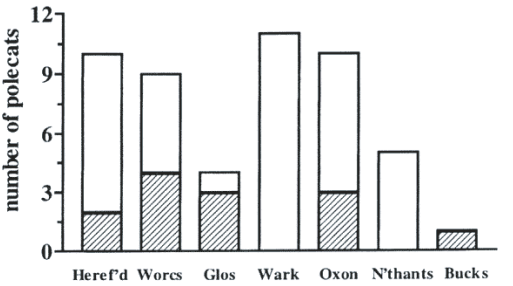

Figure 1: *The numbers of polecats with (shaded bars) and without (open bars) detectable residues of second-generation rodenticides collected in Herefordshire. Worce.ftershire, Gloucestershire, Warwickshire, Oxfordshire, NorthamplOnshire and Buckinghamshire.*

The findings from the analysis of 50 animals are consistent with and, because they are based on a larger sample, add confidence to the conclusions drawn from the first survey (Shore *et al.,* 1996). It is evident that exposure of polecats to second-generation rodenticides is geographically widespread and occurs repeatedly. The rodenticides detected in the liver reflect usage patterns, difenacoum and bromadiolone being the second-generation rodenticides most widely applied on British farms (Olney and Garthwaite, 1994; Olney *et al.,* 1994).

Table 2: *Rodenticide residues in polecats. ND = not detected. Residues were not detected in the livers of the other 37 animals analysed. Flocoumafen was not detected in any of the livers. Data from first survey are from Shore* et al. *(1996).*

Animal code	Date carcass located	Location (county)	Sex	Rodenticide concentration (µg g^{-1})		
				Difenacoum	Bromadiolone	Brodifacoum
1st survey						
P33/94	09/4/94	Herefordshire	M	0.005	0.217	0.008
P02/94	06/2/94	Worcestershire	M	0.073	0.116	ND
P42/94	28/4/94	Herefordshire	F	0.193	ND	ND
P43/94	15/4/94	Worcestershire	M	0.321	ND	ND
P13/93	25/3/94	Gloucestershire	F	0.016	ND	ND
P28/94	02/4/94	Gloucestershire	M	ND	0.039	ND
P23/93	1213/93	Gloucestershire	M	0.100	ND	ND
2nd survey						
P170/94	15/11/94	Worcestershire	F	0.125	ND	ND
P172/94	21/11/94	Oxfordshire	M	ND	0.126	ND
P18/96	15/06/96	Buckinghamshire	F	0.016	ND	ND
P14/97	03/03/97	Oxfordshire	M	ND	0.024	ND
P25/97	01/04/96	Worcestershire	M	ND	0.016	ND
P27/97	09/02/97	Oxfordshire	M	ND	0.018	ND

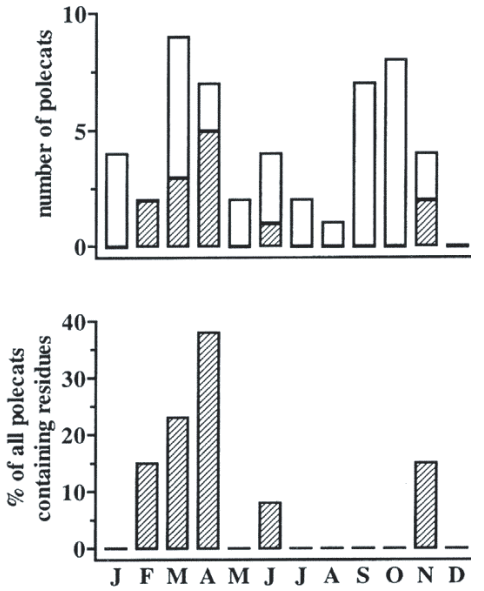

Figure 2: *Upper graph: the numbers of polecats with (shaded bars) and without (open bars) detectable residues of second-generation rodenticides collected in each month (data for different years combined). Lower graph: the % of polecats collected in each month that contained detectable residues.*

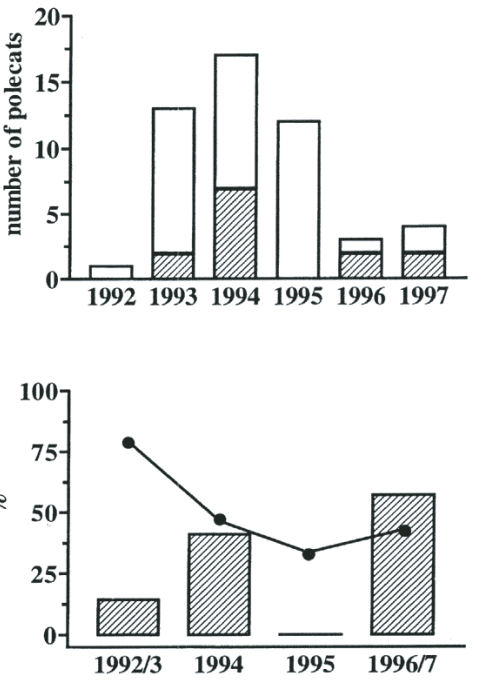

Figure 3: *Upper graph: the numbers of polecats with (shaded bars) and without (open bars) detectable residues of second-generation rodenticides collected in each year. Lower graph: Shaded bars are the % of polecats that contained detectable residues in each year. Solid line indicates the % of polecats in each year that were killed between November and April, the six month period in*

The prevalence of rodenticides in animals killed in winter and early spring most probably reflects the fact that polecats are exposed to rodenticides when they hunt on farms in early winter and are then killed on roads in subsequent months when they disperse into the wider landscape. Elimination of rodenticides from the liver is biphasic in mammals with a rapid initial phase lasting two to eight days after exposure and a slower terminal phase in which the elimination half-life exceeds 100 days (World Health Organisation. 1995). Thus, it is likely that residues would be detected in polecats several months after exposure. It is uncertain whether the general lack of detectable rodenticide in animals killed between May and October indicates that residues accumulated in the previous winter are largely metabolised and eliminated by this time, some five-ten months after the main exposure period. The duration over which rodenticides remain detectable in polecats will vary with time and extent of exposure, amongst other factors. Other causes, such as sampling different sub-sets of animals in different seasons, cannot be ruled out.

Overall, it is surprising that the proportion of individuals contaminated with rodenticides appears to be so similar in polecats, stoats, weasels and barn owls, given that the polecat has a closer association with farms and their rats than at least the other mustelids (Corbet and Harris, 1991). It is possible that direct comparison of the stoat and weasel data with those for polecats is biased by differences in methodology. Stoats and weasels were analysed for six (first and second generation) rodenticides and came largely from eastern England (McDonald *et al.,* 1998), where rodenticide use is relatively high, whereas polecats were analysed only for four second-generation rodenticides and were predominantly from western counties, where rodenticides are used less intensely. Had stoats and weasels been taken from the same areas as polecats

and analysed only for the same compounds, the proportion of animals with detectable residues may have been lower. However, this does not appear to be the case with barn owls. Direct comparison of, albeit limited, data for owls and polecats collected in the same years and region and chemically analysed by identical methods indicated that the percentage of contaminated animals did not differ significantly between the two species (Newton *et al*., in press). Thus, there is no evidence to suggest that polecats and barn owls differ in their levels of exposure to second-generation rodenticides.

It is arguable that surveys of rodenticide residues in road-killed and trapped animals may under-estimate the true level of exposure. This is because animals that have ingested a lethal dose of rodenticide are under-represented. This will also be true for surveys of carcasses found by the public if animals die in places where they are not easily found. Under-estimation of exposure may be especially pronounced in polecat samples collected throughout the year because of the seasonality of exposure. In the study by Birks (1998), five of the eleven (45%) radio-tracked animals made "heavy use" of farmyards in which rodenticides were applied and were considered vulnerable to secondary exposure. If that small sample was representative of the whole population, the true level of exposure would appear to be almost twice that detected in the present polecat carcass survey. Interestingly, if polecats killed between May and October are excluded from the carcass survey data, because it is assumed that residues accumulated in the previous winter have been eliminated, the percentage of contaminated individuals in the sample increases to 46%, the same as indicated by the radio-tracking study. Clearly, the exposure to, and subsequent pharmacokinetics of, second-generation rodenticides in polecats need to be quantified so that survey data can be interpreted with greater accuracy.

Effects of rodenticides on wildlife populations in Britain

There are few data with which to assess the effects of rodenticides on populations. Newton *et al*. (in press) found that less than 2% of individuals examined in the barn owl survey had died from poisoning by second-generation rodenticides and concluded that there was currently no evidence that rodenticides seriously affected population levels. For mustelids, there is no evidence from the WIIS of any widespread mortality of stoats, weasels or polecats although poisoned animals may not be found. It is not known whether the 9% level of secondary poisoning amongst the 11 polecats studied by Birks (1998) is at all representative of rodenticide poisonings in the whole polecat population. However, polecats are expanding their range in Britain (Birks, 1993), indicating that, whatever mortality may be caused by anticoagulants, it is not sufficient to prevent population expansion at present. Whether rodenticides will affect the ability of polecats to recolonise eastern areas of Britain, where rodenticide use is heavier, remains to be seen.

Conclusions

The data reviewed and presented in this paper indicate that at least 25-35% of individuals in populations of small mammal predators are secondarily exposed to rodenticides in Britain. It is possible that this is an under-estimate. Exposure of barn owls and polecats has been shown to be geographically widespread, occurs in many years and matches rodenticide usage patterns.

Secondary poisoning, rather than just exposure, has also been recorded in various small mammal predators in Britain. However, there is little understanding of the frequency with which this occurs in mammals or its importance compared with other causes of mortality. Studies are needed to assess the magnitude and frequency of rodenticide intake by small mammal predators and to determine the toxic effects of this exposure. It is only with such data that the importance of secondary poisoning on populations can be estimated. Any such estimates should be validated by field studies. Furthermore, given the widespread occurrence of low-level exposure and the lack of knowledge about what effects this may have, physiological and behaviourial studies on the effects of sub-lethal exposure in predators are also much needed.

References

Alterio, N. 1996. Secondary poisoning of stoats *(Mustela erminea),* feral ferrets *(Mustela furo),* and feral house cats *(Felis catus)* by the anticoagulant poison, brodifacoum. *New Zealand Journal of Zoology* 23: 331-338.

Alterio, N.; Brown, K.; Moller, H. 1997. Secondary poisoning of mustelids in a New Zealand *Nothofagus* forest. *Journal of Zoology (London)* 243: 863-869.

Berny, PJ.; Buronfosse, T.; Buronfosse, F.; Lamarque, F.; Lorgue, G. 1997. Field evidence of secondary poisoning of foxes *(Vulpes vulpes)* and buzzards *(Buteo buteo)* by bromadiolone, a 4-year survey. *Chemosphere* 35: 1817-1829.

Birks, J.D.S. 1993. The return of the polecat. *British Wildlife* 5: 16-25.

Birks, J.D.S. 1998. Secondary rodenticide poisoning risk arising from winter farmyard use by the European Polecat *Mustela putorius*. *Biological Conservation* 85: 233-240.

Blandford, P.RS. 1987. Biology of the Polecat *Mustela putorius:* a literature review. *Mammal Review* 17: 155-198.

Brown, K.P.; Alterio, N.; Moller, H. 1998. Secondary poisoning of stoats *(Mustela erminea)* at low mouse *(Mus musculus)* abundance in a New Zealand *Nothofagus* forest. *Wildlife Research* 25: 419-426.

Corbet, G.B.; Harris, S. 1991. *The Handbook of British Mammals* Third edition. Blackwell Scientific Publications, Oxford.

Cowan, D.P.; Dunsford, G.; Gill, E.; Jones, A.; Kerins, G.; MacNicoll, A.; Quy, R 1995. The impact of resistance on the use of 2nd generation anticoagulants against rats on farms in southern England. *Pesticide Science* 43: 83-93.

Duckett, J.E. 1984. Barn Owls *(Tyto alba)* and the second generation rat-baits utilised in oil palm plantations in Peninsular Malaysia. *Planter, Kuala Lumpur 60:* 3-11.

Eason, C.T.; Wright, G.R.; Batcheler, D. 1996. Anticoagulant effects and the persistence of brodifacoum in possums *(Trichosurus vulpecula)*. *New Zealand Journal of Agricultural Research* 39: 397-400.

Fletcher, M.R; Hunter, K. 1993. *Pesticide Poisoning of Animals 1992: Investigations of Suspected Incidents in Great Britain*. MAFF publications, London, 43 pp.

Fletcher, M.R.; Hunter, K.; Barnett, E.A 1994. *Pesticide Poisoning of Animals 1993: Investigations of Suspected Incidents in the United Kingdom*. MAFF publications, London, 52 pp.

Fletcher, M.R.; Hunter, K.; Barnett, E.A 1995. *Pesticide Poisoning of Animals 1994: Investigations of Suspected Incidents in the United Kingdom*. MAFF publications, London, 52 pp.

Fletcher, M.R; Hunter, K.; Barnett, E.A.; Sharp, E.A. 1996. *Pesticide Poisoning of Animals 1995: Investigations of Suspected Incidents in the United Kingdom*. MAFF publications, London, 48 pp.

Fletcher, M.R.; Hunter, K.; Barnett, E.A.; Sharp, E.A. 1997. *Pesticide Poisoning of Animals* 1996: *Investigations of Suspected Incidents in the United Kingdom*. MAFF publications, London, 50 pp.

Fletcher, M.R.; Hunter, K.; Quick, M.P.; Grave, R.C. 1992. *Pesticide Poisoning of Animals 1991: Investigations of Suspected Incidents in Great Britain*. MAFF publications, London, 39 pp.

Fletcher, M.R; Hunter, K.; Quick, M.P.; Thompson, H.M.; Greig-Smith, P.W. 1991. *Pesticide Poisoning of Animals 1990: Investigations of Suspected Incidents in Great Britain*. MAFF publications, London, 30 pp.

Grolleau,G.; Lorgue, G.; Nahas, K. 1989. Toxicité secondaire, en laboratoire, d'un rodenticide anticoagulant (bromadiolone) por des prédateurs de rongeurs champêtres: buse variable *(Buteo buteo)* et hermine *(Mustela erminea)*. *EPPO Bulletin 19:* 633-648.

Hegdal, P.L.; Blaskiewicz, R.W. 1984. Evaluation of the potential hazard to Barn Owls of talon (brodifacoum bait) used to control rats and house mice. *Environmental Toxicology and Chemistry* 3: 167-179.

Hegdal, P.L.; Colvin, B.A. 1988. Potential hazard to eastern Screech-owls and other raptors of brodifacoum bait used for vole control in orchards. *Environmental Toxicology and Chemistry* 7: 245-260.

Huckle, K.R; Hutson, D.H.; Logan, C.1.; Morrison, B.1.; Warburton, P.A. 1989. The fate of the rodenticide flocumafen in the rat: retention and elimination of a single oral dose. *Pesticide Science* 25: 297-312.

Langley, P.J.W.; Yalden, D.W. 1977. The decline of the rarer carnivores in Great Britain during the nineteenth century. *Mammal Review* 7: 95-116.

McDonald, RA.; Harris, *S.;* Turnbull, G.; Brown, P.; Fletcher, M. 1998. Anticoagulant rodenticides in stoats *(Mustela erminea* L.) and weasels *(M. nivalis* L.) in England. *Environmental Pollution 103:* 17-23.

Mendenhall, Y.M.; Pank, L.F. 1980. Secondary poisoning of owls by anticoagulant rodenticides. *Wildlife Society Bulletin* 8: 311-315.

Merson, M.H.; Byers, R.E.; Kaukeinen, D.E. 1984. Residues of the rodenticide brodifacoum in voles and raptors after orchard treatment. *Journal of Wildlife Management* 48: 212-216.

Meyer, AN.; Shankster, A.; Langton, S.D.; Jukes, G. 1995. National commensal rodent survey 1993. *Environmental Health* June 1995: 127-133.

Newton, I.; Wyllie, I.; Freestone, P. 1990. Rodenticides in British barn owls. *Environmental Pollution* 68: 10 1-117.

Newton, I.; Shore, R.F.; Wyllie, I.; Birks, J.D.S.; Dale, L. in press. Empirical evidence of side-effects of rodenticides on some predatory birds and mammals. *Proceedings of the First European Conference on Vertebrate Pest Management*.

Olney, N.J.; Garthwaite, D.G. 1994. *Pesticide Usage Survey Report* 113: *Rodenticide Usage in Great Britain on Farms Growing Arable Crops* 1992. MAFF Publications, London, 20 pp.

Olney, N.J.; Thomas, M.R.; Garthwaite, D.G. 1994. *Pesticide Usage Survey Report 122: Rodenticide Usage on Farms in Great Britain Growing Grassland & Fodder Crops 1993*. MAFF Publications, London, 21 pp.

Pannar, G.; Bratt, H.; Moore, R.; Batten, P.L. 1987. Evidence for a common binding site *in vivo* for the retention of anticoagulants in rat liver. *Human Toxicology* 6: 431.

Shawyer, C.R. 1987. *The Barn Owl in the British Isles. Its past, present and future*. The Hawk Trust, London, 113 pp.

Shore, R.F.; Birks, J.D.S.; Freestone, P.; Kitchener, A. 1996. Second generation rodenticides and polecats *(Mustela putorius)* in Britain. *Environmental Pollution* 91: 279-282.

Townsend, M.G.; Bunyan, P.J.; Odam, E.M.; Stanley, P.I.; Wardall, H.P. 1984. Assessment of secondary poisoning hazard of warfarin to least weasels. *Journal of Wildlife Management* 48: 628-632.

Walton, K.C. 1970. The polecat in Wales. *In:* Lacey, W.S. (Editor), *Welsh Wildlife in Trust*. North Wales Naturalist Trust, Bangor, pp. 98-108.

World Health Organisation 1995. *Environmental Health Criteria* 175: *Anticoagulant Rodenticides*. WHO, Geneva, 121 pp.