The Toxicity and Sub-lethal Effects of Brodifacoum in Birds and Bats

A Literature Review

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1 Introduction

The Weeds and Pests Division, Manaaki Whenua–Landcare Research, Christchurch, reviewed the scientific literature on the toxicity and sub-lethal effects in birds and bats of brodifacoum used in cereal-based baits for rodent control, and identified non-target species likely to be put at risk. This review, prepared for the Department of Conservation (DoC) in October 1992 - October 1993, is intended as a risk assessment to accompany on-going field and laboratory research on the impact of brodifacoum rat baits on non-target species and its persistence in the environment.

2 Background

Rodents, especially ship rats (*Rattus rattus*), have played a significant role in the reduction of many species of New Zealand birds, reptiles, amphibians, and invertebrates.

During the 1970s and early 1980s eradication of rodent populations from New Zealand offshore islands was seen as only a remote possibility. Since then new potent second-generation anticoagulant rodenticides have been developed, and the success of more recent eradication programmes has been attributed to their use (Taylor & Thomas 1989, 1993). Ground-baiting with brodifacoum in the form of Talon 50WB in covered bait stations has been considered to be relatively safe. However, the aerial sowing of Talon 20P has been associated with the deaths of a range of bird species, including saddlebacks and moreporks (Towns & McFadden 1991, unpubl. DoC report) and this has heightened concerns about the non-target impact of brodifacoum.

In 1991 a review of the advantages and disadvantages of existing rodenticides and rat baits for DoC recommended environmental impact studies (including monitoring of non-target populations and analysis for brodifacoum residues in non-target species) if brodifacoum baits were to be aerially sown in rodent-eradication programmes (Eason 1991, unpubl. FRI contract report).

Landcare Research has been contracted to undertake field trials to evaluate the safety of brodifacoum used in baits in bait stations and in aerially-sown cereal baits for rodent eradication. This review is intended to identify any non-target species at risk and assist in the design of these field trials.

3 Objectives

- To review the worldwide literature on the toxicity and sub-lethal effects of brodifacoum in birds and bats.
- To identify birds and bats that might be put at risk by the use of brodifacoum in cereal-based baits for rat control in New Zealand.

4 Methods

The international scientific literature on the toxicity of brodifacoum and related toxins was comprehensively reviewed. Relevant material relating to the non-target effects of brodifacoum and other similar anticoagulant toxins was collated and is summarised in this report. Researchers in DoC associated with aerial baiting using brodifacoum were contacted and interviewed, and their comments are included in this report as personal communications.

ICI, the manufacturers of brodifacoum baits, were contacted and this report includes results from a number of unpublished ICI field trials conducted to monitor the non-target effects of brodifacoum.

The distribution and feeding habits of indigenous non-target birds and bats that might be subjected to primary or secondary poisoning after use of brodifacoum in cereal-based baits for rat control were assessed by considering their feeding habits and the likelihood of their eating toxic baits.

5 Results

5.1 THE TOXICOLOGY AND PERSISTENCE OF BRODIFACOUM

Over 10 anticoagulant toxins are marketed worldwide, and these are conventionally divided into first-generation compounds such as pindone and warfarin, which were developed in the 1940s and 1950s, and second-generation compounds such as brodifacoum, which was developed in the mid 1970s (Hadler & Shadbolt 1975). Brodifacoum, like the other anticoagulant toxins, acts by interfering with the normal synthesis of vitamin K-dependent clotting factors in the liver of vertebrates. The potential insecticidal properties of these toxins are unknown. However, it has been suggested that anticoagulants are unlikely to affect invertebrates, which have different blood clotting systems from vertebrates (Shirer 1992).

The greater potency of the second-generation anticoagulant toxins is likely to be related to their accumulation and persistence in the liver after absorption (Huckle *et al.* 1988). Anticoagulants share a common binding site in the liver, but the second-generation anticoagulants have a greater binding affinity than the first-generation compounds (Parmar *et al.* 1987). Sub-lethal doses of brodifacoum can persist in the liver of sheep for over 16 weeks (Laas *et al.* 1985), and sub-lethal doses of flocoumafen, a structurally related second-generation anticoagulant, accumulate and persist in rats for 14 weeks (Huckle *et al.* 1988).

The persistence and potency of the second-generation anticoagulants means the risk of primary and secondary poisoning by these toxins is greater than that associated with the earlier anticoagulants.

5.2 ACUTE TOXICITY AND PRIMARY POISONING

Second-generation compounds such as brodifacoum differ from the earlier anticoagulants in that only a single feeding is needed to induce death. The toxicity of brodifacoum varies in both mammals (Table 1, included for comparative purposes) and birds (Table 2).

TABLE 1 THE ACUTE ORAL TOXICITY OF BRODIFACOUM IN DIFFERENT MAMMAL SPECIES (FROM GODFREY 1985)

MAMMAL SPECIES	LD_{50} (MG/KG)
pig	0.1
possum	0.17
rabbit	0.2
rat	0.27
mouse	0.4
dog	3.5
sheep	10
cat	25

TABLE 2 THE ACUTE ORAL TOXICITY OF BRODIFACOUM IN DIFFERENT BIRD SPECIES[1] (FROM GODFREY 1985)

BIRD SPECIES	LD_{50} (MG/KG)
southern black-backed gull	<0.75*
Canada goose	<0.75*
pukeko	0.95
blackbird	>3**
hedge sparrow	>3**
California quail	3.3
mallard duck	4.6
black-billed gull	<5.0*
house sparrow	>6**
silvereye	>6**
Australasian harrier	10.0
ring-necked pheasant	10.0
paradise shelduck	>20**

1 *Names follow Turbott (1990)*

* *Lowest dose tested*

** *Highest dose tested*

Small birds such as silvereyes, sparrows, blackbirds, and quail are considered more resistant to brodifacoum than some larger birds such as gulls, geese, and pukeko (Godfrey 1985). However, some large birds including Australasian harriers, pheasants, and paradise shelducks are also relatively resistant. Despite these distinctions, a wide range of small and large birds have been found dead from primary poisoning after field use of brodifacoum; e.g., saddlebacks, blackbirds, chaffinches, a house sparrow, and a hedge sparrow were found dead after Talon® 20P was aerially sown to eradicate kiore and rabbits on Stanley Island (Towns & McFadden 1991, unpubl. DoC report), and silvereyes, blackbirds, and song thrushes were found dead after Talon® 20P was used to control dama wallabies near Rotorua (D. Moore pers. comm.). A paradise shelduck, magpie, and chaffinch found dead after ground-laying of cereal-based Talon® bait for rabbit control contained residues of brodifacoum (Rammell *et al.* 1984; Williams *et al.* 1986). Robins were seen eating crumbs of Talon® 50WB dropped by Norway rats on Breaksea Island and two were found dead, although there was no significant change in population density (Taylor & Thomas 1993). The entire weka population on Tawhitinui Island was

exterminated mainly by direct consumption of Talon® 50WB intended for ship rat control (Taylor 1984).

These findings suggest that the reported differences in sensitivity (from published LD_{50} values) may be either inaccurate or irrelevant predictors of susceptability to brodifacoum. Species such as blackbirds, silvereyes, and paradise shelducks are reported to be moderately resistant, and yet these species have been killed by brodifacoum.

Sub-lethal doses of brodifacoum caused abortions and reduced lambing rates in sheep (Godfrey 1985), and concerns have been expressed about the adverse effects of small doses of anticoagulants on tawny owls (Townsend *et al.* 1980). However, there are no publications that elucidate any potential long-term effects of low-level brodifacoum exposure in birds.

There are no published LD_{50} data on the direct acute toxicity of brodifacoum orally administered to bats. However, in Latin America where paralytic bovine rabies is transmitted by the common vampire bat (*Desmodus rotundus*), cattle are given sub-lethal intramuscular doses of diphacinone, a first-generation anticoagulant related to brodifacoum. These cattle effectively act as live baits, and bats that suck blood from treated cattle die (Thompson *et al.* 1972; Mitchell 1986; Said Fernandez & Flores-Crespo 1991). It is not possible to extrapolate the toxicity of diphacinone in the vampire bats to the susceptibility of New Zealand's short and long-tailed bats (*Mystacina tuberculata* and *Chalinolobus tuberculatus*, respectively) to brodifacoum. However, it appears that bats may be susceptible to anticoagulants.

5.3 SECONDARY POISONING

It has been suggested that invertebrates are unlikely to be directly killed by brodifacoum (Shirer 1992). However, invertebrates have been seen eating baits containing brodifacoum, and residues of brodifacoum have been found in beetles collected from bait stations containing Talon® 50WB intended for rats on Stewart Island (Wright & Eason 1991, unpublished FRI contract report). Consequently, invertebrates may pose a risk of secondary poisoning to insectivorous vertebrates. One example of secondary poisoning of insectivorous birds with brodifacoum has been reported in a zoo, where avocets, rufous-throated ant pittas, golden plovers, honey creepers, finches, thrushes, warblers, and crakes died in an aviary after feeding on pavement ants and cockroaches that had eaten brodifacoum baits (Godfrey 1985).

Secondary poisoning of non-target predatory and scavenging species has been reported after use of both first and second-generation anticoagulants. The risk of secondary poisoning of non-target species is far greater with second-generation anticoagulants, such as brodifacoum, because they are not substantially metabolised and excreted before death.

In pen trials, owls have consistently survived after eating rodents poisoned with first-generation anticoagulants. For example, tawny owls fed mice poisoned with warfarin were not killed, and no physical or behavioural changes were observed. However, plasma prothrombin levels were affected, and the authors suggested that this might have deleterious effects on owl wellbeing (Townsend

et al. 1980). In a separate study, all barn owls fed rats poisoned with three first-generation anticoagulants (diphacinone, fumarin, or chlorophacinone) survived without symptoms (Mendenhall & Pank 1980).

However, barn owls were killed by two second-generation anticoagulants, difenacoum and brodifacoum. Five out of six barn owls deliberately fed rats that had eaten brodifacoum died. Difenacoum is less potent than brodifacoum and only one out of six owls fed difenacoum-poisoned rats died. In New Zealand, a pen trial with Australasian harriers has confirmed the risk of secondary poisoning with brodifacoum. One out of four harriers died after eating rabbits poisoned with brodifacoum (Godfrey 1985).

In field trials, barn owls in an oil palm plantation in Malaysia were tolerant of secondary ingestion of warfarin for rat control but the population declined from 20 breeding pairs to two individuals after use of second-generation anticoagulants, firstly coumachlor, then brodifacoum; carcasses showed signs of haemorrhaging (Duckett 1984). Screech-owls in USA were killed by secondary poisoning from brodifacoum baits used to control voles in orchards (Hegdal & Colvin 1988). In New Zealand, dead southern black-backed gulls and Australasian harriers collected after a rabbit-poisoning operation contained residues of brodifacoum (Rammell *et al.* 1984; Williams *et al.* 1986), and a morepork was found dead after Talon® 20P was aerially sown for eradication of kiore and rabbits on Stanley Island (Towns & McFadden 1991, unpubl. DoC report). However, field reports indicate that the extent of bird deaths resulting from brodifacoum use depends on the way baits are used and the behaviour of non-target species.

In the United Kingdom, brodifacoum use on farms caused secondary poisoning of tawny owls, buzzards, and corvids (Edwards & Swaine 1983, unpubl. ICI report), but secondary poisoning was reduced by pulsed *versus* saturation baiting, and by use of baits close to farm buildings (Edwards *et al.* 1984a, unpubl. ICI report). Residue levels were significantly lower in poisoned rats after pulse baiting than after saturation baiting (Edwards *et al.* 1984b, unpubl. ICI report). Barn owls in the United Kingdom are now widely exposed to brodifacoum, but not all owls exposed are likely to receive a lethal dose, and there is no evidence that brodifacoum has affected barn owl populations (Newton *et al.* 1990).

Predators may not be affected when other food sources are readily available. For example, baiting with brodifacoum in and around farm buildings in the USA did not affect local barn owls, which fed on rodents in grassland away from buildings (Hegdal & Blaskiewicz 1984).

The use of brodifacoum bait must be evaluated with a full understanding of the wildlife present in an area designated for rodent control. For example, brodifacoum baiting to control rodents in rice fields in the Philippines had no effect on wildlife (Brown *et al.* 1986, unpubl. ICI report), but there were no rodent predators present.

In New Zealand, observations by researchers suggest that the risks of both primary and secondary poisoning can be reduced by the use of baits in bait stations. Comparable numbers of brown skuas and New Zealand falcons, the main avian predators at risk, were seen before and after use of Talon® 50WB in

bait stations for eradication of Norway rats on Hawea Island (Taylor & Thomas 1989). There was no evidence of New Zealand falcons or moreporks being killed by use of Talon® 50WB in bait stations for eradication of Norway rats on Breaksea Island (Taylor & Thomas 1993).

Nevertheless, the hazards of secondary poisoning to non-target wildlife have prevented second-generation anticoagulants such as brodifacoum being registered in the USA for non-commensal (field) use (Colvin *et al.* 1991).

5.4 BIRDS AND BATS AT RISK FROM BRODIFACOUM POISONING IN NEW ZEALAND

Two species of bats and about 150 species or sub-species of birds (half of which are land and freshwater birds) are indigenous to New Zealand. Both species of bats and about 60 species of birds (Table 3) occur in areas where brodifacoum baits could be used for rat control. Any bats or birds that come into direct or indirect contact with brodifacoum baits would be at risk from primary or secondary poisoning.

Bats

Short-tailed bats are primarily insectivorous but also eat carrion and might feed on brodifacoum-poisoned carcasses. They might also eat cereal-based baits. The death of a short-tailed bat has been attributed to the consumption of cyanide bait laid for possum control (Daniels & Williams 1984). Consequently, short-tailed bats may be at risk from both primary and secondary poisoning. Long-tailed bats are thought to be entirely insectivorous and therefore at risk only from secondary poisoning.

Birds

About 30 species or sub-species of birds would probably eat cereal-based baits containing brodifacoum if they encountered them (Table 3). For example, North Island brown kiwi ate non-toxic cereal-based baits (Wanganui No.7) containing Rhodomine B that marked their faeces (Pierce & Montgomery 1992, unpublished DOC internal report). Paradise shelducks, grey ducks, weka, and robins have been seen eating cereal-based baits intended for possums. Weka, robins, and tomtits have been found dead after possum control operations using 1080 in cereal-based baits. Captive weka, kaka, parakeets, kokako, and saddlebacks ate cereal-based baits in feeding trials (Spurr 1993, in press). Cereal-based pellets normally fed to poultry are included in the diet of captive kokako and saddlebacks. New Zealand pipits eat seeds and would probably eat cereal-based baits if they encountered them in the wild. Some birds have been reported taking baits from bait stations. Weka and kea have both removed Talon® 50WB from novacoil bait stations (Taylor 1984, Taylor & Thomas 1993), and kaka have eaten apple paste from modified Romark bait stations (Sherley 1992). These species are inquisitive and could eat toxic cereal-based baits from bait stations.

TABLE 3 INDIGENOUS BIRD SPECIES OR SUB-SPECIES[1] AT RISK IN AREAS WHERE CEREAL-BASED BAITS COULD BE USED FOR RODENT CONTROL

PROBABLY WOULD EAT CEREAL-BASED BAITS IF ENCOUNTERED[2]	PROBABLY WOULD NOT EAT CEREAL-BASED BAITS IF ENCOUNTERED BUT MIGHT BE AT RISK FROM SECONDARY POISONING
North Island brown kiwi*	Australasian harrier+
South Island brown kiwi	New Zealand falcon
Stewart Island brown kiwi	Brown Skua
Little spotted kiwi	Southern black-backed gull+
Great spotted kiwi	Shining cuckoo
Paradise shelduck*+	Long-tailed cuckoo
Grey duck*	Morepork+
North Island weka*	New Zealand Kingfisher
Western weka+	North Island rifleman
Buff weka	South Island rifleman
Stewart Island weka	Welcome swallow
Pukeko	North Island fernbird
Red-billed gull	South Island fernbird
New Zealand pigeon	Stewart Island fernbird
Chatham Island pigeon	Codfish Island fernbird
North Island kaka*	Brown creeper
South Island kaka	Whitehead
Kea	Yellowhead
Red-crowned parakeet*	Grey warbler
Chatham Island red-crowned parakeet	Chatham Island warbler
Yellow-crowned parakeet	North Island fantail
Forbes' parakeet	South Island fantail
New Zealand pipit	Chatham Island fantail
North Island robin+	North Island tomtit
South Island robin+	South Island tomtit
Stewart Island robin	Chatham Island tomtit
Silvereye+	Stitchbird
North Island kokako*+	Bellbird
North Island saddleback*+	Tui
	Chatham Island tui

1 *Names follow Turbott (1990).*

2 *All species in this column are known to have eaten cereal-based baits or are considered likely to eat them.*

* *Known to have eaten cereal-based baits.*

\+ *Found dead after pest control operations using cereal-based baits (Wanganui No.7, Mapua, RS5, Talon® 20P) or Talon® 50WB.*

Insectivorous birds (e.g., riflemen, whiteheads, grey warblers, and tomtits) though listed as unlikely to eat cereal-based baits containing brodifacoum, might do so occasionally for the same reasons that they sometimes eat fruit (Spurr 1979, 1991). However, insectivorous birds are more likely to be exposed to brodifacoum by eating invertebrates that have fed on toxic baits; i.e., they are more likely to be at risk from secondary poisoning. Predatory birds (especially the Australasian harrier, New Zealand falcon, and morepork) might also be at risk from secondary poisoning by eating birds, small mammals, or invertebrates that have fed on toxic baits. Australasian harriers, for example, have been found dead after eating rabbits poisoned with brodifacoum (Rammell *et al.* 1984; Godfrey 1985; Williams *et al.* 1986), and moreporks have been found dead presumably from secondary poisoning after eating rodents poisoned with Talon® 20P (Towns & McFadden 1991, unpubl. DoC report) and 1080 (Spurr 1979, 1991). Some birds such as the Australasian harrier, weka, and southern black-backed gull could be at risk from secondary poisoning by scavenging on poisoned carcasses.

6 Conclusions

Brodifacoum has the potential to cause both primary and secondary poisoning of birds and bats. However, the adverse effects of brodifacoum on wildlife are dependent on how baits are used and on the behaviour of non-target species. Baits in bait stations are likely to be less accessible to non-target species than baits on the ground. Secondary poisoning of birds is likely where rats are a major constituent of the diet (e.g., of the weka). The risk from brodifacoum will be at its greatest when saturation baiting techniques, such as aerial sowing, are used in eradication programmes.

Indigenous New Zealand vertebrates most at risk from feeding directly on cereal-based baits containing brodifacoum are those species that are naturally inquisitive and have an omnivorous diet (birds such as weka, kaka, kea, and robins). The greatest risk is probably that of secondary poisoning to predatory and scavenging birds (especially the Australasian harrier, New Zealand falcon, southern black-backed gull, morepork, and weka). Some species are at risk from both primary and secondary poisoning. However, laboratory and field trials are essential to determine the actual risks associated with cereal-based baits containing brodifacoum and with aerial baiting strategies.

7 Recommendations

- The risk of bats and birds eating cereal-based baits should be investigated initially in laboratory and field studies using non-toxic baits.

- The risk of secondary poisoning to insectivorous non-target species should be investigated in laboratory and field studies to determine levels of brodifacoum residues and their persistence in selected invertebrates.
- The risk of primary and secondary poisoning to indigenous non-target species from brodifacoum, especially in aerially sown toxic bait in both single-pulse and repeated-pulse strategies, should be field-tested in an area containing some key non-target species identified in this report. Bird, bat, and invertebrate numbers should be monitored before and after poisoning, and residue analyses for brodifacoum in water, soil, and animals should be undertaken. The benefits of reduced aerial application rates and repellents to deter non-target species should be investigated.

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