

Environmental fate and residual persistence of brodifacoum in wildlife

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Environmental fate and residual persistence of brodifacoum in wildlife

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Summary

Project and Client

- Hawke's Bay Regional Council contracted Landcare Research (Envirolink 884-HBRC131) in Mar–Oct 2010 to review literature on the environmental toxicology of the vertebrate toxic agent brodifacoum. The information was intended to guide management and control strategies to mitigate the risks posed by the use of brodifacoum in pest programmes in the Hawke's Bay.

Objective

- Summarise current knowledge about the environmental effects of the use of brodifacoum as a vertebrate toxic agent, particularly the residual persistence of brodifacoum and potential effects on non-target wildlife and livestock.

Methods

- Scientific literature from published and unpublished sources was reviewed. This included laboratory research and field-based monitoring of brodifacoum in the environment, with special focus on New Zealand uses for pest animal management and the context of brodifacoum use in the Hawke's Bay.

Results

- Research over the last 10 years indicates that the contamination of non-target wildlife by the anticoagulant rodenticide brodifacoum is likely to be widespread and mediated through a wider range of environmental transfer pathways than are currently described, e.g. invertebrates as vectors of residues.
- Despite New Zealand field research in the 1990s that demonstrated secondary mortality in some non-target species, and the occurrence of residual brodifacoum in a range of wildlife, there has been little ongoing monitoring or investigation of the longer-term implications of the continued field use of brodifacoum for possum and rodent control.
- For example, there appears to have been no brodifacoum testing of livers from wildlife species sampled in the Hawke's Bay area since 2002 (based on the Landcare Research Toxicology Laboratory database). Samples that have been tested from the Hawke's Bay are mostly from predatory mammals (stoats, cats, weasels) or game mammals (pigs, deer) that were tested as part of formal field research, rather than ongoing monitoring. Very few native birds have been tested from the Hawke's Bay area (one North Island robin and one weka).

Conclusions

- Regional management agencies and private land managers in New Zealand use brodifacoum in bait stations for possum control (e.g. 'possum control areas' PCA

programmes) mainly because of its favourable cost-efficacy compared to other control tools, i.e. high efficacy of brodifacoum against possums, availability of baits to non-licensed users, and the relatively low cost of baits and labour required to maintain bait stations.

- There is growing evidence that even the more restricted uses of brodifacoum for commensal rodent control can result in secondary poisoning and residues in non-target wildlife. This suggests that large-scale, ongoing field applications of brodifacoum in bait stations in New Zealand are likely to be contaminating a range of non-target mammals, birds and invertebrates. For some species this could mean an as-yet unknown but potentially significant mortality through accumulation of liver residues.
- Research and monitoring data clearly show the potential for environmental transfer of brodifacoum residues and non-target mortality, but there has been no ongoing evaluation or monitoring of the longer term environmental impacts of sustained field applications of brodifacoum in New Zealand.
- The potential environmental costs of brodifacoum use need to be considered in balancing the benefits and costs of pest control. Understanding, then demonstrably managing, these risks will better enable the ongoing availability of important on-ground pest control tools to land managers.

Recommendations

- Hawke's Bay Regional Council should support research to provide basic information about how brodifacoum is most commonly transferred from bait stations into the wider environment to allow identification of the most prevalent residue transfer pathways and development of measures to reduce residue transfer, by:
 - Testing soil from under well-established and frequently refilled bait stations to determine whether residual brodifacoum concentrations are present as the result of PCA baiting programmes
 - Quantifying the amounts of bait/brodifacoum that are typically removed from bait stations to the wider environment by rodent or possum spillage and by invertebrate activity
 - Conducting a formal wildlife residue survey in areas where bait station use is widespread to gauge the extent of non-target wildlife contamination in Hawke's Bay

1 Introduction

Many regional councils require landowners within designated 'Possum Control Areas' (PCA) to maintain low possum densities. Generally in PCA, the council arranges for initial possum control to low densities, and then requires landowners to maintain possum numbers at or below a 5% residual trap-catch. A range of traps and toxic baits are available for these applications, and often councils will subsidise purchase of these. The purchase and field use of the brodifacoum bait formulations 'Talon' and 'Pestoff' do not require a controlled substances licence (National Possum Control Agencies (NPCA) 2006), making the use of brodifacoum baits in bait stations a readily accessible control tool to landowners involved in 'self-help' PCA. The Hawke's Bay Region has a relatively large PCA coverage, and probably one of the largest bait station programmes in New Zealand where brodifacoum is applied for possum control. A current estimate of use is 12–14 tonnes of brodifacoum bait per year, deployed in around 45 000 bait stations set across 430 000 ha (Campbell Leckie, pers. comm., August 2010).

To guide management and control strategies to mitigate the risks posed by the use of brodifacoum in pest programmes in the Hawke's Bay, Hawke's Bay Regional Council (HBRC) contracted Landcare Research (Envirolink 884-HBRC131) in Mar-Oct 2010 to review literature on the environmental toxicology of the vertebrate toxic agent brodifacoum. This report summarises the results of research and monitoring to date relevant to environmental fate and effects of brodifacoum, identifies new or significant findings about brodifacoum that could influence future best practice, and identifies and prioritises current information gaps.

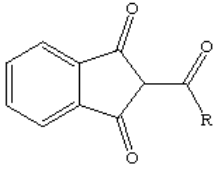
2 Background

Brodifacoum is one of the compounds in the 'family' of anticoagulants that have been used worldwide for control of rodent and other mammalian pests (e.g. Kegley et al. 2007). Application of brodifacoum bait for commensal rodent control is generally limited to 'indoor' use and bait station deployment, e.g. within a nominated distance of buildings using fixed baits in tamper-proof bait stations. In some countries, including New Zealand, bait formulations for household rodent control are available 'over the counter' to the public while in other places, such as the United Kingdom, brodifacoum use is restricted to indoor use by licensed or professional applicators.

However, New Zealand use-patterns of brodifacoum differ from most other countries, in that bait formulations (0.005% or 0.002% brodifacoum by weight) are also registered for field application against rodents and possums. Hoare and Hare (2006) provide an overview of brodifacoum use in New Zealand; bait station deployments of brodifacoum can cover considerable mainland areas and may be sustained for a number of years in certain key areas. Of the estimated 6 kg of brodifacoum (as active ingredient in bait) sold annually in New Zealand, approximately 50% is used by professional pest contractors, 30% by regional councils, 15% by the Department of Conservation (DOC) and 5% by private landowners (Hoare & Hare 2006).

Anticoagulants can be classified as indandiones or coumarins by chemical structure, and also as first-generation or second-generation according to when they were first available as rodenticides (Table 1).

Table 1 Date of development and use of first- and second-generation anticoagulant rodenticides, and their grouping by chemical structure (British Crop Protection Council 2000)

First generation	1942: Pindone 1952: Diphacinone c.1962: Chlorophacinone	Indandione	 R = allyl, aryl
	1944: Warfarin 1962: Coumatetralyl		
Second generation	1975: Difethialone		
	1976: Brodifacoum		
	1978: Bromadiolone		
	1984: Flocoumafen		
	1986: Difenacoum		

All of these compounds have a common mode of toxicity, through inhibition of the normal synthesis of vitamin K-dependent blood clotting factors in the liver (e.g. Thijssen 1995). When this inhibition occurs over a sufficient time blood will not coagulate, and typical clinical signs of anticoagulant toxicity are haemorrhage and anaemia, with death through massive haemorrhage occurring several days after a lethal exposure (Pelfrene 2001). In general, the first-generation anticoagulants (e.g. warfarin, pindone, diphacinone) are most toxic when ingested in multiple, consecutive doses whereas the second-generation anticoagulants, particularly brodifacoum, are considered 'single feed' poisons because of their greater oral toxicity. Brodifacoum is the most toxic of the second-generation anticoagulants to mammals and birds. Compared with the first-generation anticoagulants, it also has a high residual persistence in liver tissue, the main site of toxic action (e.g. Eason et al. 1996; Fisher et al. 2003). Brodifacoum is less persistent in blood, fat and muscle tissue than in liver (e.g. Laas et al. 1985), so detection of residual brodifacoum in liver tissue of mammals and birds has been a focus for monitoring its fate in the environment.

Brodifacoum, because of its broad-spectrum, high toxicity to mammals and birds, poses an unwanted hazard for non-target wildlife or domestic species that ingest bait (primary exposure) or ingest tissues of animals containing residual concentrations of brodifacoum (secondary exposure). It has a relatively high risk of causing secondary poisoning in comparison with other rodenticides (Erickson & Urban 2004) through combined high toxicity and relatively prolonged residual persistence in liver. Current restrictions in the United Kingdom and Europe on the use of brodifacoum for commensal rodent control reflect its potential for unwanted impacts through secondary poisoning (e.g. Baker et al. 2007). In New Zealand, evidence of secondary effects and brodifacoum contamination of wildlife (e.g. Eason et al. 2002) prompted the Department of Conservation (DOC) to restrict the use of brodifacoum for conservation purposes on the mainland (DOC 2000), and current DOC uses of brodifacoum bait are mostly for eradication of introduced rodents from islands (e.g. Towns & Broome 2003). However, the continuing use of bait formulations of brodifacoum for commensal rodent control in New Zealand and, particularly, for field applications in bait stations for possum control, pose significant risks of inputs of brodifacoum into the wider environment.

3 Objective

- Summarise current knowledge about the environmental effects of the use of brodifacoum as a vertebrate toxic agent, particularly the residual persistence of brodifacoum and potential effects on non-target wildlife and livestock.

4 Methods

A literature review was undertaken, covering peer-reviewed scientific publications, unpublished material available from pest management agencies, and data available through the Vertebrate Pesticide Residues Database maintained by the Landcare Research Toxicology Laboratory, with special focus on New Zealand uses for pest animal management and the context of brodifacoum use in the Hawke's Bay. Non-target species were considered in the categories of 'wildlife' (birds, invertebrates, game animals) and 'domestic animals' (pets and livestock). In particular, publications from the last decade were sought that dealt with:

- The fate of brodifacoum in soil and water
- Primary poisoning risks to non-target species in New Zealand
- Secondary poisoning risks to non-target species in New Zealand
- Environmental transfer pathways of brodifacoum and residue monitoring

5 Results

5.1 Brodifacoum in water

Bait station applications normally prevent baits directly entering waterways. Even when brodifacoum baits could potentially enter waterways, e.g. following aerial application of cereal pellets, monitoring data indicate that water contamination by residual brodifacoum is highly unlikely. Monitoring of fresh water after aerial applications of cereal pellet bait containing 20 ppm (parts per million) brodifacoum on Red Mercury Island (Morgan & Wright 1996), Lady Alice Island (Ogilvie et al. 1997), Maungatautari (217 water samples tested), Little Barrier Island and Rangitoto/Motutapu Islands (Fisher et al. in press) has found no detectable brodifacoum. On the assumption that baits entered waterways as the result of these aerial applications, factors likely to have contributed to such results are brodifacoum's overall low water-solubility, especially at acidic and neutral pH (British Crop Protection Council 2000), the adsorption of brodifacoum to organic particles (World Health Organisation 1995), and dilution with water volume and flow rate.

5.2 Brodifacoum in soil

Brodifacoum is effectively immobile in soil because of its very low water solubility. In leaching studies, only 2% of brodifacoum added to the soil leached more than 2 cm from its source in four soil types tested (World Health Organisation 1995). Once in soil, brodifacoum degrades at rates that vary with soil type. The mechanisms and pathways of brodifacoum

degradation in soil are not well described, but half-life estimates (the time taken for the residual concentration of brodifacoum to decrease by half) in soil range from 12 to 25 weeks (US EPA 1998; ICI unpublished data).

Soil immediately underneath degrading cereal bait pellets containing brodifacoum (i.e. baits placed on the ground) can become contaminated with brodifacoum. In a study at Tawharanui, low concentrations of brodifacoum (0.02–0.2 ppm) were measured in 69% of soil samples taken from underneath/around degrading baits (Craddock 2004), with the highest concentration of 0.2 ppm on day 84. After 110 days all soil concentrations were below the limit of detection of the method of analysis (<0.02 ppm). Similar monitoring after aerial application of Pestoff20R on Little Barrier Island measured a highest concentration in soil under pellets of 0.07 ppm at 153 days (Fisher et al. in press).

Because there is potential for spillage of bait from stations by possums or rats, and the fragmentation of baits within stations by invertebrate activity or weathering of pellets, baits and bait fragments may reach the soil below and around a bait station, potentially over a sustained period if bait stations have remained in the same position and been refilled over months or years. Because residual brodifacoum is unlikely to disperse widely in soil, this may result in gradually increasing concentrations of brodifacoum in the soil immediately beneath bait stations. This possibility has not been investigated and is a major gap in current understanding of potential environmental effects.

5.3 Primary poisoning risks

A variety of bait stations are used to apply brodifacoum bait for possum control (NPCA 2009). Depending on the type of bait station, they can hold from 0.2 to 2 kg of pellet bait containing 20 ppm of brodifacoum (equivalent to 0.02 g brodifacoum per kilogram of pellet bait, or 0.002% brodifacoum by weight). A ‘pulse baiting’ strategy is recommended for possums, with stations initially filled for 3–4 days and thereafter refilled every 14 days (2009). This is intended to minimise bait uptake while maintaining kill efficacy by taking into account the known progression of brodifacoum poisoning in possums. The delayed onset of brodifacoum poisoning symptoms means that possums that have ingested a lethal amount of bait (see Table 2) may continue to eat bait for several days before becoming ill and ceasing to feed (e.g. Littin et al. 2000). The mean time to death in captive possums poisoned with brodifacoum has been estimated as c. 21 days (Littin et al. 2002).

Table 2 summarises the toxicity of brodifacoum to a range of species, expressed as the ‘LD₅₀’, the amount of brodifacoum ingested relative to bodyweight (milligrams of brodifacoum per kilogram of weight; mg/kg) that will kill 50% of the population; the smaller the LD₅₀ value, the higher the susceptibility of the species. Based on the LD₅₀ and a given ‘mean’ bodyweight for each species, Table 2 also summarises the amount of brodifacoum bait that each species would need to consume to ingest a lethal dose. Ingestion of brodifacoum may be lethal or sub-lethal, depending on the amount eaten and the susceptibility of the animal. Table 2 shows that body size as well as susceptibility (LD₅₀) influences the relative amounts of bait estimated to be lethal for different species.

Table 2 Estimated lethal amounts (g) of 20 ppm brodifacoum bait for representative species

Species	LD ₅₀ brodifacoum (mg/kg)	Bodyweight (kg)	Lethal amount of 20 ppm brodifacoum bait for 50% of animals (g)
Possum	0.17	3.0	25.5
Dog	3.56	10	1780
Sheep	11.0	45	25000
Pig	1.8	45	4050
Ship rat	0.27	0.15	2.1
Sparrow	6.0	0.03	9
Pūkeko	0.95	0.9	42.8
Paradise shelduck	20	1.5	1500

Table 2 illustrates that omnivorous or granivorous birds, especially small species such as sparrows, could consume lethal amounts of bait if they had access to it. Best-practice recommendations are to raise bait stations out of the reach of ground-dwelling birds such as kiwi, weka and robins when placed in their habitats (NPCA 2009). Most bait stations have design features that are thought to exclude non-target birds from bait, but rodents, particularly mice and ship rats, are probably able to access bait from most types of station. To date, bait 'spillage' and/or removal of bait for caching elsewhere by rodents (e.g. Lund 1988) and the extent to which these activities make bait more readily available to non-targets have not been measured. Another significant source of primary non-target exposure to brodifacoum bait is spillage from bait stations by possums, and this also has not been quantified. For bait station applications, estimates of rates of spillage and the fate of spilled bait are needed to estimate the primary poisoning risk for bird species known to eat cereal pellets, such as tomtits (Spurr 1994), pūkeko and paradise shellduck (Eason & Spurr 1995).

Best practice is for stock to be excluded from areas treated with bait station areas to prevent them from directly interfering with stations to access bait (NPCA 2009). Table 2 suggests that for livestock (e.g. sheep,) primary poisoning is unlikely, as kilogram amounts of bait are needed for a lethal dose, implying the contents of multiple bait stations would need to be accessed. This also applies to other large herbivores such as deer or cattle. Monitoring of wild deer for primary exposure to bait found that 11 out of 33 wild red deer shot in areas where brodifacoum had been used had detectable residues in liver, although concentrations did not exceed 0.03 mg/kg (Eason et al. 2002). Spurr et al. (2005) found no residues in the liver of five red deer shot in his research study area during a period of brodifacoum bait use.

Even though feral pigs also require a reasonably large quantity of bait for a lethal exposure (Table 2), some individuals or groups of pigs may have increased risk of lethal or sub-lethal exposure if they learn that bait stations can be damaged to obtain bait (Morriss et al. 2005). The obvious solution is to switch to another possum control method in locations where pig damage to stations becomes widespread or recurring. However, because feral pigs also scavenge carcasses, they are subject to secondary brodifacoum exposure (see section 5.4.1).

The toxicity of brodifacoum to invertebrates is not well described, but the small number of captive studies all indicate low toxicity of brodifacoum, e.g. to large-headed tree weta (Booth et al. 2001) and Ascension Island land crabs (Pain et al. 2000). Craddock (2003) found that

captive locusts fed readily on cereal-based brodifacoum baits with no significant increase in mortality. Weight loss and mortality of captive cave weta and ground weta were not significantly higher in weta exposed to brodifacoum bait over 60 days than in non-exposed animals (Bowie & Ross 2006). Overall, this suggests that arthropod invertebrates have a much lower susceptibility to brodifacoum than mammals, such that they are not at high risk of primary poisoning risk. Until recently (Craddock 2003), however, invertebrate feeding on cereal pellet bait had not been assessed in terms of its long-term contribution to bait fragmentation/weathering and transfer of residual brodifacoum from bait stations into the wider environment. Field monitoring following brodifacoum baiting in New Zealand has detected residues in some invertebrate species, particularly those that eat baits (summarised by Booth et al. 2001). Thus invertebrates are also part of the pathway of anticoagulant residues in the environment, presenting a secondary hazard to insectivores. This is discussed further in section 5.4.3.

5.4 Secondary poisoning risks

Predatory or scavenging species have the highest risk of secondary exposure to brodifacoum through preying on live animals that contain residual brodifacoum or scavenging carcasses of animals killed by brodifacoum poisoning. The liver tissue and, in some cases, gut contents (when these contain partially digested brodifacoum bait) of prey or carcasses pose the highest hazard in terms of the potential amount of residual brodifacoum. Ingestion of brodifacoum from such sources may be lethal or sub-lethal, depending on the amount eaten and the susceptibility of the species, as demonstrated by studies on captive animals (e.g. Joermann 1998). Borst and Counotte (2002) document mortality from secondary brodifacoum exposure in captive bird species: two turkey vulture chicks that died of toxicosis after being fed rodenticide-killed mice by the adult birds. Godfrey (1985) reports that several birds in a zoo aviary died, apparently as the result of eating ants and cockroaches that had eaten brodifacoum baits.

Secondary exposure to anticoagulants including brodifacoum (indicated by the presence of residues in liver) has been described internationally in an increasing range of non-target wildlife, despite the more restricted applications of anticoagulant baits for commensal rodent control in and around buildings, e.g. in the United Kingdom (Brakes & Smith 2005) and the United States (Hoops 2005). The same pathway for brodifacoum transfer from communal rodents has been reported in New Zealand; Spurr et al. (2005) provide evidence that anticoagulant baits used in St Arnaud village and nearby farms were responsible for residues in non-target wild mammals trapped in a surrounding 8-km-radius zone. It is not surprising that the uniquely New Zealand broad-scale field use of brodifacoum in bait stations, often over extended periods, has also resulted in secondary exposure and mortality of non-target wildlife, as indicated by residue monitoring (Eason et al. 2002).

5.4.1 Mammals

Brodifacoum residues have been detected in predatory mammals in the United Kingdom and United States, including polecats/ferrets (Shore et al. 2003), coyotes, foxes, bobcats (Hosea 2000) and mountain lions (Hosea 2000; Hoops 2005). In these countries, brodifacoum use is restricted to commensal rodent control (indoor) applications yet predatory or scavenging wildlife species have been exposed. Field studies of predatory mammals (stoats, ferrets and feral cats) in New Zealand have confirmed both sub-lethal (Eason et al. 2002) and lethal

(presumably secondary exposure; Alterio 1996; Alterio & Moller 2000) to brodifacoum following bait applications for rodent, rabbit or possum control. While the by-kill of these other pest mammals may have contributed to the desired outcomes of the control operation, such cases highlight the potential for secondary poisoning of domestic cats or dogs where there is a high availability of affected (easily-caught) rats, or rat/possum carcasses (e.g. Bradley 2009).

As highly efficient, omnivorous scavengers, feral pigs are particularly prone to secondary exposure to brodifacoum and resulting residue burdens in the liver. In trials where captive pigs were fed the soft tissue of possums poisoned by brodifacoum (Eason et al. 1999), residual brodifacoum concentrations of 0.32 to 0.80 mg/kg were detected in the pigs' livers 5 days later, and were dose-dependent, i.e. the more possum tissue eaten by a pig, the higher the residual brodifacoum in liver. Brodifacoum was detected in muscle, at much lower concentration than in liver, from only one of these pigs (Eason et al. 1999). Feral pigs may also be exposed to brodifacoum by scavenging poisoned rat carcasses (Morriss et al. 2005). Eason et al. (2002) reported 21 (60%) out of 35 liver samples from feral pigs from areas where brodifacoum was being used for possum and rat control had detectable residues of brodifacoum in their livers, with concentrations ranging from 0.007 to 1.78 mg/kg. The New Zealand Food Safety Authority specifies caution periods and buffer zones for recreational hunting of wild deer and other game species in areas where brodifacoum has been used, in the context of the risk of human consumption of meat containing residual brodifacoum.

Spurr et al. (2005) found that 47.6% of hedgehogs (21 animals), sampled from the St Arnaud area during a period of brodifacoum use in bait stations, had detectable brodifacoum in liver with a mean concentration of 0.20 mg/kg. Recent monitoring of hedgehogs in Britain (Dowding et al. 2010) indicates that an overall proportion of 57.5% had detectable liver residues of at least one, and sometimes multiple, second-generation anticoagulant compounds including brodifacoum, bromadiolone and difenacoum. This is noteworthy given the restricted uses of these compounds in Britain. Hedgehogs have received relatively less attention in terms of secondary brodifacoum exposure in New Zealand, probably because they are not a common food item for people, are perceived as being less of a predator/scavenger, and also as less of a pest than other introduced mammals. However, as insectivores/omnivores, the potential exposure of hedgehogs to brodifacoum in the environment may be similarly high to that of predators and scavengers of carcasses. Hedgehogs may thus be a useful indicator species for monitoring brodifacoum residues in some parts of New Zealand, and for improving understanding of secondary environmental transfer pathways.

5.4.2 Birds

Internationally, brodifacoum and other anticoagulant residues have been detected in a range of hawk and owl species in the United Kingdom, United States and Canada (Newton et al. 2000; Mineau et al. 2003; Stone & Okoniewski 2003). Preliminary data from a recent small survey in New Zealand, using tissue from road-killed harrier hawks collected in mid-Canterbury in 2010, indicate that at least half of the hawks had detectable concentrations of at least one coumarin anticoagulant (bromadiolone, coumatetralyl, or flocoumafen) in their liver, including brodifacoum in 7 of the 13 hawks tested to date (Landcare Research, unpubl. data). Whether the prevalence of brodifacoum residues in hawks, and other

predatory/scavenging birds would be similar or higher in other regions where extensive bait station applications of brodifacoum are ongoing, e.g. Hawke's Bay, remains to be established.

Bird species likely to prey on rodents, or to scavenge possum/rodent carcasses, are at obvious risk of exposure to brodifacoum residues, and there is growing evidence that insectivorous bird species are also exposed secondarily. Dowding et al. (2006) documented mortality and brodifacoum residues in New Zealand dotterels that had apparently fed on sand hoppers (invertebrates) that had eaten bait and contained residual brodifacoum. More recently, residual brodifacoum was detected in livers of three of nine little blue penguins found dead on beaches in the Hauraki Gulf in 2009 (Fisher et al. in press). The penguins were tested soon after an aerial application of brodifacoum pellet baits for pest eradication on nearby Rangitoto and Motutapu islands to address community concerns of non-target mortality in the wider area. Overall necropsy findings suggested that the cause of penguin mortality was not brodifacoum poisoning but starvation, consistent with previous seasonal instances of 'beachings' of malnourished penguins in the area. However, the presence of low concentrations of brodifacoum in some penguins indicates an unconfirmed environmental pathway of exposure. This may have originated from the aerial application of bait on Rangitoto and Motutapu; however, the known prolonged persistence of brodifacoum in liver also meant that exposure could have occurred some months before this baiting operation (Fisher et al. in press). The ongoing use of brodifacoum for commensal rodent control (e.g. around baches, marinas, on boats) in areas where penguin burrows are present may also present an exposure pathway to penguins, with marine/intertidal invertebrates a possible secondary vector of residues.

More systematic monitoring is needed to better characterise the exposure of non-target birds to brodifacoum used for pest control. In this context morepork would be an appropriate 'focus' species, as a native predator of both rodents and invertebrates. Secondary poisoning of morepork has been confirmed, through residue testing of small numbers of carcasses recovered after one-off applications of brodifacoum bait for pest eradication from Kapiti (Empson & Miskelly 1999), Mokoia (Stephenson et al. 1999) and Chetwode (Walker & Elliott 1997) islands. No testing of morepork carcasses or radio-tracking studies of the fate of moreporks have been done where the field use of brodifacoum in an area has been ongoing over a number of years.

5.4.3 Invertebrates as residue vectors

Invertebrates appear to be less at risk of primary brodifacoum poisoning if they feed on baits, but can carry residual concentrations in their bodies after doing so. Based on a small number of captive studies, residual brodifacoum appears less persistent in invertebrates than in mammalian liver; residues were not detectable 4 days after exposure in captive weta (Booth et al. 2001), after 6 days in captive locusts (Craddock 2003), and after 1 month in land crabs (Pain et al. 2000). Brodifacoum residues were found in both the gut (3.9 µg/g) and foot tissue (1.2 µg/g) of common garden snails 14 days after they were exposed to soil mixed with ground-up bait at 2 mg brodifacoum/kg soil (Booth et al. 2003).

In a New Zealand field study, terrestrial invertebrates (weta, cockroaches, beetles and other 'miscellaneous' species) were monitored for residues before, during and after application of brodifacoum baits in stations at Tawharanui (Craddock 2003). While background 'trace'

concentrations of brodifacoum were apparently present in some invertebrates before baiting started, some invertebrates contained much higher residues of brodifacoum (up to 7.47 µg/g) during the baiting period and these were dependent on the amount of toxic bait available in stations. Invertebrates carrying brodifacoum were found to disperse up to 10 m from the loaded bait stations and residue concentrations in them decreased significantly the further away from the bait stations they were sampled. After baits were removed from stations, brodifacoum residues in invertebrates took more than 4 weeks to return to 'background' levels, and trace concentrations of brodifacoum similar to those monitored before baiting were still detectable up to 10 weeks after the bait had been removed (Craddock 2003).

These results suggest that sustained bait station applications of brodifacoum are providing localised populations of some invertebrates with an ongoing source of food, and that invertebrates feeding on baits are transporting residual concentrations of brodifacoum into the immediate area around bait stations, as a result of which secondary exposure to insectivores could occur.

5.5 Monitoring of brodifacoum residues

Restrictions of the use of brodifacoum for commensal rodent control in the United Kingdom and Europe are in recognition of its potential for unwanted impacts on biodiversity (e.g. Baker et al. 2007). These countries, despite anticoagulant use being largely restricted to commensal rodent control, have undertaken anticoagulant residue testing in non-target raptors as part of formal monitoring schemes for pesticides and persistent pollutants in the environment.

Given the much more extensive field uses of brodifacoum in New Zealand, it is noteworthy that there are no formal residue monitoring systems for brodifacoum and other anticoagulants in New Zealand wildlife. Increasing evidence of secondary non-target effects and contamination of wildlife by brodifacoum, demonstrated by field-based research in the 1990s, prompted the Department of Conservation to implement restrictions on the use of brodifacoum for conservation purposes on the mainland (DOC 2000). However there has been little (if any) ongoing effort to continue monitoring to describe the extent of residue occurrence in wildlife, despite ongoing field uses of brodifacoum by other land managers. Appendix 1 summarises the results of brodifacoum testing of livers from wildlife species sampled in the Hawke's Bay area, carried out by the Landcare Research Toxicology Laboratory. In many cases these were samples from predatory mammals (stoats, cats, weasels) or game mammals (pigs, deer) that were tested as part of formal field research, rather than ongoing monitoring – there appears to have been no further brodifacoum testing of wildlife from the region since 2002. Very few native birds have been tested from the Hawke's Bay area (one North Island robin and one weka; Appendix 1).

The long-term implications of sub-lethal brodifacoum exposure for survival or reproductive fitness of affected individuals are not known. Given the persistent nature of brodifacoum, if non-target wildlife is repeatedly being exposed to brodifacoum, the potential also exists for liver residues to accumulate and exceed the toxic threshold. This has not been fully investigated but is an important question for delayed or long-term non-target impacts, particularly where field use of brodifacoum is extensive and sustained, such as in the Hawke's Bay Region. Targeted monitoring in such areas would probably reveal widespread evidence of low-level exposure to brodifacoum – i.e. low-level brodifacoum residues present in apparently healthy individuals. The relationship between liver concentrations of

brodifacoum and mortality is unclear. Use of ‘threshold’ liver concentration as a determinant of acute toxicity in mammals or birds has been suggested, with estimates of 0.7 ppm (Gray et al. 1994; Kaukeinen et al. 2000) and 0.5 ppm (Dowding et al. 1999). However, Littin et al. (2002) measured concentrations as low as 0.33 ppm in livers of lethally poisoned possums, and sub-lethally exposed chickens (*Gallus gallus*) had liver residues of 0.45–1.00 ppm (Fisher 2009). Relatively high liver concentrations (> 1 ppm) appear more strongly associated with lethal exposure, but there is overlap between the lowest ‘lethal’ and highest ‘sub-lethal’ concentrations reported. On this basis it seems more valid to relate increasing probability of lethal exposure with increasing liver concentration – as done by Myllymäki et al. (1999), who estimated that survival probability in voles (*Microtus* sp.) started to decrease at 0.20 ppm in liver.

6 Conclusions

The use of brodifacoum in bait stations for possum control by regional management agencies and private land managers in New Zealand seems largely driven by favourable cost-efficacy in comparison to other control tools, i.e. high efficacy of brodifacoum against possums, availability of baits to non-licensed users, and the relatively low cost of baits and labour required to maintain bait stations.

There is growing evidence that even the more restricted uses of brodifacoum for commensal rodent control can result in secondary poisoning and residue burdens in non-target wildlife. This suggests that large-scale, ongoing field applications of brodifacoum in bait stations in New Zealand is likely to be causing at least contamination of a range of mammals, birds and invertebrates. For some species this could mean an as-yet undetected but potentially significant mortality through accumulation of liver residues.

Despite research and monitoring data that clearly show the potential for environmental transfer of brodifacoum residues and non-target mortality, there has been no ongoing evaluation or monitoring of the longer term environmental impacts of sustained field applications of brodifacoum.

The potential environmental costs of brodifacoum use need to be considered in balancing the benefits and costs of pest control. Understanding, then demonstrably managing, these risks will better enable the ongoing availability of important on-ground pest control tools to land managers.

7 Recommendations

- Hawke's Bay Regional Council should support research to provide basic information about how brodifacoum is most commonly transferred from bait stations into the wider environment to allow identification of the most prevalent residue transfer pathways and development of measures to reduce residue transfer, by:
 - Testing soil from under well-established and frequently refilled bait stations to determine whether residual brodifacoum concentrations are present as the result of PCA baiting programmes
 - Quantifying the amounts of bait/brodifacoum that are typically removed from bait stations to the wider environment by rodent or possum spillage and by invertebrate activity
 - Conducting a formal wildlife residue survey in areas where bait station use is widespread to gauge the extent of non-target wildlife contamination in Hawke's Bay

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Appendix 1 – Hawke’s Bay wildlife tested for brodifacoum residues

Wildlife from the Hawke’s Bay Region, where liver was tested for brodifacoum residues
(data from Vertebrate Pesticide Residues Database, Landcare Research)

Species	Date	Location	Habitat	Map Grid Ref 1	Map Grid Ref 2	Brodifacoum in liver (ppm)
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.012
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	1.3
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.07
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.029
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.48
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.042
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.65
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.24
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.035
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.012
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.21
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	1.3
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.013
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.027
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.22
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.15
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.49
Cat	03-Oct-02	Boundary Stream	Bush–Pasture Margin	28388	62265	0.078
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.01
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.38
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.05
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.05
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.53
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.69
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.18
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.55
Cat	17-May-99	Boundary Stream	Forest	28410	62260	1.02
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.01
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.06
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.66

Species	Date	Location	Habitat	Map Grid Ref 1	Map Grid Ref 2	Brodifacoum in liver (ppm)
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.18
Cat	17-May-99	Boundary Stream	Forest	28410	62260	0.96
Deer	05-Feb-98	Pureora Forest, Waipapa Ecological Area	Forest	27440	63195	0
Deer	05-Feb-98	Te Urewera	Forest	28700	63128	0
Deer	23-Nov-98	Te Urewera	Forest	28700	63128	0.01
Deer	23-Nov-98	Te Urewera	Forest	28700	63128	0.02
Deer	23-Nov-98	Te Urewera	Forest	28700	63128	0.01
Deer	23-Nov-98	Te Urewera	Forest	28700	63128	0.03
Deer	23-Nov-98	Te Urewera	Forest	28700	63128	0.01
Deer	23-Nov-98	Te Urewera	Forest	28700	63128	0.03
Deer	23-Nov-98	Te Urewera	Forest	28700	63128	0
Ferret	17-May-99	Boundary Stream	Forest	28410	62260	0.07
Ferret	17-May-99	Boundary Stream	Forest	28410	62260	0.01
Ferret	17-May-99	Boundary Stream	Forest	28410	62260	0
Ferret	17-May-99	Boundary Stream	Forest	28410	62260	0.76
Ferret	17-May-99	Boundary Stream	Forest	28410	62260	0.46
NI robin	23-Jul-97	Mainland island Northern Urewera	Forest	28530	62990	0.58
Pig	05-Feb-98	Te Urewera	Forest	28700	63128	0
Pig	05-Feb-98	Te Urewera	Forest	28700	63128	0.31
Pig	05-Feb-98	Te Urewera	Forest	28700	63128	1.09
Pig	05-Feb-98	Te Urewera	Forest	28700	63128	0
Pig	05-Feb-98	Te Urewera	Forest	28700	63128	0.04
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.53
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.78
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.25
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.91
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.39
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.06
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	1.3
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.54
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.83
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.35

Species	Date	Location	Habitat	Map Grid Ref 1	Map Grid Ref 2	Brodifacoum in liver (ppm)
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	1.32
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.47
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.84
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.15
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.31
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.6
Stoat	14-Jul-98	Otamatuna, Urewera National Park		28724	63105	0.24
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.02
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.37
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.08
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.48
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.64
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.58
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.01
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.73
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.08
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.01
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.06
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.33
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.03
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.41
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.53
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.01
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.36
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.18
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.04
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.43
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.03
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.01
Stoat	17-May-99	Boundary Stream	Forest	28410	62260	0.11
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	1.17
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.01
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.4
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.86
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.81

Species	Date	Location	Habitat	Map Grid Ref 1	Map Grid Ref 2	Brodifacoum in liver (ppm)
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.78
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.73
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.05
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.98
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.04
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.36
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.7
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.48
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.03
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.17
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.82
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.01
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.29
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	1.31
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.02
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.96
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	1.17
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.01
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.4
Weasel	17-May-99	Boundary Stream	Forest	28410	62260	0.86
Weka	06-Jul-00	Pakihi Valley	Forest	28990	63317	0.49