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Environmental Monitoring for Brodifacoum Residues after Aerial Application of Baits for Rodent Eradication

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ABSTRACT: Aerial application of brodifacoum bait for eradication of invasive rodents from islands raises concerns about environmental contamination and adverse effects on nontarget wildlife. We summarize results of environmental monitoring for brodifacoum residues after New Zealand eradications in a fenced reserve at Maungatautari and on the offshore islands Little Barrier, Rangitoto, and Motutapu. Brodifacoum was not detected in extensive freshwater monitoring at Maungatautari, or in freshwater samples from Little Barrier Island. Residual concentrations were present in soil samples from underneath degrading bait pellets on Little Barrier, and decreased to near the limit of detection by c. 100 days after application. No brodifacoum was present in marine shellfish sampled from Little Barrier, Rangitoto or Motutapu. A range of birds, including a kiwi from Little Barrier, were considered nontarget mortalities. Nine little blue penguins found dead on beaches outside the Rangitoto/Motutapu area after baiting were considered most likely to have died of starvation, despite the detection of brodifacoum in three birds. This result highlights the critical role of post-application environmental monitoring in rodent eradications, and information gaps regarding the movement, persistence, and effects of brodifacoum in the environment.

KEY WORDS: aerial application, baits, brodifacoum, laboratory testing, New Zealand, nontarget species, residues, rodenticides, soil and water

INTRODUCTION

Brodifacoum is among the most toxic of the anticoagulants to rats and mice (Erickson and Urban 2004), so rodents need to ingest a relatively small amount of bait for a lethal exposure. The combination of its delayed toxicity (Kaukeinen and Rampaud 1986) and high rodenticidal efficacy, coupled with development of brodifacoum bait formulations that are highly acceptable to rodents and can be applied aerially over large areas, has provided a valuable tool for island conservation. To date, brodifacoum baiting has been used in an estimated 71% of campaigns to eradicate introduced rodents from islands (Howald et al. 2007). An important consideration has been assessing risk to nontarget wildlife and the potential for environmental contamination. Increasingly, rodent eradication is being considered for islands that are inhabited or used by people or are close to highly populated mainland areas. Where the use of brodifacoum bait is proposed, particularly through aerial application, managers also need to address possible environmental contamination pathways that pose risks to humans, livestock, and domestic animals.

Here we describe monitoring undertaken after three New Zealand eradications involving aerial application of cereal pellet bait containing 20 ppm brodifacoum, and discuss the results in the context of environmental contamination and nontarget risk. Under current New Zealand legislation, the discharge of a contaminant (e.g., brodifacoum) to land and water (e.g., through aerial bait application) often requires consent from a local government agency. While there are currently no prescriptive environmental monitoring regimes for residual brodifacoum, concerns addressed during the consent application process for each of the eradications focused attention on the fate of brodifacoum in water and soil as potential transfer pathways to human food and nontarget wildlife. Where aerial application could result in bait entering the marine environment, this included monitoring of coastal marine fauna, especially shellfish commonly harvested for human food.

METHODS

Maungatautari Water Monitoring

The Maungatautari Ecological Island Trust (MET) aims to achieve complete pest mammal eradication in this mainland reserve in the central North Island, by pest-proof fencing and removal of pest mammals through aerial baiting and trapping within the fenced area (see www.maungatrust.org/index.asp). A pilot eradication program in two fenced enclosures on the northern (c. 32 ha) and southern (c. 76 ha) sides of the mountain was undertaken in 2004. Each enclosure received two aerial applications of Pestoff® Rodent Bait 20R at a rate of 15
kg/ha, applied in accordance with a Code of Practice (Anon. 2006). Streams flowing through both enclosures were used for human or livestock drinking supply by adjoining landowners. The resource consent specified that all water supplies drawn from the enclosures be disconnected before bait application, and to remain so until two water samples taken on consecutive days showed no brodifacoum contamination, i.e., below the analytical method detection limit (MDL). Samples from two streams in each enclosure were taken at zero hours (baseline) then at 1, 2, 3, 6, 9, 12, 24, 48, and 72 h after bait application, and thereafter at 1 week, 2 weeks, and 3 months. Further samples were taken after ≥25 mm rainfall occurred in a 24-h period. Samples were taken from the point where each stream left the enclosure and at c. 800 m downstream. Samples taken up to 48 h after bait application were analysed within 24 h of receipt by the laboratory, to facilitate reconnection of water supplies once there were two consecutive below-MDL results.

### Little Barrier Island – Water, Soil, Bait Degradation, and Marine Shellfish Monitoring

Little Barrier Island is situated in the Hauraki Gulf 80 km northeast of Auckland (see www.doc.govt.nz/parks-and-recreation/places-to-visit/auckland/warkworth-area/little-barrier-island-hauturu-nature-reserve/). The Department of Conservation (DOC) undertook two aerial applications of Pestoff® Rodent Bait 20R at 11.7 and 6.2 kg/ha in June and July 2004, and the island was declared free of Pacific rats (*Rattus exulans*) in July 2006.

Carcass searches along the island’s track network and grid-searches over c. 120 ha were undertaken during the week following each bait application. One kiwi carcass recovered was necropsied (IVABS, Massey University, NZ) with liver tissue analysed for residual brodifacoum (Table 1). Monitoring of bait degradation was used to determine timing of the release of 3 brown teal (*Anas chlorotis*) taken into captivity before the operation. At 4 sites representing grassland and forested habitats across the island, 20 bait pellets were placed under wire cages designed to exclude rodents and birds, and checked for condition scoring following the categories described by Craddock (2003a), over 4 months. Soil monitoring was undertaken after peg-marking the position of individual pellets so that soil samples could later be taken from the exact location. Soil (4-cm³ plugs), collected at days 56 and 153 after the second bait application, was stored frozen until analysis. Within 24 h after both bait applications, water samples were taken from one waterway, less than 1 m downstream from where bait pellets were visible in the water, and also from the island’s bore water supply. At 1 and 2 weeks after the second bait application, samples (Table 1) of paua (*Haliotis iris*) and scallops (*Pecten novaecelandiae*) were taken from within 5 and 50 m of the shoreline, respectively.

### Rangitoto and Motutapu Islands – Residues in Water, Wildlife, and Marine Shellfish

Rangitoto and Motutapu are connected islands in the inner Hauraki Gulf, approximately 8 km northeast of Auckland (see www.doc.govt.nz/parks-and-recreation/places-to-visit/auckland/auckland-area/rangitoto-island-scenic-reserve/ and www.doc.govt.nz/parks-and-recreation/places-to-visit/auckland/auckland-area/motutapu-island-recreation-reserve/). DOC undertook 3 aerial applications of Pestoff® Rodent Bait 20R on 19-20 June, 9 July, and 6 August 2009 with respective application rates of 22.1, 9.5, and 6.6 kg/ha. The initial high application rate was used to minimise the risk that uptake by rabbits would leave gaps in bait coverage intended for rodents (*Rattus rattus*, *R. norvegicus*, and *Mus musculus*). Roof water-collection systems were disconnected before aerial application, and roofs and animal drinking troughs cleared of any bait afterwards. Four samples from drinking supplies on Motutapu were taken approximately 2 months after the last aerial application. Three weeks after the last application, 10 pipi (*Paphies australis*) from Motutapu and 10 mussels (*Mytilus edulis*) from Rangitoto were sampled for residue testing (Table 1).

### Table 1. Testing laboratories, numbers analysed and detection limits for water, soil and animal tissue samples tested for residual brodifacoum following aerial bait application.

| Island Eradication | Sample Type | No. Tested | Testing Laboratory | MDL (ppm) |
|--------------------|-------------|------------|--------------------|-----------|
| Maungatautari      | Water       | 217        | LCR                | 0.000002  |
|                    | Soil        | 4          | AQ                 | 0.02      |
|                    | Shellfish   | 4*         | AQ                 | 0.001     |
|                    | Kiwi liver  | 1          | LCR                | 0.001     |
| Little Barrier     | Water       | 4          | AQ                 | 0.02      |
|                    | Soil        | 4          | AQ                 | 0.01      |
|                    | Shellfish   | 4*         | AQ                 | 0.001     |
| Rangitoto/Motutapu | Water       | 4          | LCR                | 0.000002  |
|                    | Shellfish   | 2*         | LCR                | 0.001     |
|                    | Penguin liver| 9        | LCR                | 0.001     |
|                    | Dolphin liver| 5        | AQ                 | 0.005     |
|                    | Dolphin ingesta| 5    | AQ                 | 0.005     |
|                    | Dog vomit   | 1          | AQ                 | 0.005     |
|                    | Pilchards   | 1*         | LCR                | 0.001     |

*Each sample consisted of 4 or 5 individual shellfish combined

LCR = Landcare Research Toxicology Laboratory, Lincoln, New Zealand
AQ = Agriquality National Chemical Residue Laboratory, Upper Hutt, New Zealand
MDL = method detection limit

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Over approximately 4 weeks following the baiting operation, national media and Internet coverage was given to assertions by various interest groups and individuals that marine wildlife found dead on local beaches outside the eradication operational area had been poisoned as a result of the aerial bait application. This coincided with cases of domestic dogs being poisoned on beaches in the area. While veterinary diagnoses and chemical testing later indicated that these cases were the result of ingesting sea slugs (*Pleurobranchaea maculata*) containing the neurotoxin tetrodotoxin, their occurrence soon after the Rangitoto/Motutapu brodifacoum applications further raised public concerns. To address these, necropsy and brodifacoum testing of 9 little blue penguin (*Eudyptula minor*) and 5 dolphin (*Delphinus sp.*) carcasses was undertaken. Samples of the dolphins’ stomach contents, of pilchards (*Sardilopis neopilchardus*) found dead on beaches, and a vomit sample from 1 of 5 that died were also tested for residual brodifacoum (Table 1).

**Residue Analyses**

Two accredited New Zealand laboratories analysed samples for brodifacoum, with method detection limit (MDL) values dependent on sample type (Table 1). The Landcare Research brodifacoum analyses used HPLC with fluorescence detection, with methods developed for different sample types based on those described by Hunter (1983), Booth et al. (1999), and Primus et al. (2001).

**RESULTS**

No brodifacoum was detected in 217 water samples from Maungatautari, in any of the 4 water samples tested from Little Barrier, or in the 4 drinking water samples from Motutapu. On Little Barrier Island, bait pellets in exclusion cages were nearly completely disintegrated by 100 d after bait application. Soil samples from a grassland site on Little Barrier had residues of 0.2 ppm (n = 2 with the same concentration) on day 56 and 0.03 ppm on day 153. Soil samples from a forested site had residues of 0.9 and 0.5 ppm on day 56 and 0.07 ppm on day 153. Brodifacoum was not detected in any of the paua and scallop samples from Little Barrier, or in pipi or mussel samples from Motutapu and Rangitoto.

On Little Barrier Island, track searches recovered carcasses of a blackbird (*Turdus merula*) and a pukeko (*Porphyrio porphyrio*). Grid searches recovered carcasses of 2 blackbirds, 4 pukeko, 14 morepork (*Ninox novaeseelandiae*), one harrier (*Circus approximans*), 2 North Island brown kiwi (*Apteryx mantelli*), and 2 kakariki (*Cyanoramphus atriceps*). The carcasses were too degraded for necropsy or liver sampling, except for one kiwi where necropsy gave a provisional diagnosis of bronchopneumonia with residual brodifacoum concentrations in the liver of 0.26 ppm.

Following the Rangitoto/Motutapu eradication, no brodifacoum was detected in 5 dolphins or their stomach contents or in whole-body samples of pilchards collected from local beaches during July 2009. In some cases degradation of penguin carcasses precluded necropsy. Of the 7 penguins examined there were no obvious signs of anticoagulant poisoning (such as haemorrhage), and in 3 of these necropsy indicated poor condition, i.e., no body fat, empty stomach. Of the total 9 penguin livers tested, no brodifacoum was detected in 6, but in 3 there were concentrations of 0.005, 0.007, and 0.17 ppm, respectively.

**DISCUSSION**

**Brodifacoum in Water**

The water monitoring implemented at Maungatautari (217 samples tested, no brodifacoum detected) appears the most comprehensive reported to date. Brodifacoum was also not detected in water samples from Little Barrier and Motutapu, consistent with previous small-scale monitoring on Red Mercury Island (Morgan and Wright 1996) and Lady Alice Island (Ogilvie et al. 1997). Interacting factors likely to have contributed such results are brodifacoum’s overall low water-solubility (which decreases with pH; British Crop Protection Council 2000), adsorption of brodifacoum to organic particles (World Health Organization 1995), and dilution with water volume and flow rate. If aerially applied baits were to enter fresh water, only a limited amount of the brodifacoum in them would enter solution, being more likely to remain bound to bait or to other organic particles present in the water or sediment. If this was occurring, it did not create detectable concentrations of brodifacoum in water that could have been used for drinking supplies.

**Bait Degradation and Brodifacoum in Soil**

Bait degradation on Little Barrier was over a similar time frame as that described by Craddock (2003a) at Tawharanui, NZ where 96.5% pellets had completely broken down by 120 d in open grassed area, although bait degradation was slightly slower in a forested site. Thus, a universal degradation time for all situations cannot be defined, especially as rainfall (Bowen et al. 1995), among other climatic factors affecting degradation, can vary from island to island. In each instance, monitoring should be used to confirm whether uneaten bait is likely to have degraded sufficiently to no longer present a nontarget hazard. Following aerial bait (Talon 20P) application on Red Mercury Island (Morgan and Wright 1996) and Lady Alice Island (Ogilvie et al. 1997), no brodifacoum was detected in topsoil sampled at one month and over days 2 to 34, respectively. Those soil samples are presumed not to have been specifically associated with degrading bait, noting that brodifacoum is relatively immobile in soil (Eason and Wickstrom 2001). Hence, any residual soil concentrations are most likely to be localized around uneaten, degrading bait, as indicated by the Little Barrier results. The relatively low brodifacoum concentrations (<1 ppm) in these samples may have been due to the presence of disintegrated bait particles in the sample, in addition to limited movement of brodifacoum from bait into the soil. A decrease in the concentrations (from maximum 0.9 ppm to minimum 0.03 ppm over c. 100 d) suggests degradation in soil over time. Degradation rate of brodifacoum in a sandy clay loam was estimated as 22.4 weeks (US EPA 1998) but probably varies with soil type, at least. Thus, soil invertebrates near degrading bait on Little Barrier may have been exposed to low concentrations of brodifacoum.
brodifacoum concentrations for a limited period. While exposure of laboratory earthworms (*Apporectodea caliginosa*) to 500 ppm brodifacoum in soil resulted in 85% mortality after 28 days’ exposure (Booth and Fisher 2003), this soil brodifacoum concentration was 25 times higher than that of bait. It is unknown whether soil concentrations in a much lower (c. 1 ppm) range, more representative of field results, would affect soil invertebrate survival or health, and for how long sublethal residual concentrations of brodifacoum persist in soil invertebrates.

**Brodifacoum in Marine Shellfish**

Following accidental spillage of 18 tonnes of PestOff® 20R into the ocean at Kaikoura, NZ, brodifacoum residues were detectable for some weeks in marine shellfish commonly harvested for human consumption (Primus et al. 2005), raising awareness and concerns about potential human exposure. An important point of difference was that the spill comprised an extremely large quantity of bait entering the ocean at one point; in contrast to aerial application, which disperses distribution of individual pellets so that, consequently, much smaller quantities of brodifacoum may enter the ocean around island shorelines. The results reported here suggest that contamination of marine shellfish is unlikely following aerial application of brodifacoum baits for rodent eradication. That there were no detectable results in marine shellfish following the Little Barrier and Rangitoto/Motutapu eradication is consistent with previous small monitoring efforts following bait applications on New Zealand islands. Two oyster samples and 3 of 4 mussel samples from Motuhi Island in 1998 were <MDL, with one mussel sample reported as 0.02 ppm. This result was identified by the analysing laboratory (Landcare Research) as having interference on the chromatogram, so was conservatively reported as being brodifacoum. Two mussel samples from aquaculture farms near Great Barrier Island (Hauraki Gulf) were also below detectable concentrations, following a 2008 rat eradication attempt.

There is a lack of information regarding potential differences in exposure pathways between sediment and water-column-feeding shellfish species and the persistence of residual brodifacoum in shellfish. On this basis, there may yet be instances where residues could occur in marine shellfish following aerial bait application, but the evidence so far suggests that the risk to humans harvesting shellfish is relatively low. Where this is a concern for proposed eradication, stipulating a no-harvest period linked to post-application monitoring is a prudent approach to confirming that there is no potential secondary human exposure as a result of consuming shellfish.

**Brodifacoum in Nontarget Wildlife**

Brodifacoum is highly toxic to mammals and birds (Erickson and Urban 2004), consequently rodent bait presents a (primary) poisoning hazard to nontarget mammals and birds that find and eat enough of it. If exposure is not lethal, residual brodifacoum can persist (for months) in the livers of mammals (Eason et al. 2002, Fisher et al. 2003) and birds (Fisher 2009), but it is eliminated more quickly (days) from blood and other tissues (e.g., Fisher 2009). Liver residues and stomach contents containing partially digested brodifacoum bait present the highest secondary hazard for mammalian and avian species that prey on rodents or scavenge carcasses (e.g., Howald et al. 1999, Shore et al. 1999). Some terrestrial invertebrates will feed on cereal-based bait and then contain residual concentrations of brodifacoum (e.g., Booth et al. 2001, Craddock 2003b, Bowie and Ross 2006). Secondary mortality of insectivorvous New Zealand dotterels (*Charadrius obscurus aquilonius*) may have occurred through this environmental pathway (Dowding et al. 1999). Unpublished evidence of suspected secondary brodifacoum poisoning of two tuatara (*Sphenodon punctatus*) held in a zoo was the basis for implementing several mitigation measures to prevent brodifacoum exposure of tuatara held in outdoor enclosures on Little Barrier (R. Griffiths, unpubl. data).

The 27 bird carcasses found on Little Barrier were of species previously reported as nontarget mortalities in other New Zealand eradications using brodifacoum (e.g., Towns and Broome 2003), and in the absence of residue testing or necropsy data, the conservative assumption is they represent nontarget mortality. Of 10 radio-tagged little spotted kiwi (*Apteryx owenii*), one was confirmed to have died of brodifacoum poisoning following rodent eradication on Kapiti Island, with hemorrhage found at necropsy, and with liver residues of 1.2 ppm (Robertson and Colbourne 2001). Wild kiwi have occasionally been recorded eating softened or degraded cereal bait, but their main prey are soil invertebrates such as earthworms, cicada nymphs, and grass grubs (Robertson et al. 1999), so both primary and secondary exposure to brodifacoum was possible for the 2 brown kiwi found dead on Little Barrier Island. Better understanding of invertebrates as a residue vector is required to identify the most likely pathways of environmental exposure by kiwi to brodifacoum, and also to direct improved nontarget risk mitigation measures for inverteivores. Most morepork carcasses were found in areas where historical densities of kiore (*Rattus exulans*) had been highest, so presenting a possible increased risk of secondary poisoning. Since the bait application in 2004, morepork have remained abundant on Little Barrier (R. Griffiths, unpubl. data). A 2009 call-count survey of kiwi on Little Barrier detected similar average calls per hour to a 2002 survey, but estimated a lower overall kiwi population than in 2002 (Wade 2009). So, although a population-level effect of non-target mortality on kiwi and morepork on Little Barrier has not been measured, some community groups consider that any nontarget bird mortality (especially iconic native species) is unacceptable.

The presence of residual brodifacoum in livers of 3 of 9 penguins cannot be confirmed as sourced from the Rangitoto/Motutapu bait applications, as brodifacoum bait stations are commonly used for commensal rodent control in New Zealand, and also for field use against brushtail possums and rodents (see Hoare and Hare 2006). Exposure of the penguins to brodifacoum before the Rangitoto/Motutapu aerial operation cannot be ruled out because brodifacoum was almost certainly being used.
in the Hauraki Gulf area, potentially around buildings or on boats in coastal areas near terrestrial penguin habitat, before June 2009. The presence of brodifacoum in the penguins also cannot be confirmed as a direct cause or contributor to their mortality, as brodifacoum can be retained in liver at sublethal concentrations, as reported in a range of live-sampled, apparently healthy mammals and birds (see Fisher 2009). Relatively high liver concentrations (<1 ppm) are more strongly associated with lethal exposure, but there is overlap between the lowest lethal and highest sublethal concentrations reported. For example, Littin et al. (2002) measured concentrations as low as 0.33 ppm in livers of lethally poisoned possums, but sublethally exposed chickens (Gallus gallus) had liver residues of 0.45-1.00 ppm (Fisher 2009). Rather than estimating a threshold liver concentration definitive of lethal brodifacoum exposure (e.g., Kaukeinen et al. 2000), it is more valid to attribute increasing certainty of lethal exposure with increasing liver concentration – as did Myllymäki et al. (1999), who estimated that survival probability in voles (Microtus sp.) started decreasing at 0.20 ppm in liver. Necropsy observations of fresh carcasses may assist in determining the cause of death (e.g., Hosea 2000, Stone and Okoniewski 2003), and in some cases be supported by information on the circumstances of carcass recovery and expert knowledge of common causes of mortality in the species concerned.

The 0.26 ppm liver concentration in the kiwi from Little Barrier Island was in the “overlap” concentration range, i.e., low certainty, possible lethal exposure. While necropsy did not indicate hemorrhage, the recovery of the carcass in the operational area soon after bait application and previous confirmation of a kiwi mortality in similar circumstances (Robertson and Colbourne 2001) support a conservative diagnosis of brodifacoum poisoning. In all of 9 penguin carcasses found on beaches outside the operational area in the month following the Rangitoto/Motutapu operation, necropsy indicated starvation with no evidence of hemorrhage considered typical of anticoagulant poisoning. In some years, many little blue penguin carcasses are washed ashore in New Zealand, probably as the result of food shortage or biotoxins (e.g., Heather and Robertson 1996). For the 6 penguins in which no brodifacoum was detected, starvation was the most likely cause of death. In 2 of the 3 penguins with detectable liver residues, starvation was also most likely because the very low brodifacoum concentrations of 0.005 and 0.007 ppm were most representative of sublethal exposure. The penguin with 0.17 ppm liver concentration was within the “overlap” range, i.e., a low-certainty, possibly lethal exposure. Because the carcass was found outside the operational area and with no hemorrhage seen at necropsy, the known seasonal occurrence of starvation in local penguin populations was considered the more likely cause of death than brodifacoum poisoning. However, it is unknown whether brodifacoum exposure in this penguin was a contributing factor to mortality.

Importance of Monitoring

While environmental sampling and subsequent analysis adds labor and operating cost to eradication programmes, monitoring data from completed eradication programmes have undoubted value as a reference for future risk assessments. When budgeting to cover mandated monitoring, generally as stipulated by the conditions of a regulatory approval, eradication planners should retain some flexibility to at least obtain additional environmental samples that can be stored pending analysis (better to have samples that don’t need testing than to need to test and not have samples). Even if the potential for brodifacoum contamination is considered low, directly addressing concerns through analysis for residues may have greater “public relations” value than the dollar cost of a laboratory test, especially if confirmation or assurance is provided by nil-detected results from a locally relevant environment. Where brodifacoum is detected in environmental samples, this contributes to future risk assessments and mitigation approaches – the detection of residual brodifacoum in little blue penguins shows the role of monitoring in identifying new information. In this case, it has raised questions about the pathways and extent of exposure in penguins and the significance of sublethal residual concentrations for longer-term survival fitness. The Rangitoto/Motutapu bait application also attracted media attention and public concern that contributed to increased publicising of both factual and inaccurate information about brodifacoum and its effects. For managers planning eradications on inhabited islands, failure to clearly address the information gaps identified by community concerns around the aerial application of brodifacoum will mean that clear justification of eradication benefits will become increasingly difficult.

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LITERATURE CITED

ANONYMOUS. 2006. Code of practice: aerial and hand broadcast application of Pestoff® Rodent Bait 20R for the intended eradication of rodents from specified areas of New Zealand. Report prepared for the New Zealand Food Safety Authority, June 2006 revision. 27 pp.

BOOTH, L. H., C. T. EASON, and E. B. SPURR. 2001. Literature review of the acute toxicity and persistence of brodifacoum to invertebrates and studies of residue risks to wildlife and people. Sci. for Conserv. 177, Dept. of Conservation, Wellington, NZ. 23 pp.

BOOTH, L. H., and P. FISHER. 2003. Toxicity and residues of brodifacoum in snails and earthworms. DOC Sci. Internal Ser. 143, Dept. of Conservation, Wellington, NZ. 14 pp.

BOOTH, L. H., S. C. OGILVIE, and C. T. EASON. 1999. Persistence of sodium monofluoroacetate (1080), pindone, cholecalciferol, and brodifacoum in possum baits under simulated rainfall. NZ J. Agric. Res. 42:107-112.

BOWEN, L. H., D. R. MORGAN, and C. T. EASON. 1995. Persistence of sodium monofluoroacetate (1080) in baits under simulated rainfall. NZ J. Agric. Res. 38:529-531.

BOWIE M., and J. ROSS. 2006. Identification of weta (Orthoptera: Anostomatidae and Rhaphidophoridae) foraging on brodifacoum cereal bait and the risk of
secondary poisoning for bird species on Quail Island, New Zealand. NZ J. Ecol. 30:219-228.

BRITISH CROP PROTECTION COUNCIL. 2000. Brodifacoum. In: C. R. Worthing and R. J. Hance (Eds.), The Pesticide Manual, 12th Ed. British Crop Protection Council, Surrey, UK.

CRADDOCK, P. 2003a. Environmental breakdown of Pestoff poison bait (20 ppm) brodifacoum at Tawharanui Regional Park, north of Auckland. Unpublished report prepared for Northern Regional Parks, Auckland Regional Council. Entomologia Consulting, NZ. 25 pp.

CRADDOCK, P. 2003b. Aspects of the ecology of forest invertebrates and the use of brodifacoum. Ph.D. thesis, University of Auckland, NZ. 206 pp.

DOWDING, J. E., E. C. MURPHY, and C. R. VEITCH. 1999. Brodifacoum residues in target and non-target species following an aerial poisoning operation on Motuihe Island, Hauraki Gulf, New Zealand. NZ J. Ecol. 23:207-214.

EASON, C. T., E. MURPHY, G. R. WRIGHT, and E. B. SPURR. 2002. Assessment of risks of brodifacoum to non-target birds and mammals in New Zealand. Ecotoxicol. 11:35-48.

EASON, C., and M. WICKSTROM. 2001. Vertebrate pesticide toxicology manual (poisons). Dept. of Conservation Tech. Ser. 23. Department of Conservation, Wellington, NZ.

ERICKSON, W., and D. URBAN. 2004. Potential risks of nine rodenticides to birds and nontarget mammals: A comparative approach. U.S. Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances, Washington, D.C.

FISHER, P. 2009. Residual concentrations and persistence of the anticoagulant rodenticides brodifacoum and diphacinone in fauna. Ph.D. thesis, Lincoln University, Lincoln, NZ.

FISHER, P., C. O’CONNOR, G. WRIGHT, and C. EASON. 2003. Persistence of four anticoagulant rodenticides in the liver of laboratory rats. DOC Sci. Internal Ser. 139, Department of Conservation, Wellington, NZ.

HEATHER, B., and H. ROBERTSON (EDITORS). 1996. The Field Guide to the Birds of New Zealand. Penguin, Auckland, NZ. 432 pp.

HOARE, J. M., and K. M. HARE. 2006. The impact of brodifacoum on non-target wildlife: Gaps in knowledge. NZ J. Ecol. 30:157-167.

HOSEA, R. C. 2000. Exposure of non-target wildlife to anticoagulant rodenticides in California. Proc. Vertebr. Pest Conf. 19:236-244.

HOWALD, G., C. J. DONLAN, J. P. GALVAN, J. C. RUSSELL, J. PARKES, A. SAMANIEGO, Y. WANG, D. VEITCH, P. GENOVESI, M. PASCAL, A. SAUNDERS, and B. TERSHY. 2007. Invasive rodent eradication on islands. Conserv. Biol. 21:1258-1268.

HOWALD, G. R., P. MINEAU, J. E. ELLIOTT, and K. M. CHENG. 1999. Brodifacoum poisoning of avian scavengers during rat control on a seabird colony. Ecotoxicology 8:431-447.

HUNTER, K. 1983. Determination of coumarin anticoagulant rodenticide residues in animal tissue by high-performance liquid chromatography: I. Fluorescence detection using post-column techniques. J. Chromatogr. 270:267-276.

KAUKAINKEN, D. E., and M. RAMPAUD. 1986. A review of brodifacoum efficacy in the U.S. and worldwide. Proc. Vertebr. Pest Conf. 12:16-50.

KAUKAINKEN, D. E., C. W. SPRAGINS, and J. F. HOBSO. 2000. Risk-benefit considerations in evaluating commensal anticoagulant impacts to wildlife. Proc. Vertebr. Pest Conf. 19:245-266.

LITTIN, K. E., C. E. O’CONOR, N. G. GREGORY, D. J. MELLOR, and C. T. EASON. 2002. Behaviour, coagulopathy and pathology of brushtail possums (Trichosurus vulpecula) poisoned with brodifacoum. Wildl. Res. 29:259-267.

MORGAN, D. R., and G. R. WRIGHT. 1996. Environmental effects of rodent Talon baiting. Part I. Monitoring for toxic residues. Pp. 5-11 in: Sci. for Conserv. 38, Department of Conservation, Wellington, NZ.

MYLLYMÄKI, A., J. PIIHLA, and H. TUURL. 1999. Predicting the exposure and risk to predators and scavengers associated with using single-dose second-generation anticoagulants against field rodents. Pp. 387-404 in: D. P. Cowan and C. J. Feare (Eds.), Advances in Vertebrate Pest Management. Filander Verlag, Fürth, Germany.

OGLIE, S. C., R. J. PIERCE, G. R. WRIGHT, L. H. BOOTH, and C. T. EASON. 1997. Brodifacoum residue analysis in water, soil, invertebrates and birds after rat eradication on Lady Alice Island. NZ J. Ecol. 22:371-379.

PRIMUS, T., J. D. EISEMANN, G. H. MATSCHIKE, C. RAMEY, and J. J. JOHNSTON. 2001. Chlorophacinone residues in rangeland rodents: An assessment of the potential risk of secondary toxicity to scavengers. Pp. 164-180 in: J. J. Johnston (Ed.), Pesticides and Wildlife. ACS Symp. Ser. 771, American Chemical Society, Washington, D.C.

PRIMUS, T., G. WRIGHT, and P. FISHER. 2005. Accidental discharge of brodifacoum baits in a tidal marine environment: A case study. Bull. Environ. Contam. Toxicol. 74:913-919.

ROBERTSON, H. A., and R. M. COULBOURNE. 2001. Survival of little spotted kiwi exposed to the rodenticide brodifacoum. J. Wildl. Manage. 65:29-34.

ROBERTSON, H. A., R. M. COULBOURNE, P. GRAHAM, P. J. MILLER, and R. J. PIERCE. 1999. Survival of brown kiwi exposed to 1080 poison used for control of brushtail possums in Northland, New Zealand. Wildl. Res. 26:209-214.

SHORE, R. F., J. D. S. BIRKS, and P. FREESTONE. 1999. Exposure of non-target vertebrates to second-generation rodenticides in Britain, with special reference to the polecat Mustela putorius. NZ J. Ecol. 23:199-206.

STONE, W. B., and J. C. OKONIEWSKI. 2003. Anticoagulant rodenticides and raptors: Recent findings from New York, 1998-2001. Bull. Environ. Contam. Toxicol. 70:34-40.

TOWNS, D. R., and K. G. BROOME. 2003. From small Maria to massive Campbell: Forty years of rat eradications from New Zealand islands. NZ J. Zool. 30:377-398.

US EPA (UNITED STATES ENVIRONMENTAL PROTECTION AGENCY). 1998. Reregistration Eligibility Decision (RED) Rodenticide Cluster. Prevention, Pesticides and Toxic Substances (7508W). EPA738(R(98(007, July 1998.

WADE, L. 2009. Hauturu (Little Barrier Island) kiwi survey July 2009. Unpublished report. Prepared for the Little Barrier Island Supporters Trust. 8 pp.

WORLD HEALTH ORGANIZATION. 1995. Anticoagulant rodenticides. P. 121 in: Environmental Health Criteria 175, WHO, Geneva, Switzerland.