Anticoagulant rodenticide brodifacoum detected in dead nestlings of an insectivorous passerine

Bryce M. Masuda[1], Penny Fisher[2] and Ian G. Jamieson[1]*

[1]Department of Zoology, University of Otago, PO Box 56, Dunedin 9054, New Zealand
[2]Landcare Research, PO Box 69040, Lincoln 7640, New Zealand
*Author for correspondence (Email: ian.jamieson@otago.ac.nz)

Published online: 7 November 2013

Abstract: The anticoagulant rodenticide brodifacoum is widely used to eradicate invasive rats from islands for the protection and restoration of populations of native species. However, brodifacoum is also highly toxic to birds. We report the first apparent case of secondary brodifacoum exposure and subsequent poisoning in nestlings of an insectivorous passerine, the Stewart Island robin (*Petroica australis rakiura*). Thirteen dead nestlings were collected 3–4 months after brodifacoum bait was applied to eradicate rats from Ulva Island, New Zealand. Twelve of these composite nestling samples contained moderate concentrations of brodifacoum (mean = 0.08 ± 0.02 μg g^{-1}; range = 0.011–0.28 μg g^{-1}) at levels comparable with those associated with mortality in adult birds of other species (0.2 μg g^{-1} in liver), which suggests exposure in the robin nestlings was lethal. However, we were unable to determine the definitive cause(s) of death because sampling of dead nestlings was opportunistic and we were unable to test live nestlings for residue as a comparison. The period between brodifacoum application and mortality in the nestlings (52–92 days) implicates secondary poisoning. Our results highlight the potential role of invertebrates as vectors of anticoagulant rodenticides in the environment, as well as the need for further research on this exposure pathway.

Keywords: *Petroica australis rakiura*; rat eradication; secondary poisoning; Stewart Island robin

Introduction

The second-generation anticoagulant rodenticide brodifacoum is used extensively worldwide for the control of commensal rodents in and around buildings, and is available over the counter (e.g. Talon™) for such uses in New Zealand and Australia (Clapperton et al. 2011; Gunja et al. 2011). Brodifacoum has also emerged as the rodenticide of choice for the eradication of invasive rodents (*Rattus* spp. and *Mus musculus*) from islands and fenced sanctuaries, towards biodiversity conservation and ecosystem restoration in countries such as New Zealand (e.g. Veitch 2002; McClelland 2011), Australia (e.g. Burbidge 2004; Priddel et al. 2011) and the United States (e.g. Garcia et al. 2002; Howald et al. 2005; Buckelew et al. 2011). Nearly all rat eradication attempts on islands using brodifacoum have been successful because of its high toxicity and persistence, delayed onset of poisoning, and in many cases the aerial broadcast application of bait (Parkes et al. 2011).

These characteristics also increase the risk of poisoning to non-target species, via consumption of toxic bait or primary exposure (e.g. Taylor & Thomas 1993; Wedding et al. 2010), or via consumption of contaminated prey or secondary exposure (e.g. Brown et al. 1998; Fournier-Chambrillon et al. 2004; Riley et al. 2007). Investigations of non-target secondary poisoning have typically focused on bird species that either prey on rodents (e.g. Mendenhall & Pank 1980; Hegdal & Colvin 1988; Stephenson et al. 1999) or scavenge carcasses (e.g. Howald et al. 1999; Henny & Elliott 2007; Redig & Arent 2008). Insectivorous birds are also at risk of secondary poisoning if they consume invertebrates that feed on bait, but there have been relatively few studies of this non-target exposure pathway (Booth et al. 2001).

In contrast to the many studies that document adult mortality in non-target wildlife following a brodifacoum application, few investigations have examined the effect of brodifacoum exposure on the reproduction of birds, particularly the risk to nestlings. Exposure of nestlings to anticoagulant rodenticides can occur if they are fed toxic bait directly. For example, nestling South Island robins (*Petroica australis*) were fed Racumin® paste (which contains the anticoagulant rodenticide coumatetralyl) by their parents (Pryde et al. 2013). Nestlings can also be secondarily exposed if they are fed contaminated prey items, such as rodenticide-killed mice that were fed to turkey vulture (*Cathartes aura*) nestlings by their parents (Borst & Counotte 2002). Insectivorous nestlings could be at risk of poisoning if they are fed contaminated invertebrates, which have been detected up to 10 weeks after the application of brodifacoum (Craddock 2003). Nestlings could also be at a greater risk than adults because of their smaller size, where less toxin is required for a lethal exposure.

Ulva Island (267 ha) is an open sanctuary in Paterson Inlet, Stewart Island, New Zealand. Norway rats (*Rattus norvegicus*) were initially eradicated from Ulva Island in 1996, because their presence resulted in the local extinction of native species such as Stewart Island robins (*Petroica a. rakiura*). The initial rat eradication was followed by the reintroduction of Stewart Island robins in 2000. Robins are insectivorous ground-feeding passerines endemic to New Zealand and are classified as 'Nationally Vulnerable' (Miskelly et al. 2008). Robins exhibit biparental care, with both males and females feeding offspring, and have a modal clutch size of two (Heather & Robertson 2005). Since being reintroduced in 2000, robins have established territories across Ulva Island, and the entire population has been intensively monitored since the release as part of a long-term study on inbreeding in reintroduced populations (Jamieson 2011; Laws & Jamieson 2011; Grueber et al. 2012).

Since the initial eradication in 1996, an average of one rat capture per year has indicated regular incursions onto Ulva Island, but rats failed to establish a breeding population until December 2010 (Masuda & Jamieson 2013). In an effort to again eradicate rats from Ulva to protect and restore native species, aerial applications of brodifacoum were conducted by the New Zealand Department of Conservation on 18 August and 20 September 2011 across the entire island. Brodifacoum was applied in the form of cereal bait at a concentration of 20 ppm, and the total sowing density across the entire island was 14 kg ha^{-1} (see Masuda & Jamieson 2013).

The application of brodifacoum on Ulva Island was followed by mortality of Stewart Island robins; adult robins were observed feeding on toxic bait pellets and 136 of the entire population of 473 (31.5%) were presumed to have died within one month of bait application because they were not observed thereafter (Masuda & Jamieson 2013). The reproduction of robins could also have been negatively affected by the application of brodifacoum if nestlings were fed bait directly, or if nestlings were fed contaminated invertebrates. Robins primarily feed on invertebrates, particularly large prey such as earthworms (Family Megascolecidae), tree weta (*Hemideina* spp.) and cicadas (Family Cicadidae) (Powlesland 2013), and adults feed these prey species to their nestlings as well (BMM pers. obs.).

In this paper, we report the concentrations of residual brodifacoum detected in the bodies of robin nestlings found dead 3–4 months after the aerial application of brodifacoum bait, along with observational evidence suggesting that mortality was associated with secondary exposure to brodifacoum.

Materials and methods

As part of the long-term monitoring of robins on Ulva Island, fieldworkers visited each robin pair (September–January) once every 7–10 days to determine their breeding status (Laws et al. 2010). During the 2011 breeding season, approximately 3–4 months after the first of two aerial applications of brodifacoum bait (i.e. between 11 November and 21 December 2011), we opportunistically collected a total of 13 dead robin nestlings from seven nests. In six of the seven nests, both nestlings were collected and tested for residue; one nest contained only a single nestling. Based on body size, weight, estimated laying date, and condition of the carcass, all nestlings were estimated to have died at between 7 and 17 days of age. Nestling carcasses from additional failed nests were too decomposed (i.e. found more than 2–3 days after death) for residue testing. We also did not collect live nestlings from other nests as a comparison because this would have conflicted with survival outcomes produced from the long-term study, as obtaining liver tissue to test for brodifacoum residue would have required lethal sampling. As a result, we were unable to compare brodifacoum residue concentrations present in nestlings that died and those that survived (see Discussion). All nestling carcasses were frozen within 8 h of being collected. A basic necropsy was conducted on each nestling, where the whole carcasses were checked for visible signs of external damage or injury, and then opened and checked for visible signs of damage to tissue and organs, lesions, or internal bleeding. The presence and appearance of any stomach and gut contents were described.

Tissues from each nestling carcass were taken for chemical analysis of brodifacoum concentration. In vertebrates, the liver is typically analysed to determine brodifacoum exposure, but the livers from individual robin nestlings were too small (< 0.5 g) to be analysed separately. To obtain an adequate quantity of tissue from each nestling, internal organs, including the gastrointestinal tract and its contents, were removed and homogenised to make a composite tissue sample for each individual nestling. These samples were extracted and analysed by the Landcare Research Toxicology Laboratory (Lincoln, New Zealand) for brodifacoum concentration using high performance liquid chromatography and fluorescence detection as described by Primus et al. (2005). The detection limit of this method is 0.001 μg g^{-1}, and the uncertainty (95% CI) was ±6%.

Results

There was no evidence of gross internal haemorrhage at necropsy. Evidence of haemorrhaging could be expected because anticoagulants block the reduction of vitamins K_1, K_2, and K_3 into biologically active vitamin K (Silverman 1980), which ultimately results in an increase in blood clotting time and uncontrolled haemorrhaging (Suttie 1985; Littin et al. 2000). However, in birds, the absence of haemorrhaging does not exclude the possibility of anticoagulant poisoning (see Discussion).

All nestlings had full gizzards and at least some digesta present in other sections of the gastrointestinal tract, in some cases (5/13) with faeces formed in the cloaca. Digesta was coloured black to dark brown and of a paste-like consistency containing distinct (2–3 mm) soft particles of unidentified food. We did not observe any green colouration in the digesta that may have indicated ingestion of bait pellets.

Brodifacoum was detected in 12 of 13 nestlings tested (mean = 0.08 ± 0.02 μg g^{-1}; range = 0.011–0.28 μg g^{-1}; Fig. 1), from six of seven nests. Brodifacoum was detected in all dead nestlings that were collected with a nestmate (n = 12); the only nestling in which brodifacoum was not detected did not have a nestmate. In general, higher brodifacoum concentrations were present in nestlings that died earlier in the season (i.e. sooner after the baiting operation), although the strength of relationship is partly influenced by one outlier (Fig. 1).

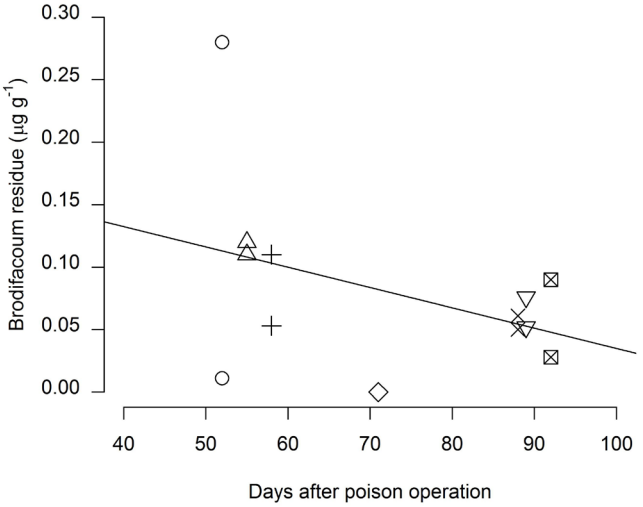

Figure 1. Brodifacoum concentration in 13 Stewart Island robin (*Petroica australis rakiura*) nestlings found dead on Ulva Island following a poison operation to eradicate Norway rats (*Rattus norvegicus*). Nestmates are indicated by paired symbols on the same day. Brodifacoum was not detected in one singleton nestling found dead on Day 71.

Discussion

Did the nestlings die from brodifacoum poisoning?

During necropsy of the robin nestlings, we did not observe internal haemorrhaging that is considered a typical sign of anticoagulant poisoning in adult birds (Rattner et al. 2011). However, anticoagulant poisoning can also occur without any apparent haemorrhage and histopathology is not necessarily diagnostic of anticoagulant poisoning (DuVall et al. 1989). Hence the absence of readily visible internal haemorrhaging in nestlings that contained residual brodifacoum does not preclude the possibility of brodifacoum poisoning per se.

The concentrations detected in composite tissue samples from robin nestlings (mean = 0.08 ± 0.02 μg g^{-1}) suggest that the robins may have died due to brodifacoum poisoning. Liver is the typical indicator tissue for monitoring exposure of wildlife to anticoagulants (e.g. Fisher et al. 2010), and previous studies that tested livers of adult birds for brodifacoum suggest that concentrations greater than 0.2 μg g^{-1} are associated with an increased risk of mortality (Fisher et al. 2011). Our results are not directly comparable with these previous studies because we tested composite tissue samples of nestlings, not liver samples of adults. As a result, we could not determine if the brodifacoum detected was in the liver, gastrointestinal tract and contents, other organs, or some combination of organs. If brodifacoum was present in the liver but not in other tissues of the nestlings, then our reported concentrations are likely to underestimate those in liver alone. The highest concentration of the anticoagulant rodenticide diphacinone detected among rat organs was in the liver, while slightly lower concentrations were detected in the lung and kidney (Yu et al. 1982). Furthermore, lower residue levels of the anticoagulant rodenticide coumatetratyl were detected in the entire gastrointestinal tract (i.e. stomach, intestines and contents) than in the rest of the body combined, although substantial variability was observed between gastrointestinal samples (O'Connor et al. 2003).

It is also possible that nestlings are more sensitive than adults (weight for weight) to the metabolic effects of brodifacoum, and thus the smaller nestlings would require relatively less brodifacoum exposure than adults to cause adverse effects and death. Although we have found no previous reported studies where residual brodifacoum has been linked to non-target mortality in avian nestlings, nestlings have been shown to be more susceptible than adults to other toxins (Cutkomp 1943; Grue & Shipley 1984). Considering the potential underestimation of brodifacoum in livers of nestlings, as well as the possible greater sensitivity, the mean concentration of brodifacoum measured in the internal organs of robin nestlings (0.08 μg g^{-1}) may well represent a lethal exposure. However, we acknowledge the limitations of this study, in that sampling of dead nestlings was opportunistic and their subsequent testing for brodifacoum was investigative. Furthermore, we were unable to test nestlings that had survived as a comparison due to the ongoing long-term study. Thus, we were unable to robustly determine a relationship between mortality and residue concentrations in our composite samples, as well as the extent (if any) of haemorrhaging. As a result, brodifacoum poisoning was implicated in the deaths of the nestlings, but we were unable to determine the definitive cause(s).

Brodifacoum poisoning has been documented in nestlings of captive, predatory turkey vultures following the consumption of contaminated mice (Borst & Counotte 2002). However, our results could be the first reported mortality in nestlings of a wild avian insectivore caused by secondary exposure to brodifacoum. Direct consumption of bait containing another anticoagulant rodenticide (coumatetralyl) appeared to result in the mortality of four South Island robin (*Petroica australis*) nestlings (Pryde et al. 2013).

How were the nestlings exposed to brodifacoum?

We did not monitor active nests to determine if nestlings were being fed poison bait directly, nor did we test invertebrate prey for brodifacoum residue. Therefore, we were unable to determine the source of brodifacoum exposure (poison bait or invertebrates) in the nestlings that died. Nevertheless, we infer secondary poisoning via invertebrates fed to nestlings, on the basis of the following evidence. First, any adults that learned to eat brodifacoum cereal pellet baits (applied August–September 2011) likely died of poisoning well before the start of the breeding season (October), when they may have fed it to nestlings. Second, by the time the dead nestlings had been found, there was little sign of bait remaining on the ground (BMM pers. obs.). Bait degradation has been monitored following aerial applications for rodent eradications in New Zealand. On Little Barrier Island, at four sites representing grassland and forested/canopy habitats, 20 bait pellets were placed under wire cages designed to exclude rodents and birds, and checked for degradation at intervals after application. Bait pellets were nearly completely disintegrated by 100 days after application (Fisher et al. 2011). Also, Craddock (2003) found that most Pestoff® bait pellets took around 80 days to completely degrade, and all pellets were degraded by 110 days during a study of bait degradation at Tawharanui Regional Park on the Mahurangi coast north of Auckland. Third, if parents had fed bait directly to offspring, we would have expected to see evidence of the green dye used in the baits, either in nestling faeces or gut contents, with potentially higher concentrations of brodifacoum in the dead nestlings.

Consuming contaminated invertebrates is a viable pathway for insectivorous birds and their offspring to be exposed to brodifacoum. Many invertebrate species feed on brodifacoum bait directly, or could ingest brodifacoum via the consumption of rodent faeces or carcasses (Spurr & Drew 1999; Craddock 2003; Hoare & Hare 2006). Brodifacoum levels in weta (*Hemideina* spp., *Gymnoplectron* spp.), which are a prey species for robins, have reached up to 7.47 μg g^{-1} immediately after a poison application (Craddock 2003). Furthermore, weta may act as vectors of brodifacoum residue for a relatively long period. Residue levels in weta were considered relatively high (0.12 μg g^{-1}) 4 weeks after bait-station applications of brodifacoum (Craddock 2003). Furthermore, brodifacoum has been shown to persist in weta for up to 4 days following consumption in one study (Booth et al. 2001) and for less than 12 days in a second study (Lloyd 1997, cited in McClelland 2002). Because invertebrates appear less sensitive than mammals to anticoagulant poisoning (Pain et al. 2000; Hoare & Hare 2006), we suggest that live invertebrates can act as spatial and temporal vectors of residual brodifacoum within a food web.

Secondary brodifacoum poisoning of insectivorous adult birds has been reported, but in far fewer cases compared with poisoning of avian predators and scavengers from ingesting dead rodents (Hoare & Hare 2006; Henny & Elliott 2007). On two occasions, it appeared that insectivorous birds died in zoo aviaries after feeding on ants and cockroaches that had eaten brodifacoum baits (Godfrey 1985; Borst & Counotte 2002). Secondary exposure to brodifacoum also appeared to cause

mortality of adult New Zealand dotterels (*Charadrius obscurus aquilonius*) that consumed sandhoppers (*Talorchestia* spp.) that had fed on bait pellets (Dowding et al. 2006). Individuals of other insectivorous bird species have been found dead after brodifacoum baiting operations, but these were believed to have died from primary poisoning (but see Robertson et al. 1999; Robertson & Colbourne 2001; Eason et al. 2002).

Recent reviews of the literature concerning the non-target effects of brodifacoum have suggested that most mortalities of insectivorous or omnivorous birds are through primary rather than secondary poisoning (Eason et al. 2002; Hoare & Hare 2006; Sánchez-Barbudo et al. 2012). However, the few studies that have explored the role of invertebrates as vectors of anticoagulant rodenticides, and our inference that the robin nestlings on Ulva Island died from secondary brodifacoum poisoning, raise the possibility that secondary exposure of insectivorous birds is more widespread than previously noted and has the potential to result in mortality. Therefore, the application of brodifacoum during or just prior to the breeding season may lead to an elevated risk of secondary poisoning in insectivorous birds, and baiting operations should be timed accordingly.

Although secondary poisoning appeared to result in the failure of at least six robin nests on Ulva Island, these failures did not appear to have any significant impact on robin productivity at a population scale. Nest survival during the nestling stage was similar before (during the 2009 and 2010 seasons) and after the baiting operation (during the 2011 season) (BMM & IGJ unpubl. data). Furthermore, had the Norway rat population not been eradicated, the robin population was unlikely to have been viable in the long term (see Masuda & Jamieson 2013). Nevertheless, conservation managers should be aware of the potential role of invertebrates as vectors of anticoagulant rodenticides to insectivorous species, particularly when assessing and mitigating non-target risk to brodifacoum exposure.

Acknowledgements

We thank the Ulva Island Trust, and B. Beaven and P. Dobbins from the Department of Conservation for logistical support during the study. E. Murphy and A. Fairweather provided helpful feedback on an earlier draft. Our long-term research project on Ulva Island, including specific aspects of this study, was conducted with approval from the University of Otago Animal Ethics Committee (79/08), Department of Conservation (SO-23570-FAU), and Ōraka Aparima Rūnaka's Kaitiaki Rōpū, and funded by the Marsden Fund, Landcare Research, Allan Wilson Centre, Department of Conservation, and University of Otago.

References

Booth LH, Eason CT, Spurr EB 2001. Literature review of the acute toxicity and persistence of brodifacoum to invertebrates. Science for Conservation 177A. Wellington, Department of Conservation.

Borst GHA, Counotte GHM 2002. Shortfalls using second-generation anticoagulant rodenticides. Journal of Zoo and Wildlife Medicine 33: 85.

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

Buckelew S, Byrd V, Howald G, MacLean S, Sheppard J 2011. Preliminary ecosystem response following invasive Norway rat eradication on Rat Island, Aleutian Islands, Alaska. In: Veitch CR, Clout MN, Towns DR eds Island invasives: eradication and management. Gland, Switzerland, IUCN. Pp. 275–279.

Burbidge AA 2004. *Montebello Renewal: Western Shield* review—February 2003. Conservation Science West Australia 5: 194–201.

Clapperton K, Maddigan F, Gillies C, Murphy E 2011. Diet of predators in *Nothofagus* forest, Nelson Lakes National Park. DOC Research & Development Series 328. Wellington, Department of Conservation. 12 p.

Craddock P 2003. Aspects of the ecology of forest invertebrates and the use of brodifacoum. Unpublished PhD thesis, University of Auckland, Auckland, New Zealand. 235 p.

Cutkomp LK 1943. Toxicity of rotenone and derris extract administered orally to birds. Journal of Pharmacology and Experimental Therapeutics 77: 238–246.

Dowding JE, Lovegrove TG, Ritchie J, Kast SN, Puckett M 2006. Mortality of northern New Zealand dotterels (*Charadrius obscurus aquilonius*) following an aerial poisoning operation. Notornis 53: 235–239.

DuVall MD, Murphy MJ, Ray AC, Reagor JC 1989. Case studies on second-generation anticoagulant rodenticide toxicities in nontarget species. Journal of Veterinary Diagnostic Investigation 1: 66–68.

Eason CT, Murphy EC, Wright GRG, Spurr EB 2002. Assessment of risks of brodifacoum to non-target birds and mammals in New Zealand. Ecotoxicology 11: 35–48.

Fisher P, Shlosberg A, Brown L, Wright G 2010. Anticoagulant residues in non-target wildlife – assessing sublethal exposure to brodifacoum without lethal sampling. In: Timm RM, Fagerstone KA eds Proceedings of the 24th Vertebrate Pest Conference. Sacramento, CA, University of California, Davis. Pp. 167–171.

Fisher P, Griffiths R, Speedy C, Broome K 2011. Environmental monitoring for brodifacoum residues after aerial application of baits for rodent eradication. In: Veitch CR, Clout MN, Towns DR eds Island invasives: eradication and management. Gland, Switzerland, IUCN. Pp. 300–304.

Fournier-Chambrillon C, Berny PJ, Coiffier O, Barbedienne P, Dassé B, Delas G, Galineau H, Mazet A, Pouzenc P, Rosoux R, Fournier P 2004. Evidence of secondary poisoning of free-ranging riparian mustelids by anticoagulant rodenticides in France: implications for conservation of European mink (*Mustela lutreola*). Journal of Wildlife Diseases 40: 688–695.

Garcia MA, Diez CE, Alvarez AO 2002. The eradication of *Rattus rattus* from Monito Island, West Indies. In: Veitch CR, Clout MN eds Turning the tide: the eradication of invasive species. Gland, Switzerland and Cambridge, UK, IUCN SSC Invasive Species Specialist Group. Pp. 116–119.

Godfrey MER 1985. Non-target and secondary poisoning hazards of "second generation" anticoagulants. Acta Zoologica Fennica 173: 209–212.

Grue CE, Shipley BK 1984. Sensitivity of nestling and adult starlings to dicrotophos, an organophosphate pesticide. Environmental Research 35: 454–465.

Grueber CE, Wallis GP, King TM, Jamieson IG 2012. Variation at innate immunity toll-like receptor genes in a bottlenecked

population of a New Zealand robin. PLoS ONE 7(9): e45011. doi:10.1371/journal.pone.0045011.
Gunja N, Coggins A, Bidny S 2011. Management of intentional superwarfarin poisoning with long-term vitamin K and brodifacoum levels. Clinical Toxicology 49: 385–390.
Heather BD, Robertson HA 2005. The field guide to the birds of New Zealand. Auckland, Viking. 440 p.
Hegdal PL, Colvin BA 1988. Potential hazard to eastern screech-owls and other raptors of brodifacoum bait used for vole control in orchards. Environmental Toxicology and Chemistry 7: 245–260.
Henny CJ, Elliott JE 2007. Toxicology. In: Bird DM, Bildstein KL eds Raptor research and management techniques manual. 2nd edn. Blaine, WA, Hancock House. Pp. 329–350.
Hoare JM, Hare KM 2006. The impact of brodifacoum on non-target wildlife: gaps in knowledge. New Zealand Journal of Ecology 30: 157–167.
Howald GR, Mineau P, Elliott JE, Cheng KM 1999. Brodifacoum poisoning of avian scavengers during rat control on a seabird colony. Ecotoxicology 8: 431–447.
Howald GR, Faulkner KR, Tershy B, Keitt B, Gellerman H, Creel EM, Grinnell M, Ortega ST, Croll DA 2005. Eradication of black rats from Anacapa Island: biological and social considerations. In: Garcelon DK, Schwemm eds Proceedings of the Sixth California Islands Symposium. National Park Service Technical Publication CHIS-05-01. Arcata, CA, Institute for Wildlife Studies. Pp. 299–312.
Jamieson IG 2011. Founder effects, inbreeding, and loss of genetic diversity in four avian reintroduction programs. Conservation Biology 25: 115–123.
Laws RJ, Jamieson IG 2011. Is lack of evidence of inbreeding depression in a threatened New Zealand robin indicative of reduced genetic load? Animal Conservation 14: 47–55.
Laws RJ, Townsend SM, Nakagawa S, Jamieson IG 2010. Limited inbreeding depression in a bottlenecked population is age but not environment dependent. Journal of Avian Biology 41: 645–652.
Littin KE, O'Connor CE, Eason CT 2000. Comparative effects of brodifacoum on rats and possums. New Zealand Plant Protection 53: 310–315.
Masuda BM, Jamieson IG 2013. Response of a reintroduced bird population to a rat reinvasion and eradication. New Zealand Journal of Ecology 37: 224–231.
McClelland PJ 2002. Eradication of Pacific rats (*Rattus exulans*) from Whenua Hou Nature Reserve (Codfish Island), Putauhinu and Rarotoka Islands, New Zealand. Turning the tide: the eradication of invasive species. Gland, Switzerland and Cambridge, UK, IUCN SSC Invasive Species Specialist Group. Pp. 173–181.
McClelland PJ 2011. Campbell Island – pushing the boundaries of rat eradications. In: Veitch CR, Clout MN, Towns DR eds Island invasives: eradication and management. Gland, Switzerland, IUCN. Pp. 204–207.
Mendenhall VM, Pank LF 1980. Secondary poisoning of owls by anticoagulant rodenticides. Wildlife Society Bulletin 8: 311–315.
Miskelly CM, Dowding JE, Elliot GP, Hitchmough RA, Powlesland RG, Robertson HA, Sagar PM, Scofield RP, Taylor GA 2008. Conservation status of New Zealand birds, 2008. Notornis 55: 117–135.
O'Connor CE, Eason CT, Endepols S 2003. Evaluation of secondary poisoning hazards to ferrets and weka from the rodenticide coumatetralyl. Wildlife Research 30: 143–146.
Pain DJ, Brooke MdL, Finnie JK, Jackson A 2000. Effects of brodifacoum on the land crab of Ascension Island. The Journal of Wildlife Management 64: 380–387.
Parkes J, Fisher P, Forrester G 2011. Diagnosing the cause of failure to eradicate introduced rodents on islands: brodifacoum versus diphacinone and method of bait delivery. Conservation Evidence 8: 100–106.
Powlesland RG 2013. South Island robin. In: Miskelly CM ed. New Zealand Birds Online. www.nzbirdsonline.org.nz
Priddel D, Carlile N, Wilkinson I, Wheeler R 2011. Eradication of exotic mammals from offshore islands in New South Wales, Australia. In: Veitch CR, Clout MN, Towns DR eds Island invasives: eradication and management. Gland, Switzerland, IUCN. Pp. 337–344.
Primus T, Wright G, Fisher P 2005. Accidental discharge of brodifacoum baits in a tidal marine environment: a case study. Bulletin of Environmental Contamination and Toxicology 74: 913–919.
Pryde MA, Pickerell G, Coats G, Hill GS, Greene TC, Murphy EC 2013. Observations of South Island Robins eating Racumin®, a toxic paste used for rodent control. New Zealand Journal of Zoology 40: 255–259.
Rattner BA, Horak KE, Warner SE, Day DD, Meteyer CU, Volker SF, Eisemann JD, Johnston JJ 2011. Acute toxicity, histopathology, and coagulopathy in American kestrels (*Falco sparverius*) following administration of the rodenticide diphacinone. Environmental Toxicology and Chemistry 30: 1213–1222.
Redig PT, Arent LR 2008. Raptor toxicology. Veterinary Clinics of North America: Exotic Animal Practice 11: 261–282.
Riley SPD, Bromley C, Poppenga RH, Uzal FA, Whited L, Sauvajot RM 2007. Anticoagulant exposure and notoedric mange in bobcats and mountain lions in urban southern California. The Journal of Wildlife Management 71: 1874–1884.
Robertson HA, Colbourne RM 2001. Survival of little spotted kiwi exposed to the rodenticide brodifacoum. The Journal of Wildlife Management 65: 29–34.
Robertson HA, Colbourne RM, Graham PJ, Miller PJ, Pierce RJ 1999. Survival of brown kiwi (*Apteryx mantelli*) exposed to brodifacoum poison in Northland, New Zealand. New Zealand Journal of Ecology 23: 225–231.
Sánchez-Barbudo IS, Camarero PR, Mateo R 2012. Primary and secondary poisoning by anticoagulant rodenticides of non-target animals in Spain. Science of The Total Environment 420: 280–288.
Silverman RB 1980. A model for a molecular mechanism of anticoagulant activity of 3-substituted 4-hydroxycoumarins. Journal of the American Chemical Society 102: 5421–5423.
Spurr EB, Drew KW 1999. Invertebrates feeding on baits used for vertebrate pest control in New Zealand. New Zealand Journal of Ecology 23: 167–173.
Stephenson BH, Minot EO, Armstrong DP 1999. Fate of moreporks (*Ninox novaeseelandiae*) during a pest control operation on Mokoia Island, Lake Rotorua, North Island, New Zealand. New Zealand Journal of Ecology 23: 233–240.
Suttie JW 1985. Vitamin K-dependent carboxylase. Annual Review of Biochemistry 54: 459–477.
Taylor RH, Thomas BW 1993. Rats eradicated from rugged Breaksea Island (170 ha), Fiordland, New Zealand. Biological Conservation 65: 191–198.
Veitch CR 2002. Eradication of Pacific rats (*Rattus exulans*) from Fanal Island, New Zealand. In: Veitch CR, Clout

MN eds Turning the tide: the eradication of invasive species. Gland, Switzerland and Cambridge, UK, IUCN SSC Invasive Species Specialist Group. Pp. 357–359.

Wedding CJ, Brunton DH, Ji W 2010. Implications of visitations by shore skinks '*Oligosoma smithi*' to bait stations containing brodifacoum in a dune system in New Zealand. Pacific Conservation Biology 16: 86–91.

Yu CC, Atallah YH, Whitacre DM 1982. Metabolism and disposition of diphacinone in rats and mice. Drug Metabolism and Disposition 10: 645–648.

Editorial Board member: Craig Barnett
Received 18 March 2013; accepted 1 September 2013