Laboratory and field studies of brodifacoum residues in relation to risk of exposure to wildlife and people

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ABSTRACT

The field use of brodifacoum baits (Talon® and PESTOFF®) to control brushtail possums (*Trichosurus vulpecula*) in New Zealand raises concerns of secondary and tertiary poisoning, resulting from the transfer of this toxicant through the food chain. In New Zealand, feral pigs (*Sus scrofa*) scavenge possum carcasses and may also access bait stations containing possum baits. We have determined the concentrations of brodifacoum in serum, muscle and liver tissue from captive pigs after both primary and secondary poisoning. Highest concentrations were found in the liver. Pigs eating 500–1776 g of bait containing 20 mg/kg brodifacoum had muscle concentrations of 0.02–0.07 mg/kg and liver concentrations of 0.72–1.38 mg/kg. Both muscle and liver concentrations appeared to be independent of the amount of bait eaten. Possums fed 400 g of bait had similar liver concentrations (0.52–1.20 mg/kg). Pigs that had eaten the soft tissue from eight poisoned possums had brodifacoum concentrations of 0.32–0.80 mg/kg in the liver, which increased in a dose-dependent manner. Brodifacoum was detected in muscle from only one of these animals. In a field survey where brodifacoum baits were currently being used to control possums and rats, 29 of 37 wild pigs, 2 of 23 goats, and 14 of 36 red deer sampled had brodifacoum concentrations in the liver ranging from 0.01 to 1.9 mg/kg. In areas where brodifacoum baiting had ceased at least 6 months previously, 12 of 19 pigs and none of the 6 goats and 15 deer sampled had residues. In view of the potential exposure of pig hunters consuming wild pig meat and offal, a precautionary approach, even in the absence of evidence of human exposure, has been recommended, i.e. some restrictions on the sustained field use of brodifacoum baits on mainland New Zealand and the Chatham Islands.

Keywords: Anticoagulant, baits, non-target species, pigs, possums.

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1. Introduction

The concentration of brodifacoum in pig serum and tissue (liver and muscle) after the primary and secondary poisoning of captive pigs, and the level of brodifacoum contamination in samples taken from feral pigs, goats and deer were assessed by Landcare Research, Lincoln, for the Department of Conservation (DOC), Science and Research Division from June 1997 to June 2000. The theoretical risks of secondary poisoning to humans (and pets) associated with brodifacoum use in New Zealand are discussed in light of the results from these residue analyses.

2. Background

Brodifacoum is a potent, second-generation, anticoagulant rodenticide developed in the mid-1970s. It is extremely persistent in target and non-target species (Eason et al. 1996). Brodifacoum has been used with success in recent rodent eradication programmes on New Zealand's offshore islands (Buckle & Fenn 1992; Robertson et al. 1993; Taylor & Thomas 1993; Towns et al. 1993). In addition to its worldwide use to control and eradicate rats, brodifacoum has also been used to eradicate rabbits (*Oryctolagus cuniculus*) from islands (Merton 1987; Towns et al. 1993). It is now commonly used in New Zealand to control brushtail possums (*Trichosurus vulpecula*) (Eason et al. 1993), where it has found favour for field use since it can control rats and possums simultaneously.

Brodifacoum differs markedly in its mode of action when compared with fast-acting toxins such as sodium monofluoroacetate (1080) or cyanide, since possums may take 3 or more weeks to die after eating a lethal dose of brodifacoum bait (Eason et al. 1994). The reported LD_{50} for brodifacoum in possums is 0.17 mg/kg (equivalent to 20 g of bait containing 20 mg/kg for a possum weighing 2–3 kg) (Godfrey 1985). However, because of the slow onset of action of brodifacoum, some possums will eat more than 1 kg of bait before dying, which can make the use of brodifacoum expensive compared with 1080 (Eason et al. 1994; Henderson et al. 1994). Nevertheless, this toxicant can be cost-effective when used after 1080, cyanide, or trapping to maintain possum numbers at very low levels.

During the mid-1990s there was an increasing use of cereal bait containing 20 mg/kg brodifacoum (Talon® and PESTOFF®) for killing possums. The difficulties with using 1080, including the increasing public concerns and the legal consents process and notifications required before using it, probably contributed to this increased user-preference for brodifacoum bait.

The toxicology of brodifacoum and the species at risk from primary and secondary poisoning in New Zealand have been reviewed elsewhere (Eason et al. 1993; Eason & Spurr 1995). The acute toxicity of brodifacoum to birds varies from an LD_{50} of < 1 mg/kg in pukeko (*Porphyrio p. melanotus*) to

> 20 mg/kg in paradise shelduck (*Tadorna variegata*). In several species of mammals, including rodents, pigs and possums, the LD_{50} is < 0.4 mg/kg. Because of its toxicity, all vertebrates that eat baits or poisoned prey containing brodifacoum residues are at risk, including humans. There are numerous documented cases of deliberate or inadvertent primary anticoagulant poisoning of humans (Barlow et al. 1982; Jones et al. 1984; Lipton & Klass 1984; Chong et al. 1986; Hoffman et al. 1988; Weitzel et al. 1990; Wallace et al. 1990; Kruse & Carlson 1992; Tasheva 1995). For example, in one fatal incident villagers in South Sumatra, Indonesia, used a 50-ppm brodifacoum rice grain bait as a food source. They attempted to remove the rodenticide by repeated washing, rinsing, and cooking before eating the rice. Because of the delay in the appearance of symptoms, it appeared to the villagers that they had been successful, thus encouraging others to eat the rice, resulting in several deaths (Tasheva 1995).

To date there are no recorded incidents of humans being poisoned after eating contaminated meat. However, a risk of secondary poisoning exists, which is compounded by the unusual persistence of this toxicant and other second-generation anticoagulants in vertebrate species compared with first-generation anticoagulants (Parmar et al. 1987). For example, brodifacoum has been shown to persist in the liver of sheep (*Ovis aries*) for 16 weeks (Laas et al. 1985) and possums for 9 months (Eason et al. 1996). However, little consideration has been given to the potential dangers to humans arising from brodifacoum residues in possum carcasses, or to other wildlife, including game species. Pigs are one of a number of species that may scavenge possum carcasses, and possums dying up to 9 months (or more) after being exposed to sub-lethal amounts of brodifacoum will contain residues, particularly in the liver, which could be transferred through the food chain (Eason et al. 1996). Also of concern is the possibility of primary poisoning of pigs and other game by their gaining access to the bait in bait stations and the subsequent secondary poisoning implications of these practices.

3. Objectives

We set out to:

- determine the concentration of brodifacoum in pig liver and muscle after primary and secondary exposure;
- survey feral game species in areas of high and low brodifacoum use and determine the extent of contamination in animals that are a probable food source for humans;
- evaluate the risk of secondary poisoning of people.

4. Methods

4.1 PRIMARY POISONING OF PIGS

Sixteen domestic pigs held in captivity were divided into four equal groups. Group 1 received non-toxic pellets; Group 2 were fed 500 g (± 31.2 g, low group), Group 3 were fed 937 g (± 34.2 g, medium group), and Group 4 were fed 1776 g (± 81.4 g, high group) of brodifacoum bait. All groups were fed the pellets over a 2-day period. Pigs in the medium- and high-dose groups received between 5 and 200 g of fortified (i.e. 100 mg/kg) brodifacoum pellets on the second day to elevate total intake to the required levels and minimise within-group variation in the amount of brodifacoum ingested. The rationale for the dosages used is as follows. The published LD_{50} values for brodifacoum in pigs vary from 0.1 mg/kg (Godfrey 1985) to 0.5–2.0 mg/kg (Dubock & Kaukeinen 1978). Since possum baits contain 20 ppm (or 20 mg/kg), a 20-kg pig would need to eat 1 kg of bait to ingest 1 mg/kg of brodifacoum, which would be approximately equivalent to an LD_{50} dose. (Half this would be approximately an LD_{25} dose, and double this would be approximately equivalent to an LD_{90}). Furthermore, these amounts could approximate the quantity of bait wild pigs might readily obtain if they fed directly from possum bait stations. Some of these would be expected to survive, and even those eating lethal amounts of bait could be hunted and eaten prior to the toxicant taking effect.

All the pigs were killed humanely 5 days after they had started eating toxic bait and before the onset of major clinical signs associated with anticoagulant toxicosis. Serum, liver and muscle tissue (hind limb) samples were taken and stored at –20°C until analysis.

4.2 SECONDARY POISONING OF PIGS

Fifty possums were offered standard brodifacoum possum bait (20 mg brodifacoum per kg of bait) for 4 days to achieve an average of 400 g intake per possum. A small percentage of possums were offered fortified brodifacoum pellets (100 mg brodifacoum per kg of bait) on the fourth night to minimise the variation in the amount of brodifacoum ingested. The possums were killed humanely 5 days after they had started eating bait. Samples of liver and hind limb were taken from six possums and retained at –20°C until analysis. Liver, kidney, heart, muscle, and portions of the small intestine were retained from all possums.

Another 16 pigs were divided into five groups and fed possum tissue over a 2-day period. Group 1 were fed tissues from possums that had not been dosed with brodifacoum (n = 4). Pigs in Group 2 were each fed tissues from one poisoned possum carcass (n = 3). Pigs in Group 3 were each fed tissues from two poisoned carcasses (n = 3), pigs in Group 4 were each fed tissues from four carcasses (n = 3), and pigs in Group 5 were each fed tissues from the equivalent of eight possum carcasses each (n = 3). Five days after the pigs had first started eating the possum meat and organs, they were killed humanely. Liver and hind limb samples were collected from the pigs at post-mortem and stored at –20°C.

4.3 FIELD SURVEY OF 'GAME' SPECIES

A sample of pigs, goats, and deer were shot by hunters in New Zealand in areas where Talon® or PESTOFF® possum bait was currently being used or in areas where these baits had been used 6 months to 5 years previously. At least 50 pigs, 24 goats and 24 deer were to be collected. The areas sampled included Northland, Coromandel, East Cape, central North Island, Nelson Lakes, Canterbury, Southland and the Chatham Islands. Pigs were hunted and caught by dogs and killed by the hunter, and deer and goats were shot. The samples were collected by DOC and Landcare Research staff. The majority of animals were sampled from public conservation lands, but some goats and pigs were sampled on private farmland and forestry land. A 50-g sample of muscle and liver was collected from each animal and frozen as soon as possible. Analysis of brodifacoum residues was carried out by the Landcare Research toxicology laboratory at Lincoln. The high performance liquid chromatography (hplc) method determines the levels of brodifacoum in animal tissues using fluorescence detection. A post-column pH switching technique is used to exploit the natural fluorescence of this compound. Difenacoum is used as an internal standard. The least detectable level in liver tissue is 0.010 µg/g.

The percentage of species contaminated was calculated and the likelihood of hunters being significantly exposed to risk of poisoning from contaminated game was estimated.

Approval was obtained from the Landcare Research Animal Ethics Committee to undertake both the captive animal trials and the field surveys (AEC application no. 97/8/4).

4.4 STATISTICS

Differences in group mean concentrations of brodifacoum in liver, muscle and serum were tested using analysis of variance (ANOVA). Significant effects were further investigated using Bonferoni adjusted pairwise comparisons. The data were square-root transformed to meet assumptions of the analysis. Where the data could not be transformed to meet the assumptions of ANOVA, Kruskal–Wallis tests were used.

5. Results

5.1 PRIMARY POISONING OF PIGS

The concentration of brodifacoum in the muscle and livers of pigs that had eaten toxic bait appeared to be independent of the amount of bait eaten over the range 500–1776 g per pig (Table 1). However, serum concentrations increased with intake of brodifacoum ($F_{3,11}$ = 46.41, $P < 0.001$). Pairwise comparison showed that serum concentrations of brodifacoum in the two high-

TABLE 1. THE AVERAGE AMOUNT OF PESTOFF® BAIT EATEN (g/PIG ± S.E.), THE CORRESPONDING AVERAGE DOSE OF BRODIFACOUM (mg/kg ± S.E.) AND THE CONCENTRATION OF BRODIFACOUM IN PIG LIVER, MUSCLE AND SERUM (mg/kg ± S.E.). VALUES WITH DIFFERENT SUPERSCRIPTS (a, b) ARE SIGNIFICANTLY ($P < 0.05$) DIFFERENT FROM EACH OTHER WITHIN EACH COLUMN.

GROUP	BAIT EATEN (mg BAIT PER PIG)	BRODIFACOUM INGESTED (mg BRODIFACOUM PER kg OF BAIT)	MEAN CONCENTRATION (± S.E). (mg BRODIFACOUM PER kg OF TISSUE)		
			LIVER+	MUSCLE+	SERUM
1	Control non-toxic	0 negligible*	0.04 ± 0.02*	0	0
2	PESTOFF® 500 g/pig (± 31.2 g)	0.57 ± 0.06	1.10 ± 0.07[a]	0.05 ± 0.006[a]	0.17 ± 0.01[a]
3	PESTOFF® 937 g/pig (± 34.2 g)	0.96 ± 0.03	1.08 ± 0.14[a]	0.05 ± 0.01[a]	0.25 ± 0.04[b]
4	PESTOFF® 1776 g/pig (± 81.4 g)	1.94 ±0.09	1.05 ± 0.06[a]	0.05 ± 0.003[a]	0.38 ± 0.06[b]

* Denotes some low-level exposure in control pigs that escaped and ate some toxic bait before recapture.

\+ Concentrations in muscle range from 0.02 to 0.07 and in liver from 0.72 to 1.38 mg/kg.

dose groups were significantly higher (Bonferoni-adjusted $P < 0.05$) than both the control and the low-dose groups. Some animals in the high-dose group had developed clinical symptoms by the time they were euthanased 5 days after dosing. The concentration of brodifacoum in the liver was over 20 times that in the muscle (Table 1).

5.2 SECONDARY POISONING OF PIGS

Possums ate an average 388 ± 41.5 (S.E.) g of bait (or 3.12 ± 0.09 mg/kg bodyweight of brodifacoum) over 4 nights. The concentration of brodifacoum in the possum tissues fed to pigs varied from 0.84 mg/kg (range 0.52–1.20) in the liver to 0.065 mg/kg (range 0.013–0.094) in the muscle (n = 6).

The concentration of brodifacoum in the liver of pigs that ate the soft tissues from poisoned possums increased in a dose-dependent manner ($H_4 = 14.045$, $P = 0.007$), with the highest concentrations present in pigs that had eaten the soft tissue and muscle from eight possums (Figure 1).

5.3 FIELD SURVEY OF 'GAME' SPECIES

A total of 56 pigs, 29 goats and 51 deer were shot. Of these 37 pigs, 23 goats and 36 deer came from areas where Talon® or PESTOFF® bait was currently being used for possum and rodent control. The remaining samples were collected from areas where Talon® and PESTOFF® baiting had ceased at least 6 months previously (19 pigs, 6 goats, and 15 deer).

About two-thirds of pigs sampled had detectable brodifacoum residue present in their livers (Table 2) at least 6 months after baiting had ceased.

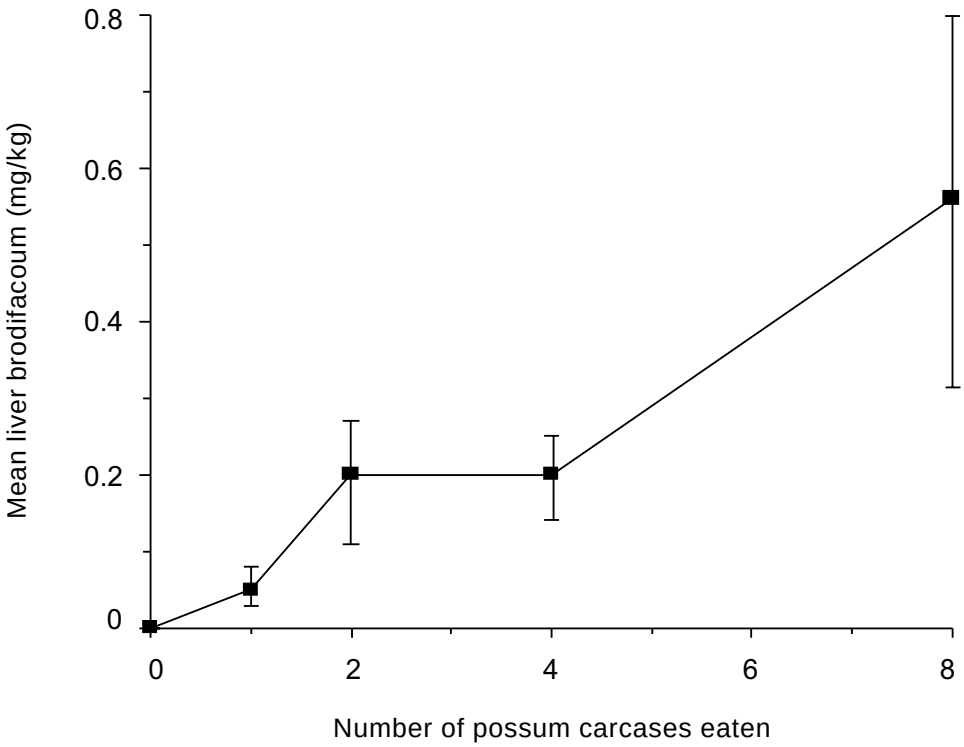

Figure 1. Brodifacoum concentrations (mg/kg) in pig liver 5 days after eating possum tissues. Range is presented as bars (in preference to S.E.) as our analysis did not assume a normally distributed error.

TABLE 2. PERCENTAGE OF FERAL GAME SPECIES WITH BRODIFACOUM RESIDUES IN LIVER.

	PIGS	GOATS	DEER
Current use	29/37 (78%)	2/23 (9%)	14/36 (39%)
Historical use	12/19 (63%)	0/6 (0%)	0/15 (0%)

In the 'current use' areas brodifacoum residues in contaminated pig livers ranged from 0.007 to 1.9 mg/kg (Appendix 1). In the 'historical use' areas pig liver contamination ranged from 0.01 to 2.4 mg/kg. Pigs with contaminated livers were still being found 17 months after Talon® baiting had ceased (Appendix 2). Pigs were the predominant species that had brodifacoum residues detected in their muscle. Generally, those samples that had a liver concentration of brodifacoum greater than 0.5 mg/kg also had detectable residue in the muscle (range 0.01–0.05 mg/kg).

The two contaminated goats had only low levels of residue in their liver (0.01 mg/kg), both of these animals were from 'current use' areas.

The brodifacoum contamination in the livers of deer ranged from 0.01 to 0.03 mg/kg. One deer muscle sample had a level of contamination of 0.02 mg/kg but unfortunately no liver sample was collected from this animal.

Sample sizes were not sufficient to determine if there was a correlation between residual levels and the length of time brodifacoum had been in use or the length of time since its use.

6. Discussion

Trials with captive pigs have indicated that significant contamination of the liver and, to a lesser extent, muscle occurs after both primary and secondary poisoning. In many of the captive pigs, and in 28 of the 41 contaminated feral animals, concentrations in the liver exceeded 0.1 mg/kg, a concentration at which bleeding disorders and death occurred in two out of 36 possums (C. Eason unpubl. data). It is difficult to predict whether in pigs, or indeed humans, a similar effect could be expected at this liver concentration. In risk assessment a precautionary approach is normal, hence we should assume that concentrations of ≥ 0.1 mg/kg are of considerable concern in any non-target species. It is clear that pigs that had repeatedly eaten possums containing high brodifacoum residues had concentrations similar to those found in possums, the original target species. There is a wide range of values in the literature for the LD_{50} of brodifacoum in dogs (0.25–3.25 mg/kg) (Hone & Mulligan 1982). Taking the conservative figure (LD_{50} 0.25 mg/kg), a 15-kg dog would need to eat 37.5 kg of possum liver containing 0.1 mg/kg, or 3.75 kg of liver containing 1 mg/kg. If the LD_{50} for humans and dogs is similar (and for most species the LD_{50} is < 1 mg/kg), a 60-kg person would need to eat approximately 15 kg of liver containing 1 mg/kg to receive an LD_{50} dose.

These calculations suggest that very large amounts of contaminated meat would need to be eaten to cause fatality. Nevertheless, we believe the levels of brodifacoum present in pig livers represent a risk to humans (and farm dogs); this risk is magnified by the persistence of the compound, which could lead to accumulation on repeated exposure. In this regard it is important to remember that a sub-lethal dose well below the LD_{50} will produce significant clotting abnormalities and some haemorrhaging. Indigenous non-target species also at risk from secondary poisoning are predatory and scavenging birds such as weka (*Gallirallus australis australis* and *Gallirallus australis hectori*), Australasian harrier (*Circus approximans*), southern black-backed gull (*Larus dominicanus*), and morepork (*Ninox novaeseelandiae*) (Eason & Spurr 1995).

The low levels of residue found in deer and goats suggest that these species may only eat the occasional pellet spilt beneath bait stations. The one deer sample that had contaminated muscle could be an exception and may reflect that particular individual animal's preference for cereal bait. However, the relatively high contamination rates of deer in 'current use' areas (39%) suggests a high level of feeding on baits by this species, although perhaps only a small number of baits by each animal.

It is well known that there is considerable individual variation in the response to anticoagulant exposure, which may in part depend on the site at which haemorrhaging occurs (e.g. cerebral versus subcutaneous haemorrhaging). This is borne out by the high levels of brodifacoum found in the livers of some pigs that had not been exposed to baits for 6 months or more.

The Department of Conservation has restricted the use of brodifacoum baits to areas where there are no feral pigs, and has moved away from reliance on brodifacoum for maintenance possum control to less-persistent toxicants.

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8. References

Barlow, A.M.; Gay, A.L.; Park, B.K. 1982: Difenacoum (Neosorexa) poisoning. *British Medical Journal 285*: 541.

Buckle, A.P.; Fenn, M.G.P. 1992: Rodent control in the conservation of endangered species. Pp 36–41 in Borrecco, J.E.; Marsh, R.E. (Eds): Proceedings of the Fifteenth Vertebrate Pest Conference. University of California, Davis, USA.

Chong, L.L.; Chan, W.K.; Ho, C.H. 1986: A case of 'superwarfarin' poisoning. *Scandanavian Journal of Haematology 36*: 314–15.

Dubock, A.C.; Kaukeinen, D.E. 1978: Brodifacoum (Talon™ rodenticide), a novel concept. Pp. 127–137 in Howard, W.E.; Marsh, R.E. (Eds): Proceedings of the Eighth Vertebrate Pest Conference. University of California, Davis, USA.

Eason, C.T.; Spurr, E.B. 1995: Review of the toxicity and impacts of brodifacoum on non-target wildlife in New Zealand. *New Zealand Journal of Zoology 22*: 371–379.

Eason, C.T.; Frampton, C.M.; Henderson, R.; Thomas, M.D.; Morgan, D.R. 1993: Sodium monofluoroacetate and alternative toxins for possum control. *New Zealand Journal of Zoology 20*: 329–334.

Eason, C.T.; Henderson, R.; Thomas, M.D.; Frampton, C.M. 1994: The advantages and disadvantages of sodium monofluoroacetate and alternative toxins for possum control. Pp. 159–166 in Seawright, A.A.; Eason, C.T. (Eds), Proceedings of the Science Workshop on 1080, *The Royal Society of New Zealand Miscellaneous Series 28*.

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

Godfrey, M.E.R. 1985: Non-target and secondary poisoning hazards of 'second generation' anticoagulants. *Acta Zoologica Fennica 173*: 209–212.

Henderson, R.J.; Frampton, C.M.; Thomas, M.D.; Eason, C.T. 1994: Field evaluations of cholecalciferol, gliftor, and brodifacoum for the control of brushtail possums (*Trichosurus vulpecula*). *Proceedings of the forty-seventh New Zealand Plant Protection Conference:* 112–116.

Hoffman, R.S.; Smilkstein, M.J.; Goldfrank, L.R. 1988: Evaluation of coagulation factor abnormalities in long-acting anticoagulant overdose. *Clinical Toxicology 26*: 233–248.

Hone, J.; Mulligan, H. 1982: Vertebrate pesticides. *Science Bulletin 89*. Department of Agriculture, New South Wales.

Jones, E.C.; Groew, G.H.; Naiman, S.C. 1984: Prolonged anticoagulation in rat poisoning. *Journal of the American Medical Association 252*: 3005–3007.

Kruse, J.A.; Carlson, R.W. 1992: Fatal rodenticide poisoning with brodifacoum. *Annals of Emergency Medicine 21*: 331–336.

Laas, F.J.; Forss, D.A.; Godfrey, M.E.R. 1985: Retention of brodifacoum in sheep tissues and excretion in faeces. *New Zealand Journal of Agricultural Research 28*: 357–359.

Lipton, R.A.; Klass E.M. 1984: Human ingestion of a 'superwarfarin' rodenticide resulting in a prolonged anticoagulant effect. *Journal of the American Medical Association 252*: 3004–3005.

Merton, D. 1987: Eradication of rabbits from Round Island, Mauritius: a conservation success story. *Dodo Journal of Jersey Wildlife Preservation Trust 24*: 19–43.

Parmar G.; Bratt H.; Moore R.; Batten P.L. 1987: Evidence for a common binding site in vivo for the retention of anticoagulants in rat liver. *Human Toxicology 6*: 431–432.

Robertson, H.A.; Colbourne, R.M.; Nieuwland, F. 1993: Survival of little spotted kiwi and other forest birds exposed to brodifacoum rat poison on Red Mercury Island. *Notornis 40*: 253–262.

Tasheva, M. 1995: Anticoagulant rodenticides. Geneva. Environment Health Criteria Publications World Health Organisation. 121 p.

Taylor, R.H.; Thomas, B.W. 1993: Rats eradicated from rugged Breaksea Island (170 ha), Fiordland, New Zealand. *Biological Conservation 65*: 191–198.

Towns, D.; McFadden, I.; Lovegrove, T. 1993: Offshore islands co-operative conservation project with ICI Crop Care Division: Phase one (Stanley Island). Department of Conservation. *Science and Research Internal Report* 138. 24 p.

Wallace, S.; Paull, P.; Worsnop, C.; Mashford, M.L. 1990: Covert self poisoning with brodifacoum, a 'superwarfarin'. *Australian and New Zealand Journal of Medicine 20*: 713–715.

Weitzel, J.N.; Sadowski, J.A.; Furie, B.C.; Moroose, R.; Kim, H.; Mount, M.E.; Murphy, M.J.; Furie, B. 1990: Surreptitious ingestion of a long-acting vitamin K antagonist / rodenticide, brodifacoum: Clinical and metabolic studies of three cases. *Blood 76*: 2555–2559.

APPENDIX 1

Brodifacoum concentrations from samples taken from areas with ongoing baiting

LOCATION	APPROX. CONTROL AREA (ha)	APPROX. VOLUME OF BAIT (kg)	TIME FRAME	DATE SAMPLED	PIGS L = LIVER M = MUSCLE (mg/kg)	DEER L = LIVER M = MUSCLE (mg/kg)	GOATS L = LIVER M = MUSCLE (mg/kg)
Otamatuna Bay of Plenty	1400	7000 Pulse baiting	9/96 to 8/98	10/96		L0.01 M<LD	
				10/96		L0.01 M<LD	
				11/96		L<LD M<LD	
				11/96		L<LD M<LD	
				11/96		L<LD M<LD	
				11/96		L<LD M<LD	
				12/96		L0.03 M<LD	
				01/97		L0.01 M<LD	
				02/97		L<LD M<LD	
				02/97		L<LD M<LD	
				02/97		L<LD M<LD	
				03/97		L<LD M<LD	
				08/97		L0.02 M<LD	
				09/97		L<LD	
				09/97		L<LD	
				10/97		L<LD	
				10/97		L<LD	
				11/97		L<LD	
				11/97		L<LD	
				12/97	L0.31 M<LD		
				12/97		L<LD	
				01/98		L<LD	
				01/98		L<LD	
				01/98		L<LD	
				08/98	L0.78 M0.02		
				08/98		L0.03	
				09/98		L<LD	
				09/98		L0.02	
				09/98		L0.03	
				09/98		L0.01	
				10/98	L1.02 M0.01		
				10/98	L0.20		
				10/98	L1.50 M0.03		
				10/98	L0.08		
				10/98		L0.01	
				10/98		L0.01	
Onepu Bay of Plenty	500	3000 Pulse baiting	12/96 to 8/98	10/97	L1.09 M0.04		
				08/98	L0.19		
				08/98	L0.10		
				08/98	L0.14		
				08/98	L1.66 M<LD		
				10/98	L0.03		
Pipiriki Wanganui	500 Bait Stations (4000)	1200 Monthly pulses	9/95 to 1/97	08/98	L0.04		
				08/98	L<LD		
				10/98	L0.16		
Te Paki Northland	800	1200 Fortnightly pulses	11/94 to 9/97	02/96	L<LD		
				10/97	L0.24		
				-	L<LD		
				-	L<LD		

TABLE A1 *continued*

LOCATION	APPROX. CONTROL AREA (ha)	APPROX. VOLUME OF BAIT (kg)	TIME FRAME	DATE SAMPLED	PIGS L = LIVER M = MUSCLE (mg/kg)	DEER L = LIVER M = MUSCLE (mg/kg)	GOATS L = LIVER M = MUSCLE (mg/kg)
Mangatutu Pureora	1000	3500 Pulse baiting	8/95- 	11/98 11/98		L0.03 L0.01	
Rotoiti Nelson Lakes	800	360 Continuous over summer months	11/97-	4/98 4/98 4/98		L<LD L<LD L<LD	
Mapara King Country	1400	8000 Pulse baiting	9/93 to 4/97	1/95 1/95 1/95 1/96 1/96 1/96 6/96 8/96 8/96 9/96	L0.007 L1.7[1] L0.21 L0.009 L1.6 M0.03 M<LD L<LD L<LD L<LD		
Waipapa Pureora	2480 (1700 bait stations)	20 400	12/95-	10/98 10/98 10/98 10/98	L1.90 M0.01 L0.52 M<LD L0.26 M<LD L0.09 M<LD		
Moehau Coromandel	4500 (1800 bait stations)	10 000	4/95-	2/98	L1.78 M0.05		
Southland	540	5000 Pulse baiting	9/93-	11/97			L<LD
Erua Forest	106 bait stations	1049 Pulse every month	95-	6/98		M0.02	
TOTAL					37	36	23

<LD = below detectable limit.
[1] Animals found dead.

APPENDIX 2

Brodifacoum concentrations from samples taken from areas where baiting ceased at least 6 months previously

LOCATION	APPROX. CONTROL AREA (ha)	APPROX. VOLUME OF BAIT(kg)	TIME FRAME	DATE SAMPLED	PIGS L = LIVER M = MUSCLE (mg/kg)	DEER L = LIVER M = MUSCLE (mg/kg)	GOATS L = LIVER M = MUSCLE (mg/kg)
Tuku Reserve & Awatotara Covenant, Chatham Islands	1000	record not kept	Large-scale possum control 4/98	4/99 5/99 5/99 5/99 - - 7/99	L0.24 L0.01 L<LD L<LD L0.11 L<LD L<LD		
Mapara King Country	1400	8000 approx. Pulse baiting	9/93 to 4/97	11/97 11/97 11/97 1/98 6/98 6/98 8/98 9/98	L<LD L<LD L<LD L<LD L0.01 L<LD L0.59 M<LD L<LD		
Onepu Bay of Plenty	500	3000 Pulse baiting	12/96 to 8/98	3/99 3/99 3/99	L<LD L2.40 L<LD		
Otamatuna Bay of Plenty	1400	7000 Pulse baiting	9/96 to 8/98	2/99 2/99 2/99 3/99 4/99 4/99 4/99 5/99 7/99 7/99 8/99 8/99 8/99 8/99 8/99 8/99 9/99 9/99 9/99 9/99 9/99	 L1.73 M0.02 L0.05 L0.04 L0.68 M0.02 L0.40 M0.02 L1.60	L<LD L<LD L<LD L<LD L<LD L<LD L<LD L<LD L<LD L<LD L<LD L<LD L<LD L<LD L<LD	
Pipiriki Wanganui	500 Bait stations (4000)	1200 Monthly pulses	9/95 to 1/97	6/99 6/99 6/99 7/99 7/99 7/99			L<LD L<LD L<LD L<LD L<LD L<LD
TOTAL					19	15	6

<LD = below detectable limit.