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SURVIVAL OF BROWN KIWI (*APTERYX MANTELLI*) EXPOSED TO BRODIFACOUM POISON IN NORTHLAND, NEW ZEALAND

Summary: Brown kiwi (*Apteryx mantelli*) in central Northland have been monitored for up to 32 months of sustained exposure to brodifacoum poison. The cereal baits were placed in bait stations to target brushtail possums (*Trichosurus vulpecula*). Annual survival of 55 radio-tagged adult kiwi in two poisoned forest patches has been high (95.9%), and similar to that in two nearby unpoisoned forest patches and in the patches before poison was used (95.3%). The two adult birds tested of the four that died in the poisoned areas had no traces of brodifacoum at the detection limit of 0.05 mg kg⁻¹. With an improved detection limit of 0.02 mg kg⁻¹, no traces of brodifacoum were found in nine eggs collected over 18 months after adults were first exposed to the poison. Of four chicks tested (detection limit: 0.005 to 0.02 mg kg⁻¹), that had apparently died of natural causes or were killed by predators, three contained traces of brodifacoum (0.01 - 0.18 mg kg⁻¹). The median survival of 39 radio-tagged chicks in the poisoned areas (36 days) was significantly better than that of 18 chicks in untreated areas nearby (15 days). Kiwi chicks may be more vulnerable than adult kiwi to accidental poisoning because of physiological or behavioural differences; however, the effects of sustained exposure to low levels of brodifacoum poison may not be visible in the adult population for several years. At this stage of the ongoing study, the benefits to kiwi of this method of pest control outweigh the costs, because chick survival is greatly improved.

Keywords: Brown kiwi; *Apteryx mantelli*; brodifacoum; poison; pest control; survival.

Introduction

The brown kiwi (*Apteryx mantelli* Bartlett) is a large (2000-3500 g) flightless nocturnal bird endemic to New Zealand. They are unusual in having a well-developed sense of smell, with their nostrils being placed near the tip of the long (90-150 mm) bill (Heather and Robertson, 1996). Adult kiwi probe for large invertebrates in the top 100 mm of the soil or from the surface litter, and occasionally eat fallen fruits (Kleinpaste, 1990). Formerly widespread in the North Island and northern South Island, the range of the brown kiwi has contracted substantially since human settlement of New Zealand about 1000 years ago and the species is now regarded as threatened (Collar, Crosby and Stattersfield, 1994). McLennan *et al.* (1996) pooled data from a number of radio-tracking studies to show that the numbers of brown kiwi were declining on the mainland at an average rate of 5.8% per year. Miller and Pierce (1995) documented the rapid decline in brown kiwi in Northland since the 1970s, and noted that apart from widespread deaths of adult kiwi from dogs (e.g., Taborsky, 1988; Pierce and Sporle, 1997), the decline coincided with the arrival of ferrets (*Mustela furo* L.) and brushtail possums (*Trichosurus vulpecula* Kerr) in Northland.

Possoms were introduced to New Zealand from Australia in 1858 to establish a fur industry (Cowan, 1990). They have become a serious pest as a vector of bovine tuberculosis, which threatens multi-billion dollar dairy, beef and deer industries, and because they cause substantial damage to ecosystems that evolved in New Zealand in the absence of browsing or predatory mammals. Up until the 1970s, possums were rare in Northland. Over the past 25 years, however, a wave of possums has swept through the region from the south.

Possoms eat kiwi eggs and occasionally kill adult kiwi in disputes over burrow occupancy (McLennan *et al.*, 1996). A much greater threat is posed indirectly via possum control operations using leg-hold traps or ground-laid cyanide baits (Robertson *et al.*, 1999). The Department of Conservation uses four main techniques to protect native forests from possums: leg-hold traps (set at least 70 cm off the ground in areas occupied by flightless birds such as kiwi), cyanide poison in a paste or encapsulated (also set 70 cm off the ground in kiwi areas), Compound 1080 (sodium monofluoroacetate) poison in cereal baits used in aerial and ground-based (hand-spread or bait station) applications, and brodifacoum poison in cereal baits placed in bait stations. Usually the last technique is

used to keep possum populations at low densities after an initial knock-down using one of the other techniques. This paper reports on the preliminary results of a study of the survival of brown kiwi exposed to brodifacoum poison (Talon® or Pestoff®) placed in Philproof® bait stations during a possum control operation which mimics a standard Department of Conservation operation.

Several studies have documented the survival of kiwi exposed to aerially-sown brodifacoum poison baits used to eradicate rats (*Rattus* spp.) off islands around the New Zealand coast. Robertson, Colbourne and Nieuwland (1993) reported that all nine radio-tagged little spotted kiwi (*Apteryx owenii* Gould) on Red Mercury Island survived at least 27 days, and Colbourne and Robertson (1997) indicated that there was no long-term problem for this population. Likewise, they reported that the little spotted kiwi population on Tiritiri Matangi Island had increased rapidly since the rat-poisoning there in 1993, even though a bird that drowned more than 10 months later contained traces (0.01 mg kg^{-1}) of brodifacoum in its liver. On Kapiti Island, Robertson and Colbourne (*unpubl. data*) estimated from band recoveries that between 2 and 5% of Little Spotted Kiwi had been accidentally poisoned during the rat eradication programme there in spring 1996; one radio-tagged bird that died from accidental poisoning contained 1.2 mg kg^{-1} of brodifacoum in its liver. No studies have followed the survival of adult kiwi during sustained exposure to brodifacoum poison presented in bait stations rather than one-off aerial applications, nor has anyone measured the toxin levels in eggs or chicks in a poisoned area.

Rammell *et al.* (1984) reported a wide range of brodifacoum levels in the livers of large birds found dead after a rabbit-control trial, ranging from 0.12 mg kg^{-1} in one of two harriers (*Circus approximans* Peale) to 4.0 mg kg^{-1} in a paradise shelduck (*Tadorna variegata* Gmelin). Godfrey (1985) estimated the acute oral toxicity of brodifacoum for a small range of introduced and native birds in New Zealand (but not kiwi) and found that the LD_{50} levels were also highly variable, with more than a 25-fold difference between black-backed gulls (*Larus dominicanus* Lichtenstein) and pukeko (*Porphyrio porphyrio* L.) each with an LD_{50} of < 0.75 , and paradise shelduck with an LD_{50} of > 20 . The published brodifacoum levels for birds may not reflect the true vulnerability of kiwi to toxins because kiwi are an endemic order of birds which have many unusual physiological features such as low, mammal-like, body temperature and basal metabolic rate (McNab, 1996). It was with this in mind that we investigated the survival of kiwi during a brodifacoum poison operation.

Methods

Study area

For our study which aims to determine and manage the threats to brown kiwi, we chose four remnant patches of broadleaf-podocarp forest in central Northland within a 5 km radius of $35^{\circ}37'S$, $174^{\circ}08'E$ (Fig. 1). These forest patches lie just to the north of the line marking the southern limit of brown kiwi distribution in Northland (Miller and Pierce, 1995).

Aponga Scenic Reserve (33 ha) and an adjoining block of privately-owned forest (22 ha), known collectively as Rarewarewa, was chosen for intensive possum control. The canopy and subcanopy of this 365 m volcanic cone and adjacent broad valley are dominated by taraire (*Beilschmiedia tarairi* A. Cunn.), with lesser amounts of towai (*Weinmannia silvicola* Sol. ex A. Cunn.), kohekohe (*Dysoxylum spectabile* Forst. f.), hinau (*Elaeocarpus dentatus* J.R. & G. Forst.), karaka (*Corynocarpus laevigatus* J.R. & G. Forst.) and puriri (*Vitex lucens* T. Kirk). The site has been modified by the pre-European construction of a pa (Maori fortress) on the highest point, and the extraction of emergent podocarps and kauri (*Agathis australis* Salisb.) in the early 1900s, but some individual large emergent totara (*Podocarpus totara* G. Benn. ex Don), rimu (*Dacrycarpus dacrydioides* A. Rich.) and kauri

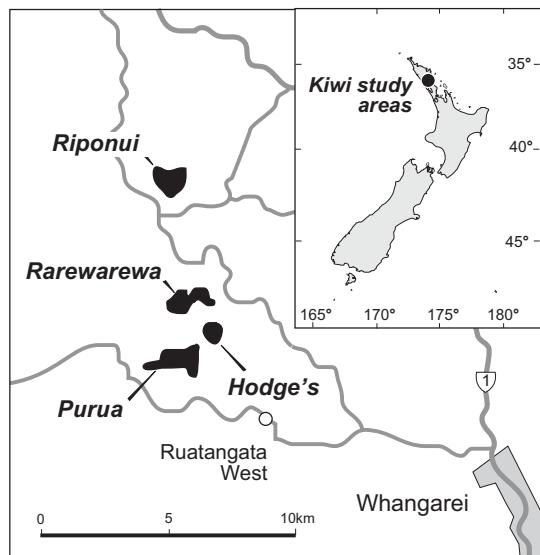


Figure 1: Map of central Northland showing the location of the four study sites.

remain. The reserve and private land have been further modified by grazing of sheep, cattle and goats, but over the past three years only 8 ha of the private forest has been unfenced and open to farm stock.

Riponui Reserve (45 ha) is on the side of a low ridge (250 m above sea level) and is deeply incised by a series of parallel streams running towards the lower boundary (130 m above sea level). The vegetation is similar to that in Rarewarewa and has also historically suffered some extraction of timber trees.

For this part of our study, the non-treatment blocks were Purua Reserve and Hodge's Bush. Purua is a 110 ha volcanic cone with very similar topography and vegetation to Rarewarewa, except for being more heavily vegetated and hence damper on the northern side. Hodge's Bush is a 35 ha patch of privately-owned forest with similar canopy composition to the other study sites, but it lies in a broad valley and is almost devoid of undergrowth as it is grazed by farm stock.

Kiwi monitoring

We used trained, muzzled labrador dogs to locate the daytime dens of brown kiwi. Between January 1994 and June 1998, we have banded, wing-tagged or radio-tagged a total of 343 individuals (83 adult males, 84 adult females, 45 juveniles and 131 nestlings) in the four study areas. In the North Island, only the male brown kiwi incubates (Heather and Robertson, 1996). To find nests, we attached a 25 g radio-transmitter (Sirtrack, Havelock North, NZ) to the tibiotarsus of most males, using two plastic hospital identification bracelets and several rounds of electrical tape. For this study, we made a concerted effort to radio-tag female kiwi in Rarewarewa so that we would have a representative sample of males and females in case they reacted differently to the poison. We checked the adult birds about once a month to determine their survival or cause of death.

We followed the fate of kiwi nests and collected infertile, rotten or abandoned eggs for brodifacoum analysis. About a week after hatching, we attached a 10 g radio-transmitter (Miles and McLennan, 1998) to the tibiotarsus of most chicks and checked them every 2-4 weeks to determine their survival or cause of death. In the 1997/98 season, we collected liver samples from chicks that had either died of natural causes or were killed by predators.

Poison operation

In May 1995, cereal and jam baits containing 0.15% 1080 were laid at Rarewarewa to knock-down the

possum population and to test if kiwi were at risk of accidental poisoning from this toxin (Robertson *et al.*, 1999). In October 1995, we started to use brodifacoum baits, placed in Philproof® bait stations (Philproof Feeders, Hamilton, NZ) nailed to the trunks of large trees. A total of 78 bait stations were placed in the 47 ha portion of the forest patch that was closed to farm stock. The bait stations were about 150 m apart on tracks that followed the edge of the bush patch and then natural contours within the area rather than a grid, because of the steep terrain. We placed 500-800 g of green-dyed and cinnamon-lured brodifacoum baits in each station about every two months from October 1995 to June 1998, except for a four month trial (December 1995 to March 1996) in 14 ha of Rarewarewa using cholecalciferol poison (Campaign®, Key Industries, Birkenhead, NZ). We used cereal brodifacoum baits (Talon 20P®, ICI Cropcare, Motueka, NZ) up until September 1997, and since then have used wax-coated cereal baits (Pestoff®, Animal Control Products, Wanganui, NZ). The poison baits contained a nominal 20 ppm or 0.02 g kg⁻¹ of brodifacoum. Possums and rats gain direct access to baits in the stations, but mice (*Mus musculus* L.) probably feed on baits spilled from the stations or on baits cached by rats. Between 24 October 1995 and 30 June 1998, 1121 kg of poison baits have been used at Rarewarewa (23.9 kg ha⁻¹).

At Riponui, we carried out a cyanide operation in October 1997 to knock the possum population down (210 possums were killed and removed from the 45 ha bush patch in a single night). We have replenished Pestoff baits monthly in 47 bait stations situated at about 150 m intervals around the perimeter of the reserve and along the internal parallel ridge system. Since 2 October 1997, 244 kg of baits have been used at Riponui Reserve (5.4 kg ha⁻¹).

For this paper, data for kiwi not exposed to brodifacoum was derived from Purua and Hodge's Bush, along with Rarewarewa from January 1994 to October 1995 and Riponui from May 1994 to October 1997.

Analysis

Residue analysis

Liver samples from dead adults and chicks, and whole eggs were analysed for traces of brodifacoum by the National Chemical Residue Analytical Laboratory of the Ministry of Agriculture at Wallaceville, New Zealand or by Landcare Research, Lincoln, New Zealand. The detection limit for brodifacoum in samples from adult kiwi was 0.05 mg kg⁻¹, but with improving analytical methods

the detection limit has been lowered to 0.02 and then 0.005 mg kg⁻¹ for the more recent samples from eggs or chicks.

Statistical analysis

Kiwi survival in poisoned and unpoisoned areas was compared using the Kaplan-Meier technique for analysis of radio-tracking data (Pollock *et al.*, 1989), with the Mantel-Haenzsel statistic approximating a χ^2 distribution with 1 degree of freedom. For the analysis of chick survival, we censored survivorship data at 182 days (6 months) of age, because at about this age the chicks become big enough to defend themselves from stoats and feral cats and so their probability of daily survival increases rapidly (*unpubl. data*).

Results

Toxin analysis

Through the entire study, 11 adults (ten males and one female) have died and four of these (all males) were in the poisoned area. Of the four birds that died in the poisoned area, two were killed or scavenged by ferrets or dogs (in February 1996 and August 1996), one died of no obvious cause (in January 1996), and one drowned in a natural tunnel during a

cyclonic storm in January 1997. The two birds killed in January and August 1996 were still in a suitable condition to obtain tissue samples, but no traces of poison were found.

We collected nine eggs that had failed to hatch during the 1997/98 breeding season in the poisoned site, but none contained detectable traces of brodifacoum.

Of the four dead chicks tested in the 1997/98 breeding season, three contained traces of brodifacoum: 0.01, 0.01 and 0.18 mg kg⁻¹. The ages at death (and hence maximum exposure time) of these three chicks were 110, 364 and 60 days respectively. The one that showed no detectable brodifacoum was only about four days old when it died and at that age may not have even ventured from the nest to feed.

Survival and productivity of kiwi

The number of adults radio-tracked, and the total time they were at risk from accidental poisoning by brodifacoum while carrying an active transmitter are given in Table 1. The survival of adult males and females did not differ significantly (Mantel-Haenzsel $\chi^2 = 1.2$, $P > 0.05$) and so their data are pooled. The annual survival of adults exposed to brodifacoum (95.9%) was not significantly different from the survival (95.3%) of adults not exposed to brodifacoum (Mantel-Haenzsel $\chi^2 = 0.2$, $P > 0.05$).

Table 1: *Survival of radio-tagged adult brown kiwi living in poisoned and unpoisoned areas. At Rarewarewa, 18 males and 10 females had been exposed to the poison for a sustained period of 32 months from late October 1995 to early May 1998, while the remainder of the Rarewarewa and Riponui samples had been exposed for shorter periods.*

Site	Period	Sex	No. of birds	Bird-years tracked	No. of deaths
Poisoned sites:					
Rarewarewa	Oct 1995-May 1998	M	28	55.1	4
		F	15	32.5	0
Riponui	Oct 1997-May 1998	M	9	6.3	0
		F	3	1.2	0
Total			55	95.2	4
Life expectancy	23.8 years				
Annual survival	95.9 %				
Unpoisoned sites:					
Rarewarewa	Jan 1994-Oct 1995	M	26	25.9	0
		F	20	10.8	0
Riponui	May 1994-Oct 1997	M	11	27.3	2
		F	5	1.2	0
Purua	May 1994-May 1998	M	19	43.4	1
		F	8	3.7	0
Hodge's	May 1994-May 1998	M	14	29.2	3
		F	7	4.1	1
Total			110	145.9	7
Life expectancy	20.8 years				
Annual survival	95.3%				

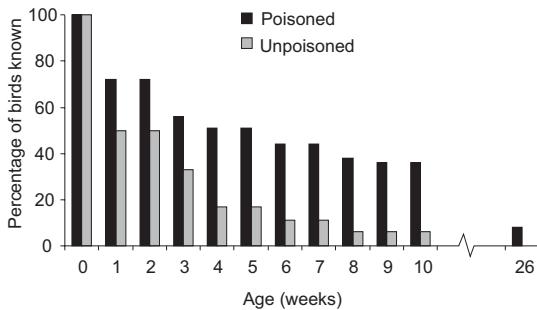


Figure 2: Survival of brown kiwi chicks radio-tagged while still in the nest. The median survival of the 39 chicks living in the poisoned areas was 36 days (5.1 weeks) compared with 15 days (2.1 weeks) for the 18 chicks living in the unpoisoned areas. After 26 weeks (182 days) three chicks were still living in the poisoned sites and none were known to be alive in the unpoisoned sites.

We found no significant difference in nesting success between the poisoned (60% of 65 nests) and unpoisoned (54% of 57 nests) areas in the 1996-98 seasons ($\chi^2_1 = 0.4$, $P > 0.05$).

The median survival of 39 radio-tagged chicks (36 days) in the poisoned forest blocks in 1996-98 (we excluded the 1995/96 season because we used 1080 poison in the early part of the season) was significantly better (Mantel-Haenzel $\chi^2 = 8.8$, $P < 0.01$) than that of 18 radio-tagged chicks (15 days) in the nearby untreated forest patches in the same years (Fig. 2). In the poisoned areas, 11 (28%) of the 39 chicks were identified as having been killed by stoats or feral cats, whereas in the unpoisoned areas 10 (56%) of the 18 chicks were killed by stoats or feral cats ($\chi^2_1 = 3.9$, $P < 0.05$).

We estimated from nesting success and survival of chicks that the productivity in the poisoned blocks was sufficient to replace adult losses, whereas in the untreated blocks, the populations were in decline (*unpubl. data*).

Discussion

There has been much debate in the popular media about the use of 1080 poison to control possums in New Zealand and the effects it has on non-target wildlife, whereas brodifacoum has not received the same scrutiny. This is ironic because brodifacoum is much more persistent and can accumulate in living tissue (Eason and Spurr, 1995), whereas 1080 is broken down very rapidly (Gooneratne *et al.*, 1995).

The LD₅₀ of brodifacoum (or any other toxin) in kiwi is not known, and we expect that there would

be a public outcry if such native birds were subjected to the rigorous testing required. The only alternative method of determining the effect of such toxins is to monitor the fate of birds following pest control operations, and to build up a database on the cause of death and level of toxins discovered in a large sample of birds.

This is the first study to have assessed the impacts of brodifacoum possum control operations on a large sample of brown kiwi, and includes data from different stages in the life-cycle. The data suggest that brodifacoum, if present, was not passed from female kiwi to their eggs, but that chicks somehow came into contact with the toxin. They may either have fed on baits or fed on invertebrates that had ingested the poison. At this stage, it seems that adults are less likely to come into contact with the toxin even though adults and chicks both feed mainly within the forest around the bait stations. Kiwi chicks may be more vulnerable than adults to accidental poisoning if they have different physiology or behaviour; for instance, kiwi chicks are more liable to feed on novel food items (Tamsin Ward-Smith, Massey University, NZ, *pers. comm.*). With a short bill, chicks are restricted to feeding on surface-dwelling invertebrates and so may be more likely to come into contact with invertebrates feeding on baits that had fallen to the ground or been cached by rodents (rats or mice), and invertebrates feeding on possum and rodent carcasses on the ground.

We had no evidence from our study to show that either rats or possums were killing kiwi chicks. The significantly better survival of kiwi chicks in the blocks treated with brodifacoum therefore suggested that the poisoning operation affected the density of stoats and cats, the main predators of kiwi chicks. We believe that these predators either died from secondary poisoning after eating dead or dying rodents and possums, or they encountered very low densities of rodents (their main prey in Northland forests) in the poisoned blocks and, following an ideal-free distribution (Krebs and Davies, 1981), they moved on to other patches where prey were more abundant. Despite only a small area of forest being poisoned and hence a rapid rate of re-invasion of predators, the poison indirectly eased predation pressure on kiwi chicks to allow the populations in the treated areas to become at least self-sustaining for the duration of the study to date. However, the gains may be only temporary if prolonged use of poison eventually results in increased populations of invertebrates, lizards and other bird species which could actually provide an increased prey base for predators.

We still do not know the long-term effect to kiwi of the sustained application of brodifacoum.

We have shown that kiwi chicks are ingesting the toxin, even though the doses recorded are generally below the levels affecting the physiology, behaviour or survival of the chicks concerned; however, one chick had a higher level (0.18 mg kg^{-1}) than the 0.12 mg kg^{-1} that was apparently fatal in a harrier (Rammell *et al.* 1984). Although we failed to detect any toxin in two adults, and the overall survival has been high during the period of exposure to the baits (up to 32 months), we are not yet confident that they are not slowly accumulating the toxin which may have fatal consequences at a later date. We also do not know whether or how fast kiwi eliminate brodifacoum from their bodies.

The use of toxins such as brodifacoum for pest control can have profound benefits to particular species, ecosystems, and to the rural economy. However, we must be aware of the possible side-effects and the potential for a backlash in public opinion about the use of such poisons unless we are able to demonstrate the gains as well as openly acknowledge the losses and risks. We must also strive to modify pest control operations to reduce the amount of poison used to the minimum amount needed to achieve the desired conservation and/or economic benefit. At this stage of our ongoing study, we acknowledge that kiwi chicks are at risk of accidental poisoning by brodifacoum baits presented in bait stations, but unless a significant cumulative effect appears in adults, or chicks are shown to die from brodifacoum poisoning, the benefits to kiwi populations outweigh the costs because chick survival is greatly improved.

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