SHORT COMMUNICATION

Bait consumption and residual concentrations of diphacinone in the Wellington tree weta (*Hemideina crassidens*) (Orthoptera: Anostostomatidae)

Penny Fisher[1,2 *], Eric B. Spurr[1], Shaun C. Ogilvie[2] and Charles T. Eason[2]
[1] Landcare Research, PO Box 40, Lincoln 7640, New Zealand
[2] Bio-Protection and Ecology Division, Lincoln University, PO Box 84, Lincoln 7640, New Zealand
* Author for correspondence (Email: fisherp@landcareresearch.co.nz)

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Abstract: To investigate the potential for mortality or sublethal effects in the tree weta (*Hemideina crassidens*) as the result of exposure to baits used for rodent control, and the potential secondary hazard to non-target species, captive weta were offered Ditrac® wax block bait containing the anticoagulant diphacinone. Bait consumption was recorded daily for the first week and then weekly. Weta were sampled in groups of four following 1, 4, 8, 16, 31, and 64 days of exposure to bait and analysed to determine the concentration of diphacinone residues in their bodies. Any changes in feeding behaviour, survival, and bodyweight were recorded. Weta found Ditrac wax block baits palatable even in the presence of natural plant food, showing steady consumption of bait over time. No mortality or weight loss was attributable to the intake of Ditrac bait. All weta that ate bait had detectable diphacinone in their bodies, but did not accumulate diphacinone, i.e. whole-body concentrations did not increase with the amount of diphacinone bait eaten over time. Field use of diphacinone bait is likely to present a low risk of mortality to weta, but the risk posed by secondary diphacinone exposure to non-target species that eat weta requires further investigation.

Keywords: non-target species; poisons; secondary exposure; vertebrate pest control

Introduction

Bait formulations containing anticoagulant compounds are used worldwide for commensal rodent control (e.g. Hadler & Buckle 1992). In New Zealand, management strategies for introduced pests such as brushtail possums (*Trichosurus vulpecula*) and rodents (*Rattus rattus*, *R. norvegicus*, *R. exulans* and *Mus musculus*) sometimes include field application of bait containing the second-generation coumarin anticoagulant brodifacoum (e.g. Innes et al. 1995). Field monitoring following bait station and broadcast application of baits has reported brodifacoum residues in native birds (e.g. Eason & Spurr 1995), introduced mammals (e.g. Spurr et al. 2005) and invertebrates (e.g. Craddock 2003). The first-generation indandione anticoagulant diphacinone used in bait station applications is therefore being investigated as an effective but less persistent alternative to brodifacoum for controlling field populations of introduced rodents in New Zealand (Gillies et al. 2006). Diphacinone is also undergoing registration in the United States for control of rats in Hawai'i using broadcast application (Johnston et al. 2005).

It has been suggested that anticoagulants lack insecticidal properties because insects do not have the same blood-clotting systems as vertebrates (Shirer 1992). Limited studies indicate that insects do not appear to be at risk of mortality from brodifacoum poisoning (Booth et al. 2001). Diphacinone is considered 'moderately toxic' to the freshwater invertebrate *Daphnia magna* (US EPA 1998). While there appear to be no comparable data regarding the acute toxicity of diphacinone to terrestrial insects, there are early reports of insecticidal properties in compounds structurally related to diphacinone (2-(diphenylacetyl)-1,3-indandione). For example, isomeric valeryl-1,3-indandiones exhibit strong insecticidal properties against houseflies (*Musca domestica*) (Kilgore et al. 1942), 2-pivalyl-1,3-indandione (pivalyl) shows toxic effects against body lice (*Pediculus humanus corporis*) (Eddy & Bushland 1948), and 0.025% pivalyl cereal baits applied in field trials for rodent control also had insecticidal properties (Crabtree & Robinson 1953). A range of terrestrial insect species, including weta, have been reported to feed on cereal-based baits used for vertebrate pest control in New Zealand (e.g. Ogilvie

et al. 1997; Spurr & Drew 1999; Craddock 2003), and thus have primary exposure to the active ingredient of the bait. We sought to ascertain whether the Wellington tree weta (*Hemideina crassidens*), a regionally common, large, native New Zealand orthopteran, would feed on a currently available diphacinone bait formulation and, if so, whether this would cause toxicity or mortality.

Insects that have low susceptibility to anticoagulant toxicity and feed on baits could carry significant concentrations of anticoagulant residues into the environment, and pose a risk of secondary exposure to insectivores and scavengers, possibly causing non-target mortality. For example, several birds in a zoo aviary died after apparently eating ants (Formicidae) and cockroaches (Blattidae) that had eaten brodifacoum baits (Godfrey 1985). However, the risk of secondary mortality of non-target species caused by anticoagulant residues in field conditions has not been assessed fully, perhaps because of the relative scale and complexity of the field studies that would be required to do so. Native New Zealand birds that eat weta, and therefore potentially could be at risk from secondary poisoning if the weta had eaten diphacinone bait, include kiwi (*Apteryx* sp.), weka (*Gallirallus australis*), morepork (*Ninox novaeseelandiae*), kaka (*Nestor meridionalis*), and saddleback (*Philesturnus carunculatus*) (Gibbs 1998). We sought to measure diphacinone residues in the bodies of weta after they had fed on baits for different periods of time, to provide a basis for theoretical assessments of the secondary hazards to predators and scavengers of weta following field applications of diphacinone baits.

Methods

Capture, housing, and husbandry of weta

Thirty-eight adult and late-instar juvenile Wellington tree weta were captured from podocarp–broadleaf coastal forest habitat near Harihari, West Coast, South Island, New Zealand (2311045E, 5781505N). Each weta was placed in a ventilated plastic container with leaf litter and transported the following morning into captive housing. Maintenance of weta in captivity was based on conditions described by Barrett (1991). Weta were housed individually in cylindrical plastic containers approximately 200-mm diameter × 300 mm high, with close-fitting 'clip-on' plastic lids. Each housing unit had two or three ventilation holes (approximately 2-cm diameter) in the sides of the container, covered over with fine metal mesh. The base of each unit had a 4-cm layer of sand covered with leaf litter from the site of capture. Two plastic test-tubes (9.5 cm long and 1.7-cm diameter) were taped vertically to opposite inner sides of each unit and filled with water so that they could hold sprigs of plants as food. Each unit contained a shelter made from hollow flax-flower stalks (150 mm long), split in half and then held together with rubber bands. The housing containers were kept under natural photoperiod on a laboratory benchtop out of direct sunlight, with ambient room temperature ranging from 10 to 25°C, and humidity of 50–75%, maintained by misting with tap water. Weta were checked twice weekly and visually confirmed to be alive for 1 month before trials began.

Native plant material was used as maintenance diet. Sprigs of five to eight leaves of five-finger (*Pseudopanax arboreus*), māhoe (*Melicytus ramiflorus*), and broadleaf (*Griselinia littoralis*) were placed in the water tubes inside each housing unit and replaced at least weekly, or more frequently if any were substantially eaten or wilting. Consumption of plant food was noted during the twice-weekly checks of the weta. Weta were also weighed every 7–10 days, although any that were moulting were not handled. Faecal pellets were only removed from the housing units during trials if they were visibly mouldy. Housing units were cleaned out thoroughly between trials.

Diphacinone bait consumption, survival and residues in weta

Pilot trial

To ascertain whether weta were likely to feed continuously on diphacinone baits and whether this would result in mortality, four weta (2 females, 2 males) were presented with a Ditrac® block bait (Pest Management Services, Paraparaumu, NZ) placed on a glass dish on the floor of the housing unit, in the presence of normal plant food, for 44 days. The baits were waxed cereal blocks dyed a pale green colour, nominally containing 0.005% diphacinone (50 ppm) as the active ingredient by weight. A sample of the fresh bait was analysed for diphacinone concentration by the Landcare Research toxicology laboratory, Lincoln, using an HPLC method based on that of Hunter (1984). Weta and baits were weighed at the start and end of the 44-day period, and regular observations of the bait and health of the weta were made.

Main trial

After the four weta in the pilot trial had survived at least 3 weeks' exposure to diphacinone baits (see Results), a larger trial was established to measure consumption of diphacinone bait by weta over time and the resultant concentrations of diphacinone in their bodies. Twenty-seven weta (18 female, 9 male) were individually presented with a Ditrac block bait on a glass dish as previously described. Weta and baits were weighed on the day baits were placed in the containers. For the first week, the baits were observed daily for fresh feeding marks, presence of crumbs on the bait

dish and presence of mould. Thereafter, bait condition was recorded and weta were confirmed to be alive at weekly intervals, during the routine replacement of plant food in the housing units. This was intended to minimise disturbance of the weta that may have affected their feeding behaviour. On days 1, 4, 8, 16, 31, and 64 after placement of baits, a sample of four weta (initially 2 male, 2 female) was randomly selected. The weta were weighed, placed in screw-top plastic specimen containers, and left overnight in a freezer at –20°C to kill them, prior to analysis for residual diphacinone. Baits from the housing units were weighed, including fragments of bait where it was possible to separate them from sand present on the dish. Seven other weta maintained in the laboratory on normal plant food were weighed and observed regularly for mortality during this trial to provide some sort of control group, as we could not obtain a non-toxic version of the Ditrac formulation to present to a formal control group.

Analyses for diphacinone concentrations in weta and bait were carried out by the Landcare Research toxicology laboratory, using an HPLC method based on that of Hunter (1984), with a limit of detection of 0.2 μg g^{-1}. Whole, frozen weta were dissected and mixed with anhydrous sodium sulphate and subsequently extracted with solvent (chloroform/acetone/formic acid). The mixture was homogenised with a tissue disperser, shaken and centrifuged. The supernatant was decanted and the extraction repeated twice more. The combined extracts were evaporated and taken up in hexane/chloroform/acetone for application to a solid-phase extraction column for clean-up. The eluent from the column was again evaporated and taken up in mobile phase for HPLC determination, which employed ion-paired chromatography and UV detection at 284 nm. Each batch of samples analysed included a spiked sample, where 50 μL of 10 μg mL^{-1} diphacinone was added to a suitable blank matrix in order to determine recovery.

Two environmental-control housing units (with one bait but no weta) were established and the baits in these were observed and weighed at each sampling interval in order to correct estimates of bait intake by weta for ambient changes in bait weight (moisture content). By the end of the trial, these baits gained weight, with a mean increase of 2.68%, and appeared slightly less sharp in outline. Bait consumption by weta was estimated by correcting the start weight of the bait using the corresponding mean change in the environmental-control baits at each sampling interval, and then subtracting the weight of the bait at sampling. From this figure, diphacinone intake by weta was estimated at each sampling interval using the measured concentration of 52.5 μg diphacinone per gram of bait (52.5 ppm), adjusted according to the weight of the individual weta at sampling (i.e. intake as micrograms of diphacinone per gram weta bodyweight).

The amount of bait consumed (or removed) by weta as a proportion of their starting bodyweight was analysed using weighted regression in a generalised linear model in Genstat (Genstat Committee 2005). There was considerable heterogeneity in the data. The relative growth rate of weta between the pretrial acclimatisation and trial periods (i.e. bodyweight gained, in g g^{-1} day^{-1}) was also compared using weighted regression.

Results

The weta brought into captivity ate the plant food provided readily and used the flax-stalk shelters during daylight. When brought into captivity, the mean weight of 11 adult females (± SE) was 3.40 ± 0.34 g and of 9 adult males was 2.82 ± 0.23 g. Overall, the weta maintained or gained weight in captivity, with the mean weight of the same 11 adult females being 3.98 ± 0.27 g, and 9 adult males 2.97 ± 0.20 g, after one month in captivity.

Diphacinone bait consumption, survival and residues in weta

Pilot trial

All four weta survived the trial, appearing healthy and responding normally to disturbance throughout the 44 days. All had nibbled the Ditrac bait by day 4. Consumption (or at least removal) of bait by weta was shown by the presence of distinctive scrapes on the bait surface and bait crumbs on the dish. Bait consumption or removal continued steadily until about day 15–20, after which it appeared to level off. Mould was first observed on the surface of the baits approximately one month after they were placed in the housing units, and covered the bait surface more extensively as the trial progressed. One of the weta continued to eat the mouldy bait, as evidenced by the presence of crumbs, although the other three did not leave any evidence of interference with bait once the mould was present. The bait blocks lost a mean of 1.6 ± 0.6 g over the trial. Laboratory analysis showed that the Ditrac blocks contained 52.5 μg g^{-1} (52.5 ppm) diphacinone, slightly higher than the nominal 0.005% by weight. The analysed concentration of diphacinone in bait was used to estimate a mean cumulative intake of 113.04 ±21.79 μg diphacinone eaten or at least removed by each of the four weta over the trial, without accounting for changes in bait weight due to environmental moisture. The frass of the four weta feeding on diphacinone baits was lighter coloured and often had a striped appearance compared with that from weta feeding on plant food only. Three of the four weta gained weight over the 44 days they had access to Ditrac bait, but one female lost approximately 60% of starting bodyweight over this time.

Main trial

All weta left some evidence of bait consumption or removal by day 8, but none had completely eaten the bait by the end of the trial. There was an increase in the amount of bait consumed or removed over time (weighted regression slope = 0.376, SE = 0.114, t_{24} = 3.29, P = 0.003), with a corresponding increase in the estimated consumption of diphacinone by each group of weta sampled at intervals up to day 64 (Fig. 1).

Spiked samples analysed alongside the whole weta yielded estimates of 86, 82 and 76% recovery of diphacinone. The method detection limit for diphacinone in invertebrate tissue was 0.2 µg g^{-1}, with an uncertainty (95% CI) of ±20%.

There was a small, but significant decrease in the residual concentration of diphacinone in the weta over time (weighted regression slope = −0.0441, SE = 0.0123, t_{24} = 3.59, P = 0.002). The mean (± SE) concentrations of diphacinone in weta showed a slight increase from weta sampled on day 1 (3.63 ± 1.59 µg g^{-1}) to those sampled on day 4 (4.85 ± 0.73 µg g^{-1}), but thereafter declined gradually to reach 0.99 ± 0.51 µg g^{-1} by day 64 (Fig. 1). Using the bodyweight of each weta at sampling to estimate the total amount of residual diphacinone contained in a weta gave mean (± SE) figures of 11.44 ± 0.88 µg diphacinone (day 1), 14.42 ± 4.36 µg (day 4), 9.91 ± 5.06 µg (day 8), 8.06 ± 11.68 µg (day 16), 6.27 ± 0.92 µg (day 31) and 3.64 ± 1.74 µg (day 64). The most residual diphacinone calculated to be present in a single weta was 24.99 µg, in a 4.23-g female sampled on day 4.

Exposure to diphacinone baits did not appear to adversely affect weta bodyweight. In fact, weight gain was greater during the trial phase than during acclimatisation (difference in slopes = 0.0194, SE_{diff} = 0.0086, t_{100} = 2.26, P = 0.026), although relative growth rates (g g^{-1} day^{-1}) showed less difference between the acclimatisation and trial phases (difference in slopes = 0.0002, SE_{diff} = 0.0001, t_{183} = 1.82, P = 0.072).

Three male weta died during the trial period – two were found dead on day 8 of sampling, with the other noted to be unresponsive on day 8 and checked and found dead on day 9. Weights of the dead weta were nearly half those recorded at the beginning of the trial. All three weta had consumed bait during the trial period, equivalent to 31.0, 29.5 and 8.09 µg diphacinone per gram bodyweight. Respective residual diphacinone concentrations in these weta were 7.9, 3.6 and 2.2 µg g^{-1}. These weta were excluded from the overall statistical analyses because the cause of their death was not certain. The amounts of bait these weta had consumed before they died were similar to those measured for other weta that remained healthy throughout the trial. Although the residual concentration in one of the dead weta was slightly higher (7.9 µg g^{-1}) than the next highest residual diphacinone concentration measured in the trial (6.2 µg g^{-1} in a female sampled on day 8), the residual concentrations of the other two dead weta were well within the range measured in surviving weta. Total whole-body residues were not calculated for the three dead weta because of the significant weight loss they underwent over a relatively short period of time and uncertainty about the actual date of death.

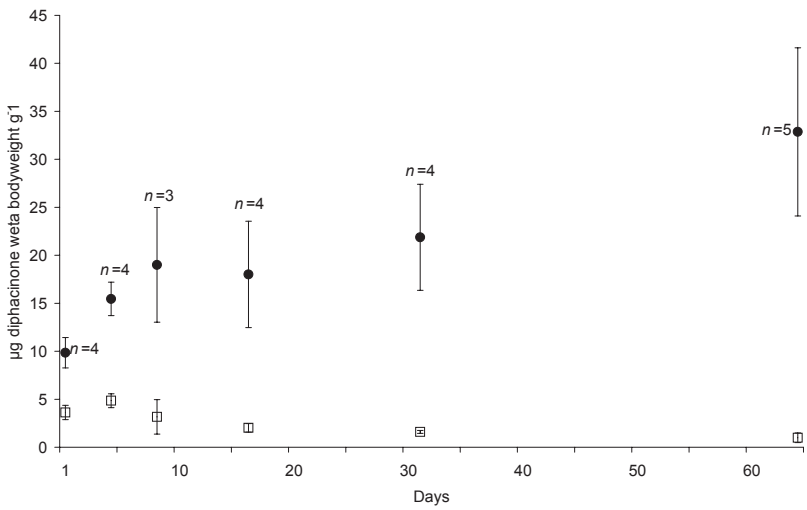

Figure 1. Mean (± SE) estimated diphacinone intake by groups of weta over time from consumption of Ditrac block baits (µg diphacinone per gram weta bodyweight) (●), and corresponding measured diphacinone residues in the whole body of the weta (□).

Discussion

A wide range of New Zealand invertebrate species, including weta, have been reported to eat cereal-based baits in field conditions (Sherley et al. 1999; Spurr & Drew 1999; Lloyd & McQueen 2000; Spurr 2000; Spurr & Berben 2004). Captive weta in our study ate Ditrac wax block bait in the presence of alternative natural plant food. This suggests that Ditrac wax block bait would offer a palatable food source to opportunistically foraging weta in the field. A field study in the North Island showed that weta were attracted to cereal-based brodifacoum bait in bait stations, and spent considerable time in contact with the baits (Craddock 2003). After the initial finding of baits by captive weta in our study, steady consumption over time suggests they were returning nightly to feed on an identified palatable food source. In some field control operations, removal of baits by weta and other invertebrates feeding on baits might be expected to hasten the physical degradation of baits, potentially decreasing their acceptability and availability to target pest animals.

No adverse effects of the consumption of the Ditrac wax block baits were detected in weta during our trial. This finding adds to recent studies indicating that insects in general have a much lower susceptibility to anticoagulants than mammals. Craddock (2003) found that captive locusts (*Locusta migratoria*) fed readily on cereal-based brodifacoum baits with no significant increase in mortality. Bowie and Ross (2006) found no significant difference in weight loss of captive cave weta (*Pleioplectron simplex*) or ground weta (*Hemiandrus* sp.) offered brodifacoum bait for 60 days compared with weta offered non-toxic bait. Although mortality appears unlikely in weta feeding on diphacinone or brodifacoum bait, the absorption, distribution, metabolism and excretion of anticoagulant rodenticides in invertebrates are poorly described. While the vitamin-K-dependent carboxylation reactions that produce blood coagulation factors are affected by anticoagulant toxicity in mammalian liver, vitamin-K-dependent metabolic processes also occur in other tissues (Vermeer et al. 1992), and these carboxylase enzyme systems are generally distributed in invertebrate systems (Walker et al. 2001). Caution should be used in extrapolating a general lack of acute effects of anticoagulants in arthropod species to nil effect in other invertebrates, or to nil effect on the long-term reproductive fitness of arthropods exposed to baits. For example, there is limited evidence for mortality in molluscs (e.g. Gerlach & Florens 2000; Primus et al. 2005) and earthworms (Booth et al. 2003) following relatively high environmental exposures to brodifacoum.

Weta eating Ditrac bait over time did not accumulate diphacinone beyond a maximum of 7.9 μg g^{-1}, i.e. whole-body concentrations did not increase with the amount of diphacinone eaten. This suggests a saturation body burden, where bait material in the gut plus any diphacinone absorbed and distributed in tissues was metabolised and/or excreted as quickly as more was ingested. Weta in the day 1 sample group were estimated to have consumed or removed bait in quantities from 12 to 27% of their bodyweight within 24 hours, suggesting that their gut was rapidly filled with bait material in various stages of digestion. Although the extent to which weta absorb and metabolise diphacinone is not known, most of the residual diphacinone detected was probably in the gut contents. In terms of estimating secondary non-target risks (see below), the distribution of residues in weta tissues is probably irrelevant as predators or scavengers are likely to eat the whole insect, or at least the abdomen. The change in colour of the frass of weta eating diphacinone bait suggests a substantial change in diet composition. If weta excrete diphacinone relatively rapidly and without extensive metabolism, they have the potential to distribute residual diphacinone in the leaf litter and soil surface via their frass.

Birds appear to be less susceptible than mammals to single doses of diphacinone; the lowest LD_{50} value (i.e. the single oral dose of diphacinone expected to cause death in 50% of a population) reported for a bird species is > 400 mg kg^{-1} in northern bobwhite quail (*Colinus virginianus*), and the lowest LC_{50} (i.e. the concentration of diphacinone in food that can be expected to cause the death of 50% of a population) is 906 μg g^{-1} (95% CI 187–35 107 μg g^{-1}) for mallards (*Anas platyrhynchos*) (US EPA 1998). The highest weta residue concentration detected in this trial (7.9 μg g^{-1}) was approximately six times less toxic than the diphacinone concentration in the Ditrac bait blocks, and 23 times less than the lower 95% confidence interval for the mallard dietary toxicity value (LC_{50}). On paper, such concentrations of diphacinone in weta represent a very low secondary hazard to birds; a 20-g bird would need to consume more than 10 kg of contaminated weta in a single feed to ingest 400 mg kg^{-1} diphacinone (as a conservative LD_{50} estimate for birds). While acute secondary diphacinone toxicity in birds that feed on weta seems highly unlikely on the basis of this simplistic 'risk of mortality' calculation, the toxicity of diphacinone in multiple rather than single intakes and the possibility of adverse sublethal effects on birds require consideration. As for other first-generation anticoagulants, the toxicity of diphacinone to mammals is enhanced by multiple, consecutive oral doses in comparison with single oral doses (US EPA 1998), and this could also be the case for birds. These aspects of the secondary risk assessment for diphacinone remain largely unquantified, and investigation in terms of lethal or sublethal outcomes in birds feeding regularly on diphacinone bait or contaminated weta is warranted.

Persistence of residues in invertebrates is an important determinant of the likelihood of secondary exposure. We located no published reports of the persistence of diphacinone in invertebrates. However, published studies indicate that brodifacoum residues are not as persistent in invertebrate tissues as they are in mammalian or avian liver. Following sublethal doses, brodifacoum residues were not detectable after 4 days in captive weta (Booth et al. 2001) and after one month in land crabs (Pain et al. 2000). Captive locusts excreted brodifacoum rapidly, indicating that long-term bioaccumulation was unlikely (Craddock 2003). However, a field-based study showed that brodifacoum residues in invertebrates took more than 4 weeks to return to background levels after brodifacoum bait was removed from bait stations, with trace concentrations of brodifacoum still detectable up to 10 weeks after the bait had been removed (Craddock 2003). Brodifacoum residues were found in both the gut and foot tissue of common garden snails 14 days after they were exposed to soil containing ground bait at 2 mg brodifacoum per kilogram of soil (Booth et al. 2003).

Our study did not set out to assess the persistence of residual diphacinone in weta after exposure to diphacinone bait ceased, although on the basis of the residual concentrations measured in weta during this study, they would probably excrete diphacinone within a period of days. Comparative pharmacokinetics, an important basis for formulating assessments of risk to non-target species and minimising environmental effects, has traditionally focused on vertebrate eutherian species. New Zealand biodiversity is characterised by its native avian, reptilian, and invertebrate species, and the use of toxic baits is a mainstay of current management strategies for vertebrate pests that threaten biodiversity values. In this context, a comparative study of the persistence of brodifacoum and diphacinone residues in tree weta would be useful to further quantify the relative risks of applying bait formulations for vertebrate pest control.

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