 DOI: 10.20417/nzjecol.40.43

Acute toxicity and risk to lizards of rodenticides and herbicides commonly used in New Zealand

Scott M. Weir[1,5*], Shuangying Yu[1,6], Ami Knox[2,7], Larry G. Talent[3], Joanne M. Monks[4] and Christopher J. Salice[1,8]

[1]The Institute of Environmental and Human Health, Department of Environmental Toxicology, Texas Tech University, 1207 S Gilbert Dr, Lubbock, TX 79416, USA
[2]University Studies, Texas Tech University, 2500 Broadway, Lubbock, TX 79409, USA
[3]Department of Natural Resources Ecology and Management, Oklahoma State University, Stillwater, OK 74074, USA
[4]Science and Policy Group, Department of Conservation, PO Box 5244, Moray Place, Dunedin 9058, New Zealand
[5]Present address: Department of Biology, Queens University of Charlotte, 1900 Selwyn Ave, Mail Stop 1264, Charlotte, NC 28207, USA
[6]Present address: Department of Environmental Science and Chemistry, Queens University of Charlotte, 1900 Selwyn Ave, Mail Stop 1264, Charlotte, NC 28207, USA
[7]Present address: Research Integrity Office, Texas Tech University Health Sciences Center, Texas Tech University, Lubbock, TX 79409, USA
[8]Present address: Environmental Science and Studies, Department of Biological Sciences, Towson University, Towson, Maryland 21252, USA
*Author for correspondence: (Email: weirs@queens.edu)

Published online: 13 May 2016

Abstract: Invasive species can have negative consequences on native reptile populations, especially on island systems. Chemical control can be a cost-effective way to control or eradicate invasive species. Chemical control is currently in use in New Zealand to limit impacts of non-native mammals and plants on a range of native biodiversity. However, it is important to consider the potential non-target risks of chemical control to native species that are likely already significantly reduced in number. We aimed to characterise the toxicity of several rodenticides and herbicides to reptiles and to provide a screening-level risk assessment of these chemicals applicable to native reptiles of New Zealand using the western fence lizard, *Sceloporus occidentalis*, as a surrogate organism. We used the Up-and-Down testing procedure to estimate oral toxicity for all compounds. We tested five rodenticides (brodifacoum, coumatetralyl, pindone, diphacinone and cholecalciferol). Only pindone was toxic to fence lizards at concentrations below 1750 μg g^{-1} (LD50 = 550 μg g^{-1}). We tested five herbicides (glyphosate, clopyralid, triclopyr, metsulfuron-methyl and haloxyfop-methyl) and one common adjuvant in glyphosate formulations (polyethoxylated tallowamine or POEA). Only triclopyr was toxic to fence lizards below 1750 μg g^{-1} (LD50 = 550 μg g^{-1}). Toxicity does not necessarily imply risk. Using the pindone concentrations in accepted bait formulations in New Zealand, a 10 g lizard would need to ingest 4.7 g of pindone bait in a single day in order to achieve toxic levels, which is extremely unlikely. We used the highest acceptable application rate for triclopyr to estimate risk for reptiles and found minimal risk of acute toxicity from triclopyr applications. Taken together, our data suggest little risk of reptile acute toxicity from the tested rodenticides or herbicides in New Zealand, but research into sub-lethal effects is also required in order to make informed decisions about the ecological impacts of chemically controlling invasive species.

Key Words: ecological risk assessment; ecotoxicology; LD50; pesticides; reptile

Introduction

Reptiles are declining globally and invasive species are considered to be one of several factors contributing to these declines (Gibbons et al. 2000). Reptiles inhabiting islands are particularly affected by the invasion of small mammals (Nogales et al. 2006). On the islands of New Zealand, invasive species have been a major issue, with invasive mammals causing declines in many native taxa including reptiles (Nelson et al. 2014). Rodenticides are applied to entire offshore islands to eradicate introduced mammals (especially rodents) from these systems (Towns & Broome 2003) and at the landscape level on New Zealand's main islands to protect biodiversity (e.g. Innes et al. 2010; Reardon et al. 2012). Specifically in New Zealand, anti-coagulant rodenticides have been used to control invasive mammals (e.g. brodifacoum; see Eason & Spurr 1995). In addition, herbicides are used throughout New Zealand's islands and, outside of agricultural uses, are often used to control invasive plant species. However, it is important to ensure that the chemicals used to control invasive species do not also negatively impact reptile populations, especially considering that greater than 50% of native New Zealand reptiles are declining or threatened (Hitchmough et al. 2013).

Chemical contaminants are an additional stressor that may be contributing to reptile declines (Gibbons et al. 2000). Reptiles remain the least studied vertebrate group relative to other terrestrial vertebrates in ecotoxicology (Sparling et al. 2010). Previous research has found that contaminant ecological risk assessments are difficult to perform for reptiles. In general, toxicity data are lacking for reptiles and current

understanding of contaminant exposure is minimal (Weir et al. 2010). In the United States, federal regulations for pesticide registrations (such as the Federal Insecticide Fungicide and Rodenticide Act) do not require testing of reptiles for pesticide registration, and this is generally the case worldwide. For terrestrial herpetofauna, results for avian species are considered representative of both amphibians and reptiles (USEPA 2004). However, there are notable instances in which reptiles have greater sensitivity to contaminants than birds or mammals (Weir et al. 2010). Therefore, research is needed to quantify the toxicity of pesticides to reptiles in order to adequately assess their ecological risk.

The effect of the rodenticides and herbicides used in New Zealand on native reptiles is difficult to determine based on currently available data (e.g. Hoare & Hare 2006a). There is evidence to suggest that New Zealand reptiles will ingest bait (Freeman et al. 1996; Hoare & Hare 2006b; Marshall & Jewell 2007; Wedding et al. 2010), and it is likely that a secondary poisoning pathway exists via their invertebrate prey (Erickson & Urban 2004); so two exposure pathways may exist for many rodenticide baits. In this paper we present an experiment that aims to quantify potential acute toxicity of several rodenticides and herbicides to lizards. Toxicity will be quantified as LD50s, defined as the median lethal dose, which represents the dose expected to cause 50% mortality in a group of individuals. We then estimate ecological risk of acute mortality of the chosen pesticides to lizards. Risk is determined by comparing the LD50s to exposure models for oral exposure. If exposure exceeds toxicity, this represents risk.

Materials and methods

Study organisms

We used the western fence lizard (*Sceloporus occidentalis*) as a surrogate species for native New Zealand reptiles. The western fence lizard has been previously vetted as a model organism for reptile ecotoxicology research (Talent et al. 2002; Suski et al. 2008). Further, the fence lizard is a relatively small lizard (12–30 g used in the current study) that feeds primarily on small invertebrates, and thus provides a reasonable biological model for similar sized native New Zealand reptiles. We are not aware of any physiological mechanisms that would cause New Zealand lizards to be significantly more sensitive to pesticides, although data are greatly lacking for reptiles in general (Weir et al. 2010). Mechanistic research on metabolic enzymes and other physiological factors that affect toxicity are very rare for reptiles. Taken together, we believe that the western fence lizard represents an adequate surrogate for New Zealand lizards (despite evolutionary distance) given the paucity of available model reptile species.

Adult male western fence lizards were acquired from the western fence lizard colony maintained at Oklahoma State University. Once received, all lizards were maintained in animal holding facilities approved by the Animal Care and Use Committee at Texas Tech University. Lizards were held individually in plastic containers measuring 11 cm deep × 15.5 cm wide × 28.5 cm long and were provided 1 kg of washed playground sand in each container as substrate. Lizards were also provided a small water dish (10 mL volume) for *ad libitum* drinking. Lizards were fed two large mealworms (approximate weight = 0.15 g each) every other day prior to initiation of experiments. The lizards were maintained on a 14:10 light dark cycle and a heat lamp was provided for 6 hours each day for thermoregulation. The heat lamp created a gradient of approximately 26–34°C in the container when the lamps were on. When the lamps were off, temperatures were maintained at 22 ± 4°C. These temperatures are higher than the average temperatures throughout New Zealand (especially in the south); however, they were optimised for the western fence lizard and also represent standard temperatures for studies of acute toxicity for vertebrate organisms. We discuss the importance of temperature in toxicity (see Discussion) and emphasise that investigating the role of temperature in toxicity should be an important future research focus, particularly for reptiles.

Acute toxicity experiments

Oral exposure was conducted as a pseudo-gavage and is generally intended to represent exposures in the field resulting from consumption of contaminated food items. The method has been described previously (Suski et al. 2008; Salice et al. 2009). Rather than intubate the lizards, repeatable and accurate dosing can be achieved with a standard #5 gelatin capsule (Torpac Inc.) and it is less stressful for the organism (Salice et al. 2009; Weir et al. 2015). Another advantage of using gelatin capsules for dose administration is that no carrier solvent is required thus minimising potential absorptive effects of the carrier. The oral dosing method requires two researchers, one to firmly hold the lizard and to pull gently but firmly on the dewlap to slowly open the lizard's mouth. The second researcher then administers the dose by pushing the capsule far into the throat of the lizard to prevent the lizard from regurgitating the capsule after administration. Gelatin capsules were filled with a known mass of the pesticide of interest. The mass of pesticide was based on the weight of the lizard to achieve a body mass-specific dose ($\mu g\ g^{-1}$).

We chose five rodenticides and five herbicides for investigation of oral acute toxicity. The rodenticides chosen were brodifacoum, coumatetralyl, diphacinone, pindone and cholecalciferol. These rodenticides are currently registered and commonly used for control of rodents in New Zealand. The commonly used toxin 1080 (sodium monofluoroacetate) was not tested because it has been already evaluated for toxicity to Australian reptiles and based on evidence from a range of taxa there is little concern about lethal effects in reptiles (McIlroy et al. 1985). Brodifacoum and coumatetralyl represent coumarin (or anticoagulant) rodenticides. Coumatetralyl is a traditional coumarin in that it requires multiple feedings to achieve toxicity, while brodifacoum is a second-generation anticoagulant with single-dose toxicity (Ware & Whitacre 2004). Pindone and diphacinone represent indandione rodenticides (also anticoagulant). Pindone requires multiple feedings while diphacinone could be considered single-dose (Ware & Whitacre 2004) or multi-dose (Erickson & Urban 2004). Cholecalciferol (also known as Vitamin D_3) is a newer rodenticide that does not belong to any specific mode of action group.

The herbicides chosen were glyphosate, triclopyr, clopyralid, metsulfuron-methyl and haloxyfop-methyl. Haloxyfop-methyl is an aryloxphenoxy propionate herbicide, glyphosate is a phosphono amino acid herbicide, triclopyr and clopyralid represent carboxylic acid herbicides, and metsulfuron-methyl is a sulfonylurea herbicide. These herbicides are currently registered for use in New Zealand. We also tested the toxicity of polyethoxylated tallowamine (POEA), which is a surfactant added to many varieties of Roundup, a common formulation of glyphosate. We tested both oral and dermal toxicity of POEA as it is a liquid at room temperature and no solvent was needed to test dermal toxicity. Roundup

toxicity to amphibians has been well established, as well as very low toxicity from glyphosate itself (Mann & Bidwell 1999). It is generally believed that the toxicity of Roundup to amphibians is due to the presence of POEA (Howe et al. 2004), as is the case with aquatic invertebrates and fish (Giesy et al. 2000). We acquired pure pesticides (purity ≥98% in all cases) and POEA from Chemservice (Westchester, PA, USA).

We modified the Up-and-Down Procedure (UDP) for estimating LD50s (OECD 2008). The UDP has produced LD50 estimates very similar to those of traditional dose-response LD50 designs (Lipnick et al. 1995), but uses considerably fewer animals (a maximum of 15 for the UDP compared to 40 or more for traditional LD50). Briefly, the UDP methodology encompasses dosing one individual at a time and changing the dose for the next individual as a result of short-term (e.g. 48 h or 96 h) outcomes. For example, if an organism is dosed at 55 μg g^{-1} and survives, the next dose is 175 μg g^{-1}. If the individual had died at 55 μg g^{-1}, the next dose would have been 17.5 μg g^{-1}. This process is repeated until one of the stopping criteria is met. Therefore, the number of lizards dosed may be different between pesticides. All individuals surviving the first 4 days are observed for 14 days, and LD50 calculations are based on mortality at 14 days.

We employed the standard UDP approach with minimal modifications. First, for what we defined as short-term results, we observed lizards for 96 h rather than the standard 48 h, because reptiles have a slower metabolism than birds and mammals. Second, we selected 1750 μg g^{-1} as our highest dose rather than the standard 2000 μg g^{-1} or 5000 μg g^{-1}, in an attempt to reduce the number of lizards used in experiments. Because no previous knowledge of the toxicity of these pesticides is available for reptiles, we used the standard UDP doses of 5.5 μg g^{-1}, 17.5 μg g^{-1}, 55 μg g^{-1}, 175 μg g^{-1}, 550 μg g^{-1} and 1750 μg g^{-1}. Lizards were observed for a 1-week acclimation period prior to the initiation of toxicity testing. Prior to dosing, lizards were housed as described above. After receiving a single dose via pseudo gavage, lizards were not provided a basking lamp (to decrease variability associated with metabolic rate and subsequent effects on toxicity) during the 14-day observation period and food was withheld for the first 96 hours of observation. All lizards were given the same standard doses (ranging from 5.5 to 1750 μg g^{-1}) as outlined in OECD guidelines (OECD 2008); therefore, resulting LD50 estimates can often be very similar as the same doses are used for all pesticides tested (see Weir et al. 2015).

The standard short-term response for toxicity is 48 hours; however, lizards have a slower metabolism than birds and mammals, so the observation period was extended. Longer observation may be especially important for some of the rodenticides tested, which may exhibit a lag between dosing and mortality (e.g. brodifacoum; Littin et al. 2000). Any lizard that survived the 96 h short term observation period was then observed for an additional 10 days to score long-term response to the pesticide. After the initial 96 h observation period, lizards were provided one mealworm every other day until the entire 14-day observation period was completed. Lizards were provided less food to decrease the potential for food/toxicity interactions and because many lizards at very high doses displayed food avoidance.

Following the 14-day observation period, all remaining lizards were euthanised according to approved methods using CO_2 exposure followed by decapitation (AVMA 2013). After decapitation, lizard carcasses were retained for later chemical analysis and were frozen at -80°C until extraction and analysis. All activities were approved by the Animal Care and Use Committee of Texas Tech University (#13012-01).

We calculated LD50s using AOT25STATPRG software developed by Westat for the USEPA (software available at: www.epa.gov/oppfead1/harmonization/docs/AOT425Setup.exe). In general, for the UPD, LD50s are calculated using maximum likelihood methods, for full details see OECD (2008).

Risk estimation

To place the rodenticide and herbicide LD50s into context, we estimated risk using two different methods for each pesticide type. For rodenticides we compared estimated daily feeding requirements to the mass of bait necessary to achieve toxicity. Our toxicity threshold was the lower confidence limit of the LD50 for each rodenticide or 1750 μg g^{-1} (the highest dose tested) if toxicity did not occur in our experiments. We assumed our toxicity estimates were applicable to native New Zealand reptiles in the absence of any other data for comparison. The USEPA Wildlife Exposure Factors Handbook provides a formula for estimating daily ingestion rates for reptiles:

$$IR = 0.013 \times BW^{0.773} \quad (1)$$

where IR is ingestion rate in g and BW is lizard body mass (g) (USEPA 1993). We estimated the mass of bait needed to elicit toxicity in 10 to 80 g lizards using the bait concentration as well as the toxicity threshold. We chose this weight range as most of the threatened reptiles in New Zealand fall within this range (Hoare et al. 2007). As an example, if the LD50 is 100 μg g^{-1}, a 10 g lizard would need to ingest 1000 μg of the rodenticide. If the bait concentration is 50 μg g^{-1}, this equates to 20 g of bait ingested to achieve toxicity. Bait concentrations came from New Zealand Department of Conservation reports listing formulations with a range of active ingredients (e.g. 170–500 μg g^{-1} for pindone; Fairweather & Fisher 2012). We estimated risk by comparing the daily feeding requirements of a 10 g lizard (approximately 0.077 g from the ingestion rate formula), to the mass of bait needed to elicit toxicity. There is a high likelihood that lizards could ingest more than the estimated daily feeding requirement. For example, lizards in this study regularly consumed 200–300 mg of food (two mealworms, every two days).

The formulae that the USEPA uses to estimate feeding rates are averaged to daily estimates, which is logical for birds and mammals that commonly feed every day. Reptiles may not have daily feeding events, so a 300 mg feeding event once during a 3-day period will average out to 100 mg per day. Because this theoretical reptile only ate once during a 3-day period, the 100 mg per day estimate actually underestimates the mass of a single feeding event. Therefore, we have also estimated risk assuming the lizard eats an optimal amount of food every day. An optimal food quantity was calculated using the 300 mg value of two mealworms for a 20 g lizard. The estimated food ingestion rate for a 20 g lizard is approximately 132 mg per day. We then used the ratio of 300 mg to 132 mg for a 20 g lizard and increased all food ingestion estimates for all lizards by the same factor (2.27), which provides an estimate of 'optimum feeding'. We calculated feeding requirements for one day and seven days with both methods. If more than 100% of daily feeding is needed to exceed toxicity thresholds, this suggests that the risk of acute toxicity is low as our models made very conservative assumptions (e.g. the lizard eats only contaminated bait, the lizard eats every day for 7 days, etc.).

For herbicides, we used previous methods (Weir et al. 2010)

to perform a screening level risk assessment of toxicity from a single herbicide application. Herbicide exposure was estimated using the standard USEPA methods for terrestrial exposure (i.e. TREX software). The TREX software uses the Kenaga Nomogram to estimate residues on common food items based on a specified application rate. We used a standard application rate (1.12 kg ha^{-1}) that is common to commercial formulations of herbicides and also represents a high-end application rate for the herbicides used in these experiments. A standard allometric field metabolic rate averaged for all reptiles is available in the USEPA Wildlife Exposure Factors Handbook (USEPA 1993). The daily dietary exposure is estimated as:

$$\text{Dose } (\mu g\ g^{-1}\ BW\ d^{-1}) = ([FMR / 1.7] \times 45) / BW \quad (2)$$

where FMR is field metabolic rate (kcal per day), BW is lizard body mass (g), 45 (μg g^{-1}) is the pesticide residue on the food item (small insects in this case) assuming a standard 1.12 kg ha^{-1} application rate, and 1.7 (kcal g^{-1}) is the assumed calorie value of small insects. Dose estimates are allometrically scaled to body mass, and we estimated exposure for reptiles ranging in body mass from 10–80 g. Exposure estimates were compared to toxicity data from the current study to estimate risk to create a risk quotient (exposure/toxicity). A risk quotient >1 suggests a potential for an adverse effect and that risk of toxicity from estimated exposure cannot be precluded (USEPA 2004). We used the lower confidence limit of the estimated LD50s for each herbicide, if toxicity did not occur at doses up to 1750 μg g^{-1} we assumed the LD50 was 1750 μg g^{-1}. Therefore, these risk estimates are conservative because the true toxicity value for these herbicides was >1750 μg g^{-1}.

Results

Few of the pesticides we tested were toxic to fence lizards below 1750 μg g^{-1} over the entire 14-day observation period (Table 1). Of the five rodenticides tested, only pindone caused toxicity below 1750 μg g^{-1} with an LD50 of 550 μg g^{-1} (235.4–778 μg g^{-1} 95% CI). All other rodenticides were essentially non-toxic to reptiles. Diphacinone caused some mortality at 1750 μg g^{-1} (2 out of 5 lizards) suggesting the LD50 is near 1750 μg g^{-1}. A similar result was found for the herbicides tested. Only triclopyr caused toxicity below 1750 μg g^{-1}, with an LD50 of 550 μg g^{-1} (228.6–664 μg g^{-1} 95% CI). All other herbicides and surfactants caused no toxicity at doses up to 1750 μg g^{-1}.

The risk of acute toxicity appears to be very low, even for compounds that elicited toxicity in our experiments. Our food ingestion rate models suggest that lizards would need to consume bait in excess of 300% of their daily food requirements to exceed the lower limit LD50 estimated for pindone (235.4 μg g^{-1}) even under assumptions of very high feeding rates (0.3 g per day for a 20 g lizard, Table 2). Using the field ingestion rate (0.077 g per day), lizards would need to consume bait >800% of their daily food requirements to reach the lower limit LD50. Because toxicity estimates (i.e. LD50s) are very high for all herbicides (even triclopyr, relatively speaking), risk estimates (i.e. risk quotients) for field applications are very low (<0.02 for triclopyr, <0.002 for other herbicides) and varied little across body masses (Table 3).

Discussion

The toxicity of the rodenticides and herbicides to fence lizards tested in these experiments was generally very low with two exceptions (pindone for rodenticides, triclopyr for herbicides). The low toxicity of most pesticides from these two classes is not surprising, as neither group is designed to be toxic to ectothermic vertebrates. Rodenticides are designed to target endothermic organisms and generally have a mechanism of action that is most effective for endothermic physiology. Many rodenticides have a long half-life in the target organism and toxicity may take many days to manifest. Because reptiles have a much slower metabolism than mammals (Nagy et al. 1999), it may take longer for toxicity to occur in reptiles than mammals given the same dose.

Because rodenticides target endothermic organisms, the

Table 1. Summary of LD50s (μg g^{-1}) for rodenticides and herbicides for western fence lizards (as a surrogate organism). CI represents 95% confidence intervals calculated by the Westat software available from the USEPA (see text).

Group	Chemical	n	Carrier	Mean lizard mass (g)	LD50	CI
Rodenticides	Pindone	10	Capsule	16.36	550.0	235.4 - 778
	Diphacinone	5	Capsule	19.59	~1750[a]	-
	Cholecalciferol (Vit D_3)	3	Capsule	17.05	> 1750	-
	Coumatetralyl	3	Capsule	16.59	> 1750	-
	Brodifacoum	3	Capsule	17.45	> 1750	-
Herbicides	Glyphosate	3	Capsule	20.31	> 1750	-
	POEA - Oral	3	Capsule	20.51	> 1750	-
	POEA - Dermal	3	N/A[b]	21.38	> 1750	-
	Clopyralid	3	Capsule	23.09	> 1750	-
	Triclopyr	12	Capsule	23.39	550.0	228.6 - 664
	Haloxyfop-methyl	3	Acetone[c]	15.59	> 1750	-
	Metsulfuron-methyl	3	Capsule	20.35	> 1750	-

[a]Two out of five lizards exposed to 1750 μg g^{-1} diphacinone died, which did not require a full LD50 test, but suggests the LD50 is near 1750 μg g^{-1}.
[b]POEA was provided as a liquid at room temperature so no solvent was used in the dermal toxicity test.
[c]Haloxyfop-methyl was provided as a liquid at room temperature. Such a small volume of liquid was provided that it was exceedingly difficult to measure out accurate weights of the pure haloxyfop-methyl into capsules. Therefore, acetone was added to the liquid haloxyfop-methyl to provide accurate doses.

Table 2. Risk summary for acute mortality of rodenticides to lizards. Risk is reported as a proportion of daily feeding needed to reach the LD50 (lower confidence limit). For the rodenticides other than pindone, toxicity did not occur with doses up to 1750 μg g^{-1}, so the LD50 is an overestimate of toxicity based on our highest dose. Further, for brevity, we provided risk estimates for only the 10 g lizards for these rodenticides as risk was always highest for 10 g lizards. For each rodenticide, we provided the greatest bait concentration permitted for use in New Zealand. A proportion greater than 100% suggests little risk as toxicity to lizards could not be achieved by feeding exclusively on bait. Risk estimates are provided using standard USEPA daily ingestion rate models as well as 'optimal food' ingestion rates which represents known quantities of food ingested by the lizards in our experiments (see text for details).

Compound[a]	Weight (g)	LD50 (μg g^{-1})	Bait (μg g^{-1})	g Bait = mortality	% 1 d FIR	% 7 d FIR	% 1 d Optimal Food	% 7 d Optimal Food
Pindone	10	235.4	500	4.7	6108	873	2691	384
	20	235.4	500	9.4	7149	1021	3149	450
	40	235.4	500	18.8	8367	1195	3686	527
	80	235.4	500	37.7	9793	1399	4314	616
Brodifacoum	10	1750	50	350.0	454072	64867	200032	28576
Diphacinone	10	1750	300	58.3	75679	10811	33339	4763
Coumatetralyl	10	1750	500	35.0	45407	6487	20003	2858
Cholecalciferol	10	1750	8000	2.2	2838	405	1250	179

[a] Sources for bait concentration are: pindone (Fairweather & Fisher 2012), brodifacoum (Broome et al. 2012a), diphacinone (Broome & Fisher 2012), coumatetralyl (Broome et al. 2012b), cholecalciferol (Fairweather & Fisher 2011).

Table 3. Summary of risk to lizards from herbicide applications. Other herbicides are glyphosate, clopyralid, haloxyfop-methyl and metsulfuron-methyl. FMR refers to field metabolic rate and is calculated by standard metabolic rates averaged across reptiles (USEPA 1993). The EED is the expected environmental dose and is a function of FMR, residue on food items (45 μg g^{-1} at 1.12 kg ha^{-1} application rate), and the estimated caloric content of the food item (1.7 kcal g^{-1}, see text for details). RQ represents risk quotient and is the EED divided by the LD50 (lower confidence limit). An RQ >1 suggests significant risk of acute toxicity. For 'other herbicides' toxicity did not occur up to 1750 μg g^{-1} in our experiments and we assumed toxicity was equal to 1750 μg g^{-1} to be conservative.

Compound	Weight (g)	LD50 (μg g^{-1})	FMR	EED (μg g^{-1})	RQ
Triclopyr	10	228.6	0.36	2.77	0.012
	20	228.6	0.67	2.57	0.011
	40	228.6	1.24	2.38	0.010
	80	228.6	2.30	2.20	0.010
Other herbicides	10	1750	0.36	2.77	0.002
	20	1750	0.67	2.57	0.001
	40	1750	1.24	2.38	0.001
	80	1750	2.30	2.20	0.001

biochemical and physiological processes necessary to achieve toxicity will be very different between reptiles and mammals (e.g. 1080 toxicity; McIlroy 1986). There does not seem to be a consistent pattern in rodenticide toxicity to reptiles based on number of feeding attempts needed to elicit toxicity (e.g. multiple or single feedings). However, there may be a pattern of indandione rodenticides showing greater toxicity than the coumarin derivative rodenticides. Only pindone and diphacinone appeared to cause acute toxicity to fence lizards. If diphacinone is defined as a multi-dose rodenticide (e.g. Erickson & Urban 2004) then there may be a pattern of multi-dose rodenticides having higher toxicity to reptiles than single-dose. There was never an indication of toxicity from coumarin derivative rodenticides. More research is needed to determine if this observation results from consistently greater toxicity of indandione rodenticides to reptiles. There may be a mechanistic explanation for greater toxicity of indandione rodenticides that is currently unknown. There is very little previous research of the toxicity of rodenticides to reptiles. However, Brooks et al. (1998) report preliminary toxicity of diphacinone to brown tree snakes (*Boiga irregularis*) and report toxicity occurring at doses of 20 (1 of 5 snakes), 40 (3 of 5 snakes), and 80 μg g^{-1} (5 of 5 snakes). Why Brooks et al. (1998) found higher toxicity than we did in our study is currently unknown. A potential confounding factor was that Brooks et al. (1998) used a carrier solvent (propylene glycol) for dosing while we used capsules without a carrier solvent. Perhaps the solvent facilitated uptake of diphacinone or altered toxicity via some other mechanism (Weir et al. 2015). An additional important consideration in comparing our data to those of Brooks et al. (1998) is inter-species differences in sensitivity. McIlroy et al. (1985) reported that larger predator/scavenger reptiles were more sensitive to 1080 than other reptiles. Our results have a similar pattern in which our smaller lizard was less sensitive than the larger brown tree snakes used by Brooks et al. (1998). Future research with diphacinone is warranted to determine how common reptile sensitivity to diphacinone occurs, and the mechanism for the noticeable differences between our data and those of Brooks et al. (1998). Size-based differences may be especially important for New Zealand reptiles considering

the large differences between tuatara (*Sphenodon punctatus*) and other smaller native lizards.

Many herbicides have plant-specific modes of action that do not affect animal physiologic processes. An example is glyphosate, the active ingredient in Roundup brand herbicides. The mode of action for glyphosate is inhibition of essential amino acid synthesis. The biochemical pathway appears to be unique to plants and some microorganisms, and is generally of low toxicity to animals (Giesy et al. 2000). However, preliminary research on glyphosates and their adjuvants suggests herbicides used in New Zealand can have measurable physiological effects on native lizards (Carpenter et al. 2016). As such, research into potential sub-lethal effects of rodenticides and herbicides on reptiles is warranted. As an example, an important sub-lethal effect of rodenticides could be coagulopathy (a condition in which the blood's ability to clot is impaired) which may not directly result in mortality, but when combined with another stressor (e.g. being attacked by a predator) may combine to result in lethal excess bleeding or hemorrhaging (Rattner et al. 2011). The toxicity of triclopyr to lizards was somewhat surprising. Triclopyr is a carboxylic acid herbicide (also known as pyridinoxy and picolinic acid herbicides), which generally has a hormone mimic mode of action (Ware & Whitacre 2004). Because the hormones are not found in animal cells, these herbicides generally do not have high toxicity to animals. It is important to note that while we recorded lizard toxicity from triclopyr, the toxicity values were still quite high (LD50 = 550 μg g^{-1}) and are probably environmentally unrealistic under normal application scenarios.

Our risk models, which included estimates of exposure, suggest little risk of acute toxicity of our chosen pesticides to lizards. The only rodenticide that was toxic at experimental doses (pindone) would require a 10 g lizard to ingest 4.7 g of bait. It is unlikely that the stomach of a 10 g lizard could accommodate 4.7 g of bait. Given that most native New Zealand reptiles experience much cooler temperatures than the 23°C used in our experiments (except during the warmest summer months), a 10 g lizard ingesting 4.7 g of bait is even more unlikely. For example, Freeman et al. (1996) provided pindone baits to skinks and allowed *ad libitum* feeding on the bait. The highest mass of bait ingested by a single lizard was 0.14 g and the mean weight of the skinks was 2.8 g. Taken together, the available information indicates that it is very unlikely that a lizard could ingest enough bait to achieve toxicity with pindone.

In most cases it seems that toxicity from a single feeding event is practically impossible because sufficient doses will not be reached. Brodifacoum is almost non-toxic to reptiles (LD50 >1750 μg g^{-1}). Assuming the LD50 is 1750 μg g^{-1}, then 17500 μg is needed to achieve toxicity in a 10 g lizard. Brodifacoum rodenticide formulations approved in New Zealand have a range of proportion of active ingredients from 20–50 μg g^{-1} (Broome et al. 2012a). A 10 g lizard would have to ingest 350 g of the 50 μg g^{-1} bait to achieve a dose of 1750 μg g^{-1}. Nevertheless, our data are preliminary, and future experimental work with longer observation periods, multiple doses, and/or the presence of a basking lamp may provide additional insights.

All risk estimates for herbicides were much lower than 1 (all <0.02) and so are unlikely to result in observable adverse effects in realistic environments. To provide some context, in order to achieve exposure levels that would create even moderate risk of acute toxicity, application rates would need to be more than 50-times greater than our assumed application rate (1.12 kg ha^{-1}). The assumed application rate of 1.12 kg ha^{-1} may itself be higher than most application rates for the herbicides of interest. While our risk estimate only takes dietary exposure into account, including other routes of exposure would likely not increase risk estimates to significant levels (Weir et al. 2010).

It is well established that the toxicity of compounds to ectothermic organisms can be highly dependent on temperature (Cairns et al. 1975). The effect of temperature is dependent on the mode of action of a given pesticide. For example, pesticides that require metabolic activation (e.g. some organophosphorous insecticides) will be expected to be more toxic at higher temperatures due to increased metabolism (Lydy et al. 1999). Conversely, pesticides that do not require metabolic activation and are broken down and excreted by metabolic processes (e.g. some pyrethroid insecticides) are expected to be less toxic at higher temperatures (Sparks et al. 1983). The toxicity of pyrethrins (the natural product pyrethroid insecticides are derived from) to Carolina anoles (*Anolis carolinensis*) is highly dependent on temperature, with lower temperatures causing a significant increase in toxicity (Talent 2005). We exposed fence lizards to pesticides under controlled standard laboratory temperatures (22 ± 4°C) to aid comparisons with other standard test data, but these temperatures are higher than those experienced by most New Zealand reptiles. We have previously shown that the presence of a heat lamp following dosing can significantly alter toxicity estimates (Weir et al. 2015). Some of the compounds tested in the current study (e.g. brodifacoum) have long half-lives (up to months) and may circulate in rodents for several days before toxicity manifests (Vandenbrouke et al. 2008). For reptiles, the length of time needed for toxicity to occur will be much longer than mammals due to decreased metabolism and absorption of the chemical following exposure.

The length of the post-dose observation period may be important for some of the pesticides we investigated. For example, while brodifacoum can exert toxicity following a single feeding event, mortality can lag significantly following the feeding event. Reports of time-to-death (in days) of some mammals following ingestion of toxic doses of brodifacoum were 5.6–8.5 for Norway rats (*Rattus norvegicus*), 14.9–45.3 for brushtail possum (*Trichosurus vulpecula*) (Littin et al. 2000), and 2–18 for rabbits (*Oryctolagus cuniculus*; Godfrey et al. 1981). If it is assumed that metabolic rate can play a role in manifestation of toxicity (in addition to toxicokinetic/toxicodynamic causes), toxicity may manifest in our lizards after 14 days of exposure. It might be prudent for future researchers to consider longer observational periods for pesticides with a known lag in the onset of toxicity. Similarly, for compounds that require multiple feedings to elicit toxicity (e.g. coumatetralyl), toxicity may be greatly underestimated using single dose toxicity estimates (Vyas & Rattner 2012).

Finally, in an attempt to contextualise our reptile toxicity data against more widely available data, we provide a summary of the available mammal and avian toxicity data for the chosen pesticides in comparison to reptiles (Table 4). Not surprisingly, the mammalian LD50s for rodenticides are generally much lower (greater toxicity) compared to reptiles. Pindone is the only rodenticide for which reptilian toxicity is even remotely close to mammalian toxicity. In addition, pindone represents the least toxic rodenticide to mammals of the five we tested. Results were more varied for birds with some rodenticides being highly toxic (e.g. brodifacoum) and some not very toxic

Table 4. Summary of available toxicity data (LD50 in μg g^{-1}) for reptiles, birds, and mammals for the chosen pesticides. Reptile data are from the current study; no other sources of reptile toxicity data were available for these pesticides, except diphacinone. Brooks et al. (1998) report an oral LD50 of approximately 40 μg g^{-1} for brown tree snakes exposed to diphacinone. Sources are provided for bird and mammal data and are generally based on large toxicity databases as many of the LD50s reported here could be traced back to registration requirements for the USA. If two or more LD50s were available (n ≥2) for a pesticide, the mean LD50 is reported and the range of the LD50s is also provided.

Chemical	Reptile	n	Range	Bird	Range	n	Source	Mammal	Range	n	Source[a]
Brodifacoum	> 1750	1	-[b]	3.24	0.26-11	3	1	5.43	0.2-25	5	1
Diphacinone	895	2	40 - 1750	1724.7	96.8-3158	4	1,2,3	2.45	2.3-2.6	2	1
Coumatetralyl	> 1750	1	-	> 2000	-	1	4	16.5	-	1	4
Cholecalciferol	> 1750	1	-	> 2000	-	1	5	13	-	1	4
Pindone	550	1	-	241	-	1	1	78.43	10.3-150	3	4
Glyphosate	> 1750	1	-	> 3851	-	1	1	3763	1658-5600	5	4
POEA	> 1750	1	-	N/A[c]	-	-	-	N/A	-	-	-
Clopyralid	> 1750	1	-	> 2000	-	1	1	4300	-	1	4
Triclopyr	550	1	-	1698	-	1	1	729	-	1	4
Haloxyfop-methyl	> 1750	1	-	> 2150	-	1	1	393	-	1	6
Metsulfuron-methyl	> 1750	1	-	> 2510	-	1	1	> 5000	-	1	4

[a]1: Pesticide Ecotoxicity Database (PED 2014), 2: Rattner et al. 2010, 3: Rattner et al. 2011, 4: Hazardous Substances Data Bank (HSDB 2011), 5: Pesticide Properties DataBase (University of Hertfordshire 2013), 6: EXTOXNET (1995).
[b]Range or sample size data are not available because insufficient toxicity data were found for this pesticide.
[c]N/A = toxicity data was not found for this taxon for this compound.

at all (e.g. coumatetralyl). Avian and reptilian sensitivity was also similar for pindone. For the herbicide data, many of the LD50s reported for mammals and birds were greater than our highest tested dose. Therefore, it is not known how reptiles and mammals compare for many of the herbicides studied. Perhaps surprisingly, the toxicity of triclopyr was greater (lower LD50) for reptiles than birds/mammals. More toxicity data across a wider range of tested doses is needed to determine how often reptiles and mammals have similar toxicity to herbicides.

The results of this research suggest that the rodenticides and herbicides used on public lands in New Zealand appear to pose little acute risk of mortality to reptiles. The one exception may be the use of pindone, as reptile and mammalian toxicity is similar, and pindone usually requires multiple doses to control rodents. More research is needed to determine the toxicity of pindone to reptiles from multiple doses and, in order to more accurately estimate risk, a more thorough exposure regime is needed for pindone that can incorporate estimates of bait take (similar to Jessop et al. 2013). Importantly, we have not considered chronic exposure nor sub-lethal toxicity (e.g. Carpenter et al. 2016). Our data represent a first attempt to quantify the toxicity of several pesticides to reptiles, but more data are needed to ensure that native New Zealand reptiles are adequately protected from the use of pesticides to control invasive species. However, relative to the danger posed by invasive species, the positive aspects of using rodenticides likely outweigh the potential toxicity to reptiles (e.g. Brown 1997).

Acknowledgements

SM Weir thanks the Helen Jones Foundation and the Texas Tech University Office of the Provost for financial support. We thank the New Zealand Lizard Technical Advisory Group and Clayson Howell for commenting on the study design. This study was partially funded by the New Zealand Department of Conservation (Science Investigation No. 4491). We thank Jo Carpenter and an anonymous reviewer for helpful comments that greatly improved the manuscript.

References

AVMA 2013. AVMA guidelines for the euthanasia of animals: 2013 edition. www.avma.org/KB/Policies/Pages/Euthanasia-Guidelines.aspx (accessed 20 April 2016).

Brooks JE, Savarie PJ, Johnston JJ 1998. The oral and dermal toxicity of selected chemicals to brown tree snakes (*Boiga irregularis*). Wildlife Research 25: 427–435.

Broome KG, Fisher P 2012. Diphacinone pesticide information review. Version 2012/1. Unpublished report docdm-25450. Hamilton, New Zealand, Department of Conservation. 53 p.

Broome KG, Fairweather AAC, Fisher P 2012a. Brodifacoum pesticide information review. Version 2012/2. Unpublished report docdm-25439. Hamilton, New Zealand, Department of Conservation. 110 p.

Broome KG, Fairweather AAC, Fisher P 2012b. Coumatetralyl pesticide information review. Version 2012/2. Unpublished report docdm-25444. Hamilton, New Zealand, Department of Conservation. 37 p.

Brown D 1997. Chetwode Island kiore and weka eradication project. Ecological Management 5: 11–20.

Cairns J Jr, Heath AG, Parker BC 1975. Temperature influence on chemical toxicity to aquatic organisms. Journal (Water Pollution Control Federation) 47: 267–280.

Carpenter JK, Monks JM, Nelson N 2016. The effect of two glyphosate formulations on a small, diurnal lizard (*Oligosoma polychroma*). Ecotoxicology 25: 548–554.

Eason CT, Spurr EB 1995. Review of the toxicity and impacts of brodifacoum on non-target wildlife in New Zealand. New Zealand Journal of Zoology 22: 371–379.

Erickson W, Urban D 2004. Potential risks of nine rodenticides to birds and nontarget mammals: a comparative approach. Environmental Fate and Effects Division, Office of

Pesticides Programs, United States Environmental Protection Agency. 230 p.
EXTOXNET 1995. Haloxyfop. pesticide information project. Primary files maintained by Oregon State University, OR, USA. http://extoxnet.orst.edu/pips/haloxyfo.htm (accessed 20 April 2016).
Fairweather AAC, Fisher P 2011. Cholecalciferol pesticide information review. Version 2011/1. Unpublished report docdm-25415. Hamilton, New Zealand, Department of Conservation. 38 p.
Fairweather AAC, Fisher P 2012. Pindone pesticide information review. Version 2012/1. Unpublished report docdm-124982. Hamilton, New Zealand, Department of Conservation. 48 p.
Freeman AB, Hickling GJ, Bannock CA 1996. Response of the skink *Oligosoma maccanni* (Reptilia: Lacertilia) to two vertebrate pest-control baits. Wildlife Research 23: 511–516.
Gibbons WJ, Scott DE, Ryan TJ, Buhlmann KA, Tuberville TD, Metts BS, Greene JL, Mills T, Leiden Y, Poppy S, Winne CT 2000. The global decline of reptiles, déjà vu amphibians. Bioscience 50: 653–666.
Giesy JP, Dobson S, Solomon KR 2000. Ecotoxicological risk assessment for Roundup herbicide. Reviews of Environmental Contamination and Toxicology 167: 35–120.
Godfrey MER, Reid TC, McAllum HJF 1981. The oral toxicity of brodifacoum to rabbits. New Zealand Journal of Experimental Agriculture 9: 23–25.
HSDB 2011. TOXNET: toxicology data network, National Library of Medicine, National Institute of Health, Bethesda, MA, USA. http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB (accessed 20 April 2016).
Hitchmough R, Anderson P, Barr B, Monks J, Lettink M, Reardon J, Tocher M, Whitaker T 2013. Conservation status of New Zealand reptiles, 2012. Wellington, New Zealand, Department of Conservation. 16 p.
Hoare JM, Hare KM 2006a. *Hoplodactylus maculatus* (common gecko) toxin consumption. Herpetological Review 37: 86–87.
Hoare JM, Hare KM 2006b. The impact of brodifacoum on non-target wildlife: gaps in knowledge. New Zealand Journal of Ecology 30: 157–167.
Hoare JM, Adams LK, Bull LS, Towns DR 2007. Attempting to manage complex predator-prey interactions fails to avert imminent extinction of a threatened New Zealand skink population. Journal of Wildlife Management 71: 1576–1584.
Howe CM, Berrill M, Pauli BD, Helbing CC, Werry K, Veldhoen N 2004. Toxicity of glyphosate-based pesticides to four North American frog species. Environmental Toxicology and Chemistry 23: 1928–1938.
Innes J, Kelly D, Overton JM, Gillies C 2010. Predation and other factors currently limiting New Zealand forest birds. New Zealand Journal of Ecology 34: 86–114.
Jessop TM, Kearney MR, Moore JL, Lockwood T, Johnston M 2013. Evaluating and predicting risk to a large reptile (*Varanus varius*) from feral cat baiting protocols. Biological Invasions 15: 1653–1663.
Lipnick RL, Cotruvo JA, Hill RN, Bruce RD, Stitzel KA, Walker AP, Chu I, Goddard M, Segal L, Springer JA, Myers RC 1995. Comparison of the Up-and-Down, conventional LD50, and Fixed-Dose acute toxicity procedures. Food and Chemical Toxicology 33: 223–231.
Littin KE, O'Connor CE, Eason CT 2000. Comparative effects of brodifacoum on rats and possums. New Zealand Plant Protection 53: 310–315.
Lydy MJ, Belden JB, Ternes MA 1999. Effects of temperature on the toxicity of M-parathion, chlorpyrifos, and pentachlorobenzene to *Chironomus tentans*. Archives of Environmental Contamination and Toxicology 37: 542–547.
Mann RM, Bidwell JR 1999. The toxicity of glyphosate and several glyphosate formulations to four species of southwestern Australian frogs. Archives of Environmental Contamination and Toxicology 36: 193–199.
Marshall JE, Jewell T 2007. Consumption of non-toxic baits used for pest mammal control by grand (*Oligosoma grande*) and Otago (*O. otagense*) skinks, and their prey. DOC Research & Development Series 272. Wellington, New Zealand, Department of Conservation. 11 p.
McIlroy JC 1986. The sensitivity of Australian animals to 1080 poison. IX. Comparisons between the major groups of animals, and the potential danger nontarget species face from 1080 poisoning campaigns. Australian Wildlife Research 13: 39–48.
McIlroy JC, King DR, Oliver AJ 1985. The sensitivity of Australian animals to 1080 poison. VIII. Amphibians and reptiles. Australian Wildlife Research 12: 113–118.
Nagy KA, Girard IA, Brown TK 1999. Energetics of free-ranging mammals, reptiles, and birds. Annual Review of Nutrition 19: 247–277.
Nelson NJ, Hitchmough RA, Monks JM 2014. New Zealand reptiles and their conservation. In: Stow A, Holwell G, Maclean N eds Austral ark. Cambridge, UK, Canterbury University Press. Pp. 382–404.
Nogales M, Rodriguez-Luengo JL, Marrero P 2006. Ecological effects and distribution of invasive non-native mammals on the Canary Islands. Mammal Review 36: 49–65.
OECD 2008. Acute oral toxicity – Up-and-Down procedure (UDP). OECD Guidelines for the Testing of Chemicals 425. Paris, France, OECD Publishing. 27 p.
PED 2014. National information system. Regional Centers for Integrated Pest Management, United States Department of Agriculture. www.ipmcenters.org/Ecotox/DataAccess.cfm (accessed 20 April 2016).
Rattner BA, Horak KE, Warner SE, Johnston JJ 2010. Acute toxicity of diphacinone in Northern bobwhite: effects on clotting and survival. Ecotoxicology and Environmental Safety 73: 1159–1164.
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.
Reardon JT, Whitmore N, Holmes KM, Judd LM, Hutcheon AD, Norbury G, Mackenzie DI 2012. Predator control allows critically endangered lizards to recover on mainland New Zealand. New Zealand Journal of Ecology 36: 141–150.
Salice CJ, Suski JG, Bazar MA, Talent LG 2009. Effects of inorganic lead on western fence lizards (*Sceloporus occidentalis*). Environmental Pollution 157: 3457–3464.
Sparks TC, Pavloff AM, Rose RL, Clower DF 1983. Temperature-toxicity relationships of pyrethroids on *Heliothis virescens* (F) (Lepidoptera: Noctuidae) and *Anthonomus grandis grandis* Boheman (Coleoptera:

Curculionidae). Journal of Economic Entomology 76: 243–246.
Sparling DW, Linder G, Bishop CA, Krest SK 2010. Recent advancements in amphibian and reptile ecotoxicology. In: Sparling DW, Linder G, Bishop CA eds Ecotoxicology of amphibians and reptiles. 2nd edn. Pensacola, FL, USA, SETAC Press. Pp. 1–13.
Suski JG, Salice C, Houpt JT, Bazar MA, Talent LG 2008. Dose-related effects following oral exposure of 2,4-dinitrotoluene on the western fence lizard, *Sceloporus occidentalis*. Environmental Toxicology and Chemistry 27: 352–359.
Talent LG 2005. Effect of temperature on toxicity of a natural pyrethrin pesticide to green anole lizards (*Anolis carolinensis*). Environmental Toxicology and Chemistry 24: 3113–3116.
Talent LG, Dumont JN, Bantle JA, Janz DM, Talent SG 2002. Evaluation of western fence lizard (*Sceloporus occidentalis*) and eastern fence lizard (*Sceloporus undulatus*) as laboratory reptile models for toxicological investigations. Environmental Toxicology and Chemistry 21: 899–905.
Towns DR, Broome KG 2003. From small Maria to massive Campbell: forty years of rat eradications from New Zealand islands. New Zealand Journal of Ecology 30: 377–398.
University of Hertfordshire 2013. The pesticide properties database (PPDB) developed by the Agriculture & Environment Research Unit (AERU), University of Hertfordshire, 2006-2013. http://sitem.herts.ac.uk/aeru/ppdb/en/ (accessed 20 April 2016).
USEPA 1993. Wildlife exposure factors handbook volume 1 of 2. Report # EPA-600-R93-187. Washington, DC, USA, Office of Research and Development. 572 p.
USEPA 2004. Overview of the ecological risk assessment process in the Office of Pesticide Programs, U.S. Environmental Protection Agency. Endangered and threatened species effects determination. Washington, D.C, Office of pesticide prevention, pesticides, and toxic substances, Office of Pesticide Programs. 92 p.
Vandenbrouke V, Bousquet-Melou A, De Backer P, Croubels S 2008. Pharmacokinetics of eight anticoagulant rodenticides in mice after a single oral administration. Journal of Veterinary Pharmacology and Therapeutics 31: 437–445.
Vyas NB, Rattner BA 2012. Critique on the use of the standardized avian acute oral toxicity test for first generation anticoagulant rodenticides. Human and Ecological Risk Assessment 18: 1069–1077.
Ware GW, Whitacre DM 2004. The Pesticide Book, 6th edition. Willoughby, OH, USA. MeisterPro Information Resources. 488 p.
Wedding CJ, Ji W, Brunton DH 2010. Implications of visitation by shore skinks *Oligosoma smithi* to bait stations containing brodifacoum in a dune system in New Zealand. Pacific Conservation Biology 16: 86–91.
Weir SM, Suski JG, Salice CJ 2010. Ecological risk of anthropogenic pollutants to reptiles: evaluating assumptions of sensitivity and exposure. Environmental Pollution 158: 3596–3606.
Weir SM, Yu S, Talent LG, Maul JD, Anderson TA, Salice CJ 2015. Improving reptile risk assessment: oral and dermal toxicity of pesticides to a common lizard species (*Sceloporus occidentalis*). Environmental Toxicology and Chemistry 34: 1778–1786.

Editorial board member: Colin O'Donnell
Received 19 August 2015; accepted 18 March 2016