New Zealand Veterinary Journal

Te Hautaka Tākuta Kararehe o Aotearoa

ISSN: 0048-0169 (Print) 1176-0710 (Online) Journal homepage: www.tandfonline.com/journals/tnzv20

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To cite this article: S Mehta, L Booth, S Hunter, M Jolly & BD Gartrell (04 May 2025): Suspected brodifacoum poisoning in tuatara (*Sphenodon punctatus*), New Zealand Veterinary Journal, DOI: 10.1080/00480169.2025.2491498

To link to this article: https://doi.org/10.1080/00480169.2025.2491498

Published online: 04 May 2025.

NEW ZEALAND VETERINARY JOURNAL
https://doi.org/10.1080/00480169.2025.2491498

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Suspected brodifacoum poisoning in tuatara (*Sphenodon punctatus*)

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ABSTRACT

Case history: Between June 2017 and April 2019, three captive tuatara from a zoological facility in the South Island of New Zealand were found unwell and admitted to veterinary care. One other tuatara from the same facility was found dead from misadventure in May 2019.
Clinical findings: All three unwell tuatara showed clinical signs of lethargy, mucous membrane pallor, and dehydration, with haematoma formation/swelling in dependent parts of the body. Fine needle aspiration and cytology of the swellings showed common features of peripheral blood, with variable other cytological findings. Haematology confirmed marked anaemia in Case 1 (PCV 5%; reference range 22–53%) and Case 2 (PCV 1%) and suspected mild anaemia in Case 3 (PCV 27%). Case 1 died 6 weeks after initial presentation, whereas Cases 2 and 3 died soon after presentation.
Pathological findings: Post-mortem examination showed general pallor of soft tissues in the three tuatara with clinical signs of coagulopathy. There was haemorrhage in the bladder wall of Case 1, while Cases 2 and 3 had haematomas (subcutaneous in Case 2 and peri-oesophageal in Case 3). The pathological diagnosis in Case 4 was death by asphyxiation following burrow collapse. Retrospective analysis showed brodifacoum was present in liver tissue at a concentration of 0.26 mg/kg in Case 3, and in skeletal muscle tissue at concentrations of 0.019 mg/kg in Case 2 and 0.035 mg/kg in the non-clinical case (Case 4).
Diagnosis: The clinical signs and post-mortem findings were consistent with anticoagulant poisoning in three tuatara, and tissue concentrations of brodifacoum demonstrated exposure in three animals, including one animal with no clinical signs of coagulopathy (Case 4). Definitive diagnosis was prevented, however, by inconsistent toxicology testing and a limited understanding of toxicity thresholds in reptiles in general, and tuatara specifically.
Clinical relevance: This case series suggests that tuatara are susceptible to anticoagulant poisoning and this has implications for both the captive management of tuatara, and also the use of rodenticides in tuatara habitat, such as offshore islands and mainland sanctuaries.

Abbreviations: AR: Anticoagulant rodenticide; LD50: Median lethal dose; SGAR: Second generation anticoagulant rodenticide

ARTICLE HISTORY
Received 30 October 2024
Accepted 3 April 2025

KEYWORDS
Anticoagulant; coagulopathy; reptile; Sphenodontidae; tuatara; wildlife pathology

Introduction

Tuatara (*Sphenodon punctatus*) is a unique reptile species native to New Zealand. It is the sole surviving member of the reptilian order Rhynchocephalia (Jones *et al.* 2009; Gemmell and Tollis 2020). Tuatara populations were once distributed throughout New Zealand. Since human settlement, tuatara populations have declined constantly to the extent that they now occupy < 0.5% of their original range (Nelson *et al.* 2002; Ramstad *et al.* 2007) and were totally eliminated from the mainland in the eighteenth century (Gaze 2001). Conservation management has led to the re-establishment of tuatara populations in some of their historic habitats (Gaze 2001). These efforts have led to an increase in the population, which is currently estimated to be around 100,000 individuals, concentrated on 39 offshore islands along the east coast of the North Island, from the Bay of Plenty to Northland, and some of the islands in the Cook Strait. Tuatara can also be found in various fenced wildlife sanctuaries and zoological institutions around the New Zealand mainland (Gaze 2001; Towns *et al.* 2007; Jones *et al.* 2009).

Rats (*Rattus* spp.) prey on the eggs and juveniles of tuatara and compete with the adults for food (Cree 2014). The increase in the tuatara population can be primarily attributed to the eradication of rats from the offshore islands by the use of anticoagulant rodenticides (AR), which include first generation anticoagulant rodenticides such as warfarin, pindone and coumatetralyl, and second generation anticoagulant rodenticides (SGAR) such as difenacoum, bromadiolone, brodifacoum and flocoumafen (Eason *et al.* 2002; Towns and Broome 2003; Brakes and Smith 2005). These SGAR are more potent and persistent

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than their first generation predecessors (Brakes and Smith 2005). The most extensively used SGAR is brodifacoum, which was developed in the mid-1970s and has been used for the eradication of rats and other mammalian predators on offshore islands, wildlife sanctuaries and national parks in New Zealand (Eason and Spurr 1995; Eason *et al.* 2002; Hoare and Hare 2006). The Department of Conservation has used brodifacoum extensively for island rodent eradications but stopped using it for mainland control operations in 2002 due to concerns about its persistence in the environment (Eason *et al.* 2002). However, it continues to be used extensively on the mainland by regional councils, community groups and private landowners (Sran *et al.* 2023).

Anticoagulant rodenticides work by inhibiting the recycling of vitamin K1. Vitamin K1 is a cofactor required for post-ribosomal carboxylation (activation) of blood clotting factors (factor II (prothrombin), factor VII, factor IX, and factor X) by the enzyme vitamin K-dependent carboxylase. AR inhibit vitamin K epoxide reductase, which normally catalyses the recycling of inactive vitamin K1 epoxide to active vitamin K1 hydroquinone. This leads to a lack of active vitamin K1 and thus inhibits the activation of blood clotting factors. In mammals, the activity of clotting factors decreases within about 12–24 hours after intoxication, coinciding with the first massive bleeding episodes (Valchev *et al.* 2008). The most potent of all SGAR is brodifacoum, which causes an approximately 100-times greater decrease in vitamin K-dependent coagulation factors than warfarin (Murphy 2018). However, resistance to brodifacoum has recently been reported in New Zealand rat populations (Sran *et al.* 2023).

Anticoagulant poisoning in reptiles may occur by two different pathways: primary or secondary (Eason and Spurr 1995). Primary poisoning occurs when an individual of a non-target species ingests the bait directly from the ground or bait stations. There are a few reported incidences of primary brodifacoum poisoning in reptiles, including the death of King's skinks (*Egernia kingii*; Bettink 2015), Galápagos land iguanas (*Conolophus subcristatus*; Harper *et al.* 2011), and Telfair's skinks (*Leiolopisma telfairii*; Merton 1987). There have also been reports where reptiles were observed to consume baits directly, but no mortality was observed; for example, Wright's skinks (*Mabuya wrightii*; Thorsen *et al.* 2000), Raukawa geckos (*Woodworthia maculata*; Hoare and Hare 2006), Duvaucel's gecko (*Hoplodactylus duvaucelii*; Christmas 1995), shore skinks (*Oligosoma smithi*; Wedding *et al.* 2010), bobtails (*Tiliqua rugosa*; Lohr and Davis 2018), and moko skinks (*Oligosoma moco*; Mauldin *et al.* 2020).

Most of the reptile species in New Zealand are insectivorous, including tuatara, and may be at risk of secondary poisoning by feeding on invertebrates or, in the case of tuatara, small rodents, that have fed on brodifacoum baits (Eason and Spurr 1995). There have been no previous reports of secondary poisoning in reptiles by feeding on invertebrates other than an anecdotal report of the deaths of two tuatara in captivity with suspected brodifacoum poisoning (R. Griffiths, unpublished data cited in Fisher *et al.* 2010). Recent studies have shown that some reptiles are less susceptible to anticoagulant toxicity than birds and mammals. For example, in a controlled experiment, Western fence lizards (*Sceloporus occidentalis*) given up to 1,750 mg/kg of brodifacoum orally showed no symptoms of acute toxicity (Weir *et al.* 2016), while Mauldin *et al.* (2020) found that turtles and boas exhibited a relative insensitivity to diphacinone and brodifacoum and lizards appeared to be somewhat more sensitive to these compounds.

The typical clinical signs of brodifacoum poisoning in vertebrates may include mild to severe coagulopathy with prolonged bleeding times, haemoptysis, ecchymosis, melaena, haematuria, and haematomas. Loss of blood may lead to dyspnoea, tachycardia, hypotension, and multiple organ failure in mammals (Woody *et al.* 1992). Sub-lethal effects of brodifacoum are difficult to determine, especially in wild animals (Brakes and Smith 2005). The effect of anticoagulant rodenticides on reptiles has not been studied as much as in mammals, either as target or non-target species (Lohr and Davis 2018). The basic mechanism of clot formation in reptiles is similar to that of the other vertebrate orders (Gentry 2004) but blood clotting times are slower in reptiles than birds and mammals both *in vivo* and *in vitro* (Zain-ul-Abedin and Katorski 1966; Hackett and Hann 1967; Kubalek *et al.* 2002).

In this article we report the findings of cases of suspected brodifacoum poisoning in three tuatara from a single institution and a fourth tuatara with evidence of exposure but no clinical signs of coagulopathy.

Case history

Three cases were identified with clinical and post-mortem findings consistent with anticoagulant poisoning from the Wildlife Pathology database at Massey University (Palmerston North, NZ) between June 2017 and April 2019. All three of these animals were tuatara held at a captive zoological institution in the South Island of New Zealand. A fourth tuatara from the same institution was found dead due to misadventure in May 2019 and was included in the investigation to assess possible exposure to brodifacoum.

Further case information regarding these four cases was gathered by reviewing veterinary hospital records from the Halifax Veterinary Centre (Nelson, NZ) and Wildbase Hospital (Massey University) where the initial three cases had been treated, post-mortem

Table 1. Relevant signalment, history, clinical and post-mortem findings, and tissue brodifacoum concentrations in four tuatara (*Sphenodon punctatus*).

Case	Hospital	Admission date	Age (years)	Sex	History	Clinical findings	Date of death	Post-mortem findings	Brodifacoum concentration (mg/g)
1	Halifax[a]	June 2017	Unknown	M	Recurring phalangeal osteomyelitis. Swelling on left side.	Dehydration, pale MM, prolonged bleeding, anaemia (PCV 5%), thrombocytopenia, mild heterophilia and hypoproteinaemia.	4/7/2017	Pallor of soft tissues, bladder wall haemorrhage, haematuria	Not assessed
2	Halifax[a]	5/3/2018	8	F	Lethargy and right chest swelling.	Anaemia (PCV 1%) with marked reticulocytosis, SC haematoma	9/3/2018	Pallor of soft tissues, SC haematomas	0.019 (skeletal muscle)
3	Wildbase[b]	4/4/2019	8	F	Sub-mandibular swelling with pale oral MM	Mild anaemia (PCV 27%), submandibular haematoma	5/4/2019	Pallor of soft tissues, peri-oesophageal and submandibular haematomas	0.26 (liver)
4	NA	NA	24	F	Found dead with head buried under a rock	Not applicable	9/5/2019	No evidence of coagulopathy	0.035 (skeletal muscle)

[a]Halifax Veterinary Centre, Nelson, NZ.
[b]Wildbase Hospital, Massey University, Palmerston North, NZ.
F = female; M = male; MM = mucous membranes; NA = not applicable; PCV = packed cell volume.

findings, laboratory tests and communication with keeping staff (Table 1). Three cases (1–3) had a history of identified illness, while Case 4 was found dead in the enclosure with no previous clinical signs of illness and was submitted to Wildbase Pathology (Massey University) directly for post-mortem examination.

Brodifacoum baits (Pestoff Rodent Baits; Orillion, Whanganui, New Zealand) had been used in bait stations around the captive institution, and within 10 m of the tuatara enclosures, continuously from 2013 until the diagnosis of suspected brodifacoum poisoning in Case 3 in 2019. At no time had baits been used at all within the enclosures.

Clinical findings

Relevant clinical findings for the four tuatara are summarised in Table 1. All three unwell tuatara showed clinical signs of lethargy, mucous membrane pallor, and dehydration, with haematoma formation/swelling in dependent parts of the body (Figure 1). Fine needle aspiration and cytology of the swellings showed common features of peripheral blood, with variable other cytological findings. Haematology confirmed marked anaemia in Case 1 (PCV 5%; reference range 22–53%) and Case 2 (PCV 1%) and mild anaemia in Case 3 (PCV 27%) (Table 2). As the diagnosis of anticoagulant poisoning was only reached retrospectively, the tuatara only received supportive care consisting of SC

Figure 1. Ante-mortem photograph showing submandibular swelling (arrow) on a tuatara (*Sphenodon punctatus*) (Case 3) that presented with clinical signs and findings consistent with brodifacoum poisoning.

Table 2. Haematology and biochemistry values of two tuatara (*Sphenodon punctatus*; Cases 1 and 3) with coagulopathy due to suspected brodifacoum poisoning.

Parameter	Case 1	Case 3	Reference range[a]
Packed cell volume (%)	5	27	22–53%
Haemoglobin (g/L)	10	–	45–91 g/L
White blood count (x 10^9/L)	17.4	4.6	1.2–21×10^9/L
Lymphocytes			
Absolute (x 10^9/L)	3.8	0.5	
Percentage	22	10	
Monocytes			
Absolute (x 10^9/L)	0.5	0.2	
Percentage	3	4	
Eosinophils			
Absolute (x 10^9/L)	0.9	1.3	
Percentage	5	28	
Heterophils			
Absolute (x 10^9/L)	12.2	2.6	
Percentage	70	56	
Total protein (g/L)	23	34	18.7–58.4 g/L
Aspartate aminotransferase (IU/L)	32	20	4–112 IU/L
Creatine kinase (IU/L)	911	1,033	
Calcium (mmol/L)	2.01	3.15	1.57–5.79 mmol/L
Phosphorus (mmol/L)	0.74	2.11	0.96–3.36 mmol/L
Glucose (mmol/L)	3.3	7.9	4.6–13.9 mmol/L
Uric acid (µmol/L)	110	169	13–500 µmol/L

[a]From Blanchard (2002).

fluids and thermal support. Case 1 died 6 weeks after initial presentation, whereas Cases 2 and 3 died soon after presentation.

Pathological findings

Post-mortem examination and histopathology was carried out by the Wildlife Pathology service at Massey University. The relevant post-mortem findings are summarised in Table 1. In summary, post-mortem examination showed general pallor of soft tissues in the three clinically affected tuatara. There was haemorrhage in the bladder wall in one tuatara (Case 1), and haematomas in two tuatara (SC in Case 2 and perioesophageal in Case 3) (Figure 2 and Figure 3). The pathological diagnosis in Case 4 was death by asphyxiation following burrow collapse.

Tissue samples were collected at post-mortem examination and stored frozen at −20°C. Brodifacoum tissue assays were carried out retrospectively on Cases 2, 3 and 4 by the Toxicology Laboratory at Manaaki Whenua – Landcare Research (Lincoln, NZ). No tissue sample was available for Case 1. The method was based on that of Jones (1996). Briefly, tissue was diced and a sub-sample was mixed with anhydrous sodium sulphate, then extracted with chloroform/acetone/ammonia. Extracts were evaporated, taken up in chloroform/hexane and cleaned up by solid-phase extraction on an aminopropyl column. The analyte was eluted from the column using 0.005 M tetrabutylammonium phosphate in methanol, evaporated and taken up in a mobile phase of methanol/water/acetic acid, for high-performance liquid chromatography analysis. The minimum detectable limit of this method was 0.001 mg/kg and all results are presented as wet weight.

Liver tissue from Case 3 had a brodifacoum concentration of 0.260 mg/kg and muscle tissue from Case 2 and Case 4 had brodifacoum concentrations of 0.019 mg/kg and 0.035 mg/kg respectively (Table 1).

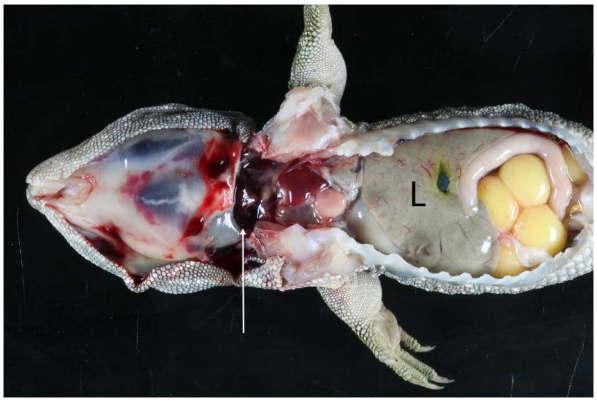

Figure 2. Post-mortem photograph of a tuatara (*Sphenodon punctatus*) (Case 3) that presented with clinical signs and findings consistent with brodifacoum poisoning showing extensive haemorrhage in the submandibular area, around the glottis and trachea (arrow). The pallor of the liver (L) is evident.

Discussion

In this case series, we have demonstrated an association between the deaths of three tuatara with severe coagulopathy and confirmed exposure to brodifacoum in two of these three animals. There was no evidence of systemic disease or trauma to otherwise explain the coagulopathy. The caveat to these results is that a fourth tuatara that had similar tissue concentrations of brodifacoum did not show signs of coagulopathy prior to death from misadventure. The clinical signs in the three affected animals were consistent with brodifacoum poisoning. Given that bait stations were never positioned within the tuatara enclosures, it is most likely these animals were exposed by secondary routes, such as the consumption of poisoned rodents or invertebrates that had been feeding on the bait stations. Secondary poisoning of animals from anticoagulant baits via rodents and invertebrates has been well documented (Hoare and Hare 2006; Nakayama *et al.* 2019), although there is only one anecdotal report of secondary

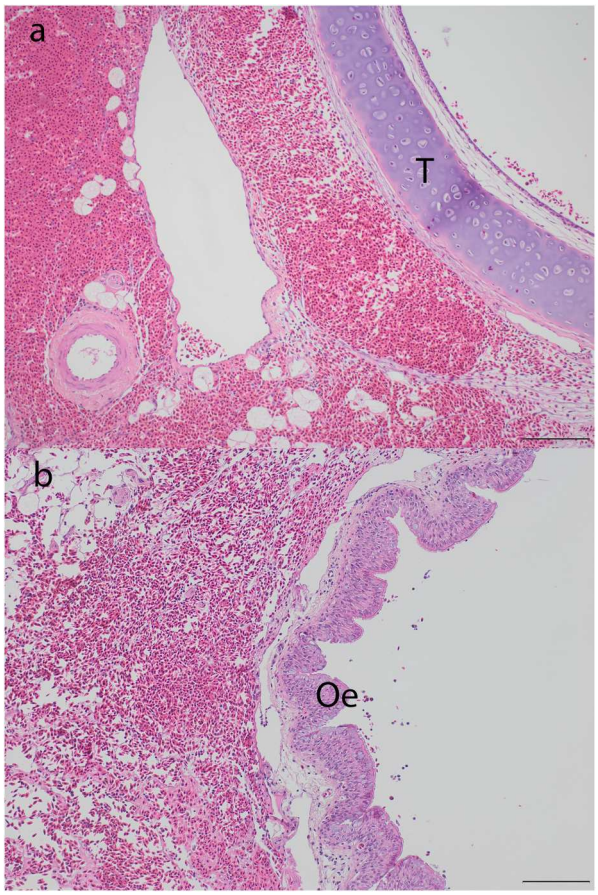

Figure 3. Photomicrographs of histological sections from a tuatara (*Sphenodon punctatus*; Case 3) showing extensive haemorrhage around a) the trachea (T) and b) the oesophagus (Oe); bars = 100 µm, H&E.

poisoning of reptiles (R. Griffiths, unpublished data cited in Fisher *et al*. 2010).

The toxicology testing in these cases was inconsistent: Case 1 was not tested and for Cases 2 and 4 only skeletal muscle was tested. Liver is considered the best tissue for anticoagulant residue testing (Booth 2019) and the sample from Case 3 is the most useful in this case series in providing supportive evidence for the role of brodifacoum in the coagulopathy and death of the tuatara. The half-life for brodifacoum in muscle is far shorter than in liver. The presence of brodifacoum in the muscle indicates a high and recent exposure, although this could well be species-dependent. The residues in the tuataras' muscle were quite low relative to that in their liver. This compares with other studies, such as that carried out by Fisher (2009), who found that residues in rat muscle were 8.6% of those found in liver, and Eason *et al.* (1996), who found the ratio of brodifacoum in muscle to that in liver in brushtailed possums (*Trichosurus vulpecula*) to be reasonably consistent at 8–11%, and 7.7% in a second study (Eason *et al.* 1999). Nevertheless, the toxicology is consistent with the three tested tuatara (Cases 2, 3 and 4) having been exposed to brodifacoum.

Interpretation of these brodifacoum concentrations is complicated by a lack of information on toxicity thresholds in reptiles in general, and tuatara in particular. The clinical effects of the anticoagulant poisoning in tuatara were not as pronounced as is seen in mammals and birds, where the clinical signs are mostly acute in nature. Mammals are highly susceptible to brodifacoum toxicity, with reported median lethal doses (LD50) of $\leq$ 1 mg/kg for rodents, 0.25–3.6 mg/kg for dogs and 5–25 mg/kg for sheep. Similarly, the LD50 for most bird species is between 3 and 20 mg/kg, but some species are particularly susceptible with LD50 values $\leq$ 1 mg/kg (Eason and Spurr 1995). It is likely that reptile species may show similar inter-species variation in susceptibility to brodifacoum.

Reported residual concentrations of brodifacoum found in reptiles vary widely. In dead reptiles, examples include 0.82 mg/kg in whole body tissue of moko skink (Mauldin *et al.* 2020), and 1.3 mg/kg in liver of King's skink (Bettink 2015). In surveillance studies, unaffected geckos (*Lepidodactylus lugubris* and *Hemidactylus frenatus*) showed a peak concentration of 0.067 mg/kg of brodifacoum in tissues (Pitt *et al.* 2015). All of the above studies reported only the brodifacoum exposure and not the clinical signs associated with poisoning in the reptiles. There is also evidence of differences between reptile species in sensitivity to anticoagulants (Mauldin *et al.* 2020). It is therefore difficult to extrapolate toxicity thresholds for brodifacoum in the tissues of tuatara, which were 0.26 mg/kg in liver and 0.019–0.035 mg/kg in skeletal muscle. More work is needed to better evaluate the effect of SGAR use on captive and wild populations of tuatara.

Two of the tuatara in this report (Cases 2 and 3) died within a week of presentation of clinical signs. However, it is possible that sub-clinical effects may have been present for much longer due either to a slow accumulation of brodifacoum from multiple feeding sessions of exposed invertebrates or to the slow metabolism of reptiles. Tuatara are adapted to much colder temperatures than most other reptiles, and measured standard metabolic rates of tuatara at 13°C are lower than that of lizards at 20°C as their adaptation to colder temperatures is mirrored by their metabolism rather than thermoregulation (Thompson and Daugherty 1998). A single dose of brodifacoum has a long half-life in the body of target animals and it may take many days for toxicity to manifest (Weir *et al.* 2016). Slow metabolism may delay the onset and prolong the occurrence of toxicity in tuatara. Some organophosphorus insecticides are more toxic at higher temperatures while some pyrethroid insecticides are less toxic at higher temperatures (Weir *et al*. 2016). It is unknown what role temperature plays in the toxicity of brodifacoum. Slow metabolic rates and lower temperatures may be one possible reason for the delayed onset of clinical signs of toxicity in tuatara. Delayed toxicity with brodifacoum has been reported in Galápagos land iguanas (*Conolophus subcristatus*) on Seymour Norte, Galápagos, where deaths were reported 2 months after a brodifacoum baiting operation (Harper *et al.* 2011) and in Telfair's skinks in Mauritius that died 3–6 weeks after poison was laid (Merton 1987).

The slow metabolic rate of the tuatara may also make them susceptible to potential sub-lethal effects associated with brodifacoum poisoning. Sub-lethal effects do not directly cause mortality but instead affect behaviour and fitness (Brakes and Smith 2005), including extreme debilitation and depressed mentation, which may lead to death by other causes like starvation, predation and accidents (Murray 2018). There are reports of brodifacoum baiting programmes where no mortality was seen despite observations of primary and secondary exposure of reptiles. For example, Wright's skinks were seen consuming brodifacoum (Talon 20) baits in the Seychelles (Thorsen *et al*. 2000), and bungarras (*Varanus gouldii*) ate poisoned dying ship rats (Burbidge 2004), and yet no mortality was observed. Any possible sub-lethal effects in these cases were not studied and there are no reports of the effects of long-term sub-lethal exposure to brodifacoum.

The deaths of these captive tuatara mean that zoological institutes that hold captive tuatara may need to reassess their pest control strategies. We have demonstrated an association between the deaths of three tuatara with severe coagulopathy and exposure to

brodifacoum in two of the affected animals, with the caveat that a fourth tuatara that had similar tissue concentrations of brodifacoum did not show signs of coagulopathy prior to death from misadventure. More research needs to be done in this field so that proper diagnosis and exposure monitoring can be implemented if similar cases occur in captive facilities. Rodent control is necessary in captive zoological institutions to prevent disease. However, preventive measures should be taken to minimise exposure to the resident tuatara to brodifacoum. These may include setting up bait stations well away from tuatara enclosures, or the use of mechanical traps in the vicinity of the enclosure.

Brodifacoum has also been used widely in the removal of rodents from offshore islands used for the conservation of tuatara. The deaths of these captive tuatara raise concerns about the use of brodifacoum for the conservation management of wild tuatara populations. Brodifacoum is aerially dropped on offshore islands to control rodents and other pests. Studies have shown that native birds living in these islands have been poisoned by brodifacoum (Eason and Spurr 1995) but there have been limited reports of any effects on reptiles. This case series suggests that brodifacoum exposure may cause coagulopathy in some tuatara. We recommend that, ideally, AR should not be used in tuatara habitat, but if they are, then careful monitoring of wild or captive populations may be needed for months following exposure to detect mortality or clinically affected animals and assess the delayed effects of poisoning.

Acknowledgements

We acknowledge Ngāti Koata as the kaitiaki for the tuatara; Todd Halsey, Hans Andersen and the staff of Halifax Veterinary Centre; and Wildbase Hospital for their care of the clinically ill tuatara. Post-mortem examinations were performed under contract with the Department of Conservation.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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