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NONTARGET MORTALITY OF NEW ZEALAND LESSER SHORT-TAILED BATS (*MYSTACINA TUBERCULATA*) CAUSED BY DIPHACINONE

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ABSTRACT: Primary and secondary poisoning of nontarget wildlife with second-generation anticoagulant rodenticides has led to restrictions on their use and to increased use of first-generation anticoagulants, including diphacinone. Although first-generation anticoagulants are less potent and less persistent than second-generation compounds, their use is not without risks to nontarget species. We report the first known mortalities of threatened New Zealand lesser short-tailed bats (*Mystacina tuberculata*) caused by diphacinone intoxication. The mortalities occurred during a rodent control operation in Pureora Forest Park, New Zealand, during the 2008–2009 Austral summer. We observed 115 lesser short-tailed bat deaths between 9 January and 6 February 2009, and it is likely that many deaths were undetected. At necropsy, adult bats showed gross and histologic hemorrhages consistent with coagulopathy, and diphacinone residues were confirmed in 10 of 12 liver samples tested. The cause of mortality of pups was diagnosed as a combination of the effects of diphacinone toxicity, exposure, and starvation. Diphacinone was also detected in two of 11 milk samples extracted from the stomachs of dead pups. Eight adults and 20 pups were moribund when found. Two adults and five pups survived to admission to a veterinary hospital. Three pups responded to treatment and were released at the roost site on 17 March, 2009. The route of diphacinone ingestion by adult bats is uncertain. Direct consumption of toxic bait or consumption of poisoned arthropod prey could have occurred. We suggest that the omnivorous diet and terrestrial feeding habits of lesser short-tailed bats make them susceptible to poisoning with the bait matrix and the method of bait delivery used. We recommend the use of alternative vertebrate pesticides, bait matrices, and delivery methods in bat habitat.

Key words: Anticoagulant rodenticides, conservation, microchiroptera, Mystacinidae, pathology, pest control, toxicants, wildlife.

INTRODUCTION

Anticoagulant rodenticides are commonly used to control rodent pests in urban and agricultural settings worldwide; they are also widely used to eradicate introduced rodents from mammal-free island ecosystems for conservation purposes (Towns and Broome 2003; Howald et al. 2007). In New Zealand, where the only native land mammals are two species of bats (King 2005), these pesticide compounds are also used to control rats (*Rattus* spp.) in mainland conservation reserves using broad-scale, sustained, ground-based applications (Innes and Barker 1999).

Anticoagulant rodenticides are categorized as either first- or second-generation compounds. Both types act by interfering with the synthesis of vitamin K-dependent blood clotting factors in the liver of vertebrates, causing fatal hemorrhaging (Buckle 1994). Second-generation compounds are more potent and more persistent in animal tissue than are first-generation compounds (Fisher et al. 2003; Erickson and Urban 2004), increasing the risk of poisoning of nontarget animals by direct consumption of poisoned bait (primary exposure) or by consumption of poisoned prey (secondary exposure) (Eason and Spurr 1995). Mortalities and sublethal poisonings, caused through both primary and secondary routes of exposure to second-generation compounds, have been recorded for a wide range of nontarget bird (e.g., Eason

et al. 2002) and mammal species (e.g., Fournier-Chambrillon et al. 2004). Intoxication of nontarget wildlife with first-generation compounds also occurs (e.g., Stone et al. 1999) but much less frequently (Erickson and Urban 2004).

Widespread concern about the continuing impacts of second-generation anticoagulant rodenticides on nontarget wildlife has led to new restrictions on their use (Department of Conservation 2000; US Environmental Protection Agency 2008) and to the investigation of more-suitable first-generation compounds, including diphacinone. Diphacinone has been considered a promising alternative to second-generation compounds because it has medium potency to rodents but relatively short persistence in animal tissue (Fisher et al. 2003). In New Zealand diphacinone has proven effective for controlling rats in mainland conservation reserves (Gillies et al. 2006). In the USA it is used to control rodents in commensal (Erickson and Urban 2004), agricultural, and rangeland settings (Salmon et al. 2007) and to eradicate rodents from small islands for conservation purposes (Witmer et al. 2007b).

Although less potent and less persistent than second-generation anticoagulants, the use of diphacinone is not without ecologic risk (Eisemann and Swift 2006; Rattner et al. 2012). Secondary poisoning of raptors that were fed mice killed with diphacinone has been demonstrated in laboratory trials (Mendenhall and Pank 1980), and residues of diphacinone have been detected in wild populations of nontarget birds (e.g., Stone et al. 2003), invertebrates (e.g., Johnston et al. 2005), and mammals (e.g., Riley et al. 2007).

We report nontarget mortalities of New Zealand lesser short-tailed bats (*Mystacina tuberculata*) due to diphacinone intoxication. These threatened, endemic bats have a terrestrial foraging habit and a broad diet comprising terrestrial, arboreal, and aerial arthropods, nectar, pollen, and fruit (Daniel 1976; Arkins et al. 1999). They have therefore been considered vulnerable to primary poisoning through consumption of toxic baits encountered while foraging (Eason and Spurr 1995) or to secondary poisoning by consumption of arthropods that had fed on toxic bait (Lloyd and McQueen 2000; Craddock 2003).

This is the first reported case of lesser short-tailed bat deaths due to anticoagulant poisoning. The incident occurred during a rodent control operation in native forest on public conservation land in New Zealand in January 2009. The mortalities were detected at two roost trees during the bats' breeding season (November–February) when pups are born and crèched in maternity roosts. We documented the mortality event and subsequent investigation to provide records for future reference.

MATERIALS AND METHODS

Study site

The bat mortalities occurred in Pikiariki Ecological Area, Pureora Forest Park, in the North Island, New Zealand (38°31′S, 175°34′E). Pikiariki Ecological Area (hereafter "Pikiariki") is a remnant of old-growth, native podocarp-hardwood forest (457 ha) within Pureora Forest Park (78,000 ha). Pikiariki has been designated an Ecological Area in recognition of its high conservation values and provides the core breeding and colonial roost tree habitat for a population of lesser short-tailed bats.

Bat mortalities

On January 9, 2009, dead and moribund lesser short-tailed bats were discovered at the bases of a maternity roost tree and a nearby colonial roost tree. At the time, a rodent control operation using diphacinone (0.005%) was taking place in Pikiariki to limit the roof rat (*Rattus rattus*) population. The active ingredient was presented in a cereal paste matrix (RatAbate®, Connovation Ltd., Auckland, New Zealand) and delivered in biodegradable plastic bait bags, each containing 300 g of paste. Bait bags were stapled to tree trunks 0.2–1 m above the ground on a 150 × 50-m grid throughout Pikiariki. Baiting commenced on 21 October 2008, with subsequent bait deployments in November and December to maintain availability to rodents.

Daily checks for further mortalities at the two roost trees continued for 34 d. An additional eight known colonial and maternity roost trees were also monitored for signs of occupancy and for mortalities. We attempted to locate any unknown roost trees which might have been occupied. Eight adult bats in apparent good health were captured in mist nets (38 mm, Avinet, City, State, USA) and fitted with radio transmitters (BD2A, Holohil Systems, Carp, Ontario, Canada), attached between the scapulae on an area of partially trimmed fur, using a latex-based contact adhesive (Ados F2®, CRC Industries New Zealand, Auckland, New Zealand). Transmitters weighed ≤0.7 g and represented <5% of bat body mass. Bats were radio-tracked to roosts during the day using a hand-held TR4 receiver (Telonics, Mesa, Arizona, USA) and a hand-held, 3-element Yagi aerial (Sirtrack, Havelock North, New Zealand). Intensive monitoring of the affected roosts continued for 5 d after the last casualty was found. The diphacinone-laced baits were removed from the field within 4 d of finding the first dead bats, following the preliminary necropsy findings.

Pathology

Dead bats were chilled and transported to Massey University, Palmerston North, New Zealand. Those in suitable condition were necropsied. Tissue samples were fixed in 10% neutral buffered formalin. Fixed tissues were embedded in paraffin, sectioned at 4 μm, and stained with hematoxylin and eosin for histologic examination. Fresh tissue samples of lung, liver, and kidney were taken from adults and pups for aerobic bacterial culture. Stomach contents of freshly dead adults were examined. Pup stomachs containing milk were excised and frozen.

Toxicology

Liver samples were frozen separately for toxicologic analysis. The diphacinone content of selected liver samples and maternal milk extracted from the stomachs of pups was determined by high-performance liquid chromatography. The method detection limit was 0.05 μg/g for liver and was undetermined for milk. The uncertainty (95% CI) was ±20%. Assays were performed by CENTOX (Centre for Environmental Toxicology), Landcare Research, Lincoln, New Zealand.

Treatment of live bats

Moribund bats were given supportive care and transported to Massey University for treatment. Admitted bats were initially hydrated twice daily with warmed subcutaneous fluids (0.9% NaCl/2.5% glucose) and dosed with subcutaneous vitamin K (Konakion, Roche, Auckland, New Zealand) at 10 mg/kg. Bats then received 10 mg/kg oral vitamin K solution twice a day, prepared by a compounding pharmacist. Treatment with vitamin K continued for 34 d. General rehabilitation continued for a further 3 wk.

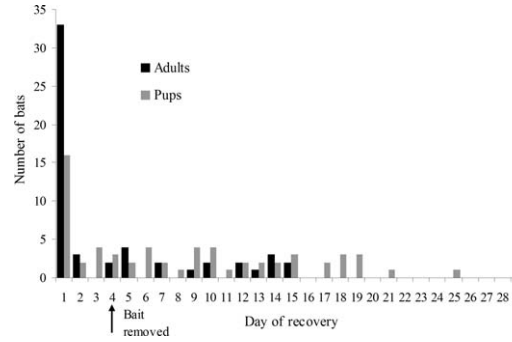

FIGURE 1. Numbers of dead and moribund lesser short-tailed bat (*Mystacina tuberculata*) adults and pups recovered each day over a 29-d period from beneath an active maternity roost tree and a nearby colonial roost tree in Pureora Forest Park, New Zealand, during a large mortality event in 2009. The large number of bats recovered on day 1 reflects an accumulation of bat bodies over an unknown period of time prior to discovery. Intensive monitoring of the affected roost trees continued for 5 d beyond the last bat recovery on day 29.

RESULTS

Mortality, morbidity, and response to treatment

We collected 118 affected bats from two roost sites over 29 d (Fig. 1). Recovery of dead and moribund adults ($n=47$ and 8, respectively) and nonvolant pups ($n=43$ dead and 20 moribund) included both sexes ($n=39$ males, 29 females, and 50 unknown). Most affected bats were located on the ground within approximately 3 m of the base of the maternity roost tree. One moribund and two dead adult bats were located at the base of a colonial roost tree 65 m east of the maternity roost. The maternity roost remained active throughout the monitoring period. No mortalities or injured bats were detected at an additional eight known maternity and

colonial roosts, and no new colonial or maternity roosts were identified through radio-tracking of eight tagged adult bats.

Affected bats found alive were lethargic and did not resist handling. Four moribund adults and nine moribund pups died shortly after collection. The remaining four adults and 11 pups found alive were transported to Massey University. Two adults and five pups survived to admission. Three pups were successfully treated and were released at the roost site on 17 March, 2009.

Pathology findings

Dead bats were in various stages of decomposition, ranging from intact to desiccated specimens, indicating that the mortalities had occurred over several days to weeks. Subcutaneous bruising and hemorrhage on the abdomen, neck, thorax, legs, and around the bones of the wings was noted on external examination of some of the fresher specimens, particularly on unfurred pups. There was dried blood in the mouth of one dead adult bat and around the anus of another, but no moribund bats exhibited signs of external bleeding.

Eleven adults and 16 pups were in suitable condition for necropsy. At necropsy, one adult and four pups that died shortly after collection were in poor body condition. Two of these pups were severely dehydrated, as evidenced by skin turgor and tacky serosal surfaces. The most frequently observed necropsy findings were subcutaneous hemorrhages, hemoperitoneum, and pulmonary hemorrhage (Table 1). Subcutaneous hemorrhages were observed in a variety of locations on both adults and pups. In two adults internal hemorrhaging was severe. There were no skeletal fractures that might indicate trauma as a cause of hemorrhage. One adult and nine pups showed no evidence of internal hemorrhaging. Only one pup showed no evidence of any hemorrhaging. Pallor of the liver was observed in one adult and one pup and pallor of the lungs in one pup.

TABLE 1. Location and incidence of hemorrhagic lesions in lesser short-tailed bat (*Mystacina tuberculata*) adults and pups with diphacinone toxicity, as determined at necropsy by gross and histologic examination, New Zealand, 2009.

	No. of bats	
Site of hemorrhage	Adults ($n=11$)	Pups ($n=16$)
Subcutaneous	4	15
Peritoneal cavity	5	5
Lungs	6	3
Heart	3	0
Skeletal muscle	2	0
Meninges	1	0
Liver	1	0
Perineal region	1	0

The stomachs of six dead adult bats contained pollen and arthropod fragments. None contained evidence of cereal bait consumption. Forty dead pups were in suitable condition for examination of the abdominal organs. Twenty-two pups had an empty stomach, 17 stomachs contained milk, and one contained pollen and arthropod fragments. Six of the milk samples may have been bat milk formula that pups received while in care, and these were excluded from toxicologic analysis. The remaining 11 samples were maternal milk.

Histopathologic examination

Histopathologic findings in five of eight adults and three of seven pups examined were indicative of coagulopathy. Significant lesions in adults included recent hemorrhage in the myocardium and pericardium ($n=2$), in the pleura or alveoli of the lung ($n=4$), in the peritoneum and the serosal surfaces of abdominal viscera ($n=2$), and in the intestinal muscular wall ($n=1$). Significant lesions in pups included atelectasis in some areas of the lung ($n=1$), diffuse moderate accumulations of hemosiderin in the cytoplasm of hepatocytes ($n=2$), free erythrocytes in the stomach lumen ($n=1$), hemorrhage within the lumen of the alveoli ($n=1$), and large, recent hemorrhages on the serosa of the kidney ($n=1$).

Microbiology

Ten species of bacteria were cultured from samples of liver, lung, and kidney from short-tailed bat adults ($n=3$) and pups ($n=1$); *Corynebacterium* sp. ($n=3$ bats), *Proteus* sp. ($n=2$), alpha hemolytic *Streptococcus* sp. ($n=2$), nonhemolytic *Streptococcus* sp. ($n=2$), *Micrococcus* sp. ($n=1$), *Staphylococcus aureus* ($n=1$), *Escherichia coli* ($n=1$), *Proteus mirabilis* ($n=1$), *Hafnia alvei* ($n=1$), and *Serratia* sp. ($n=1$). None of the bacteria isolated was considered to be a primary pathogen.

Toxicology

Diphacinone was confirmed in liver samples from four of five adults (mean concentration±SE; 0.29±0.06 μg/g, $n=4$) and six of seven pups tested (0.32±0.07 μg/g, $n=6$). Concentrations ranged from 0.19 to 0.68 μg/g of liver tissue. Diphacinone was also confirmed in two of the 11 maternal milk samples tested at concentrations of 0.23 and 0.09 μg/g.

DISCUSSION

Cause of death

Our investigation confirms a diagnosis of diphacinone-induced coagulopathy of lesser short-tailed bats. Diagnosis was supported by a history of exposure, clinical signs, gross and histologic lesions at necropsy, toxicologic analysis, and response to treatment. For 80 d leading up to the observed mortalities, diphacinone-laced baits were distributed throughout the bats' core roosting habitat. Diphacinone residues were confirmed in liver samples from both adults and pups and in two samples of maternal milk. Severe diffuse hemorrhage, as observed in the bats at the time of necropsy, is characteristic of anticoagulant poisoning (Berny 2007).

Few of the affected bats which were recovered alive during the mortality event survived for more than a few days after collection. Following ingestion of a lethal dose of an anticoagulant, there is a delay of several days until death (5–6 d in rats dosed with 3 mg/kg diphacinone) (Eason and Wickstrom 2001). It is likely, therefore, that the live bats which allowed us to approach and handle them were already severely affected by diphacinone. Furthermore stress, caused by handling and transport, could have exacerbated susceptibility to anticoagulant toxicosis (Robinson et al. 2005).

The cause of mortality of bat pups was diagnosed as a combination of the effects of diphacinone toxicity, exposure, and starvation. The presence of diphacinone in the maternal milk collected from the stomachs of dead pups provided evidence that the poison was passed from lactating adult females to their pups. This route of intoxication with pesticides has been reported for bat pups in the USA (e.g., Clark et al. 1978, 1988). The pathway of diphacinone intoxication of the adult bats at Pikiariki, however, is uncertain.

Route of exposure

Two likely routes of exposure of adult bats to diphacinone at Pikiariki are direct consumption of toxic bait or secondary poisoning after eating arthropods that had consumed toxic bait. Due to the delayed mode of action of anticoagulants and the rapid gut transit time of microbats (Klite 1965), the absence of bait in the stomachs of examined adult bats does not exclude the possibility of primary poisoning.

The method of presentation of the diphacinone-laced baits during the mortality event may have increased the potential for bait encounters and consumption by foraging adult bats. Diphacinone was formulated in cereal paste baits which were placed in biodegradable bags stapled to tree trunks rather than in pelletized cereal baits secured in bait stations. Bait acceptance trials indicate that captive lesser short-tailed bats are unlikely to consume the pelletized cereal baits typically used in vertebrate pest control operations (Lloyd 1994). However, in a separate captive trial, lesser short-tailed bats sampled cereal paste baits,

similar to the RatAbate paste used at Pikiariki, although it was uncertain whether the quantities consumed were sufficient to put bats at risk of poisoning (Beath et al. 2004).

Consumption of toxic bait by arthropods may have served as a pathway for secondary exposure of lesser short-tailed bats to diphacinone (Lloyd and McQueen 2000). Pesticide-contaminated prey have been implicated in mortalities of insectivorous bats in the USA (Clark et al. 1988; O'Shea and Clark 2002), and a wide variety of arthropod species have been observed on pelletized cereal baits in New Zealand forests (e.g., Sherley et al. 1999; Spurr and Drew 1999). Residue analysis of arthropods exposed to anticoagulant baits in laboratory and field trials confirm that they may act as vectors of these compounds (Craddock 2003; Fisher et al. 2007).

Despite evidence to support a secondary route of poisoning, no mortalities have previously been detected in wild lesser short-tailed bat populations monitored through pest control operations using pelletized cereal baits laced with anticoagulants, either when aerially broadcasted (Sedgeley and Anderson 2000) or concealed in bait stations (O'Donnell et al. 2011). Further investigation into the palatability and acceptance of a nontoxic RatAbate paste matrix to lesser short-tailed bats and forest arthropods is required.

Sensitivity to diphacinone

Species differ in their sensitivities to a particular toxicant (Erickson and Urban 2004), although there may be general trends within animal groups (McIlroy 1986). The microchiroptera may be relatively more sensitive to diphacinone than are most other mammal groups. Vampire bats (*Desmodus rotundus*) are sensitive to diphacinone, and populations in Central and South America are controlled using this toxicant in systemic or dermal applications (Arellano-Sota 1988). The acute oral median lethal dose (LD_{50}) of diphacinone determined for caged (unexercised) vampire bats is 0.91 mg/kg (Thompson et al. 1972) and in active bats may be closer to 0.3 mg/kg (Bullard and Thompson 1977). Furthermore, LD_{50} figures for acute doses of first-generation anticoagulants are typically higher than multiple doses administered over several consecutive days, suggesting that the risks associated with the use of these compounds are underestimated (Vyas and Rattner 2012). The acute oral LD_{50} of diphacinone for lesser short-tailed bats is not known (Fisher and Broome 2010), and caution must be observed when using data from similar species to predict sensitivity (McIlroy 1986).

Extent of mortalities

The number of carcasses recovered in Pikiariki is likely to be an underestimate of the total mortality of lesser short-tailed bats resulting from diphacinone intoxication. Carcass counts are unreliable estimators of mortality (Vyas, 1999). The large number of decomposed bodies found on the first day of recovery suggests that deaths had been occurring for several days to weeks. Many adult deaths may have gone undetected between initial deployment of baits in October and the start of our surveillance 80 d later on January 9. Pups are born in late December, and so would have been susceptible to poisoning for a much-shorter period, but maternal exposure may have caused prenatal losses through abortion (Robinson et al. 2005). During the interval between death and our searches, carcasses may have decomposed or been removed by scavengers, and sick animals may have been taken by predators. Furthermore, dead and moribund lesser short-tailed bats encountered in our searches were difficult to see due to their color and small size, and some bats may have been obscured from view by vegetation. An unknown number of bats may have died inside the affected roost trees, at other unidentified roost trees, or away from roost sites.

Adult bat deaths were detected for several days following removal of toxic baits from Pikiariki, most likely due to the delayed onset of symptoms in lethally dosed animals. Sublethal intoxication of lactating females, or their eventual death, may account for the extended period of pup deaths observed. The hepatic elimination half-life of diphacinone is 3 d in female laboratory rats (Fisher et al. 2003). If it is similar in lesser short-tailed bats, the clotting mechanism would begin to recover 3 d after exposure and we would not expect mortalities to continue long after bait removal. However, sublethally exposed individuals may have been affected in ways which compromised their survival beyond this period (e.g., Riley et al. 2007; Lemus et al. 2011).

The overall impact of the bat mortalities on the viability of the Pikiariki population is not known. Bats are a long-lived species with low reproductive output (Barclay and Harder 2003). We lack information on population trends and the size of the Pikiariki lesser short-tailed bat population before and after the mortality event. There is a need for baseline data on population size and long-term studies on population dynamics to monitor the population's recovery and viability.

Management implications

These findings illustrate the hazards of diphacinone use to the lesser short-tailed bat. However, the risks to nontarget species of anticoagulant rodenticide use should be weighed against the benefits of control of rodent pests (e.g., Pascal et al. 2005). The lesser short-tailed bat, classified as vulnerable by the International Union for the Conservation of Nature (O'Donnell 2008), is likely to benefit from rodent control (Pryde et al. 2005). Furthermore, the consequences of failing to reduce rodent pest impacts can be extreme, as demonstrated by the probable extinction of the New Zealand greater short-tailed bat (*Mystacina robusta*) (Daniel 1990; Worthy 1997; O'Donnell et al. 2010).

While the use of vertebrate pesticides remains a necessity, measures which reduce exposure to, and the adverse effects on, nontarget species are important (Witmer et al. 2007a; Eason et al. 2010). Determination of the route of exposure of the Pikiariki lesser short-tailed bats to diphacinone will help inform bait design and delivery to reduce the risk of further mortalities during future rodent control operations in bat habitat. The effectiveness of any operational changes in minimizing risks to bats should be evaluated by appropriate monitoring of bat populations. Until more information is available, we recommend the use of less-potent toxicants, presented in pelletized cereal baits delivered in secure bait stations, to control vertebrate pests in bat habitat.

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