Continuing Field Efficacy of Norbormide against both *Rattus rattus* (Ship Rats) and *Rattus norvegicus* (Norway Rats)

Lee Shapiro
Boffa Miskell Ltd., Auckland, New Zealand
Centre for Wildlife Management and Conservation, Lincoln University, Lincoln, New Zealand
Kirtana Kumar
Boffa Miskell Ltd., Auckland, New Zealand
David Rennison and **Margaret Brimble**
The University of Auckland, Auckland, New Zealand
Duncan MacMorran
Invasive Pest Control Ltd. and Connovation Ltd., Auckland, New Zealand
Charles Eason
Invasive Pest Control Ltd. and Connovation Ltd., Auckland, New Zealand
Centre for Wildlife Management and Conservation, Lincoln University, Lincoln, New Zealand

ABSTRACT: Norbormide is a uniquely selective rat toxicant for *Rattus* species, with rats being 100- to 150-fold more sensitive to norbormide toxicity than most other mammals and birds. Previously we reported that on completion of a 10-year program of targeted fundamental and applied synthetic chemistry and toxicology, taste aversion associated with this compound had been overcome. In 2020-2022, trials have been successfully completed on poultry farms with Norway rats and larger scale field trials were undertaken targeting ship rats using 1% norbormide paste baits. Firstly, the efficacy of norbormide-containing paste baits targeting rat infestations on poultry farms was proven with a 100% reduction of Norway rat populations on three different farms. Secondly, 100% reduction in ship rat abundance was achieved at two large field test sites; and no reduction was achieved at the untreated control site. These larger field trials are described in depth in this publication. Plans are progressing to complete product development and registration.

KEY WORDS: black rat, field trials, norbormide, Norway rat, *Rattus norvegicus*, *Rattus rattus*, ship rat

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INTRODUCTION

Anticoagulant rodenticides are the most important tools for rodent control and will remain so well into the future (Buckle and Eason 2015). Nevertheless, there are drivers for other tools in the toolbox. The emergence of physiological resistance in some populations of rodents, even to second-generation rodenticides and the discovery of residues of the second-generation anticoagulants in wildlife (Young and De Lai 1997, Stone et al. 1999, USEPA 2004, 2008; Crowell et al. 2013, Huang et al. 2016, Kotthoff et al. 2018, Walther et al. 2020, 2021) has raised non-target toxicity (Niedringhaus et al. 2021, Roos et al. 2021) and food safety concerns (Mercer et al. 2022). In addition, questions regarding humaneness of second-generation anticoagulants have been raised in the past (Littin et al. 2002, Mason and Littin 2003). Effort continues to better assess the individual and population level effects on wildlife of anticoagulants (Quinn and Swift 2018). In parallel there has been increased interest in norbormide (Campbell et al. 2015, Eason et al. 2017) as it has little or no toxicity to non-target species such as birds, livestock, and pets and does not cause residue concerns. Norbormide is a selective rat toxicant, effective against both ship rats (*Rattus rattus*) and Norway rats (*R. norvegicus*). It was originally developed in the 1960s as an antirheumatic drug (Russell 1965) and then explored as a rodenticide but discontinued in the 1970s as anticoagulant toxins became popular, taste aversion limited its effectiveness, and field efficacy results were mixed. Its mode of action is through vasoconstriction of small arteries and vasodilation of large arteries in rats, and death results from acute heart failure (Yelnosky and Lawlor 1971). It is likely to be more humane than most other rodenticides because of the relatively short time to death and short duration of symptoms of poisoning when compared with anticoagulant rodenticides (Shapiro et al. 2018, 2020).

Norbormide shows a remarkable selectivity both in toxicity and in pharmacological effect. The oral LD_{50} values for both wild and domestic Norway rats range from 5.3 to 15.0 mg/kg (Roszkowski 1965). Corresponding values for ship rats and Hawaiian/Polynesian rats (*R. exulans*) were 52 and 10 mg/kg respectively. Oral LD_{50} values were much higher in other rodent and lagomorphs, for example: hamster 140 mg/kg; guineapig 620 mg/kg; mouse 2,250 mg/kg; and rabbit about 1,000 mg/kg. The oral LD_{50} was high in other species tested; in the dog, cat, monkey, sheep, pig and chicken no effect was detectable at 1,000 mg/kg (Roszkowski et al 1964, Roszkowski 1965, Nui 1970). The acute toxicity of norbormide in rats, mammals, and birds is summarised in Table 1.

Pharmacokinetic studies indicate norbormide is readily metabolized and unlikely to be persistent (Ravindran et al.

2009a, 2009b). In-vitro and in-vivo metabolism studies in the literature have shown that, unlike some commonly used rodenticides (e.g., brodifacoum), norbormide is metabolised in rats and other rodent species through hepatic hydroxylation. Hydroxylation is a major pathway of xenobiotic metabolism (Phase 1), which makes compounds more hydrophilic and/or available for conjugation (Phase 11) and excretion. This process is consistent with its rapid excretion and clearance in in-vivo studies in rats and mice (Ravindran et al. 2009b).

To examine whether rapid metabolic clearance or species-dependent formation of a toxic metabolite play a role in the marked species-sensitivity, in vivo metabolic studies have been completed in rats and mice (Ravindran et al. 2009b). Taken together, these studies indicate that the toxicity resides with the parent compound, rather than species-dependent formation of a potent metabolite, and that species sensitivity may be controlled at the pharmacodynamic level.

Further evidence for the parent compound, namely norbormide, being responsible for acute toxicity in rats comes from comparative assessment of oral versus intravenous toxicity in rats at lethal dose. Rapid lethality occurred in rats following intravenous dosing, leaving traces of norbormide in blood and tissue, without time for the formation of metabolites. After oral administration to rats, analysis of blood taken after 10 and 30 minutes revealed neither parent compound nor its hydroxylated metabolites at these time points, suggesting that they were very rapidly cleared from the blood stream (Ravindran et al. 2009b).

Given these positive attributes it is not surprising that other research teams have explored methods of overcoming taste aversion to norbormide including encapsulation (Nadian and Lindblom 2002) and the development of analogues (Rennison et al 2007). Negating the need for encapsulation or analogues Invasive Pest Control (IPC) Ltd, the University of Auckland, and Boffa Miskell Ltd have been trialing and refining a new palatable and effective formulation of norbormide for the control of Norway and ship rats, and this research has been enabled by funding and support from Predator Free 2050 Ltd (PF2050). This development has involved optimizing routes of chemical synthesis to limit impurities and improving bait palatability to produce an effective formulation of norbormide for the control of Norway and ship rats. This represents a breakthrough with the successful development of a norbormide paste bait, developed for use in bait stations, which is highly palatable and effective on rats in cage trials (Shapiro et al. 2018).

The research to date has been methodical and has enabled the research team to ensure small-scale batches retain consistent efficacy, and the most recent step has involved working closely with commercial partners to produce larger batches of norbormide. Larger batches of norbormide have enabled further laboratory testing to confirm efficacy on Norway and ship rats. The results from previous cage trials on Norway and ship rats and control at two poultry farms (Shapiro et al. 2020) are encouraging, and have been subsequently confirmed in two additional trials, successfully targeting Norway rats on poultry farms (pers. commun., Lee Shapiro). The logical progression from these successful small-scale laboratory trials for Norway and ship rats and small-scale trials around intensive agriculture targeting Norway rats is to trial this formulation in native forest to target ship rats.

Table 1. Toxicity of norbormide to different species.
(from Roszkowski et al. 1964 and 1965)

Species	LD_{50} mg/kg
Norway rat (*Rattus norvegicus*)	5-15
Ship rat (*Rattus rattus*)	52
Hamster (*Cricetus* spp)	140
Guinea-pig (*Cavia porcellus*)	640
House mouse (*Mus musculus*)	2,250
Rabbit (*Oryctolagus cuniculus*)	>1,000
Cat (*Felis catus*)	>1,000
Dog (*Canis* spp)	>1,000
Duck (*Anas platyrhynchos*)	>1,000
Pig (*Sus scrofa*)	>1,000
Pigeon (Columbidae)	>1,000
Turkey (Meleagris)	>1,000
Sheep (*Ovis aries*)	>1,000

This publication focuses on the control of ship rats with paste bait containing 1% norbormide. Ship rats are the dominant rat found in New Zealand and are common in a variety of habitats throughout the country, particularly in lowland forests, where there is a wide range of food resources available, including seeds, fruit, invertebrates, and small native vertebrate species (Russell et al. 2019, Innes et al. 2010, Wilson et al. 2007). The decline and extinction of many native species in New Zealand have been attributed to the introduction of ship rats, with a number of native New Zealand species still under significant threat from ship rats (Brown et al. 2015). The following sections of this paper describe two field trials targeting ship rats.

METHODS

Field trials were undertaken at two sites; both were located approximately 19 km south of central Christchurch on Banks Peninsula (South Island, NZ). The two treatment sites were native forest blocks fenced off from surrounding farmland and located on the same privately-owned property. The vegetation within the trial blocks was largely native bush with a core area of remnant vegetation and regenerating native scrub.

Trial Site 1 was approximately 26.8 ha and centered around a gully running east to west and consisting predominantly of regenerating scrubland with areas of gorse and sections of native, established broadleaf forest. Trial Site 2 was approximately 26.4 ha and centered around a gully with two arms, one running east to west and the most southern of the two arms running northeast to southwest. This gully consisted of predominantly established native broadleaf forest, with sections of regenerating scrubland on the margins. Sites 1 and 2 were adjacent to each other with an average distance of 150 m between the two trial

sites apart from one section of Site 1 where a small area of scrub extended to within 60 m of Site 2. The area between the two sites was predominantly pasture habitat. To minimize any potential movement of rats between Sites 1 and 2 and to keep them independent, a line of rat traps was installed in the habitat between the two sites and run for the duration of the trial. Both sites offered good habitat for ship rats, with native trees providing fruit and seed. There had been no rodent control at either of the sites for >12 months. The habitat type and the lack of rodent control therefore made these sites suitable for this trial.

An untreated Control Site was also selected for these trials, and this was located approximately 5.5 km to the east of the treatment sites. Orton Bradley Park is a private 650-ha rural property situated on the southern shores of Lyttelton Harbour and is made up of a mixture of farmland, planted exotic trees, and native bush remnants within gullies. The untreated Control Site chosen consisted of approximately 65 ha of remnant native bush within the main gully at Orton Bradley and consists predominantly of kanuka and kowhai canopy species and five finger, kohuhu, and broadleaf.

The purpose of the untreated Control Site was to illustrate that any decrease in rat abundance at Sites 1 and 2 were due to the deployment of toxic bait containing 1% norbormide and not due to seasonal or environmental effects.

Monitoring Pre- and Post-control

Chew cards are frequently used to estimate rodent abundance in New Zealand. They are made up of rectangles of corflute card (9 × 18 cm) with corrugations between the layers of card that have sections filled with an attractant lure. The cards are folded in half and nailed to a tree trunk 30 cm above the ground, allowing pest animals to bite and chew on the card and leave unique tooth impressions that are then analyzed and identified as rat species, as distinguished from other animals (Sweetapple and Nugent 2011).

Chew cards containing peanut butter lure were deployed in lines of 10 cards with 20-m intervals between cards (180 m total distance along a line). Sites 1 and 2 each were set up with three chew card lines (lines were spaced at least 200 m apart from each other giving a total of 30 chew cards at each trial site, and the same number of chew card lines and cards were deployed at the Control Site at Orton Bradley Park. Chew cards were deployed for seven days at Sites 1 and 2 and at the Control Site for the pre monitor at each site. Chew cards were deployed at all three sites at the conclusion of the toxic control operation on 29 September and left in situ for seven days before being retrieved for analysis. Chew cards were analyzed by the research team, and given the importance of the trial, they were also sent for independent assessment by Landcare Research, NZ.

The chew card rat abundance index (CCI) was expressed as the percentage of cards at each site that recorded rat bite marks. The CCI was calculated for monitoring undertaken pre and post toxic control, and the change in CCI was used to determine the change in relative abundance of ship rats at the two treatment sites and the efficacy of paste bait containing 1% norbormide.

Pre-feeding

A total of 52 rat-specific bait stations were deployed at Site 1, and 53 were deployed at Site 2. Bait stations were deployed on a 75 × 75 m grid at each trial site. When deployed, each bait station was loaded with 200 g of non-toxic paste bait. This paste bait formulation was identical to that used in the toxic section of the trials but without the norbormide active. Pre-feeding was undertaken during late July, August, and early September 2021 and the aim was to ensure that rats were readily feeding on baits prior to deploying toxic baits. Pre-feeding is particularly important with acute toxins, as rats are cautious feeders and pre-feeding decreases the chances of rats being 'shy' feeders and eating a sub-lethal dose of toxic bait.

Pre-feeding commenced on 26 July 2021 with all bait stations being deployed at the site loaded with 200 g of non-toxic paste bait. After initial deployment, bait stations were checked on two occasions –16 August and then 8 September. During the check on 16 August, any bait stations with bait eaten were topped up with a further 100 g of non-toxic paste bait, or topped up to 200 g where bait take was ≤100 g. The trial was paused during the Covid-19 national lockdown starting on 17 August, and bait stations were checked again during the first week of September. Any remaining non-toxic pre-feed was removed from bait stations on 8 September 2021.

During the bait station checks, each station was assessed to determine if any consumption of pre-feed take had occurred, and how much pre-feed was needed to top up the stations that recorded consumption of pre-feed. Bait stations were not weighed, but an assessment of the percentage of the bait consumed was made to categorize bait take into either 100%, 75%, 50%, 25% or no bait take. The percentage take was then used to estimate the amount of bait consumption from each station in grams.

Toxic Baiting

Following the removal of the non-toxic pre-feed paste bait, bait stations at Sites 1 and 2 were left empty for six nights, and then each bait station was loaded with 200 g of paste bait containing 1% norbormide (Figure 1) over two days on 15 and 16 September 2021. Paste bait was manufactured at Connovation Ltd, East Tamaki, Auckland. Once toxic baits were deployed at Sites 1 and 2, bait stations were checked every two days for the first week, and then on two occasions during the second week, in accordance with the New Zealand Environmental Protection Authority containment approval. Seven days after toxic bait was deployed, all bait stations were weighed to determine bait take and each bait station with bait take was topped up to 200 g. All bait stations were weighed at the conclusion of the trial to determine the bait take in the second week of the trial. At the conclusion of the trial on 29 September (i.e., 13-14 days of continuous toxic bait availability), all remaining toxic bait was removed from bait stations, and post-monitoring with chew cards was started that same day.

Figure 1. Bait station and bucket of toxic paste bait (left) and 2 × 100 g balls of toxic paste bait in the bait station (right).

Regulatory Approvals and Quality Assurance

Both field trials were undertaken with an approval from the Waikato University Animal Ethics Committee (Approval #1090), a Containment Approval for field trials from the New Zealand Environmental Protection Authority (HSC100244), and a Provisional Registration from the Agricultural Compounds and Veterinary Medicines group within the Ministry for Primary Industries (Approval number V009667).

Batches of paste bait containing 1% norbormide (TB1539) was manufactured by Connovation Ltd, (36B Sir William Ave, East Tamaki, Auckland) on 4 August 2021. A 50 subsample was sent to Labtec Scientific & Technical to confirm the concentration of norbormide in the paste bait at 1.0%.

RESULTS

Trial Site 1

Rat monitoring undertaken prior to toxic baiting recorded 33.3% CCI at Site 1. Non-toxic pre-feed paste bait take was between 16 August to 8 September in which 9.5 kg of bait was eaten from 48 of the 52 bait stations. All 48 bait stations were empty when checked and the remaining four bait stations recorded no bait consumed.

Toxic baiting was undertaken over two weeks, and during the first seven days of baiting 3.75 kg of toxic bait was consumed from 41 of the 52 bait stations at Site 1, with the remaining 11 bait stations having no bait consumed. During the second seven days of baiting, 1.5 kg of toxic bait was consumed from 25 of the 52 bait stations at Site 1, with the remaining 27 bait stations having no bait consumed. Following toxic baiting, post-treatment monitoring was undertaken and found no rat chews on any of the 30 chew cards (0% CCI) from this site; only possum and mouse chew marks were recorded. The toxic treatment achieved a 100% reduction in rat chew card at Trial Site 1.

Trial Site 2

Rat monitoring undertaken prior to toxic baiting recorded 16.7% CCI at Site 2. Non-toxic pre-feed paste bait take was 8.7 kg of bait eaten from 46 of the 53 bait stations. Of the 46 bait stations with bait consumption, 41 were empty and five had partial bait consumption, while the remaining seven bait stations had no bait consumption.

Toxic baiting was undertaken over two weeks, and during the first seven days of baiting 2.34 kg of toxic bait was consumed from 31 of the 53 bait stations at Site 2, with the remaining 22 bait stations having no bait consumed. During the second seven days of baiting, 0.58 kg of toxic bait was consumed from 12 of the 52 bait stations at Site 2, with the remaining 41 bait stations having no bait consumption. Following toxic baiting, post-treatment monitoring was undertaken and found no rat chews on any of the 30 (0% CCI) chew cards from this site; only possum and mouse chew marks were recorded. The toxic treatment achieved a 100% reduction in rat abundance at Trial Site 2.

Untreated Control Site

Rat monitoring at the Control Site at Orton Bradley Park undertaken prior to toxic baiting at Treatment Sites 1 and 2 recorded 16.7% CCI, and the post-trial monitoring at the Control Site showed little change with 13.3% CCI, which illustrated that there was no significant change in ship rat abundance due to seasonal, environmental, or other influences.

CONCLUSION

Previously, our research group reported that we had overcome taste aversion and established the efficacy of norbormide-containing baits targeting rat infestations on two poultry farms and achieved 100% and 96% reductions of wild Norway rat populations (Shapiro et al. 2018, 2020).

These results in poultry have been confirmed since then; with 100% at two additional poultry farms. However, the breakthrough reported in this paper is the successful targeting of ship rats, using paste bait containing 1% of the same norbormide formulation.

The post-treatment chew card monitoring detected no ship rats in either Site 1 or Site 2, and there was no meaningful change in ship rat abundance at the untreated Control Site during the toxic trial (pre-monitor 16.7% CCI; post-monitor 13.3% CCI), which illustrates that the trial results were not due to seasonal, environmental, or other influences, but a direct result of the toxic baiting with paste bait containing 1% norbormide. The chew cards from the post-treatment monitoring of Sites 1 and 2 were sent to an expert reviewer who confirmed that there were no rat chew marks present on these cards. This reduction in the ship rat abundance at both trial sites was the first reported field trial of norbormide targeting ship rats. It represents a significant reduction in the ship rat population at both sites, and well above the minimum 80% efficacy required registration field trials in New Zealand.

The target-specific nature of norbormide will enable its use in areas where the rapid reduction in rat populations through toxic baiting may currently be problematic or not undertaken due to risks to non-target species or opposition to current broad-spectrum toxins.

This field trial, targeting ship rats, and the four recent complementary field trials targeting Norway rats represent a significant milestone in the development of norbormide for rodent control. Critical milestones are summarized as follows:

- 2008 – Fundamental and applied synthetic chemistry and toxicology
- 2020 – Success in research and development to produce palatable and consistent norbormide bait
- 2020/2022 – Accelerating progress on field trials
- 2022– Registration dossier with New Zealand Authorities (MPI/EPA)
- 2022 – Scaling-up manufacturing process
- 2023/2024 – Registration in NZ plus exploring US and other markets (planned)

The focus now is on 1) registration of the paste bait used in these field trials; 2) scaling manufacture of norbormide and the paste bait; and 3) the development of a solid bait. Solid bait is of interest because it can be even more readily cached by rats than can paste baits, and because solid bait (not paste bait) could be deployed aerially (e.g., via helicopter).

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LITERATURE CITED

Brown, K., G. Elliott, J. Innes, J. Kemp. 2015. Ship rat, stoat and possum control on mainland New Zealand. An overview of techniques, successes and challenges. Department of Conservation, Wellington, New Zealand.

Buckle, A. P., and C. T. Eason. 2015. Control methods: chemical. Pages 123-154 *in* Rodent pests and their control. 2nd Edition. CABI International, Wallingford, Oxfordshire, England.

Campbell, K. J., J. Beek, C. T. Eason, A. S. Glen, J. Godwin, F. Gould, and G. S. Baxter. 2015. The next generation of rodent eradications: innovative technologies and tools to improve species specificity and increase their feasibility on islands. Biological Conservation 185:47-58.

Crowell, M., K. G. Broome, C. T. Eason, A. A. C. Fairweather, S. Ogilvie, and E. C. Murphy. 2013. How long do vertebrate pesticides persist in living mammals? Priorities for research. DOC Research and Development Series 337. Department of Conservation, Wellington, New Zealand.

Eason, C. T., L. Shapiro, S. C. Ogilvie, C. King, M. Clout. 2017. Trends in the development of mammalian pest control technology in New Zealand. New Zealand Journal of Zoology 44(4):267-304. DOI: 10.1080/03014223.2017 .1337645.

Huang, A. C., J. E. Elliott, S. Hindmarch, S. L. Lee, F. Maisonneuve, V. Bowes, K. M. Cheng, K. Martin. 2016. Increased rodenticide exposure rate and risk of toxicosis in barn owls (*Tyto alba*) from southwestern Canada and linkage with demographic but not genetic factors. Ecotoxicology 25(6):1061-1071.

Innes, J., C. M. King, L. Bridgman, N. Fitzgerald, G. Arnold, and N. Cox. 2010. Effect of grazing on ship rat density in forest fragments of lowland Waikato, New Zealand. New Zealand Journal of Ecology 34(2): 227-232.

Kotthoff, M., H. Rudel, H. Jurling, K. Severin, S. Hennecke, A. Friesen, J. Koschorreck. 2018. First evidence of anticoagulant rodenticides in fish and suspended particulate matter: spatial and temporal distribution in German freshwater aquatic systems. Environmental Science and Pollution Research 26:7315-7325.

Littin, K. E., C. O'Connor, N. Gregory, D. Mellor, and C. T. Eason. 2002. Behaviour, coagulopathy and pathology of brushtail possums (*Trichosurus*). Wildlife Research 29:259-267.

Mason, G. J., and K. E. Littin. 2003. The humaneness of rodent pest control. Animal Welfare 12:1-37.

Mercer, M. A., J. L. Davis, J. E. Riviere, R. E. Baynes, L. A. Tell, M. Jaberi-Douraki, F. P. Maunsell, and Z. Lin. 2022 Mechanisms of toxicity and residue considerations of rodenticide exposure in food Animals: a FARAD perspective. Journal of the American Veterinary Medical Association 1:1-10.

Nadian A., and L. Lindblom. 2002. Studies on the development of a microencapsulated delivery system for norbormide, a species-specific acute rodenticide. International Journal of Pharmaceutics V 242(1-2):63-68.

Niedringhaus, K. D., N. M. Nemeth, S. Gibbs, J. Zimmerman, L. Shender, K. Slankard, H. Fenton, C. Bahnson, M. F. Dalton, E. J. Elsmo, R. Poppenga, B. Millsap, and M. G. Ruder. 2021. Anticoagulant rodenticide exposure and toxicosis in bald eagles (*Haliaeetus leucocephalus*) and golden eagles (*Aquila chrysaetos*) in the United States. PLoS ONE 16(4):e0246134.

Nui, M. 1970. Pharmacological action of norbormide. Japanese Journal of Pharmacology 66:224-236.

Quinn, N., and C. E. Swift. 2018. What do we need to know to address individual and population level effects on wildlife of anticoagulants. Proceedings of Vertebrate Pest Conference 28:235-242.

Ravindran, S., B. Hopkins, S. Bova, D. Rennison, M. Brimble and M. Tingle. 2009a. *In vitro* metabolism of norbormide in rat, mouse and guinea pig liver preparations. Environmental Toxicology and Pharmacology 27:144-148.

Ravindran, S., B. Hopkins, S. Bova and M. Tingle. 2009b. *In vivo* metabolism of norbormide in rats and mice. Environmental Toxicology Pharmacology 28:147-151.

Rennison, D., S. Bova, M. Cavalli, F. Ricchelli, A. Zulian, B. Hopkins, and M. Brimble. 2007. Synthesis and activity studies of analogues of the rat selective toxicant norbormide. Bioorganic and Medicinal Chemistry 15(8):2963-2974.

Roos, S., S. T. Campbell, G. Hartley, R. F. Shore, L. A. Walker, and J. D. Wilson. 2021. Annual abundance of common Kestrels (*Falco tinnunculus*) is negatively associated with second generation anticoagulant rodenticides. Ecotoxicology 30(4):560-574.

Roszkowski, A. P. 1965. The pharmacological properties of norbormide a selective rat toxicant. Journal of Pharmacology and Experimental Therapeutics. 149(2): 288-299.

Roszkowski, A. P., G. I. Poos, and R. J. Mohrbacher. 1964. Selective rat toxicant. Science 114:253-261.

Russell, R. U. 1965. Norbormide: a *Rattus* specific toxic agent. Journal of the Forensic Science Society 1:80-83.

Russell, J. C., J. H. Robins, and R. M. Fewster. 2019. Phylogeography of invasive rats in New Zealand. Frontiers in Ecology and Evolution 7:48.

Shapiro S, C. T. Eason, D. MacMorran, D. Rennison, M. Brimble, and M. Jackson. 2020. Preliminary field trials with a palatable form of norbormide. Proceedings of Vertebrate Pest Conference 29:134-136.

Shapiro, L., D. Rennison, M. Brimble, D. MacMorran, and C. T. Eason. 2018. Redevelopment of a rat specific rodenticide norbormide. Proceedings of Vertebrate Pest Conference 27: 47-50.

Stone, W. B., J. Okoniewski, and J. Stedlin. 1999. Poisoning of wildlife with anticoagulant rodenticides in New York. Journal of Wildlife Diseases 35:87-193.

Sweetapple, P., and G. Nugent. 2011. Chew-track-cards: a multiple-species small mammal detection device. New Zealand Journal of Ecology 35(2):153-162.

U.S. Environmental Protection Agency (US EPA). 2004. Potential risks of nine rodenticides to birds and nontarget mammals: a comparative approach. Washington, D.C.

U.S. Environmental Protection Agency. 2008. Risk mitigation decision for ten rodenticides. EPA-HQ-OPP-2006-0955-0764. Washington, D.C.

Walther, B., A. Geduhn, D. Schenke, and J. Jacob. 2021. Exposure of passerine birds to brodifacoum during management of Norway rats on farms. Science of the Total Environment 762(2):144-160.

Walther, B., A. Geduhn, D. Schenke, A. Schlotelburg, and J. Jacob. 2020. Baiting location affects anticoagulant rodenticide exposure of non-target small mammals on farms. Pest Management Science 77(2):611-619.

Wilson, D. J., M. G. Efford, S. J. Brown, J. F. Williamson, and G. J. McElrea. 2007. Estimating density of ship rats in New Zealand forests by capture-mark-recapture trapping. New Zealand Journal of Ecology 1:47-59.

Yelnosky, J. and R. Lawlor. 1971. Cardiovascular effects of norboromide. European Journal of Pharmacology 16:117-119.

Young, J., and L. De Lai. 1997. Population declines of predatory birds coincident with the introduction of Klerat rodenticide in North Queensland. Australian Bird Watcher 17:160-167.