Encapsulated sodium nitrite as a new toxicant for possum control in New Zealand

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Abstract: Sodium nitrite (NaNO₂), a commonly used food preservative, has been researched in New Zealand for the control of brushtail possums (*Trichosurus vulpecula*). In sufficiently high doses, NaNO₂ is toxic because it disrupts circulatory transport of oxygen. As NaNO₂ is very bitter, encapsulation and mixing it through a highly palatable bait formulation is necessary to effectively deliver it to target pest species. In no-choice cage trials, 12/12 possums consumed a lethal dose of toxic paste bait and died on average after 95.6 minutes (± 4.9 SE). In two-choice cage trials 7/8 possums consumed a lethal dose of toxic paste bait and died on average after 96.7 minutes (± 11.4 SE). Two field trials targeting possums using this toxic paste in bait stations reduced their abundance by 81.2% ($\pm 2.5\%$ SE) and 72.7% ($\pm 1.6\%$ SE) respectively. NaNO₂ paste, known as BaitRite, has been registered in New Zealand as a vertebrate toxic agent for controlling possums.

Keywords: brushtail possum; methaemoglobin; methaemoglobinaemia; NaNO₂; vertebrate pesticide

Introduction

In New Zealand, common brushtail possums (Trichosurus vulpecula) are a threat to native biodiversity, through the damage they cause to flora and fauna (Innes et al. 2004; Glen et al. 2012; Nugent & Morriss 2013). They also threaten the primary sector through their role as the main wildlife vector of bovine tuberculosis (Mycobacterium bovis) (Coleman & Caley 2000; PCE 2011). Possums are controlled with a variety of traps and toxins, including ground-based control with the toxins cyanide, brodifacoum and cholecalciferol, as well as aerial control with sodium fluoroacetate (1080). Control work is often undertaken on or in close proximity to farmland. When these operations are undertaken, there is therefore a risk of secondary poisoning to non-target species, including working dogs, from carcasses of poisoned possums (Meenken & Booth 1997; Eason 2002; Eason et al. 2011). Research to minimise this risk has focused on developing vertebrate toxic agents (VTAs) that have low residue, low risk to non-target animals, and animal welfare as a key consideration (Morgan et al. 2013; Eason et al. 2014; Shapiro et al. 2016b).

One compound researched for possum control has been sodium nitrite (NaNO₂), an inorganic salt commonly used to add colour and flavour to food for human consumption and as an antimicrobial agent in cured and processed meats (Binkerd & Kolari 1975; Hord et al. 2009). The chemistry and toxicology of NaNO₂ is well understood due to the numerous documented cases of accidental poisoning of humans and animals (Counter et al. 1975; Bradberry et al. 1994; Gautami et al. 1995; Vyt & Spruytte 2006). NaNO₂ has also been researched as a potential VTA for feral pigs (*Sus scrofa*) (Sullivan 1985; Cowled et al. 2008; Shapiro et al. 2016a). Research in New Zealand expanded on that of Cowled et al. (2008), carried out in Australia, and investigated the utility of NaNO₂ as a potential control tool for possums and feral pigs.

NaNO₂ has been referred to as a red blood cell toxicant (Eason & Ogilvie 2009) due to its mode of action. The protein

haemoglobin, found in red blood cells and responsible for oxygen transport, has an alternate form called methaemoglobin (MetHb) and normally accounts for less than 2% of the total haemoglobin circulating at one time (Fan et al. 1987; Bradberry 2011). Ingestion of NaNO₂ causes an elevation in the levels of MetHb (Beutler & Mikus 1961) and in high enough doses this leads to methaemoglobinaemia. Chui et al. (2005) describe methaemoglobinaemia as the potentially fatal condition where the oxidation of haemoglobin to MetHb negates its ability to bind and transport oxygen. Levels of MetHb <20% of total haemoglobin are usually asymptomatic (Bradberry 2011). At levels higher than this, symptoms of methaemoglobinaemia appear and, in humans and possums, include a bluish grey skin colour, lethargy, cerebral anoxia, chocolate-coloured blood, irregular breathing, loss of consciousness. Levels above 80% can be fatal (Fan et al. 1987; Brunning-Fann & Kaneene 1993; Fisher et al. 2008). The treatment of methaemoglobinaemia as outlined by Umbreit (2007) commonly involves the infusion of methylene blue, a compound routinely used to treat nitrate poisoning in cattle (Bolan & Kemp 2003). Following treatment with methylene blue, a rapid improvement is usually seen 30-60 minutes after its administration (Chui et al. 2005).

Lapidge and Eason (2010) noted from previous research that the lethal doses for humans, rats, and pigs, administered NaNO₂ by oral gavage, were approx. 100 mg/kg. Based on this, a 3-kg possum would require 300 mg of NaNO₂ for a lethal dose; however, this figure is based on oral gavage not delivered in bait. For the research reported here, we wanted to ensure there was a low chance of sub-lethally dosing possums and our aim was to exceed the oral gavage lethal dose several fold, paste baits containing 10% w/w sodium nitrite were trialled.

The low palatability of NaNO $_2$ was observed in early cage trials carried out by Shapiro et al. (2009), where raw NaNO $_2$ (10% w/w), mixed in paste bait, was fed to possums. Only four out of 12 possums consumed any bait, and in each case it was insufficient for a toxic effect to be observed. A proprietary encapsulation technique (Connovation Ltd) was applied to

 $NaNO_2$ for the purpose of taste masking. The purpose of the research reported here was to determine the effectiveness of encapsulated $NaNO_2$ in a paste bait, containing 10% w/w of the active ingredient, for possum control.

Materials and methods

Possum cage trials

Twenty wild common brushtail possums were captured using Victor leg-hold traps in Hororata, Canterbury, New Zealand in summer. Possums were housed individually in indoor cages at the Johnstone Memorial Animal Facility, Lincoln University. Cages were kept in a temperature controlled room (19°C \pm 5°C) and were constructed of stainless steel and measured 110 cm \times 55 cm \times 60 cm; each had a plastic box for possums to use as a den. The room lighting was kept under natural daylength lighting. Possums underwent a health check on arrival during which they were weighed, sexed, and females were screened for pouch young. Possums were fed solid pellets, made of various grains, as well as fresh vegetables, and water was available ad libitum.

Possums were acclimatised for 1 week to ensure they were eating and that their weight remained stable. Once acclimatised, the pellets and vegetables were removed from cages and individuals were fed 50 g of a non-toxic paste (Connovation Ltd), consisting of a mixture of peanut butter (35%), kibbled wheat (20%), ground maize (15%), margarine (15%), and sugar (15%). This was undertaken on two occasions 3 days apart in the week leading up to the toxic trials. Possums received the standard pellet and vegetable diet on the days between being fed the non-toxic paste, apart from the 24 hours immediately before the toxic trials where they received no vegetables and half rations of pellets. Two cage trials were conducted. The first was a no-choice bait acceptance trial to determine whether possums would consume sufficient toxic bait to receive a lethal dose. The second, a two-choice trial, was to test whether possums would still consume a lethal dose of toxic paste when also presented with non-toxic paste. For both trials the length of exposure to toxic baits was determined from the acclimatisation period, on these two occasions all possums consumed the entire 50 g of non-toxic paste bait within 4 hours.

No-choice trial

Once acclimatised to the non-toxic paste, 12 possums (six male and six female; weight range 1.78 – 3.20 kg) were each presented with approx. 50 g of the NaNO₂ paste (Connovation Ltd). Toxic bait was placed in a metal feed tray with a single tray in each cage. Each possum was observed with a single LTL Acorn 5210A motion-detecting video camera left in the cage, and footage was observed at the end of the trial. Toxic baits were weighed again after trays had been left in cages for four hours. Possums were left undisturbed for the first hour and feeding and behaviour was recorded solely via camera. For the remainder of the trial, possums were monitored by camera as well as closely observed by a researcher every 15 minutes. They were specifically monitored for symptoms of methaemoglobinaemia, which include pale extremities (including nose and gums), lethargy, ataxia, shortness of breath and tremors. The time to the onset of these symptoms, the duration of symptoms, and the time to death were also recorded.

Two-choice trial

Once acclimatised to the non-toxic paste, eight possums (four male and four female; weight range $1.60-4.18~{\rm kg}$) were each presented with approx. 50 g of the paste bait containing NaNO₂ (Connovation Ltd), the same formulation as the nochoice trial. Each possum was also presented with approx. 50 g of the non-toxic paste. The two forms of the paste bait were placed in separate compartments of a metal feed tray and this was randomised to avoid any potential effect of animals being conditioned to feeding from a particular compartment. Each possum was observed with a single LTL Acorn 5210A motion-detecting video camera left in the cage, and footage was observed at the end of the trial. Both bait types were weighed again after trays had been left in the cages for 4 hours. All monitoring of possums during this time was identical to that of the no-choice trial.

Possum field trials

Two field trial sites were established on two privately owned farms located in Canterbury, New Zealand. Field trial site one was located approx. 15 km west of Little River on Banks Peninsula (43°79' S, 172°70' E). Site two was located approx. 25 km north-east of Little River on Banks Peninsula (43°72' S, 173°08' E). Each site consisted of a treatment and a nontreatment area located 1.5 km apart within each trial site, each area was approx. 100 ha. Vegetation at both trial sites consisted of open pasture, mānuka (*Leptospermum scoparium*), tōtara (*Podocarpus totara*), various *Coprosma* species, the native tree nettle ongaonga (*Urtica ferox*), and regenerating scrubland.

The trial at site one took place over a 4-week period during June 2010, with an average overnight temperature during this period of 5.9°C (min 2.9°C and max 10.3°C). Total rainfall for the duration of the trial was 93.0 mm (NIWA, 2010). The trial at site two took place over a 4-week period from mid-June 2010 to mid-July 2010, with an average overnight temperature during this period of 5.4°C (min 2.2°C and max 9.5°C). Total rainfall for the duration of the trial was 72.6 mm (NIWA, 2010).

Relative possum abundance was measured before and after control using the NPCA Waxtags® protocol (NPCA 2010) in the treatment and non-treatment areas for both sites. Using this method, relative possum abundance is calculated as a percentage of the Waxtags® bitten by possums and recorded as a Bite Mark Index (BMI). In the treatment and non-treatment areas at each site Waxtags® were deployed on five lines with 20 tags per line and 10 m between tags. Waxtags® were left out for 7 nights and then retrieved.

In the treatment area of both sites bait stations were set up at approx. 100 m intervals, on lines spaced 150 m apart in areas where vegetation was sparse and 100 m apart in areas of thicker scrub. A total of 83 mini Philproof® bait stations were set out across site one and 65 across site two. Prefeeding at both treatment sites was carried out using non-toxic paste (Connovation Ltd), consisting of a mixture of peanut butter (35%), kibbled wheat (20%), ground maize (15%), margarine (15%), and sugar (15%). On three occasions, at 1-week intervals, approx. 200 g of this non-toxic paste was placed in each bait station. One week after the last pre-feed, any remaining non-toxic paste was removed from the bait stations and replaced with approximately 130 g of NaNO2 paste (Connovation Ltd). This replacement paste consisted of 90% non-toxic paste and 10% w/w NaNO2. Baits were checked every 2 days and replenished wherever there was less than half the original amount left in a bait station. Baits

were left out for 4 nights, after which time they were removed from bait stations.

Samples of the encapsulated NaNO $_2$ active and NaNO $_2$ paste were analysed by Flinders Cook Ltd (Technical Services) to confirm the concentration of NaNO $_2$ active before each of the trials. Samples of the encapsulated NaNO $_2$ contained 95% w/w NaNO $_2$ active and 5% encapsulant material. Samples of the NaNO $_2$ paste contained 10.0% \pm 0.3% NaNO $_2$. The method of analysis was based on an internationally recognised analytical method described in Vogel (1979).

Results

No-choice efficacy trial

In the no-choice efficacy cage trial all 12 possums (100%; 95% binomial CI 73.5 – 100%) died after consuming NaNO₂ paste Possums consumed an average of 9.49 g (\pm 1.36 SE) of bait, an average dose per possum of 360.8 mg/kg (\pm 49.75 SE) of NaNO₂(Table 1). Clinical signs first appeared on average after 20.6 minutes (\pm 1.8 SE) and possums died on average 95.6 minutes (\pm 4.9 SE) after ingesting bait. Symptoms observed included pale noses, pale gums, lethargy, ataxia, slight tremors, collapse, and death.

Two-choice trial

In the two-choice cage trial seven of the eight possums (87.5%; 95% binomial CI 47.4 – 99.7%) consumed a lethal dose of NaNO₂ paste Those seven possums consumed an average of 8.41 g (\pm 2.2 SE) of bait, an average dose per possum of 260.5 mg/kg (\pm 64.8 SE) of NaNO₂ (Table 2). Clinical signs first appeared on average after 24.0 minutes (\pm 2.9 SE) and possums died on average 96.7 minutes (\pm 11.4 SE) after ingesting bait. One of the eight possums did not consume a lethal dose of toxic paste (Table 2) but displayed clinical symptoms, including a pale nose and gums as well as being lethargic, for 45 minutes before recovering and was euthanased at the conclusion of the trial in line with our animal ethics approval document. Based on consumption, the relative palatability of the NaNO₂ paste was 66.3% compared with 33.8% for the non-toxic paste.

Using the data generated in these two cage trials and from a previous pilot trial with four possums (Hix et al, 2010), an LD_{50} for possums free feeding on paste bait containing $NaNO_2$ (10% w/w) was calculated as 121.6 mg/kg (95% CI 45.4 – 169.6 mg/kg).

Possum field trials

Before the toxic trial, possum abundance at site one was found to be 85.0% BMI (\pm 6.7 SE) in the treatment area and

Table 1. Time to appearance of symptoms, duration and time to death for possums that consumed NaNO₂ paste (cage trials).

Possum	Sex	Weight (kg)	Bait consumed (g)	Dose (mg/kg)	First appearance clinical symptoms (mins)	Time to death (mins)
1	M	2.87	11.15	388.50	15	104
2	M	2.46	8.07	328.05	35	78
3	M	3.02	14.01	463.91	15	107
4	M	1.78	6.04	339.33	22	89
5	M	2.22	6.27	282.43	23	79
6	M	3.05	6.84	224.26	20	130
7	F	3.20	6.39	199.69	30	114
8	F	2.09	6.97	333.49	16	72
9	F	3.08	8.96	290.91	20	96
10	F	2.38	5.04	211.76	15	103
11	F	2.56	21.54	741.41	20	80
12	F	2.96	12.62	426.35	16	95

Table 2. Time to appearance of symptoms, duration and time to death in possums presented non-toxic paste and NaNO₂ paste (cage trials).

Possum	Sex	Weight (kg)	Non-toxic paste consumed (g)	NaNO ₂ paste consumed (g)	Toxic dose (mg/kg)	Appearance clinical symptoms (mins)	Time to death (mins)
1	M	2.35	0.36	13.66	581.28	16	65
2	M	2.85	0.26	8.85	310.53	31	115
3	M	4.18	0.84	17.84	426.79	29	107
4	M	2.68	13.16	3.87	144.40	14	60
5	F	2.40	0.05	5.11	212.92	26	105
6	F	1.60	12.59	1.72	107.50	19	80
7	F	3.57	0.00	7.81	218.77	33	145
8	F	3.48	43.37	2.85	81.90	16	Recovered

79.0% BMI (\pm 6.0 SE) in the non-treatment area. At site two possum abundance was found to be 77.0% BMI (\pm 6.1 SE) in the treatment area and 86.0% BMI (\pm 1.9 SE) in the non-treatment area. Post-monitoring, undertaken immediately after the toxic trial, found that possum abundance at site one in the treatment area had reduced significantly (t_4 =11.09, P<0.01) to 16.0% BMI (\pm 1.0 SE). This represents a decrease in possum abundance of 81.2% (\pm 1.5 SE). Post-monitoring at site two found that possum abundance in the treatment area had also reduced significantly (t_4 =9.68, P<0.01) to 21.0% BMI (\pm 1.9 SE). This represents a decrease in possum abundance of 72.7% (\pm 3.0 SE).

There was no significant change in possum abundance in the control area of site one (t_4 =-2.44, p = 0.07) or site two (t_4 =1.20, p = 0.29). Post-treatment possum abundance was 85.0% BMI (± 4.5 SE) at site one and 81.0% BMI (± 1.0 SE) at site two.

Discussion

An encapsulated form of NaNO₂ has been developed, which has been shown, in small-scale pen and field trials, to be palatable and effective for the control of possums when presented in paste bait. The encapsulation of NaNO₂ has improved its effectiveness in possums compared with results from previous trials where possums were presented with unencapsulated NaNO₂ in the same paste bait matrix (Shapiro et al. 2009).

Sufficient sodium nitrite needs to be ingested quickly to induce fatal methaemoglobinaemia and death. NaNO₂ at high doses induces a relatively fast time to death, in possums, compared with conventional VTAs such as 1080, brodifacoum and cholecalciferol (McIlroy 1983; Jolly et al. 1993; Littin et al. 2000, 2002) but comparable to those times observed for stoats (*Mustela erminea*) and feral cats (*Felis catus*) poisoned with PAPP (Savarie et al. 1983; Eason et al. 2010).

Due to its use in food preservation, a large amount of data exists on the metabolism of NaNO₂ by humans as well as numerous other species. The time for nitrite to be eliminated from the blood is expressed in terms of plasma elimination half-life ($t_{1/2}$). Results for various species, in minutes, were collated from previous research by Lapidge and Eason (2010) and reported for sheep (29), dogs (30), ponies (34) (Schneider & Yeary 1975) and humans (42.1 ± 10.2) (Dejam et al. 2007). The excretion of nitrite is both rapid and extensive and therefore it is not accumulated in tissues (European Food Safety Authority 2009). The metabolism data indicate that in the short time from ingesting toxic NaNO₂ baits to death, possums are likely to excrete a sizeable amount of NaNO₂, and in turn present a low risk of secondary poisoning for animals that scavenge the carcasses of possums poisoned with NaNO₂.

The development of new toxins and control tools, as well as the refinement of existing control techniques, is essential to enhancing our ability to effectively administer bovine tuberculosis control and native flora and fauna protection. In the context of animal pest control in New Zealand, encapsulated NaNO2 has been shown to have suitable efficacy as an additional tool for possum control. In November 2013, NaNO2 paste, known as Bait-Rite paste (ACVM V009563), was registered in New Zealand as a VTA for the control of possums.

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References

- Beutler E, Mikus BJ 1961. The effect of methaemoglobin formation in sickle cell disease. Journal of Clinical Investigation 40: 1857–1871.
- Binkerd EF, Kolari OE 1975. The history and use of nitrate and nitrite in curing of meat. Food and Cosmetic Toxicology 13: 655–651.
- Bolan NS, Kemp PD 2003. A review of factors affecting and prevention of pasture-induced nitrate toxicity in grazing animals. Proceedings of the New Zealand Grassland Association 65: 171–178.
- Bradberry S 2011. Methaemoglobinaemia: complications of poisoning. Medicine 40(2): 59–60.
- Bradberry S, Gazzard B, Vale A 1994. Methaemoglobinaemia caused accidental contamination of drinking water with sodium nitrite. Journal of Toxicology: Clinical Toxicology 32: 173–178.
- Brunning-Fann CS, Kaneene JB 1993. The effects of nitrate, nitrite and N-nitroso compounds on human health: a review. Veterinary and Human Toxicology 35: 521–538.
- Chui JSW, Poon WT, Chan KC, Chan AYW, Buckley TA 2005. Nitrite-induced methaemoglobinaemia—aetiology, diagnosis and treatment. Anaesthesia 60: 496–500.
- Coleman J, Caley P 2000. Possums as a reservoir of bovine Tb. In: Montague TL ed The brushtail possum: biology, impact and management of an introduced marsupial. Lincoln, Manaaki Whenua Press. Pp. 92–104.
- Counter DE, Giles N, Redmond R 1975. Stored rainwater as a cause of nitrite poisoning in pigs. Veterinary Record 96: 412.
- Cowled BD, Elsworth P, Lapidge SJ 2008. Additional toxins for feral pig (*Sus scrofa*) control: identifying and testing Achilles' heels. Wildlife Research 35: 1–12.
- Dejam A, Hunter CJ, Tremonti C, Pluta RM, Hon YY, Grimes G, Partovi K, Pelletier MM, Oldfield EH, Cannon RO, Schechter AN, Gladwin MT 2007. Nitrite infusion in humans and nonhuman primates: endocrine effects, pharmacokinetics, and tolerance formation. Circulation 116: 1821–1831.
- Eason CT 2002. Technical review of sodium monofluoroacetate (1080) toxicology. Wellington, Animal Health Board. ISBN 0-478-09346-2. 25 p.

- Eason CT, Ogilvie SC 2009. A re-evaluation of potential rodenticides for aerial control of rodents. DOC Research & Development Series 312. 34 p.
- Eason CT, Murphy E, Hix S, MacMorran D 2010. The development of a new humane toxin for predator control. Integrative Zoology 1: 443–448.
- Eason CT, Miller A, Ogilvie S, Fairweather A 2011. An updated review of the toxicology and ecotoxicology of sodium fluroacetate (1080) in relation to its use as a pest control tool in New Zealand. New Zealand Journal of Ecology 35: 1–20.
- Eason CT, Miller A, MacMorran D, Murphy EA 2014. Toxicology and ecotoxicology of PAPP for pest control in New Zealand. New Zealand Journal of Ecology 38: 177–188.
- European Food Safety Authority 2009. Scientific Opinion of the Panel on Contaminants in the Food Chain on a request from the European Commission on nitrite as undesirable substances in animal feed. The EFSA Journal 1017. Pp. 1–47.
- Fan AM, Willhite CC, Book SA 1987. Evaluation of the nitrate drinking water standard with reference to infant Methemoglobinemia and potential reproductive toxicology. Regulatory Toxicology and Pharmacology 7: 135–148.
- Fisher P, O'Connor CE, Morriss G 2008. Oral toxicity of p-aminopropiophenone to Brushtail Possums (*Trichosurus vulpecula*), dama wallabies (*Macropus eugenii*), and mallards (*Anas platyrhynchos*). Journal of Wildlife Diseases 44: 655–663.
- Gautami S, Rao RN, Raghuram TC, Rajagopalan S, Bhat RV 1995. Accidental acute fatal sodium nitrite poisoning. Journal of Toxicology: Clinical Toxicology 33: 131–133.
- Glen AS, Byrom AE, Pech RP, Cruz J, Schwab A, Sweetapple P, Yockney I, Nugent G, Coleman M, Whitford J 2012. Ecology of brushtail possums in a New Zealand dryland ecosystem. New Zealand Journal of Ecology 36: 29–37.
- Hix S, MacMorran D, Shapiro S, Eason C 2010. Pilot trial of the effectiveness of encapsulated sodium nitrite in 213 paste on possums and rats. Connovation Limited Internal Report. 5 p.
- Innes J, Nugent G, Prime K, Spurr EB 2004. Responses of kukupa (*Hemiphaga novaeseelandiae*) and other birds to mammal pest control at Motatau, Northland. New Zealand Journal of Ecology 28: 73–81.
- Jolly SE, Eason CT, Frampton C 1993. Serum calcium levels in response to cholecalciferol and calcium carbonate in the Australian brushtail possum. Pesticide Biochemistry and Physiology 47: 159–164.
- Lapidge SJ, Eason CT 2010. Pharmacokinetics and methaemoglobin reductase activity as determinants of species susceptibility and non-target risks from sodium nitrite manufactured feral pig baits. Report for the Australian Government Department of the Environment, Water, Heritage and the Arts, Canberra, Australia. 18 p.

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- Littin KE, O'Connor CE, Eason CT 2000. Comparative effects of brodifacoum on rats and possums. Proceedings New Zealand Plant Protection Conference 53: 310–315.
- Littin KE, O'Connor CE, Gregory NG, Mellor DJ, Eason CT 2002. Behaviour, coagulopathy and pathology of brushtail possums (*Trichosurus vulpecula*) poisoned with brodifacoum. Wildlife Research 29: 259–267.
- McIlroy JC 1983. The sensitivity of the brushtail possum (*Trichosurus vulpecula*) to 1080 poison. New Zealand Journal of Ecology 6: 125–131.
- Meenken D, Booth LH 1997: The risk to dogs of poisoning from sodium monofluoroacetate (1080) residues in possum (*Trichosurus vulpecula*). New Zealand Journal of Agricultural Research 40: 573–576.
- Morgan D, Arrow J, Smith MP 2013. Combining Aspirin with cholecalciferol (Vitamin D3) A potential new tool for controlling possum populations. PLoS ONE 8(8) DOI: 10.1371/journal.pone.0070683.
- PCE 2011. Evaluating the use of 1080: predators, poisons and silent forests. Parliamentary Commissioner for the Environment, Wellington. 85 p.
- NIWA 2010. CliFlo: NIWA's National Climate Database on the Web. http://cliflo.niwa.co.nz/ (accessed 5 July 2010)
- NPCA 2010. Possum population monitoring using the Waxtag® method. Wellington, National Possum Control Agencies. 28 p.
- Nugent G, Morriss GA 2013. Delivery of toxic bait in clusters: a modified technique for aerial poisoning of small mammal pests. New Zealand Journal of Ecology 37: 246–255.
- Savarie PJ, Ping Pan H, Hayes DJ, Roberts JD, Dasch GJ, Felton R, Schafer EW 1983. Comparative acute oral toxicity of para-aminopropiophenone. Bulletin of Environmental Contamination and Toxicology 30: 122–126.
- Schneider NR, Yeary RA 1975. Nitrite and nitrate pharmacokinetics in the dog, sheep and pony. American Journal of Veterinary Research 36: 941–947.
- Shapiro L, Hix S, Eason CT, MacMorran D 2009. Palatability and efficacy of sodium nitrite in a paste bait to possums and pigs in cage trials. Connovation Ltd. Unpublished report. 10 p.
- Shapiro L, Eason C, Bunt C, Hix S, Aylett P, MacMorran D 2016a. Efficacy of encapsulated sodium nitrite as a new tool for feral pig management. Journal of Pest Science 89: 489–495.
- Shapiro L, MacMorran D, Ross J, Eason C 2016b. Early field experience with microencapsulated zinc phosphide paste for possum ground control in New Zealand. New Zealand Journal of Ecology 40: 386–389.
- Sullivan RM 1985. Assessment of sodium nitrite as a poison for the feral pig (*Sus scrofa*). Thesis submitted as partial requirement of the degree of Bachelor of Rural Science, University of New England, Armidale NSW, Australia.
- Umbreit J 2007. Methaemaglobin it's not just blue: a concise review. American Journal of Hematology 82: 134–144.
- Vogel AI 1979. Vogel's textbook of quantitative inorganic analysis. 4th edn. London, Longman. 356 p.
- Vyt P, Spruytte H 2006. Nitrite intoxication in sows. The Veterinary Record 158: 456.