PEST CONTROL AND INVERTEBRATES

FACTSHEET RD19

Possums are regarded as a pest species in New Zealand because they are main wildlife vector of bovine tuberculosis (TB). Populations are controlled as part of New Zealand's TBfree pest control programme, which has the secondary benefit of impeding the destruction of native biodiversity.

Possum populations are controlled by the use of trapping and poisoning. While trapping is not considered to have negative effects on invertebrates (ie any animal without a backbone such as insects, snails or worms), the application of poison bait has the potential to expose invertebrates to a range of pesticides used for possum or rodent control. These include sodium fluoroacetate (1080), anticoagulants (such as brodifacoum, pindone and diphacinone) and cyanide.

IMPACTS OF WILDLIFE AND PEST CONTROL ON NATIVE INVERTEBRATES

Numerous studies have shown that introduced mammals are an important cause of the decline or disappearance of native invertebrates. In particular, larger, flightless, ground-dwelling nocturnal species have suffered most (for example, giant weta). These groups disappeared rapidly from the mainland after mammals invaded New Zealand. For example, the Cook Strait giant weta became extinct on the lower North Island over 100 years ago but it has survived on mammal-free islands in Cook Strait.

The 'Māori' or Polynesian rat (kiore arrived) in New Zealand about 700 years ago; 150 years before a diversity of other mammals became established. Only invertebrates with resistant traits (eg high fecundity, small body size) have survived the onslaught of predators on the mainland.

Some studies have found that mammal control alters invertebrate abundance, species richness, diversity, and behaviour. Some invertebrate groups – consistently the larger invertebrates – may recover strongly. However, other studies found no response to pest control[1]. Auckland tree weta increased threefold for three years with repeated aerial 1080 operations but declined when rat densities rebounded[2]. Furthermore, mammal predators may be replaced by native predators, especially birds, so interpreting changes in the invertebrate community is difficult; these communities and their interactions are poorly understood.

INVERTEBRATES AND EXPOSURE TO POISONS

1080

1080 has high toxicity to invertebrates and can kill individual invertebrates that ingest enough bait. Current aerial application rates of bait (approximately 120 bait pellets per hectare) mean that relatively few invertebrates are exposed, so that non-target invertebrate mortality is not sufficient to have an effect at a population level. However, presentation of 1080 bait in stations has the potential to cause localised declines in invertebrate populations if invertebrates consistently are attracted to a source of bait that is present in the same place over time.

More than 180 different invertebrate groups have been found feeding on baits, but only a small proportion of baits had invertebrates on them any time, and then with only a few individuals per bait. There is no evidence that 1080-poisoning operations have any adverse impacts on invertebrate communities at a whole-forest scale[3].

Other studies have shown that aerial 1080 poison operations did not affect invertebrates at a population level (including tree weta, cave weta, cockroaches, spiders, harvestman and leaf-veined slugs) in refuges above the ground. In addition, there was no detectable change in the numbers of individually marked tree weta seen after bait application.

Anticoagulants

Captive studies indicate low toxicity of anticoagulants to invertebrates [4-6]. For example, captive locusts that fed readily on cereal-based brodifacoum baits showed no significant increase in mortality. In captive weta, weight loss and mortality was not significantly higher in weta exposed to brodifacoum bait over 60 days[5]. On this basis, consumption of anticoagulant bait by invertebrates is not expected to result in high mortality. The potential role of invertebrates as environmental vectors of residual anticoagulants is then dependent upon the persistence of residues in their tissues.

Limited data are available regarding anticoagulants in invertebrates but generally indicate lower persistence than in mammals. Following sub-lethal doses, brodifacoum residues were not detectable after four days in captive weta[4] and captive locusts appeared to excrete brodifacoum rapidly, indicating that long-term bioaccumulation was unlikely[6].

Cyanide

While cyanide is expected to have high toxicity to invertebrates that ingest it, current possum bait formulations of cyanide (paste, encapsulated pellets) are not attractive to invertebrates, and non-target invertebrate mortality is not associated with the use of this cyanide bait. Cyanide itself is highly reactive and not persistent in animal tissues.

INVERTEBRATES AS SECONDARY SOURCES OF PESTICIDE RESIDUE

1080

Invertebrates that have fed on pellets represent a potential source of secondary exposure to insectivores (animals that eat insects)[7]. Similarly invertebrates that scavenge the carcasses of poisoned animals may also acquire residues. To date, there have been no field-based evaluations of the potential risk presented to insectivores by 1080 in this context.

Invertebrates can metabolise and excrete sublethal exposures of 1080, so secondary risk will have a limited timeframe. In laboratory studies, tree weta eliminated >90% of a sublethal oral dose 1080 within 4–6 days[8]. Ants sublethally dosed with 1080 had low but detectable levels of 1080 seven days after dosing[9].

Anticoagulants

In the case of brodifacoum, invertebrates that have fed on bait have been shown to act as vectors of residues to birds, with some cases where brodifacoum was applied for rodent eradication resulting in bird mortality through secondary poisoning[10]. The extent to which this occurs as the result of possum and rodent baiting operations is currently not well understood.

REFERENCES

[1] Byrom AE, Innes J, Binny RN 2016. A review of biodiversity outcomes from possum-focused pest control in New Zealand. *Wildlife Research* 43: 228-253.

[2] Ruscoe W, Sweetapple P, Perry M, Duncan R 2012. Effects of spatially extensive control of invasive rats on abundance of native invertebrates in mainland New Zealand forests. *Conservation Biology* 27: 74–82.

[3] Spurr E 2012. 1080 impacts on invertebrate populations: A review and response to Benfield (2011). *New Zealand Journal of Forestry* 56: 46–47.

[4] Booth LH, Eason CT, Spurr EB 2001. Literature review of the acute toxicity and persistence of brodifacoum to invertebrates and studies of residue risks to wildlife and people. Science for Conservation 177. Wellington, Department of Conservation.

[5] Bowie M, Ross J 2006. Identification of weta (Orthoptera: Anostomatidae and Rhaphidophoridae) foraging on brodifacoum cereal bait and the risk of secondary poisoning for bird species on Quail Island, New Zealand. *New Zealand Journal of Ecology* 30: 219–228.

[6] Craddock P 2003. Aspects of the ecology of forest invertebrates and the use of brodifacoum. Unpublished PhD thesis, University of Auckland, Auckland, New Zealand.

[7] Lloyd BD, McQueen SM 2000. An assessment of the probability of secondary poisoning of forest insectivores following an aerial 1080 possum control operation. *New Zealand Journal of Ecology* 24: 47-56.

[8] Eason CT, Gooneratne R, Wright GR, Pierce R, Frampton CM 1993. The fate of sodium monofluoroacetate (1080) in water, mammals, and invertebrates. Proceedings of the New Zealand Plant Protection Conference 46: 297-301.

[9] Booth LH, Wickstrom ML 1999. The toxicity of sodium monofluoroacetate (1080) to *Huberia striata*, a New Zealand native ant. *New Zealand Journal of Ecology* 23: 161-165.

[10] Dowding JE, Lovegrove TG, Ritchie J, Kast SN, Puckett M 2006. Mortality of northern New Zealand dotterels (*Charadrius obscurus aquilonius*) following an aerial poisoning operation. *Notornis* 53: 235-259.

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