

Effects of rodent poisoning on *Powelliphanta traversi*

Shaun J. Bennett, Rachel J. Standish* and Ian A.N. Stringer

Ecology Group, Institute of Natural Resources, Massey University, Private Bag 11 222, Palmerston North, New Zealand

*Present address (& author for correspondence): Landcare Research, Private Bag 6, Nelson, New Zealand.

ABSTRACT

Rat predation is a threat to lowland *Powelliphanta traversi* (giant predatory land snail), and we have shown that 'press' poisoning of rodents (rats and mice) using brodifacoum baits significantly reduces rat abundance relative to non-poisoned areas. The effect on *P. t. traversi* was evident by the increase in population size, mainly due to adult migration, and a decrease in rat-damaged shells, for areas where rat predation occurs. A longer-term study is required to determine whether prolonged rat control benefits *P. t. traversi* recruitment. Mouse control was inadequate with use of brodifacoum baits. We document a concomitant rise in bird predation of *P. traversi* when rat abundance was reduced, suggesting that control of both is necessary to make real conservation gains. However, mortality related to other factors was more common than that caused by predators, possibly due to the habitat drying out periodically. We suggest that low recruitment rates, predator targeting of juveniles (i.e. blackbirds, song thrush and possibly mice and hedgehogs) and poor habitat conditions are the main threats to survival of lowland *P. traversi*.

Keywords: bird predation, blackbirds, brodifacoum, endangered land snails, Lake Papaitonga Scenic Reserve, mouse predation, *Powelliphanta traversi*, snail mortality, pest control, rat predation, snail recruitment, rodent poisoning, song thrush

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1. Introduction

New Zealand's native biota is characterised by a high degree of endemism because of its Gondwanan ancestry, long isolation from other land masses and the absence of most mammals (Cooper & Millener 1993; Daugherty et al. 1993). New Zealand lacked land mammals other than bats and seals until around 1000–1200 years ago (King 1990), so many of the traits that developed in the absence of mammalian predators (e.g. gigantism, low reproductive rates) have left endemic species vulnerable to predation after the arrival of small mammalian and avifauna predators (Daugherty et al. 1993). The introduction of large mammals has greatly affected New Zealand's vegetation (Campbell 1978), vertebrate biota (Atkinson 1978) and endemic invertebrate fauna (Ramsay 1978). The introduction of vertebrate predators in particular has caused a widespread decline in both the numbers of species of snails and the distributions of three genera with gigantic form: *Powelliphanta*, *Paryphanta* and *Placostylus* (Meads et al. 1984). Rats, mice, brush-tailed possums, pigs, hedgehogs, mustelids, blackbirds, song thrushes and cats have been cited as likely predators of *Powelliphanta* (Powell 1946; Meads et al. 1984; Walker 1997; Coad 1998; Efford 1998). All of these animals occur in the Horowhenua district.

All four rodent species (Muridae) in New Zealand have had a considerable impact on invertebrates throughout New Zealand (Ramsay 1978). Kiore (*Rattus exulans* Peale) were implicated in the extinction of two species of large land snails (*Placostylus hongii* Lesson, *Amborbytida tarangensis* Powell) on Lady Alice Island (Brook 1999), as well as in mainland extinctions and reductions of an unknown number of large invertebrates including land snails (Atkinson & Moller 1990). Meads et al. (1984) reported evidence of kiore predation on *Powelliphanta*, but the full impact on small animals in mainland New Zealand can only be inferred, because kiore disappeared from most of the North Island and northern South Island by the late 1800s (Atkinson 1973). Evidence of snail predation by both ship rat (*Rattus rattus* L.) and Norway rat (*Rattus norvegicus* Berkenhout) is more substantial (Meads et al. 1984; Devine 1997; Walker 1997; Sherley et al. 1998). In the past, rat depredations (either species) have accounted for a large proportion of damaged *Powelliphanta traversi traversi* shells (Meads et al. 1984; Devine 1997). Recently, we found little evidence of rat damage (Standish et al. 2002) but we did not search for burrow chambers where rats are known to cache snails (Meads et al. 1984). Mice (*Mus musculus* L.) have been implicated as predators of New Zealand's large land snails (Climo 1975), but there is no evidence that they are a significant predator of *Powelliphanta* (Meads et al. 1984; K. Walker, pers. comm.). However, we found that mice were successfully able to attack baited *P. t. traversi* shells smaller than 27 mm when they were offered in a laboratory feeding trial (Bennett 2001). We do not know whether or not mice attack live *Powelliphanta* in the field.

Much of New Zealand's modern conservation effort has gone towards the control or eradication of rodents. Poison has been widely used to eradicate them successfully, especially in offshore island operations (Veitch 1992). An important step in the success of these poisoning operations was the development of second-generation anticoagulant poisons such as brodifacoum,

which can kill after a single feed and allow a rodent to consume a lethal dose long before they suffer toxic effects. This prevents the development of bait shyness that can follow a sub-lethal dose or doses (Taylor 1992). Brodifacoum was used in 28 of 33 recent mammal eradication programmes undertaken by DOC on offshore islands, and the use of this poison nationally has been increasing (Innes & Barker 1999). It has also been used to control mice in crop fields (Brown & Singleton 1998), bush, scrub and pasture (Dowding et al. 1999).

Studies of the effects of rodents on land snails have shown variable benefits of rodent control. Sherley et al. (1998) found that juveniles of a subspecies of *Placostylus ambagiosus* (Suter) with shells larger than 10 mm increased in number following eight years of 'pulse' (i.e. intermittent) poisoning of rodents. Surveys of *Powelliphanta t. otakia* during 18 years of rodent control have shown a significant increase in snail numbers in the first ten years, but variable results since (Walker 1997; K. Walker, pers. comm.). However, neither study assessed the number of rodents present prior to, or during poisoning, so the effectiveness of poisoning was unknown. Our investigation aims to assess the effectiveness of rodent 'press' (i.e. constant) poisoning using brodifacoum baits, and also the effect of rodent control on survival of *P. t. traversi* at Lake Paipaitonga Scenic Reserve, southern North Island. We measure the effectiveness of the poisoning operation by comparison of the rodent (rats and mice) populations both prior to and during poisoning in two treatment and two non-treatment areas. We compare the population density of *P. t. traversi* in two non-poisoned and two poisoned areas both prior to, and after poisoning, at 12 and 19 months.

2. Methods

Two areas, with similar habitats and separated from each other by an area of wetland, were set up at Lake Paipaitonga Scenic Reserve. Each area was divided in half, and each half was either poisoned or not poisoned. The four plots were designated P1 (2.99 ha), P2 (2.94 ha), and N1 (2.92 ha), N2 (2.60 ha) for poisoned and non-poisoned areas respectively.

Seventeen Philproof[®] bait stations were placed on a 50 m × 50 m grid in each poison area. Bait stations were affixed low on trees and had sticks leant from the opening to the ground to aid rodent access. Orange-flavoured PestOff[®] possum bait (Animal Control Products Limited, Wanganui), a wax-coated cereal pellet (active ingredient 0.02 g/kg brodifacoum), was used. Six pellets were placed in each bait station to assess bait take on 20 October 1998. All but four bait stations were empty five days later, so they were all re-stocked with 100–250 g of bait (equivalent to about 38–87 pellets) depending on bait take. To maintain press poisoning, bait stations were checked and re-stocked as necessary every 21–42 days until 14 April 2000, and again on 10 July 2000. In addition, rats may have been able to access possum bait stations (stocked intermittently with a brodifacoum bait during the study) spread throughout the study site (including non-treatment areas).

Rodent activity was monitored in treatment and non-treatment areas on two occasions before poison was applied, and on 17 occasions during the poisoning programme, using tracking tunnels laid with tracking paper (after King et al. 1994). Tracking tunnels were placed next to the bait stations in P1 and P2, and on a 50 m × 50 m grid in N1 and N2. On each occasion, activity was monitored for three consecutive nights and tracking papers were replaced each night. Sanitarium Crunchy Peanut Butter was used to lure rodents through the tunnels. The average percentage of tunnels with rat and mouse tracks was used as an index of their respective abundance.

Four 20 m × 5 m sub-plots were established in each experimental plot at Lake Papaitonga ($n = 16$) for monitoring *P. t. traversi*. Terrestrial molluscs frequently have an aggregated or non-random distribution (Peake 1978), so it is better to sample them with many small quadrats rather than using a few large quadrats (Green & Young 1993). There are two forms of *P. t. traversi* present at Lake Papaitonga, namely *P. t. traversi* and *P. t. traversi* f. 'florida', but we did not distinguish between them. Plots were searched in October 1998 before the first poison was applied, and then 12 and 19 months after rodent control commenced (October 1999 and May 2000). The size and weight of each live snail was measured each time it was located. Snails of greater than 23 mm maximum length (ML) were marked with a letter-number combination using an electric engraver (Arlec), whereas smaller snails were marked with a small amount of Selley's Liquid Nails adhesive applied to the protoconch. Predators, and their relative importance to this population of *P. t. traversi*, were estimated by collecting empty shells during quadrat searches and recording the probable cause of death, using the guide of Meads et al. (1984). Mouse damage could not be distinguished from rat damage, and partial shells (i.e. heavily weathered shells that may or may not have been damaged by predators) were grouped together, since the cause of death could not be determined. Shells were discarded outside the experimental area after notes were made. Fisher's exact test and Chi-squared tests (SAS Inc. 1996) were used to analyse the effect of rodent control on the abundance of live *P. t. traversi* and empty shells.

3. Results

3.1 POISON EFFECTIVENESS

Rat activity was significantly reduced in poisoned compared with non-poisoned areas after bait stations were stocked (Fig. 1). No rats were recorded in P2 during November and December 1998 after poisoning began (Fig. 1), and the rat track indices for this area remained mostly below 5% and always below 12% thereafter (Table 1). There was a slower decline in the rat tracking index at P1, until no rat tracks were observed there by March 1999. Thereafter, the rat track index ranged between 0 and 5%, except for one occasion when it reached 14.33% (January 2000; Table 1). The rat track index at N1 was consistently higher than any other area after poisoning (Fig. 1) and this area also had the highest average rat track index (46.04%; Table 1). In contrast, the rat track indices for N2 was similar to P1 and P2 (Table 1).

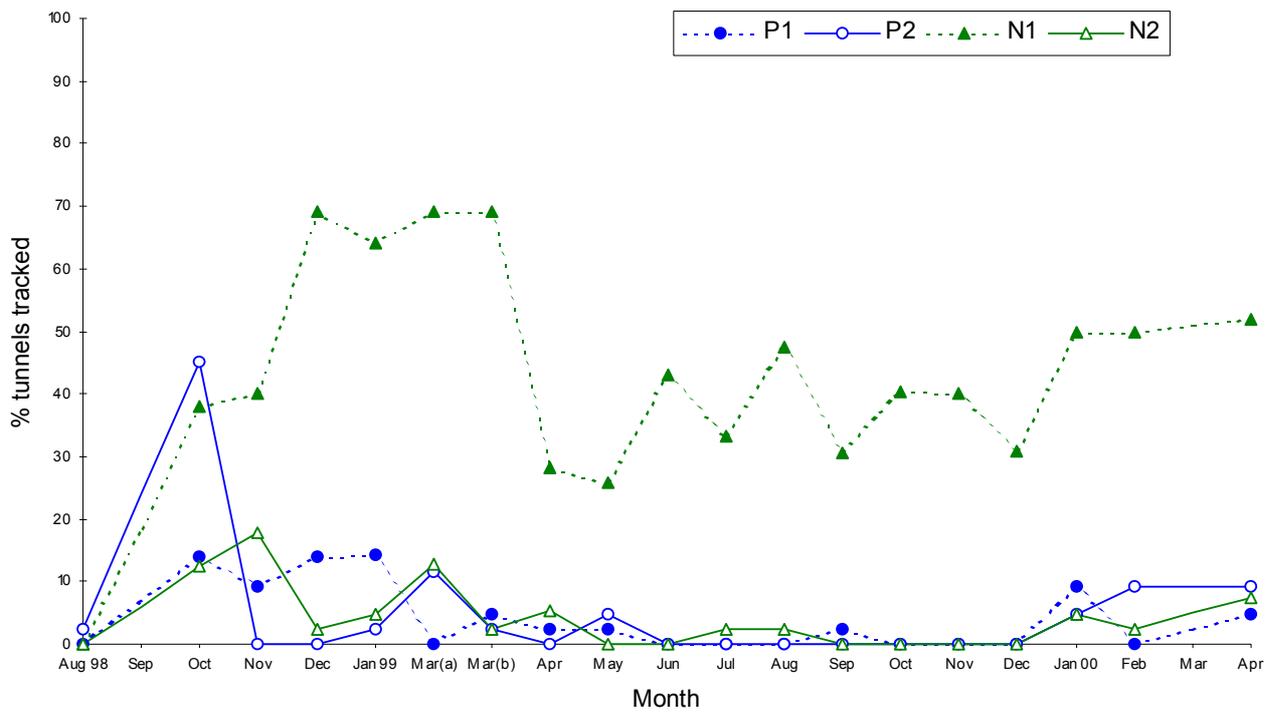


Figure 1. Rat activity in poisoned (P1 and P2) and non-poisoned (N1 and N2) areas at Lake Papaitonga.

TABLE 1. SUMMARY OF RAT TRACKING INDICES AT LAKE PAPAITONGA. TOTAL RANGE INCLUDES TRACKING INDEX VALUES OBSERVED BEFORE POISON WAS APPLIED.

AREA	PRE-POISONING MAXIMUM	TOTAL RANGE	MEAN DURING POISONING
P1	14	0-14	3.72 ± 1.21
P2	45	0-12	2.61 ± 0.96
N1	38	0-69	46.04 ± 3.56
N2	12	0-18	3.77 ± 1.19

Poison effectiveness for mice is best described as ‘patchy’ (Fig. 2). There was an immediate reduction of 93-100% in the mouse track index at P1 and P2 following the first application of poison (Fig. 2). The average mouse track index in both poison areas was much lower than in the non-poison areas (Table 2), but the mouse track index for one poison area was at least as high as a non-poison area in December 1998 and March–April 1999. The mouse track indices for P1 and P2 even exceeded both non-poison areas in April 2000. Overall, the mouse track index was generally highest in N2 (Fig. 2 and Table 2) and contrasted with the rat track indices which were highest in N1 (Fig. 1 and Table 1). Lastly, the mouse track indices during poisoning were generally higher than the corresponding rat track indices, except in N1.

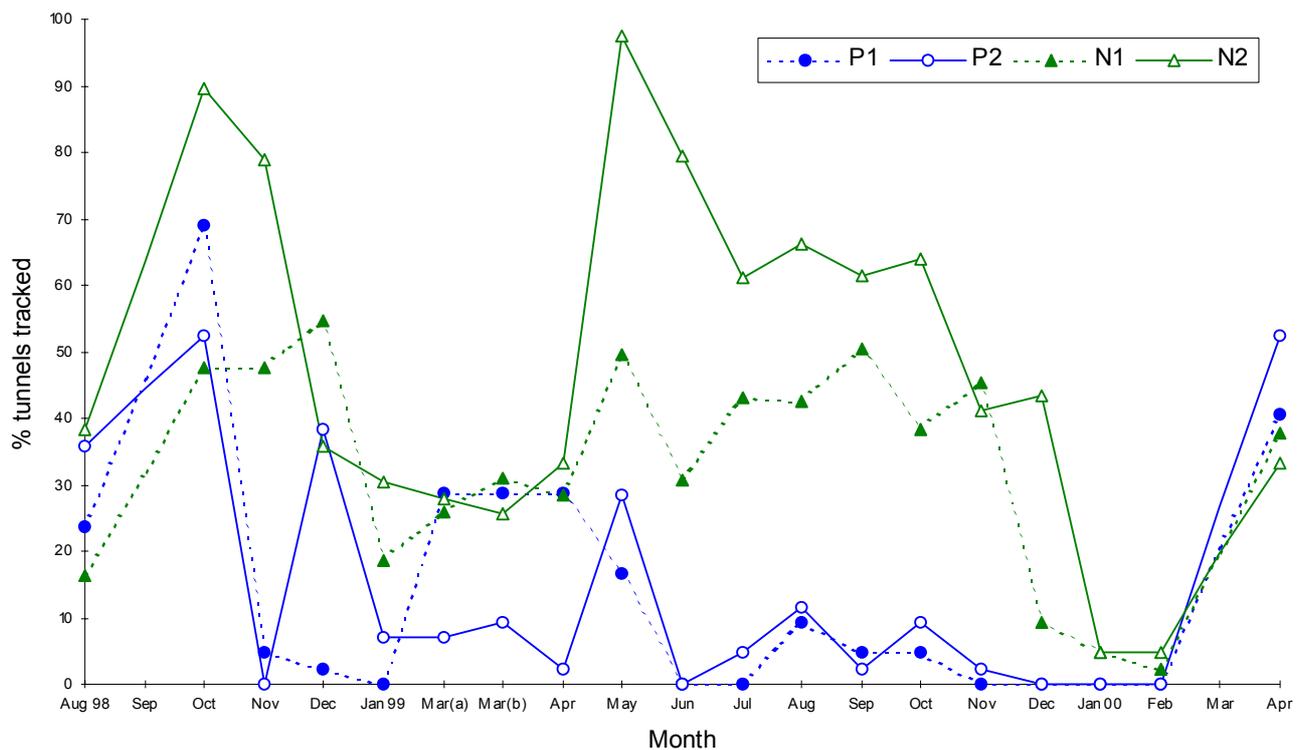


Figure 2. Mouse activity in poisoned (P1 and P2) and non-poisoned (N1 and N2) areas at Lake Papaitonga.

TABLE 2. SUMMARY OF MOUSE TRACKING INDICES AT LAKE PAPAITONGA. TOTAL RANGE INCLUDES TRACKING INDEX VALUES OBSERVED BEFORE POISON WAS APPLIED.

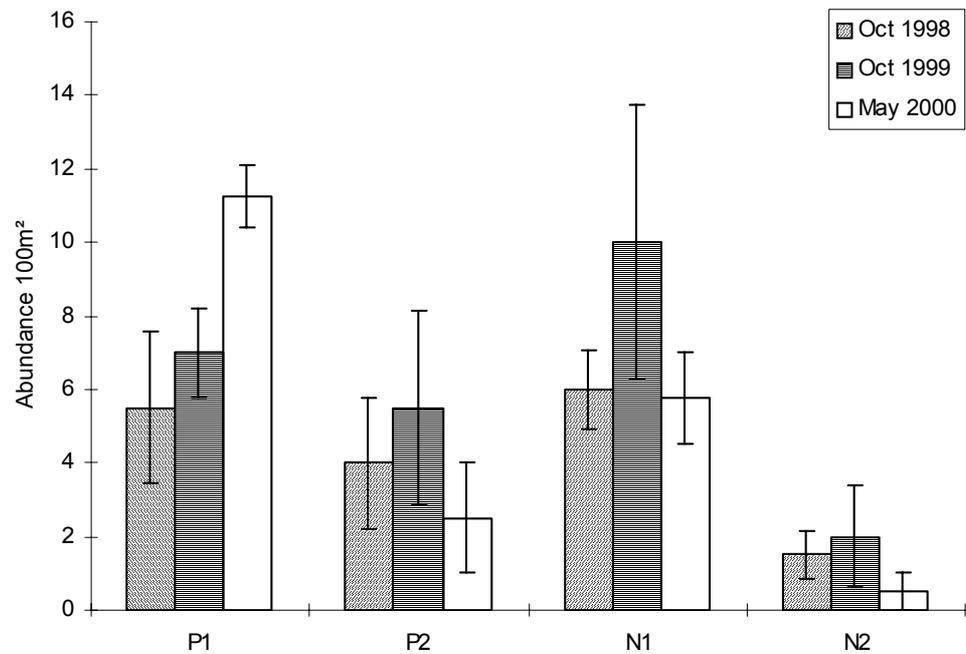
AREA	PRE-POISONING MAXIMUM	TOTAL RANGE	MEAN DURING POISONING
P1	69	0-49	9.94 ± 3.25
P2	52	0-52	10.29 ± 3.67
N1	48	2-55	32.96 ± 3.95
N2	90	5-90	46.36 ± 6.33

3.2 EFFECT OF RODENT POISONING ON *P. T. TRAVERSI*

The density of live snails increased after 12 months of poisoning in P1 and P2, but numbers also increased in non-treatment areas (Fig. 3). Comparison of adjacent poison and non-poison areas (i.e. P1 compared with N1, and P2 compared with N2) indicate that the increase in snail density between 1998 and 2000 in P1 was highly significant ($\chi^2 = 8.99$, d.f. = 2, $P = 0.011$) compared with the fluctuation in snail density over the same period in N1. However, compared with N2 over the period 1998 to 2000, the fluctuation in P2 snail density was not significant ($\chi^2 = 0.55$, d.f. = 2, $P > 0.05$).

The difference in snail abundance between areas, and between pre-treatment and post-treatment searches was highly significant ($\chi^2 = 17.47$, d.f. = 6, $P < 0.01$). The increase in snail abundance from 1998 levels, at P1 in 1999 and 2000, and at N1 in 1999, contributed most to this result. The snail abundance at

Figure 3. Mean (\pm SE) abundance of live *P. t. traversi* per quadrat in rodent-poisoned (P1 and P2) and non-poisoned (N1 and N2) areas prior to poisoning (Oct 1998) and post-poisoning at Lake Papaitonga.



P1 in 2000 (45 snails) was increased significantly ($\chi^2 = 3.89$, d.f. = 1, $P < 0.01$) from 1998 (22 snails) and 1999 (28 snails; $\chi^2 = 7.57$, d.f. = 1, $P < 0.05$). Also, the snail abundance at N1 in 1999 (40) was significantly greater than that a year previously (24 snails; $\chi^2 = 3.91$, d.f. = 1, $P < 0.05$). Lastly, the decline in the numbers of live snails between 1999 and 2000 in N1 (40 and 23 snails respectively) was significant ($\chi^2 = 4.47$, d.f. = 1, $P < 0.05$), as was the decline in snails in P2 between 1999 and 2000 (22 and 10 respectively; $\chi^2 = 3.89$, d.f. = 1, $P < 0.05$).

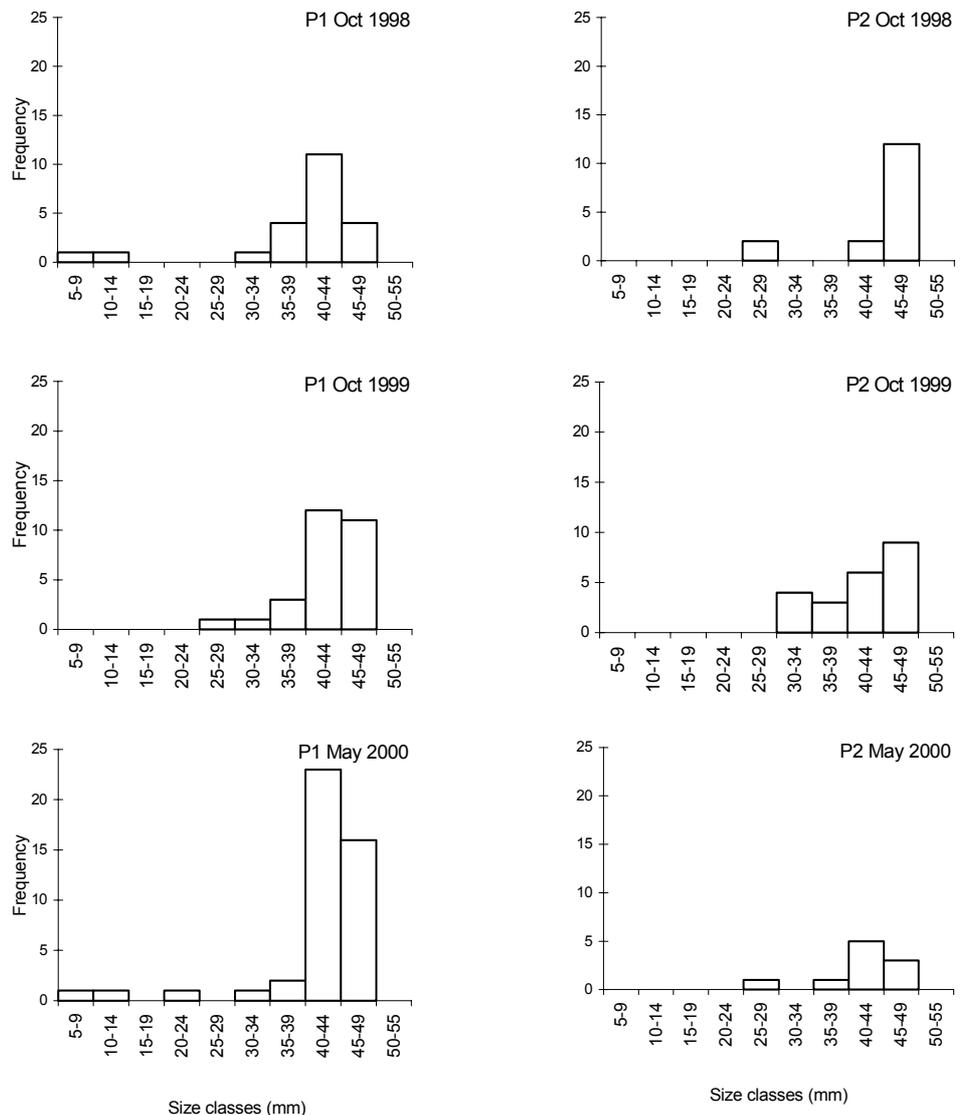
A comparison of the size-frequency distributions of snails within areas for the period of the poisoning operation suggests that the increase in density of snails was due to migration (e.g. P1 between 1999 and 2000, Fig. 4A; and N1 between 1998 and 1999, Fig. 4B) and recruitment (e.g. P1 between 1999 and 2000; Fig. 4A).

The mean (\pm SE) number of live snails recorded per 100 m² quadrat for all 48 searches was 5.13 (\pm 0.65). This gives an estimate for the *P. t. traversi* population at Lake Papaitonga (35.9 ha suitable habitat) of between 14 000 and 23 000 snails (95% CI).

3.3 PREDATION OF *P. T. TRAVERSI*

Mortality varied among experimental plots prior to poisoning (1998 data), being highest in P1 and lowest in N2 (Fig. 5). The ratio of live to dead snails was roughly equal for plots P2, N1 and N2, but much higher (3.6) for P1 based on the 1998 census. The cause of death was not usually predator-related in all areas, pre-poison and post-poison (Fig. 5). Prior to poisoning, rats were responsible for 29% of deaths in P1, 14% in N1, and none in others (Fig. 5). After poisoning in P1, rat predation, measured as a percentage of total deaths, dropped from 29% pre-poison to 15% one year after poisoning, and to 10% 19 months after poisoning. Concomitantly, in the same area, bird predation increased from 3% in October 1998 to 20% in October

Figure 4. *Powelliphanta t. traversi* size-frequency distributions (ML) in rodent-poisoned (P1 and P2) (this page) and non-poisoned (N1 and N2) (facing page) areas prior to poisoning (Oct 1998) and post-poisoning at Lake Papaitonga.

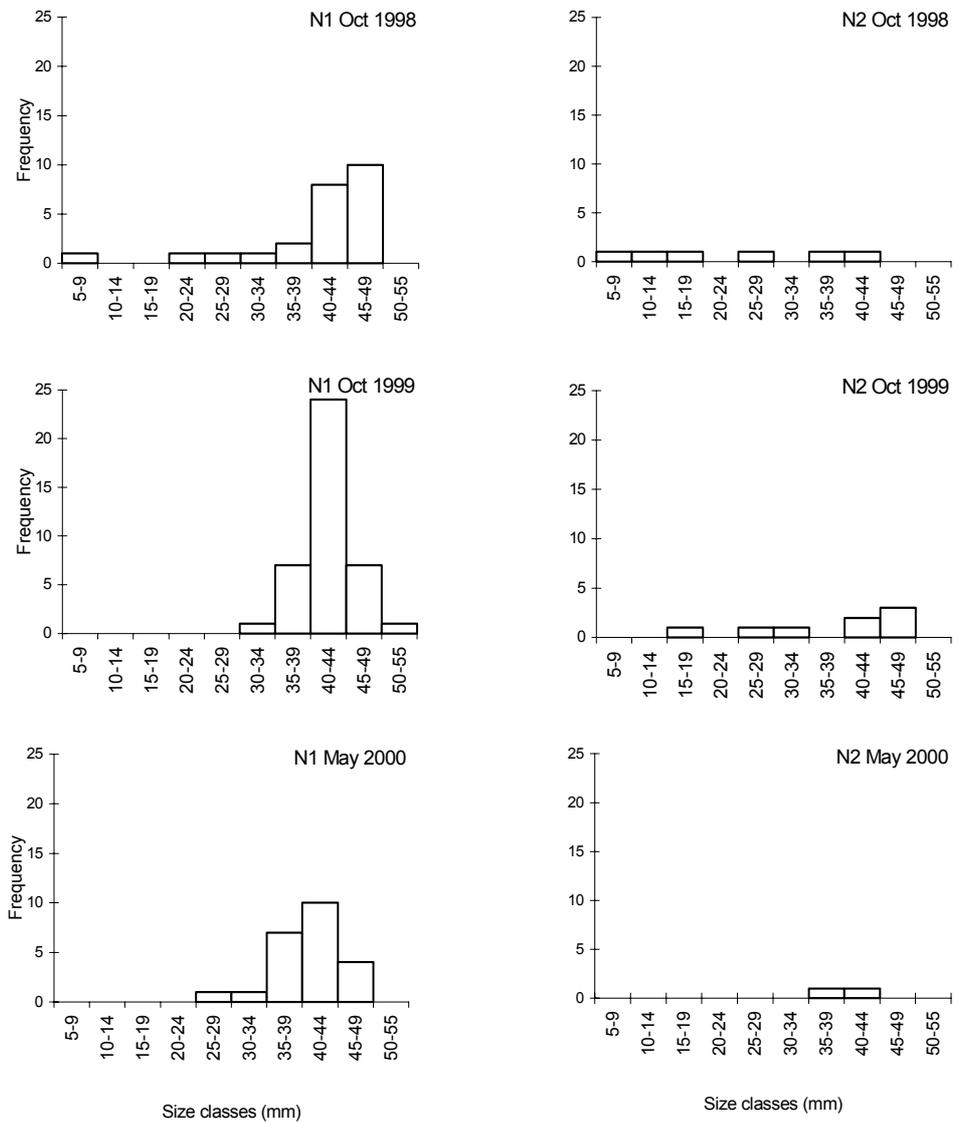


1999 and to 48% in May 2000. Indeed, bird predation increased over 1998–2000 in all areas except N2 (Fig. 5).

The majority of whole and rat-damaged shells were in the three largest size classes (40–55 mm; Fig. 6). By comparison, partial shells were distributed across all size classes and bird-damaged shells found were less than 25 mm (with the majority < 20 mm). There was a highly significant difference ($\chi^2 = 119.12$, d.f. = 27, $P < 0.01$) between the four types of empty shell when the size-frequency distributions were compared. The high number of empty whole shells between 40 and 50 mm, and bird damaged shells between 10 and 20 mm contributed most to the result.

Empty shells were found with damage consistent with both Norway rat and ship rat predation during this study. Several empty shells (and two live snails) were found that had suffered rat damage to the aperture lip (peristome) but had continued to grow, so it is apparent that snails are able to survive some degree of rodent attack. Both types of rat damage, and snails that had survived rodent attack, were also observed by Devine (1997).

Figure 4 continued



4. Discussion

We estimate that rat predation accounted for 20% of deaths of *P. t. traversi* at Lake Papaitonga prior to poisoning. This may be an underestimate, since we did not search for burrows containing rat caches. Meads et al. (1984) did search for rat burrows and found caches, which may explain their higher estimate of rat predation (69%). Moreover, some partially damaged shells (27%) may have been rat-chewed. Press poisoning using brodifacoum baits effectively reduces rat abundance relative to non-poisoned areas, and the effect on *P. t. traversi* is evident by the increase in population size, mainly due to adult migration, and a decrease in rat-damaged shells, for areas where rat predation occurs (P1). It is harder to determine mouse impacts on *P. t. traversi*, since shell damage by mice is not easily identifiable. Additionally, mice may completely destroy shells if they are small. The increase in snail abundance at area P1 post-poisoning occurred despite inconsistent control of mice in the area, indicating that mice

Figure 5. Empty *P. t. traversi* shells categorised as whole, rat-damaged, bird-damaged or partially damaged, found in: Oct 1998 prior to poisoning (a, b); May 2000 12 months after poisoning (c, d); and Oct 2000 19 months after poisoning (e, f), at Lake Papaitonga.

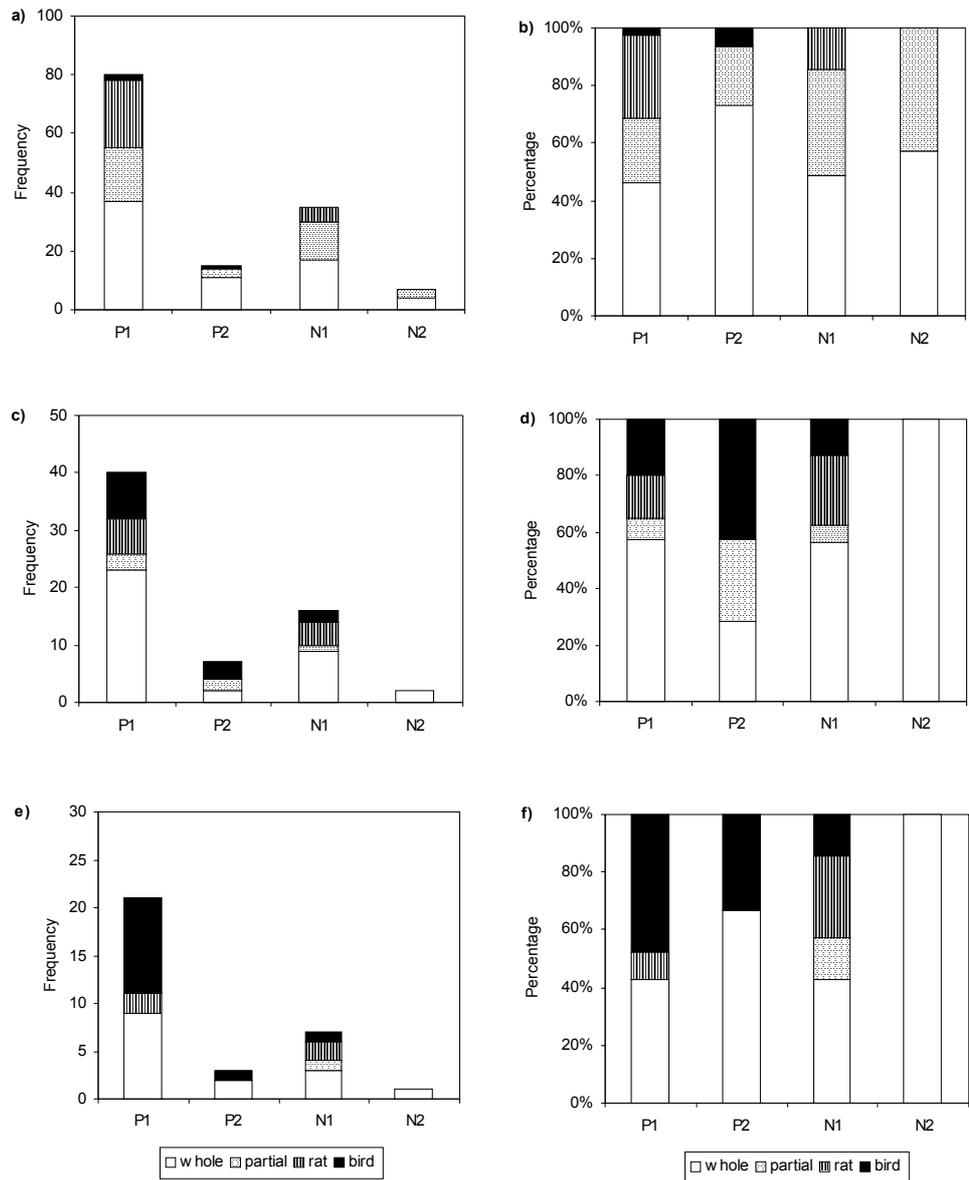
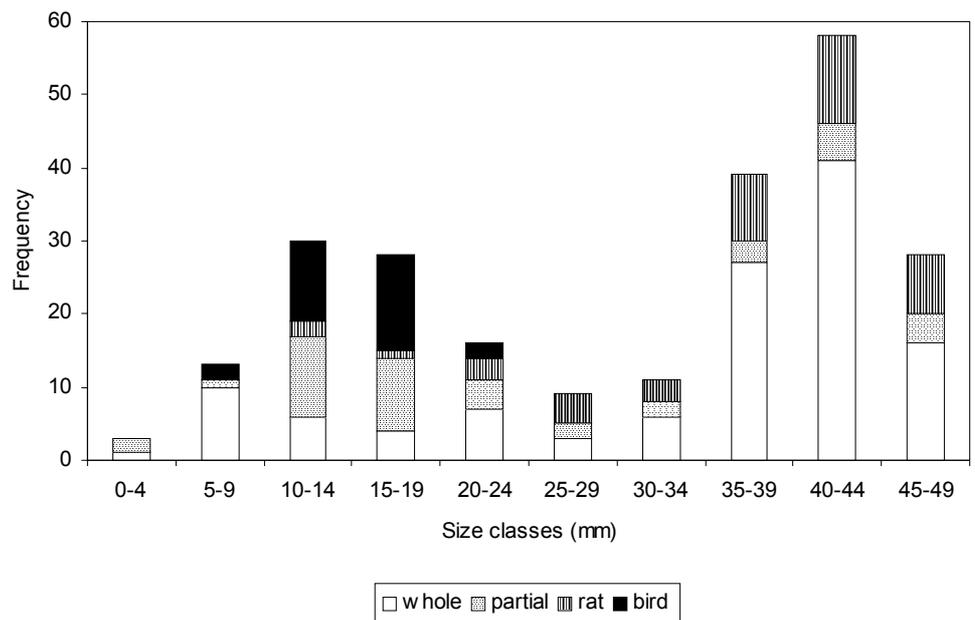


Figure 6. *Powellipbanta t. traversi* size-frequency distributions for whole, rat-damaged, bird-damaged and partially damaged shells found October 1998 to May 2000 at Lake Papaitonga experimental area.



predation of adult *P. t. traversi* is minimal. We estimate the *P. t. traversi* population size at Lake Papaitonga to be within the range of 14,000–23,000 snails, based on a mean \pm SE density of $4.92 \pm 0.71/100 \text{ m}^2$, which is slightly greater than an estimate made three years previously of 14,000–15,000 snails, based on a mean \pm SE density of $2.82 \pm 0.85/100 \text{ m}^2$ (Devine 1997). This suggests that that population is, at the very least, replacing itself.

Innes & Barker (1999) pointed out that a ‘pulse’ regime of vertebrate pest control is likely to be ineffective for invertebrates if the adults are vulnerable to predation during inter-pulse periods. Growth of *P. t. traversi* is indeterminate, and the snails do not develop any morphological attributes as adults that afford them protection from rodent predation (cf. the thickened aperture of adult *Placostylus*: Sherley et al. 1998). Rat populations can recover within 2–5 months of a single poison drop (Innes et al. 1995). The press baiting over 19 months at Lake Papaitonga successfully maintained low rat densities. Rat consumption of possum bait may account for the low rat densities in area N2. Once rodent numbers are reduced by poisoning, the remaining individuals become increasingly difficult to remove because of an increase in the relative abundance of food (Thomson 1992). Meads et al. (1984) believed that Norway rats were the primary predator of *P. t. traversi* in the Horowhenua, but recognised that both Norway rats and ship rats may exhibit similar predator sign. We think ship rats are the primary rodent predator of *P. t. traversi* at Lake Papaitonga, since they constituted the entire catch of a recent trapping effort (Devine 1997).

Mouse abundance can increase despite poison being available (Innes et al. 1995; Gillies & Pierce 1999), and mice can also increase following an initial decline after poisoning (Innes et al. 1995; Miller & Miller 1995; Hunt et al. 1998; present study). Innes et al. (1995) showed that increases in mouse abundance were highest in areas where rat abundance had been most reduced. This was also the case at Lake Papaitonga. Furthermore, mice are able to multiply more quickly than rats, and will recover quickly in the absence of bait, as we observed when mouse abundance increased from February to April 2000 while rat abundance remained low. These different responses of mice and rats, and also spatial variation in densities, highlight the importance of monitoring targeted pest species during control operations. Hunt et al. (1998) suggested that studies investigating the benefits to invertebrates of sustained rodent poisoning require about six years of data collection, including two years pre-poison monitoring to determine natural fluctuations in both snail and rodent densities. Furthermore, Dowding & Murphy (1994) showed that ship rat home ranges vary spatially and temporally, and urged caution when interpreting data from tracking tunnels. Ideally, tracking tunnel indices should be calibrated with live trapping (e.g. Brown et al. 1996).

Few studies have monitored the impact of small mammal predator control on gastropods. Churchfield et al. (1991) reported a significant increase in large gastropods when shrews were excluded from grassland plots in England over a two-year period. Sherley et al. (1998) recorded a significant increase in recruitment of *Placostylus ambagiosus parasprittus* (Powell) during rodent control. A study similar to ours in terms of habitat, location and use of brodifacoum baits has involved 18 years of rodent poisoning for protection of *Powelliphanta t. otakia* snails. The number of live snails increased from just

above zero to 10 after three years of poisoning, and to 42 after eight years of poisoning. Since then, however, the number of live snails has fluctuated, perhaps because baiting has been irregular (every 2–3 months) and restricted to a small area (300 m²), which may allow rat re-invasion (K. Walker, pers. comm.). We attributed the increase in snail density in P1 to migration because recruitment was low (e.g. $n = 2$, P1, May 2000) and even though we failed to observe recruitment, snail growth is about 1.5 mm increase in diameter per year (Meads et al. 1984), so it would take 5 mm recruits about 20 years to grow to 35 mm.

Whereas rats mostly target snails larger than 25 mm, birds (and possibly mice) target shells smaller than 25 mm, so no size class was free from predation pressure. Blackbirds (*Turdus merula* L.) and song thrushes (*Turdus philomelos* Brehm) occur throughout the reserve, and both are the most likely bird predators of *P. t. traversi* (Meads et al. 1984). It is not possible to distinguish between the damage caused by these species, even though each typically uses a different method of snail extraction. Song thrushes tend to use anvils to smash open snail shells (Heather & Robertson 1996), whereas blackbirds hammer and prise open the shells (Meads et al. 1984). Predation by rats on New Zealand's avifauna is well documented (Atkinson 1978; Brown et al. 1998); in particular, ship rats and mice are known to prey on blackbirds and song thrushes (Kikkawa 1966; Moors 1983). The release of these birds from rat predation could explain why we observed an increase in bird predation of *P. t. traversi* in areas where rat numbers were controlled, but does not explain why we observed a slight increase in bird predation in N1 where rat abundance remained high. Previous workers report no bird predation of *P. t. traversi* at Lake Papaitonga (Meads et al. 1984; Devine 1997), while we report three cases of bird predation prior to, and 25 cases during the rodent poison operation. Lastly, hedgehog damage is apparently undistinguishable from bird damage of *Powelliphanta* shells (K. Walker, pers. comm.), and so hedgehogs may be responsible for some of the 'bird predation' recorded during this study. Hedgehogs did not attack baited shells in our lab trials.

Factors unrelated to predation account for 51% of *P. t. traversi* mortality. This level is consistent for the period October 1998 to May 2000 (cf. 18% rat and 12% bird over the same period), and probably accounts for some of the partially damaged shells collected in 1998, since whole shells may have deteriorated between the death of the snail and collection. Non-predator-related death occurs across all size classes, but is particularly common for snails larger than 35 mm. It must be asked whether this is natural. It has increased since 1984, when Meads et al. (1984) collected 49 (31%) whole shells, and noted that the understorey and litter layers were reduced, although we note that they did not search measured quadrats and hence may have underscored small snails. Perhaps death by desiccation is becoming more common for *P. t. traversi* at Lake Papaitonga, although we would expect the smaller snails to be most affected (large surface area to volume ratio relative to larger snails), and this is not so. At Prouse's Bush, where predation pressure is low (Standish et al. 2002), non-predator-related death was also common, more so in leaf litter (54%) than in *Tradescantia* (29%), over the period August 1998 to July 2000. This supports

our suggestion that desiccation is a factor, since the figure is lower in *Tradescantia*, which is the moister habitat.

The apparent paucity of *P. t. traversi* recruits at Lake Papaitonga is cause for concern. Recruitment is likely to be low, as *P. t. traversi* exhibit typical *K*-selected traits (e.g. rare, few young, large young, slow maturation) but there are no data to indicate what this recruitment value is. Small dead shells are also uncommon at Lake Papaitonga (i.e. 16 of 230 were smaller than 10 mm), so unless small shells are eaten whole by predators, recruitment is infrequent, as suggested by observations of live snails. However, rapid decay of shells by humic acid may prevent accurate scoring of mortality, particularly for the smaller size classes. Growth from hatching to juvenile is likely to be faster than at any other stage of the life cycle (I. Stringer, pers. comm.), so recruits may be observed less often than older snails, but should nevertheless be observed (e.g. as in *Tradescantia* at Prouse's Bush: Standish et al. 2002). The size-frequency distribution of the population at Lake Papaitonga is skewed heavily towards larger size classes. This was also true of *P. t. traversi* in leaf litter at Prouse's Bush (Standish et al. 2002). The size-frequency distribution of *P. t. traversi* in *Tradescantia* at Prouse's Bush closely resembled a bell-shaped curve, including recruits (Standish et al. 2002). Clearly, the population with recruits is more viable than one with few or no recruits.

Our study indicates that *P. t. traversi* will benefit from rat control where rat predation is evident, whereas predation by mice is difficult to estimate and needs further research. The level of rat control necessary to benefit *P. t. traversi* can be achieved with brodifacoum baits. Mouse control with brodifacoum baits was inadequate. A long-term study is required to determine any benefit of long-term rat control to *P. t. traversi* recruitment. The concomitant rise in bird predation (and possible predation by mice and hedgehogs) with rat control suggests that control of all three is necessary to make real conservation gains. The high percentage of non-predator-related deaths, evident at Lake Papaitonga and in native habitat at Prouse's Bush, is also of concern. This is possibly due to conditions being too dry, and there is no 'easy fix' solution for making habitat conditions more suitable where the results of past activities (e.g. habitat fragmentation, drainage) are irreversible. It is ironic that an invasive weed (*Tradescantia*) appears to be a more suitable habitat for *P. t. traversi* by providing some protection from desiccation, although this hypothesis, and also the apparent low *P. t. traversi* recruitment in native habitat, requires further research.

5. Conclusions and recommendations

Rat predation continues to threaten *P. traversi*, and it is likely that adult *P. traversi* would benefit from rat control. Longer-term continuous rat control is necessary to determine the effect on *P. traversi* recruitment. Mouse predation was dismissed as unimportant by Meads et al. (1984), but we remain uncertain of their importance as predators of juvenile *P. traversi*. We highlight the importance of monitoring major predator-prey dynamics during predator control operations, especially: key predators, target species and the response of other predators. For complete protection of *P. traversi* from predation, a control programme would need to target rats, as well as blackbirds, song thrushes and possibly mice and hedgehogs. Of the threats to survival of *P. traversi*, we found that predation was secondary to non-predator-related mortality, in contrast to past observations (e.g. Meads et al. 1984).

6. Acknowledgements

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EFFECTS OF RODENT POISONING ON *POWELLIPHANTA TRAVERSI*

By Shaun J. Bennett, Rachel J. Standish and Ian A.N. Stringer

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