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Resistance in introduced populations of a freshwater snail to native range parasites

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evolution of increased competitive ability hypothesis;
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resistance.

Abstract

Introduced species provide an opportunity to examine responses to novel ecological conditions, in particular to the absence of co-evolved enemies. Introduced populations could evolve lower investment in resistance or could down-regulate their immune system as a plastic response to enemy absence. The response might have consequences for the success of introduced species. Assuming a trade-off between resistance and traits related to demographic success, an evolved change or reallocation from resistance could increase the chances of invasions. On the other hand, introduced populations could have increased resistance as a correlate of greater vigour and competitive ability among successful invaders [Sampling Bias hypothesis (SBH)]. These hypotheses make different predictions about investment in resistance in introduced populations. Using a New Zealand clonal snail (*Potamopyrgus antipodarum*), we examined the resistance of three introduced genotypes (one from the US and two from Europe) to several populations of a native range parasite (*Microphallus* sp.). One genotype (Euro A) was resistant to all native range parasite populations, consistent with the SBH. However, two remaining genotypes (Euro C and US 1) were less susceptible to parasite populations that were allopatric to their source populations. Furthermore, resistance of one genotype (US 1) collected from the introduced range was indistinguishable from its resistance when collected from the range of the parasite. Hence, there was no evidence for decreased resistance in the absence of native enemies, which is inconsistent with hypotheses that envision reduced allocation to resistance or a trade-off between competitive ability and resistance.

Introduction

Introduced species experience dramatic ecological changes that should lead to a new selection regime (Lee, 2002; Stockwell *et al.*, 2003). One prominent difference between the introduced range and native range of many invaders is the absence of co-evolved enemies; invaders often leave behind more specialized enemies than they acquire in the introduced range (Wolfe, 2002; Mitchell & Power, 2003; Torchin *et al.*, 2003). The absence of specialized enemies should favour reduced allocation to resistance, assuming such traits are costly (Moret & Schmid-Hempel, 2000; Hoang, 2001). Either an evolved response or a plastic down-regulation in resistance (Moret & Siva-Jothy, 2003; Little & Kraaijeveld, 2004) is possible in introduced populations. On the other hand, introduced populations might maintain their allocation to resistance if resistance is not costly, or if novel enemies stimulate up-regulation of the immune system (Colautti *et al.*, 2004). In contrast to these predictions, it is also possible for introduced populations to have greater resistance because of selection during invasion for more vigorous genotypes [the Sampling Bias Hypothesis (SBH); Simons, 2003], assuming vigour is associated with more resources to invest in resistance (reviewed in Sheldon & Verhulst, 1996; Jokela *et al.*, 2000; Rigby *et al.*, 2002). Introduced species can be used to address these responses to the absence of co-evolved enemies.

The level of allocation to resistance in the introduced range also might influence the probability of success of an

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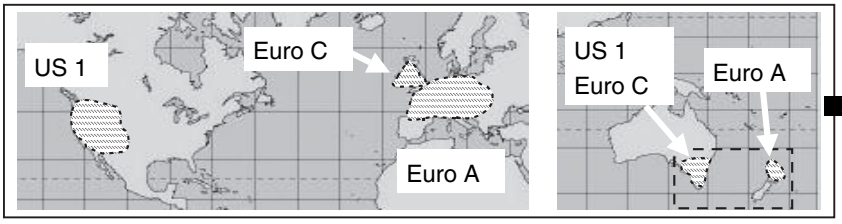

Fig. 1 Map representing the introduced range of snail genotypes (US 1, Euro A and C) and their source range where they are exposed to the parasite *Microphallus* (dashed box).

introduced species. Assuming trade-offs exist between resistance and competitive ability (Kraaijeveld & Godfray, 1997), an evolutionary response to relaxed selection for resistance might lead to increased demographic success in the introduced range. This is known as the Evolution of Increased Competitive Ability hypothesis (EICA; Blossey & Notzold, 1995; Siemann & Rogers, 2003). Similarly, if introduced species down-regulate their allocation to costly resistance through phenotypic plasticity, they could reallocate resources to growth, reproduction, or competitive ability (Crawley, 1987). We refer to this as the Plastic Increase in Competitive Ability Hypothesis (PICA). In both cases, a reduced allocation to resistance positively influences the demographic success of introduced populations. On the other hand, the success of introduced species might not depend on a change in resistance. The absence of enemies should lead to lower mortality and increased recruitment, assuming specialized enemies play regulatory roles in communities (Poulin, 1999; Mitchell & Power, 2003). Hence, the demographic success of an introduced population would result from a release from population regulation (Regulatory Release hypothesis; Maron & Vila, 2001; reviewed in Keane & Crawley, 2002; Colautti *et al.*, 2004).

In this paper, we examine resistance of an introduced species to its co-evolved enemies to answer questions about responses to relaxed enemy-driven selection and invasion success. We examined the resistance of three introduced genotypes of a clonal New Zealand endemic snail (*Potamopyrgus antipodarum*) to the parasite (*Microphallus* sp.) from the snail's native range. This snail is introduced in Australia, Europe, Great Britain and the USA (Ponder, 1988; Zaranko *et al.*, 1997). For three introduced snail genotypes (US 1 from the western USA, Euro C from Great Britain and Euro A from continental Europe; Fig. 1), we examine resistance to *Microphallus* populations from the source ranges of the snail genotypes. First, we looked at levels of resistance to *Microphallus* from populations that were sympatric and allopatric to snail source populations for these genotypes. *Microphallus* is known to be locally adapted to sympatric host populations and common genotypes (Lively, 1989; Dybdahl & Lively, 1998; Lively & Dybdahl, 2000; Lively *et al.*, 2004). If resistance had declined, we expected introduced snail genotypes to be susceptible to all source parasite populations. Second, we compared resistance of US 1 collected from the introduced range with US 1 collected from the source range and within the range of *Microphallus*.

Materials and methods

Study system

Potamopyrgus antipodarum is a freshwater snail with both clonal and sexual variants in New Zealand (Wallace, 1992; Dybdahl & Lively, 1995). Clonal populations have been introduced throughout the world, including Australia since 1895, Great Britain and Europe since 1859 and the USA since 1985 (Ponder, 1988; Zaranko, 1997). Three introduced *P. antipodarum* clones identified by allozyme genetic markers (US 1, Euro A and C) are the focus of this study (Fig. 1). US 1 is found across the western USA (M.F. Dybdahl, unpublished), Euro C is found in the United Kingdom and Euro A is found in a broad region of mainland Europe (Hauser *et al.*, 1992). Previous infection experiments showed that neither US 1 (M.F. Dybdahl, unpublished) nor Euro A (J. Jokela, personal communications) were infectible by South Island, New Zealand, populations of *Microphallus.* These results might reflect a lack of co-evolutionary history between South Island parasites and these clones. In fact, more recent data suggests that the source range for US 1 and Euro C invasions is Australia, and that the source of Euro A is New Zealand's North Island (Fig. 1), based on the distribution of clonal genotypes and mtDNA haplotypes (M.F. Dybdahl, unpublished; Stadler *et al.*, 2005) (these conclusions for US 1 and Euro C are supported by infection rates shown in Figs 2 and 4).

Microphallus sp. is a digenetic trematode found in New Zealand and Australia. *Microphallus* populations in Victoria, Australia, are sympatric with the source ranges of US 1 and Euro C, and *Microphallus* populations North Island are sympatric with the source range of Euro A (Fig. 1). However, there are no published reports or known records occurring in European or USA populations of *Potamopyrgus*. Snails become infected by ingesting an egg of *Microphallus* produced during the adult stage in the definitive host (a variety of waterfowl and wading birds). Within the snail, eggs hatch and the parasite migrates to the gonad, reproduces asexually, encysts as metacercariae and eventually replaces the gonad and sterilizes the individual. Upon ingestion by the definitive host, adult worms excyst from the metacercariae.

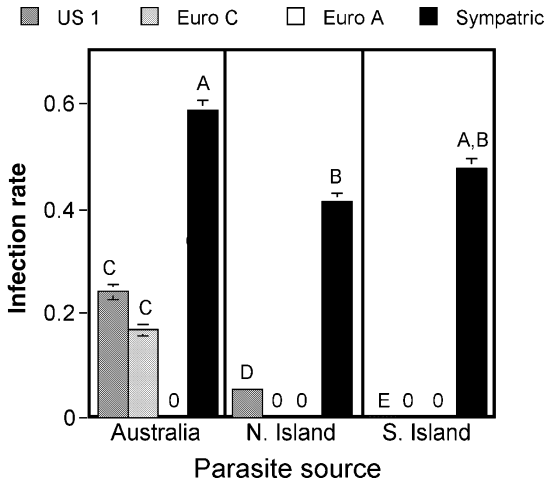

Fig. 2 Resistance of introduced clones to *Microphallus* in a laboratory infection experiment. Parasites were most infective to their sympatric hosts. All clones retained resistance to allopatric parasites, and US 1 and Euro C were most susceptible to *Microphallus* from their source range (Australia). Bars sharing the same letter are not significantly different from each other, and '0' indicates 0% infection. Vertical bars show binomial mean-square errors.

Are patterns of resistance to sympatric and allopatric parasites maintained in introduced populations? US 1, Euro C and A

To examine resistance in introduced populations, we did a laboratory infection experiment using three *Microphallus* populations that were sympatric and allopatric to each snail clone's source population. If introduced snail populations reduced their allocation to resistance, then we expected that they would be susceptible to any parasite population, regardless of geographic origin. Hence, we exposed US 1, Euro C and A to *Microphallus* from three sources: Victoria, Australia (Wilkur Creek), which is sympatric with US 1 and Euro C, the North Island (Lake Taupo), which is sympatric with Euro A and South Island (Lake Poerua), which is allopatric to the sources of all three introduced clones.

Parasites used for the infection experiment were collected from each source population in January 2003 by making large collections of snails that were then dissected to obtain *Microphallus* in the infective metacercarial stage. Laboratory mice were used for the vertebrate host (see also Lively, 1989). Two mice were assigned to each parasite source population. *Microphallus* metacercaria from approximately 20 infected snails from each source population were fed to each mouse. In the mouse, metacercaria hatch into adult worms that mate and produce eggs. Eggs are deposited in the mouse faeces. For each parasite source, faeces from the two mice were combined to avoid confounding an individual mouse with parasite source.

The snails used in the experiment were collected in their introduced range and held in the laboratory. US 1 was collected from Thousand Springs, Idaho, USA, and Euro A and C were both obtained from laboratory stock cultures collected in the field in Europe and Great Britain, respectively (Dr V. Forbes, University of Roskilde). As a positive control for infection, we also exposed field-collected snails that were collected from each parasite source. There were three replicates of the sympatric positive controls, three of US 1, two of Euro C and one of Euro A. There were 60 snails per replicate held in two-liter plastic containers. Each replicate was exposed by adding an equivalent volume of the appropriate mouse faeces mixture. After a 2-week exposure, snails were transferred to clean containers and housed with regular food and water changes. After 3.5 months, snails were dissected to determine infection status and the per cent infected per replicate.

To determine whether introduced populations retained resistance to different populations of *Microphallus*, we tested for the effects of parasite source (Australia, North Island and South Island) and host (US 1, Euro C, A and Sympatric) and their interaction using a two-way analysis of variance and planned comparisons (Ott & Longnecker, 2001). In order to meet model assumptions, per cent infection was arcsine square root transformed. We predicted that if resistance was lower in the introduced range (as predicted by the PICA and EICA), then the three clones would be susceptible to all parasite sources.

Does resistance of US 1 from introduced and source ranges vary?

To determine whether the allocation to resistance changed in the absence of parasites in the USA population of US 1, we used the results from the lab infection experiment to compare the infection rate of US 1 collected from the USA with the infection rate of US 1 collected from Australia using the Australian sympatric positive controls. For the latter, we snap-froze the snails at the end of the experiment, assayed the six-locus allozyme genotypes (Dybdahl & Lively, 1995), and calculated the frequency of the US 1 clone in this sample and its infection rate. We compared infection rates of US 1 from the two populations using 95% confidence intervals (Ott & Longnecker, 2001).

This comparison assumes that Australian *Microphallus* populations are co-evolved and locally adapted to track clonal snail genotypes in Australia (including US 1), as has been shown in New Zealand (Dybdahl & Lively, 1998; Lively & Dybdahl, 2000). In addition, it assumes that lab experimental infection rates mimic infection rates by natural *Microphallus* populations in the field. To determine whether *Microphallus* in Australia is infecting local clones in the field, we measured the frequency and infection rate of field-collected US 1 from an Australian source population (Wilkur Creek in Victoria, Australia)

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in January 2003. We dissected 77 snails, recorded infection status, and genotyped each snail using allozymes. We calculated the proportion of the total sample comprised of each clone, and the infection rate of each clone as the number infected over the total for that clone. Rare clones were defined as those making up <15% of the sample and were grouped for the statistical analysis.

To ensure that the laboratory infection experiment accurately mimicked the infectivity of the natural *Microphallus* population, we compared these field measures of relative clone infection rates (AU field in Fig. 3) with the relative experimental infection rates of these same clones (including US 1) in the Australian sympatric positive control treatment from the lab infection experiment (AU experimental). These proportions were compared using 95% confidence intervals (Ott & Longnecker, 2001).

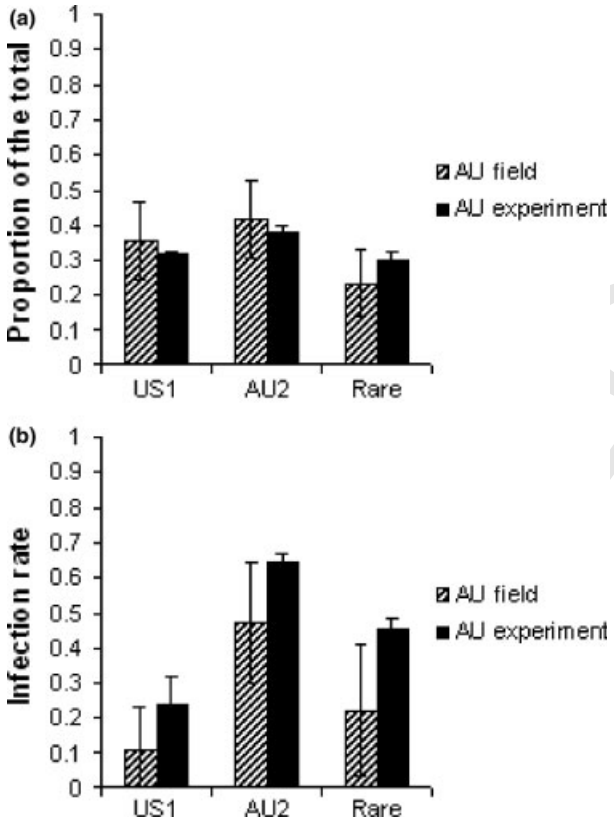

Fig. 3 Clone frequency and infection rates by the parasite *Microphallus* from the Australian source range of US 1 (Wilkur Creek, Victoria, Australia). (a) The clonal composition of natural populations of snails in the field (AU field) and in the Australian sympatric positive control treatment in the lab experiment (AU experimental). (b) Clonal infection rates in Australian field and experimental samples, as in (a). These data show that US 1 is susceptible to *Microphallus* in the field, and that the lab experiment mimicked the natural population, both in terms of clonal composition and infection rates. Vertical bars show 95% confidence intervals.

To determine whether Australian *Microphallus* are adapted to local snails including US 1, we performed an infection experiment in 2004 with Australian and North Island populations of *Microphallus*. Parasites were isolated from *P. antipodarum* collected from Wilkur Creek, Victoria, Australia and from Lake Taupo, North Island, New Zealand. These two parasite sources were used to expose snails from both populations and snails from US 1 collected at Thousand Springs, Idaho, USA. For each parasite source (Australia and North Island), we passed *Microphallus* sp. from 20 snails through each of three replicate mice. The resulting parasite eggs collected from the mouse faeces were distributed across three replicate containers with 75 snails from each of the three host sources (Australia, North Island and USA). The results were analysed using a mixed model ANOVA, executed by the General Linear Model procedure in SPSS (Norusis, 2002). Host and parasite sources were fixed effects, and mouse was a random effect nested within parasite. Infection rate was arcsine square root transformed.

Results

Are patterns of resistance to sympatric and allopatric parasites maintained in introduced populations? US 1, Euro C and A

The laboratory infection experiment showed that each of the parasite populations were infective to their sympatric host population, with infection rates ranging from 45% (North Island) to 58% (Australia) (Fig. 2). However, introduced clones were not equally resistant to all parasite sources (Fig. 2), as would be expected if reduced allocation to resistance had erased patterns of local parasite adaptation. The parasite-host clone interaction was highly significant, indicating that the infection rate of specific clones depended on the source population of the parasite (Table 1). The US 1 and Euro C clones with source range in Australia were least resistant to Australian *Microphallus*. Nevertheless, US 1 and Euro C retained resistance to allopatric parasites. Infection rates of US 1 and Euro C by *Microphallus* from the North Island and South Island were significantly lower. In contrast, the Euro A clone was resistant to all three parasite sources, including the North Island parasites which are sympatric to New Zealand populations of the Euro A genotype.

Table 1 Results from a two-way ANOVA of infection rates from the laboratory infection experiment, including the effects of parasite source (Australia, North Island, South Island) and host source (US 1, Euro A, C, and Sympatric).

Source	d.f.	Type III SS	MSE	*F*-value	*P*-value
Parasite	2	0.2402	0.1201	31.15	<0.0001
Host	3	1.9260	0.6420	166.50	<0.0001
Parasite × host	6	0.1818	0.0303	7.86	0.0006
Error	15	0.0578	0.00386		

Does resistance of US 1 from introduced and source ranges vary?

To see if the resistance of US 1 changed quantitatively between the introduced and the source range, we compared the lab experimental infection rate by Australian *Microphallus* of US 1 collected in Australia (Fig. 3b) with that of US 1 snails collected in the USA (Fig. 2). The infection rates of US 1 snails from Australia (0.24 ± 0.08) and of US 1 snails collected in the USA (0.24 ± 0.01) were indistinguishable.

The lab infection experiment closely mimicked the natural infection rates of Australian *Microphallus*. The introduced clone US 1 was the second most common clone in the Australian source population, and was infected at 11% by the local *Microphallus* population in the field (AU field in Fig. 3). The most common clone at the site (AU 2) had the highest infection rate (44%) followed by rare clones (20%). In the laboratory infection experiment, the clonal composition and clone-specific infection rates of Australian snails were similar in rank to those in the field (AU experiment in Fig. 3). This suggests that the resistance expressed in the lab infection experiment mimicked the resistance expressed in the natural population.

The above patterns assume that Australian and North Island *Microphallus* populations are locally adapted, and that Australian *Microphallus* were not simply more infective than other parasite populations. In the reciprocal cross-infection experiment with Australian and North Island *Microphallus*, we found that both parasite populations were strongly locally adapted to their sympatric snail populations (Fig. 4); the parasite-host interaction term was significant (ANOVA, d.f. 2,8; $F = 9.008$, $P = 0.009$). There was no evidence that Australian *Microphallus* were equally infective to both North Island and Australian snails. Neither the parasite (d.f. 1,8; $F = 3.012$, $P = 0.158$) nor the host (d.f. 2,8; $F = 3.537$, $P = 0.079$) main effects were significant. In addition, Australian *Microphallus* were significantly more infective to US 1 than the North Island *Microphallus* (ANOVA, d.f. 1,35; $F = 20.238$, $P < 0.0001$). This result again suggests that US 1 did not lose resistance to allopatric parasites.

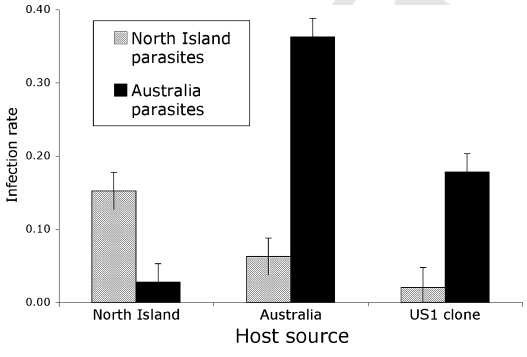

Fig. 4 Local adaptation of *Microphallus* to snails from Australian, North Island, and introduced populations of the US 1 clone by *Microphallus* sp. from Australia and North Island. US 1 retained resistance to allopatric parasites, but were susceptible to co-evolved parasites from their source range. Vertical bars show SE.

Discussion

The absence of co-evolved enemies in introduced species should favour reduced allocation to resistance that might in turn affect the demographic success of an invader. Studies of changes in resistance are rare (Maron *et al.*, 2005) and we do not know of any studies in introduced animals. Such studies are needed because several leading hypotheses for invasion success predict that allocation to resistance differs in the introduced range. We found no evidence of lower resistance in three introduced genotypes of the snail *P. antipodarum* to different populations of a co-evolved enemy *Microphallus*. In addition, there was no evidence of a change in resistance of one snail genotype (US 1) collected from the introduced range compared with the same genotype collected from the range of the parasite. However, one introduced genotype (Euro A) seems to be highly resistant, as it was not infected by either sympatric or allopatric parasites.

In the first of two tests, we exposed three introduced populations in a common garden to native range parasites that were sympatric and allopatric to their source populations. Because host-parasite co-evolution leads to genotype-specific parasite local adaptation in this system, this test allowed us to determine if introduced genotypes were susceptible to parasites with which they did not share a co-evolutionary history (Dybdahl & Lively, 1998; Lively & Dybdahl, 2000; Lively *et al.*, 2004). Susceptibility of introduced genotypes to allopatric parasite populations would have demonstrated reduced allocation to resistance. Contrary to these predictions, none of the introduced clones were susceptible to all parasite sources (Fig. 2). For two genotypes (Euro C and US 1) the pattern of infection by sympatric and allopatric parasites was consistent with local adaptation, suggesting no loss of resistance. Both snail genotypes were resistant to parasite populations that were allopatric to the source of the introduced genotypes (North and South Island of New Zealand), but not to the parasite population, i.e. sympatric with their source (Australia). The results of the cross-infection experiment (Fig. 4) show that the Australian parasite population is not simply more infective to all snail populations.

In contrast to the results for US 1 and Euro C, the Euro A clone was resistant to three parasite populations from across the range of *Microphallus.* This result is consistent with previous unpublished results for South Island parasites. It seems unlikely that the resistance of Euro A to all three parasite sources could be explained by a failure to use locally adapted parasites for two reasons. First, the mtDNA haplotype of Euro A is found in a

number of populations on the North Island of New Zealand (Stadler *et al.*, 2005; D. Drown & M.F. Dybdahl, in preparation), and well within the likely dispersal distance (Dybdahl & Lively, 1996) of the North Island parasite population used in this experiment (Lake Taupo). Second, although local adaptation by *Microphallus* leads to the highest infection rates in sympatric host populations, *Microphallus* populations are still infective to allopatric host populations and individual clones at lower rates (Lively & Dybdahl, 2000; Lively *et al.*, 2004). For example, in this study, North Island parasites are able to infect US 1, only at a lower rate, than the Australian parasites that are sympatric to the US 1 source population (Figs 2 and 4). Hence, Euro A appears to be a highly resistant genotype.

In a second assessment of change in resistance, we looked at the resistance of the US 1 genotype from the introduced and source range in the common garden lab experiment. Higher infection rates of US 1 from the introduced vs. source range would have been evidence of reduced allocation to resistance. Contrary to this prediction, US 1 collected from the introduced range and US 1 collected from its source range (Australia) were equally susceptible to parasites from Australia. These results mean that resistance of US 1 has not changed even though US 1 no longer encounters *Microphallus* in the introduced range.

Taken together, these results for US 1, Euro C and A suggest there has been no decrease in allocation to resistance, either plastic or evolved. The absence of a response might reflect an absence of selection to reduce resistance. However, a number of studies suggest that many resistance mechanisms incur fitness costs (Webster & Woolhouse, 1999; Moret & Schmid-Hempel, 2000; Hoang, 2001; reviewed in Rigby *et al.*, 2002). We also do not think that the absence of an evolved response reflects a lack of evolutionary potential in introduced clonal populations. The accumulation of mutational variation seems likely in introduced populations because of their age (up to 150 years old) and immense size (up to 800 000 individuals/m^2; Dorgelo, 1987), leading to mutation accumulation within clonal populations (Lynch & Gabriel, 1983; Lynch, 1984, 1985). Another clonal invader with large population sizes was found to exhibit genetic variation and local adaptation (Butin *et al.*, 2005). In addition, genetic variation exists for some life-history traits in the US 1 clone (Dybdahl & Kane, 2005).

The absence of a plastic decrease in resistance in US 1 might be because of immunological priming in invertebrates, where prior exposure to parasites increases resistance capacity (Little & Kraaijeveld, 2004). This is possible because both introduced and source range populations of US 1 in this experiment were collected from the field. Australian US 1 might have been exposed to *Microphallus*, and the introduced populations might have been exposed to novel parasites in the introduced range (but not infected by them). Exposure to novel parasites might maintain defensive traits, even if attacks are incompatible and unable to lead to infection (Colautti *et al.*, 2004). However, immunological priming cannot explain the absence of plastic down-regulation for Euro C and A snail clones because these experimental animals were raised in the lab and had no prior exposure. It is also possible that resource abundance, which might be typical of invaders of open niches, reduces the benefit of plastic reallocation from resistance to other fitness traits. In any case, the results for all three genotypes suggest that plastic down-regulation does not occur either because there is no benefit to reallocation, or because novel parasites stimulate immunological defences.

A greater likelihood of successful establishment and spread might be expected by introduced populations comprised of individuals more 'vigorous' than average, according to the SBH (Simons, 2003). Assuming a positive correlation between resistance and vigour, introduced genotypes might also have heightened resistance. In this study, high vigour and correlated high resistance could have been manifested in three ways: (i) if introduced clones were not infected by *Microphallus* in natural populations in their source range, (ii) if introduced clones were resistant to all experimental parasite sources, including those from the source range, or (iii) if introduced clones had lower experimental infection rates than the same clones collected from the range of *Microphallus*. Contrary to the SBH predictions, our survey of US 1 in the field showed that it was infected by *Microphallus* in the Australian source range (Fig. 3). Furthermore, US 1 and Euro C were susceptible to Australian *Microphallus* but not to populations outside their source range in the infection experiment (Figs 2 and 4). Finally, US 1 collected from the introduced range was not more resistant to *Microphallus* than US 1 collected from the source range. On the other hand, the resistance of Euro A is consistent with the SBH.

The absence of population regulation by co-evolved enemies (Regulatory Release) might provide an advantage sufficient to explain the success of some invasive species (Elton, 1958; Maron & Vila, 2001; Colautti *et al.*, 2004). One of the assumptions of this Regulatory Release hypothesis is that parasites in the native range were important regulators of their host populations (Keane & Crawley, 2002). It seems likely that *Microphallus* plays a strong role in regulating populations of *Potamopyrgus* because it sterilizes infected snails and because it regulates populations of individual clones (Dybdahl & Lively, 1998). In addition, population densities are much higher in the introduced range than they are within the range of native parasites. Abundances across 48 New Zealand streams rarely exceeded 1000 individuals/m^2 (Holomuzki & Biggs, 1999), and the highest reported densities in Australia were 50 000 individuals/m^2 (Schreiber *et al.*, 1998). In the absence of parasites, abundances in excess of 30 000 individuals/m^2 are com-

mon, and exceed 300 000 individuals/m^2 (Richards *et al.*, 2001; Hall *et al.*, 2003; Kerans *et al.*, 2005) in the western US, and up to 800 000 individuals/m^2 in Europe (Dorgelo, 1987). Thus, the absence of enemy regulation might permit greater *Potamopyrgus* densities in the introduced range.

In conclusion, the resistance of the Euro A genotype might indicate its vigour and correlated colonization advantage, consistent with the SBH. However, we found no evidence for an expected decrease in allocation to resistance by introduced genotypes of *Potamopyrgus* in their introduced ranges, as assumed under the EICA and PICA for invasion success. Introduced US 1 and Euro C were most susceptible to *Microphallus* collected from their Australian source range, as expected from local parasite adaptation, but retained resistance to allopatric parasite sources. In addition, the resistance of US 1 was identical from both the introduced and source ranges. Hence, these genotypes experienced a release from enemies, but there is no evidence for a response in resistance traits. Although it is difficult to determine the causes of invasion success, our results show that the assumptions of the EICA and PICA were not met for these three invasions of parasite-free range.

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