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The paradox of the parasites: implications for biological invasion

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The enemy-release hypothesis for biological invasions supposes that invasive species may be more successful in their introduced ranges than in their native ranges owing to the absence of coevolved natural enemies. Recent studies supporting this hypothesis have found that introduced plants and animals are less parasitized in their introduced ranges than in their native ranges. Expanding on this theory, I hypothesize that the role of enemy release may differ among the introduction, establishment and spread phases of an invasion. I present a simple model indicating that parasite release is unlikely to greatly affect the chance of establishment in populations with and without an immune subpopulation. The specific numerical relationship between the number of individuals introduced and the chance of establishment depends on a relationship between virulence, here conceptualized as the chance for the extinction of a lineage, and the fraction of the population infected at introduction. These results support the idea of a 'filter effect' in which different biological processes regulate the different phases of an invasion.

Keywords: biological invasion; enemy-release hypothesis; extinction probability; parasitism

1. INTRODUCTION

Some invasive species exhibit remarkable population growth in their introduced ranges. The enemy-release hypothesis supposes that introduced organisms may be released from regulation by natural enemies such as herbivores, predators, parasites and pathogens and thereby attain higher individual and population growth rates. Evidence for this hypothesis comes primarily from experiments with exotic plants and biological control releases (reviewed in Keane & Crawley 2002) and from two recent studies (Mitchell & Power 2003; Torchin *et al.* 2003), which found that on average the number of species parasitizing naturalized plants ($N = 473$) and animals ($N = 26$) in their introduced ranges was less than in their native ranges. These studies indicate that demographic release from parasites and pathogens may be an important factor in determining invasion success.

Invasion success, however, is a vague concept and might refer to the chance of establishment, the rate of spread, the maximum population density obtained or range size, or maximum body size and individual growth rate (Torchin *et al.* 2002). Different theories predict each of these hypothetical effects of enemy release, any or none of which may therefore occur in specific cases. For example, though Torchin *et al.* (2001) found evidence that parasite release may contribute to faster growth and greater biomass in introduced populations of the invasive green crab *Carcinus maenas*, it has not yet been shown if parasite release affects establishment probability in this species. Indeed, to my knowledge, the effect of parasite release on the chance of establishment has not yet been demonstrated for any species.

In this paper, I use a simple theoretical model to explore the possible effects of parasite release on the chance of establishment. In a typical case, the parasites of concern are transported with their hosts (in contrast to being acquired in the recipient environment). Assuming that the chance of transporting the parasite with the host is equivalent to the chance that one of the transported organisms is carrying the parasite, then the chance for parasite release declines exponentially with the number of individual hosts introduced. Therefore, if parasite release is important for invasion success, a reasonable hypothesis is that the chance of establishment also declines with the number of organisms introduced. However, this hypothesis contradicts theoretical predications (Richter-Dyn & Goel 1972) and data (Williamson 1996; Grevstad 1999) indicating that establishment probability usually increases with the number of individuals introduced because larger populations are less susceptible to demographic stochasticity and environmental variability. One possible resolution to this paradox is that there is a trade-off between parasite release and insurance against demographic fluctuations. Thus, an intermediate population size would exhibit the greatest probability of establishment. The consequences of this trade-off are easily explored. I formulate a model describing this situation and consider the chance of establishment, defined as the complement of the chance of extinction.

2. MATERIAL AND METHODS

The chance of establishment is defined as $(1 - p_{\text{ext}})$, where p_{ext} is the total probability of extinction from all causes. I consider extinctions caused by demographic stochasticity under conditions of parasitism and no parasitism. The chance of establishment, therefore, is

$$p_{\text{est}} = (p_{\text{est}} | \text{parasitized})\, p(\text{parasitized}) + (p_{\text{est}} | \text{not parasitized})(1 - p(\text{parasitized})). \quad (2.1)$$

If the probabilities that individual animals are parasitized are independent and equal with probability p, then the probability that at least one individual in an introduced population of size n carries the parasite is $(1 - q^n)$, where $q = (1 - p)$. There are many forms that the conditional probabilities of establishment could take. A simple model supposes no density dependence and that all individuals have an equal chance of persisting in the new environment. Suppose that the probabilities of extinction for the descendants of each individual of an introduced parasitized or non-parasitized population are ε_1 and ε_2, respectively. In this way, all the relevant manifestations of virulence (premature death and reductions in fitness) are integrated into the single parameter ε. Furthermore, the extinction probabilities for the lineages of all individuals are the same within a population, but differ depending on whether or not the population is infected. The probability that an introduced population of n individuals will fail is ε_1^n or ε_2^n depending on whether or not the population carries the parasite. Substituting these values in equation (2.1) and rearranging, the total probability of establishment is

$$p_{\text{est}} = 1 - \varepsilon_1^n + q^n(\varepsilon_1^n - \varepsilon_2^n). \quad (2.2)$$

One interpretation of equation (2.1) is that the infection dynamics are sufficiently rapid and the population sufficiently mixed such that if the introduced population contains a single infected individual the entire population will be exposed and suffer the fate of infection. For many introduced species this is not an unreasonable assumption. After all, the means by which non-indigenous species are commonly introduced—trade in agriculture, horticulture and raw materials—induce crowded conditions prone to epidemics. Nevertheless,

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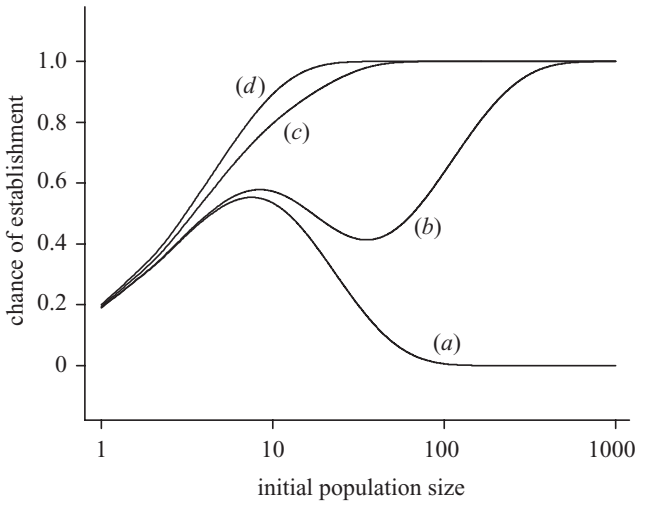

Figure 1. In populations without immunity, parasite release only enhances the chance of establishment in populations with unrealistically severe parasitism. The differences between (*c*) realistic parasite release and (*d*) no effect of parasitism, are negligible compared to parasite release from (*a*) lethal parasitism and (*b*) severe parasitism. Parameter values are as follows: (*a*) $\varepsilon_1 = 1.0$, $q = 0.95$, $\varepsilon_2 = 0.8$; (*b*) $\varepsilon_1 = 0.99$, $q = 0.95$, $\varepsilon_2 = 0.8$; (*c*) $\varepsilon_1 = 0.90$, $q = 0.95$, $\varepsilon_2 = 0.8$; (*d*) $q = 1$, $\varepsilon_2 = 0.8$.

equation (2.2) can be modified to allow a certain fraction of the population, through immunity or otherwise, to escape infection. Whereas the original model supposed that an infected individual would initiate an epidemic, possibly leading to the demise of the entire population, a more general model would permit parasitism to reduce the effective size of the population by reducing the chance of persistence only of certain lineages (the susceptible ones). Suppose that the chance of extinction for lineages of immune individuals in parasitized populations is ε_2—the same as for individuals in non-parasitized populations in the previous model—then the chance of extinction for a parasitized population is the probability that the lineages of all immune and susceptible individuals go extinct, i.e.

$$(p_{\text{ext}} \mid \text{parasitized}) = p_{\text{ext}}(\text{immune lineages})\, p_{\text{ext}}(\text{susceptible lineages}). \tag{2.3}$$

Let $\pi (0 \leqslant \pi \leqslant q)$ be the fraction of the population exhibiting immunity. In this case, the parameterized version of equation (2.1) with the chance of extinction for parasitized populations given by equation (2.3) is

$$p_{\text{est}} = 1 - (\varepsilon_2^{\pi n} \varepsilon_1^{(1-\pi)n}(1 - q^n) + (\varepsilon_2^n q^n)). \tag{2.4}$$

Two special cases of this model are of interest.

(**a**) ***Special case 1***

The generalized model (2.4) exhibits the desirable property that the original model (2.2) is recovered as a special case at $\pi = 0$, i.e. where no individuals are able to escape infection.

(**b**) ***Special case 2***

In the case of the original model, the idea was that infection of individuals after introduction might create an epidemic causing the entire introduced population to fail. The general model allows the lineages of healthy individuals, even of parasitized populations, high probabilities of persistence. Therefore, another special case of equation (2.4) is where parasitized individuals do not transmit the parasite to any uninfected individuals after introduction. Here, immune individuals are just those that were not infected before introduction, i.e. $\pi = q$. In this case, parasitized individuals merely exert a drag on the probability of population persistence, which can be conceived as a reduction in the effective size of the population.

3. RESULTS

In some cases these models indeed exhibit an optimal chance of establishment at intermediate population sizes (figure 1). In particular, if the effect of having the parasite is so severe in the new environment that the extinction of a parasitized lineage is certain, the probability of establishment exhibits the shape hypothesized (figure 1*a*; $\varepsilon_1 = 1.0$, $q = 0.95$, $\varepsilon_2 = 0.8$). However, if extinction is only very likely ($\varepsilon_1 = 0.99$) the probability of establishment first increases, then decreases, then increases again to 1 (figure 1*b*). Finally, if the consequences of parasitism are not sufficiently great ($\varepsilon_1 = 0.90$) the effects of parasite release are outweighed by the effect of increasing population size (figure 1*c*). This result is very close to the chance of establishment without parasites at all (figure 1*d*; $q = 1$).

The model with immunity shows similar behaviours (figure 2). However, in this model immunity (π) interacts with virulence (ε_1) to create a variety of different patterns. For instance, if the parasite is lethal ($\varepsilon_1 = 1.0$; figure 2*a*) even a small amount of immunity generates a considerable chance for invasion. Compare this result, in which persistence is owing to immunity, with figure 1 in which persistence is owing to lesser virulence. Similarly, if the effect of the parasite is severe ($\varepsilon_1 = 0.99$; figure 2*b*) immunity in the population can increase the chance of establishment considerably. However, if the parasite is not sufficiently virulent ($\varepsilon_1 = 0.90$; figure 2*c*) the effects of immunity are negligible. In contrast to zero or partial immunity, the special case where $\pi = q$ nowhere exhibits an optimal chance of establishment at intermediate initial population sizes (figure 2*d*). This is because there is really no possibility of parasite release: all individuals who are not immune have already been infected. Instead, the result is to reduce the chance of establishment overall, but not to cause a qualitative change in the shape of the result.

4. DISCUSSION

In this paper I have looked at the effect of one kind of enemy release—release from parasites and pathogens—on the chance of establishment for introduced populations. In the least severe case of parasitism presented here (figure 1*c*), virulence is similar to that of rinderpest south of the Zambesi River (Dobson & May 1986), surely one of the most severe epidemics ever studied. Thus, it may reasonably be inferred that parasite release is not likely to greatly affect the chance of population establishment.

This conclusion would not hold, however, if the parasite was more virulent in the introduced environment than in the former range. The usual arguments about the evolution of virulence suggest that if virulence is functionally related to transmission efficiency in reasonable ways, extremely high virulence will not evolve in the endemic region. Rather, an intermediate grade of virulence is likely to result, the details of which depend on other factors in complicated ways (May & Anderson 1990). These arguments, of course, pertain to the evolution of the parasite in the endemic region only. It does not follow, therefore, that the parasite will not be extremely lethal in a new environment with conditions to which it has not had the opportunity to adapt. The level of virulence in the new environment will depend on contingent factors related to the physiology, immunology and population biology of the host in the recipient environment. Curiously, the extreme cases cited by Dobson & May (1986) are exactly cases where epidemics were caused by introduced pathogens. These are rinderpest in Africa, the myxoma virus in

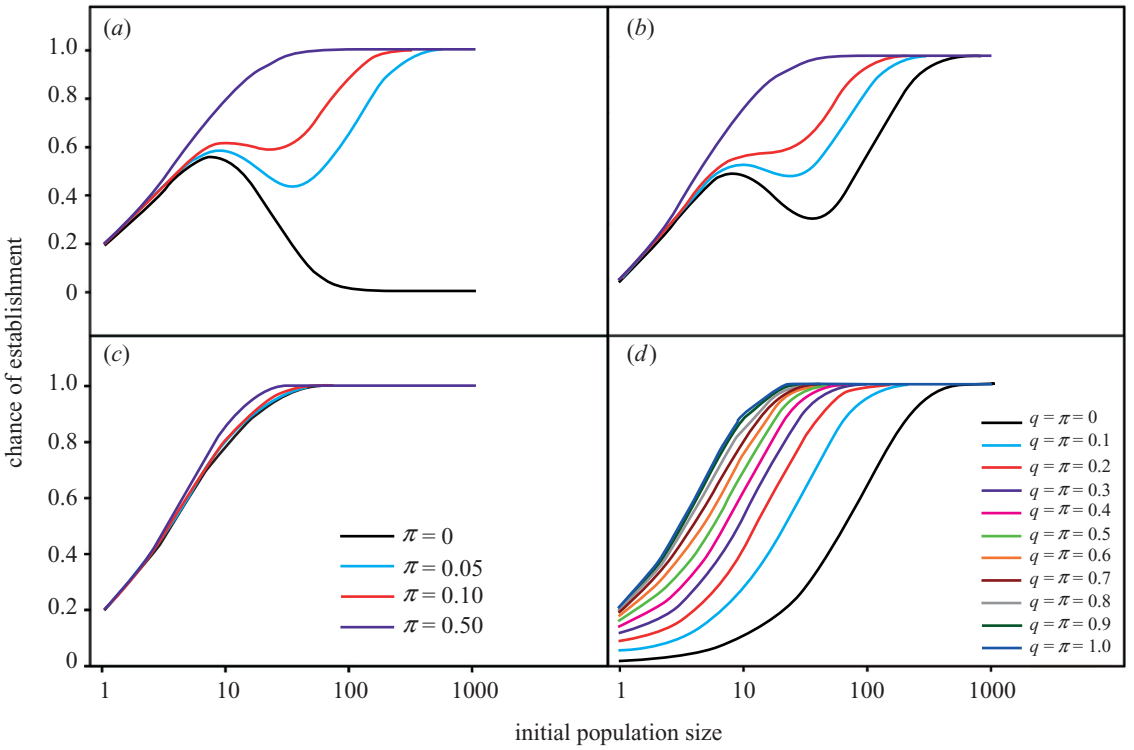

Figure 2. In populations with immunity, moderate numbers of immune individuals can lead to parasite release in populations with (*a*) lethal parasitism and (*b*) severe parasitism. However, even considerable immunity does not greatly affect parasite release in populations with (*c*) realistic levels of parasitism. A special case (*d*) is introduced populations in which only individuals infected at introduction are susceptible. In this case, infected individuals exert a drag on the chance of establishment, increasing as the fraction (π) of individuals infected approaches 1. Other parameter values are (*a*) $\varepsilon_1 = 1.0$, $q = 0.95$, $\varepsilon_2 = 0.8$; (*b*) $\varepsilon_1 = 0.99$, $q = 0.95$, $\varepsilon_2 = 0.8$; (*c*) $\varepsilon_1 = 0.90$, $q = 0.95$, $\varepsilon_2 = 0.8$; (*d*) $\varepsilon_1 = 0.99$, $q = \pi$, $\varepsilon_2 = 0.8$.

Australia and chestnut blight in North America. In such cases, host establishment probabilities like those in figure 1*a*,*b* are indeed conceivable.

The model presented here is a rather simple abstraction of the underlying biology (though simpler models have sometimes been proposed). Indeed, a more rigorous derivation of ε^n is readily available, for instance as the solution to a simple birth–death process. Moreover, future developments of this theory should explore other potentially important factors including parasitism by multiple species, density dependence, and heterogeneity and correlations in susceptibility. Additionally, this model does not make the usual distinction between microparasite and macroparasite infections. These factors are unlikely to modify the general result, however, and it is reasonable to expect that the qualitative result obtained here will continue to hold: virulence must be unusually severe if parasite release is to greatly affect the chance of population establishment.

In conclusion, the idea that the process of invasion may be conceptualized as a sequence of three steps involving introduction, initial establishment and subsequent spread in a landscape is useful (Kolar & Lodge 2001). As the mechanisms that control these processes are different, it is reasonable to suppose that different conditions will determine success in each phase. Hence, invasiveness is a composite characteristic of species that are capable of passing through multiple 'filters'. With respect to the chances for establishment and spread, invasiveness must therefore be defined with reference to the recipient environment. Though parasite release may possibly be important in permitting invasive species to spread in some environments, and has been shown to be important for individual growth, the results of this study indicate that it is unlikely to be important for establishment.

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Dobson, A. P. & May, R. M. 1986 Patterns of invasions by pathogens and parasites. In *Ecology of biological invasions of North America and Hawaii* (ed. H. A. Mooney & J. A. Drake), pp. 58–76. New York: Springer.

Grevstad, F. 1999 Experimental invasions using biological control introductions: the influence of release size on the chance of population establishment. *Biol. Invasions* **1**, 313–323.

Keane, R. M. & Crawley, M. J. 2002 Exotic plant invasions and the enemy release hypothesis. *Trends Ecol. Evol.* **17**, 164–170.

Kolar, C. S. & Lodge, D. M. 2001 Progress in invasion biology: predicting invaders. *Trends Ecol. Evol.* **16**, 199–204.

May, R. M. & Anderson, R. M. 1990 Parasite–host coevolution. *Parasitology* **100**, S89–S101.

Mitchell, C. E. & Power, A. G. 2003 Release of invasive plants from fungal and viral pathogens. *Nature* **421**, 625–627.

Richter-Dyn, N. & Goel, N. S. 1972 On the extinction of a colonizing species. *Theor. Popul. Biol.* **3**, 406–433.

Torchin, M. E., Lafftery, K. D. & Kuris, A. M. 2001 Release from parasites as natural enemies: increased performance of a globally introduced marine crab. *Biol. Invasions* **3**, 333–345.

Torchin, M. E., Lafferty, K. D. & Kuris, A. M. 2002 Parasites and marine invasions. *Parasitology* **124**, S137–S151.

Torchin, M. E., Lafferty, K. D., Dobson, A. P., McKenzie, V. J. & Kuris, A. M. 2003 Introduced species and their missing parasites. *Nature* **421**, 628–630.

Williamson, M. 1996 *Biological invasions*. London: Chapman & Hall.