Fitness costs in spatially structured environments

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Abstract

The clustering of individuals that results from limited dispersal is a double-edged sword: while it allows for local interactions to be mostly among related individuals, it also results in increased local competition. Here I show that, because they mitigate local competition, fitness costs such as reduced fecundity or reduced survival are less costly in spatially structured environments than in non spatial settings. I first present a simple demographic example to illustrate how spatial structure weakens selection against fitness costs. Then, I illustrate the importance of disentangling the evolution of a trait from the evolution of potential associated costs, using an example taken from a recent study investigating the effect of spatial structure on the evolution of host defence. In this example indeed, the differences between spatial and non-spatial selection gradients are entirely due to differences in the fitness costs, thereby undermining interpretations of the results made in terms of the trait only. This illustrates the need to consider fitness costs as proper traits in both theoretical and empirical studies.

Introduction

Most populations in nature exhibit some form of spatial structure; this can be because their habitat is fragmented, but also, even in the absence of patchiness, because there are limits to the distances an individual can disperse and because ecological interactions are usually local (Tilman and Kareiva, 1997). Theoretical models have shown that limited dispersal and localised interactions influence demographic processes, such as population growth (Law et al., 2003), epidemiological processes such as the invasion threshold of parasites (Sato et al., 1994; Keeling, 1999), and evolutionary processes, such as the evolution of dispersal (Hamilton and May, 1977; Ferrière and Le Galliard, 2001), altruistic behaviour (Lehmann and Keller, 2006; Lehmann and Rousset, 2010), reproductive effort (Pen, 2000; Lion, 2010), parasite virulence (Boots and Sasaki, 1999; Lion and Boots, 2010) and host defence (Frank, 1998; Best et al., 2011; Débarre et al., 2012). When dispersal is spatially limited, individuals aggregate within clusters (Lion and van Baalen, 2008) and related individuals tend to live next to one another. This clustering can be beneficial and is for instance key to the evolution of altruism, but it also results in increased local competition, which can annihilate

the beneficial effects of clustering (Wilson et al., 1992; Taylor, 1992; Taylor et al., 2011; Débarre et al., 2014). Consequently, traits able to alleviate this local competition can be selected for.

It is usually assumed that new or improved traits come with fitness costs, because of pleiotropic effects or metabolic costs. This is for instance the case for traits of defence against natural enemies. Mounting a defence against parasites can involve the diversion of resources that would otherwise have been used for another purpose (Sheldon and Verhulst, 1996); the chemicals used in the defence can also harm the host (auto-toxicity, Purrington, 2000). In addition to these direct costs, defence traits may also have indirect costs, such as the deterrence of mutualists, or a reduced competitive ability (Strauss et al., 2002). It is therefore common in theoretical studies to assume that the trait of interest is costly. Fitness costs are often considered as a logical necessity to avoid the evolution of Darwinian demons (Reznick et al., 2000) (a situation which, from a theoretical point of view, has a limited interest), but are seldom considered as traits under selection themselves.

Still, when comparing the evolution of a costly trait in spatial *vs.* non-spatial (well-mixed) environments, it is crucial to consider the costs as correlated traits that are also under selection. Indeed, this article shows that spatial structure mitigates fitness costs, and that this result may affect the way we interpret differences in the evolution of specific traits in spatial *vs.* non-spatial contexts, highlighting the limits of adaptationist interpretations. I first consider a simple model of a population living in a lattice, where reproduction is density-dependent. The decomposition of a selection gradient shows why selection against a reduced fecundity (or against a decreased survival) is less strong in a spatial context than in a non-spatial context. I then assume that individuals can be infected by a directly transmitted parasite, and I study the evolution of reduced susceptibility to the disease, using the same model as Best et al. (2011). Again, I decompose the selection gradient, identifying terms due to the trait itself and terms due to the associated cost, a reduced fecundity. This decomposition reveals that spatial structure does not influence the evolution of reduced susceptibility itself: the change in the evolved level of host susceptibility in spatial *vs.* non-spatial environments is instead a by-product of selection on its associated cost.

Demographic model

In this first example, we follow the density dynamics of a population of clonally reproducing individuals, when reproduction is density dependent. We assume that there is a large number of breeding sites in the population, and that each site can host at most one individual. Each site is therefore either empty ($\circ$) or occupied (S).

We denote by b individual fecundity (notation is summarised in table 1). An individual can only reproduce if there are empty sites available to host its offspring. With probability $1-g_R$, reproduction is local, meaning that the offspring can only be sent to the neighbouring breeding sites; with probability g_R, reproduction is global: the offspring can be sent to any empty breeding site in the environment (see figure 1). Death, on the other hand, is density independent, and occurs at a rate d; this is the rate at which an occupied site (S) becomes empty again ($\circ$). We denote by p_S the global density of occupied sites (number of occupied sites divided by the total number of sites in the environment); the global density of empty sites is $p_\circ = 1-p_S$. The quantity $q_{\circ|S}$ is the local density of empty sites around an occupied site, that is, the probability of finding an empty site in the neighbourhood of an occupied site. With this, the density dynamics of the density of occupied sites can be written using the following spatial moment equation (Rand, 1999; van Baalen, 1998, 2002):

$$\frac{dp_S}{dt} = b\,\left((1-g_R)\,q_{\circ|S} + g_R\,p_\circ\right)\,p_S - d\,p_S. \tag{1}$$

This population, called the "resident" population, is assumed to be at equilibrium, and we denote by $q^*_{\circ|S}$ and $p^*_\circ$ the equilibrium values of the local and global densities of empty sites, respectively. Setting equation (1) equal to zero, we obtain (Lion, 2010)

$$R^* \equiv b\,\left((1-g_R)\,q^*_{\circ|S} + g_R\,p^*_\circ\right) - d = 0. \tag{2}$$

We then assume that a mutant appears, with a different fecundity (b') and/or death rate (d'). The mutant is initially rare, and the invasion dynamics of this rare mutant are given by:

$$\frac{dp_{S'}}{dt} = b'\,\left((1-g_R)\,q_{\circ|S'} + g_R\,p^*_\circ\right)\,p_{S'} - d'\,p_{S'}. \tag{3}$$

The mutant can establish in the population when $R' > 0$, with

$$R' = b' \left((1 - g_R)\, q_{\circ|S'} + g_R\, p^*_\circ \right) - d'. \tag{4}$$

We assume that mutant and resident individuals are phenotypically close: the mutation is of small phenotypic effect, so that we can write $b' = b + \partial b$ and $d' = d + \partial d$. Consequently, the local density of empty sites seen by a mutant individual is also not too different from the local density of empty sites seen by a resident individual, so that $q_{\circ|S'} = q^*_{\circ|S} + \partial q_{\circ|S'}$. Using the definitions of R' and R^* (equations (2) and (4)), we can express the selection gradient $\partial R'$ as follows:

$$\partial R' = R' - R^* = \underbrace{\partial b\, \frac{d}{b} - \partial d}_{\partial R'_{\text{self}}} + \underbrace{(1 - g_R)\, b\, \partial q_{\circ|S'}}_{\partial R'_{\text{demo}}}. \tag{5}$$

This selection gradient is the sum of two terms. The first term, $\partial R'_{\text{self}}$, represents the direct effects of the mutation on a mutant's own fitness; it does not depend on whether reproduction is local or not. The second term, $\partial R'_{\text{demo}}$, accounts for the changes in the demographic structure of the population due to the mutation, via the term $\partial q_{\circ|S'}$, which is the change in the local density of empty sites around an mutant individual, compared to around a resident individual, at equilibrium. This second term vanishes in a non-spatial model, in which $g_R = 1$.

In this model, how spatial structure affects the invasion of the mutant is exclusively controlled by $\partial R'_{\text{demo}}$. Compared to a non-spatial setting, spatial structure favours the invasion of the mutant if $\partial q_{\circ|S'} > 0$, *i.e.*, if mutants see more empty sites around themselves than residents do. This is the crucial point of our argument.

Let us consider a mutant that has a reduced fecundity, a feature that will later be qualified as a fitness cost (the argument goes the same way if we consider changes in the death rate). In a spatial setting, reproduction is mostly local, and related individuals tend to cluster. Mutants have a lower fecundity ($\partial b < 0$), hence have more empty sites in their neighbourhood than residents do: $\partial q_{\circ|S'} > 0$, so that $\partial R'_{\text{demo}} > 0$. In both cases, though, $\partial R'_{\text{self}}$ is negative and is the leading term of the selection gradient, so that the mutant is eventually counter-selected. But it is less strongly counter-selected in a spatial context than a a

non-spatial context: spatial structure mitigates the fitness cost. Conversely, a mutant with an increased fecundity ($\partial b > 0$) sees a lower local density of empty sites ($\partial q_{\circ|S'} < 0$), yielding $\partial R'_{\text{demo}} < 0$; it is therefore less strongly favoured in a spatial context than in a non-spatial context.

Figure 2 illustrates this result; the selection gradients are calculated numerically, using the pair approximation (Matsuda et al., 1992; Nakamaru et al., 1997) to evaluate local densities. The R codes to run the model as available on figshare, `http://dx.doi.org/10.6084/m9.figshare.1183435`.

Spatial structure therefore affects the magnitude of the effect of the fitness cost: it makes fitness costs less costly. We will now see why this matters.

Evolution of host susceptibility

We now consider the evolution of a trait of defence against parasites, namely, the evolution of avoidance (or reduced susceptibility), as studied by Best et al. (2011). I use the same model and the same assumptions as Best et al., but with the notation of (Débarre et al., 2012), where the decomposition of the selection gradient used in this study was introduced. As in Best et al. (2011), we will assume that defence is costly, and that a reduced susceptibility to the infection comes at the cost of a reduced fecundity.

The basic assumptions are the same as previously (one individual per site, density-dependent reproduction), but we now assume that the individuals can be infected by a parasite. Infected individuals cannot reproduce nor recover: the infected state is a dead-end, and we denote by ν the additional mortality due to the infection (also called virulence (Read, 1994)). With a probability $1-g_T$, an infected individual can only infect its (healthy) neighbours; with probability g_T, transmission is global: an infected individual can infect any healthy individual in the population. A parameter β denotes the transmissibility of the parasite, while a parameter α denotes the susceptibility of a healthy host. With these assumptions, the dynamics of the density of sites occupied by healthy (p_S) and infected (p_I) individuals are given by the following system (notation is recapitulated in table 1):

$$\frac{dp_S}{dt} = \left[b\,\left((1-g_R)\,q_{\circ|S} + g_R\,p_\circ\right) - d\right]\,p_S - \alpha\,\beta\,\left((1-g_T)\,q_{I|S} + g_T\,p_I\right)\,p_S, \quad (6a)$$

$$\frac{dp_I}{dt} = \alpha\,\beta\,\left((1-g_T)\,q_{I|S} + g_T\,p_I\right)\,p_S - (d+\nu)\,p_I. \quad (6b)$$

As previously, we assume that the population (called the "resident" population) is at equilibrium and we use a star * to denote global and local densities evaluated at this equilibrium. We assume that a mutant appears, with a different susceptibility to the infection $\alpha' = \alpha + \partial\alpha$, and different fecundity, $b' = b + \partial b$ (the product $\partial\alpha.\partial b$ is positive). The sign of the selection gradient $\partial R'$ indicates whether these mutants can establish; Débarre et al. (2012) have shown that the selection gradient can be expressed as follows:

$$\partial R' = \underbrace{\frac{1}{H^*}\left[\partial b\,\left((1-g_R)\,q^*_{\circ|S} + g_R\,p^*_\circ\right) - \frac{B^*}{H^*}\partial\alpha\,\beta\,\left((1-g_T)\,q^*_{I|S} + g_T\,p^*_I\right)\right]}_{\partial R'_{\text{self}}}$$
$$\underbrace{+\frac{1}{H^*}\,(1-g_R)\,\partial q_{\circ|S'}\,b}_{\partial R'_{\text{demo}}} \quad \underbrace{-\frac{B^*}{H^*}\frac{1}{H^*}\,(1-g_T)\,\partial(q_{I'|S'} + q_{I|S'})\,\alpha\,\beta}_{\partial R'_{\text{epi}}}. \quad (7)$$

where

$$B^* = b\,\left((1-g_R)\,q^*_{\circ|S} + g_R\,p^*_\circ\right) - d\,, \text{ and}$$
$$H^* = \alpha\,\beta\,\left((1-g_T)\,q^*_{I|S} + g_T\,p^*_I\right).$$

[The method to derive equation (7) is detailed in Débarre et al. (2012, Appendix C). The derivation uses a next-generation approach (Diekmann et al., 1990; van den Driessche and Watmough, 2002; Hurford et al., 2010) and techniques developed in Lion and van Baalen (2007).]

We note that the expression of B^* is identical to the expression of R^* in the demographic model (equation (2)), except that this quantity is not equal to zero anymore, for the density of healthy individuals is also affected by infection dynamics (see equation (6a)).

The interpretation of the first two terms of the selection gradient (7) is the same as in the previous section: $\partial R'_{\text{self}}$, corresponds to the direct effects of the mutation on the mutants'

own fitness, and $\partial R'_{\text{demo}}$ takes into account changes in the demographic structure of the population. A third term, $\partial R'_{\text{epi}}$, corresponds to changes in the epidemiological structure of the population, via the terms $\partial q_{I'|S'}$ and $\partial q_{I|S'}$, whose sum corresponds to the changes in the density of infected individuals (resident or mutant) in the neighbourhood of a healthy mutant individual. Both $\partial R'_{\text{demo}}$ and $\partial R'_{\text{epi}}$ vanish in a non-spatial context, when reproduction and transmission are purely global ($g_R = g_T = 1$).

The selection gradient (7) conflates the effects of changes in the trait of interest (α) and the associated cost (b), but we can disentangle these effects, by noting that

$$\partial R' = \partial R'^{(\text{trait})} + \partial R'^{(\text{cost})}, \tag{8}$$

where $\partial R'^{(\text{trait})}$ is the selection gradient that we would obtain if the trait under selection had no associated cost ($b' = b$), while $\partial R'^{(\text{cost})}$ is the selection gradient obtained when mutants only carry the cost, but have the same trait as the residents ($\alpha' = \alpha$). Both can be further subdivided into direct, demographic and epidemiological components, as in equation (7).

Let us compare the global selection gradient in a purely spatial setting ($\partial R'_S$, when $g_R = g_T = 0$) and purely non-spatial setting ($\partial R'_{NS}$, when $g_R = g_T = 1$). Figure 3(a) shows that $\partial R'_S < 0 \leq \partial R'_{NS}$: lower susceptibility to the disease (an avoidance defence mechanism) evolves in a spatially structured environment, while a non-spatial environment selects for a higher susceptibility to the disease. A dissection of the selection gradient is going to tell us where this difference comes from.

Let us start with the effect of the cost, a reduced fecundity, because this effect is similar to the situation studied in the demographic model (see figure 3(c)), except that the $\partial R'_{\text{self}}$ terms now differ between the spatial and non-spatial settings (in figure 3(c), the thick grey curve and thin black curve are not exactly superimposed anymore), and there is an additional $\partial R'_{\text{epi}}$ term in the spatial setting (dot-dashed curve). The argument however remains: the cost is less costly in a spatial setting.

Let us now turn to the effect of the trait itself, in the absence of cost. Figure 3(b) illustrates the fact that $\partial R'^{(\text{trait})}_{NS}$ and $\partial R'^{(\text{trait})}_{S}$ are almost identical: the thick grey and thick black curves are almost on top of each other. In the spatial context, the effects of the demographic and epidemiological structures ($\partial R'_{\text{demo}}$ and $\partial R'_{\text{epi}}$, dashed and dot-dashed

thin black curves) compensate each other. The overall effect of spatial structure on the evolution of susceptibility to the disease is negligible.

Now going back to the global selection gradient, encompassing the trait and its associated cost (figure 3(a)), we now understand that the difference between the spatial and non-spatial settings are in fact almost entirely driven by the fitness cost, and not by the trait of interest itself.

Discussion

While evolutionary studies commonly assume that a change in trait of interest comes with an associated cost, the cost itself is seldom considered as a trait in its own right. In this article, I show that overlooking that a cost is a jointly evolving, correlated trait can lead to erroneous interpretations. I use an example taken from a recent study by Best et al. (2011), investigating the effect of spatial structure on the evolution of host susceptibility to a disease. Assuming that a lower susceptibility to a disease is associated to a lower host fecundity, Best et al. (2011) found that higher levels of host susceptibility evolve in a non-spatial setting than in a spatial setting. Decomposing selection gradients into terms due to the trait (host susceptibility) and the cost (fecundity), and dissecting these terms into a direct effect (effect of the change on the individual itself), as well as demographic and epidemiological effects (changes in the spatial structure of the population), I show that spatial structure actually almost does not influence the evolution of host susceptibility strictly speaking. Instead, spatial structure makes fitness costs less costly, which indirectly leads to lower levels of host susceptibility to the disease in a spatial setting.

Why are fitness costs less costly in a spatial setting? The model assumes that reproduction is density-dependent: there is a fixed (but large) number of breeding sites, and an individual can only reproduce if it has access to empty sites. When reproduction is local, an individual can only reproduce in the nearby sites (see figure 1). A mutant who reproduces less will have more empty sites nearby, which is beneficial to the individual and its neighbours, also likely to be mutants themselves. This effect disappears in a non-spatial setting, where an individual can send its offspring to any empty site in the entire environment. In both cases, a lower fecundity remains directly detrimental, and is therefore not selected for,

but selection against a lower fecundity is weaker in a spatial setting (see figure 2).

Best et al. (2011)'s result happens to almost be entirely due to weaker selection against the cost in a spatial setting; interpretations of the result should therefore be in terms of the cost, instead of being in terms of the trait only. There is not effect of kin selection on the evolution of host susceptibility in this model, therefore no need for an adaptationist explanation of the result, because it is the product of correlated selection on another trait, the fitness cost (Gould and Lewontin, 1979).

And yet, in a seminal model, Frank (1998) showed that increased relatedness led to more disease avoidance, equivalent to a lower host susceptibility. How does Frank's result compare to ours? Importantly, Frank's kin selection model lacks two features that are key to our model. First, there are not any explicit epidemiological dynamics in Frank's model: the probability of future attack (parameter a in his model) is a constant. In other words, the force of infection is constant, meaning that the dynamics of parasite densities are not affected by the availability of hosts in the population: there are no epidemiological feedbacks (Boots et al., 2009). Second, and more importantly, there is no density-dependence in Frank's model: the fitness of individuals does not depend on the local or global density of hosts—in other words, the host population is exponentially growing, and there are no demographic feedbacks. The absence of density-dependence has two consequences. First, the fitness cost (c in Frank's model) is not less costly in a spatial setting, because there is no local competition for space that the cost could alleviate. Second, the demographic term of the selection gradient, that compensated the epidemiological term, disappears, as if we removed the dashed curve in figure 3(b): the selection gradient in the spatial setting changes and becomes in favour of a lower susceptibility.

To conclude, our study shows that, when reproduction is density-dependent, fitness costs are less costly in a spatial setting than in a non-spatial setting. This highlights the need to consider costs as jointly evolving correlated traits in both empirical and theoretical studies, and whenever possible, to study the effects of the trait and the costs independently, to disentangle their relative contributions. On a positive note finally, the finding that spatial structure actually almost does not directly influence the evolution of host susceptibility undermines Best et al. (2011)'s dramatic conclusion that a more globalized world could lead

to lower levels of host defence against parasites.

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Tables and figures

b	Fecundity of healthy individuals
d	Death rate
β	Disease transmissibility
α	Susceptibility to the disease
ν	Additional death rate due to the disease
g_R	Probability of global reproduction
g_T	Probability of global transmission
p_x	Global density of sites of type x
$q_{x\|y}$	Local density of sites of type x around a site of type y

Table 1: Notation

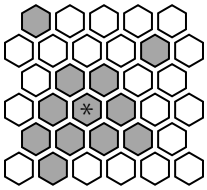

Figure 1: Density-dependent reproduction and limited dispersal lead to increased local competition. Empty sites are in white, occupied sites in grey. With purely local reproduction ($g_R = 0$), the individual in the starred site cannot reproduce, because there is currently no empty site in its neighbourhood. With purely global reproduction ($g_R = 1$), on the contrary, this individual can reproduce because it has already access to 21 empty sites.

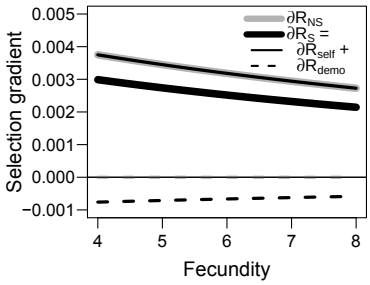

Figure 2: Selection gradients (thick curves) and their decomposition (thin curves) in the demographic model, when only individual fecundity evolves. In grey: selection gradient in a non-spatial model, $\partial R'_{NS}$ (when $g_R = 1$); in black, selection gradient in the spatial model, $\partial R'_S$ (when $g_R = 0$). The thin full curve is $\partial R'_{\text{self}}$; it is the same in both the spatial and non spatial models and appears on top of the thick grey curve; the thin dashed black curve is $\partial R'_{\text{demo}}$. Both ∂R_{NS} and ∂R_S are positive: higher values of the fecundity parameter b are favoured by selection, but $\partial R_S < \partial R_{NS}$. Parameters: $d = 1$, and each individual has $n = 4$ neighbours in the spatial model.

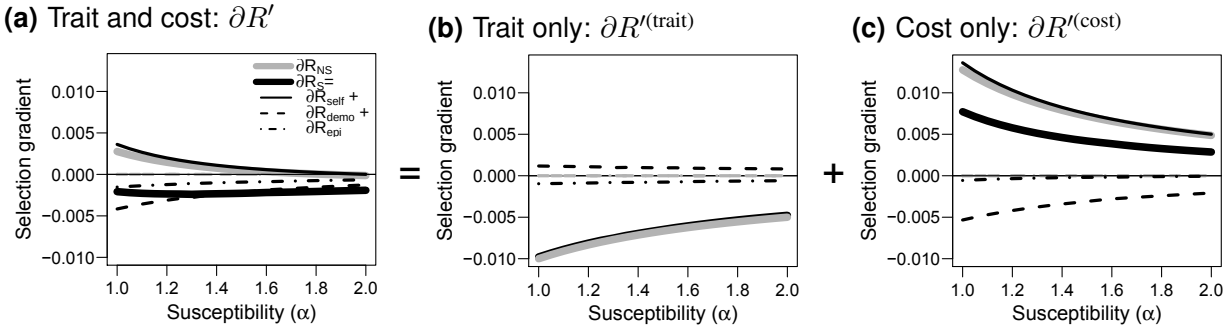

Figure 3: Selection gradients (thick curves) and their decomposition (thin curves), in the non-spatial (grey; $g_R = g_T = 1$) and spatial (black; $g_R = g_T = 0$) models. In (a), both the trait (susceptibility to the disease, α) and the cost (fecundity, b) evolve jointly. In (b), only the trait evolves, mutants have the same fecundity as residents; in (c), only the cost evolves, mutants have the same susceptibility as residents. Parameters: same as in figure 2a in Best et al. (2011): $d = 0.1$, $\beta = 1$, $\nu = 0.1$, $b(\alpha) = 4 * (-0.2 + 1.2\alpha)/(0.9 + 0.1\alpha)$, and $n = 4$ neighbours in the spatial model.