The evolution of dispersal in a Levins' type metapopulation model

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Abstract

We study the evolution of the dispersal rate in a metapopulation model with extinction and colonisation dynamics, akin to the model as originally described by Levins. To do so we extend the metapopulation model with a description of the within patch dynamics. By means of a separation of time scales we analytically derive a fitness expression from first principles for this model. The fitness function can be written as an inclusive fitness equation (Hamilton's rule). By recasting this equation in a form that emphasizes the effects of competition we show the effect of the local competition and on the local population size on the evolution of dispersal. We find that the evolution of dispersal cannot be easily interpreted in terms of avoidance of kin competition, but rather that increased dispersal reduces the competitive ability. Our model also yields a testable prediction in term of relatedness and life history parameters.

Key words: population structure, structured deme model, virulence, patch exploitation, relatedness, Hamilton's rule, inclusive fitness.

Left running title: Vincent A.A. Jansen and Renaud Vitalis
Right running title: Evolution of dispersal in metapopulations

Introduction

Natural populations are rarely well-defined entities that are genetically and demographically isolated from other populations with their own, independent dynamics. Rather, the local dynamics of populations are linked to the global dynamics through the movement of individuals among groups. Dispersal is therefore central to our understanding of the ecological and evolutionary processes that are at play in subdivided populations. A typical example of a subdivided population is the *metapopulation*: a collection of local populations that exist in patches of suitable habitat that exhibit extinction-colonisation dynamics (Levins 1969; Hanski 1999).

Dispersal influences both the local, within-patch dynamics and the global dynamics of a metapopulation: locally, dispersal balances the efflux of emigrants and the influx of immigrants, which affects local competition; globally, dispersal allows the colonisation of empty patches. In return, both the local and global effects contribute to the selection process that shapes the evolution of dispersal. Because of these various effects, and the different levels at which they operate, it is often difficult to disentangle the selective forces that drive the evolution of dispersal. Probably for this reason there is a wealth of material on the evolution of dispersal, for a large part of a theoretical nature (see Clobert et al. (2001), Bowler and Benton (2005) for reviews).

The evolutionary causes of dispersal have been classified into three broad categories (Ronce et al. 2001). If there is, firstly, spatial heterogeneity in habitat quality, dispersal enables an individual to leave a certain locality and to arrive at a locality where the conditions are supposedly better, a process referred to as "habitat selection". Yet, if there is spatial heterogeneity in habitat quality and if habitat quality is constant over time dispersal is selected against (Hastings 1983; Parvinen 1999), because it is never optimal to emit offspring to patches of potentially lower quality. If, however, the habitat quality changes over time this holds no longer true.

A second category in the evolution of dispersal therefore involves temporal heterogeneity in habitat quality. There can be a selective advantage to dispersal if habitat quality fluctuates over time. By dispersing, an individual can spread its offspring over space and thus sample a large area. If the fluctuations are not completely correlated over space this sampling will reduce the experienced fluctuations. Such a strategy is often referred to as bet-hedging (Kuno 1981; Venable and Brown 1988; Jansen and Yoshimura 1998). In a metapopulation local catastrophes cause heterogeneity in space and time. An increase in the extinction rate therefore generally leads to selection for higher dispersal rates (but see Ronce et al. 2000, Gyllenberg et al. 2002, Parvinen et al. 2003 and Parvinen, 2006).

The third category relates to local competition: by leaving its native patch an individual can reduce the strength of competition in the patch. Because dispersal often entails a reduction in the basic fitness of an individual and an increase in the fitness of individuals remaining in the patch, dispersal is often seen as an altruistic trait. Dispersal therefore needs to be considered in the light of the theory of kin selection. Central in this theory is Hamilton's rule (Hamilton 1964), which explains that an altruistic trait, i.e. a trait that incurs a fitness cost to the individual displaying it (the *actor*) but provides a fitness gain to an individual benefiting from it (the *recipient*), can evolve if the actor and the recipient are likely to share identical copies of their genes. The probability with which gene copies are shared among individuals is known as *relatedness*.

Following the seminal work of Hamilton and May (1977), the effect of local competition on the evolution of dispersal has often been studied in systems of structured demes. Within a deme, a finite number of individuals produce a large, possibly infinite, number of offspring, a fraction of which disperses. All adults die, and the access of offspring to breeding sites is granted through local competition which is determined through a lottery played among philopatric (produced within the deme) and dispersed offspring (see e.g Hamilton and May 1977; Gandon and Rousset 1999). The results of Hamilton and May (1977) have been generalised in various ways, most notably by Comins et al. (1980). The interpretation of dispersal and the importance of relatedness was highlighted in the work of Frank and Taylor (Frank 1986; Taylor 1988; Taylor and Frank 1996), and further refinements have been added by various others (Gandon and Michalakis 1999; Rousset and Billiard 2000; Rousset 2004).

Whilst the structured-deme model has arguably become the standard setting to discuss the effects of local competition on the evolution of dispersal, the leading formalism to describe the population dynamics of the metapopulation is the Levins' model (Levins 1969; Hastings and Harrison 1994; Hanski 1999). The Levins' model assumes that patches are in one of two states: they are either occupied or empty. Empty patches can get colonised, occupied patches can fall empty through local

catastrophes. The model explicitly allows for the presence of empty patches, as this is considered by many a crucial characteristic of metapopulations.

Various studies have addressed the evolution of dispersal in Levins' type metapopulation models (Heino and Hanski 2001). A formalism to study the evolution of dispersal in metapopulations has been developed (Gyllenberg and Metz 2001; Metz and Gyllenberg 2001; Parvinen 2006), and this formalism has been applied in various forms (Gyllenberg et al. 2002; Cadet et al. 2003; Parvinen et al. 2003). Because of the complexity of these models most of the results come in the form of numerical results. Some analytical results have been derived, yet only for cases where the local population size is infinite, and where the immigrations into local patches are very frequent, and therefore the metapopulation has no empty patches. Although these studies have added greatly to our understanding of the evolution of dispersal, in specific scenarios these results lack generality.

Although the study of the evolution of dispersal in structured-deme and metapopulation models have provided considerable insight in the factors that determine the evolution of dispersal, there is a marked difference between these two approaches. The work on structured-deme models are based on the assumption that the local population size is fixed and does not depend on the dispersal rate (but see Rousset and Ronce 2004). In addition, studies on such models tend to concentrate on the derivation of simple expressions for the marginal fitness, and the interpretation of these expressions in terms of genetic measurable quantities, in particular relatedness. On the other hand, studies of the evolution of dispersal in Levins' type models often include a detailed description of the local dynamics, and normally describe the dependence of local dynamics on dispersal. Unfortunately, this work has so far not led to simple and general insights and mainly yielded numerical results or expressions for fitness that are difficult to interpret in terms of the relatedness structure of a population. The gap between these two approaches is further widened by the fact that population dynamical models, such as the Levins' model, are often formulated in a way that predominantly describes local *densities*, thus making it difficult to tease out the effects of interactions between individuals. We think it is important to bridge this gap in order to formulate a theory that is based on a realistic and widely accepted ecology, such as the Levins' model, and that produces predictions in terms of easily observable quantities, in particular relatedness.

The aim of this paper is to relate the framework of Levins' type metapopulations with structured-demes models. We formulate an analytical model that is a natural extension of the Levins' model, in order to study the evolution of dispersal in metapopulations which contain empty patches. By applying a separation of time scales we describe local competition efficiently and make it analytically tractable. This enables us to derive an expression for fitness that allows interpretation and direct comparison to structured-deme models. Interestingly, we recover Hamilton's rule in this model but with a twist. We hope that our results contribute to bridging the gap that currently exists between population dynamical and genetical approaches in the study of the evolution of dispersal.

Model formulation

We start by describing the Levins' metapopulation model for one species and then extend it so that it accounts for local dynamics. We do this by assuming that local population growth is logistic and that this growth is fast relatively to the lifetime of a patch so that one can effectively assume that the equilibrium is reached instantaneously (see also Gyllenberg and Hanski (1992)). We further extend the model to allow the coexistence of different strains in a patch. If the competing strains only differ marginally in their dispersal rate, then the local competition between the strains is inherently a slow process (see Jansen and Mulder 1999). We then describe the competition between the strains at the metapopulation level by considering the invasion of empty patches and the reinvasion of occupied patches. In this way we can establish which of two competing strains wins the competition at the metapopulation level and we can therefore establish the fitness of a strain with a dispersal rate that is marginally different from the mean dispersal rate in the population. Finally, we interpret the resulting fitness function by studying how its components depend on the model parameters. By analysing this fitness function, both the evolutionary change in dispersal rate and the end-point of this process can be predicted.

The metapopulation model for a single strain

The Levins' metapopulation model describes the dynamics of a single species in a collection of patches that are either empty or occupied: occupied patches become empty through catastrophes that cause local extinction, while empty patches can be colonised from occupied patches. The fraction of occupied patches is denoted P. This changes over time as new colonizers (mP) reach empty patches $(1 - P)$ and as extinctions occur at rate eP. The Levins' metapopulation model therefore reads:

$$\dot{P} = mP(1 - P) - eP \tag{1}$$

where the dot indicates the change of P over time.

The Levins' model does not specify the dynamics within a patch. We will extend the Levins' model to account for local population dynamics. Because local populations consist of individuals, population size is a discrete variable. In what follows we will describe the size of the local population as a continuous deterministic variable. We can do this because although events that change the size of the local population, such as births and deaths, are inherently stochastic and discrete, we can consider the mean population size of an ensemble of populations that all started of from the same initial number. In this way we can describe the local dynamics in terms of the ensemble mean of a stochastic process, which is a continuous variable (see Appendix 1). To describe the change in the ensemble mean of the local population size we will follow Gyllenberg et al. (2002) and Parvinen et al. (2003) and assume that the ensemble mean of the local population size within a patch, x, obeys logistic growth with growth rate r and carrying capacity k. We also assume that individuals leave the patch with a per capita rate of γ. The local population size thus changes over time according to

$$\dot{x} = rx(1 - \frac{x}{k}) - \gamma x. \tag{2}$$

The local population size goes to a positive, stable equilibrium given by $\tilde{x} = k(1 - \frac{\gamma}{r})$ if $\gamma < r$.

If this equilibrium value is reached quickly compared to the lifetime of a patch the local dynamics are fast compared to the dynamics of the metapopulation (Goel and Richter-Dyn 1974; Barbour and Pugliese 2004). Note that this assumption is implicit to the Levins' model: if this were not the case the sizes of the subpopulations at any point in time would all be very different and one could not reasonably assume that occupied patches are all equal. In mathematical terms this assumption amounts to requiring that $r - \gamma \gg e$ (the time to reach equilibrium is proportional to $1/(r-\gamma)$, and the average lifetime of a patch is $1/e$) and ensures that virtually all local populations are at equilibrium. For the model in this paper we will assume that $r - \gamma \gg e$. If migrations into occupied patches are rare we can ignore their effect on the local dynamics (2). However, local immigrations do matter if they occur in an empty patch.

Individuals that leave their patch become dispersers (D). The total number of new dispersers per unit of time is $D(P) = \gamma \tilde{x} P$. We assume that a disperser successfully finds a patch with probability α (hence, the 'cost of dispersal' is $1 - \alpha$), and that the patch is colonised with probability $u(\gamma)$. The probability of colonisation of an empty patch depends on the dispersal rate: for instance, if the birth and death rate are given by, respectively, rx and rx^2/k, then the probability of colonisation is approximately $u(\gamma) = 1 - \gamma/r$ (see Appendix 1). The colonisation rate of empty patches is $\alpha u(\gamma)D(P)(1 - P)$. If we define $m(\gamma) = \gamma \alpha u(\gamma)k(1 - \frac{\gamma}{r})$ the colonisation rate of empty patches can be written as $m(\gamma)P(1 - P)$ and in doing so we have extended the Levins' metapopulation model (1) with a consistent description of the local dynamics, and have made the patch colonisation rate $m(\gamma)$ a function of the dispersal rate.

In the Levins' model only immigrations into empty patches are accounted for: in the limit considered here, $r - \gamma \gg e$, the local dynamics equilibrate instantaneously, hence the acquisition of an extra individual does not affect the local population size in an occupied patch. However, if we wish to retain information about the number of distinct strains that a patch has received we can denote the fraction of patches that have received i immigrants by P_i. The total fraction of occupied patches is given by $P = \sum_{i=1}^{n} P_i$. If we restrict the number of invasions to $n = 2$, (Van Baalen and Sabelis (1995a) use a similar assumption in the context of the evolution of virulence) the dynamics of the P_is are given by:

$$\begin{aligned} \dot{P}_1 &= m(\gamma)P(1 - P) - eP_1 - m(\gamma)/u(\gamma)PP_1 \\ \dot{P}_2 &= m(\gamma)/u(\gamma)PP_1 - eP_2. \end{aligned} \tag{3}$$

Note that because the birth and death rates will differ from the birth and death rates in empty patches, the probability of colonisation of already occupied patches is different from empty patches. In the formalism used here we assume that the loss of individuals through local extinctions in patches that are reinvaded is incorporated in the local dynamics (see Appendix 1) and therefore we need to compensate the reinvasion rate to take this into account. The reinvasion rate, for this reason is divided by the term $u(\gamma)$ used in the colonisation of empty patches and the rate of reinvasion is $m(\gamma)/u(\gamma)PP_1$. System (3) has as equilibrium $\tilde{P} = 1 - e/m(\gamma)$, which is identical to the equilibrium of the Levins' model (1), and the equilibrium values of P_1 and P_2 are given by $\tilde{P}_1 = \frac{m(\gamma)u(\gamma)}{m(\gamma)-e(1-u(\gamma))}\tilde{P}(1 - \tilde{P})$ and $\tilde{P}_2 = \frac{m(\gamma)}{m(\gamma)-e(1-u(\gamma))}\tilde{P}^2$. The restriction to two invasions per patch can be justified by realising that empty patches are a characteristic of a metapopulation. This implies that the colonisation rates are low, and that multiple recurrent invasions in a patch are relatively rare over the lifetime of a patch. In what follows we will outline results for more than two invasions, and show that this assumption does not qualitatively affect our results. Because our model assumes that colonisation rates are low, we will assume throughout this paper that the parameter α is sufficiently small for this to be the case. How small exactly depends on the other parameters.

Dispersal affects the metapopulation in various ways: it decreases the local population size $\tilde{x}$ (Fig. 1a), but, obviously, it also increases the number of individuals leaving a patch and decreases the probability of colonisation. As a result, the colonisation rate $m(\gamma)$ varies non-monotonically with the dispersal rate (Fig. 1b). For $\gamma = r/3$, these effects compensate one another, and the local population sizes yield the maximum number of dispersers. The occupation of the metapopulation also reaches a maximum at $\gamma = r/3$ (Fig. 1c) but since the local equilibrium size decreases with γ, the total number of individuals in the metapopulation $\tilde{P}\tilde{x}$ peaks for a lower value of γ (Fig. 1d).

Local dynamics for two competing strains

Next, we extend the model to describe two strains coexisting in the metapopulation. The strains differ marginally in their dispersal rates and they compete locally for resources. The local population size of the first strain, which has a dispersal rate γ°, is given by x°. In what follows we will refer to this widespread strain as the resident strain in the metapopulation. The local population size of the second strain, which has dispersal rate γ^*, is given by x^*. This strain will further be referred to as the mutant strain. If the two strains locally coexist, we assume the local dynamics within a patch are given by (see Appendix 1):

$$
\begin{aligned}
\dot{x}^\circ &= rx^\circ(1 - \tfrac{x^\circ + x^*}{k}) - \gamma^\circ x^\circ \\
\dot{x}^* &= rx^*(1 - \tfrac{x^\circ + x^*}{k}) - \gamma^* x^*.
\end{aligned}
\tag{4}
$$

By introducing new variables for the total density ($s = x^\circ + x^*$) and the fraction of the mutant strain ($f = x^*/s$), the local dynamics (4) can be rewritten as:

$$
\dot{s} = s\left(r(1 - \tfrac{s}{k}) - \gamma^\circ(1 - f) - \gamma^* f\right) \tag{5}
$$

$$
\dot{f} = (\gamma^\circ - \gamma^*)f(1 - f). \tag{6}
$$

As for the single strain dynamics we will assume that the dynamics of the local population size $\dot{s}$ are fast compared to the metapopulation dynamics and therefore require $r - \gamma^\circ \gg e + \alpha D$ and $r - \gamma^* \gg e + \alpha D$. Under these assumptions s quickly reaches a quasi steady state, denoted $\tilde{s}$, which can be found by putting the left hand side of equation (5) to zero:

$$
\tilde{s}(t) = k\left(1 - \frac{\gamma^\circ(1 - f(t))}{r} - \frac{\gamma^* f(t)}{r}\right) = \tilde{x}^\circ + f(t)(\tilde{x}^* - \tilde{x}^\circ) \tag{7}
$$

where $\tilde{x}^\circ$ (resp. $\tilde{x}^*$) gives the equilibrium population of the resident (resp. mutant) strain when it occupies a patch on its own: $\tilde{x}^\circ = k(1 - \tfrac{\gamma^\circ}{r})$ [resp. $\tilde{x}^* = k(1 - \tfrac{\gamma^*}{r})$]. Note that $\tilde{s}(t)$ changes over time only through $f(t)$.

Equation (6) describes the replacement of one strain by the other through competition. Although both strains share the same carrying capacity, and thus compete which each other for resources, this competition does not lead to replacement. It is the difference in the strains' dispersal rates, which manifests itself through a difference in competitive ability, that causes the replacement of the more
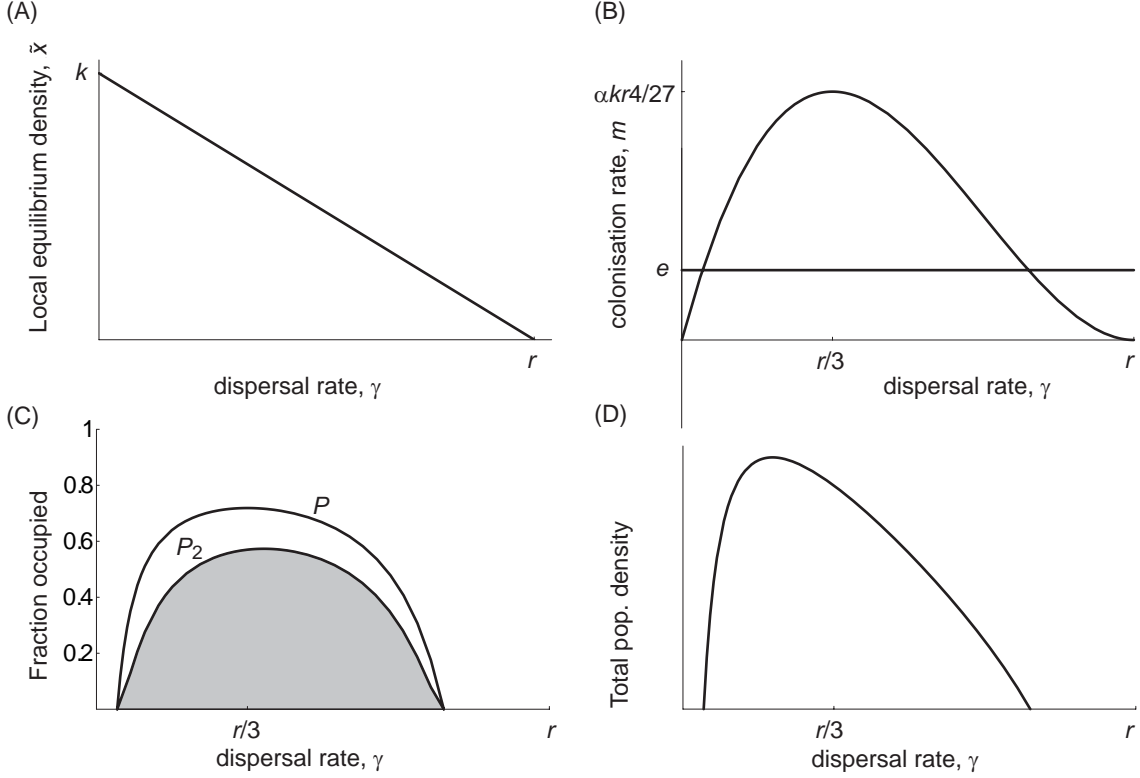
Figure 1: The effect of the dispersal rate, γ on (A) the local equilibrium population size $\tilde{x}$, (B) the colonisation rate, $m(\gamma)$, (C) the equilibrium level of occupation in the metapopulation $\tilde{P}$, and the fraction of patches that received two colonisers, $\tilde{P}_2$, and (D) the total number of individuals in the metapopulation $\tilde{x}\tilde{P}$. The horizontal line in B represents the extinction rate, and the metapopulation is viable if the colonisation rate exceeds the extinction rate. It can be seen that for very high or very low dispersal rates the metapopulation is not viable. Parameters: $\alpha = 0.001$, $r = 25$, $e = 0.1$ and $k = 100$.

dispersing strain by the more philopatric one. If this difference $(\gamma^\circ - \gamma^*)$ is small, then this process is slow – a fact that can be exploited to approximate the local dynamics accurately (Jansen and Mulder 1999). The dynamics of the fraction f is equivalent to that for logistic growth and can be solved in closed form

$$f^\phi(t) = \frac{\phi}{\phi + (1-\phi)\exp\left[(\gamma^* - \gamma^\circ)t\right]} \tag{8}$$

where the superscript refers to the initial condition (i.e., the fraction of the mutant strain at the time of invasion): $\phi = f^\phi(0)$. If the meaning is unambiguous we will suppress the superscripts. Similarly, $\tilde{s}^\phi(t) = \tilde{x}^\circ + f^\phi(t)(\tilde{x}^* - \tilde{x}^\circ)$. If $r - \gamma \gg e$ the approximation of x^* by $f\tilde{s}$ and x° by $(1-f)\tilde{s}$ provides an excellent approximation to the local dynamics, which we will henceforth use to describe the local dynamics (Fig. 2).

The initial conditions relevant for the local dynamics depend on the order in which individuals from different strains arrive in a patch. For example, consider an individual with a dispersal rate γ° that invades a patch previously founded by an individual with dispersal rate γ^*. According to our model this patch will contain on average $\tilde{x}^*$ individuals before invasion and therefore an average of $\tilde{x}^* + 1$ individuals immediately after the second invasion. The fraction of mutant individuals at the time of invasion will therefore be given by $\phi^* = f^{\phi^*}(0) = \frac{\tilde{x}^*}{\tilde{x}^*+1}$. Similarly, a patch founded by an individual with dispersal rate γ° that is reinvaded by an individual with a dispersal rate γ^* will contain a fraction $\phi^\circ = f^{\phi^\circ}(0) = \frac{1}{\tilde{x}^\circ+1}$ of mutant individuals immediately after reinvasion. The approximation of the local dynamics (5) and (6), together with the relevant initial conditions given above, allows us to calculate the fitness of the mutant strain in the metapopulation.
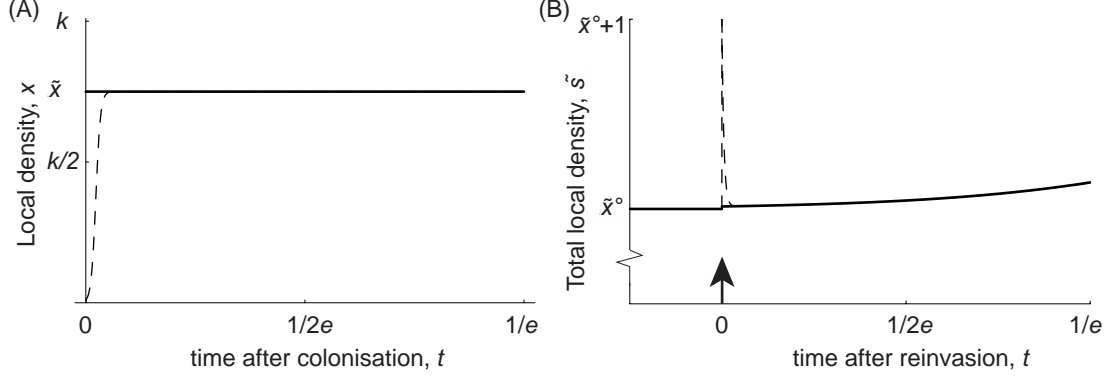
Figure 2: The local dynamics and the approximation of the local dynamics made by the Levin's model. (A) The local dynamics after colonisation of a patch. If the local population growth is logistic (dashed line, given by eqn (2)), the assumption of the Levins' model that the local population size is constant (drawn line) is justified if the growth rate is fast compared to the the lifetime of a patch. (B) The local dynamics after reinvasion of an individual. Here, we consider that at time 0, a single individual invades an occupied patch (vertical arrow). The local population dynamics of the full, non-approximated model (4) are represented by the dashed line. Apart from the initial spike that immediately follows reinvasion (vertical arrow), the dynamics are well approximated by the description of the extended Levins' model (drawn line given by (7) and (8)) which was derived using a separation of time scale argument. The sharp fall back after the initial spike is caused by the fact that the local population size falls back very quickly to its quasi equilibrium following invasion. Parameters as in Fig. 1, $\gamma = r/4$, $\gamma^* = \gamma - 0.25$ and $x(0) = 1$ in A and $x(0) = 75$ in B.

The fitness of a focal individual

Studying the evolutionary dynamics of dispersal in the metapopulation, amounts to characterizing the fitness of an individual that adopts a marginally deviant strategy for dispersal. We shall therefore calculate the expected number of dispersed offspring that a rare mutant that disperses at rate γ^* will give rise to in a population dominated by a resident strain that disperses at rate γ°. This is related to the computation of the invasion rate as a measure for fitness (Metz et al. 1992) as extended to metapopulations (Metz and Gyllenberg 2001; Gyllenberg and Metz 2001) and largely equivalent to the focal individual fitness approach pioneered by Taylor and Frank (1996) (see also Ajar 2003).

We will assume that the density for the resident population at large is given by the equilibrium of the Levins' model (3). To calculate the fitness, we follow the different fates that a focal individual may meet:

(i) The focal individual lands and successfully colonises an empty patch. This happens with probability $\alpha u(\gamma^*)(1 - \tilde{P})$. Following invasion, this patch either is hit by a catastrophe or is reinvaded. The average time elapsed before one of these events occurs is $1/(e + \alpha\tilde{D})$, during which time $\gamma^*\tilde{x}^*\alpha u(\gamma^*)\frac{1-\tilde{P}}{e+\alpha\tilde{D}} = \gamma^*\tilde{x}^*\frac{u(\gamma^*)\tilde{P}_1}{u(\gamma^\circ)\tilde{D}}$ mutants leave this patch. If the patch goes extinct nothing else will happen. However, if the patch is reinvaded, which happens with probability $\alpha\tilde{D}/(e + \alpha\tilde{D}) = \frac{m(\gamma^\circ)}{m(\gamma^\circ)-e(1-u(\gamma^\circ))}\tilde{P}$, more mutants will still be dispersed. The total number of mutants dispersed from such a patch is $\gamma^*\alpha u(\gamma^*)\frac{\alpha\tilde{D}(1-\tilde{P})}{e+\alpha\tilde{D}}\int_0^\infty f^{\phi^*}(t)\tilde{s}^{\phi^*}(t)\exp(-et)\mathrm{d}t = \gamma^*\alpha\frac{u(\gamma^*)\tilde{P}_1}{u(\gamma^\circ)}\int_0^\infty f^{\phi^*}(t)\tilde{s}^{\phi^*}(t)\exp(-et)\mathrm{d}t$, where $\exp(-et)$ is the probability that a patch has not gone extinct t time units after reinvasion. Since the density of the mutant strain is very small in the metapopulation, we need not consider the successive invasion of two mutant individuals in the same patch.

(ii) The focal individual lands and reinvades a patch already occupied. This happens with probability $\alpha\tilde{P}_1$. In that case, $\gamma^*\alpha\tilde{P}_1\int_0^\infty f^{\phi^\circ}(t)\tilde{s}^{\phi^\circ}(t)\exp(-et)\mathrm{d}t$ mutant dispersers are sent out.

Putting all this together yields the fitness of a rare mutant strain with dispersal rate γ^* in a metapopulation at equilibrium in which the dispersal rate is γ°:

$$W(\gamma^*, \gamma^\circ) = \gamma^*\alpha\tilde{P}_1\left[\frac{\tilde{x}^*u(\gamma^*)}{\alpha u(\gamma^\circ)\tilde{D}} + \int_0^\infty\left[f^{\phi^\circ}(t)\tilde{s}^{\phi^\circ}(t) + \frac{u(\gamma^*)}{u(\gamma^\circ)}f^{\phi^*}(t)\tilde{s}^{\phi^*}(t)\right]\exp(-et)\mathrm{d}t\right]$$

7

It is straightforward to check that $W(\gamma^\circ, \gamma^\circ) = 1$, i.e. that the fitness of a focal mutant that disperses at the same rate as the rest of the population is unity. The fitness expression can be rewritten using special functions, but this does not lead to transparent results.

Marginal fitness

In order to identify the possible endpoints of the evolutionary process, we calculate the marginal fitness, or fitness gradient (a change in fitness due to a marginal change in dispersal rate) as:

$$\left.\frac{\partial W(\gamma^*, \gamma^\circ)}{\partial \gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} = \frac{1}{\gamma} + \frac{\mathrm{d}\tilde{x}}{\tilde{x}\mathrm{d}\gamma}\frac{\tilde{P}_1 + \tilde{P}_2\left(\phi^{\circ 2} + \phi^{*2}\right)}{\tilde{P}} - \frac{1}{e}\frac{2\phi^\circ\phi^*\tilde{P}_2}{\tilde{P}} + \frac{\mathrm{d}u}{u\mathrm{d}\gamma}\left(\frac{\tilde{P}_1}{\tilde{P}} + \frac{\tilde{P}_2}{\tilde{P}}\phi^*\right) + \left.\frac{\tilde{P}_2}{\tilde{P}}\frac{\mathrm{d}\phi^*}{\mathrm{d}\gamma^*}\right|_{\gamma^*=\gamma}$$
(9)

(see Appendix 2 for derivation.) It is straightforward to numerically calculate the values of γ for which the marginal fitness is zero, and thus establish the Evolutionarily Singular (ES) dispersal rates. (We have avoided the word stability here because the analysis does not provide information about the evolutionary stability of the ES values. However, in all cases where we verified the stability the ES values were evolutionary and convergence stable.) Such an analysis shows:

- *The ES dispersal rate increases with the local growth rate r* (Fig. 3a). This is so mainly because strains that have a higher growth rate can afford to emit more dispersers, and in agreement with the results in Parvinen (2006).

- *The ES dispersal rate decreases with the probability of finding a patch α* (Fig. 3b). This is so because our approximation is valid mainly if the probability of finding a patch is low, and this will be a main determinant of the occupancy of the metapopulation. An increase in colonisation will decrease relatedness (Gandon and Michalakis 1999; Kisdi 2004) and put a premium on competition, leading to reduced dispersal. This contradicts Gyllenberg et al. (2002), Parvinen et al. (2003) and Parvinen (2006) but in these studies the cost of dispersal was always fairly low and not in a regime which compares to our model. Comins et al. (1980), Gandon and Michalakis (1999) find that the ES dispersal probability generally decreases with the cost of dispersal 1-α, but, as in our model, they find an increase in the ES dispersal rate for high costs and for small populations.

- *The ES dispersal rate predominantly decreases with the local carrying capacity* (Fig. 3c). For low carrying capacities the ES dispersal rate decreases as the carrying capacity increases, mainly because larger patches lead to an increase in the total number of dispersers, which leads to a reduced dispersal rate. For large carrying capacities the ES dispersal rate increases with the carrying capacity. This increase is in marked difference with structured-deme models where the dispersal rate generally decreases with the local population size (Comins et al. 1980, Gandon and Michalakis 1999). However, for the region in which the increase takes place the rate of invasion in a patch is relatively large compared to the local population growth, so that the time scale separation we applied is at the limit of its validity.

- *The ES dispersal rate increases with the extinction rate e* (Fig. 3d). The rationale is that extinctions increase the fraction of empty patches, hence the opportunity for successful colonisations. This result is in contrast with the findings of Ronce et al. (2000), Gyllenberg et al. (2006), Parvinen et al. (2003) and Parvinen (2006) who found a non-monotonic relationship. This discrepancy is most likely to be caused by the fact that at very high extinction rates the average lifetime of a patch becomes short, and the separation of time scales we have applied is not valid. At high extinction rates the local population dynamics spend a relatively long time in a phase of exponential growth, and a r-selection scenario ensues. A reduced dispersal rate boosts local growth and thus explains the evolution of smaller dispersal rates.

Although expression (9) is useful in obtaining numerical results, it is of limited assistance in understanding the biological processes that drive the evolution of dispersal in a metapopulation, which is the main motivation of this work. However, this equation can be interpreted further, by noticing that some terms are functions of the *relatedness* among mutant individuals, which measures the probability of finding two identical genes in two individuals from the same patch, relative to the
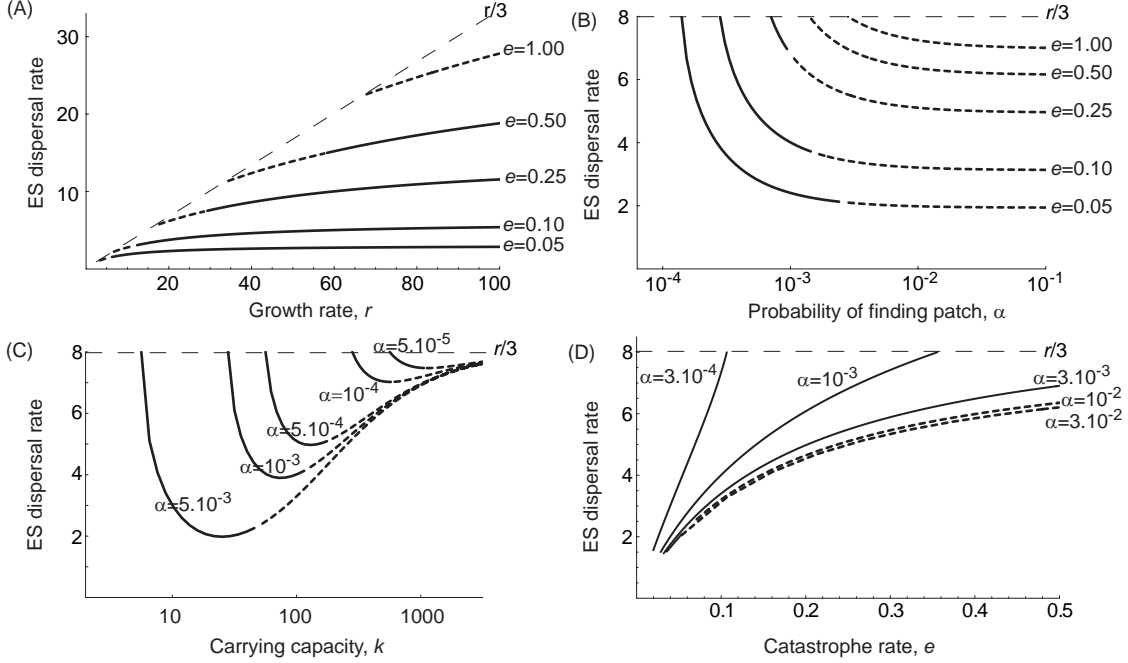
Figure 3: The ES dispersal rate as a function of (A) the local growth rate r, (B) the probability of finding a patch α, (C) the extinction rate e, and (D) the carrying capacity k. The long dashed line gives the boundary of the metapopulation viability area. This is the viability limit, because $\gamma = r/3$ implies $R = 1$, which in turn implies $\tilde{P}_2 = \tilde{P}^2 = 0$ Results are derived by setting the lhs of (9) to zero and numerically solving for γ. For the drawn curves $r - \gamma > 10(e + \alpha D)$ while for the dashed curves this was not the case. Unless otherwise indicated parameters as in Fig. 1.

probability of finding two identical genes in two individuals from the same metapopulation (Pamilo 1984; Queller and Goodnight 1989). In Appendix 3 we show that relatedness in the metapopulation is given by

$$R = \frac{\tilde{P}_1 + \tilde{P}_2 \left(\phi^{\circ 2} + \phi^{*2} \right)}{\tilde{P}}.$$

We also show that this is identical to the chance that a rare mutant encounters another mutant in its patch, which has also been proposed as a relatedness measure (Day and Taylor 1998; Van Baalen and Rand 1998; Ferriere and Le Galliard 2000). In a similar fashion we can define the probability that a rare mutant encounters a resident individual. This probability, which one could call unrelatedness, is simply $1 - R$; we will denote it as $\bar{R}$ and it is given by

$$\bar{R} = \frac{2\phi^{\circ} \phi^{*} \tilde{P}_2}{\tilde{P}}.$$

Using this we can rewrite the marginal change in fitness (9) in a much clearer form as:

$$
\begin{aligned}
\left. \frac{\partial W(\gamma^*, \gamma)}{\partial \gamma^*} \right|_{\gamma^*=\gamma^{\circ}=\gamma} &= \frac{1}{\gamma} + \frac{\mathrm{d}\tilde{x}}{\tilde{x}\mathrm{d}\gamma} R - \frac{1}{e}\bar{R} + \frac{\mathrm{d}u}{u\mathrm{d}\gamma} \frac{\tilde{P}_1 + \phi^* \tilde{P}_2}{\tilde{P}} + \frac{\tilde{P}_2}{\tilde{P}(1+\tilde{x})^2} \frac{\mathrm{d}\tilde{x}}{\mathrm{d}\gamma} \\
&= \frac{1}{\gamma} - \frac{1}{r-\gamma} R - \frac{1}{e}\bar{R} - \frac{1}{r-\gamma} \frac{\tilde{P}_1 + \phi^* \tilde{P}_2}{\tilde{P}} - \frac{\tilde{P}_2}{r-\gamma} \frac{\tilde{x}}{\tilde{P}(1+\tilde{x})^2}. \quad (10)
\end{aligned}
$$

The first term of the above equation represents the fitness change due to a change in the emission of dispersers. This is a direct effect, which relates to the increased fitness of a strain sending out more dispersers. This effect is stronger for low dispersal rates, because as dispersal increases the fraction of empty patches decreases.

The second term represents the effect of a change in the dispersal rate on the local equilibrium population: an increase in the dispersal rate of the focal mutant decreases the local population size,

9

which diminishes the potential for other mutants in the patch to send out dispersers. This is an indirect effect, since it only affects the focal mutant through an effect on related individuals in the patch. It is therefore multiplied with the factor R, which measures the fraction of mutants in a patch conditional to the focal individual being a mutant.

The third term gives the fitness reduction due to a decreased competitive ability within a patch: a strain that emits more dispersers will slowly lose a jointly occupied patch as described by (6). This means that as the focal mutant strain sends out more dispersers, the other strain that competes in the patch benefits from a competitive advantage. Hence, this term is multiplied by the probability of encountering an unrelated individual, $\bar{R} = 1 - R$. This effect is proportional to the lifetime of the patch, which is on average $1/e$.

The fourth term describes the effect of demographic stochasticity. In our model we have incorporated this through the probability $u(\gamma)$ to lose a patch through demographic extinction of a newly colonised patch. Because this probability is only applicable to patches which are colonised from an empty state, this only applies to patches in which the mutant is the first invader. Note that if the local population sizes are moderately large, end hence ϕ^* is close to 1, this term will take the approximate value of $-1/(r-\gamma)$. In what follows, and for reasons of convenience and clarity we will therefore use this approximate value.

The fifth term is somewhat unexpected: it represents the decrease in the fraction of mutants in the patch after a resident individual invades, $d\phi^*/d\gamma^*$. Because a decrease in the dispersal rate boosts the local population size, patches of individuals with a low dispersal rate are less affected by the invasion of a different type. This control is asymmetrical: by decreasing its dispersal rate the mutant can effectively reduce the initial fraction of reinvading residents in patches founded by mutants; but if the mutant reinvades a resident patch, a change in mutant dispersal rate will not affect the mutant fraction. If the local populations are small this effect might be sizable, but for larger population sizes the effect diminishes (see also Appendix 4). Moreover, if the local population dynamics are fast, which implies $r - \gamma \gg e$ the contribution of this term becomes negligible. For clarity, because its presence does not influence our results qualitatively, and because for most realistic metapopulations we expect the magnitude of this finite size effect to be small, we will not take it into account any further.

The outcome of the selection process involving direct fitness effects (i.e., that result from personal reproduction) and indirect fitness effects (i.e., that result from the reproduction of relatives) may be best understood in light of Hamilton's rule (Hamilton 1964) which predicts that the altruistic behaviour of an actor to a recipient individual shall be favoured if $bR > c$, where b is the benefit received by the recipient, c is the cost incurred to the actor, and R is a measure of the relatedness between the two. After rewriting (10) without the fifth term in the form of an inclusive fitness equation gives:

$$\left.\frac{\partial W(\gamma^*, \gamma^\circ)}{\partial \gamma^*}\right|_{\gamma^* = \gamma^\circ = \gamma} \approx \frac{1}{\gamma} - \frac{1}{r - \gamma} - \frac{1}{e} + \left(\frac{1}{e} - \frac{1}{(r - \gamma)}\right) R. \tag{11}$$

Note that this fitness function is not constructed in analogy with Hamilton's rule, but is derived from first principles. From this recast equation (which we also derived for unlimited numbers of invaders per patch, see Appendix 4) we can see that the direct fitness effect of a change in the dispersal rate can now be interpreted as the combination of the direct benefit resulting from an increased emission of dispersers (term 1) and the effect of demographic stochasticity (term 2), together with the cost of competition (the third term in (10)) which is levied over all individuals (term 3). However, because competition only affects the fitness if it operates between unrelated individuals, we need to compensate for the competition between related individuals that we now have invoked. In (11) this is accounted for by adding an indirect benefit through the interactions with related individuals. Although this leads to a representation which is compatible with the commonly held view that the avoidance of kin competition is a main selective force in the evolution of dispersal, this also suggests that this interpretation does not follow naturally from the competitive process.

The indirect fitness component (the term multiplied with R in (11)) is always positive because the Levins' model requires $r - \gamma \gg e$. The direct fitness component can be positive if many patches are unoccupied. A small increase in the dispersal rate can therefore give an enormous advantage that outweighs the cost of local competition. In that situation dispersal would not constitute an act of altruism, as this requires that the direct effect is a cost (i.e. is negative), whilst the indirect effect, which here only involves the interactions with similar individuals, is positive. At and close to the ES dispersal rate the marginal gain in fitness is zero or small. Because the indirect effect in (11) is

positive the direct effect has to be negative. Dispersal in a metapopulation in such cases can indeed be seen as an altruistic behaviour.

Because competitive replacement is only brought about by individuals with a dispersal rate that differs from that of the focal individual, replacement competition only occurs among unrelated individuals. To reveal this effect of competition we recast (10) without the fifth term in a form in which the effects with unrelated individuals are exposed:

$$\left.\frac{\partial W(\gamma^*, \gamma^\circ)}{\partial \gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} \approx \frac{1}{\gamma} - \frac{2}{r-\gamma} - \left(\frac{1}{e} - \frac{1}{r-\gamma}\right)\bar{R}. \qquad (12)$$

The first and second terms, that do not have the factor $\bar{R}$, represent the marginal fitness of a mutant that would result if different strains cannot jointly occupy a patch. Such a situation obtains if we would consider single colonisations only, in which case the fitness is given by $\frac{m(\gamma^*)}{m(\gamma)}$. The marginal gain in fitness reads $\frac{1}{m(\gamma)}\frac{dm(\gamma^*)}{d\gamma^*} = \frac{1}{\gamma} + \frac{d\tilde{x}}{\tilde{x}d\gamma} + \frac{du}{ud\gamma}$ and these terms therefore represent the increase in fitness due to the change in the colonisation rate if local interactions occur exclusively among related individuals. One could thus interpret the fitness components given by the first and second terms as representing the importance of the common good (Van Baalen and Jansen 2001).

In the extreme situation when the common good is the only driving force (i.e. if $R = 1$), the evolutionary singular dispersal rate would be $\gamma = r/3$. The effect of dispersal on the local population size $(-(r-\gamma)^{-1})$ is always negative: increased dispersal does not benefit related individuals that remain in the patch (see Discussion). Interestingly, the term $-\frac{d\tilde{x}}{\tilde{x}d\gamma}$ can be shown to be proportional to the characteristic return time of the local equilibrium (independently of the actual growth rate function that is used): the fitness depends on the stability of the local population equilibrium (Zeineddine and Jansen 2005).

The third term, multiplied with the 'unrelatedness' $\bar{R}$, comprises the effects of competition with unrelated individuals. Because $\bar{R}$ is a probability it is always positive. Furthermore, as we required for a Levins' type metapopulation that $r - \gamma \gg e$, the combined effect of competition is always negative. It follows that at an ES dispersal rate the term $\frac{1}{\gamma} - \frac{2}{r-\gamma}$ must be positive and thus that the evolutionary singular dispersal rate cannot exceed $r/3$. The local population size $\tilde{x}$ and the global population size $\tilde{x}\tilde{P}$ tend to be larger than in a metapopulation where the joint occupancy of patches is not possible (see Figs. 1a and 1d).

Putting the right hand side of equation (12) to zero, we find that the at candidate evolutionary endpoints the unrelatedness approximately obeys the equation

$$\bar{R} \approx \frac{\gamma^{-1} - 2(r-\gamma)^{-1}}{e^{-1}}$$

(this only holds approximately because we have used $r - \gamma \gg e$ to simplify the denominator and ignored the fifth term in (10).) This observation leads to a testable prediction. *In a metapopulation in which the dispersal rate is at its evolutionary endpoint the unrelatedness approximately equals the reciprocal of the dispersal rate minus twice the characteristic return time of the local population divided by the average lifetime of a patch.* All these factors are, in principle, measurable. The dispersal rate can be inferred from the probability that an individual disperses, the characteristic return time can be inferred from the dynamics of newly colonised patches or through experimental manipulation by removing individuals. The unrelatedness, $\bar{R}$, is one minus the relatedness, which can be estimated using standard methods (Queller and Goodnight 1989). Note that this relation predicts that the unrelatedness decreases, and hence the relatedness increases, with the ES dispersal rate (c.f. Gandon & Michalakis 1999).

Discussion

We have analysed the evolution of dispersal in a Levins' metapopulation model, which was extended to account for local population dynamics. We derived a fitness equation from this model and interpreted this expression in various ways. We found that the evolution of dispersal follows Hamilton's rule, i.e. there is a direct effect of a change in the dispersal rate, and an indirect effect which results from the interactions with related individuals. This shows that dispersal indeed can be seen as an altruistic

trait (see Ronce et al. 2001, Rousset 2004). We also showed that increasing dispersal is beneficial because it augments the chance to colonise empty patches. Yet, it also reduces the competitive ability within a patch as well as the local population size.

The evolution of dispersal has been studied in considerable detail (e.g. Frank 1986; Taylor 1988; Taylor and Frank 1996; Gandon and Michalakis 1999; Rousset and Billiard 2000, Rousset 2004). The predominant theoretical approach, a structured-deme model (Hamilton and May 1977; Comins et al. 1980) assumes that within a deme the local population reaches a constant equilibrium size each generation. This assumption is mainly motivated by analytical tractability (Comins et al. 1980). Instead, in order to reproduce the ecological settings of a metapopulation, we based our model on the Levins' metapopulation (Levins 1969, Hanski 1999) to describe the extinction-colonisation dynamics, and we used a logistic growth function for the local dynamics. Our model relates to various other studies of the evolution of dispersal in metapopulations (Ronce et al. 2000; Heino and Hanski 2001; Metz and Gyllenberg 2001; Gyllenberg and Metz 2001; Gyllenberg et al. 2002; Parvinen et al. 2003; Cadet et al. 2003; Parvinen 2006). Our work differs from many previous studies in that we require the colonisation rates to be small, so that at all times the metapopulations contains a relatively large proportion of empty patches, which can remain empty for relatively large periods of time.

Our work extends findings on the evolution of dispersal in metapopulations by deriving analytical expressions, and an interpretation in terms of inclusive fitness. We have achieved this by using a separation of time scales argument, which makes the fitness expressions tractable. This technique is generally applicable to the study of evolution in subdivided populations (Jansen and Mulder 1999), and offers the potential to answer hitherto unanswered questions about patch or host exploitation strategy (e.g. Van Baalen and Sabelis 1995b). We have derived our results by limiting the number of invasions per patch to two, but demonstrate that this has no major impact on our findings. We have made two major assumptions: that the metapopulation has empty patches and that the local dynamics are fast and at quasi steady state. The numerical results we have obtained are broadly in agreement with previous theoretical work on models based on structured demes and metapopulation models, albeit that we did found some departures from previous results if our assumptions became violated. Our technique is also applicable to the study of the evolution of virulence in the face of multiple recurrent infections. To deal with such cases most models make the assumption that a second invader in a patch takes over immediately, or not at all. Although this is mathematically convenient, it leaves the aspect of competition between strains and the effect of relatedness unstudied. The approach used here would apply to study this in much more detail.

We formulated a simple but realistic ecological model, and calculated the selective forces working on the evolution of dispersal. By casting the fitness equation in a form which emphasizes the competition with unrelated individuals, we reached a testable prediction: in a metapopulation in which the dispersal rate is at an evolutionary endpoint the unrelatedness (i.e one minus the coefficient of relatedness sensu Pamilo (1984) and Queller and Goodnight (1989)) approximately equals the reciprocal of the dispersal rate minus twice the characteristic return time of the local population, divided by the average lifetime of a patch. This prediction can assist in determining whether real populations are closer to the metapopulation model given here, or the structured deme model, for which the relatedness is also predicted (Frank 1986; Gandon and Rousset 1999; Gandon and Michalakis 1999). The crucial characteristic of the metapopulations in our model is that local populations are founded by a single foundress, that colonises the patch and fills it with her offspring. Although such a patch can later be reinvaded, the larger the local population size, the less impact reinvasions have and hence the relatedness increases with carrying capacity. Similarly, an increase in the cost of dispersal will make reinvasion less likely, and this leads to the evolution of higher dispersal rates. The assumption of a single foundress and high relatedness is supported by data which show that relatedness in metapopulations often is high (Ingvarsson 1998; Gerlach and Hoeck 2001; Stow et al. 2001; Verdade et al. 2002)

The main result we have obtained is that evolution of dispersal in a Levin's metapopulation can be shown analytically to follow an inclusive fitness equation. The interpretation of this equation allowed us to identify four selective forces that regulate the evolution of dispersal. Increased dispersal leads to (1) an increased ability to colonise new habitats (2) a decreased competitive ability (3) a reduction in the local population size (4) increased demographic stochasticity. As long as the average life time of a patch is large compared to the local population dynamics, these conclusions are likely to be robust. Note that the size of the local population is an important factor for the evolution of dispersal. Restrictive assumptions on the local growth rates, like assuming a fixed number of individuals per

patch, leave out a host of factors in the evolution of dispersal that are likely to be of importance in natural situations (West et al. 2002).

The *avoidance of kin competition* (i.e., local competition with related individuals) (Hamilton and May 1977; Frank 1986; Gandon and Michalakis 1999; Gandon 1999; Perrin and Mazalov 2000) is often invoked as an important factor that should select for dispersal. Our analysis suggests that this interpretation is somewhat contrived. Local competition for resources, in our model as in a structured deme model, is assumed to be fair and hence all individuals have the same chance to succeed, irrespective of their dispersal rates. The empty place which the disperser leaves behind will soon by filled by a new individual, which competes in the same fashion. It is hard then to imagine how arranging competition with unrelated genotypes (as it is suggested by the avoidance-of-kin-competition argument) leads to a selective advantage, except maybe if deleterious effects of inbreeding are taken into account (which is neither the case here, nor in most deme-structured models (but see Morgan 2002; Roze and Rousset 2005)). This is further supported by the observation that a change in the carrying capacity, which will also change the strength of local competition for resources, has no direct impact on the fitness components, apart from a change in the relatedness of the population. However, increased dispersal is detrimental to competitive ability because less offspring are retained in the patch, giving a less dispersing type a competitive advantage. We argue that the competition with unrelated individuals provides a conceptually simpler explanation for the evolution of dispersal.

Acknowledgements

Appendix 1: Justification of the model

A series of models have been formulated to extend the metapopulation model beyond the Levins model. Some of these models are of a deterministic nature, others are fully stochastic. The latter category has recently been used to generate numerical results on the evolution of dispersal (Gyllenberg et al. 2002, Parvinen et al. 2003). Although the main motivation for this model is to demonstrate the conceptual relation between evolution in a metapopulation and structured deme models, rather than to give a approximation of fully stochastic metapopulation models, we will in this appendix outline a possible relation between the model used here, and the fully stochastic metapopulation model. We should stress though, that rather than to provide a precise description of the dynamics of and evolution in a metapopulation our aim here is to provide the conceptual link between classes of metapopulation model. For this reason, this appendix outlines a possible justification of the model. If one is willing to accept that the description of the local dynamics (2) is a reasonable description, this appendix is redundant.

The fully stochastic metapopulation has a comparable structure to the model given here apart from the within patch dynamics, which is formulated as a stochastic birth-death model. The general case is presented in Metz and Gyllenberg (2001), here, we will discuss the formalism as used in Parvinen et al. (2003) and outline how it relates to our model.

Consider a single patch in which the local dynamics are given by a birth-death process. Let the probability that the patch has n° individuals with dispersal rate γ°, and n^* individuals with dispersal rate γ^* be given by P_n where n is the row vector $(n^\circ, n^*)^T$. The master equation for $n^\circ, n^* \geq 0$ of this process is given by:

$$\dot{P}_n = \alpha(D^\circ P_{n-e_1} + D^* P_{n-e_2}) + \sum_{i=1,2} b^i_{n-e_i} P_{n-e_i}$$

$$-(\sum_{i=1,2} b^i_n + d^i_n + \alpha(D^\circ + D^*) + \gamma^* n^* + \gamma^\circ n^\circ)P_n$$

$$+\gamma^\circ(n^\circ + 1)P_{n+e_1} + \gamma^*(n^* + 1)P_{n+e_2} + \sum_{i=1,2} d^i_{n+e_i} P_{n+e_i}$$

where e_i is a row vector with elements 0, apart from position i where it has a 1. If n° or $n^* < 0$ then $P_n = 0$.

Invasions into the patch and catastrophes, occur with rate $\alpha(D^\circ + D^*) + e$. The average waiting time between such events is $(\alpha(D^\circ + D^*) + e)^{-1}$. We can describe the process between invasions and catastrophes, and the changes that happen at invasions or catastrophes. If neither of these events happen the process obeys the master equation

$$\dot{P}_n = \sum_{i=1,2} b^i_{n-e_i} P_{n-e_i} - (\sum_{i=1,2} b^i_n + d^i_n + \gamma^* n^* + \gamma^\circ n^\circ)P_n$$

$$+\gamma^\circ(n^\circ + 1)P_{n+e_1} + \gamma^*(n^* + 1)P_{n+e_2} + \sum_{i=1,2} d^i_{n+e_i} P_{n+e_i}$$

Assume that at time T an invasion into this patch takes place of an individual with dispersal rate γ° (or γ^*). The effect of the invasion is that the local population will increase with the vector e_1 (or e_2). Then if $P_n(T^-)$ is the probability of the process being in state n just before the invasion, and $P_n(T^+)$ the probability to be in state n just after invasion we can calculate the probabilities just after invasion as:

$$P_{n+e_i}(T^+) = P_n(T^-). \tag{13}$$

If at time T a catastrophe takes place we have

$$P_n(T^+) = 0 \quad n > 0$$
$$P_0(T^+) = 1. \tag{14}$$

By averaging over all events (invasions and catastrophes) and all permutations in which these events occur we can find the probabilities P_n without loss of generality. Note that in doing so, we do not need to incorporate the effect of invasions on the local dynamics, as these will not take place in the time interval considered.

We are interested in the number of dispersers that a patch produces on average. We therefore study the average number of dispersers a patch typically produces. To do so we define the ensemble mean between invasions as $x = (x^\circ, x^*)^T = \sum_n nP_n$ this changes over time as

$$
\begin{aligned}
\dot{x} &= \sum_{i=1,2}\sum_n nb^i_{n-e_i}P_{n-e_i} - \sum_n n\left(\sum_{i=1,2} b^i_n + d^i_n + \gamma^* n^* + \gamma^\circ n^\circ\right)P_n \\
&\quad + \sum_n n\gamma^\circ(n^\circ + 1)P_{n+e_1} + \sum_n n\gamma^*(n^* + 1)P_{n+e_2} + \sum_{i=1,2}\sum_n nd^i_{n+e_i}P_{n+e_i} \\
&= \sum_n \sum_{i=1,2} e_i(b^i_n - d^i_n)P_n - e_1\gamma^\circ n^\circ P_n - e_2\gamma^* n^* P_n \\
&= \sum_n \begin{pmatrix} b^1_n - d^1_n - \gamma^\circ n^\circ \\ b^2_n - d^2_n - \gamma^* n^* \end{pmatrix} P_n.
\end{aligned}
$$

If an invasion occurs at time T we find

$$
x(T^+) = \sum_n nP_n(T^+) = \sum_n nP_{n-e_i}(T^-) = x(T^-) + e_i. \tag{15}
$$

(Note that if invasion is by small groups or colonies, (e.g. fruits, or clusters of seeds for plants or droplets of infectious agents for airborne pathogens) one would have to add a vector that gives the average number in such a group, where one would have to be careful to take the relatedness in the invading group into account). If we start our observation at the time of the first invasion we have $x(0) = e_i$. At this point, it is important to note that, firstly, up to this point we have made no assumptions about the nature of the process, other than that is determined by birth, death, invasions and catastrophes. Secondly, through the ensemble mean we have found a description of the local dynamics in a continuous variable, even though in the population size can only take integer values in the stochastic process on which the description is based.

If the birth and death rates b^i_n and d^i_n are linear in n then the ensemble mean is an autonomous system, which we can solve in closed form. If this is not the case we can only solve the ensemble mean if the higher moments of the ensemble are known. Although it is straightforward to derive the equations for the higher moments, to find the ensemble in closed form we need to solve an infinitely dimensional system of differential equations.

We will illustrate for the specific model as used in Parvinen et al. (2003) that the formalism used in the text is a reasonable approximation of the ensemble mean. For this model the birth rates are $b^1_n = rn^\circ$, $b^2_n = rn^*$ and the death rates are $d^1_n = rn^\circ(n^\circ + n^*)/k$, $d^2_n = rn^*(n^\circ + n^*)/k$. In this case the ensemble mean changes as

$$
\dot{x} = \left(rI - \begin{bmatrix} \gamma^\circ & 0 \\ 0 & \gamma^* \end{bmatrix}\right)x - \frac{r}{k}(c + xx^T)(e_1 + e_2), \tag{16}
$$

where c is the covariance matrix which is defined as

$$
c = \sum_n (n - x)(n - x)^T P_n. \tag{17}
$$

We will not attempt to discuss the behaviour of the ensemble mean in general, as the covariance matrix will depend on the initial conditions and the higher moments. We will discuss two scenarios that are relevant within the context of this paper.

The first scenario that we will discuss in somewhat more detail is that of an invasion in an empty patch. In this case there is only one strain present in the patch and we can study the simplified system:

$$
\dot{y} = r\left(1 - \frac{y}{k}\right) - \gamma y + \frac{r}{k}\text{var }y \tag{18}
$$

with $y(0) = 1$, where y represents the number of individuals in the patch and γ their migration rate. The behaviour of the stochastic variant of the logistic equation is well known (see e.g Goel and Richter-Dyn 1974; Barbour and Pugliese 2004): following the invasion of an individual in the patch the numbers will fluctuate in a stochastic fashion. During this initial phase the patch either will go

extinct again, or the numbers will increase and the population will become so large that stochastic extinction becomes highly improbable. The probability of the population to reach a certain size N before extinction decreases with N for small N, but then plateaus and, if the birth rate at small population sizes exceeds the death rate, for relatively small values of N approaches a constant value. Therefore there exists a critical size N, and once this size is reached the probability of the patch to go extinct through demographic stochasticity is very small (Goel and Richter-Dyn 1974). The probability to reach this critical size is

$$u(\gamma) = \frac{1}{1 + \sum_{j=1}^{N-1} \prod_{x=1}^{j} (x/k + \gamma/r)} \tag{19}$$

Although it is possible to evaluate this expression using special functions, we will approximate this expression. If k is large we find that

$$u(\gamma) \approx \frac{1}{1 + \sum_{j=1}^{N-1} \prod_{x=1}^{j} \gamma/r} = \frac{1 - \gamma/r}{1 - (\gamma/r)^N} \tag{20}$$

Since $\gamma/r < 1/2$, even for small N this goes to

$$u(\gamma) \approx 1 - \frac{\gamma}{r}. \tag{21}$$

(Note that this result depends on the choices made for the birth and death rates in the stochastic model. In general, this probability is, for large k, approximately $1 - (\gamma - d)/b$, where b (d) is the birth (death) rate for small numbers). If a local population reaches the critical size, the local population increases and the local population size goes to a stochastic long term quasi equilibrium (this is a quasi-equilibrium because in the very long run extinction is still a certainty). The distribution of the population sizes in this quasi equilibrium is narrow, and takes the form of a shifted Poisson distribution (Barbour and Pugliese 2004). For largish k the mean value of this equilibrium is approximately $k(1 - \gamma/r)$ and the ensemble mean approaches its equilibrium in a logistic fashion.

Putting all this together it follows that the ensemble mean is well approximated by $u(\gamma)x$ where x is a solution of

$$\dot{x} = rx(1 - x/k) - \gamma x. \tag{22}$$

This was confirmed by simulations of the stochastic process describing the within patch dynamics. Only in the first moments after invasion this approximation could be improved because this assumes that extinctions occur immediately after invasion. Because we assume in the main text that the time to reach equilibrium is short compared to the life time of a patch we will not further consider this aspect. Simulation results also confirmed that if a patch that is at quasi equilibrium is further invaded by an individual of the same strain, the logistic equation describes the dynamics well. By describing the local dynamics only by the deterministic change in the ensemble mean the variance in the population sizes is no longer taken into account. Because this variance is relatively small this can be justified (Barbour and Pugliese 2004) but this will have as consequence that this variance in neglected in the calculation of the relatedness. This will introduce a small error in the calculation of the relatedness used in this paper.

The second scenario that is of relevance is the invasion of a patch that is at quasi-equilibrium by an individual of a different strain. We first note that if the dispersal rates are the same, the ensemble mean of the total number of individuals is well described by the logistic equation given above. If the dispersal rates are different the total number changes slightly as the invader takes over the local population. As a consequence, there is a negative ensemble covariance between the numbers of the two strains in the local population, and this covariance initially decreases with rate $-2r$. The ensemble variances increase initially with rate $2r$. As a result, the net contribution of the variance and covariance on the differential equation governing the ensemble mean (eq 16) is small and we can therefore describe the local population dynamics following the invasion of a second strain by (16) with $c = 0$. This implies that if the mutant population is initially small, its stochastic dynamics are given by a linear birth-death process and that the growth rate of the ensemble mean is approximately equal to the difference between the birth and death rates. It is important to realise that in this case the ensemble mean incorporates the effects of local extinctions. Based on this we will use the equations for the ensemble mean to describe the local dynamics while, importantly, this description takes into account the loss through local extinctions. For reinvasion we therefore do not explicitly incorporate a term for the probability of loss through local demographic extinction.

This leads us to assume, following Metz and Gyllenberg (2001) and Parvinen et al. (2003), that for low numbers the dynamics of the invader can be represented by the deterministic equivalent of the stochastic model, albeit that we maintain the non-linearity of the logistic model. We note, however, that the calculation of the fitness for model incorporating the full stochastic behaviour following invasion of a novel strain in a finite population is not completely known and that this area would benefit from a rigorous mathematical treatment.

Appendix 2: Derivation of marginal fitness

The marginal fitness can be found by differentiation with respect to γ^* at $\gamma^* = \gamma^\circ = \gamma$. The following identities hold if $\gamma^* = \gamma^\circ = \gamma$:

$$\tilde{x} = \tilde{x}^\circ = \tilde{x}^*,$$

$$\tilde{s}^*(t) = \tilde{s}^\circ(t) = \tilde{x},$$

$$f^\phi(t) = \phi,$$

$$\left.\frac{\mathrm{d}s^\phi(t)}{\mathrm{d}\gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} = \phi\frac{\mathrm{d}\tilde{x}}{\mathrm{d}\gamma}.$$

Using these identities we find:

$$
\left.\frac{\partial W(\gamma^*,\gamma^\circ)}{\partial\gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} = \frac{W(\gamma,\gamma)}{\gamma} + \gamma\alpha\tilde{P}_1\left[\frac{1}{\alpha\tilde{D}}\frac{\mathrm{d}\tilde{x}}{\mathrm{d}\gamma} + \int_0^\infty\frac{\mathrm{d}\tilde{x}}{\mathrm{d}\gamma}\left(\phi^{\circ 2}+\phi^{*2}\right)\exp(-et)\mathrm{d}t \right.
$$
$$
\left.+ \int_0^\infty\tilde{x}\left(\frac{\mathrm{d}f^{\phi^\circ}(t)}{\mathrm{d}\gamma^*}+\frac{\mathrm{d}f^{\phi^*}(t)}{\mathrm{d}\gamma^*}\right)\exp(-et)\mathrm{d}t + \frac{\mathrm{d}u(\gamma^*)}{u(\gamma^\circ)\mathrm{d}\gamma^*}\left(\frac{\tilde{x}}{\alpha\tilde{D}}+\frac{\tilde{x}\phi^*}{e}\right)\right]_{\gamma^*=\gamma^\circ=\gamma}
$$

By using the derivative:

$$
\left.\frac{\mathrm{d}f^\phi(t)}{\mathrm{d}\gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} = -\phi(1-\phi)t + \left.\frac{\mathrm{d}\phi}{\mathrm{d}\gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} \tag{23}
$$

we find after evaluation of the integrals

$$
\left.\frac{\partial W(\gamma^*,\gamma^\circ)}{\partial\gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} = \frac{1}{\gamma} + \frac{\gamma\alpha\tilde{x}\tilde{P}_1}{e}\left[\frac{e}{\alpha\tilde{D}}\frac{\mathrm{d}\tilde{x}}{\tilde{x}\mathrm{d}\gamma} + \frac{\mathrm{d}\tilde{x}}{\tilde{x}\mathrm{d}\gamma}\left(\phi^{\circ 2}+\phi^{*2}\right) - \frac{2\phi^\circ\phi^*}{e}\right.
$$
$$
\left.+ \left(\frac{\mathrm{d}\phi^\circ}{\mathrm{d}\gamma^*}+\frac{\mathrm{d}\phi^*}{\mathrm{d}\gamma^*}\right)_{\gamma^*=\gamma^\circ=\gamma} + \frac{\mathrm{d}u(\gamma^*)}{u(\gamma^\circ)\mathrm{d}\gamma^*}\left(\frac{e}{\alpha\tilde{D}}+\phi^*\right)\right].
$$

After rearranging, and using $\gamma\alpha\tilde{x} = \frac{m}{u(\gamma)}$, $\alpha\tilde{D} = \frac{m}{u(\gamma)}\tilde{P}$, $\frac{m\tilde{P}_1}{eu(\gamma)} = \frac{\tilde{P}_2}{\tilde{P}}$ and

$$
\left.\frac{\mathrm{d}\phi^\circ}{\mathrm{d}\gamma^*}\right| = 0
$$

this gives

$$
\left.\frac{\partial W(\gamma^*,\gamma^\circ)}{\partial\gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} = \frac{1}{\gamma} + \frac{\mathrm{d}\tilde{x}}{\tilde{x}\mathrm{d}\gamma}\frac{\tilde{P}_1+\tilde{P}_2\left(\phi^{\circ 2}+\phi^{*2}\right)}{\tilde{P}} - \frac{1}{e}\frac{2\phi^\circ\phi^*\tilde{P}_2}{\tilde{P}} + \frac{\mathrm{d}u}{u\mathrm{d}\gamma}\left(\frac{\tilde{P}_1}{\tilde{P}}+\frac{\tilde{P}_2}{\tilde{P}}\phi^*\right) + \left.\frac{\tilde{P}_2}{\tilde{P}}\frac{\mathrm{d}\phi^*}{\mathrm{d}\gamma^*}\right|_{\gamma^*=\gamma}
$$

which is equation (9) in the text. By tracing back the terms in this equation through the derivation it is easy to see that the first term results from the overall effect of increasing the dispersal rate, the second term from the change in the local equilibrium size, the third term from the change of the competition process under a change of the dispersal rate, the fourth term from the change in the loss of a patch through demographic stochasticity and, finally, the last term describes the effect of a change in the initial conditions on the competition process.

Appendix 3: Relatedness and unrelatedness in a metapopulation

To calculate relatedness in the metapopulation we first describe the dynamics of two strains at the metapopulation level. The dynamics of patches which harbour only a single strain can be formulated in analogy with the Levins' model. The superscript refers to the strain that occupies the patch, the subscript to the number of invasions in the patch, and $P_1^i + P_2^i = P^i$. The variable Q denotes the fraction of patches with mixed occupancy. The metapopulation dynamics are:

$$\begin{aligned}
\dot{P}_1^\circ &= \alpha u(\gamma)D^\circ(1 - P^\circ - P^* - Q) - eP_1^\circ - (\alpha D^\circ + \alpha D^*)P_1^\circ \\
\dot{P}_2^\circ &= \alpha D^\circ P_1^\circ - eP_2^\circ \\
\dot{P}_1^* &= \alpha u(\gamma)D^*(1 - P^\circ - P^* - Q) - eP_1^* - (\alpha D^\circ + \alpha D^*)P_1^* \\
\dot{P}_2^* &= \alpha D^* P_1^* - eP_2^*
\end{aligned} \tag{24}$$

Next we describe the dynamics of the patches with mixed occupancy. We denote the fraction of patches that received the starred strain first and the other strain second by Q^*. If the order of invasion is the other way round we denote them Q°, and $Q = Q^\circ + Q^*$. The dynamics are given by

$$\begin{aligned}
\dot{Q}^* &= \alpha D^\circ P_1^* - eQ^* \\
\dot{Q}^\circ &= \alpha D^* P_1^\circ - eQ^\circ
\end{aligned}$$

and the number of dispersers is given by

$$\begin{aligned}
\alpha D^\circ &= m/u(\gamma)(P_1^\circ + P_2^\circ + (1 - \phi^*)Q^* + (1 - \phi^\circ)Q^\circ) \\
\alpha D^* &= m/u(\gamma)(P_1^* + P_2^* + \phi^* Q^* + \phi^\circ Q^\circ)
\end{aligned}$$

Because the strains are selectively neutral, the dynamics will be strongly determined by the initial conditions, in particular the fraction of starred individuals. Let a fraction ω of all individuals in the metapopulation be marked with a star. The total number of individuals in the metapopulation is $\tilde{x}\tilde{P}$ and hence the total number of marked individuals is $\omega\tilde{x}\tilde{P}$ whilst the total number of non-marked individuals is $(1-\omega)\tilde{x}\tilde{P}$. Although the fraction of starred individuals is fixed, the distribution over the different patches is not, and will equilibrate over time. At the equilibrium the dispersal pool represents a random selection of individuals and hence $\alpha D^\circ = m(1-\omega)\tilde{P}/u(\gamma)$ and $\alpha D^* = m\omega\tilde{P}/u(\gamma)$. Therefore also the fractions of the respective patches follow from this pattern of random colonisation and at equilibrium the population sizes are: $P_1^\circ = (1-\omega)\tilde{P}_1$, $P_1^* = \omega\tilde{P}_1$, $P_2^\circ = (1-\omega)^2\tilde{P}_2$, $P_2^* = \omega^2\tilde{P}_2$, $Q^\circ = Q^* = \omega(1-\omega)\tilde{P}_2$. It is easily verified that these values represent an equilibrium by substituting them into the differential equations.

The probability of picking two individuals at random from the metapopulation and both of them being marked with a star is $p_m^* = \omega^2$, the probability of picking two individuals marked with a circle from the metapopulation is $p_m^\circ = (1-\omega)^2$. The probability of picking two individuals marked with a star from the same patch is given by

$$p_p^* = \frac{\omega\tilde{P}_1 + \omega^2\tilde{P}_2 + \omega(1-\omega)\tilde{P}_2(\phi^{\circ 2} + \phi^{*2})}{\tilde{P}}.$$

Similarly, the probability of picking two individuals from the same patch that both are marked with a circle is

$$p_p^\circ = \frac{(1-\omega)\tilde{P}_1 + (1-\omega)^2\tilde{P}_2 + \omega(1-\omega)\tilde{P}_2(\phi^{\circ 2} + \phi^{*2})}{\tilde{P}}.$$

The relatedness within patches relative to the metapopulation is calculated as $R = (p_p^\circ + p_p^* - p_m^\circ - p_m^*)/(1 - p_m^\circ - p_m^*)$ (Pamilo 1984; Queller and Goodnight 1989). After rearranging this is equal to

$$R = \frac{\tilde{P}_1 + \tilde{P}_2(\phi^{\circ 2} + \phi^{*2})}{\tilde{P}}.$$

In the context of the invasion of a rare mutant relatedness can also be interpreted as the probability of a rare mutant to encounter another mutant individual in its patch. Let us think of the individuals marked with a star as selectively neutral mutants. The probability of picking such individuals from

the same patch conditional that one of the individuals is a mutant is p_p^*/ω. If the mutant is rare this probability reduces to $\lim_{\omega \to 0} \frac{p_p^*}{\omega} = R$, (see also Day and Taylor (1998)). This is interpretation of relatedness is similar to that used by Van Baalen and Rand (1998) and Ferriere and Le Galliard (2000).

In a similar vein we can calculate the probability of picking two different individuals of not being related. We can define such 'unrelatedness' as $\bar{R} = 1 - R = \tilde{R}$. It is easy to establish that this quantity equals

$$\bar{R} = \frac{2\tilde{P}_2 \phi^\circ \phi^*}{\tilde{P}}.$$

This corresponds to the probability of a rare but selectively neutral mutant encountering a non-mutant. The probability of picking two differently marked individuals from a patch is

$$p_u = \frac{2\phi^*(1 - \phi^*)\bar{Q}^* + 2\phi^\circ(1 - \phi^\circ)\bar{Q}^\circ}{\tilde{P}} = \frac{2\omega(1 - \omega)\tilde{P}_2 \phi^\circ \phi^*}{\tilde{P}}.$$

Therefore, the probability of a rare but selectively neutral mutant to encounter a non-mutant is $\lim_{\omega \to 0} \frac{p_u}{\omega} = \bar{R}$. The argument that the invasion rate of rare mutants depends on the rate with which a rare mutant encounters other such mutants is equivalent to an interpretation in terms of relatedness.

Appendix 4: multiple invasions

Here we will outline results when a patch can be invaded more than two times. We will first deal with three invasions. Throughout this appendix we will need results on the equilibrium number of patches that have received i invaders. Although this is straightforward and can be done by extending system (3) to account for a maximum of n invasions, the calculations tend to become tedious. To reduce tedium and clutter we will simplify the model somewhat by choosing $u(\gamma) = 1$. This removes the effect of stochastic extinction, from the following consideration, however, as this is a direct fitness effect it will not change under repeated invasions and therefore the fitness contribution of this effect will remain the same under repeated invasion. Solving the extended system (3) for equilibrium with $u(\gamma) = 1$ gives $\tilde{P}_i = (1 - \tilde{P})\tilde{P}^i$ for $i < n$ and $\tilde{P}_n = \tilde{P}^n$.

When every patch can be invaded up to three times we will denote the initial fractions as ϕ^{ijk} where the superscript indicates the order of the invasions. In case of 2 invasions we use a double superscript instead of a triple superscript. Note that because we now have to specify all invaders and that therefore our notation here is slightly different from that in the rest of the paper: a patch that has received first the starred strain, and then the circle has initial condition $\phi^{*\circ}$, in contrast with previous notation which used only a single superscript in this case.

The fitness if at most 3 invasions are allowed is given by

$$W(\gamma^*, \gamma^\circ) =$$

$$\gamma^* \tilde{x} \frac{\alpha(1 - \tilde{P})}{m} + \gamma^* \alpha(1 - \tilde{P})\tilde{P} \int_0^\infty f^{\phi^{*\circ}}(t)\tilde{s}^{\phi^{*\circ}}(t)\exp(-mt)\mathrm{d}t$$

$$+\gamma^* \alpha(1 - \tilde{P})\tilde{P}^2 \int_0^\infty m\exp(-mt_1) \int_0^\infty f^{\frac{f^{\phi^{*\circ}}(t_1)\tilde{s}^{\phi^{*\circ}}(t_1)}{\tilde{s}^{\phi^{*\circ}}(t_1)+1}}(t_2)\tilde{s}^{\frac{f^{\phi^{*\circ}}(t_1)\tilde{s}^{\phi^{*\circ}}(t_1)}{\tilde{s}^{\phi^{*\circ}}(t_1)+1}}(t_2)\exp(-et_2)\mathrm{d}t_2\mathrm{d}t_1$$

$$+\gamma^* \alpha \tilde{P}_1 \int_0^\infty f^{\phi^{\circ*}}(t)\tilde{s}^{\phi^{\circ*}}(t)\exp(-mt)\mathrm{d}t$$

$$+\gamma^* x \tilde{P}_1 \tilde{P} \int_0^\infty m\exp(-mt_1) \int_0^\infty f^{\frac{f^{\phi^{\circ*}}(t_1)\tilde{s}^{\phi^{\circ*}}(t_1)}{\tilde{s}^{\phi^{\circ*}}(t_1)+1}}(t_2)\tilde{s}^{\frac{f^{\phi^{\circ*}}(t_1)\tilde{s}^{\phi^{\circ*}}(t_1)}{\tilde{s}^{\phi^{\circ*}}(t_1)+1}}(t_2)\exp(-et_2)\mathrm{d}t_2\mathrm{d}t_1$$

$$+\gamma^* \alpha \tilde{P}_2 \int_0^\infty f^{\phi^{\circ\circ*}}(t)\tilde{s}^{\phi^{\circ\circ*}}(t)\exp(-et)\mathrm{d}t$$

The first term is the production of dispersers following invasion of a starred individual in an empty patch until this patch is invaded again or hit by a catastrophe. Note that the lifetime of such a patch is $(e + \alpha\tilde{D})^{-1} = m^{-1}$ and the probability that this patch is reinvaded is $\frac{\alpha\tilde{D}}{e + \alpha\tilde{D}} = \tilde{P}$. The second (third) term is the production of dispersers of such a patch after its second (third) invasion. The fourth and fifth terms represent the production of dispersers by a patch that is first colonised by an individual

with dispersal rate γ°, the sixth term the production of dispersers by a patch that is first colonised twice by an individual with growth rate γ° and then reinvaded by an individual with dispersal rate γ^*. It is easy to verify that $W(\gamma, \gamma) = 1$.

The marginal fitness is given by

$$
\left.\frac{\partial W(\gamma^*, \gamma^\circ)}{\partial \gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} = \frac{1}{\gamma} + \frac{1}{\tilde{x}}\frac{d\tilde{x}}{d\gamma}R - \frac{\tilde{P}_2}{\tilde{P}}\left[\frac{1}{m} + \frac{\tilde{P}}{e}\frac{\tilde{x}}{\tilde{x}+1}\right][\phi^{*\circ}(1-\phi^{*\circ}) + \phi^{\circ*}(1-\phi^{\circ*})]
$$

$$
-\frac{1}{e}\frac{\tilde{P}_3}{\tilde{P}}[\phi^{*\circ\circ}(1-\phi^{*\circ\circ}) + \phi^{\circ*\circ}(1-\phi^{\circ*\circ}) + \phi^{\circ\circ*}(1-\phi^{\circ\circ*})]
$$

$$
+m\tilde{P}\left[\frac{\tilde{P}_1}{\tilde{P}}\left(\frac{1}{m} + \frac{\tilde{P}}{e}\frac{\tilde{x}}{\tilde{x}+1}\right) + \frac{1}{e}\frac{\tilde{P}_2}{\tilde{P}}(\phi^{*\circ 2} + \phi^{\circ*2})\right]\frac{1}{(\tilde{x}+1)^2}\frac{d\tilde{x}}{d\gamma}
$$

with

$$
R = \frac{\tilde{P}_1 + \tilde{P}_2(\phi^{*\circ 2} + \phi^{\circ*2}) + \tilde{P}_3(\phi^{*\circ\circ 2} + \phi^{\circ*\circ 2} + \phi^{\circ\circ*2})}{\tilde{P}}.
$$

The unrelatedness is given by:

$$
\begin{aligned}
\bar{R} &= 1 - R \\
&= \frac{\tilde{P}_2 + \tilde{P}_3 - \tilde{P}_2(\phi^{*\circ 2} + \phi^{\circ*2}) - \tilde{P}_3(\phi^{*\circ\circ 2} + \phi^{\circ*\circ 2} + \phi^{\circ\circ*2})}{\tilde{P}} \\
&= \frac{\tilde{P}_2(\phi^{*\circ}(1-\phi^{*\circ}) + \phi^{\circ*}(1-\phi^{\circ*})) + \tilde{P}_3\left(\phi^{*\circ\circ}(1-\phi^{*\circ\circ}) + \phi^{\circ*\circ}(1-\phi^{\circ*\circ}) + \phi^{\circ\circ*}(1-\phi^{\circ\circ*})\right)}{\tilde{P}}.
\end{aligned}
$$

This shows that the effect of an extra individual invading a patch of finite size on reinvasion has a subtle effect on the fitness. If the local population size is large, this effect will be negligible. For large $\tilde{x}$ (corresponding to large k) the fitness is approximately:

$$
\left.\frac{\partial W(\gamma^*, \gamma^\circ)}{\partial \gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} = \frac{1}{\gamma} - \frac{1}{r-\gamma}R - \frac{\tilde{P}_2}{\tilde{P}}\left[\frac{1}{m} + \frac{\tilde{P}}{e}\right][\phi^{*\circ}(1-\phi^{*\circ}) + \phi^{\circ*}(1-\phi^{\circ*})]
$$

$$
-\frac{1}{e}\frac{\tilde{P}_3}{\tilde{P}}[\phi^{*\circ\circ}(1-\phi^{*\circ\circ}) + \phi^{\circ*\circ}(1-\phi^{\circ*\circ}) + \phi^{\circ\circ*}(1-\phi^{\circ\circ*})]
$$

The factor $[\frac{1}{m} + \frac{\tilde{P}}{e}]$ can be interpreted as the dilution factor of the relatedness following subsequent reinvasion. It can be read as the average time until the second reinvasion $1/m$, plus the lifetime of the resulting patch ($1/e$ weighed by the probability that this patch gets reinvaded ($\tilde{P}$). If we allow n invasions this factor will become $\sum_{i=0}^{n-3}\frac{\tilde{P}^i}{m} + \tilde{P}^{n-2}e$. For terms which represent patches which have already received k invaders, similarly, have a dilution term $\sum_{i=0}^{n-k-1}\frac{\tilde{P}^i}{m} + \frac{\tilde{P}^{n-k}}{e}$.

This interpretation suggests that if infinitely many invasions are allowed the marginal fitness takes the form (again ignoring finite size effects)

$$
\begin{aligned}
\left.\frac{\partial W(\gamma^*, \gamma^\circ)}{\partial \gamma^*}\right|_{\gamma^*=\gamma^\circ=\gamma} &= \frac{1}{\gamma} - \frac{1}{r-\gamma}R - \left[\sum_{i=0}^{\infty}\frac{\tilde{P}^i}{m}\right]\bar{R} \\
&= \frac{1}{\gamma} + \frac{1}{\tilde{x}}\frac{d\tilde{x}}{d\gamma}R - \frac{1}{e}\bar{R}
\end{aligned}
$$

The relatedness in this case is given by

$$
\begin{aligned}
R &= \frac{1}{\tilde{P}}\sum_{i=1}^{\infty}\tilde{P}_i\left(\left(\frac{\tilde{x}}{\tilde{x}+1}\right)^{2(i-1)} + \left(\frac{1}{\tilde{x}+1}\right)^2\sum_{j=0}^{i-2}\left(\frac{\tilde{x}}{\tilde{x}+1}\right)^{2j}\right) \\
&= (1-\tilde{P})\sum_{i=0}^{\infty}\tilde{P}^i\left(\left(\frac{\tilde{x}}{\tilde{x}+1}\right)^{2i} + \tilde{x}^2\frac{1-\left(\frac{\tilde{x}}{1+\tilde{x}}\right)^{2i}}{1+2\tilde{x}}\right) \\
&= \left(\frac{\tilde{x}}{\tilde{x}+1}\right)^2 + \frac{\tilde{P}}{1+\tilde{x}(2+\tilde{x}(1-\tilde{P}))}
\end{aligned}
$$

Note that for large $\tilde{x}$ we find that $R \approx 1 - 2/\tilde{x}$.

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