In this thesis we formulate and analyze a structured population model, with infectious disease dynamics, based on a similar life-cycle as with individuals of the Hamilton-May model. Each individual is characterized by a strategy vector (state dependent dispersal), and depending on the infectious status of the individual, it will use a strategy accordingly. We begin by assuming that every individual in the population has the same strategy, and as the population equilibrates we consider a mutant, with its own strategy, entering the population, trying to invade. We apply the theory of Adaptive dynamics to model the invasion fitness of the mutant, and to analyze the evolution of dispersal. We show that evolutionary branching is possible, and when such an event happens, the evolutionary trajectories, described by the Canonical equation of Adaptive dynamics, of two strategies evolve into the extinction of one branch. The surviving branch then evolves to the extinction of the disease.

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Evolution of Infection
State-Dependent Dispersal

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Introduction

In this thesis we formulate an infectious disease model where individuals have a similar life-cycle than those in the Hamilton-May model (1977), which is a metapopulation model, and analyze the evolution of the dispersal strategy of the individual through numerical analysis.

In the first section we present the Perron-Frobenius theorem, theory of Adaptive dynamics, and the SIR-model. The Perron-Frobenius theorem states that for a matrix of a certain form there exists a dominant eigenvalue. This dominant eigenvalue is used as a measure of fitness in the case of structured population models in discrete time. The theory of Adaptive dynamics begins with defining key components: trait vector, the fitness invasion function, the selection gradient, and the canonical equation. We furthermore give conditions for evolutionary and strong convergence stability of singular strategies in multivariate cases, and lastly show how, in a two dimensional case, can the trace and determinant of a projection matrix be used instead of the dominant eigenvalue. In the second section we present the SIR-model of infectious disease.

In the third section we present the Hamilton-May model and show how we can construct the invasion fitness of said model. Furthermore, we show that in the Hamilton-May-model the singular strategy is always an evolutionary stable strategy. We have have a large number of sites, that can be occupied by a single individual. It gives birth to a large number of offspring and a fraction of them disperse to a global dispersal pool, where they are well-mixed and then land in each site with equal probability, and only a fraction of them survive dispersal. In each sites the offspring compete for the occupancy of respective sites, and the winner is determined by a fair lottery. This is when we consider a mutant, who's dispersal strategy differs only slightly from the resident population. We formulate the fitness invasion function for this specific model, and show that of what form the singular strategy is.

In the fourth we begin with the formulation of our model, and we first consider that that the population has a single evolving trait with state dependent dispersal, where the dispersal rate of an infected individual is a function of the dispersal rate of the susceptible individual. The adults that occupy each site give birth to offspring, allowing vertical transmission of the disease from infected parents with a fixed probability. The offspring disperse, with rates dependent on their state, where they are well-mixed, and thus randomly land into sites. Surviving dispersal is assumed to be state dependent as well. After dispersal, the infection is transmitted via horizontal transmission from infected to susceptible offspring, and the dynamics is described by the SIR-model. In each site a winner of the site is determined by weighted lottery.
We then consider a rare mutant, with a different state dependent dispersal strategy, entering the population. We construct the projection matrix of the mutant and show that, in the cases presented, that the evolutionary singular strategy is attracting and evolutionarily stable. In the sixth and seventh sections we consider two evolving traits: the dispersal rate of the susceptible individual and the dispersal strategy of the infected individual. We show that, given specific parameter values, there are different evolutionary outcomes.

In the last section we discuss the results of the model, in the one trait and two trait cases, and shortly present other models where either infection affects dispersal, and some other metapopulation models.
1 Preliminaries

In this section we present mathematical tools in modeling physiologically structured population dynamics. We also present the basic theory of Adaptive Dynamics.

1.1 The Perron-Frobenius theorem

We say that a matrix $A \in \mathbb{R}^{n \times n}$ is non-negative (positive), $A \geq 0$ ($A > 0$) if all the components of $A$ are non-negative (positive), i.e. $A_{ij} \geq 0$ ($A_{ij} > 0$) for all $i, j \in \{1, \ldots, n\}$. Furthermore a non-negative matrix $A$ is primitive if there exists a strictly positive integer $k$, such that $A^k > 0$.

**Theorem 1.1.1. (Perron-Frobenius theorem)** Suppose $A \in \mathbb{R}^{n \times n}$ is a non-negative and primitive matrix. Then there exists a strictly positive and real eigenvalue $\lambda$, which has strictly positive associated left and right eigenvectors, and such that $|\lambda_i| < \lambda$ is satisfied for any other eigenvalue $\lambda_i$ of $A$.

**Proof.** For proof see Horn & Johnson (2013).

The eigenvalue $\lambda$, that the Perron-Frobenius theorem shows to exist, is also called the dominant eigenvalue.

1.2 Adaptive dynamics of structured populations

1.2.1 States

In physiologically structured population models we consider that the individuals of the populations have one specific state that they are in at a given time. These states can reflect age or physiological attributes, e.g. either being infected or susceptible to a specific disease, etc. Let $\Omega = \{\omega_1, \ldots, \omega_n\}$ be the set of states or state space (e.g. Diekmann et al. 2013). We also consider that an individual can go through a process that changes its state, which we call state transition. The state transitions we consider in the model formulated in this theses is a process where a susceptible individuals (S-state individual) gets infected, and becomes an infected individual (I-state individual), thus limiting the state space to $n = 2$, but for now we keep $n$ arbitrary.

1.2.2 Strategies and invasion fitness

We also consider that each individual has a trait vector $\bar{x} = [x_1 \ldots x_m]^T \in \mathbb{R}^m$ (also called strategy), and clonal reproduction in the population, i.e. every offspring is a genetic copy of it’s parent with the same strategy. We
apply the theory of Adaptive dynamics to model how a rare mutant, with strategy \( \bar{y} \in \mathbb{R}^m \) (that appears due random mutation and with low probability) can invade a resident population with \( K \) residents, the set of resident strategies is \( E = \{ \bar{x}_1, \bar{x}_2, \ldots, \bar{x}_K \} \), assuming that the entire population consists of individuals with strategies \( \bar{x}_k \), \( k \in \{1,2,\ldots,K\} \), and the population of residents has achieved a positive stable equilibrium. The main assumptions in Adaptive dynamics are the rarity of mutations, and small mutation steps, i.e. the norm of the difference \( \bar{y} - \bar{x}_k \) is small, but positive, for some \( k \). The key component of Adaptive dynamics is the invasion fitness of a mutant \( s_k(E, \bar{y}_k) \), which is defined as the long-term exponential growth rate of a phenotype in a given environment, i.e. the long-term exponential growth rate of a rare mutant with strategy \( \bar{y}_k \) in an environment set by \( E \). A mutant with strategy \( \bar{y}_k \) has a positive probability of invading and replacing resident \( \bar{x}_k \), if \( s_k(E, \bar{y}_k) > s_k(E, \bar{x}_k) \). By definition and with the assumption that the residents are at a stable equilibrium, we easily see that \( s_k(E, \bar{x}_k) = 0 \). We also define the selection gradient of resident \( k \) as the column vector \( \nabla s_k(E, \bar{y}_k) \mid_{\bar{y}_k=\bar{x}_k} \), where the \( l \)th component the selection gradient is

\[
\frac{\partial s_k(E, \bar{y}_k)}{\partial y_{kl}} \bigg|_{\bar{y}_k=\bar{x}_k},
\]

where \( y_{kl} \) is the \( l \)th component of \( \bar{y}_k \). (Durinx et al. 2008, Leimar 2009)

### 1.2.3 The canonical equation

We first define the Next-Generation Matrix \( L(E, \bar{y}) \) by it’s components: \( L(E, \bar{y})_{lm} \) is the expected number of offspring with state \( l \) born over the lifetime of an individual with trait vector \( \bar{y} \) that was born with state \( m \), given steady environmental conditions as specified by \( E \). Furthermore, we denote the dominant eigenvalue of \( L(E, \bar{y}) \) as \( R_0(E, \bar{y}) \). The canonical equation of Adaptive dynamics for structured population models is of the form

\[
\frac{d\bar{x}_k}{dt} = \frac{T_f}{T_s} \cdot \frac{\hat{n}_{kk} \mu_{mut}}{B(E, \bar{x}_j)} \cdot C \cdot \frac{\partial \log(R_0(E, \bar{y}))}{\partial \bar{y}} \bigg|_{\bar{y}=\bar{x}_k},
\]

where the column vector’s

\[
\frac{\partial \log(R_0(E, \bar{y}))}{\partial \bar{y}}
\]
The lth component is
\[
\frac{\partial \log(R_0(E, \bar{y}))}{\partial y_l}.
\]
Furthermore, \(T_f\) is the average age of an individual giving birth, \(T_s\) is the expected lifespan of an individual, \(\bar{n}_k\) is the equilibrium density of the \(k\)th resident, \(\mu_{\text{mut}}\) is the probability of a mutational event, and \(C\) is the mutational variance-covariance matrix. Lastly, \(B(E, \bar{x}_k) = \sum_l u_l \text{Var}(\sum_m v_m \xi_{ml})\), \(l, m \in \{1, \ldots, n\}\), where \(u = [u_1 \ldots u_n]^\top\) and \(v = [v_1 \ldots v_n]\) are the right and left eigenvectors, respectively, corresponding to the leading eigenvalue \(R_0\) of the Next-Generation Matrix \(L(\bar{x}_k, E)\), and \(\xi_{ml}\) is a random variable of the number of \(m\)-state offspring from one \(l\)-state parent with trait vector \(\bar{x}_k\).
(Diekmann & Law 1996, Durinx et al. 2008)

The trajectory of resident \(k\) in the strategy space is found by integrating the canonical equation. What the canonical equation looks like in practice is model dependent and we will return to it in section 6.3 and 7.4.

1.2.4 Singularities and classification of them

A point \(\hat{x}\) at which the selection gradient is zero is called a singular strategy (or singularity for short). We have set the theory of Adaptive dynamics to accommodate \(K\) residents, but for emergence of several residents, we need to examine what kind of singular strategies we have in the monomorphic case (i.e. when only one resident is present). Evolutionary branching, i.e. a process in which a single resident evolves to point at which selection is disruptive and the coexistence of several residents (polymorphism) is possible, occurs when a singularity is attracting and invadable by a mutant strategy near by. (Geritz et al. 1998, Geritz et al. 2016)

We define that a singularity \(\hat{x}\) is strongly convergence stable, if it is an attractor of the evolutionary dynamics for any mutational process provided the mutational step sizes are sufficiently small. (Leimar 2009)

We define the selection Hessian as a matrix \(H_{kk}\) with elements
\[
(H_{kk})_{ij} = \frac{\partial^2 s_k(E, \bar{y}_k)}{\partial y_{ki} \partial y_{kj}} \bigg|_{\bar{y}_k = \bar{x}_k = \hat{x}},
\]
and denote a matrix \(Q_{kl}\) with elements
\[
(Q_{kl})_{ij} = \frac{\partial^2 s_k(E, \bar{y}_k)}{\partial y_{ki} \partial x_{lj}} \bigg|_{\bar{y}_k = \bar{x}_k = \hat{x}}.
\]

The Jacobian \(J\) of the selection gradient is given by \(J = H + Q\), where \(H\) is a
symmetric block diagonal matrix with the selection Hessians $H_{kk}$ as blocks, and $Q$ is a matrix with blocks $Q_{jl}$. (Leimar 2009)

Leimar (2009) showed that a singularity is strongly convergent stable if the symmetric part of the Jacobian $J$ evaluated at the singularity is negative definite, and for a singularity to be evolutionarily stable (locally invadable), it is sufficient that the Hessians $H_{kk}$ of all residents are negative definite and necessary that they are negative semidefinite.

1.2.5 Invasion fitness in a structured population

To model the invasion dynamics of a rare mutant in discrete time we define the projection matrix of a mutant to be the matrix $A := A(E, y) = [a_{ij}(E, \bar{y})]$, where $a_{ij}(E, \bar{y})$ is the expected number of $\omega_i$-state descendants with trait value $y_i$ in one time step from one mutant $\omega_j$-state individual with trait value $y_j$ in an environment set by $E = \{\bar{x}_1, \ldots, \bar{x}_k\}$. What each $a_{ij}$ looks like in practice is model dependent, since the number of states vary among models, as does the life cycle of an individual. We consider that the invasion dynamics of a rare mutant, with strategy $\bar{y}$, is described by the following equation

$$M_{t+1} = AM_t$$

where $M_{t,i}$ (the $i$th component of the column vector $M_t$) is the density of $s_i$-state mutants at time $t = 0, 1, 2, \ldots, i \in \{1, \ldots, n\}$.

The projection matrix $A(E, \bar{y})$ is by its definition non-negative. Assuming that it is primitive the Perron-Frobenius theorem can be applied to the projection matrix. As shown in Metz et al. (1992) the logarithm of the dominant eigenvalue of the projection matrix $A$ is a measure of invasion fitness of the mutant. The model we formulate in this thesis assumes that generations are non-overlapping, thus the dominant eigenvalues of the Next-Generation Matrix and the projection matrix are the same, since the matrices are the same.

We will now show that if the number of states is two, then invasion fitness is determined by the trace and determinant of the projection matrix $A$. Let $A$ be a two-by-two matrix,

$$A = \begin{bmatrix} a & b \\ c & d \end{bmatrix},$$

and let $\lambda$ be the dominant eigenvalue of $A$. We first show that $\text{tr}(A)^2 - 4 \cdot \text{det}(A) \geq 0$ holds: assume that $\text{tr}(A)^2 - 4 \cdot \text{det}(A) < 0$, and equivalently

$$(a + d)^2 - 4(ad - cb) < 0 \iff a^2 + 2ad + d^2 - 4ad + 4cb < 0.$$
\[ a^2 - 2ad + d^2 + 4cb < 0 \]
\[ (a - d)^2 + 4cb < 0, \]

which is a contradiction, since \( A \) is positive, i.e. \((a - d)^2 \geq 0\) and \(4cb \geq 0\). Since \( \lambda \) is the dominant eigenvalue of \( A \), it is of the form
\[
\lambda = \frac{\text{tr}(A) + [\text{tr}(A)^2 - 4 \cdot \det(A)]^{1/2}}{2}.
\]

Assume that \( \lambda < 1 \). Now
\[
\frac{\text{tr}(A) + [\text{tr}(A)^2 - 4 \cdot \det(A)]^{1/2}}{2} < 1
\]
\[\iff 2 - \text{tr}(A) > [\text{tr}(A)^2 - 4 \cdot \det(A)]^{1/2} \geq 0 \]
\[\iff 4 - 4 \cdot \text{tr}(A) + \text{tr}(A)^2 > \text{tr}(A)^2 - 4 \cdot \det(A), \text{ and } \text{tr}(A) < 2 \]
\[\iff \text{tr}(A) - \det(A) < 1, \text{ and } \text{tr}(A) < 2. \]

Trivially \( \text{tr}(A) > 2 \) implies \( \lambda > 1 \). Thus we can use \( \text{tr}(A) - \det(A) \) as a measure for invasion fitness for small mutation steps. Furthermore, for any resident strategy \( \text{tr}(A) < 2 \) always holds. Thus by continuation, and considering small mutation steps (i.e. \( |\bar{x} - \bar{y}| < \varepsilon \), for a small \( \varepsilon \), resident strategy \( \bar{x} \), mutant strategy \( \bar{y} \)) \( \text{tr}(A) < 2 \) holds. (Metz & Leimar 2011)
2 The SIR-model of infectious diseases

A classic way of modeling infectious disease dynamics in a closed population is the SIR-model, where \( S \) stands for susceptible, \( I \) for infected, and \( R \) for recovered or removed.

Let \( S := S(t) \) be the number of susceptible individuals at time \( t \), \( I := I(t) \) be the number of infected individuals at time \( t \), and \( R = R(t) \) be the number of removed individuals at time \( t \). We consider individuals moving like molecules in a closed area, and that individuals have a positive probability of coming into contact with each other, forming a pair for a short time, and then moving on. This contact can be described as an elastic collision of molecules and the rate that this happens is proportional to \( SI \). Furthermore, \( N \) is the total number of individuals, i.e. \( S + I + R = N \). The principle on which this contact process is based on is called the Law of Mass-action. Furthermore, we consider that the time and individual is infected is exponentially distributed with parameter \( \mu \). From these assumptions we are able to formulate the SIR-model as the following system of ODEs (Ordinary Differential equations):

\[
\begin{align*}
\dot{S} &= -\beta SI \\
\dot{I} &= \beta SI - \mu I \\
\dot{R} &= \mu I,
\end{align*}
\]

where \( \beta \) is the transmission rate per unit time of the disease, and \( \mu \) is the recovery rate of an individual. The term \( \beta SI \) describes here the incidence of the disease, which is defined as the number of new infected individuals per unit time, where \( \beta \) is a combined term of the contact rate, probability of transmission, and the total population \( N \), which is constant in time, since

\[
\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0.
\]

Starting with an initially low number of infected individuals the root of the final size equation \( \log s_{\infty} = \lambda_0 (1 - s_{\infty}) \) (where \( \lambda_0 \) is the basic reproduction number of the disease and \( s_{\infty} = \lim_{t \to \infty} S(t)/N \)) shows what is the fraction of susceptibles after an epidemic. (Diekmann et al. 2013)

To illustrate the behavior of solutions Figure 1 shows the phase portrait, i.e. for each point to which general direction does the solution tend to.

In the model formulated in this thesis, we consider horizontal transmission of a disease by an ODE system similar to the SIR-model, except we consider mortality of all individuals and additional mortality of infected individuals (virulence). Furthermore, we consider that the time interval of the ODE system is finite, i.e. though the system may have asymptotically stable steady...
state, solutions will not converge to it.

Figure 1: Parameter values: $\beta = 4, \mu = 2$. 
3 The Hamilton-May -model for the evolution of dispersal

In this section we formulate and analyze the Hamilton-May model (Hamilton & May 1977).

Let $M$ be the number on distinct sites in a landscape, and at census every site is occupied by exactly one adult individual. Each individual gives birth to $F$ offspring in the beginning of each year, i.e. after census, and dies after the dispersal of offspring, thus freeing the site. These offspring disperse with probability $d$ into the global dispersal pool, i.e. the number of dispersing offspring from a single site is $Fd$. The probability of dispersal can also be referred as the dispersal strategy of a given type of individuals. In the dispersal pool the offspring are well-mixed, and then randomly land into sites, for which they compete only if they survive dispersal. Let $s$ be the probability of surviving dispersal.

We now consider a mutant, with dispersal strategy $D$, and denote $X_n$ as the fraction of sites occupied by a mutant individual in year $n$, $n \in \mathbb{Z}_+$. In a site that was occupied by a mutant in the year $n$, the number of mutant offspring that arrive at this specific site is $F(1-D) + FX_n M D s / M$, where $X_n M$ is the number of sites that are mutant sites, so the number of offspring from all mutant sites is $FX_n M$, and $1/M$ is the probability that the offspring survive dispersal arrive at the specific site we are considering, and so the $M$’s cancel. Using the same method of book-keeping, the number of resident individuals that disperse into a mutant site is $F(1 - X_n) d s$, the number of mutant individuals that disperse into a resident site is $FX_n D s$, and the number of resident individuals in a resident site is $F(1 - d) + FX_n d s$. Furthermore, we consider $F \to \infty$ and $M \to \infty$. We are now ready to formulate the invasion fitness function of a mutant, so we first compute the expected fraction of mutant sites in year $n+1$:

$$X_{n+1} = X_n \frac{F(1-D) + FX_n D s}{F(1-D) + FX_n D s + F(1 - X_n) d s} + (1 - X_n) \frac{FX_n D s}{F(1-d) + FX_n D s + F(1 - X_n) d s}.$$  

When $X_n \ll 1$, we get $X_{n+1} = X_n W(d, D) + O(X_n)$ with

$$W(d, D) := \frac{1 - D}{1 - D + ds} + \frac{D s}{1 - d + ds},$$

and use this initial growth rate as fitness, such that a mutant with strategy $D$
has a positive probability of invasion in a population with resident strategy \( d \) when \( W(d, D) > 1 \).

To find singular strategies we must first solve for which strategies \( d, D \in [0, 1] \) it holds that \( W(d, D) = 1 \):

\[
W(d, D) = \frac{1 - D}{1 - D + ds} + \frac{Ds}{1 - d + ds} = 1 \iff \frac{Ds}{1 - d + ds} = 1 - \frac{1 - D}{1 - D + ds} \\
\iff \frac{1 - d + ds}{Ds} = \frac{1 - D + ds}{ds} \\
\iff (1 - D + ds)D - (1 - d + ds)d = 0 \\
\iff (d - D)(d(s - 1) + 1 - D) = 0 \\
\iff D = d \lor D = d(s - 1) + 1,
\]

and for a singular strategy, we have \( \hat{d} = \hat{d}(s-1)+1 \iff \hat{d} = \frac{1}{2 - s} \). See Figure 2 for the Pairwise invadability plot (PIP) of the invasion fitness function. Using the classification of singular strategies from Geritz et al. (1998) we are able to determine that the singular strategy is always an attracting and evolutionary stable, since

\[
\frac{\partial^2 W}{\partial D^2}(\hat{d}, \hat{d}) = -\frac{2\hat{d}s}{1 - \hat{d} + ds} = \ldots = -2s(s - 2)^2 < 0
\]

and

\[
\frac{\partial^2 W}{\partial d \partial D}(\hat{d}, \hat{d}) + \frac{\partial^2 W}{\partial D^2}(\hat{d}, \hat{d}) = \frac{-s(2\hat{d} + s - 3\hat{d}s + \hat{d}s^2)}{(1 - \hat{d} + ds)^3} = \ldots = s(s - 2)^3 < 0
\]

for all \( s \in [0, 1] \). To summarize: in the Hamilton-May model the singular strategy is always an attracting evolutionary stable strategy (ESS).
Figure 2: Pairwise invadability plot. Area is red when mutant invasion probability is positive, i.e. when $W(d, D) > 1$. When probability of surviving dispersal is set to $s = 0.6$, the ESS is $\hat{d} \approx 0.714$. 
4 The Model

We now introduce an infectious disease into this population, which is contracted through vertical transmission with probability $p$, and through horizontal transmission before site competition. We denote the fraction of sites occupied by an infected individual in year $n$ with $Y_n$. We assume that infected individuals have separate probabilities of dispersal $\delta = \kappa d$, and survival $s_i = \sigma s$, where $\kappa, \sigma \in [0, 1]$. Furthermore, we denote the fraction of sites occupied by a susceptible individual in year $n$ as $X_n$, where $X_n = 1 - Y_n$. From here on we call a site occupied by an infected individual in the previous year as an infected site, or an $I$-state site, and a site occupied by a susceptible individual in the previous year as a susceptible site, or a $S$-state site.

4.1 Within-site dynamics

After dispersal, and before competition, the offspring randomly arrive into sites, and the horizontal transmission of the disease occurs within a given site during the time interval $[0, T]$. For $t \in [0, T]$, we denote $x_k(t)$ and $y_k(t)$, as the number of susceptible and infected individuals at time $t$, respectively, in a $k$-state site, $k \in \{S, I\}$, after dispersal. At time $t = 0$, in year $n$, the number of susceptible and infected offspring that arrive at a given site we denote as $x_{0,k}$ and $y_{0,k}$, respectively, since the initial condition, i.e. the number of individuals that arrive and compete for a given site, is dependent on the site type. The within-site dynamics in susceptible and infected sites is described by the following autonomous system:

\[
\begin{align*}
\dot{x}_k(t) &= -\beta x_k(t)y_k(t) - \mu x_k(t), \\
\dot{y}_k(t) &= \beta x_k(t)y_k(t) - (\mu + \eta)y_k(t),
\end{align*}
\]

with initial conditions $x_k(0) = x_{0,k}$ and $y_k(0) = y_{0,k}$, where $\beta$ is the transmission rate of the disease, $\mu$ is the death rate, and $\eta$ is the disease induced death rate.

For a $S$-state site the initial conditions are

\[
\begin{align*}
x_{0,S} &= F[1 - d + (1 - Y_n)ds + Y_nd(1 - p)s], \\
y_{0,S} &= FY_npd_is_i,
\end{align*}
\]

and for a $I$-state site, the initial conditions are

\[
\begin{align*}
x_{0,I} &= F[(1 - d)(1 - p) + (1 - Y_n)ds + Y_nd(1 - p)s], \\
y_{0,I} &= F[p(1 - d_i) + Y_npd_is_i].
\end{align*}
\]
4.2 Competition

Let \( t \mapsto (x_S(t), y_S(t)) \) and \( t \mapsto (x_I(t), y_I(t)) \) be solutions of (4.1) for susceptible and infected sites, respectively, with initial conditions (4.2) and (4.3), respectively. We furthermore define \((\tilde{x}_S, \tilde{y}_S) := (x_S(T), y_S(T))\) and \((\tilde{x}_I, \tilde{y}_I) := (x_I(T), y_I(T))\). The winner of the site is determined by weighted lottery, where we assume that a susceptible individual has a fixed weighing constant \( \alpha \in \mathbb{R}_+ \). Thus the probabilities of an infected individual winning a site is given by the following:

(i) for a susceptible site, we have \( \frac{\tilde{y}_S}{\alpha \tilde{x}_S + \tilde{y}_S} \),

(ii) for an infected site, we have \( \frac{\tilde{y}_I}{\alpha \tilde{x}_I + \tilde{y}_I} \).

From these probabilities we finally compute the expected fraction of infected sites in year \( n + 1 \):

\[
Y_{n+1} = \frac{\tilde{y}_S}{\alpha \tilde{x}_S + \tilde{y}_S} (1 - Y_n) + \frac{\tilde{y}_I}{\alpha \tilde{x}_I + \tilde{y}_I} Y_n =: G(Y_n).
\]

To find a stable resident equilibrium \( \hat{Y} \), for which \( \hat{Y} = G(\hat{Y}) \), we need numerical analysis. In Figure 3 we have three plots of \( Y_{n+1} = G(Y_n) \) for three different values of the within-site transmission rate \( \beta \).

![Figure 3: Fraction of sites that are infected. We see that if the disease is not infectious enough, the density of infected sites is very low. Parameter values: \( d = 0.5, p = 0.5, s = 0.5, \eta = 0.1, \mu = 0.1, \alpha = 1.25, \kappa = 0.8, \sigma = 0.7 \).](image-url)
5 Evolution of a single trait

5.1 Mutation of the dispersal strategy

We consider a mutant entering the population, and denote the mutant dispersal strategy for a susceptible mutant as $d_{\text{mut}}$ and for an infected mutant as $\delta_{\text{mut}}$. Likewise we denote the dispersal strategies of the resident population as $d$ and $\delta$ for resident susceptible and resident infected individuals, respectively. Furthermore, we assume that the resident population has already reached a stable equilibrium, when a mutant appears, i.e. $X_n = \hat{X}$ and $Y_n = \hat{Y}$, where $\hat{X} + \hat{Y} = 1$.

Equivalently to the resident, we assume that the dispersal strategies of the susceptible and infected mutant individuals are connected with the coefficient $\kappa$, the same as the resident population, i.e. $\delta_{\text{mut}} = \kappa d_{\text{mut}}$, and denote the fractions of sites with regard to residents and mutants as follows:

(i) the fraction of sites occupied by a resident susceptible individual in year $n$ as $X_n$ (sites called \textit{susceptible resident sites})
(ii) the fraction of sites occupied by a resident infected individual in year $n$ as $Y_n$ (sites called \textit{infected resident sites})
(iii) the fraction of sites occupied by a mutant susceptible individual in year $n$ as $X_n^{(\text{mut})}$ (sites called \textit{susceptible mutant site})
(iv) the fraction of sites occupied by a mutant infected individual in year $n$ as $Y_n^{(\text{mut})}$ (sites called \textit{infected mutant site})

5.2 Invasion fitness of a mutant

For a dispersing offspring of a mutant we assume that they do not have an effect on the dynamics of within-site dynamics of a site, that was previously occupied by a resident, but are affected by it.

When the mutant is not rare in a specific site, i.e. the number of mutant individuals that arrive or stay at a given site is large enough, the within-site dynamics is given by

\begin{equation}
\begin{cases}
\dot{x} = -\beta x [y + y^{(\text{mut})}] - \mu x, \\
\dot{y} = \beta x [y + y^{(\text{mut})}] - (\mu + \eta)y, \\
\dot{x}^{(\text{mut})} = -\beta x^{(\text{mut})} [y + y^{(\text{mut})}] - \mu x^{(\text{mut})}, \\
\dot{y}^{(\text{mut})} = \beta x^{(\text{mut})} [y + y^{(\text{mut})}] - (\mu + \eta)y^{(\text{mut})}.
\end{cases}
\end{equation}
with initial conditions
\[
\begin{align*}
  x_k(0) &= x_{0,k}, \\
  y_k(0) &= y_{0,k}, \\
  x_{k,\text{mut}}(0) &= x_{0,k,\text{mut}}, \\
  y_{k,\text{mut}}(0) &= y_{0,k,\text{mut}},
\end{align*}
\]

\( k \in \{S_{\text{mut}}, I_{\text{mut}}\} \), where \( x_k(t) \) and \( y_k(t) \) are the number of resident susceptible and infected individuals, respectively, at time \( t \), and \( x_{k,\text{mut}}(t) \) and \( y_{k,\text{mut}}(t) \) are the number of mutant susceptible and infected individuals, respectively, at time \( t \). The dynamics of the system stated above applies only to mutant sites, infected and susceptible.

Note that since the mutant does not affect the dynamics of the resident site dynamics, the initial conditions of \( S \) and \( I \) sites are similar to what was before, with corresponding solutions of each system. The only difference being the conversion of \( X_n \) to \( \hat{X} \), and \( Y_n \) to \( \hat{Y} \) in the initial conditions of each resident site.

The initial condition of a susceptible resident site is
\[
\begin{align*}
  x_{0,S} &= F \left[ 1 - d + \hat{X}ds + \hat{Y}(1-p)ds \right] \\
  y_{0,S} &= F \left[ \hat{Y}p\delta s_i \right]
\end{align*}
\]

Let \( t \mapsto (x_{S}(t), y_{S}(t)) \) be a solution of (4.1) with initial conditions \((x_{S}(0), y_{S}(0)) = (x_{0,S}, y_{0,S})\), and define \((\tilde{x}_{S}, \tilde{y}_{S}) := (x_{S}(T), y_{S}(T))\).

The initial condition of a infected resident site is
\[
\begin{align*}
  x_{0,I} &= F \left[ (1-p)(1-d) + \hat{X}ds + \hat{Y}(1-p)ds \right] \\
  y_{0,I} &= F \left[ p(1-\delta) + \hat{Y}p\delta s_i \right]
\end{align*}
\]

Let \( t \mapsto (x_{I}(t), y_{I}(t)) \) be a solution of (4.1) with initial condition \((x_{I}(0), y_{I}(0)) = (x_{0,I}, y_{0,I})\), and define \((\tilde{x}_{I}, \tilde{y}_{I}) := (x_{I}(T), y_{I}(T))\).

For a susceptible mutant site the initial conditions for the within-site dynamics are
\[
\begin{align*}
  x_{0,S_{\text{mut}}} &= F \left[ \hat{X}ds + \hat{Y}(1-p)ds \right], \\
  y_{0,S_{\text{mut}}} &= F \left[ \hat{Y}p\delta s_i \right], \\
  x_{0,\text{mut}}(0) &= F \left[ 1 - d_{\text{mut}} \right], \\
  y_{0,\text{mut}}(0) &= 0.
\end{align*}
\]

Let \( t \mapsto (x_{\text{mut}}(t), y_{\text{mut}}(t), x_{\text{mut}}(t), y_{\text{mut}}(t)) \) be a solution of (5.1) with
initial conditions, as stated above. We furthermore define

\[
\begin{align*}
\tilde{x}_{\text{mut}} & := x_{\text{mut}}(T), \\
\tilde{y}_{\text{mut}} & := y_{\text{mut}}(T), \\
\tilde{x}^{(\text{mut})} & := x^{(\text{mut})}(T), \\
\tilde{y}^{(\text{mut})} & := y^{(\text{mut})}(T).
\end{align*}
\]

For a infected mutant site the initial conditions for the within-site dynamics are

\[
\begin{align*}
x_{0,\text{mut}} &= F[X ds + \hat{Y}(1 - p) ds] \\
y_{0,\text{mut}} &= F[Y p \delta s i] \\
x^{(\text{mut})}_0 &= F(1 - p)(1 - d_{\text{mut}}) \\
y^{(\text{mut})}_0 &= F[1 - \delta_{\text{mut}}] p
\end{align*}
\]  

(5.5)

and the dynamics of the site is again the same as in the system above. Let \( t \mapsto (x_{\text{mut}}(t), y_{\text{mut}}(t), x^{(\text{mut})}_\text{mut}(t), y^{(\text{mut})}_\text{mut}(t)) \) be a solution of (5.1) with initial conditions, as stated above. We furthermore define

\[
\begin{align*}
\tilde{x}_{\text{mut}} & := x_{\text{mut}}(T), \\
\tilde{y}_{\text{mut}} & := y_{\text{mut}}(T), \\
\tilde{x}^{(\text{mut})} & := x^{(\text{mut})}(T), \\
\tilde{y}^{(\text{mut})} & := y^{(\text{mut})}(T).
\end{align*}
\]

These will again be used later for computing the probability of a mutant susceptible or infected individual of winning the site.

Though the mutant is assumed rare and does not affect the dynamics of a resident site, it still does have a probability of winning in a resident site, and thus need to solve what is the probability of a susceptible mutant surviving until competition, the probability of a susceptible mutant to get infected at some point during the within-site dynamics and surviving until competition, and the probability of a infected mutant surviving until competition.

Let \( z_k(t) \) be the probability that a susceptible mutant individual is still alive at time \( t \) after it has arrived at a \( k \)-state site, \( k \in \{S, I\} \), and we get the following ODE:

\[
\dot{z}_k = -(\mu + \beta y_k) z_k
\]

where \( y_k = y_k(t) \) is the number of infected resident individuals at time \( t \) in a
Solving the ODE, with the initial condition $z_k(0) = 1$, we get
\[ z_k(t) = e^{-\mu t - \beta \int_0^t y_k(\tau) d\tau}. \]

Moreover, we define
\[ z_k = z_k(T). \]

Let $q$ be the probability that an infected individual is still alive at time $T$, and we get
\[ q = e^{-(\mu + \eta)T}. \]

Let $w_k$ be the probability of a mutant susceptible getting infected during the time interval $[0, T]$, and surviving until time $T$. We get
\[ w_k = \int_0^T z_k(t) e^{-(\mu + \eta)(T-t)} y_k(t) \beta dt. \]

Let $A \coloneqq A(d, D) = [a_{ij}(d, D)]_{i,j=1,2}$ be the projection matrix of the mutant population, i.e.
\[
\begin{bmatrix}
X_{n+1}^{(mut)} \\
Y_{n+1}^{(mut)}
\end{bmatrix}
= 
\begin{bmatrix}
a_{11} & a_{12} \\
a_{21} & a_{22}
\end{bmatrix}
\begin{bmatrix}
X_n^{(mut)} \\
Y_n^{(mut)}
\end{bmatrix}
\]

where
\[
a_{11} = \frac{\alpha F d_{mut} s \hat{X} z_S(T)}{\alpha \hat{x} + \hat{y}} + \frac{\alpha F d_{mut} s \hat{Y} z_I(T)}{\alpha \hat{x} + \hat{y}} + \frac{\alpha \hat{x} S^{(mut)}}{\alpha \hat{x} S^{(mut)} + \hat{y} S^{(mut)} + \hat{y} S^{(mut)}},
\]
\[
a_{12} = \frac{\alpha F (1-p) d_{mut} s \hat{X} z_S(T)}{\alpha \hat{x} + \hat{y}} + \frac{\alpha F (1-p) d_{mut} s \hat{Y} z_I(T)}{\alpha \hat{x} + \hat{y}} + \frac{\alpha \hat{x} I^{(mut)}}{\alpha \hat{x} I^{(mut)} + \hat{y} I^{(mut)} + \hat{y} I^{(mut)}},
\]
\[
a_{21} = \frac{F d_{mut} s \hat{X} w_S}{\alpha \hat{x} + \hat{y}} + \frac{F d_{mut} s \hat{Y} w_I}{\alpha \hat{x} + \hat{y}} + \frac{\alpha \hat{x} I^{(mut)}}{\alpha \hat{x} I^{(mut)} + \hat{y} I^{(mut)} + \hat{y} I^{(mut)}},
\]
\[
a_{22} = \frac{F p \delta_{mut} s \hat{X} q + F (1-p) d_{mut} s \hat{X} w_S}{\alpha \hat{x} + \hat{y}}.
\]
The invasion fitness of a mutant $W$ is given by $W_{d}(d_{\text{mut}}) = \text{tr} A - \det A$, and the mutant has a positive probability of invading the resident population whenever $W_{d}(d_{\text{mut}}) > 1$.

5.3 Pairwise invadability plots and Resident equilibria

In Figures 4, 5, and 6 display Pairwise invadability plots and resident equilibrium plots. In the PIPs the red areas correspond with positive invasion fitness for the mutant. The resident equilibrium plots show how the value of the resident equilibrium changes through different values of the dispersal strategy $d$. The transmission rate $\beta$ was varied through different plots, but the other parameter values are as follows: $p = 0.5$, $s = 0.5$, $\mu = 0.1, \eta = 0.1, \sigma = 0.7$, $\kappa = 0.8$.

In Figure 7 we can see how different values of within-site transmission rate $\beta$ and probability of vertical transmission probability $p$ affect the singular strategy $\hat{d}$. For low values of $p$ and $\beta$, while the probability of surviving dispersal is $s = 0.5$, numerical analysis gives us the Hamilton-May equilibrium, i.e. $\hat{d} = (2 - s)^{-1} = (2 - 0.5)^{-1} = (1.5)^{-1} \approx 0.666$. For high values of $p$ and $\beta$, i.e. when almost every individual is born infected or gets infected during the within-site dynamics, numerical analysis gives us $\hat{d} = 0.757$, which coincides with the Hamilton-May equilibrium with survival probability $s_i$, since

$$\kappa d = d_i = \frac{1}{2 - s_i} = \frac{1}{2 - \sigma s} \Rightarrow 0.8 \cdot d = \frac{1}{2 - 0.7 \cdot 0.5} = \frac{1}{1.65} = 0.606060...$$

$$\Rightarrow d = 0.7575...$$
Figure 4: PIP for $\beta = 1$. When the disease is not infectious enough, there is no positive infected density, and the singular strategy $\hat{d} = 0.666\ldots$ is the Hamilton-May-singularity.

Figure 5: PIP for $\beta = 2.5$. With high enough infectiousness a positive infected density is possible, with $\hat{d} = 0.679\ldots$, but only for high enough values of $d$. 
Figure 6: PIP for $\beta = 5. \hat{d} = 0.706\ldots$

Figure 7: Values of the singular strategy $\hat{d}$ for different values of the probability of vertical transmission $p$ and horizontal transmission rate $\beta$. 

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6 Evolution of two traits

In the previous section we assumed that the dispersal strategies of the susceptible and infected individuals are connected by the coefficient $\kappa$. We now drop this assumption and let the strategies of the susceptible and infected individuals evolve separately.

Let $\bar{x} = (d, \delta) \in [0, 1] \times [0, 1]$ be the dispersal strategy of the resident population, where $d$ is the dispersal strategy of susceptible individuals and $\delta$ is the dispersal strategy of the infected individuals. Moreover the mutant strategy is given by $\bar{x}_{mut} = (d_{mut}, \delta_{mut}) \in [0, 1] \times [0, 1]$.

Even though dispersal strategies of the susceptible and infected individuals are not connected, we can use the same notation as before for the Resident Equilibrium, the Within-site dynamics, with corresponding initial conditions, and the projection matrix of the mutant.

Again, in order to compute the projection matrix of the mutant, and thus the invasion fitness of a mutant, we can use the same notation to construct the projection matrix of the mutant.

Let $A := A(\bar{x}, \bar{x}_{mut}) = A(d, \delta, d_{mut}, \delta_{mut}) = [a_{ij}(d, \delta, d_{mut}, \delta_{mut})]_{i,j=1,2}$ be the projection matrix of the mutant population, i.e.

\[
\begin{bmatrix}
X_{n+1}^{(mut)} \\
Y_{n+1}^{(mut)}
\end{bmatrix} = \begin{bmatrix}
a_{11} & a_{12} \\
a_{21} & a_{22}
\end{bmatrix} \begin{bmatrix}
X_{n}^{(mut)} \\
Y_{n}^{(mut)}
\end{bmatrix}.
\]

The components of the projection matrix are the same as on page 19, keeping in mind that $\delta$ and $\delta_{mut}$ are not connected to $d$ and $d_{mut}$, respectively.

For the invasion fitness we get

\[
W(\bar{x}_{mut}) = W(d, \delta, d_{mut}, \delta_{mut}) = \text{tr} A - \text{det} A,
\]

and the mutant strategy has a positive probability of invasion whenever $W(\bar{x}_{mut}) > 1$.

6.1 Isocline plots

In order to find singular strategies in the strategy space $[0, 1] \times [0, 1]$, we must first compute the following sets:

\[
d_{iso} = \{(d, \delta) \in [0, 1] \times [0, 1] \mid \partial_d W(d, \delta, d, \delta) = 0\} \quad (d\text{-isocline}),
\]

\[
\delta_{iso} = \{(d, \delta) \in [0, 1] \times [0, 1] \mid \partial_\delta W(d, \delta, d, \delta) = 0\} \quad (\delta\text{-isocline}).
\]

A singular strategy is a point $\hat{x} := (\hat{d}, \hat{\delta})$ in the strategy space $[0, 1] \times [0, 1]$
for which $\hat{x} \in d_{iso}$ and $\hat{x} \in \delta_{iso}$, whenever $d_{iso} \cap \delta_{iso} \neq \emptyset$.

Figures 8a, 8b, and 8c show the $d$- and $\delta$-isoclines, and directional arrows of the selection gradient at the respective points. We can see for different parameter values, here we vary $\eta$, singular strategies may or may not exist. The background indicates the value of the resident equilibrium $\hat{Y}$, e.g. black for those points $(d, \delta)$, for which $\hat{Y} = 0$, and white for those points $(d, \delta)$, for which $\hat{Y} = 1$.

![Isocline plots](image)

**Figure 8**: Isocline plots with different values of virulence $\eta$. Parameter values: $p = 0.9, s = 0.1, \sigma = 0.25, \beta = 6, \mu = 0.1, \alpha = 1.5$. Red lines represent $d$-isoclines, and black lines represent $\delta$-isoclines. The background color indicates the value of the resident equilibrium for each point $(d, \delta)$: black if $\hat{Y} = 0$, and white if $\hat{Y} = 1$.

### 6.2 Bifurcation diagrams

In the isocline plots we saw that when $\eta$ is low we have to singularities in the strategy space, and when $\eta$ is high enough the singularities vanish, since
the $d$- and $\delta$-isoclines did not intersect. We will now analyze more closely when the singularities are stable (in the strong convergence and evolutionary sense) when we smoothly vary $\eta$. We use the criterion from Leimar (2009) to determine the stability of each singularity, as presented in section 1.2.4, keeping in mind that when a singularity is strongly convergent stable and evolutionarily unstable it is a branching point only in a two dimensional strategy space (Geritz et al. 2016).

Figure 9: Evolutionary singularities as a function of virulence. (a) and (b) display the strong convergence stability of the singularities for each value of $\eta$: black corresponds to a stable singularity, and red to an unstable singularity. (c) and (d) display the evolutionary stability of the singularities of each value of $\eta$: black corresponds to a stable singularity, and red to an unstable singularity. Parameter values: $\alpha = 1.5$, $p = 0.9$, $s = 0.1$, $\sigma = 0.25$, $\beta = 6$, $\mu = 0.1$. 

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In figures 9a and 9b, for each value of $\eta$, the corresponding singularity is strong convergent stable if black, and unstable if red. Furthermore, in figures 9c and 9d, for each value of $\eta$, the corresponding singularity is evolutionarily stable if black, and unstable if red.

As we see while comparing Figures 9b and 9d we notice a range of $\eta$, for which the lower singularity is stable in the strong convergence sense, but not in the evolutionary sense. This means evolutionary branching is possible near this singularity. To explore what happens after branching, we need to construct the canonical equation.

### 6.3 The canonical equation of the monomorphic population

We use the canonical equation from section 1.2.3, and apply it for this model. Since this model describes a yearly individual, who gives birth and then dies, we can set $T_f = T_s = 1$, and choose $s_x(\bar{x}_{mut}) = \log(R_0(\bar{x}, \bar{x}_{mut}))$, where $R_0$ is the dominant eigenvalue of the Next-Generation Matrix $L(\bar{x}, \bar{x})$. The canonical equation then finally arrives to the form

$$
\frac{d\bar{x}}{d\tau} = \frac{\mu_{mut} M}{B(\bar{x}, \bar{x})} \cdot C \cdot \frac{\partial s_x(\bar{x}_{mut})}{\partial \bar{x}_{mut}} \bigg|_{\bar{x}_{mut} = \bar{x}},
$$

where $\mu_{mut}$ is the probability of mutation, $M$ the number of sites, with $\mu M \to 0$, and the covariance matrix $C$. Furthermore $B(\bar{x}, \bar{x}) = \sum_l u_l \text{Var}(\sum_m v_m \xi_{ml})$, $l, m \in \{S, I\}$, where $u = [u_S \ u_I]^\top$ and $v = [v_S \ v_I]$ are the right and left eigenvectors, respectively, of the leading eigenvalue $R_0$ of the Next-Generation Matrix $L(\bar{x}, \bar{x})$, and $\xi_{ml}$ is a random variable of the number of $m$-state sites from one $l$-state site. The random variables $\xi_{ml}$ comprise of the sum of three random variables, i.e.

$$
\xi_{ml} = \varphi_{ml} + \chi_{ml,S} + \chi_{ml,I},
$$

where $\varphi_{ml}$ is the number of home sites, $\chi_{ml,S}$ the number of $S$-state sites, and $\chi_{ml,I}$ the number of $I$-state sites from one $l$-state site. (Dunrix et al. 2008)

We assume that the covariance matrix is of the form

$$
C = \begin{bmatrix}
1 & c \\
c & 1
\end{bmatrix},
$$

with $c \in [0, 1]$. Here $C_{ij}$ is the covariance of the mutation distribution (Diekmann & Law 1996) of trait values of states $i$ and $j$ ($i, j \in \{S, I\}$). For the diagonal elements we assume the variances to be equal, and thus can be
scaled to 1. Furthermore, we assume that the mutation distribution is such that the off-diagonal can be scaled to $c$, which implies correlation of mutation of the two trait values.

We see here that, since we have non-overlapping generations, the Next-Generation Matrix $L$ is defined exactly like the projection matrix of a mutant, when $\tilde{x}_{mut} = \tilde{x}$, i.e. when $L(\tilde{x}) = A(\tilde{x}, \tilde{x})$

We notice, that $\varphi_{ml} \sim \text{Bernoulli}(p_{ml})$, where $p_{ml}$ is the probability, that offspring born in a $l$-state site, survives the site dynamics, and wins the home site as a $m$-state individual, since

$$\varphi_{ml} = \begin{cases} 1, & \text{with probability } p_{ml} \\ 0, & \text{otherwise} \end{cases}$$

Next we show that $\chi_{ml,i} \sim \text{Poisson}(\lambda_{ml,i})$, $m, l, i \in \{S, I\}$. It is enough to show that $\chi_{SS,S} \sim \text{Poisson}(\lambda_{SS,S})$, since the same type of arguments apply to all other random variables $\chi_{ml,i}$.

The number of offspring that disperse from a $S$-state site to a $S$-state site is $k = Fds\hat{X}$, and the probability that a single offspring wins the site it has arrived to is

$$q = \frac{zS\alpha}{\alpha\hat{x}_S + \hat{y}_S}.$$ 

It follows that now $\chi_{SS,S}$ is binomially distributed, with parameters $k$ and $p$, since at the limit $F \to \infty$, we have $k \to \infty$ and

$$q = \frac{zS\alpha}{\alpha\hat{x}_S + \hat{y}_S} \to 0,$$

since $\alpha\hat{x}_S + \hat{y}_S = O(F)$. Thus $\chi_{SS,S} \sim \text{Poisson}(\lambda_{SS,S})$, where

$$\lambda_{SS,S} = pk = \frac{Fds\hat{X}zS\alpha}{\alpha\hat{x}_S + \hat{y}_S}.$$ 

Below is the list of all parameters of the random variables $\varphi_{ml}$, $\chi_{ml,i}$, $m, l, i \in \{S, I\}$:

$$p_{SS} = \frac{F(1-d)zS\alpha}{\alpha\hat{x}_S + \hat{y}_S}, \quad \lambda_{SS,S} = \frac{Fds\hat{X}zS\alpha}{\alpha\hat{x}_S + \hat{y}_S}, \quad \lambda_{SS,I} = \frac{Fds\hat{Y}zI\alpha}{\alpha\hat{x}_I + \hat{y}_I},$$

$$p_{IS} = \frac{F(1-d)wS}{\alpha\hat{x}_S + \hat{y}_S}, \quad \lambda_{IS,S} = \frac{Fds\hat{X}wS}{\alpha\hat{x}_S + \hat{y}_S}, \quad \lambda_{IS,I} = \frac{Fds\hat{Y}wI}{\alpha\hat{x}_I + \hat{y}_I},$$

$$p_{SI} = \frac{F(1-p)(1-d)zI\alpha}{\alpha\hat{x}_S + \hat{y}_S}, \quad \lambda_{SI,S} = \frac{F(1-p)ds\hat{X}zS\alpha}{\alpha\hat{x}_S + \hat{y}_S}, \quad \lambda_{SI,I} = \frac{F(1-p)ds\hat{Y}zI\alpha}{\alpha\hat{x}_I + \hat{y}_I},$$

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\[
p_{II} = \frac{F[p(1 - \delta)q + (1 - p)(1 - d)w_I]}{\alpha \bar{x}_I + \bar{y}_I}, \quad \lambda_{II,S} = \frac{F\hat{X}[(1 - p)dw_S + p\delta_s q]}{\alpha \bar{x}_S + \bar{y}_S},
\]
and
\[
\lambda_{II,I} = \frac{F\hat{Y}[(1 - p)dw_I + p\delta_s q]}{\alpha \bar{x}_I + \bar{y}_I},
\]
where \(z_k, w_k,\) and \(q\) are as formulated in page 19 for the case of two traits.

Since the function \(B(\bar{x}, \bar{x})\) consists of the variances of a linear combinations of random variables, in order to simplify it, we need to prove which of the random variables are independent, and if not independent, construct the joint distribution in order to evaluate their covariances.

We easily see that for fixed \(m, l\), i.e. \(\xi_{ml} = \varphi_{ml} + \chi_{ml,S} + \chi_{ml,I}\), the random variables \(\varphi_{ml}\) and \(\chi_{ml,S}\) are independent, as are \(\varphi_{ml}\) and \(\chi_{ml,I}\), since the probability of an offspring, that does not disperse, winning the home site is independent of the probability of a dispersing offspring winning a site. Moreover \(\chi_{ml,S}\) and \(\chi_{ml,I}\) are independent, since an offspring that disperse into a \(S\)-state have not effect on the probability of offspring winning in a \(I\)-state site, and vice versa.

Thus, for fixed \(m, l\), we have
\[
\begin{align*}
\text{Cov}(\varphi_{ml}, \chi_{ml,S}) &= 0, \\
\text{Cov}(\varphi_{ml}, \chi_{ml,I}) &= 0, \\
\text{Cov}(\chi_{ml,S}, \chi_{ml,I}) &= 0.
\end{align*}
\]

With similar arguments we can show that \(\varphi_{ij}\) and \(\chi_{kl,m}\) are independent for all \(i, j, k, l, m \in \{S, I\}\), as well as \(\chi_{hi,j}\) and \(\chi_{kl,m}\) for all \(h, i, j, k, l, m \in \{S, I\}\).

We notice that \(\varphi_{SS}\) and \(\varphi_{IS}\) are not independent, and compute the following:
\[
\begin{align*}
\text{Cov}(\varphi_{SS}, \varphi_{IS}) &= E(\varphi_{SS}\varphi_{IS}) - E(\varphi_{SS})E(\varphi_{IS}) \\
&= 0 \cdot 0 \cdot q_{00} + 1 \cdot 0 \cdot q_{10} + 0 \cdot 1 \cdot q_{01} + 1 \cdot 1 \cdot q_{11} - p_{SS}p_{IS} \\
&= -p_{SS}p_{IS},
\end{align*}
\]
where \(q_{ij} = \Pr(\varphi_{SS} = i, \varphi_{IS} = j), i, j \in \{0, 1\}\), with \(q_{11} = 0\), since only one offspring can win any single site.

Similarly \(\varphi_{SI}\) and \(\varphi_{II}\) are not independent, so we compute the following:
\[
\begin{align*}
\text{Cov}(\varphi_{SI}, \varphi_{II}) &= E(\varphi_{SI}\varphi_{II}) - E(\varphi_{SI})E(\varphi_{II}) \\
&= 0 \cdot 0 \cdot q_{00} + 1 \cdot 0 \cdot q_{10} + 0 \cdot 1 \cdot q_{01} + 1 \cdot 1 \cdot q_{11} - p_{SI}p_{II}
\end{align*}
\]
where \( q_{ij} = \Pr(\varphi_{SI} = i, \varphi_{II} = j) \), \( i,j \in \{0,1\} \), with \( q_{11} = 0 \), since only one offspring can win any single site.

Finally we can simplify \( B(\bar{x}, \bar{x}) \):

\[
B(\bar{x}, \bar{x}) = \sum_{l \in \{S, I\}} u_l \Var(\sum_{m \in \{S, I\}} v_m \xi_{ml}) \\
= u_S \Var(v_S \xi_{SS} + v_I \xi_{IS}) + u_I \Var(v_S \xi_{SI} + v_I \xi_{II}) \\
= u_S \Var(v_S \varphi_{SS} + v_S \chi_{SS,I} + v_I \varphi_{IS} + v_I \chi_{IS,I} + v_I \chi_{IS,I}) \\
+ u_I \Var(v_S \varphi_{SI} + v_S \chi_{SI,I} + v_I \varphi_{II} + v_I \chi_{II,I} + v_I \chi_{II,I}) \\
= u_S \{v_S^2 [\Var(\varphi_{SS}) + \Var(\chi_{SS,I}) + \Var(\chi_{IS,I})] \\
+ v_I^2 [\Var(\varphi_{SS}) + \Var(\chi_{SS,I}) + \Var(\chi_{IS,I})] \\
+ 2v_S^2 [\Cov(\varphi_{SS}, \chi_{SS,I}) + \Cov(\varphi_{SS}, \chi_{SS,I}) + \Cov(\chi_{SS,I}, \chi_{SS,I})] \\
+ 2v_I^2 [\Cov(\varphi_{SS}, \chi_{SS,I}) + \Cov(\chi_{SS,I}, \chi_{SS,I}) + \Cov(\chi_{SS,I}, \chi_{SS,I})] \\
+ 2v_S v_I [\Cov(\varphi_{SS}, \varphi_{IS} + \Cov(\varphi_{SS}, \chi_{IS,I}) + \Cov(\varphi_{IS}, \chi_{IS,I}) \\
+ \Cov(\chi_{SS,I}, \varphi_{IS} + \Cov(\chi_{SS,I}, \chi_{IS,I} + \Cov(\chi_{IS,I}, \chi_{IS,I}) \\
+ \Cov(\chi_{SS,I}, \varphi_{II} + \Cov(\chi_{SS,I}, \chi_{II,I} + \Cov(\chi_{II,I}, \chi_{II,I}) \\
+ \Cov(\chi_{SS,I}, \varphi_{II} + \Cov(\chi_{SS,I}, \chi_{II,I} + \Cov(\chi_{II,I}, \chi_{II,I}) \} \\
\}
\]

+ u_I \{v_S^2 [\Var(\varphi_{SI}) + \Var(\chi_{SI,I}) + \Var(\chi_{SI,I})] \\
+ v_I^2 [\Var(\varphi_{SI}) + \Var(\chi_{SI,I}) + \Var(\chi_{SI,I})] \\
+ 2v_S v_I [\Cov(\varphi_{SS}, \varphi_{IS}) + \Cov(\chi_{SS,I}, \chi_{IS,I}) \\
+ \Cov(\chi_{SS,I}, \varphi_{IS} + \Cov(\chi_{SS,I}, \chi_{IS,I}) + \Cov(\chi_{IS,I}, \chi_{IS,I}) \\
+ \Cov(\chi_{SS,I}, \varphi_{II} + \Cov(\chi_{SS,I}, \chi_{II,I} + \Cov(\chi_{II,I}, \chi_{II,I}) \\
+ \Cov(\chi_{SS,I}, \varphi_{II} + \Cov(\chi_{SS,I}, \chi_{II,I} + \Cov(\chi_{II,I}, \chi_{II,I}) \} \\
\}
\]

= u_S \{v_S^2 [p_{SS}(1 - p_{SS}) + \lambda_{SS,S} + \lambda_{SS,I}] \\
+ v_I^2 [p_{IS}(1 - p_{IS}) + \lambda_{IS,S} + \lambda_{IS,I} - 2v_S v_I p_{SS} p_{IS}] \\
+ u_I \{v_S^2 [p_{SI}(1 - p_{SI}) + \lambda_{SI,S} + \lambda_{SI,I}] \\
+ v_I^2 [p_{II}(1 - p_{II}) + \lambda_{II,S} + \lambda_{II,I} - 2v_S v_I p_{SI} p_{II}] \}.
\]
In figures 10a, 10b, 10c, and 10d show the trajectories in the strategy space given by integrating canonical equation in evolutionary time for a monomorphic population. The initial conditions and the value of the virulence $\eta$ vary. The parameter values, that do not vary, are as follow: $c = 0.25$, $\alpha = 1.5$, $p = 0.9$, $s = 0.1$, $\sigma = 0.25$, $\beta = 6$, $\mu = 0.1$.

Figures 10a and 10b (with $\eta = 0$ and $\eta = 0.2$, respectively) show that, given the initial condition $\bar{x}_0 = [0.6 \ 0.4]^T$ the trajectories converge to the attracting singularity, which is strongly convergent stable.

Figures 10c and 10d (with $\bar{x}_0 = [0.6 \ 0.4]^T$ and $\bar{x}_0 = [0.9 \ 0.5]^T$, respectively) show that with high enough $\eta$, the trajectories follow the $d$-isocline in figure 8c at page 24.

Figure 10: Trajectories of the canonical equation for different initial conditions and values of $\eta$. Parameter values: $c = 0.25$, $\alpha = 1.5$, $p = 0.9$, $s = 0.1$, $\sigma = 0.25$, $\beta = 6$, $\mu = 0.1$. 
7 The dimorphic population

Since we were able to show, with certain parameter values, that evolutionary branching is possible, we need to construct and formulate the model to accommodate a polymorphic population of residents. Geritz et al. (2016) showed that "generically, expanding polymorphisms around evolutionarily singular strategies initially evolve towards becoming dimorphisms". Thus, at the evolutionary time scale we can assume that in this model a monomorphic population branches into a dimorphism.

Again we follow the same process of yearly behavior as before: single individuals produce $F$ offspring, that either disperse or stay at the home site, after dispersal go through a period of time in which contraction of the disease within the site is possible, and finally the winner of the site is chosen by weighted lottery, susceptibles assumed to have an advantage in competition via the constant $\alpha$.

Consider now two established residents with dispersal strategies $\vec{x}_1 = (d_1, \delta_1)$ and $\vec{x}_2 = (d_2, \delta_2)$, with $\vec{x}_1, \vec{x}_2 \in [0, 1] \times [0, 1]$. Let $X_{1,n}$ and $Y_{1,n}$ be the fraction of susceptible and infected sites, respectively, of the first resident in year $n \in \mathbb{Z}_+$, and $X_{2,n}$ and $Y_{2,n}$ be the fraction of susceptible and infected sites, respectively, of the second resident in year $n \in \mathbb{Z}_+$. Moreover, we have that $X_{1,n} + Y_{1,n} + X_{2,n} + Y_{2,n} = 1$ must hold.

To simplify notation and terminology we say that a site is a $S_1$-state site, if it is occupied by a susceptible resident of the first kind, $S_2$-state site, if it is occupied by a susceptible resident of the second kind, $I_1$-state site, if it is occupied by an infected resident of the first kind, and $I_2$-state site, if it is occupied by an infected resident of the second kind.

7.1 The within-site dynamics of the dimorphic population

As before the within-site dynamics for a resident only population is described by the following ODE system:

\begin{equation}
\begin{aligned}
\dot{x}^{(1)}_k &= -\beta x^{(1)}_k (y^{(1)}_k + y^{(2)}_k) - \mu x^{(1)}_k , \\
\dot{y}^{(1)}_k &= \beta x^{(1)}_k (y^{(1)}_k + y^{(2)}_k) - (\mu + \eta) y^{(1)}_k , \\
\dot{x}^{(2)}_k &= -\beta x^{(2)}_k (y^{(1)}_k + y^{(2)}_k) - \mu x^{(2)}_k , \\
\dot{y}^{(2)}_k &= \beta x^{(2)}_k (y^{(1)}_k + y^{(2)}_k) - (\mu + \eta) y^{(2)}_k ,
\end{aligned}
\end{equation}

where $x^{(1)}_k$ and $y^{(1)}_k$ are the numbers of susceptible and infected individuals, respectively, of the first resident, and $x^{(2)}_k$ and $y^{(2)}_k$ are the numbers of suscep-
tible and infected individuals, respectively, of the second resident in a \(k\)-state site.

Furthermore, the initial conditions of the ODEs depend on which site we are looking at.

For a \(S_1\)-state site, the initial conditions are:

\[
\begin{align*}
\begin{cases}
x^{(1)}_{0,S_1} &= F[(1 - d_1) + X_{1,n}d_1s + Y_{1,n}(1 - p)d_1s], \\
y^{(1)}_{0,S_1} &= FY_{1,n}p\delta_1s_i, \\
x^{(2)}_{0,S_1} &= F[X_{2,n}d_2s + Y_{2,n}(1 - p)d_2s], \\
y^{(2)}_{0,S_1} &= FY_{2,n}p\delta_2s_i.
\end{cases}
\]

(7.2)

For a \(I_1\)-state site, the initial conditions are:

\[
\begin{align*}
\begin{cases}
x^{(1)}_{0,I_1} &= F[(1 - p)(1 - d_1) + X_{1,n}d_1s + Y_{1,n}(1 - p)d_1s], \\
y^{(1)}_{0,I_1} &= F[p(1 - \delta_1) + Y_{1,n}p\delta_1s_i], \\
x^{(2)}_{0,I_1} &= F[X_{2,n}d_2s + Y_{2,n}(1 - p)d_2s], \\
y^{(2)}_{0,I_1} &= FY_{2,n}p\delta_2s_i.
\end{cases}
\]

(7.3)

For a \(S_2\)-state site, the initial conditions are:

\[
\begin{align*}
\begin{cases}
x^{(1)}_{0,S_2} &= F[X_{1,n}d_1s + Y_{1,n}(1 - p)d_1s], \\
y^{(1)}_{0,S_2} &= FY_{1,n}p\delta_1s_i, \\
x^{(2)}_{0,S_2} &= F[1 - d_2 + X_{2,n}d_2s + Y_{2,n}(1 - p)d_2s], \\
y^{(2)}_{0,S_2} &= FY_{2,n}p\delta_2s_i.
\end{cases}
\]

(7.4)

For a \(I_2\)-state site, the initial conditions are:

\[
\begin{align*}
\begin{cases}
x^{(1)}_{0,I_2} &= F[X_{1,n}d_1s + Y_{1,n}(1 - p)d_1s], \\
y^{(1)}_{0,I_2} &= FY_{1,n}p\delta_1s_i, \\
x^{(2)}_{0,I_2} &= F[(1 - p)(1 - d_2) + X_{2,n}d_2s + Y_{2,n}(1 - p)d_2s], \\
y^{(2)}_{0,I_2} &= F[p(1 - \delta_2) + Y_{2,n}p\delta_2s_i].
\end{cases}
\]

(7.5)

Let \(t \mapsto (x^{(1)}_k(t), y^{(1)}_k(t), x^{(2)}_k(t), y^{(2)}_k(t))\) be a solution of the ODE system of a \(k\)-state site, \(k \in \{S_1, I_1, S_2, I_2\}\), with initial conditions

\[
\begin{align*}
\begin{cases}
x^{(1)}_k(0) &= x^{(1)}_{0,k}, \\
y^{(1)}_k(0) &= y^{(1)}_{0,k}, \\
x^{(2)}_k(0) &= x^{(2)}_{0,k}, \\
y^{(2)}_k(0) &= y^{(2)}_{0,k}.
\end{cases}
\]

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and define
\[
\begin{aligned}
&\begin{cases}
\hat{x}_k^{(1)} := x_k^{(1)}(T), \\
\hat{y}_k^{(1)} := y_k^{(1)}(T), \\
\hat{x}_k^{(2)} := x_k^{(2)}(T), \\
\hat{y}_k^{(2)} := y_k^{(2)}(T).
\end{cases}
\end{aligned}
\]

7.2 The resident equilibrium of the dimorphic population

Now we are able to compute \(X_{1,n+1}, Y_{1,n+1}, X_{2,n+1},\) and \(Y_{2,n+1}:\)

\[
X_{1,n+1} = X_{1,n} \frac{\alpha \hat{x}_1^{(1)}}{\alpha(\hat{x}_1^{(1)} + \hat{x}_1^{(2)}) + \hat{y}_1^{(1)} + \hat{y}_1^{(2)}} + Y_{1,n} \frac{\alpha \hat{x}_1^{(1)}}{\alpha(\hat{x}_1^{(1)} + \hat{x}_1^{(2)}) + \hat{y}_1^{(1)} + \hat{y}_1^{(2)}} + X_{2,n} \frac{\alpha \hat{x}_2^{(1)}}{\alpha(\hat{x}_2^{(1)} + \hat{x}_2^{(2)}) + \hat{y}_2^{(1)} + \hat{y}_2^{(2)}} + Y_{2,n} \frac{\alpha \hat{x}_2^{(1)}}{\alpha(\hat{x}_2^{(1)} + \hat{x}_2^{(2)}) + \hat{y}_2^{(1)} + \hat{y}_2^{(2)}}
\]

\[
Y_{1,n+1} = X_{1,n} \frac{\gamma^{(1)}}{\gamma(\hat{x}_1^{(1)} + \hat{x}_1^{(2)}) + \hat{y}_1^{(1)} + \hat{y}_1^{(2)}} + Y_{1,n} \frac{\gamma^{(1)}}{\gamma(\hat{x}_1^{(1)} + \hat{x}_1^{(2)}) + \hat{y}_1^{(1)} + \hat{y}_1^{(2)}} + X_{2,n} \frac{\gamma^{(1)}}{\gamma(\hat{x}_2^{(1)} + \hat{x}_2^{(2)}) + \hat{y}_2^{(1)} + \hat{y}_2^{(2)}} + Y_{2,n} \frac{\gamma^{(1)}}{\gamma(\hat{x}_2^{(1)} + \hat{x}_2^{(2)}) + \hat{y}_2^{(1)} + \hat{y}_2^{(2)}}
\]

\[
X_{2,n+1} = X_{1,n} \frac{\alpha \hat{x}_2^{(2)}}{\alpha(\hat{x}_1^{(1)} + \hat{x}_2^{(2)}) + \hat{y}_1^{(1)} + \hat{y}_1^{(2)}} + Y_{1,n} \frac{\alpha \hat{x}_2^{(2)}}{\alpha(\hat{x}_1^{(1)} + \hat{x}_2^{(2)}) + \hat{y}_1^{(1)} + \hat{y}_1^{(2)}} + X_{2,n} \frac{\alpha \hat{x}_2^{(2)}}{\alpha(\hat{x}_2^{(1)} + \hat{x}_2^{(2)}) + \hat{y}_2^{(1)} + \hat{y}_2^{(2)}} + Y_{2,n} \frac{\alpha \hat{x}_2^{(2)}}{\alpha(\hat{x}_2^{(1)} + \hat{x}_2^{(2)}) + \hat{y}_2^{(1)} + \hat{y}_2^{(2)}}
\]

with \(Y_{2,n} = 1 - X_{1,n} - Y_{1,n} - X_{2,n},\) and we will use a more convenient notation

\[
(X_{1,n+1}, Y_{1,n+1}, X_{2,n+1}) = G(X_{1,n}, Y_{1,n}, X_{2,n}).
\]

Thus the stable resident equilibrium \((\hat{X}_1, \hat{Y}_1, \hat{X}_2, \hat{Y}_2)\) is found by solving

\[
(\hat{X}_1, \hat{Y}_1, \hat{X}_2) = G(\hat{X}_1, \hat{Y}_1, \hat{X}_2).
\]
7.3 The projection matrix of an invading mutant in the dimorphic population

We assume that the resident population has reached an equilibrium, where the density of infected sites is positive, and consider now a rare mutant entering the population, with dispersal strategy $\bar{x}_{\text{mut}} = (d_{\text{mut}}, \delta_{\text{mut}}) \in [0, 1] \times [0, 1]$. In sites that were occupied by a mutant susceptible or infected individual, $S_{\text{mut}}$-state or $I_{\text{mut}}$-state sites, respectively, the within-site dynamics is described by the following ODE system:

\[
\begin{align*}
&\dot{x}_k^{(1)} = -\beta x_k^{(1)} \left[ y_k^{(1)} + y_k^{(2)} + y_k^{(\text{mut})} \right] - \mu x_k^{(1)}, \\
&\dot{y}_k^{(1)} = \beta x_k^{(1)} \left[ y_k^{(1)} + y_k^{(2)} + y_k^{(\text{mut})} \right] - (\mu + \eta) y_k^{(1)}, \\
&\dot{x}_k^{(2)} = -\beta x_k^{(2)} \left[ y_k^{(1)} + y_k^{(2)} + y_k^{(\text{mut})} \right] - \mu x_k^{(2)}, \\
&\dot{y}_k^{(2)} = \beta x_k^{(2)} \left[ y_k^{(1)} + y_k^{(2)} + y_k^{(\text{mut})} \right] - (\mu + \eta) y_k^{(2)}, \\
&\dot{x}_k^{(\text{mut})} = -\beta x_k^{(\text{mut})} \left[ y_k^{(1)} + y_k^{(2)} + y_k^{(\text{mut})} \right] - \mu x_k^{(\text{mut})}, \\
&\dot{y}_k^{(\text{mut})} = \beta x_k^{(\text{mut})} \left[ y_k^{(1)} + y_k^{(2)} + y_k^{(\text{mut})} \right] - (\mu + \eta) y_k^{(\text{mut})},
\end{align*}
\]

$k \in \{S_{\text{mut}}, I_{\text{mut}}\}$, where $x_k^{(1)}$, $y_k^{(1)}$, $x_k^{(2)}$, and $y_k^{(2)}$ are the number of resident susceptible and infected, as before, and $x_k^{(\text{mut})}$ and $y_k^{(\text{mut})}$ are the number of mutant susceptible and infected, respectively.

The initial conditions of $S_1$, $I_1$, $S_2$, and $I_2$ sites are similar to what was as before, with corresponding solutions of each system, with the only difference being the converting $X_{1,n}$ to $X_1$, $Y_{1,n}$ to $Y_1$, etc. Moreover, the initial conditions of the resident sites exclude mutant dispersers, since mutant individuals do not affect the within-site dynamics of resident sites, but are affected by it.

Since the mutant is rare, we assume that dispersal from a $S_{\text{mut}}$-site to a $I_{\text{mut}}$, and to itself, is negligible, as is dispersal from a $I_{\text{mut}}$-site to a $S_{\text{mut}}$, and to itself, is negligible. Furthermore, the initial conditions of a $S_{\text{mut}}$-site and a $I_{\text{mut}}$ are

\[
\begin{align*}
&x_{0,S_{\text{mut}}}^{(1)} = F[\hat{X}_1 d_1 s + \hat{Y}_1 (1 - p) d_1 s], \\
y_{0,S_{\text{mut}}}^{(1)} = F\hat{Y}_1 p \delta_1 s_1, \\
x_{0,S_{\text{mut}}}^{(2)} = F[\hat{X}_2 d_2 s + \hat{Y}_2 (1 - p) d_2 s], \\
y_{0,S_{\text{mut}}}^{(2)} = F\hat{Y}_2 p \delta_2 s_1, \\
x_{0,S_{\text{mut}}}^{(\text{mut})} = F(1 - d_{\text{mut}}) \\
y_{0,S_{\text{mut}}}^{(\text{mut})} = 0,
\end{align*}
\]

\text{(7.7)}
and

\[
\begin{aligned}
    x_{0, I_{\text{mut}}}^{(1)} &= F[\hat{X}_1 d_1 s + \hat{Y}_1 (1 - p) d_1 s], \\
    y_{0, I_{\text{mut}}}^{(1)} &= F\hat{Y}_1 p \delta_1 s, \\
    x_{0, I_{\text{mut}}}^{(2)} &= F[\hat{X}_2 d_2 s + \hat{Y}_2 (1 - p) d_2 s], \\
    y_{0, I_{\text{mut}}}^{(2)} &= F\hat{Y}_2 p \delta_2 s, \\
    x_{0, I_{\text{mut}}}^{(\text{mut})} &= F(1 - p)(1 - d_{\text{mut}}), \\
    y_{0, I_{\text{mut}}}^{(\text{mut})} &= Fp(1 - \delta_{\text{mut}}),
\end{aligned}
\] (7.8)

respectively.

Let \( t \mapsto (x_k^{(1)}(t), y_k^{(1)}(t), x_k^{(2)}(t), y_k^{(2)}(t), x_k^{(\text{mut})}(t), y_k^{(\text{mut})}(t)) \) be a solution of the within-site dynamics of a \( k \)-state site, \( k \in \{S_{\text{mut}}, I_{\text{mut}}\} \), with the corresponding initial condition, and define

\[
\begin{aligned}
    \tilde{x}_k^{(1)} &:= x_k^{(1)}(T), \\
    \tilde{y}_k^{(1)} &:= y_k^{(1)}(T), \\
    \tilde{x}_k^{(2)} &:= x_k^{(2)}(T), \\
    \tilde{y}_k^{(2)} &:= y_k^{(2)}(T), \\
    \tilde{x}_k^{(\text{mut})} &:= x_k^{(\text{mut})}(T), \\
    \tilde{y}_k^{(\text{mut})} &:= y_k^{(\text{mut})}(T).
\end{aligned}
\]

For a dispersing mutant offspring, since they do not have effect on the dynamics of within-site dynamics of a resident site but are affected by it, i.e., as in the monomorphic dynamics, we need to construct the probabilities of surviving the dynamics of each resident site.

Let \( z_k(t) \) be the probability of a mutant resident of being alive at time \( t \) in a \( k \)-state site, \( k \in \{S_1, I_1, S_2, I_2\} \). We get

\[
\dot{z}_k(t) = -z_k(t)(\mu + \beta(y_k^{(1)}(t) + y_k^{(2)}(t))),
\]

where \( y_k^{(1)}(t) \) and \( y_k^{(2)}(t) \) are the number of infected individuals of resident 1 and 2, respectively, at time \( t \in [0, T] \), in a \( k \)-state site. Solving this ODE with initial conditions \( z(0) = 1 \) we get

\[
z_k(t) = e^{-\mu t - \beta \int_0^t (y_k^{(1)}(\tau) + y_k^{(2)}(\tau)) d\tau},
\]

and define

\[
z_k := z_k(T),
\] (7.9)
where \( z_k \) is now the probability of a susceptible individual on still being alive and not infected at time \( T \) in a \( k \)-state site.

Let \( w_k \) be the probability that a susceptible individual becomes infected at some time \( t \in [0, T] \), and surviving until time \( t = T \) as an infected individual in a \( k \)-state site, \( k \in \{ S_1, I_1, S_2, I_2 \} \). We get

\[
(7.10) \quad w_k = \int_0^T z_k(t)e^{-(\mu + \eta)(T-t)}(y_k^{(1)}(t) + y_k^{(2)}(t))\beta dt.
\]

Finally let \( q \) be the probability of an infected individual surviving until competition. We get \( q = e^{-(\mu + \eta)T} \).

We are now able to formulate the projection matrix \( A := [a_{ij}(\bar{x}_1, \bar{x}_2, \bar{x}_{\text{mut}})]_{i,j=1,2} \), for which

\[
(7.11) \quad \begin{bmatrix} X_n^{(\text{mut})} \\ Y_n^{(\text{mut})} \end{bmatrix} = \begin{bmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{bmatrix} \begin{bmatrix} X_n^{(\text{mut})} \\ Y_n^{(\text{mut})} \end{bmatrix}.
\]

The components \( a_{ij} \) are as follows:

\[
a_{11} = \frac{Fd_{\text{mut}}s\hat{X}_1z_{S_1}^2}{\alpha[\hat{x}_1^{(1)} + \hat{x}_2^{(2)} + \gamma_{S_1}] + \hat{y}_{S_1} + \gamma_{S_1}^2} + \frac{Fd_{\text{mut}}s\hat{Y}_1z_{I_1}^2}{\alpha[\hat{x}_2^{(1)} + \hat{x}_2^{(2)} + \gamma_{I_2}] + \hat{y}_{I_2} + \gamma_{I_2}^2},
\]

\[
a_{12} = \frac{F(1-p)d_{\text{mut}}s\hat{X}_1z_{S_2}^2}{\alpha[\hat{x}_1^{(1)} + \hat{x}_2^{(2)} + \gamma_{S_1}] + \hat{y}_{S_1} + \gamma_{S_1}^2} + \frac{F(1-p)d_{\text{mut}}s\hat{Y}_1z_{I_2}^2}{\alpha[\hat{x}_2^{(1)} + \hat{x}_2^{(2)} + \gamma_{I_2}] + \hat{y}_{I_2} + \gamma_{I_2}^2},
\]

\[
a_{21} = \frac{Fd_{\text{mut}}s\hat{X}_1w_{S_1}}{\alpha[\hat{x}_1^{(1)} + \hat{x}_2^{(2)} + \gamma_{S_1}] + \hat{y}_{S_1} + \gamma_{S_1}^2} + \frac{Fd_{\text{mut}}s\hat{Y}_1w_{I_1}}{\alpha[\hat{x}_2^{(1)} + \hat{x}_2^{(2)} + \gamma_{I_2}] + \hat{y}_{I_2} + \gamma_{I_2}^2},
\]

\[
a_{22} = \frac{Fd_{\text{mut}}s\hat{X}_2w_{S_2}}{\alpha[\hat{x}_1^{(1)} + \hat{x}_2^{(2)} + \gamma_{S_1}] + \hat{y}_{S_1} + \gamma_{S_1}^2} + \frac{Fd_{\text{mut}}s\hat{Y}_2w_{I_2}}{\alpha[\hat{x}_2^{(1)} + \hat{x}_2^{(2)} + \gamma_{I_2}] + \hat{y}_{I_2} + \gamma_{I_2}^2}.
\]
\[ a_{22} \equiv F \frac{\tau_2 H_2 \left[ p \delta_{\text{mut}} x_1 q + (1 - p) d_{\text{mut}} sw_1 \right]}{\alpha \left[ x_{1}^{(1)} + x_{1}^{(2)} + y_{1}^{(1)} + y_{1}^{(2)} \right]} + F \frac{\tau_2 H_2 \left[ p \delta_{\text{mut}} x_2 q + (1 - p) d_{\text{mut}} sw_2 \right]}{\alpha \left[ x_{2}^{(1)} + x_{2}^{(2)} + y_{2}^{(1)} + y_{2}^{(2)} \right]} + \frac{\tau_2 H_2 \left[ p \delta_{\text{mut}} x_1 q + (1 - p) d_{\text{mut}} sw_1 \right]}{\alpha \left[ x_{1}^{(1)} + x_{1}^{(2)} + y_{1}^{(1)} + y_{1}^{(2)} \right]}, \quad j \in \{1, 2\} \]

7.4 The canonical equation for the dimorphic population

The canonical equation for a dimorphic population is

\[ \frac{d\bar{x}}{d\tau} = \frac{\mu_{\text{mut}} M(\bar{X}_j + \bar{Y}_j)}{B(\bar{x}_j, \bar{x}_1, \bar{x}_2)} \cdot C \cdot \frac{\partial \log(R_0(\bar{x}_1, \bar{x}_2, \bar{x}_{\text{mut}}))}{\partial \bar{x}_{\text{mut}}} \bigg|_{\bar{x}_{\text{mut}} = \bar{x}_j}, \quad j \in \{1, 2\} \]

where \( \mu_{\text{mut}} \) is the probability of mutation, \( M \) the number of sites, with \( \mu M \to 0 \), and the covariance matrix \( C \) is

\[ C = \begin{bmatrix} 1 & c \\ c & 1 \end{bmatrix}, \]

with \( c \in [0, 1] \), using the same arguments as in the monomorphic case. Furthermore \( B(\bar{x}_j, \bar{x}_1, \bar{x}_2) = \sum_l u_l \text{Var}(\sum_m v_m \xi_{ml}) \), \( l, m \in \{S_j, I_j\} \), where \( u = \begin{bmatrix} u_{S_j} & u_{I_j} \end{bmatrix}^T \) and \( v = \begin{bmatrix} v_{S_j} & v_{I_j} \end{bmatrix} \) are the right and left eigenvectors, respectively, of the leading eigenvalue \( R_0 \) of the Next-Generation Matrix \( L(\bar{x}_j, \bar{x}_1, \bar{x}_2) \), and \( \xi_{ml} \) is a random variable of the number of \( m \)-state sites from one \( l \)-state site. (Durinx. et al. 2008)

As in the monomorphic case, \( L \) is the same as the projection matrix of the mutant, since we have a discrete time model with non-overlapping generations. Thus \( L(\bar{x}_j, \bar{x}_1, \bar{x}_2) = A(\bar{x}_1, \bar{x}_2, \bar{x}_j) \).

The offspring of a \( l \) state parent either stay at the home site or disperse to sites of all states, and win the sites as a \( m \)-state individual. Thus the random variable \( \xi_{ml} \) is comprised of the sum of five random variables, i.e.

\[ \xi_{ml} = \varphi_{ml} + X_{ml,S_1} + X_{ml,I_1} + X_{ml,S_2} + X_{ml,I_2}, \]
\[ p_{s_j s_j} = \frac{F(1 - d_j) z_j}{\alpha [\tilde{z}_{s_j}^{(1)} + \tilde{z}_{s_j}^{(2)}] + \tilde{y}_{s_j}^{(1)} + \tilde{y}_{s_j}^{(2)}}; \]

\[ \lambda_{s_j s_i, s_i} = \frac{F d_j \hat{X}_1 z_i}{\alpha [\tilde{z}_{s_i}^{(1)} + \tilde{z}_{s_i}^{(2)}] + \tilde{y}_{s_i}^{(1)} + \tilde{y}_{s_i}^{(2)}}, \lambda_{s_j s_i, i_1} = \frac{F d_j \hat{X}_1 z_i}{\alpha [\tilde{z}_{i_1}^{(1)} + \tilde{z}_{i_1}^{(2)}] + \tilde{y}_{i_1}^{(1)} + \tilde{y}_{i_1}^{(2)}}, \]

\[ \lambda_{s_j s_i, s_2} = \frac{F d_j \hat{X}_2 z_{s_2}}{\alpha [\tilde{z}_{s_2}^{(1)} + \tilde{z}_{s_2}^{(2)}] + \tilde{y}_{s_2}^{(1)} + \tilde{y}_{s_2}^{(2)}}, \lambda_{s_j s_i, i_2} = \frac{F d_j \hat{X}_2 z_{i_2}}{\alpha [\tilde{z}_{i_2}^{(1)} + \tilde{z}_{i_2}^{(2)}] + \tilde{y}_{i_2}^{(1)} + \tilde{y}_{i_2}^{(2)}}, \]

\[ p_{l_j s_j} = \frac{F(1 - p)(1 - d_j) z_i}{\alpha [\tilde{z}_{s_j}^{(1)} + \tilde{z}_{s_j}^{(2)}] + \tilde{y}_{s_j}^{(1)} + \tilde{y}_{s_j}^{(2)}}; \]

\[ \lambda_{l_j s_i, s_i} = \frac{F d_j s \hat{X}_1 z_i}{\alpha [\tilde{z}_{s_i}^{(1)} + \tilde{z}_{s_i}^{(2)}] + \tilde{y}_{s_i}^{(1)} + \tilde{y}_{s_i}^{(2)}}, \lambda_{l_j s_i, i_1} = \frac{F d_j s \hat{X}_1 z_i}{\alpha [\tilde{z}_{i_1}^{(1)} + \tilde{z}_{i_1}^{(2)}] + \tilde{y}_{i_1}^{(1)} + \tilde{y}_{i_1}^{(2)}}, \]

\[ \lambda_{l_j s_i, s_2} = \frac{F d_j s \hat{X}_2 z_{s_2}}{\alpha [\tilde{z}_{s_2}^{(1)} + \tilde{z}_{s_2}^{(2)}] + \tilde{y}_{s_2}^{(1)} + \tilde{y}_{s_2}^{(2)}}, \lambda_{l_j s_i, i_2} = \frac{F d_j s \hat{X}_2 z_{i_2}}{\alpha [\tilde{z}_{i_2}^{(1)} + \tilde{z}_{i_2}^{(2)}] + \tilde{y}_{i_2}^{(1)} + \tilde{y}_{i_2}^{(2)}}, \]

\[ p_{l_j i_j} = \frac{F[(1 - p)(1 - d_j) w_{i_1} + p(1 - \delta_j) q]}{\alpha [\tilde{z}_{i_j}^{(1)} + \tilde{z}_{i_j}^{(2)}] + \tilde{y}_{i_j}^{(1)} + \tilde{y}_{i_j}^{(2)}}; \]

\[ \lambda_{l_j i_j, s_1} = \frac{F \hat{X}_1 [(1 - p)d_j sw_{s_1} + p \delta_j s_1 q]}{\alpha [\tilde{z}_{s_1}^{(1)} + \tilde{z}_{s_1}^{(2)}] + \tilde{y}_{s_1}^{(1)} + \tilde{y}_{s_1}^{(2)}}, \lambda_{l_j i_j, i_1} = \frac{F \hat{X}_1 [(1 - p)d_j sw_{i_1} + p \delta_j s_1 q]}{\alpha [\tilde{z}_{i_1}^{(1)} + \tilde{z}_{i_1}^{(2)}] + \tilde{y}_{i_1}^{(1)} + \tilde{y}_{i_1}^{(2)}}, \]

\[ \lambda_{l_j i_j, s_2} = \frac{F \hat{X}_2 [(1 - p)d_j sw_{s_2} + p \delta_j s_1 q]}{\alpha [\tilde{z}_{s_2}^{(1)} + \tilde{z}_{s_2}^{(2)}] + \tilde{y}_{s_2}^{(1)} + \tilde{y}_{s_2}^{(2)}}, \lambda_{l_j i_j, i_2} = \frac{F \hat{X}_2 [(1 - p)d_j sw_{i_2} + p \delta_j s_1 q]}{\alpha [\tilde{z}_{i_2}^{(1)} + \tilde{z}_{i_2}^{(2)}] + \tilde{y}_{i_2}^{(1)} + \tilde{y}_{i_2}^{(2)}}. \]
where \( z_k, q, \) and \( w_k \) are as formulated on page 35. Furthermore, \( \varphi_{ml} \) and \( \lambda_{ml,k}, m, l \in \{S_j, I_j\}, k \in \{S_1, I_1, S_2, I_2\} \), are independent random variables, since the probability of winning the home site is independent from the probability of a dispersing offspring winning a foreign site. And lastly, \( \lambda_{S_is_j,k} \) and \( \lambda_{I_is_j,k} \), as well as \( \lambda_{S_is_j,k} \) and \( \lambda_{I_is_j,k} \), are independent for all \( k \in \{S_1, I_1, S_2, I_2\} \).

The random variables \( \varphi_{S_is_j} \) and \( \varphi_{I_is_j} \) are not independent, since if a susceptible individual wins the home site, an infected cannot. Thus we compute

\[
\text{Cov}(\varphi_{S_is_j}, \varphi_{I_is_j}) = E(\varphi_{S_is_j} \varphi_{I_is_j}) - E(\varphi_{S_is_j})E(\varphi_{I_is_j})
= 0 \cdot 0 \cdot q_{00} + 1 \cdot 0 \cdot q_{10} + 0 \cdot 1 \cdot q_{01} + 1 \cdot 1 \cdot q_{11} - p_{S_is_j}p_{I_is_j}
= -p_{S_is_j}p_{I_is_j},
\]

where \( q_{k_1k_2} = \text{Pr}(\varphi_{S_is_j} = k_1, \varphi_{I_is_j} = k_2), k_1, k_2 \in \{0, 1\}, \) with \( q_{11} = 0, \) since only one offspring can win any single site.

Also the random variables \( \varphi_{S_iI_1} \) and \( \varphi_{I_iI_1} \) are not independent, since if a susceptible individual wins the home site, an infected cannot. Thus we compute

\[
\text{Cov}(\varphi_{S_iI_1}, \varphi_{I_iI_1}) = E(\varphi_{S_iI_1} \varphi_{I_iI_1}) - E(\varphi_{S_iI_1})E(\varphi_{I_iI_1})
= 0 \cdot 0 \cdot q_{00} + 1 \cdot 0 \cdot q_{10} + 0 \cdot 1 \cdot q_{01} + 1 \cdot 1 \cdot q_{11} - p_{S_iI_1}p_{I_iI_1}
= -p_{S_iI_1}p_{I_iI_1},
\]

where \( q_{k_1k_2} = \text{Pr}(\varphi_{S_iI_1} = k_1, \varphi_{I_iI_1} = k_2), k_1, k_2 \in \{0, 1\}, \) with \( q_{11} = 0, \) since only one offspring can win any single site.

Finally, we are able to simplify \( B(\bar{x}_j, \bar{x}_j) \) after some algebra:

\[
B(\bar{x}_j, \bar{x}_1, \bar{x}_2) = \sum_{i \in \{S_j, I_j\}} u_i \text{Var}(\sum_{m \in \{S_i, I_i\}} v_m \xi_{ml})
= u_{S_i} \text{Var}(v_{S_i}s_j, s_j + v_{I_i} \xi_{I_j} s_j) + u_{I_j} \text{Var}(v_{S_i}s_j, I_j + v_{I_i} \xi_{I_j} I_j)
= \ldots
= u_{S_j} \left\{ v_{S_j}^2 [p_{S_is_j}(1 - p_{S_is_j}) + \lambda_{S_j,s_j,s_j} + \lambda_{S_j,s_j,I_1} + \lambda_{S_j,s_j,s_2} + \lambda_{S_j,s_j,I_2}]
+ v_{I_j}^2 [p_{I_is_j}(1 - p_{I_is_j}) + \lambda_{I_is_j,s_j} + \lambda_{I_is_j,I_1} + \lambda_{I_is_j,s_2} + \lambda_{I_is_j,I_2}]
- 2v_{S_j}v_{I_j}p_{S_is_j}p_{I_is_j} \right\}
+ u_{I_j} \left\{ v_{S_j}^2 [p_{S_is_j}(1 - p_{S_is_j}) + \lambda_{S_j,s_j,s_j} + \lambda_{S_j,I_1,s_j} + \lambda_{S_j,I_1,I_1} + \lambda_{S_j,s_j,s_2} + \lambda_{S_j,I_1,I_2}]
+ v_{I_j}^2 [p_{I_is_j}(1 - p_{I_is_j}) + \lambda_{I_is_j,s_j} + \lambda_{I_is_j,I_1} + \lambda_{I_is_j,s_2} + \lambda_{I_is_j,I_2}]
\right\}
\]

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7.5 Evolution of two coexisting residents

As shown in Geritz et al. (2016) at a branching point the direction of branching in a two dimensional trait space is parallel to the eigenvector of the dominant eigenvalue of the Hessian $H$, that has elements

$$(H)_{ij} = \frac{\partial^2 W(\bar{x}, \bar{y})}{\partial y_i \partial y_j} \bigg|_{\bar{y} = \bar{x} = \hat{x}},$$

of the monomorphic invasion fitness $W$ (see eq. (6.2) on page 23).

In order to integrate the canonical equation for residents $x_1$ and $x_2$, we have computed the Hessian $H$, solved the eigenvector corresponding to the leading eigenvalue of $H$, and chosen two points near the singularity, on both sides, such that they are on the line spanned by the eigenvector, as initial conditions for the canonical equation.

Figures 11a and 11b show the evolution of $d_1$ and $d_2$, as well as $\delta_1$ and $\delta_2$, respectively. Furthermore, Figures 12a and 12b show what is the resident equilibrium value during evolution. Initially the residents evolve in parallel, but opposite, directions. We see that at evolutionary time $\tau \approx 8940$ the resident $\bar{x}_2$ goes extinct, and resident $\bar{x}_1$ is now the only resident in the population. Thus the dimorphic population has become a monomorphic population, so the single resident continues evolving. Since resident $\bar{x}_1$ had already evolved far enough from the attracting singularity during coexistence, it evolves to the Hamilton-May singularity, and thus the disease also dies out. This can be seen in Figure 8b from page 24, since it shows that if one is north enough from the attracting singularity, evolution toward the disease dying out is inevitable.
Figure 11: Figures show the evolution of resident strategies $\bar{x}_1$ and $\bar{x}_2$, with susceptible strategies in (a) and infected strategies in (b). Parameter values: $s = 0.1$, $c = 0.25$, $a = 1.5$, $p = 0.9$, $s = 0.1$, $\sigma = 0.25$, $\beta = 6$, $\mu = 0.1$, and $\eta = 0.2$.

Figure 12: The evolution of the resident equilibrium values. (a) Time interval is from 0 to 10000. (b) Time interval is from 8700 to 9100, to see more closely at which point of evolutionary time $\tau$ does resident $\bar{x}_2$ go extinct. Parameter values: $s = 0.1$, $c = 0.25$, $a = 1.5$, $p = 0.9$, $s = 0.1$, $\sigma = 0.25$, $\beta = 6$, $\mu = 0.1$, and $\eta = 0.2$.
8 Discussion

In this thesis we have shown that in a two dimensional trait space evolutionary branching of dispersal is possible with certain parameter values. After branching the two residents initially evolved in opposite directions, with roughly equal fraction of sites to each resident. During evolution, the resident at a higher point of $\delta$ then began occupying a larger fraction of sites, which then lead the second resident to extinction. At this time the only resident left entered a monomorphic case, and the trajectory found by integrating the canonical equation lead the resident towards the extinction of the disease. In contrast, with lower values of the virulence $\eta$, we showed that there can exists a strongly attracting, evolutionary stable singular strategy. Should the initial conditions of the canonical equation lie in the basin of attraction, with a positive density of infected sites, the evolutionary trajectory found by integrating the canonical equation would lead the single resident to this singularity, as seen in Figure 8b. Furthermore, should $\eta$ be high enough, so that no singular strategy would exist in the strategy space, starting with a single resident we showed that the trajectory found by integrating the canonical equation, with any initial condition from the strategy space, evolution lead to the extinction of the disease, as seen in Figure 8c.

With only a single trait evolving we were able to recover the Hamilton-May singularity, when the density of infected sites was zero, and show that other singular strategies exist when the disease was infectious enough such that the density of infected sites was positive. In these few cases observed we were able to determine that the singularities were attracting and evolutionarily stable, as is the Hamilton-May singularity. Whether branching is possible is still unknown, since the seven parameters we had gave a lot of options in which to vary, and may take a long time to analyze numerically.

The effect of infectious disease dynamics on dispersal and/or the evolution of dispersal has been studied in many models. Lion et al. (2006) formulated and analyzed a spatial lattice model, where each site has a specific number of neighbors, and each site can be empty, occupied by a susceptible or infected individual, and no vertical transmission was assumed. Sites where occupied via birth from neighboring sites, and a susceptible site could be infected if it had a infected neighbor. Migration rate of the infected host was considered the evolutionary trait, and fitness was determined by a spatial fitness function.

Débarre et al. (2012) formulated and analyzed a spatially structured population model, where individual sites could be empty, or occupied by one susceptible or infected individual. Empty sites were occupied via local or global dispersal of offspring, local occupying meaning neighboring empty
sites, and global meaning randomly dispersing into an empty site. Furthermore, individuals occupying sites could die, with higher mortality given to those individuals who were infected. Both susceptible and infected individuals could give birth, with no vertical transmission of the disease. The virulence and transmission rate of the disease were considered the evolving traits, and were analyzed separately.

Iritani and Iwasa (2014) formulated and analyzed a metapopulation model, with the individual having the same type of life cycle as in the Hamilton-May model (1977). Here they considered infinitely many demes that supported several adult individuals. At the beginning of each year each individual gives birth to a large number of offspring, and in each deme a fixed fraction of offspring are infected. Dispersal and surviving dispersal is state dependent, i.e. whether an individual is infected or not, assuming the probability of surviving dispersal is lower for a infected than a susceptible. Furthermore, after dispersal a fixed fraction of infected offspring at each deme dies, and the many winners of each deme give birth to the next generation. The dispersal rates of susceptible and infected individuals were considered the evolutionary traits, and fitness was determined by the sum of fitness in foreign demes and in the home deme. A similar approach was taken in Iritani (2015) with the additional assumption that a susceptible could be infected, and an infected could be cured during dispersal.

North and Godfray (2017) formulated and analyzed a metapopulation model where sites are created in clusters, that occur randomly, with the number of sites in each cluster given by a Poisson-distribution. In the cluster a site is determined by it’s distance from the center of the cluster. They consider two cases: fixed number of clusters, or dynamic. Each site can be empty, or occupied by a susceptible or infected individual. Empty sites are occupied by migrants from either susceptible or infected sites, with respective migration rates, depending also on the distance of the focal site, and occupants can die with respective rates to infected and susceptible individuals. The infected sites produce pathogen propagules, that transmit the disease to susceptible individual, which can happen also with the offspring of an infected site migrating to a susceptible site.

Ronce and Promislow (2010) formulated and analyzed a metapopulation model, with similar life-cycle of and individual as in the Hamilton-May model (1977), but with great changes: each site is occupied by an adult individual, that gives birth to a large number of offspring, that disperse at a fixed probability, but the adult survives to the next breeding season with an age specific probability. Furthermore, the number of offspring is age specific. The additional assumption is that offspring cannot overtake a site that is already occupied by an adult, and thus are competing for ownership of sites.
that are empty, i.e. the occupying adult had died after giving birth, and so the offspring that have not dispersed in a still occupied site have no chance of winning it.
References


