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Dispersal, disease and life-history evolution

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Abstract

Discrete-time susceptible–infective–susceptible (S–I–S) disease transmission models can exhibit bistability (alternative stable equilibria) over a wide range of parameter values. We illustrate the richness generated by such 'simple' non-linear systems in the study of two patch epidemic models with disease-enhanced or disease-suppressed dispersal. Dispersal between patches can have a profound impact on local patch disease dynamics. In fact, dispersal between patches may give rise to bistability in parameter regimes without bistability in single patch models. © 2001 Elsevier Science Inc. All rights reserved.

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1. Introduction

The impact of 'disease' on animal populations in a broad sense is part of the study of host-parasite interactions (see [3,30,32,37–40]). Over the last 20 years (with some notable exceptions) the study of disease dynamics has focused on models with human hosts (see [3,5,12–18,20,21,30,37,38]). Models that incorporate population and disease dynamics as well as dispersal are rare, regardless of the type of hosts. Here, we study the role of dispersal on disease dynamics in populations capable of supporting complex dynamics. Local (single patch) disease dynamics are modeled via an S–I–S epidemic process that does not include disease-induced mortality. This is unrealistic since most diseases in animal populations impact host mortality. However, our choice of framework is a function of our driving questions: Are simple discrete-time epidemic models bistable? Does dispersal give rise to bistability? Our setting assumes that complexity comes

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exclusively from the (disease-free) population dynamics so that we can focus exclusively on the role of dispersal on disease dynamics.

Single patch discrete-time S–I–S epidemic models are capable of generating complex (chaotic) dynamics, a situation not shared by *classical* continuous-time epidemic models [1,2,12,14–18]. Typically, the reproductive number of the disease R_0 is the key threshold parameter. R_0 less than 1 most often guarantees the global asymptotic stability of the disease-free equilibrium (the disease dies out) while R_0 bigger than 1 typically supports the existence and global asymptotic stability of a unique endemic equilibrium (disease persists).

Non-constant transmission rates can generate multiple stable equilibria in single patch models. Examples of epidemic models exhibiting this behavior were first established for continuous-time epidemic models by Castillo-Chavez et al. [13–15], Dushoff et al. [25] and Huang et al. [35]. Recently, examples using simpler continuous-time models were constructed by Hadeler and Castillo-Chavez [30], Hadeler and van den Driessche [31], Feng et al. [27], Kribs-Zaleta and Velasco-Hernández [36], and van den Driessche and Watmough [46]. The results of Feng et al. [27], Huang et al. [35] and Hadeler and Castillo-Chavez [30] have far reaching implications for the implementation of effective public health policies for HIV and tuberculosis, and that of van den Driessche and Watmough [46] have important theoretical implications since they illustrate the possibility of multiple stable equilibria for simple epidemic processes (S–I–S epidemic models based on a Volterra integral equation). The results of Kribs-Zaleta and Velasco-Hernández [36] focus on diseases at borders, a topic first addressed in [28].

Multiple stable equilibria are possible in *classical* discrete-time epidemic models (that is, models with typical forces of infection). An example using a discrete-time susceptible–exposed–infective–susceptible (S–E–I–S) epidemic model has been recently developed [4]. Here, we show that bistability in simple discrete-time S–I–S epidemic models with or without dispersion between patches is possible. Albeit, bistability in a single patch is generated following the approach in [46]. This approach can be avoided but it requires an additional dimension for the epidemic process (see [4]). Since our desire is to look at the role of dispersal in the simplest possible setting, we follow the approach in [46].

The paper is organized as follows: In Section 2, we study single patch discrete-time S–I–S models and establish conditions for the occurrence of a unique stable endemic equilibrium point under various recruitment regimes of new susceptibles (no bistability). In Section 3, we illustrate the possibility of bistability in single patch discrete-time S–I–S models. Section 4 focuses on two patch discrete-time S–I–S models with disease-enhanced and disease suppressed dispersal between patches. Conclusions are in Section 5 and proofs are collected in Appendix A.

2. Single patch S–I–S epidemic models

In a single patch, the dynamics of the total population size in generation t, denoted by $T(t)$, is typically governed by equations of the form

$$T(t+1) = f(T(t)) + \gamma T(t), \tag{1}$$

where γ denotes the 'probability' of survivorship per generation while f denotes the local birth or recruitment function. An epidemic process is built on 'top' of the demographic pattern generated

by f. Density regulation, modeled by Eq. (1), can support complex dynamics for $T(t)$. To guarantee control over the local dynamics (no matter how complex they are) it is assumed that the disease does not affect T in a *significant* way, that is, we *ignore* disease-induced mortality. The absence of disease-induced mortality does not imply that the population does not experience death. Fixing the local population dynamics and building an epidemic processes on them is quite common for continuous-time epidemic processes but less common for discrete-time epidemic models [1,2,12–19].

$S(t)$ denotes the population of susceptibles; $I(t)$ denotes the population of the infected, assumed infectious; $T(t) \equiv S(t) + I(t)$ denotes the total population size at generation t, and, $T_\infty \equiv \lim_{t\to\infty} T(t)$ denotes the demographic steady state for the total population whenever it exists. Individuals are assumed to survive with constant probability γ (die with probability $1-\gamma$) each generation while infected individuals recover with probability $1-\sigma$ (do not recover with constant probability σ). It is assumed that susceptible individuals become infected with probability $1-G$ per generation (remain susceptible with non-constant probability function G). Here, we further assume that $g(y) \equiv G(y\alpha(y))$, where the transmission function $\alpha \equiv \alpha(y)$, that is, α models the impact of prevalence ($y \equiv I(t)/T(t)$) on G. In general, $G : [0,\infty) \to [0,1]$ is a monotone concave probability function with $G(0) = 1$; $G'(x) < 0$ and $G''(x) \geqslant 0$ for all $x \in [0,\infty)$, and, $\alpha : [0,1] \to [0,\infty)$ is a smooth function.

It is also assumed that the disease is not fatal; all recruits are susceptible; the recruitment function depends on the total population; time is measured in generations; and, recovery from disease does not give permanent or temporary immunity. Model construction assumes implicitly a sequential process. At each generation, the fraction $(1-\gamma)$ of each class is removed (death); then surviving susceptibles become infected with probability $(1-G)$; while, independently, surviving infectives recover with probability $(1-\sigma)$. The use of this sequential approach simplifies the analysis without limiting the nature of the results.

These assumptions and definitions lead to the following discrete-time single patch S–I–S model with no dispersion

$$\begin{aligned} S(t+1) &= f(T(t)) + \gamma g(y(t))S(t) + \gamma(1-\sigma)I(t), \\ I(t+1) &= \gamma(1-g(y(t)))S(t) + \gamma\sigma I(t), \end{aligned} \tag{2}$$

where $0 < \gamma, \sigma < 1$ and $T(t) > 0$. Model (2) reduces to the model of Castillo-Chavez and Yakubu [10,18] whenever the transmission function α is a constant. Whether or not epidemics built on populations with complex dynamics exhibit qualitatively similar dynamics is part of the questions of interest (see [10,18]).

2.1. Asymptotically bounded growth

To gain some understanding on the role of dispersal, population dynamics, and disease on life-history evolution, we look at the dynamics of our model under specific functional forms for the recruitment function f (forms commonly found in the literature). If new recruits arrive on a single patch at the positive constant rate Λ per generation and there is no dispersion (Eq. (1) with $f(T(t)) = \Lambda$), then $T_\infty = \Lambda/(1-\gamma)$. If the birth or recruitment process is governed by Ricker's Equation ($f(T(t)) = T(t)\exp(r - kT(t))$, where r and k are positive constants) and there is no

dispersion then Eq. (1) implies that the total population, on each patch, will eventually reach the positive steady state

$$T_\infty = \frac{r - \ln(1-\gamma)}{k}$$

whenever

$$0 < r < \frac{2 + (1-\gamma)\ln(1-\gamma)}{(1-\gamma)}$$

($T(t)$ would exhibit complex local dynamics for larger values of r (see [18])).

System (2) can be analyzed if one assumes that the total population has reached a demographic equilibrium before the disease invades. In other words, it is assumed that the total population has reached the positive steady state T_∞, that is, we set $T(t) = T_\infty$, $x(t) = S(t)/T_\infty$ and $y(t) = I(t)/T_\infty$ in System (2). The resulting one-dimensional autonomous 'limiting system' for $y(t)$ is therefore given by

$$y(t+1) = \gamma(1 - g(y(t)))(1 - y(t)) + \gamma\sigma y(t). \tag{3}$$

Simulations support the conclusion that Eq. (3) exhibits the same qualitative dynamics as those of System (2). Theoretical results on the qualitative dynamics equivalence of autonomous and non-autonomous systems have been established by Thieme [45], in the context of continuous-time dynamical systems and by Zhao [48] for discrete-time dynamical systems. Their results support this type of simplification under very general conditions. The following results depend on the assumption that Eq. (3) and System (2) have the same qualitative dynamics whenever there is a unique positive stable equilibrium T_∞ [10].

The basic reproductive number, $\mathscr{R}_0$, determines the asymptotic behavior of System (3).

$$\mathscr{R}_0 = \begin{cases} \gamma\sigma & \text{if } \alpha(0) = 0, \\ \frac{-\gamma\alpha(0)G'(0)}{1-\gamma\sigma} & \text{if } \alpha(0) \neq 0, \end{cases} \tag{4}$$

gives the average number of secondary infections generated by a small pioneer population of infected (assumed infectious) individuals over their life-time. Epidemiologically (and typically), if $\mathscr{R}_0 > 1$ the number of infectives grows while if $\mathscr{R}_0 < 1$ the number of infectives would decrease to zero regardless of initial conditions. This is what happens when $\alpha(y)$ is a constant function. We collect these results in the following theorem.

Theorem 2.1. *Let $\alpha \equiv \alpha_0$ be a positive constant.*

(a) *If $\mathscr{R}_0 < 1$, then the solutions $(x(t),\ y(t))$ of System* (3) *approach the disease-free equilibrium, $(1, 0)$, as $t \to \infty$.*

(b) *If $\mathscr{R}_0 > 1$, then the solutions $(x(t),\ y(t))$ of System* (3) *approach a unique positive endemic equilibrium, $(1 - \overline{y}, \overline{y}) \in (0, \infty) \times (0, \infty)$, as $t \to \infty$.*

Proof. The reproduction function for the proportion of infected individuals of Eq. (3) is given by

$$h(y) = \gamma(1 - G(\alpha_0 y))(1 - y) + \gamma\sigma y,$$

where $h:[0,1]\to[0,1]$, $h(0)=0$ and $0\leqslant y\leqslant 1$. The set of iterates of h is equivalent to the set of density sequence generated by Eq. (3). Differentiation with respect to y gives

$$h'(y)=-\gamma(1-G(\alpha_0 y))-\alpha_0\gamma G'(\alpha_0 y)(1-y)+\gamma\sigma,$$
$$h''(y)=2\alpha_0\gamma G'(\alpha_0 y)-\alpha_0^2\gamma(1-y)G''(\alpha_0 y).$$

To establish the result, we will show that $\mathscr{R}_0<1$ implies $0<h'(0)<1$, $\mathscr{R}_0>1$ implies $h'(0)>1$, and, $h''(y)<0$ for $y\in(0,1]$.

$0<\mathscr{R}_0<1$ implies that $0<h'(0)=-\alpha_0\gamma G'(0)+\gamma\sigma<1$. Therefore, the fixed point $\{0\}$ is locally stable under h-iteration. Since $G'<0$ and $G''\geqslant 0$ we have that, $h''(y)<0$ for $y\in[0,1]$. The monotonicity condition on h' and the fact that $h'(0)<1$ imply that $h'(y)<1$ or $h(y)<y$ for $y\in(0,1]$. Hence, $\{y(t)\}_{t\geqslant 0}$, a strictly decreasing sequence bounded below by zero, converges to the only fixed point of h in the interval $[0,1]$, zero. This proves condition (a).

$\mathscr{R}_0>1$ implies that $h'(0)=-\alpha_0\gamma G'(0)+\gamma\sigma>1$ and, therefore, the fixed point $\{0\}$ is locally unstable under h-iteration. Let $\overline{y}$ denote the smallest positive fixed point of h in $[0,1]$, and note that $h(1)=\gamma\sigma<1$. The intermediate value theorem guarantees the existence of the positive fixed point $\overline{y}\in(0,1)$ satisfying $h(\overline{y})=\overline{y}$ and $h(y)>y$ for $y\in(0,\overline{y})$ and, consequently, $h'(\overline{y})\leqslant 1$. Since $h''(y)<0$ implies that $h'(y)<h'(\overline{y})\leqslant 1$ for $y\in(\overline{y},1)$, then $\int_{\overline{y}}^{y}h'(x)\mathrm{d}x<\int_{\overline{y}}^{y}\mathrm{d}x$ and, we have $h(y)<y$ for $y>\overline{y}$. Hence, h has a unique positive fixed point $\overline{y}\in(0,1)$. Furthermore, $h(y)>y$ for $y\in(0,\overline{y})$ and $h(y)<y$ for $y\in(\overline{y},1]$.

To establish the global stability of $\overline{y}$, we first prove the non-existence of non-trivial two-cycles for h. First, we show that $1+h'(y)>0$. Note that $1+h'(y)=1-\gamma(1-G(\alpha_0 y))-\alpha_0\gamma G'(\alpha_0 y)(1-y)+\gamma\sigma\geqslant 1-\gamma+\gamma\sigma>0$. Hence, $1+h'(y)\neq 0$ for $y\in(0,1]$. Suppose h has a non-trivial 2-cycle $\{p,q\}$, where $p,q\in[0,1]$, then $h(p)=q$ and $h(q)=p$ where $p\neq q$. The mean value theorem guarantees the existence of a point r between p and q such that $h'(r)=h(p)-h(q)/(p-q)=-1$, and $1+h'(r)=0$, a contradiction. Hence, h has no non-trivial 2-cycles in $[0,1]$. From a result of Cull [22]; McCluskey and Muldowney [41]; the non-existence of non-trivial 2-cycles for h implies global stability of the positive fixed point $\overline{y}$.

When the transmission function $\alpha\equiv\alpha(y)$ is not a constant function, Theorem 2.1 does not apply. However, $\mathscr{R}_0<1$ implies $0<h'(0)<1$ and $\mathscr{R}_0>1$ implies $h'(0)>1$. If, in addition, $\alpha(y)+y\alpha'(y)>0$ and $2\alpha'(y)+y\alpha''(y)\leqslant 0$, then $h''(y)<0$ for $y\in(0,1]$ and the following result follows.

Corollary 2.2. *Let*

$$\alpha(y)+y\alpha'(y)>0$$

and

$$2\alpha'(y)+y\alpha''(y)\leqslant 0.$$

(a) *If* $\mathscr{R}_0<1$, *then the solutions* $(x(t),\ y(t))$ *of System* (3) *approach the disease-free equilibrium*, $(1,0)$, *as* $t\to\infty$.
(b) *If* $\mathscr{R}_0>1$, *then the solutions* $(x(t),y(t))$ *of System* (3) *approach a unique positive endemic equilibrium*, $(1-\overline{y},\overline{y})\in(0,\infty)\times(0,\infty)$, *as* $t\to\infty$.

The outline of the proof of Corollary 2.2 is in Appendix A.

2.2. Geometric growth

If new recruits are assumed to arrive at the positive per-capita rate μ per generation, that is, if $f(T(t)) = \mu T(t)$, then Eq. (1) becomes the linear difference equation

$$T(t+1) = (\mu+\gamma)T(t), \tag{5}$$

that is,

$$T(t) = (\mu+\gamma)^{t}T(0). \tag{6}$$

The demographic basic reproductive number is defined by

$$\mathscr{R}_{\mathrm{d}} = \frac{\mu}{1-\gamma},$$

where $\mathscr{R}_{\mathrm{d}}$ is a dimensionless quantity that gives the average number of descendants produced by a typically small pioneer population ($T(0)$) over its life-time. $\mathscr{R}_{\mathrm{d}} > 1$ implies that the population invades at a geometric rate while $\mathscr{R}_{\mathrm{d}} < 1$ leads to extinction. We now build an epidemic process on a population with disease-free geometric growth.

The study of the dynamics of System (2) can be simplified via the use of proportions. In fact, the use of the new variables

$$x(t) = \frac{S(t)}{T(t)} \quad \text{and} \quad y(t) = \frac{I(t)}{T(t)}, \tag{7}$$

reduces System (2) with $f(T(t)) = \mu T(t)$ to

$$\begin{aligned} x(t+1) &= \frac{\mu}{\mu+\gamma} + \frac{\gamma}{\mu+\gamma}x(t)g(y(t)) + \frac{\gamma}{\mu+\gamma}(1-\sigma)y(t), \\ y(t+1) &= \frac{\gamma}{\mu+\gamma}x(t)(1-g(y(t))) + \frac{\gamma}{\mu+\gamma}\sigma y(t). \end{aligned} \tag{8}$$

Since $x(t)+y(t) = 1$ for all t in System (8), then all solutions live on the invariant line $\{(x,y) \in [0,\infty)\times[0,\infty) \mid x+y=1\}$. The substitution $x(t) = 1-y(t)$ reduces System (8) to a one-dimensional autonomous system for $y(t)$, namely,

$$y(t+1) = \frac{\gamma}{\mu+\gamma}(1-y(t))(1-g(y(t))) + \frac{\gamma}{\mu+\gamma}\sigma y(t). \tag{9}$$

From Eq. (9) we compute the basic reproductive number

$$\mathscr{R}_0 = \begin{cases} \frac{\gamma\sigma}{(1-\mathscr{R}_{\mathrm{d}})\gamma+\mathscr{R}_{\mathrm{d}}} & \text{if } \alpha(0) = 0, \\ \frac{-\gamma\alpha(0)G'(0)}{(1-\gamma)(\mathscr{R}_{\mathrm{d}}-1)+1-\gamma\sigma} & \text{if } \alpha(0) \neq 0. \end{cases} \tag{10}$$

$\mathscr{R}_0$ is easily derived from the linearization of Eq. (9) near $(x_\infty, y_\infty) \equiv (1,0)$, that is, from

$$y(t+1) \approx \frac{\gamma}{\mu+\gamma}(-\alpha(0)G'(0)+\sigma)y(t).$$

If $\mathscr{R}_{\mathrm{d}} = 1$ (no demographic impact), then $\mathscr{R}_0$ reduces to $\mathscr{R}_0 = \gamma\sigma$ or $\mathscr{R}_0 = -\gamma\alpha(0)G'(0)/(1-\gamma\sigma)$, where $1/(1-\gamma\sigma)$ denotes the average death-adjusted length of the infectious period in genera-

tions; γ is the proportion of surviving susceptibles who can be invaded by the disease; and, $-\alpha(0)G'(0)$ is the maximum rate of infection per infective (no dispersion) (see [18]). If $\mathscr{R}_d \neq 1$, then demography impacts disease dynamics, that is $\mathscr{R}_0$. In fact, $1/((1-\gamma)(\mathscr{R}_d - 1) + 1 - \gamma\sigma)$ gives the demographic death-adjusted infectious period measured in generations. Hence, $\mathscr{R}_0$ decreases with population growth ($\mathscr{R}_d > 1$) and increases with population decay ($0 < \mathscr{R}_d < 1$) as all new recruits are assumed to be susceptibles. We collect these results in the following theorem.

Theorem 2.3. *In System* (8), *let*

$$\alpha(y) + y\alpha'(y) > 0$$

and

$$2\alpha'(y) + y\alpha''(y) \leqslant 0.$$

Then,

(a) *if* $\mathscr{R}_d < 1$, *the total population*, $T \equiv S + I$, *decreases to zero at a geometric rate*; $\mathscr{R}_d > 1$ *implies that the total population increases at a geometric rate*; $\mathscr{R}_d = 1$ *implies that the total population remains fixed at its initial value.*

(b) *If* $\mathscr{R}_d > 1$ *and* $\mathscr{R}_0 < 1$, *then the proportion* I/T *of infectives in the total population tends to* 0 *as* $t \to \infty$, *while the proportion* S/T *of susceptibles in the total population tends to* 1 *as* $t \to \infty$. *Hence*, $(S/T, I/T)$ *tends to the disease-free equilibrium* $(1, 0)$, *where* S *is increasing at the same geometric rate as* T.

(c) *If* $\mathscr{R}_d > 1$ *and* $\mathscr{R}_0 > 1$, *then the proportion* I/T *of infectives in the total population tends to a positive number*, $\overline{I/T}$ *as* $t \to \infty$, *and the proportion* S/T *of susceptibles in the total population also tends to a positive number* $1 - (\overline{I/T})$ *as* $t \to \infty$. *Hence*, $(S/T, I/T)$ *tends to an endemic equilibrium.* I, S *and* T *are increasing at the same geometric rate.*

(d) *If* $\mathscr{R}_d < 1$ *and* $\mathscr{R}_0 < 1$, *then the proportion* I/T *of infectives in the total population tends to* 0 *as* $t \to \infty$, *while the proportion* S/T *of susceptibles in the total population tends to* 1 *as* $t \to \infty$. *Hence*, $(S/T, I/T)$ *tends to the disease-free equilibrium* $(1, 0)$. *Hence*, S *is decreasing to zero at the same geometric rate as* T.

(e) *If* $\mathscr{R}_d < 1$ *and* $\mathscr{R}_0 > 1$, *then the proportion* I/T *of infectives in the total population tends to a positive number* $\overline{I/T}$ *as* $t \to \infty$, *and the proportion* S/T *of susceptibles in the total population also tends to a positive number* $1 - (\overline{I/T})$ *as* $t \to \infty$. *Hence*, $(S/T, I/T)$ *tends to an endemic equilibrium. Hence*, I, S *and* T *are decreasing at the same geometric rate.*

Proof. Recall that, $T(t+1) = (\mu + \gamma)T(t)$ and $\mathscr{R}_d = \mu/(1-\gamma)$. Hence, $\mathscr{R}_d > 1$ implies T increases geometrically and $\mathscr{R}_d < 1$ implies T decreases geometrically. To establish the result, we prove that if $\mathscr{R}_0 < 1$, then the solutions $(x(t),\ y(t))$ of System (8) approach the equilibrium $(1, 0)$, as $t \to \infty$. If $\mathscr{R}_0 > 1$, we proceed exactly as in the proof of Theorem 2.1 and Corollary 2.2 to prove that the solutions $(x(t),\ y(t))$ of System (8) approach a unique positive endemic equilibrium, $(\overline{x}, \overline{y}) \in (0, \infty) \times (0, \infty)$, as $t \to \infty$.

For the proof of Theorem 2.3, the reproduction function for the infected individuals of System (8) is

$$h(y) = \frac{\gamma}{\mu+\gamma}(1-g(y))(1-y) + \frac{\gamma}{\mu+\gamma}\sigma y,$$

where $h : [0,1] \to [0,1]$. Now, we proceed exactly as in the proof of Theorem 2.1 and proof of Corollary 2.2 to establish the result.

3. Bistability

Typically, epidemic models have a unique stable equilibrium with the reproductive number of the disease serving as a threshold parameter. If the reproduction number is less than 1, the disease dies out and if the reproduction number is bigger than 1, the disease persists (Theorem 2.1, Corollary 2.2 and Theorem 2.3). When the transmission rate $\alpha \equiv \alpha(y)$ is a non-constant function, then two stable equilibria for System (3) are possible (bistability).

In order to illustrate this phenomenon explicitly, it is assumed throughout this section that infections are modeled as Poisson processes and that $\alpha(y) = y$ so that $g(y) = e^{-y^2}$ [17,18]. The approximation $e^{-y^2} \approx 1 - y^2$ is used instead to make it possible to compute explicit formulae. Hence, the use of $g(y^2) = 1 - y^2$ leads to the new well-posed system:

$$\begin{aligned} x(t+1) &= \frac{\mu}{\mu+\gamma} + \frac{\gamma}{\mu+\gamma}x(t)(1-y(t)^2) + \frac{\gamma}{\mu+\gamma}y(t)(1-\sigma), \\ y(t+1) &= \frac{\gamma}{\mu+\gamma}y(t)^2x(t) + \frac{\gamma}{\mu+\gamma}\sigma y(t). \end{aligned} \tag{11}$$

System (8) is not capable of supporting bistability when the transmission rate α is a constant. The use of $\alpha(y) = y$ in System (11) implies $\alpha(y) + y\alpha'(y) = 2y \geqslant 0$ and $2\alpha'(y) + y\alpha''(y) = 2 > 0$. $\mathscr{R}_0 = \gamma\sigma/((1-\mathscr{R}_d)\gamma + \mathscr{R}_d)$ simplifies to $\mathscr{R}_0 = \gamma\sigma/(\mu+\gamma)$, that is, $\mathscr{R}_0 < 1$ always. An increase in σ, the probability that an infective does not recover in the time interval, can give rise to two stable equilibria (bistability). Theorem 3.1 collects these results on bistability for System (11).

Theorem 3.1.

(a) *If*

$$0 < \mathscr{R}_0 < 1 - \frac{\gamma}{4(\mu+\gamma)},$$

then the solutions $(S/T, I/T)$ *of System* (11) *approach the disease-free equilibrium*, (1,0), *as* $t \to \infty$.

(b) *If*

$$\mathscr{R}_0 = 1 - \frac{\gamma}{4(\mu+\gamma)},$$

then System (11) *has an unstable endemic equilibrium at* $\left(\frac{1}{2}, \frac{1}{2}\right)$ *coexisting with the locally asymptotically stable disease-free equilibrium at* $(1,0)$.

(c) *If*

$$\mathscr{R}_0 > 1 - \frac{\gamma}{4(\mu+\gamma)},$$

then System (11) *has an unstable endemic equilibrium at*

$$\left(\frac{1}{2}\left(1+\sqrt{1-\frac{4(\mu+\gamma)}{\gamma}(1-\mathscr{R}_0)}\right),\quad \frac{1}{2}\left(1-\sqrt{1-\frac{4(\mu+\gamma)}{\gamma}(1-\mathscr{R}_0)}\right)\right)$$

and a locally asymptotically stable endemic equilibrium at

$$\left(\frac{1}{2}\left(1-\sqrt{1-\frac{4(\mu+\gamma)}{\gamma}(1-\mathscr{R}_0)}\right),\quad \frac{1}{2}\left(1+\sqrt{1-\frac{4(\mu+\gamma)}{\gamma}(1-\mathscr{R}_0)}\right)\right)$$

coexisting with the locally asymptotically stable disease-free equilibrium at $(1,0)$.

The proof of Theorem 3.1 is in Appendix A. $\mathscr{R}_0$ is a function of σ (the probability that an infective does not recover), γ (the survival probability) and μ (the per-capita birth rate). To illustrate numerically this bifurcation for System (11), we vary σ while keeping γ and μ fixed.

Example 1. Set the following parameter values in System (11):

$\mu = 0.01$ and $\gamma = 0.98$.

The disease-free equilibrium $(1,0)$ is locally stable for all values of the parameter, and in Example 1 it is globally stable whenever $\mathscr{R}_0 < 0.7525$. A saddle node (bistability) bifurcation occurs at $\mathscr{R}_0 \approx 0.7525$. When $\mathscr{R}_0 \approx 0.7525$, an unstable endemic equilibrium appears, and for values of $\mathscr{R}_0$ in the interval (0.7525, 1) the system has two stable equilibria (an endemic equilibria coexisting with the disease-free equilibrium (see Fig. 1 and Theorem 3.1)).

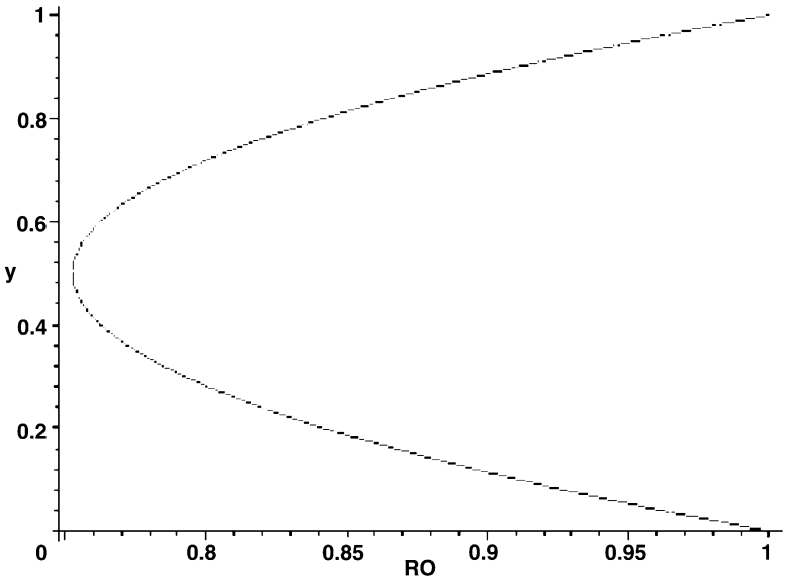

Fig. 1. The relationship between the proportion of infectives and $\mathscr{R}_0$.

4. Two-patch S–I–S epidemic models with dispersion

Dispersal is a major mechanism for the generation and support of species diversity [37,38]. The role that disease plays in the support of pathogen and host diversity is a central question in evolutionary biology particularly in the study of disease evolution [3,16–19,38,39]. Extensive work has been carried out on the role of dispersal on life-history evolution of plants. Should seeds go 'dormant', germinate or disperse [19,20,23,24,33]?

The study of the role of dispersal on human disease dynamics has been carried out in the context of influenza (see [6–9,43,44]) and tuberculosis (see [13]). Boroyan et al. [6–9], Castillo-Chavez et al. [13] and Rvachev et al. [43,44] have looked at the role of dispersal (travel) on the spatial dynamics of influenza and tuberculosis. In the context of host-parasite interactions, it is known that parasites often affect host behavior making them more susceptible to predators (fish swimming closer to the surface or diminish mobility in mammals, see [34]). Rhinoviruses (causative agent of the common cold) reproduce inside the cells that line nasal passages. Viruses are shed from these cells into nasal secretions which trickle out through a runny nose or blast out in droplets during a sneeze. The possibilities of planting rhinoviruses from infected to susceptible hosts occur after the infected host's fingers contact the host's nose and a handshake follows or when susceptible hosts' noses inhale droplets secreted by infected hosts who sneeze [26]. Consequently, virus transmission depends on host's mobility. If the virus were to reproduce so fast that hosts become too ill to disperse then rhinoviruses released would be confined to a single patch. Who should disperse, susceptibles or infectives? Dogs infected by rabies, for example, are more 'mobile' than those who are not [47]. The examples described above do not quite fit the framework that we have introduced in this article but they are illustrative of the issues that we have approached in a simpler setting. Our primary objective here is to look at the role of dispersal as an agent that promotes disease persistence. Hence, we formulate and analyze an S–I–S epidemic model with dispersal of individuals between two patches, Patches 1 and 2.

In Patch $i \in \{1,2\}$, $S_i(t)$ denotes the population of susceptibles; $I_i(t)$ denotes the population of the infected, assumed infectious; $T_i(t) \equiv S_i(t) + I_i(t)$ denotes the total population size at generation t; and, $T_{i\infty} \equiv \lim_{t\to\infty} T_i(t)$ denotes the demographic steady state for the total population whenever it exists; individuals survive with constant probability γ_i (die with probability $1-\gamma_i$) each generation while infected individuals recover with probability $1-\sigma_i$ (do not recover with constant probability σ_i); and, it is assumed that susceptible individuals become infected with probability $1-G_i$ per generation (remain susceptible with non-constant probability function G_i), and $g_i(y_i) \equiv G_i(y_i\alpha_i(y_i))$ and $y_i \equiv I_i(t)/T_i(t)$.

We couple Patches 1 and 2 with a simple exchange of a fixed fraction of the population per generation. For each Patch $i \neq j \in \{1,2\}$, let $D_{i\mathrm{S}}$ and $D_{i\mathrm{I}}$ be the fraction of the susceptible and infective populations that disperse from Patch i to j, respectively. This leads to the following system of equations for the two-patch disease dynamics:

$$\begin{aligned}
S_1(t+1) &= (1-D_{1\mathrm{S}})\widetilde{S}_1(t) + D_{2\mathrm{S}}\widetilde{S}_2(t),\\
S_2(t+1) &= D_{1\mathrm{S}}\widetilde{S}_1(t) + (1-D_{2\mathrm{S}})\widetilde{S}_2(t),\\
I_1(t+1) &= (1-D_{1\mathrm{I}})\widetilde{I}_1(t) + D_{2\mathrm{I}}\widetilde{I}_2(t),\\
I_2(t+1) &= D_{1\mathrm{I}}\widetilde{I}_1(t) + (1-D_{2\mathrm{I}})\widetilde{I}_2(t),
\end{aligned} \tag{12}$$

where $\widetilde{S}_i(t) = f_i(T_i(t)) + \gamma_i g_i(y_i)S_i(t) + \gamma_i(1-\sigma_i)I_i(t)$, $\widetilde{I}_i(t) = \gamma_i(1-g_i(y_i))S_i(t) + \gamma_i\sigma_i I_i(t)$ and $0 \leqslant D_{i\mathrm{S}}, D_{i\mathrm{I}} \leqslant 1$. The dispersion coefficients, $D_{i\mathrm{S}}$ and $D_{i\mathrm{I}}$, denote the probability of dispersion by susceptible and infective individuals from Patch i to j, respectively; while γ_i denotes the probability of survival in Patch i.

If Patch 2 is empty and there is no dispersion between the two patches ($D_{1\mathrm{S}} = D_{1\mathrm{I}} = D_{2\mathrm{S}} = D_{2\mathrm{I}} = \widetilde{S}_2(t) = \widetilde{I}_2(t) = 0$), then System (12) reduces to the single patch model, System (1). In the absence of dispersal, System (12) models two independent patches. Next, we illustrate the potential role of dispersal on patches with identical local dynamics ($f_1 = f_2$) via simple examples.

4.1. Identical local patch dynamics

It is known that dispersion between patches could alter local dynamics [4,11,16,17,19,20,23,24,29,33]. A disease destined to go extinct in Patch i, local basic reproductive number $\mathscr{R}_{0i} < 1$, could persist in the full system in the presence of dispersion (Example 2). If dispersion rates between two patches are symmetric, then System (12) exhibits the same qualitative dynamics as System (1), a one patch system with no dispersion. The joint dynamics live on the invariant set of identical population sizes

$$M = \{(S_1, S_2, I_1, I_2) \mid S_1 = S_2 \text{ and } I_1 = I_2\}.$$

This result appears to be valid only under the assumption of identical local patch ($f_1 = f_2$), local disease dynamics and identical initial conditions. Asymmetric initial conditions, even in the presence of identical local patch dynamics, can generate multiple attractors. Some initial conditions outside of the invariant set M (out-of-phase populations) give rise to dynamics that live outside of M throughout the entire life-history of the local populations. We collect these results below.

Lemma 1. *In System* (12), *the set of identical local densities*

$$\{(S_1, S_2, I_1, I_2) \mid S_1 = S_2 \text{ and } I_1 = I_2\}$$

is invariant if $\gamma_1 = \gamma_2, \sigma_1 = \sigma_2, \alpha_1 = \alpha_2, f_1 = f_2, G_1 = G_2, D_{1\mathrm{S}} = D_{2\mathrm{S}}$ *and* $D_{1\mathrm{I}} = D_{2\mathrm{I}}$.

To prove Lemma 1, note that $S_1(t) = S_2(t)$ and $I_1(t) = I_2(t)$ implies that $\widetilde{S}_1(t) = \widetilde{S}_2(t)$ and $\widetilde{I}_1(t) = \widetilde{I}_2(t)$ whenever $\gamma_1 = \gamma_2$, $\sigma_1 = \sigma_2$, $\alpha_1 = \alpha_2$, $f_1 = f_2$ and $G_1 = G_2$. Consequently, $(1 - D_{1\mathrm{S}})\widetilde{S}_1(t) + D_{2\mathrm{S}}\widetilde{S}_2(t) = S_1(t+1) = S_2(t+1)$, and $(1 - D_{1\mathrm{I}})\widetilde{I}_1(t) + D_{2\mathrm{I}}\widetilde{I}_2(t) = I_1(t+1) = I_2(t+1)$. Therefore, $\{(S_1, S_2, I_1, I_2) \mid S_1 = S_2 \text{ and } I_1 = I_2\}$ is an invariant set.

4.2. Disease-enhanced versus disease-suppressed dispersion

Do disease-induced changes in behavior improve the chances of disease persistence? An extreme case of disease-enhanced dispersal is modeled using System (12) with $D_{i\mathrm{S}} = 0$, $D_{i\mathrm{I}} > 0$. Susceptible individuals are confined to a patch while the infectives are allowed to disperse between patches. Disease-suppressed dispersal occurs in System (12) whenever $D_{i\mathrm{S}} > D_{i\mathrm{I}}$ while disease-enhanced dispersal occurs when $D_{i\mathrm{I}} > D_{i\mathrm{S}}$. If $D_{i\mathrm{I}} = 0$ while $D_{i\mathrm{S}} > 0$, then susceptible individuals

disperse between the two patches while the infectives are confined to a patch. One-way disease-suppressed dispersion from Patches 1 to 2 leads to the following system:

$$\begin{aligned} x_1(t+1) &= (1-D_{1S})\widetilde{x}_1(t),\\ x_2(t+1) &= D_{1S}\widetilde{x}_1(t)+\widetilde{x}_2(t)),\\ y_1(t+1) &= \widetilde{y}_1(t),\\ y_2(t+1) &= \widetilde{y}_2(t), \end{aligned} \tag{13}$$

where $0 < D_{1S} < 1$, and from System (11),

$$\widetilde{x}_i(t) = \frac{\mu_i}{\mu_i+\gamma_i} + \frac{\gamma_i}{\mu_i+\gamma_i}x_i(t)(1-y_i(t)^2) + \frac{\gamma_i}{\mu_i+\gamma_i}y_i(t)(1-\sigma_i),$$

and

$$\widetilde{y}_i(t) = \frac{\gamma_i}{\mu_i+\gamma_i}y_i(t)^2x_i(t) + \frac{\gamma_i}{\mu_i+\gamma_i}\sigma_i y_i(t).$$

Notice that Model (13) assumes geometric growth for the total population.

Whenever $D_{1S} = 0$, System (13) reduces to System (11). System (13) has a locally asymptotically stable disease-free equilibrium point at

$$\left(\frac{(1-D_{1S})\mu_1}{\mu_1+\gamma_1 D_{1S}}, \left(\frac{D_{1S}\mu_1}{\mu_1+\gamma_1 D_{1S}} + \frac{\mu_2}{\mu_2+\gamma_2}\right)\frac{\mu_2+\gamma_2}{\mu_2}, 0, 0\right),$$

and some positive initial population sizes lead to the extinction of the disease in the two patches. In fact, if

$$D_{1S} > 1 - \frac{4(1-q_1\sigma_1)}{q_1(p_1+4(1-q_1\sigma_1))} \text{ or } D_{1S} < \frac{\left(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2\right)q_1}{2(1-q_1\sigma_1)+\left(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2\right)q_1},$$

then System (13), where

$$p_i = \frac{\mu_i}{\mu_i+\gamma_i} \quad \text{and} \quad q_i = \frac{\gamma_i}{\mu_i+\gamma_i}$$

for each $i \in \{1,2\}$, has no endemic equilibrium population size. If

$$D_{1S} = 1 - \frac{4(1-q_1\sigma_1)}{q_1(p_1+4(1-q_1\sigma_1))} = \frac{\left(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2\right)q_1}{2(1-q_1\sigma_1)+\left(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2\right)q_1},$$

then System (13) has a unique unstable endemic equilibrium population size at the point

$$\left(\frac{2(1-q_1\sigma_1)}{q_1}, \quad \frac{2(1-D_{1S})p_2q_1(1-q_2\sigma_2)}{q_2((1-D_{1S})p_2q_1+2D_{1S}(1-q_1\sigma_1))}, \quad \frac{1}{2}, \frac{(1-D_{1S})p_2q_1+2D_{1S}(1-q_1\sigma_1)}{2(1-D_{1S})p_2q_1}\right).$$

Furthermore, the relationships

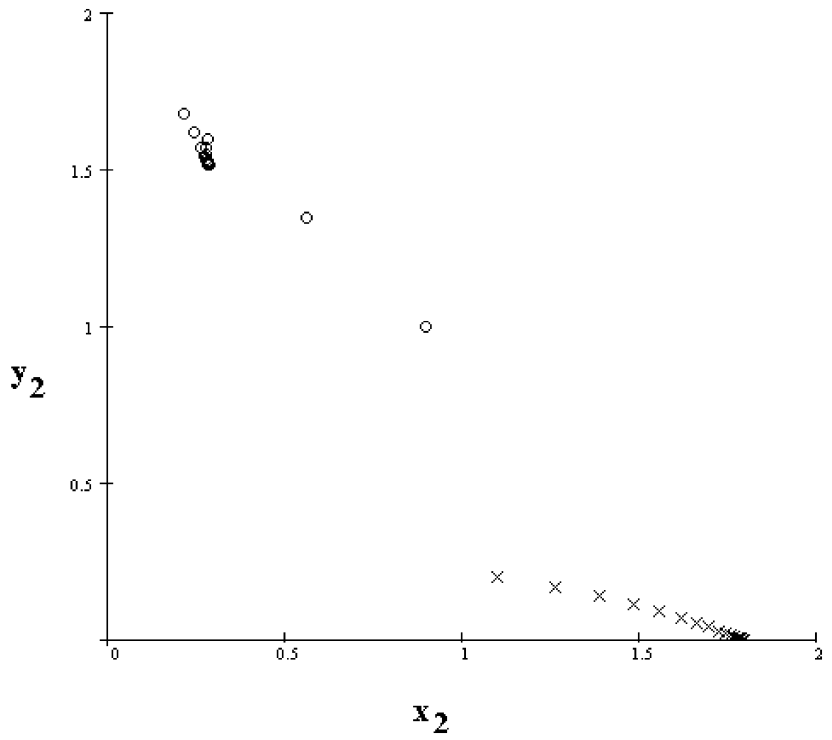

Fig. 2. (Bistability) Some initial conditions converge to (0.2, 0.286, 0, 1.514) while others converge to (0.2, 1.8, 0, 0) in Example 2 with $D_{1S} = 0.5$.

$$D_{1S} < 1 - \frac{4(1-q_1\sigma_1)}{q_1(p_1+4(1-q_1\sigma_1))} \text{ and } D_{1S} > \frac{\left(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2\right)q_1}{2(1-q_1\sigma_1)+\left(2\sqrt{\frac{1-q_2\sigma_2}{q_2}}-p_2\right)q_1}$$

imply that System (13) has two endemic equilibria. Hence, disease persistence depends on initial conditions. Consequently, System (13) is capable of supporting two endemic equilibria coexisting with the locally asymptotically stable disease-free equilibrium (bistability), with or without dispersion between patches.

The assumption of exclusive disease-driven dispersal, reduces System (12) to the following system of equations:

$$\begin{aligned} x_1(t+1) &= \widetilde{x}_1(t), \\ x_2(t+1) &= \widetilde{x}_2(t), \\ y_1(t+1) &= (1-D_{1I})\widetilde{y}_1(t) + D_{2I}\widetilde{y}_2(t), \\ y_2(t+1) &= D_{1I}\widetilde{y}_1(t) + (1-D_{2I})\widetilde{y}_2(t). \end{aligned} \tag{14}$$

System (14) reduces to System (11) whenever $D_{1I} = D_{2I} = 0$. System (14), like the corresponding single-patch model, System (11), has a locally asymptotically stable disease-free equilibrium at

$$(1, 1, 0, 0).$$

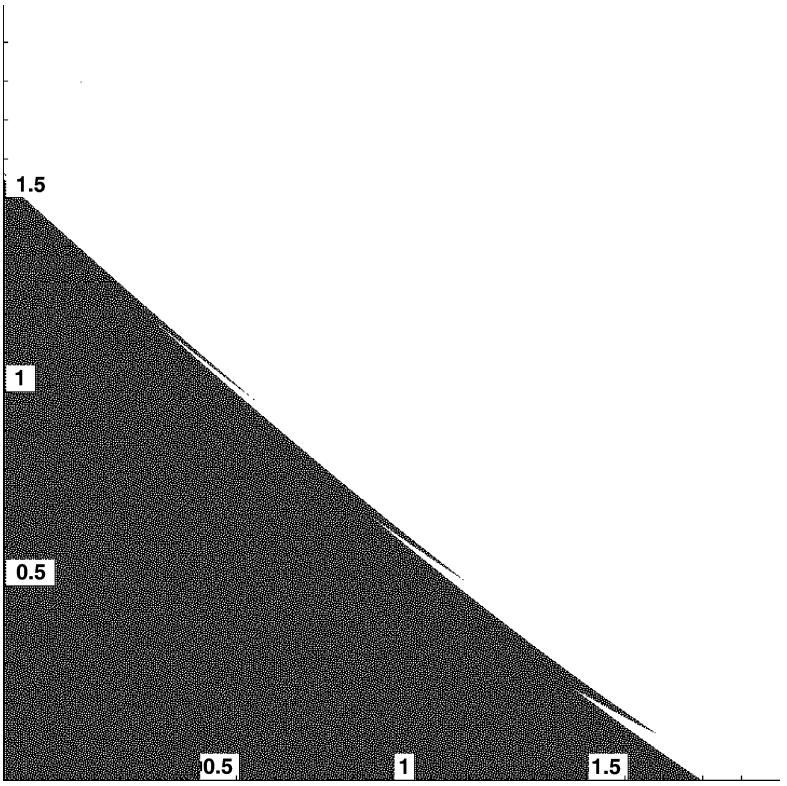

Fig. 3. Basin of attraction of (0.2, 0.286, 0, 1.514) (white region) versus that of (0.2, 1.8, 0, 0) (black region) in Example 2 with $D_{1S} = 0.5$. On the horizontal axis $0 \leqslant x_1 \leqslant 2$ and on the vertical axis $0 \leqslant x_2 \leqslant 2$.

Some positive initial population sizes lead to the extinction of the disease in Patches 1 and 2 regardless of the parameter values. System (14), like System (13), assumes geometric growth for the total population and supports bistability with or without dispersion.

Disease persistence in multi-patch systems with dispersal between patches is possible even in situations where the disease is on the brink of extinction without dispersal. To illustrate this possibility we consider the disease-suppressed dispersal model, System (13). It has two fixed points with infectives in Patch 2 and no infectives in Patch 1 at the following levels:

$$\left(\frac{(1-D_{1S})\mu_1}{\mu_1+\gamma_1 D_{1S}},\quad \frac{\mu_2+\gamma_2}{2\mu_2}\left(B\pm\sqrt{(B^2-C)}\right),\quad 0,\quad \frac{2\mu_2(1-q_2\sigma_2)}{(\mu_2+\gamma_2)q_2(B\pm\sqrt{(B^2-C)})}\right),$$

where

$$B=\frac{D_{1S}\mu_1}{\mu_1+\gamma_1 D_{1S}}+\frac{\mu_2}{\mu_2+\gamma_2},\quad C=\frac{4\mu_2^2(\mu_2+\gamma_2(1-\sigma_2))}{\gamma_2(\mu_2+\gamma_2)},\ B>\sqrt{C}\quad\text{and}\quad q_2=\frac{\gamma_2}{\mu_2+\gamma_2}.$$

Our simulations illustrate these possibilities:

Example 2. In System (13), let

$$\mu_1 = \mu_2 = 0.3, \quad \gamma_1 = \gamma_2 = 0.9, \quad \sigma_1 = \sigma_2 = 0.9,$$

the local basic reproduction number in each Patch $i \in \{1, 2\}$ is

$$\mathscr{R}_{0i} = \frac{\gamma_i \sigma_i}{(1 - \mathscr{R}_{id})}\gamma_i + \mathscr{R}_{id} = 0.675 < 1 - \frac{\gamma_i}{4(\mu_i + \gamma_i)} = 0.8125.$$

Theorem 3.1 implies that, regardless of initial conditions, the disease-free equilibrium is globally asymptotically stable and the disease dies out on each patch if there is no dispersion. Adding dispersal in Example 2 gives rise to bistability where there is no bistability in the single patch models without dispersion. Example 2 with $D_{1S} = 0.5$ supports two coexisting stable equilibria at (0.2, 0.286, 0, 1.514) and (0.2, 1.8, 0, 0) (bistability, see Figs. 2 and 3).

Adding dispersal of susceptibles from Patches 1 to 2 alters the local single patch dynamics. The disease persists in Patch 2 with dispersion where there is no disease without dispersion. In Fig. 3, the white region is in the basin of attraction of (0.2, 0.286, 0, 1.514) while the black region is in the basin of attraction of (0.2, 1.8, 0, 0). The boundary of the basins of attraction in the $x_1 - x_2$ plane is not fractal in nature (Fig. 3).

5. Conclusion

The study of the impact of disease and dispersal on life-history evolution has received little attention. The focus has often been on dispersal [11,15,19,20,23,24,33] or disease [1–3,8,10,14,15,18,27,30,35,36,42,46]. Here, we have focused on the joint impact of dispersal and disease on the life-history evolution of populations with potentially complex population dynamics. We have looked at the role of dispersal in a metapopulation setting where in every generation a *proportion* of the hosts moves from Patches i to j (where $i \neq j$). We have looked at various examples and illustrated possible disease dynamics on populations with potentially complex demography (e.g. complex life-histories).

In earlier work, we modeled a simple epidemic process on populations with rich and highly complex population dynamics [10,18]. An S–I–S epidemic process was built on the life-history of a population under a non-linear intraspecific competition. The T-dynamics were governed by Eq. (1), $T(t+1) = f(T(t)) + \gamma T(t)$, and were clearly independent of the transmission function α. We vary α while keeping all other parameters fixed in order to keep the T-dynamics fixed. The earlier work appeared to illustrate the possibility of having a population $T(t)$ living on a two cycle while $I(t)$, infected individuals, remained at the same level [10,18]. Hence, in the presence of a non-fatal disease, the T-dynamics may differ from I-dynamics in a single patch. This numerical result is unlikely to be atypical since it is possible to increase the level of complexity of an epidemic process within a single patch (bistability) even when the T-dynamics are at a globally stable fixed point. We illustrated this last result with a simple model (see [46] for the analogue and more general continuous-time versions). Hence, disease is likely to have a dramatic impact on local life-history evolution even when it is non-fatal. Many questions remain on the impact of fatal and non-fatal diseases on life-history evolution on a single patch (see [4,26]).

Disease-induced and disease-suppressed dispersal appear to play a critical role on the generation and support of multiple attractors and, in the process, increase the likelihood of disease persistence. Example 2, a disease-suppressed dispersal model with new recruits under geometric growth, shows the possibility of disease persistence and bistability (disease-free equilibrium is globally asymptotically stable and the disease dies out on each patch). In other words, dispersion enhances persistence and hence promotes diversity.

The fragmentation of the landscape (increasing the number of patches) gives rise to drastically different aggregate dynamics of the full system and these results may have serious implications in the fields of conservation and evolutionary biology. The re-establishment of connections via *dispersal corridors* in fragmented landscapes not only reduces the level of fragmentation but may in fact reduce the likelihood that a species goes extinct [19]. In other words, adding a number of tiny non-connected patches to the total size of preserved areas may not be as critical as connecting preserved areas via dispersal corridors.

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Appendix A

Proof of Corollary 2.2. The reproduction function for the infected individuals of Eq. (3) is given by

$$h(y) = \gamma(1 - g(y))(1 - y) + \gamma\sigma y,$$

where $h : [0,1] \to [0,1]$, $h(0) = 0$ and $0 \leqslant y \leqslant 1$. The set of iterates of h is equivalent to the set of density sequence generated by Eq. (3). Differentiation with respect to y gives

$$h'(y) = \gamma(-(1 - y)g'(y) - 1 + g(y) + \sigma),$$
$$h''(y) = \gamma(2g'(y) - (1 - y)g''(y)),$$

where

$$g'(y) = (\alpha(y) + y\alpha'(y))G'(y\alpha(y))$$

and

$$g''(y) = (2\alpha'(y) + y\alpha''(y))G'(y\alpha(y)) + (\alpha(y) + y\alpha'(y))^2 G''(y\alpha(y)).$$

Now, proceed exactly as in the proof of Theorem 2.1.

Proof of Theorem 2.2. Recall that, $T_i(t+1) = (\mu_i + \gamma_i)T_i(t)$ and $\mathscr{R}_{id} = \mu_i/(1-\gamma_i)$. Hence, $\mathscr{R}_{id} > 1$ implies T_i increases geometrically and $\mathscr{R}_{id} < 1$ implies T_i decreases geometrically. To establish the result, we prove that if $\mathscr{R}_{0i} \leqslant 1$, then the solutions $(x_i(t), y_i(t))$ of System (8) approach the equilibrium $(1,0)$, as $t \to \infty$. If $\mathscr{R}_{0i} > 1$, we proceed exactly as in the Proof of Theorem 2.1 to prove that the solutions $(x_i(t), y_i(t))$ of System (8) approach a unique positive endemic equilibrium, $(\overline{x}_i, \overline{y}_i) \in (0,\infty) \times (0,\infty)$, as $t \to \infty$.

For the Proof of Theorem 2.2, the reproduction function for the infected individuals of System (8) is

$$h_i(y_i) = \frac{\gamma_i}{\mu_i + \gamma_i}(1 - g_i(y_i))(1 - y_i) + \frac{\gamma_i}{\mu_i + \gamma_i}\sigma_i y_i,$$

where $h_i : [0,1] \to [0,1]$. We now, proceed exactly as in the proof of Theorem 2.1 to establish the result. □

Proof of Theorem 3.1. The reproduction function for the proportion of infected individuals of System (11) is

$$h_i(y_i) = \frac{\gamma_i}{\mu_i + \gamma_i} y_i^2(1 - y_i) + \frac{\gamma_i}{\mu_i + \gamma_i}\sigma_i y_i,$$

where $h_i : [0,1] \to [0,1]$.

Notice that $\{0\}$ is a locally asymptotically stable fixed point of h_i for all values of the parameters. To prove (a), note that $0 < \mathscr{R}_{0i} < 1 - \gamma_i/(4(\mu_i + \gamma_i))$ implies that h_i has no other fixed points in the interval (0,1]. Consequently, $h_i(y_i) < y_i$ for each $y_i \in (0,1]$, and the fixed point $\{0\}$ is globally stable under h_i iterations. Hence, the disease-free equilibrium point $(1,0)$ is globally stable in System (11). $\mathscr{R}_{0i} = 1 - \gamma_i/(4(\mu_i + \gamma_i))$ implies that the fixed points of h_i are $\{0\}$ and $\{\frac{1}{2}\}$. Since $\{0\}$ is locally asymptotically stable, the result is immediate.

To prove (c) notice that $\mathscr{R}_{0i} > 1 - \gamma_i/(4(\mu_i + \gamma_i))$ implies that the fixed points of h_i are $\{0\}$,

$$\left\{\frac{1}{2}\left(1 - \sqrt{1 - \frac{4(\mu_i + \gamma_i)}{\gamma_i}(1 - \mathscr{R}_{0i})}\right)\right\}$$

and

$$\left\{\frac{1}{2}\left(1 + \sqrt{1 - \frac{4(\mu_i + \gamma_i)}{\gamma_i}(1 - \mathscr{R}_{0i})}\right)\right\}.$$

Since $\{0\}$ is locally asymptotically stable, the fixed point

$$\left\{\frac{1}{2}\left(1 - \sqrt{1 - \frac{4(\mu_i + \gamma_i)}{\gamma_i}(1 - \mathscr{R}_{0i})}\right)\right\}$$

is unstable and

$$\left\{\frac{1}{2}\left(1+\sqrt{1-\frac{4(\mu_i+\gamma_i)}{\gamma_i}(1-\mathscr{R}_{0i})}\right)\right\}$$

is locally asymptotically stable. This establishes Proof of Theorem 3.1.

References

[1] L. Allen, A.M. Burgin, Comparison of deterministic and stochastic SIS and SIR models in discrete time, Math. Biosci. 163 (2000) 1.
[2] L. Allen, Some discrete-time SI, SIR and SIS epidemic models, Math. Biosci. 124 (1994) 83.
[3] R.M. Anderson, R.M. May, Infectious Diseases of Humans: Dynamics and Control, Oxford University, Oxford, 1992.
[4] R. Arreola, A. Crossa, M.C. Velasco, Discrete-time S–E–I–S models with dispersal between two patches, Biometric Department, MTBI Cornell University Technical Report, 2000.
[5] N.T.J. Bailey, The simple stochastic epidemic: a complete solution in terms of known functions, Biometrika 50 (1963) 235.
[6] V.O. Baroyan, L.A. Genchikov, L.A. Rvachev, V.A. Shashkov, An attempt at large-scale influenza epidemic modelling by means of a computer, Bull. Int. Epidemiol. Assoc. 18 (1969) 22.
[7] V.O. Baroyan, L.A. Rvachev, Deterministic epidemic models for a territory with a transport network, Kibernetika 3 (1967) 67.
[8] V.O. Baroyan, L.A. Rvachev, K. Frank et al., Model of influenza epidemics spread through the territory of Bulgaria and its significance, J. Epidemiol. Microbiol. Infect. Dis., Sofia 15 (1978) 168.
[9] V.O. Baroyan, L.A. Rvachev, Y.G. Ivannikov, Modelling and Prediction of Influenza Epidemics in USSR, Moscow, Gamelei Institute of Epidemiology and Microbiology, 1977.
[10] J.H. Barrera, A. Cintron-Arias, N. Davidenko, L.R. Denogean, S.R. Franco-Gonzalez, Dynamics of a two-dimensional discrete-time SIS model, Biometric Department, MTBI Cornell University Technical Report, 1999.
[11] J. Best, V. Pasour, N.Tisch, C. Castillo-Chavez, Delayed density dependence and the dynamic consequences of dispersal between patches (preprint, 2001).
[12] F. Brauer, C. Castillo-Chavez, Mathematical Models in Population Biology and Epidemiology, Texts in Applied Mathematics, Springer, New York (2001).
[13] C. Castillo-Chavez, A. Capurro, M. Zellner, J.X. Velasco-Hernandez, El transporte público y la dinámica de la tuberculosis a nivel poblacional. A portaciones mathematicas, Ser. Commun. 22 (1998) 209.
[14] C. Castillo-Chavez, K. Cooke, W. Huang, S.A. Levin, The role of long periods of infectiousness in the dynamics of acquired immunodeficiency syndrome (AIDS), J. Math. Biol. 27 (1988) 373.
[15] C. Castillo-Chavez, H.W. Hethcote, V. Andreasen, S.A. Levin, W.M. Liu, Cross immunity in the dynamics of homogeneous and heterogeneous populations, in: L.J. Cross, T.G. Hallam, S.A. Levin (Eds.), Mathematical Ecology, 1988, p. 303.
[16] C. Castillo-Chavez, S. Blower, P. van den Driessche, D. Kirschner, A.-A. Yakubu, in: Mathematical Approaches For Emerging and Reemerging Infectious Diseases: An Introduction to Models, Methods and Theory, Springer, New York, in press.
[17] C. Castillo-Chavez, A-A. Yakubu, Discrete-time S–I–S models with complex dynamics, in: Proc. WCNA 2000, Nonlinear Anal. TMA, in press.
[18] C. Castillo-Chavez, A-A. Yakubu, Discrete-time S–I–S models with simple and complex population dynamics, in: C. Castillo-Chavez, S. Blower, P. van den Driessche, D. Kirschner, A.-A. Yakubu (Eds.), Mathematical Approaches For Emerging and Reemerging Infectious Diseases: An Introduction to Models, Methods and Theory, Springer, Berlin, in press.
[19] C. Castillo-Chavez, A.A. Yakubu, Intraspecific competition, dispersal and disease dynamics in discrete-time patchy environments, in: C. Castillo-Chavez, S. Blower, P. van den Driessche, D. Kirschner, A-A. Yakubu (Eds.),

Mathematical Approaches For Emerging and Reemerging Infectious Diseases: An Introduction to Models, Methods and Theory, Springer, Berlin, in press.
[20] D. Cohen, S.A. Levin, The interaction between dispersal and dormancy strategies in varying and heterogeneous environments, in: E. Teramoto, M. Yamaguti, Mathematical Topics in Population Biology, Morphogenesis and Neurosciences, Proc. Kyoto, Springer, Berlin, 1985, 1987, p. 110.
[21] K.L. Cooke, J.A. Yorke, Some equations modelling growth processes and gonorrhea epidemics, Math. Biosci. 16 (1973) 75.
[22] P. Cull, Local and global stability for population models, Biol. Cybern. 54 (1986) 141.
[23] M. Doebeli, Dispersal and dynamics, Theoret. Popul. Biol. 47 (1995) 82.
[24] M. Doebeli, G.D. Ruxton, Evolution of dispersal rates in metapopulation models: branching and cylic dynamics in phenotype space, Evolution 51 (1997) 1730.
[25] J. Dushoff, W. Huang, C. Castillo-Chavez, Backwards bifurcations and catastrophe in simple models of fatal diseases, J. Math. Biol. 36 (1998) 227.
[26] P.W. Ewald, Evolution of Infectious Disease, Oxford University, Oxford, New York, 1994.
[27] Z. Feng, C. Castillo-Chavez, A. Capurro, A model for tuberculosis with exogenous reinfection, Theoret. Popul. Biol. 57 (2000) 235.
[28] J. Flores, C. Garcia, C.W. Melgar, C. Saenz, Dynamics of Rubella virus in populations with different vaccination policies, Biometric Department (BU-1424-M), MTBI Cornell University Technical Report, 1997.
[29] P.A. Gonzalez, R. Saenz, B. Sanchez, The influence of dispersal between two patches on the dynamics of a disease. Biometric Department, MTBI Cornell University Technical Report, 2000.
[30] K.P. Hadeler, C. Castillo-Chavez, A core group model for disease transmission, Math. Biosci. 128 (1995) 41.
[31] K.P. Hadeler, P. van den Driessche, Backward bifurcation in epidemic control, Math. Biosci. 146 (1997) 15.
[32] M.P. Hassell, R.C. Anderson, J.E. Cohen et al., Impact of diseases on host populations (group report), in: R.M. Anderson, R.M. May (Eds.), Population Biology of Infectious diseases, Springer, New York, 1982.
[33] A. Hastings, Complex interactions between dispersal and dynamics: lessons from coupled logistic equations, Ecology 74 (1993) 1362.
[34] J.C. Holmes, W.M. Bethel, Modification of intermediate host behaviour by parasites, in: E.V. Canning, C.A. Wright (Eds.), Behavioural aspects of parasite transmission, Zool. J. Linn. Soc. (Suppl. 1) 51 (1972) 123.
[35] W. Huang, K.L. Cooke, C. Castillo-Chavez, Stabilitiy and bifurcation for a multiple-group model for the dynamics of HIV/AIDS transmission, SIAM J. Appl. Math. 52 (1992) 835.
[36] C.M. Kribs-Zaleta, J.X. Velasco-Hernández, A simple vaccination model with a backward bifurcation, Math. Biosci. 164 (2000) 183.
[37] S.A. Levin, Encyclopedia of Biodiversity, in: S.A. Levin (Ed.), vol. 1, Academic Press, New York, 2000.
[38] S.A. Levin, R.T. Paine, Disturbance, patch formation and community structure, Proc. Nat. Acad. Sci. (USA) 71 (1974) 2744.
[39] R.M. May, Simple mathematical models with very complicated dynamics, Nature 261 (1977) 459.
[40] R.M. May, Stability and Complexity in Model Ecosystems, Princeton University, Princeton, NJ, 1974.
[41] C.C. McClusky, J.C. Muldowney, Bendixson-Dulac criteria for difference equations, J. Dyn. Diff. Eq. 10 (1998) 567.
[42] I. Nåsell, The quasi-stationary distribution of the closed endemic SIS model, Adv. Appl. Prob. 28 (1996) 895.
[43] L.A. Rvachev, Computer modelling experiment on large-scale epidemic, Dok. USSR Acad. Sci. 180 (1968) 294.
[44] L.A. Rvachev, I.M. Longini, A mathematical model for the global spread of influenza, Math. Biosci. 75 (1985) 1.
[45] H.R. Thieme, Convergence results and a Poincaré–Bendixson trichotomy for asymptotically autonomous differential equations, J. Math. Biol. 30 (1992) 755.
[46] P. van den Driessche, J. Watmough, A simple SIS epidemic model with a backward bifurcation, J. Math. Biol. 40 (2000) 525.
[47] G.P. Youmans, P.Y. Paterson, H.M. Sommers, The Biological and Clinical Basis of Infectious Diseases, Saunders, Philadelphia, PA, 1985.
[48] X.-Q. Zhao, Asymptotic behavior for asymptotically periodic semiflows with applications, Commun. Appl. Nonlinear Anal. 3 (1996) 43.