Dispersal between two patches in a discrete time SEIS model

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Abstract

Dispersal and dormancy are two of the fundamental evolutionary mechanisms used by nature to support and generate ecological diversity. In this investigation, we focus on the role of disease-enhanced or disease-suppressed dispersal on the dynamics of populations in a multi-patch system. Single patch systems, which are capable of supporting simple and complex dynamics, are studied both analytically and numerically. The impact of disease and dispersal is also studied numerically. Our results are compared to those in the literature that focused on dispersal in disease free multipatch systems.

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1   Introduction

Nature uses dispersion to support and generate ecological diversity. In [9], Hastings investigated the role of dispersal on local dynamics in discrete-time models. Hastings' model consists of two patches connected by dispersion. He showed that dispersal between patches can stabilize a system that is chaotic when there is no dispersion. Hastings also showed that dispersal between patches can lead to the creation of multiple attractors with fractal basin boundaries. Castillo-Chavez and Yakubu [3] in a recent paper explored the effects of dispersion using different intraspecific competitive regimes on patchy environments. In addition, they incorporated an S-I-S epidemic model to the dispersion model and wrote explicit equations for the dispersal of susceptible and infectious individuals between patches. Their model provides a framework to answer several interesting questions such as: Do complex population dynamics drive disease dynamics (see [1] and [3])? or, can dispersal help eliminate a disease or help it become established?

In this paper we focus on how dispersal between two patches affects the dynamics of a disease in a population. First we formulate and analyze a single patch discrete time Susceptible-Exposed-Infectious-Susceptible (SEIS) model. In the single patch model we obtain thresholds for the persistence of a disease. We also study the role of different recruitment functions, such as Ricker's model, constant recruitment, and geometric growth on single-patch disease dynamics. Ricker's model is known to generate complex dynamics. We use different functions to describe the frequency dependent probability that an individual remains susceptible or becomes infectious. In the two patch model we study the effects of dispersal of susceptibles, that is, infectives and latent individuals are assumed to be sedentary.

2   S-E-I-S Single Patch Model

In this section we introduce and analyze an S-E-I-S model in a single patch; which models the dynamics of a disease that divides a population in three classes. These classes are: susceptibles (individuals that do not have the disease or have recovered from it), exposed individuals (who have the disease but do not transmit it), and infectious (individuals that have and transmit the disease). We assume that the disease is not lethal and does not give partial or permanent immunity. The latter statement implies that an infectious

individual becomes susceptible to the disease again after treatment. Also, we assume that a susceptible individual has to be in the exposed class before becoming infectious. In the same way, an exposed individual must become infectious before recuperating and becoming susceptible.

In order to construct the equations for the model, we define the notation that will be used in the rest of this paper. At generation t, the number of susceptibles is denoted by S_t; E_t represents the number of exposed individuals; and I_t is the number of infectious. Hence the total population represented by T_t is given by $T_t = S_t + E_t + I_t$. This model allows for the birth of new individuals. The number of new individuals that enter the system per generation is given by a recruitment function f. We assume that an individual first survives, with probability γ, and then changes class (or not); i.e., the demographic dynamics happen before the disease dynamics. The probability that an exposed individual stays exposed is σ, and δ is the probability that an infected individual does not recuperate. The probability that a susceptible does not become exposed is given by a function G; thus $0 \leq G \leq 1$, where G is a function of the proportion of infectives. If there are no infectious, then the probability that a susceptible does not become exposed to the disease is 1, thus $G(0) = 1$. As the proportion of infectives increases, the probability that a susceptible does not become exposed decreases; thus $G' < 0$. Aside from these conditions we will require that $G'' \geq 0$. With these assumptions the discrete time S-E-I-S model is

$$
\left.
\begin{array}{rcl}
S_{t+1} & = & f(T_t) + \gamma G(\frac{I_t}{T_t})S_t + \gamma(1-\delta)I_t, \\
E_{t+1} & = & \gamma(1 - G(\frac{I_t}{T_t}))S_t + \gamma\sigma E_t, \\
I_{t+1} & = & \gamma(1-\sigma)E_t + \gamma\delta I_t, \\
T_{t+1} & = & S_{t+1} + E_{t+1} + I_{t+1} = f(T_t) + \gamma T_t.
\end{array}
\right\}
\tag{1}
$$

In this section, we analyze system (1) where the new recruits are governed by geometric growth ($f(T_t) = \mu T_t$), constant recruitment ($f(T_t) = \Lambda$), and Ricker's equation ($f(T_t) = T_t e^{r-kT_t}$)

2.1 Geometric Growth

In this case the recruitment function $f(T_t)$ is of the form $f(T_t) = \mu T_t$; i.e., the number of new individuals in generation $t + 1$ is proportional to the individuals present in generation t. Hence, the total population is governed

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by

$$T_{t+1} = (\mu + \gamma) T_t, \tag{2}$$

which implies geometric growth. Hence, the fate of the population depends on the value of $\mu + \gamma$, in other words, on the demographic basic reproductive number, $R_d = \frac{\mu}{1-\gamma}$ (a dimensionless quantity that gives the number of descendants produced by small pioneer populations over their life-time [2]). $R_d < 1$ implies that the population goes extinct; $R_d = 1$ implies that the population remains constant; and $R_d > 1$ implies unbounded geometric growth.

This recruitment function makes the system homogeneous of order one; hence the system can support geometric solutions. We use the homogeneity property to rescale the system using $x_t = \frac{S_t}{T_t}$, $y_t = \frac{E_t}{T_t}$, and $z_t = \frac{I_t}{T_t}$. The rescaled system becomes:

$$\left. \begin{aligned} x_{t+1} &= (1-q) + qG(z_t)x_t + q(1-\delta)z_t \\ y_{t+1} &= q(1-G(z_t))x_t + q\sigma y_t \\ z_{t+1} &= q(1-\sigma)y_t + q\delta z_t \end{aligned} \right\} \tag{3}$$

where $q = \frac{\gamma}{\mu+\gamma}$ and $x_t + y_t + z_t = 1$. Rescaling makes the analysis easier; but care must be excercised in the interpretation of results. It is important to note that while the actual number of a class of individuals may be approaching zero, the proportion may not. Similarly, the number of a class may approach infinity as the proportion goes to a value less than 1, including zero. Since, $x_t = 1 - y_t - z_t$, System (3) reduces to the following two dimensional system:

$$\left. \begin{aligned} y_{t+1} &= q(1-G(z_t))(1 - y_t - z_t) + q\sigma y_t \\ z_{t+1} &= q(1-\sigma)y_t + q\delta z_t \end{aligned} \right\}. \tag{4}$$

2.1.1   Equilibria and Stability

To analyze the stability of equilibria we use the Jury test [5]. The Jacobian for System (4) is

$$J(y, z) = q \begin{pmatrix} \sigma - (1 - G(z)) & -(1 - G(z)) - G'(z)(1 - y - z) \\ 1 - \sigma & \delta \end{pmatrix}. \tag{5}$$

The stability of the disease free equilibrium (d.f.e.), where the proportion (number) of infectious and exposed individuals is zero, is obtained from

$$J = J(0, 0) = q \begin{pmatrix} \sigma & -G'(0) \\ 1 - \sigma & \delta \end{pmatrix},$$

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and the Jury criteria. Since $trace(J) = q(\sigma + \delta) \geq 0$ and $\det(J) = q^2(\sigma\delta + G'(0)(1 - \sigma))$, then the d.f.e is locally stable whenever

$$q(\sigma + \delta) < 1 + q^2(\sigma\delta + G'(0)(1 - \sigma)) < 2,$$

or equivalently when

$$-q^2 G'(0)(1 - \sigma) < 1 + q^2\sigma\delta - q\delta - q\sigma < 2 - q^2 G'(0)(1 - \sigma) - q\delta - q\sigma.$$

Notice that the second part of the inequality is always true. Hence, the stability condition reduces to

$$\frac{-q^2 G'(0)(1 - \sigma)}{(1 - q\delta)(1 - q\sigma)} < 1.$$

Therefore, we can define the basic reproductive number R_0 as

$$R_0 = \frac{-q^2 G'(0)(1 - \sigma)}{(1 - q\delta)(1 - q\sigma)}, \tag{6}$$

and the condition for local asymptotic stability of the d.f.e is given by $R_0 < 1$.

R_0 is the number of secondary infections that an infectious individual produces when rare, that is, in a population of mostly susceptible individuals. To give an epidemiological interpretation of R_0 we first consider $R_d = 1$, that is, we exclude demographic considerations. In this case, $\frac{1}{1-q\delta} = \frac{1}{1-\gamma\delta}$, which is the death-adjusted number of generations that an individual stays in the infectious class before recovery or death; likewise, $\frac{1}{1-q\sigma} = \frac{1}{1-\gamma\sigma}$ is the death adjusted number of generations that an individual stays in the exposed class. We observe that $-q^2 G'(0)$ is the maximum rate of infection per individual [2]. If σ is close to 1, then $(1 - \sigma)$, the probability that an exposed becomes infectious is small, thus the number of infectious is reduced. Hence $(1 - \sigma)$ reduces the maximal infection rate, $-q^2 G'(0)$, per individual.

When $R_d \neq 1$, $\frac{1}{1-q\sigma}$ is the average number of generations that an individual who survives stays in the exposed class before becoming infectious; while $\frac{1}{1-q\delta}$ is the average number of generations that an infectious individual who survived takes to recover.

Theorem 2.1. *The disease free equilibrium is globally stable whenever it is locally stable.*

Proof. We will show this by exhibiting a function that meets the Lyapunov conditions for stability [6] .

Let $F : [0,1] \times [0,1] \to [0,1] \times [0,1]$ be defined by

$$F(y,z) = q((1 - G(z))(1 - y - z) + \sigma y, (1 - \sigma)y + \delta z),$$

that is, F is the reproduction function of System (4). Note that $F(0,0) = (0,0)$, *i.e.*, $(0,0)$ is a fixed point of F. Now, define $V : [0,1] \times [0,1] \to [0,1]$ by

$$V(y,z) = y - \frac{qG'(0)}{1 - q\delta} z$$

We will show that V is a Lyapunov function for F. Clearly, V is continuous on its domain and $V(0,0) = 0$. Moreover, $V(0,0) > 0 \ \forall (y,z) \neq (0,0)$. Since $F((0,1] \times \{0\}) \subset (0,1] \times (0,1]$ and $F(\{0\} \times (0,1]) \subset (0,1] \times (0,1]$, then to show global stability of $(0,0)$ it is sufficient to prove that $V(F(y,z)) < V(y,z)$ for $(y,z) \in (0,1] \times (0,1]$.

Now,

$$
\begin{aligned}
V[F(y,z)] &= q[(1 - G(z))(1 - y - z) + \sigma y] - q\frac{qG'(0)}{1 - q\delta}[(1 - \sigma)y + \delta z] \\
&\leq q(1 - G(z)) + q\left[\sigma - \frac{qG'(0)(1 - \sigma)}{1 - q\delta}\right]y - q\left[\frac{q\delta G'(0)}{1 - q\delta}\right]z \\
&\leq q\left[\sigma - \frac{qG'(0)(1 - \sigma)}{1 - q\delta}\right]y - qG'(0)\left[\frac{q\delta}{1 - q\delta} + 1\right]z \\
&= \frac{q\sigma(1 - q\delta) - q^2 G'(0)(1 - \sigma)}{1 - q\delta}y - \frac{qG'(0)}{1 - q\delta}z.
\end{aligned}
$$

In order for $V[F(y,z)] < V(y,z)$, we need to have

$$\frac{q\sigma(1 - q\delta) - q^2 G'(0)(1 - \sigma)}{1 - q\delta} < 1,$$

which is equivalent to

$$\frac{-q^2 G'(0)(1 - \sigma)}{(1 - q\delta)(1 - q\sigma)} < 1,$$

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and since

$$R_0 = \frac{-q^2 G'(0)\,(1-\sigma)}{(1-q\delta)\,(1-q\sigma)},$$

the condition for global stability of the d.f.e is

$$R_0 < 1.$$

Therefore, the disease free equilibrium is globally stable whenever it is locally stable. $\qquad\square$

Endemic Equilibrium In order to find conditions for existence and uniqueness of an endemic equilibrium we consider

$$
\begin{aligned}
y_\infty &= q(1 - G(z_\infty))(1 - y_\infty - z_\infty) + q\sigma y_\infty & (7)\\
z_\infty &= q(1-\sigma)y_\infty + q\delta z_\infty. & (8)
\end{aligned}
$$

From Equation (8)

$$z_\infty = B y_\infty, \tag{9}$$

where $B = \frac{q(1-\sigma)}{1-q\delta}$. Replacing Equation (9) into Equation (7) we get

$$(1 - q\sigma)y_\infty = q(1 - G(B y_\infty))(1 - (1 + B)y_\infty).$$

If we let

$$M(y_\infty) = (1 - q\sigma)y_\infty \tag{10}$$

and

$$H(y_\infty) = q(1 - G(B y_\infty))(1 - (1 + B)y_\infty), \tag{11}$$

then the existence of an endemic equilibrium (y_∞, z_∞) is established whenever these two functions intersect with $y_\infty \in (0, 1)$. We show the existence of a unique $y_\infty \in (0, 1)$ such that $M(y_\infty) = H(y_\infty)$. Since $M(y_\infty)$ is a line that passes through $(0, 0)$, then to find conditions for the existence of the intersection we need only analyze the behavior of $H(y_\infty)$.

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Note that $H(y_\infty)$ also passes through $(0,0)$, $H(y_\infty) \geq 0$ for $y_\infty \in (0, \frac{1}{1+B})$; and, $\lim_{y_\infty \to \infty} H(y_\infty) = -\infty$. Thus, we have at least one endemic equilibrium when $M'(0) < H'(0)$, *i.e.*, when

$$(1 - q\sigma) < -qBG'(0).$$

Hence, an endemic equilibrium exists when

$$\frac{-qBG'(0)}{(1 - q\sigma)} > 1,$$

that is, when $R_0 > 1$. Therefore, the existence of an endemic equilibrium brings instability to the d.f.e.

Note that when positive y_∞ exists, $y_\infty < 1$, since when $y_\infty > 0$, $M(y_\infty) > 0$, so we must have $H(y_\infty) > 0$, and thus we need

$$0 < 1 - (1 + B)y_\infty \quad \text{or} \quad y_\infty < \frac{1}{1 + B} < 1.$$

We observe that

$$H''(y_\infty) = q[2B(1 + B)G'(By_\infty) - B^2 G''(By_\infty)(1 - (1 + B)y_\infty)] < 0$$

since $G'(By_\infty) < 0$ and $G''(By_\infty) \geq 0$. Hence, $H(y_\infty)$ is concave down. This result implies uniqueness of y_∞.

We summarize these results in the following theorem:

Theorem 2.2. *If $R_0 > 1$, then there exists a unique endemic equilibrium of System (4).*

2.2 Constant Recruitment and Ricker's Equation

In this section we consider constant recruitment and Ricker recruitment. Ricker's equation allows for the possibility of fixing the disease free dynamics (demography) to various degrees of complexity (fixed points to chaos). Hence, it allows the possibility of studying whether or not the demography drives disease dynamics (Barrera *et. al.* [1]).

Hence, we consider the recruitment functions $f(T_t) = \Lambda$ and $f(T_t) = T_t e^{r - kT_t}$. These functions make System (1) nonhomogeneous. To simplify the analysis, we consider an equivalent limiting system, which qualitative

dynamics behave similar to the original system under some assumptions [14]. The limiting system is found by substituting T_t by $T_\infty = \lim_{t \to \infty} T_t$.

$$\left.\begin{array}{rcl} S_{t+1} & = & f(T_\infty) + \gamma G(\frac{I_t}{T_\infty})S_t + \gamma(1-\delta)I_t \\ E_{t+1} & = & \gamma(1 - G(\frac{I_t}{T_\infty}))S_t + \gamma\sigma E_t \\ I_{t+1} & = & \gamma(1-\sigma)E_t + \gamma\delta I_t \end{array}\right\} \tag{12}$$

where $T_\infty = \frac{\Lambda}{1-\gamma}$ for $f(T_t) = \Lambda$ and $T_\infty = \frac{r - \ln(1-\gamma)}{k}$ for $f(T_t) = T_t e^{r-kT_t}$. If $f(T_t) = \Lambda$, the total population at generation $t+1$ is given by $T_{t+1} = \Lambda + \gamma T_t$, and since $0 < \gamma < 1$, then T_∞ is always stable and positive. When $f(T_t) = T_t e^{r-kT_t}$, T_∞ is stable and positive whenever $0 \le r \le \frac{2}{1-\gamma} + \ln(1-\gamma)$.

Using the unjustified substitution, $S_t = T_\infty - E_t - I_t$ reduce the System 12 to

$$\left.\begin{array}{rcl} E_{t+1} & = & \gamma(1 - G(\frac{I_t}{T_\infty}))\left(T_\infty - E_t - I_t\right) + \gamma\sigma E_t \\ I_{t+1} & = & \gamma(1-\sigma)E_t + \gamma\delta I_t \end{array}\right\} \tag{13}$$

2.2.1 Equilibria and Stability

Consider the local stability of the disease free equiliibrium. The Jacobian matrix of System (13) at $(0,0)$ is

$$J(0,0) = \begin{pmatrix} \gamma\sigma & -\gamma G'(0) \\ \gamma(1-\sigma) & \gamma\delta \end{pmatrix}.$$

The Jury test implies that $(0,0)$ is locally asymptotically stable whenever the following inequality is satisfied

$$\gamma(\sigma + \delta) - \gamma^2(1-\sigma)G'(0) < 1 + \sigma\delta\gamma^2 < 2 - \gamma^2(1-\sigma)G'(0)$$

or, equivalently, when

$$-\gamma^2(1-\sigma)G'(0) < 1 + \sigma\delta\gamma^2 - \gamma(\sigma + \delta) < 2 - \gamma^2(1-\sigma)G'(0) - \gamma(\sigma + \delta).$$

The second part of this inequality is always true. Hence, the condition for the asymptotic local stability of $(0,0)$ for System (13) is

$$\frac{-\gamma^2(1-\sigma)G'(0)}{(1-\gamma\sigma)(1-\gamma\delta)} < 1,$$

and thus, we define

$$R_0 = \frac{-\gamma^2 (1 - \sigma) G'(0)}{(1 - \gamma\sigma)(1 - \gamma\delta)}. \tag{14}$$

The interpretation of R_0 in this case is analogous to that of (6) when $R_d = 1$.

Theorem 2.3. *The disease free equilibrium of System (13) is globally stable whenever it is locally asymptotically stable.*

Proof. The proof of this theorem is like that of Theorem 2.1. In this case the Lyapunov function is $V(E, I) = E - \frac{\gamma G'(0)}{1 - \gamma\delta} I$. □

Endemic Equilibrium To find conditions for the existence of an endemic equilibrium, consider

$$\left.\begin{array}{rcl} E_\infty & = & \gamma(1 - G(\frac{I_\infty}{T_\infty}))(T_\infty - E_\infty - I_\infty) + \gamma\sigma E_\infty \\ I_\infty & = & \gamma(1 - \sigma)E_\infty + \gamma\delta I_\infty \end{array}\right\} \tag{15}$$

System (15) is similar to the one obtained in section 2.1.1. The procedure to find conditions for the existence and uniqueness of the endemic equilibrium is similar. Thus we state the following result without further ado.

Theorem 2.4. *System (15) has a unique endemic equilibrium when $R_0 > 1$.*

The proof patterns the procedure of section 2.1.1. Now, I_∞ satisfies $0 < I_\infty < T_\infty$.

3 Examples

In this section we use specific forms of the probability function G to obtain conditions for the stability of the endemic equilibrium. First, we consider the probability that an encounter between a susceptible and an infectious does not produce a new exposed is given by a Poisson process. Thus the probability that a susceptible does not become exposed is given by $G\left(\frac{I_t}{T_t}\right) = e^{-\alpha\frac{I_t}{T_t}}$ (where α is a parameter that measures the impact of the proportion of infectives), as it was used in [2]. Although we have a specific function for

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G, we are still not able to find a specific value for the endemic equilibrium. So, we consider a simpler function, namely $G\left(\frac{I_t}{T_t}\right) = 1 - \frac{I_t}{T_t}$, obtained by searching for the simplest probablity function (random mixing). Clearly, $G\left(\frac{I_t}{T_t}\right) = e^{-\alpha \frac{I_t}{T_t}}$, and $G\left(\frac{I_t}{T_t}\right) = 1 - \frac{I_t}{T_t}$ satisfy the conditions given in Section 2: $G(0) = 1$, $G'(\frac{I_t}{T_t}) < 0$, $G''(\alpha \frac{I_t}{T_t}) \geq 0$, and $0 \leq G \leq 1$.

First we consider $G\left(\frac{I_t}{T_t}\right) = e^{-\alpha \frac{I_t}{T_t}}$. If $f(T_t) = \mu T_t$, then we substitute $G'(0)$ in (6) from Section 2.1.1 and we get that

$$R_0 = \frac{q^2 \alpha (1 - \sigma)}{(1 - q\delta)(1 - q\sigma)},$$

where $q = \frac{\gamma}{\mu + \gamma}$. Theorem 2.1 implies that $(1, 0, 0)$ is globally asymptotically stable when $R_0 < 1$. Theorem 2.2 implies that a unique endemic equilibrium exists when $R_0 > 1$. Simulations with Dynamics [12] show that different trajectories converge to a fixed positive equilibrium (see Figure 3). Now, if $f(T_t) = T_t e^{r - kT_t}$ or , $f(T_t) = \Lambda$, substituting $G'(0)$ in (14) gives

$$R_0 = \frac{\gamma^2 \alpha (1 - \sigma)}{(1 - \gamma\sigma)(1 - \gamma\delta)},$$

Theorems 2.3 and 2.4, implies that disease free equilibrium $(T_\infty, 0, 0)$ is globally stable whenever $R_0 < 1$, and guarantee the existence of a unique endemic equilibrium when $R_0 > 1$. Again the likelihood of the local stability of the unique endemic equilibrium is supported by simulations (Figure 3).

Now we consider $G\left(\frac{I_t}{T_t}\right) = 1 - \frac{I_t}{T_t}$ and $f(T_t) = \mu T_t$, then

$$R_0 = \frac{q^2 (1 - \sigma)}{(1 - q\delta)(1 - q\sigma)}. \tag{16}$$

The disease free equilibrium is globally asymptotically stable when $R_0 < 1$. In this case we can find the values for y_∞ and z_∞ explicitly, they are:

$$\begin{bmatrix} y_\infty \\ z_\infty \end{bmatrix} = \begin{bmatrix} \frac{\left(1 - R_0^{-1}\right)\left(\mu + \gamma(1 - \delta)\right)}{\gamma^2} \\ \frac{\gamma(1 - \sigma)\left(1 - R_0^{-1}\right)}{\gamma^2(2 - \sigma - \delta) + \mu} \end{bmatrix}. \tag{17}$$

If $f(T_t) = \Lambda$ or $f(T_t) = T_t e^{r - kT_t}$ then

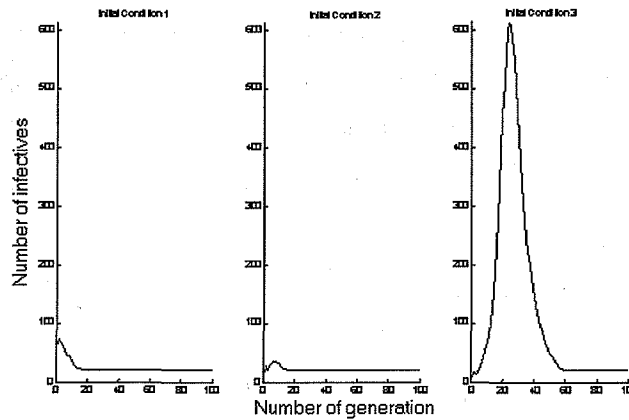$$R_0 = \frac{\gamma^2 (1 - \sigma)}{(1 - \gamma\sigma)(1 - \gamma\delta)}.$$

Figure 1: These are examples of how with different initial conditions the trajectories stabilize at the unique positive endemic state.

The disease free equilibrium is globally asymptotically stable whenever $R_0 < 1$. To find the unique endemic equilibrium we must solve System (15). Some algebra shows

$$
\begin{bmatrix} S_\infty \\ E_\infty \\ I_\infty \end{bmatrix} = \begin{bmatrix} T_\infty - E_\infty - I_\infty \\ \frac{(1-\gamma\delta)}{(1-\sigma)} \left(\frac{(1+\gamma^2\sigma\delta)(R_o-1)}{\gamma^2(1-\sigma)+\gamma(1-\gamma\delta)} \right) \frac{T_\infty}{\gamma} \\ \frac{(1+\gamma^2\sigma\delta)(R_o-1)}{\gamma(1-\sigma)+(1-\gamma\delta)} \frac{T_\infty}{\gamma} \end{bmatrix},
$$

where $T_\infty = \frac{\Lambda}{1-\gamma}$ for $f(T_t) = \Lambda$ and $T_\infty = \frac{r-\ln(1-\gamma)}{k}$ for $f(T_t) = T_t e^{r-kT_t}$.

4 Multipatch Model and Dispersal

The local dynamics of a single patch have been analyzed in the past sections; we have considered different recruitment functions as well as different probability functions for the rate of not becoming exposed to the disease. We found conditions for stability of the disease free equilibrium and for the existence of a unique endemic equilibrium. We also performed numerical simulations to support the local asymptotic stability of the endemic equilibrium.

Now we couple populations living in two patches via the dispersal of individuals. We are interested in exploring questions such as: Can dispersal help eradicate the disease in one patch? in all patches? Can dispersal of one

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class of individuals be relevant enough as to change the behavior of the total population? These questions are relevant because some diseases, like rabies, enhance the dispersal of the infected individuals [8]; while others diminish their capacity to disperse.

First we present analytical models for the dispersion of all individuals by using methods introduced by Hastings [9], and Castillo-Chavez and Yakubu [3]. We provide examples of how this general model can be adjusted to fit disease enhanced or disease suppressed dispersal. Finally we focus only on the dispersal of susceptibles via simulations.

4.1 General Dispersion Model. All Classes Disperse

Let $X_t{}^i$ be the population size of type X in patch i at time t, and let $\tilde{X}_t^i$ be the population in class X on patch i at time t after the local dynamics have occurred (right before dispersal occurs), i.e., assume that local dynamics occur before dispersal. Where $X = \{S, E, I\}$, and $i = \{1, 2, ..., N\}$ Hence,

$$\tilde{S}_t{}^i = f_i(T_t^i) + \gamma_i G_i(\tfrac{I_t^i}{T_t^i})S_t^i + \gamma_i(1 - \delta_i)I_t^i$$
$$\tilde{E}_t{}^i = \gamma_i(1 - G_i(\tfrac{I_t^i}{T_t^i}))S_t^i + \gamma_i\sigma_i E_t^i \qquad .$$
$$\tilde{I}_t{}^i = \gamma_i(1 - \sigma_i)E_t^i + \gamma_i\delta_i I_t^i$$

Then, the model where all classes disperse is

$$
\left.
\begin{aligned}
S_{t+1}^i &= (1 - \sum_{\substack{j=1 \\ j \neq i}}^{N} d_{ijS})\tilde{S}_t{}^i + \sum_{\substack{j=1 \\ j \neq i}}^{N} d_{jiS}\tilde{S}_t{}^j \\
E_{t+1}^i &= (1 - \sum_{\substack{j=1 \\ j \neq i}}^{N} d_{ijE})\tilde{E}_t{}^i + \sum_{\substack{j=1 \\ j \neq i}}^{N} d_{jiE}\tilde{E}_t{}^j \\
I_{t+1}^i &= (1 - \sum_{\substack{j=1 \\ j \neq i}}^{N} d_{ijI})\tilde{I}_t{}^i + \sum_{\substack{j=1 \\ j \neq i}}^{N} d_{jiI}\tilde{I}_t{}^j
\end{aligned}
\right\} \qquad (18)
$$

where d_{ijX} is the proportion of individuals in class X that disperse from patch i to patch j.

If we wish to consider disease suppressed dispersal, i.e., dispersal where only "healthy" individuals disperse, then $d_{ijE} = d_{ijI} = 0 \; \forall i, j = \{1, 2, ..., N\}$. Likewise, we consider disease enhanced dispersal, which arises from diseases where infectives or exposed are more likely to disperse; then $d_{ijS} = 0 \; \forall i$, $j = \{1, 2, ..., N\}$. To gain insight on the effects of dispersal on the dynamics of the disease we ran simulations using both MatLab and Dynamics.

4.2 Susceptibles Disperse: A Numerical Perspective

Consider System (1), and allow susceptibles to disperse between two patches. Then, the system that gives the population at generation $t + 1$ in patch i is

$$\left.\begin{aligned}
S_{t+1}^i &= (1 - d_i)[f_i(T_t^i) + \gamma_i G_i(\tfrac{I_t^i}{T_t^i})S_t^i + \gamma_i(1 - \delta_i)I_t^i] \\
&\quad + d_j[f_j(T_t^j) + \gamma_j G_j(\tfrac{I_t^j}{T_t^j})S_t^j + \gamma_j(1 - \delta_j)I_t^j] \\
E_{t+1}^i &= \gamma_i(1 - G_i(\tfrac{I_t^i}{T_t^i}))S_t^i + \gamma_i \sigma_i E_t^i \\
I_{t+1}^i &= \gamma_i(1 - \sigma_i)E_t^i + \gamma_i \delta_i I_t^i
\end{aligned}\right\} \text{Patch } i \qquad (19)$$

where $i = \{1, 2\}$, $j = \{1, 2\}$, $j \neq i$.

We consider the symmetric situation where the recruitment function and the probability of infection function are the same in both patches, i.e., $f_1 = f_2$ and $G_1 = G_2$.

Next, we show the results of simulations when $f(T_t) = \mu T_t$ and $G\left(\frac{I_t}{T_t}\right) = 1 - \frac{I_t}{T_t}$ in both patches. In this case we use the normalized System (4). To study the long run effects of dispersal on the disease, we consider different combinations of dispersal rates. We plot dispersal from patch 1 to patch 2 (d_1) versus dispersal from patch 2 to patch 1 (d_2). For every combination (d_1, d_2) we observe what happens to the proportions of infectious individuals in both patches. If the proportion is zero, then we call the patch disease free (DF); if it is not, then we call the patch endemic (E).

From simulations we observe that some combinations of dispersal produce changes in the disease dynamics. For example, dispersal causes the emergence of an endemic equilibrium even though $R_0 < 1$, or vice versa, an endemic equilibrium may disappear although $R_0 > 1$. These possibilities can be seen from regions clearly defined by lines (see Figures 4.2 and 4.2). However, when both patches have similar disease dynamics without dispersion, no combination of dispersal provokes a simultaneous change of behavior in both patches. For example, if both patches have an endemic equilibrium without dispersal then there are no dispersal values that produce simultaneous stable disease free equilibria. In addition if the disease dynamics are exactly the same in both patches; i.e., all parameters are equal then symmetric behavior is observed.

Our simulations have led to the following conjetures

Conjecture 4.1. *If* $R_{0_i} < 1$ *for all* $i \in \{1, 2\}$ *then the full two-patch system can not have an endemic equilibrium.*
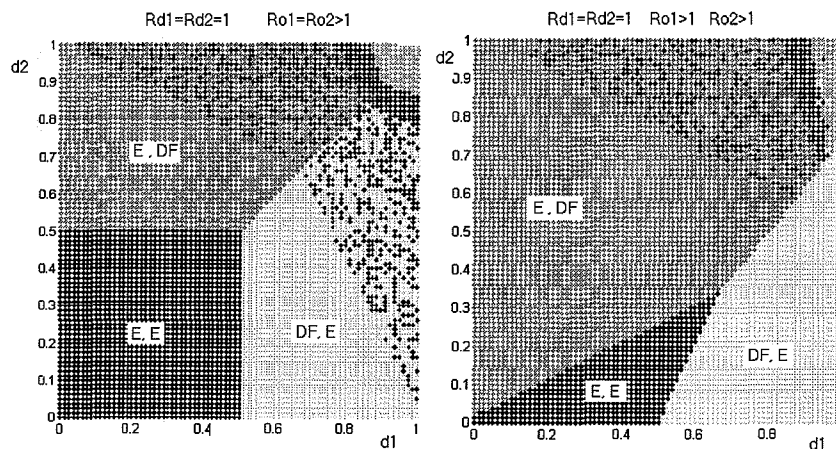
Figure 2: In this figure we observe symmetric behavior when disease and population dynamics are the same in both patches. The symmetry is broken when the R_{0_i}'s are different. The points in the upper right corner of both graphs are points that diverge or become negative; they are indicative of a Hopf Bifurcation.[4]

Conjecture 4.2. *If $R_{0_i} > 1$ for all $i \in \{1, 2\}$ then the full two-patch system can not have a stable disease free equilibrium.*

Ricker's Equation

Here we present the results of simulations that investigate the behavior of the total population and the population of infectives when susceptibles are allowed to disperse. We use the dispersion model (19), where $f(T_t) = T_t e^{r - kT_t}$ and $G\left(\frac{I_t}{T_t}\right) = 1 - \frac{I_t}{T_t}$.

The simulations show that the quantitative behavior of each patch does not change, that is, if a patch has a stable disease-free equilibrium in the absence of dispersion then, dispersion does not create an endemic state. However, the qualitative behavior of the patch does change. Dispersion creates multiple attractors or stabilizes chaotic behavior; our results agree with the results in [9] (see Figures 4.2 and 4.2).

In Figure 4.2 we compare the behavior of the total population and that of the infectious with and without dispersion. When there is no dispersion, the total population in patch 1 has chaotic behavior while the total population in patch 2 has period 3 (see Figure 4.2 (a) and (b) top). Also, the infectious
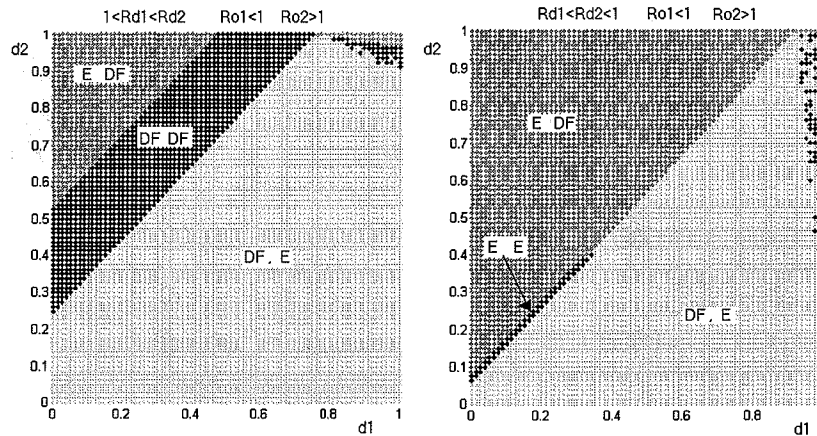
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Figure 3: Disease dynamics in both patches are different when there is not dispersion. In the left we see that for some combinations of dispersion the system becomes disease free, while in the right, the system develops an endemic equilibrium.

population of Patch 2 follows the dynamics of the total population (see Figure 4.2 (c) top), while the infectious population of Patch 1 is almost constant[1]. When dispersion is introduced (bottom Figure 4.2), we see that the behavior of Patch 1 stabilizes into a period 6 cycle and patch 2 undergoes a period doubling bifurcation. Hence, dispersion can stabilize chaotic behavior.

In addition to stabilizing chaotic behavior, dispersion is capable of creating multiple attractors [9]. An example of this is presented in Figure 4.2. Without dispersion, Patch 1 has chaotic behavior, while Patch 2 has periodic behavior. When dispersal is allowed, both patches support at least two attractors.

5 Conclusions

We have extended the discrete-time S-I-S model of Castillo-Chavez and Yakubu [2] to an S-E-I-S model. Our model allows for the study of diseases like respiratory infections. In the single patch S-E-I-S model, we obtain thresholds for the persistence of the disease. These thresholds differ from those obtained by Castillo-Chavez and Yakubu due to the presence of the exposed class. When
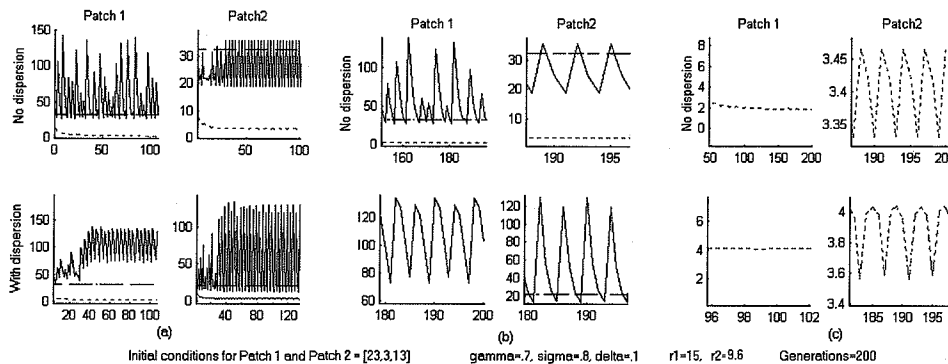
Figure 4: (a) top:behavior of Patch 1 and Patch 2 without dispersion. bottom: behavior of the patches with dispersion. (b) Details to see the behavior of the total populaiton. (c) Details to see the behavior of the infecitous in each patch.

comparing the basic reproductive numbers of both models we observe that the value corresponding to the S-E-I-S model is less than the one corresponding to the S-I-S model. Hence it is easier to eliminate the disease if there is an exposed class.

In the two patch S-E-I-S model with dispersion we obtain multiple attractors where, without dispersion there would not be any. We also observe that dispersal can stabilize chaotic behavior, as well create stable periodic attractors (without dispersion there would be chaos). These results agree with those obtained by Alan Hastings in a two patch ecological model without disease dynamics [7],[9]. The emergence of chaotic attractors due to dispersion gives opportunity for ecological diversity.

Moreover, when a population exhibits geometric growth, and there is a disease in a two patch system, dispersal can help a disease establish, where without dispersal the disease would perish. Likewise, dispersal can help eradicate a disease where without dispersal it would invade. However, when the two patch system has an endemic equilibrium in the absence of dispersion, dispersion can not free the system of disease and viceversa.
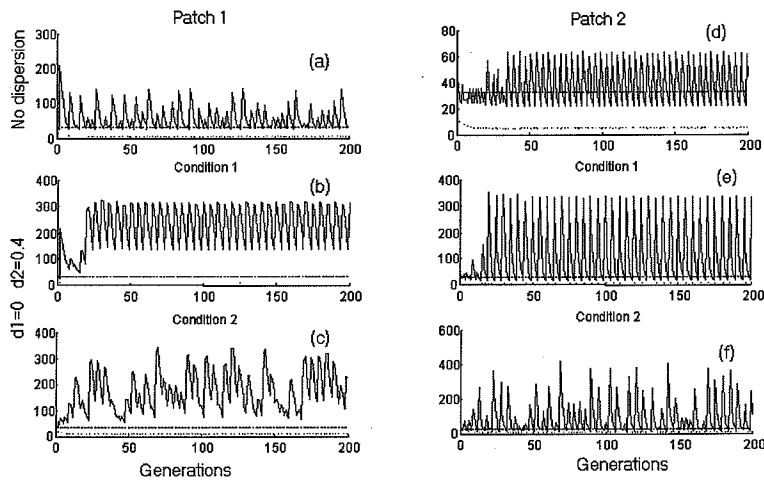
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Figure 5: Behavior of patches 1 and 2 with different initial conditions.

Appendix: MatLab Programs

```
function doublebif3(v0,w0,y0,z0,pts,c1,c2,c3,c4,c5,c6,c7,c8,its,fig)
figure;
hold on;
p1=[c1 c2 c3 c4];
p2=[c5 c6 c7 c8];
d1=linspace(0,1,pts);
d2=linspace(1,0,pts);
[D1,D2]=meshgrid(d1,d2);
V=v0.*ones(pts,pts);
W=w0.*ones(pts,pts);
Y=y0.*ones(pts,pts);
Z=z0.*ones(pts,pts);
for k=1:its
new_V=latent(D1,D2,V,W,Y,Z,p1,c6);
new_W=infected(D1,D2,V,W,Y,Z,p1);
new_Y=latent(D2,D1,Y,Z,V,W,p2,c2);
new_Z=infected(D2,D1,Y,Z,V,W,p2);
V=new_V;
```

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```
W=new_W;
Y=new_Y;
Z=new_Z;
end %for k
for j=1:pts
for i=1:pts
if (W(i,j)<0) | (Z(i,j)<0) %cuidado!
plot(D1(i,j),D2(i,j),'r.');
elseif(W(i,j)<=0.00000001) & (Z(i,j)<=0.00000001) %DF,DF
plot(D1(i,j),D2(i,j),'b.');
elseif(W(i,j)>0.00000001) & (Z(i,j)<=0.00000001) %E,DF
plot(D1(i,j),D2(i,j),'g.');
elseif (W(i,j)<=0.00000001) & (Z(i,j)>0.00000001) %DF,E
plot(D1(i,j),D2(i,j),'y.');
elseif (W(i,j)>0.00000001) & (Z(i,j)>0.00000001) % E,E
plot(D1(i,j),D2(i,j),'k.');
else
plot(D1(i,j),D2(i,j),'c.');
end %if
end %for i
end %for j
xlabel('d1');
ylabel('d2');
title(['Number ' num2str(fig)])
function t=latent(d1,d2,v,w,y,z,p,c)
t=p(1).*(w.*((1-d1).*(p(2)-v-w)+d2.*(c-y-z))./p(2)+p(4).*v);
function s=infected(d1,d2,v,w,y,z,p)
s=p(1).*((1-p(4)).*v+p(3).*w);
```

This program, tplot3, plots the trajectories that the populations follow as
time progresses.

```
function tplot3(u0,v0,w0,x0,y0,z0,c1,c2,c3,c4,c5,c6,c7,c8,c9,c10,D1,D2,its)
%Here u0, v0, w0, x0, y0, z0 are vectors of initial conditions
p1=[c1 c2 c3 c4 c9];
p2=[c5 c6 c7 c8 c10];
```

```
%c1-gamma1
%c2-r1
%c3-delta1
%c4-sigma1
%c9-k1
%c5-gamma2
%c6-r2
%c7-delta2
%c8-sigma2
%c10-k2

u=u0(1).*ones(1,its);
v=v0(1).*ones(1,its);
w=w0(1).*ones(1,its);
x=x0(1).*ones(1,its);
y=y0(1).*ones(1,its);
z=z0(1).*ones(1,its);
t1=ones(1,its);
t2=ones(1,its);

for k=1:(its-1)
t1(k)=u(k)+v(k)+w(k);
t2(k)=x(k)+y(k)+z(k);

u(k+1)=sucep(0,0,u(k),v(k),w(k),x(k),y(k),z(k),p1,p2,t1(k),t2(k));
v(k+1)=latent(u(k),v(k),w(k),p1,t1(k));
w(k+1)=infected(v(k),w(k),p1);
x(k+1)=sucep(0,0,x(k),y(k),z(k),u(k),v(k),w(k),p2,p1,t2(k),t1(k));
y(k+1)=latent(x(k),y(k),z(k),p2,t2(k));
z(k+1)=infected(y(k),z(k),p2);
end %for k

t1(its)=u(its)+v(its)+w(its);
t2(its)=x(its)+y(its)+z(its);

U=ones(3,its);
V=ones(3,its);
W=ones(3,its);
```

```
X=ones(3,its);
Y=ones(3,its);
Z=ones(3,its);
T1=ones(3,its);
T2=ones(3,its);

for j=1:3
U(j,:)=u0(j).*ones(1,its);
V(j,:)=v0(j).*ones(1,its);
W(j,:)=w0(j).*ones(1,its);
X(j,:)=x0(j).*ones(1,its);
Y(j,:)=y0(j).*ones(1,its);
Z(j,:)=z0(j).*ones(1,its);
end

for k=1:(its-1)
T1(:,k)=U(:,k)+V(:,k)+W(:,k);
T2(:,k)=X(:,k)+Y(:,k)+Z(:,k);
U(:,k+1)=sucep(D1,D2,U(:,k),V(:,k),W(:,k),X(:,k),
               Y(:,k),Z(:,k),p1,p2,T1(:,k),T2(:,k));
V(:,k+1)=latent(U(:,k),V(:,k),W(:,k),p1,T1(:,k));
W(:,k+1)=infected(V(:,k),W(:,k),p1);
X(:,k+1)=sucep(D2,D1,X(:,k),Y(:,k),Z(:,k),U(:,k),
               V(:,k),W(:,k),p2,p1,T2(:,k),T1(:,k));
Y(:,k+1)=latent(X(:,k),Y(:,k),Z(:,k),p2,T2(:,k));
Z(:,k+1)=infected(Y(:,k),Z(:,k),p2);
end %for k

T1(:,its)=U(:,its)+V(:,its)+W(:,its);
T2(:,its)=X(:,its)+Y(:,its)+Z(:,its);

t1inf=(c2-log(1-c1))/c9;
t2inf=(c6-log(1-c5))/c10;

T1inf=t1inf.*ones(1,its);
T2inf=t2inf.*ones(1,its);

figure;
```

```
hold on;
%plots for patch 1
subplot(421)
title('Patch 1')
hold on
plot(t1,'b') %CAREFUL! this only plots
plot(w,'r:') % one initial condition when d1=d2=0
plot(T1inf,'k--')
ylabel('No dispersion')
hold on;

subplot(423)
hold on
plot(T1(1,:),'b')
plot(W(1,:),'r:')
plot(T1inf,'k--')
title('Condition 1')
subplot(425)
hold on
plot(T1(2,:),'b')
plot(W(2,:),'r:')
plot(T1inf,'k--')
title('Condition 2')
ylabel(['d1 = ' num2str(D1) ', d2 = ' num2str(D2)])

subplot(427)
hold on
plot(T1(3,:),'b')
plot(W(3,:),'r:')
plot(T1inf,'k--')
title('Condition 3')

%plots for patch 2

subplot(422)
title('Patch2')
hold on;
plot(t2,'b')
```

```
plot(z,'r:')
plot(T1inf,'k--')

subplot(424)
hold on
plot(T2(1,:),'b')
plot(Z(1,:),'r')
plot(T2inf,'k--')
title('Condition 1')

subplot(426)
hold on
plot(T2(2,:),'b')
plot(Z(2,:),'r:')
plot(T2inf,'k--')
title('Condition 2')

subplot(428)
hold on
plot(T2(3,:),'b')
plot(Z(3,:),'r:')
plot(T2inf,'k--')
title('Condition 3')
legend(['Total' num2str(j)],['Infectives'num2str(j)])
legend('Total','Infectives')
%functions called in the program:

function r=sucep(d1,d2,u,v,w,x,y,z,p,q,t1,t2)
r=(1-d1).*(t1.*exp(p(2)-p(5).*t1)+
    p(1).*u.*(u+v)./t1+(1-p(3)).*p(1).*w)+
     d2.*(t2.*exp(q(2)-q(5).*t2)+
      q(1).*x.*(x+y)./t2+(1-q(3)).*q(1).*z);

function t=latent(u,v,w,p,t)
t=p(1).*(u.*w./t+p(4).*v);

function s=infected(v,w,p)
s=p(1).*((1-p(4)).*v+p(3).*w);
```

References

[1] Barrera, J. H., Cintron-Arias, A., Davidenko, N., Denogean, L. R., Franco-Gonzalez, S. R., Dynamics of a two-dimensional discrete-time SIS model, Biometrics Unit, Tech. Rep., BU-0000-M, Cornell University, 1999.

[2] Castillo-Chavez, C.,Yakubu, A. A., Discrete-time S-I-S models with complex dynamics, preprint.

[3] Castillo-Chavez, C., Yakubu A. A., Intraspecific competition, dispersal and disease dynamics in discrete-time patchy environments, *IMA*, to appear.

[4] Devaney, R.L., An introduction to chaotic dynamical systems, The Benjamin/Cummings Publishing Company, Inc. 1986.

[5] Edelstein-Keshet, L., *Mathematical models in biology*, McGraw-Hill, 1988.

[6] Elaydi, S. N., *An introduction to difference equations*, 2nd Ed., Springer-Verlag, 1999.

[7] Flores-Torres, E., Interaction between dispersal and dynamics: Coupled Ricker's Equation . Biometrics Unit, Tech. Rep.,MTBI Cornell University. 1999

[8] Hanski, I. A., Gilpin, M. E., *Metapopulation biology*, Academic Press, 1997.

[9] Hastings, A., Complex interactions between dispersal and dynamics: Lessons from coupled logistic equations, *Ecology*, **74** No.5 (1993), 1362-1372.

[10] May, R. M., *Stability and complexity in model ecosystems*, Princeton University Press, 1974.

[11] May, R. M., Simple mathematical models with very complicated dynamics, *Nature*, **261** (1977), 459-469.

[12] Nusse, H.E., Yorke, J. A., *Dynamics: numerical explorations*, 2nd Ed., Springer-Verlag, 1998.

[13] Strogatz, S. H., *Nonlinear dynamics and chaos*, 8th Ed., Perseus Books, 1998.

[14] Thieme, H. R., Convergence results and a Poincaré-Bendixon trichotomy for asymptotically autonomous differential equations, *J. Math. Biol.,* **30** (1992), 755-763.

[15] Velazquez, J. P., SIS nonlinear discrete-time models with two competing strains, Biometrics Unit, Tech. Rep., BU-0000-M, Cornell University, 1999.

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