HOST DEMOGRAPHIC ALLEE EFFECT, FATAL DISEASE, AND MIGRATION: PERSISTENCE OR EXTINCTION

AVNER FRIEDMAN† AND ABDUL-AZIZ YAKUBU‡

Abstract. In this paper, we focus on biodiversity, a major problem for ecosystem resilience. We use extensions of the susceptible-infected (SI) epidemic model of Hilker et al. to study how population persistence or extinction of a vulnerable species relates to habitat dependent Allee thresholds, fatal disease dynamics, and migration rates in both discrete and continuum sets of compartments. We analyze the migration-linked models and establish verifiable conditions that guarantee host population persistence (with or without infected individuals) or extinction.

Key words. Allee effect, extinction, migration, patches, persistence

AMS subject classifications. 34L30, 35B50, 35G30, 92D25, 92D30, 92D40

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1. Introduction. The more biologically diverse the web of life within an ecosystem, the more resilient it is; conversely, the less biologically diverse, the more fragile. According to recent estimates, scientists have named over 1.7 million species of animals, plants, and algae. However, they have yet to describe many other species of plants, invertebrate animals, and lichens, and the number of the species that are known to scientists continues to increase substantially every year. Tragically, as fast as these new species are being identified, others and their habitat are being destroyed at alarming rates [27], [29]. As a result, the world’s biodiversity is declining at an unprecedented rate. The list of recently extinct animals include the Tasmanian tiger (Thylacin), Plains zebra (Quagga), passenger pigeon or wild pigeon (Ectopistes migratorius), golden toad (Bufo periglenes), Caribbean monk seal (Monachus tropicalis), Pyrenean ibex (Capra pyrenaica pyrenaica), Bubal hartebeest (Alcelaphus buselaphus buselaphus), Javan tiger (Panthera tigris sondaica), Tecopa pupfish (Cyprinodon nevadensis calidae), and Baiji river dolphin (Lipotes vexillifer). In addition, dramatic declines have been observed in many species over the last few decades from locations all over the world. The list of rapidly declining species include Seychelles scops owl (Otus insularis), African wild dog (Lycaon pictus), crab-eating or long-tailed macaque (Macaca fascicularis), Verreaux’s sifaka (Propithecus verreauxi), and one-third of sea fisheries. These declines are perceived as one of the most critical threats to global biodiversity. Some of the causes of the species declines and extinctions include disease, habitat destruction and modification, exploitation, pollution, pesticide use, newly introduced species, and increased ultraviolet-B radiation (UV-B).

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†Mathematical Biosciences Institute and Mathematics Department, The Ohio State University, Columbus, OH 43210 (afriedman@math.osu.edu).

‡Department of Mathematics, Howard University, Washington, DC 20059 (ayakubu@howard.edu).
In this paper, we use mathematical models to study the impact of disease, animal migrations, and Allee effect on maintaining biodiversity. Transmission of infectious diseases in human and animal populations is not always homogeneous through an endemic geographical area but usually spotty and depends on two prime factors: local habitat (single patch) population dynamics and migration. Since most species are not restricted to one region, we are seeking to understand the “global” question of survival of species, that is, not just survival in one isolated region. This raises the question of how movement among animals between different locations affects their survival when a disease occurs in one or several locations. West Nile Virus, for example, spread rapidly along the East Coast of the U.S., most likely due to the movements of migratory birds.

As another example, Phocine and canine distemper viruses disease outbreaks pose one of the greatest global disease threats to wild carnivores including lions, African wild dogs, and several types of seals. In May of 2002, a distemper infection outbreak started in the Danish seal population at the Kattegat Isle of Anholt. The disease later spread to Sweden and Norway in the following month. A second outbreak was observed in mid-June in the Netherlands. Subsequently, the disease spread to Germany and Denmark in an eastern direction, and to Belgium, France, Great Britain, and Ireland to the West. In Germany, approximately 7,500 harbor seals died during the distemper virus disease epidemics [28].

Migration or diffusion between patches can have dramatic impact on local disease dynamics [3], [2], [4], [5], [6], [7], [8], [9], [10], [11], [12], [20], [21], [23], [26], [30]. Among the mathematical models that consider the Allee effect and disease dynamics [1], [2], [5], [8], [9], [15], [16], [14], [17], [18], [19], [22], [24], [25], [26], [30], [31], [32], [33], [34], models that incorporate the Allee effect in host demographics, disease dynamics, and migration or diffusion in a single model are very few. The Allee effect can be caused by difficulties in finding mating partners at small population densities, genetic inbreeding, demographic stochasticity, or a reduction in cooperative interactions [15]. In a recent paper, Friedman and Yakubu [22] used a single patch susceptible-infected (SI) epidemic model of Hilker et al. [25] without migration or diffusion to prove that if a healthy stable host population at the disease-free equilibrium is subject to the strong Allee effect, then a small number of infected individuals with a fatal disease can cause the host population to go extinct. To prove the single patch result, Friedman and Yakubu used the relative position of the disease threshold to the Allee threshold and to the host population carrying capacity to derive verifiable conditions that guarantee the persistence or the extinction of the host population. When the Allee effect occurs in the host demographics and a fatal disease invades the host population in a single patch with no migration, then the Allee threshold is effectively increased [1], [5], [6], [7], [8], [9], [10], [12], [13], [15], [16], [14], [19], [22], [24], [25], [26], [30].

In the absence of movement, a single patch is said to be low-risk if any small perturbation to the disease-free equilibrium leads to the host population persistence (with or without infected individuals) [3], [4]. The single patch is said to be high-risk if a small perturbation to the disease-free equilibrium leads to the host population extinction [3], [4]. In the present paper, we introduce two extensions of the SI epidemic model of Hilker et al. [25], an ODE SI epidemic model that consists of \( n \) dispersal-linked discrete sets of compartments and a PDE epidemic model with diffusion-linked continuum sets of compartments. We use the SI epidemic model extensions with migration to study the impact of spatial heterogeneity and habitat connectivity on the single patch results in [22]. In particular, we derive verifiable conditions that guarantee host population persistence (with or without infected individuals) or extinction in the migration-linked models. As in the single patch SI epidemic model without movement,
we show that when initial host population size on each patch is below the minimum patch Allee threshold, then the migration-linked (discrete or continuous) model leads to host population extinction.

Others have studied disease spread in epidemic models with either a discrete or continuum set of patches but without the demographic Allee effect [3], [4], [7], [11], [20], [21]. For example, using a two-patch SIS epidemic model without the Allee effect, Allen et al. [3], [4] considered the case where the disease persists in only one of the two patches, a high-risk patch. They showed that when the patches are linked by migration of the susceptible and infected individuals, then it is possible for the disease to persist in both patches of the dispersal-linked two-patch model. However, if the migration pattern is altered so that only infected individuals move between the two patches, then it is possible to drive all the infected individuals in the population extinction and persistence is a function of both migration pattern and initial host population sizes.

In section 3, we show that the n-dispersal-linked SI model with the strong Allee effect in the demographic equation.

This paper is organized as follows: In section 2, we introduce the ODE n-patch dispersal-linked SI model with the strong Allee effect in the demographic equation. In section 3, we show that the n-patch model is well posed. Conditions for host population extinction and persistence are derived in sections 4 and 5, respectively. In section 6, we introduce the PDE SI epidemic model with diffusion. Illustrative examples and concluding remarks are presented in sections 7 and 8, respectively.

2. SI epidemic patch model. To introduce the SI epidemic patch model, we let \( n \geq 2 \) be the number of patches and \( \Omega = \{1, 2, \ldots, n\} \). At time \( t \geq 0 \), in the absence of diffusion, the total population in patch \( j \in \Omega \) is

\[
p_j(t) = s_j(t) + i_j(t),
\]

where \( s_j(t) \) and \( i_j(t) \) denote the number of susceptible and infected individuals, respectively. In the presence of diffusion, the total population and infected individuals in each patch \( j \) are described by the following system of differential equations:

\[
\begin{align*}
\frac{dp_j}{dt} &= r_j(1 - p_j)(p_j - u_j)p_j - \alpha_j i_j + \delta \sum_{k \in \Omega} (L_{jk} p_k - L_{kj} p_j), \\
\frac{di_j}{dt} &= [-A_j + (\sigma_j - 1)p_j - \sigma_j i_j]i_j + \delta \sum_{k \in \Omega} (L_{jk} i_k - L_{kj} i_j),
\end{align*}
\]

where on each patch \( j \in \Omega \), \( A_j = \alpha_j + d_j + r_j u_j \), \( \delta \) is a positive diffusion coefficient for the total population, \( u_j \in (0, 1) \) is the Allee threshold, and \( L_{jk} \) is the degree of movement from patch \( k \) to patch \( j \). On each patch \( j \in \Omega \), the model parameters \( \alpha_j \), \( d_j \), \( r_j \), and \( \sigma_j \) are positive constants [25]. We make the following assumptions:

(A1) The matrix \( L = (L_{jk}) \) is symmetric, nonnegative, and irreducible, and \( L_{kk} = 0 \) for all \( k \in \Omega \). Thus, \( L_{jk} \geq 0 \) for all \( j, k \in \Omega \), and for any \( j, k \in \Omega \), \( j \neq k \), there exists a sequence \( j_1, j_2, \ldots, j_t \) such that

\[
L_{jj_1} > 0, \quad L_{jj_1j_2} > 0, \ldots, \quad L_{jj_{t-1}j_t} > 0, \quad L_{jj_k} > 0.
\]

(A2) We shall also impose throughout this paper the initial conditions

\[
0 \leq i_j(0) \leq p_j(0) \leq 1 \quad \text{for all} \quad j \in \Omega.
\]
When there is no diffusion between the patches, $\delta = 0$ and model (2.1) reduces to the following single patch model of Hilker et al. [25]:

\begin{align}
\frac{dp_j}{dt} &= r_j(1-p_j)(p_j-u_j)p_j - \alpha_j i_j, \\
\frac{di_j}{dt} &= [-A_j + (\sigma_j - 1)p_j - \sigma_j i_j]i_j.
\end{align}

When $\sigma_j \leq 1$ and there is no diffusion, then $\frac{di_j}{dt} < 0$ and the population of infectives decreases to zero. Consequently, we assume throughout the paper that $\sigma_j > 1$ for each patch $j \in \Omega$. In what follows, we shall use the notation $\vec{p}(t) = (p_1(t), p_2(t), \ldots, p_n(t))$ and $\vec{i}(t) = (i_1(t), i_2(t), \ldots, i_n(t))$.

### 3. Bounds and order of solutions

To indicate the well posedness of the patch model, we prove that if

\begin{equation}
0 \leq i_j(0) \leq p_j(0) \leq 1 \quad \text{for all } j \in \Omega,
\end{equation}

then

\begin{equation}
0 \leq i_j(t) \leq p_j(t) \leq 1 \quad \text{for all } t > 0 \quad \text{and} \quad j \in \Omega.
\end{equation}

More precisely, we have the following theorem.

**Theorem 3.1.**

\begin{itemize}
  \item[(3.3)] If $i_j(0) > 0$ for all $j \in \Omega$, then $i_j(t) > 0$ for all $t > 0$ and $j \in \Omega$.
  \item[(3.4)] If $p_j(0) < 1$ for all $j \in \Omega$, then $p_j(t) < 1$ for all $t > 0$ and $j \in \Omega$.
  \item[(3.5)] If $0 < i_j(0) \leq p_j(0)$ for all $j \in \Omega$, then $0 < i_j(t) \leq p_j(t)$ for all $t > 0$ and $j \in \Omega$.
\end{itemize}

The conclusion that (3.1) implies (3.2) then follows by approximation.

**Proof of (3.3).** If the assertion (3.3) is not true, then there exists a smallest $\tilde{t} > 0$ such that

\[ i_j(t) > 0 \quad \text{for all } t < \tilde{t}, \quad j \in \Omega \]

and

\[ i_j(\tilde{t}) = 0 \quad \text{if } j \in \Lambda \neq \emptyset \quad \text{and} \quad i_j(\tilde{t}) > 0 \quad \text{if } j \in \Lambda^c, \]

where $\Lambda \subset \Omega$ and $\Lambda^c = \Omega \setminus \Lambda$. Note that $\Lambda^c \neq \emptyset$, for otherwise $i_j(\tilde{t}) = 0$ for all $j \in \Omega$ and, by uniqueness, also $i_j(t) \equiv 0$ for all $t \geq 0$, which contradicts the assumption that $i_j(0) > 0$ in (A.2). Clearly,

\begin{equation}
\frac{di_j(\tilde{t})}{dt} \leq 0 \quad \text{for all } j \in \Lambda.
\end{equation}

On the other hand, by (2.1), if $j \in \Lambda$, then

\[ \frac{di_j(\tilde{t})}{dt} = \delta \sum_{k=1}^{n} (L_{jk}i_k(\tilde{t}) - L_{kj}i_j(\tilde{t})) = \delta \sum_{k \in \Lambda^c} L_{jk}i_k(\tilde{t}) \]
and \( i_k(\bar{t}) > 0 \). Hence, by (3.6), \( L_{jk} = 0 \) if \( j \in \Lambda \) and \( k \in \Lambda^c \), which is a contradiction to the assumption that \( (L_{jk}) \) is irreducible. \( \square \)

**Proof of (3.4).** If the assertion (3.4) is not true and \( j \in \Omega \), then there exists a smallest \( \bar{t} > 0 \) such that \( p_j(t) < 1 \) for all \( 0 < t < \bar{t}, j \in \Omega \), and \( p_j(\bar{t}) = 1 \) if \( j \in \Lambda, \Lambda \neq \emptyset \) and \( p_j(\bar{t}) < 1 \) if \( j \in \Lambda^c \). Clearly,

\[
\frac{dp_j(\bar{t})}{dt} \geq 0 \quad \text{for all } j \in \Lambda.
\]

Hence, by (2.1), if \( j \in \Lambda \), then

\[
\frac{dp_j(\bar{t})}{dt} = \alpha_j i_j(\bar{t}) + \delta \sum_{k=1}^{n} (L_{jk} p_k(\bar{t}) - L_{k,j} p_j(\bar{t})) = -\alpha_j i_j(\bar{t}) + \delta \sum_{k \in \Lambda^c} L_{jk}(p_k(\bar{t}) - 1).
\]

Recalling (3.7) we conclude that \( i_j(\bar{t}) = 0 \) and \( L_{jk} = 0 \) if \( j \in \Lambda \) and \( k \in \Lambda^c \). But since \( (L_{jk}) \) is irreducible, \( \Lambda^c = \emptyset \) and then, by uniqueness, \( p_j(t) \equiv 1, i_j(t) \equiv 0 \) for all \( j \in \Lambda, t \geq 0 \), which is a contradiction to the assumption in (3.4). \( \square \)

**Proof of (3.5).** If the assertion (3.5) is not true, then there exists a smallest \( \bar{t} > 0 \) such that \( i_j(t) < p_j(t) \) for all \( 0 < t < \bar{t}, j \in \Omega \), and

\[
i_i(\bar{t}) = p_i(\bar{t}) \quad \text{for some } l,
\]

(3.8)

\[
i_j(\bar{t}) \leq p_j(\bar{t}) \quad \text{for all } j \in \Omega.
\]

Then also

\[
\frac{d}{dt} \left( p_j(\bar{t}) - i_j(\bar{t}) \right) \leq 0.
\]

(3.10)

Since, \( A_l = r_l u_l + \alpha_l + d_l \), it follows from (2.1) for \( j = l \) and from (3.8)–(3.9) that

\[
\frac{d}{dt} \left( p_l(\bar{t}) - i_l(\bar{t}) \right) = r_l (1 - p_l(\bar{t})) (p_l(\bar{t}) - u_l) p_l(\bar{t}) - \alpha_l p_l(\bar{t}) + (A_l + p_l(\bar{t})) p_l(\bar{t}) + \delta \sum_{k=1}^{n} L_{lk}(p_k(\bar{t}) - i_k(\bar{t}))) > 0,
\]

which is a contradiction to (3.10). \( \square \)

**4. Host population extinction.** The set of disease-free equilibrium points of the single patch model without diffusion, model (2.3), are

\[
(p_{0j}, i_{0j}) = (0, 0), \quad (p_{1j}, i_{1j}) = (u_j, 0), \quad \text{and} \quad (p_{2j}, i_{2j}) = (1, 0).
\]

The corresponding disease-free equilibrium (DFE) points of the diffusion-linked \( n \)-patch, model (2.1), include

\[
\vec{0} = (0, 0, 0, \ldots, 0, 0)
\]

and

\[
\left( \vec{\gamma}, \vec{0} \right) = (1, 1, \ldots, 1, 0, 0, \ldots, 0).
\]
In model (2.3), due to the presence of the strong Allee effect, \((p_{0j}, i_{0j})\) is locally asymptotically stable. Furthermore, any solution of model (2.3) with \(p_j(0) < u_j\) satisfies

\[
p_j(t) \downarrow 0 \text{ as } t \to \infty.
\]

The following theorem establishes a similar result for model (2.1).

**Theorem 4.1 (host population extinction).** In model (2.1), assume that

\[
0 < p_j(0) + \varepsilon < u = \min_{1 \leq i \leq n} \{u_i\} \text{ for all } j \in \Omega
\]

for some \(\varepsilon > 0\). Then the solution

\[
\left( \vec{p}(t), \vec{i}(t) \right)
\]

satisfies

\[
p_j(t) < u - \varepsilon \text{ for all } t > 0 \text{ and } j \in \Omega.
\]

Moreover,

\[
\sum_{j \in \Omega} p_j(t) \leq C e^{-\gamma t},
\]

where \(C\) and \(\gamma\) are positive constants; hence

\[
\left( \vec{p}(t), \vec{i}(t) \right) \to \vec{0} \text{ as } t \to \infty.
\]

Thus, the discrete diffusion-linked population goes extinct whenever the smallest Allee threshold is far from each initial local patch population. This dynamical behavior is driven by the Allee effect alone and is independent of the disease epidemics. That is, with or without migration between the patches, at small population sizes each patch \(j \in \Omega\) is a high-risk patch.

**Proof.** We claim that

\[
p_j(t) < u - \varepsilon \text{ for all } t > 0 \text{ and } j \in \Omega.
\]

Indeed, otherwise there exists a smallest \(\tilde{t} > 0\) and a nonempty subset \(\Lambda\) of \(\Omega\) such that

\[
p_k(\tilde{t}) = u - \varepsilon \text{ for all } k \in \Lambda \text{ and } p_j(t) < u - \varepsilon \text{ for all } t < \tilde{t} \text{ and } j \in \Omega \setminus \Lambda.
\]

This implies that

\[
\frac{dp_k(\tilde{t})}{dt} \geq 0 \text{ for all } k \in \Lambda.
\]

However, by model (2.1), we have

\[
\frac{dp_k(\tilde{t})}{dt} = r_k(1 - p_k(\tilde{t}))(p_k(\tilde{t}) - u_k)p_k(\tilde{t}) - \alpha_k i_k + \delta \sum_{l \in \Omega}(L_{kl}p_l(\tilde{t}) - L_{lk}p_k(\tilde{t})),
\]

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and if \( k \in \Lambda \),
\[
 r_k (1 - p_k (\bar{\bar{t}})) (p_k (\bar{\bar{t}}) - u_k) p_k (\bar{\bar{t}}) = r_k (-\varepsilon) (1 - \bar{u} + \varepsilon) (\bar{u} - \varepsilon) < 0.
\]
Furthermore, since \( p_l (\bar{\bar{t}}) \leq p_k (\bar{\bar{t}}) \) for all \( l \in \Omega \),
\[
 \delta \sum_{l \in \Omega} (L_{kl} p_l (\bar{\bar{t}}) - L_{lk} p_k (\bar{\bar{t}})) = \delta \sum_{l \in \Omega} L_{kl} (p_l (\bar{\bar{t}}) - p_k (\bar{\bar{t}})) \leq 0.
\]
However, \( \frac{dp_k (\bar{\bar{t}})}{dt} < 0 \), which is a contradiction to (4.1).

Now, let
\[
P(t) = \sum_{j \in \Omega} p_j (t).
\]
Then
\[
 P'(t) = \sum_{j \in \Omega} \frac{dp_j (t)}{dt} = \sum_{j \in \Omega} (r_j (1 - p_j) (p_j - u_j) p_j - \alpha_j i_j) < -\varepsilon \bar{t} (1 - \bar{u}) \sum_{j \in \Omega} p_j (t),
\]
where \( \bar{t} = \min_{1 \leq j \leq n} \{ r_j \} \). Hence,
\[
 \sum_{j \in \Omega} p_j (t) < \left( \sum_{j \in \Omega} p_j (0) \right) e^{-\varepsilon \bar{t} (1 - \bar{u}) t},
\]
and by (3.4), \( 0 < i_j (t) < p_j (t) \) for all \( t > 0 \) and \( j \in \Omega \). Consequently,
\[
 \left( \vec{p} (t), \vec{i} (t) \right) \to \vec{0} \text{ as } t \to \infty.
\]

Using the single patch model with no diffusion, model (2.3), Friedman and Yakubu [22] showed that host population extinction is possible on each local patch \( j \in \Omega \) whenever \( p_j (0) > u_j \). To state the single patch result, we introduce the patch \( j \in \Omega \) disease threshold,
\[
 (4.2) \quad P_{Tj} = \frac{A_j}{\sigma_j - 1},
\]
and the patch \( j \in \Omega \) basic reproduction number,
\[
 (4.3) \quad R_{0j} = \frac{\sigma_j}{A_j + 1}.
\]
In model (2.3), \( P_{Tj} \) is the point at which the linear infecteds nullcline crosses the horizontal axis. \( P_{Tj} > 1 \) is equivalent to \( R_{0j} < 1 \), and \( 0 < P_{Tj} < 1 \) is equivalent to \( R_{0j} > 1 \). Note that \( R_{0j} < 1 \) if and only if \( \lambda_{0j} = -A_j + \sigma_j - 1 < 0 \), where \( \lambda_{0j} \) is the largest eigenvalue of the Jacobian matrix of model (2.3) at the DFE (1, 0).

If \( R_{0j} < 1 \), then the DFE (1, 0) of model (2.3) is asymptotically stable and patch \( j \) is low-risk at high densities, whereas if \( R_{0j} > 1 \), then (1, 0) is not stable, and patch \( j \) is high-risk at both low and high densities. In order to determine the asymptotic stability of the DFE \((\bar{\bar{T}}, \bar{\bar{O}}) \) of model (2.1), the migration-linked model, we consider the Jacobian matrix evaluated at \((\bar{\bar{T}}, \bar{\bar{O}}) \), \( J_{(\bar{\bar{T}}, \bar{\bar{O}})} \), which is a symmetric matrix, and compute its maximal eigenvalue, \( \lambda_{0j} \).
If $\delta$ is small, then, as can be easily computed,

$$\lambda_\delta = \max_{j \in \Omega} \{\lambda_{0j} + \delta L_j\} + O(\delta^2),$$

where $L_j = \sum_{k \in \Omega} L_{kj}$. Hence, $\lambda_\delta > 0$ if $\max_{j \in \Omega} \lambda_{0j} > 0$ (or if $\max_{j \in \Omega} R_{0j} > 1$). For general $\delta$, the eigenvalues of $J(\mathbf{1}, \mathbf{0})$ are the roots of the characteristic equations

$$M \equiv \begin{vmatrix} m_1 - \lambda & \delta L_{12} & \delta L_{13} & \cdots & \delta L_{1n} \\ \delta L_{12} & m_2 - \lambda & \delta L_{23} & \cdots & \delta L_{2n} \\ \cdots & \cdots & \cdots & \cdots & \cdots \\ \delta L_{1n} & \delta L_{2n} & \cdots & \cdots & m_n - \lambda \end{vmatrix} = 0,$$

and

$$N \equiv \begin{vmatrix} n_1 - \lambda & \delta L_{12} & \delta L_{13} & \cdots & \delta L_{1n} \\ \delta L_{12} & n_2 - \lambda & \delta L_{23} & \cdots & \delta L_{2n} \\ \cdots & \cdots & \cdots & \cdots & \cdots \\ \delta L_{1n} & \delta L_{2n} & \cdots & \cdots & n_n - \lambda \end{vmatrix} = 0,$$

where for each $j \in \Omega$, $m_j = -r_j(1 - u_j) - \delta \sum_{k \in \Omega} L_{kj}$ and $n_j = -A_j + \sigma_j - 1 - \delta \sum_{k \in \Omega} L_{kj}$. If $n = 2$, the roots of $M$ are negative, and the maximal root of $N$ is

$$\frac{(n_1 + n_2) + \sqrt{(n_1 - n_2)^2 + 4(\delta L_{12})^2}}{2},$$

which is easily seen to be larger than $\max_{j \in \Omega} \lambda_{0j}$. We conjecture that, in general,

$$\lambda_\delta > \max_{j \in \Omega} \lambda_{0j}.$$

For future reference we state the following result.

**Theorem 4.2.** If

$$\lambda_\delta > 0,$$

then the DFE, $(\mathbf{1}, \mathbf{0})$, of model (2.1) is not stable. That is, there exists a small neighborhood $V$ of $(\mathbf{1}, \mathbf{0})$ such that if $(\mathbf{p}(0), \mathbf{i}(0))$ does not lie on either the local stable or center manifold of $(\mathbf{1}, \mathbf{0})$, then

$$\left(\mathbf{p}(t), \mathbf{i}(t)\right) \notin V \text{ for all } t > 0,$$

provided that $(\mathbf{p}(0), \mathbf{i}(0)) \notin V$.

In single patch models, it is known that a small number of infected individuals can lead to host population extinction when there is an Allee effect in the host demographics [19], [22]. However, most species migrate to take advantage of food, shelter, and water, which vary with seasons, or life stage. Examples include salmon migrating thousands of miles back to their spawning grounds, huge flocks of sandhill cranes migrating across the northern skies, or caribou crossing rivers in the fall. Thus, in order to protect biodiversity we must be very careful to prevent any fatal disease infection among species when the Allee effect is present. When the species is spread over several
different regions with some movement among them, the question is how careful must
we be in order to prevent a collapse of the entire species under initial small infection
in some of the regions. The next theorem gives a condition under which any small
infection leads to collapse of the total population. In an earlier paper, we considered
such a problem for one isolated region with no migration. Here, the condition we give
involves not each region separately, but rather all the regions at once. This condi-
tion is satisfied if on each patch \( j \in \Omega \), the disease transmission rate, \( \alpha_j \), and Allee
threshold, \( u_j \), are large while the local intrinsic per-capita growth rate, \( r_j \), and disease
threshold, \( P_{Tj} \), are small, while \( \min_{j \in \Omega} R_{0j} > 1 \). That is, independent of initial pop-
ulation sizes, the species goes extinct in the discrete dispersal-link model whenever
the “global” disease transmission rate is high while the “global” intrinsic per-capita
growth rate is small, while the Allee thresholds are large and \( \min_{j \in \Omega} R_{0j} > 1 \).

**Theorem 4.3** (host population extinction). In model (2.1), assume that
\[
\lambda_0 > 0
\]
and, for any \( l \in \Omega \), if \( y_l < y_l < 1 \) and \( 0 < y_j < 1 \) for all \( j \in \Omega, j \neq l \), then
\[
\sum_{j \in \Omega} \left\{ r_j(1 - y_j)(y_j - u_j)y_j - \frac{\alpha_j(\sigma_j - 1)}{\sigma_j}(y_j - P_{Tj}) \right\} < 0.
\]
Then every solution of model (2.1) satisfies
\[
\left( \overrightarrow{p}(t), \overrightarrow{i}(t) \right) \rightarrow \overrightarrow{0} \text{ as } t \rightarrow \infty,
\]
provided that \( (\overrightarrow{p}(0), \overrightarrow{i}(0)) \) does not lie on either the (local) stable or center manifolds
of \( (\overrightarrow{1}, \overrightarrow{0}) \).

Remark. Friedman and Yakubu [22] proved that in model (2.3), if
\[
0 < P_{Tj} < u_j \text{ for all } j \in \Omega
\]
and
\[
\max_{u_j \leq y_j \leq 1} \left\{ r_j(1 - y_j)(y_j - u_j)y_j - \frac{\alpha_j(\sigma_j - 1)}{\sigma_j}(y_j - P_{Tj}) \right\} \leq \varepsilon
\]
for some sufficiently small \( \varepsilon > 0 \), then every solution of model (2.3) with \( 1 - \eta < p_j(0) \leq 1 \) for any sufficiently small \( \eta > 0 \) and \( i_j(0) > 0 \) satisfies
\[
p_j(t) \rightarrow 0 \text{ and } i_j(t) \rightarrow 0 \text{ as } t \rightarrow \infty.
\]
That is, \((1, 0)\) is not stable and each patch \( j \in \Omega \) is high-risk at both low and high
densities whenever condition (4.7) holds.

Condition (4.7) means that the \( i_j \)-nullcline lies “almost completely” above the
\( p_j \)-nullcline for all \( u_j < p_j < 1 \). Similarly, condition (4.6) means that the \( i_j \)-nullcline
lies “well” above the \( p_j \)-nullcline. This condition is satisfied, for example, if \( \frac{\alpha_j}{A_j} \) and
\( \frac{\sigma_j}{\bar{r}_j} \) are sufficiently large.

**Proof of Theorem 4.3.** By Theorem 4.2, we can choose a small neighborhood \( V \)
of \( (\overrightarrow{1}, \overrightarrow{0}) \) such that
\[
\left( \overrightarrow{p}(t), \overrightarrow{i}(t) \right) \notin V \text{ for all } t > 0.
\]
For \( \varepsilon > 0 \) sufficiently small, let \( \tilde{U}_\varepsilon \) denote the domain
\[
\left\{ \left( \vec{p}, \vec{i} \right) : \vec{u} - \varepsilon < p_j < 1, \ 0 < i_j < p_j \text{ for at least one } j \in \Omega \right\}
\]
and set
\[
U_\varepsilon = \tilde{U}_\varepsilon \setminus V.
\]
Consider the function
\[
Q(t) = Q \left( \vec{p}(t), \vec{i}(t) \right) = \sum_{j \in \Omega} \left( p_j(t) - \ln(i_j(t)) \right)
\]
on \( U_\varepsilon \). We note that
\[
(4.9)
\]
\[
Q(t) \geq -Q_0 \text{ (} Q_0, \text{ a positive constant) for all } t \geq 0 \text{ such that } \left( \vec{p}(t), \vec{i}(t) \right) \in U_\varepsilon
\]
and
\[
\frac{dQ}{dt} = \sum_{j \in \Omega} \left( \frac{dp_j(t)}{dt} - \frac{1}{i_j} \frac{di_j(t)}{dt} \right) \equiv B_1 - \delta B_2,
\]
where
\[
B_1 = \sum_{j \in \Omega} \left( (r_j(1 - p_j)(p_j - u_j)p_j - \alpha_ji_j) + A_j - (\sigma_j - 1)p_j + \sigma_ji_j \right)
\]
and
\[
B_2 = \sum_{j \in \Omega} \left( \sum_{k \in \Omega} \frac{(L_{jk}i_k - L_{kj}i_j)}{i_j} \right).
\]
By (4.6), if \( \varepsilon \) is sufficiently small, then \( B_1 \leq -\delta < 0 \) in \( U_\varepsilon \), where \( \delta \) is a positive constant. For each pair \((j, k)\), the terms
\[
\frac{(L_{jk}i_k - L_{kj}i_j)}{i_j} + \frac{(L_{jk}i_j - L_{kj}i_k)}{i_k}
\]
from \( B_2 \) have the form
\[
L_{jk} \left( \frac{x}{x - 1} \right) + L_{jk} \left( \frac{1}{x} - 1 \right) = L_{jk} \left( x + \frac{1}{x} - 2 \right) \geq 0,
\]
where \( x = \frac{i_k}{i_j} \geq 0 \). Hence, \( -\delta B_2 \leq 0 \). We conclude that along the trajectory \((\vec{p}(t), \vec{i}(t))\),
\[
\frac{dQ}{dt} \left( \vec{p}(t), \vec{i}(t) \right) \leq -\delta
\]
as long as \((\vec{p}(t), \vec{i}(t))\) remains in \( U_\varepsilon \). So, if \((\vec{p}(t), \vec{i}(t))\) remains in \( U_\varepsilon \) for all \( t \geq 0 \), then \( Q(t) \rightarrow -\infty \) as \( t \rightarrow \infty \), which is a contradiction to (4.9). Also, by (4.8), \((\vec{p}(t), \vec{i}(t))\) does not enter the \( V \)-neighborhood of \((\vec{1}, \vec{1})\). Hence, there exists a \( \tilde{t} > 0 \) such that \((\vec{p}(\tilde{t}), \vec{i}(\tilde{t})) \) exists in \( \tilde{U}_\varepsilon \setminus V \) and
\[
p_j(\tilde{t}) < \vec{u} - \varepsilon \text{ for all } j \in \Omega.
\]
By Theorem 4.1 it then follows that \((\vec{p}(t), \vec{i}(t)) \rightarrow \vec{0} \) as \( t \rightarrow \infty \). □
5. Host population persistence with and without infecteds. In the absence of disease and migration, a major consequence of the strong Allee effect is the existence of a critical threshold, the Allee threshold, below which the population is likely to go extinct while persistence is possible at population densities above the Allee threshold. In this section, we show that with or without the explicit fatal disease dynamics, migration intensity and initial population densities interact to promote species persistence.

We first consider the disease-free model (2.3),

\[ \frac{dp_j}{dt} = r_j (1 - p_j)(p_j - u_j)p_j + \delta \sum_{k \in \Omega} (L_{jk}p_k - L_{kj}p_j), \quad j \in \Omega, \]

**Theorem 5.1 (population persistence).** In model (5.1), assume

\[ \overline{\mu} + \varepsilon < p_j(0) < 1 \quad \text{for all} \quad j \in \Omega \]

for some \( \varepsilon > 0 \), where \( \overline{\mu} = \max_{1 \leq i \leq n} \{u_i\} \). Then the solution

\[ (p_1(t), p_2(t), \ldots, p_n(t)) \]

satisfies

\[ \overline{\mu} + \varepsilon < p_j(t) \quad \text{for all} \quad t > 0 \quad \text{and} \quad j \in \Omega. \]

By Theorem 5.1, in the absence of the disease, the diffusion-linked population persists uniformly whenever each initial local patch population exceeds the biggest Allee threshold. Thus, migrating populations that are subjected to a strong Allee effect might be less vulnerable to “global” extinction if conservation efforts are successful on each local habitat.

**Proof.** If the assertion of the theorem is not true, then there exist a smallest \( \tilde{t} > 0 \) and a subset \( \Lambda \) of \( \Omega \), \( \Lambda \neq \emptyset \), such that

\[ p_k(\tilde{t}) = \overline{\mu} + \varepsilon \quad \text{for all} \quad k \in \Lambda \quad \text{and} \quad \overline{\mu} + \varepsilon < p_j(t) \quad \text{for all} \quad t < \tilde{t} \quad \text{and} \quad j \in \Omega \setminus \Lambda. \]

This implies that

\[\frac{dp_k(\tilde{t})}{dt} \leq 0 \quad \text{for all} \quad k \in \Lambda.\]

On the other hand,

\[ \frac{dp_k(\tilde{t})}{dt} = r_k (1 - p_k(\tilde{t}))(p_k(\tilde{t}) - u_k)p_k(\tilde{t}) + \delta \sum_{l \in \Omega} (L_{kl}p_l(\tilde{t}) - L_{lk}p_k(\tilde{t})), \]

\[ r_k (1 - p_k(\tilde{t}))(p_k(\tilde{t}) - u_k)p_k(\tilde{t}) = r_k \varepsilon (1 - \overline{\mu} - \varepsilon) (\overline{\mu} + \varepsilon) > 0, \]

and

\[ \delta \sum_{l \in \Omega} (L_{kl}p_l(\tilde{t}) - L_{lk}p_k(\tilde{t})) = \delta \sum_{l \in \Omega} L_{lk} (p_l(\tilde{t}) - (\overline{\mu} + \varepsilon)) \geq 0. \]

Hence, \( \frac{dp_k(\tilde{t})}{dt} > 0 \), which is a contradiction of (5.2). \( \Box \)
In contrast to the extinction result, Theorem 4.3, if the Allee thresholds are small and each initial local patch population exceeds the biggest Allee threshold, we obtain in Theorem 5.2 that the population persists (with or without infected individuals) on each patch $j \in \Omega$ of the dispersal-linked model (2.1) whenever the disease transmission rate, $\alpha_j$, is small while the local intrinsic per-capita growth rate, $r_j$, is large, and $\min_{j \in \Omega} R_{0j} > 1$.

**Theorem 5.2 (host population persistence).** In model (2.1), assume that

$$\lambda_{S} > 0,$$

$$\bar{\pi} + \varepsilon < p_j(0) \leq 1 \text{ for all } j \in \Omega,$$

and

$$\min_{j \in \Omega} \left[ r_j (1 - u_j)(y_j - u_j)y_j - \alpha_j y_j \right]_{y_j = \bar{\pi} + \varepsilon} > 0 .$$

Then the model solution

$$\left( \overrightarrow{p}(t), \overrightarrow{i}(t) \right)$$

satisfies

$$\bar{\pi} + \varepsilon < p_j(t) \text{ for all } t > 0 \text{ and } j \in \Omega.$$

**Proof.** We proceed as in the proof of Theorem 5.1, but use the fact that $i_j(\tilde{t}) < p_j(\tilde{t})$ and (5.3) to get a contradiction as before.

In the next theorem, we prove persistence of the infected individuals, assuming, in addition to (5.3), that

$$\sigma_j - 1) \bar{\pi} > A_j \text{ for all } j \in \Omega.$$

That is, we prove that if the Allee thresholds are large and each initial local patch population density exceeds the biggest Allee threshold, then the population persists (with infected individuals) on each patch $j \in \Omega$ of the discrete dispersal-linked model (2.1) whenever the disease transmission rate, $\alpha_j$, is small while the local intrinsic per-capita growth rate, $r_j$, is large, while $\min_{j \in \Omega} R_{0j} > 1$.

**Theorem 5.3 (disease persistence).** In model (2.1), if

$$\lambda_{S} > 0,$$

$$\bar{\pi} + \varepsilon < p_j(0) \leq 1 \text{ for all } j \in \Omega,$$

$$\sum_{i \in \Omega} i_l(0) > 0,$$

and conditions (5.3), (5.4) are satisfied, then the solution

$$\left( \overrightarrow{p}(t), \overrightarrow{i}(t) \right)$$
satisfies
\[ \sum_{l \in \Omega} i_l(t) \geq \eta > 0 \text{ for all } t \geq 0, \]
where \( \eta \) is a positive constant.

**Proof.** Let
\[ I(t) = \sum_{l \in \Omega} i_l(t). \]
It is enough to show that there exists \( \varepsilon_0 > 0 \) such that, for any \( t > 0 \), if \( 0 < I(t) < \varepsilon_0 \), then
\[ (5.5) \quad I'(t) \geq \gamma I(t), \quad \gamma > 0, \]
where \( \gamma = \frac{1}{2} \min_{l \in \Omega} (\sigma_l - 1) \) is a positive constant independent of \( t \). By Theorem 5.2, \( p_j(t) > \bar{\pi} + \varepsilon \) for all \( t > 0 \) and some \( \varepsilon > 0 \), so that
\[
\frac{dI}{dt} = \sum_{l \in \Omega} [-A_l + (\sigma_l - 1)p_l(t) - \sigma_l \varepsilon_0] i_l(t) \\
> \sum_{l \in \Omega} [-A_l + (\sigma_l - 1)(\bar{\pi} + \varepsilon) - \sigma_l \varepsilon_0] i_l(t) \\
\geq \sum_{l \in \Omega} [(\sigma_l - 1)\varepsilon - \sigma_l \varepsilon_0] i_l(t) \quad \text{by (5.4)}. \]
Choosing
\[ \varepsilon_0 = \frac{1}{2} \min_{l \in \Omega} \frac{\sigma_l - 1}{\sigma_l} \varepsilon, \]
the assertion (5.5) follows. \( \square \)

By Theorems 4.1 and 5.3, some patch \( l \in \Omega \) is high-risk at both low and high densities whenever \( \lambda_l > 0 \) and both conditions (5.3) and (5.4) hold.

**6. SI epidemic PDE model.** In this section, we extend the results of the previous sections to the case where instead of the discrete set of compartments of model (2.1),
\[
\left( \begin{array}{c} \vec{p}(t) \\ \vec{i}(t) \end{array} \right),
\]
we have a continuum
\[
(p(x,t), i(x,t)),
\]
where \( x \) varies in a bounded domain \( G \) in \( \mathbb{R}^n \) (\( n \geq 1 \)) with boundary \( \partial G \). In this case, the movement among compartments is replaced by the dispersion operator
\[ \Delta = \sum_{j=1}^{n} \frac{\partial^2}{\partial x_j^2}, \]
where \( x = (x_1, x_2, \ldots, x_n) \). Thus, we have
\[
(6.1) \quad \begin{cases}
\frac{\partial p}{\partial t} - \Delta p = r(x)(1-p)(p-u(x))p - \alpha(x)i, \\
\frac{\partial i}{\partial t} - \Delta i = [-A(x) + (\sigma(x) - 1)p - \sigma(x)]i,
\end{cases}
\]
where \( p = p(x, t), \ i = i(x, t), \ A(x) = \alpha(x) + d(x) + r(x)u(x), \) and \( 0 < u(x) < 1. \) We assume no-flux boundary conditions,

\[
\frac{\partial p}{\partial \nu} = \frac{\partial i}{\partial \nu} = 0 \text{ on } \partial G, \ t > 0,
\]

where \( \nu \) is the outward normal, and prescribe the initial conditions

\[
p(x, 0) = p_0(x), \ i(x, 0) = i_0(x) \text{ for } x \in \overline{G}.
\]

We assume that

\[
0 \leq i_0(x) \leq p_0(x) \leq 1 \text{ for } x \in \overline{G},
\]

that the functions

\( r, \ u, \ \alpha, \ d, \ \sigma, \ i_0, \) and \( p_0 \)

are in a H"older class \( C^\alpha(G), \) that \( \partial G \) is in \( C^{2+\alpha} \), and that

\[
\frac{\partial p_0}{\partial \nu} = \frac{\partial i_0}{\partial \nu} = 0 \text{ on } \partial G.
\]

Then by standard theory of parabolic PDEs, there exists a unique solution of (6.1)–(6.3) for all \( t > 0 \) with

\[
D_t p, \ D_t^2 p, \ D_t i, \ \text{and } D_t^2 i
\]

\( \alpha \)-H"older continuous in \( x \) and \( (\alpha/2) \)-H"older continuous in \( t \), uniformly in \( G \times [t_0, T] \) for any \( 0 < t_0 < T < \infty, \) and \( p, i \) are continuous for \( x \in \overline{G} \) and \( t \geq 0. \)

In the following result, we indicate the well-posedness of the PDE model with continuous dispersion.

**Theorem 6.1.** If

\[
0 < i_0(x) < p_0(x) < 1 \text{ for } x \in \overline{G},
\]

then the solution \((p(x, t), i(x, t))\) of (6.1) satisfies the inequalities

\[
0 < i(x, t) < p(x, t) < 1 \text{ for } x \in \overline{G}, \ t > 0.
\]

*Proof.* The assertion \( i(x, t) > 0 \) follows from the (strong) maximum principle, where we use one of the no-flux boundary condition for \( i. \) As long as \( p(x, t) < 1, \) we have

\[
\frac{\partial p}{\partial t} - \Delta p < -\alpha(x)i(x, t) < 0
\]

if \( p(x, t) > u(x). \) Hence, again by the (strong) maximum principle or by the comparison principle, \( p(x, t) < 1 \) for all \( x \in \overline{G}, \ t > 0. \)

We finally prove that \( p - i > 0. \) Proceeding by contradiction, suppose this inequality holds for \( x \in \overline{G}, \ 0 \leq t < \bar{t}, \) and \( (p - i)(x_0, \bar{t}) = 0 \) for some \( x_0 \in \overline{G}. \) As in the proof of Theorem 3.1, by comparing the right-hand sides of model (6.1) we have

\[
\frac{\partial (p - i)}{\partial t} - \Delta (p - i) > 0
\]
near the point \((x_0, 0)\). Hence, by the maximum principle and the no-flux conditions (6.2), \((p - i)\) cannot take minimum 0 at \((x_0, 0)\), which is a contradiction. 

We introduce positive constants \(\underline{u}, \overline{u}\) such that

\[
0 < \underline{u} \leq u(x) < \overline{u} < 1 \quad \text{for all } x \in \overline{O}.
\]

As in the model with discrete dispersion, in the continuous diffusion-linked model, we obtain that the total population goes extinct whenever each initial local patch population density is below the smallest Allee threshold.

**Theorem 6.2** (host population extinction). In model (6.1), assume that

\[
0 < p_0(x) < \underline{u} \quad \text{for all } x \in \overline{G}.
\]

Then the solution

\[
(p(x, t), i(x, t))
\]

satisfies

\[
p(x, t) < \underline{u} - \varepsilon \quad \text{for all } t > 0 \text{ and some } \varepsilon > 0,
\]

and

\[
p(x, t) \leq Ce^{-\gamma t} \quad \text{for all } x \in \overline{G} \text{ and } t > 0,
\]

where \(C\) and \(\gamma\) are positive constants. Hence,

\[
(p(x, t), i(x, t)) \to \vec{0} \quad \text{as } t \to \infty.
\]

**Proof.** As long as \(p < \underline{u} - \varepsilon\), there holds

\[
\frac{\partial p}{\partial t} - \Delta p < 0.
\]

Hence, by the maximum principle, \(p < \underline{u} - \varepsilon\) for all \(x \in \overline{G}\) and \(t > 0\). But then, by (6.1),

\[
\frac{\partial p}{\partial t} - \Delta p \leq -\gamma \varepsilon
\]

for some positive constant \(\gamma\), and (6.6) then follows by comparison of \(p\) with \(Ce^{-\gamma t}\). 

We denote the DFE of (6.1), the solution of

\[
p(x, t) \equiv 1 \quad \text{and} \quad i(x, t) \equiv 0,
\]

by \((1, 0)\). We consider the case when \((1, 0)\) is not stable in the following sense. There exists a neighborhood \(V_\varepsilon\) of \((1, 0)\) defined by

\[
(6.7) \quad V_\varepsilon \equiv \{1 - \varepsilon_0 < p(x) < 1, \ 0 < i(x) < \varepsilon_0 \text{ for all } x \in \overline{G}\}
\]

for some small \(\varepsilon_0 > 0\) and initial condition \((p_0(x), i_0(x)) \notin V_\varepsilon\) such that

\[
(6.8) \quad (p_0, i_0) \in V_\delta \text{ for some small } \delta > \varepsilon_0 \quad \text{and} \quad (p(x, t), i(x, t)) \notin V_\varepsilon \text{ for all } t > 0.
\]
The next theorem is similar to Theorem 4.3. As in the remark following the statement of Theorem 4.3, we shall assume that

\[(6.9) \quad \frac{\sigma(x)}{A(x)} \quad \text{and} \quad \frac{\alpha(x)}{r(x)}\]

are sufficiently large and, in particular,

\[(6.10) \quad P_T(x) \equiv \frac{A(x)}{\sigma(x) - 1} < u\]

uniformly for \(x \in \Omega\). Independent of initial population sizes, we obtain that the species goes extinct “globally” in the continuous dispersal-link model whenever at each location the disease transmission rate is high and the Allee threshold is far from the disease threshold at that location.

**Theorem 6.3 (host population extinction).** Under conditions (6.9) and (6.10), if

\[(p_0, i_0) \quad \text{is as in (6.8)},\]

then the solution

\[(p(x, t), i(x, t))\]

of (6.1), satisfies

\[\max_{x \in \Omega} p(x, t) \to 0 \quad \text{as} \quad t \to \infty.\]

**Proof.** Let

\[\tilde{U}_\varepsilon \equiv \{(p, i) : u - \varepsilon < p(x) < 1, 0 < i(x) < p(x) \quad \text{for at least some point} \quad x \in \Omega\}\]

and set \(U_\varepsilon = \tilde{U}_\varepsilon \setminus V_{i_0}\). Consider the function

\[Q(t) = \int_\Omega [p(x, t) - \ln(i(x, t))] \, dx.\]

By model (6.1),

\[\frac{dQ}{dt} = K(t) + \int_\Omega (\Delta p) \, dx - \int_\Omega \frac{\Delta i}{i} \, dx,\]

where

\[(6.11) \quad K(t) = \int_\Omega \left\{r(x)(1 - p)(p - u(x))p - \alpha(x)i\right\} + [A(x) - (\sigma(x) - 1)p + \sigma(x)i] dx.\]

Clearly,

\[\int_\Omega \Delta p = \int_{\partial \Omega} \frac{\partial p}{\partial \nu} = 0,\]

\[\int_\Omega \frac{\Delta i}{i} = \int_{\partial \Omega} \frac{\partial i}{\partial \nu} \cdot 1 - \int \nabla i \cdot \nabla \frac{1}{i} = \int \frac{\nabla i \cdot \nabla \frac{1}{i}}{i^2} \geq 0.\]
Hence,

\[ \frac{dQ}{dt} \leq K(t). \]  

By the choice of \((p_0, i_0)\) we see that if \((p(x, t), i(x, t))\) belongs to \(U_\varepsilon\) for all \(t > 0\), then it also belongs to \(\tilde{U}_\varepsilon\) for all \(t > 0\). But then, for any \(t > 0\), there exists a point \(x_t\) such that \(p(x_t, t) > u - \varepsilon\) and by Hölder continuity, 

\[ p(x, t) > u - \varepsilon \quad \text{if} \quad x \in S_t, \]

where \(S_t\) is a neighborhood of \(x_t\) with volume \(\geq c > 0\), \(c\) depending only on \(\varepsilon\). Using assumptions (6.9) and (6.10), we find that the second [...] in (6.11) is negative and it controls the first [...] , so that, by (6.12),

\[ \frac{dQ}{dt} \leq -\gamma \quad \text{for some positive constant } \gamma. \]

Noting, however, that \(Q\) is bounded from below, the last inequality cannot hold for all \(t > 0\). Hence, \((p(\cdot, t), i(\cdot, t))\) must exit \(\tilde{U}_\varepsilon\) at some time \(t = \tau\), and then the assertion of Theorem 6.3 follows from Theorem 6.2. 

We next consider extensions of Theorems 5.1–5.3. First, as in Theorem 5.1 we prove that, in the absence of the disease, the continuous diffusion-linked population persists at each location, whenever the initial population is far from the largest Allee threshold.

**Theorem 6.4** (population persistence). In model (6.1), if
\[ i_0(x) \equiv 0, \quad p_0(x) > \overline{u} + \varepsilon \quad \text{for all } x \in \overline{G} \quad \text{and} \quad \varepsilon > 0, \]

then the model solution
\[ (p(x, t), i(x, t)) \]

satisfies
\[ \overline{u} + \varepsilon < p(x, t) \quad \text{for all } x \in \overline{G}, \quad t > 0. \]

The proof is similar to the proof of Theorem 6.2, using the maximum principle. As in Theorem 5.2, next we prove that if the initial population density at each location is far from the biggest Allee threshold, then the population persists (with or without infected individuals) in the continuous dispersal-linked model whenever the disease transmission rate at each location, \(\alpha(x)\), and the Allee threshold, \(u(x)\), are small while the intrinsic per-capita growth rate at that location, \(r(x)\), is large.

**Theorem 6.5** (host population persistence). In model (6.1), if
\[ i_0(x) \geq 0, \quad p_0(x) > \overline{u} + \varepsilon \quad \text{for all } x \in \overline{G}, \quad \varepsilon > 0, \]

and
\[ \min_{x \in \overline{G}} \left[ r(x)(1 - y)(y - u(x))y - \alpha(x)y \right]_{y=\overline{u}+\varepsilon} > 0, \]

then the model solution
\[ (p(x, t), i(x, t)) \]
satisfies
\[ \pi + \varepsilon < p(x, t) \text{ for all } x \in \overline{G}, \ t > 0. \]

The proof follows by the maximum principle, as in the previous two theorems.

In order to establish persistence of the infected population, we introduce the function
\[ I(t) = \int_G i(x, t)dx \]
and the condition
\[ (\sigma(x) - 1)\pi > A(x) \text{ for all } x \in \overline{G}. \]  

As in Theorem 5.3, we prove in Theorem 6.6 that if the maximum Allee threshold is sufficiently large, then it is possible to have the persistence of the infected individuals in the continuous dispersal-linked model.

**Theorem 6.6 (disease persistence).** In model (6.1), under conditions (6.13)–(6.15), if
\[ I(0) > 0, \]
then
\[ I(t) \geq \varepsilon_0 \text{ for all } t \text{ sufficiently large and some positive constant } \varepsilon_0. \]

**Proof.** If \( I(t) < \varepsilon_0 \) for some point \( t = \tilde{t} \), then
\[ \frac{dI(\tilde{t})}{dt} = \int_G \left[-A(x) + (\sigma(x) - 1)p(x, \tilde{t}) - \sigma(x)i(x, \tilde{t})\right]i(x, \tilde{t})dx. \]

Since \( \pi + \varepsilon < p(x, t) \) (by Theorem 6.5), using (6.15) we get
\[ \frac{dI(\tilde{t})}{dt} > \gamma \int_G i(x, \tilde{t})dx = \gamma I(\tilde{t}) \]
for some positive constant \( \gamma \), provided that \( \varepsilon_0 \) is sufficiently small, depending on \( \varepsilon \). It follows that \( I(t) > \varepsilon_0 \) for some \( t = t_0 \), which depends on \( I(0) \), and then \( I(t) > \varepsilon_0 \) for all \( t > t_0 \). \( \square \)

**7. Illustrative examples.** In this section, we use two specific examples to illustrate the fact that if one isolated patch is low-risk and the others are high-risk, then migration can drive the total population to the brink of extinction, or it can lead to persistence of the population in both patches. In both examples, given below, we consider model (2.1) with \( n = 2 \) and parameters
\[ r_1 = \alpha_1 = 0.016667, \ d_1 = 0.00482, \ \sigma_1 = 1.23, \ u_1 = 0.2, \]
\[ r_2 = \alpha_2 = 0.016667, \ d_2 = 0.00482, \ \sigma_2 = 1.241, \text{ and } u_2 = 0.2. \]

When there is no migration, then the population persists locally in patch 1 while it goes to extinction in patch 2 \[22\], as shown in Figure 1. That is, without migration patch 1 is low-risk while patch 2 is high-risk.
Fig. 1. The initial condition \((p_1(0), i_1(0), p_2(0), i_2(0)) = (1, 0.0001, 0.0101, 0.01)\) leads to population persistence in patch 1 and population extinction in patch 2 when there are no migrations in Example 1.

Fig. 2. The initial condition \((p_1(0), i_1(0), p_2(0), i_2(0)) = (1, 0.0001, 0.0101, 0.01)\) leads to population collapse in both patches when there is migration in Example 1 and \(\delta = 0.001\), where \(L_{12} = L_{21} = 1\).
**Example 1.** We take initial value

\[(p_1(0), i_1(0), p_2(0), i_2(0)) = (1, 0.0001, 0.0101, 0.01).\]

Migration with \(\delta = 0.001\) and \(L_{12} = L_{21} = 1\) leads to the total collapse of the migration-linked total population, as shown in Figure 2.

In Example 1, species isolation that promotes local species persistence can be used as a conservation strategy for maintaining biodiversity.

**Example 2.** To study the role of initial conditions on Figures 1–2, we now consider Example 1 with the initial condition

\[(p_1(0), i_1(0), p_2(0), i_2(0)) = (1, 0.0001, 1, 0.0001).\]

As in Figure 1, Figure 3 shows that when there is no migration, the population persists locally in patch 1 while it goes extinct locally in patch 2 [22]. That is, without migration patch 1 is low-risk while patch 2 is high-risk.

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**Fig. 3.** The initial condition \((p_1(0), i_1(0), p_2(0), i_2(0)) = (1, 0.0001, 1, 0.0001)\) leads to population persistence in patch 1 and population extinction in patch 2 when there are no migrations in Example 2.

When there is migration with \(\delta = 0.001\) and \(L_{12} = L_{21} = 1\), unlike Figure 2, Figure 4 shows that the population persists in both patches of the migration-linked model.

Unlike in Example 1, in Example 2, linkages of species habitats that offset local extinctions can be used as a conservation strategy for maintaining biodiversity.

**8. Conclusion.** We have extended the single patch epidemic model of Hilker et al. [24], [25], [26], from an SI epidemic model with the strong Allee effect in the host demographics and no movement, to a multipatch model with dispersal between the patches and a reaction-diffusion model on a continuous spatial domain. The presence of the strong Allee effect adds to these models the possibility of population extinction.
as the disease disappears, a concern for species conservation efforts in maintaining biodiversity. Our extended models are based on fairly simple biologically relevant assumptions and could be applied to host populations with fatal infectious diseases that have similar demographic Allee effect and epidemiological structures.

Using the discrete and continuous diffusion-linked models, we obtain verifiable conditions that lead to the host population extinction for most initial population densities and conditions that guarantee host/disease persistence for some initial population densities. In particular, we proved that the host population goes extinct in the migration-linked models whenever the initial host population density on each patch \( j \) is lower than the smallest Allee threshold. We also proved that, when the initial host population size on each patch \( j \) is higher than the largest Allee threshold, then the host population goes extinct in the migration-linked models whenever each infected population nullcline, \( i_{j \text{-nullcline}} \), lies “well” above each host population nullcline, \( p_{j \text{-nullcline}} \). However, when the \( p_{j \text{-nullcline}} \) on each patch \( j \) is “sufficiently” elevated in the sense of (5.3), then high host population densities can lead to host population persistence in the migration-linked model whenever the largest eigenvalue of the Jacobian matrix \( J_{(\vec{\Gamma}, \vec{b})} \) is positive. These results on how species persistence or extinction relates to (habitat dependent) Allee thresholds, migrations, and fatal disease dynamics may be useful in conservation biology. For example, the endangered African wild dog \( Lycaon pictus \) exhibits the Allee effect and is vulnerable to fatal diseases like rabies, distemper, and anthrax. African wild dogs are nomadic throughout most of the year and are known to wander in ranges that may cover approximately 580 square miles (1,500 square km) [22], [25]. Our extended models can be used to investigate how the Allee threshold of one subpopulation of the African wild dog in a natal pack at a geographical location is influenced by the collective migrations of several African wild dog populations from different natal packs with different Allee thresholds.

Fig. 4. The initial condition \((p_1(0), i_1(0), p_2(0), i_2(0)) = (1, 0.0001, 1, 0.0001)\) leads to population persistence on all patches when there is migration in Example 1 and \( \delta = 0.001 \), where \( L_{12} = L_{21} = 1 \).
Using a two-patch model, we show, by simulations, that migration between low- and high-risk patches can endanger the low-risk population whenever the high-risk population density is below its Allee threshold. However, migration can save the high-risk population from extinction whenever the initial host population densities on both patches are sufficiently high. These results show that, depending on the size of an endangered population, isolation and habitat connectivity can be effective strategies for maintaining biodiversity.

REFERENCES


