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Transmission dynamics for vector-borne diseases in a patchy environment

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Abstract In this paper, a mathematical model is derived to describe the transmission and spread of vector-borne diseases over a patchy environment. The model incorporates into the classic Ross–MacDonald model two factors: disease latencies in both hosts and vectors, and dispersal of hosts between patches. The basic reproduction number $\mathcal{R}_0$ is identified by the theory of the next generation operator for structured disease models. The dynamics of the model is investigated in terms of $\mathcal{R}_0$. It is shown that the disease free equilibrium is asymptotically stable if $\mathcal{R}_0 < 1$, and it is unstable if $\mathcal{R}_0 > 1$; in the latter case, the disease is endemic in the sense that the variables for the infected compartments are uniformly persistent. For the case of two patches, more explicit formulas for $\mathcal{R}_0$ are derived by which, impacts of the dispersal rates on disease dynamics are also explored. Some numerical computations for $\mathcal{R}_0$ in terms of dispersal rates are performed which show visually that the impacts could be very complicated: in certain range of the parameters, $\mathcal{R}_0$ is increasing with respect to a dispersal rate while in some other range, it can be decreasing with respect to the same dispersal rate. The results can be useful to health organizations at various levels for setting guidelines or making policies for travels, as far as malaria epidemics is concerned.

Keywords Vector-borne diseases · Patch · Dispersal · Latency · Basic reproduction number · Non-local infection · Persistence

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1 Introduction

Vector-borne diseases are transmitted through cross infections between a vector species and a host species. Vector can be mosquitoes, ticks, triatomines bugs, sandflies, and blackflies (Confalonieri et al. 2007); while a host is typically human but can also be other animals. A typical example of such diseases is malaria, which is a mosquito-borne disease that has spread to more than one hundred countries, mostly in tropical and sub-tropical regions. Each year, 300–500 million infection cases are reported, among which around a million cases result in deaths. Thus, malaria still remains a threat to human beings in many places in the world. Other vector-borne diseases include, but are limited to, dengue and Japanese encephalitis (mosquito-borne) and Lyme disease (tick-borne).

Mathematical models can help understand the dynamics of transmission and spread of an infectious disease and thereby, provide guides and suggestions for the control of the disease. In the context of malaria, the earliest model is the Ross–MacDonald model (see, e.g., Ross 1910; Macdonald 1957 or Aron and May 1982), which is given by the following system of ordinary differential equations:

$$\begin{cases} \dfrac{\mathrm{d}I_h}{\mathrm{d}t} = ac_1 I_m \dfrac{N - I_h}{N} - d_1 I_h, \\ \dfrac{\mathrm{d}I_m}{\mathrm{d}t} = ac_2 (M - I_m) \dfrac{I_h}{N} - d_2 I_m. \end{cases} \tag{1.1}$$

Here, I_h and I_m represent the populations of the infectious classes of human beings and female mosquitoes, respectively. N and M are the total populations of human beings and female mosquitoes, which were assumed to be constants. The model is a result of ignoring the latency within both hosts and mosquitoes and assuming no immunity of the recovered individuals (thus, the terms $N - I_h$ and $M - I_m$ present the populations of the susceptible humans and mosquitoes). The constant a is the mosquito biting rate; c_1 is the probability that a bite by an infective mosquito to a susceptible human will cause infection of the human; and c_2 is the probability that a bite by a susceptible mosquito of an infective human individual will cause infection of the mosquito. It is assumed that the average durations of infection for human and mosquitoes are $1/d_1$ and $1/d_2$ individually. By analyzing this mathematical model, both Ross and Macdonald found that it would be possible to eradicate the disease without killing all vector mosquitoes. This was in contrast to the traditional belief that malaria could be wiped out only by eradicating all vector mosquitoes, which turned out to be impossible in practice. Indeed, by looking at the basic reproduction number for this model given by

$$\mathcal{R}_0 = \frac{ac_1}{d_1} \frac{M}{N} \frac{ac_2}{d_2}, \tag{1.2}$$

one knows that any measure(s) that can bring $\mathcal{R}_0$ to a value less than 1 would eventually drive the disease to extinction. Obviously, among the possible measures are, for example, controlling the mosquito population M (e.g., by spraying mosquito

pesticides) to a sufficiently lower level and controlling the biting rate a (e.g., by using mosquito nets).

The Ross–MacDonald model is a simple example showing how mathematical modeling can provide insights into the mechanism of malaria transmission and spread, by which, some effective measures can be suggested to control the disease. It can also be applied to other vector-borne disease. This simple model is mathematically tractable in the sense that long term solution behavior of model (1.1) can be fully determined by the quantity $\mathcal{R}_0$ which is explicitly calculated by the model parameters. However, it is highly simplified and biologically inaccurate in the sense that many biological factors are ignored. Among such factors are latencies of the developments of parasites/virus within hosts (such as humans) and vectors (such as mosquitoes), and the spatial heterogeneity of the habitats of hosts and vectors. In recent years, researchers have started incorporating these missing factors into the model, resulting in various modifications of (1.1).

Along the line of latency, in Aron and May (1982), Chamchod and Britton (2011), and Lou and Zhao (2011), a discrete delay is introduced into their models to account for the latency within vectors (mosquitoes); in Ruan and Xiao (2008), two discrete delays are added into model (1.1), one accounting for the latency in humans and the other for that in vectors (mosquitoes); and in Xiao and Zou (2013), two probability distributions are used to account for the variation of latencies in host (human) and vector (mosquito) populations.

As far as spatial heterogeneity is concerned, there are two types: (A) continuous spatial heterogeneity and (B) discrete spatial heterogeneity. With respect to (A), proposed and studied in Lou and Zhao (2010) is a model with spatial diffusion and advection of vectors (mosquitoes), as well as the seasonality of the parameters; and in Lou and Zhao (2011) a spatially non-local model with latency in vectors (mosquitoes) is discussed. In the context of (B), there are also works (Arino et al. 2011; Auger et al. 2008; Cosner et al. 2009; Gao and Ruan 2012), extending the model (1.1) into patch models. However, the patch models in Arino et al. (2011), Auger et al. (2008), and Cosner et al. (2009); Gao and Ruan (2012) have all ignored the latencies which have been shown to have a significant impact on the disease dynamics. This motivates us to derive a more realistic patch model that not only contains dispersal of humans but also incorporates the latencies in both humans and mosquitoes.

In this paper, we will follow the approach in Li and Zou (2010) and Li and Zou (2009) where patch models with non-local infections are derived and analyzed. Making use of the infection age as well as the typical method of characteristics for structured populations, we derive a model involving a patchy environment that has two discrete delays, accounting for the latencies in hosts and vectors, respectively. The model also contains spatially non-local terms accounting for non-local infections resulting from the dispersal of hosts during the latent period. We point out that, as in Arino et al. (2011) and Auger et al. (2008), we assume vectors cannot fly/jump/run the distances between the patches, and hence, only hosts can disperse between patches in our model. This is in contrast to the model in Cosner et al. (2009) and Gao and Ruan (2012) where both hosts (humans) and vectors (mosquitoes) can disperse between patches. In this context, the models in Cosner et al. (2009) and Gao and Ruan (2012) are suitable when the patches are ponds or other small aquatic environments within a city/region so that mosquitoes

can fly between these patches; while a scenario for the models in Arino et al. (2011) and Auger et al. (2008) and in this paper is that the patches are cities or even countries so that the vectors (mosquitoes) can not fly the distances between these patches.

The rest of the paper is organized as below. In Sect. 2, we derive (not propose) the model rigorously, starting from an age structured system of first order partial differential equations and using the method of characteristics. In Sect. 3, we address the well-posedness by proving the non-negativeness and boundedness of solutions. In Sect. 4, we identify the basic reproduction number $\mathcal{R}_0$ of the model by using the abstract theory for structured disease models developed by Thieme (2009). As expected, $\mathcal{R}_0$ plays a threshold role in the sense that when $\mathcal{R}_0 < 1$, the disease free equilibrium (DFE) is asymptotically stable (Sect. 4); if $\mathcal{R}_0 > 1$ the DFE is unstable and the disease is endemic in the sense that the infected components of the model are uniformly persistent (Sect. 5). In Sect. 6, we focus on the two-patch case where more explicit conditions are obtained, and impacts of dispersal rates in all different compartments on $\mathcal{R}_0$ are also explored. In the last section, Sect. 7, we summarize our main results and discuss the biological implications of our results.

2 Model formulation for general patch model with fixed latency

Assume that the host and vector (such as human and *Anopheles* mosquito) populations are distributed over n patches. Here, depending on the situation, patches could be towns, cities or countries etc. Use $N_i(t)$ and $M_i(t)$ to denote the total population of hosts and vectors in patch i, respectively. In the presence of a vector-borne disease (e.g., malaria), the total populations are divided into compartments of susceptible and infected classes. Assume that there is a fixed infection latent period of length τ_1 within the host and another fixed latent period τ_2 within the vector. Although latencies differ from individual to individual in general, for simplicity, here we assume fixed latencies which can be considered as an approximation of the mean latencies within hosts and vectors. We can also assume that $\tau_2 \leq \tau_1$ (for malaria, it is known that the latency in humans ranges from 7 to 30 days and the latency in mosquitoes is approximately 10 days). Due to latencies, the infected classes are further divided into latent and infectious classes for both hosts and vectors. Let S_{ij}, L_{ij} and I_{ij} be the sub-populations of the susceptible, latent and infectious classes respectively, with the first sub-index i specifying the i-th patch ($i = 1, 2, ..., n$), and the second sub-index j representing host for $j = 1$ and vector for $j = 2$.

As we pointed out at the end of the introduction, we assume that the distances between two patches are sufficiently large such that the vector is unable to disperse between the patches. Then, the sub-populations of the vectors can be described by the following differential equations:

$$\begin{cases} \dfrac{\mathrm{d}S_{i2}(t)}{\mathrm{d}t} = \beta_{i2}M_i(t) - d_{i2}S_{i2}(t) - a_i c_{i2} S_{i2}(t)\dfrac{I_{i1}(t)}{N_i(t)}, \\ \dfrac{\mathrm{d}L_{i2}(t)}{\mathrm{d}t} = -d_{i2}L_{i2}(t) + a_i c_{i2} S_{i2}(t)\dfrac{I_{i1}(t)}{N_i(t)} - a_i c_{i2} S_{i2}(t-\tau_2)\dfrac{I_{i1}(t-\tau_2)}{N_i(t-\tau_2)}e^{-d_{i2}\tau_2}, \\ \dfrac{\mathrm{d}I_{i2}(t)}{\mathrm{d}t} = -d_{i2}I_{i2}(t) + a_i c_{i2} S_{i2}(t-\tau_2)\dfrac{I_{i1}(t-\tau_2)}{N_i(t-\tau_2)}e^{-d_{i2}\tau_2}, \end{cases} \tag{2.1}$$

where β_{i2}, d_{i2} and a_i are the birth, death and biting rates respectively in patch i, and c_{i2} is the probability that a contact between a susceptible vector with an infectious host (i.e. a bite by a susceptible mosquito of an infectious human) in patch i will cause infection. In this work, we further assume that $\beta_{i2} = d_{i2}$ for $i = 1, 2, \ldots, n$, so that the birth and death of the vectors are balanced in each patch, implying that the total vector population $M_i(t)$ in patch i remains a constant as $M_i'(t) = 0$ under the above assumption. Hence, we will simply write M_i to replace $M_i(t)$.

On the host side, dispersal between patches is common. This, together with the latency within hosts, will result in the so-called non-local infections, meaning that one host may get infected in one patch, but start to infect vectors in another patch. To model this phenomenon, we follow the ideas in Li and Zou (2010) and Li and Zou (2009) to make use of the infection age a. Let $l_i(t, a)$ be the density of the infected hosts in patch i (i=1,2,...,n) with the infection age a at time t. Similar to the equations incorporating the natural age structure in Metz and Diekmann (2000), the densities $l_i(t, a)$, $(i = 1, 2, ...n)$ are described by the following system of first-order partial differential equations

$$\frac{\partial}{\partial t} l_i(t,a) + \frac{\partial}{\partial a} l_i(t,a) = -(d_{i1} + \bar{d}_{i1}(a) + \gamma_i) l_i(t,a) + \sum_{j=1}^{n} D_{ij}(a) l_j(t,a) - \sum_{j=1}^{n} D_{ji}(a) l_i(t,a), \quad (2.2)$$

where $D_{ij}(a)$ is the dispersal rate from patch j to patch i of the infected hosts at the infection age a; d_{i1} is the natural death rate of hosts, $\bar{d}_{i1}(a)$ stands for disease induced mortality rate and γ_i is the recovery rate, all in patch i. Although $D_{ii}(a) = 0$, $i = 1, \ldots, n$, we keep $D_{ii}(a)$ $(i = 1, \ldots, n)$ in the equations for the convenience of notations. We ignore delays and loss for the movements of hosts between patches, otherwise things will become mathematically more intractable.

From the definition of $l_i(t, a)$, the host population in the latent and infectious class in patch i at time t can be expressed by

$$L_{i1}(t) = \int_0^{\tau_1} l_i(t,a)\,\mathrm{d}a \quad \text{and} \quad I_{i1}(t) = \int_{\tau_1}^{\infty} l_i(t,a)\,\mathrm{d}a. \quad (2.3)$$

The fact that d_{i1} and γ_i are bounded and the assumption of $\bar{d}_{i1}(a)$ below imply that

$$l_i(t, \infty) = 0. \quad (2.4)$$

Noting that the population with zero infection age is nothing but the population of new infected, we have

$$l_i(t, 0) = a_i c_{i1} \frac{I_{i2}(t) S_{i1}(t)}{N_i(t)}, \quad (2.5)$$

where c_{i1} is the probability that a contact between an infectious host and a susceptible vector (i.e. a bite by an infectious mosquito to a susceptible human being) will result in a successful new infection of a susceptible host in patch i .

For convenience of showing the main idea to build the patch model, we further assume that the disease induced mortality rates and the dispersal rates in system (2.2) are piecewise constants:

$$\bar{d}_{i1}(a)=\begin{cases}0, & 0\le a\le\tau_1,\\ \mu_i, & a>\tau_1,\end{cases}\qquad D_{ij}(a)=\begin{cases}D^L_{ij}, & 0\le a\le\tau_1,\\ D^I_{ij}, & a>\tau_1,\end{cases}\qquad i,j=1,2,...n. \tag{2.6}$$

It follows from (2.2)–(2.4) and (2.6) that

$$\begin{aligned}\frac{\mathrm{d}I_{i1}(t)}{\mathrm{d}t}=&-\int_{\tau_1}^{\infty}\frac{\partial l_i(t,a)}{\partial a}\,\mathrm{d}a-\int_{\tau_1}^{\infty}\left(d_{i1}+\bar{d}_{i1}(a)\right)l_i(t,a)\,\mathrm{d}a\\&+\int_{\tau_1}^{\infty}\sum_{j=1}^{n}D_{ij}(a)l_j(t,a)\,\mathrm{d}a-\int_{\tau_1}^{\infty}\sum_{j=1}^{n}D_{ji}(a)l_i(t,a)\,\mathrm{d}a\\=&\,l_i(t,\tau_1)-(d_{i1}+\mu_i+\gamma_i)I_{i1}(t)+\sum_{j=1}^{n}D^I_{ij}I_{j1}(t)-\sum_{j=1}^{n}D^I_{ji}I_{i1}(t).\end{aligned} \tag{2.7}$$

Similarly, from (2.2)–(2.3) and (2.5)–(2.6), we obtain

$$\frac{\mathrm{d}L_{i1}(t)}{\mathrm{d}t}=a_ic_{i1}I_{i2}(t)\frac{S_{i1}(t)}{N_i(t)}-d_{i1}L_{i1}(t)-l_i(t,\tau_1)+\sum_{j=1}^{\infty}D^L_{ij}c_{j1}(t)-\sum_{j=1}^{\infty}D^L_{ji}c_{i1}(t). \tag{2.8}$$

The term $l_i(t,\tau_1)$ in (2.7) and (2.8) can be determined by applying the method of characteristics to (2.2) in the same way as in Li and Zou (2010) and Li and Zou (2009), and we give the details below.

For fixed $\xi>0$, let

$$U_i^{\xi}(t)=l_i(t,t-\xi),\qquad \text{for } \xi\le t\le\xi+\tau_1,\ \ i=1,2,...,n.$$

Then,

$$\begin{aligned}\frac{\mathrm{d}}{\mathrm{d}t}U_i^{\xi}(t)=&\ \frac{\partial}{\partial t}l_i(t,a)|_{a=t-\xi}+\frac{\partial}{\partial a}l_i(t,a)|_{a=t-\xi}\\=&-(d_{i1}+\bar{d}_{i1}(t-\xi))l_i(t,t-\xi)+\sum_{j=1}^{n}D_{ij}(t-\xi)l_j(t,t-\xi)\\&-\sum_{j=1}^{n}D_{ji}(t-\xi)l_i(t,t-\xi)\end{aligned} \tag{2.9}$$

$$= -d_{i1} l_i(t, t-\xi) + \sum_{j=1}^{n} D_{ij}^L l_j(t, t-\xi) - \sum_{j=1}^{n} D_{ji}^L l_i(t, t-\xi)$$
$$= -d_{i1} U_i^\xi(t) + \sum_{j=1}^{n} D_{ij}^L U_i^\xi(t) - \sum_{j=1}^{n} D_{ji}^L U_i^\xi(t).$$

Using vector notation $U^\xi(t) = \left(U_1^\xi(t), U_2^\xi(t), ..., U_n^\xi(t)\right)^T$ where T represents the transpose of a vector, (2.9) is rewritten as

$$\frac{\mathrm{d}}{\mathrm{d}t} U^\xi(t) = B U^\xi(t), \tag{2.10}$$

where the coefficient matrix B is given by

$$B = \begin{bmatrix} -d_{11} - \sum_{j=1}^{n} D_{j1}^L & D_{12}^L & \cdots & D_{1n}^L \\ D_{21}^L & -d_{21} - \sum_{j=1}^{n} D_{j2}^L & \cdots & D_{2n}^L \\ \vdots & \vdots & \ddots & \vdots \\ D_{n1}^L & D_{n2}^L & \cdots & -d_{n1} - \sum_{j=1}^{n} D_{jn}^L \end{bmatrix}.$$

Integrating system (2.10) for $t \in [\xi, \xi + \tau_1]$ yields

$$U^\xi(t) = e^{B(t-\xi)} \left(U_1^\xi(\xi), U_2^\xi(\xi), \ldots, U_n^\xi(\xi)\right)^T, \quad \xi \le t \le \xi + \tau_1. \tag{2.11}$$

From the definition of $U_i^\xi(t)$ and equalities (2.5), it follows that

$$\begin{aligned} U^\xi(t) &= e^{B(t-\xi)} \left(l_1(\xi, 0), l_2(\xi, 0), \ldots, l_n(\xi, 0)\right)^T \\ &= e^{B(t-\xi)} \left(a_1 c_{11} I_{12}(\xi) \frac{S_{11}(\xi)}{N_1(\xi)}, \cdots, a_n c_{n1} I_{n2}(\xi) \frac{S_{n1}(\xi)}{N_n(\xi)}, \right)^T, \quad \xi \le t \le \xi + \tau_1. \end{aligned} \tag{2.12}$$

For $t \ge \tau_1$, let $l(t, \tau_1) = (l_1(t, \tau_1), l_2(t, \tau_1, \ldots, l_n(t, \tau_1)))^T$. Then

$$\begin{aligned} l(t, \tau_1) &= U^{t-\tau_1}(t) \\ &= e^{B\tau_1} \left(a_1 c_{11} I_{12}(t-\tau_1) \frac{S_{11}(t-\tau_1)}{N_1(t-\tau_1)}, \ldots, a_n c_{n1} I_{n2}(t-\tau_1) \frac{S_{n1}(t-\tau_1)}{N_n(t-\tau_1)}\right)^T. \end{aligned} \tag{2.13}$$

Denoting the matrix $e^{B\tau_1}$ by $P = [p_{ij}(\tau_1)]_{n \times n}$, (2.13) becomes

$$l_i(t, \tau_1) = \sum_{j=1}^{n} p_{ij}(\tau_1) a_j c_{j1} I_{j2}(t-\tau_1) \frac{S_{j1}(t-\tau_1)}{N_j(t-\tau_1)}. \tag{2.14}$$

Since B is essentially positive, $P(t) = [p_{ij}(t)]_{n\times n} = e^{Bt}$ is a positive matrix for all $t > 0$ (see, e.g., Smith 1995).

Since our focus is not on the demography of the host, we will adopt the simplest demographic equation $N_i'(t) = K_{i1} - d_{i1}N_i(t)$ for patch i ($i = 1, 2, \ldots, n$), which leads to the following equation for the susceptible host population in patch i:

$$\frac{\mathrm{d}S_{i1}(t)}{\mathrm{d}t} = K_{i1} - d_{i1}S_{i1}(t) + \gamma_i I_{i1}(t) - a_i c_{i1} I_{i2}(t)\frac{S_{i1}(t)}{N_i(t)} + \sum_{j=1}^{n} D_{ij}^S S_{j1}(t) - \sum_{j=1}^{n} D_{ji}^S S_{i1}(t). \tag{2.15}$$

Here, similarly as matrices D^L and D^I, we also keep $D_{ii}^S (= 0)$, $i = 1, \ldots, n$ in the equations. Substituting equalities (2.14) back into the L_{i1} and I_{i1} equations in systems (2.7) and (2.8), and pulling the resulting equations with (2.1) and (2.15) together, we know that the sub-populations in all compartments for $t \geq \tau_1$ are described by the following system of delay differential equations (DDEs):

$$\begin{cases}
\dfrac{\mathrm{d}S_{i1}(t)}{\mathrm{d}t} = K_{i1} - d_{i1}S_{i1}(t) + \gamma_i I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D_{ij}^S S_{j1}(t) - \sum_{j=1}^{n} D_{ji}^S S_{i1}(t) \\
\qquad\qquad - a_i c_{i1} I_{i2}(t)\dfrac{S_{i1}(t)}{N_i(t)}, \\
\dfrac{\mathrm{d}L_{i1}(t)}{\mathrm{d}t} = a_i c_{i1} I_{i2}(t)\dfrac{S_{i1}(t)}{N_i(t)} - d_{i1}L_{i1}(t) + \displaystyle\sum_{j=1}^{n} D_{ij}^L L_{j1}(t) - \sum_{j=1}^{n} D_{ji}^L L_{i1}(t) \\
\qquad - \displaystyle\sum_{j=1}^{n} p_{ji}(\tau_1) a_i c_{i1} I_{i2}(t-\tau_1)\frac{S_{i1}(t-\tau_1)}{N_i(t-\tau_1)}, \\
\dfrac{\mathrm{d}I_{i1}(t)}{\mathrm{d}t} = -(d_{i1} + \gamma_i + \mu_i) I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D_{ij}^I I_{j1}(t) - \sum_{j=1}^{n} D_{ji}^I I_{i1}(t) \\
\qquad + \displaystyle\sum_{j=1}^{n} p_{ij}(\tau_1) a_j c_{j1} I_{j2}(t-\tau_1)\frac{S_{j1}(t-\tau_1)}{N_j(t-\tau_1)}, \\
\dfrac{\mathrm{d}S_{i2}(t)}{\mathrm{d}t} = d_{i2}M_i - d_{i2}S_{i2}(t) - a_i c_{i2} S_{i2}(t)\dfrac{I_{i1}(t)}{N_i(t)}, \\
\dfrac{\mathrm{d}L_{i2}(t)}{\mathrm{d}t} = -d_{i2}L_{i2}(t) + a_i c_{i2} S_{i2}(t)\dfrac{I_{i1}(t)}{N_i(t)} - a_i c_{i2} S_{i2}(t-\tau_2)\dfrac{I_{i1}(t-\tau_2)}{N_i(t-\tau_2)} e^{-d_{i2}\tau_2}, \\
\dfrac{\mathrm{d}I_{i2}(t)}{\mathrm{d}t} = -d_{i2}I_{i2}(t) + a_i c_{i2} S_{i2}(t-\tau_2)\dfrac{I_{i1}(t-\tau_2)}{N_i(t-\tau_2)} e^{-d_{i2}\tau_2},
\end{cases} \tag{2.16}$$

with $N_i(t) = S_{i1}(t) + L_{i1}(t) + I_{i1}(t)$ and $M_i = S_{i2}(t) + L_{i2}(t) + I_{i2}(t)$, for $i = 1, 2, \ldots, n$. In the equation of I_{i1}, we find that the recruitment consists of two

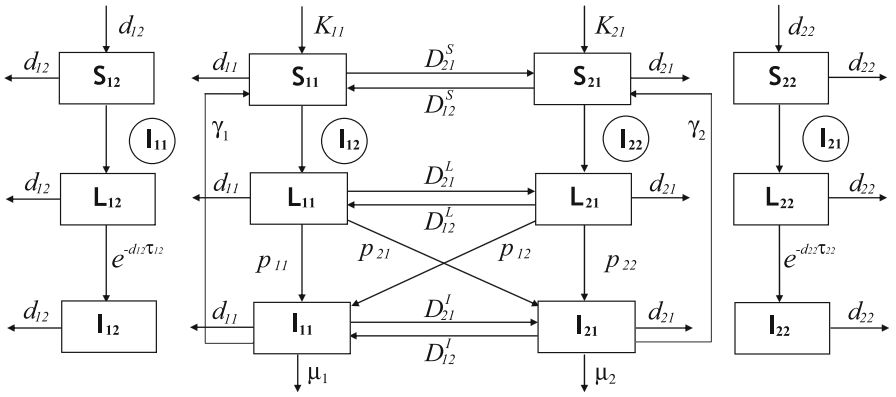

Fig. 1 Transmission diagram for $t \geq \tau_1$ for two-patch model.

parts: one directly results from travel of infectious individuals, while the other is a result of mobility of latent individuals with $p_{ij}(\tau_1)$ accounting for the probability that a host infected in patch j can survive the latent period $[0, \tau_1]$ and move to patch i at the end of the latent period. For $n = 2$, the transmission dynamics described by system (2.16) can be visualized by the transmission diagram in Fig. 1.

For $t \in [0, \tau_2)$, no new infected hosts and vectors will become infectious and hence, the disease dynamics is governed by the following system of ODEs:

$$
\begin{cases}
\dfrac{\mathrm{d}S_{i1}(t)}{\mathrm{d}t} = K_{i1} - d_{i1}S_{i1}(t) + \gamma_i I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D_{ij}^S S_{j1}(t) - \sum_{j=1}^{n} D_{ji}^S S_{i1}(t) \\
\qquad\qquad - a_i c_{i1} I_{i2}(t) \dfrac{S_{i1}(t)}{N_i(t)}, \\
\dfrac{\mathrm{d}L_{i1}(t)}{\mathrm{d}t} = a_i c_{i1} I_{i2}(t) \dfrac{S_{i1}(t)}{N_i(t)} - d_{i1}L_{i1}(t) + \displaystyle\sum_{j=1}^{n} D_{ij}^L L_{j1}(t) - \sum_{j=1}^{n} D_{ji}^L L_{i1}(t), \\
\dfrac{\mathrm{d}I_{i1}(t)}{\mathrm{d}t} = -(d_{i1} + \gamma_i + \mu_i) I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D_{ij}^I I_{j1}(t) - \sum_{j=1}^{n} D_{ji}^I I_{i1}(t), \\
\dfrac{\mathrm{d}S_{i2}(t)}{\mathrm{d}t} = d_{i2}M_i - d_{i2}S_{i2}(t) - a_i c_{i2} S_{i2}(t) \dfrac{I_{i1}(t)}{N_i(t)}, \\
\dfrac{\mathrm{d}L_{i2}(t)}{\mathrm{d}t} = -d_{i2}L_{i2}(t) + a_i c_{i2} S_{i2}(t) \dfrac{I_{i1}(t)}{N_i(t)}, \\
\dfrac{\mathrm{d}I_{i2}(t)}{\mathrm{d}t} = -d_{i2}I_{i2}(t),
\end{cases}
\tag{2.17}
$$

while for $t \in [\tau_2, \tau_1)$, the disease dynamics is given by another system of DDEs:

$$\begin{cases} \dfrac{\mathrm{d}S_{i1}(t)}{\mathrm{d}t} = K_{i1} - d_{i1}S_{i1}(t) + \gamma_i I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^S_{ij}S_{j1}(t) - \sum_{j=1}^{n} D^S_{ji}S_{i1}(t) \\ \qquad\qquad - a_i c_{i1} I_{i2}(t)\dfrac{S_{i1}(t)}{N_i(t)}, \\ \dfrac{\mathrm{d}L_{i1}(t)}{\mathrm{d}t} = a_i c_{i1} I_{i2}(t)\dfrac{S_{i1}(t)}{N_i(t)} - d_{i1}L_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^L_{ij}L_{j1}(t) - \sum_{j=1}^{n} D^L_{ji}L_{i1}(t), \\ \dfrac{\mathrm{d}I_{i1}(t)}{\mathrm{d}t} = -(d_{i1} + \gamma_i + \mu_i)I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^I_{ij}I_{j1}(t) - \sum_{j=1}^{n} D^I_{ji}I_{i1}(t), \\ \dfrac{\mathrm{d}S_{i2}(t)}{\mathrm{d}t} = d_{i2}M_i - d_{i2}S_{i2}(t) - a_i c_{i2} S_{i2}(t)\dfrac{I_{i1}(t)}{N_i(t)}, \\ \dfrac{\mathrm{d}L_{i2}(t)}{\mathrm{d}t} = -d_{i2}L_{i2}(t) + a_i c_{i2} S_{i2}(t)\dfrac{I_{i1}(t)}{N_i(t)} - a_i c_{i2} S_{i2}(t-\tau_2)\dfrac{I_{i1}(t-\tau_2)}{N_i(t-\tau_2)}e^{-d_{i2}\tau_2}, \\ \dfrac{\mathrm{d}I_{i2}(t)}{\mathrm{d}t} = -d_{i2}I_{i2}(t) + a_i c_{i2} S_{i2}(t-\tau_2)\dfrac{I_{i1}(t-\tau_2)}{N_i(t-\tau_2)}e^{-d_{i2}\tau_2}, \end{cases} \tag{2.18}$$

Note that in (2.16)–(2.18), the equations for $L_{i2}(t)$ are decoupled from the rest, and this fact will be used to simplify the analysis in Sect. 5. Also,it is obvious that the long term disease dynamics is represented by the system of DDEs (2.16) which will be, therefore, the main focus of our analysis in the subsequent sections.

It is natural to assume that the dispersal matrix $D^S = (D^S_{ij})$ is irreducible, otherwise the patchy environment can be further split into smaller irreducible environments isolated from each other. As the behavior of individuals in latent period generally remains the same as that of susceptible individuals, we assume that $D^L = (D^L_{ij})$ is also irreducible. But in this paper, it can be seen in the following sections that the irreducibility of $D^L = (D^L_{ij})$ is only necessary for addressing disease persistence in Sect. 5. The rest of the results, particularly those on computing the basic reproduction number, remain valid (see, Thieme 2009) without the assumption.

3 Well-posedness

Realistically, initial values for all variables in the model should be non-negative:

$$S_{ij}(0) \geq 0,\ I_{ij}(0) \geq 0,\ L_{i1}(0) \geq 0, \quad \text{for } i = 1, 2, ..., n; \quad j = 1, 2. \tag{3.1}$$

With such a set of initial values given, one can solve (2.17) to get a unique solution for $t \in [0, \tau_2]$ which can be easily shown to be non-negative in $[0, \tau_2]$. Using the values of this solution in the interval $[0, \tau_2]$, one can further solve the DDE system (2.18) to get a unique and non-negative solution defined for $t \in [\tau_2, \tau_1]$. The combination of these two solutions gives the initial functions for the DDE system (2.16) on $[0, \tau_1] = [0, \tau_2] \cup [\tau_2, \tau_1]$. This non-negative initial function, by the fundamental theory of

DDEs, will result in a unique solution of system (2.16) for $t \geq \tau_1$ which is also non-negative on the maximal interval of existence $[\tau_1, t_{max})$. Details of the theory validating the above argument can be found in, e.g., Hale and Verduyn Lunel (1993) and Smith (1995). Similar arguments, but for another non-vector-borne disease model with non-local infections on a patch environment, can be found in Li and Zou (2010).

Next, we show that the solutions of system (2.16) remain bounded. Firstly the boundedness of S_{i2}, L_{i2} and I_{i2} is obvious since $0 \leq S_{i2} \leq M_i, 0 \leq L_{i2} \leq M_i, 0 \leq I_{i2} \leq M_i$ and M_i is a constant. To prove the boundedness of S_{i1}, L_{i1} and I_{i1}, it suffices to show that $N_i(t)$ is bounded. Let $N(t)$ be the total population of hosts in the n patches,i.e.,

$$N(t) = \sum_{i=1}^{n} N_i = \sum_{i=1}^{n} S_{i1}(t) + \sum_{i=1}^{n} L_{i1}(t) + \sum_{i=1}^{n} I_{i1}(t).$$

Then,

$$\begin{aligned} \dot{N}(t) &= \sum_{i=1}^{n} \dot{S}_{i1}(t) + \sum_{i=1}^{n} \dot{L}_{i1}(t) + \sum_{i=1}^{n} \dot{I}_{i1}(t) \\ &= \sum_{i=1}^{n} K_{i1} - \sum_{i=1}^{n} d_{i1} N_i(t) - \sum_{i=1}^{n} \mu_i I_{i1}(t) \\ &\leq \sum_{i=1}^{n} K_{i1} - \sum_{i=1}^{n} \underline{d}_1 N_i(t) \\ &= \bar{K}_1 - \underline{d}_1 N(t), \end{aligned} \tag{3.2}$$

where $\underline{d}_1 = \min_{1 \leq i \leq n}(d_{i1})$ and $\bar{K}_1 = \sum_{j=1}^{n} K_{i1}$. By the comparison theorem, we conclude that $N(t)$ is bounded with $\limsup_{t \to \infty} N(t) \leq \bar{K}_1 / \underline{d}_1$. Consequently, $N_i(t)$, $i = 1, 2, \cdots, n$, are also bounded, and so are S_{i1}, L_{i1} and I_{i1} by the relations $0 \leq S_{i1} \leq N_i, 0 \leq L_{i1} \leq N_i, 0 \leq I_{i1} \leq N_i$.

The *a priori* boundedness of solutions to system (2.16) implies that all solutions with initial conditions satisfying initial condition (3.1) exist globally, that is, exist for all $t \in [\tau, \infty)$ (see Hale and Verduyn Lunel 1993).

Summarizing the above, we have established the following theorem.

Theorem 3.1 *For any given initial values satisfying initial condition* (3.1)*, the model system consisting of* (2.16)–(2.18) *has a unique solution which is non-negative and bounded for all* $t \geq 0$.

As we have seen above, systems (2.18) and (2.17) only describe the disease dynamics on the transient intervals $[0, \tau]$ and $[\tau_2, \tau_1]$ respectively, and the long term disease dynamics is described by (2.16). In the rest of this paper, we will only investigate the dynamics of (2.16).

4 Disease free equilibrium and basic reproduction number

A disease free equilibrium of model (2.16) is the equilibrium with the infection related components being zeros. That is, such an equilibrium has the form

$$E_0 = (\bar{S}_{11}^0, \ldots, \bar{S}_{n1}^0, \bar{S}_{12}^0, \ldots, \bar{S}_{n2}^0, \underbrace{0, \ldots, 0}_{4n}).$$

Denote $\bar{S}_1^0 = (\bar{S}_{11}^0, \ldots, \bar{S}_{n1}^0)$ and $\bar{S}_2^0 = (\bar{S}_{12}^0, \ldots, \bar{S}_{n2}^0)$. It is immediately noticed that $\bar{S}_2^0 = (M_1, \ldots, M_n)$ and $\bar{S}_1^0$ satisfies the following linear algebraic system

$$Q\bar{S}_1^0 = K, \tag{4.1}$$

where $K = (K_{11}, \ldots, K_{n1})^T$ and

$$Q = \begin{pmatrix} d_{11} + \sum_{j=1}^n D_{j1}^S & -D_{12}^S & \cdots & -D_{1n}^S \\ -D_{21}^S & d_{21} + \sum_{j=1}^n D_{j2}^S & \cdots & -D_{2n}^S \\ \vdots & \vdots & \ddots & \vdots \\ -D_{n1}^S & -D_{n2}^S & \cdots & d_{n1} + \sum_{j=1}^n D_{jn}^S \end{pmatrix}.$$

The irreducibility of D^S ensures that Q is also irreducible. Since Q has non-positive off-diagonal entries and positive column sums, Q is a non-singular M-matrix. This shows that system (4.1) has a unique positive solution $\bar{S}_0^1 = Q^{-1}K > 0$, implying that system (2.16) only admits one disease free equilibrium E_0.

The basic reproduction number of a disease model is usually closely related to the stability of the disease free equilibrium. To proceed further, we linearize system (2.16) at E_0. Note that the equations of S_{i1}, S_{i2}, L_{i1} and L_{i2} decouple from the equations for I_{i1} and I_{i2} which read

$$\begin{cases} \dfrac{\mathrm{d}I_{i1}(t)}{\mathrm{d}t} = -(d_{i1} + \gamma_i + \mu_i)I_{i1}(t) + \displaystyle\sum_{j=1}^n D_{ij}^I I_{j1}(t) - \sum_{j=1}^n D_{ji}^I I_{i1}(t) \\ \qquad\qquad + \displaystyle\sum_{j=1}^n p_{ij}(\tau_1)a_j c_{j1} I_{j2}(t-\tau_1), \\ \dfrac{\mathrm{d}I_{i2}(t)}{\mathrm{d}t} = -d_{i2}I_{i2}(t) + a_i c_{i2}\dfrac{M_i}{\bar{S}_{i1}^0} e^{-d_{i2}\tau_2} I_{i1}(t-\tau_2). \end{cases} \tag{4.2}$$

Let

$$\mathbf{F}_1 = \begin{pmatrix} 0 & F_1 \\ 0 & 0 \end{pmatrix}, \quad \mathbf{F}_2 = \begin{pmatrix} 0 & 0 \\ F_2 & 0 \end{pmatrix}, \quad \mathbf{V} = \begin{pmatrix} V_1 & 0 \\ 0 & V_2 \end{pmatrix},$$

where the $n \times n$ matrices F_1, F_2, V_1 and V_2 are defined as below:

$$\begin{aligned}
&(F_1)_{ij} = p_{ij}(\tau_1) a_j c_{j1}, \quad i, j = 1, \ldots, n,\\
&F_2 = diag\left(a_1 c_{12} e^{-d_{12}\tau_2} \frac{M_1}{\bar{S}^0_{11}}, \ldots, a_n c_{n2} e^{-d_{n2}\tau_2} \frac{M_n}{\bar{S}^0_{n1}} \right),\\
&(V_1)_{ij} = \begin{cases} d_{i1} + \gamma_i + \mu_i + \sum_{k=1}^{n} D^I_{ki}, & \text{for} \quad j = i, \\ -D^I_{ij} \quad \text{for} \quad j \neq i, \end{cases}\\
&V_2 = diag\,(d_{12}, d_{22}, \ldots, d_{n2}).
\end{aligned}$$

Denote $I_1(t) = (I_{11}, \cdots, I_{n1})$ and $I_2(t) = (I_{12}, \cdots, I_{n2})$ and let $I(t) = (I_1(t), I_2(t))$. Obviously, the equations in system (4.2) containing the components of $I(t)$ actually decouple from the rest, giving a subsystem:

$$\frac{\mathrm{d}}{\mathrm{d}t} I(t) = \mathbf{F}_1 I(t - \tau_1) + \mathbf{F}_2 I(t - \tau_2) - \mathbf{V} I(t). \tag{4.3}$$

Since system (2.16) (hence (4.3)) is not an ODE system, the recipe for calculating the spectral radius of the next generation *matrix* given in Driessche and Watmough (2002) cannot be directly applied to define the basic reproduction number for this model. Below, we will adopt a more general notion of the next generation *operator* which provides an analogue of the next generation *matrix* for structured models described by *infinite* dimensional systems, including system (2.16). We now follow the approach in Diekmann and Heesterbeek (2000) and Thieme (2009) to define the basic reproduction number $\mathcal{R}_0$ for our model. To this end, we need to identify the next generation operator for our model.

Assume that the populations of hosts and vectors are settled at E_0 and there is no infectious individual before the time $t = 0$. Suppose that at $t = 0$, there are some infectious individuals introduced into this patchy environment. Then near E_0, the infectious populations $I_1(t)$ and $I_2(t)$ are governed by system (4.3). Note that the first two terms in system (4.3) track new infections while the last term takes care of evolution with respect to time, tracking the survival and dispersal of the infected individuals.

Define a positive linear operator $\mathcal{F} : R^n_+ \times R^n_+ \to R^n_+ \times R^n_+$ by

$$\mathcal{F}(\sigma) = (F_1\sigma_2, F_2\sigma_1) \quad \text{for} \quad \sigma = (\sigma_1, \sigma_2) \in R^n_+ \times R^n_+.$$

Let $U(t) = (U_1(t), U_2(t))$ be the semi-group generated by

$$\frac{\mathrm{d}}{\mathrm{d}t} I(t) = -\mathbf{V} I(t),$$

that is

$$U(t)\sigma = (e^{-V_1 t}\sigma_1, e^{-V_2 t}\sigma_2), \quad \text{for} \quad \sigma = (\sigma_1, \sigma_2) \in R_+^n \times R_+^n.$$

Now, let the initial distribution $I_0 = (I_0^1, I_0^2)$, where $I_0^1 = (I_{11}(0), \ldots, I_{n1}(0))$ and $I_0^2 = (I_{12}(0), \ldots, I_{n2}(0))$ be given. Due to the latency within hosts, the production of new infectious hosts will not occur before $t = \tau_1$, and hence, the number of the cumulative new infectious hosts is given by

$$\int_{\tau_1}^{\infty} F_1[U_2(t-\tau_1) I_0^2]\,\mathrm{d}t = \int_0^{\infty} F_1[U_2(t) I_0^2]\,\mathrm{d}t, \quad t \geq \tau_1. \tag{4.4}$$

Similarly, the number of the cumulative new infectious vectors is

$$\int_{\tau_2}^{\infty} F_2[U_1(t-\tau_2) I_0^1]\,\mathrm{d}t = \int_0^{\infty} F_2[U_1(t) I_0^1]\,\mathrm{d}t, \quad t \geq \tau_2. \tag{4.5}$$

From Eqs. (4.4) and (4.5), the distribution of all new infections caused by the initial distribution I_0 for $t \geq \max\{\tau_1, \tau_2\}$ is

$$\int_0^{\infty} (F_1[U_2(t) I_0^2],\ F_2[U_1(t) I_0^1])\ \mathrm{d}t = \int_0^{\infty} \mathcal{F} U(t) I_0\ \mathrm{d}t.$$

This identifies the next generation operator $\mathcal{T}$ of the model:

$$\mathcal{T}(\sigma) := \int_0^{\infty} \mathcal{F}[U(t)\sigma]\,\mathrm{d}t, \quad \text{for} \quad \sigma = (\sigma_1, \sigma_2) \in R_+^n \times R_+^n. \tag{4.6}$$

The basic reproduction number is then defined as the spectral radius of $\mathcal{T}$: $\mathcal{R}_0 = \rho(\mathcal{T})$ (see, e.g. , Diekmann and Heesterbeek 2000; Thieme 2009). Note that (4.6) can be rewritten as

$$\mathcal{T}(\sigma) = \int_0^{\infty} \mathbf{F} e^{-\mathbf{V}t}\sigma\ \mathrm{d}t = \left(\int_0^{\infty} \mathbf{F} e^{-\mathbf{V}t}\,\mathrm{d}t \right) \sigma \quad \text{for} \quad \sigma = (\sigma_1, \sigma_2) \in R_+^n \times R_+^n, \tag{4.7}$$

where $\mathbf{F} = \mathbf{F}_1 + \mathbf{F}_2$. Thus, $\mathcal{R}_0$ can be further expressed as

$$\mathcal{R}_0 := \rho(\mathcal{T}) = \rho\left(\int_0^\infty \mathbf{F}e^{-\mathbf{V}t}\,\mathrm{d}t\right) = \rho\left(\mathbf{F}\int_0^\infty e^{-\mathbf{V}t}\,\mathrm{d}t\right) = \rho(\mathbf{F}\mathbf{V}^{-1}). \tag{4.8}$$

As in all disease models, by the biological meaning of the basic reproduction number $\mathcal{R}_0$, the stability of E_0 should be equivalent to $\mathcal{R}_0 < 1$, provided that $\mathcal{R}_0$ is correctly identified. We will confirm this below for our model.

It is easy to calculate the characteristic equation of system (4.2) as

$$\begin{aligned}&|zE_{n\times n} + Q| \cdot |zE_{n\times n} + D_2| \cdot |(zE_{n\times n}+V_1)\,(zE_{n\times n}+V_2)\\ &\quad -F_2e^{-z\tau_2}F_1e^{-z\tau_1}| = 0,\end{aligned} \tag{4.9}$$

where $D_2 = diag\,(d_{12}, \ldots, d_{n2})$. The other matrices in (4.9) have been defined before. Let

$$\Delta_1(z) = |zE_{2n\times 2n} + Q|\,, \quad \Delta_2(z) = |zE_{2n\times 2n} + D_2|\,,$$
$$\Delta_3(z, \tau_1, \tau_2) = \left|(zE_{n\times n} + V_1)\,(zE_{n\times n} + V_2) - F_2e^{-z\tau_2}F_1e^{-z\tau_1}\right|.$$

Obviously $\Delta_2(z) = 0$ has roots $-d_{i2} < 0,\ i = 1, 2, \ldots, n$. Note that for the matrix $Q = [Q_{ij}]_{n\times n}$,

$$\sum_{j\neq i}|-Q_{ji}| = \sum_{j=1}^{n} D^S_{ji} < d_i + \sum_{j=1}^{n} D^S_{ji} = \left|-d_i - \sum_{j=1}^{n} D^S_{ji}\right| = |-Q_{ii}|\,. \tag{4.10}$$

According to the Gershgorin Circle Theorem (see, e.g.,Varga 2004), any root z of $\Delta_1(z) = 0$ satisfies

$$|z + Q_{ii}| \leq \sum_{j\neq i}|-Q_{ji}| < |-Q_{ii}| = |Q_{ii}|,$$

and hence, must have negative real part. Therefore, the stability of E_0 is fully determined by the distribution of the roots of the equation

$$\Delta_3(z, \tau_1, \tau_2) = 0, \tag{4.11}$$

which is the characteristic equation of (4.3). Noting that $\mathbf{F}_1, \mathbf{F}_2$ and $-\mathbf{V}$ have non-negative off-diagonal entries, (4.3) is a monotone system, therefore, the stability of the trivial solution is equivalent to that of the corresponding ODE system obtained by dropping the two discrete delays (see e.g., Smith 1987, 1995):

$$\frac{\mathrm{d}}{\mathrm{d}t}I(t) = (\mathbf{F}_1 + \mathbf{F}_2 - \mathbf{V})I(t) = (\mathbf{F} - \mathbf{V})I(t). \tag{4.12}$$

Then

$$\max\{Re(z) : \Delta_3(z, \tau_1, \tau_2) = 0\} < 0 \ (> 0) \quad \text{if and only if} \quad s(\mathbf{F} - \mathbf{V}) < 0 \ (> 0),$$

where $s(\mathbf{F} - \mathbf{V})$ is the stability modulus of $\mathbf{F} - \mathbf{V}$ defined as the maximal real part of all eigenvalues of the matrix $\mathbf{F} - \mathbf{V}$. By Theorem 2 in Driessche and Watmough (2002), $s(\mathbf{F} - \mathbf{V}) < 0 \ (> 0)$ is equivalent to $\rho(\mathbf{F}\mathbf{V}^{-1}) < 1 \ (> 1)$. Hence, E_0 is asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$.

Summarizing the above, we have proved the following theorem.

Theorem 4.1 *If $\mathcal{R}_0 = \rho(\boldsymbol{FV}^{-1}) < 1$, then E_0 is asymptotically stable; when $\mathcal{R}_0 > 1$, E_0 is unstable.*

5 Disease persistence and endemic equilibrium

We have seen that when $\mathcal{R}_0 > 1$, the DFE is unstable. In this section we will show that under this circumstances, the disease will persist; moreover, there exists an endemic equilibrium.

As we mentioned in Sect. 2, in systems (2.16)–(2.18), L_{i2} actually decouples from the rest. Thus, we only need to consider the following subsystem as a result of omitting the $\dot{L}_{i2}(t)$ equations in (2.17), (2.18) and (2.16):

$$\begin{cases} \dfrac{\mathrm{d}S_{i1}(t)}{\mathrm{d}t} = K_{i1} - d_{i1}S_{i1}(t) + \gamma_i I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^S_{ij}S_{j1}(t) - \sum_{j=1}^{n} D^S_{ji}S_{i1}(t) \\ \qquad\qquad - a_i c_{i1} I_{i2}(t)\dfrac{S_{i1}(t)}{N_i(t)}, \\ \dfrac{\mathrm{d}L_{i1}(t)}{\mathrm{d}t} = a_i c_{i1} I_{i2}(t)\dfrac{S_{i1}(t)}{N_i(t)} - d_{i1}L_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^L_{ij}L_{j1}(t) - \sum_{j=1}^{n} D^L_{ji}L_{i1}(t), \\ \dfrac{\mathrm{d}I_{i1}(t)}{\mathrm{d}t} = -(d_{i1} + \gamma_i + \mu_i)I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^I_{ij}I_{j1}(t) - \sum_{j=1}^{n} D^I_{ji}I_{i1}(t), \\ \dfrac{\mathrm{d}S_{i2}(t)}{\mathrm{d}t} = d_{i2}M_i - d_{i2}S_{i2}(t) - a_i c_{i2} S_{i2}(t)\dfrac{I_{i1}(t)}{N_i(t)}, \\ \dfrac{\mathrm{d}I_{i2}(t)}{\mathrm{d}t} = -d_{i2}I_{i2}(t), \end{cases} \quad t \in [0, \tau_2), \tag{5.1}$$

$$
\begin{cases}
\dfrac{\mathrm{d}S_{i1}(t)}{\mathrm{d}t} = K_{i1} - d_{i1}S_{i1}(t) + \gamma_i I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^S_{ij}S_{j1}(t) - \sum_{j=1}^{n} D^S_{ji}S_{i1}(t) \\
\qquad\qquad - a_i c_{i1} I_{i2}(t)\dfrac{S_{i1}(t)}{N_i(t)}, \\
\dfrac{\mathrm{d}L_{i1}(t)}{\mathrm{d}t} = a_i c_{i1} I_{i2}(t)\dfrac{S_{i1}(t)}{N_i(t)} - d_{i1}L_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^L_{ij}L_{j1}(t) - \sum_{j=1}^{n} D^L_{ji}L_{i1}(t), \\
\dfrac{\mathrm{d}I_{i1}(t)}{\mathrm{d}t} = -(d_{i1} + \gamma_i + \mu_i)I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^I_{ij}I_{j1}(t) - \sum_{j=1}^{n} D^I_{ji}I_{i1}(t), \\
\dfrac{\mathrm{d}S_{i2}(t)}{\mathrm{d}t} = d_{i2}M_i - d_{i2}S_{i2}(t) - a_i c_{i2} S_{i2}(t)\dfrac{I_{i1}(t)}{N_i(t)}, \\
\dfrac{\mathrm{d}I_{i2}(t)}{\mathrm{d}t} = -d_{i2}I_{i2}(t) + a_i c_{i2} S_{i2}(t-\tau_2)\dfrac{I_{i1}(t-\tau_2)}{N_i(t-\tau_2)}\, e^{-d_{i2}\tau_2},
\end{cases}
\qquad t \in [\tau_2, \tau_1), \tag{5.2}
$$

and

$$
\begin{cases}
\dfrac{\mathrm{d}S_{i1}(t)}{\mathrm{d}t} = K_{i1} - d_{i1}S_{i1}(t) + \gamma_i I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^S_{ij}S_{j1}(t) - \sum_{j=1}^{n} D^S_{ji}S_{i1}(t) \\
\qquad\qquad - a_i c_{i1} I_{i2}(t)\dfrac{S_{i1}(t)}{N_i(t)}, \\
\dfrac{\mathrm{d}L_{i1}(t)}{\mathrm{d}t} = a_i c_{i1} I_{i2}(t)\dfrac{S_{i1}(t)}{N_i(t)} - d_{i1}L_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^L_{ij}L_{j1}(t) - \sum_{j=1}^{n} D^L_{ji}L_{i1}(t) \\
\qquad\qquad - \displaystyle\sum_{j=1}^{n} p_{ji}(\tau_1) a_i c_{i1} I_{i2}(t-\tau_1)\dfrac{S_{i1}(t-\tau_1)}{N_i(t-\tau_1)}, \\
\dfrac{\mathrm{d}I_{i1}(t)}{\mathrm{d}t} = -(d_{i1} + \gamma_i + \mu_i)I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^I_{ij}I_{j1}(t) - \sum_{j=1}^{n} D^I_{ji}I_{i1}(t) \\
\qquad\qquad + \displaystyle\sum_{j=1}^{n} p_{ij}(\tau_1) a_j c_{j1} I_{j2}(t-\tau_1)\dfrac{S_{j1}(t-\tau_1)}{N_j(t-\tau_1)}, \\
\dfrac{\mathrm{d}S_{i2}(t)}{\mathrm{d}t} = d_{i2}M_i - d_{i2}S_{i2}(t) - a_i c_{i2} S_{i2}(t)\dfrac{I_{i1}(t)}{N_i(t)}, \\
\dfrac{\mathrm{d}I_{i2}(t)}{\mathrm{d}t} = -d_{i2}I_{i2}(t) + a_i c_{i2} S_{i2}(t-\tau_2)\dfrac{I_{i1}(t-\tau_2)}{N_i(t-\tau_2)}\, e^{-d_{i2}\tau_2},
\end{cases}
\qquad t \geq \tau_1. \tag{5.3}
$$

The break-down of the model into (5.1)–(5.3) starting with the ODE system (5.1) seems to suggest that it is more convenient to establish persistence in $\Re^{5n}$. Consider the scenario in which there is no infectious individual in the patchy environment before

$t = 0$. At $t = 0$, let $(S_1(0), S_2(0), L_1(0), I_1(0), I_2(0)) = (S_1^0, S_2^0, L_1^0, I_1^0, I_2^0)$ where $S_j^0 = (S_{1j}^0, \ldots, S_{nj}^0) \in \Re_+^n$, $L_1^0 = (L_{11}^0, \ldots, L_{n1}^0) \in \Re_+^n$ and $I_j^0 = (I_{1j}^0, \ldots, I_{nj}^0) \in \Re_+^n$ for $j = 1, 2$. With this initial condition, by Theorem 3.1 , there is a unique solution to the model (5.1)–(5.3), denoted by

$$\Big(S_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0),\ S_2(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0),\ L_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0),$$
$$I_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0),\ I_2(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0)\Big),$$

which is non-negative and bounded. For the convenience of notations, we sometimes omit the initial values when referring to the solution and simply write $(S_1(t), S_2(t), L_1(t), I_1(t), I_2(t))$ if there is no confusion. Then we further have the following observations:

(O1) $S_j(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) \gg 0$ for $j = 1, 2$ and $t > 0$. Here and in the sequel, the notation $\gg$ means that all components are positive.

(O2) If $I_1^0 = 0$ and $I_2^0 = 0$, then $I_{i1}(t) = 0$ and $I_{i2}(t) = 0$ for all $t \geq 0$. This can be shown by applying the constant-variation formula to systems (5.1)–(5.3) consecutively.

(O3) Assume that either $I_1^0 > 0$ (i.e., $I_1^0 \geq 0$ but $I_1^0 \neq 0$, i.e., at least one component is positive) or $I_2^0 > 0$, meaning that the disease is brought to at least one patch either by hosts or vectors at $t = 0$. In this case, we can show that $I_1(t) > 0$ or $I_2(t) > 0$ for $t \in [0, \tau_2]$ from system (5.1). Moving on to the interval $[\tau_2, \tau_1]$ and by (5.2), we further know that $I_1(t) > 0$ or $I_2(t) > 0$ for $t \in [0, \tau_1]$. Finally, for $t > \tau_1$, from system (5.3) and by the irreducibility and positivity of the matrix $P = e^{B\tau_1}$, we conclude that $I_1(t) \gg 0$ and $I_2(t) \gg 0$ for $t \geq \tau_1$. Thus components of $I_1(t)$ and $I_2(t)$ are positive for $t \geq \tau_1$.

(O4) If both $I_1^0 > 0$ and $I_2^0 > 0$ are true, then repeating the argument for (O3) concludes that $I_1(t)$ and $I_2(t)$ are positive for $t \geq 0$.

(O5) If both $I_1^0 = 0$ and $I_2^0 = 0$ are true, but $L_1^0 > 0$, then there is as least one component of L_1^0 that is positive. Assume $L_{i_0 1} > 0$ for some $i_0 \in \{1, 2, \ldots, n\}$, that is,

$$L_{i_0 1}(0) = \int_0^{\tau_1} l_{i_0}(0, a)\, \mathrm{d}a > 0,$$

which implies that $l_{i_0}(0, a_0) > 0$ for some $a_0 \in [0, \tau_1]$. By formula (2.2) for $l(t, a)$ and equation (2.13), we can extend $U^{\xi}(t)$ for $\xi > 0$ to include $U^{-a_0}(t)$, leading to

$$\begin{aligned} l(\tau_1 - a_0, \tau_1) &= U^{-a_0}(\tau_1 - a_0) \\ &= e^{B(\tau_1 - a_0)}\, (l_1(0, a_0),\ l_2(0, a_0),\ \ldots,\ l_n(0, a_0)) \\ &> 0. \end{aligned}$$

Now by the continuity of $I(t, a)$,

$$I_{i1}(\tau_1 - a_0) = \int_{\tau_1}^{\infty} I_i(\tau_1 - a_0, a)\, \mathrm{d}a > 0, \quad \text{for all } i \in \{1,\ 2,\ ...,\ n\}.$$

Repeating the argument for (O3) with a shifting of initial time, we conclude that $I_1(t)$ and $I_2(t)$ are positive for $t \geq 2\tau_1 - a_0$.

Denote

$$\mathbf{X} = \Re_+^{5n} = \{(X_1, X_2, Y_1, Z_1, Z_2) : X_j \in \Re_+^n,\ Y_1 \in \Re_+^n,\ Z_j \in \Re_+^n,\ j = 1, 2\}$$
$$\mathbf{X}_0 = \{(X_1, X_2, Y_1, Z_1, Z_2) \in X : Y_1 \gg 0,\ Z_1 \gg 0,\ Z_2 \gg 0\},$$

and let $\partial\mathbf{X}_0 = \mathbf{X} \setminus \mathbf{X}_0$. Then

$$\partial\mathbf{X}_0 = \{(X_1, X_2, Y_1, Z_1, Z_2) \in \mathbf{X} : Z_{i1} = 0 \text{ or } Z_{i2} = 0 \text{ or } Y_{i1} = 0, \text{ at least for one } i\}.$$

We have seen from the above that both $\mathbf{X}$ and $\mathbf{X}_0$ are positive invariant sets for the solution semi-flow $\Phi(t)$ of model (5.1)–(5.3). It is obvious that $\partial\mathbf{X}_0$ is relatively closed in $\mathbf{X}$. Theorem 3.1 and (3.2) also confirm that (5.1)–(5.3) are point-dissipative systems in $\mathbf{X}$ since the bounded set $[0, \bar{K}_1/\underline{d}_1]^{5n}$ attracts all orbits of (5.1)–(5.3) in $\mathbf{X}$.

Next, let

$$\Omega_{\partial 1} = \{(S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) \in \mathbf{X} : (S_1(t), S_2(t), L_1(t), I_1(t), I_2(t)) \in \partial\mathbf{X}_0, \text{ for } t > 0\}.$$

We first prove the following lemma which shows that $\Omega_{\partial 1}$ can also be characterized by the following set

$$\Omega_{\partial 2} = \Big\{(S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) \in X : I_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) = 0,$$
$$I_2(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) = 0, L_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) = 0 \text{ for } t > 0\Big\}.$$

Lemma 5.1 $\Omega_{\partial 2} = \Omega_{\partial 1} =: \Omega_{\partial}$.

Proof Indeed $\Omega_{\partial 2} \subset \Omega_{\partial 1}$ is obvious, so we only need to show $\Omega_{\partial 1} \subset \Omega_{\partial 2}$. Let $(S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) \in \Omega_{\partial 1}$. We need to show that $I_i(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) = 0,\ i = 1, 2$ and $L_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) = 0$ for all $t > 0$. Assume the opposite, that is, there exist an i and a $t_0 > 0$ such that either (A) $I_{i1}(t_0, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) > 0$; or (B) $I_{i2}(t_0, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) > 0$; or (C) $L_{i1}(t_0, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) > 0$. We show below that each of these three cases will lead to a contradiction.

With respect to (A), we have three cases: (A-1) $t_0 \in [0, \tau_2)$; (A-2) $t_0 \in [\tau_2, \tau_1)$; (A-3) $t_0 \in [\tau_1, \infty)$. In case (A-1), similar to (O3) above, we know from system (5.1) that

$$I_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0)) > 0, \quad I_2(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) \geq 0 \text{ for } t \in [t_0, \tau_2);$$

and from system (5.2) that

$$I_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0)) > 0, \quad I_2(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) > 0 \text{ for } t \in [\tau_2, \tau_1);$$

and finally from system (5.3) that,

$$I_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0)) \gg 0, \quad I_2(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) \gg 0 \text{ for } t \in [\tau_1, \infty).$$

This implies that for $t \geq \tau_1$,

$$\Big(S_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0),\ S_2(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0),\ I_1(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0),$$
$$I_2(t, S_1^0, S_2^0, L_1^0, I_1^0, I_2^0)\Big) \in \mathbf{X}_0,$$

which is a contradiction to $(S_1^0, S_2^0, L_1^0, I_1^0, I_2^0) \in \Omega_{\partial 1}$. For (A-2) and (A-3), similar arguments will also lead to contradictions.

With respect to (B), there are also three cases: (B-1) $t_0 \in [0, \tau_2)$; (B-2) $t_0 \in [\tau_2, \tau_1)$; (B-3) $t_0 \in [\tau_1, \infty)$. By similar arguments, each of these cases will lead to a contradiction as well.

For the case (C), by the observation of (O5) and similar arguments, we can also get a contradiction.

Summarizing the three cases, we conclude that $\Omega_{\partial 1} = \Omega_{\partial 2}$, which will be denoted by Ω_∂ in the sequel. □

The next lemma establishes weak persistence of the disease in the sense that both I_1 and I_2 persist.

Lemma 5.2 *Assume that $\mathcal{R}_0 > 1$. Then there is an $\varepsilon > 0$ such that for any solution of (5.3) that eventually enters X_0, we have*

$$\limsup_{t\to\infty} \max\{I_{ij}(t), i = 1, \ldots, n; \quad j = 1, 2\} \geq \varepsilon. \tag{5.4}$$

Proof For the sake of contradiction, assume that (5.4) is false. Then, for any $\varepsilon > 0$, there is a $T_1 > \tau_1$ such that

$$0 < I_{ij}(t) < \varepsilon \text{ for } t \geq T_1, \ i = 1, \ldots, n; \quad j = 1, 2. \tag{5.5}$$

It follows from (5.3) that

$$\frac{\mathrm{d}S_{i1}}{\mathrm{d}t} \geq (K_{i1} - a_i c_{i1}\varepsilon) - d_{i1}S_{i1} + \sum_{j=1}^{n} D_{ij}^S S_{j1} - \sum_{j=1}^{n} D_{ji}^S S_{i1}, \quad t \geq T_1, \tag{5.6}$$

and

$$\frac{\mathrm{d}S_{i1}}{\mathrm{d}t} \leq (K_{i1} + \gamma_i\varepsilon) - d_{i1}S_{i1} + \sum_{j=1}^{n} D_{ij}^S S_{j1} - \sum_{j=1}^{n} D_{ji}^S S_{i1}, \quad t \geq T_1. \tag{5.7}$$

The above inequalities suggest the following two comparison systems for $S_1(t)$:

$$\frac{\mathrm{d}Y_{i2}}{\mathrm{d}t} = (K_{i1} - a_i c_{i1}\varepsilon) - d_{i1}S_{i2} + \sum_{j=1}^{n} D_{ij}^{S} Y_{j2} - \sum_{j=1}^{n} D_{ji}^{S} Y_{i2}, \quad t \geq T_1, \tag{5.8}$$

and

$$\frac{\mathrm{d}Y_{i3}}{\mathrm{d}t} = (K_{i1} + \gamma_i\varepsilon) - d_{i1}S_{i1} + \sum_{j=1}^{n} D_{ij}^{S} Y_{j3} - \sum_{j=1}^{n} D_{ji}^{S} Y_{i3}, \quad t \geq T_1. \tag{5.9}$$

The comparison theorem (see, e.g., Smith 1995) shows that

$$Y_2(t) \leq S_1(t) \leq Y_3(t), \quad \text{for } t \geq T_1. \tag{5.10}$$

Similarly, from the $L_1(t)$ equation in (5.3), we have

$$\frac{\mathrm{d}L_{i1}(t)}{\mathrm{d}t} \leq a_i c_{i1}\varepsilon - d_{i1}L_{i1}(t) + \sum_{j=1}^{n} D_{ij}^{L} L_{j1}(t) - \sum_{j=1}^{n} D_{ji}^{L} L_{i1}(t), \quad t \geq T_1, \tag{5.11}$$

suggesting the comparison system for $L_1(t)$,

$$\frac{\mathrm{d}Y_{i4}(t)}{\mathrm{d}t} = a_i c_{i1}\varepsilon - d_{i1}Y_{i4}(t) + \sum_{j=1}^{n} D_{ij}^{L} Y_{j4}(t) - \sum_{j=1}^{n} D_{ji}^{L} Y_{i4}(t), \quad t \geq T_1, \tag{5.12}$$

and then, leading to

$$0 \leq L_1(t) \leq Y_4(t), \quad \text{for } t \geq T_1, \tag{5.13}$$

where $Y_4(t)$ satisfies (5.12).

Note system (5.9) has a globally asymptotically stable equilibrium $\bar{Y}_2(\varepsilon) = Q_s^{-1}K_2(\varepsilon)$ where $K_2(\varepsilon) = (K_{11} + \gamma_1\varepsilon, \ldots, K_{n1} + \gamma_n\varepsilon)$, and system (5.8) has a globally asymptotically equilibrium $\bar{Y}_3(\varepsilon) = Q_s^{-1}K_3(\varepsilon)$ where $K_3(\varepsilon) = (K_{11} - a_1c_{11}\varepsilon, \ldots, K_{n1} - a_nc_{n1}\varepsilon)$. Similarly, system (5.12) also has a globally asymptotically stable positive equilibrium $\bar{Y}_4(\varepsilon)$. Notice that $\bar{Y}_2(\varepsilon)$, $\bar{Y}_3(\varepsilon)$ and $\bar{Y}_4(\varepsilon)$ are all continuous in ε with $\bar{Y}_2(\varepsilon) \to \bar{S}_1^0$, $\bar{Y}_3(\varepsilon) \to \bar{S}_1^0$ and $\bar{Y}_4(\varepsilon) \to 0$ as $\varepsilon \to 0$. Hence, for any given $\eta > 0$, there is an $\varepsilon_0 \leq \eta$ such that

$$\bar{S}_1^0 - \hat{\eta} \leq \bar{Y}_2(\varepsilon), \quad \bar{Y}_3(\varepsilon) \leq \bar{S}_1^0 + \hat{\eta} \text{ and } 0 \leq \bar{Y}_4(\varepsilon) \leq \hat{\eta} \text{ for } \varepsilon \in (0, \varepsilon_0), \tag{5.14}$$

where $\hat{\eta}$ denotes the n-dimensional vector with all components equal to η. Thus, for sufficiently large t, we have

$$\bar{S}_1^0 - \hat{\eta} \leq S_1(t) \leq \bar{S}_1^0 + \hat{\eta} \text{ and } 0 \leq L_1(t) \leq \hat{\eta}. \tag{5.15}$$

Choose $\eta < \min\{\bar{S}^0_{i1} : i = 1, \ldots, n\}$ and $\varepsilon \in (0, \varepsilon_0)$. Then for t sufficiently large,

$$0 < \bar{S}^0_{i1} - \eta < N_i(t) = S_{i1}(t) + L_{i1}(t) + I_{i1}(t) \le \bar{S}^0_{i1} + 3\eta, \quad i = 1, \ldots, n. \tag{5.16}$$

Next we consider the $S_2(t)$ equation in system (5.3). By relations (5.5), (5.15) and (5.16), we know that for sufficiently large t,

$$\frac{I_{i1}(t)}{N_i(t)} \le \frac{\varepsilon}{\bar{S}^0_{i1} - \eta} \le \frac{\eta}{\bar{S}^0_{i1} - \eta}.$$

This together with system (2.18) leads to

$$\frac{\mathrm{d}S_{i2}(t)}{\mathrm{d}t} \ge d_{i2}M_i - d_{i2}S_{i2}(t) - a_i c_{i2}\frac{\eta}{\bar{S}^0_{i1} - \eta}S_{i2}(t). \tag{5.17}$$

By an analogous argument, we conclude that for t sufficiently large,

$$S_{i2}(t) \ge \frac{d_i(\bar{S}^0_{i1} - \eta)}{d_i(\bar{S}^0_{i1} - \eta) + a_i c_{i1}\eta}M_i =: \bar{Y}_{i5}(\eta). \tag{5.18}$$

Obviously, $Y_{i5}(\eta)$ is continuous in η and $Y_{i5}(0) = M_i$.

We now apply the above estimates to the $I'_{i1}(t)$ and $I'_{i2}(t)$ equations in system (5.3), yielding the following

$$\begin{cases} \dfrac{\mathrm{d}I_{i1}}{\mathrm{d}t} \ge -(d_{i1} + \gamma_i + \mu_i)I_{i1}(t) + \displaystyle\sum_{j=1}^{n} D^I_{ij}I_{j1}(t) - \sum_{j=1}^{n} D^I_{ji}I_{i1}(t) \\ \qquad\qquad + \displaystyle\sum_{j=1}^{n} p_{ij}(\tau_1)a_j c_{j1}h_j(\eta)I_{j2}(t - \tau_1), \\ \dfrac{\mathrm{d}I_{i2}}{\mathrm{d}t} \ge -d_{i2}I_{i2} + a_i c_{i2}\, e^{-d_{i2}\tau_2}g_i(\eta)I_{i1}(t - \tau_2), \end{cases} \tag{5.19}$$

where

$$h_i(\eta) = \frac{\bar{S}^0_{i1} - \eta}{\bar{S}^0_{i1} + 3\eta}, \quad g_i(\eta) = \frac{\bar{Y}_{i5}(\eta)}{\bar{S}^0_{i1} + 3\eta}, \quad i = 1, \ldots, n.$$

Consider the following comparison system obtained from the right-hand side of system (5.19):

$$\frac{\mathrm{d}W(t)}{\mathrm{d}t} = \mathbf{G}_1(\eta)W(t - \tau_1) + \mathbf{G}_2(\eta)W(t - \tau_2) - \mathbf{V}W(t), \tag{5.20}$$

where the matrix $\mathbf{V}$ is as in Sect. 3.4, and

$$\mathbf{G}_1(\eta) = \begin{pmatrix} 0 & G_1(\eta) \\ 0 & 0 \end{pmatrix}, \quad \mathbf{G}_1(\eta) = \begin{pmatrix} 0 & 0 \\ G_1(\eta) & 0 \end{pmatrix},$$

with

$$G_1(\eta) = \begin{pmatrix} b_{11}(\tau_1)a_1c_{11}h_1(\eta) & b_{12}(\tau_1)a_2c_{21}h_2(\eta) & \cdots & b_{1n}(\tau_1)a_nc_{n1}h_n(\eta) \\ b_{21}(\tau_1)a_1c_{11}h_1(\eta) & b_{22}(\tau_1)a_2c_{21}h_2(\eta) & \cdots & b_{2n}(\tau_1)a_nc_{n1}h_n(\eta) \\ \vdots & \vdots & \ddots & \vdots \\ b_{n1}(\tau_1)a_1c_{11}h_1(\eta) & b_{n2}(\tau_1)a_2c_{21}h_2(\eta) & \cdots & b_{nn}(\tau_1)a_nc_{n1}h_n(\eta) \end{pmatrix},$$

and

$$G_2(\xi) = \begin{pmatrix} a_1c_{12}e^{-d_{12}\tau_2}g_1(\eta) & 0 & \cdots & 0 \\ 0 & a_2c_{22}e^{-d_{22}\tau_2}g_2(\eta) & \cdots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & a_nc_{n2}e^{-d_{n2}\tau_2}g_n(\eta) \end{pmatrix}.$$

This linear delay system is monotone and hence, the stability/instability of the trivial solution is independent of τ_1 and τ_2. Let $\mathbf{G}(\eta) = \mathbf{G}_1(\eta)+\mathbf{G}_2(\eta)$. By the same argument for the stability of system (4.12), we know that if $\rho(\mathbf{G}(\eta)\mathbf{V}^{-1}) > 1$, then the trivial solution of system (5.20) is unstable, implying that system (5.20) has unbounded solutions, since it is a linear system. Note that $h_i(\eta)$ and $g_i(\eta)$ are continuous in η with $h_i(0) = 1$ and $g_i(0) = M_i/\bar{S}^0_{i1}$. This implies that $\mathbf{G}_1(\eta) \to \mathbf{F}_1(\eta)$ and $\mathbf{G}_2(\eta) \to \mathbf{F}_2(\eta)$, and hence, $\mathbf{G}(\eta) \to \mathbf{F}$ as $\eta \to 0$. Now since $\mathcal{R}_0 = \rho(\mathbf{F}\mathbf{V}^{-1}) > 1$, by continuity, we can choose η sufficiently small so that $\rho(\mathbf{G}(\eta)\mathbf{V}^{-1}) > 1$, and therefore, system (5.20) has unbounded solutions; by (5.19) and the comparison theorem for delay differential equations (see, e.g., Smith 1995), system (5.3) also has unbounded solutions, contradicting to the results in Theorem 3.1. This proves the lemma. □

We are now in the position to state and prove the main results in this section.

Theorem 5.1 *Assume that $\mathcal{R}_0 > 1$. Then the disease is uniformly persistent in the sense that there exists an $\varepsilon > 0$ such that for any solution of system (5.3) that eventually enters $\boldsymbol{X_0}$, we have*

$$\liminf_{t\to\infty} I_{ij}(t) \geq \varepsilon \text{ for } i = 1, \ldots, n; \quad j = 1, 2. \tag{5.21}$$

Moreover, there exists a positive (endemic) equilibrium, that is, an equilibrium with all components positive.

Proof Note that $\bar{S}^0 = (\bar{S}_1^0, \bar{S}_2^0)$ is globally asymptotically stable in $\Re_+^{2n}/\{0\}$ for the system consisting of the S_{i1} and S_{21} equations resulting from setting $I_{i1} = 0$ and $I_{21} = 0$ in (2.16). Moreover, by Lemmas 5.1-5.2, E_0 is an isolated invariant set in $\mathbf{X}$, and the stable manifold of E_0 does not intersect the interior of $\mathbf{X}_0$. Also note that every orbit in Ω_∂ converges to E_0 (hence E_0 is an isolated equilibrium in $\mathbf{X}$). By Theorem 4.6 in Thieme (1993), it follows that the model system (2.16)–(2.18) is uniformly persistent with respect to $(\mathbf{X}_0, \partial\mathbf{X}_0)$, and hence (5.21) holds. Further by Theorem 2.4 in Zhao (1995), there is an equilibrium in $\mathbf{X}_0$, denoted by $E^* = (S_1^*, S_2^*, L_1^*, I_1^*, I_2^*)$, where $S_1^* \geq 0$, $S_2^* \geq 0$, $L_1^* \gg 0$, $I_1^* \gg 0$ and $I_2^* \gg 0$. From the I_{i2} equations in system (2.18) and the fact that $I_1^* \gg 0$ and $I_2^* \gg 0$, it follows that $S_2^* \gg 0$. So, it remains to show that $S_1^* \gg 0$ as well. Firstly, we claim that $S_1^* > 0$, otherwise, the S_{i1} equations would lead to $I_{i1}^* = -K_{i1}/\gamma_i < 0$, a contradiction. Rewrite the $S_{i1}'(t)$ equations as

$$\dot{S}_1(t) = [Q - Q_1(t)]S_1(t) + [K + M(t)],$$

where Q and K as in Sect. 3.2, $M(t) = (\gamma_1 I_{11}(t), \cdots, \gamma_n I_{21}(t))^T$ and

$$Q_1(t) = diag\left(\frac{a_1 c_{11} I_{12}(t)}{N_1(t)}, \frac{a_2 c_{21} I_{22}(t)}{N_2(t)}, \ldots, \frac{a_n c_{n1} I_{n2}(t)}{N_n(t)}\right).$$

Since $S_1^* = S_1(t, S_1^*, S_2^*, I_1^*, I_2^*)$, S_1^* can be expressed by

$$S_1^* = e^{\int_0^t [Q - Q_1(\xi)]\, d\xi} S_1^* + \int_0^t e^{\int_s^t [Q - Q_1(\xi)]\, d\xi} [K + M(\xi)]\, d\xi.$$

By the cooperative and irreducible property of the matrix $Q - Q_1(t)$ and the positivity of $[K + M(\xi)]$, we conclude that $S_1^* \gg 0$. Thus, E^* is positive, completing the proof. □

6 A simple case: $n = 2$ (two patches)

In the previous sections, we have seen that $\mathcal{R}_0 = \rho(\mathbf{F}\mathbf{V}^{-1})$ plays a threshold role. All parameters in system (2.16) are included in the two matrices $\mathbf{F}$ and $\mathbf{V}$, directly or indirectly. We particularly emphasize that the dispersal rates in the three compartments (susceptible, latent and infectious) enter $\mathbf{F}$ and $\mathbf{V}$ in various ways, and hence, we expect to see their effects on $\mathcal{R}_0$ in different ways. Unfortunately, for general n, it is very difficult (if not impossible) to investigate the impact of these dispersal rates on $\mathcal{R}_0$ in explicit form. In this section, we will focus on the simplest patchy environment: two patches, with the hope of obtaining some more explicit and helpful information on how $\mathcal{R}_0$ depends on the various dispersal rates.

When $n = 2$, the matrix B becomes

$$B = \begin{bmatrix} -d_{11} - D_{21}^L & D_{12}^L \\ D_{21}^L & -d_{21} - D_{12}^L \end{bmatrix},$$

and accordingly, the non-local infection matrix $P(\tau_1) = e^{\tau_1 B}$ is given by

$$P(\tau_1) = \left(p_{ij}(\tau_1)\right)_{2\times 2} = \begin{bmatrix} \frac{e^{-d_{11}\tau_1}\left(D_{12}^L + D_{21}^L e^{-(D_{12}^L + D_{21}^L)\tau_1}\right)}{D_{12}^L + D_{21}^L} & \frac{e^{-d_{11}\tau_1} D_{12}^L \left(1 - e^{-(D_{12}^L + D_{21}^L)\tau_1}\right)}{D_{12}^L + D_{21}^L} \\ \frac{e^{-d_{21}\tau_1} D_{21}^L \left(1 - e^{-(D_{12}^L + D_{21}^L)\tau_1}\right)}{D_{12}^L + D_{21}^L} & \frac{e^{-d_{21}\tau_1}\left(D_{12}^L e^{-(D_{12}^L + D_{21}^L)\tau_1} + D_{21}^L\right)}{D_{12}^L + D_{21}^L} \end{bmatrix}. \quad (6.1)$$

The matrices $\mathbf{F}$ and $\mathbf{V}$ in this case have the following expressions:

$$\mathbf{F} = \begin{bmatrix} 0 & 0 & p_{11}(\tau_1)a_1c_{11} & p_{12}(\tau_1)a_2c_{21} \\ 0 & 0 & p_{21}(\tau_1)a_1c_{11} & p_{22}(\tau_1)a_2c_{21} \\ \frac{a_1c_{12}e^{-d_{12}\tau_2}M_1}{\bar{S}_{11}^0} & 0 & 0 & 0 \\ 0 & \frac{a_2c_{22}e^{-d_{22}\tau_2}M_2}{\bar{S}_{21}^0} & 0 & 0 \end{bmatrix},$$

$$\mathbf{V} = \begin{bmatrix} d_{11} + \gamma_1 + \mu_1 + D_{21}^I & -D_{12}^I & 0 & 0 \\ -D_{21}^I & d_{21} + \gamma_2 + \mu_2 + D_{12}^I & 0 & 0 \\ 0 & 0 & d_{12} & 0 \\ 0 & 0 & 0 & d_{22} \end{bmatrix},$$

where

$$\bar{S}_{11}^0 = \frac{D_{12}^S K_{21} + D_{12}^S K_{11} + d_{21}K_{11}}{d_{21}d_{11} + d_{21}D_{21}^S + D_{12}^S d_{11}}, \quad \bar{S}_{21}^0 = \frac{d_{11}K_{21} + D_{21}^S K_{21} + D_{21}^S K_{11}}{d_{21}d_{11} + d_{21}D_{21}^S + D_{12}^S d_{11}}, \quad (6.2)$$

are the components of the solution of the algebraic equation $Qx = K$, regardless of the irreducibility of the matrix Q. With Maple's help, the basic reproduction number is calculated as

$$\mathcal{R}_0 = \rho(\mathbf{F}\mathbf{V}^{-1}) = \frac{1}{2}\sqrt{2r_3s_2 + 2r_4s_4 + 2r_1s_1 + 2r_2s_3 + 2\sqrt{Z}}, \quad (6.3)$$

where

$$\begin{cases} Z = {r_3}^2{s_2}^2 + 2r_3s_2r_4s_4 + 2r_3s_1r_1s_2 - 2r_3s_2r_2s_3 + {r_4}^2{s_4}^2 - 2r_4s_4r_1s_1 \\ \quad + 2r_4s_3r_2s_4 + {r_1}^2{s_1}^2 + 2r_1s_1r_2s_3 + {r_2}^2{s_3}^2 + 4r_3s_1r_2s_4 + 4r_4s_3r_1s_2, \\ r_1 = a_1c_{12}e^{-d_{12}\tau_2}M_1(d_{21} + \gamma_2 + \mu_2 + D_{12}^I)/\left(\det(V_1)\,\bar{S}_{11}^0\right), \\ r_2 = a_1c_{12}e^{-d_{12}\tau_2}M_1D_{12}^I/\left(\det(V_1)\,\bar{S}_{11}^0\right), \\ r_3 = a_2c_{22}e^{-d_{22}\tau_2}M_2D_{21}^I/\left(\det(V_1)\,\bar{S}_{21}^0\right), \\ r_4 = a_2c_{22}e^{-d_{22}\tau_2}M_2(d_{11} + \gamma_1 + \mu_1 + D_{21}^I)/\left(\det(V_1)\,\bar{S}_{21}^0\right), \\ s_1 = p_{11}(\tau_1)a_1c_{11}/d_{12}, \quad s_2 = p_{12}(\tau_1)a_2c_{21}/d_{22}, \\ s_3 = p_{21}(\tau_1)a_1c_{11}/d_{12}, \quad s_4 = p_{22}(\tau_1)a_2c_{21}/d_{22}. \end{cases} \quad (6.4)$$

The expression of the basic reproduction number is still very complicated. For simplicity, we further confine ourselves to two even simpler scenarios on the dispersions between the two patches: (i) only susceptible individuals disperse; (ii) only susceptible and exposed groups disperse. For the case when all three classes of hosts disperse, we only explore it numerically.

Since we are interested in the impact of dispersions, it is natural and helpful to compare with the case when the two patches are isolated, that is, D^S, D^L and D^I are all zero matrices. In this fully isolated situation, the disease free equilibrium is

$$E_0^1 = (K_{11}/d_{11},\ K_{21}/d_{21},\ M_1,\ M_2, 0,\ 0,\ 0,\ 0,\ 0,\ 0),$$

and the basic reproduction number is

$$\mathcal{R}_{01} = \max(\mathcal{R}_{11},\ \mathcal{R}_{21}), \tag{6.5}$$

where

$$\mathcal{R}_{11} = \sqrt{\frac{{a_1}^2 c_{12} c_{11} \mathrm{e}^{(-d_{11}\tau_1 - d_{12}\tau_2)} M_1}{\frac{K_{11}}{d_{11}} (d_{11} + \gamma_1 + \mu_1)\, d_{12}}}, \quad \mathcal{R}_{21} = \sqrt{\frac{{a_2}^2 c_{22} c_{21} \mathrm{e}^{(-d_{21}\tau_1 - d_{22}\tau_2)} M_2}{\frac{K_{21}}{d_{21}} (d_{21} + \gamma_2 + \mu_2)\, d_{22}}}. \tag{6.6}$$

Clearly, $\mathcal{R}_{11}$ and $\mathcal{R}_{21}$ are the local basic reproduction numbers for each patch. Applying the results in work (Xiao and Zou 2013) to each patch, we have the following theorem on the disease dynamics in each patch.

Theorem 6.1 *If $\mathcal{R}_{i1} < 1$, then the disease free equilibrium (DFE): $(K_{i1}/d_{i1},\ 0,\ 0,\ M_i,\ 0,\ 0)$ is asymptotically stable; moreover if $\mu_i = \gamma_i = 0$, then the DFE is globally asymptotically stable. If $\mathcal{R}_{i1} > 1$, the disease uniformly persists in the population in patch i, $i = 1, 2$.*

6.1 Only susceptible individuals disperse

When only susceptible individuals can travel between patches, the dispersal matrices D^L and D^I are zero matrices. A scenario for such an assumption is that all infected individuals are prohibited (e.g., by health authorities) from traveling. In this case, $r_2 = r_3 = s_2 = s_3 = 0$. Denoting the global basic reproduction number (GBRN) by $\mathcal{R}_{02}$ now, (6.3) then yields

$$\begin{aligned}\mathcal{R}_{02} &= \sqrt{\frac{r_4 s_4 + r_1 s_1 + \sqrt{(r_4 s_4 - r_1 s_1)^2}}{2}} = \sqrt{\max\{r_4 s_4, r_1 s_1\}} \\ &= \max\{\sqrt{r_4 s_4}, \sqrt{r_1 s_1}\} = \max\{\mathcal{R}_{12}, \mathcal{R}_{22}\},\end{aligned} \tag{6.7}$$

where

$$\mathcal{R}_{i2} = \sqrt{\frac{{a_i}^2 c_{i1} c_{i2} \mathrm{e}^{-d_{i1}\tau_1} \mathrm{e}^{-d_{i2}\tau_1} M_i}{\bar{S}^0_{i1} d_{i2} (d_{i1} + \gamma_i + \mu_i)}}, \quad i = 1, 2, \tag{6.8}$$

and $(\bar{S}^0_{11},\ \bar{S}^0_{21})$ are given by (6.2). Here, $\mathcal{R}_{i2}$ can be explained as a dispersal mediated reproduction number for patch i.

The impact of the latency on $\mathcal{R}_{02}$ can be directly seen from (6.7)–(6.8), while the impact of the dispersions of susceptible hosts on $\mathcal{R}_{02}$ is reflected by (6.7)–(6.8), and the dependence of $\bar{S}^0_{2i}$ on D^S_{12} and D^S_{21} will be explicitly explored below for a special case.

Assume that the two patches are "identical" in the sense that $K_{11}/d_{11} = K_{21}/d_{21}$ and $\mathcal{R}_{11} = \mathcal{R}_{21}$, and we consider the impact of host dispersal on the GBRN in the case that all other parameters are fixed. Then, in terms of D^S_{21} and D^S_{12}, we have three cases.

(i) Suppose $D^S_{21} < D^S_{12}$. Then

$$\begin{aligned}
&D^S_{12}\frac{K_{21}}{d_{21}} > D^S_{21}\frac{K_{11}}{d_{11}}\\
&\Rightarrow d_{11}D^S_{12}K_{21} > D^S_{21}K_{11}d_{21}\\
&\Rightarrow d_{11}D^S_{12}K_{21} + d_{11}D^S_{12}K_{11} + d_{11}d_{21}K_{11} > d_{21}d_{11}K_{11} + K_{11}d_{21}D^S_{21}\\
&\quad + D^S_{12}d_{11}K_{11}\\
&\Rightarrow d_{11}(D^S_{12}K_{21} + D^S_{12}K_{11} + d_{21}K_{11}) > K_{11}(d_{21}d_{11} + d_{21}D^S_{21} + D^S_{12}d_{11})\\
&\Rightarrow \frac{D^S_{12}K_{21} + D^S_{12}K_{11} + d_{21}K_{11}}{d_{21}d_{11} + d_{21}D^S_{21} + D^S_{12}d_{11}} > \frac{K_{11}}{d_{11}}.
\end{aligned}$$

Hence, we have $\bar{S}^0_{11} > \frac{K_{11}}{d_{11}}$. Similarly, we have $\bar{S}^0_{21} < \frac{K_{21}}{d_{21}}$. Thus, with such an asymmetric dispersal pattern, we have

$$\mathcal{R}_{12} < \mathcal{R}_{11} = \mathcal{R}_{21} < \mathcal{R}_{22},$$

$$\mathcal{R}_{02} = \mathcal{R}_{22} = \max(\mathcal{R}_{12},\ \mathcal{R}_{22}) > \max(\mathcal{R}_{11},\ \mathcal{R}_{21}) = \mathcal{R}_{01}.$$

(ii) Symmetrically, if $D^S_{21} > D^S_{12}$, then

$$\mathcal{R}_{12} > \mathcal{R}_{11} = \mathcal{R}_{21} > \mathcal{R}_{22},$$

$$\mathcal{R}_{02} = \mathcal{R}_{12} = \max(\mathcal{R}_{12},\ \mathcal{R}_{22}) > \max(\mathcal{R}_{11},\ \mathcal{R}_{21}) = \mathcal{R}_{01}.$$

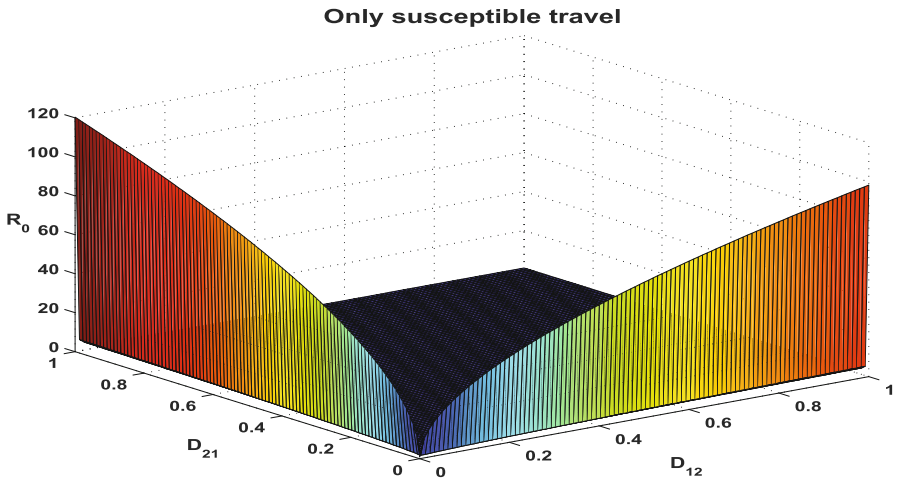

Fig. 2 Dependence of $\mathcal{R}_{02}$ on D_{12}^S and D_{21}^S

(iii) If the dispersions are symmetric, i.e., $D_{21}^S = D_{12}^S$, it is obvious that

$$\mathcal{R}_{12} = \mathcal{R}_{11} = \mathcal{R}_{21} = \mathcal{R}_{22},$$

$$\mathcal{R}_{02} = \mathcal{R}_{12} = \max(\mathcal{R}_{12},\ \mathcal{R}_{22}) = \max(\mathcal{R}_{11},\ \mathcal{R}_{21}) = \mathcal{R}_{01}.$$

It is immediately seen from (i)–(ii) that if the dispersions between two "identical" patches are asymmetric, the dispersal will increase the GBRN $\mathcal{R}_{02}$ of the model.

Even when the two patches are not necessarily "identical" in the above sense (i.e., without assuming $K_{11}/d_{11} = K_{21}/d_{21}$ and $\mathcal{R}_{11} = \mathcal{R}_{21}$), we can also observe the effect of D_{21}^S and D_{21}^S on the GBRN. For example, if we fix $D_{21}^S \geq 0$ and let $D_{12}^S \to \infty$, we find that $\bar{S}_{21}^0 \to 0$ and $\bar{S}_{11}^0 \to (K_{11} + K_{21})/d_{11}$, implying that $\mathcal{R}_{02} \to \infty$. Thus larger D_{12}^S can always bring $\mathcal{R}_{02}$ to a value lager than 1, meaning that such unbalanced travels of hosts (e.g., human) can enhance the survival of the vector-borne disease (e.g., malaria) in a global sense. Fixing $D_{12}^S \geq 0$ and letting $D_{21}^S \to \infty$ will lead to the same conclusion. This conclusion can be confirmed by numerically computing the GBRN. To this end, we adtop the following parameter values from Chitnis et al. (2008) where malaria disease is considered:

$$\begin{cases} K_{11} = K_{21} = 0.0694 \text{ humans} \cdot \text{day}^{-1}, \\ M_1 = 4000 \text{ mosquitoes}, \quad M_2 = 6000 \text{ mosquitoes}, \\ d_{11} = 0.0014 \text{ day}^{-1}, \quad d_{12} = 0.13 \text{ day}^{-1}, \quad d_{21} = 0.0014 \text{ day}^{-1}, \quad d_{22} = 0.13 \text{ day}^{-1}, \\ a_1 = 0.35 \text{ day}^{-1}, \quad a_2 = 0.35 \text{ day}^{-1}, \\ e_{11} = 0.3, \quad e_{12} = 0.0.022, \quad e_{21} = 0.3, \quad e_{22} = 0.022, \\ \gamma_1 = 0.01 \text{ day}^{-1}, \quad \gamma_2 = 0.01 \text{ day}^{-1}, \quad \tau_1 = 20 \text{ days}, \quad \tau_2 = 10 \text{ days}, \\ \mu_1 = 5e-5 \text{ humans} \cdot \text{day}^{-1}, \quad \mu_2 = 5e-5 \text{ humans} \cdot \text{day}^{-1}. \end{cases} \tag{6.9}$$

With these values, numerical computation of $\mathcal{R}_0^2$ by (6.7)–(6.8) in terms of D_{12}^S and D_{21}^S is plotted in Fig. 2. By further fixing one of the two dispersion rates, one may

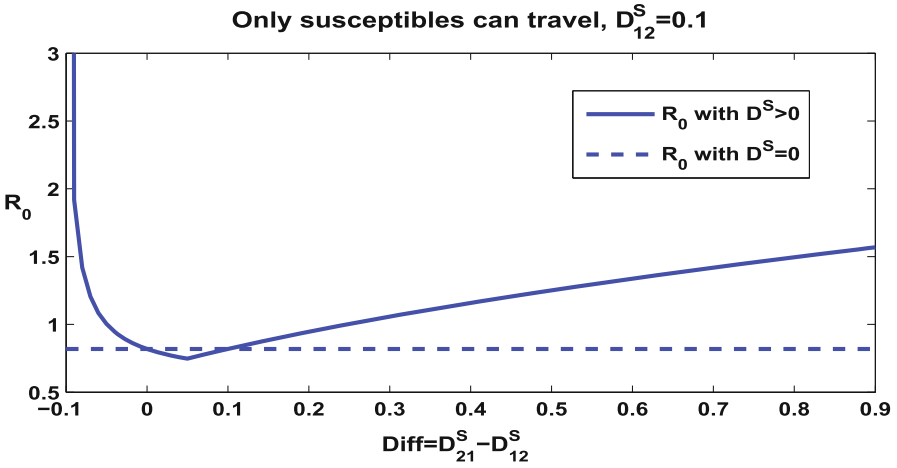

Fig. 3 Dependence of $\mathcal{R}_{02}$ on diff = $D_{12}^S - D_{21}^S$ when D_{21}^S is fixed at 0.1

more visually see the dependence of $\mathcal{R}_{02}$ on the other dispersion rate. To illustrate, we further fix $D_{21}^S = 0.1$. The dependence of $\mathcal{R}_{02}$ on $D_{12}^S - D_{21}^S = D_{12}^S - 0.1$ is plotted in Fig. 3.

6.2 Only susceptible and latent individuals disperse

When hosts become infectious, they usually have disease symptoms which will limit their outdoor activities as well as travels. However, latent hosts generally do not know that they have been infected, and therefore, under these circumstances, both susceptible and latent individuals behave the same, including disperse between patches, Corresponding to such a scenario are the assumptions that $D^I = 0$ but $D^S \neq 0$ and $D^L \neq 0$. From (6.4), we still have $r_2 = r_3 = 0$ but $s_2 \neq 0$ and $s_3 \neq 0$ in this case, and by (6.3), the GBRN, denoting by $\mathcal{R}_{03}$ now, simplifies to

$$\mathcal{R}_{03} = \sqrt{\frac{(r_4 s_4 + r_1 s_1) + \sqrt{(r_4 s_4 - r_1 s_1)^2 + 4 r_4 r_1 s_3 s_2}}{2}} \tag{6.10}$$

which can be further expressed in terms of $\mathcal{R}_{12}$ and $\mathcal{R}_{22}$ as

$$\mathcal{R}_{03} = \sqrt{\frac{[\mathcal{R}_{12}]^2 \eta_1 + [\mathcal{R}_{22}]^2 \eta_2 + \sqrt{Z}}{2}} \tag{6.11}$$

where Z now becomes

$$\begin{cases} Z = ([\mathcal{R}_{12}]^2 \eta_1 - [\mathcal{R}_{22}]^2 \eta_2)^2 + 4\,[\mathcal{R}_{12}\,\mathcal{R}_{22}]^2\,\eta_1\,\eta_2 \frac{p_{12}(\tau_1) p_{21}(\tau_1)}{p_{11}(\tau_1) p_{22}(\tau_1)}, \\ \eta_i = p_{ii}(\tau_1) \mathrm{e}^{d_{i1}\tau_1}, \quad i = 1, 2. \end{cases} \tag{6.12}$$

By dropping the last term in the formula for Z, we see that in general,

$$\mathcal{R}_{03} \geq \max\{\mathcal{R}_{12}\eta_1, \mathcal{R}_{22}\eta_2\}. \tag{6.13}$$

If the dispersal of latent hosts is unidirectional, the last term in the formula for Z vanishes and the above inequality becomes an equality. For example, if $D_{21}^L = 0$, then $p_{21}(\tau_1) = 0$, $\eta_1 = 1$, $\eta_2 = e^{-D_{12}\tau_1}$ and hence, (6.11) and (6.12) yield

$$\mathcal{R}_{03} = \max\{\mathcal{R}_{12}\sqrt{\eta_1}, \mathcal{R}_{22}\sqrt{\eta_2}\} = \max\{\mathcal{R}_{12}, \mathcal{R}_{22}e^{-D_{12}\tau_1/2}\} \leq \max\{\mathcal{R}_{12}, \mathcal{R}_{22}\} = \mathcal{R}_{02}. \tag{6.14}$$

Unidirectional travels become possible when different patches (e.g., countries) have different public health systems, or the health officials in different patches disagree on severity of a disease, resulting in different restrictions on travels.

One may obtain some information on how D^L affect the GBRN by further exploring (6.11)–(6.12) numerically. For example, if we fix the parameters as below (Chitnis et al. 2008),

$$\begin{cases} K_{11} = K_{21} = 0.0926 \text{ humans} \cdot \text{day}^{-1}, \\ M_1 = 6000 \text{ mosquitoes}, \quad M_2 = 6000 \text{ mosquitoes}, \\ d_{11} = 0.0014 \text{ day}^{-1}, \quad d_{12} = 0.13 \text{ day}^{-1}, \quad d_{21} = 0.0014 \text{ day}^{-1}, \quad d_{22} = 0.13 \text{ day}^{-1}, \\ a_1 = 0.5 \text{ day}^{-1}, \quad a_2 = 0.2 \text{ day}^{-1}, \\ e_{11} = 0.45, \quad e_{12} = 0.022, \quad e_{21} = 0.24, \quad e_{22} = 0.022, \\ \gamma_1 = 0.017 \text{ day}^{-1}, \quad \gamma_2 = 0.01 \text{ day}^{-1}, \quad \tau_1 = 20 \text{ day}, \quad \tau_2 = 10 \text{ day}, \\ \mu_1 = 9e-5 \text{ humans} \cdot \text{day}^{-1}, \quad \mu_2 = 1.8e-5 \text{ humans} \cdot \text{day}^{-1} \end{cases} \tag{6.15}$$

then computations of (6.11) and (6.12) show the impact of D_{12}^L and D_{21}^L on $\mathcal{R}_0^3$ as in Fig. 4. Cross sections of Fig. 4 at $D_{12}^L = 0,\ 0.1,\ 0.57,\ 0.9$ are given in Fig. 5; cross sections of Fig. 4 at $D_{21}^L = 0,\ 0.1,\ 0.57,\ 0.9$ are shown in Fig. 6.

From Figs. 4, 5 and 6, it is interesting to notice that the impact of the dispersions of latent class on the GBRN would be different from that of the susceptible class: while unbalanced dispersal rates of susceptible hosts always enhance the survival of the disease by increasing the GBRN, it is possible that unbalanced dispersions of latent class will *increase* the GBRN and it is also possible that unbalanced dispersions of latent class will *decrease* the GBRN.

7 Conclusion and discussion

We have derived a system of delay differential equations to describe the transmission dynamics of general vector-borne diseases in a patchy environment (with malaria being a prototype) in which the development latencies of parasites or virus (parasitic or viral vector-borne diseases: malaria protozoans or dengue virus) within both hosts and vectors, as well as the travels of hosts between patches are incorporated. Our model only applies to large scale patchy environments in which vectors cannot disperse the distances between the patches. This is in contrast to the patch models in

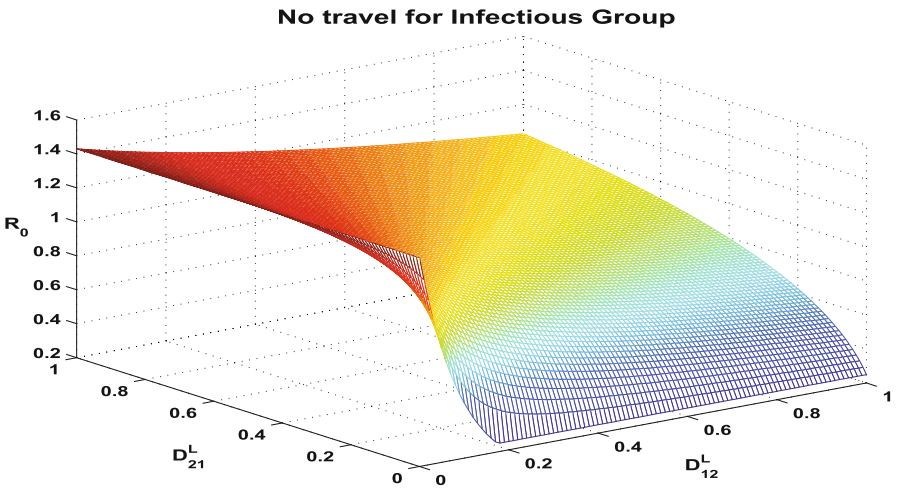

Fig. 4 Dependence of $\mathcal{R}_{03}$ on D_{12}^L and D_{21}^L when other parameters are fixed by (6.15)

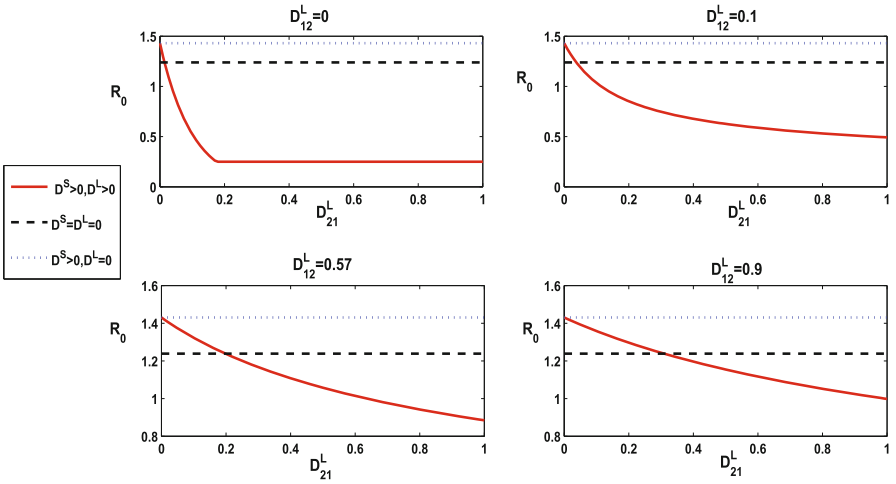

Fig. 5 Cross sections of 4 at $D_{12}^L = 0,\ 0.1,\ 0.57,\ 0.9$

Arino et al. (2011), Auger et al. (2008), Cosner et al. (2009) and Gao and Ruan (2012) where vectors (mosquitoes) can also disperse between the patches. The co-existence of development latency in hosts and the travels of hosts between patches results in the so-called non-local infections, meaning that infectious hosts in a patch come not only from the same patch but also from all the other patches.

For this structured disease model, which is an infinite dimensional system, we have applied the theory of the next generation operator to explicitly compute explicitly global basic reproduction number (GBRN). We have shown that a small scale disease invasion will be unsuccessful if $\mathcal{R}_0 < 1$ in the sense that the disease free equilibrium is asymptotically stable. If $\mathcal{R}_0 > 1$, by applying the persistence theory (Thieme 1993;

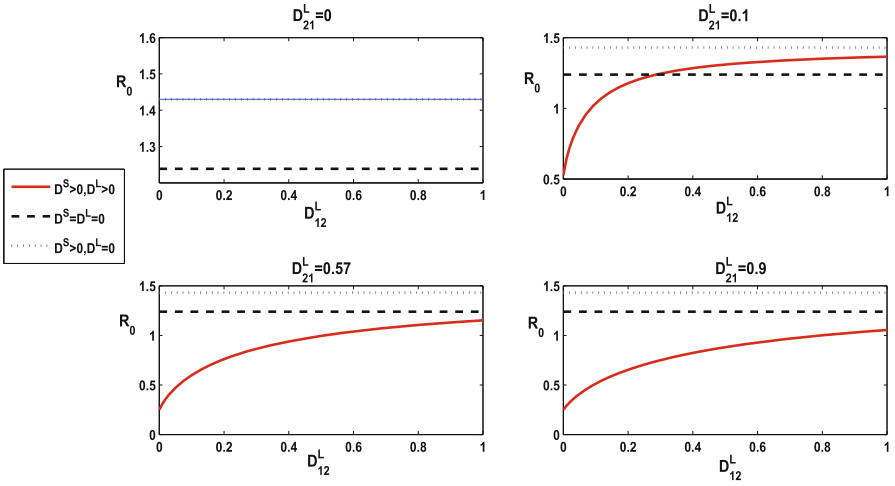

Fig. 6 Cross sections of 4 at $D_{21}^L = 0,\ 0.1,\ 0.57,\ 0.9$

Zhao 1995), we have shown that the disease will persist uniformly in all patches. In the special case of two patches, we are able to obtain an explicit formula for the $\mathcal{R}_0$ by which we can explore the impact of the dispersal rates of susceptible and latent hosts in various situations. For example, we have observed numerically that the impact of the dispersal of susceptible hosts on the GBRN can differ from that of the latent hosts in that the former always tends to increase the GBRN, while the latter may also, in addition to increasing the GBRN, decrease the GBRN. Take (6.14) as an example which is obtained under the assumption $D_{21}^L = 0$. Now if $\mathcal{R}_{12} < 1 < \mathcal{R}_{22}$, then (6.14) implies that small D_{12}^L will keep $\mathcal{R}_{03} > 1$ but large D_{12}^L will lead to $\mathcal{R}_{03} < 1$. Although more information may be obtained by more detailed and careful analysis of the formula for the GBRN, we decide not to proceed further along this line in this already lengthy paper. We point out that such information on the impacts of hosts' travels between patches on disease epidemics is useful to the health organizations of various levels for setting guidelines or making policies for travels in the context of vector-borne disease control. For instance, our result in Sect. 6.1, visualized in Figs. 2 and 3, seems to suggest that unbalanced travels of susceptible host between patches should be avoided because the GBRN attains its minimum at the balanced travel pattern.

When $\mathcal{R}_0 > 1$, the uniform persistence of a vector-borne disease in all patches also implies the existence of a global endemic equilibrium (GEE), which is an equilibrium with all components positive. The stability of this GEE, as in most disease models, is a mathematically challenging problem, and we leave it for a future project.

We have assumed that in different patches, the latent periods are constants and identical for all hosts and vectors, respectively. It is biologically more reasonable to consider different and non-constant latent periods for both hosts and vectors in different patches, due to the variations of climate and geographic conditions. Interested readers may follow the framework of this paper to generalize the model to include such cases. The theory of the next generation operator is still applicable in such a generalization,

but the similar probability matrix $P(\tau)$ accounting the non-local infections due to dispersions of hosts in the latent period is much more complicated.

We conclude the paper by pointing out an observation revealed by this work which is specific for vector-borne diseases, compared with patch models for directly transmitted diseases. To better explain the comparison, let us choose Arino et al. (2005) where a patch model is proposed and studied for a direct transmission disease with multiple strains. When there is only one strain, in the special case of isotropic mobility for all compartments and assuming that all parameters are equal in all patches, then the model behavior reduces to that of a one-strain epidemic model *without spatial dynamics*, meaning that all patches can be considered as a single patch. Same conclusion holds for the patch model with non-local infections for a direct transmission disease discussed in Li and Zou (2010). However, in our model, even in the case of isotropic mobility for all compartments and assuming that all parameters are equal are all patches, spatial dynamics is still possible. This can be clearly demonstrated by the dependence of the GBRN on the vector population sizes M_i, $i = 1, 2$, in (6.4) and (6.3), concluding that the sizes of the vector population in each patch also play a role.

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