Mathematical Biosciences 281 (2016) 128–138

Contents lists available at ScienceDirect

Mathematical Biosciences

journal homepage: www.elsevier.com/locate/mbs

# Vector-borne diseases models with residence times – A Lagrangian perspective

Derdei Bichara[a,b,*], Carlos Castillo-Chavez[c]

[a] Department of Mathematics, California State University, Fullerton, United States
[b] Center for Computational and Applied Mathematics, 800 N. State College Blvd, Fullerton, CA 92831, United States
[c] Simon A. Levin Mathematical, Computational and Modeling Science Center, Arizona State University, Tempe, AZ 85287, United States

ARTICLE INFO

*Article history:*
Received 23 September 2015
Revised 6 September 2016
Accepted 6 September 2016
Available online 10 September 2016

*MSC:*
92D30
34D23
34A34
34C12

*Keywords:*
Vector-borne diseases
Human dispersal
Basic reproduction number
Global stability
Nonlinear dynamical systems

ABSTRACT

A multi-patch and multi-group modeling framework describing the dynamics of a class of diseases driven by the interactions between vectors and hosts structured by groups is formulated. Hosts' dispersal is modeled in terms of patch-residence times with the nonlinear dynamics taking into account the *effective* patch-host size. The residence times basic reproduction number $\mathcal{R}_0$ is computed and shown to depend on the relative environmental risk of infection. The model is robust, that is, the disease free equilibrium is globally asymptotically stable (GAS) if $\mathcal{R}_0 \leq 1$ and a unique interior endemic equilibrium is shown to exist that is GAS whenever $\mathcal{R}_0 > 1$ whenever the configuration of host-vector interactions is irreducible. The effects of *patchiness* and *groupness*, a measure of host-vector heterogeneous structure, on the basic reproduction number $\mathcal{R}_0$, are explored. Numerical simulations are carried out to highlight the effects of residence times on disease prevalence.

© 2016 Elsevier Inc. All rights reserved.

## 1. Introduction

Vector-borne diseases, a major public health problem around the world, are responsible for over one million death and hundreds of millions cases each year [51,65] and so diminishing their impact is a worldwide priority. Travel, climate change and trade have significantly altered vector-borne diseases dynamics [10,26,38,52,53]. Ross [56] was the first to model a vector borne disease dynamics. Ross's paper [56] and follow up work [57–59] laid the foundation of what is known today as the field of mathematical or theoretical epidemiology. There is an extensive literature associated with the study of vector-host interactions in the context of human diseases ([2,4,6,14–16,20–24,31,40,41,43,44] and the references therein). Sparse theoretical results exist on the role of geographical heterogeneity on the spread of vector-borne diseases, mostly via metapopulation models [1,3,5,18,28,54,61,66,69], that assume that the movement of host is "permanent"; this approach has been referred as Eulerian [30,47,48]. A Lagrangian perspective considers the movement of individuals across patches in a framework where the hosts' origin or identity are never lost. This approach, useful in the study of the role of movement of individuals in highly connected settings albeit it has received limited attention [18,25,34,54,60].

The concept of Langragian and Eulerian approaches were implemented by Okubo et al. [47,48] in modeling the diffusion and aggregation of animal populations in ecology. This nomenclature has been used in the context of epidemic models by Cosner et al. [18]. The use of a Lagrangian approach in the study of the dynamics and control of vector-borne diseases has also been explored in [25,31] prior this work. Specifically, Dye and Hasibeder [25,31] considered the study of vector-born dynamics via $SIS - SI$ type host-vector models in the context of $n$ patch systems. Rodriguez and Torres-Sorando [54] used a Lagrangian perspective via the incorporation of short-time visitations to multiple patches, also in the context of vector borne disease. In [60], authors also considered a patchy Ross–Macdonald model and derived patch specific basic reproduction number in order to identify which patch is a source or a sink. More recently, Iggidr et al. [34] introduced a general $SIR - SI$ multi group deriving necessary and sufficient conditions for the existence of a sharp threshold [34]. Their [34]

* Corresponding author.
*E-mail addresses:* dbichara@fullerton.edu, bichara112@gmail.com (D. Bichara), ccchavez@asu.edu (C. Castillo-Chavez).

http://dx.doi.org/10.1016/j.mbs.2016.09.006
0025-5564/© 2016 Elsevier Inc. All rights reserved.

abstract setting did not incorporate residence times explicitly, albeit their general infection terms technically may allow for their inclusion. The study in Iggidr et al. [34] and related papers, with the exception of [25,31], assume that hosts and vectors are residents or members of particular patch or group. Our framework can handle multiple levels of organization including the host's age or socio-economic structure (see [42,64] for the age factors and [8,37,49] for the socio-economics' role). Since vector transmission is often determined by the vectors' place of residence, it is often useful to decouple the host's structure from that of vectors' population whenever possible.

In this paper, we consider a vector-host model where the host population is structured by groups/classes that interact with non-mobile vectors living in multiple patches/environments. The hosts' groups may be defined by socioeconomic background, gender, or age. The vectors' patches represent the vectors' "space", which include schools, farms, workplaces etc. Hosts, in general, will distribute their time in a multitude of vectors' places of residence (patches). In our setup, we assume that the spatial scale under consideration is such that ignoring vector mobility across patches is acceptable. There are evidences that such an assumption is reasonable, for example, Dengue and Chikungunya's urban vectors *Aedes aegypti* rarely travel more than a few tens of meters during their lifespan [1,50]; the mainly rural but urban adapted vector *Aedes albopictus* have maximum dispersal of 400–600 m [33,45]; according to [9,45], the vectors *Aedes albopictus* are unlikely to travel long distance due to wind speed variability, in fact, they exhibit a tendency to fly closer to the ground, desisting to fly during heavy winds; the adult *Anopheles*( vector of malaria) does not fly more than 2 km [63]; and, *Anopheles gambiae*'s (the main malaria vector in Africa) maximal flight distance is 10 km [36]. In short, the spread of vector-borne diseases, in many instances, is primarily due to hosts' dispersal. Therefore, it is assumed here as in [5,69] that vectors do not abandon their geographical environment or patch. There are alternative modes of mosquitoes dispersal like those generated by trade, including the used-tires' trade [46,55].

The host population is structured into $n$ groups with dispersal modeled via the residence times matrix $\mathbb{P} = (p_{ij})_{\substack{1\le i\le n,\\ 1\le j\le m}}$, where $p_{ij}$ denotes the proportion of time that a host member of Groups $i$ spends in Patch $j$. The use of this approach impacts the temporal dynamics of the *effective* host population size in each patch. Host *effective* population size *per* patch, that is the number of hosts of each group at time $t$ in Patch $j$, $j = 1, 2, \ldots, m$; is computed using the entries of the matrix $\mathbb{P}$ as weights. The density of *effective* infected host per patch account for both *effective* population and *effective* infected population size in each patch.

The host *effective* population size has not been incorporated in the literature using a Lagrangian approach in the context of vector-borne diseases before [18,54] (but see [11]). Our formulation generalize the case where vectors and hosts are defined by jointly inhabited patches [18,34,54]. We prove that the disease free equilibrium is GAS if $\mathcal{R}_0 \le 1$ and that a unique endemic equilibrium exists and is GAS if $\mathcal{R}_0 > 1$ whenever the multi-patch, multi-group system is irreducible. This approach has been used in the study of a general *SIS* model in the context of communicable diseases [7].

The paper is organized as follow. Section 2 is devoted to the derivation and basic properties of the model; Section 3 deals with the stability analysis of the disease free equilibrium (DFE) and the endemic equilibrium. Section 4, highlights the role of heterogeneity in term of patch and group variability on the basic reproduction number; Section 5 highlights tour results in the context of 2 groups, 2 patches and 2 groups and 3 patches via simulations. Section 6 collects our conclusions and thoughts on the usefulness of this approach and list possible extensions.

## 2. Derivation of the model

We consider the dynamics of human-vector interactions within a population composed of $n$ social groups and $m$ environments or patches. We denote by $N_{h,i}$ the host population in social group $i$, $i = 1, \ldots, i$, and $N_{v,j}$ vector population in Patch $j$, $j = 1, \ldots, m$. The susceptible and infected host populations in group $i$, $i = 1, \ldots, n$ , at time $t$, are denoted by $S_{h,i}(t)$ and $I_{h,i}(t)$, respectively. It is assumed that the total host population in each group is constant, that is $N_{h,i} = S_{h,i}(t) + I_{h,i}(t)$; that the disease in the host is captured by an *SIS* epidemic model while the vectors' dynamics follows an *SI* framework. The vector population in each patch is composed by $S_{v,j}$ and $I_{v,j}$, the susceptible and infected vector populations in Patch $j$, $j = 1, \ldots, m$, respectively.

The entries of the residence times matrix $\mathbb{P}$ denote the proportion of time that individuals of different groups spend in each patches; specifically $p_{ij}$ represents the proportion of time that members of group $i$ spend in Patch $j$ ($p_{ij} \ge 0$ for all $j$ and $\sum_{j=1}^{m} p_{ij} = 1$ for all $i$). The susceptible individuals of group $i$ ($S_{h,i}$) are generated through birth at the per-capita rate $\mu_i$ and they recover from infection at the per-capita rate $\gamma_i$. It is assumed that all offsprings are susceptible and that the disease does not confer immunity. The birth of susceptible individuals in group $i$ is compensated by deaths, maintaining constant host population size in each group. The host population is at risk of infection in every patches from its interaction with local infected vectors ($I_{v,j}$, $j = 1, \ldots, m$). Hence, the dynamics of the the susceptible host of group $i$, for $i = 1, \ldots, n$, is given by:

$$\dot{S}_{h,i} = \mu_i N_{h,i} + \gamma_i I_{h,i} - \sum_{j=1}^{m} b_j(N_h, N_{v,j})\beta_{v,h} p_{ij} S_{h,i} \frac{I_{v,j}}{N_{v,j}} - \mu_i S_{h,i}$$

where $b_j(N_h, N_{v,j})$ is the number of mosquito bites per human per unit of time [13,15,27,29] in Patch $j$. $b_j(N_h, N_{v,j})$ is assumed to be a function of the number of host in Patch $j$; a population that includes visitors from other patches.

The dynamics of infected hosts of group $i$, $i = 1, \ldots, n$, is modeled as follows

$$\dot{I}_{h,i} = \sum_{j=1}^{m} b_j(N_h, N_{v,j})\beta_{vh} p_{ij} S_{h,i} \frac{I_{v,j}}{N_{v,j}} - (\mu_i + \gamma_i) I_{h,i} \tag{1}$$

The susceptible vectors in Patch $j$ are replenished via constant recruitment $\Lambda_{v,j}$, subject to death at the per-capita rate $\mu_v$ and removed (through harvesting and spraying) at the per-capita rate $\delta_j$. We suppose that the natural per-capita vectors' death rates are the same in all patches. Though, the vectors do not move across patches, the susceptible mosquitoes in Patch $j$ ($S_{v,j}$) may, of course, be infected by infected hosts of any group while visiting Patch $j$. The *effective* proportion of infected individuals in Patch $j$ is therefore given by

$$\frac{\sum_{i=1}^{n} p_{ij} I_{h,i}}{\sum_{k=1}^{n} p_{kj} N_{h,k}}$$

Hence, the dynamics of susceptible vector in Patch $j$, $j = 1, \ldots, m$ in patch $j$ at time $t$ is given by

$$\dot{S}_{v,j} = \Lambda_{v,i} - a_j \beta_{hv} S_{v,j} \frac{\sum_{i=1}^{n} p_{ij} I_{h,i}}{\sum_{k=1}^{n} p_{kj} N_{h,k}} - (\mu_v + \delta_j) S_{v,j}$$

where $a_j$ is the number of bites per mosquito per unit of time in Patch $j$, assumed to be constant.

The dynamics of infected vectors in Patch $j$ is given by

$$\dot{I}_{v,j} = a_j \beta_{hv} S_{v,j} \frac{\sum_{i=1}^{n} p_{ij} I_{h,i}}{\sum_{k=1}^{n} p_{kj} N_{h,k}} - (\mu_v + \delta_j) I_{v,j} \tag{2}$$

We know that the total number of bites by mosquitoes ($a_j N_{v,j}$ in Patch $j$) should equal the total number of bites received by

**Table 1**
Description of the parameters used in System (4).

| Parameters | Description |
|---|---|
| $\beta_{vh}$ | Infectiousness of human to mosquitoes per biting |
| $\beta_{hv}$ | Infectiousness of mosquitoes to humans per biting |
| $a_j$ | Biting rate in Patch $j$ |
| $\mu_i$ | *Per capita* humans' birth and death rate for Group $i$ |
| $\gamma_i$ | *Per capita* Recovery rate for Group $i$ |
| $p_{ij}$ | Proportion of time individual in Group $i$ spend in Patch $j$ |
| $\mu_v$ | *Per capita* natural death rate of mosquitoes |
| $\delta_j$ | *Per capita* death rate of control of mosquitoes in Patch $j$ |

humans ($b_j(N_h, N_v)\sum_{k=1}^{n} p_{kj}N_{h,k}$) [2,13,44]. In our case, this conservation of contact rates should be satisfied in each patch. Hence, for Patch $j$, we have:

$$a_j N_{v,j} = b_j(N_h, N_{v,j})\sum_{k=1}^{n} p_{kj}N_{h,k}.$$

This implies that:

$$b_j(N_h, N_{v,j}) = \frac{a_j N_{v,j}}{\sum_{k=1}^{n} p_{kj}N_{h,k}} \tag{3}$$

Hence, the disease dynamics for $n$ host groups interacting in $m$ different environments subjected to resident vectors is completely described by the following system:

$$\begin{cases} \dot{S}_{h,i} = \mu_i N_{h,i} + \gamma_i I_{h,i} - \beta_{vh} S_{h,i}\sum_{j=1}^{m} a_j p_{ij}\dfrac{I_{v,j}}{\sum_{k=1}^{n} p_{kj}N_{h,k}} - \mu_i S_{h,i} \\ \dot{I}_{h,i} = \beta_{vh} S_{h,i}\sum_{j=1}^{m} a_j p_{ij}\dfrac{I_{v,j}}{\sum_{k=1}^{n} p_{kj}N_{h,k}} - (\mu_i+\gamma_i) I_{h,i} \\ \dot{S}_{v,j} = \Lambda_{v,j} - a_j\beta_{hv} S_{v,j}\dfrac{\sum_{i=1}^{n} p_{ij}I_{h,i}}{\sum_{k=1}^{n} p_{kj}N_{h,k}} - (\mu_v+\delta_j) S_{v,j} \\ \dot{I}_{v,j} = \beta_{hv} S_{v,j}\dfrac{\sum_{i=1}^{n} p_{ij}I_{h,i}}{\sum_{k=1}^{n} p_{kj}N_{h,k}} - (\mu_v+\delta_j) I_{v,j}, \end{cases} \tag{4}$$

with $i = 1, \ldots, n$ and $j = 1, \ldots, m$. The parameters used in Model (4) are defined in Table 1 and the flow diagram of the model is provided in Fig. 1.

The total host population is constant and since total vector population dynamics are given by

$$\dot{N}_{v,j} = \Lambda_{v,j} - (\mu_v + \delta_j)N_{v,j},$$

we can deduce that

$$\limsup_{t\to+\infty} N_{v,j} = \frac{\Lambda_{v,j}}{\mu_v+\delta_j} := \bar{N}_{v,j}$$

And so, the vector population is asymptotically constant in each patch. The use of the asymptotic theory on triangular systems [12,68], applied to System (4), leads to the following equivalent autonomous system:

$$\begin{cases} \dot{I}_{h,i} = \beta_{vh}(N_{h,i} - I_{h,i})\sum_{j=1}^{m} a_j p_{ij}\dfrac{I_{v,j}}{\sum_{k=1}^{n} p_{kj}N_{h,k}} - (\mu_i+\gamma_i) I_{h,i}, \\ \quad \forall i = 1, 2\ldots., n. \\ \dot{I}_{v,j} = a_j\beta_{hv}(\bar{N}_{v,j} - I_{v,j})\dfrac{\sum_{i=1}^{n} p_{ij}I_{h,i}}{\sum_{k=1}^{n} p_{kj}N_{h,k}} - (\mu_v+\delta_j) I_{v,j}, \\ \quad \forall j = 1, 2, \ldots, m. \end{cases} \tag{5}$$

Here, we have that human risk in Patch $j$ is defined by $a_j\beta_{vh}$ and so, Patch $j$ is riskier than Patch $l$ whenever $a_j > a_l$. The modeling framework is quite flexible. For example, the case $n = m$ covers the interactions between $n$ host groups (or classes) in $n$ patches or the case when hosts and vectors are co-residents. The case $p_{ij} = 0$ for all $j \neq i$ or $p_{ii} = 1$ when $n = m$ leads to a collection of isolated

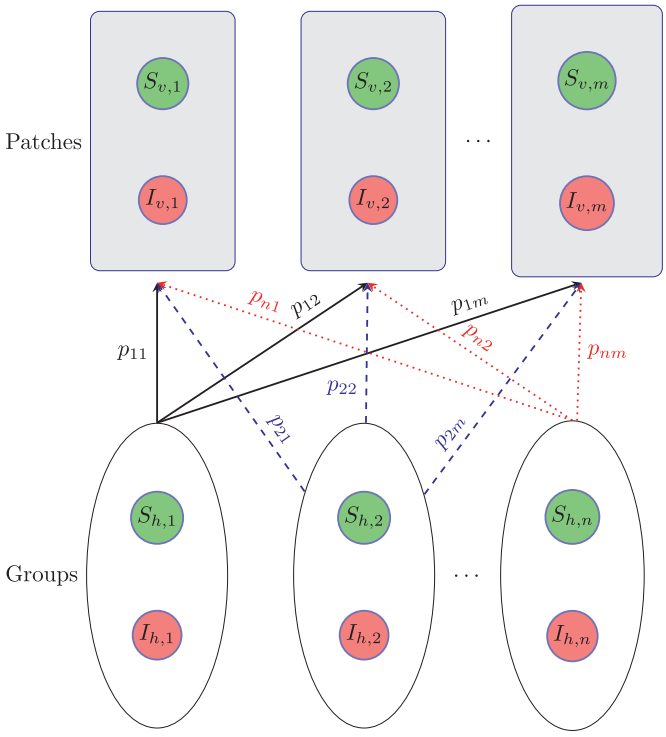



**Fig. 1.** Flow diagram of the model.

classical Ross-Macdonald models. For $n \neq m$, hosts are structured in groups like children, farmers, retired people and vectors are distributed in patches or meeting places like home, school, farm, work place or mosquito breeding site, etc. People from the considered groups visit patches and spend certain amount of time and get possibly infected whereby.

Dye and Hasibeder [25,31] models involve $n$ host and $m$ vector patches under the assumption that only the vectors move. The models in [18,54,60], have incorporated residence times explicitly but their modeling does not account for the *effective* patch population size. In fact, in [18,54], the pattern of movement between patches does not produce any "net" change on the total population per patch at any given time. For example, it is assumed (in [54]), that the total population of patch $j$ is $N/k$ where $N$ the overall human population and $k$ the number of patches. In [18,60], the total population in each patch $j$ is $N_j$ (or $H_j$ in their notations) regardless of the movement of individuals between patches. Similar remarks hold for the Dengue's two-patch model in [39]. In our case, the host population in each patch is the sum of visiting individuals of different groups *weighted by the proportion of time* they spend in each patch. This means that, at time $t$, at any given Patch $j$, the host population is $p_{1j}N_{h,1} + p_{2j}N_{h,2} + \cdots + p_{nj}N_{h,n}$, the *effective* population size of Patch $j$. Moreover, this approach is well suited for better intervention strategies through the knowledge of $a_j$, $j = 1, \ldots, m$ or the residence time matrix $\mathbb{P} = (p_{ij})_{\substack{1\le i\le n,\\ 1\le j\le m}}$. For example, if a particular host group is more affected by the disease in consideration, that may lead to the patch within which the infection had occurred, that is the patch "source" of infection. That could help steer control measures such as DDT in the "infectious" patch or social distancing the "infected" group to mitigate the disease burden.

System (5), could be written in a compact form as follows

$$\begin{cases} \dot{I}_h = \beta_{vh}\mathrm{diag}(N_h - I_h)\mathbb{P}\mathrm{diag}(a)\mathrm{diag}(\mathbb{P}^t N_h)^{-1} I_v - \mathrm{diag}(\mu+\gamma) I_h \\ \dot{I}_v = \beta_{hv}\mathrm{diag}(a)\mathrm{diag}(N_v - I_v)\mathrm{diag}(\mathbb{P}^t N_h)^{-1}\mathbb{P}^t I_h - \mathrm{diag}(\mu_v+\delta) I_v \end{cases} \tag{6}$$

where $I_h = [I_{h,1}, I_{h,2}, \ldots, I_{h,n}]^t$, $I_v = [I_{v,1}, I_{v,2}, \ldots, I_{v,m}]^t$, $N_h = [N_{h,1}, N_{h,2}, \ldots, N_{h,n}]^t$, $\tilde{N}_v = [\tilde{N}_{v,1}, \tilde{N}_{v,2}, \ldots, \tilde{N}_{v,m}]^t$, $\delta = [\delta_1, \delta_2, \ldots, \delta_m]^t$, $a = [a_1, a_2, \ldots, a_m]^t$ and $\mu = [\mu_1, \mu_2, \ldots, \mu_n]^t$.

We end this section by showing that the solutions of Model (5) are positive and bounded, or in other words, that the model is biologically grounded.

**Lemma 1.1.** *The region defined by*

$$\Omega = \left\{ (I_h, I_v) \in \mathbb{R}_+^{n+m} \;\middle|\; I_h \le N_h,\ I_v \le \tilde{N}_v \right\}$$

*is a compact attracting positively invariant set for System (6).*

**Proof.** The set $\Omega$, a subset $\mathbb{R}^{n+m}$, is clearly closed and bounded and hence a compact. The right-hand side of System (6) could be written as $A(I_h, I_v)(I_h, I_v)^t$ where

$$A(I_h, I_v) = \begin{pmatrix} -\text{diag}(\mu+\gamma) & \beta_{vh}\text{diag}(N_h - I_h)\mathbb{P}\text{diag}(a)\text{diag}(\mathbb{P}^t N_h)^{-1} \\ \beta_{hv}\text{diag}(a)\text{diag}(\tilde{N}_v - I_v)\text{diag}(\mathbb{P}^t N_h)^{-1}\mathbb{P}^t & -\text{diag}(\mu_v + \delta) \end{pmatrix}$$

Since, $I_n \le N_h$ and $I_v \le \tilde{N}_v$, the matrix $A(I_h, I_v)$ is Metzler. Hence, the positive orthant $\mathbb{R}_+^{n+m}$ is invariant. At $I_h = N_h$, we have $\dot{I}_h = -\text{diag}(\mu+\gamma)I_h \le 0$. Similarly, at $I_v = \tilde{N}_v$, we have $\dot{I}_v = -\text{diag}(\mu_v + \delta)I_v \le 0$. Hence, the vector field of (6) is pointed inward from the faces of $\Omega$. □

## 3. Equilibria and global stability

In the absence of infected vectors in all patches, Model (6) supports a unique, disease free equilibrium (DFE), given by $E_0 = \mathbf{0}_{\mathbb{R}^{n+m}}$. The basic reproduction number, defined as the average number of secondary cases produced of by an infected individual during its lifetime, is computed using the next generation method [19,67]. The right hand side of (6) could be written as $\mathcal{F} + \mathcal{V}$ where

$$\mathcal{F}(I_h, I_v) = \begin{pmatrix} \beta_{vh}\text{diag}(N_h)\mathbb{P}\text{diag}(a)\text{diag}(\mathbb{P}^t N_h)^{-1} I_v \\ \beta_{hv}\text{diag}(a)\text{diag}(N_v)\text{diag}(\mathbb{P}^t N_h)^{-1}\mathbb{P}^t I_h \end{pmatrix} \quad \text{and}$$

$$\mathcal{V}(I_h, I_v) = \begin{pmatrix} -\text{diag}(\mu+\gamma)I_h \\ -\text{diag}(\mu_v + \delta)I_v \end{pmatrix}$$

Let $F = D\mathcal{F}(I_h, I_v)$ and $V = D\mathcal{V}(I_h, I_v)$ evaluated at the DFE. We obtain:

$$F = \begin{pmatrix} 0 & \beta_{vh}\text{diag}(N_h)\mathbb{P}\text{diag}(a)\text{diag}(\mathbb{P}^t N_h)^{-1} \\ \beta_{hv}\text{diag}(a)\text{diag}(N_v)\text{diag}(\mathbb{P}^t N_h)^{-1}\mathbb{P}^t & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} -V_h & 0 \\ 0 & -V_v \end{pmatrix}$$

where $V_h = \text{diag}(\mu+\gamma)$ and $V_v = \text{diag}(\mu_v + \delta)$.

The basic reproduction number is the spectral radius of the next generation matrix:

$$-FV^{-1} = \begin{pmatrix} \mathbf{0}_{n,n} & M_{vh} \\ M_{hv} & \mathbf{0}_{m,m} \end{pmatrix}$$

where

$$M_{hv} = \beta_{hv}\text{diag}(a)\text{diag}(\mathbb{P}^t N_h)^{-1}\text{diag}(N_v)\mathbb{P}^t V_h^{-1}$$

and

$$M_{vh} = \beta_{vh}\text{diag}(N_h)\mathbb{P}\text{diag}(\mathbb{P}^t N_h)^{-1}\text{diag}(a)V_v^{-1}$$

Notice also that since $(-FV)^2 = \text{diag}(M_{vh}M_{hv}, M_{hv})$, we can deduce that the basic reproduction number is $\mathcal{R}_0^2 = \rho(M_{vh}M_{hv})$.

More precisely, we have that

$$M_{hv} = \begin{pmatrix} \frac{a_1\beta_{hv}p_{11}N_{v,1}}{\sum_{i=1}^n p_{i1}N_{h,i}(\mu_1+\gamma_1)} & \frac{a_1\beta_{hv}p_{21}N_{v,1}}{\sum_{i=1}^n p_{i1}N_{h,i}(\mu_2+\gamma_2)} & \cdots & \frac{a_1\beta_{hv}p_{n1}N_{v,1}}{\sum_{i=1}^n p_{i1}N_{h,i}(\mu_n+\gamma_n)} \\ \frac{a_2\beta_{hv}p_{12}N_{v,2}}{\sum_{i=1}^n p_{i2}N_{h,i}(\mu_1+\gamma_1)} & \frac{a_2\beta_{hv}p_{22}N_{v,2}}{\sum_{i=1}^n p_{i2}N_{h,i}(\mu_2+\gamma_2)} & \cdots & \frac{a_2\beta_{hv}p_{n2}N_{v,2}}{\sum_{i=1}^n p_{i2}N_{h,i}(\mu_n+\gamma_n)} \\ \vdots & \vdots & \cdots & \vdots \\ \frac{a_m\beta_{hv}p_{1m}N_{v,m}}{\sum_{i=1}^n p_{im}N_{h,i}(\mu_1+\gamma_1)} & \frac{a_m\beta_{hv}p_{2m}N_{v,m}}{\sum_{i=1}^n p_{im}N_{h,i}(\mu_2+\gamma_2)} & \cdots & \frac{a_m\beta_{hv}p_{nm}N_{v,m}}{\sum_{i=1}^n p_{im}N_{h,i}(\mu_n+\gamma_n)} \end{pmatrix}$$

and

$$M_{vh} = \begin{pmatrix} \frac{a_1\beta_{vh}p_{11}N_{h,1}}{\sum_{i=1}^n p_{i1}N_{h,i}(\mu_v+\delta_1)} & \frac{a_2\beta_{vh}p_{12}N_{h,1}}{\sum_{i=1}^n p_{i2}N_{h,i}(\mu_v+\delta_2)} & \cdots & \frac{a_m\beta_{hv}p_{1m}N_{h,1}}{\sum_{i=1}^n p_{im}N_{h,i}(\mu_v+\delta_m)} \\ \frac{a_1\beta_{vh}p_{21}N_{h,2}}{\sum_{i=1}^n p_{i1}N_{h,i}(\mu_v+\delta_1)} & \frac{a_2\beta_{vh}p_{22}N_{h,2}}{\sum_{i=1}^n p_{i2}N_{h,i}(\mu_v+\delta_2)} & \cdots & \frac{a_m\beta_{vh}p_{2m}N_{h,2}}{\sum_{i=1}^n p_{im}N_{h,i}(\mu_v+\delta_m)} \\ \vdots & \vdots & \cdots & \vdots \\ \frac{a_1\beta_{vh}p_{n1}N_{h,n}}{\sum_{i=1}^n p_{i1}N_{h,i}(\mu_v+\delta_1)} & \frac{a_2\beta_{vh}p_{n2}N_{h,n}}{\sum_{i=1}^n p_{i2}N_{h,i}(\mu_v+\delta_2)} & \cdots & \frac{a_m\beta_{hv}p_{nm}N_{h,n}}{\sum_{i=1}^n p_{im}N_{h,i}(\mu_v+\delta_m)} \end{pmatrix}$$

Note that the matrices $M_{hv}$ and $M_{vh}$ are of $(m, n)$ and $(n, m)$ size, respectively. The matrix $M_{vh}$ represents the new human cases due to infected mosquitoes whereas $M_{hv}$ represents the new mosquito cases due to humans. In fact, the elements of $M_{vh}$ and $M_{hv}$ have specific biological interpretations, for instance,

- For $i = 1, \ldots, n$ and $j = 1, \ldots, m$, we have $(m_{hv})_{ji} = \frac{a_j\beta_{hv}p_{ij}\tilde{N}_{v,j}}{(\mu_i+\gamma_i)\sum_{l=1}^n p_{lj}N_{h,l}}$ represents the average number of secondary vector (of Patch $j$) cases of infection produced by a single infected of group $i$ during his/her infectious period.
- Similarly, for $i = 1, \ldots, n$ and $j = 1, \ldots, m$, we have $(m_{vh})_{ij} = \frac{a_j\beta_{hv}p_{ij}N_{h,i}}{(\mu_v+\delta_j)\sum_{l=1}^n p_{lj}N_{h,l}}$ represents the average number of secondary human (of group $i$) cases generated by an infected vector (of Patch $j$) during her infectious period.
- The overall number of new cases produced by an infected mosquitoes in Patch $j$ is the sum of the elements of the $j$th column of $M_{vh}$, that is,

$$\begin{aligned} \sum_{i=1}^n (m_{vh})_{ij} &= \sum_{i=1}^n \frac{a_j\beta_{hv}p_{ij}N_{h,i}}{(\mu_v+\delta_j)\sum_{l=1}^n p_{lj}N_{h,l}} \\ &= \frac{a_j\beta_{hv}}{(\mu_v+\delta_j)\sum_{l=1}^n p_{lj}N_{h,l}} \sum_{i=1}^n p_{ij}N_{h,i} \\ &= \frac{a_j\beta_{hv}}{(\mu_v+\delta_j)}. \end{aligned}$$

The overall new vector cases generated by an infected human of group $i$ is the sum of the first column of $M_{hv}$, namely,

$$\begin{aligned} \sum_{j=1}^m (m_{hv})_{ji} &= \sum_{j=1}^m \frac{a_j\beta_{hv}p_{ij}\tilde{N}_{v,j}}{(\mu_i+\gamma_i)\sum_{l=1}^n p_{lj}N_{h,l}} \\ &= \frac{\beta_{hv}}{(\mu_i+\gamma_i)} \sum_{j=1}^m \frac{a_j p_{ij}\tilde{N}_{v,j}}{\sum_{l=1}^n p_{lj}N_{h,l}}. \end{aligned}$$

The matrix

$$\begin{pmatrix} 0 & M_{vh} \\ M_{vh} & 0 \end{pmatrix}$$

or equivalently the matrix

$$\begin{pmatrix} 0 & \mathcal{N}_{vh} \\ \mathcal{N}_{vh} & 0 \end{pmatrix}$$

where $\mathcal{N}_{vh} = \text{diag}(N_h)\mathbb{P}\text{diag}(\mathbb{P}^t N_h)^{-1}\text{diag}(a)$ and $\mathcal{N}_{hv} = \text{diag}(a)\text{diag}(\mathbb{P}^t N_h)^{-1}\text{diag}(\tilde{N}_v)\mathbb{P}^t$ is what authors in [34] called host-vector network. It is proven in [34] that the matrix $-FV^{-1}$ is irreducible if and only if $M_{hv}M_{vh}$ and $M_{vh}M_{hv}$ are both irreducible. Notice that, even if $n = m$, the irreducibility of $\mathbb{P}$ is neither necessary nor sufficient to ensure the irreducibility of $M_{hv}M_{vh}$ and $M_{vh}M_{hv}$. See the Section 5.1 for counter examples.

**Remark 2.1.** If we suppose that $n = m$ and $p_{ij} = 0$, $\forall\{i, j\} \in \{1, 2, \ldots, n\}^2$ and $i \neq j$ then $\mathbb{P} = I_n$. Hence, $M_{hv}$ and $M_{vh}$ are diagonal matrices and so is their product $M_{hv}M_{vh}$. Hence,

$$\mathcal{R}_0^2 = \max\{(\mathcal{R}_0^1)^2, (\mathcal{R}_0^2)^2, \ldots, (\mathcal{R}_0^n)^2\}$$

where

$$(\mathcal{R}_0^i)^2 = \frac{a_i^2 \beta_{vh}\beta_{hv}\tilde{N}_{v,i}}{\mu_v(\mu_i + \gamma_i)N_{h,i}}$$

For each $i$, the basic reproduction number $(\mathcal{R}_0^i)^2$ is the one derived from the classical Ross model.

**Theorem 2.1.** *Under the assumption that the host-vector network is irreducible, we have that*

1. *If $\mathcal{R}_0 \leq 1$, the DFE is globally asymptotically stable.*
2. *If $\mathcal{R}_0 > 1$, the DFE is unstable.*

**Theorem 2.2.** *Under the assumption that the vector-host network is irreducible, we have that if $\mathcal{R}_0 > 1$ then there exists a unique endemic equilibrium that is globally asymptotically stable.*

Theorems 2.1 and 3.2 can be obtained by using Smith's results [62]. Indeed, it is immediate that System (6) is cooperative, strongly concave and its Jacobian is irreducible. Hence, the theorems can be obtained following Smith's results [62] (Theorem 3.1 and Corollary 3.2). A similar method using Hirsch's theorem [32] is outlined by Iggidr et al. in [35] for an *SIS* metapopulation model.

A sharp threshold results of a multi-group vector-borne disease model has been obtained in [34] by using nicely crafted Lyapunov functions and elements of graph theory.

## 4. Effects of heterogeneity

In this section, we take a closer look to the effect of heterogeneity on the basic reproduction number and provide simple bounds for the basic reproduction number that may be useful in applications. We also compare the effects of *patchiness* (the role of variable number of patches/environments) and *groupness* (the role of variable hosts' groups) on the basic reproduction number. We denote $\mathcal{R}_0^2(m, n)$ the basic reproduction number for $n$ groups and $m$ patches.

The basic reproduction number is the spectral radius of $M_{vh}M_{hv}$. This matrix is supposed irreducible with entries are given by:

$$r_{ij} = \frac{\beta_{vh}\beta_{hv}N_{h,i}}{\mu_j + \gamma_j}\sum_{k=1}^{m}\frac{a_k^2 p_{ik}p_{jk}N_{v,k}}{(\sum_{l=1}^{n} p_{lk}N_{h,l})^2(\mu_v + \delta_k)} \quad \forall i, j = 1, \ldots, n. \tag{7}$$

$\mathbb{R} = (r_{ij})$ is an $n \times n$ matrix. The basic reproduction number is also the spectral radius of $M_{hv}M_{vh}$, a matrix with entries

$$\tilde{r}_{ij} = \frac{a_i a_j \beta_{hv}\beta_{vh}N_{v,i}}{(\mu_v + \delta_j)(\sum_{l=1}^{n} p_{li}N_{h,l})(\sum_{l=1}^{n} p_{lj}N_{h,l})}\sum_{k=1}^{n}\frac{p_{ki}p_{kj}N_{h,k}}{\mu_k + \gamma_k} \quad \forall i, j = 1, \ldots, m. \tag{8}$$

The next theorem collects a set of inequalities that identify lower and upper bounds for the basic reproduction number.

**Theorem 3.1.**

1. $\min_{j=1,\ldots,n} L_j \leq \mathcal{R}_0^2(n, m) \leq \max_{j=1,\ldots,n} L_j$ *where*

$$L_j = \frac{\beta_{hv}\beta_{hv}}{\mu_j + \gamma_j}\sum_{k=1}^{m}\frac{a_k^2 p_{jk}N_{v,k}}{(\sum_{l=1}^{n} p_{lk}N_{h,l})(\mu_v + \delta_k)}$$

2. $\min_{i=1,\ldots,n} L_i^\sharp \leq \mathcal{R}_0^2(n, m) \leq \max_{i=1,\ldots,n} L_i^\sharp$ *where*

$$L_i^\sharp = \sum_{k=1}^{m}\frac{a_k^2 \beta_{hv}\beta_{hv}p_{ik}N_{h,i}N_{v,k}}{(\sum_{l=1}^{n} p_{lk}N_{h,l})^2(\mu_v + \delta_k)}\left(\sum_{j=1}^{n}\frac{p_{jk}}{\mu_j + \gamma_j}\right)$$

3. $\min_{j=1,\ldots,m} L_j^\diamond \leq \mathcal{R}_0^2(n, m) \leq \max_{j=1,\ldots,m} L_j^\diamond$ *where*

$$L_j^\diamond = \frac{a_j \beta_{hv}\beta_{vh}}{(\mu_v + \delta_j)(\sum_{l=1}^{n} p_{lj}N_{h,l})}\sum_{k=1}^{n}\frac{p_{kj}N_{h,k}}{\mu_k + \gamma_k}\left(\sum_{i=1}^{m}\frac{a_i p_{ki}N_{v,i}}{\sum_{l=1}^{n} p_{li}N_{h,l}}\right)$$

4. $\min_{i=1,\ldots,m} L_i^\dagger \leq \mathcal{R}_0^2(n, m) \leq \max_{i=1,\ldots,m} L_i^\dagger$ *where*

$$L_i^\dagger = \frac{a_i \beta_{hv}\beta_{vh}N_{v,i}}{\sum_{l=1}^{n} p_{li}N_{h,l}}\sum_{k=1}^{n}\frac{p_{ki}N_{h,k}}{\mu_k + \gamma_k}\left(\sum_{j=1}^{m}\frac{a_j p_{kj}}{(\mu_v + \delta_j)\sum_{l=1}^{n} p_{lj}N_{h,l}}\right)$$

**Proof.**

1. Since $M_{vh}M_{hv}$ is a nonnegative irreducible matrix, the basic reproduction number $\mathcal{R}_0^2 = \rho(M_{vh}M_{hv})$ satisfy the Frobenius' inequality:

$$\min_j r_j(M_{vh}M_{hv}) \leq \mathcal{R}_0^2(n, m) \leq \max_j r_j(M_{vh}M_{hv})$$

where $r_j(M_{vh}M_{hv}) = \sum_{i=1}^{n} r_{ij}$ and $r_{ij}$ are given by (7). We have:

$$\begin{aligned} r_j(M_{vh}M_{hv}) &= \sum_{i=1}^{n} r_{ij} \\ &= \sum_{i=1}^{n}\frac{\beta_{vh}\beta_{hv}N_{h,i}}{\mu_j + \gamma_j}\sum_{k=1}^{m}\frac{a_k^2 p_{ik}p_{jk}N_{v,k}}{(\sum_{l=1}^{n} p_{lk}N_{h,l})^2(\mu_v + \delta_k)} \\ &= \frac{\beta_{vh}\beta_{hv}}{\mu_j + \gamma_j}\sum_{k=1}^{m}\frac{a_k^2 p_{jk}N_{v,k}}{(\sum_{l=1}^{n} p_{lk}N_{h,l})^2(\mu_v + \delta_k)}\sum_{i=1}^{n} p_{ik}N_{h,i} \\ &= \frac{\beta_{vh}\beta_{hv}}{\mu_j + \gamma_j}\sum_{k=1}^{m}\frac{a_k^2 p_{jk}N_{v,k}}{(\sum_{l=1}^{n} p_{lk}N_{h,l})(\mu_v + \delta_k)} \\ &:= L_j \end{aligned}$$

2. This inequality is obtained in the same way as 1 this time, by summing over the columns of $M_{vh}M_{hv}$ and using Frobenius' inequality.
3. By considering the fact that $\mathcal{R}_0^2(n, m)$ is also the spectral radius of matrix $M_{hv}M_{vh}$, the Frobenius' inequality leads to

$$\min_j \tilde{r}_j(M_{hv}M_{vh}) \leq \mathcal{R}_0^2(n, m) \leq \max_j \tilde{r}_j(M_{hv}M_{vh}),$$

where $\tilde{r}_j(M_{hv}M_{vh}) = \sum_{i=1}^{m} \tilde{r}_{ij}$ and $\tilde{r}_{ij}$ are given by (8). It follows that,

$$\begin{aligned} \tilde{r}_j(M_{hv}M_{vh}) &= \sum_{i=1}^{m} \tilde{r}_{ij} \\ &= \sum_{i=1}^{m}\frac{a_i a_j \beta_{hv}\beta_{vh}N_{v,i}}{(\mu_v + \delta_j)(\sum_{l=1}^{n} p_{li}N_{h,l})(\sum_{l=1}^{n} p_{lj}N_{h,l})} \\ &\quad \times \sum_{k=1}^{n}\frac{p_{ki}p_{kj}N_{h,k}}{\mu_k + \gamma_k} \end{aligned}$$

$$= \frac{a_j\beta_{hv}\beta_{vh}}{(\mu_v+\delta_j)(\sum_{l=1}^n p_{lj}N_{h,l})}\sum_{i=1}^m \frac{a_i p_{ki}N_{v,i}}{(\sum_{l=1}^n p_{li}N_{h,l})} \times \sum_{k=1}^n \frac{p_{kj}N_{h,k}}{\mu_k+\gamma_k}$$
$$:= L_j^{\diamond}$$

4. Let $r_i(M_{hv}M_{vh})$ denote the sum of the entries along the $i$th row of $M_{hv}M_{vh}$. We have:

$$\tilde{r}_i(M_{hv}M_{vh}) = \sum_{j=1}^m \tilde{r}_{ij} = \sum_{j=1}^m \frac{a_i a_j \beta_{hv}\beta_{vh}N_{v,i}}{(\mu_v+\delta_j)(\sum_{l=1}^n p_{li}N_{h,l})(\sum_{l=1}^n p_{lj}N_{h,l})} \times \sum_{k=1}^n \frac{p_{ki}p_{kj}N_{h,k}}{\mu_k+\gamma_k}$$
$$= \frac{a_i\beta_{hv}\beta_{vh}N_{v,i}}{(\sum_{l=1}^n p_{li}N_{h,l})}\sum_{j=1}^m \frac{a_j N_{v,i}}{(\mu_v+\delta_j)(\sum_{l=1}^n p_{lj}N_{h,l})} \times \sum_{k=1}^n \frac{p_{ki}p_{kj}N_{h,k}}{\mu_k+\gamma_k}$$
$$:= L_i^{\dagger}$$

We deduce the inequality as in 3. □

Note that the bounds $L_j$ and $L_j^{\sharp}$ can be interpreted biologically. $L_j$ is the sum of the products of the number of secondary cases of infections on mosquitoes (of Patch $k$, $k=1,\dots,m$) produced by infected host of Group $j$ ($\frac{a_k\beta_{hv}\tilde{N}_{v,k}}{\mu_j+\gamma_j}$) and secondary cases of infections on hosts (of Group $j$) produced by infected mosquitoes in Patch $k$ ($\frac{a_k\beta_{vh}}{\mu_v+\delta_k}\frac{p_{jk}}{\sum_{l=1}^n p_{lk}N_{h,l}}$) for $k=1,\dots,m$.

Similarly, $L^{\sharp}$ is the sum of the product between the number of secondary cases produced by infected mosquitoes (of Patch $k$) on hosts, that is, $\frac{a_k\beta_{hv}p_{ik}N_{h,i}}{(\sum_{l=1}^n p_{lk}N_{h,l})(\mu_v+\delta_k)}$, and the secondary mosquito cases of infection produced by infected hosts during their infectious period, that is, $\sum_{j=1}^n \frac{a_k}{\mu_j+\gamma_j}\frac{p_{jk}}{\sum_{l=1}^n p_{lk}N_{h,l}}$.

**Theorem 3.2.** *If the residence time matrix is of rank one then an explicit expression of the basic reproduction number for the general system is given by*

$$\mathcal{R}_0^2(m,n) = \frac{\beta_{vh}\beta_{hv}}{(\sum_{l=1}^n p_l N_{h,l})^2}\sum_{k=1}^m \frac{a_k^2 N_{v,k}}{(\mu_v+\delta_k)}\left(\sum_{i=1}^n \frac{p_i^2 N_{h,i}}{\mu_i+\gamma_i}\right)$$

**Proof.** Let us suppose that the residence time matrix $\mathbb{P}$ is of rank 1. There exist $x\in\mathbb{R}_+^n$ and $y\in\mathbb{R}_+^m$ such that $\mathbb{P}=xy^T$. Since $\mathbb{P}$ is stochastic, we can deduce that $x_i\sum_{j=1}^m y_j = 1$ for $i=1,2,\dots,n$. Therefore, $\mathbb{P}$ could be written as $\mathbb{P}=\mathbb{1}p^t$ where $p\in\mathbb{R}^m$ and $\sum_{i=1}^m p_i = 1$. Hence, the matrices $M_{vh}M_{hv}$ and $M_{hv}M_{vh}$ are also of rank one. Therefore the trace of $M_{vh}M_{hv}$ is only positive eigenvalue of $M_{vh}M_{hv}$. Hence, by using (7), we obtain:

$$\mathcal{R}_0^2(m,n) = \sum_{i=1}^n \frac{\beta_{vh}\beta_{hv}N_{h,i}}{\mu_i+\gamma_i}\sum_{k=1}^m \frac{a_k^2 p_i^2 N_{v,k}}{(\sum_{l=1}^n p_l N_{h,l})^2(\mu_v+\delta_k)} = \frac{\beta_{vh}\beta_{hv}}{(\sum_{l=1}^n p_l N_{h,l})^2}\sum_{i=1}^n \frac{p_i^2 N_{h,i}}{\mu_i+\gamma_i}\sum_{k=1}^m \frac{a_k^2 N_{v,k}}{(\mu_v+\delta_k)} = \frac{\beta_{vh}\beta_{hv}}{(\sum_{l=1}^n p_l N_{h,l})^2}\sum_{k=1}^m \frac{a_k^2 N_{v,k}}{(\mu_v+\delta_k)}\left(\sum_{i=1}^n \frac{p_i^2 N_{h,i}}{\mu_i+\gamma_i}\right)$$

□

If we assume that "virtual" dispersal does not induce any substantial change in the population of each patch, i.e $p_i N_{h,i} = N_{h,i}$ at any time and that $\mu_i=\mu$ and $\gamma_i=\gamma$ for all $i=1,2,\dots,n$, then we recover the result of Dye and Hasibeder [25,31], namely,

$$\mathcal{R}_0^2(m,n) = \mathcal{R}_0^2(m,1),$$

where $\mathcal{R}_0^2(m,1)$ is the basic reproduction number corresponding of $m$ patches of vectors and a single host group. Similarly, if $\delta_j=\delta$ and $a_j=a$ for all $j=1,2,\dots,m$, we obtain

$$\mathcal{R}_0^2(m,n) = \mathcal{R}_0^2(1,n),$$

where $\mathcal{R}_0^2(1,n)$ is the basic reproduction number corresponding of $n$ host groups and a single patch of vectors.

For $m$ patches and one group, the basic reproduction number is given by

$$\mathcal{R}_0^2(m,1) = \frac{\beta_{vh}\beta_{hv}}{(\mu+\gamma)N_h}\sum_{k=1}^m \frac{a_k^2 p_{1k}^2 N_{v,k}}{(\mu_v+\delta_k)}.$$

The basic reproduction number associated with single group and single environment turns out to be the classical $\mathcal{R}_0^2$, that is, $\mathcal{R}_0^2(1,1) = \frac{a^2\beta_{vh}\beta_{hv}}{(\mu+\gamma)(\mu_v+\delta)}\frac{N_v}{N_h}$. We arrive at the following result:

**Lemma 3.1.** *We have*

$$\mathcal{R}_0^2(m,1) \geq \mathcal{R}_0^2(1,1)$$

**Proof.**

$$\mathcal{R}_0^2(m,1) = \frac{\beta_{vh}\beta_{hv}}{(\mu_+\gamma_1)N_h}\sum_{k=1}^m \frac{a_k^2 p_{1k}^2 \bar{N}_{v,k}}{(\mu_v+\delta_k)} \geq \frac{\beta_{vh}\beta_{hv}}{(\mu_+\gamma_1)N_h}\frac{a_k^2 p_{11}^2 \bar{N}_{v,1}}{(\mu_v+\delta_1)} := \mathcal{R}_0^2(1,1) \quad (9)$$

since $p_{11}=1$ for a single patch and single host. □

**Remark 3.1.** In Lemma 3.1, we are comparing the basic reproduction number of the $m$ patches and 1 group case with the one of 1 patch and 1 group case. And so, the $p_{11}$ in the RHS of the inequality in the proof of Lemma 3.1, is seen both as the $p_{11}$ of the single patch, single group case and the $m$ patches, single group case.

Lemma 3.1 states that, for a single group, the presence of patch/environmental heterogeneity might increase the basic reproduction number.

## 5. Special cases and simulations

In this section, we provide examples of cases where the number of patches and number of groups are either equal or non-equal, that is, we highlight in a limited way the role of *patchiness* and *groupness*. We start off by the case of two patches and two groups to showcase that even when the residence times matrix $\mathbb{P}$ is square, its irreducibility is neither necessary nor sufficient to ensure the irreducibility of the next generation matrix. This implies that the disease either dies out or persists in all patches and groups. We then consider the three patches and two groups for which the disease persists in all groups and patches in an attempt to see how the differential in residence times leads to a differential in the disease burden for hosts and vectors.

### 5.1. The two patches and two groups case

As stated at the derivation of the model (Section 2), this system could model either the case where there are two patches within which there are hosts and vectors or it could model the case where there are two groups of hosts interacting in two different patches.

The basic reproduction number, for $n = 2$, $m = 2$ is $\rho(M_{vh}M_{hv})$, where

$$M_{hv} = \begin{pmatrix} \frac{a_1\beta_{hv}p_{11}\bar{N}_{v,1}}{(p_{11}N_{h,1}+p_{21}N_{h,2})(\mu_1+\gamma_1)} & \frac{a_1\beta_{hv}p_{21}\bar{N}_{v,1}}{(p_{11}N_{h,1}+p_{21}N_{h,2})(\mu_2+\gamma_2)} \\ \frac{a_2\beta_{hv}p_{12}\bar{N}_{v,2}}{(p_{12}N_{h,1}+p_{22}N_{h,2})(\mu_1+\gamma_1)} & \frac{a_2\beta_{hv}p_{22}\bar{N}_{v,2}}{(p_{12}N_{h,1}+p_{22}N_{h,1})(\mu_2+\gamma_2)} \end{pmatrix}$$

and

$$M_{vh} = \begin{pmatrix} \frac{a_1\beta_{vh}p_{11}N_{h,1}}{(p_{11}N_{h,1}+p_{21}N_{h,2})(\mu_v+\delta_1)} & \frac{a_2\beta_{vh}p_{12}N_{h,1}}{(p_{12}N_{h,1}+p_{22}N_{h,2})(\mu_v+\delta_2)} \\ \frac{a_1\beta_{vh}p_{21}N_{h,2}}{(p_{11}N_{h,1}+p_{21}N_{h,2})(\mu_v+\delta_1)} & \frac{a_2\beta_{vh}p_{22}N_{h,2}}{(p_{12}N_{h,1}+p_{22}N_{h,2})(\mu_v+\delta_2)} \end{pmatrix}$$

We have that

$$M_{vh}M_{hv} = \begin{pmatrix} m_{11} & m_{12} \\ m_{21} & m_{22} \end{pmatrix}$$

where

$$m_{11} = \frac{a_1^2\beta_{hv}\beta_{vh}p_{11}^2\bar{N}_{v,1}N_{h,1}}{(p_{11}N_{h,1}+p_{21}N_{h,2})^2(\mu_1+\gamma_1)(\mu_v+\delta_1)} + \frac{a_2^2\beta_{hv}\beta_{vh}p_{12}^2\bar{N}_{v,2}N_{h,1}}{(p_{12}N_{h,1}+p_{22}N_{h,2})^2(\mu_1+\gamma_1)(\mu_v+\delta_2)},$$

$$m_{12} = \frac{a_1^2\beta_{hv}\beta_{vh}p_{11}p_{21}\bar{N}_{v,1}N_{h,1}}{(p_{11}N_{h,1}+p_{21}N_{h,2})^2(\mu_2+\gamma_2)(\mu_v+\delta_1)} + \frac{a_2^2\beta_{hv}\beta_{vh}p_{12}p_{22}\bar{N}_{v,2}N_{h,1}}{(p_{12}N_{h,1}+p_{22}N_{h,2})^2(\mu_2+\gamma_2)(\mu_v+\delta_2)},$$

$$m_{21} = \frac{a_1^2\beta_{hv}\beta_{vh}p_{11}p_{21}\bar{N}_{v,1}N_{h,2}}{(p_{11}N_{h,1}+p_{21}N_{h,2})^2(\mu_1+\gamma_1)(\mu_v+\delta_1)} + \frac{a_2^2\beta_{hv}\beta_{vh}p_{12}p_{22}\bar{N}_{v,2}N_{h,2}}{(p_{12}N_{h,1}+p_{22}N_{h,2})^2(\mu_1+\gamma_1)(\mu_v+\delta_2)},$$

and

$$m_{22} = \frac{a_1^2\beta_{hv}\beta_{vh}p_{21}^2\bar{N}_{v,1}N_{h,2}}{(p_{11}N_{h,1}+p_{21}N_{h,2})^2(\mu_2+\gamma_2)(\mu_v+\delta_1)} + \frac{a_2^2\beta_{hv}\beta_{vh}p_{22}^2\bar{N}_{v,2}N_{h,2}}{(p_{12}N_{h,1}+p_{22}N_{h,2})^2(\mu_2+\gamma_2)(\mu_v+\delta_2)}$$

We observe, that even for the case $n = m$, the irreducibility of $\mathbb{P}$ is nor necessary not sufficient to ensure the irreducibility of $M_{vh}M_{hv}$ and $M_{hv}M_{vh}$. Indeed, if $p_{12} = 0$, $p_{21} > 0$ and $p_{22} > 0$, the residence time matrix is given by

$$\begin{pmatrix} 1 & 0 \\ p_{21} & p_{22} \end{pmatrix}$$

is reducible whereas

$$M_{vh}M_{hv} = \begin{pmatrix} \frac{a_1^2\beta_{hv}\beta_{vh}p_{11}^2\bar{N}_{v,1}N_{h,1}}{(p_{11}N_{h,1}+p_{21}N_{h,2})^2(\mu_1+\gamma_1)(\mu_v+\delta_1)} & \frac{a_1^2\beta_{hv}\beta_{vh}p_{11}p_{21}\bar{N}_{v,1}N_{h,1}}{(N_{h,1}+p_{21}N_{h,2})^2(\mu_2+\gamma_2)(\mu_v+\delta_1)} \\ \frac{a_1^2\beta_{hv}\beta_{vh}p_{21}\bar{N}_{v,1}N_{h,2}}{(N_{h,1}+p_{21}N_{h,2})^2(\mu_1+\gamma_1)(\mu_v+\delta_1)} & \frac{a_1^2\beta_{hv}\beta_{vh}p_{21}^2\bar{N}_{v,1}N_{h,2}}{(N_{h,1}+p_{21}N_{h,2})^2(\mu_2+\gamma_2)(\mu_v+\delta_1)} + \frac{a_2^2\beta_{hv}\beta_{vh}\bar{N}_{v,2}}{N_{h,2}(\mu_2+\gamma_2)(\mu_v+\delta_2)} \end{pmatrix}$$

is irreducible. Similarly, the residence times matrix

$$\mathbb{P} = \begin{pmatrix} 0 & 1 \\ 1 & 0 \end{pmatrix}$$

is irreducible while the non-diagonal entries of $M_{vh}M_{hv}$ are equal to zero, that is, $m_{12} = m_{21} = 0$. Hence, $M_{vh}M_{hv}$ is not irreducible. If the matrices $M_{hv}M_{vh}$ and $M_{vh}M_{hv}$ are not both irreducible, we may obtain boundary equilibria for which the disease dies out in some hosts' groups and vectors' patches while persisting in others. See Fig. 7a and b for instance.

### 5.2. The three patches and two groups case

As an illustrative example, we consider System (5) for the case $n = 2$ groups and $m = 3$ patches. The basic reproduction number is the spectral radius of

$$-FV^{-1} = \begin{pmatrix} 0 & 0 & & M_{vh} & \\ 0 & 0 & & & \\ & & 0 & 0 & 0 \\ M_{hv} & & 0 & 0 & 0 \end{pmatrix} \tag{10}$$

where

$$M_{hv} = \begin{pmatrix} \frac{a_1\beta_{hv}p_{11}\bar{N}_{v,1}}{(p_{11}N_{h,1}+p_{21}N_{h,2})(\mu_1+\gamma_1)} & \frac{a_1\beta_{hv}p_{21}\bar{N}_{v,1}}{(p_{11}N_{h,1}+p_{21}N_{h,2})(\mu_2+\gamma_2)} \\ \frac{a_2\beta_{hv}p_{12}\bar{N}_{v,2}}{(p_{12}N_{h,1}+p_{22}N_{h,2})(\mu_1+\gamma_1)} & \frac{a_2\beta_{hv}p_{22}\bar{N}_{v,2}}{(p_{12}N_{h,1}+p_{22}N_{h,1})(\mu_2+\gamma_2)} \\ \frac{a_3\beta_{hv}p_{13}\bar{N}_{v,3}}{(p_{13}N_{h,1}+p_{23}N_{h,2})(\mu_1+\gamma_1)} & \frac{a_3\beta_{hv}p_{23}\bar{N}_{v,3}}{(p_{13}N_{h,1}+p_{23}N_{h,1})(\mu_2+\gamma_2)} \end{pmatrix}$$

and

$$M_{vh} = \begin{pmatrix} \frac{a_1\beta_{vh}p_{11}N_{h,1}}{(p_{11}N_{h,1}+p_{21}N_{h,2})(\mu_v+\delta_1)} & \frac{a_2\beta_{vh}p_{12}N_{h,1}}{(p_{12}N_{h,1}+p_{22}N_{h,2})(\mu_v+\delta_2)} \\ \frac{a_1\beta_{vh}p_{21}N_{h,2}}{(p_{11}N_{h,1}+p_{21}N_{h,2})(\mu_v+\delta_1)} & \frac{a_2\beta_{vh}p_{22}N_{h,2}}{(p_{12}N_{h,1}+p_{22}N_{h,2})(\mu_v+\delta_2)} \\ \frac{a_3\beta_{vh}p_{13}N_{h,1}}{(p_{13}N_{h,1}+p_{23}N_{h,2})(\mu_v+\delta_3)} & \\ \frac{a_3\beta_{vh}p_{23}N_{h,2}}{(p_{13}N_{h,1}+p_{23}N_{h,2})(\mu_v+\delta_3)} & \end{pmatrix}$$

For purposes of simulations, we use the following baseline parameters with the ranges given in parentheses.

$$\beta_{hv} = 0.5(0.001 - 0.54), \quad \beta_{vh} = 0.41(0.3 - 0.9)$$
$$\frac{1}{\mu_v} = 20(10 - 30) \text{ days}, \frac{1}{\mu_1} = 75 \times 365 \text{ days}$$
$$a_1 = 0.5 \text{ day}^{-1}, \quad a_2 = 0.4 \text{ day}^{-1}, \quad a_3 = 0.3 \text{ day}^{-1}$$
$$\frac{1}{\mu_2} = 73 \times 365 \text{ days}, \quad \frac{1}{\gamma_1} = 7 \text{ days}, \quad \frac{1}{\gamma_2} = 6 \text{ days},$$
$$\delta_1 = 0.001 \text{ day}^{-1}, \quad \delta_2 = 0.01, \quad \delta_3 = 0.08 \text{ day}^{-1}.$$

The values of $\beta_{hv}$, $\beta_{vh}$ and $\mu_v$ are taken from [17]. The host populations and the recruitments of vectors for the 3 patches are taken as

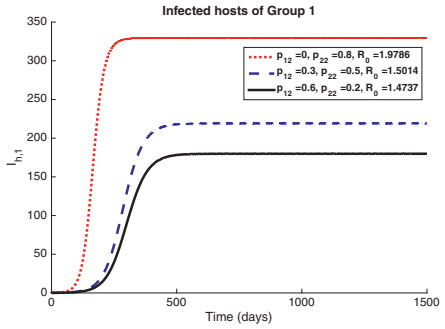
$$N_{h,1} = 4000, \quad N_{h,2} = 4500, \quad \Lambda_{v,1} = 1000,$$
$$\Lambda_{v,2} = 1000, \quad \Lambda_{v,3} = 950.$$

Unless otherwise stated, we fix $p_{13} = 0.1$ and $p_{23} = 0.2$, carrying out System (5) simulations that focus on the effects of non-fixed residence times matrix entries on the prevalence of hosts and vectors.

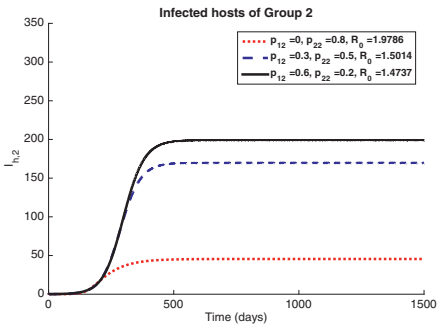
Fig. 2 displays the dynamics of infected hosts of Group 1 (Fig. 2a) and Group 2 (Fig. 2b). The level of endemicity of individuals of Group 1 seems to decrease as proportion of time in Patch 2 ($p_{12}$) increases; probably because as $p_{12}$ increases, $p_{11}$ decreases. In other words, individuals of Group 1 spend more time in the less riskier Patch 2 ($a_2 = 0.4$) than in the riskier Patch 1 ($a_1 = 0.5$). We see that the less time that individuals spend in riskier environment the less likely that they will become infected, as one would expect.

In Fig. 2b, the level of endemicity of hosts in Group 2 seems to decrease as $p_{22}$ increases or equivalently as $p_{21}$ decreases ( $p_{21} + p_{22} + p_{23} = 1$ and $p_{23}$ is fixed). It is so because individuals are increasing their residence time in Patch 2 ($a_2 = 0.4$) rather than in Patch 1.

Figs. 3 and 4 offer an overview on how the dynamics of vectors change as the proportion of time that individuals of Group 1 and

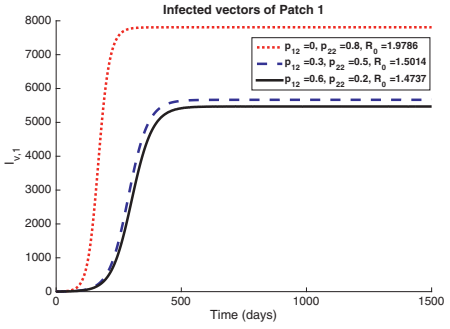



(a) The level of prevalence of host of group 1 seems to decrease as $p_{12}$ increases (and hence $p_{11}$ decreases).

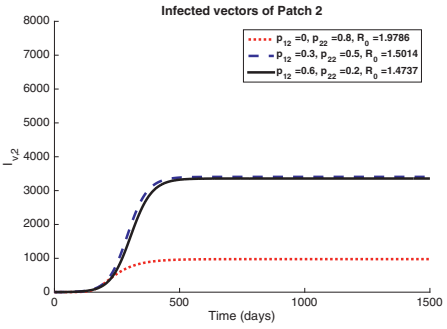



(b) The level of prevalence of host of group 2 seems to decrease with respect to $p_{22}$.

**Fig. 2.** Dynamics of $I_{h,1}$ and $I_{h,2}$ for different values of $p_{ij}$.




(a) The level of prevalence of vectors of Patch 1 is decreasing as $p_{12}$ increases and $p_{22}$ increases.




(b) The level of prevalence of host of group 2

**Fig. 3.** Dynamics of $I_{v,1}$ and $I_{v,2}$ for different values of $p_{ij}$.

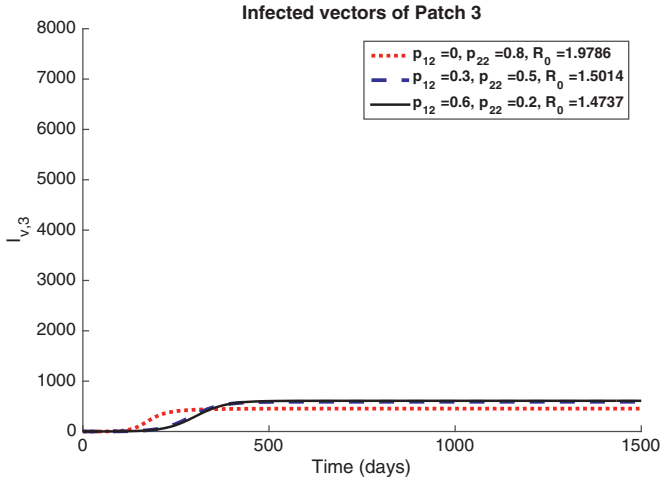



**Fig. 4.** Dynamics of vectors in Patch 3 ($I_{v,3}$) for different values of $p_{ij}$.

Group 2 spend in environments 1, 2 and 3 varies. For the selected residence times matrix entries, the prevalence of vector in environment 1 (see Fig. 3a) is at its highest if $p_{12} = 0$ and $p_{22} = 0.8$. With this configuration, $p_{11} = 0.9$ and Patch 1 has the highest *effective* population size. Moreover, Patch 1 has the highest biting rate, leading to high level of vector infections in that patch.

Though $\Lambda_{v,1} = \Lambda_{v,2}$, the prevalence of vectors Patch 2 (Fig. 3b) is lower than of Patch 1 (Fig. 3a), regardless of the combination of the chosen residence times entries. This is because the *effective* population of Patch 2 is less than the *effective* population of Patch 1 for all the three selected residence time configurations and also because $a_1 > a_2$.

Fig. 4 represent the dynamics of the vectors in Patch 3. The number of infected vector in this patch is much less when compared to the number of infected in Patches 1 and 2. Again, the *effective* population size of Patch 3, with $p_{13} = 0.1$ and $p_{23} = 0.2$, is much less when compared to those in Patches 1 and 2. Additionally, we also have by assumption that $a_3 < a_2 < a_1$.

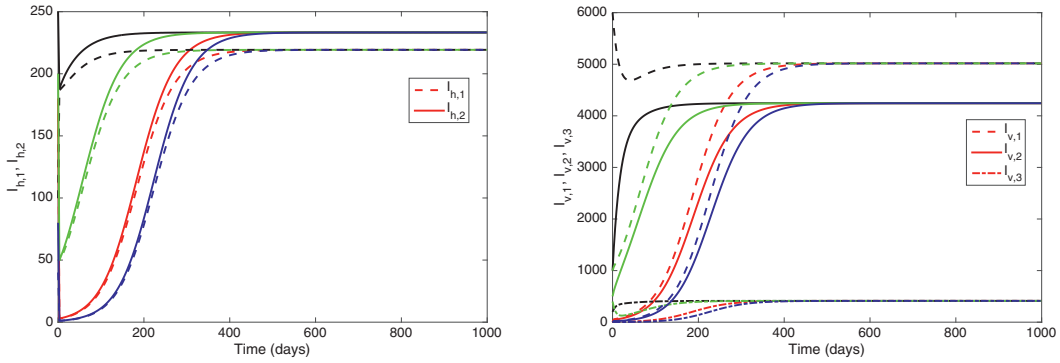
For the vectors' prevalence, we obtain similar results as in Figs. 3 and 4 even if when biting rates are equal in all the three patches. This last comment highlights the role of the *effective* population per patch.

**Remark 4.1.** For all the selected combination of the residence times matrix entries in Figs. 2–4, the matrices $M_{vh}M_{hv}$ and $M_{hv}M_{vh}$ are irreducible and $\mathcal{R}_0 > 1$ and so the curves of the infected hosts and vectors in those figures are reaching an endemic equilibrium level in accordance with Theorem 2.2.

In Figs. 5 and 6, we consider the case where the residence time matrix is fixed as follows:

$$\mathbb{P} = \begin{pmatrix} 0.4 & 0.3 & 0.3 \\ 0.4 & 0.4 & 0.2 \end{pmatrix}$$

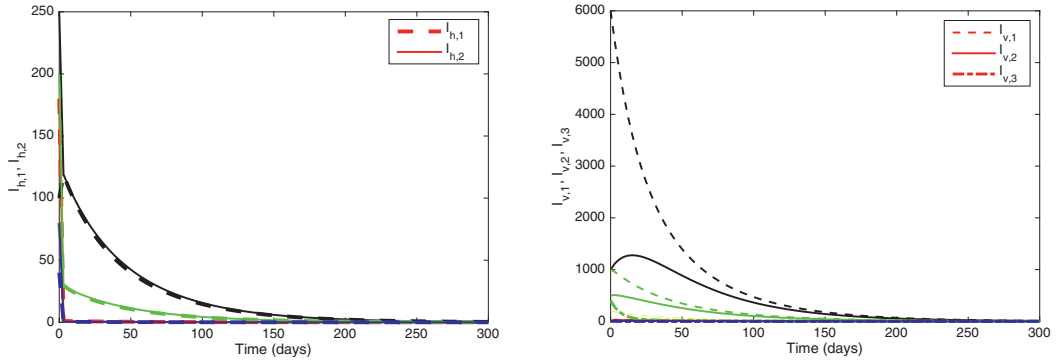
In that case, the matrices $M_{hv}M_{vh}$ and $M_{vh}M_{hv}$ are both irreducible. We sketch the trajectories of System (5) for different initial conditions. Since, with these values of parameters and residence times matrix, the basic reproduction number is $\mathcal{R}_0(3,2) = 1.4771$, and so the result of Theorem 2.2 should hold and Fig. 5 confirms that. More precisely, Fig. 5a shows that the trajectories of infected individuals of Groups 1 and 2 converge




(a) Dynamics of infected hosts of Group 1 and Group 2.

(b) Dynamics of infected vectors of Patch 1 and Patch 2.

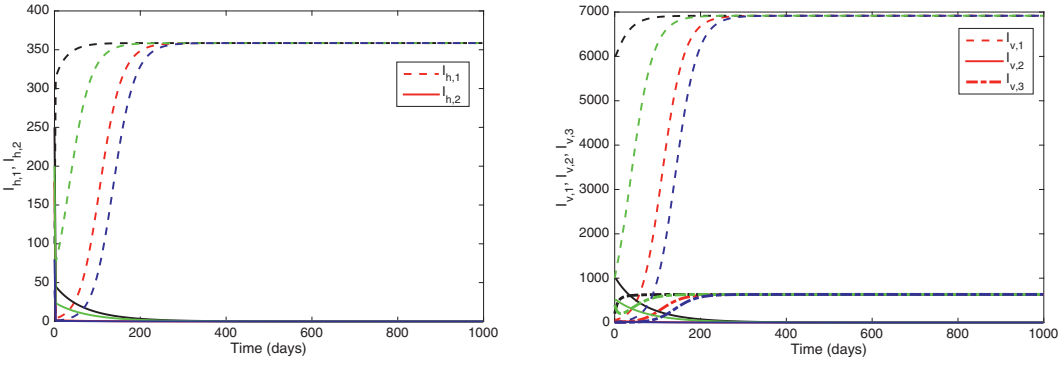
**Fig. 5.** Trajectories of System (5), with $n = 2$ groups and $m = 3$ patches with 4 different initial conditions. The trajectories are converging toward a unique interior endemic equilibrium.




(a) Dynamics of infected hosts of Group 1 and Group 2.

(b) Dynamics of infected vectors of Patch 1 and Patch 2.

**Fig. 6.** Trajectories of System (5), with $n = 2$ groups and $m = 3$ Patches with 4 different initial conditions. With $\beta_{hv} = 0.2$ and $\beta_{vh} = 0.4$, we have $\mathcal{R}_0(3, 2) = 0.6353$ and the trajectories are converging toward the disease free equilibrium.




(a) Dynamics of infected hosts of Group 1 and Group 2.

(b) Dynamics of infected vectors of Patch 1, Patch 2 and Patch 3.

**Fig. 7.** Trajectories of System (5), with $n = 2$ groups and $m = 3$ patches with 4 different initial conditions. The disease dies out for the host of Group 2 whereas it persists for those of Group 1. Similarly, the disease dies out for the vector of Patch 2 but persists for the vectors of Patches 1 and 3.

to the same interior endemic equilibrium for four different initial conditions, namely $IC_1 = [I_{h,1}(0) = 180,\ I_{h,2}(0) = 180,\ I_{v,1}(0) = 0,\ I_{v,2}(0) = 0,\ I_{v,3}(0) = 0]$ (solid red for $I_{h,1}$ and dashed red for $I_{h,2}$ in Fig. 5a), $IC_2 = [I_{h,1}(0) = 100,\ I_{h,2}(0) = 250,\ I_{v,1}(0) = 6000,\ I_{v,2}(0) = 1000,\ I_{v,3}(0) = 200]$ (solid black for $I_{h,1}$ and dotted black for $I_{h,2}$ in Fig. 5a), $IC_3 = [I_{h,1}(0) = 80,\ I_{h,2}(0) = 200,\ I_{v,1}(0) = 1000,\ I_{v,2}(0) = 500,\ I_{v,3}(0) = 400]$ (solid green for $I_{h,1}$ and dotted green for $I_{h,2}$ in Fig. 5a) and $IC_4 = [I_{h,1}(0) = 40,\ I_{h,2}(0) = 80,\ I_{v,1}(0) = 1,\ I_{v,2}(0) = 2,\ I_{v,3}(0) = 3]$ (solid blue for $I_{h,1}$ and dotted blue for $I_{h,2}$ in Fig. 5a).

Similarly, Fig. 5b displays the trajectories of infected vectors in the three considered environments. For all the above-mentioned initial conditions, these trajectories converge to their interior endemic equilibrium level.

Fig. 6 sketches the case where the values of all the parameters are the same as above but where $\beta_{hv} = 0.2$ and $\beta_{vh} = 0.4$. In

this case, the basic reproduction number is $\mathcal{R}_0 = 0.6353$ which is less than one. As we can see in Fig. 6a and b, the trajectories of infected hosts of the two groups and the infected vectors of the three patches are converging to zero for the above four initial conditions. This suggests that the DFE is globally asymptotically stable and confirms the result of Theorem 2.1.

Now, we consider the case where the configuration of the Group-Patch network is not irreducible. If we assume that $p_{12} = p_{21} = p_{23} = 0$, the residence times matrix becomes

$$\mathbb{P} = \begin{pmatrix} 0.7 & 0 & 0.3 \\ 0 & 1 & 0 \end{pmatrix}.$$

This imply that $M_{hv} = \begin{pmatrix} \frac{a_1\beta_{vh}\tilde{N}_{v,1}}{N_{h,1}(\mu_1+\gamma_1)} & 0 \\ 0 & \frac{a_2\beta_{vh}\tilde{N}_{v,2}}{N_{h,2}(\mu_2+\gamma_2)} \\ \frac{a_3\beta_{vh}\tilde{N}_{v,3}}{N_{h,1}(\mu_1+\gamma_1)} & 0 \end{pmatrix}$ and $M_{vh} = \begin{pmatrix} \frac{a_1\beta_{hv}}{\mu_v+\delta_1} & 0 & \frac{a_3\beta_{hv}}{\mu_v+\delta_3} \\ 0 & \frac{a_2\beta_{hv}}{\mu_v+\delta_2} & 0 \end{pmatrix}$. Hence, the matrices $M_{vh}M_{hv}$ and $M_{hv}M_{vh}$ are not both irreducible and hence Theorem 2.2 does not hold, as shown in Fig. 7, where a boundary equilibrium appears. In this case, members of Group 2 spend all their time in Patch 2 and hence are isolated from the rest of groups and patches. The basic reproduction number of Group 2 in Patch 2 is $(\mathcal{R}_0(2,2))^2 = \frac{a_2^2\beta_{vh}\beta_{hv}N_{v,2}}{(\mu_v+\delta_2)(\mu_2+\gamma_2)N_{h,2}} = 0.8$. The diseases dies out from the hosts of Group 2 (see Fig. 7a, solid curves) and vectors of Patch 2 (Fig. 7b, solid curves). Members of Group 1 are connected to Patch 1 and Patch 3. Hence, the corresponding basic reproduction number is $(\mathcal{R}_0^{1,1,3})^2 = \frac{a_1^2\beta_{vh}\beta_{hv}N_{v,21}}{(\mu_v+\delta_1)(\mu_1+\gamma_1)N_{h,1}} + \frac{a_3^2\beta_{vh}\beta_{hv}N_{v,3}}{(\mu_v+\delta_3)(\mu_1+\gamma_1)N_{h,1}} = 1.8549$. Hence, the disease persists among the members of Group 1(see Fig. 7a, dashed curves) and vectors of Patches 1 and 3 (see Fig. 7b, dashed curves). This case offers a glimpse on how disease dynamics when some groups are strongly connected to some environments while other groups are isolated.

## 6. Conclusion and discussion

Modeling vector-borne interactions have often been based on well-mixed models that make it difficult to address effectively the role of host mobility on vector borne disease dynamics. Here, we consider a Lagrangian framework where hosts' dispersal is modeled via the proportion of time that individuals spend in different environments. In the process, we are forced to account, for time variations in *effective* population size within each patch/environment. The kind of natural adjustment that can significantly alter the quantitative and qualitative dynamics of vector borne dynamics in geographically heterogeneous system; here within spatial scales that make it possible to neglect vector mobility.

And so, we consider a general *SIS* framework to account for the host dynamics and an *SI* framework to account for the vector dynamics. The transmission terms must make adjustments to account for the *effective* population size generated by the residence time matrix. This is handled via the use of a modified frequency-dependent incidence model that accounts for the *effective* density of infected hosts within each patch at any time. We compute the basic reproduction number $\mathcal{R}_0^2(\mathbb{P}, m, n)$ for the general host-vector model and prove that the disease free equilibrium is globally asymptotically stable (GAS) if $\mathcal{R}_0^2(\mathbb{P}, m, n) \leq 1$. We also show that there exists a unique interior endemic equilibrium that is GAS whenever $\mathcal{R}_0^2(\mathbb{P}, m, n) > 1$ in the irreducible case, that is, when the hosts' groups and vector patches are strongly connected. When irreducibility does not hold, the existence of boundary equilibria is identified. In addition, we provide explicit expression for the basic reproduction number whenever the residence time matrix $\mathbb{P}$ is of rank one. Finally, we briefly explore the role of variability in the number of patches and groups on the basic reproduction number, $\mathcal{R}_0^2(\mathbb{P}, m, n)$ and in the process, bounds for $\mathcal{R}_0^2(\mathbb{P}, m, n)$ are identified.

Our results generalize those of [18,39,54] since our models account for the time-dependant *effective* patch population size. The approach we considered here includes the case where the hosts' structure is defined by residency ( see Section 5.1) as well as the case when the hosts' structure is defined by groups or classes that are independent from the spatially explicit patches.

In short, the contributions of this manuscript are primarily tied to the *effective* population size, a function of the mobility matrix $\mathbb{P} = (p_{ij})_{\substack{1\leq i\leq n,\\ 1\leq j\leq m}}$, where the $p_{ij}$ denotes the proportion of time the host of group $i$ (or the member of a well defined class $i$) spends in environment $j$. We explicitly study the role of the matrix $\mathbb{P}$ on $\mathcal{R}_0$ and connected the dynamics to the reducibility and irreducibility structure of the system. Theorems were established and examples provided on the role of *pachiness* and *groupness* on the disease dynamics.

## Acknowledgments

We are grateful to the handling editor and two anonymous reviewers for their helpful comments and suggestions which led to an improvement of this paper. C.C.C is supported in part by grant #1R01GM100471-01 from the National Institute of General Medical Sciences (NIGMS) at the National Institutes of Health. The contents of this manuscript are solely the responsibility of the authors and do not necessarily represent the official views of DHS or NIGMS. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

## References

[1] B. Adams, D.D. Kapan, Man bites mosquito: understanding the contribution of human movement to vector-borne disease dynamics, PloS One 4 (2009).
[2] J. Arino, C. Bowman, A. Gumel, S. Portet, Effect of pathogen-resistant vectors on the transmission dynamics of a vector-borne disease., J Biol. Dyn. 1 (2007) 320–346.
[3] J. Arino, A. Ducrot, P. Zongo, A metapopulation model for malaria with transmission-blocking partial immunity in hosts, J. Math. Biol. 64 (2012) 423–448.
[4] J.L. Aron, R.M. May, The population dynamics of malaria, in: The Population Dynamics of Infectious diseases: Theory and Applications, Springer, 1982, pp. 139–179.
[5] P. Auger, E. Kouokam, G. Sallet, M. Tchuente, B. Tsanou, The ross-macdonald model in a patchy environment, Math. Biosci. 216 (2008) 123–131.
[6] N.T. Bailey, et al., The biomathematics of malaria. the biomathematics of diseases: 1., in: The biomathematics of malaria. The Biomathematics of Diseases: 1., 1982.
[7] D. Bichara, Y. Kang, C. Castillo-Chavez, R. Horan, C. Perrings, SIS and SIR models under virtual dispersal, Bull. Math. Biol. 77 (2015) 2004–2034.
[8] R. Biritwum, J. Welbeck, G. Barnish, Incidence and management of malaria in two communities of different socio–economic level, in accra, ghana, Ann. Trop. Med. Parasitol. 94 (2000) 771–778.
[9] D.D. Bonnet, D.J. Worcester, The dispersal of aedes albopictus in the territory of hawaii, Am. J. Trop. Med. Hyg. 1 (1946) 465–476.
[10] C. Castillo-Chavez, R. Curtiss, P. Daszak, S.A. Levin, O. Patterson-Lomba, C. Perrings, G. Poste, S. Towers, Beyond ebola: lessons to mitigate future pandemics, Lancet Glob. Health 3 (2015) e354–e355.
[11] C. Castillo-Chavez, B. Song, J. Zhangi, An epidemic model with virtual mass transportation: the case of smallpox, Bioterrorism: Math. Model. Appl. Homel. Secur. 28 (2003) 173.
[12] C. Castillo-Chavez, H.R. Thieme, Asymptotically autonomous epidemic models, in: O. Arino, A. D.E., M. Kimmel (Eds.), Mathematical Population Dynamics: Analysis of Heterogeneity, Theory of Epidemics, vol. One, Wuerz, 1995.
[13] C. Castillo-Chavez, J.X. Velasco-Hernandez, S. Fridman, Modeling contact structures in biology, in: S.A. Levin (Ed.), Frontiers in Mathematical Biology, vol. 100, Springer, 1994, pp. 454–491.
[14] N. Chitnis, Using Mathematical Models in Controlling The Spread of Malaria, University of Arizona, 2005 Ph.d thesis.
[15] N. Chitnis, J.M. Cushing, J.M. Hyman, Bifurcation analysis of a mathematical model for malaria transmission, SIAM J. Appl. Math. 67 (2006) 24–45. (electronic)
[16] N. Chitnis, J.M. Hyman, J. Cushing, Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model, Bull. Math. Biol. (2008).

[17] N. Chitnis, J.M. Hyman, C.A. Manore, Modelling vertical transmission in vector-borne diseases with applications to rift valley fever, J. Biol. Dyn. 7 (2013) 11–40.
[18] C. Cosner, J. Beier, R. Cantrell, D. Impoinvil, L. Kapitanski, M. Potts, A. Troyo, S. Ruan, The effects of human movement on the persistence of vector-borne diseases, J. Theor. Biol. 258 (2009) 550–560.
[19] O. Diekmann, J.A.P. Heesterbeek, J.A.J. Metz, On the definition and the computation of the basic reproduction ratio $R_0$ in models for infectious diseases in heterogeneous populations, J. Math. Biol. 28 (1990) 365–382.
[20] K. Dietz, transmission and control of arbovirus diseases, in: D. Ludwig, K. Cooke (Eds.), SIMS 1974 Utah Conference Proceedings, 1975, pp. 104–121.
[21] K. Dietz, Epidemiologic interference of virus population, J. Math. Biol. 8 (1979) 291–300.
[22] K. Dietz, Models for vector-borne parasitic diseases, in: Vito Volterra Symposium on Mathematical Models in Biology (Rome, 1979), in: Lecture Notes in Biomath., Springer, Berlin, vol. 39, 1980, pp. 264–277.
[23] Y. Dumont, F. Chiroleu, Vector control for the chikungunya disease, Math. Biosci. Eng 7 (2010) 313–345.
[24] Y. Dumont, F. Chiroleu, C. Domerg, On a temporal model for the chikungunya disease: modeling, theory and numerics, Math. Biosci. 213 (2008) 80–91.
[25] C. Dye, G. Hasibeder, Population dynamics of mosquito-borne disease: effects of flies which bite some people more frequently than others, Trans. R. Soc. Tropical Med. Hyg. 80 (1986) 69–77.
[26] A. Elbers, C. Koenraadt, R. Meiswinkel, Mosquitoes and culicoides biting midges: vector range and the influence of climate change, Rev. Sci. Tech. Off. Int. Epiz 34 (2015) 123–137.
[27] F. Forouzannia, A.B. Gumel, Mathematical analysis of an age-structured model for malaria transmission dynamics, Math. Biosci. 247 (2014) 80–94.
[28] D. Gao, S. Ruan, A multipatch malaria model with logistic growth populations, SIAM J. Appl. Math. 72 (2012) 819–841.
[29] S.M. Garba, A.B. Gumel, M.A. Bakar, Backward bifurcations in dengue transmission dynamics, Math. Biosci. 215 (2008) 11–25.
[30] D. Grünbaum, Translating stochastic density-dependent individual behavior with sensory constraints to an Eulerian model of animal swarming, J. Math. Biol. 33 (1994) 139–161.
[31] G. Hasibeder, C. Dye, Population dynamics of mosquito-borne disease: Persistence in a completely heterogeneous environment, Theor. Popul. Biol. 33 (1988) 31–53.
[32] M. Hirsch, The dynamical system approach to differential equations, Bull. AMS 11 (1984) 1–64.
[33] N.A. Honório, W.d. C. Silva, P.J. Leite, J.M. Gonçalves, L.P. Lounibos, R.L.-d. Oliveira, Dispersal of aedes aegypti and aedes albopictus (diptera: Culicidae) in an urban endemic dengue area in the state of rio de janeiro, brazil, Memórias do Instituto Oswaldo Cruz 98 (2003) 191–198.
[34] A. Iggidr, G. Sallet, M.O. Souza, On the dynamics of a class of multi-group models for vector-borne diseases, J. Math. Anal. Appl. 441 (2016) 723–743.
[35] A. Iggidr, G. Sallet, B. Tsanou, Global stability analysis of a metapopulation sis epidemic model, Math. Popul. Stud. 19 (2012) 115–129.
[36] C. Kaufmann, H. Briegel, Flight performance of the malaria vectors anopheles gambiae and anopheles atroparvus, J. Vector Ecol. 29 (2004) 140–153.
[37] K. Koram, S. Bennett, J. Adiamah, B. Greenwood, Socio-economic risk factors for malaria in a peri-urban area of the gambia, Trans. R. Soc. Tropical Med. Hyg. 89 (1995) 146–150.
[38] G. Kuno, Review of the factors modulating dengue transmission, Epidemiol. Rev. 17 (1995) 321–335.
[39] S. Lee, C. Castillo-Chavez, The role of residence times in two-patch dengue transmission dynamics and optimal strategies, J. Theor. Biol. 374 (2015) 152–164.
[40] G. Macdonald, Epidemiological basis of malaria control, Bull. World Health Organ. 15 (1956a) 613.
[41] G. Macdonald, The Epidemiology and Control of Malaria, Oxford University Press, London, 1957.
[42] P. Martens, L. Hall, Malaria on the move: human population movement and malaria transmission., Emerg. Infectious Dis. 6 (2000) 103.
[43] A.G. Ngwa, On the population dynamics of the malaria vector, Bull. Math. Biol. (2006).
[44] A.G. Ngwa, W. Shu, A mathematical model for endemic malaria with variable human and mosquito population, Math. Comput. Model. 32 (2000) 747–763.
[45] M. Niebylski, G. Craig Jr, Dispersal and survival of aedes albopictus at a scrap tire yard in missouri., J. Am. Mosquito Control Assoc. 10 (1994) 339–343.
[46] R. Novak, A north american model to contain the spread of aedes albopictus through tire legislation., Parassitologia 37 (1995) 129–139.
[47] A. Okubo, Diffusion and ecological problems: mathematical models, Biomathematics 10 (1980).
[48] A. Okubo, S.A. Levin, Diffusion and Ecological Problems: Modern Perspectives, vol. 14, Springer Science & Business Media, 2013.
[49] O. Onwujekwe, B. Uzochukwu, S. Eze, E. Obikeze, C. Okoli, O. Ochonma, Improving equity in malaria treatment: relationship of socio-economic status with health seeking as well as with perceptions of ease of using the services of different providers for the treatment of malaria in nigeria, Malar. J. 7 (2008) 1–10.
[50] World Health Organization, Dengue Control, World Health Organization, 2015. http://www.who.int/denguecontrol/mosquito/en/ (accessed 14.09.16).
[51] World Health Organization, et al., A Global Brief on Vector-Borne Diseases, World Health Organization, 2014. http://www.who.int/campaigns/world-health-day/2014/global-brief/en/ (accessed 14.09.16).
[52] C. Perrings, C. Castillo-Chavez, G. Chowell, P. Daszak, E.P. Fenichel, D. Finnoff, R.D. Horan, A.M. Kilpatrick, A.P. Kinzig, N.V. Kuminoff, et al., Merging economics and epidemiology to improve the prediction and management of infectious disease, EcoHealth 11 (2014) 464–475.
[53] P. Reiter, Climate change and mosquito-borne disease: knowing the horse before hitching the cart, Revue scientifique et technique-Office international des épizooties 27 (2014).
[54] D.J. Rodríguez, L. Torres-Sorando, Models of infectious diseases in spatially heterogeneous environments, Bull. Math. Biol. 63 (2001) 547–571.
[55] D. Roiz, R. Eritja, R. Escosa, J. Lucientes, E. Marques, R. Melero-Alcibar, S. Ruiz, R. Molina, A survey of mosquitoes breeding in used tires in spain for the detection of imported potential vector species, J. Vector Ecol. 32 (2007) 10–15.
[56] R. Ross, The Prevention of Malaria, John Murray, 1911.
[57] R. Ross, An application of the theory of probabilities to the study of a priori pathometry. Part i, Proc. R. Soc. Lond. A 92 (1916) 204–230.
[58] R. Ross, H. Hudson, An application of the theory of probabilities to the study of a priori pathometry. Part ii, Proc. R. Soc. Lond. A 93 (1917) 212–225.
[59] R. Ross, H. Hudson, An application of the theory of probabilities to the study of a priori pathometry. Part iii, Proc. R. Soc. Lond. A 93 (1917) 225–240.
[60] N.W. Ruktanonchai, D.L. Smith, P. De Leenheer, Parasite sources and sinks in a patched ross-macdonald malaria model with human and mosquito movement: implications for control, Math. Biosci. 279 (2016) 90–101.
[61] D.L. Smith, J. Dushoff, F.E. McKenzie, The risk of a mosquito-borne infection in a heterogeneous environment, PLoS Biol. 2 (2004) e368.
[62] H. Smith, Cooperative systems of differential equations with concave nonlinearities, Nonlinear Anal.: Theory, Meth. Appl. 10 (1986) 1037–1052.
[63] M.P. Stürchler, The vector and measures against mosquito bites, in: P. Schlagenhauf-Lawlor (Ed.), Travelers' Malaria, 2001, pp. 88–105. BC Decker, ch. 8
[64] M.M. Thiboutot, S. Kannan, O.U. Kawalekar, D.J. Shedlock, A.S. Khan, G. Sarangan, P. Srikanth, D.B. Weiner, K. Muthumani, Chikungunya: a potentially emerging epidemic, PLoS Negl Trop Dis 4 (2010) e623.
[65] M.A. Tolle, Mosquito-borne diseases, Curr. Probl. Pediatric Adolesc. Health Care 39 (2009) 97–140.
[66] L. Torres-Sorando, D.J. Rodrìguez, Models of spatio-temporal dynamics in malaria, Ecol. Model. 104 (1997) 231–240.
[67] P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci. 180 (2002) 29–48.
[68] M. Vidyasagar, Decomposition techniques for large-scale systems with nonadditive interactions: Stability and stabilizability., IEEE Trans. Autom. Control 25 (1980) 773–779.
[69] Y. Xiao, X. Zou, Transmission dynamics for vector-borne diseases in a patchy environment, J. Math. Biol. 69 (2014) 113–146.