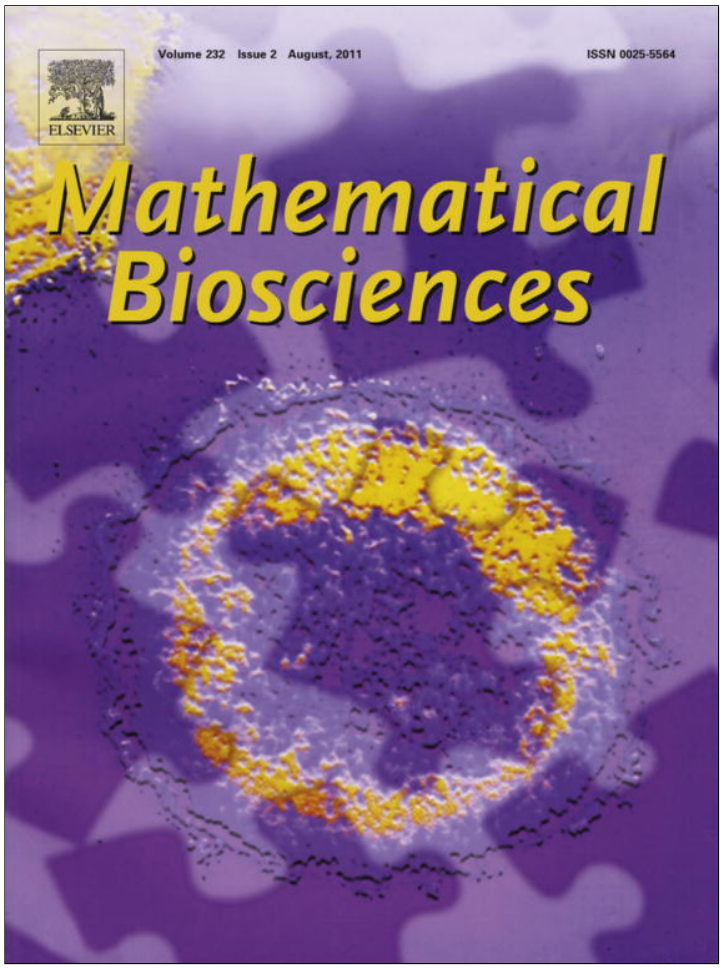

Mathematical Biosciences 232 (2011) 110–115

Contents lists available at ScienceDirect

Mathematical Biosciences

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

An SIS patch model with variable transmission coefficients ☆

Daozhou Gao, Shigui Ruan *

Department of Mathematics, University of Miami, Coral Gables, FL 33124-4250, USA

ARTICLE INFO

Article history:
Received 9 February 2011
Received in revised form 2 May 2011
Accepted 7 May 2011
Available online 18 May 2011

Keywords:
Basic reproduction number
Media coverage
Patch model
Infectious disease
Global stability
Uniform persistence

ABSTRACT

In this paper, an SIS patch model with non-constant transmission coefficients is formulated to investigate the effect of media coverage and human movement on the spread of infectious diseases among patches. The basic reproduction number $\mathcal{R}_0$ is determined. It is shown that the disease-free equilibrium is globally asymptotically stable if $\mathcal{R}_0 \leqslant 1$, and the disease is uniformly persistent and there exists at least one endemic equilibrium if $\mathcal{R}_0 > 1$. In particular, when the disease is non-fatal and the travel rates of susceptible and infectious individuals in each patch are the same, the endemic equilibrium is unique and is globally asymptotically stable as $\mathcal{R}_0 > 1$. Numerical calculations are performed to illustrate some results for the case with two patches.

1. Introduction

It has been observed that media coverage can affect the spread and control of infectious diseases (see [9] and the references cited therein). During outbreaks of serious infectious diseases such as the SARS outbreak in 2003 and the H1N1 influenza pandemic in 2009, public media has massive reports on the number of infections and deaths per day, the locations where these happen, the symptoms of the disease, the proper protections to decrease the possibility of being infected, etc. People follow the reports and thus choose to protect themselves by reducing their social activities and direct contacts with others, especially with those high-risk groups, which could therefore lead to a reduction of effective contacts between susceptible individuals and infectious individuals. In a recent paper [3], Cui et al. proposed an SIS epidemiological model incorporating media coverage

$$\begin{aligned} \frac{dS}{dt} &= A - dS - \beta(I)\frac{SI}{S+I} + \gamma I, \\ \frac{dI}{dt} &= \beta(I)\frac{SI}{S+I} - (d+v+\gamma)I, \end{aligned} \tag{1.1}$$

where the transmission coefficient $\beta(I)$ is a non-increasing function of the number of the infectious individuals. They defined a threshold for (1.1) below which all orbits converge to the disease-free equilibrium and above which all orbits with $I(0) > 0$ converge to a unique endemic equilibrium.

In this paper, we shall study an SIS patch model for the transmission of an infectious disease with population dispersal between p patches. Within a single patch, our model is based on that of Cui et al. [3]. Let $S_i(t)$ and $I_i(t)$ denote, respectively, the number of susceptible and infectious individuals in patch i at time t. The population dynamics is described by the following system of ordinary differential equations with non-negative initial conditions:

$$\begin{aligned} \frac{dS_i}{dt} &= A_i - d_iS_i - \beta_i(I_i)\frac{S_iI_i}{S_i+I_i} + \gamma_iI_i + \sum_{j=1}^{p} m_{ij}S_j, \quad 1 \leqslant i \leqslant p, \\ \frac{dI_i}{dt} &= \beta_i(I_i)\frac{S_iI_i}{S_i+I_i} - (d_i+v_i+\gamma_i)I_i + \sum_{j=1}^{p} n_{ij}I_j, \quad 1 \leqslant i \leqslant p. \end{aligned} \tag{1.2}$$

In patch i, $A_i > 0$ is the recruitment rate, $d_i > 0$ is the natural death rate, $\gamma_i > 0$ is the recovery rate and $v_i \geqslant 0$ is the disease-induced death rate. The transmission coefficient in patch i is $\beta_i(I_i) = a_i - b_if_i(I_i)$, where a_i is the usual transmission coefficient without considering the impact of media reported number of infective individuals, b_i is the maximum reduced transmission coefficient due to the media effect and $f_i(I_i)$ is a saturation function to measure the impact of the reported number of infected individuals. Similar to Cui et al. [3], we assume that

$$a_i > b_i \geqslant 0, \quad f_i(0) = 0, \quad f_i(I_i) \in C^1([0,\infty)) \quad \text{with } f_i'(I_i) \geqslant 0, \quad \lim_{I_i\to\infty} f_i(I_i) = 1 \quad \text{for } i = 1,\ldots,p.$$

Typical examples of $f_i(I_i)$ with such properties are $1 - k_i/(k_i + I_i^{n_i})$ with $k_i > 0$ and $n_i > 0$, and $1 - e^{-k_iI_i}$ with $k_i > 0$. When $b_i = 0$ for

☆ Research was partially supported by NSF Grant DMS-1022728.
* Corresponding author.
E-mail addresses: dzgao@math.miami.edu (D. Gao), ruan@math.miami.edu (S. Ruan).

0025-5564/$ - see front matter © 2011 Elsevier Inc. All rights reserved.
doi:10.1016/j.mbs.2011.05.001

$i = 1,2,\ldots,p$, i.e., the media impact is ignored, model (1.2) with two patches was studied in Salmani and van den Driessche [11]. The immigration rates from patch j to patch i for $i \neq j$ of susceptible and infectious humans are denoted, respectively, by $m_{ij} \geqslant 0$ and $n_{ij} \geqslant 0$, while the emigration rates of susceptible and infectious humans in patch i are denoted, respectively, by $-m_{ii} \geqslant 0$ and $-n_{ii} \geqslant 0$. For simplicity, deaths and births during travel are neglected. Thus, we have

$$\sum_{j=1}^{p} m_{ji} = 0 \quad \text{and} \quad \sum_{j=1}^{p} n_{ji} = 0 \quad \text{for } i = 1,2,\ldots,p.$$

Unless otherwise indicated, the travel rate matrices $(m_{ij})_{p\times p}$ and $(n_{ij})_{p\times p}$ are assumed to be irreducible.

The organization of this paper is as follows. In Section 2, the basic reproduction number $\mathcal{R}_0$ is defined and it is shown to be a threshold of the disease dynamics. Namely, the disease can be eradicated if $\mathcal{R}_0 \leqslant 1$ and will be endemic if $\mathcal{R}_0 > 1$. In Section 3, we consider the special case when susceptible and infectious individuals have identical travel rates and there is no disease-induced death, and present a global qualitative analysis. In the final section, we conclude with some numerical examples and a brief discussion.

2. Threshold dynamics

We first introduce some notations which will be used throughout this paper. Let $\mathbb{R}^p_+ = \{x \in \mathbb{R}^p : x_i \geqslant 0 \text{ for } 1 \leqslant i \leqslant p\}$ be the positive orthant in $\mathbb{R}^p$ and $\operatorname{Int}\mathbb{R}^p_+ = \{x \in \mathbb{R}^p : x_i > 0 \text{ for } 1 \leqslant i \leqslant p\}$ be the interior of $\mathbb{R}^p_+$. We write $x \leqslant y$ and $y \geqslant x$ whenever $y - x \in \mathbb{R}^p_+$, $x < y$ and $y > x$ whenever $y - x \in \mathbb{R}^p_+$ and $x \neq y$, and $x \ll y$ and $y \gg x$ whenever $y - x \in \operatorname{Int}\mathbb{R}^p_+$. If $x, y \in \mathbb{R}^p_+$ and $x \leqslant y$, we let $[x,y] = \{z \in \mathbb{R}^p_+ : x \leqslant z \leqslant y\}$.

Let $N_i(t) = S_i(t) + I_i(t)$ be the total population in patch i at time t, and let the new infection term in patch i equal zero whenever $N_i = 0$ [5]. The following result indicates that model (1.2) is mathematically and biologically well posed.

Theorem 2.1. *Consider system (1.2) with non-negative initial conditions. Then the system has a unique solution defined for all time $t \geqslant 0$, and all disease state variables remain non-negative. Moreover, the total population $N(t) = \sum_{i=1}^{p} N_i(t)$ is bounded.*

Proof. The vector field defined by (1.2) is Lipschitzian in each compact set in $\mathbb{R}^{2p}_+$, so the initial value problem has a unique solution which exists for all $t \geqslant 0$ [25]. The non-negative property of state variables can be immediately verified.

Let $\mathcal{A} = \sum_{i=1}^{p} A_i$ and $\mathcal{D} = \min_{1\leqslant i\leqslant p} d_i$. Since

$$\frac{dN}{dt} = \sum_{i=1}^{p}(A_i - d_i N_i - v_i I_i) \leqslant \sum_{i=1}^{p}(A_i - d_i N_i) \leqslant \mathcal{A} - \mathcal{D}N,$$

by a comparison theorem, $N(t)$ is bounded above by $\max\{\mathcal{A}/\mathcal{D}, N(0)\}$. □

2.1. Basic reproduction number

Let the right hand side of (1.2) be zero, one can verify that model (1.2) always admits a disease-free equilibrium (DFE), denoted by $E_0 = \left(S_1^0, S_2^0, \ldots, S_p^0, 0, 0, \ldots, 0\right)$. Indeed, there is a DFE if and only if $S^0 = \left(S_1^0, S_2^0, \ldots, S_p^0\right)$ satisfies $B(S^0)^T = \mathbf{A}$, where $B = (\delta_{ij} d_i - m_{ij})_{p\times p}$ and $\mathbf{A} = (A_1, A_2, \ldots, A_p)^T$. Here δ_{ij} denotes the Kronecker delta (i.e. 1 when $i = j$ and 0 otherwise). It follows from Corollary 4.3.2 in Smith [13] that B^{-1} is a positive matrix. Hence $S^0 = (B^{-1}\mathbf{A})^T \gg 0$ guarantees the existence and uniqueness of the disease-free equilibrium.

Now, we calculate the basic reproduction number of (1.2). Using the recipe of van den Driessche and Watmough [18], we have

$$F = (\delta_{ij} a_i)_{p\times p} \quad \text{and} \quad V = (\delta_{ij}(d_i + v_i + \gamma_i) - n_{ij})_{p\times p}.$$

Therefore, the basic reproduction number is $\mathcal{R}_0 = \rho(FV^{-1})$, where ρ denotes the spectral radius and it is the same as that of the classical model with fixed transmission coefficients.

Observe that $\mathcal{R}_0$ is independent of the parameters A_i, b_i for $i = 1,2,\ldots,p$, and the travel rates of susceptible individuals. It is easy to see that $\mathcal{R}_0$ is increasing in a_i while it is decreasing with respect to d_i, v_i and γ_i. The following estimation on the basic reproduction number was already showed by Wang and Mulone [20] and Salmani and van den Driessche [11] for $p = 2$, so here is an interesting generalization for general p.

Proposition 2.2. *Let $\mathcal{R}_0^{(i)} = a_i/(d_i + v_i + \gamma_i)$ be the basic reproduction number for patch i in isolation and write $\widetilde{\mathcal{R}}_0^{(i)} = a_i/(d_i + v_i + \gamma_i - n_{ii})$ as a modified reproduction number that contains travel of infectives out of patch i. Then*

$$\max\left\{\max_{1\leqslant i\leqslant p}\widetilde{\mathcal{R}}_0^{(i)}, \min_{1\leqslant i\leqslant p}\mathcal{R}_0^{(i)}\right\} \leqslant \mathcal{R}_0 \leqslant \max_{1\leqslant i\leqslant p}\mathcal{R}_0^{(i)}.$$

Proof. The inequality $\max_{1\leqslant i\leqslant p}\widetilde{\mathcal{R}}_0^{(i)} \leqslant \mathcal{R}_0 \leqslant \max_{1\leqslant i\leqslant p}\mathcal{R}_0^{(i)}$ follows a similar analysis used in the proof of Theorem 3.4 in Gao and Ruan [4]. It then suffices to prove that $\min_{1\leqslant i\leqslant p}\mathcal{R}_0^{(i)} \leqslant \mathcal{R}_0$.

Let $c_i = d_i + v_i + \gamma_i$ for $i = 1,2,\ldots,p$ and $s(\cdot)$ denote the spectral bound of a matrix. Since V has a positive inverse, FV^{-1} is a positive matrix. Using the Perron-Frobenius theorem, $\mathcal{R}_0 = s(FV^{-1})$ is a simple eigenvalue of FV^{-1} associated to a positive eigenvector $\mathbf{v}$ and any eigenvector $\mathbf{w} > 0$ of FV^{-1} is a positive multiple of $\mathbf{v}$ (see [13]). Hence, $FV^{-1}\mathbf{v} = \mathcal{R}_0\mathbf{v}$, which is equivalent to $-VF^{-1}\mathbf{v} = -\frac{1}{\mathcal{R}_0}\mathbf{v}$, where

$$-VF^{-1} = (n_{ij})_{p\times p}F^{-1} - diag\left\{1/\mathcal{R}_0^{(1)}, 1/\mathcal{R}_0^{(2)}, \ldots, 1/\mathcal{R}_0^{(p)}\right\}.$$

Since $-VF^{-1}$ is a quasi-positive and irreducible matrix and $\mathbf{v}$ is positive, we conclude that $s(-VF^{-1}) = -1/\mathcal{R}_0$. The facts $M_L \leqslant -VF^{-1} \leqslant M_U$ and $s((n_{ij})_{p\times p}F^{-1}) = 0$ imply that

$$s(M_L) = -\max_{1\leqslant i\leqslant p}\frac{1}{\mathcal{R}_0^{(i)}} \leqslant s(-VF^{-1}) = -1/\mathcal{R}_0 \leqslant s(M_U) = -\min_{1\leqslant i\leqslant p}\frac{1}{\mathcal{R}_0^{(i)}},$$

where

$$M_L = (n_{ij})_{p\times p}F^{-1} - \max_{1\leqslant i\leqslant p}\frac{1}{\mathcal{R}_0^{(i)}}\cdot diag\{1,1,\ldots,1\},$$

$$M_U = (n_{ij})_{p\times p}F^{-1} - \min_{1\leqslant i\leqslant p}\frac{1}{\mathcal{R}_0^{(i)}}\cdot diag\{1,1,\ldots,1\}.$$

A direct simplification completes the proof of the proposition. □

Remark 2.3. By the results in Hadeler and Thieme [6], $s(-VF^{-1})$ depends in a monotone way on the travel rate of infectious humans n_{ij} for $i,j = 1,2,\ldots,p$ and $i \neq j$. More precisely, it is always strictly decreasing or strictly increasing or it is constant. So is $\mathcal{R}_0 = -1/s(-VF^{-1})$.

Like in the single patch model (1.2) or many other epidemic models, we have the global stability of the DFE for system (1.2) as $\mathcal{R}_0 < 1$.

Theorem 2.4. *The DFE of system (1.2) is globally asymptotically stable (GAS) if $\mathcal{R}_0 \leqslant 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. From Theorem 2 in van den Driessche and Watmough [18], E_0 is locally asymptotically stable if $\mathcal{R}_0 < 1$, but unstable if $\mathcal{R}_0 > 1$.

Now it suffices to prove that all solutions converge to the DFE when $\mathcal{R}_0 \leqslant 1$. The inequalities $S_i/N_i \leqslant 1$ and $\beta_i(I_i) \leqslant a_i$ yield

$$\frac{dI_i}{dt} \leqslant a_i I_i - (d_i + \nu_i + \gamma_i) I_i + \sum_{j=1}^{p} n_{ij} I_j, \quad 1 \leqslant i \leqslant p.$$

By applying the algorithm in Kamgang and Sallet [8], we know that the DFE is GAS whenever $\mathcal{R}_0 \leqslant 1$ (or like in Sun et al. [16], by a standard comparison theorem). □

2.2. Uniform persistence

Using the techniques of persistence theory [24], we can show the uniform persistence of the disease and the existence of at least one endemic equilibrium when $\mathcal{R}_0 > 1$. Thus, the basic reproduction number $\mathcal{R}_0$ is a threshold parameter of the disease dynamics. The proof below is analogous to those of Theorem 2.3 in Wang and Zhao [21] and Theorem 3.7 in Gao and Ruan [4].

Theorem 2.5. *For model (1.2), if $\mathcal{R}_0 > 1$, then the disease is uniformly persistent, i.e., there exists a constant $\kappa > 0$ such that every solution $\phi_t(\mathbf{x}_0) \equiv (S_1(t), \ldots, S_p(t), I_1(t), \ldots, I_p(t))$ of system (1.2) with $\mathbf{x}_0 \equiv (S_1(0), \ldots, S_p(0), I_1(0), \ldots, I_p(0)) \in \mathbb{R}^p_+ \times \mathbb{R}^p_+ \setminus \{0\}$ satisfies*

$$\liminf_{t\to\infty} I_i(t) > \kappa \quad \text{for } i = 1, 2, \ldots, p,$$

and (1.2) admits at least one endemic equilibrium.

Proof. Let

$$\begin{aligned} X &= \{(S_1, \ldots, S_p, I_1, \ldots, I_p) : S_i \geqslant 0,\ I_i \geqslant 0,\ i = 1, 2, \ldots, p\},\\ X_0 &= \{(S_1, \ldots, S_p, I_1, \ldots, I_p) \in X : I_i > 0,\ i = 1, 2, \ldots, p\},\\ \partial X_0 &= X \setminus X_0 = \{(S_1, \ldots, S_p, I_1, \ldots, I_p) \in X : I_i = 0\\ &\qquad \text{for some } i \in \{1, 2, \ldots, p\}\}. \end{aligned}$$

It suffices to prove that ∂X_0 repels uniformly the solutions of system (1.2) in X_0. Clearly, ∂X_0 is relatively closed in X. It is immediate that X and X_0 are positively invariant. Theorem 2.1 implies that system (1.2) is point dissipative.

Denote $M_\partial = \{\mathbf{x}_0 \in \partial X_0 : \phi_t(\mathbf{x}_0) \in \partial X_0 \text{ for } t \geqslant 0\}$ and $D = \{\mathbf{x}_0 \in X : I_i = 0,\ i = 1, 2, \ldots, p\}$. Obviously, $D \subset M_\partial$. On the other hand, we have $I_1(0) + \cdots + I_p(0) > 0$ for any $\mathbf{x}_0 \in \partial X_0 \backslash D$. By the irreducibility of the travel rate matrix $(n_{ij})_{p\times p}$, we know that $\phi_t(\mathbf{x}_0) \in X_0$ for all $t > 0$. Therefore, $\mathbf{x}_0 \notin M_\partial$ and $M_\partial \subset D$, which implies that $M_\partial = D$.

The disease-free equilibrium E_0 is the unique equilibrium in M_∂. Let $W^s(E_0)$ be the stable manifold of E_0. We now show that $W^s(E_0) \cap X_0 = \emptyset$ when $\mathcal{R}_0 > 1$. Let

$$M_\epsilon = F - V - diag\{a_1\epsilon + b_1\epsilon - b_1\epsilon^2, \ldots, a_p\epsilon + b_p\epsilon - b_p\epsilon^2\}.$$

Since $s(F - V) > 0$ if and only if $\mathcal{R}_0 > 1$, there is an $\epsilon_1 > 0$ such that $s(M_\epsilon) > 0$ for $\epsilon \in [0, \epsilon_1]$. Choose η small enough such that

$$S_i(0)/N_i(0) \geqslant 1 - \epsilon_1 \quad \text{and} \quad f_i(I_i(0)) \leqslant \epsilon_1 \quad \text{for } i = 1, \ldots, p,\ \ \|\mathbf{x}_0 - E_0\| \leqslant \eta.$$

We claim that $\limsup_{t\to\infty} \|\phi_t(\mathbf{x}_0) - E_0\| > \eta$ for $\mathbf{x}_0 \in X_0$, where $\|\cdot\|$ is the usual Euclidean norm. Suppose not, after translation, we have $\|\phi_t(\mathbf{x}_0) - E_0\| \leqslant \eta$ for all $t \geqslant 0$ and hence

$$\frac{dI_i}{dt} \geqslant (a_i - b_i\epsilon_1)(1 - \epsilon_1) I_i - (d_i + \nu_i + \gamma_i) I_i + \sum_{j=1}^{p} n_{ij} I_j, \quad 1 \leqslant i \leqslant p.$$

Notice that M_{ϵ_1} has a positive eigenvalue $s(M_{\epsilon_1})$ associated to a positive eigenvector. It follows from a comparison theorem that $I_i(t) \to \infty$ as $t \to \infty$ for $i = 1, 2, \ldots, p$, a contradiction.

Since E_0 is globally stable in M_∂, it follows that $\{E_0\}$ is an isolated invariant set and acyclic. By Theorem 4.6 in Thieme [17], system (1.2) is uniformly persistent with respect to $(X_0, \partial X_0)$. Furthermore, by Theorem 2.4 in Zhao [22], we know that system (1.2) has an equilibrium $\bar{E} = (\bar{S}_1, \ldots, \bar{S}_p, \bar{I}_1, \ldots, \bar{I}_p) \in X_0$. The first equation of (1.2) ensures that $\bar{S}_i > 0$ for $i = 1, \ldots, p$. This means that $\bar{E}$ is an endemic equilibrium of system (1.2). □

Remark 2.6. Neither the travel of susceptible individuals nor the media coverage affects the persistence and extinction of the disease. By Proposition 2.2, if $\mathcal{R}_0^{(i)} > 1$ (or $\leqslant 1$) for $i = 1, 2, \ldots, p$, then $\mathcal{R}_0 > 1$ (or $\leqslant 1$). Biologically, this means that the disease persists or dies out in each isolated patch then remains persistent or extinct, respectively, when human movement occurs.

3. Model with restrictions

In the case where there is no disease-induced death (i.e., $\nu_i = 0$ for $i = 1, 2, \ldots, p$) and susceptible and infectious individuals have identical travel rates (i.e., $m_{ij} = n_{ij}$ for $i, j = 1, 2, \ldots, p$), the dynamics of the individuals are governed by the following model:

$$\begin{aligned} \frac{dS_i}{dt} &= A_i - d_i S_i - \beta_i(I_i)\frac{S_i I_i}{S_i + I_i} + \gamma_i I_i + \sum_{j=1}^{p} m_{ij} S_j, \quad 1 \leqslant i \leqslant p,\\ \frac{dI_i}{dt} &= \beta_i(I_i)\frac{S_i I_i}{S_i + I_i} - (d_i + \gamma_i) I_i + \sum_{j=1}^{p} m_{ij} I_j, \quad 1 \leqslant i \leqslant p. \end{aligned} \tag{3.1}$$

Sun et al. [16] presented a global qualitative analysis for system (3.1) with two-patch when $\mathcal{R}_0 > 1$. Here we study the model with an arbitrary number of patches by using the theory of monotone dynamical systems [13].

Theorem 3.1. *If $\mathcal{R}_0 > 1$, then system (3.1) has a unique endemic equilibrium which is globally asymptotically stable relative to $\mathbb{R}^p_+ \times \mathbb{R}^p_+ \setminus \{0\}$.*

Proof. Adding the two equations in system (3.1) leads to

$$\frac{dN_i}{dt} = A_i - d_i N_i + \sum_{j=1}^{p} m_{ij} N_j, \quad 1 \leqslant i \leqslant p. \tag{3.2}$$

Obviously, system (3.2) has a unique equilibrium, labeled by $N^* = (N_1^*, N_2^*, \ldots, N_n^*)$, which is equal to $S^0 = \left(S_1^0, S_2^0, \ldots, S_p^0\right)$ and is globally asymptotically stable for (3.2). System (3.1) is then equivalent to the following system

$$\begin{aligned} \frac{dN_i}{dt} &= A_i - d_i N_i + \sum_{j=1}^{p} m_{ij} N_j, \quad 1 \leqslant i \leqslant p,\\ \frac{dI_i}{dt} &= \beta_i(I_i)\frac{N_i - I_i}{N_i} I_i - (d_i + \gamma_i) I_i + \sum_{j=1}^{p} m_{ij} I_j, \quad 1 \leqslant i \leqslant p. \end{aligned} \tag{3.3}$$

Since $N_i(t) \to N_i^*,\ i = 1, 2, \ldots, p$, as $t \to \infty$, (3.3) gives the following limit system

$$\frac{dI_i}{dt} = h_i(I_1, \ldots, I_p) = \beta_i(I_i)\frac{N_i^* - I_i}{N_i^*} I_i - (d_i + \gamma_i) I_i + \sum_{j=1}^{p} m_{ij} I_j, \quad i = 1, 2, \ldots, p. \tag{3.4}$$

Let $h : \mathbb{R}^p_+ \to \mathbb{R}^p$ denote the vector field described by (3.4) and ψ_t denote the corresponding flow. For any $\alpha \in (0, 1)$ and any $(I_1, \ldots, I_p) \in \operatorname{Int}\mathbb{R}^p_+ \cap \mathbb{D}$ with $\mathbb{D} = [0, N^*]$, there hold

$$\begin{aligned} &\beta_i(\alpha I_i)\frac{N_i^* - \alpha I_i}{N_i^*}\alpha I_i - (d_i + \gamma_i)\alpha I_i + \sum_{j=1}^{p} m_{ij}\alpha I_j\\ &> \alpha\left(\beta_i(I_i)\frac{N_i^* - I_i}{N_i^*} I_i - (d_i + \gamma_i) I_i + \sum_{j=1}^{p} m_{ij} I_j\right), \quad i = 1, 2, \ldots, p, \end{aligned}$$

that is, $h(\alpha(I_1,\ldots,I_p)) \gg \alpha h(I_1,\ldots,I_p)$. Thus h is strongly sublinear on $\mathbb{D}$. In addition, $\mathbb{D}$ is positively invariant for (3.4) since

$$h_i(N^*) = -(d_i+\gamma_i)N_i^* + \sum_{j=1}^{p} m_{ij}N_j^* = -A_i - \gamma_i N_i^* < 0, \quad i = 1,2,\ldots,p.$$

Note that the Jacobian matrix of system (3.4) at the origin, $Dh(0)$, satisfies $s(Dh(0)) = s(F-V) > 0$. It is easy to see that Corollary 3.2 in Zhao and Jing [23] also holds if $\mathbb{R}_+^p$ is replaced by a positively invariant order interval in $\mathbb{R}_+^p$. Therefore, system (3.4) has a positive equilibrium $I^* = \left(I_1^*, I_2^*, \ldots, I_p^*\right) \in \mathbb{D}$, which is globally asymptotically stable in $\mathbb{D}\setminus\{0\}$. It is clear from the strong monotonicity of ψ_t that $S_i^* = N_i^* - I_i^* > 0$ for $i = 1,2,\ldots,p$. Hence (3.1) admits a unique positive equilibrium $E^* = \left(S_1^*, S_2^*, \ldots, S_p^*, I_1^*, I_2^*, \ldots, I_p^*\right)$.

Next, we prove that every non-trivial solution to (3.4) in $\mathbb{R}_+^p$ converges to I^*. We claim that (3.4) has no equilibrium in $\mathbb{R}_+^p \setminus \mathbb{D}$. Assume, by contrary, that $I^{**} = \left(I_1^{**}, \ldots, I_p^{**}\right)$ is an equilibrium of (3.4) in $\mathbb{R}_+^p \setminus \mathbb{D}$. It follows from the strong monotonicity of the flow ψ_t that $I^* \ll I^{**}$. From (3.4), we have $M^*(I^*)^T = 0$ and $M^{**}(I^{**})^T = 0$, where $M^* = \left((\beta_i(I_i^*)(N_i^* - I_i^*)/N_i^* - (d_i+\gamma_i))\delta_{ij} + m_{ij}\right)_{p\times p}$ and $M^{**} = \left((\beta_i(I_i^{**})(N_i^* - I_i^{**})/N_i^* - (d_i+\gamma_i))\delta_{ij} + m_{ij}\right)_{p\times p}$. Then $s(M^*) = s(M^{**}) = 0$, which is in contradiction to

$$M^* - M^{**} = \left(\left(\beta_i(I_i^*)(N_i^* - I_i^*)/N_i^* - \beta_i(I_i^{**})(N_i^* - I_i^{**})/N_i^*\right)\delta_{ij}\right)_{p\times p} > 0.$$

So, I^* is the unique non-trivial equilibrium of (3.4) in $\mathbb{R}_+^p$. For any $\mathbf{y}_0 \in \mathbb{R}_+^p \setminus \mathbb{D}$, we have $\mathbf{y}_0 \ll lN^*$ for sufficiently large $l > 1$ and therefore $\psi_t(\mathbf{y}_0) \ll \psi_t(lN^*)$ for $t \geqslant 0$. Note that $h(lN^*) \ll 0$ for $l \geqslant 1$, so $\psi_t(lN^*) \to I^*$ as $t \to \infty$. This means that $\psi_t(\mathbf{y}_0)$ enters into $\mathbb{D}$ for large t and thus $\psi_t(\mathbf{y}_0)$ approaches I^* as $t \to \infty$.

Since both systems (3.2) and (3.4) are locally (globally) asymptotically stable, system (3.3) has $\mathcal{E}^* = \left(N_1^*, N_2^*, \ldots, N_p^*, I_1^*, I_2^*, \ldots, I_p^*\right)$ as a locally asymptotically stable state [19]. A comparison theorem implies that all orbits of (3.3) are forward bounded, while the proof of Theorem 2.5 indicates that no orbit of system (3.3) starting at a point in $\mathbb{R}_+^p \times \mathbb{R}_+^p \setminus \{0\}$ tends to $\left(N_1^*, \ldots, N_p^*, 0, \ldots, 0\right)$ if $\mathcal{R}_0 > 1$. It then follows a similar argument used in the proof of Theorem 4.2 in Seibert and Suarez [12] that $\mathcal{E}^*$ is globally asymptotically stable for system (3.3) relative to $\mathbb{R}_+^p \times \mathbb{R}_+^p \setminus \{0\}$. So is E^* for system (3.1). □

Remark 3.2. The above approach works for a class of SIS patch models where there is no disease-induced death and susceptible and infectious individuals travel at the same rates. As far as we know, most of the existing global results on these models are only for two-patch case. With our approach, for example, one can generalize Theorem 2.7 in Wang and Mulone [20] and Theorem 3.3 in Jin and Wang [7] to arbitrary number of patches where the respective limit system is strongly sublinear in the positive orthant and hence Corollary 3.2 in Zhao and Jing [23] can be applied directly.

Remark 3.3. The existence and global asymptotic stability of the positive equilibrium of system (3.4) can also be proved in a manner similar to the proof for Theorem 2 in Cosner et al. [1]. Clearly, our result is a generalization of Theorem 3.1 in Salmani and van den Driessche [11] where two patches are concerned and there is no impact of media coverage ($b_i = 0$ for $i = 1,2$).

Remark 3.4. The endemic equilibrium E^* for (3.1) is also linearly stable. To prove this, it is equivalent to consider the stability of the Jacobian matrix of system (3.3) at $\mathcal{E}^*$, i.e.,

$$J(\mathcal{E}^*) = \begin{pmatrix} ((-d_i)\delta_{ij} + m_{ij})_{p\times p} & 0_{p\times p} \\ \left(\beta_i(I_i^*)(I_i^*/N_i^*)^2\delta_{ij}\right)_{p\times p} & M' \end{pmatrix},$$

where $M' = \left(\left(\beta_i'(I_i^*)\frac{N_i^* - I_i^*}{N_i^*}I_i^* + \beta_i(I_i^*)\frac{N_i^* - 2I_i^*}{N_i^*} - (d_i+\gamma_i)\right)\delta_{ij} + m_{ij}\right)_{p\times p}$. Obviously, $s(((-d_i)\delta_{ij} + m_{ij})_{p\times p}) < 0$. Meanwhile, $s(M') < 0$ is proved by observing that $s(M^*) = 0$ and

$$M' - M^* = \left(\left(\beta_i'(I_i^*)\frac{N_i^* - I_i^*}{N_i^*}I_i^* - \beta_i(I_i^*)\frac{I_i^*}{N_i^*}\right)\delta_{ij}\right)_{p\times p} < 0.$$

Consequently, all eigenvalues of $J(\mathcal{E}^*)$ have negative real parts.

A combination of Theorems 2.4 and 3.1 yields a complete description of the dynamics of system (3.1) as follows.

Corollary 3.5. *For model (3.1), the disease-free equilibrium E_0 is globally asymptotically stable if $\mathcal{R}_0 \leqslant 1$, and the endemic equilibrium E^* exists and is globally asymptotically stable on the non-negative orthant minus the disease-free state if $\mathcal{R}_0 > 1$.*

Remark 3.6. The media coverage has no influence on the dynamics of disease transmission of system (3.1). However, the final infected size in each patch can be strictly reduced with more media coverage when the disease persists (i.e., $\mathcal{R}_0 > 1$). Such media-induced reduction was demonstrated in Sun et al. [16] by numerical simulations. In fact, this is equivalent to say that $\partial I_i^*/\partial b_j < 0$ for $i,j = 1,2,\ldots,p$. Since $\left(I_1^*, I_2^*, \ldots, I_p^*\right)$ is the unique positive solution of the following equations

$$\beta_i(I_i)\frac{N_i^* - I_i}{N_i^*}I_i - (d_i+\gamma_i)I_i + \sum_{j=1}^{p} m_{ij}I_j = 0, \quad i = 1,2,\ldots,p, \tag{3.5}$$

it follows from the implicit function theorem that $\partial I_i^*/\partial b_j$ exists. We consider without loss of generality the sign of $\partial I_i^*/\partial b_1$. Taking partial derivatives of (3.5) with respect to b_1 gives

$$M'\left(\frac{\partial I_1^*}{\partial b_1}, \frac{\partial I_2^*}{\partial b_1}, \ldots, \frac{\partial I_p^*}{\partial b_1}\right)^T = \left(f_1(I_1^*)\frac{N_1^* - I_1^*}{N_1^*}I_1^*, 0, \ldots, 0\right)^T,$$

where M' is defined in Remark 3.4. Note that M' has a negative inverse, thus $\partial I_i^*/\partial b_1 < 0$ for $i = 1,2,\ldots,p$.

When $\mathcal{R}_0 > 1$ for system (1.2), the following result shows that the existence, uniqueness and global attractivity of the endemic equilibrium still hold if the disease has mild effect on the travel of infectious humans (i.e., $n_{ij} \approx m_{ij}$ for $i,j = 1,2,\ldots,p$) and the disease-induced death is seldom (i.e., $v_i \approx 0$ for $i = 1,2,\ldots,p$). We omit the proof which is similar to that of Theorem 3.4 in Jin and Wang [7] by applying Theorem 4.3 and Remark 4.2 in Smith and Zhao [15] and Corollary 2.3 in Smith and Waltman [14].

Theorem 3.7. *Let $P = (m_{ij})_{p\times p}$ and $Q = (n_{ij})_{p\times p}$ be the travel rate matrices for the susceptible and infectious classes, respectively, and $\vec{v} = (v_1, v_2, \ldots, v_p)$ be the vector formed by the disease-induced death rates. Assume that all parameters in (1.2) are fixed except n_{ij} for $i,j = 1,2,\ldots,p$ and v_i for $i = 1,2,\ldots,p$, and $\mathcal{R}_0 > 1$ when $Q = P$ and $\vec{v} = 0_{1\times p}$. Then there is a $\tau > 0$ such that for any Q and $\vec{v}$ with $\|Q - P\| < \tau$ and $\|\vec{v}\| < \tau$, (1.2) has a unique endemic equilibrium $E^*(Q,\vec{v})$, which is globally attractive with respect to $\mathbb{R}_+^p \times \mathbb{R}_+^p \setminus \{0\}$. Here $\|\cdot\|$ is the Frobenius norm if '$\cdot$' is a matrix and the Euclidean norm if '$\cdot$' is a vector.*

We end this section with a result on the number of endemic equilibria for a special case of the two-patch model:

$$\begin{aligned} \frac{dN_i}{dt} &= A_i - d_iN_i - v_iI_i - m_{ji}(N_i - I_i) + m_{ij}(N_j - I_j), \quad i,j = 1,2,\ i \neq j, \\ \frac{dI_i}{dt} &= \beta_i(I_i)\frac{N_i - I_i}{N_i}I_i - (d_i + v_i + \gamma_i)I_i, \quad i,j = 1,2,\ i \neq j. \end{aligned} \tag{3.6}$$

Namely, when the infectious individuals in each patch do not travel to the other patch, we cannot obtain multiple endemic equilibria by

choosing suitable saturation functions and parameter values, which is different from the two-patch model in Jin and Wang [7].

Theorem 3.8. *System (3.6) has at most one endemic equilibrium if $m_{12} \geqslant 0$ and $m_{21} \geqslant 0$.*

Proof. Assume that $E^* = (N_1^*, N_2^*, I_1^*, I_2^*)$ and $\widehat{E} = (\widehat{N}_1, \widehat{N}_2, \widehat{I}_1, \widehat{I}_2)$ are two distinct positive equilibria of (3.6). Then they must satisfy the following four equations

$$A_i - d_i N_i - v_i I_i - m_{ji}(N_i - I_i) + m_{ij}(N_j - I_j) = 0, \quad i,j = 1,2,\ i \neq j,$$
$$\beta_i(I_i)\frac{N_i - I_i}{N_i} - (d_i + v_i + \gamma_i) = 0, \quad i,j = 1,2,\ i \neq j.$$

Solving N_1, N_2 in terms of I_1, I_2 from the first two and the last two equations gives

$$N_i = ((d_j + m_{ij})(A_i - (v_i - m_{ji})I_i - m_{ij}I_j) + m_{ij}(A_j - m_{ji}I_i - (v_j - m_{ij})I_j))/\Delta \tag{3.7}$$

and

$$N_i = I_i/(1 - \Lambda_i/\beta_i(I_i)), \tag{3.8}$$

respectively, where $\Lambda_i = d_i + v_i + \gamma_i$, $i,j = 1,2, i \neq j$ and $\Delta = (d_1 + m_{21})(d_2 + m_{12}) - m_{12}m_{21} > 0$. Thus, I_j can be solved in terms of I_i from (3.7) and (3.8) as follows:

$$(d_j + v_j)m_{ij}I_j = (d_j + m_{ij})A_i + m_{ij}A_j - ((d_j + m_{ij})v_i - d_j m_{ji})I_i - \frac{I_i\Delta}{1 - \Lambda_i/\beta_i(I_i)} \tag{3.9}$$

for $i,j = 1,2$ and $i \neq j$, or

$$I_j = \frac{(d_j + m_{ij})A_i + m_{ij}A_j - ((d_j + m_{ij})v_i - d_j m_{ji})I_i - I_i\Delta/(1 - \Lambda_i/\beta_i(I_i))}{(d_j + v_j)m_{ij}} \tag{3.10}$$

if $m_{ij} \neq 0$. Next the proof is naturally divided into three cases.

Case 1. $m_{12} > 0, m_{21} > 0$. Note that $I_i^* \neq \widehat{I}_i$ for $i = 1,2$, since otherwise it follows from (3.8) and (3.10) that $E^* = \widehat{E}$. For $i = 1,2, N_i^* > 0$ and $\widehat{N}_i > 0$ imply that $\beta_i(I_i) > \Lambda_i$, $I_i \in [\min\{I_i^*, \widehat{I}_i\}, \max\{I_i^*, \widehat{I}_i\}]$. We differentiate the right hand side of (3.10), denoted by $g_i(I_i)$, with respect to $I_i \in \left[\min\left\{I_i^*, \widehat{I}_i\right\}, \max\left\{I_i^*, \widehat{I}_i\right\}\right]$ and obtain

$$\begin{aligned}\frac{dg_i(I_i)}{dI_i} &= \frac{d_j m_{ji} - (d_j + m_{ij})v_i}{(d_j + v_j)m_{ij}} \\ &\quad - \frac{\Delta}{(d_j + v_j)m_{ij}}\left(\frac{\beta_i(I_i)}{\beta_i(I_i) - \Lambda_i} - \frac{I_i\beta_i'(I_i)\Lambda_i}{(\beta_i(I_i) - \Lambda_i)^2}\right) \\ &\leqslant \frac{d_j m_{ji} - (d_j + m_{ij})v_i - \Delta}{(d_j + v_j)m_{ij}} = \Theta_i < 0, \quad i,j \\ &= 1,2, \text{ and } i \neq j.\end{aligned}$$

Direct algebraic manipulations yield

$$\begin{aligned}&(d_1 m_{12} - (d_1 + m_{21})v_2 - \Delta)(d_2 m_{21} - (d_2 + m_{12})v_1 - \Delta) \\ &\quad - (d_1 + v_1)(d_2 + v_2)m_{12}m_{21} \\ &\quad = (d_1 + v_1)(d_2 + v_2)(d_1 d_2 + d_1 m_{12} + d_2 m_{21}) \\ &\quad = (d_1 + v_1)(d_2 + v_2)\Delta > 0,\end{aligned}$$

which is equivalent to $\Theta_1 \cdot \Theta_2 > 1$. Without loss of generality, let $I_1^* < \widehat{I}_1$. Hence,

$$\frac{dg_1(I_1)}{dI_1}\frac{dg_2(I_2)}{dI_2} > 1 \Rightarrow \frac{dg_1(I_1)}{dI_1} < \left(\frac{dg_2(I_2)}{dI_2}\right)^{-1} < 0, I_1 \in [I_1^*, \widehat{I}_1].$$

This means that in the I_1I_2-plane, after the point (I_1^*, I_2^*), the curve of $I_2 = g_1(I_1)$ is below the curve of $I_1 = g_2(I_2)$. So the two curves cannot intersect again at $(\widehat{I}_1, \widehat{I}_2)$.

Case 2. $m_{12} > 0$ and $m_{21} = 0$, or $m_{12} = 0$ and $m_{21} > 0$. It suffices to prove the result under the first condition. The negativity of the derivative of the right side of (3.9) with respect to I_2 ($i = 2, j = 1$) means that $I_2^* = \widehat{I}_2$. Once again, the negativity of the derivative of the right side of (3.9) with respect to I_1 ($i = 1, j = 2$) means that $I_1^* = \widehat{I}_1$. It follows (3.8) that $E^* = \widehat{E}$, which is a contradiction.

Case 3. $m_{12} = m_{21} = 0$. The negativity of the derivative of the right side of (3.9) with respect to I_i means that $I_i^* = \widehat{I}_i$ for $i = 1,2$. So $E^* = \widehat{E}$, a contradiction. □

Remark 3.9. An elementary but lengthy argument shows that system (3.6) can have up to four biologically meaningful equilibria in $\mathbb{R}_+^4$ if $m_{12} \geqslant 0$ and $m_{21} \geqslant 0$, that is, the DFE E_0, two one-patch disease-free steady states, and the endemic equilibrium. This is the same as the classic endemic model with $b_i = 0$, $i = 1,2$ (see [11,2]).

4. Examples and discussion

As mentioned earlier, the media effect alone cannot drive an endemic disease extinct, but it plays a significant role in reducing the number of infectives and its proportion to the total population. To investigate this, we carry out a numerical example for the two-patch model.

Consider the saturation functions $f_i(I_i) = 1 - k_i/(k_i + I_i)$ for $i = 1,2$ with $k_1 = 30$ and $k_2 = 50$, and take parameters in system (1.2) as follows: $A_1 = 20$, $a_1 = 0.10$, $d_1 = 3.6 \times 10^{-5}$, $v_1 = 0.02$, $\gamma_1 = 0.09$, $A_2 = 15$, $a_2 = 0.22$, $d_2 = 4.0 \times 10^{-5}$, $v_2 = 0.05$, $\gamma_2 = 0.05$, $b_2 = 0.11$. For these parameter values, the respective basic reproduction numbers for both patches are $\mathcal{R}_0^{(1)} = 0.9088 < 1$ and $\mathcal{R}_0^{(2)} = 2.1991 > 1$. If the two patches are disconnected, the disease eventually dies out in patch 1 while it persists in patch 2.

We fix the travel rates by letting $m_{12} = 0.10$, $m_{21} = 0.08$, $n_{12} = 0.08$, $n_{21} = 0.06$, thus $\mathcal{R}_0 = 1.6208$. Therefore, the disease becomes endemic in both patches and there exists an endemic equilibria. If we let b_1 vary from 0 to 0.05, the curves of the final sized infectives I_1^* and I_2^* against b_1 are depicted in Fig. 1(a). Here numerical calculations indicate that the endemic equilibrium is unique for each $b_1 \in [0, 0.05]$ and is locally stable. Both I_1^* and I_2^* are strictly decreasing with respect to b_1 which means stronger media coverage in patch 1 is beneficial to individuals in both patches.

If we keep all parameter values unchanged except that $v_2 = 0.03$, $n_{12} = 0.04$ and $n_{21} = 0.02$, then Fig. 1(b) shows how I_1^* and I_2^* vary with b_1 from 0 to 0.05. Here I_1^* is decreasing in b_1 but I_2^* is increasing in b_1. However, their proportions to the total population in each patch are strictly decreasing. Basically, appropriate media alert is helpful to disease control.

In this paper, we proposed a multi-patch model to study the influence of media coverage and human movement on disease transmission. Our results show that the basic reproduction number $\mathcal{R}_0$ is a threshold parameter of the disease dynamics. Particularly, either all positive solutions approach the disease-free equilibrium ($\mathcal{R}_0 \leqslant 1$) or a unique endemic equilibrium ($\mathcal{R}_0 > 1$) provided that the disease is non-fatal and susceptible and infectious individuals have the same travel rates. There are some unanswered questions with our model. For example, the non-existence of multiple endemic equilibria is unclear even for $p = 2$. Can the model exhibit more complicated dynamical behaviors like Hopf bifurcation? Is there a possibility that media coverage has negative effect on controlling of infectious diseases?

We can generalize the current model in many aspects. A more realistic model should include the impact of media on the dispersal

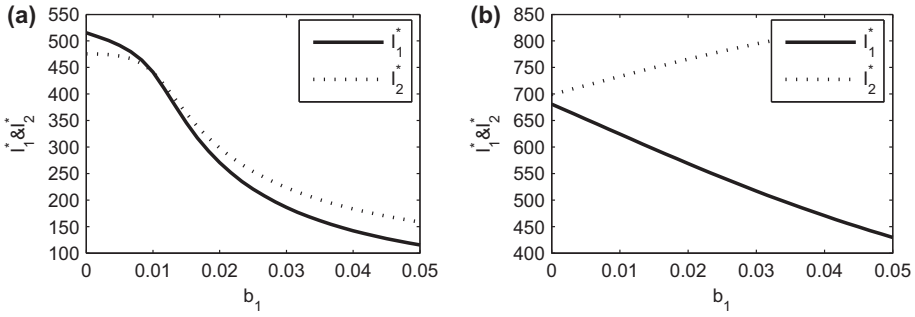

Fig. 1. The dependence of I_1^* and I_2^* on b_1.

rates. Sometimes it is better to consider the transmission coefficient as a function of the ratio I_i/N_i in patch i. There is a difference between the time when data is collected and the time when audiences get to know it, so it may be reasonable to consider a system of delay differential equations. One can also incorporate media effect in other ways such as that in Mummert and Weiss [10].

Acknowledgment

The authors thank Chris Cosner, Long Zhang and Xiao-Qiang Zhao for helpful discussions on this project.

References

[1] C. Cosner, J.C. Beier, R.S. Cantrell, D. Impoinvil, L. Kapitanski, M.D. Potts, A. Troyo, S. Ruan, The effects of human movement on the persistence of vector-borne diseases, J. Theor. Biol. 258 (2009) 550.
[2] J. Cui, Y. Takeuchi, Y. Saito, Spreading disease with transport-related infection, J. Theor. Biol. 239 (2006) 376.
[3] J. Cui, X. Tao, H. Zhu, A SIS infection model incorporating media coverage, Rocky Mount. J. Math. 38 (2008) 1323.
[4] D. Gao, S. Ruan, A multi-patch malaria model with logistic growth populations, submitted for publication.
[5] D. Greenhalgh, Hopf bifurcation in epidemic models with a latent period and nonpermanent immunity, Math. Comput. Model. 25 (2) (1997) 85.
[6] K.P. Hadeler, H.R. Thieme, Monotone dependence of the spectral bound on the transition rates in linear compartmental models, J. Math. Biol. 57 (2008) 697.
[7] Y. Jin, W. Wang, The effect of population dispersal on the spread of a disease, J. Math. Anal. Appl. 308 (2005) 343.
[8] J.C. Kamgang, G. Sallet, Computation of threshold conditions for epidemiological models and global stability of the disease-free equilibrium (DFE), Math. Biosci. 213 (2008) 1.
[9] R. Liu, J. Wu, H. Zhu, Media/psychological impact on multiple outbreaks of emerging infectious diseases, Comput. Math. Methods Med. 8 (2007) 153.
[10] A. Mummert, H. Weiss, Get the news out loudly and quickly: modeling the influence of the media on limiting infectious disease outbreaks, 2010. Available from: <arXiv:1006.5028v2>.
[11] M. Salmani, P. van den Driessche, A model for disease transmission in a patchy environment, Discrete Contin. Dyn. Syst. Ser. B 6 (2006) 185.
[12] P. Seibert, R. Suarez, Global stabilization of nonlinear cascade systems, Syst. Control Lett. 14 (1990) 347.
[13] H.L. Smith, Monotone Dynamical Systems: An Introduction to the theory of Competitive and Cooperative Systems, Mathematical Surveys and Monographs, vol. 41, AMS, Providence, RI, 1995.
[14] H.L. Smith, P. Waltman, Perturbation of a globally stable steady state, Proc. Amer. Math. Soc. 127 (1999) 447.
[15] H.L. Smith, X.-Q. Zhao, Dynamics of a periodically pulsed bio-reactor model, J. Differ. Equat. 155 (1999) 368.
[16] C. Sun, Y. Wei, J. Arino, K. Khan, Effect of media-induced social distancing on disease transmission in a two patch setting, Math. Biosci. 230 (2011) 87.
[17] H.R. Thieme, Persistence under relaxed point-dissipativity (with application to an endemic model), SIAM J. Math. Anal. 24 (1993) 407.
[18] P. van den Driessche, J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci. 180 (2002) 29.
[19] M. Vidyasagar, Decomposition techniques for large-scale systems with nonadditive interactions: stability and stabilizability, IEEE Trans. Automat. Control 25 (1980) 773.
[20] W. Wang, G. Mulone, Threshold of disease transmission in a patch environment, J. Math. Anal. Appl. 285 (2003) 321.
[21] W. Wang, X.-Q. Zhao, An epidemic model in a patchy environment, Math. Biosci. 190 (2004) 97.
[22] X.-Q. Zhao, Uniform persistence and periodic coexistence states in infinite-dimensional periodic semiflows with applications, Can. Appl. Math. Quart. 3 (1995) 473.
[23] X.-Q. Zhao, Z.-J. Jing, Global asymptotic behavior in some cooperative systems of functional differential equations, Can. Appl. Math. Quart. 4 (1996) 421.
[24] X.-Q. Zhao, Dynamical Systems in Population Biology, Springer, New York, 2003.
[25] Z. Zhang, T. Ding, W. Huang, Z. Dong, Qualitative Theory of Differential Equations, vol. 101, AMS, Providence, RI, 1992.