

Modeling direct and indirect disease transmission using multi-group model



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ABSTRACT

The survival of pathogens outside the host in the environment is an important factor for some diseases transmission. Environment-to-individuals is an indirect mode of transmission, which also plays a role in the dynamics of some disease. In this paper, we proposed a general multi-group epidemic model with nonlinear direct and indirect transmission incidence rates, nonlinear pathogen shedding rates, and common environmental contamination for indirect transmission. Under the certain assumptions, the basic reproduction number of the model is identified. We proved the global stability of the equilibria by using global Lyapunov functions with the specific coefficients and graph-theoretical approach theorem, which are determined by the basic reproduction number. The main result of our model is that we give the specific coefficients of global Lyapunov functions. From the discussion of our model, we conclude that our model contains earlier cholera models, brucellosis models, and other general disease multi-group models as special cases.

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1. Introduction

Infectious disease is a serious public health problem in some areas of the world, such as influenza [18,49], malaria [19], dengue [20], cholera [30], hantavirus [16], brucellosis [4], foot-and-mouth disease virus [38], infectious canine hepatitis virus [46], childhood diseases measles and rubella [7]. Some of these diseases are spread by pathogens outside the host, and the survival of pathogens outside the host is highly important for the epidemiological dynamics of brucellosis [1], cholera [3], hantavirus [17], infectious canine hepatitis virus [46], and influenza [49]. Mathematical modeling suggested that an indirect mode of transmission via

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the natural environment (e.g. respiratory droplets, fomites, fungal spores, waterborne pathogens) was an epidemic pattern for disease transmission [31]. This mechanism is in contrast to direct transmission which postulates that the pathogen is acquired through an infectious contact with an infected individual. For diseases transmitted through indirect pathways with the pathogens to survive outside of its host has also been used to play a vital role in the transmission dynamics process of hantavirus [17,31], cholera [3,8,27,28,32,39,40] and brucellosis [1,13,14,25,26]. Most of these studies are focused on multiple transmission pathways which involve both direct and indirect modes. Mukandavire et al. [27,28] introduced a dynamic model of cholera in Zimbabwe, the direct transmission (fast transmission pathway) is bilinear incidence and the indirect transmission (slow transmission pathway) is saturating incidence. Eisenberga et al. [8] proposed a cholera model in a patchy environment with water and human movement, Hou et al. [14] represented a brucellosis model for animal diseases, Shuai and van den Driessche [32] studied a cholera model with differential infectivity, the direct and indirect transmission of these three models are nonlinear incidence. Under the reasonable assumption of nonlinear incidence, the global stability of endemic equilibrium is given when basic reproduction number is larger than one.

In reality, due to the heterogeneity in host population, the host population can be divided into multiple groups. Multi-group model is a class of highly heterogeneous models with complex interactions among distinct groups corresponding to different infection source targets for specific purpose in studying disease transmission dynamics. Its applications became widely noticed in literature since 1970s [12,21], but large scale utilizations are just recently due to its mathematical breakthrough and surging computing capacity for simulations. In recently years, many multi-group models have been introduced to describe the transmission dynamics of infectious diseases [10,11,24,35,36,48]. The difficulty of global dynamics of multi-group models lies in establishing uniqueness and global stability of endemic equilibrium when basic reproduction number is larger than one. Guo et al. [10,11] firstly proved the global stability of endemic equilibrium of multi-group models with bilinear incidence of cross infection by using global Lyapunov functions and graph-theoretical approach. Li and Shuai [24] further made the proof more integrated to simpler formulas and became a standard procedure. Afterwards, a series of papers involving multi-group models utilized the methods to establish global dynamics as shown in reference [35,36,48]. However, most of the modeling work focused on analyzing the spread of infectious diseases uses a direct transmission mechanism, and there are few studies to analyze the multi-group dynamic model which incorporates both direct and indirect transmission. In paper [25], Li et al. proposed a multi-group brucellosis dynamical model for bilinear direct and indirect transmission incidence with different environmental contamination between cattle and sheep in the public farm, and the global dynamic behavior of the model was also given. In paper [26], Li et al. studied a multi-group sheep brucellosis model with common environmental contamination, but they could not give the global stability of the endemic equilibrium. Hence, taking an example of brucellosis, we want to study the global dynamic behavior of general multi-group *SEI-W* type model for the transmission of brucellosis that involves both nonlinear direct and indirect transmission, and common environmental contamination with indirect bacteria transmission for different groups. For our model, general forms for direct and indirect transmission include mass action, saturating incidence and other incidence. Our model contained earlier cholera models in [3,27,28,39,40] and brucellosis models in [1,13,25,26] as special cases. We proved the global stability of the equilibria by using global Lyapunov functions and graph-theoretical approach theorem, which are determined by the basic reproduction number. Our conclusions also contained the global stability of endemic equilibrium which was not addressed in previous paper [26]. The proofs of the main results of our paper exploited the method of constructing Lyapunov functions and a graph-theoretical technique in estimating the derivatives of Lyapunov functions, and also improved the related works in [15,42,43]. In this paper we gave the specific coefficients of global Lyapunov functions which are absent from previous papers [10,11,24,32,35,36,45,48].

The organization of this paper is as follows. In Section 2, we construct a multi-group brucellosis model with common environmental contamination of bacteria for different groups, and give some dynamic analysis

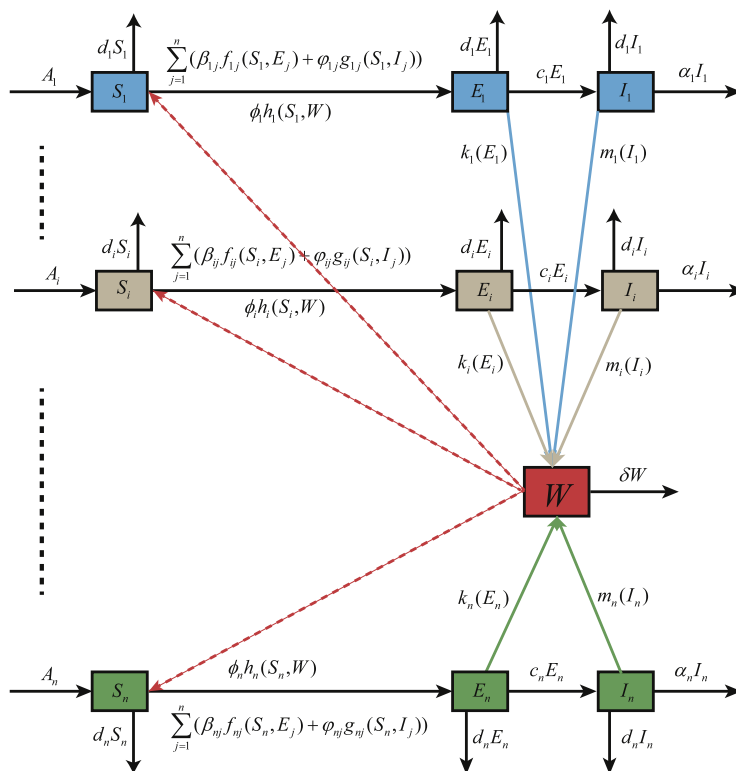


Fig. 1. Transmission diagram on the dynamical transmission of brucellosis with common environmental contamination in public farm.

on the disease-free equilibrium and the endemic equilibrium. Some specific examples are given in Section 3 and some conclusions are included in Section 4.

2. Main model and the global dynamical behavior

Brucellosis is an infectious and contagious bacterial disease caused by members of the genus *Brucella* [26]. Brucellosis can transmit to other individuals through directed contacting with infected individuals or indirect transmission by *Brucella* in the environment [25]. Here, we study more general forms for direct and indirect transmission of multi-group brucellosis dynamical model with common environmental contamination, and give the global dynamic behavior of the equilibrium. Suppose the host populations are divided into n homogeneous groups, each group i is further compartmentalized into S_i , E_i , and I_i , which denote the subpopulations that are susceptible, exposed, and infectious to the disease, respectively. Let W denote the number of pathogens in the common environment, which are shed by the exposed (E_i) and infected (I_i) individuals in group i . We define $k_i(E_i)$ and $m_i(I_i)$ as the pathogen shedding rate of the exposed and infected individuals in group i , respectively. $\sum_{i=1}^n (k_i(E_i) + m_i(I_i))$ denotes the pathogen shedding rate by individuals in E_1, \dots, E_n and I_1, \dots, I_n . The nonlinear coupling term $\beta_{ij} f_{ij}(S_i, E_j)$ and $\varphi_{ij} g_{ij}(S_i, I_j)$ represent the direct cross infection from the exposed individual and infectious individual of group j to the susceptible individual of group i . The nonlinear coupling term $\phi_i h_i(S_i, W)$ represents the indirect transmission from pathogen W to the susceptible individual of group i . Based on the characteristics of the spread of brucellosis with our previous model [26], the dynamical transmission of brucellosis with common environmental contamination in public farm are demonstrated in the following flowchart (see Fig. 1). Hence, the multi-group brucellosis model with common environmental contamination in public farm is described as the following ordinary differential equations:

$$\begin{cases} \frac{dS_i}{dt} = A_i - d_i S_i - \sum_{j=1}^n (\beta_{ij} f_{ij}(S_i, E_j) + \varphi_{ij} g_{ij}(S_i, I_j)) - \phi_i h_i(S_i, W), \\ \frac{dE_i}{dt} = \sum_{j=1}^n (\beta_{ij} f_{ij}(S_i, E_j) + \varphi_{ij} g_{ij}(S_i, I_j)) + \phi_i h_i(S_i, W) - (d_i + c_i) E_i, \\ \frac{dI_i}{dt} = c_i E_i - (d_i + \alpha_i) I_i, \\ \frac{dW}{dt} = \sum_{i=1}^n (k_i(E_i) + m_i(I_i)) - \delta W. \end{cases} \quad i = 1, 2, \dots, n. \quad (1)$$

The model describes the spread of an infectious disease in a heterogeneous population and incorporates both within the i -th group and inter-group direct/indirect transmission. The multi-group model is also suitable for investigating the spatial spread of waterborne diseases such as cholera and hemorrhagic fever with renal syndrome (HFRS). In group i , A_i denotes the recruitment individuals of susceptible compartment S_i , d_i describes natural mortality rate of individuals, and α_i represents mortality rate due to the disease, δ is removal rate of pathogen, and c_i is clinical outbreak rate from exposed compartment to infectious compartment. β_{ij} and φ_{ij} describe the direct transmission rate from the exposed individual and infectious individual of group j to the susceptible individual of group i , respectively. ϕ_i denotes the indirect transmission rate from pathogen W to the susceptible individual of group i . All parameters in (1) are nonnegative constants.

System (1) also has one disease-free equilibrium $P_0 = (S_1^0, 0, 0, \dots, S_n^0, 0, 0, 0)$, where $S_i^0 = \frac{A_i}{d_i}$. For each group i , adding the three equations in (1) gives

$$(S_i + E_i + I_i)' = A_i - d_i(S_i + E_i + I_i) - \alpha_i I_i \leq A_i - d_i(S_i + E_i + I_i).$$

Then it follows that

$$\lim_{t \rightarrow \infty} \sup (S_i + E_i + I_i) \leq \frac{A_i}{d_i}, \quad \lim_{t \rightarrow \infty} \sup W \leq \frac{\sum_{i=1}^n \left(k_i\left(\frac{A_i}{d_i}\right) + m_i\left(\frac{A_i}{d_i}\right) \right)}{\delta}.$$

Therefore, omega limit sets of system (1) are contained in the following bounded region in the non-negative cone of \mathbb{R}^{3n+1} :

$$X = \left\{ (S_i, E_i, I_i, W) \in \mathbb{R}_+^{3n+1} \mid 0 \leq (S_i + E_i + I_i) \leq \frac{A_i}{d_i}, 0 \leq W \leq \frac{\sum_{i=1}^n \left(k_i\left(\frac{A_i}{d_i}\right) + m_i\left(\frac{A_i}{d_i}\right) \right)}{\delta}, i = 1, 2, \dots, n \right\}.$$

It can be verified that region X is positively invariant with respect to system (1).

Based on biological considerations, we can also assume that $f_{ij}(0, E_j) = f_{ij}(S_i, 0) = 0$, $g_{ij}(0, I_j) = g_{ij}(S_i, 0) = 0$, $h_i(0, W) = h_i(S_i, 0) = 0$ and $f_{ij}(S_i, E_j) > 0$, $g_{ij}(S_i, I_j) > 0$, $h_i(S_i, W) > 0$ for $S_i, E_j, I_j, W > 0$. We also assume that $f_{ij}(S_i, E_j)$, $g_{ij}(S_i, I_j)$ and $h_i(S_i, W)$ are sufficiently smooth. We assume the basic assumptions on functions $f_{ij}(S_i, E_j)$, $g_{ij}(S_i, I_j)$, $h_i(S_i, W)$, $k_i(E_i)$ and $m_i(I_i)$ as follows:

(H1) Define $F_i(S_i) = \lim_{E_j \rightarrow 0^+} \frac{f_{ij}(S_i, E_j)}{E_j}$, $G_i(S_i) = \lim_{I_j \rightarrow 0^+} \frac{g_{ij}(S_i, I_j)}{I_j}$, $H_i(S_i) = \lim_{W \rightarrow 0^+} \frac{h_i(S_i, W)}{W}$. Then for $0 < S_i \leq \frac{A_i}{d_i}$, $0 < F_i(S_i)$, $G_i(S_i)$, $H_i(S_i) \leq \infty$.

(H2) $f_{ij}(S_i, E_j) \leq F_i(S_i) E_j$, $g_{ij}(S_i, I_j) \leq G_i(S_i) I_j$, $h_i(S_i, W) \leq H_i(S_i) W$ for all $E_i, I_i, W_i > 0$ and $0 < S_i \leq \frac{A_i}{d_i}$.

(H3) $F_i(S_i)$, $G_i(S_i)$ and $H_i(S_i)$ are monotone increasing for all $0 < S_i \leq \frac{A_i}{d_i}$.

(H4) $k_i(E_i)$, $m_i(I_i)$ only vanish at 0, which are also monotone nondecreasing, and $\frac{k_i(E_i)}{E_i}$, $\frac{m_i(I_i)}{I_i}$ are monotone nonincreasing for all $E_i, I_i > 0$, $i = 1, 2, \dots, n$, where $k'_i(E_i)$ and $m'_i(I_i)$ are the differential of $k_i(E_i)$ and $m_i(I_i)$, respectively.

Note that the class of $f_{ij}(S_i, E_j)$, $g_{ij}(S_i, I_j)$, $h_{ij}(S_i, W_j)$ satisfying (H1–H3) and $k_i(E_i)$, $m_i(I_i)$ satisfying (H4) include many common incidence functions, which is shown in Table 1.

Table 1

Parameters value in system (1).

Group	$f(S_i, E_j)$	$g(S_i, I_j)$	$h(S_i, W)$	$k_i(E_i) + m_i(I_i)$	Reference
2	$S_i E_j$	$S_i I_j$	$S_i W$	$k_i E_i + k_i I_i$	Li et al. [26]
1	0	SI	$\frac{SW}{b+W}$	kI	Mukandavire et al. [27,28]
1	0	$\frac{SI}{1+\alpha I^2}$	0	0	Xiao and Ruan [47]
1	0	$S^q I (q > 0)$	0	0	Wang et al. [44]
1	0	$\frac{SI}{b+\theta S}$	0	0	Anderson and May [2]
n	0	$f_i(S_i)g_j(I_j)$	$f_i(S_i)h(W)$	0	Shuai and van den Driessche [33]
n	0	$f_i(S_i)g_j(I_j)$	0	0	Yuan and Wang [48]
n	0	$S_i E_j$	$S_i I_j$	$g(\mathbf{E}, \mathbf{I})$	Wang and Cao [45]

2.1. The basic reproduction number

Now we derive the basic reproduction number R_0 of system (1), according to the concepts of next generation matrix and the basic reproduction number present in [5,6,41]. Here we define a new vector $x = (E_1, \dots, E_n, I_1, \dots, I_n, W)^T$, which only contains infected variables first by disease state. Considering the following auxiliary system:

$$\begin{cases} \frac{dE_i}{dt} = \sum_{j=1}^n (\beta_{ij} f_{ij}(S_i, E_j) + \varphi_{ij} g_{ij}(S_i, I_j)) + \phi_i h_i(S_i, W) - (d_i + c_i) E_i, \\ \frac{dI_i}{dt} = c_i E_i - (d_i + \alpha_i) I_i, \\ \frac{dW}{dt} = \sum_{i=1}^n (k_i(E_i) + m_i(I_i)) - \delta W. \end{cases} \quad i = 1, 2, \dots, n. \quad (2)$$

Assume that $f_{ij}(S_i, E_j), g_{ij}(S_i, I_j), h_i(S_i, W), k_i(E_i)$ and $m_i(I_i)$ satisfy (H1–H4). Following the recipe from van den Driessche and Watmough [41], it is also easy to obtain

$$F = \begin{pmatrix} \beta_{11} F_1(S_1^0) & \cdots & \beta_{1n} F_1(S_1^0) & \varphi_{11} G_1(S_1^0) & \cdots & \varphi_{1n} G_1(S_1^0) & \phi_1 H_1(S_1^0) \\ \vdots & & \vdots & \vdots & & \vdots & \vdots \\ \beta_{n1} F_n(S_n^0) & \cdots & \beta_{nn} F_n(S_n^0) & \varphi_{n1} G_n(S_n^0) & \cdots & \varphi_{nn} G_n(S_n^0) & \phi_n H_n(S_n^0) \\ 0 & \cdots & 0 & 0 & \cdots & 0 & 0 \\ \vdots & & \vdots & \vdots & & \vdots & \vdots \\ 0 & \cdots & 0 & 0 & \cdots & 0 & 0 \\ 0 & \cdots & 0 & 0 & \cdots & 0 & 0 \end{pmatrix}_{(2n+1) \times (2n+1)},$$

$$V = \begin{pmatrix} \text{diag}\{d_1 + c_1, \dots, d_n + c_n\} & 0 & 0 \\ \text{diag}\{c_1, \dots, c_n\} & \text{diag}\{d_1 + \alpha_1, \dots, d_n + \alpha_n\} & 0 \\ K_1 & K_2 & \delta \end{pmatrix}_{(2n+1) \times (2n+1)},$$

$$K_1 = \begin{pmatrix} -k'_1(0) & \cdots & -k'_n(0) \end{pmatrix}_{1 \times n}, K_2 = \begin{pmatrix} -m'_1(0) & \cdots & -m'_n(0) \end{pmatrix}_{1 \times n}.$$

The inverse of V equals

$$V^{-1} = \begin{pmatrix} \text{diag}\{\frac{1}{d_1+c_1}, \dots, \frac{1}{d_n+c_n}\} & 0 & 0 \\ \text{diag}\{\frac{c_1}{(d_1+c_1)(d_1+\alpha_1)}, \dots, \frac{c_n}{(d_n+c_n)(d_n+\alpha_n)}\} & \text{diag}\{\frac{1}{d_1+\alpha_1}, \dots, \frac{1}{d_n+\alpha_n}\} & 0 \\ K_3 & K_4 & \frac{1}{\delta} \end{pmatrix}_{(2n+1) \times (2n+1)},$$

$$K_3 = \begin{pmatrix} \frac{(d_1+\alpha_1)k'_1(0)+c_1m'_1(0)}{\delta(d_1+c_1)(d_1+\alpha_1)} & \cdots & \frac{(d_n+\alpha_n)k'_n(0)+c_nm'_n(0)}{\delta(d_n+c_n)(d_n+\alpha_n)} \end{pmatrix}_{1 \times n},$$

$$K_4 = \begin{pmatrix} \frac{c_1m'_1(0)}{\delta(d_1+c_1)(d_1+\alpha_1)} & \cdots & \frac{c_nm'_n(0)}{\delta(d_n+c_n)(d_n+\alpha_n)} \end{pmatrix}_{1 \times n}.$$

Then, the next-generation matrix is $C = FV^{-1}$, and the basic reproduction number R_0 of system (1) can be defined as the spectral radius of C (see [5,6,41]), that is

$$R_0 = \rho(FV^{-1}),$$

where

$$C = \left(\frac{\beta_{ij}F_{ij}(S_i^0)}{d_i + c_i} + \frac{\varphi_{ij}c_iG_{ij}(S_i^0)}{(d_i + c_i)(d_i + \alpha_i)} + \phi_iH_i(S_i^0) \frac{(d_i + \alpha_i)k'_i(0) + c_im'_i(0)}{\delta(d_i + c_i)(d_i + \alpha_i)} \right)_{1 \leq i, j \leq n}.$$

2.2. Global stability of the disease-free equilibrium of system (1)

In this subsection, we study the global stability of the equilibria. Firstly, we investigate the global stability of disease-free equilibrium when the basic reproduction number $R_0 \leq 1$. For the disease-free equilibrium P_0 of system (1), we have the following property.

Let $M = F - V$, and define $s(M) = \max\{\operatorname{Re}\lambda : \lambda \text{ is an eigenvalue of } M\}$ [34]. By the Theorem 2 of van den Driessche and Watmough [41], there hold two equivalences:

$$R_0 > 1 \Leftrightarrow s(M) > 0, R_0 < 1 \Leftrightarrow s(M) < 0.$$

Theorem 2.1. Suppose that assumptions (H1)–(H4) hold, then the following conclusions hold for system (1). The disease-free equilibrium P_0 of system (1) is globally asymptotically stable if $R_0 \leq 1$, and unstable if $R_0 > 1$.

Proof. To prove the local stability of disease-free equilibrium P_0 of system (1), all eigenvalues of the Jacobian matrix with system (1) at disease-free equilibrium P_0 have negative real parts. The Jacobian matrix is

$$J|_{P_0} = \begin{pmatrix} M & \mathbf{0} \\ J_1 & \Lambda \end{pmatrix}_{(3n+1) \times (3n+1)},$$

where $\Lambda = \operatorname{diag}\{-d_1, \dots, -d_n\}$, and

$$J_1 = - \begin{pmatrix} \beta_{11}F_1(S_1^0) & \cdots & \beta_{1n}F_1(S_1^0) & \varphi_{11}G_1(S_1^0) & \cdots & \varphi_{1n}G_1(S_1^0) & \phi_1H_1(S_1^0) \\ \vdots & & \vdots & \vdots & & \vdots & \vdots \\ \beta_{n1}F_n(S_n^0) & \cdots & \beta_{nn}F_n(S_n^0) & \varphi_{n1}G_n(S_n^0) & \cdots & \varphi_{nn}G_n(S_n^0) & \phi_nH_n(S_n^0) \end{pmatrix}_{n \times (2n+1)}.$$

The corresponding characteristic equation of $J|_{P_0}$ is

$$|\lambda \mathbf{E} - J|_{P_0}|_{(3n+1) \times (3n+1)} = (\lambda_1 + d_1) \cdots (\lambda_n + d_n) |\lambda \mathbf{I} - M|_{(2n+1) \times (2n+1)}.$$

When $s(M) < 0$, then all eigenvalues of the Jacobian matrix $J|_{P_0}$ have negative real parts. By the Theorem 2 of van den Driessche and Watmough [41], the disease-free equilibrium P_0 of system (1) is locally asymptotically stable when $R_0 < 1$. If $R_0 > 1$, then $s(M) > 0$ and $s(J|_{P_0}) > 0$, the disease-free equilibrium P_0 of system (1) is unstable.

Using assumptions (H2) and (H3), it is easy to obtain

$$f_{ij}(S_i, E_j) \leq F_i(S_i^0)E_j, g_{ij}(S_i, I_j) \leq G_i(S_i^0)I_j, h_i(S_i, W) \leq H_i(S_i^0)W.$$

One can obtain the following equation by using assumption (H4),

$$\frac{k_i(E_i)}{E_i} \leq \lim_{E_i \rightarrow 0} \frac{k_i(E_i)}{E_i} = \lim_{E_i \rightarrow 0} \frac{k_i(E_i) - k_i(0)}{E_i - 0} = k'_i(0),$$

which also means $k'_i(0)E_i \geq k_i(E_i)$. Similarity, we have $m'_i(0)I_i \geq m_i(I_i)$. Hence, system (2) will become the following system

$$\frac{dx}{dt} \leq (F - V)x. \quad (3)$$

Let $b \geq 0$ be the left eigenvector of the nonnegative matrix $V^{-1}F$ with respect to the eigenvalue $\rho(V^{-1}F) = R_0$, that is, $b^T V^{-1}F = R_0 b^T$. Define the Lyapunov function $L_1 = b^T V^{-1}x$. Then the derivative of L along the system (2) is

$$\frac{dL_1}{dt} = b^T V^{-1}x' \leq b^T V^{-1}(F - V)x = b^T V^{-1}Fx - b^T x \leq (R_0 - 1)b^T x.$$

If $R_0 < 1$, then $\frac{dL_1}{dt} \leq 0$. Let

$$\Psi = \{(S_i, E_i, I_i, W) \in X \mid \frac{dL_1}{dt} = 0, i = 1, 2, \dots, n\}.$$

If $R_0 < 1$, $\frac{dL_1}{dt} = 0$ implies that $b^T x = 0$, thus $E_i = 0, I_i = 0, W = 0, i = 1, 2, \dots, n$. Therefore, the largest invariant set of Ψ is the singleton P_0 when $R_0 < 1$.

If $R_0 = 1$, $\frac{dL_1}{dt} = 0$ implies that $f_{ij}(S_i, E_j) = F_i(S_i^0)E_j$, $g_{ij}(S_i, I_j) = G_i(S_i^0)I_j$, $h_i(S_i, W) = H_i(S_i^0)W$ for all $1 \leq i, j \leq n$. It follows from assumption (H3) that $S_i = S_i^0$ or $E_j = I_j = W = 0$ for all $j = 1, 2, \dots, n$. Therefore, it can be verified that the largest invariant set of Ψ where $\frac{dL_1}{dt} = 0$ is the singleton P_0 when $R_0 = 1$. By LaSalle's Invariance Principle [22], P_0 is globally asymptotically stable in the region X when $R_0 \leq 1$.

If $R_0 > 1$ and $x > 0$, it follows that

$$(R_0 - 1)b^T x > 0. \quad (4)$$

Hence, there must exist $\frac{dL_1}{dt} > 0$ in a small enough neighborhood of P_0 in the interior of X when $R_0 > 1$. Therefore, solutions in the interior of X sufficiently close to P_0 move away from P_0 , and which implies that P_0 is unstable. This completes the proof of Theorem 2.1.

2.3. The uniform persistence of system (1)

The following result shows that $R_0 > 1$ actually implies that system (1) admits at least one endemic equilibrium and the disease is uniformly persistent.

Theorem 2.2. *Let (H1)–(H4) hold and $R_0 > 1$. Then system (1) admits at least one (componentwise) positive equilibrium, and there is a positive constant ϵ such that every solution $(S_i(t), E_i(t), I_i(t), W(t))$ of system (1) with $(S_i(0), E_i(0), I_i(0), W(0)) \in \mathbb{R}_+^n \times \text{Int } \mathbb{R}_+^{2n+1}$ satisfies*

$$\min\{\liminf_{t \rightarrow \infty} E_i(t), \liminf_{t \rightarrow \infty} I_i(t), \liminf_{t \rightarrow \infty} W(t)\} \geq \epsilon, i = 1, 2, \dots, n.$$

Proof. We show first that

$$\limsup_{t \rightarrow \infty} \max_i \{E_i(t), I_i(t), W(t)\} > \epsilon, i = 1, 2, \dots, n. \quad (5)$$

Considering the following system

$$\frac{dS_i}{dt} = A_i - d_i S_i. \quad (6)$$

Using Corollary 3.2 in Zhao et al. [51], it then follows that system (6) has a unique positive equilibrium $(S_i^0, i = 1, 2, \dots, n)$ and which is globally asymptotically stable.

As to $R_0 > 1 \Leftrightarrow s(M) > 0$, choose $\varepsilon_1 > 0$ small enough such that $s(M - \varepsilon_1 M_0) > 0$, where

$$M_0 = \begin{pmatrix} \beta_{11} & \cdots & \beta_{1n} & \varphi_{11} & \cdots & \varphi_{1n} & \phi_1 \\ \vdots & & \vdots & \vdots & & \vdots & \vdots \\ \beta_{n1} & \cdots & \beta_{nn} & \varphi_{n1} & \cdots & \varphi_{nn} & \phi_n \\ 0 & \cdots & 0 & 0 & \cdots & 0 & 0 \\ \vdots & & \vdots & \vdots & & \vdots & \vdots \\ 0 & \cdots & 0 & 0 & \cdots & 0 & 0 \\ 0 & \cdots & 0 & 0 & \cdots & 0 & 0 \end{pmatrix}_{(2n+1) \times (2n+1)}.$$

Let us consider a perturbed system

$$\frac{dS_i}{dt} = A_i - d_i S_i - \varepsilon \left(F_i(S_i) \sum_{j=1}^n \beta_{ij} + G_i(S_i) \sum_{j=1}^n \varphi_{ij} + \phi_i H_i(S_i) \right). \quad (7)$$

From our previous analysis of system (6), we can restrict $\varepsilon > 0$ small enough such that (7) admits a unique positive equilibrium $(S_i^0(\varepsilon), i = 1, 2, \dots, n)$ which is globally asymptotically stable. Since $S_i^0(\varepsilon)$ is continuous in ε , so we can further restrict ε small enough such that $F_i(S_i^0(\varepsilon)) > F_i(S_i^0) - \varepsilon_1, G_i(S_i^0(\varepsilon)) > G_i(S_i^0) - \varepsilon_1, H_i(S_i^0(\varepsilon)) > H_i(S_i^0) - \varepsilon_1, i = 1, 2, \dots, n$.

For the sake of contradiction of (5), that there is a $T > 0$ such that $E_i(t) < \varepsilon, I_i(t) < \varepsilon, W(t) < \varepsilon, i = 1, 2, \dots, n$, for all $t \geq T$. Then for $t \geq T$, we have

$$\begin{aligned} \frac{dS_i}{dt} &= A_i - d_i S_i - \sum_{j=1}^n (\beta_{ij} f_{ij}(S_i, E_j) + \varphi_{ij} g_{ij}(S_i, I_j)) - \phi_i h_i(S_i, W) \\ &\geq A_i - d_i S_i - \left(F_i(S_i) \sum_{j=1}^n \beta_{ij} E_j + G_i(S_i) \sum_{j=1}^n \varphi_{ij} I_j + \phi_i H_i(S_i) W \right) \\ &\geq A_i - d_i S_i - \varepsilon \left(F_i(S_i) \sum_{j=1}^n \beta_{ij} + G_i(S_i) \sum_{j=1}^n \varphi_{ij} + \phi_i H_i(S_i) \right). \end{aligned}$$

Since the equilibrium $(S_i^0(\varepsilon), i = 1, 2, \dots, n)$ of (7) is globally asymptotically stable and $F_i(S_i^0(\varepsilon)) > F_i(S_i^0) - \varepsilon_1$. There is a $T_1 > T > 0$ such that $F_i(S_i(t)) \geq F_i(S_i^0) - \varepsilon_1$ for $t > T_1$. Further, there must be a $T_2 > T_1 > T > 0$ such that $f_{ij}(S_i, E_j) \geq (F_i(S_i^0) - \varepsilon_1) E_j$ for $t > T_2$. Similarly, we have $g_{ij}(S_i, I_j) \geq (G_i(S_i^0) - \varepsilon_1) I_j$ and $h_i(S_i, W) \geq (H_i(S_i^0) - \varepsilon_1) W$ for $t > T_2$. As a consequence, for $t > T_2$, there holds

$$\begin{cases} \frac{dE_i}{dt} \geq \sum_{j=1}^n (\beta_{ij} (F_i(S_i^0) - \varepsilon_1) E_j + \varphi_{ij} (G_i(S_i^0) - \varepsilon_1) I_j) + \phi_i (H_i(S_i^0) - \varepsilon_1) W - (d_i + c_i) E_i, \\ \frac{dI_i}{dt} = c_i E_i - (d_i + \alpha_i) I_i, \\ \frac{dW}{dt} = \sum_{i=1}^n (k_i(E_i) + m_i(I_i)) - \delta W. \end{cases} \quad i = 1, 2, \dots, n.$$

Considering the following system

$$\begin{cases} \frac{dE'_i}{dt} = \sum_{j=1}^n (\beta_{ij}(F_i(S_i^0) - \varepsilon_1)E'_j + \varphi_{ij}(G_i(S_i^0) - \varepsilon_1)I'_j) + \phi_i(H_i(S_i^0) - \varepsilon_1)W' - (d_i + c_i)E'_i, \\ \frac{dI'_i}{dt} = c_iE'_i - (d_i + \alpha_i)I'_i, \\ \frac{dW'}{dt} = \sum_{i=1}^n (k_i(E'_i) + m_i(I'_i)) - \delta W'. \end{cases} \quad i = 1, 2, \dots, n.$$

Since the matrix M_1 has positive eigenvalue $s(M_1)$ with a positive eigenvector. It is easy to see that $(E'_i(t), I'_i(t), W'(t)) \rightarrow (\infty, \infty, \infty)$ as $t \rightarrow \infty, i = 1, 2, \dots, n$. Using the comparison principle of Smith and Waltman [34], we also know that $(E_i(t), I_i(t), W(t)) \rightarrow (\infty, \infty, \infty)$ as $t \rightarrow \infty, i = 1, 2, \dots, n$. Which leads to a contradiction.

Define

$$X_0 = \{(S_i, E_i, I_i, W) \in X | E_i, I_i, W > 0, i = 1, 2, \dots, n\}, \partial X_0 = X | X_0.$$

The following will show that system (1) is uniformly persistent with respect to $(X_0, \partial X_0)$. By the form of system (1), it is easy to see that both X and X_0 are positively invariant and ∂X_0 is relatively closed in X . Furthermore system (1) is point dissipative. Let

$$M_\partial = \{(S_i(0), E_i(0), I_i(0), W(0)) | (S_i(t), E_i(t), I_i(t), W(t)) \in \partial X_0, \forall t \geq 0, i = 1, 2, \dots, n\}.$$

We claim that

$$M_\partial = \{(S_1(t), 0, 0, \dots, S_n(t), 0, 0, 0) | S_i(t) \geq 0, i = 1, 2, \dots, n\} \quad (8)$$

Noting that $\{(S_1(t), 0, 0, \dots, S_n(t), 0, 0, 0) | S_i(t) \geq 0, i = 1, 2, \dots, n\} \subseteq M_\partial$. We only need to prove $M_\partial \subseteq \{(S_1(t), 0, 0, \dots, S_n(t), 0, 0, 0) | S_i(t) \geq 0, i = 1, 2, \dots, n\}$. Assume $(S_i(0), E_i(0), I_i(0), W(0)) \in M_\partial, i = 1, 2, \dots, n$. It suffices to show that $E_i(t) = 0, I_i(t) = 0, W(t) = 0, \forall t \geq 0$. Suppose not. Then there exist an $i_0, 1 \leq i_0 \leq n$, and $t_0 \geq 0$ such that $E_{i_0}(t_0) > 0, I_{i_0}(t_0) = 0, W(t_0) = 0$. We partition $\{1, 2, \dots, n\}$ into two sets Q_1 and Q_2 such that

$$\begin{cases} E_i(t_0) = 0, I_i(t_0) = 0, W(t_0) = 0, \forall i \in Q_1 \\ E_i(t_0) > 0, I_i(t_0) = 0, W(t_0) = 0, \forall i \in Q_2 \end{cases}$$

Q_1 is non-empty due to the definition of M_∂ . Q_2 is non-empty since $E_{i_0}(t_0) > 0, I_{i_0}(t_0) = 0, W(t_0) = 0$. For any $i \in Q_2$, we have

$$\frac{dI_i(t_0)}{dt} = c_i E_i(t_0) - (d_i + \alpha_i) I_i(t_0) = c_i E_i(t_0).$$

It follows that there is an $\eta > 0$ small enough such that $I_i(t) > 0, i \in Q_2$ for $t_0 < t < t_0 + \eta$. This means that $(S_i(t), E_i(t), I_i(t), W(t))$ does not belong $\partial X_0, i = 1, 2, \dots, n$ for $t_0 < t < t_0 + \eta$, which contradicts the assumption that $(S_i(0), E_i(0), I_i(0), W(0)) \in M_\partial, i = 1, 2, \dots, n$. The claim (8) is proved.

As to P_0 is globally asymptotically stable for system (1). It is clear that there is only a equilibria P_0 in M_∂ , by afore-mentioned claim, it then follows that P_0 is isolated invariant set in X , $W^s(P_0) \cap X_0 = \emptyset$. Clearly, every orbit in M_∂ converges to P_0 , P_0 is acyclic in M_∂ . Using Theorem 4.6 in Thieme [37] or Theorem 1.3.1 and Remark 1.3.1 in Zhao [50], we conclude that the system (1) is uniformly persistent with respect to $(X_0, \partial X_0)$. This completes the proof of Theorem 2.2.

2.4. Global stability of the endemic equilibrium of system (1)

If $R_0 > 1$, then it follows from Theorem 2.2 that system (1) is uniformly persistent, together with the uniform boundedness of solutions of (1) in the interior of X , which implies that (1) admits at least one endemic equilibrium in the interior of X (see also in [9], Proposition 3.3 in [23], and Theorem D.3 in [34]). Let $P^* = (S_1^*, E_1^*, I_1^*, \dots, S_n^*, E_n^*, I_n^*, W^*)$ be a positive endemic equilibrium of system (1), we will show its global asymptotic stability in the interior of the feasible region X . Here $S_i^*, E_i^*, I_i^*, W^* > 0, i = 1, 2, \dots, n$ satisfies the following algebraic equations:

$$\begin{cases} A_i - d_i S_i^* - \sum_{j=1}^n (\beta_{ij} f_{ij}(S_i^*, E_j^*) + \varphi_{ij} g_{ij}(S_i^*, I_j^*)) - \phi_i h_i(S_i^*, W^*) = 0, \\ \sum_{j=1}^n (\beta_{ij} f_{ij}(S_i^*, E_j^*) + \varphi_{ij} g_{ij}(S_i^*, I_j^*)) + \phi_i h_i(S_i^*, W^*) - (d_i + c_i) E_i^* = 0, \\ c_i E_i^* - (d_i + \alpha_i) I_i^* = 0, \\ \sum_{i=1}^n (k_i(E_i^*) + m_i(I_i^*)) - \delta W^* = 0, \end{cases} \quad i = 1, 2, \dots, n.$$

Theorem 2.3. Suppose that assumptions (H1)–(H4) hold and matrix $[\beta_{ij}]_{1 \leq i, j \leq n}$ and $[\varphi_{ij}]_{1 \leq i, j \leq n}$ are irreducible. If $R_0 > 1$, and $f_{ij}(S_i, E_j), g_{ij}(S_i, I_j), h_i(S_i, W)$ satisfy the following conditions: for all $S_i, E_j, I_j, W > 0, 1 \leq i, j \leq n$,

$$\Theta_1 = \left(1 - \frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)}\right) (S_i^* - S_i) \leq 0, \quad (9)$$

$$\Theta_2 = \left(1 - \frac{E_j}{E_j^*} \frac{f_{ii}(S_i, E_i^*) f_{ij}(S_i^*, E_j^*)}{f_{ii}(S_i^*, E_i^*) f_{ij}(S_i, E_j)}\right) \left(\frac{f_{ii}(S_i^*, E_i^*) f_{ij}(S_i, E_j)}{f_{ii}(S_i, E_i^*) f_{ij}(S_i^*, E_j^*)} - 1\right) \leq 0, \quad (10)$$

$$\Theta_3 = \left(1 - \frac{I_j}{I_j^*} \frac{f_{ii}(S_i, E_i^*) g_{ij}(S_i^*, I_j^*)}{f_{ii}(S_i^*, E_i^*) g_{ij}(S_i, I_j)}\right) \left(\frac{f_{ii}(S_i^*, E_i^*) g_{ij}(S_i, I_j)}{f_{ii}(S_i, E_i^*) g_{ij}(S_i^*, I_j^*)} - 1\right) \leq 0, \quad (11)$$

$$\Theta_4 = \left(1 - \frac{W}{W^*} \frac{f_{ii}(S_i, E_i^*) h_i(S_i^*, W^*)}{f_{ii}(S_i^*, E_i^*) h_i(S_i, W)}\right) \left(\frac{f_{ii}(S_i^*, E_i^*) h_i(S_i, W)}{f_{ii}(S_i, E_i^*) h_i(S_i^*, W^*)} - 1\right) \leq 0, \quad (12)$$

then the endemic equilibrium P^* of system (1) is unique and globally asymptotically stable in the interior of X .

Proof. We prove that P^* is globally asymptotically stable in the interior of X , which also implies that the endemic equilibrium is unique. Now we consider the following equation:

$$V_{i1} = \int_{S_i^*}^{S_i} \frac{f_{ii}(\xi, E_i^*) - f_{ii}(S_i^*, E_i^*)}{f_{ii}(\xi, E_i^*)} d\xi + E_i - E_i^* - E_i^* \ln \frac{E_i}{E_i^*},$$

$$V_{i2} = I_i - I_i^* - I_i^* \ln \frac{I_i}{I_i^*}, V_3 = W - W^* - W^* \ln \frac{W}{W^*}.$$

For $V_{i1}, i = 1, 2, \dots, n$, differentiating and using the equilibrium equations give

$$\begin{aligned}
\frac{dV_{i1}}{dt} &= \left(1 - \frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)}\right) S_i' + \left(1 - \frac{E_i^*}{E_i}\right) E_i' \\
&= \left(1 - \frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)}\right) \left(A_i - d_i S_i - \left(\sum_{j=1}^n (\beta_{ij} f_{ij}(S_i, E_j) + \varphi_{ij} g_{ij}(S_i, I_j)) + \phi_i h_i(S_i, W)\right)\right) \\
&\quad + \left(1 - \frac{E_i^*}{E_i}\right) \left(\sum_{j=1}^n (\beta_{ij} f_{ij}(S_i, E_j) + \varphi_{ij} g_{ij}(S_i, I_j)) + \phi_i h_i(S_i, W) - (d_i + c_i) E_i\right) \\
&= \left(1 - \frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)}\right) (d_i(S_i^* - S_i) + \sum_{j=1}^n \beta_{ij} f_{ij}(S_i^*, E_j^*) \left(1 - \frac{f_{ij}(S_i, E_j)}{f_{ij}(S_i^*, E_j^*)}\right) \\
&\quad + \sum_{j=1}^n \varphi_{ij} g_{ij}(S_i^*, I_j^*) \left(1 - \frac{g_{ij}(S_i, I_j)}{g_{ij}(S_i^*, I_j^*)}\right) + \phi_i h_i(S_i^*, W^*) \left(1 - \frac{h_i(S_i, W)}{h_i(S_i^*, W^*)}\right)) \\
&\quad + \left(1 - \frac{E_i^*}{E_i}\right) \left(\sum_{j=1}^n \beta_{ij} f_{ij}(S_i^*, E_j^*) \left(\frac{f_{ij}(S_i, E_j)}{f_{ij}(S_i^*, E_j^*)} - \frac{E_i}{E_i^*}\right)\right. \\
&\quad + \sum_{j=1}^n \varphi_{ij} g_{ij}(S_i^*, I_j^*) \left(\frac{g_{ij}(S_i, I_j)}{g_{ij}(S_i^*, I_j^*)} - \frac{E_i}{E_i^*}\right) + \phi_i h_i(S_i^*, W^*) \left(\frac{h_i(S_i, W)}{h_i(S_i^*, W^*)} - \frac{E_i}{E_i^*}\right)) \\
&\leq \sum_{j=1}^n \beta_{ij} f_{ij}(S_i^*, E_j^*) \left(2 - \frac{E_i}{E_i^*} - \frac{E_i^* f_{ij}(S_i, E_j)}{E_i f_{ij}(S_i^*, E_j^*)} - \frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)} + \frac{f_{ii}(S_i^*, E_i^*) f_{ij}(S_i, E_j)}{f_{ii}(S_i, E_i^*) f_{ij}(S_i^*, E_j^*)}\right) \\
&\quad + \sum_{j=1}^n \varphi_{ij} g_{ij}(S_i^*, I_j^*) \left(2 - \frac{E_i}{E_i^*} - \frac{E_i^* g_{ij}(S_i, I_j)}{E_i g_{ij}(S_i^*, I_j^*)} - \frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)} + \frac{f_{ii}(S_i^*, E_i^*) g_{ij}(S_i, I_j)}{f_{ii}(S_i, E_i^*) g_{ij}(S_i^*, I_j^*)}\right) \\
&\quad + h_i(S_i^*, W^*) \left(2 - \frac{E_i}{E_i^*} - \frac{E_i^* h_i(S_i, W)}{E_i h_i(S_i^*, W^*)} - \frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)} + \frac{f_{ii}(S_i^*, E_i^*) h_i(S_i, W)}{f_{ii}(S_i, E_i^*) h_i(S_i^*, W^*)}\right).
\end{aligned} \tag{13}$$

Where the inequality follows inequality (9). Let

$$\begin{aligned}
F_{ij} &= 2 - \frac{E_i}{E_i^*} - \frac{E_i^* f_{ij}(S_i, E_j)}{E_i f_{ij}(S_i^*, E_j^*)} - \frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)} + \frac{f_{ii}(S_i^*, E_i^*) f_{ij}(S_i, E_j)}{f_{ii}(S_i, E_i^*) f_{ij}(S_i^*, E_j^*)}, \\
G_{ij} &= 2 - \frac{E_i}{E_i^*} - \frac{E_i^* g_{ij}(S_i, I_j)}{E_i g_{ij}(S_i^*, I_j^*)} - \frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)} + \frac{f_{ii}(S_i^*, E_i^*) g_{ij}(S_i, I_j)}{f_{ii}(S_i, E_i^*) g_{ij}(S_i^*, I_j^*)}, \\
H_i &= 2 - \frac{E_i}{E_i^*} - \frac{E_i^* h_i(S_i, W)}{E_i h_i(S_i^*, W^*)} - \frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)} + \frac{f_{ii}(S_i^*, E_i^*) h_i(S_i, W)}{f_{ii}(S_i, E_i^*) h_i(S_i^*, W^*)}, \\
D_i(x_i) &= -\frac{x_i}{x_i^*} + \ln \frac{x_i}{x_i^*}, \Phi(a) = 1 - a + \ln a.
\end{aligned}$$

Then it is easy to verify that

$$\Phi(a) = 1 - a + \ln a \leq 0, \forall a > 0, \tag{14}$$

the equality holds only when $a = 1$. Furthermore, under the condition of the inequalities (10), it is easy to obtain

$$\begin{aligned}
F_{ij} &= \Phi \left(\frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)} \right) - \ln \left(\frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)} \right) + \Phi \left(\frac{E_i^* f_{ij}(S_i, E_j)}{E_i f_{ij}(S_i^*, E_j^*)} \right) - \ln \left(\frac{E_i^* f_{ij}(S_i, E_j)}{E_i f_{ij}(S_i^*, E_j^*)} \right) \\
&\quad - \frac{E_i}{E_i^*} + \frac{E_j}{E_j^*} + \Phi \left(\frac{E_j f_{ii}(S_i, E_i^*) f_{ij}(S_i^*, E_j^*)}{E_j^* f_{ii}(S_i^*, E_i^*) f_{ij}(S_i, E_j)} \right) - \ln \left(\frac{E_j f_{ii}(S_i, E_i^*) f_{ij}(S_i^*, E_j^*)}{E_j^* f_{ii}(S_i^*, E_i^*) f_{ij}(S_i, E_j)} \right) \\
&\quad + \left(1 - \frac{E_j f_{ii}(S_i, E_i^*) f_{ij}(S_i^*, E_j^*)}{E_j^* f_{ii}(S_i^*, E_i^*) f_{ij}(S_i, E_j)} \right) \left(\frac{f_{ii}(S_i^*, E_i^*) f_{ij}(S_i, E_j)}{f_{ii}(S_i, E_i^*) f_{ij}(S_i^*, E_j^*)} - 1 \right) \\
&\leq \frac{E_j}{E_j^*} - \frac{E_i}{E_i^*} - \ln \left(\frac{f_{ii}(S_i^*, E_i^*)}{f_{ii}(S_i, E_i^*)} \right) - \ln \left(\frac{E_i^* f_{ij}(S_i, E_j)}{E_i f_{ij}(S_i^*, E_j^*)} \right) - \ln \left(\frac{E_j f_{ii}(S_i, E_i^*) f_{ij}(S_i^*, E_j^*)}{E_j^* f_{ii}(S_i^*, E_i^*) f_{ij}(S_i, E_j)} \right) \\
&= \frac{E_j}{E_j^*} - \ln \frac{E_j}{E_j^*} + \ln \frac{E_i}{E_i^*} - \frac{E_i}{E_i^*} = D_i(E_i) - D_j(E_j).
\end{aligned} \tag{15}$$

Similarly, using inequalities (11) and (12), we can obtain

$$G_{ij} \leq \frac{I_j}{I_j^*} - \ln \frac{I_j}{I_j^*} + \ln \frac{E_i}{E_i^*} - \frac{E_i}{E_i^*} = D_i(E_i) - D_j(I_j). \tag{16}$$

$$H_i \leq \frac{W}{W^*} - \ln \frac{W}{W^*} + \ln \frac{E_i}{E_i^*} - \frac{E_i}{E_i^*} = D_i(E_i) - D(W). \tag{17}$$

Taking $\beta'_{ij} = \beta_{ij} f_{ij}(S_i^*, E_j^*)$, $\varphi'_{ij} = \varphi_{ij} g_{ij}(S_i^*, I_j^*)$, $\phi'_i = \phi_i h_i(S_i^*, W^*)$. Using inequalities (15), (16) and (17), we can obtain

$$\begin{aligned}
\frac{dV_{i1}}{dt} &\leq \sum_{j=1}^n \beta_{ij} f_{ij}(S_i^*, E_j^*) F_{ij} + \sum_{j=1}^n \varphi_{ij} g_{ij}(S_i^*, I_j^*) G_{ij} + \phi_i h_i(S_i^*, W^*) H_i \\
&\leq \sum_{j=1}^n (\beta'_{ij} (D_i(E_i) - D_j(E_j)) + \varphi'_{ij} (D_i(E_i) - D_j(I_j))) + \phi'_i (D_i(E_i) - D(W)).
\end{aligned} \tag{18}$$

Similarly, for V_{i2} and V_3 , it is easy to obtain

$$\begin{aligned}
\frac{dV_{i2}}{dt} &= \left(1 - \frac{I_i}{I_i^*} \right) I'_i = \left(1 - \frac{I_i}{I_i^*} \right) \left(c_i E_i - c_i E_i^* \frac{I_i}{I_i^*} \right) = c_i E_i^* \left(\frac{E_i}{E_i^*} - \frac{I_i}{I_i^*} + \Phi \left(\frac{E_i I_i^*}{E_i^* I_i} \right) - \ln \frac{E_i I_i^*}{E_i^* I_i} \right) \\
&\leq c_i E_i^* \left(\frac{E_i}{E_i^*} - \ln \frac{E_i}{E_i^*} + \ln \frac{I_i}{I_i^*} - \frac{I_i}{I_i^*} \right) = c_i E_i^* (D_i(I_i) - D_i(E_i)).
\end{aligned} \tag{19}$$

$$\begin{aligned}
\frac{dV_3}{dt} &= \left(1 - \frac{W}{W^*} \right) W' = \left(1 - \frac{W}{W^*} \right) \left(\sum_{i=1}^n (k_i(E_i) + m_i(I_i)) - \sum_{i=1}^n (k_i(E_i^*) + m_i(I_i^*)) \frac{W}{W^*} \right) \\
&= \sum_{i=1}^n k_i(E_i^*) \left(1 + \frac{k_i(E_i)}{k_i(E_i^*)} - \frac{W}{W^*} - \frac{k_i(E_i) W^*}{k_i(E_i^*) W} \right) + \sum_{i=1}^n m_i(I_i^*) \left(1 + \frac{m_i(I_i)}{m_i(I_i^*)} - \frac{W}{W^*} - \frac{m_i(I_i) W^*}{m_i(I_i^*) W} \right) \\
&\leq \sum_{i=1}^n k_i(E_i^*) \left(\left(\frac{k_i(E_i)}{k_i(E_i^*)} - 1 \right) \left(1 - \frac{k_i(E_i^*) E_i}{k_i(E_i) E_i^*} \right) + \frac{E_i}{E_i^*} - \ln \frac{E_i}{E_i^*} + \ln \frac{W}{W^*} - \frac{W}{W^*} \right) \\
&\quad + \sum_{i=1}^n m_i(I_i^*) \left(\left(\frac{m_i(I_i)}{m_i(I_i^*)} - 1 \right) \left(1 - \frac{m_i(I_i^*) I_i}{m_i(I_i) I_i^*} \right) + \frac{I_i}{I_i^*} - \ln \frac{I_i}{I_i^*} + \ln \frac{W}{W^*} - \frac{W}{W^*} \right) \\
&\leq \sum_{i=1}^n k_i(E_i^*) \left(\frac{E_i}{E_i^*} - \ln \frac{E_i}{E_i^*} + \ln \frac{W}{W^*} - \frac{W}{W^*} \right) + \sum_{i=1}^n m_i(I_i^*) \left(\frac{I_i}{I_i^*} - \ln \frac{I_i}{I_i^*} + \ln \frac{W}{W^*} - \frac{W}{W^*} \right) \\
&= \sum_{i=1}^n (k_i(E_i^*) (D(W) - D_i(E_i)) + m_i(I_i^*) (D(W) - D_i(I_i))).
\end{aligned} \tag{20}$$

The first inequality of (20) uses the inequality (14), which follows from

$$\begin{aligned}
 M_1 &= 1 + \frac{k_i(E_i)}{k_i(E_i^*)} - \frac{W_i}{W_i^*} - \frac{W_i^* k_i(E_i)}{W_i k_i(E_i^*)} \\
 &= \frac{k_i(E_i)}{k_i(E_i^*)} - 1 - \frac{E_i}{E_i^*} + \frac{k_i(E_i^*) E_i}{k_i(E_i) E_i^*} + 2 + \frac{E_i}{E_i^*} - \frac{W_i}{W_i^*} - \frac{k_i(E_i^*) E_i}{k_i(E_i) E_i^*} - \frac{W_i^* k_i(E_i)}{W_i k_i(E_i^*)} \\
 &= \frac{k_i(E_i)}{k_i(E_i^*)} - 1 - \frac{E_i}{E_i^*} + \frac{k_i(E_i^*) E_i}{k_i(E_i) E_i^*} + \frac{E_i}{E_i^*} - \frac{W_i}{W_i^*} \\
 &\quad + \Phi \left(\frac{k_i(E_i^*) E_i}{k_i(E_i) E_i^*} \right) - \ln \frac{k_i(E_i^*) E_i}{k_i(E_i) E_i^*} + \Phi \left(\frac{W_i^* k_i(E_i)}{W_i k_i(E_i^*)} \right) - \ln \frac{W_i^* k_i(E_i)}{W_i k_i(E_i^*)} \\
 &\leq \left(\frac{k_i(E_i)}{k_i(E_i^*)} - 1 \right) \left(1 - \frac{k_i(E_i^*) E_i}{k_i(E_i) E_i^*} \right) + \frac{E_i}{E_i^*} - \ln \frac{E_i}{E_i^*} + \ln \frac{W_i}{W_i^*} - \frac{W_i}{W_i^*}.
 \end{aligned}$$

Similarity, we can obtain

$$\begin{aligned}
 M_2 &= 1 + \frac{m_i(I_i)}{m_i(I_i^*)} - \frac{W_i}{W_i^*} - \frac{W_i^* m_i(I_i)}{W_i m_i(I_i^*)} \\
 &\leq \left(\frac{m_i(I_i)}{m_i(I_i^*)} - 1 \right) \left(1 - \frac{m_i(I_i^*) I_i}{m_i(I_i) I_i^*} \right) + \frac{I_i}{I_i^*} - \ln \frac{I_i}{I_i^*} + \ln \frac{W_i}{W_i^*} - \frac{W_i}{W_i^*}.
 \end{aligned}$$

The second inequality of (20) mainly follows from assumption (H4). Using assumption (H4), we can obtain the following inequality

$$\left(\frac{k_i(E_i)}{k_i(E_i^*)} - 1 \right) \leq 0, \left(1 - \frac{k_i(E_i^*) E_i}{k_i(E_i) E_i^*} \right) \geq 0, 0 < E_i \leq E_i^*,$$

and

$$\left(\frac{k_i(E_i)}{k_i(E_i^*)} - 1 \right) > 0, \left(1 - \frac{k_i(E_i^*) E_i}{k_i(E_i) E_i^*} \right) < 0, E_i > E_i^*.$$

Hence, it is easy to obtain

$$\left(\frac{k_i(E_i)}{k_i(E_i^*)} - 1 \right) \left(1 - \frac{k_i(E_i^*) E_i}{k_i(E_i) E_i^*} \right) \leq 0, E_i > 0.$$

In the same way, we can also obtain

$$\left(\frac{m_i(I_i)}{m_i(I_i^*)} - 1 \right) \left(1 - \frac{m_i(I_i^*) I_i}{m_i(I_i) I_i^*} \right) \leq 0, I_i > 0.$$

In order to prove the global stability of P^* for system (1), we need to define the following Lyapunov function

$$V = \sum_{i=1}^n v_i \left(V_{i1} + \frac{1}{c_i E_i^*} \frac{\phi'_i \sum_{i=1}^n m_i(I_i^*)}{\sum_{i=1}^n (k_i(E_i^*) + m_i(I_i^*))} V_{i2} + \frac{\phi'_i}{\sum_{i=1}^n (k_i(E_i^*) + m_i(I_i^*))} V_3 \right) + \sum_{i,j=1}^n v_j \frac{\varphi'_{ji}}{c_i E_i^*} V_{i2}. \quad (21)$$

Then the derivative of V along solutions of system (1) is

$$\frac{dV}{dt} = \sum_{i=1}^n v_i \left(V'_{i1} + \frac{1}{c_i E_i^*} \frac{\phi'_i \sum_{i=1}^n m_i(I_i^*)}{\sum_{i=1}^n (k_i(E_i^*) + m_i(I_i^*))} V'_{i2} + \frac{\phi'_i}{\sum_{i=1}^n (k_i(E_i^*) + m_i(I_i^*))} V'_3 \right) + \sum_{i,j=1}^n v_j \frac{\phi'_{ji}}{c_i E_i^*} V'_{i2}.$$

Through using inequalities (18), (19) and (20), it is easy to obtain

$$\begin{aligned} \frac{dV}{dt} &\leq \sum_{i=1}^n v_i \left(\sum_{j=1}^n (\beta'_{ij}(D_i(E_i) - D_j(E_j)) + \varphi'_{ij}(D_i(E_i) - D_j(I_j))) + \phi'_i(D_i(E_i) - D(W)) \right) \\ &\quad + \sum_{i=1}^n v_i \left(\frac{\phi'_i \sum_{i=1}^n m_i(I_i^*)}{\sum_{i=1}^n (k_i(E_i^*) + m_i(I_i^*))} (D_i(I_i) - D_i(E_i)) \right) + \sum_{i,j=1}^n v_j \varphi'_{ji}(D_i(I_i) - D_i(E_i)) \\ &\quad + \sum_{i=1}^n v_i \left(\frac{\phi'_i}{\sum_{i=1}^n (k_i(E_i^*) + m_i(I_i^*))} \sum_{i=1}^n (k_i(E_i^*)(D(W) - D_i(E_i)) + m_i(I_i^*)(D(W) - D_i(I_i))) \right) \\ &= \sum_{i=1}^n v_i \left(\sum_{j=1}^n (\beta'_{ij}(D_i(E_i) - D_j(E_j)) + \varphi'_{ij}(D_i(E_i) - D_j(I_j))) \right) + \sum_{i,j=1}^n v_j \varphi'_{ji}(D_i(I_i) - D_i(E_i)) \\ &= \sum_{i=1}^n v_i \left(\sum_{j=1}^n (\beta'_{ij}(D_i(E_i) - D_j(E_j)) + \varphi'_{ij}(D_i(E_i) - D_j(I_j))) \right) + \sum_{i,j=1}^n v_i \varphi'_{ij}(D_j(I_j) - D_j(E_j)) \\ &= \sum_{i,j=1}^n v_i ((\beta'_{ij} + \varphi'_{ij})(D_i(E_i) - D_j(E_j))). \end{aligned}$$

Due to matrix $[\beta_{ij}]_{1 \leq i,j \leq n}$ and $[\varphi_{ij}]_{1 \leq i,j \leq n}$ are irreducible, so the matrix $[\beta'_{ij} + \varphi'_{ij}]_{1 \leq i,j \leq n}$ is also irreducible. According to the methods and conclusions in [24], since $[\beta'_{ij} + \varphi'_{ij}]_{1 \leq i,j \leq n}$ is irreducible, there exist constants $v_i > 0, i = 1, 2, \dots, n$ such that

$$\sum_{i,j=1}^n v_i ((\beta'_{ij} + \varphi'_{ij})(D_i(E_i) - D_j(E_j))) = 0.$$

Therefore, the function V as defined in the Theorem 3.1 of [24] is a Lyapunov function for system (1), namely, $V' \leq 0$ for all P^* belong to in the interior of X . From inequalities (18), (19) and (20), we know that $V' = 0$ iff

$$S_i = S_i^*, E_i = E_i^*, I_i = I_i^*, W = W^*, i = 1, 2, \dots, n.$$

So the only compact invariant subset of the set where $V' = 0$ is the singleton $\{P^*\}$. By LaSalle's Invariance Principle [22], P^* is globally asymptotically stable in the interior of X when $R_0 > 1$. The proof is end.

Remark. We remark that Lyapunov functions for similar models have been used in many papers. In this paper we gave the specific coefficients of global Lyapunov functions which are absent from previous papers [10,11,24,32,35,36,45,48]. The specific coefficients are shown in the equation (21), which are

$$\frac{1}{c_i E_i^*} \frac{\phi'_i \sum_{i=1}^n m_i(I_i^*)}{\sum_{i=1}^n (k_i(E_i^*) + m_i(I_i^*))}, \frac{\phi'_i}{\sum_{i=1}^n (k_i(E_i^*) + m_i(I_i^*))} \text{ and } \sum_{i,j=1}^n v_j \frac{\varphi'_{ji}}{c_i E_i^*}, \text{ respectively.}$$

3. Some specific examples

In this section, we will give some specific cases to illustrate the usefulness of the results. From the previous section, we know that there are many general incidence functionals satisfy assumptions (H1)–(H4). So we only need to verify these incidence functionals satisfy equation (9), (10), (11) and (12).

Case 1. The transmission incidence functions $f_{ij}(S_i, E_j)$, $g_{ij}(S_i, I_j)$ and $h_{ij}(S_i, W_j)$ are bilinear, which are $f_{ij}(S_i, E_j) = S_i E_j$, $g_{ij}(S_i, I_j) = S_i I_j$ and $h_i(S_i, W) = S_i W$. Using these bilinear incidences for equation (9), (10), (11) and (12), we can obtain

$$\Theta_1 = \left(1 - \frac{S_i^* E_i^*}{S_i E_i^*}\right) (S_i^* - S_i) \leq 0, \Theta_2 = \left(1 - \frac{E_j}{E_j^*} \frac{S_i E_i^* S_i^* E_j^*}{S_i^* E_i^* S_i E_j}\right) \left(\frac{S_i^* E_i^* S_i E_j}{S_i E_i^* S_i^* E_j^*} - 1\right) = 0.$$

Similarity, it is easy to obtain $\Theta_3 = 0$ and $\Theta_4 = 0$, which implies that equation (9), (10), (11) and (12) hold, it is also meaning the endemic equilibrium of the model is globally asymptotically stable when the basic reproduction number is larger than 1. For the special case of our previous brucellosis model in paper [26], we can obtain the endemic equilibrium is also globally asymptotically stable. Hence, our model with bilinear incidence contains earlier cholera models in [40], brucellosis models in [13,25,26], and the general disease multi-group models [10,11,35] as special cases.

Case 2. In paper [27], the direct and indirect transmission incidences are $f(S, I) = SI$ (bilinear) and $h(S, W) = \frac{SW}{1+\alpha W}$ (saturating), respectively. And the model is

$$\begin{cases} \frac{dS}{dt} = \mu(S + I + R) - (\beta_h SI + \beta_e \frac{SW}{1+\alpha W}) - \mu S, \\ \frac{dI}{dt} = \beta_h SI + \beta_e \frac{SW}{1+\alpha W} - (\mu + \gamma)I, \\ \frac{dR}{dt} = \gamma I - \mu R, \\ \frac{dW}{dt} = \xi I - \delta W. \end{cases} \quad (22)$$

Using the transmission incidences $f(S, I) = SI$ and $h(S, W) = \frac{SW}{1+\alpha W}$ for equation (9), (10), (11) and (12), we can obtain

$$\begin{aligned} \Theta_1 &= \left(1 - \frac{f(S^*, I^*)}{f(S, I^*)}\right) (S^* - S) = \left(1 - \frac{S^* I^*}{S I^*}\right) (S^* - S) \leq 0, \Theta_3 = 0, \\ \Theta_2 &= \left(1 - \frac{I}{I^*} \frac{f(S, I^*) f(S^*, I^*)}{f(S^*, I^*) f(S, I)}\right) \left(\frac{f(S^*, I^*) f(S, I)}{f(S, I^*) f(S^*, I^*)} - 1\right) = \left(1 - \frac{I}{I^*} \frac{S I^* S^* I^*}{S^* I^* S I}\right) \left(\frac{S^* I^* S I}{S I^* S^* I^*} - 1\right) = 0, \\ \Theta_4 &= \left(1 - \frac{W}{W^*} \frac{f(S, I^*) h(S^*, W^*)}{f(S^*, I^*) h(S, W)}\right) \left(\frac{f(S^*, I^*) h(S, W)}{f(S, I^*) h(S^*, W^*)} - 1\right) \\ &= \left(1 - \frac{W}{W^*} \frac{S I^* \frac{S^* W^*}{1+\alpha W^*}}{S^* I^* \frac{S W}{1+\alpha W}}\right) \left(\frac{S^* I^* \frac{S W}{1+\alpha W}}{S I^* \frac{S^* W^*}{1+\alpha W^*}} - 1\right) \\ &= \left(1 - \frac{1 + \alpha W}{1 + \alpha W^*}\right) \left(\frac{\frac{W}{1+\alpha W}}{\frac{W^*}{1+\alpha W^*}} - 1\right) = \frac{-\alpha(W - W^*)^2}{W^*(1 + \alpha W)(1 + \alpha W^*)} \leq 0. \end{aligned}$$

Hence, the endemic equilibrium of paper [27] is globally asymptotically stable when the basic reproduction number is larger than 1. Compared with the proof of global stability of endemic equilibrium in paper [27], our method is more simple and effective, and easy to understand. The model with direct bilinear transmission rate and indirect saturating transmission rate contains cholera models in [3,27,28]. In the same way, if the incidence functions satisfy $f_{ij}(S_i, E_j) = \frac{S_i E_j}{1+\alpha E_j}$ and $g_{ij}(S_i, I_j) = \frac{S_i I_j}{1+\alpha I_j}$, we can also obtain $\Theta_1 \leq 0, \Theta_2 \leq 0$ and $\Theta_3 \leq 0$.

Remark. If the incidence function satisfies $f_{ij}(S_i, E_j) = \frac{S_i E_j}{b + \theta S_i}$ and $f_{ij}(S_i, E_j) = S_i^q E_j$ ($q \geq 1$), it is also easy to verify $\Theta_1 \leq 0, \Theta_2 = 0, \Theta_3 = 0, \Theta_4 = 0$ and $\Theta'_4 = 0$. Unfortunately, we can not verify equation (11) holds when the incidence function satisfies $f_{ij}(S_i, E_j) = \frac{S_i E_j}{b + \alpha E_j^2}$ and $f_{ij}(S_i, E_j) = \frac{S_i E_j}{S_i + E_i + I_i}$, we leave this for future investigation.

4. Discussion and conclusion

In order to model the importance of the survival of pathogens outside the host and heterogeneities in disease transmission, we have proposed a multi-group compartmental model with direct and indirect transmission, nonlinear incidence rates and nonlinear pathogen shedding rate functions. If nonlinear incidence rates and nonlinear pathogen shedding rate functions satisfy assumptions (H1)–(H4), then the disease-free equilibrium of system (1) is globally asymptotically stable when the basic reproduction number is smaller than 1. Further, if nonlinear incidence rates also satisfy equation (10), (11), (12) and (13), then the endemic equilibrium of system (1) is globally asymptotically stable when the basic reproduction number is larger than 1, and the disease will persist at a constant endemic level. For our model, general forms for direct and indirect transmission include both mass action, saturating incidence and other incidence. Our model contains earlier cholera models in [3,27,28,39,40] and brucellosis models in [1,13,25,26] as special cases. The proofs of the our main results exploit the method of constructing Lyapunov functions and a graph-theoretical technique in estimating the derivatives of Lyapunov functions. We also give the specific coefficients of global Lyapunov functions which are absent from previous papers [10,11,24,32,35,36,45,48].

The stability of a delayed multi-group SIS epidemic model with nonlinear incidence rates and patch structure has been studied in paper [29]. Combined with the results of our paper and the previous paper [29], some further works on multi-group epidemic models with latency, vaccination group, or path structure can be done. Furthermore, from our previous analysis we know that some nonlinear incidence rates do not satisfy assumptions (H1)–(H4) and equation (10), (11), (12) and (13). Such as $f_{ij}(S_i, E_j) = \frac{S_i E_j}{b + \alpha E_j^2}$ or $f_{ij}(S_i, E_j) = \frac{S_i E_j}{S_i + E_i + I_i}$, which would result in various dynamical behaviors, and we will leave this for future investigation.

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References

- [1] B. Alnseba, B. Chahrazed, M. Pierre, A model for ovine brucellosis incorporating direct and indirect transmission, *J. Biol. Dyn.* 4 (2010) 2–11.
- [2] R.M. Anderson, R.M. May, Population biology of infectious diseases I, *Nature* 280 (1979) 361–367.
- [3] C.T. Codeço, Endemic and epidemic dynamics of cholera: the role of the aquatic reservoir, *BMC Infect. Dis.* 1 (1) (2001).
- [4] M.J. Corbel, *Brucellosis in Humans and Animals*, World Health Organization, 2006.
- [5] O. Diekmann, J.A.P. Heesterbeek, J.A.J. Metz, On the definition and computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations, *J. Math. Biol.* 28 (1990) 365–382.
- [6] O. Diekmann, J.A.P. Heesterbeek, M.G. Roberts, The construction of next-generation matrices for compartmental epidemic models, *J. R. Soc. Interface* 7 (2010) 873–885.
- [7] S.F. Dowell, Seasonal variation in host susceptibility and cycles of certain infectious diseases, *Emerg. Infect. Dis.* 7 (2001) 369–374.

- [8] M.C. Eisenberga, Z.S. Shuai, J.H. Tien, P. van den Driessche, A cholera model in a patchy environment with water and human movement, *Math. Biosci.* 246 (2013) 105–112.
- [9] H.I. Freedman, S.G. Ruan, M.X. Tang, Uniform persistence and flows near a closed positively invariant set, *J. Dynam. Differential Equations* 6 (1994) 583–600.
- [10] H. Guo, M.Y. Li, Z. Shuai, Global stability of the endemic equilibrium of multigroup SIR epidemic models, *Can. Appl. Math. Q.* 14 (2006) 259–284.
- [11] H. Guo, M.Y. Li, Z. Shuai, A graph-theoretic approach to the method of global Lyapunov functions, *Proc. Amer. Math. Soc.* 136 (2008) 2793–2802.
- [12] H.W. Hethcote, An immunization model for a heterogeneous population, *Theor. Popul. Biol.* 14 (1978) 338–349.
- [13] Q. Hou, X.D. Sun, J. Zhang, Y.J. Liu, Y.M. Wang, Z. Jin, Modeling the transmission dynamics of brucellosis in Inner Mongolia Autonomous Region, China, *Math. Biosci.* 242 (2013) 51–58.
- [14] Q. Hou, X.D. Sun, Y.M. Wang, B.X. Huang, Z. Jin, Global properties of a general dynamic model for animal diseases: a case study of brucellosis and tuberculosis transmission, *J. Math. Anal. Appl.* 414 (2014) 424–433.
- [15] G. Huang, J.L. Wang, J. Zu, Global dynamics of multi-group dengue disease model with latency distributions, *Math. Methods Appl. Sci.* 38 (13) (2015) 2703–2718.
- [16] C.B. Jonsson, L.T.M. Figueiredo, O. Vapalahti, A global perspective on hantavirus ecology, epidemiology, and disease, *Clin. Microbiol. Rev.* 23 (2) (2010) 412.
- [17] E.R. Kallio, J. Klingstro, E. Gustafsson, T. Manni, A. Vaheri, H. Henttonen, O. Vapalahti, Å. Lundkvist, Prolonged survival of Puumala hantavirus outside the host: evidence for indirect transmission via the environment, *J. Gen. Virol.* 87 (2006) 2127–2134.
- [18] E. Kenah, D.L. Chao, L. Matrajt, M.E. Halloran, I.M. Longini Jr., The global transmission and control of influenza, *PLoS ONE* 6 (5) (2011) e19515.
- [19] A. Kiszewski, A. Mellinger, A. Spielman, P. Malaney, S.E. Sachs, J. Sachs, A global index representing the stability of malaria transmission, *Am. J. Trop. Med. Hyg.* 70 (5) (2004) 486–498.
- [20] J.L. Kyle, E. Harris, Global spread and persistence of dengue, *Annu. Rev. Microbiol.* 62 (2008) 71–92.
- [21] A. Lajmanovich, J.A. York, A deterministic model for gonorrhea in a nonhomogeneous population, *Math. Biosci.* 28 (1976) 221–236.
- [22] J.P. Lasalle, *The Stability of Dynamical Systems*, Regional Conference Series in Applied Mathematics, SIAM, Philadelphia, 1976.
- [23] M.Y. Li, J.R. Graef, L. Wang, J. Karsai, Global dynamics of a SEIR model with varying total population size, *Math. Biosci.* 160 (1999) 191–213.
- [24] M.Y. Li, Z. Shuai, Global-stability problem for coupled systems of differential equations on networks, *J. Differential Equations* 248 (2010) 1–20.
- [25] M.T. Li, G.Q. Sun, Y.F. Wu, J. Zhang, Z. Jin, Transmission dynamics of a multi-group brucellosis model with mixed cross infection in public farm, *Appl. Math. Comput.* 237 (2014) 582–594.
- [26] M.T. Li, G.Q. Sun, J. Zhang, Z. Jin, X.D. Sun, Y.M. Wang, B.X. Huang, Y.H. Zheng, Transmission dynamics and control for a brucellosis model in Hinggan League of Inner Mongolia, China, *Math. Biosci. Eng.* 11 (2014) 1115–1137.
- [27] Z. Mukandavire, S. Liao, J. Wang, H. Gaff, D.L. Smith, J.G. Morris Jr., Estimating the reproductive numbers for the 2008–2009 cholera outbreaks in Zimbabwe, *Proc. Natl. Acad. Sci. USA* 108 (2011) 8767–8772.
- [28] Z. Mukandavire, D.L. Smith, J.G. Morris Jr., Cholera in Haiti: reproductive numbers and vaccination coverage estimates, *Sci. Rep.* 3 (2013) 997.
- [29] Y. Muroya, T. Kuniya, J.L. Wang, Stability analysis of a delayed multi-group SIS epidemic model with nonlinear incidence rates and patch structure, *J. Math. Anal. Appl.* 425 (2015) 415–439.
- [30] A. Mutreja, D.W. Kim, N.R. Thomson, T.R. Connor, J.H. Lee, S. Kariuki, N.J. Croucher, S.Y. Choi, S.R. Harris, M. Lebens, S.K. Niyogi, E.J. Kim, T. Ramamurthy, J. Chun, J.L.N. Wood, J.D. Clemens, C. Czerkinsky, G.B. Nair, J. Holmgren, J.L. Parkhill, G. Dougan, Evidence for several waves of global transmission in the seventh cholera pandemic, *Nature* 477 (2011) 462–465.
- [31] F. Sauvage, M. Langlais, N.G. Yoccoz, D. Pontier, Modelling hantavirus in fluctuating populations of bank voles: the role of indirect transmission on virus persistence, *J. Anim. Ecol.* 72 (2003) 1–13.
- [32] Z.S. Shuai, P. van den Driessche, Global dynamics of cholera models with differential infectivity, *Math. Biosci.* 234 (2011) 118–126.
- [33] Z.S. Shuai, P. van den Driessche, Global stability of infectious disease models using Lyapunov functions, *SIAM J. Appl. Math.* 73 (4) (2013) 1513–1532.
- [34] H.L. Smith, P. Waltman, *The Theory of the Chemostat*, Cambridge University Press, 1995.
- [35] R. Sun, Global stability of the endemic equilibrium of multigroup SIR models with nonlinear incidence, *Comput. Math. Appl.* 60 (2010) 2286–2291.
- [36] R. Sun, J. Shi, Global stability of multigroup epidemic model with group mixing and nonlinear incidence rates, *Appl. Math. Comput.* 218 (2011) 280–286.
- [37] H.R. Thieme, Persistence under relaxed point-dissipativity (with application to an endemic model), *SIAM J. Math. Biosci.* 166 (1993) 407–435.
- [38] G.R. Thomson, W. Vosloo, A.D.S. Bastos, Foot and mouth disease in wildlife, *Virus Res.* 91 (2003) 145–161.
- [39] J.J. Paul Tian, J. Wang, Global stability for cholera epidemic models, *Math. Biosci.* 232 (2011) 31–41.
- [40] J.H. Tien, D.J.D. Earn, Multiple transmission pathways and disease dynamics in a waterborne pathogen model, *Bull. Math. Biol.* 72 (2010) 1506.
- [41] 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.
- [42] J.L. Wang, X.N. Liu, J.M. Pang, D.M. Hou, Global dynamics of a multi-group epidemic model with general exposed distribution and relapse, *Osaka J. Math.* 52 (2015) 117–138.

- [43] J.L. Wang, H.Y. Shu, Global dynamics of a multi-group epidemic model with latency, relapse and nonlinear incidence rate, *Math. Biosci. Eng.* 13 (1) (2016) 209–225.
- [44] X. Wang, Y.D. Tao, X.Y. Song, Pulse vaccination on SEIR epidemic model with nonlinear incidence rate, *Appl. Math. Comput.* 210 (2009) 398–404.
- [45] Y. Wang, J.D. Cao, Global dynamics of multi-group SEI animal disease models with indirect transmission, *Chaos Solitons Fractals* 69 (2014) 81–89.
- [46] L.W. Woods, Adenoviral diseases, in: E.S. Williams, I.K. Barker (Eds.), *Infectious Diseases of Wildlife Mammals*, 3rd edn., Manson, London, 2001, pp. 202–212.
- [47] D. Xiao, S. Ruan, Global analysis of an epidemic model with nonmonotone incidence rate, *Math. Biosci.* 208 (2007) 419–429.
- [48] Z. Yuan, L. Wang, Global stability of epidemiological models with group mixing and nonlinear incidencerates, *Nonlinear Anal. RWA* 11 (2010) 995–1004.
- [49] J. Zhang, Z. Jin, G.Q. Sun, X.D. Sun, Y.M. Wang, B.X. Huang, Determination of original infection source of H7N9 avian influenza by dynamical model, *Sci. Rep.* 4 (2014).
- [50] X.Q. Zhao, *Dynamical Systems in Population Biology*, Springer, New York, 2003.
- [51] X.Q. Zhao, Z.-J. Jing, Global asymptotic behavior in some cooperative systems of functional differential equations, *Can. Appl. Math. Q.* 4 (1996) 421–444.