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Global properties of a general dynamic model for animal diseases: A case study of brucellosis and tuberculosis transmission

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ABSTRACT

Animal diseases such as brucellosis and tuberculosis can be transmitted through an environmentally mediated mechanism, but the topics of most modeling work are based on infectious contact and direct transmission, which leads to the limited understanding of the transmission dynamics of these diseases. In this paper, we propose a new deterministic model which incorporates general incidences, various stages of infection and a general shedding rate of the pathogen to analyze the dynamics of these diseases. Under the biologically motivated assumptions, we derive the basic reproduction number R_0, show the uniqueness of the endemic equilibrium, and prove the global asymptotically stability of the equilibria. Some specific examples are used to illustrate the utilization of our results. In addition, we elaborate the epidemiological significance of these results, which are very important for the prevention and control of animal diseases.

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1. Introduction

Animal diseases, can be transmitted not only to other animals, but also to humans with exposure to infected animals or ingestion of contaminated water and dust in the environment. For example, brucellosis, caused by intracellular bacteria of the genus Brucella, can infect a wide variety of wild and domestic animal hosts, and is regarded as one of the most important zoonosis [15]. Although some animal diseases are well controlled in some developed countries, such as bovine brucellosis in Australia, most animal diseases are still common clinical diseases in the world [19]. Particularly, many animal diseases represent a significant public health burden on developing countries and continue receiving worldwide attention.

As for infectious animal diseases, the incubation period varies with species and stages of gestation at infection. Progression of the brucellosis infection can take two weeks to five months before the occurrence of the clinical signs, and goes through several distinct stages. The infectivity of an infected animal is determined by two main factors: brucella shed by exposed and infected animals which can survive for several weeks, or even months in the feces or contaminated environment and the frequency at which the host is in contact with others [2,14]. For the brucellosis infection, the transmissibility increases from the exposed stage to the infectious stage, and it is difficult to find the infected animals in the exposed period through the method of serum detection. For the tuberculosis (TB) infection, the usual route of infection is by inhaling infected droplets which are expelled from the lungs by coughing. Because the course of disease is slow, taking months or years to kill an infected animal, an animal can spread the disease to many other herd mates before it begins to manifest clinical

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signs [21]. Therefore, contact with undetected infected domestic animals and ingestion of the pathogen in the environment are the major ways of spreading the disease. On the other hand, it is worth noting note that an asymptomatic disease carrier may have a low pathogen load while maintaining a high infectivity due to risky behaviors.

Transmission dynamics of certain animal diseases are complicated with the multiple interaction between the animal host and the pathogen in the environment, which results in both direct animal-to-animal and indirect environment-to-animal transmission pathways. In addition, the treatment of infected animals is rarely attempted because of the high cost and the larger goal of eliminating the disease. Then the standard control measure applied to animal diseases is vaccination, test and culling. It is noteworthy that these control measures play an important role in the prevalence of some animal diseases and the impact of intervention measures needs to be carefully investigated using mathematical models. The present work aims to understand the global dynamics of these animal diseases in a general mathematical model which has a potential to incorporate different factors into a unified framework. The study tends to be crucial for effective prevention and intervention against animal diseases outbreak.

Many dynamic models have been proposed to study the complex dynamics of certain animal diseases [1,3,5,8,10,16–18]. However, most of these studies focused on analyzing the spread of animal diseases with the direct transmission (contact transmission). Therefore, one difficulty in studying infectious diseases dynamics in animals is the coupling between its multiple transmission pathways which lead to combination of animal-environment epidemiological models. In the present paper, a general dynamic model of infectious animal diseases is proposed involving both direct and indirect transmission, nonlinear incidence, and various infection stages of infectious individuals. On very general and biologically plausible assumptions of the incidence, the birth of individuals, shedding rate of pathogenic bacteria, and removal functions, we analyze the uniqueness of the endemic equilibrium and the global stability of the equilibria, which are determined by the basic reproduction number R_0: if $R_0 \leqslant 1$, the disease-free equilibrium is globally asymptotically stable; if $R_0 > 1$, there exists a unique endemic equilibrium which is the globally asymptotically stable.

This work is structured as follows. Section 1 gives the general introduction of the study background. In Section 2, the dynamic model for infectious diseases is presented. We derive the basic reproduction number and show the uniqueness of the endemic equilibrium in Section 3. In Section 4, the global stability of the disease-free equilibrium and the endemic equilibrium are established. Some examples are shown in Section 5. A brief summary is given in Section 6.

2. The model and notations

Based on common characteristics of the spread of these animal diseases, we divide the animal population into three compartments: the susceptible compartment $S(t)$, the exposed compartment $E(t)$ and the infectious compartment $I(t)$. Normally, exposed and infected animals can shed pathogenic bacteria into the environment through abortion or animal secretions, which can be *harvested* by susceptible individuals who would become infected depending on the ingested dose. Therefore, infected or exposed animals would spread the bacteria in two ways: direct and indirect modes of transmission. Define $B(t)$ as the pathogens in the environment. We propose the following compartmental model for the dynamics of infectious animal diseases based on the combination of an *SEI* model and an environmental component:

$$\begin{cases} \dfrac{dS}{dt} = m(S) - n(S)\big(\rho(E) + f(I) + g(B)\big), \\ \dfrac{dE}{dt} = n(S)\big(\rho(E) + f(I) + g(B)\big) - (e+\sigma)E, \\ \dfrac{dI}{dt} = \sigma E - \varphi(I), \\ \dfrac{dB}{dt} = \omega(E) + h(I) - \theta(B). \end{cases} \tag{1}$$

Here, the intrinsic growth rate of the susceptible class is given by $m(S)$ with all the newly produced animals assumed to be susceptible. $n(S)$ is a contact function. eE is the elimination rate of the exposed class. The transfer rate from the exposed class into the infected class is given by σE. The elimination rate of the infected animals, including the disease induced death rate, is denoted by $\varphi(I)$. We define $\omega(E)$ and $h(I)$ as the pathogen shedding rate of the exposed and infected animals respectively. $\theta(B)$ represents the disinfection rate and decaying rate of pathogen in the environment. Moreover, the functions m, n, ρ, f, g, ϕ, φ, ω, h and θ are assumed to be sufficiently smooth so that solutions to (1) with nonnegative initial conditions exist and are unique. The following basic assumptions are to make the model biologically significant:

(H_1) There exists $S_0 > 0$ such that the equation $m(S_0) = 0$ with

$$\big(m(S) - m(S_0)\big)(S - S_0) < 0 \quad \text{for } S \neq S_0.$$

(H_2) $n(S) \geqslant 0$, for $S \geqslant 0$; $n'(S) > 0$ for $S > 0$.
(H_3) $\rho(0) = f(0) = g(0) = 0$, and $\rho(E), f(I), g(B) > 0$, for $E, I, B > 0$.
(H_4) The functions $\rho(E), f(I), g(B)$ satisfy the following conditions for $E, B, I > 0$:

$$\rho'(E) > 0, \qquad f'(I) > 0, \qquad g'(B) > 0$$

and

$$\rho''(E) \leqslant 0, \qquad f''(I) \leqslant 0, \qquad g''(B) \leqslant 0.$$

(H_5) $\omega(x), h(x) \geqslant 0$, and $\omega(x) \leqslant h(x)$, for $x \geqslant 0$; $\omega'(E), h'(I) > 0$ for $E, I > 0$.
(H_6) $\varphi(0) = 0$, $\varphi(I) > 0$ and $\varphi'(I) > 0$ for $I > 0$, there exists constant $\mu_1 > 0$ such that

$$\varphi(I) \geqslant \mu_1 I.$$

(H_7) $\theta(0) = 0$, $\theta(B) > 0$ and $\theta'(B) > 0$ for $B > 0$.

Assumption (H_1) ensures that the host population has carried capacity $S_0 > 0$ when there is no disease. With biological considerations, we are interested in solutions that are nonnegative and bounded. In fact, adding the first three equations of (1) yields that

$$(S + E + I)' = m(S) - eE - \varphi(I) \leqslant m(S) - \mu(E + I),$$

where $\mu = \min\{e, \mu_1\}$. Choose Π sufficiently large such that $\Pi \geqslant \mu S_0 + \max\{m(S)\}$. So we have

$$(S + E + I)' \leqslant m(S) - \mu(E + I) \leqslant \Pi - \mu(S + E + I),$$

then it follows that

$$\limsup_{t\to\infty}(S + E + I) \leqslant \frac{\Pi}{\mu}, \qquad \limsup_{t\to\infty} B \leqslant \theta^{-1}\left(h\left(\frac{\Pi}{\mu}\right)\right).$$

Therefore, the set

$$\Omega = \left\{(S, E, I, B) \in R_+^4 \colon S, E, I, B \geqslant 0, S + E + I \leqslant \frac{\Pi}{\mu}, B \leqslant \theta^{-1}\left(h\left(\frac{\Pi}{\mu}\right)\right)\right\}$$

is the positively invariant set for system (1).

The assumption (H_2) represents a common contact function, such as $\frac{S^q}{1+MS^q}$ with $q > 0$ and $M \geqslant 0$. The assumption (H_3) indicates that incidence rate is greater than or equal to 0. The first three inequalities in assumption (H_4) implies that increased exposure, infection and pathogens in the environment lead to higher incidence rate. The last three inequalities in (H_4) is a common assumption for nonlinear incidence. Possible forms of ρ, f are βE^p and βI^p with constants $\beta, p > 0$, saturation incidences $\frac{\beta E^p}{1+ME^p}$ and $\frac{\beta I^p}{1+MI^p}$ with constants β, p and $M > 0$ [12]; possible forms of g are $\frac{\lambda B}{1+TB}$ and $\eta(1 - e^{-\alpha B})$ with constants $\lambda, \eta, \alpha > 0$ and $T \geqslant 0$ [4,11]. In our system, this assumption regulates $\rho(E)$, $f(I)$ or $g(B)$ as a biologically realistic incidence based on a consequence of saturation effects: when the number of the exposed, the infective, or the environmental pathogen concentration is high, the incidence rate will respond more slowly than the linear increase in E, I and B. The assumption (H_5) states that the pathogen concentration increases with the growing number of the infected and exposed individuals. The assumption (H_6) implies that the more the infected animals are, the more infected animals to be eliminated will be. The assumption (H_7) shows that the disinfection and decaying rate of pathogen is monotonically increasing for $B > 0$.

3. Equilibria and the basic reproduction number

Since the equation $m(S) = 0$ has a single solution S_0 and $\rho(0) = f(0) = g(0) = \varphi(0) = \omega(0) = h(0) = \theta(0) = 0$, it is easy to see that the system (1) admits a unique disease-free equilibrium $P_0 = (S_0, 0, 0, 0)$.

Now we derive the basic reproduction number of system (1) by the next generation matrix formulated in Diekmann et al. [6] and van den Driessche and Watmough [20]. We write

$$\begin{pmatrix} \frac{dE}{dt} \\ \frac{dI}{dt} \\ \frac{dB}{dt} \end{pmatrix} = \begin{pmatrix} n(S)(\rho(E) + f(I) + g(B)) \\ 0 \\ 0 \end{pmatrix} - \begin{pmatrix} (e+\sigma)E \\ -\sigma E + \varphi(I) \\ -h(I) - \omega(E) + \theta(B) \end{pmatrix} = \mathcal{F} - \mathcal{V},$$

where $\mathcal{F}$ denotes the rate of occurrence of new infections and $\mathcal{V}$ denotes the transfer rate of individuals into or out of each population set. The next generation matrix is defined as FV^{-1}, where F and V are 3×3 Jacobian matrices given by

$$F = \begin{pmatrix} n(S_0)\rho_E(0) & n(S_0)f_I(0) & n(S_0)g_B(0) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} e+\sigma & 0 & 0 \\ -\sigma & \varphi_I(0) & 0 \\ -\omega_E(0) & -h_I(0) & \theta_B(0) \end{pmatrix}.$$

The basic reproduction number is defined as the spectral radius of the nonnegative matrix FV^{-1}, so we obtain the basic reproduction number by

$$R_0 = \rho\left(FV^{-1}\right) = \frac{n(S_0)(\rho_E(0)\theta_B(0) + g_B(0)\omega_E(0))}{(e+\sigma)\theta_B(0)} + \frac{\sigma n(S_0)(f_I(0)\theta_B(0) + g_B(0)h_I(0))}{(e+\sigma)\varphi_I(0)\theta_B(0)} = R_0^E + R_0^{EB} + R_0^I + R_0^{IB}, \tag{2}$$

where $R_0^E = \frac{n(S_0)\rho_E(0)}{e+\sigma}$, $R_0^{EB} = \frac{n(S_0)g_B(0)\omega_E(0)}{(e+\sigma)\theta_B(0)}$, $R_0^I = \frac{\sigma n(S_0)f_I(0)}{(e+\sigma)\varphi_I(0)}$, $R_0^{IB} = \frac{\sigma n(S_0)g_B(0)h_I(0)}{(e+\sigma)\varphi_I(0)\theta_B(0)}$. Eq. (2) clearly shows that R_0 depends on two factors: R_0^E (R_0^I) is the average number of the individuals infected by a single exposed (infectious) individual in a fully susceptible population. R_0^{EB} (R_0^{IB}) is the average number of the individuals infected by the pathogen which are excreted into the environment by the exposed (infectious) individual. Therefore, R_0 denotes the total average number of infections.

We now try to establish some sufficient conditions for the existence of the endemic equilibrium (S^*, E^*, I^*, B^*). Since it would be somehow unrealistic to attempt to solve the system (1) in its greatest generality, some additional conditions are imposed on the functional coefficients. Suppose the followings:

(H_8) m is strictly decreasing on $[0,\infty)$ with $m(0) > 0$.
(H_9) $\frac{\rho(E)+f(G(E))+g(H(E))}{(e+\sigma)E}$ is nonincreasing on $(0,\infty)$, where $G(E)$ and $H(E)$ are defined as the following Eq. (4).

The endemic equilibrium $P^*(S^*, E^*, I^*, B^*)$ of system (1) is determined by equations

$$\begin{cases} m(S^*) = n(S^*)\left(\rho(E^*) + f(I^*) + g(B^*)\right), \\ (e+\sigma)E^* = n(S^*)\left(\rho(E^*) + f(I^*) + g(B^*)\right), \\ \sigma E^* = \varphi(I^*), \\ \omega(E^*) + h(I^*) = \theta(B^*). \end{cases} \tag{3}$$

From the last two equations in (3), we can obtain

$$I^* = \varphi^{-1}(\sigma E^*) \triangleq G(E^*), \qquad B^* = \theta^{-1}\left(\omega(E^*) + h(G(E^*))\right) \triangleq H(E^*). \tag{4}$$

Substituting (4) into the first two equations in (3) gives

$$m(S^*) = n(S^*)\left(\rho(E^*) + f(G(E^*)) + g(H(E^*))\right),$$
$$(e+\sigma)E^* = n(S^*)\left(\rho(E^*) + f(G(E^*)) + g(H(E^*))\right).$$

Define

$$\Psi_1(S,E) \triangleq m(S) - n(S)\left(\rho(E) + f(G(E)) + g(H(E))\right),$$
$$\Psi_2(S,E) \triangleq n(S)\left(\rho(E) + f(G(E)) + g(H(E))\right) - (e+\sigma)E.$$

Since $\Psi_1(S,E)$ is strictly decreasing for $S > 0$ and $\Psi_1(0,E)\cdot\Psi_1(S_0,E) < 0$ for $E > 0$, the equation $\Psi_1(S,E) = 0$ can be uniquely solved with S as a function of E for all E. That is to say, there is a function $S = \varepsilon_1(E)$ which satisfies

$$\frac{m(\varepsilon_1(E))}{n(\varepsilon_1(E))} = \rho(E) + f(G(E)) + g(H(E)) \triangleq \Phi(E).$$

Since $\frac{m}{n}$ is strictly decreasing and Φ is strictly increasing, it follows that ε_1 is strictly decreasing. Note that deriving from (4), $\lim_{E\to\frac{\Pi}{\mu}} \varepsilon_1(E) = 0$.

We also note that $\Psi_2(S,E)$ is strictly increasing for $S > 0$ and $\Psi_2(0,E) < 0$ for all E while it is not necessarily true that $\Psi_2(S_0,E) > 0$. So the same approach would not be used to solve the equation $\Psi_2(S,E) = 0$. Since we are searching for a unique endemic equilibrium and for a uniquely corresponding E^*, we only need the local solvability of the equation $\Psi_2(S,E) = 0$ on a certain condition.

We assume for the moment that the equation $\Psi_2(S,E) = 0$ may also be uniquely solved with S as a function of E (locally for E). That is, there is a function $S = \varepsilon_2(E)$ which satisfies

$$n(\varepsilon_2(E)) = \frac{(e+\sigma)E}{\Phi(E)}.$$

Since the function $\frac{(e+\sigma)E}{\Phi(E)}$ is increasing on $(0,\infty)$ and the function n is strictly increasing, it follows that ε_2 is increasing.

Since ε_1 is strictly decreasing, ε_2 is increasing and $\lim_{E\to\frac{\Pi}{\mu}} \varepsilon_1(E) = 0$, the curves defined by $S = \varepsilon_1(E)$ and $S = \varepsilon_2(E)$ have a common point (S^*, E^*) with $S^* > 0, E^* > 0$ on condition that if and only if $\varepsilon_1(0) > \varepsilon_2(0)$ or $n(\varepsilon_1(0)) > n(\varepsilon_2(0))$.

Since $\varepsilon_1(0) = S_0$ and $n(\varepsilon_2(0)) = \lim_{E\to 0}\frac{(e+\sigma)E}{\Phi(E)}$, the existence condition is $n(S_0) > \lim_{E\to 0}\frac{(e+\sigma)E}{\Phi(E)}$. Using L'Hospital rule and the basic reproduction number of the system (1) as defined in (2), this condition can be rewritten as $R_0 > 1$.

We have shown that if the equation $\Psi_2(S, E) = 0$ is solvable with S as a function of E, then the necessary and sufficient condition for the existence of positive (S^*, E^*) is that $R_0 > 1$. In this case, we have

$$\Psi_2(S, E) = (e+\sigma)E\left(\frac{n(S)\Phi(E)}{(e+\sigma)E} - 1\right)$$

and $\Psi_2(S_0, E)$ is positive for E in a vicinity of 0 if $R_0 > 1$. It follows that the equation $\Psi_2(S, E)$ is solvable with S as a function of E (locally for E), that is, we have shown that the existence of positive (S^*, E^*) is equivalent to the valid condition $R_0 > 1$. So system (1) have a unique positive solutions $P^* = (S^*, E^*, I^*, B^*)$.

According to the above discussion, the following results can be summarized:

Theorem 1. *Assume that conditions (H_2)–(H_9) hold. Then there is a unique positive endemic equilibrium $P^* = (S^*, E^*, I^*, B^*)$ of system (1) if and only if $R_0 > 1$.*

Remark. We note that conditions (H_8), (H_9) (combined with $R_0 > 1$) are sufficient for the existence of the endemic equilibrium rather than necessary. For instance, if one assumes that the removal rate $\varphi(I)$ of the infected animals is influenced by the resource which is used to monitor and cull infected animals, such as $\varphi(I) = \mu I + \frac{cI}{a+I}$ with constants $\mu, c, a > 0$, the disease-free equilibrium may coexist with multiple positive endemic equilibria when $R_0 < 1$. On the other hand, if $\frac{\rho}{(e+\sigma)E}$, $\frac{f}{\varphi}$, $\frac{g}{\theta}$, $\frac{\omega}{(e+\sigma)E}$ and $\frac{h}{\varphi}$ are nonincreasing, then assumption (H_9) holds. In fact, at the endemic equilibrium P^*, $f(G(E)) = f(I)$, $g(H(E)) = g(B)$, so we obtain

$$\left(\frac{f(G(E))}{(e+\sigma)E}\right)' = \frac{f_I(I)\frac{1}{\varphi_I(I)}\sigma(e+\sigma)E - (e+\sigma)f(I)}{(e+\sigma)^2E^2} = (e+\sigma)\frac{f_I(I)\varphi(I) - \varphi_I(I)f(I)}{\varphi_I(I)(e+\sigma)^2E^2} \leqslant 0$$

and

$$\begin{aligned}\left(\frac{g(H(E))}{(e+\sigma)E}\right)' &= \frac{g_B(B)\frac{1}{\theta_B(B)}(\omega_E(E) + h_I(I)\frac{1}{\varphi_I(I)}\sigma)(e+\sigma)E - (e+\sigma)g(B)}{(e+\sigma)^2E^2}\\ &= \frac{g_B(B)\theta_E(B)(e+\sigma)E - g(B)\theta_B(B)(e+\sigma)}{\theta_B(B)(e+\sigma)^2E^2} \leqslant 0,\end{aligned}$$

where

$$E\theta_E(B) - \theta(B) = E\omega_E(E) - \omega(E) + \frac{h_I\varphi(I) - \varphi_I(I)h(I)}{\varphi_I(I)} \leqslant 0,$$

which is equivalent to $E\theta_E(B) \leqslant \theta(B)$. Therefore, assumption (H_9) is satisfied.

4. Stability of the disease-free and the endemic equilibrium

In this section, we study the global stability of disease-free equilibrium and the endemic equilibrium of system (1). It is important for us to understand the extinction and persistence of infectious animal diseases.

4.1. Stability of the disease-free equilibrium

In this subsection, we investigate the global stability of disease-free equilibrium when the basic reproduction number $R_0 \leqslant 1$. It is perhaps also worth noting that the stability of the equilibria depends essentially on the behavior of the function $\frac{\rho}{(e+\sigma)E}$, $\frac{f}{\varphi}$, $\frac{g}{\theta}$, $\frac{\omega}{(e+\sigma)E}$, $\frac{h}{\varphi}$, and then we make the following assumption:

(H_{10}) $\frac{\rho}{(e+\sigma)E}$, $\frac{f}{\varphi}$, $\frac{g}{\theta}$, $\frac{\omega}{(e+\sigma)E}$ and $\frac{h}{\varphi}$ are nonincreasing on $(0, \infty)$.

Theorem 2. *Assume that conditions (H_1)–(H_7) and (H_{10}) are satisfied. If $R_0 \leqslant 1$, the disease-free equilibrium P_0 of system (1) is globally asymptotically stable in Ω; if $R_0 > 1$, P_0 is unstable and system (1) is uniformly persistent in the interior of Ω.*

Proof. Since the function $\frac{\rho}{\phi}$, $\frac{f}{\varphi}$, $\frac{g}{\theta}$, $\frac{\omega}{\phi}$ and $\frac{h}{\varphi}$ are nonincreasing, we have

$$\frac{n(S)\rho(E)}{(e+\sigma)E} \leqslant \lim_{E\to 0}\frac{n(S_0)\rho(E)}{(e+\sigma)E} = \frac{n(S_0)\rho_E(0)}{e+\sigma} \triangleq b_1,$$

$$\frac{n(S)f(I)}{\varphi(I)} \leqslant \lim_{I\to 0}\frac{n(S_0)f(I)}{\varphi(I)} = \frac{n(S_0)f_I(0)}{\varphi_I(0)} \triangleq b_2,$$
$$\frac{n(S)g(B)}{\theta(B)} \leqslant \lim_{B\to 0}\frac{n(S_0)g(B)}{\theta(B)} = \frac{n(S_0)g_B(0)}{\theta_B(0)} \triangleq b_3,$$
$$\frac{\omega(E)}{(e+\sigma)E} \leqslant \lim_{E\to 0}\frac{\omega(E)}{(e+\sigma)E} = \frac{\omega_E(0)}{e+\sigma}, \qquad \frac{h(I)}{\varphi(I)} \leqslant \lim_{I\to 0}\frac{h(I)}{\varphi(I)} = \frac{h_I(0)}{\varphi_I(0)}.$$

Define

$$J = \begin{pmatrix} 1 & 0 & 0 \\ -\frac{\sigma}{e+\sigma} & 1 & 0 \\ -\frac{\omega_E(0)}{e+\sigma} & -\frac{h_I(0)}{\varphi_I(0)} & 1 \end{pmatrix}, \qquad (a_1,a_2,a_3) = (b_1,b_2,b_3)J^{-1}.$$

Thus, $a_1 = R_0 \leqslant 1$, we define the Lyapunov function

$$L = R_0E + a_2I + a_3B.$$

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

$$\begin{aligned}\frac{dL}{dt} &= R_0\left(\frac{n(S)\rho(E)}{(e+\sigma)E}, \frac{n(S)f(I)}{\varphi(I)}, \frac{n(S)g(B)}{\theta(B)}\right)\big((e+\sigma)E, \varphi(I), \theta(B)\big)^{\tau} \\ &\quad - (R_0,a_2,a_3)\begin{pmatrix} 1 & 0 & 0 \\ -\frac{\sigma}{e+\sigma} & 1 & 0 \\ -\frac{\omega(E)}{(e+\sigma)E} & -\frac{h(I)}{\varphi(I)} & 1 \end{pmatrix}\big((e+\sigma)E, \varphi(I), \theta(B)\big)^{\tau} \\ &\leqslant R_0\left(\frac{n(S_0)\rho_E(0)}{e+\sigma}, \frac{n(S_0)f_I(0)}{\varphi_I(0)}, \frac{n(S_0)g_B(0)}{\theta_B(0)}\right)\big((e+\sigma)E, \varphi(I), \theta(B)\big)^{\tau} \\ &\quad - (R_0,a_2,a_3)\begin{pmatrix} 1 & 0 & 0 \\ -\frac{\sigma}{e+\sigma} & 1 & 0 \\ -\frac{\omega_E}{e+\sigma} & -\frac{h_I}{\varphi_I} & 1 \end{pmatrix}\big((e+\sigma)E, \varphi(I), \theta(B)\big)^{\tau} \\ &= (R_0-1)(b_1,b_2,b_3)\big((e+\sigma)E, \varphi(I), \theta(B)\big)^{\tau} \leqslant 0.\end{aligned}$$

Let

$$\Psi = \left\{(S,E,I,B) \in \Omega \;\middle|\; \frac{dL}{dt} = 0\right\}.$$

If $R_0 < 1$, $\frac{dL}{dt} = 0$ implies that $n(S)(\rho(E) + f(I) + g(B)) = 0$. And then $S = S_0$, $E = 0, I = 0, B = 0$. Therefore, the largest invariant set of Ψ is P_0. Supposing $R_0 = 1$, $\frac{dL}{dt} = 0$ implies for $E, I > 0$ that

$$\frac{n(S)\rho(E)}{(e+\sigma)E} = \frac{n(S_0)\rho(E)}{(e+\sigma)E} = b_1, \qquad \frac{n(S)f(I)}{\varphi(I)} = \frac{n(S_0)f(I)}{\varphi(I)} = b_2.$$

So $S = S_0$, P_0 is the only invariant set of Ψ. By LaSalle's invariance principle, P_0 is globally asymptotically stable in Ω if $R_0 \leqslant 1$.

If $R_0 > 1$, $\frac{dL}{dt} > 0$ in a neighborhood of P_0 in the interior of Ω, solutions sufficiently close to P_0 will go away from P_0, which implies that P_0 is unstable. With a uniform persistence result from [7], it can be shown that system (1) is uniformly persistent when $R_0 > 1$. □

4.2. Stability of the endemic equilibrium

In this subsection, it is assumed that the system (1) admits a positive endemic equilibrium and shows its global asymptotic stability in the interior of the feasible region Ω. However, it should be pointed out that conditions (H_8) and (H_9) are sufficient for the existence of the endemic equilibrium rather than necessary. Therefore, we do not assume that (H_8) and (H_9) are satisfied to establish our result. For our purpose, we assume that

(A_1) For $S \geqslant 0$,

$$\big(m(S) - m(S^*)\big)\big(n(S) - n(S^*)\big) < 0 \quad \text{for } S \neq S^*.$$

(A_2) For $E, I > 0$,

$$\big(\omega(E) - \omega(E^*)\big)\left(\frac{\omega(E)}{(e+\sigma)E} - \frac{\omega(E^*)}{(e+\sigma)E^*}\right) \leqslant 0,$$
$$\big(h(I) - h(I^*)\big)\left(\frac{h(I)}{\varphi(I)} - \frac{h(I^*)}{\varphi(I^*)}\right) \leqslant 0.$$

(A_3) For $E, I, B > 0$,

$$\big(\rho(E) - \rho(E^*)\big)\left(\frac{\rho(E)}{(e+\sigma)E} - \frac{\rho(E^*)}{(e+\sigma)E^*}\right) \leqslant 0,$$
$$\big(f(I) - f(I^*)\big)\left(\frac{f(I)}{\varphi(I)} - \frac{f(I^*)}{\varphi(I^*)}\right) \leqslant 0,$$
$$\big(g(B) - g(B^*)\big)\left(\frac{g(B)}{\theta(B)} - \frac{g(B^*)}{\theta(B^*)}\right) \leqslant 0.$$

In fact, if assumption (H_{10}) holds, it is easy to find that above assumptions (A_2) and (A_3) are satisfied.

Theorem 3. *Assume that conditions (H_1)–(H_7) and (A_1)–(A_3) are satisfied. If $R_0 > 1$, the endemic equilibrium (S^*, E^*, I^*, B^*) is globally asymptotically stable in the interior of Ω.*

Proof. For system (1), we consider the following Lyapunov function:

$$L = c_1 \int_{S^*}^{S} \frac{n(x) - n(S^*)}{n(x)}\,dx + c_1\big(E - E^* - E^* \ln\big(E/E^*\big)\big) + c_2 \int_{I^*}^{I} \frac{\varphi(x) - \varphi(I^*)}{\varphi(x)}\,dx + c_3 \int_{B^*}^{B} \frac{\theta(x) - \theta(B^*)}{\theta(x)}\,dx.$$

Here $c_i > 0$, $i = 1, 2, 3$, are constants to be specified later. Differentiating L with t along solution curves of system (1) and using equilibrium equations (3) to simplify, one can obtain

$$\begin{aligned}
\frac{dL}{dt} &= c_1\left(m(S) - (e+\sigma)E - m(S)\frac{n(S^*)}{n(S)} + n\big(S^*\big)\big(\rho(E) + f(I) + g(B)\big) + (e+\sigma)E^* - n(S)\big(\rho(E) + f(I) + g(B)\big)\frac{E^*}{E}\right) \\
&\quad + c_2\left(\sigma E - \varphi(I) - \sigma E\frac{\varphi(I^*)}{\varphi(I)} + \varphi\big(I^*\big)\right) + c_3\left(\omega(E) + h(I) - \theta(B) - \big(\omega(E) + h(I)\big)\frac{\theta(B^*)}{\theta(B)} + \theta\big(B^*\big)\right) \\
&= c_1\big(m(S) - m\big(S^*\big)\big)\left(1 - \frac{n(S^*)}{n(S)}\right) + c_1 n\big(S^*\big)\rho\big(E^*\big)\left(2 + \frac{\rho(E)}{\rho(E^*)} - \frac{n(S^*)}{n(S)} - \frac{E}{E^*} - \frac{n(S)\rho(E)E^*}{n(S^*)\rho(E^*)E}\right) \\
&\quad + c_1 n\big(S^*\big) f\big(I^*\big)\left(2 + \frac{f(I)}{f(I^*)} - \frac{n(S^*)}{n(S)} - \frac{E}{E^*} - \frac{n(S)f(I)E^*}{n(S^*)f(I^*)E}\right) \\
&\quad + c_1 n\big(S^*\big) g\big(B^*\big)\left(2 + \frac{g(B)}{g(B^*)} - \frac{n(S^*)}{n(S)} - \frac{E}{E^*} - \frac{n(S)g(B)E^*}{n(S^*)g(B^*)E}\right) \\
&\quad + c_2\sigma E^*\left(\frac{E}{E^*} - \frac{\varphi(I)}{\varphi(I^*)} - \frac{E\varphi(I^*)}{E^*\varphi(I)} + 1\right) + c_3\omega\big(E^*\big)\left(\frac{\omega(E)}{\omega(E^*)} - \frac{\theta(B)}{\theta(B^*)} - \frac{\omega(E)\theta(B^*)}{\omega(E^*)\theta(B)} + 1\right) \\
&\quad + c_3 h\big(I^*\big)\left(\frac{h(I)}{h(I^*)} - \frac{\theta(B)}{\theta(B^*)} - \frac{h(I)\theta(B^*)}{h(I^*)\theta(B)} + 1\right).
\end{aligned}$$

Using (A_1), we obtain

$$\big(m(S) - m\big(S^*\big)\big)\left(1 - \frac{n(S^*)}{n(S)}\right) \leqslant 0. \tag{5}$$

We consider the function $\nu(x) = 1 - x + \ln x$, which is nonpositive for $x > 0$ and $\nu(x) = 0$ if and only if $x = 1$. By inequality (A_3), we have

$$\begin{aligned}
\frac{E}{E^*} - \frac{\varphi(I)}{\varphi(I^*)} - \frac{E\varphi(I^*)}{E^*\varphi(I)} + 1 &= \nu\left(\frac{E\varphi(I^*)}{E^*\varphi(I)}\right) + \frac{E}{E^*} - \ln\frac{E}{E^*} - \frac{\varphi(I)}{\varphi(I^*)} + \ln\frac{\varphi(I)}{\varphi(I^*)} \\
&\leqslant \frac{E}{E^*} - \ln\frac{E}{E^*} - \frac{\varphi(I)}{\varphi(I^*)} + \ln\frac{\varphi(I)}{\varphi(I^*)},
\end{aligned} \tag{6}$$

$$\frac{\omega(E)}{\omega(E^*)}-\frac{\theta(B)}{\theta(B^*)}-\frac{\omega(E)\theta(B^*)}{\omega(E^*)\theta(B)}+1=\left(\frac{\omega(E)}{\omega(E^*)}-1\right)\left(1-\frac{\omega(E^*)E}{\omega(E)E^*}\right)+\nu\left(\frac{\omega(E)\theta(B^*)}{\omega(E^*)\theta(B)}\right)+\nu\left(\frac{\omega(E^*)E}{\omega(E)E^*}\right)$$
$$+\frac{E}{E^*}-\ln\frac{E}{E^*}-\frac{\theta(B)}{\theta(B^*)}+\ln\frac{\theta(B)}{\theta(B^*)}$$
$$\leqslant \frac{E}{E^*}-\ln\frac{E}{E^*}-\frac{\theta(B)}{\theta(B^*)}+\ln\frac{\theta(B)}{\theta(B^*)}, \tag{7}$$

and

$$\frac{h(I)}{h(I^*)}-\frac{\theta(B)}{\theta(B^*)}-\frac{h(I)\theta(B^*)}{h(I^*)\theta(B)}+1=\left(\frac{h(I)}{h(I^*)}-1\right)\left(1-\frac{h(I^*)\varphi(I)}{h(I)\varphi(I^*)}\right)+\nu\left(\frac{h(I)\theta(B^*)}{h(I^*)\theta(B)}\right)+\nu\left(\frac{h(I^*)\varphi(I)}{h(I)\varphi(I^*)}\right)$$
$$+\frac{\varphi(I)}{\varphi(I^*)}-\ln\frac{\varphi(I)}{\varphi(I^*)}-\frac{\theta(B)}{\theta(B^*)}+\ln\frac{\theta(B)}{\theta(B^*)}$$
$$\leqslant \frac{\varphi(I)}{\varphi(I^*)}-\ln\frac{\varphi(I)}{\varphi(I^*)}-\frac{\theta(B)}{\theta(B^*)}+\ln\frac{\theta(B)}{\theta(B^*)}. \tag{8}$$

Similarly, using assumption (A_2), we obtain

$$2+\frac{\rho(E)}{\rho(E^*)}-\frac{n(S^*)}{n(S)}-\frac{E}{E^*}-\frac{n(S)\rho(E)E^*}{n(S^*)\rho(E^*)E}=\left(\frac{\rho(E)}{\rho(E^*)}-1\right)\left(1-\frac{\rho(E^*)E}{\rho(E)E^*}\right)+\nu\left(\frac{n(S^*)}{n(S)}\right)$$
$$+\nu\left(\frac{n(S)\rho(E)E^*}{n(S^*)\rho(E^*)E}\right)+\nu\left(\frac{\rho(E^*)E}{\rho(E)E^*}\right)$$
$$\leqslant 0,$$
$$2+\frac{f(I)}{f(I^*)}-\frac{n(S^*)}{n(S)}-\frac{E}{E^*}-\frac{n(S)f(I)E^*}{n(S^*)f(I^*)E}=\left(\frac{f(I)}{f(I^*)}-1\right)\left(1-\frac{f(I^*)\varphi(I)}{f(I)\varphi(I^*)}\right)+\nu\left(\frac{n(S^*)}{n(S)}\right)$$
$$+\nu\left(\frac{n(S)f(I)E^*}{n(S^*)f(I^*)E}\right)+\nu\left(\frac{f(I^*)\varphi(I)}{f(I)\varphi(I^*)}\right)$$
$$+\frac{\varphi(I)}{\varphi(I^*)}-\ln\frac{\varphi(I)}{\varphi(I^*)}-\frac{E}{E^*}+\ln\frac{E}{E^*}$$
$$\leqslant \frac{\varphi(I)}{\varphi(I^*)}-\ln\frac{\varphi(I)}{\varphi(I^*)}-\frac{E}{E^*}+\ln\frac{E}{E^*} \tag{9}$$

and

$$2+\frac{g(B)}{g(B^*)}-\frac{n(S^*)}{n(S)}-\frac{E}{E^*}-\frac{n(S)g(B)E^*}{n(S^*)g(B^*)E}\leqslant\frac{\theta(B)}{\theta(B^*)}-\ln\frac{\theta(B)}{\theta(B^*)}-\frac{E}{E^*}+\ln\frac{E}{E^*}. \tag{10}$$

Let

$$x_1=E,\qquad x_2=\varphi(I),\qquad x_3=\theta(B).$$

Combining (5)–(10) and using the definition of m_{ij} in (12), we have

$$\frac{dL}{dt}\leqslant\sum_{i,j=1}^{3}c_i m_{ij}\left(\frac{x_j}{x_j^*}-\ln\frac{x_j}{x_j^*}-\frac{x_i}{x_i^*}+\ln\frac{x_i}{x_i^*}\right), \tag{11}$$

where

$$M=(m_{ij})_{3\times3}=\begin{pmatrix}1 & n(S^*)f(I^*) & n(S^*)g(B^*)\\ \sigma E^* & 1 & 0\\ \omega(E^*) & h(I^*) & 1\end{pmatrix}. \tag{12}$$

According to the methods and conclusions in [9], since M is irreducible, there exist constants $c_i>0$, $i=1,2,3$ such that

$$\sum_{i,j=1}^{3}c_i m_{ij}\left(\frac{x_j}{x_j^*}-\ln\frac{x_j}{x_j^*}-\frac{x_i}{x_i^*}+\ln\frac{x_i}{x_i^*}\right)\equiv 0. \tag{13}$$

Moreover, the equality $\frac{dL}{dt}=0$ holds if and only if $S=S^*$, $E=E^*$, $I=I^*$, $B=B^*$. Thus, P^* is the only invariant set of model (1) in $\{(S,E,I,B)\in\Omega\colon \frac{dL}{dt}=0\}$, so the endemic equilibrium P^* has the global asymptotic stability in the interior of Ω by LaSalle's Invariance Principle. □

5. Several specific examples

To illustrate the usefulness of the results, we consider the following the nonlinear system:

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \alpha SE - \beta SI - \lambda Sg(B) - \mu S, \\ \dfrac{dE}{dt} = \alpha SE + \beta SI + \lambda Sg(B) - (\mu + \sigma)E, \\ \dfrac{dI}{dt} = \sigma E - (c + \mu)I, \\ \dfrac{dB}{dt} = \gamma(I + E) - dB, \end{cases} \tag{14}$$

where Λ, α, β, λ, μ, d, γ and $\sigma > 0$, $m(S) = \Lambda - \mu S$, $n(S) = S$, $\rho(E) = \alpha E$, $f(I) = \beta I$, $\varphi(I) = (c + \mu)I$, $\omega(E) = \gamma E$, $h(I) = \gamma I$, $\theta(B) = dB$, the function $g(B)$ is strictly monotonously increasing. It is easy to see that assumptions (H_1)–(H_7), (H_8) and (A_1) are automatically satisfied.

If $g(B) = \frac{\lambda B}{1+TB}$, assumptions (H_{10}) and (A_3) hold. In fact, assumptions (H_{10}) and (A_3) can be satisfied by the following nonlinear incidence functions:

$$\frac{X_i^p}{1 + T_i X_i^p}, \quad i = 1, 2, 3,$$

where $X_1 = E$, $X_2 = I$, $X_3 = B$, $0 < p \leqslant 1$, $T_i \geqslant 0$.

When $g(B) = \eta(1 - e^{-\alpha B})$, one can obtain

$$\left(\frac{1 - e^{-\alpha B}}{B}\right)' = \frac{(\alpha B + 1)e^{-\alpha B} - 1}{B^2}.$$

Let $\Gamma(B) = (\alpha B + 1)e^{-\alpha B} - 1$, so $\Gamma' = -\alpha^2 B e^{-\alpha B} \leqslant 0$ for $B \geqslant 0$, which means that $\frac{\Gamma(B)}{B^2}$ is a monotonously decreasing function. So there is the following result:

$$\frac{\Gamma(B)}{B^2} \leqslant \lim_{B \to 0} \frac{(\alpha B + 1)e^{-\alpha B} - 1}{B^2} = -\frac{\alpha^2}{2},$$

$\frac{1-e^{-\alpha B}}{B}$ is also a monotonously decreasing function, and then (H_{10}) and (A_3) are satisfied. If $g(B) = k \ln(1 + \frac{\lambda B}{k})$ [13], using the above method, we can prove that the function $\frac{\ln(1+\frac{\lambda B}{k})}{B}$ is monotonously decreasing, which implies that assumptions (H_{10}) and (A_3) hold. In these cases, if $R_0 \leqslant 1$, the disease-free equilibrium is globally asymptotically stable, and system (14) has a unique endemic equilibrium, which is also globally asymptotically stable if $R_0 > 1$.

6. Conclusions and discussions

Animal diseases and zoonoses not only have resulted enormous economic losses, but also has become a serious public health problem around the world. In order to better understand the dynamics of these disease transmission, a general SEIB dynamic model with nonlinear demographic functions, nonlinear incidence and removal functions is proposed, based on the common characteristics of infectious animal diseases. Under very general biological assumptions, we have derived the basic reproduction number R_0 and analyzed the existence and the global stability of equilibria. The results indicate that the disease-free equilibrium is globally asymptotically stable if $R_0 \leqslant 1$, which means that the extinction of infectious diseases is independent of initial sizes of the populations and the disease will be eliminated; for $R_0 > 1$, there is a unique positive endemic equilibrium which is globally asymptotically stable, implying that the disease always remains endemic and persists at a unique endemic equilibrium, no matter how small the size of the initial infection is.

On the other hand, nonlinear functions are used in model (1) to describe incidence and removal rate. These nonlinear elements can lead to complex dynamic behavior. In fact, the stability of equilibria depends on the properties of the function $\frac{\rho}{(e+\sigma)E}$, $\frac{f}{\varphi}$, $\frac{g}{\theta}$, $\frac{\omega}{(e+\sigma)E}$ and $\frac{h}{\varphi}$. If these functions are nonincreasing, dynamical properties of the model (1) cannot be an oscillations. However, if these functions (at least one function) are strictly monotonic increasing, model (1) may have other dynamical features such as oscillations. We leave other dynamical properties for further research.

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References

[1] L.J.S. Allen, D.A. Flores, R.K. Ratnayake, J.R. Herbold, Discrete-time deterministic and stochastic models for the spread of rabies, Appl. Math. Comput. 132 (2002) 271–292.
[2] G.G. Alton, The epidemiology of brucella melitensis in sheep and goats, in: J.M. Verger, M. Plommet (Eds.), Brucella Melitensis, in: CEC Seminars, vol. 32, Martinus Nijoff, Dordrecht–Boston–Lancaster, 1985, pp. 187–196.
[3] D. Andrew, M. Mary, The population dynamics of brucellosis in the Yellowstone National Park, Ecology 77 (4) (1996) 1026–1036.
[4] R. Breban, J.M. Drake, D.E. Stallknecht, P. Rohani, The role of environmental transmission in recurrent avian influenza epidemics, PLoS Comput. Biol. 5 (4) (2009) e1000346, http://dx.doi.org/10.1371/journal.pcbi.1000346.
[5] R.S. Cantrell, C. Cosner, W.F. Fagan, Brucellosis, boties, and brainworms: The impact of edge habitats on pathogen transmission and species extinction, J. Math. Biol. 42 (2001) 95–119.
[6] O. Diekmann, J.A.P. Heesterbeek, J.A.J. Metz, On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations, J. Math. Biol. 28 (1990) 365–382.
[7] 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.
[8] J. González-Guzmán, R. Naulin, Analysis of a model of bovine brucellosis using singular perturbations, J. Math. Biol. 33 (1994) 211–234.
[9] H.B. Guo, M.Y. Li, Z.S. Shuai, Global dynamics of a general class of multistage models for infectious diseases, SIAM J. Appl. Math. 72 (1) (2012) 261–279.
[10] K. Hampson, J. Dushoff, J. Bingham, G. Brückner, Y.H. Ali, A. Dobson, Synchronous cycles of domestic dog rabies in sub-Saharan Africa and the impact of control efforts, Proc. Natl. Acad. Sci. USA 104 (2007) 7717–7727.
[11] Q. Hou, X.D. Sun, J. Zhang, Y.J. Liu, Y.M. Wang, Z. Jin, Modeling the transmission dynamics of sheep brucellosis in inner Mongolia autonomous region, China, Math. Biosci. 242 (2013) 51–58.
[12] W.M. Liu, S.A. Levin, Influence of nonlinear incidence rates upon the behavior of SIRS epidemiological models, J. Math. Biol. 23 (1986) 187–204.
[13] H. McCallum, N. Barlow, J. Hone, How should pathogen transmission be modelled?, Trends Ecol. Evol. 16 (6) (2001) 295–300.
[14] P. Nicoletti, The epidemiology of bovine brucellosis, Adv. Vet. Sci. Comp. Med. 24 (1980) 69–98.
[15] G. Pappas, P. Papadimitriou, N. Akritidis, L. Christou, E.V. Tsianos, The new global map of human brucellosis, Lancet, Infect. Dis. 6 (2006) 91–99.
[16] M.J. Perterson, W.E. Grant, D.S. Davis, Simulation of host-parasite interactions within a resource management framework: import of brucellosis on bison population dynamics, Ecol. Model. 54 (1991) 299–320.
[17] M.G. Roberts, The dynamics and control of bovine tuberculosis in possums, Math. Med. Biol. 9 (1) (1992) 19–28.
[18] S. Roy, Terry F. McElwain, Y. Wan, A network control theory approach to modeling and optimal control of zoonoses: Case study of brucellosis transmission in sub-Saharan Africa, PLoS Negl. Trop. Dis. 5 (10) (2011) e1259, http://dx.doi.org/10.1371/journal.pntd.0001259.
[19] M.M.H. Sewell, D.W. Brocklesby, Animal Diseases in the Tropics, fourth ed., Baillie're Tindall, London, 1990.
[20] 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.
[21] WHO, Global tuberculosis control, epidemiology, strategy, financing, WHO report 2009, http://www.who.int/tb/publications/global_report/2009/en/index.html, 2009.