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Global properties of vector–host disease models with time delays

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Abstract Since there exist extrinsic and intrinsic incubation periods of pathogens in the feedback interactions between the vectors and hosts, it is necessary to consider the incubation delays in vector–host disease transmission dynamics. In this paper, we propose vector–host disease models with two time delays, one describing the incubation period in the vector population and another representing the incubation period in the host population. Both distributed and discrete delays are used. By constructing suitable Liapunov functions, we obtain sufficient conditions for the global stability of the endemic equilibria of these models. The analytic results reveal that the global dynamics of such vector–host disease models with time delays are completely determined by the basic reproduction number. Some specific cases with discrete delay are studied and the corresponding results are improved.

Keywords Vector–host disease model · Time delay · Global stability · Liapunov functional · Basic reproduction number

Mathematics Subject Classification 92D30

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1 Introduction

The transmission of vector-borne diseases depends upon the attributes and requirements of at least three different living organisms: the pathologic agent (a virus, protozoa, bacteria, or helminth), the vector (mosquitoes, sandflies, or ticks), and the human host. For example, dengue, malaria, Raft Valley fever, West Nile virus, and Yellow fever are transmitted through mosquitoes; sandflies are the vector for Leishmaniasis; and Lyme disease is a tick-borne disease (Graz 1999). Due to their high morbidity and mortality, vector-borne diseases continue to pose a significant burden worldwide (Gubler 1998). In recent years, a number of factors such as global warming and environment change have contributed to the emergence and resurgence of vector-borne diseases (Harrus and Baneth 2005; Sutherst 2004).

Mathematical modeling of vector-borne diseases started with the pioneer work of Ross (1911) who constructed a system of two ordinary differential equations to describe changes in densities of susceptible and infected vectors (mosquitoes) and hosts (humans) and provided a quantitative understanding of the transmission dynamics of malaria. Macdonald (1957) extended Ross' basic model and introduced the concept of basic reproduction number, which is defined as the average number of secondary cases produced by an index case during its infectiousness period. The Ross–Macdonald model has been extended by many researchers to include more features, see Aron and May (1982), Anderson and May (1991), Chitnis et al. (2006), Gao and Ruan (2012), Koella (1991), Lou and Zhao (2010), Smith and McKenzie (2004), and the references cited therein. The modeling scheme has also been modified to describe the transmission dynamics of some other vector-borne diseases, such as Chagas disease (Velasco-Hernández 1994), dengue (Esteva and Vargas 1998), Leishmaniasis (Dye 1996), West Nile virus (Bowman et al. 2005), etc.

Time delays occur naturally in vector-borne diseases due to processes in the development of pathogens in the vector population that take a significant amount of time, particularly compared to the lifespan of the vector population. This incubation period is called the *extrinsic incubation period*. Besides the incubation period in the vector population, vector-borne pathogens also have an incubation period within the host population, which is called the *intrinsic incubation period*. In Ruan et al. (2008), generalized the Ross–Macdonald type model by incorporating two discrete time delays to represent the extrinsic incubation period in the vector population and the intrinsic incubation period in the host population, respectively. They investigated the dynamics of the generalized Ross–Macdonald model with two delays by evaluating the basic reproduction number and showed that prolonging the incubation periods in either host (human) or vector (mosquito) population could reduce the prevalence of infection. Recently, vector–host disease models with time delays have been extensively studied by some researchers, see Atkinson et al. (2007), Fan et al. (2010), Lou and Zhao (2010), Martcheva and Prosper (2013), and Vargas-De-León (2012). Since the incubation latencies in both the vector and host populations may differ from individual to individual, it has been suggested that distributed delays may be more suitable to describe such latent periods (Takeuchi et al. 2000; Wei et al. 2008; Xiao and Zou 2013). In this paper, we extend the Ross–Macdonald type vector–host disease model with two discrete delays in Ruan et al. (2008) by using distributed delays to describe

the extrinsic and intrinsic incubation periods and by including both the susceptible and infective vectors and hosts. By constructing suitable Liapunov functionals we investigate the global dynamics of the model and its variants.

This paper is organized as follows. In Sect. 2 we introduce the vector-borne disease models with distributed delays. We discuss the equilibria and their stability in Sect. 2.1, show the persistence of the disease in Sect. 2.2, and establish the global stability of the disease-free and the endemic equilibria, respectively, in Sect. 2.3. In Sect. 3, we consider an extended model with direct transmission and establish the global stability of the endemic equilibrium. In Sect. 4, we discuss the special case when the distributed delays reduce to discrete delays and present some results on the global stability in the Ross–Macdonald vector–host model with discrete delays, which are related to the models considered in Ruan et al. (2008) and Wei et al. (2008). Our results show that the global stability of the positive equilibria in these models is completely determined by the basic reproduction number.

2 The vector–host model with distributed delay

Divide the host population into three compartmental classes: the number of susceptible individuals $S_h(t)$, the number of infective individuals $I_h(t)$, and the number of recovered or immune individuals $R_h(t)$. The vector population is divided into two groups: susceptible vectors $S_v(t)$, and infective vectors $I_v(t)$. Since we focus on the effect of incubation periods on the global dynamics, without loss of generality we assume that the total populations of hosts N_h and vectors N_v are constants. Similar assumption has been made in Ross (1911), Macdonald (1957), Anderson and May (1991), Ruan et al. (2008), Wei et al. (2008), Xiao and Zou (2013), etc. Let Λ_h and Λ_v denote the birth rate of the hosts and vectors, respectively, and μ_h, μ_v are the nature death rate of the hosts and vectors, respectively. In addition, let α_h be the recovery rate of the host population. b is the average rate of biting on hosts by a single vector (number of bites per unit time), and the hosts are always sufficient in abundance, so that it is reasonable to assume that the biting rate b is constant. Thus, the number of bites on hosts per unit time per host is $\frac{b}{N_h}$. A susceptible vector becomes infected upon biting an infected host I_h with a biting rate b and the probability of transmission of the disease is given by $\widetilde{\beta}_v$. Furthermore, it is assumed that infected vectors bite at the same rate as susceptible vectors, namely b. We denote by $\widetilde{\beta}_h$ the probability of transmission of the pathogen from infected vector to susceptible host.

Since there exist extrinsic and intrinsic incubation periods for the interactions between vectors and hosts (Ruan et al. 2008), it is reasonable to consider the incubation delays in both vectors and hosts. In reality, however, the incubation period is not a number but an interval during which the maturation of the parasite occurs in different hosts and vectors. Hence, we assume that the incubation period is a distributed parameter (see Cushing 1977; McDonald 1978). Since it is possible that some hosts recovered from parasitemia during this incubation period (Smith and McKenzie 2004), of those individuals recovered from infected τ unit times ago, only a proportion $\frac{b}{N_h}\widetilde{\beta}_h \int_0^{\tau_h} \widetilde{g}_h(\tau) S_h(t-\tau) I_v(t-\tau) e^{-(\alpha_h+\mu_h)\tau} d\tau$ is infectious at the present time t. Here the kernel function $\widetilde{g}_h(\tau)$ represents the probability distribution of the infec-

tivity of the parasites in host population where the time taken to become infectious is τ, which is a random variable. Similarly, based on the facts that the average total rate of contacts between hosts and vectors must be conserved (Ross 1911, P_{667}), we can show that $\frac{b}{N_h}\widetilde{\beta}_v \int_0^{\tau_v} \widetilde{g}_v(\tau) S_v(t-\tau) I_h(t-\tau) e^{-\mu_v \tau} d\tau$ gives the incidence of new cases of infection for the vectors at the present time t. The kernel function $\widetilde{g}_v(\tau)$ represents the infectivity on susceptible vectors during the intrinsic incubation period. Here, τ_h and τ_v are, respectively, the upper limits of the parasite's incubation periods in hosts and vectors. The terms $e^{-\mu_v \tau}$ and $e^{(-\mu_h+\alpha_h)\tau}$ account for the individual survival probability in vectors and hosts, respectively.

For simplicity, we introduce the following notation:

$$\beta_h = \frac{b}{N_h}\widetilde{\beta}_h, \quad \beta_v = \frac{b}{N_h}\widetilde{\beta}_v, \quad g_v(\tau) = e^{-\mu_v \tau}\widetilde{g}_v(\tau), \quad g_h(\tau) = \widetilde{g}_h(\tau) e^{-(\mu_h+\alpha_h)\tau}.$$

Moreover, we impose the following assumptions.

(i) $g_v(\tau) \geq 0$ and $g_h(\tau) \geq 0$ for $0 \leq \tau \leq h$ and are continuous on $[0, h]$;
(ii) $g_v(\tau)$ and $g_h(\tau)$ satisfy

$$\int_0^{\tau_v} g_v(\tau) d\tau = a_v, \qquad \int_0^{\tau_h} g_h(\tau) d\tau = a_h.$$

Let $\mathcal{C}$ denote the Banach space $C([-h, 0], \mathbb{R})$ of continuous functions mapping the interval $[-h, 0]$ into $\mathbb{R}$ equipped with the sup-norm $||\psi|| = \sup_{-h \leq \theta \leq 0} |\psi(\theta)|$. The nonnegative cone of $\mathcal{C}$ is defined as $\mathcal{C}^+ = C([-h, 0], \mathbb{R}_+)$. Under the above assumptions, our model under consideration with distributed incubation delays can be formulated as follows:

$$\begin{aligned}
\frac{dS_h(t)}{dt} &= \Lambda_h - \beta_h S_h(t) I_v(t) - \mu_h S_h(t), \\
\frac{dI_h(t)}{dt} &= \beta_h \int_0^{\tau_h} g_h(\tau) S_h(t-\tau) I_v(t-\tau) d\tau - (\mu_h + \alpha_h) I_h(t), \\
\frac{dR_h(t)}{dt} &= \alpha_h I_h(t) - \mu_h R_h(t), \\
\frac{dS_v(t)}{dt} &= \Lambda_v - \beta_v S_v(t) I_h(t) - \mu_v S_v(t), \\
\frac{dI_v(t)}{dt} &= \beta_v \int_0^{\tau_v} g_v(\tau) S_v(t-\tau) I_h(t-\tau) d\tau - \mu_v I_v(t).
\end{aligned}$$

Since the variable $R_h(t)$ does not appear in the other equations, it is sufficient to analyze the behavior of the model without the equation of $R_h(t)$. Thus, we investigate the following system:

$$\begin{cases}
\frac{dS_h(t)}{dt} = \Lambda_h - \beta_h S_h(t) I_v(t) - \mu_h S_h(t), \\
\frac{dI_h(t)}{dt} = \beta_h \int_0^{\tau_h} g_h(\tau) S_h(t-\tau) I_v(t-\tau) d\tau - (\mu_h + \alpha_h) I_h(t), \\
\frac{dS_v(t)}{dt} = \Lambda_v - \beta_v S_v(t) I_h(t) - \mu_v S_v(t), \\
\frac{dI_v(t)}{dt} = \beta_v \int_0^{\tau_v} g_v(\tau) S_v(t-\tau) I_h(t-\tau) d\tau - \mu_v I_v(t).
\end{cases} \tag{2.1}$$

Associated with system (2.1), we also consider the following initial conditions

$$\begin{cases} S_h(\theta) = \phi_1(\theta),\ I_h(\theta) = \phi_2(\theta),\ S_v(\theta) = \phi_3(\theta), I_v(\theta) = \phi_4(\theta),\ \text{for } \theta \in [-h, 0], \\ \phi_1(0) > 0,\ \phi_3(0) > 0,\ \phi_i(\theta) \geq 0, \theta \in [-h, 0],\ h = \max\{\tau_v, \tau_h\}, \phi_i \in \mathcal{C}^+;\ i = 1, \cdots, 4. \end{cases} \tag{2.2}$$

By the fundamental theory of functional differential equations, we can show that system (2.1) with initial conditions (2.2) has a unique solution $(S_h(t), I_h(t), S_v(t), I_v(t))$ which is nonnegative for all $t \geq 0$. Moreover, the first equation of (2.1) implies that $\limsup_{t\to\infty} S_h(t) \leq \frac{\Lambda_h}{\mu_h}$. From the first two equations of (2.1), we get

$$\begin{aligned} \int_0^{\tau_h} g_h(\tau) S_h'(t-\tau) d\tau + I_h'(t) &= \Lambda_h \int_0^{\tau_h} g_h(\tau) d\tau \\ &\quad - \mu_h \int_0^{\tau_h} g_h(\tau) S_h(t-\tau) d\tau - (\mu_h + \alpha_h) I_h(t) \\ &\leq \Lambda_h a_h - (\mu_h + \alpha_h) I_h(t). \end{aligned}$$

Let $x_h(t) = \int_0^{\tau_h} g_h(\tau) S(t-\tau) d\tau$. Then, we have $x_h(t) \leq \frac{a_h \Lambda_h}{\mu_h}$ for $t \geq 0$. Choose sufficiently small $\widetilde{\mu}_h$ such that $\widetilde{\mu}_h < \mu_h$. Obviously, we have $\widetilde{\mu}_h \leq \mu_h + \alpha_h$. Thus, we have

$$(x_h(t) + I_h(t))' \leq \Lambda_h a_h - (\mu_h + \alpha_h) I_h(t) \leq 2\Lambda_h a_h - \widetilde{\mu}_h (x_h(t) + I_h(t)),$$

and thus $\limsup_{t\to\infty} (x_h(t) + I_h(t)) \leq \frac{2\Lambda_h a_h}{\widetilde{\mu}_h}$. Since $x_h(t) \geq 0$, it is easy to obtain that $\limsup_{t\to\infty} I_h(t) \leq \frac{2\Lambda_h a_h}{\widetilde{\mu}_h}$. Similarly, it is easy to obtain that $\limsup_{t\to\infty} S_v(t) \leq \frac{\Lambda_v}{\mu_v}$. Choose a sufficiently small positive number $\widetilde{\mu}_v$ such that $\widetilde{\mu}_v < \mu_v$. Thus, we have $\limsup_{t\to\infty} I_v(t) \leq \frac{2\Lambda_v a_v}{\widetilde{\mu}_v}$.

Let

$$\begin{aligned} \Omega = \{&(S_h, I_h, S_v, I_v) \in \mathcal{C}^+ \times \mathcal{C}^+ \times \mathcal{C}^+ \times \mathcal{C}^+ : ||S_h|| \\ &\leq \frac{\Lambda_h}{\mu_h}, ||I_h|| \leq \frac{2\Lambda_h a_h}{\widetilde{\mu}_h}, ||S_v|| \leq \frac{\Lambda_v}{\mu_v}, ||I_v|| \leq \frac{2\Lambda_v a_v}{\widetilde{\mu}_v}\}. \end{aligned}$$

It can be verified that the region Ω is positively invariant with respect to system (2.1) with (2.2). Therefore, for system (2.1), we have the following proposition.

Proposition 2.1 *There exists a unique solution $(S_v(t), I_v(t), S_h(t), I_h(t))$ of system (2.1) with initial conditions (2.2). Moreover, all the solutions of system (2.1) are nonnegative for all $t \geq 0$ and ultimately uniformly bounded in Ω.*

2.1 Equilibria and their stabilities

Let $\tilde{E} = (\tilde{S}_h, \tilde{I}_h, \tilde{S}_v, \tilde{I}_v)$ be an equilibrium. It must satisfy the following equations

$$\Lambda_h - \beta_h \tilde{S}_h \tilde{I}_v - \mu_h \tilde{S}_h = 0,$$

$$\beta_h \int_0^{\tau_h} g_h(\tau)\tilde{S}_h\tilde{I}_v d\tau - (\mu_h+\alpha_h)\tilde{I}_h = 0,$$
$$\Lambda_v - \beta_v\tilde{S}_v\tilde{I}_h - \mu_v\tilde{S}_v = 0,$$
$$\beta_v \int_0^{\tau_v} g_v(\tau)\tilde{S}_v\tilde{I}_h d\tau - \mu_v\tilde{I}_v = 0. \tag{2.3}$$

Using $\int_0^{\tau_v} g_v(\tau)d\tau = a_v$, $\int_0^{\tau_h} g_h(\tau)d\tau = a_h$, and letting

$$\mathcal{R}_0 = \frac{\beta_h\beta_v a_h a_v \Lambda_h \Lambda_v}{(\mu_h+\alpha_h)\mu_h\mu_v^2},$$

it is easy to see that if $\mathcal{R}_0 < 1$, system (2.1) has a disease-free equilibrium $E_0 = (S_v^0, 0, S_h^0, 0)$, where $S_v^0 = \frac{\Lambda_v}{\mu_v}$, $S_h^0 = \frac{\Lambda_h}{\mu_h}$. Apart from E_0, if $\mathcal{R}_0 > 1$, system (2.1) has a unique endemic equilibrium $E^* = (S_h^*, I_h^*, S_v^*, I_v^*)$, where

$$S_h^* = \frac{\Lambda_h\mu_v(\beta_v I_h^* + \mu_v)}{I_h^*(\beta_h\beta_v a_v\Lambda_v + \beta_v\mu_h\mu_v) + \mu_h\mu_v^2},\ S_v^* = \frac{\Lambda_v}{\beta_v I_h^* + \mu_v},$$
$$I_v^* = \frac{\beta_v a_v \Lambda_v I_h^*}{\mu_v(\beta_v I_h^* + \mu_v)},\ I_h^* = \frac{\mu_h\mu_v^2(\mathcal{R}_0 - 1)}{\beta_h\beta_v a_v\Lambda_v + \beta_v\mu_h\mu_v}.$$

Following Driessche and Watmough (2002), $\mathcal{R}_0$ can be defined the basic reproduction number of model (2.1).

Now we consider the linearized system of (2.1) at an equilibrium $\tilde{E} = (\tilde{S}_h, \tilde{I}_h, \tilde{S}_v, \tilde{I}_v)$. Let

$$x_h(t) = S_h(t) - \tilde{S}_h,\quad x_v(t) = S_v(t) - \tilde{S}_v,\quad y_h(t) = I_h(t) - \tilde{I}_h,\quad y_v(t) = I_v(t) - \tilde{I}_v.$$

We obtain the following linearized system

$$\begin{cases} x_h'(t) = -\beta_h\tilde{I}_v x_h(t) - \beta_h\tilde{S}_h y_v(t) - \mu_h x_h(t), \\ y_h'(t) = \beta_h\tilde{I}_v\int_0^{\tau_h} g_h(\tau)x_h(t-\tau)d\tau + \beta_h\tilde{S}_h\int_0^{\tau_h} g_h(\tau)y_v(t-\tau)d\tau - (\mu_h+\alpha_h)y_h(t), \\ x_v'(t) = -\beta_v\tilde{I}_h x_v(t) - \beta_v\tilde{S}_v y_h(t) - \mu_v x_v(t), \\ y_v'(t) = \beta_v\tilde{I}_h\int_0^{\tau_v} g_v(\tau)x_v(t-\tau)d\tau + \beta_v\tilde{S}_v\int_0^{\tau_v} g_v(\tau)y_h(t-\tau)d\tau - \mu_v y_v(t). \end{cases} \tag{2.4}$$

From (2.4), we obtain the following characteristic equation at the disease-free equilibrium E_0

$$(\lambda+\mu_h)(\lambda+\mu_v)\Big[(\lambda+\mu_v)(\lambda+\mu_h+\alpha_h)$$
$$-\frac{\beta_h\beta_v\Lambda_v\Lambda_h}{\mu_h\mu_v}\int_0^{\tau_h} g_h(\tau)e^{-\lambda\tau}d\tau\int_0^{\tau_v} g_v(\tau)e^{-\lambda\tau}d\tau\Big] = 0. \tag{2.5}$$

It is clear that both $\lambda = -\mu_h$ and $\lambda = -\mu_v$ are roots (2.5). All other roots of (2.5) are determined by the following equation:

$$H(\lambda) := (\lambda + \mu_v)(\lambda + \mu_h + \alpha_h) - Q_\lambda = 0, \tag{2.6}$$

where, for simplicity, we use the notation

$$Q_\lambda = \frac{\beta_h \beta_v \Lambda_v \Lambda_h}{\mu_h \mu_v} \int_0^{\tau_h} g_h(\tau) e^{-\lambda\tau} d\tau \int_0^{\tau_v} g_v(\tau) e^{-\lambda\tau} d\tau.$$

For the case $\mathcal{R}_0 < 1$, we suppose on the contrary that E_0 is not locally asymptotically stable. Suppose that λ is a root of $H(\lambda)$ with $\mathrm{Re}\lambda \geq 0$, which implies that $|e^{-\lambda\tau}| \leq 1$ for any $\tau \geq 0$. However, from (2.6) it is easy to obtain the following inequalities

$$|Q_\lambda| \leq \frac{\beta_h \beta_v \Lambda_v \Lambda_h a_h a_v}{\mu_h \mu_v}, \quad \mu_v \leq |\lambda + \mu_v|, \quad \mu_h + \alpha_h \leq |\lambda + \mu_h + \alpha_h|. \tag{2.7}$$

From (2.6) and (2.7), we obtain that

$$\mu_v(\mu_h + \alpha_h) \leq |\lambda + \mu_v||\lambda + \mu_h + \alpha_h| = |Q_\lambda| \leq \frac{\beta_h \beta_v \Lambda_v \Lambda_h a_h a_v}{\mu_h \mu_v}, \tag{2.8}$$

which is a contraction. Hence, if $\mathcal{R}_0 < 1$, then the disease-free equilibrium E_0 of system (2.1) is locally asymptotically stable.

For the case $\mathcal{R}_0 > 1$, it is directly seen that

$$H(0) = \mu_v(\mu_h + \alpha_h) - \frac{\beta_h \beta_v \Lambda_v \Lambda_h a_h a_v}{\mu_h \mu_v} = \mu_v(\mu_h + \alpha_h)(1 - \mathcal{R}_0) < 0$$

and

$$\lim_{\lambda \to +\infty} H(\lambda) = +\infty$$

holds for $\lambda \in \mathbb{R}$. Therefore, system (2.1) has at least one positive real root. Hence, if $\mathcal{R}_0 > 1$, the disease-free equilibrium E_0 is unstable.

Summarizing the above discussion, we obtain the following results.

Proposition 2.2 *If $\mathcal{R}_0 < 1$, the disease-free equilibrium E_0 of system (2.1) is locally asymptotically stable; If $\mathcal{R}_0 > 1$, the disease-free equilibrium E_0 is unstable.*

Now we explore the stability of the endemic equilibrium E^*. By system (2.4) and direct calculations, the characteristic equation at E^* has one negative root $-\mu_v$ and the other roots can be determined by the following equation

$$(\lambda + \mu_h + \alpha_h)(\lambda + \mu_v + \beta_v I_h^*)(\lambda + \mu_h + \beta_h I_v^*)$$

$$= (\lambda + \mu_h)\beta_h S_h^* \int_0^{\tau_h} e^{-\lambda\tau} g_h(\tau) d\tau \beta_v S_v^* \int_0^{\tau_v} e^{-\lambda\tau} g_v(\tau) d\tau. \tag{2.9}$$

We claim that Eq. (2.9) has no roots with positive real part. In fact, dividing both sides by $\lambda + \mu_h$, we have

$$\begin{aligned} LHS &\stackrel{def}{=} \frac{(\lambda + \mu_h + \alpha_h)(\lambda + \mu_v + \beta_v I_h^*)(\lambda + \mu_h + \beta_h I_v^*)}{\lambda + \mu_h} \\ RHS &\stackrel{def}{=} \beta_h \beta_v S_h^* S_v^* \int_0^{\tau_h} e^{-\lambda\tau} g_h(\tau) d\tau \int_0^{\tau_v} e^{-\lambda\tau} g_v(\tau) d\tau. \end{aligned} \tag{2.10}$$

If λ is a root with $\mathrm{Re}\lambda \geq 0$, it follows from (2.10) that

$$\begin{aligned} &|LHS| > |(\lambda + \alpha_h + \mu_h)||(\lambda + \mu_v)| \geq (\alpha_h + \mu_h)\mu_v, \\ &|RHS| \leq \beta_h \beta_v S_h^* S_v^* a_h a_v. \end{aligned} \tag{2.11}$$

Using the relation (2.3) of the endemic equilibrium, we obtain

$$|LHS| > |(\alpha_h + \mu_h)\mu_v = \frac{\beta_h S_h^* I_v^* a_h}{I_h^*} \frac{\beta_v S_v^* I_h^* a_v}{I_v^*} = a_h a_v \beta_h \beta_v S_h^* S_v^* \geq |RHS|.$$

It is a contradiction. Thus, Eq. (2.9) cannot have any roots with positive real part. In conclusion, we summarize the above result in the following lemma.

Proposition 2.3 *If $\mathcal{R}_0 > 1$, then the endemic equilibrium $E^* = (S_h^*, I_h^*, S_v^*, I_v^*)$ of system (2.1) is locally asymptotically stable.*

2.2 Uniform persistence

Now we present some conditions to ensure the uniform persistence of system (2.1). The persistence theory for infinite-dimensional systems was first developed by Hale and Waltman (1989). In recent years, the methods and techniques have been employed by other authors to study delay models (see, Freedman and Ruan 1995; Kuang 1993; Ma et al. 2004; Zhao 2003; Smith and Zhao 2001).

To obtain the persistence result in system (2.1), we first introduce the following notation and terminology. Let $X = \mathcal{C}^+ \times \mathcal{C}^+ \times \mathcal{C}^+ \times \mathcal{C}^+$, and (X, d) be a complete metric space with metric d, and X_0 be an open subset of X. Set $\partial X_0 := X / X_0$ the boundary of X. Clearly, ∂X_0 is a closed subset of X, $X = X_0 \bigcup \partial X_0$, and $X_0 \bigcap \partial X_0 = \emptyset$.

Definition 2.4 A lower semicontinuous function $p : X \to \mathbb{R}_+$ is called *a generalized distance function* for the semiflow $\Phi(t) : X \to X$ if for every $x \in (X_0 \bigcap p^{-1}(0)) \bigcup p^{-1}(0, \infty)$, we have $p(\Phi(t)x) > 0, \forall t > 0$.

Now, we use the following persistence theorem from Theorem 3 in Smith and Zhao (2001).

Lemma 2.5 *Let p be a generalized distance function for the semiflow $\Phi(t) : X \to X$ with $\Phi(t)X_0 \subset X_0$ for all $t \geq 0$. Assume that*

(P1) $\Phi(t) : X \to X$ *has a global attractor* A;
(P2) *There exists a finite sequence* $M = \{M_1, \cdots, M_k\}$ *of disjoint, compact, and isolated invariant sets in* ∂X_0 *with the following properties:*
(i) $\cup_{x\in M_\partial}\omega(x) \subset \cup_{i=1}^{k} M_i$;
(ii) *No subset of* M *forms a cycle in* ∂X_0;
(iii) *Each* M_i *is isolated in* X;
(iv) $W^s(M_i) \bigcup p^{-1}(0, \infty) = \emptyset$, $1 \le i \le k$.

there exists $\delta > 0$ *such that* $\liminf_{t\to+\infty} p(\Phi(t)x) \ge \delta$ *for all* $x \in X_0$.

We now state and prove the following result on uniform persistence of system (2.1).

Theorem 2.6 *Let* $\mathcal{R}_0 > 1$. *Then system* (2.1) *is uniformly persistent in* Int Ω.

Proof Set

$$\begin{aligned} X &= \{(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta), \phi_4(\theta)) : \phi_1(\theta) \\ &\quad \ge 0, \phi_2(\theta) \ge 0, \phi_3(\theta) \ge 0, \phi_4(\theta) \ge 0, -h \le \theta \le 0\}, \\ X_0 &= \{(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta), \phi_4(\theta)) : \phi_1(\theta) > 0, \phi_2(\theta) > 0, \phi_3(\theta) > 0, \\ &\quad \phi_4(\theta) > 0, -h \le \theta < 0\}, \\ \partial X_0 &= X/\ X_0. \end{aligned}$$

Obviously, X_0 is an open set relative to X. Let $x(t, \phi)$ be the unique solution of system (2.1) with $x_0(\phi) = \phi$. Let $\Phi : X \to X$ be the Poincaré map associated with system (2.1); *i.e.*, $\Phi(t) = x_t(\phi)$ for all $\phi \in X$. It is easy to see that $\Phi(t)(X_0) \subset X_0$ for all $t \ge 0$. Note that Proposition 2.1 implies that $\Phi : X \to X$ is point dissipative and Φ^{n_0} is compact whenever $n_0 T > \tau$. Thus, it follows from Magal and Zhao (2005) that $\Phi(t)$ admits a global attractor A in X. Now it suffices to show that $\Phi(t)$ is uniformly persistent with respect to $(X_0, \partial X_0)$.

To attain this end, we have to verity that the condition (*P2*) of Lemma 2.5 is satisfied.

First, it follows from Proposition 2.1 that both X and X_0 are positively invariant. ∂X_0 should be relatively closed in X. In fact, let U be a bounded set of X, and k_1, k_2 be such that for any $(\phi_1, \phi_2, \phi_3, \phi_4) \in U$, $\phi_1 < k_1, \phi_3 < k_2$, and $||\phi_2|| < k_1$, $||\phi_4|| < k_2$. Consider the solution $(\bar{S}_v(t), \bar{I}_v(t), \bar{S}_h(t), \bar{I}_h(t))$ with initial conditions $(\bar{S}_v(0) = k_1, \bar{I}_{v_0} = \phi_2(\theta), \bar{S}_h(0) = k_2, \bar{I}_{h_0} = \phi_4(\theta))$. By a completely similar argument as the one used in Fan et al. (2010), we can show that for any solution $(S_v(t), I_v(t), S_h(t), I_h(t))$ with initial conditions from U, we have $S_v(t) < \bar{S}_v(t), I_v(t) < \bar{I}_v(t), S_h(t) < \bar{S}_h(t), I_h(t)) < \bar{I}_h(t)$ for all $t \in [0, t_0]$.

Set

$$M_\partial = \{\phi \in \partial X_0 : \Phi(t)\phi \text{ satisfies system (2.1) and } \Phi(t)\phi \in \partial X_0, \forall t \ge 0\}.$$

We now claim that $M_\partial = \{(\frac{\Lambda_v}{\mu_v}, 0, \frac{\Lambda_h}{\mu_h}, 0)\}$. Assuming $\phi \in M_\partial$, it suffices to show that $I_v(t) = I_h(t) = 0, \forall t \ge 0$. Assume on the contrary that there exists $t_0 > 0$ such that either (a) $I_v(t_0) > 0, I_h(t_0) = 0$; or (b) $I_v(t_0) = 0, I_h(t_0) > 0$.

In case (a), from the second equation of (2.1), we have

$$\dot{I}_h(t)|_{t=t_0} = \beta_h \int_0^{\tau_h} g_h(\tau) S_h(t_0 - \tau) I_v(t_0 - \tau) d\tau > 0.$$

Hence, there is a sufficiently small constant ε_0 such that $I_h(t) > 0, \forall t \in (t_0, t_0+\varepsilon_0)$. On the other hand, from $I_v(t_0) > 0$, we obtain a positive ε_1 $(0 < \varepsilon_1 < \varepsilon_0)$ such that $I_v(t) > 0, \forall t \in (t_0, t_0 + \varepsilon_1)$. Thus, we obtain $I_v(t) > 0$, $I_h(t) > 0, \forall \in (t_0, t_0 + \varepsilon)$. This is in contradiction with the assumption that $\Phi(t)\phi \in M_\partial$. Similarly we can show that the case (b) does not hold.

Let $\Omega_2 = \bigcup_{x \in A} \omega(x)$, where A is the global attractor of P restricted to ∂X_0. We now show that $\Omega_2 = \{E_0\}$. In fact, it follows from $\Omega_2 \subseteq M_\partial$ and the first and third equations of (2.1). We have $\lim_{t\to+\infty} S_h(t) = \frac{\Lambda_h}{\mu_h}$, $\lim_{t\to+\infty} S_v(t) = \frac{\Lambda_v}{\mu_v}$. Thus, $\{E_0\}$ is the isolated compact invariant set for $\Phi(t)$ in M_∂.

Finally we need to show that $W^s(E_0) \bigcap X_0 = \emptyset$, where $W^s(E_0)$ is a stable set of E_0 for $\Phi(t)$. Assume on the contrary, there exists a positive orbit $(S_v(t), I_v(t), S_h(t)), I_h(t)) \in X_0$ of (2.1) such that

$$\lim_{t\to+\infty} S_v(t) = \frac{\Lambda_v}{\mu_v}, \quad \lim_{t\to+\infty} I_v(t) = 0, \quad \lim_{t\to+\infty} S_h(t) = \frac{\Lambda_h}{\mu_h}, \quad \lim_{t\to+\infty} I_h(t) = 0. \tag{2.12}$$

Then, from (2.12) for any sufficiently small enough constant $\varepsilon_2 > 0$, there exists a positive constant $T_0 = T_0(\varepsilon_2)$ such that

$$S_v(t) > \frac{\Lambda_v}{\mu_v} - \varepsilon_2 > 0, \ S_h(t) > \frac{\Lambda_h}{\mu_h} - \varepsilon_2, \ \forall t \geq T_0. \tag{2.13}$$

Since $\mathcal{R}_0 = \frac{\beta_h \beta_v a_h a_v \Lambda_h \Lambda_v}{(\mu_h+\mu_v)\mu_h \mu_v^2} > 1$, for the above given ε_2, we have

$$\frac{a_h a_v \beta_h \beta_v}{(\mu_h + \alpha_h)\mu_v} \left(\frac{\Lambda_h}{\mu_h} - \varepsilon_2\right)\left(\frac{\Lambda_v}{\mu_v} - \varepsilon_2\right) > 1. \tag{2.14}$$

From the second and fourth equations of system (2.1) and using (2.13), we obtain for any $t > T_0$ that

$$I_h'(t) \geq \beta_h \left(\frac{\Lambda_h}{\mu_h} - \varepsilon_2\right) \int_0^{\tau_h} g_h(\tau) I_v(t - \tau) d\tau - (\mu_h + \alpha_h) I_h(t),$$
$$I_v'(t) \geq \beta_v \left(\frac{\Lambda_v}{\mu_v} - \varepsilon_2\right) \int_0^{\tau_v} g_v(\tau) I_h(t - \tau) d\tau - \mu_v I_v(t).$$

Using the mean value theorem for integrals, we know that for any time t, there is a t_ξ such that

$$\int_0^{\tau_v} g_v(\tau) I_h(t - \tau) d\tau = a_v I_h(t_\xi) \text{ for } t - h \leq t_\xi \leq t. \tag{2.15}$$

Thus, there exist T_0 such that $t > T_0 + \tau$,

$$I_h'(t) \geq \beta_h \left(\frac{\Lambda_h}{\mu_h} - \varepsilon_2\right) \int_0^{\tau_h} g_h(\tau) I_v(t-\tau) d\tau - (\mu_h + \alpha_h) I_h(t),$$
$$I_v'(t) \geq a_v \beta_v \left(\frac{\Lambda_v}{\mu_v} - \varepsilon_2\right) I_h(t_\xi) - \mu_v I_v(t). \tag{2.16}$$

If $(I_v(t), I_h(t)) \to (0,0)$ as $t \to \infty$, then by a standard comparison argument (see Smith 1995, Theorem 5.1.1, Page 78), the solution $(x_v(t), y_h(t))$ of the following equations

$$x_v'(t) = a_v \beta_v \left(\frac{\Lambda_v}{\mu_v} - \varepsilon_2\right) y_h(\xi_t) - \mu_v x_v(t),$$
$$y_h'(t) = \beta_h \left(\frac{\Lambda_h}{\mu_h} - \varepsilon_2\right) \int_0^{\tau_h} g_h(\tau) x_v(t-\tau) d\tau - (\mu_h + \alpha_h) y_h(t) \quad \text{for } t > T_0 + \tau \tag{2.17}$$

with initial conditions $x_v(\theta) = \psi_2(\theta)$, $y_h(\theta) = \psi_4(\theta)$ for $\theta \in [-h, 0]$ and $\forall t \in [T_0, T_0 + \tau]$ converges to $(0,0)$ as well.

Set

$$W(t) = \frac{a_h \beta_h}{\mu_v} \left(\frac{\Lambda_h}{\mu_h} - \varepsilon_2\right) \int_0^{\tau_h} g_h(\tau) x_v(t-\tau) d\tau + y_h(t) + (\mu_h + \alpha_h) \int_{t_\xi}^{t} y_h(s) ds. \tag{2.18}$$

By calculating the derivative of $W(t)$, we obtain that

$$\frac{dW(t)}{dt}\Big|_{(2.17)} = \left[\frac{a_h a_v \beta_h \beta_v}{\mu_v} \left(\frac{\Lambda_h}{\mu_h} - \varepsilon_2\right) \left(\frac{\Lambda_v}{\mu_v} - \varepsilon_2\right) - (\mu_h + \alpha_h)\right] y(t_\xi) > 0.$$

It follows from (2.14) that $W(t)$ goes to infinity or approaches a positive limit as $t \to \infty$ for $\mathcal{R}_0 > 1$. On the other hand, by our assumption, we have shown that $\lim_{t\to\infty} x_v(t) = 0, \lim_{t\to\infty} y_h(t) = 0$. Thus, from (2.18), we have $W(t) \to 0$ as $t \to \infty$. This is a contradiction. Therefore, we have $W^s(E_0) \bigcap X_0 = \emptyset$. By the acyclicity theorem on uniform persistence for maps, it follows from Theorem 3.1.1 in Zhao (2003) that the semiflow $\Phi(t) : X \to X$ is also uniformly persistent with respect to X_0. Therefore, from Theorem 4.5 in Magal and Zhao (2005), we obtain that system (2.1) admits a positive solution $\Phi(t)\phi$ with $\phi \in X_0$.

Let $\rho(x) = d(x, \partial X_0)$. It follows that $\Phi(t) : X_0 \to X_0$ has a compact global attractor A_0. Since $A_0 = \Phi(T)A_0$, we see that $\phi_i(0) > 0$ for all $i = 1, \ldots, 4$. Let $M_0 = \bigcup_{t\in[0,T]} \phi(t) A_0$. Thus, we can obtain $\varphi_i(0) > 0$ for all $\varphi_i \in M_0, i = 1, \ldots, 4$. Moreover, $M_0 \subset X_0$ and $\lim_{t\to\infty} d(\Phi(t)\phi, M_0) = 0$. Now we define a continuous function by

$$p(\phi) = \min_{1\leq i\leq 4} \{\phi_i(0)\}, \ \forall \phi \in X.$$

Obviously, $p : X \to \mathbb{R}_+$. Since M_0 is a compact subset of X_0, it follows that $\inf_{\phi\in M_0} p(\phi) = \min_{\phi\in M_0} p(\phi)$. Therefore, there exists a positive constant $\delta > 0$ such that

$$\liminf_{t\to+\infty}(S_h(t,\phi_1), I_h(t,\phi_2), S_v(t,\phi_3), I_v(t,\phi_4)) = \liminf_{t\to+\infty} p(\Phi(t)x) \geq \delta.$$

This completes the proof of Theorem 2.6. □

2.3 Global stability

In the previous section, we have established the local stability of the equilibria and have shown that the incubation period delays have no effect on the local stability of the equilibria for system (2.1). In this section, our objective is to derive global stability results. Inspired by recent works of Huang and Takeuchi (2011), McCluskey (2010), Shuai and van den Driessche (2013), Vargas-De-León (2012), and Yang et al. (2015), we construct a suitable Liapunov functional to establish the global stability of the endemic equilibrium E^*. We have the following theorem.

Theorem 2.7 *If $\mathcal{R}_0 > 1$, then the endemic equilibrium E^* of system (2.1) is globally asymptotically stable.*

Proof By Proposition 2.3, it suffices to show the global attractiveness of E^*. Let $f(x) = x - 1 - \ln x$ for $x > 0$. Because of the complexity of the expressions, we define the Liapunov functional in components and take the derivative of each component separately. Set

$$\begin{aligned} U_{S_v}(t) &= f\left(\frac{S_v(t)}{S_v^*}\right), \quad U_{I_v}(t) = f\left(\frac{I_v(t)}{I_v^*}\right), \\ U_{+I_v}(t) &= \int_0^{\tau_v} g_v(\tau)\int_{t-\tau}^{t} f\left(\frac{S_v(\sigma)I_h(\sigma)}{S_v^*I_h^*}\right)d\sigma\, d\tau. \end{aligned} \tag{2.19}$$

First, we calculate the time derivatives of $U_{s_v}(t)$ and $U_{I_v}(t)$ along system (2.1) obtain that

$$\begin{aligned} \frac{dU_{S_v}(t)}{dt} &= \frac{1}{S_v^*}\left(\frac{S_v(t)-S_v^*}{S_v(t)}\right)\frac{dS_v(t)}{dt} \\ &= \frac{1}{S_v^*}\left(\frac{S_v(t)-S_v^*}{S_v(t)}\right)(\Lambda_v - \beta_v S_v(t)I_h(t) - \mu_v S_v(t)) \\ &= \frac{1}{S_v^*}\left(\frac{S_v(t)-S_v^*}{S_v(t)}\right)\left[\beta_v S_v^* I_h^* - \beta_v S_v(t)I_h(t)) + \mu_v(S_v^* - S_v(t))\right] \\ &= -\frac{\mu_v(S_v(t)-S_v^*)^2}{S_v^* S_v(t)} + \beta_v I_h^*\left(1 - \frac{S_v^*}{S_v(t)} - \frac{S_v(t)I_h(t)}{S_v^*I_h^*} + \frac{I_h(t)}{I_h^*}\right). \\ \frac{dU_{I_v}(t)}{dt} &= \frac{1}{I_v^*}\left(\frac{I_v(t)-I_v^*}{I_v(t)}\right)\frac{dI_v(t)}{dt} \end{aligned} \tag{2.20}$$

$$
\begin{aligned}
&= \frac{1}{I_v^*}\left(\frac{I_v(t)-I_v^*}{I_v(t)}\right)\left(\beta_v\int_0^{\tau_v} g_v(\tau)S_v(t-\tau)I_h(t-\tau)d\tau-\mu_v I_v(t)\right)\\
&= \frac{1}{I_v^*}\left(\frac{I_v-I_v^*}{I_v(t)}\right)\left[\beta_v S_v^* I_h^*\int_0^{\tau_v} g_v(\tau)\left(\frac{S_v(t-\tau)I_h(t-\tau)}{S_v^*I_h^*}-\frac{\mu_v I_v(t)}{\beta_v a_v S_v^* I_h^*}\right)\right]d\tau\\
&= \frac{\beta_v S_v^* I_h^*}{I_v^*}\int_0^{\tau_v} g_v(\tau)\left[1+\frac{S_v(t-\tau)I_h(t-\tau)}{S_v^*I_h^*}-\frac{I_v}{I_v^*}\right.\\
&\quad \left.-\frac{S_v(t-\tau)I_h(t-\tau)I_v^*}{S_v^*I_h^*I_v(t)}\right]d\tau. \qquad (2.21)
\end{aligned}
$$

Taking the time derivative of $U_{+I_v}(t)$ with respect to t, we obtain

$$
\begin{aligned}
\frac{dU_{+I_v}(t)}{dt} &= \frac{d}{dt}\int_0^{\tau_v} g_v(\tau)\int_{t-\tau}^{t} f\left(\frac{S_v(\sigma)I_h(\sigma)}{S_v^*I_h^*}\right)d\sigma d\tau\\
&= \int_0^{\tau_v} g_v(\tau)\left[f\left(\frac{S_v(t)I_h(t)}{S_v^*I_h^*}\right)-f\left(\frac{S_v(t-\tau)I_h(t-\tau)}{S_v^*I_h^*}\right)\right]d\tau\\
&= \int_0^{\tau_v} g_v(\tau)\left(\frac{S_v(t)I_h(t)}{S_v^*I_h^*}-\ln\frac{S_v(t)I_h(t)}{S_v^*I_h^*}-\frac{S_v(t-\tau)I_h(t-\tau)}{S_v^*I_h^*}\right.\\
&\quad \left.+\ln\frac{S_v(t-\tau)I_h(t-\tau)}{S_v^*I_h^*}\right)d\tau. \qquad (2.22)
\end{aligned}
$$

Set

$$
U_v(t) = \frac{a_v}{\beta_v I_h^*}U_{S_v}(t)+\frac{I_v^*}{\beta_v S_v^* I_h^*}U_{I_v}(t)+U_{+I_v}(t).
$$

By using (2.20)–(2.22) and calculating the time derivative of $U_v(t)$ with respect to t, we obtain

$$
\begin{aligned}
\frac{dU_v(t)}{dt} &= -\frac{\mu_v a_v(S_v(t)-S_v^*)^2}{\beta_v I_h^* S_v^* S_v(t)}+a_v\left(1-\frac{S_v^*}{S_v(t)}-\frac{S_v(t)I_h(t)}{S_v^*I_h^*}+\frac{I_h(t)}{I_h^*}\right)\\
&\quad +\int_0^{\tau_v} g_v(\tau)\left[1+\frac{S_v(t-\tau)I_h(t-\tau)}{S_v^*I_h^*}-\frac{I_v}{I_v^*}-\frac{S_v(t-\tau)I_h(t-\tau)I_v^*}{S_v^*I_h^*I_v(t)}\right]d\tau\\
&\quad +\int_0^{\tau_v} g_v(\tau)\left(\frac{S_v(t)I_h(t)}{S_v^*I_h^*}-\ln\frac{S_v(t)I_h(t)}{S_v^*I_h^*}-\frac{S_v(t-\tau)I_h(t-\tau)}{S_v^*I_h^*}\right.\\
&\quad \left.+\ln\frac{S_v(t-\tau)I_h(t-\tau)}{S_v^*I_h^*}\right)d\tau\\
&= -\frac{\mu_v a_v(S_v(t)-S_v^*)^2}{\beta_v I_h^* S_v^* S_v(t)}+\int_0^{\tau_v} g_v(\tau)\left[2-\frac{S_v^*}{S_v(t)}-\frac{I_v(t)}{I_v^*}\right.\\
&\quad \left.-\frac{S_v(t-\tau)I_h(t-\tau)I_v^*}{S_v^*I_h^*I_v(t)}+\frac{I_h(t)}{I_h^*}\right.
\end{aligned}
$$

$$- \ln \frac{S_v(t) I_h(t)}{S_v^* I_h^*} + \ln \frac{S_v(t-\tau) I_h(t-\tau)}{S_v^* I_h^*} \Bigg] d\tau. \tag{2.23}$$

Similarly, set

$$\begin{aligned} U_{S_h}(t) &= f\left(\frac{S_h(t)}{S_h^*}\right), \quad U_{I_h}(t) = f\left(\frac{I_h(t)}{I_h^*}\right), \\ U_{+I_h}(t) &= \int_0^{\tau_h} g_h(\tau) \int_{t-\tau}^{t} f\left(\frac{S_h(\sigma) I_v(\sigma)}{S_h^* I_v^*}\right) d\sigma d\tau, \\ U_h(t) &= \frac{a_h}{\beta_h I_v^*} U_{S_h}(t) + \frac{I_h^*}{\beta_h S_h^* I_v^*} U_{I_h}(t) + U_{+I_h}(t). \end{aligned} \tag{2.24}$$

Calculating the time derivative of the functions in (2.24) with respect to t along system (2.1), we obtain

$$\begin{aligned} \frac{dU_{S_h}(t)}{dt} &= \frac{1}{S_h^*}\left(\frac{S_h(t) - S_h^*}{S_h(t)}\right) \frac{dS_h(t)}{dt} \\ &= -\frac{\mu_h (S_h(t) - S_h^*)^2}{S_h^* S_h(t)} + \beta_h I_v^* \left(1 - \frac{S_h^*}{S_h(t)} - \frac{S_h(t) I_v(t)}{S_h^* I_v^*} + \frac{I_v(t)}{I_v^*}\right) d\tau, \\ \frac{dU_{I_h}(t)}{dt} &= \frac{1}{I_h^*}\left(\frac{I_h(t) - I_h^*}{I_h(t)}\right) \frac{dI_h(t)}{dt} \\ &= \frac{\beta_h S_h^* I_v^*}{I_h^*} \int_0^{\tau_h} g_h(\tau) \Bigg[1 + \frac{S_h(t-\tau) I_v(t-\tau)}{S_h^* I_v^*} \\ &\quad - \frac{I_h(t)}{I_h^*} - \frac{S_h(t-\tau) I_v(t-\tau) I_h^*}{S_h^* I_v^* I_h(t)}\Bigg] d\tau, \\ \frac{dU_{+I_h}(t)}{dt} &= \int_0^{\tau_h} g_h(\tau) \left(\frac{S_h(t) I_v(t)}{S_h^* I_v^*} - \ln \frac{S_h(t) I_v(t)}{S_h^* I_v^*} - \frac{S_h(t-\tau) I_v(t-\tau)}{S_h^* I_v^*}\right. \\ &\quad \left. + \ln \frac{S_h(t-\tau) I_v(t-\tau)}{S_h^* I_v^*}\right) d\tau. \end{aligned} \tag{2.25}$$

Thus, from (2.24)–(2.25), we have

$$\begin{aligned} \frac{dU_h(t)}{dt} &= -\frac{\mu_h a_h (S_h(t) - S_h^*)^2}{\beta_h I_v^* S_h^* S_h(t)} + \int_0^{\tau_h} g_h(\tau) \Bigg[2 - \frac{S_h^*}{S_h(t)} \\ &\quad - \frac{I_h(t)}{I_h^*} - \frac{S_h(t-\tau) I_v(t-\tau) I_h^*}{S_h^* I_v^* I_h(t)} + \frac{I_v(t)}{I_v^*} \\ &\quad - \ln \frac{S_h(t) I_v(t)}{S_h^* I_v^*} + \ln \frac{S_h(t-\tau) I_v(t-\tau)}{S_h^* I_v^*}\Bigg] d\tau. \end{aligned} \tag{2.26}$$

Set $U_1(t) = \frac{U_v(t)}{a_v} + \frac{U_h(t)}{a_h}$. Using $\int_0^{\tau_h} g_h(\tau)d\tau = a_h$, $\int_0^{\tau_v} g_v(\tau)\,d\tau = a_v$, and (2.23) and (2.26), we obtain

$$\begin{aligned}
\frac{dU_1(t)}{dt} &= \frac{dU_h(t)}{a_h dt} + \frac{dU_v(t)}{a_v dt} \\
&= -\frac{(S_v(t)-S_v^*)^2}{\beta_v I_h^* S_v^* S_v(t)} + 2 - \frac{S_v^*}{S_v(t)} - \frac{I_v(t)}{I_v^*} + \frac{I_h(t)}{I_h^*} - \ln\frac{S_v(t)I_h(t)}{S_v^* I_h^*} \\
&\quad -\frac{1}{a_v}\int_0^{\tau_v} g_v(\tau)\left[\frac{S_v(t-\tau)I_h(t-\tau)I_v^*}{S_v^* I_h^* I_v(t)} - \ln\frac{S_v(t-\tau)I_h(t-\tau)}{S_v^* I_h^*}\right]d\tau \\
&\quad -\frac{\mu_h(S_h(t)-S_h^*)^2}{\beta_h I_v^* S_h^* S_h(t)} + 2 - \frac{S_h^*}{S_h(t)} - \frac{I_h(t)}{I_h^*} + \frac{I_v(t)}{I_v^*} - \ln\frac{S_h(t)I_v(t)}{S_h^* I_v^*} \\
&\quad -\frac{1}{a_h}\int_0^{\tau_h} g_h(\tau)\left[\frac{S_h(t-\tau)I_v(t-\tau)I_h^*}{S_h^* I_v^* I_h(t)} - \ln\frac{S_h(t-\tau)I_v(t-\tau)}{S_h^* I_v^*}\right]d\tau.
\end{aligned} \tag{2.27}$$

Equation (2.27) can be reduced to the following

$$\begin{aligned}
\frac{dU_1(t)}{dt} &= -\frac{\mu_v(S_v(t)-S_v^*)^2}{\beta_v I_h^* S_v^* S_v(t)} - \frac{\mu_h(S_h(t)-S_h^*)^2}{\beta_h I_v^* S_h^* S_h(t)} + 1 - \frac{S_v^*}{S_v(t)} + \ln\frac{S_v^*}{S_v(t)} \\
&\quad +\frac{1}{a_v}\int_0^{\tau_v} g_v(\tau)\left(1 - \frac{S_v(t-\tau)I_h(t-\tau)I_v^*}{S_v^* I_h^* I_v(t)}\right. \\
&\quad \left. +\ln\frac{S_v(t-\tau)I_h(t-\tau)I_v^*}{S_v^* I_h^* I_v(t)}\right]d\tau + 1 - \frac{S_h^*}{S_h(t)} \\
&\quad +\ln\frac{S_h^*}{S_h(t)} + \frac{1}{a_h}\int_0^{\tau_h} g_h(\tau)\left(1 - \frac{S_h(t-\tau)I_v(t-\tau)I_h^*}{S_h^* I_v^* I_h(t)}\right. \\
&\quad \left. +\ln\frac{S_h(t-\tau)I_v(t-\tau)I_h^*}{S_h^* I_v^* I_h(t)}\right)d\tau \\
&= -\frac{\mu_v(S_v(t)-S_v^*)^2}{\beta_v I_h^* S_v^* S_v(t)} - f\left(\frac{S_v^*}{S_v(t)}\right) \\
&\quad -\frac{1}{a_v}\int_0^{\tau_v} g_v(\tau) f\left(\frac{S_v(t-\tau)I_h(t-\tau)I_v^*}{S_v^* I_h^* I_v(t)}\right)d\tau \\
&\quad -\frac{\mu_h(S_h(t)-S_h^*)^2}{\beta_h I_v^* S_h^* S_h(t)} - f\left(\frac{S_h^*}{S_h(t)}\right) \\
&\quad -\frac{1}{a_h}\int_0^{\tau_h} g_h(\tau) f\left(\frac{S_h(t-\tau)I_v(t-\tau)I_h^*}{S_h^* I_v^* I_h(t)}\right)d\tau.
\end{aligned} \tag{2.28}$$

For Eq. (2.28), using $f(x) = x - 1 - \ln x > 0$, $x \geq 0$, we obtain that $\frac{dU_1(t)}{dt} \leq 0$ for $S_v, I_v, S_h, I_h > 0$, and $\frac{dU_1(t)}{dt} = 0$ if and only if $S_v(t-\tau) = S_v^*$, $I_v(t-\tau) = I_v^*$, $S_h(t-\tau) = S_h^*$, $I_h(t-\tau) = I_h^*$ for all $\tau \in [0, h]$. The largest compact invariant set in

$$\Omega_1 = \{(S_v(t), I_v(t), S_h(t), I_h(t)) | \dot{U}_1(t) = 0\}$$

is $\{E^*\}$. By Proposition 2.3 and LaSalle's invariance principle (see Kuang 1993), we can conclude that the endemic equilibrium E^* of system (2.1) is globally asymptotically stable. □

Next we discuss the global stability of the disease-free equilibrium.

Theorem 2.8 *If $\mathcal{R}_0 \leq 1$, then the disease-free equilibrium E_0 of system (2.1) is globally asymptotically stable.*

Proof Consider the following Liapunov function:

$$\begin{aligned} U_2(t) = & \frac{a_v \beta_h S_v^0}{\mu_v} f\left(\frac{S_v(t)}{S_v^0}\right) + f\left(\frac{S_h(t)}{S_h^0}\right) + \frac{\beta_h}{\mu_v} I_v(t) \\ & + \frac{1}{a_h S_h^0} I_h(t) + \frac{\beta_h \beta_v}{\mu_v} \int_0^{\tau_v} g_v(\tau) \int_{t-\tau}^{t} S_v(\sigma) I_h(\sigma) d\sigma d\tau \\ & + \frac{\beta_h}{a_h S_h^0} \int_0^{\tau_h} g_h(\tau) \int_{t-\tau}^{t} S_h(\sigma) I_v(\sigma) d\sigma d\tau. \end{aligned} \tag{2.29}$$

Along the solutions of (2.1), by directly calculating the derivative of $U_2(t)$, we obtain that

$$\begin{aligned} \frac{dU_2(t)}{dt} = & -\frac{a_v \beta_h (S_v(t) - S_v^0)^2}{S_v(t)} - \frac{\mu_h (S_h(t) - S_h^0)^2}{S_h^0 S_h(t)} \\ & + \frac{a_v \beta_h \beta_v S_v^0}{\mu_v} \left(1 - \frac{1}{\mathcal{R}_0}\right) I_h(t) \leq 0 \quad \text{for} \quad \mathcal{R}_0 \leq 1. \end{aligned}$$

From the above discussion and Proposition 2.2, we can conclude that the disease-free equilibrium E_0 is globally asymptotically stable.

3 The extended model with direct transmission

There are evidences showing that in urban centers vector-borne diseases (such as Chagas' disease) are not only transmitted by the vectors but also by contaminated blood of infected hosts through blood transfusions (see Kitchen and Chiodini 2006; Rassi et al. 2010). It has been pointed out that some vector-borne diseases are characterized by the existence of a chronic stage that can last from 10 to 20 years. The existence of the long chronic stage enhances the likelihood of transmission by blood transfusions especially through the asymptomatic patients. The lack of adequate screening of blood samples has increased the chances of vector-borne diseases transmission by blood transfusion. Similar to model (2.1), here we still denote by $S_v(t)$ and $S_h(t)$ the number of susceptible vector and host populations at times t, respectively. Let $I_v(t)$ and $I_h(t)$ be the number of infective vector and infective host populations, respectively. R_h is

the number of recovered host individuals. $\beta_{h_2}\int_0^{\tau_h} g_{h_2}(\tau)S_h(t-\tau)I_h(t-\tau)d\tau$ is the incidence of new cases of infection in the direct transmission at the present time t. Dropping the equation for $R_h(t)$, we obtain the following distributed delay model with direct transmission:

$$\begin{cases} \frac{dS_h(t)}{dt} = \Lambda_h - \beta_{h_1}S_h(t)I_v(t) - \beta_{h_2}S_h(t)I_h(t) - \mu_h S_h(t), \\ \frac{dI_h(t)}{dt} = \beta_{h_1}\int_0^{\tau_h} g_{h_1}(\tau)S_h(t-\tau)I_v(t-\tau)d\tau \\ \qquad +\beta_{h_2}\int_0^{\tau_h} g_{h_2}(\tau)S_h(t-\tau)I_h(t-\tau)d\tau - (\mu_h+\alpha_h)I_h(t), \\ \frac{dS_v(t)}{dt} = \Lambda_v - \beta_v S_v(t)I_h(t) - \mu_v S_v(t), \\ \frac{dI_v(t)}{dt} = \beta_v\int_0^{\tau_v} g_v(\tau)S_v(t-\tau)I_h(t-\tau)d\tau - \mu_v I_v(t). \end{cases} \tag{3.1}$$

The initial conditions and parameters in (3.1) are same as those in (2.1). All parameters are positive constants.

Similar to Sect. 2.1, we can show that there exists a positively invariant compact set Ω_0 for model (3.1). The basic reproduction number of system (3.1) can be evaluated as follows:

$$\hat{\mathcal{R}}_0 = \frac{\Lambda_h}{\mu_h}\left(\frac{a_{h1}a_v\beta_{h_1}}{\mu_h+\alpha_h}\frac{\beta_v}{\mu_v}\frac{\Lambda_v}{\mu_v} + \frac{\beta_{h_2}a_{h2}}{\mu_h+\alpha_h}\right),$$

where,

$$a_{h_1} = \int_0^{\tau_h} g_{h1}(\tau)d\tau,\ a_{h_2} = \int_0^{\tau_h} g_{h_2}(\tau)d\tau,\ a_v = \int_0^{\tau_v} g_v(\tau)d\tau.$$

Direct calculation shows that system (3.1) always has an infection-free equilibrium $M_0 = (\frac{\Lambda_h}{\mu_h}, 0, \frac{\Lambda_v}{\mu_v}, 0)$. If $\hat{\mathcal{R}}_0 > 1$, system (3.1) has a unique positive equilibrium $M^* = (S_h^*, I_h^*, S_v^*, I_v^*)$, where

$$\begin{aligned} S_h^* &= \frac{\Lambda_h\mu_v(\beta_v I_h^* + \mu_v)}{\mu_v(\beta_{h_2}I_h^* + \mu_h)(\beta_v I_h^* + \mu_v) + \Lambda_v a_v\beta_{h_1}\beta_v I_h^*}, \quad I_v^* = \frac{\Lambda_v a_v\beta_v I_h^*}{\mu_v(\beta_v I_h^* + \mu_v)}, \\ S_v^* &= \frac{\Lambda_v}{\beta_v I_h^* + \mu_v}, \end{aligned} \tag{3.2}$$

and I_h^* is the unique positive root of the following equation

$$c_0 I_h^2 + c_1 I_h + c_2 = 0,$$

where,

$$\begin{aligned} c_0 &= (\mu_h+\alpha_h)\mu_v a_v\beta_{h_2}\beta_v > 0, \\ c_1 &= (\mu_h+\alpha_h)(\beta_{h_2}\mu_v^2 + \mu_h\mu_v\beta_v + a_v\Lambda_v\beta_{h_1}\beta_v) - \Lambda_v\mu_v\beta_{h_2}\beta_v, \\ c_2 &= (\mu_h+\alpha_h)\mu_h\mu_v^2(1-\mathcal{R}_0) < 0. \end{aligned}$$

Similar to the proof of Theorem 2.6, it is easy to show that if $\hat{\mathcal{R}}_0 > 1$, then system (3.1) is uniformly persistent in $\text{int}\Omega_0$ (the interior of Ω_0).

In the following, by constructing suitable Liapunov functions, we show the global stability of equilibria in system (3.1) for the same infectivity indexes $a_{h1} = a_{h2}$. Thus, let $a_{h1} = a_{h2} = \widetilde{a}_h$, we have following results

Theorem 3.1 *If $\hat{\mathcal{R}}_0 > 1$, then the endemic equilibrium M^* of system (3.1) is globally stable.*

Proof Let $f(x) = x - 1 - \ln x, x > 0$. By constructing the following Liapunov functional

$$\begin{aligned} V_1(t) = {} & \widetilde{a}_h S_h^* f\left(\frac{S_h(t)}{S_h^*}\right) + I_h^* f\left(\frac{I_h(t)}{I_h^*}\right) \\ & + \beta_{h_1} S_h^* I_v^* \int_0^{\tau_h} g_h(\tau) \int_{t-\tau}^{t} f\left(\frac{S_h(\sigma) I_v(\sigma)}{S_h^* I_v^*}\right) d\sigma d\tau \\ & + \beta_{h_2} S_h^* I_h^* \int_0^{\tau_h} g_h(\tau) \int_{t-\tau}^{t} f\left(\frac{S_h(\sigma) I_h(\sigma)}{S_h^* I_h^*}\right) d\sigma d\tau \\ & + \frac{\widetilde{a}_h \beta_{h_1} S_h^* I_v^*}{\beta_v I_h^*} f\left(\frac{S_v(t)}{S_v^*}\right) + \frac{\beta_{h_1} S_h^* (I_v^*)^2}{\beta_v S_v^* I_h^*} f\left(\frac{I_v(t)}{I_v^*}\right) \\ & + \beta_{h_1} S_h^* I_v^* \int_0^{\tau_h} g_v(\tau) \int_{t-\tau}^{t} f\left(\frac{S_v(\sigma) I_h(\sigma)}{S_v^* I_h^*}\right) d\sigma d\tau, \end{aligned} \tag{3.3}$$

along solutions of system (3.1), we have

$$\begin{aligned} \frac{dV_1(t)}{dt} \le {} & -\widetilde{a}_h \beta_{h_1} S_h^* I_v^* \left(f\left(\frac{S_h(t)}{S_h^*}\right) + f\left(\frac{S_v(t)}{S_v^*}\right)\right) \\ & - \beta_{h_1} S_h^* I_v^* \int_0^{\tau_v} g_v(\tau) f\left(\frac{S_h(t-\tau) I_v(t-\tau) I_h^*}{S_h^* I_v^* I_h(t)}\right) d\tau \\ & - \beta_{h_1} S_h^* I_v^* \int_0^{\tau_v} g_v(\tau) f\left(\frac{S_v(t-\tau) I_h(t-\tau) I_v^*}{S_v^* I_h^* I_v(t)}\right) d\tau \\ & - \widetilde{a}_h \beta_{h_2} S_h^* I_h^* f\left(\frac{S_h(t)}{S_h^*}\right) \\ & - \beta_{h2} S_h^* I_h^* \int_0^{\tau_h} g_h(\tau) f\left(\frac{S_h(t-\tau) I_h(t-\tau) I_h^*}{S_h^* I_h^* I_h(t)}\right) d\tau \le 0. \end{aligned}$$

Similar to the proof in Theorem 2.7, we can complete the proof of Theorem 3.1. Here, we omit the details proof. □

Similar to Theorem 2.8, using the following Liapunov functional

$$V_2(t) = \frac{a_v \beta_{h_1} S_v^0}{\mu_v} f\left(\frac{S_v(t)}{S_v^0}\right) + f\left(\frac{S_h(t)}{S_h^0}\right) + \frac{\beta_{h_1}}{\mu_v} I_v(t) + \frac{1}{\widetilde{a}_h S_h^0} I_h(t)$$

$$+\frac{\beta_{h_1}}{\mu_v}\int_0^{\tau_v}\int_{t-\tau}^{t} g_v(\tau)S_v(\sigma)I_h(\sigma)d\sigma d\tau$$
$$+\frac{\beta_{h_1}}{\widetilde{a}_h S_h^0}\int_0^{\tau_h}\int_{t-\tau}^{t} g_h(\tau)S_h(\sigma)I_v(\sigma)d\sigma d\tau$$
$$+\frac{\beta_{h_2}}{\widetilde{a}_h S_h^0}\int_0^{\tau_h} g_h(\tau)\int_{t-\tau}^{t} S_h(\sigma)I_h(\sigma)d\sigma d\tau,$$

along solutions of system (3.1), we have

$$\frac{dV_2(t)}{dt} = -\frac{a_v\beta_h(S_v(t)-S_v^0)^2}{S_v(t)} - \frac{\mu_h(S_h(t)-S_h^0)^2}{S_h^0 S_h(t)}$$
$$+\frac{a_v\beta_{h_1}\beta_v S_v^0}{\mu_v}\left(1-\frac{1}{\hat{\mathcal{R}}_0}\right)I_h(t) \le 0 \quad \text{for} \quad \hat{\mathcal{R}}_0 \le 1.$$

Thus, we can establish the following global stability of the disease-free equilibrium M_0 for system (3.1).

Theorem 3.2 *If $\hat{\mathcal{R}}_0 \le 1$, then the disease-free equilibrium M_0 of system (3.1) is globally stable.*

4 Vector–host disease models with discrete delays

In this section, we first consider vector–host model (2.1) with discrete delays. Let the kernels

$$g_h(\xi_1) = e^{-(\mu_h+\alpha_h)\xi_1}\delta(\xi_1-\tau_1), \quad g_v(\xi_2) = e^{-\mu_v\xi_2}\delta(\xi_2-\tau_2),$$

where $\delta(\cdot)$ is the Dirac delta function. From model (2.1) we obtain the following model, which is a generalization of the delayed Ross–Macdonald model for malaria transmission considered in Ruan et al. (2008):

$$\begin{cases} \frac{dS_h(t)}{dt} = \Lambda_h - \beta_h S_h(t)I_v(t) - \mu_h S_h(t), \\ \frac{dI_h(t)}{dt} = \beta_h S_h(t-\tau_1)I_v(t-\tau_1)e^{-(\mu_h+\alpha_h)\tau_1} - (\mu_h+\alpha_h)I_h(t), \\ \frac{dS_v(t)}{dt} = \Lambda_v - \beta_v S_v(t)I_h(t) - \mu_v S_v(t), \\ \frac{dI_v(t)}{dt} = \beta_v S_v(t-\tau_2)I_h(t-\tau_2)e^{-\mu_v\tau_2} - \mu_v I_v(t) \end{cases} \tag{4.1}$$

with the initial conditions

$$S_h(\theta) = \varphi_1(\theta),\ I_h(\theta) = \varphi_2(\theta),\ S_v(\theta) = \varphi_3(\theta),$$
$$I_v(\theta) = \varphi_4(\theta),\ \theta \in [-\tau, 0],\ \tau = \max\{\tau_1, \tau_2\},$$

where $\varphi_i(\theta) \in C([-\tau, 0], \mathbb{R}_+)$. All parameters are positive constant. The basic reproduction number of (4.1) is given by

$$\bar{\mathcal{R}}_0 = \frac{\beta_h \beta_v \Lambda_h \Lambda_v e^{-(\mu_h+\alpha_h)\tau_1} e^{-\mu_v \tau_2}}{(\mu_h + \alpha_h)\mu_h \mu_v^2}.$$

System (4.1) always has a trivial equilibrium $\bar{E}_0 = (\frac{\Lambda_h}{\mu_h}, 0, \frac{\Lambda_v}{\mu_v}, 0)$ and a positive equilibrium $\bar{E}^* = (\bar{S}_h^*, \bar{I}_h^*, \bar{S}_v^*, \bar{I}_v^*)$ if $\bar{\mathcal{R}}_0 > 1$. Following the techniques in Ruan et al. (2008), one can establish the global asymptotic stability of the trivial equilibrium $\bar{E}_0$ for $\bar{\mathcal{R}}_0 < 1$ and obtain sufficient conditions for the local asymptotic stability of the positive equilibrium $\bar{E}^*$ by analyzing the associated characteristic equations. Here we study the global stability of the positive equilibrium of system (4.1).

Similar to Theorem 2.8 in Sect. 2, we consider the following Liapunov function

$$\begin{aligned}\bar{W}(t) = {} & \frac{1}{\beta_h \bar{I}_v^*} f\left(\frac{S_h(t)}{\bar{S}_h^*}\right) + \frac{\bar{I}_h^*}{\beta_h e^{-(\mu_h+\alpha_h)\tau_1} \bar{S}_h^* \bar{I}_v^*} f\left(\frac{I_h(t)}{\bar{I}_h^*}\right) \\ & + \int_{t-\tau_1}^{t} f\left(\frac{S_h(\sigma) I_v(\sigma)}{\bar{S}_h^* \bar{I}_v^*}\right) d\sigma + \frac{1}{\beta_v \bar{I}_h^*} f\left(\frac{S_v(t)}{\bar{S}_v^*}\right) \\ & + \frac{\bar{I}_v^*}{\beta_v e^{-\mu_v \tau_2} \bar{S}_v^* \bar{I}_h^*} f\left(\frac{I_v(t)}{\bar{I}_v^*}\right) + \int_{t-\tau_2}^{t} f\left(\frac{S_v(\sigma) I_h(\sigma)}{\bar{S}_v^* \bar{I}_h^*}\right) d\sigma. \end{aligned} \tag{4.2}$$

Along solutions of system (4.1), we have

$$\begin{aligned}\frac{d\bar{W}(t)}{dt} = {} & -\frac{\mu_h (S_h(t) - \bar{S}_h^*)^2}{\beta_h S_h(t) \bar{S}_h^* \bar{I}_v^*} - \frac{\mu_v (S_v(t) - \bar{S}_v^*)^2}{\beta_v S_v(t) \bar{S}_v^* \bar{I}_h^*} - f\left(\frac{\bar{S}_h^*}{S_h(t)}\right) \\ & - f\left(\frac{S_h(t-\tau_1) I_v(t-\tau_1) \bar{I}_h^*}{\bar{S}_h^* \bar{I}_v^* I_h(t)}\right) \\ & - f\left(\frac{\bar{S}_v^*}{S_v(t)}\right) - f\left(\frac{S_v(t-\tau_2) I_h(t-\tau_2) \bar{I}_v^*}{\bar{S}_v^* \bar{I}_h^* I_v(t)}\right). \end{aligned} \tag{4.3}$$

Using $f(x) = x - 1 - \ln x \geq 0$ for $x > 0$, $\bar{\mathcal{R}}_0 > 1$, we have $\frac{d\bar{W}(t)}{dt} \leq 0$. Let

$$\bar{\Omega} = \{(S_v, I_v, S_h, I_h) \in X | \frac{d\bar{W}(t)}{dt} = 0\}.$$

I We can verify that $\frac{d\bar{W}(t)}{dt} = 0$ if and only if $S_h(t) = \bar{S}_h^*$, $I_h(t) = \bar{I}_h^*$, $S_v(t) = \bar{S}_v^*$, $I_v(t) = \bar{I}_v^*$, Hence, the largest invariant set in $\bar{\Omega}$ is the singleton $\{\bar{E}^*\}$. By LaSalle's invariance principle, we obtain the following result.

Theorem 4.1 *If $\bar{\mathcal{R}}_0 > 1$, then the positive equilibrium $\bar{E}^*$ of system (4.1) is globally asymptotically stable.*

Corresponding to system (3.1), now we consider the following discrete delay model with direct transmission

$$\begin{cases} \frac{dS_h(t)}{dt} = \Lambda_h - \beta_{h_1} S_h(t) I_v(t) - \beta_{h_2} S_h(t) I_h(t) - \mu_h S_h(t), \\ \frac{dI_h(t)}{dt} = \beta_{h_1} S_h(t-\tau_1) I_v(t-\tau_1) e^{-(\mu_h+\alpha_h)\tau_1} \\ \qquad +\beta_{h_2} S_h(t-\tau_1) I_h(t-\tau_1) e^{-(\mu_h+\alpha_h)\tau_1} - (\mu_h+\alpha_h) I_h(t), \\ \frac{dS_v(t)}{dt} = \Lambda_v - \beta_v S_v(t) I_h(t) - \mu_v S_v(t), \\ \frac{dI_v(t)}{dt} = \beta_v S_v(t-\tau_2) I_h(t-\tau_2) e^{-\mu_v \tau_2} - \mu_v I_v(t). \end{cases} \tag{4.4}$$

The initial conditions of (4.4) are similar to those in (4.1). All parameters are positive constants. By directly computing, the basic reproduction number of system (4.4) is given by

$$\hat{\hat{\mathcal{R}}}_0 = \frac{\beta_{h_1}\beta_v \Lambda_h \Lambda_v e^{-(\mu_h+\alpha_h)\tau_1} e^{-\mu_v \tau_2}}{\mu_h \mu_v^2 (\mu_h+\alpha_h)} + \frac{\beta_{h_2} \Lambda_h e^{-(\mu_h+\alpha_h)\tau_1}}{\mu_h(\mu_h+\alpha_h)}.$$

There exist two possible equilibria (*i.e.*, the trivial equilibrium $\mathcal{E}_0$ and the positive equilibrium $\mathcal{E}^*$ if $\hat{\hat{\mathcal{R}}}_0 > 1$). In Wei et al. (2008), Wei et al. investigated the dynamics of system (4.4) with $\tau_1 = 0$. However, complete global dynamics of (4.4) have not been studied. By similar analysis in Theorems 3.1–3.2 in Sect. 3, we have the following result.

Theorem 4.2 *If $\hat{\hat{\mathcal{R}}}_0 < 1$, then the trivial equilibrium $\mathcal{E}_0$ of system (4.4) is globally asymptotically stable; If $\hat{\hat{\mathcal{R}}}_0 > 1$, then the positive equilibrium $\mathcal{E}^*$ of system (4.4) is globally asymptotically stable.*

5 Numerical simulations

In this section, we present some numerical simulations to show the impact of the time delays on stability of the endemic equilibrium in system (2.1). We choose some parameter values from Chitnis et al. (2006), Ruan et al. (2008), Smith and McKenzie (2004) (see Table 1). The units of all parameters are day^{-1} except for τ_h, τ_v which are in days. The gamma distribution Cushing (1977)

$$g_{n,b_i}(\tau) = \frac{n^n \tau^{(n-1)}}{(n-1)! b_i^n} e^{-n\tau/b_i}, \quad i = 1, 2, \tag{5.1}$$

is used, where $b_i > 0$ is the mean value and $n \geq 1$ is an integer-valued shape parameter.

First, let $g_h(\tau) = g_{n_1,b_1}(\tau)$, $g_v(\tau) = g_{n_2,b_2}(\tau)$, $\Lambda_h = 0.001$, $\beta_h = 0.001$, $\mu_h = 0.00004$, $\mu_v = 0.0333$, $\alpha_h = 0.002$, $\Lambda_v = 6$, $\beta_v = 0.025$, $b_1 = 20$, $b_2 = 14$, $n_1 = 2$, $n_2 = 3$. The infection-free equilibrium of model (2.1) is stable for $\mathcal{R}_0 = 0.4947$ with $\tau_h = 12$, $\tau_v = 8$ (see Fig. 1a). The endemic equilibrium of model (2.1) is stable when $\mathcal{R}_0 = 2.8519$ with $\tau_h = 12$, $\tau_v = 8$ (see Fig. 1b) and when $\mathcal{R}_0 = 2.8519$ with $\tau_h = 22$, $\tau_v = 12$, $\beta_h = 0.006$, $\beta_v = 0.025$ (see Fig. 1c). These verify the conclusions in Theorems 2.7–2.8.

Table 1 Model parameters and their interpretations

Description	Parameter	Range	References
Human birth rate	Λ_h	0.0027–0.01	Chitnis et al. (2006), Ruan et al. (2008) , Smith and McKenzie (2004)
Natural death rate of humans	μ_h	1×10^{-6}–2×10^{-4}	Chitnis et al. (2006), Ruan et al. (2008), Smith and McKenzie (2004)
Rate of humans recovery	α_h	0.010–0.05	Chitnis et al. (2006), Ruan et al. (2008), Smith and McKenzie (2004)
Mosquito birth rate	Λ_v	3–8	Chitnis et al. (2006), Ruan et al. (2008), Smith and McKenzie (2004)
Natural death rate of mosquitoes	μ_v	0.050–0.1	Chitnis et al. (2006), Ruan et al. (2008), Smith and McKenzie (2004)
Parasite transmission probability			
From mosquito to human	β_h	0.0010–0.8	Chitnis et al. (2006), Ruan et al. (2008), Smith and McKenzie (2004)
Parasite transmission probability			
From human to mosquito	β_v	0.0072–0.64	Chitnis et al. (2006), Ruan et al. (2008), Smith and McKenzie (2004)
Incubation periodic for *P. vivax* in humans	τ_1	$5-24$	Chitnis et al. (2006), Ruan et al. (2008), Smith and McKenzie (2004)
Incubation periodic in mosquitoes	τ_2	5–18	Chitnis et al. (2006), Ruan et al. (2008), Smith and McKenzie (2004)

From Fig. 1, it can been seen that the endemic equilibrium of system (2.1) depends on the delays and the equilibrium level of infective individuals of system (2.1) decreases with the increase of the delays τ_h and τ_v. But there is no change in the stability of the endemic equilibrium. It remains stable irrespective of the delay parameters.

Next we show that system (3.1) keeps similar dynamics with two different infectivity indexes $g_{h_1}(\tau)$, $g_{h_2}(\tau)$, see Fig. 2. That is, the densities in components of system (3.1) go toward their corresponding endemic equilibrium values M^* when the basic reproduction number $\hat{\mathcal{R}}_0 > 1$. Here, we let $g_{h_1}(\tau) = g_{n_1,b_1}(\tau)$, $g_{h_2}(\tau) = g_{\widetilde{n}_1,\widetilde{b}_1}(\tau)$, $g_v(\tau) = g_{n_2,b_2}(\tau)$ in Eq. (5.1). The parameter values used in these simulations are same as in Fig. 1 except for $\widetilde{n}_1 = 1,\ 4, \widetilde{b}_1 = 10,\ 16$.

Finally, in Fig. 3 we show that the endemic equilibrium of system (4.4) is stable and it is varying as the incubation delay τ_1 and τ_2 vary, respectively. Here $\beta_{h_1} =$

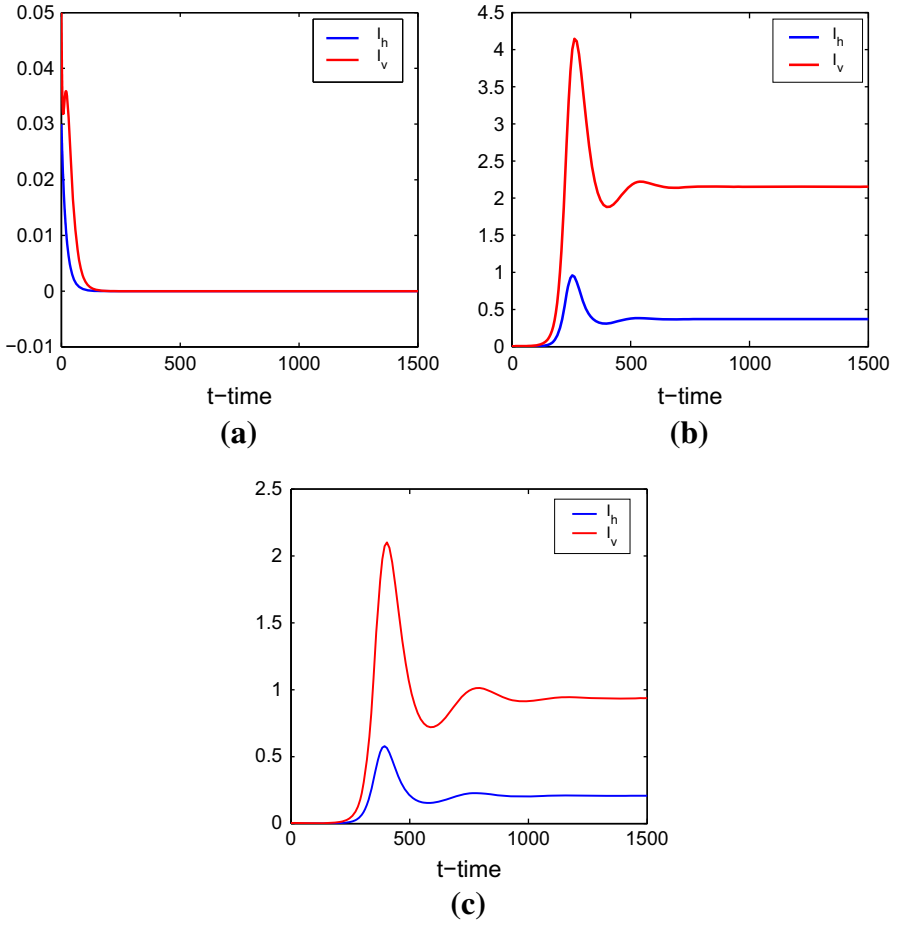

Fig. 1 **a** The infection-free equilibrium of model (2.1) is asymptotically stable for $\mathcal{R}_0 = 0.4947$ with $\tau_h = 12$, $\tau_v = 8$. The endemic equilibrium of model (2.1) is asymptotically stable for **b** $\mathcal{R}_0 = 2.8519$ with $\tau_h = 12$, $\tau_v = 8$ and **c** for $\mathcal{R}_0 = 2.8519$ with $\tau_h = 22$, $\tau_v = 12$ in the *right* figure

0.006, $\beta_{h_2} = 0.0001$. All the parameter values used in these simulations are given in Table 1. The numerical simulations show that the endemic equilibrium values are more sensitive to the incubation period τ_2 in vector population than in the incubation period τ_1 in the host population.

6 Discussion

It has been observed that the discrete time delays may cause oscillations, bifurcations, and chaotic behaviour in vector–host epidemic models Martcheva and Prosper (2013). In this paper, we have investigated the effect the distributed delays on the vector–host disease dynamics.

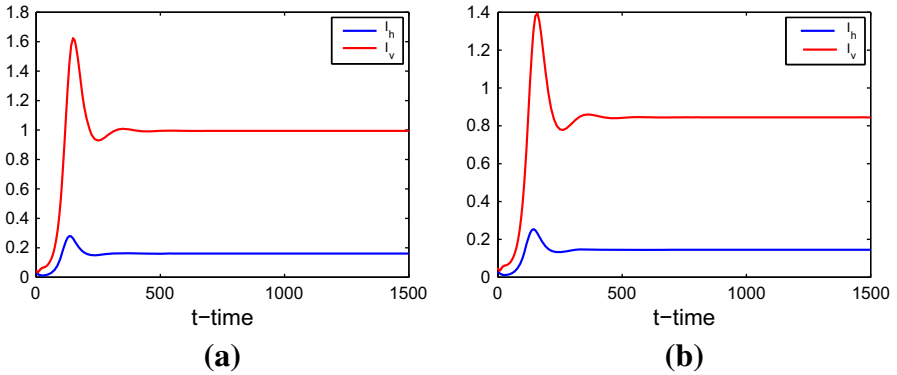

Fig. 2 The endemic equilibrium of model (3.1) is asymptotically stable for **a** $\hat{\mathcal{R}}_0 = 3.6668$ with $\tau_h = 10, \tau_v = 8, n_1 = 2, b_1 = 20, \widetilde{n}_1 = 1, \widetilde{b}_1 = 10$, and **b** $\hat{\mathcal{R}}_0 = 3.6668$ with $\tau_h = 12, \tau_v = 10, n_1 = 2, b_1 = 10, \widetilde{n}_1 = 4, \widetilde{b}_1 = 16$

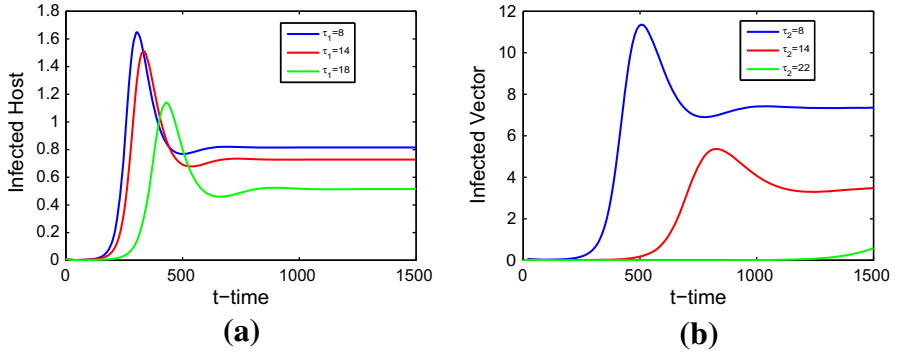

Fig. 3 The endemic equilibrium of infected host of system (4.4) is asymptotically stable for $\hat{\tilde{\mathcal{R}}}_0 > 1$. **a** $\tau_2 = 8$ and increasing the incubation period in the hosts τ_1 will decrease the numbers of the infective host populations, and **b** $\tau_1 = 8$ and the endemic equilibrium value of the infective vectors decreases and eventually approaches to zero as the incubation periods in the vectors τ_2 increases

Firstly, two distributed time delays, representing the intrinsic and extrinsic incubation periods of parasites within the hosts and vectors, respectively, have been incorporated in the vector–host disease models. By applying the persistence theory recently developed by Magal and Zhao (2005) for infinite-dimensional systems, we have shown that the model is uniformly persistent. By constructing suitable Liapunov functionals, we have studied the global stability of equilibria in systems (2.1) and (3.1), respectively. Our results show that the global stability of the equilibria in the distributed delay models is completely determined by the basic reproductive number $\mathcal{R}_0$; *i.e.,* if $\mathcal{R}_0 < 1$, the disease-free equilibrium is globally asymptotically stable; if $\mathcal{R}_0 > 1$, a unique endemic equilibrium exists and is globally asymptotically stable. As a result, no non-constant periodic solutions can exist for all positive values of parameters. These results suggest that introducing the intrinsic and extrinsic incubation periods of parasites within the hosts and vectors does not change the stability (note that the basic reproductive ratio is delay dependent in this case).

Our methods and results also apply to Ross–MacDonald type vector–host disease models with discrete delays. For a simplified two-component (infective vectors and hosts) Ross–MacDonald malaria model with two discrete delays, Ruan et al. (2008, Theorem 2.3) showed that under some conditions, the positive equilibrium $\bar{E}^*$ is locally asymptotically stable for $\bar{\mathcal{R}}_0 > 1$. For a four-component (susceptible vectors and hosts, and infective vectors and hosts) vector–host disease models with two discrete delays, in Theorem 4.1 we have showed that $\bar{E}^*$ is not only locally asymptotically stable but also global asymptotically stable for $\bar{\mathcal{R}}_0 > 1$. Thus, we not only generalize the model and results in Ruan et al. (2008) but also confirm the numerical simulation conjecture in Ruan et al. (2008, Theorem 2.3) *that the positive equilibrium $\bar{E}^*$ of the delay Ross–Macdonald is asymptotically stable for all delays values as long as the basic reproduction number is greater than one.* In Theorem 4.2 we have also showed that the positive equilibrium $\mathcal{E}^*$ of (4.4) is indeed globally asymptotically stable for $\hat{\bar{\mathcal{R}}}_0 > 1$. Thus, Hopf bifurcation does not occur in our models when the delays vary and there are no bifurcating periodic solutions due to the increase of the delay values. Hence, we have improved the stability results in Wei et al. (2008).

Our obtained results show that vector–host disease models with incubation delays have some interesting features. First, the disease can persist in a population when the number of vectors is greater than a given threshold. Secondly, the prevalence of infection in vectors and hosts depends directly on the basic reproduction number and the relationship is nonlinear. Thirdly, the model has a stable positive equilibrium when the basic reproduction number is greater than one. This means that temporary intervention can lead to a temporary reduction of prevalence, when the intervention is relaxed prevalence again increases to the original values (Koella 1991; Smith and McKenzie 2004). Our analytic results have also showed that incubation periods can play significant role in affecting the disease transmission. That is, prolong (via medical drug or control measures) either of the incubation periods could reduce the numbers of the infected host and vector populations and thus control the disease. Of course, as Ruan et al. (2008) pointed out, the longer incubation period may let the exposed hosts and vectors to spread the parasites to different locations. Therefore, it is more reasonable to consider the combined effect of varying incubation periods and spatial structure in order to model vector–host interactions and understand the spatial spread of vector-borne diseases. For example, introduce incubation periods into the multi-patch vector–host disease models (Cosner et al. 2009; Gao et al. 2014). We leave these for future consideration.

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