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Global dynamics of a time-delayed echinococcosis transmission model

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Abstract

In this paper, we present a time-delayed echinococcosis transmission model to explore effective control and prevention strategies. We first give the basic reproduction number R_0. It is shown that if $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable, and if $R_0 > 1$, the disease persists. We further show that the endemic equilibrium is globally asymptotically stable for a special case. Numerical simulations are performed to illustrate our analytic results. We give some sensitivity analysis of some parameters and give some useful comments on controlling the transmission of echinococcosis.

Keywords: echinococcosis transmission; uniform persistence; Lyapunov functional; global stability

1 Introduction

Echinococcosis, also called hydatid disease, hydatidosis, or echinococcal disease, is a parasitic disease of tapeworms of the Echinococcus type. The disease occurs in most areas of the world and currently affects about one million people. In some areas of South America, Africa, and Asia up to 10% of certain populations are affected [1]. In 2010, it caused about 1,200 deaths down from 2,000 in 1990 [2]. The economic cost of the disease is estimated to be around 3×10^9 USD a year. It can affect both humans and other animals such as pigs, cows, and horses [1]. The most common form found is cystic echinococcosis (also known as unilocular echinococcosis), which is caused by Echinococcus granulosus. The second most common form is alveolar echinococcosis, which is caused by Echinococcus multilocularis.

Like many other parasite infections, the course of Echinococcus infection is complex. The worm has a life cycle that requires definitive hosts and intermediate hosts. Definitive hosts are normally carnivores such as dogs, while intermediate hosts are usually herbivores such as sheep and cattle. Humans function as accidental hosts, because they are usually a dead-end for the parasitic infection cycle.

There are three development stages in the life cycle of Echinococcus, including egg, larva, and adult. An adult worm resides in the small intestine of a definitive host. Afterwards, gravid proglottids release eggs that are passed in the feces of the definitive host. The egg is then ingested by an intermediate host. The egg then hatches in the small intestine of the intermediate host and releases an oncosphere that penetrates the intestinal wall and moves through the circulatory system into different organs, in particular the liver

and lungs. Once it has invaded these organs, the oncosphere develops into a cyst. The cyst then slowly enlarges, creating protoscolices and daughter cysts within the cyst. The definitive host then becomes infected after ingesting the cyst-containing organs of the infected intermediate host. After ingestion, the protoscolices attach to the intestine. They then develop into adult worms and the cycle starts all over again.

In China, there are 22 provinces, autonomous regions, and municipalities reported with cystic echinococcosis (CE) which was caused by Echinococcus granulosus and Echinococcus multilocularis [3, 4]. The main endemic areas are in the western and northwestern provinces and autonomous regions: Xinjiang, Gansu, Ningxia, Inner Mongolia, Qinghai, Tibet [5], and Sichuan [6, 7], where extensively developed livestock husbandry maintains stable transmission cycles of Echinococcus granulosus. The number of domestic animals being faced with the infection of echinococcosis is more than 10^8, in which the amount of dogs is at least 5×10^6 [8].

Much has been done in terms of modeling and analysis of disease transmission of Echinococcus (see [9–13]). In [14], in order to explore effective control and prevention measures the authors proposed a deterministic model to study the transmission dynamics of echinococcosis in Xinjiang. The results showed that the dynamics of the model was completely determined by the basic reproductive number R_0. Du *et al.* [15] proposed an echinococcosis transmission model with saturation incidence, and they also established a threshold type result, which states that when $R_0 < 1$, the disease will die out; when $R_0 > 1$ and the recovery rate of dogs is very small, the disease will persist.

In this paper, we focus on the Echinococcus granulosus, which is the most common cause of human hydatid disease. The egg needs 5 to 6 months to develop into a larva in the intermediate hosts, and protoscoleces may develop into adult worms in about 1.5 to 2 months [16] in the definitive hosts. In view of realistic considerations, we take two time delays into account, to describe the time needed from egg to larva and from larva to adult, respectively. In fact, from the expression of R_0 in Section 2, we can see those delays reduce the values of R_0. Therefore, the neglect of the delays overestimated the infection risk.

The purpose of this paper is to study the global dynamics of a time-delayed Echinococcus transmission model. In Section 2, we present the model and prove its wellposedness, also we introduce the basic reproduction number R_0. In Section 3, we show the global stability of the disease-free equilibrium when $R_0 < 1$. In Section 4, we show that the disease is uniformly persistent when $R_0 > 1$. In Section 5, by constructing Lyapunov functionals, we show that the endemic equilibrium is globally asymptotically stable. In Section 6, we perform some sensitivity analysis of several model parameters and give some useful comments on controlling the transmission of echinococcosis.

2 Model formulation

We divide the definitive hosts population (mainly the dogs) into three subclasses: the susceptible population, the exposed population, and the infected population, denoted by $S_1(t)$, $E_1(t)$, and $I_1(t)$, respectively, and $N_1(t) = S_1(t) + E_1(t) + I_1(t)$ is the total number of definitive hosts. The definitive hosts are infected by means of eating infected, cyst-containing organs.

We divide the intermediate hosts population into three subclasses: the susceptible population ($S_2(t)$), the exposed population ($E_2(t)$) and the infected population ($I_2(t)$), and $N_2(t) = S_2(t) + E_2(t) + I_2(t)$ is the total number of intermediate hosts. The intermediate

hosts are infected via the ingestion of eggs. Since eggs are released by the infected definitive hosts, we assume that the amount of eggs is proportional to the amount of infected definitive hosts. It follows from [14] that the parameters of the humans do not affect the dynamical behaviors of the echinococcosis model. Hence in the paper we only consider definitive hosts and intermediate hosts in our model.

An infectious individual can contact a finite number of individuals in one time unit in a large population. The standard incidence rate seems more reasonable than the bilinear incidence rate in [14]. Therefore, in our model, we discuss the dynamic behavior for an echinococcosis model with standard incidence rate. Then we take the following model:

$$\begin{aligned}
&\frac{dS_1(t)}{dt} = A_1 - \frac{\beta_1 S_1(t) I_2(t)}{N_1(t)} - d_1 S_1(t) + \sigma I_1(t),\\
&\frac{dE_1(t)}{dt} = \frac{\beta_1 S_1(t) I_2(t)}{N_1(t)} - d_1 E_1(t) - \beta_1 e^{-d_1\tau_1} \frac{S_1(t-\tau_1) I_2(t-\tau_1)}{N_1(t-\tau_1)},\\
&\frac{dI_1(t)}{dt} = \beta_1 e^{-d_1\tau_1} \frac{S_1(t-\tau_1) I_2(t-\tau_1)}{N_1(t-\tau_1)} - (d_1+\sigma) I_1(t),\\
&\frac{dS_2(t)}{dt} = A_2 - \frac{\beta_2 S_2(t) I_1(t)}{N_2(t)} - d_2 S_2(t),\\
&\frac{dE_2(t)}{dt} = \frac{\beta_2 S_2(t) I_1(t)}{N_2(t)} - d_2 E_2(t) - \beta_2 e^{-d_2\tau_2} \frac{S_2(t-\tau_2) I_1(t-\tau_2)}{N_2(t-\tau_2)},\\
&\frac{dI_2(t)}{dt} = \beta_2 e^{-d_2\tau_2} \frac{S_2(t-\tau_2) I_1(t-\tau_2)}{N_2(t-\tau_2)} - (d_2+\varepsilon_2) I_2(t).
\end{aligned} \tag{1}$$

All parameters are assumed nonnegative. For the definitive hosts population, A_1 describes the annual recruitment rate; d_1 is the natural death rate; σ denotes the recovery rate of transition from infected to susceptible definitive hosts, including the natural recovery rate and recovery due to anthelmintic treatment; $\frac{\beta_1 S_1(t) I_2(t)}{N_1(t)}$ describes the transmission of echinococcosis between susceptible definitive hosts and infectious intermediate hosts after the ingestion of cyst-containing organs of the infected intermediate hosts. For the intermediate hosts, A_2 is the annual recruitment rate; d_2 is the death rate; $\frac{\beta_2 S_2(t) I_1(t)}{N_2(t)}$ describes the transmission of echinococcosis to intermediate hosts by the ingestion of Echinococcus eggs in the environment, ε_2 is the death-induced death rate. τ_1 is the time needed for eggs to develop into larvae in the intermediate hosts, and τ_2 is the time needed for protoscoleces to develop into adult worms in the definitive hosts.

It is easy to see that the equations for $E_1(t)$ and $E_2(t)$ can be rewritten as two integral equations:

$$E_1(t) = \int_{t-\tau_1}^{t} e^{-d_1(t-\theta)} \frac{\beta_1 S_1(\theta) I_2(\theta)}{N_1(\theta)}\, d\theta, \tag{2}$$

$$E_2(t) = \int_{t-\tau_2}^{t} e^{-d_2(t-\theta)} \frac{\beta_2 S_2(\theta) I_1(\theta)}{N_2(\theta)}\, d\theta. \tag{3}$$

Let $\tau = \max\{\tau_1, \tau_2\}$, in view of (2) and (3), the initial conditions for system (1) take the form of

$$\begin{aligned}
&S_1(\theta) = \phi_1(\theta) \geq 0, \qquad E_1(\theta) = \phi_2(\theta) \geq 0, \qquad I_1(\theta) = \phi_3(\theta) \geq 0,\\
&S_2(\theta) = \phi_4(\theta) \geq 0, \qquad E_2(\theta) = \phi_5(\theta) \geq 0, \qquad I_2(\theta) = \phi_6(\theta) \geq 0, \quad \theta \in [-\tau, 0],
\end{aligned} \tag{4}$$

and

$$\begin{aligned}
&\sum_{i=1}^{3}\phi_i(\theta) > 0, \qquad \sum_{i=4}^{6}\phi_i(\theta) > 0, \quad \forall\theta \in [-\tau, 0],\\
&\phi_2(0) = \int_{-\tau_1}^{0} e^{d_1\theta}\frac{\beta_1\phi_1(\theta)\phi_6(\theta)}{\sum_{i=1}^{3}\phi_i(\theta)}\,d\theta,\\
&\phi_5(0) = \int_{-\tau_2}^{0} e^{d_2\theta}\frac{\beta_2\phi_4(\theta)\phi_3(\theta)}{\sum_{i=4}^{6}\phi_i(\theta)}\,d\theta,
\end{aligned} \tag{5}$$

where $\Phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6) \in C^+([-\tau, 0], \mathbb{R}^6_+)$, the space of continuous functions mapping $[-\tau, 0]$ into $\mathbb{R}^6_+$.

By a similar proof to Theorem 1 of [14], we can show the following.

Lemma 2.1 *The solutions of system* (1) *with initial conditions* (4) *and* (5) *satisfy* $S_1(t) > 0$, $E_1(t) \geq 0$, $I_1(t) \geq 0$, $S_2(t) > 0$, $E_2(t) \geq 0$, $I_2(t) \geq 0$ *for all* $t > 0$.

For any $\varepsilon > 0$, we define Ω_ε as

$$\Omega_\varepsilon = \left\{(S_1, E_1, I_1, S_2, E_2, I_2) \in \mathbb{R}^6_+ : S_1 + E_1 + I_1 \leq \frac{A_1}{d_1} + \varepsilon, S_2 + E_2 + I_2 \leq \frac{A_2}{d_2} + \varepsilon\right\}.$$

Lemma 2.2 *All solutions of system* (1) *with initial conditions* (4) *and* (5) *ultimately turn into region* Ω_ε *as* $t \to \infty$.

Proof From system (1), we have

$$\begin{aligned}
&\frac{dN_1(t)}{dt} = A_1 - d_1N_1(t),\\
&\frac{dN_2(t)}{dt} = A_2 - d_2N_2(t) - \varepsilon_2I_2(t) \leq A_2 - d_2N_2(t).
\end{aligned}$$

For the system

$$\frac{dx(t)}{dt} = A_i - d_ix(t), \quad i = 1, 2,$$

the equilibrium $x^* = \frac{A_i}{d_i}$ is globally asymptotically stable. By the comparison principle, it follows that

$$\limsup_{t\to\infty}\big(N_1(t), N_2(t)\big) \leq \left(\frac{A_1}{d_1}, \frac{A_2}{d_2}\right).$$

Hence, for any $\varepsilon > 0$, there is a $t_1 > 0$ such that

$$\begin{aligned}
&S_1(t) + E_1(t) + I_1(t) = N_1(t) \leq \frac{A_1}{d_1} + \varepsilon,\\
&S_2(t) + E_2(t) + I_2(t) = N_2(t) \leq \frac{A_2}{d_2} + \varepsilon \quad \text{for all } t \geq t_1.
\end{aligned}$$

This completes the proof. □

Remark 2.1 Lemma 2.2 tells us that all feasible solutions of model (1) enter or remain in the region Ω_ε as t becomes large enough. Hence, the dynamics of model (1) can be considered only in Ω_ε.

It is easy to check that $E_0 = (\frac{A_1}{d_1}, 0, 0, \frac{A_2}{d_2}, 0, 0)$ is the disease-free equilibrium of (1), and it exists for all nonnegative values of the parameters. According to the idea in [17], we obtain the basic reproduction number

$$R_0 = \sqrt{\frac{\beta_1\beta_2 e^{-d_1\tau_1}e^{-d_2\tau_2}}{(d_1+\sigma)(d_2+\varepsilon_2)}}.$$

Remark 2.2 Near the disease-free equilibrium E_0, each infected intermediate host produces $\frac{\beta_1 e^{-d_1\tau_1}}{d_2+\varepsilon_2}$ new infected definitive hosts over its expected infectious period, and each definitive host produces $\frac{\beta_2 e^{-d_2\tau_2}}{d_1+\sigma}$ new infected intermediate hosts over its expected infectious period. The square root arises from the two 'generations' required for an infected definitive host or intermediate host to 'reproduce' itself.

3 Global stability of E_0

For the disease-free equilibrium E_0, we will show that the disease dies out if $R_0 < 1$.

Theorem 3.1 *The disease-free equilibrium* $E_0 = (\frac{A_1}{d_1}, 0, 0, \frac{A_2}{d_2}, 0, 0)$ *is unstable if* $R_0 > 1$, *and it is globally asymptotically stable if* $R_0 < 1$.

Proof The characteristic equation of system (1) at E_0 is

$$(\lambda + d_1)^2(\lambda + d_2)^2 f(\lambda) = 0, \tag{6}$$

where

$$f(\lambda) = \lambda^2 + (d_1 + \sigma + d_2 + \varepsilon_2)\lambda + (d_1+\sigma)(d_2+\varepsilon_2) - \beta_1\beta_2 e^{-d_1\tau_1}e^{-d_2\tau_2}e^{-\lambda(\tau_1+\tau_2)}.$$

Clearly, (6) has four negative roots $\lambda_1 = \lambda_2 = -d_1$, $\lambda_3 = \lambda_4 = -d_2$. Therefore, the stability of E_0 is determined by the distribution of roots of $f(\lambda) = 0$.

Note that if $R_0 = \sqrt{\frac{\beta_1\beta_2 e^{-d_1\tau_1}e^{-d_2\tau_2}}{(d_1+\sigma)(d_2+\varepsilon_2)}} > 1$, then $f(0) = (d_1+\sigma)(d_2+\varepsilon_2) - \beta_1\beta_2 e^{-d_1\tau_1}e^{-d_2\tau_2} < 0$, and $f(+\infty) = \infty$. Hence, $f(\lambda) = 0$ has at least one positive root and E_0 is unstable.

If $R_0 < 1$, we define $m_1 = d_1 + \sigma + d_2 + \varepsilon_2$, $m_2 = (d_1+\sigma)(d_2+\varepsilon_2)$. Let $\lambda = u + iv$ with $u, v \in \mathbb{R}$ be a root of $f(\lambda) = 0$. Then we have

$$\begin{aligned} &u^2 - v^2 + m_1 u + m_2 = R_0^2 m_2 e^{-u(\tau_1+\tau_2)}\cos\big((\tau_1+\tau_2)v\big),\\ &2uv + m_1 v = -R_0^2 m_2 e^{-u(\tau_1+\tau_2)}\sin\big((\tau_1+\tau_2)v\big), \end{aligned}$$

thus,

$$\begin{aligned} &\big(u^2+v^2\big)^2 + 2u^2(m_1u+m_2) + (m_1u+m_2)^2 + 2m_1uv^2 + \big[(d_1+\sigma)^2 + (d_2+\varepsilon_2)^2\big]v^2\\ &\quad = R_0^4 m_2^2 e^{-2u(\tau_1+\tau_2)}. \end{aligned} \tag{7}$$

If $u \geq 0$, then each term in the left hand side of (7) is nonnegative, furthermore, $(m_1 u + m_2)^2 \geq m_2^2$, while for the right hand side of (7), $R_0^4 m_2^2 e^{-2u(\tau_1+\tau_2)} \leq R_0^4 m_2^2 < m_2^2$, a contradiction. This shows that all roots of $f(\lambda) = 0$ must have negative real parts. Therefore, E_0 is locally asymptotically stable.

To complete the proof of Theorem 3.1, we only need to show that E_0 is globally attractive under the condition $R_0 < 1$. From system (1), we obtain

$$\begin{aligned}
&\frac{dI_1(t)}{dt} \leq \beta_1 e^{-d_1\tau_1} I_2(t-\tau_1) - (d_1+\sigma) I_1(t),\\
&\frac{dI_2(t)}{dt} \leq \beta_2 e^{-d_2\tau_2} I_1(t-\tau_2) - (d_2+\varepsilon_2) I_2(t).
\end{aligned}$$

Consider the following linear system:

$$\begin{aligned}
&\frac{dx_1(t)}{dt} = \beta_1 e^{-d_1\tau_1} x_2(t-\tau_1) - (d_1+\sigma) x_1(t),\\
&\frac{dx_2(t)}{dt} = \beta_2 e^{-d_2\tau_2} x_1(t-\tau_2) - (d_2+\varepsilon_2) x_2(t),
\end{aligned} \tag{8}$$

this is a cooperative and irreducible system of delay differential equations with a unique equilibrium $(0,0)$; the characteristic equation at $(0,0)$ is

$$f(\lambda) = 0.$$

Since all roots of $f(\lambda) = 0$ have negative real parts, E_0 is locally asymptotically stable. Therefore,

$$\lim_{t\to\infty} \big(x_1(t), x_2(t)\big) = (0,0).$$

An application of the standard comparison argument yields

$$\lim_{t\to\infty} \big(I_1(t), I_2(t)\big) = (0,0).$$

Hence, we have the following limiting system:

$$\begin{aligned}
&\frac{dS_1(t)}{dt} = A_1 - d_1 S_1(t),\\
&\frac{dE_1(t)}{dt} = -d_1 E_1(t),\\
&\frac{dS_2(t)}{dt} = A_2 - d_2 S_2(t),\\
&\frac{dE_2(t)}{dt} = -d_2 E_2(t),
\end{aligned}$$

which implies that

$$\lim_{t\to\infty} \big(S_1(t), E_1(t), S_2(t), E_2(t)\big) = \left(\frac{A_1}{d_1}, 0, \frac{A_2}{d_2}, 0\right).$$

Thus, according to the theory of asymptotically autonomous semiflows [18], we find that the disease-free equilibrium E_0 is globally asymptotically stable if $R_0 < 1$. This completes the proof. □

4 Uniform persistence

Using the method in [17], we now consider the issue of disease persistence.

Theorem 4.1 *If $R_0 > 1$, then there exists an $\eta > 0$ such that every solution $(S_1(t), E_1(t), I_1(t), S_2(t), E_2(t), I_2(t))$ of system* (1) *with initial conditions* (4) *and* (5), $\phi_3(0) \neq 0$, $\phi_6(0) \neq 0$ *satisfies*

$$\liminf_{t\to\infty}\big(I_1(t), I_2(t)\big) \geq (\eta, \eta).$$

Proof Let

$$X = \big\{\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6) \in C^+\big([-\tau, 0], \mathbb{R}^6_+\big) | \phi \text{ satisfies condition (5)}\big\},$$
$$X_0 = \big\{\phi \in X | \phi_3(0) \neq 0, \phi_6(0) \neq 0\big\}.$$

It follows that

$$\partial X_0 = X \setminus X_0 = \big\{\phi \in X | \phi_3(0) = 0 \text{ or } \phi_6(0) = 0\big\}.$$

We also define

$$M_\partial = \big\{\phi \in X | \Phi(t)\phi \in \partial X_0, \forall t \geq 0\big\}.$$

Let u_t be the solution of (1), let $\Phi(t) : X \to X$ be the solution semiflow associated with (1); that is, $\Phi(t)\phi = u_t(\phi)$, $\phi \in X$, $t \geq 0$. By Lemmas 2.1 and 2.2, the solutions of (1) are ultimately bounded, thus the semiflow $\Phi(t)$ is point dissipative on X, and $\Phi(t) : X \to X$ is compact for all $t > \tau$. By [19], it then follows that $\Phi(t)$ admits a global attractor, which attracts every bounded set in X.

Claim 1: There is a $\delta > 0$, such that, for any $\phi \in X_0$,

$$\limsup_{t\to\infty} \big\| \Phi(t)\phi - E_0 \big\| \geq \delta. \tag{9}$$

First we consider the following system:

$$\begin{aligned} \frac{dy_1(t)}{dt} &= \beta_1 e^{-d_1\tau_1}(1-\varepsilon)y_2(t-\tau_1) - (d_1+\sigma)y_1(t), \\ \frac{dy_2(t)}{dt} &= \beta_2 e^{-d_2\tau_2}(1-\varepsilon)y_1(t-\tau_2) - (d_2+\varepsilon_2)y_2(t). \end{aligned} \tag{10}$$

For sufficiently small $\varepsilon > 0$, let $\lambda_1(\varepsilon)$ be the principal eigenvalue of system (10). Since $R_0 > 1$, by Corollary 5.5.2 in [20] we have $\lambda_1(0) > 0$. We then can restrict ε to be small enough such that $\lambda_1(\varepsilon) > 0$. For this ε, there is a $\delta > 0$ such that

$$\frac{x_1}{x_1 + x_2 + x_3} > 1 - \varepsilon > 0,$$

and

$$\frac{x_4}{x_4+x_5+x_6} > 1-\varepsilon > 0, \quad \forall \left|(x_1,x_2,x_3,x_4,x_5,x_5) - E_0\right| < \delta.$$

Suppose (9) does not hold, then there is a $\phi \in X_0$, such that

$$\limsup_{t\to\infty} \left\| \Phi(t)\phi - E_0 \right\| < \delta. \tag{11}$$

Then there exists a $t_2 > t_1$, such that for all $t \geq t_2$, we have

$$\left\| \Phi(t)\phi - E_0 \right\| < \delta.$$

For any $\varepsilon > 0$, we can choose $t_3 > t_2$, such that for all $t \geq t_3$,

$$\frac{S_1(t)}{N_1(t)} > 1-\varepsilon, \qquad \frac{S_2(t)}{N_2(t)} > 1-\varepsilon.$$

From system (1), when $t \geq t_3$, we have

$$\begin{aligned} &\frac{dI_1(t)}{dt} \geq \beta_1 e^{-d_1\tau_1}(1-\varepsilon)I_2(t-\tau_1) - (d_1+\sigma)I_1(t), \\ &\frac{dI_2(t)}{dt} \geq \beta_2 e^{-d_2\tau_2}(1-\varepsilon)I_1(t-\tau_2) - (d_2+\varepsilon_2)I_2(t). \end{aligned} \tag{12}$$

Let $(v_1, v_2)^T$ be the positive right eigenvector associated with $\lambda_1(\varepsilon)$ for system (10), we can choose $r > 0$ small enough such that

$$rv_1 e^{\lambda_1(\varepsilon)t} \leq I_1(t), \qquad rv_2 e^{\lambda_1(\varepsilon)t} \leq I_2(t), \quad \forall t \in [t_3, t_3+\tau].$$

It is easy to see that $re^{\lambda_1(\varepsilon)t}(v_1,v_2)^T$ satisfies (10) for $t \geq t_3$. Then by the comparison principle, we get

$$\big(I_1(t), I_2(t)\big) \geq re^{\lambda_1(\varepsilon)t}(v_1,v_2) \quad \text{for all } t \geq t_3.$$

Since $\lambda_1(\varepsilon) > 0$, we have $\lim_{t\to\infty}(I_1(t), I_2(t)) = (\infty,\infty)$, a contradiction to (11). Thus (9) holds.

Denote the ω-limit set of the solution of system (1) starting in $\phi \in X$ by $\omega(\phi)$.

Claim 2: $\bigcup_{\phi\in M_\partial} \omega(\phi) = E_0$.

For any $\phi \in M_\partial$, we have $I_1(t,\phi) \equiv 0$ or $I_2(t,\phi) \equiv 0$. If $I_1(t,\phi) \equiv 0$, then from system (1), we get $\lim_{t\to\infty} S_2(t,\phi) = \frac{A_2}{d_2}$, $\lim_{t\to\infty} E_2(t,\phi) = 0$, $\lim_{t\to\infty} I_2(t,\phi) = 0$. By the theory of asymptotically autonomous semiflows [18], it follows that $\lim_{t\to\infty} S_1(t,\phi) = \frac{A_1}{d_1}$, $\lim_{t\to\infty} E_1(t,\phi) = 0$. If $I_2(t,\phi) \equiv 0$, again from system (1), we get $\lim_{t\to\infty} E_1(t,\phi) = 0$, $\lim_{t\to\infty} I_1(t,\phi) = 0$; furthermore, we obtain $\lim_{t\to\infty} S_1(t,\phi) = \frac{A_1}{d_1}$, $\lim_{t\to\infty} S_2(t,\phi) = \frac{A_2}{d_2}$, $\lim_{t\to\infty} E_2(t,\phi) = 0$. Therefore, we have $\bigcup_{\phi\in M_\partial} \omega(\phi) = E_0$.

Define $p : X \to \mathbb{R}_+$ by

$$p(\Phi) = \min\{\phi_3(0), \phi_6(0)\}, \quad \forall \Phi \in X.$$

It is easy to see that $p^{-1}(0,\infty) \subset X_0$. By (1),

$$I_1(t) = e^{-(d_1+\sigma)t}\left[I_1(0) + \int_0^t \beta_1 e^{-d_1\tau_1} e^{(d_1+\sigma)\rho} \frac{S_1(\rho-\tau_1)I_2(\rho-\tau_1)}{N_1(\rho-\tau_1)}\, d\rho\right]$$

and

$$I_2(t) = e^{-(d_2+\varepsilon_2)t}\left[I_2(0) + \int_0^t \beta_2 e^{-d_2\tau_2} e^{(d_2+\varepsilon_2)\rho} \frac{S_2(\rho-\tau_2)I_1(\rho-\tau_2)}{N_2(\rho-\tau_2)}\, d\rho\right].$$

Then $I_i(t) > 0$ for all $t > 0$, $i = 1, 2$, $\phi_3(0) \neq 0$, $\phi_6(0) \neq 0$. It follows that p has the property that if either $p(\phi) = 0$ and $\phi \in \partial X_0$, or $p(\phi) > 0$, then $p(\Phi(t)(\phi)) > 0$ for all $t > 0$. Hence, p is a generalized distance function for the semiflow $\Phi(t) : X \to X$ (see [21]). By Claim 2, we see that any forward orbit of $\Phi(t)$ in M_∂ converges to E_0. By Claim 1, we see that E_0 is an isolated invariant set in X, and that $W^s(E_0) \cap X_0 = \emptyset$, where $W^s(E_0)$ is the stable manifold of E_0. By [21], it then follows that there exists an $\eta > 0$ such that $\liminf_{t\to\infty} p(\Phi(t)\phi) \geq \eta$ for any $\phi \in X_0$. This implies that $\liminf_{t\to\infty} I_i(t) \geq \eta$, $i = 1, 2$. This completes the proof. □

5 Global stability of endemic equilibrium

In this section, we will study the global stability of endemic equilibrium of system (1). For simplicity, we assume that $\varepsilon_2 = 0$, we find that when $R_0 > 1$, system (1) has one endemic equilibrium; when $R_0 \leq 1$, there is no endemic equilibrium, system (1) has only the disease-free equilibrium E_0.

Theorem 5.1 *Assume that* $\varepsilon_2 = 0$. *If* $R_0 > 1$, *system* (1) *has a unique endemic* (*positive*) *equilibrium* $E^* = (S_1^*, E_1^*, I_1^*, S_2^*, E_2^*, I_2^*)$. *More specifically,*

(i) *If* $\sigma > 0$, $E^* = (S_1^*, E_1^*, I_1^*, S_2^*, E_2^*, I_2^*)$ *is given by*

$$S_1^* = \frac{A_1(A_1 + \sigma I_1^*)}{d_1(A_1 + \beta_1 I_2^*)},$$

$$S_2^* = \frac{A_2^2}{d_2(A_2 + \beta_2 I_1^*)},$$

$$E_1^* = \frac{\beta_1(1 - e^{-d_1\tau_1})S_1^* I_2^*}{A_1},$$

$$E_2^* = \frac{\beta_2(1 - e^{-d_2\tau_2})S_2^* I_1^*}{A_2},$$

$$I_1^* = \frac{-a_1 + \sqrt{a_1^2 - 4a_0a_2}}{2a_0},$$

$$I_2^* = \frac{\sigma[R_0^2 A_2(d_1 + \sigma - \sigma e^{-d_1\tau_1}) + \beta_2 A_1 e^{-d_1\tau_1}]I_1^*}{\beta_1\beta_2 A_1 e^{-d_1\tau_1} + \beta_1 A_2(d_1 + \sigma - \sigma e^{-d_1\tau_1})} + \frac{(d_1 + \sigma - \sigma e^{-d_1\tau_1})(R_0^2 - 1)A_1A_2}{\beta_1\beta_2 A_1 e^{-d_1\tau_1} + \beta_1 A_2(d_1 + \sigma - \sigma e^{-d_1\tau_1})},$$

where

$$
\begin{aligned}
a_0 &= \beta_1 A_2 \sigma R_0^2\left(d_1 + \sigma - \sigma e^{-d_1\tau_1}\right) + \beta_1\beta_2 A_1 \sigma e^{-d_1\tau_1},\\
a_1 &= \beta_1 A_1 A_2\left[\sigma e^{-d_1\tau_1}\left(1 - R_0^2\right) + R_0^2\left(d_1 + \sigma - \sigma e^{-d_1\tau_1}\right)\right] + \beta_1\beta_2 A_1^2 e^{-d_1\tau_1},\\
a_2 &= A_1^2 A_2 \beta_1 e^{-d_1\tau_1}\left(1 - R_0^2\right).
\end{aligned}
$$

(ii) *If* $\sigma = 0$, $E^* = (S_1^*, E_1^*, I_1^*, S_2^*, E_2^*, I_2^*)$ *is given by*

$$
\begin{aligned}
S_1^* &= \frac{A_1 d_2 (A_1\beta_2 + A_2 d_1 e^{d_1\tau_1})}{d_1\beta_2(A_1 d_2 + A_2\beta_1 e^{-d_2\tau_2})},\\
S_2^* &= \frac{A_2 d_1 (A_2\beta_1 + A_1 d_2 e^{d_2\tau_2})}{d_2\beta_1(A_2 d_1 + A_1\beta_2 e^{-d_1\tau_1})},\\
E_1^* &= \frac{A_1 A_2 d_2 (e^{d_1\tau_1} - 1)(R_0^2 - 1)}{\beta_2(A_1 d_2 + A_2\beta_1 e^{-d_2\tau_2})},\\
E_2^* &= \frac{A_1 A_2 d_1 (e^{d_2\tau_2} - 1)(R_0^2 - 1)}{\beta_1(A_2 d_1 + A_1\beta_2 e^{-d_1\tau_1})},\\
I_1^* &= \frac{A_1 A_2 d_2 (R_0^2 - 1)}{\beta_1\beta_2 A_2 e^{-d_2\tau_2} + \beta_2 A_1 d_2},\\
I_2^* &= \frac{A_1 A_2 d_1 (R_0^2 - 1)}{\beta_1\beta_2 A_1 e^{-d_1\tau_1} + \beta_1 A_2 d_1}.
\end{aligned}
\tag{13}
$$

For the special case $\varepsilon_2 = \sigma = 0$, that is, the disease-induced death rate of infected livestock population is zero ($\varepsilon_2 = 0$), we also assume that the infected dogs will not recover ($\sigma = 0$). In this case, as regards the stability of the endemic equilibrium, we have the following theorems.

Theorem 5.2 *Assume that* $\varepsilon_2 = \sigma = 0$. *If* $R_0 > 1$, *the endemic equilibrium* $E^* = (S_1^*, E_1^*, I_1^*, S_2^*, E_2^*, I_2^*)$ *of system* (1) *is locally asymptotically stable, where* E^* *is denoted by* (13).

Proof The linearization of system (1) at E^* is

$$
(\lambda + d_1)^2(\lambda + d_2)^2 g(\lambda) = 0, \tag{14}
$$

where

$$
g(\lambda) = \lambda^2 + \left(\frac{\beta_1 I_2^*}{N_1^*} + d_1 + \frac{\beta_2 I_1^*}{N_2^*} + d_2\right)\lambda + \left(\frac{\beta_1 I_2^*}{N_1^*} + d_1\right)\left(\frac{\beta_2 I_1^*}{N_2^*} + d_2\right) - d_1 d_2 e^{-\lambda(\tau_1+\tau_2)}.
$$

It is easy to see that (14) has four negative roots $\lambda_1 = \lambda_2 = -d_1$, $\lambda_3 = \lambda_4 = -d_2$. Therefore, the stability of E^* is determined by the distribution of roots of $g(\lambda) = 0$.

We define $n_1 = \frac{\beta_1 I_2^*}{N_1^*} + d_1$, $n_2 = \frac{\beta_2 I_1^*}{N_2^*} + d_2$. Let $\lambda = a + ib$ with $a, b \in \mathbb{R}$ be a root of $g(\lambda) = 0$. Then we have

$$
\begin{aligned}
a^2 - b^2 + (n_1 + n_2)a + n_1 n_2 &= d_1 d_2 e^{-a(\tau_1+\tau_2)}\cos\big((\tau_1 + \tau_2)b\big),\\
2ab + (n_1 + n_2)b &= -d_1 d_2 e^{-a(\tau_1+\tau_2)}\sin\big((\tau_1 + \tau_2)b\big),
\end{aligned}
$$

it follows that

$$\begin{aligned}&\left(a^2+b^2\right)^2+\left((n_1+n_2)a+n_1n_2\right)^2+2ab^2(n_1+n_2)+b^2\left(n_1^2+n_2^2\right)\\&\quad+2a^2\left((n_1+n_2)a+n_1n_2\right)-\left(d_1d_2e^{-a(\tau_1+\tau_2)}\right)^2=0.\end{aligned}\tag{15}$$

If $a\geq 0$, then the left hand side of (15) is greater than or equal to $(n_1n_2)^2-(d_1d_2e^{-a(\tau_1+\tau_2)})^2>0$, a contradiction. This shows that all roots of $g(\lambda)=0$ have negative real parts. Therefore, E^* is locally asymptotically stable. This completes the proof of Theorem 5.2. □

We can further give the global stability of the endemic equilibrium E^*.

Theorem 5.3 *Assume that* $\varepsilon_2=\sigma=0$. *If* $R_0>1$, *the endemic equilibrium* $E^*=(S_1^*,E_1^*,I_1^*,S_2^*,E_2^*,I_2^*)$ *of system* (1) *is globally asymptotically stable*, *where* E^* *is denoted by* (13).

Proof In Theorem 5.2, we have given the local stability of E^*. We now prove the global attractivity of E^*.

The numbers of the total populations $N_1(t)$ and $N_2(t)$ satisfy

$$\begin{aligned}&\frac{dN_1(t)}{dt}=A_1-d_1N_1(t),\\&\frac{dN_2(t)}{dt}=A_2-d_2N_2(t),\end{aligned}$$

it follows that

$$\lim_{t\to\infty}\left(N_1(t),N_2(t)\right)=\left(\frac{A_1}{d_1},\frac{A_2}{d_2}\right).$$

Therefore, $S_1(t)$, $I_1(t)$, $S_2(t)$, $I_2(t)$ satisfy the following limit system:

$$\begin{aligned}&\frac{dS_1(t)}{dt}=A_1-\tilde{\beta}_1S_1(t)I_2(t)-d_1S_1(t),\\&\frac{dI_1(t)}{dt}=\tilde{\beta}_1e^{-d_1\tau_1}S_1(t-\tau_1)I_2(t-\tau_1)-d_1I_1(t),\\&\frac{dS_2(t)}{dt}=A_2-\tilde{\beta}_2S_2(t)I_1(t)-d_2S_2(t),\\&\frac{dI_2(t)}{dt}=\tilde{\beta}_2e^{-d_2\tau_2}S_2(t-\tau_2)I_1(t-\tau_2)-d_2I_2(t),\end{aligned}\tag{16}$$

where $\tilde{\beta}_1=\frac{d_1\beta_1}{A_1}$, $\tilde{\beta}_2=\frac{d_2\beta_2}{A_2}$.

Consider the following Lyapunov functional:

$$\begin{aligned}L_1(t)&=e^{-d_1\tau_1}\left[S_1-S_1^*-S_1^*\ln\left(\frac{S_1}{S_1^*}\right)\right]+I_1-I_1^*-I_1^*\ln\left(\frac{I_1}{I_1^*}\right)\\&\quad+\tilde{\beta}_1e^{-d_1\tau_1}\int_0^{\tau_1}\left[S_1(t-r)I_2(t-r)-S_1^*I_2^*-S_1^*I_2^*\ln\left(\frac{S_1(t-r)I_2(t-r)}{S_1^*I_2^*}\right)\right]dr.\end{aligned}$$

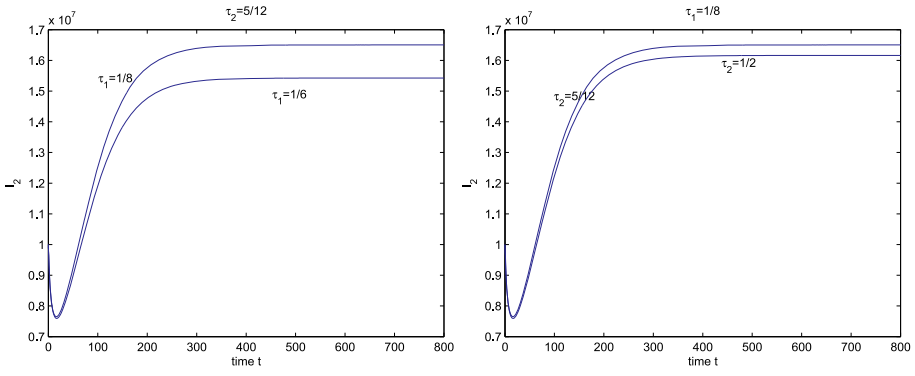

Figure 1 The influence of τ_1 and τ_2 on the number of infected intermediate hosts I_2.

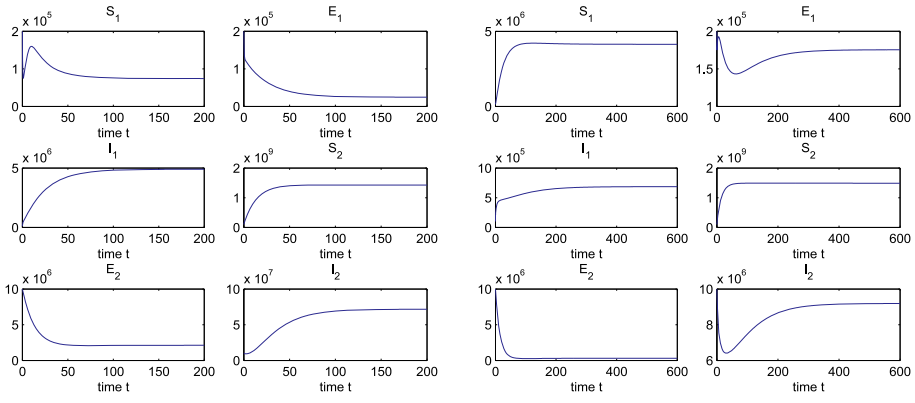

Figure 2 Long-term behavior of the population of each class when $R_0 > 1$. In the left figure, we take $\varepsilon_2 = \sigma = 0$. In the right figure, we take $\varepsilon_2 = 0.01$, $\sigma = 2$.

Note that $1 - x \le \ln(\frac{1}{x})$, $x \in R^+$, with equality only if $x = 1$, then the derivative of L_1 along the solution of (16) satisfies

$$\begin{aligned}
\frac{dL_1}{dt} &= -d_1 e^{-d_1\tau_1}\frac{(S_1 - S_1^*)^2}{S_1} + \tilde{\beta}_1 e^{-d_1\tau_1} S_1^* I_2^* \left(1 - \frac{S_1^*}{S_1} + \frac{I_2}{I_2^*} - \frac{I_1}{I_1^*}\right) \\
&\quad + \tilde{\beta}_1 e^{-d_1\tau_1} S_1^* I_2^* \left(1 - \frac{S_1(t-\tau_1)I_2(t-\tau_1)I_1^*}{S_1^* I_2^* I_1}\right. \\
&\quad \left. + \ln\big(S_1(t-\tau_1)I_2(t-\tau_1)\big) - \ln\big(S_1(t)I_2(t)\big)\right) \\
&\le \tilde{\beta}_1 e^{-d_1\tau_1} S_1^* I_2^* \left(\ln\left(\frac{S_1}{S_1^*}\right) + \frac{I_2}{I_2^*} - \frac{I_1}{I_1^*} + \ln\left(\frac{S_1^* I_2^* I_1}{S_1(t-\tau_1)I_2(t-\tau_1)I_1^*}\right)\right. \\
&\quad \left. + \ln\big(S_1(t-\tau_1)I_2(t-\tau_1)\big) - \ln\big(S_1(t)I_2(t)\big)\right) \\
&= \tilde{\beta}_1 e^{-d_1\tau_1} S_1^* I_2^* \left(\ln\left(\frac{I_2^* I_1}{I_1^* I_2}\right) + \frac{I_2}{I_2^*} - \frac{I_1}{I_1^*}\right).
\end{aligned}$$

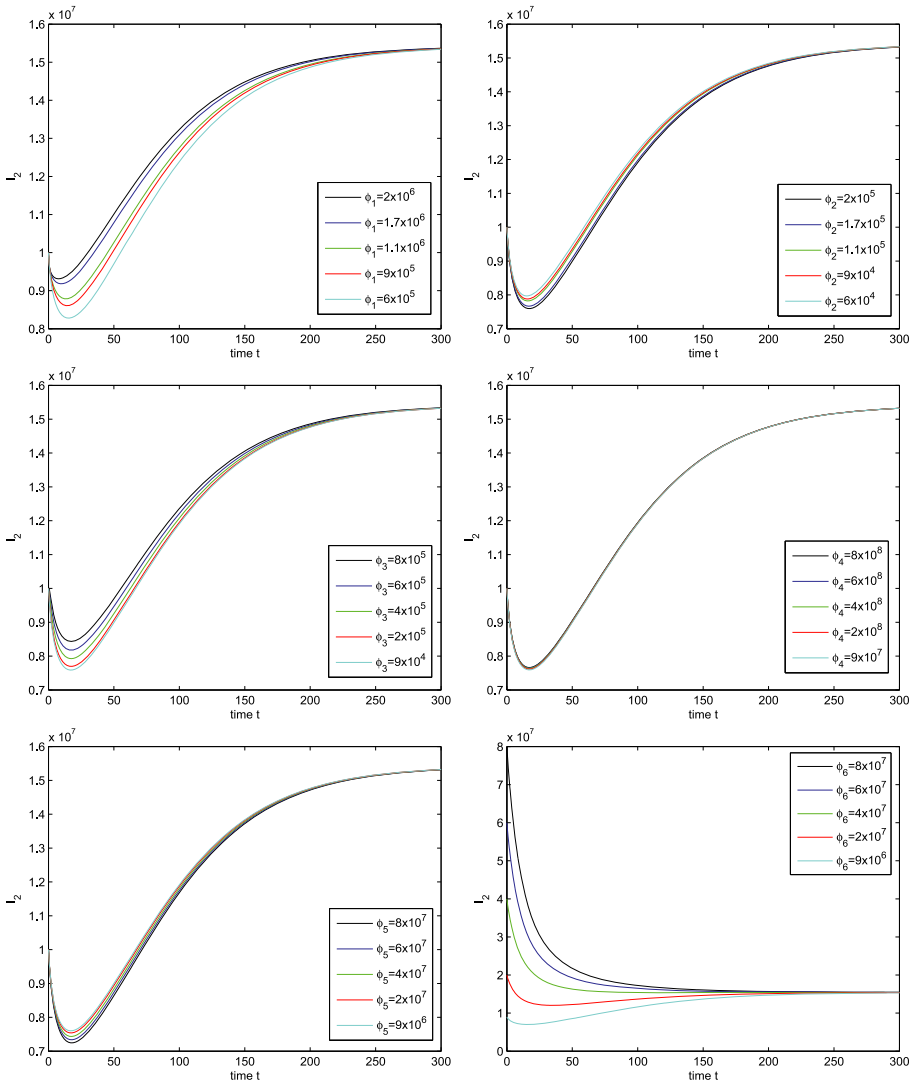

Figure 3 The influence of the initial conditions on the number of infected intermediate hosts, I_2.

If we construct another Lyapunov functional,

$$L_2(t) = e^{-d_2\tau_2}\left[S_2 - S_2^* - S_2^*\ln\left(\frac{S_2}{S_2^*}\right)\right] + I_2 - I_2^* - I_2^*\ln\left(\frac{I_2}{I_2^*}\right) + \tilde{\beta}_2 e^{-d_2\tau_2}\int_0^{\tau_2}\left[S_2(t-r)I_1(t-r) - S_2^*I_1^* - S_2^*I_1^*\ln\left(\frac{S_2(t-r)I_1(t-r)}{S_2^*I_1^*}\right)\right]dr,$$

by a similar computation to $\frac{dL_1}{dt}$, we obtain

$$\frac{dL_2}{dt} \le \tilde{\beta}_2 e^{-d_2\tau_2} S_2^* I_1^*\left(\ln\left(\frac{I_1^* I_2}{I_2^* I_1}\right) + \frac{I_1}{I_1^*} - \frac{I_2}{I_2^*}\right).$$

Finally we choose the Lyapunov functional

$$L(t) = \tilde{\beta}_2 e^{-d_2\tau_2} S_2^* I_1^* L_1(t) + \tilde{\beta}_1 e^{-d_1\tau_1} S_1^* I_2^* L_2(t),$$

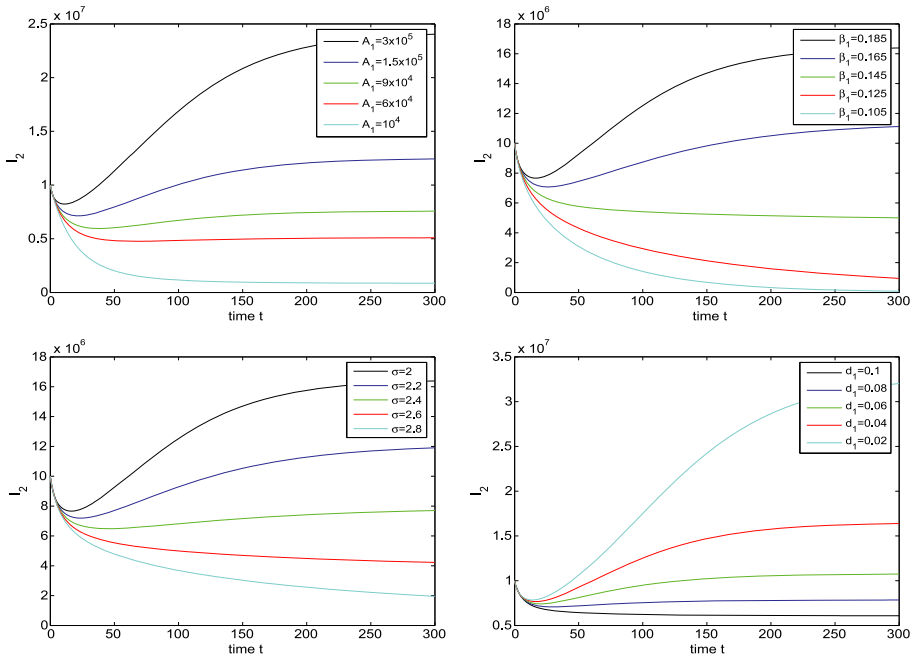

Figure 4 The influence of parameters A_1, β_1, σ, and d_1 on the number of infected intermediate hosts, I_2.

then we get

$$\frac{dL}{dt} \leq 0.$$

Let $M \subset E = \{(S_1, I_1, S_2, I_2) : \frac{dL}{dt} = 0\}$ be the largest invariant set with respect to system (16), we can show that $M = \{(S_1^*, I_1^*, S_2^*, I_2^*)\}$. By the Lasalle invariance principle, $(S_1^*, I_1^*, S_2^*, I_2^*)$ is globally attractive. From (2) and (3), we have $E_1(t) \to E_1^*$, $E_2(t) \to E_2^*$ as $t \to \infty$. Now we see that $E^* = (S_1^*, E_1^*, I_1^*, S_2^*, E_2^*, I_2^*)$ is globally attractive. This completes the proof. □

6 Numerical simulations

In this section, we carry out numerical simulations to illustrate our analytic results. Since all the parameters are not easy to find, we will assume some parameters.

In view of [22], we fix $\beta_1 = 0.185$ year^{-1}, $d_1 = 0.04$ year^{-1}, $d_2 = 0.07$ year^{-1}, we first choose $\sigma = 2$ year^{-1}, $\varepsilon_2 = 0$ year^{-1} (see [14]), $\tau_1 = \frac{1}{8} \sim \frac{1}{6}$ year, $\tau_2 = \frac{5}{12} \sim \frac{1}{2}$ year (see [16]).

First we take $A_1 = 2 \times 10^5$ year^{-1}, $A_2 = 1.05 \times 10^8$ year^{-1}, $\beta_2 = 1.11$ year^{-1}. Fixing $\tau_2 = 5/12$ year, we vary τ_1 from $\frac{1}{8}$ year to $\frac{1}{6}$ year, and from Figure 1 we see the disease of the intermediate hosts population I_2 persists; as τ_1 increases, the infection level becomes lower. If we fix $\tau_1 = 1/8$ year and vary τ_2 from $\frac{5}{12}$ year to $\frac{1}{2}$ year, also Figure 1 shows that as τ_2 increases, I_2 decreases. This figure implies that large time delays are beneficial to disease control. Also we see that τ_1 has a bigger impact than τ_2 on the infection level of I_2.

Then we take $\varepsilon_2 = \sigma = 0$; we get $R_0 = 8.4188$, and by Theorem 5.3, we find that E^* is globally asymptotically stable (see the left figure of Figure 2). But if we choose $\varepsilon_2 = 0.01$, $\sigma = 2$, then we get $R_0 = 1.1027$, and from the right figure of Figure 2, we see that there is still an endemic equilibrium E^* which is globally asymptotically stable.

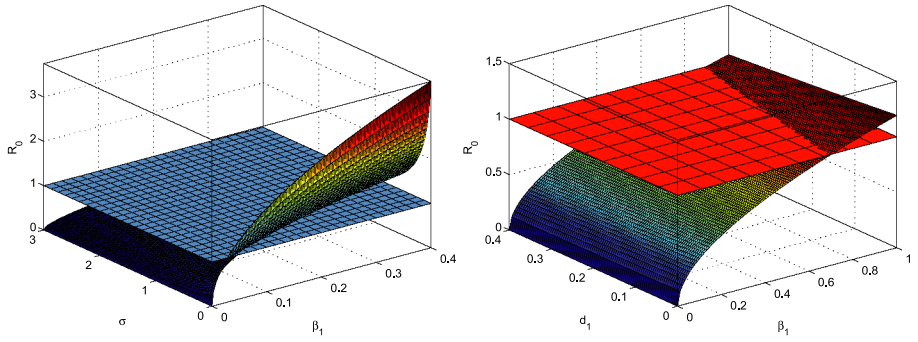

Figure 5 Surface plots of R_0 as a function of β_1, d_1, and σ.

We now investigate the impact of the initial conditions on the number of infected intermediate hosts, $I_2(t)$. From Figure 3, we can see that ϕ_1, ϕ_3, ϕ_6 have a stronger impact than the other initial values, ϕ_4 has almost no influence on $I_2(t)$. These figures show that it is very important to control the amount of susceptible and infected definitive hosts.

Next we want to see the influence of different parameters on the amount of infected intermediate hosts $I_2(t)$. The influence of A_1, β_1, σ, d_1 on $I_2(t)$ is shown in Figure 4. From the expression of R_0, we can see that R_0 decreases if β_1 decreases, or σ increases, or d_1 increases. Figure 4 also verifies this.

Figure 5 depicts the relationship of R_0 as a function of β_1, d_1, and σ, we can see that R_0 is not always less than 1.

Remark 6.1 Although A_1 does not affect the number of R_0, reducing A_1 can decrease the infection level of I_2 (see Figure 4).

Based on the above analysis, we now give some control strategies by adjusting the parameters A_1, β_1, σ, and d_1. (1) Decrease A_1 by reducing the birth rate of newborn puppies. (2) β_1 can be reduced by the following measures. Livestock slaughtering regulations and health education should be implemented in endemic areas. Infected offal should be treated harmlessly. Definitive hosts should be barred from slaughter houses. (3) σ can be increased through increasing the frequency of anticestodal drugs (*e.g.*, praziquantel). (4) In order to increase d_1, we can kill the infected definitive hosts and the stray dog populations.

Competing interests
The authors declare that they have no competing interests.

Authors' contributions
JL proposed the model and completed the main part of this manuscript, LL checked all the theorems and polished the language, XF enhanced the revised version, and JF revised the numerical simulation part. All the authors read and approved the manuscript.

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