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GLOBAL ANALYSIS FOR AN EPIDEMICAL MODEL OF VECTOR-BORNE PLANT VIRUSES WITH DISEASE RESISTANCE AND NONLINEAR INCIDENCE*

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Abstract Vector-borne disease models play an important role in understanding the mechanism of plant disease transmission. In this paper, we study a vector-borne model with plant disease resistance, disease exposed period and nonlinear incidence. We compute the basic reproduction number, determine the implicit locations of equilibria and then investigate their global stability by generalizing a classic geometric approach to higher dimensional systems. Higher dimensions cause greater difficulties such as the construction of the transformation matrix and the estimate of the *Lozinskiĭ* measure in this geometric approach. For a complete control of vector-borne diseases, a quantitative way is provided by the given expression of the basic reproduction number, from which we need not only increasing plant disease resistance but also decreasing the contact rate between infected plants and susceptible vectors instead of a single one of them.

Keywords Nonlinear incidence, plant disease resistance, vector-borne model, global stability.

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1. Introduction

Diseases caused by plant viruses seriously restrict the yield and quality of crops in China and around the world. There are many kinds of plant viruses and almost 1100 species of plant viruses have been reported (see [37]). Tomato spotted wilt virus (TSWV) is one of them. The spotted wilt disease of tomato was first described in 1915 in Australia by Brittlebank in [2]. The name tomato spotted wilt virus was first established by Samuel in [27] for characterizing the pathogenic agent as a virus. TSWV is one of the deadliest viruses with a host range of more than 1000 plant species belonging to more than 85 families including tomato, bean, lettuce, groundnut, pepper, potato and tobacco (see [14]).

TSWV is transmitted by at least 8 species of thrips and the western flower thrip (WFT) is reported to be the most important vector due to its wide distribution (see [34]). WFT life cycle consists of 6 stages: egg, two larvae stages, two transformation stages (prepupae and pupae), and an adult stage. The TSWV must be acquired by thrips during the first larval stage. Thus, only immature thrips, which acquire

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the TSWV when they are on the first larvae stage, can grow into adults that can transmit the virus. At the beginning, WFT with virus appeared in the western United States and has now spread to many countries largely through international trade. In China, symptoms of diseases on tomatoes is observed to be resembling as WFT in Sichuan province for the first time in 1944 (see [31]). Since then, increasing number of tospoviruses have been found in many regions of China and they cause significant losses of vegetables and high-value crops such as tomato, peanut, tobacco and so on (see [4, 9, 21, 31]).

Recently, mathematical models of vector-borne plant diseases have attracted the interest of many researchers, under the effect of host demography in [6], influence of weather in [23], temperature and seasonal variations in [22], different ages in [16, 28, 35] and so on. A four-dimensional model of soil-borne diseases is presented in [6] and is further developed in [29] to a five-dimensional model. After a long-term interaction, mutual adaptation, mutual selection and even co-evolution with pathogens, plants gradually acquire a series of complex defense mechanisms to protect themselves, i.e., disease resistance. For instance, five different genes for TSWV resistance in tomato have been described in [7, 12]. Thus, plant disease resistance is an important factor to be considered in modeling disease spreading. This is the motivation for us to investigate vector-borne plant diseases with disease resistance. In this paper, we study a epidemic model for TSWV transmitted by WFT with plant disease resistance, disease exposed period and nonlinear incidence. Assume that

(H1) The total number of plants is a positive constant. Diseases are not infectious during the exposed period. Plants have permanent immunity after recovery;

(H2) The susceptible plants can be infected not only by the infected vectors but also by the infected plants;

(H3) Susceptible vectors can be infected only by infected plants. Once infected, it will carry the virus for life. Further, the new born vectors are susceptible.

As in [6, 29], the total number can always be maintained by adding a new plant after the death of a plant and these new plants are susceptible. Thus, we assume that the total number of plants is a positive constant in (H1). Since infectious disease model is the simplification of reality and idealization to a certain extent, the more factors to consider the more complex the model is, the more difficult it is to analyze. So we assume that the disease is not infectious during the exposed period and plants have permanent immunity after recovery in (H1) as indicated in [35, 36, 38]. As in [18, 31], the mechanical damage of plants caused by field operation and the bite of WFT causes the transmission of TSWT between plants through body fluids. Therefore, we give assumption (H2). Assumption (H3) is based on the experimental observation of tomato plants in greenhouse in [18], where it is assumed that the vector is infected via biting on infected host plants. Once infected, it carries the virus for life as in [16].

Our model takes form

$$\begin{cases} \dot{S} = f(I) + cE + bI - \mu S - \left(\dfrac{\beta_{vh}Y}{1+\alpha_{vh}Y} + \dfrac{\beta_{hh}I}{1+\alpha_{hh}I}\right) S, \\ \dot{E} = \left(\dfrac{\beta_{vh}Y}{1+\alpha_{vh}Y} + \dfrac{\beta_{hh}I}{1+\alpha_{hh}I}\right) S - (c+\varepsilon+\mu)E, \\ \dot{I} = \varepsilon E - (b+\mu+\gamma+d)I, \\ \dot{R} = \gamma I - \mu R, \\ \dot{X} = \Lambda - \beta_{hv} X I - mX, \\ \dot{Y} = \beta_{hv} X I - mY, \end{cases} \tag{1.1}$$

where $S(t), E(t), I(t), R(t), X(t), Y(t)$ are the numbers of susceptible individuals, exposed individuals, infected individuals, recovered individuals and the densities of the susceptible vectors, infected vectors, respectively. To be more realistic, we use nonlinear incidence for plant disease. By assumption (H1) the total number of plants is a constant K. In order to keep the total number constant, we assume that the number of replants equals to $\mu K + dI$, where μK is the natural deaths and dI is the infected deaths. So $f(I) := \mu K + dI$. Here parameters $\beta_{hv}, \beta_{vh}, ..., c, d$ are all nonnegative constants and their biological meanings are given in Table 1.

Table 1. Description of parameters in system (1.1)

Parameter	Description	value
β_{hv}	infection ratio between infected hosts and susceptible vectors	fitting
β_{vh}	biting rate of an infected vector on the susceptible hosts	fitting
β_{hh}	infection incidence between infected and susceptible hosts	fitting
α_{vh}	determines the degree of infection saturation between the vector and the host	fitting
α_{hh}	determines the degree of infection saturation between hosts	fitting
γ	the conversion rate of infected hosts to recovered hosts	fitting
μ	natural death rate of hosts	[18]
Λ	birth or immigration of insect vectors	[18]
ε	is the constant rate for the exposed hosts becoming infectious	[24]
m	natural death rate of insect vectors	[35]
b	rate of the infectious individual become susceptible individual without treatment	[1]
c	rate of the exposed individual become susceptible individual without treatment	fitting
d	disease-induced mortality of infected hosts	fitting

Let $N(t)$ be the sum of total insect vectors. By the last two equations in system (1.1) we get

$$\dot{N} = \dot{X} + \dot{Y} = \Lambda - mN,$$

from which $N(t) \to \Lambda/m$ as $t \to +\infty$. Since we care about the long-time dynamical behavior, $N(t)$ is regarded as a constant Λ/m, hence, in system (1.1) we replace X by $\Lambda/m - Y$. Thus, the last two equations in (1.1) actually become one equation

$$\dot{Y} = \beta_{hv}\left(\frac{\Lambda}{m} - Y\right) I - mY.$$

Then, system (1.1) can be reduced to

$$\begin{cases} \dot{S} = \mu(K - S) + (b + d)I + cE - \left(\dfrac{\beta_{vh}Y}{1 + \alpha_{vh}Y} + \dfrac{\beta_{hh}I}{1 + \alpha_{hh}I}\right) S, \\ \dot{E} = \left(\dfrac{\beta_{vh}Y}{1 + \alpha_{vh}Y} + \dfrac{\beta_{hh}I}{1 + \alpha_{hh}I}\right) S - \xi E, \\ \dot{I} = \varepsilon E - \eta I, \\ \dot{Y} = \beta_{hv}\left(\dfrac{\Lambda}{m} - Y\right) I - mY \end{cases} \tag{1.2}$$

because of the independence of $\dot{S}, \dot{E}, \dot{I}, \dot{Y}$ on R as shown in system (1.1), where $\xi := c + \varepsilon + \mu$ and $\eta := b + \mu + \gamma + d$. Thus, as in [11, 29], the research about the stability for system (1.1) is equivalent to that of system (1.2).

In this paper we consider system (1.2) in set Ω, where

$$\Omega := \left\{(S, E, I, Y) \in \mathbb{R}^4 : \ S, E, I, Y \geq 0, 0 < S + E + I \leq K, 0 \leq Y \leq \frac{\Lambda}{m}\right\}.$$

Set Ω is a closed and positively invariant set of system (1.2), which is proved in next section. The organization of this paper is as follows. In section 2, for system (1.2) we prove the positively invariance of Ω, compute the basic reproduction number and give conditions for the existence of equilibria. In section 3, global asymptotic stability of the unique disease-free equilibrium is studied. In section 4, we give condition for global asymptotic stability of endemic equilibria. In section 5, some numerical simulations are done to illustrate our theoretical results.

2. Positively invariance of Ω and existence of equilibria

In this section we first prove the positively invariance of Ω for system (1.2) and then compute the equilibria for different cases of the basic reproduction number R_0.

Biologically, we do not consider the case that $(S(0), E(0), I(0), R(0)) = (0, 0, 0, K)$ because in such case we consider another family of plants by assumption (H1). From the sense of mathematics, for any given initial value $(S(0), E(0), I(0), Y(0))$ satisfying $S(0), E(0), I(0), Y(0) \geq 0, 0 < S(0) + E(0) + I(0) \leq K, 0 \leq Y(0) \leq \Lambda/m$, the orbit of (1.2) does not pass through $(0, 0, 0, Y(t))$ for all $t \neq 0$ because $(S(t) + E(t) + I(t))' = \mu K - \mu(S + E + I) - \gamma I$. In fact, this equation means that $S(t) + E(t) + I(t)$ always increases when $S(t) + E(t) + I(t)$ gets close to 0.

In the proof of positively invariance we only need to consider the solutions starting $(0, 0, 0, Y(0))$, where $Y(0) \in [0, \Lambda/m]$, and the solutions starting on the boundary of Ω, i.e.,

$$\Omega_1 := \Omega \cap \{S = 0\}, \qquad \Omega_2 := \Omega \cap \{S \neq 0, E = 0\},$$
$$\Omega_3 := \Omega \cap \{SE \neq 0, I = 0\}, \qquad \Omega_4 := \Omega \cap \{SEI \neq 0, S + E + I = K\},$$
$$\Omega_5 := \Omega \cap \{SEI \neq 0, S + E + I \neq K, Y = 0\}, \Omega_6 := \Omega \cap \{SEI \neq 0, S + E + I \neq K, Y = \frac{\Lambda}{m}\}.$$

For the case that the initial value lies on Ω_1, we get

$$\dot{S} = \mu K + (b + d)I(0) + cE(0) > 0.$$

Additionally, if $I(0) = 0$,

$$\dot{I} = \varepsilon E(0) > 0.$$

If $I(0) \neq 0, Y(0) = 0$,

$$\dot{Y} = \beta_{h\nu} I(0)\Lambda/m > 0.$$

If $I(0) \neq 0, Y(0) = \Lambda/m$,

$$\dot{Y} = -\Lambda < 0.$$

If $I(0) \neq 0, E(0) = 0$, for $t \in (0, \epsilon)$ we get

$$E(t) = e^{-\xi t} \int_0^t e^{\xi\sigma} \left(\frac{\beta_{vh} Y(\sigma)}{1 + \alpha_{vh} Y(\sigma)} + \frac{\beta_{hh} I(\sigma)}{1 + \alpha_{hh} I(\sigma)} \right) S(\sigma) d\sigma > 0.$$

Thus, the orbit starting from Ω_1 goes into the inner of Ω. Similarly to this case, we can prove that the orbit starting from Ω_i $(i = 2, ..., 6)$ goes into the inner of Ω. By the statement above the definition of Ω, we actually know that the orbit starting from any point in the inner of Ω does not pass $(0, 0, 0, Y(0))$, where $Y(0) \in [0, \Lambda/m]$. The positively invariance of Ω is proved.

The basic reproduction number, denoted by R_0, is the expected number of secondary cases produced by a typical infected individual in a completely susceptible population during its entire period of infectiousness (see [8]). In the following, we use the method of next generation matrix (see [10]) to calculate R_0. Let $x := (S, E, I, Y)^{\mathsf{T}}$, we rewrite system (1.2) as $dx/dt = \mathcal{F}(x) - \mathcal{V}(x)$, where

$$\mathcal{F}(x) = \begin{pmatrix} 0 \\ \left(\frac{\beta_{vh} Y}{1+\alpha_{vh} Y} + \frac{\beta_{hh} I}{1+\alpha_{hh} I} \right) S \\ 0 \\ 0 \end{pmatrix},$$

$$\mathcal{V}(x) = \begin{pmatrix} -\mu(K - S) - cE - bI - dI + \left(\frac{\beta_{vh} Y}{1+\alpha_{vh} Y} + \frac{\beta_{hh} I}{1+\alpha_{hh} I} \right) S \\ \xi E \\ -\varepsilon E + \eta I \\ -\beta_{hv} \left(\frac{\Lambda}{m} - Y \right) I + mY \end{pmatrix}.$$

According to [10], we get

$$F := \left. \frac{\partial(\mathcal{F}_2(x), \mathcal{F}_3(x), \mathcal{F}_4(x))}{\partial(E, I, Y)} \right|_{S=K, E=0, I=0, Y=0} = \begin{pmatrix} 0 & \beta_{hh} K & \beta_{vh} K \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

$$V := \left. \frac{\partial(\mathcal{V}_2(x), \mathcal{V}_3(x), \mathcal{V}_4(x))}{\partial(E, I, Y)} \right|_{S=K, E=0, I=0, Y=0} = \begin{pmatrix} \xi & 0 & 0 \\ -\varepsilon & \eta & 0 \\ 0 & -\beta_{hv} \frac{\Lambda}{m} & m \end{pmatrix}.$$

Thus, the next generation matrix FV^{-1} defined in [10] is

$$\frac{1}{m\xi\eta}\cdot\begin{pmatrix} K\varepsilon\left(m\beta_{hh}+\beta_{hv}\beta_{vh}\frac{\Lambda}{m}\right) & K\xi\left(m\beta_{hh}+\beta_{hv}\beta_{vh}\frac{\Lambda}{m}\right) & K\beta_{vh}\xi\eta \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

from which we get the basic reproduction number

$$R_0 := \rho(FV^{-1}) = \frac{K\varepsilon(m^2\beta_{hh}+\beta_{hv}\beta_{vh}\Lambda)}{m^2\xi\eta}. \tag{2.1}$$

Here $\rho(\cdot)$ is the spectral radius function.

Theorem 2.1. *In Ω, the disease-free equilibrium $E_0 : (K, 0, 0, 0)$ is the unique equilibrium of system (1.2) when $R_0 \le 1$; System (1.2) has exactly two equilibria E_0 and*

$$E_c : \left(K - \frac{\varepsilon(\mu+\gamma)+\mu\eta}{\mu\varepsilon}I^*, \frac{\eta}{\varepsilon}I^*, I^*, \frac{\beta_{hv}\Lambda}{m^2+m\beta_{hv}I^*}I^*\right)$$

when $R_0 > 1$. Here

$$I^* = \frac{-B+\sqrt{B^2-4AC}}{2A}, \tag{2.2}$$

where

$$\begin{aligned} A =& \beta_{vh}\beta_{hv}\Lambda\alpha_{hh}(\mu\eta+\varepsilon\gamma+\varepsilon\mu)+\big(\beta_{hh}(\mu\eta+\varepsilon\gamma+\varepsilon\mu)+\mu\xi\eta\alpha_{hh}\big)(\Lambda\alpha_{vh}\beta_{hv}+m\beta_{hv}),\\ B =& (\mu\eta+\varepsilon\gamma+\varepsilon\mu)(\beta_{vh}\beta_{hv}\Lambda+m^2\beta_{hh})+\mu(\Lambda\alpha_{vh}\beta_{hv}+m\beta_{hv})(\xi\eta-K\varepsilon\beta_{hh})\\ &+\mu\alpha_{hh}(m^2\xi\eta-K\varepsilon\beta_{vh}\beta_{hv}\Lambda),\\ C =& \mu\xi\eta m^2(1-R_0). \end{aligned}$$

It is easy to check that $A > 0$ and $C < 0$ when $R_0 > 1$. So, I^* is positive. On the other hand, actually we need $\mu\varepsilon K - [\varepsilon(\mu+\gamma)+\mu\eta]I^*$ to be positive in Theorem 2.1 when $R_0 > 1$.

Proof. To get equilibria of system (1.2), we need to solve

$$\begin{cases} \mu K + dI + cE + bI - \mu S - \left(\dfrac{\beta_{vh}Y}{1+\alpha_{vh}Y}+\dfrac{\beta_{hh}I}{1+\alpha_{hh}I}\right)S = 0,\\ \left(\dfrac{\beta_{vh}Y}{1+\alpha_{vh}Y}+\dfrac{\beta_{hh}I}{1+\alpha_{hh}I}\right)S - \xi E = 0,\\ \varepsilon E - \eta I = 0,\\ \beta_{hv}\left(\dfrac{\Lambda}{m}-Y\right)I - mY = 0. \end{cases} \tag{2.3}$$

From (2.3),

$$S = K - \frac{\varepsilon(\mu+\gamma)+\mu\eta}{\mu\varepsilon}I, \quad E = \frac{\eta}{\varepsilon}I, \quad Y = \frac{\beta_{hv}\Lambda}{m^2+m\beta_{hv}I}I$$

and I satisfies $\left(AI^2+BI+C\right)I=0$, where A,B,C are expressed in the statement of this theorem. We observe that system (1.2) always has the disease-free equilibrium E_0.

Clearly, $A>0$ and it is easy to check that

$$\begin{aligned}B >&(\mu\eta+\varepsilon\gamma+\varepsilon\mu)(\beta_{vh}\beta_{hv}\Lambda+m^2\beta_{hh})+\mu(\Lambda\alpha_{vh}\beta_{hv}+m\beta_{hv})\xi\eta(1-R_0)\\&+\mu\alpha_{hh}m^2\xi\eta((1-R_0)\end{aligned}$$

because

$$\xi\eta-K\varepsilon\beta_{hh}>\xi\eta(1-R_0)$$

and $m^2\xi\eta-K\varepsilon\beta_{vh}\beta_{hv}\Lambda>m^2\xi\eta((1-R_0)$ by the expression of R_0 given in (2.1).

When $R_0<1$, we get $B>0$ and $C>0$. Thus, $AI^2+BI+C=0$ has no positive roots. When $R_0=1$, we get $B>0$ and $C=0$. Equation $AI^2+BI+C=0$ has nonzero root $I=-B/A$, which is negative. Thus, E_0 is the unique equilibrium of system (1.2) when $R_0\leq 1$. When $R_0>1$, we have $C<0$. Equation $AI^2+BI+C=0$ has a unique positive root I^*, given in (2.2). Thus, system (1.2) has exactly two equilibria E_0 and E_c, given in the statement of this theorem. □

3. The global stability of disease-free equilibrium E_0

In this section, we study the stability of the disease-free equilibrium E_0.

Theorem 3.1. *Equilibrium E_0 of system* (1.2) *is unstable if $R_0>1$.*

Proof. We compute the Jacobian matrix at E_0 and get

$$J(E_0)=\begin{pmatrix}-\mu & c & d+b-\beta_{hh}K & -\beta_{vh}K\\ 0 & -\xi & \beta_{hh}K & \beta_{vh}K\\ 0 & \varepsilon & -\eta & 0\\ 0 & 0 & \beta_{hv}\frac{\Lambda}{m} & -m\end{pmatrix}.$$

Thus the characteristic equation at the disease-free equilibrium E_0 is

$$\begin{vmatrix}\lambda+\mu & -c & \beta_{hh}K-d-b & \beta_{vh}K\\ 0 & \lambda+\xi & -\beta_{hh}K & -\beta_{vh}K\\ 0 & -\varepsilon & \lambda+\eta & 0\\ 0 & 0 & -\beta_{hv}\frac{\Lambda}{m} & \lambda+m\end{vmatrix}=0. \tag{3.1}$$

From (3.1) we obtain an eigenvalue $\lambda_1=-\mu$ and the other three eigenvalues $\lambda_2,\lambda_3,\lambda_4$ are roots of equation $\lambda^3+b_1\lambda^2+b_2\lambda+b_3=0$, where

$$\begin{aligned}&b_1:=\xi+\eta+m>0,\\&b_2:=\xi\eta+(\xi+\eta)m-\varepsilon\beta_{hh}K,\\&b_3:=m\xi\eta-\varepsilon K\left(\beta_{hh}m+\beta_{hv}\beta_{vh}\frac{\Lambda}{m}\right).\end{aligned}$$

It is easy to see that $b_3 < 0$ when $R_0 > 1$ by the expression of R_0. When $R_0 > 1$, we have $\lambda_2\lambda_3\lambda_4 = -b_3 > 0$, implying that at least one of eigenvalues is positive. Therefore, E_0 is unstable when $R_0 > 1$. □

Theorem 3.2. *Equilibrium E_0 of system (1.2) is globally asymptotically stable if $R_0 < 1$.*

Proof. From system (1.2), we have

$$\begin{cases} \dot{E} \le \beta_{vh} KY + \beta_{hh} KI - \xi E, \\ \dot{I} \le \varepsilon E - \eta I, \\ \dot{Y} \le \beta_{hv} \dfrac{\Lambda}{m} I - mY. \end{cases}$$

Consider the following comparison system

$$\begin{cases} \dot{Z}_1 = \beta_{hh} KZ_2 + \beta_{vh} KZ_3 - \xi Z_1, \\ \dot{Z}_2 = \varepsilon Z_1 - \eta Z_2, \\ \dot{Z}_3 = \beta_{hv} \dfrac{\Lambda}{m} Z_2 - mZ_3. \end{cases} \tag{3.2}$$

The coefficient matrix of (3.2) at $(0, 0, 0)$ is

$$\begin{pmatrix} -\xi & \beta_{hh} K & \beta_{vh} K \\ \varepsilon & -\eta & 0 \\ 0 & \beta_{hv} \frac{\Lambda}{m} & -m \end{pmatrix}. \tag{3.3}$$

The characteristic equation of (3.3) is $\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0$, where

$$\begin{aligned} c_1 &:= \xi + \eta + m > 0, \\ c_2 &:= \xi\eta + (\xi + \eta)m - \varepsilon\beta_{hh} K, \\ c_3 &:= m\xi\eta - \varepsilon K \left(\beta_{hh} m + \beta_{hv}\beta_{vh} \frac{\Lambda}{m} \right). \end{aligned}$$

When $R_0 < 1$, we get $m^2\xi\eta > \varepsilon K(\beta_{hh} m^2 + \beta_{hv}\beta_{vh}\Lambda)$, which implies that $c_2, c_3 > 0$. Thus, when $R_0 < 1$, we obtain $c_2, c_3 > 0$ and

$$\begin{aligned} c_1c_2 - c_3 &= (\xi + \eta + m)(\xi\eta + (\xi + \eta)m - \varepsilon\beta_{hh} K) - m\xi\eta - \varepsilon K \left(\beta_{hh} m + \beta_{hv}\beta_{vh} \frac{\Lambda}{m} \right) \\ &= (\xi + \eta)(\xi\eta + (\xi + \eta)m - \varepsilon\beta_{hh} K) + m^2(\xi + \eta) + \varepsilon K\beta_{hv}\beta_{vh} \frac{\Lambda}{m} > 0. \end{aligned}$$

By the Routh-Hurwitz theorem [13], all eigenvalues of (3.3) have negative real parts. Thus $(0, 0, 0)$ of the linear system (3.2) is globally asymptotically stable. Thus, for (3.2) solution $\big(Z_1(t), Z_2(t), Z_3(t)\big) \to (0, 0, 0)$ as $t \to +\infty$ when $R_0 < 1$, where $Z_1(0), Z_2(0), Z_3(0) > 0$. We claim that $Z_1(t) > 0$, for all $t \in (0, +\infty)$. In fact, if it is not true, then there exists $t_1 > 0$, such that

$$\begin{cases} Z_1(t_1) = 0, \\ \dot{Z}_1(t_1) \le 0, \\ Z_1(t) > 0 \quad \text{for all} \quad t \in (0, t_1). \end{cases} \tag{3.4}$$

For $t \in (0, t_1]$,

$$Z_2(t) = e^{-\eta t}\left(Z_2(0) + \int_0^t \varepsilon Z_1(s) e^{\eta s} ds\right) > 0, \tag{3.5}$$

$$Z_3(t) = e^{-mt}\left(Z_3(0) + \int_0^t \beta_{hv} \frac{\Lambda}{m} Z_2(s) e^{ms} ds\right) > 0. \tag{3.6}$$

Then

$$\dot{Z}_1(t_1) = \beta_{hh} K Z_2(t_1) + \beta_{vh} K Z_3(t_1) - \xi Z_1(t_1) > 0,$$

which contradicts $\dot{Z}_1(t_1) \leq 0$ given in (3.4). Therefore, this claim is correct. Further, by (3.5) and (3.6) we get $Z_2(t) > 0$ and $Z_3(t) > 0$ for all $t \in (0, +\infty)$.

By the Comparison Principle (see, e.g., [30, Theorem B.1]), we have $E(t) \leq Z_1(t), I(t) \leq Z_2(t), Y(t) \leq Z_3(t)$ for all $t \geq 0$ for solution $(E(t), I(t), Y(t))$ satisfying $E(0) \leq Z_1(0), I(0) \leq Z_2(0), Y(0) \leq Z_3(0)$. Hence, together with the positivity of the solution we conclude that $\big(E(t), I(t), Y(t)\big) \to (0, 0, 0)$ as $t \to +\infty$.

From the fourth equation in system (1.1) we get

$$R(t) = e^{-\mu t} R(0) + e^{-\mu t} \int_0^t \gamma I(s) e^{\mu s} ds. \tag{3.7}$$

If $\lim_{t\to+\infty} \int_0^t \gamma I(s) e^{\mu s} ds$ exists, then $R(t) \to 0$ as $t \to +\infty$. If $\lim_{t\to+\infty} \int_0^t \gamma I(s) e^{\mu s} ds$ does not exist, $\int_0^t \gamma I(s) e^{\mu s} ds \to +\infty$ as $t \to \infty$ and, hence,

$$\lim_{t\to+\infty} R(t) = \lim_{t\to+\infty} \gamma I(t)/\mu = 0. \tag{3.8}$$

Thus $R(t) \to 0$ as $t \to +\infty$ in any case. Duo to $S + E + I + R = K$, we obtain $S(t) \to K$ as $t \to +\infty$. Therefore, $\big(S(t), E(t), I(t), Y(t)\big) \to (K, 0, 0, 0)$ as $t \to +\infty$ when $R_0 < 1$. Finally, the globally asymptotically stability of E_0 follows from the globally asymptotically stability of $(0, 0, 0)$ of linear system (3.2). □

4. The global stability of endemic equilibrium E_c

In this section, we analyze the stability of the equilibrium E_c of system (1.2). To do this, we compute the Jacobian matrix $J(E_c)$ of the vector field of system (1.2) at E_c and obtain

$$J(E_c) = \begin{pmatrix} -\mu - \zeta & c & d + b - \frac{\beta_{hh} S^*}{(1+\alpha_{hh} I^*)^2} & -\frac{\beta_{vh} S^*}{(1+\alpha_{vh} Y^*)^2} \\ \zeta & -\xi & \frac{\beta_{hh} S^*}{(1+\alpha_{hh} I^*)^2} & \frac{\beta_{vh} S^*}{(1+\alpha_{vh} Y^*)^2} \\ 0 & \varepsilon & -\eta & 0 \\ 0 & 0 & \beta_{hv}\left(\frac{\Lambda}{m} - Y^*\right) & -\vartheta \end{pmatrix},$$

where (S^*, E^*, I^*, Y^*) denotes the coordinate of E_c given in Theorem 2.1 and

$$\zeta := \frac{\beta_{vh} Y^*}{1 + \alpha_{vh} Y^*} + \frac{\beta_{hh} I^*}{1 + \alpha_{hh} I^*}, \qquad \vartheta := \beta_{hv} I^* + m.$$

The corresponding characteristic equation is $\lambda^4+a_1\lambda^3+a_2\lambda^2+a_3\lambda+a_4=0$, where

$$\begin{aligned}
a_1 &:= \mu+\xi+\eta+\zeta+\vartheta,\\
a_2 &:= \mu(\xi+\eta)+(\mu+\xi+\eta)\vartheta+(\varepsilon+\mu+\eta+\vartheta)\zeta+\xi\eta-\frac{\varepsilon\beta_{hh}S^*}{(1+\alpha_{hh}I^*)^2},\\
a_3 &:= \mu\left(\xi\eta-\frac{\beta_{hh}S^*}{(1+\alpha_{hh}I^*)^2}\right)+\mu(\xi+\eta)\vartheta+(\varepsilon\mu+\varepsilon\gamma+\mu\eta)\zeta+(\varepsilon+\mu+\eta)\zeta\vartheta\\
&\quad+\vartheta\left(\xi\eta-\frac{\varepsilon\beta_{hh}S^*}{(1+\alpha_{hh}I^*)^2}-\frac{\varepsilon\beta_{hv}\beta_{vh}S^*\Lambda}{m\vartheta(1+\alpha_{vh}Y^*)^2}\right)+\frac{\varepsilon\beta_{hv}\beta_{vh}S^*Y^*}{\vartheta(1+\alpha_{vh}Y^*)^2},\\
a_4 &:= \mu\vartheta\left(\xi\eta-\frac{\varepsilon\beta_{hh}S^*}{(1+\alpha_{hh}I^*)^2}-\frac{\varepsilon\beta_{hv}\beta_{vh}S^*\Lambda}{m\vartheta(1+\alpha_{vh}Y^*)^2}\right)+\mu\frac{\varepsilon\beta_{hv}\beta_{vh}S^*Y^*}{\vartheta(1+\alpha_{vh}Y^*)^2}\\
&\quad+(\varepsilon\mu+\varepsilon\gamma+\mu\eta)\zeta\vartheta.
\end{aligned}\tag{4.1}$$

Define

$$\Upsilon := a_3(a_1a_2-a_3)-a_1^2a_4. \tag{4.2}$$

Theorem 4.1. *If $R_0>1$, equilibrium E_c of system (1.2) is locally asymptotically stable if and only if $\Upsilon>0$, where Υ is defined in (4.2).*

Proof. In order to prove this theorem, we only need to prove that all eigenvalues of the characteristic equation $\lambda^4+a_1\lambda^3+a_2\lambda^2+a_3\lambda+a_4=0$ have negative real parts, where a_i $(i=1,...,4)$ are given in (4.1). So, by the Routh-Hurwitz Theorem we need to prove

$$a_1>0,\begin{vmatrix} a_1 & a_3\\ 1 & a_2\end{vmatrix}>0,\begin{vmatrix} a_1 & a_3 & 0\\ 1 & a_2 & a_4\\ 0 & a_1 & a_3\end{vmatrix}>0,\begin{vmatrix} a_1 & a_3 & 0 & 0\\ 1 & a_2 & a_4 & 0\\ 0 & a_1 & a_3 & 0\\ 0 & 1 & a_2 & a_4\end{vmatrix}>0.$$

Straight computation shows that it is equivalent to prove $a_1,a_2,a_3,a_4>0$ and $a_3(a_1a_2-a_3)-a_1^2a_4>0$. Since Υ is defined as $a_3(a_1a_2-a_3)-a_1^2a_4$ in (4.2) and is required as $\Upsilon>0$ in the statement of Theorem (4.1), we only need to prove that $a_1,a_2,a_3,a_4>0$.

Let

$$\begin{aligned}
G &:= \xi\eta-\frac{\varepsilon\beta_{hh}S^*}{(1+\alpha_{hh}I^*)^2},\qquad \Theta := \frac{\varepsilon\beta_{hv}\beta_{vh}S^*Y^*}{(1+\alpha_{vh}Y^*)^2},\\
H &:= \xi\eta-\frac{\varepsilon\beta_{hh}S^*}{(1+\alpha_{hh}I^*)^2}-\frac{\varepsilon\beta_{hv}\beta_{vh}S^*\Lambda}{m\vartheta(1+\alpha_{vh}Y^*)^2}.
\end{aligned}$$

Since E_c is an equilibrium, by (2.3) we get

$$\left(\frac{\beta_{vh}Y^*}{1+\alpha_{vh}Y^*}+\frac{\beta_{hh}I^*}{1+\alpha_{hh}I^*}\right)S^*-\xi E^*=0,\qquad \varepsilon E^*-\eta I^*=0.$$

Then

$$\frac{\varepsilon\beta_{hh}S^*}{1+\alpha_{hh}I^*}+\frac{\varepsilon\beta_{hv}\beta_{vh}S^*\Lambda}{m\vartheta(1+\alpha_{vh}Y^*)}=\xi\eta,$$

implying $G > 0, H > 0$. Thus, by the expressions of $a_1, ..., a_4$ given in (4.1) we get

$$\begin{aligned}
a_1 &= \mu + \xi + \eta + \zeta + \vartheta > 0,\\
a_2 &= \mu(\xi + \eta) + (\mu + \xi + \eta)\vartheta + (\varepsilon + \mu + \eta + \vartheta)\zeta + G > 0,\\
a_3 &= \mu G + \mu(\xi + \eta)\vartheta + (\varepsilon\mu + \varepsilon\gamma + \mu\eta)\zeta + (\varepsilon + \mu + \eta)\zeta\vartheta + \vartheta H + \Theta > 0,\\
a_4 &= \mu\vartheta H + \mu\Theta + (\varepsilon\mu + \varepsilon\gamma + \mu\eta)\zeta\vartheta > 0.
\end{aligned}$$

The proof is finished. □

In the following, using the geometric approach given in [11,17,20] we discuss the global stability of E_c. As defined in [3], system (1.2) is *uniformly persistent* in $\mathring{\Omega}$ if there exists a constant $\hat{\rho} > 0$ such that any solution $\big(S(t), E(t), I(t), Y(t)\big)$ of (1.2) with the initial value $\big(S(0), E(0), I(0), Y(0)\big) \in \mathring{\Omega}$ satisfies

$$\min\left\{\liminf_{t\to+\infty} S(t), \liminf_{t\to+\infty} E(t), \liminf_{t\to+\infty} I(t), \liminf_{t\to+\infty} Y(t)\right\} \geq \hat{\rho}, \tag{4.3}$$

where $\mathring{\Omega}$ is the inner of Ω. Note that this definition is called *uniformly strongly persistent* in [32].

Lemma 4.1. *System* (1.2) *is uniformly persistent in* $\mathring{\Omega}$ *when* $R_0 > 1$.

Proof. Since Ω is bounded and positively invariant, system (1.2) is point dissipative in Ω (see [15]). Thus, there exists a compact set M_0 such that all solutions of system (1.2) initiated in Ω ultimately goes into M_0. So conditions $(C_{4.2})$ of [32, Theorem 4.6] hold for M_0.

Let $\omega(x_0)$ be the ω-limit set of the orbit starting from $x_0 \in \mathring{\Omega}$ and

$$\Xi_1 := \{x_* \in \partial\Omega |\ \ x(t, x_*) \in \partial\Omega, \forall t > 0\}, \qquad \Xi_2 := \bigcup_{x_*\in\Xi_1} \omega(x_*),$$

where $x(t, x_*) = (S(t), E(t), I(t), Y(t))$ is the solution of system (1.2). Clearly, $\Xi_1, \Xi_2 \subseteq \partial\Omega$. By the expression of system (1.2), all solutions starting in $\partial\Omega$ but not on the S-axis ultimately goes into $\mathring{\Omega}$. Thus $\Xi_1 = \{(S, 0, 0, 0) |\ \ 0 \leq S \leq K\}$ and $\Xi_2 = \{E_0\}$, because S-axis is a 1-dimensional stable sub-manifold of E_0. In order to prove that system (1.2) is uniformly persistent in $\mathring{\Omega}$, by [32, Theorem 4.6] it suffices to prove that E_0 is a weak repeller (see [32, p408]) for $\mathring{\Omega}$, i.e., every solution starting from $x_0 \in \mathring{\Omega}$ satisfies

$$\limsup_{t\to+\infty} d\left(x(t, x_0), E_0\right) > 0. \tag{4.4}$$

In fact, if $W^s(E_0)\bigcap\mathring{\Omega} \neq \emptyset$, there exists a solution $\big(\tilde{S}(t), \tilde{E}(t), \tilde{I}(t), \tilde{Y}(t)\big)$ with the initial value $\big(\tilde{S}(0), \tilde{E}(0), \tilde{I}(0), \tilde{Y}(0)\big)$ in $\mathring{\Omega}$ such that $\big(\tilde{S}(t), \tilde{E}(t), \tilde{I}(t), \tilde{Y}(t)\big) \to E_0$ as $t \to +\infty$, where $W^s(E_0)$ denotes the stable manifold of E_0. Define a continuous function

$$g(\nu) := \varepsilon(K - \nu)\left(\frac{\beta_{hh}m}{1 + \alpha_{vh}\nu} + \frac{\beta_{hv}\beta_{vh}\Big(\Lambda - m\nu\Big)}{m(1 + \alpha_{hh}\nu)}\right) - m\xi\eta$$

for $\nu \geq 0$. Clearly, $g(0) = (R_0 - 1)m\xi\eta > 0$ because $R_0 > 1$. Hence there exists a sufficiently small $\hat{\nu} \in (0, K)$ such that $g(\nu) > 0$ for all $\nu \in [0, \hat{\nu}]$. On the other hand, there exists a $T > 0$ such that for all $t \geq T$

$$K - \hat{\nu} \leq \tilde{S}(t) \leq K + \hat{\nu}, \quad 0 \leq \tilde{E}(t) \leq \hat{\nu}, \quad 0 \leq \tilde{I}(t) \leq \hat{\nu}, \quad 0 \leq \tilde{Y}(t) \leq \hat{\nu}.$$

We claim that there exists a $\hat{T} > T$ such that $\tilde{E}(\hat{T})\tilde{I}(\hat{T})\tilde{Y}(\hat{T}) \neq 0$. Otherwise, either $\tilde{E}(t) \equiv 0$ or $\tilde{I}(t) \equiv 0$ or $\tilde{Y}(t) \equiv 0$ for all $t \geq T$, which implies $\tilde{E}(t) \equiv \tilde{I}(t) \equiv \tilde{Y}(t) \equiv 0$ by the expression of (1.2). This contradicts that $\big(\tilde{S}(0), \tilde{E}(0), \tilde{I}(0), \tilde{Y}(0)\big)$ in $\mathring{\Omega}$. So there exists a $\hat{T} > T$ such that $\tilde{E}(\hat{T})\tilde{I}(\hat{T})\tilde{Y}(\hat{T}) \neq 0$. For $t \geq \hat{T}$ we get

$$\begin{cases} \dot{\tilde{E}} \geq \left(\dfrac{\beta_{vh}\tilde{Y}}{1+\alpha_{vh}\hat{\nu}} + \dfrac{\beta_{hh}\tilde{I}}{1+\alpha_{hh}\hat{\nu}} \right)(K-\hat{\nu}) - \xi\tilde{E}, \\ \dot{\tilde{I}} \geq \varepsilon\tilde{E} - \eta\tilde{I}, \\ \dot{\tilde{Y}} \geq \beta_{hv}\left(\dfrac{\Lambda}{m} - \hat{\nu}\right)\tilde{I} - m\tilde{Y}. \end{cases}$$

At $(0,0,0)$, linear system

$$\begin{cases} \dot{E} = \left(\dfrac{\beta_{vh}Y}{1+\alpha_{vh}\hat{\nu}} + \dfrac{\beta_{hh}I}{1+\alpha_{hh}\hat{\nu}} \right)(K-\hat{\nu}) - \xi E, \\ \dot{I} = \varepsilon E - \eta I, \\ \dot{Y} = \beta_{hv}\left(\dfrac{\Lambda}{m} - \hat{\nu}\right) I - mY \end{cases} \tag{4.5}$$

has Jacobian matrix

$$\hat{A} = \begin{pmatrix} -\xi & \frac{\beta_{hh}(K-\hat{\nu})}{1+\alpha_{hh}\hat{\nu}} & \frac{\beta_{vh}(K-\hat{\nu})}{1+\alpha_{vh}\hat{\nu}} \\ \varepsilon & -\eta & 0 \\ 0 & \beta_{hv}\left(\frac{\Lambda}{m} - \hat{\nu}\right) & -m \end{pmatrix}.$$

Let $\hat{\lambda}$ be the maximum eigenvalue of $\hat{A}$. It is not hard to prove $\hat{\lambda} > 0$ by the expression of $\hat{A}$ and $g(\hat{\nu}) > 0$. Since all off-diagonal elements in $\hat{A}$ are nonnegative, by the Perron-Frobenius Theorem (see [33]) there is a positive eigenvector $\hat{n} = (\hat{n}_1, \hat{n}_2, \hat{n}_3)$ corresponding to $\hat{\lambda}$ and $|\hat{n}| = 1$. There exists a positive l such that $l\hat{n}_1 < \tilde{E}(\hat{T}), l\hat{n}_2 < \tilde{I}(\hat{T}), l\hat{n}_3 < \tilde{Y}(\hat{T})$. Moreover, the solution $(E(t), I(t), Y(t))$ with initial value $(E(\hat{T}), I(\hat{T}), Y(\hat{T})) = (l\hat{n}_1, l\hat{n}_2, l\hat{n}_3)$ of linear system (4.5) goes to positive infinity as $t \to +\infty$. By the comparison principle (see [25]) we get $\tilde{E}(t) > E(t), \tilde{I}(t) > I(t), \tilde{Y}(t) > Y(t)$. Hence, $\tilde{E}(t) \to +\infty, \tilde{I}(t) \to +\infty, \tilde{Y}(t) \to +\infty$ as $t \to +\infty$. This contradicts $\tilde{E}(t) \to 0, \tilde{I}(t) \to 0, \tilde{Y}(t) \to 0$ as $t \to +\infty$. Then we get

$$W^s(E_0) \bigcap \mathring{\Omega} = \emptyset. \tag{4.6}$$

If (4.4) does not hold for some solution $x(t, x_0)$, then $\limsup_{t\to+\infty} d\big(x(t,x_0), E_0\big) = 0$, implying $\lim_{t\to+\infty} x(t,x_0) = E_0$. This contradicts (4.6). Therefore, (4.4) holds for every solution starting from $x_0 \in \mathring{\Omega}$. By [32, Theorem 4.6], system (1.2) is uniformly persistent in $\mathring{\Omega}$ when $R_0 > 1$. □

Having Lemma 4.1, (4.3) holds for some $\hat{\rho}$. It is clear that such $\hat{\rho}$ is less than $\min\{K, \Lambda/m\}$ by the definition of Ω. Let ρ be the maximum of all values of $\hat{\rho}$.

Theorem 4.2. *If $R_0 > 1$, equilibrium E_c of system (1.2) is globally asymptotically stable when*

$$\mu > \max\left\{ \frac{2\beta_{vh}K\Lambda}{m\rho(1+\alpha_{vh}\rho)^2}, \frac{2\beta_{hh}K}{(1+\alpha_{hh}\rho)^2} - 2(b+d) - \gamma \right\}. \tag{4.7}$$

Proof. Let

$$\Gamma := \{(S,E,I,Y) \in \Omega |\ \rho \le S \le K, \rho \le E \le K, \rho \le I \le K, \rho \le Y \le \Lambda/m\}.$$

Clearly, Γ is compact and absorbing because Ω is positively invariant. For system (1.2), define

$$P(t) := \begin{pmatrix} \frac{1}{E(t)} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{I(t)} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{I(t)} & 0 & 0 \\ 0 & 0 & \frac{1}{Y(t)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{Y(t)} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{1}{Y(t)} \end{pmatrix}$$

for $t \ge 0$, where $(S(t), E(t), I(t), Y(t))$ is the solution of system (1.2) with the initial value $(S(0), E(0), I(0), Y(0)) \in \Gamma$. It is not hard to check that $P(t)$ is invertible and then we define

$$Q(t) := \frac{dP(t)}{dt} P^{-1}(t) + P(t) J^{[2]}(t) P^{-1}(t),$$

where

$$J(t) := \begin{pmatrix} -\mu - q_4 & c & d+b-q_1 & -q_2 \\ q_4 & -\xi & q_1 & q_2 \\ 0 & \varepsilon & -\eta & 0 \\ 0 & 0 & q_3 & -\beta_{hv} I - m \end{pmatrix}$$

is the Jacobian matrix of (1.2), $J^{[2]}(t)$ is its second additive compound matrix (see [19]) and

$$q_1 = \frac{\beta_{hh} S}{(1+\alpha_{hh} I)^2},\quad q_2 = \frac{\beta_{vh} S}{(1+\alpha_{vh} Y)^2},\quad q_3 = \beta_{hv}\left(\frac{\Lambda}{m} - Y\right),\quad q_4 = \frac{\beta_{vh} Y}{1+\alpha_{vh} Y} + \frac{\beta_{hh} I}{1+\alpha_{hh} I}.$$

By straight computations, we write $Q(t)$ as

$$Q(t) = \begin{pmatrix} Q_{11} & Q_{12} & Q_{13} \\ Q_{21} & Q_{22} & Q_{23} \\ Q_{31} & Q_{32} & Q_{33} \end{pmatrix},$$

where

$$Q_{11} = M_{11} - \frac{\dot{E}}{E},\quad Q_{12} = \left(\tfrac{I}{E} q_1, \tfrac{I}{E}(q_1 - b - d)\right),\quad Q_{13} = \left(\tfrac{Y}{E} q_2, \tfrac{Y}{E} q_2, 0\right).$$

$$Q_{21} = \begin{pmatrix} \varepsilon \\ 0 \end{pmatrix},\qquad Q_{22} = \begin{pmatrix} M_{22} - \frac{\dot{I}}{I} & c \\ q_4 & M_{44} - \frac{\dot{I}}{I} \end{pmatrix},\qquad Q_{23} = \begin{pmatrix} 0 & 0 & \frac{Y}{I} q_2 \\ 0 & 0 & -\frac{Y}{I} q_2 \end{pmatrix}.$$

$$Q_{31} = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix},\qquad Q_{32} = \begin{pmatrix} \frac{I}{Y} q_3 & 0 \\ 0 & \frac{I}{Y} q_3 \\ 0 & 0 \end{pmatrix},\qquad Q_{33} = \begin{pmatrix} M_{33} - \frac{\dot{Y}}{Y} & c & b+d-q_1 \\ q_4 & M_{55} - \frac{\dot{Y}}{Y} & q_1 \\ 0 & \varepsilon & M_{66} - \frac{\dot{Y}}{Y} \end{pmatrix}$$

and

$$M_{11}=-\mu-q_4-\xi,\qquad M_{22}=-\mu-q_4-\eta,\qquad M_{33}=-\mu-q_4-\beta_{vh}I-m,$$
$$M_{44}=-\xi-\eta,\qquad M_{55}=-\xi-\beta_{hv}I-m,\qquad M_{66}=-\eta-\beta_{hv}I-m.$$

For $z:=(z_1,z_2,z_3,z_4,z_5,z_6)\in\mathbb{R}^6$, we use $\max\{|z_1|,|z_2|+|z_3|,|z_4|+|z_5|+|z_6|\}$ as its norm $|z|$. Let $\sigma(Q(t))$ be the *Lozinskiĭ* measure (see [5]) with respect to this norm. As described in [26], for given $t\geq 0$ we get $\sigma\big(Q(t)\big)\leq\max\{\mathrm{g}_1(t),\mathrm{g}_2(t),\mathrm{g}_3(t)\}$, where

$$\mathrm{g}_i(t):=\sigma_1(Q_{ii})+\sum_{j\in\{1,2,3\}\setminus\{i\}}|Q_{ij}|,\qquad i=1,2,3,$$

and σ_1 denotes the *Lozinskiĭ* measure with respect to the l_1 norm (see [20]), $|Q_{ij}|(i,j=1,2,3)$ are matrix norms with respect to the l_1 norm. Further computations show that

$$\mathrm{g}_1(t)<-\mu,\quad \mathrm{g}_2(t)<-\mu+2\frac{\beta_{vh}\Lambda K}{m\rho(1+\alpha_{vh}\rho)^2},\quad \mathrm{g}_3(t)<-\mu+\omega,$$

where $\omega=\max\big\{0,2\beta_{hh}K/(1+\alpha_{hh}\rho)^2-2(b+d)-\gamma\big\}$.

Let

$$\bar{b}:=\min\left\{\mu,\mu-\frac{2\beta_{vh}\Lambda K}{m\rho(1+\alpha_{vh}\rho)^2},\mu-\omega\right\}.$$

By (4.7), we have $\bar{b}>0$. Thus for $t\geq 0$

$$\mathrm{g}_1(t)<-\bar{b},\qquad \mathrm{g}_2(t)<-\bar{b},\qquad \mathrm{g}_3(t)<-\bar{b}.$$

Therefore, for $t\geq 0$

$$\frac{1}{t}\int_0^t\sigma(Q(s))ds\leq\frac{1}{t}\int_0^t-\bar{b}ds=-\bar{b}.$$

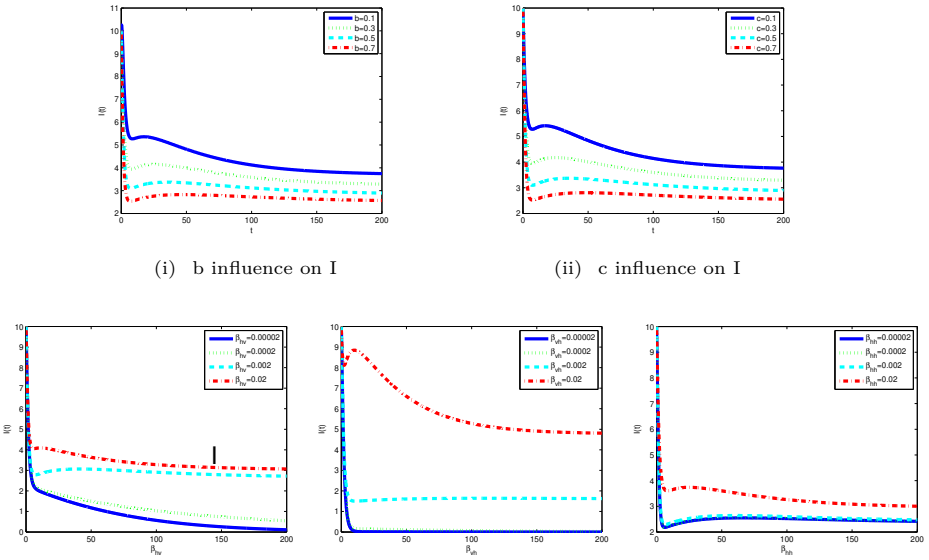
It follows that

$$\limsup_{t\to+\infty}\sup_{(S(0),E(0),I(0),Y(0))\in\Gamma}\frac{1}{t}\int_0^t\sigma(Q(s))ds\leq-\bar{b}<0.$$

By [20, Theorem 3.5], equilibrium E_c of system(1.2) is globally asymptotically stable. □

5. Numerical simulations and conclusion remarks

In this section, we perform some numerical simulations to support the analytic results presented in the above sections. We first give the analysis of infection number I affected by some parameters. As shown in Fig. 1(i), (ii), the number of infected hosts I decreases effectively as the rates b, c of the infectious or exposed individual becoming susceptible individual without treatment increases. As shown in Fig. 1(iii), (iv), (v), the number of infected individual I decreases as infection ratios $\beta_{hv},\beta_{vh},\beta_{hh}$ decrease. Moreover, β_{hv},β_{vh} have a greater influence on I than β_{hh}.



(i) b influence on I (ii) c influence on I

(iii) β_{hv} influence on I (iv) β_{vh} influence on I (v) β_{hh} influence on I

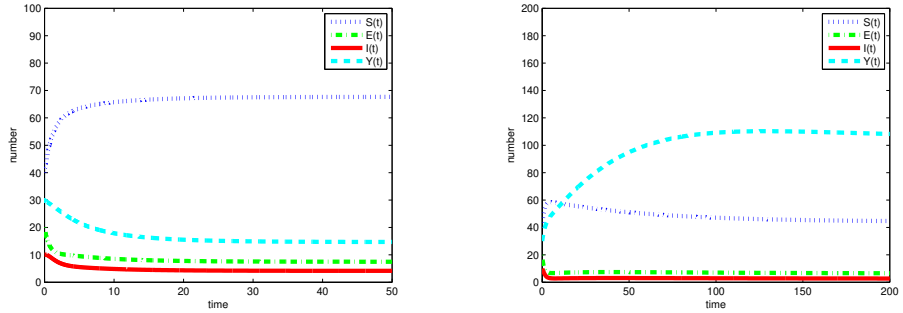
Figure 1. Change of I

Thus, strategies such as drug control and biological control that either decrease biting rates β_{hv}, β_{vh} or increase disease resistance b, c could be recommended.

Taking $K = 80, \Lambda = 10, \beta_{hv} = 0.02, \beta_{vh} = 0.01, \beta_{hh} = 0.005, \alpha_{hv} = 0.02, \alpha_{vh} = 0.02, \alpha_{hh} = 0.02, c = 0.1, d = 0.1, \mu = 0.6, \varepsilon = 0.5, m = 0.2, b = 0.1, \gamma = 0.1$ and initial condition $(S(0) = 40, E(0) = 20, I(0) = 10, Y(0) = 30)$, we get $R_0 = 2.2564 > 1$ and find that condition (4.7) holds. E_c is globally asymptotically stable as shown in Fig. 2(i). However, similar as in [1, 18, 24, 35] we take $K = 80, \Lambda = 10, \beta_{hv} = 0.002, \beta_{vh} = 0.004, \beta_{hh} = 0.01, \alpha_{hv} = 0.02, \alpha_{vh} = 0.02, \alpha_{hh} = 0.01, c = 0.6, d = 0.6, \mu = 0.01, \varepsilon = 0.5, m = 0.02, b = 0.5, \gamma = 0.1$ and initial condition $(S(0) = 40, E(0) = 20, I(0) = 10, Y(0) = 30)$. Then $R_0 = 6.2542 > 1$ but condition (4.7) does not hold. We observe in Fig. 2(ii) that E_c is still globally asymptotically stable. This means that condition (4.7) in Theorem 4.2 is just sufficient for the globally asymptotically stability of E_c but not necessary.

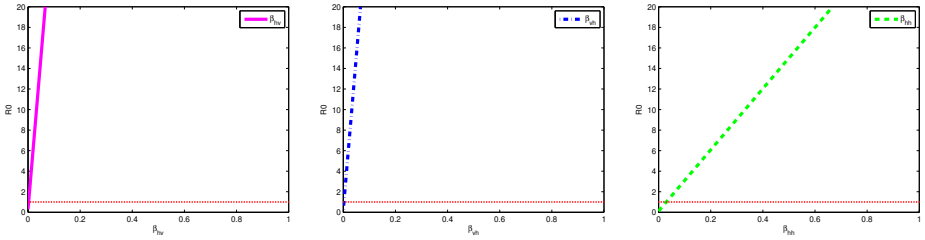
From the expression of (2.1) we can see that R_0 is a strictly increasing function with respect to parameters β_{hv}, β_{vh} and β_{hh}, and a strictly decreasing function with respect to parameters b and c. We present some figures to show how the basic reproduction number R_0 changes in terms of various values of contact rates. In Fig. 3 we observe that as the value of the parameters β_{hv}, β_{vh} and β_{hh} increases, the value of R_0 rapidly exceeds 1. Further, the transmission of disease from host to vector or vector to host plays a more important role than that from host to host because of the slopes shown in Fig. 3.

In Fig. 4(i) we observe that R_0 decreases as b and c increase, whereas the value of R_0 is always greater than 1. Thus, the single increasing of plant disease resistance is insufficient for the complete control of disease. However, the increasing of plant disease resistance can lower R_0 to be less than 1 if at the same we reduce the contact rate between infected plants and susceptible vectors as shown in Fig. 4(ii).



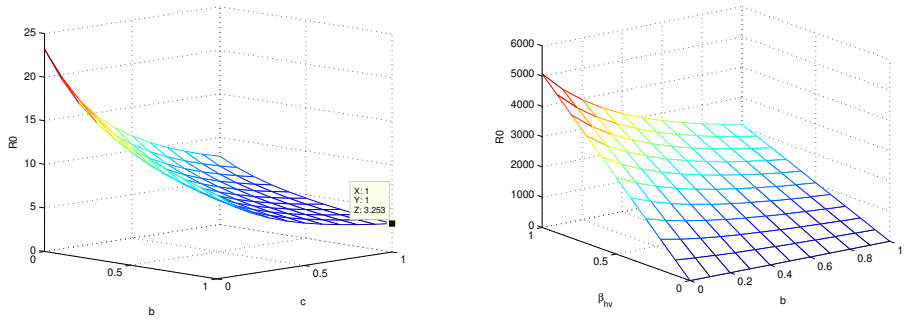
(i) $R_0 > 1$, condition (4.7) is satisfied (ii) $R_0 > 1$, condition (4.7) is not satisfied

Figure 2. Stability of endemic equilibrium E_c



(i) β_{hv} (ii) β_{vh} (iii) β_{hh}

Figure 3. The dependence of R_0 on infection rates



(i) The dependence of R_0 on b and c (ii) The dependence of R_0 on b and β_{hv}

Figure 4. Change of R_0

As an conclusion, we propose a vector-borne plant epidemic mathematical model with disease resistance. We calculate the basic reproduction number R_0, which is important for the dynamics, and investigate the existence and global stability of equilibria. By our main results, the system has a unique equilibrium E_0 when $R_0 < 1$, which is disease-free equilibrium and globally asymptotically stable. It implies that the disease dies out eventually. When $R_0 > 1$, the system has a

unique endemic equilibrium E_c, which is globally stable under some conditions. It implies that the disease will persist. As we know from technology, global stability of equilibria is important and difficult for many biological models. In our paper we obtain main results by generalizing the geometric approach given in [20] to higher dimensional systems. Higher dimensions cause greater difficulties in calculations such as the construction of matrix function $P(t)$ and the division of matrix function $Q(t)$.

We have to point out that system (1.1) may be more complicated and higher-dimensional if we consider WFT to have 6 development stages: egg-larvae1-larvae2-prepupae-pupae-adult as in [18]. Then its dynamical analysis will become more difficult.

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