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 Lozinski\'i 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 TSWV 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
\[
\begin{align*}
\dot{S} &= f(I) + cE + bI - \mu S - \left( \frac{\beta_{vh}Y}{1 + \alpha_{vh}Y} + \frac{\beta_{hh}I}{1 + \alpha_{hh}I} \right) S, \\
\dot{E} &= \left( \frac{\beta_{vh}Y}{1 + \alpha_{vh}Y} + \frac{\beta_{hh}I}{1 + \alpha_{hh}I} \right) S - (\varepsilon + \varepsilon + \mu)E, \\
\dot{I} &= \varepsilon E - (b + \mu + \gamma + d)I, \\
\dot{R} &= \gamma I - \mu R, \\
\dot{X} &= \Lambda - \beta_{hv} XI - mX, \\
\dot{Y} &= \beta_{hv} XI - mY,
\end{align*}
\]

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 $\mu K$ is the natural deaths and $dI$ is the infected deaths. So $f(I) := \mu K + dI$. Here parameters $\beta_{hv}, \beta_{vh}, \ldots, c, d$ are all nonnegative constants and their biological meanings are given in Table 1.

| Parameter | Description | value |
|-----------|-------------|-------|
| $\beta_{hv}$ | infection ratio between infected hosts and susceptible vectors | fitting |
| $\beta_{vh}$ | biting rate of an infected vector on the susceptible hosts | fitting |
| $\beta_{hh}$ | infection incidence between infected and susceptible hosts | fitting |
| $\alpha_{vh}$ | determines the degree of infection saturation between the vector and the host | fitting |
| $\alpha_{hh}$ | determines the degree of infection saturation between hosts | fitting |
| $\gamma$ | the conversion rate of infected hosts to recovered hosts | fitting |
| $\mu$ | natural death rate of hosts | $[18]$ |
| $\Lambda$ | birth or immigration of insect vectors | $[18]$ |
| $\varepsilon$ | 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 $\Lambda/m$, 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
\[
\begin{align*}
\dot{S} &= \mu(K - S) + (b + d)I + cE - \left(\frac{\beta_{vh}Y}{1 + \alpha_{vh}Y} + \frac{\beta_{hh}I}{1 + \alpha_{hh}I}\right)S, \\
\dot{E} &= \left(\frac{\beta_{vh}Y}{1 + \alpha_{vh}Y} + \frac{\beta_{hh}I}{1 + \alpha_{hh}I}\right)S - \xi E, \\
\dot{I} &= \varepsilon E - \eta I, \\
\dot{Y} &= \beta_{hv} \left(\frac{\Lambda}{m} - Y\right)I - mY,
\end{align*}
\]  
(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 $\Omega$, 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 $\Omega$ 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 $\Omega$, 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 $\Omega$ and existence of equilibria

In this section we first prove the positively invariance of $\Omega$ 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 $\Omega$, i.e.,
\[
\begin{align*}
\Omega_1 := \Omega \cap \{S = 0\}, & & \Omega_2 := \Omega \cap \{S \neq 0, E = 0\}, \\
\Omega_3 := \Omega \cap \{SE \neq 0, I = 0\}, & & \Omega_4 := \Omega \cap \{SEI \neq 0, S + E + I = K\}, \\
\Omega_5 := \Omega \cap \{SEI \neq 0, S + E + I \neq K, Y = 0\}, & & \Omega_6 := \Omega \cap \{SEI \neq 0, S + E + I \neq K, Y = \frac{\Lambda}{m}\}.
\end{align*}
\]

For the case that the initial value lies on $\Omega_1$, 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 \( I(0) \neq 0 \), \( Y(0) = 0 \),
\[
\dot{Y} = \beta_{hv} I(0) \Lambda/m > 0.
\]

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 \( \Omega_1 \) goes into the inner of \( \Omega \). Similarly to this case, we can prove that the orbit starting from \( \Omega_i \) \( (i = 2, \ldots, 6) \) goes into the inner of \( \Omega \). By the statement above the definition of \( \Omega \), we actually know that the orbit starting from any point in the inner of \( \Omega \) does not pass \((0,0,0,Y(0))\), where \( Y(0) \in [0, \Lambda/m] \). The positively invariance of \( \Omega \) 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 \( x := (S,E,I,Y)^T \), we rewrite system (1.2) as \( dx/dt = F(x) - V(x) \), where

\[
F(x) = \begin{pmatrix}
0 \\
\frac{\beta_{vh} Y}{1 + \alpha_{vh} Y} + \frac{\beta_{hh} I}{1 + \alpha_{hh} I} & 0 \\
0 & 0 \\
0 & 0
\end{pmatrix} S,
\]

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

According to \([10]\), we get

\[
F := \frac{\partial (F_2(x), F_3(x), F_4(x))}{\partial (E, I, Y)} \bigg|_{S=K,E=0,I=0,Y=0} = \begin{pmatrix}
0 & \beta_{hh} K & \beta_{vh} K \\
0 & 0 & 0 \\
0 & 0 & 0
\end{pmatrix},
\]

\[
V := \frac{\partial (V_2(x), V_3(x), V_4(x))}{\partial (E, I, Y)} \bigg|_{S=K,E=0,I=0,Y=0} = \begin{pmatrix}
\xi & 0 & 0 \\
-\varepsilon & \eta & 0 \\
0 & -\beta_{hv} \frac{\Lambda}{m} m
\end{pmatrix}.
\]
Thus, the next generation matrix \( F V^{-1} \) defined in [10] is

\[
\frac{1}{m\xi\eta} \begin{pmatrix}
K\varepsilon \left( m\beta_{hh} + \beta_{hv}\beta_{ch}\frac{\lambda}{m} \right) & K\xi \left( m\beta_{hh} + \beta_{hv}\beta_{ch}\frac{\lambda}{m} \right) & \beta_{ch}\xi\eta \\
0 & 0 & 0 \\
0 & 0 & 0
\end{pmatrix},
\]

from which we get the basic reproduction number

\[
R_0 := \rho(FV^{-1}) = \frac{K\varepsilon m^2\beta_{hh} + \beta_{hv}\beta_{ch}\Lambda}{m^2}\frac{\mu\xi\eta}{\mu\varepsilon + \mu\eta + \mu\varepsilon + \mu\xi\eta}\frac{\mu\xi\eta}{\mu\varepsilon + \mu\eta + \mu\varepsilon + \mu\xi\eta}.
\]

(2.1)

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

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

\[
I^* = \frac{-B + \sqrt{B^2 - 4AC}}{2A},
\]

(2.2)

where

\[
A = \beta_{ch}\beta_{hv}\Lambda\alpha_{hh}(\mu\eta + \varepsilon\gamma + \varepsilon\mu) + (\beta_{hh}(\mu\eta + \varepsilon\gamma + \varepsilon\mu) + \mu\xi\eta\alpha_{hh})(\Lambda\alpha_{eh}\beta_{hv} + m\beta_{hv}),
\]

\[
B = (\mu\eta + \varepsilon\gamma + \varepsilon\mu)(\beta_{vh}\beta_{hv}\Lambda + m^2\beta_{hh}) + \mu(\Lambda\alpha_{eh}\beta_{hv} + m\beta_{hv})(\xi\eta - K\varepsilon\beta_{hh})
\]

\[
+ \mu\alpha_{hh}(m^2\xi\eta - K\varepsilon\beta_{vh}\beta_{hv}\Lambda),
\]

\[
C = \mu\xi\eta m^2(1 - R_0).
\]

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

\[
\begin{cases}
\mu K + dI + bE + bI - \mu S - \left( \frac{\beta_{ch}Y}{1 + \alpha_{eh}Y} + \frac{\beta_{hh}I}{1 + \alpha_{hh}I} \right) S = 0, \\
\left( \frac{\beta_{ch}Y}{1 + \alpha_{eh}Y} + \frac{\beta_{hh}I}{1 + \alpha_{hh}I} \right) S - \xi E = 0, \\
\xi E - \eta I = 0, \\
\beta_{hv}\left( \frac{\Lambda}{m} - Y \right) I - mY = 0.
\end{cases}
\]

(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 \((AI^2 + BI + C)I = 0\), 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
\[
B > (\mu \eta + \varepsilon \gamma + \varepsilon \mu)(\beta_{vh} \beta_{hv} \Lambda + m^2 \beta_{hh}) + \mu(\Lambda \alpha_{vh} \beta_{hv} + m \beta_{hv}) \xi \eta(1 - R_0) + \mu \alpha_{hh} m^2 \xi \eta(1 - R_0)
\]
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
\[
J(E_0) = \begin{pmatrix}
-\mu & c & d + b - \beta_{hh}K - \beta_{vh}K \\
0 & -\xi & \beta_{hh}K \\
0 & \varepsilon & -\eta \\
0 & 0 & \beta_{hv} \frac{\Lambda}{m} - m
\end{pmatrix}.
\]

Thus the characteristic equation at the disease-free equilibrium \(E_0\) is
\[
\begin{vmatrix}
\lambda + \mu & -c & \beta_{hh}K - d - b & \beta_{vh}K \\
0 & \lambda + \xi & -\beta_{hh}K & -\beta_{vh}K \\
0 & -\varepsilon & \lambda + \eta & 0 \\
0 & 0 & -\beta_{hv} \frac{\Lambda}{m} & \lambda + m
\end{vmatrix} = 0.
\]

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
\[
b_1 := \xi + \eta + m > 0,
b_2 := \xi \eta + (\xi + \eta)m - \varepsilon \beta_{hh}K,
b_3 := m \xi \eta - \varepsilon K \left(\beta_{hh}m + \beta_{hv} \beta_{vh} \frac{\Lambda}{m}\right).
\]
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 $\lambda_2 \lambda_3 \lambda_4 = -b_1 > 0$, 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

$$
\begin{align*}
\dot{E} &\leq \beta_{ch} K Y + \beta_{hh} K I - \xi E, \\
\dot{I} &\leq \varepsilon E - \eta I, \\
\dot{Y} &\leq \beta_{hv} \frac{\Lambda}{m} I - mY.
\end{align*}
$$

Consider the following comparison system

$$
\begin{align*}
\dot{Z}_1 &= \beta_{hh} K Z_2 + \beta_{ch} K Z_3 - \xi Z_1, \\
\dot{Z}_2 &= \varepsilon Z_1 - \eta Z_2, \\
\dot{Z}_3 &= \beta_{hv} \frac{\Lambda}{m} Z_2 - mZ_3.
\end{align*}
$$

(3.2)

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

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

(3.3)

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

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

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

$$
c_1 c_2 - c_3 = (\xi + \eta + m)(\xi \eta + (\xi + \eta) m - \varepsilon \beta_{hh} K) - m \xi \eta + \varepsilon K \left( \beta_{hh} m + \beta_{hv} \beta_{ch} \frac{\Lambda}{m} \right)
$$

$$
= (\xi + \eta)(\xi \eta + (\xi + \eta) m - \varepsilon \beta_{hh} K) + m^2 (\xi + \eta) + \varepsilon K \beta_{hv} \beta_{ch} \frac{\Lambda}{m} > 0.
$$

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 $(Z_1(t), Z_2(t), Z_3(t)) \to (0, 0, 0)$ 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

$$
\begin{align*}
Z_1(t_1) &= 0, \\
\dot{Z}_1(t_1) &\leq 0, \\
Z_1(t) &> 0 \text{ for all } t \in (0, t_1).
\end{align*}
$$

(3.4)
For \( t \in (0, t_1] \),

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

\[
Z_3(t) = e^{-mt} \left( Z_3(0) + \int_0^t \frac{\Delta}{m} Z_2(s)e^{ms}ds \right) > 0. \tag{3.6}
\]

Then

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

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 \( (E(t), I(t), Y(t)) \rightarrow (0, 0, 0) \) as \( t \rightarrow +\infty \).

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

\[
\lim_{t \rightarrow +\infty} R(t) = \lim_{t \rightarrow +\infty} \gamma I(t)/\mu = 0. \tag{3.8}
\]

Thus \( R(t) \rightarrow 0 \) as \( t \rightarrow +\infty \) in any case. Due to \( S + E + I + R = K \), we obtain \( S(t) \rightarrow K \) as \( t \rightarrow +\infty \). Therefore, \( (S(t), E(t), I(t), Y(t)) \rightarrow (K, 0, 0, 0) \) as \( t \rightarrow +\infty \) 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).

\[ \square \]

### 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

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

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

\[
\zeta := \frac{\beta_{vh} Y^*}{1+\alpha_{vh} Y^*} + \frac{\beta_{hh} I^*}{1+\alpha_{hh} I^*}, \quad \vartheta := \beta_{hv} I^* + m.
\]
The corresponding characteristic equation is \( \lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4 = 0 \), where

\[
a_1 := \mu + \xi + \eta + \zeta + \vartheta,
\]

\[
a_2 := \mu(\xi + \eta) + (\mu + \xi + \eta)\vartheta + (\varepsilon + \mu + \eta + \vartheta)\zeta + \xi\eta - \frac{\varepsilon \beta_{hh} S^*}{(1 + \alpha_{hh} I^*)^2},
\]

\[
a_3 := \mu \left( \xi \eta - \frac{\beta_{hh} S^*}{(1 + \alpha_{hh} I^*)^2} + \mu(\xi + \eta)\vartheta + (\varepsilon + \mu + \eta + \vartheta)\zeta + (\varepsilon + \mu + \eta)\vartheta \right)
+ \vartheta \left( \xi \eta - \frac{\beta_{hh} S^*}{(1 + \alpha_{hh} I^*)^2} - \frac{\varepsilon \beta_{hv} \beta_{vh} S^* \Lambda}{m\vartheta(1 + \alpha_{vh} Y^*)^2} \right) + \frac{\varepsilon \beta_{hv} \beta_{vh} S^* Y^*}{\vartheta(1 + \alpha_{vh} Y^*)^2}.
\]

\[
a_4 := \mu \vartheta \left( \xi \eta - \frac{\beta_{hh} S^*}{(1 + \alpha_{hh} I^*)^2} - \frac{\varepsilon \beta_{hh} \beta_{vh} S^* \Lambda}{m\vartheta(1 + \alpha_{vh} Y^*)^2} \right) + \frac{\varepsilon \beta_{hv} \beta_{vh} S^* Y^*}{\vartheta(1 + \alpha_{vh} Y^*)^2}
+ (\varepsilon + \mu + \eta)\vartheta \zeta \vartheta.
\]

Define

\[
\Upsilon := a_3(a_1 a_2 - a_3) - a_1^2 a_4.
\]

**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 \( \Upsilon \) 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 \( a_i \) (\( i = 1, \ldots, 4 \)) are given in (4.1). So, by the Routh-Hurwitz Theorem we need to prove

\[
a_1 > 0, \quad \begin{vmatrix} a_1 & a_3 \ 1 & a_2 \end{vmatrix} > 0, \quad \begin{vmatrix} a_1 & a_3 & 0 \ 1 & a_2 & a_4 \ 0 & a_1 & a_3 \end{vmatrix} > 0, \quad \begin{vmatrix} a_1 & a_3 & 0 & 0 \ 1 & a_2 & a_4 & 0 \ 0 & 1 & a_3 & 0 \ 0 & 1 & 2 & a_4 \end{vmatrix} > 0.
\]

Straight computation shows that it is equivalent to prove \( a_1, a_2, a_3, a_4 > 0 \) and \( a_3(a_1 a_2 - a_3) - a_1^2 a_4 > 0 \). Since \( \Upsilon \) is defined as \( a_3(a_1 a_2 - a_3) - a_1^2 a_4 \) 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

\[
G := \xi \eta - \frac{\varepsilon \beta_{hh} S^*}{(1 + \alpha_{hh} I^*)^2}, \quad \Theta := \frac{\varepsilon \beta_{hv} \beta_{vh} S^* Y^*}{(1 + \alpha_{vh} Y^*)^2},
\]

\[
H := \xi \eta - \frac{\varepsilon \beta_{hh} S^*}{(1 + \alpha_{hh} I^*)^2} - \frac{\varepsilon \beta_{hv} \beta_{vh} S^* \Lambda}{m\vartheta(1 + \alpha_{vh} Y^*)^2}.
\]

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

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

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

\[
\begin{align*}
    a_1 &= \mu + \xi + \eta + 3 + \theta > 0, \\
    a_2 &= \mu(\xi + \eta) + (\mu + \xi + \eta)\theta + (\epsilon + \mu + \eta + \theta)\zeta + G > 0, \\
    a_3 &= \mu G + (\epsilon + \mu + \eta)\xi + (\epsilon + \mu + \eta)\zeta + \theta H + \Theta > 0, \\
    a_4 &= \mu \theta H + \mu \Theta + (\epsilon + \mu + \eta)\zeta \theta > 0.
\end{align*}
\]

The proof is finished. \qed

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 $\hat{\Omega}$ if there exists a constant $\hat{\rho} > 0$ such that any solution $(S(t), E(t), I(t), Y(t))$ of (1.2) with the initial value $(S(0), E(0), I(0), Y(0)) \in \hat{\Omega}$ 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 $\hat{\Omega}$ is the inner of $\Omega$. Note that this definition is called uniformly strongly persistent in [32].

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

**Proof.** Since $\Omega$ is bounded and positively invariant, system (1.2) is point dissipative in $\Omega$ (see [15]). Thus, there exists a compact set $M_0$ such that all solutions of system (1.2) initiated in $\Omega$ 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 $\omega$-limit set of the orbit starting from $x_0 \in \hat{\Omega}$ and

\[
\Xi_1 := \{ x_s \in \partial \hat{\Omega} \mid x(t, x_s) \in \partial \Omega, \forall t > 0 \}, \quad \Xi_2 := \bigcup_{x_s \in \Xi_1} \omega(x_s),
\]

where $x(t, x_s) = (S(t), E(t), I(t), Y(t))$ is the solution of system (1.2). Clearly, $\Xi_1, \Xi_2 \subseteq \partial \hat{\Omega}$. By the expression of system (1.2), all solutions starting in $\partial \hat{\Omega}$ but not on the $S$-axis ultimately goes into $\Omega$. Thus $\Xi_1 = \{ (S, 0, 0, 0) \mid 0 \leq S \leq K \}$ 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 $\hat{\Omega}$, by [32, Theorem 4.6] it suffices to prove that $E_0$ is a weak repeller (see [32, p408]) for $\Omega$, i.e., every solution starting from $x_0 \in \Omega$ satisfies

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

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

\[
g(\nu) := \epsilon(K - \nu) \left( \frac{\beta_{hh} m}{1 + \alpha_{eh} \nu} + \frac{\beta_{hv} \beta_{eh}}{m(1 + \alpha_{hh} \nu)} \right) - m \xi \eta
\]

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

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

$$
\begin{align*}
\dot{E} &\geq \left( \frac{\beta_{ch} \hat{Y}}{1 + \alpha_{ch} \hat{\nu}} + \frac{\beta_{hh} \hat{I}}{1 + \alpha_{hh} \hat{\nu}} \right) (K - \hat{\nu}) - \xi \hat{E}, \\
\dot{I} &\geq \varepsilon \hat{E} - \eta \hat{I}, \\
\dot{Y} &\geq \beta_{hy} \left( \frac{\Lambda}{m} - \hat{\nu} \right) \hat{I} - m \hat{Y}.
\end{align*}
$$

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

$$
\begin{align*}
\dot{E} &= \left( \frac{\beta_{ch} Y}{1 + \alpha_{ch} \nu} + \frac{\beta_{hh} I}{1 + \alpha_{hh} \nu} \right) (K - \nu) E, \\
\dot{I} &= \varepsilon E - \eta I, \\
\dot{Y} &= \beta_{hy} \left( \frac{\Lambda}{m} - \nu \right) I - m Y
\end{align*}
$$

has Jacobian matrix

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

Let $\bar{\lambda}$ be the maximum eigenvalue of $\hat{A}$. It is not hard to prove $\bar{\lambda} > 0$ 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 $\bar{\lambda}$ and $|\hat{n}| = 1$. There exists a positive $l$ such that $l \hat{n}_1 < \hat{E}(T), l \hat{n}_2 < \hat{I}(T), l \hat{n}_3 < \hat{Y}(T)$. Moreover, the solution $(E(t), I(t), Y(t))$ with initial value $(E(T), I(T), Y(T)) = (l \hat{n}_1, l \hat{n}_2, l \hat{n}_3)$ of linear system (4.5) goes to positive infinity as $t \to +\infty$. By the comparison principle (see [25]) we get $E(t) > E(t), I(t) > I(t), Y(t) > Y(t)$. Hence, $E(t) \to +\infty, I(t) \to +\infty, Y(t) \to +\infty$ as $t \to +\infty$. This contradicts $\hat{E}(t) \to 0, \hat{I}(t) \to 0, \hat{Y}(t) \to 0$ as $t \to +\infty$. Then we get

$$
W^*(E_0) \bigcap \Omega = \emptyset.
$$

(4.6)

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

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

(4.7)
Proof. Let 
\[ \Gamma := \{(S, E, I, Y) \in \Omega \mid \rho \leq S \leq K, \rho \leq E \leq K, \rho \leq I \leq K, \rho \leq Y \leq \Lambda/m \} \].

Clearly, \( \Gamma \) is compact and absorbing because \( \Omega \) is positively invariant. For system (1.2), define
\[
P(t) := \begin{pmatrix}
\frac{1}{\tau(t)} & 0 & 0 & 0 & 0 \\
0 & \frac{1}{\tau(t)} & 0 & 0 & 0 \\
0 & 0 & \frac{1}{\tau(t)} & 0 & 0 \\
0 & 0 & 0 & \frac{1}{\tau(t)} & 0 \\
0 & 0 & 0 & 0 & \frac{1}{\tau(t)}
\end{pmatrix}
\]
for \( t \geq 0 \), 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
\[
J(t) := \begin{pmatrix}
-\mu - q_4 & c & d + b - q_1 & -q_2 & 0 \\
q_4 & -\xi & q_1 & q_2 & 0 \\
0 & \varepsilon & -\eta & 0 & 0 \\
0 & 0 & q_3 & -\beta_h I - m & 0
\end{pmatrix}
\]
is the Jacobian matrix of (1.2), \(J^2(t)\) is its second additive compound matrix (see [19]) and
\[
q_1 = \frac{\beta_{hh} S}{(1 + \alpha_{hh} I)^2}, \quad q_2 = \frac{\beta_{hh} S}{(1 + \alpha_{hh} Y)^2}, \quad q_3 = \beta_{hh} \left( \frac{\Lambda}{m} - Y \right), \quad q_4 = \frac{\beta_{eh} Y}{1 + \alpha_{eh} Y} + \frac{\beta_{hh} I}{1 + \alpha_{hh} I}.
\]

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
\[
Q_{11} = M_{11} - \frac{E}{E'}, \quad Q_{12} = \left( \frac{\varepsilon}{E} q_1, \frac{\varepsilon}{E} (q_1 - b - d) \right), \quad Q_{13} = \left( \frac{\gamma}{E} q_2, \frac{\gamma}{E} q_2, 0 \right). \]
\[
Q_{21} = \begin{pmatrix}
\varepsilon \\
0
\end{pmatrix}, \quad Q_{22} = \begin{pmatrix}
M_{22} - \frac{\beta}{\mu} & c & \frac{\beta}{\mu} \\
q_4 & M_{44} - \frac{\beta}{\mu} & \frac{\beta}{\mu}
\end{pmatrix}, \quad Q_{23} = \begin{pmatrix}
0 & 0 & \frac{\gamma}{\beta} q_2 \\
0 & 0 & \frac{\gamma}{\beta} q_2
\end{pmatrix},
\]
\[
Q_{31} = \begin{pmatrix}
0 \\
0 \\
0
\end{pmatrix}, \quad Q_{32} = \begin{pmatrix}
\frac{\varepsilon}{\tau} q_3 & 0 \\
0 & \frac{\varepsilon}{\tau} q_3 \\
0 & 0
\end{pmatrix}, \quad Q_{33} = \begin{pmatrix}
M_{33} - \frac{\varepsilon}{\tau} & c & b + d - q_1 \\
q_4 & M_{55} - \frac{\varepsilon}{\tau} & \frac{\gamma}{\beta} q_2 \\
0 & \varepsilon & M_{66} - \frac{\gamma}{\beta}
\end{pmatrix}.
\]
and
\[
M_{11} = -\mu - q_4 - \xi, \quad M_{22} = -\mu - q_4 - \eta, \quad M_{33} = -\mu - q_4 - \beta_{vh}I - m, \\
M_{44} = -\xi - \eta, \quad M_{55} = -\xi - \beta_{hv}I - m, \quad M_{66} = -\eta - \beta_{hv}I - m.
\]
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 Lozinski\u00ed measure (see [5]) with respect to this norm. As described in [26], for given \( t \geq 0 \) we get \( \sigma(Q(t)) \leq \max \{ g_1(t), g_2(t), g_3(t) \} \), where
\[
g_i(t) := \sigma_1(Q_{ii}) + \sum_{j \in \{1,2,3\} \setminus \{i\}} |Q_{ij}|, \quad i = 1, 2, 3,
\]
and \( \sigma_1 \) denotes the Lozinski\u00ed measure with respect to the \( l_1 \) norm (see [20]).

Let
\[
\bar{b} := \min \left\{ \mu, \mu - \frac{2\beta_{vh}AK}{\rho(1+\alpha_{vh}\rho)^2}, \mu - \omega \right\}.
\]
By \( (4.7) \), we have \( \bar{b} > 0 \). Thus for \( t \geq 0 \)
\[
g_1(t) < -\bar{b}, \quad g_2(t) < -\bar{b}, \quad g_3(t) < -\bar{b}.
\]
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, \( \beta_{hv}, \beta_{vh} \) have a greater influence on \( I \) than \( \beta_{hh} \).
Thus, strategies such as drug control and biological control that either decrease biting rates $\beta_{hv}, \beta_{vh}$ 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 $\beta_{hv}, \beta_{vh}$ and $\beta_{hh}$, 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 $\beta_{hv}, \beta_{vh}$ and $\beta_{hh}$ 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).
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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