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Dynamical Behavior of a Stage-structure Epidemic Model

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Abstract: In this paper, we investigate a host-vector epidemic model with stage-structure for the vector. Analytical results reveal that the model has two disease-free equilibria, one (uninfected host population only) is always unstable, the other (the uninfected hosts and vectors only) is global asymptotically stable when the basic reproduction number R_0 isn't larger than unity. For the basic reproductive number R_0 is greater than one, a unique endemic equilibrium exists and the disease is uniformly persistent. Furthermore, using the theory of centre manifold it is shown that the model exhibits the phenomenon of backward bifurcation, where the stable disease-free equilibrium coexists with a stable endemic equilibrium. In the end, numerical simulations are presented to illustrate the effect of stage structure on dynamics of the disease transmission, and also verify the analyzed results.

Keywords: Host-vector; epidemic model; stage structure; backward bifurcation; stability

1 Introduction

Mathematical models provide a powerful tool to understand the spread and control of epidemic diseases. Among all kind of diseases, host-vector diseases are one of the major growing issues due to the frequent occurrence of natural disasters and environmental degradation around the world, which creates conditions suitable for breeding of vectors so that host-vector diseases are constantly emerging. Host-vector diseases are spread among hosts by vectors. For example, human and animal diseases including dengue fever, malaria, Chagas' disease, scrub typhus and so on. Also, plant virus diseases such as African cassava mosaic virus, tobacco mosaic virus, potato virus Y, beet yellows virus, cucumber mosaic virus, rice dwarf virus and pine wilt disease are transmitted by arthropod vectors [1-2].

In many epidemiological models, stage-structure has received wide attention in recent years due to the facts that individuals in a population may grow through several stage of physiology, such as the immature and mature [3-5]. For instance, chickenpox, measles, and scarlet fever always occur in immaturity, while other infectious diseases such as typhus, gonorrhea, syphilis, schistosomiasis cutaneous, diphtheria and sexually transmitted diseases spread or have more opportunities to break out in maturity. In paper [6], Xiao and Chen classify individuals as belonging to either immature or mature and suppose that the immature population is susceptible to infection, while the mature population does not contract the disease. They studied the dynamical behavior of an SIS infectious disease model with stage structure. Jia and Li[7] suppose that the mature population is susceptible to the infection, while the immature population does not contract the diseases, and analyzed the SIR epidemic model with stage structure.

Moreover, some epidemics such as Chagas' disease, hepatitis C virus (HCV), gonorrhea and other sexually transmitted diseases, may develop through several stage of infections, and have different ability to transmit these infections in different stages of infection[8,9]. For example, in the case of Chagas' disease [8], the acute stage follows the invasion of the blood stream by the protozoan T. cruzi. After the acute phase, which lasts one or two months, then the infected individuals enter the chronic stage and stay there for variable duration that lasts from 10 to 20 years. A stage-structured epidemic model with a nonlinear incidence was studied in paper [10], where the period of infection was partitioned into the stage-1 and stage-2 according to the developing process of infection, and global dynamics of the model was rigorously established. Other epidemic models with stage structure are presented and studied in [11-16].

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According to a survey from the above-mentioned references, all the results in [6-16] focused on stage structure for hosts. However, for many kind of vector-host diseases, stage-structure for the vector is an important factor that affects disease transmission. There are some kinds of vector-host disease which are only spread among hosts by the immature stage of the vector, such as cysticercosis and Scrub typhus. For instance, Scrub typhus is transmitted to humans and rodents by some species of trombiculid mites ("chiggers", Leptotrombidium deliense and others). The mite is very small (0.2 – 0.4mm) and can only be seen through a microscope or magnifying glass. The adult mites have a four-stage lifecycle: egg, larva,nymph and adult. The larva is the only stage (chigger) that can transmit the disease to humans and other vertebrates, since the other life stages (nymph and adult) do not feed on vertebrate animals. Both the nymph and the adult are free-living in the soil [17]. While some infectious diseases such as malaria, dengue fever, West Nile virus, pine wilt disease [18] and so on are spread only by adult vectors.

Consequently, realistic study of host-vector disease transmission in a population needs considering stage structure for the vector, each stage being homogeneous. However, up till now no one has considered the effect of stage structure for the vector on the dynamics of vector–host diseases. Motivated by the above discussions, in this paper, we aim to model the transmission dynamics of a vector–host disease with stage structure for the vector and gain insights in the dynamics of the system. The remaining part of this paper is organized as follows: in Section 2, we mainly formulate our model. In Section 3, we show mathematical analysis to establish the existence and stability results of the disease-free equilibria for the proposed model. In Section 4, detail analysis of the endemic equilibria and the existence of a backward bifurcation is studied. In Section 5, the Uniformly persistence of the disease is analyzed. In Section 6, some numerical simulations are given to verify the analyzed results, and also illustrate the effect of stage structure on the spread of the vector-host diseases. The paper ends with a conclusion.

2 Model description

In this section we formulate a host-vector disease model with stage structure to describe the transmission dynamics of a vector borne disease. First, we suppose that the model satisfies the following assumptions:

(1) The total host population $N_h(t)$ is divided into two distinct subclasses which are susceptible and infectious, with densities denoted by $S_h(t)$ and $I_h(t)$, respectively, so that $N_h(t) = S_h(t) + I_h(t)$.

(2) The total vector population is divided into two stage groups: immature or mature, and they are denoted by $M_v(t)$ and $N_v(t)$, respectively.

(3) Only mature vectors are capable of spreading diseases. So, we divide the total mature vector population $N_v(t)$ into two distinct epidemiological subclasses which are susceptible and infectious, with densities denoted by $S_v(t)$ and $I_v(t)$, respectively, so that $N_v(t) = S_v(t) + I_v(t)$.

(4) The virus in vectors does not cause the death of vectors, and does not influence the propagation of vectors.

(5) The immature vector population only grows up to susceptible mature vector population, and the susceptible mature vector population experiences crowing effects.

(6) Linear function $b_2 N_v(t)$is used as the birth term of the immature vector population.

Using the above assumptions, then the stage-structured vector-host disease model can be written as:

$$\begin{cases} S_h'(t) = b_1 - \beta_1 S_h(t) I_v(t) - \mu_h S_h(t), \\ I_h'(t) = \beta_1 S_h(t) I_v(t) - (\mu_h + \delta_h) I_h(t), \\ M_v'(t) = b_2(S_v(t) + I_v(t)) - (\mu_v + \eta) M_v(t), \\ S_v'(t) = \eta M_v(t) - \beta_2 S_v(t) I_h(t) - \mu_v S_v(t) - \alpha S_v^2(t), \\ I_v'(t) = \beta_2 S_v(t) I_h(t) - \mu_v I_v(t), \end{cases} \tag{1}$$

where $b_1, b_2, \mu_h, \delta_h, \mu_v, \eta, \beta_1, \beta_2, \alpha$ are positive. b_1 is the total recruitment of the hosts. μ_h and μ_v are respectively the natural death rate of infected hosts and vectors. δ_h is the disease-caused death rate of infected hosts. η is the conversion rate from immature vectors to mature vectors. α is the rate of crowing effects of the susceptible hosts. β_1 and β_2 are respectively the rate of biting from susceptible hosts to infected hosts and susceptible vectors to infected vectors.

System (1) will be analyzed in the biological domain Γ given by

$$\Gamma = \left\{ (S_h, I_h, M_v, S_v, I_v) \in R_5^+ \,|0 \le S_h + I_h \le b_1/\mu_h, M_v \ge 0, S_v \ge 0, I_v \ge 0. \right\}$$

then, it is easy to verify that Γ is positively invariant of system (1).

3 Disease-free equilibrium

Direct calculations show that the system (1) has two disease-free equilibria, they are

$E_0\,(b_1/\mu_h, 0, 0, 0, 0)$(uninfected host population only), which always exists and $E_1\left(S_h^0, 0, M_v^0, S_v^0, 0\right)$ (only uninfected hosts and vectors), which exists when $\sigma = b_2\eta - \mu_v(\mu_v+\eta) > 0$,where $S_h^0 = b_1/\mu_h, M_v^0 = b_2\sigma/\alpha(\mu_v+\eta)^2, S_v^0 = \sigma/\alpha(\mu_v+\eta)$.

To ensure the existence of equilibrium E_1, in the following, we always assume that $\sigma > 0$.

Theorem 1 *The local stability of disease-free equilibria for system (1) is as follows.*
(1) The disease-free equilibrium $E_0\,(b_1/\mu_h, 0, 0, 0, 0)$ (uninfected host population only) is always unstable.
(2) The disease-free equilibrium $E_1\left(S_h^0, 0, M_v^0, S_v^0, 0\right)$ (only uninfected hosts and vectors) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$, where

$$R_0 = \frac{\beta_1\beta_2 S_h^0 S_v^0}{\mu_v(\mu_h+\delta_h)} = \frac{b_1\sigma\beta_1\beta_2}{\mu_h\mu_v\alpha(\mu_h+\delta_h)(\mu_v+\eta)} \tag{2}$$

is the reproduction number of system (1).

Proof. (1) Linearizing system (1) about the disease-free equilibrium E_0, there exist five eigenvalues, three of the eigenvalues are $-\mu_h, -\mu_v, -(\mu_h+\delta_h)$, the other two eigenvalues can be obtained by solving equation

$$\lambda^2 + (2\mu_v+\eta)\lambda - \sigma = 0. \tag{3}$$

Since $2\mu_v+\eta > 0$ and $\sigma > 0$, so there exists one positive and one negative real roots for Eq. (3), Thus, E_0 is unstable.
(2) The characteristic equation of the system (1) at its disease free equilibrium E_1 is given by

$$(\lambda+\mu_h)[\lambda^2 + (2\mu_v+\mu+2\sigma/(\mu_v+\eta))\,\lambda + \sigma][\lambda^2 + (\mu_h+\delta_h+\mu_v)\lambda + \mu_v(\mu_h+\delta_h) - \beta_1\beta_2 S_h^0 S_v^0] = 0. \tag{4}$$

There are five eigenvalues corresponding to Eq. (4). One of the eigenvalues is$-\mu_h$. The other four eigenvalues can be obtained by solving

$$\lambda^2 + (2\mu_v+\eta+2\sigma/(\mu_v+\eta))\,\lambda + \sigma = 0, \tag{5}$$

and

$$\lambda^2 + (\mu_h+\delta_h+\mu_v)\lambda + \mu_v(\mu_h+\delta_h) - \beta_1\beta_2 S_h^0 S_v^0 = 0. \tag{6}$$

Since $2\mu_v+\eta+2\sigma/(\mu_v+\eta) > 0$ and $\sigma > 0$, therefore the two roots of (5) have negative real parts. While for Eq.(6), the two roots of (6) have negative real parts if and only if $R_0 < 1$. Therefore, the disease-free equilibrium E_1 is locally asymptotically stable if $R_0 < 1$ and unstable is $R_0 > 1$. ■

To realize the global stability of the disease-free equilibrium, we establish the following theorem.

Theorem 2 *The disease-free equilibrium E_1 of system (1) is global asymptotically stable if $R_0 \le 1$ and $M_v(t) \le M_v^0$ for $t \ge 0$.*

Proof. Construct the following Lyapunov functional

$$\begin{aligned} V(t) = {} & c_1\left(S_h(t) - S_h^0\ln(S_h(t)/S_h^0)\right) + c_2 I_h(t) + c_3\left(M_v(t) - M_v^0 - M_v^0\ln(M_v(t)/M_v^0)\right) \\ & + c_4\left(S_v(t) - S_v^0\ln(S_v(t)/S_v^0)\right) + c_5 I_v(t), \end{aligned}$$

where $c_1 = c_2 = 1, c_3 = c_4\eta M_v^0/(b_2 S_v^0), c_4 = \mu_h + \delta_h/(\beta_2 S_v^0), c_5 = c_4$.

Since at the disease-free equilibrium E_1, the third and fourth equation of system (1) can be rewritten as

$$\begin{cases} M_v'(t) = (b_2/M_v^0)[-S_v(t)(M_v(t)-M_v^0) + M_v(t)(S_v(t)-S_v^0)] + b_2 I_v(t), \\ S_v'(t) = (\eta/S_v^0)[-M_v(t)(S_v(t)-S_v^0) + S_v(t)(M_v(t)-M_v^0)] - \alpha S_v(t)(S_v(t)-S_v^0) - \beta_2 S_v(t) I_h(t). \end{cases} \tag{7}$$

Therefore, calculate the derivative of $V(t)$ along the solution of (1), then from (7) we have

$$\begin{aligned} V'(t) = {} & (-c_1\mu_h/S_h(t))(S_h(t)-S_h^0)^2 - c_1\beta_1 S_h(t) I_v(t) + c_1\beta_1 S_h^0 I_v(t) + c_2\beta_1 S_h(t) I_v(t) \\ & - c_2(\mu_h+\delta_h) I_h(t) - (c_3 b_2 S_v(t)/M_v^0 M_v(t))(M_v(t)-M_v^0)^2 \\ & + (c_3 b_2/M_v^0)(M_v(t)-M_v^0)(S_v(t)-S_v^0) + (c_3 b_2 I_v(t)/M_v(t))(M_v(t)-M_v^0) \\ & - c_4\alpha(S_v(t)-S_v^0)^2 - (c_4\eta M_v(t)/S_v^0 S_v(t))(S_v(t)-S_v^0)^2 \\ & + (c_4\eta/S_v^0)(M_v(t)-M_v^0)(S_v(t)-S_v^0) - c_4\beta_2 S_v(t) I_h(t) \\ & + c_4\beta_2 S_v^0 I_h(t) + c_5\beta_2 S_v(t) I_h(t) - c_5\mu_v I_v(t). \end{aligned}$$

By some rearrangement, it follows that

$$\begin{aligned}V'(t) &= (-c_1\mu_h/S_h(t))(S_h(t)-S_h^0)^2 - c_4\alpha(S_v(t)-S_v^0)^2 + (c_2-c_1)\beta_1 S_h(t)I_v(t)\\ &+[c_4\beta_2 S_v^0 - c_2(\mu_h+\delta_h)]I_h(t) + [c_1\beta_1 S_h^0 - c_5\mu_v]I_v(t)\\ &+(c_3b_2I_v(t)/M_v(t))(M_v(t)-M_v^0) - (c_3b_2S_v(t)/M_v^0M_v(t))(M_v(t)-M_v^0)^2\\ &+[(c_3b_2/M_v^0)+(c_4\eta/S_v^0)](M_v(t)-M_v^0)(S_v(t)-S_v^0) - (c_4\eta M_v(t)/S_v^0S_v(t))(S_v(t)-S_v^0)^2.\end{aligned}$$

After some reduction, then we have

$$\begin{aligned}V'(t) &= -(c_1\mu_h/S_h(t))(S_h(t)-S_h^0)^2 - c_4\alpha(S_v(t)-S_v^0)^2\\ &-(\eta/S_v^0)\left[\sqrt{(S_v(t)/M_v(t))}(M_v(t)-M_v^0) - \sqrt{(M_v(t)/M_v^0)}(S_v(t)-S_v^0)\right]^2\\ &+(\mu_v(\mu_h+\delta_h)/\beta_2 S_v^0)[R_0-1]I_v(t) + (c_3b_2I_v(t)/M_v(t))(M_v(t)-M_v^0).\end{aligned} \tag{8}$$

Therefore, it follows from (8) that $V'(t)\le 0$ in Ω when$M_v(t)\le M_v^0$ for all $t\ge 0$. The equation $V'(t)=0$ hold if and only if $S_h(t)=S_h^0, I_h(t)=0, M_v(t)=M_v^0, S_v(t)=S_v^0, I_v(t)=I_v^0$.

Thus, we prove the global stability of the disease in Ω. The maximal compact invariant set in $\{S_h(t), I_h(t), M_v(t), S_v(t), I_v(t), \in \Omega : V'(t)=0\}$is $\{E_1\}$ when $R_0\le 1$ and $M_v(t)\le M_v^0$ for and $t\ge 0$. From the LaSalle's invariance principle, we finish the proof of Theorem 2. ■

Remark 3 *From Theorem 2 we can see that when $R_0\le 1$, then the disease-free equilibrium E_1 of system (1) is globally asymptotically stable if the immature vector population size $M_v(t)$ is not larger than the immature vector equilibrium level M_v^0. That is, the immature vector density threshold for a successful disease eradication is $M_v(t)=M_v^0$. If $M_v(t)\le M_v^0$ and $R_0\le 1$ then disease can be eradicated, otherwise pathogen persists though $R_0\le 1$. Therefore, it is necessary to take some strategies to reduce the vector populations at its youth stage for vector-borne disease prevention.*

4 Disease equilibrium

In order to find equilibria (endemic equilibria) of the system (1) where at least one of the infected components of the system (1) is non-zero, we need to take the following steps.

Let $E_2\left(\tilde{S}_h, \tilde{I}_h, \tilde{M}_v, \tilde{S}_v, \tilde{I}_v\right)$represents any arbitrary endemic equilibrium of the model (1). Solving the equations of the system (1) at steady state gives

$$\tilde{S}_h = \frac{b_1\alpha\mu_v^2(\mu_v+\eta)}{\alpha\mu_h\mu_v^2(\mu_v+\eta)+\sigma\beta_1\beta_2(\mu_v+\beta_2\tilde{I}_h)\tilde{I}_h}, \quad \tilde{M}_v = \frac{b_2\sigma(\mu_v+\beta_2\tilde{I}_h)^2}{\alpha\mu_v^2(\mu_v+\eta)^2},$$

$$\tilde{S}_v = \frac{\sigma(\mu_v+\beta_2\tilde{I}_h)}{\alpha\mu_v(\mu_v+\eta)}, \quad \tilde{I}_v = \frac{\sigma\beta_2\tilde{I}_h(\mu_v+\beta_2\tilde{I}_h)}{\alpha\mu_v^2(\mu_v+\eta)}.$$

If $\tilde{I}_h\neq 0$, then substituting $\tilde{S}_h$,$\tilde{I}_v$ in the second equation of the system (1) at steady state, we obtain after some calculations the following quadratic equation:

$$f(\tilde{I}_h) = a_2\tilde{I}_h^2 + a_1\tilde{I}_h + a_0 = 0, \tag{9}$$

where $a_2=\sigma\beta_1\beta_2^2(\mu_h+\delta_h)>0$, $a_1=\sigma\beta_1\beta_2\left[\mu_v(\mu_h+\delta_h)-b_1\beta_2\right]$, $a_0=\alpha\mu_h\mu_v^2(\mu_h+\delta_h)(\mu_v+\eta)(1-R_0)$, and R_0 given in (2) is the reproduction number of system (1). Clearly the coefficient a_2 is always positive, and a_0 is positive (negative) if R_0 is less than (greater than) unity, respectively. Since $a_2>0$, the existence of the positive solutions of Eq. (9) will depend on the signs of a_1 and a_0. If $R_0>1$, then there are two roots of Eq. (9) of which one root is positive and thus there is a unique endemic equilibrium. If $R_0=1$, then $a_1>0$ and there is no positive solution of (9). Since equilibria depend continuously on R_0which shows that there exists an interval to the left of R_0 on which there are two positive equilibria

$$\tilde{I}_{h1} = \frac{-a_1-\sqrt{a_1^2-4a_2a_0}}{2a_2}, \quad \tilde{I}_{h2} = \frac{-a_1+\sqrt{a_1^2-4a_2a_0}}{2a_2}.$$

If $a_0>0$ and either$a_1\ge 0$, or $a_1^2<4a_2a_0$, there are no positive solutions of (9) and thus there are no endemic equilibria. For different range of these parameters the following results are established.

Theorem 4 *The model (1) has:*
(1) a unique endemic equilibrium in Ω if $a_0<0\Leftrightarrow R_0>1$.
(2) a unique endemic equilibrium in Ω if $a_1<0$ and $a_0=0$ or $a_1^2-4a_2a_0=0$.
(3) two endemic equilibriia in Ω if $a_0>0, a_1<0$ and $a_1^2-4a_2a_0>0$.
(4) no endemic equilibria otherwise.

Case (3) of Theorem 4 indicates the possibility of a backward bifurcation (where the locally-asymptotically stable disease-free equilibrium co-exists with a locally-asymptotically stable endemic equilibrium when $R_0 < 1$. In fact, set the discriminant $a_1^2 - 4a_2a_0$ to be zero and solve for the critical value of R_0, denoted by R_c, then we have

$$R_c = 1 - \frac{a_1^2}{4a_2\mu_h\mu_v^2\alpha(\mu_h+\delta_h)(\mu_v+\eta)}.$$

It is clear that $R_c < R_0$ is equivalent to $a_1^2 - 4a_2a_0 > 0$ and therefore, backward bifurcation would occur for values of R_0 such that $R_c < R_0 < 1$.

Here, we apply the center manifold theory approach to explore the existence of backward bifurcation of system (1).

Theorem 5 *If $\alpha > \mu_h\sigma/(\mu_v+\eta)$, then system (1) undergoes a backward bifurcation at $R_0 = 1$.*

Proof. Let β_2 be the bifurcation parameter. From $R_0 = 1$ we have$\beta_2^* = \frac{\mu_h\mu_v\alpha(\mu_h+\delta_h)(\mu_v+\eta)}{b_1\sigma\beta_1}$.

The matrix of the linearzed system about the disease free equilibrium E_1 is

$$A = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & -b_1S_h^0 \\ 0 & -(\mu_h+\delta_h) & 0 & 0 & b_1S_h^0 \\ 0 & 0 & -(\mu_v+\eta) & b_2 & b_2 \\ 0 & -\beta_2^*S_v^0 & \eta & -(b_2\eta+\sigma)/(\mu_v+\eta) & 0 \\ 0 & \beta_2^*S_v^0 & 0 & 0 & -\mu_v \end{pmatrix}$$

It is easy to calculate that the Jacobian A of the linearized system has a simple zero eigenvalue and all the other eigenvalues have negative real parts. Hence, the center manifold theory can be used to analyze the dynamics of the system (1). It can be shown that the Jacobian matrix of A at $\beta_2 = \beta_2^*$ has a right eigenvector given by the solution of the system $Aw = 0$ and defined as $w = (w_1, w_2, w_3, w_4, w_5)^T$, where

$$w_1 = -\left(b_1\beta_1/\mu_h^2\right)w_5, w_2 = (\alpha\mu_v(\mu_v+\eta)/(\beta_2^*\sigma))w_5, w_3 = (b_2/(\mu_v+\eta))w_5, w_4 = w_5, w_5 = w_5.$$

We then computer the left eigenvector given by the solution of the system $v^TA = 0$ and defined as $v = (v_1, v_2, v_3, v_4, v_5)^T$, where

$$v_2 = (\mu_h\mu_v/(b_1\beta_1))v_5, v_5 = v_5, v_1 = v_3 = v_4 = 0.$$

Next, by the methods of Theorem 4 in [19], we have to determine a and b. It can be shown that

$$\begin{aligned} a &= \sum_{k,i,j=1}^{n} v_kw_iw_j\frac{\partial^2 f_k}{\partial x_i\partial x_j}(E_1,\beta_2^*) = \beta_1w_5w_1v_2 + \beta_2w_2w_4v_5 \\ &= v_5w_5^2[-(\mu_v\beta_1/\mu_h) + (\alpha\mu_v(\mu_v+\eta)/\sigma)(\sigma+2\mu_v(\mu_v+\eta))] \end{aligned} \tag{10}$$

$$b = \sum_{k,i=1}^{n} v_kw_i\frac{\partial^2 f_k}{\partial x_i\partial\beta_2}(E_1,\beta_2^*) = v_5w_5S_v^0 > 0 \tag{11}$$

It follows from the expression (10) that the coefficient $a > 0$ whenever $\alpha > (\mu_h\sigma/(\mu_v+\eta))$.

Hence, by Theorem 4 in paper [19], system (1) undergoes a backward bifurcation if the inequality $\alpha > (\mu_h\sigma/(\mu_v+\eta))$ holds. ■

5 Uniformly persistence

The purpose of this section is to discuss the uniformly persistence of system (1). Before giving the main theorem, we first need the following Lemma.

Lemma 6 *For the following system*

$$\begin{cases} S_h'(t) = b_1 - \mu_hS_h(t), \\ M_v'(t) = b_2S_v(t) - (\mu_v+\eta)M_v(t), \\ S_v'(t) = \eta M_v(t) - \mu_vS_v(t) - \alpha S_v^2(t), \end{cases} \tag{12}$$

there exists a unique positive equilibrium $E^*(S_h^0, M_v^0, S_v^0)$, and it is globally asymptotically stable.

By direct computation, the existence of the unique positive equilibrium $E^*(S_h^0, M_v^0, S_v^0)$ is obvious, and the globally asymptotically stability can be obtained by constructing Lyapunov functional and using LaSalle's invariance principle, here we omit it.

Theorem 7 *If $R_0 > 1$, then system (1) is uniformly persistent, i.e., there is a positive constant such that every positive solution $(S_h(t), I_h(t), M_v(t), S_v(t), I_v(t))$of (1) satisfies that*

$$\lim_{t\to+\infty}\inf S_h(t) \geq \varepsilon, \lim_{t\to+\infty}\inf I_h(t) \geq \varepsilon, \lim_{t\to+\infty}\inf M_v(t) \geq \varepsilon, \lim_{t\to+\infty}\inf S_v(t) \geq \varepsilon, \lim_{t\to+\infty}\inf I_v(t) \geq \varepsilon.$$

Proof. Define

$$X = \{(S_h, I_h, M_v, S_v, I_v) : S_h \geq 0, M_v \geq 0, S_v > 0, I_h \geq 0, I_v \geq 0\},$$
$$X_0 = \{(S_h, I_h, M_v, S_v, I_v) \in X : I_h > 0, I_v > 0\},$$

we have

$$\partial X_0 = X \backslash X_0 = \{(S_h, I_h, M_v, S_v, I_v) \in X : I_h I_v = 0\}.$$

From system (1), it follows that X and X_0 are positively invariant, and ∂X_0 is relatively closed in X. Set

$$M_\partial = \{(S_h(0), I_h(0), M_v(0), S_v(0), I_v(0)) \in \partial X_0 : (S_h(t), I_h(t), M_v(t), S_v(t), I_v(t)) \\ satisfies\ (1)\ and\ (S_h(t), I_h(t), M_v(t), S_v(t), I_v(t)) \in \partial X_0,\ \forall t \geq 0\}.$$

We now show that

$$M_\partial = \left\{(S_h, I_h, M_v, S_v, I_v) \in X : I_h^2 + I_v^2 = 0\right\}. \tag{13}$$

Assume $(S_h(0), I_h(0), M_v(0), S_v(0), I_v(0)) \in M_\partial$. It suffices to show that $I_h^2(t) + I_v^2(t) = 0$ for all $t \geq 0$. Suppose not, then there exists at least a point $(S_h(0), I_h(0), M_v(0), S_v(0), I_v(0)) \in M_\partial$ such that $I_h(0) > 0$ or $I_v(0) > 0$.
Case (I) If $I_v(0) = 0$ and $I_h(0) > 0$, then it is clear from the second equation of (1)

$I_h(t) \geq I_h(0)e^{-(\mu_h+\delta_h)t} > 0$for all $t > 0$.

From the fourth equation of system (1) that

$S_v'(t) \geq S_v(t)\left[-(\mu_v + \beta_2 I_h(t)) - \alpha S_v(t)\right]$ for all $t > 0$.

Then from $S_v(0) > 0$, we have

$S_v(t) \geq S_v(0)\exp\int_0^t \left[-(\mu_v + \beta_2 I_h(t)) - \alpha S_v(t)\right] ds > 0$ for all $t > 0$.

Consequently, by the fifth equation of system (1) we have$I_v'(t) > -\mu_v I_v(t)$ for all $t > 0$. Thus $I_v(t) > 0$ for all $t > 0$.
Case (II) If $I_v(0) > 0$ and $I_h(0) = 0$.

Similar to the inference of Case (I), we can easily show that $I_h(t) > 0$ for all $t > 0$if $I_v(0) > 0$ and $I_h(0) = 0$. Here we omit it.

This shows that $(S_h(t), I_h(t), M_v(t), S_v(t), I_v(t)) \notin \partial X_0$. Hence $(S_h(0), I_h(0), M_v(0), S_v(0), I_v(0)) \notin M_\partial$ which leads to a contradiction. It indicates that $M_\partial \subseteq \{(S_h, 0, M_v, S_v, 0) : S_h \geq 0, M_v > 0, S_v > 0\}$. This proves (13).

It is clear that E_0 is the unique equilibrium in M_∂. We now show that E_0 repels the solution in X_0.

Suppose $(S_h(0), I_h(0), M_v(0), S_v(0), I_v(0))$ is a solution of system (1) with $(S_h(0), I_h(0), M_v(0), S_v(0), I_v(0)) \in X_0$. We now claim that $\lim_{t\to+\infty} \sup \max\{I_h(t), I_v(t)\} > \delta$.

Suppose, for the sake of contradiction, that there is a $T > 0$ such that $I_h(t) \leq \delta, I_v(t) \leq \delta$ for $t \geq T$. Then by the first, third and fourth equations of system (1), we have

$$\begin{cases} S_h'(t) \geq b_1 - (\mu_h + \beta_1\delta)S_h(t), \\ M_v'(t) \geq b_2 S_v(t) - (\mu_v + \eta)M_v(t) \\ S_v'(t) \geq \eta M_v(t) - (\mu_v + \beta_v\delta)S_v(t) - \alpha S_v^2(t) \end{cases}$$

For $t > T$. Consider the following systems:

$$\begin{cases} \bar{S}_h'(t) = b_1 - (\mu_h + \beta_1\delta)S_h(t) \\ \bar{M}_v'(t) = b_2 S_v(t) - (\mu_v + \eta)M_v(t) \\ \bar{S}_v'(t) = \eta M_v(t) - (\mu_v + \beta_v\delta)S_v(t) - \alpha S_v^2(t) \end{cases} \tag{14}$$

By Lemma 6, we can restrict δ to be small enough such that (14) admits a positive equilibrium $(\bar{S}_h^0, \bar{M}_v^0, \bar{S}_v^0)$,where

$$\bar{S}_h^0 = \frac{b_1}{\mu_h + \beta_1\delta}, \bar{M}_v^0 = \frac{b_2[b_2\eta - (\mu_v + \beta_2\delta)(\mu_v + \eta)]}{\alpha(\mu_v + \eta)^2}, \bar{S}_v^0 = \frac{b_2\eta - (\mu_v + \beta_2\delta)(\mu_v + \eta)}{\alpha(\mu_v + \eta)}. \tag{15}$$

Furthermore, the unique positive equilibrium $(\bar{S}_h^0, \bar{M}_v^0, \bar{S}_v^0)$ of system (14) is globally asymptotically stable.

From (15), then we can choose θ small enough such that

$$\bar{S}_h^0 > S_h^0 - \theta, \bar{M}_v^0 > M_v^0 - \theta, \bar{S}_v^0 > S_v^0 - \theta, \tag{16}$$

where $S_h^0 = b_1/\mu_h, M_v^0 = b_2\sigma/(\alpha(\mu_v+\eta)^2), S_v^0 = \sigma/(\alpha(\mu_v+\eta))$..

By (16) and the standard comparison principle, there is a $\tau > 0$ such that $S_h(t) \geq S_h^0 - \theta$, $M_v(t) \geq M_v^0 - \theta, S_v(t) \geq S_v^0 - \theta$for $t > T + \tau$. Consequently, for $t > T + \tau$, we have

$$\begin{cases} I_h'(t) \geq \beta_1 I_v(t)(S_h^0 - \theta) - (\mu_h + \delta_h)I_h(t), \\ I_v'(t) \geq \beta_2 I_h(t)(S_v^0 - \theta) - \mu_v I_v(t). \end{cases}$$

Consider auxiliary system

$$\begin{cases} \hat{I}_h'(t) = \beta_1 \hat{I}_v(t)(S_h^0 - \theta) - (\mu_h + \delta_h)\hat{I}_h(t), \\ \hat{I}_v'(t) = \beta_2 \hat{I}_h(t)(S_v^0 - \theta) - \mu_v \hat{I}_v(t). \end{cases} \tag{17}$$

The coefficient matrix $\hat{J}$ of the right hand of (17) is defined by

$$\hat{J} = \begin{pmatrix} -(\mu_h + \delta_h) & \beta_1(S_h^0 - \theta) \\ \beta_2(S_v^0 - \theta) & -\mu_v \end{pmatrix}.$$

The characteristic equation of the above matrix is $\lambda^2 + q_1\lambda + q_2 = 0$, where

$$q_1 = \mu_h + \delta_h + \mu_v > 0, \quad q_2 = \mu_v(\mu_h + \delta_h) - \beta_1\beta_2(S_h^0 - \theta)(S_v^0 - \theta).$$

When $R_0 > 1$, we have $\beta_1\beta_2 S_h^0 S_v^0 - \theta > \mu_v(\mu_h + \delta_h)$. Therefore, we can choose $\theta > 0$ small enough such that $\beta_1\beta_2(S_h^0 - \theta)(S_v^0 - \theta) > \mu_v(\mu_h + \delta_h)$, that is $q_2 < 0$, thus there exists and only exists one positive eigenvector λ_m of $\hat{J}$. By the theory of linear system, it is easy to see that any positive solution of (16) tends to infinity. Then by the standard comparison principle, we have $I_h(t) \to +\infty, I_v(t) \to +\infty$ as $t \to +\infty$. This contradicts$I_h(t) \leq \delta, I_v(t) \leq \delta$ for $t \geq T$. This proves $\lim\limits_{t\to+\infty} \sup \max\{I_h(t), I_v(t)\} > \delta$.

Hence, $W^S(E_0) \cap X_0 \neq \emptyset$. Clearly, every forward orbit in M_∂ converges to E_0. By Theorem 4.6 of [20] we are able to conclude that the system (1) is uniformly persistent with respect to $(X_0, \partial X_0)$. This completes the proof of Theorem 7. ■

6 Numerical simulations

Some numerical simulations are presented in this section to support the analytical results and show the effect of stage structure on the spread of the vector-borne diseases.

Example 1 *(the existence of back bifurcation) Take* $b_1 = 0.9861, b_2 = 0.2311, \beta_1 = 0.6815, \beta_2 = 0.2347, \eta = 0.5949, \delta_h = 0.3049, \mu_h = 0.5249, \mu_v = 0.1333$ *then* $a_1 < 0$*, and choose* α *as bifurcation parameter, the backward bifurcation occurs (see Fig.1 and Fig.2). Bifurcation diagram Fig. 1 shows the change in equilibrium level of infective hosts with the change in* α *but in this figure change in infective hosts is shown with respect to corresponding value of* R_0*. From Fig.1, it is clear that if the reproductive number* R_0 *satisfies that* $R_c < R_0 < 1$*, then backward bifurcation occur for system (1), where there exists two endemic equilibria and the smaller one is unstable and the larger one is stable. This verifies Theorems 4.*

For the above same bifurcation data, bifurcation diagram Fig. 2 shows the equilibrium level of infective hosts is plotted against α*. From this bifurcation diagram, it can see that with increase in* α*, the equilibrium level of infective hosts decreases, and exceeding a threshold, the backward bifurcation occurs, and multiple infective equilibria occur. Fig. 2 also verifies the conclusions in Theorem 5.*

By Theorem 4, it is clear that the reproduction number R_0 *and the critical value* R_c *which satisfies* $a_1^2 - 4a_2a_0 = 0$ *are threshold condition that determine the existence of disease and disease-free equilibrium. Therefore, the quantities* R_0*and* R_c *can provide us more important information for analyzing the effect of stage structure on the spread of the vector–host disease. In the following, we will give two examples to illustrate it.*

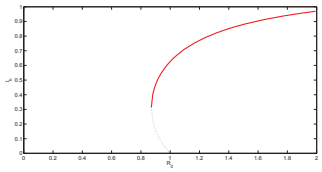

Figure 1: The backward bifurcation diagram of system (1) with respect to R_0 for the parameter values given in Example 1.

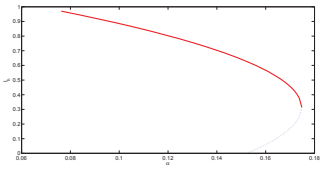

Figure 2: The backward bifurcation diagram of system (1) with respect to α for the parameter values given in Example 1.

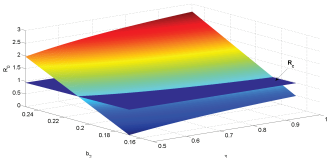

Figure 3: Surface plot of R_0 as a function of b_2, η for the parameters given in Example 2.

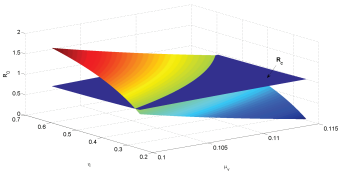

Figure 4: Surface plot of R_0 as a function of η, μ_v for the parameters given in Example 3.

Example 2 *Take $\mu_v = 0.1333$ and keep all the other parameter values fixed in Example 1 except b_2 and η which are chosen as parameters. Fig. 3 gives the change in value of R_0 and R_c with the change in b_2 and η. From Fig.3, it is clear that R_0 increases with the increase of b_2 and η. And $R_0 < R_c < 1$ if b_2 and η are close to 0.16 and 0.5 respectively. In this case, by Theorem 4 there only exists the disease-free equilibrium and by Theorem 2, it is globally asymptotically stable, that is the disease is tend to be extinct. However, as b_2 and η become large then we have $R_0 \geq R_c$ and even $R_0 > 1$. By Theorem 4, there exists at least one disease-equilibrium and by Theorem 7 the disease is persistent when $R_0 > 1$. This suggests that b_2 and η will cause the spread of the vector-borne disease.*

Example 3 *Take $b_2 = 0.1689$ and keep all the other parameter values fixed in Example 1 except η and μ_v which are chosen as parameters. Fig. 4 gives the change in value of R_0 and R_c with the change in η and μ_v. From Fig.4, it is clear that R_0 increases with the increase of η and the decrease of μ_v. And $R_0 < R_c < 1$ if η and μ_v are close to 0.1 and 0.115, respectively, then by Theorem 4 there only exists the disease-free equilibrium and by Theorem 2, it is globally asymptotically stable, that is, the disease tends to be extinct. However, as η becomes large and μ_v becomes small then we have $R_0 \geq R_c$ and even $R_0 > 1$. By Theorem 4, there exists at least one disease-equilibrium and by Theorem 7 the disease is persistent when $R_0 > 1$. This suggests that η will cause the spread of the vector-borne disease while μ_v will reduce the spread of the vector-borne disease.*

Remark 8 *Example 2-3 indicate that the reproduction number R_0 increases with the increase of b_2 and η while decrease with the increase of μ_v. Therefore, to prevent or control vector-borne disease, it is significant to take some effective strategies at larvae stage, such as use of physical strategies, use of pesticides, biological control and so on to reduce the birth rate b_2 and the conversation rate η, and increase the death rate μ_v of the immature vector.*

7 Conclusion and discussion

In this section, we mainly formulate and analyze a vector-borne disease model with state structure. Qualitative analysis of model (1) with stage structure and crowing effect of the susceptible vectors reveals that if the basic reproduction number $R_0 < 1$, the phenomena of backward bifurcation in the system occurs. The presence of a backward bifurcation has practically important consequences for the control of infectious diseases, where the endemic equilibria and the disease-free equilibrium can coexist. By Theorem 5, we show that the existence of a backward bifurcation depends on parameters of the model, and can be regulated by the crowing effect rate of the susceptible vector population. In Theorem 2, we show that the disease-free equilibrium E_1 is globally asymptotically stability if $R_0 \leq 1$ and the immature vector population size $M_v(t)$ is not larger than the immature vector equilibrium level M_v^0. The uniformly persistence of system (1) when $R_0 > 1$ is shown by Theorem 7. Finally, to illustrate the impact of stage structure on the spread of host-vector diseases, also to support the analytical results, some numerical examples are provided to system (1). It indicates that the growth

rate b_2 and the conversion rate η from immature vectors to mature vectors will cause the spread of vector-borne disease, while the death rate μ_v will reduce the vector-borne disease spread. Therefore taking some effective strategies at immature stage, such as use of physical strategies, use of pesticides, biological control and so on is necessary to vector-host disease prevention and control.

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References

[1] K-B. G. Scholth, S. Adkins, H. Czosnek, P. Palukaitis, E. Jacquot, T. Hohn, B. Hohn, K. Saunders, T. Candresse, P. Ahlquist,C. Hemenway and G. Foster. Top 10 plant viruses in molecular plant pathology. *Molecular Plant Pathology*, 12(2011):938–954.

[2] R. M. Giblin-Davis. Interactions of nematodes with insects, Nematode Interactions. Chapman and Hall, London, UK. 1993.

[3] W. G. Aiello, H. I. Freedman and J. Wu. Analysis of a model representing stage-structured population growth with state-dependent time delay. *IAM J. Appl. Math.*, 52(1992):855–869.

[4] Y. Cao, J. Fan and T. C. Gard. The effects of state-dependent time delay on a stage-structured population growth model. *Nonlinear Anal-real.*, 19(1992):95–105.

[5] X. Song and L. Chen. Modelling and analysis of a single species system with stage structure and harvesting. *Math. Comput. Model.*, 36(2002):67-82.

[6] Y. Xiao and Y. Chen. Analysis of a SIS epidemic model with stage structure and a delay. *Commun. Nonlinear Sci. Numer. Simul.*, 6(2001):35-39.

[7] J. Jia and Q. Li. Qualitative analysis of an SIR epidemic model with stage structure. *Appl. Math. Comput.*, 193(2007):106-115.

[8] J. X. Velasco-Hernandez. An epidemiological model for the dynamics of Chagas disease. *BioSystems*, 26(1991):127-134.

[9] J. M. Hyman, J. Li and E. A. Stanley. The differential infectivity and staged progression models for the transmission of HIV. *Math. Biosci.*, 155(1999):77-109.

[10] L. Cai, X. Li and M. Ghosh. Global stability of a stage-structured epidemic model with a nonlinear incidence. *Appl. Math. Comput.*, 214(2009):73-82.

[11] C. Wu and P. Weng. Stability analysis of a stage structured SIS model with general incidence rate. *Nonlinear. Anal-real.*, 11(2010):1826-1834.

[12] X. Zhang, H. Huo, X.Sun and Q. Fu. The differential susceptibility SIR epidemic model with stage structure and pulse vaccination. *Nonlinear. Anal-real.*, 11(2010):2634-2646.

[13] T. Zhang, J. Liu and Z. Teng. Stability of Hopf bifurcation of a delayed SIRS epidemic model with stage Structure. *Nonlinear. Anal-real.*, 11(2010):293-306.

[14] X. Shi, J. Cui and X. Zhou. Stability and Hopf bifurcation analysis of an eco-epidemic model with a stage structure. *Nonlinear. Anal-real.*, 74(2011):1088-1106.

[15] P. Klepac and H. Caswell. The stage-structured epidemic: linking disease and demography with a multi-state matrix approach model. *Theor. Ecol.*, 4(2011):301-319.

[16] Y. Liu, S. Gao and Y. Luo. Impulsive epidemic model with differential susceptibility and stage structure. *Appl. Math. Model.*, 36(2012):370-378.

[17] World health organization. Frequently Asked Questions Scrub Typhus. 2008.

[18] B. G. Zhao, K. Futai, R. Jack, J. R. Sutherland and Y. Takeuchi. Pine Wilt Disease. Springer, New York. 2008.

[19] C. Castillo-Chavez and B. Song. Dynamical models of tuberculosis and their application. *Math. Biosci. Eng.*,1(2004):361-404.

[20] H. R. Thieme. Persistence under relaxed point-dissipativity with an application to an epidemic model. *SIAM J. Math. Anal.*,24(1993):407-435.