Advances in Difference Equations
a SpringerOpen Journal

RESEARCH Open Access

Impact of predator on the host–vector disease model with stage structure for the vector

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Abstract

In this paper, we propose a host–vector–predator model with stage structure for the vector to explore the impact of biological control agents on host–vector dynamics and disease control. Here the total vector population is divided into two physiological subclasses which are immature and mature subclasses. Holling type II functional response is used to portray the interactions between vectors and predators. Stability analysis of the equilibria demonstrates that the basic reproduction number gives the threshold condition determining the persistence and extinction of the disease. Furthermore, the phenomenon of Hopf bifurcation occurs when predators are introduced. The stability of limit cycle arising from a Hopf bifurcation is rigorously investigated. Finally, numerical simulations are given to show the validity of analytical results, and the comparative results of disease dynamics with and without predators.

Keywords: Vector–host diseases; Disease control; Stage structure; Hopf bifurcation; Persistence

1 Introduction

Host–vector diseases, such as malaria, dengue fever, tobacco mosaic virus, pine wilt disease, and so forth, are transmitted to the host population (e.g. humans and plants) by biological agents (arthropods) called vectors who carry the disease without getting themselves. Recent analysis demonstrates that the impact of environmental change and frequent occurrence of natural disasters on complex host–parasite relationships for vector-borne diseases is clear [1, 2]. Vector-borne diseases remain a serious threat to humans, livestock, and plants, and thus the control of such diseases is of great economic and healthcare concern.

Vector control is one of the few proven ways to reduce transmission of many vector-borne diseases. The mostly adopted methods to control vectors include physical control, such as burning, clearness, burying, bednets, and so on, spraying insecticides [3], vaccination [4, 5], environment control [6]. As a newly proposed method of vector-borne diseases, Sterile Insect Technique (SIT) [7] has obtained some applications in vector-borne disease control though it is still in its infancy.

One potential approach to vector control is to use biological enemies (biocontrol agents) of the vectors. Biological control shows no environmental contamination and vector resistance, less maintenance costs, and more safety compared with insecticide, environment

control, and Sterile Insect Technique, respectively. Moreover, it will be conducive to ecological diversity and environment protection. Biological agents have obtained successful application in controlling a variety of host–vector diseases. For human diseases, entomogenous fungi were adopted as promising biopesticides for sleeping sickness and tick control [8, 9]. Predators were introduced to control many diseases such as dengue fever, malaria, Lyme disease and tick disease [10–14]. Several recent studies have shown that predators have caused decline in local cases of malaria in India [15, 16] and dengue fever in Vietnam and Thailand [17, 18]. In addition, biological agents were used in tree diseases such as pine wilt disease in Japan and China [19, 20], as well as in crop diseases such as the cassava mosaic virus in sub-Saharan Africa and the tomato leaf curl virus in India [21, 22].

However, biological control of vectors is still only partially understood compared with biological control of herbivorous pests, which has long been established and widely applied in pest management aiming to directly reduce pest populations by pest enemies [23, 24], and many good research results of interactions between the biological agents (predators) and the pests (prey) have been stated in some papers (see, for example, [25–27] etc.). This is primarily caused by the complexity of interactions among the host, vector, and predator populations. The predators affect the spread of pathogen and the interactions between the hosts and the vectors by preying on the vectors, and the interactions between the hosts and the vectors, between the pathogen and the hosts will in turn impact the vector–predator dynamics.

Mathematical modeling provides strong support for understanding the complex dynamics of epidemic and ecological systems as well as in decision making process on disease prevention and disease control. Different mathematical models were proposed in [28, 29] to explore the effect of Wolbachia, which shortened the lifespan of the vector (the infected mosquito Sedes aegypti), on the transmission of dengue. Moore et al. [30] first adopted a host–vector–predator mathematical model to investigate how the predators affect the persistence or extinction of vector-borne diseases. In [31], authors considered the disease dynamics for a class of host–vector models with the effect of predators, and the nonlinear dynamics caused by predators. Okamoto and Amarasek [32] gave the comparative analysis of three classes of biocontrol agents: pathogen, predator/parasitoid, and competitor of the vector controlling diseases by reducing vector densities. However, all the above studies focused on the effect of predators on vector-borne disease control without stage structure.

In reality, individuals in a population may grow through several stages of physiology, such as immature and mature. Some epidemic models with physiology stage for the hosts have been investigated recently (see [33–38]). However, for vector-borne diseases, there are some kinds of vector–host diseases which are only spread among hosts by the immature vectors such as cysticercosis and Scrub typhus [39]. Some infectious diseases, such as malaria, dengue fever, West Nile virus, pine wilt disease [40], and so on, are spread only by the adult vectors. Consequently, to study realistically the host–vector disease transmission in a host population, we must consider the model to include stage structure for the vectors, each stage being homogeneous.

Motivated by the above discussions, in this paper we develop a host–vector–predator model with stage structure for the vectors to ask how the interactions between the vectors and the biocontrol agents indirectly reduce the prevalence of a vector-borne disease in the host population, how the predators cause the change in the host–pathogen dynamics,

and how the stage structure impacts the disease dynamics with and without predators. The remaining part of this paper is organized as follows: In Sect. 2, we mainly formulate our model. In Sect. 3, we establish the existence and stability results of the disease and disease-free equilibria of the model, and the phenomenon of Hopf bifurcation is rigorously studied. In Sect. 4, numerical simulations are given to show the validity of our results and to compare the disease dynamics with and without predators. The paper ends with a conclusion.

The contributions of this paper can be summed as follows: (1) The coupled host–vector–predator model with stage structure is considered, where the total vector population is divided into immature and mature subclasses. (2) We analyze the dynamics of the model by Routh–Hurwitz criteria and bifurcation theory. Theoretical results show that the introduction of predators leads to the occurrence of Hopf bifurcation. Meanwhile, the introduction of predators is greatly helpful to disease control. The effect of stage structure on disease transmission is also investigated. (3) Finally, numerical simulations are given to show the validity of analytical results, and the comparative results of disease dynamics with and without predators are presented.

2 Model description

To model the interactions among the host population, the vector population with stage structure, and the predator population, we divide the total vector population into two stage groups, immature vectors $M_v(t)$ and mature vectors $N_v(t)$, and assume that only mature vectors have the ability to transmit the disease to host populations. Therefore $N_v(t)$ can be divided into two subclasses, susceptible and infectious, with densities denoted by $S_v(t)$ and $I_v(t)$, respectively, so that $N_v(t) = S_v(t) + I_v(t)$. It is assumed that the virus in vectors does not cause the death of vectors and does not influence the propagation of vectors. The birth rate of the immature vector is assumed to be proportional to the density of mature vectors with proportionality parameter b_2. We assume that mature vector populations experience growing effects with rate parameter α. We introduce the predator population for preying on the vectors which transmits the disease among hosts, and the interactions between the predators and the vectors are portrayed by Holling type II functional response, which is $h_2 N_v(t)P_2(t)/(1 + a_2 N_v(t))$, where h_2 and a_2 are the capturing rate (or the attacking rate) and the satiety rate of the predator $P_2(t)$, respectively, and $P_2(t)$ denotes the predator population of the mature vector population. The total host population is split into susceptible and infectious subclasses, with sizes denoted by $S_h(t)$ and $I_h(t)$, respectively.

Using the above assumptions, we obtain the following host–vector–predator dynamical model:

$$
\begin{cases}
\frac{dS_h(t)}{dt} = b_1 - \beta_1 S_h(t) I_v(t) - \mu_h S_h(t), \\
\frac{dI_h(t)}{dt} = \beta_1 S_h(t) I_v(t) - (\mu_h + \delta_h) I_h(t), \\
\frac{dM_v(t)}{dt} = b_2 (S_v(t) + I_v(t)) - (\mu_{v1} + d) M_v(t), \\
\frac{dS_v(t)}{dt} = d M_v(t) - \beta_2 S_v(t) I_h(t) - \mu_{v2} S_v(t) \\
\qquad - \alpha S_v(t)((S_v(t) + I_v(t)) - \frac{h_2 S_v(t) P_2(t)}{1 + a_2((S_v(t) + I_v(t))}, \\
\frac{dI_v(t)}{dt} = \beta_2 S_v(t) I_h(t) - \mu_{v2} I_v(t) \\
\qquad - \alpha I_v(t)((S_v(t) + I_v(t)) - \frac{h_2 I_v(t) P_2(t)}{1 + a_2((S_v(t) + I_v(t))}, \\
\frac{dP_2(t)}{dt} = \frac{\gamma_2 h_2 ((S_v(t) + I_v(t)) P_2(t)}{1 + a_2((S_v(t) + I_v(t))} - e_2 P_2(t),
\end{cases}
\tag{1}
$$

with initial conditions

$$S_h(0) \geq 0, \qquad I_h(0) \geq 0, \qquad M_v(0) \geq 0,$$
$$S_v(0) \geq 0, \qquad I_v(0) \geq 0, \qquad P_2(0) \geq 0,$$

where b_1 is the recruitment rate of the host populations. μ_h is the natural death rate of the infected host population, μ_{v1} and μ_{v2} are respectively the natural death rate of the immature and mature vectors. δ_h is the disease-caused death rate of the infected hosts. d is the conversion rate from immature vectors to mature vectors. β_1 and β_2 are respectively the rate of biting from susceptible hosts to infected vectors and susceptible vectors to infected hosts. γ_2 and e_2 respectively denote the conversion factor and the natural mortality of the predator populations.

Without considering the effect of predators on host–vector disease model, (1) can be reduced as follows:

$$\begin{cases} \frac{dS_h(t)}{dt} = b_1 - \beta_1 S_h(t) I_v(t) - \mu_h S_h(t), \\ \frac{dI_h(t)}{dt} = \beta_1 S_h(t) I_v(t) - (\mu_h + \delta_h) I_h(t), \\ \frac{dM_v(t)}{dt} = b_2(S_v(t) + I_v(t)) - (\mu_{v1} + d) M_v(t), \\ \frac{dS_v(t)}{dt} = dM_v(t) - \beta_2 S_v(t) I_h(t) - \mu_{v2} S_v(t) \\ \qquad - \alpha S_v(t)((S_v(t) + I_v(t)), \\ \frac{dI_v(t)}{dt} = \beta_2 S_v(t) I_h(t) - \mu_{v2} I_v(t) \\ \qquad - \alpha I_v(t)((S_v(t) + I_v(t)), \end{cases} \tag{2}$$

with initial conditions

$$S_h(0) \geq 0, \qquad I_h(0) \geq 0, \qquad M_v(0) \geq 0, \qquad S_v(0) \geq 0, \qquad I_v(0) \geq 0.$$

3 Dynamics of model (1)

In this section, we mainly study the dynamics of model (1). As preliminary results, first we give the equilibria of (1).

Lemma 3.1 *The equilibria for model* (1) *are as follows.*

(i) *The boundary equilibrium* $E_0(b_1/\mu_h, 0, 0, 0, 0, 0)$ *always exists.*

(ii) *The predator-absent disease-free equilibrium* $E_1(S_h^0, 0, M_v^0, S_v^0, 0, 0)$ *exists if* $\sigma > 0$, *where*

$$S_h^0 = \frac{b_1}{\mu_h}, \qquad M_v^0 = \frac{b_2 S_v^0}{\mu_{v1} + d}, \qquad S_v^0 = \frac{\sigma}{(\mu_{v1} + d)\alpha}, \qquad \sigma = b_2 d - \mu_{v2}(\mu_{v1} + d).$$

(iii) *The predator-absent disease equilibrium* $E_2(S_h^*, I_h^*, M_v^*, S_v^*, I_h^*, 0)$ *exists if* $\sigma > 0$ *and* $R_{01} > 1$, *where*

$$S_h^* = \frac{b_1(\mu_{v2} + \alpha S_v^0 + \beta_2 I_h^*)}{\mu_h(\mu_{v2} + \alpha S_v^0 + \beta_2 I_h^*) + \beta_1 \beta_2 S_v^0 I_h^*},$$
$$I_h^* = \frac{\mu_h(\mu_{v2} + \alpha S_v^0)(R_{01} - 1)}{\beta_2(\mu_h + \beta_1 S_v^0)},$$

$$M_v^* = \frac{b_2 S_v^0}{(\mu_{v1}+d)}, \qquad S_v^* = \frac{(\mu_{v2}+\alpha S_v^0)S_v^0}{\mu_{v2}+\alpha S_v^0+\beta_2 I_h^*}, \qquad I_v^* = \frac{\beta_2 S_v^0 I_h^*}{\mu_{v2}+\alpha S_v^0+\beta_2 I_h^*},$$
$$S_v^0 = \frac{\sigma}{(\mu_{v1}+d)\alpha}, \qquad R_{01} = \frac{\beta_1\beta_2 S_h^0 S_v^0}{(\mu_h+\delta_h)(\mu_{v2}+\alpha S_v^0)}.$$

(iv) *The predator-present disease-free equilibrium* $E_3(S_h^1, 0, M_v^1, S_v^1, 0, P_2^1)$ *exists if* $\sigma > 0$ *and* $R_1 > 1$, *where* σ *is given in case* (*i*), *and*

$$S_h^1 = S_h^0 = \frac{b_1}{\mu_h}, \qquad M_v^1 = \frac{b_2 S_v^1}{\mu_{v1}+d}, \qquad S_v^1 = \frac{e_2}{\gamma_2 h_2 - a_2 e_2},$$
$$P_2^1 = \frac{\alpha(S_v^0 - S_v^1)(1+a_2 S_v^1)}{h_2}, \qquad R_1 = \frac{\sigma(\gamma_2 h_2 - a_2 e_2)}{(\mu_{v1}+d)\alpha e_2}.$$

(v) *The predator-present disease equilibrium* $E_4(\hat{S}_h, \hat{I}_h, \hat{M}_v, \hat{S}_v, \hat{I}_v, \hat{P}_2)$ *exists if* $\sigma > 0$, $R_1 > 1$, *and* $R_{02} > 1$, *where* σ *and* R_1 *are given in cases* (i) *and* (iv), *respectively, and*

$$\hat{S}_h = \frac{b_1(\mu_{v2}+\alpha S_v^1+\beta_2\hat{I}_h+\frac{h_2 P_2^1}{1+a_2 S_v^1})}{\mu_h(\mu_{v2}+\alpha S_v^1+\beta_2\hat{I}_h+\frac{h_2 P_2^1}{1+a_2 S_v^1})+\beta_1\beta_2 S_v^1\hat{I}_h},$$
$$\hat{I}_h = \frac{\mu_h(\mu_{v2}+\alpha S_v^1+\frac{h_2 P_2^1}{1+a_2 S_v^1})(R_{02}-1)}{\beta_2(\mu_h+\beta_1 S_v^1)},$$
$$\hat{M}_v = \frac{b_2 S_v^1}{(\mu_{v1}+d)}, \qquad \hat{S}_v = \frac{(\mu_{v2}+\alpha S_v^1+\frac{h_2 P_2^1}{1+a_2 S_v^1})S_v^1}{\mu_v+\alpha S_v^1+\beta_2\hat{I}_h+\frac{h_2 P_2^1}{1+a_2 S_v^1}},$$
$$\hat{I}_v = \frac{\beta_2 S_v^1\hat{I}_h}{\mu_{v2}+\alpha S_v^1+\beta_2\hat{I}_h+\frac{h_2 P_2^1}{1+a_2 S_v^1}}, \qquad S_v^1 = \frac{e_2}{\gamma_2 h_2 - a_2 e_2},$$
$$\hat{P}_2 = P_2^1 = \frac{\alpha(S_v^0 - S_v^1)(1+a_2 S_v^1)}{h_2},$$
$$R_{02} = \frac{\beta_1\beta_2 S_h^0 S_v^1}{(\mu_h+\delta_h)(\mu_{v2}+\alpha S_v^0)}.$$

The equilibria E_i $(i = 0, 1, 2, 3, 4)$ of Lemma 3.1 can be obtained by direct computation. Here we omit it.

According to the next generation matrix proposed in [41, 42], R_{01} and R_{02} given in Lemma 3.1 are the basic reproduction numbers of system (1) and system (2), respectively. It is clear that $R_{02} < R_{01}$ if $R_1 > 1$ (where the condition $R_1 > 1$ ensures $S_v^0 > S_v^1$, therefore the equilibrium density of the predator is larger than zero). That is, the basic reproduction number has been lessened by introducing the predators.

3.1 Local stability and existence of Hopf bifurcation for system (1)

In this subsection, we shall investigate the local properties of the equilibria and Hopf bifurcation for system (1).

Theorem 3.1 *For the predator-absent equilibria* E_0, E_1, *and* E_2 *of system* (1), *we have*:

(i) *The equilibrium* E_0 *is a saddle point, which is unstable.*

(ii) *If* $R_1 < 1$, $\gamma_2 h_2 - a_2 e_2 > 0$, *and* $R_{01} < 1$, *then the predator-absent disease-free equilibrium* E_1 *is locally asymptotically stable.*

(iii) *If* $R_1 < 1$, $\gamma_2 h_2 - a_2 e_2 > 0$, *and* $R_{01} > 1$, *then the predator-absent disease equilibrium* E_2 *is locally asymptotically stable.*

Here E_0, E_1, and E_2 are given in Lemma 3.1.

Proof By some mathematical deductions and using the Routh–Hurwitz criteria, we can obtain the proof of Theorem 3.1. Here we omit it. □

By Theorem 3.1, we obtain the following corollary.

Corollary 3.1 *The equilibria of system* (2) *are as follows.*

(i) *The boundary equilibrium* $E_0(b_1/\mu_h, 0, 0, 0, 0)$ *is a saddle point, which is unstable.*

(ii) *If* $R_{01} < 1$, *then the disease-free equilibrium* $E_1(S_h^0, 0, M_v^0, S_v^0, 0)$ *is locally asymptotically stable.*

(iii) *If* $R_{01} > 1$, *then the disease equilibrium* $E_2(S_h^*, I_h^*, M_v^*, S_v^*, I_h^*)$ *is locally asymptotically stable,*

where S_h^0, M_v^0, S_v^0, S_h^*, I_h^*, M_v^*, S_v^*, *and* I_h^* *are given in Lemma* 3.1.

From Corollary 3.1, the basic reproduction number R_{01} of system (2) is the threshold to decide the disease persistence and extinction without predators.

When $R_1 > 1$, system (1) has the predator-present equilibria E_3 and E_4 given in Lemma 3.1. In the following, we will give the stability results of E_3 and E_4.

Theorem 3.2 *For the predator-present equilibria* E_3 *and* E_4 *of system* (1), *we have*:

(i) *The predator-present disease-free equilibrium* E_3 *is locally asymptotically stable if* $R_{02} < 1$ *and* $C_1C_2 - C_3 > 0$, *in which case the vector–host disease can be eradicated when a predator population* P_2 *is introduced. When* $C_1C_2 - C_3 = 0$ *and* $R_{02} < 1$, *then system* (1) *undergoes a Hopf bifurcation at* E_3, *in which case, despite oscillations occurring between the predator and the vector, predation can eliminate the pathogen and the vector population is greater than zero.*

(ii) *The predator-present disease equilibrium* E_4 *is locally asymptotically stable if* $R_{02} > 1$ *and* $C_1C_2 - C_3 > 0$, *in which case the vector-borne diseases persist though the predators are introduced in the system. When* $C_1C_2 - C_3 = 0$ *and* $R_{02} > 1$, *then system* (1) *undergoes a Hopf bifurcation at* E_4, *in which case predation causes oscillations among host, vector, and predator populations, and the disease cannot be eliminated though predators are introduced in the system.*

Here

$$C_1 = \mu_{v1} + \mu_{v2} + d + 2\alpha S_v^1 + G_1, \tag{3}$$

$$C_2 = G_2G_3 + (\mu_{v1} + d)\left(\mu_{v2} + 2\alpha S_v^1 + G_1\right) - b_2 d, \tag{4}$$

$$C_3 = (\mu_{v1} + d)G_2G_3, \tag{5}$$

$$G_1 = \frac{h_2 P_2^1}{(1 + a_2 S_v^1)^2}, \tag{6}$$

$$G_2 = \frac{h_2 S_v^1}{1 + a_2 S_v^1}, \tag{7}$$

$$G_3 = \frac{\gamma_2 h_2 P_2^1}{(1 + a_2 S_v^1)^2}, \tag{8}$$

where S_v^1 and P_2^1 are given in Lemma 3.1.

Proof (i) By some mathematical deductions and rearrangement, the characteristic polynomial corresponding to the disease-free equilibrium E_3 can be rewritten as

$$f(\lambda) = (\lambda + \mu_h)\big(\lambda^2 + B_1\lambda + B_2\big)\big(\lambda^3 + C_1\lambda^2 + C_2\lambda + C_3\big) = 0, \tag{9}$$

where

$$B_1 = \mu_h + \delta_h + \mu_{v2} + \alpha S_v^1 + \frac{h_2 P_2^1}{1 + a_2 S_v^1},$$

$$B_2 = (\mu_h + \delta_h)(1 - R_{02})\left[\mu_{v2} + \alpha S_v^1 + \frac{h_2 P_2^1}{1 + a_2 S_v^1}\right],$$

C_1, C_2, and C_3 are given in Theorem 3.2.

Obviously, Eq. (9) has a real root $\lambda_1 = -\mu_h$, two roots $\lambda_{2,3}$ with negative real parts if $R_{02} < 1$, and the other three eigenvalues can be obtained by solving

$$\lambda^3 + C_1\lambda^2 + C_2\lambda + C_3 = 0. \tag{10}$$

For Eq. (10), three eigenvalues have negative real parts if they satisfy the Routh–Hurwitz criteria, such that $C_i > 0$, $i = 1, 2, 3$ and $C_1C_2 - C_3 > 0$. From the above expressions, we see that $C_1 > 0$, $C_3 > 0$, and if $C_1C_2 - C_3 > 0$, then we have $C_2 > 0$. Thus, E_3 is locally asymptotically stable if $R_{02} < 1$ and $C_1C_2 - C_3 > 0$.

Choose h_2 as a bifurcation parameter and let h^* be the solution of equation $C_1C_2 - C_3 = 0$, that is, $C_1(h_2^*)C_2(h_2^*) - C_3(h_2^*) = 0$, then Eq. (10) can be rewritten into

$$\big(\lambda^2 + C_2\big)(\lambda + C_1) = 0, \tag{11}$$

which has three roots

$$\lambda_1 = +i\sqrt{C_2}, \qquad \lambda_2 = -i\sqrt{C_2}, \qquad \lambda_3 = -C_1.$$

For $h_2 \in (h_2^* - \delta, h_2^* + \delta)$ $(\delta > 0)$, the roots are in general of following form:

$$\lambda_1(h_2) = w_1(h_2) + iw_2(h_2), \qquad \lambda_2(h_2) = w_1(h_2) - iw_2(h_2), \qquad \lambda_3(h_2) = -C_1(h_2).$$

Now we verify the transversality condition

$$\mathrm{Re}\left[\frac{d\lambda_i}{dh_2}\right]_{|h_2 = h_2^*} \neq 0, \quad i = 1, 2, 3. \tag{12}$$

Substituting $\lambda_1(h_2) = w_1(h_2) + iw_2(h_2)$ in (11) and calculating the derivative, we get

$$E_1(h_2)w_1'(h_2) - E_2(h_2)w_2'(h_2) + F(h_2) = 0,$$
$$E_2(h_2)w_1'(h_2) + E_1(h_2)w_2'(h_2) + G(h_2) = 0,$$

where

$$E_1(h_2) = 3w_1^2(h_2) + 2C_1(h_2)w_1(h_2) + C_2(h_2) - 3w_2^2(h_2),$$
$$E_2(h_2) = 6w_1(h_2)w_2(h_2) + 2C_1(h_2)w_2(h_2),$$
$$F(h_2) = w_1^2(h_2)C_1'(h_2) + w_1(h_2)C_2'(h_2) + C_3'(h_2) - w_2^2(h_2)C_1'(h_2),$$
$$G(h_2) = 2w_1(h_2)w_2(h_2)C_1'(h_2) + w_2(h_2)C_2'(h_2).$$

Since $E_1(h_2^*)F(h_2^*) + E_2(h_2^*)G(h_2^*) \neq 0$, thus we have

$$\operatorname{Re}\left[\frac{d\lambda_i}{dh_2}\right]_{|h_2=h_2^*} = w_1'(h_2)_{|h_2=h_2^*} = \frac{E_1(h_2^*)F(h_2^*) + E_2(h_2^*)G(h_2^*)}{E_1^2 + E_2^2} \neq 0, \quad i = 1, 2, \tag{13}$$

and

$$\operatorname{Re}\left[\frac{d\lambda_3}{dh_2}\right]_{h_2=h_2^*} = -\operatorname{Re}\left[\frac{dC_1(h_2)}{dh_2}\right]_{h_2=h_2^*} = -\frac{(1 + ae_2S_v^0)e_2}{\alpha\gamma_2(h_2^*)^2} \neq 0. \tag{14}$$

Here S_v^0 is given in Lemma 3.1.

Thus, system (1) undergoes a Hopf bifurcation at E_3 when $R_1 > 1$, $R_{02} < 1$, and h_2 passes through the critical value h_2^* such that $C_1(h_2^*)C_2(h_2^*) - C_3(h_2^*) = 0$.

(ii) By some mathematical deduction and rearrangement, the characteristic polynomial corresponding to E_4 can be rewritten as

$$\left(\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3\right)\left(\lambda^3 + C_1\lambda^2 + C_2\lambda + C_3\right) = 0, \tag{15}$$

where C_1, C_2, C_3 are given in (3), (4), and (5), respectively, and

$$A_1 = \mu_h + \beta_1\hat{I}_v + \mu_h + \delta_h + \mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2S_v^1} > 0,$$

$$\begin{aligned} A_2 &= (\mu_h + \beta_1\hat{I}_v)(\mu_h + \delta_h) + (\mu_h + \beta_1\hat{I}_v)\left(\mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2S_v^1}\right) \\ &\quad + (\mu_h + \delta_h)\left(\mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2S_v^1}\right) - \beta_1\beta_2\hat{S}_h\hat{S}_v \\ &= (\mu_h + \beta_1\hat{I}_v)(\mu_h + \delta_h) + (\mu_h + \beta_1\hat{I}_v)\left(\mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2S_v^1}\right) \\ &\quad + (\mu_h + \delta_h)\left(\mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2S_v^1}\right) - (\mu_h + \delta_h)\left(\mu_{v2} + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2S_v^1}\right) \\ &> (\mu_h + \beta_1\hat{I}_v)\left(\mu_h + \delta_h + \mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2S_v^1}\right) > 0, \end{aligned}$$

$$
\begin{aligned}
A_3 &= (\mu_h + \beta_1\hat{I}_v)(\mu_h + \delta_h)\left(\mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2 S_v^1}\right) - \mu_h\beta_1\beta_2\hat{S}_h\hat{S}_v \\
&= (\mu_h + \beta_1\hat{I}_v)(\mu_h + \delta_h)\left(\mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2 S_v^1}\right) \\
&\quad - \mu_h(\mu_h + \delta_h)\left(\mu_{v2} + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2 S_v^1}\right) > 0,
\end{aligned}
$$

where $\hat{I}_v$, $\hat{I}_h$, $\hat{S}_v$, $\hat{S}_h$, $\hat{P}_2$, and S_v^1 are given in Lemma 3.1, and

$$
\begin{aligned}
A_1A_2 - A_3 > {} & \left(\mu_h + \beta_1\hat{I}_v + \mu_h + \delta_h + \mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2 S_v^1}\right)(\mu_h + \beta_1\hat{I}_v) \\
& \cdot \left[(\mu_h + \delta_h) + \left(\mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2 S_v^1}\right)\right] - (\mu_h + \beta_1\hat{I}_v)(\mu_h + \delta_h) \\
& \cdot \left(\mu_{v2} + \beta_2\hat{I}_h + \alpha S_v^1 + \frac{h_2\hat{P}_2}{1 + a_2 S_v^1}\right) > 0.
\end{aligned}
$$

Therefore, by the Routh–Hurwitz criteria, equation $(\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3) = 0$ has three roots with negative real parts. For the equation $\lambda^3 + C_1\lambda^2 + C_2\lambda + C_3 = 0$, by the analysis results of disease-free E_3 in Theorem 3.2, we have that the disease equilibrium E_4 is locally asymptotically stable if $R_{02} > 1$ and $C_1C_2 - C_3 > 0$, and if $R_{02} > 1$, then system (1) undergoes a Hopf bifurcation at the disease equilibrium E_4 when h_2 passes through the critical value h_2^* such that $C_1(h_2^*)C_2(h_2^*) - C_3(h_2^*) = 0$.

Hence the proof of Theorem 3.2. □

Remark 1 From Theorem 3.2, we find that the introduction of predation into vectors results in the occurrence of Hopf bifurcation for system (1), and the condition $C_1C_2 - C_3 = 0$ is the threshold to determine the existence of Hopf bifurcation. On the other hand, when $R_{01} > 1$ then the disease persists. By introducing predators to the vectors, it is clear that the basic reproduction number R_{02} takes the role of threshold determining the persistence and extinction of the disease. If $R_{02} > 1$, then the vector-borne diseases will persist. If $R_{02} < 1$, then the vector-borne diseases will tend to die out. That is to say, reducing the value of R_2 will promote the positive effect of predators on disease control. In the next numerical simulation, we will further analyze the relationship between some predator parameters and the value of R_{02}, and present some simulation figures to show the positive role of predators in disease control.

3.2 Direction and stability of limit cycle

In this subsection, we study the stability of a limit cycle caused by the predator preying on the mature vector population. Before giving the main results, we first discuss the following subsystem:

$$
\begin{cases}
\frac{dM_v(t)}{dt} = b_2N_v(t) - (\mu_{v1} + d)M_v(t), \\
\frac{dN_v(t)}{dt} = dM_v(t) - \mu_{v2}N_v(t) - \alpha N_v^2(t) - \frac{h_2N_v(t)P_2(t)}{1+a_2N_v(t)}, \\
\frac{dP_2(t)}{dt} = \frac{\gamma_2h_2N_v(t)P_2(t)}{1+a_2N_v(t)} - e_2P_2(t),
\end{cases}
\tag{16}
$$

where $N_v(t)$ is the total mature vector population density at time t, that is, $N_v(t) = S_v(t) + I_v(t)$.

For system (16), there always exist a predator-free equilibrium $E_0(M_v^0, N_v^0, 0)$ and a predator-present equilibrium $E_1(\tilde{M}_v, \tilde{N}_v, \tilde{P}_2)$ if $R_1 > 1$, where $N_v^0 = S_v^0$, $\tilde{M}_v = M_v^1$, $\tilde{N}_v = S_v^1$, $\tilde{P}_2 = P_2^1$, and M_v^0, S_v^0, M_v^1, S_v^1, P_2^1 are given in Lemma 3.1.

It is easy to find that the equilibrium E_0 of system (16) is locally asymptotically stable if $R_1 < 1$ and unstable if $R_1 > 1$. The characteristic polynomial at E_1 of system (16) is

$$\lambda^3 + C_1\lambda^2 + C_2\lambda + C_3 = 0.$$

Then, by Theorem 3.2, we have the following results of the local stability and existence of Hopf bifurcation for system (16).

Corollary 3.2 *The local stability and existence of Hopf bifurcation of system* (16) *are as follows*:

(i) $E_0(M_v^0, N_v^0, 0)$ *is locally asymptotically stable if* $R_1 < 1$ *and unstable if* $R_1 > 1$.

(ii) $E_1(\tilde{M}_v, \tilde{N}_v, \tilde{P}_2)$ *is locally asymptotically stable if* $R_1 > 1$ *and* $C_1C_2 - C_3 > 0$.

(iii) *When* h_2 *passes through the critical value* h_2^*, *which satisfies* $C_1(h_2^*)C_2(h_2^*) - C_3(h_2^*) = 0$, *system* (16) *undergoes a Hopf bifurcation at the equilibrium* $E_1(\tilde{M}_v, \tilde{N}_v, \tilde{P}_2)$,

where C_1, C_2, C_3 *are given in* (3), (4), *and* (5), *respectively.*

In the following, we apply the center manifold theorem to study the stability of the limit cycle arising from Hopf bifurcation. For convenience, we first translate the origin of the coordinate system to the equilibrium $E_1(\tilde{M}_v, \tilde{N}_v, \tilde{P}_2)$ by writing

$$\bar{M}_v = M_v - \tilde{M}_v, \qquad \bar{I}_v = I_v - \tilde{I}_v, \qquad \bar{P}_2 = P_2 - \tilde{P}_2,$$

then from (16) we have

$$\frac{d}{dt}\begin{pmatrix} \bar{M}_v \\ \bar{N}_v \\ \bar{P}_2 \end{pmatrix} = \tilde{J}\begin{pmatrix} \bar{M}_v \\ \bar{N}_v \\ \bar{P}_2 \end{pmatrix} + \begin{pmatrix} \varphi_1 \\ \varphi_2 \\ \varphi_3 \end{pmatrix}, \tag{17}$$

where

$$\tilde{J} = \begin{pmatrix} -(\mu_{v1} + d) & b_2 & 0 \\ d & -(\mu_{v2} + 2\alpha S_v^1 + G_1) & -G_2 \\ 0 & G_3 & 0 \end{pmatrix} \tag{18}$$

and the nonlinear terms φ_1, φ_2, and φ_3 are given by

$$\varphi_1 = 0, \qquad \varphi_2 = -G_4\bar{N}_v^2 - G_5\bar{N}_v\bar{P}_2, \qquad \varphi_3 = -G_6\bar{N}_v\bar{P}_2, \tag{19}$$

where

$$G_4 = \alpha + \frac{a_2h_2P_2^1}{(1 + a_2S_v^1)^3}, \qquad G_5 = \frac{h_2}{(1 + a_2S_v^1)^2}, \qquad G_6 = \frac{a_2h_2\gamma_2P_2^1}{(1 + a_2S_v^1)^3}.$$

When the Hopf bifurcation characteristic equation holds, then the eigenvalues of $\tilde{J}$ are $\lambda_{1,2} = \pm i\eta$, $\lambda_3 = \theta$, where $\theta = -C_1$, $\eta^2 = C_2 = G_2G_3 + (\mu_{v1} + d)(\mu_{v2} + 2\alpha S_v^1 + G_1) - b_2 d$.

If the eigenvectors of $\tilde{J}$ associated with $\lambda_{1,2}$ are $W_1 \pm iW_2$, and the eigenvector corresponding to λ_3 is W_3, then it can be shown that the matrix $P = (W_2, W_1, W_3)$ is nonsingular. Furthermore,

$$P^{-1}\tilde{J}P = \begin{pmatrix} 0 & -\eta & 0 \\ \eta & 0 & 0 \\ 0 & 0 & \theta \end{pmatrix} \tag{20}$$

and P is given by

$$P = [p_{ij}] \quad (i, j = 1, 2, 3), \tag{21}$$

where

$$p_{11} = 0, \qquad p_{12} = 1, \qquad p_{13} = \frac{b_2}{\mu_{v1} + d + \theta},$$
$$p_{21} = \frac{\eta}{b_2}, \qquad p_{22} = \frac{\mu_{v1} + d}{b_2},$$
$$p_{23} = 1, \qquad p_{31} = -\frac{G_3(\mu_{v1} + d)}{b_2\eta}, \qquad p_{32} = \frac{G_3}{b_2}, \qquad p_{33} = \frac{G_3}{\theta},$$

and

$$Q = P^{-1} = \Delta[q_{ij}] \quad (i, j = 1, 2, 3), \tag{22}$$

where $\Delta = \det P^{-1}$ and

$$q_{11} = p_{22}p_{33} - p_{23}p_{32}, \qquad q_{12} = p_{32}p_{13} - p_{12}p_{33}, \qquad q_{13} = p_{12}p_{23} - p_{13}p_{22},$$
$$q_{21} = p_{31}p_{23} - p_{21}p_{33}, \qquad q_{22} = p_{11}p_{11} - p_{31}p_{13}, \qquad q_{23} = p_{21}p_{13} - p_{11}p_{23},$$
$$q_{31} = p_{21}p_{32} - p_{22}p_{31}, \qquad q_{32} = p_{31}p_{12} - p_{11}p_{32}, \qquad q_{33} = p_{11}p_{22} - p_{12}p_{21}.$$

Let the linear transformation

$$\bar{Y} = PW, \tag{23}$$

where $\bar{Y} = (\bar{M}_v, \bar{N}_v, \bar{P}_2)^T$ and $W = (x_1, x_2, x_3)^T$, then we have

$$W = P^{-1}\bar{Y},$$

where P is given by (21).

Substituting (23) into (17), we get

$$\frac{d(PW)}{dt} = \tilde{J}PW + H(PW),$$

where $H(PW) = H(\bar{Y}) = (\varphi_1, \varphi_2, \varphi_3)^T$, which implies that

$$\frac{dW}{dt} = \left(P^{-1}\tilde{J}P\right)W + P^{-1}H(PW), \tag{24}$$

where $P^{-1}\tilde{J}P$ is a constant matrix given by (20).

Now system (24) can be written as

$$\begin{cases} \dot{x} = Ax + F_1(x,y), \\ \dot{y} = By + F_2(x,y), \end{cases} \tag{25}$$

where $x = (x_1, x_2)^T$, $y = (x_3)$, A and B are the constant matrices given by

$$A = \begin{pmatrix} 0 & -\eta \\ \eta & 0 \end{pmatrix}, \qquad B = (\theta), \tag{26}$$

and F_1, F_2 are functions of C^2.

We can write system (24) as

$$\frac{d}{dt}\begin{pmatrix} x_1 \\ x_2 \\ x_3 \end{pmatrix} = \begin{pmatrix} 0 & -\eta & 0 \\ \eta & 0 & 0 \\ 0 & 0 & \theta \end{pmatrix}\begin{pmatrix} x_1 \\ x_2 \\ x_3 \end{pmatrix} + Q\begin{pmatrix} \varphi_1 \\ \varphi_2 \\ \varphi_3 \end{pmatrix}. \tag{27}$$

From (27) we have that

$$\frac{dx_3}{dt} = \theta x_3 + \Delta(q_{31}\varphi_1 + q_{32}\varphi_2 + q_{33}\varphi_3). \tag{28}$$

To prove the stability of limit cycle, we give the following two lemmas.

Lemma 3.2 ([43]) *System* (27) *has a local center manifold* $y = \psi(x)$, $K < \delta$, *where* ψ *is in* C^2. *The function* $\psi(x)$ *can be approximated arbitrarily closed as a Taylor series as proved by the following theorem.*

Lemma 3.3 ([43]) *Let* $\phi : S^n \to S^m$ *be* C^1 *in a neighborhood of origin,* $\phi(0) = 0$, $\phi'(0) = 0$, *and* $M\phi(x) = O(|x|^p)$ *as* $x \to \infty$, *then* $\psi(x) = \phi(x) + O(|x|^p)$ *as* $x \to \infty$, *where* $M\phi(x) = \phi'(x)[Bx + F_2(x, \phi(x))] - A\phi(x) - F_1(x, \phi(x))$ *and* $p > 1$.

Hence, by Lemma 3.3, the center manifold up to a quadratic approximation can be described by

$$x_3 = \psi(x_1, x_2) = \frac{1}{2}\left(a_{11}x_1^2 + 2a_{12}x_1x_2 + a_{22}x_2^2\right). \tag{29}$$

Then it follows that

$$\frac{dx_3}{dt} = \begin{pmatrix} a_{11}x_1 + a_{12}x_2 & a_{12}x_1 + a_{22}x_2 \end{pmatrix}\begin{pmatrix} 0 & -\eta \\ \eta & 0 \end{pmatrix}\begin{pmatrix} x_1 \\ x_2 \end{pmatrix}. \tag{30}$$

On the other hand, by (28) and (29) we have

$$\frac{dx_3}{dt} = \frac{\theta}{2}\left(a_{11}x_1^2 + 2a_{12}x_1x_2 + a_{22}x_2^2\right) + \Delta(q_{31}\varphi_1 + q_{32}\varphi_2 + q_{33}\varphi_3). \tag{31}$$

From (23), we have

$$\begin{aligned} &\bar{M}_\nu = p_{11}x_1 + p_{12}x_2 + p_{13}x_3, \qquad \bar{N}_\nu = p_{21}x_1 + p_{22}x_2 + p_{23}x_3, \\ &\bar{P}_2 = p_{31}x_1 + p_{32}x_2 + p_{33}x_3. \end{aligned} \tag{32}$$

By substituting $\bar{M}_\nu$, $\bar{N}_\nu$, and $\bar{P}_2$ from (32) into Eq. (19) and rearranging, we then obtain φ_1, φ_2, and φ_3 as follows:

$$\begin{aligned} \varphi_1 &= 0, \\ \varphi_2 &= -G_4(p_{21}x_1 + p_{22}x_2 + p_{23}x_3)^2 - G_5(p_{21}x_1 + p_{22}x_2 + p_{23}x_3) \\ &\quad \cdot (p_{31}x_1 + p_{32}x_2 + p_{33}x_3), \\ &= -G_4(p_{21}x_1 + p_{22}x_2)^2 - G_4p_{23}\Lambda(p_{21}x_1 + p_{22}x_2) \\ &\quad - G_5(p_{21}x_1 + p_{22}x_2)(p_{31}x_1 + p_{32}x_2) \\ &\quad - \frac{1}{2}G_5p_{33}\Lambda(p_{21}x_1 + p_{22}x_2) - \frac{1}{2}G_5p_{23}\Lambda(p_{31}x_1 + p_{32}x_2), \\ \varphi_3 &= -G_6\bar{N}_\nu\bar{P}_2 \\ &= -G_6(p_{21}x_1 + p_{22}x_2)(p_{31}x_1 + p_{32}x_2) \\ &\quad - \frac{1}{2}G_6p_{33}\Lambda(p_{21}x_1 + p_{22}x_2) - \frac{1}{2}G_6p_{23}\Lambda(p_{31}x_1 + p_{32}x_2), \end{aligned} \tag{33}$$

where $\Lambda = a_{11}x_1^2 + 2a_{12}x_1x_2 + a_{22}x_2^2$.

Now it follows from (30) and (31) that

$$\begin{aligned} \frac{dx_3}{dt} &= \begin{pmatrix} a_{11}x_1 + a_{12}x_2 & a_{12}x_1 + a_{22}x_2) \end{pmatrix} \begin{pmatrix} 0 & -\eta \\ \eta & 0 \end{pmatrix} \begin{pmatrix} x_1 \\ x_2 \end{pmatrix} \\ &= \frac{\theta}{2}\left(a_{11}x_1^2 + 2a_{12}x_1x_2 + a_{22}x_2^2\right) + \Delta(q_{31}\varphi_1 + q_{32}\varphi_2 + q_{33}\varphi_3) \\ &= x_1^2\left[\frac{\theta}{2}a_{11} - \Delta\left(q_{32}\left(G_4p_{21}^2 + G_5p_{21}p_{31}\right) + q_{33}G_6p_{21}p_{31}\right)\right] + x_1x_2 \\ &\quad \cdot \left[\theta a_{12} - \Delta\left(q_{32}\left(2G_4p_{21}p_{22} + G_5(p_{21}p_{32} + p_{22}p_{31})\right) + q_{33}G_6(p_{21}p_{32} + p_{22}p_{31})\right)\right] \\ &\quad + x_2^2\left[\frac{\theta}{2}a_{22} - \Delta\left(q_{32}\left(G_4p_{22}^2 + G_5p_{22}p_{32}\right) + q_{33}G_6p_{22}p_{32}\right)\right] + o\left(\rho^2\right), \end{aligned} \tag{34}$$

where $o(\rho^2)$ represents higher order term when $\rho^2 \to 0$, $\rho = \sqrt{x^2 + y^2}$.

Comparing the coefficient of x_1^2, x_1x_2, and x_2^2 of both sides of Eq. (34), we have

$$\begin{pmatrix} -\frac{\theta}{2} & \eta & 0 \\ -\eta & -\theta & \eta \\ - & -\eta & -\frac{\theta}{2} \end{pmatrix} \begin{pmatrix} a_{11} \\ a_{12} \\ a_{22} \end{pmatrix} = \begin{pmatrix} \Omega_1 \\ \Omega_2 \\ \Omega_3 \end{pmatrix}, \tag{35}$$

where

$$\begin{aligned}
\Omega_1 &= -\Delta\big(q_{32}\big(G_4p_{21}^2 + G_5p_{21}p_{31}\big) + q_{33}G_6p_{21}p_{31}\big),\\
\Omega_2 &= -\Delta\big(q_{32}\big(2G_4p_{21}p_{22} + G_5(p_{21}p_{32} + p_{22}p_{31})\big) + q_{33}G_6(p_{21}p_{32} + p_{22}p_{31})\big),\\
\Omega_3 &= -\Delta\big(q_{32}\big(G_4p_{22}^2 + G_5p_{22}p_{32}\big) + q_{33}G_6p_{22}p_{32}\big).
\end{aligned}$$

It can be easily shown that

$$\begin{aligned}
a_{11} &= -\frac{[\eta^2(\Omega_1 + \Omega_3) + \frac{\theta}{2}(\eta\Omega_2 + \theta\Omega_1)]}{(\frac{\theta^3}{4} + \eta^2\theta)},\\
a_{12} &= -\frac{[\frac{\theta^2\Omega_2}{4} + \frac{\eta\theta}{2}(\Omega_3 - \Omega_1)]}{(\frac{\theta^3}{4} + \eta^2\theta)},\\
a_{13} &= -\frac{[\frac{\theta^2\Omega_3}{2} - \frac{\theta}{2}\eta\Omega_2 + \eta^2(\Omega_1 + \Omega_3)]}{(\frac{\theta^3}{4} + \eta^2\theta)}.
\end{aligned} \tag{36}$$

Then the flow on center manifold is governed by the two-dimensional system

$$\dot{x} = Ax + F_1(x, \psi\big((x)\big). \tag{37}$$

Now we give the center manifold theorem by the following theorem to determine the asymptotic behaviors of the solution of (25).

Theorem 3.3 *Suppose that the zero solution of* (37) *is asymptotically stable* (*unstable*), *then the zero solution of* (25) *is asymptotically stable* (*unstable*).

System (37) can be written as

$$\frac{d}{dt}\begin{pmatrix} x_1 \\ x_2 \end{pmatrix} = \begin{pmatrix} 0 & -\eta \\ \eta & 0 \end{pmatrix}\begin{pmatrix} x_1 \\ x_2 \end{pmatrix} + \begin{pmatrix} \Gamma \\ \Sigma \end{pmatrix}, \tag{38}$$

where $\Gamma = q_{11}\varphi_1 + q_{12}\varphi_2 + q_{13}\varphi_3 + o(\rho^2)$, $\Sigma = q_{21}\varphi_1 + q_{22}\varphi_2 + q_{23}\varphi_3 + o(\rho^2)$, Λ, φ_1, φ_2, and φ_3 are given in (33).

The stability of the limit cycle arising from a Hopf bifurcation is determined by the sign of the quantity Π, where

$$\begin{aligned}
\Pi = {} & \Gamma_{111} + \Sigma_{112} + \Gamma_{122} + \Sigma_{222} + \frac{1}{\eta}\big[\Gamma_{12}(\Gamma_{11} + \Gamma_{22})\\
& - \Sigma_{12}(\Sigma_{11} + \Sigma_{22}) - \Gamma_{11}\Sigma_{11} + \Gamma_{22}\Sigma_{22}\big],
\end{aligned} \tag{39}$$

where Γ_{ijk} denotes the partial derivative $\frac{\partial^3\Gamma}{\partial x_i\partial x_j\partial x_k}$ at the origin and quantities with two subscripts represent order partial derivatives at the origin.

If $\Pi < 0$, the bifurcation limit cycle is stable and the Hopf bifurcation is called supercritical; if $\Pi > 0$, the bifurcation limit cycle is unstable and the Hopf bifurcation is called subcritical.

Here

$$\Gamma_{111} = -q_{12}(6a_{11}G_4p_{21}p_{23} + 3a_{11}G_5p_{21}p_{23} + 3a_{11}G_5p_{23}p_{31}),$$

$$\Sigma_{112} = -q_{22}(2a_{11}G_4p_{22}p_{23} + a_{11}G_5p_{22}p_{23} + a_{11}G_5p_{23}p_{32}),$$

$$\Gamma_{122} = -q_{12}(2a_{22}G_4p_{21}p_{23} + a_{22}G_5p_{21}p_{33} + a_{22}G_5p_{23}p_{31}),$$

$$\Sigma_{112} = -q_{22}(6a_{11}G_4p_{22}p_{23} + 3a_{22}G_5p_{22}p_{33} + 3a_{22}G_5p_{23}p_{32}),$$

$$\Gamma_{11} = -2q_{12}\left(G_4p_{21}^2 + G_5p_{21}p_{31}\right) - 2q_{13}G_6p_{21}p_{31},$$

$$\Gamma_{22} = -2q_{12}\left(G_4p_{22}^2 + G_5p_{22}p_{32}\right) - 2q_{13}G_6p_{22}p_{32},$$

$$\Sigma_{11} = -2q_{22}\left(G_4p_{21}^2 + G_5p_{21}p_{31}\right) - 2q_{23}G_6p_{21}p_{31},$$

$$\Sigma_{22} = -2q_{22}\left(G_4p_{22}^2 + G_5p_{22}p_{32}\right) - 2q_{23}G_6p_{22}p_{32},$$

$$\Gamma_{12} = -q_{12}\left[2G_4p_{21}^2 + G_5(p_{21}p_{32} + p_{22}p_{31})\right] - q_{13}G_6(p_{21}p_{32} + p_{22}p_{31}),$$

$$\Sigma_{12} = -q_{22}\left[2G_4p_{21}p_{22} + G_5(p_{21}p_{32} + p_{22}p_{31})\right] - q_{23}G_6(p_{21}p_{32} + p_{22}p_{31}).$$

The sign of Π can be obtained by putting the values of Γ_{111}, Σ_{112}, Γ_{122}, Σ_{222}, Γ_{11}, Γ_{22}, Σ_{11}, Σ_{22}, Γ_{12}, Σ_{12} in Eq. (39).

Based on the above results, for system (1), we have the following.

Theorem 3.4 *If $R_1 > 1$, $R_{02} < 1$, and $C_1C_2 - C_3 < 0$, then for system (1), there exists a stable limit cycle in the (M_v, S_v, P_2) space, in which case, despite oscillations, predation will lead to the elimination of the virus from the system, and uninfected vectors still exist.*

Proof From the characteristic equation (9) of equilibrium E_3 of system (1), we have $\lim_{t\to\infty}(S_h(t), I_h(t), I_v(t)) = (S_h^0, 0, 0)$ if $R_1 > 1$ and $R_{02} < 1$. Then by Lemmas 3.2, 3.3 and Theorem 3.3 there exists a stable limit cycle in the (M_v, S_v, P_2) space for system (1) if $R_1 > 1$, $R_{02} < 1$ and $C_1C_2 - C_3 < 0$.

This completes the proof of Theorem 3.4. □

By numerical simulation, we show that there exists a stable limit cycle in the $(S_h, I_h, M_v, S_v, I_v, P_2)$ space for system (1) if $R_1 > 1$, $R_{02} > 1$ and $C_1C_2 - C_3 < 0$ (see Fig. 4–5).

4 Numerical simulation of the system dynamics

In this subsection, numerical simulations are given to show the stability and bifurcation of system (1) (see Fig. 1–5) and the effect of stage structure on disease dynamics with and without predators (see Fig. 6–7).

Example 1 We take $b_1 = 0.7978$, $b_2 = 0.9020$, $\mu_h = 0.3485$, $\delta_h = 0.8706$, $\mu_{v1} = \mu_{v2} = 0.2381$, $d = 0.8202$, $\beta_1 = 0.9050$, $\beta_2 = 0.6740$, $\alpha = 0.1464$ (all the parameters are stochastically chosen for illustrative purpose only). Numerical calculations give $R_{01} = 3.1489 > 1$ and $C_1C_2 - C_3 = -0.0138 < 0$. It follows from Corollary 3.1 that the disease equilibrium of system (2) is locally asymptotically stable, that is, the vector-borne disease will persist in the absence of predators (see Fig. 1).

Example 2 We take $a_2 = 0.6497$, $e_2 = 0.1356$, $\gamma_2 = 0.7855$, $h_2 = 0.6425$ and keep other parameters unchanged in Example 1. Numerical calculations give $R_1 = 6.2465 > 1$ and

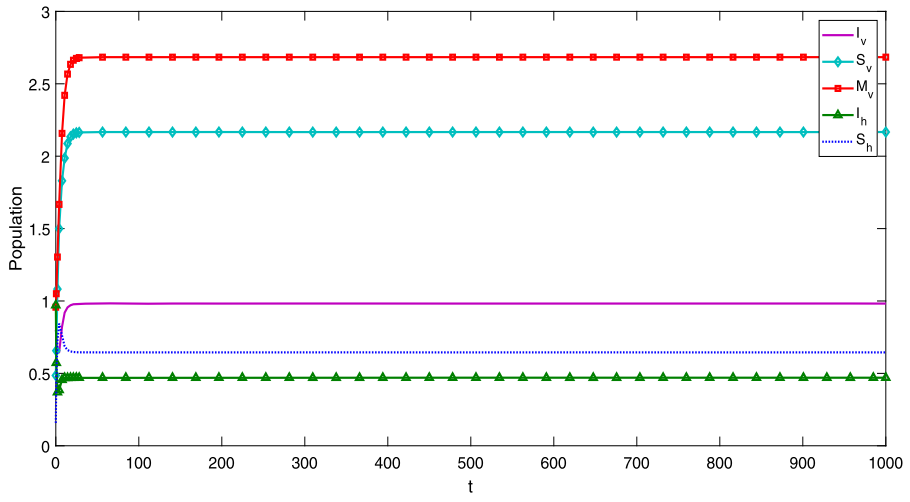

Figure 1 Solution curves of system (2) with parameters given in Example 1

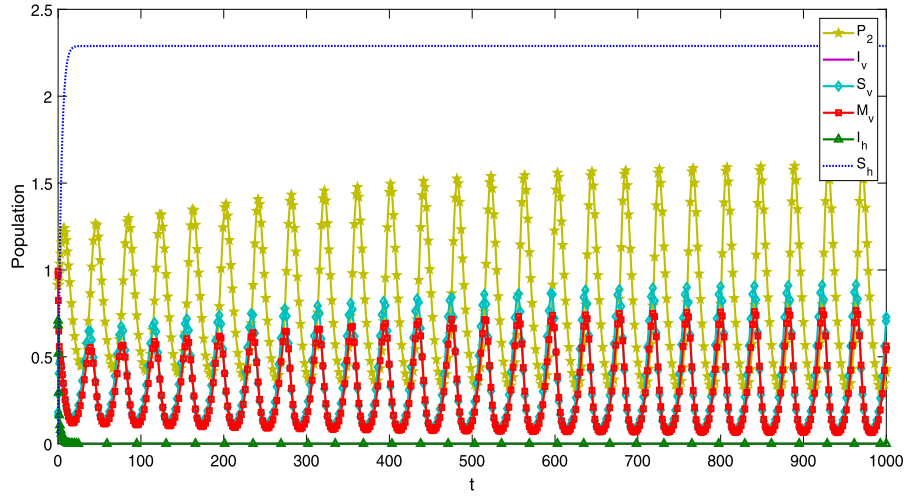

Figure 2 Solution curves of system (1) with parameters given in Example 2

$R_{02} = 0.5331 < 1$. It follows from Theorem 3.2 that the vector-borne disease will tend to die out (see Fig. 2). By calculations, $C_1C_2 - C_3 = -0.0138 < 0$, then by Theorem 3.4 there exists a stable limit cycle in the (M_v, S_v, P_2) space for system (1) (see Fig. 3).

Example 3 Keep all the parameters fixed in Example 2 except for $h_2 = 0.3841$. By calculation the values of R_1 and $C_1C_2 - C_3$ remain the same as in Example 2 and $R_{02} = 1.0396 > 1$, then by Theorem 3.2 the vector-borne disease persists, but the equilibrium levels of the infected hosts and vectors have been greatly lessened when the predator P_2 is added (see Fig. 4). Moreover, Fig. 5 illustrates that there exists a stable limit cycle for system (1) if $R_1 > 1$, $R_{02} < 1$, and $C_1C_2 - C_3 < 0$.

Example 4 Take $\mu_{v1} = 0.1$ and keep all the parameters unchanged in Example 1 except for parameters b_2 and d. Then $R_{01} < 1$ if b_2 and d are close to 0.48 and 0.1, respectively.

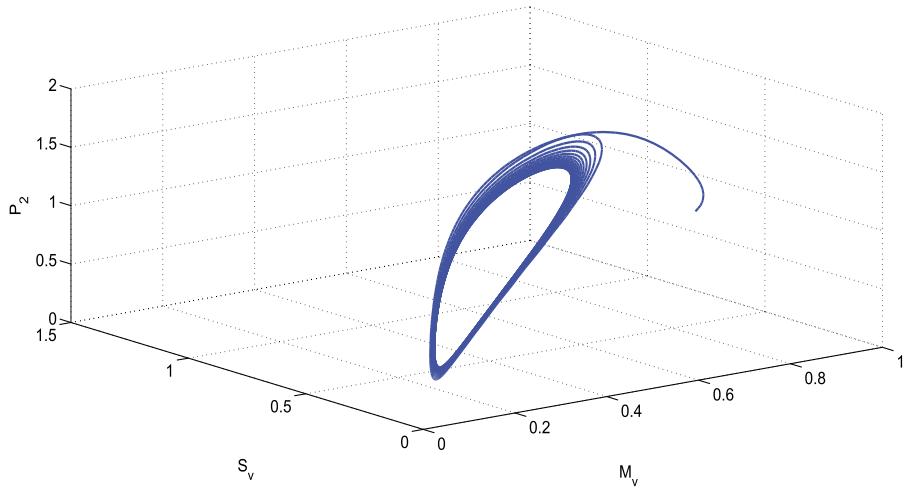

Figure 3 Stable limit cycle in the (M_v, S_v, P_2) space for system (1) with parameters given in Example 2

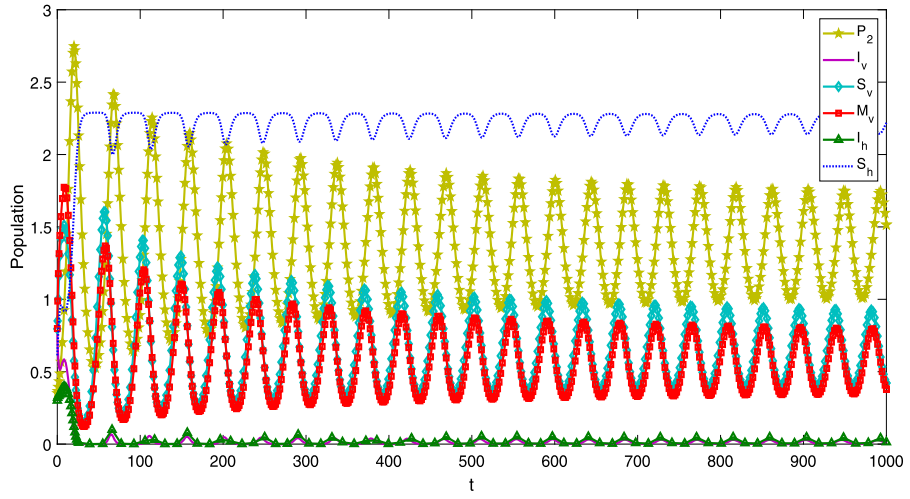

Figure 4 Solution curves of system (1) with parameters given in Example 3

But if b_2 and d are large, then we have $R_{01} > 1$. This suggests that in the absence of the predator, the birth rate b_2 of the immature vectors and the conversion rate d from the immature vectors to the mature vectors will cause the spread of the vector-borne disease (see the left-hand side of Fig. 6). Take $\mu_{v1} = 0.1$ and keep all the parameters unchanged in Example 2 except for parameters b_2 and d, then from the right-hand side of Fig. 6 it is clear that when predators are present, an increase in the vector birth rate b_2 and the conversion rate d from the immature vectors to the mature vectors will reduce the value of the reproduction number R_{02}, and therefore lessen the prevalence of disease, because an increase in the vector birth rate and the conversion rate leads to an increase in the predator population.

Example 5 Take $b_2 = 0.7343$ and keep all the parameters unchanged in Example 4 except for parameters μ_{v1} and d. Then $R_{01} < 1$ if d and u_{v1} are close to 0.1 and 0.2, respectively.

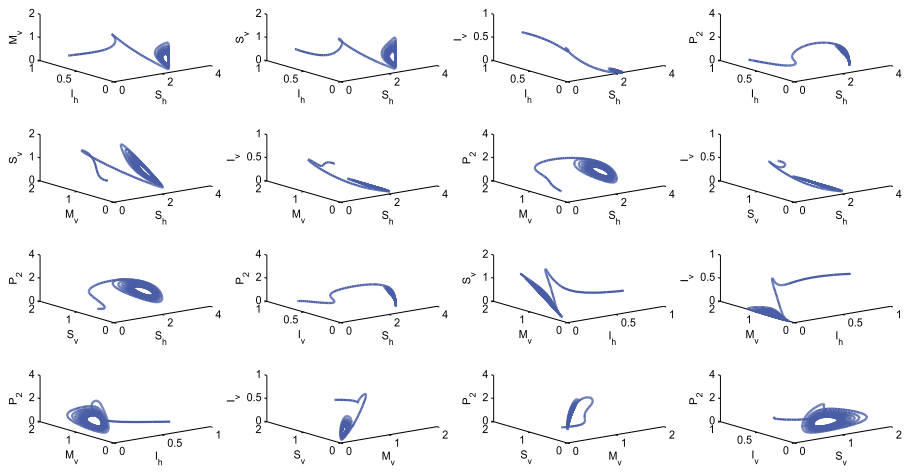

Figure 5 Stable limit cycle for system (1) with parameters given in Example 3

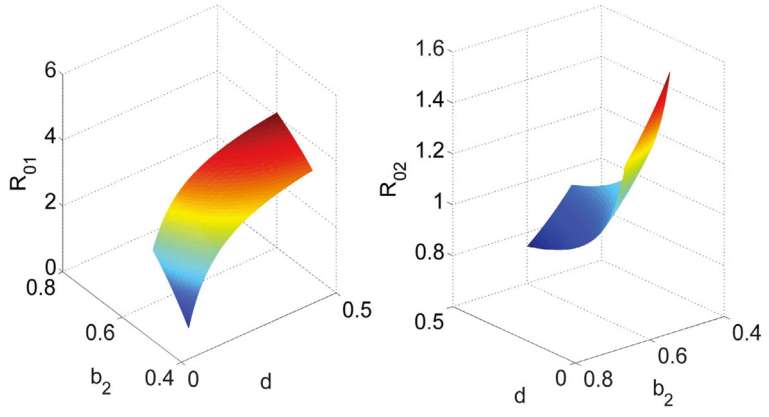

Figure 6 Surface plot of the basic reproduction numbers R_{01} and R_{02} as functions of b_2 and d for the parameters given in Example 4

But if d is large and u_{v1} is small, then we have $R_{01} > 1$. This suggests that the conversion rate d from the immature vectors to the mature vectors will cause the spread of the vector-borne disease in the absence of predators, while the natural death rate u_{v1} of the immature vector can reduce the vector disease spread (see the left-hand side of Fig. 7). When the predators are introduced, we find from the right-hand side of Fig. 7 that an increase in the conversion rate d from the immature vectors to the mature vectors leads to a decline in the value of the reproduction number R_{02}, while an increase in the natural death rate of the immature vectors leads to an increase in the value of the reproduction number R_{02}, because an increase in the immature vector mortality rate μ_{v1} indirectly leads to an increase in the predator densities.

Remark 2 From Figs. 2–3 and Figs. 4–5, it is clear that periodic solutions exist when a predator species P_2 is introduced in the system, suggesting the occurrence of a super-critical Hopf bifurcation. From Figs. 4–5, the hosts, the vectors, including the immature

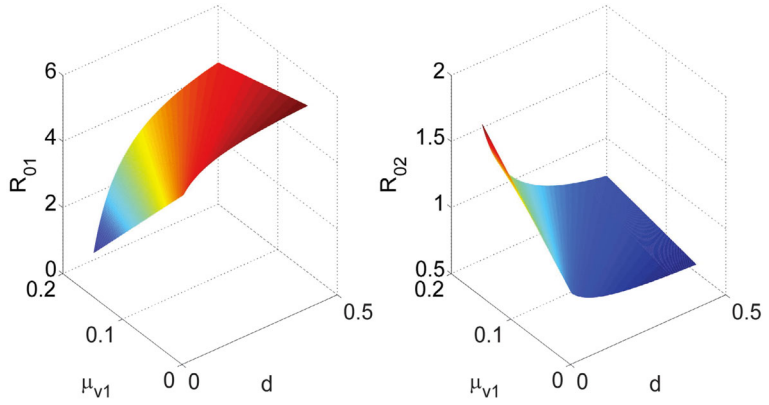

Figure 7 Surface plot of the basic reproduction numbers R_{01} and R_{02} as functions of μ_{v1} and d for the parameters given in Example 5

and mature ones, and the predators oscillate, leading to the persistence of the vector-transmitted disease when the predator species P_2 is added. By enhancing predators' attack rate h_2, the level of infected hosts I_h and vectors I_v will decline, and when $h_2 = 0.6425$, from Figs. 2–3 we can see that predation brings about the extinction of the disease, despite the oscillations between the predators and the vectors. That is to say, introducing predators to vectors is advantageous to vector-borne disease control, and the larger the capturing rate h_2 is, the better control of disease will be.

Remark 3 From the left-hand sides of Figs. 6 and 7, we find that the stage structure has different effects on disease transmission dynamics with and without predators. When predators are introduced, small values of the birth rate b_2 and the conversion rate d, and large value of the natural death rate μ_{v1} will lead to the increase in the value of the basic reproduction number R_{02}, and therefore increase the risk of disease spread. In the absence of predators, we find that the reproduction number R_{01} increases with the increase in the birth rate b_2 and the conversion rate d of the immature vectors, and with the decrease in the natural death rate μ_{v1} of the immature vectors. Therefore, for vector–host disease prevention and control in the absence of the predator, it is necessary to take some strategies to reduce the birth rate b_2 and the conversion rate d, and enlarge the natural death rate μ_{v1} at vectors' larva stage, such as use of physical strategies, use of pesticides, biological control, and so on.

From Sect. 3, we find that the predator P_2 brings about complicated dynamics of system (1), and we also find that introducing the predator has certain benefits to disease control. It is clear from Figs. 2–5 that the larger the value of capturing rate h_2 is, the lower equilibrium levels of infected hosts and vectors will be.

5 Conclusions and discussions

In this paper, we mainly formulate and analyze a host–vector–predator model with stage structure for the vectors. The period of growth for the vectors is divided into immature and mature stages according to the transmission properties (e.g., cysticercosis and scrub typhus are spread among hosts by the immature vectors, while some infectious diseases, such as malaria, dengue fever, West Nile virus, and pine wilt disease, are transmitted

among hosts by the mature vectors). The interactions between the predator and the vector are modeled by Holling type II functional response. Corollary 3.1 reveals that in the absence of the predator, the reproduction number R_{01} of system (2) provides the threshold which decides the persistence and extinction of vector-borne diseases. If $R_{01} < 1$, the diseases will tend to die out, while if $R_{01} > 1$ then the diseases will persist (see Fig. 1). When the predators are added, we find from Theorem 3.2 that the diseases still persist if $R_{02} > 1$, but the equilibrium levels of the infected hosts and vectors have been lessened (see Fig. 2). If $R_{02} < 1$, then by Theorem 3.2 the diseases can be eradicated (see Fig. 3). Therefore, predators have positive effects on disease control. Moreover, by Theorem 3.2 predation causes the phenomenon of limit cycle arising from a Hopf bifurcation (see Fig. 2–5). If $R_{02} < 1$ and $C_1C_2 - C_3 > 0$, then there exists a stable limit cycle in the (M_v, S_v, P) space for system (1). Though periodic oscillation occurs between the vectors and the predators, the diseases can be eradicated by the effect of predators on vectors (see Fig. 2–3). However, if $R_{02} > 1$ and $C_1C_2 - C_3 > 0$, then there exists a stable limit cycle for system (1), and the diseases cannot be eradicated (see Fig. 4–5). Finally, the effect of stage structure on disease spread with and without predators has been illustrated (see Fig. 6–7). From the left-hand sides of Figs. 6 and 7, it is clear that in the absence of predators, the reproduction number R_{01} increases with the increase in the birth rate b_2 and the conversion rate d of the immature vectors, while R_{01} increases with the decrease in the natural death rate μ_{v1} of the immature vectors. Therefore, it is necessary to take some strategies to reduce the birth rate b_2 and the conversion rate d and enlarge the natural death rate μ_{v1} at vectors' larva stage through the use of physical strategies, use of pesticides, biological control, and so on. From the right-hand sides of Figs. 6 and 7, we find that after introducing the predators, the effect of b_2, d, and μ_{v1} on disease transmission is inverse compared with the effect of b_2, d, and μ_{v1} on disease transmission in the absence of predators. Therefore, to exert the best effect of predators on disease control, we should increase the values of the birth rate b_2 and the conversion rate d to certain degree, and decrease the value of the natural death rate μ_{v1} of vectors at the immature stage.

Acknowledgements
The authors would like to thank the editor and two anonymous reviewers for their valuable suggestions and comments.

Funding
The work is supported by the National Natural Science Foundation of China (grant no. 71701082 and 71271103), the Six Talents Peak Foundation of Jiangsu Province, and the Innovative Foundation for Doctoral Candidate of Jiangsu Province, China (grant no. CXZZ13_0687).

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

Authors' contributions
All authors contributed equally to the writing of this paper. All authors read and approved the final manuscript.

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Publisher's Note

Received: 3 February 2018 Accepted: 22 August 2018 Published online: 15 September 2018

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