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Research Article

Global Dynamics of a Host-Vector-Predator Mathematical Model

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A mathematical model which links predator-vector(prey) and host-vector theory is proposed to examine the indirect effect of predators on vector-host dynamics. The equilibria and the basic reproduction number R_0 are obtained. By constructing Lyapunov functional and using LaSalle's invariance principle, global stability of both the disease-free and disease equilibria are obtained. Analytical results show that R_0 provides threshold conditions on determining the uniform persistence and extinction of the disease, and predator density at any time should keep larger or equal to its equilibrium level for successful disease eradication. Finally, taking the predation rate as parameter, we provide numerical simulations for the impact of predators on vector-host disease control. It is illustrated that predators have a considerable influence on disease suppression by reducing the density of the vector population.

1. Introduction

Host-vector diseases are infectious diseases caused by an infectious microbe transmitted by a blood (or sap-) sucking arthropod called vectors, which carry the disease without getting it themselves. For instance, human and animal diseases are such as malaria, dengue fever, West Nile virus, Chagas disease, sleeping sickness, and Lyme disease. Host-vector disease also affects other living organisms, such as plants. Examples of vector-borne infections in crops include tobacco mosaic virus (TMV), tomato spotted wilt virus (TSWV), tomato yellow leaf curl virus (TYLCV), cucumber mosaic virus (CMV), and potato virus Y (PVY) [1]. Vector-borne infections in trees include the pine wilt disease and the red ring disease in palms [2].

Recently, the frequent occurrence of natural disasters and environmental degradation around the world creates conditions suitable for breeding of vectors so that vector-borne diseases are constantly emerging. It is estimated that almost three-fourths of the new infectious diseases in recent years belong to vector-borne infectious disease. Vector-borne diseases remain a serious global threat to humans, livestock, and crops and cause great economic losses in agriculture and forestry; thus; control of such diseases is of great economic and public health concern.

The present mode of controlling vector-borne disease includes using bednets, spraying insecticides [3], and sterile insect technique (SIT) [4–6]. Effective vaccines and treatment control have not been adopted for most of the diseases except that malaria, which is preventable and curable when treatment and prevention measures are taken properly. One potential approach to control vector-borne disease is to introduce biological enemies (biocontrol agents) of the vector. Compared to insecticide method and sterile insect technique, introducing biological enemies (biocontrol agents) of the vector is more safe and cost-efficient. Moreover, it will lead to ecological balance and environment protection. Biological control of vectors has been successfully applied in controlling a variety of disease pathogens, including crop diseases such as the tomato leaf curl virus in India and the cassava mosaic virus in sub-Saharan Africa [7, 8], as well as tree diseases such as pine wilt disease in Japan and China [9, 10]. For human diseases, Entomogenous fungi are used as promising biopesticides for tick and sleeping sickness control [11, 12]. Predators have been introduced as biological control agents of vectors for various diseases such as malaria, dengue fever,

tick disease, and Lyme disease [13–20]. Several recent studies suggested that predators led to a decline in local cases of dengue fever in Vietnam and Thailand [21, 22] and malaria in India [23, 24].

Mathematical models provide a powerful tool to understand the dynamics of disease spreading through a population and in decision-making process regarding disease prediction and disease control. Since Ross [25] first proposed a malaria model in 1911, many authors have been attracted to mathematical modeling, and very large good results on the subject have been presented. For example, the authors in [26] studied the global dynamics and back bifurcation of a vector-borne disease model with horizontal transmission in host population, which includes exposed classes both in host and vector populations and extended models studied in [27, 28].

However, compared with biological control of herbivorous pests, which has long been established as a major component of pest management programmers and is aimed to direct decrease pest densities by pest enemies [29, 30], biological control of vectors has been seldom investigated based on mathematical models. The main reason is that modeling the biological control of vectors is a complex interaction, mainly including the virus-host interaction, vector-host interaction, and vector- (prey-) enemy interaction, which is more complicated than the only pest-enemy interaction [31, 32] of biological pest control.

Up to know, only several authors have studied the biological control of vector to reduce the disease incidence by mathematical models. For examples, Moore et al. [33] first proposed a host-vector-predator model to study the effect of predator on the transmission and control of vector-borne disease. The efficacy of three types of biocontrol agents: predator/parasitoid, competitor, and pathogen of the vector, is compared in [34] to reduce disease incidence. Zhou and Yao [35] improved the model proposed in [33], study the disease control threshold, and limit cycles with persistence of disease or without disease. However, both papers [33, 35] focus on the constant total host populations, and the hosts and vectors are simply divided into susceptible and infective ones.

Motivated by the above considerations, in this paper we consider a new host-vector-predator model, where both total host and vector population are time-dependent population size and the total host population is divided into four subpopulations of susceptible hosts, exposed hosts, infectious hosts, and recovered hosts. Furthermore, the total vector population is divided into three subpopulations of susceptible hosts, exposed hosts, and infectious hosts. The aim is to explore the global dynamics of the proposed host-vector-predator model and the impacts of predator on host-vector disease control.

The rest of the paper is organized as follows. In Section 2, we present a formulation of the mathematical model. The equilibria and the basic reproduction number are given in Section 3. In Section 4, global stability analysis of the equilibria is investigated. In Section 5, we use some numerical simulations to explore the impact of predators on the prevalence of vector-borne disease. Conclusions are given in Section 6.

2. Model Formulation

The basic model for the transmission dynamics of vector-borne disease with predator control is given by the following deterministic system of nonlinear differential equations:

$$\begin{aligned}
\frac{dS_h(t)}{dt} &= b_1 - \beta_1 S_h(t) I_v(t) - \mu_h S_h(t),\\
\frac{dE_h(t)}{dt} &= \beta_1 S_h(t) I_v(t) - (\alpha_h + \mu_h) E_h(t),\\
\frac{dI_h(t)}{dt} &= \alpha_h E_h(t) - (\gamma_h + \delta_h + \mu_h) I_h(t),\\
\frac{dR_h(t)}{dt} &= \gamma_h I_h(t) - \mu_h R_h(t),\\
\frac{dS_v(t)}{dt} &= b_2 - \beta_2 S_v(t) I_h(t) - \mu_v S_v(t) - h S_v(t) P_v(t),\\
\frac{dE_v(t)}{dt} &= \beta_2 S_v(t) I_h(t) - \alpha_v E_v(t) - \mu_v E_v(t) - h E_v(t) P_v(t),\\
\frac{dI_v(t)}{dt} &= \alpha_v E_v(t) - \mu_v I_v(t) - h I_v(t) P_v(t),\\
\frac{dP_v(t)}{dt} &= \varepsilon h (S_v(t) + E_v(t) + I_v(t)) P_v(t) - e P_v(t),
\end{aligned} \tag{1}$$

with initial conditions

$$\begin{aligned}
&S_h(0) \geq 0, \quad E_h(0) \geq 0, \quad I_h(0) \geq 0,\\
&R_h(0) \geq 0, \quad S_v(0) \geq 0, \quad E_v(0) \geq 0,\\
&I_v(0) \geq 0, \quad P_v(0) \geq 0,
\end{aligned} \tag{2}$$

where the total host population at time t denoted by $N_h(t)$ is divided into four subpopulations of susceptible hosts $S_h(t)$, exposed hosts $E_h(t)$, infectious hosts $I_h(t)$, and recovered hosts $R_h(t)$, so that $N_h(v) = S_h(t) + E_h(t) + I_h(t) + R_h(t)$. The total vector population at time t denoted by $N_v(t)$ is split into susceptible vectors $S_v(t)$, exposed vectors $E_v(t)$ and infectious vectors $I_v(t)$, so that $N_v(t) = S_v(t)+E_v(t)+I_v(t)$. The predator of the vector at time t is represented by $P_v(t)$. b_1 and b_2 are, respectively, the recruitment rate of hosts and vectors. Parameters β_1 and β_2 are, respectively, the rate of biting from susceptible hosts to infected hosts and susceptible vectors to infected vectors. μ_h and μ_v are, respectively, the natural death rate of infected hosts and vectors. Exposed hosts and vectors develop symptoms of the disease and move to the infectious class at rates α_h and α_v, respectively. γ_h is the natural recovery rate from the infected hosts. δ_h is the disease-caused death rate of infected hosts. h and ε are, respectively, the predation rates and conversation rate of the predator to the vector. e is the mortality of the predator including naturally, being preyed both by humans and other animals.

Adding the host equations in (1), we have

$$\frac{dN_h(t)}{dt} = b_1 - \mu_h N_h(t) - \delta_h I_h(t), \tag{3}$$

and from the last four equations of system (1), we have

$$\begin{aligned} \frac{dN_v(t)}{dt} &= b_2 - \mu_v N_v(t) - hN_v(t) P_v(t), \\ \frac{dP_v(t)}{dt} &= \varepsilon h N_v(t) P_v(t) - eP_v(t). \end{aligned} \tag{4}$$

By (3) and (4), we have

$$\begin{aligned} \frac{dN_h(t)}{dt} &\le b_1 - \mu_h N_h(t), \\ \frac{dN_v(t)}{dt} &\le b_2 - \mu_v N_v(t), \\ \frac{dP_v(t)}{dt} &= \varepsilon h N_v(t) P_v(t) - eP_v(t). \end{aligned} \tag{5}$$

Let

$$\begin{aligned} \overline{\Gamma} = \Bigg\{ & \left(S_h, E_h, I_h, R_h, S_v, E_v, I_v, P_v\right) \in R_8^+ \\ & \mid 0 \le S_h + E_h + I_h + R_h \le \frac{b_1}{\mu_h}, \\ & 0 \le S_v + E_v + I_v \le \frac{b_2}{\mu_v}, P_v \ge 0 \Bigg\}; \end{aligned} \tag{6}$$

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

Since the fourth equation for $R_h(t)$ in system (1) does not influence the dynamics behavior of system, we can omit the equations for $R_h(t)$. System (1) in the invariant space $\overline{\Gamma}$ can be written as the following seven dimensional nonlinear systems:

$$\begin{aligned} \frac{dS_h(t)}{dt} &= b_1 - \beta_1 S_h(t) I_v(t) - \mu_h S_h(t), \\ \frac{dE_h(t)}{dt} &= \beta_1 S_h(t) I_v(t) - \left(\alpha_h + \mu_h\right) E_h(t), \\ \frac{dI_h(t)}{dt} &= \alpha_h E_h(t) - mI_h(t), \\ \frac{dS_v(t)}{dt} &= b_2 - \beta_2 S_v(t) I_h(t) - \mu_v S_v(t) - hS_v(t) P_v(t), \\ \frac{dE_v(t)}{dt} &= \beta_2 S_v(t) I_h(t) - \alpha_v E_v(t) - \mu_v E_v(t) - hE_v(t) P_v(t), \\ \frac{dI_v(t)}{dt} &= \alpha_v E_v(t) - \mu_v I_v(t) - hI_v(t) P_v(t), \\ \frac{dP_v(t)}{dt} &= \varepsilon h \left(S_v(t) + E_v(t) + I_v(t)\right) P_v(t) - eP_v(t), \end{aligned} \tag{7}$$

where $m = \gamma_h + \delta_h + \mu_h$. Let

$$\begin{aligned} \Gamma = \Bigg\{ & \left(S_h, E_h, I_h, S_v, E_v, I_v, P_v\right) \in R_7^+ \\ & \mid 0 \le S_h + E_h + I_h \le \frac{b_1}{\mu_h}, \\ & 0 \le S_v + E_v + I_v \le \frac{b_2}{\mu_v}, P_v \ge 0 \Bigg\}. \end{aligned} \tag{8}$$

Obviously, for system (7), the region Γ is positively invariant.

3. The Equilibria and the Basic Reproduction Number

Lemma 1. *The equilibria of system* (7) *are as follows.*

(i) *If the predator is present, that is,* $P_v(t) > 0$ *for any* $t \in [0, +\infty)$*, then there exist a disease-free equilibrium* $E_1(S_h^0, 0, 0, S_v^0, 0, 0, \widetilde{P}_v)$ *and a disease equilibrium* $E_2(\widetilde{S}_h, \widetilde{E}_h, \widetilde{I}_h, \widetilde{S}_v, \widetilde{E}_v, \widetilde{I}_v, \widetilde{P}_v)$ *if* R_0 *is larger than one.*

(ii) *If the predator is absent, that is,* $P_v(t) \equiv 0$ *for any* $t \in [0, +\infty)$*, then there exist a disease-free equilibrium* $E_3((b_1/\mu_h)0, 0, (b_2/\mu_v), 0, 0, 0)$ *and a disease equilibrium* $E_4(\widehat{S}_h, \widehat{E}_h, \widehat{I}_h, \widehat{S}_v, \widehat{E}_v, \widehat{I}_v, 0)$ *if* R_1 *is larger than one.*

where

$$S_h^0 = \frac{b_1}{\mu_h}, \qquad S_v^0 = \widetilde{N}_v,$$

$$\widetilde{S}_h = \frac{Q_1 Q_2 Q_3 \left(\mu_v + h\widetilde{P}_v + \beta_2 \widetilde{I}_h\right)}{\alpha_h \alpha_v \beta_1 \beta_2 \widetilde{N}_v},$$

$$\widetilde{I}_h = \frac{R_0^2 - 1}{Q_1^2 Q_2^2 Q_3 Q_4 \mu_h \left(\mu_h \beta_2 Q_3 + \alpha_v \beta_1 \beta_2 \widetilde{N}_v\right)},$$

$$\widetilde{E}_h = \frac{Q_2}{\alpha_h} \widetilde{I}_h, \qquad \widetilde{S}_v = \frac{b_2}{\mu_v + h\widetilde{P}_v + \beta_2 \widetilde{I}_h},$$

$$\widetilde{E}_v = \frac{\alpha_v \beta_2 \widetilde{N}_v \widetilde{I}_h \left(\mu_v + h\widetilde{P}_v\right)}{\alpha_v Q_3 \left(\mu_v + h\widetilde{P}_v + \beta_2 \widetilde{I}_h\right)},$$

$$\widetilde{I}_v = \frac{\alpha_v \beta_2 \widetilde{N}_v \widetilde{I}_h}{Q_3 \left(\mu_v + h\widetilde{P}_v + \beta_2 \widetilde{I}_h\right)},$$

$$\widehat{S}_h = \frac{Q_1 Q_2 Q_5 \mu_v \left(\mu_v + \beta_2 \widehat{I}_h\right)}{\alpha_h \alpha_v \beta_1 \beta_2 b_2},$$

$$\begin{aligned}
\widehat{I}_h &= \frac{R_1^2 - 1}{Q_1^2 Q_2^2 Q_5 \mu_h \left[\mu_h \mu_v \beta_2 Q_5 + \alpha_v \beta_1 \beta_2 b_2\right]},\\
\widehat{E}_h &= \frac{Q_2}{\alpha_h}\widehat{I}_h, \qquad \widehat{S}_v = \frac{b_2}{\mu_v + \beta_2 \widehat{I}_h},\\
\widehat{E}_v &= \frac{\mu_v \widehat{I}_v}{\alpha_v}, \qquad \widehat{I}_v = \frac{b_2 \alpha_v \beta_2 \widehat{I}_h}{\mu_v Q_5 \left(\mu_v + \beta_2 \widehat{I}_h\right)},\\
Q_1 &= \alpha_h + \mu_h, \qquad Q_2 = m = \gamma_h + \delta_h + \mu_h,\\
Q_3 &= \alpha_v + \mu_v + h\widetilde{P}_v, \qquad Q_4 = \left(\mu_v + h\widetilde{P}_v\right),\\
Q_5 &= \alpha_v + \mu_v, \qquad \widetilde{N}_v = \frac{e}{\varepsilon h},\\
\widetilde{P}_v &= \frac{b_2 - \mu_v \widetilde{N}_v}{h\widetilde{N}_v} \quad \text{if } \frac{b_2}{\mu_v} > \frac{e}{\varepsilon h}.
\end{aligned} \tag{9}$$

Here (b_2/μ_v) is the equilibrium level of total vector population of system (7) *without predators, while $(e/\varepsilon h)$ is the equilibrium level of total vector population of system* (7) *with predators. $(b_2/\mu_v) > (e/\varepsilon h)$ is the sufficient and necessary conditions to ensure $\widetilde{P}_v > 0$. Therefore, from the viewpoint of biological meaning, if the predators are present, we always assume that $(b_2/\mu_v) > (e/\varepsilon h)$.*

$R_0 = \sqrt{(\alpha_h \alpha_v \beta_1 \beta_2 b_1 \widetilde{N}_v/\mu_h Q_1 Q_2 Q_3 Q_4)}$ and $R_1 = \sqrt{(\alpha_h \alpha_v b_1 b_2 \beta_1 \beta_2/\mu_h \mu_v^2 Q_1 Q_2 Q_5)}$, respectively, are the basic reproduction number of system (7) and system (7) without predators by [36, 37].

Remark 2. It is not difficult to find that R_0 is less than R_1. That is, predation results into a reduction in the basic reproduction number R_0 of system (7).

4. Global Stability Analysis

The purpose of this section is to discuss the global stability of the disease-free and disease equilibria of system (7) to obtain the control condition under which diseases can be eradicated by predators preying on vectors. Before giving the main proof, we first give the following Lemma.

Lemma 3. *For system* (4)*, the unique positive equilibrium $(\widetilde{N}_v, \widetilde{P}_v)$ is globally asymptotically stable, where $\widetilde{N}_v$ and $\widetilde{P}_v$ are given in Lemma 1.*

By constructing Lyapunov function $V(t) = (\varepsilon/2)(N_v(t) - \widetilde{N}_v)^2 + \widetilde{N}_v(P_v(t) - \widetilde{P}_v - \widetilde{P}_v \ln(P_v(t)/\widetilde{P}_v))$ and using LaSalle's invariance principle, it is not difficult to prove that the positive equilibrium $(\widetilde{N}_v, \widetilde{P}_v)$ is globally asymptotically stable. Here we omit it.

Theorem 4. *If $R_0 \le 1$ and for any $t \in [0, +\infty)$, $P_v(t) \ge \widetilde{P}_v$; then, the disease-free equilibrium E_1 of system* (7) *is globally asymptotically stable.*

Proof. We construct the following Lyapunov functional:

$$\begin{aligned}
L(t) = {} & w_0 \left(S_h(t) - S_h^0 - S_h^0 \ln \frac{S_h(t)}{S_h^0}\right) + w_1 E_h(t)\\
& + w_2 I_h(t) + w_3 \left(S_v(t) - S_v^0 - S_v^0 \ln \frac{S_v(t)}{S_v^0}\right)\\
& + w_4 E_v(t) + w_5 I_v(t)\\
& + w_6 \left(P_v(t) - \widetilde{P}_v - \widetilde{P}_v \ln \frac{P_v(t)}{\widetilde{P}_v}\right),
\end{aligned} \tag{10}$$

where $w_0 = w_1 = (\alpha_h/Q_1)$, $w_2 = 1$, $w_3 = w_4 = (b_1 \alpha_h \alpha_v \beta_1/\mu_h Q_1 Q_3 Q_4)$, $w_5 = (b_1 \alpha_h \beta_1/\mu_h Q_1 Q_4)$, $w_6 = (w_3/\varepsilon)$.

Calculating the derivative of $L(t)$ along the solution of (7) yields that

$$\begin{aligned}
\frac{dL(t)}{dt} = {} & w_0 \left(\frac{S_h(t) - S_h^0}{S_h(t)}\right)\left[b_1 - \beta_1 S_h(t) I_v(t) - \mu_h S_h(t)\right]\\
& + w_1 \left[\beta_1 S_h(t) I_v(t) - (\alpha_h + \mu_h) E_h(t)\right]\\
& + w_2 \left[\alpha_h E_h(t) - m I_h(t)\right]\\
& + w_3 \left(\frac{S_v(t) - S_v^0}{S_v(t)}\right)\\
& \times \left[b_2 - \beta_2 S_v(t) I_h(t) - \mu_v S_v(t) - h S_v(t) P_v(t)\right]\\
& + w_4 \left[\beta_2 S_v(t) I_h(t) - \alpha_v E_v(t)\right.\\
& \qquad \left. - \mu_v E_v(t) - h E_v(t) P_v(t)\right]\\
& + w_5 \left[\alpha_v E_v(t) - \mu_v S_v(t) - h I_v(t) P_v(t)\right]\\
& + w_6 \left(\frac{P_v(t) - \widetilde{P}_v}{P_v(t)}\right)\\
& \times \left[\varepsilon h \left(S_v(t) + E_v(t) + I_v(t)\right) - e\right] P_v(t).
\end{aligned} \tag{11}$$

By the equilibrium conditions $b_1 = \mu_h S_h^0$ and $b_2 = \mu_v S_v^0 + h S_v^0 \widetilde{P}_v$, it follows that

$$\begin{aligned}
& \frac{dL(t)}{dt}\\
& = -\mu_h w_0 \frac{\left(S_h(t) - S_h^0\right)^2}{S_h(t)} - \mu_v w_3 \frac{\left(S_v(t) - S_v^0\right)^2}{S_v(t)}\\
& \quad + \left(w_1 - w_0\right)\beta_1 S_h(t) I_v(t) + \left(w_4 - w_3\right)\beta_2 S_v(t) I_h(t)\\
& \quad + \left(w_2 \alpha_h - w_1 Q_1\right) E_h(t) + \left(w_5 \alpha_v - w_4 Q_3\right) E_v(t)
\end{aligned}$$

$$+\left[w_3\beta_2\widetilde{N}_v - w_2Q_2\right]I_h(t) + \left[w_0\beta_1\frac{b_1}{\mu_h} - w_5Q_4\right]I_v(t)$$
$$+ w_3h\frac{\left(S_v(t) - S_v^0\right)}{S_v(t)}\left(S_v^0\widetilde{P}_v - S_v(t)P_v(t)\right)$$
$$+ w_4hE_v(t)\left(\widetilde{P}_v - P_v(t)\right)$$
$$+ w_5hI_v(t)\left(\widetilde{P}_v - P_v(t)\right) + w_6\varepsilon hI_v(t)\left(P_v(t) - \widetilde{P}_v\right)$$
$$\times\left(N_v(t) - \widetilde{N}_v\right). \tag{12}$$

Since

$$w_3h\frac{\left(S_v(t) - S_v^0\right)}{S_v(t)}\left(S_v^0\widetilde{P}_v - S_v(t)P_v(t)\right)$$
$$+ w_4hE_v(t)\left(\widetilde{P}_v - P_v(t)\right)$$
$$= -w_3h\widetilde{P}_v\frac{\left(S_v(t) - S_v^0\right)^2}{S_v(t)}$$
$$+ w_3h\left(S_v(t) - S_v^0\right)\left(\widetilde{P}_v - P_v(t)\right), \tag{13}$$

then using $S_h^0 = b_1/\mu_h$, $S_v^0 = \widetilde{N}_v$ we have

$$\frac{dL(t)}{dt}$$
$$= -\mu_h w_0\frac{\left(S_h(t) - (b_1/\mu_h)\right)^2}{S_h(t)} - w_3\left(\mu_v + h\widetilde{P}_v\right)\frac{\left(S_v(t) - \widetilde{N}_v\right)^2}{S_v(t)}$$
$$+ Q_2\left(R_0^2 - 1\right)I_h(t) + \left[w_3h\left(S_v(t) - \widetilde{N}_v\right)\right.$$
$$\left. + w_4hE_v(t) + w_5hI_v(t)\right]$$
$$\times\left(\widetilde{P}_v - P_v(t)\right)$$
$$+ w_6\varepsilon h\left(P_v(t) - \widetilde{P}_v\right)\left(N_v(t) - \widetilde{N}_v\right). \tag{14}$$

After some rearrangement, we have

$$\frac{dL(t)}{dt}$$
$$= -\mu_h w_0\frac{\left(S_h(t) - (b_1/\mu_h)\right)^2}{S_h(t)} - w_3\left(\mu_v + h\widetilde{P}_v\right)\frac{\left(S_v(t) - \widetilde{N}_v\right)^2}{S_v(t)}$$
$$+ Q_2\left(R_0^2 - 1\right)I_h(t) + h\left(w_5 - w_3\right)I_v(t)\left(\widetilde{P}_v - P_v\right). \tag{15}$$

Since $P_v(t) \geq \widetilde{P}_v$, then we have $N_v(t) \leq \widetilde{N}_v$ for any $t \in [0, +\infty)$. Otherwise, if $N_v(t) > \widetilde{N}_v$ for any $t \in [0, +\infty)$, then $\varepsilon hN_v(t) - e > 0$; thus, from the last equation of system (7), we have $(dP_v(t)/dt) > 0$. On the other hand, by Lemma 1, $\lim_{t\to+\infty}P_v(t) = \widetilde{P}_v$, so for any $t \in [0, +\infty)$, $P_v(t) < \widetilde{P}_v$. This contradicts $P_v(t) \geq \widetilde{P}_v$. So $N_v(t) \leq \widetilde{N}_v$ when $P_v(t) \geq \widetilde{P}_v$ for any $t \in [0, +\infty)$. Thus, $(dL(t)/dt) \leq 0$ if $R_0 \leq 1$ and $P_v(t) \geq \widetilde{P}_v$ for any $t \in [0, +\infty)$. Furthermore, $(dL(t)/dt) = 0$ if and only if $S_h = S_h^0$, $S_v = S_v^0 = \widetilde{N}_v$, $E_h = E_v = I_h = I_v = 0$ and $P_v = \widetilde{P}_v$. Consequently, the largest compact invariant set in $\{(S_h(t), E_h(t), I_h(t), S_v(t), E_v(t), I_v(t), P_v(t)) \in \Gamma : dL/dt = 0\}$ when $R_0 \leq 1$ is the singleton E_1. Then by Lyapunov-LaSalle theorem, the equilibrium E_1 is globally stable if $R_0 \leq 1$ and $P_v(t) \geq \widetilde{P}_v$ for any $t \in [0, +\infty)$. □

Remark 5. From Theorem 4 we can see that when $R_0 \leq 1$ the disease-free equilibrium E_1 of system (7) is globally asymptotically stable if the predator population size $P_v(t)$ is not less than the predator equilibrium level $\widetilde{P}_v$, which depends on the total equilibrium vector density $\widetilde{N}_v$. That is, the predator density threshold for successful disease eradication is $P_v(t) = \widetilde{P}_v$. If $P_v(t) \geq \widetilde{P}_v$ and $R_0 \leq 1$ then disease can be eradicated; otherwise; pathogen persists though predators are introduced and $R_0 \leq 1$.

Similar to the proof of Theorem 4, we have the following corollary to show that the disease-free equilibrium E_3 of system (7) in absence of predators is global asymptotically stable.

Corollary 6. *If $R_1 \leq 1$ then the disease-free equilibrium E_3 of system (7) without predators is globally asymptotically stable.*

A global stability result for the endemic equilibrium E_2 of the system (7) is given below.

Theorem 7. *The endemic equilibrium state E_2 of system (7) is globally asymptotically stable if*

$$R_0 > 1, \qquad \alpha_v = \frac{\beta_2\widetilde{S}_v\widetilde{I}_h}{\widetilde{E}_v}, \tag{16}$$

where $\widetilde{S}_v$, $\widetilde{I}_h$ and $\widetilde{E}_v$ are the disease equilibrium value of susceptible vectors, infected hosts, and exposed vectors of system (7).

Proof. Consider the following Lyapunov functional:

$$V(t)$$
$$= c_1\left(S_h(t) - \widetilde{S}_h\ln\frac{S_h(t)}{\widetilde{S}_h}\right) + c_2\left(E_h(t) - \widetilde{E}_h\ln\frac{E_h(t)}{\widetilde{E}_h}\right)$$
$$+ c_3\left(I_h(t) - \widetilde{I}_h\ln\frac{I_h(t)}{\widetilde{I}_h}\right) + c_4\left(S_v(t) - \widetilde{S}_v\ln\frac{S_v(t)}{\widetilde{S}_v}\right)$$
$$+ c_5\left(E_v(t) - \widetilde{E}_v\ln\frac{E_v(t)}{\widetilde{E}_v}\right) + c_6\left(I_v(t) - \widetilde{I}_v\ln\frac{I_v(t)}{\widetilde{I}_v}\right)$$
$$+ c_7\left(P_v(t) - \widetilde{P}_v\ln\frac{P_v(t)}{\widetilde{P}_v}\right), \tag{17}$$

where $c_2 = c_1$, $c_3 = (c_1\beta_1 a_1/d_1)$, $c_4 = c_5 = (c_1\beta_1 a_1/\beta_2 a_2)$, $c_6 = (c_5\beta_2 a_2/d_2)$, $c_7 = (c_4/\varepsilon)$, $\widetilde{S}_h\widetilde{I}_v = a_1$, $\widetilde{S}_v\widetilde{I}_h = a_2$, $d_1 = \alpha_h\widetilde{E}_h$, $d_2 = \alpha_v\widetilde{E}_v$.

Calculate the derivative of $V(t)$ along the solution of (1); this yields that

$$\begin{aligned}
&\frac{dV(t)}{dt} \\
&= c_1\left(1-\frac{\widetilde{S}_h}{S_h(t)}\right)\left[b_1-\beta_1 S_h(t) I_v(t)-\mu_h S_h(t)\right] \\
&\quad + c_2\left(1-\frac{\widetilde{E}_h}{E_h(t)}\right)\left[\beta_1 S_h(t) I_v(t)-(\alpha_h+\mu_h) E_h(t)\right] \\
&\quad + c_3\left(1-\frac{\widetilde{I}_h}{I_h(t)}\right)\left[\alpha_h E_h(t)-mI_h(t)\right] \\
&\quad + c_4\left(1-\frac{\widetilde{S}_v}{S_v(t)}\right) \\
&\quad \times\left[b_2-\beta_2 S_v(t) I_h(t)-\mu_v S_v(t)-hS_v(t) P_v(t)\right] \\
&\quad + c_5\left(1-\frac{\widetilde{E}_v}{E_v(t)}\right) \\
&\quad \times\left[\beta_2 S_v(t) I_h(t)-\alpha_v E_v(t)-\mu_v E_v(t)-hE_v(t) P_v(t)\right] \\
&\quad + c_6\left(1-\frac{\widetilde{I}_v}{I_v(t)}\right) \\
&\quad \times\left[\alpha_v E_v(t)-\mu_v I_v(t)-hI_v(t) P_v(t)\right] \\
&\quad + c_7\left(1-\frac{\widetilde{P}_v}{P_v(t)}\right) \\
&\quad \times\left[\varepsilon h\left(S_v(t)+E_v(t)+I_v(t)\right) P_v(t)-eP_v(t)\right].
\end{aligned} \tag{18}$$

System (7) satisfied the following relations at equilibrium point:

$$\begin{aligned}
b_1 &= \beta_1\widetilde{S}_h\widetilde{I}_v+\mu_h\widetilde{S}_h, \\
(\alpha_h+\mu_h) &= \frac{\beta_1\widetilde{S}_h\widetilde{I}_v}{\widetilde{E}_h}, \\
m &= \frac{\alpha_h\widetilde{E}_h}{\widetilde{I}_h}, \\
b_2 &= \beta_2\widetilde{S}_v\widetilde{I}_h+\mu_v\widetilde{S}_v+h\widetilde{S}_v\widetilde{P}_v, \\
(\alpha_v+\mu_v) &= \frac{\beta_2\widetilde{S}_v\widetilde{I}_h}{\widetilde{E}_v}-h\widetilde{P}_v, \\
\mu_v &= \frac{\alpha_v\widetilde{E}_v}{\widetilde{I}_v}-h\widetilde{P}_v, \\
e &= \varepsilon h\widetilde{N}_v, \qquad \widetilde{N}_v = \widetilde{S}_v+\widetilde{E}_v+\widetilde{I}_v.
\end{aligned} \tag{19}$$

Substituting b_1 up to e in the above equation, we obtain

$$\begin{aligned}
&\frac{dV(t)}{dt} \\
&= -c_1\mu_h\frac{\left(S_h(t)-\widetilde{S}_h\right)^2}{S_h(t)}-c_4\mu_v\frac{\left(S_v(t)-\widetilde{S}_v\right)^2}{S_v(t)} \\
&\quad + c_1\left(1-\frac{\widetilde{S}_h}{S_h(t)}\right)\left[\beta_1\widetilde{S}_h\widetilde{I}_v-\beta_1 S_h(t) I_v(t)\right] \\
&\quad + c_2\left(1-\frac{\widetilde{E}_h}{E_h(t)}\right)\left[\beta_1 S_h(t) I_v(t)-E_h\frac{\beta_1\widetilde{S}_h\widetilde{I}_v}{\widetilde{E}_h}\right] \\
&\quad + c_3\left(1-\frac{\widetilde{I}_h}{I_h(t)}\right)\left[\alpha_h E_h(t)-\alpha_h\widetilde{E}_h\frac{I_h(t)}{\widetilde{I}_h}\right] \\
&\quad + c_4\left(1-\frac{\widetilde{S}_v}{S_v(t)}\right) \\
&\quad \times\left[\beta_2\widetilde{S}_v\widetilde{I}_h-\beta_2 S_v(t) I_h(t)+h\widetilde{S}_v\widetilde{P}_v-hS_v(t) P_v(t)\right] \\
&\quad + c_5\left(1-\frac{\widetilde{E}_v}{E_v(t)}\right) \\
&\quad \times\left[\beta_2 S_v(t) I_h(t)-\beta_2\widetilde{S}_v\widetilde{I}_h\frac{E_v(t)}{\widetilde{E}_v}\right. \\
&\qquad \left.-hE_v(t) P_v(t)+hE_v(t)\widetilde{P}_v\right] \\
&\quad + c_6\left(1-\frac{\widetilde{I}_v}{I_v(t)}\right) \\
&\quad \times\left[\alpha_v E_v(t)-\alpha_v\widetilde{E}_v\frac{I_v}{\widetilde{I}_v(t)}-hI_v(t) P_v(t)+hI_v(t)\widetilde{P}_v\right] \\
&\quad + c_7\left(1-\frac{\widetilde{P}_v}{P_v(t)}\right) \\
&\quad \times\left[\varepsilon h\left(S_v(t)+E_v(t)+I_v(t)-\widetilde{S}_v-\widetilde{E}_v-\widetilde{I}_v\right)\right] P_v(t).
\end{aligned} \tag{20}$$

Set

$$\begin{aligned}
&\frac{S_h}{\widetilde{S}_h}=x_1, \quad \frac{E_h}{\widetilde{E}_h}=x_2, \quad \frac{I_h}{\widetilde{I}_h}=x_3, \\
&\frac{S_v}{\widetilde{S}_v}=x_4, \quad \frac{E_v}{\widetilde{E}_v}=x_5, \quad \frac{I_v}{\widetilde{I}_v}=x_6, \\
&\frac{P_v}{\widetilde{P}_v}=x_7, \quad \widetilde{S}_v\widetilde{P}_v=a_3, \quad \widetilde{E}_v\widetilde{P}_v=a_4, \\
&\widetilde{I}_v\widetilde{P}_v=a_5;
\end{aligned} \tag{21}$$

then, from the above equation, we obtain

$$\begin{aligned}
&\frac{dV(t)}{dt} \\
&= -c_1\mu_h\frac{\left(S_h(t)-\widetilde{S}_h\right)^2}{S_h(t)} - c_4\mu_v\frac{\left(S_v(t)-\widetilde{S}_v\right)^2}{S_v(t)} \\
&\quad + c_1\beta_1a_1 - c_1\beta_1a_1x_1x_6 - c_1\beta_1a_1\frac{1}{x_1} + c_1\beta_1a_1x_6 \\
&\quad + c_2\beta_1a_1 + c_2\beta_1a_1x_1x_6 - c_2\beta_1a_1x_2 - c_2\beta_1a_1\frac{x_1x_6}{x_2} \\
&\quad + c_3d_1 + c_3d_1x_2 - c_3d_1x_3 - c_3d_1\frac{x_2}{x_3} \\
&\quad + c_4\beta_2a_2 + c_4\beta_2a_2x_3 - c_4\beta_2a_2x_3x_4 - c_4\beta_2a_2\frac{1}{x_4} \\
&\quad + c_4ha_3 - c_4ha_3x_4x_7 - c_4ha_3\frac{1}{x_4} + c_4ha_3x_7 \\
&\quad + c_5\beta_2a_2 + c_5\beta_2a_2x_3x_4 - c_5\beta_2a_2x_5 - c_5\beta_2a_2\frac{x_3x_4}{x_5} \\
&\quad - c_5ha_4x_5x_7 + c_5ha_4x_5 + c_5ha_4x_7 - c_5ha_4 \\
&\quad + c_6d_2 + c_6d_2x_5 - c_6d_2\frac{x_5}{x_6} - c_6d_2x_6 \\
&\quad - c_6ha_5x_6x_7 + c_6ha_5x_6 + c_6ha_5x_7 - c_6ha_5 \\
&\quad + c_7\varepsilon ha_3x_4x_7 + c_7\varepsilon ha_4x_5x_7 + c_7\varepsilon ha_5x_6x_7 \\
&\quad - c_7\varepsilon ha_3x_7 - c_7\varepsilon ha_4x_7 - c_7\varepsilon ha_5x_7 \\
&\quad - c_7\varepsilon ha_3x_4 - c_7\varepsilon ha_4x_5 - c_7\varepsilon ha_5x_6 \\
&\quad + c_7\varepsilon ha_3 + c_7\varepsilon ha_4 + c_7\varepsilon ha_5.
\end{aligned} \tag{22}$$

After some rearrangement, we have

$$\begin{aligned}
&\frac{dV(t)}{dt} \\
&= -c_1\mu_h\frac{\left(S_h(t)-\widetilde{S}_h\right)^2}{S_h(t)} - c_5\mu_v\frac{\left(S_v(t)-\widetilde{S}_v\right)^2}{S_v(t)} \\
&\quad + \left(c_1\beta_1a_1 - c_6d_2\right)x_6 + \left(c_3d_1 - c_2\beta_1a_1\right)x_2 \\
&\quad + \left(c_4\beta_2a_2 - c_3d_1\right)x_3 + \left(c_6d_2 - c_5\beta_2a_2\right)x_5 \\
&\quad + \left(c_2\beta_1a_1 - c_1\beta_1a_1\right)x_1x_6 + \left(c_5\beta_2a_2 - c_4\beta_2a_2\right)x_3x_4 \\
&\quad + \left(c_7\varepsilon - c_4\right)ha_3x_4x_7 + \left(c_7\varepsilon - c_5\right)ha_4x_5x_7 \\
&\quad + \left(c_7\varepsilon - c_6\right)ha_5x_6x_7 \\
&\quad + \left(c_4a_3 + c_5a_4 + c_6a_5 - c_7\varepsilon a_3 - c_7\varepsilon a_4 - c_7\varepsilon a_5\right)hx_7 \\
&\quad + \left(c_7\varepsilon ha_4 + c_7\varepsilon ha_5 - c_5ha_4 - c_6ha_5\right) \\
&\quad + \left(c_4ha_3 + c_7\varepsilon ha_3 - c_4ha_3\frac{1}{x_4} - c_7\varepsilon ha_3x_4\right) \\
&\quad + c_1\beta_1a_1 - c_1\beta_1a_1\frac{1}{x_1} - c_2\beta_1a_1\frac{x_1x_6}{x_2} + c_2\beta_1a_1 \\
&\quad - c_3d_1\frac{x_2}{x_3} + c_3d_1 + c_4\beta_2a_2 - c_4\beta_2a_2\frac{1}{x_4} \\
&\quad - c_5\beta_2a_2\frac{x_3x_4}{x_5} + c_5\beta_2a_2 - c_6d_2\frac{x_5}{x_6} + c_6d_2.
\end{aligned} \tag{23}$$

By some reduction, it follows that

$$\begin{aligned}
&\frac{dV(t)}{dt} \\
&= -c_1\mu_h\frac{\left(S_h(t)-\widetilde{S}_h\right)^2}{S_h(t)} - c_5\mu_v\frac{\left(S_v(t)-\widetilde{S}_v\right)^2}{S_v(t)} \\
&\quad + c_4ha_3\left(2 - x_4 - \frac{1}{x_4}\right) \\
&\quad + c_1\beta_1a_1\left(6 - \frac{1}{x_1} - \frac{x_1x_6}{x_2} - \frac{x_2}{x_3} - \frac{1}{x_4} - \frac{x_3x_4}{x_5} - \frac{x_5}{x_6}\right).
\end{aligned} \tag{24}$$

Since the arithmetic mean is greater than or is equal to the geometric mean, then

$$\begin{aligned}
&2 - x_4 - \frac{1}{x_4} \geq 0, \\
&\frac{1}{x_1} + \frac{x_1x_6}{x_2} + \frac{x_2}{x_3} + \frac{1}{x_4} + \frac{x_3x_4}{x_5} + \frac{x_5}{x_6} \geq 6.
\end{aligned} \tag{25}$$

Thus, it follows from (24) that $(dV(t)/dt) \leq 0$ in Ω. The equation $(dV(t)/dt) = 0$ holds if and only if $x_1 = x_2 = x_3 = x_4 = x_5 = x_6 = 1$; that is, $S_h = \widetilde{S}_h$, $E_h = \widetilde{E}_h$, $I_h = \widetilde{I}_h$, $S_v = \widetilde{S}_v$, $E_v = \widetilde{E}_v$, $I_v = \widetilde{I}_v$, $P_v = \widetilde{P}_v$. Therefore, we prove the global stability of the disease in Γ. The maximal compact invariant set in $\{S_h, E_h, I_h, S_v, E_v, I_v, P_v \in \Gamma : dV(t)/dt = 0\}$ is $\{E_2\}$ when $R_0 > 1$ and $\alpha_v = (\beta_3\widetilde{S}_v\widetilde{I}_h/\widetilde{E}_v)$. From the LaSalle's invariance principle, we finish the proof of Theorem 7. □

Similar to the proof of Theorem 7, we have the following corollary to show that the disease equilibrium E_4 of system (7) in absence of predators is globally asymptotically stable.

Corollary 8. *The endemic equilibrium state E_4 of system (7) without predators is globally asymptotically stable if $R_1 > 1$.*

Remark 9. From Theorems 4 and Theorem 7, Corollary 6 and Corollary 8 we find that $R_0 = 1$ and $R_1 = 1$ provide threshold conditions on determining the uniform persistence and extinction of the disease with and without

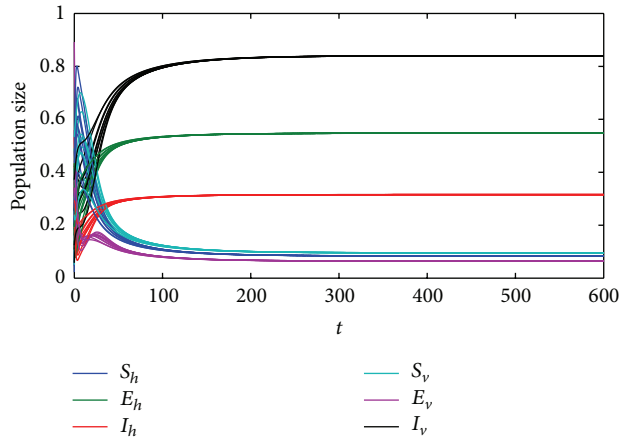

FIGURE 1: Global asymptotically stability of the unique disease equilibrium of system (7) without predators when $R_1 > 1$.

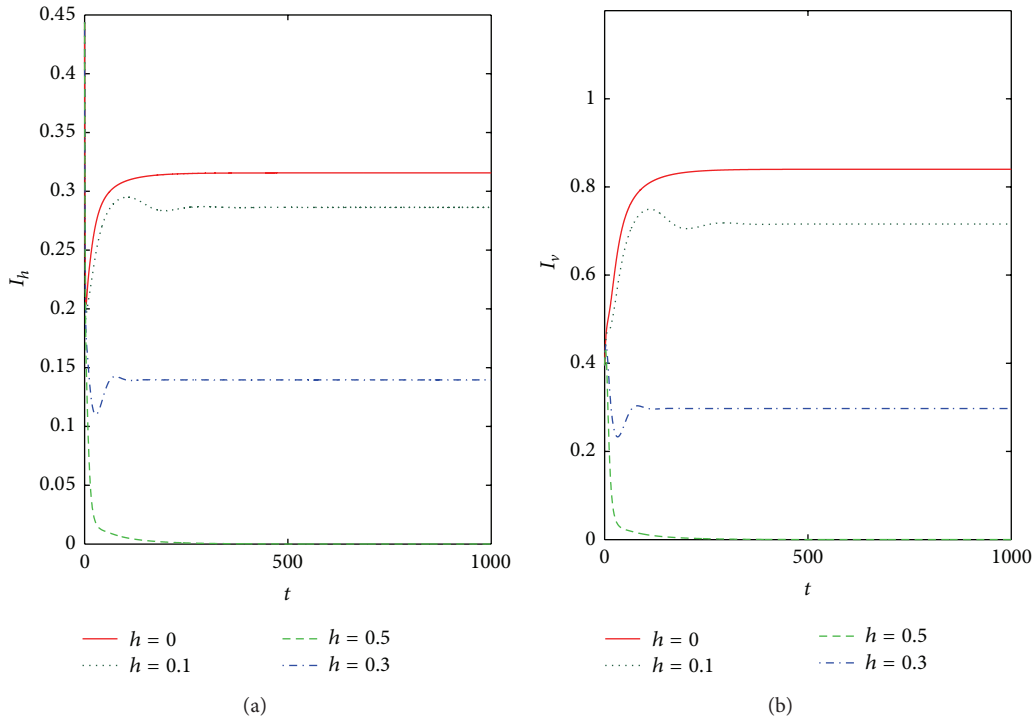

FIGURE 2: The population densities of the infected hosts and vectors of system (7) without and with predators.

predators, respectively. Moreover, we also find that the disease is persistent without predators if $R_1 > 1$, then by introducing predators, the disease will tend to be extinct if $R_0 \leq 1$.

5. Numerical Simulations

In this section, we examine the effects of predators on the transmission dynamics of vector-borne diseases by some numerical simulations. We choose the following set of parameter values (it should be stated that these parameters are chosen for illustrative purpose only and may not necessarily be realistic epidemiologically): $b_1 = 0.786098$, $b_2 = 0.190028$, $\beta_1 = 0.961509$, $\beta_2 = 0.535061$, $\mu_h = 0.914518$, $\mu_v = 0.0150508$, $\alpha_h = 0.645664$, $\alpha_v = 0.19636$, and $m = 1.120469$; By simple calculations, the basic reproduction number $R_1 = 11.2826 > 1$; then, by Corollary 8, the disease equilibrium of system (7) without predators is globally asymptotically stable. That is, the disease persists without predators (see Figure 1).

Choose $e = 0.1$, $\varepsilon = 0.15$; we consider the effect of predators on disease control by comparing equilibrium level of infected hosts and vectors with different values of h in absence of the predators, that is, $h = 0$; then, by the Corollary 8 disease persists (see the solid line of Figure 2). By introducing predators of the vector population, we find that the equilibrium infection levels have been reduced a bit if the

predation rate h is equal to 0.1; however, the disease persists (see the dotted line of Figure 2). Increasing the predation rate such that $h = 0.3$, we find that though the disease still persists, the equilibrium levels of infected hosts and vectors have been greatly lessened (see the dot and dashed line of Figure 2). By enhancing the predation rate h such that $h = 0.5 > h^* = 0.4755$ (h^* satisfies that $R_0 = 1$), then $R_0 = 0.9411 < 1$; therefore, by Theorem 4 the vector-borne disease can be eradicated by introducing vector predators (see the dashed line of Figure 2).

Remark 10. From Figures 1 and 2, we find that predation has a positive effect on vector-host disease control by reducing vector density. Furthermore, disease can be eradicated if predation rate h is large enough such that $R_0(h) \leq 1$ and the predator density $P_v(t)$ satisfies $P_v(t) \geq \widetilde{P}_v$, where $\widetilde{P}_v$ is the predator equilibrium level.

6. Conclusions

In this paper, we propose a host-vector-predator coupled model with variable host and vector population size to investigate the effect of predators on vector-borne disease control by analyzing the global stability of the disease-free and disease equilibria. It is shown that the basic reproduction number R_0 characterizes the disease transmission dynamics: if $R_0 \leq 1$, then there exists only the disease-free equilibrium which is globally asymptotically stable when predator density at any time keeps larger or equal to its equilibrium level; that is, disease tends to be extinct when predators are introduced, and if $R_0 > 1$, then there is a disease equilibrium which is globally asymptotically stable; that is, disease still persists though predators are introduced. As corollaries, the globally stability of system (7) without predators is given. To examine the effect of predator on disease control, numerical simulations are given by choosing to focus on parameter h, which is predation rate. We conclude that predation leads to decrease of equilibrium levels both for infected host and vector population; as h increases, then the infected host and vector equilibrium population will be lessened. Furthermore, if $h > h^*$ (h^* satisfies that $R_0 = 1$), then vector-borne diseases can be eradicated.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

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