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Research Article

Analysis of a Simple Vector-Host Epidemic Model with Direct Transmission

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Vector-host epidemic models with direct transmission are proposed and analyzed. It is shown that the stability of the equilibria in the proposed models can be controlled by the basic reproduction number of the disease transmission. One model considers that the dynamics of human hosts and vectors are described by SIS and SI model, respectively, where the global asymptotical stability for the equilibria of the model is analyzed by constructing Lyapunov function, respectively. The other model considers that the dynamics of the human hosts and vectors are described by SIRS and SI model, respectively, where the global stability of the disease-free equilibrium and the persistence of the disease in the model are also analyzed, respectively.

1. Introduction

Mathematical models of infectious disease have proven to be valuable component for public health planing and responses, as well as an important application of population biology. A simple model may play a significant role in the development of a better understanding of the infectious disease and the various preventive strategies used against it [1–9]. Recently, mathematical models concerning the emergence and reemergence of the vector-host infectious disease have been proposed and analyzed. For example, Esteva and Vargas [10] have investigated an ordinary differential equation compartmental model for the spread of dengue fever. Their results suggested that the disease can be controlled by the threshold parameter R_0 and could persist if and only if R_0 exceeds 1. In [11], a vector-host epidemic mathematical model with demographic structure has been investigated, where the threshold condition for control of the vector diseases transmission has been obtained and the dynamical behavior of the model is globally performed. Epidemiological models with vector host are numerous in the literature [12–16], and we also refer the reader to [1–3] for a general reference since they are not detailed here.

In the aforementioned modeling work on vector-host disease transmissions, many authors consider that these infections diseases, such as malaria, dengue fever, West Nile virus, and so forth, are transmitted to the human population by insects or vectors (e.g., infected mosquitoes). However, some evidences show that direct transmission is possible through blood transfusion, vertically or through needlestick injury. Such models with direct transmission in addition to vector transmission have been also reported in malaria and in Chagas diseases. For example, in the paper in [17], an epidemic model of a vector-host disease with direct transmission and the vector-mediated transmission has been investigated. Recently, the paper in [18] has modeled and analyzed an age-since-infection structured model of Chagas diseases with direct transmission and vector transmission. In this paper, we shall investigate the transmission of a simple vector-host infectious disease by compartmental epidemiological model. A type Ross-MacDonald model for vector-host infectious disease is widely applied, where the host and vector population are divided into the susceptible and infected individuals. Here, we first consider a vector-host epidemic model with direct and vector transmissions. To explore dynamics of the solution of the nonlinear system of differential equations governing the infectious diseases, some mathematical methods and ideas are applied [19–23] in recent years. One of our aims in this paper is to show that the disease-free equilibrium and the endemic equilibrium are, respectively, the global stability by constructing suitable Lyapunov functional. Our results show that the equilibria of the model can be controlled by the basic reproduction number R_0. That is, if R_0 is less than one, the disease-free equilibrium is globally asymptotically stable, and in such a case the endemic equilibrium does not exist; if R_0 is greater than one, then the disease persists and the unique endemic equilibrium is globally asymptotically stable.

Then, we extend the above model by taking into account that the dynamics of the human hosts and vectors are described by SIRS and SI model, respectively. Mathematical analysis of the dynamical behavior of the equilibria in this model is performed. The global stability of the disease-free equilibrium and the persistence of the disease in the model are obtained, respectively.

The paper is organized as follows. In Section 2, a vector-host epidemic model with direct and vector transmissions is presented, where the dynamics of the human hosts and vectors are described by SIS and SI model, respectively. In Section 3, the global stability of equilibria in the model is investigated by constructing suitable Lyapunov function. In Section 4, an extended vector-host epidemic model with direct and vector transmissions is investigated, where the dynamics of the human hosts and vectors are described by SIRS and SI model, respectively. The paper ends with brief remarks.

2. The Model with Host SIS

In this section, a vector-host epidemic model with direct and vector transmissions is presented and investigated, where the dynamics of the host is described by SIS model. It is assumed that there is no immunity in vector population and host population, and the total host population $N_H(t)$ is partitioned into two distinct epidemiological subclasses which are susceptibles and infectious subclasses, with the sizes denoted by $S_H(t)$ and $I_H(t)$, respectively, and the total vector population $N_V(t)$ is divided into susceptibles and infectious, with the sizes denoted by $S_V(t)$ and $I_V(t)$, respectively.

The proposed model satisfies the following assumptions.

(H_1) Susceptible hosts can be infected via two routes of transmission, that is, directly, through a contact with an infected individual (possibly as a result of blood transfusion), and through being bitten by an infectious vector. Thus, we denote the rate of direct transmission by β_1 so that the incidence of new infections via this route is given by a standard incidence rate $\beta_1 S_H(t) I_H(t)/N_H$. We denote the biting rate that a pathogen-carrier vector has to susceptible hosts as β_2, and the incidence of new infections transmitted by the vectors is given again by a standard incidence rate $\beta_2 S_H(t) I_V(t)/N_H$.

(H_2) It is assumed that the host total population N_H is constant. The birth rate and the per-capita natural mortality rate of host are equal, μ. ϕ is the recovery rate of infective hosts.

(H_3) The vector total population N_V is constant, and η is the per-capita natural mortality rate of vector. The host infectious-to-vector susceptible transmission rate is given by $\beta S_V(t) I_H(t)/N_H$.

The dynamics of this infectious disease in the host and vector populations can be described by the following system of nonlinear differential equations:

$$\begin{aligned}
\frac{dS_H}{dt} &= \mu N_H - \frac{\beta_1 S_H I_H}{N_H} - \frac{\beta_2 S_H I_V}{N_V} + \phi I_H - \mu S_H,\\
\frac{dI_H}{dt} &= \frac{\beta_2 S_H I_V}{N_H} + \frac{\beta_1 S_H I_H}{N_H} - (\mu + \phi) I_H,\\
\frac{dS_V}{dt} &= \eta N_V - \eta S_V - \frac{\beta S_V I_H}{N_H},\\
\frac{dI_V}{dt} &= \frac{\beta S_V I_H}{N_H} - \eta I_V.
\end{aligned} \tag{2.1}$$

The feasible region for system (2.1) is R_+^4 (the positive orthant of R_+^4). System (2.1) is obviously well-posed. In order to analyze system (2.1), let $s_h = S_H/N_H$, $i_h = I_H/N_H$, $s_v = S_V/N_V$, $i_V = I_V/N_V$, and $m = N_V/N_H$. For the convenience, here, we still write s_h, i_h, s_v, and i_v as S_H, I_H, S_V, and I_V. So system (2.1) can be reduced to the following equations:

$$\begin{aligned}
\frac{dS_H}{dt} &= \mu - \mu S_H - \beta_2 m S_H I_V - \beta_1 S_H I_H + \phi I_H,\\
\frac{dI_H}{dt} &= \beta_2 m S_H I_V + \beta_1 S_H I_H - (\mu + \phi) I_H,\\
\frac{dS_V}{dt} &= \eta - \eta S_V - \beta S_V I_H,\\
\frac{dI_V}{dt} &= \beta S_V I_H - \eta I_V.
\end{aligned} \tag{2.2}$$

3. Stability of the Equilibria of System (2.2)

It is easy to verify that all of the solutions of system (2.2) exist and are nonnegative. Let

$$\Gamma = \left\{(S_H, I_H, S_V, I_V) \in R_+^4 : S_H, I_H, S_V, I_V \geq 0, S_H + I_H = 1, S_V + I_V = 1\right\}. \tag{3.1}$$

It can be verified that Γ is positively invariant with respect to (2.2). Direct calculation shows that system (2.2) has always the disease-free equilibrium $E_0 = (1, 0, 1, 0)$ in Γ. Let $R_0 = (\beta_2 m/(\mu + \phi))(\beta/\eta) + \beta_1/(\mu + \phi)$. If $R_0 > 1$, then system (2.2) has the unique endemic equilibrium $E^* = (S_H^*, I_H^*, S_V^*, I_V^*)$ in Γ, where

$$S_H^* = \frac{(\mu + \phi)(\eta + \beta I_H^*)}{\beta_1(\eta + \beta I_H^*) + \beta\beta_2 m}, \qquad S_V^* = \frac{\eta}{\eta + \beta I_H^*}, \qquad I_V^* = \frac{\beta I_H^*}{\eta + \beta I_H^*}, \tag{3.2}$$

and I_H^*, which is the unique positive solution of the following equation is given as:

$$f(I_H^*) = a_2(I_H^*)^2 + a_1 I_H^* + a_0, \tag{3.3}$$

where

$$\begin{gathered} a_2 = \beta\beta_1 > 0, a_1 = \eta\beta_1 + \beta\beta_2 m - \beta\beta_1 + (\mu + \phi)\beta, \\ a_0 = -\eta(\mu + \phi)(R_0 - 1) < 0. \end{gathered} \tag{3.4}$$

Remark 3.1. According to Theorem 2 in [24], R_0 is called the basic reproduction number. It represents the average number of people infected directly and indirectly that single infectious host can generate in a totally susceptible population of hosts and vectors.

Now we shall investigate the local geometric properties of the equilibria of the system (2.2). We first give the following results.

Theorem 3.2. *If $R_0 < 1$, the disease-free equilibrium E_0 of model (2.2) is locally asymptotically stable, and is unstable if $R_0 > 1$.*

Proof. Linearizing around the disease-free equilibrium E_0, we obtain the following characteristic equation:

$$(\lambda_1 + \mu)(\lambda_2 + \eta)\left[\lambda^2 + (\eta + \mu + \phi - \beta_1)\right]\lambda + (\mu + \phi)\eta(1 - R_0) = 0. \tag{3.5}$$

Let

$$f(\lambda) = \lambda^2 + A_1\lambda + A_0, \tag{3.6}$$

where

$$A_1 = \eta + \mu + \phi - \beta_1,$$
$$A_0 = (\mu + \phi)\eta(1 - R_0). \tag{3.7}$$

Since it follows that $\mu+\phi > \beta_1$ from $R_0 < 1$, thus $A_0 > 0$ and $A_1 > 0$. So $f(\lambda)$ have two negative roots. So, all of the eigenvalues of the characteristic equation (3.5) are negative real parts. Hence, the equilibrium E_0 is locally asymptotically stable in the interior of Γ. This completes the proof of Theorem 3.2. □

Theorem 3.3. *If $R_0 < 1$, the disease-free equilibrium E_0 of the model (2.2) is globally asymptotically stable.*

Proof. To establish the global stability of the disease-free equilibrium E_0, we construct the following Lyapunov function:

$$L(S_H, I_H, S_V, I_V) = \left(S_H - S_H^* - S_H^* \log \frac{S_H}{N_H}\right) + I_H + \frac{\mu + \phi}{\beta}\left[\left(S_V - S_V^* - S_V^* \log \frac{S_V}{N_H}\right) + I_V\right]. \tag{3.8}$$

By directly calculating the derivation of L along the solution of (2.2), we obtain

$$\begin{aligned}
\frac{dL}{dt} &= (S_H - S_H^*)\frac{S_H'}{S_H} + I'_H + (S_V - S_V^*)\frac{S_V'}{S_V} + I'_V \\
&= \frac{S_H - S_H^*}{S_H}\left[\mu - \mu S_H + \phi I_H - \beta_1 S_H I_H - \beta_2 S_H I_V\right] + \left[\beta_1 S_H I_H + \beta_2 S_H I_V - (\mu + \phi) I_H\right] \\
&\quad + \frac{S_V - S_V^*}{S_V}\left[\eta - \beta S_V I_H - \eta S_V\right] + \left[\beta S_V I_H - \eta I_V\right].
\end{aligned} \tag{3.9}$$

Using $I_H = 1 - S_H$, $S_H^* = 1$, and $S_V^* = 1$, we have

$$\begin{aligned}
\frac{dL}{dt} &= \frac{(S_H - 1)}{S_H}\left[(\mu + \phi)(1 - S_H) - (\beta_1 S_H I_H + \beta_2 S_H I_V)\right] + \left[\beta_1 S_H I_H + \beta_2 S_H I_V - (\mu + \phi) I_H\right] \\
&\quad - \eta\frac{\mu + \phi}{\beta}\frac{(S_V - 1)^2}{S_V} - \frac{\mu + \phi}{\beta}\beta S_V I_H \frac{S_V - 1}{S_V} + \frac{\mu + \phi}{\beta}\left[\beta S_V I_H - \eta I_V\right] \\
&= -(\mu + \phi)\frac{(S_H - 1)^2}{S_H} - \frac{\eta(\mu + \phi)}{\beta}\frac{(S_V - 1)^2}{S_V} - \frac{\eta(\mu + \phi)}{\beta}(1 - R_0) I_V \le 0.
\end{aligned} \tag{3.10}$$

Noting that $dL/dt = 0$ if and only if $S_H = S_H^*$, $S_V = S_V^*$, and $I_H = I_V = 0$, therefore, the largest compact invariant set in $\{(S_H, I_H, S_V, I_V) \in \Gamma : dL/dt = 0\}$ is the singleton $\{E_0\}$, where E_0 is the disease-free equilibrium in system (2.2). By LaSalle's invariant principle [19], E_0 is globally asymptotically stable in Γ.

This completes the proof of Theorem 3.3. □

Now we shall investigate the local geometric properties of the endemic equilibria of system (2.2). We have the following results.

Theorem 3.4. *If $R_0 > 1$, the endemic equilibrium E^* of system (2.2) is locally asymptotically stable.*

Proof. Since the human and vector populations remain constant in Γ, therefore, letting $S_H = 1 - I_H$ and $S_V = 1 - I_V$, system (2.2) in the invariant set Γ can be written as the equivalent to the following two-dimensional nonlinear system:

$$\begin{aligned} \frac{dI_H}{dt} &= \beta_2 m(1 - I_H)I_V + \beta_1(1 - I_H)I_H - (\mu + \phi)I_H, \\ \frac{dI_V}{dt} &= \beta(1 - I_V)I_H - \eta I_V. \end{aligned} \tag{3.11}$$

Thus, the characteristic equation of E^* is

$$\begin{aligned} f(\lambda) &= \lambda^2 + B_1\lambda + B_0 = 0, \\ B_1 &= \beta_2 m I_V^* + 2\beta_1 I_H^* + \mu + \phi - \beta_1 + \beta I_H^* + \eta, \\ B_0 &= (\beta_2 m I_V^* + \beta_1 I_H^*)\eta + \beta_2 m\beta I_V^* + \beta\beta_1 (I_H^*)^2 > 0. \end{aligned} \tag{3.12}$$

Using $\mu + \phi = \beta_1(1 - I_H^*) + \beta_2 m(1 - I_H^*)(I_V^*/I_H^*) > \beta_1(1 - I_H^*)$, it is easy to verify that $B_1 > 0$. Therefore, from (3.12), we obtain that the eigenvalues of $J(E^*)$ have two negative real parts. Therefore E^* is locally asymptotically stable for $R_0 > 1$. □

Finally, we shall give the global stability of the endemic equilibrium E^*. We have the following results

Theorem 3.5. *If $R_0 > 1$, the endemic equilibrium E^* of the model (2.2) is globally asymptotically stable.*

Proof. Let us construct the following Lyapunov function

$$\begin{aligned} V(S_H, I_H, S_V, I_V) = {} & k_1\left(S_H - S_H^* - S_H^* \log\frac{S_H}{N_H}\right) + k_2\left(I_H - I_H^* - I_H^* \log\frac{I_H}{N_H}\right) \\ & + k_3\left(S_V - S_V^* - S_V^* \log\frac{S_V}{N_H}\right) + k_4\left(I_V - I_V^* - I_V^* \log\frac{I_V}{N_H}\right), \end{aligned} \tag{3.13}$$

where

$$k_1 = k_2 = \beta S_V^* I_H^*, \qquad k_3 = k_4 = \beta_2 m S_H^* I_V^* + \beta_1 S_H^* I_H^*. \tag{3.14}$$

By directly calculating the derivation of $V(t)$ along the solution of (2.2), we have

$$
\begin{aligned}
]\frac{dV}{dt} &= k_1(S_H - S_H^*)\frac{S_H'}{S_H} + k_2(I_H - I_H^*)\frac{I_H'}{I_H} + k_3(S_V - S_V^*)\frac{S_V'}{S_V} + k_4(I_H - I_H^*)\frac{I_H'}{I_H} \\
&= \frac{S_H - S_H^*}{S_H}\left[\mu - \mu S_H + \phi I_H - \beta_1 S_H I_H - \beta_2 S_H I_V\right] \\
&\quad + \frac{I_H - I_H^*}{I_H}\left[\beta_1 S_H I_H + \beta_2 S_H I_V - (\mu + \phi) I_H\right] \\
&\quad + \frac{S_V - S_V^*}{S_V}\left[\eta - \beta S_V I_H - \eta S_V\right] + \frac{I_V - I_V^*}{I_V}\left[\beta S_V I_H - \eta I_V\right] \\
&= -\beta S_V^* I_H^* (\mu + \phi)\frac{(S_H - S_H^*)^2}{S_H} - \eta(\beta_2 m S_H^* I_V^* + \beta_1 S_H^* I_H^*)\frac{(S_V - S_V^*)^2}{S_V} \\
&\quad + \beta S_V^* I_H^* (\beta_2 m S_H^* I_V^* + \beta_1 S_H^* I_H^*) \\
&\quad \times \left[\frac{(S_H - S_H^*)}{S_H} - \frac{(S_H - S_H^*)}{S_H(\beta_2 m S_H^* I_V^* + \beta_1 S_H^* I_H^*)} \times (\beta_2 S_H I_V + \beta_1 S_H I_H)\right] \\
&\quad - \beta S_V^* I_H^* (\beta_2 m S_H^* I_V^* + \beta_1 S_H^* I_H^*)\left[\frac{I_H - I_H^*}{I_H I_H^*} + \frac{I_H^*}{I_H}\frac{\beta_2 m S_H I_V + \beta_1 S_H I_H}{\beta_2 m S_H^* I_V^* + \beta_1 S_H^* I_H^*} + \frac{I_H^*}{I_H}\right] \\
&\quad - \beta S_V^* I_H^* (\beta_2 m S_H^* I_V^* + \beta_1 S_H^* I_H^*) \\
&\quad \times \left[\frac{(S_V - S_V^*)}{S_V}\left(1 - \frac{\beta S_V I_H}{\beta S_V^* I_H^*}\right) + \frac{I_V - I_V^*}{I_V}\frac{\beta S_V I_H}{\beta S_V^* I_H^*} - \frac{I_V - I_V^*}{I_V}\right] \\
&= -\beta S_V^* I_H^* (\mu + \phi)\frac{(S_H - S_H^*)^2}{S_H} - \eta(\beta_2 m S_H^* I_V^* + \beta_1 S_H^* I_H^*)\frac{(S_V - S_V^*)^2}{S_V} \\
&\quad - \beta S_V^* I_H^* (\beta_2 m S_H^* I_V^* + \beta_1 S_H^* I_H^*) \\
&\quad \times \left[\frac{S_H^*}{S_H} + \frac{S_V^*}{S_V} + \frac{S_H I_H^* (\beta_2 m I_V + \beta I_H)}{S_H^* I_H (\beta_2 m I_V^* + \beta I_H^*)} + \frac{S_V I_H (\beta_2 m I_V^* + \beta I_H^*)}{S_V^* I_H^* (\beta_2 m I_V + \beta I_H)} - 4\right].
\end{aligned}
\tag{3.15}
$$

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

$$
\frac{S_H^*}{S_H} + \frac{S_V^*}{S_V} + \frac{S_H I_H^* (\beta_2 m I_V + \beta I_H)}{S_H^* I_H (\beta_2 m I_V^* + \beta I_H^*)} + \frac{S_V I_H (\beta_2 m I_V^* + \beta I_H^*)}{S_V^* I_H^* (\beta_2 m I_V + \beta I_H)} \geq 4, \quad \forall S_H, I_H, S_V, I_V \geq 0. \tag{3.16}
$$

Hence, it follows from (3.15) that we obtain $dV/dt \leq 0$. Noting that $dV/dt = 0$ if and only if $S_H = S_H^*$, $S_V = S_V^*$, $I_H = I_H^*$, and $I_V = I_V^*$, therefore, the largest compact invariant set in $\{(S_H, I_H, S_V, I_V) \in \Gamma : dV/dt = 0\}$ is the singleton $\{E^*\}$, where E^* is the disease-free equilibrium in system (2.2). By LaSalle's invariant principle, E^* is globally asymptotically stable in Γ.

This completes the proof of Theorem 3.5. □

Remark 3.6. In this section, by constructing suitable Lyapunov function, it is established in Theorems 3.3 and 3.5 that R_0 is a sharp threshold parameter and completely determines the global stability of (2.2) in the feasible region Γ. We can extend model (2.1) to more stage progression compartments model and establish the global stability of the model by constructing Lyapunov functions of the form $W(x_1, x_2, \ldots, x_n) = \sum_{i=1}^{n} k_i(x_i - x_i^* - x_i^* \log x_i / x_I^*)$.

4. The Model with Host SIRS

In this section, we shall extend the model (2.1) by considering that the dynamics of the host is described by SIRS model. It is assumed that the host populations are constant that is, $S_H + I_H + R_H = N_H$ (*constant*). Similar to model (2.2), by using dimensionless, we obtain $S_H + I_H + R_H = 1$, and $S_V + I_V = 1$. Thus we consider the following differential equation model:

$$\begin{aligned}
\frac{dS_H}{dt} &= \mu - \mu S_H - \beta_2 m S_H I_V + \phi I_H - \beta_1 S_H I_H + \delta(1 - I_H - S_H), \\
\frac{dI_H}{dt} &= \beta_2 m S_H I_V + \beta_1 S_H I_H - (\mu + \phi + \gamma) I_H, \\
\frac{dI_V}{dt} &= \beta(1 - I_V) I_H - \eta I_V,
\end{aligned} \tag{4.1}$$

where γ is the rate at which the host populations acquire immunity. δ is the per-capita rate of loss of immunity in host populations. The other variables and parameters are the same as those of model (2.1). Let $\Omega = \{(S_H, I_H, I_V) \in R_+^3 \mid 0 \le S_H + I_H \le 1, 0 \le I_V \le 1\}$. It is easy to verify that Ω is positively invariant. Now we first investigate the existence of equilibria of (4.1). Letting the equations of system (4.1) with the right-hand side be zero, obviously, $E^0 = (1, 0, 0)$ is always the disease-free equilibrium of system (4.1), and letting $\Re_0 = (\beta\beta_2 m/(\mu + \phi + \gamma)\eta) + (\beta_1/(\mu + \phi + \gamma))$, we can obtain that the unique endemic equilibrium of system (4.1) $E^* = (S_H^*, I_H^*, I_V^*)$ for $\Re_0 > 1$, S_H^*, I_V^* satisfies the following relations:

$$S_H^* = \frac{(\mu + \phi + \gamma) I_H^*}{\beta_2 m I_V^* + \beta_1 I_H^*}, \qquad I_V^* = \frac{\beta I_H^*}{\beta I_H^* + \eta}, \tag{4.2}$$

and I_H^* is the positive solution of the following quadratic polynomial:

$$\beta_1\beta\left(1 + \frac{\gamma}{\mu + \delta}\right)(I_H^*)^2 + \left[(\beta\beta_2 m + \beta_1\eta)\frac{\gamma}{\mu + \delta} + \beta(\mu + \delta) - \beta_1\beta\right] I_H^* + (\mu + \phi + \gamma)\eta(1 - \Re_0) = 0. \tag{4.3}$$

By linearizing system (4.1) around the disease-free equilibrium E^0 of system (4.1) and analyzing the characteristic equation of E^0, it is easy to obtain the following results.

Theorem 4.1. *If $\Re_0 < 1$, the disease-free equilibrium E^0 of system (4.1) is locally asymptotically stable, and is unstable if $\Re_0 > 1$.*

Theorem 4.2. *If $\Re_0 < 1$, then the infection-free equilibrium E^0 of system (4.1) is globally asymptotically stable in Ω.*

Proof. From the last two equations of system (4.1), we have

$$\begin{aligned} I_H' &\le \beta_2 m I_V + \left(\beta_1 - (\mu + \phi + \gamma)\right) I_H, \\ I_V' &\le \beta I_H - \eta I_V. \end{aligned} \tag{4.4}$$

Let us consider the following equations:

$$\begin{aligned} Z_1' &= \beta_2 m Z_2 + \left(\beta_1 - (\mu + \phi + \gamma)\right) Z_1, \\ Z_2' &= \beta Z_1 - \eta Z_2. \end{aligned} \tag{4.5}$$

From $\Re_0 < 1$, we have $\beta\beta_2 m + \beta_1\eta < (\mu + \phi + \gamma)\eta$. It is easy to show that, if $\beta\beta_2 m + \beta_1\eta < (\mu + \phi + \gamma)\eta$ for any solutions of (4.5) with nonnegative initial values, we have $\lim_{t\to\infty} Z_i(t) = 0, i = 1, 2$. Let $0 < I_H(0) \le Z_1(0)$, and $0 < I_V(0) \le Z_2(0)$. If $(Z_1(t), Z_2(t))$ is a solution of system (4.5) with nonnegative initial values $(Z_1(0), Z_1(0))$, then, by comparison principle, we have $I_H(t) \le Z_1(t)$, and $I_V(t) \le Z_2(t)$ for all sufficiently large t. Hence, we have $\lim_{t\to\infty} I_H(t) = 0$, and $\lim_{t\to\infty} I_V(t) = 0$. The maximal compact invariant subset in $\{(S_H, I_H, I_V) \in \Omega : I_H' = I_V' = 0\}$ consists of the S_H-axis. From this set, it is easy to obtain that $S_H \to 1, I_H = 0$, and $I_V = 0$ for $t \ge 0$. It follows that all trajectories starting in Ω approach E^0 for $\Re_0 < 1$.

This completes the proof of Theorem 4.2. □

Theorem 4.3. *If $\Re_0 > 1$, the disease of system (4.1) is uniformly persistent in IntΩ.*

Proof. Similar to the proof of Theorem 3.4 in [23], we choose $X = \Omega$, $X_1 = \operatorname{int}\Omega$, $X_2 = bd(\Omega)$. It is easy to obtain that $Y_2 = \{(S, 0, 0) : 0 < S \le 1\}$ and $\Omega_2 = \bigcup_{y\in Y_2} \omega(y) = \{E^0\}$, and $\{E^0\}$ is an isolated compact invariant set in X. Furthermore, letting $M = \{E^0\}$, thus, M is an acyclic isolated covering of Ω_2.

Now we only need to show that $\{E^0\}$ is a weak repeller for X_1. Suppose that there exists a positive orbit (S_H, I_H, I_V) of (4.1) such that

$$\lim_{t\to+\infty} S_H(t) = 1, \qquad \lim_{t\to+\infty} I_H(t) = 0, \qquad \lim_{t\to+\infty} I_V(t) = 0. \tag{4.6}$$

Since $\Re_0 > 1$, there exists a small enough $\varepsilon > 0$ such that

$$\beta\beta_2 m(1-\varepsilon)^2 + \beta_1(1-\varepsilon)\eta > (\mu + \phi + \gamma)\eta. \tag{4.7}$$

From (4.1), we choose $t_0 > 0$ large enough such that, when $t \ge t_0$, we have

$$\begin{aligned} I_H' &> \beta_2 m(1-\varepsilon) I_V + \left(\beta_1(1-\varepsilon) - (\mu + \phi + \gamma)\right) I_H, \\ I_V' &> \beta(1-\varepsilon) I_H - \eta I_V. \end{aligned} \tag{4.8}$$

Consider the following matrix M_ε defined by

$$M_\varepsilon = \begin{pmatrix} \beta_1(1-\varepsilon) - (\mu + \phi + \gamma) & \beta_2 m(1-\varepsilon) \\ \beta(1-\varepsilon) & -\eta \end{pmatrix}. \tag{4.9}$$

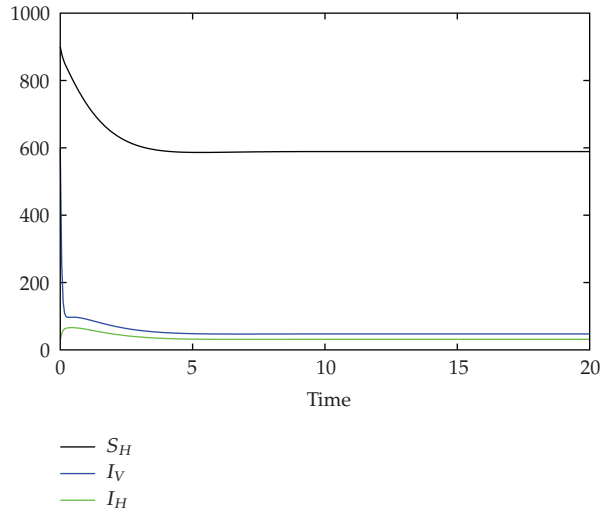

Figure 1: Variation of S_H, I_H, and I_V with time for the parameter values $\mu = 0.00042$, $\beta_1 = 0.00004$, $\beta_2 = 0.00002$, $\beta = 0.00003$, $\alpha = 0.01$, $m = 0.005$, $\gamma = 0.0012$, and $\delta = 0.00002$ when $\Re_0 = 17.0124$.

Since M_ε admits positive off-diagonal element, the Perron-Frobenius Theorem [19] implies that there is a positive eigenvector $v = (v_1, v_2)$ for the maximum eigenvalue λ^* of M_ε. From (4.7), we see that the maximum eigenvalue λ^* is positive. Let us consider the following system:

$$\begin{aligned} \frac{du_1}{dt} &= \beta_2 m(1-\varepsilon)u_2 + \big(\beta_1(1-\varepsilon) - (\mu+\phi+\gamma)\big)u_1, \\ \frac{du_2}{dt} &= \beta(1-\varepsilon)u_1 - \eta u_2. \end{aligned} \tag{4.10}$$

Let $u(t) = (u_1(t), u_2(t))$ be a solution of (4.10) through (lv_1, lv_2) at $t = t_0$, where $l > 0$ satisfies $lv_1 < I_H(t_0)$, and $lv_2 < I_V(t_0)$. Since the semiflow of (4.10) is monotone and $M_\varepsilon v > 0$, it follows that $u_i(t)$ are strictly increasing and $u_i(t) \to +\infty$ as $t \to +\infty$, contradicting the eventual boundedness of positive solutions of system (4.1). Thus, E^0 is weak repeller for X_1.

This completes the proof of Theorem 4.3. □

Remark 4.4. In this section, although we have not discussed the stability of E^* in model (4.1) (this can be achieved via a tedious process, involving the determination of the signs of the eigenvalues of the corresponding Jacobian), numerical simulation (Figure 1) confirms that the equilibrium (E^*) in model (4.1) is stable whenever it exists.

5. The Concluding Remarks

Malaria, dengue fever, and so forht are very sever vector-host disease in some developing countries where hygienic and cultural conditions are inadequate. Despite the improvements in these conditions in the past decades, the endemic levels of these diseases have not tended to decrease; on the contrary, the endemic level in some countries has increased from initial incidence being about 60 per 100,000 yearly to the present 110 per 100,000 [25]. In this paper,

mathematical models for a vector-host disease transmission are proposed and analyzed. Our models seem to be quite robust in their qualitative behavior. The constant human recruitment rate and exponential natural death, as well as vector population with asymptotically constant, are incorporated into the model. The basic reproduction numbers of the model (2.1) and the extended models (4.1) are obtained, respectively. The dynamics behavior of the models is determined by their basic reproduction number, respectively. That is, if $R_0(\mathfrak{R}_0) \leq 1$, the disease-free equilibrium is globally asymptotically stable. If $R_0 > 1$, the disease persists and the unique endemic equilibrium is globally asymptotically stable. Additionally, we show that, if $\mathfrak{R}_0 > 1$, system (4.1) has a unique positive equilibrium. Numerical simulations suggest that the unique endemic equilibrium is globally asymptotically stable whenever it exists, and we conjecture that the unique positive equilibrium is globally asymptotically stable. The simple model treated in this paper shows that direct transmission rate has played a very important role into the diseases transmission, besides indirect transmission rate, mean duration of host carriers, mean life of vector in the environment, the transmission rate of the host infected to vector susceptible, and so forth.

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