Research Article

Stability Analysis of a Vector-Borne Disease with Variable Human Population

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Received 25 November 2012; Revised 1 March 2013; Accepted 2 March 2013

Academic Editor: Ferenc Hartung

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A mathematical model of a vector-borne disease involving variable human population is analyzed. The varying population size includes a term for disease-related deaths. Equilibria and stability are determined for the system of ordinary differential equations. If \( R_0 \leq 1 \), the disease-“free” equilibrium is globally asymptotically stable and the disease always dies out. If \( R_0 > 1 \), a unique “endemic” equilibrium is globally asymptotically stable in the interior of feasible region and the disease persists at the “endemic” level. Our theoretical results are sustained by numerical simulations.

1. Introduction

Vector-borne diseases such as malaria, dengue fever, plague, and West Nile fever are infectious diseases caused by the influx of viruses, bacteria, protozoa, or rickettsia which are primarily transmitted by disease-transmitting biological agents, called vectors. A vector-borne disease is transmitted by a pathogenic microorganism from an infected host to another organism results to form from an infection by blood-feeding arthropods [1].

Vector-borne diseases, in particular, mosquito-borne disease, are transmitted to humans by blood-sucker mosquitoes, which have been a big problem for the public health in the world. The literature dealing with the mathematical theory on vector-borne diseases is quite extensive. Many mathematical models concerning the emergence and reemergence of the vector-host infectious disease have been proposed and analyzed in the literature [2, 3].

By direct transmission models, we mean that the infection moves from person to person directly, with no environmental source, intermediate vector, or host. In a vector-host model, direct transmission may take place by transfusion-related transmission, transplantation-related transmission, and needle-stick-related transmission [4]. Some models have been developed to study the dynamics of a vector-borne disease that considers a direct mode of transmission in human host population [5–7].

Mathematical modeling has proven to play an important role in gaining some insights into the transmission dynamics of infectious diseases and suggest control strategies. Appropriate mathematical models can provide a qualitative assessment for the problem. Some mathematical models discussed in [8–10] provide, best understanding about the dynamics and control of infectious diseases. Immense literature on the use of mathematical models for communicable diseases is available [11, 12]. The assumption of constant population size in epidemiological models is usually valid when we study the diseases of short duration with limited effects on mortality. It may not be valid when dealing with endemic diseases such as malaria, which has a high mortality rate. Ngwa and Shu [13] assumed density-dependent death rates in both vector and human populations, so that the total populations are varying with time that includes disease-related deaths. Esteva and Vargas [14] analyzed the effect of variable host population size and disease-induced death rate. Recently Ozair et. al analyzed vector-host disease model with nonlinear incidence [15].
In this paper, based on the ideas posed in [6, 14], we develop and analyze a vector-host disease model considering a direct mode of transmission as well as a variable human population. The aim of this paper is to establish stability properties of equilibria and the threshold parameter $R_0$ that completely determines the existence of endemic or disease-free equilibrium. If $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable. If $R_0 > 1$, a unique endemic equilibrium exists and is globally asymptotically stable under parametric restrictions. However, in numerical simulations it is shown that the disease still can be "endemic" even if the conditions are violated.

The rest of the paper is organized as follows. In Section 2, we present a formulation of the extended mathematical model. The dimensionless formulation of proposed model is carried out in Section 3. Section 4 devotes existence and uniqueness of "endemic" equilibria. In Section 5, we use Lyapunov function theory to show global stability of disease-free equilibrium and geometric approach to prove global stability of "endemic" equilibrium. Discussions and simulations are done in Section 6.

2. Model Formulation

The human population is partitioned into subclasses of individuals who are susceptible, infectious, and recovered, with sizes denoted by $S_h(t)$, $I_h(t)$, and $R_h(t)$, respectively. The vector population is subdivided into susceptible and infectious vectors, with sizes denoted by $S_v(t)$ and $I_v(t)$, respectively. The mosquito population does not have an immune class, since their infective period ends with their death. Thus, $N_h(t) = S_h(t) + I_h(t) + R_h(t)$ and $N_v(t) = S_v(t) + I_v(t)$ are, respectively, the total human and vector populations at time $t$. The model is given by the following system of differential equations:

$$\frac{dS_h(t)}{dt} = b_1N_h - \frac{\beta_1 S_h I_h}{N_v} - \mu_h S_h,$$

$$\frac{dI_h(t)}{dt} = \frac{\beta_1 S_h I_h}{N_v} - \mu_h I_h - \gamma I_h - \delta_h I_h,$$

$$\frac{dR_h(t)}{dt} = \gamma h - \mu_h R_h,$$

$$\frac{dS_v(t)}{dt} = \mu_v N_v - \frac{\beta_2 S_v I_h}{N_h} - \mu_v S_v,$$

$$\frac{dI_v(t)}{dt} = \beta_2 S_v I_h - \mu_v I_v.$$

In model (1), $b_1$ is the recruitment rate of humans into the population which is assumed to be susceptible. Susceptible hosts get infected via two routes of transmission, through a contact with an infected individual and through being bitten by an infectious vector. We denote the infection rate of susceptible individuals which results from effective contact with infectious individuals by $\beta_1$ and $\beta_2$ is the infection rate of susceptible humans resulting due to the biting of infected vectors. The incidence of new infections via direct and indirect route of transmission is given by the standard incidence form $\beta_1(S_h I_h/N_v)$ and $\beta_2(S_v I_h/N_h)$, respectively. The term $\mu_h$ is the natural mortality rate of humans. We assume that infectious individuals acquire permanent immunity by the rate $\gamma$. The infectious humans suffer from disease-induced death at a rate $\delta_h$. The recruitment and natural death rate of vector population is assumed to be $\mu_v$. The susceptible vectors become infectious as a result of biting effect of infectious humans at a rate $\beta_v$, so that the incidence of newly infected vectors is again given by standard incidence form $\beta_v(S_v I_h/N_h)$. The total human population is governed by the following equation:

$$\frac{dN_h}{dt} = b_1N_h - \mu_h N_h - \delta_h I_h. \quad (2)$$

3. Dimensionless Formulation

Denote $s_h = S_h/N_h$, $i_h = I_h/N_h$, $r_h = R_h/N_h$, $s_v = S_v/N_v$, and $i_v = I_v/N_v$. It is easy to verify that $s_h$, $i_h$, $r_h$, $s_v$, and $i_v$ satisfy the following system (see the Appendix details for):

$$\frac{ds_h(t)}{dt} = b_1 \left(1 - s_h\right) - \beta_1 s_h i_h - \beta_2 s_h i_v + \delta s_h i_h,$$

$$\frac{di_h(t)}{dt} = \beta_1 s_h i_h + \beta_2 s_h i_v - \left(b_1 + \gamma + \delta_h\right) i_h + \delta_h i_h^2,$$

$$\frac{dr_h(t)}{dt} = \gamma h - b_1 r_h + \delta_h i_h r_h,$$

$$\frac{ds_v(t)}{dt} = \mu_v \left(1 - s_v\right) - \beta_2 s_v i_h,$$

$$\frac{di_v(t)}{dt} = \beta_2 s_v i_h - \mu_v i_v,$$

where solutions are restricted to $s_h + i_h + r_h = 1$ and $s_v + i_v = 1$. Before analyzing the unnormalized model (1) and (2), we consider the normalized model (3) by scaling, and so we can study the following reduced system that describes the dynamics of the proportion of individuals in each class

$$\frac{ds_h(t)}{dt} = b_1 \left(1 - s_h\right) - \beta_1 s_h i_h - \beta_2 s_h i_v + \delta s_h i_h,$$

$$\frac{di_h(t)}{dt} = \beta_1 s_h i_h + \beta_2 s_h i_v - \left(b_1 + \gamma + \delta_h\right) i_h + \delta_h i_h^2,$$

$$\frac{dr_h(t)}{dt} = \beta_2 s_v i_h + \delta s_v i_h,$$

determining $r_h$ from

$$\frac{dr_h(t)}{dt} = \gamma h i_h - b_1 r_h + \delta_h i_h r_h,$$

or from $r_h = 1 - s_h - i_h$ and $s_v$ from $s_v = 1 - i_v$, respectively. The correlation between normalized and unnormalized models is explained in the Appendix. Throughout this work, we study the reduced system (4) in the closed, positively invariant set

$$\Gamma = \{(s_h, i_h, i_v) \in R_3^+ : 0 \leq s_h + i_h \leq 1, 0 \leq i_v \leq 1\},$$

where $R_3^+$ denotes the nonnegative cone of $R^3$ with its lower dimensional faces.
4. Existence of Equilibria

We seek the conditions for the existence and stability of the disease-“free” equilibrium (DFE) $E_0(s_{00}, 0, 0)$ and the “endemic” proportion equilibrium $E^*(s^*_h, i^*_h, i^*_v)$. Obviously, $E_0(1, 0, 0) \in \Gamma$ is the DFE of (4), which exists for all positive parameters. The Jacobian matrix of (4) at an arbitrary point $E(s_h, i_h, i_v)$ takes the following form:

$$f(E) = 
\begin{pmatrix}
-b_v - \beta_i i_v - \delta_i i_v - \delta_h i_h & - (\beta_i - \delta_i) s_h & -b_v s_h \\
\beta_v s_h + b_v i_v & \beta_v s_h - (b_v + \gamma_h + \delta_h) + 2 \delta_i i_h & -\beta_v s_h \\
0 & \beta_v (1 - i_v) & -\beta_v s_h - \mu_v 
\end{pmatrix}.$$  

(6)

To analyze the stability of DFE, we calculate the characteristic equation of $J(E)$ at $E = E_0$ as follows:

$$(\lambda + b_1) \left( \lambda^2 + \lambda \left( \mu_v + b_1 + \gamma_h + \delta_h - \beta_1 \right) + \mu_v \left( b_1 + \gamma_h + \delta_h \right) (1 - R_0) \right),$$  

(7)

where

$$R_0 = \frac{\beta_v}{b_1 + \gamma_h + \delta_h} + \frac{\beta_i \beta_3}{\mu_v (b_1 + \gamma_h + \delta_h)}.$$  

(8)

By Routh Hurwitz criteria [16], all roots of (7) have negative real parts if and only if $R_0 < 1$. So, $E_0$ is locally asymptotically stable for $R_0 < 1$. If $R_0 > 1$, the characteristic equation (7) has positive eigenvalue, and $E_0$ is thus unstable. We established the following theorem.

Theorem 1. The disease-free equilibrium is locally asymptotically stable whenever $R_0 < 1$ and unstable for $R_0 > 1$. 

In order to find the “endemic” equilibrium of (4), we set the right hand side of (4) equal to zero and get

$$i_v^* = \frac{\beta_2 s_h^*}{\mu_v + \beta_3 i_v^*},$$  

(9)

$$s_h^* = \frac{b_1 \left( \mu_v + \beta_3 i_v^* \right) \left( b_1 + \beta_3 - \delta_h \right) i_v^*}{\left( \mu_v + \beta_3 i_v^* \right) \left( b_1 + \beta_3 - \delta_h \right) i_v^* + \beta_2 \beta_3 i_v^*},$$  

where $i_v^*$ is a positive solution of the equation

$$f (i_v^*) = A_3 i_v^* + A_2 i_v^* + A_1 i_v^* + A_0 = 0,$$  

(10)

where

$$A_3 = \beta_3 \delta_h (\beta_1 - \beta_2),$$  

$$A_2 = \beta_2 \beta_3 \delta_h + \mu_v \delta_h - \beta_3 \left( b_1 + \gamma_h + \delta_h \right) (\beta_1 - \beta_2),$$  

$$A_1 = b_1 \left( \mu_v \delta_h + \beta_1 \beta_3 \right) - \left( b_1 + \gamma_h + \delta_h \right) \left( \beta_2 \beta_3 + b_1 \beta_3 + \mu_v (\beta_1 - \beta_2) \right),$$_

(11)

$$A_0 = \left( b_1 + \gamma_h + \delta_h \right) b_1 \mu_v (R_0 - 1).$$

From right hand side of (5), we have $\gamma_i h^* (b_1 - \delta_i i_v^*) r_v^* > 0$ and second equation of (9) $\beta_i \mu_v + \beta_2 \beta_3 - \mu_v \delta_h > \beta_3 \delta_h i_v^*$, which means that

$$0 < i_v^* \leq \min \left\{ 1, \frac{b_1}{\delta_h} \left( \frac{\beta_1 \mu_v + \beta_2 \beta_3}{\mu_v \delta_h - 1} \right) \frac{\mu_v}{\beta_3} \right\}.$$  

(12)

If $(\beta_1 \mu_v + \beta_2 \beta_3) / \mu_v \delta_h \leq 1$, there is no positive $i_v^*$, and therefore the only equilibrium point in $\Gamma$ is $E_0$. Note that this is a special case of $R_0 < 1$.

Assume that $R_0 > 1$.

(1) If $\beta_3 > \delta_h$, then $A_1 > 0$, we have $f (-\infty) < 0$, $f (\infty) > 0$ and $f (0) = A_0 > 0$. Further, $f (1) < 0$ (if $b_1 / \delta_h \geq 1$) and $f (b_1 / \delta_h) > 0$. Thus, there exists unique $i^*_h$ such that $f (i^*_h) = 0$ (see Figure 1).

(2) If $\beta_3 = \delta_h$, then $A_1 = 0$ and $f (i^*_h) = A_2 i^*_h + A_1 i^*_h + A_0$, where $A_2 = \beta_2 \beta_3 \delta_h + b_1 \beta_3 \delta_h > 0$. We observe that $f (-\infty) > 0$, $f (\infty) > 0$ and $f (0) = A_0 > 0$. Moreover, $f (1) < 0$ (if $b_1 / \delta_h \geq 1$) and $f (b_1 / \delta_h) > 0$. Therefore, there exists unique $i^*_h$ such that $f (i^*_h) = 0$ (see Figure 2).

(3) If $\beta_3 < \delta_h$, then $A_1 < 0$, we have $f (-\infty) > 0$, $f (\infty) < 0$ and still $f (0) = A_0 > 0$, $f (1) < 0$ (if $b_1 / \delta_h \geq 1$), $f (b_1 / \delta_h) < 0$. In this case, we can say that there is only one root or three roots in the interval $(0, 1)$ if $b_1 / \delta_h \geq 1$ or $(0, b_1 / \delta_h)$ if $b_1 / \delta_h < 1$.

We know that $f (i^*_h) = 0$ has three real roots if and only if

$$\frac{\beta^2}{4} + \frac{\beta^3}{27} \leq 0.$$  

(13)
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\[ p = \frac{A_1}{A_3} - \frac{(A_2)^2}{3(A_3)^2}, \]

\[ q = \frac{A_0}{A_3} - \frac{A_1A_2}{3(A_3)^2} + \frac{2(A_2)^3}{27(A_3)^3}, \]

or

\[ \tilde{R}_1 = \frac{18A_0A_1A_2A_3 - 4A_0(A_2)^3 - 4(A_1)^3A_3 + (A_1)^2(A_2)^2}{27(A_0)^2(A_3)^2} \geq 1. \]

If \( \tilde{R}_1 < 1 \), there is unique \( i^*_h \) such that \( f(i^*_h) = 0 \) in the feasible interval.

If \( \tilde{R}_1 > 1 \), there are three different real roots for \( f(i^*_h) = 0 \) say \( i^*_{h1}, i^*_{h2}, i^*_{h3} \) \( (i^*_{h1} < i^*_{h2} < i^*_{h3}) \). Note that, differentiating with respect to \( i^*_h \), we obtain

\[ f'(i^*_h) = 3A_3i^*_h - 2A_2i^*_h + A_1. \]

The three different real roots for \( f(i^*_h) = 0 \) are in the feasible interval if and only if the following inequalities are satisfied:

\[ 0 < \frac{-A_2}{3A_3} < 1, \]

\[ f'(0) = A_1 < 0, \]

\[ f'(1) = 3A_3 + 2A_2 + A_1 < 0 \quad \left( \text{if } \frac{b_1}{\delta_h} \geq 1 \right), \]

\[ f'(\frac{b_1}{\delta_h}) = 3A_3\left(\frac{b_1}{\delta_h}\right)^2 + 2A_2\left(\frac{b_1}{\delta_h}\right) + A_1 < 0 \quad \left( \text{if } \frac{b_1}{\delta_h} < 1 \right). \]

If \( \tilde{R}_1 = 1 \), there are three real roots for \( f(i^*_h) = 0 \), in which at least two are identical. Similarly, if inequalities (17) are satisfied, then there are three real roots for \( f(i^*_h) = 0 \) in the feasible interval, say \( i^*_{h1}, i^*_{h2}, i^*_{h3} \) \( (i^*_{h1} = i^*_{h2}) \).

Assume that \( R_0 = 1 \).

(1) If \( \beta_1 = \delta_h \), then \( A_3 = 0 \) and (10) reduces to

\[ i^*_h (A_3 i^*_h + A_1) = 0, \]

which implies that \( i^*_h = 0 \) or \( i^*_h = -A_1/A_2 \), which is positive but it lies outside the interval \((0, 1)\) if \( b_1/\delta_h \geq 1 \) or \((0, b_1/\delta_h)\) if \( b_1/\delta_h < 1 \).
In summary, regarding the existence and the number of the “endemic” equilibria, we have the following.

**Theorem 2.** Suppose that $\beta_1 \geq \delta_h$. There is always a disease-free equilibrium for system (4), if $R_0 > 1$, then there is a unique “endemic” equilibrium $E^*$ $(s^*_i, i^*_i, \Gamma^*_i)$ with coordinates satisfying (9) and (10) besides the disease-free equilibrium.

### 5. Global Dynamics

#### 5.1. Global Stability of the Disease-Free Equilibrium

In this subsection, we analyze the global behavior of the equilibria for system (4). The following theorem provides the global property of the disease-free equilibrium $E_0$ of the system.

**Theorem 3.** If $R_0 \leq 1$, then the infection-free equilibrium $E_0$ is globally asymptotically stable in the interior of $\Gamma$.

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

$$L(t) = \mu_s i_h(t) + \beta_2 i_v(t).$$

Calculating the time derivative of $L$ along (4), we obtain

$$L'(t) = \mu_s i'_h(t) + \beta_2 i'_v(t)$$

$$= \mu_s [\beta_1 s h_i + \beta_2 s i'_v] - (b_1 + \gamma_h + \delta_h) i_h$$

$$+ \delta_h i'_h + \beta_2 (\beta_3 s h_i - \mu i_v)$$

$$= \mu_s [\beta_1 (1 - i_h) i_h + \beta_2 (1 - i_h) i_v - (b_1 + \gamma_h + \delta_h) i_h$$

$$+ \delta_h i'_h + \beta_2 (\beta_3 (1 - i_v) i_h - \mu i_v)$$

$$= \mu_s [\beta_1 i_h - \beta_1 i'_h] + \beta_2 i_v - \beta_2 i'_h$$

$$= \mu_s [\beta_1 i_h - \beta_1 i'_h] + \beta_2 i_v - \beta_2 i'_h - (b_1 + \gamma + \delta_h) i_h$$

$$+ \delta_h i'_h + \beta_2 (\beta_3 i_h - \beta_3 i'_h - \mu i_v)$$

$$= \mu_s \beta_1 i_h - \mu_s \beta_1 i'_h + \mu_s \beta_2 i_v - \mu_s \beta_2 i'_h$$

$$- \mu_v (b_1 + \gamma + \delta_h) i_h + \mu_v \delta_h i'_h + \beta_2 \beta_3 i_h - \beta_2 \beta_3 i'_h -$$

$$\beta_2 \mu v i_v$$

$$= -\mu_v (b_1 + \gamma + \delta_h) (1 - R_0) i_h - \mu_v (\beta_1 - \delta_h) i'_h$$

$$+ \mu s \beta_2 i_v - \beta_2 i'_h.$$

(20)

Thus, $L'(t)$ is negative if $R_0 \leq 1$ and $L' = 0$ if and only if $i_h = 0$. Consequently, the largest compact invariant set in $(S_h, s_h, i_v) \in \Gamma$, $L' = 0$, when $R_0 \leq 1$, is the singleton $\{E_0\}$. Hence, LaSalle’s invariance principle [16] implies that “$E_0$” is globally asymptotically stable in $\Gamma$. This completes the proof.

#### 5.2. Global Stability of “Endemic” Equilibrium

Here, we use the geometrical approach of Li and Muldowney to investigate the global stability of the endemic equilibrium $E^*$ in the

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(2) If $\beta_1 > \delta_h$, then $A_3 > 0$, we have $i'_h (A_3 i^*_h + A_2 i^*_h + A_1) = 0$, which implies that $i^*_h = 0$ or $i^*_h$ is the solution of the equation

$$g(i^*_h) = A_3 i^*_h + A_2 i^*_h + A_1 = 0,$$

(18)

where $g(-\infty) > 0$, $g(\infty) > 0$, and $g(0) = A_1 < 0$. Moreover, $g(1) < 0$ (if $b_1/\delta_h \geq 1$) and $g(b_1/\delta_h) < 0$ if $b_1/\delta_h < 1$. Therefore, there exists no $i^*_h$ such that $g(i^*_h) = 0$ in the interval $(0, 1)$ if $b_1/\delta_h \geq 1$ or $(0, b_1/\delta_h)$ if $b_1/\delta_h < 1$. 

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Figure 5: $b_1 = 2; \beta_1 = 0.025; \beta_2 = 0.65; \beta_3 = 0.75; \gamma_h = 0.000045; \mu_s = 0.2; \beta_1 > \delta_h = 0.000025; R_0 = 1.23.$

Figure 6: $b_1 = 2; \beta_1 = 0.0025; \beta_2 = 0.65; \beta_3 = 0.75; \gamma_h = 0.000045; \mu_s = 0.2; \beta_1 = \delta_h = 0.000025; R_0 = 1.22.$
feasible region $\Gamma$. We have omitted the detailed introduction of this approach, and we refer the interested readers to see [17]. We summarize this approach below.

Consider a $C^1$ map $f : x \mapsto f(x)$ from an open set $D \subset \mathbb{R}^n$ to $\mathbb{R}^n$ such that each solution $x(t, x_0)$ to the differential equation

$$x' = f(x),$$

is uniquely determined by the initial value $x(0, x_0)$. We have the following assumptions:

$(H_1)$ $D$ is simply connected;

$(H_2)$ there exists a compact absorbing set $K \subset D$;

$(H_3)$ (21) has a unique equilibrium $E$ in $D$.

Let $P : x \mapsto P(x)$ be a nonsingular $(\frac{n}{2}) \times (\frac{n}{2})$ matrix-valued function which is $C^1$ in $D$ and a vector norm $|\cdot|$ on $\mathbb{R}^N$, where $N = (\frac{n}{2})$.

Let $\mu$ be the Lozinski measure with respect to the $|\cdot|$. Define a quantity $\bar{d}_2$ as

$$\bar{d}_2 = \limsup_{t \to \infty} \sup_{x \in K} \frac{1}{t} \int_0^t \mu(B(x(s, x_0))) \, ds,$$  \hspace{1cm} (22)

where $B = [P_j P^{-1} + \frac{1}{2} J[j]] P^{-1}$, the matrix $P_j$ is obtained by replacing each entry $p$ of $P$ by its derivative in the direction of $f$, $(p_{ij})_j$, and $J[j]$ is the second additive compound matrix of the Jacobian matrix $J$ of (21). The following result has been established in Li and Muldowney [17].

**Theorem 4.** Suppose that $(H_1)$, $(H_2)$, and $(H_3)$ hold, the unique endemic equilibrium $E^*$ is globally stable in $\Gamma$ if $\bar{d}_2 < 0$.

Obviously $\Gamma$ is simply connected and $E^*$ is a unique endemic equilibrium for $R_0 > 1$ in $\Gamma$. To apply the result of the above theorem for global stability of endemic equilibrium $E^*$, we first prove the uniform persistence of (4) when the threshold parameter $R_0 > 1$, by applying the acyclicity Theorem (see [18]).

**Definition 5** (see [19]). The system (4) is uniformly persistent, that is, there exists $c > 0$ (independent of initial conditions), such that $\liminf_{t \to \infty} s_i(t) \geq c$, $\liminf_{t \to \infty} v_i(t) \geq c$, $\liminf_{t \to \infty} l_i(t) \geq c$. Let $X$ be a locally compact metric space with metric $d$ and let $\Gamma$ be a closed nonempty subset of $X$ with boundary $\Gamma^*$ and interior $\Gamma^\circ$. Clearly, $\Gamma^\circ$ is a closed subset of $\Gamma$. Let $\Phi_0$ be a dynamical system defined on $\Gamma$. A set $B$ in $X$ is said to be invariant if $\Phi(B(t)) = B$. Define $M_0 := \{x \in \Gamma : \Phi(t, x) \in \Gamma, \text{for all } t \geq 0\}$. 


Lemma 6 (see [18]). Assume that

(a) $\Phi_t$ has a global attractor;
(b) there exists $M = \{M_1, \ldots, M_k\}$ of pair-wise disjoint, compact and isolated invariant set on $\partial \Gamma$ such that

1. $\bigcup_{x \in M_3} \omega(x) \subseteq \bigcup_{j=1}^{k} M_j$;
2. no subsets of $M$ form a cycle on $\partial \Gamma$;
3. each $M_j$ is also isolated in $\Gamma$;
4. $W^s(M_j) \cap \Gamma = \phi$ for each $1 \leq j \leq k$, where $W^s(M_j)$ is stable manifold of $M_j$. Then $\Phi_t$ is uniformly persistent with respect to $\Gamma$.

Proof: We have $\Gamma = \{(s_h, i_h, i_v) \in R^3, 0 \leq s_h + i_h < 1, 0 \leq i_v < 1\}$, $\Gamma^* = \{(s_h, i_h, i_v) \in R^3, s_h > 0\}$, $\partial \Gamma = \Gamma / \Gamma^*$. Obviously $M_3 = \partial \Gamma$. Since $\Gamma$ is bounded and positively invariant, so there exists a compact set $M$ in which all solutions of system (4) initiated in $\Gamma$ ultimately enter and remain forever. On $s_h$-axis we have $s''_h = b_1(1-s_h)$ which means $s_h \to 1$ as $t \to \infty$. Thus, $E_0$ is the only omega limit point on $\partial \Gamma$, that is, $\omega(x) = E_0$ for all $x \in M_3$. Furthermore, $M = E_0$ is a covering of $\Omega = \bigcup_{x \in M_3} \omega(x)$, because all solutions initiated on the $s_h$-axis converge to $E_0$. Also $E_0$ is isolated and acyclic. This verifies that hypothesis (1) and (2) hold. When $R_0 > 1$, the disease-“free” equilibrium (DFE) $E_0$ is unstable from Theorem 1 and also $W^s(M) = \partial \Gamma$. Hypothesis (3) and (4) hold. Therefore, there always exists a global attractor due to ultimate boundedness of solutions.

The boundedness of $\Gamma$ and the above lemma imply that (4) has a compact absorbing set $K \subset \Gamma$ [19]. Now we shall prove that the quantity $\alpha_2 < 0$. We choose a suitable vector norm $\| \|$ in $R^3$ and a $3 \times 3$ matrix-valued function

$$P(x) = \begin{pmatrix} 1 & 0 & 0 \\ 0 & \frac{i_h}{i_v} & 0 \\ 0 & 0 & \frac{i_h}{i_v} \end{pmatrix}.$$ (23)

Obviously, $P$ is $C^1$ and nonsingular in the interior of $\Omega$. Linearizing system (4) about an endemic equilibrium $E^*$ gives the following Jacobian matrix:

$$J(E^*)$$

$$= \begin{pmatrix} -\frac{b_1}{s_h} & 0 & -\beta s_h \\ 0 & -\beta s_h & 0 \\ \beta i_h + \beta s_v & \beta s_h - (\beta_i + \gamma_v + \delta_v) + 2\delta_i i_h & -\beta s_h \end{pmatrix}.$$ (24)
The second additive compound matrix of $J(E^*)$ is given by

$$J^{[2]} = \begin{pmatrix} M_{11} & \beta_2 s_h M_{22} & -\beta_2 s_h M_{33} \\ 0 & \beta_1 i_h + \beta_2 i_v & 0 \end{pmatrix},$$

where

$$M_{11} = -\frac{b_h}{s_h} + \frac{b_1 s_h}{s_h} - (b_1 + y_h + \delta_h) + 2\delta_h i_h,$$

$$M_{22} = -\frac{b_1}{s_h} - \beta_3 i_h - \mu_v,$$

$$M_{33} = \beta_1 s_h - (b_1 + y_h + \delta_h) + 2\delta_h i_h - \beta_3 i_h - \mu_v.$$

The matrix $B = P_T P^{-1} + P_T^{[2]} P^{-1}$ can be written in block form as

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}.$$

with

$$B_{11} = -\frac{b_h}{s_h} + \beta_1 s_h - (b_1 + y_h + \delta_h) + 2\delta_h i_h,$$

$$B_{12} = \begin{pmatrix} \beta_2 s_h i_h \beta_2 s_h i_v \\ \beta_2 s_h i_h \end{pmatrix},$$

$$B_{21} = \begin{pmatrix} \frac{i_h}{i_v} \beta_3 (1 - i_v) \\ 0 \end{pmatrix},$$

$$B_{22} = \begin{pmatrix} Q_{11} & Q_{12} \\ Q_{21} & Q_{22} \end{pmatrix},$$

where

$$Q_{11} = -\frac{b_h}{s_h} - \beta_3 i_h - \mu_v,$$

$$Q_{12} = - (\beta_1 - \delta_h) s_h,$$

$$Q_{21} = \beta_1 i_h + \beta_2 i_v,$$

$$Q_{22} = \beta_1 s_h - (b_1 + y_h + \delta_h) + 2\delta_h i_h - \beta_3 i_h - \mu_v.$$

Consider the norm in $R^3$ as $||u, v, w|| = \max(||u||, |v| + |w|)$ where $(u, v, w)$ denotes the vector in $R^3$. The Lozinskii measure with respect to this norm is defined as

$$\mu(B) = \sup(g_1, g_2),$$

where

$$g_1 = \mu_1 (B_{11}) + |B_{12}|, \quad g_2 = \mu_1 (B_{22}) + |B_{21}|.$$

From system (4), we can write

$$\frac{i_h'}{i_h} = \beta_1 s_h + \beta_3 s_h \frac{i_v}{i_h} - (b_1 + y_h + \delta_h) + \delta_h i_h,$$

$$\frac{i_v'}{i_v} = \beta_3 (1 - i_v) \frac{i_h}{i_v} - \mu_v.$$

Since $B_{11}$ is a scalar, its Lozinskii measure with respect to any vector norm in $R^3$ will be equal to $B_{11}$. Thus

$$|B_{11}| = -\frac{b_h}{s_h} + \beta_1 s_h - (b_1 + y_h + \delta_h) + 2\delta_h i_h,$$

and $g_1$ will become

$$g_1 = \frac{i_h'}{i_h} - \frac{b_1}{s_h} + \delta_h i_h,$$

and

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$$g_1 = \frac{i_h'}{i_h} - \frac{b_1}{s_h} + \delta_h i_h.$$
Also \(|B_{21}| = (i_h/i_v)\beta_3(1 - i_v), \quad |B_{12}| \) and \(|B_{21}| \) are the operator norms of \(B_{12}\) and \(B_{21}\) which are mapping from \(R^2\) to \(R\) and from \(R\) to \(R^2\) respectively, and \(R^2\) is endowed with the \(l_1\) norm. \(\mu_1(B_{22})\) is the Lozinski\' measure of \(2 \times 2\) matrix \(B_{22}\) with respect to \(l_1\) norm in \(R^2\);

\[
\mu(B_{22}) = \sup \left\{ \frac{i_v}{i_h} \left( \frac{i_h}{i_v} - \beta_3 i_h - \beta_1 i_h \right) + \beta_2 i_v \left( \frac{i_h}{i_v} \right) + (\beta_1 - \delta_h) s_h + \beta_1 s_h \right. \\
- (b_1 + \gamma_h + \delta_h) + 2\delta_h - \beta_3 i_h - \mu i_i
\]

if \(\beta_1 \leq \gamma_h/2\). Hence

\[
g_2 \leq \frac{i_v}{i_h} - \frac{i_v}{i_v} - b_1 + \delta_h i_h - \beta_3 i_h - \mu v + \left( \frac{i_h}{i_v} \right) \beta_3 (1 - i_v) \\
= \frac{i_v}{i_h} - b_1 + \delta_h i_h - \beta_3 i_h.
\]

Thus,

\[
\mu(B) = \sup \{g_1, g_2\} \\
\leq \frac{i_v}{i_h} - b_1 + \delta_h \\
\leq \frac{i_v}{i_h} - \beta_3,
\]

where \(\overline{\beta}_3 = \min(\gamma_h/2, b_1/2)\). Since (4) is uniformly persistent when \(R_0 > 1\), so for \(T > 0\) such that \(t > T\) implies \(i_h(t) \geq c, \quad i_v(t) \geq c\) and \((1/t) \log i_h(t) < (\overline{\beta}_3/2)\) for all \((s_h(0), i_h(0), i_v(0)) \in K\). Thus,

\[
\frac{1}{t} \int_0^t \mu(B) dt < \frac{\log i_h(t)}{t} < -\overline{\beta}_3 < -\beta_3/2.
\]

for all \((s_h(0), i_h(0), i_v(0)) \in K\), which further implies that \(\overline{\beta}_3 < 0\). Therefore, all the conditions of Theorem 4 are satisfied. Hence, unique endemic equilibrium \(E^*\) is globally stable in \(\Gamma\).

6. Discussions and Simulations

This paper deals with a vector-host disease model which allows a direct mode of transmission and varying human population. It concerns diseases with long duration and substantial mortality rate (e.g., malaria). Our main results are concerned with the global dynamics of transformed proportionate system. We have constructed Lyapunov function to show the global stability of disease-“free” equilibrium and the geometric approach is used to prove the global stability of “endemic” equilibrium. The epidemiological correlations between the two systems (normalized and unnormalized) have also been discussed. The dynamical behavior of the proportionate model is determined by the basic reproduction number of the disease. The model has a globally asymptotically stable disease-“free” equilibrium whenever \(R_0 \leq 1\) (Figures 3 and 4). When \(R_0 > 1\), the disease persists at an “endemic” level (Figures 5 and 6) if \(\beta_1 < \min(b_1/2, \gamma_h/2)\). Figures 7, 8, 9, and 10 describe numerically “endemic” level of infectious individuals and infectious vectors under the condition \(\beta_1 \leq \min(b_1/2, \gamma_h/2)\). We here question that what are the dynamics of the proportionate system (4) even if the condition \(\beta_1 < \min(b_1/2, \gamma_h/2)\) is not satisfied? We see numerically that if \(\delta_h, \gamma_h/2 < \beta_1 < b_1/2\) or \(\beta_1 < \delta_h < b_1/2\) then infectious individuals and infectious vectors will
the dimensionless form (3). If $\delta_h = 0$ and $b_1 = \mu_h$, then $N_h(t)' = 0$ and so $N_h(t)$ remains fixed at its initial value $N_{h0}$. In this case, the system (1) becomes the model with constant population whose dynamics are the same as the proportionate system (3). Hence, the solutions with initial conditions $S_{h0} + I_{h0} + R_{h0} = N_{h0}$ tend to $(N_{h0}, 0, 0)$ if $R_0 \leq 1$ and to $N_{h0}(s_0, i_0, r_0^*)$ if $R_0 > 1$. In the rest of this section, we suppose that $\delta_h > 0$. From system (1) and (2), the trivial equilibrium $E = (0, 0, 0, 0)$ can be easily obtained. Assume that $E_* = (N_*^h, S_*^h, I_*^h, R_*^h, I_*^s)$ is the endemic equilibrium of system (1) and (2), where $N_*^h = S_*^h + I_*^h + R_*^s$. This equilibrium exists if and only if the following equations are satisfied
\[
\frac{S_*^h}{N_*^h} = \frac{Q(\beta_3 \alpha_h + \mu \delta_h)}{\beta_1 (\beta_2 \alpha_h + \mu \delta_h) + \beta_3 \beta_3 \delta_h},
\]
\[
\frac{I_*^s}{N_*^h} = \frac{\alpha_h}{\delta_h},
\]
\[
\frac{R_*^s}{N_*^h} = \frac{\gamma_h \alpha_h}{\mu \delta_h},
\]
\[
\frac{I_*^s}{N_*^h} = \frac{\beta_3 \alpha_h N_v}{(\beta_3 \alpha_h + \mu_s) N_*^h},
\]
where $\alpha_h = b_1 - \mu_h$ and $Q = \mu_h + \gamma_h + \delta_h$. We introduce the parameters
\[
R_1 = \begin{cases} \frac{b_1}{\mu_h}, & \text{if } R_0 \leq 1, \\ \frac{b_1}{\mu_h + \delta_h i_*^h}, & \text{if } R_0 > 1, \end{cases}
\]
\[
R_2 = \begin{cases} \frac{\beta_1}{\mu_h + \gamma_h + \delta_h}, & \text{if } R_0 \leq 1, \\ \frac{\mu_h + \gamma_h + \delta_h}{\mu_r (\mu_h + \gamma_h + \delta_h)} + \frac{\beta_2 \beta_3 \alpha_h (1 - i_*^s)}{\mu_r (\mu_h + \gamma_h + \delta_h)} & \text{if } R_0 > 1. \end{cases}
\]
From (2) we have for $t \to \infty$
\[
\frac{dN_h}{dt} = N_h (b_1 - \mu_h - \delta_h i_*^h)
\]
\[
\to \begin{cases} N_h (b_1 - \mu_h), & \text{if } R_0 \leq 1, \\ N_h (b_1 - \mu_h - \delta_h i_*^h), & \text{if } R_0 > 1. \end{cases}
\]
By the definition of $R_1$, we have following threshold result.

**Theorem A.1.** The total population $N_h(t)$ for the system (1) decreases to zero if $R_1 < 1$ and increases to $\infty$ if $R_1 > 1$ as $t \to \infty$. The asymptotic rate of decrease is $\mu_h (R_1 - 1)$ if $R_0 \leq 1$, and the asymptotic rate of increase is $(\mu_h + \delta_h i_*^h)(R_1 - 1)$ when $R_0 > 1$.

**Theorem A.2.** Suppose $R_1 > 1$, for $t \to \infty$, $(S_h(t), I_h(t), R_h(t))$ tend to $(\infty, 0, 0)$ if $R_2 < 1$ and tend to $(\infty, \infty, \infty)$ if $R_2 > 1$. 
Proof: Since \( i'_v \to 0 \) as \( t \to \infty \), so in the limiting case the proportion of infectious mosquitoes is related to the proportion of infectious humans as

\[
i_v = \frac{\beta_v (1 - i_v) i_h}{\mu_v}, \tag{A.4}\n\]

thus, the equation for \( I_v(t) \) has limiting form

\[
\frac{dI_v(t)}{dt} = (\mu_h + \gamma_h + \delta_h) (R_2 - 1) I_v, \tag{A.5}\n\]

which shows that \( I_v(t) \) decreases exponentially if \( R_2 < 1 \) and increases exponentially if \( R_2 > 1 \).

The solution \( R_h(t) \) is given by

\[
R_h(t) = R_{h0} e^{\mu_d t} + \gamma_h e^{-\mu_d t} \int_0^t I_s(s) e^{\mu_d s} ds. \tag{A.6}\n\]

From the exponential nature of \( I_h(t) \), it follows that \( I_h(t) \) declines exponentially if \( R_2 < 1 \) and grows exponentially if \( R_2 > 1 \).

Suppose \( R_1 = 1 \), then \( b_1 = \mu_h \) corresponding to \( R_1 < 1 \) and the differential equation for \( N_h(t) \) will have the form

\[
\frac{dN_h(t)}{dt} = -\delta_h h, \tag{A.7}\n\]

which means that \( N_h(t) \) is bounded for all \( t > 0 \), the equilibria \((N_h^*, 0, 0, 0)\) have one eigenvalue zero, and the other eigenvalues have negative real parts. Therefore, each orbit approaches an equilibrium point.

If \( R_0 > 1 \), the disease becomes “endemic.” From the global stability of \( E^* \) and the equation

\[
\frac{dN_h(t)}{dt} = \delta_h \left[ \left( \frac{b_1 - \mu_h}{\delta_h} - i_h \right) (i_h - i_h^*) \right] N_h, \tag{A.8}\n\]

we observe that \((N_h, S_h, I_h, R_h) \) approaches to \((0, 0, 0, 0, 0)\) or \((\infty, \infty, \infty, \infty, \infty)\) if \( R_1 < 1 \) or \( R_1 > 1 \). From the global stability of \( i_h^* \), we have \( N_h(t) \) converges to some \( N_h^* \) as \( t \) approaches to \( \infty \). Since \( S_h = S_h/N_h, \ i_h = I_h/N_h, \ r_h = R_h/N_h, \) so we have \( S_h^* = s_h^* N_h^*, \ i_h^* = i_h^* N_h^*, \ r_h^* = r_h^* N_h^* \). All the above discussion is summarized in Table 1.

### Acknowledgment

This work was supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education, Science and Technology (MEST) (2012-000599).

### References


