

# Global dynamics of vector-borne diseases with horizontal transmission in host population

Abid Ali Lashari<sup>a,\*</sup>, Gul Zaman<sup>b,1</sup>

<sup>a</sup> Center for Advanced Mathematics and Physics, National University of Sciences and Technology, H-12, Islamabad, Pakistan

<sup>b</sup> Department of Mathematics, University of Malakand, Chakdara Dir (Lower), Khyber Puhtoonkhwa, Pakistan

## ARTICLE INFO

### Article history:

Received 31 August 2010

Received in revised form 9 December 2010

Accepted 9 December 2010

### Keywords:

Epidemic model

Backward bifurcation

Global stability

Numerical simulation

## ABSTRACT

The paper presents the dynamical features of a vector–host epidemic model with direct transmission. First, we extended the model by taking into account the exposed individuals in both human and vector population with the impact of disease related deaths and total time dependent population size. Using Lyapunov function theory some sufficient conditions for global stability of both the disease-free equilibrium and the endemic equilibrium are obtained. For the basic reproductive number  $R_0 > 1$ , a unique endemic equilibrium exists and is globally asymptotically stable. Furthermore, it is found that the model exhibits the phenomenon of backward bifurcation, where the stable disease-free equilibria coexists with a stable endemic equilibrium. Finally, numerical simulations are carried out to investigate the influence of the key parameters on the spread of the vector-borne disease, to support the analytical conclusion and illustrate possible behavioral scenarios of the model.

© 2010 Elsevier Ltd. All rights reserved.

## 1. Introduction

Vector-borne diseases are infectious diseases caused by pathogens transmitted by insects, ticks, bacteria and protozoa which are primarily transmitted by disease transmitting biological agents (arthropods) called vectors, who carry the disease without getting it themselves. Vector-borne infections are major killers, particularly of children in developing countries. Over the past decade, more comprehensive and transparent methods of measuring health have improved understanding of the importance of these diseases. The World Health Organization reports annually on the numbers of deaths and DALYs (disability adjusted life years, a composite measure of health status combining premature death and sickness during life), by disease category in different regions of the world [1]. Despite technological advances and increasing affluence in many regions, vector-borne infectious diseases remain amongst the most important causes of global health illness. Malaria is one of the most prevalent vector-borne diseases whose vectors are the mosquitoes. The mosquitoes are vectors of a number of infectious diseases most prominent among which are dengue, yellow fever, St Louis Encephalitis, Japanese Encephalitic, and West Nile Fever, caused by the West Nile Virus. There are also some other vectors like the assassin bugs, causing the Chagas disease, fleas transmitting the plague from its normal host to humans, or from human to human, and ticks which transmit the most prevalent vector-borne diseases in North America.

Both deterministic and stochastic population models are important in characterization for understanding the relationship of vector-borne diseases with ecological communities. The model first proposed by Ross [2] and subsequently modified

\* Corresponding author.

E-mail addresses: [abidlshr@yahoo.com](mailto:abidlshr@yahoo.com) (A.A. Lashari), [gzaman@uom.edu.pk](mailto:gzaman@uom.edu.pk) (G. Zaman).

<sup>1</sup> Tel.: +92 051 9085 5551; fax: +92 051 9085 5552.

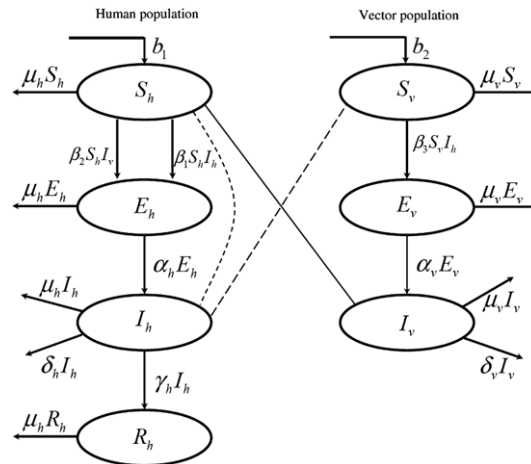


Fig. 1. The flow chart represents the interaction and transfer diagram of both human and vector.

by Macdonald [3] has influenced both the modeling and the application of control strategies to vector-borne disease. Models of malaria that investigate complications arising from host superinfection, immunity, and other factors are based on this fundamental model [4–7]. The model has also influenced the mathematical analysis of many other vector-borne diseases [8,9]. Infectious disease such as malaria, dengue fever, West Nile virus, and so forth, are transmitted to human population by vectors. However direct transmission (transfusion related transmission, transplantation related transmission, and needle–stick-related transmission) is also possible [10].

In recent years, the phenomenon of the backward bifurcations has arisen the interests in disease control (see [11–13]). In this case, the basic reproduction number cannot describe the necessary disease elimination effort any more. In a backward bifurcation, disease control is only feasible if basic reproductive number  $R_0$  is reduced further to values below another sub-threshold less than unity. Clearly, this phenomenon has important public health implication, since it renders the classical requirement of reproduction number being less than unity to be insufficient (in general) for disease elimination. Thus, it is important to identify backward bifurcations and establish thresholds for the control of diseases. The purpose of this paper is to study the backward bifurcation and global dynamics of a vector–host epidemic model with direct transmission.

In this work we extend the model of Cai and Li [14] to include exposed individuals, disease induced death rate and time dependent total population size in both host and vector population. We first establish stability results for the proposed model. Analysis of this model reveals that there are two equilibria which are the disease-free equilibria and the endemic equilibria. Further, it is shown that the model exhibits the phenomenon of backward bifurcation where the locally asymptotically stable disease-free equilibrium co-exists with a locally asymptotically stable endemic equilibrium when  $R_0 < 1$ . Then, we use Lyapunov function theory to present global asymptotical stability. It is proved that the global dynamics are completely determined by the basic reproduction number  $R_0$ . If  $R_0 \leq 1$ , the disease-free equilibrium is globally stable. If  $R_0 > 1$ , a unique endemic equilibrium exists and is globally asymptotically stable. Finally, numerical simulations are carried out to investigate the influence of the key parameters on the spread of the vector-borne disease, to support the analytical conclusion and illustrate possible behavioral scenarios of the model. This new assumption is biologically much more plausible than the previous assumption without the exposed class and disease related death rate in both host and vector population.

The paper is organized as follows. In Section 2, we present a formulation of the extended mathematical model. We show mathematical analysis to establish stability results for the proposed model in Section 3. We present detail analysis of the endemic equilibria and the existence of a backward bifurcation in Section 4. In Section 5, we use Lyapunov function theory to show global stability of both disease-free and endemic equilibrium, respectively. Parameters estimation and numerical results are discussed in Section 6. Finally, we give conclusion.

## 2. Model frame work

The model presented in [14] studied the analysis of a simple vector–host epidemic model with direct transmission. In their work, the host population was divided in three subclasses susceptible host  $S_h$ , infectious host  $I_h$  and recovered host  $R_h$  with total constant population and vector population into susceptible vectors  $S_v$  and infectious vectors  $I_v$  without disease related death rate. In this section, we extend their model by including the following features:

1. Exposed class in both host and vector population denoted by  $E_h$  and  $E_v$ , respectively.
2. Disease induced death rates in both host and vector population denoted by  $\delta_h$  and  $\delta_v$ , respectively.
3. Total time dependent population of both host and vectors such that  $N_h = S_h + E_h + I_h + R_h$  and  $N_v = S_v + E_v + I_v$ .

We consider a compartmental model that divide the host and vector populations into different classes. In our model the total host population at time  $(t)$ , denoted by  $N_h(t)$ , is split into four distinct epidemiological subclasses which are susceptible ( $S_h(t)$ ), exposed ( $E_h(t)$ ), infectious ( $I_h(t)$ ) and recovered ( $R_h(t)$ ) subclasses of host, so that  $N_h = S_h + E_h + I_h + R_h$  while, the total vector population at time  $(t)$ , denoted by  $N_v(t)$  is split into susceptible ( $S_v(t)$ ), exposed ( $E_v(t)$ ) and infectious ( $I_v(t)$ ), subclasses of vectors, so that  $N_v = S_v + E_v + I_v$ . The immune class in the vector population does not exist, since the mosquitoes once infected never recover from infection, that is, their infection period ends with their death. Complete interaction and transfer diagram of both host (human) and vector population is depicted in Fig. 1. The compartmental deterministic mathematical model can be represented analytically by the following nonlinear system of seven ordinary differential equations:

$$\begin{aligned}
 \frac{dS_h}{dt} &= b_1 - \beta_1 S_h I_h - \beta_2 S_h I_v - \mu_h S_h, \\
 \frac{dE_h}{dt} &= \beta_1 S_h I_h + \beta_2 S_h I_v - \alpha_h E_h - \mu_h E_h, \\
 \frac{dI_h}{dt} &= \alpha_h E_h - \gamma_h I_h - \mu_h I_h - \delta_h I_h, \\
 \frac{dR_h}{dt} &= \gamma_h I_h - \mu_h R_h, \\
 \frac{dS_v}{dt} &= b_2 - \beta_3 S_v I_h - \mu_v S_v, \\
 \frac{dE_v}{dt} &= \beta_3 S_v I_h - \alpha_v E_v - \mu_v E_v, \\
 \frac{dI_v}{dt} &= \alpha_v E_v - \mu_v I_v - \delta_v I_v,
 \end{aligned} \tag{1}$$

with initial conditions

$$S_h(0) \geq 0, \quad E_h(0) \geq 0, \quad I_h(0) \geq 0, \quad R_h(0) \geq 0, \quad S_v(0) \geq 0, \quad E_v(0) \geq 0, \quad I_v(0) \geq 0. \tag{2}$$

Here  $b_1$  is the recruitment rate of human (assumed susceptible). Susceptible human can be infected via two routes of transmission, that is, directly, through a contact with an infected individual (possibly as a result of transfusion related transmission, transplantation related transmission, and needle–stick-related transmission) and through being bitten by an infectious vector. Thus, we denote the rate of direct transmission of the disease by  $\beta_1$ ,  $\beta_2$  is the biting rate that a pathogen-carrier mosquito has of susceptible human,  $\mu_h$  is the natural mortality rate of human, exposed humans develop clinical symptoms of the disease and move to the infectious class at rate  $\alpha_h$ ,  $\gamma_h$  is the recovery rate of human. We assume that a disease may be fatal to some infectious host, so deaths due to disease can be included in the model using the disease related death rate from infectious class,  $\delta_h$ . It is assumed that recovered individuals acquire lifelong immunity against re-infection. Similarly  $b_2$  is the constant recruitment rate of susceptible vectors population by birth and  $\beta_3$  represents biting rate of per susceptible vector per host per unit time,  $\mu_v$  is the natural mortality rate of vectors population. Exposed vectors develop symptoms of disease and move to the infectious class at rate  $\alpha_v$  and infectious vectors die due to disease at a rate  $\delta_v$ . The total human population dynamics is given by

$$\frac{dN_h}{dt} = b_1 - \mu_h N_h - \delta_h I_h. \tag{3}$$

The given initial conditions (2) make sure that  $N_h(0) \geq 0$ . Thus the total population  $N_h(t)$  remains positive and bounded for all finite time  $t > 0$ . The total dynamics of vector population is

$$\frac{dN_v}{dt} = b_2 - \mu_v N_v - \delta_v I_v. \tag{4}$$

It follows from (3) and (4) that

$$\frac{dN_h}{dt} \leq b_1 - \mu_h N_h, \quad \frac{dN_v}{dt} \leq b_2 - \mu_v N_v. \tag{5}$$

Then

$$\lim_{t \rightarrow \infty} \text{Sup} N_h \leq \frac{b_1}{\mu_h} \quad \text{and} \quad \lim_{t \rightarrow \infty} \text{Sup} N_v \leq \frac{b_2}{\mu_v}.$$

Thus the feasible region for the system (1) is  $\Omega = \{(S_h, E_h, I_h, R_h, S_v, E_v, I_v) \in R^7_+, V_1 \leq \frac{b_1}{\mu_h}, V_2 \leq \frac{b_2}{\mu_v}\}$ .

**Proposition 1.** Let  $(S_h, E_h, I_h, R_h, S_v, E_v, I_v)$  be the solution of the system (1) with initial conditions (2) and the closed set  $\Omega = \{(S_h, E_h, I_h, R_h, S_v, E_v, I_v) \in R^7_+, V_1 \leq \frac{b_1}{\mu_h}, V_2 \leq \frac{b_2}{\mu_v}\}$ . Then  $\Omega$  is positively invariant and attracting under the flow described by (1).

**Proof.** Consider the following Lyapunov function

$$V(t) = (V_1(t), V_2(t)) = (S_h + E_h + I_h + R_h, S_v + E_v + I_v). \quad (6)$$

Its time derivative is

$$\frac{dV}{dt} = (b_1 - \mu_h V_1 - \delta_h I_h, b_2 - \mu_v V_2 - \delta_v I_v). \quad (7)$$

It is easy to prove that

$$\begin{cases} \frac{dV_1}{dt} \leq b_1 - \mu_h V_1 \leq 0, & \text{for } V_1 \geq \frac{b_1}{\mu_h}, \\ \frac{dV_2}{dt} \leq b_2 - \mu_v V_2 \leq 0, & \text{for } V_2 \geq \frac{b_2}{\mu_v}. \end{cases} \quad (8)$$

It follows from (8) that  $\frac{dV}{dt} \leq 0$  which implies that  $\Omega$  is positively invariant set. On the other hand, a standard comparison theorem [15] can be used to show that  $0 \leq (V_1, V_2) \leq (V_1(0)e^{-\mu_h t} + \frac{b_1}{\mu_h}(1 - e^{-\mu_h t}), V_2(0)e^{-\mu_v t} + \frac{b_2}{\mu_v}(1 - e^{-\mu_v t}))$ . Thus as  $t \rightarrow \infty$ ,  $0 \leq (V_1, V_2) \leq (\frac{b_1}{\mu_h}, \frac{b_2}{\mu_v})$  and we can conclude that  $\Omega$  is an attracting set.  $\square$

Furthermore, the model (1) is well-posed epidemiologically and mathematically. Hence, it is sufficient to study the dynamics of this basic model in  $\Omega$ .

### 3. Disease-free equilibrium

In order to understand dynamical behavior of the system (1), we set right hand side of all equations in the system (1) equal to zero. Direct calculations shows that the system (1) has a disease-free equilibrium point given by

$$E_1 = (S_h^0, 0, 0, 0, S_v^0, 0, 0),$$

where  $S_h^0 = \frac{b_1}{\mu_h}$  and  $S_v^0 = \frac{b_2}{\mu_v}$ . The dynamics of the disease is described by the quantity  $R_0$  as follows:

$$R_0 = \frac{b_1}{\mu_h} \left( \frac{\alpha_h \alpha_v b_2 \beta_2 \beta_3}{\mu_v Q_1 Q_2 Q_3 Q_4} + \frac{\alpha_h \beta_1}{Q_1 Q_2} \right), \quad (9)$$

with  $Q_1 = \alpha_h + \mu_h$ ,  $Q_2 = \gamma_h + \mu_h + \delta_h$ ,  $Q_3 = \alpha_v + \mu_v$  and  $Q_4 = \mu_v + \delta_v$ . The threshold quantity  $R_0$ , is called the basic reproduction number of the disease [16,17]. It represents the expected average number of new infections produced directly and indirectly by a single infective when introduced into a completely susceptible population. For classical epidemic models, it is common that the basic reproduction number is threshold in a sense that, when the basic reproduction number  $R_0 < 1$ , on average each infected individual infects fewer than one individual, and the disease dies out. If  $R_0 > 1$ , on average each infected individual, infects more than one other individual, so we would expect the disease to spread.

**Theorem 3.1.** *If  $R_0 < 1$ , then the disease-free equilibrium point  $E_1$  of the model (1) is locally asymptotically stable, otherwise unstable.*

**Proof.** The local stability of the disease-free equilibrium solution can be examined by linearizing the system (1) around  $E_1$ . This gives the Jacobian matrix  $J_1$  as follows

$$J_1 = \begin{bmatrix} -\mu_h & 0 & -\beta_1 \frac{b_1}{\mu_h} & 0 & 0 & 0 & -\beta_2 \frac{b_1}{\mu_h} \\ 0 & -Q_1 & \beta_1 \frac{b_1}{\mu_1} & 0 & 0 & 0 & \beta_2 \frac{b_1}{\mu_h} \\ 0 & \alpha_h & -Q_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & -\beta_3 \frac{b_2}{\mu_v} & 0 & -\mu_v & 0 & 0 \\ 0 & 0 & \beta_3 \frac{b_2}{\mu_v} & 0 & 0 & -Q_3 & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha_v & -Q_4 \end{bmatrix}. \quad (10)$$

The characteristic equation of the above matrix is

$$(\lambda + \mu_h)(\lambda + \mu_h)(\lambda + \mu_v)(\lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4) = 0, \quad (11)$$

where

$$\begin{aligned} a_1 &= Q_1 + Q_2 + Q_3 + Q_4, \\ a_2 &= Q_1Q_2 + Q_1Q_3 + Q_1Q_4 + Q_2Q_3 + Q_2Q_4 + Q_3Q_4 - \frac{b_1\alpha_h\beta_1}{\mu_h}, \\ a_3 &= Q_1Q_3Q_4 + Q_2Q_3Q_4 + (Q_3 + Q_4) \left( Q_1Q_2 - \frac{b_1\alpha_h\beta_1}{\mu_h} \right), \\ a_4 &= Q_1Q_2Q_3Q_4(1 - R_0). \end{aligned}$$

There are seven eigenvalues corresponding to Eq. (11). Three of the eigenvalues,  $-\mu_h$  with multiplicity two and  $-\mu_v$  have negative real part. The other four eigenvalues can be obtained by solving

$$\delta(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4. \tag{12}$$

These four eigenvalues have negative real part if they satisfy the Routh–Hurwitz Criteria [18], such that  $a_i > 0$  for  $i = 1, 2, 3, 4$ , with  $a_1a_2a_3 > a_2^2 + a_1^2a_4$ . For  $R_0 < 1$ ,  $Q_1Q_2 > b_1\alpha_h\beta_1/\mu_h$ , we obtain  $a_i > 0$  for  $i = 1, 2, 3, 4$ . Thus all the eigenvalues of the characteristic equation (11) have negative real parts if and only if  $R_0 < 1$ , which shows that the disease-free equilibrium  $E_1$  is locally asymptotically stable. □

**Remark.** For  $R_0 \geq 1$  or equivalently  $a_4 < 0$ , we have  $\delta(0) < 0$  and  $\lim \delta(\lambda) \rightarrow +\infty$  when  $\lambda \in \mathbb{R}$  and  $\lambda \rightarrow +\infty$ . Then, there exists  $\lambda^* > 0$  such that  $\delta(\lambda^*) = 0$ , which proves the instability of the disease-free equilibrium.

#### 4. Endemic equilibria and backward bifurcation

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

Let  $E_2 = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*)$  represents any arbitrary endemic equilibrium of the model (1). Solving the equations of the system (1) at steady state gives,

$$\begin{aligned} S_h^* &= \frac{Q_1Q_2Q_3Q_4(\mu_v + \beta_3I_h^*)}{\alpha_h\beta_1Q_3Q_4(\mu_v + \beta_3I_h^*) + \alpha_h\alpha_v b_2\beta_2\beta_3}, & E_h^* &= \frac{Q_2}{\alpha_h} I_h^*, & R_h^* &= \frac{\gamma_h}{\mu_h} I_h^*, & S_v^* &= \frac{b_2}{\mu_v + \beta_3I_h^*}, \\ E_v^* &= \frac{\beta_3 b_2 I_h^*}{Q_3(\mu_v + \beta_3 I_h^*)}, & I_v^* &= \frac{\beta_3 \alpha_v b_2 I_h^*}{Q_3 Q_4 (\mu_v + \beta_3 I_h^*)}. \end{aligned}$$

If  $I_h^* \neq 0$ , then substituting  $S_h^*, I_h^*$  in the first equation of the system (1) at steady state, we obtain after some calculations the following quadratic equation:

$$f(I_h) = aI_h^2 + bI_h + c = 0, \tag{13}$$

where

$$\begin{aligned} a &= \beta_1\beta_3Q_1Q_2Q_3Q_4, \\ b &= (\beta_1\mu_v + \beta_3\mu_h)Q_1Q_2Q_3Q_4 + b_2\alpha_v\beta_2\beta_3Q_1Q_2 - b_1\alpha_h\beta_1\beta_3Q_3Q_4, \\ c &= \mu_h\mu_vQ_1Q_2Q_3Q_4(1 - R_0). \end{aligned} \tag{14}$$

Clearly the coefficient  $a$  is always positive, and  $c$  is positive (negative) if  $R_0$  is less than (greater than) unity, respectively. Since  $a > 0$ , the existence of the positive solutions of Eq. (13) will depend on the signs of  $b$  and  $c$ . If  $R_0 > 1$ , then there are two roots of Eq. (13) of which one root is positive and thus there is a unique endemic equilibrium. If  $R_0 = 1$ , then  $c = 0$  and there is a unique nonzero solution of (13),  $I = -b/a$ , which is positive if and only if  $b < 0$ . If  $b < 0$  there is a positive endemic equilibrium for  $R_0 = 1$ . Since equilibria depend continuously on  $R_0$  which shows that there exists an interval to the left of  $R_0$  on which there are two positive equilibria

$$I_1 = \frac{-b - \sqrt{b^2 - 4ac}}{2a}, \quad I_2 = \frac{-b + \sqrt{b^2 - 4ac}}{2a}.$$

If  $c > 0$  and either  $b \geq 0$  or  $b^2 < 4ac$ , there are no positive solutions of (13) and thus there are no endemic equilibria. For different range of these parameters the following results are established.

**Theorem 4.1.** *The model (1) has:*

- (i) a unique endemic equilibrium in  $\Omega$  if  $c < 0 \Leftrightarrow R_0 > 1$ ;
- (ii) a unique endemic equilibrium in  $\Omega$  if  $b < 0$ , and  $c = 0$  or  $b^2 - 4ac = 0$ ;
- (iii) two endemic equilibria in  $\Omega$  if  $c > 0$ ,  $b < 0$  and  $b^2 - 4ac > 0$ ;
- (iv) no endemic equilibria otherwise.

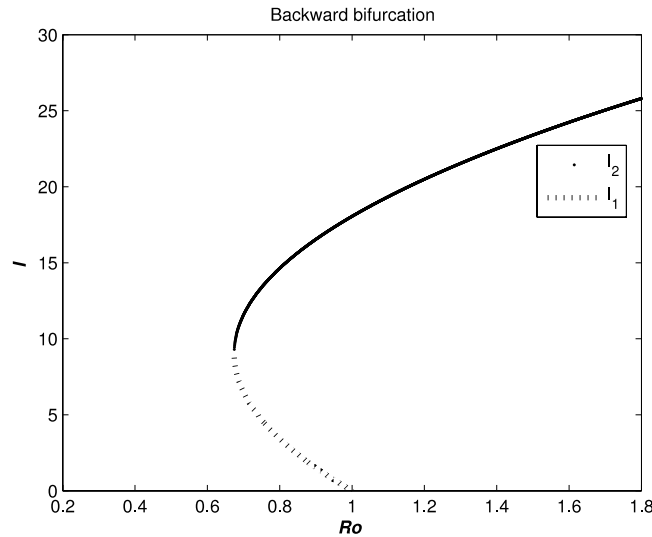


Fig. 2. The figure of  $I_h^*$ ,  $I_1$  and  $I_2$  versus  $R_0$  that shows a backward bifurcation with endemic equilibria when  $R_0 < 1$ .

Case (iii) of Theorem 4.1 indicates the possibility of a backward bifurcation (where the locally-asymptotically stable disease-free equilibrium co-exists with a locally-asymptotically stable endemic equilibrium when  $R_0 < 1$ , see, for instance, [19,20]) in the model (1) when  $R_0 < 1$ . To find backward bifurcation, we set the discriminant  $b^2 - 4ac$  to be zero and solved for the critical value of  $R_0$ , denoted by  $R_c$  is given by

$$R_c = 1 - \frac{b^2}{4a\mu_h\mu_vQ_1Q_2Q_3Q_4}.$$

Thus,  $R_c < R_0$  is equivalent to  $b^2 - 4ac > 0$  and therefore, backward bifurcation would occur for values of  $R_0$  such that  $R_c < R_0 < 1$ . This is illustrated by simulating the model with the following set of parameter values:  $b_1 = 36$ ,  $\beta_1 = 0.01$ ,  $\mu_h = 0.1$ ,  $\alpha_h = 0.01$ ,  $\gamma_h = 0.001$ ,  $\delta_h = 0.001$ ,  $b_2 = 100$ ,  $\beta_3 = 0.0072$ ,  $\mu_v = 0.09$ ,  $\alpha_v = 0.001$ , and  $\delta_v = 0.99$ . The associated bifurcation diagram is depicted in Fig. 2. Thus, Fig. 2 clearly shows the co-existence of two locally-asymptotically stable equilibria when  $R_0 < 1$ , confirming that the model (1) undergoes the phenomenon of backward bifurcation.

**Lemma 4.1.** The basic model (1) undergoes backward bifurcation when case (iii) of Theorem 4.1 holds with  $R_c < R_0 < 1$ .

**Corollary 4.2.** System (1) has a backward bifurcation at  $R_0 = 1$  if and only if  $b < 0$ .

**Proof.** For sufficiency, let us consider the graph of  $y = g(I) = aI^2 + bI + c$ . Since  $R_0 = 1$  implies  $c = 0$  thus  $g(0) = 0$ , hence the graph passes through the origin. Further, if  $b < 0$ , we have  $g(I) = 0$ , has a positive root  $I = -b/a$ . If we increase  $c$  from  $c = 0$  to some positive value  $c > 0$ , since  $g(I)$  is continuous function of  $c$  guarantees that there will be some open interval  $(0, \epsilon)$  containing  $c$ , on which  $g(I) = 0$  has two positive real roots. In other words, we have shown that it is possible that there exist two endemic equilibria when  $R_0 < 1$ . The necessity is obvious, since if  $b \geq 0$ , (13) has no positive real roots when  $R_0 < 1$ . □

The epidemiological significance of the phenomenon of backward bifurcation is that the classical requirement of  $R_0 < 1$  is, although necessary, no longer sufficient for disease elimination. In such a scenario, disease elimination would depend on the initial sizes of the sub-populations (state variables) of the model. That is, the presence of backward bifurcation in the model (1) suggests that the feasibility of controlling disease when  $R_0 < 1$  could be dependent on the initial sizes of the sub-population of the model.

Now we analyze the local stability of the endemic equilibrium when  $R_0 > 1$ .

**Theorem 4.2.** When  $R_0 > 1$ , the unique endemic equilibrium state  $E_2$  is locally asymptotically stable if  $\beta_1 \geq \frac{Q_1Q_2}{\alpha_h b_1}$ .

**Proof.** Linearization of the system (1) about an endemic equilibrium  $E_2$  gives the Jacobian matrix:

$$J_2 = \begin{bmatrix} -\beta_1 I_h^* - \beta_2 I_v^* - \mu_h & 0 & -\beta_1 S_h^* & 0 & 0 & -\beta_2 S_h^* \\ \beta_1 I_h^* + \beta_2 I_v^* & -Q_1 & \beta_1 S_h^* & 0 & 0 & \beta_2 S_h^* \\ 0 & \alpha_h & -Q_2 & 0 & 0 & 0 \\ 0 & 0 & -\beta_3 S_v^* & -\beta_3 I_h^* - \mu_v & 0 & 0 \\ 0 & 0 & \beta_3 S_v^* & \beta_3 I_h^* & -Q_3 & 0 \\ 0 & 0 & 0 & 0 & \alpha_v & -Q_4 \end{bmatrix}. \tag{15}$$

To discuss the properties of the endemic equilibrium we make an elementary row-transformation for the Jacobian matrix  $J_2$  to obtain the following matrix:

$$J^* = \begin{bmatrix} -M_1 & 0 & -\beta_1 S_h^* & 0 & 0 & -\beta_2 S_h^* \\ 0 & -Q_1 & \frac{\mu_h \beta_1 S_h^*}{M_1} & 0 & 0 & \frac{\mu_h \beta_2 S_h^*}{M_1} \\ 0 & 0 & -Q_2 + \frac{\alpha_h \mu_h \beta_1 S_h^*}{Q_1 M_1} & 0 & 0 & \frac{\alpha_h \mu_h \beta_2 S_h^*}{Q_1 M_1} \\ 0 & 0 & 0 & -\beta_3 I_h^* - \mu_v & 0 & \frac{M_2}{l} \\ 0 & 0 & 0 & 0 & -Q_3 & \frac{l}{\alpha_v \mu_v M_2} \\ 0 & 0 & 0 & 0 & 0 & -Q_4 - \frac{\alpha_v \mu_v M_2}{Q_3(\beta_3 I_h^* + \mu_v)} \end{bmatrix} \tag{16}$$

where

$$M_1 = \beta_1 I_h^* + \beta_2 I_v^* + \mu_h, \quad M_2 = \frac{\alpha_h \mu_h \beta_2 \beta_3 S_h^* S_v^*}{-Q_1 Q_2 M_1 + \alpha_h \mu_h \beta_1 S_h^*}.$$

The eigenvalues are

$$\begin{aligned} \lambda_1 &= -M_1 < 0, & \lambda_2 &= -Q_1 < 0, & \lambda_3 &= -Q_2 + \frac{\alpha_h \mu_h \beta_1 S_h^*}{Q_1 M_1}, & \lambda_4 &= -\beta_3 I_h^* - \mu_v < 0, \\ \lambda_5 &= -Q_3 < 0, & \lambda_6 &= -Q_4 - \frac{\alpha_v \mu_v M_2}{Q_3(\beta_3 I_h^* + \mu_v)}, \end{aligned}$$

$\lambda_3 < 0$ , if and only if

$$Q_1 Q_2 M_1 - \alpha_h \mu_h \beta_1 S_h^* > 0. \tag{17}$$

By using the value of  $M_1$  with some little rearrangement, we can rewrite Eq. (17)

$$\begin{aligned} &\beta_1 \beta_3^2 Q_3 Q_4 (b_1 \alpha_h \beta_1 - Q_1 Q_2 \mu_h) I_h^{*2} + 2 Q_1 Q_2 Q_3 Q_4 \mu_h \mu_v \beta_1 \beta_2 (R_0 - 1) I_h^* \\ &+ Q_1 Q_2 Q_3 Q_4 \mu_h \mu_v^2 \beta_1 (R_0 - 1) + Q_1 Q_2 \mu_h \mu_v \alpha_v b_2 \beta_2 \beta_3 R_0 > 0. \end{aligned} \tag{18}$$

Thus all the coefficients of Eq. (18) are positive if  $R_0 > 1$  and  $b_1 \geq \frac{Q_1 Q_2}{\alpha_h \beta_1}$ . Also the condition  $Q_1 Q_2 M_1 > \alpha_h \mu_h \beta_1 S_h^*$  shows that  $M_2 > 0$ . Hence  $\lambda_6 < 0$ . Thus all the eigenvalues have negative real parts, which shows that the  $E_2$  is locally asymptotically stable.  $\square$

### 5. Global stability analysis

The following theorem provides the global property of the disease-free equilibrium  $V_1$  of the system (1).

**Theorem 5.1.** *If  $R_0 \leq 1$ , then the disease-free equilibrium of the system (1) is globally asymptotically stable on  $\Omega$ .*

**Proof.** To establish the global stability of the disease-free equilibrium  $E_1$ , we construct the following Lyapunov function:

$$V(t) = W_1 \left( S_h - S_h^0 - S_h^0 \log \frac{S_h}{S_h^0} \right) + W_2 E_h + W_3 I_h + W_4 \left( S_v - S_v^0 - S_v^0 \log \frac{S_v}{S_v^0} \right) + W_5 E_v + W_6 I_v, \tag{19}$$

where  $W_i$ , for  $i = 1, 2, \dots, 6$  are some positive constants to be chosen later. Calculating the time derivative of  $V$  along the solutions of system (1), we obtain

$$\begin{aligned} V'(t) &= W_1 \left( \frac{S_h - S_h^0}{S_h} \right) [b_1 - \beta_1 S_h I_h - \beta_2 S_h I_v - \mu_h S_h] + W_2 [\beta_1 S_h I_h + \beta_2 S_h I_v - Q_1 E_h] \\ &+ W_3 [\alpha_h E_h - Q_2 I_h] + W_4 \left( \frac{S_v - S_h^0}{S_v} \right) [b_2 - \beta_3 S_v I_h - \mu_v S_v] \\ &+ W_5 [\beta_3 S_v I_h - Q_3 E_v] + W_6 [\alpha_v E_v - Q_4 I_v], \end{aligned} \tag{20}$$

where  $\prime$  denotes the derivative with respect to time  $t$ . Using  $S_h^0 = \frac{b_1}{\mu_h}$  and  $S_v^0 = \frac{b_2}{\mu_v}$  in (20), we have

$$\begin{aligned} V'(t) &= -\mu_h W_1 \frac{(S_h - S_h^0)^2}{S_h} - \mu_v W_4 \frac{(S_v - S_v^0)^2}{S_v} + (W_5 - W_4) \beta_3 S_v I_h + (W_2 - W_1) [\beta_1 S_h I_h + \beta_2 S_h I_v] \\ &+ (W_3 \alpha_h - W_2 Q_1) E_h + (W_6 \alpha_v - W_5 Q_3) E_v \\ &+ \left[ \frac{W_4 b_2 \beta_3}{\mu_v} + \frac{W_1 b_1 \beta_1}{\mu_h} - W_3 Q_2 \right] I_h \left[ \frac{W_1 b_1 \beta_2}{\mu_h} - W_6 Q_4 \right] I_v. \end{aligned} \tag{21}$$

Let us choose  $W_1 = W_2 = \alpha_h/Q_1$ ,  $W_3 = 1$ ,  $W_4 = W_5 = \frac{b_1\alpha_h\alpha_v\beta_2}{\mu_h Q_1 Q_3 Q_4}$ ,  $W_6 = \alpha_h b_1 \beta_2 / \mu_h Q_1 Q_4$  and rewriting Eq. (21) with some little rearrangement, we get

$$V'(t) = -\frac{\alpha_h \mu_h (S_h - S_h^0)^2}{Q_1 S_h} - \frac{b_1 \alpha_h \alpha_v \beta_2 \mu_v (S_v - S_v^0)^2}{\mu_h Q_1 Q_3 Q_4 S_v} - Q_2 (1 - R_0) I_h. \tag{22}$$

Thus  $V'(t)$  is negative if  $R_0 \leq 1$ . Also note that,  $V'(t) = 0$  if and only if  $S_h = S_h^0, S_v = S_v^0, E_h = I_h = R_h = 0, E_v = I_v = 0$ . Therefore the largest compact invariant set in  $\{(S_h, E_h, I_h, R_h, S_v, E_v, I_v) \in \Omega : V'(t) = 0\}$  is the singleton  $\{E_1\}$ , where  $E_1$  is the disease-free equilibrium point. Hence LaSalle’s invariant principle [21] then implies that  $E_1$  is globally asymptotically stable in  $\Omega$ . This completes the proof.  $\square$

A global stability result for the endemic equilibrium  $E_2$  of the system (1) is given below.

**Theorem 5.2.** *If  $R_0 > 1$ , then the endemic equilibrium  $E_2$  of the system (1) is globally asymptotically stable on  $\Omega$  if*

$$\begin{cases} \mu_h = \frac{b_1}{S_h^*}, \\ \mu_v = \frac{b_2}{S_v^*}, \\ \alpha_h = \frac{Q_1 Q_2}{2\beta_1 S_h^*}, \\ \alpha_v = \frac{Q_3 Q_4 \beta_1}{\beta_2 \beta_3 S_v^*}. \end{cases} \tag{23}$$

**Proof.** Define the Lyapunov function

$$L(t) = \frac{1}{\beta_1 S_h^*} (S_h - S_h^* \log S_h) + \frac{1}{\beta_3 S_v^*} (S_v - S_v^* \log S_v) + \frac{1}{\beta_1 S_h^*} E_h + \frac{2}{Q_2} I_h + \frac{1}{\beta_3 S_v^*} E_v + \frac{\beta_2}{Q_4 \beta_1} I_v. \tag{24}$$

Calculating the time derivative of  $L$  along the solutions of the system (1), we obtain

$$\begin{aligned} L'(t) = & \frac{1}{\beta_1 S_h^*} (S_h - S_h^*) \left( \frac{b_1}{S_h} - \beta_1 I_h - \beta_2 I_v - \mu_h \right) + \frac{1}{\beta_3 S_v^*} (S_v - S_v^*) \left( \frac{b_2}{S_v} - \beta_3 I_h - \mu_v \right) \\ & + \frac{1}{\beta_1 S_h^*} (\beta_1 S_h I_h + \beta_2 S_h I_v - Q_1 E_h) + \frac{2}{Q_2} (\alpha_h E_h - Q_2 I_h) + \frac{1}{\beta_3 S_v^*} (\beta_3 S_v I_h - Q_3 E_v) + \frac{\beta_2}{Q_4 \beta_1} (\alpha_v E_v - Q_4 I_v). \end{aligned} \tag{25}$$

After some rearrangement we have

$$L'(t) = -\frac{\mu_h}{\beta_1} \left( \frac{S_h}{S_h^*} + \frac{S_h^*}{S_h} - 2 \right) - \frac{\mu_v}{\beta_3} \left( \frac{S_v}{S_v^*} + \frac{S_v^*}{S_v} - 2 \right). \tag{26}$$

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

$$\frac{S_h}{S_h^*} + \frac{S_h^*}{S_h} \geq 2 \quad \text{and} \quad \frac{S_v}{S_v^*} + \frac{S_v^*}{S_v} \geq 2. \tag{27}$$

Thus, the condition (23) ensures that  $L'(t) \leq 0$  for all  $(S_h, E_h, I_h, R_h, S_v, E_v, I_v) \in \Omega$ , and the strict equality  $L'(t) = 0$  holds only for  $S_h = S_h^*, S_v = S_v^*, E_h = E_h^*, I_h = I_h^*, R_h = R_h^*, E_v = E_v^*,$  and  $I_v = I_v^*$ . Then, the equilibrium state  $E_2$  is the only positively invariant set of the system (1) contained entirely in  $\Omega = \{(S_h, E_h, I_h, R_h, S_v, E_v, I_v), S_h = S_h^*, S_v = S_v^*, E_h = E_h^*, I_h = I_h^*, R_h = R_h^*, E_v = E_v^*\}$  and hence by the asymptotic stability theorem [21], the positive endemic equilibrium state  $E_2$  is globally asymptotically stable on  $\Omega$ .  $\square$

### 6. Numerical results and discussion

In this section the model is solved using Runge–Kutta fourth order scheme. The techniques in [22–24] can be used for solving a wide range of problems whose mathematical models yield system of differential equations. The values of some of the parameters in the model are dictated by reality, e.g. the death rates of the humans and mosquitoes, the duration of the infectious period in the human, disease induced death rate of human and mosquitoes, etc. As we have pointed out, a person infected with the dengue virus is only infectious during the viremia period, which lasts around three days. The recovery rate should be equal to 1/3 per day and not the inverse of the length of the illness. The values of the parameters determined by nature are  $\mu_h = 0.000039$  per day, corresponding to a life expectancy of human is 70 years,  $\mu_v = 0.1$  per day corresponding to a mosquito mean life of 10 days,  $\beta_1$  and  $\beta_2$  are the transmission probabilities of dengue from human



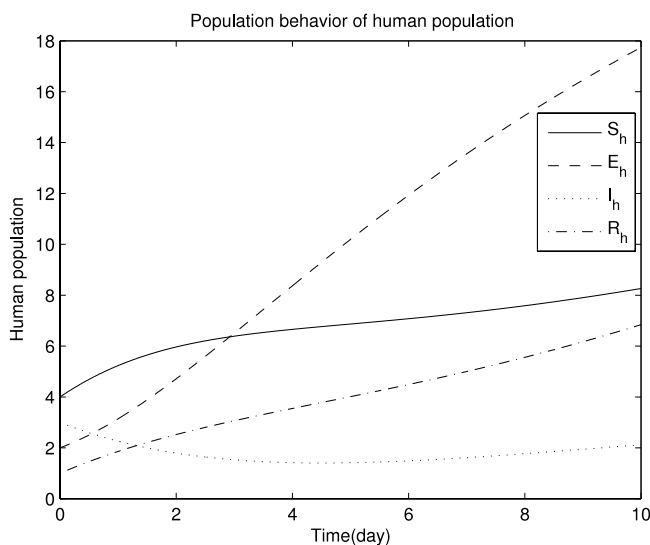


Fig. 3. The plot shows the human population.

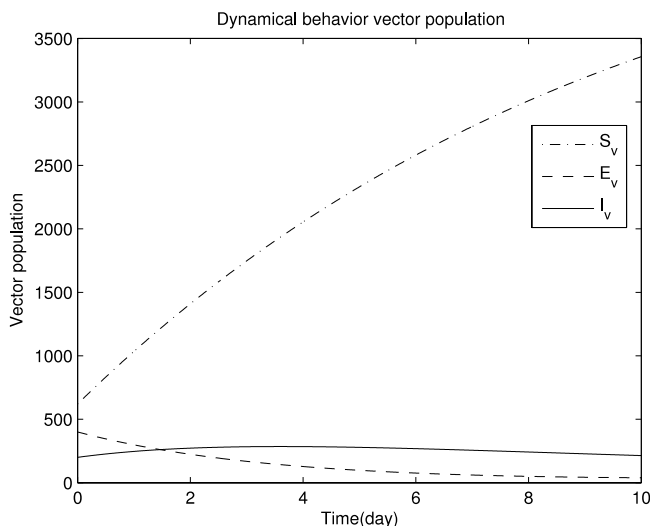


Fig. 4. The plot shows the vector population.

Table 1

Parameters used for numerical simulation.

Notation	Parameter definition	Value	Resource
$b_1$	Recruitment rate of humans	$2.5 \text{ day}^{-1}$	[25]
$\mu_h^{-1}$	Average human life expectancy	70 years	[26]
$\alpha_h$	Progression rate from $E_h$ to $I_h$ class	$(0, 1) \text{ day}^{-1}$	[20]
$\gamma_h$	Recovery rate for humans	$1/3 \text{ day}^{-1}$	[26]
$\delta_h$	Disease-induced death rate for humans	$10^{-3} \text{ day}^{-1}$	[25]
$b_2$	Recruitment rate of mosquitoes	$500 \text{ day}^{-1}$	[27]
$\mu_v^{-1}$	Average lifespan of mosquitoes	(4, 14) days	[8,27]
$\delta_v$	Disease-induced death rate for mosquitoes	negligible $\text{day}^{-1}$	[8]

to human and vector to human population respectively,  $\beta_3$  is the transmission probability of dengue from human to vector population,  $\alpha_v$  is the progression rate from  $E_v$  to  $I_v$  class; these parameters are arbitrarily chosen. For illustration purposes, we consider the parameters value in Table 1, with  $\beta_1 = 0.00001$ ,  $\beta_2 = 0.0012$ ,  $\beta_3 = 0.001$ , and  $\alpha_v = 0.20$  for numerical simulation. These values are biologically feasible. Fig. 3 represents the population of human while Fig. 4 represents the vector population.

## 7. Conclusion

In this work we extended the model by taking into account exposed individuals, disease induced death rate and time dependent total population size in both host and vector population. As in epidemiological models, the model has two steady states, an uninfected steady state where the disease is not present; and an endemically infected steady state. We first established stability results and obtained that there are two equilibria which are the disease-free equilibria and the endemic equilibria. We also shown that the model exhibits the phenomenon of backward bifurcation where the locally asymptotically stable disease-free equilibrium co-exists with a locally asymptotically stable endemic equilibrium when  $R_0 < 1$ . Then, we developed Lyapunov functions to present the global stability of both the disease-free and endemic states. It is proved that the global dynamics are completely determined by the basic reproduction number  $R_0$ . If  $R_0 \leq 1$ , the disease-free equilibrium is globally stable. If  $R_0 > 1$ , a unique endemic equilibrium exists and is globally asymptotically stable. We believe that this new assumption and analysis is biologically much more plausible than the previous assumption without the exposed classes and disease induced death rate in both human and vector population.

## References

- [1] The World Health Report 2004: Changing History, WHO, Geneva, Switzerland.
- [2] R. Ross, The Prevention of Malaria, second edition, Murray, London, 1911.
- [3] G. Macdonald, The analysis of equilibrium in malaria, *Trop. Dis. Bull.* 49 (1952) 813–828.
- [4] J.L. Aron, R.M. May, The Population Dynamics of Infectious Diseases, Chapman & Hall, London, 1982.
- [5] K. Dietz, L. Molineaux, A. Thomas, A malaria model tested in the African savannah, *Bull. World Health Org.* 50 (1974) 347–357.
- [6] H.M. Wei, X.Z. Li, M. Martcheva, An epidemic model of a vector-borne disease with direct transmission and time delay, *J. Math. Anal. Appl.* 342 (2008) 895–908.
- [7] H.W. Hethcote, The mathematics of infectious diseases, *SIAM Rev.* 42 (4) (2000) 599–653.
- [8] Z. Feng, V. Hernandez, Competitive exclusion in a vector-host model for the dengue fever, *J. Math. Biol.* 35 (1997) 523–544.
- [9] Z. Qiu, Dynamical behavior of a vector-host epidemic model with demographic structure, *Comput. Math. Appl.* 56 (2008) 3118–3129.
- [10] V. Wiwanitkit, Unusual mode of transmission of dengue, *J. Infect Dev Ctries.* 30 (2009) 51–54.
- [11] J. Dushoff, W. Huang, C.C. Chavez, Backwards bifurcations and catastrophe in simple models of fatal diseases, *J. Math. Biol.* 36 (1998) 227–248.
- [12] S.M. Garba, A.B. Gumel, M.R.A. Bakar, Backward bifurcations in dengue transmission dynamics, *Math. Biosci.* 215 (2008) 11–25.
- [13] J. Hul, D. Zhu, Global stability and periodicity on SIS epidemic models with backward bifurcation, *Comput. Math. Appl.* 50 (2005) 1271–1290.
- [14] L. Cai, X. Li, Analysis of a simple vector-host epidemic model with direct transmission, *Discrete Dyn. Nat. Soc.* (2010) doi:10.1155/2010/679613.
- [15] V. Lakshmikantham, S. Leela, A.A. Martynyuk, Stability Analysis of Nonlinear Systems, Marcel Dekker Inc., New York, Basel, 1989.
- [16] R.M. Anderson, R.M. May, Infectious Diseases of Humans: Dynamics and Control, Second ed., Oxford University Press, 1991.
- [17] P. van den Driessche, J. Watmough, Reproduction number and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci.* 180 (2002) 29–48.
- [18] L.J.S. Allen, An Introduction to Mathematical Biology, Pearson Education Ltd., USA, 2007.
- [19] K.P. Hadeler, P. van den Driessche, Backward bifurcation in epidemic control, *Math. Biosci.* 146 (1997) 15–35.
- [20] O. Sharomi, C.N. Podder, A.B. Gumel, E.H. Elbasha, J. Watmough, Role of incidence function in vaccine-induced backward bifurcation in some HIV models, *Math. Biosci.* 210 (2007) 436–463.
- [21] J.P. LaSalle, The Stability of Dynamical Systems, SIAM, Philadelphia, PA, 1976.
- [22] O.D. Makinde, Adomian decomposition approach to a SIR epidemic model with constant vaccination strategy, *Appl. Math. Comput.* 184 (2007) 842–848.
- [23] Y. Khan, Q. Wu, Homotopy perturbation transform method for nonlinear equations using He's polynomials, *Comput. Math. Appl.* (2010) doi:10.1016/j.camwa.2010.08.022.
- [24] Y. Khan, An effective modification of the Laplace decomposition method for nonlinear equations, *Int. J. Nonlinear Sci. Numer. Simul.* 10 (2009) 1373–1376.
- [25] F.A.B. Coutinho, M.N. Burattini, L.F. Lopez, E. Massad, Threshold conditions for a non-autonomous epidemic system describing the population dynamics of dengue, *Bull. Math. Biology* 68 (2006) 2263–2282.
- [26] K. Patanarapelert, I.M. Tang, Effect of time delay on the transmission of dengue fever, *I. J. Biol. Life Sci.* 3 (2007) 238–246.
- [27] L. Esteva, C. Vargas, Analysis of a dengue disease transmission model, *Math. Biosci.* 150 (1998) 131–151.