Research Article

Mathematical Analysis of a Malaria Model with Partial Immunity to Reinfection

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A deterministic model with variable human population for the transmission dynamics of malaria disease, which allows transmission by the recovered humans, is first developed and rigorously analyzed. The model reveals the presence of the phenomenon of backward bifurcation, where a stable disease-free equilibrium coexists with one or more stable endemic equilibria when the associated reproduction number is less than unity. This phenomenon may arise due to the reinfection of host individuals who recovered from the disease. The model in an asymptotical constant population is also investigated. This results in a model with mass action incidence. A complete global analysis of the model with mass action incidence is given, which reveals that the global dynamics of malaria disease with reinfection is completely determined by the associated reproduction number. Moreover, it is shown that the phenomenon of backward bifurcation can be removed by replacing the standard incidence function with a mass action incidence. Graphical representations are provided to study the effect of reinfection rate and to qualitatively support the analytical results on the transmission dynamics of malaria.

1. Introduction

Malaria is a mosquito-borne disease caused by a parasite. It is endemic and widespread in tropical and subtropical regions, including much of sub-Saharan Africa, Asia, and the Americas. Malaria is still a public health problem today. Every year, there are more than 225 million cases of malaria, killing around 781,000 people according to the World Health Organization's 2010 World Malaria Report [1].

In humans, malaria is caused due to infection by one of four Plasmodium species [2, 3]. Transmission from mosquito to human occurs during a bite by an infectious mosquito. A mosquito becomes infected when it takes a blood meal from an infected human. Once ingested, the parasite gametocytes taken up in the blood will further differentiate into gametes and then fuse in the mosquito's gut. Gametocytes are responsible for transmission of the parasite from humans by mosquitoes bite. Fertilization of the parasite occurs in the mosquito gut, and after a short period of replication and development, the cycle of transmission may begin anew.

One of the most complex features of the epidemiology of malaria is the dynamic interaction between infection and immunity. A better understanding of this interaction is important for evaluating the impact of malaria control activities. An important phenomenon is noticed that the changes with age reflect the slow acquisition of an immunity that reduces illness but does not completely block infection [4, 5]. In endemic areas, children younger than five years have repeated and often serious attacks of malaria. The survivors

develop and maintain partial immunity that reduces the severity of the disease but does not prevent subsequent infections. Thus, in these areas older children and adults often have become asymptomatic carriers of infection [6]. In areas of low malaria transmission, immunity develops slowly and may take years or decades and probably never results in sterile immunity [7]. Therefore, humans are susceptible to reinfections. Incomplete immunity to malaria complicates disease control strategies [8, 9] as the partially immune individuals suffer only mild infections, and might not seek medical attention but continue to transmit the parasite in the community.

The enormous public health burden inflicted by malaria disease necessitates the use of mathematical modeling and analysis to gain insights into its transmission dynamics, and to determine effective control strategies. The earliest malaria transmission models can be traced to the model formulated by Ross in 1911 [9]. He used a mathematical model and showed that bringing a mosquito population below a certain threshold was sufficient to eliminate malaria. This threshold naturally depended on biological factors such as the biting rate and vectorial capacity. To estimate infection and recovery rates, MacDonald extended the Ross model in 1957 [9]. Macdonald's model shows that reducing the number of mosquitoes is an inefficient control strategy. Moreover, this would have little effect on the epidemiology of malaria in areas of intense transmission. Since then, the emergence and reemergence of malaria diseases have promoted many author's interest in mathematical modeling to describe and to predict the transmission dynamics of malaria in the literature (see, e.g., [10–16] and the references therein). In paper [12], Dietz et al. applied the Garki model to show that the duration of acquired immunity in humans in malaria depends on repeated exposure. In paper [13], Niger and Gumel constructed a mathematical model that includes multiple infected and recovered classes, to assess the role of the partial immunity on the transmission dynamics of malaria in a human population. Their results reveals the presence of the phenomenon of backward bifurcation in the standard incidence model with the disease-induced death in the human population. Recently, a transmission model of human malaria in a partially immune population is formulated in Wan and Cui's paper [14]. They established the basic reproduction number and explicit subthreshold conditions for the model, and showed that if the disease induced death rate is large enough, the model undergoes a backward bifurcation. Li [15] formulated a malaria transmission model with partial immunity in humans and showed that the established model having the same reproductive number but different numbers of progression stages can exhibit different transient dynamics. Thus, the above mentioned models always let the recovered individuals return into the susceptible class to explore the transmission dynamics of diseases. But this only takes states of complete immunity and full susceptibility in consideration. In addition, various vector-borne disease model concerning malaria transmission have been established and discussed [17–20]. For example, in paper [17], Yang et al have investigated global stability of an epidemic model for vector-borne disease, however, they assumed that the immunity of the recovered population have never lose.

Motivated by the recent work of [13, 15], in this paper, we shall continue to construct a malaria transmission model with partial immunity to reinfection in the recovered human population. Our purpose is to explore the transmission dynamics of the malaria and to assess the role of partial immunity to reinfection on the transmission dynamics of malaria in a human population.

The organization of this paper is as follows: in the next section, the standard incidence malaria model, which incorporates the partial immunity to reinfection, is formulated. The existence and stability of the equilibria, and the phenomena of the backward bifurcation are, respectively, explored in Sections 2.2 and 2.3. Graphical representations are provided to study the effect of reinfection rate in Section 2.4. In Section 3, the associated mass action incidence model is formulated, and mathematical results such as existence and local stability of equilibria are provided in Section 3.2. Our main theorems for the global stability of equilibria for the mass action model and the proofs are given in Section 3.3. The paper ends with a conclusion in Section 4.

2. Model Formulation

We formulate a model for the spread of malaria in the human and mosquito population, with the total population size at time t given by $\overline{N}_h(t)$ and $\overline{N}_v(t)$, respectively. The total human population is divided into three epidemiological classes: $\overline{S}_h(t)$, $\overline{I}_h(t)$, and $\overline{R}_h(t)$, which denote, respectively, the number of the susceptible, infective, and immune class at time t. Thus, $\overline{N}_h(t) = \overline{S}_h(t) + \overline{I}_h(t) + \overline{R}_h(t)$. The susceptible human population is generated by the recruitment of humans (assumed susceptible) into the community at a rate Λ_h, μ_h, and γ_h are, respectively, the natural death rate and recovery rate in human hosts population. Also, some disease-induced death in human population contributes to an additional population decrease at the constant rate δ_h.

Due to its short life, a mosquito never recovers from the infection, and we may not consider the recovered class in this population. Thus, the total vector population $\overline{N}_v(t)$ is divided into the susceptible class, $\overline{S}_v(t)$, and infective class, $\overline{I}_v(t)$, so that $\overline{N}_v(t) = \overline{S}_v(t) + \overline{I}_v(t)$. Susceptible mosquitoes vectors are generated at a rate Λ_v by birth, μ_v is the per capita mortality rate of mosquitoes. Let β_h be the transmission probability from vector to human, and β_v be the transmission probability from human to vector. The parameter b is the average number of bites per mosquito per day. This rate depends on a number of factors, in particular, climatic ones, but for simplicity in this paper we assume b to be a constant. The parameter σ ($0 \leq \sigma \leq 1$) determines the degree of partial protection for the recovered individuals given by a primary infection: $\sigma = 0$ implies complete protection, and $\sigma = 1$ implies no protection. Taking into account the assumptions made above, the interaction between human hosts and the mosquito vector population with partial immunity to reinfection in

host population is described by the following system of equations:

$$\frac{d\overline{S}_h}{dt} = \Lambda_h - \frac{b\beta_h \overline{S}_h \overline{I}_v}{\overline{N}_h} - \mu_h \overline{S}_h,$$

$$\frac{d\overline{I}_h}{dt} = \frac{b\beta_h \overline{S}_h \overline{I}_v}{\overline{N}_h} + \frac{\sigma b\beta_h \overline{R}_h \overline{I}_v}{\overline{N}_h} - (\gamma_h + \delta_h + \mu_h)\overline{I}_h,$$

$$\frac{d\overline{R}_h}{dt} = \gamma_h \overline{I}_h - \frac{\sigma b\beta_h \overline{R}_h \overline{I}_v}{N_h} - \mu_h \overline{R}_h,$$

$$\frac{d\overline{S}_v}{dt} = \Lambda_v - \frac{b\beta_v \overline{S}_v \overline{I}_h}{\overline{N}_h} - \mu_v \overline{S}_v,$$

$$\frac{d\overline{I}_v}{dt} = \frac{b\beta_v \overline{S}_v \overline{I}_h}{\overline{N}_h} - \mu_v \overline{I}_v.$$

$$(1)$$

The total humans host and mosquitoes vector populations $\overline{N}_h = \overline{S}_h + \overline{I}_h + \overline{R}_h$ and $\overline{N}_v = \overline{S}_v + \overline{I}_v$ are governed, respectively, by

$$\frac{d\overline{N}_h}{dt} = \Lambda_h - \mu_h \overline{N}_h - \delta_h \overline{I}_h,$$

$$\frac{d\overline{N}_v}{dt} = \Lambda_v - \mu_v \overline{N}_v.$$

$$(2)$$

It is easily seen that for the mosquitoes vector population the corresponding total population size is asymptotically constant: $\lim_{t \to \infty} \overline{N}_v(t) = \Lambda_v/\mu_v$. This implies that in our model we assume without loss of generality that $\overline{N}_v(t) = \Lambda_v/\mu_v$, for all $t \geq 0$, provided that $\overline{S}_v(0) + \overline{I}_v(0) = \Lambda_v/\mu_v$. Let

$$S_h = \frac{\overline{S}_h}{\Lambda_h/\mu_h}, \qquad I_h = \frac{\overline{I}_h}{\Lambda_h/\mu_h}, \qquad R_h = \frac{\overline{R}_h}{\Lambda_h/\mu_h},$$

$$S_v = \frac{\overline{S}_v}{\Lambda_v/\mu_v}, \qquad I_v = \frac{\overline{I}_v}{\Lambda_v/\mu_v}, \qquad N_h = \frac{\overline{N}_h}{\Lambda_h/\mu_h}.$$

$$(3)$$

Since $N_h = S_h + I_h + R_h$ and $S_v + I_v = 1$. Using $R_h = N_h - S_h - I_h$ and $S_v = 1 - I_v$, system (1) is reduced to the following four-dimensional nonlinear system of ODEs:

$$\frac{dS_h}{dt} = \mu_h - \frac{bm\beta_h S_h I_v}{N_h} - \mu_h S_h,$$

$$\frac{dI_h}{dt} = \frac{bm\beta_h S_h I_v}{N_h} + \frac{\sigma bm\beta_h (N_h - S_h - I_h) I_v}{N_h} - \alpha_1 I_h,$$

$$\frac{dI_v}{dt} = \frac{b\beta_v (1 - I_v) I_h}{N_h} - \mu_v I_v,$$

$$\frac{dN_h}{dt} = \mu_h - \mu_h N_h - \delta_h I_h,$$

$$(4)$$

where $m = (\Lambda_v/\mu_v)/(\Lambda_h/\mu_h)$ and $\alpha_1 = \gamma_h + \mu_h + \delta_h$.

2.1. Basic Properties of the Model. Since the model (4) monitors humans host and mosquitoes vector populations, it is plausible to assume that all its state variables and parameters are nonnegative for all $t \geq 0$. Further, it can be shown that the region Ω given by

$$\Omega = \left\{ (S_h, I_h, N_h, I_v) \in R_+^4 : \right.$$

$$\left. 0 \leq S_h + I_h \leq N_h \leq 1, \ 0 \leq I_v \leq 1 \right\}$$

$$(5)$$

is positively invariant with respect to system (4). Thus, every solution of the model (4), with initial conditions in Ω remains there for $t > 0$. Therefore, it is sufficient to consider the dynamics of the flow generated by (4) in Ω. In this region, the model can be considered as been epidemiologically and mathematically well posed.

2.2. Stability of Disease-Free Equilibria. The stability of the disease-free equilibrium state can be obtained from studying the eigenvalues of the Jacobian matrix evaluated at the equilibrium point. If all the eigenvalues have negative real parts, then the equilibrium point is stable. The disease-free equilibrium for the system (4) is $E_0(1, 0, 1, 0)$. The Jacobian matrix at the disease-free equilibrium E_0 is

$$J = \begin{pmatrix} -\mu_h & 0 & 0 & -bm\beta_h \\ 0 & -\alpha_1 & 0 & bm\beta_h \\ 0 & -\delta_h & -\mu_h & 0 \\ 0 & b\beta_v & 0 & -\mu_v \end{pmatrix}.$$

$$(6)$$

The characteristic equation of the above matrix is

$$(\lambda - \mu_h)(\lambda - \mu_h)(\lambda^2 + (\alpha_1 + \mu_v)\lambda + \alpha_1 \mu_v(1 - \mathbb{R}_0)) = 0,$$

$$(7)$$

where $\mathbb{R}_0 = b^2 m\beta_h \beta_v/\mu_v \alpha_1$. There are four eigenvalues corresponding to (7). Two of the eigenvalues $\lambda_1, \lambda_2 = -\mu_h$ have negative real parts. The other two eigenvalues can be obtained from the equation

$$\lambda^2 + (\alpha_1 + \mu_v)\lambda + \alpha_1 \mu_v(1 - \mathbb{R}_0) = 0. \qquad (8)$$

Applying the Routh-Hurwitz criteria for a quadratic polynomial. It is easy to see that both the coefficients of (8) are positive if an only if $\mathbb{R}_0 < 1$. Thus, all roots of (8) are with negative real parts if $\mathbb{R}_0 < 1$, and one of its roots is with positive real part if $\mathbb{R}_0 > 1$. Therefore, the disease-free equilibrium (DFE) E_0 is locally asymptotically stable if $\mathbb{R}_0 < 1$ and unstable if $\mathbb{R}_0 > 1$. Thus, we have the following result.

Theorem 1. *The uninfected equilibrium E_0 is locally asymptotically stable if $\mathbb{R}_0 < 1$ and unstable if $\mathbb{R}_0 > 1$ in Ω.*

From Theorem 1, the threshold quantity $\mathbb{R}_0$, is called the basic reproduction number of system (4). The basic reproduction number, $\mathbb{R}_0$ measures the average number of new malaria infections generated by a single infected individual in a completely susceptible population [21]. Theorem 1 also implies that malaria can be eliminated from the community (when $\mathbb{R}_0 < 1$) if the initial sizes of the subpopulations of

the model are in the basin of attraction of the disease-free equilibrium (DFE) (E_0). To ensure that disease elimination is independent of the initial sizes of the subpopulations, it is necessary to show that the DFE is globally asymptotically stable (GAS) if $\mathbb{R}_0 < 1$. This is explored for a special case in Section 3.2.

2.3. The Existence of Endemic Equilibria and Backward Bifurcation.

In this section, conditions for the existence of endemic equilibria and phenomenon of backward bifurcation in system (4) will be determined. In order to do this, we let

$$E^* = (S_h^*, I_h^*, N_h^*, I_v^*) \qquad (9)$$

represent an arbitrary endemic equilibrium of the model (4). Setting the right-hand sides of the equations in (4) to zero and solving them in terms of I_h^* gives the following expressions for the state variables of the model

$$S_h^* = \frac{(\mu_h - \delta I_h^*)(\alpha_2 I_h^* + \mu_h \mu_v)}{b^2 m \mu_h \beta_h \beta_v I_h^* + (\mu_h - \delta I_h^*)(\alpha_2 I_h^* + \mu_h \mu_v)}, \qquad (10)$$

$$N_h^* = \frac{\mu_h - \delta_h I_h^*}{\mu_h}, \qquad I_v^* = \frac{\mu_h b \beta_v I_h^*}{\alpha_2 I_h^* + \mu_h \mu_v}, \qquad (11)$$

where $\alpha_2 = \mu_h b \beta_v - \delta_h \mu_v$, and I_h^* is determined from the following equation:

$$K_1 I_h^{*4} + K_2 I_h^{*3} + K_3 I_h^{*2} + K_4 I_h^* + K_5 = 0, \qquad (12)$$

where

$$K_1 = \delta_h^2 \alpha_1 \alpha_2^2,$$

$$K_2 = \alpha_1 \left(-2\delta_h \alpha_2 (\mu_h \alpha_2 - \delta_h \mu_h \mu_v) - \delta_h \alpha_2 \mu_h b^2 m \beta_h \beta_v \right)$$
$$\quad - \mu_h \delta_h \alpha_2 \alpha_3 \sigma b^2 m \beta_h \beta_v,$$

$$K_3 = \alpha_1 \left((\mu_h \alpha_2 - \delta_h \mu_h \mu_v)^2 - 2\mu_h^2 \mu_v \delta_h \alpha_2 \right.$$
$$\quad + \mu_h b^2 m \beta_h \beta_v (\mu_h \alpha_2 - \delta_h \mu_h \mu_v) \Big)$$
$$\quad + \mu_h^2 \delta_h \alpha_2 b^2 m \beta_h \beta_v + \mu_h \alpha_3 \sigma b^2 m \beta_h \beta_v \qquad (13)$$
$$\quad \times \left((\mu_h \alpha_2 - \delta_h \mu_h \mu_v) + \mu_h b^2 m \beta_h \beta_v \right),$$

$$K_4 = \alpha_1 \left(2\mu_h^2 \mu_v (\mu_h \alpha_2 - \delta_h \mu_h \mu_v) + \mu_h^3 \mu_v b^2 m \beta_h \beta_v \right)$$
$$\quad - \mu_h^2 b^2 m \beta_h \beta_v (\mu_h \alpha_2 - \delta_h \mu_h \mu_v)$$
$$\quad - \sigma \mu_h^3 b^2 m \beta_h \beta_v \left(b^2 m \beta_h \beta_v - \alpha_3 \mu_v \right),$$

$$K_5 = \alpha_1 \mu_h^4 \mu_v^2 (1 - \mathbb{R}_0),$$

and $\alpha_3 = \mu_h + \delta_h$.

It follows from (13) that $K_1 > 0$. Further, $K_5 > 0$ whenever $\mathbb{R}_0 < 1$. Thus, the number of possible positive real roots for (12) depends on the signs of K_2, K_3 and K_4. This can be analyzed using the Descartes Rule of Signs on the quartic $f(I_h^*) = K_1 I_h^{*4} + K_2 I_h^{*3} + K_3 I_h^{*2} + K_4 I_h^* + K_5$. The various

possibilities for the roots of $f(I_h^*)$ are tabulated in Table 1. We have the following result from the various possibilities enumerated in Table 1.

Theorem 2. *The system* (4) *has a unique endemic equilibrium E^* if $\mathbb{R}_0 > 1$ and Cases 1–3 and 6 are satisfied; it could have more than one endemic equilibrium if $\mathbb{R}_0 > 1$ and Cases 4, 5, 7, and 8 are satisfied; it could have 2 or more endemic equilibria if $\mathbb{R}_0 < 1$ and Cases 2–8 are satisfied.*

The existence of multiple endemic equilibria when $\mathbb{R}_0 < 1$ (is shown in Table 1). Table 1 suggests the possibility of backward bifurcation (see [22–24]), where the stable DFE coexists with a stable endemic equilibrium, when the reproduction number is less than unity. Thus, the occurrence of a backward bifurcation has an important implications for epidemiological control measures, since an epidemic may persist at steady state even if $\mathbb{R}_0 < 1$. This is explored below by using Centre Manifold Theory (see, e.g., [25] and the references therein).

Now, we shall establish the conditions on parameter values that cause a backward bifurcation to occur in system (4), based on the use of Center Manifold theory, of the paper in Castillo-Chavez and Song [25].

Theorem 3. *Let one consider the following general system of ordinary differential equations with a parameter ϕ:*

$$\frac{dx}{dt} = f(x, \phi), \quad f : \mathbb{R}^n \times \mathbb{R} \longrightarrow \mathbb{R}^n, \quad f \in C^2 (\mathbb{R} \times \mathbb{R}). \qquad (14)$$

Without loss of generality, it is assumed that $x = 0$ is an equilibrium for system (14) *for all values of the parameter ϕ. Assume that*

(A1) *$A = D_x f(0, 0)$ is the linearized matrix of system* (14) *around the equilibrium $x = 0$ with ϕ evaluated at 0. Zero is a simple eigenvalues of A and all other eigenvalue of A have negative real parts;*

(A2) *Matrix A has a nonnegative right eigenvector $\mathbf{w}$ and a left eigenvector $\mathbf{v}$ corresponding to the zero eigenvalue.*

Let f_k be the kth component of f and

$$a_1 = \sum_{i,j,k=1}^{5} v_k w_i w_j \frac{\partial^2 f_k (0,0)}{\partial x_i \partial x_j},$$
$$b_1 = \sum_{i,k=1}^{5} v_k w_i \frac{\partial^2 f_k (0,0)}{\partial x_i \partial \beta_h}. \qquad (15)$$

The local dynamics of system (14) *around 0 are totally determined by a_1 and b_1.*

(i) *In the case where $a_1 > 0$, $b_1 > 0$, one has that when $\phi < 0$ with $|\phi|$ close to zero, $x = 0$ is unstable; when $0 < \phi \ll 1$, $x = 0$ is unstable and there exists a negative and locally asymptotically stable equilibrium;*

(ii) *In the case where $a_1 < 0$, $b_1 < 0$, one has that when $\phi < 0$ with $|\phi|$ close to zero, $x = 0$ is locally asymptotically*

TABLE 1: Number of possible positive real roots of $f(I_h^*)$ for $\mathbb{R}_0 > 1$ and $\mathbb{R}_0 < 1$.

Cases	K_1	K_2	K_3	K_4	K_5	$\mathbb{R}_0$	Number of sign change	Number of positive real roots
1	+	+	+	+	+	$\mathbb{R}_0 < 1$	0	0
	+	+	+	+	−	$\mathbb{R}_0 > 1$	1	1
2	+	−	−	−	+	$\mathbb{R}_0 < 1$	2	0, 2
	+	−	−	−	−	$\mathbb{R}_0 > 1$	1	1
3	+	+	−	−	+	$\mathbb{R}_0 < 1$	2	0, 2
	+	+	−	−	−	$\mathbb{R}_0 > 1$	1	1
4	+	−	+	−	+	$\mathbb{R}_0 < 1$	4	0, 2, 4
	+	−	+	−	−	$\mathbb{R}_0 > 1$	3	1, 3
5	+	−	−	+	+	$\mathbb{R}_0 < 1$	2	0, 2
	+	−	−	+	−	$\mathbb{R}_0 > 1$	3	1, 3
6	+	+	+	−	+	$\mathbb{R}_0 < 1$	2	0, 2
	+	+	+	−	−	$\mathbb{R}_0 > 1$	1	1
7	+	+	−	+	+	$\mathbb{R}_0 < 1$	2	0, 2
	+	+	−	+	−	$\mathbb{R}_0 > 1$	3	1, 3
8	+	−	+	+	+	$\mathbb{R}_0 < 1$	2	0, 2
	+	−	+	+	−	$\mathbb{R}_0 > 1$	3	1, 3

stable and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$, $x = 0$ is locally asymptotically stable, and there exists a positive unstable equilibrium;

(iii) In the case where $a_1 > 0$, $b_1 < 0$, one has that when $\phi < 0$ with $|\phi|$ close to zero, $x = 0$ is unstable and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$, $x = 0$ is stable and a positive unstable equilibrium appears;

(iv) In the case where $a_1 < 0$, $b_1 > 0$, one has that when $\phi < 0$ changes from negative to positive, $x = 0$ changes its stability from stable to unstable. Correspondingly a_1 negative unstable equilibrium becomes positive and locally asymptotically stable. Particularly, if $a_1 > 0$ and $b_1 > 0$, then a backward bifurcation occurs at $\phi = 0$.

To apply the center manifold method, the following simplification and change of variables are made on the model (4). First of all, let $x_1 = S_h$, $x_2 = I_h$, $x_3 = R_h$, $x_4 = S_v$, and $x_5 = I_v$, so that $N_h = x_1 + x_2 + x_3$ and $N_v = x_4 + x_5$. Further, by using the vector notation $X = (x_1, x_2, x_3, x_4, x_5)^T$, the system (4) can be written in the form $(dX/dt) = (f_1, f_2, f_3, f_4, f_5)^T$ as follows:

$$\frac{dx_1}{dt} = f_1 = \mu_h - \frac{bm\beta_h x_1 x_5}{x_1 + x_2 + x_3} - \mu_h x_1,$$

$$\frac{dx_2}{dt} = f_2 = \frac{bm\beta_h x_1 x_5}{x_1 + x_2 + x_3} + \frac{\sigma bm\beta_h x_3 x_5}{x_1 + x_2 + x_3} - \alpha_1 x_2,$$

$$\frac{dx_3}{dt} = f_3 = \gamma_h x_2 - \frac{\sigma bm\beta_h x_3 x_5}{x_1 + x_2 + x_3} - \mu_h x_3, \quad (16)$$

$$\frac{dx_4}{dt} = f_4 = \mu_v - \frac{b\beta_v x_4 x_2}{x_1 + x_2 + x_3} - \mu_v x_4,$$

$$\frac{dx_5}{dt} = f_5 = \frac{b\beta_v x_4 x_2}{x_1 + x_2 + x_3} - \mu_v x_5.$$

Choose β_h as a bifurcation parameter and solving $\mathbb{R}_0 = 1$ gives

$$\beta_h = \beta_h^* = \frac{\mu_v \alpha_1}{b^2 m \beta_v}. \quad (17)$$

The Jacobian matrix evaluated at disease-free equilibrium $(1, 0, 0, 1, 0)$ with $\beta_h = \beta_h^*$ is

$$J = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & -bm\beta_h \\ 0 & -\alpha_1 & 0 & 0 & bm\beta_h \\ 0 & \gamma_h & -\mu_h & 0 & 0 \\ 0 & -b\beta_v & 0 & -\mu_v & 0 \\ 0 & b\beta_v & 0 & 0 & -\mu_v \end{pmatrix}. \quad (18)$$

It can be easily seen that the Jacobian J of the linearized system has a simple zero eigenvalue and all other eigenvalues have negative real parts. Hence, the center manifold theory can be used to analyze the dynamics of the system (16). For the case when $R_0 = 1$, it can be shown that the Jacobian matrix J has a right eigenvector (corresponding to the zero eigenvalue) given by $\mathbf{w} = [w_1 \ w_2 \ w_3 \ w_4 \ w_5]^T$, where

$$w_1 = \frac{-bm\beta_h}{\mu_h} w_5, \qquad w_2 = \frac{bm\beta_h}{\alpha_1} w_5, \qquad w_3 = \frac{\gamma_h bm\beta_h}{\mu_h \alpha_1} w_5,$$

$$w_4 = \frac{-b^2 m\beta_h \beta_v}{\mu_v \alpha_1} w_5, \qquad w_5 = w_5 > 0.$$

$$\quad (19)$$

Similarly, the components of the left eigenvector of J (corresponding to the zero eigenvalue), denoted by $\mathbf{v} = [v_1 \ v_2 \ v_3 \ v_4 \ v_5]$, are given by

$$v_1 = v_3 = v_4 = 0, \qquad v_2 = \frac{b\beta_v}{\alpha_1} v_5, \qquad v_5 = v_5 > 0. \quad (20)$$

Computation of a_1: for the transformed system (16), the associated non-zero partial derivatives of f (evaluated at the DFE) which we need in the computation of a_1 are given by

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_5} = \frac{\partial^2 f_2}{\partial x_5 \partial x_2} = -bm\beta_h,$$

$$\frac{\partial^2 f_2}{\partial x_3 \partial x_5} = \frac{\partial^2 f_2}{\partial x_5 \partial x_3} = -bm\beta_h + \sigma bm\beta_h,$$

$$\frac{\partial^2 f_5}{\partial x_1 \partial x_2} = \frac{\partial^2 f_5}{\partial x_2 \partial x_1} = -b\beta_v, \qquad \frac{\partial^2 f_5}{\partial x_2^2} = -2b\beta_v,$$

$$\frac{\partial^2 f_5}{\partial x_2 \partial x_3} = \frac{\partial^2 f_5}{\partial x_3 \partial x_2} = -b\beta_v, \qquad \frac{\partial^2 f_5}{\partial x_2 \partial x_4} = \frac{\partial^2 f_5}{\partial x_4 \partial x_2} = b\beta_v. \tag{21}$$

Direct calculations shows that

$$a_1 = 2b\left(-m\beta_h v_2 w_2 w_5 - m\beta_h v_2 w_3 w_5 + \sigma m\beta_h v_2 w_3 w_5\right.$$

$$- \beta_v v_5 w_1 w_2 - \beta_v v_5 w_2^2 \tag{22}$$

$$\left. - \beta_v v_5 w_2 w_3 + \beta_v v_5 w_2 w_4\right).$$

Computation of b_1: Substituting the vectors $\mathbf{v}$ and $\mathbf{w}$ and the respective partial derivatives (evaluated at the DFE) into the expression

$$b_1 = \sum_{i,k=1}^{5} v_k w_i \frac{\partial^2 f_k(0,0)}{\partial x_i \partial \beta_h} \tag{23}$$

gives $b_1 = bm v_2 w_5 > 0$. Since the coefficient b_1 is automatically positive, it follows that the sign of the coefficient a_1 decides the local dynamics around the disease-free equilibrium for $\beta_h = \beta_h^*$. Based on Theorem 3, system (4) will undergo backward bifurcation if the coefficient a_1 is positive. The coefficient a_1 is positive if and only if

$$\sigma > \frac{1}{\gamma_h \mu_v}\left(\mu_v\left(\gamma_h + \mu_h\right) + b\mu_h \beta_v - \mu_v \delta_h\right). \tag{24}$$

Thus, we have the following result.

Theorem 4. *The system* (4) *exhibits backward bifurcation whenever the condition* (24) *holds.*

The backward bifurcation phenomenon is illustrated by simulating the system (4) with the following set of parameter values $\mu_h = 0.00004$, $\mu_v = 0.015$, $\Lambda_v = 4$, $\Lambda_h = 3$, $\beta_v = 0.2$, $b = 0.4$, $\delta_h = 0.0011$, $\gamma_h = 0.0005$ (so that, $a_1 > 0$ and $\mathbb{R}_0 < 1$). Figure 1 depicts the associated backward bifurcation diagram.

2.4. The Effect of the Reinfection. We further investigate the effect of the reinfection parameter σ and the transmission probability from an infectious human to a susceptible vector β_h on the associated backward bifurcation region, as a function of the average life span of mosquitoes ($1/\mu_v$). The backward bifurcation region is illustrated (Figures 2–4) by simulating the model (4) with the following set of parameter
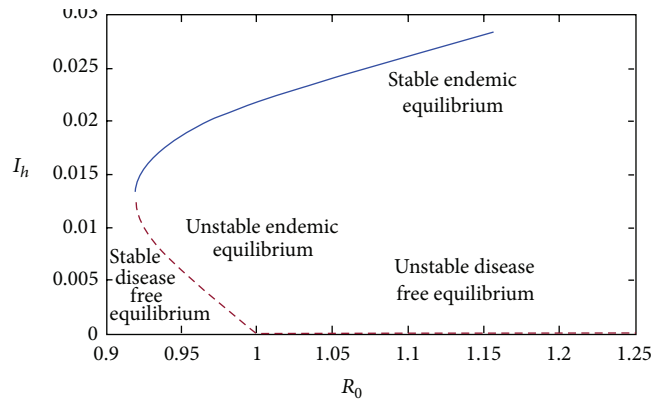

FIGURE 1: Simulations of the model (4) illustrating the phenomenon of backward bifurcation.

values (note that the parameters are chosen in order to illustrate the backward bifurcation region, and may not all be realistic epidemiologically), $\Lambda_h = 30$, $\Lambda_v = 24$, $\beta_v = 0.09$, $\delta_h = 0.2$, $\gamma_h = 0.0005$, $\mu_h = 0.00004$, $b = 0.4$, $\mu_v = 0.2$. Also to be noted is, the parameter values are chosen such that $a_1 > 0$, $b_1 > 0$ and $\mathbb{R}_0 < 1$ (so that backward bifurcation occurs).

Solving for $a_1 > 0$ in terms of $0 \leqslant \sigma \leqslant 1$ and $\beta_h > 0$ (i.e., fixing all parameters in the expression for a_1 except β_h and σ) we obtained the backward bifurcation region for β_h. Figure 2 depicted the results obtained for $\sigma = 0.5$, it shows that the region for backward bifurcation (for β_h) increases as the average life span of vectors ($1/\mu_v$) decreases. For instance, when the average life span of vectors is 20 days ($\mu_v = 0.05$), the backward bifurcation region for β_h is $\beta_h \in [0.10381, 0.35556]$, as shown in Figure 2(a). When the average life span of vectors is decreased to 10 days ($\mu_v = 0.1$), the backward bifurcation region for β_h increases to $\beta_h \in [0.2077, 0.71083]$ (Figure 2(b)). Furthermore, when the average life span of vectors is decreased to 5 days ($\mu_v = 0.2$), the backward bifurcation region for β_h increases to $\beta_h \in [0.4153, 1.4217]$ (Figure 2(c)). Similar results are obtained for the cases $\sigma = 0.6$ (Figures 3(a), 3(b), and 3(c)) and $\sigma = 1$ (Figures 4(a), 4(b), and 4(c)), from which it is evident that the backward bifurcation regions for β_h increase with increasing values of the reinfection rate σ. These results are tabulated in Table 2 ($1/\mu_v$ represents average life span of vectors.)

3. The Mass Action Model

In this section, we shall investigate the dynamics of system (1) if mass action incidence is used instead of the standard incidence function. Thus the resulting (mass action) model is

$$S_h'(t) = \Lambda_h - b\beta_h S_h(t) I_v(t) - \mu_h S_h(t),$$

$$I_h'(t) = b\beta_h S_h(t) I_v(t) + \sigma b\beta_h R_h(t) I_v(t)$$

$$- \left(\mu_h + \gamma_h + \delta_h\right) I_h(t),$$
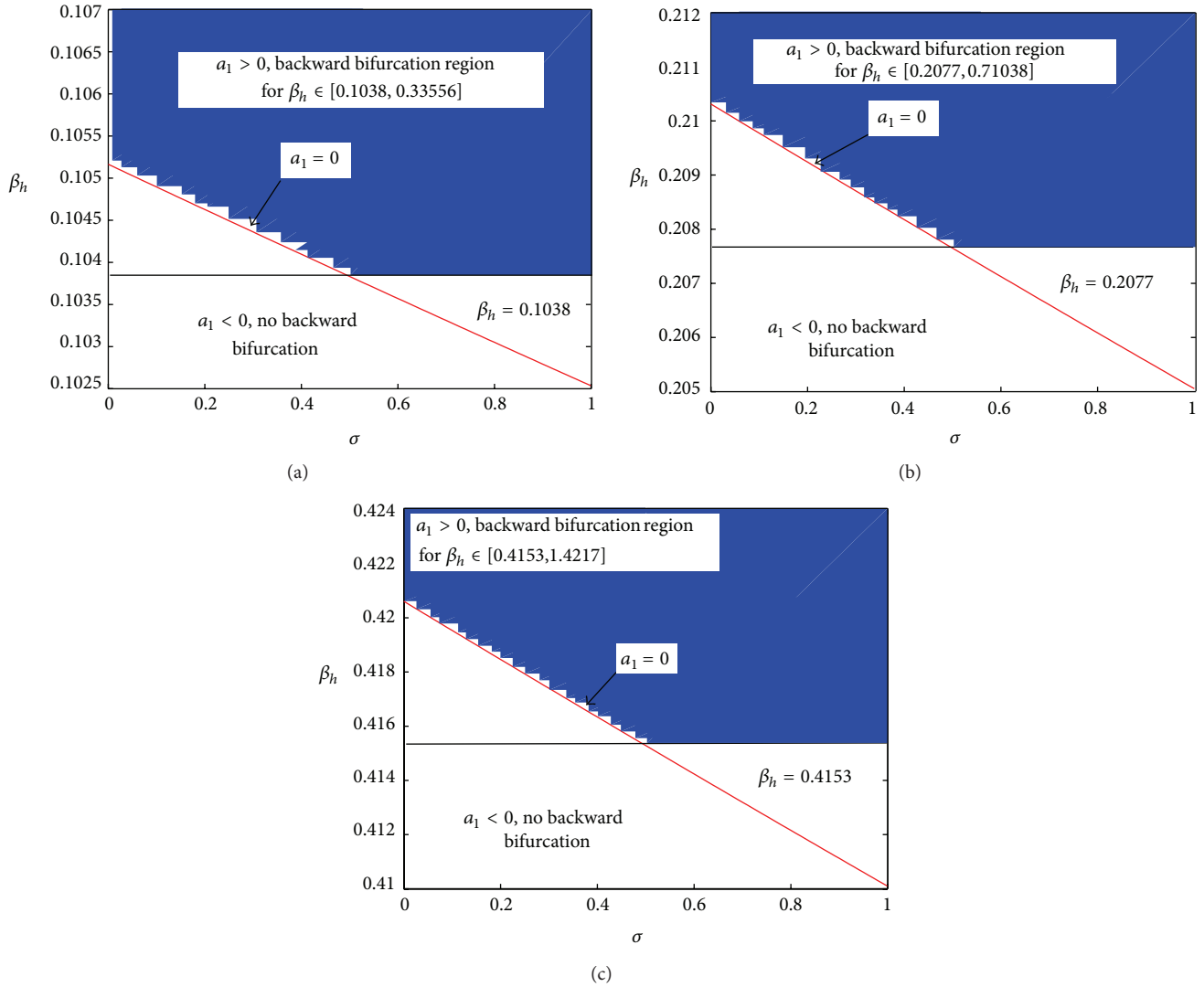
(a)

(b)

(c)

FIGURE 2: Backward bifurcation regions for the model (4) in the σ-β_h parameter space corresponding to $\sigma = 0.5$ and various ranges of β_h. Parameter values used are: $\Lambda_h = 30$, $\Lambda_v = 24$, $\delta_h = 0.2$, $\gamma_h = 0.0005$, $\mu_h = 0.00004$, $b = 0.4$, $\mu_v = 0.0015$, $\beta_v = 0.09$. In (a) $\mu_v = 0.05$, $\mathbb{R}_0 = 0.0014046$ (backward bifurcation region for β_h is $\beta_h \in [0.1038, 0.35556]$), (b) $\mu_v = 0.1$, $\mathbb{R}_0 = 0.00035113$ (backward bifurcation region for β_h is $\beta_h \in [0.2077, 0.71083]$), and (c) $\mu_v = 0.2$, $\mathbb{R}_0 = 0.0000087788$ (backward bifurcation region for β_h is $\beta_h \in [0.4153, 1.9468]$). With the above set of parameter values, $a_1 > 0$, $b_1 > 0$, and $\mathbb{R}_0 < 1$.

$$R'_h(t) = \gamma_h I_h(t) - \sigma b \beta_h R_h(t) I_v(t) - \mu_h R_h(t),$$

$$S'_v(t) = \Lambda_v - b \beta_v I_h(t) S_v(t) - \mu_v S_v(t),$$

$$I'_v(t) = b \beta_v I_h(t) S_v(t) - \mu_v I_v(t),$$

(25)

where the prime ($\prime$) stands for the derivative with respect to time t and initial conditions $S_h(0) \geq 0$, $I_h(0) \geq 0$, $R_h(0) \geq 0$, and $S_v(0) \geq 0$, $I_v(0) \geq 0$.

3.1. Basic Properties: Positivity and Invariant Regions. The dynamics of the total human population, obtained by adding first three equations in the model (25), is given by

$$N'_h(t) = \Lambda_h - \mu_h N_h(t) - \delta_h I_h(t).$$

(26)

Thus, we have

$$\frac{\Lambda_h}{\mu_h + \delta_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h + \delta_h} \right) e^{-(\mu_h + \delta_h)t}$$

$$\leq N_h(t) \leq \frac{\Lambda_h}{\mu_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h} \right) e^{-\mu_h t}.$$

(27)

Thus, for a low level of disease induced death rate ($\delta_h \approx 0$) total human population could eventually assume a steady-state value. Motivated by this, we consider a human population which assumes a steady-state value Λ_h/μ_h stationary. Similarly, the dynamics of the total mosquito population, obtained by adding last two equations in the model (25), is given by, $N'_v(t) = \Lambda_v - \mu_v N_v(t)$, so that, $N_v(t) = \Lambda_v/\mu_v$ as $t \to \infty$.

(a)

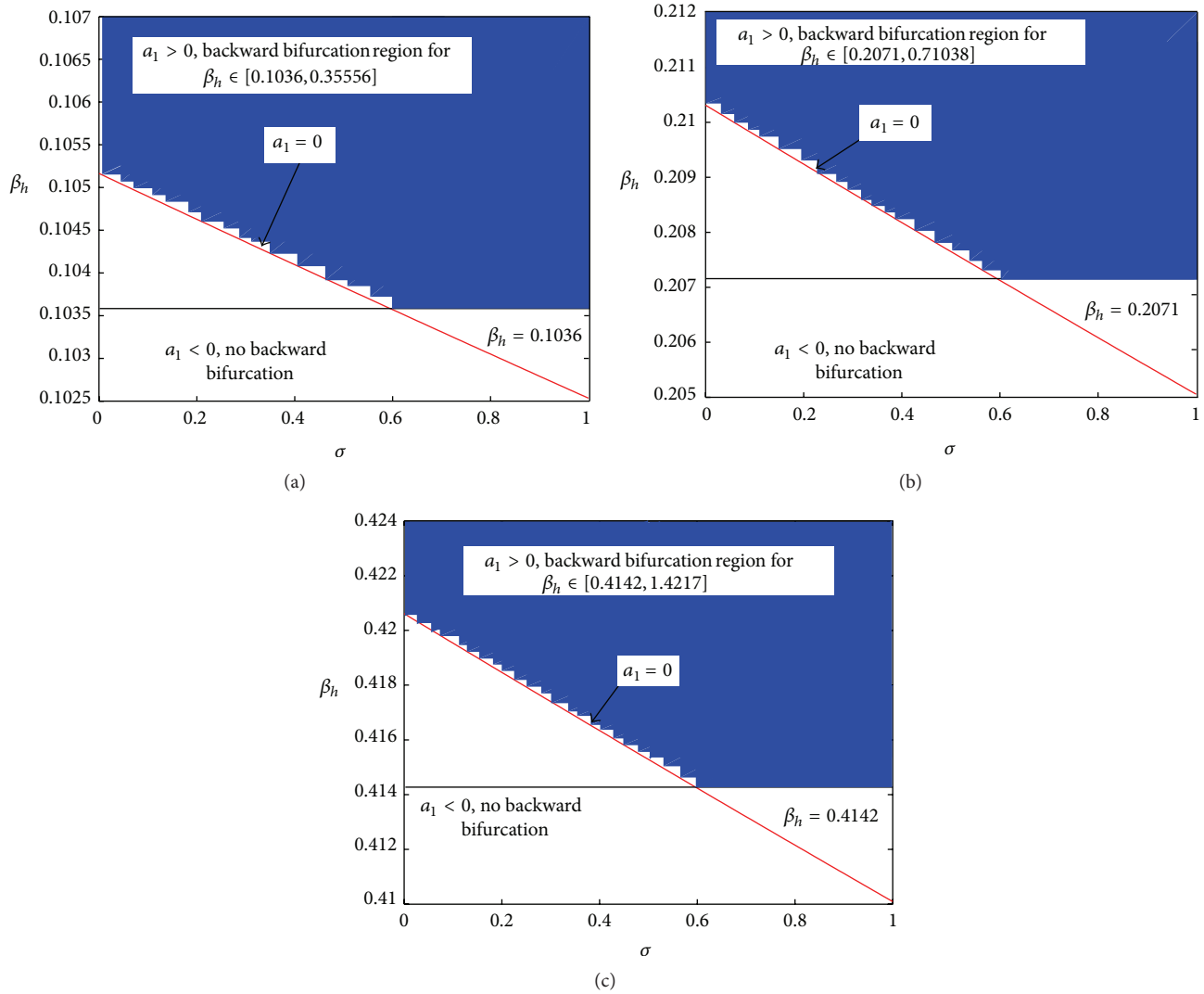(b)

(c)

FIGURE 3: Backward bifurcation regions for the model (4) in the σ-β_h parameter space corresponding to $\sigma = 0.6$ and various ranges of β_h. Parameter values used are: $\Lambda_h = 30, \Lambda_v = 24, \delta_h = 0.2, \gamma_h = 0.0005, \mu_h = 0.00004, b = 0.4, \mu_v = 0.0015, \beta_v = 0.09$. In (a) $\mu_v = 0.05$, $\mathbb{R}_0 = 0.0014046$ (backward bifurcation region for β_h is $\beta_h \in [0.1036, 0.35556]$), (b) $\mu_v = 0.1$, $\mathbb{R}_0 = 0.00035113$ (backward bifurcation region for β_h is $\beta_h \in [0.2071, 0.71083]$), and (c) $\mu_v = 0.2$, $\mathbb{R}_0 = 0.0000087788$ (backward bifurcation region for β_h is $\beta_h \in [0.4142, 1.4217]$). With the above set of parameter values, $a_1 > 0$, $b_1 > 0$, and $\mathbb{R}_0 < 1$.

Let $H(t) = (S_h(t), I_h(t), R_h(t))$ and $V(t) = (S_v(t), I_v(t))$. Based on the above discussion, we define a region

$$\Gamma = \left\{ (H(t), V(t)) \in R_+^3 \times R_+^2 \mid 0 \leq H(t) \right.$$

$$\left. \leq \frac{\Lambda_h}{\mu_h}, \ 0 \leq V(t) \leq \frac{\Lambda_v}{\mu_v} \right\}. \tag{28}$$

It is easy to verify that Γ is positively invariant with respect to the system (25). In this part, it is sufficient to consider the dynamics of the flow generated by (25) in Γ.

3.2. Equilibrium and Local Stability. In this section, we investigate the existence and local stability of equilibria of system (25). Obviously, the system (25) always has

a disease-free equilibrium $E_{\text{mass}}^0(\Lambda_h/\mu_h, 0, 0, \Lambda_v/\mu_v, 0)$. Let $E_{\text{mass}}^*(S_h^*, I_h^*, R_h^*, S_v^*, I_v^*)$ represents any arbitrary endemic equilibrium of the model (25). Solving the equations in (25) at steady state gives

$$S_h^* = \frac{\Lambda_h \mu_v (b\beta_v I_h^* + \mu_v)}{b^2 \Lambda_v \beta_h \beta_v I_h^* + \mu_h \mu_v (b\beta_v I_h^* + \mu_v)},$$

$$R_h^* = \frac{\gamma_h \mu_v (b\beta_v I_h^* + \mu_v) I_h^*}{b^2 \Lambda_v \sigma \beta_h \beta_v I_h^* + \mu_h \mu_v (b\beta_v I_h^* + \mu_v)}, \tag{29}$$

$$S_v^* = \frac{\Lambda_v}{b\beta_v I_h^* + \mu_v}, \qquad I_v^* = \frac{b\Lambda_v \beta_v I_h^*}{\mu_v (b\beta_v I_h^* + \mu_v)},$$
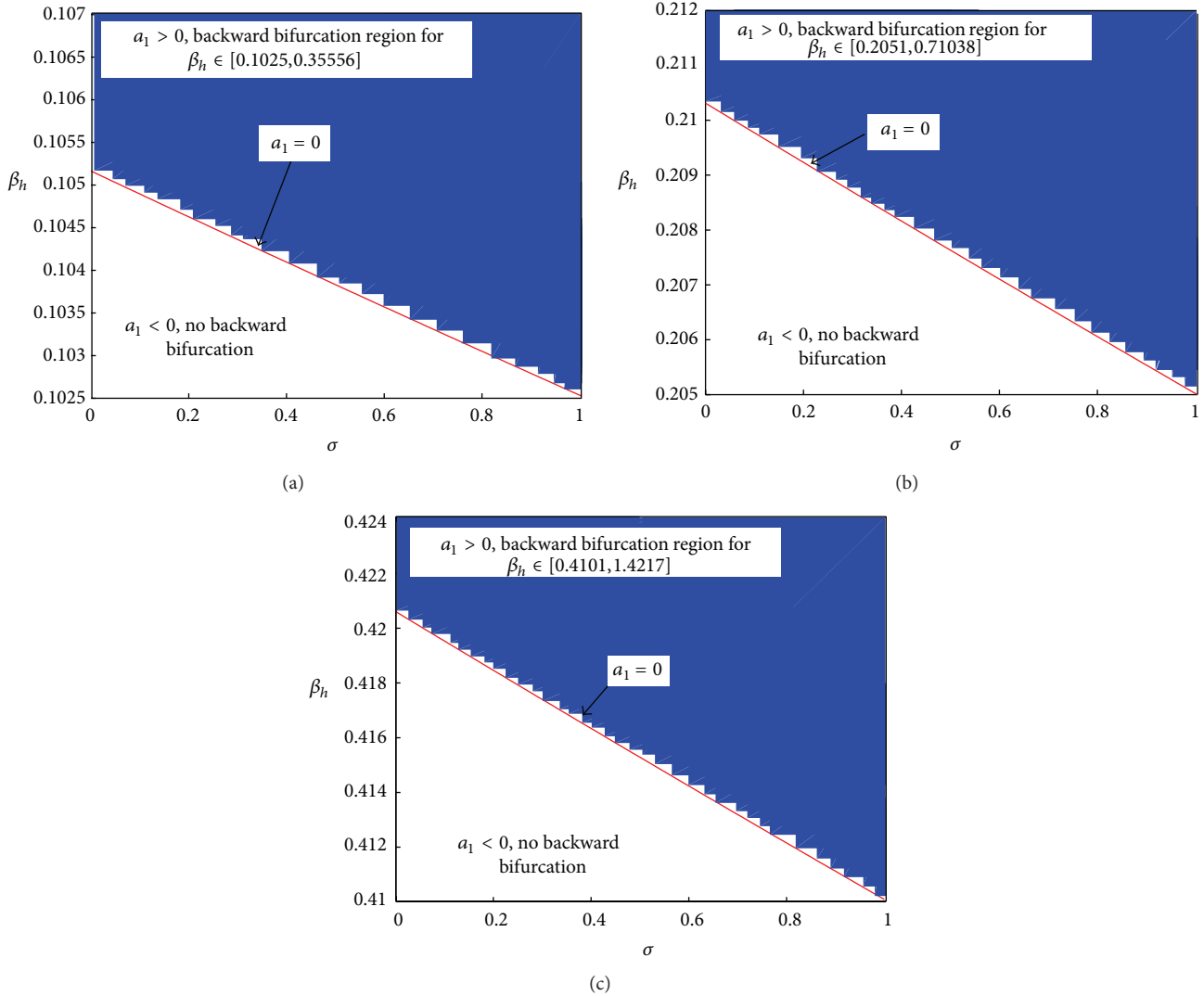
(a)

(b)

(c)

FIGURE 4: Backward bifurcation regions for the model (4) in the σ-β_h parameter space corresponding to $\sigma = 1$ and various ranges of β_h. Parameter values used are: $\Lambda_h = 30, \Lambda_v = 24, \delta_h = 0.2, \gamma_h = 0.0005, \mu_h = 0.00004, b = 0.4, \mu_v = 0.0015, \beta_v = 0.09$. In (a) $\mu_v = 0.05, \mathbb{R}_0 = 0.0014046$ (backward bifurcation region for β_h is $\beta_h \in [0.1025, 0.35556]$), (b) $\mu_v = 0.1, \mathbb{R}_0 = 0.00035113$ (backward bifurcation region for β_h is $\beta_h \in [0.2051, 0.71083]$), and (c) $\mu_v = 0.2, \mathbb{R}_0 = 0.0000087788$ (backward bifurcation region for β_h is $\beta_h \in [0.4101, 1.4217]$). With the above set of parameter values, $a_1 > 0$, $b_1 > 0$, and $\mathbb{R}_0 < 1$.

where I_h^* is the positive root of the following quadratic equation,

$$d_1 I_h^{*2} + d_2 I_h^* + d_3^* = 0, \tag{30}$$

with

$$
\begin{aligned}
d_1 &= b^2 \beta_v^2 \left(\mu_h + \delta_h\right) \left(\sigma b \Lambda_v \beta_h + \mu_h \mu_v\right) \left(b \Lambda_v \beta_h + \mu_h \mu_v\right) \\
&\quad + \gamma_h b^2 \beta_v^2 \mu_h \mu_v \left(b \Lambda_v \beta_h + \mu_h \mu_v\right) > 0, \\
d_2 &= b \beta_v \mu_h \mu_v^2 \left(\mu_h + \gamma_h + \delta_h\right) \left(b \Lambda_v \beta_h + \mu_h \mu_v\right) \\
&\quad + b \beta_v \mu_h \mu_v^2 \left(\mu_h + \delta_h\right) \left(\sigma b \Lambda_v \beta_h + \mu_h \mu_v\right) + \gamma_h b \beta_v \mu_h^2 \mu_v^3
\end{aligned}
$$

$$- b^3 \Lambda_v \Lambda_h \beta_h \beta_v^2 \left(b \sigma \Lambda_v \beta_h + \mu_v \mu_h\right),$$

$$d_3 = \left(\mu_h + \gamma_h + \delta_h\right) \mu_h^2 \mu_v^4 \left(1 - \frac{b^2 \Lambda_h \Lambda_v \beta_h \beta_v}{\left(\mu_h + \gamma_h + \delta_h\right) \mu_h \mu_v^2}\right). \tag{31}$$

The dynamics of the model (25) are analyzed by $\mathbb{R}_{\text{mass}}$ given by

$$\mathbb{R}_{\text{mass}} = \frac{b^2 \Lambda_h \Lambda_v \beta_h \beta_v}{\left(\mu_h + \gamma_h + \delta_h\right) \mu_h \mu_v^2}. \tag{32}$$

The threshold quantity $\mathbb{R}_{\text{mass}}$ is the basic reproduction number of the system (25). It can be derived from the Jacobian matrix of the system (25) at the disease-free equilibrium E_{mass}^0 together with the assumption of local asymptotical stability

TABLE 2: Backward Bifurcation Ranges for β_h for Various Values of $1/\mu_v$ and σ.

Average life span of vectors $(1/\mu_v)$	$\sigma = 0.5$	$\sigma = 0.6$	$\sigma = 1$
20 days	$\beta_h \in [0.1038, 0.35556]$	$\beta_h \in [0.1036, 0.35556]$	$\beta_h \in [0.1025, 0.35556]$
10 days	$\beta_h \in [0.2077, 0.71083]$	$\beta_h \in [0.2071, 0.71083]$	$\beta_h \in [0.2051, 0.71083]$
5 days	$\beta_h \in [0.4153, 1.4217]$	$\beta_h \in [0.4142, 1.4217]$	$\beta_h \in [0.4101, 1.4217]$

of E^0_{mass} [21]. From (31), we see that $\mathbb{R}_{mass} > 1$ if and only if, $d_3 < 0$. Since $d_1 > 0$, (30) has a unique positive root in feasible region Ω. If $\mathbb{R}_{mass} < 1$, then $d_3 > 0$. Also $b^2 \Lambda_h \Lambda_v \beta_h \beta_v < (\mu_h + \gamma_h + \delta_h)\mu_h\mu_v$, is equivalent to $\mathbb{R}_{mass} < 1$. Hence, $d_2 > 0$. Thus, by considering the shape of the graph $f(I_h) = d_1 I_h^2 + d_2 I_h + d_3$ (and noting that $d_1 > 0$), we have that there will be zero endemic equilibria in this case. Therefore, we can conclude that if $\mathbb{R}_{mass} < 1$, (30) has no positive root in the feasible region Γ. If, $\mathbb{R}_{mass} = 1$, (30) has a unique positive root in the feasible region Γ. This result is summarized below.

Theorem 5. *System* (25) *always has the infection-free equilibrium* $E^0_{mass} = ((\Lambda_h/\mu_h), 0, 0, (\Lambda_v/\mu_v), 0)$. *If* $\mathbb{R}_{mass} > 1$, *system* (25) *has a unique endemic equilibrium* $E^*_{mass} = (S^*_h, I^*_h, R^*_h, S^*_v, I^*_v)$ *defined by* (29) *and* (30).

Linearizing the system (25) around the disease-free equilibrium E^0_{mass} yields the following characteristic equation:

$$(\lambda + \mu_h)^2 \left[\lambda^2 + (\mu_h + \gamma_h + \delta_h + \mu_v)\lambda \right.$$
$$\left. + (\mu_h + \gamma_h + \delta_h)\mu_v - b^2 \beta_h \beta_v \frac{\Lambda_h \Lambda_v}{\mu_h \mu_v} \right] = 0. \tag{33}$$

Two of the roots of the characteristic equation (33) $\lambda_{1,2} = -\mu_h$, have negative real parts. The other two roots can be determine from the quadratic term in (33) and have negative real parts if and only if $\mathbb{R}_{mass} < 1$. Therefore, the disease-free equilibrium E^0_{mass} is locally asymptotically stable for $\mathbb{R}_{mass} < 1$. When $\mathbb{R}_{mass} > 1$, E^0_{mass} becomes an unstable equilibrium point, and the endemic equilibrium E^*_{mass} emerges in Γ. This result is summarized below.

Theorem 6. *The disease-free equilibrium* E^0_{mass} *of system* (25) *is locally asymptotically stable if* $\mathbb{R}_{mass} < 1$ *and unstable if* $\mathbb{R}_{mass} > 1$.

In order to discuss the stability of the endemic equilibrium E^*_{mass} and to simplify our calculations, we assume both humans and mosquitoes populations are at steady state. Thus, using $N_v = S_h + I_h + R_h = \lambda_v/\mu_h$, and $N_v = S_v + I_v = \lambda_v/\mu_v$, system (25) in the invariant space Γ can be written as the following equivalent three dimensional nonlinear system of ODEs:

$$S'_h(t) = \Lambda_h - b\beta_h S_h(t) I_v(t) - \mu_h S_h(t),$$

$$I'_h(t) = b\beta_h S_h(t) I_v(t) + \sigma b\beta_h (N_h - S_h(t) - I_h(t))$$

$$\times I_v(t) - (\mu_h + \gamma_h + \delta_h) I_h(t),$$

$$I'_v(t) = b\beta_v I_h(t) (N_v - I_v(t)) - \mu_v I_v(t). \tag{34}$$

Now, linearization of system (34) about an endemic equilibrium E^*_{mass} gives the following characteristic equation:

$$\begin{vmatrix} \lambda + \mu_h + b\beta_h I^*_v & 0 & b\beta_h S^*_h \\ b\beta_h (\sigma - 1) I^*_v & \lambda + \alpha_1 + b\sigma\beta_h I^*_v & -\dfrac{\alpha_1 I^*_h}{I^*_v} \\ 0 & -\dfrac{\mu_v I^*_v}{I^*_h} & \lambda + b\beta_v I^*_h + \mu_v \end{vmatrix} = 0, \tag{35}$$

where, $\alpha_1 = \mu_h + \gamma_h + \delta_h$. Expanding (35) gives

$$\lambda^3 + Q_1 \lambda^2 + Q_2 \lambda + Q_3 = 0, \tag{36}$$

where

$$Q_1 = \mu_h + b\beta_h I^*_v + \alpha_1 + b\sigma\beta_h I^*_v + \mu_v + b\beta_v I^*_h > 0,$$

$$Q_2 = (\mu_h + b\beta_h I^*_v)(\alpha_1 + b\sigma\beta_h I^*_v + \mu_v + b\beta_v I^*_h)$$
$$+ b\beta_v I^*_h (\alpha_1 + b\sigma\beta_h I^*_v) + \mu_v b\sigma\beta_h I^*_v > 0,$$

$$Q_3 = (\mu_h + b\beta_h I^*_v) [b\beta_v I^*_h (\alpha_1 + b\sigma\beta_h I^*_v) + \mu_v b\sigma\beta_h I^*_v]$$
$$+ b^2 \beta_h^2 (1 - \sigma) I^*_v S^*_h \frac{\mu_v I^*_v}{I^*_h} > 0. \tag{37}$$

From the second equation of system (34) at steady state E^*, we have

$$\alpha_1 = \frac{b\beta_h S^*_h I^*_v (1 - \sigma) + b\sigma N_h \beta_h I^*_v}{I^*_h} - b\sigma\beta_h I^*_v. \tag{38}$$

Using (37), direct calculations show that

$$Q_1 Q_2 - Q_3$$

$$= \left(\underline{\mu_h + b\beta_h I_v^*} + \alpha_1 + b\sigma\beta_h I_v^* + \underline{\mu_v + b\beta_v I_h^*} \right)$$

$$\times \left[\underline{b\beta_v I_h^* (\alpha_1 + b\sigma\beta_h I_v^*) + \mu_v b\sigma\beta_h I_v^*} \right.$$

$$\left. + \left(\mu_h + \underline{b\beta_h I_v^*} \right) \left(\underline{\alpha_1 + b\sigma\beta_h I_v^*} + \mu_v + b\beta_v I_h^* \right) \right]$$

$$- \left[\underline{(\mu_h + b\beta_h I_v^*) \left[b\beta_v I_h^* (\alpha_1 + b\sigma\beta_h I_v^*) + \mu_v b\sigma\beta_h I_v^* \right]} \right.$$

$$\left. + b^2 \beta_h^2 (1-\sigma) I_v^* S_h^* \frac{\mu_v I_v^*}{I_h^*} \right].$$

(39)

Obviously, the term $(\mu_h + b\beta_h I_v^*)$ in first bracket times the term $(b\beta_v I_h^*(\alpha_1 + b\sigma\beta_h I_v^*) + \mu_v b\sigma\beta_h I_v^*)$ in the second bracket is $(\mu_h + b\beta_h I_v^*)[b\beta_v I_h^*(\alpha_1 + b\sigma\beta_h I_v^*) + \mu_v b\sigma\beta_h I_v^*]$. Multiplying the terms under straight line and using (30), we have $\mu_v b\beta_h I_v(\alpha_1 + b\sigma\beta_h I_v^*) > b^2 \beta_h^2 (1-\sigma) I_v^* S_h^* (\mu_v I_v^*/I_h^*)$. Hence, $Q_1 Q_2 - Q_3 > 0$. Thus, by Routh Hurwitz criteria, the following result is established.

Theorem 7. *The endemic equilibrium E^*_{mass} of the reduced model* (34) *is locally asymptotically stable if* $\Re_{mass} > 1$.

3.3. Global Stability of the Equilibria. In this section, the global stability of the equilibria of system (34) will be explored. First, we claim the following theorem.

Theorem 8. *If* $\Re_{mass} \leq 1$, *then the infection-free-equilibrium E^0_{mass} of system* (34) *is globally asymptotically stable in* Γ.

Proof. To establish the global stability of the disease-free equilibrium E^0_{mass}, we construct the following Lyapunov function

$$L(t) = b\beta_v I_h(t) + (\mu_h + \gamma_h + \delta_h) I_v(t). \tag{40}$$

Calculating the derivative of L (where a dot represents differentiation with respect to t) along the solutions of (34) we obtain

$$L'(t) = b\beta_v I_h'(t) + (\mu_h + \gamma_h + \delta_h) I_v'(t),$$

$$= b\beta_v \left[b\beta_h S_h(t) I_v(t) + b\sigma\beta_h (N_h - S_h(t) - I_h(t)) I_v(t) \right.$$

$$\left. - (\mu_h + \gamma_h + \delta_h) I_h(t) \right]$$

$$+ (\mu_h + \gamma_h + \delta_h) \left[b\beta_v (N_v - I_v(t)) I_h(t) - \mu_v I_v(t) \right],$$

$$= b^2 \beta_v \beta_h (S_h(t) + \sigma (N_h - S_h(t) - I_h(t)))$$

$$\times I_v(t) - (\mu_h + \gamma_h + \delta_h) \mu_v I_v(t)$$

$$- b(\mu_h + \gamma_h + \delta_h) \beta_v I_h(t) I_v(t),$$

$$= (\mu_h + \gamma_h + \delta_h) \mu_v$$

$$\times \left[\frac{b^2 \beta_v \beta_h}{(\mu_h + \gamma_h + \delta_h) \mu_v} \right.$$

$$\times (S_h(t) + \sigma(N_h - S_h(t) - I_h(t))) - 1 \left] I_v(t) \right.$$

$$- b(\mu_h + \gamma_h + \delta_h) \beta_v I_h(t) I_v(t),$$

$$\leq (\mu_h + \gamma_h + \delta_h) \mu_v I_v(t) (R_{mass} - 1)$$

$$- b(\mu_h + \gamma_h + \delta_h) \beta_v I_h(t) I_v(t).$$

(41)

Thus, $L'(t) \leq 0$ if $R_{mass} \leq 1$ with $L'(t) = 0$ if and only if $I_v(t) = 0$. Thus, from the second and the first equation of system (34), we have $\lim_{t\to\infty} I_h(t) = 0$, and $\lim_{t\to\infty} S_h(t) = \Lambda_h/\mu_h$. Therefore, the largest compact invariant set in $\{(S_h(t), I_h(t), I_v(t)) \in \alpha : L'(t) = 0\}$ is the singleton $\{(\Lambda_h/\mu_h, 0, 0)\}$ in Γ. Using the LaSalle's invariant principle [26], the infection-free equilibrium E^0_{mass} is globally asymptotically stable for $R_{mass} \leq 1$ in Γ. The epidemiological implication of the above result is that malaria will be eliminated from the population if R_{mass} can be brought to (and maintained at) a value less than unity. Thus, the substitution of standard incidence with mass action incidence in the model (1) removes the phenomenon of backward bifurcation. The result of Theorem 8 is illustrated numerically by simulating the model (30), for the case $R_{mass} < 1$, using various initial conditions. The solution profiles obtained shows convergence to the DFE, as depicted in Figure 5.

Now, we investigate the global stability of the endemic equilibrium E^*_{mass}. We notice that when the incomplete immunity term $0 < \sigma < 1$, system (30) is no longer competitive. To investigate the global stability of E^*_{mass}, we adopted a general approach of Li and Muldowney [27, 28], which is developed for higher dimensional systems irrespective if they are competitive. While the approach of Li and Muldowney has been successfully applied to many classes of epidemic models, we demonstrated in the present paper, for the first time, that this approach is also applicable to vector-host model which is non-competitive. □

We briefly state the approach developed recently in Li and Muldowney as follows:

Let $G \subset R^n$ be an open set and $f : x \mapsto f(x) \in R^n$ be a C^1 function for $x \in G$. Consider the following differential equation:

$$x' = f(x). \tag{42}$$

Let $x(t, x_0)$ denote the solution of (42) satisfying $x(0, x_0) = x_0$. We make the following two assumptions.

(H_1) There exists a compact absorbing set $K \subset G$.

(H_2) Equation (42) has a unique equilibrium $\bar{x}$ in G.
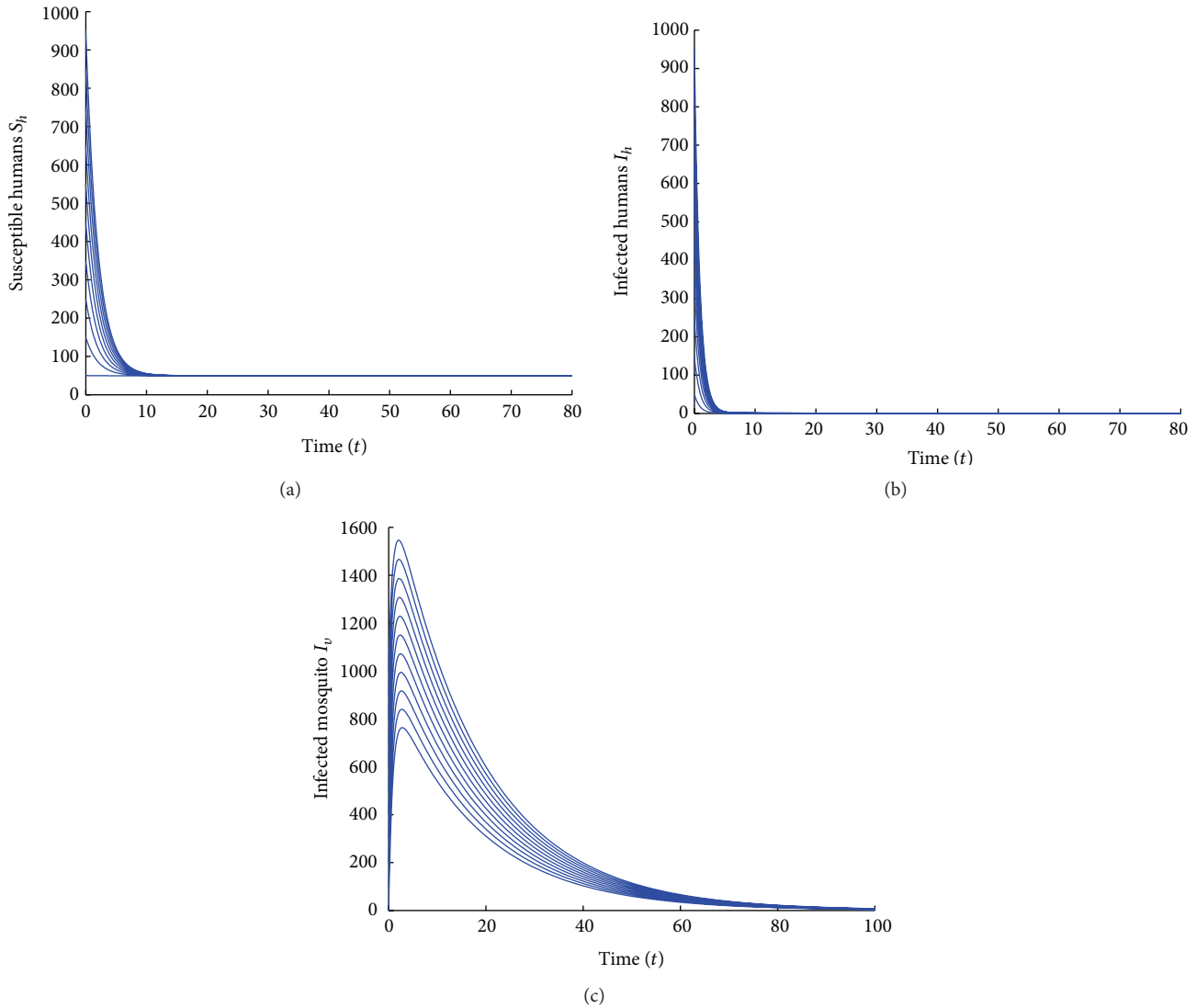
FIGURE 5: Simulations of the model (5) showing (a) the number of susceptible humans, (b) the number of infected humans, (c) the number of infected mosquitoes, as a function of time using the parameters: $b = 0.5$, $\Lambda_h = 25$, $\Lambda_V = 500$, $\beta_1 = 0.00001$, $\beta_2 = 0.005$, $\mu_h = 0.5$ $\mu_v = 0.06$, $\sigma = 0.8$, $\delta_h = 0.0001$, and $\gamma_h = 0.6$ (so that $\mathbb{R}_0 = 0.0789 < 1$).

Let $Q : x \mapsto Q(x)$ be an $\binom{n}{2} \times \binom{n}{2}$ matrix-valued function, that is, it is C^1 and $Q^{-1}(x)$ exists for $x \in G$. Let μ be a Lozinskiĭ measure on $R^{d \times d}$, where $d = \binom{n}{2}$. Define a quantity q_2 as

$$\overline{q}_2 = \lim_{t \to \infty} \sup \sup_{x_0 \in E} \frac{1}{t} \int_0^t \mu\left(M\left(x\left(s, x_0\right)\right)\right) ds, \qquad (43)$$

where $M = Q_f Q^{-1} + Q J^{[2]} Q^{-1}$, the matrix Q_f is obtained by replacing each entry q_{ij} of Q by its derivative in the direction of f, $(q_{ij})_f$, and $J^{[2]}$ is the second additive compound matrix of the Jacobian matrix J of system (42). The following results have been established in Li and Muldowney [27, 28].

Theorem 9. *For system* (42), *assume that G is a simple connected and that the assumptions* (H_1) *and* (H_2) *hold. Then, the unique equilibrium $\overline{x}$ is globally asymptotically stable in G*

if there exist a function $Q(x)$ and a Lozinskiĭ measure μ such that $\overline{q}_2 < 0$.

From the above discussion, we know that Γ is simply connect and E^*_{mass} is the unique positive equilibrium for $\mathbb{R}_{\text{mass}} > 1$ in Γ.

To apply the result of Theorem 9 to investigate the global stability of the infective equilibrium E^*_{mass}, we first state and prove the following result.

Theorem 10. *If $\mathbb{R}_{\text{mass}} > 1$, then system* (34) *is uniformly persistent, that is, there exists $\epsilon > 0$ (independent of initial conditions), such that $\lim\inf_{t \to \infty} S_h(t) > \epsilon$, $\lim\inf_{t \to \infty} I_h(t) > \epsilon$, and $\lim\inf_{t \to \infty} I_v(t) > \epsilon$.*

Proof. Similar to the proof of Theorem 3.4 in [29], we choose $X = \Gamma$, $X_1 = \text{int}\,\Gamma$, $X_2 = bd(\Gamma)$. It is easy to obtain that

$Y_2 = \{(S, 0, 0) : 0 < S \leq 1\}$, $\Gamma_2 = \bigcup_{y \in Y_2} \Gamma(y) = \{E^0\}$, and $\{E^0\}$ is an isolated compact invariant set in X. Furthermore, let $M = \{E^0\}$, thus, M is an acyclic isolated covering of Γ_2.

Now, we only need to show that $\{(\Lambda_h/\mu_h, 0, 0)\}$ is a weak repeller for X_1. Suppose that there exists a positive orbit (S_h, I_h, I_v) of (34) such that

$$\lim_{t \to +\infty} S_h(t) = \frac{\Lambda_h}{\mu_h}, \qquad \lim_{t \to +\infty} I_h(t) = 0, \qquad \lim_{t \to +\infty} I_v(t) = 0.$$
(44)

Since $\mathbb{R}_{\text{mass}} > 1$, there exists a small enough $\varepsilon > 0$, such that

$$b\Lambda_h \Lambda_v \beta_h \beta_v (1 - \varepsilon)^2 > (\mu_h + \gamma_h + \delta_h) \mu_h \mu_v^2.$$
(45)

From (34), we choose $t_0 > 0$ large enough such that when $t \geq t_0$, we have

$$I_h'(t) > b\beta_h (1 - \varepsilon) \frac{\Lambda_h}{\mu_h} I_v(t) - (\mu_h + \gamma_h + \delta_h) I_h(t),$$

$$I_v'(t) > b\beta_v (1 - \varepsilon) \frac{\Lambda_v}{\mu_v} I_h(t) - \mu_v I_v(t).$$
(46)

Consider the following matrix M_ε defined by

$$M_\varepsilon = \begin{pmatrix} -(\mu_h + \gamma_h + \delta_h) & b\beta_h (1 - \varepsilon) \frac{\Lambda_h}{\mu_h} \\ b\beta_v (1 - \varepsilon) \frac{\Lambda_v}{\mu_v} & \mu_v \end{pmatrix}.$$
(47)

Since M_ε admits positive off-diagonal element, the Perron-Frobenius Theorem [26] implies that there is a positive eigenvector $v = (v_1, v_2)$ for the maximum eigenvalue λ^* of M_ε. From (45), we see that the maximum eigenvalue λ^* is positive. Let us consider the following system:

$$u_1'(t) = b\beta_h (1 - \varepsilon) \frac{\Lambda_h}{\mu_h} u_2(t) - (\mu_h + \gamma_h + \delta_h) u_1(t),$$

$$u_2'(t) = b\beta_v (1 - \varepsilon) \frac{\Lambda_v}{\mu_v} u_1(t) - \mu_v u_2(t).$$
(48)

Let $u(t) = (u_1(t), u_2(t))$ be a solution of (48) through (lv_1, lv_2) at $t = t_0$, where $l > 0$ satisfies $lv_1 < I_h(t_0)$, $lv_2 < I_v(t_0)$. Since the semiflow of (48) 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 (48). Thus, E^0 is weak repeller for X_1. The proof is completed. □

From Theorem 10 and the boundedness of solutions, it follows that a compact set $M \subset \Gamma$ exists in system (34). Therefore, in Theorem 9, both assumptions (H_1) and (H_2) are satisfied for $\mathbb{R}_{\text{mass}} > 1$.

Now, we apply Theorem 9 to investigate the global stability of the endemic equilibrium E^*_{mass} in the feasible region Γ. For the global stability of the endemic equilibrium E^*_{mass}, we have the following theorem.

Theorem 11. *If $\mathbb{R}_{\text{mass}} > 1$, then the infective equilibrium E^*_{mass} of system* (48) *is globally asymptotically stable in* int Γ.

Proof. The Jacobian matrix J evaluated at a general solution (S_h, I_h, I_v) of system (34) is

$$J = \begin{pmatrix} -(\mu_h + b\beta_h I_v) & 0 & -b\beta_h S_h \\ b\beta_h (1 - \sigma) I_v & -(\mu_h + \gamma_h + \delta_h) - b\sigma\beta_h I_v & \begin{matrix} b\beta_h S_h (1 - \sigma) \\ +\sigma b\beta_h (N_h - I_h) \end{matrix} \\ 0 & b\beta_v (N_v - I_v) & -(b\beta_v I_h + \mu_v) \end{pmatrix},$$
(49)

and its corresponding second compound matrix $J^{[2]}$ takes the form

$$J^{[2]} = \begin{pmatrix} \begin{matrix} -(\mu_h + b\beta_h I_v + \mu_h \\ +\gamma_h + b\sigma\beta_h I_v) \end{matrix} & \begin{matrix} b\beta_h S_h (1 - \sigma) \\ +\sigma b\beta_h (N_h - I_h) \end{matrix} & b\beta_h S_h \\ b\beta_v (N_v - I_v) & \begin{matrix} -(\mu_h + b\beta_h I_v \\ +b\beta_v I_h + \mu_v) \end{matrix} & -\delta_h \\ 0 & b\beta_h (1 - \sigma) I_v & \begin{matrix} -(m + \mu_v + b\beta_v I_h \\ +b\sigma\beta_h I_v) \end{matrix} \end{pmatrix}.$$
(50)

Set the function $P(x) = P(S_h, I_h, I_v) = \text{diag}(1, I_h/I_v, I_h/I_v)$. Then $P_f P^{-1} = \text{diag}(0, I_h'/I_h - I_v'/I_v, I_h'/I_h - I_v'/I_v)$. Moreover,

$$B = P_f P^{-1} + P J^{[2]} P^{-1}$$

$$= \begin{pmatrix} -(\mu_h + b\beta_h I_v + \mu_h & (b\beta_h S_h (1-\sigma) & \\ \quad +\gamma_h + \delta_h + b\sigma\beta_h I_v) & \quad +\sigma b\beta_h (N_h - I_h)) \dfrac{I_v}{I_h} & b\beta_h S_h \dfrac{I_v}{I_h} \\ & \dfrac{I_h'}{I_h} - \dfrac{I_v'}{I_v} - (\mu_h + \mu_v & \\ b\beta_v (N_v - I_v) \dfrac{I_h}{I_v} & \quad + b\beta_h I_v & 0 \\ & \quad + b\beta_v I_h) & \\ & & \dfrac{I_h'}{I_h} - \dfrac{I_v'}{I_v} - \mu_h \\ 0 & b\beta_h (1-\sigma) I_v & \quad -\gamma_h - \delta_h - \mu_v \\ & & \quad -b\beta_v I_h - b\sigma\beta_h I_v \end{pmatrix}, \tag{51}$$

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

where $B_{11} = -(\mu_h + b\beta_h I_v + \mu_h + \gamma_h + \delta_h + b\sigma\beta_h I_v)$, $B_{12} = [(b\beta_h S_h + \sigma b\beta_h (N_h - S_h - I_h))I_v/I_h, b\beta_h S_h I_v/I_h]$, $B_{21} = [b\beta_v (N_v - I_v)(I_h/I_v), 0]^T$, and

$$B_{22}$$

$$= \begin{pmatrix} \dfrac{I_h'}{I_h} - \dfrac{I_v'}{I_v} - (\mu_h + \delta_h + \mu_v) & 0 \\ \quad -b\beta_h I_v - b\beta_v I_h & \\ b\beta_h I_v (1-\sigma), & \dfrac{I_h'}{I_h} - \dfrac{I_v'}{I_v} - (+\mu_h + \gamma_h + \delta_h + \mu_v) \\ & \quad -b\theta\beta_h I_v - b\beta_v I_h \end{pmatrix}. \tag{52}$$

Let (u, v, w) be the vectors in R^3. We choose a norm in R^3 as $|(u, v, w)| = \max\{|u|, |v| + |w|\}$, and let μ be the corresponding Lozinskiĭ measure. From the paper [28], we have the following estimate:

$$\mu(B) \le \sup\{g_1, g_2\}, \tag{53}$$

where

$$g_1 = \mu_1(B_{11}) + |B_{12}|, \qquad g_2 = \mu_1(B_{22}) + |B_{21}|. \tag{54}$$

Here, $|B_{12}|$, $|B_{21}|$ are matrix norm with respect to the l_1 vector norm, and μ_1 is the Lozinskiĭ measure with respect to l_1 norm. Thus, we have

$$\mu_1(B_{11}) = -(\mu_h + b\beta_h I_v + \mu_h + \gamma_h + \delta_h + b\sigma\theta\beta_h I_v),$$

$$|B_{12}| = (b\beta_h S_h + \sigma b\beta_h (N_h - S_h - I_h)) \frac{I_v}{I_h}, \tag{55}$$

$$|B_{21}| = b\beta_v (N_v - I_v) \frac{I_h}{I_v}.$$

According to paper [28], $\mu_1(B_{22})$ can be evaluated as follows:

$$\mu_1(B_{22}) = \max\left\{ \frac{I_h'}{I_h} - \frac{I_v'}{I_v} - (\mu_h + \mu_v + b\beta_v I_h + \sigma b\beta_h I_v), \right.$$

$$\frac{I_h'}{I_h} - \frac{I_v'}{I_v} - b\sigma\beta_h I_v - b\beta_v I_h$$

$$\left. -\mu_h - \delta_h - \gamma_h - \mu_v \right\},$$

$$= \frac{I_h'}{I_h} - \frac{I_v'}{I_v} - (\mu_h + \mu_v + b\beta_v I_h + \sigma b\beta_h I_v). \tag{56}$$

Thus,

$$g_1 = -(\mu_h + b\beta_h I_v + \mu_h + \delta_h + \gamma_h + b\sigma\beta_h I_v)$$

$$+ (b\beta_h S_h + \sigma b\beta_h (N_h - S_h - I_h)) \frac{I_v}{I_h},$$

$$g_2 = b\beta_v (N_v - I_v) \frac{I_h}{I_v} + \frac{I_h'}{I_h} - \frac{I_v'}{I_v}$$

$$- (\mu_h + \mu_v + b\beta_v I_h + \sigma b\beta_h I_v). \tag{57}$$

From system (34), we have

$$\frac{I_h'}{I_h} = (b\beta_h S_h + \sigma b\beta_h (N_h - S_h - I_h)) \frac{I_v}{I_h} - (\mu_h + \delta_h + \gamma_h),$$

$$\frac{I_v'}{I_v} = b\beta_v (N_v - I_V) \frac{I_h}{I_v} - \mu_v. \tag{58}$$
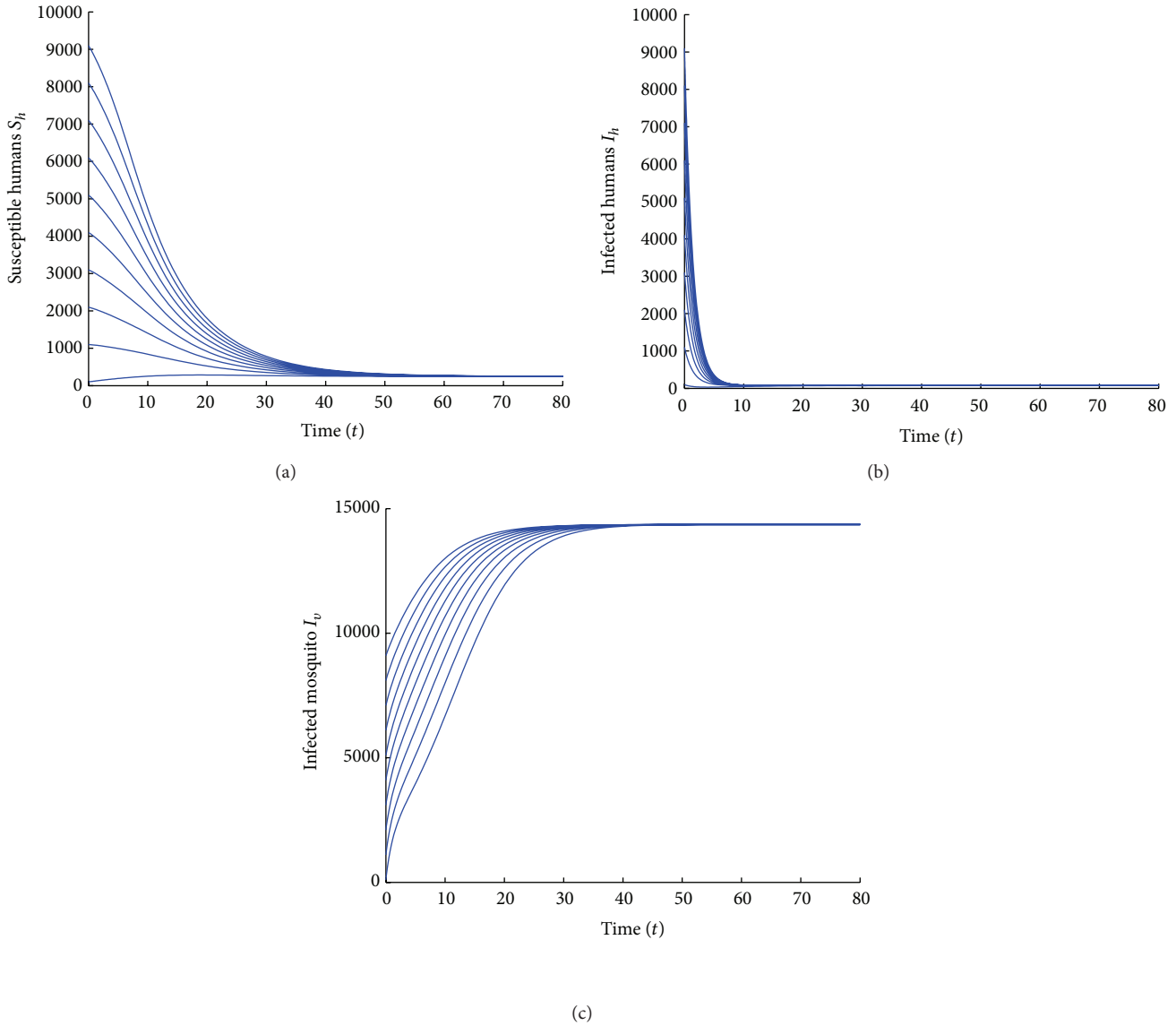
(a)

(b)

(c)

FIGURE 6: Simulations of the model (5) showing (a) the number of susceptible humans, (b) the number of infected humans, (c) the number of infected mosquitoes, as a function of time using the parameters: $b = 0.5$, $\Lambda_h = 25$, $\Lambda_v = 500$, $\beta_1 = 0.00001$, $\beta_2 = 0.005$, $\mu_h = 0.03$, $\mu_v = 0.03$, $\sigma = 0.8$, $\delta_h = 0.0001$, and $\gamma_h = 0.6$ (so that $\mathbb{R}_0 = 9.1843 > 1$).

Using (58) in (57) gives

$$g_1 = \frac{I_h'}{I_h} - \left(\mu_h + b\beta_h I_v + b\sigma\beta_h I_v\right),$$

$$g_2 = \frac{I_h'}{I_h} - \left(\mu_h + b\beta_v I_h + \sigma b\beta_h I_v\right). \tag{59}$$

From Theorem 9 we know that for the uniform persistence constant $\varepsilon > 0$, there exists a time $T > 0$ independent of $x(0) \in \Gamma$, the compact absorbing set, such that

$$I_h(t) > \varepsilon, \qquad I_v(t) > \varepsilon \tag{60}$$

for $t > T$. Thus, from (59) and (60), we get

$$g_1 \le \frac{I_h'}{I_h} - \left(\mu_h + b\theta\beta_h\varepsilon + b\sigma\theta\beta_h\varepsilon\right),$$

$$g_2 \le \frac{I_h'}{I_h} - \left(\mu_h + b\beta_v\varepsilon + \sigma b\beta_h\varepsilon\right). \tag{61}$$

Therefore, from (61), we have $\mu(B) \le (I_h'/I_h) - \eta$ for $t > T$, where

$$\eta = \min\left\{\mu_h + b\beta_h\varepsilon + b\sigma\theta\beta_h\varepsilon, \mu_h + b\beta_v\varepsilon + \sigma b\beta_h\varepsilon\right\}. \tag{62}$$

Then along each solution $(S_h(t), I_h(t), I_v(t))$ such that $(S_h(0), I_h(0), I_v(0)) \in \Gamma$ for $t > T$, give the following:

$$\frac{1}{t}\int_0^t \mu(B)\,ds \le \frac{1}{t}\int_0^T \mu(B)\,ds + \frac{1}{t}\ln\frac{I_h(t)}{I_v(t)} - \eta\frac{t-T}{t}, \quad (63)$$

which implies that $\overline{q}_2 \le -\eta/2 < 0$. This proves that the unique infective equilibrium E^*_{mass} is globally asymptotically stable whenever it exists.

This completes the proof. □

Remark 12. The epidemiological implication of Theorem 11 is that malaria would persist in the population if $R_{\mathrm{mass}} > 1$. Theorem 11 is illustrated numerically by simulating the model (25), for the case $R_{\mathrm{mass}} > 1$ using various initial conditions. The convergence of the solutions to E^*_{mass} for the case $R_{\mathrm{mass}} > 1$, is depicted in Figure 6.

4. Conclusions

This paper presents a deterministic model for the transmission dynamics of malaria with partial immunity to reinfection. The basic reproduction number of the model is obtained. The proposed model with standard incidence rate, undergoes the phenomenon of backward bifurcation, where the stable disease-free equilibrium coexists with one or more stable endemic equilibrium as the basic reproduction number ($\mathbb{R}_0$) is less than unity. In comparison with the corresponding results of the model with mass action incidence, we can conclude that this phenomenon arises due to the use of standard incidence rate. This study suggests that in some regions where malaria is inducing the varying total populations, it is difficult to control malaria due to the occurrence of backward bifurcation phenomenon. If we ignore the disease-induced rate, and consider an asymptotical constant host population, the standard incidence model results in a model with mass action incidence. In this case, the dynamics of the model are relatively simple. That is, the global dynamics of malaria disease with reinfection is completely determined by the associated reproduction number $\mathbb{R}_{\mathrm{mass}}$. If $\mathbb{R}_{\mathrm{mass}} < 1$, the disease-free equilibrium is globally *asymptotically* stable, so the disease always dies out. If $\mathbb{R}_{\mathrm{mass}} > 1$, the disease-free equilibrium becomes unstable while the endemic equilibrium emerges as the unique positive equilibrium and it is globally *asymptotically* stable in the interior of the feasible region, and once the disease appears, it eventually persists at the unique endemic equilibrium level. Therefore, we have shown that the backward bifurcation property can be removed by replacing the standard incidence function in the model with a mass action incidence. The numerical simulations suggest also that the region of backward bifurcation increases with increasing rate of partial protection (σ) of recovered individuals. The region of backward bifurcation for the model increases with decreasing average life span of mosquitoes.
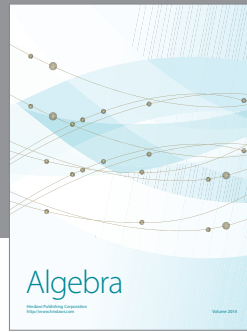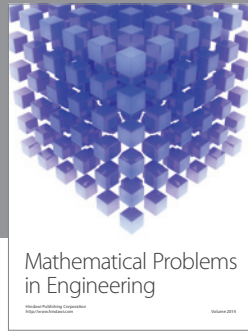
Acknowledgments

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