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Research Article

Global Stability of Malaria Transmission Dynamics Model with Logistic Growth

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Mathematical models become an important and popular tools to understand the dynamics of the disease and give an insight to reduce the impact of malaria burden within the community. Thus, this paper aims to apply a mathematical model to study global stability of malaria transmission dynamics model with logistic growth. Analysis of the model applies scaling and sensitivity analysis and sensitivity analysis of the model applied to understand the important parameters in transmission and prevalence of malaria disease. We derive the equilibrium points of the model and investigated their stabilities. The results of our analysis have shown that if $R_0 \leq 1$, then the disease-free equilibrium is globally asymptotically stable, and the disease dies out; if $R_0 > 1$, then the unique endemic equilibrium point is globally asymptotically stable and the disease persists within the population. Furthermore, numerical simulations in the application of the model showed the abrupt and periodic variations.

1. Introduction

Malaria is a mosquito-borne disease caused by Plasmodium parasite, which is transmitted through the bites of an infected mosquito. In 2017, the World Health Organization report reveals estimations of 216 million malaria cases and 445 thousand deaths due to malaria were registered worldwide in 2016. However, the most malaria cases and deaths were shared by the WHO Africa region, which account for 90% of cases and 91% deaths. The most predominant malaria parasite in the WHO Africa region is *Plasmodium falciparum*, accounting for 99% of malaria cases in 2016 [1].

Malaria is entirely preventable and treatable disease if the recommended interventions are properly applied. Individuals should have taken some aggressive measurements to decline malaria burden. Personal protection measures are the first line of defense against mosquito-borne diseases. Mosquito repellent is a method used for personal protection; and these are the substances used for exposed skin to prevent human-mosquito contact. Insecticide Treated Bed Nets (ITNs) are used for individuals against malaria to reduce the morbidity of childhood malaria (below five years of age) by 50% and global child mortality by 20–30% [2, 3]. When used on a large scale, ITNs are supposed to represent efficient tools for malaria vector control but there is a limitation of resistance to insecticides used for a saturated net. The resistance of the most important African malaria *Anopheles gambiae* to protrude is already widespread in several West African countries [4, 5].

Nowadays, mathematical models become an important and popular tools to understand the transmission dynamics of the disease and give an insight to reduce the impact of malaria burden in the society. This is because mathematical modeling can answer the following questions raised by the public health authorities and policy makers to make the correct decisions: (1) how severe will the epidemics be? (2) How long will it last? (3) How effective will an intervention be? (4) What are the effective measures to control and eliminate an endemic disease? The earliest malaria model study originated from Ross in 1911 [6] and later modification made by Macdonald [7]. Some further extensions of Ross-Macdonald models for malaria were described in [8–13]. Tumwiine et al. [13] define the reproduction number, R_0, and show the existence and stability of the disease-free equilibrium and an endemic equilibrium.

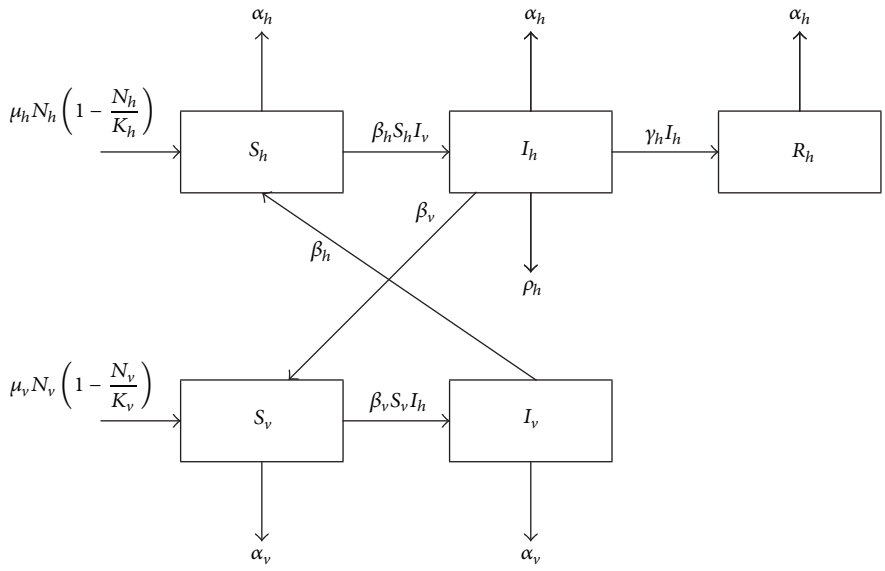

Figure 1: The compartmental model for malaria transmission.

Recently, many works on host-vector interaction models have been done in [14–24]. In [18, 20, 22, 25], global stability of equilibria has been investigated using suitable Lyapunov functions; and their results show that the disease-free and endemic equilibrium points become globally asymptotically stable if $R_0 \leq 1$ and $R_0 > 1$, respectively. Application of the optimal control theory becomes an important tool for investigating the efficiency of joint control intervention strategies to minimize the impact of malaria disease and cost-effectiveness of implementing them [19, 21, 23, 24]. Their studies suggest that the optimal control strategies can effectively reduce the malaria disease.

Motivated by the above studies, we extend the model presented in [14] by taking into account a logistic model with population dependent birth rates for both human and vector populations that describes self-limiting growth of both the human host and mosquito vector populations. We consider logistic malaria model as no population can grow exponentially at all time, in general. A number of populations initially grow exponentially, but, due to competition and limited resources availability, their population size decline, after some time, to a stable size K, called the maximum *carrying capacity*. The competition for limited resources (including food, territory, light, water, and oxygen) decreases the fertility or survival of individuals. Furthermore, this paper presents application of the model to study abrupt and periodic variations of malaria and sensitivity analysis applied to understand the important parameters in transmission and prevalence of the malaria disease. The purpose of this work is to investigate the global stability of both disease-free and endemic equilibrium points.

2. Malaria Model

We consider that the total human host population, N_h, at a time t^*, is divided into three disjoint compartments: susceptible S_h, infectious I_h, and recovered R_h. The total vector population, N_v, at a time t^*, is divided into two mutually exclusive subpopulations of individuals who are susceptible, S_v, and infectious, I_v. The susceptible human and vector populations are recruited at the rates B_h and B_v, respectively, where $B_h = \mu_h N_h(1 - N_h/K_h)$ and $B_v = \mu_v N_v(1 - N_v/K_v)$. The susceptible human and vector populations decrease due to natural death at a rate α_h for humans and α_v for vectors, and those that move to the infected classes at a rate $\beta_h I_v$ and $\beta_v I_h$, respectively. The infected human population grows as a result of new infection at a rate $\beta_h S_h I_v$ and decline due to natural mortality, disease induced death, and recovery at a rate α_h, ρ_h, and γ_h, respectively. For details, see the schematic diagram of the model in Figure 1. The state variables and parameters for the model are described in "State Variables, Parameters, Descriptions, and Their Dimensions of Malaria Model" section.

The model has made the following assumptions: both the total sizes of human and vector populations not being constant; all variables and parameters involving the model assumed to be nonnegative; all newborns susceptible to infection; mosquitoes not dying because of infection; no recovery compartment for infected mosquitoes; and the recovered human population developing permanent immunity. From the schematics diagram of transmission of malaria between human and mosquito (see Figure 1), we have the governed differential equations which describe the dynamics of malaria,

$$\frac{dS_h}{dt^*} = \mu_h N_h\left(1-\frac{N_h}{K_h}\right) - \beta_h S_h I_v - \alpha_h S_h,$$

$$\frac{dI_h}{dt^*} = \beta_h S_h I_v - \alpha_h I_h - \rho_h I_h - \gamma_h I_h,$$

$$\frac{dR_h}{dt^*} = \gamma_h I_h - \alpha_h R_h,$$

$$\frac{dS_v}{dt^*} = \mu_v N_v \left(1 - \frac{N_v}{K_v}\right) - \beta_v S_v I_h - \alpha_v S_v,$$
$$\frac{dI_v}{dt^*} = \beta_v S_v I_h - \alpha_v I_v, \tag{1}$$

with initial conditions

$$S_h(0) = S_{h0},$$
$$I_h(0) = I_{h0},$$
$$R_h(0) = R_{h0}, \tag{2}$$
$$S_v(0) = S_{v0},$$
$$I_v(0) = I_{v0}.$$

At all times, $N_h = S_h + I_h + R_h$ and $N_v = S_v + I_v$. Moreover, their differential equations are satisfying

$$\frac{dN_h}{dt^*} = \mu_h N_h \left(1 - \frac{N_h}{K_h}\right) - \alpha_h N_h - \rho_h I_h,$$
$$\frac{dN_v}{dt^*} = \mu_v N_v \left(1 - \frac{N_v}{K_v}\right) - \alpha_v N_v, \tag{3}$$

respectively. We may notice that the vector population equation is completely decoupled from the human equations which is physically reasonable.

If we eliminate R_h and S_v and add the total population equations, then we finally have

$$\frac{dS_h}{dt^*} = \mu_h N_h \left(1 - \frac{N_h}{K_h}\right) - \beta_h S_h I_v - \alpha_h S_h,$$
$$\frac{dI_h}{dt^*} = \beta_h S_h I_v - \alpha_h I_h - \rho_h I_h - \gamma_h I_h,$$
$$\frac{dI_v}{dt^*} = \beta_v (N_v - I_v) I_h - \alpha_v I_v, \tag{4}$$
$$\frac{dN_h}{dt^*} = \mu_h N_h \left(1 - \frac{N_h}{K_h}\right) - \alpha_h N_h - \rho_h I_h,$$
$$\frac{dN_v}{dt^*} = \mu_v N_v \left(1 - \frac{N_v}{K_v}\right) - \alpha_v N_v.$$

2.1. Basic Properties. Since the model system (1) involves human and mosquito populations, all its associated variables and parameters are nonnegative.

Theorem 1. *Solutions of the model system (1) with positive initial data will remain nonnegative for all time $t \geq 0$.*

Proof. Let $\Omega = \{(S_h, I_h, R_h, S_v, I_v \in R^5 : S_{h0} > 0,\ I_{h0} > 0,\ R_{h0} > 0,\ I_{v0} > 0,\ S_{v0} > 0)\}$. Then it follows from the second equation of malaria model (1) that

$$\frac{dS_h}{dt} = \mu_h N_h \left(1 - \frac{N_h}{K_h}\right) - S_h \beta_h I_v - \alpha_h S_h \geq -S_h (\beta_h I_v + \alpha_h), \tag{5}$$

so that

$$\frac{dS_h}{S_h} \geq -(\beta_h I_v + \alpha_h)\, dt. \tag{6}$$

Hence,

$$S_h(t) \geq S_{h0} \exp(-\beta_h I_v - \alpha_h)\, t \geq 0. \tag{7}$$

Similarly, it can be shown that $I_h \geq 0$, $R_h \geq 0$, $S_v \geq 0$, and $I_v \geq 0$ for all time $t \geq 0$.

This completes the proof. □

2.1.1. Invariant Region. The malaria model (1) will be analyzed in biologically feasible region. Thus, the feasible solutions set for the model written by

$$\Omega = \left\{(S_h, I_h, R_h, S_v, I_v) \in R_+^5 : S_h + I_h + R_h \leq \frac{K_h(\mu_h - \alpha_h)}{\mu_h},\ S_v + I_v \leq \frac{K_v(\mu_v - \alpha_v)}{\mu_v}\right\} \tag{8}$$

is positively invariant and then the model is biologically meaningful and mathematically well posed in the domain Ω. The proof is omitted for simplicity.

3. Model Analysis

To analyze the malaria model in system (4), we use the normalized quantities instead of the actual populations. Since N_h and N_v may vary, these scales are not suitable for use in the scaling. However, the typical choice for logistic models is to use the sustainable populations K_h and K_v for the scales. In the present case, we shall also consider varying K_h and K_v. It is, therefore, convenient to write $K_h = K_h^0 g_h(t)$ and $K_v = K_v^0 g_v(t)$, where K_h^0 and K_v^0 are typical sizes and where g_h and g_v take care of the time variations. At the moment, we just assume that $g_h(t) = g_v(t) = 1$. We shall scale the time t^* with the quantity $1/\mu_h$ by setting $t = \mu_h t^*$. The scaling is then $S_h = K_h^0 s_h$, $I_h = K_h^0 i_h$, $R_h = K_h^0 r_h$, $N_h = K_h^0 n_h$, $S_v = K_v^0 s_v$, $I_v = K_v^0 i_v$, $N_v = K_v^0 n_v$, and $t^* = (1/\mu_h)t$. The dimensionless parameters of the model become $\beta = \beta_h N_v/\mu_h$, $\alpha = \alpha_h/\mu_h$, $\gamma = (\rho_h + \gamma_h)/\mu_h$, $\nu = \beta_v N_h/\mu_v$, $\delta = \alpha_v/\mu_v$, and $\varepsilon = \mu_h/\mu_v \ll 1$.

The scaled equations then become

$$\frac{dn_h}{dt} = n_h(1 - n_h) - \alpha n_h - \rho i_h,$$
$$\frac{ds_h}{dt} = n_h(1 - n_h) - \beta s_h i_v - \alpha s_h,$$
$$\frac{di_h}{dt} = \beta s_h i_v - (\alpha + \rho + \gamma) i_h, \tag{9}$$
$$\varepsilon \frac{dn_v}{dt} = n_v(1 - n_v) - \delta n_v,$$
$$\varepsilon \frac{di_v}{dt} = \nu(n_v - i_v) i_h - \delta i_v,$$

subject to suitable initial conditions,

$$\begin{aligned} n_h(0) &= n_{h0}, \\ i_h(0) &= i_{h0}, \\ s_h(0) &= s_{h0}, \\ n_v(0) &= n_{v0}, \\ i_v(0) &= i_{v0}. \end{aligned} \tag{10}$$

3.1. Stability of Disease-Free Equilibrium and Reproduction Number. Our model (9) admits two disease-free equilibria, namely, $E_0 = (0, 0, 0, 0, 0)$ and $E_1 = (1-\alpha, 1-\alpha, 0, 1-\delta, 0)$, where α and δ lie between 0 and 1, and a unique endemic equilibrium, $E_2 = (n_{h*}, s_{h*}, i_{h*}, n_{v*}, i_{v*})$. The equilibria of the system (9) are obtained by setting the right side equal to zero.

The basic reproduction number, R_0, is the single most important parameter in epidemiological modeling. It measures the average number of the secondary infections caused by a single infective in an entirely susceptible population during its whole infectious period [26]. To derive the basic reproduction number R_0 of model (9), we use the next generation matrix approach described in [27–29]. The infected compartments of system (9) are i_h and i_v. Following [29], the new infection matrix F and the transition matrix V are given, respectively, by

$$\begin{aligned} F &= \begin{bmatrix} 0 & \beta(1-\alpha) \\ \nu(1-\delta) & 0 \end{bmatrix}, \\ V &= \begin{bmatrix} \alpha+\rho+\gamma & 0 \\ 0 & \delta \end{bmatrix}. \end{aligned} \tag{11}$$

Hence, the basic reproduction number, R_0, is the dominant eigenvalue of the next generation matrix FV^{-1} and becomes

$$\begin{aligned} R_0 &= \rho\left(FV^{-1}\right), \\ &= \sqrt{\frac{\beta(1-\alpha)}{(\alpha+\rho+\gamma)}\frac{\nu(1-\delta)}{\delta}} \\ &= \sqrt{R_{0vh} \times R_{0hv}}. \end{aligned} \tag{12}$$

From (12), it is noted that the reproduction number depends on the product of the number of humans that one mosquito infects through its infectious lifetime, R_{0vh}, and the number of mosquitoes that one human infects through its infectious lifetime, R_{0hv}.

Theorem 2. *The disease-free equilibrium point, E_0, is locally asymptotically stable if all eigenvalues of the characteristic equation of the variational matrix lie below zero.*

Proof. At the equilibrium point, E_0, the variational matrix is given by

$$J(E_0) = \begin{bmatrix} 1-\alpha & 0 & -\rho & 0 & 0 \\ 1 & -\alpha & 0 & 0 & 0 \\ 0 & 0 & -(\alpha+\rho+\gamma) & 0 & 0 \\ 0 & 0 & 0 & 1-\delta & 0 \\ 0 & 0 & 0 & 0 & -\delta \end{bmatrix}. \tag{13}$$

The characteristic equation may be written as $\det[J(E_0) - \lambda I] = 0$. It implies

$$\begin{aligned} &(1-\alpha-\lambda)(-\alpha-\lambda)(-\alpha-\rho-\gamma-\lambda)(1-\delta-\lambda) \\ &\quad \cdot (-\delta-\lambda) = 0. \end{aligned} \tag{14}$$

Clearly, we have

$$\begin{aligned} \lambda_1 &= 1-\alpha > 0, \quad \text{as } \alpha < 1, \\ \lambda_2 &= -\alpha < 0, \\ \lambda_3 &= -(\alpha+\rho+\gamma) < 0, \\ \lambda_4 &= 1-\delta > 0 \quad \text{as } \delta < 1, \\ \lambda_5 &= -\delta < 0. \end{aligned} \tag{15}$$

Thus, this shows that the solution, E_0, is unstable since λ_1 and λ_4 lie above zero. □

Theorem 3. *The disease-free equilibrium point, E_1, is locally asymptotically stable if $R_0 < 1$.*

Proof. The variational matrix at the equilibrium point, E_1, becomes

$$\begin{aligned} &J(E_1) \\ &= \begin{bmatrix} \alpha-1 & 0 & -\rho & 0 & 0 \\ 2\alpha-1 & -\alpha & 0 & 0 & \beta(\alpha-1) \\ 0 & 0 & -(\alpha+\rho+\gamma) & 0 & \beta(1-\alpha) \\ 0 & 0 & 0 & \delta-1 & 0 \\ 0 & 0 & \nu(1-\delta) & 0 & -\delta \end{bmatrix}. \end{aligned} \tag{16}$$

Thus, the characteristic equation of the variational matrix is given by

$$(\alpha-1-\lambda)(-\alpha-\lambda)(\delta-1-\lambda)\left(\lambda^2 + a_1\lambda + a_0\right) = 0, \tag{17}$$

where

$$\begin{aligned} a_0 &= \delta(\alpha+\rho+\gamma)\left(1-R_0^2\right), \\ a_1 &= \delta+\alpha+\gamma+\rho. \end{aligned} \tag{18}$$

The characteristic polynomial in (17) has roots $\lambda_1 = \alpha - 1$, $\lambda_2 = \delta - 1$, $\lambda_3 = -\alpha$, with negative real parts since $0 < \alpha, \delta < 1$. By Routh-Hurwitz criterion [28], the other roots λ_4 and

λ_5 have negative real parts if both a_0 and a_1 lie above zero. From the second equation of (18), $a_1 > 0$, and from the first equation of (18), $a_0 > 0$, when $R_0 < 1$. Hence, the disease-free equilibrium point, E_1, is locally asymptotically stable. □

Theorem 4. *The disease-free equilibrium point, E_1, is globally asymptotically stable in Ω if $R_0 \leq 1$; otherwise it is unstable.*

Proof. Consider the Lyapunov function

$$V(i_h, i_v) = ai_h + bi_v, \tag{19}$$

where

$$a = \nu \frac{1-\delta}{\delta(\alpha+\gamma+\rho)}, \qquad b = \frac{\varepsilon}{\delta}. \tag{20}$$

The time derivative of the function V along the solutions of (9) becomes

$$\begin{aligned}
\dot{V}(i_h, i_v) &= a\frac{di_h}{dt} + b\frac{di_v}{dt} \\
&= a\left(\beta s_h i_v - (\alpha+\gamma+\rho) i_h\right) \\
&\quad + b\left(\frac{\nu(n_v - i_v) i_h - \delta i_v}{\varepsilon}\right) \\
&= \left(a\beta(1-\alpha) - \frac{b\delta}{\varepsilon}\right) i_v \\
&\quad + \left(\frac{b\nu}{\varepsilon}(1-\delta-i_v) - a(\alpha+\gamma+\rho)\right) i_h \\
&= \left(\frac{\beta\nu(1-\alpha)(1-\delta)}{\delta(\alpha+\gamma+\rho)} - 1\right) i_v - \frac{\nu}{\delta} i_h i_v \\
&= \left(R_0^2 - 1\right) i_v - \frac{\nu}{\delta} i_h i_v \leq 0.
\end{aligned} \tag{21}$$

Thus $\dot{V} \leq 0$ if $R_0 \leq 1$ and the equality $\dot{V} = 0$ holds if and only if $i_h = i_v = 0$. Therefore, the largest compact invariant set in $\{(i_h, i_v) \in \Omega : \dot{V} = 0\}$ is the singleton $\{E_1\}$, where E_1 is the disease-free equilibrium. LaSalle's Invariant Principle [30] implies that E_1 is globally asymptotically stable in Ω. □

3.2. Stability of Endemic Equilibrium. To find the endemic equilibrium E_2, we shall keep the assumptions about n_{v*} and i_{v*} from the singular perturbation equations (see the fourth and fifth equations of system (9)) and then focus on the first three equations of system (9). We consider the equilibrium solutions, using i_{h*} as the basic quantity. From the third equation of (9), we have an expression for s_{h*}:

$$s_{h*} = \frac{\alpha+\gamma+\rho}{\beta\nu(1-\delta)}\left(\delta + \nu i_{h*}\right). \tag{22}$$

Addition of the second and third equations of system (9) gives

$$n_{h*} - \frac{\gamma}{\alpha} i_{h*} - i_{h*} - s_{h*} = 0. \tag{23}$$

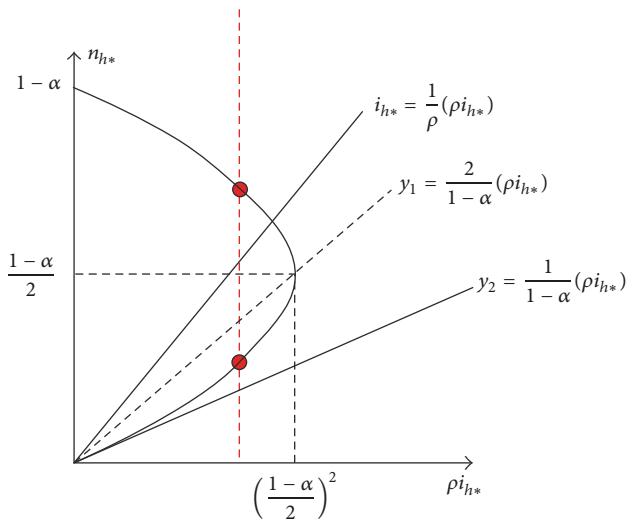

Figure 2: Graph illustrating the behaviour of the solutions of the equation between n_{h*} and i_{h*}.

Now, let us consider the first equation of (9) which connects n_{h*} and i_{h*}. That is,

$$n_{h*}(1 - n_{h*}) - \alpha n_{h*} - \rho i_{h*} = 0. \tag{24}$$

Equation (24) has only real and positive solutions for n_{h*} if $\rho i_{h*} < ((1-\alpha)/2)^2$, since the maximum value of $n_{h*}(1-n_{h*}) - \alpha n_{h*}$ is $(1-\alpha)^2/4$. If $\rho i_{h*} < (1-\alpha)^2/4$, then (24) has two solutions:

$$n_{h*}^+, n_{h*}^- = \frac{1-\alpha}{2} \pm \sqrt{\frac{(1-\alpha)^2}{4} - \rho i_{h*}}, \tag{25}$$

for each value of ρi_{h*}.

One might wonder what happens when the inequality is violated and $\rho i_{h*} > ((1-\alpha)/2)^2$. The solution of (24) will then be majorized by the equation

$$\frac{dy}{dt} = y(1-y) - \alpha y - \left(\frac{1-\alpha}{2}\right)^2 \quad \text{when } \rho i_{h*} = \left(\frac{1-\alpha}{2}\right)^2, \tag{26}$$

which has only one unstable equilibrium point at $y = ((1-\alpha)/2)$ and otherwise tends to 0. Moreover, it is clear that n_{h*} needs to be larger or equal to i_{h*}. Let us, therefore, consider n_{h*}^+ and n_{h*}^- in the light of this restriction. The various situations are best described in terms of the graph shown in Figure 2.

The parabola is the locus of the solutions for n_{h*}^+ and n_{h*}^- when $0 \leq \rho i_{h*} \leq (1-\alpha)^2/4$. The line, $i_{h*} = (1/\rho)(\rho i_{h*})$, defines the limit of the region for solutions fulfilling $i_{h*} \leq n_{h*}$. In the graph, the upper solution (upper red dot) is acceptable since $n_{h*}^+ > i_{h*}$, whereas the lower solution is outside the region and hence unacceptable. Further inspection of the graph shows the following.

Table 1: Number of possible positive real roots of (27) for $R_0 < 1$ and $R_0 > 1$.

Cases	a	b	c	R_0	Number of sign changes	Number of possible positive real roots
1	+	+	+	<1	0	0
2	+	+	−	>1	1	1
3	+	−	−	>1	1	1

(1) If $1 - \alpha \leq \rho$, both solutions, n_{h*}^{-} and n_{h*}^{+}, are larger than i_{h*} (consider the line y_2).

(2) If $\rho < (1-\alpha)/2$, only n_{h*}^{+} is larger than i_{h*} when i_{h*} is positive (consider the line y_1).

(3) For $(1-\alpha)/2 < \rho < 1-\alpha$, one or both of the solutions are acceptable.

The main conclusion is that there are acceptable solutions with respect to size for all i_{h*} where $0 \leq \rho i_{h*} \leq (1-\alpha)^2/4$.

Assume that $\rho i_{h*} < (1-\alpha)^2/4$, where only n_{h*}^{+} is acceptable with respect to size. Then substituting expressions (22) and (25) into the equation in (23) and simplifying lead to the following quadratic equation:

$$a i_{h*}^2 + b i_{h*} + c = 0, \tag{27}$$

where

$$\begin{aligned} a &= \left(1 + \frac{\gamma}{\alpha} + \frac{\alpha+\gamma+\rho}{\beta(1-\delta)}\right)^2, \\ b &= \left(1 + \frac{\gamma}{\alpha} + \frac{\alpha+\gamma+\rho}{\beta(1-\delta)}\right)\left(\frac{\delta(\alpha+\gamma+\rho)}{\beta\nu(1-\delta)}\left(2 - R_0^2\right)\right) \\ &\quad + \rho, \\ c &= \left(\frac{\delta(\alpha+\gamma+\rho)}{\beta\nu(1-\delta)}\right)^2 (1 - R_0) + \left(\frac{1-\alpha}{2}\right)^2. \end{aligned} \tag{28}$$

From (27), it can easily be seen that $a > 0$. Further, if $R_0 > 1$, then $c < 0$. Thus, the number of possible positive real roots of (27) can depend on the signs of b. This can be analyzed using the Descartes rule of signs on the quadratic polynomial (27). The different possibilities for the roots of (27) are tabulated in Table 1.

Thus, the malaria model has a unique endemic equilibrium if $R_0 > 1$ and whenever cases 2 and 3 are satisfied. Hence, the endemic equilibrium then becomes

$$\begin{aligned} n_{h*} &= \left(1 + \frac{\gamma}{\alpha} + \frac{\alpha+\gamma+\rho}{\beta(1-\delta)}\right) i_{h*} + \frac{\delta(\alpha+\gamma+\rho)}{\beta\nu(1-\delta)}, \\ s_{h*} &= \frac{\delta(\alpha+\gamma+\rho)}{\beta\nu(1-\delta)} + \frac{\alpha+\gamma+\rho}{\beta(1-\delta)} i_{h*}, \\ n_{v*} &= 1 - \delta, \\ i_{v*} &= (1-\delta)\frac{\nu i_{h*}}{\delta + \nu i_{h*}}, \end{aligned} \tag{29}$$

and i_{h*} is the unique positive root of (27).

Theorem 5. *If $R_0 > 1$, then the endemic equilibrium E_2 of the malaria model (9) is globally asymptotically stable in Ω.*

Proof. We shall propose the Lyapunov function

$$\begin{aligned} V(n_h, s_h, i_h, n_v, i_v) &= a_1\left(n_h - n_{h*} - n_{h*}\ln\frac{n_h}{n_{h*}}\right) \\ &\quad + a_2\left(s_h - s_{h*} - s_{h*}\ln\frac{s_h}{s_{h*}}\right) \\ &\quad + a_3\left(i_h - i_{h*} - i_{h*}\ln\frac{i_h}{i_{h*}}\right) \\ &\quad + a_4\left(n_v - n_{v*} - n_{v*}\ln\frac{n_v}{n_{v*}}\right) \\ &\quad + a_5\left(i_v - i_{v*} - i_{v*}\ln\frac{i_v}{i_{v*}}\right), \end{aligned} \tag{30}$$

where

$$\begin{aligned} a_1 &= a_2 = a_3 = a_4 = 1, \\ a_5 &= \frac{\varepsilon}{\nu}. \end{aligned} \tag{31}$$

The Lyapunov function V is continuous for all $n_h, s_h, i_h, n_v, i_v > 0$. The time derivative of the function V along the solutions of system (9) becomes

$$\begin{aligned} \frac{dV}{dt} &= a_1\left(1 - \frac{n_{h*}}{n_h}\right)\frac{dn_h}{dt} + a_2\left(1 - \frac{s_{h*}}{s_h}\right)\frac{ds_h}{dt} \\ &\quad + a_3\left(1 - \frac{i_{h*}}{i_h}\right)\frac{di_h}{dt} + a_4\left(1 - \frac{n_{v*}}{n_v}\right)\frac{dn_v}{dt} \\ &\quad + a_5\left(1 - \frac{i_{v*}}{i_v}\right)\frac{di_v}{dt} \\ &= a_1\left(1 - \frac{n_{h*}}{n_h}\right)\left(n_h(1-n_h) - \alpha n_h - \rho i_h\right) \\ &\quad + a_2\left(1 - \frac{s_{h*}}{s_h}\right)\left(n_h(1-n_h) - \beta s_h i_v - \alpha s_h\right) \\ &\quad + a_3\left(1 - \frac{i_{h*}}{i_h}\right)\left(\beta s_h i_v - (\alpha+\gamma+\rho) i_h\right) \\ &\quad + a_4\left(1 - \frac{n_{v*}}{n_v}\right)\left(\frac{n_v(1-n_v) - \delta n_v}{\varepsilon}\right) \\ &\quad + a_5\left(1 - \frac{i_{v*}}{i_v}\right)\left(\frac{\nu(n_v - i_v) i_h - \delta i_v}{\varepsilon}\right). \end{aligned} \tag{32}$$

From the equilibrium point of the malaria model (9), we have the following relations:

$$\begin{aligned} \alpha &= \frac{n_{h*}(1-n_{h*}) + \rho i_{h*}}{n_{h*}}, \\ \alpha + \gamma + \rho &= \frac{\beta s_{h*} i_{v*}}{i_{h*}}, \\ \delta &= 1 - n_{v*}, \end{aligned}$$

$$\nu = \frac{\delta i_{\nu*}}{\left(n_{\nu*} - i_{\nu*}\right) i_{h*}},$$
$$n_{h*}\left(1 - n_{h*}\right) = \beta s_{h*} i_{\nu*} + \alpha s_{h*}. \tag{33}$$

By adding and subtracting $\beta s_{h*} i_{\nu*}, \beta s_{h*} i_{\nu*}$ $(n_h i_{\nu*}/n_{h*} i_\nu)$ and using (31) and (33) in (32), after intensive simplification, we have

$$\begin{aligned}
\frac{dV}{dt} &= n_h\left(1 - n_h\right)\left(2 - \frac{n_{h*}}{n_h} - \frac{s_{h*}}{s_h}\right) \\
&+ \alpha s_{h*}\left(2 - \frac{n_h}{n_{h*}} - \frac{s_h}{s_{h*}}\right) \\
&+ \beta s_{h*} i_{\nu*}\left(3 - \frac{i_h}{i_{h*}} - \frac{n_h i_{\nu*}}{n_{h*} i_\nu} + \frac{s_h}{s_{h*}}\frac{i_{h*}}{i_h}\frac{i_\nu}{i_{\nu*}}\right) \\
&+ \beta s_{h*} i_{\nu*}\left(1 - \frac{i_{\nu*}}{i_\nu}\right)\left(\frac{i_\nu}{i_{\nu*}} - \frac{n_h}{n_{h*}}\right) \\
&+ \rho i_{h*}\left(\frac{n_{h*}}{n_h} - 1\right)\left(\frac{n_h}{n_{h*}} + \frac{i_h}{i_{h*}}\right) \\
&+ n_{\nu*} i_{\nu*}\left(\frac{n_\nu i_h}{n_{\nu*} i_{h*}} - \frac{i_\nu}{i_{\nu*}}\right)\left(1 - \frac{i_{\nu*}}{i_\nu}\right) \\
&- \frac{1}{\varepsilon}\left(n_\nu - n_{\nu*}\right)^2 \\
&- i_{h*} i_{\nu*}\left(1 - \frac{i_h}{i_{h*}}\right)\left(1 - \frac{i_\nu}{i_{\nu*}}\right).
\end{aligned} \tag{34}$$

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

$$\begin{aligned}
\left(2 - \frac{n_{h*}}{n_h} - \frac{s_{h*}}{s_h}\right) &\le 0, \\
\left(2 - \frac{n_h}{n_{h*}} - \frac{s_h}{s_{h*}}\right) &\le 0, \\
\left(3 - \frac{i_h}{i_{h*}} - \frac{n_h i_{\nu*}}{n_{h*} i_\nu} + \frac{s_h}{s_{h*}}\frac{i_{h*}}{i_h}\frac{i_\nu}{i_{\nu*}}\right) &\le 0.
\end{aligned} \tag{35}$$

Also,

$$\left(1 - \frac{i_{\nu*}}{i_\nu}\right)\left(\frac{i_\nu}{i_{\nu*}} - \frac{n_h}{n_{h*}}\right) \le 0, \tag{36}$$

if

$$\begin{aligned}
\frac{i_\nu}{i_{\nu*}} &\le \frac{n_h}{n_{h*}} \quad \text{when } i_\nu \ge i_{\nu*}, \\
\frac{i_\nu}{i_{\nu*}} &\ge \frac{n_h}{n_{h*}} \quad \text{when } i_\nu \le i_{\nu*},
\end{aligned} \tag{37}$$

$$\left(\frac{n_{h*}}{n_h} - 1\right)\left(\frac{n_h}{n_{h*}} + \frac{i_h}{i_{h*}}\right) \le 0, \quad \text{if } n_h \ge n_{h*}.$$

Furthermore,

$$\left(\frac{n_\nu i_h}{n_{\nu*} i_{h*}} - \frac{i_\nu}{i_{\nu*}}\right)\left(1 - \frac{i_{\nu*}}{i_\nu}\right) \le 0, \tag{38}$$

if

$$\begin{aligned}
\frac{n_\nu i_h}{n_{\nu*} i_{h*}} &\le \frac{i_\nu}{i_{\nu*}} \quad \text{when } i_{\nu*} \le i_\nu, \\
\frac{n_\nu i_h}{n_{\nu*} i_{h*}} &\ge \frac{i_\nu}{i_{\nu*}} \quad \text{when } i_{\nu*} \ge i_\nu.
\end{aligned} \tag{39}$$

Hence, it follows from (34), (35), (36), and (38) that $dV/dt \le 0$ in Ω. Thus, the equality $dV/dt = 0$ holds only when $n_h = n_{h*}$, $s_h = s_{h*}$, $i_h = i_{h*}$, $n_\nu = n_{\nu*}$, and $i_\nu = i_{\nu*}$. Therefore, the largest compact invariant set in $\{n_h, s_h, i_h, n_\nu, i_\nu \in \Omega : \dot{V} = 0\}$ is the singleton $\{E_2\}$, where E_2 is the endemic equilibrium. From the LaSalle's invariant principle [30], the unique equilibrium E_2 of system (9) is globally asymptomatically stable for $R_0 > 1$. □

3.3. Sensitivity Analysis. To understand the relative importance of parameters which are responsible for transmission and prevalence of malaria disease, described in the model (9), we perform a sensitivity analysis. Sensitivity indices help us to measure the relative change in a state variable while a parameter changes. The normalized sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter [27]. We calculate the sensitivity indices of R_0 to assess which parameter has a great impact on R_0 and hence the greatest effect in determining whether the disease dies out or persists with population.

Let P be the generic parameter of model (9). We, now only, derive the normalized sensitivity index of R_0 to each of the parameters involved in R_0, defined by the ratio of the relative change in R_0 to the relative change in the parameter P; that is,

$$\Pi_P^{R_0} = \frac{P}{R_0}\frac{\partial R_0}{\partial P}. \tag{40}$$

This index shows how sensitive R_0 is to a change in the parameter P. We notice that

$$\Pi_\beta^{R_0} = \frac{\beta}{R_0}\frac{\partial R_0}{\partial \beta} = \frac{\beta}{R_0}\sqrt{\frac{\nu\left(1-\alpha\right)\left(1-\delta\right)}{\delta\left(\alpha+\gamma+\rho\right)}}\frac{\partial\sqrt{\beta}}{\partial\beta} = \frac{1}{2}. \tag{41}$$

This indicates that $\Pi_\beta^{R_0}$ does not depend on any parameter value. Similarly, for the other parameters, we have

$$\begin{aligned}
\Pi_\nu^{R_0} &= \frac{1}{2}, \\
\Pi_\alpha^{R_0} &= -\frac{\beta\nu\alpha\left(1-\delta\right)\left(1+\gamma+\rho\right)}{2R_0^2\delta\left(\alpha+\gamma+\rho\right)^2} \\
\Pi_\gamma^{R_0} &= -\frac{\gamma}{2\left(\alpha+\gamma+\rho\right)},
\end{aligned}$$

Table 2: Some parameter values of the malaria model; all parameters are nondimensional.

Parameter	Baseline value	Source
β	0.0220	[31]
ν	0.4800	[31]
γ	0.1096	[32]
δ	0.2538	[31]
α	0.1455	[31]
ρ	0.8182	[31]

Table 3: Sensitivity indices of R_0 to parameters for the malaria model.

Parameter	Sensitivity index
β	+0.5000
ν	+0.5000
γ	−0.0511
δ	−0.6266
α	−0.1528
ρ	−0.3812

$$\Pi_{\rho}^{R_0} = -\frac{\rho}{2(\alpha+\gamma+\rho)},$$
$$\Pi_{\delta}^{R_0} = -\frac{\beta\nu(1-\alpha)}{2R_0^2\delta(\alpha+\gamma+\rho)^2}. \quad (42)$$

We evaluate the above sensitivity indices, in Table 3, using the parameter values in Table 2. The basic reproduction number, R_0, is most sensitive to the contact rates of human to vector and vector to human, with $\pi_{\beta}^{R_0} = 0.5000$ and $\pi_{\nu}^{R_0} = 0.5000$ as it can be seen in Table 3. This shows that any increase (decrease) by 10% in β or ν will increase (decrease) by 5% in R_0. The other parameters with highest sensitivity indices are δ, with $\pi_{\delta}^{R_0} = -0.6266$, and ρ, with $\pi_{\rho}^{R_0} = -0.3812$. Increasing (decreasing) δ by 10% will decrease (increase) in R_0 by 6.266% and increasing (decreasing) ρ by 10% will decrease (increase) in R_0 by 3.812% or vice versa. The rest of parameters, γ and α, have less significant effect in R_0.

In conclusion, the vector death rate, the human induced death rate, and the contact rates are important parameters in the model which have a significant impact on prevalence and transmission of the malaria disease; these parameters are able to control so that an intensive effort/work has to be done to eradicate the malaria disease from the population. Furthermore, one can understand from the sensitivity indices that vector control is the most effective control strategy.

4. Application of the Model

In this section, we present more simulations illustrating the abrupt and periodic variations of the model. We fix a reasonable parameter values of the model for numerical simulations.

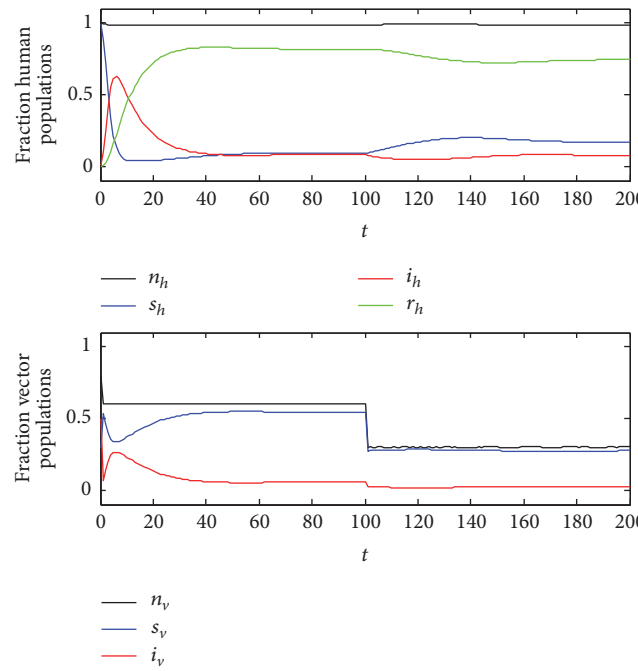

Figure 3: Numerical simulation for the fractions of human and vector population versus time with constant parameter values, $\varepsilon = 0.01$, $\alpha = 0.01$, $\beta = 2$, $\rho = 0.01$, $\gamma = 0.1$, $\delta = 0.4$, and $\nu = 0.5$, and initial values of the fractions of human host and mosquito vector $n_{h0} = 1.0$, $s_{h0} = 0.99$, $i_{h0} = 0.01$, $n_{v0} = 0.8$, $i_{v0} = 0.5$, and $K_h = 1$. Steps down the vector population by 50%.

We allow the mosquito sustainable level, $K_v(t) = K_v^0 g_v(t)$, noted in Section 3, to vary with respect to time. However, we first keep K_h fixed in order to investigate the impact of fast variation in K_v on the human populations. Periodic variations in K_v are shown in these plots for different periods. Plots of the abrupt changes in the sustainable populations, $K_h = K_h^0 g_h(t)$ (see in Section 3) and K_v, are located in Figures 3–7, whereas plots of periodic variations are shown in Figures 8 and 9.

Abrupt changes in the human and mosquito populations may, for example, be due to intensive spraying of the mosquitoes some massive emigration (refugee camps) or immigration for the humans. In Figures 3 and 4, steps down and up in vector sustainable population about 50% are plotted. These plots show that the transition occurs very fast and the system adjusts quickly to the new equilibrium. In Figures 5–7 increases about 50% and 100% and decrease about 50% in K_h are shown. Step change in humans needs caution since it may lead to unphysical solutions. In Figure 5, after a transient, the solution converges to the new equilibrium. An increase in K_h in Figure 6 may lead to an increase in the fraction of the human susceptible population for small time intervals, but not much dramatic change is shown. However, the slow change in human population after transient is shown in Figure 7. In the plots of periodic variation, one observes that the fast variation is quickly adapted by the vector population. This is because of the fast time scale for the mosquito population. Most of the figures show that the humans follow a

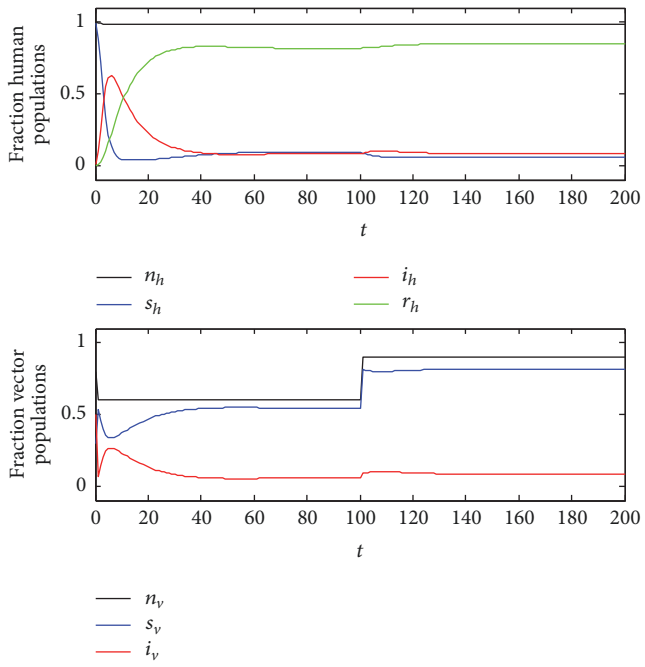

FIGURE 4: Numerical simulation for the fractions of human and vector population versus time with constant parameter values the same as in Figure 3 and steps up the vector population by 50%.

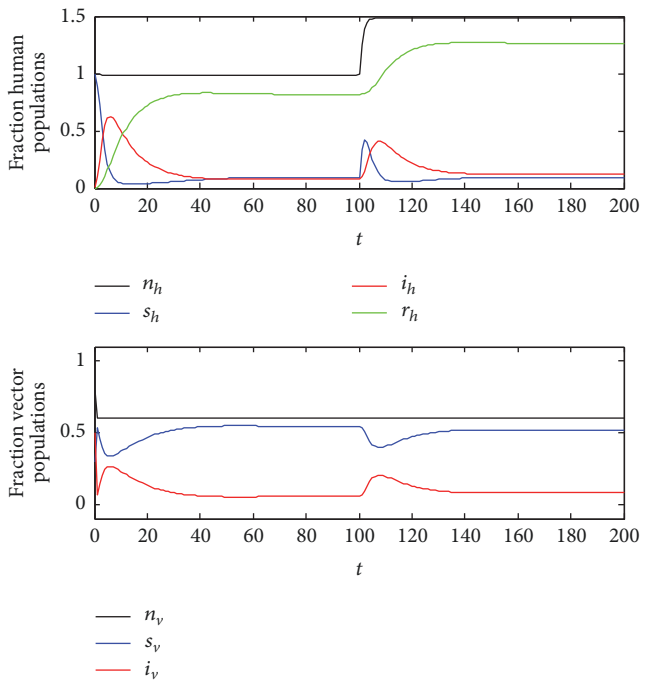

FIGURE 5: Numerical simulation for the fractions of human and vector population versus time with constant parameter values the same as in Figure 3 and steps up the human population by 50%.

slow variations relative to the vectors. Therefore, it is possible to say that fast variations in K_v do not imply large variation in human population. In general, for periods shorter than $1/\mu_h$, the human population do not in practice show the variations

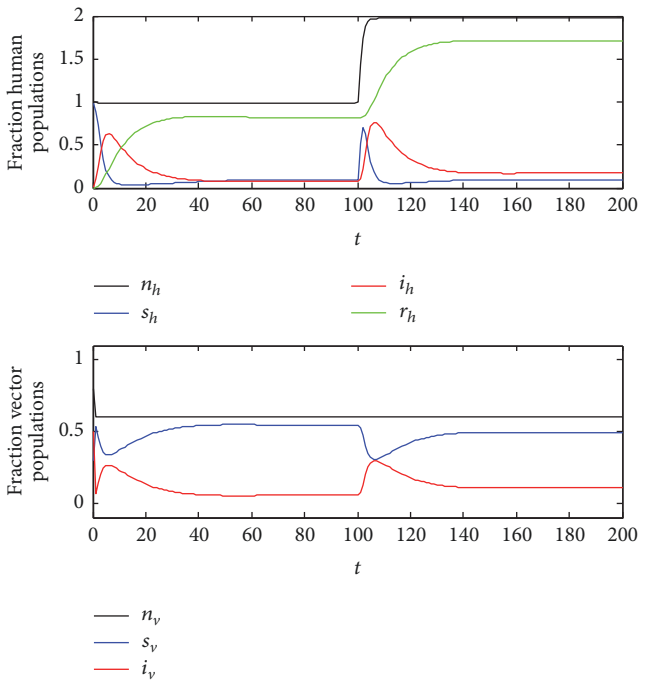

FIGURE 6: Numerical simulation for the fractions of human and vector population versus time with constant parameter values the same as in Figure 3 and increasing the human population by 100%.

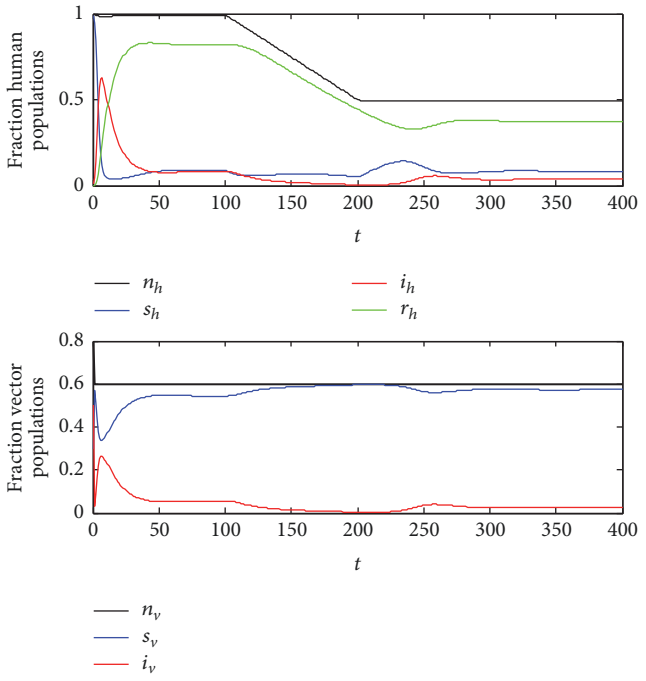

FIGURE 7: Numerical simulation for the fractions of human and vector population versus time with constant parameter values the same as in Figure 3 and $\varepsilon = 0.001$, and steps down the human population by 50% in the form of a constant drop over a time interval of length 100.

in the vector populations, but for long periods they do, but apparently weaker.

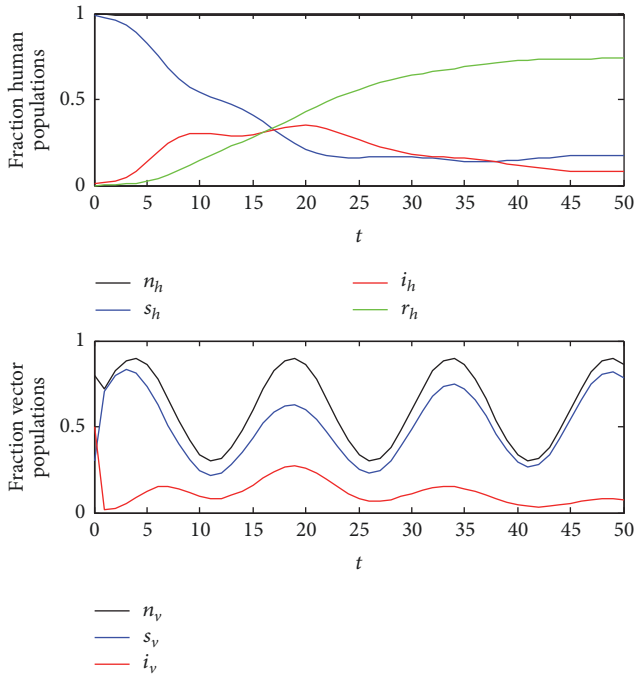

FIGURE 8: Numerical simulation for the fractions of human and vector population versus time with constant parameter values, $\varepsilon = 0.001$, $\alpha = 0.01$, $\beta = 0.7$, $\rho = 0.01$, $\gamma = 0.1$, $\delta = 0.4$, and $\nu = 0.5$, and initial values of the fractions of human host and mosquito vector, $n_{h0} = 1.0$, $s_{h0} = 0.99$, $i_{h0} = 0.01$, $n_{v0} = 0.8$, and $i_{v0} = 0.5$, and the sustainable levels $K_h = 1$ and periodic variations in K_v given by $K_v = 1 + 0.5\sin(2\pi t/15)$.

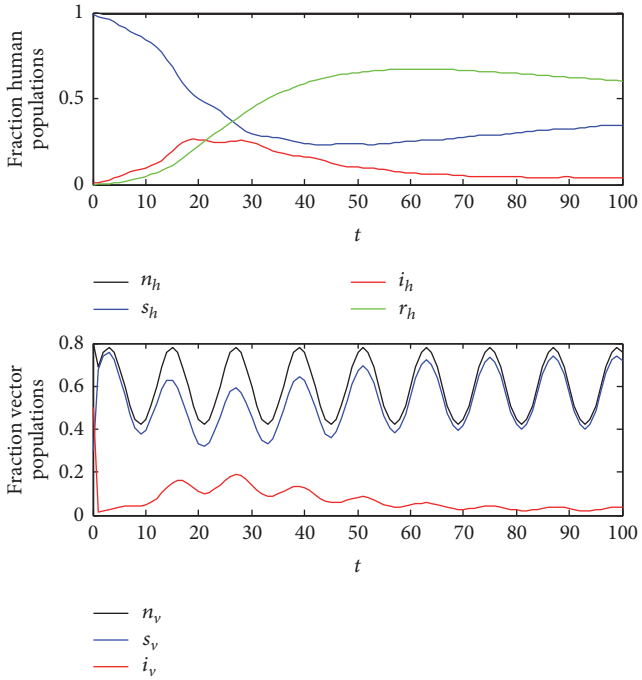

FIGURE 9: Periodic variations in K_v with $K_v = 1 + 0.3\sin(\pi t/6)$, for $K_h = 1$, and the other parameters and initial values remain fixed as in Figure 8 apart from $\beta = 0.5$ over the time interval [0 100].

5. Conclusions

In this work, we developed and analyzed a logistic malaria model to study the global stability of both disease-free and endemic equilibrium points. Mathematically, we formulated a five-dimensional system of deterministic ordinary differential equations and defined the domain where the model is epidemiologically feasible and mathematically well-posed. The model used the next generation matrix approach to obtain an explicit formula for a reproduction number, R_0, which is the expected number of secondary cases produced by a single infectious individual during its entire period of infectiousness in a fully susceptible populations.

Qualitative analysis of the model determines stability analysis of the equilibrium points. Accordingly, we obtained two diseases-free equilibrium points E_0 and E_1. The equilibrium point, E_0, is unstable and unphysical, while the equilibrium point, E_1, becomes both locally and globally stable whenever $R_0 < 1$ and $R_0 \leq 1$, respectively. We also have shown that the endemic equilibrium point, E_2, is globally asymptotically stable if $R_0 > 1$. Furthermore, sensitivity analysis of the model shows that the human induced death rate, the contact rates (human to mosquito or vice versa), and mosquito death rate have a significant effect on transmission and prevalence of the malaria disease. Moreover, numerical simulations are carried out in the application of the model to investigate how variations in the sustainable level of the vectors affect the human population. One can see from these simulations that fast variations in K_v do not lead to large variations in the human population.

State Variables, Parameters, Descriptions, and Their Dimensions of Malaria Model

S_h: Number of susceptible humans at a time t^*
I_h: Number of infected humans at a time t^*
R_h: Number of recovered humans at a time t^*
S_v: Number of susceptible mosquitoes at a time t^*
I_v: Number of infected mosquitoes at a time t^*
N_h: The total human population at a time t^*
N_v: The total mosquito population at a time t^*
K_h: Sustainable level of human population at a time t^*
K_v: Sustainable level of mosquito population at a time t^*
μ_h: Per capita birth rate of human population. Dimension: Time^{-1}
α_h: Per capita natural death rate for humans. Dimension: Time^{-1}
ρ_h: Per capita disease-induced death rate for humans. Dimension: Time^{-1}
β_h: The human contact rate. Dimension: $\text{Mosquitoes}^{-1} \times \text{Time}^{-1}$
γ_h: Per capita recovery rate for humans. Time^{-1}
μ_v: Per capita birth rate of mosquitoes. Dimension: Time^{-1}
α_v: Per capita natural death rate of mosquitoes. Dimension: Time^{-1}
β_v: The mosquito contact rate. Dimension: $\text{Humans}^{-1} \times \text{Time}^{-1}$.

Conflicts of Interest

The author declares that there are no conflicts of interest regarding the publication of this paper.

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