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Research Article

Stability Analysis and Optimal Control of a Vector-Borne Disease with Nonlinear Incidence

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The paper considers a model for the transmission dynamics of a vector-borne disease with nonlinear incidence rate. It is proved that the global dynamics of the disease are completely determined by the basic reproduction number. In order to assess the effectiveness of disease control measures, the sensitivity analysis of the basic reproductive number R_0 and the endemic proportions with respect to epidemiological and demographic parameters are provided. From the results of the sensitivity analysis, the model is modified to assess the impact of three control measures; the preventive control to minimize vector human contacts, the treatment control to the infected human, and the insecticide control to the vector. Analytically the existence of the optimal control is established by the use of an optimal control technique and numerically it is solved by an iterative method. Numerical simulations and optimal analysis of the model show that restricted and proper use of control measures might considerably decrease the number of infected humans in a viable way.

1. Introduction

Vector-borne diseases are infectious diseases caused by viruses, bacteria, protozoa, or rickettsia which are primarily transmitted by disease transmitting biological agents, called vectors. Vector-borne diseases, in particular, mosquito-borne diseases such as malaria, dengue fever, and West Nile Virus that are transmitted to humans by blood-sucker mosquito, have been big problem for the public health in the world. The literature dealing with the mathematical theory and dynamics of vector-borne diseases are quite extensive.

Many mathematical models concerning the emergence and reemergence of the vector-host infectious disease have been proposed and analyzed in the literature [1, 2].

Mathematical modeling became considerable important tool in the study of epidemiology because it helped us to understand the observed epidemiological patterns, disease control and provide understanding of the underlying mechanisms which influence the spread of disease and may suggest control strategies. The model formulation and its simulation with parameter estimation allow us to test for sensitivity and comparison of conjunctures. The foundations of the modern mathematical epidemiology based on the compartment models were laid in the early 20th century [3].

The incidence of a disease is the number of infection per unit time and plays an important role in the study of mathematical epidemiology. In classical epidemiological bilinear incidence rate βSI and standard incidence rate $\beta(S/N)I$ are frequently used, where β is the probability of transmission per contact, S is susceptible, and I is infective individuals. However, actual data and evidence observed for many diseases show that dynamics of disease transmission are not always as simple as shown in these rates. There are a number of biological mechanisms which may result in nonlinearities in the transmission rates. In 1978, Capasso and Serio [4] introduced a saturated incidence rate $g(I)S$ in an epidemic models. This is important because the number of effective contacts between infective and susceptible individuals may saturate at high infective levels due to overcrowding of infective individuals or due to protective measures endorsed by susceptible individuals. A variety of nonlinear incidence rates have been used in epidemic models [5–10]. In [10], an epidemic model with nonlinear incidences is proposed to describe the dynamics of diseases spread by vectors, (mosquitoes), such as malaria, yellow fever, dengue and so on.

Optimal control theory is a powerful mathematical tool to make decision involving complex dynamical systems [11]. For example, what percentage of the population should be vaccinated as time evolves in a given epidemic model to minimize both the number of infected people and the cost of implementing the vaccination strategy. The desired outcome depends on the particular situation. New drug treatments and combinations of drugs are under constant development. The optimal treatment scheme for patients remains the subject of intense debate. Further, optimal control methods have been used to study the dynamics of some diseases (see [12, 13] and the references therein).

Recently, a number of mathematical models have been proposed to study the transmission dynamics of vector-borne diseases. Cai and Li [1] describes the dynamics of a vector-borne disease considering that the infection moves from person to person directly with no environmental source and intermediate vector or host. There have been applications of optimal control methods to epidemiological models, namely, Blayneh et al. [14], Okosun and Makinde [15], Lashari and Zaman [16, 17], and so forth. Lashari and Zaman [17] used personal protection, blood screening, and vector-reduction strategies as optimal control to reduce the transmission of a vector-borne disease. Kar and Batabyal [18] analyzed a nonlinear epidemic model and used optimal control technique to reduce the disease burden with a vaccination program.

In this work, we consider a vector host epidemic model with nonlinear incidence rate. Our aim is to carry out qualitative behavior and present a rigorous analysis of the resulting model to investigate the parameters to show how they affect the vector-borne disease transmission. We perform sensitivity analysis of the basic reproductive number and the endemic equilibrium with respect to epidemiological and demographic parameters. From the sensitivity analysis, we find that the reproductive number is most sensitive to the biting and mortality rates of mosquito. Further, the treatment rate of infectious humans is also

a sensitive parameter for equilibrium proportion of infectious humans. These suggest us to develop strategies that target the mosquito biting rate, mosquito death rate, and treatment of infectious individuals in controlling the disease. Based on sensitivity analysis, we formulate an optimal control problem to minimize the number of infected human using three main efforts as control measures. Unfortunately, there is no vaccine nor specific treatment against vector-borne disease is available; that is why the main measures to limit the impact of such epidemic have to be considered. Therefore, we look at time-dependent prevention, treatment efforts and breeding sites destruction, for which optimal control theory is applied.

This paper is organized as follows. The model is developed in Section 2. The analysis of global stability of the equilibria of the model is investigated in Section 3. Section 4 focuses on the sensitivity analysis. Section 5 describes the extended model with three control measures and numerical simulations are presented in Section 6. Finally, conclusions are summarized in Section 7.

2. Model Formulation

The total human population, denoted by $N_h(t)$, is split into susceptible individuals ($S_h(t)$) and infected individuals ($I_h(t)$) so that $N_h(t) = S_h(t) + I_h(t)$. Whereas, the total vector population, denoted by $N_v(t)$, is subdivided into susceptible vectors ($S_v(t)$) and infectious vectors ($I_v(t)$). Thus $N_v(t) = S_v(t) + I_v(t)$.

The dynamics of the disease are described by the following system of differential equations:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \frac{b\beta_1 S_h I_v}{1+\alpha_1 I_v} - \frac{\beta_3 S_h I_h}{1+\alpha_2 I_h} - \mu_h S_h + \gamma_h I_h,\\
\frac{dI_h}{dt} &= \frac{b\beta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\beta_3 S_h I_h}{1+\alpha_2 I_h} - \mu_h I_h - \gamma_h I_h,\\
\frac{dS_v}{dt} &= \Lambda_v - \frac{b\beta_2 I_h S_v}{1+\alpha_3 I_h} - \mu_v S_v,\\
\frac{dI_v}{dt} &= \frac{b\beta_2 I_h S_v}{1+\alpha_3 I_h} - \mu_v I_v.
\end{aligned} \tag{2.1}$$

Susceptible humans are recruited at a rate Λ_h, whereas susceptible vectors are generated by Λ_v. We assume that the number of bites per vector per host per unit time is φ, the proportion of infected bites that gives rise to the infection is r, and the ratio of vector numbers to host numbers is ξ. Let $b = \varphi r \xi$, let β_1 be the transmission rate from vector to human, and let β_2 be the transmission rate from human to vector. β_3 is the transmission probability from human to human. μ_h is natural death rate of human, μ_v is death rate of vectors, respectively. We assume that infectious individuals do not acquire permanent immunity and become susceptible again by the rate γ_h. Further we assume that incidence terms for human population and vector population that transmit disease are saturation interactions and are given by $b\beta_1 S_h I_v/(1+\alpha_1 I_v)$, $\beta_3 S_h I_h/(1+\alpha_2 I_h)$, and $b\beta_2 I_h S_v/(1+\alpha_3 I_h)$, where α_1, α_2, and α_3 determine the level at which the force of infection saturates.

Obviously, $\Delta = \{(S_h, I_h, S_v, I_v) \in R^4 : S_h + I_h = \Lambda_h/\mu_h, S_v + I_v = \Lambda_v/\mu_v\}$ is positively invariant, system (2.1) is dissipative, and the global attractor is contained in Δ.

The total dynamics of vector population are $dN_v/dt = \Lambda_v - \mu_v N_v$. Thus we can assume without loss of generality that $N_v = \Lambda_v/\mu_v$ for all $t \geq 0$ provided that $S_v(0) + I_v(0) = \Lambda_v/\mu_v$. On Δ, $S_v = \Lambda_v/\mu_v - I_v$. Therefore, we attack system (2.1) by studying the subsystem

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \frac{b\beta_1 S_h I_v}{1+\alpha_1 I_v} - \frac{\beta_3 S_h I_h}{1+\alpha_2 I_h} - \mu_h S_h + \gamma_h I_h,\\
\frac{dI_h}{dt} &= \frac{b\beta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\beta_3 S_h I_h}{1+\alpha_2 I_h} - \mu_h I_h - \gamma_h I_h,\\
\frac{dI_v}{dt} &= \frac{b\beta_2}{\mu_v}\frac{(\Lambda_v - \mu_v I_v) I_h}{1+\alpha_3 I_h} - \mu_v I_v.
\end{aligned} \tag{2.2}$$

From biological considerations, we study system (2.2) in the closed set $\Omega = \{(S_h, I_h, I_v) \in R_+^3 : S_h + I_h = \Lambda_h/\mu_h, I_v \leq \Lambda_v/\mu_v\}$, where R_+^3 denotes the nonnegative cone of R^3 including its lower dimensional faces. It can be easily verified that Ω is positively invariant with respect to (2.2).

3. Mathematical Analysis of the Model

The dynamics of the disease are described by the basic reproduction number R_0. The threshold quantity R_0 is called the reproduction number, which is defined as the average number of secondary infections produced by an infected individual in a completely susceptible population. The basic reproduction number of model (2.2) is given by the expression

$$R_0 = \frac{\beta_3 \Lambda_h}{\mu_h(\mu_h + \gamma_h)} + \frac{b^2\beta_1\beta_2\Lambda_h\Lambda_v}{\mu_v^2\mu_h(\mu_h + \gamma_h)}. \tag{3.1}$$

Direct calculation shows that system (2.2) has two equilibrium states. For $R_0 \leq 1$, the only equilibrium is disease-free equilibrium $E_0 = (\Lambda_h/\mu_h, 0, 0)$. For $R_0 > 1$, there is an additional equilibrium $E^*(S_h^*, I_h^*, I_v^*)$ which is called endemic equilibrium, where

$$\begin{aligned}
S_h^* &= \frac{\Lambda_h - \mu_h I_h^*}{\mu_h},\\
I_v^* &= \frac{b\beta_2\Lambda_v I_h^*}{\mu_v^2 + (\alpha_3\mu_v^2 + b\beta_2\mu_v) I_h^*},
\end{aligned} \tag{3.2}$$

and I_h^* is the root of the following quadratic equation.

$$a_1 I_h^{2*} + a_2 I_h^* + a_3 = 0, \tag{3.3}$$

with

$$\begin{aligned}
a_1 &= \alpha_2 \mu_h b^2 \beta_1 \beta_2 \Lambda_v + \left(\alpha_3 \mu_v^2 + b\beta_2 \mu_v + \alpha_1 b \beta_2 \Lambda_v\right)\left[\beta_3 \mu_h + \alpha_2 \mu_h (\mu_h + \gamma_h)\right], \\
a_2 &= \mu_h \left(b^2 \beta_1 \beta_2 \Lambda_v + \beta_3 \mu_v^2\right) + \alpha_2 \left(\mu_v^2 \mu_h (\mu_h + \gamma_h) - b^2 \beta_1 \beta_2 \Lambda_h \Lambda_v\right) \\
&\quad + \left(\alpha_3 \mu_v^2 + b\beta_2 \mu_v + \alpha_1 b \beta_2 \Lambda_v\right)\left(\mu_h (\mu_h + \gamma_h) - \Lambda_h \beta_3\right), \\
a_3 &= \mu_h (\mu_h + \gamma_h) \mu_v^2 (1 - R_0).
\end{aligned} \tag{3.4}$$

From (3.3), we see that $R_0 > 1$ if and only if $a_3 < 0$. Since $a_1 > 0$, (3.3) has a unique positive root in feasible region. If $R_0 < 1$, then $a_3 > 0$. Also, it can be easily seen that $a_2 > 0$ for $R_0 < 1$. Thus, by considering the shape of the graph of (3.3) (and noting that $a_3 > 0$), we have that there will be zero (positive) endemic equilibrium in this case. Therefore, we can conclude that if $R_0 < 1$, (3.3) has no positive root in the feasible region. If, $R_0 > 1$, (3.3) has a unique positive root in the feasible region. This result is summarized below.

Theorem 3.1. *System* (2.2) *always has the infection-free equilibrium* E_0. *If* $\mathcal{R}_0 > 1$, *system* (2.2) *has a unique endemic equilibrium* $E^* = (S_h^*, I_h^*, I_v^*)$ *defined by* (3.2) *and* (3.3).

3.1. Global Stability of Disease-Free Equilibrium

In this subsection, we analyze the global behavior of the equilibria for system (2.2). The following theorem provides the global property of the disease-free equilibrium E_0 of the system.

Theorem 3.2. *If* $R_0 \leq 1$, *then the infection-free equilibrium* E_0 *is globally asymptotically stable in the interior of* Ω.

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

$$L(t) = I_h(t) + b\beta_1 \frac{\Lambda_h}{\mu_h \mu_v} I_v(t). \tag{3.5}$$

Calculating the time derivative of L along the solutions of system (2.2), we obtain

$$\begin{aligned} L'(t) &= I_h'(t) + b\beta_1 \frac{\Lambda_h}{\mu_h \mu_v} I_v'(t) \\ &= \frac{b\beta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\beta_3 S_h I_h}{1+\alpha_2 I_h} - (\mu_h + \gamma_h) I_h + b\beta_1 \frac{\Lambda_h}{\mu_h \mu_v} \left\{ \frac{b\beta_2 \Lambda_v}{\mu_v (1+\alpha_3 I_h)} I_h - \frac{b\beta_2 I_v I_h}{1+\alpha_3 I_h} - \mu_v I_v \right\} \\ &\leq \frac{b\beta_1 \Lambda_h}{\mu_h} I_v + \frac{\beta_3 \Lambda_h}{\mu_h} I_h - (\mu_h + \gamma_h) I_h + b\beta_1 \frac{\Lambda_h}{\mu_h \mu_v} \left\{ \frac{b\beta_2 \Lambda_v}{\mu_v} I_h - \frac{b\beta_2 I_v I_h}{1+\alpha_3 I_h} - \mu_v I_v \right\} \\ &= -(\mu_h + \gamma_h) I_h (1 - R_0) - b^2 \beta_1 \beta_2 \frac{\Lambda_h}{\mu_h \mu_v} \frac{I_v I_h}{1+\alpha_3 I_h}. \end{aligned} \tag{3.6}$$

Thus $L'(t)$ is negative if $R_0 \leq 1$. When $R_0 < 1$, the derivative $L' = 0$ if and only if $I_h = 0$, while in the case $R_0 = 1$, the derivative $L' = 0$ if and only if $I_h = 0$ or $I_v = 0$. Consequently, the largest compact invariant set in $\{(S_h, I_h, I_v) \in \Omega, L' = 0\}$, when $R_0 \leq 1$, is the singelton E_0. Hence, LaSalle's invariance principle [19] implies that E_0 is globally asymptotically stable in Ω. This completes the proof. □

3.2. Global Stability of the Endemic Equilibrium

Here, we use the geometrical approach of Li and Muldowney to investigate the global stability of the endemic equilibrium E^* in the feasible region Ω. We have omitted the detailed introduction of this approach and we refer the interested readers to see [20]. We summarize this approach below.

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

$$x' = f(x) \tag{3.7}$$

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

(H_1) D is simply connected;

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

(H_3) Equation (3.7) has unique equilibrium $\overline{x}$ in D.

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

Let μ be the Lozinskiĭ measure with respect to the $|\cdot|$. Define a quantity $\overline{q}_2$ as

$$\overline{q}_2 = \limsup_{t \to \infty} \sup_{x_0 \in K} \frac{1}{t} \int_0^t \mu(B(x(s, x_0)))ds, \tag{3.8}$$

where $B = P_f P^{-1} + P J^{[2]} P^{-1}$, the matrix P_f is obtained by replacing each entry p of P by its derivative in the direction of f, $(p_{ij})_f$, and $J^{[2]}$ is the second additive compound matrix of the

Jacobian matrix J of (3.7). The following result has been established in Li and Muldowney [20].

Theorem 3.3. *Suppose that* (H_1), (H_2) *and* (H_3) *hold, the unique endemic equilibrium* E^* *is globally stable in* Ω *if* $\overline{q}_2 < 0$.

Obviously Ω is simply connected and E^* is unique endemic equilibrium for $R_0 > 1$ in Ω. To apply the result of the above theorem for global stability of endemic equilibrium E^*, we first state and prove the following result.

Lemma 3.4. *If* $R_0 > 1$, *then the system* (2.2) *is uniformly persistent; that is, there exists* $c > 0$ *(independent of initial conditions), such that* $\liminf_{t\to\infty} S_h \geq c$, $\liminf_{t\to\infty} I_h \geq c$, *and* $\liminf_{t\to\infty} I_v \geq c$,

Proof. Let Φ be semidynamical system (2.2) in $(R_0^+)^3$, let F be a locally compact metric space, and let $\Gamma_0 = \{(S_h, I_h, I_v) \in \Omega : I_v = 0\}$. Γ_0 is a compact subset of Ω and Ω/Γ_0 is positively invariant set of system (2.2). Let $P : F \to R_0^+$ be defined by $P(S_h, I_h, I_v) = I_v$ and set $S = \{(S_h, I_h, I_v) \in \Omega : P(S_h, I_h, I_v) < \phi\}$, where ϕ is sufficiently small so that

$$\frac{\beta_3\Lambda_h}{\mu_h(\mu_h+\gamma_h)(1+\alpha_2\phi)} + \frac{b^2\beta_1\beta_2\Lambda_h\Lambda_v(1-(\mu_v/\Lambda_v)\phi)}{\mu_v^2\mu_h(\mu_h+\gamma_h)(1+\alpha_3\phi)} > 1. \tag{3.9}$$

Assume that there is a solution $x \in S$ such that for each $t > 0$ $P(\Phi(x,t)) < P(x) < \phi$. Let us consider

$$L(t) = \frac{b\beta_1\Lambda_h}{\mu_h\mu_v}(1-\delta^*)I_v + I_h, \tag{3.10}$$

where δ^* is sufficiently small so that

$$\frac{\beta_3\Lambda_h}{\mu_h(\mu_h+\gamma_h)(1+\alpha_2\phi)} + \frac{b^2\beta_1\beta_2\Lambda_h\Lambda_v(1-(\mu_v/\Lambda_v)\phi)(1-\delta^*)}{\mu_v^2\mu_h(\mu_h+\gamma_h)(1+\alpha_3\phi)} > 1. \tag{3.11}$$

By direct calculation we have

$$\begin{aligned} L'(t) &\geq (\mu_h+\gamma_h)\left(\frac{\beta_3\Lambda_h}{\mu_h(\mu_h+\gamma_h)(1+\alpha_2\phi)} + \frac{b^2\beta_1\beta_2\Lambda_h\Lambda_v(1-(\mu_v/\Lambda_v)\phi)(1-\delta^*)}{\mu_v^2\mu_h(\mu_h+\gamma_h)(1+\alpha_3\phi)} - 1\right)I_h \\ &\quad + \frac{b\beta_1\Lambda_h}{\mu_h}\delta^* I_v, \\ L'(t) &\geq \alpha L(t), \end{aligned} \tag{3.12}$$

where

$$\alpha=\min\left\{(\mu_h+\gamma_h)\left(\frac{\beta_3\Lambda_h}{\mu_h(\mu_h+\gamma_h)(1+\alpha_2\phi)}+\frac{b^2\beta_1\beta_2\Lambda_h\Lambda_v(1-(\mu_v/\Lambda_v)\phi)(1-\delta^*)}{\mu_v^2\mu_h(\mu_h+\gamma_h)(1+\alpha_3\phi)}-1\right),\frac{\mu_v\delta^*}{1-\delta^*}\right\}. \tag{3.13}$$

This implies that $L(t)\to\infty$ as $t\to\infty$. However $L(t)$ is bounded on Ω. According to [21, Theorem 1] the proof is completed. □

The boundedness of Ω and the above lemma imply that (2.2) has a compact absorbing set $K\subset\Omega$ [22]. Now we shall prove that the quantity $\overline{q}_2<0$. We choose a suitable vector norm $|\cdot|$ in R^3 and a 3×3 matrix valued function

$$P(x)=\begin{pmatrix}1&0&0\\0&\dfrac{I_h}{I_v}&0\\0&0&\dfrac{I_h}{I_v}\end{pmatrix}. \tag{3.14}$$

Obviously P is C^1 and nonsingular in the interior of Ω. Linearizing system (2.2) about an endemic equilibrium E^* gives the following Jacobian matrix.

$$J(E^*)=\begin{pmatrix}-\dfrac{b\beta_1I_v}{1+\alpha_1I_v}-\dfrac{\beta_3I_h}{1+\alpha_2I_h}-\mu_h & -\dfrac{\beta_3S_h}{(1+\alpha_2I_h)^2}+\gamma_h & -\dfrac{b\beta_1S_h}{(1+\alpha_1I_v)^2}\\ \dfrac{b\beta_1I_v}{1+\alpha_1I_v}+\dfrac{\beta_3I_h}{1+\alpha_2I_h} & \dfrac{\beta_3S_h}{(1+\alpha_2I_h)^2}-(\mu_h+\gamma_h) & \dfrac{b\beta_1S_h}{(1+\alpha_1I_v)^2}\\ 0 & \dfrac{b\beta_2}{\mu_v}\dfrac{\Lambda_v-\mu_vI_v}{(1+\alpha_3I_h)^2} & -\dfrac{b\beta_2I_h}{(1+\alpha_3I_h)}-\mu_v\end{pmatrix}. \tag{3.15}$$

The second additive compound matrix of $J(E^*)$ is given by

$$J^{[2]}=\begin{pmatrix}M_{11} & \dfrac{b\beta_1S_h}{(1+\alpha_1I_v)^2} & \dfrac{b\beta_1S_h}{(1+\alpha_1I_v)^2}\\ \dfrac{b\beta_2}{\mu_v}\dfrac{\Lambda_v-\mu_vI_v}{(1+\alpha_3I_h)^2} & M_{22} & -\dfrac{\beta_3S_h}{(1+\alpha_2I_h)^2}+\gamma_h\\ 0 & \dfrac{b\beta_1I_v}{1+\alpha_1I_v}+\dfrac{\beta_3I_h}{1+\alpha_2I_h} & M_{33}\end{pmatrix}, \tag{3.16}$$

where

$$
\begin{aligned}
M_{11} &= -\frac{b\beta_1 I_v}{1+\alpha_1 I_v} - \frac{\beta_3 I_h}{1+\alpha_2 I_h} - \mu_h + \frac{\beta_3 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h),\\
M_{22} &= -\frac{b\beta_1 I_v}{1+\alpha_1 I_v} - \frac{\beta_3 I_h}{1+\alpha_2 I_h} - \mu_h - \frac{b\beta_2 I_h}{(1+\alpha_3 I_h)} - \mu_v,\\
M_{33} &= \frac{\beta_3 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h) - \frac{b\beta_2 I_h}{(1+\alpha_3 I_h)} - \mu_v.
\end{aligned}
\tag{3.17}
$$

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

$$
B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix},
\tag{3.18}
$$

with

$$
\begin{aligned}
B_{11} &= -\frac{b\beta_1 I_v}{1+\alpha_1 I_v} - \frac{\beta_3 I_h}{1+\alpha_2 I_h} - \mu_h + \frac{\beta_3 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h),\\
B_{12} &= \left(\frac{b\beta_1 S_h}{(1+\alpha_1 I_v)^2} \frac{I_v}{I_h}, \frac{b\beta_1 S_h}{(1+\alpha_1 I_v)^2} \frac{I_v}{I_h} \right),\\
B_{21} &= \begin{pmatrix} \left(\frac{I_h}{I_v}\right) \frac{b\beta_2}{\mu_v} \frac{\Lambda_v - \mu_v I_v}{(1+\alpha_3 I_h)^2} \\ 0 \end{pmatrix},\\
B_{22} &= \begin{pmatrix} Q_{11} & Q_{12} \\ Q_{21} & Q_{22} \end{pmatrix},
\end{aligned}
\tag{3.19}
$$

where

$$
\begin{aligned}
Q_{11} &= \frac{I_v}{I_h}\left(\frac{I_h}{I_v}\right)_f - \frac{b\beta_1 I_v}{1+\alpha_1 I_v} - \frac{\beta_3 I_h}{1+\alpha_2 I_h} - \mu_h - \frac{b\beta_2 I_h}{(1+\alpha_3 I_h)} - \mu_v,\\
Q_{12} &= -\frac{\beta_3 S_h}{(1+\alpha_2 I_h)^2} + \gamma_h,\\
Q_{21} &= \frac{b\beta_1 I_v}{1+\alpha_1 I_v} + \frac{\beta_3 I_h}{1+\alpha_2 I_h},\\
Q_{22} &= \frac{I_v}{I_h}\left(\frac{I_h}{I_v}\right)_f + \frac{\beta_3 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h) - \frac{b\beta_2 I_h}{(1+\alpha_3 I_h)} - \mu_v,\\
\frac{I_v}{I_h}\left(\frac{I_h}{I_v}\right)_f &= \frac{I_h'}{I_h} - \frac{I_v'}{I_v}.
\end{aligned}
\tag{3.20}
$$

Consider the norm in R^3 as $|(u,v,w)| = \max(|u|, |v| + |w|)$ where (u,v,w) denotes the vector in R^3. The Lozinskiĭ measure with respect to this norm is defined as $\mu(B) \leq \sup(g_1, g_2)$, where

$$g_1 = \mu_1(B_{11}) + |B_{12}|, \qquad g_2 = \mu_1(B_{22}) + |B_{21}|. \tag{3.21}$$

From system (2.2) we can write

$$\begin{aligned} \frac{I_h'}{I_h} &= \frac{b\beta_1 S_h}{1+\alpha_1 I_v}\frac{I_v}{I_h} + \frac{\beta_3 S_h}{1+\alpha_2 I_h} - (\mu_h + \gamma_h), \\ \frac{I_v'}{I_v} &= \frac{b\beta_2}{\mu_v}\frac{(\Lambda_v - \mu_v I_v)}{1+\alpha I_h}\frac{I_h}{I_v} - \mu_v. \end{aligned} \tag{3.22}$$

Since B_{11} is a scalar, its Lozinskiĭ measure with respect to any vector norm in R^1 will be equal to B_{11}. Thus

$$\begin{aligned} B_{11} &= -\frac{b\beta_1 I_v}{1+\alpha_1 I_v} - \frac{\beta_3 I_h}{1+\alpha_2 I_h} - \mu_h + \frac{\beta_3 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h), \\ |B_{12}| &= \frac{b\beta_1 S_h}{(1+\alpha_1 I_v)^2}\frac{I_v}{I_h}, \end{aligned} \tag{3.23}$$

and g_1 will become

$$\begin{aligned} g_1 &= -\frac{b\beta_1 I_v}{1+\alpha_1 I_v} - \frac{\beta_3 I_h}{1+\alpha_2 I_h} - \mu_h + \frac{\beta_3 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h) + \frac{b\beta_1 S_h}{(1+\alpha_1 I_v)^2}\frac{I_v}{I_h} \\ &\leq -\frac{b\beta_1 I_v}{1+\alpha_1 I_v} - \frac{\beta_3 I_h}{1+\alpha_2 I_h} - \mu_h + \frac{\beta_3 S_h}{(1+\alpha_2 I_h)} - (\mu_h + \gamma_h) + \frac{b\beta_1 S_h}{(1+\alpha_1 I_v)}\frac{I_v}{I_h} \\ &\leq \frac{I_h'}{I_h} - \mu_h - \frac{b\beta_1 I_v}{1+\alpha_1 I_v} - \frac{\beta_3 I_h}{1+\alpha_2 I_h}. \end{aligned} \tag{3.24}$$

Also $|B_{21}| = (I_h/I_v)(b\beta_2/\mu_v)((\Lambda_v - \mu_v I_v)/(1+\alpha_3 I_h)^2)$, $|B_{12}|$ and $|B_{21}|$ are the operator norms of B_{12} and B_{21} which are mapping from R^2 to R and from R to R^2, respectively, and R^2 is

endowed with the l_1 norm. $\mu_1(B_{22})$ is the Lozinskiĭ measure of 2×2 matrix B_{22} with respect to l_1 norm in R^2.

$$\begin{aligned}
&\mu(B_{22})\\
&= \operatorname{Sup}\left\{ \frac{I_v}{I_h}\left(\frac{I_h}{I_v}\right)_f - \frac{b\beta_1 I_v}{1+\alpha_1 I_v} - \frac{\beta_3 I_h}{1+\alpha_2 I_h} - \mu_h - \frac{b\beta_2 I_h}{(1+\alpha_3 I_h)} - \mu_v + \frac{b\beta_1 I_v}{1+\alpha_1 I_v} + \frac{\beta_3 I_h}{1+\alpha_2 I_h}, \right.\\
&\qquad \left. \frac{I_v}{I_h}\left(\frac{I_h}{I_v}\right)_f + \frac{\beta_3 S_h}{(1+\alpha_2 I_h)^2} - (\mu_h + \gamma_h) - \frac{b\beta_2 I_h}{(1+\alpha_3 I_h)} - \mu_v - \frac{\beta_3 S_h}{(1+\alpha_2 I_h)^2} + \gamma_h \right\}\\
&= \frac{I_v}{I_h}\left(\frac{I_h}{I_v}\right)_f - \mu_h - \frac{b\beta_2 I_h}{(1+\alpha_3 I_h)} - \mu_v.
\end{aligned} \tag{3.25}$$

Hence

$$\begin{aligned}
g_2 &= \frac{I_h'}{I_h} - \frac{I_v'}{I_v} + \left(\frac{I_h}{I_v}\right)\frac{b\beta_2}{\mu_v}\frac{\Lambda_v - \mu_v I_v}{(1+\alpha_3 I_h)^2} - \mu_h - \frac{b\beta_2 I_h}{(1+\alpha I_h)} - \mu_v\\
&\le \frac{I_h'}{I_h} - \frac{I_v'}{I_v} + \left(\frac{I_h}{I_v}\right)\frac{b\beta_2}{\mu_v}\frac{\Lambda_v - \mu_v I_v}{(1+\alpha_3 I_h)} - \mu_h - \frac{b\beta_2 I_h}{(1+\alpha_3 I_h)} - \mu_v\\
&\le \frac{I_h'}{I_h} - \frac{I_v'}{I_v} + \frac{I_v'}{I_v} - \mu_h - \frac{b\beta_2 I_h}{(1+\alpha_3 I_h)}\\
&\le \frac{I_h'}{I_h} - \mu_h - \frac{b\beta_2 I_h}{(1+\alpha_3 I_h)}.
\end{aligned} \tag{3.26}$$

Thus,

$$\mu(B) = \operatorname{Sup}\{g_1, g_2\} \le \frac{I_h'}{I_h} - \mu_h. \tag{3.27}$$

Since (2.2) is uniformly persistent when $R_0 > 1$, so for $T > 0$ such that $t > T$ implies $I_h(t) \ge c$, $I_v(t) \ge c$ and $(1/t)\log I_h(t) < \mu/2$ for all $(S_h(0), I_h(0), I_v(0)) \in K$. Thus

$$\frac{1}{t}\int_0^t \mu(B)dt < \frac{\log I_h(t)}{t} - \mu < \frac{-\mu}{2} \tag{3.28}$$

for all $(S_h(0), I_h(0), I_v(0)) \in K$, which further implies that $\overline{q}_2 < 0$. Therefore all the conditions of Theorem 3.3 are satisfied. Hence unique endemic equilibrium E^* is globally stable in Ω.

4. Sensitivity Analysis

We would like to know different factors that are responsible for the disease transmission and prevalence. In this way we can try to reduce human mortality and morbidity due

Table 1: Values for parameters used for sensitivity analysis.

Parameter	Value	Reference
Λ_h	0.00011	[23]
Λ_v	0.13	[23]
b	0.5	[23]
γ_h	0.7	Assumed
β_1	0.022	[23]
β_2	0.48	[23]
β_3	0.004	Assumed
α	5	Assumed
μ_h	0.000016	[23]
μ_v	0.033	[23]

Table 2: Sensitivity indices of R_0 to parameters for the model, evaluated at the parameter values given in Table 1.

Parameter	Description	Sensitivity index
b	Rate of biting of a host by mosquito	1.97493
γ_h	Loss of immunity	−0.999977
β_1	Probability of transmission from mosquitoes to host	0.987467
β_2	Probability of transmission from host to mosquitoes	0.987467
β_3	Probability of transmission from infectious human to susceptible human	0.0125332
Λ_h	Recruitment rate of susceptible hosts	1
Λ_v	Recruitment rate of susceptible mosquitoes	0.987467
μ_v	Death rate of mosquitoes	−1.97493
μ_h	Death rate of hosts	−1.00002

to disease. Initial disease transmission depends upon the reproductive number whereas disease prevalence is directly related to the endemic equilibrium point. The class of infectious humans is the most important class because it represents the persons who may be clinically ill and is directly related to the disease induced deaths. We will calculate the sensitivity indices of the reproductive number, R_0, and the endemic equilibrium point with respect to the parameters given in Table 1 for the model. By the analysis of these indices we could determine which parameter is more crucial for disease transmission and prevalence.

Definition 4.1. The normalized forward sensitivity index of a variable, h, that depends differentiably on a parameter, l, is defined as $\gamma_l^h = (\partial h/\partial l) \times (l/h)$.

Table 2 represents sensitivity indices of model parameters to R_0.

By analyzing sensitivity indices we observe that the most sensitive parameters are biting rate of mosquitoes b and death rate of mosquitoes μ_v. The reproductive number (R_0) is directly related to the biting rate of mosquitoes and inversely related to the death rate

Table 3: The sensitivity indices of the state variables at the endemic equilibrium, x_i, to the parameters, p_j, for parameter values given in Table 1.

	S_h^*	I_h^*	I_v^*
Λ_h	0.998946	1.50275	0.00011
Λ_v	−0.00108296	0.516688	1.45019
b	−0.00401088	1.91363	2.57621
γ_h	0.00314305	−1.49958	−1.30657
β_1	−0.00302661	1.44402	1.25817
β_2	−0.000984278	0.469608	1.31805
β_3	−0.000116517	0.0555912	0.0484363
α_1	0.00194365	−0.927333	−0.80798
α_2	6.33734×10^{-6}	−0.0030236	−0.00263445
α_3	0.0000407045	−0.0194205	−0.0545075
μ_h	−0.998946	−1.50279	−1.30937
μ_v	0.00206723	−0.986295	−2.76823

of mosquitoes. We can say that an increase (or decrease) in biting rate b by 10% increases (or decreases) R_0 by 20%. Similarly increase (or decrease) in death rate of mosquitoes by 10% decreases (or increases) R_0 by 20%. This suggests that strategies that can be applied in controlling the disease are to target the mosquito biting rate and death rate such as the use of insecticide-treated bed nets and indoor residual spray.

4.1. Sensitivity Indices of Endemic Equilibrium

We have numerically calculated the sensitivity indices at the parameter values given in Table 1. The most sensitive parameter for I_h^* is mosquito biting rate. Change in mosquito biting rate is directly related to change in I_h^* and inversely related to change in γ_h. This suggests that personal protection and human treatment strategies can lead to marvelous decrease in I_h^*. The most sensitive parameter for I_v^* is mosquito death rate μ_v, followed by mosquito biting rate. We observe that I_v^* can be reduced by personal protection, larvcide adulticide, and so forth.

The analysis of the sensitivity indices of R_0, I_h^*, and I_v^* suggests us that three controls, personal protection, larvacide, and adulticide and treatment of infectious humans, can play an effective role to control the disease. The sensitivity indices for S_h^*, I_h^*, and I_v^* with respect to all parameters are given in Table 3.

5. Analysis of Optimal Control

In this section, model (2.1) is extended to assess the impact of some control measures, namely, prevention, treatment, and spray of insecticide against vector. In the human population, the associated force of infection is reduced by a factor of $(1 - u_1)$ and the reproduction rate of the mosquito population is reduced by a factor of $(1 - u_3)$. It is assumed that under the successful

control efforts the mortality rate of mosquito population increases at a rate proportional to u_3, where $c > 0$ is a rate constant. The per capita recovery rate is proportional to u_2, where $r > 0$ is a rate constant. One has

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - (1-u_1)\frac{b\beta_1 S_h I_v}{1+\alpha_1 I_v} - \frac{\beta_3 S_h I_h}{1+\alpha_2 I_h} - \mu_h S_h + \gamma_h I_h,\\
\frac{dI_h}{dt} &= (1-u_1)\frac{b\beta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\beta_3 S_h I_h}{1+\alpha_2 I_h} - (\mu_h + \gamma_h + ru_2) I_h,\\
\frac{dS_v}{dt} &= \Lambda_v(1-u_3) - (1-u_1)\frac{b\beta_2 I_h S_v}{1+\alpha_3 I_v} - \mu_v S_v - cu_3 S_v,\\
\frac{dI_v}{dt} &= (1-u_1)\frac{b\beta_2 I_h S_v}{1+\alpha_3 I_v} - \mu_v I_v - cu_3 I_v.
\end{aligned} \tag{5.1}$$

The control variable u_1 represents the use of drugs or vaccine which are preventive measures to minimize vector human contacts. The control function u_2 represents the treatment supplied to the infected humans. The control function u_3 represents the level of larvacide and adulticide used for vector control applied at those places at which vector breeding occurs.

To investigate the optimal level of efforts that would be needed to control the disease, we give the objective functional J, which is to minimize the number of infected human and the cost of applying the control u_1, u_2, u_3. One has

$$J = \int_0^{t_f} \left(A_1 I_h + A_2 I_v + A_3 S_v + \frac{1}{2}\left(B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2 \right) \right) dt, \tag{5.2}$$

where A_1, A_2, and A_3 are positive weights. We choose a quadratic cost on the controls and this is similar with what is in other literature on epidemic controls [15]. With the given objective function $J(u_1, u_2, u_3)$, our goal is to minimize the number of infected humans, while minimizing the cost of control $u_1(t)$, $u_2(t)$, and $u_3(t)$. We seek an optimal control u_1^*, u_2^*, u_3^* such that

$$J\left(u_1^*, u_2^*, u_3^*\right) = \min\{ J(u_1, u_2, u_3) \mid u_1, u_2, u_3 \in U \}, \tag{5.3}$$

where $U = \{(u_1, u_2, u_3)$ such that u_1, u_2, u_3 measurable with $0 \le u_1 \le 1,\ 0 \le u_2 \le 1,\ 0 \le u_3 \le 1\}$ is the control set. The necessary conditions that an optimal must satisfy come from the

Pontryagin's Maximum Principle [24]. This principle converts (5.1) and (5.2) into a problem of minimizing pointwise a Hamiltonian H, with respect to u_1, u_2, and u_3

$$\begin{aligned} H = {} & A_1 I_h + A_2 I_v + A_3 S_v + \frac{1}{2}\left(B_1 u_1^2 + B_2 u_2^2 + B_3 u_3^2\right) \\ & + \lambda_1\left(\Lambda_h - (1-u_1)\frac{b\beta_1 S_h I_v}{1+\alpha_1 I_v} - \frac{\beta_3 S_h I_h}{1+\alpha_2 I_h} - \mu_h S_h + \gamma_h I_h\right) \\ & + \lambda_2\left((1-u_1)\frac{b\beta_1 S_h I_v}{1+\alpha_1 I_v} + \frac{\beta_3 S_h I_h}{1+\alpha_2 I_h} - (\mu_h + \gamma_h + ru_2) I_h\right) \\ & + \lambda_3\left(\Lambda_v(1-u_3) - (1-u_1)\frac{b\beta_2 I_h S_v}{1+\alpha_3 I_v} - \mu_v S_v - cu_3 S_v\right) \\ & + \lambda_4\left((1-u_1)\frac{b\beta_2 I_h S_v}{1+\alpha_3 I_v} - \mu_v I_v - cu_3 I_v\right), \end{aligned} \tag{5.4}$$

where λ_i for $i = 1, \ldots, 4$ are adjoint variables. In the following we will state and prove the existence of the optimal control by using the result by Fleming and Rishel [25].

Theorem 5.1. *There exists an optimal control (u_1^*, u_2^*, u_3^*) that minimize J over U subject to the control system* (5.1). *Further, for the system* (5.1), *there exists adjoint variables λ_i, $i = 1, \ldots, 4$ satisfying*

$$\begin{aligned} \frac{d\lambda_1}{dt} &= (\lambda_1 - \lambda_2) b\beta_1 (1-u_1)\frac{I_v}{1+\alpha_1 I_v} + (\lambda_1 - \lambda_2)\beta_3 \frac{I_h}{1+\alpha_2 I_h} + \lambda_1 \mu_h, \\ \frac{d\lambda_2}{dt} &= (\mu_h + \gamma_h + ru_2)\lambda_2 - \lambda_1 \gamma_h + (\lambda_1 - \lambda_2)\beta_3 \frac{S_h}{(1+\alpha_2 I_h)^2} + (\lambda_3 - \lambda_4)(1-u_1) b\beta_2 \frac{S_v}{1+\alpha_3 I_v} - A_1, \\ \frac{d\lambda_3}{dt} &= (\lambda_3 - \lambda_4)(1-u_1) b\beta_2 \frac{I_h}{1+\alpha_3 I_v} + (\mu_v + cu_3)\lambda_3 - A_3, \\ \frac{d\lambda_4}{dt} &= (\lambda_1 - \lambda_2) b\beta_1 (1-u_1)\frac{S_h}{(1+\alpha_1 I_v)^2} + (\lambda_4 - \lambda_3)(1-u_1)\alpha b\beta_2 \frac{I_h S_v}{(1+\alpha_3 I_v)^2} + (\mu_v + cu_3)\lambda_4 - A_2, \end{aligned} \tag{5.5}$$

with transversality conditions $\lambda_i(t_f) = 0$, $i = 1, \ldots, 4$. The optimal controls are given by

$$\begin{aligned} u_1^* &= \min\left\{\max\left\{0, \frac{1}{B_1}\left[(\lambda_2 - \lambda_1) b\beta_1 \frac{S_h I_v}{1+\alpha_1 I_v} + (\lambda_4 - \lambda_3)\frac{b\beta_2 I_h S_v}{1+\alpha_3 I_v}\right]\right\}, 1\right\}, \\ u_2^* &= \min\left\{\max\left\{0, \frac{1}{B_2}[\lambda_2 r I_h]\right\}, 1\right\}, \\ u_3^* &= \min\left\{\max\left\{0, \frac{1}{B_3}[\lambda_3(\Lambda_v + cS_v) + \lambda_4 c I_v]\right\}, 1\right\}. \end{aligned} \tag{5.6}$$

Proof. The integrand of the objective functional J given by (5.2) is a convex function of u_1, u_2, u_3 and the state system satisfies the Lipschitz property with respect to the state variables since state solutions are bounded. The existence of an optimal control follows [25]. The equations governing the adjoint variables are obtained by differentiation of the Hamiltonian function with respect to S_h, I_h, S_v, and I_v, respectively, evaluated at the optimal control. To get the characterization of the optimal control given by (5.6), solving the equations

$$\frac{\partial H}{\partial u_1} = 0, \qquad \frac{\partial H}{\partial u_2} = 0, \qquad \frac{\partial H}{\partial u_3} = 0, \tag{5.7}$$

on the interior of the control set and using the property of the control space U, we can derive the desired characterization (5.6). □

6. Numerical Results and Discussions

Here, we investigate numerically the effect of the optimal control strategies on the spread of the disease in a population.

6.1. Use of Preventive Measures ($u_1 \neq 0$) and Treatment ($u_2 \neq 0$)

With this strategy, only the preventive control u_1 on the vector biting rate and treatment u_2 is used to optimize the objective function J, while the control u_3 on reducing the vector population is set to zero. Figure 1 shows no significant difference in the number of infected mosquitoes I_v between the case with control and the case without while there is a significant difference in the number of infected humans I_h with control and the case without control. The control profile is shown in Figure 1(d); the control u_1 rise to the upper bound after 400 (days), while control u_2 rise to the upper bound after 50 (days). This shows that an effective and optimal use of preventive measure in the population without the use of larvacide against the vector will not be beneficial if total elimination of the disease is desirable in the community.

6.2. Use of Preventive Measures ($u_1 \neq 0$) and Larvacide Only ($u_3 \neq 0$)

With this strategy, we set the control u_2 to zero and use only preventive control u_1 and larvacide u_3 to optimize the objective function J. In Figures 2(a) and 2(b) show a significant difference in the infected humans I_h and infected mosquitoes I_v, respectively, with control compared to the situation where there is no control. More specifically, we observe a decrease in I_h and I_v while an increase was observed in the uncontrolled cases. The control profile is shown in Figure 2(d), where we see that the control u_3 is at the upper bound for $t = 450$ (days).

6.3. Use of Treatment ($u_2 \neq 0$) and Larvacide ($u_3 \neq 0$)

With this strategy, we set control u_1 to zero, while the control u_2 and the control u_3 are both used to optimize the objective function J. In Figures 3(a) and 3(b), we observed that

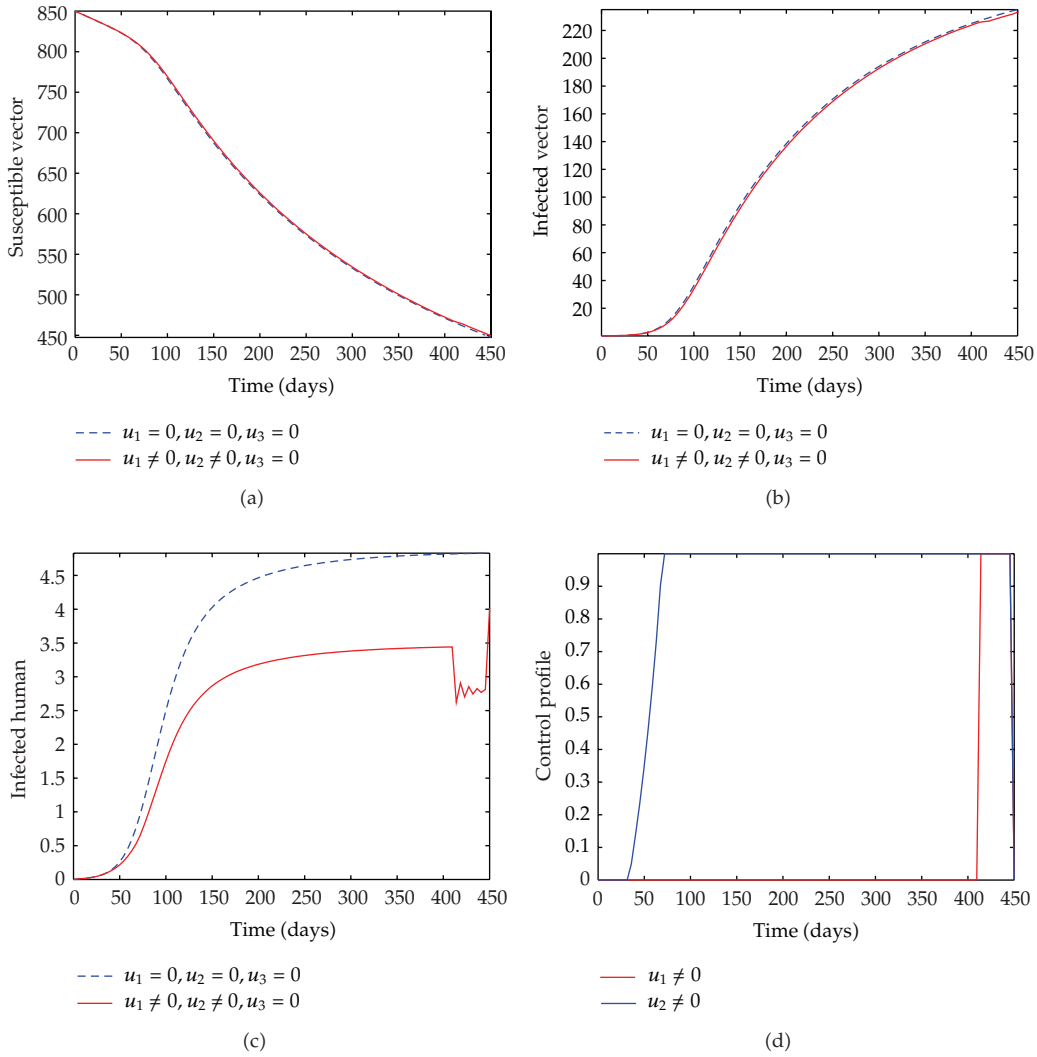

Figure 1: Simulations of the malaria model showing the effect of control strategies on malaria transmission.

the control strategies resulted in a decrease in the number of infected humans (I_h), infected mosquitoes (I_v), and susceptible humans (S_h) while an increase is observed in the number of infected humans (I_h) and infected mosquitoes (I_v) in strategy without control. The control profile is shown in Figure 3(d), where we see that the control u_3 is at the upper bound for $t = 450$ (days) and the control u_2 is at the upper bound for $t = 440$ days and then dropped to the lower bound at the final time.

6.4. *Use of Preventive Measures* ($u_1 \neq 0$)*, Treatment* ($u_2 \neq 0$)*, and Larvacide* ($u_3 \neq 0$)

With this strategy, the control u_1, control u_2, and the control u_3 are all used to optimize the objective function J. In Figures 4(a) and 4(b), we observed that the control strategies

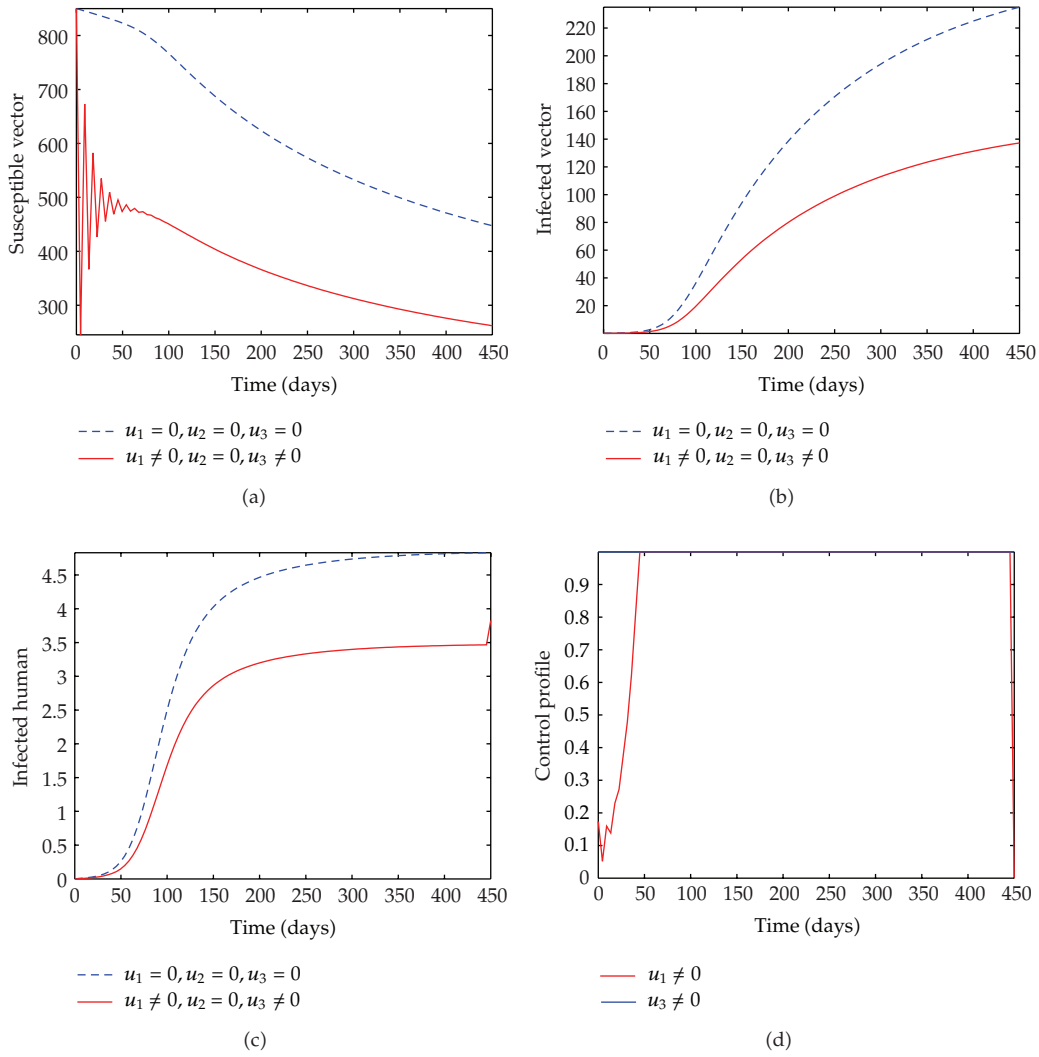

Figure 2: Simulations of the malaria model showing the effect of control strategies on malaria transmission.

resulted in a decrease in the number of infected humans (I_h), infected mosquitoes, (I_v) and susceptible humans (S_h) while an increase is observed in the number of infected humans (I_h) and infected mosquitoes (I_v) in strategy without control. The control profile is shown in Figure 4(d), where we see that the control u_1 is at the upper bound for $t = 440$ (days) before dropping gradually to the lower bound, the control u_2 is at the upper bound for $t = 440$ (days) and then dropped to the lower bound at the final time, and the control u_3 is maintained at the upper bound for $t = 450$ (days).

7. Conclusion

In this paper, we have studied a vector host epidemic model with saturated incidence rate. The global stability of the disease-free steady state is established by direct Lyapunov

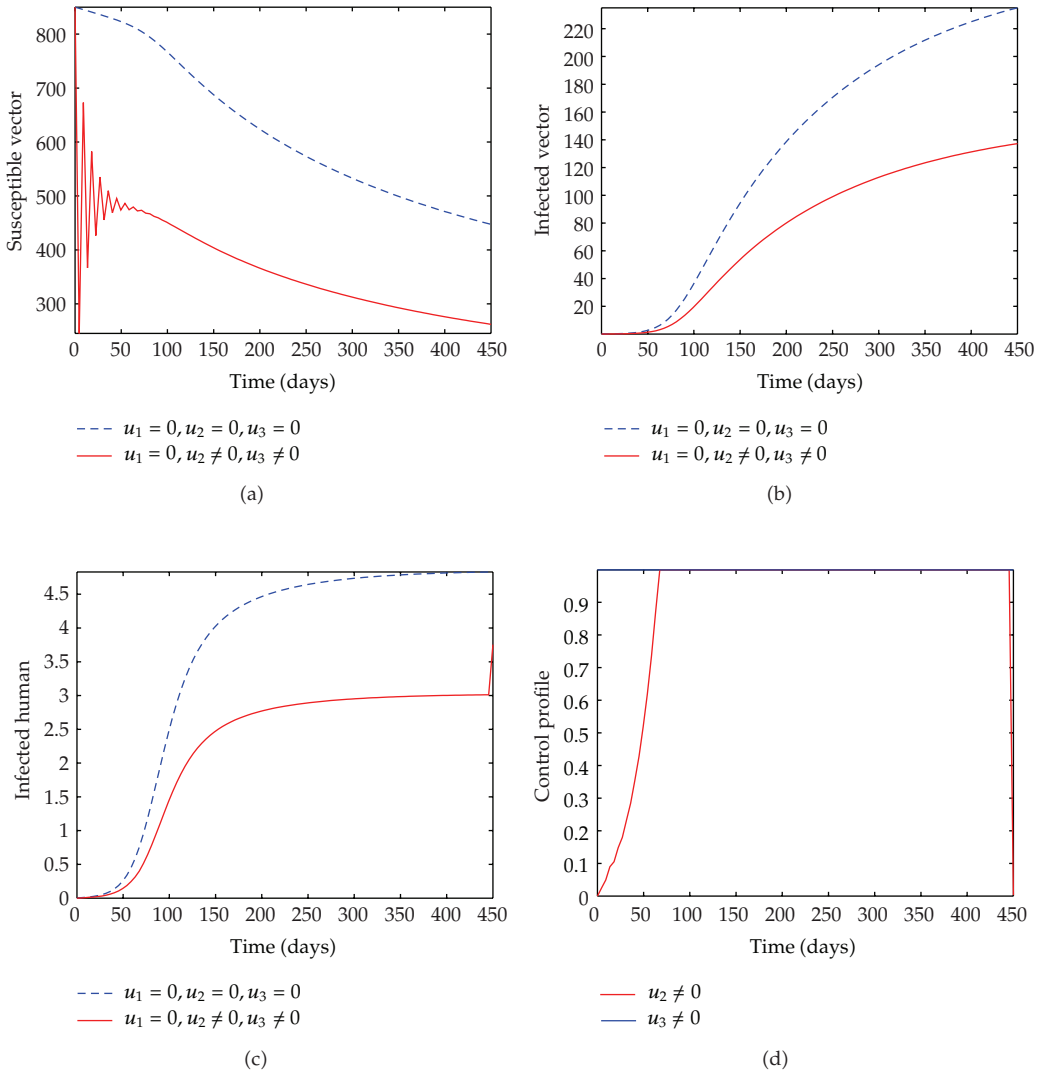

Figure 3: Simulations of the malaria model showing the effect of control strategies on malaria transmission.

method and a geometric approach is used for the global stability of the endemic steady state. The model has a globally asymptotically stable disease-free solution whenever the basic reproduction number R_0 is less than or equal unity and has a unique positive globally asymptotically stable endemic equilibrium whenever R_0 exceeds unity. We found also from the sensitivity indices analysis that the most sensitive parameters are mosquito biting and death rates. The paper was also extended to asses the impact of some control measures. By the application of optimal control theory, we derived and analyzed the conditions for optimal control of the disease with personal protection, treatment and spray of insecticides. The optimal control has a very desirable effect for reducing the number of infected individuals and comparison between optimal control and without control is shown in figures. From our numerical results we found that an effective and optimal use of preventive measure in the population without the use of larvacide against the vector will not be beneficial

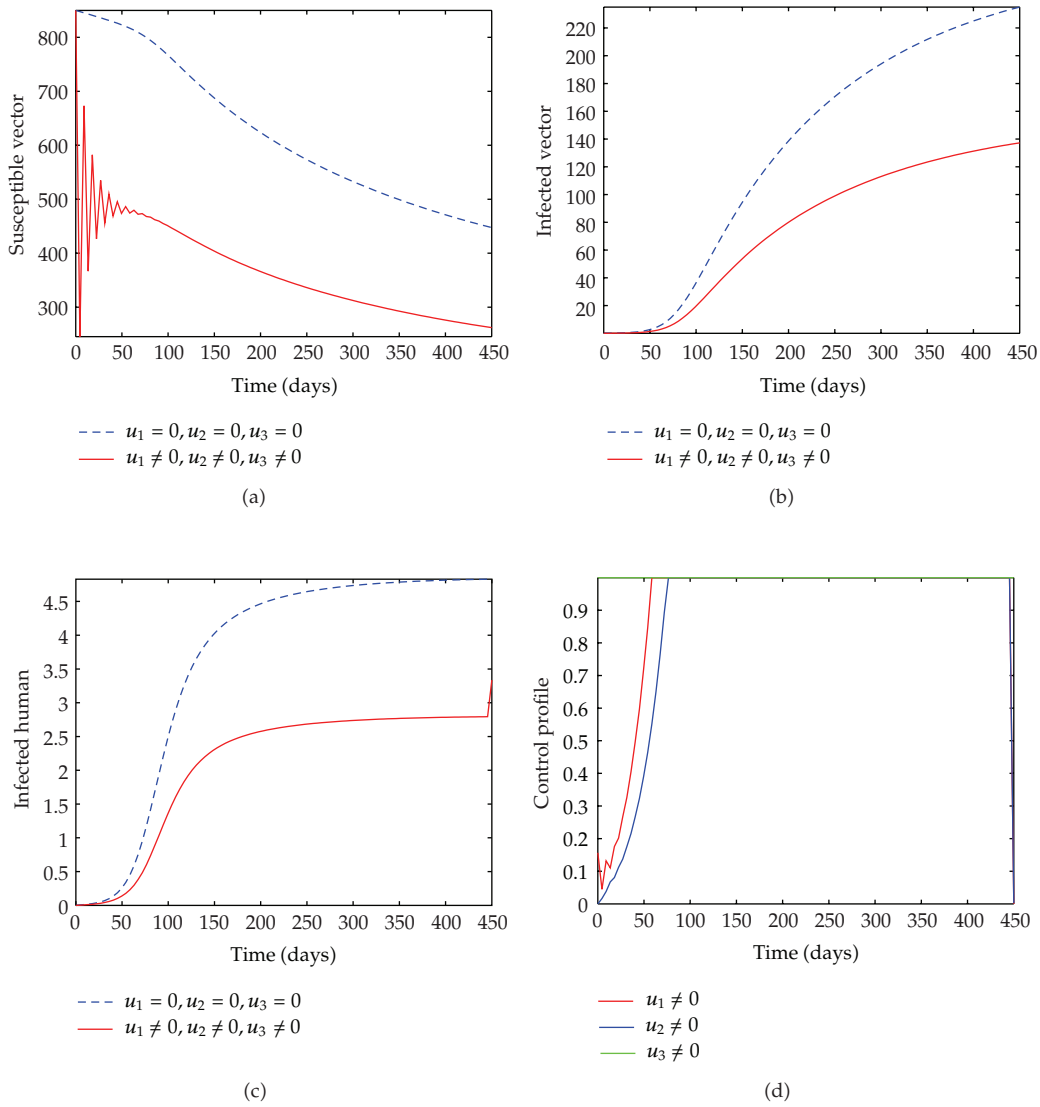

Figure 4: Simulations of the malaria model showing the effect of control strategies on malaria transmission.

if total elimination of the disease is desirable in the community. Control programs that follow these strategies can effectively reduce the spread of a vector-borne disease in the community.

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