

Research Article

Modeling and Stability Analysis of the Dynamics of Malaria Disease Transmission with Some Control Strategies

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In this study, we proposed and analyzed a nonlinear deterministic mathematical model of malaria transmission dynamics. In addition to the previous approaches, we incorporated the class of aware people and other control measures. We established the wellposedness of the model, and the asymptotic behavior of the solutions is rigorously studied depending on the basic reproduction number R_0 . The model system admits two equilibrium points: disease-free and disease-persistent equilibrium points. The analytical result of the model system revealed that the disease-free equilibrium point is both locally as well as globally asymptotically stable whenever $R_0 < 1$ while the disease-persistence equilibrium point is globally asymptotically stable whenever $R_0 > 1$. Moreover, the forward bifurcation phenomenon of the model system for $R_0 = 1$ was analyzed by using center manifold theory. A sensitivity analysis of the basic reproduction number was performed to identify parameters that will cause to trigger the transmission of malaria disease and should be targeted by control strategies. Then, the model was extended to the optimal control problem, with the use of three time-dependent controls, namely, preventive measures (treated bednets and indoor residual spraying), continuous awareness campaigns to susceptible individuals, and treatment for infected individuals. By using Pontryan's maximum principle, necessary conditions for the transmission of malaria disease were derived. Numerical simulations are illustrated by using MATLAB ode45 to validate the theoretical results of the model. The numerical findings of the optimal model suggested that integrated control strategies are better than a sole intervention to eliminate malaria disease.

1. Introduction

Nowadays, developing countries are challenging with a lot of problems, such as climatic change, racism, terrorism, unemployment, poverty, and public health crises. Among them, malaria disease is continued as a killer death and a major public health issue [1]. It is a contagious illness and spreads between people through the bites of infected female *Anopheles* mosquitoes [2, 3]. The most common species that can cause to human illness are *Plasmodium vivax*, *Plasmodium falciparum*, *Plasmodium ovale*, *Plasmodium malariae*, and *Plasmodium knowlesi*, and their distribution may vary from place to place [1]. The most common symptoms of malaria include high fever, joint pains, abdominal pains, headaches, vomiting, nausea, diarrhea, and extreme fatigue [3, 4, 5, 6, 7]. Approximately 241 million cases and 627,000 deaths were reported globally in 2020, with 95% of cases occurring in the African Region [8]. Utilizing bed nets that have been treated, antimalarial drugs,

and indoor residual spraying are among the most effective malaria prevention measures [9].

A thorough understanding of the disease's transmission process and its control is necessary to create effective intervention measures and mitigations. Mathematical models help in describing the evolution of infectious diseases [10, 11, 12, 13, 14, 15, 16, 17, 18]. The first published paper on the dynamics of malaria that demonstrates the life cycle of the malaria parasite dates back to Sir Ross [19], which is SIR type. Recently, various scholars have studied the dynamics of malaria transmission using mathematical modeling approaches to combat it. For example, in 2022, Collins and Duffy [20] proposed and analyzed the dynamics of the malaria transmission model in Nigeria that takes into account drug resistance, treatment, and the use of mosquito nets as preventative measures. Their findings confirmed that unless better control strategies are put into place, the disease is likely to remain endemic in Nigeria. In 2021, Keno et al. [21] developed and analyzed a mathematical

model of malaria transmission with optimal controls. Their study showed that the most cost-effective ways to reduce malaria transmission are through the combination of control measures, such as treatment and indoor residual spraying. In 2020, Ghosh et al. [17] formulated and analyzed SEIRS for human populations and SEI for vector populations with optimal control and relapse. Their result revealed that treated bed nets and indoor residue sprays are the best strategies to eliminate malaria transmission. In 2018, Leiton et al. [22] presented an SEIRS-SI mathematical model for malaria transmission with optimal control in Colombia. Their finding concluded that to prevent malaria transmission the integrated control strategies must hold effectively. In 2022, Keno et al. [23] presented a nonlinear mathematical model of malaria transmission with three control strategies, namely treated bed nets, treatment, and indoor residual spraying. Both forward and backward bifurcation were present in their model, and (treatment and treated bed nets) are the best ways to reduce the burden of malaria transmission among control measures. In 2022, Gautam et al. [24] developed a mathematical model for malaria transmission dynamics that included cross-border travel between a low-endemic country (Nepal) and a high-endemic country (India) with optimal control. They reaffirmed that their study offers crucial insights into a potential barrier that cross-border mobility may pose to malaria elimination programs. Even though most countries have policies or strategies that are being made to eradicate malaria disease, it is continued as a challenge, especially for developing countries. For the best of our knowledge, mathematical models that deal with the impact of awareness creation for susceptible individuals are very rare. Thus, we are motivated to fill this gap and consider other alternative control measures.

The remaining parts of this study are outlined as follows: The model description and underlying assumptions are covered in Section 2. The basic properties of the model are discussed in Section 3. An analysis of the model is given in Section 4. The optimal control problem is presented in Section 5. Numerical simulations and discussions of the model are covered in Section 6. The conclusion is provided in Section 7.

2. Mathematical Model Formulation and Description

The model formulation is adopted from [25] by making some modifications by adding the class of aware individuals compartment for controlling malaria disease, and it makes the entire of human population to be divided into four epidemiological compartments, namely, susceptible individuals $S_h(t)$; aware susceptible individuals $A_h(t)$; infectious individuals $I_h(t)$; and recovered individuals $R_h(t)$. Furthermore, the total number of mosquitoes is classified into two groups: susceptible mosquitoes $S_m(t)$ and infected mosquitoes $I_m(t)$. The following assumptions are taken into account in the formulation of our model.

- (i) We assumed that all newly recruited humans and mosquitoes are not carriers of malaria infection.

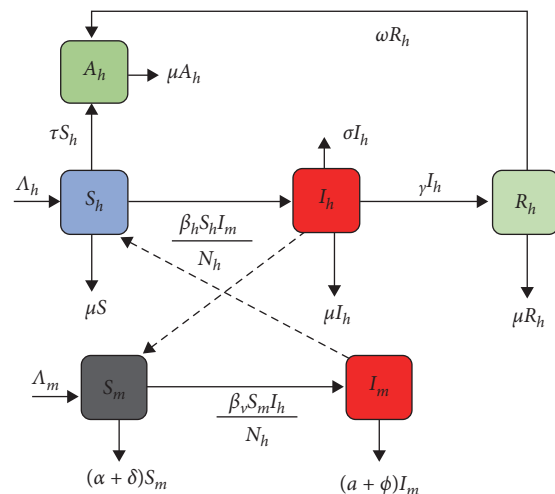


FIGURE 1: The flowchart of malaria transmission model.

- (ii) We considered only female anopheles mosquitoes in our recent study.
- (iii) In addition to natural death, we assumed that other human activities contribute for the mortality of mosquitoes.
- (iv) We assumed that mosquitoes feed only human blood, and vertical transmission of the infection between them is not under consideration.
- (v) We assumed that infected mosquitoes remain infectious until they die.

Keeping the above discussions in mind, the new model is portrayed in Figure 1, and state variables and parameters are presented in Tables 1 and 2, respectively.

The governing nonlinear differential equations of the model, as per the flowchart, are as follows:

$$\dot{S}_h = \Lambda_h - \frac{\beta_h S_h I_m}{N_h} - (\mu + \tau) S_h, \quad (1a)$$

$$\dot{A}_h = \tau S_h - \mu A_h + \omega R_h, \quad (1b)$$

$$\dot{I}_h = \frac{\beta_h S_h I_m}{N_h} - (\sigma + \mu + \gamma) I_h, \quad (1c)$$

$$\dot{R}_h = \gamma I_h - (\omega + \mu) R_h, \quad (1d)$$

$$\dot{S}_m = \Lambda_m - \frac{\beta_m S_m I_h}{N_h} - (\alpha + \delta) S_m, \quad (1e)$$

$$\dot{I}_m = \frac{\beta_m S_m I_h}{N_h} - (\alpha + \phi) I_m, \quad (1f)$$

TABLE 1: State variables of the model and its description.

Notation	Description
$S_h(t)$	The class of susceptible human population at given time t
$A_h(t)$	The class of aware human population at a given time t
$I_h(t)$	The class of infectious human population at a given time t
$R_h(t)$	The class of recovered human population at a given time t
$S_m(t)$	The class of susceptible female Anopheles mosquitoes at a given time t
$I_m(t)$	The class of infectious female Anopheles mosquitoes at a given time t

TABLE 2: Model parameters and its description.

Notation	Description
Λ_h	The recruitment rate of susceptible individuals
Λ_m	The recruitment rate of susceptible female Anopheles mosquitoes population
τ	The progression rate of susceptible individuals into the class of aware individuals
β_h	The rate in which susceptible humans get infected by infected mosquitoes
β_m	The rate in which susceptible mosquitoes get infected by infectious individuals
μ	The natural death rate of individuals
α	The natural death rate of mosquitoes
σ	The disease induced-death rate of infectious individuals
ω	The progression rate of recovered individuals to the class of aware individuals
γ	The recovery rate of infectious individuals
δ	The mortality rate of susceptible mosquitoes population due to human activities
ϕ	The mortality rate of infected mosquitoes population due to human activities

with initial conditions:

$$\begin{aligned}
 S_h(0) &= S_{h0} > 0, \\
 A_h(0) &= A_{h0} > 0, \\
 I_h(0) &= I_{h0} \geq 0, \\
 R_h(0) &= R_{h0} \geq 0, \\
 S_m(0) &= S_{m0} > 0, \\
 I_m(0) &= I_{m0} \geq 0,
 \end{aligned}
 \tag{2}$$

$$\dot{x} = \begin{bmatrix} S_h \\ A_h \\ I_h \\ R_h \\ S_m \\ I_m \end{bmatrix}, f(x) = \begin{bmatrix} \Lambda_h - \frac{\beta_h S_h I_m}{N_h} - (\mu + \tau) S_h, \\ \tau S_h - \mu A_h + \omega R_h \\ \frac{\beta_h S_h I_m}{N_h} - (\sigma + \mu + \gamma) I_h, \\ \gamma I_h - (\omega + \mu) R_h, \\ \Lambda_m - \frac{\beta_m S_m I_h}{N_h} - (\alpha + \delta) S_m, \\ \frac{\beta_m S_m I_h}{N_h} - (\alpha + \phi) I_m, \end{bmatrix}.
 \tag{4}$$

and

$$\begin{aligned}
 N_h(t) &= S_h(t) + A_h(t) + I_h(t) + R_h(t), \\
 N_m(t) &= S_m(t) + I_m(t).
 \end{aligned}
 \tag{3}$$

3. Basic Properties of the Model

Lemma 1 (Existence and Uniqueness of Model Solutions). *The unique solution in $C(\mathbb{R}^+, \mathbb{R}_+^6)$, $\forall t \geq 0$, is admissible for the model system (1) with initial condition (2).*

Proof. we can rewrite the system (1) in the form of $\dot{x} = f(x)$, where

It is straightforward to see that all the right-hand-side components of the function $f(x)$ are continuously differentiable almost everywhere in $C(\mathbb{R}^+, \mathbb{R}_+^6)$, which implies that $f(x)$ is of a class C^1 on \mathbb{R}_+^6 for all $t \geq 0$. Hence, by Picard–Lindelöf theorem [26], the model system (1) has a unique solution locally in \mathbb{R}_+^6 for all $t \geq 0$. This completes the proof. \square

Since we are dealing with populations, it is necessary to ensure the nonnegativity and boundedness of model solutions.

Lemma 2 (Nonnegativity of Model Solutions). *The solution set $\{S_h(t), A_h(t), I_h(t), R_h(t), S_m(t), I_m(t)\}$ of model system (1) remains nonnegative for all future time, $t \geq 0$ under the initial condition (2) in the closed region $\Delta \subset \mathbb{R}_+^6$.*

Proof. Consider the 1st equation in the system (1),

$$\dot{S}_h = \Lambda_h - \frac{\beta_h S_h I_m}{N_h} - (\mu + \tau) S_h. \quad (5)$$

Then, after solving for $S_h(t)$, we obtained the following:

$$S_h(t)\eta(t) = S_h(0) + \Lambda_h \int_0^t \eta(\varepsilon) d\varepsilon, \quad (6)$$

for all time $t \geq 0$, where the function $\eta(t)$ is the integrating factor given as follows:

$$\eta(t) = \exp\left(\int_0^t \frac{\beta_h I_m(\varepsilon)}{N_h(\varepsilon)} - (\mu + \tau) d\varepsilon\right). \quad (7)$$

From Equation (6), it is clear that the function $S_h(t)$ is positive for all $t \geq 0$, since $\eta(t) > 0$.

Likewise, consider the 5th equation in the system (1)

$$\dot{S}_m = \Lambda_m - \frac{\beta_m S_m I_h}{N_h} - (\alpha + \delta) S_m. \quad (8)$$

Then, after solving Equation (9), the result can be implicitly expressed as follows:

$$S_m(t)\kappa(t) = S_m(0) + \Lambda_m \int_0^t \kappa(\varepsilon) d\varepsilon, \quad (9)$$

for all time $t \geq 0$, where the function $\kappa(t)$ is the integrating factor given by the following:

$$\kappa(t) = \exp\left(\int_0^t \frac{\beta_m I_h(\varepsilon)}{N_h(\varepsilon)} - (\alpha + \delta) d\varepsilon\right). \quad (10)$$

Hence, from Equation (9), the function $S_m(t)$ is also positive for all time $t \geq 0$. It can be shown in similar way that: $A_h \geq 0$, $I_h \geq 0$, $R_h \geq 0$ and $I_m \geq 0$. Therefore, all the solutions that start in the positive region remain positive. \square

Lemma 3 (Boundedness of Model Solutions). *The malaria model (1) with initial condition (2) in \mathbb{R}_+^6 is positively invariant in the feasible region $\Delta = \Delta_h \times \Delta_v \subset \mathbb{R}_+^4 \times \mathbb{R}_+^2$, where*

$$\begin{aligned} \Delta_h &= \left\{ (S_h(t), A_h(t), I_h(t), R_h(t)) \in \mathbb{R}_+^4 : S_h(t) + A_h(t) + I_h(t) + R_h(t) \leq \frac{\Lambda_h}{\mu} \right\}, \\ \Delta_m &= \left\{ (S_m(t), I_m(t)) \in \mathbb{R}_+^2 : S_m(t) + I_m(t) \leq \frac{\Lambda_m}{\alpha} \right\}. \end{aligned} \quad (11)$$

Proof. Adding all the model equations of (1), we obtain the following:

$$\begin{aligned} \dot{N}_h(t) &= \dot{S}_h(t) + \dot{A}_h(t) + \dot{I}_h(t) + \dot{R}_h(t) \\ \Rightarrow \dot{N}_h(t) &= \Lambda_h - \mu N_h - \sigma I_h. \end{aligned} \quad (12)$$

The positivity of model equations (1) helps us to arrive from

$$\dot{N}_h(t) \leq \Lambda_h - \mu N_h \Rightarrow \dot{N}_h(t) + \mu N_h \leq \Lambda_h. \quad (13)$$

Solving the differential inequality, we obtained the relation as follows:

$$N_h(t) \leq \frac{\Lambda_h}{\mu} + \left[N_h(0) - \frac{\Lambda_h}{\mu} \right] e^{-\mu t}. \quad (14)$$

If $N_h(0) \leq \frac{\Lambda_h}{\mu}$, then we can claim that $N_h(t) \leq \frac{\Lambda_h}{\mu}$ for all $t \geq 0$. This confirms that the feasible solution set of the human population is given by the following:

$$\Delta_h = \{ (S_h, A_h, I_h, R_h) \in \mathbb{R}_+^4 : N_h(0) \leq N_h(t) \leq \frac{\Lambda_h}{\mu} \}.$$

Similarly, the time derivative of the total mosquito population, $N_m(t)$ along model solutions (1), is obtained and given in compact form by the following:

$$\Delta_m = \left\{ (S_m, I_m) \in \mathbb{R}_+^2 : N_m(0) \leq N_m(t) \leq \frac{\Lambda_m}{\alpha} \right\}. \quad (15)$$

Therefore, the proof of the theorem is already completed. \square

4. Analytical Analysis of the Model

4.1. Malaria Disease Free-Equilibrium (DFE) Point. To obtain the DFE, we set the right-hand sides of model system (1) to zero at $I_h = I_m = 0$. Thus, the DFE point of the model is denoted by D_f and given by the following:

$$D_f = (S_h^*, A_h^*, I_h^*, R_h^*, S_m^*, I_m^*) = \left(\frac{\Lambda_h}{\mu + \pi}, \frac{\tau \Lambda_h}{\mu(\mu + \tau)}, 0, 0, \frac{\Lambda_m}{\alpha + \delta}, 0 \right).$$

4.2. Basic Reproduction Number. This section devotes to find the basic reproductive number R_0 , which is an important ratio used to predict the asymptotic behavior of the disease distribution. It is defined as the average number of new cases resulted when a single infection is introduced into completely susceptible populations. We compute R_0 based on [27, 28] and denoting the vector for the newly infected by N and the rate of transfer by T where

$$N = \begin{bmatrix} \frac{\beta_h S_h I_m}{N_h} \\ \frac{\beta_m S_m I_h}{N_h} \end{bmatrix}, T = \begin{bmatrix} (\sigma + \mu + \gamma) I_h \\ (\alpha + \phi) I_m \end{bmatrix}. \quad (16)$$

The Jacobian matrix of N and T at DFE point D_f are given by F and V , where

$$F = \begin{bmatrix} 0 & \frac{\beta_h \mu}{\mu + \tau} \\ \frac{\beta_m \Lambda_m \mu}{\Lambda_h (\alpha + \delta)} & 0 \end{bmatrix}, V = \begin{bmatrix} (\sigma + \mu + \gamma) & 0 \\ 0 & (\alpha + \phi) \end{bmatrix}. \quad (17)$$

Then, the inverse of V is computed and given by the following:

$$V^{-1} = \begin{bmatrix} \frac{1}{(\sigma + \gamma + \mu)} & 0 \\ 0 & \frac{1}{(\alpha + \phi)} \end{bmatrix}. \quad (18)$$

Finally, the next-generation matrix is given by the following:

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\beta_h \mu}{(\mu + \tau)(\alpha + \phi)} \\ \frac{\beta_m \Lambda_m \mu}{\Lambda_h (\alpha + \delta)(\sigma + \gamma + \mu)} & 0 \end{bmatrix}. \quad (19)$$

The two eigenvalues of FV^{-1} are as follows:

$$\lambda_1 = \sqrt{\frac{\beta_h \beta_m \Lambda_m \mu^2}{\Lambda_h (\tau + \mu)(\alpha + \delta)(\alpha + \phi)(\sigma + \gamma + \mu)}}, \quad (20)$$

$$\lambda_2 = -\sqrt{\frac{\beta_h \beta_m \Lambda_m \mu^2}{\Lambda_h (\tau + \mu)(\alpha + \delta)(\alpha + \phi)(\sigma + \gamma + \mu)}}.$$

Indeed, the spectral radius of FV^{-1} is as follows:

$$R_0 = \max\{\lambda_1, \lambda_2\} = \sqrt{\frac{\beta_h \beta_m \Lambda_m \mu^2}{\Lambda_h (\tau + \mu)(\alpha + \delta)(\alpha + \phi)(\sigma + \gamma + \mu)}}. \quad (21)$$

4.3. Malaria Disease Persistent Equilibrium Point of the Model.

The disease persistent equilibrium point of the model system (1) is denoted by D_p with $D_p = (S_h^{**}, A_h^{**}, I_h^{**}, R_h^{**}, S_m^{**}, I_m^{**})$. To find the components of D_p , we take all the right hand-sides of system (1) equal to zero at $I_h \neq 0, I_m \neq 0$ as follows:

$$\begin{cases} \Lambda_h - \frac{\beta_h S_h I_m}{N_h} - (\mu + \tau) S_h = 0, \\ \tau S_h - \mu A_h + \omega R_h = 0, \\ \frac{\beta_h S_h I_m}{N_h} - (\sigma + \mu + \gamma) I_h = 0, \\ \gamma I_h - (\omega + \mu) R_h = 0, \\ \Lambda_m - \frac{\beta_m S_m I_h}{N_h} - (\alpha + \delta) S_m = 0, \\ \frac{\beta_m S_m I_h}{N_h} - (\alpha + \phi) I_m = 0. \end{cases} \quad (22)$$

From the first equation of system (1), we have that

$$S_h^{**} = \frac{N_h \Lambda_h}{\beta_h I_m^{**} + k_1 N_h}, \quad (23)$$

and also from the fifth equation of system (1), we have the following:

$$S_m^{**} = \frac{\Lambda_m N_h}{\beta_m I_h^{**} + k_4 N_h}. \quad (24)$$

Then, substituting Equation (24) into the sixth equation of system (1) yields

$$I_m^{**} = \frac{\beta_m \Lambda_m I_h^{**}}{k_5 (\beta_m I_h^{**} + k_4 N_h)}. \quad (25)$$

At the end, substituting Equations (23) and (25) into the third equation of system (1) and solving for I_h^{**} results

$$I_h^{**} = \frac{\beta_m \Lambda_h^2 - k_1 k_2 k_4 k_5 N_h}{k_2 \beta_m \Lambda_m + k_1 k_2 k_5 \beta_m}, \quad (26)$$

provided that

$$\beta_m \Lambda_h^2 > k_1 k_2 k_4 k_5 N_h. \quad (27)$$

The remaining components of D_p are calculated based on the expression I_h^{**} as follows:

$$R_h^{**} = \frac{\gamma I_h^{**}}{k_3},$$

$$A_h^{**} = \frac{1}{\mu} \left[\frac{\tau N_h \Lambda_h k_5 (\beta_m I_h^{**} + k_4 N_h)}{\beta_h \beta_m \Lambda_m I_h^{**} + k_1 k_5 N_h (\beta_m I_h^{**} k_4 N_h)} + \frac{\omega \gamma I_h^{**}}{k_3} \right], \quad (28)$$

where

$$\begin{aligned} k_1 &= (\mu + \tau), \\ k_2 &= (\gamma + \mu + \sigma), \\ k_3 &= (\mu + \omega), \\ k_4 &= (\alpha + \delta), \\ k_5 &= \alpha + \phi. \end{aligned} \quad (29)$$

4.4. Stability Analysis of Model Equilibrium Points. In the subsequent theorems, we will examine the obtained equilibria,

$$J(D_f) = \begin{bmatrix} -(\mu + \tau) & 0 & 0 & 0 & 0 & \frac{-\beta_h \mu}{\mu + \tau} \\ \tau & -\mu & 0 & \omega & 0 & 0 \\ 0 & 0 & -(\sigma + \gamma + \mu) & 0 & 0 & \frac{\beta_h \mu}{\mu + \tau} \\ 0 & 0 & \gamma & -(\mu + \omega) & 0 & 0 \\ 0 & 0 & \frac{-\beta_m \Lambda_m \mu}{\Lambda_h(\alpha + \delta)} & 0 & -(\alpha + \delta) & 0 \\ 0 & 0 & \frac{\beta_m \Lambda_m \mu}{\Lambda_h(\alpha + \delta)} & 0 & 0 & -(\alpha + \phi) \end{bmatrix}. \quad (30)$$

The characteristics equation $|J(D_f) - \lambda I_{2 \times 2}| = 0$ admits four eigenvalues:

$$\begin{aligned} \lambda_1 &= -\mu < 0, \\ \lambda_2 &= -(\mu + \tau) < 0, \\ \lambda_3 &= -(\alpha + \delta) < 0, \\ \lambda_4 &= -(\mu + \omega) < 0, \end{aligned} \quad (31)$$

and the remaining eigenvalues of $J(D_f)$ are determined from the submatrix

$$J_5(D_f) = \begin{bmatrix} -(\sigma + \gamma + \mu) & \frac{\beta_h \mu}{\mu + \tau} \\ \frac{\beta_m \Lambda_m \mu}{\Lambda_h(\alpha + \delta)} & -(\alpha + \phi) \end{bmatrix}. \quad (32)$$

The characteristic polynomial of $J_5(D_f)$ is expressed by the following:

$$\begin{aligned} \varphi(\lambda) &= \lambda^2 + ((\gamma + \mu + \sigma) + (\alpha + \phi))\lambda \\ &\quad + (\gamma + \mu + \sigma)(\alpha + \phi) - \frac{\beta_h \beta_m \Lambda_m \mu^2}{(\tau + \mu)(\alpha + \delta)\Lambda_h} \\ &= A_0 \lambda^2 + A_1 \lambda + A_2 = 0, \end{aligned} \quad (33)$$

which are D_f and D_p of model system (1), both in local and global sense.

Theorem 1. *The malaria-free equilibrium point of model system (1), $D_f = (S_h^*, A_h^*, I_h^*, R_h^*, S_m^*, I_m^*)$ is locally asymptotically stable whenever $R_0 < 1$ otherwise unstable.*

Proof. The Jacobian matrix of the system (1) from DFE, D_f , is given by the following:

where

$$\begin{aligned} A_0 &= 1, \\ A_1 &= (\alpha + \phi) + (\gamma + \sigma + \mu), \\ A_2 &= (\alpha + \phi)(\gamma + \sigma + \mu) - \frac{\beta_h \beta_m \Lambda_m \mu^2}{\Lambda_h(\mu + \tau)(\alpha + \delta)} \\ &= (\alpha + \phi)(\gamma + \sigma + \mu)(1 - R_0^2) \\ &\leq (\alpha + \phi)(\gamma + \sigma + \mu)(1 - R_0^2). \end{aligned} \quad (34)$$

Thus, $A_2 = (\alpha + \phi)(\gamma + \sigma + \mu)(1 - R_0^2) > 0$ provided that $(1 - R_0^2) > 0$, which leads to $R_0 < 1$. According to Routh–Hurwitz criteria, it can be seen that the characteristic polynomial $\varphi(\lambda)$ has all negative real roots whenever $R_0 < 1$. Hence, the DFE point D_f is locally asymptotically stable. But for $R_0 > 1$, there is sign change on A_2 , and D_f is unstable. The epidemiological implication of this theorem is that the transmission of malaria can be controlled from the community when $R_0 < 1$; otherwise, reinvasion will occur within the society. \square

Theorem 2. *The DFE point $D_f = (S_h^*, A_h^*, I_h^*, R_h^*, S_m^*, I_m^*)$ of model system (1) is globally asymptotically stable whenever $R_0 < 1$ otherwise unstable.*

Proof. To evaluate the global stability of D_f of model system (1), we formulate a suitable Lyapunov function

$$L : \mathbb{R}_+^2 \rightarrow \mathbb{R} \\ (I_h, I_m) \rightarrow \frac{(\alpha + \phi)(\tau + \mu)I_h}{\beta_h \mu} + I_m. \quad (35)$$

It is easy to observe that L is of class C^1 on the interior of Δ , and the time derivative of L along the solutions of Equation (1) is given by the following:

$$\begin{aligned} \dot{L} &= \frac{(\alpha + \phi)(\tau + \mu)}{\beta_h \mu} \left(\frac{\beta_h S_h I_m}{N_h} - (\sigma + \gamma + \mu)I_h \right) + \frac{\beta_m S_m I_h}{N_h} - (\alpha + \phi)I_m, \\ \Rightarrow \dot{L} &= \left\{ \frac{\beta_m \Lambda_m \mu^2}{\Lambda_h (\alpha + \delta)} - \frac{(\tau + \mu)(\alpha + \phi)(\gamma + \sigma + \mu)}{\beta_h \mu} \right\} I_h - (\alpha + \phi)I_m \left\{ 1 - \frac{S_h}{N_h} \right\}, \\ &\leq \left\{ \frac{\beta_m \Lambda_m \mu^2}{\Lambda_h (\alpha + \delta)} - \frac{(\alpha + \phi)(\tau + \mu)(\gamma + \sigma + \mu)}{\beta_h \mu} \right\} I_h, \\ &= \frac{(\alpha + \phi)(\tau + \mu)(\sigma + \gamma + \mu)}{\beta_h \mu} \left(\frac{\beta_m \beta_h \Lambda_m \mu^2}{\Lambda_h (\tau + \mu)(\alpha + \phi)(\alpha + \delta)(\sigma + \gamma + \mu)} - 1 \right), \\ &= \frac{(\alpha + \phi)(\tau + \mu)(\sigma + \gamma + \mu)}{\mu \beta_h} (R_0^2 - 1). \end{aligned} \quad (36)$$

Here, $\dot{L} \leq 0$ since $R_0 < 1$ and $\dot{L} = 0 \Leftrightarrow I_h = I_m = 0$. Hence, L is a Lyapunov function on Δ . Additionally, the singleton set D_f is the largest compact invariant set in $\{(S_h, A_h, I_h, R_h, S_m, I_m) \in \Delta : \dot{L} = 0\}$. As a result, by LaSalle's invariant principle [29], D_f is globally asymptotically stable in the interior of Δ for $R_0 < 1$. \square

Theorem 3. The disease persistent equilibrium point of the model system (1) $D_p = (S_h^{**}, A_h^{**}, I_h^{**}, R_h^{**}, S_m^{**}, I_m^{**})$ is globally asymptotically stable on Δ for $R_0 > 1$.

Proof. To prove this theorem, we employ the Dulac's criterion [30] by taking $\varrho(I_h, I_m) = \frac{1}{I_h I_m}$ as the candidate of Dulac's function for model system (1) and

$$\Gamma = (S_h, A_h, I_h, R_h, S_m, I_m).$$

Since $\varrho(I_h, I_m) > 0, \forall I_h, I_m > 0$, we have that

$$\Sigma(S_h, A_h, I_h, R_h, S_m, I_m) = \frac{d\varrho\Gamma}{dt} = \frac{d\varrho S_h}{dt} + \frac{d\varrho A_h}{dt} + \frac{d\varrho I_h}{dt} + \frac{d\varrho R_h}{dt} + \frac{d\varrho S_m}{dt} + \frac{d\varrho I_m}{dt}. \quad (37)$$

Then, we have that

$$\begin{aligned} \Sigma(S_h, A_h, I_h, R_h, S_m, I_m) &= \frac{\partial}{\partial S_h} \left(\frac{1}{I_h I_m} \left(\Lambda_h - \frac{\beta_h S_h I_m}{N_h} - (\mu + \tau)S_h + \omega R_h \right) \right) \\ &\quad + \frac{\partial}{\partial A_h} \left(\frac{1}{I_h I_m} (\tau S_h - \mu A_h + \omega R_h) \right) \\ &\quad + \frac{\partial}{\partial I_h} \left(\frac{1}{I_h I_m} \left(\frac{\beta_h S_h I_m}{N_h} - (\sigma + \gamma + \mu)I_h \right) \right) \\ &\quad + \frac{\partial}{\partial R_h} \left(\frac{1}{I_h I_m} (\gamma I_h - (\mu + \omega)R_h) \right) \\ &\quad + \frac{\partial}{\partial S_m} \left(\frac{1}{I_h I_m} \left(\Lambda_m - \frac{\beta_m S_m I_h}{N_h} - (\alpha + \delta)S_m \right) \right) \\ &\quad + \frac{\partial}{\partial I_m} \left(\frac{1}{I_h I_m} \left(\frac{\beta_m S_m I_h}{N_h} - (\alpha + \phi)I_m \right) \right) \\ &= - \left[\frac{\beta_h}{I_h N_h} + \frac{\mu + \tau}{I_h I_m} + \frac{\mu}{I_h I_m} + \frac{\beta_h S_h}{I_h^2 N_h} \right. \\ &\quad \left. + \frac{\mu + \omega}{I_h I_m} + \frac{\beta_m}{N_h I_m} + \frac{\alpha + \delta}{I_h I_m} \right] < 0. \end{aligned} \quad (38)$$

This implies Dulac's function ϱ , and model system (1) does not contain any periodic orbits in $\mathbb{R}^6 \setminus \{I_h, I_m = 0\}$. As a result, whenever $R_0 > 1$, the disease persistent equilibrium point D_p is globally asymptotically stable. In epidemiological point of view, the above result suggests that there will be fluctuation in the transmission of malaria in society, which makes it challenging to allocate resources for the control of malaria transmission. \square

4.5. Bifurcation Analysis of the Model. In this section, we look at the concept of the bifurcation, which includes the infection-free equilibrium, D_f at $R_0 = 1$, by employing Center Manifold theory [31] for system (1). To apply this, variable changes are made on the system (1) as follows $S_h = x_1, A_h = x_2, I_h = x_3, R_h = x_4, S_m = x_5$ and $I_m = x_6$, so that $N_h = x_1 + x_2 + x_3 + x_4$. Further, by using vector notation $x = (x_1, x_2, x_3, x_4, x_5, x_6)^T$, the model system (1) can be written as $\frac{dx}{dt} = (f_1, f_2, f_3, f_4, f_5, f_6)^T$ where

$$\frac{dx_1}{dt} = \Lambda_h - \frac{\beta_h x_1 x_6}{x_1 + x_2 + x_3 + x_4} - (\mu + \tau)x_1 = f_1, \quad (39a)$$

$$\frac{dx_2}{dt} = \tau x_1 - \mu x_2 + \omega x_4 = f_2, \quad (39b)$$

$$\frac{dx_3}{dt} = \frac{\beta_h x_1 x_6}{x_1 + x_2 + x_3 + x_4} - (\gamma + \sigma + \mu)x_3 = f_3, \quad (39c)$$

$$\frac{dx_4}{dt} = \gamma x_3 - (\mu + \omega)x_4 = f_4, \quad (39d)$$

$$\frac{dx_5}{dt} = \Lambda_m - \frac{\beta_m x_5 x_3}{x_1 + x_2 + x_3 + x_4} - (\alpha + \delta)x_5 = f_5, \quad (39e)$$

$$\frac{dx_6}{dt} = \frac{\beta_m x_5 x_3}{x_1 + x_2 + x_3 + x_4} - (\alpha + \phi)x_6 = f_6. \quad (39f)$$

The approach consists of evaluating the system's linearized matrix (39) at the DFE point, D_f , which is given by the following:

$$J(D_f) = \begin{bmatrix} -(\mu + \tau) & 0 & 0 & 0 & 0 & \frac{-\beta_h \mu}{\mu + \tau} \\ \tau & -\mu & 0 & \omega & 0 & 0 \\ 0 & 0 & -(\gamma + \sigma + \mu) & 0 & 0 & \frac{\beta_h \mu}{\mu + \tau} \\ 0 & 0 & \gamma & -(\mu + \omega) & 0 & 0 \\ 0 & 0 & \frac{-\beta_m \Lambda_m \mu}{\Lambda_h(\alpha + \delta)} & 0 & -(\alpha + \delta) & 0 \\ 0 & 0 & \frac{\beta_m \Lambda_m \mu}{\Lambda_h(\alpha + \delta)} & 0 & 0 & -(\alpha + \phi) \end{bmatrix}. \quad (40)$$

If we choose $\beta_h = \beta_h^*$ as the bifurcation parameter for $R_0 = 1$, then the solution to the problem of finding β_h from $R_0 = 1$ gives $\beta_h = \beta_h^* = \frac{\Lambda_h(\alpha + \delta)(\tau + \mu)(\sigma + \gamma + \mu)}{\beta_m \Lambda_m \mu^2}$. After some algebraic manipulations on Equation (40), the Jacobian of the system $J_{(\beta_h = \beta_h^*)}$ has eigenvalues

$$\begin{aligned} \lambda_1 &= -\mu, \\ \lambda_2 &= -(\tau + \mu), \\ \lambda_3 &= -(\mu + \omega), \\ \lambda_4 &= -(\alpha + \delta), \\ \lambda_5 &= 0, \\ \lambda_6 &= -[(\sigma + \gamma + \mu) + (\alpha + \phi)], \end{aligned} \quad (41)$$

which indicates that it has a single simple zero eigenvalue and that all other eigenvalues are real and negative. Therefore, the DFE is a nonhyperbolic equilibrium for $\beta_h = \beta_h^*$. With the above simple zero eigenvalue, $Jw = 0$ determines the right eigenvector, $w = (w_1, w_2, w_3, w_4, w_5, w_6)^T$, where

$$\begin{aligned} w_1 &= \frac{-\beta_h \mu}{(\mu + \tau)^2} w_6, \\ w_2 &= w_6 > 0, \\ w_3 &= \frac{1}{\mu} \left[\frac{\gamma \omega \beta_h \mu}{(\tau + \mu)(\mu + \omega)(\sigma + \gamma + \mu)} - \frac{\tau \beta_h \mu}{(\mu + \tau)^2} \right] w_6, \\ w_4 &= \frac{\beta_h \mu}{(\sigma + \gamma + \mu)(\tau + \mu)} w_6, \\ w_5 &= \frac{\gamma \beta_h \mu}{(\sigma + \gamma + \mu)(\tau + \mu)(\mu + \omega)} w_6, \\ w_6 &= \frac{-\beta_h \beta_m \Lambda_m \mu^2}{\Lambda_h(\sigma + \gamma + \mu)(\alpha + \delta)^2(\tau + \mu)} w_6. \end{aligned} \quad (42)$$

The left eigenvectors $v = (v_1, v_2, v_3, v_4, v_5, v_6)$ that correspond to the simple zero eigenvalues above are obtained from $vJ = 0$ and are given by the following:

$$\begin{aligned}\nu_1 &= 0, \\ \nu_2 &= 0, \\ \nu_4 &= 0, \\ \nu_5 &= 0, \\ \nu_3 &= \frac{(\beta_m \Lambda_m \mu)}{\Lambda_h(\alpha + \delta)(\gamma + \sigma + \mu)} \nu_6,\end{aligned}\quad (43)$$

which suggests that ν_6 is free. Let x_k represent state variables for $k = 1, 2, 3, 4, 5, 6$, and let f_k represent the k th component of the right-hand sides of model (1). The signs of the two coefficients a and b , which are completely responsible for the local dynamics of system (1) around D_f , where

$$\begin{aligned}a &= \sum_{k,i,j=1}^6 \nu_k \omega_i \omega_j \frac{\partial^2 f_k(D_f)}{\partial x_i \partial x_j} \\ b &= \sum_{i,k=1}^6 \nu_k \omega_i \left(\frac{\partial^2 f_k(D_f)}{\partial x_i \partial \beta} \right)_{\beta=\beta^*}.\end{aligned}\quad (44)$$

According to the left eigenvector, we then have that

$$\begin{aligned}a &= \nu_3 \sum_{i,j=1}^6 w_i w_j \frac{\partial^2 f_3(D_f)}{\partial x_i \partial x_j} + \nu_6 \sum_{i,j=1}^6 w_i w_j \frac{\partial^2 f_6(D_f)}{\partial x_i \partial x_j} \\ &= \nu_3 w_1 w_6 \frac{\partial}{\partial x_1} \left(\frac{\partial f_3(D_f)}{\partial x_6} \right) + \nu_3 w_6 w_1 \frac{\partial}{\partial x_6} \left(\frac{\partial f_3(D_f)}{\partial x_1} \right) \\ &\quad + \nu_6 w_3 w_5 \frac{\partial}{\partial x_3} \left(\frac{\partial f_6(D_f)}{\partial x_5} \right) + \nu_6 w_5 w_3 \frac{\partial}{\partial x_5} \left(\frac{\partial f_6(D_f)}{\partial x_3} \right) \\ &= -2 \left[\frac{\beta_h \mu \nu_3}{(\mu + \tau)^2} + \frac{\beta_h^2 \mu^2 \nu_6}{(\alpha + \delta)(\tau + \mu)^2(\sigma + \gamma + \mu)^2} \right] w_6^2 < 0,\end{aligned}\quad (45)$$

and

$$\begin{aligned}b &= \nu_3 \sum_{i=1}^6 w_i \frac{\partial^2 f_3(D_f)}{\partial x_i \partial \beta_h} + \nu_6 \sum_{i=1}^6 w_i \frac{\partial^2 f_6(D_f)}{\partial x_i \partial \beta} \\ &= \nu_3 w_3 \frac{\partial}{\partial x_6} \left(\frac{\partial f_3(D_f)}{\partial \beta_h} \right) + \nu_6 w_3 \frac{\partial}{\partial x_3} \left(\frac{\partial f_6(D_f)}{\partial \beta^*} \right) \\ &= \frac{1}{x_1 + x_2} [\nu_3 \omega_6 x_1 + \nu_6 \omega_3 x_5], \\ &= \frac{1}{x_1 + x_2} \left[\nu_3 x_1 + \frac{\nu_6 \beta_h \mu}{(\tau + \mu)(\sigma + \gamma + \mu)} x_5 \right] \omega_6 > 0.\end{aligned}\quad (46)$$

Since the coefficient $a < 0$ and $b > 0$, the model experiences forward bifurcation, which suggests that $R_0 < 1$ is both a necessary and sufficient condition for the cessation of malaria transmission.

4.6. Sensitivity Analysis of the Basic Reproductive Number. To ascertain the model's robustness to parameter values, we conducted a sensitivity analysis on the model's basic

reproductive number. Mathematically, the normalized forward sensitivity index of a variable to a parameter is a differentiable function of the parameter and is defined using partial derivatives based on the classical definition given in [32].

Definition 1. The sensitivity and elasticity of quantity R_0 with respect to the parameter ν is given by the following:

$$\Phi_\nu^{R_0} = \frac{\partial R_0}{\partial \nu} \cdot \frac{\nu}{R_0} \quad ([31]). \quad (47)$$

Here, for example $\Phi_\nu^{R_0} = \frac{\partial R_0}{\partial \nu} \cdot \frac{\nu}{R_0} \equiv +0.1$ means, increasing (or decreasing) the value of ν by 10%, always result to increase (or decrease) the value of R_0 by 10%, whereas $\Phi_\nu^{R_0} \equiv -0.1$ means increasing (or decreasing) the value of ν by 10%, always result to decrease (or increase) R_0 by 10%.

We computed the sensitivity indices of system (1) in a similar manner, and the results are as follows:

$$\begin{aligned}\Phi_{\beta_h}^{R_0} &= \frac{1}{2} > 0, \quad \Phi_{\beta_v}^{R_0} = \frac{1}{2} > 0, \quad \Phi_{\Lambda_v}^{R_0} = \frac{1}{2} > 0, \\ \Phi_\tau^{R_0} &= \frac{-\tau}{2(\tau + \mu)} < 0, \\ \Phi_\gamma^{R_0} &= \frac{-\gamma}{2(\sigma + \gamma + \mu)} < 0, \\ \Phi_\delta^{R_0} &= \frac{-\delta}{2(\alpha + \delta)} < 0, \\ \Phi_\phi^{R_0} &= \frac{-\phi}{2(\alpha + \phi)} < 0.\end{aligned}\quad (48)$$

Here, we want to notice the biological impossibility of increasing mortality rates in the presence of declining malaria transmission rates, so we did not conduct a sensitivity analysis of those mortality rates.

According to the sensitivity analysis index, parameters with positive indexes β_h , β_v , and Λ_v have an adverse effect on the ability to control the spread of malaria, whereas parameters with negative indexes τ , δ , ϕ , and γ have an advantageous effect. Table 3 provides the summarize of the sensitivity analysis of model system (1).

4.6.1. Interpretation of Sensitivity Indices. It is noted from Table 3 that when the values of β_h , β_v , and Λ_v rise while the values of the other parameters remain constant, the value of R_0 rises. By increasing the values of these parameters, the secondary infection, R_0 , will also increase, which means malaria transmission will revive within the society. On the other hand, while the other parameters remain constant, increasing the values of γ , ϕ , τ , and δ causes the value of the secondary infection, R_0 , to decrease. This indicates a reduction in the transmission of malaria as a result of the population's overall infection rate falling as these parameters' values are raised. As a result, the model's sensitivity analysis showed that in order to stop the transmission of malaria among the population, authorities, and health practitioners should increase parameters with negative indices and decrease those with positive indices.

TABLE 3: Elasticity index of model system (1).

Parameter	Elasticity index
β_h	+ ve
β_m	+ ve
Λ_m	+ ve
τ	- ve
γ	- ve
δ	- ve
ϕ	- ve

5. Extension of the Model into Optimal Control

In this section, we extend our model system (1) to the optimal control problem by including three time-dependent control strategies, $c_1(t)$, $c_2(t)$, and $c_3(t)$, where

- (i) $c_1(t)$ stands the efforts intended for susceptible individuals to create awareness through all media outlets.
- (ii) $c_2(t)$ stands the initiatives aimed at providing adequate control measures of malaria.
- (iii) $c_3(t)$ stands the efforts made to assist infected people with continuous treatment.

For the seek of simplicity, we represent $c_1(t) = c_1$, $c_2(t) = c_2$, and $c_3(t) = c_3$. After incorporating the above intervention strategies into model (1), we get an optimal control model described by the following:

$$\dot{S}_h(t) = \Lambda_h - (1 - c_1) \frac{\beta_h S_h I_m}{N_h} - (\mu + \tau + c_2) S_h, \quad (49a)$$

$$\dot{A}_h(t) = (\tau + c_2) S_h - \mu A_h + \omega R_h, \quad (49b)$$

$$\dot{I}_h(t) = (1 - c_1) \frac{\beta_h S_h I_m}{N_h} - (\sigma + \mu + \gamma + c_3) I_h, \quad (49c)$$

$$\dot{R}_h(t) = (\gamma + c_3) I_h - (\omega + \mu) R_h, \quad (49d)$$

$$\dot{S}_m(t) = \Lambda_m - \frac{\beta_m S_m I_h}{N_h} - (\alpha + \delta) S_m, \quad (49e)$$

$$\dot{I}_m(t) = \frac{\beta_m S_m I_h}{N_h} - (\alpha + \phi) I_m, \quad (49f)$$

where $\{0 \leq c_i \leq 1, i = 1, 2, 3, [0, t_f]\}$ is assumed to be Lebesgue measurable [33]. The ultimate goal is to obtain the optimal levels of the controls that optimize the objective function defined by the following:

$$J(c_1, c_2, c_3) = \min_{c_1, c_2, c_3} \int_0^{t_f} \left[A_1 I_h + \frac{1}{2} (B_1 c_1^2 + B_2 c_2^2 + B_3 c_3^2) \right] d\tau, \quad (50)$$

where t_f is fixed final time, and A_1 is the weight constant of the infected population, while B_1, B_2 , and B_3 are constants that balance the cost of control strategies at time t . Since the measure of cost function is not linear in its condition, thus we take the quadratic function as frequently used in [34, 35]. Here, we want to find the optimal triple (c_1^*, c_2^*, c_3^*) such that

$$J(c_1^*, c_2^*, c_3^*) = \min \{J(c_1, c_2, c_3) : c_1, c_2, c_3 \in C\}, \quad (51)$$

where $C = \{(c_1, c_2, c_3) | 0 \leq c_i \leq 1, i = 1, 2, 3\}$ is the set of acceptable controls.

5.1. Existence of an Optimal Control

Theorem 4. *There exists an optimal control (c_1^*, c_2^*, c_3^*) that minimizes the objective functional $J(c_1, c_2, c_3)$, subject to the control system (49).*

Proof. To show the existence of optimal control, we denote the right-hand sides of system (49) by $z(t, \vec{x}, \vec{c})$. Then, to prove the existence of optimal control we followed the classical result of [36, 37]. To achieve this, the following conditions must met.

- (i) z is of class C^1 and there exists a constant φ such that

$$|z(t, 0, 0)| \leq K, |z_{\vec{x}}(t, \vec{x}, \vec{c})| \leq K(1 + |\vec{c}|), |z_{\vec{c}}(t, \vec{x}, \vec{c})| \leq K, \quad (52)$$

- (ii) The admissible set of all solutions to system (49) with corresponding control in C_{ad} is nonempty,
- (iii) There exist functions a and b such that $z(t, \vec{x}, \vec{c}) = a(t, \vec{x}) + b(t, \vec{x}) \vec{c}$,
- (iv) The control set $C = [0, 1] \times [0, 1] \times [0, 1]$ is closed, convex and compact,
- (v) The integrand of the objective function is convex in C .

To verify condition (1), rewrite

$$z(t, \vec{x}, \vec{c}) = \begin{bmatrix} \Lambda_h - (1 - c_1) \frac{\beta_h S_h I_m}{N_h} - (\tau + \mu + c_2) S_h \\ (\tau + c_2) S_h - \mu A_h + \omega R_h \\ (1 - c_1) \frac{\beta_h S_h I_m}{N_h} - (\gamma + \sigma + \mu + c_3) I_h \\ (\gamma + c_3) I_h - (\mu + \omega) R_h \\ \Lambda_m - \frac{\beta_m S_m I_h}{N_h} - (\alpha + \delta) S_m \\ \frac{\beta_m S_m I_h}{N_h} - (\alpha + \phi) I_m \end{bmatrix}. \quad (53)$$

From Equation (53), it is clear that $|z(t, 0, 0)| = \Lambda_h + \Lambda_m$ and that $z(t, \vec{x}, \vec{c})$ is a class of C^1 .

Moreover, consider

$$|z_{\vec{x}}(t, \vec{x}, \vec{c})| = \left| \begin{bmatrix} -(1-c_1)\frac{\beta_h I_m}{N_h} - a_1 & 0 & 0 & 0 & 0 & -(1-c_1)\frac{\beta_h S_h}{N_h} \\ (\tau+c_2) & -\mu & 0 & \omega & 0 & 0 \\ (1-c_1)\frac{\beta_h I_m}{N_h} & 0 & -a_2 & 0 & 0 & (1-c_1)\frac{\beta_h S_h}{N_h} \\ 0 & 0 & \gamma+c_3 & -(\mu+\omega) & 0 & 0 \\ 0 & 0 & \frac{-\beta_m S_m}{N_h} & 0 & \frac{\beta_m I_h}{N_h} - (\alpha+\delta) & 0 \\ 0 & 0 & \frac{\beta_m S_m}{N_h} & 0 & \frac{\beta_m I_h}{N_h} & -(\alpha+\phi) \end{bmatrix} \right|, \quad (54)$$

where $a_1 = (\tau + \mu + c_2)$, $a_2 = (\gamma + \mu + \sigma + c_3)$ and

$$|z_{\vec{c}}(t, \vec{x}, \vec{c})| = \left| \begin{bmatrix} \frac{\beta_h S_h I_m}{N_h} & -S_h & 0 \\ 0 & S_h & 0 \\ \frac{-\beta_h S_h I_m}{N_h} & 0 & -I_h \\ 0 & 0 & I_h \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \right|. \quad (55)$$

Since S_h, A_h, I_h, R_h, S_m and I_m are constrained, a constant K exists such that

$$|z(t, 0, 0)| \leq K, |z_{\vec{x}}(t, \vec{x}, \vec{c})| \leq K(1 + |\vec{c}|), |z_{\vec{c}}(t, \vec{x}, \vec{c})| \leq K. \quad (56)$$

This suggests that condition (i) holds.

A unique solution to system (49) for a constant control exists in accordance with condition (i), which also implies that condition (ii) is satisfied.

Furthermore, we have the following:

$$z(t, \vec{x}, \vec{c}) = \begin{bmatrix} \Lambda_h - \frac{\beta_h S_h I_m}{N_h} - (\tau + \mu)S_h \\ \tau S_h - \mu A_h + \omega R_h \\ \frac{\beta_h S_h I_m}{N_h} - (\gamma + \sigma + \mu)I_h \\ \gamma I_h - (\mu + \omega)R_h \\ \Lambda_m - \frac{\beta_m S_m I_h}{N_h} - (\alpha + \delta)S_m \\ \frac{\beta_m S_m I_h}{N_h} - (\alpha + \phi)I_m \end{bmatrix} + \begin{bmatrix} \frac{\beta_h S_h I_m}{N_h} & -S_h & 0 \\ 0 & S_h & 0 \\ \frac{-\beta_h S_h I_m}{N_h} & 0 & -I_h \\ 0 & 0 & I_h \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} c_1 \\ c_2 \\ c_3 \end{bmatrix}. \quad (57)$$

As a result, condition (iii) is met. The control set C is compact because it is closed and bounded. This confirms the state (iv). Finally, in order to show the convexity of the integrand in the objective functional $f(t, \vec{x}, \vec{c})$, we must ensure that for any two control vectors \vec{c} and \vec{d} and a constant $n \in [0, 1]$, the following inequality holds:

$$(1-n)f(t, \vec{x}, \vec{c}) + nf(t, \vec{x}, \vec{d}) \geq f(t, \vec{x}, (1-n)\vec{c} + n\vec{d}), \quad (58)$$

where

$$f(t, \vec{x}, \vec{c}) = A_1 I_h + \frac{1}{2} [B_1 (\vec{c}_1)^2 + B_2 (\vec{c}_2)^2 + B_3 (\vec{c}_3)^2]. \quad (59)$$

Further,

$$(1-n)f(t, \vec{x}, \vec{c}) + nf(t, \vec{x}, \vec{d}) = A_1 I + \frac{1-n}{2} [B_1(\vec{c}_1)^2 + B_2(\vec{c}_2)^2 + B_3(\vec{c}_3)^2] + \frac{n}{2} [B_1(\vec{d}_1)^2 + B_2(\vec{d}_2)^2 + B_3(\vec{d}_3)^2], \quad (60)$$

and

$$f(t, \vec{x}, (1-n)\vec{c} + n\vec{d}) = A_1 I_h + \frac{1}{2} [B_1[(1-n)\vec{c}_1 + n\vec{d}_1]^2 + B_2[(1-n)\vec{c}_2 + n\vec{d}_2]^2 + B_3[(1-n)\vec{c}_3 + n\vec{d}_3]^2]. \quad (61)$$

After some mathematical computations, we obtained that

$$\begin{aligned} & \frac{B_1}{2} [n(1-n)(c_1 - d_1)^2] + \frac{B_2}{2} [n(1-n)(c_2 - d_2)^2] + \frac{B_3}{2} [n(1-n)(c_3 - d_3)^2] \\ & \Rightarrow (1-n)f(t, \vec{x}, \vec{c}) + nf(t, \vec{x}, \vec{d}) - f(t, \vec{x}, (1-n)\vec{c} + n\vec{d}) \geq 0 \end{aligned} \quad (62)$$

This implies that condition (v) is fulfilled.

Hence, the proof of the theorem is completed. \square

$$H(t, x, c, \xi) = \frac{dJ}{dt} + \sum_{i=1}^6 \xi_i f_i = L(t, x, c) + \sum_{i=1}^6 \xi_i f_i, \quad (64)$$

5.2. The Hamiltonian and Optimality System. The Pontryagin et al. [38] maximum principle formulates the necessary conditions that the optimal control must meet. To solve the optimal control problem (50) with the help of Equation (49), we have to construct the Lagrangian function as follows:

$$L(t, x, c) = \frac{dJ}{dt} = A_1 I_h + \frac{1}{2} [B_1 c_1^2(t) + B_2 c_2^2(t) + B_3 c_3^2(t)]. \quad (63)$$

The Pontryagin's Maximum Principle converts the system (49) and (50) into a problem of minimizing pointwise Hamiltonian (H) with respect to the controls c_1 , c_2 , and c_3 as follows:

where $L(t, x, c) = A_1 I + \frac{1}{2} [B_1 c_1^2(t) + B_2 c_2^2(t) + B_3 c_3^2(t)]$ and f_i , $i = 1, 2, 3, 4, 5, 6$ are the right hand side components of Equation (49) and ξ_i , $i = 1, 2, 3, 4, 5, 6$ are costate variables to be determined. The appropriate partial derivatives of H with respect to the state variables can be used to obtain these costate variables.

Theorem 5. Suppose we have optimal controls c_1^* , c_2^* , and c_3^* and S_h, A_h, I_h, R_h, S_m and I_m solutions of the respective state system that minimizes the objective functional (50) over C , there exist costate variables, ξ_1, \dots, ξ_6 satisfying

$$\begin{cases} \dot{\xi}_1 = \left[\frac{(1-c_1^*)\beta_h I_m^*}{N_h} + (\tau + \mu + c_2) \right] \xi_1 - (\tau + c_2^*)\xi_2 - \frac{(1-c_1^*)\xi_3 \beta_h I_m^*}{N_h}, \\ \dot{\xi}_2 = \mu \xi_2, \\ \dot{\xi}_3 = -A_1 + (\sigma + \gamma + \mu + c_3^*)\xi_3 - (\gamma + c_3^*)\xi_4 + \frac{\beta_m \xi_5 S_m^*}{N_h} - \frac{\beta_m \xi_6 S_m^*}{N_h}, \\ \dot{\xi}_4 = (\mu + \omega)\xi_4 - \omega \xi_2 \\ \dot{\xi}_5 = \frac{\beta_m I_h^* (\xi_5 - \xi_6)}{N_h} + (\alpha + \delta)\xi_5, \\ \dot{\xi}_6 = \frac{(1-c_1^*)\xi_1 \beta_h S_h^*}{N_h} - \frac{(1-c_1^*)\beta_h \xi_3 S_h^*}{N_h} + (\alpha + \phi)\xi_6, \end{cases} \quad (65)$$

with transversality conditions:

$$\xi_1(t_f) = \xi_2(t_f) = \xi_3(t_f) = \xi_4(t_f) = \xi_5(t_f) = \xi_6(t_f) = 0. \quad (66)$$

Furthermore, the ideal control set $\{c_1^*(t), c_2^*(t), c_3^*(t)\}$, which minimizes $J(c_1, c_2, c_3)$ over the region C , is obtained and given in compact notation as follows:

$$\begin{cases} c_1^*(t) = \max \left\{ 0, \min \left(1, \left(\frac{\beta_h S_h^* I_m^*}{N_h} \right) \left(\frac{\xi_3 - \xi_1}{B_1} \right) \right) \right\}, \\ c_2^*(t) = \max \left\{ 0, \min \left(1, \frac{S_h^* (\xi_1 - \xi_2)}{B_2} \right) \right\}, \\ c_3^*(t) = \max \left\{ 0, \min \left(1, \frac{(\xi_3 - \xi_4) I_h^*}{B_3} \right) \right\}. \end{cases} \quad (67)$$

Proof. Differentiating the Hamiltonian, H with respect to state variables provides the following:

$$\begin{aligned} \dot{\xi}_1 &= -\frac{\partial H}{\partial S_h} = \left[\frac{(1 - c_1^*) \beta_h I_m^*}{N_h} + (\tau + \mu + c_2) \right] \xi_1 - (\tau + c_2^*) \xi_2 - \frac{(1 - c_1^*) \xi_3 \beta_h I_m^*}{N_h}, \\ \dot{\xi}_2 &= -\frac{\partial H}{\partial A_h} = \mu \xi_2, \\ \dot{\xi}_3 &= -\frac{\partial H}{\partial I_h} = -A_1 + (\sigma + \gamma + \mu + c_3^*) \xi_3 - (\gamma + c_3^*) \xi_4 + \frac{\beta_m \xi_5 S_m^*}{N_h} - \frac{\beta_m \xi_6 S_m^*}{N_h}, \\ \dot{\xi}_4 &= -\frac{\partial H}{\partial R_h} = (\mu + \omega) \xi_4 - \omega \xi_2, \\ \dot{\xi}_5 &= -\frac{\partial H}{\partial S_m} = \frac{\beta_m I_h^* (\xi_5 - \xi_6)}{N_h} + (\alpha + \delta) \xi_5, \\ \dot{\xi}_6 &= -\frac{\partial H}{\partial I_m} = \frac{(1 - c_1^*) \xi_1 \beta_h S_h^*}{N_h} - \frac{(1 - c_1^*) \beta_h \xi_3 S_h^*}{N_h} + (\alpha + \phi) \xi_6. \end{aligned} \quad (68)$$

Moreover, to compute the optimality solutions, we follow the methodology outlined in [39],

and

$$\begin{aligned} \frac{\partial H}{\partial c_1} &= 0, c_1 = c_1^*, \\ \frac{\partial H}{\partial c_2} &= 0, c_2 = c_2^*, \\ \frac{\partial H}{\partial c_3} &= 0, c_3 = c_3^*. \end{aligned} \quad (69)$$

$$\begin{cases} c_3^*(t) = 0, \Delta_3 \leq 0, \\ c_3^*(t) = \Delta_3, 0 < \Delta_3 < 1, \\ c_3^*(t) = 1, \Delta_3 > 1, \end{cases} \quad (72)$$

where

$$\begin{aligned} \Delta_1 &= \left(\frac{\beta_h S_h^* I_m^*}{N_h} \right) \left(\frac{\xi_3 - \xi_1}{B_1} \right), \\ \Delta_2 &= \frac{S_h^* (\xi_1 - \xi_2)}{B_2}, \\ \Delta_3 &= \frac{(\xi_3 - \xi_4) I_h^*}{B_3}. \end{aligned} \quad (73)$$

Therefore, by standard arguments, including control bounds, it follows that

$$\begin{cases} c_1^*(t) = 0, \Delta_1 \leq 0, \\ c_1^*(t) = \Delta_1, 0 < \Delta_1 < 1, \\ c_1^*(t) = 1, \Delta_1 > 1, \end{cases} \quad (70)$$

$$\begin{cases} c_2^*(t) = 0, \Delta_2 \leq 0, \\ c_2^*(t) = \Delta_2, 0 < \Delta_2 < 1, \\ c_2^*(t) = 1, \Delta_2 > 1, \end{cases} \quad (71)$$

Hence, the required result. \square

5.3. Analysis of the Control Reproductive Number. As usual, employing the next-generation method, we calculate the control reproductive number as follows:

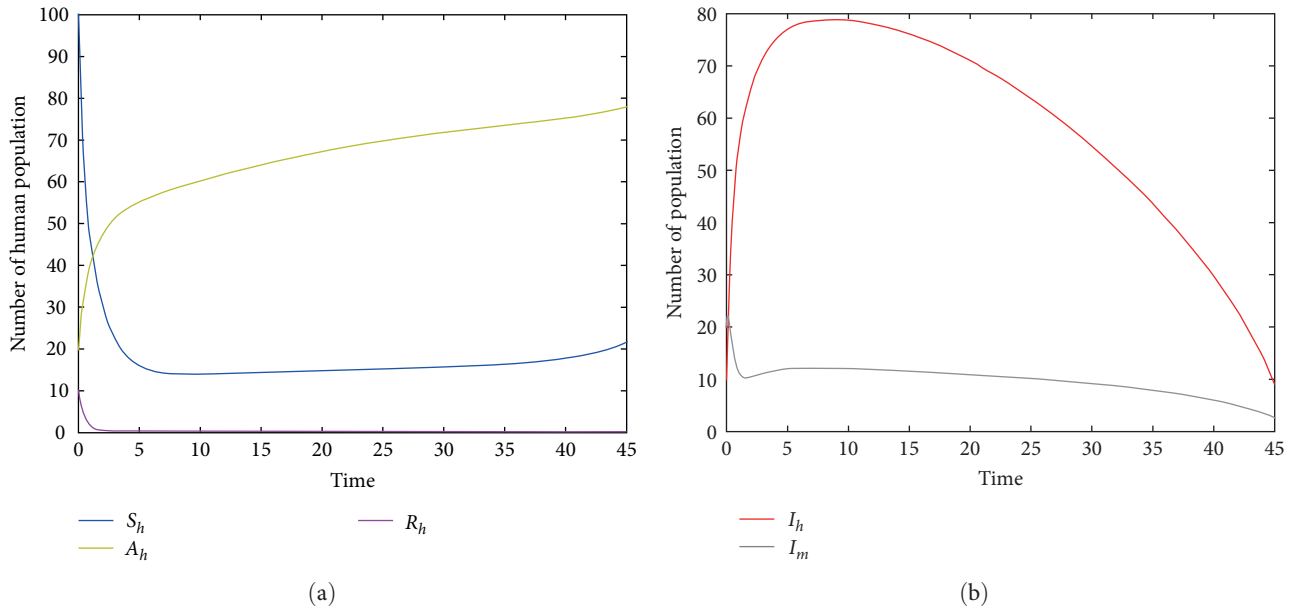


FIGURE 2: (a, b) The numerical simulation result showing the population when $R_0 = 0.3812 < 1$.

$$F_i(X) = \begin{bmatrix} \frac{(1-c_1)\beta_h S_h I_m}{N_h} \\ \frac{\beta_m S_m I_h}{N_h} \end{bmatrix}, V_i(X) = \begin{bmatrix} (\sigma + \gamma + \mu + c_3)I_h \\ (\alpha + \delta)I_v \end{bmatrix}. \quad (74)$$

Then, after some mathematical calculation using the next-generation method, the control reproductive number is denoted by R_{con} and given by the following:

$$R_{\text{con}} = \sqrt{\frac{(1-c_1)\beta_h \beta_m \mu^2 \Lambda_m}{\Lambda_h (\alpha + \delta)^2 (\tau + \mu + c_2) (\sigma + \gamma + \mu + c_3)}}. \quad (75)$$

In the absence of prevention strategies c_1 , c_2 , and c_3 , Equation (75) reduces to Equation (21). It is easy to claim that $R_{\text{con}} \leq R_0$, for $0 \leq c_i \leq 1$, $i = 1, 2, 3$, due to possibility of infection reduction in the presence of control strategies.

Moreover,

$$\begin{aligned} \lim_{c_1 \rightarrow 1} R_{\text{con}} &= 0, \\ \lim_{c_2 \rightarrow 1} R_{\text{con}} &= \sqrt{\frac{(1-c_1)\beta_h \beta_m \mu^2 \Lambda_m}{\Lambda_h (\alpha + \delta)^2 (\tau + \mu + 1) (\sigma + \gamma + \mu + c_3)}} \ll R_0, \\ \lim_{c_3 \rightarrow 1} R_{\text{con}} &= \sqrt{\frac{(1-c_1)\beta_h \beta_m \mu^2 \Lambda_m}{\Lambda_h (\alpha + \delta)^2 (\tau + \mu + c_2) (\sigma + \gamma + \mu + 1)}} \ll R_0, \end{aligned} \quad (76)$$

which means that very high public awareness creation through all media sources about malaria ($c_1 \rightarrow 1$), adequate access to malaria prevention measures, which are treated bed nets and

indoor residual spraying ($c_2 \rightarrow 1$), and also continuous anti-malaria drugs for infected individuals ($c_3 \rightarrow 3$) have a positive effect on the reduction or elimination of malaria transmission within the society.

6. Numerical Results and Discussions

In this section, we carried out some numerical results of model (1) to establish the validity of the theoretical results using MATLAB ode45 solver by using initial conditions: $S_{h0} = 100$, $I_{h0} = 10$, $S_{m0} = 1,000$, $I_{m0} = 20$ taken in [40] and assumptions $A_{h0} = 20$, $R_{h0} = 10$. It is important to notice that parameter values used in the simulations are for illustrative purposes only and may not be epidemiologically accurate.

For parameter values: $\Lambda_h = 10$, $\beta_h = 5.9$, $\Lambda_m = 500$, $\beta_m = 0.955$, $\sigma = 0.051$, $\mu = 0.05$, $\alpha = 1/15$, $\gamma = 0.00991$, $\omega = 1.9$, $\tau = 0.2$, $\delta = 9.91$, $\phi = 1.89$, $\pi = 0.28$, the numerical simulation of model system (1) is illustrated. Figures 2(a) and 2(b) show for $R_0 = 0.3812$, the class of aware population $A_h(t)$ and susceptibles populations $S_h(t)$ are increasing, while recovered populations are decreasing. At the same condition, infected human and vector populations are decreasing but not sufficient to eradicate malaria disease. This finding supports the analytical result that the DFE point is locally asymptotically stable for $R_0 < 1$ as stated in Theorem 1. This result indicates that malaria disease will gradually decrease or eliminate from the population. Figures 3(a) and 3(b) shows that all solution curves approaches almost to non-zero components or to their endemic equilibrium point components for parameter values $\Lambda_h = 10$, $\beta_h = 5.99$, $\Lambda_v = 600$, $\beta_v = 9.05$, $\sigma = 0.051$, $\mu = 0.05$, $\alpha = 1$, $\gamma = 0.01991$, $\omega = 0.0075$, $\tau = 0.1892$, $\delta = 11.99$, $\phi = 1.89$, $\pi = 0.18$. This result revealed that the global asymptotic stability of the malaria disease persistent equilibrium point, which occurs when $R_0 = 2.9713 > 1$, indicates that the disease will persist in the population.

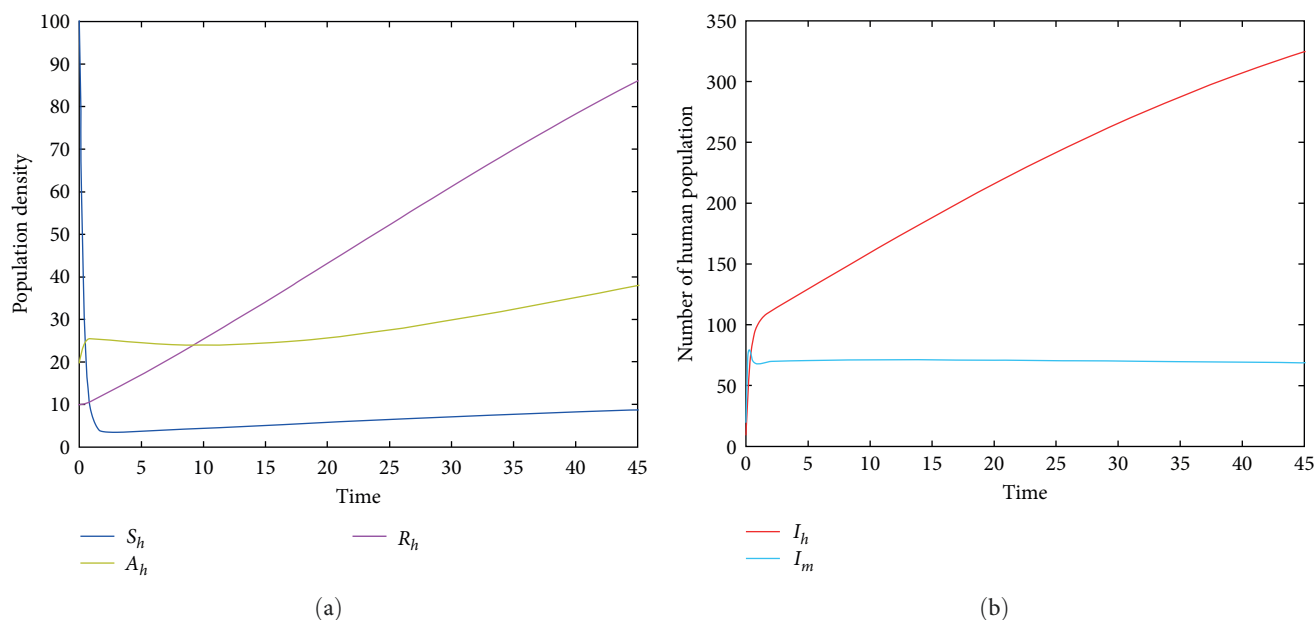


FIGURE 3: (a, b) The numerical simulation result showing the population when $R_0 = 2.9713 > 1$.

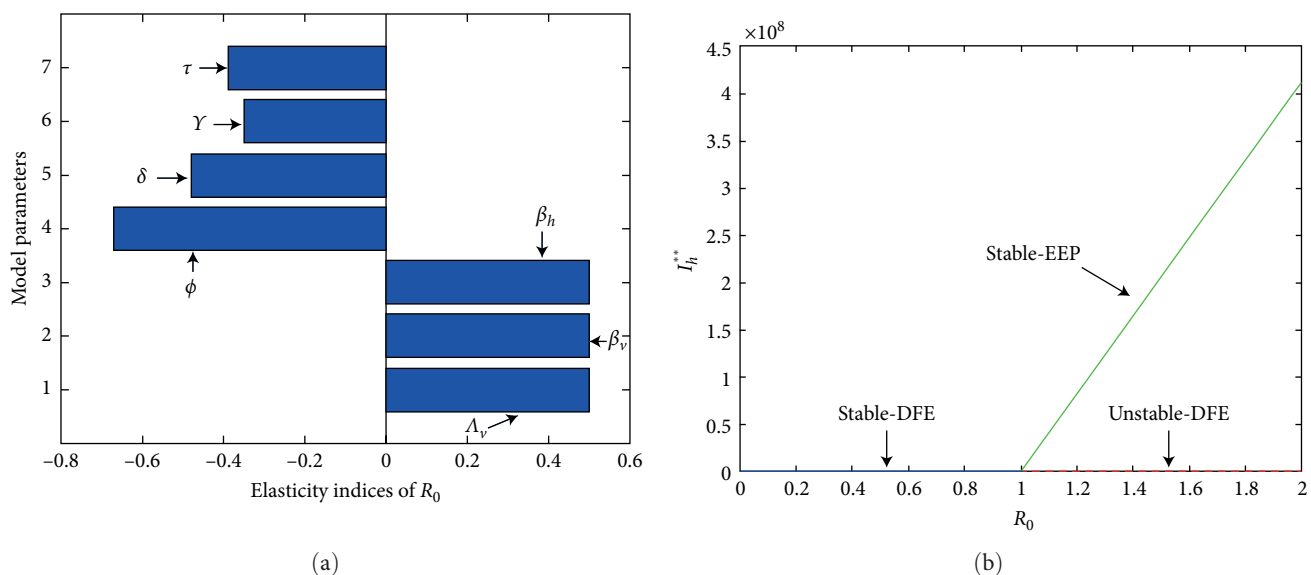


FIGURE 4: Simulations reflect the sensitivity and bifurcation analysis of model system (1): (a) the numerical result of sensitivity indices of the model system and (b) the forward bifurcation phenomenon of the model system.

The simulation of sensitivity analysis is displayed in Figure 4(a). The baseline parameter values with positive index will result a major effect on the frequency of the ailment spread, while parameters with negative index help to reduce the disease. In Figure 4(b), for $R_0 < 1$, the model system (1) has stable DFE, and it has no endemic equilibrium point. While for $R_0 > 1$, a stable endemic equilibrium appeared, and a stable disease-free became unstable. Hence, the transcritical bifurcation arises.

Figure 5(a) depicts that a large value of τ or γ or δ or ϕ in the presence of β_h can lead to a small value of R_0 . That is to say, if we increase the awareness creation, recovery rate of infected individuals and environmental cleaning (chemical

spraying), the spread of malaria disease will decrease in the population. Figure 5(b) reflects that a large value of τ or ϕ leads to a small value of R_0 . Therefore, by increasing public awareness creation and chemical spraying, we can reduce the spread of malaria disease.

6.1. Numerical Results of the Optimal Control Model. In this section, we examine the effects of various control measures on the eradication of malaria disease through some numerical results.

Figures 6(a) and 6(b) show the effectiveness of public awareness in the reduction of both infected human and

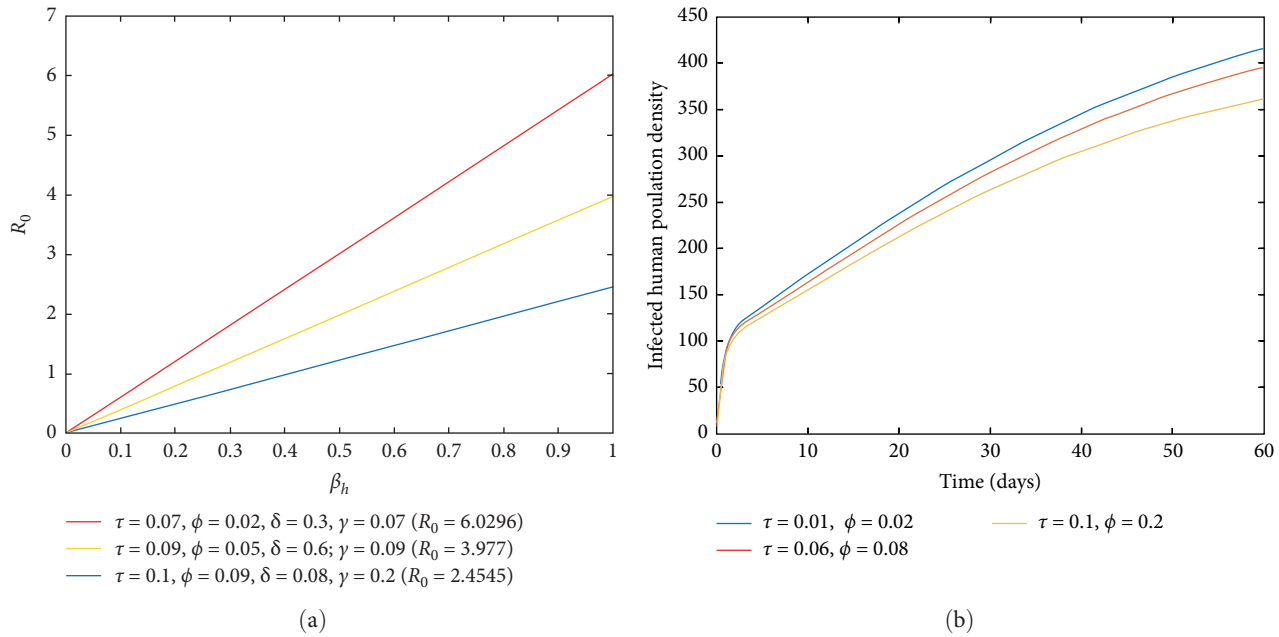


FIGURE 5: The effect of newly added model parameters on R_0 and infected human populations: (a) the effects of τ, δ, ϕ , and γ on R_0 in the presence of β_h and (b) the effect of τ and ϕ on the number of infected human populations.

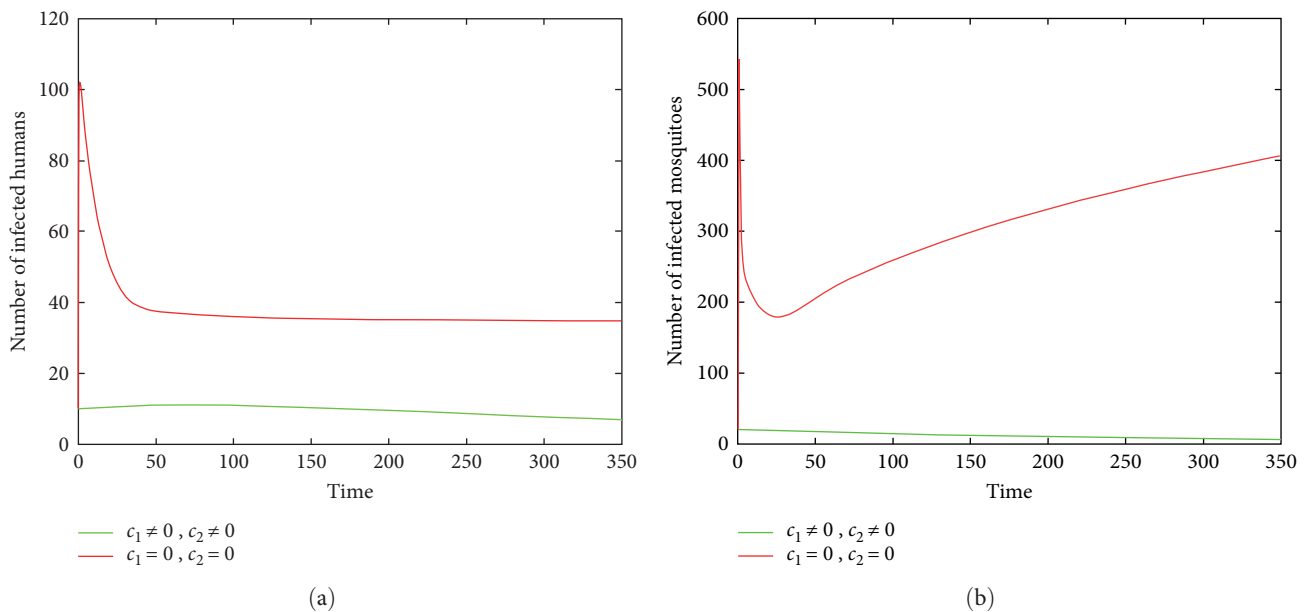


FIGURE 6: Simulations present the dynamics of infected humans and infected mosquitoes populations with and without control measures c_1 and c_2 : (a) the dynamics of infected human populations with and without control measures c_1 and c_2 and (b) the dynamics of infected mosquito populations with and without control measures c_1 and c_2 .

mosquitoes population. As we observed, in the absence of a public awareness campaign, the number of infected mosquitoes increases and, in turn, this leads to an increase in the numbers of the infected human population, while in the presence of a public awareness campaign, the numbers of infected mosquitoes dramatically decrease and also infected human populations decrease at the same time. Therefore, in order to stop the spread of malaria, policymakers must focus on raising public awareness to the fullest extent possible and

ensuring that there are enough preventive controls available. Figures 7(a) and 7(b) show that the number of recovered human populations is higher under the control strategy than in the case without control, while the number of susceptible mosquito populations is lower under the control strategy than in the case without control. Therefore, in order to stop the spread of malaria in the community, policymakers and the government must place a high priority on providing ongoing treatment for those who are infected.

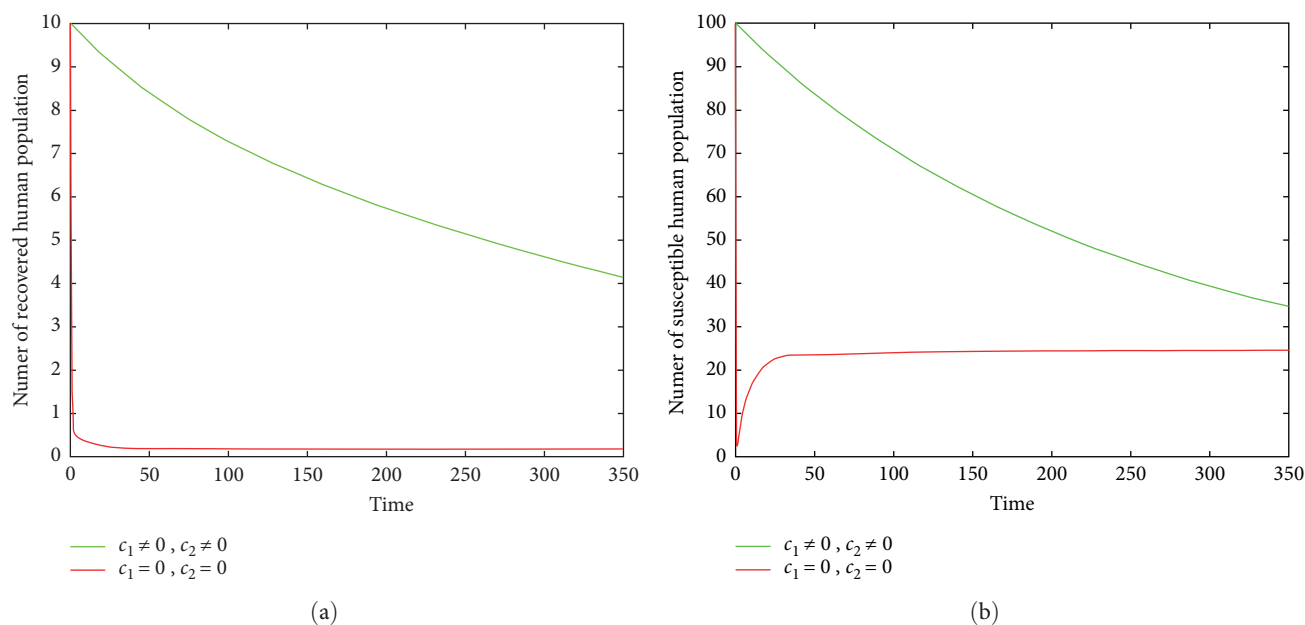


FIGURE 7: Simulations present the dynamics of recovered humans and susceptible mosquitoes populations with and without control measure c_3 : (a) the dynamics of recovered human populations with and without control measure c_3 and (b) the dynamics of susceptible mosquito populations with and without control measure c_3 .

7. Conclusion

In this study, a nonlinear deterministic mathematical model of malaria transmission is formulated and analyzed, dividing susceptible into another aware human population. Under the given domain, the mathematical as well as epidemiological well-posedness of the model is proved. Malaria disease reproduction number (threshold) of the model was obtained and provided the scenarios to stick or eradicate in the population. The stability of the disease-free and endemic equilibrium points was examined, and the DFE point was found to be asymptotically stable when $R_0 < 1$ and the existence of bifurcation at $R_0 = 1$ was analyzed, and it was shown that when $R_0 = 1$ the model undergo forward bifurcation phenomenon. We found that $R_0 < 1$ is the sufficient condition to eradicate malaria disease.

The sensitivity analysis of the basic reproduction number of the model revealed that parameters with negative indices are likely to decrease the disease while parameters with positive indices are likely to increase the expansion of malaria disease in the society. The numerical simulation of the model revealed that the numbers of infected human populations decrease when the biting rate of susceptible humans by infected mosquitoes decrease. The qualitative study proved the existence and characterization of an optimal control solution. Also, various control strategies were tested and showed that the combination of continuous public awareness creation and access to adequate control measures are more effective in the control of malaria disease transmission.

Data Availability

The data used to supplement the findings of this study are from previously published articles and cited in relevant places.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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