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A Mathematical Model of Malaria Transmission in Democratic Republic of the Congo

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Abstract

In this paper, we used an SIR-SI model to formulate a mathematical model of malaria transmission in Democratic Republic of the Congo. We evaluated the basic reproduction number, and studied the existence of local stability of disease-free and endemic equilibria. The global stability of the disease-free equilibrium (DFE) and endemic equilibrium was also studied. It was shown that the model exhibits backward bifurcation phenomenon where the stable (DFE) exists with a stable endemic equilibrium. Numerical simulations were conducted to confirm our analytic results. Next, we used the optimal control (the use of treated bet net $u_1(t)$ and treatment with drugs $u_2(t)$) as a tool for reducing the total number of malaria infected individuals and that of infected mosquitoes respectively. Our numerical simulations results indicate that the number of infected individuals always lead to decrease under the use of treated bet net and treatment with drugs controls respectively.

Keywords: Malaria; Stability Analysis; Backward Bifurcation; Optimal Control.

Introduction

Malaria is a contagious disease caused by infected female Anopheles mosquitoes. In 2016, 216 million malaria cases occurred in the world compared with 237 and 211 million cases in 2010 and 2015 respectively. Fifteen countries are accounted for 80% of global malaria deaths, all of them are in Africa (Sub-Saharan Africa), except India [1]. The Democratic Republic of the Congo (DRC) is one of the two highest leading contributors to the global burden of illness due to malaria [2]. Daily in the DRC, more than 400 children die, and nearly half of the deaths is caused by malaria [3]. Mathematical models are very important tools in understanding the dynamics of the vector-transmitted disease such as malaria. Furthermore, optimal control is a very useful and important field in both theoretical and applications. A lot of research has been conducted on the mathematical modeling of malaria transmission since the first model was introduced by Ronald Ross [4]. Juan Wnang, *et al.* considered SIR-SI malaria endemic model [5]. They derived the $\mathcal{R}_0$ and investigated the local and global stability of disease free equilibrium. They proved that the unique endemic equilibrium is globally asymptotically stable under certain specific conditions. According to their results their model shows backward bifurcation when $\mathcal{R}_0 < 1$. Edoardo Beretta, *et al.* analyzed a mathematical model for malaria transmission with asymptomatic carriers in a two age groups in human population [6]. Their results show that the key parameters that can be identified such as a threshold level, built on these parameters for example mosquitoes biting rate and the mortality rate of mosquitoes. When $\mathcal{R}_0 < 1$ the endemic equilibrium exist, and when $\mathcal{R}_0 < 1$ the disease die out. Xiaomei Feng, *et al.* considered a deterministic malaria transmission model with standard incidence rate and treatment, with the assumption that a part of the individuals who are recovered or treated from the disease spontaneously, shift to the recovered class with temporary immunity, while others re-enter the susceptible class [7]. Their stability analysis shows that when $\mathcal{R}_0 < 1$, two endemic equilibrium may exists and the backward bifurcation is possible for malaria to persist even if $\mathcal{R}_0 < 1$. Their numerical simulations support their analytical results. They used their model to simulate real data from China 2003-2013. According to their simulation results malaria can be eradicated in China. They computed their $\mathcal{R}_0$ and obtained 0.0161. G.R. Kelatlhegile, *et al.* formulated and analyzed a mathematical model for the prevention of a disease that spread horizontally and vertically in a population of varying size [8]. They investigated the stability analysis of their model and their results show that there exists more than one endemic equilibrium. for the model when $\mathcal{R}_0 < 1$. Their simulation results justified their analytical results. S.kim, *et al.* Studied and analyzed SIR-SI vector-

bias malaria transmission model to determine how it affects the dynamics of the disease [9]. Their results show that considering the vector-bias effect in different areas help to predicate the dynamics future of malaria and to make decisions for establishing control strategy. Liming Cai, *et al*. introduced a mathematical model for malaria transmission with an asymptomatic compartment in human and exposed compartments in both human and mosquito [10]. They investigated the stability analysis of their model and their simulation results suggest that the total spread of the disease is high if all individuals show symptoms upon infection. Furthermore, they applied the optimal control theory to the model by using bed-net and treatment as important tools for reducing the number of symptomatic and asymptomatic individuals. According to their simulation results the optimal control strategies always lead to decrease in the symptomatic infectious individuals and may lead to increase the number of asymptomatic infectious individuals. S.R. Gani and S.V. Halawar formulated a nonlinear mathematical model to control the spread of infectious disease using the role of awareness programs by media and antiviral treatment [11]. According to their results the most effective strategies to control the disease are the combination of 3 control measures (the successful campaign of awareness programs by media, controlling effort that alters infectious cases receiving treatment and strengthening effort made on awareness campaign programs) are the most cost effective control strategies and this indicates that the implementation of the three control measures is necessary in order to control the disease outbreak. D. Khamis, *et al*. used an optimal control frame work based on coupled models of mosquito pop- ulation dynamics malaria epidemiology to investigate the cost-effectiveness of combining vector control with drug therapies in homogeneous environments with and without vector control migration [12]. Their results shows that the combining vector control and drug therapies is the most effective and efficient use of resources. Recently, Fuzzy Optimal Control is the most useful in control theory see [13-15].

In this article, we formulate a new model, which is different from (J.Wang, *et al*.) and all the above mentioned mathematical models of malaria transmission in the sense that, we divided recovered human population into two groups, the first group has partial immunity after recovery and they re-enter the susceptible class again. The other group may be infected immediately again after recovery, by an infectious mosquitoes with transmission probability of recovered human $\delta < 1$, which is less than the probability of susceptible human [5].

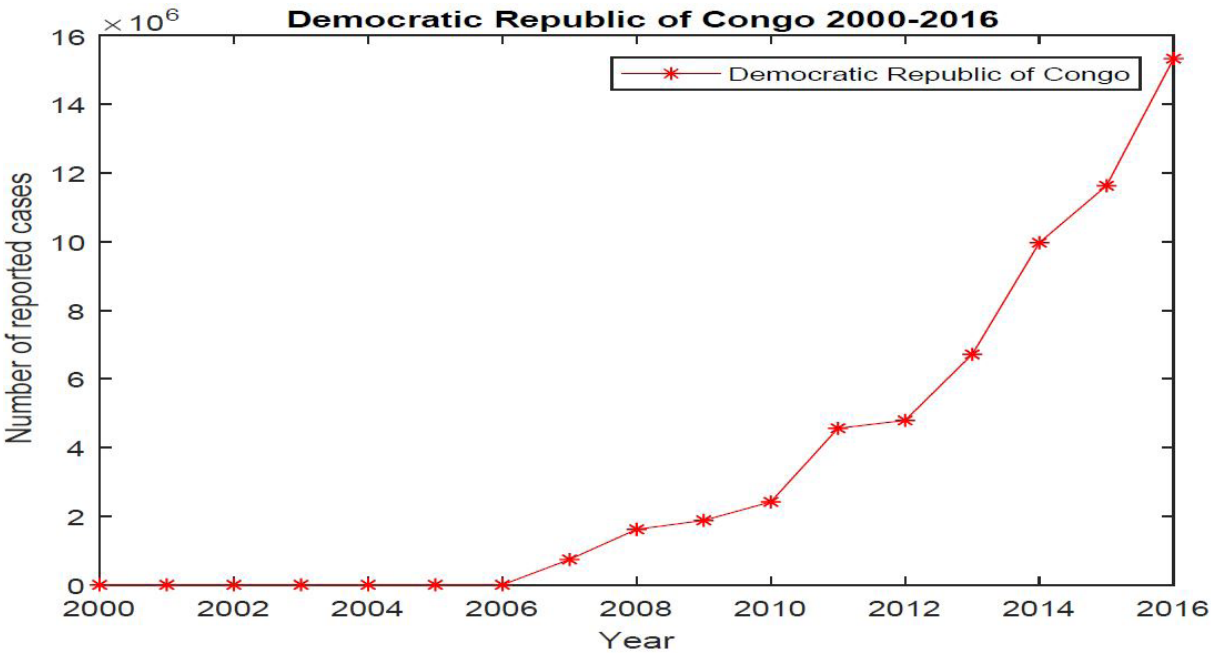

Figure 1: Democratic Republic of the Congo, Reported cases of malaria from WHO [23,24]

Moreover, we applied the optimal control theory to our model in terms of treated bed-net use and treatment with drugs to control the disease. This article is structured as follows: we introduced the model description in Section 2. In Section 3, stability analysis of the model: including positivity and boundedness of the solution, existence of equilibria and backward bifurcation respectively, and global asymptotic stability of diseasefree equilibrium is proved. Numerical simulations have been presented in Section 4. Sensitivity analysis of R_0 is given in Section 5. In Section 6, numerically, we investigated optimal control strategy and applied it to (bet-net use and treatment with drugs). Section 7, is made up of conclusion of our article.

Mathematical Model

Model Description

The human population of our model is divided into three classes: Susceptible human $S_h(t)$, Infectious human $I_h(t)$, and Recovered human $R_h(t)$ and the mosquitoes population is in two classes: Susceptible mosquito $S_m(t)$, and Infectious mosquito $I_m(t)$. $N_h(t)$ and

$N_m(t)$ denote the total population of human and mosquitoes respectively, so that $N_h(t) = S_h + I_h + R_h$ and $N_m = S_m + I_m$. We assume that the bite from mosquito is randomly and the recovered humans are in two groups as mentioned earlier, one of the groups with partial immunity re-enter the susceptible class again and the other without immunity become infectious again. Also we assumed that the total population of the human and mosquito are constant and that mosquitoes do not recover from the infected class. All the above mentioned assumptions lead to the following system:

$$\begin{cases} \frac{dS_h}{dt} = \mu N_h - \frac{a\gamma_h S_h I_m}{N_h} - \mu S_h + \rho R_h, \\ \frac{dI_h}{dt} = \frac{a\gamma_h S_h I_m}{N_h} - (\mu + \alpha) I_h + \frac{a\delta\gamma_h R_h I_m}{N_h}, \\ \frac{dR_h}{dt} = \alpha I_h - \frac{a\delta_h R_h I_m}{N_h} - (\mu + \rho) R_h, \\ \frac{dS_m}{dt} = \psi N_m - \frac{a\gamma_m I_h S_m}{N_h} - \psi S_m, \\ \frac{dI_m}{dt} = \frac{a\gamma_m I_h S_m}{N_h} - \psi I_m(t), \end{cases} \tag{2.1}$$

With: $S_h(0) > 0$, $I_h(0) \geq 0$, $R_h(0) \geq 0$, $S_m(0) > 0$, $I_m(0) \geq 0$.

Where the average life span of humans in endemic area is $\frac{1}{\mu}$ that is μ is the natural death rate of humans. Similarly, the average life span of mosquitoes is $\frac{1}{\psi}$, where ψ is the natural death rate of mosquitoes. Moreover *a* is the mosquitoes biting rate and the human can loss their immunity at rate ρ. The humans and mosquitoes transmission rates are γ_h and γ_m respectively. Also the infectious individual can be recovered at rate α and the transmission property of the human recovery is given by δ. In addition to that, all the parameter values were listed in Table 2.

Model Analysis

Positivity and boundedness of the solutions

Since the total population of human and mosquitoes are constants, that is $\frac{dN_h}{dt} = \frac{dN_m}{dt} = 0$, then in order to simplify model (2.1) we normalize the human and mosquito population. We denote $s_h = \frac{S_h}{N_h}$, $i_h = \frac{I_h}{N_h}$, $rh = \frac{R_h}{N_h}$, $s_m = \frac{S_m}{N_m}$, $i_m = \frac{I_m}{N_m}$, $q = \frac{N_m}{N_h}$, and define in the positive region as

$$\Sigma = \{(s_h, i_h, r_h, s_m, i_m) \in R_+^5 : 0 \leq s_h + i_h + r_h = 1, \ 0 \leq S_m + i_m \leq 1\}$$

Sine $s_m + i_m = N_m = 1$, this implies that $s_m = 1 - i_m$, the system (2.1) can be written as

$$\begin{cases} \frac{ds_h}{dt} = \mu - aq\gamma_h s_h i_m - \mu s_h + \rho r_h, \\ \frac{di_h}{dt} = aq\gamma_h s_h i_m - (\mu + \alpha) i_h + aq\delta\gamma_h r_h i_m, \\ \frac{dr_h}{dt} = \alpha i_h - aq\delta\gamma_h r_h i_m - (\mu + \rho) r_h, \\ \frac{ds_m}{dt} = \psi - a\gamma_m i_h s_m - \psi s_m, \\ \frac{di_m}{dt} = a\gamma_m i_h s_m - \psi i_m, \end{cases} \tag{3.1}$$

Sine $s_m = 1 - i_m$ then we can omit s_m from the model (3.1), and rearranged as

$$\begin{cases} \frac{ds_h}{dt} = \mu - aq\gamma_h s_h i_m - \mu s_h + \rho r_h, \\ \frac{di_h}{dt} = aq\gamma_h s_h i_m - (\mu + \alpha) i_h + aq\delta\gamma_h r_h i_m, \\ \frac{dr_h}{dt} = \alpha i_h - aq\delta\gamma_h r_h i_m - (\mu + \rho) r_h, \\ \frac{di_m}{dt} = a\gamma_m (1 - i_m) i_h - \psi i_m, \end{cases} \tag{3.2}$$

Where the model (3.2) is defined on the feasible region

$$\Upsilon = \{(s_h, i_h, r_h, i_m) \in R_+^4 : 0 \leq s_h + i_h + r_h = 1, \ 0 \leq i_m \leq 1\},$$

Existence of Equilibria

It is clear that the system (3.2) has a disease-free equilibrium $P_0 = (1, 0, 0, 0)$. We used the next generation matrix method in [16,17], to find the reproduction number $\mathcal{R}_0$ at P_0, defined F as the matrix for new infectious individuals and V is the matrix of remaining transfer individuals. For more information about F,V and the next generation matrix technique see [16-19].

$$F = \begin{bmatrix} aq\gamma_h s_h i_m + a\delta\gamma_h r_h i_m \\ 0 \\ a\gamma_m (1 - i_m) i_h \end{bmatrix}, \quad \text{and} \quad V = \begin{bmatrix} (\mu + \alpha) i_h \\ -\alpha i_h + aq\delta\gamma_h r_h i_m + (\mu + \rho) r_h \\ \psi i_m \end{bmatrix},$$

Differentiate F and V partially with respect to: $i_h,\ r_h,\ i_m$ at $P_0(1,\ 0,\ 0,\ 0)$ we obtained:

$$f = \begin{bmatrix} 0 & 0 & aq\gamma_h \\ 0 & 0 & 0 \\ a\gamma_m & 0 & 0 \end{bmatrix}, \quad \text{and} \quad v = \begin{bmatrix} (\mu + \alpha) & 0 & 0 \\ -\alpha & (\mu + \rho) & 0 \\ 0 & 0 & \psi \end{bmatrix},$$

$$fv^{-1} = \begin{bmatrix} 0 & 0 & aq\gamma_h \\ 0 & 0 & 0 \\ a\gamma_m & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{(\mu+\alpha)} & 0 & 0 \\ \frac{\alpha}{(\mu+\alpha)(\mu+\rho)} & \frac{1}{(\mu+\alpha)} & 0 \\ 0 & 0 & \frac{1}{\psi} \end{bmatrix} = \begin{bmatrix} 0 & 0 & \frac{aq\gamma_h}{\psi} \\ 0 & 0 & 0 \\ \frac{a\gamma_m}{(\mu+\alpha)} & 0 & 0 \end{bmatrix},$$

The reproduction number $\mathcal{R}_0$ is given by the spectral radius of fv^{-1} which is denoted by $\sigma(fv^{-1})$ and defined as:

$$\mathcal{R}_0 = \sigma\left(fv^{-1}\right) = \sqrt{\frac{a^2 q\gamma_h\gamma_m}{\psi(\mu + \alpha)}} \tag{3.3}$$

Where σ is the spectral radius of the next generation matrix fv^{-1}.

Local stability of disease-free equilibrium: In this subsection, we investigate the stability of disease-free equilibrium P_0 by considering the model (3.2), by taking the Jacobian matrix and obtained

$$J(P_0) = \begin{bmatrix} -\mu & 0 & \rho & -aq\gamma_h \\ 0 & -(\mu + \alpha) & 0 & aq\gamma_h \\ 0 & \alpha & -(\mu + \rho) & 0 \\ 0 & a\gamma_m & 0 & -\psi \end{bmatrix} = 0,$$

to find the eginvalues of the above matrix we use determinant

$$|J(P_0) - \lambda I| = \begin{vmatrix} -\mu - \lambda & 0 & \rho & -aq\gamma_h \\ 0 & -(\mu + \alpha) - \lambda & 0 & aq\gamma_h \\ 0 & \alpha & -(\mu + \rho) - \lambda & 0 \\ 0 & a\gamma_m & 0 & -\psi - \lambda \end{vmatrix} = 0,$$

the two eigenvalues of the above jacobian matrix are clearly negative $(i.e\ \lambda_1 = -\mu\ and\ \lambda_2 = -(\mu + p))$. The other two roots can be characterized as:

$$\lambda^2 + b_1\lambda + b_2 = 0, \tag{3.4}$$

Where

$$b_1 = \mu + \alpha,$$
$$b_2 = \psi(\mu + \alpha)\left(1 - \mathcal{R}_0^2\right),$$

and $\mu > 0,\ \alpha > 0,$ so $b_1 > 0$.

Using the Routh-Hurwitz Criterion [20] it can be seen that the two eigenvalues of the characteristic equation (3.4) have negative real parts if and only if

$$b_1 > 0, b_1 b_2 > 0, \tag{3.5}$$

if $\mathcal{R}_0 < 1$, then $b_2 > 0$, hence $b_1 b_2 > 0$,

Thus, we have the following theorem.

Theorem 3.1. *The disease-free equilibrium P_0 of model (3.2) is locally asymptotically stable if and only if $\mathcal{R}_0 < 1$ and the inequalities (3.5) are satisfied.*

Endemic Equilibrium and Bifurcation: To find the endemic equilibrium of the system (3.2), let: $\frac{ds_h}{dt} = \frac{di_h}{dt} = \frac{dr_h}{dt} = \frac{ds_m}{dt} = \frac{di_m}{dt} = 0$ after simple calculation we get an endemic equilibrium $P^* = \left(S_h^*,\ i_h^*,\ r_h^*,\ i_m^*\right)$ for the model (3.2) as

$$\begin{aligned} s_h^* &= \frac{a\mu\gamma_m(1-i_m^*)((\mu+\rho)+aq\delta\gamma_h i_m^*)+\rho\alpha\psi i_m^*}{(aq\gamma_h i_m^*+\mu)(a\gamma_m(1-i_m^*)((\mu+\rho)+aq\delta\gamma_h i_m^*))}, \; i_h^* = \frac{\psi i_m^*}{a\gamma_m\,(1-i_m^*)}, \\ r_h^* &= \frac{\alpha\psi i_m^*}{a\gamma m(1-i_m^*)((\mu+\rho)+aq\delta\gamma_h i_m^*)}, \end{aligned} \tag{3.6}$$

Then we substitute equation (3.6) into the second equation of system (3.2), to obtain a quadratic equation defined by

$$g\,(i_m^*) = c_1 i_m^{*2} + c_2 i_m^* + c_3 = 0, \tag{3.7}$$

Where:

$$\begin{aligned} c_1 &= a^2 q^2 \delta \gamma_h^2 \psi \mu, \\ c_2 &= aq\delta\mu^2\psi\gamma_h + aq\psi\gamma_h\mu(\mu+\rho+\alpha) + a\mu q\gamma_h\gamma_m\,(a(\mu+\rho) + q\gamma_h(\delta - a)), \\ c_3 &= (\mu+\alpha)(\mu+\rho)\psi\mu\left[1-\mathcal{R}_0^2\right], \end{aligned} \tag{3.8}$$

Case	c_1	c_2	c_3	$\mathcal{R}_0$	No. of sign changes	No. of possible real roots
1	+	+	+	$\mathcal{R}_0 < 1$	0	0
2	+	+	-	$\mathcal{R}_0 > 1$	1	1
3	+	-	+	$\mathcal{R}_0 < 1$	2	0,2
4	+	-	-	$\mathcal{R}_0 > 1$	1	1

Table 1: Number of possible real roots of equation (3.7)

Consequently, from equations (3.7), (3.8) and Table 1, it is easy to see that the possibility of more than one endemic equilibria exist, thus, backward bifurcations phenomenon may occur. Consequently, we have the following theorem.

Theorem 3.2. The system (3.2) has a unique endemic equilibrium P^*, if $\mathcal{R}_0 > 1$ and When cases 2, 4 are satisfied and the system (3.2) could have more than one endemic equilibrium if $\mathcal{R}_0 < 1$ and case 3 is satisfied

That is the following conditions are true for model (3.2)

(i) if $c_3 < 0$ then $\mathcal{R}_0 > 1$, exactly the system (3.2) have one endemic equilibrium.
(ii) Exactly one unique endemic equilibrium if $c_2 < 0$ and $c_2 = 0$ or $c_2^2 - 4c_2c_3 = 0$,
(iii) Exactly two endemic equilibrium if $c_3 > 0$, (i.e. $\mathcal{R}_0 < 1$), $c_2 < 0$, and $c_2^2 - 4c_1c_3 > 0$,
(iv) No endemic equilibrium otherwise.

The system (3.1) can be written in the form $x'(t) = F(X)$, where $X = (x_1, x_2, x_3, x_4, x_5)^T = (s_h, i_h, r_h, s_m, i_m)^T$ *and* $F(X) = (f_1, f_2, f_3, f_4, f_5)^T$, denotes the right hand sides of the system (3.2) as:

$$\begin{cases} \frac{dx_1}{dt} = f_1 = \mu - aq\gamma_h x_1 x_5 - \mu x_1 + \rho x_3, \\ \frac{dx_2}{dt} = f_2 = aq\gamma_h x_1 x_5 - (\mu+\alpha)x_2 + aq\delta\gamma_h x_3 x_5, \\ \frac{dx_3}{dt} = f_3 = \alpha x_2 - aq\delta\gamma_h x_3 x_5 - (\mu+\rho)x_3, \\ \frac{dx_4}{dt} = f_4 = \psi - a\gamma_m x_2 x_5 - \psi x_5, \\ \frac{dx_5}{dt} = f_5 = a\gamma_m x_2 x_4 - \psi x_5, \end{cases} \tag{3.9}$$

We evaluate the Jacobian matrix of the system (3.9) at P_0, which is denoted by $J(P_0)$, and it is given by

$$J(P_0) = \begin{bmatrix} -\mu & 0 & \rho & 0 & -aq\gamma_h \\ 0 & -(\mu+\alpha) & 0 & 0 & aq\gamma_h \\ 0 & \alpha & -(\mu+\rho) & 0 & 0 \\ 0 & -a\gamma_m & 0 & -\psi & 0 \\ 0 & a\gamma_m & 0 & 0 & -\psi \end{bmatrix} = 0,$$

Next, we consider $\mathcal{R}_0 = 1$. Furthermore, let $\gamma_h = \gamma_h^*$ chosen to be the bifurcation parameter. Solving equation (3.3) for γ_h with $\mathcal{R}_0 = 1$ yields

$$\gamma_h = \gamma_h^* = \frac{(\mu + \alpha)\psi}{a^2 q \gamma_m},$$

after simple calculation it is easy to see that $J(P_0)$ with $\gamma_h = \gamma_h^*$ denoted by $J_{\gamma_h^*}$, has a simple zero eigenvalue and other eigenvalues have negative real part. i.e $\left(\lambda_1 = 0,\ \lambda_1 = -\mu,\ \lambda_3 = -(\mu+\rho),\ \lambda_4 = -\frac{1}{2}(\alpha+2\mu) - \frac{1}{2}\sqrt{\alpha^2 - 4a^2 q\gamma_h\gamma_m},\ \lambda_4 = -\frac{1}{2}(\alpha+2\mu) + \frac{1}{2}\sqrt{\alpha^2 - 4a^2 q\gamma_h\gamma_m}\right)$.

Hence, the dynamics of the system (3.8) satisfy the conditions of the centre manifold theory [21]. Moreover, we show that the system (3.8) is equivalent to system (3.2), underdoes backward bifurcation at $\mathcal{R}_0 = 1$, then we apply the Center Manifold Theorem to test the dynamics of (3.2) close to $\gamma_h = \gamma_h^*$.

The Jacobian matrix of system (3.2), denoted by $J(P_0)$ at $\gamma_h = \gamma_h^*$, has a right and a left eigenvectors associated with zero eigenvalue, given by: $w = (w_1, w_2, w_3, w_4, w_5)^T$, and $v = (v_1, v_2, v_3, v_4, v_5)$ respectively.

Where

$$w_1 = \frac{-aq\gamma_h^*(\mu+\alpha+\rho)w_5}{(\mu+\rho)(\mu+\rho)}, w_2 = \frac{aq\gamma_h^* w_5}{(\mu+\alpha)}, w_3 = \frac{a\alpha q\gamma_h^* w_5}{(\mu+\rho)(\mu+\alpha)}, w_4 = \frac{-a^2\gamma_m q\gamma_h^* w_5}{\psi(\psi+\alpha)}, w_5 = w_5. \tag{3.10}$$

and

$$v_1 = 0,\ v_2 = v_2,\ v_3 = 0,\ v_4 = 0,\ v_5 = \frac{aq\gamma_h^* v_2}{\psi}. \tag{3.11}$$

In order to show the existence of backward bifurcation, we compute $\frac{\partial^2 f_i}{\partial x_i \partial x_j}$, where i, j = 1, 2, 3, 4, 5 at P_0 and obtain

$$\begin{aligned} &\frac{\partial^2 f_2}{\partial x_1 \partial x_5} = \frac{\partial^2 f_2}{\partial x_5 \partial x_1} = aq\gamma_h,\ \frac{\partial^2 f_2}{\partial x_3 \partial x_5} = \frac{\partial^2 f_2}{\partial x_5 \partial x_3} = aq\delta\gamma_h,\ \frac{\partial^2 f_2}{\partial x_2 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_2} = a\gamma_m \\ &\frac{\partial^2 f_5}{\partial x_2 \partial x_4} = \frac{\partial^2 f_5}{\partial x_4 \partial x_2} = a\gamma_h, \end{aligned} \tag{3.12}$$

Now we compute a and b defined in Theorem 4.1 [21] of Castillo-Chavez and Song as follows

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}\left(0,\ \gamma_h^*\right) = \sum_{i,j=1}^{n} v_2 w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}\left(0,\ \gamma_h^*\right) + \sum_{i,j=1}^{n} v_5 w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j}\left(0,\ \gamma_h^*\right) \tag{3.13}$$

$$\begin{aligned} a &= \sum_{k,i,j=1}^{n} 2v_2 w_1 w_5 \frac{\partial^2 f_2}{\partial x_1 \partial x_5}\left(0,\ \gamma_h^*\right) + \sum_{i,j=1}^{n} 2v_2 w_3 w_5 \frac{\partial^2 f_2}{\partial x_3 \partial x_5}\left(0,\ \gamma_h^*\right) + \sum_{i,j=1}^{n} 2v_5 w_2 w_4 \frac{\partial^2 f_5}{\partial x_2 \partial x_5}\left(0,\ \gamma_h^*\right) \\ &= 2aq\gamma_h v_2 w_1 w_5 + 2aq\delta\gamma_h v_2 w_3 w_5 + 2a\gamma_m v_5 w_2 w_4 \end{aligned}$$

and

$$b = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial \gamma_h}\left(0,\ \gamma_h^*\right) = \sum_{i,j=1}^{n} v_2 w_i \frac{\partial^2 f_2}{\partial x_i \partial \gamma_h}\left(0,\ \gamma_h^*\right) = a^2 q^2 \gamma_h^* v_2^2 > 0 \tag{3.14}$$

Since the factor b is always positive, then the model (3.2) undergoes backward bifurcation at $R_0 = 1$, whenever

$$a = 2aq\gamma_h v_2 w_1 w_5 + 2aq\delta\gamma_h v_2 w_3 w_5 + 2a\gamma_m v_5 w_2 w_4 > 0, \tag{3.15}$$

Then we can establish from the above discussions the following theorem.

Theorem 3.3. *The system (3.2) undergoes a backward bifurcation at $\mathcal{R}_0$ whenever inequality (3.15) holds*

Local stability of endemic equilibrium

Theorem 3.4. For $\mathcal{R}_0 > 1$, the endemic equilibrium P^* of the system (3.2) is locally asymptotically stable if the condition (3.18) is satisfied, otherwise unstable.

Proof At P^*, the jacobian matrix J (P^*) evaluated as

$$J(P^*) = \begin{bmatrix} -\mu - z_1 & 0 & \rho & -z_2 \\ z_1 & -z_3 & z_4 & z_5 \\ 0 & \alpha & -z_6 & -z_5 \\ 0 & z_7 & 0 & -z_8 - \psi \end{bmatrix}, \tag{3.16}$$

where $z_1 = aq\gamma_h i_m^*, z_2 = aq\gamma_h s_h^*, z_3 = (\mu + \alpha), z_4 = aq\delta\gamma_h r_h^*, z_5 = aq\delta\gamma_h i_m^*, z_6 = (\mu + \rho)$, $z_7 = a\gamma_m (1 - i_m^*)$, $z_8 = a\gamma_m i_h^*$

The roots of the matrix (3.16) can be determined from the following characteristics equation

$$f(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0, \tag{3.17}$$

Where

$$a_1 = z_1 + z_3 + z_6 + z_8 + \mu + \psi,$$

$$a_2 = z_3 (z_1 + \mu) + z_6 (z_1 + \mu) + (z_1 + \mu) (z_8 + \psi) + z_3 z_6 + z_3 (z_8 + \psi) + z_6 (z_8 + \psi) - (\alpha z_4 + z_5 z_7),$$

$$a_3 = (z_1 + \mu) (z_3 z_6 + z_3 (z_8 + \psi) + z_6 (z_8 + \psi) - \alpha z_4 - z_5 z_7) + (z_3 z_6 - \alpha z_4) (z_8 + \psi) + z_5 z_7 (z_4 - z_6) + z_7 - \alpha\rho z_1,$$

$$a_4 = (z_3 z_6 - \alpha z_4) (z_1 + \mu) + (z_4 z_5 - z_5 z_6) z_7 (z_1 + \mu) + (z_5 z_7 - \alpha (z_8 + \psi)) \rho z_1 + z_1 z_2 + z_6 z_7.$$

The Rouuth-Hurtwiz criteria [22] for equation (3.17) will give four negative eigenvalues if and only if $a_i > 0$, for i = 1, 2, 3, 4 and the following condition is satisfied

$$a_1 a_2 a_3 > a_3^2 + a_1^2 a_4, \tag{3.18}$$

Thus, the system (3.2) about. And P^* is locally asymptotically stable if $\mathcal{R}_0 > 1$ and the condition (3.18) is satisfied.

Global Asymptotic Stability of Disease-Free Equilibrium

Theorem 3.5. The disease-free equilibrium P_0 of model (3.2) is globally asymptotically stable if $R_0 < 1$ and satisfies $\delta < 1$.

Proof. To prove the global stability of P_0 we define the Lyapunov function as

$$\begin{aligned} V &= a\gamma_m i_h + (\mu + \alpha) i_m \\ V' &= a\gamma_m i_h' + (\mu + \alpha) i_m' \\ &= a\gamma_m \left[aq\gamma_h s_h i_m - (\mu + \alpha) i_h + aq\delta\gamma_h r_h i_m\right] + (\mu + \alpha) \left[a\gamma_m i_h s_m - \psi i_m\right] \\ &\leq a\gamma_m aq\gamma_h (s_h + r_h) i_m - \psi(\mu + \alpha) i_m - (\mu + \alpha) a\gamma_m i_h i_m \\ &\leq \psi(\mu + \alpha) \left[\frac{a\gamma_m aq\gamma_h (s_h + r_h)}{\psi(\mu + \alpha)} - 1\right] i_m - (\mu + \alpha) a\gamma_m i_h i_m \\ &\leq \psi(\mu + \alpha) i_m \left[\mathcal{R}_0^2 (s_h + i_h) - 1\right] - (\mu + \alpha) a\gamma_m i_h i_m \\ &\leq \psi(\mu + \alpha) i_m \left[\mathcal{R}_0^2 - 1\right] - (\mu + \alpha) a\gamma_m i_h i_m \\ &\leq \psi(\mu + \alpha) i_m \left[\mathcal{R}_0^2 - 1\right] \end{aligned}$$

Then from $\mathcal{R}_0 < 1$ and $\delta < 1$, we have $V' = 0$ if and only if $i_m = 0$. And from the second equation of (3.2), we have $\lim_{t\to\infty} i_h(t) = 0$ also from the first and third equations of (3.2) we have $\lim_{t\to\infty} r_h(t) = 0$, and $\lim_{t\to\infty} s_h(t) = 1$, $\lim_{t\to\infty} s_m(t) = 1$, respectively. Furthermore, the largest compact invariant set in $(s_h, i_h, r_h, s_m, i_m) \in \Upsilon : V' = 0$ is the singleton P_0, where P_0= is the disease-free equilibrium point. Using the LaSalle's invariant principle, it implies that P_0 is globally asymptotically stable in Υ if $R_0 < 1$ and $\delta < 1$ Hence proved.

Numerical Simulations

We used our model to simulate malaria reported data of the DRC, Figure 1 shows that the reported malaria case was less than 2000. From 2000-2006, and started to increase after that to more than 15 million cases at 2016 [23,24]. However, we ignore the data from 2000-2006, from our simulations, because there was a war and conflicts between the local communities in DRC at that period of time. All the parameter values for our model are presented in Table 2. According to these parameter values, we implemented numerical simulations of our model and obtained a suitable fitting between the infected human of model (2.1) and the malaria reported cases of (DRC) from WHO, from 2007- 2016, see (Figures 2a and 2b). In (Figures 2a, 2b and Figure 4a) we used the parameter values in Table 2 to simulate the model compartments. Moreover the influence of initial size of susceptible mosquitoes on the number of human malaria cases in DRC (Figure 4b). The basic reproduction number of (DRC) is estimated to be 4.5589 and the disease is endemic. (Figures 5a and 5b) show that solution of the model (2.1) with parameter values given in Table 2 for Democratic Republic of the Congo.

Parameter	Parameter description	Value	References
a	Mosquitoes bitting rate	0.4	[25]
ψ	Natural death rate of mosquitoes	0.1	[25]
μ	Natural death rate of humans	4.72×10^{-5}	[26]
ρ	Loss of immunity rate for humans	2.74×10^{-3}	[27]
α	Recovery rate of humans	3.5×10^{-3}	[25]
γ_h	Transmission rate in humans	0.048	[27]
γ_m	Transmission rate in mosquito	0.48	[27]
δ	Transmission probability of recovered human	0.043	fitting

Table 2: Description of parameters of the model (2.1)

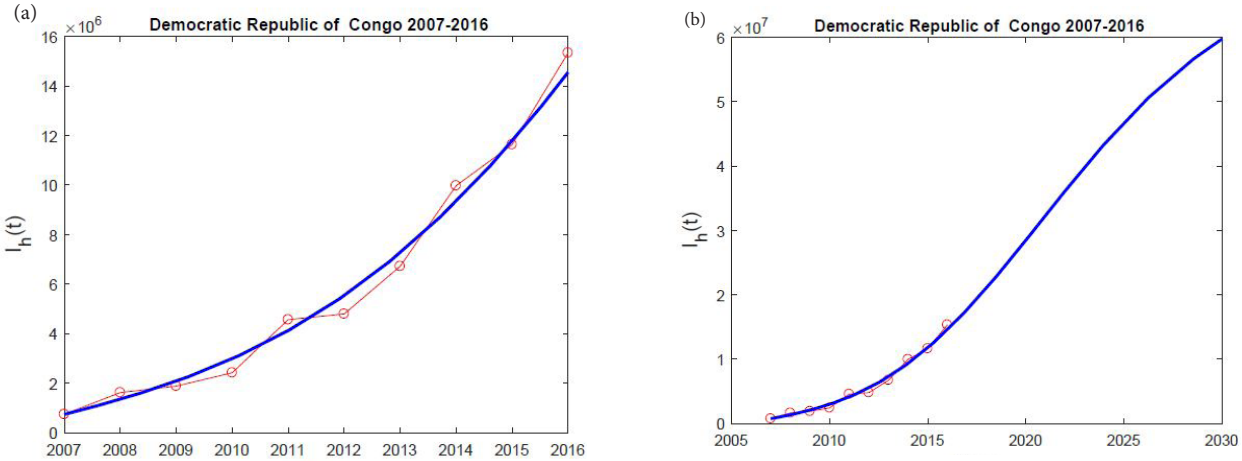

Figure 2: Comparisons of the reported malaria cases from WHO (red curve) and the solution of infectious human $I_h(t)$ for model (2.1), **(a)**: Simulation of malaria reported cases in Democratic Republic of the Congo from 2007 to 2016, **(b)**: Prediction of malaria cases for Democratic Republic of the Congo 2007 to 2030.

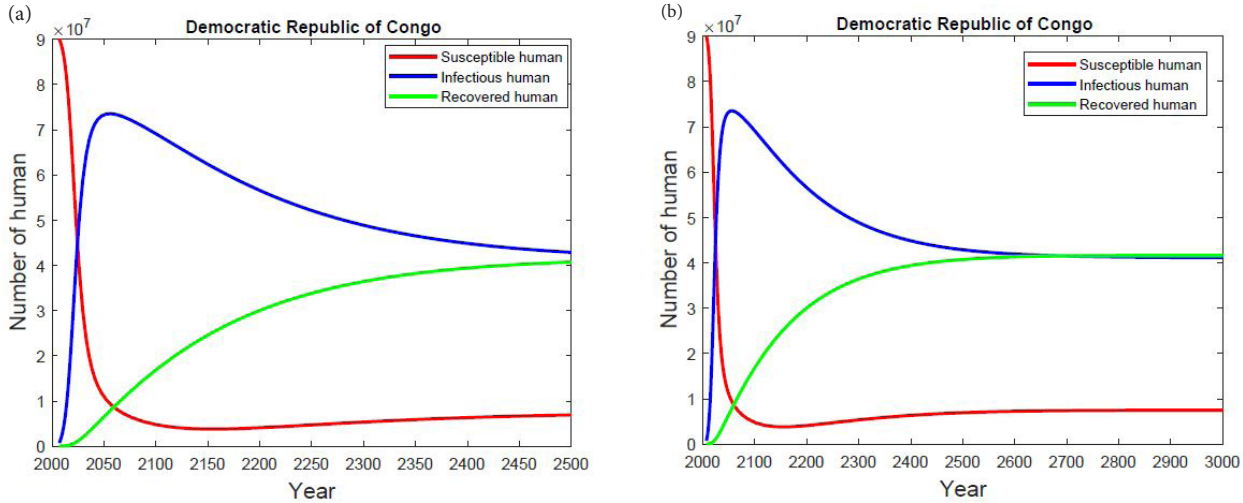

Figure 3: Solution of the model (2.1) with parameter for Democratic Republic of the Congo, **(a)** Simulation the number of human from 2007 to 2500, **(b)** Simulation the number of human from 2007 to 3000

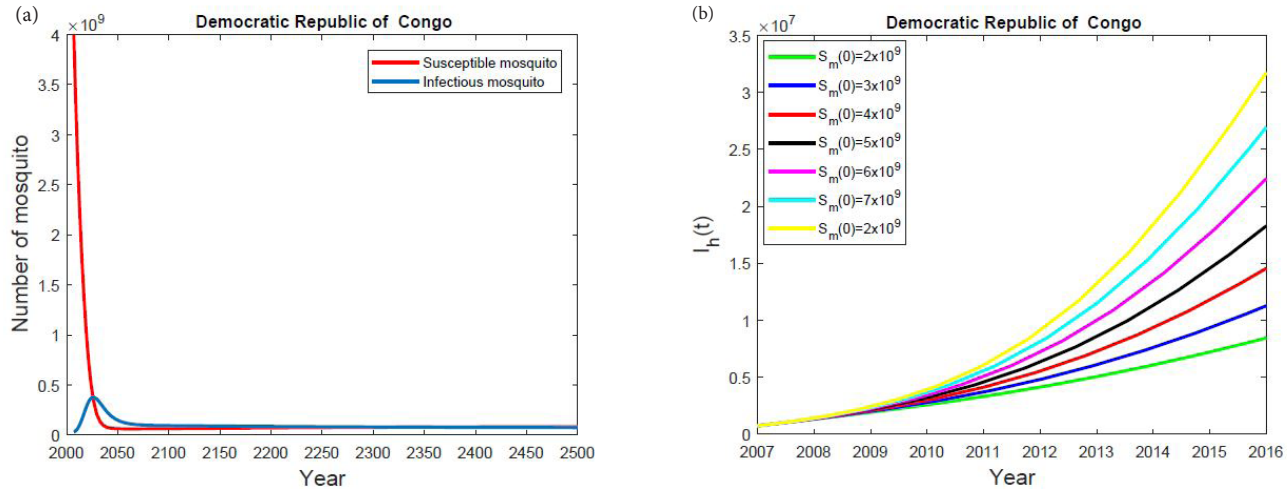

Figure 4: (a): Solution of the model (2.1) with parameters for Democratic Republic of the Congo with the number of mosquito from 2007 to 2500 **(b)**: The influence of initial size of susceptible mosquito on the number of human malaria in Democratic Republic of the Congo

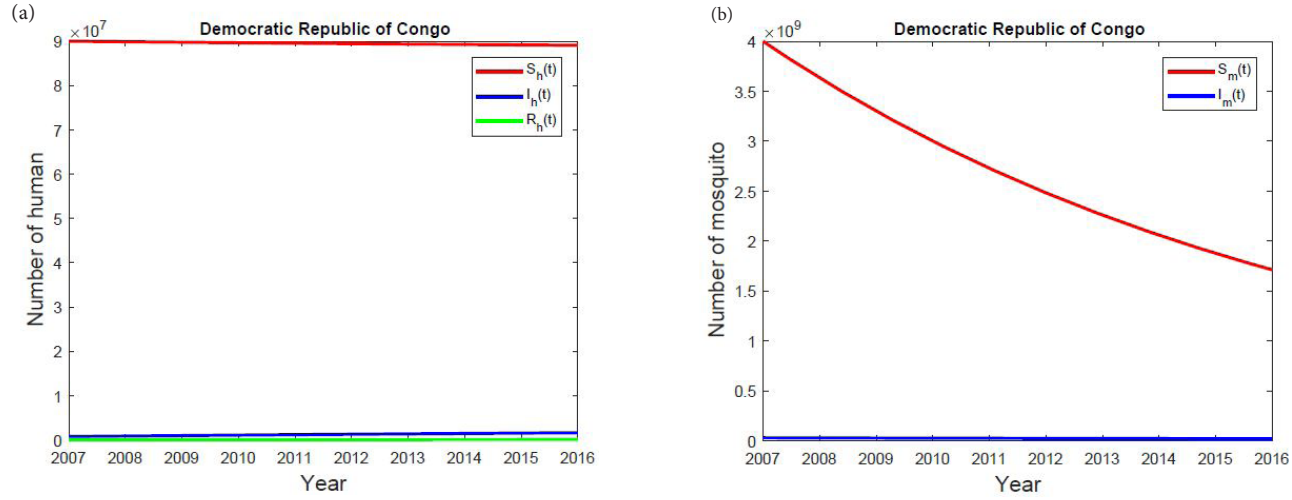

Figure 5: Solution of the model (2.1) with parameter for Democratic Republic of the Congo when $\mathcal{R}_0 < 1$, **(a)** Simulation the number of human from 2007 to 2016, **(b)** Simulation the number of mosquito from 2007 to 2016

Sensitivity Analysis

We used sensitivity analysis to determine the influence of the model parameter values used on the model. This analysis provides information on our model parameters that have important impact of theoretical model for malaria transmission in relationship to the basic reproduction number $\mathcal{R}_0$. In order to perform this analysis, we use the normalized forward sensitivity index of a variable to a parameter.

	Parameter	Sensitivity index
1	a	1
2	μ	-0.75
3	α	0.59
4	ψ	-0.5
5	γ_h	0.5
7	γ_m	0.5
8	q	0.5

Table 3: Sensitivity indices of $\mathcal{R}_0$ to parameters for model (3.1)

Definition 5.1. The normalized forward sensitivity index of a variable r that depends differentially on a parameters is defined as:

$$\gamma_r^s = \frac{\partial r}{\partial s} \times \frac{s}{r}, \tag{5.1}$$

In detail the sensitivity indices of $\mathcal{R}_0$ based on the computed are listed in Table 3. The parameters are ordered in such a way that it begins from the biggest sensitive to the smallest sensitive one. The most sensitive parameter from Table 3, are mosquitoes bitting rate, natural death rate of humans, loss of immunity rate for humans, recovery rate of humans, transmission rate in humans, transmission rate in mosquito, natural death rate of mosquito (a, μ, α, ψ, γ_h, γ_m), and the least parameter is q. At the endemic equilibrium for the model (2.1)

(a) If the value of *a* is decreased to 0.087 or less and the other values are maintained same then $\mathcal{R}_0 < 1$ (0.9916).
(b) If we increased the value of α from $3.5\times10^{-3} to\ 7.9\times10^{-2}$ then $\mathcal{R}_0 < 1$, i.e. $\mathcal{R}_0$ is decreased from 4.5589 to 0.9978.
(c) If the value of γ_h is decreased to 0.023 and the other parameters maintains the same then $\mathcal{R}_0 < 1$, (0.9979).
(d) If the value of γ_m is decreased to 0.023 and the other parameters maintains same then $\mathcal{R}_0 < 1$, (0.9979).

Optimal Control

Optimal control theory deals with the problem of finding control law for a given system such that a certain optimality criterion is achieved [29]. Optimal control is the process of determining and state trajectories for a dynamic system over a period of time to minimize a performance index [30].

In this section, we make use of Pontryagin's Principle in order to find the necessary conditions that establish the presence of optimal control of the malaria transmission model. We include time dependent controls into our model and attempt to explore the suitable optimal control strategy for setting the malaria under the control. Many optimal control strategies used to control and reduce malaria transmission with different cost depends on many factors such as: treated bed nets, treatment with drugs, indoor residual spray(IRS), Long-Lasting Insecticide Treated Net (LLITN), the insecticide spray on the breeding grounds the mosquitoes, etc. Also using of the Sterile Insect Technique (SIT), will reduce the mosquito population which helps to reduce malaria transmission in the specific area for more information see [31-33]. Because the malaria is spread widely in the DRC, specially in the country side which has many forest and heavy rainfall that increase the mosquitoes population. We used two control variables, $u_1(t)$ and $u_2(t)$ which represents the effort on preventing malaria infections through the use of bed-nets and treatment with drugs respectively to see the effects of them on malaria transmission on the Congo,DR. Our objective function is similar to and it's given by

$$J(u_1,u_2)=\int_0^{t_f}\left(AI_h+c_1u_1^2+c_2u_2^2\right)dt, \tag{6.1}$$

Where A is the balancing cost factor due to scale and c_1, c_2 denote the weighting constants for making use of bet-nets and treatment with drugs which has the potential of reducing the spread of the disease [9,28]. Consequently, we attempt to expect an optimal controls u_1^* and u_2^* such that,

$$J(u_1^*,u_2^*)=\min J(u_1,u_2),\Gamma=\{(u_1,u_2)|0\le u_i\le 1,i=1,2\}. \tag{6.2}$$

$$\begin{cases}\frac{dS_h}{dt}=\mu N_h-a\left(1-u_1\right)\frac{\gamma_h S_h I_m}{N_h}-\mu S_h+\rho R_h,\\ \frac{dI_h}{dt}=a\left(1-u_1\right)\frac{\gamma_h S_h I_m}{N_h}-\left(\mu+u_2\alpha\right)I_h+\frac{a\delta\gamma_h R_h I_m}{N_h},\\ \frac{dR_h}{dt}=u_2\alpha I_h-\frac{a\delta\gamma_h R_h I_m}{N_h}-(\mu+\rho)R_h,\\ \frac{dS_m}{dt}=\psi N_m-a\left(1-u_1\right)\frac{\gamma_m I_h S_m}{N_h}-\psi S_m,\\ \frac{dI_m}{dt}=a\left(1-u_1\right)\frac{\gamma_m I_h S_m}{N_h}-\psi I_m,\end{cases} \tag{6.3}$$

The optimal control must conform to the necessary conditions that is emanated from the Pontryagin Maximum Principle [34].This concept transforms the equations (6.2) and (6.3) into a type of problem characterised by minimizing pointwise a Hamililtonian H, with respect to u_1 and u_2:

$$\begin{aligned}H&=AI_h(t)+c_1u_1^2+c_2u_2^2\\&+\lambda_{S_h}\left\{\mu N_h-a\left(1-u_1\right)\frac{\gamma_h S_h I_m}{N_h}-\mu S_h+\rho R_h\right\}\\&+\lambda_{I_h}\left\{a\left(1-u_1\right)\frac{\gamma_h S_h I_m}{N_h}-\left(\mu+u_2\alpha\right)I_h+\frac{a\delta\gamma_h R_h I_m}{N_h}\right\}\\&+\lambda_{R_h}\left\{u_2\alpha I_h-\frac{a\delta\gamma_h R_h I_m}{N_h}-(\mu+\rho)R_h\right\}\\&+\lambda_{S_m}\left\{\psi N_m-a\left(1-u_1\right)\frac{\gamma_m I_h s_m}{N_h}-\psi S_m\right\}\\&+\lambda_{I_m}\left\{a\left(1-u_1\right)\frac{\gamma_m I_h s_m}{N_h}-\psi I_m\right\}\end{aligned} \tag{6.4}$$

Where $\lambda_{S_h}, \lambda_{I_h}, \lambda_{R_h}, \lambda_{S_m}, \lambda_{I_m}$ represents the adjoint variables. The system solution is attained by suitably taking partial derivatives of the Hamiltonian (6.4) with respect to the associated state variable.

Theorem 6.1. Given optimal controls u_1^*, u_2^* and solutions S_h, I_h, R_h, S_m, I_m of the corresponding state System (6.3) and (6.4) that minimize J (u_1, u_2) over Γ. Then there exists adjoint variables $\lambda_{S_h}, \lambda_{I_h}, \lambda_{R_h}, \lambda_{S_m}, \lambda_{I_m}$ satisfying

$$\frac{-d\lambda_i}{dt} = \frac{\partial H}{\partial i} \tag{6.5}$$

Where $i = S_h, I_h, R_h, S_m, I_m$ with transversality conditions

$$\lambda_{S_h}(t_f) = \lambda_{I_h}(t_f) = \lambda_{R_h}(t_f) = \lambda_{S_m}(t_f) = \lambda_{I_m}(t_f) = 0, \tag{6.6}$$

and

$$u_1^* = \min\left\{1, \max\left\{0, \frac{1}{2c_1}\left[a\gamma_h S_h I_m\left(\lambda_{I_h} - \lambda_{S_h}\right) + a\gamma_m I_h S_m\left(\lambda_{I_m} - \lambda_{S_m}\right)\right]\right\}\right\} \tag{6.7}$$

$$u_2^* = \min\left\{1, \max\left\{0, \frac{1}{2c_2}\left[\alpha I_h\left(\lambda_{I_h} - \lambda_{R_h}\right)\right]\right\}\right\} \tag{6.8}$$

Proof: Theorem 4.1 and Corollary 4.1 of [34] gives. the conditions of possible existence of an optimal control based on the convexity of the integrand of J (u_1, u_2) with respect to u_1 and u_2 a priori boundedness of the state solutions, and the resulting Lipschitz characteristics of the state system of the ODE's with the state variables [34]. The Hamiltonian function determines at the optimal control level leads to the adjoint variables. Thus, the adjoint equations can be reordered as

$$\begin{aligned}
\frac{d\lambda_{S_h}}{dt} &= \mu\lambda_{S_h} + a(1-u_1)\frac{\gamma_h I_m}{N_h}(\lambda_{S_h} - \lambda_{I_h}) + a(1-u_1)\frac{\gamma_h I_m S_h}{N_h^2}(\lambda_{I_h} - \lambda_{S_h}) + \\
&\quad \frac{a\delta\gamma_h R_h I_m}{N_h^2}\left(\lambda_{I_h} - \lambda_{R_h}\right) + a(1-u_1)\frac{\gamma_m I_h S_m}{N_h^2}(\lambda_{I_m} - \lambda_{S_m}), \\
\frac{d\lambda_{I_h}}{dt} &= -A + a(1-u_1)\frac{\gamma_h S_h I_m}{N_h^2}\left(\lambda_{I_h} - \lambda_{S_h}\right) + (\mu + u_2\alpha)\lambda_{I_h} + \frac{a\delta\gamma_h R_h I_m}{N_h^2}\left(\lambda_{I_h} - \lambda_{R_h}\right) \\
&\quad + a(1-u_1)\frac{\gamma_m I_h S_m}{N_h^2}(\lambda_{I_m} - \lambda_{S_m}) + a(1-u_1)\frac{\gamma_m S_m}{N_h}(\lambda_{S_m} - \lambda_{I_m}) - u_2\alpha\lambda_R, \\
\frac{d\lambda_{R_h}}{dt} &= a(1-u_1)\frac{\gamma_h S_h I_m}{N_h^2}\left(\lambda_{I_h} - \lambda_{S_h}\right) + \frac{a\delta\gamma_h I_m}{N_h}\left(\lambda_{R_h} - \lambda_{I_h}\right) + a(1-u_1)\frac{\gamma_m I_h S_m}{N_h^2}(\lambda_{I_m} - \lambda_{S_m}) \\
&\quad + \frac{a\delta_h R_h I_m}{N_h^2}\left(\lambda_{I_h} - \lambda_{R_h}\right) + (\mu + \rho)\lambda_{R_h} - \rho\lambda_{S_h}, \\
\frac{d\lambda_{S_m}}{dt} &= a(1-u_1)\frac{\gamma_m I_h}{N_h}(\lambda_{S_m} - \lambda_{I_m}) + \psi\lambda_{S_m}, \\
\frac{d\lambda_{Im}}{dt} &= a(1-u_1)\frac{\gamma_h S_h}{N_h}\left(\lambda_{S_h} - \lambda_{I_h}\right) + \frac{a\delta\gamma_h R_h}{N_h}\left(\lambda_{R_h} - \lambda_{I_h}\right) + \psi\lambda_{I_m},
\end{aligned} \tag{6.9}$$

Numerical Simulations for Optimal Control

In this subsection, we used MATLAB program to obtain the numerical simulation solutions. Table 3 presents the parameter values used for these simulations

Prevention of malaria through the use of treated bed-net (u_1) only: Malaria prevention (treated bed net) control (u_1) was used to optimized the objective function *J*, while we set the other control u_2 to zero. We observed in Figure 6(a) that these is a significant difference between the control ($u_1 \neq 0$, $u_2 = 0$) and without control ($u_1 = 0$, $u_2 = 0$). It was also observed in Figure 6(a) that the number of malaria infected humans is still increasing even after the activation of the control. This implies that this strategy is not effective in controling the number of malaria infected humans I_h. In Figure 6(b), there is a significant difference between the control ($u_1 \neq 0$, $u_2 = 0$) and without control ($u_1 = 0$, $u_2 = 0$). The use of the bed net reduces the number of infected mosquitoes I_m and would eventually reduce the spread of malaria. Hence the use of bed net as a strategy to control infected mosquitoes yields a positive result.

Prevention of malaria through treatment with drugs (u_2) only: In this strategy, treatment effort (u_2) was employed to optimize the objective function *J*, while the prevention through the use of bed net (u_1) was set to zero. It can be seen that there is no significant difference between the two graphs represented as (Figures 7a and 7b) respectively. It was also observed that the strategy used in reducing the number of malaria infected mosquitoes and that of humans respectively is not the best. This is due the to the fact that the number malaria humans I_h and malaria infected mosquitoes (I_m) are still increasing. Hence the use of treatment (u_2) control only as a strategy to reduced the number of malaria, malaria infected mosquitoes and malaria infected humans respectively is not effective.

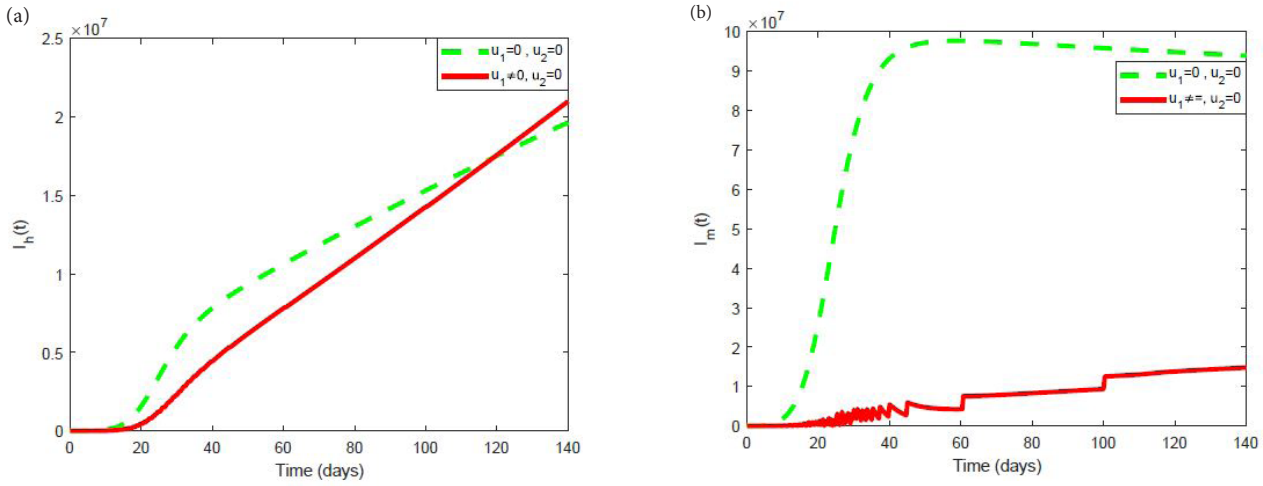

Figure 6: Simulations of the model showing the effect of malaria prevention only on transmission. Figure 6 **(a)** and **(b)** represents the behavior infected humans and infected mosquitoes respectively. Dashed line represents system without control ($u_1 = 0$, $u_2 = 0$) and solid line shows the system with control ($u_1 \neq 0$, $u_2 = 0$)

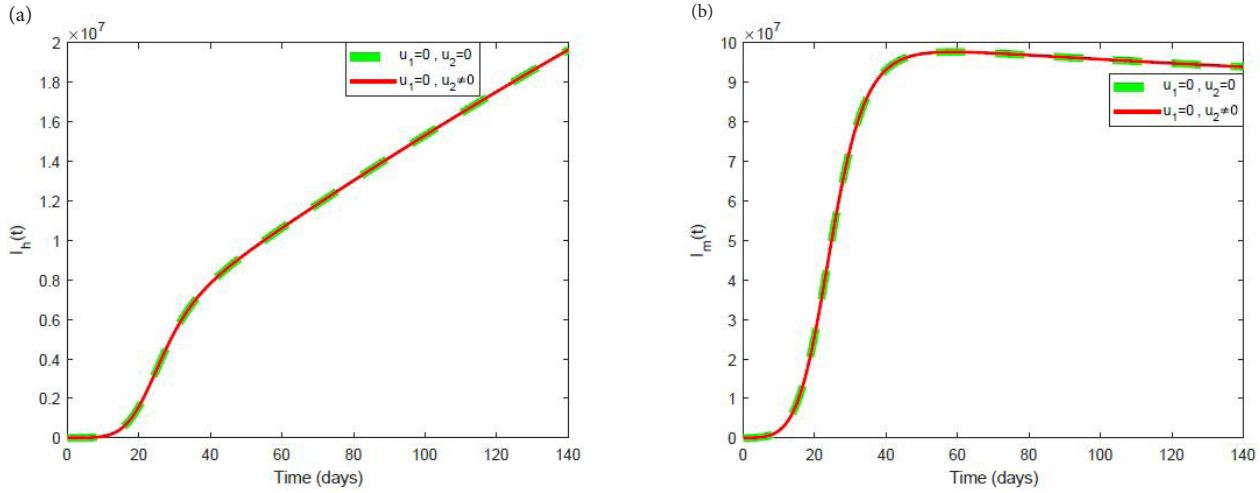

Figure 7: Simulations of the model showing the effect of malaria treatment only on transmission. Figures 7 **(a)** and **(b)** represents the behavior infected humans and infected mosquitoes respectively. Dashed line represents system without control ($u_1 = 0$, $u_2 = 0$) and solid line shows the system with control ($u_1 = 0$, $u_2 \neq 0$)

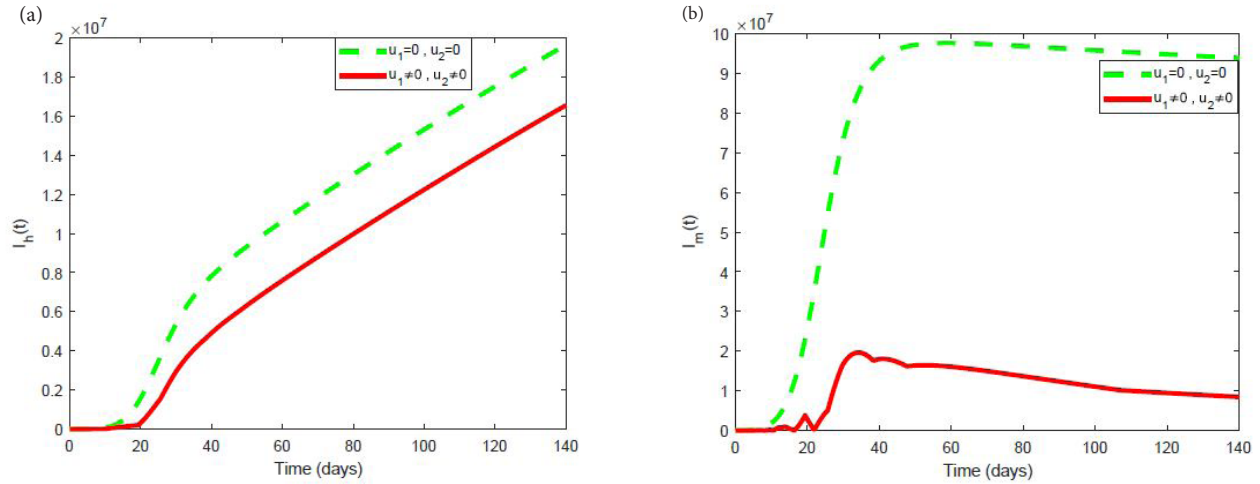

Figure 8: Simulations of the model showing the effect of malaria prevention only on transmission. Figures 6 **(a)** and **(b)** represents the behavior infected mosquitoes and infected human respectively. Dashed line represents system without control ($u_1 = 0$, $u_2 = 0$) and solid line shows the system with control ($u_1 \neq 0$, $u_2 \neq 0$)

Prevention of malaria through the use of treated bed net (u_1) and treatment with drugs (u_2) controls: In this strategy, all the two controls are explored in order to optimize the objective function. It is obvious in Figure 8a and 8b that there is a significant difference between the malaria infected mosquitoes and infected humans without control and that of malaria, malaria infected humans with control. It was observed in both that the activation of the two controls mechanisms are able to reduce the number of malaria infected

mosquitoes and that of malaria infected humans (Figures 8a and 8b). Though the impact of this strategy is greater in Figure 8(b) as compared to Figure 8(a). This means that strategy is able to reduce more infected mosquitoes than that of malaria infected humans.

Conclusion

In this paper, we studied an SIR-SI malaria transmission model. The population used for this model is made up of mosquitoes and humans. The recovered human population was divided into two groups, one group can be infected immediately by the disease to joined infectious class $I_h(t)$, and the other group recovered with immunity and re-enter the susceptible class $S_h(t)$. The fundamental property of the model is investigated, in addition to, the basic reproduction number $\mathcal{R}_0$. The equilibria of the model is studied, and the disease-free equilibrium is found to be locally and globally asymptotically stable when $\mathcal{R}_0 < 1$ respectively. We applied the center manifold theory to study the stability of endemic equilibrium and the results were that our model is asymptotically stable. The Pontryagin's Maximum Principle is used to determine the fundamental conditions necessary for elective control of malaria through the use of treated bed-net $(u_1(t))$ and treatment with drugs $(u_2(t))$ in the community. Our numerical simulation results suggest that using $u_1(t)$ and $u_2(t)$ together we can reduce the number of malaria infected individuals and malaria infected mosquitoes in the Democratic Republic of the Congo. In our future work we consider adding Indoor Residual Spray (IRS) and Long-Lasting Insecticide Treated Net (LLITN) malaria control strategies to our model. We would also try to incorporate the incubation stage of humans as well as that of mosquitoes in our future model.

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