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# Backward bifurcation analysis in SIRS-SI of the dynamics of malaria transmission model with treatment

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**Abstract**

In this paper, we developed a mathematical model which describes the dynamics of malaria transmission with treatment based on the SIRS-SI framework, using the system of ordinary differential equations (ODE). In addition, we derive a condition for the existence of equilibrium points of the model and investigate their stability and the existence of backward bifurcation for the model. Our result shows that if the reproduction number $R_0$ is less than 1 the disease-free equilibrium point is stable so that the disease dies out. If $R_0$ is greater than 1, then the disease-free equilibrium point is unstable. In this, the endemic state has a unique equilibrium and the disease persists within the human population. A qualitative study based on bifurcation theory reveals that backward bifurcation may occur. The stable disease-free equilibrium of the model coexists with the stable endemic equilibrium when the basic reproduction number is less than one. Numerical simulations were carried out using a mat lab to support our analytical solutions. And these simulations show how treatment affects the dynamics of the human and mosquito population.

Keywords: SIRS-SI, equilibrium points, backward bifurcation, reproduction number, Numerical simulations
2020 MSC: 93A30, 37G10, 37M05

## 1 Introduction

Malaria is an infectious disease caused by a parasite known as plasmodium. Carrier of plasmodium parasite is the female anopheles mosquito that causes the destruction of red blood cells in humans and animals through bites. Malaria can be transmitted through blood transfusion, sharing needles, or congenital. The infection can lead to serious complications affecting the brain, lungs, kidneys and other organs [17, 21]. Clinical symptoms such as fever, pain, chills and sweats may develop a few days after infected mosquito bites [22, 23]. Malaria is one of the most prevalent and lethal human infections throughout the world. An estimated 40 percent of the world's population lives in malaria endemic areas. Most cases and deaths occur in sub-Saharan Africa. It causes an estimated 300 to 500 million cases and 1.5 to 2.7 million deaths each year worldwide. Africa shares 80 percent of the cases and 90 percent of deaths [8, 20]. The epidemiological patterns of malaria usually vary with season in the case of transmission. The complexity of the disease control process, ways of transmission, cost of the control program and resistance of the parasite to anti-malarial drugs, and vectors to insecticides, are some of the challenges. As stated above there is a variation in

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disease patterns and transmission dynamics from place to place, by season and according to varying environmental circumstances [13, 18].

The approaches in the planning and implementation of prevention and control activities also vary based on local realities. Since malaria increases morbidity and mortality, it continues to inflict major public health and socioeconomic burdens in developing countries [3, 16, 26]. It is clear that poverty, while not a disease in itself, is a contributing factor not only for malaria but also for almost all diseases that face mankind. Because of poverty, communities may have poor sanitation and poor drainage, and these two factors allow the mosquitoes to breed in ever greater numbers. Many researchers have developed a mathematical model of malaria transmission[9, 26]. SIR with nonlinear infection rate and the use of vaccination against the human population was analyzed in [4, 13]. The effect of treatment as a control variable on malaria transmission system was studied in [2, 14]. Model of malaria transmission was investigated in [1, 12] by considering the existence of human-to-human transmission through blood transfusions and through malaria-infected pregnant women (congenital). In this current work, we discuss the effectiveness of the use of drugs in a malaria transmission model based on [17, 7, 19]. In our model we assumed that the vaccination at the right time can move susceptible human beings directly to recovered class and by considering the assumption that humans belong to recovered class have possibility to be susceptible, i.e., we consider a SIRS-SI model [1, 6]. Moreover, we also consider the treatment like application of vaccine and spraying as introduced in [15, 19, 24]. The global and local stability analysis of the model is then performed to reveal the effects of treatments on population dynamics [10, 11, 25, 27].

## 2 Model Formulation and Analysis

### 2.1 Model formulation

The endemic model of malaria transmission with treatment considered in our study is SIRS in human population and SI in mosquito population which divides human population into three classes namely susceptible human($S_h$), infected human ($I_h$) and recovered ($R_h$) and divides mosquito population into two class's namely susceptible mosquito ($S_m$) and infected mosquito($I_m$). The mosquito compartment does not include an immune class as mosquitoes never recover from the infection that is their infective period ends with their death due to their relatively short life cycle. Thus the immune class in the mosquito population is negligible and death occurs equally in all groups. The model is formulated for the spread of malaria in the human and mosquito population with the total population size at time (t) denoted $N_h$(t) and $N_m$(t)respectively. The basic model incorporates a set of assumptions.

By considering the above assumptions, the mathematical model which describe the dynamics of malaria in the human and mosquito populations using a system of nonlinear ordinary differential equation becomes

$$\frac{dS_h}{dt} = \lambda_h + \sigma R_h - (a\beta_1 I_h + b\beta_2 I_m)S_h - (\theta + \mu_h)S_h \tag{2.1}$$

$$\frac{dI_h}{dt} = \eta I_h + (a\beta_1 I_h + b\beta_2 I_m)S_h - (\mu_h + \alpha + k\gamma)I_h \tag{2.2}$$

$$\frac{dR_h}{dt} = k\gamma I_h - (\mu + \sigma)R_h + \theta S_h \tag{2.3}$$

$$\frac{dS_m}{dt} = \lambda_m - (c\beta_3 I_h + \mu_m + \rho)S_m \tag{2.4}$$

Table 1: **The state variable for the malaria model** (2.1) – (2.5)

| No | symbol | description | unit |
|---|---|---|---|
| 1 | $S_h$ | the number of susceptible humans | number |
| 2 | $I_h$ | the number of infected humans | number |
| 3 | $R_h$ | the number of recovered humans | number |
| 4 | $S_m$ | the number of susceptible mosquitoes | number |
| 5 | $I_m$ | the number of infected mosquitoes | number |
| 6 | $N_h$ | the total human populations | number |
| 7 | $N_m$ | the total mosquito populations | number |

Table 2: **The parameters for the malaria model** (2.1) – (2.5)

| No | symbol | description | unit |
|---|---|---|---|
| 1 | $\lambda_h$ | rate of individuals and new born migrate to susceptible class | $time^{-1}$ |
| 2 | $a$ | average number of blood transfusion per unit time | $time^{-1}$ |
| 3 | $\beta_1$ | chances of disease transmission to susceptible human from infected human | $time^{-1}$ |
| 4 | $b$ | average number of infected mosquito bites on susceptible human per unit t | $time^{-1}$ |
| 5 | $\beta_2$ | chances of disease transmission from infected mosquitoes to susceptible humans | $time^{-1}$ |
| 6 | $\theta$ | the number of susceptible rate of susceptible humans move to recovered class due to vaccination | $time^{-1}$ |
| 7 | $\mu_h$ | natural death rate of humans in each class | $time^{-1}$ |
| 8 | $\eta$ | rate of new born baby can be infected due to congenital | $time^{-1}$ |
| 9 | $k$ | the rate of human recovery | $time^{-1}$ |
| 10 | $\gamma$ | the effectiveness of anti-malarial drugs | $time^{-1}$ |
| 11 | $\alpha$ | death rate of humans due to malaria | $time^{-1}$ |
| 12 | $\mu_m$ | death rate of mosquitoes in each class | $time^{-1}$ |
| 13 | $\rho$ | death rate of mosquitoes in the two classes due to spraying | $time^{-1}$ |
| 14 | $\sigma$ | rate of recovered peoples move to susceptible class | $time^{-1}$ |
| 15 | $c$ | average number of susceptible mosquito bites on infected humans per unit t | $time^{-1}$ |
| 16 | $\beta_3$ | the chances of disease transmission from infected humans to susceptible mosquitoes | $time^{-1}$ |
| 17 | $\lambda_m$ | Per capita birth rate for mosquitoes | $time^{-1}$ |

$$\frac{dI_m}{dt} = \lambda_m - c\beta_3 I_h S_m - (\mu_m + \rho) I_m \tag{2.5}$$

where $\theta, k\gamma, \rho$ are treatment terms with initial conditions $S_h(0) = S_{ho}, I_h(0) = I_{h0}, R_h(0) = R_{h0}, S_m(0) = S_{m0}, I_m(0) = I_{m0}, N_h(0) = N_{h0}, N_m(0) = N_{m0}$. The total human population, $N_h$ and the total mosquito population, $N_m$ are given by:

$$\begin{aligned} N_h &= S_h + I_h + R_h \\ N_m &= S_m + I_m \end{aligned} \tag{2.6}$$

## 2.2 Analysis of the Model

### 2.2.1 Positivity of Solutions

**Theorem 2.1.** Let the initial condition be

$$\begin{aligned} \Omega H &= \{S_h(0) = S_{ho} \geq 0, I_h(0) = I_{h0} \geq 0, R_h(0) = R_{h0} \geq 0\} \\ \Omega M &= \{S_m(0) = S_{m0} \geq 0, I_m(0) = I_{m0} \geq 0\} \in D \end{aligned} \tag{2.7}$$

Then the solution $S_h(t), I_h(t), R_h(t), S_m(t), I_m(t)$ of the system (2.1)-(2.5)is positive for all, $t \geq 0$

**Proof**: From the differential equation (2.1) we have

$$\frac{dS_h}{dt} = \lambda_h + \sigma R_h - (a\beta_1 I_h + b\beta_2 + I_m) S_h - (\theta + \mu_h) S_h \geq -(a\beta_1 I_h + b\beta_2 + I_m) S_h - (\theta + \mu_h) S_h).$$

Without loss of generality the inequality can be expressed as

$$\frac{dS_h}{dt} \geq -(a\beta_1 I_h + b\beta_2 + I_m + \theta + \mu_h) S_h.$$

Integrating both sides gives

$$\int \frac{dS_h}{S_h} \geq -\int (a\beta_1 I_h + b\beta_2 + I_m + \theta + \mu_h) dt$$

$$S_h(t) \geq e^{-(a\beta_1 I_h + b\beta_2 + I_m + \theta + \mu_h)t + c} = A e^{-(a\beta_1 I_h + b\beta_2 + I_m + \theta + \mu_h)t},$$

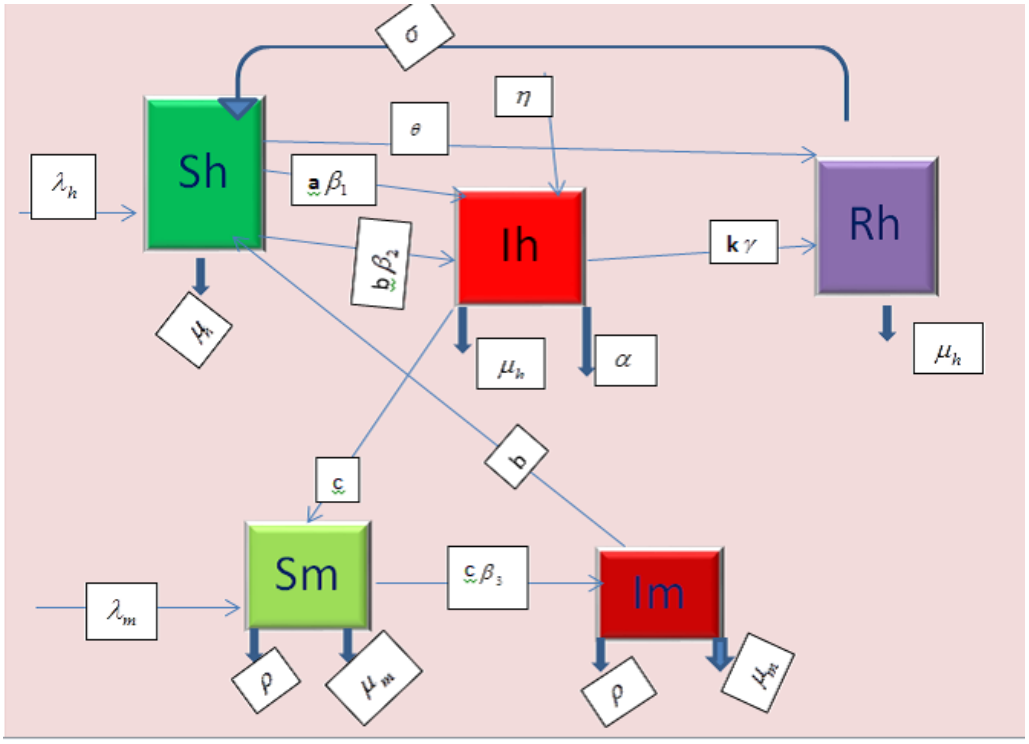


Figure 1: The compartmental model for malaria transmission flow diagram

where $A = e^c$, at $t = 0$, $S_h(0) = A \geq 0$. Thus,

$$S_h(t) \geq e^{-(a\beta_1 I_h + b\beta_2 + I_m + \theta + \mu_h)t + c} \geq 0.$$

Without loss of generality the equations $(2.1) - (2.5)$ can be proved in the same way and all are positive as $t \longrightarrow \infty$.

### 2.2.2 Basic Reproduction Number

Intuitively from the epidemiological point of view, the basic reproduction number $(R_0)$ is the average number of new cases (infections), that one infected case will generate during its entire infectious life time. It is very important in determining whether the disease persists in the population or die out. We use the next generation method to compute $(R_0)$. Let us assume that there are n compartments of which the first m compartments correspond to infected individuals. Let

- $F_i(x)$ be the rate of appearance of new infections in compartments i,
- $V_i^+$ is the rate of transfer of individuals into compartment i by all other means,
- $V_i^-$ is the rate of transfer of individual out of the $i^{th}$ and compartment
- $F_i(x)$ be the rate of appearance of new infections in compartments.

It is assumed that each function is continuously differentiable at least twice in each variable. The disease transmission model consists of nonnegative initial conditions together with the following system of equations:

$$\frac{dx_i}{dt} = f_i = F_i(x)V_i(x), i = 1, 2, 3, ...$$

where $V_i(x) = V_i^+ - V_i^-$ The next step is the computation of the square matrices $F$ and $V$ of order $m \times m$, where $m$ is the number of infected classes, defined by

$$F = \left[\frac{\partial F_i}{\partial X_j}(x_0)\right]$$

and

$$V = \left[\frac{\partial V_i}{\partial X_j}(x_0)\right]$$

with $1 \leq 1, j \leq m$ such that $F$ is nonnegative, $V$ is a non-singular matrix and $E_0$ is the disease-free equilibrium point. Consequently

$$F = \begin{bmatrix} f_{11} \\ f_{22} \end{bmatrix} = \begin{bmatrix} (a\beta_1 I_h + b\beta_2 I_m)S_h \\ c\beta_3 I_h S_m \end{bmatrix}$$

By linearization approach the associated matrix $F$ at disease free equilibrium point $E_o$ is given by

$$F = \begin{bmatrix} \frac{\partial f_{11}}{\partial I_h} & \frac{\partial f_{11}}{\partial I_m} \\ \frac{\partial f_{22}}{\partial I_h} & \frac{\partial f_{22}}{\partial I_m} \end{bmatrix} = \begin{bmatrix} a\beta_1 S_h & b\beta_2 S_h \\ c\beta_3 S_m & 0 \end{bmatrix}$$

Similarly,

$$V = \begin{bmatrix} v_{11} \\ v_{22} \end{bmatrix} = \begin{bmatrix} \frac{\partial v_{11}}{\partial I_h} & \frac{\partial v_{11}}{\partial I_m} \\ \frac{\partial v_{22}}{\partial I_h} & \frac{\partial v_{22}}{\partial I_m} \end{bmatrix} = \begin{bmatrix} \mu_h + \sigma + k\gamma - \eta & 0 \\ 0 & -(\mu_m + \rho) \end{bmatrix}$$

Thus,

$$V^{-1} = \begin{bmatrix} \frac{-1}{\mu_h+\sigma+k\gamma-\eta} & 0 \\ 0 & \frac{-1}{\mu_m+\rho} \end{bmatrix}$$

Since $F$ is non-negative and $V$ is non-singular, and then $V^{-1}$is non-negative and also $FV^{-1}$ is nonnegative. Hence the matrix $FV^{-1}$ is called the next generation matrix for the model. Finally, the basic reproduction number $R_0$ (reproduction ratio) is given by $R_0 = \rho(FV^{-1})$ where $A = FV^{-1}$. Then $\rho(A)$ denotes the spectral radius of a matrix $A$ and the spectral radius, $\rho(FV^{-1})$ is the biggest non negative eigenvalue of the next generation matrix. Equilibrium point of the system of differential equations $(2.1) - (2.5)$ can be obtained by simultaneously solving the following equations.

$$\frac{S-h}{dt} = 0, \frac{I-h}{dt} = 0, \frac{R-h}{dt} = 0, \frac{s-m}{dt} = 0, \frac{I-m}{dt} = 0$$

The system of differential equations $(2.1) - (2.5)$ disease states $F$ and the transfer states $V$ with respect to $I_h$ and $I_m$ at the disease free equilibrium.

$$E_0 = (S_h, I_h, R_h, S_m, I_m) = (S_h^*, 0, R_h^*, S_m^*, 0)$$

where

$$S_h^* = \frac{\lambda_h(\mu_h + \sigma)}{\mu_h(\theta + \sigma + \mu_h)},\ R_h^* = \frac{\lambda_h \theta}{\mu_h(\theta + \sigma + \mu_h)}, S_m^* = \frac{\lambda_m}{\mu_m + \rho}$$

Hence,

$$F = \begin{bmatrix} \frac{a\beta_1\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h} & \frac{b\beta_2\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h} \\ \frac{c\beta_3\lambda_m}{\mu_m+\rho} & 0 \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{-1}{\mu_h+\sigma+k\gamma-\eta} & 0 \\ 0 & \frac{-1}{\mu_m+\rho} \end{bmatrix}$$

Thus,

$$FV^{-1} = \begin{bmatrix} \frac{a\beta_1\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h} & \frac{b\beta_2\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h} \\ \frac{c\beta_3\lambda_m}{\mu_m+\rho} & 0 \end{bmatrix} \begin{bmatrix} \frac{-1}{\mu_h+\sigma+k\gamma-\eta} & 0 \\ 0 & \frac{-1}{\mu_m+\rho} \end{bmatrix}$$

Basic reproduction number $R_0$ is the largest positive eigenvalues of the matrix $K = FV^{-1}$ [5]from which we obtain

$$R_0 = \frac{b_1 + \sqrt{b_1^2 + 4b_2 b_3}}{2}$$

where

$$b_1 = \frac{a\beta_1\lambda_h(\mu_h + \sigma)}{\mu_h(\theta + \sigma\mu_h)(-\eta + \mu_h + \alpha + k\gamma)}$$

$$b_2 = \frac{b\beta_2\lambda_h(\mu_h + \sigma)}{(\mu_m + \rho)(\theta + \alpha + \mu_h)}$$

$$b_3 = \frac{c\beta_3\lambda_m}{(\mu_m + \rho)(-\eta + \mu_h + \alpha + k\gamma)}$$

### 2.2.3 Local Stability of Disease Free Equilibrium Point

The equilibrium is obtained by equating the right hand side of the system $(2.1)-(2.5)$ to zero.The disease-free equilibrium $(DFE)$ of the model is the steady-state solution of the model in the absence of the disease $(malaria)$.Hence, the DFE of the malaria model $(2.1)-(2.5)$ is given by

$$E=(S_h^*,I_h^*,R_h^*,S_m^*,I_m^*)=\left(\frac{\lambda_h\left(\mu_h+\sigma\right)}{\mu_h\left(\theta+\sigma+\mu_h\right)},0,\frac{\lambda_h\theta}{\mu_h\left(\theta+\sigma+\mu_h\right)},\frac{\lambda_m}{\mu_m+\rho},0\right)$$

**Theorem 2.2.** The disease free state $E_0$, locally asymptotically stable if $R_0\leq 1$ and unstable if $R_0\geq 1$

**Proof**: The Jacobin matrix of the system $(2.1)-(2.5)$ evaluated at the disease free equilibrium point $E_0$, is given by

$$J\left(E_0\right)=\begin{bmatrix}-\left(\theta+\mu_h\right) & 0 & \sigma & 0 & 0\\ 0 & \eta-\left(\mu_h+\alpha+k\gamma\right) & 0 & 0 & b\beta_2\frac{\lambda_h(\mu+\sigma)}{\mu_h(\theta+\sigma+\mu_h)}\\ 0 & k\gamma & -\left(\mu_h+\sigma\right) & 0 & 0\\ 0 & -c\beta_3\frac{\lambda_m}{\mu_m+\rho} & 0 & -\left(\mu_h+\rho\right) & 0\\ 0 & c\beta_3\frac{\lambda_m}{\mu_m+\rho} & 0 & 0 & -\left(\mu_m+\rho\right)\end{bmatrix}$$

We need to show that all the eigenvalues of $J\left(E_0\right)$ are negative. As the first and the fourth columns contain only the diagonal terms which form the two negative eigenvalues $-\left(\theta+\mu_h\right)$ and $-\left(\mu_m+\rho\right)$, the other three eigenvalues can be obtained from the sub matrix, $J_1\left(E_0\right)$, formed by excluding the first and fourth rows of $J\left(E_0\right)$ and the third column of $J\left(E_0\right)$. Hence we have

$$J_1\left(E_0\right)=\begin{bmatrix}\eta-\left(\mu_h+\alpha+k\gamma\right) & 0 & b\beta_2\frac{\lambda_h(\mu+\sigma)}{\mu_h(\theta+\sigma+\mu_h)}\\ k\gamma & -\left(\mu_h+\sigma\right) & 0\\ c\beta_3\frac{\lambda_m}{\mu_m+\rho} & 0 & -\left(\mu_m+\rho\right)\end{bmatrix}$$

In the same way, the second column of $J_1\left(E_0\right)$, contains only the diagonal term which forms a negative eigenvalue, $-\left(\mu_h+\sigma\right)$. The remaining two eigenvalues are obtained from the sub-matrix;

$$J_2\left(E_0\right)=\begin{bmatrix}\eta-\left(\mu_h+\alpha+k\gamma\right) & b\beta_2\frac{\lambda_h(\mu+\sigma)}{\mu_h(\theta+\sigma+\mu_h)}\\ c\beta_3\frac{\lambda_m}{\mu_m+\rho} & -\left(\mu_m+\rho\right)\end{bmatrix}$$

The eigenvalues of matrix $J_2\left(E_0\right)$ are the roots of characteristic equation of the matrix:

$$\left|J_2\left(E_0\right)-\lambda\right|=\begin{vmatrix}\left(\eta-\left(\mu_h+\alpha+k\gamma\right)\right)-\lambda & b\beta_2\frac{\lambda_h(\mu+\sigma)}{\mu_h(\theta+\sigma+\mu_h)}\\ c\beta_3\frac{\lambda_m}{\mu_m+\rho} & -\left(\mu_m+\rho\right)-\lambda\end{vmatrix}=0$$

Then the characteristic equation is:

$$\left(\left(\eta-\left(\mu_h+\alpha+k\gamma\right)\right)-\lambda\right)\left(-\left(\mu_m+\rho\right)-\lambda\right)-\left(b\beta_2\frac{\lambda_h\left(\mu+\sigma\right)}{\mu_h\left(\theta+\sigma+\mu_h\right)}\right)\left(c\beta_3\frac{\lambda_m}{\mu_m+\rho}\right)=0$$

$$\lambda^2+\left(-\mu_m-\rho\right)\left(\eta-\mu_h-\alpha-k\gamma\right)-\lambda\left(-\mu_m-\rho\right)-\lambda\left(\eta-\mu_h-\alpha-k\gamma\right)-\left(c\beta_3\frac{\lambda_m}{\mu_m+\rho}\right)\left(b\beta_2\frac{\lambda_h\left(\mu+\sigma\right)}{\mu_h\left(\theta+\sigma+\mu_h\right)}\right)=0$$

$$\lambda^2+a_1\lambda+a_2=0$$

where

$$a_1=\mu_m+\rho-\eta+\mu_h+\alpha+k\gamma,$$

$$a_2=-\eta\mu_m+\mu_h\mu_m+\alpha\mu_m+k\gamma\mu_m-\rho\eta+\rho\mu_h+\alpha\rho+k\rho\gamma-c\beta_3\frac{\lambda_m}{\mu_m+\rho}b\beta_2\frac{\lambda_h\left(\mu+\sigma\right)}{\mu_h\left(\theta+\sigma+\mu_h\right)}$$

The remaining eigenvalues $\lambda_4$ and $\lambda_5$ are found by solving the characteristic equation above and hence we can see that $\lambda_4=\frac{-a_1-\sqrt{a_1^2-4a_2}}{2}$ and $\lambda_5=\frac{-a_1+\sqrt{a_1^2-4a_2}}{2}$

### 2.2.4 Global Stability of the Disease Free Equilibrium Point

**Theorem 2.3.** The disease free equilibrium point $E_0$ of the system $(2.1)-(2.5)$ is globally stable in region D if $R_0 < 1$ where $D = D_h \times D_m; D_h = \left\{(S_h, I_h, R_h) \in \mathbb{R}^3, S_h \geq 0, I_H \geq 0, R_h \geq 0, N_h \leq \frac{\lambda_h}{\mu_h}\right\}, D_m = \left\{(S_m, I_m) \in \mathbb{R}^2, S_m \geq 0, I_H \geq 0, N_m \leq$

**Proof**:
The equation of the infected components can be written in terms of

$$\begin{vmatrix} \frac{dS_h}{dt} \\ \frac{dI_h}{dt} \end{vmatrix} = |F - V| \begin{vmatrix} I_h \\ I_m \end{vmatrix} = \begin{vmatrix} a\beta_1 I_h + b\beta_2 I_m - \left(\frac{a\beta_1 I_h}{N_h} + \frac{b\beta_2 I_m}{N_m}\right) S_h \\ c\beta_3 I_h - \left(\frac{c\beta_3 I_h}{N_h}\right) S_m \end{vmatrix}$$

Then

$$\begin{vmatrix} \frac{dS_h}{dt} \\ \frac{dI_h}{dt} \end{vmatrix} \leq |F - V| \begin{vmatrix} I_h \\ I_m \end{vmatrix} \tag{2.8}$$

All the eigenvalues of the matrix $[F - V]$ have negative real parts. It follows that the system of linear differential inequality (2.8) is stable whenever $R_0 < 1$. Consequently by comparison theorem we have $I_h \longrightarrow 0$ and $I_m \longrightarrow 0$ as $t \longrightarrow \infty$.This follows that $(I_h, I_m) \longrightarrow (0, 0)$ and the remaining equation of system $(2.1) - (2.5)$ give us the solution $E_0 = (S_h, 0, R_h, S_m, 0)$.Therefore $(S_h, I_h, R_h, S_m, I_m) \longrightarrow E_0$ as $t \longrightarrow \infty$. Hence, the disease free equilibrium point is globally asymptotically stable whenever $R_0 < 1$.

### 2.2.5 Local Stability of Endemic Equilibrium Point

The equilibrium points are obtained by equating the right hand side of the system $(2.1) - (2.5)$ to zero. Endemic equilibrium of the model is the steady-state solution of the model in the presence of the disease (malaria).

**Theorem 2.4.** The positive endemic equilibrium point $E_0$ of the system is locally asymptotically stable if $R_0 > 1$.

**Proof**: The Jacobin matrix of the system evaluated at endemic equilibrium point $E_0$, is obtained as

$$J(E_0) = \begin{bmatrix} -(k_1 I_h^* + k_2 I_m^* + \theta + \mu_h) & -k_1 S_h^* & \sigma & 0 & k_2 S_h^* \\ k_1 I_h^* + k_2 I_h^* & -(\tau - k_1 S_h^*) & 0 & 0 & k_2 S_h^* \\ \theta & k\gamma & -(\mu_h + \sigma) & 0 & 0 \\ 0 & -k_3 S_m^* & 0 & -(k_3 I_h^* + \mu_m + \rho) & 0 \\ 0 & k_3 S_m^* & 0 & k_3 I_h & -(\mu_m + \rho) \end{bmatrix}$$

Now we show that, if $R_0 > 1$, then $Tr[J(E^*)] < 0$ and $Det[J(E^*)] > 0$

$$J(E_0) = \begin{bmatrix} -(p + \theta + \mu_h) & -z & \sigma & 0 & -m \\ p & -(\tau - z) & 0 & 0 & m \\ \theta & k\gamma & -(\mu_h + \sigma) & 0 & 0 \\ 0 & -q & 0 & -(r + \mu_m + \rho) & 0 \\ 0 & q & 0 & r & -(\mu_m + \rho) \end{bmatrix}$$

where $p = k_1 I_h^* + k_2 I_m^*, q = k_3 S - m^*, z = k_1 S_h^8, m = k_2 S_h^*, r = k_3 S_h^*$ here the condition $Tr[J(E^*)] < 0$ is satisfied. The next thing is to compute determinant of the system and to show $Det[J(E^*)] > 0$ $Det[J(E^*)] > 0$

$$Det[J(E^*)] = \begin{bmatrix} -x & -z & \sigma & 0 & -m \\ p & -y & 0 & 0 & m \\ \theta & k\gamma & -c & 0 & 0 \\ 0 & -q & 0 & -w & 0 \\ 0 & q & 0 & r & -v \end{bmatrix}$$

where $x = p + \theta + \mu_h, y = \tau - z, c = \mu_h + \sigma, w = r + \mu_m + \rho, v = \mu_m + \rho$
Thus

$$Det[J(E^*)] = xcmq(w - r) + pcmq(w - r) + \theta\sigma mq(w - r) + pmv(d\sigma + cv) + \theta\sigma(ywv - xycwv)$$

From

$$xcmq(w - r) > 0, xcmq(r + \mu_m + \rho - r) > 0, xcmq(\mu_m + \rho) > 0$$

and

$$-[xycwv] = -[(p+\theta+\mu_h)(\tau-z)(\mu+\sigma)(r+\mu_m+\sigma)(\mu_m+p)] =$$
$$-[\tau(p+\theta+\mu_h)(-z)(r+\mu_m+\sigma)(\mu_m+\rho)] \geq 0$$

From the above equation $Det[J(E^*)] > 0$. There fore $R_0 > 1$, because $Det[J(E^*)] > 0$ and $Tr[J(E^*)] < 0$. This implies that the equilibrium point $E^*$ of the system is locally asymptotically stable.

### 2.2.6 Bifurcation Analysis of the model

**Existence of backward bifurcation**

We shall use the following theorem in [9], to show that the system $(2.1)-(2.5)$ exhibits backward bifurcation at $R_0 = 1$

**Theorem 2.5.** Consider the following general system of ordinary differential equations with a parameter $\phi$

$$\frac{dx}{dt} = f(x,\phi), f : \phi \in \mathbb{R}^n, x \in \mathbb{R}^n \tag{2.9}$$

Without loss of generality, it is assumed that 0 is equilibrium for system (2.9) for all values of the parameter $\phi$ , (that is $f(0,\phi) = 0$). Assume

$(A_1)$ $A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_i}(0,0)\right)$ is the linearized matrix of system above around the equilibrium 0 with $\phi$ evaluated at 0.

$(A_2)$ Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts;

$(A_3)$ Matrix A has a non-negative right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.

Let $f_k$ be the $k^{th}$component of f and

$$a = \sum_{i,j,k=1}^{5} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$$
$$b = \sum_{i,j,k=1}^{5} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0) \tag{2.10}$$

The local dynamics of system (2.10) around 0 are totally determined by a and b.

1. $a > 0, b > 0$. when $\phi < 0$ with $|\phi| << 1$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \phi << 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium.
2. $a < 0, b < 0$. When $\phi < 0$ with $|\phi| << 1$, 0 is unstable; when $0 < \phi << 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium,
3. $a > 0, b < 0$. When $\phi < 0$ with $|\phi| << 1$, 0 is unstable;and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi << 1$, 0 is stable, and a positive unstable equilibrium appears,
4. $a < 0, b > 0$. When $\phi$ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

Particularly, if $a > 0$ and $b > 0$, then a backward bifurcation occurs at $\phi = 0$. To apply the above result, the following simplification and change of variables are made on the system $(2.1) - (2.5)$

Let $S_h = x_1, I_h = x_2, R_h = x_3, S_m = x_4, I_m = x_5$ , $N_h = x_1 + x_2 + x_3$ and $N_m = x_4 + x_5$ Moreover, by using vector notation $x = (x_1, x_2, x_3, x_4, x_5)^T$, the system can be written in the form $\frac{df}{dt} = (f_1, f_2, f_3, f_4, f_5)^T$ as follows

$$\frac{dx_1}{dt} = f_1 = \lambda_h + \sigma x_3 - (a\beta_1 I_h + b\beta_2 I_m) x_1 - (\theta + \mu_h) x_1$$
$$\frac{x_2}{dt} = f_2 = \eta x_2 + (a\beta_1 x_2 + b\beta_2 x_5) x_1 - (\alpha + \mu_h + k\gamma) x_2$$
$$\frac{x_3}{dt} = f_3 = k\gamma x_2 - (\mu + \sigma) x_3 + \theta x_1$$
$$\frac{x_4}{dt} = f_4 = \lambda_m - (c\beta_3 x_2 + \mu_m + \rho) x_4$$
$$\frac{x_5}{dt} = f_5 = c\beta_3 x_2 x_4 - (\mu_m + \rho) x_5$$

Choose $a\beta_2 = (a\beta_2)^*$ as bifurcation parameter, then at $R_0 = 1$ we obtain

$$a\beta_2 = (a\beta_2)^* := \frac{b_1\sqrt{b_1^2 + 4b_2b_3}}{2} = R_0.$$

So the disease free equilibrium $E_0$ is locally stable when $a\beta_2 < (a\beta_2)^*$ and is unstable when $a\beta_2 > (a\beta_2)^*$. Then $(a\beta_2)^*$ is a bifurcation value. The linearized matrix of the system above around the disease free equilibrium $E_0$ and evaluated at $(a\beta_2)^*$ is given by $J\left(E_0, (a\beta_2)^*\right)$ and

$$J\left(E_0, (a\beta_2)^*\right) = \begin{bmatrix} -(\theta+\mu_h) & 0 & \sigma & 0 & 0 \\ 0 & \eta-(\mu_h+\alpha+k\gamma) & 0 & 0 & \beta_2 S_h \\ 0 & k\gamma & -(\mu_h+\sigma) & 0 & 0 \\ 0 & -c\beta_3 & 0 & -(\mu_h+\rho) & 0 \\ 0 & c\beta_3 & 0 & 0 & -(\mu_m+\rho) \end{bmatrix}$$

where $S_h = \frac{\lambda_h(\mu+\sigma)}{\mu_h(\theta+\sigma+\mu_h)}$ and $S_m = \frac{\lambda_m}{\mu_m+\rho}$. The eigenvalues $\lambda$ of $J\left(E_0, (a\beta_2)^*\right)$ given by the equation above are roots of characteristics of the form

$$\lambda^2 + a_1\lambda + a_2 = 0.$$

From this

$$\lambda_1 = \frac{-a_1 - \sqrt{a_1^2 - 4a_2}}{2}, \lambda_2 = \frac{-a_1 + \sqrt{a_1^2 - 4a_2}}{2}.$$

If $a_1 = \sqrt{a_1^2 4a_2}\lambda = 0$, then now in this case assume that $a_1 = \sqrt{a_1^2 4a_2}$, that is $\lambda = 0$. The right eigenvector $w = (w_1, w_2, w_3, w_4, w_5)^T$ associated with this simple zero eigenvalue can be obtained from $J\left(E_0, (a\beta_2)^*\right).w = 0$. As a result we have

$$w_1 = \frac{\sigma}{\theta+\mu_h}w_3, w_2 = \frac{\sigma+\mu_h}{k\gamma}w_3, w_3 = \frac{b\beta_2 S_h}{\mu_h+\alpha+k\gamma-\eta}, w_4 = \frac{(\mu+\rho)}{c\beta_3 S_m}w_4.$$

Further, the left eigenvector $v = (v_1, v_2, v_3, v_4, v_5)^T$ corresponding to the simple zero eigenvalue is obtained from $v.J\left(E_0, (a\beta_2)^*\right)$ as

$$v_1 = 0, v_3 = 0, v_4 = 0, v_2 = v_2 > 0, v_5 = \frac{b\beta_2 S_h}{\mu_h+\rho}v_2$$

**Computation of $a$**

By computing the second-order partial derivatives at the disease free equilibrium point we have

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_j} = 0, j = 1, 2, 4$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_j} = 0, j = 2, 3, 4, 5$$

$$\frac{\partial^2 f_2}{\partial x_3 \partial x_j} = 0, j = 1, 2, 3, 4, 5$$

$$\frac{\partial^2 f_2}{\partial x_4 \partial x_j} = 0, j = 1, 2, 3, 4, 5$$

$$\frac{\partial^2 f_2}{\partial x_5 \partial x_j} = 0, j = 2, 3, 4, 5$$

where as

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \frac{\partial^2 f_2}{\partial x_2 \partial x_1} = a\beta_2$$

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_5} = \frac{\partial^2 f_2}{\partial x_5 \partial x_1} = a\beta_2.$$

Similarly,

$$\frac{\partial^2 f_5}{\partial x_1 \partial x_j} = 0, j = 1, 2, 3, 4, 5$$

$$\frac{\partial^2 f_5}{\partial x_2 \partial x_j} = 0, j = 1, 2, 3, 5$$

$$\frac{\partial^2 f_5}{\partial x_3 \partial x_j} = 0, j = 1, 2, 3, 4, 5$$

$$\frac{\partial^2 f_5}{\partial x_4 \partial x_j} = 0, j = 1, 3, 4, 5$$

$$\frac{\partial^2 f_5}{\partial x_5 \partial x_j} = 0, j = 1, 2, 3, 4, 5$$

where

$$\frac{\partial^2 f_5}{\partial x_2 \partial x_4} = \frac{\partial^2 f_5}{\partial x_4 \partial x_2} = c\beta_3, j = 1, 3, 4, 5.$$

Then

$$\begin{aligned}
a &= v_2 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}(0,0) + v_5 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j}(0,0) \\
&= v_2 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}(0,0) + \frac{b\beta_2 S_h}{\mu_m + \rho} v_2 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j}(0,0) \\
&= v_2 \left[ \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}(0,0) + \frac{b\beta_2 S_h}{\mu_m + \rho} \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j}(0,0) \right] \\
&= v_2 \left[ w_1 w_2 (a\beta_1) + w_5 w_1 (a\beta_2) + \frac{b\beta_2 S_h}{\mu_m + \rho} v_2 w_2 w_4 c\beta_3 \right] \\
&= v_2 \left[ a\beta_1 \left( \frac{\sigma}{\theta + \mu_h} \right) w_3 \left( \frac{\sigma}{k\gamma} \right) w_3 + \frac{\mu_h + \alpha + k\gamma - \eta}{b\beta_2 S_h} w_2 \left( \frac{\sigma}{\theta + \mu_h} \right) w_3 + \right. \\
&\qquad \left. \frac{b\beta_2 S_h}{\mu_m + \rho} - \left( \frac{c\beta_3 S_m}{\mu_m + \rho} \right) w_2 \right] \\
&= v_2 \left[ a\beta_1 \left( \frac{\sigma}{\theta + \mu_h} \right) \left( \frac{\mu_h + \sigma}{k\gamma} \right) w_3^2 + \left( \frac{\mu_h + \alpha + k\gamma - \eta}{b\beta_2 S_h} \right) \left( \frac{\mu_h + \sigma}{k\gamma} \right) \left( \frac{\sigma}{\theta + \mu_h} \right) w_3^2 - \right. \\
&\qquad \left. \left( \frac{b\beta_2 S_h}{\mu_m + \rho} \right) \left( \frac{c\beta_3 S_m}{\mu_m + \rho} \right) w_3^2 \right] \\
&= \left( \frac{\mu_h + \sigma}{k\gamma} \right) \left[ a\beta_1 \left( \frac{\sigma}{\theta + \mu_h} \right) + \left( \frac{\mu_h + \alpha + k\gamma - \eta}{b\beta_2 S_h} \right) \left( \frac{\sigma}{\theta + \mu_h} \right) - \right. \\
&\qquad \left. \frac{b\beta_2 S_h}{(\mu_m + \rho)^2} (c\beta_3 S_m) \left( \frac{\mu_h + \sigma}{k\gamma} \right) \right] v_2 w_3^2
\end{aligned}$$

**Computation of** $b$

To compute $b$ we need to find the second order derivatives of $f_2$ and $f_5$ with respect to $x_i$ and $b\beta_2$ at the disease free equilibrium point. Direct computation shows

$$\frac{\partial^2 f_2}{\partial x_i \partial b\beta_2} = 0, j = 1, 2, 3, 4$$

$$\frac{\partial^2 f_5}{\partial x_i \partial b\beta_2} = 0, j = 1, 2, 3, 4, 5$$

and

$$\frac{\partial^2 f_2}{\partial x_i \partial b\beta_2} = b\beta_2$$

$$b = \sum_{i,k=1}^{5} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial b\beta_2}(0,0) = b\beta_2 v_k w_i > 0$$

Hence the following result holds. The malaria model $(2.1)-(2.5)$ exhibits backward bifurcation at $R_0 = 1$ whenever $a$ is positive. But $a > 0$ if and only if

$$b\beta_2 S_h \frac{(c\beta_3 S_m)}{(\mu_m+\rho)^2}\left(\frac{\mu_h+\sigma}{k\gamma}\right)^2 < \left[\left(\frac{\mu_h+\sigma}{k\gamma}\right)\left(\frac{\sigma}{\theta+\mu_h}\right)\left(a\beta_1 + \frac{\mu_h+\alpha+k\gamma-\eta}{b\beta_2 S_h}\right)\right]$$

Therefore backward bifurcation exhibits when this condition holds and given us

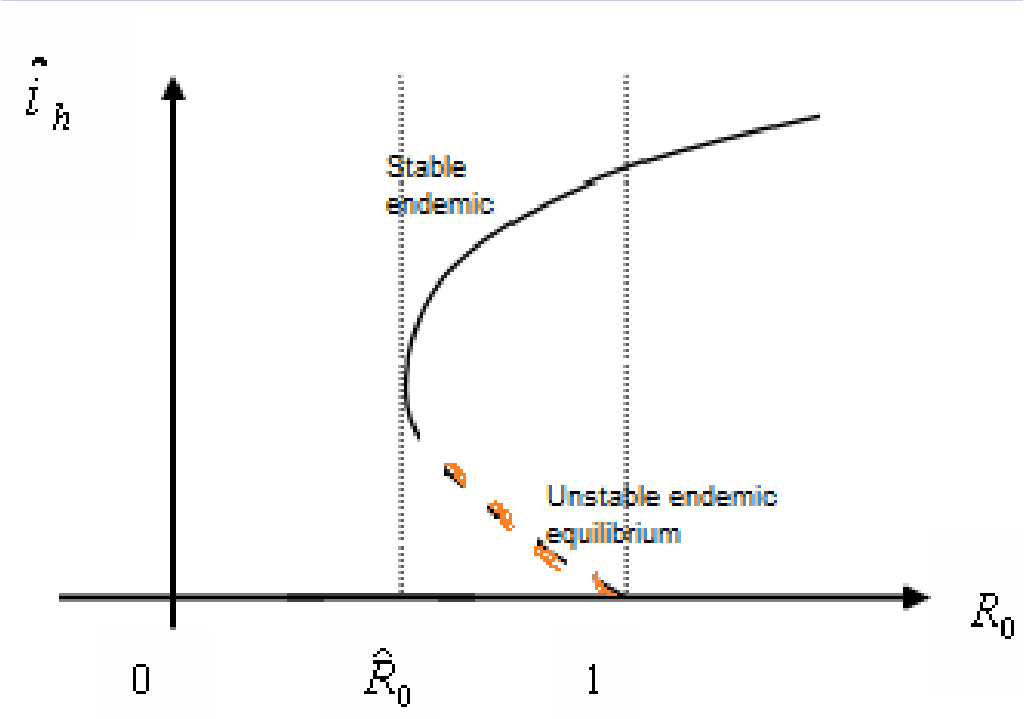


Figure 2: The backward bifurcation of the malaria model equation $(2.1)-(2.5)$

## 3 Numerical Simulations and Result

In this section we present numerical simulations of the model which is carried out using Matlab ode45. In this simulation, the population dynamics are observed in conditions such that $R_0 < 1$ and $R_0 > 1$. Thus we would like to show that the effect of vaccination, anti-malarial drug, and spraying in a situation where the disease does not spread. The data of parameters is gathered from Ethiopia, Oromia, West Arsi Zone, Siraro District Health Center were malaria disease is highly endemic and some parameters relied on the studies conducted by various reliable sources as indicated in Table 1. In Figure 3, the fractions of the populations, $S_h$ ,$I_h$ and $R_h$ are plotted versus time. The susceptible populations will initially decreases with time and then increases and the fractions of infected human populations decrease. The reproduction number is below one and the disease-free equilibrium point $E_0 = (S_h, 0, R_h)$ is stable. The susceptible and infected mosquito population decreases over time as shown in the figure.

## 4 Discussions and Conclusion

### 4.1 Discussions

House spraying with residual Insecticides and mosquito bed nets are the major intervention measures used in these days to prevent malaria transmission. These methods reduce the contact rates between the mosquitoes and humans. Other measures employ the use of anti-malarial drugs which have the effect of reducing the infectivity of the human host. Indoor Residual Spraying (IRS) reduces mosquito longevity and it also reduces mosquito fertility. This strategy is also likely to kill mosquitoes that rest indoors after feeding so it would increase the chances of killing infected mosquitoes. Indoor residual spraying increases the mosquito death rate $\mu_m$ and $\rho$, and reduces the

Table 3: **In 2016 Siraro District Health Center malaria concerned data were used for the simulations**

| No | Parameters | Values |
|---|---|---|
| 1 | $S_h\,(0)$ | 0.993 |
| 2 | $I_h\,(0)$ | 0.0043 |
| 3 | $R_h\,(0)$ | 0.0032 |
| 4 | $S_m\,(0)$ | 0.6 |
| 5 | $I_m\,(0)$ | 0.3 |
| 6 | $N_h\,(0)$ | 185739 |
| 7 | $N_m\,(0)$ | 5000 |
| 8 | $\lambda_h$ | 0.027 |
| 9 | $a$ | 0.038 |
| 10 | $\beta_1$ | 0.02 |
| 11 | $b$ | 0.13 |
| 12 | $\beta_2$ | 0.710 |
| 13 | $\theta$ | 0.3 |
| 14 | $\mu_h$ | 0.0004 |
| 15 | $\eta$ | 0.005 |
| 16 | $k$ | 0.611 |
| 17 | $\gamma$ | 0.2 |
| 18 | $\alpha$ | 0.05 |
| 19 | $\mu_m$ | 0.04 |
| 20 | $\rho$ | 0.5 |
| 21 | $\sigma$ | 0.001369 |
| 22 | $c$ | 0.022 |
| 23 | $\beta_3$ | 0.072 |
| 24 | $\lambda_m$ | 0.13 |

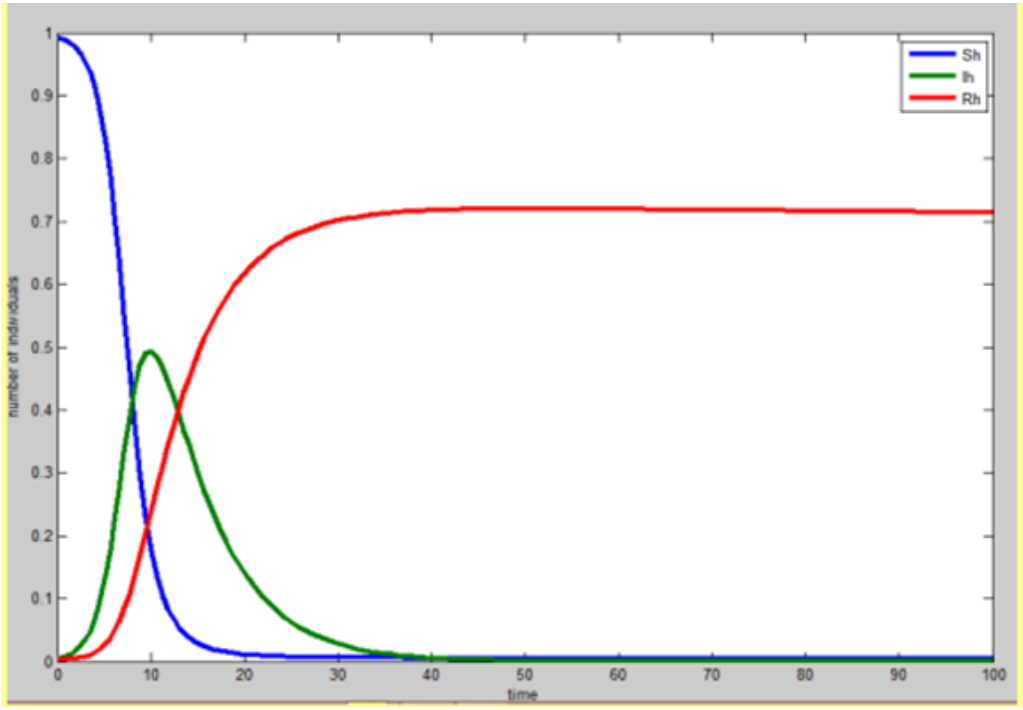


Figure 3: Simulation of the model (2.1) − (2.5) for susceptible, infected and recovered human population with the numerical values of $E_0 = \{1.4482, 0, 6.6313, 0.2407, 0\}$ and $E^* = \{0.10996326, 0.1640617, 168.62127, 0.00000241, 0.000007568\}$

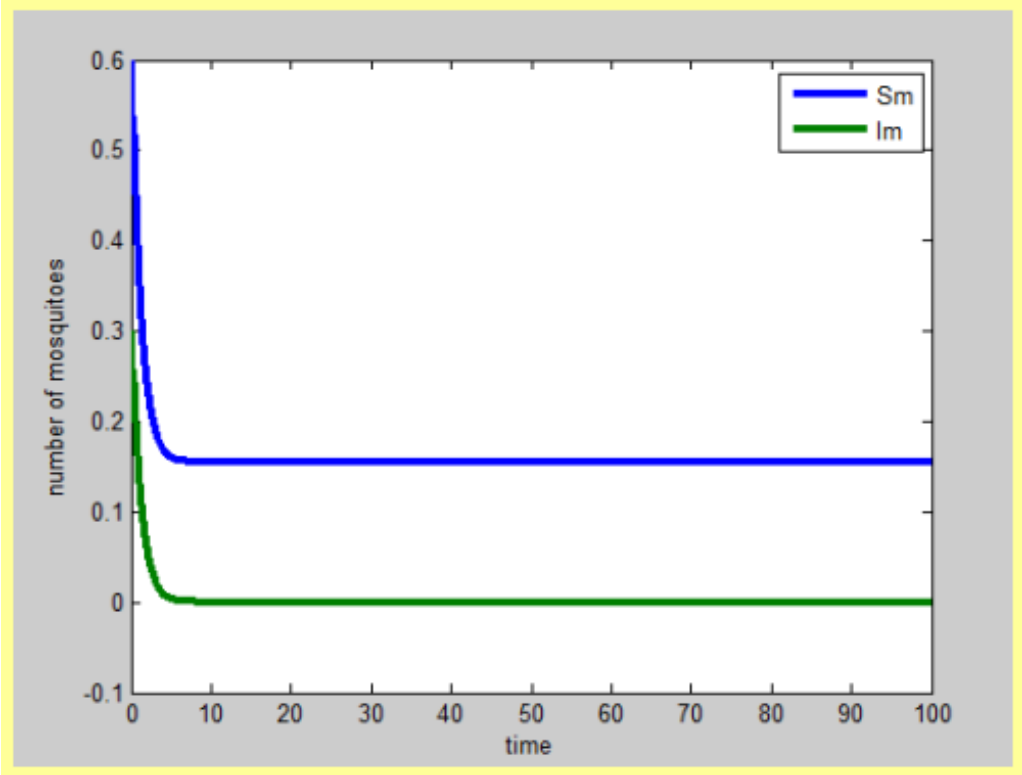


Figure 4: Simulation of the model equation (2.1) – (2.5) for susceptible and infected mosquito population

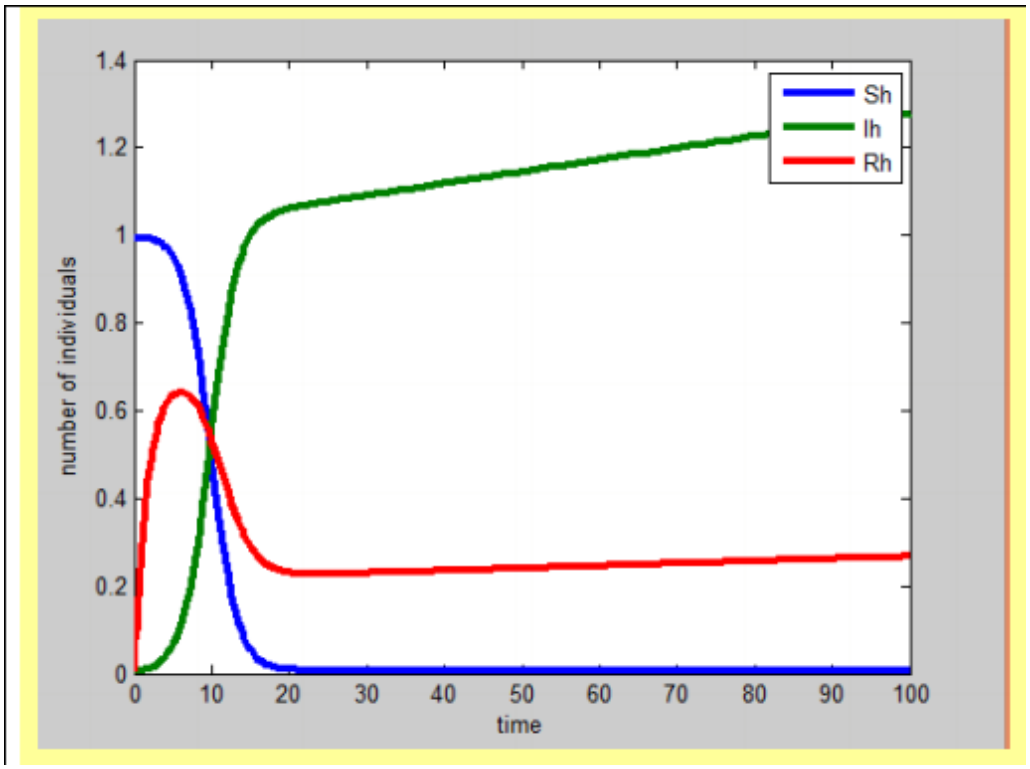


Figure 5: Simulation of the model (2.1) – (2.5) for$R_0 = 23.934$, with parameter values $\lambda_h = 0.027, \mu_h = 0.0004, \beta_1 = 0.02, \beta_2 = 0.710, \beta_3 = 0.072, \theta \in [0,1] = 0.45964, \eta = 0.1710, \rho \in [0,1], \sigma = 0.601369, \gamma \in [0.01,1], a = 0.038, b = 0.13, c = 0.022, \mu_m = 0.04, \lambda_m = 0.13$

number of mosquitoes, simultaneously it reduces disease transmission by blood transfusion. Increasing $\mu_m$ and $\rho$ can

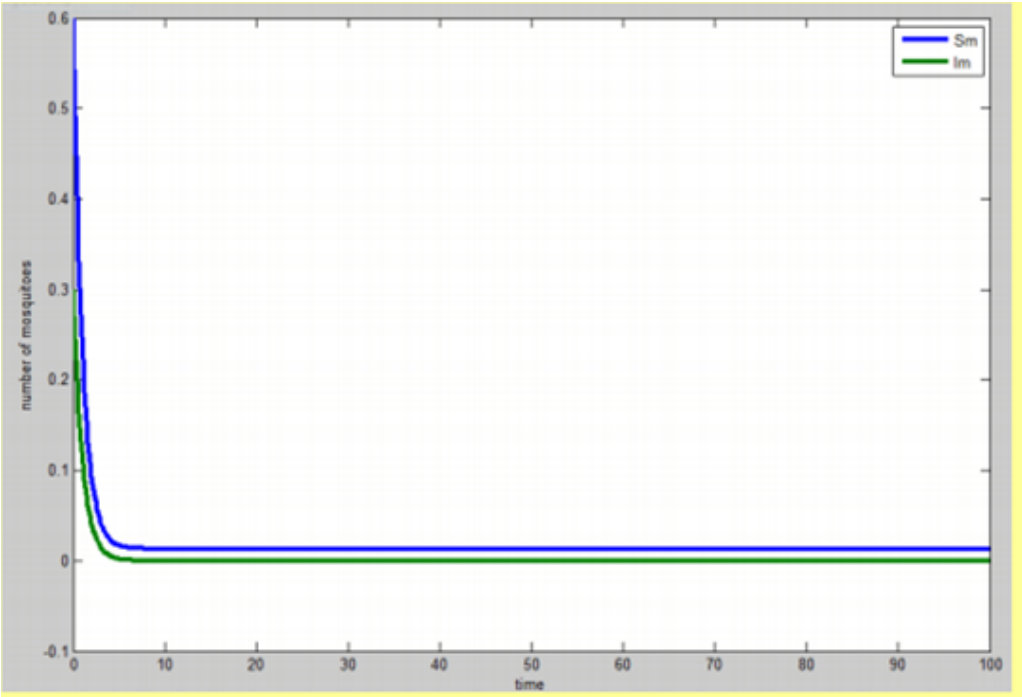


Figure 6: Simulation of the model (2.1) − (2.5) for susceptible and infected mosquitoes when $\lambda_m$ decreased from 0.13 to 0.013 and when $(\mu_m + \rho)$ is increased from 0.84 to 0.9984

be effective in reducing the malaria burden. The other intervention measure, Insecticide-Treated bed Nets, prevent mosquito-human contacts which reduce the number of bites per mosquito. Reducing the number of blood meals that each female mosquito gets would also lower the mosquito recruitment rate, $\lambda_m$, which in turn reduces the number of mosquitoes. This is found to be the most effective control strategy in reducing disease transmission. Therefore, all these control strategies are an effective way of controlling most of the parameters which are involved in our model. In order to determine the efficient way to tackle malaria, and reduce malaria mortality, it is necessary to know the relative importance of the different factors responsible for its transmission and prevalence. The basic reproduction number is such an important tool, which allows us to determine the importance of the parameters in the disease transmission. From our computation of the basic reproduction number we have noticed that $R_0$ is independent of the rate of immunity loss. We have also noticed that an increasing mosquito to human contact rate $\beta_2$, the mosquito recruitment rate $\lambda_m$, and the human to mosquito contact rate $\beta_3$, leads to an increase in the basic reproduction number and this leads to for disease to be hard controlling. Furthermore, we have seen that bringing $R_0$ less than unity is necessary but not sufficient condition for the disease eradication.

### 4.2 Conclusion

In this paper, a deterministic mathematical model for malaria transmission with treatment has been presented. It was showed that there exists a domain in which the model is mathematically and epidemiological well posed. The next generation matrix was used to derive the basic reproduction number $R_0$, which is the average number of new cases that one infected case, will generate. The disease free equilibrium of the model was proved to be locally asymptotically stable whenever $R_0$ less than unity. It is also showed that the disease free equilibrium is globally asymptotically stable provided that the basic reproduction number is less than some threshold. The possibility of multiple endemic equilibrium point was discussed. It was shown that the model undergo backward bifurcation phenomenon. The stable disease free equilibrium co-exists with the stable endemic equilibrium. Bringing the disease (malaria) induced death rate below some threshold was shown to be sufficient to eliminate backward bifurcation. Thus along with treated bed nets, and insecticides that would reduce the mosquito population there is a need for effective drug and efficient treatment which reduce the number of malaria induced death rate. We notice that in order to reduce the basic reproduction number below 1, intervention strategies need to be focused on treatment and reduction of the contact between mosquito and human host. Thus, there is a need for effective drugs, treated bed nets, and insecticides that would reduce the mosquito population and keep the human population stable.

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