Advances in Applied Mathematics and Mechanics
Adv. Appl. Math. Mech., Vol. **5**, No. 6, pp. 857-871

DOI: 10.4208/aamm.12-m12137
December 2013

# Mathematical Modelling of Malaria with Treatment

Mini Ghosh*

*School of Advanced Sciences, VIT University, Chennai Campus, Chennai, India*

Received 20 October 2012; Accepted (in revised version) 30 April 2013

Available online 6 September 2013

**Abstract.** This paper proposes a Susceptible-Infective-Susceptible (SIS) model to study the malaria transmission with treatment by considering logistic growth of mosquito population. In this work, it is assumed that the treatment rate is proportional to the number of infectives below the capacity and is constant when the number of infectives is greater than the capacity. We find that the system exhibits backward bifurcation if the capacity is small and it gives bi-stable equilibria which makes the system more sensitive to the initial conditions. The existence and stability of the equilibria of the model are discussed in-detail and numerical simulations are presented to illustrate the numerical results.

**AMS subject classifications**: 92D30, 37N25

**Key words**: Malaria, treatment, simulation.

## 1 Introduction

The malaria is a mosquito-borne infectious disease of humans and other animals that is caused by protists (i.e., a type of microorganism) of the genus Plasmodium. Based upon the current understanding, there are four species of Plasmodium which are responsible for malaria in humans: P. Falciparum, P. Vivax, P. Ovale, and P. Malariae. Out of these, the majority of deaths are caused by P. Falciparum and P. Vivax. The remaining two, P. Ovale, and P. Malariae, cause a generally milder form of malaria that is rarely fatal. Furthermore, the zoonotic species P. Knowlesi, prevalent in Southeast Asia, causes malaria in macaques but can also cause severe infections in humans. Normally, malaria is significant in tropical and subtropical regions because of several reasons. For example, the heavy rainfall, warm temperatures, and stagnant waters provide habitats ideal for mosquito larvae. In this regard, the disease transmission can be reduced by preventing mosquito bites by distribution of mosquito nets and insect repellents, or with mosquito-control measures such as spraying insecticides and draining stagnant water.

*Corresponding author.
*Email:* minighosh@vit.ac.in (M. Ghosh)

 

The P. Falciparum dominates in majority of malarial related deaths in Africa and South East Asia. It is responsible for nearly 80% of all malaria cases and nearly 90% of deaths, [1]. However in India roughly half of the cases of malaria are caused by the P. Falciparum, and half by the P. Vivax, [2]. In most of the tropical countries including India, the emergence of malaria has taken place and it has become endemic in the North-Eastern part of India, where this disease is spread by a lethal parasite called the Plasmodium Falciparum. Though, there are several experimental studies related to the surveys of malaria in different regions (see [3–10]), but the dynamics of malaria is very complex and there is a strong need to understand the transmission dynamics of malaria and the environmental factors which influence it.

Mathematical modeling is very helpful in understanding the dynamics of any infectious diseases and malaria is one of the diseases which is studied efficiently using modeling approaches. Sir Ronald Ross was the first to formulate a mathematical model for the P. Falciparum malaria in India by involving both, the human and mosquito population, [11]. This model was very simple and later it was modified by several researchers, (see [12–20]). Mathematical modeling of control of malaria by considering different aspects of controls has been discussed in [21–24].

Treatment of infective is an important parameter to control the spread of the disease related to malaria. In the classical epidemic models, the treatment rate of infectives is assumed to proportional to the number of infectives but this fact is applicable to the developing countries which have limited resources. As in case of limited resources, it is not possible to provide treatment to all infectives if the size of infective class is very large. Hence here we apply following treatment rate function which is described in [25]:

$$T(I)=\begin{cases} rI, & \text{if } 0\leq I\leq I_0, \\ k, & \text{if } I>I_0. \end{cases} \tag{1.1}$$

Where $I$ denotes infective class and $k=rI_0$. This means that the treatment rate is proportional to the number of the infectives when the capacity of treatment is not reached, and otherwise takes the maximal capacity. This describes the situation where patients have to be hospitalized: the number of hospital beds is limited. This is true also for the case where medicines are not sufficient.

In this paper, therefore, an SIS non-linear mathematical model is proposed and analyzed by incorporating treatment. In most of the existing malaria models, the population demography for mosquitoes has been assumed as of constant recruitment and death type. In the present work, we assume that the density of the mosquito population follows a generalized logistic model such that its growth rate decreases but its death rate increases as population density increases towards its carrying capacity with respect to the environment. This assumption is more realistic and reasonable as the mosquito population density is high in the regions that are conducive to its growth such as rivers and ponds as well as man-made habitats e.g., water storage tank, rice fields, barrels, irrigation channels, ditches, field wells etc. We analyze the model using the stability theory of

the differential equations. We show that the approach of reducing the basic reproduction number $R_0$ below one is not enough to eradicate the disease from the population and one need to lower the $R_0$ much below one, to have disease free equilibrium to be stable.

The remaining of this paper is organized as follows: Section 2 describes the model, Section 3 describes the existence and stability of different equilibria of the model, Section 4 demonstrates the numerical results. The paper ends with brief discussion in Section 5.

## 2 The model

We consider here an SIS model, where the human population density $N_1(t)$ is divided into two classes namely, the susceptible class $X_1(t)$ and the infective class $Y_1(t)$. The mosquito population density $N_2(t)$ is divided into the susceptible class $X_2(t)$ and the infective class $Y_2(t)$. It is assumed that mosquito population is growing logistically and the population demography for human is of constant recruitment and death type. Keeping in view the above and by considering the criss-cross interaction of the mosquito population with the human population, a mathematical model can be formulated as follows:

$$\dot{X}_1 = A - d_1 X_1 - \beta_1 X_1 Y_2 + \nu_1 Y_1 + T(Y_1), \tag{2.1a}$$

$$\dot{Y}_1 = \beta_1 X_1 Y_2 - (\nu_1 + \alpha_1 + d_1) Y_1 - T(Y_1), \tag{2.1b}$$

$$\dot{N}_1 = A - d_1 N_1 - \alpha_1 Y_1, \tag{2.1c}$$

$$\dot{X}_2 = \left(b_2 - a' \frac{r_2}{K_2} N_2\right) N_2 - \left\{d_2 + (1-a') \frac{r_2}{K_2} N_2\right\} X_2 - \beta_2 X_2 Y_1 - \alpha_2 X_2, \tag{2.1d}$$

$$\dot{Y}_2 = \beta_2 X_2 Y_1 - \left\{\alpha_2 + d_2 + (1-a') \frac{r_2}{K_2} N_2\right\} Y_2, \tag{2.1e}$$

$$\dot{N}_2 = r_2 N_2 \left(1 - \frac{N_2}{K_2}\right) - \alpha_2 N_2, \tag{2.1f}$$

$$N_1 = X_1 + Y_1, \qquad N_2 = X_2 + Y_2, \tag{2.1g}$$

$$X_1(0) > 0, \qquad Y_1(0) \geq 0, \qquad X_2(0) \geq 0, \qquad Y_2(0) \geq 0. \tag{2.1h}$$

And the treatment rate function is defined as follows:

$$T(Y_1) = \begin{cases} rY_1, & \text{if } 0 \leq Y_1 \leq \hat{Y}_{10}, \\ k, & \text{if } Y_1 > \hat{Y}_{10}, \quad \text{where } k = r\hat{Y}_{10}. \end{cases}$$

In model (2.1), $A$ is the constant immigration rate of the human population; $d_1$ is the natural death rate constant; $\beta_1$ is transmission rate of malaria from infected mosquitoes to susceptible humans; $\nu_1$ is the natural recovery rate coefficient of the human population; $\alpha_1$ is the disease related death rate constant; $b_2$ and $d_2$ are the birth and the death rate constants corresponding to the mosquito population; $r_2 = b_2 - d_2$ is the growth rate coefficient of the mosquito population; $K_2$ is the carrying capacity of the mosquito population in the

natural environment; $\alpha_2$ is the death rate of mosquitoes due to control measures ($r_2>\alpha_2$); $\beta_2$ is transmission coefficient of malaria from infected humans to susceptible mosquito and $0\leq a'\leq 1$ is a constant [26], which governs the logistic birth and logistic death of the mosquito population. Since $X_1+Y_1=N_1$ and $X_2+Y_2=N_2$, it is sufficient to consider the following system:

$$\dot{Y_1}=\beta_1(N_1-Y_1)Y_2-(\nu_1+\alpha_1+d_1)Y_1-T(Y_1), \tag{2.2a}$$
$$\dot{N_1}=A-d_1N_1-\alpha_1Y_1, \tag{2.2b}$$
$$\dot{Y_2}=\beta_2(N_2-Y_2)Y_1-\left\{\alpha_2+d_2+(1-a')\frac{r_2}{K_2}N_2\right\}Y_2, \tag{2.2c}$$
$$\dot{N_2}=r_2N_2\left(1-\frac{N_2}{K_2}\right)-\alpha_2N_2. \tag{2.2d}$$

Since the system (2.2) is autonomous, the effects of $N_2$ on the spread of malaria can be qualitatively studied by taking its asymptotic values as $t\to\infty$ in the last equation of the system (2.2). Thus we have

$$\text{for } N_2(0)>0, \quad \lim_{t\to\infty}\sup N_2=\frac{K_2}{r_2}(r_2-\alpha_2)=\bar{N_2}.$$

Now it suffices to study the global behavior of the system (2.2) by the following system of equations:

$$\dot{Y_1}=\beta_1(N_1-Y_1)Y_2-(\nu_1+\alpha_1+d_1)Y_1-T(Y_1), \tag{2.3a}$$
$$\dot{N_1}=A-d_1N_1-\alpha_1Y_1, \tag{2.3b}$$
$$\dot{Y_2}=\beta_2(\bar{N_2}-Y_2)Y_1-\left\{\alpha_2+d_2+(1-a')\frac{r_2}{K_2}\bar{N_2}\right\}Y_2. \tag{2.3c}$$

## 3 Existence of equilibria

It is easy to visualize the disease free equilibrium $E_0(0,A/d_1,0)$. An endemic equilibrium of the system (2.3) satisfies the following algebraic equations:

$$\beta_1(N_1-Y_1)Y_2-(\nu_1+\alpha_1+d_1)Y_1-T(Y_1)=0, \tag{3.1a}$$
$$A-d_1N_1-\alpha_1Y_1=0, \tag{3.1b}$$
$$\beta_2(\bar{N_2}-Y_2)Y_1-\left\{\alpha_2+d_2+(1-a')\frac{r_2}{K_2}\bar{N_2}\right\}Y_2=0. \tag{3.1c}$$

When $0<Y_1\leq\hat{Y}_{10}$, the system (3.1) becomes

$$\beta_1(N_1-Y_1)Y_2-(\nu_1+\alpha_1+d_1+r)Y_1=0, \tag{3.2a}$$
$$A-d_1N_1-\alpha_1Y_1=0, \tag{3.2b}$$
$$\beta_2(\bar{N_2}-Y_2)Y_1-\left\{\alpha_2+d_2+(1-a')\frac{r_2}{K_2}\bar{N_2}\right\}Y_2=0. \tag{3.2c}$$

When $Y_1 > \hat{Y}_{10}$, the system (3.1) becomes

$$\beta_1(N_1-Y_1)Y_2-(\nu_1+\alpha_1+d_1)Y_1-k=0, \tag{3.3a}$$
$$A-d_1N_1-\alpha_1Y_1=0, \tag{3.3b}$$
$$\beta_2(\bar{N}_2-Y_2)Y_1-\left\{\alpha_2+d_2+(1-a')\frac{r_2}{K_2}\bar{N}_2\right\}Y_2=0. \tag{3.3c}$$

The system (3.2) admits a unique positive solution $E^*(Y_1^*,N_1^*,Y_2^*)$, provided

$$\beta_1\beta_2\frac{A}{d_1}N_2^* > (\nu_1+\alpha_1+d_1+r)\left\{\alpha_2+d_2+(1-a')\frac{r_2}{K_2}N_2^*\right\}, \tag{3.4}$$

and $Y_1^*,N_1^*$, and $Y_2^*$ are given by

$$Y_1^* = \frac{\beta_1\beta_2\frac{A}{d_1}N_2^*-C_1(\nu_1+\alpha_1+d_1+r)}{\beta_2\left[\beta_1\left(1+\frac{\alpha_1}{d_1}\right)N_2^*+\nu_1+\alpha_1+d_1+r\right]},$$
$$N_1^* = \frac{A-\alpha_1Y_1^*}{d_1},$$
$$Y_2^* = \frac{(\nu_1+\alpha_1+d_1+r)Y_1^*}{\beta_1(N_1^*-Y_1^*)}.$$

Let

$$\Re_0 = \frac{\beta_1\beta_2\frac{A}{d_1}N_2^*}{(\nu_1+\alpha_1+d_1+r)\left\{\alpha_2+d_2+(1-a')\frac{r_2}{K_2}N_2^*\right\}}.$$

Then $\Re_0$ is the basic reproduction number for the system (2.3) and $E^*(Y_1^*,N_1^*,Y_2^*)$ is an endemic equilibrium of (2.3) if and only if

$$1<\Re_0\le 1+\left[\frac{\beta_1\beta_2N_2^*\left(1+\frac{\alpha_1}{d_1}\right)}{C_1(\nu_1+\alpha_1+d_1+r)}+\frac{\beta_2}{C_1}\right]\hat{Y}_{10}.$$

Now to get the positive solution of the system (3.3), we get following from the second and third equations respectively:

$$N_1 = \frac{A-\alpha_1Y_1}{d_1}, \tag{3.5a}$$
$$Y_2 = \frac{\beta_2N_2^*Y_1}{\beta_2Y_1+C_1}, \tag{3.5b}$$

where

$$C_1 = \alpha_2+d_2+(1-a')\frac{r_2}{K_2}N_2^*.$$

Substituting these values of $N_1$ and $Y_2$ into the third equation of the system (3.3), we get following quadratic in $Y_1$,

$$\left[\beta_1\beta_2 N_2^*\left(1+\frac{\alpha_1}{d_1}\right)+\beta_2(\nu_1+\alpha_1+d_1)\right]Y_1^2-\left[\beta_1\beta_2 N_2^*\frac{A}{d_1}-\beta_2 k-C_1(\nu_1+\alpha_1+d_1)\right]Y_1 +C_1 k=0.$$

For this quadratic to have real positive roots, we first need the coefficient of $Y_1$, say $B$, to be negative, which implies

$$-B=\beta_1\beta_2 N_2^*\frac{A}{d_1}-\beta_2 k-C_1(\nu_1+\alpha_1+d_1)>0,$$

i.e.,

$$R_0 C_1(\nu_1+\alpha_1+d_1+r)>\beta_2 k+C_1(\nu_1+\alpha_1+d_1).$$

This gives,

$$\Re_0>1+\frac{\beta_2 k-C_1 r}{C_1(\nu_1+\alpha_1+d_1+r)}. \tag{3.6}$$

Additionally we want the discriminant $\Delta$ to be nonnegative, i.e.,

$$\Delta=B^2-4kC_1\left[\beta_1\beta_2 N_2^*\left(1+\frac{\alpha_1}{d_1}\right)+\beta_2(\nu_1+\alpha_1+d_1)\right]\geq 0,$$

which implies

$$\Re_0\geq 1+\frac{\beta_2 k-C_1 r}{C_1(\nu_1+\alpha_1+d_1+r)}+\frac{C_2}{(\nu_1+\alpha_1+d_1+r)}=:p_0 \tag{3.7}$$

or

$$R_0\leq 1+\frac{\beta_2 k-C_1 r}{C_1(\nu_1+\alpha_1+d_1+r)}-\frac{C_2}{(\nu_1+\alpha_1+d_1+r)}, \tag{3.8}$$

where

$$C_2=\sqrt{\frac{4k\left[\beta_1\beta_2 N_2^*\left(1+\frac{\alpha_1}{d_1}\right)+\beta_2(\nu_1+\alpha_1+d_1)\right]}{C_1}}.$$

Note that coefficient of $Y_1$ is negative under the condition (3.6), so it can be observed that the condition (3.7) is necessary and sufficient for the existence of real positive roots of the last quadratic in $Y_1$. Let these positive roots are given by

$$Y_1^{**}=\frac{-B-\sqrt{\Delta}}{2\left[\beta_1\beta_2 N_2^*\left(1+\frac{\alpha_1}{d_1}\right)+\beta_2(\nu_1+\alpha_1+d_1)\right]}, \tag{3.9a}$$

$$Y_1^{***}=\frac{-B+\sqrt{\Delta}}{2\left[\beta_1\beta_2 N_2^*\left(1+\frac{\alpha_1}{d_1}\right)+\beta_2(\nu_1+\alpha_1+d_1)\right]}. \tag{3.9b}$$

After finding these values of $Y_1$ corresponding values of $N_1$ and $Y_2$ can be found using the Eqs. (3.5a) and (3.5b) respectively. Thus we have two more equilibrium point, say, $E_1(Y_1^{**},N_1^{**},Y_2^{**})$ and $E_2(Y_1^{***},N_1^{***},Y_2^{***})$. And $E_1$ is an endemic equilibrium of (3.3) if $Y_1^{**}>\hat{Y}_{10}$. Similarly $E_2$ is an endemic equilibrium of (3.3) if $Y_1^{***}>\hat{Y}_{10}$. Let us consider the condition under which $Y_1^{**}>\hat{Y}_{10}$. By the definition, we see that it is equivalent to

$$-B-\sqrt{\Delta}>2\Big[\beta_1\beta_2N_2^*\Big(1+\frac{\alpha_1}{d_1}\Big)+\beta_2(\nu_1+\alpha_1+d_1)\Big]\hat{Y}_{10}. \tag{3.10}$$

This implies that

$$B+2\Big[\beta_1\beta_2N_2^*\Big(1+\frac{\alpha_1}{d_1}\Big)+\beta_2(\nu_1+\alpha_1+d_1)\Big]\hat{Y}_{10}<0. \tag{3.11}$$

It follows from the definition of $B$ that

$$\Re_0>1+\frac{\beta_2k-C_1r}{C_1(\nu_1+\alpha_1+d_1+r)}+\frac{2\big[\beta_1\beta_2N_2^*\big(1+\frac{\alpha_1}{d_1}\big)+\beta_2(\nu_1+\alpha_1+d_1)\big]\hat{Y}_{10}}{C_1(\nu_1+\alpha_1+d_1+r)}=:p_1. \tag{3.12}$$

Further, (3.10) implies that

$$\Big[B+2\Big\{\beta_1\beta_2N_2^*\Big(1+\frac{\alpha_1}{d_1}\Big)+\beta_2(\nu_1+\alpha_1+d_1)\Big\}\hat{Y}_{10}\Big]^2>\Delta. \tag{3.13}$$

By direct calculation it can be seen that (3.13) is equivalent to

$$\Re_0<1+\Big[\frac{\beta_1\beta_2N_2^*\Big(1+\frac{\alpha_1}{d_1}\Big)}{C_1(\nu_1+\alpha_1+d_1+r)}+\frac{\beta_2}{C_1}\Big]\hat{Y}_{10}=:p_2. \tag{3.14}$$

Hence, $Y_1^{**}>\hat{Y}_{10}$ holds if and only if inequalities (3.12) and (3.14) are valid. Moreover, if $\Re_0\le p_1$ or $\Re_0\ge p_2$, we have $Y_1^{**}\ge\hat{Y}_{10}$ By similar arguments as above, we see that $Y_1^{***}>\hat{Y}_{10}$ if (3.12) holds or

$$p_2<\Re_0\le p_1. \tag{3.15}$$

Furthermore, $Y_1^{***}<\hat{Y}_{10}$ if

$$\Re_0\le\min\{p_1,p_2\}. \tag{3.16}$$

Summarizing the discussion above, we have the following conclusions.

**Theorem 3.1.** *$E^*=(Y_1^*,N_1^*,Y_2^*)$ is an endemic equilibrium of* (2.3) *if and only if $1<\Re_0\le p_2$. Furthermore, $E^*$ is the unique endemic equilibrium of* (2.3) *if $1<\Re_0\le p_2$ and one of following conditions is satisfied:*

*(i)* $\Re_0<p_0$.

*(ii)* $p_0 \leq \Re_0 < p_1$.

Note that

$$C_1 r > \left[\beta_1\beta_2 N_2^*\left(1+\frac{\alpha_1}{d_1}\right)+\beta_2(\nu_1+\alpha_1+d_1)\right]\hat{Y}_{10}$$

is equivalent to that $p_1 < p_2$. We also have the following theorem.

**Theorem 3.2.** *$E_1$ and $E_2$ do not exist when $\Re_0 < p_0$. Further, if $\Re_0 \geq p_0$, we get following:*

*(i) If*

$$C_1 r > \left[\beta_1\beta_2 N_2^*\left(1+\frac{\alpha_1}{d_1}\right)+\beta_2(\nu_1+\alpha_1+d_1)\right]\hat{Y}_{10},$$

*then both $E_1(Y_1^{**},N_1^{**},Y_2^{**})$ and $E_2(Y_1^{***},N_1^{***},Y_2^{***})$ exist when $p_1 < \Re_0 < p_2$.*

*(ii) If*

$$C_1 r > \left[\beta_1\beta_2 N_2^*\left(1+\frac{\alpha_1}{d_1}\right)+\beta_2(\nu_1+\alpha_1+d_1)\right]\hat{Y}_{10},$$

*then $E_1$ does not exist but $E_2$ exists if $\Re_0 \geq p_2$.*

*(iii) Let*

$$C_1 r \leq \left[\beta_1\beta_2 N_2^*\left(1+\frac{\alpha_1}{d_1}\right)+\beta_2(\nu_1+\alpha_1+d_1)\right]\hat{Y}_{10}.$$

*Then $E_1$ does not exist. Further, $E_2$ exists when $p_2 < \Re_0$ and $E_2$ does not exist when $\Re_0 \leq p_2$.*

**Corollary 3.1.** *Eq.* (2.3) *has a backward bifurcation with endemic equilibria when $\Re_0 < 1$ if*

$$C_1 r > \left[\beta_1\beta_2 N_2^*\left(1+\frac{\alpha_1}{d_1}\right)+\beta_2(\nu_1+\alpha_1+d_1)\right]\hat{Y}_{10}$$

*and $p_0 < 1$.*

*Proof.* This corollary is a simple consequence of (i) of Theorem 3.2. □

**Theorem 3.3.** *$E_0$ is asymptotically stable if $\Re_0 < 1$ and is unstable if $\Re_0 \geq 1$. $E^*$ is asymptotically stable if $Y_1^* < \hat{Y}_{10}$.*

*Proof.* It is easy to proof that $E_0$ is asymptotically stable if $\Re_0 < 1$ and is unstable if $\Re_0 \geq 1$, so we omit it. Now to check the local stability of the equilibrium $E^*$ which exists only when $Y_1^* < \hat{Y}_{10}$, we find the following variational matrix of the system (2.3) at the equilibrium $E^*$,

$$M^* = \begin{pmatrix} -(\beta_1 Y_2^* + \nu_1 + \alpha_1 + d_1 + r) & \beta_1 Y_2^* & \beta_1(N_1^* - Y_1^*) \\ -\alpha_1 & -d_1 & 0 \\ \beta_2(N_2^* - Y_2^*) & 0 & -[\beta_2 Y_1^* + C_1] \end{pmatrix}.$$

The characteristic polynomial corresponding to matrix $M^*$ is

$$\psi^3 + b_1\psi^2 + b_2\psi + b_3 = 0,$$

where

$$b_1=\beta_1 Y_2^*+\nu_1+\alpha_1+2d_1+r+\beta_2 Y_1^*+C_1,$$
$$b_2=(\beta_1 Y_2^*+\nu_1+\alpha_1+d_1+r)(d_1+\beta_2 Y_1^*)+\beta_1 Y_2^* C_1+\alpha_1\beta_1 Y_2^*+d_1[\beta_2 Y_1^*+C_1],$$
$$b_3=\beta_1 Y_2^*(\alpha_1+d_1)[\beta_2 Y_1^*+C_1]+\beta_2 Y_1^* d_1(\nu_1+\alpha_1+d_1+r).$$

We note here that $b_1>0$ and also $b_1b_2-b_3>0$. Hence by the Routh-Hurwitz criteria the equilibrium $E^*$, if it exists, is locally asymptotically stable. □

# 4 Simulation

The system (2.3) is simulated for various sets of parameters by fourth order Runge-Kutta method using the package XPP, [27]. In Figs. 1-6, ($N_1$, $Y_1$) phase planes are drawn

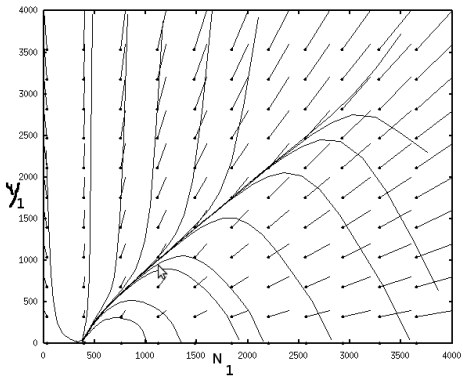


Figure 1: Phase plot of $Y_1$ verses $N_1$ showing infection free equilibrium to be stable when $R_0=0.78<1$ for the parameter values $r=1.2$, $A=300$, $d_1=0.8$, $\beta_1=\beta_2=0.0005$, $\nu_1=0.1$, $\alpha_1=0.01$, $\alpha_2=0.4$, $d_2=3$, $a'=0.6$, $r_2=5$, $K_2=100000$, $\hat{Y}_{10}=200$.

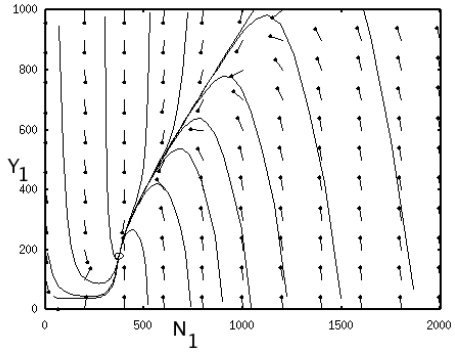


Figure 2: Phase plot of $Y_1$ verses $N_1$ showing the existence of only $E^*$ which is stable when $1<R_0<p_2$ and $R_0<p_0$ for the parameter values $r=1.2$, $A=300$, $d_1=0.8$, $\beta_1=\beta_2=0.0008$, $\nu_1=0.1$, $\alpha_1=0.01$, $\alpha_2=0.4$, $d_2=3$, $a'=0.6$, $r_2=5$, $K_2=100000$, $\hat{Y}_{10}=200$.

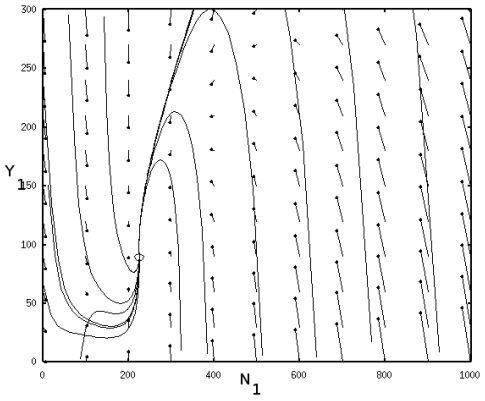


Figure 3: Phase plot of $Y_1$ verses $N_1$ showing the existence of only $E^*$ which is stable when $1<R_0<p_2$ and $p_0<R_0<p_1$ for the parameter values $r=1.19$, $A=185$, $d_1=0.8$, $\beta_1=\beta_2=0.0009$, $\nu_1=0.05$, $\alpha_1=0.05$, $\alpha_2=0.4$, $d_2=3$, $a'=0.7$, $r_2=5$, $K_2=100000$, $\hat{Y}_{10}=90$.

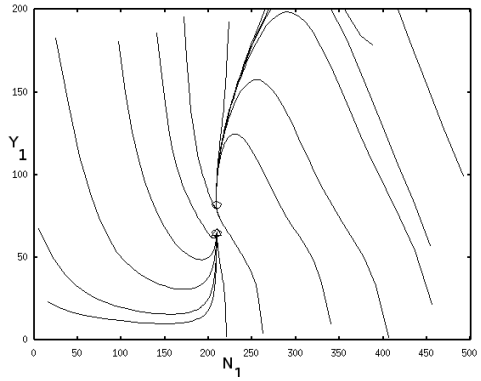


Figure 4: Phase plot of $Y_1$ verses $N_1$ showing bi-stability when $p_0<R_0<1$ and $p_1<R_0<p_2$ for the parameter values $r=1.2$, $A=168$, $d_1=0.8$, $\beta_1=\beta_2=0.0009$, $\nu_1=0.02$, $\alpha_1=0.01$, $\alpha_2=0.4$, $d_2=3$, $a'=0.6$, $r_2=5$, $K_2=100000$, $\hat{Y}_{10}=65$.

which confer the existence and the stability of different equilibria of the system (2.3). Fig. 1 is showing the global stability of disease free equilibrium point $E_0(0,375,0)$ when $R_0=0.78<1$ and other equilibria do not exist. Fig. 2 is describing the situation when only $E^*(179.82,372.75,2458.2)$ exists and is stable. Here $R_0=1.997$ which satisfies the condition mentioned in the Theorem 3.1(i). Fig. 3 is supporting the statement in Theorem 3.1(ii), where only $E^*(89.34967,225.666,1522.124)$ is stable for $R_0=1.7249$. In Fig. 4, we have shown bi-stability where the equilibrium $E_2(81.81,208.98,1274.8)$ and the equilibrium point $E^*(64.85,209.19,1013.5)$ are stable and $E_1(65.56,209.18,1024.46)$ is saddle. This supports the analytical results stated in Theorem 3.2(i). Figs. 5-6 are showing the existence and the stability of the equilibrium $E_2$ alone which confer the Theorem 3.2(ii) and Theorem 3.2(iii) respectively. And in this case $E_2$ comes out to be $(257.434,371.782,3479.128)$ and $(234.076,309.574,3933.995)$ respectively. Here the infection free equilibrium $E_0$ is un-

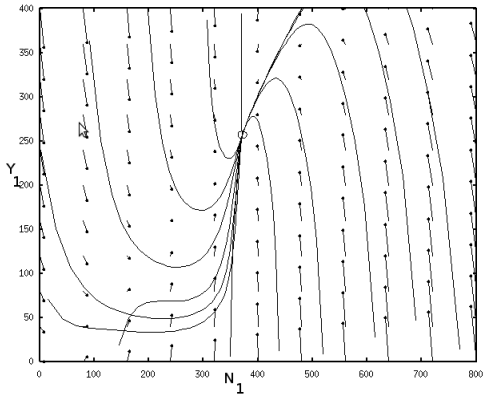


Figure 5: Phase plot of $Y_1$ verses $N_1$ showing the existence of only $E_2$ which is stable when $p_1 < p_2$, $p_0 < R_0$ and $R_0 > p_2 > 1$ for the parameter values $r = 1.2$, $A = 300$, $d_1 = 0.8$, $\beta_1 = \beta_2 = 0.0008$, $\nu_1 = 0.1$, $\alpha_1 = 0.01$, $\alpha_2 = 0.4$, $d_2 = 3$, $a' = 0.6$, $r_2 = 5$, $K_2 = 100000$, $\hat{Y}_{10} = 70$.

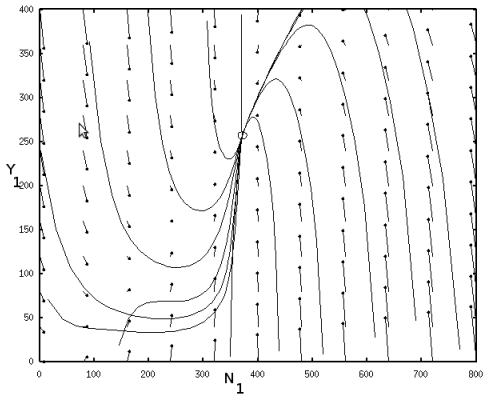


Figure 6: Phase plot of $Y_1$ verses $N_1$ showing the existence of only $E_2$ which is stable when $R_0 > p_0$, $p_1 > p_2$ and $p_2 < R_0$ for the parameter values $r = 1.2$, $A = 250$, $d_1 = 0.8$, $\beta_1 = \beta_2 = 0.001$, $\nu_1 = 0.1$, $\alpha_1 = 0.01$, $\alpha_2 = 0.4$, $d_2 = 3$, $a' = 0.6$, $r_2 = 5$, $K_2 = 100000$, $\hat{Y}_{10} = 70$.

stable. From all these phase-plane diagram it is clear that when ever $E_2$ exists, the number of infectives i.e., $Y_1^{***}$ is always greater than the number of infectives corresponding to the other equilibria if they exist. This fact is more clear from the bifurcation diagrams (see Figs. 7-10). Fig. 7 is obtained by considering the recruitment rate $A$ as the critical parameter. The horizontal axis is labelled with the appropriate value of the reproduction number $R_0$ corresponding to this bifurcation parameter $A$. It is observed that when the reproduction number $R_0$ is between 0 to 0.480954, the infection free equilibrium alone is stable, for $0.480954 < R_0 < 1$ we have bi-stability where either the infection free equilibrium is stable or the equilibrium $E_2$ is stable. For $1 < R_0 < 1.050146$, again we have bi-stability where we have two stable endemic equilibria. Here either the equilibrium $E^*$ is stable or the equilibrium $E_2$ is stable. The equilibrium $E_1$ when it exists is always

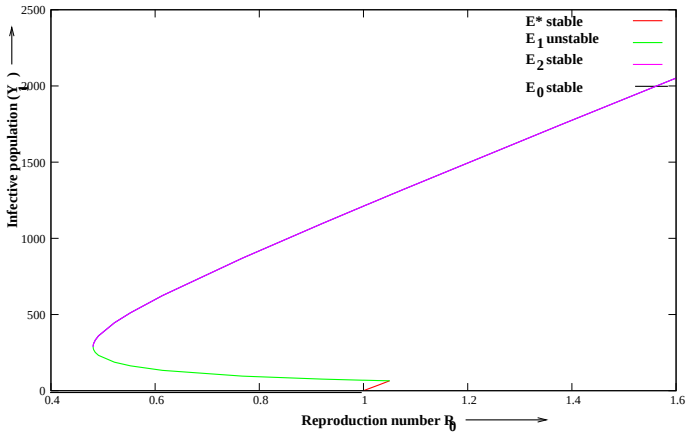


Figure 7: The variation of equilibrium level of the infective population with the reproduction number showing the backward bifurcation from an endemic equilibrium at $R_0=1.050146$ for the parameters values $r=0.8$, $d_1=0.014$, $\beta_1=\beta_2=0.00012$, $\nu_1=0.02$, $\alpha_1=0.01$, $\alpha_2=0.4$, $d_2=2$, $a'=0.6$, $r_2=3$, $K_2=100000$, $\hat{Y}_{10}=65$ when the critical parameter is $A$.

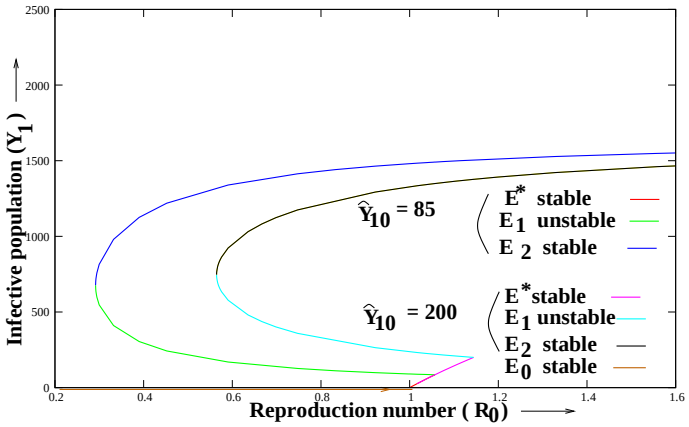


Figure 8: The variation of equilibrium level of the infective population with the reproduction number showing the backward bifurcation from an endemic equilibrium at $R_0=1.056781$ for the parameters $r=0.8$, $A=40$, $d_1=0.014$, $\nu_1=0.02$, $\alpha_1=0.01$, $\alpha_2=0.4$, $d_2=2$, $a'=0.7$, $r_2=3$, $K_2=100000$ for $\hat{Y}_{10}=85$ and 200 when the critical parameter is $\beta_1=\beta_2$.

saddle. The second bifurcation diagram (see Fig. 8) is obtained by considering the transmission coefficient $\beta_1=\beta_2$ as the critical parameter. Here too we get similar plot showing the bi-stability and the backward bifurcation. From these two plots it is clear that just reducing the reproduction number $R_0$ below one is not always sufficient to eliminate the disease from the population. As there is a backward bifurcation so we need to reduce $R_0$ well below one to make the unique infection free equilibrium to be stable. In Figs. 9-10, bifurcation diagram is obtained by taking the threshold value of the infective population $\hat{Y}_{10}$ as the critical parameter. Here it is noted that this parameter is involved in the treatment. As it is assumed that treatment is proportional to the number of infective until the

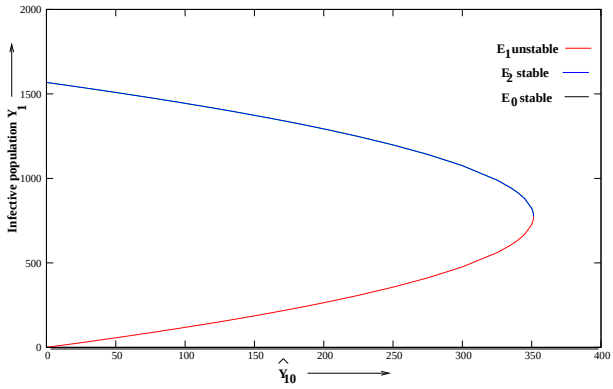


Figure 9: The variation of equilibrium level of the infective population with $\hat{Y}_{10}$ when $R_0 = 0.9226 < 1$ for the parameters $r = 0.8$, $A = 40$, $d_1 = 0.014$, $\beta_1 = \beta_2 = 0.0001$, $\nu_1 = 0.02$, $\alpha_1 = 0.01$, $\alpha_2 = 0.4$, $d_2 = 2$, $a' = 0.7$, $r_2 = 3$, $K_2 = 100000$ where $\hat{Y}_{10}$ is the critical parameter.

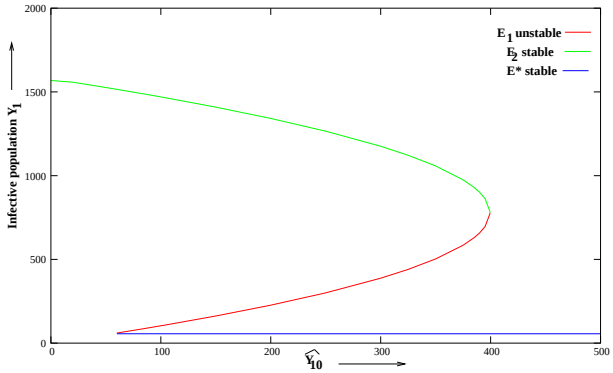


Figure 10: The variation of equilibrium level of the infective population with $\hat{Y}_{10}$ when $R_0 = 1.036636 > 1$ for the parameters $r = 0.8$, $A = 40$, $d_1 = 0.014$, $\beta_1 = \beta_2 = 0.000106$, $\nu_1 = 0.02$, $\alpha_1 = 0.01$, $\alpha_2 = 0.4$, $d_2 = 2$, $a' = 0.7$, $r_2 = 3$, $K_2 = 100000$ where $\hat{Y}_{10}$ is the critical parameter.

infective population reaches a threshold value $\hat{Y}_{10}$ and after that the treatment function is a constant. Due to this fact in this figure we see that the equilibrium level of the infective population decreases with the increase in $\hat{Y}_{10}$ until it comes to a saturation point. Fig. 9 is describing the situation when the reproduction number $R_0 < 1$ and either the infection free equilibrium $E_0$ is stable or the equilibrium $E_2$ is stable. Here when we increase $\hat{Y}_{10}$ the equilibrium level of the infective population (corresponding to the equilibrium $E_2$) decreases until we arrive at $\hat{Y}_{10} = 351.34693$ where increasing it further does not have any effect and in this case only the infection free equilibrium $E_0$ is stable. Fig. 10 is obtained when the reproduction number $R_0 > 1$ and we can see that the equilibrium level of the infective population decreases with the increase in $\hat{Y}_{10}$ until $\hat{Y}_{10} = 60$. And increasing $\hat{Y}_{10}$ further gives bi-stability where either the equilibrium $E_2$ is stable or the equilibrium $E^*$ is stable. The equilibrium $E_1$ is saddle in between these two nontrivial equilibria. Then

increasing $\hat{Y}_{10}$ further gives the stability of the equilibrium $E^*$ alone. This is the situation when the medical facilities are more than sufficient and hence there is no effect of improving it further. From all these results it is clear that increasing the capacity of treatment has positive impact in reducing the infection prevalence of the disease irrespective of $R_0 < 1$ or $R_0 > 1$. From Fig. 8, it is easy to see that there is a sift of bifurcation diagram with the increase in $\hat{Y}_{10}$. This tells us that the threshold value of $R_0$ for disease free equilibrium to be stable can be increased with the increase in the capacity of treatment. And it is easy to visualize that with the further increase in the capacity of treatment, we may not get backward bifurcation. Also from Figs. 9-10, it is clear that increasing the capacity of treatment leads to decrease in the equilibrium level of infective population which leads to reduction in infection prevalence of the disease.

## 5 Conclusions

This paper has presented a mathematical model for the malaria with treatment. We have discussed different equilibria and their stability conditions and performed simulation which is also consistent with the analytic results. Similar to the other SIS models with treatment, the presented malaria model too shows backward bifurcation which makes this system vulnerable to initial data and also in this case $R_0 < 1$ is not sufficient for system to tend to disease free equilibrium point. Since the bi-stability occurs, system may tend to endemic equilibrium for some set of initial values. We have shown that there is some threshold value which should be much below one, and if $R_0$ is less than that then only disease free equilibrium will be globally stable. Otherwise for $R_0 < 1$ too, the local stability of disease free equilibrium point is guaranteed only when the level of initial infectious invasion is much low. Furthermore, we have presented numerical simulation to describe the analytical results and bifurcation results, and the simulation results illustrate the presented model well.

## Acknowledgments

The author thanks the handling editor and anonymous referees for their valuable comments and suggestions which led to an improvement of the original manuscript.

## References

[1] World Health Organization (WHO) and WHO Global Malaria Programme. See `http://www.who.int/topics/malaria/en/`, `http://www.who.int/malaria/about_us/en/index.html`.

[2] M. MARTCHEVA AND F. HOPPENSTEADT, *India's approach to eliminate Plasmodium falciparum malaria: a modelling perspective*, J. Biological Systems, 18(4) (2010), pp. 867–891.

[3] S. C. DAS, M. BHUYAN, L. BARUAH AND P. K. TALUKDAR, *Mosquito survey in Tripura*, Indian J. Malariology, 28 (1991), pp. 129–134.

[4] V. P. SHARMA, *Re-emergence of malaria in India*, Indian J. Med. Research, 103 (1996), pp. 26–45.
[5] V. P. SHARMA, *Fighting malaria in India*, Current Science, 75 (1998), pp. 1127–1140.
[6] V. P. SHARMA, *Battling the malaria iceberg with chloroquine in India*, Malaria J., 6 (2007), pp. 105.
[7] M. SITE, *Malaria in India*, `http://www.malariasite.com/MALARIA/malaria-india.htm`.
[8] D. H. HAMER, M. P. SINGH, B. J. WYLIE, K. YEBOAH-ANTWI, J. TUCHMAN, M. DESAI, V. UDHAYAKUMAR, P. GUPTA, M. I. M. I. BROOKS, M. M. SHUKLA, K. AWASTHY, L. SABIN, W. B. MACLEOD, A. P. DASH AND N. SINGH, *Burden of malaria in pregnancy in Jharkhand State, India*, Malaria J., 8 (2009), pp. 209.
[9] V. SINGH, N. MISHRA, G. AWASTHI, A. P. DASH AND A. DAS, *Why is it important to study malaria epidemiology in India?* Trends Paras, 25(10) (2009), pp. 452–457.
[10] V. P. SHARMA, *Artemisinin drugs in the treatment of P. Falciparum Malaria in India*, Curr. Sci., 90(10) (2006), pp. 1323–1324.
[11] R. ROSS, *The prevention of Malaria*, Eng. Edn. John Murray London, 4-10 (1911).
[12] J. RADCLIFFE, *The initial geographical spread of host-vector and carrier borne epidemics*, J. Appl. Prob., 10 (1973), pp. 703–717.
[13] J. RADCLIFFE, *The periodicity of endemic malaria*, J. Appl. Prob., 11(3) (1997), pp. 562–567.
[14] K. DIETZ, L. MOLINEAUX AND A. THOMAS, *A Malaria model tested in the African Savannah*, Bull. Wld. Hlth. Org., 50 (1974), pp. 347–357.
[15] L. MOLINEAUX, K. DIETZ AND A. THOMAS, *Further epidemiological evaluation of a malaria model*, Bull. Wld. Hlth. Org., 56 (1978), pp. 565–571.
[16] N. T. J. BAILEY, The Biomathematics of Malaria, Griffin, London, 1982.
[17] G. A. NGWA AND W. S. SHU, *A mathematical model for endemic malaria with variable human and mosquito populations*, Math. Comput. Model., 32 (2000), pp. 747–763.
[18] N. CHITNIS, J. M. CUSHING AND J. M. HYMAN, *Bifurcation Analysis of mathematical model for malaria transmission*, SIAM J. Appl. Math., 67(1) (2006), pp. 24–45.
[19] N. CHITNIS, J. M. HYMAN AND J. M. CUSHING, *Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model*, Bull Math Biol., 70 (2008), pp. 1272–1296.
[20] SANDIP MANDAL, RAMRUP SARKAR AND SOMDATTA SINHA, *Mathematical models od malaria-a review*, Malaria J., 10 (2011), pp. 202.
[21] BYUL NIM KIM, KYEONGAH NAH, CHAESHIN CHU, SANG UK RYU, YONG HAN KANG AND YONGKUK KIM, *Optimal control strategy of plasmodium vivax Malaria transmission in Korea*, Osong Public Health and Research Perspectives, 3(3) (2012), pp. 128–136.
[22] H. DIAZ, A. A. RAMIREZ, A. OLARTE AND C. CLAVIJO, *A model for the control of malaria using genetically modified vectors*, J. Theoretical Biology, 276(1) (2011), pp. 57–66.
[23] ABID ALI LASHARI AND GUL ZAMAN, *Optimal control of a vector borne disease with horizontal transmission*, Nonlinear Anal. Real World Appl., 13(1) (2012), pp. 203–212.
[24] MINI GHOSH, ABID ALI LASHARI AND XUE-ZHI LI, *Biological control of malaria: a mathematical model*, Appl. Math. Comput., 219 (15), pp. 7923–7939.
[25] W. D. WANG, *Backward bifurcation of an epidemic model with treatment*, Math. Biosci., 201 (2006), pp. 58–71.
[26] L. Q. GAO AND H. W. HETHCOTE, *Disease transmission models with density dependent demographics*, J. Math. Bio., 32 (1992), pp. 717–731.
[27] B. ERMENTROUT, Simulating, Analyzing, and Animating Dynamical Systems: A Guide to XPPAUT for Researchers and Students, 1st edition, SIAM, Philadelphia, PA, 2002.