

# Transmission Dynamics of Malaria in Ghana

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## Abstract

In this paper, a deterministic mathematical model to investigate the transmission dynamics of malaria in Ghana is formulated taking into account human and mosquito populations. The model consists of seven non-linear differential equations which describe the dynamics of malaria with 4 variables for humans and 3 variables for mosquitoes. The state vector for the model is  $(S_h, E_h, I_h, R, S_m, E_m, I_m)$  where  $S_h, E_h, I_h, R, S_m, E_m$  and  $I_m$  respectively represent populations of susceptible humans, exposed humans, infectious humans, recovered humans, susceptible mosquitoes, exposed mosquitoes and infectious mosquitoes. Stability analysis of the model is performed and we make use of the next generation method to derive the basic reproduction number  $R_0$ . A mathematical analysis of the dynamic behaviour indicates that the estimated model has a unique endemic equilibrium point and malaria will persist in Ghana. The basic reproduction number for Ghana is found to be  $R_0 = 0.8939$ . Further, both the disease-free and endemic equilibria are locally asymptotically stable. Numerical simulations indicate that reducing current biting rate of female Anopheles mosquitoes by 1/16 could assist Ghana to achieve malaria free status by the year 2037. If, in addition, the number of days it takes to recover from malaria infection were reduced to three 3 days malaria free status could be achieved by the year 2029.

**Keywords:** deterministic mathematical model, basic reproduction number, stability analysis and female Anopheles mosquitoes

## 1. Introduction

Malaria is a life-threatening disease caused by a protozoan parasite called Plasmodium, which lives part of its life in humans and part in Anopheles mosquitoes. The disease is endemic in tropical and subtropical regions, including Africa, Asia, Latin America, the Middle East and some parts of Europe. According to the Anti-Malaria Drug Policy for Ghana document in 2009, Malaria remains hyper endemic in Ghana and is the single most important cause of mortality and morbidity especially among children under five years, pregnant women and the poor (UNICEF - At a glance: Ghana, [www.unicef.org](http://www.unicef.org)).

Chitnis (2005) proposed a model similar to the malaria model in this paper. The main differences of our model, from that of Chitnis (2005) is that we have excluded the infection of female Anopheles mosquito by recovered humans, because we assume that these humans do not have sufficient plasmodium parasites in their bodies to transmit the infection to mosquitoes. Also, in our model, the infectious humans recover with clinical treatment and the death of the female Anopheles mosquito is caused by natural death rate and insecticides. Further this paper fills in an information gap pertaining to the dynamics of transmission of malaria in Ghana. To the best of our knowledge enough work has not been done in this area to assist health workers and policy makers to make informed decisions. In this paper we use clinical malaria data from Ghana Health Service at WHO Website to develop a mathematical model to investigate and understand the disease transmission dynamics in Ghana taking both host and vector populations into account. Our model will be non-linear ordinary differential equations. Stability analysis and numerical simulations are performed. The simulations are conducted using MATLAB's ode45. Some of the assumptions in this paper on mathematical modeling of malaria are based on studies by Chitnis (2005), Chitnis et al. (2006), Danso-Addo (2009) and Mwamtobe (2010).

## 2. Method

The model is based on important intervention strategies currently relevant in Ghana such as clinical treatment

and the mortality of the female Anopheles mosquito which is caused not only by natural death rate, but by indoor residual spraying (IRS) and insecticide treated bed nets (ITNs). We divide the human population into 4 classes: the Susceptible, denoted,  $S_h$ , representing the fraction of host population that is susceptible to infection; the Exposed, denoted  $E_h$ , being the fraction of population who are infected but not infectious in that they cannot transmit the infection. There is also the Infectious class, denoted  $I_h$ , corresponding to persons who have the malaria infection and can transmit it to persons in a susceptible class. Finally, the recovered, denoted by  $R$ , being people who recover from the infection through clinical treatment and are endowed with temporary immunity. These latter humans can not transmit the infection to mosquitoes because we assume that they have no plasmodium parasites in their bodies. People enter the susceptible class, either through birth or through immigration (assume to be at a constant rate). There is a finite probability of movement to the exposed class when a susceptible person is bitten by an infectious anopheles mosquito. The parasite is then passed on to humans in the form of sporozoites. The clinical onset of the disease is characterized by the parasite entering the blood stream as merozoites. Later there is a movement of infectious humans to recovery class where they acquire some immunity to malaria. After some further time, this immunity is lost and they revert to the susceptible class. Humans leave the population through natural death and those in the infectious class have additional disease-induced death rate.

We do not include the immigration of infectious humans because we assume that most people who are sick will not travel. The movement of Exposed humans are excluded because, given the short time of the exposed stage, the number of exposed people is small. We do make a simplifying assumption that there is no immigration of recovered humans.

The female Anopheles mosquito population is divided into 3 classes: Susceptible  $S_m$ , Exposed  $E_m$  and Infectious  $I_m$ . Anopheles male mosquitoes are not included in the model because only female mosquitoes bite humans for blood meals. Female mosquitoes enter the susceptible class through birth. The parasite (in the form of gametocytes) enters the mosquito, with some probability, when the mosquito bites an infectious human and the mosquito moves from the Susceptible to the Exposed class. After some period of time, dependent on the ambient temperature and humidity, the parasite develops into sporozoites and enters the mosquito's salivary glands; and the mosquito moves from the exposed class to the infectious class. The mosquito remains infectious for life. Mosquitoes leave the population through natural death rate and death caused by insecticides. We assume that longevity of the female Anopheles mosquitoes is unaffected by the parasite infection and they do not die from the infection. There is no super infection of the disease. Mosquitoes cannot survive without human host as they need human blood to feed their developing eggs.

The parameters in Table 1 and the state variables in Table 2 are used in Figure 1 to formulate the malaria model.

Table 1. Mode parameters and their interpretations for the malaria model (1)

| Parameter     | Description  |
|---------------|--|
| $\Psi$        | Recruitment rate of humans   |
| $\rho$        | Recruitment rate of mosquitoes   |
| $\alpha_h$    | Force of infection of humans from susceptible state to exposed state   |
| $\alpha_m$    | Force of infection of mosquitoes from susceptible state to exposed state   |
| $\beta_h$     | Rate of progression of humans from the exposed state to the infectious state   |
| $\beta_m$     | Rate of progression of mosquitoes from the exposed state to the infectious state                                     |
| $\tau$        | Clinical treatment-recovery rate of humans from the infectious state to the recovered state                          |
| $\mu$         | Natural death rate for humans  |
| $\omega$      | Death of mosquitoes caused by natural death rate and insecticides  |
| $\pi$         | Disease-induced death rate for humans  |
| $\varphi$     | Rate of loss immunity for humans   |
| $\theta_{mh}$ | Probability of transmission of infection from an infectious mosquito to a susceptible human provided there is a bite |
| $\theta_{hm}$ | Probability of transmission of infection from an infectious human to a susceptible mosquito provided there is a bite |
| $\phi$        | Biting rate of mosquitoes  |

Table 2. The state variables for the malaria model (1)

| Parameter | Description                                     |
|-----------|---|
| $S_h(t)$  | Number of susceptible humans at time $t$        |
| $E_h(t)$  | Number of exposed humans at time $t$            |
| $I_h(t)$  | Number of infectious humans at time $t$         |
| $R(t)$    | Number of recovered (immune) humans at time $t$ |
| $S_m(t)$  | Number of susceptible mosquitoes at time $t$    |
| $E_m(t)$  | Number of exposed mosquitoes at time $t$        |
| $I_m(t)$  | Number of infectious mosquitoes at time $t$     |
| $N_h(t)$  | Total human population at time $t$              |
| $N_m(t)$  | Total mosquito population at time $t$           |

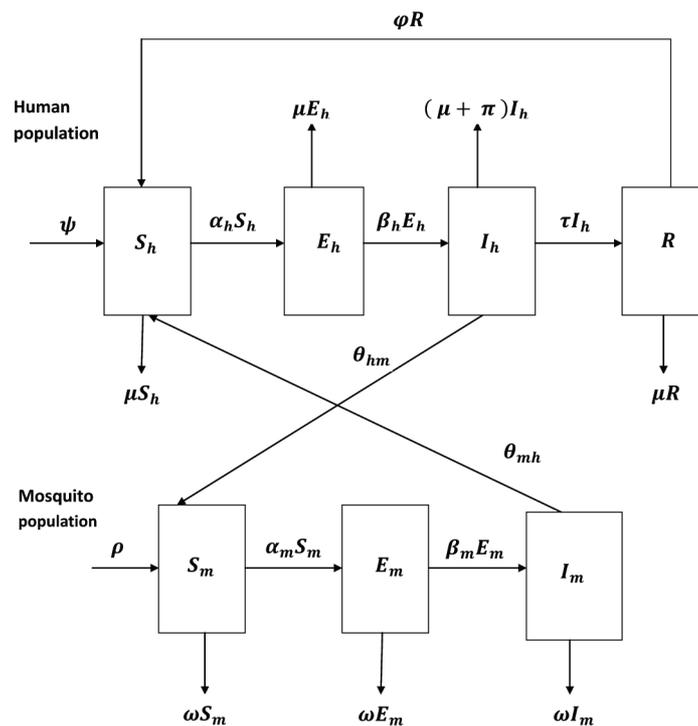


Figure 1. Flowchart of malaria incidence in human and mosquito populations

2.1 Equations of the Malaria Model

Applying the assumptions, definitions of state variables and parameters above, the system of non-linear differential equations which describe the dynamics of malaria are formulated below:

$$\left. \begin{aligned}
 \frac{dS_h}{dt} &= \psi + \varphi R - \alpha_h S_h - \mu S_h \\
 \frac{dE_h}{dt} &= \alpha_h S_h - \beta_h E_h - \mu E_h \\
 \frac{dI_h}{dt} &= \beta_h E_h - \tau I_h - (\mu + \pi) I_h \\
 \frac{dR}{dt} &= \tau I_h - \varphi R - \mu R \\
 \frac{dS_m}{dt} &= \rho - \alpha_m S_m - \omega S_m \\
 \frac{dE_m}{dt} &= \alpha_m S_m - \beta_m E_m - \omega E_m \\
 \frac{dI_m}{dt} &= \beta_m E_m - \omega I_m
 \end{aligned} \right\} \tag{1}$$

with initial conditions

$$S_h(0) = S_{h0}, E_h(0) = E_{h0}, I_h(0) = I_{h0}, R(0) = R_0, S_m(0) = S_{m0}, E_m(0) = E_{m0}, I_m(0) = I_{m0},$$

where  $\alpha_h = \frac{\theta_{mh}\phi I_m}{N_h}$  and  $\alpha_m = \frac{\theta_{hm}\phi I_h}{N_h}$ . For the purpose of this paper, we refer to Equation (1) as malaria model. The total population sizes are  $N_h = S_h + E_h + I_h + R$  and  $N_m = S_m + E_m + I_m$  with their differential equations

$$\frac{dN_h}{dt} = \psi - \mu N_h - \pi I_h \tag{2}$$

and

$$\frac{dN_m}{dt} = \rho - \omega N_m$$

### 3. Analysis of the Model

#### 3.1 Invariant Region

The invariant region can be obtained by the following theorem.

**Theorem 1** *The solutions set to the malaria model are feasible for all  $t > 0$  if they enter the invariant region  $\Omega = \Omega_h \times \Omega_m$ .*

*Proof.* Let  $\Omega = (S_h, E_h, I_h, R, S_m, E_m, I_m) \in R_+^7$  be any solution of the malaria model with non-negative initial conditions. In absence of the malaria, that is  $I_h = 0$ , Equation (2) becomes

$$\frac{dN_h}{dt} + \mu N_h \leq \psi$$

and solving for  $N_h$  we have,  $N_h \leq \frac{\psi}{\mu} + Ce^{-\mu t}$ . Using the initial conditions at  $t = 0$ ,  $N_h(0) = N_{h0}$ , we have

$$N_h \leq \frac{\psi}{\mu} + \left(N_{h0} - \frac{\psi}{\mu}\right)e^{-\mu t}.$$

Applying the theorem of differential inequality (Birkhoff & Rota, 1982), we obtain,

$$0 \leq N_h \leq \frac{\psi}{\mu} \text{ as } t \rightarrow \infty.$$

Therefore, as  $t \rightarrow \infty$ , the human population  $N_h$  approaches  $K = \frac{\psi}{\mu}$ , the parameter  $K$  is usually called the carrying capacity (Namaweje, 2011). Hence all feasible solution set of the human population of the malaria model enters the region  $\Omega_h = \{(S_h, E_h, I_h, R) \in R_+^4 : S_h > 0, E_h \geq 0, I_h \geq 0, R \geq 0, N_h \leq \frac{\psi}{\mu}\}$ .

Similarly, the feasible solutions set of the mosquito population enters the region  $\Omega_m = \{(S_m, E_m, I_m) \in R_+^3 : S_m > 0, E_m \geq 0, I_m \geq 0, N_m \leq \frac{\rho}{\omega}\}$ . Therefore, the feasible solutions set for malaria model given by  $\Omega = \Omega_h \times \Omega_m$  is positive-invariant and hence it is biologically meaningful and mathematically well-posed in the domain  $\Omega$ .

#### 3.2 Positivity of Solutions

**Lemma 1** *Let the initial data be  $\{(S_h(0), S_m(0)) > 0, (E_h(0), I_h(0), R(0), E_m(0), I_m(0)) \geq 0\} \in \Omega$ . Then the solution set  $(S_h(t), E_h(t), I_h(t), R(t), S_m(t), E_m(t), I_m(t))$  of the system malaria model is positive  $\forall t > 0$ .*

*Proof.* From the first equation in the model (1), we have

$$\frac{dS_h}{dt} = \psi + \varphi R - \alpha_h S_h - \mu S_h \geq -\alpha_h S_h - \mu S_h$$

therefore,

$$S_h(t) \geq S_h(0)e^{-(\alpha_h + \mu)t} \geq 0$$

From the second equation of model malaria model,

$$\frac{dE_h}{dt} = \alpha_h S_h - \beta_h E_h - \mu E_h \geq -(\beta_h + \mu)E_h$$

hence,

$$E_h(t) \geq E_h(0)e^{-(\beta_h + \mu)t} \geq 0.$$

Similarly, it can be shown that the remaining equations in system malaria model are also positive  $\forall t > 0$ , because  $e^\eta > 0 \eta \in R$ .

### 3.3 Disease-Free Equilibrium

Disease-free equilibrium points (DFE) are steady state solutions where there is no malaria in the human population or Plasmodium parasite in the mosquito population. Let define the “diseased” classes as the human or mosquito populations that are either exposed or infectious; that is,  $E_h, I_h, E_m$  and  $I_m$ . In absence of the disease, this implies that ( $E_h = I_h = E_m = I_m = 0$ ) and when the right-hand side of a nonlinear system (1) is set to zero, we have,

$$\left. \begin{aligned} S_h^{e_0} &= \frac{\psi}{\mu} \\ S_m^{e_0} &= \frac{\rho}{\omega} \end{aligned} \right\}$$

Therefore, the disease-free equilibrium point of the malaria model (6) is given by

$$E_0 = (S_h^{e_0}, E_h^{e_0}, I_h^{e_0}, R^{e_0}, S_m^{e_0}, E_m^{e_0}, I_m^{e_0}) = \left( \frac{\psi}{\mu}, 0, 0, 0, \frac{\rho}{\omega}, 0, 0 \right)$$

which represents the state in which there is no infection(in the absence of malaria) in the society.

### 3.4 Basic Reproduction Number

We use the next generation operator approach as described by Diekmann et al. (1990) to define the basic reproduction number,  $R_0$ , as the number of secondary infections that one infectious individual would create over the duration of the infectious period, provided that everyone else is susceptible. When  $R_0 < 1$ , each infected individual produces on average less than one new infected individual, so we would expect the disease to die out. On the other hand, if  $R_0 > 1$ , each individual produces more than one new infected individual, so we would expect the disease to spread in the population. This means that the threshold quantity for eradicating the disease is to reduce the value of  $R_0$  to be less than one. We determine  $R_0$  using the next generation operator approach. The associated next generation matrices are

$$F = \begin{bmatrix} 0 & 0 & 0 & \theta_{mh}\phi \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\theta_{hm}\phi\mu\rho}{\psi\omega} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} (\beta_h + \mu) & 0 & 0 & 0 \\ -\beta_h & (\tau + \mu + \pi) & 0 & 0 \\ 0 & 0 & (\beta_h + \omega) & 0 \\ 0 & 0 & -\beta_h & \omega \end{bmatrix},$$

and

$$FV^{-1} = \begin{bmatrix} 0 & 0 & \frac{\beta_m\theta_{mh}\phi}{\omega(\beta_m+\omega)} & \frac{\theta_{mh}\phi}{\omega} \\ -\beta_h & (\tau + \mu + \pi) & 0 & 0 \\ \frac{\beta_h\theta_{hm}\phi\mu\rho}{\psi\omega(\beta_h + \mu)(\tau+\mu+\pi)} & \frac{\theta_{hm}\phi\mu\rho}{\psi\omega(\tau+\mu+\pi)} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

Let  $p = \frac{\beta_m\theta_{mh}\phi}{\omega(\beta_m+\omega)}$ ,  $q = \frac{\theta_{mh}\phi}{\omega}$ ,  $k = \frac{\beta_h\theta_{hm}\phi\mu\rho}{\psi\omega(\beta_h + \mu)(\tau+\mu+\pi)}$  and  $l = \frac{\theta_{hm}\phi\mu\rho}{\psi\omega(\tau + \mu + \pi)}$ . We can now calculate the eigenvalues  $\lambda$  to determine the basic reproduction number  $R_0$  by taking the spectral radius (dominant eigenvalue) of the matrix  $FV^{-1}$ . We have  $\lambda^2(\lambda^2 - kp) = 0 \Rightarrow \lambda = 0$  or  $\lambda = \pm \sqrt{kp}$ . From the four eigenvalues, the dominant eigenvalue of the matrix  $FV^{-1}$  is  $\lambda = \sqrt{kp}$ . Therefore the basic reproduction number  $R_0 = \sqrt{kp}$ . Hence

$$R_0 = \sqrt{\frac{\phi^2\rho\beta_h\beta_m\theta_{hm}\theta_{mh}\mu}{\psi\omega(\beta_h + \mu)(\tau + \mu + \pi)(\beta_m + \omega)\omega}} \tag{3}$$

The threshold parameter  $R_0$  can be defined as square roots of the product of number of humans one mosquito infects during its infectious lifetime  $R_{0h}$  and number of mosquitoes one human infects during the duration of the infectious period  $R_{0m}$ , provided all humans and mosquitoes are susceptible. Therefore,

$$R_0 = \sqrt{R_{0h} \times R_{0m}} = \sqrt{\frac{\beta_h\theta_{mh}\phi\mu}{\psi(\beta_h + \mu)(\tau + \mu + \pi)} \times \frac{\beta_m\theta_{hm}\phi\rho}{(\beta_m + \omega)\omega^2}} \tag{4}$$

hence

$$R_{0h} = \frac{\beta_h \theta_{mh} \phi \mu}{\psi (\beta_h + \mu) (\tau + \mu + \pi)}$$

and

$$R_{0m} = \frac{\beta_m \theta_{hm} \phi \rho}{(\beta_m + \omega) \omega^2}$$

The basic reproduction number can be used to determine the local stability of the disease free equilibrium point.

### 3.5 Local Stability of Disease-Free Equilibrium

The local stability of the disease-free equilibrium can be analyzed using the Jacobian matrix of the malaria model at the disease free equilibrium point. Referring to the results of Van den Driessche and Watmough (2002), the following theorem holds.

**Theorem 2** *The disease free equilibrium point for the malaria model is locally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ .*

*Proof.* The Jacobian matrix  $J$  of the malaria model (1) with  $S_h = N_h - (E_h + I_h + R)$  and  $S_m = N_m - (E_m + I_m)$  at the disease-free equilibrium point is given by

$$J = \begin{bmatrix} -(\beta_h + \mu) & 0 & 0 & 0 & \theta_{mh} \phi \\ \beta_h & -(\tau + \mu + \pi) & 0 & 0 & 0 \\ 0 & \tau & -(\varphi + \mu) & 0 & 0 \\ 0 & \frac{\theta_{hm} \phi \mu \rho}{\psi \omega} & 0 & -(\beta_m + \omega) & 0 \\ 0 & 0 & 0 & \beta_m & -\omega \end{bmatrix}. \tag{5}$$

The eigenvalues of the Jacobian matrix are the solutions of the characteristic equation  $|J - \lambda I| = 0$ . Set  $A_1 = \omega$ ,  $A_2 = (\beta_m + \omega)$ ,  $A_3 = (\tau + \mu + \pi)$ ,  $A_4 = (\beta_h + \mu)$  and  $K^* = \frac{\phi^2 \rho \theta_{hm} \theta_{mh} \beta_h \beta_m \mu}{\psi \omega}$ . Then

$$\lambda^4 + B_1 \lambda^3 + B_2 \lambda^2 + B_3 \lambda + B_4 = 0, \tag{6}$$

where

$$\left. \begin{aligned} B_1 &= A_4 + A_3 + A_2 + A_1 \\ B_2 &= A_4(A_3 + A_2 + A_1) + A_3(A_2 + A_1) + A_2 A_1 \\ B_3 &= A_4 A_3 A_2 + A_4 A_3 A_1 + A_4 A_2 A_1 + A_3 A_2 A_1 \\ B_4 &= A_4 A_3 A_2 A_1 - K \end{aligned} \right\}.$$

Using the Routh-Hurwitz Criteria on (6), we can prove that all roots of the polynomial (6) have negative real parts. For the characteristic polynomial in (6), when  $n = 4$ , the Routh-Hurwitz criteria are  $B_1 > 0, B_2 > 0, B_3 > 0, B_4 > 0$  the determinants of Hurwitz matrices are positive. Hence all the eigenvalues of the jacobian (5) have negative real part and  $R_0 < 1$ . Therefore disease-free equilibrium point is stable.

Conversrly, if  $R_0 > 1$  it implies that  $B_4 < 0$  and since the remaining coefficients ( $B_1, B_2$  and  $B_3$ ) of the polynomial (6) are positive, then all the roots of this polynomial cannot have negative real parts. Therefore, the disease-free equilibrium point is unstable.

### 3.6 The Endemic Equilibrium Point

Endemic equilibrium points are steady state solutions where the disease persists in the population (all state variables are positive). That is, malaria infection will persists in the population and the endemic equilibrium point ( $EEP$ ) of the model is given by  $EEP = (S_h^{e_1}, E_h^{e_1}, I_h^{e_1}, R^{e_1}, S_m^{e_1}, E_m^{e_1}, I_m^{e_1}) > 0$ . To derive the  $EEP$ , we have to solve

$$\left. \begin{aligned} \psi + \varphi R - \frac{\theta_{mh} \phi I_m S_h}{N_h} - \mu S_h &= 0 \\ \frac{\theta_{mh} \phi I_m S_h}{N_h} - (\beta_h + \mu) E_h &= 0 \\ \beta_h E_h - (\tau + \mu + \pi) I_h &= 0 \\ \tau I_h - (\varphi + \mu) R &= 0 \\ \rho - \frac{\theta_{hm} \phi I_h S_m}{N_h} - \omega S_m &= 0 \\ \frac{\theta_{hm} \phi I_h S_m}{N_h} - (\beta_m + \omega) E_m &= 0 \\ \beta_m E_m - \omega I_m &= 0 \end{aligned} \right\}.$$

After some algebraic manipulation, we get

$$\begin{aligned}
 A[I_h^{e_1}]^2 + BI_h^{e_1} + C &= 0 \\
 A &= R_0^2 L \omega \phi \tau - \phi^2 \mu \theta_{hm} (\varphi + \mu) (M + L) \\
 B &= R_0^2 \mu \omega^2 N_h^2 \varphi \tau - \omega \phi (\varphi + \mu) [L(\psi R_0^2 - \psi - \mu N_h) - M\psi] \\
 C &= \mu \omega^2 N_h^2 \psi (\varphi + \mu) [R_0^2 - 1],
 \end{aligned}$$

where  $L = \mu N_h \theta_{hm}$  and  $M = \omega \theta_{mh} R_{om}$ . Therefore,

$$\begin{aligned}
 I_h^{e_1} &= \frac{-B + \sqrt{B^2 - 4AC}}{2A} = \Phi \\
 S_h^{e_1} &= \frac{\mu \theta_{hm} \phi \Phi + \omega \psi}{R_0^2 \omega \mu}, \quad E_h^{e_1} = \frac{\tau + \mu + \pi}{\beta_h} \Phi, \quad R^{e_1} = \frac{\tau}{\varphi + \mu} \Phi, \\
 S_m^{e_1} &= \frac{N_h \rho}{\theta_{hm} \phi \Phi + \omega N_h}, \quad E_m^{e_1} = \frac{R_{0m} \omega^2 \Phi}{\beta_m (\theta_{hm} \phi \Phi + \omega N_h)}, \quad I_m^{e_1} = \frac{R_{0m} \omega \Phi}{(\theta_{hm} \phi \Phi + \omega N_h)}
 \end{aligned}$$

We now consider the possibility of multiple endemic equilibria for equation (6). It may also indicate three distinct situations which we have to consider depending on the signs of  $B$  and  $C$  since  $A$  is always positive. The  $C$  is negative if  $R_0 < 1$  and positive if  $R_0 > 1$ . Hence the three situations will lead to the following theorem.

**Theorem 3** *The malaria model (1) has,*

- 1) *Precisely one unique endemic equilibrium if  $C < 0 \iff R_0 < 1$  .*
- 2) *Precisely one unique endemic equilibrium if  $B < 0$  and  $C = 0$  or  $B^2 - 4AC > 0$  .*
- 3) *Precisely two endemic equilibria if  $C > 0$  ,  $B < 0$  and*
- 4) *No endemic otherwise.*

### 3.7 Local Stability of the Endemic Equilibrium

The stability of the endemic equilibrium of the malaria model can be analysed using the Centre Manifold Theory described by Castillo-Chavez and Song (2004).

To apply this theorem we make the following change of variables in the malaria model.

Let  $x_1 = S_h$ ,  $x_2 = E_h$ ,  $x_3 = I_h$ ,  $x_4 = R$ ,  $x_5 = S_m$ ,  $x_6 = E_m$  and  $x_7 = I_m$ .

The malaria model is written in the form,

$$\frac{dX_i}{dt} = H(X_i)$$

where  $X_i = (x_1, x_2, \dots, x_7)^T$  and  $H = (h_1, h_2, \dots, h_7)^T$  are transposed matrices. The malaria model becomes

$$\begin{aligned}
 \frac{dx_1}{dt} &= \psi + \varphi x_4 - \frac{\Psi^* \phi \mu x_7 x_1}{\psi} - \mu x_1 = h_1 \\
 \frac{dx_2}{dt} &= \frac{\Psi^* \phi \mu x_7 x_1}{\psi} - (\beta_h + \mu) x_2 = h_2 \\
 \frac{dx_3}{dt} &= \beta_h x_2 - (\tau + \mu + \pi) x_3 = h_3 \\
 \frac{dx_4}{dt} &= \tau x_3 - (\varphi + \mu) x_4 = h_4 \\
 \frac{dx_5}{dt} &= \rho - \frac{\theta_{hm} \phi \mu x_3 x_5}{\psi} - \omega x_5 = h_5 \\
 \frac{dx_6}{dt} &= \frac{\theta_{hm} \phi \mu x_3 x_5}{\psi} - (\beta_m + \omega) x_6 = h_6 \\
 \frac{dx_7}{dt} &= \beta_m x_6 - \omega x_7 = h_7
 \end{aligned} \tag{7}$$

where  $N_h = x_1 + x_2 + x_3 + x_4$  and  $N_m = x_5 + x_6 + x_7$  with  $\Psi^* = \theta_{mh}$ .

Let  $\Psi^*$  be the bifurcation parameter, the system (7) is linearized at disease free equilibrium point when  $\Psi = \Psi^*$  with  $R_0 = 1$ . Thus  $\Psi^*$  can be solved from (3) when  $R_0 = 1$  as

$$\Psi^* = \frac{\psi(\beta_h + \mu)(\tau + \mu + \pi)(\beta_m + \omega)\omega^2}{\phi^2\rho\beta_h\beta_m\theta_{hm}\mu}$$

Then zero is a simple eigenvalue of the following Jacobian matrix,  $J_{bif}$  with the application of the bifurcation parameters.

$$\begin{bmatrix} -\mu & 0 & 0 & \varphi & 0 & 0 & -\Psi\phi \\ 0 & F_h & 0 & 0 & 0 & 0 & \Psi\phi \\ 0 & \beta_h & C & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau & -(\varphi + \mu) & 0 & 0 & 0 \\ 0 & 0 & D & 0 & -\omega & 0 & 0 \\ 0 & 0 & E & 0 & 0 & F_m & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_m & -\omega \end{bmatrix}$$

where  $C = -(\tau + \mu + \pi)$ ,  $D = -\frac{\theta_{hm}\phi\mu\rho}{\psi\omega}$ ,  $E = \frac{\theta_{hm}\phi\mu\rho}{\psi\omega}$ ,  $F_h = -(\beta_h + \mu)$  and  $F_m = -(\beta_m + \omega)$ .

A right eigenvector associated with the eigenvalue zero is  $w = (w_1, w_2, \dots, w_7)$ . We have the following right eigenvector

$$w_1 = \frac{\varphi w_4 - \Psi\phi w_7}{\mu}, \quad w_2 = \frac{\Psi\phi w_7}{\beta_h + \mu}, \quad w_3 = \frac{\beta_h w_2}{\tau + \mu + \pi}, \quad w_4 = \frac{\tau w_3}{\varphi + \mu}, \quad w_5 = -\frac{\theta_{hm}\phi\mu\rho w_3}{\psi\omega^2},$$

$$w_6 = \frac{\theta_{hm}\phi\mu\rho w_3}{\psi\omega(\beta_m + \omega)}, \quad w_7 = w_7 > 0$$

and the left eigenvector satisfying  $vw = 1$  is  $v = (v_1, v_2, \dots, v_7)$ . The left eigenvector is given as follows:

$$v_1 = 0, \quad v_2 = v_2 > 0, \quad v_3 = \frac{(\beta_h + \mu)v_2}{\beta_h}, \quad v_4 = 0, \quad v_5 = 0, \quad v_6 = \frac{\beta_m v_7}{\beta_m + \omega}, \quad v_7 = \frac{\Psi\phi v_2}{\omega}.$$

Therefore

$$a = v_2 w_2 w_7 \frac{\partial^2 h_2}{\partial x_2 \partial x_7} + v_2 w_3 w_7 \frac{\partial^2 h_2}{\partial x_3 \partial x_7} + v_6 w_6 w_3 \frac{\partial^2 h_6}{\partial x_6 \partial x_3} + v_6 w_7 w_3 \frac{\partial^2 h_6}{\partial x_7 \partial x_3}$$

$$= v_2 w_2 w_7 \left(-\frac{\Psi\phi\mu}{\psi}\right) + v_2 w_3 w_7 \left(-\frac{\Psi\phi\mu}{\psi}\right) + v_6 w_6 w_3 \left(-\frac{\theta_{hm}\phi\mu}{\psi}\right) + v_6 w_7 w_3 \left(-\frac{\theta_{hm}\phi\mu}{\psi}\right)$$

$$= -\frac{\phi\mu}{\psi} \left[ v_2 w_7^2 \Psi^2 \phi \left( \frac{\tau + \mu + \pi + \beta_h}{(\beta_h + \mu)(\tau + \mu + \pi)} \right) + v_6 w_3 \theta_{hm} \left( \frac{\theta_{hm}\phi^2\mu\rho\beta_h\Psi}{\psi\omega(\beta_m + \omega)(\tau + \mu + \pi)(\beta_h + \mu)} + 1 \right) \right] < 0$$

Similarly

$$b = v_2 w_7 \frac{\partial^2 h_2}{\partial x_7 \partial \Psi} = v_2 w_7 \phi > 0.$$

Hence  $a < 0$  and  $b > 0$ . Therefore the following theorem holds.

**Theorem 1** *The malaria model has a unique endemic equilibrium which is locally asymptotically stable when  $R_0 < 1$  and unstable when  $R_0 > 1$ .*

#### 4. Results and Discussion

The parameters in the malaria model were estimated using clinical malaria data and demographic statistics of Ghana. Those that were not available were obtained from literature published by researchers in malaria endemic countries which have similar environmental conditions compared to Ghana. According to the Ghana Living Standards Survey Report of the Fifth Round (GLSS 5), 2008, the estimated number of households in Ghana is 5.5 million. Conservatively, it is assumed that there are 10 female Anopheles mosquitoes per household in Ghana. The female Anopheles mosquito population is then approximately given by:  $5,500,000 \times 10 = 55,000,000$  mosquitoes.

The Table 3 below shows the estimated parameters and their sources for the model (1). The rates are given per day.

Table 3. Mode parameters and their interpretations for the malaria model (1)

| Parameter     | Value        | Source                                 |
|---------------|--------------|--|
| $\Psi$        | 0.00005079   | (2010 est.) by 2011 CIA World Factbook |
| $\rho$        | 0.071        | Niger, 2008                            |
| $\beta_h$     | 1/14         | Malaria.com, 2011                      |
| $\beta_m$     | 1/11         | Chitnis, 2005                          |
| $\tau$        | 1/7          | Tumwiine et al., 2004                  |
| $\mu$         | 1/(64365.25) | At a glance: Ghana, UNICEF, 2012       |
| $\omega$      | 1/25         | Estimated                              |
| $\pi$         | 0.0000027    | World Malaria Report 2010 for Ghana    |
| $\varphi$     | 1/91.3125    | Estimated                              |
| $\theta_{mh}$ | 0.42         | Estimated                              |
| $\theta_{hm}$ | 0.0655       | Estimated                              |
| $\phi$        | 0.4          | Chitnis, 2005                          |

After substituting the estimated parameter values in Table 3 into the malaria model, we obtain the basic reproduction number from (3) as  $R_0 = 0.8939$ . Since  $R_0 = 0.8939 < 1$ , hence malaria disease can be eliminated or eradicated in the susceptible population in Ghana.

4.1 Local Stability of the Disease-free Equilibrium

Using (6), we have  $\lambda^4 + 0.3853\lambda^3 + 0.05209\lambda^2 + 0.002868\lambda + 0.00001075 = 0$  Since the coefficients of the polynomial are positive, it follows by the Routh-Hurwitz stability criteria that, the disease-free equilibrium point is asymptotically stable. This means that malaria free society can be achieved.

4.2 The Endemic Equilibrium Point

The quadratic equation for calculating the value of  $I_h^{e1}$  is given below:

$$0.0072[I_h^{e1}]^2 + 34705I_h^{e1} - 3.1111 = 0 \tag{8}$$

Since  $C = -3.1111 < 0$  in (8) and also  $R_0 < 1$ , the estimated malaria model for Ghana has one unique endemic equilibrium point.

The bifurcation parameter is given by

$$\Psi^* = \frac{0.00005079 (0.07147) (0.1429) (0.13091) (0.04)^2}{(0.4)^2 (0.071) (1/14) (1/11) (0.42) (0.00004278)} = 0.0820$$

The estimate of  $a$  is given by

$$a = -\frac{(0.4) (0.00004278)}{(0.00005079)} [0.05644 + 0.07901] = -0.33692 (0.13545) = -0.04564 < 0$$

Similarly the parameter estimate is given by

$$b = v_2w_7 \frac{\partial^2 h_2}{\partial x_7 \partial \Psi} = v_2w_7\phi = (1)(1)(0.4) > 0$$

Since  $a < 0$  and  $b > 0$ , by the Centre Manifold Theory described by Castillo-Chavez and Song (2004) the endemic equilibrium point is locally asymptotically stable. This means malaria will persist in Ghana.

### 5. Numerical Simulations

A numerical simulation of the estimated malaria model is conducted to explore scenarios of the dynamics of the disease in the human population. The time-axes in all phase portraits below start from the year 2000. We mainly consider the effects of varying key parameters responsible for controlling malaria

- 1) Reducing the biting rate of mosquitoes.
- 2) The treatment rate of infectious humans.
- 3) Combining the reduction in the biting rate of mosquitoes and the increase in the treatment rate of infectious humans.

The biting rate of mosquitoes can be reduced by using the Insecticide-treated bed nets (ITN) and Indoor residual spraying (IRS). The values of the biting rate of mosquitoes, transmission rate of infection from an infectious mosquito to a susceptible human, rate of loss of immunity for humans and the mosquito population are reduced by 1/16, while the values of the other parameters are maintained. This is illustrated in the Figure 2(a).

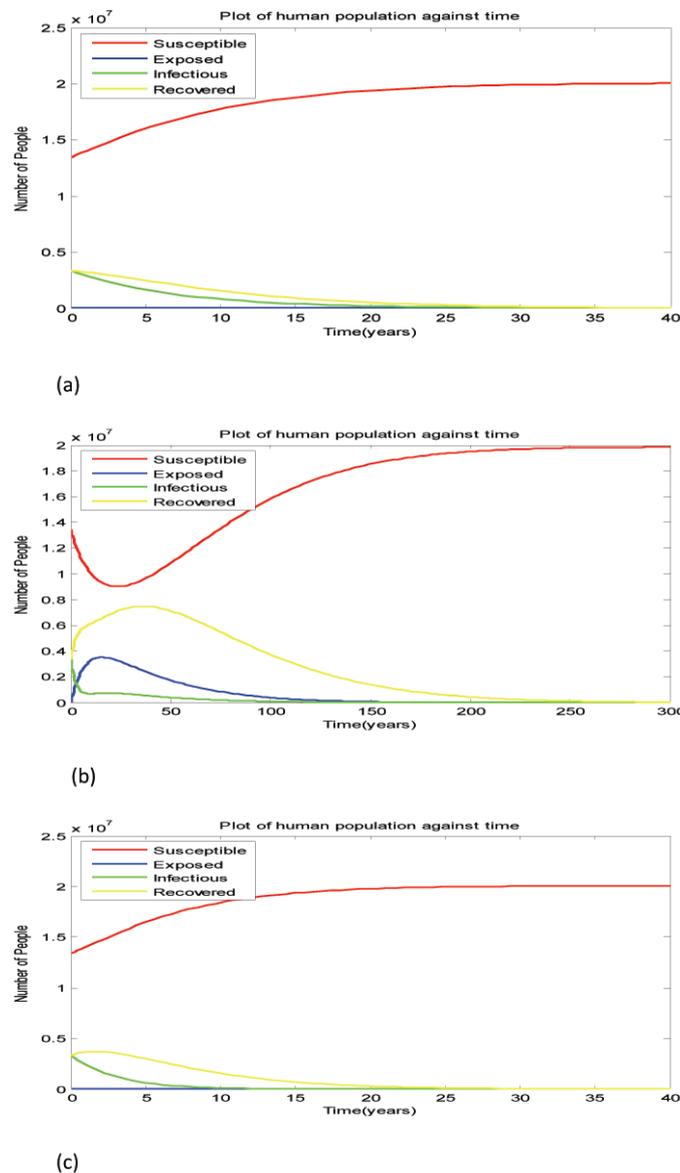


Figure 2. Simulation plots

Increasing the treatment rate will reduce the transmission rate of infection from an infectious human to a susceptible

mosquito and the rate of loss of immunity for humans. Increasing the treatment rate to  $1/3$  and reducing the transmission rate  $\theta_{hm}$  and rate of loss of immunity to 0.18 and  $1/39.1339$  respectively, give the phase portrait diagram below. From Figure 2(b), if the treatment rate is increased by  $1/3$ , Ghana will achieve malaria free status by the year 2285. Comparing the two interventions, we conclude that the most influential parameter in controlling the disease (malaria) is to reduce the mosquito biting rate.

We consider the effects of combining the two interventions in controlling malaria disease. The effects of combining the two interventions in controlling malaria disease are shown in Figure 2(c). When the two interventions are combined, Ghana could have malaria free status by 2029. So the intervention practices that involve both prevention and treatment controls yield a relatively better result.

## 6. Conclusion

In this paper a mathematical model is formulated from the host and vector populations in the transmission of malaria in Ghana. The Malaria model has a unique endemic equilibrium point which is locally asymptotically stable. Further, the model indicates that malaria disease can be eliminated or eradicated in the susceptible population and malaria free society can be achieved in Ghana by the year 2037 if the current mosquito biting rate is reduced by  $1/16$ . In Figure 2(c), when the two interventions are combined, Ghana will have malaria free status by 2029. Innovative strategies to minimize or reduce the biting rate need to be identified and implemented soon.

The Model assumes a varying host and vector populations. Further research into situations where by a deliberate injection of genetically modified mosquitoes into the mosquitoes to reduce the transmission rate needs to be investigated.

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