Journal of the Nigerian Association of Mathematical Physics
Volume 47 (*July, 2018 Issue*), pp39 –46

MATHEMATICAL MODEL FOR THE CONTROL OF MALARIA USING EDUCATION-BASED INTERVENTION

Achie I., Soladoye Sikiru O. and Shehu U.

Department of Mathematics and Statistics, Kaduna Polytechnic, Kaduna.

Abstract

In this study, we modified a mathematical model for the transmission dynamics of malaria by incorporating behavioural change via education as another control strategy against the spread of malaria. Analytical studies is carried out to determine the local stability of model and result indicates that the disease-free equilibrium of the system is locally asymptotically stable if $R_0 < 1$***; implying that behavioural change plays a significant role towards achieving a malaria-free society.***

Keywords**:** Reproduction number, behavioural change, Local stability

1.0 Introduction

Malaria is a life-threatening disease is caused by the single-celled genus plasmodium parasites and five parasite species have been identified to cause malaria in humans. These include *P. vivax, P. ovale, P. malariae, P. falciparum,* and *P. knowlesi*, *Plasmodium falciparum (P. falciparum)* causes most of the severe diseases and deaths and is most prevalent in Africa [1]. Malaria parasites are transmitted to humans through the bites of infected Anopheles mosquitoes, called "malaria vectors", which bite mainly between dusk and dawn. The main symptoms of malaria include fatigue, chill, headache, abdominal and back pain, diarrhoea, sometimes vomiting and fever. The parasite is responsible for the greatest number of deaths and clinical cases in the tropics especially among pregnant women and children below ages of 5. Severe malaria infection can lead to serious complications affecting brain, lungs, kidneys and other organs [2]. Global efforts to control and eliminate malaria have saved an estimated 3.3 million lives since 2000 [3]. However, the global incidence of malaria is increasing especially in Sub Saharan Africa. This is attributed to factors such as poverty, war and insurgency, and collapse of health care systems, poor environmental sanitation and management, prevailing political situation and inadequate funding by the government. Also, emerging drug and insecticide resistance threaten to reverse recent gains [1].

Mathematical models have been used to understand the epidemiology of infectious diseases such as malaria in a given population. Mathematical modelling of malaria began in 1911 with the Ross' model. MacDonald did a major extension on the Ross model [4]. Since then, various mathematical models of malaria transmission have been developed which take into account various possible scenarios in the spread of malaria. Several interventions have been recommended to curb the rising burden of the disease in endemic regions. These interventions form the pillar of the global campaign for effective malaria intervention, particularly in sub-Saharan Africa. A compartmental model where human population follow SEIRS pattern and mosquitoes follow SEI pattern in which the effect of environment on malaria transmission was considered and developed [5].

In a recent study, a SEIR model with sensitivity analysis to compare intervention strategies for malaria control for two representative areas of high and low transmission was derived [6]. Also derived was deterministic model to investigate the transmission dynamics of malaria in Ghana [7]. A mathematical model was used to study the effects of malaria preventive measures [8]. An observation was made that when interventions such as education are introduced in the fight against infectious diseases, trends improve in the population [9].

Therefore, the malaria model proposed in this study is an extension of a model, where the human population follows the susceptible-exposed-infectious-recovered (SEIR) pattern and the mosquito population follows susceptible-exposed-infectious (SEI) pattern [10]. We incorporate an additional compartment to represent the protected human population and a parameter to enable us to determine the effect of behavioural change as another control strategy against the spread of malaria.

The rest of this study is organized thus: the full description of the model is given in Section 2. Section 3 gives the existence of the disease free equilibrium, derivation of the reproduction number and the local stability of the disease free equilibrium. In section 4, numerical experiments of the model are performed and the conclusion is given in section 5.

2. Model description and formulation

Our malaria model consists of human and mosquito populations. The human population is divided into five classes; Susceptible humans, $S_h(t)$, Protected humans, $P_h(t)$, Exposed humans, $E_h(t)$, Infectious humans, $I_h(t)$ and Recovered humans, $R_h(t)$. Thus, the total population of humans is given by

$$N_h(t) = S_h(t) + P_h(t) + E_h(t) + I_h(t) + R_h(t)$$

Corresponding Author**:** Achie i., Email: achieismailu@gmail.com, Tel: +2348028566982

On the other hand, the mosquito population is divided into three classes; Susceptible mosquitoes, $S_m(t)$, Exposed mosquitoes, $E_m(t)$ and Infectious mosquitoes, $I_m(t)$. Hence, we have the total mosquito population as

$N_m(t) = S_m(t) + E_m(t) + I_m(t)$

Our model was based on the following assumptions

i. Apart from dying due to other causes, infectious humans die as a result of the disease.
ii. The infected mosquito remain infectious for life and die as due to the disease
iii. Recovered humans acquire immunity to the disease for some period of time and loses the immunity to become susceptible again
iv. Susceptible humans that sufficiently acquire or cultivate health-enhancing behaviours will prevent themselves from having contact with mosquitoes.

The state variables in Table 1 and parameters in Table 2 are used in Figure 1 to formulate the model for malaria control (2.1) – (2.8).

Table 1: State Variables of the Model

State variables	Description
$S_h(t)$	Number of human host susceptible to malaria infection at time t
$E_h(t)$	Number of human host exposed to malaria infection at time t
$I_h(t)$	Number of Infectious human host at time t
$R_h(t)$	Number of Recovered human host 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
$P_h(t)$	Number of protected humans at time t

Table 2: Parameters of the model

Parameter	Description
Λ_h	Recruitment rate of the susceptible humans
Λ_m	Recruitment rate of the susceptible mosquitoes
b	Biting rate of the mosquito
β_h	Probability that a bite by an infectious mosquito results in transmission of the disease to humans
β_m	Probability that a bite results in transmission of parasite to a susceptible mosquito
μ_h	Per capital death rate of humans
μ_m	Per capital death rate of humans
δ_h	Disease-induced death rate of humans
δ_m	Disease-induced death rate of mosquito
α_h	Per capital rate of progression of humans from exposed state to infectious state
α_m	Per capital rate of progression of mosquito from the exposed state to infectious
r	Per capital recovery rate for humans from the infectious state to the recovered state
ω	Per capital rate of loss of immunity in humans
v_h	Proportion of antibody produced by humans in response to the incidence of infection caused by mosquitoes
v_m	Proportion of antibody produced by mosquito in response to the incidence of infection caused be humans
e	Per capital rate of behavioural change

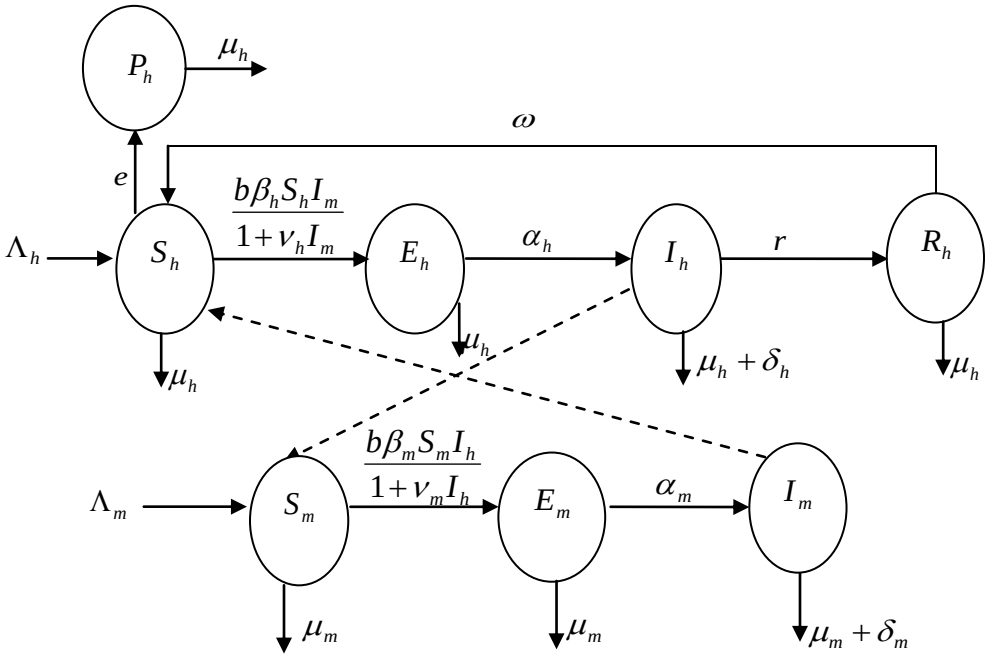

Figure 1: Epidemiological Flow Diagram for the Malaria Control Model

2.1 Equations of the Model

We obtain an 8-dimensional non-linear system of ordinary differential equations describing the transmission of malaria.

$$\frac{dS_h}{dt} = \Lambda_h - \frac{b\beta_h S_h(t) I_m(t)}{1+\nu_h I_m(t)} - \mu_h S_h(t) - eS_h(t) + \omega R_h(t) \quad (2.1)$$

$$\frac{dP_h}{dt} = eS_h(t) - \mu_h P_h(t) \quad (2.2)$$

$$\frac{dE_h}{dt} = \frac{b\beta_h S_h(t) I_m(t)}{1+\nu_h I_m(t)} - (\alpha_h + \mu_h) E_h(t) \quad (2.3)$$

$$\frac{dI_h}{dt} = \alpha_h E_h(t) - (r + \mu_h + \delta_h) I_h(t) \quad (2.4)$$

$$\frac{dR_h}{dt} = rI_h(t) - (\mu_h + \omega) R_h(t) \quad (2.5)$$

$$\frac{dS_m}{dt} = \Lambda_m - \frac{b\beta_m S_m(t) I_h(t)}{1+\nu_m I_h(t)} - \mu_m S_m(t) \quad (2.6)$$

$$\frac{dE_m}{dt} = \frac{b\beta_m S_m(t) I_m(t)}{1+\nu_m I_h(t)} - (\alpha_m + \mu_m) E_m(t) \quad (2.7)$$

$$\frac{dI_m}{dt} = \alpha_m E_m(t) - (\mu_m + \delta_m) I_m(t) \quad (2.8)$$

Together with the initial conditions:

$S_h(0) = S_{0h},\ P_h(0) = P_{oh},\ E_h(0) = E_{0h},\ I_h(0) = I_{0h},\ R_h(0) = R_{0h},\ S_m(0) = S_{0m},\ E_m(0) = E_{0m},\ I_m(0) = I_{0m}$

3. Analysis of the Model

3.1 Disease-free equilibrium (DFE)

We obtained the disease free equilibrium by setting the right hand side of the model equations (2.1)-(2.8) to zero, given that in the absence of the disease we have $E_h = 0, I_h = 0, E_m = 0, I_m = 0, R_h = 0$

Therefore, the disease-free equilibrium point is given by

$$E_0 = (S_h^*, P_h^*, E_h^*, I_h^*, R_h^*, S_m^*, E_m^*, I_m^*) = \left(\frac{\Lambda_h}{\mu_h + e}, \frac{e\Lambda_h}{\mu_h(\mu_h + e)}, 0, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0\right) \quad (2.9)$$

3.2 Basic Reproduction Number

The basic reproduction number denoted by, R_0, is an important parameter which is used to study the behaviour of epidemiological models. This is defined as the average number of secondary infectious infected by an infective individual during whose whole cause of disease in the case that all members of the population are susceptible. It is an important threshold parameter that determines whether or not, an infection will spread through a given population

We apply the next generation matrix technique to obtain the basic reproduction number, R_0, by considering the infected compartments of the system (2.1)–(2.8) [11].

If F_i be the rate of appearance of new infection in the i compartment and V_i be the rate of transfer of individuals out of i, given the disease-free equilibrium, then R_0 is the spectral radius (largest eigenvalue) of the next generation matrix denoted by $G = FV^{-1}$

Now, let $x = (E_h, I_h, E_m, I_m)^T$ which can be written in the form

$\frac{dx}{dt} = F_i(x) - V_i(x)$, where

$$F_i(x) = \begin{pmatrix} \frac{b\beta_h S_h I_m}{1+\nu_h I_m} \\ 0 \\ \frac{b\beta_m S_m I_h}{1+\nu_m I_h} \\ 0 \end{pmatrix} \quad \text{and} \quad V_i(x) = \begin{pmatrix} (\alpha_h + \mu_h)E_h \\ (r+\delta_h+\mu_h)I_h - \alpha_h E_h \\ (\alpha_m+\mu_m)E_m \\ (\mu_m+\delta_m)I_m - \alpha_m E_m \end{pmatrix}$$

Evaluating the Jacobian matrix of $F(x)$ at the disease–free equilibrium, E_0 , gives

$$\mathrm{F} = \begin{pmatrix} 0 & 0 & 0 & \frac{b\beta_h\Lambda_h}{\mu_h+e} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{b\beta_m\Lambda_m}{\mu_m} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{2.10}$$

Similarly, evaluating $V(x)$ at disease-free equilibrium, E_0, yields

$$\mathrm{V} = \begin{pmatrix} \alpha_h+\mu_h & 0 & 0 & 0 \\ -\alpha_h & r+\mu_h+\delta_h & 0 & 0 \\ 0 & 0 & \alpha_m+\mu_m & 0 \\ 0 & 0 & -\alpha_m & \mu_m+\delta_m \end{pmatrix}$$

Thus,

$$V^{-1} = \begin{pmatrix} \frac{1}{\alpha_h+\mu_h} & 0 & 0 & 0 \\ \frac{\alpha_h}{(\alpha_h+\mu_h)(r+\delta_h+\mu_h)} & \frac{1}{r+\delta_h+\mu_h} & 0 & 0 \\ 0 & 0 & \frac{1}{\alpha_m+\mu_m} & 0 \\ 0 & 0 & \frac{\alpha_m}{(\alpha_m+\mu_m)(\mu_m+\delta_m)} & \frac{1}{\mu_m+\delta_m} \end{pmatrix} \tag{2.11}$$

Hence,

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{b\alpha_m\beta_h\Lambda_h}{\mu_h(\mu_m+\delta_m)(\alpha_m+\mu_m)} & \frac{b\beta_h\Lambda_h}{\mu_h(\mu_m+\delta_m)} \\ 0 & 0 & 0 & 0 \\ \frac{b\alpha_h\beta_m\Lambda_m}{\mu_m(r+\delta_h+\mu_h)(\alpha_h+\mu_h)} & \frac{b\beta_m\Lambda_m}{\mu_m(r+\delta_h+\mu_h)} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{2.12}$$

Thus,

$$R_0 = \sqrt{\frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h+e)(\alpha_h+\mu_h)(r+\delta_h+\mu_h)(\alpha_m+\mu_m)(\mu_m+\delta_m)\mu_m}} \tag{2.13}$$

is the largest eigenvalue of the next-generation matrix (2.12),

$$R_0 = b\sqrt{\frac{\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h+e)(\alpha_h+\mu_h)(r+\delta_h+\mu_h)(\alpha_m+\mu_m)(\mu_m+\delta_m)\mu_m}}$$

This can be written as

$$R_0 = \sqrt{R_h R_m} \tag{2.14}$$

Where $R_h = \dfrac{b\alpha_h\beta_h\Lambda_h}{(\mu_h+e)(\alpha_h+\mu_h)(r+\delta_h+\mu_h)}$ and $R_m = \dfrac{b\alpha_m\beta_m\Lambda_m}{(\alpha_m+\mu_m)(\mu_m+\delta_m)\mu_m}$

R_h, describes the number of humans that one infectious mosquito infects over its expected infection period in a completely susceptible human population. While R_m is the number of mosquitoes infected by one infectious human during the period of infectiousness in a completely susceptible mosquito population.

3.3 Stability of Disease-free Equilibrium

The basic reproduction number (2.13) can be used to determine the local stability of the disease-free equilibrium point for the system (2.1) – (2.8). The local stability can be analyzed using the Jacobian matrix of the system (2.1)-(2.8).

Theorem 1

The disease-free equilibrium point, E_0, for the system (2.1)-(2.8) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:

We evaluate the Jacobian matrix of the system (2.1) – (2.8) at the disease – free equilibrium, E_0, and thus obtain

$$J(E_0) = \begin{pmatrix} J_{11} & 0 & 0 & 0 & J_{15} & 0 & 0 & J_{18} \\ J_{21} & J_{22} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & J_{33} & 0 & 0 & 0 & 0 & J_{38} \\ 0 & 0 & J_{43} & J_{44} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & J_{54} & J_{55} & 0 & 0 & 0 \\ 0 & 0 & 0 & J_{64} & 0 & J_{66} & 0 & 0 \\ 0 & 0 & 0 & J_{74} & 0 & 0 & J_{77} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & J_{87} & J_{88} \end{pmatrix} \tag{2.15}$$

Where,

$J_{11} = -(\mu_h + e),\ J_{15} = \omega,\ J_{18} = -\dfrac{b\beta_h\Lambda_h}{\mu_h+e},\ J_{21} = e,\ J_{22} = -\mu_h,\ J_{33} = -(\alpha_h+\mu_h),\ J_{38} = \dfrac{b\beta_h\Lambda_h}{\mu_h+e},\ J_{43} = \alpha_h,\ J_{44} = -(r+\mu_h+\delta_h),\ J_{54} = r,$

$J_{55} = -(\mu_h+\omega),\ J_{64} = -\dfrac{b\beta_m\Lambda_m}{\mu_m},\ J_{66} = \mu_m,\ J_{74} = \dfrac{b\beta_m\Lambda_m}{\mu_m},\ J_{77} = -(\alpha_m+\mu_m),\ J_{87} = \alpha_m,\ J_{88} = -(\mu_m+\delta_m)$

Hence, the characteristic equation of (2.15) is given as

$$\begin{vmatrix} J_{11}-\lambda & 0 & 0 & 0 & \omega & 0 & 0 & -\dfrac{b\beta_h\Lambda_h}{\mu_h+e} \\ e & J_{22}-\lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & J_{33}-\lambda & 0 & 0 & 0 & 0 & \dfrac{b\beta_h\Lambda_h}{\mu_h+e} \\ 0 & 0 & \alpha_h & J_{44}-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & r & J_{55}-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & -\dfrac{b\beta_m\Lambda_m}{\mu_m} & 0 & J_{66}-\lambda & 0 & 0 \\ 0 & 0 & 0 & \dfrac{b\beta_m\Lambda_m}{\mu_m} & 0 & 0 & J_{77}-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m & J_{88}-\lambda \end{vmatrix} = 0 \tag{2.16}$$

We need to show that all the eigenvalues of the characteristic equation (2.15) are negative. Clearly we observe that the second column of (3.15) contain only the diagonal terms so that we have

$$(-\mu_h-\lambda)\begin{vmatrix} J_{11}-\lambda & 0 & 0 & \omega & 0 & 0 & -\dfrac{b\beta_h\Lambda_h}{\mu_h+e} \\ 0 & J_{33}-\lambda & 0 & 0 & 0 & 0 & \dfrac{b\beta_h\Lambda_h}{\mu_h+e} \\ 0 & \alpha_h & J_{44}-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & r & J_{55}-\lambda & 0 & 0 & 0 \\ 0 & 0 & -\dfrac{b\beta_m\Lambda_m}{\mu_m} & 0 & J_{66}-\lambda & 0 & 0 \\ 0 & 0 & \dfrac{b\beta_m\Lambda_m}{\mu_m} & 0 & 0 & J_{77}-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha_m & J_{88}-\lambda \end{vmatrix} = 0 \tag{2.17}$$

Also, the fifth column of (2.17) contains only the diagonal term which gives

$$(-\mu_h-\lambda)(-\mu_m-\lambda)\begin{vmatrix} J_{11}-\lambda & 0 & 0 & \omega & 0 & -\frac{b\beta_h\Lambda_h}{\mu_h+e} \\ 0 & J_{33}-\lambda & 0 & 0 & 0 & \frac{b\beta_h\Lambda_h}{\mu_h+e} \\ 0 & \alpha_h & J_{44}-\lambda & 0 & 0 & 0 \\ 0 & 0 & r & J_{55}-\lambda & 0 & 0 \\ 0 & 0 & \frac{b\beta_m\Lambda_m}{\mu_m} & 0 & J_{77}-\lambda & 0 \\ 0 & 0 & 0 & 0 & \alpha_m & J_{88}-\lambda \end{vmatrix}=0 \quad (2.18)$$

In the same way, the first column of equation (2.18) contains only the diagonal term. Evaluating further yields

$$(-\mu_h-\lambda)(-\mu_m-\lambda)(-(e+\mu_h)-\lambda)\begin{vmatrix} J_{33}-\lambda & 0 & 0 & 0 & \frac{b\beta_h\Lambda_h}{\mu_h+e} \\ \alpha_h & J_{44}-\lambda & 0 & 0 & 0 \\ 0 & r & J_{55}-\lambda & 0 & 0 \\ 0 & \frac{b\beta_m\Lambda_m}{\mu_m} & 0 & J_{77}-\lambda & 0 \\ 0 & 0 & 0 & \alpha_m & J_{88}-\lambda \end{vmatrix}=0 \quad (2.19)$$

Similarly, the third column of equation (2.19) contains only the diagonal terms so that we obtain

$$(-\mu_h-\lambda)(-\mu_m-\lambda)(-(e+\mu_h)-\lambda)(-(\mu_h+\omega)-\lambda)\begin{vmatrix} J_{33}-\lambda & 0 & 0 & \frac{b\beta_h\Lambda_h}{\mu_h+e} \\ \alpha_h & J_{44}-\lambda & 0 & 0 \\ 0 & \frac{b\beta_m\Lambda_m}{\mu_m} & J_{77}-\lambda & 0 \\ 0 & 0 & \alpha_m & J_{88}-\lambda \end{vmatrix}=0 \quad (2.19)$$

Hence,

$(-\mu_h-\lambda)(-\mu_m-\lambda)(-(e+\mu_h)-\lambda)(-(\mu_h+\omega)-\lambda)=0$

So that, $\lambda_1=-\mu_h,\ \lambda_2=-\mu_m,\ \lambda_3=-(\mu_h+e),\ \lambda_4=-(\mu_h+\omega)$ and

$$\begin{vmatrix} -(\alpha_h+\mu_h)-\lambda & 0 & 0 & \frac{b\beta_h\Lambda_h}{\mu_h+e} \\ \alpha_h & -(r+\mu_h+\delta_h)-\lambda & 0 & 0 \\ 0 & \frac{b\beta_m\Lambda_m}{\mu_m} & -(\alpha_m+\mu_m)-\lambda & 0 \\ 0 & 0 & \alpha_m & -(\mu_m+\delta_m)-\lambda \end{vmatrix}=0 \quad (2.20)$$

Therefore, solving the determinant of (2.20) yields the characteristics equation

$$(\lambda+\alpha_h+\mu_h)(\lambda+r+\mu_h+\delta_h)(\lambda+\alpha_m+\mu_m)(\lambda+\mu_m+\delta_m)-\frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h+e)\mu_m}=0 \quad (2.21)$$

Now, let $B_1=\alpha_h+\mu_h,\ B_2=r+\mu_h+\delta_h,\ B_3=\alpha_m+\mu_m,\ B_4=\mu_m+\delta_m$ so that equation (2.21) becomes

$$(\lambda+B_1)(\lambda+B_2)(\lambda+B_3)(\lambda+B_4)-\frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h+e)\mu_m}=0 \quad (2.22)$$

Therefore, expanding equation (2.22) yields

$$A_4\lambda^4+A_3\lambda^3+A_2\lambda^2+A_1\lambda+A_0=0 \quad (2.23)$$

Where,

$$\begin{aligned} A_4&=1 \\ A_3&=B_1+B_2+B_3+B_4 \\ A_2&=(B_1+B_2)(B_3+B_4)+B_1B_2+B_3B_4 \\ A_1&=(B_1+B_2)B_3B_4+(B_3+B_4)B_1B_2 \\ A_0&=B_1B_2B_3B_4-\frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h+e)\mu_m}=B_1B_2B_3B_4(1-R_0{}^2) \end{aligned} \quad (2.24)$$

We apply Routh-Hurwitz criterion to prove that all roots of the polynomial (2.23) have negative real parts if and only if the coefficients, A_i, are positive and the determinants of the matrices, $H_i>0$. For $i=0,1,2,3,4$. Therefore, from equation (2.24), we see that $A_1>0,\ A_2>0,\ A_3>0,$ and $A_4>0$, since B_1,B_2,B_3,B_4 are all positive. That is, $H_1=A_3>.0$

$$H_2 = \begin{vmatrix} A_3 & A_4 \\ A_1 & A_2 \end{vmatrix} = A_2A_3 - A_1A_4 > 0$$

$$H_3 = \begin{vmatrix} A_3 & A_4 & 0 \\ A_1 & A_2 & A_3 \\ 0 & A_0 & A_1 \end{vmatrix} > 0 \quad \text{and} \quad H_4 = \begin{vmatrix} A_3 & A_4 & 0 & 0 \\ A_1 & A_2 & A_3 & A_4 \\ 0 & A_0 & A_1 & A_2 \\ 0 & 0 & 0 & A_0 \end{vmatrix} > 0$$

Therefore, all the eigenvalues of the polynomial (2.23) have negative real parts, implying that $\lambda_5 < 0,\ \lambda_6 < 0,\ \lambda_7 < 0,\ \lambda_8 < 0$. Hence, since all the values of $\lambda_i < 0, \text{for } i = 1,2,3,4,5,6,7,8$ when $R_0 < 1$ we conclude that the disease-free equilibrium point is locally asymptotically stable.

However, if $R_0 > 1$, we observe that $A_0 < 0$ and by Descartes' rule of signs, there is exactly one sign change in the sequence, A_4, A_3, A_2, A_1, A_0. of the coefficients of the polynomial (2.10). This implies that, there exists one eigenvalue with positive real part. Hence, the disease-free equilibrium point will be unstable.

Conclusion

We propose a new model for malaria control consisting of an 8-dimenssional system of ordinary differential equations. The Disease Free Equilibrium is established for the system (2.1)-(2.8). A reproduction number R_0, obtained using the next generation matrix method. The local stability of the Disease Free Equilibrium of the model was determined using the Routh Hurwitz condition for stability. Our results indicate that all the eigenvalues are negative, that is, $\lambda_i < 0$ for $i = 1,2,3,4,5,6,7,8$, when $R_0 < 1$. This implies that the disease free equilibrium is locally asymptotically stable, indicating that malaria can be eradicated from the entire population. Therefore, we conclude that behavioural change plays a vital role in the fight against the spread and most importantly eradication of malaria, if implemented in control programmes alongside other intervention strategies.

References

[1] World Health Organization (2014). Fact Sheet on Malaria. Electronic, www.who.int/mediacentre/factsheets/fs094/en/

[2] World Health Organization (2017), World Malaria Report. WHO Global Malaria Programme

[3] World Health Organization (2016), World malaria report, WHO Press, Switzerland. http//www.who.int/malaria

[4] Koella K. C., (2014). On the use of mathematical models of malaria transmission, Acta Tropica, 49, pp. 1-25.

[5] Yang H. M., (2016). Malaria transmission model for different levels of acquired immunity and temperature-dependent parameters (vector), Revista de Satude Publica, 34, pp. 223{231.

[6] Chitnis N., (2016). Using Mathematical Models in Controlling the Spread of Malaria, Ph.D. thesis, Program in Applied Mathematics, University of Arizona, Tucson, AZ.

[7] Franncis T. O., Gabriel A. O. and George T. A. (2017). "Transmission Dynamics of Malaria in Ghana" Journal of Mathematics Research. Vol. 4. No. 6, http://dx.doi.org/10.5539/jmr.v4n6p22

[8] Monroe P.G. and Catlla, A.J. (2012). Modeling the Effects of Malaria Preventive Measures.

[9] Maliyoni M., Mwamtobe P. M. M., Hove-Musekwa S. D. and Tchuenche J. M. (2016). Modelling the Role of Diagnosis, Treatment, and Health Education on Multidrug-Resistant Tuberculosis Dynamics, International Scholarly Research Network ISRN Biomathematics Volume, Article ID 459829, 20 pages.

[10] Olaniyi S., and Obabiyi O. S. (2013). Mathematical Model for Malaria Transmission Dynamics In Human and Mosquito Populations with Nonlinear Forces of Infection, International Journal of Pure and Applied Mathematics, Volume 88 No. 1, 125-156. ISSN: 1314-3395 (on-line version), url: http://www.ijpam.eu

[11] Diekmnn, O. and Heesterbeek J. A. P. (2017). Mathematical Epidemiology of Infectious Disease: ‘’Application of Matrix Theory to Model Building, Analysis and Interpretation. JAMA 271(9): 698-702