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Mathematical Model for the Transmission Dynamics and Control of Malaria by Incorporating Behavioural Change

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Abstract

In this paper, we propose a mathematical model for the transmission dynamics of malaria by incorporating behavioral change via education as a control strategy against the spread of malaria. Analytical study is carried out to investigate the local stability of the system, given a threshold parameter known as the basic reproduction number R_0, which is derived using the next generation matrix method. Result obtained showed that, disease-free equilibrium of the system is locally asymptotically stable if $R_0 < 1$. Numerical simulation carried out on the system shows that behavioral change plays a significant role towards achieving a malaria-free society.

Keywords: Malaria, infections, Disease, mosquito, Equilibrium.

Introduction

Malaria is an infectious disease cause by plasmodium parasite and transmitted between humans through bite of female anopheles mosquitoes, (cobremeskel and krogstad, 2015). Malaria an ancient disease having a huge social, economic and health burden. It is predominantly present in the tropical countries (mandal *et al.*). Even though the disease has been investigated for hundreds of years, it still remains a major public health problem with 91 countries. The global record of malaria in 2015 was 212 million new cases and 429000 deaths. Across Africa, millions of people

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still lack access to the tools they need to prevent and treat the disease (WHO, 2016). Malaria has for many years been considered as a global issue, and many epidemiologist and other scientists invest their efforts in learning the dynamics of malaria and to control its transmission. From interactions with those scientists, mathematicians have developed a significant and effective tool, namely, mathematical models of malaria, given an insight into the interaction between the host and a vector population, the dynamics of malaria, how to control malaria transmission and eventually how to eradicate it (cobremeskel and krogstad, 2015).

Malaria is one of the most devastating infectious diseases in the world, infecting millions of people annually and is a major cause of mortality. The World Health Organization reported that in 2016, there were 216 million cases of malaria and about 90% of reported cases occurred in Africa (WHO 2018).This life-threatening disease is caused by the single-celled genus plasmodium parasites which are transmitted through bites of infected anopheles mosquitoes, biting mainly between dusk and dawn. Plasmodium vivax, plasmodium ovale, plasmodium malariae, plasmodium falciparum, and plasmodium knowlesi are five parasite species identified to cause malaria in humans. Plasmodium falciparum (P. falciparum) causes most of the severe diseases and deaths which is most prevalent in Sub-Saharan Africa. Children below age 5 and pregnant women are most susceptible to the disease. In particular, malaria claims the life of a child every 2 minutes. The major symptoms of malaria include fatigue, chill, headache, abdominal and back pain, diarrhoea, vomiting and fever. Severe malaria infection can result in serious complications affecting brain, lungs, kidneys and other organs (WHO, 2014). Despite global efforts to control malaria, the incidence of the disease is increasing in endemic regions such as, sub-Saharan Africa. While Africa accounts for 91%of malaria deaths worldwide, Nigeria being the most populous country on the continent accounted for 24% of malaria deaths globally in 2016 (WHO, 2018).

Mathematical modelling has been used as a tool to understand the dynamics of infectious diseases. From 1911 till present, various mathematical models have been derived which take into account various possible scenarios of the transmission dynamics of malaria. Some of these include Koella (1991), Chitnis (2005), Tumwiine et al. (2008), Peter (2010), Adamu and Kimbir (2013), Adamu *et al* (2017). In another attempt to alleviate the problem of malaria transmission, Olaniyi and Obabiyi (2013) developed a model that considered the impact of antibodies produced by both human and mosquito populations in response to the presence of malaria parasites. But we observe that in many regions where the disease burden is high, very few people live above the poverty level. In other words, humans will be able to boost production of antibodies with the intake of the right food or supplements when hunger and poverty has been eradicated knowing that malaria affects some of the poorest regions of the world, with very limited resources (Chitnis, 2005). However, Maliyoni et al., (2012) observed that when interventions such as education are introduced in the fight against infectious diseases, trends improve in the population.

In this paper, we propose a mathematical model that incorporates an education-based behavioral change as an extension of Olaniyi and Obabiyi (2013) who used a system of seven dimensional ODE to modeling the transmission the transmission of plasmodium falciparum malaria between humans and mosquitos with nonlinear forces of infection in form of saturated incidence rate, these incidence rate produce anti bodies in response to the presence of parasite coursing malaria in both human and mosquito population. They investigated the stability analysis of (DFE) and according to their result, (DFE) was asymptotically stable when $R_0 < 1$, and unstable when $R_0 > 1$.

By extension, we incorporated the protected humans' population which follows as susceptible-protected-exposed-infectious-recovered (SPEIR) pattern and the mosquito population follows susceptible-exposed-infectious (SEI) pattern. Hence, our aim is to determine the effect of behavioral change as a control strategy against the spread of malaria.

Model Formulation

The model comprises of two interacting (human and mosquito) populations as developed by Olaniyi and Obabiyi (2013). A modified version is presented in Fig. 2 below which incorporates an additional class of humans called, protected humans.

State variables and parameters of the models

Table 1: Description of the state variables of the models

State variables	Description
$S_h(t)$	Number of human host susceptible to malaria infection at time t
$P_h(t)$	Number of protected human host 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

Table 2: Description of the model Parameters of the models

State variables	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 human
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 exposed state to infectious
r	Per capital recovery rate for humans from the infectious state of 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 be humans
v_h	Proportion of antibody produced by mosquito in response to the incidence of infection caused be humans
e	Per capital rate of behavioural change.

Epidemiological Flow Diagrams

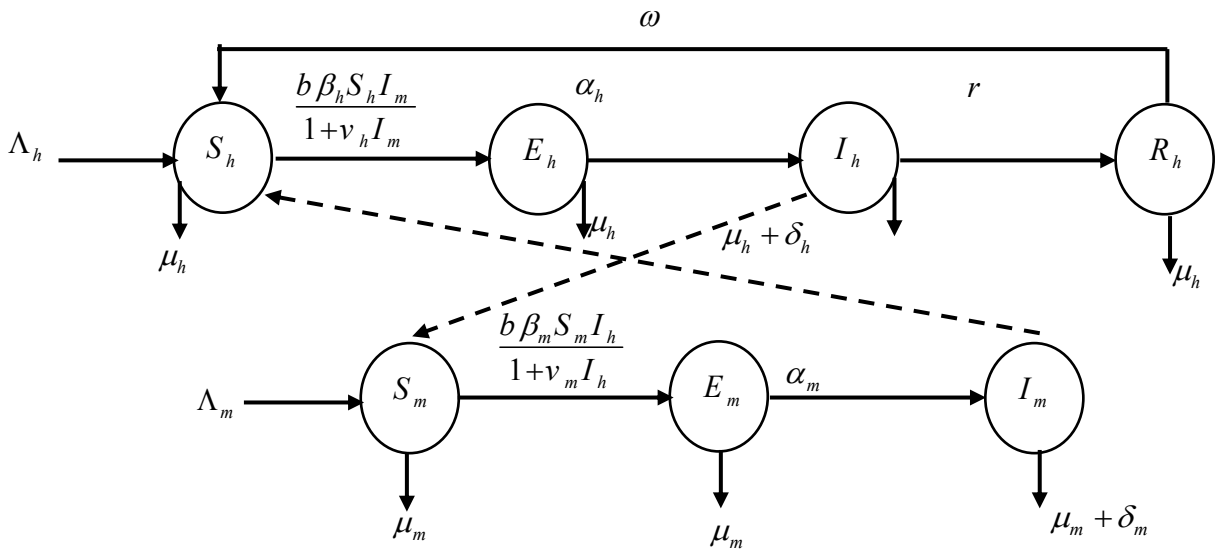

Fig. 1: Epidemiological flow diagram for the existing model by Olaniyi & Obabiyi (2013)

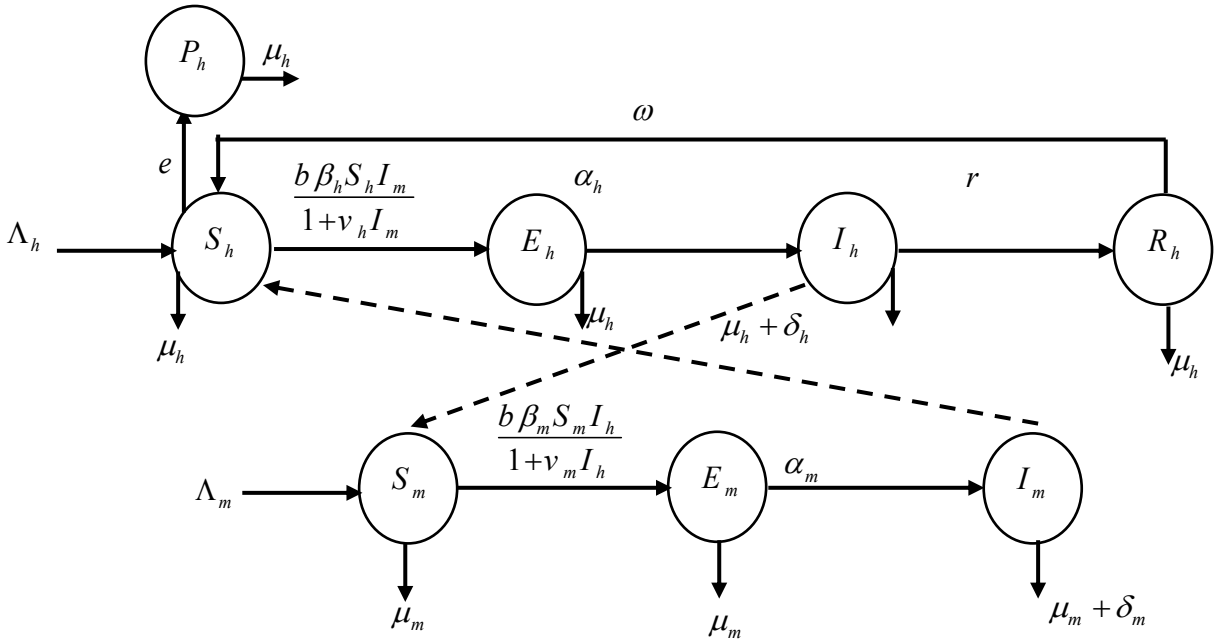

Fig. 2: Epidemiological flow diagram of the model with behavioral change (modified model)

Model Equations

From the epidemiological flow diagram in Fig. 1, the following model equations are obtained

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

$$\frac{dE_h}{dt} = \frac{b\beta_h S_h(t) I_m(t)}{1+v_h I_m(t)} - (\alpha_h + \mu_h) E_h(t) \quad (2)$$

$$\frac{dI_h}{dt} = \alpha_h E_h(t) - (r + \mu_h + \delta_h) I_h(t) \quad (3)$$

$$\frac{dR_h}{dt} = rI_h(t) - (\mu_h + \omega) R_h(t) \quad (4)$$

$$\frac{dS_m}{dt} = \Lambda_m - \frac{b\beta_m S_m(t) I_h(t)}{1+v_m I_h(t)} - \mu_m S_m(t) \quad (5)$$

$$\frac{dE_m}{dt} = \frac{b\beta_m S_m(t) I_h(t)}{1+v_m I_h(t)} - (\alpha_m + \mu_m) E_m(t) \quad (6)$$

$$\frac{dI_m}{dt} = \alpha_m E_m(t) - (\mu_m + \delta_m) I_m(t) \quad (7)$$

Together with the initial conditions:

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

But, Fig. 2 shows an additional compartment in the human population called the protected compartment, denoted by $P_h(t)$. We assume that susceptible humans can be protected from contacts with mosquitoes at a rate *e* when they exhibit positive behavioral change. Thus, equation of the modified model for the transmission dynamics of the disease is given by the following system of ordinary differential equations

$$\frac{dS_h}{dt} = \Lambda_h - \frac{b\beta_h S_h(t) I_m(t)}{1+v_h I_m(t)} - (\mu_h + e) S_h(t) + \omega R_h(t) \quad (8)$$

$$\frac{dP_h}{dt} = eS_h(t) - \mu_h P_h(t) \quad (9)$$

$$\frac{dE_h}{dt} = \frac{b\beta_h S_h(t) I_m(t)}{1+v_h I_m(t)} - (\alpha_h + \mu_h) E_h(t) \quad (10)$$

$$\frac{dI_h}{dt} = \alpha_h E_h(t) - (r + \mu_h + \delta_h) I_h(t) \quad (11)$$

$$\frac{dR_h}{dt} = rI_h(t) - (\mu_h + \omega) R_h(t) \quad (12)$$

$$\frac{dS_m}{dt} = \Lambda_m - \frac{b\beta_m S_m(t) I_h(t)}{1+v_m I_h(t)} - \mu_m S_m(t) \quad (13)$$

$$\frac{dE_m}{dt} = \frac{b\beta_m S_m(t) I_h(t)}{1+v_m I_h(t)} - (\alpha_m + \mu_m) E_m(t) \quad (14)$$

$$\frac{dI_m}{dt} = \alpha_m E_m(t) - (\mu_m + \delta_m) I_m(t) \quad (15)$$

Together with the initial conditions:

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

MODEL ANALYSIS

Existence of Disease-free Equilibrium Point

The disease free equilibrium point E_0, of the system of equations is obtained by setting the equations to zero. When $E_h=0, I_h=0, R_h=0, E_m=0, I_m=0$ is given by

$$E_0=\left(S_h^*, P_h^*, E_h^*, I_h^*, R_h^*, S_m^*, E_m^*, I_m^*\right)=\left(\frac{\Lambda_h}{\mu_h+e}, \frac{e\Lambda_m}{\mu_m}, 0, 0\right) \tag{16}$$

Basic Reproduction Number

The basic reproduction number denoted by, R_0, is an important parameter which is used to study the behavior 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 by Diekmann and Heesterbeek (2000) to obtain the basic reproduction number, R_0, by considering the diseased compartments of the system (8) - (15). The rate of appearance of new infections and the transition rate are given respectively by

$$F_1=\begin{pmatrix} \frac{b\beta_h S_h I_m}{1+v_h I_m} \\ 0 \\ \frac{b\beta_m S_m I_h}{1+v_m I_h} \\ 0 \end{pmatrix} \quad \text{and} \quad v_i=\begin{pmatrix} (\alpha_h+\mu_h)E_h \\ (r+\delta_h+\mu_h)I_h-\alpha_h E_h \\ (\alpha_m+\mu_m)E_m \\ (\mu_m+\delta_m)I_m-\alpha_m E_m \end{pmatrix}.$$

Evaluating the Jacobian matrix of F_i and V_i, at the disease–free equilibrium, E_0 We obtain

$$F=\begin{pmatrix} 0 & 0 & 0 & \frac{b\beta_h\Lambda_h}{\mu_h+e} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{b\beta_m\Lambda_m}{\mu_m+e} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \text{ and } v=\begin{pmatrix} \alpha_h+\mu_h & 0 & 0 & 0 \\ -\alpha_h & r+\mu_h+\delta_h & 0 & 0 \\ 0 & 0 & \alpha_m+\mu_m & 0 \\ 0 & 0 & -\alpha_m & \mu_m+\delta_m \end{pmatrix},$$

So that

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

Hence

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

Therefore, we evaluate the characteristics equation, $\left|FV^{-1}-\lambda I\right|=0$ and obtained basic reproduction number as:

$$R_0=\sqrt{\frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h+e)(\alpha_h+\mu_h)(r+\delta_h+\mu_h)(\mu_m+\delta_m)\mu_m}} \tag{17}$$

Local Stability of Disease-free Equilibrium

The basic reproduction number (16) is used to analyze the local stability of the equilibrium point of the system (8)-(15).

Proposition

The disease-free equilibrium, E_0, is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$

Proof:

The Jacobian of the system (8)–(15) evaluated at the disease-free equilibrium point E_0, is obtained as

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

Where

$$J_{11}=-(\mu_h+e),\ J_{15}=\omega, J_{18}=-\frac{b\beta_h\Lambda_h}{\mu_h+e}, J_{21}=e, J_{22}=-\mu_h, J_{33}=-(\alpha_h+\mu_h), J_{38}=\frac{b\beta_h\Lambda_h}{\mu_h+e},$$

$$J_{43}=\alpha_h, J_{44}=-(r+\mu_h+\delta_h), J_{54}=r, J_{55}=-(\mu_h+\omega), J_{64}=-\frac{b\beta_m\Lambda_m}{\mu_m}, J_{66}=\mu_m,$$

$$J_{74}=\frac{b\beta_m\Lambda_m}{\mu_m}, J_{77}=-(\alpha_m+\mu_m), J_{87}=\alpha_m, J_{88}=-(\mu_m+\delta_m).$$

The characteristic equation of (17) is given as

$$\begin{vmatrix} J_{11}-\lambda & 0 & 0 & 0 & \omega & 0 & 0 & -\frac{b\beta_h\Lambda_h}{\mu_h+e} \\ e & J_{22}-\lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & J_{33}-\lambda & 0 & 0 & 0 & 0 & \frac{b\beta_h\Lambda_h}{\mu_h+e} \\ 0 & 0 & \alpha_h & J_{44}-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & r & J_{55}-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & -\frac{b\beta_m\Lambda_m}{\mu_m+e} & 0 & J_{66}-\lambda & 0 & 0 \\ 0 & 0 & 0 & \frac{b\beta_m\Lambda_m}{\mu_m} & 0 & 0 & J_{77}-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m & J_{88}-\lambda \end{vmatrix} = 0. \quad (18)$$

We need to show that all the eigenvalues of the characteristic equation (18) are negative. Thus, evaluating the equation and simplifying (18) yields

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

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

$$\begin{vmatrix} -(\alpha_h+\mu_h)-\lambda & 0 & 0 & \frac{b\beta_h\Lambda_h}{\mu_h+e} \\ \alpha_h & -(r+\mu_h+\delta_h)-\lambda & 0 & 0 \\ 0 & \frac{b\beta_m\Lambda_m}{\mu_m} & -(\alpha_m+\mu_m)-\lambda & 0 \\ 0 & 0 & \alpha_m & -(\mu_m+\delta_m)-\lambda \end{vmatrix} = 0. \quad (19)$$

Therefore, simplifying (19) further we obtain

$$(\lambda+\alpha_h+\mu_h)(\lambda+r+\mu_h+\delta_h)(\lambda+\alpha_m+\mu_m)(\lambda+r+\mu_m+\delta_m)-\frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h+e)\mu_m}=0 \quad (20)$$

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, we obtain

$$(\lambda+B_1)(\lambda+B_2)(\lambda+B_3)(\lambda+B_4)-\frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h+e)\mu_m} \quad (21)$$

By expanding and simplifying (21), we get

$$A_4\lambda^4+A_3\lambda^3+A_2\lambda^2+A_1\lambda+A_0=0 \quad (22)$$

Where,

$$A_4=1$$
$$A_3=B_1+B_2+B_3+B_4$$
$$A_2=(B_1+B_2)(B_3+B_4)+B_1B_2+B_3B_4$$
$$A_1=(B_1+B_2)B_3B_4+(B_3+B_4)B_1B_2 \quad (23)$$

$$A_0 = B_1B_2B_3B_4 - \frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h + e)\mu_m} = B_1B_2B_3B_4\left(1 - R_0^2\right)$$

Thus, applying the Routh-Hurwitz criterion which states that all roots of the polynomial (22) have negative real parts if and only if the coefficients, A_i, are positive and the determinants of the matrices, $H_i > 0$ .For $i = 1,2,3,4$. Therefore, from equation (23), 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, $H_1 = A_3 > 0$.

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

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

Therefore, all the eigenvalues of the polynomial (22) have negative real parts, implying that $\lambda_5 < 0, \lambda_6 < 0, \lambda_7 < 0, \lambda_8 < 0.$ Hence, since all the values of $\lambda_i < 0$, 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, (Polyanin and Manzhirov 2007), 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 (22), implying that, there exists one eigenvalue with positive real part. Hence, the disease-free equilibrium point will be unstable.

Numerical Experiments

Numerical experiments were performed using MATLAB to study and compare the behavior of the Olaniyi and Obabiyi (2013) model and the model with behavioral change given by the system (8)-(15) on the human populations.

Table 3: Parameter values and initial variables

Parameter	1	2	3	4	5	6	Source
Λ_h	0.000215	0.000215	0.000215	0.000215	0.000215	0.000215	Olaniyi&Obabiyi(2013)
Λ_m	0.007	0.007	0.007	0.007	0.007	0.007	Olaniyi&Obabiyi(2013)
B	0.12	0.12	0.12	0.12	0.12	0.12	Olaniyi&Obabiyi(2013)
β_h	0.1	0.1	0.1	0.1	0.1	0.1	Olaniyi&Obabiyi(2013)
β_m	0.09	0.09	0.09	0.09	0.09	0.09	Olaniyi&Obabiyi(2013)
μ_h	0.0000548	0.0000548	0.0000548	0.0000548	0.0000548	0.0000548	Olaniyi&Obabiyi(2013)
μ_m	1/15	1/15	1/15	1/15	1/15	1/15	Olaniyi&Obabiyi(2013)
δ_h	0.001	0.001	0.001	0.001	0.001	0.001	Olaniyi&Obabiyi(2013)
δ_m	0.01	0.01	0.01	0.01	0.01	0.01	Olaniyi&Obabiyi(2013)
α_h	1/17	1/17	1/17	1/17	1/17	1/17	Olaniyi&Obabiyi(2013)
α_m	1/18	1/18	1/18	1/18	1/18	1/18	Olaniyi&Obabiyi(2013)
R	0.05	0.05	0.05	0.05	0.05	0.05	Olaniyi&Obabiyi(2013)
ω	1/730	1/730	1/730	1/730	1/730	1/730	Olaniyi&Obabiyi(2013)
v_h	0	0	0	0.5	0.5	0.5	Olaniyi&Obabiyi(2013)
v_m	0	0	0	0.5	0.5	0.5	Olaniyi&Obabiyi(2013)
E	0.25	0.5	0.75	0.25	0.5	0.75	Assumed
$S_h(0)$	100	100	100	100	100	100	Olaniyi&Obabiyi(2013)
$E_h(0)$	20	20	20	20	20	20	Olaniyi&Obabiyi(2013)
$I_h(0)$	10	10	10	10	10	10	Olaniyi&Obabiyi(2013)
$R_h(0)$	0	0	0	0	0	0	Olaniyi&Obabiyi(2013)
$S_m(0)$	1000	1000	1000	1000	1000	1000	Olaniyi&Obabiyi(2013)
$E_m(0)$	20	20	20	20	20	20	Olaniyi&Obabiyi(2013)
$I_m(0)$	30	30	30	30	30	30	Olaniyi&Obabiyi(2013)
$P_h(0)$	5	5	5	5	5	5	Assumed

Two approaches were deployed in conducting the numerical the experiments. First, we considered a case where no antibody is produced as a form of immune response to the presence of malaria parasites while varying the rate of behavioral change. Figures 3, 4, and show the varying effect of behavioral change on the infected human populations while parameter values in Table 3 remained unchanged and $R_0 < 1$.

Secondly, we considered implementing the proportions of antibodies produced by susceptible humans and mosquitoes in response to the presence of malaria parasites at 50% and varied the proportion of behavioral change of protected humans as shown in Figures 6, 7, and 8

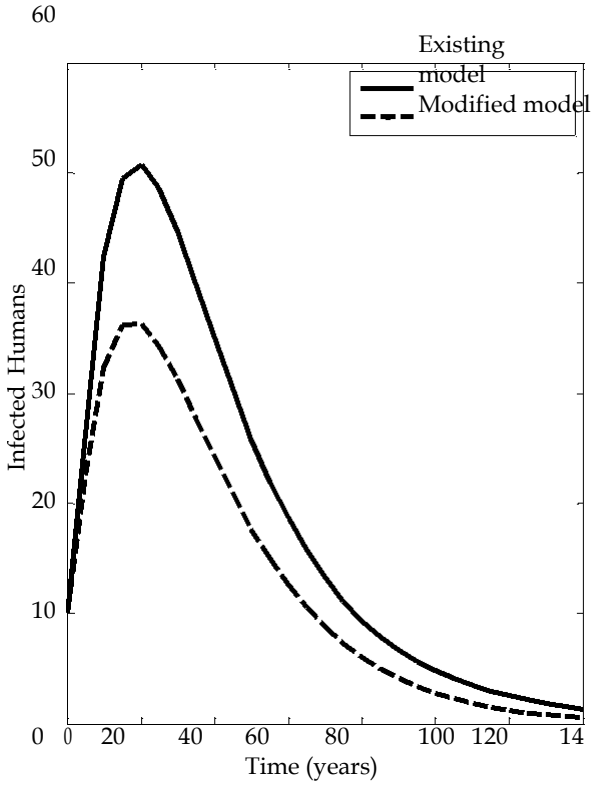

Fig. 3: The behavior of the models when e=0.25

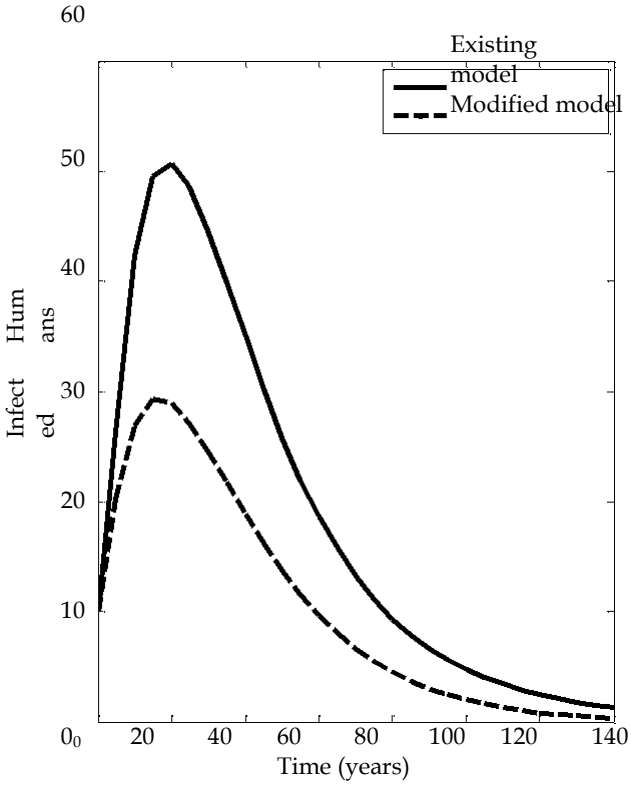

Fig. 4: The behavior of the model e=0.5

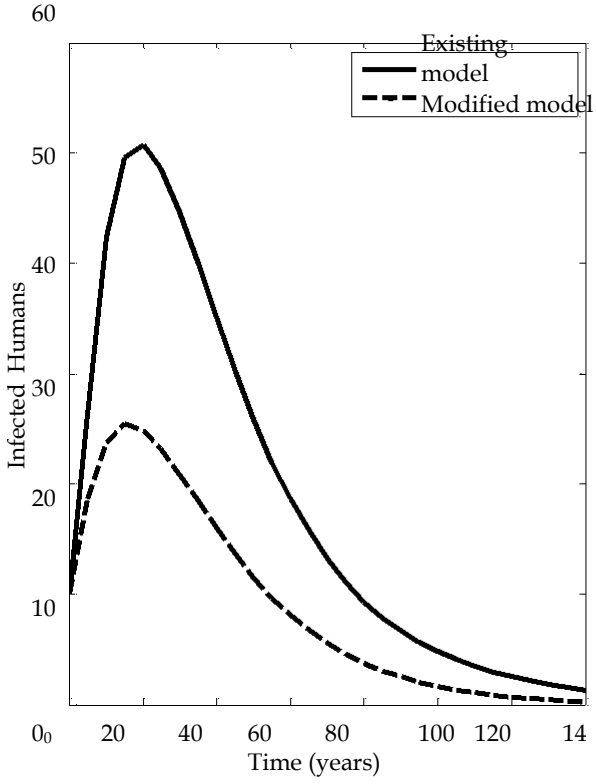

Fig. 5: The behavior of the models when e =0.75

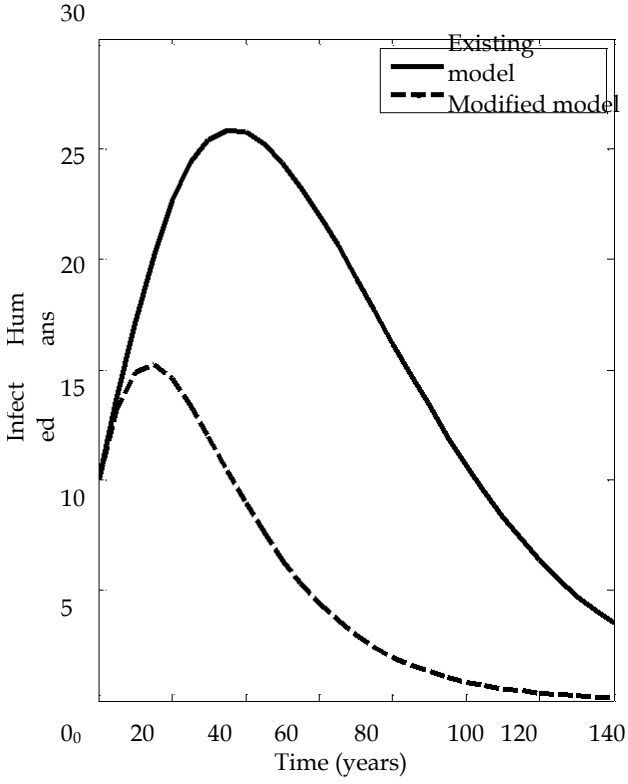

Fig. 6: The behavior of the models when e =0.25

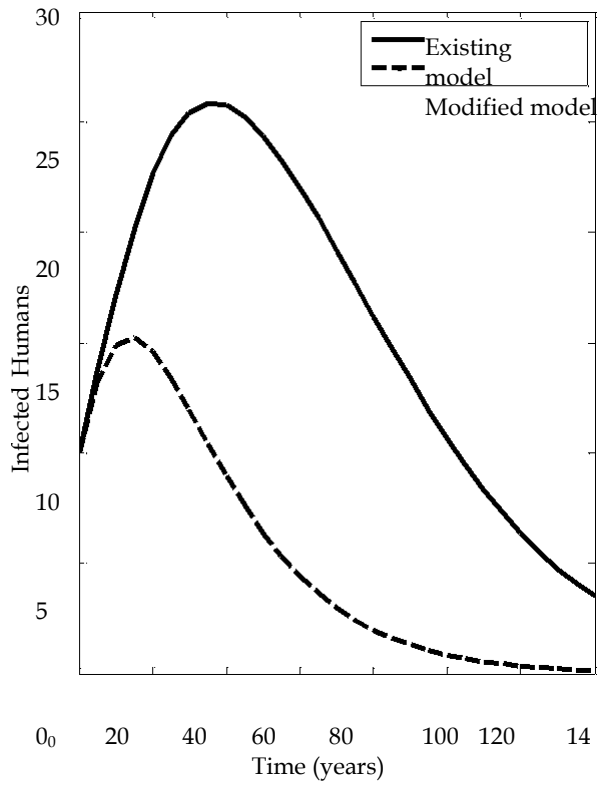

Fig. 7: The behavior of the models when e =0.5

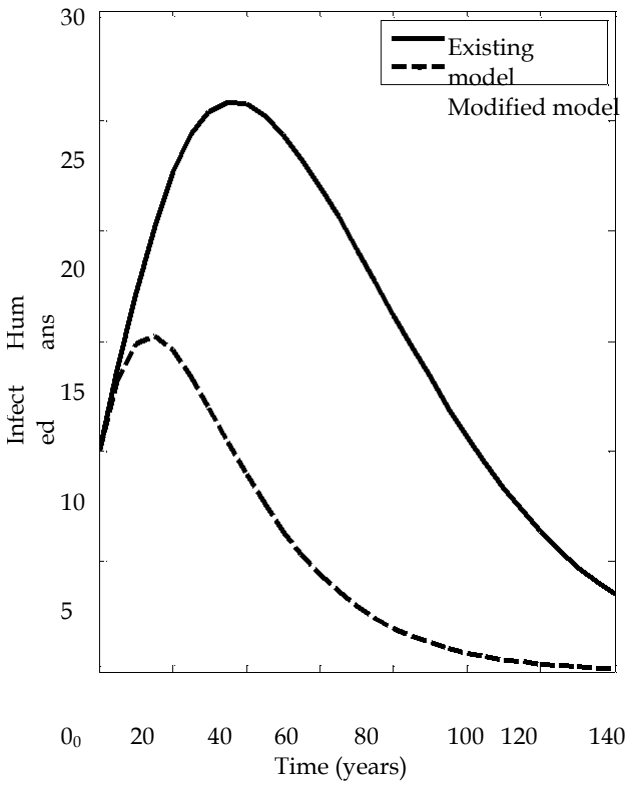

Fig. 8: The behavior of the models when e =0.7

Discussion

The disease-free equilibrium for the system was established. The next generation matrix method was used to derive an explicit formula the basic reproduction number R_0. Further studies was carried out using Routh-Hurwitz criteria and results showed that the disease-free equilibrium point is locally asymptotically stable when $R_0 < 1$, indicating that malaria eradication is possible within the population.

The numerical experiment showed that when the production of antibody is suspended in both susceptible humans and mosquitoes, and varying the rate of behavioral change results in further decrease in the population of susceptible, exposed, infectious and recovered humans respectively. On the other hand, there is an increase in the population of protected humans indicating the effect of preventing contact with mosquitoes and how trends improve in the population. Similarly, results indicate that when we combine the intervention of antibodies and behavioral change as illustrated in Figures 6-8, there is a greater improvement in controlling the spread of the disease in the entire human population compared to single intervention as illustrated in Figures 3-5. In other words, the combined intervention yields greater improvement in the population and hastens the time at which malaria is eradicated from the population.

Conclusion

This paper is an extension of Olaniyi and Obabiyi (2013) model for transmission dynamics of malaria where by education-based behavioral change, *e*, was incorporated leading to an additional compartment in the human population called, the protected humans. And this gave rise to an 8x8 system of non-linear ordinary differential equations. The disease-free equilibrium for the system was established. Both local stability analysis and numerical simulations of the system indicates that behavioral change will significantly improve the control and eradication of malaria when deployed with other control or preventive measures already being implemented to combat the disease. Furthermore, behavioral change will drastically reduce the disease burden in regions where the level of education is high unlike areas with poor education where the disease continues to thrive. Therefore, massive and continuous health education necessary for all members of communities invaded with infectious diseases such as malaria.

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