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Full Length Research Article

MODELLING THE EFFECT OF EDUCATION-BASED INTERVENTION IN THE CONTROL OF MALARIA

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ABSTRACT
In this study, we propose a mathematical model for the transmission dynamics of malaria by incorporating behavioural 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 obtained using the next generation matrix method. Result 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 behavioural change significantly alters the dynamics of malaria infection towards achieving a malaria-free society in finite time.

Keywords: Education, behavioural change, Disease-Free Equilibrium, Basic reproduction number, Local stability

INTRODUCTION

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 (WHO) 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 transmission dynamics of infectious diseases. From the year 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, 2007), (Tumwiine et al, 2008), (Peter, 2010), (Francis et al, 2012), (Adamu and Kimbir, 2013), (Xiongwei et al, 2014), (Bakare and Nwozo, 2015), (Adamu et al, 2015), (Sunisa et al, 2015), (Abadi and Harald, 2015), (Bakary 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. It has been observed 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 developed a mathematical model that incorporates an education-based behavioural change as an extension of (Olaniyi and Obabiyi, 2013) where the human population follows the susceptible-exposed-infectious-recovered (SEIR) pattern and the mosquito population follows susceptible-exposed-infectious (SEI) pattern. Hence, our aim is to determine the effect of behavioural change as a control strategy against the spread of malaria.

MATERIALS AND METHODS

The model comprises of two interacting (human and mosquito) populations as developed by (Olaniyi and Obabiyi, 2013). And a modified version is presented which incorporates an additional class of humans called, protected humans. Thus, the human population follows the susceptible-protected-exposed-infectious-recovered (SPEIR) pattern and the mosquito population follows susceptible-exposed-infectious (SEI) pattern.

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

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Table 2: Description of the parameters of the models

Parameter	Description
Λ_h	Recruitment rate into the susceptible humans
Λ_m	Recruitment rate into 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 capita death rate of humans
μ_m	Per capita death rate of mosquito
δ_h	Disease-induced death rate of humans
δ_m	Disease-induced death rate of mosquito
α_h	Per capita rate of progression of humans from exposed state to infectious state
α_m	Per capita rate of progression of mosquito from the exposed state to infectious
r	Per capita recovery rate for humans from the infectious state to the recovered state
ω	Per capita rate of loss of immunity in humans
ν_h	Proportion of antibody produced by humans in response to the incidence of infection caused by mosquitoes
ν_m	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

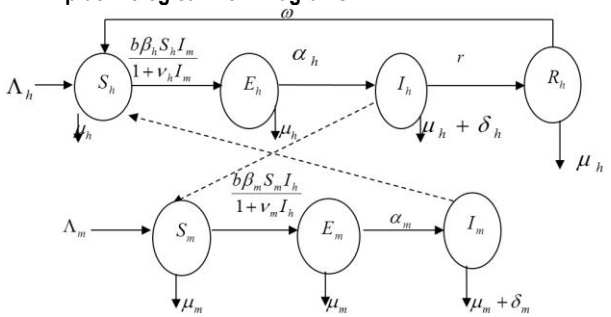

Figure 1: Epidemiological flow diagram for the existing model by (Olaniyi and Obabiyi, 2013)

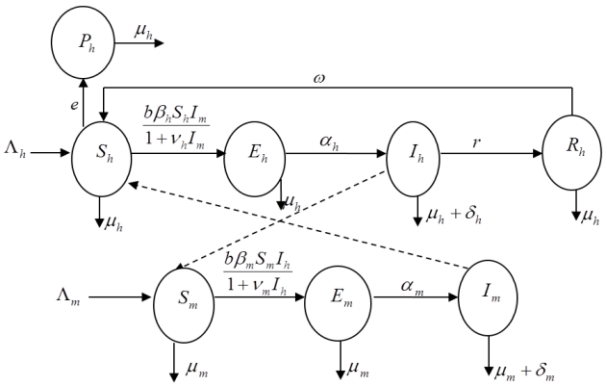

Figure 2: Epidemiological flow diagram of the model with behavioural change (modified model)

Model formulation

From the epidemiological flow diagram in Figure 1, Olaniyi and Obabiyi (2013) obtained the model equations (1) – (7) given below;

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

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

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

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

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

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

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

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, Figure 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 behavioural change. Thus, equation of the modified model for the transmission dynamics of the disease is given by the following system of ordinary differential equations

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

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

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

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

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

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

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

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

Together with the initial conditions:

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

RESULTS

Existence of Disease-free Equilibrium Point

The disease free equilibrium point E_0, of the system (8) - (15) is obtained by setting the right hand side of (8)-(15) equal to zero when $E_h = 0,\ I_h = 0,\ R_h = 0,\ E_m = 0,\ I_m = 0$ and is given by

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

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. (Diekmann et al, 1990), defined the basic reproduction number as the the expected number of secondary cases produced, in a completely susceptible population, by a typical infective individual. 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) and is given by

$$R_0 = \sqrt{\frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h + e)(\alpha_h + \mu_h)(r + \delta_h + \mu_h)(\alpha_m + \mu_m)(\mu_m + \delta_m)\mu_m}} \quad (16)$$

Local Stability of Disease-free Equilibrium

The basic reproduction number (16) is used to analyse 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:

$$J(E_0) = \begin{pmatrix} J_{11} & 0 & 0 & 0 & J_{15} & 0 & 0 & J_{18} \\ J_{21} & J_{22} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & J_{33} & 0 & 0 & 0 & 0 & J_{38} \\ 0 & 0 & J_{43} & J_{44} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & J_{54} & J_{55} & 0 & 0 & 0 \\ 0 & 0 & 0 & J_{64} & 0 & J_{66} & 0 & 0 \\ 0 & 0 & 0 & J_{74} & 0 & 0 & J_{77} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & J_{87} & J_{88} \end{pmatrix} \quad (17)$$

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

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

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

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

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

$$(\lambda+B_1)(\lambda+B_2)(\lambda+B_3)(\lambda+B_4) - \frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h+e)\mu_m} = 0 \quad (21)$$

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,

$$\begin{aligned} A_4 &= 1 \\ A_3 &= B_1+B_2+B_3+B_4 \\ A_2 &= (B_1+B_2)(B_3+B_4)+B_1B_2+B_3B_4 \\ A_1 &= (B_1+B_2)B_3B_4+(B_3+B_4)B_1B_2 \\ A_0 &= B_1B_2B_3B_4 - \frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m}{(\mu_h+e)\mu_m} \\ &= B_1B_2B_3B_4(1-R_0{}^2) \end{aligned} \quad (23)$$

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 $i=0,1,2,3,4$. 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,

$$H_1=A_3>.0$$

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

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

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 behaviour of the Olaniyi and Obabiyi (2013) model and the model with behavioural change given by the system (8)-(15) on the human populations.

Table 3: Parameter values and initial variables

Parameter	Experiment 1	2	3	4	5	6	Source
Λ_h	0.000215	0.000215	0.000215	0.000215	0.000215	0.000215	Olaniyi and Obabiyi (2013)
Λ_m	0.007	0.007	0.007	0.007	0.007	0.007	Olaniyi and Obabiyi (2013)
b	0.12	0.12	0.12	0.12	0.12	0.12	Olaniyi and Obabiyi (2013)
β_h	0.1	0.1	0.1	0.1	0.1	0.1	Olaniyi and Obabiyi (2013)
β_m	0.09	0.09	0.09	0.09	0.09	0.09	Olaniyi and Obabiyi (2013)
μ_h	0.0000548	0.0000548	0.0000548	0.0000548	0.0000548	0.0000548	Olaniyi and Obabiyi (2013)
μ_m	1/15	1/15	1/15	1/15	1/15	1/15	Olaniyi and Obabiyi (2013)
δ_h	0.001	0.001	0.001	0.001	0.001	0.001	Olaniyi and Obabiyi (2013)
δ_m	0.01	0.01	0.01	0.01	0.01	0.01	Olaniyi and Obabiyi (2013)
α_h	1/17	1/17	1/17	1/17	1/17	1/17	Olaniyi and Obabiyi (2013)
α_m	1/18	1/18	1/18	1/18	1/18	1/18	Olaniyi and Obabiyi (2013)
r	0.05	0.05	0.05	0.05	0.05	0.05	Olaniyi and Obabiyi (2013)
ω	1/730	1/730	1/730	1/730	1/730	1/730	Olaniyi and Obabiyi (2013)
v_h	0	0	0	0.5	0.5	0.5	Olaniyi and Obabiyi (2013)
v_m	0	0	0	0.5	0.5	0.5	Olaniyi and Obabiyi (2013)
E	0.25	0.50	0.75	0.25	0.50	0.75	Assumed
$S_h(0)$	100	100	100	100	100	100	Olaniyi and Obabiyi (2013)
$E_h(0)$	20	20	20	20	20	20	Olaniyi and Obabiyi (2013)
$I_h(0)$	10	10	10	10	10	10	Olaniyi and Obabiyi (2013)
$R_h(0)$	0	0	0	0	0	0	Olaniyi and Obabiyi (2013)
$S_m(0)$	1000	1000	1000	1000	1000	1000	Olaniyi and Obabiyi (2013)
$E_m(0)$	20	20	20	20	20	20	Olaniyi and Obabiyi (2013)
$I_m(0)$	30	30	30	30	30	30	Olaniyi and 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 behavioural change. Figures 3, 4, and 5 show the varying effect of behavioural 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 behavioural change of protected humans as shown in Figures 6, 7, and 8

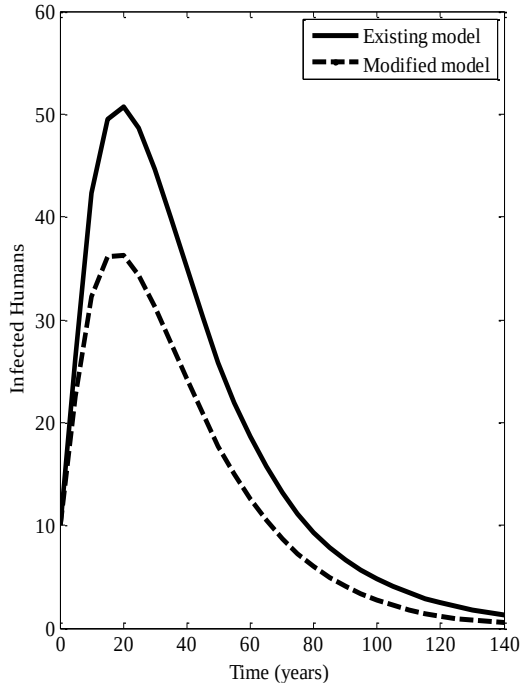

Figure 3: The behaviour of the models when $e = 0.25$

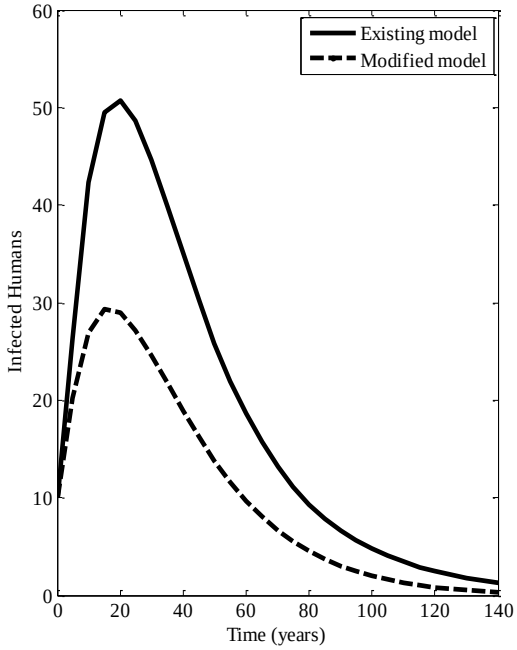

Figure 4: The behaviour of the models when $e = 0.5$

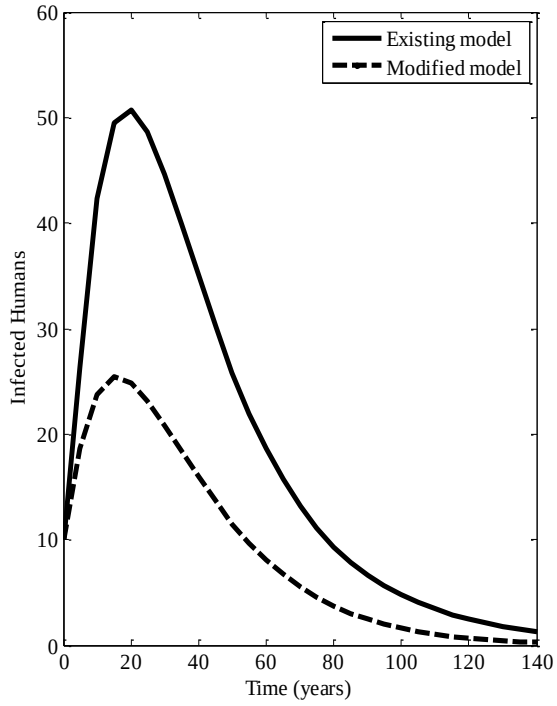

Figure 5: The behaviour of the models when $e = 0.75$

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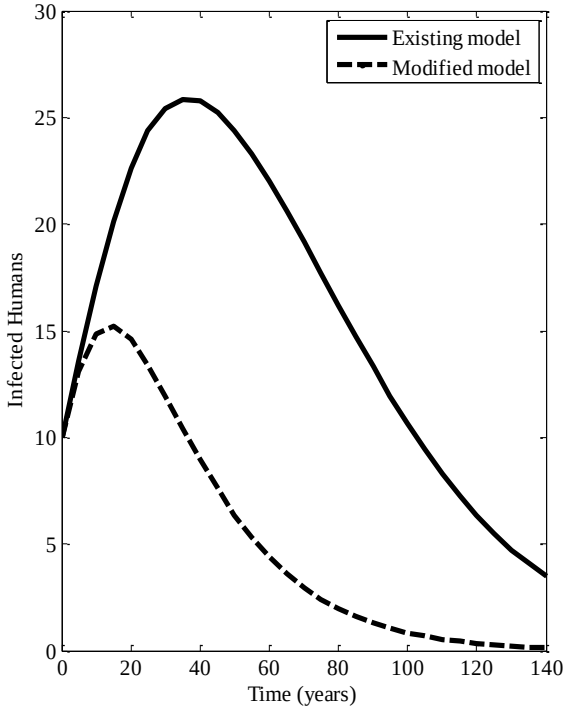

Figure 6: The behaviour of the models when $e = 0.25$

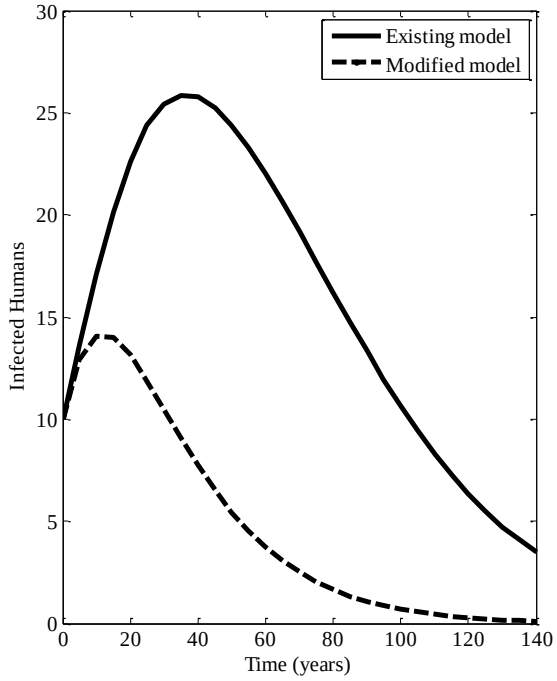

Figure. 7: The behaviour of the models when $e = 0.5$

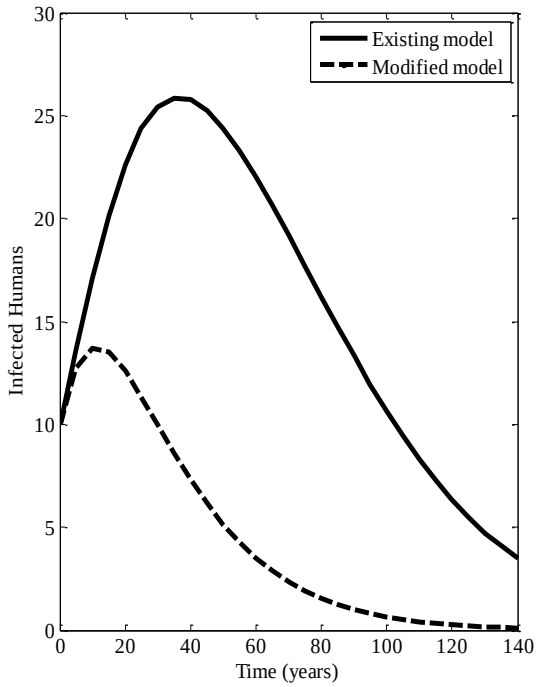

Figure. 8: The behaviour of the models when $e = 0.75$

DISCUSSION

The disease-free equilibrium for the system (8)-(15) 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 behavioural change results in further decrease in the population of infectious humans as shown in Figures 3–5. Similarly, results indicate that when we combine the intervention of antibodies and behavioural 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

Incorporating an education-based intervention strategy into the model due to (Olaniyi and Obabiyi, 2013) yields better performance of the modified model. Stability analysis of the modified model revealed an asymptotically stable Disease free equilibrium under specified conditions. Malaria can be eradicated in finite time when $R_0 < 1$. Otherwise, it persists in the population.

Furthermore, behavioural 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 is

highly recommended as an intervention alongside other forms of intervention for all members of communities invaded with malaria.

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