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Sensitivity Analysis of Mathematical Model for Malaria Transmission with Saturated Incidence Rate

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Authors' contributions

This work was carried out in collaboration between all authors. Author MARENO designed the study, performed the analysis, wrote the protocol and wrote the first draft of the manuscript. Author AE performed the analysis, managed the analyses of the study and assisted in writing of the draft. Author NAH reviewed the language of the final draft. Author CY managed the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

Malaria is a life threatening vector borne disease caused by parasites that are transmitted to people through the bites of infected female *Anopheles* mosquitoes. In this paper, we study and analyze mathematical model of ordinary differential equations for human and mosquito with saturated incidence function. The stability of the system was analyzed for the Malaria-Free Equilibrium (MFE) through the reproduction number $\mathcal{R}_0$ which was obtained using the next generation matrix method. The MFE is locally asymptotical stable if $\mathcal{R}_0 < 1$ and unstable otherwise. Moreover, our sensitivity analysis shows that the most effective parameter is, a, mosquito biting rate and the less effective one is α_h, human progression rate. Our numerical simulations show that, reducing the biting rate of mosquitoes will reduce the number of exposed humans as well as infected individuals and increase the number of treated individuals. This can be achieved by increasing the proportion of antibodies.

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1 Introduction

According to [1], Malaria is a life-threatening disease caused by parasites that are transmitted to people through the bites of infected female Anopheles mosquitoes. It is preventable and curable. In 2016, there were an estimated 216 million cases of malaria in 91 countries, an increase of 5 million cases over 2015. Malaria deaths reached 445000 in 2016. The total funding for malaria control and elimination reached an estimated US 2.7 billion in 2015. Nearly half of the world's population is at risk of the malaria disease with most of the malaria cases and deaths occurring in the sub-saharan Africa. The female infected mosquitoes carry a parasite called Plasmodium. The mosquitoes take the blood meal from human which is needed for their egg production and such blood meals are the link between the human and the mosquito host in the parasite life [2]. There are four common species of plasmodium that cause malaria in humans which include;*Plasmodium falciparum, Plasmodium vivax, Plasmodium malariae*, and *Plasmodium ovale*. Recently, some human cases of malaria have also occurred with *Plasmodium knowlesi*, which is a species that infects animals. Among the species, *Plasmodium falciparum* is the most deadly and accounts for 80 percent of malaria cases and 90 percent of death [3, 4].

The use of mathematical modeling has played a unique role in comparing the effects of control strategies, used individually or in packages [5]. It can also be used to project how infectious diseases progress, to show the likely outcome of an epidemic, and help inform public health interventions [6]. Understanding the epidemiology of emerging and re-emerging of infectious diseases in a population produces a healthy environment for living. Mathematical models are used in likening, designing, implementing, evaluating and optimizing several detection, prevention and control plans [7].

Mathematical modelling of malaria has flourished since the days of Ross [8], who was the first to model the dynamics of malaria transmission and Macdonald [9, 10, 11] who expounded on Ross's work, introducing the theory of superinfection. In the work of Chitnis et al. [5], they perform the sensitivity analysis on a mathematical model of malaria transmission to determine the relative importance of the model parameters to disease transmission and prevalence. They also studied the sensitivity indices of the reproduction number and the endemic equilibrium point to the parameters at the baseline value. In a Ph.D. dissertation, Chitnis [12] described a compartmental model for malaria transmission, based on a model by Nqwa and Shun [13]. He defined a reproductive number, $\mathcal{R}_0$, as the expected number of secondary cases that one infected individual would cause through the duration of the infectious period. Also he showed the existence and stability of the disease free and endemic equilibrium points. He also computed the sensitivity indices of $\mathcal{R}_0$ and the endemic equilibrium to the parameters in the model.

There has been a high incidence and prevalence of malaria in the last few decades due to increasing parasite drug-resistance and mosquito insecticide-resistance.This calls for a comparative knowledge of the effectiveness and efficacy of different control strategies which are useful and cost-effective in the malaria control programs. It is from this background that we developed a vector-host mathematical model for the transmission dynamics of malaria to examine the sensitive parameters that play vital roles in the dynamical spread and control of the disease. The paper is organized as follows: In Section 2, we describe the formulation of the model. Section 3 is devoted for the analysis of the model; the basic reproduction number is also computed. The stability of the disease free equilibrium is investigated as well as the existence of the endemic equilibrium. Section 4 has the sensitivity analysis of the basic reproduction number. Section 5 is devoted for the numerical simulations . The conclusion is discussed in Section 6.

2 Mathematical Model

2.1 Model description

The total population size, N_h, of the human population is sub-divided into five classes namely Susceptible human,S_h, Exposed human, E_h, Infected human, I_h, Treated human, T_h, and Recovered human, R_h, so that

$$N_h = S_h + E_h + I_h + T_h + R_h \tag{2.1}$$

The mosquitoes population N_m is also sub-divided into three classes namely Susceptible mosquitoes S_m, Exposed mosquitoes E_m and Infected mosquitoes I_m, so that

$$N_m = S_m + E_m + I_m \tag{2.2}$$

We assume that susceptible human population increases by birth or immigration at the rate Λ_h, human can die at any stage by natural causes and mosquitoes do not recover from infection. Also the infected humans after treatment move from infected to the treatment class for treatment. With a biting rate of a, there is an infection from the infected human to a susceptible mosquito at a rate of β_{hm}. The recovered humans becomes susceptible to the disease after they have been fully recovered at a rate ρ. The susceptible human population is reduced by a natural death rate of μ. The class E_h of exposed humans is generated after the mosquito bites a susceptible human at a rate a. At this stage, individuals do not show any signs and symptoms of malaria. The exposed human class is reduced by a rate α_h, which is the human progression rate from the exposed human to the Infected human class and also by a natural death rate of μ. When the exposed humans start showing signs of malaria, they leave the expose class and join the infected class at a rate of α_h. After treatment, a rate γ, leaves the infected class to join the treated class. The infected class is further decreased by a disease induced death rate δ_h and a natural death rate of μ. A rate σ leaves the treatment class to the recovery class after they have fully recovered. The class is further decreased by a natural death rate of μ. A class of recovered human, R_h, is generated when the infected human respond fully to the treatment given to them. There is a natural death rate of μ. Also a rate ρ of the recovered humans joins the susceptible class again.

In the case of the mosquitoes, there is a recruitment of Λ_m, into the susceptible class. With a biting rate of a, the infected mosquito transfer the plasmodium parasite to the susceptible human at a rate of β_{mh}. The susceptible mosquitoes also decreases by a natural death rate of η. A class of exposed mosquito is generated after they have bitten an infected human. A rate α_m leaves the exposed class of mosquitoes to the infected class of mosquitoes. There is a natural death rate of η. After the mosquitoes leaves the exposed class, a class of Infected mosquitoes is formed. A rate α_m leaves the expose class to join the infected class. The infected mosquitoes are reduced by a disease induce and natural death rate of δ_m and η respectively.

With the above formulations and assumptions, we have the following system of differential equations:

$$\begin{cases} \frac{dS_h}{dt} = \Lambda_h - \frac{a\beta_{hm}S_hI_m}{1+v_hI_m} + \rho R_h - \mu S_h, \\ \frac{dE_h}{dt} = \frac{a\beta_{hm}S_hI_m}{1+v_hI_m} - (\alpha_h + \mu)E_h, \\ \frac{dI_h}{dt} = \alpha_h E_h - (\gamma + \mu + \delta_h)I_h, \\ \frac{dT_h}{dt} = \gamma I_h - (\mu + \sigma)T_h, \\ \frac{dR_h}{dt} = \sigma T_h - (\mu + \rho)R_h, \\ \frac{dS_m}{dt} = \Lambda_m - \frac{a\beta_{mh}S_mI_h}{1+v_mI_h} - \eta S_m, \\ \frac{dE_m}{dt} = \frac{a\beta_{mh}S_mI_h}{1+v_mI_h} - (\eta + \alpha_m)E_m, \\ \frac{dI_m}{dt} = \alpha_m E_m - (\eta + \delta_m)I_m, \end{cases} \tag{2.3}$$

With the initial conditions: $S_h(0) > 0, E_h(0) \geq 0, I_h(0) \geq 0, T_h(0) \geq 0, R_h(0) \geq 0, S_m(0) > 0, E_m(0) \geq 0, I_m(0) \geq 0$.

3 Model Analysis

3.1 Positivity and boundedness of the solutions

Since the model (2.3) characterizes interaction between host (human) and vector (mosquito) populations, it is important to state that all the model variables and parameters are non-negative with respect to time, thus $t \geq 0$. The system (2.3) will be considered in the epidemiologically- feasible region $\Omega = \Omega_h \times \Omega_m \subset \mathbb{R}^5_+ \times \mathbb{R}^3_+$ with,

$$\Omega_h = \{S_h, E_h, I_h, T_h, R_h \in \mathbb{R}^5_+ : N_h \leq \frac{\Lambda_h}{\mu}\}, \tag{3.1}$$

and

$$\Omega_m = \{S_m, E_m, I_m \in \mathbb{R}^3_+ : N_m \leq \frac{\Lambda_m}{\eta}\}, \tag{3.2}$$

It can be shown that the region Ω is a positively invariant set and global attractive of the system (2.3), this means any trajectory indicated any where in the non-negative region $\mathbb{R}^8_+$ of the phase space ultimately enters the feasible region Ω and remains in Ω thereafter.

Lemma 3.1. The region

$$\Omega_h = \{S_h, E_h, I_h, T_h, R_h, S_m, E_m, I_m \in \mathbb{R}^8_+ : N_h \leq \frac{\Lambda_h}{\mu}, N_m \leq \frac{\Lambda_m}{\eta}\},$$

is positively invariant region for the model (2.3)

Proof.

$$\begin{aligned} &\tfrac{dN_h}{dt} = \Lambda_h - \mu N_h - \delta_h I_h, \\ &\tfrac{dN_h}{dt} \leq \Lambda_h - \mu N_h, \\ &\lim_{t \longrightarrow \infty} N_h(t) \leq \tfrac{\Lambda_h}{\mu}, \end{aligned} \tag{3.3}$$

$$\begin{aligned} &\tfrac{dN_m}{dt} = \Lambda_m - \eta N_m - \delta_m I_m, \\ &\tfrac{dN_m}{dt} \leq \Lambda_m - \eta N_m, \\ &\lim_{t \longrightarrow \infty} N_m(t) \leq \tfrac{\Lambda_m}{\eta}, \end{aligned} \tag{3.4}$$

□

3.2 Reproduction number and existence of equilibrium

The malaria-free equilibrium (MFE) is a point at which the population is free from the malaria disease. The MFE of the system (2.3) is denoted by P_0 and is given by
$P_0 = (S_{h0}, E_{h0}, I_{h0}, T_{h0}, R_{h0}, S_{m0}, E_{m0}, I_{m0}) = (S_{h0}, 0, 0, 0, 0, S_{m0}, 0, 0) = (\frac{\Lambda_h}{\mu}, 0, 0, 0, 0, \frac{\Lambda_m}{\eta}, 0, 0)$
Let

$$\mathcal{F} = \begin{bmatrix} \frac{a\beta_{hm} S_h I_m}{1+v_m I_m} \\ 0 \\ \frac{a\beta_{mh} S_m I_h}{1+v_m I_h} \\ 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} (\alpha_h + \mu) E_h \\ -\alpha_h E_h + (\gamma + \mu + \delta_h) I_h \\ (\eta + \alpha_m) E_m \\ -\alpha_m E_m + (\eta + \delta_m) I_m \end{bmatrix}.$$

by differentiating $\mathcal{F}$ and $\mathcal{V}$ partially with respect to: E_h, I_h, E_m and I_m at MFE P_0, we get,

$$f = D[\mathcal{F}(P_0)] = \begin{bmatrix} 0 & 0 & 0 & \frac{a\beta_{hm}\Lambda_h}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{a\beta_{mh}\Lambda_m}{\eta} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, v = D[\mathcal{V}(P_0)] = \begin{bmatrix} \alpha_h+\mu & 0 & 0 & 0 \\ -\alpha_h & \gamma+\mu+\delta_h & 0 & 0 \\ 0 & 0 & \eta+\alpha_m & 0 \\ 0 & 0 & -\alpha_m & \eta+\delta_m \end{bmatrix}$$

$$fv^{-1} = \begin{bmatrix} 0 & 0 & 0 & \frac{a\beta_{hm}\Lambda_h}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{a\beta_{mh}\Lambda_m}{\eta} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{(\alpha_h+\mu)} & 0 & 0 & 0 \\ \frac{\alpha_h}{(\alpha_h+\mu)(\gamma+\mu+\delta_h)} & \frac{1}{(\gamma+\mu+\delta_h)} & 0 & 0 \\ 0 & 0 & (\eta+\alpha_m) & 0 \\ 0 & 0 & \frac{\alpha_m}{(\eta+\alpha_m)(\eta+\delta_m)} & \frac{1}{(\eta+\alpha_m)} \end{bmatrix}.$$

$$= \begin{bmatrix} 0 & 0 & \frac{a\beta_{hm}\Lambda_h\alpha_m}{\mu(\eta+\alpha_m)(\eta+\delta_m)} & \frac{a\beta_{hm}\Lambda_h}{(\eta+\delta_m)} \\ 0 & 0 & 0 & 0 \\ \frac{a\beta_{mh}\Lambda_m\alpha_h}{\eta(\alpha_h+\mu)(\gamma+\mu+\delta_h)} & \frac{a\beta_{mh}\Lambda_m}{\eta(\gamma+\mu+\delta_h)} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Then $\mathcal{R}_0$, is given by spectral radius of fv^{-1} which is denoted by $\rho(fv^{-1})$ and defined as:

$$\mathcal{R}_0 = \rho(fv^{-1}) = \sqrt{\frac{a^2\beta_{hm}\beta_{mh}\Lambda_h\Lambda_m\alpha_h\alpha_m}{\mu\eta(\alpha_h+\mu)(\gamma+\mu+\delta_h)(\eta+\alpha_m)(\eta+\delta_m)}}, \tag{3.5}$$

3.2.1 Stability of the Malaria-Free Equilibrium

In this subsection, we investigate the stability of the MFE P_0, by evaluating the Jacobian matrix of system (2.3) at $P_0 = (\frac{\Lambda_h}{\mu}, 0, 0, 0, 0, \frac{\Lambda_m}{\eta}, 0, 0)$ and obtained

$$J(P_0) =$$

$$\begin{bmatrix} -\mu & 0 & 0 & 0 & \rho & 0 & 0 & -\frac{a\beta_{hm}\Lambda_h}{\mu} \\ 0 & -(\alpha_h+\mu) & 0 & 0 & 0 & 0 & 0 & \frac{a\beta_{hm}\Lambda_h}{\mu} \\ 0 & \alpha_h & -(\gamma+\mu+\delta_h) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma & -(\mu+\sigma) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma & -(\mu+\rho) & 0 & 0 & 0 \\ 0 & 0 & -\frac{a\beta_{mh}\Lambda_m}{\eta} & 0 & 0 & -\eta & 0 & 0 \\ 0 & 0 & \frac{a\beta_{mh}\Lambda_m}{\eta} & 0 & 0 & 0 & -(\alpha_m+\eta) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m & -(\delta_m+\eta) \end{bmatrix}$$

It is clear that $\lambda_1 = -\mu$,$\lambda_2 = -\eta$, $\lambda_3 = -(\mu+\sigma)$, $\lambda_4 = -(\mu+\rho)$ are negative eigenvalues and the sign of the other eigenvalues can be determined by the equation

$$G(\lambda) = c_4\lambda^4 + c_3\lambda^3 + c_2\lambda^2 + c_1\lambda + c_0 = 0 \tag{3.6}$$

where:

$$\begin{aligned} c_4 &= 1, \\ c_3 &= k_1 + k_2 + k_5 + k_6, \\ c_2 &= k_1k_2 + k_1k_5 + k_1k_6 + k_2k_5 + k_2k_6 + k_5k_6, \\ c_1 &= k_1k_2k_5 + k_1k_2k_6 + k_1k_5k_6 + k_2k_5k_6, \\ c_0 &= k_1k_2k_5k_6(1-\mathcal{R}_0^2), \end{aligned}$$

such that: $k_1 = (\alpha_h + \mu), k_2 = (\gamma + \mu + \delta_h), k_3 = (\mu + \sigma), k_4 = (\mu + \rho), k_5 = (\alpha_m + \eta)$, $k_6 = (\delta_m + \eta)$. Clearly it can be seen that all the roots of equation (3.6) have negative real parts, by applying the Routh-Hurwitz Criterion if and only if the factors c_i, are positive for $i = 0, 1, 2, 3, 4$ and the determinants $D_i > 0$, for $i = 1, 2, 3, 4$. From (3.6) clearly $c_1 > 0, c_2 > 0, c_3 > 0, c_4 > 0$. Moreover,if $\mathcal{R}_0 < 1$ then $c_0 > 0$. Also

$$D_1 = c_3 > 0, D_2 = \begin{vmatrix} c_3 & c_4 \\ c_1 & c_2 \end{vmatrix} > 0, D_3 = \begin{vmatrix} c_3 & c_4 & c_0 \\ c_1 & c_2 & c_3 \\ 0 & c_0 & c_1 \end{vmatrix} > 0, D_4 = \begin{vmatrix} c_3 & c_4 & 0 & 0 \\ c_1 & c_2 & c_3 & c_4 \\ 0 & c_0 & c_1 & c_2 \\ 0 & 0 & 0 & c_0 \end{vmatrix} > 0, \tag{3.7}$$

Thus, all the eigenvalues of $J(P_0)$ have negative real parts whenever $\mathcal{R}_0 < 1$, and P_0 is said to be locally asymptotically stable. However, if $\mathcal{R}_0 > 1$ then $c_0 < 0$ and by Descartes rule of signs [14, 15], there exist exactly one sign change in c_4, c_3, c_2, c_1, c_0 of factors of the equation (3.6). So, there is one eigenvalue with non-negative real part then the MFE P_0 is unstable when $\mathcal{R}_0 > 1$, which indicates an existence of an endemic equilibrium.

Theorem 3.2. System (2.3) has the MFE point P_0 if $\mathcal{R}_0 < 1$,which is locally asymptotically stable and unstable if $\mathcal{R}_0 > 1$.

4 Sensitivity Analysis

Table 1: Description and values of parameters of the model (2.3).

Parameter	Parameter Description	Value	References
Λ_h	Human recruitment rate	0.0250	Assumed
Λ_m	Mosquito recruitment rate	0.035	Assumed
v_h	Proportion of antibody produced by human	0.29	Assumed
v_m	Proportion of antibody produced by mosquito	0.21	Assumed
γ	treatment rate of the infectious individual	0.14	Assumed
μ	Human natural death rate	4.74×10^{-5}	Assumed
δ_h	Disease induced death rate of human	0.001	[14]
δ_m	Disease induced death rate of mosquito	0.01	[14]
α_h	Human progression rate from E_h to I_h	0.08333	[15]
α_m	Mosquito progression rate from E_m to I_m	0.48	[15]
σ	Recovery rate through the treatment	3.5×10^{-3}	[16]
β_{hm}	Transmission rate from infected human to susceptible mosquito	0.48	[16]
β_{mh}	Transmission rate from infected mosquito to susceptible human	0.048	[16]
a	Mosquito biting rate	0.33	[17]
η	Mosquito natural death rate	0.1	[17]
ρ	Loss of immunity rate	2.74×10^{-3}	[17]

Sensitivity indices permit us to measure the proportional change in a state variable when a parameter changes. Normally the sensitivity analysis of the model is determined by using the partial derivatives of the outcome with respect to it's parameters.

Definition: The normalized forward sensitivity of index for a variable y, which depends on a parameter q, denoted by γ_q^y is defined as

$$\gamma_q^y = \frac{\partial y}{\partial q} \times \frac{q}{y},$$

We consider that change of the state variable parallels with a change in the value of $\mathcal{R}_0$ in our model simulation. Since the reproduction number is a function of the parameters, then we can evaluate the relative sensitivity of $\mathcal{R}_0$ for every parameter that $\mathcal{R}_0$ depends on.The parameters μ and η are simply not considered because they are natural death rate of humans and mosquitoes respectively.

In order to determine how to reduce human morbidness due to malaria it is better to know the relative importance of different factors that changes the spread of the disease. The parameters are listed in Table 2 in such a way that they begin from the most sensitive to the least sensitive one.The signs on sensitivity indices indicate the direction of the change for each parameter.The most sensitive parameter is a the mosquitoes biting rate and the least sensitive one is α_h, which is human progression rate from E_h to I_h.

(i) if we decrease the value of a from 0.33 to 0.25 and the other parameter values remain the same then the value of $\mathcal{R}_0$ is reduces from 0.0137 to 0.0104.
(ii) if we reduce the value of β_{hm} from 0.48 to 0.35 and the other parameters remain the same then the value of $\mathcal{R}_0$ reduces from 0.0137 to 0.01168
(iii) if we reduce the value of β_{hm} from 0.048 to 0.035 and the other parameters remains the same then the value of $\mathcal{R}_0$ reduces from 0.0137 to 0.0117

Table 2: Sensitivity indices of $\mathcal{R}_0$ to parameters for model (2.3)

	Parameter	Sensitivity index
1	a	+1
2	β_{hm}	+0.5
3	β_{mh}	+0.5
4	Λ_h	+0.5
5	Λ_m	+0.5
6	μ	-0.5
7	γ	-0.4963
8	α_m	+ 0.0862
9	δ_m	-0.0455
10	δ_h	-0.0035
11	α_h	0.000028435

5 Numerical Simulations

In this section, we study the numerical simulations of our model. The graphs in Fig 1 show the simulations of malaria model showing the varying effect of the ratio of antibodies, v_h on the human population. Fig 1(a) indicates that an increase in the antibody in human greatly increases the number of susceptible human. In the case of the exposed human, increasing the antibody sharply reduces the number of exposed human as depicted in Fig.1(b). Also, an increase in the antibody increases the number of infected humans and vice verse, this is shown in Fig 1(c). Fig 1(d) shows that when the antibody is increased, it doesn't show any initial changes until after day 10, after which the number of treated humans reduces drastically. Fig 2 shows the simulation of malaria model showing the varying effect of the ratio of antibody v_m on the mosquitoes population S_m. From Fig 2(a), we clearly see that an increase or decrease in the antibody does not affect the susceptible mosquitoes. However, Fig 2(b) indicates that the number of magnitude of the exposed mosquitoes decreases as the antibody increases. Thus, increasing the antibodies reduces the expose mosquito population depicted in Fig 2(c). In general, it is observed that an increase in antibody greatly reduces the infected mosquitoes.

Fig.3 shows the simulations of malaria model showing the effect of vector biting rate a on the susceptible and infected human. It is clearly seen from Fig 3(a) that as the biting rate of mosquitoes increases, the population of the susceptible humans reduces drastically. On the other hand, as the biting rate a of the mosquitoes increase, the number of infected human also increases.

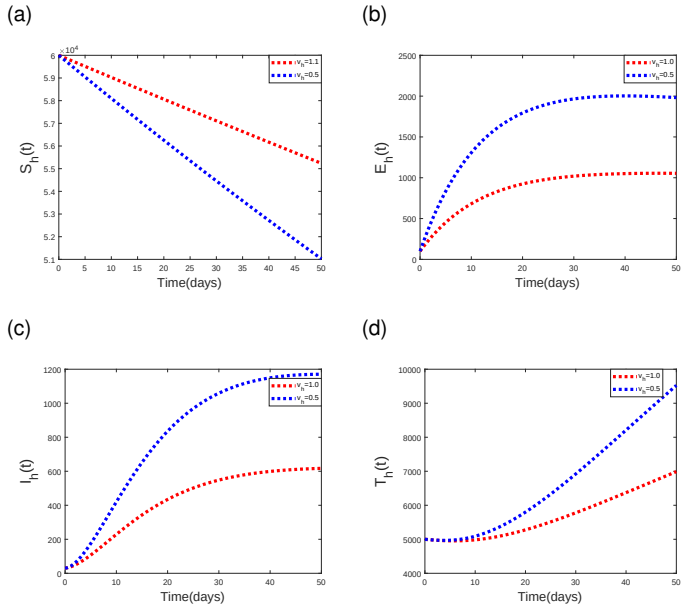

Figure 1: Simulations of malaria model (2.3) showing the varying effect of the ratio of antibody v_h on the human population $S_h(t)$ Fig 1 (a),$E_h(t)$ Fig 1 (b) $I_h(t)$ Fig 1 (c) and $T_h(t)$ Fig 1 (d). when $R_0 < 1$. All Parameter values used are listed in Table 1

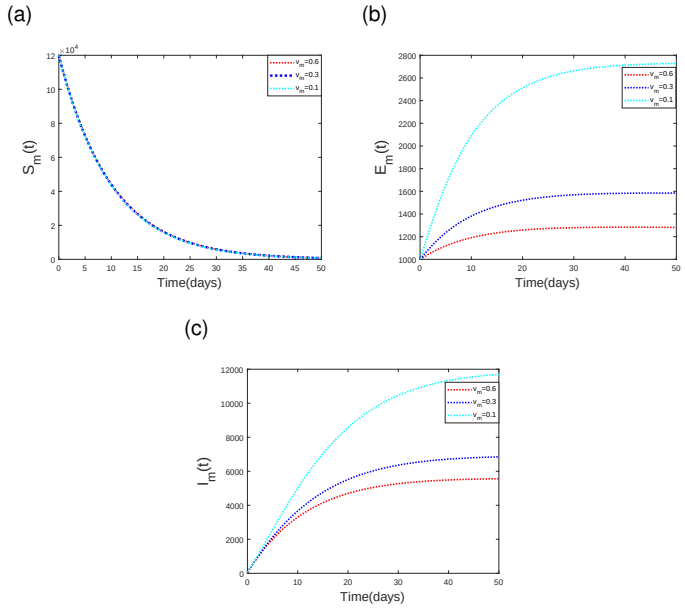

Figure 2: Simulations of malaria model (2.3) showing the varying effect of the ratio of antibody v_m on the mosquitoes population $S_m(t)$ Fig (a),$E_m(t)$ Fig 2 (b) and $I_m(t)$ Fig 2 (c), when $R_0 < 1$. All Parameter values used are listed in Table 1

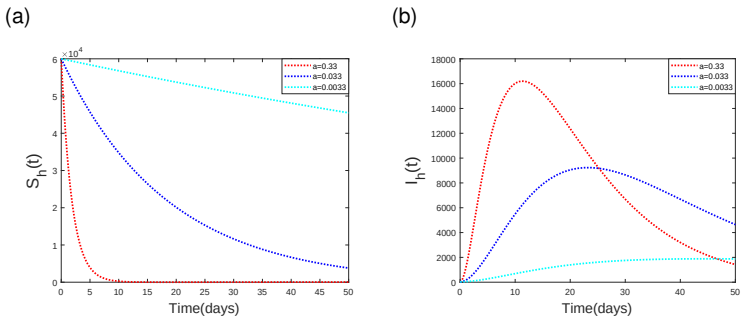

Figure 3: Simulations of malaria model (2.3) showing the effect of vector biting rate a on the human population $S_h(t)$ Fig 3 (a) and infected human $I_h(t)$. Fig 3 (b), as a function of time. Parameter values used are listed in Table 1

6 Conclusion

The sensitivity analysis of the ordinary differential equations model of malaria transmission with saturated incidence function was studied. Basic properties of the model were discussed. The malaria-free equilibrium, P_0 was shown to be locally asymptotically stable whenever $\mathcal{R}_0 < 1$. Our sensitivity analysis shows that the most effective parameter is, a, mosquito biting rate and the less effective one is, α_h, human progression rate from E_h to I_h. Furthermore, our numerical simulations showed that increasing the antibodies is the best strategy to reduce the number of exposed humans and infected individuals, which increases the number of treated humans and controlls the disease.

Competing Interests

Authors have declared that no competing interests exist.

References

[1] Malaria, Key facts.
Available: https://www.who.int/news-room/fact-sheets/detail/malaria.

[2] Centre for Disease Control and prevention (CDC), Anopheles mosquitoes.
Available:http://www.cdc.gov/malaria/about/biology/mosquitoes/index.html

[3] WHO: Malaria, International travel and health.
Available:www.int/ith/disease/ malaria/en.

[4] Flahault A, Le Menach A, Mekenzie EF, Smith DL. The unexpected importance of mosquito oviposition behaviour for malaria: non productive larva habitats can be sources for malaria transmission. Malaria Journal. 2005;4.

[5] Chitnis N, Hyman JM, Cushing JM. Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. Bulletin of Mathematical Biology; 2008. DOI:10.1007/s11538-008-9299-0

[6] Rosa S, Torres DFM. Parameter estimation, sensitivity analysis and optimal control of a periodic epidemic model with application to HRSV in Florida. Stat. Optim. Inf. Comput. 2018;6:139-149.

[7] Ojo MM, Gbadamosi B, Olukayode A, Oluwaseun OR. Sensitivity analysis of dengue model with saturated incidence rate. Open Access Library Journal. 2018;5:e4413. DOI:https://doi.org/10.4236/oalib.1104413

[8] Ross R. The prevention of malaria, 2 edn. Murray, London; 1911.

[9] Macdonald G. The analysis of infection rates in diseases in which super infection occurs. Trop. Dis. Bull. 1950;47;907-915.

[10] Macdonald G. The analysis of sporozoite rate, Trop. Dis Bull. 1952;49:569-585.

[11] Macdonald G. The epidemiology and control of malaria. Oxford University Press, London; 1957.

[12] Chitnis N. Using mathematical models in controlling the spread of malaria. Ph.D. thesis, University of Arizona, Tucson, Arizona, USA; 2005.

[13] Ngwa GA, Shu WS. A mathematical model for the endemic malaria with variable human and mosquito population. Math. Comput. Model. 2000;32:747-763.

[14] Olaniyi S, Obabiyi OS. Mathematical model for malaria transmission dynamics in human and mosquito populations with nonlinear forces of infection. International Journal of Pure and Applied Mathematics. 2013;88:125-156.

[15] Vanden Driessche P, Watmough J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Mathematical Biosciences. 2002;180:29-48.

[16] Chitnis N, Hyman J, Cushing J. Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. Bull. Math. Biol. 2008;70:1272-1296.

[17] CDC. Anopheles mosquitoes. Centers for Disease Control and Prevention; 2015.

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