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DETERMINING SENSITIVE PARAMETERS IN THE TRANSMISSION DYNAMIC S OF MALARIA DISEASE USING MATHEMATICAL MODEL APPROACH

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ABSTRACT: *The challenge posed by malaria disease calls for an urgent need for a better understanding of important parameters in the disease transmission and development of prevention and control of the spread of malaria disease. In this work, a mathematical model for the dynamics of malaria disease is developed and analyzed. There is existence of disease free equilibrium and endemic equilibrium point of the model, the local stability of disease free equilibrium is obtained using Jacobian matrix which is locally asymptotically stable whenever the basic reproduction number is less than unity. Finally, the results obtained in Table 2, Figure 6 and Figure 8 from sensitivity analysis reveal that malaria disease can be controlled if the biting rate of mosquito is eliminated in the population.*

KEYWORDS: Mathematical model, Disease free equilibrium, Basic reproduction number, Sensitivity, Positivity, Boundedness of solution.

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INTRODUCTION

Malaria disease occurs due to the bites of female anopheles mosquitoes. Plasmodium parasite is transmitted into the human body when an infected female anopheles mosquito makes bites. Plasmodium parasites making the human liver as their home multiply their population and start infecting red blood cells of the human. A variety of plasmodium parasites exist. Mainly four types of plasmodium cause malaria disease among the human viz., falciparum, vivax, ovale and plasmodium malaria [1,11,12,13,14,19]. [17,22,23,24,25,26] developed a mathematical model to study the transmission of malaria; in fact, their study showed clearly that effective treatment offered to about fifty percent of the infected population together with about fifty percent prevention rate is all that is required to eliminate the disease. [1,9] developed a model for the dynamic spread of malaria where humans and vectors interact and infect each other. Aftermath, the model was analyzed to gain more insight into the dynamics of the spread of malaria in the human and vector population. Also, the numerical analysis of the model shows that the most effective strategies for controlling malaria is to reduce the vector biting rate and increase human treatment. [16] study a mathematical model of Ebola virus disease, it was shown that the model was well- posed; both disease and endemic equilibria for the models were obtained. Their results obtained so far from sensitivity analysis strongly shows that the spread of Ebola virus disease in the population depend on effective contact rate and was concluded that the best way to control the Ebola virus disease in the population is to minimize the contact rate. [9,10] developed a mathematical model of Ebola virus and malaria disease, the models were analyzed for stability and it was established that the disease free equilibrium of each model and their co-infections were locally and globally asymptotically stable. In their result, it was show that reducing the contact rate of Ebola-malaria co-infection is the best method to curtain co-infection. Invariably, this study analyzed malaria disease model detached from [9,10] of Ebola virus- Malaria disease co-infections model in-order to have broad knowledge of this infection.

Formulation of malaria disease model

Invariably, human acquires malaria infection following effective contact with infected mosquitoes at a rate $\lfloor_M$, given by

$$\lambda_M = \frac{\beta_M a I_V}{N_V} \qquad (1)$$

where β_M is the transmission probability from mosquito to human provided that there is a contact between the human and the mosquito and `` $a$ `` is the number of mosquito bites that one person has per unit time.

Thus, the rate of change of susceptible population is given by;

$$\frac{dS_H}{dt} = \pi_H - \lambda_M S_H - \mu S_H + \phi_1 R_M \qquad (2)$$

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A fraction ε_2 of new infected individuals with low immunity move to the exposed class (E_M) and the remaining fraction $(1-\varepsilon_2)$ move to the infected class (I_M). The exposed class population increases by those that are treated (at the rate $\rho\phi_3$). The exposed class decreases by progression to infected individuals (at the rate κ_M), those that are treated (at the rate τ_2), natural death (at the rate μ). Thus;

$$\frac{dE_M}{dt} = \varepsilon_2\lambda_M S_M - (\kappa_M + \mu)E_M - \tau_2 E_M \quad (3)$$

The population of individual infected with malaria is generated by a fraction of the new infected individuals with low immunity (at the rate $1-\varepsilon_2$) and progression to infected individuals from the exposed class. The population decreases by treatment of the infected individuals (at the rate τ_3), those who are successfully treated and recovered (at the rate r), natural death (at the rate μ) and disease induced death (at the rate δ_{IM}).

Therefore,

$$\frac{dI_M}{dt} = (1-\varepsilon_2)\lambda_M S_H + \kappa_M E_M - (\tau_3 + r + \delta_{IM} + \mu)I_M \quad (4)$$

The recovered population is generated by treatment (at the rate τ_2 and τ_3) of the exposed and infected class respectively and those who are successfully treated and recovered (at the rate r). There is a decrease in the population due to natural death (at the rate μ) and those that lose immunity (at the rate ϕ_1). Hence;

$$\frac{dR_M}{dt} = \tau_2 E_M + \tau_3 I_M + rI_M - (\phi_1 + \mu)R_M \quad (5)$$

Susceptible mosquitoes (S_V) are generated at a constant (recruitment rate π_V) and acquire malaria infection following effective contacts with humans infected with malaria at a rate λ_v, where the force of infection λ_V is given by;

$$\lambda_v = \frac{\beta_M b I_M}{N_H} \quad (6)$$

b is the number of human bites one mosquito has per unit time, β_M is the transmission probability from human to mosquito. Newly infected mosquitoes move to exposed class and they are assumed to suffer death (at a rate μ_V. Hence;

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$$\frac{dS_V}{dt} = \pi_V - \lambda_V S_V - \mu_V S_V \quad (7)$$

The exposed mosquitoes consist of newly infected mosquitoes and their population diminishes by progression into infected class (at the rate σ_V) and death of the mosquitoes (at the rate μ_V). Therefore;

$$\frac{dE_V}{dt} = \lambda_V S_V - (\sigma_V + \mu_V)E_V \quad (8)$$

The infected mosquitoes have those that progress from exposed class and diminish by the death of the mosquitoes (at the rate μ_V). Hence,

$$\frac{dI_V}{dt} = \sigma_v E_V - \mu_V I_V \quad (9)$$

Applying the assumptions, definitions of compartmental variables and parameters described in Table 1, and the system of nonlinear differential equations that describe the dynamics of malaria disease transmission are formulated and presented below:

$$\frac{dS_H}{dt} = \pi_H - \lambda_M S_H + \phi_1 R_M - \mu S_H$$

$$\frac{dE_M}{dt} = \varepsilon_2 \lambda_M S_H - (\kappa_M + \mu)E_M - \tau_2 E_M$$

$$\frac{dI_M}{dt} = (1-\varepsilon_2)\lambda_M S_H + \kappa_M E_M - (\tau_3 + r + \delta_{IM} + \mu)I_M$$

$$\frac{dR_M}{dt} = \tau_2 E_M + \tau_3 I_M + r I_M - (\phi_1 + \mu)R_M \quad (10)$$

$$\frac{dS_V}{dt} = \pi_V - \lambda_V S_V - \mu_V S_V$$

$$\frac{dE_V}{dt} = \lambda_V S_V - (\sigma_V + \mu_V)E_V$$

$$\frac{dI_V}{dt} = \sigma_V E_V - \mu_V I_V$$

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Where $\lambda_M = \frac{\beta_M b I_V}{N_H}$ and $\lambda_V = \frac{\beta_V b I_M}{N_V}$

Table 1. Definitions of parameters and variables used in the model formulation

PARAMETERS/ VARIABLES	DEFINITIONS
S_H	Susceptible individuals
E_M	Malaria exposed individuals
I_M	Malaria infected individuals
R_M	Malaria recovered individuals
S_V	Susceptible vectors (mosquitoes)
E_V	Exposed vectors (mosquitoes)
I_V	Infected vectors (mosquitoes)
π_H , π_V	Recruitment rate of human and vectors respectively
μ	Human death rate
μ_V	vectors (mosquitoes)death rate
τ_2 , τ_3	Treatment rate for malaria exposed, malaria infected individuals
ε_2	Fraction of individuals with low immunity, Infected with malaria disease
δ_{IM}	Malaria induced death rate for classes E_M , I_M
κ_M	Progression rate for malaria
β_M , β_V	Transmission probability from mosquito to human and human to mosquito respectively
σ_V	Progression rate of vectors (mosquitoes)
r	Recovery rate of malaria
b	Number of mosquito bites per unit time

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λ_M , λ_V	Force of infection from mosquito to human and from human to mosquito respectively
ϕ_1	Rate of loss of immunity

Positivity and boundedness of solutions of malaria model

For the malaria model (10) to be epidemiologically meaningful, it is important to prove that all solutions with nonnegative initial data will remain nonnegative for all time $t > 0$

Consider the biologically-feasible region, $\prod_M = \prod_H \times \prod_V \subset \Re_+^7$ then

$\prod_H = \left\{(S_H, E_M, I_M, R_M,) \varepsilon \Re_+^4 : N_H \leq \frac{\pi_H}{\mu}\right\}$ and $\prod_V = \left\{(S_V, E_V, I_V) \varepsilon \Re_+^3 : N_V \leq \frac{\pi_V}{\mu_V}\right\}$, it will be proved that $\prod_H$ is positively invariant.

Therefore, the malaria model is well posed and epidemiologically meaningful. Hence, it is sufficient to study dynamics of basic model in the region $\prod_M$.

1. Existence of disease free equilibrium (DFE) of malaria model

Disease-free equilibrium point is the steady state solution where there is no infection or disease in the entire population which implies that $(I_M = I_V = 0)$

$$\pi_H - \mu_h S_H = 0, \Rightarrow S_H = \frac{\pi_H}{\mu_h}$$

$$\pi_v - \mu_v S_v = 0, \Rightarrow S_v = \frac{\pi_v}{\mu_v}$$

Hence, the disease-free equilibrium point of the malaria model (1) is given by, $E_0 = (S_H, E_M, I_M, R_M, S_V, E_V, I_V) = \left(\frac{\pi_H}{\mu_H}, 0,0,0,0, \frac{\pi_v}{\mu_v}, 0,0\right)$

Which represent the state in which there is no malaria infection in the population

2. Basic reproduction number

In this section, the threshold parameter that governs the spread of a disease which is called the basic reproduction number is determined. To obtain the reproduction number, the next generation matrix method is the spectral radius of the next generation matrix (Driessche et al., 2002).

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The rate of appearance of new infection in compartment *i* gives the followings:

$$F=\begin{pmatrix} 0 & 0 & 0 & 0 & \frac{\varepsilon_2\beta_M b\mu_V\pi_H}{\pi_V\mu} \\ 0 & 0 & 0 & 0 & \frac{(1-\varepsilon_2)\beta_M b\mu_V\pi_H}{\pi_V\mu} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_V b\mu\pi_V}{\pi_H\mu_V} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$ and the rate of transfer individual in or out of compartment obtained as;

$$V=\begin{pmatrix} \kappa_M+\mu+\tau_2 & 0 & 0 & 0 & 0 \\ -\kappa_M & \tau_3+r+\delta_{IM}+\mu & 0 & 0 & 0 \\ -\tau_2 & -\tau_3-r & \phi_1+\mu & 0 & 0 \\ 0 & 0 & 0 & \sigma_V+\mu_V & 0 \\ 0 & 0 & 0 & -\sigma_V & \mu_V \end{pmatrix}$$

The reproduction number R_M for malaria model is given by $R_M=\sigma(FV^{-1})$, where σ denotes the spectral radius of the dominant eigenvalue of the next generation matrix FV^{-1}, Therefore,

$$R_M=\frac{\sqrt{(\kappa_M+\mu+\tau_2)(\tau_3+r+\delta_{IM}+\mu)\frac{\beta_V b\lambda_V\mu}{\pi_H}\sigma_V\left[\frac{\varepsilon_2\beta_M b\pi_V\pi_H\kappa_M}{\pi_V\mu}+\frac{(1-\varepsilon_2)\beta_M b\pi_V\pi_H\kappa_M}{\pi_V\mu}(\kappa_M+\mu+\tau_2)\right]}}{(\kappa_M+\mu+\tau_2)(\tau_3+r+\delta_{IM}+\mu)(\sigma_V+\mu)\mu_V}$$

3. Local stability of disease free equilibrium of the malaria model

Theorem 2

The disease free equilibrium point is locally asymptotically stable if $R_M<1$ and unstable if $R_M>1$. Then the theorem implies the disease can be eliminated from the community.

Proof: To prove local stability of disease free equilibrium, the Jacobian matrix of the system (1) at the disease free equilibrium E_0 is obtained

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$$J(E_0)=\begin{pmatrix} -\mu & 0 & 0 & \phi_1 & 0 & 0 & -\frac{\beta_M b\mu_V\pi_H}{\pi_V\mu} \\ 0 & -(\kappa_M+\mu+\tau_2) & 0 & 0 & 0 & 0 & \frac{\varepsilon_2\beta_M b\mu_V\pi_H}{\pi_V\mu} \\ 0 & \kappa_M & -(\tau_3+r+\delta_{IM}+\mu) & 0 & 0 & 0 & \frac{(1-\varepsilon_2)\beta_M b\mu_V\pi_H}{\pi_V\mu} \\ 0 & \tau_2 & (\tau_3+r) & -(\phi_1+\mu) & 0 & 0 & 0 \\ 0 & 0 & \frac{-\beta_V b\pi_V\mu}{\mu_V\pi_H} & 0 & -\mu_V & 0 & 0 \\ 0 & 0 & \frac{\beta_V b\pi_V\mu}{\mu_V\pi_H} & 0 & 0 & -(\sigma_V+\mu_V) & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_V & -\mu_V \end{pmatrix}$$

For simplicity let: $T_1=\frac{\beta_M b\mu_V\pi_H}{\pi_V\mu}$, $T_2=\kappa_M+\mu+\tau_2$, $T_3=\frac{\varepsilon_2\beta_M b\mu_V\pi_H}{\pi_V\mu}$, $T_4=\tau_3+r+\delta_{IM}+\mu$, $T_5=\frac{(1-\varepsilon_2)\beta_M b\mu_V\pi_H}{\pi_V\mu}$, $T_6=\tau_3+r$, $T_7=\phi_1+\mu$, $T_8=\frac{\beta_V b\pi_V\mu}{\mu_V\pi_H}$ $T_{10}=\sigma_V+\mu_V$

Therefore

$$|J-\lambda I|=\begin{vmatrix} -\mu-\lambda & 0 & 0 & \phi_1 & 0 & 0 & -T_1 \\ 0 & -T_2-\lambda & 0 & 0 & 0 & 0 & T_3 \\ 0 & \kappa_M & -T_4-\lambda & 0 & 0 & 0 & T_5 \\ 0 & \tau_2 & T_6 & -T_7-\lambda & 0 & 0 & 0 \\ 0 & 0 & -T_8 & 0 & -\mu_V-\lambda & 0 & 0 \\ 0 & 0 & T_9 & 0 & 0 & -T_{10}-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_V & -\mu_V-\lambda \end{vmatrix}=0$$

Now, expand along first column

$$(-\mu-\lambda)\begin{vmatrix} -T_2-\lambda & 0 & 0 & 0 & 0 & T_3 \\ \kappa_M & -T_4-\lambda & 0 & 0 & 0 & T_5 \\ \tau_2 & T_6 & -T_7-\lambda & 0 & 0 & 0 \\ 0 & T_8 & 0 & \mu_V-\lambda & 0 & 0 \\ 0 & T_9 & 0 & 0 & -T_{10}-\lambda & 0 \\ 0 & 0 & 0 & 0 & \sigma_V & \mu_V-\lambda \end{vmatrix}=0$$

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Therefore $\lambda_1 = -\mu$, or $\begin{vmatrix} -T_2-\lambda & 0 & 0 & 0 & 0 & T_3 \\ \kappa_M & -T_4-\lambda & 0 & 0 & 0 & T_5 \\ \tau_2 & T_6 & -T_7-\lambda & 0 & 0 & 0 \\ 0 & T_8 & 0 & \mu_V-\lambda & 0 & 0 \\ 0 & T_9 & 0 & 0 & -T_{10}-\lambda & 0 \\ 0 & 0 & 0 & 0 & \sigma_V & \mu_V-\lambda \end{vmatrix} = 0$

Also, expand along third columns obtain

$$(-T_7-\lambda)\begin{vmatrix} T_2-\lambda & 0 & 0 & 0 & T_3 \\ \kappa_M & T_4-\lambda & 0 & 0 & T_5 \\ 0 & T_8 & -\mu_V-\lambda & 0 & 0 \\ 0 & T_9 & 0 & -T_{10}-\lambda & 0 \\ 0 & 0 & 0 & \sigma_V & \mu_V-\lambda \end{vmatrix} = 0$$

$\Rightarrow \lambda_2 = -T_7$ or $\begin{vmatrix} T_2-\lambda & 0 & 0 & 0 & T_3 \\ \kappa_M & T_4-\lambda & 0 & 0 & T_5 \\ 0 & T_8 & -\mu_V-\lambda & 0 & 0 \\ 0 & T_9 & 0 & -T_{10}-\lambda & 0 \\ 0 & 0 & 0 & \sigma_V & \mu_V-\lambda \end{vmatrix} = 0$

Similarly, expand along third columns

$$-(\mu_V-\lambda)\begin{vmatrix} -T_2-\lambda & 0 & 0 & T_3 \\ \kappa_M & -T_4-\lambda & 0 & T_5 \\ 0 & T_9 & -T_{10}-\lambda & 0 \\ 0 & 0 & \sigma_V & -\mu_V-\lambda \end{vmatrix} = 0$$

$\therefore \lambda_3 = -\mu_V$

or $\begin{vmatrix} -T_2-\lambda & 0 & 0 & T_3 \\ \kappa_M & -T_4-\lambda & 0 & T_5 \\ 0 & T_9 & -T_{10}-\lambda & 0 \\ 0 & 0 & \sigma_V & -\mu_V-\lambda \end{vmatrix} = 0$

Expand the remaining along first row

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$$(-T_2-\lambda)\begin{vmatrix} -T_4-\lambda & 0 & T_5 \\ T_9 & T_{10}-\lambda & 0 \\ 0 & \sigma_V & -\mu_V-\lambda \end{vmatrix}$$

$$-T_3\begin{vmatrix} \kappa_M & -T_4-\lambda & 0 \\ 0 & T_9 & -T_{10}-\lambda \\ 0 & 0 & \sigma_V \end{vmatrix}=0$$

$$-(T_2-\lambda)\left[(T_4-\lambda)(-T_{10}-\lambda)(-\mu_V-\lambda)+T_5T_9\sigma_V\right]-T_3T_9\kappa_M\sigma_V=0$$

After expansion and simplification obtain the following polynomial equation

$$C_0\lambda^4+C_1\lambda^3+C_2\lambda^2+C_3\lambda+C_4=0 \quad (11)$$

Where

$$C_0=1$$

$$C_1=\mu_V+T_2+T_{10}$$

$$C_2=T_2\mu_V+T_4\mu_V+T_2T_4+T_2T_{10}+\mu_VT_{10}+T_4T_{10}$$

$$C_3=\mu_VT_2T_4+\mu_VT_2T_{10}+T_2T_4T_{10}+\mu_VT_4T_{10}-\sigma_VT_5T_9 \; C_4=\mu_VT_2T_4T_{10}-T_2T_5T_9\sigma_V-T_3T_9\kappa_M\sigma_V$$

By Routh-Hurwitz criterion,

$$C_1>0$$

which means that,

$$2\mu_V+\kappa_M+\mu+\tau_2+\sigma_V$$

Also,

$$C_2>0$$

Means that,

$$\mu_V(\kappa_M+\mu+\tau_2)+\mu_V(\tau_3+r+\delta_{IM}+\mu)+(\kappa_M+\mu+\tau_2)(\tau_3+r+\delta_{IM}+\mu)+(\kappa_M+\mu+\tau_2)(\sigma_V+\mu_V)+\mu_V(\sigma_V+\mu_V)+(\tau_3+r+\delta_{IM}+\mu)(\sigma_V+\mu_V) \qquad C_3>0$$

means that

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$\mu_V(\kappa_M+\mu+\tau_2)(\tau_3+r+\delta_{IM}+\mu)+\mu_V(\kappa_M+$
$\mu+\tau_2)(\sigma_V+\mu_V)+(\kappa_M+\mu+\tau_2)(\tau_3+r+\delta_{IM}+\mu)$
$(\sigma_V+\mu_V)+\mu_V(\tau_3+r+\delta_{IM}+\mu)(\sigma_V+\mu_V)-$
$\sigma_V\frac{(1-\varepsilon_2)\beta_M b\mu_V\pi_H}{\pi_V\mu}\frac{\beta_V b\pi_V\mu}{\mu_V\pi_H}$ finally,

$C_4>0$

which means that

$$\mu_V(\kappa_M+\mu+\tau_2)(\tau_3+r+\delta_{IM}+\mu)(\sigma_V+\mu_V)-(\kappa_M+\mu+\tau_2)$$
$$\frac{(1-\varepsilon_2)\beta_M b\mu_V\pi_H}{\pi_V\mu}\frac{\beta_V b\pi_V\mu}{\mu_V\pi_H}\sigma_V-\frac{\varepsilon_2\beta_M b\mu_V\pi_H}{\pi_V\mu}\frac{\beta_V b\pi_V\mu}{\mu_V\pi_H}\kappa_M\sigma_V$$

$\Rightarrow R_M<1$

Thus, the disease free equilibrium is locally asymptotically stable if $\Rightarrow R_M<1$.

4. Existence and Stability of Endemic Equilibrium of Malaria Model

The endemic equilibrium is denoted by $E_1^{**}=(S_H^{**},E_M^{**},I_M^{**},R_M^{**},S_V^{**},E_V^{**},I_V^{**})$ and it occurs when the disease persists in the community. To obtain, equate all the model equations (10) to zero. Then obtain:

$$S_H^{**}=\frac{\pi_H+\phi_1R_M^{**}}{\lambda_M^{**}+\mu}\quad E_M^{**}=\frac{\varepsilon_2\lambda_M^{**}S_H^{**}}{\kappa_M+\mu+\tau_2}=\frac{\varepsilon_2\lambda_M^{**}S_H^{**}}{K_1},$$
$$I_M^{**}=\frac{(1-\varepsilon_2)\lambda_M^{**}S_H^{**}+\kappa_ME_M^{**}}{\tau_3+r+\delta_{IM}+\mu}=\frac{(1-\varepsilon_2)\lambda_M^{**}S_H^{**}+\kappa_ME_M^{**}}{K_2}$$
$$R_M^{**}=\frac{\tau_2E_M^{**}+\tau_3I_M^{**}+rI_M^{**}}{\phi_1+\mu}=\frac{\tau_2E_M^{**}+\tau_3I_M^{**}+rI_M^{**}}{K_3}$$

$$S_V^{**}=\frac{\pi_V}{\lambda_V^{**}+\mu},\quad E_V^{**}=\frac{\lambda_V^{**}S_V^{**}}{K_4},\quad I_V^{**}=\frac{\sigma_VE_V^{**}}{\mu_V}$$

For computational convenience, the expressions above can be rewrite in terms of $\lambda_M^{**}S_H^{**}$ and $\lambda_V^{**}S_V^{**}$.

Therefore

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$$E_M^{**} = \frac{\varepsilon_2 \lambda_M^{**} S_H^{**}}{K_1} = P_1 \lambda_M^{**} S_H^{**}, \qquad I_M^{**} = \frac{[K_1(1-\varepsilon_2) + \kappa_M \varepsilon_2]\lambda_M^{**} S_H^{**}}{K_1 K_2} = P_2 \lambda_M^{**} S_H^{**},$$

$$R_M^{**} = \frac{\tau_2 E_M^{**} + \tau_3 I_M^{**} + r I_M^{**}}{K_3}$$

$$= \frac{K_1(\tau_3(1-\varepsilon_2) + r(1-\varepsilon_2))\lambda_M^{**} S_H^{**} + (K_2\tau_2 + \kappa_M\tau_3 + r\kappa_M)\varepsilon_2 \lambda_M^{**} S_H^{**}}{K_1 K_2 K_3}$$

$$= P_3 \lambda_M^{**} S_H^{**} \qquad (12)$$

$$E_V^{**} = \frac{\lambda_V^{**} S_V^{**}}{K_4} = q_1 \lambda_V^{**} S_V^{**}$$

$$I_V^{**} = \frac{\sigma_V \lambda_V^{**} S_V^{**}}{\mu_V K_4} = q_2 \lambda_V^{**} S_V^{**}$$

Note that the force of infections defined by

$$\lambda_M^{**} = \frac{\beta_M b I_V^{**}}{N_V^{**}} \Rightarrow \lambda_M^{**} N_V^{**} = \lambda_M^{**}(S_V^{**} + E_V^{**} + I_V^{**}) = \beta_M b I_V^{**} \qquad (13)$$

$$\lambda_V^{**} = \frac{\beta_V b I_M^{**}}{N_M^{**}} \Rightarrow \lambda_V^{**} N_M^{**}$$

And $= \lambda_V^{**}(S_H^{**} + E_M^{**} + I_M^{**} + R_M^{**}) = \beta_V b I_M^{**}$ (14)

Substituting the expressions in (13) into (14)

$$\lambda_M^{**}(S_V^{**} + q_1 \lambda_V^{**} S_V^{**} + q_2 \lambda_V^{**} S_V^{**}) = \beta_M b q_2 \lambda_V^{**} S_V^{**}$$

Divide through S_V^{**}, obtain

$$\lambda_M^{**}(1 + q_1 \lambda_V^{**} + q_2 \lambda_V^{**}) = \beta_M b q_2 \lambda_V^{**} \Rightarrow \lambda_M^{**} = \frac{\beta_M b q_2 \lambda_V^{**}}{1 + q_1 \lambda_V^{**} + q_2 \lambda_V^{**}}, \text{ i.e. } \lambda_M^{**} = \frac{\beta_M b q_2 \lambda_V^{**}}{1 + q_3 \lambda_V^{**}} \qquad (15)$$

Also, substitute the expressions in (9) into (11) give,

$$\lambda_V^{**}(S_H^{**} + P_1 \lambda_M^{**} S_H^{**} + P_2 \lambda_M^{**} S_H^{**} + P_3 \lambda_M^{**} S_H^{**})$$
$$= \beta_V b P_2 \lambda_M^{**} S_H^{**}$$

Divide through S_H^{**}, obtain

$$\lambda_V^{**}(1 + P_1 \lambda_M^{**} + P_2 \lambda_M^{**} + P_3 \lambda_M^{**}) = \beta_V b P_2 \lambda_M^{**}$$

$$\Rightarrow \lambda_V^{**} = \frac{\beta_V b P_2 \lambda_M^{**}}{1 + (P_1 + P_2 + P_3)\lambda_M^{**}},$$

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i.e. $\lambda_V^{**} = \dfrac{\beta_V b P_2 \lambda_M^{**}}{1 + P_4 \lambda_M^{**}}$ (16)

From equation (12)

$$\lambda_M^{**}(1 + q_3 \lambda_V^{**}) = \beta_M b q_2 \lambda_V^{**} \quad (17)$$

Substitute (16) into (17) obtain

$$\lambda_M^{**}(1 + q_3 \lambda_V^{**}) = \beta_M b q_2 \frac{\beta_V b P_2 \lambda_M^{**}}{1 + P_4 \lambda_M^{**}} \Rightarrow \lambda_M^{**}(1 + q_3 \lambda_V^{**})(1 + P_4 \lambda_M^{**}) = \beta_M b q_2 \beta_V b P_2 \lambda_M^{**}$$

$$(1 + q_3 \lambda_V^{**})(1 + P_4 \lambda_M^{**}) = \beta_M b q_2 \beta_V b P_2 = R_M^2$$

Therefore, $1 + q_3 \lambda_V^{**} = R_M^2$ or $1 + P_4 \lambda_M^{**} = R_M^2$

So that $\lambda_V^{**} = \dfrac{R_M^2 - 1}{q_3} > 0$ or $\lambda_M^{**} = \dfrac{R_M^2 - 1}{P_4} > 0$, whenever $R_M > 1$. (18)

Then, the components of E_1 can be obtained by substituting the unique value of λ_M^{**} and λ_V^{**}, given by (15), into the expressions in (16). Thus, the following result is established.

Lemma 1 The model (10) has a unique endemic (positive) equilibrium, given by E_1, whenever $R_M > 1$.

5. Sensitivity analysis of the model parameters for malaria model

Sensitivity analysis was carried out to determine the model robustness to parameter values. This helps to identify the parameters that have a high impact on the reproductive number R_M which need to be worked with in simulation by varying those parameters on the entire population. The sensitivity indices was calculated using normalized forward sensitivity method, which is defined as the ratio of the relative change in R_M to the relative change in the parameter "P": $Z_P^{R_M} = \dfrac{\partial R_M}{\partial P} \times \dfrac{P}{R_M}$.

Table 2: Sensitivity values on the basic reproduction number R_M of malaria model

Parameters	Sensitivity indices
b	1.000000000
μ	0.7915725085
κ_M	0.6501567840
σ_V	0.5000000005

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β_M	0.5000000000
β_V	0.5000000000
π_V	0.5000000000
ε_2	0.3076489170
δ_{IM}	0.09214891265
r	0.03685956506
τ_2	0.003806791715
τ_3	0.002395871729
π_H	0.000000000000

6. Numerical Simulation Results

The analytical results of this study are illustrated by carrying out numerical simulations of the models using parameter values obtained from literature on Table 3 and the results were displayed below.

Table 3. Parameters values used for the numerical simulation

Parameters	Values	Sources
δ_{IM}	0.05	Smith and HoveMusekwa (2008)
κ_M	0.071	Malariajournal.com (2011)
β_M	0.03	Amoah-Mensah et al. (2018)
ε_2	0.6	Estimated
τ_3	0.0013	CDC (2014).
τ_2	0.0018	[10]
π_V	400	[11]
π_H	1800	[11]
μ	0.2	Estimated
μ_V	0.05	Malariajournal.com (2011)
β_V	0.09	Blayneh & Kwon (2009)
σ_V	0.1	Chitnis et al. (2006)
ϕ_1	0.02	Adewale et al. (2015)
r	0.02	Abu-Raddad et al. (2006)
b	0.4	Chitnis (2008)

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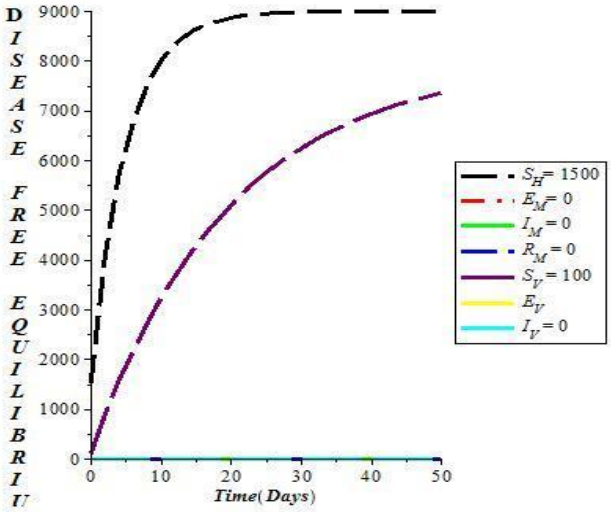

Fig.1: Graph showing disease free equilibrium point of malaria disease model at different time

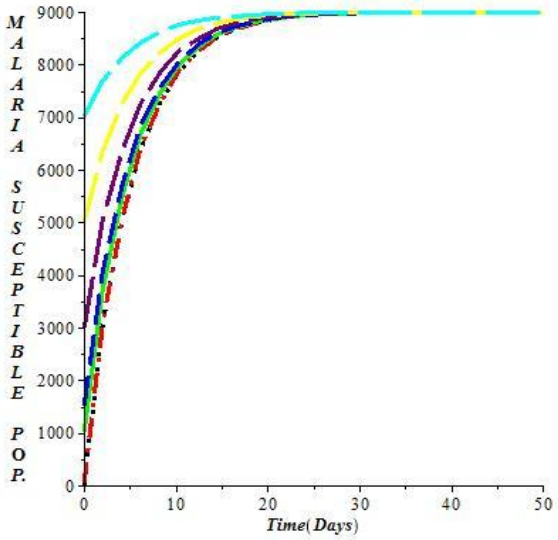

Fig. 2: Graph showing global stability of endemic equilibrium point of malaria disease model at different time

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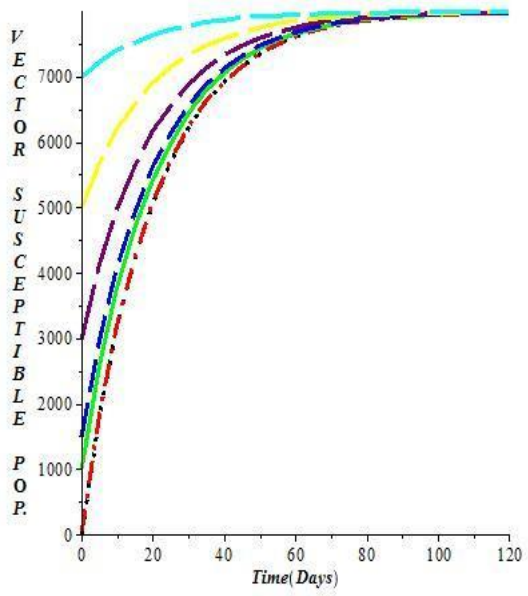

Fig. 3: Graph showing global stability of endemic equilibrium point of malaria disease model at different time

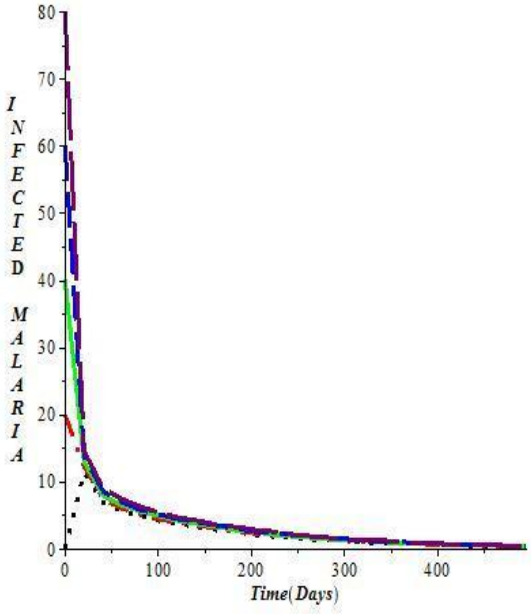

Fig. 4: Graph showing the global stability of endemic equilibrium point of malaria disease model at different time

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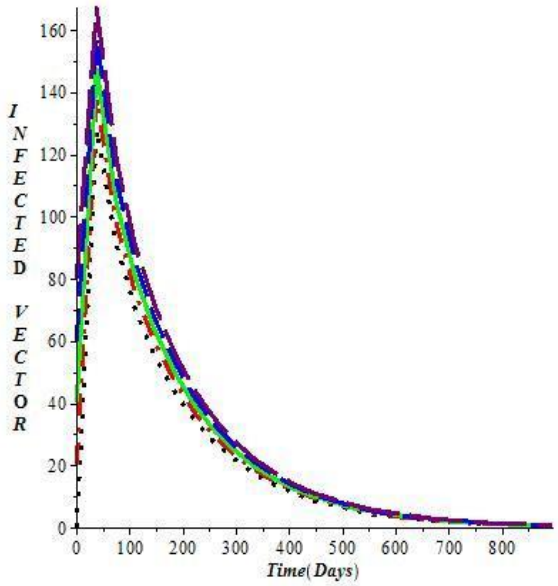

Fig. 5: Graph showing the global stability of endemic equilibrium point of malaria disease model at different time

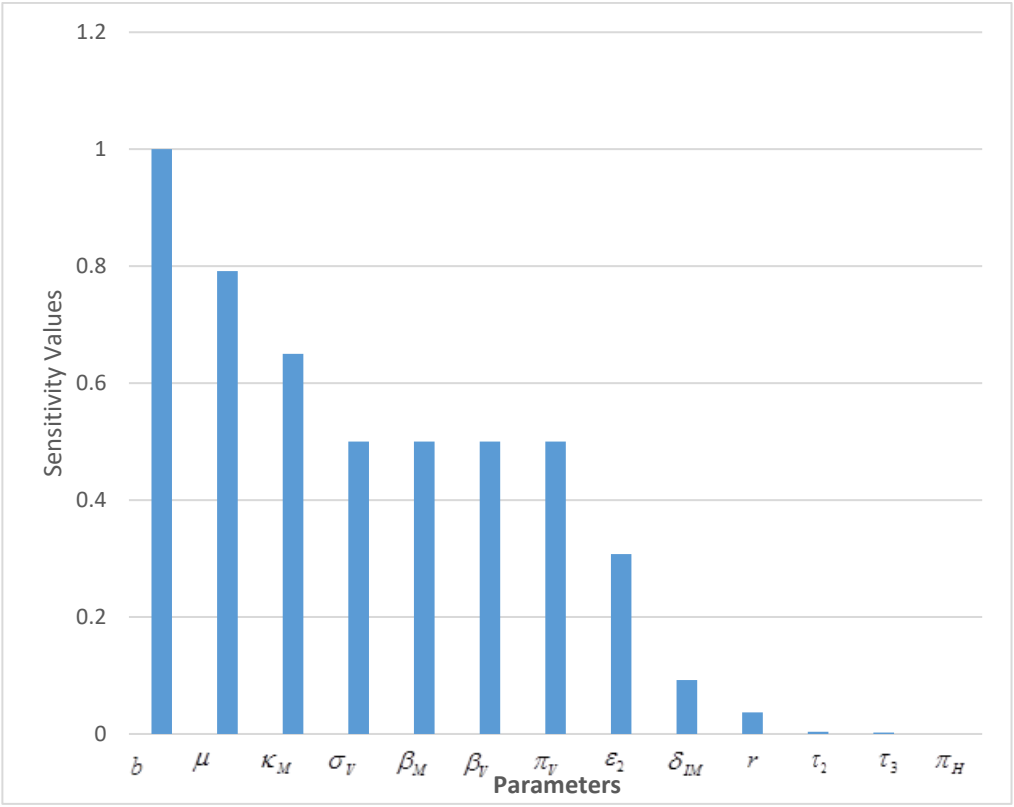

Fig. 6: Chart of the sensitivity indices on the basic reproduction number of malaria model

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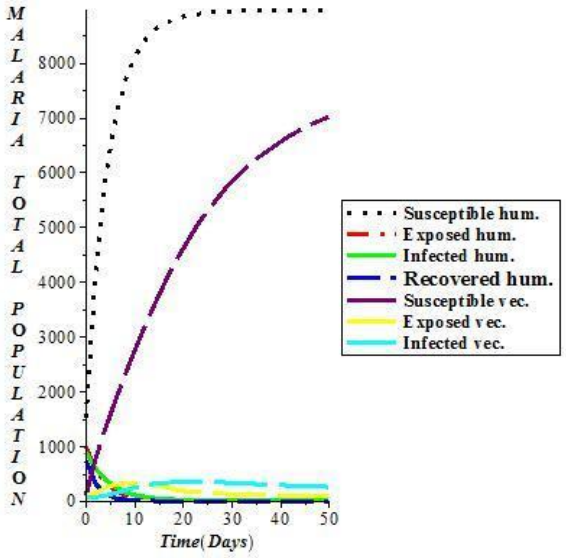

Fig. 7: Graph represents the initial behavior of the malaria disease model

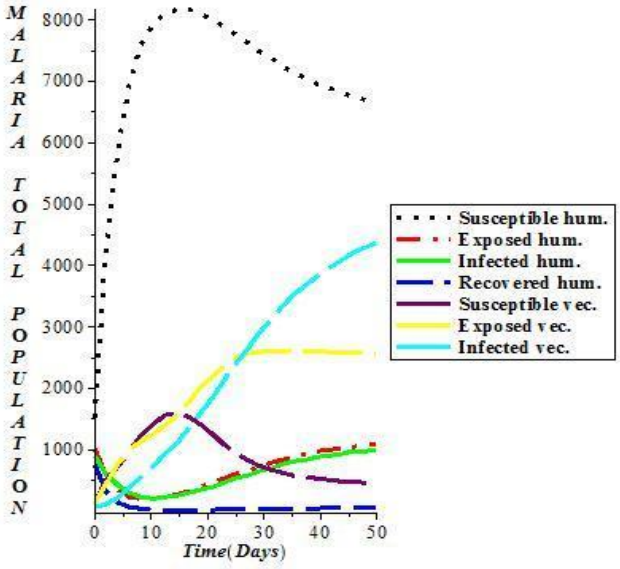

Fig. 8: Graph of increasing the most positive sensitive index value (biting rate) on malaria disease model

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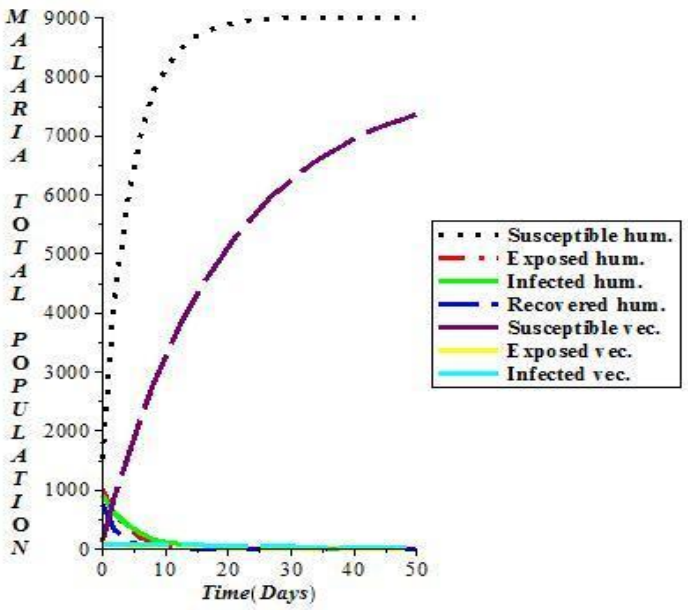

Fig. 9: Graph of eliminating the most positive sensitive index value (biting rate) on malaria disease model only

DISCUSSION OF RESULTS

Figure 1 shows the disease free equilibrium point of malaria disease, as it shows that there is always susceptibility in the population while infected individual disappear. This established the theorem 1. Also, Figures 2, 3, 4 and 5 show the global stability of malaria indicates the initial values the system converges to the same point.

Figure 7 illustrates the behavior of the malaria model with initial values. It was found initially that the proportion of susceptible malaria population increases rapidly before decline and remains steady because of recovery loss of immunity and death rate. Exposed malaria population rapidly decreases as a result of infection. Also, the infected malaria population decreases steadily as a result of treatment and recovery rate. Recovered population decreases due to the fact that those who have the infections leave the infected class after receiving the treatment and remain constant for some time but because of loss immunity. Susceptible vector population goes up sharply because of increases in recruitment rate. In the exposed vector population there is an increase in population but later decline because of the death rate in class. Infected vector population rises because of exposure to disease.

Figure 8 shows susceptible human and susceptible vector increases initially between zero to eleven days, after that it starts decreasing as a result of increasing in biting rate. Exposed human, infected human, recovered human, exposed vector and infected vector keep increasing as well. Figure 9 shows that when biting rate was eliminated from the compartment, susceptible human and susceptible vector increases while exposed human, infected human, recovered human, exposed vector and infected vector drastically reduced to the nearest minimum.

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CONCLUSION

In this work, a deterministic model for the dynamics of malaria disease is presented and analyzed, the model was analyzed for stability and it was established that the disease free equilibrium of the model was locally and globally asymptotically stable whenever the basic reproduction number is less than unity. Similarly, there exists endemic situation when the basic reproduction numbers of malaria disease are greater than unity. Conclusively, the results obtained in Table 2, Figure 6 and Figure 8 from sensitivity analysis reveal that the spread of a malaria disease within the population depend on biting rate of mosquito, which need to be controlled as suggested from Figure 9 for us to have malaria disease free environment in the population.

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