Bifurcation Analysis on Malaria using SEIR Model for Human and SEI Model for Vector

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Abstract

This paper aims to carry out the bifurcation analysis on the newly formulated mathematical model on malaria disease. The positivity of the model was done while the disease-free equilibrium of the model was established. The Basic reproduction number (R_{0M}) of the model was determined, which proved the quick spread of the

disease whenever the time is greater than one and curbing or reduction in spreading the disease if it is lesser than one. We carried out the model sensitivity analysis in which the effect of each epidemiological term on the spread of malaria infection in society was revealed. It was shown finally that mosquitoes' contact rate has a more significant influence on the basic reproduction number of malaria. Also, mosquitoes' death rate has an effect on the spread of this infectious disease.

Keywords: Bifurcation, Basic reproduction number, Prophylactics, Disease free equilibrium

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I. INTRODUCTION

Malaria is an infection of the blood that is transmitted from person to person by mosquitoes. The disease has been recognized for thousands of years and once was found almost everywhere except in the most northern areas of the world. Malaria has been eliminated in North America, Western Europe, and Russia. However, it remains a severe problem in most of the tropical and subtropical world. Millions of people continue to be infected every year, and close to one million of them die. However, around 1,500 people in a year are diagnosed with malaria in the United Kingdom after returning from abroad. (World Malaria Report, 2011).

Malaria is a life-threatening blood disease caused by parasites transmitted to humans through the bite of the *Anopheles* mosquito. Once an infected mosquito bites a human and transmits the parasites, those parasites multiply in the host's liver before infecting and destroying red blood cells. The disease can be controlled and treated if diagnosed early. Unfortunately, this is not possible in some areas lacking medical facilities, where malaria outbreaks can occur. There are more than 100 types of plasmodium parasites, which can infect a variety of species. Scientists have identified five types that specifically infect humans: Plasmodium falciparum - located worldwide in tropical and suburban areas but predominately in Africa.

An estimated 1 million people are killed by this strain every year. The strain can multiply rapidly and adhere to blood vessel walls in the brain, causing the rapid onset of severe malaria, including cerebral malaria. *P* falciparum - Common worldwide, this is the most life-threatening form of malaria. This parasite has an incubation period of 5-12 days (Shortt *et al.*,1951). Plasmodium vivax (henceforth P-vivax) - located in Latin America, Africa, and Asia, is arguably the most widespread due to the high population of Asia. This strain has a dormant liver stage that can activate and invade the blood after months or years, causing many patients to relapse. It has an incubation period of 8-13 days.

Infections can sometimes lead to life-threatening rupture of the spleen. If people are treated only with chloroquine, this type of malaria can hide in the liver and return later (Cowman and Crab, 2006). Plasmodium ovale is located mainly in West Africa, and it is biologically and morphologically similar to P-vivax. However, unlike P-vivax, this strain can affect individuals who are negative with the duff blood group, which is the case for many residents of sub-Saharan Africa. This explains the greater prevalence of *P-ovale* (rather than *P-vivax*) in most of Africa. *P ovale* - rarely found outside Africa. This form of malaria has an incubation period of 8-17 days and can hide in the liver of partially treated people and return later (Cowman and Crab, 2006).

Plasmodium malaria -found worldwide and is the only human malaria parasite to have a three-day cycle. If left untreated, *P. malaria* can cause a long-lasting, chronic infection that can last a lifetime and which may cause nephrotic syndrome. This form of malaria has an incubation of 2-4 weeks. If untreated, the infection can last for many years. Plasmodium knowlesi - located in Southeast Asia and associated with macaques (a type

of monkey). This strain has a 24 hour cycle and can multiply rapidly once a patient is infected, causing an uncomplicated case to become serious very quickly. (World Malaria Report 2014)

Evans *et al.* (2004) carried out a study where 12% of malaria patients had bacteremia. Though severe malaria and bacteremia were not positively associated, patients who smeared positive for malaria and those smears negative for it could not be distinguished based on symptoms alone. The recommendation from this study was that infants with symptoms of severe malaria but smear-negative for it should be given antibiotics. Lashari *et al.* (2013) researched on backward bifurcation and optimal control of a vector-borne disease. They concluded that the preventive practices are very effective in reducing the incidence of infectious hosts and vectors. Feng *et al* (2015) worked on stability and backward bifurcation in a malaria transmission model with applications to control malaria in China.

In this paper, bifurcation analysis on malaria with the aid of a mathematical model using susceptible, exposed, infected and recovered model for human and susceptible, exposed and infected model for vector were used to determine how to curb or reduced the spread of malaria disease in the community and society at large.

II. MODEL FORMULATION

Despite the work done by the researchers as mentioned above and those that could not be mentioned here, there is some vacuum which was left out like bifurcation analysis on malaria mathematical model using SEIR model for humans and SEI model for the vector. The human population is not constant since migration, emigration, mortality, and immortality occur. The susceptible humans are increased by the recruitment of people (either by birth or immigration) into the population at π_h rate, recovered individuals from malaria class at β rate. This class of susceptible human is decreased by natural death and transmission rate of an infected individual. As a result, the exposed individuals (E_M) increased by the infection of slow progressors at the rate $(1 - \mathcal{E}_1)$. This class E_M reduces by natural death of people in this class by μ_h , those that received treatment at rate τ_3 of exposed individuals and by the rate at which malaria spread or progressed at κ_1 . The population of infected individuals (I_M) increased by a fraction \mathcal{E}_1 of the newly infected individuals and by progression rate κ_1 of individual that exposed to malaria but had left exposed class. This class is reduced by natural death of people in this class by μ_h , induced mortality of malaria due to infection at the rate δ_2 and by treatment given to infected individuals at the rate au_2 . The recovery here means recover from the disease. The population of recovered class (R_M) increased by the group of individuals who received treatment in the infected class at the rate τ_2 and by those treated at exposed class at the rate τ_3 but later joined the recovery class. This recovery class was reduced by the natural death of people in this class, and individuals totally recovered from malaria at a rate β that moved back to the susceptible class.

The total vector population $N_V(t)$, is also divided into various components which are susceptible $S_V(t)$, of mosquitoes, exposed $E_V(t)$, of mosquitoes, and infected $I_V(t)$, of mosquitoes. The susceptible vector class was increased by the recruitment of vectors (anopheles mosquitoes) into the population at the rate π_v . This class was decreased by the natural death of vector at the rate μ_v and by transmitted rate (λ_v) of susceptible vectors. The exposed vector (mosquito) class increased by the transmission rate of susceptible vectors that left susceptible class of vector. This same class E_v decreased by natural death of exposed vectors and progression of vectors at the rate σ_v . The infected vectors class was increased by the progressive vectors which left exposed vectors class and joined infected vectors class. This class decreased by the natural death of vectors in this class.

Therefore, the new mathematical model formulated as follows

$$\frac{dS_H}{dt} = (1-\ell)\pi_H - \lambda_m S_H + \beta R_m - \mu_H S_H$$
(1)

$$\frac{dE_m}{dt} = (1 - \varepsilon_1)\lambda_m S_H - \mu_H E_m - \kappa_1 E_m - \tau_3 E_m$$
(2)

$$\frac{dI_m}{dt} = \varepsilon_1 \lambda_m S_H - \mu_H I_m - \delta I_m + \kappa_1 E_m - \tau_2 I_m$$
(3)

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$$\frac{dR_m}{dt} = \tau_2 I_m - \mu_H R_m - \beta R_m + \tau_3 E_m \tag{4}$$

$$\frac{dS_V}{dt} = \pi_V - \lambda_V S_V - \mu_V S_V \tag{5}$$

$$\frac{dL_{v}}{dt} = \lambda_{v}S_{v} - \mu_{v}E_{v} - \sigma_{v}E_{v}$$

$$\frac{dI_{v}}{dt} = \sigma E_{v} - \mu I$$
(6)

$$\frac{dt}{dt} = \frac{dt}{dt}$$
(7)

$$\lambda_{m} = \frac{\omega_{m} dI_{v}}{N_{v}}$$
$$\lambda_{v} = \frac{\omega_{v} b [E_{m} + \eta_{1} I_{m}]}{N_{H}}$$



FIGURE 2.1: Flow chart of malaria model (humans) 2.1. Transmission rate of malaria (human) and vector.

$$\lambda_{M} = \frac{\omega_{M} A I_{V}}{N_{V}}, \tag{8}$$

$$\lambda_{V} = \frac{\omega_{V} B (E_{M} + \eta_{1} I_{M})}{N_{H}}, \tag{9}$$

Where ω_M are human capital biting rate by mosquito and ω_V is the biting rate of mosquitoes, also A is the probability that a bite by an infected mosquito on a susceptible human will transfer the infection to human and B is the transmission probability for mosquito infection.

III. POSITIVITY OF SOLUTION

There is necessity to prove that all mentioned variables are non-negative for all t > 0 in order to show that Malaria model is epidemiologically meaningful and well posed.

Theorem 1: If the initial values $S_H(0)$, $E_M(0)$, $I_M(0)$, $R_M(0)$, $S_V(0)$, $E_V(0)$, $I_V(0)$ are non negatives, then the solution $\{S_H(t), E_M(t), I_M(t), R_M(t), S_V(t), E_V(t), I_V(t)\}$ of system (1 to 7) is non negatives for all $t \ge 0$. However,

$$\begin{split} \limsup_{t \to \infty} N_H(t) &\leq \frac{(1-\ell)\pi_H}{\mu_H} \text{ and } \limsup_{t \to \infty} N_V(t) \leq \frac{\pi_V}{\mu_V} \\ N_H(0) &\leq \frac{(1-\ell)\pi_H}{\mu_H}, \text{ then, } N_H(t) \leq \frac{(1-\ell)\pi_H}{\mu_H} \text{ and} \\ \text{Also, if} \quad & \text{if } N_V(0) \leq \frac{\pi_V}{\mu_V}, \text{ then } N_V(t) \leq \frac{\pi_V}{\mu_V} \end{split}$$

In particular, the region

$$\Omega = \{ (S_H, E_M, I_M, R_M, S_V, E_V, I_V) \in \mathfrak{R}^7_+ : S_H + E_M + I_M + R_M \le \frac{(1-\ell)\pi_H}{\mu_H} \text{ and } S_V + E_V + I_V \}$$
(10)

is positively invariant. The proof is neglected for clarity. The model has disease free equilibrium at the point $\varepsilon_0 = \{\frac{(1-\ell)\pi_H}{\mu_H}, 0, 0, 0, \frac{\pi_V}{\mu_V}, 0, 0\}$.

3.1. Basic Reproduction Number

This can be determined by using Next generation matrix method.(Watmough and Driessche, 2002). Using their approach, $(F.V^{-1})$, where,

$$F_{i} = \begin{bmatrix} F_{1} \\ F_{2} \\ F_{3} \\ F_{4} \\ F_{5} \end{bmatrix} = \begin{bmatrix} (1 - \varepsilon_{1})\lambda_{m}S_{H} \\ \varepsilon_{1}\lambda_{m}S_{H} \\ 0 \\ \lambda_{\nu} \\ 0 \end{bmatrix}$$
(11)
$$V_{1} = \begin{bmatrix} V_{1} \\ V_{2} \\ V_{3} \\ V_{4} \\ V_{5} \end{bmatrix} = \begin{bmatrix} \mu_{H}E_{m} + k_{1}E_{m} + \tau_{3}E_{m} \\ (\mu_{H} + \delta + \tau_{2})I_{m} - k_{1}E_{m} \\ (\mu_{H} + \delta + \tau_{2})I_{m} - k_{1}E_{m} \\ \tau_{2}I_{m} + (\mu_{H} + \beta)R_{m} - \tau_{3}E_{m} \\ (\mu_{\nu} + \sigma_{\nu})E_{\nu} \\ -\sigma_{\nu}E_{\nu} + \mu_{\nu}I_{\nu} \end{bmatrix}$$
(12)

The jacobian representation of F is given as:

$$J_{m}(E_{m}, I_{m}, R_{m}, E_{v}, I_{v}) = \begin{bmatrix} \frac{\partial f_{1}}{\partial E_{m}} & \frac{\partial f_{1}}{\partial I_{m}} & \frac{\partial f_{1}}{\partial R_{m}} & \frac{\partial f_{1}}{\partial E_{v}} & \frac{\partial f_{1}}{\partial I_{v}} \\ \frac{\partial f_{2}}{\partial E_{m}} & \frac{\partial f_{2}}{\partial I_{m}} & \frac{\partial f_{2}}{\partial R_{m}} & \frac{\partial f_{2}}{\partial E_{v}} & \frac{\partial f_{2}}{\partial I_{v}} \\ \frac{\partial f_{3}}{\partial E_{m}} & \frac{\partial f_{3}}{\partial I_{m}} & \frac{\partial f_{3}}{\partial R_{m}} & \frac{\partial f_{3}}{\partial E_{v}} & \frac{\partial f_{3}}{\partial I_{v}} \\ \frac{\partial f_{4}}{\partial E_{m}} & \frac{\partial f_{4}}{\partial I_{m}} & \frac{\partial f_{4}}{\partial R_{m}} & \frac{\partial f_{4}}{\partial E_{v}} & \frac{\partial f_{4}}{\partial I_{v}} \\ \frac{\partial f_{5}}{\partial E_{m}} & \frac{\partial f_{5}}{\partial I_{m}} & \frac{\partial f_{5}}{\partial R_{m}} & \frac{\partial f_{5}}{\partial E_{v}} & \frac{\partial f_{5}}{\partial I_{v}} \end{bmatrix}$$

$$(13)$$

The eigen values of FV^{-1} gives:

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & \frac{(1-\ell)\pi_{h}\mu_{\nu}\omega_{m}a(1-\varepsilon_{1})}{\pi_{\nu}\mu_{h}} \\ 0 & 0 & 0 & 0 & \frac{\varepsilon_{1}\omega_{m}a(1-\ell)\pi_{h}\mu_{\nu}}{\pi_{\nu}\mu_{h}} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\omega_{\nu}b\pi_{\nu}\mu_{\nu}}{(1-\ell)\pi_{h}\mu_{\nu}} & \frac{\omega_{\nu}b\eta_{1}\pi_{\nu}\mu_{\nu}}{(1-\ell)\pi_{h}\mu_{\nu}} & 0 & 0 & 0 \\ \frac{\omega_{\nu}b\pi_{\nu}\mu_{\nu}}{(1-\ell)\pi_{h}\mu_{\nu}} & \frac{\omega_{\nu}b\eta_{1}\pi_{\nu}\mu_{\nu}}{(1-\ell)\pi_{h}\mu_{\nu}} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ -\tau_{3} & -\tau_{2} & 0 & 0 & 0 \\ 0 & 0 & K_{13} & K_{14} & 0 \\ 0 & 0 & 0 & -\sigma_{\nu} & \mu_{\nu} \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & \frac{(1-\varepsilon_{1})\sigma_{\nu}\omega_{m}a(1-\ell)\pi_{h}}{\pi_{\nu}\mu_{k}} & \frac{(1-\varepsilon_{1})\omega_{m}a(1-\ell)\pi_{h}}{\pi_{\nu}\mu_{h}} \\ 0 & 0 & 0 & \frac{\varepsilon_{1}\omega_{m}a(1-\ell)\pi_{h}\sigma_{\nu}}{\pi_{\nu}\mu_{k}} & \frac{\varepsilon_{1}\omega_{m}a(1-\ell)\pi_{h}}{\pi_{\nu}\mu_{h}} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\omega_{l}b\pi_{\nu}\mu_{h}}{(1-\ell)\pi_{h}\mu_{\nu}K_{11}} & \frac{\omega_{l}b\pi_{\nu}\mu_{l}\eta_{l}}{(1-\ell)\pi_{h}\mu_{\nu}K_{12}} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$(15)$$

The above equation is equation (16), Then, the eigenvalues which is the basic reproduction number for malaria model in this research gives:

$$\begin{array}{c} 0 \\ 0 \\ \frac{0}{\sqrt{\pi_{h}\mu_{v}K_{11}K_{12}\pi_{v}K_{14}\omega_{v}b\pi_{v}\omega_{m}a\pi_{h}\sigma_{v}(K_{12}-K_{12}\varepsilon_{1}+\eta_{1}k_{1}-\eta_{1}k_{1}\varepsilon_{1}+\eta_{1}\varepsilon_{1}K_{11})}}{\pi_{h}\mu_{v}K_{11}K_{12}\pi_{v}K_{14}} \\ \frac{\sqrt{\pi_{h}\mu_{v}K_{11}K_{12}\pi_{v}K_{14}\omega_{v}b\pi_{v}\omega_{m}a\pi_{h}\sigma_{v}(K_{12}-K_{12}\varepsilon_{1}+\eta_{1}k_{1}-\eta_{1}k_{1}\varepsilon_{1}+\eta_{1}\varepsilon_{1}K_{11})}}{\pi_{h}\mu_{v}K_{11}K_{12}\pi_{v}K_{14}} \\ \end{array} \right]$$
(17)
where, $K_{11} = (\mu_{H} + k_{1} + \tau_{3}), \ K_{12} = (\mu_{H} + \delta + \tau_{2}), \ K_{13} = (\mu_{H} + \beta), \ K_{14} = (\mu_{v} + \sigma_{v})$

The threshold quantity R_{0m} is the basic reproduction number of the malaria model in this work. It measures the average number of new secondary infections generated by a single infected individual in his or her infective period in a susceptible population (Adewale *et al*, 2009).

3.2. Local Stability of DFE of the Model

Theorem 2: The disease free equilibrium of the system (1-7) is globally asymptotically stable whenever $(1 - \varepsilon_1) \lambda^{**} {}_{_{H}} S^{**}_{_{H}}$

$$E_m^{**} = \frac{(V - V_1)V - mV_H}{K_{11}} \text{ and unstable when } R_{0m} > 1.$$

Proof: It follows that $S_H = N_H^* - E_M - I_M - R_M$ and $S_V = N_V^* - E_V - I_V$ at steady state. The proof is based on using the comparison theorem (Lakshantham *et al*, 1989) to prove global stability. The rate of change of the variables representing the infected components of malaria system can be written as follows:

$$\frac{dE_m}{dt} = (1 - \varepsilon_1)\lambda_m (N_H^* - E_M - I_M - R_M) - K_{11}E_m$$
(18)

$$\frac{dI_m}{dt} = \varepsilon_1 \lambda_m (N_H^* - E_M - I_H - R_M) - K_{12} I_m + \kappa_1 E_m$$
⁽¹⁹⁾

$$\frac{dR_m}{dt} = \tau_2 I_m - K_{13} R_m + \tau_3 E_m \tag{20}$$

$$\frac{dE_{v}}{dt} = \lambda_{v} (N_{v}^{*} - E_{v} - I_{v}) - K_{14} E_{v}$$
(21)

$$\frac{dI_{\nu}}{dt} = \sigma_{\nu}E_{\nu} - \mu_{\nu}I_{\nu}$$
⁽²²⁾

Here, the invariance region is given by:

$$D^{*} = \left\{ \left(E_{m}, I_{m}, R_{m}.E_{\nu}, I_{\nu} \right) \in R^{5}_{+} : E_{m}, I_{m}, R_{m}.E_{\nu}, I_{\nu} \leq N^{+} \right\}$$
(23)

Then, the associated reproduction number denoted by R_{0m} , where

$$R_{0m} = \frac{\sqrt{\pi_{h}\mu_{v}K_{11}K_{12}\pi_{v}K_{14}\omega_{v}b\pi_{v}\omega_{m}a\pi_{h}\sigma_{v}(K_{12} - K_{12}\varepsilon_{1} + \eta_{1}k_{1} - \eta_{1}k_{1}\varepsilon_{1} + \eta_{1}\varepsilon_{1}K_{11})}{\pi_{h}\mu_{v}K_{11}K_{12}\pi_{v}K_{14}}$$
Where $K_{n} = \kappa_{n} + \kappa_{n}$

Where $K_{11} = \kappa_1 + \mu_H + \tau_3$, $K_{12} = \mu_H + \delta_2 + \tau_2$, $K_{13} = \mu_H + \beta$, $K_{14} = \mu_V + \sigma_V$ The disease free equilibrium of the model is globally asymptotically stable in D^* if $R_{0m} < 1$.

$$\begin{bmatrix} \frac{dE_m}{dt} \\ \frac{dI_m}{dt} \\ \frac{dE_w}{dt} \\ \frac{dE_w}{dt} \\ \frac{dI_w}{dt} \\ \frac{dI_w}{dt}$$

$$F - V = \begin{bmatrix} -K_{11} & 0 & 0 & 0 & \frac{\pi_{h}\mu_{\nu}\omega_{m}a(1-\varepsilon_{1})}{\pi_{\nu}\mu_{h}} \\ -k_{1} & -K_{12} & 0 & 0 & \frac{\varepsilon_{1}\omega_{m}a\pi_{h}\mu_{\nu}}{\pi_{\nu}\mu_{h}} \\ \tau_{3} & \tau_{2} & -K_{13} & 0 & 0 \\ \frac{\omega_{\nu}b\pi_{\nu}\mu_{\nu}}{\pi_{h}\mu_{\nu}} & \frac{\omega_{\nu}b\eta_{1}\pi_{\nu}\mu_{\nu}}{\pi_{h}\mu_{\nu}} & 0 & -K_{14} & 0 \\ 0 & 0 & 0 & \sigma_{\nu} & -\mu_{\nu} \end{bmatrix} \begin{bmatrix} \frac{dE_{m}}{dt} \\ \frac{dI_{m}}{dt} \\ \frac{dE_{m}}{dt} \\ \frac{d$$

All eigenvalues of matrix (F-V) have negative real parts (Castillo-Chavez *et al* (2002), Van den Driessche and Watmough (2002))

$$\begin{vmatrix} -K_{11} - \lambda & 0 & 0 & 0 & \frac{\pi_{h} \mu_{\nu} \omega_{m} a(1 - \varepsilon_{1})}{\pi_{\nu} \mu_{h}} \\ -k_{1} & -K_{12} - \lambda & 0 & 0 & \frac{\varepsilon_{1} \omega_{m} a \pi_{h} \mu_{\nu}}{\pi_{\nu} \mu_{h}} \\ \tau_{3} & \tau_{2} & -K_{13} - \lambda & 0 & 0 \\ \frac{\omega_{\nu} b \pi_{\nu} \mu_{\nu}}{\pi_{h} \mu_{\nu}} & \frac{\omega_{\nu} b \eta_{1} \pi_{\nu} \mu_{\nu}}{\pi_{h} \mu_{\nu}} & 0 & -K_{14} - \lambda & 0 \\ 0 & 0 & 0 & \sigma_{\nu} & -\mu_{\nu} - \lambda \end{vmatrix} = 0$$
(29)

From the matrix above, it gives

$$\lambda^{5} - (-\mu_{\nu} - K_{14} - K_{13} - K_{12} - K_{11})\lambda^{4} - (-K_{11}K_{12} - K_{11}K_{13} - K_{11}K_{14} - K_{11}\mu_{\nu} - K_{12}K_{13} - K_{12}K_{14} - K_{12}\mu_{\nu} - K_{13}K_{14} - K_{13}\mu_{\nu} - K_{14}\mu_{\nu})\lambda^{3} - (ab\epsilon_{1}\eta_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} - ab\epsilon_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} + ab\omega_{m}\omega_{\nu}\sigma_{\nu} - K_{11}K_{12}K_{13} - K_{11}K_{12}K_{14} - K_{11}K_{12}\mu_{\nu} - K_{11}K_{13}K_{14} - K_{11}K_{13}\mu_{\nu} - K_{11}K_{14}\mu_{\nu} - K_{12}K_{13}K_{14} - K_{12}K_{13}\mu_{\nu} - K_{12}K_{14}\mu_{\nu} - K_{13}K_{14}\mu_{\nu})\lambda^{2} - (abK_{11}\epsilon_{1}\eta_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} + abK_{13}\epsilon_{1}\eta_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} - ab\epsilon_{1}\eta_{1}\kappa_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} - abK_{12}\epsilon_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} - abK_{13}\epsilon_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} + ab\eta_{1}\kappa_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} + abK_{12}\omega_{m}\omega_{\nu}\sigma_{\nu} + abK_{13}\omega_{m}\omega_{\nu}\sigma_{\nu} - K_{11}K_{12}K_{13}K_{14} - K_{11}K_{12}K_{13}\mu_{\nu} - K_{11}K_{12}K_{14}\mu_{\nu} - K_{11}K_{13}K_{14}\mu_{\nu} - K_{12}K_{13}K_{14}\mu_{\nu})\lambda - abK_{11}K_{13}\epsilon_{1}\eta_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} + abK_{13}\epsilon_{1}\eta_{1}\kappa_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} + abK_{12}K_{13}\epsilon_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} - abK_{13}\eta_{1}\kappa_{1}\omega_{m}\omega_{\nu}\sigma_{\nu} - abK_{12}K_{13}\omega_{m}\omega_{\nu}\sigma_{\nu} + \mu_{\nu}K_{14}K_{13}K_{12}K_{11}$$
(30)

Thus, from the malaria model equation, the characteristic equation given by $\lambda^5 + a_5 \lambda^4 + a_4 \lambda^3 + a_3 \lambda^2 + a_2 \lambda^1 + a_1 = 0$ Applying Routh–Hurwitz criteria of order five (5).

IV. BIFURCATION ANALYSIS FOR THE MODEL Here the type of bifurcation that malaria model in this research exhibit shall be determined. Let $S_H = x_1, E_m = x_2, I_m = x_3, R_m = x_4, S_V = x_5, E_V = x_6, I_V = x_7$

So that
$$N_H = x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7$$
 (31)

By using the vector notion, $X = (x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7)^T$, the model can be written in the form. dV

$$\frac{dX}{dt} = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)^T$$

$$\frac{dx_1}{dt} = f_1 = \pi_H - \frac{\omega_m a x_1 x_7}{x_5 + x_6 + x_7} + \beta x_4 - \mu_H x_1$$

$$\frac{dx_2}{dt} = f_2 = \frac{(1 - \varepsilon_1)\omega_m a x_1 x_7}{x_5 + x_6 + x_7} - K_{11} x_2$$

$$\frac{dx_3}{dt} = f_3 = \frac{\varepsilon_1 \omega_m a x_1 x_7}{x_5 + x_6 + x_7} - K_{12} x_3 + \kappa_1 x_2$$

$$\frac{dx_4}{dt} = f_4 = \tau_2 x_3 - K_{13} x_4 - \tau_3 x_2$$

$$\frac{dx_5}{dt} = f_5 = \pi_V - \frac{\omega_V b(x_2 + \eta_1 x_3) x_5}{x_1 + x_2 + x_3 + x_4} - \mu_V x_5$$

$$\frac{dx_6}{dt} = f_6 = \frac{\omega_V b(x_2 + \eta_1 x_3) x_5}{x_1 + x_2 + x_3 + x_4} - K_{14} x_6$$

$$\frac{dx_7}{dt} = f_7 = \sigma_V x_6 - \mu_V x_7$$
the Jacobian of (32) is given by
$$(32)$$

$$J(\varepsilon_{0}) = \begin{pmatrix} -\mu_{h} & 0 & 0 & \beta & 0 & 0 & \frac{-\omega_{m}a\pi_{h}\mu_{V}}{\pi_{V}\mu_{h}} \\ 0 & -K_{11} & 0 & 0 & 0 & 0 & \frac{(1-\varepsilon_{1})\omega_{m}a\pi_{h}\mu_{V}}{\pi_{V}\mu_{h}} \\ 0 & \kappa_{1} & -K_{12} & 0 & 0 & 0 & \frac{\varepsilon_{1}\omega_{m}a\pi_{h}\mu_{V}}{\pi_{V}\mu_{h}} \\ 0 & \tau_{3} & \tau_{2} & -K_{13} & 0 & 0 & 0 \\ 0 & \frac{-\omega_{V}b\pi_{V}\mu_{h}}{\pi_{h}\mu_{V}} & \frac{-\omega_{V}b\pi_{V}\mu_{h}\eta_{1}}{\pi_{h}\mu_{V}} & 0 & -\mu_{V} & 0 & 0 \\ 0 & \frac{\omega_{V}b\pi_{V}\mu_{h}}{\pi_{h}\mu_{V}} & \frac{\omega_{V}b\pi_{V}\mu_{h}\eta_{1}}{\pi_{h}\mu_{V}} & 0 & 0 & -K_{14} & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_{V} & -\mu_{V} \end{pmatrix}$$
(33)

where

$$K_{11} = (\mu_H + \kappa_1 + \tau_3)$$

$$K_{12} = (\mu_H + \delta_2 + \tau_2)$$

$$K_{13} = (\mu_H + \beta)$$

$$K_{14} = (\mu_V + \sigma_V)$$

Recall that

$$R_{0m} = \frac{\sqrt{\pi_h \mu_v K_{11} K_{12} \pi_v K_{14} \omega_v b \pi_v \omega_m a \pi_h \sigma_v (K_{12} - K_{12} \varepsilon_1 + \eta_1 k_1 - \eta_1 k_1 \varepsilon_1 + \eta_1 \varepsilon_1 K_{11})}{\pi_h \mu_v K_{11} K_{12} \pi_v K_{14}}$$

 $\omega_m = \omega_m^*$ is choose as bifurcation parameter where the case $R_{OM} = 1$ shall be considered and solved for ω_m

Then,
$$\omega_m = \omega_m^* = \frac{(\pi_H \mu_V K_{11} K_{12} \pi_V K_{14})^2}{\pi_H \mu_V K_{11} K_{12} K_{14} \pi_V \omega_V a \pi_H \sigma_V (K_{12} - K_{12} \varepsilon_1 + \eta_1 \kappa_1 - \eta_1 \kappa_1 \varepsilon_1 + \eta \varepsilon_1 K_{11})}$$

To analyze the dynamics of (32) we compute the eigenvectors of the jacobian of (33) at the DFE. It can be shown that this jacobian has a night eigenvector given by $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)$

$$\begin{bmatrix} -\mu & 0 & 0 & \beta & 0 & 0 & -\omega_{m} aG \\ 0 & -K_{11} & 0 & 0 & 0 & 0 & (1-c_{1})\omega_{m} aG \\ 0 & \tau_{3} & \tau_{2} & -K_{13} & 0 & 0 & 0 \\ 0 & -\omega_{v} by & -\omega_{v} b\eta_{1} y & 0 & -\mu_{v} & 0 & 0 \\ 0 & \omega_{v} by & \omega_{v} b\eta_{1} y & 0 & 0 & -K_{14} & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_{v} & -\mu_{v} \end{bmatrix} \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$
(34)
where $y = \frac{\mu_{h} \pi_{v}}{\pi_{h} \mu_{v}}$ and $G = \frac{\mu_{v} \pi_{h}}{\pi_{v} \mu_{h}}$
Then,
 $-\mu_{h} w_{1} + \beta w_{4} - G \omega_{m} aw_{7} = 0$
 $-K_{11} w_{2} + (1-\varepsilon_{1})\omega_{m} aw_{7} G = 0$
 $K_{10} w_{2} - K_{12} w_{3} + \varepsilon_{10} \omega_{m} w_{7} = 0$
 $-\omega_{v} by w_{2} - \omega_{v} by w_{3} - \mu_{v} w_{5} = 0$
 $\omega_{v} by w_{2} + \omega_{v} b\eta_{1} yw_{3} - K_{14} w_{6} = 0$
 $\sigma_{v} w_{6} - \mu_{v} w_{7} = 0$ (35)
Solving equation (35) gives
 $w_{1} = \frac{\beta w_{4} - \omega_{m} aw_{7}G}{\mu_{h}}$
 $w_{2} = \frac{(1-\varepsilon_{1})\omega_{m} aw_{7}G}{K_{11}} , w_{3} = \frac{\kappa_{1} w_{2} + w_{7} \varepsilon_{1} \omega_{m} aG}{K_{12}}$
 $w_{4} = \frac{\tau_{3} w_{2} + \tau_{2} w_{3}}{K_{13}} , w_{5} = \frac{-\omega_{v} by w_{2} - \omega_{v} by \eta_{1} w_{3}}{\mu_{v}}$
 $w_{6} = \frac{\omega_{v} by w_{2} + \omega_{v} b\eta_{1} yw_{3}}{K_{14}}$

The jacobian J_m has left eigenvector (associated with the zero eigenvalue) given by

$$V = (v_{1}, v_{2}, v_{3}, v_{4}, v_{5}, v_{6}, v_{7})$$

$$J_{m} = \begin{bmatrix} -\mu_{h} & 0 & 0 & 0 & 0 & 0 \\ 0 & -K_{11} & k_{1} & \tau_{3} & -\omega_{v}by & \omega_{v}by & 0 \\ 0 & 0 & -K_{12} & \tau_{2} & -\omega_{v}b\eta_{1}y & \omega_{v}b\eta_{1}y & 0 \\ \beta & 0 & 0 & -K_{13} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_{v} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -K_{14} & \sigma_{v} \\ -\omega_{m}aG & (1-\varepsilon_{1})\omega_{m}aG & \varepsilon_{1}\omega_{m}aG & 0 & 0 & 0 & -\mu_{v} \end{bmatrix} \begin{bmatrix} 0 \\ v_{1} \\ v_{2} \\ v_{3} \\ v_{4} \\ v_{5} \\ v_{6} \\ v_{7} \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$
(36)

$$-\mu v_{1} = 0 \Rightarrow v_{1} = 0$$

$$-K_{11}v_{2} + k_{1}v_{3} + \tau_{3}v_{4} - \omega_{v}byv_{5} + \omega_{v}byv_{6} = 0$$

$$-K_{12}v_{3} + \tau_{2}v_{4} - \omega_{v}b\eta_{1}yv_{5} + \omega_{v}b\eta_{1}yv_{6} = 0$$

$$\beta v_{1} - K_{13}v_{4} = 0 \Rightarrow v_{4} = 0$$

$$-\mu_{v}v_{5} = 0 \Rightarrow v_{5} = 0$$

$$-K_{14}v_{6} + \sigma_{v}v_{7} = 0$$

$$-\omega_{m}aGv_{1} + (1 - \varepsilon_{1})\omega_{m}aGv_{2} + \varepsilon_{1}\omega_{m}aGv_{3} - \mu_{v}v_{7} = 0$$

$$\therefore v_{1} = 0; v_{4} = 0; v_{5} = 0.$$
Solving equation (37) we have
$$v_{2} = \frac{-(\omega_{v}byv_{6} + \kappa_{1}v_{3})}{K_{11}}$$

$$v_{3} = \frac{\omega_{v}by\eta_{1}v_{6}}{K_{13}}$$

$$v_{6} = \frac{\sigma_{v}v_{7}}{K_{14}}$$

$$v_{7} = \frac{(1 - \varepsilon_{1})\omega_{m}aGv_{2} + \varepsilon_{1}\omega_{m}aGv_{3}}{\mu_{v}}$$

$$v_{7} = v_{2}$$
 and $v_{7} = v_{2}$ are free left eigen vector.

 $v_3 = v_3$ and $v_6 = v_6$ are free left eigen vector.

Where
$$y = \frac{\mu_H \pi_V}{\mu_V \pi_H}$$

 $G = \frac{\mu_V \pi_H}{\mu_H \pi_V}, K_{11} = (\mu_H + \kappa_1 + \tau_3), K_{12} = (\mu_H + \delta + \tau_2), K_{13} = (\mu_H + \beta), K_{14} = (\mu_V + \sigma_V)$

According to coefficient a and b as defined by Castillo Chavez and Song, it follows that for the system (1-7), the associated non-zero partial derivatives at disease free equilibrium are calculated thus at

$$a = \sum_{i=j=1,k=1}^{7} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} \quad and \quad b = \sum_{i=j=1}^{7} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \omega_m^*}$$

$$\frac{v_2 w_1 w_5 \partial^2 f_2}{\partial x_1 \partial x_5} = 0, \frac{v_2 w_1 w_6 \partial^2 f_2}{\partial x_1 \partial x_6} = 0, \frac{v_2 w_1 w_2 \partial^2 f_2}{\partial x_1 \partial x_7} = \frac{v_2 w_1 w_7 (1 - \varepsilon_1) \omega_m a \mu_v}{\pi_v}$$

$$\frac{v_2 w_5 w_1 \partial^2 f_2}{\partial x_5 \partial x_1} = 0, \frac{v_2 w_5 w_2 \partial^2 f_2}{\partial x_5 \partial x_2} = 0, \frac{v_2 w_5 w_3 \partial^2 f_2}{\partial x_5 \partial x_3} = 0, \frac{v_2 w_5 w_7 \partial^2 f_2}{\partial x_5 \partial x_7} = \frac{-v_2 w_5 w_7 (1 - \varepsilon_1) \omega_m a \pi_h \mu_v^2}{\mu_h \pi_v^2}$$

$$\frac{v_2 w_6 w_1 \partial^2 f_2}{\partial x_6 \partial x_1} = 0, \frac{v_2 w_6 w_2 \partial^2 f_2}{\partial x_6 \partial x_2} = 0, \frac{v_2 w_6 w_3 \partial^2 f_2}{\partial x_6 \partial x_3} = 0, \frac{v_2 w_6 w_7 \partial^2 f_2}{\partial x_6 \partial x_7} = \frac{-v_2 w_6 w_7 (1 - \varepsilon_1) \omega_m a \pi_h \mu_v^2}{\mu_h \pi_v^2}$$

$$\frac{v_2 w_7 w_1 \partial^2 f_2}{\partial x_7 \partial x_1} = \frac{v_2 w_7 w_1 (1 - \varepsilon_1) \omega_m a \mu_v}{\pi_v} 0, \frac{v_2 w_7 w_2 \partial^2 f_2}{\partial x_7 \partial x_2} = 0, \frac{v_2 w_7 w_3 \partial^2 f_2}{\partial x_7 \partial x_3} = 0, \frac{v_2 w_7 w_4 \partial^2 f_2}{\partial x_7 \partial x_4} = 0$$

$$\frac{v_2 w_7 w_5 \partial^2 f_2}{\partial x_7 \partial x_5} = -\frac{v_2 w_7 w_5 (1 - \varepsilon_1) \omega_m a \mu_v^2 \pi_h}{\pi_v^2 \mu_h} 0, \frac{v_2 w_7 w_6 \partial^2 f_2}{\partial x_7 \partial x_6} = \frac{-v_2 w_7 w_6 (1 - \varepsilon_1) \omega_m a \pi_h \mu_v^2}{\pi_v^2 \mu_h}$$
When k = 3
$$\frac{v_3 w_1^2 \partial^2 f_3}{\partial x_1^2} = 0, \frac{v_3 w_1 w_2 \partial^2 f_3}{\partial x_1 \partial x_2} = 0, \frac{v_3 w_1 w_3 \partial^2 f_3}{\partial x_1 \partial x_3} = 0, \frac{v_3 w_1 w_4 \partial^2 f_3}{\partial x_1 \partial x_4} = 0$$

$$\frac{v_{3}w_{1}w_{5}\partial^{2}f_{3}}{\partial x_{1}\partial x_{5}} = 0, \frac{v_{3}w_{1}w_{6}\partial^{2}f_{3}}{\partial x_{1}\partial x_{6}} = 0, \frac{v_{3}w_{1}w_{7}\partial^{2}f_{3}}{\partial x_{1}\partial x_{7}} = \frac{v_{3}w_{1}w_{7}\mathcal{E}_{1}\omega_{m}a\mu_{V}}{\pi_{V}}$$

$$\frac{v_{3}w_{5}w_{4}\partial^{2}f_{3}}{\partial x_{5}\partial x_{4}} = 0, \frac{v_{3}w_{5}w_{6}\partial^{2}f_{3}}{\partial x_{5}\partial x_{6}} = 0, \frac{v_{3}w_{5}w_{7}\partial^{2}f_{3}}{\partial x_{5}\partial x_{7}} = \frac{-v_{3}w_{5}w_{7}\mathcal{E}_{1}\omega_{m}a\pi_{h}\mu_{v}^{2}}{\mu_{h}\pi_{v}^{2}}$$

$$\frac{v_{3}w_{6}w_{4}\partial^{2}f_{3}}{\partial x_{6}\partial x_{4}} = 0, \frac{v_{3}w_{6}w_{5}\partial^{2}f_{3}}{\partial x_{6}\partial x_{5}} = 0, \frac{v_{3}w_{6}w_{7}\partial^{2}f_{3}}{\partial x_{6}\partial x_{7}} = \frac{-v_{3}w_{6}w_{7}\mathcal{E}_{1}\omega_{m}a\pi_{h}\mu_{v}^{2}}{\mu_{h}\pi_{v}^{2}}$$

$$\frac{v_{3}w_{7}w_{1}\partial^{2}f_{3}}{\partial x_{7}\partial x_{1}} = \frac{w_{7}w_{1}v_{3}\mathcal{E}_{1}\omega_{m}a\mu_{v}}{\pi_{v}}, \frac{v_{3}w_{7}w_{2}\partial^{2}f_{3}}{\partial x_{7}\partial x_{2}} = 0, \frac{v_{3}w_{7}w_{3}\partial^{2}f_{3}}{\partial x_{7}\partial x_{3}} = 0, \frac{v_{3}w_{7}w_{4}\partial^{2}f_{3}}{\partial x_{7}\partial x_{4}} = 0$$

$$\frac{v_{3}w_{7}w_{5}\partial^{2}f_{3}}{\partial x_{7}\partial x_{5}} = \frac{-v_{3}w_{7}w_{5}\mathcal{E}_{1}\omega_{m}a\pi_{h}\mu_{v}^{2}}{\mu_{h}\pi_{v}^{2}}, \frac{v_{3}w_{7}w_{6}\partial^{2}f_{3}}{\partial x_{7}\partial x_{6}} = \frac{-v_{3}w_{7}w_{6}\mathcal{E}_{1}\omega_{m}a\pi_{h}\mu_{v}^{2}}{\mu_{h}\pi_{v}^{2}}, \frac{v_{3}w_{7}w_{6}\partial^{2}f_{3}}{\partial x_{7}\partial x_{5}} = \frac{-v_{3}w_{7}w_{6}\mathcal{E}_{1}\omega_{m}a\pi_{h}\mu_{v}^{2}}{\mu_{h}\pi_{v}^{2}}, \frac{v_{3}w_{7}w_{6}\partial^{2}f_{3}}{\partial x_{7}\partial x_{6}} = \frac{-v_{3}w_{7}w_{6}\mathcal{E}_{1}\omega_{m}a\pi_{h}\mu_{v}^{2}}{\mu_{h}\pi_{v}^{2}}, \frac{v_{3}w_{7}w_{6}\partial^{2}f_{6}}{\partial x_{2}\partial^{2}f_{6}} = \frac{-v_{3}w_{7}w_{7}\mathcal{E}_{1}\omega_{m}a\pi_{h}\mu_{v}^{2}}{\mu_{h}\pi_{v}^{2}}, \frac{w_{3}w_{7}w_{6}\partial^{2}f_{6}}{\partial x_{2}\partial^{2}f_{6}} = 0, \frac{w_{6}w_{2}w_{3}\partial^{2}f_{6}}{\partial x_{2}\partial x_{1}} = 0, \frac{w_{6}w_{3}w_{2}\partial^{2}f_{6}}{\partial x_{2}\partial x_{1}} = 0, \frac{w_{6}w_{3}w_{2}\partial^{2}f_{6}}{\partial x_{2}\partial x_{1}} = 0, \frac{w_{6}w_{3}w_{2}\partial^{2}f_{6}}{\partial x_{3}\partial x_{1}} = 0, \frac{w_{6}w_{3}w_{2}\partial^{2}f_{6}}$$

Since the first partial derivatives of f_6 with respect to x_4, x_5, x_5, x_6 and x_7 give constant values then there second partial derivatives are zeros. Therefore, a shall be computed thus

$$a = \sum_{i=j=1,k=1}^{7} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}$$

= $\frac{(1-\varepsilon_1)\omega_m a\mu_v v_2}{\pi_v} (2w_1 w_7 - \frac{2w_5 w_7 \pi_h \mu_v}{\mu_h \pi_v} - \frac{2w_6 w_7 \pi_h \mu_v}{\mu_h \pi_v} - \frac{2w_7^2 \pi_h \mu_v}{\mu_h \pi_v}) +$
 $\frac{\varepsilon_1 \omega_m a\mu_v v_3}{\pi_v} (2w_1 w_7 - \frac{2w_5 w_7 \pi_h \mu_v}{\mu_h \pi_v} - \frac{2w_6 w_7 \pi_h \mu_v}{\mu_h \pi_v} - \frac{2w_7^2 \pi_h \mu_v}{\mu_h \pi_v}) + \frac{\omega_v b\mu_h v_6}{\pi_h} (2w_2 w_3 - 2w_3^2 \eta_1)$
= $\frac{2\omega_m a\mu_v w_7}{\pi_v} (v_2 - \varepsilon_1 v_2 - \varepsilon_1 v_3) (w_1 - \frac{\pi_h \mu_v}{\mu_h \pi_v} (w_5 - w_6 - w_7)) + \frac{2\omega_v b\mu_h v_6 w_3}{\pi_h} (w_2 - w_3 \eta_1)$ (38)

Therefore, a > 0

 ∂x_3^2

The associated non vanishing derivatives of f can be shown in order to determine the sign of b as thus

$$b = \sum_{i=k=1}^{7} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \omega_m^*}$$

Recall that if $v_1 = v_4 = v_5 = 0$, then k = 2, 3, 6 and 7 shall be computed. For k=2,

$$v_2 w_5 \frac{\partial^2 f_2}{\partial x_5 \partial \omega_m^*} = 0, v_2 w_6 \frac{\partial^2 f_2}{\partial x_6 \partial \omega_m^*} = 0 \text{ and } v_2 w_7 \frac{\partial^2 f_2}{\partial x_7 \partial \omega_m^*} = \frac{v_2 w_7 (1 - \varepsilon_1) \omega_m a \pi_h \mu_v}{\pi_v \mu_h}$$

For k=3

$$v_{3}w_{1}\frac{\partial^{2}f_{3}}{\partial x_{1}\partial \omega_{m}^{*}} = 0 \cdot v_{3}w_{2}\frac{\partial^{2}f_{3}}{\partial x_{2}\partial \omega_{m}^{*}} = 0, v_{3}w_{3}\frac{\partial^{2}f_{3}}{\partial x_{3}\partial \omega_{m}^{*}} = 0 \cdot v_{3}w_{4}\frac{\partial^{2}f_{3}}{\partial x_{4}\partial \omega_{m}^{*}} = 0, v_{3}w_{5}\frac{\partial^{2}f_{3}}{\partial x_{5}\partial \omega_{m}^{*}} = 0, v_{3}w_{5}\frac{\partial^{2}f_{5}}{\partial x_{5}\partial \omega_{m}^{*}} = 0, v_{5}\frac{\partial^{2}f_{5}}{\partial x_{5}\partial \omega_{m}^{*}} = 0, v_{5}$$

When k = 6 and 7, the second partial derivatives give zeroes because their first partial derivatives did not contain ω_m^*

Hence,

$$b = \frac{v_2 w_7 (1 - \varepsilon_1) \omega_m a \pi_h \mu_v}{\pi_v \mu_h} + \frac{v_3 w_7 \varepsilon_1 a \pi_h \mu_v}{\pi_v \mu_h}$$
$$b = \frac{a w_7 \pi_h \mu_v}{\pi_v \mu_h} (\varepsilon_1 v_3 + v_2 \omega_m - \varepsilon_1 v_2 \omega_m)) > 0.$$

By finding the associated non-zero partial derivatives at disease free equilibrium, it follows that; the malaria model exhibits backward bifurcation for a > 0 and b > 0.

The reason behind it is that, it could be seen that the fast Progressor (\mathcal{E}_1) can never be greater than one because of the fact that it was based on the fractional rate per time in which the time could be in seconds or minutes.

V. SENSITIVITY ANALYSIS OF THE MODEL

The sensitivity analysis of the basic reproduction number parameters is carried out in order to know how crucial each parameter is to the disease transmission and to determine which of parameters causes most reduction in R_{0M} . This analysis will help in knowing the parameters to be targeted for intervention strategies in the control or prevention of the malaria disease in the society.

The sensitivity index is defined as the ratio of the relative change in R_{0M} to the relative change in the parameter say "w", its sensitivity value shall be calculated as $X_W^{R_{0M}} = \frac{\partial R_{0M}}{\partial W} \times \frac{W}{R_{0M}}$. This was carried out for each parameter found in the basic reproduction number. Thus, the constitution to be in the basic reproduction number.

each parameter found in the basic reproduction number. Thus, the sensitivity table is

| PARAMETERS | SENSITIVITY VALUES |
|-----------------------|--------------------|
| μ_H | -0.0012602552 |
| τ ₂ | -0.001341720874 |
| $	au_3$ | -0.00449626227 |
| K ₁ | -0.4524307633 |
| δ_2 | -0.00000463732 |
| η_1 | 0.001374637745 |
| A | 0.49999999999 |
| μ_{ν} | -0.7941539729 |
| σ_{v} | 0.2941539727 |
| ω _m | 0.500000000 |
| <i>E</i> ₂ | -0.3323630596 |
| ω_V | 0.4999999999999 |
| В | 0.4999999999 |

| TABLE 4.1: Numerical Sensitivity | of Malaria with Values |
|----------------------------------|------------------------|
|----------------------------------|------------------------|

The table 4.1 revealed that a contact rate ($\omega_M = 0.5$) of malaria from infected mosquito to innocent human has greater influence in the spread of malaria disease. Malaria which is transmitted by female anopheles mosquitoes that bite malaria infected human serves as vector for malaria. Hence, malaria can be reduced or eliminated if there is provision of prophylactics for human being so that they would not be able to contact malaria. Also, nursing of predators that will feed on mosquito will lead to reduction in population of mosquito as shown on the table 4.2 ($\mu_V = -0.7941539729$) have greater influence in reducing the spread of malaria disease, although the death rate value of mosquito is negative but the higher the death rate, the lesser the basic reproduction number which implies that the lower the rate of spreading malaria disease in the society. Also the transmission rate of malaria from infected mosquito to innocent human (B = 0.4999) is the same with biting rate of susceptible mosquito on infected humans with malaria (A = 0.4999) because once infected mosquitoes are many in the society, there will be rapid spread of malaria in such society. More so, increase in both mosquito biting rate and the rate of transmitting malaria on basic reproduction number for malaria (R_{0M}), the higher the value of basic reproduction number which leads to endemicity of malaria disease in the society and vice versa. The figures below shows the output of our simulations



Figure 5.2: Graph of R_{0M} against Mosquito death rate (μ_4).



Figure 5.3: Graph of I_M against time (t) with different values of ω_M .



Figure 5.4: Graph of S_H against time (t) with different values of ω_M .



Figure 5.5: Graph of E_M against time (t) with different values of ω_M .



Figure 5.6: Graph of E_M against time (t) with different values of τ_3 .



Figure 5.7: Graph of I_V against time (t) with different values of μ_V .







Figure 5.9: Graph of S_V against time (t) with different values of μ_V .



Figure 5.10: Susceptible human for malaria against basic reproduction number of malaria.







Figure 5.12: Exposed human for malaria against basic reproduction number of malaria.



Figure 5.13: Recovered human for malaria against basic reproduction number of malaria.



Figure 5.14: Infected mosquitoes with malaria against basic reproduction number of malaria.

VI. DISCUSSION OF RESULT

Figure 5.3 shows the effect of increment of malaria contact rate on the infected individual with malaria with respect to time in months. We noticed that when the contact rate was 1.033, the infected curve rises differently from other values. So, the higher the contact rate, the more the number of infected individuals and vice versa. Also, in **Figure 5.4**, The graph shows the different curves of the susceptible individual at a different rate of malaria contact with respect to time (t) in months. We observed that the higher the rate of contact, the lower the curve of the susceptible individual in the class of malaria. The curve is expected because malaria is a reinfected disease that cannot be totally eliminated in the life of human beings, especially in Africa. So, as the susceptible individuals contact the disease, the population in this class will reduce. The lesser the contact rate of malaria in the susceptible class, the more the population of the susceptible class. In **Figure 5.5**: This graph reveals the effect of the contact rate of malaria on the exposed class of malaria. At the time (t) equals zero, the exposed class is assumed to be at the maximum of 2000, and the tested rate of this class gradually fell from the shape of

the graph. It implies that the increment in contact rate yields decreases in the class of exposed individual to malaria.

This graph (5.6) describes how treatment on infected class affects exposed class of malaria. It shows that the population of exposed classes at the peak stage starts to fall in more significant numbers as an infected class treatment increases. According to this research, there is a difference in the shapes of the exposed class of malaria from that of the infected class because the exposed class populations are yet to spread the disease. After the treatment is given to a set of populations with malaria but could not spread the disease, those with malaria will get healed, and the population of this class reduced. Therefore, it leads to eradicating malaria, especially when the treatment rate is high irrespective of their values. This graph (5.7) shows the significance of mosquito death over the infected mosquito population. Therefore, the higher the death rate of the vector, the lesser the number of infected vectors (mosquito) that can transmit the malaria infection. The graph (5.8) shows the death rate of mosquitoes' effect on exposed mosquitoes (vector). Its increment will reduce the population of exposed mosquitoes; thereby, an increment in mosquito death rate leads to a higher fall on exposed mosquitoes. So this results in the reduction of the spread of malaria. The plot (5.9) shown the different curves generated by the increment in the death rate of mosquitoes' effect on susceptible mosquitoes. Generally, the death rate of mosquitoes would reduce the mosquito population. However, different values of its death rate revealed that the higher the death rate, the lesser the susceptible population of mosquitoes. So thereby the spread of malaria disease will be reduced.

Figures 5.10 - 5.13 shows backward bifurcation (subcritical) diagrams for populations of susceptible individuals (S) and exposed vector (E). These are the result of exogenous reinfection from the recovered class of malaria to the susceptible class of malaria. These diagrams marked a convex shape in which biologically, this convexity in macroparasite models correspond to the pair formation effects in sexually reproducing parasites as in the case of mosquitoes. Meanwhile, **Figure (5.11 - 5.14)** showing forward bifurcation (supercritical) diagrams for populations of exposed individuals (E_M), infected individuals (I_M), recovered individuals (R_M), and infected vectors (I_V) as well. Here the blue region shows that the basic reproduction number R_{0M} is less than unity which implies that the malaria disease is locally and globally asymptotically stable. So malaria is greater than one ($R_{0M}>1$), which implies that the threshold R_{0M} is locally and globally asymptotically unstable. The red region represents the endemic region for malaria, where the disease will be beyond control. So, a small positive asymptotically stable equilibrium appears, and the disease-free equilibrium loses its stability. However, this shows that those that have recovered from malaria that joined the susceptible class can be re-infected with malaria again and exposed vector class can still spread the malaria infection.

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