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Authors' contributions

This work was carried out in collaboration between both authors. Authors MARENO and IKA assisted in developing the model equations, writing of the draft, numerical simulations and review of the final draft. Both authors read and approved the final manuscript.

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Abstract

Our model is made up of two sections: In the first section, we study a simple SEIR model, estimated the reproduction number, discussed the disease-free and endemic equilibria using the Routh-Hurwitz criterion and second additive compound matrix respectively. A global stability of disease-free and the endemic equilibria was performed using Lasselle's invariance principle of Lyapunov functions. In the second section of our model, we considered SEIR-SEI model of malaria transmission between humans and mosquitoes. We estimated the reproduction number and discussed the stability of the disease-free and endemic equilibria. The disease-free equilibrium was locally asymptotically stable if the reproduction number is less than one and unstable if the reproduction number is greater than one in both models. Numerical simulations were conducted using Matlab software to confirm our analytic results. Our findings were that, Malaria may be controlled by reducing the contact rate between human and mosquitoes population and malaria transmission respectively.

Keywords: Mathematical model; disease-free equilibrium; endemic equilibrium; Lyapunov function; locally asymptotically stable.



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1 Introduction

Malaria is an infectious disease caused by plasmodium parasite and transmitted between humans through bites of female Anopheles mosquitoes [1]. Malaria is an ancient disease having a huge social, economic, and health burden. It is predominantly present in the tropical countries [2]. Even though the disease has been investigated for hundreds of years it still remains a major public health problem with 91 countries. The global record of malaria in 2015 was 212 million new cases and 429000 deaths. Across Africa, millions of people still lack access to the tools they need to prevent and treat the disease [3]. Malaria has for many years been considered as a global issue, and many epidemiologists and other scientists invest their effort in learning the dynamics of malaria and to control its transmission. From interactions with those scientists, mathematicians have developed a significant and effective tool, namely mathematical models of malaria, how to control malaria transmission, and eventually how to eradicate it [1]. A huge set of epidemiology models have been formulated mathematically, analyzed and applied to many infectious diseases.

In 1999 [4] developed and analyzed an SEIRS model to study the dynamics and transmission of malaria, involving variable human and mosquito populations. According to their results, there is a threshold parameter R_0 and the disease can persist if and only if $R_0 > 1$ and the Disease-Free Equilibrium (DFE) always exists and is locally stable if $R_0 < 1$, and unstable if $R_0 > 1$. Their model was also globally stable when $R_0 \leq 1$. They confirmed their results with numerical simulations. Their model provides a frame work for studying control strategies for the containment of malaria.

Another model which is related to this work is that of Olaniyi and Obabiyi [5], they used a system of sevendimensional ODE'S to modeling the transmission of plasmodium falciparum malaria between humans and mosquitoes with non-linear forces of infection in form of saturated incidence rates, these incidence rates produce antibodies in response to the presence of parasite causing malaria in both human and mosquito populations. They investigated the stability analysis of (DFE) and according to their results, (DFE) was asymptotically stable when $R_0 < 1$, and unstable when $R_0 > 1$. They also determined the existence of the unique Endemic Equilibrium (EE) under certain conditions, and their numerical simulation confirms the analytical result.

Nita and Gupta [6], modelled the basic of SEIR model and applied it to vector borne disease (malaria). They carried out the sensitivity analysis of the model using data from India. According to their results, the sensitivity analysis was very important, and it is the most sensitive aspect to be taken care of in their model.

Jia Li [7], developed an SEIR malaria model with stage-structured mosquitoes. They included metamorphic stages in the mosquito population and a simple stage mosquito population is introduced, were the mosquito population is divided into two classes namely, the aquatic stage in one class and all adults in the other class. According to their results the different dynamical behaviour of the models in their study, compared to other the bahaviour of most classical epidemiological models, and the possible occurrence of backward bifurcation make control of malaria more difficult.

Otieno et al. [8], studied a mathematical model of the dynamics of malaria with four-time dependent control measures in Kenya. These are insecticide treated bed nets, treatment, indoor residual spray and intermittent preventive treatment of malaria in pregnancy. They considered constant control parameters and calculated the reproduction number of disease-free and endemic equilibria, followed by the sensitivity analysis and numerical simulations of the optimal control problem using reasonable parameters. According to their model, if $R_0 \leq 1$, then the disease-free equilibrium is globally asymptotically stable. Also if $R_0 > 1$ then the endemic equilibrium exists and it's globally asymptotically stable. Their model exhibits backward bifurcation at $R_0 = 1$. They concluded that control programs that follow these strategies can effectively reduce the spread of malaria disease in different malaria transmission settings in Kenya.

Agyeing et al. [9], presented a deterministic SIS model for the transmission dynamics of malaria, a lifethreatening disease transmitted by mosquitoes. They considered four species of the parasite genus plasmodium that cause human malaria. According to their paper, some species of the parasite evolved into strains that are resistant to treatment. They developed a mathematical model and included all the available species and strains for a given city. Their model has disease-free equilibrium which is global attractor when the reproduction number of each species or strain is less than one. Their model possesses quasi-endemic equilibria and local asymptotic stability was established for the two of the species. Their numerical simulations suggest that the species or strain with the highest reproduction number exhibits competitive exclusion.

Traore et al. [10], proposed a mathematical model of nonautonomous ordinary differential equations describing the dynamics of malaria transmission with age structure for the vector population. They divided the into four classes. These are the susceptible, exposed, infectious and recovered classes. They considered the biting rate of mosquitoes as a positive periodic function which depends on climate factors. They obtained a basic reproduction number of the model and proved that it is the threshold parameter between the extinction and the persistence of the disease. They used the comparison theorem and the theory of uniform persistence to prove that if the basic reproduction number is less than one, then their disease-free equilibrium is globally stable, then there exists at least one positive periodic solution.

In this paper, we use SEIR model and apply it to malaria transmission between mosquitoes and human. We extend the model in [1] by introducing exposed class for humans and mosquitoes. Our main objective of this study is to investigate the stability analysis for disease-free equilibrium, and endemic equilibrium, and also to study the important parameters in the transmission of malaria and try to develop effective ways for controlling the disease. We assume that the recovered human individuals do not enter into the susceptible class again, and mosquitoes do not recover from malaria. We study the stability analysis of the DFE and EE equilibria. The rest of the paper is organized as follows: In section 2, we present the SEIR model description and derive the basic reproduction number. Model analysis consisting of the stability analysis of disease-free and endemic equilibria is discussed in section 3. In section 4 we apply the SEIR model to malaria transmission. We use numerical simulation to show the dynamical behaviour of our results in section 5. Section 6 is made up of discussion of our results. We ended the paper with a conclusion in section 7.

2 Mathematical Model

2.1 Model description and basic reproduction number

The Population of our model is divided into four compartments: Susceptible Humans (S), Exposed Humans (E), Infectious Humans (I) and Removed Humans (R). The interaction between the four compartments is shown in the schematic diagram in Fig. 1, below:



Fig. 1. Schematic diagram of Malaria transmission

Model Assumptions

The following assumptions were made in the model:

(i) The number of infected people increases at a rate proportional to both the number of infectious and the number of susceptible.

- (ii) Humans move from Exposed to Infectious compartments with progression rate α_1
- (iii) The rate of removal of infectious to recovered compartment is proportional to the number of infectious.
- (iv) A human can die at any stage by natural causes. Therefore μ is taken as natural death rate.
- (v) The recovered human individuals do not enter into the susceptible class again, and mosquitoes do not recover from malaria.

The model equations are given by:

$$\begin{cases}
\frac{ds}{dt} = \Lambda - \beta SI - \mu S \\
\frac{dE}{dt} = \beta SI - (\alpha_1 + \mu)E \\
\frac{dI}{dt} = \alpha_1 E - (\alpha_2 + \mu + \delta)I \\
\frac{dR}{dt} = \alpha_2 I - \mu R
\end{cases}$$
(2.1)

With $S(0) > 0, E(0) \ge 0, I(0) \ge 0$ and $R(0) \ge 0$

where, Λ is the recruitment rate of the population, β effective infection rate μ is the natural death rate, δ is the disease induced death rate, α_1 is developing rate of exposed (humans) becoming infectious and α_2 is the recovered rate of humans.

Table 1. Parameters Descriptions for the SEIR Model

Parameter name	Parameter description
Λ	Recruitment rate of susceptible.
β	Infection rate(effective infection rate)
α_1	Developing rate of exposed (humans) becoming infectious
α_2	Recover rate of humans (removal rate)
μ	Natural death rate
δ	induce death rate

The variable R of the system (2.1) does not appear in the first three equations in the analysis; we only consider the first three equations of the system (2.1) as:

$$\begin{cases} \frac{ds}{dt} = \Lambda - \beta SI - \mu S \\ \frac{dE}{dt} = \beta SI - (\alpha_1 + \mu)E \\ \frac{dI}{dt} = \alpha_1 E - (\alpha_2 + \mu + \delta)I \end{cases}$$
(2.2)

Adding above three equations of system (2.2), we have:

$$\frac{d}{dt}(S+E+I+R) = \Lambda - \mu N - \alpha_2 I - \delta I$$

$$\frac{d}{dt}(S+E+I+R) \le \Lambda - \mu N$$
(2.3)

It follows that:

ac

$$\lim_{t \to \infty} Sup \left(S + E + I + R \right) \le \frac{\Lambda}{\mu}.$$
(2.4)

Thus the feasible region of the system (2.2) is given by

$$\Gamma = \{(S, E, I): S + E + I + R \le \frac{\Lambda}{\mu}, S > 0, E \ge 0, I \ge 0, R \ge 0\}$$

is positively invariant. Next, we discuss the basic reproduction number of the system (2.2) by using the next generation matrix approach [11]. It is clear to see that the system (2.2) has the disease-free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0, 0)$.

Let $X = (S, E, I)^T$, then system (2.2) can be written as

$$X' = F(X) - V(X)$$

Where

$$F(X) = \begin{bmatrix} \beta IS \\ 0 \\ 0 \end{bmatrix} \quad \text{And} \quad V(X) = \begin{bmatrix} (\alpha_1 + \mu)E \\ -\alpha_1 E + (\alpha_2 + \mu + \delta)I \\ -\Lambda + \beta SI + \mu S \end{bmatrix}$$

The Jacobian matrices of F(X) and V(X) at the disease-free equilibrium, E_0 are respectively

$$DF(E_0) = \begin{bmatrix} 0 & \frac{\beta \Lambda}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, DV(E_0) = \begin{bmatrix} (\alpha_1 + \mu) & 0 & 0 \\ -\alpha_1 & (\alpha_2 + \mu + \delta) & 0 \\ 0 & \frac{\beta \Lambda}{\mu} & \mu \end{bmatrix}$$

Let:

$$\mathcal{F} = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix} \quad \text{And} \quad \mathcal{V} = \begin{bmatrix} V & 0 \\ \mathcal{J}_1 & \mathcal{J}_2 \end{bmatrix}$$

Where:

$$F = \begin{bmatrix} 0 & \frac{\beta \Lambda}{\mu} \\ 0 & 0 \end{bmatrix} \quad \text{And} \quad V = \begin{bmatrix} (\alpha_1 + \mu) & 0 \\ -\alpha_1 & (\alpha_2 + \mu + \delta) \end{bmatrix}$$

The reproduction number is given by the spectral radius of FV^{-1} that is

$$R_{0} = \sigma(FV^{-1}) = \begin{bmatrix} 0 & \frac{\beta\Lambda}{\mu} \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{(\alpha_{1}+\mu)} & 0 \\ \frac{\alpha_{1}}{(\alpha_{1}+\mu)(\alpha_{2}+\mu+\delta)} & \frac{1}{(\alpha_{2}+\mu+\delta)} \end{bmatrix}$$

$$R_{0} = \frac{\alpha_{1}\beta\Lambda}{\mu(\alpha_{1}+\mu)(\alpha_{2}+\mu+\delta)}$$
(2.5)

Theorem 1: The disease-free equilibrium $E_0\left(\frac{\Lambda}{\mu}, 0, 0\right)$ of the system (2.2) is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

3 Model Analysis

3.1 Disease-free equilibrium

In this section, we investigate the local geometrical properties of the disease-free equilibrium $E_0 = (\frac{\Lambda}{\mu}, 0, 0)$ by considering the linearized system of ODE's(2.2), by taking the Jacobian matrix and obtained

$$J(S, E, I) = \begin{bmatrix} -\beta I - \mu & 0 & -\beta S \\ \beta I & -(\alpha_1 + \mu) & \beta S \\ 0 & \alpha_1 & -(\alpha_2 + \mu + \delta) \end{bmatrix}$$
(3.1)

The local stability of the equilibrium may be determined from the Jacobian matrix (3.1). This implies that the Jacobian matrix for the disease-free equilibrium is given by

$$J(E_0) = \begin{bmatrix} -\mu & 0 & -\frac{\beta\Lambda}{\mu} \\ 0 & -(\alpha_1 + \mu) & \frac{\beta\Lambda}{\mu} \\ 0 & \alpha_1 & -(\alpha_2 + \mu + \delta) \end{bmatrix}$$
(3.2)

The determinant of (3.2) is given by

$$|J(E_0) - \lambda I| = \begin{vmatrix} -\mu - \lambda & 0 & -\frac{\beta \Lambda}{\mu} \\ 0 & -(\alpha_1 + \mu) - \lambda & \frac{\beta \Lambda}{\mu} \\ 0 & \alpha_1 & -(\alpha_2 + \mu + \delta) - \lambda \end{vmatrix} = 0$$
(3.3)

Lemma 1: The disease-free equilibrium Eo is locally asymptotically stable.

It follows that the characteristic equation of $J(E_0)$ computed from equation (3.3) is given by

$$\begin{split} \lambda^3 + (3\mu + \alpha_1 + \alpha_2 + \delta)\lambda^2 + (4\mu^3 + 2\alpha_1\mu + 2\alpha_2\mu + 2\mu\delta + \alpha_1\delta + \alpha_1\alpha_2 - \alpha_1\frac{\beta\Lambda}{\mu})\lambda \\ + (\mu^3 + \alpha_1\mu^2 + \alpha_2\mu^2 + \delta\mu^2 + \alpha_1\delta\mu + \alpha_1\alpha_2\mu - \alpha_1\beta\Lambda) = 0 \end{split}$$

We can write the characteristic equation above as:

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0 \tag{3.4}$$

Where:

$$a_1 = 3\mu + \alpha_1 + \alpha_2 + \delta$$

$$a_2 = 4\mu^3 + 2\alpha_1\mu + 2\alpha_2\mu + 2\mu\delta + \alpha_1\delta + \alpha_1\alpha_2 - \alpha_1\frac{\beta\Lambda}{\mu}$$

$$a_3 = \mu^3 + \alpha_1\mu^2 + \alpha_2\mu^2 + \delta\mu^2 + \alpha_1\delta\mu + \alpha_1\alpha_2\mu - \alpha_1\beta\Lambda$$

Using the Routh-Hurwitz criterion [12], it can be seen that all the eigenvalues of the characteristic equation (3.4) have negative real part if and only if:

$$a_1 > 0, a_2 > 0, a_3 > 0, a_1 a_2 - a_3 > 0$$
 (3.5)

Theorem 2: E_0 is locally asymptotically stable if and only if inequalities (3.5) are satisfied.

3.1.1 Global stability of disease-free equilibrium

To investigate the global stability of E_0 we consider the Lyapunov function [10]:

$$V = \alpha_1 E + (\alpha_1 + \mu)I$$
 Then

$$\frac{dv}{dt} = \alpha_1 \frac{dE}{dt} + (\alpha_1 + \mu) \frac{dI}{dt} = \alpha_1 [\beta SI - (\alpha_1 + \mu)E] + (\alpha_1 + \mu)[\alpha_1 E - (\alpha_2 + \mu + \delta)I]$$

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$$\leq \left(\frac{\alpha_1\beta\Lambda}{\mu} - (\alpha_1 + \mu)(\alpha_2 + \mu + \delta)\right)I = (\alpha_1 + \mu)(\alpha_2 + \mu + \delta)[R_0 - 1]I \leq 0, if R_0 < 1.$$

The maximal compact invariant set in $\{(S, E, I) \in \Gamma: \frac{dI}{dt} = 0\}$ Using Lasalle's invariance principle [13]. We have the following theorem.

Theorem 3: if $R_0 < 1$, then the disease- free equilibrium E_0 is globally asymptotically stable and the disease dies out , but if $R_0 > 1$, then E_0 is unstable.

3.2 Existence of endemic equilibrium

In this section, we consider a situation in which there is persistence of malaria in the population. We denote $E^* = (S^*, E^*, I^*)$ as the endemic equilibrium of the system (2.2). We also obtain

$$S^* = \frac{(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{\alpha_1 \beta} \quad , E^* = \frac{\alpha_1 \beta \Lambda - \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{\alpha_1 \beta(\alpha_1 + \mu)} \text{ and } I^* = \frac{\alpha_1 \beta \Lambda - \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{\beta(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}$$

From system of ODE's (2.2) and the linearized system we obtained:

$$J(E^*) = \begin{bmatrix} -\beta I^* - \mu & 0 & -\beta S^* \\ \beta I^* & -(\alpha_1 + \mu) & \beta S^* \\ 0 & \alpha_1 & -(\alpha_2 + \mu + \delta) \end{bmatrix}$$
$$J(E^*) = \begin{bmatrix} -[\frac{\alpha_1 \beta \Lambda - \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}] - \mu & 0 & -[\frac{(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{\alpha_1}] \\ \frac{\alpha_1 \beta \Lambda - \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)} & -(\alpha_1 + \mu) & \frac{(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)}{\alpha_1} \\ 0 & \alpha_1 & -(\alpha_2 + \mu + \delta) \end{bmatrix}$$

We determine the local stability of the positive equilibrium E^* , by using the following lemma.

Lemma 2 [14,15]: Let *H* be a 3×3 real matrix. If tr(H), det(H) and $det(H^{[2]})$ are all negative, then all the eigenvalues of *H* have negative real part.

Definition 1 [14,15,16] Let *B* be a real $m \times m$ matrix. The second additive compound matrix of $B = (b_{ij})$ for m = 3 is defined as

$$B^{[2]} = \begin{bmatrix} b_{11} + b_{22} & b_{23} & -b_{13} \\ b_{32} & b_{11} + b_{33} & b_{12} \\ -b_{31} & b_{21} & b_{22} + b_{33} \end{bmatrix}$$
(3.6)

Theorem 3: The positive equilibrium E^* of the system (2.2) is locally asymptotically stable if $R_0 > 1$.

Proof: We construct a second additive compound matrix $J^{[2]}(E^*)$ of $J(E^*)$ and obtain

$$J^{[2]}(E^*) = \begin{bmatrix} -(\beta I^* + 2\mu + \alpha_1) & \beta s^* & \beta s^* \\ \alpha_1 & -(\beta I^* + 2\mu + \alpha_2 + \delta) & \beta S \\ 0 & \beta I^* & -(2\mu + \alpha_1 + \alpha_2 + \delta) \end{bmatrix}$$
(3.7)

Then from the above:

 $\begin{aligned} tr(J(E^*)) &= -(\beta I^* + 3\mu + \alpha_1 + \alpha_2 + \delta) < 0 \\ &= -[(3\mu + \alpha_1 + \alpha_2 + \delta) + \alpha_1\beta\Lambda - \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta) < 0 \\ \text{If } (3\mu + \alpha_1 + \alpha_2 + \delta) + \alpha_1\beta\Lambda > \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta) \\ \det(J(E^*)) &= -[\mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta) - \alpha_1\beta\Lambda] < 0 \\ \text{If } \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta) > \alpha_1\beta\Lambda \end{aligned}$

Next, we calculate the determinant of $J^{[2]}(E^{**})$ in (3.7) and obtained:

$$det[J^{[2]}(E^*)] = -[a^2g + aged + b^2g + cga + cged + b - 2agb - bged - cgb - dg - a] < 0$$

If $a^2g + aged + b^2g + cga + cged + b > 2agb + bged + cgb + dg + a$

Where $a = \alpha_1 \beta \Lambda$, $b = \mu(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)$, $c = (2\mu + \alpha_1)(\alpha_1 + \mu)(\alpha_2 + \mu + \delta)$

 $d = (\alpha_1 + \mu)(\alpha_2 + \mu + \delta)$, $e = (2\mu + \alpha_2 + \delta)$, $g = \alpha_1 + 2\mu + \alpha_2 + \delta$ Note that if $R_0 > 1$

Thus $det[J^{[2]}(E^*)] < 0$. This completes the proof.

3.2.1 Global stability of endemic equilibrium

To discuss the global stability of E^* we consider the following lyapunov function [13,17].

$$V = \left(S - S^* - S^* ln \frac{S}{S^*}\right) + \left(E - E^* - E^* ln \frac{E}{E^*}\right) + \frac{\mu + \alpha_1}{\alpha_1} \left(I - I^* - I^* ln \frac{I}{I^*}\right)$$
$$\frac{dV}{dt} = \frac{d}{dt} \left(S - S^* - S^* ln \frac{S}{S^*}\right) + \frac{d}{dt} \left(E - E^* - E^* ln \frac{E}{E^*}\right) + \frac{\mu + \alpha_1}{\alpha_1} \frac{d}{dt} \left(I - I^* - I^* ln \frac{I}{I^*}\right)$$
$$\frac{dV}{dt} = \left(1 - \frac{S^*}{S}\right) \frac{dS}{dt} + \left(1 - \frac{E^*}{E}\right) \frac{dE}{dt} + \frac{\mu + \alpha_1}{\alpha_1} \left(1 - \frac{E^*}{E}\right) \frac{dI}{dt}$$

By the expression $\Lambda = \beta S^* I^* + \mu S^*$ of system (2.2) and substituting the derivatives of ODE's (2.2).

$$\begin{split} \frac{dv}{dt} &= \left(1 - \frac{S^*}{S}\right) [\Lambda - \beta SI - \mu S] + \left(1 - \frac{E^*}{E}\right) [\beta SI - (\alpha_1 + \mu)E] + \\ &= \frac{\mu + \alpha_1}{\alpha_1} \left(1 - \frac{I^*}{I}\right) [\alpha_1 E - (\alpha_2 + \mu + \delta)I] \\ \frac{dv}{dt} &= \left(1 - \frac{S^*}{S}\right) [\beta S^* I^* + \mu S^* - \beta SI - \mu S] + \left(1 - \frac{E^*}{E}\right) [\beta SI - (\alpha_1 + \mu)E] \\ &+ \frac{\mu + \alpha_1}{\alpha_1} \left(1 - \frac{I^*}{I}\right) [\alpha_1 E - (\alpha_2 + \mu + \delta)I] \\ \frac{dv}{dt} &= \left(1 - \frac{S^*}{S}\right) [-\mu (S - S^*) + \beta S^* I^* - \beta SI] + \left(1 - \frac{E^*}{E}\right) [\beta SI - (\alpha_1 + \mu)E] \\ &+ \frac{\mu + \alpha_1}{\alpha_1} \left(1 - \frac{I^*}{I}\right) [\alpha_1 E - (\alpha_2 + \mu + \delta)I] \end{split}$$

Since $\alpha_1 E^* = (\alpha_2 + \mu + \delta)I^*$ this implies that:

$$\begin{split} \beta S^* I &- \frac{\mu + \alpha_1}{\alpha_1} (\alpha_2 + \mu + \delta) I = \beta S^* I - (\alpha_1 + \mu) \frac{I^*}{I} E^* = [\beta S^* I - (\alpha_1 + \mu) E^*] \frac{I^*}{I} = 0\\ \frac{dV}{dt} &= -\mu \left[\frac{(s - s^*)^2}{s} \right] + 3(\alpha_1 + \mu) E^* - \beta S^* I^* \frac{S^*}{s} - \beta S I \frac{E^*}{E} - (\alpha_1 + \mu) \\ &- \mu \left[\frac{(s - s^*)^2}{s} \right] + (\alpha_1 + \mu) E^* [3 - \frac{S^*}{s} - \frac{S}{s^*} \frac{E^*}{E} \frac{I^*}{I} - \frac{E^*}{E} \frac{I^*}{I}] \le 0 \end{split}$$

Since the arithmetic mean is greater than or equal to the geometric mean of the quantities

$$\frac{S^*}{S}, \frac{S}{S^*} \frac{E^*}{E} \frac{I^*}{I}, \frac{E^*}{E} \frac{I^*}{I} \text{ that is: } \frac{S^*}{S} + \frac{S}{S^*} \frac{E^*}{E} \frac{I^*}{I} + \frac{E^*}{E} \frac{I^*}{I} - 3 \ge 0 \text{ then } \frac{dV}{dt} = 0 \text{ , holds when } S = S^*, E = E^*$$

And $I = I^*$, so that the maximal compact invariant set in $\{(S, E, I) \in \Gamma: \frac{dV}{dt} = 0\}$ is singleton $\{E^*\}$ using Lasalle's invariance principle, we have the following theorem

Theorem 4: If $R_0 > 1$ the endemic equilibrium E*of the system (2.2) is globally asymptotically stable.

4 Application of the Model to Malaria Transmission

4.1 Model description and basic reproduction number

The model is formulated for both human population as well as mosquito population at time t. We divide the human population into four classes: Susceptible S_H , Exposed E_H , Infectious I_H , and Recovery Human R_H , and that of the population the mosquitoes is divided into three classes they are susceptible S_V , Exposed E_V , Infectious I_v respectively.

The interaction between the human and mosquitoes is shown in the schematic diagram in Fig. 2, below:



Fig. 2. Schematic diagram of Malaria transmission between Humans and Mosquitoes.

The model equations are given by:

$$\begin{cases} \frac{dS_H}{dt} = \Lambda_H - \beta_H S_H I_H - \mu_H S_H \\ \frac{dE_H}{dt} = \beta_H S_H I_H - (\alpha_{1H} + \mu_H) E_H \\ \frac{dI_H}{dt} = \alpha_{1H} E_H - (\alpha_{2H} + \mu_H + \delta) I_H \\ \frac{dR_H}{dt} = \alpha_{2H} I_H - \mu_H R_H \\ \frac{dS_V}{dt} = \Lambda_V - \beta_V S_V I_V - \mu_V S_V \\ \frac{dE_V}{dt} = \beta_V S_V I_V - (\alpha_{1V} + \mu_V) E_V \\ \frac{dI_V}{dt} = \alpha_{1V} E_V - \mu_V I_V \end{cases}$$
(4.1)

With the initial condition: $S_H(0) > 0$, $E_H(0) \ge 0$, $I_H(0) \ge 0$, $R_H(0) \ge 0$, $S_V(0) > 0$, $E_V(0) \ge 0$, $I_V(0) \ge 0$.

 Table 2. Parameters description of the malaria transmission model.

Parameter	Parameters description
$\Lambda_{\rm H}$	Recruitment rate for humans
Λ_{v}	Recruitment rate for mosquitoes
$\alpha_{1\mathrm{H}}$	Developing rate of exposed (humans) becoming infectious
α_{2H}	Recover rate of humans (removal rate)
μ_H	Natural death rate for humans
δ	Induce death rate for humans
α_{1V}	Developing rate of exposed (mosquitoes) becoming infectious
$\mu_{\rm V}$	Natural death rate for mosquito
q_H	Probability of transmission of infection from an infectious mosquitoes to a susceptible humans
q_V	Probability of transmission of infection from an infectious humans to a susceptible mosquitoes
η_V	Mosquitoes biting rate
β_{H}	Infection rate $q_H \times \eta_V$ of humans
β_V	Infection rate $q_v \times \eta_v$ mosquitoes

We also consider the following equations:

$$N_H(t) = S_H(t) + E_H(t) + I_H(t) + R_H(t)$$
(4.2)

Then the derivative of $N_H(t)$ with respect to t is given by:

$$\begin{split} \frac{dN_H}{dt} &\leq \Lambda_{\rm H} - \mu_H N_H - \delta I_H \\ \lim_{t \to \infty} N_H(t) &\leq \frac{\Lambda_{\rm H}}{\mu_H}. \end{split}$$

Thus the feasible region of the system (4.1) for the human is given by

$$\Gamma^* = \{ (S_H, E_H, I_H, R_H) : S_H + E_H + I_H + R_H \le \frac{\Lambda_H}{\mu_H}, S_H > 0, E_H \ge 0, I_H \ge 0, R_H \ge 0 \}$$

Is positively invariant and let: $N_V(t) = S_V(t) + E_V(t) + I_V(t)$. (4.3)

The derivative of $N_V(t)$ with respect to t is given by:

$$\frac{dN_V}{dt} \le \Lambda_V - \mu_V N_V$$
$$\lim_{t \to \infty} N_V(t) \le \frac{\Lambda_V}{\mu_V}$$

Thus the feasible region of the system (4.1) of the mosquito is given by

$$\Gamma^{**} = \{(S_V, E_V, I_V): S_V + E_V + I_V \leq \frac{\Lambda_V}{\mu_V}, S_V > 0, E_V \geq 0, I_V \geq 0\}$$
 is positively invariant

It is easy to see that the system (4.1) has the disease-free equilibrium

 $E_{0HV} = (\frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, \frac{\Lambda_V}{\mu_V}, 0, 0)$. The basic reproduction number R_0 for the human and mosquito will be derived by using the next generation matrix in [6,11] as $R_1 = \sqrt{R_{0H}R_{0V}}$, and R_1 for the model (4.1) is calculated and it is given by:

$$R_1 = \sqrt{\frac{\Lambda_V \Lambda_H \alpha_{1V} \alpha_{1H} \beta_H \beta_V}{\mu_H \mu_V^2 (\mu_V + \alpha_{1V}) (\mu_H + \alpha_{1H} + \delta)}}$$
(4.4)

Theorem 5: The disease-free equilibrium $E_{0HV}\left(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, \frac{\Lambda_V}{\mu_V}, 0, 0\right)$ of the system of the ODE's (4.1) is asymptotically stable if $R_1 < 1$ and unstable if $R_1 > 1$.

4.2 Model Analysis

4.2.1 Disease-free equilibrium

In this section, we investigate the local geometrical properties of the disease-free equilibrium $E_{0HV}\left(\frac{\Lambda_{\rm H}}{\mu_{\rm H}}, 0, 0, 0, \frac{\Lambda_{\rm V}}{\mu_{\rm V}}, 0, 0\right)$ by considering the linearized system of ODE's (4.1),by taking the Jacobian matrix and obtained.

$$J_{HV}(S_H, E_H, I_H, R_H, S_V, E_V, I_V) = \begin{bmatrix} J_0 & J_2 \\ J_1 & J_3 \end{bmatrix}$$
(4.5)

Where:

The local stability of the disease-free equilibrium determined from the Jacobian matrix (4.5). This implies that the Jacobian matrix of the disease-free equilibrium is given by:

The determinant of (4.6) is given by:

$$|J(E_{0HV}) - \lambda I| = \begin{vmatrix} J_0 & J_2 \\ J_1 & J_3 \end{vmatrix} = 0$$
(4.7)

Where:

The eigenvalues of the (4.7) are given by:

Clearly $\lambda_1 = -\mu_H$ (repeated), $\lambda_2 = -\mu_V$ are negatives and

$$\lambda^4 + c_1\lambda^3 + c_2\lambda^2 + c_3\lambda + c_4 = 0 \tag{4.8}$$

By Using the Routh-Hurwitz criterion [12], it can be seen that all the eigenvalues of the characteristic equation (4.8) have negative real part if and only if:

$$\begin{cases} c_1 > 0, \ c_2 > 0, c_3 > 0, c_4 > 0, c_1 c_2 - c_3 > 0, c_1 c_2 c_3 - c_3^2 - c_1^2 c_4 > 0, \\ c_1 c_2 c_3 c_4 - c_0 c_3^2 c_4 > 0 \end{cases}$$
(4.9)

Where: $c_1 = \alpha_1 + 2\mu_H + \alpha_2 + \delta + 2\mu_V + \alpha_3$.

$$c_{2} = 2\alpha_{1}\mu_{V} + 4\mu_{V}\mu_{H} + 2\alpha_{2}\mu_{V} + 2\delta\mu_{V} + \alpha_{1}\alpha_{3} + \alpha_{2}\alpha_{3} + \delta\alpha_{3} + +2\alpha_{3}\mu_{H} + \alpha_{3}\mu_{V}^{3} - \frac{\alpha_{3}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\beta_{H}\Lambda_{H}}{\mu_{H}}$$

$$c_{3} = \alpha_{1}\alpha_{2} + \alpha_{1}\mu_{H} + \alpha_{1}\delta + \alpha_{2}\mu_{H} + \mu_{H}^{2} + \delta\mu_{H} + 2\alpha_{1}\alpha_{2}\mu_{V} + 2\alpha_{1}\mu_{V}\mu_{H} + 2\alpha_{1}\delta\mu_{V} + 2\alpha_{1}\mu_{V}\mu_{H} + 2\alpha_{1}\alpha_{2}\alpha_{3} + \alpha_{1}\alpha_{3}\mu_{H} + \alpha_{1}\alpha_{3}\delta + \alpha_{2}\alpha_{3}\mu_{H} + \alpha_{3}\mu_{H}^{2} + \alpha_{1}\alpha_{3}\delta\mu_{V}^{3} + 2\alpha_{3}\delta\mu_{V}^{3}\mu_{H} + \alpha_{3}^{3}\mu_{V}^{3} + \alpha_{3}\delta\mu_{V}^{3} - \frac{\alpha_{1}\alpha_{3}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{2\alpha_{3}\mu_{H}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{3}\delta\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{2\mu_{V}\beta_{H}\Lambda_{H}}{\mu_{H}} - \frac{\alpha_{3}\beta_{H}\Lambda_{H}}{\mu_{H}}$$

$$c_{4} = \alpha_{1}\alpha_{2}\alpha_{3}\mu_{V}^{3} + \alpha_{1}\alpha_{3}\mu_{V}^{3}\mu_{H} + \alpha_{1}\alpha_{3}\delta\mu_{V}^{3} + \alpha_{2}\alpha_{3}\mu_{V}^{3}\mu_{H} + \alpha_{3}\mu_{V}^{3}\mu_{H}^{2} + \delta\alpha_{3}\mu_{V}^{3}\mu_{H} + \frac{\alpha_{3}\beta_{V}\Lambda_{V}\beta_{H}\Lambda_{H}}{\mu_{V}\mu_{H}} - \frac{\alpha_{3}\beta_{H}\Lambda_{H}\mu_{V}}{\mu_{V}} - \frac{\beta_{H}\Lambda_{H}\mu_{V}^{2}}{\mu_{V}} - \frac{\alpha_{1}\alpha_{2}\alpha_{3}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{1}\alpha_{3}\delta\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{2}\alpha_{3}\mu_{H}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{3}\beta_{H}\Lambda_{H}\mu_{V}}{\mu_{V}} - \frac{\alpha_{3}\beta_{H}\Lambda_{H}\mu_{V}}{\mu_{V}} - \frac{\alpha_{3}\beta_{H}\Lambda_{H}\mu_{V}}{\mu_{V}} - \frac{\alpha_{3}\alpha_{H}\mu_{S}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{3}\alpha_{H}\mu_{S}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{3}\alpha_{H}\mu_{S}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{1}\alpha_{3}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{1}\alpha_{3}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{1}\alpha_{3}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{1}\alpha_{3}\beta_{H}\mu_{S}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{1}\alpha_{3}\beta_{H}\mu_{S}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{1}\alpha_{3}\beta_{H}\mu_{S}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{1}\alpha_{3}\beta_{V}\Lambda_{V}}{\mu_{V}} - \frac{\alpha_{1}\alpha_{3}\beta_{V}\Lambda_{V}}{\mu_$$

It can be seen that all the eigenvalues have negative real parts and therefore the disease-free equilibrium is Locally Asymptotically Stable.

4.2.2 Endemic equilibrium

In this section, we consider a situation in which all the steady states coexist in the equilibrium. We denote $E^*_{HV} = (S_H^*, E_H^*, I_H^*, R_H^*, S_V^*, E_V^*, I_V^*)$ as the endemic equilibrium of the system (4.1). We also obtain

$$S_{H}^{*} = \frac{(\alpha_{1H} + \mu_{H})(\alpha_{2H} + \mu_{H} + \delta)}{\alpha_{1H}\beta_{H}}, \qquad E_{H}^{*} = \frac{\alpha_{1H}\beta_{H}\Lambda_{H} - \mu_{H}(\alpha_{1H} + \mu_{H})(\alpha_{2H} + \mu_{H} + \delta)}{\alpha_{1H}\mu_{H}\beta_{H}(\alpha_{1H} + \mu_{H})}$$
$$I_{H}^{*} = \frac{\alpha_{1H}\beta_{H}\Lambda_{H} - \mu_{H}(\alpha_{1H} + \mu_{H})(\alpha_{2H} + \mu_{H} + \delta)}{\beta_{H}(\alpha_{1H} + \mu_{H})(\alpha_{2H} + \mu_{H} + \delta)}, \qquad R_{H}^{*} = \frac{\alpha_{2H}[\alpha_{1H}\beta_{H}\Lambda_{H} - \mu_{H}(\alpha_{1H} + \mu_{H})(\alpha_{2H} + \mu_{H} + \delta_{H})]}{\mu_{H}\beta_{H}(\alpha_{1H} + \mu_{H})(\alpha_{2H} + \mu_{H} + \delta)}$$
$$S_{V}^{*} = \frac{\mu_{V}(\alpha_{1V} + \mu_{V})}{\alpha_{1V}\beta_{V}}, \qquad E_{V}^{*} = \frac{\alpha_{1V}\beta_{V}\Lambda_{V} - \mu_{V}^{2}(\alpha_{1V} + \mu_{V})}{\alpha_{1V}\beta_{V}(\alpha_{1V} + \mu_{V})}, \qquad I_{V}^{*} = \frac{\alpha_{1V}\beta_{V}\Lambda_{V} - \mu_{V}^{2}(\alpha_{1V} + \mu_{V})}{\mu_{V}\beta_{V}(\alpha_{1V} + \mu_{V})}$$

The local stability of the endemic equilibrium determined from the Jacobian matrix (4.5). This implies that the Jacobian matrix of the endemic equilibrium is given by:

$$J_{HV}(S_{H}^{*}, E_{H}^{*}, I_{H}^{*}, R_{H}^{*}, S_{V}^{*}, E_{V}^{*}, I_{V}^{*}) = J(E_{HV}^{*}) = \begin{bmatrix} J_{0} & J_{2} \\ J_{1} & J_{3} \end{bmatrix}$$
(4.10)

Where:

$$J_{0} = \begin{bmatrix} -\beta_{H}I_{H}^{*} - \mu_{H} & 0 & -\beta_{H}S_{H}^{*} \\ 0 & -(\alpha_{1H} + \mu_{H}) & \beta_{H}S_{H}^{*} \\ 0 & \alpha_{1H} & -(\alpha_{2H} + \mu_{H} + \delta) \end{bmatrix}, \quad J_{1} = \begin{bmatrix} 0 & 0 & \alpha_{2H} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$
$$J_{2} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad J_{3} = \begin{bmatrix} -\mu_{H} & 0 & 0 & 0 \\ 0 & -\beta_{V}I_{V}^{*} - \mu_{V} & 0 & -\beta_{V}S_{V}^{*} \\ 0 & 0 & -(\alpha_{1V} + \mu_{V}) & \beta_{V}S_{V}^{*} \\ 0 & 0 & \alpha_{1V} & -\mu_{V} \end{bmatrix}$$

The eigenvalues of the (4.10) are given by:

Clearly
$$\lambda_1 = -\left[\frac{(\alpha_{1H} + \mu_H)(\alpha_{2H} + \mu_H + \delta)}{\alpha_{1H}} + \mu_H\right], \lambda_2 = -\mu_H, \lambda_3 = -\frac{\alpha_{1V\beta_V}\Lambda_V}{\mu_V(\alpha_{1V} + \mu_V)}$$
 are negatives and
 $\lambda^4 + d_1\lambda^3 + d_2\lambda^2 + d_3\lambda + d_4 = 0$
(4.11)

By Using the Routh-Hurwitz criterion [12], it can be seen that all the eigenvalues of the characteristic equation (4.11) have negative real part if and only if:

$$\begin{cases} d_1 > 0, \, d_2 > 0, d_3 > 0, d_4 > 0, d_1 d_2 - d_3 > 0, d_1 d_2 d_3 - d_3^2 - d_1^2 d_4 > 0, \\ d_1 d_2 d_3 d_4 - d_0 d_3^2 d_4 > 0 \end{cases}$$
(4.12)

Where: $d_1 = \alpha_{1\nu} + \mu_V + \delta + \alpha_{2H} + 3\mu_H + \alpha_{1H}$ $d_2 = \alpha_{1H}\alpha_{2H} + 4\alpha_{1H}\mu_H + 2\delta\alpha_{1H} + \alpha_{1H}\mu_V + 2\alpha_{2H}\mu_H + 4\mu_H^2 + \delta\mu_H + 2\mu_H\mu_V + 2\mu_H\alpha_{1V} + \alpha_{2H}\mu_{1V} + \alpha_{1H}\alpha_{2H}\mu_{1V} + \alpha_{1H}\mu_{2}\mu_{1V} + \alpha_{1H}\mu_{2}^2 + \delta\alpha_{1H}\mu_V$ $d_3 = \alpha_{1H}\alpha_{2H}\mu_V + \alpha_{1H}\alpha_{2H}\alpha_{1V} + \alpha_{1H}\mu_V\mu_H + \alpha_{1H}\mu_V\mu_V + \alpha_{1H}\mu_S + \alpha_{1V} + \alpha_{1H}\mu_H^2 + \delta\alpha_{1H}\mu_V$ $+\delta\alpha_{1H}\alpha_{1V} + \delta\alpha_{1H}\mu_V + \alpha_{1H}\mu_V\alpha_{1V} + \alpha_{1H}\mu_V\mu_H + \alpha_{1H}\beta_V\alpha_{1V} + \alpha_{1H}\mu_V\mu_V + \alpha_{2H}\mu_V\mu_H + \mu_{R}\alpha_{2H}\alpha_{1V}$ $+\mu_H^2\alpha_{2H}^2\alpha_{2H} + \mu_H^2\mu_V + \mu_H^2\alpha_{1V} + \alpha_{1H}\mu_V\mu_V + \delta\mu_H^2 + 2\mu_H^2\mu_V + 2\alpha_{1V}\mu_V\mu_H + \alpha_{2H}\alpha_{1V} + \alpha_{2H}\beta_V\alpha_{1V} + \alpha_{2H}\beta_V\alpha_{1V} + \alpha_{2H}\beta_V\alpha_{1V} + \alpha_{2H}\beta_V\alpha_{1V} + \alpha_{2H}\beta_V\alpha_{1V} + \alpha_{2H}\beta_V\alpha_{1V} + \delta\alpha_{1H}\mu_H + \alpha_{2H}\alpha_{2H}\mu_V + \alpha_{1H}\alpha_{2H}\mu_V + \alpha_{1H}\alpha_{2H}\mu_V\mu_H + \alpha_{1H}\alpha_{2H}\mu_V\mu_H + \alpha_{1H}\alpha_{2H}\mu_V\mu_H + \alpha_{1H}\alpha_{2H}\mu_V\mu_H + \alpha_{1H}\alpha_{2H}\mu_V\mu_H + \delta\alpha_{1H}\mu_H^2 + \delta\alpha_{1H}\mu_H^2 + \delta\alpha_{1H}\mu_H\beta_V\alpha_{1V} + \alpha_{1H}\mu_H\beta_V\alpha_{1V} + \alpha_{1H}\mu_H\beta_V\alpha_{1V} + \alpha_{1H}\mu_H\beta_V\mu_V + \alpha_{2H}\mu_H\beta_V\alpha_{1V} + \alpha_{1H}\mu_H\beta_V\mu_V + \alpha_{2H}\mu_H\beta_V\alpha_{1V} + \alpha_{1H}\mu_H\beta_V\mu_V + \alpha_{2H}\mu_H\beta_V\alpha_{1V} + \alpha_{1H}\mu_H\beta_V\mu_V + \alpha_{2H}\mu_H\beta_V\mu_V + \alpha_{1H}\mu_H\alpha_{1V}\mu_V + \alpha_{2H}\mu_H^2\mu_V + \alpha_{1H}\mu_H\alpha_{1V}\beta_V + \alpha_{2H}\mu_H\beta_V\alpha_{1V} + \delta\mu_H^2\alpha_{1V} + \delta\alpha_{1H}\mu_V\mu_V + \alpha_{2H}\mu_H\beta_V\mu_V + \alpha_{1H}\mu_H\alpha_{1V}\mu_V + \alpha_{2H}\mu_H\beta_V\mu_V + \alpha_{1H}\mu_H\alpha_{1V}\mu_V + \alpha_{2H}\mu_H\alpha_{1V}\mu_V + \alpha_{1H}\mu_H\alpha_{1V}\mu_V +$

5 Numerical Simulations

In this section, we present the numerical simulation of our models. All the parameters used in this section are displayed in Table 3, Table 4 and under Figures.

Parameter	Parameter Description		Source	
Λ	Recruitment rate of susceptible.	1.2	[18]	
β	Infection rate(effective infection rate)	0.001	Assumption	
α_1	Developing rate of exposed (humans)	0.1	Assumption	
α_2	Recover rate of humans(removal rate)	0.0035	[6]	
μ	Natural death rate	0.03	[18]	
δ	induce death rate	0.089	[18]	

Table 3. Parameters values of mod	el	1	l
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Table 4. Parameters values of n	model	2
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Parameter	Description	Value	Source
$\Lambda_{ m H}$	Recruitment rate for human	1.2	Assumption
$\Lambda_{\mathbf{v}}$	Recruitment rate for mosquitoes	0.7	Assumption
$\alpha_{1\mathrm{H}}$	Developing rate of exposed (humans) becoming infectious	0.1	Assumption
α_{2H}	Recover rate of human.(removal rate)	0.0035	[6]
μ_H	Natural death rate for humans	0.01146	Assumption
δ	induce death rate for humans	0.068	Assumption
α_{1V}	Developing rate of exposed (mosquitoes) becoming infectious	0.083	[6]
μ_{v}	Natural death rate for mosquitoes	0.05	[19]
q_H	Probability of transmission of infection from an infectious mosquitoes to a susceptible humans	0.022	[20]
q_V	Probability of transmission of infection from an infectious humans to a susceptible mosquitoes	0.24	[20]
η _v	Mosquito biting rate	0.29	[15]
$\dot{\beta}_{H}$	Infection rate $q_H \times \eta_V$ of humans	0.00638	[20]
β_V	Infection rate $q_v \times \eta_v$ mosquitoes	0.0696	[20]

5.1 Sensitivity analysis of the basic reproduction numbers

We investigate the nature of the model by conducting sensitivity analysis of the reproductive numbers R_0 , R_1 for model (2.1) and (4.1) respectively.

- (a) At the disease-free equilibrium for (2.1): $\Lambda = 1.2, \beta = 0.001, \alpha_1 = 0.1, \alpha_2 = 0.0035, \mu = 0.03, \delta = 0.089$, $R_0 < 1$.
 - (i) If the value of β is increased to 0.08 or more and the values of α_1, α_2, μ , Λ and δ maintains same then $R_0 > 1$.
 - (ii) If the value of Λ is increased to 10 or more and the values of α_1, α_2, μ , β and δ maintains same then $R_0 > 1$
- (b) At the endemic equilibrium for (4.1): $\beta_V = 0.0696$, $\beta_H = 0.0064$, $\alpha_{1V} = 0.083$, $\alpha_{1H} = 0.1$, $\mu_H = 0.01146$, $\mu_V = 0.05$, $\delta = 0.068$, $R_1 > 1$.
 - (i) If the values of $\beta_{\rm H}$ and $\beta_{\rm v}$ is decreased to 0.000064 and 0.000696 respectively or more and the values of the others parameters maintains same then $R_1 < 1$.
 - (ii) If the values of α_{1H} and α_{1v} is decreased to 0.001 and 0.00083 respectively or more and the values of the others parameters maintains same then $R_1 < 1$.



Fig. 3. Time responses of the state variables *S*, *E*, *I*, and *R* with initial conditions s(0) = 0.83, E(0) = 0.08, I(0) = 0.07, R(0) = 0.02, against the time and $R_0 = 0.2512$. Where the Parameters: $\Lambda = 1.2$, $\beta = 0.001$, $\alpha_1 = 0.1$, $\alpha_2 = 0.0035$, $\mu = 0.03$, $\delta = 0.089$. Only the susceptible class exists. The population of Exposed, Infective, and Recovery classes approach zero and reaches disease-free equilibrium.



Fig. 4. Time responses of the state variables *S*, *E*, *I*, and *R* with initial conditions s(0) = 0.83, E(0) = 0.08, I(0) = 0.07, R(0) = 0.02, against the time and $R_0 > 1$. Where the parameters: $\Lambda = 1.2$, $\beta = 0.08$, $\alpha_1 = 0.1$, $\alpha_2 = 0.0035$, $\mu = 0.03$, $\delta = 0.089$. All the distinct classes coexist in the population and therefore approach endemic equilibrium.



Fig. 5. Time responses of the state variables *S*, *E*, *I*, and *R* with initial condition s(0) = 0.83 E(0) = 0.08, I(0) = 0.07, R(0) = 0.02, against the time and $R_0 > 1$. Where the parameters: $\Lambda = 10$, $\beta = 0.001$, $\alpha_1 = 0.1$, $\alpha_2 = 0.0035$, $\mu = 0.03$, $\delta = 0.089$. When the value of Λ was increased from 1.2 to 10, all the classes in the population reappeared, making the equilibrium endemic.



Fig. 6. Time responses of the state variables S_H , E_H , I_H , and R_H with initial conditions s(0) = 0.8 E(0) = 0.1, I(0) = 0.08, R(0) = 0.02, against the time and $R_1 < 1$. Where the parameters: $\Lambda_H = 1.2$, $\beta_H = 0.001$, $\alpha_{1H} = 0.1$, $\alpha_{2H} = 0.0035$, $\mu_H = 0.03$, $\delta = 0.089$. Only the susceptible humans class exists. The populations of Exposed, Infective, and Recovery human classes approach zero and reaches disease-free equilibrium



Fig. 7. Time responses of the state variables S_H , E_H , I_H , and R_H with initial conditions s(0) = 0.8E(0) = 0.1, I(0) = 0.08, R(0) = 0.02, against the time and $R_1 > 1$. Where the parameters: $\Lambda_H = 1.2$, $\beta_H = 0.08$, $\alpha_{1H} = 0.1$, $\alpha_{2H} = 0.0035$, $\mu_H = 0.03$, $\delta = 0.089$. All the distinct classes of humans coexist in the population and therefore approach endemic equilibrium



Fig. 8. Time responses of the state variables S_V , E_V , and I_V , with initial conditions $S_V(0) = 0.7$ $E_V(0) = 0.2$, $E_V(0) = 0.1$, against the time and $R_1 < 1$. Where the parameters: $\Lambda_V = 0.7$, $\beta_V = 0.0696$, $\alpha_{1V} = 0.083$, $\mu_V = 0.165$. Only the susceptible mosquitoes class exists. The populations of Exposed and Infective, Mosquito classes approach zero and reaches disease-free equilibrium



Fig. 9. Time responses of the state variables S_V, E_V , and I_V , with initial conditions $S_V(0) = 0.7$ $E_V(0) = 0.2$, $E_V(0) = 0.1$, against the time and $R_1 > 1$. Where the parameters: $\Lambda_V = 0.7, \beta_V = 0.696, \alpha_{1V} = 0.083, \mu_V = 0.165$. All the distinct classes of mosquitoes coexist in the populations.







Fig. 12. Relationship between R_0 and Λ when $R_0 < 1$.







Fig. 14. Relationship between R_1 and α_{1V} when $R_1 < 1$.



Fig. 15. Relationship between R_1 and β_H when $R_1 < 1$.



Fig. 16. Relationship between R_1 and β_V when $R_1 < 1$.



Fig. 17. Relationship between R_1 and δ when $R_1 < 1$.

6 Discussion of Results

In this paper, we studied the dynamics of an SEIR Model (2.1) and applied it to malaria transmission between human and mosquito, we derived the basic reproduction number and discussed the existence and stability of Disease-Free Equilibrium (DFE) and Endemic Equilibrium (EE) of model (2.1) and applied them to model transmission between human and mosquitoes (4.1). Our analysis shows that if the reproduction number is less than one then the (DFE) is locally asymptotically stable, this implies that only susceptible is present and the other populations reduces to zero, and the disease dies out. And if the reproduction number is greater than one then (DFE) is unstable, for (2.1) and (4.1) respectively. This has been verified numerically by simulations in Figs. 3, 6, 8. And if the reproduction number is greater than one then the (EE) is unstable stable, this implies that all the populations are exists, for (2.1) and (4.1) respectively. This situation has been verified numerically by simulations in Figs. 4, 5, 7, 9 respectively. And the Figs. 10, 11, 12, 13, 14, 15, 16, 17 shows the graphs of $R_0 < I$ and $R_1 < I$ in terms of $\alpha_1, \alpha_2, \delta$, Λ and $\delta, \alpha_{1V}, \beta_H, \beta_V$ respectively.

Our Sensitivity analysis shows that the most effective parameters are infection rate β , infection rate of human β_H , infection rate of mosquito β_V for model (2.1) and (4.1) respectively, the Simulation results show that in epidemic situation of model (4.1), both the populations human and mosquitoes will be exists and get infected. These results are helpful in predicting malaria transmission, and how to find effective way of malaria prevention and control in the model.

Clearly, from the numerical simulations, the (DFE) is asymptotically stable whenever the reproduction number less than one and the (EE) is unstable when the reproduction number is greater than one of model (2.1) and (4.1) respectively. We notice that in order to reduce the basic reproduction number below one, we need to focused on reduction of the contact between mosquitoes and human.

7 Conclusion

In this paper, we presented two models using a deterministic system of ordinary differential equations. These are an SEIR model followed by an SEIR-SEI model which describes the transmission of malaria. We established that our models are locally asymptotically stable when the associated reproduction numbers are less than one but unstable, when they are greater than one. According to our results malaria cannot only be controlled by reducing the infection rate between humans and mosquitoes, but also by reducing the contact rate between the mosquitoes and humans, and the use of active malaria drugs, insecticides, and, treated bed nets would reduce the mosquitoes population, and that will keep the human population stable. In future, we will include optimal control approach to control the spread of malaria and bifurcation analysis in this model.

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Competing Interests

Authors have declared that no competing interests exist.

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