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Mathematical Model for Malaria Disease Transmission

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Abstract

Malaria is one of the fatal diseases caused by plasmodium parasites and transmitted to humans through biting of the female of *Anopheles* mosquitoes. We proposed a deterministic mathematical model for simulating Malaria disease transmission between humans and mosquitoes. The basic reproduction number $\mathcal{R}_0$ determined by using the next-generation matrix approach. Stability conditions for the model equilibrium points with respect to $\mathcal{R}_0$ derived and we show that the forward bifurcation occurred. When $\mathcal{R}_0 < 1$ or $\mathcal{R}_0 > 1$ the Malaria disease die out or spread, respectively. The sensitivity analysis for the basic reproduction number $\mathcal{R}_0$ fulfilled locally and globally. The model simulation was found by using Runge–Kutta fourth order method in MATLAB. Furthermore, The effects of the important parameters investigated and the obtained results presented in graphical forms. Also, we obtained that the simulation results agree with the stability analysis for $\mathrm{E}_{\mathrm{def}}$. We discussed the impacts of the Malaria disease control interventions on the important parameter for Malaria disease transmission. Recommendation for control and eradicating Malaria disease transmission provided.

Keywords: Malaria Disease, Mathematical Model, Forward Bifurcation, Sensitivity Analysis.
2010 MSC: 92B05, 34A12, 34A34.

1. Introduction

Malaria is a disease caused by protozoan parasite of the genus Plasmodium [1]. Four species of Plasmodium gene are responsible of Malaria disease infection *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale*, and *Plasmodium malariae* [1, 2]. Malaria parasite transmitted to humans via biting of infected female of *Anopheles* mosquitoes and transmitted to mosquitoes due to taking blood meal from infected human [3]. Also, Malaria parasite transmitted through sharing needle with infected person or a transfusion infected blood[4]. In the year 2021 World Health Organization (WHO) estimated 247 million infection cases by Malaria disease and 619000 deaths globally. The Africa region is the most affected region by Malaria disease where WHO estimated 234 million infection cases and 593000 deaths for the year 2021 [5]. Moreover, another regions in the world affected by Malaria disease such as South–East Asia, Eastern Mediterranean,

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Western Pacific, and Americans recorded infections and deaths cases by Malaria disease. Furthermore, Malaria disease cause many dangerous for pregnancies women, their fetus, and the new child [5].

Mathematical models have been used to help for simulating epidemics disease dynamics including Malaria disease [6]. The first mathematical model for Malaria disease transmission found since one hundred year ago by Roland Rose in 1911 [7]. Many deterministic mathematical models developed for simulating the Malaria disease transmission in [8, 9, 10, 11, 12, 13, 14]. Some authors have included climate change effect in their models in [15, 16], and some authors included Malaria disease control interventions in [17, 18, 19]. Other authors have used optimal control in [20, 21, 22]. In [9], Chitins considering a temporary immunity period for recovered humans. In [23], Koutou et al. model examined multi populations for hosts according to the host immunity for Malaria disease. In [24], Mangongo et al. added relapse ignorant infected humans compartment to their model. In [25], the effect of climate variability considered in the model and optimal control.

In this work we will use the deterministic SEIR–SEI model invented in [10] with some modifications for simulating Malaria disease transmission between humans and mosquitoes. Furthermore, we well analysis the model and also determine the important parameters for the Malaria disease transmission. Thus, we provided recommendations for control and eradicate the Malaria disease transmission.

The next sections organized as follows. In Section 2, we formulate a mathematical model for simulating Malaria disease transmission between humans and vector. Additionally, the model analysis and equilibrium points stability. In Section 3, we have done sensitivity analysis for the basic reproduction number $\mathcal{R}_0$ and determined the important parameter for Malaria disease transmission. In Section 4, the numerical simulation for the model. In Section 5, the impact of Malaria disease control interventions on our model results will discuss and recommendations for control and eradicating Malaria disease will provide. In Section 6, we will conclude the article.

2. Mathematical Formulation

In light of the model in [10] we construct SEIR–SEI model for simulating Malaria disease transmission between humans and mosquitoes. The SEIR model for humans population and SEI for vector population. The humans population divided into four classes *Susceptible* S_h, *Exposed* E_h, *Infected* I_h, and *Recovered* R_h at time t. The recovered humans gaining temporary immunity against Malaria parasite and after period of time become *Susceptible* S_h again. The vector population female of *Anopheles* mosquitoes divided into three classes *Susceptible* S_m, *Exposed* E_m, and *Infectious* I_m at time t. The *Infected* and *Recovered* humans able to transmission Malaria parasite to mosquitoes. Figure 1 show the Malaria disease transmission dynamics.

The total of humans and vector populations given by N_h and N_m, respectively as follows:

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

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

Define the incidence functions κ_h for Malaria transmission from mosquitoes to humans and κ_m from humans to mosquitoes. The incidence functions similar to the incidence functions have used in [8, 9, 26].

$$\kappa_h = \frac{\theta\beta_h I_m}{N_h}, \tag{2.3}$$

$$\kappa_m = \frac{\theta\beta_m I_h}{N_h} + \frac{\theta\overline{\beta_m} R_h}{N_h}. \tag{2.4}$$

We assuming that the mosquitoes are only *Anopheles* female, the humans and mosquitoes populations are constant, and all new recruitment in humans population are susceptible for Malaria disease.

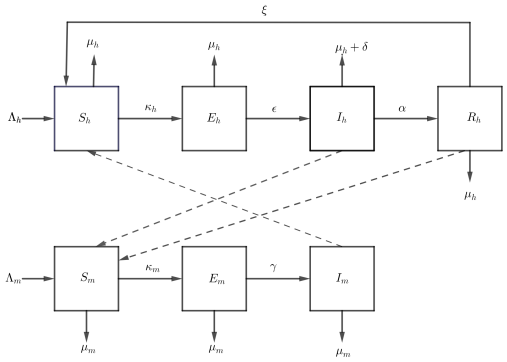

Figure 1: Transmission diagram for Malaria disease dynamic.

From the previous assumptions and schematic diagram in Figure 1 the mathematical model for Malaria disease transmission between humans and mosquitoes given by

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \kappa_h S_h - \mu_h S_h + \xi R_h,\\
\frac{dE_h}{dt} &= \kappa_h S_h - (\epsilon + \mu_h) E_h,\\
\frac{dI_h}{dt} &= \epsilon E_h - (\alpha + \mu_h + \delta) I_h,\\
\frac{dR_h}{dt} &= \alpha I_h - (\xi + \mu_h) R_h,\\
\frac{dS_m}{dt} &= \Lambda_m - \kappa_m S_m - \mu_m S_m,\\
\frac{dE_m}{dt} &= \kappa_m S_m - (\gamma + \mu_m) E_m,\\
\frac{dI_m}{dt} &= \gamma E_m - \mu_m I_m.
\end{aligned} \tag{2.5}$$

Table 1: Table explain the description of the parameters in the model (2.5).

parameter	Description	Value	Source
Λ_h	**Recruitment rate of humans population**	**2500**	[35]
Λ_m	**Recruitment rate of mosquitoes population**	**1000**	[35]
μ_h	**Human natural death rate**	**0.00004212**	[36]
δ	**Human death rate due to Malaria disease**	**0.0003454**	[36]
μ_m	**Mosquitoes death rate**	**0.033**	[9]
θ	**The rate of mosquito biting human**	**18.0**	[36]
β_h	**The rate of Malaria parasite transmission from infectious mosquito into susceptible human**	**0.022**	[9]
β_m	**The rate of Malaria parasite transmission from infected human into susceptible mosquito**	**0.8333**	[36]
$\overline{\beta_m}$	**The rate of Malaria parasite transmission from recovered humans into susceptible mosquitoes**	**0.08333**	[36]
ϵ	**The rate of human progression from *Exposed* class into *Infected* class**	**0.08333**	[36]
α	**The rate of human recovered from Malaria disease**	**0.1429**	[10]
ξ	**The rate of humans losing immunity**	**0.045**	Assumed
γ	**The rate of mosquito progression from *Exposed* class to *Infectious* class**	**0.083**	[9]

Subject to initial conditions

$$\begin{aligned} &S_h(0) \geqslant 0, \quad E_h(0) \geqslant 0, \quad I_h(0) \geqslant 0, \quad R_h(0) \geqslant 0, \\ &S_m(0) \geqslant 0, \quad E_m(0) \geqslant 0, \quad I_m(0) \geqslant 0. \end{aligned} \tag{2.6}$$

All variables and parameters in the model (2.5) are positive or non–negative. The Table 1 explain the descriptions of the parameters in the Malaria disease transmission model (2.5).

2.1. Feasible Region and Postivity of Solution

Theorem 2.1. *There is exist of a feasible region Ω such that the solutions of the model (2.5) contained and bounded.*

Proof. Adding the equations of humans classes in the model (2.5) and the equation (2.1), yield

$$\begin{aligned} \frac{dN_h}{dt} &= \Lambda_h - \mu_h N_h + \delta I_h, \\ \frac{dN_h}{dt} &\leqslant \Lambda_h - \mu_h N_h. \end{aligned} \tag{2.7}$$

The solution of the ordinary differential equation (2.7) is $N_h(t) \leqslant \left(N_h(0) - \frac{\Lambda_h}{\mu_h}\right) e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}$ and as $t \to \infty \quad 0 \leqslant N_h(t) \leqslant \frac{\Lambda_h}{\mu_h}$. Thus the feasible region for humans population given as following

$$\Omega_h = \{(S_h, E_h, I_h, R_h) \in \mathbb{R}^4_+ : S_h(t) + E_h(t) + I_h(t) + R_h(t) \leqslant \frac{\Lambda_h}{\mu_h}\}. \tag{2.8}$$

The same for mosquitoes population equations and we obtain the feasible region

$$\Omega_m = \{(S_m, E_m, I_m) \in \mathbb{R}^3_+ : S_m(t) + E_m(t) + I_m(t) \leqslant \frac{\Lambda_m}{\mu_m}\}. \tag{2.9}$$

From (2.8) and (2.9) the Malaria disease transmission model (2.5) feasible region will be $\Omega = \Omega_h \times \Omega_m$

□

Theorem 2.2. *The solutions of the model* (2.5) *with the initial data* (2.6) *are non–negative for all time* $t > 0$.

Proof. The proof will attain through Theorem B.1 in [27]. Suppose that there is exist of t^* such that $S_h(t^*) = 0$ and $S'_h(t^*) \leqslant 0$ and $S_h(t) > 0, E_h(t) > 0, I_h(t) > 0, R_h(t) > 0, S_m(t) > 0, E_m(t) > 0, I_m(t) > 0$ for $0 < t < t^*$. From the *Susceptible* human equation in model (2.5)

$$\begin{aligned}\frac{dS_h}{dt^*} &= \Lambda_h - \kappa_h(t^*)S_h(t^*) - \mu_h S_h(t^*) + \xi R_h(t^*),\\ \frac{dS_h}{dt^*} &= \Lambda_h + \xi R_h(t^*) > 0.\end{aligned}$$

And that is a contradiction then $S_h(t) > 0$.

In the *Exposed* humans equations in (2.5) if we assume $E_h(t^*) = 0$ for $t^* > 0$ and $E_h(t) > 0$ for $t \in [0, t^*)$ and by integration yield

$$E_h(t^*) = e^{-(\epsilon+\mu_h)t^*} \times \int_0^{t^*} \frac{\theta\beta_h I_m(\tau)S_h(\tau)}{N_h} e^{(\epsilon+\mu_h)\tau} d\tau + E_h(0)e^{-(\epsilon+\mu_h)t^*} > 0.$$

Then we contradict $E_h(t^*) = 0$. With the same argument we can prove that the rest of the model (2.5) quantities are positive for all time $t > 0$. □

2.2. Malaria Disease Free Equilibrium $\mathcal{E}_{dfe}$

The Malaria disease free equilibrium $\mathcal{E}_{dfe}$ of the model (2.5) given by

$$\mathcal{E}_{dfe} = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0\right). \tag{2.10}$$

2.3. The Basic Reproduction Number $\mathcal{R}_0$

P. van den Driessche et al. [28] defined the basic reproduction number $\mathcal{R}_0$ as the expected number of secondary infected cases that produced in completely susceptible population by one infected case. Using the next generation matrix approach in [28, 29] we

obtain

$$\mathcal{F} = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\theta\beta_h\Lambda_h}{N_h\mu_h} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\theta\beta_m\Lambda_m}{N_h\mu_m} & \frac{\theta\overline{\beta_m}\Lambda_m}{N_h\mu_m} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$\mathcal{V} = \begin{bmatrix} (\epsilon+\mu_h) & 0 & 0 & 0 & 0 \\ -\epsilon & (\alpha+\mu_h+\delta) & 0 & 0 & 0 \\ 0 & -\alpha & (\xi+\mu_h) & 0 & 0 \\ 0 & 0 & 0 & (\gamma+\mu_m) & 0 \\ 0 & 0 & 0 & -\gamma & \mu_m \end{bmatrix}$$

The basic reproduction number $\mathcal{R}_0$ determined by spectral radius of the matrix $\mathcal{F}\mathcal{V}^{-1}$ hereby $\mathcal{R}_0 = \rho(\mathcal{F}\mathcal{V}^{-1})$.

$$\mathcal{R}_0 = \sqrt{\frac{\theta^2\beta_h\epsilon\gamma((\xi+\mu_h)\beta_m+\alpha\overline{\beta_m})\Lambda_m\mu_h}{\mu_m^2\Lambda_h(\epsilon+\mu_h)(\alpha+\mu_h+\delta)(\xi+\mu_h)(\gamma+\mu_m)}}. \tag{2.11}$$

As exhibited in [29] the basic reproduction number $\mathcal{R}_0$ can be written as $\mathcal{R}_0 = \sqrt{\mathcal{R}_{0h}\mathcal{R}_{0m}}$ where $\mathcal{R}_{0h} = \frac{\theta\beta_h\epsilon\mu_h}{\Lambda_h(\epsilon+\mu_h)(\alpha+\mu_h+\delta)(\xi+\mu_h)}$ is the number of humans infected by Malaria disease due to one infectious mosquito. While $\mathcal{R}_{0m} = \frac{\theta\gamma((\xi+\mu_h)\beta_m+\alpha\overline{\beta_m})\Lambda_m}{\mu_m^2(\gamma+\mu_m)}$ is the number of misquotes infected by Malaria disease by one infected human.

2.4. Locally Asymptotically Stability for $\mathcal{E}_{dfe}$

Theorem 2.3. *The Malaria disease free equilibrium $\mathcal{E}_{dfe}$ is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

2.5. Globally Asymptotically Stability for $\mathcal{E}_{dfe}$

Theorem 2.4. *The Malaria disease free equilibrium point $\mathcal{E}_{dfe}$ is globally asymptotically stable if $\mathcal{R}_0 < 1$.*

Proof. We need to show that the conditions $\mathbf{H}_1$ and $\mathbf{H}_2$ in [30] are holds when $\mathcal{R}_0 < 1$. Rewrite the system (2.5) as subsystems where $X = (S_h, S_m)$, $Y = (E_h, I_h, R_h, E_m, I_m)$, and $X^* = \left(\frac{\Lambda_h}{\mu_h}, \frac{\Lambda_m}{\mu_m}\right)$, yield

$$\frac{dX}{dt} = F(X,Y),$$
$$\frac{dY}{dt} = G(X,Y), \quad G(X,0) = 0.$$

The reduced system $\frac{dX}{dt} = F(X,0)$ obtained as following:

$$\frac{dS_h}{dt} = \Lambda_h - \mu_h S_h, \tag{2.12}$$
$$\frac{dS_m}{dt} = \Lambda_m - \mu_m S_m. \tag{2.13}$$

Solutions of (2.12) and (2.13) are $S_h(t) = \frac{\Lambda_h}{\mu_h} + (S_h(0) - \frac{\Lambda_h}{\mu_h})e^{-\mu_h t}$ and $S_m(t) = \frac{\Lambda_m}{\mu_m} + (S_m(0) - \frac{\Lambda_m}{\mu_m})e^{-\mu_m t}$. Hence as $t \to \infty$ $S_h(t) \to \frac{\Lambda_h}{\mu_h}$, $S_m(t) \to \frac{\Lambda_m}{\mu_m}$ thereby X^* is globally asymptotically stable.

The matrix $A = D_Y G(X^*, 0)$ is an M–matrix given by

$$A = \begin{bmatrix} -l_1 & 0 & 0 & 0 & \theta\beta_h \\ \epsilon & -l_2 & 0 & 0 & 0 \\ 0 & \alpha & -l_3 & 0 & 0 \\ 0 & \tau_1 & \tau_2 & -l_4 & 0 \\ 0 & 0 & 0 & \gamma & -\mu_m \end{bmatrix}$$

Where $l_1 = (\epsilon + \mu_h), l_2 = (\alpha + \mu_h + \delta), l_3 = (\xi + \mu_h), l_4 = (\gamma + \mu_m), \tau_1 = \frac{\theta\beta_m\Lambda_m\mu_h}{\Lambda_h\mu_m}$, and $\tau_2 = \frac{\theta\overline{\beta_m}\Lambda_m\mu_h}{\Lambda_h\mu_m}$. We find the matrix $\hat{G}(X, Y)$ as follows

$$\hat{G}(X, Y) = \begin{bmatrix} \theta\beta_h I_h \left(1 - \frac{S_h}{N_h}\right) \\ 0 \\ 0 \\ \theta(\beta_m I_h + \overline{\beta_m} R_h)[\frac{\Lambda_m\mu_h}{\Lambda_h\mu_m} - \frac{S_m}{N_h}] \\ 0 \end{bmatrix}$$

The total population of humans and mosquitoes in steady state $N_h = \frac{\Lambda_h}{\mu_h}$, and $N_m = \frac{\Lambda_m}{\mu_m}$ thus $\hat{G}(X, Y) \geqslant 0$ in Ω and $\mathcal{E}_{dfe}$ is globally asymptotically stable whenever $\mathcal{R}_0 < 1$. □

2.6. Existence of Malaria Disease Endemic Equilibrium $\mathcal{E}_{ee}$

The endemic equilibrium point for the Malaria disease transmission model (2.5) the steady state when the Malaria disease spread. Determine the endemic equilibrium through equating all equations in the model (2.5) by zero to obtaining the equilibrium point $\mathcal{E}_{ee} = (S_h^*, E_h^*, I_h^*, R_h^*, S_m^*, E_m^*, I_m^*)$, hence

$$S_h^* = \frac{\mu_m l_1 l_2 l_4 \Lambda_h}{\theta\beta_h \epsilon\gamma\mu_h\Lambda_m} I_h^* + \frac{N_h}{\mathcal{R}_0^2}, \tag{2.14}$$

$$E_h^* = \frac{l_2}{\epsilon} I_h^*, \tag{2.15}$$

$$R_h^* = \frac{\alpha}{l_3} I_h^*, \tag{2.16}$$

$$S_m^* = \frac{l_3 N_h \Lambda_m}{\theta\pi I_h^* + \mu_m l_3 N_h}, \tag{2.17}$$

$$E_m^* = \frac{\theta\pi\Lambda_m I_h^*}{\theta l_4 \pi I_h^* + \mu_m l_3 l_4 N_h}, \tag{2.18}$$

$$I_m^* = \frac{\theta\pi\Lambda_m I_h^*}{\theta\mu_m l_4 \pi I_h^* + \mu_m^2 l_3 l_4 N_h}. \tag{2.19}$$

I_h^* is the positive root of the following equation

$$\mathcal{A}_1 {I_h^*}^2 + \mathcal{A}_2 I_h^* + \mathcal{A}_3 = 0. \tag{2.20}$$

Whereby

$$\mathcal{A}_1 = \frac{\epsilon\theta l_4\pi\mu_h}{l_3\mathcal{R}_0^2}\left(\frac{\theta^2\beta_h\gamma\mu_h\Lambda_m}{\mu_m l_4\Lambda_h} + \theta - \frac{\mu_m\xi\alpha\mathcal{R}_0^2}{\mu_h}\right),$$
$$\mathcal{A}_2 = \frac{\mu_m\epsilon\theta l_4\Lambda_h}{\mathcal{R}_0^2}\left(\pi\mathcal{R}_0^2 - \pi - 1\right) + \frac{\mu_m^2 l_4\Lambda_h}{\mu_h}\left(\xi\alpha - l_1l_2l_3\right),$$
$$\mathcal{A}_3 = \frac{\mu_m^2 l_3 l_4\Lambda_h^2}{\mu_h\mathcal{R}_0^2}\left(1 - \mathcal{R}_0^2\right).$$

With $\pi = (\xi + \mu_h)\beta_m + \alpha\overline{\beta_m}$.

There is a possibility for two endemic equilibrium points from the quadratic equation (2.20) at $\mathcal{R}_0 < 1$. Particularly, the coefficient $\mathcal{A}_1$ is always positive while $\mathcal{A}_3$ positive when $\mathcal{R}_0 < 1$ and negative at $\mathcal{R}_0 > 1$.

Theorem 2.5. *The Malaria disease transmission model (2.5) has:*

1. *A unique endemic equilibrium point if* $\mathcal{A}_3 < 0 \Longleftrightarrow \mathcal{R}_0 > 1$.
2. *A unique endemic equilibrium if* $\mathcal{A}_2 < 0$ *and* $A_3 = 0$ *or* $\mathcal{A}_2^2 - 4\mathcal{A}_1\mathcal{A}_3 = 0$.
3. *Two endemic equilibrium if* $\mathcal{A}_2 < 0, \mathcal{A}_3 > 0$ *and* $\mathcal{A}_2^2 - 4\mathcal{A}_1\mathcal{A}_3 > 0$.
4. *No endemic equilibrium otherwise.*

2.7. Locally Asymptotically Stability for $\mathcal{E}_{ee}$

Theorem 2.6. *The endemic equilibrium* $\mathcal{E}_{ee}$ *is locally asymptotically stable for* $\mathcal{R}_0 > 1$ *but close to* $\mathcal{R}_0 = 1$.

Proof. The proof will preform by using the Centre Manifold Theory as described in [Theorem 4.1 ,[31]]. Starting by changing the variables in the system (2.5) therefore $S_h = z_1, E_h = z_2, I_h = z_3, R_h = z_4, S_m = z_5, E_m = z_6, I_m = z_7$ and $z = (z_1, z_2, z_3, z_4, z_5, z_6, z_7)^T$. The model (2.5) written as

$$\begin{aligned}
\frac{dz_1}{dt} &= f_1 = \Lambda_h - \kappa_h z_1 - \mu_h z_1 + \xi z_4,\\
\frac{dz_2}{dt} &= f_2 = \kappa_h z_1 - (\epsilon + \mu_h) z_2,\\
\frac{dz_3}{dt} &= f_3 = \epsilon z_2 - (\alpha + \mu_h + \delta) z_3,\\
\frac{dz_4}{dt} &= f_4 = \alpha z_3 - (\xi + \mu_h) z_4,\\
\frac{dz_5}{dt} &= f_5 = \Lambda_m - \kappa_m z_5 - \mu_m z_5,\\
\frac{dz_6}{dt} &= f_6 = \kappa_m z_5 - (\gamma + \mu_m) z_6,\\
\frac{dz_7}{dt} &= f_7 = \gamma z_6 - \mu_m z_7.
\end{aligned} \tag{2.21}$$

The bifurcation parameter chosen to be β_h and at $\mathcal{R}_0 = 1$ obtaining $\beta_h = \beta_h^* = \frac{\mu_m^2 l_1 l_2 l_3 l_4 \Lambda_h}{\theta^2 \epsilon \gamma \pi \mu_h \Lambda_m}$.
The linearization matrix for the system (2.21) at $\mathcal{E}_{dfe}$ and $\beta_h = \beta_h^*$ is given as follows

$$D_z f = \begin{bmatrix} -\mu_h & 0 & 0 & \xi & 0 & 0 & -\theta\beta_h^* \\ 0 & -l_1 & 0 & 0 & 0 & 0 & \theta\beta_h^* \\ 0 & \epsilon & -l_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha & -l_3 & 0 & 0 & 0 \\ 0 & 0 & -\tau_1 & -\tau_2 & -\mu_m & 0 & 0 \\ 0 & 0 & \tau_1 & \tau_2 & 0 & -l_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma & -\mu_m \end{bmatrix}. \tag{2.22}$$

It is clear that the matrix (2.22) has one zero eigenvalue and six eigenvalues with negative real part and then we can use [Theorem 4.1, [31]].

The right eigenvectors associated with the zero eigenvalue $\omega = (\omega_1, \omega_2, \omega_3, \omega_4, \omega_5, \omega_6, \omega_7)^T$ are

$$\begin{aligned} \omega_1 &= -\frac{l_4 \mu_m \Lambda_h}{\theta \epsilon \mu_h^2 \Lambda_m} (l_1 l_2 l_3 - \xi \alpha \epsilon)\, \omega_6 < 0, \quad \omega_2 = \frac{l_2}{\epsilon}\omega_3 > 0, \\ \omega_3 &= \frac{l_4 l_3}{l_3\tau_1 + \alpha\tau_2}\omega_6 > 0, \quad \omega_4 = \frac{\alpha l_4}{l_3\tau_1 + \alpha\tau_2}\omega_6 > 0, \\ \omega_5 &= -\frac{l_4}{\mu_m}\omega_6 < 0, \quad \omega_6 = \omega_6 > 0, \\ \omega_7 &= \frac{\gamma}{\mu_m}\omega_6 > 0. \end{aligned}$$

The left eigenvectors corresponding to the zero eigenvalue $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)$ are given by

$$\begin{aligned} v_1 &= v_5 = 0, \\ v_2 &= \frac{\mu_m}{\theta\beta_h^*}v_7 > 0, \quad v_3 = \frac{\epsilon}{l_1}v_2 > 0, \\ v_4 &= \frac{\gamma\tau_2}{l_3 l_4}v_7 > 0, \quad v_6 = \frac{\gamma}{l_4}v_7 > 0, \\ v_7 &= v_7 > 0. \end{aligned}$$

Compute a and b in [Theorem 4.1 ,[31]] then determine the non–vanishing partial derivatives for f_2 and f_6. Firstly, to compute a the non–vanishing partial derivatives are

$$\begin{aligned} \frac{\partial^2 f_2}{\partial z_1 \partial z_7} &= \frac{\partial^2 f_2}{\partial z_7 \partial z_1} = \frac{\theta\beta_h^*\mu_h}{\Lambda_h}, \\ \frac{\partial^2 f_6}{\partial z_5 \partial z_3} &= \frac{\partial^2 f_6}{\partial z_3 \partial z_5} = \frac{\theta\beta_m\mu_h}{\Lambda_h}, \\ \frac{\partial^2 f_6}{\partial z_5 \partial z_4} &= \frac{\partial^2 f_6}{\partial z_4 \partial z_5} = \frac{\theta\overline{\beta_m}\mu_h}{\Lambda_h}. \end{aligned}$$

Now we obtain

$$a = \sum_{k,j,i=1}^{n} v_k \omega_i \omega_j \frac{\partial^2 f_k}{\partial z_i \partial z_j}(0,0) = 2\frac{\theta\mu_h}{\Lambda_h}\left[v_2\omega_1\omega_7\beta_h^* + v_6\omega_5\omega_3\beta_m + v_6\omega_5\omega_4\overline{\beta_m}\right] < 0.$$

For b we need to find the non–vanishing partial derivative for f_2 only and we obtain

$$\frac{\partial^2 f_2}{\partial z_7 \partial \beta_h^*} = \theta.$$

Thus

$$b = \sum_{k,i=1}^{n} \nu_k \omega_i \frac{\partial^2 f_k}{\partial z_i \partial \beta_h^*}(0,0) = \nu_2 \omega_7 \theta > 0.$$

Since $a < 0$ and $b > 0$ from item (iv) in Theorem 4.1 in [31] the endemic equilibrium point $\mathcal{E}_{ee}$ is locally asymptotically stable at $\mathcal{R}_0 > 1$. Also, forward bifurcations determined for the model which mean that the Malaria disease transmission eradicated at $\mathcal{R}_0 < 1$ and spread at $\mathcal{R}_0 > 1$. □

3. Sensitivity Analysis

In this section, sensitivity analysis of the basic reproduction number $\mathcal{R}_0$ will be done locally and globally. Through the sensitivity analysis we will determine the impact of the model (2.5) parameters on Malaria disease transmission.

3.1. Local Sensitivity Analysis

Applying the normalized forward sensitivity index approach in [32, 33] to obtain the sensitivity analysis for $\mathcal{R}_0$ and parameter h we compute $\Upsilon_h^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial h} \times \frac{h}{\mathcal{R}_0}$. The results presented in Table 2 and we find that the most sensitive parameters for $\mathcal{R}_0$ are μ_m, β_h, Λ_m, and α. We can say that as θ increase(or decrease) then $\mathcal{R}_0$ increase(or decrease) by 10% and as Λ_m increase(or decrease) then $\mathcal{R}_0$ decrease(or increase) by 11.422%.

3.2. Global Sensitivity Analysis

Sensitivity analysis for the basic reproduction number $\mathcal{R}_0$ will be achieved by using Partial Ranking Correlation Coefficient(PRCC) method described in [34] to calculate the sensitivity indices for $\mathcal{R}_0$ parameters. Latin Hybercube Sampling(LHS) in [34, 24] it is a sampling method based on Monte Carlo simulation used for generating samples for $\mathcal{R}_0$ uncertain parameters and it will use for determine PRCC values for each uncertain parameter. We preformed the PRCC for the basic reproduction number $\mathcal{R}_0$ and the obtained results presented in Table 2. Also, we obtain that the highly PRCC value parameters are μ_m, Λ_m, β_h, β_m, γ, and δ. It will be meaningful to consider that the parameters relating to transfer Malaria disease, and the parameters relating to amount of mosquitoes have great influence on $\mathcal{R}_0$.

From the local and global sensitivity analysis of $\mathcal{R}_0$ we find that the utmost sensitive parameters are μ_m, Λ_m, θ, β_h, and α. Furthermore, we deduced that reducing the contact between humans and mosquitoes, shorting mosquitoes lifespan, and effective Malaria disease drug will lead to reducing Malaria disease transmission.

Table 2: Table shows sensitivity index and PRCC values for $\mathcal{R}_0$ to the model (2.5) parameters.

parameter	$\gamma_{\mathbf{h}}^{\mathcal{R}_0}$	**PRCC**
θ	1.0	0.0538
β_h	0.5	0.5818
Λ_m	0.5	0.5940
Λ_h	-0.5	-0.6108
$\frac{\beta_m}{\overline{\beta}_m}$	0.3796	0.5733
	0.1204	0.0520
ϵ	0.00025260	-0.0159
α	-0.4984	0.0178
δ	-0.0015	-0.5683
ξ	-0.1203	0.0006
γ	0.2845	0.6133
μ_m	-1.1422	-0.8947
μ_h	0.4991	0.3387

4. Numerical Simulation

In this section, the numerical simulation for the model(2.5) will be done through using Runge–Kutta fourth order method in MATLAB R2015a and the parameters values from Table 1. In particularly, we are interest in the effects of the critical parameters that determined in Section 3 in the Malaria Transmission model (2.5) classes.

Figure 2, shows the effect of θ on *Exposed* and *Infected* humans classes. We obtain that as

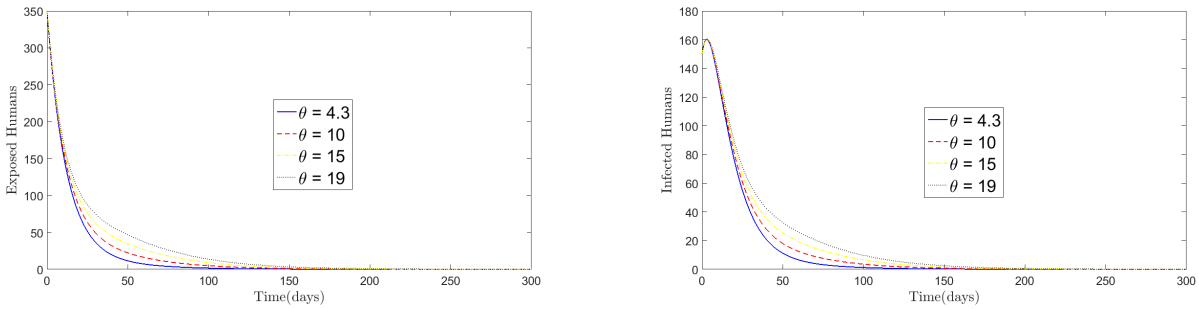

Figure 2: The effect of θ on humans *Exposed* and *Infected* classes.

θ increase *Exposed* and *Infected* humans classes increases too. In Figure 3, we obtain that increase of θ cause increase in *Exposed* and *Infectious* mosquitoes classes. From Figures 2 and 3 we deduced that increase of contact rate between humans and mosquitoes lead to increase of Malaria disease transmission and increase Malaria infection cases.

Figure 4, shows that increase the mosquitoes death rate μ_m lead to decrease *Exposed* and *Infectious* mosquitoes classes. Which means shorting lifespan for mosquitoes able to transmitted the Malaria parasite. From Figure 5, we obtain that increase of μ_m lead to decrease *Exposed* and *Infected* human classes. Which mean reduce Malaria infection cases.

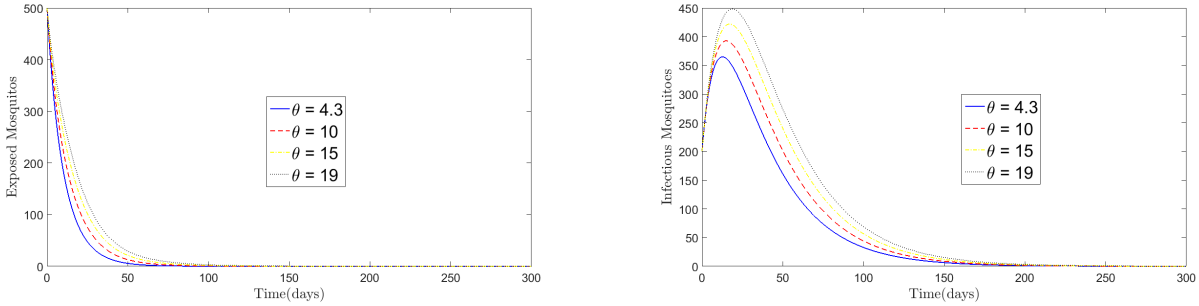

Figure 3: The effect of θ on mosquitoes *Exposed* and *Infectious* classes.

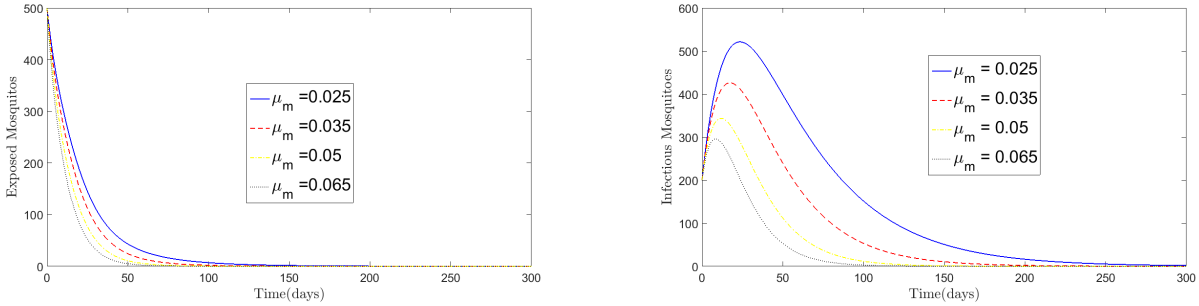

Figure 4: The effect of μ_m on mosquitoes *Exposed* and *Infectious* classes.

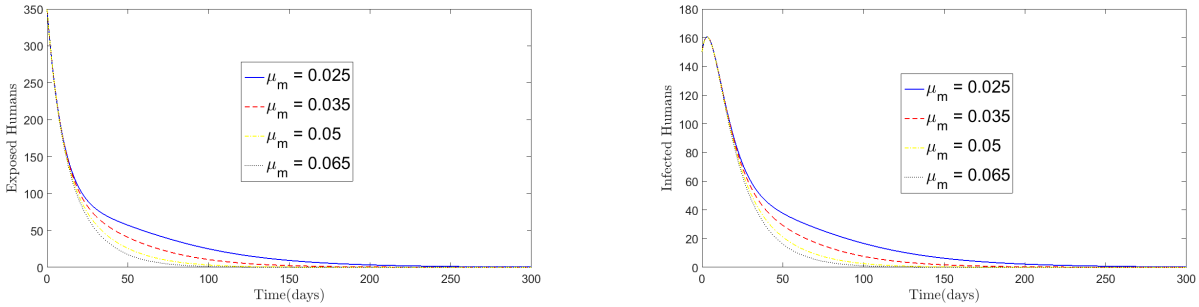

Figure 5: The effect of μ_m on humans *Exposed* and *Infected* classes.

From Figure 6, we obtain that increase of mosquitoes new recruitment rate Λ_m lead to increase mosquitoes per capita. Furthermore, the *Exposed*, and *Infectious* mosquitoes classes increases thereby Malaria disease transmission risk increase too. In Figure 7, we see that increasing of β_h lead to increase the *Exposed* and *Infected* humans classes. As a result of increase of Malaria parasite transmission from mosquitoes to humans thus Malaria cases increase. In Figure 8, we see that increase of recovering rate for infected human α leads to decrease the *Infected* humans class and increase *Recovered* humans class.

5. Malaria disease control interventions

According to the Center for Disease Control and Prevention [37] the control interventions against Malaria disease are Insecticide–Treated bed Nets(ITN), Indoor Residual

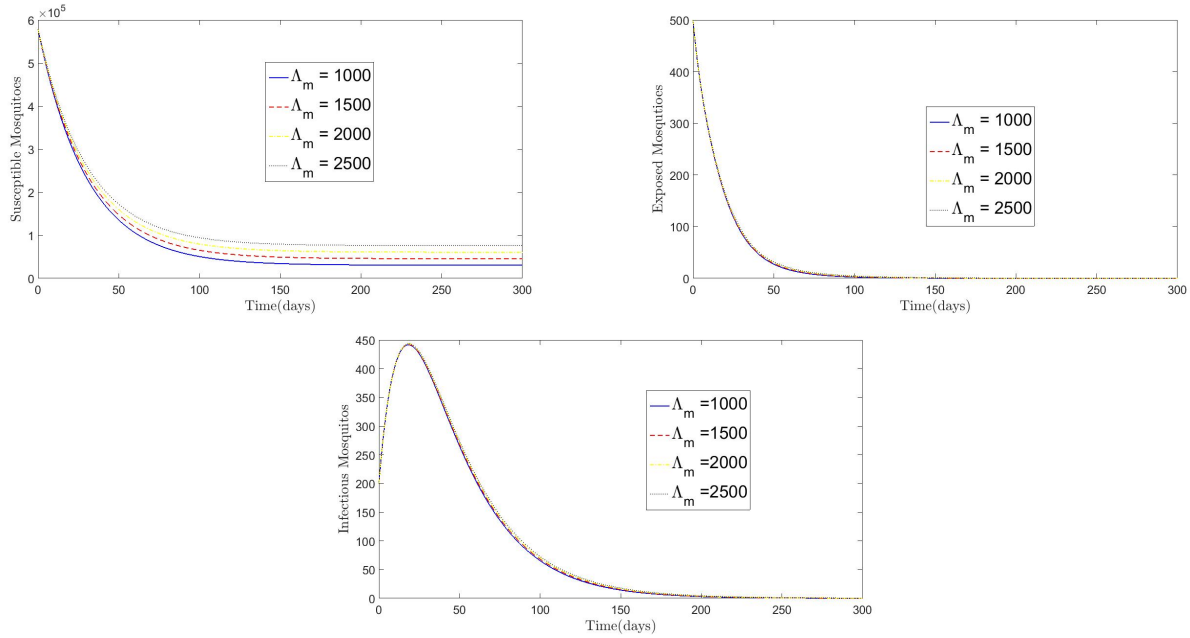

Figure 6: The effect of Λ_m on mosquitoes population.

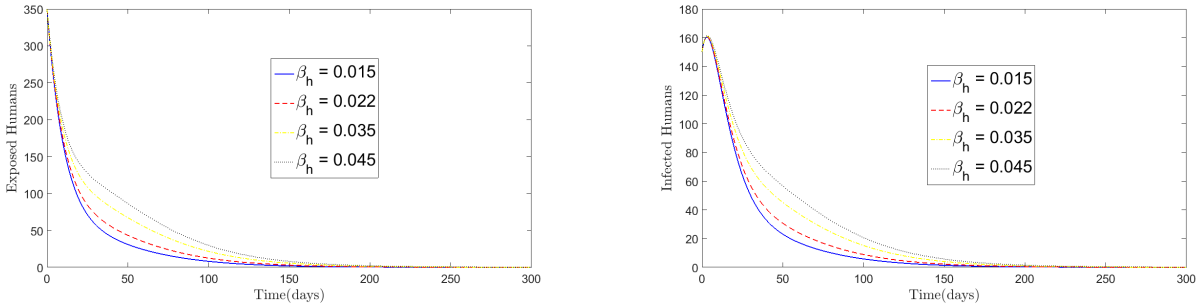

Figure 7: The effect of β_h on humans *Exposed* and *Infected* classes.

Spraying(IRS), Intermittent preventive treatment of malaria for pregnant women (IPTp), and Intermittent preventive treatment of malaria for infancy (IPTi).

According to the results deduced in Sections 3 and 4 we will discuss effects of Malaria disease interventions for controlling and eradicating Malaria disease.

The interventions (ITN) and (IRS) lead to reduce the contact between humans and mosquitoes and shorting lifespan of mosquitoes therefore reducing θ and increase μ_m. In addition, using (ITN) lead to decrease Λ_m since (ITN) prevent *Anopheles* females from take blood meal for eggs production. From [38, 39] we can use interventions that guarantee increase μ_m and decrease Λ_m such as Space spraying, and Larval control measures.

The interventions (IPTp) and (IPTi) increase the immunity for apprentices women and infancy. The parameter β_h can be reduced through (IPTp) and (IPTi) and effective anti–malaria drugs. Using effective Malaria disease drugs help in increase numbers of humans recovered from the Malaria infections which mean increase of α.

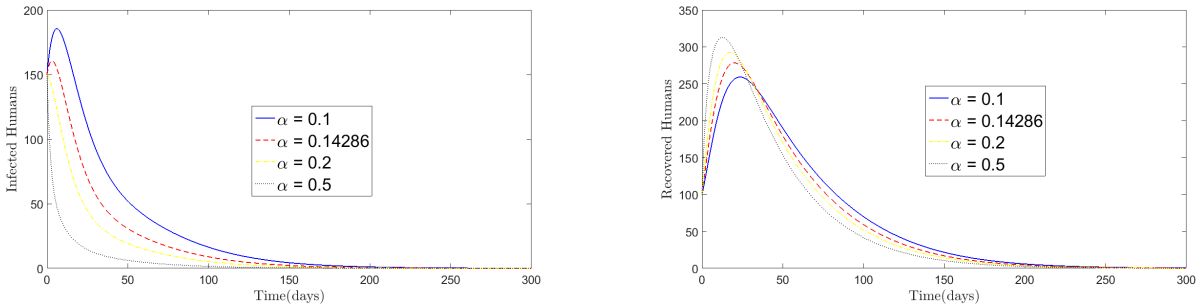

Figure 8: The effect of α on humans *Infected* and *Recovered* classes.

For control and eradicate Malaria disease transmission we recommending reducing the contact between humans and mosquitoes, reducing the amount of mosquitoes per capita, and effective treatment for infected human individuals.

6. conclusion

In this article, a deterministic model for Malaria disease transmission between humans and mosquitoes developed. The feasible region, positive solution, and the basic reproduction number $\mathcal{R}_0$ presented in details. Existence and stability analysis for the model equilibrium points discussed in details and forward bifurcation occurred. We found that when $\mathcal{R}_0 < 1$ the Malaria disease die out and at $\mathcal{R}_0 > 1$ the Malaria disease spread. Through the sensitivity analysis for $\mathcal{R}_0$ we determined the utmost sensitive parameters for $\mathcal{R}_0$. Using Runge–Kutta fourth order method in MATLAB for the model simulation and we investigated the effects of important parameters for Malaria disease transmission in the model (2.5). Furthermore, the obtained results presented in graphical forms. We found that the simulation results agree with the stability of $\mathcal{E}_{\mathtt{def}}$. We discussed the effects of Malaria disease control interventions on the critical parameters for Malaria disease transmission. Lastly, Recommendation for control and eradicating Malaria disease transmission provided.

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