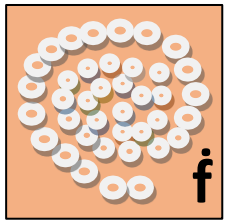

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Stability analysis of a Malaria epidemic model transmission dynamics

I. Aliyu*, R.T. Abah†, and F.O. Ogunfiditimi‡

Abstract

Malaria is a vector-borne disease that has been haunting mankind and still remains a major public health issue. It is caused by the parasites, from the genus Plasmodium, which spread to people through the bite of infected adult female mosquitoes of the Anopheles species. In 2017, an estimated 219 million cases occurred worldwide with children accounting for 61% of deaths. In this paper, we formulate a mathematical model that reduces malaria transmission dynamics and investigate the basic properties of the model; the disease Free Equilibrium and the basic reproduction number of the model were obtained. The Jacobian matrix stability technique was used to analysed the stability of the disease free equilibrium. The analysis shows that the disease free equilibrium is locally asymptotically stable that is $R_0 < 1$.This discovery indicates that, malaria transmission can be reduced or eliminated from the population.

Keywords and phrases: model formulation; properties of the model; equilibrium points; basic reproduction number; stability analysis

1 Introduction

Malaria is a vector-borne disease that has been haunting mankind and still remains a major public health issue. It is caused by the parasites, from the genus Plasmodium, which spread to people through the bite of infected adult female mosquitoes of the Anopheles species. There are more than 200 species of the genus Plasmodium identified to be parasitic to reptiles, birds, and mammals. Four Plasmodium species are known to cause human malaria, namely, Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale, and Plasmodium malariae. Plasmodium falciparum tends to be the species causing the most complications and has high mortality if untreated. Humans occasionally become infected with Plasmodium species that normally infect animals, such as Plasmodium knowlesi, but there are no reports of human-mosquito-human transmission of such zoonotic forms of malaria [1]. Although this disease is preventable and curable, malaria continues to claim the lives of more than $435,000$ people each year, largely in Africa. In 2017, an estimated 219 million cases occurred worldwide with children accounting for 61% of deaths. Plasmodium falciparum is the most prevalent malaria parasite in the African region, accounting for 99.7% of estimated malaria cases in 2017 [1]. Mathematical models have been useful in malaria control efforts and they become even more important when malaria elimination is in the foreseeable future [2]. Sir Ronald Ross was the first person to develop a mathematical model which aimed at studying malaria transmission and control [3]. He developed the model in 1916 which had a basic deterministic formula [4]. The models have been employed in malaria control studies and they have been very helpful in controlling malaria [5]. Malaria transmission models play a significant role in understanding the dynamics of the disease [6]. They have long been applied to assess the possible means of intervention ([7]; [8]). In their study, the dynamics have investigated through deterministic

*Corresponding Author; Ibrahim.aliyu@fedpolybida.edu.ng; Department of Mathematics Federal Polytechnic Bida, Niger State, Nigeria

†Department of Mathematics University Abuja, Abuja, Nigeria; E-mail: roseline.abah@uniabuja.edu.ng

‡Department of Mathematics University Abuja, Abuja, Nigeria; E-mail: viieh@yahoo.com

models [9]. Many studies have been carry out on the transmission dynamics and control of malaria. [10] developed a mathematical model and used it to demonstrate the effects of varying the transmission parameters in order to suggest control of malaria infection level to a tolerable limit. This was done in order to have an environment where malaria transmission is reduced to a tolerable level. They observed that reducing the probability that a susceptible mosquito becomes infected is a good target for intervention in the spread of malaria. But model did not include many features in malaria transmission, such as awareness, protected populations. [11] presented a Modeling and Optimal Control Analysis for Malaria Transmission with Role of Climate Variability. They applied the optimal control theory to describe the optimal control model that incorporates three controls, namely, using treated bed net, treatment of infected with antimalaria drugs, and indoor residual spraying strategy. The Pontryagin's maximum principle is introduced to obtain the necessary condition for the optimal control problem. [11] observed that the numerical simulation of optimality system and cost-effectiveness analysis reveals that the combination of treated bed net and treatment is the most optimal and least-cost strategy to minimize the malaria. Unfortunelly the recovered population progresses back to susceptible without precaution or protection against mosquito bite. Dynamics and control measures for malaria using a mathematical epidemiological model by [12]. The research investigates the transmission dynamics of malaria disease and the different ways the disease can be controlled. They concluded that, if the breeding sites of mosquitoes are reduced and insecticides are well sprayed, it will go a long way to eradicate the vector in the population. But their model did not provide way of reducing the rate at which the recovered population join the susceptible population. In 2020, a mathematical model approach on Impact of Awareness to Control Malaria Disease was developed by [13]. The model assumed that, due to the effect of awareness campaign, the aware infected individuals avoid contact with mosquitoes. The model simulation results have significantly shown that awareness is an important factor to fight against this deadly disease. Thus, the spread of this illness could be prevented through effective awareness strategies in regions, where it has rapid spread. It is not sufficiently realistic or suitable for the aware population to only avoid contact with the infected population. Despite the existing malaria control strategies, reported cases of malaria are still quite high and the disease remains one of the health problems with major causes of death for developing countries. Researchers have developed some malaria models to help proffer solutions to the disease but the problem persists in many countries, where most cases are found in Africa. For the above reason, there is need for more research on malaria to eradicate or reduce the transmission. The purpose of this research is develop and analyze a mathematical model of endemic malaria transmission dynamics that incoporating protection population and a population that accessed malaria intervention .

2 Methodology/Model formulation

The model is formulated by considering the human and mosquito populations divided into subgroups. The human population consists of protected humans P_h, susceptible humans S_h, exposed humans E_h, infected humans I_h, treated humans T_h, untreated humans L_h, recovered humans R_h, and accessed malaria intervention humans A_h . The mosquito population consists of susceptible mosquitoes S_m, exposed mosquitoes E_m and infected mosquitoes I_m. The total population sizes at time (t) for humans and mosquitoes are denoted by $N_h(t)$ and $N_m(t)$, respectively. In the model incorporate control parameters for the protected human population, susceptible human population and the exposed human population, denoted by y, y_1 and y_2 respectively. $P_h(t)$ represents the number of individuals that are protected from malaria infection, $S_h(t)$ represents the number of individuals not infected with the malaria parasite but vulnerable to the infection at time (t), $E_h(t)$ represents individuals who are infected with malaria parasite but not yet infectious, $I_h(t)$ represents individuals infected with malaria and are capable of transmitting the disease to susceptible mosquitoes, $T_h(t)$represents individuals that have been infected of malaria and treated, $L_h(t)$represents individuals infected with malaria but not treated, $R_h(t)$ represents individuals who have recovered from the disease and started taking all forms of precautions against malaria and $A_h(t)$ represents individuals who have accessed malaria intervention

and adhered to all forms of precaution against mosquito bite. The protected humans $P_h(t)$ individuals are recruited at the rate $\gamma\omega$ where γ is the Proportion of recruited human protected from birth, susceptible humans $S_h(t)$ are recruited at the rate ω. Individuals in this class die naturally at the rate μ_h or proceed to the exposed class by acquiring malaria through contact with infectious mosquitoes at a rate τ_h or move to the join the population that have accessed malaria intervention at the rate d. Exposed individuals E_h either progress to the infectious class after the development of clinical symptoms at the rate $\alpha_h$or move to the join the population that have accessed malaria intervention at the $\alpha_1(1-y_2)$, when $y_2 = 0$, means that the campaign is effective on the exposed population, some of the exposed individuals can move to the population that accessed intervention, when $y_2 = 1$, it means that the campaign is not effective on the exposed population, therefore no exposed individual move to join the population that accessed intervention . Infectious individuals I_h move to the treated population at the rate of ϕ and untreated population at the rate κ, infected individuals die due to malaria disease at rate θ and natural death μ_h. The treated T_h and untreated L_h individuals recovered at the rate σ and φ, respectively and die natural at the rate μ_h. The recovered population either die naturally at the rate μ_h or progress to the population that accessed malaria intervention at the rate β. Population that accessed malaria intervention A_h either moves to susceptible population due to wane in practicing precautions against mosquito bite at the rate η or progress to protected population with time at the rate $\delta(1-\eta)$. Natural death occurs in this population at the rate μ_h. Susceptible mosquitoes S_M are recruited at the rate ω_m and acquire malaria infection through contact with humans infected with malaria at the rate τ_m. Mosquito population is also reduced as a result of natural death at the rate μ_m or move into the exposed class E_m at the rate α_m and progresses to the class of infected mosquitoes I_m. All classes of mosquitoes die naturally at the rate μ_m .

2.1 Model Assumptions

The following assumptions are made in order to formulate the equations of the model:

- Considers the population of those who are aware of malaria through campaign as a separate compartment.
- The entire recovered human population proceed to access malaria intervention after recovery.
- Mosquito never recover from infection, that is, the vector die after infecting human.
- Having accessed malaria intervention; people will take all necessary precaution, such as cleaning environment, spray of insecticide, use of mosquito net and all other form of precaution against mosquito bites.
- Total human and mosquito populations are not constant.
- All parameters in the model being nonnegative.
- No recovered human move to susceptible class.

2.2 Schematic Representation of the Model

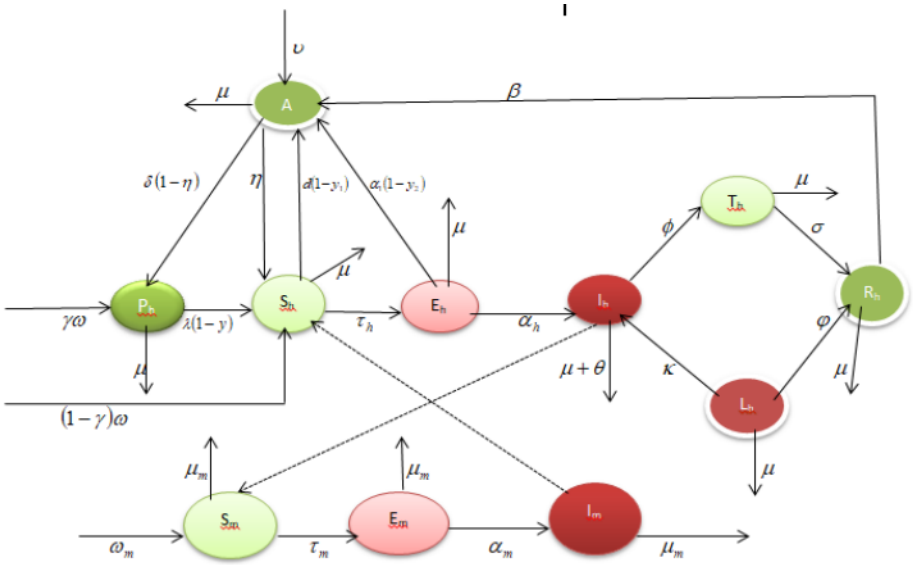

Figure 1: Schematic representation of the malaria transmission model

2.3 Model Equations

$$\frac{dP_h}{dt} = \gamma\omega_h + \delta(1-\eta)A_h - (\lambda(1-y) + \mu_h)P_h \tag{1}$$

$$\frac{dS_h}{dt} = (1-\gamma)\omega_h + \eta A_h + \lambda(1-y)P_h - \tau_h S_h - (\mu_h + d(1-y_1))S_h \tag{2}$$

$$\frac{dE_h}{dt} = \tau_h S_h - \alpha_h E_h - (\alpha_1(1-y_2) + \mu_h)E_h \tag{3}$$

$$\frac{dI_h}{dt} = \alpha_h E_h - (\phi + \kappa + \mu_h + \theta_h)I_h \tag{4}$$

$$\frac{dT_h}{dt} = \phi I_h - (\sigma + \mu_h)T_h \tag{5}$$

$$\frac{dL_h}{dt} = \kappa I_h - (\varphi + \mu_h)L_h \tag{6}$$

$$\frac{dR_h}{dt} = \sigma T_h + \varphi L_h - (\beta + \mu_h)R_h \tag{7}$$

$$\frac{dA_h}{dt} = \nu + \beta R_h + \alpha_1(1-y_2)E_h + d(1-y_1)S_h - (\delta(1-\eta) + \eta + \mu_h)A_(h) \tag{8}$$

$$\frac{dS_m}{dt} = \omega_m - \tau_m S_m - \mu_m S_m \tag{9}$$

$$\frac{dE_m}{dt} = \tau_m S_m - \alpha_m E_m + \mu_m E_m \tag{10}$$

$$\frac{dI_m}{dt} = \alpha_m E_m - \mu_m I_m \tag{11}$$

Together with initial conditions

$$\left\{\begin{array}{ccc} P_h(0) > P_h0, & S_h(0) > S_{h0}, & E_h(0) > E_{h0}, \\ I_h(0) > I_{h0}, & T_h(0) > T_{h0}, & L_{h0} > L_{h0}, \\ A_{h0} > A_{h0}, & R_h(0) > R_{h0}, & S_m(0) > S_{m0}, \\ & E_m(0) > E_{m0}, & I_m(0) > I_{m0} \end{array}\right\}. \tag{12}$$

Where $N_h = P_h + S_h + E_h + I_h + T_h + U_h + R_h + A_h$ is the total population of host (humans) and $N_m = S_m + E_m + I_m$ is the total population of vector (mosquitoes). $T_h = \frac{b\rho_h I_m}{N_h}$ is the force of infection of host and $T_m = \frac{b\rho_m I_m}{N_m}$ is the force of infection of the vector.

2.4 Model parameter and variable description

The following Table 2 gives a summary of the variables' descriptions used herein:

Table 1: Definition of Variables

Variables	Description
P_h	Total protection of human population at time t
S_h	Total population of susceptible human at time t
E_h	Total population of exposed human at time t
I_h	Total population of infected human at time t
T_h	Total population of treated human at time t
L_h	Total population of untreated and ignorant human at time t
R_h	Total population of recovered human at time t
A_h	Total population of humans that accessed malaria intervention at time t
S_m	Total population of susceptible mosquitoes at time t
E_m	Total population of exposed mosquitoes at time t
I_m	Total population of infected mosquitoes at time t

Table 2: Definition of Parameters

Variables	Description
ω	Rate of human recruitment
γ	Proportion of recruited human protected from birth
λ	Rate at which protected humans loses their protection
μ_h	Natural death rate of human
b	Biting rate of mosquitoes
ρ_h	Probability of biting by an infected mosquito to susceptible human
α_h	Rate at which exposed humans become infected
α_1	Rate at which exposed humans progresses to access malaria intervention
ϕ	Rate at which infected humans are treated
κ	Rate at which human ignore treatment
φ	Rate at which untreated humans recovered
β	Rate of progression to access malaria intervention from recovering by humans
d	Rate of progression to intervention class from susceptible by human
η	Rate of waning from intervention class to susceptible class
δ	Rate at which human become protected from the disease
θ	Death rate for human due to infection
σ	Rate at which treated human recovered from disease
α_m	Rate at which exposed mosquitoes become infected
ρ	Transmission rate on a susceptible mosquito
ν	Rate of intervention
μ_m	Natural death rate for mosquitoes
ω_m	Recruitment rate of mosquitoes
y	control parameter for protected individuals
y_1	rate of awareness of intervention for susceptible individuals
y_2	rate of awareness of intervention for exposed individuals

2.5 Basic Properties of the Model

2.5.1 Positivity of the Model

Here we show that the model equations (1) to (11) together with (12) to be epidemically meaningful and mathematically correct.

Theorem 2.1. *It is always necessary to show that solutions $P_h, S_h, E_h, I_h, T_h, R_h, A_h, S_m, E_m, I_m$ of the model equations (1) to (11) together with (12) are positive for all time $t \geq 0$.*

Proof. From equation (1) in the model

$$\frac{dP_h}{dt} = \gamma\omega_h + \delta(1-\eta)A_h - (\lambda(1-y) + \mu_h)P_h - \mu_h S_h$$

$$\frac{dP_h}{dt} \geq -(\lambda(1-y) + \mu_h)P_h \tag{13}$$

Applying separation of variables and integrating both sides of (13) we have,

$$P_h(t) \geq Be^{-\int_0^t (\lambda(1-y)+\mu_h)t} \tag{14}$$

At $P_h(0) = P_{h0}$ that is $t = 0$, equation (14) becomes

$$P_h(t) \geq B \tag{15}$$

Therefore, equation (14) becomes

$$P_h(t) \geq P_{h0} e^{-\int_0^t (\lambda(1-y)+\mu_h)t} \tag{16}$$

The inequality in equation (16) shows that $P_h(t) \geq P_{h0} \geq 0$ and is always positive for all t. Similarly, from the susceptible human population S_h in the model that is,

$$\frac{dS_h}{dt} = (1-\gamma)\omega_h + \eta A + \Lambda(1-y)P - T_h S_h - (\lambda_h + d(1-y_1))S_h$$

$$\frac{dS_h}{dt} \geq -(T_h + \mu_h + d(1-y_1))S_h \tag{17}$$

By applying separation of variable and integrating both sides of equation (17), we have.

$$S_h(t) \geq De^{-\int_0^t (T_h+\mu_h+d(1-y_1))t} \tag{18}$$

At $t = 0$ that is $S_h(0) = S_{h0}$, equation (18) becomes

$$S_h(0) \geq S_{h0} \tag{19}$$

Therefore, equation (18) becomes

$$S_h(t) \geq S_{h0} e^{-\int_0^t (\lambda_h+\mu_h)du} \geq 0 \tag{20}$$

The inequality in equation (20) shows that $S_h(t) \geq S_{h0} \geq 0$ and is always positive for all t.

Similarly, using the same procedure, the remaining state variables of the model equations (1) to (11) will positive for all time t.

2.5.2 Invariant Region

The invariant region is used to determine where the model solution is bounded. The model equation (1) to (11) is divided into two parts, the human population and the mosquitos' population. The total human population is represented by $N_h = P_h + S_h + E_h + I_h + T_h + U_h + R_h + A_h$. The total mosquitoes population is $N_m = S_m + E_m + I_m$.

Theorem 2.2. *Let* $\Omega_h = \{P_h, S_h, E_h, I_h, T_h, R_h, A_h, S_m, E_m, I_m \in \Re_+^8 : N_h(t) \leq \frac{\omega_h}{\mu_h}\}$ *and* $\Omega_m = \{S_m, E_m, I_m \in \Re_+^3 : N_m(t) \leq \frac{\omega_m}{\mu_m}\}$ *so that* $\Omega = \Omega_h \times \Omega_m \in \Re_+^8 \times \Re_+^3$. *The biologically feasible region of the* Ω *of the model equation (1) to (11) is positively invariant.*

Proof. From the total human population represented by $N_h = P_h + S_h + E_h + I_h + T_h + U_h + R_h + A_h$, It is clear that,

$$\frac{dN_h}{dt} \leq \omega_h - \mu_h N_h \tag{21}$$

Applying separation of variable and integrating, we have,

$$\omega_h - \mu_h N_h \leq D_1 e^{-}\mu_h t \tag{22}$$

At $t = 0$, $N_h(t) = N_0$ equation (22) becomes

$$\omega_h - \mu_h N_h = D_1 t$$

Therefore, equation (22) becomes

$$\omega_h - \mu_h N_h \leq (\omega_h - \mu_h N_h)e^{-\mu_h t}$$

$$N_h(t) \leq \frac{\omega_h}{\mu_h} - \frac{(\omega_h - \mu_h N_h)e^{-\mu_h t}}{\mu_h} \tag{23}$$

if $t \to \infty$ equation (23) becomes

$$N_h(t) \leq \frac{\omega_h}{\mu_h} \tag{24}$$

This implies that $0 \leq N_h(t) \leq \frac{\omega_h}{\mu_h}$.

Thus, for the host population, the feasible solution set of the model remain within the region and is given by $\Omega_h = \{P_h, S_h, E_h, I_h, T_h, R_h, A_h, S_m, E_m, I_m \in \Re_+^8 : N_h(t) \leq \frac{\omega_h}{\mu_h}\}$.

Similarly, the equation given by the total vector (mosquitoes) population in the model equation (1) to (11), that is

$$\frac{dN_m}{dt} = \omega_m - \mu_m N_m \tag{25}$$

By solving the equation (25), we have

$$N_m(t) \leq \frac{\omega_m}{\mu_m} \tag{26}$$

From equation (26), if $t \to \infty, N_m(t) \to \frac{\omega_m}{\mu_m}$.

Thus, for the total mosquitoes population the feasible solution set of the model remain within the region and is given by $\Omega_v = \{(S_m, E_m, I_m) \in \mathbb{R}_+^3 : N_m(t) \leq \frac{\omega_v}{\mu_v}\}$.

Therefore, the feasible solution set for the model equations (1) to (11) together with initial conditions given by $\Omega = \Omega_h \times \Omega_m$, is a positive invariant and hence it is biologically meaningful and well posed in the domain Ω.

3 Analysis of the Equilibrium Points

By equilibrium of a given set of equation(s), we mean the state of the set of equation(s) remaining the same at all times. Therefore, by equilibrium point, we mean the point in value where the disease level remains the same at all times. This means that the rate of change of any of the compartments in the disease dynamics is equal to zero for all times [14].

3.1 Disease Free Equilibrium Point

The disease free equilibrium point is a steady state solution whereby there is no disease. At this state, there is no malaria parasite on the humans and mosquitoes population on a dynamical system. To obtain equilibrium states, we equate the model equations (1) to (11) to zero. That is,

$$\frac{dP}{dt} = \frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dI_h}{dt} = \frac{dT_h}{dt} = \frac{dL}{dt} = \frac{dR_h}{dt} = \frac{dA_h}{dt} = \frac{dS_m}{dt} = \frac{dE_m}{dt} = \frac{dI_m}{dt} = 0 \tag{27}$$

In the absent of malaria, $P_h = E_h = I_h = E_v = I_v = 0$ and model equations (1) to (11) becomes

$$\gamma\omega_h + \delta(1-\eta)A - (\lambda(1-y) + \mu_h)P_h \tag{28}$$

$$(1-y)\omega_h + \eta A + \lambda(1-y)P - T_h S_h - (\mu_h + d(1-y_1))S_h = 0 \tag{29}$$

$$\omega_m - T_m S_m - \mu S_m = 0 \tag{30}$$

Solving the equations (28) to (30) for P_h, S_h and S_m, we have,

$$P_h = \frac{\gamma\omega_h}{\lambda(1-y) + \mu_h}, \quad S_h = \frac{(\Lambda(1-y) + (1-y)\mu_h)\omega_h}{(\mu_h + \lambda(1-y))(\mu_h + d(1-y_1))} \quad \text{and} \quad S_m = \frac{\omega_m}{\mu_m}.$$

Hence, the malaria free equilibrium point E_0 is,

$$E_0 = \left(\frac{\gamma\omega_h}{\lambda(1-y) + \mu_h}, \frac{(\Lambda(1-y) + (1-y)\mu_h)\omega_h}{(\mu_h + \lambda(1-y))(\mu_h + d(1-y_1))}, 0, 0, 0, 0, 0, 0\frac{\omega_m}{\mu_m}, 0, 0\right) \tag{31}$$

3.2 Basic Reproduction Number (R_0)

The basic reproduction number is one of the fundamental concepts in mathematical biology that determines the future of an epidemic. According to [15], the basic reproduction number R_0 is the expected number of secondary cases produced in a completely susceptible population, by a typical infective individual. It is one of the most useful threshold parameters, which characterize mathematical problems concerning infectious diseases. If R_0 is less than unity, this implies that, on average an infected individual produces less than one new infected individual during the infectious period and infection can be wiped out. Conversely if R_0 is greater than unity, then each infected individual produces, on average, more than one new infection, and the disease spreads in the population. For a single infected compartment, R_0 is the product of the infection rate and the mean duration of the infection. But for complicated models, this definition of R_0 is insufficient. We therefore compute the basic reproduction number R_0 of the model equations (1) to (11), using next generation matrix approach by ([16] ; [17]; [15]; [18]). That is,

$$\mathcal{R}_0 = \rho(FV^{-1}) \tag{32}$$

where,

- $\rho(A)$ is the spectral radius of matrix A (or the maximum modulus of the eigenvalues of A).
- F_i is the rate of appearance of new infections in compartment I,
- V_i^+ is the transfer of individuals into compartment I, and
- V_i^- is the transfer of individuals out of compartment I.

We have $F = \left[\frac{\partial \mathcal{F}_i(E_0)}{\partial x_j}\right]$ and $V = \left[\frac{\partial \mathcal{V}_i(E_0)}{\partial x_j}\right]$, with $1 \geq i, j \leq m$, where m represents the infected classes, E_0 is the disease-free equilibrium, and $\mathcal{V}_\rangle = \mathcal{V}_i^-(x) - \mathcal{V}_i^+(x)$.

From the model equations (1) to (11), we obtain $\mathcal{R}_{0h}$ and $\mathcal{R}_{0m}$ at the disease-free equilibrium E_0 by rewriting the newly infective classes of human and mosquito populations as:

$$\left\{\begin{array}{l} \dfrac{dE_h}{dt} = \dfrac{b\rho_h I_m S_h}{N_h} - (\alpha_h + \alpha_1 + \mu_h)E_h \\ \dfrac{dI_h}{dt} = \alpha_h E_h - (\phi + \kappa + \mu_h + \theta_h)I_h \\ \dfrac{dE_m}{dt} = \dfrac{b\rho_h I_h S_m}{N_m} - (\alpha_m + \mu_m)E_m \\ \dfrac{dI_m}{dt} = \alpha_m E_m - \mu_m I_m \end{array}\right\}. \tag{33}$$

From equation (33), we obtain

$$F_i = \begin{bmatrix} \frac{b\rho_h I_m S_h}{N_h} \\ 0 \\ \frac{b\rho_m I_h S_m}{N_m} \\ 0 \end{bmatrix} \tag{34}$$

Differentiating equation (34) partially with respect to E_h, I_h, E_m, I_m, that is, $\frac{\partial \mathcal{F}_i}{\partial E_h}$, $\frac{\partial \mathcal{F}_i}{\partial I_h}$ and $\frac{\partial \mathcal{F}_i}{\partial I_m}$ we obtained,

$$F_i = \begin{bmatrix} 0 & 0 & 0 & \frac{b\rho_h S_h}{N_h} \\ 0 & 0 & 0 & 0 \\ 0 & b\rho_m & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{35}$$

At the disease-free equilibrium point E_0, equation (35) becomes,

$$F_iE_0 = \begin{bmatrix} 0 & 0 & 0 & \frac{\omega b\rho_h(\lambda(1-y)+(1-y)\mu_h)}{\mu_h(\mu_h+\lambda(1-y))} \\ 0 & 0 & 0 & 0 \\ 0 & b\rho_m & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{36}$$

Also from equation (33), we obtain $\mathcal{V}_i$, that is,

$$\mathcal{V}_i = \begin{bmatrix} (\alpha_h+\alpha_1+\mu_h)E_h \\ (\phi+\kappa+\mu_h+\theta_h)I_h - \alpha_h E_h \\ (\alpha_m+\mu_m)E_m \\ \mu_m I_m - alpha_m E_m \end{bmatrix} \tag{37}$$

Differentiating equation (37) partially with respect to E_h, I_h, E_m, I_m, that is, $\frac{\partial \mathcal{V}_i}{\partial E_h}$, $\frac{\partial \mathcal{V}_i}{\partial I_h}$ and $\frac{\partial \mathcal{V}_i}{\partial I_m}$ we obtained,

$$\mathcal{V}_i = \begin{bmatrix} (\alpha_h+\alpha_1+\mu_h) & 0 & 0 & 0 \\ -\alpha_h & (\phi+\kappa+\mu_h+\theta_h) & 0 & 0 \\ 0 & 0 & (\alpha_m+\mu_m) & 0 \\ 0 & 0 & -\alpha_m & \mu_m \end{bmatrix} \tag{38}$$

Taking the inverse of (38), we have,

$$\mathcal{V}^{-1} = \begin{bmatrix} \frac{1}{(\alpha_h+\alpha_1+\mu_h)} & 0 & 0 & 0 \\ -\frac{\alpha_h}{(\alpha_h+\alpha_1+\mu_h)(\phi+\kappa+\mu_h+\theta_h)} & \frac{1}{(\phi+\kappa+\mu_h+\theta_h)} & 0 & 0 \\ 0 & 0 & \frac{1}{(\alpha_m+\mu_m)} & 0 \\ 0 & 0 & -\frac{\alpha_m}{(\alpha_m+\mu_m)\mu_m} & \frac{1}{\mu_m} \end{bmatrix} \tag{39}$$

Let

$$a_0 = \frac{1}{(\alpha_h+\alpha_1+\mu_h)}, \quad a_1 = -\frac{\alpha_h}{(\alpha_h+\alpha_1+\mu_h)(\phi+\kappa+\mu_h+\theta_h)},$$
$$a_2 = \frac{1}{(\phi+\kappa+\mu_h+\theta_h)}, \quad a_3 = \frac{1}{(\alpha_m+\mu_m)}, \quad a_4 = -\frac{\alpha_m}{(\alpha_m+\mu_m)\mu_m}, \quad a_5 = \frac{1}{\mu_m}$$

Then,

$$\mathcal{V}^{-1} = \begin{bmatrix} a_0 & 0 & 0 & 0 \\ a_1 & a_2 & 0 & 0 \\ 0 & 0 & a_3 & 0 \\ 0 & 0 & a_4 & a_5 \end{bmatrix} \tag{40}$$

Therefore, the product of equations (36) and (40) yields

$$F\mathcal{V}^{-1} = \begin{bmatrix} 0 & 0 & a_4\frac{\omega_h b\rho_h(\lambda(1-y)+(1-y)\mu_h)}{\mu_h(\mu_h+\lambda(1-y))} & a_5\frac{\omega_h b\rho_h(\lambda(1-y)+(1-y)\mu_h)}{\mu_h(\mu_h+\lambda(1-y))} \\ 0 & 0 & 0 & 0 \\ a_1 b\rho_m & a_2 b\rho_m & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{41}$$

Let

$$a_6 = a_4\frac{\omega_h b\rho_h(\lambda(1-y)+(1-y)\mu_h)}{\mu_h(\mu_h+\lambda(1-y))}, \quad a_7 = a_5\frac{\omega_h b\rho_h(\lambda(1-y)+(1-y)\mu_h)}{\mu_h(\mu_h+\lambda(1-y))},$$
$$a_8 = a_1 b\rho_m, \quad a_9 = a_2 b\rho_m$$

Then, equation (41) becomes,

$$F\mathcal{V}^{-1} = \begin{bmatrix} 0 & 0 & a_6 & a_7 \\ 0 & 0 & 0 & 0 \\ a_8 & a_9 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{42}$$

Then the characteristic equation of equation (43) is given by $\det(FV^{-1} - kI) = 0$, these imply that

$$\left|F\mathcal{V}^{-1}\right| = \begin{vmatrix} -k & 0 & a_6 & a_7 \\ 0 & -k & 0 & 0 \\ a_8 & a_9 & -k & 0 \\ 0 & 0 & 0 & -k \end{vmatrix} = 0 \tag{43}$$

Therefore, the characteristic equation of equation (43) is given as:

$$-k[-k(k^2)] - a_6k(-a_8k) + a_7k(0) = 0 \tag{44}$$

Simplifying further, we have

$$k^2(k^2 + a_6a_8) = 0 \tag{45}$$

This implies that,

$$k^2 = 0 \quad \text{or} \quad k^2 = -a_6a_8 \Rightarrow k = \pm\sqrt{a_6a_8} \tag{46}$$

The dominant eigenvalue is $k = \pm\sqrt{a_6a_8}$. Therefore, The basic reproductive number for human and mosquito populations are:

$$\mathcal{R}_{0h} = \sqrt{\frac{\omega_h b\rho_h(\lambda(1-y) + (1-y)\mu_h)\alpha_h}{\mu_h(\mu_h + \lambda(1-y))(\alpha_h + \alpha_1(1-y_2) + \mu_h)(\phi + \kappa + \mu_h + \theta_h)}} \tag{47}$$

$$\mathcal{R}_{0m} = \sqrt{\frac{b\rho_h\alpha_m}{(\alpha_m + \mu_m)\mu_m}} \tag{48}$$

Thus,

$$\mathcal{R}_0 = \sqrt{\frac{\omega_h b\rho_h(\lambda(1-y) + (1-y)\mu_h)\alpha_h b\rho_h\alpha_m}{\mu_h(\mu_h + \lambda(1-y))(\alpha_h + \alpha_1(1-y_2) + \mu_h)(\phi + \kappa + \mu_h + \theta_h)(\phi + \kappa + \mu_h + \theta_h)(\alpha_m + \mu_m)\mu_m}} \tag{49}$$

4 Local Stability Analysis of the Disease Free Equilibrium point

Theorem 4.1. *The disease free equilibrium of the system of the model equation (1) to (11) is locally asymptotically stable when $\mathcal{R}_0 < 1$ or unstable when $\mathcal{R}_0 > 1$.*

Proof.

Following the approach by [19], that is, the criteria for stability or instability of the disease free equilibrium of the model equations (1) to (11) shall be established by solving the characteristic equation of the Jacobian matrix of the model equations at the disease free equilibrium point E_0.

The Jacobian matrix of the model equations is given as:

$$J = \begin{pmatrix} u_0 & 0 & 0 & 0 & 0 & 0 & 0 & \delta(1-\eta) & 0 & 0 \\ 0 & & & & & & & & & \\ \lambda(1-y) & u_1 & 0 & 0 & 0 & 0 & 0 & \eta & 0 & 0 \\ -\frac{b\rho_h S_h}{N_h} & & & & & & & & & \\ 0 & T_h & u_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{b\rho_h S_h}{N_h} & & & & & & & & & \\ 0 & 0 & \alpha_h & u_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & & & & & & & & & \\ 0 & 0 & 0 & \phi & u_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & & & & & & & & & \\ 0 & 0 & 0 & \kappa & 0 & u_5 & 0 & 0 & 0 & 0 \\ 0 & & & & & & & & & \\ 0 & 0 & 0 & 0 & \sigma & \phi & u_6 & 0 & 0 & 0 \\ 0 & & & & & & & & & \\ 0 & d(1-y_1) & \alpha_1(1-y_2) & 0 & 0 & 0 & \beta & u_7 & 0 & 0 \\ 0 & & & & & & & & & \\ 0 & 0 & 0 & -\frac{b\rho_m S_m}{N_m} & 0 & 0 & 0 & 0 & u_8 & 0 \\ 0 & & & & & & & & & \\ 0 & 0 & 0 & \frac{b\rho_m S_m}{N_m} & 0 & 0 & 0 & 0 & T_m & u_9 \\ 0 & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m \\ u_{10} & & & & & & & & & \end{pmatrix} \tag{50}$$

where

$$u_0 = -(\mu_h + \lambda(1-y)), \quad u_1 = -(T_h + d(1-y_1+\mu_h)), \quad u_2 = -(\alpha_h + \alpha_1(1-y_2) + \mu_h),$$
$$u_3 = -(\phi + \kappa + \mu_h + \theta), \quad u_4 = -(\sigma + \mu_h)), \quad u_5 = -(\phi + \mu_h),$$
$$u_6 = -(\beta + \mu_h), \quad u_7 = -(\delta(1-eta) + \eta + \mu_h), \quad u_8 = -(T_m + \mu_m),$$
$$u_9 = -(\alpha_m + \mu_m), \quad a_{10} = -\mu_m$$

Evaluating the Jacobian matrix (48) at the disease free equilibrium point E_0, we have

$$J(E_0) = \begin{pmatrix} a_{10} & 0 & 0 & 0 & 0 & 0 & 0 & a_{11} & 0 & 0 \\ 0 & & & & & & & & & \\ 0 & a_{13} & 0 & 0 & 0 & 0 & 0 & 0 & a_{27} & 0 \\ a_{14} & & & & & & & & & \\ 0 & 0 & a_{15} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{16} & & & & & & & & & \\ 0 & 0 & 0 & a_{17} & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{29} & & & & & & & & & \\ 0 & 0 & 0 & 0 & a_{18} & 0 & 0 & 0 & 0 & 0 \\ a_{31} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & a_{19} & 0 & 0 & 0 & 0 \\ a_{32} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{20} & 0 & 0 & 0 \\ a_{36} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{30} & 0 & 0 \\ a_{37} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m & 0 \\ a_{33} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{26} \\ a_{34} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{38} & & & & & & & & & \end{pmatrix} \tag{51}$$

Where,

$$a_{10} = -(\mu_h + \lambda(1-y)), \quad a_{11} = \delta(1-\eta), \quad a_{12} = \lambda(1-y_1),$$
$$a_{13} = -(d(1-y_1+\mu_h)), \quad a_{14} = \frac{b\rho_h(\lambda(1-y)+(1-y))\mu_h}{(\mu_h+\lambda(1-y))}, \quad a_{15} = -(\alpha_h + \alpha_1(1-y_2) + \mu_h),$$
$$a_{16} = \frac{b\rho_h(\lambda(1-y)+(1-y))\mu_h}{(\mu_h+\lambda(1-y))}, \quad a_{17} = -(\phi+\kappa+\mu_h+\theta), \quad a_{18} = -(\sigma+\mu_h),$$
$$a_{19} = -(\phi+\mu_h), \quad a_{20} = (\beta+\mu_h), \quad a_{21} = d(1-y_1),$$
$$a_{22} = \alpha_1(1-y_2), \quad a_{23} = -(\delta(1-\eta)+\lambda_h), \quad a_{24} = -b\rho_m, \quad a_{25} = b\rho_m, \quad a_{26} = -(\alpha_m+\mu_m)$$

Following the approach by [20], that is, reduce row echelon form, which we applied on equation (50),

we obtain

$$J(E_0) = \begin{pmatrix} a_{10} & 0 & 0 & 0 & 0 & 0 & 0 & a_{11} & 0 & 0 \\ 0 & & & & & & & & & \\ 0 & a_{13} & 0 & 0 & 0 & 0 & 0 & 0 & a_{27} & 0 \\ a_{14} & & & & & & & & & \\ 0 & 0 & a_{15} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{16} & & & & & & & & & \\ 0 & 0 & 0 & a_{17} & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{29} & & & & & & & & & \\ 0 & 0 & 0 & 0 & a_{18} & 0 & 0 & 0 & 0 & 0 \\ a_{31} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & a_{19} & 0 & 0 & 0 & 0 \\ a_{32} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{20} & 0 & 0 & 0 \\ a_{36} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{30} & 0 & 0 \\ a_{37} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m & 0 \\ a_{33} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{26} \\ a_{34} & & & & & & & & & \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_{38} & & & & & & & & & \end{pmatrix} \tag{52}$$

Where,

$$a_{27} = \eta + \frac{\lambda\delta(1-y)(1-\eta)}{\lambda(1-y)+\mu_h} \quad a_{29} = \frac{\alpha_h b\rho_m(\lambda(1-y)+(1-y))\mu_h}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)},$$

$$a_{30} = -(\delta(1-\eta)+\eta+\mu_h) + \frac{d(1-y_1)}{(d(1-y_1+\mu_h))}(\eta + \frac{\lambda\delta(1-y)(1-\eta)}{\lambda(1-y)+\mu_h}),$$

$$a_{31} = \frac{\phi\alpha_h b\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)},$$

$$a_{32} = \frac{\kappa\alpha_h b\rho_m(\lambda(1-y)+(1-y))\mu_h}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)},$$

$$a_{33} = \frac{\alpha_h b^2{\rho^2}_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)},$$

$$a_{34} = \frac{\alpha_h b^2{\rho^2}_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)},$$

$$a_{36} = \frac{\phi\kappa\alpha_h b\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)},$$

$$a_{37} = \{(\frac{d(1-y_1)}{(d(1-y_1)+\mu_h)} + \frac{\phi\kappa\alpha_h b\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(d(1-y_1)+\lambda_h)(\phi+\mu_h)(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)} + \frac{\alpha_1(1-y_2)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)} \frac{b\rho(\lambda(1-y)+(1-\gamma)\mu_h)}{(\mu_h+\lambda(1-y))} + (\frac{1}{(d(1-y_1))((\phi+\kappa+\mu_h+\theta))}) (\frac{\beta\phi\kappa\alpha_h b\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\beta+mu_h)(\phi+\mu_h)(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)})\},$$

$$a_{38} = -\mu - \frac{\alpha_m\alpha_h b^2{\rho^2}_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_m+\mu_m)(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)}$$

The characteristic matrix equation of matrix (52) is given as:

$$\left\{\begin{array}{l} ((-(\mu_h+\lambda(1-y))-\lambda)(-(d(1-y_1+\mu_h)-\lambda)-\lambda))(-(\alpha_h+(\alpha_1(1-y_2)+\mu_h)+\mu_h)-\lambda)+ \\ (-(\phi+\kappa+\mu_h+\theta)-\lambda)(-(\sigma+\mu_h)-\lambda)(-(\phi+\mu_h)-\lambda)(-(\beta+\mu_h)-\lambda) \\ (-(\delta(1-\eta)+\eta+\mu_h)+\frac{d(1-y_1)}{(d(1-y_1+\mu_h))}(\eta+\frac{\lambda\delta(1-y)(1-\eta)}{\lambda(1-y)+\mu_h})-\lambda)(-\mu_m-\lambda) \\ (-(\alpha_m+\mu_m)-\lambda)(-(\mu+\frac{\alpha_m\alpha_h b^2{\rho^2}_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_m+\mu_m)(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)})-\lambda) \end{array}\right\} = 0. \tag{53}$$

Thus, the eigenvalues are:

$$\left\{\begin{array}{l} \lambda_1 = -(\mu_h + \lambda(1-y)) < 0, \\ \lambda_2 = -(d(1-y_1+\mu_h-\lambda)) < 0, \\ \lambda_3 = -(\alpha_h + (\alpha_1(1-y_2)+\mu_h)) < 0, \\ \lambda_4 = -(\phi+\kappa+\mu_h+\theta) < 0, \\ \lambda_5 = -(\sigma+\mu_h) < 0, \\ \lambda_6 = -(\phi+\mu_h) < 0, \\ \lambda_7 = -(\beta+\mu_h) < 0, \\ \lambda_8 = -(\delta(1-\eta)+\eta+\mu_h) + \dfrac{d(1-y_1)}{(d(1-y_1+\mu_h))}(\eta + \dfrac{\lambda\delta(1-y)(1-\eta)}{\lambda(1-y)+\mu_h}) < 0, \\ \lambda_9 = -\mu_m < 0 \\ \lambda_{10} = -(\alpha_m+\mu_m) < 0, \\ \lambda_{11} = -(\mu + \dfrac{\alpha_m\alpha_h b^2 \rho^2{}_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_m+\mu_m)(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)}) < 0 \end{array}\right\} \quad (54)$$

5 Discussions and Conclusion

From equation (52), all the eigenvalues are negative, meaning they lie strictly to the left of the imaginary axis, this indicates that the equilibrium point of system (1)–(11) is locally asymptotically stable. In practical terms, any small disturbance to the system will decay over time, and the model trajectories will return to equilibrium rather than diverge. A higher treatment rate reduces the duration of infectiousness and pushes the eigenvalues further negative, strengthening stability. If large, this reduces the effectiveness of interventions, but because the eigenvalues remain negative, the system still returns to equilibrium just more slowly. When recovery dominates over death, the infectious pool shrinks faster, which is reflected in negative eigenvalues ensuring convergence to equilibrium. Rate of progression to awareness from recovering humans increases the flow into the aware class, reducing susceptibility and supporting the decay of perturbations. Rate of progression to awareness from susceptible moves individuals away from the susceptible pool, decreasing the force of infection and reinforcing local stability. Rate of waning from awareness back to susceptible: This weakens stability by replenishing the susceptible pool, but as long as eigenvalues remain negative, its impact does not destabilize the system. Rate at which humans become protected strengthens resilience of the population by lowering effective susceptibility, which is consistent with stability. Rate at which treated humans recover complements untreated recovery by shortening the infectious period even more, which again supports stability. It is clear from equation (52) that all the eigenvalues are negative and less than zero. $\lambda_5 and \lambda_6$ in (52), implies that the disease will die of when the rates at which treated and untreated humans recovered is greater than death due malaria. Therefore the solution of model equation (1) to (11) is locally asymptotically stable. We have formulated and analysed a mathematical model that reduces malaria transmission dynamics and investigate the basic properties of the model. The Disease Free Equilibrium points and the basic reproduction number of the model were obtained. Our investigation of the basic properties of model equations shows that the model is mathematically correct. The Jacobian matrix stability technique was used to analyzed the stability of the disease free equilibrium. The analysis shows that the disease free equilibrium is locally asymptotically stable that is $(R_0 < 1)$.

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