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ASYMPTOTIC STABILITY OF MALARIA DYNAMICS WITH VIGILANT HUMAN COMPARTMENT

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Abstract: A continuous-time and discrete-age-structured compartmental model for malaria transmission in a two-interacting human and mosquito populations is formulated. The model incorporates a class of vigilant humans who adhere to the malaria vector control measures of the World Health Organization with a view to preventing the human-mosquito contacts. An epidemiological threshold called the basic reproduction number of the model is derived and a qualitative analysis of the model is carried out to investigate the asymptotic stability of the equilibria. A locally asymptotically stable disease-free equilibrium at the basic reproduction number less than unity is proved via the analysis of characteristic equation. Whereas, the existence of a locally asymptotically stable endemic equilibrium is established at the basic reproduction number greater than unity based on the use of center manifold theory of bifurcation. In addition, a sensitivity analysis is performed to examine the contributory effects of the model parameters on the transmission and spread of the malaria disease with respect to the basic reproduction number.

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1. Introduction

Malaria is one of the infectious diseases that have continued to be a subject of major concern to the public health. This is why 25th of April is set aside as the world's annual malaria day for a global alertness against the disease. The world malaria report [17] shows that, in 2013, an estimated 3.3 billion people worldwide were at risk of acquiring malaria with populations living in sub-Saharan Africa having the highest risk. Approximately 80% of malaria cases and 90% of deaths are estimated to occur in most countries of this sub-Saharan Africa (see, [16]).

The disease is caused by parasite of genus *Plasmodium* and is transmitted to human through the bites of an infectious female *anopheles* mosquito. The presence of the infection in human is characterized by paroxysms of chills, fever, headache, pain and vomiting; if left untreated, the infection can be life-threatening [10, 13].

The use of mathematics to explain the transmission and spread of malaria in a two-interacting populations has been extensively studied by researchers over the years. Notable among these studies are [1, 3, 6, 7, 9, 10]. Since humans may not have equal likelihood of getting infected, it is reasonable to incorporate age-structure in designing malaria model (see, for instance, [7] where human compartment is divided into two age groups, namely juveniles and adults). Further, in the absence of an effective malaria vaccination, intervention strategies known as vector control measures become inevitable in order to ensure malaria-free population. These measures include the use of; insecticide-treated bed-nets (ITNs), indoor residual spraying (IRS) and mosquito-repellent lotions to prevent human-mosquito contacts as well as prophylaxis (the use of drug to prevent malaria) and integrated vector management as prescribed by the World Health Organisation (see, [15-17]).

In this study, we design and analyse a mathematical compartmental model that considers a discrete-age-structure without dividing human compartment into age groups and incorporates a new class of human (vigilant human compartment) that adheres to the vector control measures. The rest of this study is organized as follows. Section 2 deals with the formulation of the model and shows its basic qualitative properties. In Section 3, asymptotic stability cum sensitivity analyses are performed. Finally, concluding remarks are provided in Section 4.

2. Model Formulation

The formulation of malaria model requires the interaction between the discrete-age-structured humans and adult female *anopheles* mosquitoes populations. The total human population size at continuous-time t and discrete-age a_i, denoted by $N_h(t, a_i)$, where $i = 0, 1, 2, ..., L$ and a_L is the maximum age of humans in the population, is subdivided into four compartments namely susceptible humans $S_h(t, a_i)$ (those who are not currently harbouring the disease but are liable to be infected), exposed humans $E_h(t, a_i)$ (those who are infected with the malaria parasites but are incapable of transmitting the disease), infectious humans $I_h(t, a_i)$ (those already infected and are able to transmit the disease), and vigilant humans $V_h(t, a_i)$ (those who are wary of malaria and guide against it by adhering to the vector control measures). Hence, we have

$$N_h(t, a_i) = S_h(t, a_i) + E_h(t, a_i) + I_h(t, a_i) + V_h(t, a_i).$$

The total mosquito population at time t, denoted by $N_m(t)$, is subdivided into susceptible mosquitoes $S_m(t)$, exposed mosquitoes $E_m(t)$ and infectious mosquitoes $I_m(t)$, so that

$$N_m(t) = S_m(t) + E_m(t) + I_m(t).$$

The dynamics of the model is formulated under the following assumptions:

- That only humans are vigilant
- That humans are recruited into either susceptible or vigilant compartment
- That exposed humans progress to either become infectious or vigilant if they are quickly treated (i.e. if under prophylaxis)
- That all infectious humans that recover become vigilant only
- That adherence to vector control measures by the vigilant humans is strict and does not result into re-infection
- That mosquitoes are recruited through susceptible class only
- That the infected susceptible mosquitoes are the exposed mosquitoes who are not yet infectious.
- That the exposed mosquitoes progress to become infectious only
- That the infectious mosquitoes remain infectious until death.

Therefore, we describe the transmission dynamics of malaria by the following system of ordinary differential equations:

$$\dot{S}_h(t,a_i) = \sum_{i=0}^{L}(1-\tau)\lambda_h(a_i)N_h(t,a_i) - \frac{b\beta_h(a_i)S_h(t,a_i)I_m(t)}{N_h(t,a_i)} - \mu_h(a_i)S_h(t,a_i) \quad (2.1a)$$

$$\dot{E}_h(t,a_i) = \sum_{i=0}^{L}\frac{b\beta_h(a_i)S_h(t,a_i)I_m(t)}{N_h(t,a_i)} - (\alpha_h(a_i)+\mu_h(a_i))E_h(t,a_i) \quad (2.1b)$$

$$\dot{I}_h(t,a_i) = \sum_{i=0}^{L}(1-\theta)\alpha_h(a_i)E_h(t,a_i) - (\gamma(a_i)+\mu_h(a_i))I_h(t,a_i) \quad (2.1c)$$

$$\dot{V}_h(t,a_i) = \sum_{i=0}^{L}\tau\lambda_h(a_i)N_h(t,a_i) + \theta\alpha_h(a_i)E_h(t,a_i) + \gamma(a_i)I_h(t,a_i) - \mu_h(a_i)V_h(t,a_i) \quad (2.1d)$$

$$\dot{S}_m = \lambda_m N_m - \frac{b\beta_m S_m(t)I_h(t,a_i)}{N_h(t,a_i)} - \mu_m S_m(t) \quad (2.1e)$$

$$\dot{E}_m = \frac{b\beta_m S_m(t)I_h(t,a_i)}{N_h(t,a_i)} - (\alpha_m+\mu_m)E_m(t) \quad (2.1f)$$

$$\dot{I}_m = \alpha_m E_m(t) - \mu_m I_m(t), \quad (2.1g)$$

where a dot represents the differentiation with respect to time. The accompanying initial conditions of system (2.1) are given by:

$$\begin{cases} S_h(0,a_i) = S_{0h}(a_i),\ E_h(0,a_i) = E_{0h}(a_i), I_h(0,a_i) = I_{0h}(a_i), \\ V_h(0,a_i) = V_{0h}(a_i), S_m(0) = S_{0m},\ E_m(0) = E_{0m},\ I_m(0) = I_{0m}. \end{cases} \quad (2.2)$$

The parameters of the model (2.1) are described as follows:

Parameter	Description
$\lambda_h(a_i)$	Per capita recruitment rate of humans at discrete-age (a_i)
τ	Proportion of human population that is born vigilant
λ_m	Per capita recruitment rate of mosquitoes
b	Biting rate of the mosquito
$\beta_h(a_i)$	Probability that a bite by an infectious mosquito results into infection in human
β_m	Probability that a bite results in transmission of parasite to a susceptible mosquito
$\mu_h(a_i)$	Per capita death rate of human at discrete-age (a_i)
μ_m	Per capita death rate of mosquito
$\alpha_h(a_i)$	Per capita rate of progression of exposed humans to infectious humans at discrete-age (a_i)
θ	Proportion of exposed humans that becomes vigilant
α_m	Per capita rate of progression of mosquitoes from the exposed state to the infectious state
$\gamma(a_i)$	Per capita recovery rate of infectious humans to vigilant humans at discrete-age (a_i)

Table 1: Description of the model parameters.

To conveniently carry out the analysis of the formulated model (2.1), we rescale the state variables by dividing the number of the individuals in the sub-populations by their respective total number of populations. This process is achieved by making the following change of variables:

$$\bar{S}_h(t,a_i)=\frac{S_h(t,a_i)}{N_h(t,a_i)},\quad \bar{E}_h(t,a_i)=\frac{E_h(t,a_i)}{N_h(t,a_i)},\quad \bar{I}_h(t,a_i)=\frac{I_h(t,a_i)}{N_h(t,a_i)},$$

$$\bar{V}_h(t,a_i)=\frac{V_h(t,a_i)}{N_h(t,a_i)},\bar{S}_m(t)=\frac{S_m(t)}{N_m(t)},\bar{E}_m(t)=\frac{E_m(t)}{N_m(t)},\bar{I}_m(t)=\frac{I_m(t)}{N_m(t)}.$$

So that,
$\bar{S}_h(t,a_i)+\bar{E}_h(t,a_i)+\bar{I}_h(t,a_i)+\bar{V}_h(t,a_i)=1$ and $\bar{S}_m(t)+\bar{E}_m(t)+\bar{I}_m(t)=1$. Noting that this rescaling follows from the assumption that $\lambda_h(a_i)=\mu_h(a_i)$ and $\lambda_m=\mu_m$. Thus, after dropping of bars, $(\bar{})$, and if we let $\sigma=\frac{N_m}{N_h}$, model

(2.1) leads to the following system of equations:

$$\begin{cases} \frac{dS_h(t,a_i)}{dt} = (1-\tau)\lambda_h(a_i) - \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h(t,a_i)I_m - \mu_h(a_i)S_h(t,a_i) \\ \frac{dE_h(t,a_i)}{dt} = \sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h(t,a_i)I_m - (\alpha_h(a_i)+\mu_h(a_i))E_h(t,a_i) \\ \frac{dI_h(t,a_i)}{dt} = \sum_{i=0}^{L}(1-\theta)\alpha_h(a_i)E_h(t,a_i) - (\gamma(a_i)+\mu_h(a_i)I_h(t,a_i) \\ \frac{dV_h(t,a_i)}{dt} = \tau\lambda_h(a_i) + \theta\alpha_h(a_i)E_h(t,a_i) + \gamma(a_i)I_h(t,a_i) - \mu_h(a_i)V_h(t,a_i) \\ \frac{dS_m}{dt} = \lambda_m - b\beta_m S_m(t)I_h(t,a_i) - \mu_m S_m(t) \\ \frac{dE_m}{dt} = b\beta_m S_m(t)I_h(t,a_i) - (\alpha_m+\mu_m)E_m(t) \\ \frac{dI_m}{dt} = \alpha_m E_m(t) - \mu_m I_m(t) \end{cases} \tag{2.3}$$

2.1. Basic Qualitative Properties

Since model (2.3) represents interaction between human and mosquito populations, it makes sense to state that all the parameters involved are non-negative. It is also pertinent to show that all the state variables of the model are non-negative for all times.

To begin with, summing up the first four and the last three of the malaria model (2.3), we have

$$\frac{dN_h}{dt} = \mu_h(a_i)\,(1-N_h)$$

and

$$\frac{dN_m}{dt} = \mu_m\,(1-N_m)\,,$$

so that,
$N_h = 1 + (N_h(0)-1)\exp(-\mu_h(a_i)t)$ and $N_m = 1 + (N_m(0)-1)\exp(-\mu_m t)$ respectively. So the malaria model (2.3) will be analysed in a positively invariant region $\mathfrak{D} = \mathfrak{D}_h \times \mathfrak{D}_m \subset \mathbb{R}^4_+ \times \mathbb{R}^3_+$ with

$$\mathfrak{D}_h = \left\{(S_h(t,a_i), E_h(t,a_i), I_h(t,a_i), V_h(t,a_i)) \in \mathbb{R}^4_+ : S_h + E_h + I_h + V_h = 1\right\}$$

and

$$\mathfrak{D}_m = \left\{(S_m, E_m, I_m) \in \mathbb{R}^3_+ : S_m + E_m + I_m = 1\right\}\ .$$

2.1.1. Positivity of Solutions

Here, we show that the state variables of the model (2.3) are positive for all times.

Theorem 2.1. *The solutions,* $S_h(t,a_i), E_h(t,a_i), I_h(t,a_i), V_h(t,a_i), S_m(t), E_m(t), I_m(t)$*, of the malaria model* (2.3) *with non-negative initial data* (2.2) *in* $\mathfrak{D}$ *remain non-negative for all times* $t > 0$*.*

Proof. Consider the first equation of system (2.3) for a given non-negative initial condition $S_{0h}(a_i)$ in $\mathfrak{D}$ and suppose that there exist $\beta > 0$ and $\mu > 0$ such that $\beta_h(a_i) < \beta$ and $\mu_h(a_i) < \mu$, then

$$\begin{aligned}\frac{dS_h(t,a_i)}{dt} &= (1-\tau)\lambda_h(a_i) - \sum_{i=0}^{L}\left[b\beta_h(a_i)\sigma S_h(t,a_i)I_m - \mu_h(a_i)S_h(t,a_i)\right] \\ &\geq -\left(b\beta\sigma I_m + \mu\right)\sum_{i=0}^{L} S_h(t,a_i),\end{aligned}$$

so that,

$$\frac{d}{dt}\sum_{i=0}^{L} S_h(t,a_i) \geq -(L+1)\left(b\beta\sigma I_m + \mu\right)\sum_{i=0}^{L} S_h(t,a_i)$$

which on integration becomes

$$\sum_{i=0}^{L} S_h(t,a_i) \geq \sum_{i=0}^{L} S_{0h}(a_i)\exp\left[-(L+1)\left(\int_0^t b\beta\sigma I_h(\zeta)d\zeta + \mu t\right)\right] > 0, \quad \forall\, t > 0.$$

Hence, it is necessary and sufficient that $S_h(t,a_i) \geq 0, \forall\ a_i$ and $t > 0$. By similar argument, it can be shown that $E_h(t,a_i) \geq 0$, $I_h(t,a_i) \geq 0$, $V_h(t,a_i) \geq 0$, $S_m(t) \geq 0$, $E_m(t) \geq 0$ and $I_m(t) \geq 0$ for all times $t > 0$. This completes the proof. □

3. Asymptotic Stability and Sensitivity Analysis

At this juncture, we analyse the behaviour of the dynamics governed by model (2.3) as its solutions approach the disease-free and endemic equilibria. The contributory effects of the model parameters with respect to the basic reproduction number of the model is also performed through sensitivity analysis.

3.1. Disease-Free Equilibrium

The steady-state solution of model (2.3) when the diseased classes; $E_h(t, a_i) = I_h(t, a_i) = 0$ and $E_m(t) = I_m(t) = 0$ is referred to as the disease-free equilibrium point denoted by $\mathcal{E}_0$. This is obtained as

$$\mathcal{E}_0 = ((1-\tau),\ 0,\ 0,\ \tau,\ 1,\ 0,\ 0)\,. \tag{3.1}$$

To examine the local stability of $\mathcal{E}_0$ given by (3.1), it is important to first obtain the basic reproduction number, $\mathcal{R}_0$, defined as the average number of secondary infections caused by a typical infectious individual in a completely susceptible population. The next generation matrix method of computing $\mathcal{R}_0$ described by van den Driessche and Watmough [14] is explored on the model (2.3) as follows:
Let $x = (E_h(t, a_i), I_h(t, a_i), E_m(t), I_m(t), S_h(t, a_i), V_h(t, a_i), S_m(t),)^T$, so that model (2.3) can be written as $\dfrac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x)$. Considering only the diseased compartments, the rate of appearance of new infections and transition rate are given, respectively, by

$$\mathcal{F}_i = \begin{pmatrix} \displaystyle\sum_{i=0}^{L} b\beta_h(a_i)\sigma S_h(t, a_i) I_m(t) \\ 0 \\ b\beta_m S_m I_h(t, a_i) \\ 0 \end{pmatrix}$$

and

$$\mathcal{V}_i = \begin{pmatrix} \displaystyle\sum_{i=0}^{L} (\alpha_h(a_i) + \mu_h(a_i)) E_h(t, a_i) \\ \displaystyle\sum_{i=0}^{L} (\gamma(a_i) + \mu_h(a_i)) I_h(t, a_i) - (1-\theta)\alpha_h(a_i) E_h(t, a_i) \\ (\alpha_m + \mu_m) E_m \\ \mu_m I_m - \alpha_m E_m \end{pmatrix}.$$

Then the non-negative matrix $\mathbf{F}$ of the new infection terms and non-singular matrix $\mathbf{V}$ of transition terms are, respectively, obtained as

$$\mathbf{F} = \begin{pmatrix} 0 & 0 & 0 & \sum_{i=0}^{L} b\beta_h(a_i)\sigma(1-\tau) \\ 0 & 0 & 0 & 0 \\ 0 & b\beta_m & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$\mathbf{V} = \begin{pmatrix} \sum_{i=0}^{L}(\alpha_h(a_i)+\mu_h(a_i)) & 0 & 0 & 0 \\ -\sum_{i=0}^{L}(1-\theta)\alpha_h(a_i) & \sum_{i=0}^{L}(\gamma(a_i)+\mu_h(a_i)) & 0 & 0 \\ 0 & 0 & (\alpha_m+\mu_m) & 0 \\ 0 & 0 & -\alpha_m & \mu_m \end{pmatrix}.$$

Consequently, we obtain the spectral radius of the matrix $\mathbf{FV}^{-1}$, known as the the basic reproduction number of the malaria model (2.3) as

$$\mathcal{R}_0(a_i) = \sqrt{\sum_{i=0}^{L} \frac{b^2\beta_h(a_i)\sigma\alpha_h(a_i)\beta_m\alpha_m(1-\theta)(1-\tau)}{(\alpha_h(a_i)+\mu_h(a_i))(\gamma(a_i)+\mu_h(a_i))(\alpha_m+\mu_m)\mu_m}}. \tag{3.2}$$

We next state and prove the local stability of $\mathcal{E}_0$ with respect to (3.2).

Theorem 3.1. *The disease-free equilibrium, $\mathcal{E}_0$, of the system* (2.3) *is locally asymptotically stable if $\mathcal{R}_0(a_i) < 1$ and unstable if $\mathcal{R}_0(a_i) > 1$.*

Proof. The Jacobian of the malaria model (2.3) evaluated at the disease-free equilibrium point, $\mathcal{E}_0$, is obtained as

$$J(\mathcal{E}_0) = \begin{pmatrix} J_{11} & 0 & 0 & 0 & 0 & 0 & J_{17} \\ 0 & J_{22} & 0 & 0 & 0 & 0 & J_{27} \\ 0 & J_{32} & J_{33} & 0 & 0 & 0 & 0 \\ 0 & J_{42} & J_{43} & J_{44} & 0 & 0 & 0 \\ 0 & 0 & J_{53} & 0 & J_{55} & 0 & 0 \\ 0 & 0 & J_{63} & 0 & 0 & J_{66} & 0 \\ 0 & 0 & 0 & 0 & 0 & J_{76} & J_{77} \end{pmatrix}, \tag{3.3}$$

where

$$J_{11} = -\mu_h(a_i), \qquad J_{17} = -b\beta_h(a_i)\sigma(1-\tau), \quad J_{22} = -(\alpha_h(a_i) + \mu_h(a_i)),$$

$$J_{27} = b\beta_h(a_i)\sigma(1-\tau), \quad J_{32} = (1-\theta)\alpha_h(a_i), \qquad J_{33} = -(\gamma(a_i) + \mu_h(a_i)),$$

$$J_{42} = \theta\alpha_h(a_i), \qquad J_{43} = \gamma(a_i), \qquad J_{44} = -\mu_h(a_i),$$

$$J_{53} = -b\beta_m, \qquad J_{55} = -\mu_m, \qquad J_{63} = b\beta_m,$$

$$J_{66} = -(\alpha_m + \mu_m), \qquad J_{76} = \alpha_m, \qquad J_{77} = -\mu_m.$$

A necessary and sufficient condition for local asymptotic stability is for the real part of the eigenvalue to be in the negative half plane [2]. Thus, we need to show that $J(\mathcal{E}_0)$ given by (3.3) has all its eigenvalues with negative real part. To this purpose, it is obvious from (3.3) that $-\mu_h(a_i)$ (twice) and $-\mu_m$ are the three of the seven eigenvalues of $J(\mathcal{E}_0)$ since the first, fourth and the fifth columns contain only the diagonal terms. Hence, the other four eigenvalues can be obtained from the sub-matrix, $J^*(\mathcal{E}_0)$, given by

$$J^*(\mathcal{E}_0) = \begin{pmatrix} J^*_{11} & 0 & 0 & J^*_{14} \\ J^*_{21} & J^*_{22} & 0 & 0 \\ 0 & J^*_{32} & J^*_{33} & 0 \\ 0 & 0 & J^*_{43} & J^*_{44} \end{pmatrix}.$$

where $J^*_{11} = J_{33}$, $J^*_{14} = J_{27}$, $J^*_{21} = J_{32}$, $J^*_{22} = J_{33}$, $J^*_{32} = J_{63}$, $J^*_{33} = J_{66}$, $J^*_{43} = J_{76}$ and $J^*_{44} = J_{77}$. In what follows, the characteristic equation of $J^*(\mathcal{E}_0)$ is of the form

$$(\lambda + Q_1)(\lambda + Q_2)(\lambda + Q_3)(\lambda + Q_4) - Q_5 = 0, \tag{3.4}$$

where $Q_1 = \alpha_h(a_i) + \mu_h(a_i)$, $Q_2 = \gamma(a_i) + \mu_h(a_i)$, $Q_3 = \alpha_m + \mu_m$, $Q_4 = \mu_m$ and $Q_5 = \sum_{i=0}^{L} b^2\beta_h(a_i)\sigma\alpha_h(a_i)(1-\theta)(1-\tau)\beta_m\alpha_m$. Further expansion of (3.4) gives

$$C_4\lambda^4 + C_3\lambda^3 + C_2\lambda^2 + C_1\lambda + C_0 = 0, \tag{3.5}$$

where

$$\begin{cases} C_4 = 1 \\ C_3 = Q_1 + Q_2 + Q_3 + Q_4 \\ C_2 = (Q_1 + Q_2)(Q_3 + Q_4) + Q_1Q_2 + Q_3Q_4 \\ C_1 = (Q_1 + Q_2)Q_3Q_4 + (Q_3 + Q_4)Q_1Q_2 \\ C_0 = Q_1Q_2Q_3Q_4 - Q_5. \end{cases} \tag{3.6}$$

It is easy to see that C_0 can be written in terms of $\mathcal{R}_0(a_i)$ as

$$C_0 = Q_1Q_2Q_3Q_4(1 - \mathcal{R}_0^2). \tag{3.7}$$

If in (3.7) $\mathcal{R}_0(a_i) < 1$, then $C_0 > 0$. Since the coefficients $C_i,\ i = 1, 2, 3, 4$ and the Hurwitz matrices of the polynomial (3.5) are positive, using Routh-Hurwitz criterion (see,[8]), all the eigenvalues of (3.5) have negative real parts. Therefore, the disease-free equilibrium, $\mathcal{E}_0$, is stable. Otherwise, whenever $\mathcal{R}_0(a_i) > 1$ then $C_0 < 0$. By Descartes' rule of signs [12], there exists one eigenvalue with positive real part. Hence, $\mathcal{E}_0$ is unstable for $\mathcal{R}_0(a_i) > 1$. □

The epidemiological implication of the above result is that the malaria disease governed by model (3) can be eliminated from the population whenever an influx by infectious individuals is small such that $\mathcal{R}_0(a_i) < 1$.

3.2. Endemic Equilibrium

The steady-state solution of model (2.3) when all the state variables are positive is referred to as the endemic equilibrium point denoted and given by

$$\mathcal{E}_e = (S_h^*(a_i), E_h^*(a_i), I_h^*(a_i), V_h^*(a_i), S_m^*, E_m^*, I_m^*)\,. \tag{3.8}$$

It is burdensome to obtain the explicit form of the endemic equilibrium point of the model (2.3). However, the existence and local stability of $\mathcal{E}_e$ shall be explored using a center manifold theory of bifurcation analysis described in Castillo-Chavez and Song [4]. To this purpose, let the malaria model (2.3) be written in the vector form

$$\frac{dX}{dt} = F(X),$$

where
$X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7)^T$ and $F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)^T$ with
$S_h(t, a_i) = x_1, E_h(t, a_i) = x_2, I_h(t, a_i) = x_3, V_h(t, a_i) = x_4, S_m(t) = x_5,$

$E_m(t) = x_6$, and $I_m(t) = x_7$. Then model (2.3) becomes

$$\begin{cases} \frac{dx_1}{dt} = (1-\tau)\lambda_h(a_i) - b\beta_h(a_i)\sigma x_1 x_7 - \mu_h(a_i)x_1 := f_1 \\ \frac{dx_2}{dt} = b\beta_h(a_i)\sigma x_1 x_7 - (\alpha_h(a_i) + \mu_h(a_i))x_2 := f_2 \\ \frac{dx_3}{dt} = (1-\theta)\alpha_h(a_i)x_2 - (\gamma(a_i) + \mu_h(a_i))x_3 := f_3 \\ \frac{dx_4}{dt} = \tau\lambda_h(a_i) + \theta\alpha_h(a_i)x_2 + \gamma(a_i)x_3 - \mu_h(a_i)x_4 := f_4 \\ \frac{dx_5}{dt} = \lambda_m - b\beta_m x_5 x_3 - \mu_m x_5 := f_5 \\ \frac{dx_6}{dt} = b\beta_m x_5 x_3 - (\alpha_m + \mu_m)x_6 := f_6 \\ \frac{dx_7}{dt} = \alpha_m x_6 - \mu_m x_7 := f_7 \end{cases} \tag{3.9}$$

At $\mathcal{R}_0(a_i) = 1$ in (3.2), the bifurcation parameter $\beta_h(a_i)$ can be obtained as

$$\beta_h^*(a_i) = \frac{\sum_{i=0}^{L}(\alpha_h(a_i) + \mu_h(a_i))(\gamma(a_i) + \mu_h(a_i))(\alpha_m + \mu_m)\mu_m}{b^2\sigma\alpha_h(a_i)\beta_m\alpha_m(1-\theta)(1-\tau)}. \tag{3.10}$$

The linearized matrix of the system (3.9) around the disease-free equilibrium $\mathcal{E}_0$ and evaluated at $\beta_h^*(a_i)$ is given by

$$J(\mathcal{E}_0, \beta_h^*(a_i)) = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & -A_1 \\ 0 & -A_2 & 0 & 0 & 0 & 0 & A_1 \\ 0 & A_3 & -A_4 & 0 & 0 & 0 & 0 \\ 0 & A_5 & \gamma & -\mu_h(a_i) & 0 & 0 & 0 \\ 0 & 0 & -b\beta_m & 0 & -\mu_m & 0 & 0 \\ 0 & 0 & b\beta_m & 0 & 0 & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & \alpha_m & -\mu_m \end{pmatrix}, \tag{3.11}$$

where $A_1 = b\beta_h^*(a_i)\sigma(1-\tau)$, $A_2 = (\alpha_h(a_i) + \mu_h(a_i))$, $A_3 = (1-\theta)\alpha_h(a_i)$, $A_4 = (\gamma(a_i) + \mu_h(a_i))$, $A_5 = \theta\alpha_h(a_i)$ and $A_6 = (\alpha_m + \mu_m)$. The eigenvalues of $J(\mathcal{E}_0, \beta_h^*(a_i))$ are the roots of the characteristic equation given by

$$(\lambda + \mu_h(a_i))^2(\lambda + \mu_m)\mathfrak{M}(\lambda) = 0 = 0, \tag{3.12}$$

where $\mathfrak{M}(\lambda)$ is a polynomial of degree four whose roots are all negative except one zero eigenvalue. The left eigenvector, $\mathbf{v} = (v_1, v_2, ..., v_7)$, corresponding to

the simple zero eigenvalue of (3.11) is obtained from $\mathbf{v}J(\mathcal{E}_0, \beta_h^*(a_i)) = 0$ as

$$\begin{cases} v_1 = 0, \quad v_2 = \dfrac{b\beta_m\alpha_m\alpha_h(a_i)(1-\theta)}{(\alpha_h(a_i)+\mu_h(a_i))}, \\ v_3 = b\beta_m\alpha_m, \quad v_4 = 0, v_5 = 0, \\ v_6 = \alpha_m(\gamma(a_i)+\mu_h(a_i)) \\ v_7 = (\gamma(a_i)+\mu_h(a_i))(\alpha_m+\mu_m) \end{cases} \tag{3.13}$$

Further, the right eigenvector, $\mathbf{w} = (w_1, w_2, ..., w_7)^T$, associated with this simple zero eigenvalue can be obtained from $J(\mathcal{E}_0, \beta_h^*(a_i))\mathbf{w} = 0$. As a result, we have

$$\begin{cases} w_1 = -\displaystyle\sum_{i=0}^{L} \frac{(\alpha_h(a_i)+\mu_h(a_i))(\gamma(a_i)+\mu_h(a_i))(\alpha_m+\mu_m)\mu_m w_7}{b\beta_m\alpha_m(1-\theta)\alpha_h(a_i)\mu_h(a_i)} \\ w_2 = \dfrac{(\gamma(a_i)+\mu_h(a_i))(\alpha_m+\mu_m)\mu_m w_7}{b\beta_m\alpha_m(1-\theta)\alpha_h(a_i)} \\ w_3 = \dfrac{(\alpha_m+\mu_m)\mu_m w_7}{b\beta_m\alpha_m} \\ w_4 = \dfrac{(\alpha_m+\mu_m)(\theta\mu_h(a_i)+\gamma(a_i))\mu_m w_7}{b\beta_m\alpha_m(1-\theta)\mu_h(a_i)} \\ w_5 = -\dfrac{(\alpha_m+\mu_m)\mu_m w_7}{\alpha_m} \\ w_6 = \dfrac{\mu_m w_7}{\alpha_m} \\ w_7 = \dfrac{\alpha_h(a_i)+\mu_h(a_i)}{B} \end{cases}, \tag{3.14}$$

where

$$\begin{aligned} B = \quad & \sum_{i=0}^{L} \mu_m(\gamma(a_i)+\mu_h(a_i))(\alpha_h(a_i)+\mu_h(a_i)+\alpha_m+\mu_m) \\ & +(\alpha_h(a_i)+\mu_h(a_i))(\alpha_m+\mu_m)(\mu_m+\gamma(a_i)+\mu_h(a_i)). \end{aligned}$$

It should be noted that the components of $\mathbf{w}$ and $\mathbf{v}$ are obtained so that $\mathbf{v}.\mathbf{w} = 1$

as required in [4]. All the second-order partial derivatives of $f_i, i = 1, 2..., 7$, from the system (3.9) are zero at point $(\mathcal{E}_0, \beta_h^*(a_i))$ except the following

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_7} = \frac{\partial^2 f_1}{\partial x_7 \partial x_1} = -b\beta_h^*(a_i)\sigma, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_7} = \frac{\partial^2 f_2}{\partial x_7 \partial x_1} = b\beta_h^*(a_i)\sigma,$$

$$\frac{\partial^2 f_5}{\partial x_3 \partial x_5} = \frac{\partial^2 f_5}{\partial x_5 \partial x_3} = -b\beta_m, \quad \frac{\partial^2 f_6}{\partial x_3 \partial x_5} = \frac{\partial^2 f_6}{\partial x_5 \partial x_3} = b\beta_m$$

with

$$\frac{\partial^2 f_1}{\partial x_7 \partial \beta_h(a_i)} = -b\sigma(1-\tau), \quad \frac{\partial^2 f_2}{\partial x_7 \partial \beta_h(a_i)} = b\sigma(1-\tau).$$

The direction of the bifurcation at $\mathcal{R}_0(a_i) = 1$ is determined by the signs of the bifurcation coefficients **a** and **b**, obtained from the above partial derivatives, given, respectively, by

$$\begin{aligned} \mathbf{a} &= \sum_{k,i,j=1}^{7} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_{02}, \beta_h^*) \\ &= -\frac{2\mu_m w_7^2 (\gamma(a_i) + \mu_h(a_i))(\alpha_m + \mu_m)^2}{\alpha_m} \\ &\quad \times \left[\sum_{i=0}^{L} \frac{(\gamma(a_i) + \mu_h(a_i))(\alpha_h(a_i) + \mu_h(a_i))\mu_m}{b\mu_h(a_i)\alpha_h(a_i)\beta_m(1-\theta)(1-\tau)} + 1 \right] \end{aligned} \tag{3.15}$$

and

$$\begin{aligned} \mathbf{b} &= \sum_{k,i=1}^{7} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_h(a_i)}(E_{02}, \beta_h^*(a_i)) \\ &= \frac{b^2 \sigma \beta_m \alpha_m \alpha_h(a_i)(1-\theta)(1-\tau) w_7}{(\alpha_h(a_i) + \mu_h(a_i))}. \end{aligned} \tag{3.16}$$

Since $a < 0$ and $b > 0$, it follows from Theorem 4.1(iv) (see, [4]) that the malaria model (2.3) exhibits a supercritical (forward) bifurcation and the endemic equilibrium $\mathcal{E}_e$ is locally asymptotically stable. This result is theorized hereunder:

Theorem 3.2. *The malaria model governed by* (2.3) *exhibits a forward bifurcation at the threshold* $\mathcal{R}_0(a_i) = 1$ (*or, equivalently, there exists an endemic equilibrium,* $\mathcal{E}_e$, *which is locally asymptotically stable whenever* $\mathcal{R}_0(a_i) > 1$ *but near* $\mathcal{R}_0(a_i) = 1$).

The implication of the above result is that a small inflow of infectious individuals into a completely susceptible population will lead to the persistence of the disease in the community whenever $\mathcal{R}_0(a_i) > 1$. It should be noted that the summation over all the discrete ages will result to a net basic reproduction number about the maximum age a; that is, $\mathcal{R}_0(a) = \sum_{i=0}^{L} \mathcal{R}_0(a_i) > 1$.

3.3. Sensitivity Analysis

Following the idea in [5, 11], we perform a sensitivity analysis of the model (2.3) in order to determine the contributory effects of the model parameters on the transmission and spread of the malaria disease.

Definition 3.1. The normalized forward-sensitivity index of a variable, v, that depends differentiably on a parameter, p, is defined as:

$$\Upsilon_p^v = \frac{\partial v}{\partial p} \times \frac{p}{v}. \tag{3.17}$$

In particular, sensitivity indices of the basic reproduction number, $\mathcal{R}_0(a_i)$, with respect to the model parameters are computed. For example, using (3.17), we have

$$\begin{aligned}
\Upsilon_b^{\mathcal{R}_0(a_i)} &= \frac{\partial \mathcal{R}_0(a_i)}{\partial b} \times \frac{b}{\mathcal{R}_0(a_i)} = 1, \\
\Upsilon_\tau^{\mathcal{R}_0(a_i)} &= \frac{\partial \mathcal{R}_0(a_i)}{\partial \tau} \times \frac{\tau}{\mathcal{R}_0(a_i)} = -\frac{1}{2}\left(\frac{\tau}{1-\tau}\right), \\
\Upsilon_{\mu_m}^{\mathcal{R}_0(a_i)} &= \frac{\partial \mathcal{R}_0(a_i)}{\partial \mu_m} \times \frac{\mu_m}{\mathcal{R}_0(a_i)} = -\frac{1}{2}\left(\frac{\alpha_m + 2\mu_m}{\alpha_m + \mu_m}\right), \\
\Upsilon_\theta^{\mathcal{R}_0(a_i)} &= \frac{\partial \mathcal{R}_0(a_i)}{\partial \theta} \times \frac{\theta}{\mathcal{R}_0(a_i)} = -\frac{1}{2}\left(\frac{\theta}{1-\theta}\right).
\end{aligned} \tag{3.18}$$

The sensitivity index (S.I) of $\mathcal{R}_0(a_i)$ with respect to other parameters of the model can be computed in the same fashion as (3.18) above. We summarize the overall sensitivity analysis of the model by showing the signs of (S.I) in the table hereunder.

Parameter	S.I	Parameter	S.I
b	+ve	θ	−ve
$\beta_h(a_i)$	+ve	τ	−ve
$\alpha_h(a_i)$	+ve	$\mu_h(a_i)$	−ve
β_m	+ve	μ_m	−ve
α_m	+ve	$\gamma(a_i)$	−ve

Table 2: The sensitivity indices of $\mathcal{R}_0(a_i)$.

The sign of the sensitivity index (S.I) plays a key role in determining how the parameters of the model relate to the basic reproduction number, $\mathcal{R}_0(a_i)$, of the model. In Table 2, the positive sign of (S.I) shows a direct relation of $\mathcal{R}_0(a_i)$ to the parameters in this category while the negative sensitivity index shows an inverse relation of $\mathcal{R}_0(a_i)$ to the parameters. With sensitivity analysis, one can get insight on the appropriate intervention strategies to prevent and control the transmission and spread of the malaria in the community. For example, $\Upsilon_b^{\mathcal{R}_0(a_i)} = 1$ means that increasing (or decreasing) b by 10% increases (or decreases) $\mathcal{R}_0(a_i)$ by the same measure. This suggests that an increase (or decrease) in the exposure to the mosquito bites will lead to the persistence (or reduction) of the disease in the population. Further, if more than half of the human population are born vigilant, say, $\tau = 0.8$, then we have $\Upsilon_\tau^{\mathcal{R}_0(a_i)} = -2.0$. This implies that increasing (or decreasing) τ by 10% decreases (or increases) $\mathcal{R}_0(a_i)$ by 20%. The same is true for the parameter θ. Hence the need for humans to be more vigilant by strictly adhering to the malaria vector control measures of the World Health Organization (WHO).

4. Conclusion

A mathematical compartmental model for the malaria transmission in a two-interacting population of human and mosquito has been studied. The formulated model with discrete-age-structured human population incorporates a class of vigilant human who adheres to the vector control measures. Asymptotic behaviour of the model around the equilibria is examined and the contributory effects of the model parameters are determined in relation to the basic reproduction number, $\mathcal{R}_0(a_i)$. The disease-free equilibrium is shown to be locally asymptotically stable when $\mathcal{R}_0(a_i) < 1$. Here, it is important to raise an objection that the disease-free state may not always be stable since the net basic reproduction number $\mathcal{R}_0(a) < L + 1$ across all the possible ages in the popula-

tion. Therefore there is need for vigilant compartment of individuals in which everyone adhere to the rules of keeping the mosquitoes at bay. The endemic equilibrium is proved to be locally asymptotically stable whenever $\mathcal{R}_0(a_i) > 1$. It is further shown that the incidence of the disease can be curtailed if high proportions of the susceptible and exposed humans are vigilant.

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