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Modeling the Inflow of Exposed and Infected Migrants on the Dynamics of Malaria

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Abstract. Malaria is currently a life-threatening vector borne disease which is endemic in most of the developing and underdeveloped countries associated with poor health care systems. In this study, a host-vector mathematical model that takes into account the inflow of human migrants who have been exposed or infected with malaria is formulated and analysed. The reproduction number of the mosquito vector population is derived and used as a threshold quantity for determining the existence of the model trivial and realistic steady states. The Routh-Hurwitz criterion and some stability theorems of Metzler matrices are used to show that the realistic disease free equilibrium is both locally and globally asymptotically stable whenever the disease reproductive number is less than one. We derived an equation for the model endemic condition and used Descartes Rule of Sign Change to established the conditions for the model to admit one or three endemic equilibrium state(s). It is further shown that in the absence of inflow of exposed or infected migrants, the model admits a globally asymptotically unique endemic equilibrium when $R_0 > 1$ and two endemic equilibria when $R_0 < 1$. Our local sensitivity analysis revealed that the adults mosquito removal and biting rates were respectively the most significant contributing parameters to the spread of malaria. The numerical simulations results suggested that the exposed and infected immigrants have no significant impact on the dynamical behaviour of the model population sub-classes.

1. Introduction

Malaria is currently a life-threatening vector borne disease which is endemic in most of the developing and underdeveloped countries associated with challenging health care systems. More particularly, malaria is highly endemic in sub-Saharan Africa characterized with poor hygienic conditions which serve as suitable breeding site for malaria vectors [1]. Plasmodium parasites and female Anopheles mosquitoes are respectively the causal agent and transmitting vectors of malaria. Among the most vulnerable groups to malaria are expectant mothers and infants under five years of age [1–3]. Common symptoms of malaria include: fever, chills, headache, pain, anaemia and vomiting [1,4]. The World Health Organization (WHO) reported that in 2022 alone, there were two hundred and forty nine million malaria cases recorded globally. Ninety four percent of theses cases

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were recorded in Africa. For example, Ghana recorded within the same period five million three hundred and fifteen thousand five hundred and ninety three (5315593) and eleven thousand five hundred and fifty seven (11557) estimated malaria cases and deaths respectively [3]. Currently, population migration caused by climate change induced factors and conflicts constitute a major threat to the malaria control programs.

Mathematical modeling has become a significant tool box for understanding disease transmission dynamics and evaluating the effectiveness of disease control strategies [5]. These models generally explain the dynamics of infections, provide/estimate the thresholds indicators that determine whether the disease will persist or die out [6–8].According to Mukhaktar et al. [9], mathematically modeling malaria can help better understand the disease dynamics and further unveil how certain factors such as human migration influence the disease transmission process, In that regard, several modeling studies have been conducted concerning human migration and malaria. Authors in [9] assessed how human mobility impact the malaria disease burden in South Sudan. Aprianti et al. [10] examined the effect of susceptible immigrants on the spread of malaria in Indonesia. Yiga et al. [11] analysed a malaria transmission model that takes into consideration the combined effect of infected immigrants and other variables that depend on temperature and rainfall. Maliki et al. [2] modelled the control of malaria in a population with infected immigrants. Witboi et al. [12] presented a malaria population dynamics model with human migrants. Yacheur et al. [13] studied the importation of malaria infections from sub-Saharan Africa to northern Africa and the absorption effect of the immigrants. Researchers in [14, 15] formulated and analyzed mathematical models for malaria disease dynamics that considered malaria vaccination campaigns and inflow of infective immigrants. Ahkrizal et al. [16] formulated a malaria dynamics model capturing the inflow of exposed and infected migrants and the recovery of exposed individuals.

In the above mentioned literature, little attention is given to the aquatic phase of the malaria vectors. Even though, the population of adults mosquitoes responsible for disseminating malaria infections is proportional to the density of the aquatic mosquitoes. It is therefore necessary to take into consideration the aquatic stages of the vector in a malaria model [11, 17]. Hence, in this study, in order to explore the impact of exposed and infected individuals on the endemic condition of malaria, we extend the malaria models formulated in [11] to include the exposed vectors and the model in [16] to capture the aquatic stage of the Anopheles female mosquito without the relapse factor of the recovered individuals. The rest of the organization of the paper is as follows: section two takes care of the model formulation and analysis, in section three, the sensitivity analysis results is presented, population simulations is carried out in section four and the conclusion is presented in section five.

2. Malaria Model Development

The model considered the interactions of humans (hosts) and female Anopheles mosquitoes (vectors). Humans (hosts) are classified into susceptible (S_h), exposed (E_h), infected (I_h) and recovered (R_h) sub-classes. As a result, the total human population at any given time t is:

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t) \tag{1}$$

The human/host population is sustained at a constant birth rate π_h and immigration rate M. Hence, the susceptible human class is generated at a rate $(\pi_h + (1 - p_1 - p_2)M)$, where p_1 and p_2 are the immigration rate of exposed and infected migrants respectively. Recovered immigrants are assumed to be susceptible to malaria. Susceptible humans become exposed to malaria infections through effective contact with infected female Anopheles mosquitoes during blood meal at a rate λ_h. Exposed humans progress to infected class at rate γ. The size of exposed humans is augmented as results of immigration of humans at a rate $p_1 M$. It is common to find people in settings with limited health facilities resorting to self medication after being bitten by mosquitoes or when a family member is suspected of suffering from malaria. Hence, in this model it is assumed that exposed individuals recover from malaria at a rate ω. The density of the infected humans is reduced following treatment at rate τ or due to malaria induced mortality at a rate δ. The size of the infected humans is augmented due to migration of infected individuals at rate $p_2 M$. Recovered individuals lose their immunity and join the susceptible sub-class at a rate φ. The constant μ_h is the human removal rate from each human compartment.

Also, the vector (Anopheles mosquito) population is stratified into immature and adult mosquito sub-populations. The immature female Anopheles mosquito sub-population includes the mosquito eggs, larvae and pupae stages.

These aquatic stages are represented by a single compartment denoted by (A_m). The aquatic vector (A_m) is generated from the eggs laid by the matured mosquitoes (susceptible, exposed and infected) at a rate $\pi_m \left(1 - \frac{A_m}{K}\right)(S_m + E_m + I_m)$.

The population of aquatic vector is bounded above by the carrying capacity of the aquatic environment (K). The aquatic mosquito population declines due to natural death at a rate μ_a. The aquatic mosquitoes mature into susceptible mosquitoes at a rate ψ. The matured mosquito is further stratified into susceptible (S_m), exposed (E_m) and infected (I_m) vectors. The susceptible vectors become exposed to malaria parasites during blood meal from infectious (infected) humans at a rate λ_m. Exposed vectors (E_m) subsequently become infected at a rate σ. As the results of natural death at a rate μ_m, the densities of adult mosquito populations ((S_m)), (E_m), (I_m) decrease. Thus, at any time t, the aquatic and adult malaria vector populations (A_m and N_{am}) satisfy:

$$A_m(t) \leq K, \quad N_{am}(t) = S_m(t) + E_m(t) + I_m(t) \tag{2}$$

$\lambda_h = \frac{b\beta_h I_m}{N_h}$ and $\lambda_m = \frac{b\beta_m I_h}{N_h}$ are respectively the forces of infection for the human and female Anopheles mosquitoes. The schematic diagram (figure 1) describes the transmission dynamics of malaria in an interacting human and mosquito populations. The model parameters are presented in Table (1).

TABLE 1. Parameter description with their values and sources

Parameter	Description	Value[Range]	Reference	Unit
π_h	Human recruitment rate	0.03	[11]	Day^{-1}
M	Immigration rate of human	0.001	[11]	Day^{-1}
p_1	Immigration rate of exposed humans	0.2	[11,16]	Day^{-1}
p_2	Immigration rate of infected humans	0.2	[11,16]	Day^{-1}
μ_h	Natural mortality rate of human	1/21900	[11]	Day^{-1}
β_h	Probability of transmission of infections from an infectious human to a susceptible mosquito (vector)	0.00021	[11]	-
γ	Progression rate from exposed humans to infected humans	1/20	[11]	Day^{-1}
ω	Progression rate from exposed humans to recovered humans	0.055	[16]	Day^{-1}
τ	Progression rate from infected humans to recovered humans	1/30	[11]	Day^{-1}
φ	Progression rate from recovered humans to susceptible humans	$1/(20 \times 365)$	[11]	Day^{-1}
δ	Malaria induced death for humans	0.001	[11]	Day^{-1}
π_m	Anopheles mosquito egg deposition rate	6	[17,18]	Day^{-1}
K	Carrying capacity for immature mosquitoes	40000	[18]	Space
b	Female Anopheles mosquito biting rate	0.94[0.1-1]	[18]	Day^{-1}
β_m	Probability of transmission of infections from an infected Anopheles mosquito to a susceptible human	0.00021	[11]	-
ψ	Maturity rate of immature mosquitoes	0.08	[19]	Day^{-1}
σ	Progression rate from exposed mosquitoes to infected mosquitoes	0.091	[18]	Day^{-1}
μ_m	Natural mortality rate of adult mosquitoes	0.11346	[17]	Day^{-1}
μ_a	Natural mortality rate of immature mosquito	0.1042	[18,19]	Day^{-1}

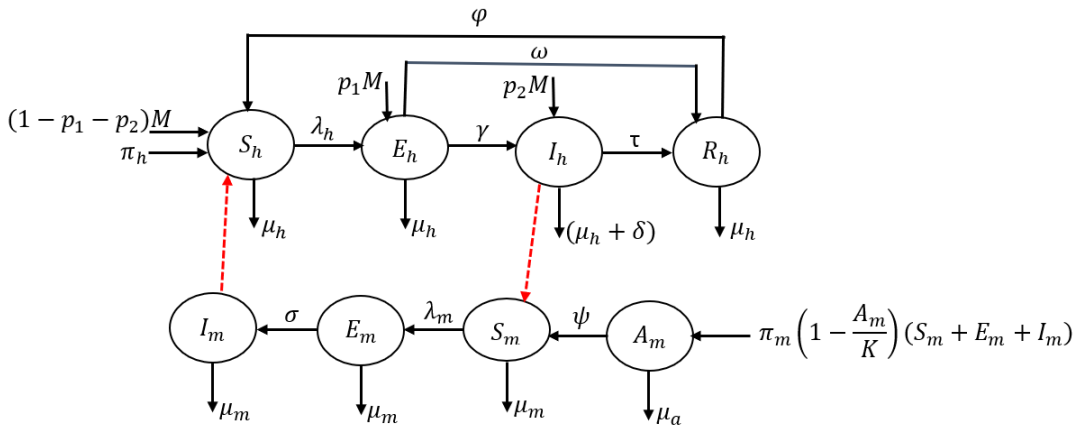

FIGURE 1. Schematic diagram for malaria transmission dynamics with human immigrants

Based on figure 1, the following system of equations is derived:

$$\begin{cases} \frac{dS_h}{dt} = \pi_h + (1-p_1-p_2)M + \varphi R_h - (\lambda_h+\mu_h)S_h \\ \frac{dE_h}{dt} = p_1M + \lambda_h S_h - g_0 E_h \\ \frac{dI_h}{dt} = p_2M + \gamma E_h - g_1 I_h \\ \frac{dR_h}{dt} = \tau I_h + \omega E_h - g_2 R_h \\ \frac{dA_m}{dt} = \pi_m\left(1-\frac{A_m}{K}\right)(S_m+E_m+I_m) - g_3 A_m \\ \frac{dS_m}{dt} = \psi A_m - (\lambda_m+\mu_m)S_m \\ \frac{dE_m}{dt} = \lambda_m S_m - g_4 E_m \\ \frac{dI_m}{dt} = \sigma E_m - \mu_m I_m \end{cases} \tag{3}$$

where: $g_0 = (\omega+\gamma+\mu_h)$, $g_1 = (\tau+\delta+\mu_h)$, $g_2 = (\varphi+\mu_h)$, $g_3 = (\psi+\mu_a)$ and $g_4 = (\sigma+\mu_m)$

2.1. **Boundedness of Solution.**

Theorem 1. *For non-negative initial values $S_h(0)$, $E_h(0)$, $I_h(0)$, $T_h(0)$, $A_m(0)$, $S_m(0)$ and $I_m(0)$ of system (3), each element of the solution set $\{S_h(t),\ E_h(t),\ I_h(t),\ T_h(t),\ A_m(t),\ S_m(t)\ I_m(t)\}$ is non-negative and bounded $\forall\ t \geq 0$.*

Proof. Considering the first differential equation in system (3):

$$\frac{dS_h}{dt} = \pi_h + (1 - p_1 - p_2)M + \varphi R_h - (\lambda_h + \mu_h)S_h \tag{4}$$

$$\implies \frac{dS_h}{dt} \geq -(\lambda_h + \mu_h)S_h$$

$$\implies \int \frac{1}{S_h} dS_h \geq -\int (\lambda_h + \mu_h)dt$$

$$\implies S_h(t) \geq S_h(0)e^{-(\mu_h t + \int_0^t \lambda_h(x)dx)} \geq 0$$

Similarly:

$$\frac{dE_h}{dt} = p_1 M + \lambda_h S_h - g_0 E_h \implies E_h(t) \geq E_h(0)e^{-g_0 t} \geq 0$$

$$\frac{dI_h}{dt} = p_2 M + \gamma E_h - g_1 I_h \implies I_h(t) \geq I_h(0)e^{-g_1 t} \geq 0$$

$$\frac{dR_h}{dt} = \omega E_h + \tau I_h - g_2)R_h \implies R_h(t) \geq R_h(0)e^{-g_2 t} \geq 0$$

$$\frac{dA_m}{dt} = \pi_m \left(1 - \frac{A_m}{K}\right)(S_m + E_m + I_m) - g_3 A_m \implies A_m(t) \geq A_m(0)e^{-g_3 t} \geq 0$$

$$\frac{dS_m}{dt} = \psi A_m - (\lambda_m + \mu_m)S_m \implies S_m(t) \geq S_m(0)e^{-(\mu_m t + \int_0^t \lambda_m(x)dx)} \geq 0$$

$$\frac{dE_m}{dt} = \lambda_m S_m - g_4 E_m \implies S_m(t) \geq S_m(0)e^{-g_4 t} \geq 0$$

$$\frac{dI_m}{dt} = \lambda_m S_m - \mu_m I_m \implies I_m(t) \geq I_m(0)e^{-\mu_m t} \geq 0$$

Therefore, for $\forall$ t $\geq$ 0, the state variables of the model have non-negative solutions.

2.2. **Invariant Region.** This section is dedicated to finding the region over which the solution set of our malaria model system of equations is well posed.

Theorem 2. *The feasible region in which the solution set of the model system of equations make biological sense is the set;*

$$\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_m \subset \mathbb{R}_+^4 \times \mathbb{R}_+^4 \tag{5}$$

where

$$\mathcal{D}_h = \left\{(S_h, E_h, I_h, R_h) \in \mathbb{R}_+^4 : S_h + E_h + I_h + R_h \leq \frac{M + \pi_h}{\mu_h}\right\} \tag{6}$$

and

$$\mathcal{D}_m = \left\{(A_m,\ S_m,\ E_m,\ I_m) \in \mathbb{R}_+^4 :\ A_m \leq K,\ S_m + E_m + I_m \leq \frac{\psi K}{\mu_m}\right\} \tag{7}$$

Proof.

Firstly, we determine the subset $\mathcal{D}_h$.

The human (host) population N_h at any given time t is:

$$N_h = S_h + E_h + I_h + R_h \tag{8}$$

Taking the differential of both sides of equation (8) and simplifying gives:

$$\begin{aligned} &\frac{dN_h}{dt} = M + \pi_h - \mu_h N_h - \delta I_h \\ \Longrightarrow\ &\frac{dN_h}{dt} \leq M + \pi_h - \mu_h N_h \text{ (in the absence of malaria induced mortality)} \\ \Longrightarrow\ &\frac{dN_h}{N_h - \frac{M+\pi_h}{\mu_h}} \leq -\mu_h dt \end{aligned} \tag{9}$$

Integrating the last inequality in (9) and taking the limit as $t \to +\infty$, yield: $N_h \to \frac{M+\pi_h}{\mu_h}$

Consequently, the following result is obtained

$$0 \leq N_h \leq \frac{M + \pi_h}{\mu_h} \tag{10}$$

Therefore:

$$\mathcal{D}_h = \left\{ (S_h,\ E_h,\ I_h,\ R_h) \in \mathbb{R}^4_+ : S_h + E_h + I_h + R_h \leq \frac{M + \pi_h}{\mu_h} \right\} \tag{11}$$

Secondly, the subset $\mathcal{D}_m$ is determined. At any point in time, the mosquito (vector) population satisfies:

$$A_m \leq K, \quad N_{am} = S_m + E_m + I_m \tag{12}$$

$$\begin{aligned} \text{Now,}\quad & N_{am} = S_m + E_m + I_m \\ \Longrightarrow\ & \frac{d}{dt}(N_{am}) = \frac{d}{dt}(S_m + E_m + I_m) \\ \Longrightarrow\ & \frac{dN_{am}}{dt} = \frac{dS_m}{dt} + \frac{dE_m}{dt} + \frac{dI_m}{dt} \\ \Longrightarrow\ & \frac{dN_{am}}{dt} \leq \psi K - \mu_m N_{am} \\ \Longrightarrow\ & N_{am} - \frac{\psi K}{\mu_m} \leq \left(N_{am}(0) - \frac{\psi K}{\mu_m} \right) e^{-\mu_m t} \\ \Longrightarrow\ & N_{am} \leq \frac{\psi K}{\mu_m} \quad \text{as} \quad t \to +\infty. \end{aligned}$$

$$\text{Therefore,}\quad \mathcal{D}_m = \left\{ (A_m,\ S_m,\ E_m,\ I_m) \in \mathbb{R}^4_+ :\ A_m \leq K,\quad S_m + E_m + I_m \leq \frac{\psi K}{\mu_m} \right\} \tag{13}$$

Thus, the feasible region for system (3) is the set:

$$\mathcal{D} = \mathcal{D}_h \times \mathcal{D}_m\ \subset \mathbb{R}^4_+ \times \mathbb{R}^4_+ \tag{14}$$

2.3. **Model Equilibrium Points.** To discuss the model equilibrium points, we consider two cases. The case where there is inflow of exposed and infected migrants ($p_1, p_2 > 0$). For this scenario, there is no disease free equilibrium and the model admits only the endemic equilibrium to be determine later. The second case is when there is no immigration of exposed and infected humans ($p_1 = p_2 = 0$). In this case, the computation of the model disease-free equilibria, is summarized in the theorem below. This approach is adopted from [20–22].

Theorem 3. *For convenience, we define the threshold parameter*

$$\mathcal{N} = \frac{\pi_m \psi}{\mu_m(\psi + \mu_a)} = \frac{\pi_m \psi}{g_3 \mu_m} \tag{15}$$

as the mosquito net reproduction or extinction number, then if:

(1) $\mathcal{N} \leq 1$, *system (3) admits a trivial disease-free equilibrium (TDFE) (which corresponds to a population without mosquitoes) given by:*

$$\xi_0 = (S_h^*,\ 0,\ 0,\ 0,\ \ 0,\ 0,\ 0,\ 0) \tag{16}$$

(2) $\mathcal{N} > 1$ *(mosquitoes persist in the community), system (3) admits a realistic disease-free equilibrium (RDFE) (since it corresponds to the existence of mosquitoes in the population) given by:*

$$\xi_1 = (S_h^*,\ 0,\ 0,\ 0,\ 0,\ A_m^*,\ S_m^*,\ 0) \tag{17}$$

where: $S_h^* = \frac{M+\pi_h}{\mu_h}$, $A_m^* = K\left(1 - \frac{1}{\mathcal{N}}\right)$ *and* $S_m^* = \frac{\psi K}{\mu_m}\left(1 - \frac{1}{\mathcal{N}}\right)$

Proof.

Suppose, $\left(S_h^*,\ E_h^*,\ I_h^*,\ R_h^*,\ A_m^*,\ S_m^*,\ E_m^*,\ I_m^*\right)$ is any arbitrary disease-free equilibrium point. Setting system (3) to zero with the condition that there are no infections at the disease-free equilibrium, that is, $p_1 = p_2 = E_h^* = I_h^* = R_h^* = E_m^* = I_m^* = 0$, gives: $S_h^* = \frac{M+\pi_h}{\mu_h}$ for the first equation.

Also, it is not hard to see from system (3) that

$$S_m^* + E_m^* + I_m^* = \frac{\psi A_m^*}{\mu_m} \tag{18}$$

Hence, from the sixth equation of system (3), we see that A_m^* satisfies:

$$\frac{\psi \pi_m}{\mu_m}\left(1 - \frac{A_m^*}{K}\right) A_m^* - g_3 A_m^* = 0 \tag{19}$$

$$\implies A_m^* = 0 \text{ or } A_m^* = K\left(1 - \frac{1}{\mathcal{N}}\right) \tag{20}$$

$$\text{Now } A_m^* = 0 \implies S_m^* = 0 \text{ and } A_m^* = K\left(1 - \frac{1}{\mathcal{N}}\right) \implies S_m^* = \frac{\psi K}{\mu_m}\left(1 - \frac{1}{\mathcal{N}}\right) \tag{21}$$

Hence, ξ_0 and ξ_1 are obtained respectively from $A_m^* = 0$ and $A_m^* = K\left(1 - \frac{1}{\mathcal{N}}\right)$. Clearly, the magnitude of $\mathcal{N}$ dictates the existence of the model disease-free equilibrium points.

$\mathcal{N}$ is a threshold quantity known as the vector offspring number or vector net reproduction number [18, 19, 23]. In general, $\mathcal{N}$ can be interpreted as a measure of the average number of new adult female Anopheles mosquitoes produced by one reproductive Anopheles mosquito during its entire reproductive life. It is expressed as a product of the egg deposition rate π_m, the fraction of immature mosquito that survive and develop into adult Anopheles mosquito $\frac{\psi}{\psi+\mu_a}$ and the average life span of adult Anopheles mosquito $\frac{1}{\mu_m}$. Thus, if $\mathcal{N} > 1$, the mosquito population persists in the community, otherwise if $\mathcal{N} \leq 1$, the malaria vector population becomes extinct and the local transmission of malaria cannot take place. It is worth noting that the trivial disease-free equilibrium (TDFE) corresponds to the absence of female Anopheles mosquitoes in the community. Hence, the TDFE is biologically less meaningful.

2.4. **The Basic Reproductive Number.** In epidemiology, the basic reproductive number (R_o) is a threshold quantity that is used to determine the extent of severity of the epidemics. In this study, the method of next generating matrix is adopted to compute the model R_o. Expressing our model differential equations in the form $\frac{dX}{dt} = (\mathcal{F} - \mathcal{V})X^T$ where X^T denotes the transpose of $X = (E_h,\ I_h,\ E_m,\ I_m)$, $\mathcal{F}$ and $\mathcal{V}$ are vectors denoting the rate of generation of new infections and transfer rates respectively, gives:

$$\mathcal{F} = \begin{pmatrix} p_1 M + \lambda_h S_h \\ p_2 M \\ \lambda_m S_m \\ 0 \end{pmatrix} \text{ and } \mathcal{V} = \begin{pmatrix} g_0 E_h \\ -\gamma E_h + g_1 I_h \\ g_4 E_m \\ -\sigma E_m + \mu_m I_m \end{pmatrix} \tag{22}$$

Evaluating the Jacobian matrices F and V of $\mathcal{F}$ and $\mathcal{V}$ at the RDFE gives respectively:

$$F = \begin{pmatrix} 0 & 0 & 0 & \frac{b\beta_h S_h^*}{N_h^*} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{b\beta_m S_m^*}{N_h^*} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} g_0 & 0 & 0 & 0 \\ -\gamma & g_1 & 0 & 0 \\ 0 & 0 & g_4 & 0 \\ 0 & 0 & -\sigma & \mu_m \end{pmatrix} \tag{23}$$

From the expression of V, the inverse of V is:

$$V^{-1} = \begin{pmatrix} \frac{1}{g_0} & 0 & 0 & 0 \\ \frac{\gamma}{g_0 g_1} & \frac{1}{g_1} & 0 & 0 \\ 0 & 0 & \frac{1}{g_4} & 0 \\ 0 & 0 & \frac{1}{g_4 \mu_m} & \frac{1}{\mu_m} \end{pmatrix} \tag{24}$$

Hence, the next generation matrix FV^{-1} is given by:

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\sigma b\beta_h S_h^*}{g_4 N_h^* \mu_m} & \frac{b\beta_h S_h^*}{N_h^* \mu_m} \\ 0 & 0 & 0 & 0 \\ \frac{\gamma b\beta_m S_m^*}{g_0 g_1 N_h^*} & \frac{b\beta_m S_m^*}{g_1 N_h^*} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{25}$$

Solving for λ in the relation $\left|FV^{-1} - \lambda I\right| = 0$, where I is a unit matrix and λ an eigenvalue of FV^{-1}, we get the dominant eigenvalue as:

$$\lambda_{max} = R_0 = \sqrt{\frac{\sigma\gamma b^2\beta_h\beta_m S_h^* S_m^*}{g_0 g_1 g_4 N_h^{*2}\mu_m}} \tag{26}$$

Taking $N_h^* = S_h^*$ and simplifying the expression in (26), we obtain the reproductive number of the model given by:

$$\begin{aligned} R_0 &= \sqrt{\frac{\sigma\gamma b^2\beta_h\beta_m\mu_h K\psi}{g_0 g_1 g_4 (M+\pi_h)\mu_m^2}\left(1 - \frac{1}{\mathcal{N}}\right)} \\ &= \sqrt{R_{0h} \times R_{0m}} \end{aligned} \tag{27}$$

where:

$$R_{0h} = \frac{\gamma b\beta_h\mu_h}{g_0 g_1 (M+\pi_h)} \quad \text{and} \quad R_{0m} = \frac{\sigma b\beta_m K\psi}{g_4\mu_m^2}\left(1 - \frac{1}{\mathcal{N}}\right)$$

The threshold quantities R_{0h} and R_{0m} characterized the contributions of malaria disease spread from human to mosquito (host to vector) and from mosquito to human (vector to host) respectively. R_{0h} represents the number of secondary cases of Anopheles mosquitoes one infectious (infected or treated) human will generate in a completely susceptible population of Anopheles mosquitoes during its infectious phase. Similarly, R_{0m} can be interpreted as the number of secondary human cases generated by an infected Anopheles mosquito in an entirely susceptible human population over the course of its life time as infectious [2].

2.5. **Stability of Malaria-Free Equilibrium.**

2.5.1. *Local Stability of Malaria-Free Equilibrium.*

Theorem 4. *The RDFE* $(\xi_1) = \left(\frac{M+\pi_h}{\mu_h},\ 0,\ 0,\ 0,\ 0,\ K\left(1 - \frac{1}{\mathcal{N}}\right),\ \frac{K\psi}{\mu_m}\left(1 - \frac{1}{\mathcal{N}}\right),\ 0\right)$ *with* $\mathcal{N} > 1$ *is locally asymptotically stable (LAS) if* $R_0 < 1$ *and unstable if* $R_0 > 1$

Proof. Let matrix J_0 be the Jacobian matrix of system (3) evaluated at the RDFE (ξ_1). Thus,

$$J_0 = \begin{pmatrix} -\mu_h & 0 & 0 & \varphi & 0 & 0 & 0 & -\frac{b\beta_h S_h^*}{N_h^*} \\ 0 & -g_0 & 0 & 0 & 0 & 0 & 0 & \frac{b\beta_h S_h^*}{N_h^*} \\ 0 & \gamma & -g_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & \omega & \tau & -g_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\left(g_3 + \frac{\pi_m S_m^*}{K}\right) & \frac{\pi_m}{\mathcal{N}} & \frac{\pi_m}{\mathcal{N}} & \frac{\pi_m}{\mathcal{N}} \\ 0 & 0 & -\frac{b\beta_m S_m^*}{N_h^*} & 0 & \psi & -\mu_m & 0 & 0 \\ 0 & 0 & \frac{b\beta_m S_m^*}{N_h^*} & 0 & 0 & 0 & -g_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma & -\mu_m \end{pmatrix} \tag{28}$$

It is not hard to see that the matrix in (28) admits two negative eigenvalues, namely $\lambda_1 = -\mu_h$ and $\lambda_2 = -g_2$. Using the matrix reduction method, the remaining eigenvalues can be obtained from the sub-matrix in (29) below:

$$J_1 = \begin{pmatrix} -g_0 & 0 & 0 & 0 & 0 & \frac{b\beta_h S_h^*}{N_h^*} \\ \gamma & -g_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\left(g_3 + \frac{\pi_m S_m^*}{K}\right) & \frac{\pi_m}{\mathcal{N}} & \frac{\pi_m}{\mathcal{N}} & \frac{\pi_m}{\mathcal{N}} \\ 0 & -\frac{b\beta_m S_m^*}{N_h^*} & \psi & -\mu_m & 0 & 0 \\ 0 & \frac{b\beta_m S_m^*}{N_h^*} & 0 & 0 & -g_4 & 0 \\ 0 & 0 & 0 & 0 & \sigma & -\mu_m \end{pmatrix} \tag{29}$$

The characteristic equation of the sub-matrix in (29) is given by

$$(\lambda + g_0)(\lambda + g_1)(\lambda + g_4)(\lambda + \mu_m)\left(\lambda^2 + \mathcal{S}\lambda + \mathcal{P}\right) = 0 \tag{30}$$

where:

$\mathcal{S} = \frac{\pi_m S_m^*}{K} + g_3 + \mu_m$, and $\mathcal{P} = \frac{\pi_m \mu_m S_m^*}{K}$

It can clearly be seen from (30) that four eigenvalues of the sub-matrix in (29) $\lambda_3 = -g_0$, $\lambda_4 = -g_1$, $\lambda_5 = -g_4$, and $\lambda_6 = -\mu_m$ are negative. Also, the nature of the remaining two eigenvalues of the sub-matrix in (29) are determined from :

$$\lambda^2 + \mathcal{S}\lambda + \mathcal{P} = 0 \tag{31}$$

Since, $\mathcal{S}$ and $\mathcal{P}$ are positive whenever $\mathcal{N} > 1$, it implies that the two remaining eigenvalues of the sub-matrix J_1 are stricly negative. Consequently, all eigenvalues of the matrix J_0 are real and negative. Hence, according to the Routh-Hurwitz stability criterion, the malaria realistic disease-free equilibrium state ξ_1 is locally asymptotically stable when $\mathcal{N} > 1$ and $R_0 < 1$ and unstable otherwise.

2.5.2. *Global Stability of Malaria-Free Equilibrium.* Following [19, 24–27], the global stability of a system equilibrium point can be established by first expressing the system in a triangular form as follows:

$$\begin{cases} \frac{dY_s}{dt} = B_1\left(Y_s - Y_{RDFE}\right) + B_{12}Y_i \\ \frac{dY_i}{dt} = B_2 Y_i \end{cases} \tag{32}$$

Here, Y_s and Y_i denotes the compartments of non-transmitting and transmitting hosts and vectors respectively, with $Y_s = (S_h,\ R_h,\ A_m,\ S_m)^T$ $Y_i = (E_h,\ I_h,\ E_m,\ I_m)^T$ and $Y_{RDFE} = \left(S_h^*,\ R_h^*,\ A_m^*,\ S_m^*\right) = \left(\frac{M+\pi_h}{\mu_h}, K\left(1-\frac{1}{\mathcal{N}}\right), \frac{\psi K}{\mu_m}\left(1-\frac{1}{\mathcal{N}}\right)\right)$

$$\left(Y_s - Y_{RDFE}\right) = \begin{cases} S_h - \frac{M+\pi_h}{\mu_h} \\ R_h \\ A_m - K\left(1-\frac{1}{\mathcal{N}}\right) \\ S_m - \frac{K\psi}{\mu_m}\left(1-\frac{1}{\mathcal{N}}\right) \end{cases} \tag{33}$$

$$B_1 = \frac{\partial Y_s}{\partial(S_h,\ R_h,\ A_m,\ S_m)} \tag{34}$$

$$B_{12} = \frac{\partial Y_s}{\partial(E_h,\ I_h,\ E_m,\ I_m)} \tag{35}$$

$$B_2 = \frac{\partial Y_i}{\partial(E_h,\ I_h,\ E_m,\ I_m)} \tag{36}$$

Using our model system of equations system (3), we get:

$$B_1 = \begin{pmatrix} -\mu_h & \varphi & 0 & 0 \\ 0 & -g_2 & 0 & 0 \\ 0 & 0 & -\left(\frac{\pi_m S_m^*}{K} + \psi + \mu_a\right) & \frac{\pi_m}{\mathcal{N}} \\ 0 & 0 & \psi & -\mu_m \end{pmatrix} \tag{37}$$

$$B_{12} = \begin{pmatrix} 0 & 0 & 0 & -\frac{b\beta_h S_h^*}{N_h^*} \\ \omega & \tau & 0 & 0 \\ 0 & 0 & \frac{\pi_m}{\mathcal{N}} & \frac{\pi_m}{\mathcal{N}} \\ 0 & -\frac{b\beta_m S_m^*}{N_h^*} & 0 & 0 \end{pmatrix} \tag{38}$$

$$B_2 = \begin{pmatrix} -g_2 & 0 & 0 & \frac{b\beta_h S_h^*}{N_h^*} \\ \gamma & -g_1 & 0 & 0 \\ 0 & \frac{b\beta_m S_m^*}{N_h^*} & -g_4 & 0 \\ 0 & 0 & \sigma & -\mu_m \end{pmatrix} \tag{39}$$

From the above we formulate the theorem as follows.

Theorem 5. *The system* $\frac{dY_s}{dt} = B_1(Y_s - Y_{RDFE}) + B_{12}Y_i$ *is globally asymptotically stable (GAS) at the RDFE when all eigenvalues of matrix* B_1 *have negative real parts and* B_2 *is a Metzler matrix.*

Proof.

Clearly, two eigenvalues of the matrix B_1 are $\lambda_1 = -\mu_h$ and $\lambda_2 = -g_2$. Thus, applying the method of matrix reduction, B_1 reduces to the sub-matrix:

$$\mathcal{A} = \begin{pmatrix} -\left(\frac{\pi_m S_m^*}{K} + g_3\right) & \frac{\pi_m}{\mathcal{N}} \\ \psi & -\mu_m \end{pmatrix} \tag{40}$$

The nature of the remaining two eigenvalues of B_1 are determined from characteristic equation:

$$\lambda^2 + \left(g_3 + \mu_m + \frac{\pi_m S_m^*}{K}\right)\lambda + \pi_m\psi\left(1 - \frac{1}{\mathcal{N}}\right) = 0 \tag{41}$$

Since in equation (41), $g_3 + \mu_m + \frac{\pi_m S_m^*}{K} > 0$ and $\pi_m\psi\left(1 - \frac{1}{\mathcal{N}}\right) > 0$ whenever $\mathcal{N} > 1$, we conclude using the Routh-Hurwitz stability condition, that the eigenvalues λ_3 and λ_4 have negative real parts. Hence, all the eigenvalues of the matrix B_1 have negative real parts.

Additionally, B_2 is clearly a Metzler matrix (since all the off diagonal entries are non negative). Thus, we conclude that the system

$$\frac{dY_s}{dt} = B_1(Y_s - Y_{RDFE}) + B_{12}Y_i \tag{42}$$

is GAS at the realistic disease free equilibrium [19,24–27].

2.6. **Malaria Endemic Equilibrium.** Let $\xi_2 = (S_h^{**}, E_h^{**}, I_h^{**}, R_h^{**}, A_m^{**}, S_m^{**}, E_m^{**}, I_m^{**})$ be the endemic equilibrium (EE) point for the malaria model, then setting system (3) to zero, the following system of solutions is obtained

$$\begin{cases} S_h^{**} = Q_0\frac{(g_0 b\varphi\tau\beta_m\mu_h)I_h^{**2} + [Q_1 b\beta_m\mu_h + \mu_m(M+\pi_h)]I_h^{**} + Q_1\mu_m(M+\pi_h)}{[Q_2\sigma b^2\beta_h\beta_m\mu_h\psi A_m^{**} + g_4 b\beta_m\mu_h\mu_m(M+\pi_h)]I_h^{**} + g_0 g_2 g_4\mu_m^2(M+\pi_h)^2} \\ E_h^{**} = \frac{Q_3 I_h^{**3} + Q_4 I_h^{**2} + Q_5 I_h^{**} + Q_6}{Q_7 I_h^{**2} + Q_8 I_h^{**} + Q_9} \\ R_h^{**} = \frac{\tau I_h^{**} + \omega E_h^{**}}{g_2} \\ A_m^{**} = 0 \text{ or } A_m^{**} = K\left(1 - \frac{1}{\mathcal{N}}\right) \\ S_m^{**} = 0 \text{ or } S_m^{**} = \frac{(M+\pi_h)\psi A_m^{**}}{b\beta_m\mu_h I_h^{**} + \mu_m(M+\pi_h)} \\ E_m^{**} = 0 \text{ or } E_m^{**} = \frac{b\beta_m\mu_h S_m^{**} I_h^{**}}{g_4(M+\pi_h)} \\ I_m^{**} = 0 \text{ or } I_m^{**} = \frac{\sigma b\beta_m\mu_h S_m^{**} I_h^{**}}{g_4\mu_m(M+\pi_h)} \\ \text{here, } I_h^{**} \text{ satisfies : } q_3 I_h^{**3} + q_2 I_h^{**2} + q_1 I_h^{**} + q_0 = 0 \end{cases} \tag{43}$$

Where:

$Q_0 = \frac{g_4\mu_m(M+\pi_h)}{\mu_h}$

$Q_1 = p_1\varphi\omega M + g_0 g_2((1-p_1-p_2)M + \pi_h)$

$Q_2 = g_0 g_2 - \varphi\omega$

$$Q_3 = \frac{1}{M+\pi_h} g_0 g_4 \varphi\tau\sigma b^3 \beta_h \beta_m^2 \mu_h \mu_m \psi A_m^{**}$$
$$Q_4 = \frac{1}{M+\pi_h} g_4 \varphi\tau\sigma b^2 \beta_h \beta_m \mu_h \psi A_m^{**} \left(Q_1 b\beta_m\mu_m + \frac{M+\pi_h}{\mu_h} g_0\varphi\tau\right) +$$
$$g_4 p_1 M b\beta_m\mu_m \left(\frac{Q_2\sigma b^2\beta_h\beta_m\mu_h\psi A_m^{**}}{M+\pi_h} + g_0 g_2 g_4 b\beta_m\mu_h\mu_m\right)$$
$$Q_5 = g_4 b\beta_m\mu_m^2 \left(\sigma b\beta_h\psi A_m^{**}(Q_1 + p_1 M Q_2) + 2g_0 g_2 g_4 p_1 M\mu_m(M+\pi_h)\right)$$
$$Q_6 = \frac{g_0 g_2 p_1 M}{\mu_h} \left(\mu_m^2 g_4 (M+\pi_h)\right)^2$$
$$Q_7 = \frac{g_0 g_4 b\beta_m\mu_h\mu_m}{M+\pi_h} \left(Q_2\sigma b^2\beta_h\beta_m\psi A_m^{**} + g_0 g_2 g_4 b\beta_m\mu_m(M+\pi_h)\right)$$
$$Q_8 = g_0 g_4 \mu_m^2 \left(Q_2\sigma b^2\beta_h\beta_m\psi A_m^{**} + 2g_0 g_2 g_4 b\beta_m\mu_m(M+\pi_h)\right)$$
$$Q_9 = \frac{g_2}{\mu_h} \left(g_0 g_4 \mu_m^2 (M+\pi_h)\right)^2$$

$$q_3 = g_0 g_4 b\beta_m\mu_m \{\frac{\sigma b^2\beta_h\beta_m\mu_h A_m^{**}}{M+\pi_h} (g_0 g_1\mu_h + \varphi\gamma(\mu_h+\delta)) + g_0 g_1 g_2 g_4 b\beta_m\mu_h\mu_m\}$$

$$q_2 = g_0 g_1 g_4 \mu_m^2 (M+\pi_h)(\frac{Q_2\sigma b^2\beta_h\beta_m\psi A_m^{**}}{M+\pi_h} + 2g_0 g_2 g_4 b\beta_m\mu_m -$$
$$\frac{\gamma\sigma b^2\beta_h\beta_m A_m^{**}}{M+\pi_h}(g_4 Q_1 b\beta_m\mu_h\mu_m + g_0 g_4\varphi\tau\mu_m^2(M+\pi_h) -$$
$$g_4 M b\beta_m\mu_h\mu_m(p_1\gamma + g_0 p_2)(\frac{\alpha\sigma b^2\beta_h\beta_m A_m^{**}}{M+\pi_h} + g_0 g_2 g_4 b\beta_m\mu_m)$$

$$q_1 = \frac{g_4\mu_m^2(M+\pi_h)}{\mu_h}\{g_0^2 g_1 g_2 g_4 \mu_m^2 (M+\pi_h) - \frac{Q_1\gamma\sigma b^2\beta_h\beta_m\mu_h\psi A_m^{**}}{M+\pi_h} -$$
$$g_0 p_2 M(\frac{Q_2\sigma b^2\beta_h\beta_m\mu_h\psi A_m^{**}}{M+\pi_h} + 2g_0^2 g_2 g_4 b\beta_m\mu_h\mu_m\}$$

$$q_0 = -\frac{g_0 g_2}{\mu_h} M(p_1\gamma + g_0 p_2 \left(g_4\mu_m^2(M+\pi_h)\right)^2$$

To analyse the disease endemic condition, we consider the polynomial function:

$$f(I_h^{**}) = q_3 I_h^{**3} + q_2 I_h^{**2} + q_1 I_h^{**} + q_0 = 0 \tag{44}$$

There is enough evidence that the polynomial in (44) admits a positive solution on the interval $[0, +\infty)$ since: $f(0) = q_0 < 0$ and $\lim_{I_h^{**}\to+\infty} f(I_h^{**}) = +\infty$. Next, we employ Descartes' Rule of Signs Change to explore more information on the roots of the polynomial $f(I_h^{**})$ (see table 2).

TABLE 2. Number (#) of Possible Positive Roots of $f(I_h^{**})$

Case	q_3	q_2	q_1	q_0	# of sign change	# of roots
(i)	+	+	+	−	1	1
(ii)	+	+	−	−	1	1
(iii)	+	−	+	−	3	1, 3
(iv)	+	−	−	−	1	1

Based on the results in table 2, we claim that in the presence of importation of malaria infections, system (3) may admit one or three endemic equilibrium state(s). To better understand any possible impact of the inflow of humans who have been exposed or infected with malaria parasites on the disease endemic condition, we now consider the endemic relation in the absence of exposed and infected immigrants.

To that effect, we set $p_1 = p_2 = 0$ into (44) and simplify to obtain:

$$I_h^{**}\left(a_2 I_h^{**2} + a_1 I_h^{**} + a_0\right) = 0 \tag{45}$$

Equation (45) implies

$$I_h^{**} = 0 \quad \text{or} \quad a_2 I_h^{**2} + a_1 I_h^{**} + a_0 = 0 \tag{46}$$

where:

$$a_2 = b\beta_m\mu_h\{\frac{\sigma b^2\beta_h\beta_m\mu_h A_m^{**}}{M+\pi_h}\left(g_0 g_1\mu_h + \varphi\gamma(\mu_h+\delta)\right) + g_0 g_1 g_4 b\beta_m\mu_h\mu_m\} \tag{47}$$

$$\begin{aligned} a_1 =& g_1\mu_h\mu_m\left(Q_2\sigma b^2\beta_h\beta_m\psi A_m^{**} + 2g_0 g_2 g_4 b\beta_m\mu_m(M+\pi_h)\right) - \\ & \gamma\sigma b^2\beta_h\beta_m\mu_h\psi A_m^{**}\left(g_2 b\beta_m\mu_h + \varphi\tau\mu_m\right) \end{aligned} \tag{48}$$

$$a_0 = g_0 g_1 g_2 g_4\mu_m^3(M+\pi_h)^2\left(1 - R_0^2\right) \tag{49}$$

With $I_h^{**} = 0$ in (46), we retrieve the TDFE when $A_m^{**} = 0$ and the RDFE when $A_m^{**} = K\left(1 - \frac{1}{\mathcal{N}}\right)$ Furthermore, a solution to the quadratic equation in (46) can be obtained using the quadratic formula, that is :

$$I_h^{**} = \frac{-a_1 \pm \sqrt{a_1^2 - 4a_0 a_2}}{2a_2} \tag{50}$$

The expression in (50) leads to the following theorem:

Theorem 6. *In the absence of inflow of exposed and infected human migrants, the malaria model represented by system (3) admits:*

(i) *one unique EE if* $a_0 < 0$, *that is* $R_0 > 1$

(ii) *one unique EE if* $a_1 < 0$, *and* $R_0 = 1$ *or* $a_1^2 - 4a_0 a_2 = 0$

(iii) *two EE if* $a_1 < 0$ *and* $a_0 > 0$ *that is* $R_0 < 1$ *or* $a_1^2 - 4a_0 a_2 > 0$

(iv) *no EE otherwise.*

We deduce from case (i) of theorem 6 that for a specific case where the parameter accounting for the importation of malaria infections is zero, system 3 admits a unique EE when $R_0 > 1$. This suggests that even in the absence of importation of malaria infections from elsewhere, malaria epidemics can continue to propagate in the population. The occurrence of two endemic equilibria when R_0 does not exceed one, case (iii) of theorem 6 shows that the model bifurcate backwardly.

That is, two stable equilibrium states coexist when the reproductive number of the model is less than one. In this case, $R_0 < 1$ even though necessary is no more enough for the elimination of malaria. To obtain the value of R_0 say, R_0^c at which backward bifurcation takes place in this case, we set the discriminant (Δ) of equation 46 to zero and solve for R_0^c. That is

$$\Delta = 0 \implies a_1^2 - 4a_0a_2 = 0$$
$$\implies R_0^c = \sqrt{1 - \frac{a_1^2}{4a_2g_0g_1g_2g_4\mu_m^3(M+\pi_h)^2}}$$

Hence, for values of R_0 between $R_0^c < R_0 < 1$, system 3 in the absence of importation of malaria infections experiences backward bifurcation.

2.7. **Global Stability of the Malaria Endemic Equilibrium Point.** In what follows, we explore the long term behavior of the unique endemic equilibrium point whenever it exist.

Consider the Lyapunov candidate:

$$\mathcal{L}\left(S_h^{**},\ E_h^{**},\ I_h^{**},\ R_h^{**},\ A_m^{**},\ S_m^{**},\ E_m^{**},\ I_m^{**}\right)$$
$$= \left((S_h - S_h^{**}) - S_h^{**}\ln\frac{S_h}{S_h^{**}}\right) + \left((E_h - E_h^{**}) - E_h^{**}\ln\frac{E_h}{E_h^{**}}\right) + \left((I_h - I_h^{**}) - I_h^{**}\ln\frac{I_h}{I_h^{**}}\right)$$
$$+ \left((R_h - R_h^{**}) - R_h^{**}\ln\frac{R_h}{R_h^{**}}\right) + \left((A_m - A_m^{**}) - A_m^{**}\ln\frac{A_m}{A_m^{**}}\right) + \left((S_m - S_m^{**}) - S_m^{**}\ln\frac{S_m}{S_m^{**}}\right)$$
$$+ \left((E_m - E_m^{**}) - E_m^{**}\ln\frac{E_m}{E_m^{**}}\right) + \left((I_m - I_m^{**}) - I_m^{**}\ln\frac{I_m}{I_m^{**}}\right)$$

Taking the time derivative of $\mathcal{L}$ gives:

$$\frac{d\mathcal{L}}{dt} = \left(1 - \frac{S_h^{**}}{S_h}\right)\frac{dS_h}{dt} + \left(1 - \frac{E_h^{**}}{E_h}\right)\frac{dE_h}{dt} + \left(1 - \frac{I_h^{**}}{I_h}\right)\frac{dI_h}{dt} + \left(1 - \frac{R_h^{**}}{R_h}\right)\frac{dR_h}{dt}$$
$$+ \left(1 - \frac{A_m^{**}}{A_m}\right)\frac{dA_m}{dt} + \left(1 - \frac{S_m^{**}}{S_m}\right)\frac{dS_m}{dt} + \left(1 - \frac{E_m^{**}}{E_m}\right)\frac{dE_m}{dt} + \left(1 - \frac{I_m^{**}}{I_m}\right)\frac{dI_m}{dt}$$
$$= \left(\frac{S_h - S_h^{**}}{S_h}\right)[\pi_h + (1-p_1-p_2)M + \varphi R_h - (\lambda_h + \mu_h)S_h] + \left(\frac{E_h - E_h^{**}}{E_h}\right)(p_1M + \lambda_hS_h - g_0E_h)$$
$$+ \left(\frac{I_h - I_h^{**}}{I_h}\right)(p_2M + \gamma E_h - g_1I_h) + \left(\frac{R_h - R_h^{**}}{R_h}\right)(\tau I_h + \omega E_h - g_2R_h)$$
$$+ \left(\frac{A_m - A_m^{**}}{A_m}\right)\left[\pi_m\left(1 - \frac{A_m}{K}\right)N_{am} - g_3A_m\right] + \left(\frac{S_m - S_m^{**}}{S_m}\right)[\psi A_m - (\lambda_m + \mu_m)S_m]$$
$$+ \left(\frac{E_m - E_m^{**}}{E_m}\right)(\lambda_mS_m - g_4E_m) + \left(\frac{I_m - I_m^{**}}{I_m}\right)(\sigma E_m - \mu_mI_m)$$
$$= \pi_h + (1-p_1-p_2)M + \varphi R_h + (\lambda_h + \mu_h)S_h^{**} - (\pi_h + (1-p_1-p_2)M + \varphi R_h)\frac{S_h^{**}}{S_h} \tag{51}$$

$$
\begin{aligned}
&-(\lambda_h+\mu_h)S_h+p_1M+\lambda_hS_h+g_0E_h^{**}-g_0E_h-(p_1M+\lambda_hS_h)\frac{E_h^{**}}{E_h}+p_2M+\gamma E_h+g_1I_h^{**}\\
&-g_1I_h-(p_2M+\gamma E_h)\frac{I_h^{**}}{I_h}+\tau I_h+\omega E_h+g_2R_h^{**}-g_2R_h-(\tau I_h+\omega E_h)\frac{R_h^{**}}{R_h}+N_{am}\pi_m+N_{am}\pi_m\frac{A_m^{**}}{K}\\
&+g_3A_m^{**}-N_{am}\pi_m\frac{A_m}{K}-g_3A_m-N_{am}\pi_m\frac{A_m^{**}}{A_m}+\psi A_m+\lambda_mS_m+g_4E_m^{**}-g_4E_m-\lambda_mS_m\frac{E_m^{**}}{E_m}\frac{S_m^{**}}{S_m}\\
&+\sigma E_m+\mu_mI_m^{**}-\mu_mI_m-\sigma E_m\frac{I_m^{**}}{I_m}\\
&=\mathcal{L}^+-\mathcal{L}^-
\end{aligned}
$$

where

$$
\begin{aligned}
\mathcal{L}^+&=\pi_h+M+\varphi R_h+(\lambda_h+\mu_h)S_h^{**}+(p_1+p_2)M\frac{S_h^{**}}{S_h}+\lambda_hS_h+g_0E_h^{**}+\gamma E_h+g_1I_h^{**}+\tau I_h\\
&+\omega E_h+g_2R_h^{**}+N_{am}\pi_m+N_{am}\pi_m\frac{A_m^{**}}{K}+g_3A_m^{**}+\psi A_m+(\lambda_m+\mu_m)S_m^{**}+\lambda_mS_m+g_4E_m^{**}\\
&+\sigma E_m+\mu_mI_m^{**}\\
\mathcal{L}^-&=(\pi_h+M+\varphi R_h)\frac{S_h^{**}}{S_h}+(\lambda_h+\mu_h)S_h+g_0E_h+(p_1M+\lambda_hS_h)\frac{E_h^{**}}{E_h}+(p_2M+\gamma E_h)\frac{I_h^{**}}{I_h}+g_1I_h\\
&+g_2R_h+(\tau I_h+\omega E_h)\frac{R_h^{**}}{R_h}+N_{am}\pi_m\frac{A_m}{K}+g_3A_m+N_{am}\pi_m\frac{A_m^{**}}{A_m}+\lambda_mS_m\frac{E_m^{**}}{E_m}+g_4E_m+\mu_mI_m\\
&+\sigma E_m\frac{I_m^{**}}{I_m}
\end{aligned}
\tag{52}
$$

Since the model parameters and state variables are non-negative, it follows from (52) that $\frac{d\mathcal{L}}{dt}\leq 0$ if $\mathcal{L}^+\leq\mathcal{L}^-$ and $\frac{d\mathcal{L}}{dt}=0$ if and only if $S_h^{**}=S_h,\ E_h^{**}=E_h,\ I_h^{**}=I_h,\ R_h^{**}=R_h,\ A_m^{**}=A_m,\ S_m^{**}=S_m,\ E_m^{**}=E_m,$ and $I_m^{**}=I_m$

Therefore, the largest compact invariant set within the model's invariant region is the singleton $\{S_h^{**},\ E_h^{**},\ I_h^{**},\ R_h^{**},\ A_m^{**},\ S_m^{**},\ E_m^{**},\ I_m^{**}\}$. Hence, by the Lasalle's invariant principle [28], the unique endemic equilibrium of system (3) is globally asymptotically stable whenever it exists.

3. Local Sensitivity Analysis

In this section, local sensitivity analysis is carried out to determine the parameters that mostly contribute to disease spread or increase (R_0). These parameters should be targeted during any intervention aimed at combating the malaria infections. Using the normalized forward sensitivity index relation:

$$
\Gamma_x^w=\frac{\partial w}{\partial x}\times\frac{x}{w}
\tag{53}
$$

and the model parameter values provided in Table 1 we compute the values for sensitivity indices of the parameters of the model reproductive number, (R_0) as presented in Table 3.

Table 3. The values of the sensitivity indices

Parameter	Sensitivity index
b	+1.00
β_h	+0.50
β_m	+0.50
π_h	−0.4839
M	−0.0161
μ_h	+0.4991
ω	−0.2618
γ	+0.2620
τ	−0.4848
δ	−0.0291
π_m	$+1.98 \times 10^{-4}$
k	+0.50
ψ	+0.4999
σ	+0.2775
μ_a	-1.12×10^{-4}
μ_m	−1.2779

If the sign of the sensitivity index of a given parameter of R_0 is positive, it means R_0 is directly proportional to that parameter. That is, an increase (decrease) in the parameter value when other parameters remain constant would result in an increase (decrease) in disease incidence. Conversely, if the sign of the sensitivity index of a given parameter is negative, then R_0 is indirectly proportional to that parameter [29]. From table 3, it is clear that an increase in the parameters: b, β_h, β_m, K,ψ. γ, π_m , and σ will lead to an increase in the disease spread (R_0) while an increase in the parameters: μ_m, τ, ω and μ_a will result in a reduction of the disease spread R_0 and vice versa.

4. Numerical Simulations

In order to explore the possible impact of the exposed and infected human immigrants on the dynamical behaviour of the malaria model sub-populations, system (3) is simulated using the following assumed set of initial condition values of the state variables:
$\{S_h(0), E_h(0), I_h(0), R_h(0) A_m(0), S_m(0), E_m(0), I_m(0)\} = \{700, 350, 100, 0, 5000, 1000, 300, 120\}\}$ and the parameter values provided in Table 1. The results (figures 10-13) suggest that the exposed and infected human immigrants have no influence on the population density of the humans and mosquitoes in the community. It can also be observed from the simulation results that the population of the immature and susceptible Anopheles mosquitoes remain high in the community. This implies that efficient vector control

strategies for both the immature and mature Anopheles mosquitoes are urgently required if malaria is to be eradicated from the population.

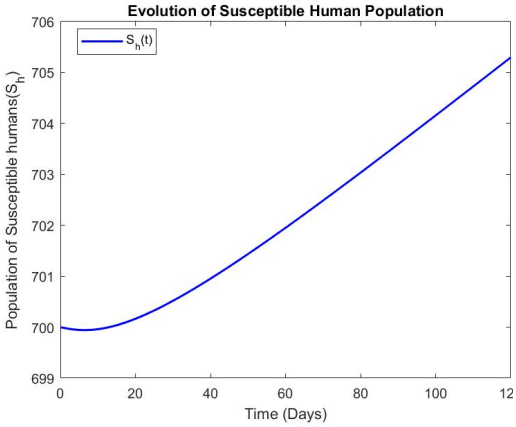

FIGURE 2. Susceptible Humans

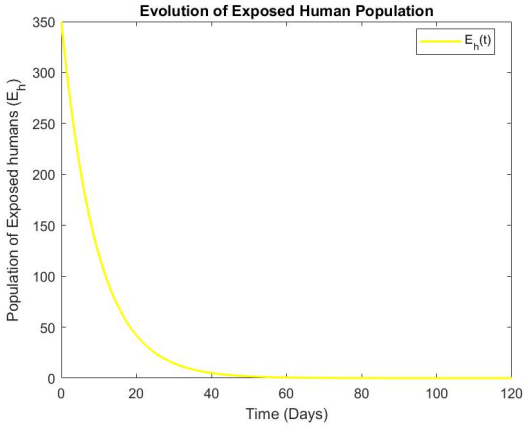

FIGURE 3. Exposed Humans

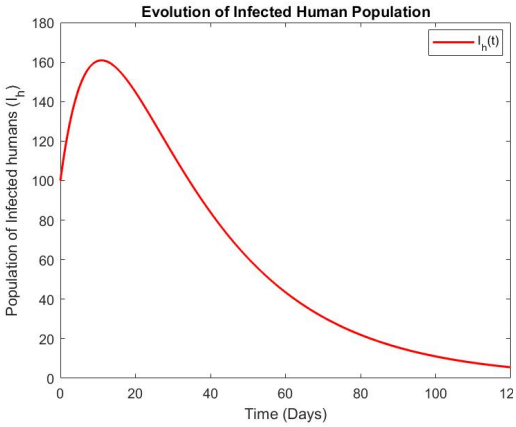

FIGURE 4. Infected Humans

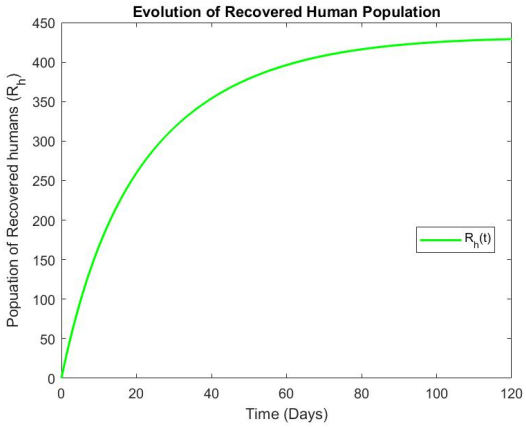

FIGURE 5. Recovered Humans

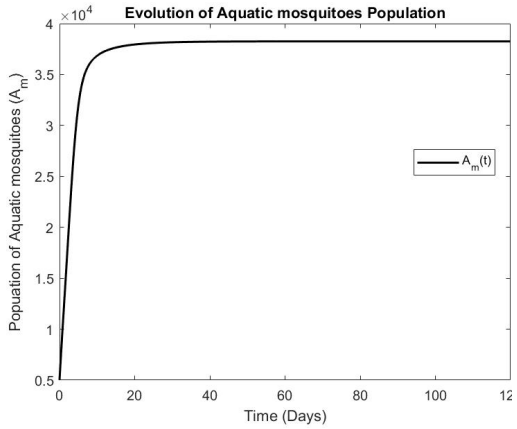

FIGURE 6. Aquatic Mosquito

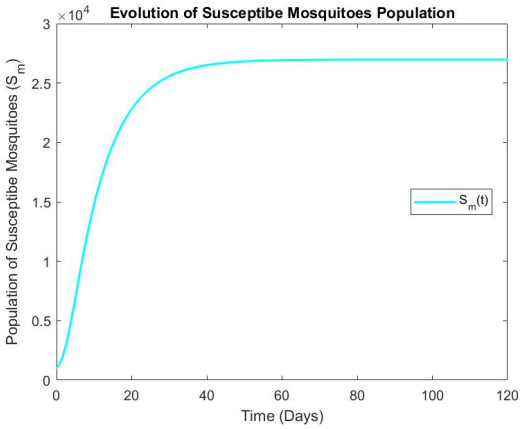

FIGURE 7. Susceptible Mosquito

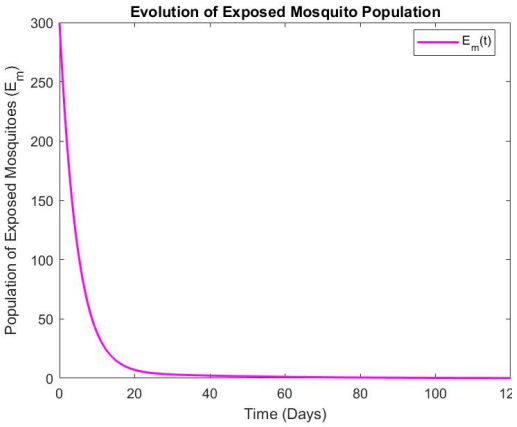

FIGURE 8. Exposed Mosquitoes

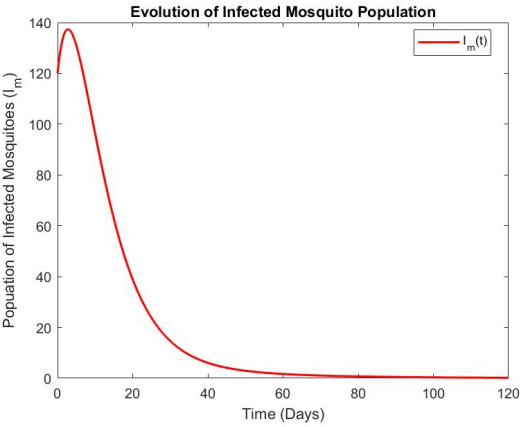

FIGURE 9. Infected Mosquitoes

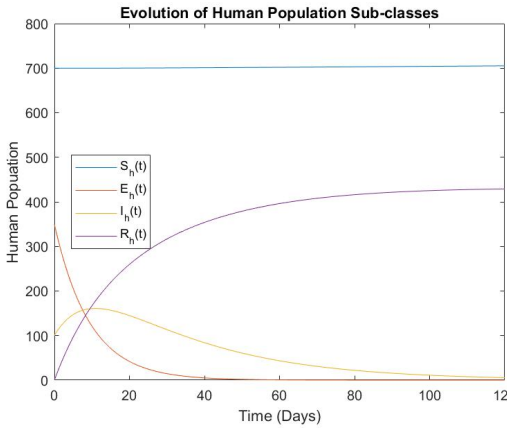

FIGURE 10. Human classes with $p_1 = p_2 = 0.2$

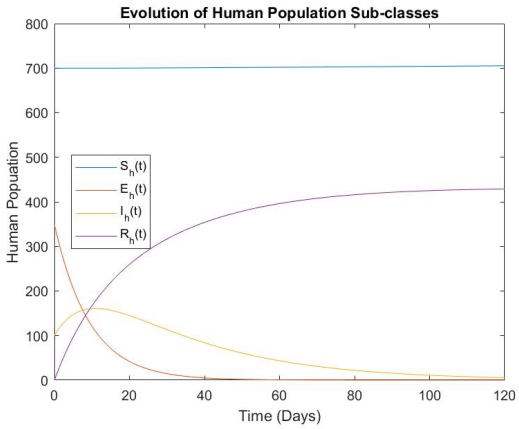

FIGURE 11. Human classes with $p_1 = p_2 = 0$

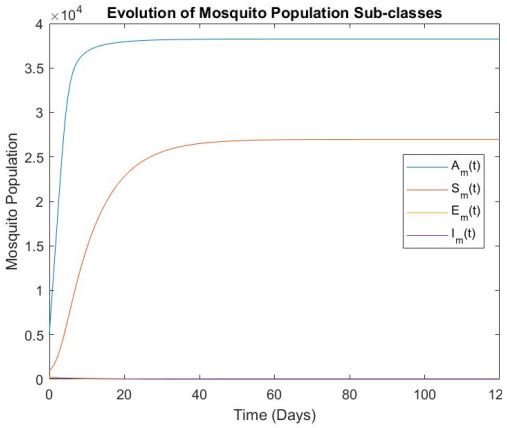

FIGURE 12. Mosquito classes with $p_1 = p_2 = 0.2$

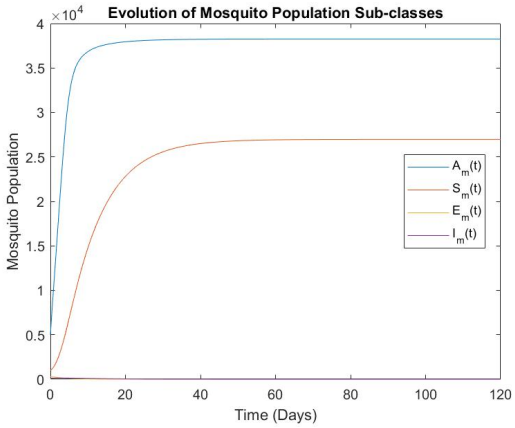

FIGURE 13. Mosquito classes with $p_1 = p_2 = 0$

5. Conclusion

In this study, a deterministic compartmental model for malaria dynamics that takes into consideration the inflow of exposed and infected migrants and the recovery of exposed humans is formulated and analysed. In the absence of inflow of exposed and infected humans from elsewhere, the model disease free states are obtained and the biologically desired infection-free equilibrium point (RDFE) is shown to be both locally and globally asymptotically stable when the disease reproduction number (R_0) is less than one and unstable if $R_0 > 1$. Furthermore, we derived the equation for the endemic condition and used the Descartes rule of sign change to establish the conditions for the model to admit one or three endemic equilibrium state(s). For a special case of no inflow of exposed or infected migrants, we proved that the model admits a global asymptotic stable unique endemic equilibrium if $R_0 > 1$ and two endemic equilibria when $R_0 < 1$. The results from our local sensitivity analysis revealed that adult mosquito removal and biting rates (μ_m and b) are respectively the most sensitive parameters to the spread of malaria. This suggests that malaria vector control remains a key factor for consideration in the elimination of malaria epidemics. Our numerical simulation graphical results indicate that the inflow of exposed and infected migrants has no significant impact on the dynamical behavior of the model population sub-classes. Thus, we recommend that real immigrants data is used to fit the malaria model and explore more on the disease dynamics in the presence of exposed or infected human immigrants.

References

[1] V. Yiga, H. Nampala, J. Tumwiine, Analysis of the model on the effect of seasonal factors on malaria transmission dynamics, J. Appl. Math. 2020 (2020) 1–19.

[2] O. S. Maliki, N. Romanus, B. O. Onyemegbulem, A mathematical modelling of the effect of treatment in the control of malaria in a population with infected immigrants, Appl. Math. 9 (2018) 1238–1257.

[3] W. H. Organization, et al., WHO malaria policy advisory group (MPAG) meeting report, 18–20 April 2023, World Health Organization, 2023.

[4] S. Olaniyi, K. Okosun, S. Adesanya, R. Lebelo, Modelling malaria dynamics with partial immunity and protected travellers: optimal control and cost-effectiveness analysis, J. Biol. Dyn. 14 (2020) 90–115.

[5] M. Y. Li, An introduction to mathematical modeling of infectious diseases, Vol. 2, Springer, 2018.

[6] S. Osman, O. D. Makinde, A mathematical model for co-infection of listeriosis and anthrax diseases, Int. J. Math. Math. Sci. 2018 (2018) 1725671.

[7] N. Chitnis, J. M. Hyman, J. M. Cushing, Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model, Bull. Math. Biol. 70 (2008) 1272–1296.

[8] W. A. Iddrisu, I. Iddrisu, A.-K. Iddrisu, et al., Modeling cholera epidemiology using stochastic differential equations, J. Appl. Math. 2023 (2023) 7232395.

[9] A. Y. Mukhtar, J. B. Munyakazi, R. Ouifki, Assessing the role of human mobility on malaria transmission, Math. Biosci. 320 (2020) 108304.

[10] E. Aprianti, J. Jaharuddin, E. H. Nugrahani, The effect of susceptible immigrants in a system dynamic on the spread of malaria in indonesia, J. Teori Apl. Mat. 6 (2022) 777–788.

[11] V. Yiga, H. Nampala, J. Tumwiine, Stability analysis of a malaria transmission model for the effect of infected immigrants with temperature and rainfall dependent parameters, Int. J. Math. Model. Comp. 12 (2022) 115–130.

[12] P. Witbooi, G. Abiodun, M. Nsuami, A model of malaria population dynamics with migrants, Math. Biosci. Eng. 18 (2021) 7301–7317.

[13] S. Yacheur, A. Moussaoui, A. Tridane, Modeling the imported malaria to north africa and the absorption effect of the immigrants, Math. Biosci. Eng. 16 (2019) 967–989.

[14] O. D. Makinde, K. O. Okosun, Impact of chemo-therapy on optimal control of malaria disease with infected immigrants, BioSystems 104 (2011) 32–41.

[15] P. Duve, S. Charles, J. Munyakazi, R. Lühken, P. Witbooi, A mathematical model for malaria disease dynamics with vaccination and infected immigrants, Math. Biosci. Eng. 21 (2024) 1082–1109.

[16] A. Ahkrizal, J. Jaharuddin, E. H. Nugrahani, Dynamics system in the seir-si model of the spread of malaria with recurrence, Jambura J. Biomath. 4 (2023) 31–36.

[17] B. Traoré, B. Sangaré, S. Traoré, et al., A mathematical model of malaria transmission with structured vector population and seasonality, J. Appl. Math. 2017 (2017) 6754097.

[18] F. Agusto, A. Gumel, P. Parham, Qualitative assessment of the role of temperature variations on malaria transmission dynamics, J. Biol. Syst. 23 (2015) 1550030.

[19] H. Abboubakar, J. C. Kamgang, N. L. Nkamba, D. Tieudjo, L. Emini, Modeling the dynamics of arboviral diseases with vaccination perspective, Biomath 4 (2015) 1507241.

[20] K. Okuneye, A. B. Gumel, Analysis of a temperature-and rainfall-dependent model for malaria transmission dynamics, Math. Biosci. 287 (2017) 72–92.

[21] A. Abidemi, N. A. B. Aziz, Analysis of deterministic models for dengue disease transmission dynamics with vaccination perspective in johor, malaysia, Int. J. Appl. Comp. Math. 8 (2022) 45.

[22] A. Abidemi, N. A. B. Aziz, Optimal control strategies for dengue fever spread in johor, malaysia, Comp. Meth. Progr. Biomed. 196 (2020) 105585.

[23] N. Hussaini, K. Okuneye, A. B. Gumel, Mathematical analysis of a model for zoonotic visceral leishmaniasis, Infect. Dis. Model. 2 (2017) 455–474.

[24] Y. Dumont, F. Chiroleu, C. Domerg, On a temporal model for the chikungunya disease: modeling, theory and numerics, Math. Biosci. 213 (2008) 80–91.

[25] S. Osman, G. T. Tilahun, S. D. Alemu, W. M. Onsongo, Analysis of the dynamics of rabies in north shewa, ethiopia, Italian J. Pure Appl. Math. 48 (2022) 877–902.

[26] Y. A. Liana, N. Shaban, G. Mlay, A. Phibert, African trypanosomiasis dynamics: Modelling the effects of treatment, education, and vector trapping, Int. J. Math. Math. Sci. 2020 (2020) 3690472.

[27] A. Hassan, N. Shaban, Onchocerciasis dynamics: modelling the effects of treatment, education and vector control, J. Biol. Dyn. 14 (2020) 245–268.

[28] J. P. La Salle, The stability of dynamical systems, SIAM, 1976.

[29] M. Martcheva, An introduction to mathematical epidemiology, Vol. 61, Springer, 2015.