

Research Article

Analysis of the Model on the Effect of Seasonal Factors on Malaria Transmission Dynamics

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Received 12 August 2020; Revised 10 November 2020; Accepted 19 November 2020; Published 19 December 2020

Academic Editor: Yansheng Liu

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Malaria is one of the world's most prevalent epidemics. Current control and eradication efforts are being frustrated by rapid changes in climatic factors such as temperature and rainfall. This study is aimed at assessing the impact of temperature and rainfall abundance on the intensity of malaria transmission. A human host-mosquito vector deterministic model which incorporates temperature and rainfall dependent parameters is formulated. The model is analysed for steady states and their stability. The basic reproduction number is obtained using the next-generation method. It was established that the mosquito population depends on a threshold value θ , defined as the number of mosquitoes produced by a female *Anopheles* mosquito throughout its lifetime, which is governed by temperature and rainfall. The conditions for the stability of the equilibrium points are investigated, and it is shown that there exists a unique endemic equilibrium which is locally and globally asymptotically stable whenever the basic reproduction number exceeds unity. Numerical simulations show that both temperature and rainfall affect the transmission dynamics of malaria; however, temperature has more influence.

1. Introduction

Malaria is one of the world's most prevalent epidemic despite a series of control and eradication measures. It is caused by the *Plasmodium* parasite transmitted between humans through the bite of a female *Anopheles* mosquito as it seeks blood necessary for oviposition [1]. The malaria parasites of humans are *Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium ovale*, and *Plasmodium vivax* [2]. *Plasmodium falciparum* and *Plasmodium vivax* are the most prevalent species in the tropical areas and temperate regions, respectively [3]. The life cycle of a mosquito begins as an egg, it hatches into a larva which turns into a pupa, then after about two to four days of pupation, the mosquito emerges as an adult [4]. On biting a human host, the female *Anopheles* mosquito injects sporozoites into the blood of the human host. The sporozoite form of the *Plasmodium* parasite multiplies in the host's liver before developing into the

gametocyte form which is released in the bloodstream and is ingested by a female *Anopheles* mosquito during a future blood meal [5]. Malaria has a long incubation period so symptoms can occur 7-30 days after the infection. Symptoms of malaria include fever, headache, body aches, chills, and vomiting [6]. Severe malaria can develop when the infection is not treated and may result in organ failure or even death. Examples of severe malaria include cerebral malaria, severe anemia, distress, kidney failure, acidosis, and hypoglycemia [7]. Pregnant women and children aged under 5 years are the most vulnerable groups affected by malaria [8].

There are about 450 species of the *Anopheles* mosquito; however, only about 35-40 transmit malaria. The *Anopheles gambiae*, *Anopheles arabiensis*, and *Anopheles coluzzii* of the *Anopheles gambiae* species complex, and *Anopheles funestus* of the *Anopheles funestus* species are major mosquito vector species of malaria in sub-Saharan Africa [9-11].

Unlike humans, mosquitoes are ectotherms (they do not regulate their own body temperatures) [12]. Both the *Anopheles* mosquito vector and *Plasmodium* malaria parasites have highly temperature-dependent life cycles [13]. The aquatic immature *Anopheles* habitats are also strongly dependent upon rainfall and local hydrodynamics. Change in climatic factors may establish conditions favourable for the malaria parasite and vector development and reproduction leading to the occurrence of malaria in previously disease-free areas, or change the intensity of malaria transmission due to changes in biting patterns determined by seasonal factors [14]. Malaria prevalence in the African tropics has been attributed to favourable environmental conditions for larval development, and parasite maturation within the infected mosquito [15–17]. Temperature plays a major role in the life cycle of both the *Anopheles* mosquito vector and the *Plasmodium* malaria parasites. Numerous studies have shown that mosquito vectors are more active at warmer temperatures [12, 18–20]. Rainfall provides breeding sites for the mosquitoes thus increasing the number of mosquito larval habitats [17, 21]. The impact of temperature and rainfall is therefore significant in the transmission dynamics of malaria.

Mathematical models have been developed over the years to gain insight into malaria transmission dynamics and aid its control and eradication. Ross [22] developed a simple susceptible-infective-susceptible (SIS) malaria model which explained the relationship between the number of mosquitoes and the incidence of malaria in humans. It was noted that there is a threshold for the number of mosquitoes below which malaria can be sufficiently eliminated. Macdonald [23] proposed a model in which it was shown that reducing the number of mosquitoes is not a sufficient control strategy, with the assumption that the amount of infective material to which a population is exposed remains unchanged. Mosquito vector longevity was identified as the single most important variable in the force of transmission. Aron [24] and Bailey [25] considered models with acquired immunity to malaria that depends on exposure to malaria infection. Tumwiine et al. [7] considered a host-vector malaria model with delays in the development of immature mosquitoes into adult mosquitoes that transmit malaria, based on susceptible-infective-susceptible (SIS) for humans and susceptible-infective (SI) for mosquito vectors. It was established that the bigger the proportion of young mosquitoes that survives the developmental period, the higher the susceptible vector population and the lower the susceptible human host population. It was suggested that the infected human population can be reduced if the adult mosquito population is controlled. Martens et al. [3], Craig et al. [26] and Bouma et al. [27] showed that environmental and climatic factors play an important role in the geographical distribution and transmission of malaria.

The majority of the malaria models ignore the role of aquatic mosquito stages since they are not involved in the spread of malaria. However, the survival of the aquatic mosquitoes increases the adult mosquito population that is responsible for the spread of malaria. It is therefore important to include the aquatic mosquito population in

the study of the effect of temperature and rainfall on malaria transmission since they are highly affected by these factors.

The dynamic process-based mathematical models play a significant role that can provide strategic insights into the effects of seasonal factors on malaria transmission. Several studies have investigated the impact of seasonality and climate factors on malaria transmission [12, 19, 20, 28–32]. Beck-Johnson et al. [12] used a temperature-dependent, stage-structured delayed differential equation model to investigate how climate determines malaria risk and found out that adult mosquito dynamics is highly affected by temperature sensitivities and juvenile dynamics influences adult age structure. Their model combined with the Detinova curve predicts the peak temperature for potentially infectious mosquitoes at 30°C, whereas when combined with the Paaijman's curve, it predicts peak temperature at 28°C. Ngarakana-Gwasira et al. [31] assessed the impact of temperature on malaria transmission dynamics. It was shown that the malaria burden increases with the increase in temperature with an optimum temperature window of 30°C–32°C. Mukhtar et al. [30] developed and analysed a human host-mosquito vector disease-based model that included temperature and rainfall. The model was used to investigate the potential impact of climatic conditions on malaria prevalence in two climatically distinct regions of South Sudan. It was found out that malaria is more severe in the tropical region than in the hot semiarid.

In this paper, a malaria transmission model with temperature- and rainfall-dependent parameters is studied. The present model differs from the models proposed by Mukhtar et al. [30] and Bhujju et al. [33] in that it assumes that interaction coefficients between humans and mosquitoes are constants and also ignores the exposed class in the mosquito population. In addition, the stability analysis of the steady states is also carried out.

This paper is organised as follows: Section 2 presents the model formulation. In Section 3, the stability of equilibria, sensitivity, and bifurcation analysis are presented. In Section 4, numerical simulation is performed. The discussion of results is presented in Section 5.

2. Model Formulation

A human host-mosquito vector model is formulated to study the transmission dynamics of malaria using a deterministic model. The total human population $N_H(t)$ is divided into the epidemiological classes: susceptible humans $S_H(t)$, exposed humans $E_H(t)$, infectious humans $I_H(t)$, and recovered humans $R_H(t)$. Individuals are recruited into the susceptible class through birth and immigration at a constant rate Λ_H . It is assumed that there is no recruitment of infective humans and vertical transmission due to malaria. Susceptible humans enter the exposed class with the interaction coefficient β_H after being bitten by an infected female *Anopheles* mosquito. This is because the sporozoites injected by the infected female *Anopheles* mosquito have not yet developed into gametocytes in the bloodstream of the human and so cannot infect susceptible

mosquitoes. Exposed humans progress to the infectious class at the rate ρ . Infected humans either cure at the rate ν to join the recovered class or die due to malaria at a rate δ . Recovered humans lose their immunity at a rate σ . Humans in all compartments die due to natural causes at the rate μ_H .

The total mosquito population $M_T(t)$ is divided into the aquatic mosquito population $M_A(t)$ and the adult mosquito population $N_V(t)$. The aquatic mosquito population consists of the eggs, larvae, and pupae stages. The total adult female *Anopheles* mosquito population is divided into susceptible $S_V(t)$ and infective $I_V(t)$ mosquitoes. There is no recovered class for mosquitoes because they do not cure from malaria throughout their lifetime. It is assumed that mosquitoes do not die from malaria due to their short lifespan. Adult female *Anopheles* mosquitoes lay eggs at a temperature-dependent rate $L(T)$, and the aquatic mosquito population increase is constrained by the carrying capacity of the environment K [33]. Aquatic mosquitoes mature and develop into adult mosquitoes at a temperature- and rainfall-dependent rate $\lambda(T, R)$. Susceptible mosquitoes become infected with interaction coefficient β_V through biting infected humans. It is assumed that aquatic mosquitoes die at a temperature-dependent death rate $\mu_A(T)$ while adult mosquitoes die at a temperature-dependent natural death rate $\mu_V(T)$. It is also assumed that all variables presented in each compartment are differentiable with respect to time and all parameters are nonnegative except that $\delta \geq 0$.

2.1. Model Equations. The human and mosquito populations are governed by the following system of ordinary differential equations.

$$\frac{dS_H}{dt} = \Lambda_H - \beta_H S_H I_V - \mu_H S_H + \sigma R_H, \quad (1)$$

$$\frac{dE_H}{dt} = \beta_H S_H I_V - (\rho + \mu_H) E_H, \quad (2)$$

$$\frac{dI_H}{dt} = \rho E_H - (\mu_H + \nu + \delta) I_H, \quad (3)$$

$$\frac{dR_H}{dt} = \nu I_H - (\sigma + \mu_H) R_H, \quad (4)$$

$$\frac{dM_A}{dt} = L(T) \left(1 - \frac{M_A}{K}\right) (S_V + I_V) - (\lambda(T, R) + \mu_A(T)) M_A, \quad (5)$$

$$\frac{dS_V}{dt} = \lambda(T, R) M_A - \beta_V S_V I_H - \mu_V(T) S_V, \quad (6)$$

$$\frac{dI_V}{dt} = \beta_V S_V I_H - \mu_V(T) I_V, \quad (7)$$

together with

$$\begin{aligned} N_H(t) &= S_H(t) + E_H(t) + I_H(t) + R_H(t), \\ N_V(t) &= S_V(t) + I_V(t), \end{aligned} \quad (8)$$

and

$$M_T(t) = N_V(t) + M_A(t). \quad (9)$$

It can be shown that the total human population is bounded by Λ_H/μ_H and the total adult mosquito population is bounded by $(\lambda(T, R)M_A)/(\mu_V(T))$. Therefore, the solution set of the system (1)–(7) is bounded in $\mathcal{D} = \{(S_H, E_H, I_H, R_H) \in \mathbb{R}_+^4 : 0 \leq N_H \leq (\Lambda_H/\mu_H), (M_A, S_V, I_V) \in \mathbb{R}_+^3 : 0 \leq N_V \leq (\lambda(T, R)M_A)/(\mu_V(T))\}$.

Theorem 1. For system (1)–(7) if $S_H(0) > 0, E_H(0) > 0, I_H(0) > 0, R_H(0) > 0, M_A(0) > 0, S_V(0) > 0, I_V(0) > 0$, then $S_H(t) > 0, E_H(t) > 0, I_H(t) > 0, R_H(t) > 0, M_A(t) > 0, S_V(t) > 0$ and $I_V(t) > 0$ for all $t > 0$.

Proof. Define a set $\mathcal{H} = \{t > 0 : S_H(t) > 0, E_H(t) > 0, I_H(t) > 0, R_H(t) > 0, M_A(t) > 0, S_V(t) > 0, I_V(t) > 0\}$.

It is assumed by contradiction that if the set \mathcal{H} defined above is bounded, then \mathcal{H} has a supremum τ . Now, define τ as

$$\tau = \sup \{t > 0 : S_H(t) > 0, E_H(t) > 0, I_H(t) > 0, R_H(t) > 0, M_A(t) > 0, S_V(t) > 0, I_V(t) > 0, 0 \leq t \leq \tau\}. \quad (10)$$

Since $S_H(t), E_H(t), I_H(t), R_H(t), M_A(t), S_V(t)$ and $I_V(t)$ are continuous, then $\tau > 0$. If $\tau < \infty$, then it is necessary that $S_H(\tau) = 0$ or $E_H(\tau) = 0$ or $I_H(\tau) = 0$ or $R_H(\tau) = 0$ or $M_A(\tau) = 0$ or $S_V(\tau) = 0$ or $I_V(\tau) = 0$.

From equation (1),

$$\frac{dS_H}{dt} = \Lambda_H + \sigma R_H - (\beta_H I_V + \mu_H) S_H. \quad (11)$$

Let $P(t) = \exp(\mu_H t + \int_0^t \beta_H I_V(s) ds)$ and note that $P(0) = 1$ and $P(t) > 0$ for all $t > 0$.

Consider

$$\begin{aligned} \int_0^\tau \frac{d}{dt} [S_H(t) P(t)] dt &= \int_0^\tau P(t) [\Lambda_H + \sigma R_H] dt, \\ S_H(\tau) P(\tau) - S_H(0) P(0) &= \int_0^\tau (\Lambda_H + \sigma R_H(t)) P(t) dt, \\ S_H(\tau) &= P(\tau)^{-1} \left[S_H(0) + \int_0^\tau (\Lambda_H + \sigma R_H(t)) P(t) dt \right]. \end{aligned} \quad (12)$$

Therefore, $S_H(\tau) > 0$ since all parameters are positive. Applying the above reasoning to the remaining equations of system (1)–(7) shows that $E_H(\tau) > 0, I_H(\tau) > 0, R_H(\tau) > 0, M_A(\tau) > 0, S_V(\tau) > 0, I_V(\tau) > 0$; thus, $\tau = \infty$. This contradicts τ being a supremum of \mathcal{H} ; thus, \mathcal{H} is not bounded.

This confirms the positivity of solutions for all $t > 0$. The model is epidemiological and mathematically well posed.

3. Model Analysis

In this section, the equilibrium points of the system (1)–(7) are obtained and analysed for their stability. It is established that system (1)–(7) has two disease-free equilibrium points and one endemic equilibrium point.

Theorem 2. *System (1)–(7) has two disease-free equilibrium points*

$$\begin{aligned} E_{01}(S_H, E_H, I_H, R_H, M_A, S_V, I_V) \\ = \left[\frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, 0, 0 \right], E_{02}(S_H, E_H, I_H, R_H, M_A, S_V, I_V) \\ = \left[\frac{\Lambda_H}{\mu_H}, 0, 0, 0, \frac{K[L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\lambda(T, R)}, \right. \\ \left. \frac{K[L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\mu_V(T)}, 0 \right], \end{aligned} \quad (13)$$

whose existence depends on parameter θ , where

$$\theta = \frac{L(T)\lambda(T, R)}{\mu_V(T)(\lambda(T, R) + \mu_A(T))}. \quad (14)$$

Proof. Setting the right-hand side (RHS) of system (1)–(7) equal to zero gives either $M_A = 0$ or $M_A = (K[L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))])/(L(T)\lambda(T, R))$.

For $M_A = 0$, it implies that $S_V = I_V = E_H = I_H = R_H = 0$ and $S_H = \Lambda_H/\mu_H$. Therefore, there exists a disease-free equilibrium point $E_{01}(S_H, E_H, I_H, R_H, M_A, S_V, I_V) = [(\Lambda_H/\mu_H), 0, 0, 0, 0, 0, 0]$. For $M_A = (K[L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))])/(L(T)\lambda(T, R))$ with $I_H = 0$, it implies that $I_V = R_H = E_H = 0$, $S_H = \Lambda_H/\mu_H$ and $S_V = (K[L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))])/(L(T)\mu_V(T))$. Therefore, there exists a disease-free equilibrium point

$$\begin{aligned} E_{02}(S_H, E_H, I_H, R_H, M_A, S_V, I_V) \\ = \left[\frac{\Lambda_H}{\mu_H}, 0, 0, 0, \frac{K[L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\lambda(T, R)}, \right. \\ \left. \frac{K[L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\mu_V(T)}, 0 \right]. \end{aligned} \quad (15)$$

E_{02} is positive and exists only if $L(T)\lambda(T, R) > \mu_V(T)(\lambda(T, R) + \mu_A(T))$, which gives the condition of existence

of E_{02} as $\theta > 1$, where $\theta = (L(T)\lambda(T, R))/(\mu_V(T)(\lambda(T, R) + \mu_A(T)))$.

3.1. Basic Reproduction Number. According to Diekmann et al. [34], the basic reproduction number is the expected number of secondary cases produced by a typical infected individual during its entire period of infectiousness in a completely susceptible population.

The basic reproduction number is obtained using the next-generation matrix method as described by Diekmann et al. [34].

The basic reproduction number is the spectral radius of the next-generation matrix FV^{-1} , where $F = \partial \mathcal{F}_j$ and $V = \partial \mathcal{V}_j$ are computed at the disease-free equilibrium point of the system.

$$\mathcal{R}_0 = \sigma(FV^{-1}). \quad (16)$$

Consider the infected subsystem of system (1)–(7) below.

$$\begin{aligned} \frac{dE_H}{dt} &= \beta_H S_H I_V - \rho E_H - \mu_H E_H, \\ \frac{dI_H}{dt} &= \rho E_H - (\mu_H + \nu + \delta) I_H, \\ \frac{dI_V}{dt} &= \beta_V S_V I_H - \mu_V(T) I_V. \end{aligned} \quad (17)$$

The vector of new infections \mathcal{F} and the vector formed by other transfers \mathcal{V} are given by

$$\mathcal{F} = \begin{bmatrix} \beta_H S_H I_V \\ 0 \\ \beta_V S_V I_H \end{bmatrix}, \quad (18)$$

and

$$\mathcal{V} = \begin{bmatrix} (\mu_H + \rho) E_H \\ -\rho E_H + (\mu_H + \nu + \delta) I_H \\ \mu_V(T) I_V \end{bmatrix}. \quad (19)$$

For the disease-free equilibrium point E_{01} , the matrix FV^{-1} has only one eigenvalue equal to zero; thus, the basic reproduction number is zero for this case. This implies that if an infective individual is introduced into the population at the steady-state E_{01} , the disease will not spread

due to the absence of the parasite-transmitting mosquito vectors.

For the disease-free equilibrium point E_{02} , the matrices F and V are computed as follows:

$$F = \begin{bmatrix} 0 & 0 & \frac{\beta_H \Lambda_H}{\mu_H} \\ 0 & 0 & 0 \\ 0 & \frac{K\beta_V[L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\mu_V(T)} & 0 \end{bmatrix}, \quad (20)$$

and

$$V = \begin{bmatrix} \mu_H + \rho & 0 & 0 \\ -\rho & \mu_H + \nu + \delta & 0 \\ 0 & 0 & \mu_V(T) \end{bmatrix},$$

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu_H + \rho} & 0 & 0 \\ \frac{\rho}{(\rho + \mu_H)(\mu_H + \nu + \delta)} & \frac{1}{\mu_H + \nu + \delta} & 0 \\ 0 & 0 & \frac{1}{\mu_V(T)} \end{bmatrix}. \quad (21)$$

Thus, the next-generation matrix is given by

$$FV^{-1} = \begin{bmatrix} 0 & 0 & \frac{\beta_H \Lambda_H}{\mu_H \mu_V(T)} \\ 0 & 0 & 0 \\ \frac{\rho K \beta_V [L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\mu_V(T)(\mu_H + \nu + \delta)(\mu_H + \rho)} & \frac{K \beta_V [L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\mu_V(T)(\mu_H + \nu + \delta)} & 0 \end{bmatrix}. \quad (22)$$

The eigenvalues of FV^{-1} are

$$0, -\frac{1}{\mu_V(T)} \sqrt{\frac{\rho \beta_H \beta_V \Lambda_H K [L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\mu_H(\mu_H + \nu + \delta)(\mu_H + \rho)}},$$

$$-\frac{1}{\mu_V(T)} \sqrt{\frac{\rho \beta_H \beta_V \Lambda_H K [L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\mu_H(\mu_H + \nu + \delta)(\mu_H + \rho)}}. \quad (23)$$

Therefore, the basic reproduction number \mathcal{R}_0 is given by

$$\mathcal{R}_0 = \frac{1}{\mu_V(T)} \sqrt{\frac{\rho \beta_H \beta_V \Lambda_H K [L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\mu_H(\mu_H + \nu + \delta)(\rho + \mu_H)}}. \quad (24)$$

\mathcal{R}_0 exists only if $L(T)\lambda(T, R) > \mu_V(T)(\lambda(T, R) + \mu_A(T))$. Expressing \mathcal{R}_0 in terms of θ gives

$$\mathcal{R}_0 = \sqrt{\frac{\rho \beta_H \beta_V \Lambda_H K (\lambda(T, R) + \mu_A(T)) (\theta - 1)}{L(T)\mu_V^2(T)\mu_H(\mu_H + \nu + \delta)(\mu_H + \rho)}}. \quad (25)$$

\mathcal{R}_0 exists only if the term under the square root is non-negative, that is, $\theta \geq 1$; otherwise, if $\theta < 1$, there is no growth of the mosquito population, and malaria will not develop in the community since mosquito vectors are important for the spread of malaria.

3.2. Sensitivity Analysis. Malaria control and eradication strategies should target important parameters which have a high impact on the basic reproduction number. A sensitivity analysis of \mathcal{R}_0 to the various parameters is thus presented in this section. The basic reproduction number is explicitly determined by the parameters $\rho, \beta_H, \beta_V, \Lambda_H, L(T), \lambda(T, R), \mu_V(T), \mu_H, \mu_A(T), \nu$ and δ . The sensitivity indices of \mathcal{R}_0 to these parameters are computed using the approach in Chitnis et al. [35].

Definition 3. The sensitivity index of a variable u that depends continuously on a parameter p is defined as

$$\Upsilon_p^u = \frac{\partial u}{\partial p} \cdot \frac{p}{u}, \quad (26)$$

where u is a differentiable function of p .

Thus, by the definition above, the formula used to derive an expression for the sensitivity of \mathcal{R}_0 to a parameter p is given by

$$\Upsilon_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \cdot \frac{p}{\mathcal{R}_0}. \quad (27)$$

Table 1 shows the sensitivity indices of \mathcal{R}_0 to the parameters (independent of temperature and rainfall variations) determining its value. Parameter values $\Lambda_H = 0.031$, $\mu_H = 1/23178$, $\beta_H = \beta_V = 0.00021$, $\rho = 1/20$, $\nu = 1/30$, $\delta = 0.00000638$, and $K = 1000000$ are used.

The MATLAB computer software program is used in the simulation of the sensitivity of \mathcal{R}_0 with respect to temperature and rainfall since the temperature-rainfall-dependent parameters $\mu_V(T)$, $L(T)$, $\lambda(T, R)$, and $\mu_A(T)$ are given by nonlinear functions. The results are shown in Figure 1.

3.2.1. Interpretation of Sensitivity Analysis. The natural death rate μ_H and the disease-induced death rate δ of the human population are the most and least sensitive parameters, respectively. A positive value in the sensitivity index shows that if the parameter is increased when all other parameters are kept constant, the value of \mathcal{R}_0 increases, while for a negative sensitivity index, when the parameter value is increased with all other parameters kept constant, the value of \mathcal{R}_0 decreases. In Figure 1(a), rainfall is fixed at 10 mm. It is observed that \mathcal{R}_0 is most sensitive to temperatures within the ranges 17°C–20°C and 37°C–40°C. This is in agreement with Githeko et al. [16] in which the lower-end range and the upper-end range of disease transmission are established at 14°C–18°C and 35°C–40°C, respectively. The sensitivity

TABLE 1: Numerical values of sensitivity indices of \mathcal{R}_0 .

Parameter symbol	Sensitivity index
μ_H	-0.5011
β_H	0.5
β_V	0.5
Λ_H	0.5
ν	-0.4993
ρ	0.0004
δ	-0.0001

indices for temperatures between 17°C and 25°C are positive, whereas those for temperatures between 25°C and 35°C are negative. In Figure 1(b), the temperature is fixed at 25°C. It is observed that the sensitivity to rainfall reduces with more rainfall received. Indices for daily rainfall below 25 mm are positive, whereas indices for rainfall above 25 mm are negative.

3.3. Local Stability of the Disease-Free Equilibrium. The Jacobian matrix of the system (1)–(7) evaluated at the disease-free equilibrium point E_{01} is shown below.

$$\mathcal{J}_{E_{01}} = \begin{bmatrix} -\mu_H & 0 & 0 & \sigma & 0 & 0 & -\beta_H \frac{\Lambda_H}{\mu_H} \\ 0 & -(\rho + \mu_H) & 0 & 0 & 0 & 0 & \beta_H \frac{\Lambda_H}{\mu_H} \\ 0 & \rho & -(\mu_H + \nu + \delta) & 0 & 0 & 0 & 0 \\ 0 & 0 & \nu & -(\mu_H + \sigma) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\lambda(T, R) + \mu_A(T)) & L(T) & L(T) \\ 0 & 0 & 0 & 0 & \lambda(T, R) & -\mu_V(T) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_V(T) \end{bmatrix}. \quad (28)$$

The eigenvalues of $\mathcal{J}_{E_{01}}$ are $-\mu_H$, $-\mu_V(T)$, $-(\mu_H + \sigma)$, $-(\mu_H + \nu + \sigma)$, $-(\rho + \mu_H)$ and the zero points of the polynomial.

$\mathcal{Z}^2 + (\mu_V(T) + \mu_A(T) + \lambda(T, R))\mathcal{Z} + \mu_V(T)(\lambda(T, R) + \mu_A(T)) - \lambda(T, R)L(T) = 0$, where \mathcal{Z} is the eigenvalue.

The zero points of a polynomial of order two have negative real parts if and only if its coefficients and constant terms are positive. Thus, the disease-free equilibrium E_{01} is stable only if

$$\frac{\lambda(T, R)L(T)}{\mu_V(T)(\lambda(T, R) + \mu_A(T))} < 1. \quad (29)$$

Theorem 4. *The disease-free equilibrium point E_{02} of system (1)–(7) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. It has already been shown that the disease-free equilibrium E_{02} only exists if $(L(T)\lambda(T, R))/(\mu_V(T)(\lambda(T, R) + \mu_A(T))) > 1$. If this condition is satisfied, then \mathcal{R}_0 is real and positive. Thus, the basic reproduction number \mathcal{R}_0 is biologically consistent. Using the theorem by van den Driessche and Watmough [36], the disease-free equilibrium E_{02} is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

3.4. Global Stability of the Disease-Free Equilibrium. The global stability of the disease-free equilibrium is investigated

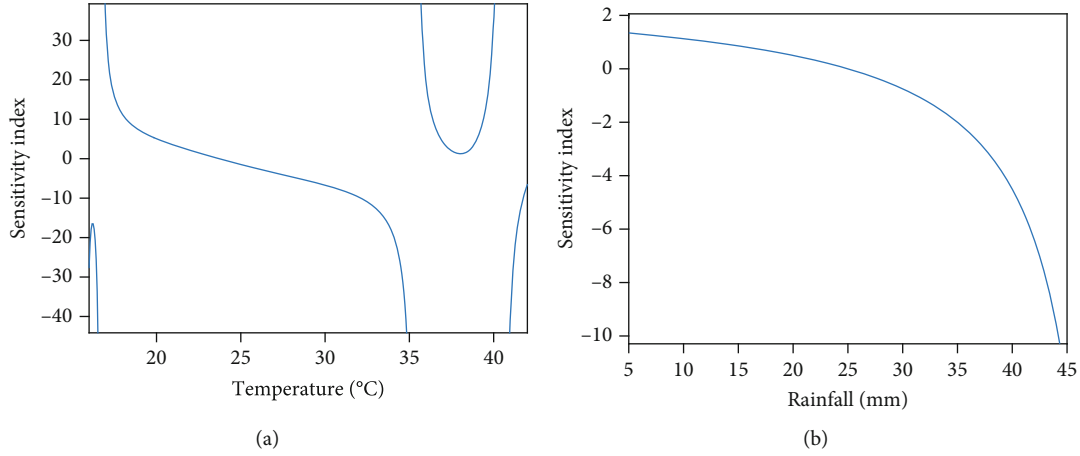


FIGURE 1: (a) Sensitivity indices of the basic reproduction number with respect to temperature. (b) Sensitivity indices of the basic reproduction number with respect to rainfall.

using a theorem by Castillo-Chavez et al. [37]. System (1)–(7) can be expressed in terms of

$$\begin{aligned} \frac{dX}{dt} &= F(X, Z) \\ \frac{dZ}{dt} &= G(X, Z), G(X, 0) = 0, \end{aligned} \quad (30)$$

where $X \in \mathbb{R}^m$ denotes the number of uninfected individuals and $Z \in \mathbb{R}^n$ denotes the number of infected individuals.

The following conditions (H1) and (H2) must be satisfied provided $\mathcal{R}_0 < 1$ to guarantee global asymptotic stability.

(H1) For $dX/dt = F(X, 0)$, X^* is globally asymptotically stable

(H2) $G(X, Z) = MZ - \widehat{G}(X, Z)$, $\widehat{G}(X, Z) \geq 0$ for $(X, Z) \in \Omega$, where $M = D_Z G(X^*, 0)$ is an M -matrix and Ω is the region where the model makes biological sense. From system (1)–(7)

$$F(X, Z) = \begin{bmatrix} \Lambda_H - \beta_H S_H I_V - \mu_H S_H + \sigma R_H \\ \nu I_H - (\sigma + \mu_H) R_H \\ L(T) \left(1 - \frac{M_A}{K} \right) (S_V + I_V) - (\lambda(T, R) + \mu_A(T)) M_A \\ \lambda(T, R) M_A - \beta_V S_V I_H - \mu_V(T) S_V \end{bmatrix}, \quad (31)$$

and

$$G(X, Z) = \begin{bmatrix} \beta_H S_H I_V - (\rho + \mu_H) E_H \\ \rho E_H - (\mu_H + \nu + \delta) I_H \\ \beta_V S_V I_H - \mu_V(T) I_V \end{bmatrix}. \quad (32)$$

To investigate condition (H1),

$$F(X, 0) = \begin{bmatrix} \Lambda_H - \mu_H S_H \\ 0 \\ L(T) \left(1 - \frac{M_A}{K} \right) S_V - (\lambda(T, R) + \mu_A(T)) M_A \\ \lambda(T, R) M_A - \mu_V(T) S_V \end{bmatrix}. \quad (33)$$

It has already been established that the threshold values for the human and mosquito populations are Λ_H/μ_H and $(\lambda(T, R) M_A)/\mu_V(T)$, respectively; thus, there is convergence in Ω . Therefore, X^* is globally asymptotically stable.

To investigate condition (H2) for the disease-free equilibrium point E_{01}

$$M = \begin{bmatrix} -(\rho + \mu_H) & 0 & a_2 \\ \rho & -(\mu_H + \nu + \delta) & 0 \\ 0 & 0 & -\mu_V(T) \end{bmatrix}, \quad (34)$$

where $a_2 = \beta_H \Lambda_H/\mu_H$

$$\widehat{G}(X, Z) = \begin{bmatrix} \beta_H I_V \left(\frac{\Lambda_H}{\mu_H} - S_H \right) \\ 0 \\ -\beta_V S_V I_H \end{bmatrix}. \quad (35)$$

Since $\widehat{G}_3(X, Z) < 0$, condition (H2) is violated. Therefore, the disease-free equilibrium point E_{01} may not be globally asymptotically stable.

For the disease-free equilibrium point E_{02}

$$M = \begin{bmatrix} -(\rho + \mu_H) & 0 & a_2 \\ \rho & -(\mu_H + \nu + \delta) & 0 \\ 0 & a_1 & -\mu_V(T) \end{bmatrix}, \quad (36)$$

where $a_1 = (\beta_V K [L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]) / (L(T)\mu_V(T))$ and $a_2 = \beta_H \Lambda_H / \mu_H$

$$\hat{G}(X, Z) = \begin{bmatrix} \beta_H I_V \left(\frac{\Lambda_H}{\mu_H} - S_H \right) \\ 0 \\ \beta_V I_H \left(\frac{K [L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]}{L(T)\mu_V(T)} - S_V \right) \end{bmatrix}. \quad (37)$$

It has already been established that the threshold value of the human population is Λ_H / μ_H . Therefore, $S_H \leq \Lambda_H / \mu_H$. Similarly, $S_V \leq (K [L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]) / (L(T)\mu_V(T))$. This shows that $\hat{G}(T, R) \geq 0$; thus, equilibrium point E_{02} is globally asymptotically stable.

3.5. Bifurcation Analysis. In this subsection, the centre manifold theorem by Castillo-Chavez and Song [38] is used to investigate the bifurcation behaviour of system (1)–(7) when the basic reproduction number $\mathcal{R}_0 = 1$.

Theorem 5. Consider a general system of ODEs with a parameter ϕ

$$\frac{dx}{dt} = f(x, \phi), f : \mathbb{R}^n \times \mathbb{R} \longrightarrow \mathbb{R}^n, f \in \mathbb{C}^2(\mathbb{R}^n \times \mathbb{R}). \quad (38)$$

Without loss of generality, it is assumed that 0 is an equilibrium for system (38) for all values of the parameter ϕ , that is

$$f(0, \phi) \equiv 0 \text{ for all } \phi. \quad (39)$$

Assume

A1. $A = D_x f(0, 0) = (\partial f_i / \partial x_j)$ is the linearisation matrix of system (38) around the equilibrium 0 with ϕ evaluated at 0. Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts.

A2. Matrix A has a nonnegative right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.

Let f_k be the k^{th} component of f and

$$\begin{aligned} a &= \sum_{k,i,j=1}^n v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0, 0), \\ b &= \sum_{k,i=1}^n v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0, 0). \end{aligned} \quad (40)$$

The local dynamics of (38) around 0 are totally determined by a and b .

- (i) $a > 0, b > 0$, when $\phi < 0$ with $|\phi| < 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \phi < 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium.
- (ii) $a < 0, b < 0$, when $\phi < 0$ with $|\phi| < 1$, 0 is unstable; when $0 < \phi < 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium.
- (iii) $a > 0, b < 0$, when $\phi < 0$ with $|\phi| < 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi < 1$, 0 is stable, and a positive unstable equilibrium.
- (iv) $a < 0, b > 0$, when ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable.

A change in notations of the variables is used such that $S_H = x_1, E_H = x_2, I_H = x_3, R_H = x_4, M_A = x_5, S_V = x_6$, and $I_V = x_7$. For $\mathcal{R}_0 = 1$, let β_H^* be a bifurcation parameter with bifurcation value β_H^* ,

$$\beta_H^* = \frac{L(T)\mu_H\mu_V(T)^2(\mu_H + \nu + \delta)(\rho + \mu_H)}{\rho\beta_V\Lambda_H K [L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))]} \quad (41)$$

System (1)–(7) becomes

$$f_1(x_1, x_2, x_3, x_4, x_5, x_6, x_7) = \Lambda_H - \beta_H x_1 x_7 - \mu_H x_1 + \sigma x_4, \quad (42)$$

$$f_2(x_1, x_2, x_3, x_4, x_5, x_6, x_7) = \beta_H x_1 x_7 - (\rho + \mu_H) x_2, \quad (43)$$

$$f_3(x_1, x_2, x_3, x_4, x_5, x_6, x_7) = \rho x_2 - (\mu_H + \nu + \delta) x_3, \quad (44)$$

$$f_4(x_1, x_2, x_3, x_4, x_5, x_6, x_7) = \nu x_3 - (\sigma + \mu_H) x_4, \quad (45)$$

$$f_5(x_1, x_2, x_3, x_4, x_5, x_6, x_7) = L(T) \left(1 - \frac{x_5}{K} \right) (x_6 + x_7) - (\lambda(T, R) + \mu_A(T)) x_5, \quad (46)$$

$$f_6(x_1, x_2, x_3, x_4, x_5, x_6, x_7) = \lambda(T, R) x_5 - \beta_V x_6 x_3 - \mu_V(T) x_6, \quad (47)$$

$$f_7(x_1, x_2, x_3, x_4, x_5, x_6, x_7) = \beta_V x_6 x_3 - \mu_V(T) x_7. \quad (48)$$

$E_{02}(x_1, x_2, x_3, x_4, x_5, x_6, x_7) = [(\Lambda_H / \mu_H), 0, 0, 0, (Kd / (L(T)\lambda(T, R))), (Kd / (L(T)\mu_V(T))), 0]$ is a disease free equilibrium point for system (42)–(48), where $d = L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))$.

The Jacobian matrix of system (42)–(48) evaluated at E_{02} is given by

$$\mathcal{J}_{E_{02}} = \begin{bmatrix} -J_1 & 0 & 0 & \sigma & 0 & 0 & -\frac{\beta_H \Lambda_H}{\mu_H} \\ 0 & -J_2 & 0 & 0 & 0 & 0 & \frac{\beta_H \Lambda_H}{\mu_H} \\ 0 & \rho & -J_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & \nu & -J_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -J_5 & \frac{\mu_V(T)(\lambda(T, R) + \mu_A(T))}{\lambda(T, R)} & \frac{\mu_V(T)(\lambda(T, R) + \mu_A(T))}{\lambda(T, R)} \\ 0 & 0 & -\frac{\beta_V K d}{L(T)\mu_V(T)} & 0 & \lambda(T, R) & -J_6 & 0 \\ 0 & 0 & \frac{\beta_V K d}{L(T)\mu_V(T)} & 0 & 0 & 0 & -J_7 \end{bmatrix} \quad (49)$$

where $J_1 = \mu_H$, $J_2 = (\rho + \mu_H)$, $J_3 = (\mu_H + \nu + \delta)$, $J_4 = (\mu_H + \sigma)$, $J_5 = (L(T)\lambda(T, R))/(\mu_V(T))$, and $J_6 = J_7 = \mu_V(T)$.

The right eigenvector $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T$, corresponding to the zero eigenvalue, is computed using $\mathcal{J}_{E_{02}} w = 0$ which yields

$$\begin{aligned} -J_1 w_1 + \sigma w_4 - \frac{\beta_H \Lambda_H}{\mu_H} w_7 &= 0, \\ -J_2 w_2 + \frac{\beta_H \Lambda_H}{\mu_H} w_7 &= 0, \\ \rho w_2 - J_3 w_3 &= 0, \\ \nu w_3 - J_4 w_4 &= 0, \\ -J_5 w_5 + \frac{\mu_V(T)(\lambda(T, R) + \mu_A(T))}{\lambda(T, R)} w_6 + \frac{\mu_V(T)(\lambda(T, R) + \mu_A(T))}{\lambda(T, R)} w_7 &= 0, \\ -\frac{\beta_V K d}{L(T)\mu_V(T)} w_3 + \lambda(T, R) w_5 - J_6 w_6 &= 0, \\ \frac{\beta_V K d}{L(T)\mu_V(T)} w_3 - J_7 w_7 &= 0. \end{aligned} \quad (50)$$

Setting $w_7 = 1$ gives,

$$\begin{aligned} w_3 &= \frac{L(T)\mu_V(T)^2}{\beta_V K d}, \\ w_4 &= \frac{L(T)\nu\mu_V(T)^2}{\beta_V K d(\mu_H + \sigma)}, \\ w_2 &= \frac{L(T)\mu_V(T)^2(\mu_H + \nu + \delta)}{\rho\beta_V K d}, \\ w_1 &= \frac{1}{\mu_H} \left(\frac{L(T)\nu\sigma\mu_V(T)^2}{\beta_V K d(\mu_H + \sigma)} - \frac{\beta_H \Lambda_H}{\mu_H} \right), \end{aligned} \quad (51)$$

and

$$\begin{aligned} -\frac{L(T)\lambda(T, R)^2}{\mu_V(T)} w_5 + \mu_V(T)(\lambda(T, R) + \mu_A(T)) w_6 \\ = -\mu_V(T)(\lambda(T, R) + \mu_A(T)), \end{aligned} \quad (52)$$

$$\lambda(T, R) w_5 - \mu_V(T) w_6 = \mu_V(T). \quad (53)$$

Solving equations (52) and (53) simultaneously gives

$$\begin{aligned} w_5 &= 0, \\ w_6 &= -1. \end{aligned} \quad (54)$$

The left eigenvector $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)$, corresponding to the zero eigenvalue, is computed using $v \mathcal{J}_{E_{02}} = 0$ which yields

$$\begin{aligned} -J_1 v_1 &= 0, \\ -J_2 v_2 + \rho v_3 &= 0, \\ -J_3 v_3 + \nu v_4 - \frac{\beta_V K d}{L(T)\mu_V(T)} v_6 + \frac{\beta_V K d}{L(T)\mu_V(T)} v_7 &= 0, \\ \sigma v_1 - J_4 v_4 &= 0, \\ \frac{\mu_V(T)(\lambda(T, R) + \mu_A(T))}{\lambda(T, R)} v_5 - J_6 v_6 &= 0, \\ \frac{\beta_H \Lambda_H}{\mu_H} v_1 + \frac{\beta_H \Lambda_H}{\mu_H} v_2 + \frac{\mu_V(T)(\lambda(T, R) + \mu_A(T))}{\lambda(T, R)} v_5 - J_7 v_7 &= 0. \end{aligned} \quad (55)$$

It follows that $v_1 = v_4 = 0$, setting $v_3 = v_6 = 1$ gives

$$\begin{aligned} v_2 &= \frac{\rho}{\rho + \mu_H}, \\ v_5 &= \frac{\lambda(T, R)}{(\lambda(T, R) + \mu_A(T))}, \\ v_7 &= \frac{L(T)\mu_V(T)(\mu_H + v + \delta) + \beta_V Kd}{\beta_V Kd}. \end{aligned} \quad (56)$$

Since $v_1 = v_4 = 0$, the values of a and b are obtained from

$$\begin{aligned} a &= \sum_{i,j=1}^7 v_2 w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j} + \sum_{i,j=1}^7 v_3 w_i w_j \frac{\partial^2 f_3}{\partial x_i \partial x_j} \\ &\quad + \sum_{i,j=1}^7 v_5 w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j} + \sum_{i,j=1}^7 v_6 w_i w_j \frac{\partial^2 f_6}{\partial x_i \partial x_j} \\ &\quad + \sum_{i,j=1}^7 v_7 w_i w_j \frac{\partial^2 f_7}{\partial x_i \partial x_j}, \\ b &= \sum_{k,i=1}^7 v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_H^*}, \end{aligned} \quad (57)$$

where the partial derivatives computed at E_{02} are

$$\begin{aligned} \frac{\partial^2 f_2}{\partial x_1 \partial x_7} &= \beta_H, \\ \frac{\partial^2 f_5}{\partial x_5 \partial x_6} &= -\frac{L(T)}{K}, \\ \frac{\partial^2 f_5}{\partial x_5 \partial x_7} &= -\frac{L(T)}{K}, \\ \frac{\partial^2 f_6}{\partial x_3 \partial x_6} &= -\beta_V, \\ \frac{\partial^2 f_7}{\partial x_3 \partial x_6} &= \beta_V, \\ \frac{\partial^2 f_2}{\partial x_7 \partial \beta_H^*} &= \frac{\Lambda_H}{\mu_H}. \end{aligned} \quad (58)$$

It follows that

$$\begin{aligned} a &= \frac{2\rho\beta_H}{\mu_H(\rho + \mu_H)} \left(\frac{L(T)v\sigma\mu_V(T)^2}{\beta_V Kd(\mu_H + \sigma)} - \frac{\beta_H \Lambda_H}{\mu_H} \right) + \frac{2L(T)\mu_V(T)^2}{Kd} \\ &\quad - \frac{2L(T)\mu_V(T)^2(\beta_V Kd + L(T)\mu_V(T)(\mu_H + v + \delta))}{\beta_V K^2 d^2}, \\ b &= \frac{\rho\beta_H \Lambda_H}{\mu_H(\rho + \mu_H)} > 0. \end{aligned} \quad (59)$$

Since $b > 0$, the model undergoes a backward bifurcation if $a > 0$, that is

$$\begin{aligned} &\frac{2\rho\beta_H L(T)v\sigma\mu_V(T)^2}{\mu_H \beta_V Kd(\mu_H + \rho)(\mu_H + \sigma)} + \frac{2L(T)\mu_V(T)^2}{Kd} \\ &> \frac{2\rho\beta_H^2 \Lambda_H}{\mu_H^2(\mu_H + \rho)} + \frac{2L(T)\mu_V(T)^2 \alpha}{\beta_V K^2 d^2}, \end{aligned} \quad (60)$$

where $\alpha = (\beta_V Kd + L(T)\mu_V(T)(\mu_H + v + \delta))$.

Remark 6. Existence of a backward bifurcation when $a > 0$ means that there is a possibility of coexistence of an endemic equilibrium and the disease-free equilibrium when $\mathcal{R}_0 < 1$. In this case, the strategy of reducing the basic reproduction number to a value less than unity would not be sufficient for the eradication of malaria.

3.6. Existence and Stability of the Endemic Equilibrium

Theorem 7. System (1)–(7) has a unique endemic equilibrium when $\mathcal{R}_0 > 1$.

Proof. Setting the right-hand side of the equations in system (1)–(7) to zero shows that either $I_H = 0$ or $I_H = (K\rho\beta_H \beta_V \Lambda_H(\sigma + \mu_H)d - L(T)\mu_H\mu_V(T)^2c)/(Kd\beta_H\beta_V(c - \rho\sigma v) + L(T)\beta_V\mu_H\mu_V(T)c)$, where $d = L(T)\lambda(T, R) - \mu_V(T)(\lambda(T, R) + \mu_A(T))$ and $c = (\mu_H + v + \delta)(\sigma + \mu_H)(\rho + \mu_H)$. $I_H = 0$ corresponds to the disease-free equilibrium.

Expressing I_H above in terms of \mathcal{R}_0 using $d = (\mathcal{R}_0^2 \mu_V^2 \mu_H L(T)c)/(K\rho\beta_H\beta_V\Lambda_H(\sigma + \mu_H))$ gives

$$I_H = \frac{\rho\Lambda_H\mu_V(T)(\sigma + \mu_H)(\mathcal{R}_0^2 - 1)}{\mathcal{R}_0^2 \mu_V(T)(c - \rho\sigma v) + \beta_V \rho\Lambda_H(\sigma + \mu_H)}. \quad (61)$$

Thus, there exists a unique endemic equilibrium point $E_1 = (S_H^*, E_H^*, I_H^*, R_H^*, M_A^*, S_V^*, I_V^*)$ only if $\mathcal{R}_0^2 > 1$, that is, $\mathcal{R}_0 > 1$ since $c > \rho\sigma v$. The endemic equilibrium is given by

$$\begin{aligned} S_H^* &= \frac{L(T)\mu_V(T)[\Lambda_H(\sigma + \mu_H) + \sigma v I_H^*](\beta_V I_H^* + \mu_V(T))}{(\sigma + \mu_H)[(K\beta_H d + L(T)\mu_H\mu_V(T))\beta_V I_H^* + L(T)\mu_H\mu_V(T)^2]}, \\ E_H^* &= \frac{K\beta_H\beta_V d I_H^*[\Lambda_H(\sigma + \mu_H) + \sigma v I_H^*]}{(\rho + \mu_H)(\sigma + \mu_H)[(K\beta_H d + L(T)\mu_H\mu_V(T))\beta_V I_H^* + L(T)\mu_H\mu_V(T)^2]}, \\ I_H^* &= \frac{\rho\Lambda_H\mu_V(T)(\sigma + \mu_H)(\mathcal{R}_0^2 - 1)}{\mathcal{R}_0^2 \mu_V(T)(c - \rho\sigma v) + \beta_V \rho\Lambda_H(\sigma + \mu_H)}, \\ R_H^* &= \frac{v I_H^*}{\sigma + \mu_H}, \\ M_A^* &= \frac{Kd}{L(T)\lambda(T, R)}, \\ S_V^* &= \frac{Kd}{L(T)(\beta_V I_H^* + \mu_V(T))}, \\ I_V^* &= \frac{K\beta_V d I_H^*}{L(T)\mu_V(T)(\beta_V I_H^* + \mu_V(T))}. \end{aligned} \quad (62)$$

The Jacobian matrix of system of equations (1)–(7) evaluated at the endemic equilibrium point E_1 is given by

$$\mathcal{J}_{E_1} = \begin{bmatrix} -e_1 & 0 & 0 & \sigma & 0 & 0 & -e_2 \\ e_3 & -e_4 & 0 & 0 & 0 & 0 & e_2 \\ 0 & \rho & -e_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & \nu & -e_6 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -e_7 & e_8 & e_8 \\ 0 & 0 & -e_9 & 0 & e_{10} & -e_{11} & 0 \\ 0 & 0 & e_9 & 0 & 0 & e_{12} & -e_{13} \end{bmatrix}, \quad (63)$$

where $e_1 = \beta_H I_H^* + \mu_H$, $e_2 = \beta_H S_H^*$, $e_3 = \beta_H I_V^*$, $e_4 = \rho + \mu_H$, $e_5 = \mu_H + \nu + \delta$, $e_6 = \sigma + \mu_H$, $e_7 = ((L(T)(S_V^* + I_V^*))/K) - (\lambda(T, R) + \mu_A(T))$, $e_8 = L(T)(1 - (M_A^*/K))$, $e_9 = \beta_V S_V^*$, $e_{10} = \lambda(T, R)$, $e_{11} = \beta_V I_H^* + \mu_V$, $e_{12} = \beta_V I_H^*$, and $e_{13} = \mu_V(T)$.

The characteristic polynomial of the Jacobian matrix \mathcal{J}_{E_1} is obtained as

$$\mathcal{Z}^7 + a_1 \mathcal{Z}^6 + a_2 \mathcal{Z}^5 + a_3 \mathcal{Z}^4 + a_4 \mathcal{Z}^3 + a_5 \mathcal{Z}^2 + a_6 \mathcal{Z} + a_7 = 0, \quad (64)$$

where

$$a_1 = e_1 + e_4 + e_5 + e_6 + e_7 + e_{11} + e_{13},$$

$$a_2 = e_1(e_4 + e_5 + e_6 + e_7 + e_{11} + e_{13}) + e_4(e_5 + e_6 + e_7 + e_{11}) + e_5(e_6 + e_7 + e_{11} + e_{13}) + e_6(e_7 + e_{11} + e_{13}) + e_7(e_{11} + e_{13}) + e_{13}(e_4 + e_7) - e_8 e_{10},$$

$$a_3 = e_1 e_4 (e_5 + e_6 + e_7 + e_{11} + e_{13}) + e_1 e_5 (e_7 + e_{11} + e_{13}) + e_1 e_6 (e_7 + e_{11} + e_{13}) + e_4 e_5 (e_6 + e_7 + e_{11} + e_{13}) + e_4 e_6 (e_7 + e_{11} + e_{13}) + e_5 e_6 (e_7 + e_{11}) + e_7 e_{11} (e_1 + e_4 + e_5 + e_6 + e_7) - e_8 e_{10} (e_1 + e_4 + e_5 + e_{12}) + e_{11} e_{13} (e_1 + e_4 + e_5 + e_6 + e_7) - e_8 e_{13} (e_4 + e_5 + e_6) - e_2 e_9 \rho + e_1 e_7 e_{13} - e_8 e_{10} e_{13},$$

$$a_4 = e_2 e_9 \rho (e_3 + e_6 - e_7 - e_{11} + e_{12} - e_1) + e_1 e_6 e_{13} (e_4 + e_5 + e_7 + e_{11}) + e_1 e_4 e_{13} (e_5 + e_7) + e_1 e_7 (e_5 e_{13} + e_4 e_{11} + e_{11} e_{13} + e_4 e_6) + e_4 e_5 (e_6 e_{13} + e_7 e_{13} + e_{11} e_{13} + e_1 e_7 + e_1 e_{11} + e_6 e_{11} - e_8 e_{10} + e_6 e_7) + e_4 e_7 (e_6 e_{13} + e_{11} e_{13} + e_1 e_6 + e_6 e_{11}) + e_5 e_{13} (e_6 e_7 + e_6 e_{11} + e_8 e_{10} + e_7 e_{11} + e_1 e_{11}) - e_1 e_{10} (e_8 e_{12} + e_8 e_{13}) + e_6 e_8 + e_5 e_8 - e_6 e_{11} (e_1 e_7 + e_5 e_7 - e_4 e_8 + e_1 e_4 + e_1 e_5) + e_8 e_{10} (e_4 e_{12} - e_4 e_{13} - e_5 e_{12} - e_6 e_{12} - e_4 e_{13} - e_5 e_6 - e_1 e_4) + e_6 e_{11} e_{13} (e_4 + e_7) - e_3 \nu \rho \sigma + e_1 e_5 e_7 e_{11},$$

$$a_5 = e_1 e_4 e_5 (e_6 e_7 + e_6 e_{11} + e_6 e_{13} + e_7 e_{11} - e_8 e_{10} + e_7 e_{13} + e_{11} e_{13}) + e_1 e_5 e_6 (e_7 e_{11} - e_8 e_{10} + e_7 e_{13} + e_{11} e_{13}) + e_4 e_6 e_8 (e_{10} e_{13} - e_5 e_{10}) + e_2 e_9 \rho (e_3 e_6 - e_1 e_6 + e_1 e_7 - e_1 e_{11} + e_3 e_{11} - e_6 e_{11} - e_7 e_{11} - e_8 e_{10} + e_1 e_{12} - e_3 e_{12} + e_6 e_{12} + e_7 e_{12}) + e_4 e_5 e_6 (e_7 e_{11} + e_7 e_{13}) - e_8 e_{10} (e_1 e_4 e_{12} + e_1 e_5 e_{12} + e_1 e_5 e_{13} + e_1 e_6 e_{13} + e_1 e_6 e_{12} + e_4 e_5 e_{12} + e_1 e_4 e_{13} + e_5 e_6 e_{13}) + e_7 e_{11} (e_1 e_4 e_{13} + e_4 e_6 e_{13}) + e_{11} e_{13} (e_4 e_5 e_6 + e_4 e_5 e_7 + e_1 e_6 e_7 + e_1 e_5 e_7) - e_3 \nu \rho \sigma (e_7 + e_{11} + e_{13}),$$

$$a_6 = e_1 e_4 e_5 (e_6 e_7 e_{11} - e_6 e_8 e_{10} + e_6 e_7 e_{13} + e_6 e_{11} e_{13} - e_8 e_{10} e_{12} - e_8 e_{10} e_{13} + e_7 e_{11} e_{13}) - e_2 e_9 \rho (e_3 e_6 e_7 - e_1 e_6 e_{11} - e_1 e_7 e_{11} + e_1 e_8 e_{10} + e_3 e_6 e_{11} + e_3 e_7 e_{11} - e_3 e_8 e_{10} - e_6 e_7 e_{11} + e_6 e_8 e_{10} + e_1 e_6 e_{12} + e_1 e_7 e_{12} - e_3 e_6 e_{12} - e_3 e_7 e_{12}) + e_1 e_4 e_6 (e_7 e_{11} e_{13} - e_8 e_{10} e_{12} - e_8 e_{10} e_{13}) - e_1 e_5 e_6 (e_8 e_{10} e_{12} - e_7 e_{11} e_{13} + e_8 e_{10} e_{13}) - e_4 e_5 e_6 (e_8 e_{10} e_{12} - e_7 e_{11} e_{13}) - e_4 e_5 e_7 e_8 e_{10} e_{13} - e_1 e_4 e_6 e_7 e_9 \rho - \nu \rho \sigma (e_3 e_7 e_{11} - e_3 e_8 e_{10} + e_3 e_7 e_{13} + e_3 e_{11} e_{13}),$$

$$a_7 = e_1 e_2 e_6 e_9 \rho (e_8 e_{10} - e_7 e_{11}) + e_2 e_3 e_6 e_9 e_{12} \rho (e_7 e_{11} - e_8 e_{10}) + e_1 e_4 e_5 e_6 (e_7 e_{11} e_{13} - e_8 e_{10} e_{12}) + e_2 e_6 e_7 e_9 e_{12} \rho (e_1 - e_3) + e_3 \rho \sigma (e_8 e_{10} e_{12} \nu - e_7 e_{11} e_{13} \nu + e_8 e_{10} e_{13}) - e_1 e_4 e_5 e_6 e_8 e_{10} e_{13}. \quad (65)$$

By the Routh-Hurwitz criteria, the endemic equilibrium point E_1 is locally stable provided

$$a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0, a_5 > 0, a_6 > 0, a_7 > 0,$$

$$a_1 a_2 > a_3,$$

$$a_1 a_2 a_3 - a_1^2 a_4 - a_3^2 > 0,$$

$$2a_1 a_4 a_5 - a_1^2 a_4^2 - a_1 a_2^2 a_5 + a_2 a_3 a_5 - a_3^2 a_4 - a_5^2 > 0,$$

$$2a_1 a_2 a_5 a_6 + a_1^2 a_3 a_4 a_6 - a_1^3 a_6^3 - a_1^2 a_4 a_5 - a_1 a_2^2 a_5^2 - a_1 a_2 a_3^2 a_6 + a_1 a_2 a_3 a_4 a_5 - 3a_1 a_3 a_5 a_6 + 2a_1 a_4 a_5^2 + a_2 a_3 a_5^2 + a_3 a_6 - a_3 a_4 a_5 - a_5^3 > 0, \quad (66)$$

and

$$a_1^2 a_3 a_4 a_6^2 - a_1^3 a_6^3 - 3a_1^2 a_2 a_4 a_6 a_7 + 2a_1^2 a_2 a_5 a_6^2 + a_1^2 a_4^3 a_7 - a_1^2 a_4^2 a_5 a_6 + 3a_1^2 a_6^2 a_7 - a_1 a_3^2 a_7^2 + 2a_1 a_2^2 a_3 a_6 a_7 + a_1 a_2^2 a_4 a_5 a_7 - a_1 a_2^2 a_5^2 a_6 - a_1 a_2 a_3^2 a_6^2 - a_1 a_2 a_3 a_4^2 a_7 + a_1 a_2 a_3 a_4 a_5 a_6 + 3a_1 a_2 a_4 a_7^2 - a_1 a_2 a_5 a_6 a_7 + a_1 a_3 a_4 a_6 a_7 - 3a_1 a_3 a_5 a_6^2 - 2a_1 a_4^2 a_5 a_7 + 2a_1 a_4 a_5^2 a_6 - 3a_1 a_6 a_7^2 + a_2^2 a_3 a_6 a_7 - a_2 a_3 a_4 a_5 a_7 + a_2 a_3 a_5^2 a_6 - a_2 a_5 a_7^2 + a_3^2 a_6^2 + a_3^2 a_4^2 a_7 - a_3^2 a_4 a_5 a_6 - 2a_3 a_4 a_7^2 + 3a_3 a_5 a_6 a_7 + a_4 a_5^2 a_7 - a_5^3 a_6 + a_7^3 > 0. \quad (67)$$

The stability of the endemic equilibrium E_1 is demonstrated in the numerical simulations (see Section 4).

Theorem 8. *The endemic equilibrium point E_1 for system (1)–(7) is globally asymptotically stable in \mathcal{D} if $\mathcal{R}_0 > 1$.*

Proof. Consider a Lyapunov function of the form

$$\begin{aligned} L(t) = & \left(S_H - S_H^* - S_H^* \ln \left(\frac{S_H}{S_H^*} \right) \right) + \left(E_H - E_H^* - E_H^* \ln \left(\frac{E_H}{E_H^*} \right) \right) \\ & + \left(I_H - I_H^* - I_H^* \ln \left(\frac{I_H}{I_H^*} \right) \right) + \left(R_H - R_H^* - R_H^* \ln \left(\frac{R_H}{R_H^*} \right) \right) \\ & + \left(M_A - M_A^* - M_A^* \ln \left(\frac{M_A}{M_A^*} \right) \right) + \left(S_V - S_V^* - S_V^* \ln \left(\frac{S_V}{S_V^*} \right) \right) \\ & + \left(I_V - I_V^* - I_V^* \ln \left(\frac{I_V}{I_V^*} \right) \right). \end{aligned} \quad (68)$$

Differentiating $L(t)$ with respect to time gives

$$\begin{aligned} \frac{dL}{dt} = & \left(\frac{S_H - S_H^*}{S_H} \right) \frac{dS_H}{dt} + \left(\frac{E_H - E_H^*}{E_H} \right) \frac{dE_H}{dt} + \left(\frac{I_H - I_H^*}{I_H} \right) \frac{dI_H}{dt} \\ & + \left(\frac{R_H - R_H^*}{R_H} \right) \frac{dR_H}{dt} + \left(\frac{M_A - M_A^*}{M_A} \right) \frac{dM_A}{dt} \\ & + \left(\frac{S_V - S_V^*}{S_V} \right) \frac{dS_V}{dt} + \left(\frac{I_V - I_V^*}{I_V} \right) \frac{dI_V}{dt}, \end{aligned}$$

$$\begin{aligned} \frac{dL}{dt} = & \left(\frac{S_H - S_H^*}{S_H} \right) [\Lambda_H - \beta_H S_H I_V - \mu_H S_H + \sigma R_H] \\ & + \left(\frac{E_H - E_H^*}{E_H} \right) [\beta_H S_H I_V - (\rho + \mu_H) E_H] \\ & + \left(\frac{I_H - I_H^*}{I_H} \right) [\rho E_H - (\mu_H + \nu + \delta) I_H] \\ & + \left(\frac{R_H - R_H^*}{R_H} \right) [\nu I_H - (\sigma + \mu_H) R_H] + \left(\frac{M_A - M_A^*}{M_A} \right) \\ & \cdot \left[L(T) S_V + L(T) I_V - \frac{L(T) S_V M_A}{K} \right. \\ & \left. - \frac{L(T) I_V M_A}{K} - (\lambda(T, R) + \mu_A(T)) M_A \right] \\ & + \left(\frac{S_V - S_V^*}{S_V} \right) [\lambda(T, R) M_A - \beta_V I_H S_V - \mu_V(T) S_V] \\ & + \left(\frac{I_V - I_V^*}{I_V} \right) [\beta_V S_V I_H - \mu_V(T) I_V], \end{aligned}$$

$$\begin{aligned} \frac{dL}{dt} = & \left(\frac{S_H - S_H^*}{S_H} \right) [\Lambda_H - \beta_H (S_H - S_H^*) (I_V - I_V^*) \\ & - \mu_H (S_H - S_H^*) + \sigma (R_H - R_H^*)] + \left(\frac{E_H - E_H^*}{E_H} \right) \\ & \cdot [\beta_H (S_H - S_H^*) (I_V - I_V^*) - (\rho + \mu_H) (E_H - E_H^*)] \\ & + \left(\frac{I_H - I_H^*}{I_H} \right) [\rho (E_H - E_H^*) - (\mu_H + \nu + \delta) (I_H - I_H^*)] \end{aligned}$$

$$\begin{aligned} & + \left(\frac{R_H - R_H^*}{R_H} \right) [\nu (I_H - I_H^*) - (\sigma + \mu_H) (R_H - R_H^*)] \\ & + \left(\frac{M_A - M_A^*}{M_A} \right) [L(T) (S_V - S_V^*) + L(T) (I_V - I_V^*) \\ & - \frac{L(T) (S_V - S_V^*) (M_A - M_A^*)}{K} - \frac{L(T) (I_V - I_V^*) (M_A - M_A^*)}{K} \\ & - (\lambda(T, R) + \mu_A(T)) (M_A - M_A^*)] + \left(\frac{S_V - S_V^*}{S_V} \right) \\ & \cdot [\lambda(T, R) (M_A - M_A^*) - \beta_V (I_H - I_H^*) (S_V - S_V^*) \\ & - \mu_V(T) (S_V - S_V^*)] + \left(\frac{I_V - I_V^*}{I_V} \right) \\ & \cdot [\beta_V (S_V - S_V^*) (I_H - I_H^*) - \mu_V(T) I_V]. \\ \frac{dL}{dt} = & \frac{\Lambda_H (S_H - S_H^*)}{S_H} - \frac{\beta_H I_V (S_H - S_H^*)^2}{S_H} + \frac{\beta_H I_V^* (S_H - S_H^*)^2}{S_H} \\ & - \frac{\mu_H (S_H - S_H^*)^2}{S_H} + \frac{\sigma R_H (S_H - S_H^*)}{S_H} - \frac{\sigma R_H^* (S_H - S_H^*)}{S_H} \\ & + \frac{\beta_H S_H I_V (E_H - E_H^*)}{E_H} - \frac{\beta_H S_H I_V^* (E_H - E_H^*)}{E_H} \\ & - \frac{\beta_H S_H^* I_V (E_H - E_H^*)}{E_H} + \frac{\beta_H S_H^* I_V^* (E_H - E_H^*)}{E_H} \\ & - \frac{(\rho + \mu_H) (E_H - E_H^*)^2}{E_H} + \frac{\rho E_H (I_H - I_H^*)}{E_H} - \frac{\rho E_H^* (I_H - I_H^*)}{E_H} \\ & - \frac{(\mu_H + \nu + \delta) (I_H - I_H^*)^2}{I_H} + \frac{\nu I_H (R_H - R_H^*)}{R_H} \\ & - \frac{\nu I_H^* (R_H - R_H^*)}{R_H} - \frac{(\sigma + \mu_H) (R_H - R_H^*)^2}{R_H} \\ & + \frac{L(T) S_V (M_A - M_A^*)}{M_A} - \frac{L(T) S_V^* (M_A - M_A^*)}{M_A} \\ & + \frac{L(T) I_V (M_A - M_A^*)}{M_A} - \frac{L(T) I_V^* (M_A - M_A^*)}{M_A} \\ & - \frac{L(T) S_V (M_A - M_A^*)^2}{K M_A} + \frac{L(T) S_V^* (M_A - M_A^*)^2}{K M_A} \\ & - \frac{L(T) I_V (M_A - M_A^*)^2}{K M_A} + \frac{L(T) I_V^* (M_A - M_A^*)^2}{K M_A} \\ & - \frac{(\lambda(T, R) + \mu_A(T)) (M_A - M_A^*)^2}{M_A} \\ & + \frac{\lambda(T, R) M_A (S_V - S_V^*)}{S_V} - \frac{\lambda(T, R) M_A^* (S_V - S_V^*)}{S_V} \\ & - \frac{\beta_V I_H (S_V - S_V^*)^2}{S_V} + \frac{\beta_V I_H^* (S_V - S_V^*)^2}{S_V} - \frac{\mu_V(T) (S_V - S_V^*)^2}{S_V} \\ & + \frac{\beta_V S_V I_H (I_V - I_V^*)}{I_V} - \frac{\beta_V S_V I_H^* (I_V - I_V^*)}{I_V} - \frac{\beta_V S_V^* I_H (I_V - I_V^*)}{I_V} \\ & + \frac{\beta_V S_V^* I_H^* (I_V - I_V^*)}{I_V} - \frac{\mu_V(T) (I_V - I_V^*)^2}{I_V}. \end{aligned} \quad (69)$$

Collecting positive terms together and negative parts together gives

$$\frac{dL}{dt} = A - B, \quad (70)$$

TABLE 2: Parameter values for the human and mosquito populations.

Parameter	Symbol	Value	Unit	Source
Recruitment rate of humans	Λ_H	0.031	day ⁻¹	[40]
Natural death rate of humans	μ_H	1/23178	day ⁻¹	[41]
Interaction coefficient between susceptible humans and infected mosquitoes	β_H	0.00021	-	[42]
Rate of loss of immunity	σ	1/(20 × 365)	day ⁻¹	[43]
Progression rate from exposed class	ρ	1/20	day ⁻¹	[43]
Recovery rate	ν	1/30	day ⁻¹	[43]
Disease-induced death rate	δ	0.00000638	day ⁻¹	[33]
Egg deposition rate	$L(T)$	$-0.153T^2 + 8.61T - 97.7$	day ⁻¹	[30]
Aquatic mosquito death rate	$\mu_A(T)$	$1.0257 - 0.094T + 0.0025T^2$	day ⁻¹	[30]
Interaction coefficient between susceptible mosquitoes and infected humans	β_V	0.00021	-	[42]
Adult mosquito death rate	$\mu_V(T)$	$-\ln(0.522 - 0.000828T^2 + 0.0367T)$	day ⁻¹	[30]
Carrying capacity of the environment	K	1000000	Dimensionless	[30]

where

$$\begin{aligned}
A = & \frac{\Lambda_H(S_H - S_H^*)}{S_H} + \frac{\beta_H I_V^*(S_H - S_H^*)^2}{S_H} + \frac{\sigma R_H(S_H - S_H^*)}{S_H} \\
& + \frac{\beta_H I_V S_H(E_H - E_H^*)}{E_H} + \frac{\beta_H I_V^* S_H^*(E_H - E_H^*)}{E_H} \\
& + \frac{\rho E_H(I_H - I_H^*)}{I_H} + \frac{\nu I_H(R_H - R_H^*)}{R_H} + \frac{L(T)S_V(M_A - M_A^*)}{M_A} \\
& + \frac{L(T)I_V(M_A - M_A^*)}{M_A} + \frac{L(T)S_V^*(M_A - M_A^*)^2}{KM_A} \\
& + \frac{L(T)I_V^*(M_A - M_A^*)^2}{KM_A} + \frac{\lambda(T, R)M_A(S_V - S_V^*)}{S_V} \\
& + \frac{\beta_V I_H^*(S_V - S_V^*)^2}{S_V} + \frac{\beta_V S_V I_H(I_V - I_V^*)}{I_V} + \frac{\beta_V S_V^* I_H^*(I_V - I_V^*)}{I_V},
\end{aligned} \tag{71}$$

and

$$\begin{aligned}
B = & \frac{\beta_H I_V(S_H - S_H^*)^2}{S_H} + \frac{\mu_H(S_H - S_H^*)^2}{S_H} + \frac{\sigma R_H^*(S_H - S_H^*)}{S_H} \\
& + \frac{\beta_H S_H I_V^*(E_H - E_H^*)}{E_H} + \frac{\beta_H S_H^* I_V(E_H - E_H^*)}{E_H} \\
& + \frac{(\rho + \mu_H)(E_H - E_H^*)^2}{E_H} + \frac{\rho E_H^*(I_H - I_H^*)}{I_H} \\
& + \frac{(\mu_H + \nu + \delta)(I_H - I_H^*)^2}{I_H} + \frac{\nu I_H^*(R_H - R_H^*)}{R_H} \\
& + \frac{(\sigma + \mu_H)(R_H - R_H^*)^2}{R_H} + \frac{L(T)S_V^*(M_A - M_A^*)}{M_A} \\
& + \frac{L(T)I_V^*(M_A - M_A^*)}{M_A} + \frac{L(T)S_V(M_A - M_A^*)^2}{KM_A} \\
& + \frac{L(T)I_V(M_A - M_A^*)^2}{KM_A} + \frac{(\lambda(T, R) + \mu_A(T))(M_A - M_A^*)^2}{M_A}
\end{aligned}$$

$$\begin{aligned}
& + \frac{\lambda(T, R)M_A^*(S_V - S_V^*)}{S_V} + \frac{\beta_V I_H(S_V - S_V^*)^2}{S_V} \\
& + \frac{\mu_V(T)(S_V - S_V^*)}{S_V} + \frac{\beta_V S_V I_H^*(I_V - I_V^*)}{I_V} \\
& + \frac{\beta_V S_V^* I_H(I_V - I_V^*)}{I_V} + \frac{\mu_V(T)(I_V - I_V^*)^2}{I_V}.
\end{aligned} \tag{72}$$

Hence, if $A < B$, then $dL/dt \leq 0$. Note that $dL/dt = 0$ if and only if $S_H = S_H^*$, $E_H = E_H^*$, $I_H = I_H^*$, $R_H = R_H^*$, $M_A = M_A^*$, $S_V = S_V^*$, and $I_V = I_V^*$. Thus, the largest compact invariant set in $[(S_H^*, E_H^*, I_H^*, R_H^*, M_A^*, S_V^*, I_V^*) \in \mathcal{D} : dL/dt = 0]$ is the singleton set E_1 . By Lasalle's invariant principles [39], it implies that E_1 is globally asymptotically stable in \mathcal{D} if $A < B$.

4. Numerical Simulations

In this section, initial conditions $S_H(0) = 5000000$, $E_H(0) = 200$, $I_H(0) = 500$, $R_H(0) = 300$, $M_A(0) = 200000$, $S_V(0) = 300000$, $I_V(0) = 1000$ are used to perform numerical simulations of system (1)–(7) using the MATLAB computer software program. Parameter values used are given in Table 2. The replication temperature range 16°C–42°C used for analysis is established from the condition given in Section 3, that is, $L(T) > \mu_V(T)$ as shown in Figure 2.

4.1. Parameters Determining the Aquatic Mosquito Maturation Rate. According to Mukhtar et al. [30], the maturation rate of mosquitoes to adulthood is temperature-rainfall dependent governed by the total number of eggs laid per adult mosquito per oviposition $\omega(T)$, daily survival probability of rainfall-dependent eggs $P_1(R)$, daily survival probability of rainfall-dependent larvae $P_2(R)$, daily survival

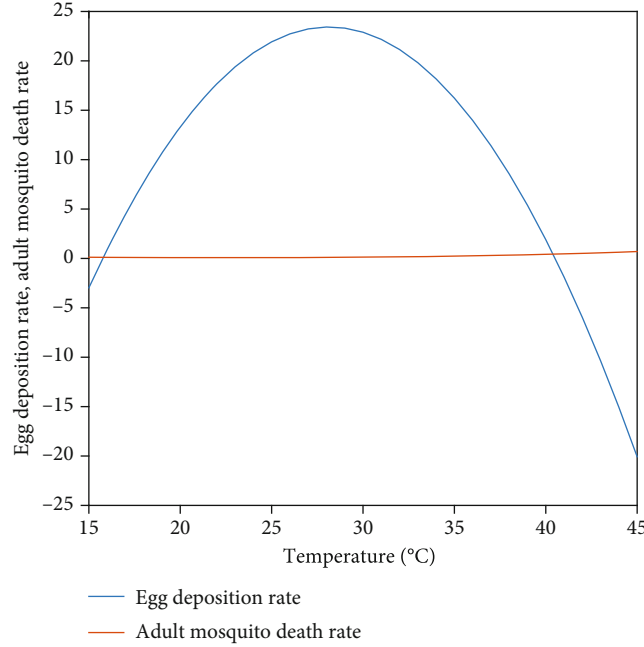


FIGURE 2: Egg deposition rate and adult mosquito death rate versus temperature.

probability of the rainfall-dependent pupae $P_3(R)$, daily survival probability of the temperature-dependent larvae $P_2(T)$, and the temperature-dependent duration of the immature mosquito development $T_{EA}(T)$ given by

$$\begin{aligned}
 \omega(T) &= \frac{-0.153T^2 + 8.61T - 97.7}{\mu_V(T)}, \\
 P_1(R) &= \frac{4 \times 0.93}{2500} R(50 - R), \\
 P_2(R) &= \frac{4 \times 0.25}{2500} R(50 - R), \\
 P_3(R) &= \frac{4 \times 0.75}{2500} R(50 - R), \\
 P_2(T) &= \exp(0.06737 - 0.00554T), \\
 T_{EA}(T) &= \frac{1}{-0.00094T^2 + 0.049T - 0.552}.
 \end{aligned} \tag{73}$$

It is assumed that aquatic mosquitoes cannot survive at daily rainfall beyond 50 mm. The maturation rate is given by

$$\lambda(T, R) = \frac{\omega(T)P_1(R)P_2(R)P_3(R)P_2(T)}{T_{EA}(T)}. \tag{74}$$

In Figure 3, daily temperature and rainfall are fixed at $T = 25^\circ\text{C}$ and $R = 10$ mm, respectively. Results in Figure 3(a) show a sharp fall in the susceptible human population and a rise in the infectious human population within the first 50 days. The infectious human population then falls due to recovery and death. Figure 3(b) shows a fall in the susceptible vector population with a rise in the infectious vector popula-

tion. It is observed from Figure 3 that the steady state in both populations is stable. Thus, the endemic equilibrium $(S_H^*, E_H^*, I_H^*, R_H^*, M_A^*, S_V^*, I_V^*)$ of the model is locally stable.

In Figure 4, temperatures within the 16°C - 42°C range are taken at a fixed amount of daily rainfall 10 mm to investigate the impact of temperature on the various compartments of the mosquito population. Temperature values of 20°C , 25°C , 30°C , 35°C , and 40°C are considered. In Figure 4(a), it is observed that the aquatic mosquito population is lowest at 40°C at all times and highest at 25°C . Thus, a temperature of 25°C is favourable for replication in the mosquito population. Figure 4(a) also shows a fall in the aquatic mosquito population with time at temperature 40°C . This shows that the aquatic mosquitoes hardly survive at very high temperatures. A sharp fall in the susceptible mosquito population is observed at all temperatures in Figure 4(b). Figure 4(c) shows the change in the infectious mosquito population with time. The highest rate of infection in the vector population is observed at 25°C and the lowest rate at 40°C . The comparison of Figures 4(a) and 4(c) shows that although the aquatic mosquito population grows faster at 30°C than 20°C , the infection rate on the other hand is higher at 20°C compared to 30°C .

Therefore, an increase in the aquatic mosquito population does not necessarily imply that there will be a corresponding rise in the infection rate. These results show that temperature greatly affects the transmission dynamics of malaria as there is a significant difference in the number of infected mosquitoes at different temperature values. Malaria is more effectively transmitted at 25°C as compared to other temperature values considered.

Figure 5 shows the impact of daily rainfall at a fixed temperature. According to Mukhtar et al. [30], aquatic mosquitoes cannot survive at daily rainfall beyond 50 mm which

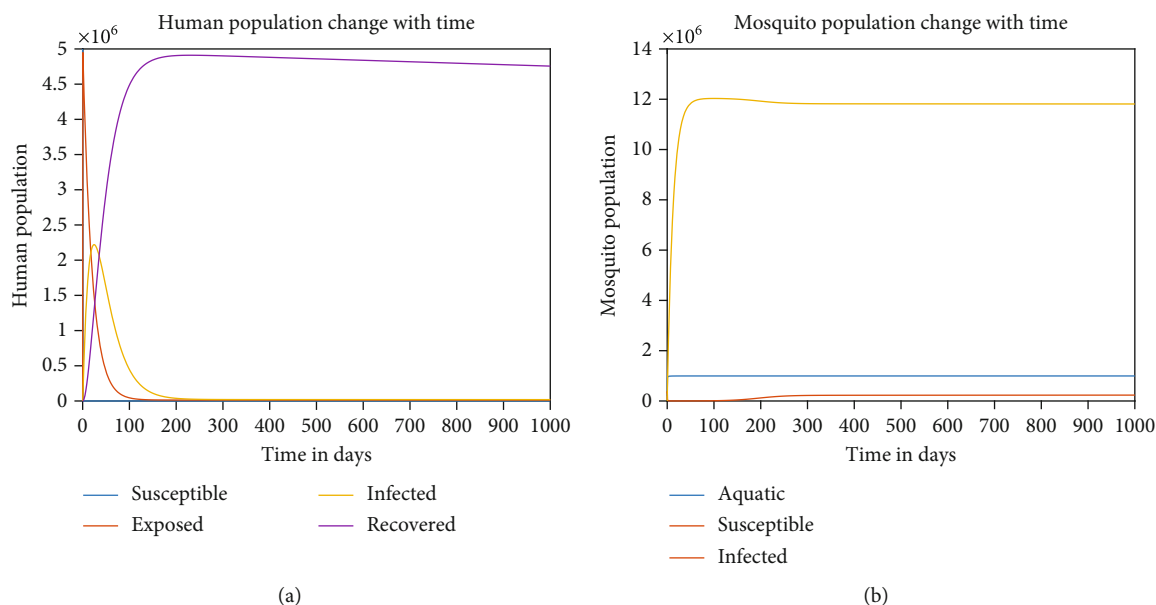


FIGURE 3: (a) Change in the human population with time over a period of 1000 days. (b) Change in mosquito population with time over a period of 1000 days.

limits the growth of the vector population. Therefore, values below 50 mm, that is, (10 mm, 20 mm, 30 mm, 40 mm) are considered at a fixed temperature 25°C. The aquatic vector population growth is observed to be lowest at rainfall value 30 mm and highest at 40 mm as shown in Figure 5(a). This is because more rainfall received provides breeding sites for the mosquitoes. Variations in the aquatic mosquito population due to temperature differences are observed to be more than the variations due to rainfall differences; thus, temperature affects the aquatic mosquito population more than rainfall. A rise followed by a sharp fall in the susceptible mosquito population is observed in Figure 5(b) at all values of rainfall. The infection rate in the vector population is highest at 30 mm of daily rainfall. This shows that the malaria parasite is more efficiently transmitted in the mosquito population at 30 mm as compared to other values considered. Similar to the observation from Figure 5, comparing Figure 5(a) and Figure 5(c), the aquatic mosquito population is higher at 40 mm than it is at 30 mm; however, the infection is seen to be higher at 30 mm compared to 40 mm. This suggests that a higher aquatic mosquito population does not necessarily lead to higher infection rates. From Figure 5, there is a variation in the infection rate for the different rainfall values; thus, rainfall affects the transmission of malaria.

5. Discussion

In this paper, a malaria transmission model with temperature and rainfall dependent parameters is formulated. The analysis of the model reveals that the model is mathematically and epidemiologically well posed. Further analysis shows that there are two disease-free equilibrium points, one without the mosquito population (E_{01}) and the other with the mosquito population (E_{02}). It is found out that the existence

and stability of the disease-free equilibria are dependent on the vector reproduction number (θ). θ is a threshold parameter defined as the number of mosquitoes produced by a female *Anopheles* mosquito throughout its lifetime, which is entirely governed by temperature-rainfall-dependent parameters (that is egg deposition rate, maturation rate of aquatic mosquitoes to adulthood, and the death rates of both adult and aquatic mosquitoes). Seasonal factors highly determine the population size of mosquito vectors which transmit malaria because mosquito replication depends on the value of θ . It was shown that the mosquito population replicates only if $\theta > 1$. The basic reproduction number \mathcal{R}_0 for the model is computed using the next-generation method, and it was shown that \mathcal{R}_0 only exists if $\theta > 1$. Malaria transmission depends on the mosquito vectors which survive only if $\theta > 1$. The disease-free equilibrium point E_{01} is stable if $\theta < 1$ which means that if an infective individual is introduced into the community at this point, malaria does not spread due to the absence of the mosquito vectors. The disease-free equilibrium point E_{02} exists if $\theta > 1$ and is stable if additionally $\mathcal{R}_0 < 1$. This means that if an infected individual is introduced into the community, malaria will not spread if $\mathcal{R}_0 < 1$; otherwise, there will be an outbreak. There is a unique endemic equilibrium if $\mathcal{R}_0 > 1$. This would suggest that malaria remains in the community as long as $\mathcal{R}_0 > 1$; thus, it is important to keep the basic reproduction number below unity.

In order to establish the temperature range within which mosquitoes replicate, the threshold parameter θ was investigated. It was revealed that a replication range of 16°C–42°C is favourable, and it is this range that was used in the proceeding analysis, because any temperature outside this range was assumed not to be favourable for mosquito reproduction. Sensitivity analysis of the model revealed that the basic

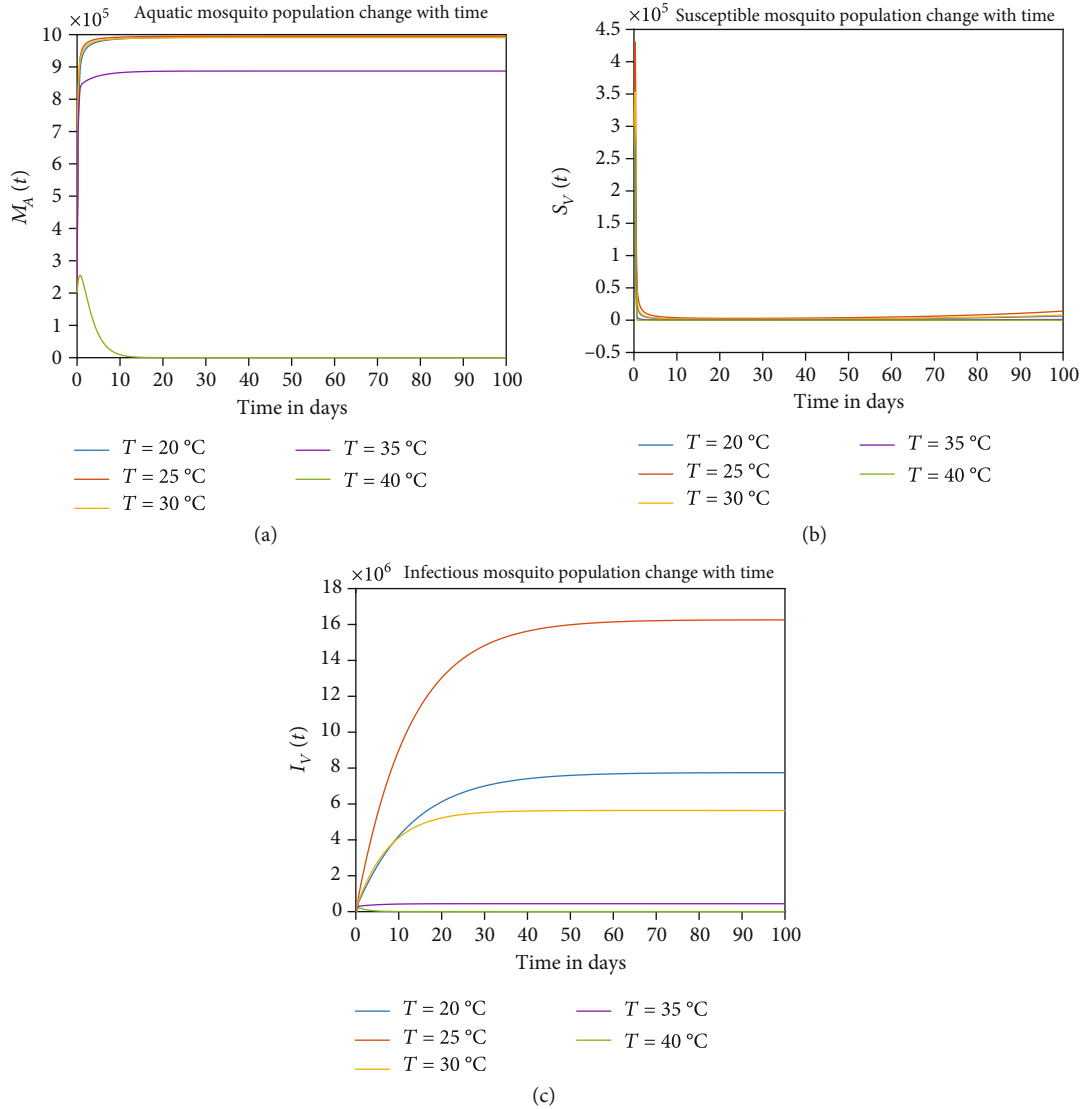


FIGURE 4: Change in the mosquito population over time for temperatures values $T = 20^\circ\text{C}$, $T = 25^\circ\text{C}$, $T = 30^\circ\text{C}$, $T = 35^\circ\text{C}$, and $T = 40^\circ\text{C}$. (a) The number of aquatic mosquitoes over a period of 100 days. (b) The number of susceptible mosquitoes over a period of 100 days. (c) The number of infectious mosquitoes over a period of 100 days.

reproduction number \mathcal{R}_0 is highly sensitive to temperature variations within 17°C - 20°C and 37°C - 40°C temperature ranges. The sensitivity of the basic reproduction number to rainfall variations reduces with more rainfall received. It was observed that sensitivity indices are positive below 25 mm and negative above 25 mm. This result shows that when the rainfall received is below 25 mm, a reduction in the amount of rainfall reduces malaria endemicity while an increase in the amount of rainfall received leads to a rise in malaria endemicity. Rainfall increments in this case create more breeding sites for the mosquitoes in the form of water pools which aid mosquito population increase. Reduction in rainfall would reduce the breeding sites and thus reduce the mosquito population. On the other hand, when the rainfall received is above 25 mm, a reduction in the amount of rainfall received increases malaria endemicity whereas an increase in rainfall reduces malaria endemicity. This is

because excessive rainfall flushes out breeding sites thus reduces the mosquito population. Sensitivity indices due to temperature variations are observed to be greater than those due to rainfall variations. This implies that malaria transmission is more sensitive to temperature changes than rainfall changes; thus, more attention should be directed to temperature variations.

Numerical simulations of the model were performed to investigate the effect of temperature and rainfall on malaria transmission. Temperature values 20°C , 25°C , 30°C , 35°C , and 40°C were considered; it was revealed that malaria is more effectively transmitted at temperature 25°C (this is in agreement with [30, 31]). Daily rainfall below 50 mm, that is, 10 mm, 20 mm, 30 mm, and 40 mm were considered, since it was assumed that mosquitoes hardly survive rainfall above 50 mm as breeding sites are flushed out. It was noted that that daily rainfall of 30 mm is favourable for malaria transmission

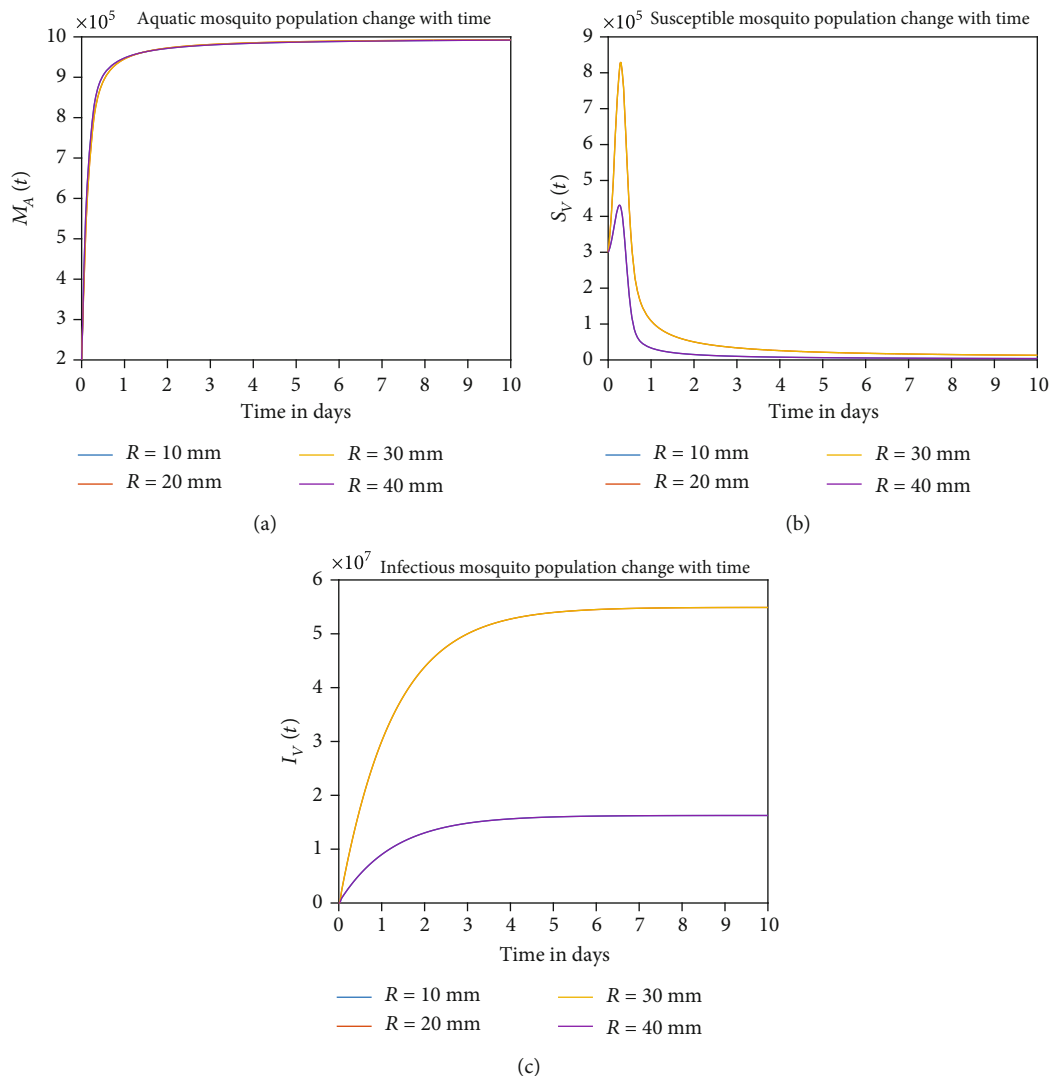


FIGURE 5: Change in mosquito population over time for rainfall values $R = 10$ mm, $R = 20$ mm, $R = 30$ mm, and $R = 40$ mm. (a) The number of aquatic mosquitoes over a period 10 days. (b) The number of susceptible mosquitoes over a period of 10 days. (c) The number of infectious mosquitoes over a period of 10 days.

as compared to other values considered. Mass malaria control programmes such as the distribution of mosquito nets should be implemented when the temperature is 25 C and daily rainfall is 30 mm. In agreement with sensitivity analysis, it was observed that variations in malaria infection due to temperature differences were more than the variations due to rainfall differences. Therefore, temperature affects the transmission dynamics of malaria more than rainfall. It was also shown that the growth of the aquatic mosquito population does not necessarily lead to higher infections. Therefore, vector control measures should target adult mosquitoes more, since most of the aquatic mosquitoes do not survive to adulthood to participate in malaria transmission.

Data Availability

The data (parameter values) used in this study were obtained from the literature.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Acknowledgments

Victor Yiga is grateful for the support from the Regional University Forum for Capacity Building in Agriculture (RUFORUM), Kyambogo University, and Mbarara University of Science and Technology to carry out this research.

References

- [1] F. E. G. Cox, "History of the discovery of the malaria parasites and their vectors," *Parasites & Vectors*, vol. 3, no. 1, p. 5, 2010.
- [2] S. Mandal, R. Sarkar, and S. Sinha, "Mathematical models of malaria - a review," *Malaria Journal*, vol. 10, no. 1, p. 202, 2011.

- [3] P. Martens, R. Kovats, S. Nijhof et al., "Climate change and future populations at risk of malaria," *Global Environmental Change*, vol. 9, no. 1, pp. S89–107, 1999.
- [4] M. B. Hoshen and A. P. Morse, "A weather-driven model of malaria transmission," *Malaria Journal*, vol. 3, no. 1, p. 32, 2004.
- [5] E. Meibalan and M. Marti, "Biology of malaria transmission," *Perspectives in Medicine Cold Spring Harbor*, vol. 7, no. 3, pp. 3–16, 2017.
- [6] World Health Organisation, *Stakeholders Meeting on Maternal Interventions Vigilance: Safety Monitoring and Surveillance in Vaccine and Other Research Settings*, World Health Organisation, Geneva, Switzerland, 2018.
- [7] J. Tumwiine, L. S. Luboobi, and J. Y. T. Mugisha, "Modelling the effect of treatment and mosquito control on malaria transmission," *International Journal of Management and Systems*, vol. 21, pp. 107–124, 2005.
- [8] World Health Organisation, *World Malaria Report*, World Health Organisation, Geneva, Switzerland, 2018.
- [9] T. M. Lunde, M. N. Bayoh, and B. Lindtjørn, "How malaria models relate temperature to malaria transmission," *Parasites & Vectors*, vol. 6, no. 1, 2013.
- [10] C. Mendis, J. L. Jacobsen, A. Gamage-Mendis et al., "*Anopheles arabiensis* and *An. funestus* are equally important vectors of malaria in Matola coastal suburb of Maputo, southern Mozambique," *Medical and Veterinary Entomology*, vol. 14, no. 2, pp. 171–180, 2000.
- [11] M. E. Sinka, M. J. Bangs, S. Manguin et al., "A global map of dominant malaria vectors," *Parasites & Vectors*, vol. 5, no. 1, 2012.
- [12] L. M. Beck-Johnson, W. A. Nelson, K. P. Paaijmans, A. F. Read, M. B. Thomas, and O. N. Bjørnstad, "The effect of temperature on *Anopheles* mosquito population dynamics and the potential for malaria transmission," *PLoS One*, vol. 8, no. 11, p. e79276, 2013.
- [13] S. E. Eikenberry and A. B. Gumel, "Mathematical modeling of climate change and malaria transmission dynamics: a historical review," *Journal of Mathematical Biology*, vol. 77, no. 4, pp. 857–933, 2018.
- [14] F. J. Colón-González, A. M. Tompkins, R. Biondi, J. P. Bizimana, and D. B. Namanya, "Assessing the effects of air temperature and rainfall on malaria incidence: an epidemiological study across Rwanda and Uganda," *Geospatial Health*, vol. 11, no. 1s, pp. 18–37, 2016.
- [15] P. A. Eckhoff, "A malaria transmission-directed model of mosquito life cycle and ecology," *Malaria Journal*, vol. 10, no. 1, p. 303, 2011.
- [16] A. K. Githeko, S. W. Lindsay, U. E. Confalonieri, and J. A. Patz, "Climate change and vector-borne diseases: a regional analysis," *Bulletin of the World Health Organisation*, vol. 78, pp. 1136–1147, 2000.
- [17] G. Zhou, N. Minakawa, A. K. Githeko, and G. Yan, "Association between climate variability and malaria epidemics in the East African highlands," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 101, no. 8, pp. 2375–2380, 2004.
- [18] M. N. Bayoh and S. W. Lindsay, "Effect of temperature on the development of the aquatic stages of *Anopheles gambiae sensu stricto* (Diptera: Culicidae)," *Bulletin of Entomological Research*, vol. 93, no. 5, pp. 375–381, 2003.
- [19] L. M. Beck-Johnson, W. A. Nelson, K. P. Paaijmans, A. F. Read, M. B. Thomas, and O. N. Bjørnstad, "The importance of temperature fluctuations in understanding mosquito population dynamics and malaria risk," *Royal Society Open Science*, vol. 4, no. 3, article 160969, 2017.
- [20] K. Okuneye, S. E. Eikenberry, and A. B. Gumel, "Weather-driven malaria transmission model with gonotrophic and sporogonic cycles," *Journal of Biological Dynamics*, vol. 13, no. 1, pp. 1–37, 2018.
- [21] S. S. Imbahale, K. P. Paaijmans, W. R. Mukabana, R. van Lammeren, A. K. Githeko, and W. Takken, "A longitudinal study on *Anopheles* mosquito larval abundance in distinct geographical and environmental settings in western Kenya," *Malaria Journal*, vol. 10, no. 1, article 81, 2011.
- [22] R. Ross, *The Prevention of Malaria*, Murray, London, 1911.
- [23] G. Macdonald, *The Epidemiology and Control of Malaria*, Oxford University Press, 1957.
- [24] J. L. Aron, "Acquired immunity dependent upon exposure in an SIRS epidemic model," *Mathematical Biosciences*, vol. 88, no. 1, pp. 37–47, 1988.
- [25] N. T. Bailey, *The Biomathematics of Malaria*, Charles Griffin and Company Ltd, 1982.
- [26] M. H. Craig, R. W. Snow, and D. le Sueur, "A climate-based distribution model of malaria transmission in sub-Saharan Africa," *Parasitology Today*, vol. 15, no. 3, pp. 105–111, 1999.
- [27] M. J. Bouma, H. E. Sondorp, and H. J. van der Kaay, "Climate change and periodic epidemic malaria," *The Lancet*, vol. 343, no. 8910, p. 1440, 1994.
- [28] S. W. Lindsay and M. H. Birley, "Climate change and malaria transmission," *Annals of Tropical Medicine and Parasitology*, vol. 90, no. 5, pp. 573–588, 2016.
- [29] Y. Lou and X. Q. Zhao, "A climate-based malaria transmission model with structured vector population," *SIAM Journal on Applied Mathematics*, vol. 70, no. 6, pp. 2023–2044, 2010.
- [30] A. Y. Mukhtar, J. B. Munyakazi, and R. Oufiki, "Assessing the role of climate factors on malaria transmission dynamics in South Sudan," *Mathematical Biosciences*, vol. 310, pp. 13–23, 2019.
- [31] E. T. Ngarakana-Gwasira, C. P. Bhunu, and E. Mashonjowa, "Assessing the impact of temperature on malaria transmission dynamics," *Afrika Matematika*, vol. 25, no. 4, pp. 1095–1112, 2014.
- [32] P. E. Parham and E. Michael, "Modelling climate change and malaria transmission," *Modelling Parasite Transmission and Control*, vol. 673, no. 2010, pp. 184–199, 2010.
- [33] G. Bhuju, G. R. Phaijoo, and D. B. Gurung, "Mathematical study on impact of temperature in malaria disease transmission dynamics," *Advances in Computer Science*, vol. 1, no. 2, pp. 1–8, 2018.
- [34] O. Diekmann, J. A. P. Heesterbeek, and J. A. Metz, "On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations," *Journal of Mathematical Biology*, vol. 28, no. 4, pp. 365–382, 1990.
- [35] N. Chitnis, J. M. Hyman, and J. M. Cushing, "Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model," *Bulletin of Mathematical Biology*, vol. 70, no. 5, pp. 1272–1296, 2008.
- [36] P. van den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," *Mathematical Biosciences*, vol. 180, no. 1–2, pp. 29–48, 2002.

- [37] C. Castillo-Chavez, S. Blower, P. Driesschevan den, D. Kirschner, and A. A. Yakubu, Eds., *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: an Introduction*, vol. 1, no. 229, 2002Springer Science and Business Media, 2002.
- [38] C. Castillo-Chavez and B. Song, "Dynamical models of tuberculosis and their applications," *Mathematical Biosciences and Engineering*, vol. 1, no. 2, pp. 361–404, 2004.
- [39] J. P. La Salle, *The Stability of Dynamical system*, SIAM, Philadelphia, 1976.
- [40] Uganda Bureau of Statistics, *The National Population and Housing Census 2014–Main Report*, Uganda Bureau of Statistics, Kampala, Uganda, 2016.
- [41] G. Pison, "The population of the world," *Population and Sociétés*, vol. 569, pp. 1–8, 2019.
- [42] D. Sunita and B. Nisha, "A model for malaria transmission dynamics with varying human interaction coefficients," *International Journal of Simulation Systems, Science and Technology*, vol. 20, no. 4, 2019.
- [43] K. Blayneh, Y. Cao, and H.-D. Kwon, "Optimal control of vector-borne diseases: treatment and prevention," *Discrete and Continuous Dynamical Systems*, vol. 11, no. 3, pp. 587–611, 2009.