

Global Stability Analysis of the Mathematical Model for Malaria Transmission between Vector and Host Population

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Abstract

In this paper, we discuss a mathematical model of malaria transmission between vector and host population. We study the basic qualitative properties of the model, the boundedness and non-negativity, calculate all equilibria, and prove the global stability of them and the behaviour of the model when the basic reproduction ratio R_0 is greater than one or less than one. The global stability of equilibria is established by using Lyapunov method. Graphical representations of the calculated parameters and their effects on disease eradication are provided.

Keywords

Malaria Transmission, Global Stability, Lyapunov Function

1. Introduction

Malaria is a life-threatening disease spread to humans by certain types of mosquitoes. According to scientific studies of National Institutes of Health (NIH), Malaria is the fifth most deadly infectious disease [1]. It is mostly found in tropical countries. One of the deadliest forms of malaria is cerebral malaria, where the patient experiences severe neurological symptoms, such as seizures and coma, the condition is lethal without treatment. Symptoms can range from mild to life-threatening. Mild symptoms include fever, chills, and headache. Severe symptoms include fatigue, confusion, seizures, muscle aches, nausea, vomiting, diarrhea and difficulty breathing [2]. Infants, children under 5 years, pregnant women, travellers, and people with HIV or AIDS are at higher risk of severe infection. Malaria can be prevented by avoiding mosquito bites and through me-

dication. Environmental factors are more significant than genetics in determining a person immunity, according to scientific studies of National Institutes of Health (NIH) [1]. Treatments can prevent mild cases from worsening as mentioned by the World Health Organization (WHO) [2], where the NIH clinical trials [3]-[5] have found that one dose of an antibody-drug safely protected healthy, non-pregnant adults from malaria infection during an intense six-month malaria season in Mali, Africa. The antibody was up to 88.2% effective at preventing infection over a 24-week period, demonstrating for the first time that a monoclonal antibody can prevent malaria infection in an endemic region. According to the World Malaria Report (WHO) in 2023 [2], there were 249 million cases of malaria in 2022 compared to 244 million cases in 2021. The estimated number of malaria deaths stood at 608,000 in 2022 compared to 610,000 in 2021. The WHO African Region continues to bear a disproportionately high share of the global malaria burden. In 2022, the region was home to about 94% of all malaria cases and 95% of deaths. Children under 5 years of age accounted for about 78% of all malaria deaths in the region. Four African countries accounted for just over half of all malaria deaths worldwide: Nigeria (26.8%), the Democratic Republic of the Congo (12.3%), Uganda (5.1%), and Mozambique (4.2%) [2].

The Centers for Disease Control and Prevention reported that a number of Malaria cases were transmitted within the USA in Texas and Florida, and became very important to understand the interactions of between climate, mosquitoes and pathogens mosquitoes transmit to humans, and this was the importance of mathematical modelling. For the last 20 years, researchers focused on understanding the impacts of environmental change on the ecology of insect vectors and the diseases they transmit, combining field studies and modelling approaches, statistically and mathematically to explain the interactions. This approach advances our capacity to engage in more relational science, where factors are not understood as fixed and independent forces, but as dynamic and interdependent. Relations between variables can be expressed by nonlinear relationship that can be easily grasped by machine learning algorithms and other data science tools. Computers have made it easier to deal with larger datasets. The automation of data assimilation can lead to predicting where and when to expect the transmission of some vector-borne diseases. Mathematical models also show how the stability of natural systems can collapse following small changes in the environment, and that has clear implications about why we need to worry as climate change continues its current course [6]. Mathematical modelling has been used by epidemiologists as a tool to predict the occurrence of epidemics of infectious diseases, and also as a tool for guiding research for eradication of malaria at the present time [7].

The route of mathematical modelling was explained in [8] and gave an insight of Malaria modelling by mathematical tools. Mathematical models have been used to provide an explicit framework for understanding malaria transmission dynamics in human population for over 100 years.

The SIR-SI model represents an advancement over Ross’s SIS [9] model by incorporating the relationship between human hosts and vector (mosquito) compartments. It categorizes humans into three groups: susceptible, infectious, and recovered, while vectors are divided into susceptible and infectious categories. Importantly, unlike its predecessor, individuals in this model do not revert to susceptibility once they acquire immunity. The model discussed in [10] addresses the impact of environmental immunity on the mathematical modelling of malaria transmission between vector and host populations. However, it lacks some of the mathematical analysis, and therefore, the primary objective of this paper is to conduct a comprehensive mathematical analysis.

Our study aims to explore the fundamental qualitative properties of the proposed model, including the investigation of the global stability. To validate our theoretical findings, we utilized numerical simulations. Finally, we provide a thorough discussion of the results obtained, offering insights into the dynamics of malaria transmission within the context of environmental immunity.

2. Model Formulation

In this section, we study the global stability of the SIR-SI model ([10] and references therein). The model comprises five compartments: the number of susceptible healthy human to the disease, S_h , the number of the infected human I_h , the number of the recovered humans R_h , the number of susceptible mosquitoes (or vector) S_v , and the number of infected mosquitoes (or vector) I_v . The environmental immunity parameter was incorporated into the infectious and recovered classes to examine its impact on the basic reproduction number and the equilibrium points. The schematic diagram of the model is illustrated in **Figure 1**. The outlines of the adopted model are [10]:

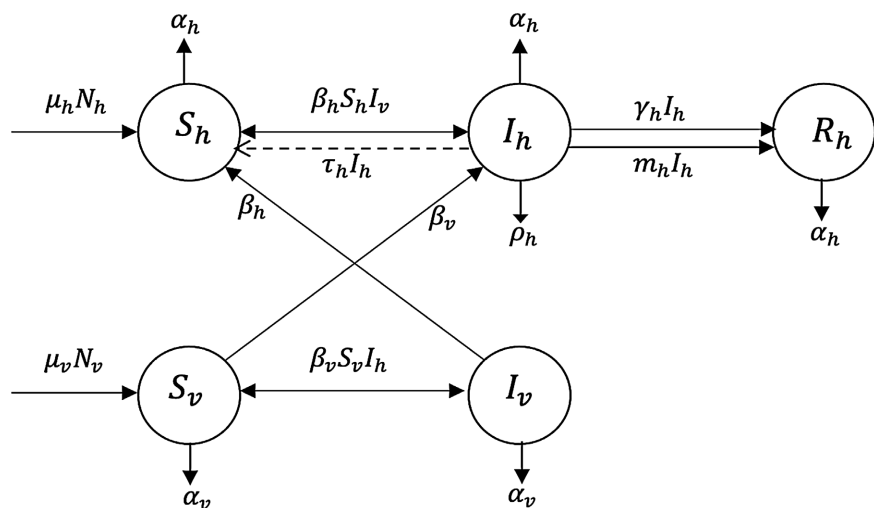


Figure 1. The schematic diagram of the SIR-SI co-infection dynamics within host-vector.

A_1 . The model considers the interactions between five compartments: S_h , I_h , R_h , S_v , I_v . Total human population at time t is denoted by $N_h(t)$ while

the total vector (mosquito) population at t is $N_v(t)$.

A_2 . The parameters of birth/migration and death-rates of the susceptible humans are μ_h and α_h , respectively.

A_3 . The parameters of birth/migration and death-rates of the susceptible vectors are μ_v and α_v , respectively.

A_4 . The rates of I_h are: contact/transmission β_h , recovery γ_h , disease induced death ρ_h , environmental immunity due to nutrition and supplement m_h , and which infected humans enter the susceptible class at rate τ_h .

A_5 . The infected vectors' contact/transmission rate is β_v .

Based on Assumptions A_1 to A_5 , the model was formulated as:

$$dS_h/dt = S'_h(t) = \mu_h N_h(t) - \beta_h S_h(t) I_v(t) - \alpha_h S_h(t) + \tau_h I_h(t) \tag{1a}$$

$$dI_h/dt = I'_h(t) = \beta_h S_h(t) I_v(t) - (\alpha_h + \gamma_h + \rho_h + m_h + \tau_h) I_h(t) \tag{1b}$$

$$dR_h/dt = R'_h(t) = (\gamma_h + m_h) I_h(t) - \alpha_h R_h(t) \tag{1c}$$

$$dS_v/dt = S'_v(t) = \mu_v N_v(t) - \beta_v S_v(t) I_h(t) - \alpha_v S_v(t) \tag{1d}$$

$$dI_v/dt = I'_v(t) = \beta_v S_v(t) I_h(t) - \alpha_v I_v(t) \tag{1e}$$

3. Basic Qualitative Properties

In this section, we study the basic qualitative properties of system (1a)-(1e) including the non-negativity, boundness, and stability. To prove that the solution of the system (1a)-(1e) is non-negative and bounded; we apply the following lemma.

Lemma 1. *The solutions of system (1a)-(1e) are nonnegative and bounded.*

Proof. We have that

$$S'_h|_{S_h=0} = \mu_h N_h + \tau_h I_h \geq 0 \text{ for all } I_h \geq 0, \quad I'_h|_{I_h=0} = \beta_h S_h I_v \geq 0 \text{ for all } S_h, I_v \geq 0,$$

$$R'_h|_{R_h=0} = (\gamma_h + m_h) I_h \geq 0 \text{ for all } I_h \geq 0, \quad S'_v|_{S_v=0} = \mu_v N_v > 0,$$

$$I'_v|_{I_v=0} = \beta_v S_v I_h \geq 0 \text{ for all } S_v, I_h \geq 0.$$

This guarantees that: $(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t)) \in \mathbb{R}_{\geq 0}^5$ for all $t \geq 0$ when $(S_h(0), I_h(0), R_h(0), S_v(0), I_v(0)) \in \mathbb{R}_{\geq 0}^5$. This proves the non-negativity of the solution.

Next, let:

$$\Psi(t) = S_h(t) + I_h(t) + R_h(t) + S_v(t) + I_v(t),$$

then

$$\begin{aligned} \Psi' &= \mu_h N_h + \mu_v N_v - \alpha_h S_h - (\alpha_h + \rho_h) I_h - \alpha_h R_h - \alpha_v S_v - \alpha_v I_v, \\ &\leq \mu_h N_h + \mu_v N_v - \phi [S_h + I_h + R_h + S_v + I_v] = \mu_h N_h + \mu_v N_v - \phi \Psi, \end{aligned}$$

where $\phi = \min\{\alpha_h, \alpha_h + \rho_h, \alpha_v\}$, thus, $0 \leq \Psi(t) \leq \Delta_1$ if $\Psi(0) \leq \Delta_1$ for $t \geq 0$, where $\Delta_1 = (\mu_h N_h + \mu_v N_v) / \phi$. Since S_h, I_h, R_h, S_v , and I_v are all nonnegative, then $0 \leq S_h, I_h, R_h, S_v, I_v \leq \Delta_1$, if $S_h(0) + I_h(0) + R_h(0) + S_v(0) + I_v(0) \leq \Delta_1$. This proves the boundedness of the solutions. ■

4. Steady-State Solutions

The model has two equilibrium points. To compute the disease-free equilibrium, the change with time was set to zero. The resulting system of nonlinear algebraic equations, then, was solved symbolically, using Matlab (see Appendices for Matlab-coding):

$$\mu_h N_h - \beta_h S_h I_v - \alpha_h S_h + \tau_h I_h = 0 \quad (2a)$$

$$\beta_h S_h I_v - \alpha_h I_h - \gamma_h I_h - \rho_h I_h - m_h I_h - \tau_h I_h = 0 \quad (2b)$$

$$\gamma_h I_h - \alpha_h R_h + m_h I_h = 0 \quad (2c)$$

$$\mu_v N_v - \beta_v S_v I_h - \alpha_v S_v = 0 \quad (2d)$$

$$\beta_v S_v I_h - \alpha_v I_v = 0 \quad (2e)$$

4.1. Disease Free Equilibrium \mathcal{D}_0

This occurs when there is no infection, *i.e.* $I_h = I_v = 0$, and hence Equations (2a)-(2e) become:

$$\mu_h N_h - \alpha_h S_h = 0,$$

$$-\alpha_h R_h = 0,$$

$$\mu_v N_v - \alpha_v S_v = 0.$$

Solving for S_h , R_h and S_v , we obtain:

$$S_h^0 = \frac{\mu_h N_h}{\alpha_h},$$

$$R_h^0 = 0,$$

$$S_v^0 = \frac{\mu_v N_v}{\alpha_v},$$

which gives the disease-free equilibrium as:

$$\mathcal{D}_0 = (S_h^0, I_h^0, R_h^0, S_v^0, I_v^0) = ((\mu_h N_h)/\alpha_h, 0, 0, (\mu_v N_v)/\alpha_v, 0).$$

4.2. Basic Reproduction Number \mathcal{R}_0

The Basic reproduction number \mathcal{R}_0 , is the number of secondary infection caused by one infected individual in a wholly susceptible population. Here, it is obtained using next generation matrix [11]. We first define the matrices F and V as follows:

$$F = \begin{pmatrix} \beta_h S_h I_v \\ \beta_v S_v I_h \end{pmatrix}, \quad \text{gives} \quad \partial F(\mathcal{D}_0) = \begin{pmatrix} 0 & \beta_h S_h^0 \\ \beta_v S_v^0 & 0 \end{pmatrix}$$

and,

$$V = \begin{pmatrix} -\xi I_h \\ -\alpha_v I_v \end{pmatrix}, \quad \text{gives} \quad \partial V^{-1} = \begin{pmatrix} \frac{-1}{\xi} & 0 \\ 0 & -\beta_h S_h / \alpha_v \end{pmatrix}.$$

Therefore,

$$\partial FV^{-1} = \begin{pmatrix} 0 & \frac{-\beta_h S_h^0}{\alpha_v} \\ \frac{-\beta_v S_v^0}{\xi} & 0 \end{pmatrix},$$

and hence: \mathcal{R}_0 is the dominant eigenvalue. Therefore,

$$\mathcal{R}_0 = \sqrt{\frac{\beta_v S_v^0 \beta_h S_h^0}{\xi \alpha_v}}.$$

4.3. Endemic Equilibrium \mathcal{D}_1

At the endemic equilibrium state, the disease cannot be completely eliminated, but instead, it stays within the population. To maintain the disease within the population, the model requires that the values of S_h, I_h, R_h, S_v, I_v are not to be equal to zero at the equilibrium state. The endemic equilibrium represents a constant state of the model, where infected humans and vectors indicate the presence of the infection, *i.e.*, $I_h = I_v \neq 0$. From the model Equations (1a)-(1e). Solving for $S_h^*, I_h^*, R_h^*, S_v^*, I_v^*$, we have from Equations (1a)-(1e):

$$I_v^* = \frac{\beta_v S_v^* I_h^*}{\alpha_v}, \tag{3a}$$

$$S_v^* = \frac{\mu_v N_v}{\beta_v I_h^* + \alpha_v}, \tag{3b}$$

$$R_h^* = \frac{(\gamma_h + m_h) I_h^*}{\alpha_h}, \tag{3c}$$

$$S_h^* = \frac{\mu_h N_h + \tau_h I_h^*}{\beta_h I_v^* + \alpha_h}, \tag{3d}$$

to find I_h^* , we use the basic reproduction number to obtain:

$$I_h^* = \frac{\beta_h S_h^* I_v^*}{\xi} \text{ i.e. } I_h^* = \frac{\xi \alpha_h \alpha_v^2 (\mathcal{R}_0^2 - 1)}{\beta_h \beta_v \mu_v N_v \psi + \xi \alpha_h \alpha_v \beta_v}, \tag{3e}$$

where, $\xi = \alpha_h + \gamma_h + \rho_h + m_h + \tau_h$. Therefore Endemic equilibrium, $\mathcal{D}_1 = (S_h^*, I_h^*, R_h^*, S_v^*, I_v^*)$ exists if $\mathcal{R}_0 > 1$.

5. Global Stability Analysis

Stability analysis is at the heart of dynamical analysis. Only stable solutions can be noticed experimentally. Therefore, in this section we examine the global asymptotic stability of all equilibria by establishing suitable Lyapunov functions [12] and applying the Lyapunov-LaSalle asymptotic stability theorem (L-LAST) [13]-[15]. Let a function $L_j(S_h, I_h, S_v, I_v)$ and $\tilde{\mathcal{M}}_j$ be the largest invariant subset of

$$\mathcal{M}_j = \left\{ (S_h, I_h, R_h, S_v, I_v) : \frac{dL_j}{dt} = 0 \right\}, \quad j = 0, 1.$$

Define a function

$$F(v) = v - 1 - \ln v.$$

Theorem 1. *If $\mathcal{R}_0 \leq 1$, the disease-free equilibrium \mathcal{D}_0 is globally asymptotically stable (G.A.S).*

Proof. Define

$$L_0 = S_h^0 F\left(\frac{S_h}{S_h^0}\right) + I_h + \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} S_v^0 F\left(\frac{S_v}{S_v^0}\right) + \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} I_v,$$

note that, $L_0 > 0$ for all $S_h, I_h, S_v, I_v > 0$, and $L_0(S_h^0, 0, S_v^0, 0) = 0$. By differentiating L_0 along the solutions of system of OD Equations (1a)-(1e):

$$\begin{aligned} \frac{dL_0}{dt} &= \left(1 - \frac{S_h^0}{S_h}\right) (\mu_h N_h - \beta_h S_h I_v - \alpha_h S_h + \tau_h I_h) \\ &\quad + \beta_h S_h I_v - (\alpha_h + \gamma_h + \rho_h + m_h + \tau_h) I_h \\ &\quad + \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} \left(1 - \frac{S_v^0}{S_v}\right) [\mu_v N_v - \beta_v S_v I_h - \alpha_v S_v] \\ &\quad + \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} \beta_v S_v I_h - \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} \alpha_v I_v \\ &= \left(1 - \frac{S_h^0}{S_h}\right) (\mu_h N_h - \alpha_h S_h) + \tau_h I_h \left(1 - \frac{S_h^0}{S_h}\right) \\ &\quad + \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} \left(1 - \frac{S_v^0}{S_v}\right) (\mu_v N_v - \alpha_v S_v) \\ &\quad + \left(\beta_h S_h^0 - \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} \alpha_v\right) I_v \\ &= \left(1 - \frac{S_h^0}{S_h}\right) (\mu_h N_h - \alpha_h S_h) + \tau_h I_h \left(1 - \frac{S_h^0}{S_h}\right) \\ &\quad + \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} \left(1 - \frac{S_v^0}{S_v}\right) (\mu_v N_v - \alpha_v S_v) \\ &\quad + \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} \alpha_v \left(\frac{\beta_h S_h^0}{\alpha_v} \frac{\beta_v S_v^0}{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h} - 1\right) I_v. \end{aligned}$$

Using the equilibrium condition: $\mu_h N_h = \alpha_h S_{h_0}$, and $\mu_v N_v = \alpha_v S_{v_0}$ we get:

$$\begin{aligned} \frac{dL_0}{dt} &= -\alpha_h \frac{(S_h - S_h^0)^2}{S_h} + \tau_h I_h \left(1 - \frac{S_h^0}{S_h}\right) \\ &\quad - \alpha_v \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} \frac{(S_v - S_v^0)^2}{S_v} \\ &\quad + \frac{\alpha_h + \gamma_h + \rho_h + m_h + \tau_h}{\beta_v S_v^0} \alpha_v (\mathcal{R}_0^2 - 1) I_v. \end{aligned}$$

Since $\mathcal{R}_0^2 < \mathcal{R}_0 < 1$ and $S_h^0/S_h \geq 1$, then $L_0' \leq 0$ for all $S_h, I_h, S_v, I_v > 0$. In

addition $L'_0 = 0$ when $S_h = S_h^0$, $S_v = S_v^0$ and $I_h = R_h = I_v = 0$. The solution of system (1a)-(1e) tends to $\mathcal{M} = \{\mathcal{D}_0\}$. Therefore, by applying Lyapunov-LaSalle asymptotic stability theorem (L-LAST) [13]-[15], we obtain that the disease-free equilibrium \mathcal{D}_0 is G.A.S. ■

Theorem 2. Let $\mathcal{R}_0 > 1$. If $I_h^*/S_h^* \leq \alpha_h/\tau_h$. Then, the infected steady state \mathcal{D}_1 is (G.A.S.). In particular, is equivalent to:

$$1 < \mathcal{R}_0 \leq \mathcal{E} + 1$$

where,

$$\begin{aligned} \mathcal{E} &= \frac{\mathcal{E}_1 \alpha_h (\alpha_v \mu_v N_v \mathcal{E}_1 + \tau_h \xi \alpha_h \alpha_v^3 (\mathcal{R}_0^2 - 1)) (\mathcal{E}_2 + \alpha_v \mathcal{E}_1)}{\tau_h \xi \alpha_h \alpha_v^2 (\mathcal{E}_1 [\beta_h \beta_v \mu_v N_v + \alpha_h \alpha_v^2] + \alpha_h \alpha_v \mathcal{E}_2 (\mathcal{R}_0^2 - 1))}, \\ \mathcal{E}_1 &= \xi \alpha_h \alpha_v \beta_v + \beta_h \beta_v \mu_v N_v \psi, \\ \mathcal{E}_2 &= \beta_h \xi \alpha_h \alpha_v^2, \\ \psi &= \alpha_h + \gamma_h + \rho_h + m_h + \tau_h. \end{aligned}$$

Proof. Define the global Lyapunov functional for Endemic equilibrium \mathcal{D}_1 ,

$$\begin{aligned} L_1 &= S_h^* F\left(\frac{S_h}{S_h^*}\right) + I_h^* F\left(\frac{I_h}{I_h^*}\right) + \frac{\beta_h S_h^* I_v^*}{\beta_v S_v^* I_h^*} S_v^* F\left(\frac{S_v}{S_v^*}\right) + \frac{\beta_h S_h^* I_v^*}{\beta_v S_v^* I_h^*} I_v^* F\left(\frac{I_v}{I_v^*}\right) \\ &+ \frac{\tau_h}{2(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} \left[(S_h - S_h^*) + (I_h - I_h^*) \right]^2. \end{aligned}$$

The time derivative of L_1 computed along solutions of the model is

$$\begin{aligned} \frac{dL_1}{dt} &= \left(1 - \frac{S_h^*}{S_h}\right) S_h' + \left(1 - \frac{I_h^*}{I_h}\right) I_h' + \frac{\beta_h S_h^* I_v^*}{\beta_v S_v^* I_h^*} \left(1 - \frac{S_v^*}{S_v}\right) S_v' + \frac{\beta_h S_h^* I_v^*}{\beta_v S_v^* I_h^*} \left(1 - \frac{I_v^*}{I_v}\right) I_v' \\ &+ \frac{\tau_h}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} \left[(S_h - S_h^*) + (I_h - I_h^*) \right] (S_h' + I_h'). \\ \frac{dL_1}{dt} &= \left(1 - \frac{S_h^*}{S_h}\right) (\mu_h N_h - \beta_h S_h I_v - \alpha_h S_h + \tau_h I_h) + \left(1 - \frac{I_h^*}{I_h}\right) (\beta_h S_h I_v - \xi I_h) \\ &+ \frac{\beta_h S_h^* I_v^*}{\beta_v S_v^* I_h^*} \left(1 - \frac{S_v^*}{S_v}\right) (\mu_v N_v - \beta_v S_v I_v - \alpha_v S_v) \\ &+ \frac{\beta_h S_h^* I_v^*}{\beta_v S_v^* I_h^*} \left(1 - \frac{I_v^*}{I_v}\right) (\beta_v S_v I_h - \alpha_v I_v) + \frac{\tau_h}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} \\ &\times \left[(S_h - S_h^*) + (I_h - I_h^*) \right] \left[\mu_h N_h - \alpha_h (S_h + I_h) - (\gamma_h + \rho_h + m_h) I_h \right]. \end{aligned}$$

From the steady-states solution:

$$\begin{aligned} \mu_h N_h &= \beta_h S_h^* I_v^* + \alpha_h S_h^* - \tau_h I_h^*, \\ \mu_h N_h &= \alpha_h (S_h^* + I_h^*) + (\gamma_h + \rho_h + m_h) I_h^*, \\ \mu_v N_v &= \beta_v S_v^* I_h^* + \alpha_v S_v^* \end{aligned}$$

we get:

$$\begin{aligned} \frac{dL_1}{dt} &= \alpha_h \left(1 - \frac{S_h^*}{S_h}\right) [S_h^* - S_h] + \tau_h \left(1 - \frac{S_h^*}{S_h}\right) [I_h - I_h^*] \\ &+ \left(1 - \frac{S_h^*}{S_h}\right) [\beta_h S_h^* I_v^* - \beta_h S_h I_v] + \left(1 - \frac{I_h^*}{I_h}\right) \beta_h S_h I_v - \left(1 - \frac{I_h^*}{I_h}\right) \xi I_h \\ &+ \frac{\beta_h S_h^* I_v^*}{\beta_v S_v^* I_h^*} \left(1 - \frac{S_v^*}{S_v}\right) [\alpha_v S_v^* - \alpha_v S_v] + \frac{\beta_h S_h^* I_v^*}{\beta_v S_v^* I_h^*} \left(1 - \frac{S_v^*}{S_v}\right) [\beta_v S_v^* I_v^* - \beta_v S_v I_v] \\ &+ \frac{\beta_h S_h^* I_v^*}{\beta_v S_v^* I_h^*} \left(1 - \frac{I_v^*}{I_v}\right) \beta_v S_v I_h - \frac{\beta_h S_h^* I_v^*}{\beta_v S_v^* I_h^*} \left(1 - \frac{I_v^*}{I_v}\right) \alpha_v I_v \\ &+ \frac{\tau_h}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} [(S_h - S_h^*) + (I_h - I_h^*)] [(S_h^* - S_h) + (I_h^* - I_h)] \\ &+ \frac{\tau_h (\gamma_h + \rho_h + m_h)}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} (I_h^* - I_h) [(S_h - S_h^*) + (I_h - I_h^*)], \end{aligned}$$

since:

$$\tau_h \left(1 - \frac{S_h^*}{S_h}\right) [I_h - I_h^*] = \tau_h (I_h - I_h^*) \frac{(S_h - S_h^*)^2}{S_h S_h^*} + \frac{\tau_h}{S_h^*} (S_h - S_h^*) (I_h - I_h^*),$$

therefore,

$$\begin{aligned} \frac{dL_1}{dt} &= \frac{-\alpha_h S_h^*}{S_n S_h^*} [S_h - S_h^*]^2 - \frac{\tau_h (I_h - I_h^*)}{S_h S_h^*} [S_h - S_h^*]^2 + \frac{\tau_h (S_h - S_h^*) (I_h - I_h^*)}{S_h^*} \\ &+ \beta_h S_h^* I_v^* \left(1 - \frac{S_h^*}{S_h}\right) - \beta_h S_h^* I_v^* \frac{S_h I_v I_h^*}{S_h^* I_v^* I_h} + \beta_h S_h^* I_v^* \\ &- \frac{\beta_h S_h^* I_v^* \alpha_v}{\beta_v S_v^* I_h^* S_v} [S_v - S_v^*]^2 + \beta_h S_h^* I_v^* \left(1 - \frac{S_v^*}{S_v}\right) - \beta_h S_h^* I_v^* \frac{S_v I_h I_v^*}{S_v^* I_h^* I_v} \\ &+ \beta_h S_h^* I_v^* + \frac{\tau_h \alpha_h S_h S_h^*}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_n^* S_n S_n^*} (S_n - S_n^*)^2 \\ &- \frac{\tau_h}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} [\alpha_h + \gamma_h + \rho_h + m_h] (I_h - I_h^*)^2 \\ &- \frac{\tau_h}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} [2\alpha_h + \gamma_h + \rho_h + m_h] (I_h - I_h^*) (S_h - S_h^*). \\ \frac{dL_1}{dt} &= - \left[\alpha_h S_h^* + \tau_h (I_h - I_h^*) + \frac{\tau_h \alpha_h S_h S_h^*}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} \right] \frac{(S_h - S_h^*)^2}{S_h S_h^*} \\ &- \frac{\beta_h S_h^* I_v^* \alpha_v}{\beta_v S_v^* I_h^* S_v} (S_v - S_v^*)^2 \\ &- \frac{\tau_h}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} [\alpha_h + \gamma_h + \rho_h + m_h] (I_h - I_h^*)^2 \\ &+ \beta_h S_h^* I_v^* \left[4 - \frac{S_h^*}{S_h} - \frac{S_h I_v I_h^*}{S_h^* I_v^* I_h} - \frac{S_v^*}{S_v} - \frac{S_v I_h I_v^*}{S_v^* I_h^* I_v} \right] \\ &+ \left[\frac{\tau_h}{S_h^*} - \frac{\tau_h}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} [2\alpha_h + \gamma_h + \rho_h + m_h] \right] (I_h - I_h^*) (S_h - S_h^*) \end{aligned}$$

$$\begin{aligned} \frac{dL_1}{dt} = & - \left[\alpha_h S_h^* - \tau_h I_h^* + \tau_h I_h + \frac{\tau_h \alpha_h S_h S_h^*}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} \right] \frac{(S_h - S_h^*)^2}{S_h S_h^*} \\ & - \frac{\beta_h S_h^* I_v^* \alpha_v}{\beta_v S_v^* I_h^* S_v} (S_v - S_v^*)^2 \\ & - \frac{\tau_h}{(2\alpha_h + \gamma_h + \rho_h + m_h) S_h^*} [\alpha_h + \gamma_h + \rho_h + m_h] (I_h - I_h^*)^2 \\ & + \beta_h S_h^* I_v^* \left[4 - \frac{S_h^*}{S_h} - \frac{S_h I_v I_h^*}{S_h^* I_v^* I_h} - \frac{S_v^*}{S_v} - \frac{S_v I_h I_v^*}{S_v^* I_h^* I_v} \right]. \end{aligned}$$

If $I_h^*/S_h^* \leq \alpha_h/\tau_h$, then $\frac{dL_1}{dt} \leq 0$ for all S_h, I_h, R_h, S_v, I_v . The solutions of system (1-5) limit to \mathcal{M} , the largest invariant subset of

$$\left\{ (S_h^*, I_h^*, R^*, S_v^*, I_v^*) \in R \mid \frac{dL_1}{dt} = 0 \right\}.$$

Therefore by LaSalle invariant principle, if $I_h^*/S_h^* \leq \alpha_h/\tau_h$, then Endemic equilibrium \mathcal{D}_1 is globally asymptotically stable.

Finally, we show that $I_h^*/S_h^* \leq \alpha_h/\tau_h$. Using S_h^* and I_h^* of Equations ((3d), (3e)), we have

$$\alpha_h S_h^* - \tau_h I_h^* \equiv \frac{\tau_h \xi \alpha_h \alpha_v^2}{E_1} [\mathcal{E} + 1 - \mathcal{R}_0^2]$$

Therefore, $I_h^*/S_h^* \leq \alpha_h/\tau_h$, if $1 < \mathcal{R}_0^2 \leq \mathcal{E} + 1$.

6. Numerical Simulations

In this section, the global stability of the system’s equilibria will be illustrated numerically. The simulations were done using the appropriate commands/package in Matlab. We use the values of the parameters presented in **Table 1**. In addition, we make a comparison between single-infection and co-infection.

Table 1. Model parameters.

Parameter	Value	Parameter	Value	Parameter	Value
$\mu_h N_h$	0.3	β_h	0.2	α_h	0.06
$\mu_v N_v$	0.07	β_v	0.09	α_v	0.067
τ_h	0.02	ρ_h	3	γ_h	1.2
		m_h	2		

Stability of the Equilibria

Now, we will present some numerical simulations to approve our theoretical results of system (1) with parameters values given in **Table 1**. We consider three different initial conditions (states) as:

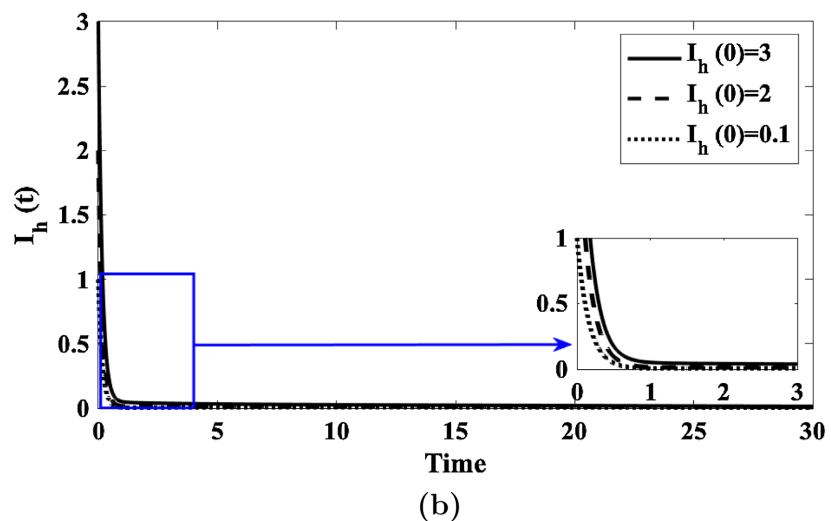
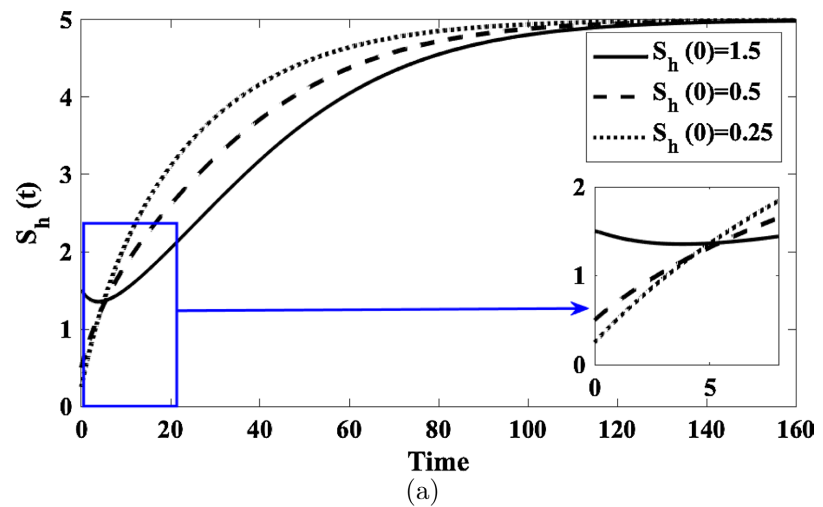
$$\mathbf{C1:} (S_h(0), I_h(0), R_h(0), S_v(0), I_v(0)) = (1.5, 3, 3, 0.8, 1),$$

$$\mathbf{C2:} (S_h(0), I_h(0), R_h(0), S_v(0), I_v(0)) = (0.5, 2, 2, 0.5, 0.5),$$

C3: $(S_h(0), I_h(0), R_h(0), S_v(0), I_v(0)) = (0.25, 1, 1, 0.25, 0.2)$.

Selecting the values of β_h, β_v and fixing the other parameters, **Table 1**, leads to the following situations:

Situation 1. (Stability of Disease-free equilibrium \mathcal{D}_0):, we have $\mathcal{R}_0 = 0.22 < 1$. **Figure 2** shows that the trajectories tend to the disease-free equilibrium $\mathcal{D}_0 = (5, 0, 0, 1.04, 0)$ for all initials. This demonstrates that, \mathcal{D}_0 is G.A.S based on Theorem 1. As time increases, one may notice, by taking $S_h = 1.5$ as an initial value, S_h decreases for a short time, then increases and tends to \mathcal{D}_0 . Taking a lower values for $S_h(0) (= 0.5, 0.25)$, it tends to increase and reach \mathcal{D}_0 . A similar behaviour was seen for S_v . The recovered human R_h , started at 3, increased for a short time, then increase to tend to \mathcal{D}_0 . By decreasing the starting value, the density of the recovered human tends to increase less but decreases after a short time. When the initial density of the infected population of human I_h starts at 3 will decrease as time increases, a similar behaviour was seen for I_v , where it increased for a shorter time and then decreased as time grew.



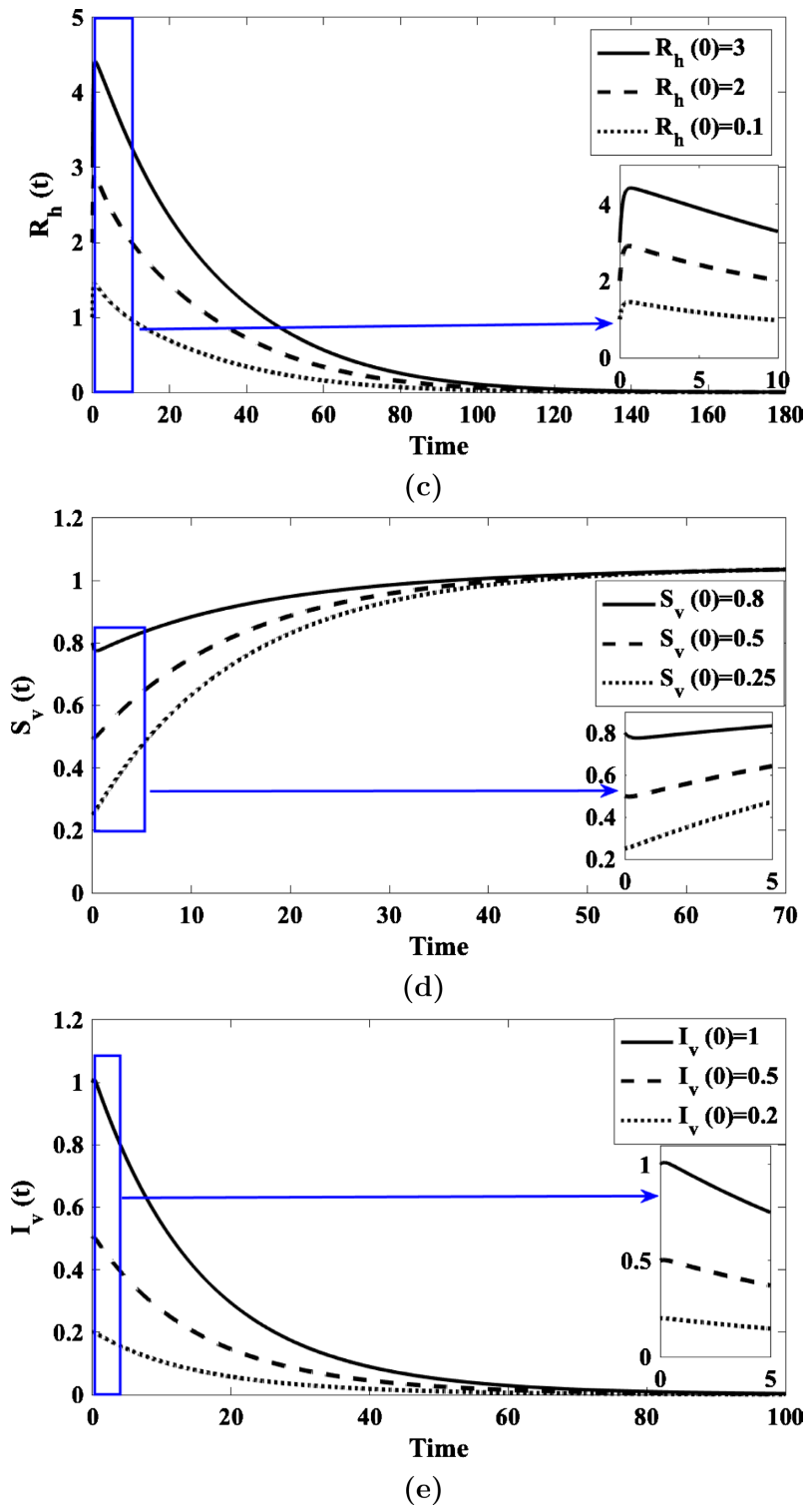
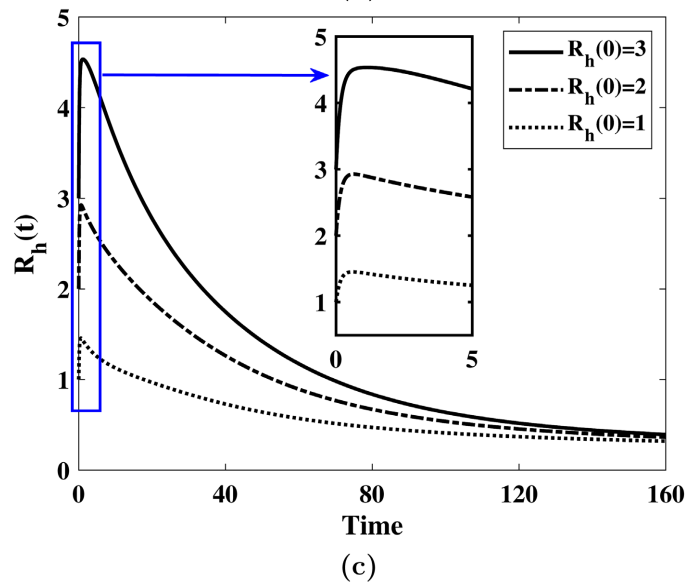
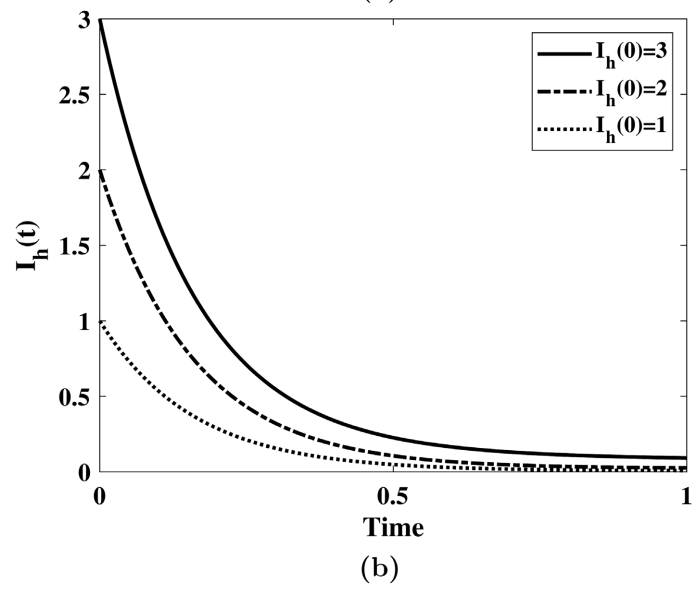
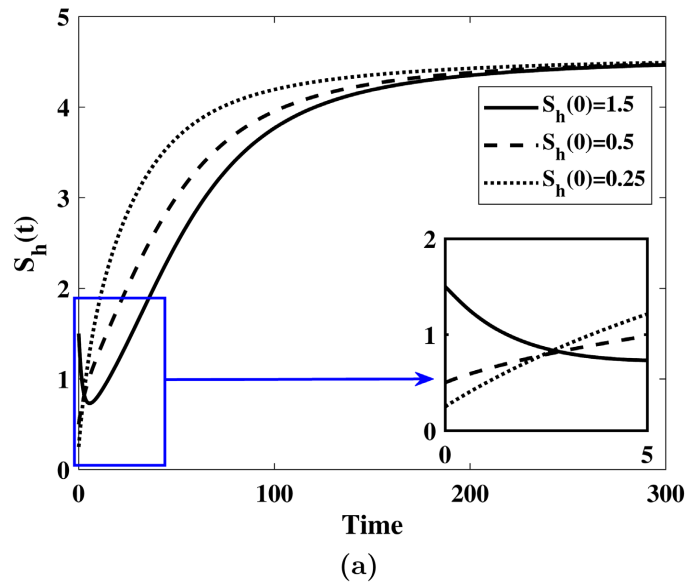


Figure 2. Solutions of system (1a) when $\mathcal{R}_0 \leq 1$, (a) Susceptible humans, (b) Infected humans, (c) Recovered humans, (d) Susceptible mosquitoes and (e) Infected mosquitoes

Situation 2. (Stability of Endemic equilibrium \mathcal{D}_1): In increasing the values of the transmission rate $\beta_h = 0.45$ and $\beta_v = 0.2$, $\mathcal{R}_0 = 1.0571 > 1$, **Figure 3** shows that: with increasing time, the variables of the system tend to the



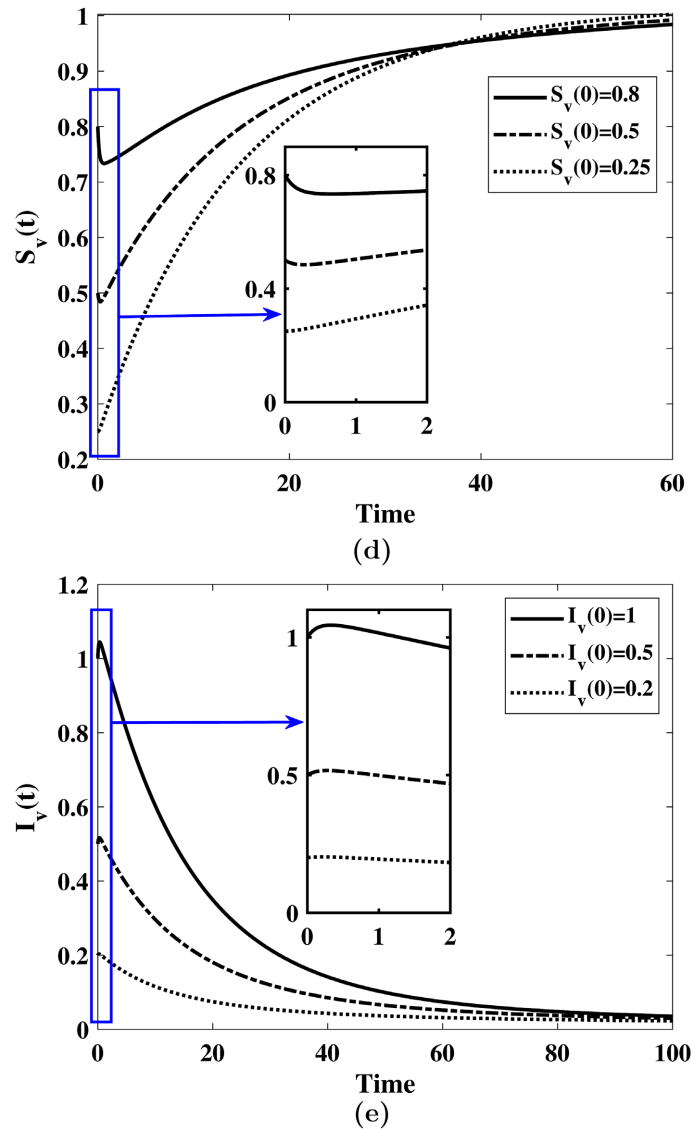


Figure 3. Solutions of system (1a) when $\mathcal{R}_0 \geq 1$, (a) Susceptible humans, (b) Infected humans, (c) Recovered humans, (d) Susceptible mosquitoes and (e) Infected mosquitoes

Endemic equilibrium $\mathcal{D}_1 = (4.5344, 0.0045, 0.2380, 1.0310, 0.0137)$ for the different initial conditions. A similar behaviour to situation 1 was observed but with a higher value of the drop-down and-up. This demonstrates that, \mathcal{D}_1 is G.A.S based on Theorem 2.

The figures show that with different initial values, the solution always tends to the G.A.S. equilibrium points when the data of the parameters involved within the system satisfy the condition on \mathcal{R}_0 .

7. Conclusion

The interaction rates of human/mosquitos had an impact on increasing the infectious disease as time increased. The environmental effect on Malaria spread is a very complex factor, climate change, heavy rainfall and humidity provide good

breeding areas for mosquitos to breed. The early diagnosis of these areas and the treatments or the controlling activities such as insecticidal spraying will help in decreasing the transmission rate and hence controlling the epidemic.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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