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Analysis of SIR Mathematical Model for Malaria disease with the inclusion of Infected Immigrants

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Abstract: *In this paper, the mathematical and stability analyses of the SIR model of malaria with the inclusion of infected immigrants are analyzed. The model consists of SIR compartments for the human population and SI compartments for the mosquito population. Susceptible humans become infected if they are bitten by infected mosquitoes and then they move from susceptible class to the infected class. In the similar fashion humans from infected class will go to recovered class after getting recovered from the disease. A susceptible mosquito becomes infected after biting an infected person and remains infected till death. The reproduction number R_0 of the model is calculated using the next generation matrix method. Local asymptotical stabilities of the steady states are discussed using the reproduction number. If the average number of secondary infections caused by an average infected, called the basic reproduction number, is less than one a disease will die out otherwise there will be an epidemic. The global stability of the equilibrium points is proved using the Lyapunov function and LaSalle Invariance Principle. The results of the mathematical analysis of the model are confirmed by the simulation study. It is concluded that the infected immigrants will contribute positively and increase the disease in the population. Thus, it is recommended to prevent infected immigrants so as to bring the disease under control.*

Keywords*: Infected immigrants, Reproduction number, Steady states, Local stability, Lyapunov function.*

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I. Introduction

Malaria is one of the diseases that have their presence constantly in human population. It is caused by the entry of the malaria parasite called *Plasmodium* into the bloodstream, due to the bite of an infected female *Anopheles* mosquito. A single bite by a malaria-carrying mosquito can lead to extreme sickness or death. Malaria starts with an extreme cold, followed by high fever and severe sweating. These symptoms can be accompanied by joint pains, abdominal pains, headaches, vomiting, and extreme fatigue [**1**].

According to the estimations of World Health Organization (WHO) in 2015, 3.2 billion persons were at risk of infection and 2.4 million new cases were detected with 438,000 cases of deaths. However sub-Saharan Africa remains the most vulnerable region with high rate of deaths due to malaria **[2]**.

To reduce the impact of malaria on the globe, considerable scientific efforts have been put forward including the construction and analysis of mathematical models. The first mathematical model to describe the transmission dynamics of malaria disease has been developed by Ross **[3]**. According to Ross, if the mosquito population can be reduced to below a certain threshold, then malaria can be eradicated from the human population. Later, Macdonald modified the Ross model by including super infection and shown that the reduction of the number of mosquitoes has a little effect on the epidemiology of malaria in areas of intense transmission **[4]**. Nowadays, several kinds of mathematical models have been developed so as to help the concerned bodies in reducing the death rate due to malaria **[4]**. In spite of the continuous efforts being made, it has still been remained difficult to eradicate malaria completely from the human world. Hence, there is a need for developing new models and for continuing research **[2]**.

The use of mathematical modeling has a significant role in understanding the theory and practice of malaria disease transmission and control. The mathematical modeling can be used in figuring out decisions that are of significant importance on the outcomes and provide complete examinations that enter into decisions in a way that human reasoning and debate cannot **[5]**.

Several health reports and studies in the literature address that malaria is increasing in rigorousness, causing significant public health and socioeconomic trouble **[6, 7]**. Malaria remains the world's most common vector-borne disease. Despite decades of global eradication and control efforts, the disease is reemerging in areas where control efforts were once effective and emerging in areas thought free of the disease. The global spread necessitates a concerted global effort to combat the spread of malaria. The present study illustrates the use of mathematical modeling and analysis to gain insight into the transmission dynamics of malaria in a

population, with main objective on determining optimal control measures. In order to manage the disease, one needs to understand the dynamics of the spread of the disease. Some health scientists have tried to obtain some insight in the transmission and elimination of malaria using mathematical modeling **[8]**.

Years have been exhausted in finding ways to control and completely remove malaria from the human population, but all efforts have been in vain. The disease was once endemic and confined to certain parts of the world, but has now even spread to areas which were previously free of the disease. Even when eradicated for a period of time, it recurs in certain areas repeatedly. One major factor which has contributed to the wide spread nature of malaria is infected human immigration and travel. An area with an uninfected population of mosquitoes can also get infected when an infected individual enters the area and is bitten by these mosquitoes **[9]**.

There are no dormant forms of malaria. If the parasite enters the body, it will surely cause a disease, unlike certain other conditions in which the diseased state does not occur even for years after infection. It is logical to assume that infected humans will be unable to travel or migrate due to the symptoms brought on by the disease. However, there is a period of around 10 days to 4 weeks from the moment of infection to the actual onset of disease, and unaware people might travel during this time. During this period, the disease cannot be diagnosed by blood tests either as the parasite multiplies in the liver, thus allowing the infection to be carried to a new place. Such people will become infected after a certain period of dormancy. As a result of this, immigration of infected people has a huge impact on the spread of malaria within, as well as, among populations **[9]**.

Even if the infected immigrants are not introducing the parasite to a new population, their entry into an already infected population will cause an increase in the infected mosquitoes of the area as they will be biting more number of infected people. Therefore this paper will present the effect of infected immigrants on the spread and dynamics of Malaria by using an SIR mathematical model.

In this paper, the disease-free equilibrium points are calculated and the reproduction number of the model is formulated. Analysis of the stability of these disease-free equilibrium points are also given in detail. The local and global stability analysis of the disease-free equilibrium points are determined by the basic reproduction number.

This paper is organized as follows. In section 2, the mathematical model of the problem is formulated. Section 3 provides the mathematical analysis of the model. In section 4, numerical simulations are performed. In order to illustrate the mathematical model given the results and discussion are given in section 5. In the last section, section 6, conclusions are drawn for the results discussed for the given model.

II. Formulation of the Model

The endemic model of malaria transmission considered in this study is *SIR* in human population and *SI* in mosquito population. The model is formulated for the spread of malaria in the human and mosquito population with the total population size at time *t* denoted by $N_h(t)$ and $N_v(t)$ respectively.

The human populations are further compartmentalized into epidemiological classes as susceptible $S_h(t)$, infected $I_h(t)$, and recovered $R_h(t)$. The mosquito populations are similarly compartmentalized into epidemiological classes as susceptible $S_v(t)$ and infected $I_v(t)$. The vector component of the model does not include an immune class as mosquitoes never recover from the infection, that is, their infected period ends with their death due to their relatively short lifecycle. Hence $N_h(t) = S_h(t) + I_h(t) + R_h(t)$ and $N_v(t) = S_v(t) + I_v(t)$.

Thus, the immune class in the mosquito population is negligible and natural death occurs equally in all groups. The model can be used for diseases that persist in a population for a long period of time with vital dynamics. The present basic model is built on a set of assumptions mentioned as follows: Both the human and vector total population sizes are assumed to be constants. The recovered individuals in human population develop permanent immunity. The populations in compartments of both humans and vectors are non-negative which are proved in theorem 1, and so are all the parameters involved in the model (See Table 1). All newborns are susceptible to infection and the development of malaria starts when the infected female mosquito bites the human host. Individuals move from one class to the other as their status with respect to the disease evolves. Humans enter the susceptible class through birth rate μ_h and recruitment rate π_h, leave from the susceptible class through death rate α_h and infected rate $\beta_h S_h$. Human enter the infected class through immigration rate $\delta_h I_h$ and infected rate $\beta_h S_h$. It leaves the infected class through the recovered rate $\gamma_h I_h$. All human individuals, whatever their status, are subject to a natural death, which occurs at a rate α_h and disease induced death rate ρ_h.

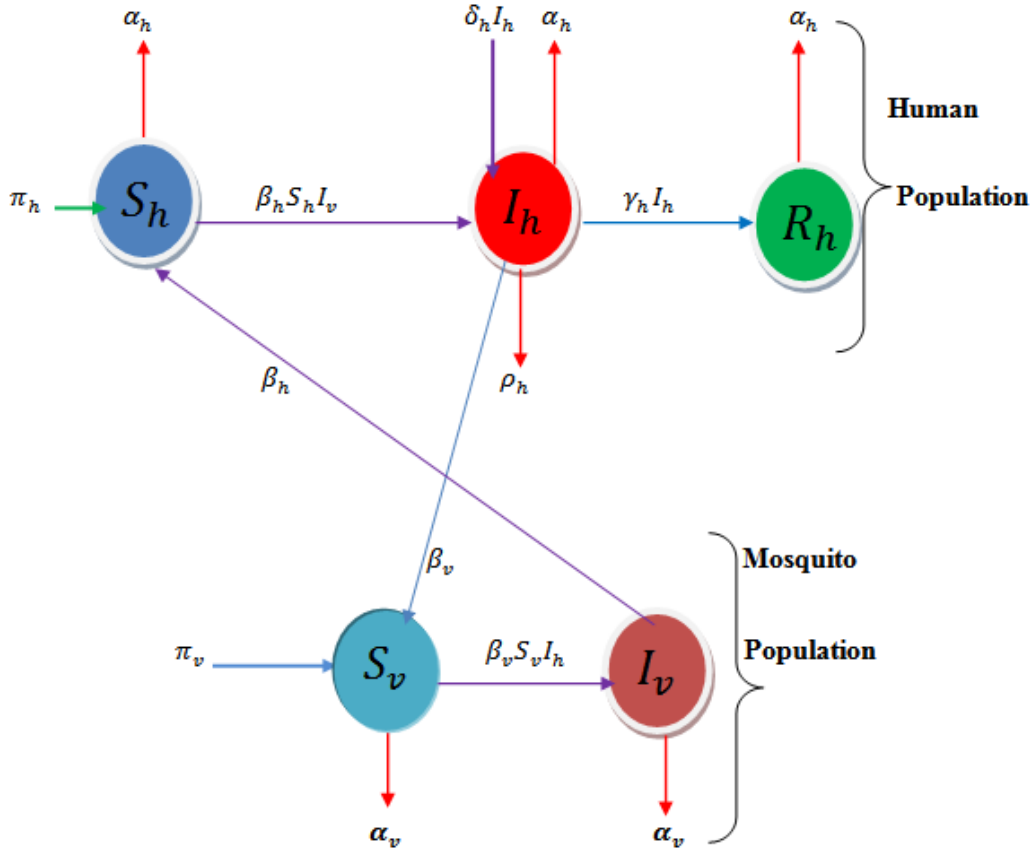

Table 1 Descriptions of model variables

$\boldsymbol{S_h}$**:** Number of susceptible humans
$\boldsymbol{I_h}$**:** Number of infected humans
$\boldsymbol{R_h}$**:** Number of recovered humans
$\boldsymbol{S_v}$**:** Number of susceptible mosquitoes
$\boldsymbol{I_v}$**:** Number of infected mosquitoes
$\boldsymbol{N_h}$**:** The total human population.
$\boldsymbol{N_v}$**:** The total mosquito population

Table 2 Descriptions of model parameters

$\boldsymbol{\alpha_h}$: Natural death rate for humans
$\boldsymbol{\rho_h}$: Disease-induced death rate for humans
$\boldsymbol{\delta_h}$: Infected migration rate for humans
$\boldsymbol{\beta_h}$: The human contact rate
$\boldsymbol{\alpha_v}$: Natural death rate for mosquitoes
$\boldsymbol{\gamma_h}$: Recovery rate for humans
$\boldsymbol{\pi_h}$: Recruitment rate of humans
$\boldsymbol{\beta_v}$: The mosquito contact rate
$\boldsymbol{\pi_v}$: Recruitment rate of mosquitoes

By considering the above assumptions and the notations of variables and parameters, the ordinary differential equations describing the dynamics of malaria in the human and mosquito populations take the form as

$$dS_h/dt = \pi_h - \beta_h S_h I_v - \alpha_h S_h \quad (1)$$

$$dI_h/dt = \beta_h S_h I_v + \delta_h I_h - \rho_h I_h - \gamma_h I_h - \alpha_h I_h \quad (2)$$

$$dR_h/dt = \gamma_h I_h - \alpha_h R_h \quad (3)$$

$$dS_v/dt = \pi_v - \beta_v S_v I_h - \alpha_v S_v \quad (4)$$

$$dI_v/dt = \beta_v S_v I_h - \alpha_v I_v \quad (5)$$

Further, the initial conditions of the model are denoted by $S_h(0) = S_{h0}$, $I_h(0) = I_{h0}$, $R_h(0) = R_{h0}$ and $S_v(0) = S_{v0}$, $I_v(0) = I_{v0}$. The total population sizes N_h and N_v of humans and mosquitoes can be determined by

$$S_h + I_h + R_h = N_h \quad (6)$$

$$N_h + I_v = N_v \quad (7)$$

In this model, the terms $\alpha_h S_h$, $\alpha_h I_h$ and $\alpha_h R_h$ refer to the total number of removed susceptible, infected and recovered humans per unit of time due to natural death. The terms $\alpha_v S_v$ and $\alpha_v I_v$ are the number of removed susceptible and infected mosquito populations per unit of time due to natural death. The term $\rho_h I_h$ is the number of removed human population because of the disease per unit of time, whereas $\gamma_h I_h$ is the total recovered human population per unit of time. The term $\beta_h S_h I_v$ denotes the rate at which the infected human hosts I_h get infected by the mosquito vector I_v, and $\beta_v S_v I_h$ refers to the rate at which the susceptible mosquitoes S_v are infected by the infected human hosts I_h at a time t. Thus, both the terms $\beta_h S_h I_v$ and $\beta_v S_v I_h$ are important parts of the model as they describe the interactions between the two populations.

III. Mathematical analysis of the model

The mathematical analysis of the model described by the system (1-5) is presented here. The model represented by the systems of coupled differential equation (1-5) will be analyzed in the feasible region and since the model represents the populations all the state variables and the parameters are assumed to be positive. The invariant region for the model (1-5) is

$$\Omega_h = \{ (S_h, R_h, I_h) \in \mathbb{R}^3_+ : S_h + R_h + I_h \leq \pi_h/\alpha_h \}$$

$$\Omega_v = \{ (S_v, I_v) \in \mathbb{R}^2_+ : S_v + I_v \leq \pi_v/\alpha_v \}$$

Therefore, the solutions of the system of ordinary differential equations (1-5) are feasible for all $t > 0$ if they enter the invariant region $\Omega = \Omega_h \times \Omega_v$.

3.1 Positivity of the solutions

In order that the model equations (1-5) are biologically and epidemiologically meaningful and well posed it is appropriate to show that the solutions of all the state variables are non-negative. This requirement is stated as a theorem and its proof is provided as follows:

Theorem 1*:* If $S_h(0) > 0$, $I_h(0) > 0$, $R_h(0) > 0$, $S_v(0) > 0$ and $I_v(0) > 0$ then the solution region $\{S_h(t), I_h(t), R_h(t), S_v(t), I_v(t)\}$ of the system of equations (1-5) is always non-negative.

Proof: To show the positivity of the solution of the dynamical system (1-5), each differential equation is considered separately and shown that its solution is positive.

Positivity of infected mosquito population: Considering the fifth differential equation of the system of differential equations (1-5) it can be shown that $dI_v/dt = \beta_v S_v I_h - \alpha_v I_v \geq -\alpha_v I_v$. Now, separation of the variables reduces it to $dI_v/I_v \geq -\alpha_v dt$. On integrating it yields to the solution $I_v(t) \geq I_{v0}\, e^{-\int_0^t \alpha_v ds} > 0$. Thus, it is clear from the solution that $I_v(t)$ is positive since the initial value I_{v0} and the exponential functions are always positive.

Positivity of infected human population: Considering the second differential equation of the system of differential equations (1-5) and that can be rewritten as $dI_h/dt = \beta_h S_h I_v + \delta_h I_h - (\rho_h + \gamma_h + \alpha_h) I_h \geq -(\rho_h + \gamma_h + \alpha_h) I_h$. Separating the variables it yields to $dI_h/I_h \geq -(\rho_h + \gamma_h + \alpha_h) dt$. Further, integrate to find the solution as $I_h(t) \geq I_{h0}\, e^{-\int_0^t (\rho_h + \gamma_h + \alpha_h) ds} > 0$. It is clear from the solution that $I_h(t)$ is positive since $I_{h0} > 0$ and the exponential function is always positive.

Positivity of susceptible human population: Considering the first differential equation of the system of differential equations (1-5) it can be shown that $dS_h/dt = \pi_h - \beta_h S_h I_v - \alpha_h S_h$.

Since π_h is a positive quantity, the equation can be expressed as an inequality as $dS_h/dt \geq -\beta_h S_h I_v - \alpha_h S_h$. Using the technique of separation of variables and up on integration gives $S_h(t) \geq S_{h0} e^{-\int_0^t (\beta_h I_v + \alpha_h) ds}$. But, for any value of the exponent, the exponential term is always a non-negative quantity, that is $e^{-\int_0^t (\beta_h I_v + \alpha_h) ds} \geq 0$. Also it is assumed that $S_{h0} > 0$. Thus, it is clear from the solution that $S_h(t)$ is positive

Positivity of susceptible mosquito population: By observing at the fourth differential equation of the dynamical systems (1-5) and that can be expressed as $dS_v/dt = \pi_v - \beta_v S_v I_h - \alpha_v S_v$. Since π_v is a positive quantity it can be rewritten as $dS_v/S_v \geq -(\beta_v I_h + \alpha_v) dt$

Now, integration leads to the solution $S_v(t) \geq S_{v0} e^{-\int_0^t (\beta_v I_h + \alpha_v) ds}$. Note that for any value of the exponent, the exponential term is always a non-negative quantity, that is $e^{-\int_0^t (\beta_v I_h + \alpha_v) ds} \geq 0$

Thus, it is clear from the solution that $S_v(t)$ is positive since $S_{v0} > 0$ and the exponential functions are always positives.

Positivity of recovered human population: Consider the third differential equation of the system of differential equations (1-5) and express it as $dR_h/dt = \gamma_h I_h - \alpha_h R_h \geq -\alpha_h R_h$. Now, separation of the variables leads to $dR_h/R_h \geq -\alpha_h dt$. Further, the integration gives the solution as $R_h(t) \geq R_{h0} e^{-\int_0^t \alpha_h ds} > 0$. It is clear from the solution that $R_h(t)$ is positive since $R_{h0} > 0$ and also the exponential function is always positive.

3.2 Boundedness of the solution region

In order that the model equations (1-5) are biologically and epidemiologically meaningful and well posed it is appropriate to show that the solutions of all the state variables are bounded. This requirement is stated as a theorem and its proof is provided as follows:

Theorem 2: The non-negative solutions characterized by theorem 1 are bounded.

Proof: It suffices to prove that the total living population size is bounded for all $t > 0$. That is, the solutions lie in the bounded region.

Boundedness of total human population: The rate of change of total human population size $N_h(t) = S_h(t) + Iht + Rht$ can be obtained as $dNhdt = dShdt + dIhdt + dRhdt = \pi h - \beta hShIv - \alpha h Sh + \beta hShIv + \delta hIh - \rho hIh - \gamma hIh - \alpha hIh + \gamma hIh - \alpha hRh$. After simplification it reduces to $dNhtdt = \pi_h - \alpha_h N_h + (\delta_h - \rho_h)$. Further, in case if the death rate of humans due to malaria disease is considered to be zero, i.e., $(\delta_h - \rho_h) = 0$ then it is obtained as $dN_h(t)/dt = \pi_h - \alpha_h N_h$. The solution of this differential equation is found to be $N_h(t) = \pi_h/\alpha_h + [N_{h0} - \pi_h/\alpha_h]e^{-\alpha_h t}$ showing that $N_h(t) \to \pi_h/\alpha_h$ as $t \to \infty$. The term N_{h0} denotes the initial total human population and is a positive quantity. It can be interpreted that the total human population grows and asymptotically converges to a positive quantity given by π_h/α_h under the condition that humans do not die due to malaria infection. Thus π_h/α_h is an upper bound of the total human population $N_h(t)$ that is $N_h(\infty) \leq \pi_h/\alpha_h$. Whenever the initial human population starts off low below π_h/α_h then it grows over time and finally reaches the upper asymptotic value π_h/α_h. Similarly, whenever the initial human population starts off higher than π_h/α_h then it decays over time and finally reaches the lower asymptotic value π_h/α_h.

Boundedness of total mosquito population: Just similar to the above, the rate of change of total mosquito population size $N_v(t) = S_v(t) + I_v(t)$ can be obtained by adding up the fourth and fifth equations of model (1-5) as $[dN_v(t)/dt] = [dS_v(t)/dt] + [dI_v(t)/dt] = \pi_v - \beta_v S_v I_h - \alpha_v S_v + \beta_v S_v I_h - \alpha_v I_v$. Further, it is simplified as $[dN_v(t)/dt] = \pi_v - \alpha_v N_v$. Thus, the solution of this differential equation is found to be $N_v(t) = \pi_v/\alpha_v + [N_{v0} - (\pi_v/\alpha_v e^{-\alpha_v t})]$. This shows that $N_v(t) \to \pi_v/\alpha_v$ as $t \to \infty$ since the term N_{v0} denotes the initial total mosquito population and it a positive quantity. It can be interpreted that the total mosquito population grows and asymptotically converges to a positive quantity given by π_v/α_v. Thus, π_v/α_v is an upper bound of the total mosquito population $N_v(t)$ i.e. $N_v(\infty) \leq \pi_v/\alpha_v$.

3.3 Disease Free Equilibrium

Disease – free equilibrium points are steady state solutions where there is no malaria in the human population or plasmodium parasite in the mosquito population. We can define the diseased classes as the human or mosquito populations that are infected that is, I_h and I_v. In the absence of the disease this implies that $I_h = 0$ and $I_v = 0$ and when the right hand side of the fourth and fifth differential equations of a non –linear system differential equations (1-5) is set zero we have:

$$\pi_h - \beta_h S_h I_v - \alpha_h S_h = 0 \quad (8)$$

$$\beta_h S_h I_v + \delta_h I_h - \rho_h I_h - \gamma_h I_h - \alpha_h I_h = 0 \quad (9)$$

$$\gamma_h I_h - \alpha_h R_h = 0 \quad (10)$$

$$\pi_v - \beta_v S_v I_h - \alpha_v S_v = 0 \quad (11)$$

$$\beta_v S_v I_h - \alpha_v I_v = 0 \quad (12)$$

The above equations (8) to (12) reduce to a pair of relations as $\pi_h - \alpha_h S_h = 0$ and $\pi_v - \alpha_v S_v = 0$. Further, these imply that ${S_h}^0 = \pi_h/\alpha_h$ and ${S_v}^0 = \pi_v/\alpha_v$. Thus, the disease – free equilibrium point of the malaria model formulated in (1-5) above is given by

$$E_0 = \{S_h^0, \quad I_h^0, \quad R_h^0, \quad S_v^0, \quad I_v^0\} = \{\pi_h/\alpha_h, \quad 0, \quad 0, \quad \pi_v/\alpha_v, \quad 0\} \quad (13)$$

Thus, the state E_0 represents that there is no infection or the malaria disease is absent in both the human and mosquito populations.

3.4 Basic Reproduction Number

Generally, the next generation operator approach as described by Diekmann et al. (1990) is used to find the basic reproduction number R_0 as the number of secondary infections that one infected individual would create over the duration of the infected period, provided that everyone else is susceptible. Reproduction number R_0 is the threshold for many epidemiology models as it determines whether a disease can invade a population or not. When $R_0 < 1$ each infected individual produces on average less than one new infected individual so it is expected that the disease dies out. On the other hand if $R_0 > 1$ then each individual produces more than one new infected individual so it is expected that the disease would spread in the population. This means that the threshold quantity for eradicating the disease is to reduce the value of R_0 to be less than one. The following steps are followed to determine the basic reproduction number R_0 by using the next generation approach.

In the next generation method, R_0 is defined as the largest eigenvalue of the next generation matrix. The formulation of this matrix involves determining two classes, infected and non-infected, from the model. That is, the basic reproduction number cannot be determined from the structure of the mathematical model alone but depends on the definition of infected and uninfected compartments. Assuming that there are n compartments of which the first m compartments to infected individuals **[12]**.

Let $V_i(x) = V_i^-(x) - V_i^+(x)$ where $V_i^+(x)$ is the rate of transfer of individuals into compartment i by all other means and $V_i^-(x)$ is the rate of transfer of individual out of the i^{th} compartment. It is assumed that each function is continuously differentiable at least twice in each variable. The disease transmission model consists of nonnegative initial conditions together with the following system of equations: $\dot{x}_i = h_i(x) = F_i(x) - V_i(x),\ i = 1,2,3,\ldots n$ where $\dot{x}$ is the rate of change of x.

The next is the computation of the square matrices F and V of order $m \times m$, where m is the number of infected classes, defined by $F = [\partial F_i(x_0)/\partial x_j]$ and $V = [\partial V_i(x_0)/\partial x_j]$ with $1 \le i, j \le m$, such that F is nonnegative, V is a non-singular matrix and x_0 is the disease – free equilibrium point (DFE).

Since F is nonnegative and V nonsingular, then V^{-1} is nonnegative and also FV^{-1} is nonnegative. Hence the matrix of FV^{-1} is called the next generation matrix for the model.

Finally the basic reproduction number R_0 is given by

$$R_0 = \rho(FV^{-1}) \tag{14}$$

Here in (14), $\rho(A)$ denotes the spectral radius of matrix A and the spectral radius is the biggest nonnegative eigenvalue of the next generation matrix. Hence, the column matrices F_i and V_i are defined as

$$F_i = \begin{bmatrix} \beta_h S_h I_v \\ \beta_v S_v I_h \end{bmatrix} \tag{15}$$

$$V_i = \begin{bmatrix} (\rho_h + \gamma_h + \alpha_h - \delta_h) I_h \\ \alpha_v I_v \end{bmatrix} \tag{16}$$

The partial derivatives of (8) with respect to $(I_h,\ I_v)$ and the Jacobian matrix of F_i at the disease – free equilibrium point (6) takes the form as

$$F = \begin{bmatrix} 0 & \beta_h S_h \\ \beta_v S_v & 0 \end{bmatrix} = \begin{bmatrix} 0 & \beta_h \pi_h/\alpha_h \\ \beta_v \pi_v/\alpha_v & 0 \end{bmatrix} \tag{17}$$

Similarly, the partial derivatives of (16) with respect to (I_h, I_v) and the Jacobian matrix of V_i at the disease – free equilibrium point (13) takes the form as

$$V = \begin{bmatrix} \rho_h + \gamma_h + \alpha_h - \delta_h & 0 \\ 0 & \alpha_v \end{bmatrix} \tag{18}$$

The inverse of the matrix V is given as

$$V^{-1} = \begin{bmatrix} (\rho_h + \gamma_h + \alpha_h - \delta_h)^{-1} & 0 \\ 0 & \alpha_v{}^{-1} \end{bmatrix} \tag{19}$$

Now both FV^{-1} and $FV^{-1}(E_0)$ are computed as

$$FV^{-1} = \begin{bmatrix} 0 & \beta_h \pi_h/\alpha_h \alpha_v \\ \beta_v \pi_v/\alpha_v(\rho_h + \gamma_h + \alpha_h - \delta_h) & 0 \end{bmatrix} \tag{20}$$

$$FV^{-1}(E_0) = \begin{bmatrix} 0 & \beta_h \pi_h/\alpha_h \alpha_v \\ \beta_v \pi_v/\alpha_v(\rho_h + \gamma_h + \alpha_h - \delta_h) & 0 \end{bmatrix} \tag{21}$$

From (21), it is now possible to calculate the eigenvalues to determine the basic reproduction number R_0 by taking the spectral radius of the matrix FV^{-1}. Thus it is computed by $|FV^{-1}(E_0) - \lambda I| = 0$, and it yields

$$\lambda = \pm\sqrt{\left(\frac{\beta_h \pi_h}{\alpha_h \alpha_v}\right)\left(\frac{\beta_v \pi_v}{\alpha_v(\rho_h + \gamma_h + \alpha_h - \delta_h)}\right)}$$

Thus, the dominant eigenvalue of the matrix FV^{-1} or the basic reproduction number is given by

$$R_0 = \sqrt{\left(\frac{\beta_h \pi_h}{\alpha_h \alpha_v}\right)\left(\frac{\beta_v \pi_v}{\alpha_v(\rho_h + \gamma_h + \alpha_h - \delta_h)}\right)} \tag{22}$$

From this, it can be quantified that higher values of β_h, π_h, β_v and π_v can allow the outbreak of the disease. Conversely, for small values of β_h, π_h, β_v and π_v the disease dies out. The reproduction number is a powerful parameter which measures the existence and stability of the disease in the human and mosquito population. If $(\beta_h \pi_h)(\beta_v \pi_v) < (\alpha_h \alpha_v^{\,2})(\rho_h + \gamma_h + \alpha_h - \delta_h)$, i.e., $R_0 < 1$ the disease-free equilibrium is the only equilibrium point and then the disease dies out. If $(\beta_h \pi_h)(\beta_v \pi_v) < (\alpha_h \alpha_v^{\,2})(\rho_h + \gamma_h + \alpha_h - \delta_h)$ i.e., $R_0 > 1$ the unique endemic equilibrium exists and the disease persists within the human and mosquito population.

3.5 Stability analysis of the Disease Free Equilibrium point

The equilibriums are obtained by equating the right hand side of the system of differential equations (1-5) to zero. Disease-free equilibrium (DFE) of the model is the steady-state solution of the model in the absence of the malaria disease. Hence, the DFE of the given malaria model (1-5) is given by

$$E_0 = \{S_h^0, \quad I_h^0, \quad R_h^0, \quad S_v^0, \quad I_v^0\} = \{\pi_h/\alpha_h, \quad 0, \quad 0, \quad \pi_v/\alpha_v, \quad 0\}$$

Theorem 1: The DFE, E_0 of the system (1-5) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.
Proof: Consider the following functions so as to find the Jacobian matrix:

$dS_h/dt = \pi_h - \beta_h S_h I_v - \alpha_h S_h = f_1\ (S_h, \quad I_h, \quad R_h, \quad S_v, \quad I_v)$
$dI_h/dt = \beta_h S_h I_v + \delta_h I_h - \rho_h I_h - \gamma_h I_h - \alpha_h I_h = f_2(S_h, \quad I_h, \quad R_h, \quad S_v, \quad I_v)$
$dR_h/dt = \gamma_h I_h - \alpha_h R_h = f_3(S_h, \quad I_h, \quad R_h, \quad S_v, \quad I_v)$
$dS_v/dt = \pi_v - \beta_v S_v I_h - \alpha_v S_v = f_4(S_h, \quad I_h, \quad R_h, \quad S_v, \quad I_v)$
$dI_v/dt = \beta_v S_v I_h - \alpha_v I_v = f_5(S_h, \quad I_h, \quad R_h, \quad S_v, \quad I_v)$

Thus, the Jacobian matrix is given by

$$J = \begin{pmatrix} \partial f_1/\partial S_h & \partial f_1/\partial I_h & \partial f_1/\partial R_h & \partial f_1/\partial S_v & \partial f_1/\partial I_v \\ \partial f_2/\partial S_h & \partial f_2/\partial I_h & \partial f_2/\partial R_h & \partial f_2/\partial S_v & \partial f_2/\partial I_v \\ \partial f_3/\partial S_h & \partial f_3/\partial I_h & \partial f_3/\partial R_h & \partial f_3/\partial S_v & \partial f_3/\partial I_v \\ \partial f_4/\partial S_h & \partial f_4/\partial I_h & \partial f_4/\partial R_h & \partial f_4/\partial S_v & \partial f_4/\partial I_v \\ \partial f_5/\partial S_h & \partial f_5/\partial I_h & \partial f_5/\partial R_h & \partial f_5/\partial S_v & \partial f_5/\partial I_v \end{pmatrix}$$

Hence,

$$J = \begin{pmatrix} -\beta_h I_v - \alpha_h & 0 & 0 & 0 & -\beta_h S_h \\ \beta_h I_v & \delta_h - \rho_h - \gamma_h - \alpha_h & 0 & 0 & \beta_h S_h \\ 0 & \gamma_h & -\alpha_h & 0 & 0 \\ 0 & -\beta_v S_v & 0 & -\alpha_v & 0 \\ 0 & \beta_v S_v & 0 & \beta_v I_h & -\alpha_v \end{pmatrix}$$

Therefore, the Jacobian matrix of model formulated at the disease-free equilibrium E_0 is

$$J(E_0) = \begin{pmatrix} -\alpha_h & 0 & 0 & 0 & -\beta_h \pi_h / \alpha_h \\ 0 & \delta_h - \rho_h - \gamma_h - \alpha_h & 0 & 0 & \beta_h \pi_h / \alpha_h \\ 0 & \gamma_h & -\alpha_h & 0 & 0 \\ 0 & -\beta_v \pi_v / \alpha_v & 0 & -\alpha_v & 0 \\ 0 & \beta_v \pi_v / \alpha_v & 0 & 0 & -\alpha_v \end{pmatrix} \quad (23)$$

The characteristic equation $|J(E_0) - \lambda I| = 0$ of (23) is expanded and simplified as follows:

$$\begin{vmatrix} -\alpha_h - \lambda & 0 & 0 & 0 & -\beta_h \pi_h / \alpha_h \\ 0 & (\delta_h - \rho_h - \gamma_h - \alpha_h) - \lambda & 0 & 0 & \beta_h \pi_h / \alpha_h \\ 0 & \gamma_h & -\alpha_h - \lambda & 0 & 0 \\ 0 & -\beta_v \pi_v / \alpha_v & 0 & -\alpha_v - \lambda & 0 \\ 0 & \beta_v \pi_v / \alpha_v & 0 & 0 & -\alpha_v - \lambda \end{vmatrix} = 0$$

$$(\alpha_h + \lambda) \begin{vmatrix} (\delta_h - \rho_h - \gamma_h - \alpha_h) - \lambda & 0 & 0 & \beta_h \pi_h / \alpha_h \\ \gamma_h & -\alpha_h - \lambda & 0 & 0 \\ -\beta_v \pi_v / \alpha_v & 0 & -\alpha_v - \lambda & 0 \\ \beta_v \pi_v / \alpha_v & 0 & 0 & -\alpha_v - \lambda \end{vmatrix} = 0$$

$$-(\alpha_h + \lambda)^2 \begin{vmatrix} (\delta_h - \rho_h - \gamma_h - \alpha_h) - \lambda & 0 & \beta_h \pi_h / \alpha_h \\ -\beta_v \pi_v / \alpha_v & -\alpha_v - \lambda & 0 \\ \beta_v \pi_v / \alpha_v & 0 & -\alpha_v - \lambda \end{vmatrix} = 0$$

$$(\alpha_h + \lambda)^2 (-\alpha_v - \lambda) \{ [(\delta_h - \rho_h - \gamma_h - \alpha_h) - \lambda](-\alpha_v - \lambda) - (\beta_h \pi_h / \alpha_h)(\beta_v \pi_v / \alpha_v) \} = 0$$

$$(\alpha_h + \lambda)^2 (-\alpha_v - \lambda) [\lambda^2 + \omega\lambda + \alpha_v \omega - (\beta_h \pi_h / \alpha_h)(\beta_v \pi_v / \alpha_v)] = 0$$

$$(\alpha_h + \lambda)^2 = 0, \quad (\alpha_v + \lambda) = 0, \qquad [\lambda^2 + \omega\lambda + \alpha_v \omega(1 - {R_0}^2)] = 0$$

Here $\omega = \rho_h + \gamma_h + \alpha_h - \delta_h$. Hence the eigenvalues of (16) are

$\lambda_1 = \lambda_2 = -\alpha_h < 0$

$\lambda_3 = -\alpha_v < 0$

$$\lambda_4 = \frac{-\omega - \sqrt{\omega^2 - 4\alpha_v \omega(1 - {R_0}^2)}}{2} < 0$$

$$\lambda_5 = \frac{-\omega + \sqrt{\omega^2 - 4\alpha_v \omega(1 - {R_0}^2)}}{2} < 0 \; if \; R_0 < 1$$

Therefore the DFE, E_0 of the system of differential equations (1-5) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Theorem 2: If $R_0 < 1$, then the disease free equilibrium point E_0 is globally asymptotically stable and the disease dies out, but if $R_0 > 1$, then E_0 is unstable.

Proof: Consider the following Lyapunov function to show the global stability of E_0.

$$V(I_h, I_v) = (\alpha_v/\beta_h)I_h + I_v$$

Then, the time derivative of V is

$$dV/dt = (\alpha_v/\beta_h)(dI_h/dt) + (dI_v/dt)$$
$$dV/dt = (\alpha_v/\beta_h)\left(\beta_h S_h I_v + \delta_h I_h - \rho_h I_h - \gamma_h I_h - \alpha_h I_h\right) + \beta_v S_v I_v - \alpha_v I_v$$
$$dV/dt = (\alpha_v/\beta_h)\beta_h S_h I_v - (\alpha_v/\beta_h)\left(\rho_h + \gamma_h + \alpha_h - \delta_h\right)I_h + \beta_v S_v I_h - \alpha_v I_v$$
$$dV/dt = \alpha_v S_h I_v - (\alpha_v/\beta_h)\left(\rho_h + \gamma_h + \alpha_h - \delta_h\right)I_h + \beta_v S_v I_h - \alpha_v I_v$$
$$dV/dt = \beta_v S_v I_h - (\alpha_v/\beta_h)\left(\rho_h + \gamma_h + \alpha_h - \delta_h\right)I_h + \alpha_v S_h I_v - \alpha_v I_v$$
$$dV/dt = \left[\beta_v S_v - (\alpha_v/\beta_h)\left(\rho_h + \gamma_h + \alpha_h - \delta_h\right)\right]I_h - \alpha_v(1 - S_h)I_v$$
$$dV/dt \leq \left[\beta_v S_v - (\alpha_v/\beta_h)\left(\rho_h + \gamma_h + \alpha_h - \delta_h\right)\right] I_h$$
$$dV/dt = \left[\left(\beta_v \pi_v/\alpha_v\right) - (\alpha_v/\beta_h)\left(\rho_h + \gamma_h + \alpha_h - \delta_h\right)\right] I_h$$
$$dV/dt = \left(\left[{R_0}^2 \alpha_h \alpha_v\left(\rho_h + \gamma_h + \alpha_h - \delta_h\right)/\beta_h \pi_h\right] - (\alpha_v/\beta_h)\left(\rho_h + \gamma_h + \alpha_h - \delta_h\right)\right) I_h$$
$$dV/dt = (\alpha_v/\beta_h)\left(\rho_h + \gamma_h + \alpha_h - \delta_h\right)\left[(\alpha_h/\pi_h){R_0}^2 - 1\right]I_h$$

Thus, it is possible to establish that $dV/dt < 0$ if $R_0 < 1$ and $dV/dt = 0$ if $I_h = 0$, $I_v = 0$ since α_h/π_h is always less than one. Therefore, the largest compact invariant set in $\{(S_h, I_h, R_h, S_v, I_v) \in \Omega : dV/dt = 0\}$ is the singleton set $E0$ in Ω. From LaSalle's invariant principle [10], every solution that starts in the region Ω approaches E_0 as $t \to \infty$ and hence the DFE E_0 is globally asymptotically stable $R_0 < 1$ in Ω.

IV. Numerical Simulations

In this section a numerical simulations of the model is presented which is carried out using a DE Discover 2.6.4. The values of the parameters used in the model are given in Table 3 and 4.

Table 3 Parameter values

Parameter	Values	Reference
α_h	0.05	[11]
π_v	25	Assumed
δ_h	0.001	Assumed
β_h	0.01	[11]
α_v	2	Assumed
γ_h	0.9	[11]
π_h	10	Assumed
β_v	0.005	[11]
ρ_h	0.01	[11]

Assuming the parameter values in table 3 with the initial conditions $S_{h0} = 120$, $I_{h0} = 20$, $R_{h0} = 18$, $S_{v0} = 110$ and $I_{v0} = 240$ were used for the simulation shown in figure 2 below. In figure 2, the fractions of the populations S_h, I_h, R_h, S_v and I_v are plotted versus time. The susceptible human populations will initially decreases with time and then increases and the fractions of infected human populations decrease. The reproduction number is less than one and thus the disease free equilibrium point $E_0 = (S_h^0, I_h^0, R_h^0, S_v^0, I_v^0) = (\pi_h/\alpha_h, 0, 0, \pi_v/\alpha_v, 0)$ is stable. The susceptible and infected mosquito population decreases over time as shown in the figure 2 indicating that the malaria outbreak will not occur in the population.

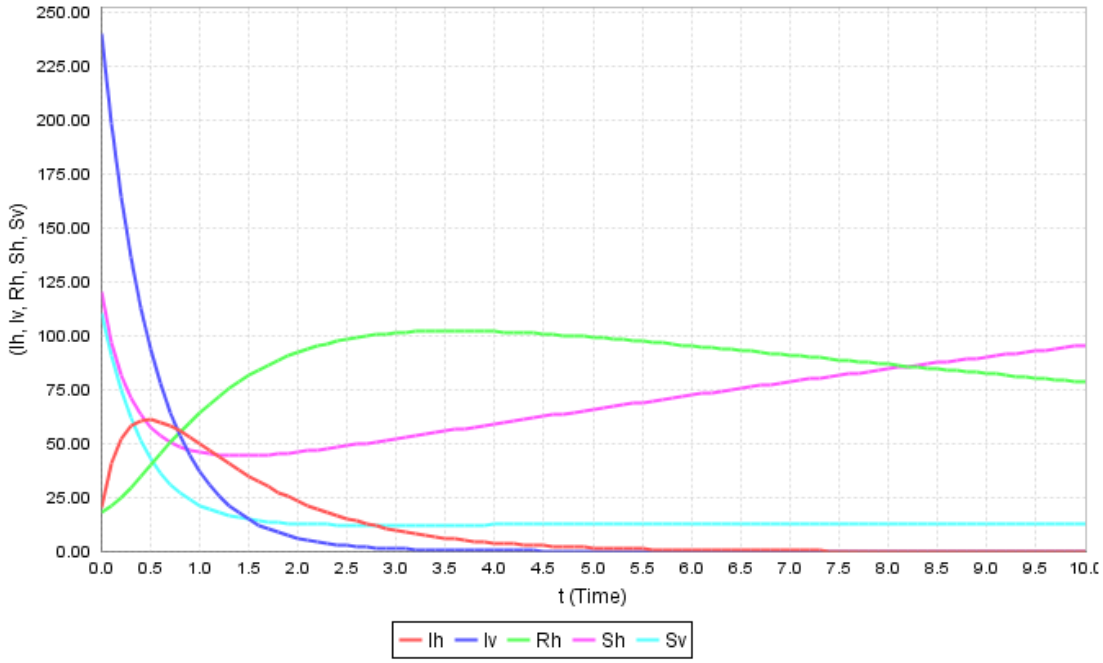

Figure 2: **Numerical simulation of the model with respect to time for parameter values in Table 3 and $\mathbf{R_0 = 0.255288}$. The initial population size are $\mathbf{S_{h0} = 120}$, $\mathbf{I_{h0} = 20}$, $\mathbf{R_{h0} = 18}$, $\mathbf{S_{v0} = 110}$ and $\mathbf{I_{v0} = 240}$.**

Table 4 Parameter values

Parameter	Values	Reference
α_h	0.05	[11]
π_v	125	Assumed
δ_h	0.1	Assumed
β_h	0.01	[11]
α_v	0.06	[11]
γ_h	0.9	[11]
π_h	2.5	[11]
β_v	0.005	[11]
ρ_h	0.001	Assumed

By considering the parameter values in table 4 and the initial conditions $S_{h0} = 360$, $I_{h0} = 40$, $R_{h0} = 36$, $S_{v0} = 220$ and $I_{v0} = 480$ the mathematical simulation of model (1-5) is conducted and the results are given in figure 3. In figure 3, the fractions of the populations S_h, I_h, R_h, S_v and I_v are plotted versus time and also in figure 4, the fractions of the populations S_h, I_h and R_h are plotted versus time. The susceptible mosquito populations will initially decreases with time and then increases as the immigration rate increases which in turn have an effect on the susceptible human population to be bitten by the mosquito and infected by malaria as well the malaria diseases persists in the population. Therefore, the reproduction number is greater than one and the disease free equilibrium point $E_0 = (S_h^0, I_h^0, R_h^0, S_v^0, I_v^0) = (\pi_h/\alpha_h, 0, 0, \pi_v/\alpha_v, 0)$ is unstable. The susceptible mosquito population increases over time as shown in the figure 3 and showing that a malaria outbreak will occur.

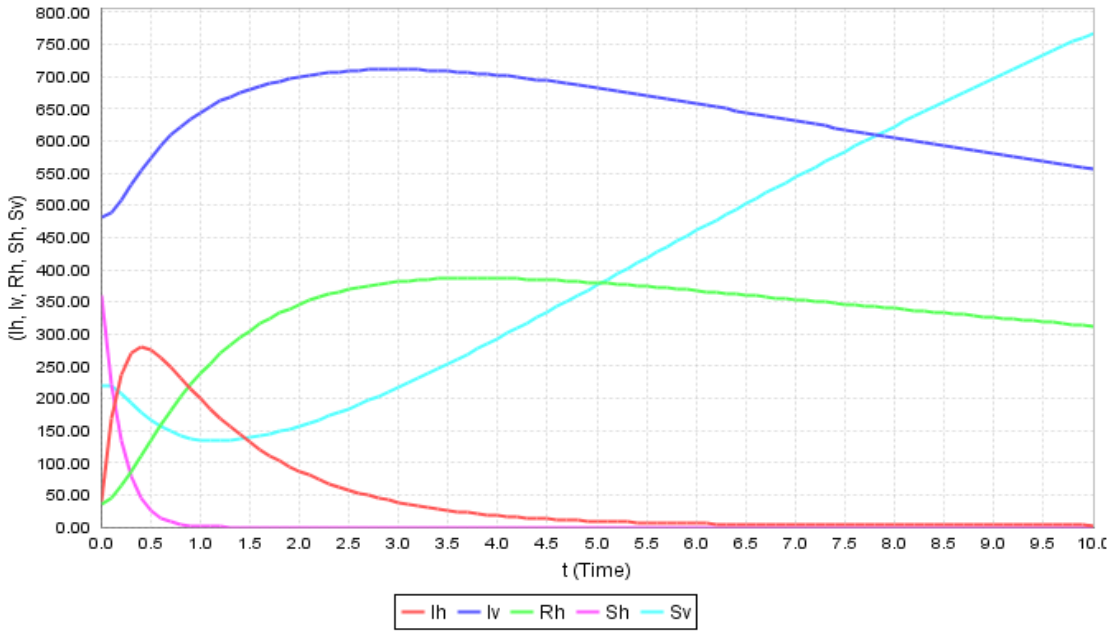

Figure 3: Numerical simulation of the model with respect to time for parameter values in Table 4 and $\mathbf{R_0 = 10.09971}$. The initial population size are $\mathbf{S_{h0} = 360}$, $\mathbf{I_{h0} = 40}$, $\mathbf{R_{h0} = 36}$, $\mathbf{S_{v0} = 220}$ and $\mathbf{I_{v0} = 480}$.

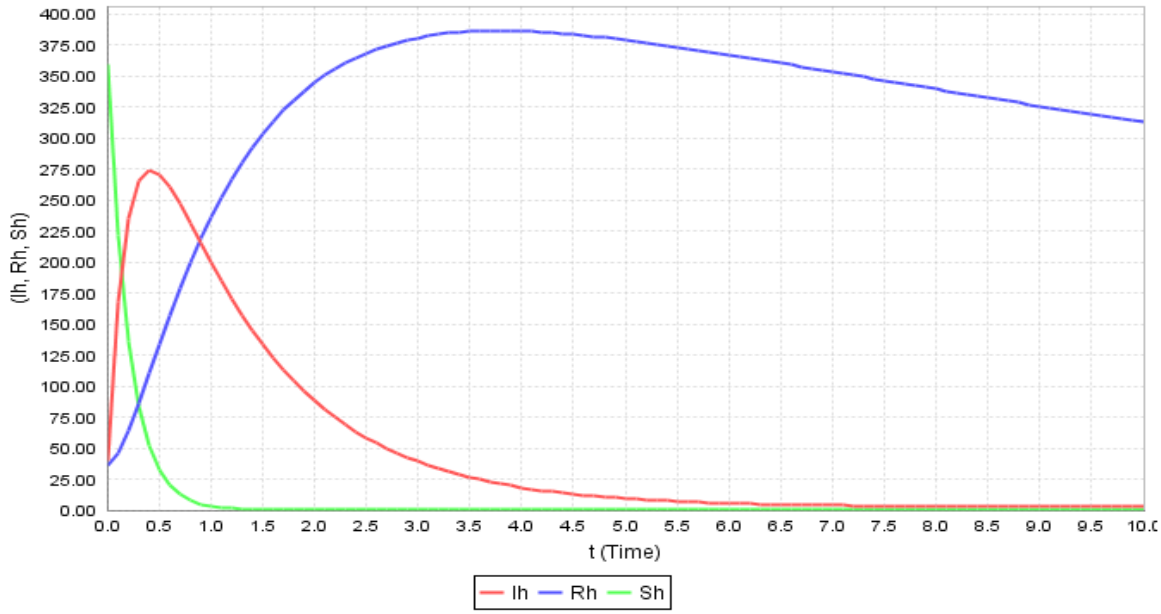

Figure 4: The fractions of the populations $\mathbf{S_h}$, $\mathbf{I_h}$ and $\mathbf{R_h}$ versus time with $\mathbf{R_0 = 10.09971}$ and the initial population size are $\mathbf{S_{h0} = 360}$, $\mathbf{I_{h0} = 40}$, $\mathbf{R_{h0} = 36}$, and $\mathbf{I_{v0} = 480}$.

V. Results and discussion

In this paper, the dynamics of an SIR model (1-5) is studied and applied to malaria transmission between human and mosquito populations. The basic reproduction number is derived and the existence and stability of Disease-Free Equilibrium DFE of model (1-5) are discussed. The analysis shows that if the reproduction number is less than one then the DFE is locally and globally asymptotically stable, this implies that only susceptible is present and the other populations reduces to zero, and the disease dies out as it is shown in figure 2. And if the reproduction number is greater than one then DFE is unstable, for the model (1-5). This has been verified by numerical simulation in figure 3.

Clearly, from the numerical simulations, the DFE is locally asymptotically stable whenever the reproduction number is less than one for the model (1-5). It is also noticed that in order to reduce the basic reproduction number below one, it is very necessary to give a focus on reduction of the infected immigration rate of the human population. Hence for the immigration of an infected human population from one place to the

other place it is recommended that to be tested for the malaria before immigration to decrease the malaria infection.

VI. Conclusion

In this paper, a model for malaria is formulated taking into account both the human and mosquito populations. An SIR model with infected immigrants to infected human is formulated for humans and an SI model is formulated for mosquito with constant recruitment for both human and mosquito population. Mosquito dynamics is studied along with human dynamics because mosquito population determines to a large extent whether a malaria outbreak will occur or not.

Further, the positivity and boundedness of the solution of the model developed is verified to discover that the model equation is mathematically and epidemiologically well posed. The disease free equilibrium theory is applied to the model developed to study the stability analysis.

In particular, the stability properties were investigated by paying more attention to the basic reproduction number and Lyapunov function. The existing work is expanded by putting the missing detail i.e., incorporating the infected immigrants to infected human compartments to SIR model and making reasonable contributions in malaria control. From the numerical results, it is found that prevention of infected immigrants have a strong impact on the malaria disease control.

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