A STUDY OF MATHEMATICAL MODELLING OF MALARIA

A DISSERTATION

SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE

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IN

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Submitted by

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DECLARATION

I, **Jackline G Julias** Roll number 2K21/MSCMAT/55 student of Master of Science in Mathematics, hereby affirm that the project's dissertation is “The Study of mathematical modeling of Malaria” which I have submitted to the Department of Applied Mathematics at Delhi Technological University in part fulfilment of the criterion for the award of master of science, is unique and wasn't copied without proper attribution from any other sources. No degree, diploma, associateship, fellowship, or other title or recognition of a similar nature has previously been awarded on the basis of this work.

Place: Delhi Name: JACKLINE G JULIAS

Date: 23/05/2023

CERTIFICATION

I hereby certify that the project dissertation titled "**A Study of Mathematical Modeling of Malaria**" which is submitted by Jackline G Julias, Roll no 2K21/MSCMAT/55, Department of Applied Mathematics, Delhi Technological University, Delhi in partial fulfillment of the requirement for the award of the degree of Master Of Science, is a record of the project work carried out by student under my supervision. To the best of Knowledge, this work has not been submitted in partial fulfillment of any Degree or Diploma to this university or elsewhere.

Place: Delhi Name: **Dr. VIVEK KUMAR AGGARWAL**

Date: 23/05/2023

ABSTRACT

Malaria is a disease that can be transferred from person to person through female Anopheles mosquito bites and is brought on by the Plasmodium parasite. A mathematical model is utilized to mathematical equations to describe the dynamics of malaria and the compartments in the human population. that capture the links between the pertinent compartmental properties. The goal of the study is to understand the key factors that influence the transmission and spread of the endemic malaria disease and to try to identify effective strategies and tactics for its prevention and control via the use of mathematical modelling. The malaria model is a system of ordinary differential equations (ODEs) developed using basic mathematical modelling methods. The study also looks at the stability of the equilibrium points for the model. The findings demonstrate that the sickness vanishes and the disease-free equilibrium point is stable if the reproduction number, R0, is smaller than 1. The disease-free equilibrium becomes unstable if R0 rises above 1. There, the endemic situation has a special balance, re-invasion is always possible, and human infection continues to spread. Matlab software was used to give the numerical results. These simulations aid in illuminating population behavior through time as well as the consistency of endemic and disease-free equilibrium points.

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With self-effacement, I give thanks to Almighty God, who through his blessings, love, and protection enabled and guide me in this study in good health and courage.

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LIST OF ABBREVIATIONS

IRS- Indoore Residual spray

LLINs- Long lasting insecticide treated nets

ITNs- Insecticide treated nets

SIR -Suceptible, Infected, Recoverd

DFE- Disease free equilibrium

EE- Endemic equilibrium

ODE- Odinary differential Equation

HIV- Human immunodeficiency virus

WHO -World health Organization

CHAPTER 1

INTRODUCTION TO THE STUDY

1.0. Introduction

The infectious disease is carried by female Anopheles mosquitoes, which bite humans known as malaria, which is brought on by the Plasmodium parasite and spread from person to person. Despite being preventable and treatable, malaria infection causes illness and life-threatening symptoms, and it continues to be a global burden with a high estimated death rate in Sub-Saharan Africa, primarily among young infants and expectant mothers.

Backdrop of malaria is a global hazard, the mosquito feeding cycle and its involvement in malaria transmission, the incidence of malaria in Tanzania, a statement of the issue that needs to be resolved, and the study's goal are all covered in this chapter. Finally, a project road map that follows the report section's format is provided.

1.1. Study's Background

Malaria is common and deadliest infections in the Sub-Saharan region, malaria accounts for the majority of the 3000 daily fatalities that occur there. Young children, especially those Specific population risk categories include children under five who have not yet acquired protective immunity, pregnant women (including HIV-infected pregnant women who are non-immune, semi-immune, and immune to HIV) and pregnant women. Fever is the most frequent sign of malaria which is frequently accompanied by other symptoms like exhaustion, shakiness, weakness, and excessive sleeping [1]. Cerebral malaria, which affects roughly 575000 children annually in Africa and has a 10–40% fatality rate, can be brought on by malaria. [3], [4],[5]. If left

untreated, cerebral malaria can harm the brain and cause brain damage. Learning difficulties are experienced by 5-20% of survivors.

When malaria elimination is anticipated in the near future, mathematical models become even more crucial [11]. They have proved helpful in attempts to control malaria. Sir Ronald Ross was the person first to introduce a mathematical model for understanding the transmission and treatment of malaria [9]. He developed a model with a purely deterministic formula in 1916 [8]. The models have been employed in studies to fight malaria, and they have proven to be highly successful [10]. Understanding the dynamics of the disease is greatly aided by malaria transmission models [25]. They have been used for a very long time to evaluate potential intervention strategies [26, 27]. Numerous research have used deterministic models to study dynamics, whereas others have used stochastic models [28, 29]. [30, 31]. Some studies either ignore the effects of climate or take them into account by using the power of infection. Ronald Ross' work was improved upon by MacDonald in 1957. He developed the Ross-MacDonald model, which is known for having a latency time in both human and mosquito populations [14].

1.1.2 Malaria transmission.

Female Anopheles mosquitoes carry the Plasmodium-genus protozoan parasite that causes malaria [4]. Despite the fact that there are over a hundred distinct species, only four—P falciparum, P vivax, P malariae, and P ovale—are primarily at blame for all human ailments. Infections, the most severe illness, and the bulk of mortality in Africa are all brought on by P falciparum [19]. The parasites reproduce in the liver of the human body, infecting red blood cells [20]. Figure 1 depicts the life cycle of the malaria

parasite within the human body.

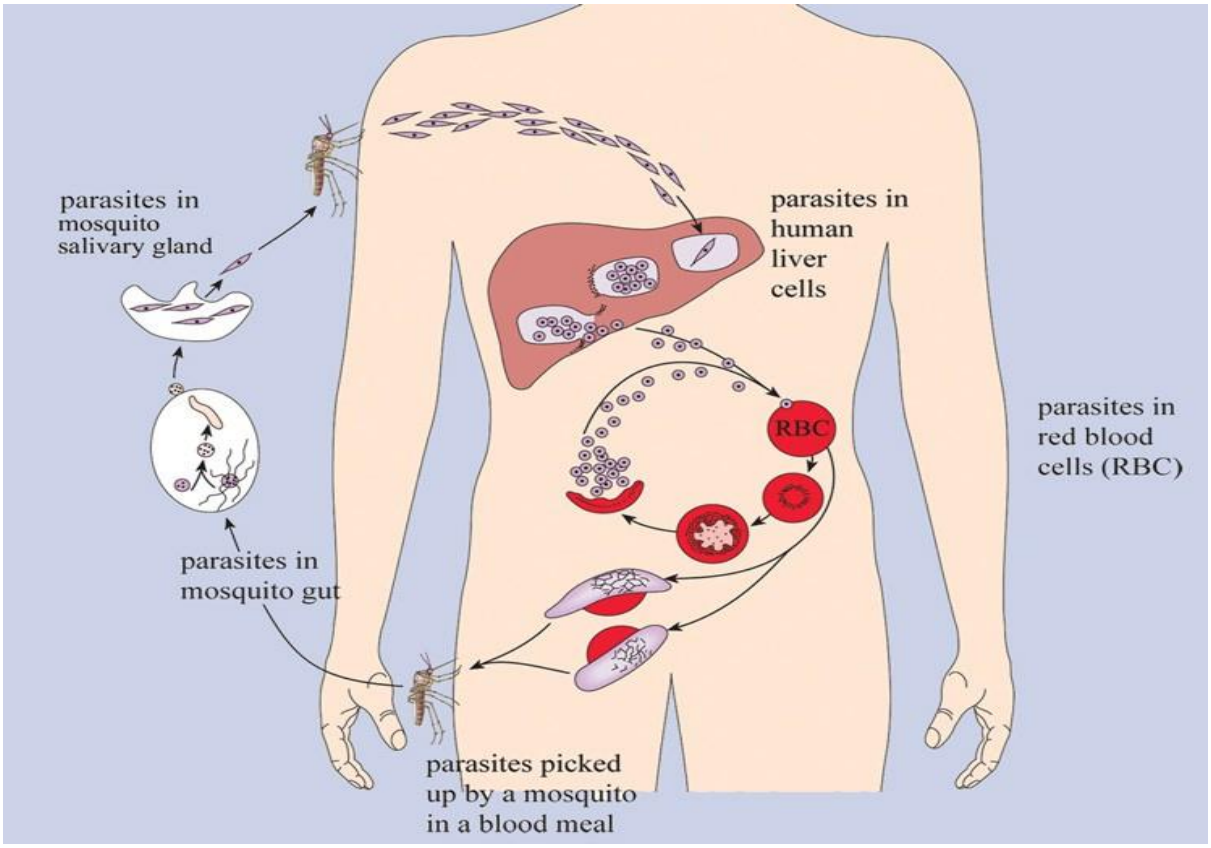

Fig 1. The life cycle of the malaria parasite can be seen in Figure 1 within the human body taken from Infectious Disease Book 5: Evolving Infections published by the Open University in 2003.

The natural ecology of malaria involves serial infections of human, additionally, malaria parasites use female anopheles mosquitoes as hosts. Prior to infecting the red blood cells in humans, parasites reproduce and proliferate in the cells of the liver. In blood, parasites develop inside red blood cells, consume them, and then release merozoite offspring that continue the cycle by feeding on additional red blood cells. Gametocytes, a stage of the parasite known as malaria that lives in the blood, cause malaria symptoms. Female Anopheles mosquitoes pick up the when a mosquito consumes blood, gametocytes start a fresh cycle of expansion and reproduction.

The parasites are discovered in mosquito salivary glands between 10 and 18 days later as sporozoites. Through the saliva of mosquitoes, the parasites inject themselves into people's bodies can spread illness. By transmitting disease from one person to another, the mosquito serves as a host vector for illness.

1.1.3 Effective strategies for malaria control.

To control the spread of malaria, numerous malaria vector control interventions have been put out and used. Most strategies concentrate on controlling vectors, adult mosquitoes. All stages of mosquito development are killed by them, deter adult mosquitoes from congregating in a particular area, and aggravate mosquitoes that come into touch with the interventions. In order to control and possibly eradicate malaria, In endemic areas, the World Health Organisation (WHO) has endorsed the use of long-

lasting insecticide-treated nets (LLINs), indoor residual spraying (IRS), and anti-malaria medications [21]. Another critical stage in managing the mosquito population is looking at environmental factors like temperature and rainfall, which are essential in anticipating disease epidemics. Accurate seasonal climatic forecasts of meteorological factors enable the use of malaria models that incorporate early warning systems in endemic areas [24].

1.2 Malaria in Tanzania

East Africa's African Great Lakes region includes Tanzania, the United Republic of. It borders Uganda to the north; Kenya to the northeast; the Comoro Islands and the Indian Ocean to the east; Mozambique and Malawi to the south; Zambia to the southwest; Rwanda, Burundi, and the Democratic Republic of the Congo to the southwest; and the Comoro Islands and the Indian Ocean to the east. Africa's highest mountain, Mount Kilimanjaro, is located in northeastern Tanzania. Tanzania is the most populated nation wholly south of the equator, with a population of 63.59 million, according to the United Nations.

The number of cases and fatalities from malaria reported in 2021 was greater than in 2020, at over 4.5 million cases and 1920 deaths, respectively.

1.3 Statement of the problem.

Although it is avoidable and treated, malaria continues to be a health issue for the developing globe, with Tanzania bearing a disproportionately heavy burden of the disease. Anopheles gambiae has become malaria transmission over time. The species due to its fast capacity to adapt is resistant to a variety of methods used to reduce mosquito populations as well as to changing environmental conditions. The result is it

still contributes significantly to the transmission of malaria. Among the prevention strategies that Tanzania in particular, where malaria is widespread, has supported the use of insecticide-treated nets (ITNs, LLINs). Despite of Intervention malaria cases has still been recorded and the cases have now started to increase.

1.4. Objectives of the study and Scope of the study.

Studying mathematical modelling of malaria is the key goal. The SIR model is applied to the human-mosquito the spread of malaria. This study's primary objective is to undertake stability studies for endemic and disease-free environments, as well as to study key factors influencing malaria transmission and attempt to create efficient malaria control strategies. We suppose that neither mosquitoes nor humans recover from malaria, nor do the recovered human beings re-enter the vulnerable class. We look at how stable the DFE and EE equilibria are. We describe the SIR model and determine the fundamental reproduction number in chapter 2. Discussion is had regarding the stability analysis of endemic and disease-free equilibria in a model. In the section titled "SIR model is applied to malaria transmission," model analysis that includes stability analyses of endemic with the discussion of disease-free equilibrium. I demonstrate the dynamical behaviour of our results in section 3 of chapter 3 using numerical simulation.

CHAPTER 2

2.1 Model Formulation

The mathematical model of malaria used in this study was SIR in the human population and SI in the vector population. With a total population size of Nh(t) and Nv(t), respectively, the model is constructed to represent the spread of malaria in the human and mosquito populations. Sh(t) and Sv(t) are the classes of human and vector population at risk of getting the disease. Human populations are further divided into epidemiological classifications as susceptible Sh(t), infected Ih(t), and recovered Rh(t) human populations and vector population is devided in susceptible Sv(t) and Infected Iv(t). The model's vector component doesn't have an immune class because they never recover from infections, and their short lifespan means that their infectious time always ends in death. Therefore, the model can be applied to chronic diseases with vital dynamics that affect a population over a long period of time. The model's premises include the total population sizes of both people and mosquitoes are thought to be constant. The recovered individuals do not re-enter the vulnerable class and receive a lifetime immunity. The compartmentalized human and vector populations are both non-negative. The infectious female mosquito that transmits malaria bites the human host, making all infants susceptible to infection. The infection does not cause the vectors to perish.

2.2 Basic model description reproductive

Susceptible Humans Pollution (S), Infectious Humans Population (I), and Removed Human Population (or Died) Humans (R) are the three compartments that make up the population in our model.

The interaction of the schematic diagram in Fig. 2.1 below displays compartments:

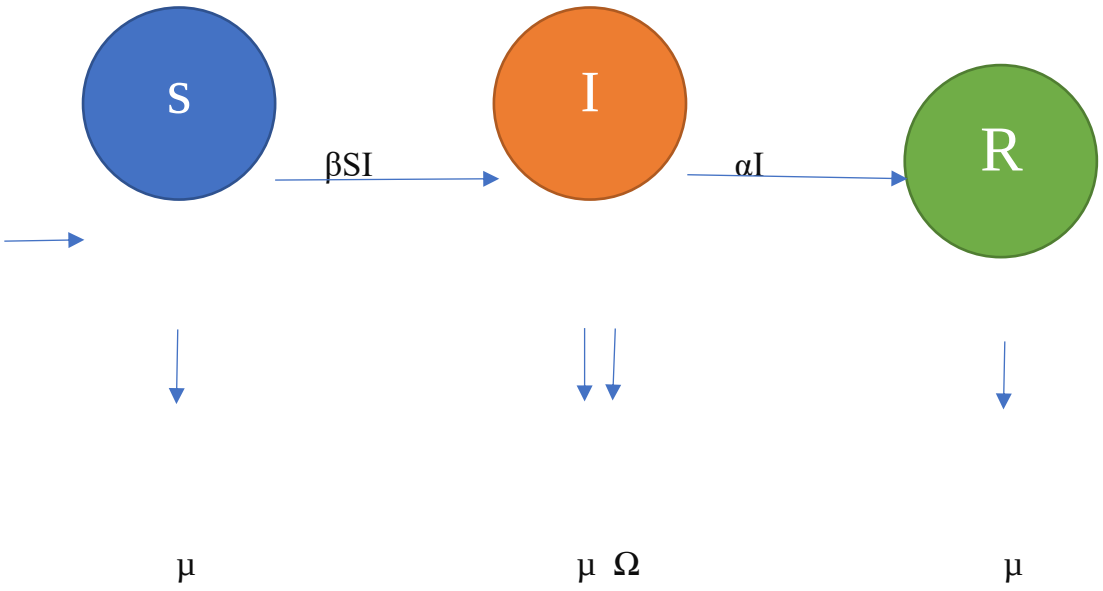

Fig. 1. Flowchart of the Malaria Transmission

The model equations are given by

$$\frac{dS}{dt} = \wedge - \beta SI - S\mu \tag{2.1}$$

$$\frac{dI}{dt} = \beta SI - \Omega I - \alpha I - I\mu$$

$$\frac{dR}{dt} = \alpha I - \mu R$$

It is possible to calculate the total population sizes N by

$$S + I + R = N$$

with initial circumstances

$S(0) > 0,\ I(0) \geq 0$ and $R(0) \geq 0$

Table 1. Descriptions of the SIR Model's parameters

Parameter Name	
∧	Recruitment of susceptible individuals
B	Infectious Rate
A	Rate of recover
μ	Rate of natural death
Ω	Rate of Induced Death

2.3. Analysis of the Model.

2.3.1 Disease free equilibrium

By taking into account the ODES (1) system and setting the derivative to zero, I research the area geometrical characteristics of the equilibrium free from illness E0 in this section.

Clearly the first equilibrium point $(S\ I\ R) = (\wedge/\mu, 0,0)$

I acquire by using the Jacobian matrix.

$$J(S,I) = \begin{bmatrix} -\beta i - \mu & -\beta \\ \beta i & \beta S - (\alpha + \mu) \end{bmatrix} \qquad (2.3.1)$$

The Jacobian matrix (3.1) can be used to assess the equilibrium's local stability. According to this, the Jacobian matrix for the equilibrium without illness is represented by

$$J(1,0) = \begin{bmatrix} -\mu & -\beta \\ 0 & \beta - (\alpha + \mu) \end{bmatrix} \qquad (2.3.2)$$

To get Eugen Values, the determinant of (3.2) we obtain

$$|J((1,0) - \lambda I)| = \begin{vmatrix} -\mu - \lambda & -\beta \\ 0 & \beta - (\alpha + \mu) - \lambda \end{vmatrix} \qquad (2.3.3)$$

The characteristics equation becomes:

$$(-\mu - \lambda)(\beta - (\alpha + \mu) - \lambda) = 0$$

$$\lambda 1 = -\mu,\ \lambda 2 = \beta - (\alpha + \mu) \qquad (2.3.4)$$

And clearly, all eigenvalues may be seen to have only negative real components if.

$$(\alpha + \mu) > \beta$$

And hence it is locally asymptotic stable

2.3.2 Endemic Equilibrium's presence

In this part, I analyse a scenario in which malaria is present among a population.

I indicate E*=(S,1,R) as an endemic equilibrium point obtained as (2.3.5)

$$S = \frac{\mu + \alpha}{\beta}$$

$$I = \frac{\beta\mu - \mu(\mu + \alpha)}{\beta(\mu + \alpha)}$$

$$R = \frac{\alpha}{\mu}\left[\frac{\beta\mu - \mu(\mu + \alpha)}{\beta(\mu + \alpha)}\right]$$

The Jacobian Matrix becomes

$$J(E*) = \begin{bmatrix} -\beta I - \mu & -\beta S \\ \beta I & \beta S - (\alpha + \mu) \end{bmatrix}$$

Substituting the values of (3.4) we get

$$J(E*) = \begin{bmatrix} \frac{\beta\mu}{\mu + \alpha} & -(\mu + \alpha) \\ \frac{\beta\mu}{\mu + \alpha} - \mu & 0 \end{bmatrix}$$

We obtain

$$|J(E* - \lambda I)| = \begin{vmatrix} \frac{\beta\mu}{\mu + \alpha} - \lambda & -(\mu + \alpha) \\ \frac{\beta\mu}{\mu + \alpha} - \mu & 0 - \lambda \end{vmatrix} = 0$$

Note: for the system to be stable asymptotic stable (R>1)

$$trace(|J(E* - \lambda I)|) < 0 \; and \; \det(|J(E* - \lambda I)| > 0) \qquad (2.3.6)$$

and hence all the real part are negative

2.4. Application of the Model to Study Malaria

There are three groups that make up the human population: Susceptible S_h, Infected I_h, and Recovered R_h. The model is created for both the current human population and the

current number of mosquitoes t, additionally, the mosquito population is split into two groups Susceptible $S_{v,}$ and Infected $I_{v.}$

The interaction between Human and mosquito is shown in the figure below

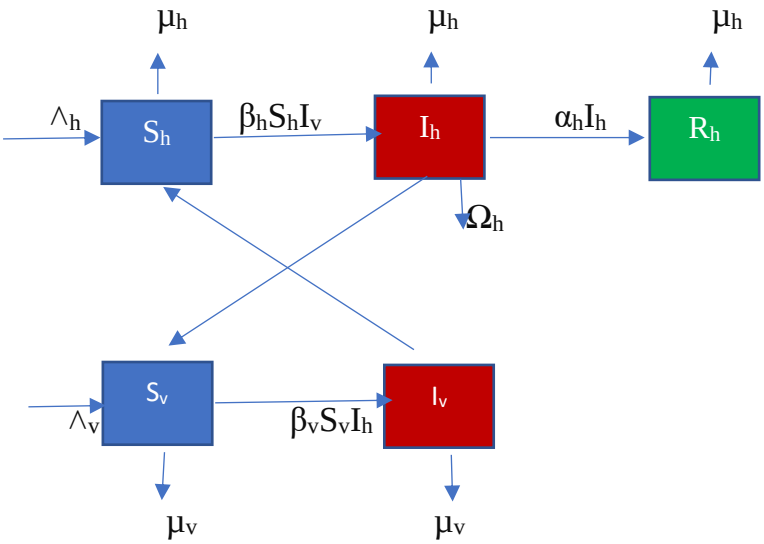

Table 2. Parameters Descriptions for the SIR Model for malaria Transmission

Parameter	Parameter description
Sh	No of susceptible human at time t
Ih	No of infected human at time t
Rh	No of recovered human at time t
Sv	No of susceptible human at time t
Iv	No of infected human at time t
Nh	Total human population at time t
Nv	Total mosquito population at time t
Λ_h	Human Recruitment rate at time t
μ_h	Per capital human population natural death rate
α_h	Per capita recovery rate of human
β_h	Human contact rate mosquito dimention

Ω_h	Per capital disease induced rate for human at time t
$\wedge_v$	Per capital birth rate of mosquito
μ_v	Per capital natura death rate of mosquito
β_v	Mosquito contact rate human dimension

The recruitment rates of humans and mosquitoes are indicated in this model by the letters $\wedge_h$ and $\wedge_v$ are respectively. The phrases used in our model as $\mu_h S_{h,,}$ $\mu_h I_h$ and $\mu_h R_h$ refer to the amount of recovered, infected, and susceptible persons removed during a specified period of time. The phrases $\mu_v S_v$ and $\mu_v I_v$ represent the proportion of susceptible and diseased mosquito populations eliminated in a certain amount of time. The term $\Omega_h I_h$ represents the proportion of people who have been removed from society due to disease during a specified period of time. while $\alpha_h I_h$ represents the total number of recovered humans over a given period of time. The word $\beta_h S_h I_v$ refers to the rate at which the mosquito vector Iv infects the human hosts Sh and $\beta_v S_v I_h$ the rate at which the human hosts Ih infect the susceptible mosquitoes Sv at time t. Thus, As a result, both of these concepts play crucial roles in the model that describes the interaction between the two populations.

From our model, The Model equations are given as:

$$\frac{dSh}{dt} = \wedge h - \beta hShIv - \mu hSh$$

$$\frac{dIh}{dt} = \beta hShIv - \Omega hIh - \mu hIh - \alpha hIh \qquad (2.4.1)$$

$$\frac{dRh}{dt} = \alpha hIh - \mu hRh$$

$$\frac{dSv}{dt} = \wedge v - \beta vSvIh - \mu vSv$$

$$\frac{dIh}{dt} = \beta vSvIh - \mu vIv$$

It is possible to calculate population sizes overall of N_h and N_v which can be established

$$S_h + I_h + R_h = N_h \quad (2.4.2)$$

$$S_v + I_v = N_v \quad (2.4.3)$$

$$N_h(t) = S_h(t) + I_h(t) + R_h(t)$$

Afterward, the derivative of $N_h(t)$ according to t is given by

$$\frac{dNh}{dt} = \wedge h - \mu hNh - \Omega hIh$$

$$\lim_{t\to\infty} Nh(t) \leq \frac{\wedge h}{\mu h}$$

Thus, the system is positively invariant with

$$Sh + Ih + Rh \leq \frac{\wedge h}{\mu h}, Sh > 0, Ih \geq 0, Rh \geq 0$$

And let;

$$N_V(t) = S_V(t) + I_V(t)$$

following the derivative of $N_v(t)$ according to t is given by

$$\frac{dNv}{dt} = \wedge h - \mu v Nv$$

$$\lim_{t\to\infty} Nv(t) \leq \frac{\wedge v}{\mu v}$$

Again, Positive invariance of the system is with

$$Sv + Iv \leq \frac{\wedge v}{\mu v}, Sh > 0, Ih \geq 0$$

Hence the system's disease-free equilibrium is clearly seen at $E_{0hv} = (\frac{\wedge h}{\mu h}, 0,0, \frac{\wedge v}{\mu v}, 0)$.

The essential reproductive number for people and mosquitoes will be produced using the next generation matrix R_0.

2.5 Basic Reproductive Number

The amount of secondary illnesses that a single infected person may spread within a vulnerable community is calculated using the next-generation matrix, and denoted by the reproductive number R0.

In the system of Ordinary Differential equation, we have two disease classes which are I_h and I_v.

In these two equations, we Let F denote the disease class and V denote transfer class.

Where F and V are given by

$$F = \begin{bmatrix} \beta h Sh Iv \\ \beta v Sv Ih \end{bmatrix}$$

$$V = \begin{bmatrix} (\Omega h + \mu h + \alpha h) Ih \\ \mu v Iv \end{bmatrix}$$

The differentials of F and V with respect to I_h and I_v at the disease-free equilibrium

$E_{0hv} = (\frac{\wedge h}{\mu h}, 0,0, \frac{\wedge v}{\mu v}, 0)$ is given by

$$DF(E_0) = \begin{bmatrix} 0 & \frac{\beta h \wedge h}{\mu h} \\ \frac{\beta v \wedge v}{\mu v} & 0 \end{bmatrix}$$

And

$$DV(E_0) = \begin{bmatrix} \Omega h + \mu h + \alpha h & 0 \\ 0 & \mu v \end{bmatrix}$$

$Ro = \sigma(FV^{-})$

This gives the basic reproductive number as

$$R_0 = \sqrt{\frac{\beta h \wedge h \beta v \wedge v}{\mu h \mu v^2 (\Omega h + \mu h + \alpha h)}}$$

Hence The disease-free equilibrium $E_{0hv} = (\frac{\wedge h}{\mu h}, 0,0, \frac{\wedge v}{\mu v}, 0)$ of the system (2.4.1) of

Ordinary differential equations is asymptotically stable if Ro <1 and unstable if $R_0 \geq 1$.

2.6 **Analysis of the SIR Model**

2.6.1 Existence and stability of Disease-free equilibrium

This section looked at the local characteristics of an equilibrium without sickness.

$E_{0hv} = (\frac{\wedge h}{\mu h}, 0,0, \frac{\wedge v}{\mu v}, 0)$

The disease-free equilibrium, E_0 which is the steady state of the model in there is absence of infection. This is obtained from the system (2.4.1) by setting the right-hand side equal to 0, and assuming that $i_h = 0$ and $i_v = 0$, where i_h and i_v refer to the equilibrium points. The local stability of E_0 is then determined from the signs of the eigenvalues of the Jacobian matrix. At the disease-free equilibrium, E_0, the Jacobian matrix is given by

$$J(E0) = \begin{bmatrix} -\mu h & 0 & -\beta hSh \\ 0 & -(\Omega + \alpha + \mu) & \beta hSh \\ 0 & \beta vSv & -\mu v \end{bmatrix}$$

The characteristic equation becomes

$$J(E0 - \lambda I) = \begin{bmatrix} -\mu h - \lambda & 0 & -\beta hSh \\ 0 & -(\Omega + \alpha + \mu) - \lambda & \beta hSh \\ 0 & \beta vSv & -\mu v - \lambda \end{bmatrix}$$

This gives

$$-(\mu h + \lambda)\big((\alpha + \mu + \Omega - \lambda)(\mu v - \lambda) - \beta vSv\beta hSh\big) = 0$$

The roots of the characteristic equation are the eigenvalues of the Jacobian matrix. It is clear that the characteristic equation has the negative eigenvalue λ_1 which is negative. It is again clear that the equation has some positive roots. As a result, we draw the conclusion that the disease-free equilibrium is unstable

2.6.2 Existence and stability of endemic equilibrium

An endemic equilibrium is a model's steady state with an infected human population and Infected vector population which is given by E = (S I R) which follows the condition $s_{h*} > 0$, $i_{h*} > 0$, $i_{v*} > 0$.

From the system (2.4.1) of the ordinary differential equation we obtain

$$Sh^* = \left(\frac{\wedge h}{\mu h} - \frac{\beta h(\Omega h + \alpha h + \mu h)}{\mu h \beta h}\right) * \left(\frac{\wedge h - \mu h(\Omega \mathrm{h} + \alpha \mathrm{h} + \mu \mathrm{h})}{\beta h \beta v Sv\left(\wedge h - (\Omega h + \alpha h + \mu h)\right)}\right)$$

$$Ih^* = \left(\frac{\wedge h - \mu h(\Omega + \alpha + \mu)}{\beta h \beta v Sv\left(\wedge h - (\Omega + \alpha + \mu)\right)}\right)$$

$$Iv^* = \frac{\Omega h + \alpha h + \mu h}{\beta h(\frac{\wedge h - (\Omega + \alpha + \mu)}{\mu h})}$$

To determine the stability of the equilibrium at the steady state of the model we obtain the Jacobian matrix from the system (2.4.1) of ODS

$$J(E1) = \begin{bmatrix} -\beta h Iv - \mu h & 0 & -\beta h Sh \\ \beta h Iv & -(\Omega + \alpha + \mu) & \beta h Sh \\ 0 & \beta v Sv & -\mu v \end{bmatrix}$$

Thus, the Characteristics equation of the Jacobian matrix can be written as

$$J(E1 - \lambda I) = \begin{bmatrix} -\beta h Iv - \mu h - \lambda & 0 & -\beta h Sh \\ \beta h Iv & -(\Omega + \alpha + \mu) - \lambda & \beta h Sh \\ 0 & \beta v Sv & -\mu v - \lambda \end{bmatrix} = 0$$

This gives

$$-(\beta h Iv + \mu h) - \lambda\left(\left((\Omega + \alpha + \mu + \lambda)(\mu v + \lambda) - \beta h Sh \beta v Sv\right) + \beta h^2 \beta v Sh Sv Iv\right)$$

$$= 0$$

I can infer that the Endemic Equilibrium state is stable because it is obvious that all Eigenvalues have negative values.

CHAPTER 3

3.0 Numerical Solution

In this section, the SIR model's numerical solution for the spread of malaria is investigated, and the table contains a list of all the variables that were employed. Fig, Table for model 2

Parameter	Description	Value	Source
$\wedge_h$	Human Recruitment rate at time t	1.2	[32]
β_h	Human contact rate mosquito dimension	0.00638	[35]
α_h	Per capita recovery rate of human	0.0035	[32]
μ_h	Per capital human population natural death rate	0.01146	Assumption
Ω_h	Per capital disease induced rate for human at time t	0.0068	Assumption
$\wedge_v$	Per capital birth rate of mosquito at time t	0.7	Assumption
β_v	Mosquito contact rate human dimension	0.00696	[35]
μ_v	Per capital natura death rate of mosquito	0.05	[34]

Nature of the models of the system of ODE (2.4.1) are investigated by conducting analysis of the basic reproductive number

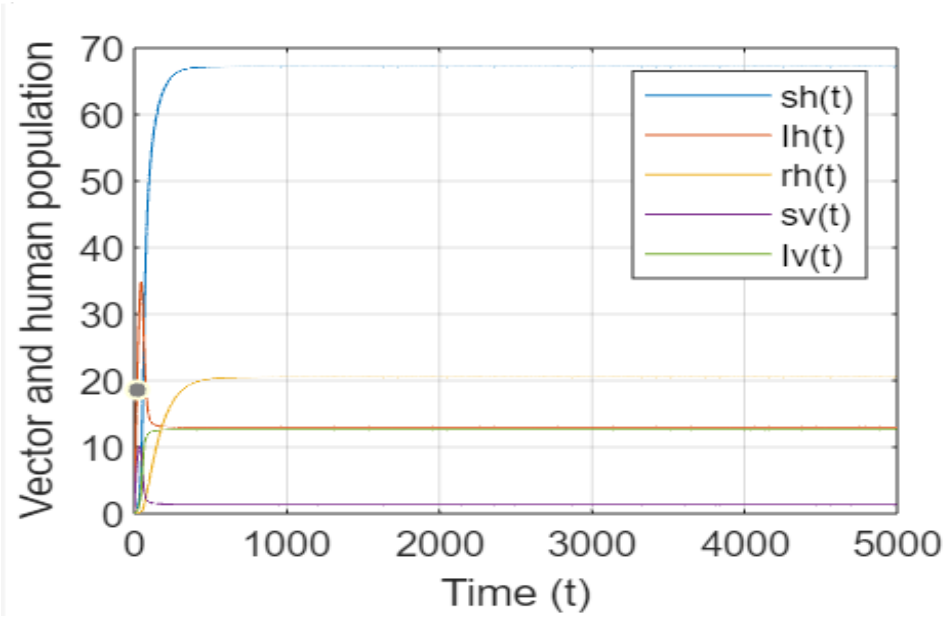

Fig4. Numerical solution of the SIR model with Time response and initial condition S0(0.89), I0(0.07), R0(0.02)

Against time and R0= 0.57

With parameters: ∧ = 1.2, Bh=0.0638, αh=0.0035, μh=0.01146, Ωh=0.0068

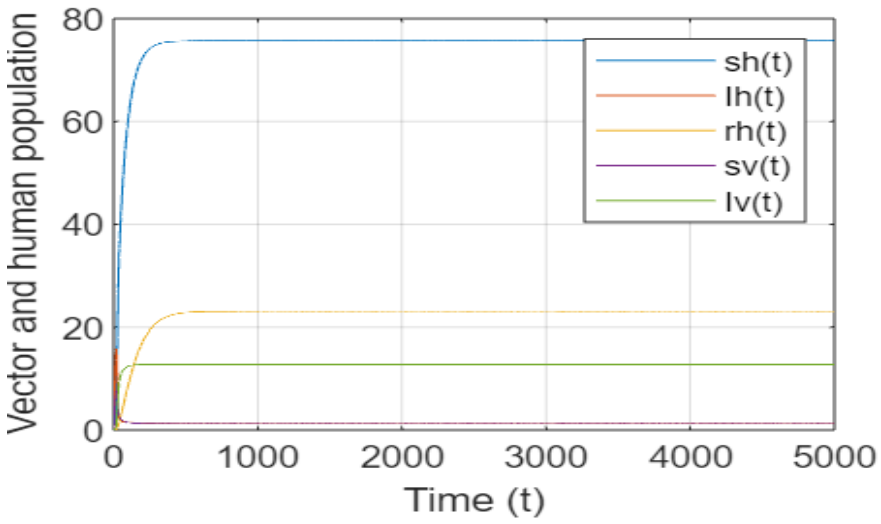

Fig 5. Numerical solution of the SIR model with Time response and initial condition $S_{0h}(0.89)$, $I_{0h}(0.07)$, $R_{0h}(0.02)$ against time with parameters: $\wedge = 1.2$, $\beta_h=0.638$, $\alpha_h=0.0035$, $\mu_h=0.01146$, $\Omega_h=0.0068$ and $R_0=2.4177$ susceptible human increases and infected human decreases and infected vector increased with decreased time and remains constant over time hence endemic is unstable

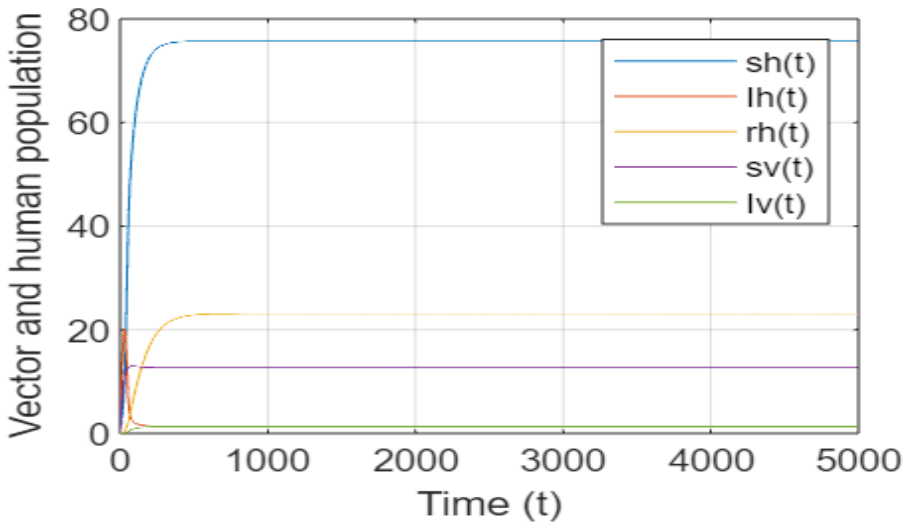

Fig 7. Numerical solution of the SIR model with Time response and initial condition S0(0.89), I0(0.07), R0(0.02) against time with parameters: ∧ = 1.2, β_h =0.00638, β_v =0.0000696 α_h=0.0035, μ_h=0.01146, Ω_h=0.0068 and R_0=0.18699 susceptible human and vector increases and infected human decreases and infected vector decreased with decreased time and approaches zero. Hence endemic equilibrium point is stable

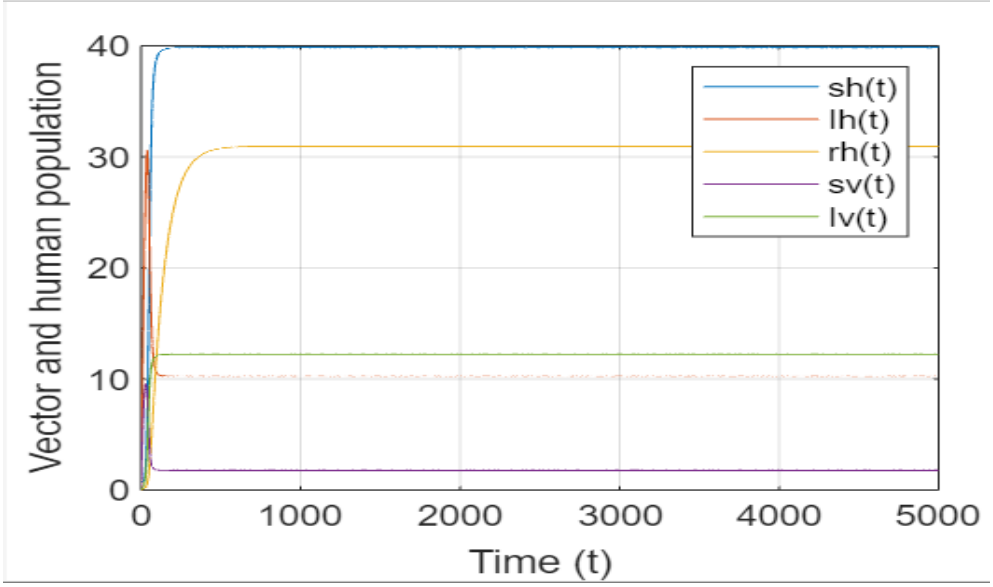

Fig 8. Numerical solution of the SIR model with Time response and initial condition S0(0.89), I0(0.07), R0(0.02) against time with parameters: ∧ = 1.2, β_h = β_v 0.08638, α_h=0.0035, μ_h=0.01146, Ω_h=0.0068 and R_0>1 suspectable human are increasing in less time and infected human and vector increased with time and maintain constant. Hence the system is not stable

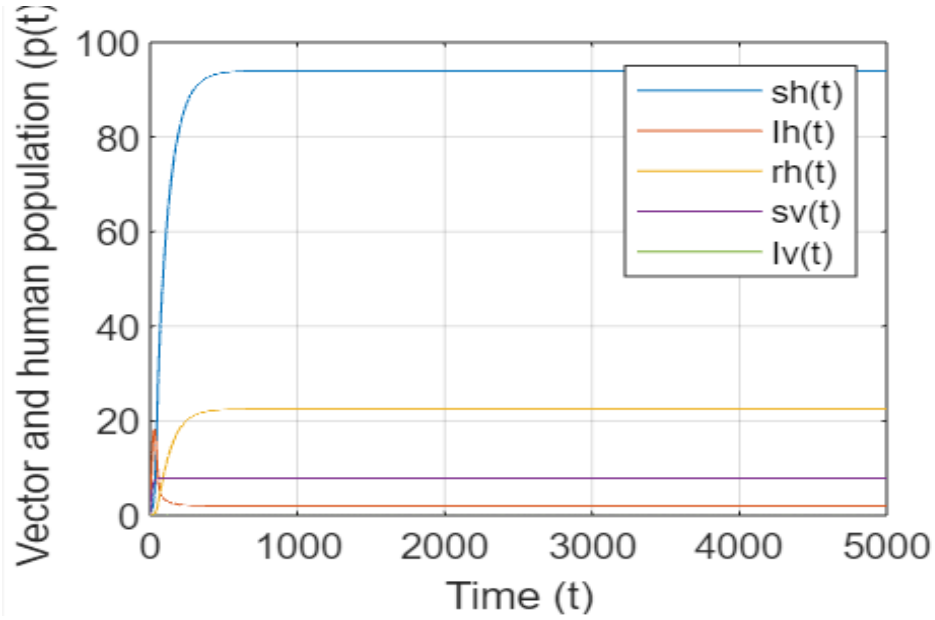

Fig 10. Numerical solution of the SIR model with Time response and initial condition S0(0.89), I0(0.07), R0(0.02) against time with parameters: ∧ = 1.2, β_h =0.0059, β_v =0.000069 and R0=0.1669 suspectable human are increasing in less time and infected human and vector increased with time and maintain constant. Hence the system is stable

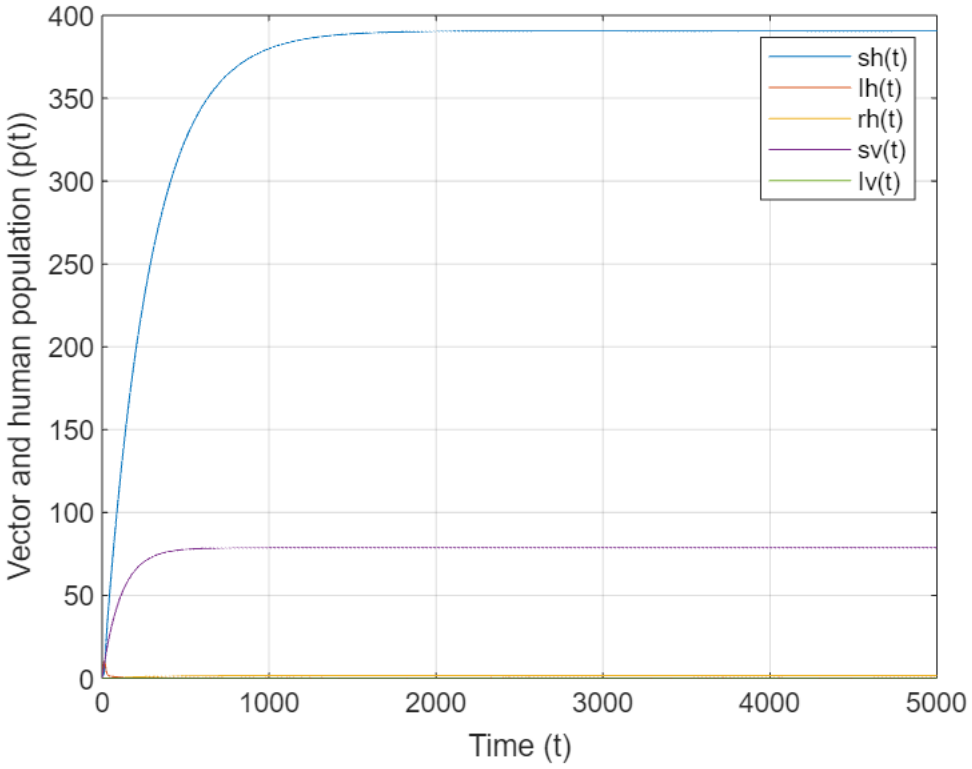

Fig 10. Numerical solution of the SIR model with Time response and initial condition S0(0.89), I0(0.07), R0(0.02) against time with parameters: ∧ = 1.2, β_h =0.0059, β_v =0.0000696, α_h=0.15 and R0=0.0815 suspectable human are increasing in less time and infected population is approximately zero. Hence the system equilibrium is stable

3.1 Discussions and Conclusion

In this section, the dynamic of SIR model in application to Malaria diseases transmission is studied between human and vector population. The model's fundamental reproduction number, stability, and equilibrium are discussed between human and vector population.

The analysis demonstrates that the disease-free equilibrium is stable and the disease dies out over time if the basic reproductive number is less than one, and unstable if the basic reproductive number is more than one.

This has been shown by the numerical solution on figures 1,2,3… additionally, if the number is less than one, the endemic equilibrium is stable, and if the number is greater than one, the endemic equilibrium is unstable. This has been shown in figures.

From the numerical solution we have seen that β is the sensitive parameter such that the interaction between an infection-prone individual and a vector with parameter β_h and the interaction between susceptible vector and infected human with parameter β_v very sensitive.

The numerical solution shows that in equilibrium point, both Infected human and vector population have existed with reproductive number g less than 1 and in Endemic equilibrium point the infected human and vector are approaching zero with reproductive number that and is smaller than one, which is consistent with the stability of the endemic equilibrium point.

Additionally, it has been noted that as the interaction between susceptible vectors and infected humans decline and declines with the interaction between Infected vectors and susceptible humans and the increase of recovery. the disease's reproductive number decreases, and eventually it will go extinct

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