ADAMA SCIENCE AND TECHNOLOGY UNIVERSITY
SCHOOL OF APPLIED NATURAL SCIENCES

APPLIED MATHEMATICS PROGRAM

MATHEMATICAL MODELING OF THE DYNAMICS OF MALARIA AND ITS TRANSMISSION WITH TREATMENT

MSc. Thesis

By:

Lemma Ebissa Atoma

In the Partial Fulfillment of the Requirements of Degree
of Master of Science by applied Mathematics

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Approval Sheet

We, the undersigned, members of the Board of Examiners of the final open defense by **Lemma Ebissa Atoma** have read and evaluated his thesis in titled **"mathematical modeling of the dynamics of Malaria and its transmission with treatment "** and Examined the candidate. This is therefore to certify that the thesis has been accepted in partial fulfillment of the requirement of the degree of Master of Science in applied Mathematics.

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Declaration

I declare that this thesis is my original work and that all source materials used for this thesis have been properly cited and acknowledged. This thesis has been submitted in partial fulfillment of the requirements for MSc. degree in applied Mathematics at Adama Science and Technology University. I earnestly declare that this thesis is not submitted to any other institution any where for the award of any academic degree, diploma, or certificate.

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Abstract

Malaria is a deadly disease transmitted to human through the bite of infected female mosquitoes. It can also be transmitted through blood transfusion from infected mother (congenitally). In this thesis we developed a mathematical model which describes the dynamics of malaria transmission with treatment based on SIRS-SI frame work,using the system of ordinary differential equations(ODE).In addition we derive a conditions for the existence of equilibrium points of the model and investigate their stability.Our results shows that if the reproduction R_0 *is less than* 1 *the disease free equilibrium point is stable, so that the disease dies out.If* R_0 *is greater than* 1*, then the disease free equilibrium point is unstable.In this the endemic state has unique equilibrium and the disease persists within the human population.A qualitative study based on bifurcation theory reveals that backward bifurcation may occur. The stable disease free equilibrium of the model coexists with the stable endemic equilibrium when the basic reproduction number is less than one.Numerical simulations were carried out using Matlab to support our analytical solutions.And these simulations show that how treatment affect the dynamics of human and mosquito population.*
[1]

[1]Key Words:-Malaria model,SIRS-SI model,stability Analysis,treatment,backward bifurcation

List of Abbreviations

DFE Disease Free Equilibrium

R0 Reproduction number

SIRS Susceptible Infected Recovered Susceptible

SI Susceptible Infected

DDT Dichlorodiphenyltrichloroethane

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CHAPTER ONE

1 Introduction

1.1 Back ground of the study

Malaria is an infectious disease caused by a parasite known as plasmodium. Carrier of plasmodium parasite is the female anopheles mosquito that causes the destruction of red blood cells in humans and animals through bites. Malaria can be transmitted through blood transfusion, sharing needles, or congenital. The infection can lead to serious complications affecting the brain, lungs, kidneys and other organs. Clinical symptoms such as fever, pain, chills and sweats may develop a few days after infected mosquito bites[28]. Malaria is one of the most prevalent and lethal human infections throughout the world. An estimated 40 percent of the world's population lives in malaria endemic areas. Most cases and deaths occur in sub-Saharan Africa. It causes an estimated 300 to 500 million cases and 1.5 to 2.7 million deaths each year worldwide. Africa shares 80 percent of the cases and 90 percent of deaths [29].The environmental conditions in the tropics are the prime factor for malaria being endemic. Malaria control is challenging due to many factors. The complexity of the disease control process, ways of transmission, cost of the control program and resistance of the parasite to anti-malarial drugs, and vectors to insecticides, are some of the challenges. As stated above there is a variation in disease patterns and transmission dynamics from place to place, by season and according to varying environmental circumstances.

The approaches in the planning and implementation of prevention and control activities also vary based on local realities. Since malaria increases morbidity and mortality, it continues to inflict major public health and socioeconomic burdens in developing countries. Many researchers have developed a mathematical model of malaria transmission. SIR with nonlinear infection rate and the use of vaccination against the human population was analyzed in [2].

The effect of treatment as a control variable on malaria transmission system was studied in[3]. Model of malaria transmission was investigated in[1] by considering the existence of human-to-human transmission through blood transfusions and through malaria-infected pregnant women (congenital). In this current work, we discuss the effectiveness of the use of drugs in a malaria transmission model

based on[22].In our model we assumed that the vaccination at the right time can move susceptible human beings directly to recovered class and by considering the assumption that humans belong to recovered class have possibility to be susceptible, i.e., we consider a SIRS-SI model[1]. Moreover, we also consider the application of vaccine and spraying as introduced in [26, 22]. Stability (global and local stability) analysis is then performed to reveal the effects of treatments on population dynamics.

1.2 Statement of the problem

Malaria has for many years been considered as a global issue, and many epidemiologists and other scientists invest their effort in understanding the dynamics of malaria and to control its transmission. From interactions with those scientists, mathematicians have developed a significant and effective tool, namely mathematical models of malaria, giving an insight into the interaction between the host and vector population, the dynamics of malaria, how to control malaria transmission, and eventually how to eradicate it. Mathematical models for transmission dynamics of malaria are useful in providing better insights into the behavior of the disease. The models have played great roles in influencing the decision making processes regarding intervention strategies for preventing and controlling the insurgence of malaria.

The model presents in this work consists of three compartments in humans and two compartments in mosquitoes. In this work we assume that the vaccination at the right time can cause susceptible human beings can move directly to recovered class and by considering the assumption that humans belong to recovered class have possibilities to be susceptible ,i.e, we consider SIRS-SI model. Therefore, this research work address the following basic questions:

1. What are the basic assumptions to develop mathematical model which describes the dynamics of malaria and its transmission with treatment?
2. What are the impact of treatment on the dynamics of malaria and its transmission?
3. What are the biological interpretations of the solution of the mathematical problem?

1.3 Objective of the study

1.3.1 General Objective

The general objective of this research work is to understand the dynamics of malaria and its transmission with treatment.

1.3.2 Specific Objectives

The specific objectives of the study are to:

1. develop a mathematical model which describes the dynamics of malaria and its transmission with treatment.
2. determine the conditions for the local and global stability of the system.
3. Examine the biological implications of the analysis.
4. Interpret the biological implication of the solutions of the mathematical model.

1.4 Significance of the study

The main significances of this thesis work are

1. To develop awareness about the dynamics of malaria and its transmission with treatment.
2. To suggest the possible intervention mechanisms.
3. To initiate other researchers to under take farther extension and precise mathematical analysis.

CHAPTER TWO

2 Literature Review

In different time; many researchers develop different mathematical models of infectious disease especially for malaria. In this section, we review the mathematical models developed by different researches to study the dynamics of malaria and its transmission that are directly related to the objectives of this study.
Mathematical models for transmission dynamics of malaria are useful in providing better insights into the behavior of the disease. Mathematical models have played great roles in influencing the decision making processes regarding intervention strategies for preventing and controlling the insurgence of malaria.
The study on malaria using mathematical modeling began in 1911 with Ronald Ross[25]. He introduced the first deterministic two-dimensional model with one variable representing humans and the other representing mosquitoes where it was shown that the reduction of mosquito population below a certain threshold level was sufficient to eradicate malaria. From the Ross model, several models have been developed by researchers who extended his model by considering different factors such as latent period of infection in mosquitoes and humans, age-related differential susceptibility to malaria in human population, acquired immunity and genetic heterogeneity of host and parasite.The major advantage in Rosss models was his ability to provide a suitable control strategy through the transmission threshold criterion which is based on the reproductive capacity of the parasite and is termed as basic reproductive number (Ro). Although the idea of threshold was first introduced by Ross, it originated from Fishers net reproductive value for a parasite, [7].
Ross did not consider the latency period of the parasite in mosquitoes and their survival during that period in his model. This resulted in the model predicting a rapid progress of the epidemic in human and a higher equilibrium prevalence of infectious mosquitoes.
After about 40 years, George Macdonald, in the 1950s, reasserted the value of mathematical epidemiology based on 20 years of fieldwork. He modified Ross's model by integrating biological information of latency in the mosquito due to malaria parasite development, and concerned the survivor ship of adult female mosquito as the weakest element in the malaria cycle.
However, in this case, it was shown that reducing the number of mosquitoes

is an inefficient control strategy that would have little effect on the epidemiology of malaria in areas of intense transmission. This provided the basis for a massive World Health Organization (WHO) coordinated campaign, which focused on using the insecticide Dichlorodiphenyltrichloroethane (DDT) that killed mosquitoes, which resulted in the elimination of malaria transmission among 500 million people in Africa, [15, 21]. Macdonald termed the latency period as t_m , and introduced the Exposed (E_m) class in the mosquitoes. Therefore, in his model the mosquito population is divided into three compartments (SEI), and the model studies the time evolution of the exposed (E_m) and infected (I_m) classes in mosquito,[18].

Further extension was described by Anderson and May[23], where the latency of infection in humans was introduced by making the additional exposed class in humans. This modification further reduces the long term prevalence of both the infected humans and mosquitoes. Thus, all other mathematical models that exist for malaria dynamics are developed from the three basic models explained earlier by incorporating different factors to make them biologically more realistic in explaining disease prevalence and prediction[27]. For examples, a number of epidemiological studies[13, 11, 14] considered the inclusion of the recovered class which incorporates a time dependent immunity developed on recovery from infection in humans. Further work on acquired immunity in malaria has been conducted by Aron [10] and Bailey [20]. Their models take into account that acquired immunity to malaria depends on continuous exposure to reinfection. Moreover, some models have integrated other factors such as: environmental effects [12, 8, 16]; mosquitoes resistance to insecticides and resistance of some parasite strains to anti-malaria drugs[24].

Total population size was assumed to be constant for all malaria models which came before Ngwa and Shus model, [7]. They proposed an immunity model in which disease related death rate is considered to be significantly high, and the total population is not constant. The Ngwa-Shu model consists of four compartments in humans Susceptible (Sh) , Exposed (Eh) , Infected (Ih) and Immune (Rh) and three compartments in mosquitoes Susceptible (Sm), Exposed (Em), and Infected (Im). Mathematical analysis of the model shows that the basic reproductive number, R_0 , can describe the malaria transmission dynamics of the disease, where a globally stable disease-free state exists if $R_0 < 1$, while for $R_0 > 1$, the endemic equilibrium becomes globally stable. This model explicitly shows the role of inclusion of demographic effects (net population growth) in

predicting the number of fatalities that may arise as a result of the disease.
Some of the more recent papers on the mathematical modeling of malaria have included environmental effects Yang[8] describes a compartmental model where humans follow an SEIRS-type (with more than one immune class for humans) pattern and mosquitoes follow a Susceptible-Exposed-Infectious (SEI) pattern. Additionally, some of the parameters related to mosquitoes are now a function of temperature. These include the time taken for mosquito eggs to develop into adults and the time taken for Plasmodium gametocytes ingested by the mosquito to develop into sporozoites and migrate to the salivary glands (the incubation time in the mosquito).
Yang et al. [17] proposed SIR for the human and SI for the vector compartment model.But in their model, Yang et. al[17] assumed that the number of births for human and mosquito are independent of the total human and mosquito population. This model was modified after wards by Abadi and Krogstad [32] by making the number of births for human and mosquito dependent of the total human and mosquito population.However, Abadi and Krogstad made an assumption that, once the humans enter the recovered class they never go to the susceptible class again.But after some period, the recovered humans lose their immunity and return to the susceptible class[33].Thus, our model is a modification of the model[32] SIR for the human and SI for vector compartment model by introducing the assumption that humans belong to recovered class have possibility to be susceptible that is we consider double immune class for human compartment and we also assume the vaccination at the right time can cause susceptible human beings to recovered class and additionally by considering the existence of human to human transmission of malaria through blood transfusions from malaria infected pregnant women (congenitally). Therefore, our model is SIRS for human population and SI for mosquito population.

CHAPTER THREE

3 Model Formulation and Analysis

3.1 Model formulation

The endemic model of malaria transmission with treatment considered in our study is SIRS in human population and SI in mosquito population which divides human population into three classes namely susceptible human (S_h), infected human (I_h) and recovered (R_h) and divides mosquito population into two classes namely susceptible mosquito (S_m) and infected mosquito (I_m). The mosquito compartment does not include an immune class as mosquitoes never recover from the infection, that is their infective period ends with their death due to their relatively short life cycle. Thus the immune class in the mosquito population is negligible and death occurs equally in all groups. The model is formulated for the spread of malaria in the human and mosquito population with the total population size at time (t) denoted $N_h(t)$ and $N_m(t)$respectively. The basic model incorporates a set of assumptions. These assumptions are:

- Individuals who are born and migrate to the susceptible class has a constant rate of λ_h.
- Humans in susceptible class can move into the infected class due to blood transfusion at a rate of $a\beta 1$(with a is average number of blood transfusion per unit time and β_1 is the chances of disease transmission to susceptible human from infected human) or by an infected mosquito bite at a rate of $b\beta_2$ (with b is average number of infected mosquito bites on susceptible human per unit time and β_2 is the chances of disease transmission from infected mosquitoes to susceptible humans).
- Humans in susceptible class can move into the recovered class due to vaccination at a rate θ.
- Humans insusceptible class may die at a rate of μh.
- A newborn baby can be infected malaria due to congenital with a rate η.
- Humans in infected class can move to the recovered class due to the use of anti-malarial drugs with a rate of γ_k (with k is the rate of human recovery and γ is the effectiveness of anti-malarial drugs).

- Humans in infected class can die at a rate of μ_h and death due to malaria at a rate of α.
- Humans in recovered class (R_h) can die at a rate of μ_h.
  Farther more:
- Mosquitoes are born and migrate to susceptible class with a constant rate of λ_m.
- Mosquitoes in susceptible class may move into the infected class for biting infected humans at a rate of $c\beta_3$ (with c is average number of susceptible mosquito bites on infected humans per unit time and β_3 is the chances of disease transmission from infected humans to susceptible mosquitoes) or can die at a rate μ_m.
- Mosquitoes in susceptible class and infected class can die because the use of spraying at a rate of ρ.
- Mosquito in infected class can die at a rate μ_m.
  Table1.1The state variable for the malaria model(1)-(5)

NO	symbol	description	unit
1	S_h	the number of susceptible humans	number
2	I_h	the number of infected humans	number
3	R_h	the number of recovered humans	number
4	S_m	the number of susceptible mosquito	number
5	I_m	the number of infected mosquitoes	number
6	N_h	the total human population	number
7	N_m	the total mosquito population	number

Table1.2The parameters for the malaria model(1)-(5)

NO	symbol	description	unit
1	λ_h	rate of individuals and new born migrate to susceptible class	number(time^{-1})
2	a	average number of blood transfusion per unit time	time^{-1}
3	β_1	chances of disease transmission to susceptible human from infected human	time^{-1}
4	b	average number of infected mosquito bites on susceptible human per unit time	time^{-1}
5	β_2	chances of disease transmission from infected mosquitoes to susceptible humans	time^{-1}
6	θ	rate of susceptible humans move to recovered class due to vaccination	time^{-1}
7	μ_h	death rate of humans in each class	time^{-1}
8	η	rate of new born baby can be infected due to congenital	time^{-1}
9	k	is the rate of human recovery	time^{-1}
10	γ	is the effectiveness of anti-malarial drugs	time^{-1}
11	α	death rate of humans due to malaria	time^{-1}
12	μ_m	death rate of mosquitoes in each class	time^{-1}
13	ρ	death rate of mosquitoes in the two classes due to spraying	time^{-1}
14	σ	rate of recovered peoples move to susceptible class	time^{-1}
15	c	average number of susceptible mosquito bites on infected humans per unit time	time^{-1}
16	β_3	is the chances of disease transmission from infected humans to susceptible mosquitoes	time^{-1}
17	λ_m	Per capita birth rate for mosquitoes	number(time^{-1})

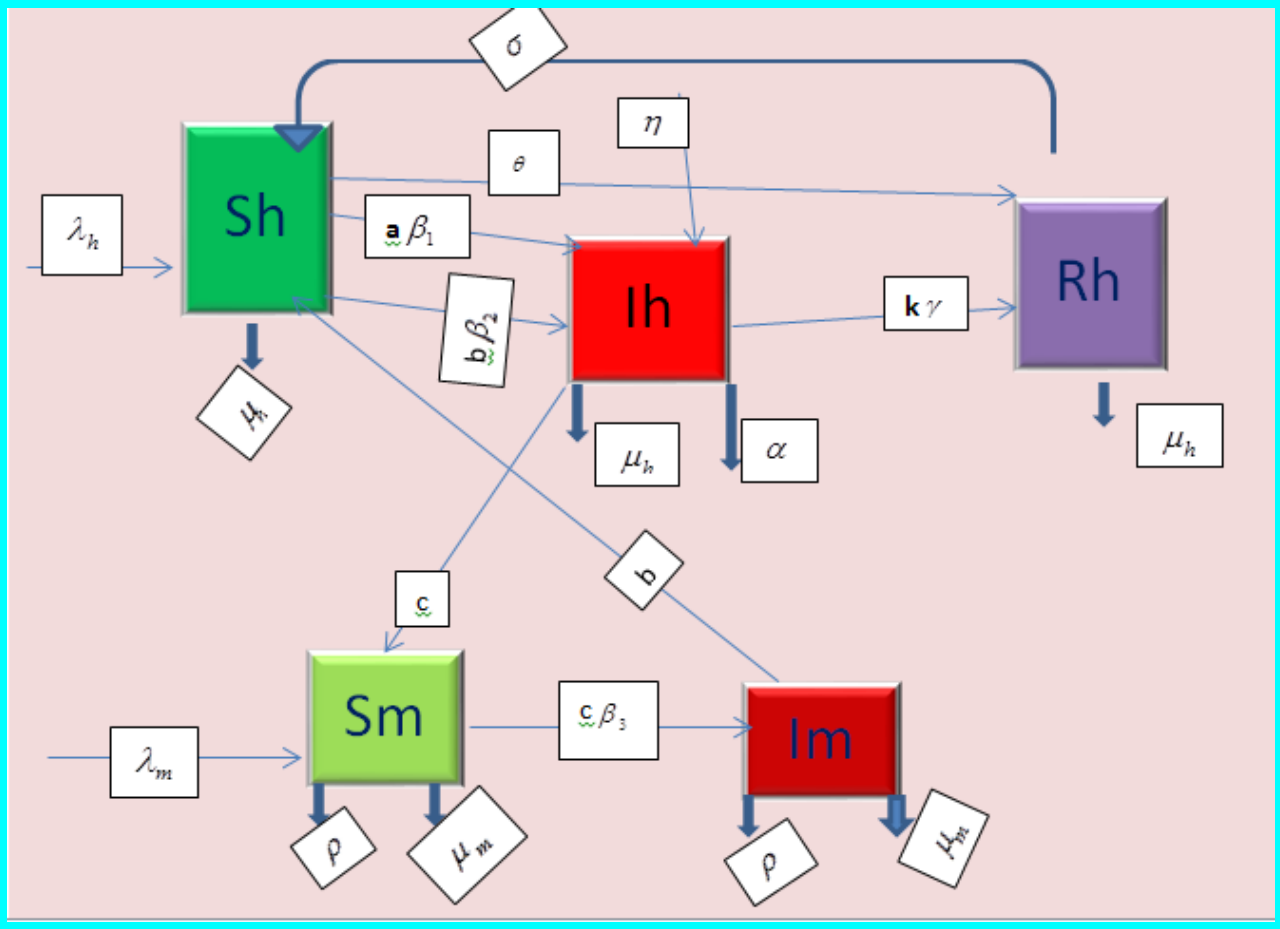

Figure 1: The compartmental model for malaria transmission

By considering the above assumptions, the mathematical model which describe the dynamics of malaria in the human and mosquito populations using a system of nonlinear ordinary differential equation becomes

$$\frac{dS_h}{dt} = \lambda_h + \sigma R_h - (a\beta_1 I_h + b\beta_2 I_m)S_h - (\theta + \mu h)S_h \tag{1}$$

$$\frac{dI_h}{dt} = \eta + (a\beta_1 I_h + b\beta_2 I_m)S_h - (\mu h + \alpha + k\gamma)I_h \tag{2}$$

$$\frac{dR_h}{dt} = k\gamma I_h - (\mu + \sigma)R_h + \theta S_h \tag{3}$$

$$\frac{dS_m}{dt} = \lambda m - (c\beta_3 I_h + \mu_m + \rho)S_m \tag{4}$$

$$\frac{dI_m}{dt} = c\beta_3 I_h S_m - (\mu_m + \rho)I_m \tag{5}$$

Where θ, $k\gamma$, ρ are treatment terms and with initial conditions

$S_h(0) = S_{h0}$
$I_h(0) = I_{h0}$
$R_h(0) = I_{h0}$
$S_m(0) = S_{m0}$
$I_m(0) = I_{m0}$
$N_h = N_{h0}$
$N_m(0) = N_{m0}$
such that all the initial values are non negative.

- The total population N_h of human population and the total population of mosquito N_m are given by:

$$\begin{aligned} S_h + I_h + R_h &= N_h \\ S_m + I_m &= N_m \end{aligned} \tag{6}$$

Then

$$\begin{aligned} \frac{dN_h}{dt} &= \frac{dS_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt} \\ &= \lambda h + \sigma R_h - (a\beta_1 + b\beta_2 I_m)S_h \\ &-(\theta + \mu h)S_h + \eta I_h + (a\beta_1 I_h \\ &+b\beta_2 I_m)S_h - (\mu h + \alpha + k\gamma)I_h \\ &+k\gamma I_h - (\mu + \sigma)R_h + \theta S_h \\ &= \lambda h - \mu_h S_h + \eta I_h - \mu_h I_h - \alpha I_h - \mu_h R_h \\ &= \lambda h - (S_h + Ih + Rh)\mu_h + \eta I_h - \alpha I_h \\ &= \lambda h - \mu_h N_h + (\eta - \alpha)I_h \end{aligned} \tag{7}$$

Similarly

$$\begin{aligned} \frac{dN_m}{dt} &= \frac{dS_m}{dt} + \frac{dI_m}{dt} \\ &= \lambda m - (c\beta_3 I_h + \mu m + \rho)S_m + c\beta_3 I_m S_m - (\mu_m + \rho)I_m \\ &= \lambda m - c\beta_3 I_h S_m + \mu m S_m + \rho S_m + c\beta_3 I_h S_m - \mu_m I_m + \rho I_m \\ &= \lambda m - (S_m + I_m)\mu m - (S_m + I_m)\rho \\ &= \lambda m - \mu m N_m - \rho N_m \\ &= \lambda m - (\mu m + \rho)N_m \end{aligned} \tag{8}$$

In this model, λ_h and λ_m denotes total birth rates of humans and mosquitoes respectively.The terms μS_h, μI_h and μR_h refer to the total number of removed susceptible, infected and recovered humans per unit time respectively.
The term $(\rho+\mu_m)S_m$ and $(\rho+\mu_m)I_m$ are the number of susceptible and infected mosquito populations per unit time.The term αI_h is the number of removed human population because of disease per unit time and the term ηI_h is the number of new infected baby due to congenital.
$k\gamma I_h$ is the total recovered human population due to use of anti-malarial drugs per unit time. The term $b\beta_2 I_m S_h$ denotes the rate at which the human hosts S_h get infected by the mosquito vector I_m and the term $a\beta_1 I_h S_h$ denotes the rate at which the human hosts S_h get infected by blood transfusion. $c\beta_3 I_h S_m$ refers to the rate at which the susceptible mosquitoes S_m are infected by the human hosts I_h at a time t.Thus both these terms are important parts of the model describing the interaction between the two population.

3.2 Analysis of Model

3.2.1 Positivity of Solutions

- let the initial condition be

$$\Omega_H = \{S_h(0) = S_{h0} \geq 0, I_h(0) = I_{h0} \geq 0, R_h(0) = R_{h0} \geq 0\}$$

and

$$\Omega_M = \{S_m(0) = S_{m0} \geq 0, I_m(0) = I_{m0} \geq 0\} \in \Omega$$

Then the solution $S_h(t), I_h(t), R_h(t), S_m(t), I_m(t)$ of the system (1)-(5) is positive for all, $t > 0$
proof
from model (1) we have

$$\frac{dS_h}{dt} = \lambda_h + \sigma R_h - (a\beta_1 I_h + b\beta_2 I_m)S_h - (\theta + \mu h)S_h$$

$$\begin{aligned}\frac{dS_h}{dt} &= \lambda_h + \sigma R_h - (a\beta_1 I_h + b\beta_2 I_m)S_h - (\theta + \mu h)S_h \\ &\geq -(a\beta_1 I_h + b\beta_2 I_m)S_h - (\theta + \mu h)S_h\end{aligned}$$

Without loss of generality the inequality can be expressed as

$$\frac{dS_h}{dt} \geq -[a\beta_1 I_h + b\beta_2 I_m + \theta + \mu h]S_h$$

integrating both side gives

$$\int \frac{1}{S_h} dS_h \geq -\int [a\beta_1 I_h + b\beta_2 I_m + \theta + \mu h]d(t) + c$$

$$S_h(t) \geq e^{-[a\beta_1 I_h + b\beta_2 I_m + \theta + \mu h]t + c} = Ae^{-[a\beta_1 I_h + b\beta_2 I_m + \theta + \mu h]t}$$

where $A = e^c$ at, $t = 0$, $S_h(0) = A \geq 0$
Thus,

$$S_h(t) \geq e^{-[a\beta_1 I_h + b\beta_2 I_m + \theta + \mu h]t + c} \geq 0 \tag{9}$$

From the second equation of the dynamical model system of equation(2)

$$\frac{dI_h}{dt} = \eta I_h + (a\beta_1 I_h + b\beta_2 I_m)S_h - (\mu h + \alpha + k\gamma)I_h$$

Without loss of generality this can be expressed, as an inequality as

$$\frac{dI_h}{dt} \geq -kI_h, \qquad where\ k = \mu_h + \alpha + k\gamma - \eta$$

Upon integrating this by using separation of variables we have the first order linear ordinary differential equation and can be solved to obtain particular solution as

$$\frac{dI_h}{dt} \geq -kI_h,$$

$$\int \frac{dI_h}{dt} \geq \int -kI_h$$

$$I_h(t) \geq I_{h0}e^{-kt}$$

Here I_{h0} is integral constant and represents initial population of infected human compartment and hence it is positive quantity and the exponential function always positive as $t \to \infty$ the analytical solution leads to $I_h(t) > 0$.Therefore the population size of infected human compartment $I_h(t)$ is always positive.
From the third equation of the dynamical model system of equation(3)

$$\frac{dR_h}{dt} = k\gamma I_h - (\mu + \sigma)R_h + \theta S_h$$

Without loss of generality this can be expressed, as an inequality as

$$\frac{dI_h}{d_t} \geq -qR_h, \qquad where\ q = \mu + \sigma$$

integrating this by using separation of variables we have the first order linear ordinary differential equation and can be solved to obtain a particular solution as

$$\frac{dR_h}{d_t} \geq -qR_h,$$

$$\int \frac{dR_h}{d_t} \geq \int -qR_h$$

$$R_h(t) \geq R_{h0}e^{-qt}$$

Here R_{h0} is integral constant and represents initial population of recovered human compartment and hence it is positive quantity and the exponential function always positive as $t \to \infty$ the analytical solution leads to $R_h(t) > 0$.Therefore the population size of recovered human compartment $R_h(t)$ is always positive. From the fourth equation of the dynamical model system of equation(4)

$$\frac{dS_m}{dt} = \lambda m - (c\beta_3 I_h + \mu_m + \rho)S_m$$

Without loss of generality this can be expressed, as an inequality as

$$\frac{S_m}{dt} \geq -(c\beta_3 I_h + \mu_m + \rho)S_m, \qquad r = c\beta_3 I_h + \mu_m + \rho$$

Up on integrating the inequality its analytic solution is obtained as

$$\frac{dS_m}{d_t} \geq -rS_m,$$

$$\int \frac{dS_m}{d_t} \geq \int -rS_m \qquad where\ r = c\beta_3 I_h + \mu_m + \rho$$

$$S_m(t) \geq S_{m0}e^{-rt}$$

Since $S_{m0} \geq 0$ and the exponential function always positive , it is clear that $S_m(t) \geq 0$. From the fifth equation of the dynamical model system of equation(5)

$$\frac{dI_m}{dt} = c\beta_3 I_h S_m - (\mu_m + \rho)I_m$$

Without loss of generality this can be expressed, as an inequality as

$$\frac{dI_m}{dt} \geq -(\mu_m + \rho)I_m, \qquad p = (\mu_m + \rho)$$

Up on integrating the inequality its analytic solution is obtained as

$$\frac{dI_m}{d_t} \geq -pI_m,$$

$$\int \frac{dI_m}{d_t} \geq \int -pI_m$$

$$I_m(t) \geq I_{m0}e^{-pt}$$

Here I_{m0} is integral constant and represents initial population of infected mosquito compartment and hence it is positive quantity and the exponential function always positive as $t \to \infty$ the analytical solution leads to $I_m(t) > 0$.Therefore the population size of infected mosquito compartment $I_m(t)$ is always positive. Therefore,for all t,$S_h(t) \geq 0$, $I_h(t) \geq 0$,$R_h(t) \geq 0$, $S_m(t) \geq 0$, $I_m(t) \geq 0$.

3.2.2 Boundedness of the solution

Let D be a domain of the system (1)-(5) and the total human population size N(t) is given by:
$N(t) = S_h(t) + I_h(t) + R_h(t)$ implies that the system (1)-(5)

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt}$$

$$= \lambda h + \sigma R_h - (a\beta_1 I_h + b\beta_2 I_m)S_h$$
$$-(\theta + \mu h)S_h + \eta I_h + (a\beta_1 I_h + b\beta_2 I_m)S_h$$
$$-(\mu h + \alpha + k\gamma)I_h + k\gamma I_h - (\mu + \sigma)R_h + \theta S_h$$
$$= \lambda h - \mu h(S_h + I_h + R_h) - (\alpha + k\gamma - \eta)I_h$$

$$\frac{dN_h}{dt} \leq \lambda_h - \mu_h N_h \tag{10}$$

$$\frac{dN_h}{dt} + \mu_h N_h \leq \lambda_h \tag{11}$$

multiplying both sides of (11) by $e^{\mu t}$ gives

$$\begin{aligned} e^{\mu t}\frac{dN_h}{dt} + \mu_h N_h e^{\mu t} &\leq \lambda_h e^{\mu t} \\ \frac{d}{d_t}(N_h e^{\mu t}) &\leq \lambda_h e^{\mu t} \end{aligned} \tag{12}$$

integrating both sides of (12) gives

$$N_h e^{\mu t} \leq \frac{\lambda_h}{\mu_h} e^{\mu t} + c \tag{13}$$

where c is a constant of integration and
Dividing(13) by $e^{\mu t}$ gives

$$N_h \leq \frac{\lambda_h}{\mu_h} + ce^{-\mu t} \tag{14}$$

using initial conditions at $t = 0, N_h(0) = N_{h0}$

$$\begin{aligned} N_{h0} &\leq \frac{\lambda_h}{\mu_h} + c \\ \Rightarrow N_{h0} - \frac{\lambda_h}{\mu_h} &\leq c \end{aligned} \tag{15}$$

$$N_h \leq \frac{\lambda_h}{\mu_h} + (N_{h0} - \frac{\lambda_h}{\mu_h})\, e^{-\mu t} \tag{16}$$

applying theorem of differential inequality [30] we obtain

$$0 \leq N_h \leq \frac{\lambda_h}{\mu_h} \tag{17}$$

as $t \to \infty$.
Therefore as $t \to \infty$ in (16) the human population approaches to $k = \frac{\lambda_h}{\mu_h}$(that is$N_h \to k = \frac{\lambda_h}{\mu_h}$), the parameter k usually called carrying capacity [31].Hence all solutions set of the human populations of the model (1)-(5) are bounded. Therefore $N \in \left[0, \frac{\lambda_h}{\mu_h}\right]$
$\mathrm{D}_h = \left\{(S_h, I_h, R_h) \in \mathbb{R}^3; S_h > 0, I_h \geq 0, R_h \geq 0, N_h \leq \frac{\lambda_h}{\mu_h}\right\}$
similarly,the solutions set of the mosquito population are bounded.
$D_m = \left\{(S_m, I_m) \in \mathbb{R}^2 : \ S_m > 0, \ I_m \geq 0, \ N_m \leq \frac{\lambda_m}{\mu_m}\right\}$.
Therefore,the solutions for model(1)-(5) is given by
$D = \left\{(S_h, I_h, R_h, S_m, I_m) \in \mathbb{R}^5 : (S_h, I_h, R_h) \geq 0, \ (S_m, I_m) > 0, \ N_h \leq \frac{\lambda_h}{\mu_h}, \ N_m \leq \frac{\lambda_m}{\mu_m}\right\}$
All the Solutions remains positive for all time t and the model (1)-(5) is biologically meaningful and mathematically bounded in domain D.

3.2.3 Existence of Disease Free Equilibrium Point

The disease-free equilibrium point is steady state solutions where there is no malaria infection.Thus disease free equilibrium point,E_0,for the malaria model(1)-(5)implies that $I_h^* = 0$ and $I_m^* = 0$ and solving (1),(3) and (5) yields

$$S_h^* = \frac{\lambda_h(\mu_h + \sigma)}{\mu_h(\theta + \sigma + \mu_h)}$$

$$R_h^* = \frac{\lambda_h\theta}{\mu_h(\theta + \sigma + \mu_h)}$$

and

$$S_m^* = \frac{\lambda_m}{\mu_m + \rho}$$

respectively. thus we are obtain

$$E_0 = \left(\frac{\lambda_h(\mu_h + \sigma)}{\mu_h(\theta + \sigma + \mu_h)}, 0, \frac{\lambda_h\theta}{\mu_h(\theta + \sigma + \mu_h)}, \frac{\lambda_m}{\mu_m + \rho}, 0\right)$$

3.2.4 Basic Reproduction number

Intuitively from the epidemiological point of view, the basic reproduction number(R_0) is the average number of new cases(infections), that one infected case will generate during its entire infectious lifetime. It is very important in determining whether the disease persist in the population or die out. We use the next generation method to compute R_0. Let us assume that there are n compartments of which the first m compartments correspond to infected individuals.
Let

- $F_i(x)$ be the rate of appearance of new infections in compartments i,
- V_i^+ is the rate of transfer of individuals into compartment i by all other means and
- V_i^- 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 non negative initial conditions together with the following system of equations:

$$\frac{dx_i}{dt} = f_i = F_i(x)V_i(x), \quad i = 1, 2, 3, ... \tag{18}$$

where $Vi(x)=Vi^-$ - Vi^+
The next step is the computation of the square matrices F and V of order mxm, where m is the number of infected classes, defined by

$$F = \left[\frac{\partial F_i}{\partial X_j}(X_0)\right]$$

and

$$V = \left[\frac{\partial V_i}{\partial X_j}(X_0)\right]$$

with 1 $\leq$i,j$\leq$m
Such that F is non negative, V is a non-singular matrix and E_0 is the disease-free equilibrium point .

Consequently,

$$\mathbf{F} = \begin{pmatrix} F_{11} \\ F_{12} \end{pmatrix} \quad = \begin{pmatrix} f_{11} \\ f_{12} \end{pmatrix} \quad = \begin{pmatrix} [a\beta_1 I_h + b\beta_2 I_m]S_h \\ [c\beta_3 I_h S_m] \end{pmatrix}$$

By linearization approach the associated matrix F at disease free equilibrium point E_0 is given by

$$\mathbf{F} = \begin{pmatrix} \frac{\partial f_1}{\partial I_h} & \frac{\partial f_1}{\partial I_m} \\ \frac{\partial f_2}{\partial I_h} & \frac{\partial f_2}{\partial I_m} \end{pmatrix} \quad = \begin{pmatrix} a\beta_1 S_h & b\beta_2 S_h \\ c\beta_3 S_m & 0 \end{pmatrix}$$

similarly

$$\mathbf{V} = \begin{pmatrix} V_{11} \\ V_{12} \end{pmatrix} \quad = \begin{pmatrix} v_{11} \\ v_{12} \end{pmatrix} \quad = \begin{pmatrix} \frac{\partial v_1}{\partial I_h} & \frac{\partial v_1}{\partial I_m} \\ \frac{\partial v_2}{\partial I_h} & \frac{\partial v_2}{\partial I_m} \end{pmatrix} \quad = \begin{pmatrix} \mu_h + \sigma + k\gamma - \eta & 0 \\ 0 & -(\mu_m + \rho) \end{pmatrix}$$

$$\mathbf{V}^{-1} = \begin{pmatrix} \frac{1}{-(\mu_h+\sigma+k\gamma-\eta)} & 0 \\ 0 & \frac{1}{-(\mu_m+\rho)} \end{pmatrix}$$

Since F is non-negative and V is non-singular, then $V^{-1}$is non-negative and also $FV^{-1}$is non-negative. Hence the matrix of FV^{-1} is called as the next generation matrix for the model. Finally, the basic reproduction number R_0 (reproduction ratio) is given by R_0= $\rho(FV^{-1})$ where $A = FV^{-1}$.

Then $\rho(A)$ denotes the spectral radius of a matrix A and the spectral radius, $\rho(FV^{-1})$, is the biggest non negative eigenvalue of the next generation matrix.

Equilibrium point of equation (1)-(5) by simultaneously solving the following equation

$$\frac{dS_h}{dt} = 0, \frac{dI_h}{dt} = 0, \frac{dR_h}{dt} = 0, \frac{dS_m}{dt} = 0, \frac{dI_m}{dt} = 0$$

The differential equations of (1)-(5) disease states F and the transfer states V with respect to I_h and I_m at the disease free equilibrium is given as

$\mathrm{E}_0(S_h, I_h, R_h, S_m, I_m) = (S_h^*, 0, R_h^*, S_m^*, 0)$

, where

$$S_h^* = \frac{\lambda_h(\mu_h + \sigma)}{\mu_h(\theta + \sigma + \mu_h)}$$

$$R_h^* = \frac{\lambda_h(\theta)}{\mu_h(\theta + \sigma + \mu_h)}$$

$$S_m^* = \frac{\lambda_m}{\mu_m + \rho}$$

Hence

$$F = \begin{pmatrix} \frac{a\beta_1\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h)} & \frac{b\beta_2\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h)} \\ \frac{c\beta_3\lambda_m}{\mu_m+\rho} & 0 \end{pmatrix}$$

$$\mathbf{V}^{-1} = \begin{pmatrix} \frac{1}{(\mu_h+\sigma+k\gamma-\eta)} & 0 \\ 0 & \frac{1}{(\mu_m+\rho)} \end{pmatrix}$$

$$\mathbf{FV}^{-1} = \begin{pmatrix} \frac{a\beta_1\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h)} & \frac{b\beta_2\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h)} \\ \frac{c\beta_3\lambda_m}{\mu_m+\rho} & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{(\mu_h+\sigma+k\gamma-\eta)} & 0 \\ 0 & \frac{1}{(\mu_m+\rho)} \end{pmatrix}$$

Basic reproductive number R_0 is the largest positive eigenvalues of the matrix $K = FV^{-1}$from which we obtain

$\mathrm{R}_0 = \frac{b_1+\sqrt{b_1^2+4b_2b_3}}{2}$

where

$$b_1 = \frac{a\beta_1\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma\mu_h)(-\eta+\mu_h+\alpha+k\gamma)}$$

$$b_2 = \frac{b\beta_2\lambda_h(\mu_h+\sigma)}{(\mu_m+\rho)\mu_h(\theta+\alpha+\mu_h)}$$

$$b_3 = \frac{c\beta_3\lambda_m}{(\mu_m+\rho)(-\eta+\mu_h+\alpha+k\gamma)}$$

3.2.5 Local Stability of Disease Free Equilibrium Point

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

$$E_0=(\mathrm{S}_h^*, I_h^*, R_h^*, S_m^*, I_m^*) = \left(\frac{\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h)}, 0 \ , \frac{\lambda_h\theta}{\mu_h(\theta+\sigma+\mu_h)}, \frac{\lambda_m}{\mu_m+\rho}, 0\right)$$

Theorem 3.2:The disease free state ,E_0,is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

proof

The Jacobin matrix of the system (1)-(5) evaluated at the disease free equilibrium point E_0,is given by

$$J(E_0)=\begin{pmatrix} -(\theta+\mu_h) & 0 & \sigma & 0 & 0 \\ 0 & \eta-(\mu_h+\alpha+k\gamma) & 0 & 0 & b\beta_2\frac{\lambda_h(\mu+\sigma)}{\mu_h(\theta+\sigma+\mu_h)} \\ 0 & k\gamma & -(\mu_h+\sigma) & 0 & 0 \\ 0 & -c\beta_3\frac{\lambda_m}{\mu_m+\rho} & 0 & -(\mu+\rho) & 0 \\ 0 & c\beta_3\frac{\lambda_m}{\mu_m+\rho} & 0 & 0 & -(\mu_m+\rho) \end{pmatrix}$$

We need to show that all the eigenvalues of $J(E_0)$ are negative.As the first and the fourth columns contain only the diagonal terms which form the two negative eigenvalues,$-(\theta+\mu_h)$,$-(\mu_m+\rho)$,the other three eigenvalues can be obtained from the sub-matrix,$J_1(E_0)$,formed by excluding the first and the fourth rows and columns of $J(E_0)$. Hence we have

$$J_1(E_0)=\begin{pmatrix} \eta-(\mu_h+\alpha+k\gamma) & 0 & b\beta_2\frac{\lambda_h(\mu+\sigma)}{\mu_h(\theta+\sigma+\mu_h)} \\ k\gamma & -(\mu_h+\sigma) & 0 \\ c\beta_3\frac{\lambda_m}{\mu_m+\rho} & 0 & -(\mu_m+\rho) \end{pmatrix}$$

In the same way the second column of $J_1(E_0)$contains only the diagonal term which forms a negative eigenvalue,$-(\mu+\sigma)$.The remaining two eigenvalues are obtained from sub-matrix;

$$J_1(E_0)=\begin{pmatrix} \eta-(\mu_h+\alpha+k\gamma) & b\beta_2\frac{\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h)} \\ c\beta_3\frac{\lambda_m}{\mu_m+\rho} & -(\mu_m+\rho) \end{pmatrix}$$

The eigenvalues of matrix $J_2(E_0)$ are the roots of characteristic equation of the matrix.

$$\mathrm{J}_1(E_0)=\begin{pmatrix} [\eta-(\mu_h+\alpha+k\gamma)]-\lambda & b\beta_2\frac{\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h)} \\ c\beta_3\frac{\lambda_m}{\mu_m+\rho} & [-(\mu_m+\rho)]-\lambda \end{pmatrix}$$

Then the characteristic equation is:

$$[(\eta-\mu_h-\alpha-k\gamma)-\lambda][(-\mu_m-\rho)-\lambda]-c\beta_3\frac{\lambda_m}{\mu_m+\rho}.b\beta_2\frac{\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h)}=0$$

$$(-\mu_m-\rho)(\eta-\mu_h-\alpha-k\gamma)-\lambda(-\mu_m-\rho)-\lambda(\eta-\mu_h-\alpha-k\gamma)+\lambda^2-c\beta_3\frac{\lambda_m}{\mu_m+\rho}.b\beta_2\frac{\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h)}=0$$

$$\lambda^2+a_1\lambda+a_2=0 \tag{21}$$

where

$$a_1=\mu_m+\rho-\eta+\mu_h+\alpha+k\gamma$$

$$a_2=-\eta\mu_m+\mu_h\mu_m+\alpha\mu_m+k\gamma\mu_m-\rho\eta+\rho\mu_h+\alpha\rho+\rho\gamma k-c\beta_3\frac{\lambda_m}{\mu_m+\rho}.b\beta_2\frac{\lambda_h(\mu_h+\sigma)}{\mu_h(\theta+\sigma+\mu_h)}$$

The remaining eigenvalues $\lambda_4$andλ_5 are found by solving equation (21)

$$\lambda^2+a_1\lambda+a_2=0$$

We can see that $\lambda_4=\frac{-a_1-\sqrt{a_1^2-4a_2}}{2}$ and $\lambda_5=\frac{-a_1+\sqrt{a_1^2-4a_2}}{2}$.
Since all the eigenvalues λ_1, λ_2, λ_3, λ_4 and λ_5 have negative real parts.This implies that the disease free equilibrium point is locally asymptotically stable for $R_0<1$.

3.2.6 Global Stability of Disease Free Equilibrium Point

Theorem 3.3:The disease free equilibrium point E_0 of the system (1)-(5) globally stable in region D if $R_0 < 1$. Where $D = D_h \text{x} D_m$; $D_h = \Big\{(S_h,\ I_h,\ R_h) \in \mathbb{R}^3;\ S_h > 0,\ I_h \geq 0,\ R_h \geq 0,\ N_h \leq \frac{\lambda_h}{\mu_h}\}$
$\text{D}_m = \{(S_m\,,\, I_m) \in \mathbb{R}^2;\ S_m > 0,\ I_m \geq 0,\ N_m \leq \frac{\lambda_m}{\mu_m}\}$
proof :-The equation of the infected components can be written in terms of

$$\begin{vmatrix} \frac{dI_h}{dt} \\ \frac{dI_m}{dt} \end{vmatrix} = [F - V] \begin{vmatrix} I_h \\ I_m \end{vmatrix} - \begin{vmatrix} (a\beta_1 I_h + b\beta_2 I_m) - (\frac{a\beta_1 I_h}{N_h} + \frac{b\beta_2 I_m}{N_m})S_h \\ (c\beta_3 I_h) - (\frac{c\beta_3 I_h}{N_h})S_m \end{vmatrix}$$

Then

$$\begin{vmatrix} \frac{dI_h}{dt} \\ \frac{dI_m}{dt} \end{vmatrix} \leq [F - V] \begin{vmatrix} I_h \\ I_m \end{vmatrix}$$

All the eigenvalues of the matrix $[F - V]$ have negative real parts.It follows that the system of linear differential inequality (22) is stable when ever $R_0 < 1$. Consequently by comparison theorem we have $I_h \to 0$ and $I_m \to 0$ as $t \to \infty$.This follows that $(I_h, I_m) \to (0, 0)$ and the remaining equation of system (1)-(5) give us the solution $E_0 = \{S_h, 0, R_h, S_m, 0\}$.Therefore $(S_h, I_h, R_h, S_m, I_m) \to E_0$ as $t \to \infty$.Hence, the disease free equilibrium is globally asymptotically stable whenever $R_0 < 1$.

3.2.7 Existence of Endemic Equilibrium Point

Endemic equilibrium point is a steady-state solution ,where the disease persists in the population.This equilibrium implies that if the carrier population is present in the system, then the infection will be transmitted to the human and the mosquito population. Endemic equilibrium E_1 of our model is obtained by setting right hand side equal to zero.

$$\frac{dS_h}{dt} = 0, \frac{dI_h}{dt} = 0, \frac{dR_h}{dt} = 0, \frac{dS_m}{dt} = 0, \frac{dI_m}{dt} = 0$$

Therefore to obtain the endemic equilibrium point we solve the system of equations simultaneously

$$\begin{aligned} 0 &= \lambda h + \sigma R_h^* - (k_1 I_h^* + k_2 I_m^*)S_h^* - (\theta + \mu h)S_h^* \\ 0 &= (k_1 I_h^* + k_2 I_m^*)S_h^* - \tau I_h^* \\ 0 &= k\gamma I_h^* - (\mu + \sigma)R_h^* + \theta S_h^* \\ 0 &= \lambda m - (k_3 I_h^* + \mu_m + \rho)S_m^* \\ 0 &= k_3 I_h^* S_m^* - (\mu_m + \rho)I_m^* \end{aligned} \tag{23}$$

Where $k_1 = a\beta_1, k_2 = b\beta_2, k_3 = c\beta_3, \tau = \mu_h + \alpha + k\gamma - \eta$.
Hence

$$S_h^{**} = \frac{\lambda_h + \sigma R_h}{k_1 I_h^* + k_2 I_m^* + \theta + \mu}$$

$$I_h^{**} = \frac{k_2 I_m^* S_h^*}{\mu_h + \alpha + k\gamma - (k_1 S_h^* + \eta)}$$

$$R_h^{**} = \frac{k\gamma I_h^* + \theta S_h^*}{\mu + \sigma}$$

$$S_m^{**} = \frac{\lambda_m}{(k_3 I_h^* + \mu_m + \rho)}$$

$$I_m^{**} = \frac{k_3 I_h^* S_m^*}{\mu_m + \rho}$$

obviously the rate of malaria transmission by blood transfusion is lower than that of transmission by bite of infected anopheles mosquito.There fore the result of I_h^{**} is always positive. The local stability of endemic equilibrium point is stated and proved hereunder.

3.2.8 Local Stability of Endemic Equilibrium Point

The equilibrium are obtained by equating the right hand side of the system (1)-(5) to zero. Endemic equilibrium of the model is the steady-state solution of the model in the presence of the disease (malaria).
Theorem 3.4:The positive endemic equilibrium point E^* of the system of equation (23) is locally asymptotically stable if $R_0 > 1$.
proof
The Jacobin matrix of the system (23) evaluated at endemic equilibrium point E^*,is obtained as

$$J(E^*)=\begin{pmatrix} -(k_1 I_h^* + k_2 I_m^* + \theta + \mu_h) & -k_1 S_h^* & \sigma & 0 & -k_2 S_h^* \\ (k_1 I_h^* + k_2 I_m^*) & -(\tau - k_1 S_h^*) & 0 & 0 & k_2 S_h^* \\ \theta & k\gamma & -(\mu_h + \sigma) & 0 & 0 \\ 0 & -k_3 S_m^* & 0 & -(k_3 I_h^* + \mu_m + \rho) & 0 \\ 0 & k_3 S_m^* & 0 & k_3 I_h^* & -(\mu_m + \rho) \end{pmatrix}$$

Now we will show that, If $R_0 > 1$ then $Tr[J(E^*)] < 0$ and $Det[J(E^*) > 0$.

$$J(E^*)=\begin{pmatrix} -(p + \theta + \mu_h) & -z & \sigma & 0 & -m \\ p & -(\tau - z) & 0 & 0 & m \\ \theta & k\gamma & -(\mu_h + \sigma) & 0 & 0 \\ 0 & -q & 0 & -(r + \mu_m + \rho) & 0 \\ 0 & q & 0 & r & -(\mu_m + \rho) \end{pmatrix}$$

Where $p = k_1 I_h^* + k_2 I_m^*$, $q = k_3 S_m^*$, $z = k_1 S_h^*$, $m = k_2 S_h^*$, $r = k_3 I_h^*$
here the condition $Tr[J(E^*)] < 0$ is satisfied.
The next thing to compute determinant of the system

To show $Det[J(E^*) > 0$ consider

$$Det(J(E^*)) = \begin{vmatrix} -x & -z & \sigma & 0 & -m \\ p & -y & 0 & 0 & m \\ \theta & k\gamma & -c & 0 & 0 \\ 0 & -q & 0 & -w & 0 \\ 0 & q & 0 & r & -v \end{vmatrix}$$

Where $x = p + \theta + \mu_h$, $y = \tau - z$, $c = (\mu + \sigma)$, $w = (r + \mu_m + \sigma)$, $v = (\mu_m + \rho)$

$$Det[J(E^*)] = xcmq(w - r) + pcmq(w - r) + \theta\sigma mq(w - r) + pwv(d\sigma + cv) + \theta\sigma(ywv - xycwv)$$

from

$$xcmq(w - r) > 0$$

$$xcmq(r + \mu_m + \rho - r) > 0$$

$$xcmq(\mu_m + \rho) > 0$$

from

$$-[xycwv]$$

$$-[(p + \theta + \mu_h)(\tau - z)(\mu + \sigma)(r + \mu_m + \sigma)(\mu_m + \rho)]$$

$$-[\tau(p + \theta + \mu_h)(-z)(\mu + \sigma)(r + \mu_m + \sigma)(\mu_m + \rho)] \geq 0$$

From the above computation $Det[J(E^*)] > 0$.Therefore $R_0 > 1$, because of $Tr[J(E^*)] < 0$ and $Det[J(E^*) > 0$.This implies that endemic equilibrium point E^* of the system of equation (23) is locally asymptotically stable.

3.3 Bifurcation Analysis of the model

3.3.1 Backward bifurcation

We shall use the following theorem in [4], to show that the system (1)-(5) exhibits backward bifurcation at $R_0 = 1$.
Theorem 3.5:- Consider the following general system of ordinary differential equations with a parameter ϕ

$$\frac{dx}{dt} = f(x, \phi), f : \phi \in \mathbb{R}^n,\ x \in \mathbb{R}^n, \tag{25}$$

Without loss of generality, it is assumed that 0 is an equilibrium for system (25) for all values of the parameter ϕ, (that is $f(0, \phi) \equiv 0$). Assume
(A1) $A = Dxf(0, 0) = (\frac{\partial f_i}{\partial x_i}(0, 0))$ is the linearized matrix of system above around the equilibrium 0 with ϕ evaluated at 0.
(A2) Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts;
(A3) Matrix A has a non-negative right eigenvector w and a left eigenvector v corresponding to

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

$$\begin{aligned} a &= \sum_{i,j,k=1}^{5} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0). \\ b &= \sum_{i,k=1}^{5} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0) \end{aligned} \tag{26}$$

The local dynamics of system (26) around 0 are totally determined by a and b.

1. $a > 0$,$b > 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 < \phi \ll 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium.
2. $a < 0$,$b < 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable; when $0 < \phi \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium;
3. $a > 0$,$b < 0$. When $\phi < 0$ with $|\phi| \ll 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi \ll 1$, 0 is stable, and a positive unstable equilibrium appears;
4. $a < 0$,$b > 0$. When ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

Particularly, if $a > 0$ and $b > 0$, then a backward bifurcation occurs at $\phi = 0$. To apply the above result, the following simplification and change of variables are made on the system (1)-(5) let $S_h = x_1,\ I_h = x_2,\ R_h = x_3,\ S_m = x_4,\ I_m = x_5,\ N_h = x_1 + x_2 + x_3$ and $N_m = x_4 + x_5$

Moreover, by using vector notation $x = (x_1, x_2, x_3, x_4, x_5)^T$, the system can be written in the form $\frac{dx}{dt} = (f_1, f_2, f_3, f_4, f_5)^T$ as follow

$$\begin{aligned} \frac{dx_1}{dt} &= f_1 = \lambda h + \sigma x_3 - (a\beta_1 I_h + b\beta_2 I_m)x_1 - (\theta + \mu h)x_1 \\ \frac{dx_2}{dt} &= f_2 = \eta x_2 + (a\beta_1 x_2 + b\beta_2 x_5)x_1 - (\mu h + \alpha + k\gamma)x_2 \\ \frac{dx_3}{dt} &= f_3 = k\gamma x_2 - (\mu + \sigma)x_3 + \theta x_1 \\ \frac{dx_4}{dt} &= f_4 = \lambda m - (c\beta_3 x_2 + \mu_m + \rho)x_4 \\ \frac{dx_5}{dt} &= f_5 = c\beta_3 x_2 x_4 - (\mu_m + \rho)x_5 \end{aligned}$$

Choose $a\beta_2$=$(a\beta_2)^*$ as bifurcation parameter, then at $R_0 = 1$ we obtain
$b\beta_2 = (a\beta_2)^*$:= $\frac{b_1 + \sqrt{b_1^2 + 4b_2 b_3}}{2}$=$\mathrm{R}_0$
So that the disease free equilibrium E_0 is locally stable when $a\beta_2$<$(a\beta_2)^*$ and is unstable.Thus $(a\beta_2)^*$ is bifurcation value.

The linearized matrix of the system above around disease free equilibrium E_0 and evaluated at $(a\beta_2)^*$ is given by $J(E_0, (a\beta_2)^*)$

$$J(E_0, (a\beta_2)^*) = \begin{pmatrix} -(\theta+\mu_h) & 0 & \sigma & 0 & 0 \\ 0 & \eta-(\mu_h+\alpha+k\gamma) & 0 & 0 & b\beta_2 S_h \\ 0 & k\gamma & -(\mu_h+\sigma) & 0 & 0 \\ 0 & -c\beta_3 S_m & 0 & -(\mu+\rho) & 0 \\ 0 & c\beta_3 S_m & 0 & 0 & -(\mu_m+\rho) \end{pmatrix}$$

where
$S_h = \frac{\lambda_h(\mu+\sigma)}{\mu_h(\theta+\sigma+\mu_h)}$ and $S_m = \frac{\lambda_m}{\mu_m+\rho}$
The eigenvalues λ of $J(E_0, (a\beta_2)^*)$ given by equation above are roots of characteristics of the form
$\lambda^2 + a_1\lambda + a_2 = 0$ from this $\lambda_1 = \frac{-a_1-\sqrt{a_1^2-4a_2}}{2}$,$\lambda_2 = \frac{-a_1+\sqrt{a_1^2-4a_2}}{2}$, if $\mathrm{a}_1 = \sqrt{a_1^2-4a_2}$ $\lambda = 0$
now in this case assume that $a_1 = \sqrt{a_1^2-4a_2}$, i.e $\lambda = 0$
The right eigenvector $w = (w_1, w_2, w_3, w_4, w_5)^T$, associated with this simple zero eigenvalue can be obtained from $J(E_0, (a\beta_2)^*).w = 0$
As a result we have

$$w_1 = \frac{\sigma}{\theta+\mu_h}w_3,\ w_2 = \frac{\mu_h+\sigma}{k\gamma}w_3\ w_2 = \frac{b\beta_2 sh}{\mu_h+\alpha+k\gamma-\eta},\ w_2 = -\frac{(\mu+\rho)}{c\beta_3 sm}w_4$$

Further ,the left eigenvector $V = (V_1, V_2, V_3, V_4, V_5)^T$ corresponding to the simple zero eigenvalue is obtained from $v.J(E_0, (a\beta_2)^*) = 0$
as

$$V_1 = 0,\ V_3 = 0,\ V_4 = 0,\ v_2 = v_2 > 0,\ V_5 = \frac{b\beta_2 sh}{\mu_m+\rho}v_2$$

Computation of a

By computing the second-order partial derivatives at the disease free equilibrium point we have

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_j} = 0,\ j = 1, 3, 4$$

$$\frac{\partial^2 f_2}{\partial x_2 \partial x_j} = 0,\ j = 2, 3, 4, 5$$

$$\frac{\partial^2 f_2}{\partial x_3 \partial x_j} = 0,\ j = 1, 2, 3, 4, 5$$

$$\frac{\partial^2 f_2}{\partial x_4 \partial x_j} = 0,\ j = 1, 2, 3, 4, 5$$

$$\frac{\partial^2 f_2}{\partial x_5 \partial x_j} = 0,\ j = 2, 3, 4, 5$$

Where as

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \frac{\partial^2 f_2}{\partial x_2 \partial x_1} = a\beta_1$$

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_5} = \frac{\partial^2 f_2}{\partial x_5 \partial x_1} = b\beta_2$$

Similarly

$$\frac{\partial^2 f_5}{\partial x_1 \partial x_j} = 0,\ j = 1, 2, 3, 4, 5$$

$$\frac{\partial^2 f_5}{\partial x_2 \partial x_j} = 0,\ j = 1, 2, 3, 5$$

$$\frac{\partial^2 f_5}{\partial x_3 \partial x_j} = 0,\ j = 1, 2, 3, 4, 5$$

$$\frac{\partial^2 f_5}{\partial x_4 \partial x_j} = 0,\ j = 1, 3, 4, 5$$

$$\frac{\partial^2 f_5}{\partial x_5 \partial x_j} = 0,\ j = 1, 2, 3, 4, 5$$

Where as

$$\frac{\partial^2 f_5}{\partial x_2 \partial x_4} = \frac{\partial^2 f_5}{\partial x_4 \partial x_2} = c\beta_3$$

Then

$$a = v_2 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}(0,0) + v_5 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j}(0,0)$$

$$= v_2 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}(0,0) + \frac{b\beta_2 sh}{\mu_m + \rho} v_2 \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j}(0,0)$$

$$= v_2[\sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_2}{\partial x_i \partial x_j}(0,0) + \frac{b\beta_2 sh}{\mu_m + \rho} \sum_{i,j=1}^{5} w_i w_j \frac{\partial^2 f_5}{\partial x_i \partial x_j}(0,0)]$$

$$= v_2[w_1 w_2 (a\beta_1) + w_5 w_1 (b\beta_2) + \frac{b\beta_2 sh}{\mu_m + \rho} w_2 w_4 c\beta_3]$$

$$= v_2[a\beta_1 (\frac{\sigma}{\theta + \mu_h}) w_3 (\frac{\sigma}{k\gamma}) w_3 + \frac{\mu_h + \alpha + k\gamma - \eta}{b\beta_2 sh} w_2 (\frac{\sigma}{\theta + \mu_h}) w_3 + \frac{b\beta_2 sh}{\mu_m + \rho} - (\frac{c\beta_3 s_m}{\mu_m + \rho}) w_2 w_2]$$

$$= v_2[a\beta_1 (\frac{\sigma}{\theta + \mu_h})(\frac{\mu_h + \sigma}{k\gamma}) w_3^2 + \frac{\mu_h + \alpha + k\gamma - \eta}{b\beta_2 sh} (\frac{\mu_h + \sigma}{k\gamma})(\frac{\sigma}{\theta + \mu_h}) w_3^2 - \frac{b\beta_2 sh}{\mu_m + \rho} \frac{c\beta_3 s_m}{\mu_m + \rho}^2 w_3^2]$$

$$= (\frac{\mu_h + \sigma}{k\gamma})[a\beta_1 (\frac{\sigma}{\theta + \mu_h}) + \frac{\mu_h + \alpha + k\gamma - \eta}{b\beta_2 sh} (\frac{\sigma}{\theta + \mu_h}) - \frac{b\beta_2 sh(c\beta_3 s_m)}{(\mu_m + \rho)^2} (\frac{\mu_h + \sigma}{k\gamma}] v_2 w_3^2$$

Computation of b

To compute b we need to find the second order derivatives of f_2 and f_5 with respect to x_i and $b\beta_2$ at the disease free equilibrium point. Direct computation shows

$$\frac{\partial^2 f_2}{\partial x_i \partial b\beta_2} = 0, \ i = 1, 2, 3, 4$$

$$\frac{\partial^2 f_5}{\partial x_i \partial b\beta_2} = 0, \ i = 1, 2, 3, 4, 5$$

and

$$\frac{\partial^2 f_2}{\partial x_5 \partial b\beta_2} = b\beta_2$$

$$b = \sum_{i,k=1}^{5} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial b\beta_2}(0,0) = b\beta_2 v_k w_i > 0$$

Hence the following result holds. The malaria model (1)-(5) exhibits backward bifurcation at $R_0 = 1$ whenever a is positive.But $a > 0$ if and only if

$$\frac{b\beta_2 sh(c\beta_3 s_m)}{(\mu_m + \rho)^2} (\frac{\mu_h + \sigma}{k\gamma})^2 < [(\frac{\mu_h + \sigma}{k\gamma})(\frac{\sigma}{\theta + \mu_h})[a\beta_1 + \frac{\mu_h + \alpha + k\gamma - \eta}{b\beta_2 sh}]]$$

CHAPTER FOUR

4 Numerical Simulations and Result

In this chapter we present a numerical simulations of the model which is carried out using Matlab ode45.

In this simulation, the population dynamics are observed in conditions such that $R_0 < 1$and $R_0 > 1$. Thus we would like to show the effect of vaccination, anti-malarial drug, and spraying in a situation where the disease does nt spread. The data of parameters is gathered from Westi Arsi Zone,Siraro woreda Health Center were malaria disease is highly endemic.And some parameters relied on the studies conducted by various reliable sources.

In 2016 at Siraro woreda malaria concerned data:

NO	parameter	values	references
1	S_{ho}	0.993	data
2	I_{ho}	0.0043	data
3	R_{ho}	0.0032	data
4	S_m	0.6	assumed
5	I_m	0.3	assumed
6	N_h	185739	data
7	N_m	5000	assumed
8	λ_h	0.0277	[1]
9	a	0.038	[1]
10	β_1	0.02	[1]
4	b	0.13	[33]
5	β_2	0.710	[33]
6	θ	0.3	[33]
7	μ_h	0.0004	[33]
8	η	0.005	[16]
9	k	0.611	[33]
10	γ	0.2	[16]
11	α	0.05	[33]
12	μ_m	0.04	[23]
13	ρ	0.5	[33]
14	σ	0.001369	[33]
15	c	0.022	[23]
16	β_3	0.072	[33]
17	λ_m	0.13	[33]

were used for the simulations. In Figure 4.1, the fractions of the populations, S_h ; I_h and R_h are plotted versus time. The susceptible populations will initially decreases with time and then increases and the fractions of infected human populations decrease. The reproduction number is below one and the disease-free equilibrium point $E_0 = (S_h^*, 0, R_h^*)$ is stable.The susceptible and infected mosquito population decreases over time as shown in Figure 4.2.

Simulation of the model(1)-(5) for Susceptible,Infected and Recovered human population.

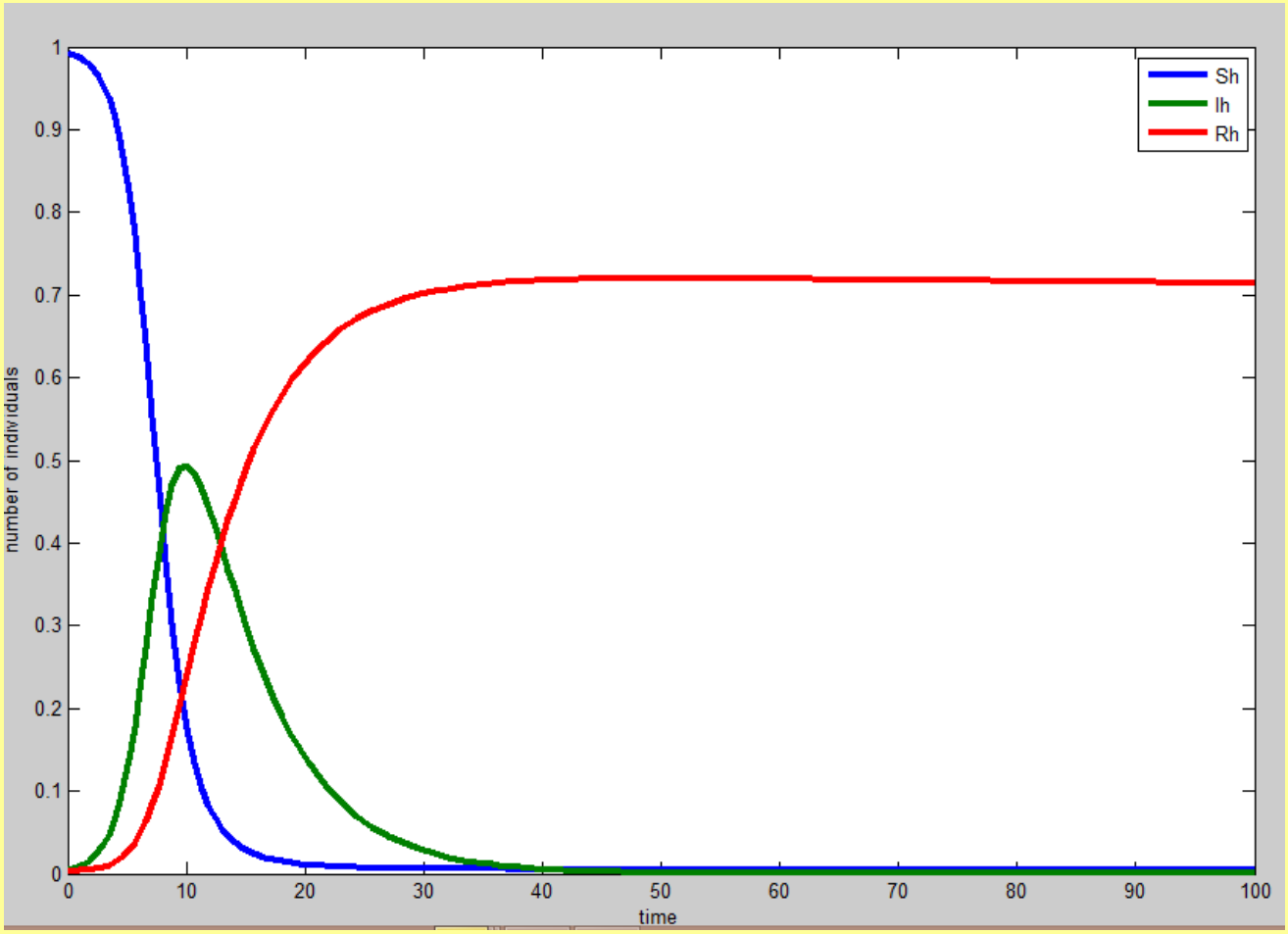

Figure 2: Simulation of the model(1)-(5) for susceptible,infected and recovered human population with the numerical value of $E_0 = \{1.4482, 0, 6.6313, 0.2407, 0\}$ and $E^* = \{0.10996326, 0.1640617, 168.62127, 0.00000241, 0.000007568\}$

Simulation of the model(1)-(5) for susceptible and infected mosquito population.

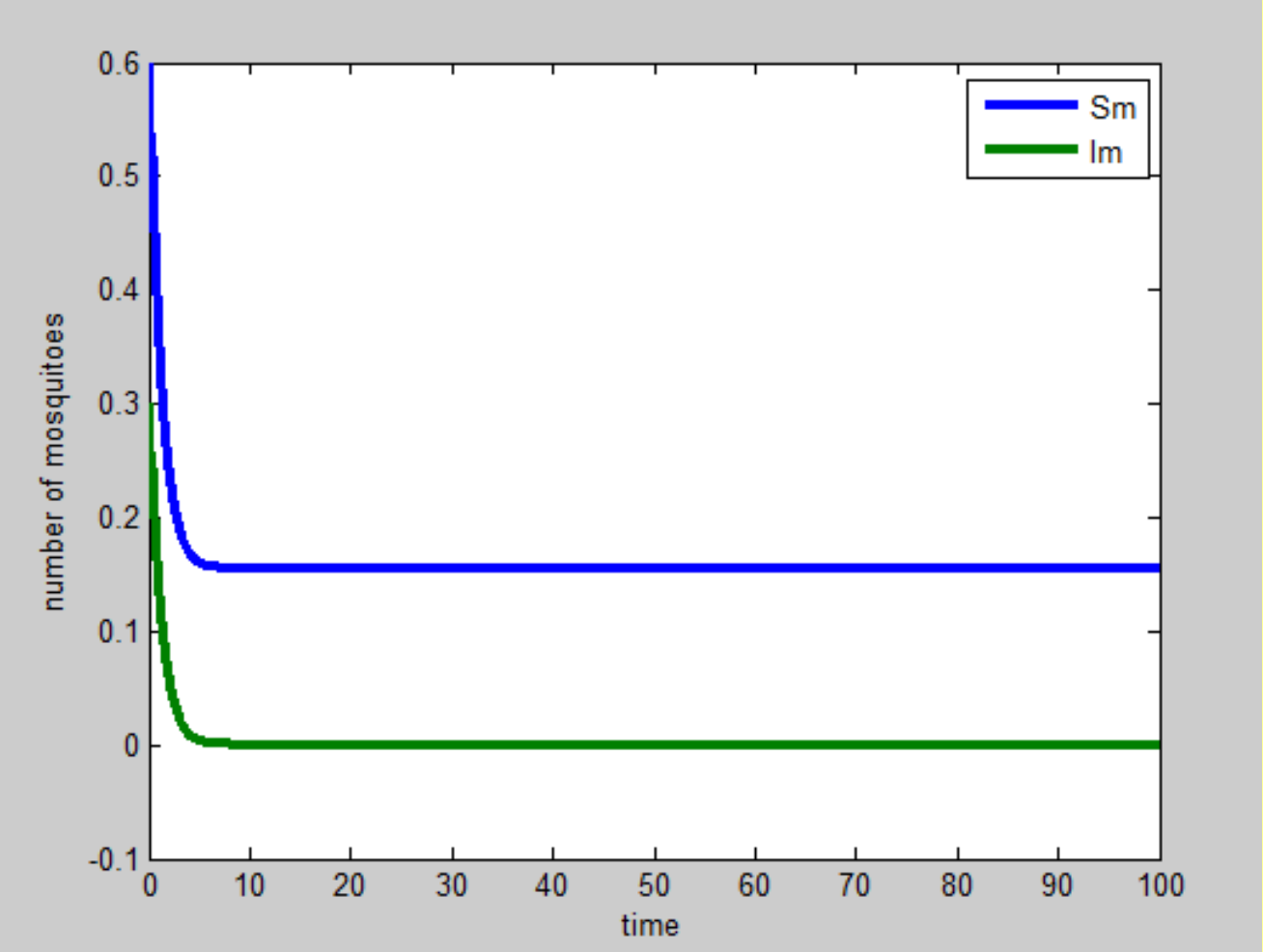

Figure 3: Simulation of the model(1)-(5) for susceptible and infected mosquito population.

Simulation of the model(1)-(5) for human population with treatment given.

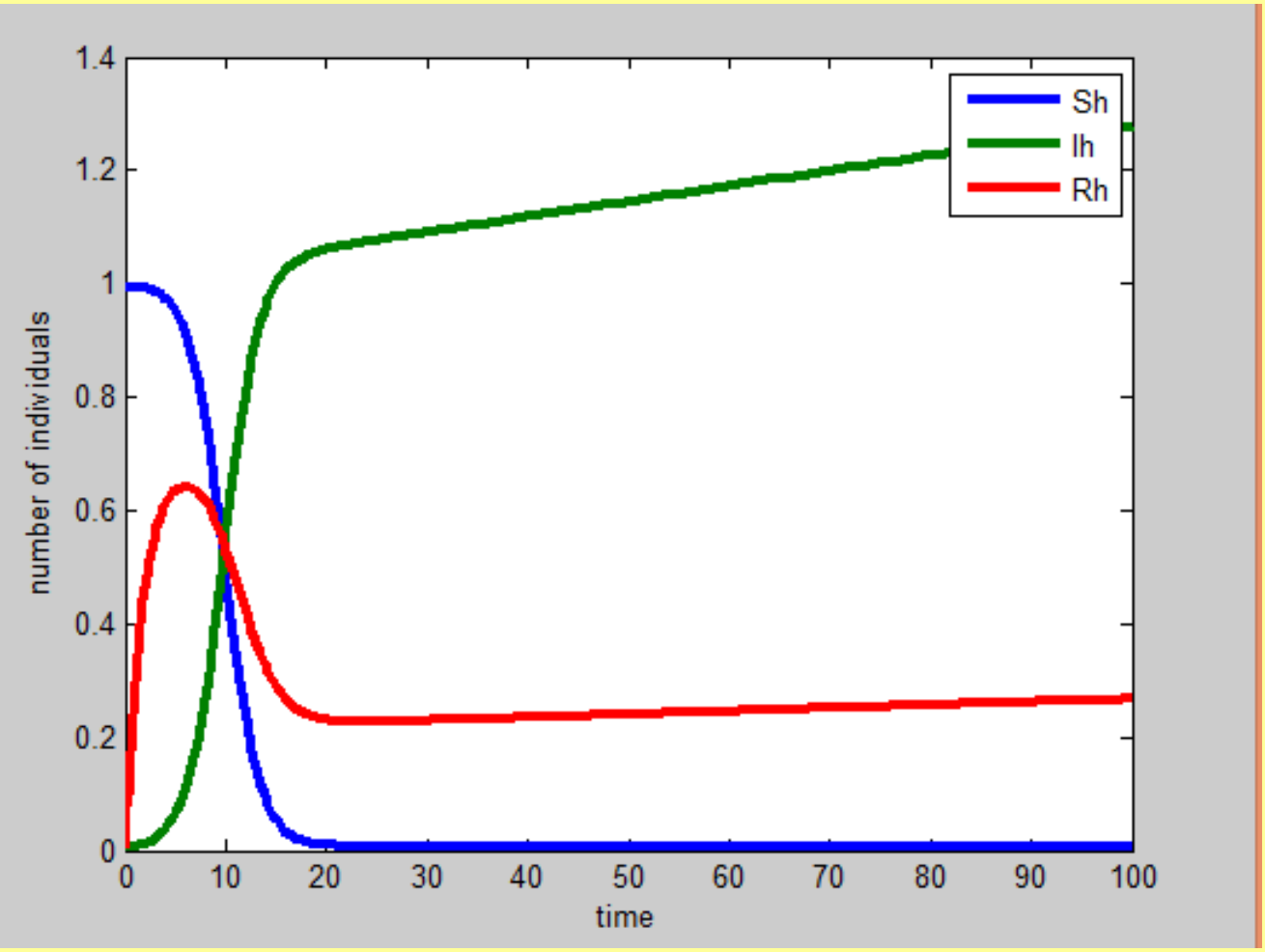

Figure 4: Simulation of the model(1)-(5) for $R0 = 23.934$, with parameter values $\lambda_h = 0.027$, $\mu_h = 0.0004$, $\beta_1 = 0.02$, $\beta_2 = 0.710$, $\beta_3 = 0.072$,$\theta \in [0, 1] = 0.45964$, $\eta = 0.1710$, $\rho \in [0, 1]$, $\sigma = 0.601369$, $\gamma \in [0.01, 1]$, $a = 0.038$, $b = 0.13$, $c = 0.022$, $\mu_m = 0.04$, $\lambda_m = 0.13$

Simulation of the model(1)-(5) for susceptible and infected mosquitoes with treatment given

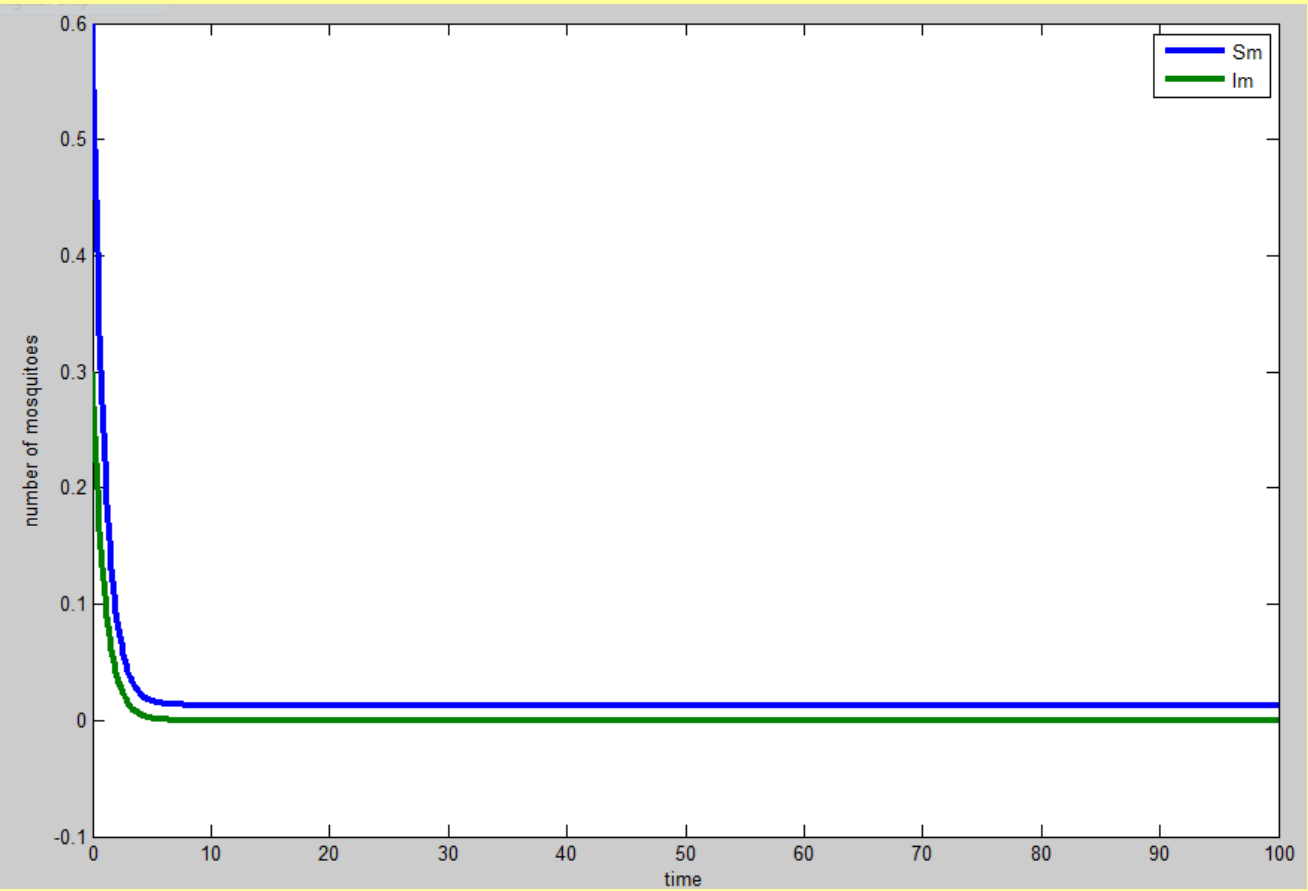

Figure 5: Simulation of the model(1)-(5) for susceptible and infected mosquitoes when λ_m decreased from 0.13 to 0.013 and when $(\mu m + \rho)$ increased from 0.84 to 0.9984.

CHAPTER FIVE

5 Discussions and Conclusion

5.1 Discussions

House spraying with residual Insecticides and mosquito bed nets are the major intervention measures used in these days to prevent malaria transmission. These methods reduce the contact rates between the mosquitoes and humans. Other measures employ the use of anti-malarial drugs which have the effect of reducing the infectivty of the human host. Indoor Residual Spraying (IRS) reduces mosquito longevity and it also reduces mosquito fertility. This strategy is also likely to kill mosquitoes that rest indoors after feeding so it would increase the chances of killing infected mosquitoes.Indoor residual spraying increases the mosquito death rate μ_m and ρ, and reduces the number of mosquitoes, simultaneously it reduces disease transmission by blood transfusion. Increasing μ_m and ρ can be effective in reducing the malaria burden. The other intervention measure, Insecticide-Treated bed Nets, prevent mosquito-human contacts which reduce the number of bites per mosquito. Reducing the number of blood meals that each female mosquito gets would also lower the mosquito recruitment rate, λ_m, which in turn reduces the number of mosquitoes. This is found to be the most effective control strategy in reducing disease transmission. Therefore, all these control strategies are an effective way of controlling most of the parameters which are involved in our model. In order to determine the efficient way to tackle malaria, and reduce malaria mortality, it is necessary to know the relative importance of the different factors responsible for its transmission and prevalence. The basic reproduction number is such an important tool, which allows us to determine the importance of the parameters in the disease transmission. From our computation of the basic reproduction number we have noticed that R_0 is independent of the rate of immunity loss. We have also noticed that an increasing mosquito to human contact rate β_2, the mosquito recruitment rate λ_m , and the human to mosquito contact rate β_3, leads to an increase in the basic reproduction number and this leads to for disease to be hard controlling. Furthermore, we have seen that bringing R_0 less than unity is necessary but not sufficient condition for the disease eradication.

5.2 Conclusion

In this study, a deterministic mathematical model for malaria transmission with treatment has been presented. It was showed that there exists a domain in which the model is mathematically and epidemiological well posed. The next generation matrix was used to derive the basic reproduction number R_0, which is the average number of new cases that one infected case will generate. The disease free equilibrium of the model was proved to be locally asymptotically stable whenever R_0 is less than unity. It is also showed that the disease free equilibrium is globally asymptotically stable provided that the basic reproduction number is less than unity. The possibility of multiple endemic equilibrium point was discussed. It was shown that the model undergo backward bifurcation phenomenon. The stable disease free equilibrium coexist with the stable endemic equilibrium. Bringing the disease (malaria) induced death rate below unity was shown to be sufficient to eliminate backward bifurcation. Thus along with treated bed nets, and insecticides that would reduce the mosquito population there is a need for effective drug and efficient treatment which reduce the number of malaria induced death rate.

We notice that in order to reduce the basic reproduction number below 1, intervention strategies need to be focused on treatment and reduction of the contact between mosquito and human host. Thus, there is a need for effective drugs, treated bed nets, and insecticides that would reduce the mosquito population and keep the human population stable.

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6 Appendices

Appendix A matlab;

```
function start
%-------------------------------------------------------------------------
% Starting script to the module ' SIR model '
%-------------------------------------------------------------------------%

% Implements the basic SIR model , and plots simulation results
%
% User Section 1 : Definition of model parameters
%
%
% These parameters are passed to the function that calculates the
% derivatives .
%
% NOTE: Do not change the name 'param' !
%
param.f1=0.1452 ; % set the parameter 'f1,f2 of the model
param.f2=0.41004;
param.f3=0.0011676; % set the parameter f3 of the model
param.f4=0.59018;% set the parameter f4 of the model
param.k1=0.0037; %set the parameter k1 of the model
param.s1=3.001369; %set the parameter s1 of the model
param.k2=0.122;%set the parameter k2 of the model
param.t2=0.4;%set the parameter t2 of the model
% This is the title string for the plot window .
model_title =  ' SIR model ' ;
% User  Section 2 : Definition of initial conditions
% Initialconditions are the values of all variables at time zero .
%
% NOTE: Do not change the name 'initial'! Define the initial values
% in the same order as the derivatives
initial.S =.993; % set the initial value of 'S'
initial.I = .00423 ; % set the initial value of 'I'
```

```
 initial.R =.0032 ; % set the initial  value of 'R'
% User Section 3 : Definition of the simulation time
end_time=100;
% User Section 4 : Definition of the ODE system
  function deriv = ode_system(t , x, param )
% Function to calculate derivatives of the SIR model
% Input :
%    t : Time (not used in this example because there is no explicit
% time dependence ) .
%    x : Vector of the current values of all variables in the same
% order as you defined the inital values : S , I , R
%   param : Used to pass parameter values .
% Output :
% deriv : Column vector of derivatives , must be the same order as the
% input vectorx .
S = x( 1 ) ;
I = x( 2 ) ;
R = x( 3 ) ;
dS =param.k1+param.s1*R-(param.f1+param.f2) * S * I ;
dI = (param.f1+param.f2)*S*I-param.f3*I ;
% Note : because S+I+R=constant , this equation could actually be omitted
% and R at any time point could simply be calculated as  N  S  I .
dR = param.k2*I-param.f4*R+param.t2*S ;
deriv = [ dS ; dI ; dR ] ;
end
% Now we solve the ODE system and plot the results
% Calculate and print R0 on the screen
N = initial.S + initial.R + initial.I  ;
R0 = param.f1*param.f2* N /param.f3*param.f4
% Extract initial values from the  'initial' structure and collect them
% in acolumn vector for use in  'ode45' .
initial_values =[ ] ;
variable_names = fieldnames (initial ) ;
 for i= 1:length (variable_names )
 initial_values = [initial_values ; initial.(variable_names{i})] ;
end
```

```
% integrate the ODE system
 [t,y] = ode45(@(t,x)ode_system (t, x, param),...
 [0 end_time ],...
initial_values ...
,[])  ;
% prepare legend texts
 legend_texts = cell(length (variable_names ),1) ;
for i=1:length(variable_names )
text =[variable_names{i} '(t)'] ;
legend_texts{i} =text ;
end
% plot the results
plot(t,y ,'linewidth',3) ;
xlabel('time') ;
ylabel('number of individuals');
legend ('Sh','Ih','Rh') ;

end
```

7 Biography

The researcher was born in Ethiopia, Oromia region, West Showa administrative Zone from his father Mr. Ebissa Atoma and his mother Gudine Shanko on may 28, 1986. He attended his education from grade 1-8 at Jarso Dire Gada primary school starting from September, 1994. He then finished his secondary school at Gedo Senior Secondary school. He has taken his National Secondary School Leaving Examination in 2003 and joined Jimma Teachers' Training College in 2003. Then graduated from Jimma in 2006 in dipiloma of teaching Mathematics. He joined for degree program Arba Minch university in 2008 and graduated in BEd Mathematics. Currently he is working at shashemane Woreda Hursa Simbo high School.