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# MATHEMATICAL MODEL FOR THE DYNAMICS AND CONTROL OF MALARIA



A THESIS SUBMITTED IN PARTIAL  
FULFILLMENT OF THE REQUIREMENTS FOR  
THE DEGREE OF MASTERS IN MATHEMATICS  
At  
ADAMA SCIENCE AND TECHNOLOGY UNIVERSITY  
SCHOOL OF APPLIED NATURAL SCIENCES

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## **Certification**

This is to certify that the thesis prepared by Teshome Kasu, entitled "Mathematical Model for the Dynamic and control of Malaria" and submitted in partial fulfillment of the requirement for the degree of Master's of Science in Applied Mathematics (Specialization in Modeling) .

As a member of the board of examiners of the MSc. Thesis open defense examination, we certify that we have read and evaluated the thesis prepared by Teshome Kasu and examined the candidate. We recommended that the thesis be accepted as fulfilling the thesis requirement for the degree of Master's of Science in Applied Mathematics.

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### Abbreviation

DEF	- Disease Free Equilibrium
EE	- Endemic Equilibrium
FMoH	- Federal Ministry of Health
IRS	- Indoor Residual Spraying
ITNS	- Insecticide-Treated Nets
SEIR	- Susceptible-Exposed-Infectious-Recovered
SE	- Susceptible-Exposed-Infectious
SI	- Susceptible-Infectious
SIR	- Susceptible-Infectious-Recovered
SIS	- Susceptible-Infectious-Susceptible
WHO	- World Health Organization

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## Abstract

In this study a deterministic mathematical model is developed to investigate the spread of malaria.

The model has five non-linear differential equations which describe the spread of malaria with three state variables for humans and two state variables for mosquitoes which are  $S_h, E_h, I_h, S_v$  and,  $I_v$  .

Analysis of the model showed that there exists a domain where the model is epidemiologically and mathematically well- posed.

The existence and stability of disease-free and endemic malaria equilibria are analyzed. The key to the analysis is the definition of the basic reproductive number which was derived by use of next generation method.

The disease-free equilibrium is locally asymptotically stable, if the reproduction number is less than one and globally asymptotically stable, if the reproduction number is greater than one.

And the endemic equilibrium exists provided that the basic reproductive number is greater than one. Ordinary differential equations were used to model malaria where humans and mosquitoes interact and infect each other.

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# Chapter 1

## INTRODUCTION

### 1.1 Background of the study

#### 1.1.1 The Biology of malaria

Malaria is one of the most fatal diseases in the world. The symptoms that characterized malaria may have been observed as far back as the prehistoric period, through the classical era, but it was not until the European renaissance period that the name malaria was derived from the Medieval Italian word, mal and aria meaning “bad air”, thinking that the foul vapours emanating from the stagnate water swamps was the cause of fever, a major symptom of the disease [22].

It is believed by some researchers that malaria must have been responsible for the fall of the Roman Empire following an archaeological discovery of the presence of malaria in the bones of a Roman child who died 1500 years ago.

The cause of malaria was not known from the dawn of history until later part of the 19th century when Charles Laveran discovered the malaria parasite in human blood in Africa. Few years later, Giovanni Grassi and Raimondo Filetti used the word plasmodium to name the malaria parasite and in 1897, Ronald Ross demonstrated that plasmodium parasite can be transmitted from infected humans to mosquitoes [11].

### 1.1.2 Life Cycle of malaria

Malaria is a widely spread infectious disease, which is also one of the most prevalent diseases in tropical and subtropical areas.

It is a vector-borne disease. Instead of transmitted directly from human to human, malaria parasites are transmitted between humans through mosquito bite. The malaria parasite has a complicated life cycle involving a mosquito and a human, which can be identified in three phases namely the sporozoite phase, merozoite (or erythrocytic) phase and gametocyte phase. The merozoite phase starts and ends with human host whereas the parasites in the first and third stages need both the mosquito and human environments to thrive.

The female anopheles mosquito requires blood meal to nurture its eggs and during the process of blood feeding it injects the malaria parasite in form of sporozoites that preoccupy its salivary glands into the body of its human host at the site of bite. These sporozoites are conveyed via the circulatory system to the liver after evading innate immune cells, in which they invade hepatic cell.

Each of these sporozoite penetrates a liver cell using it to reproduce asexually through a process often referred to as exoerythrocytic schizogony culminating in the production of merozoites, which are released into the bloodstream. During the process of schizogony an infected hepatic cell or red blood cell passes through four metamorphic stages namely young ring, old ring, young trophozoite and old trophozoite to become a schizont. However, this process may vary depending on the plasmodium species.

For instance, for some malaria parasite such as *plasmodium vivax* and *plasmodium ovale*, the development of certain trophozoites is arrested at earlier stages to form some temporarily dormant cell termed hypnozoites, which may reactivate after some weeks, months, or years being responsible for relapses of the disease. Once these merozoites are released into bloodstream, each starts another round of asexual replication using a red blood cell and after approximately 48 hours, except *plasmodium malariae* that maintains a 72 hours cycle, each surviving merozoite from any of the other three species produces a second

generation of merozoites. Immediately after the erythrocyte invasion, the plasmodium flaciparum parasite has the appearance of a ring and after about 12 hours it gradually adopts a more solid appearance known as a 'young trophozoite' which continues to grow after 24 hours to become a schizont or segment and after about 12 hours later ruptures to release daughter parasites that infect other erythrocytes [22].

The production of second and subsequent generations of merozoites increases the level of parasitemia creating intermittent fever paroxysms and other disease symptoms due to inflammations from continuous rupturing of infected erythrocytes. Plasmodium falciparum merozoites attack all red blood cells, not just the young or old cell, as do other types and a patient with this type of malaria can die within hours of the first symptoms [3, 22]. Prolonged fever destroys so many red blood cells causing blockage of the blood vessels in vital organs (especially the kidneys), which in some cases culminates in the enlargement of the spleen [8]. When malaria infection is left untreated for a long time, it can lead to many complications including severe anemia. There may be brain damage, leading to coma and convulsions.

The kidneys and liver may also fail [22]. An infected red blood cell committed to a further generation of merozoites, passes through a period of schizogony. The period starts from an immature ring stage, through trophozoite stage to a mature schizont, and eventually bursts to release merozoites. As an alternative to continuous merozoite replication cycles, some of these merozoites differentiate into sexual forms of the parasite called gametocyte. These gametocytes, made up of the male form (microgametocytes) and the female form (macrogametocytes) are later picked up by a female anopheles mosquito during blood feeding. Fertilization occurs in the stomach of the mosquito as a microgamete becomes flagellated and penetrates a macrogamete to form zygote. The zygote develops into a motile form Oocinete and penetrates the midgut wall of mosquito for further development into an asexual form Oocyst. After rounds of multiple replications the Oocyst ruptures to release sporozoites, which migrate to the salivary gland of the mosquito waiting to be injected into the skin of the human host [10].

Typical symptoms of malaria infectious start with headache, followed by cyclical fevers and chills, and sometimes even coma. The period of cyclical fevers lasts several days, during which time a high probability of dying has been observed for children, since their immune system are weak. Such fever can also lead to abortions of pregnant women. There are some other possible symptoms such as vogue, sweating, anemia, bloody stools, convulsion, myalgia, diarrhea, and vomiting.

## 1.2 Statement of the problem

Malaria is an infectious disease and widespread in tropical and subtropical regions for thousands of years. The environmental conditions in the tropical areas are the prime factor for malaria being endemic. The moderate-to-warm temperatures, high humidity and water bodies allow mosquito and parasites to produce [16]. The epidemiological patterns of malaria usually vary with seasonal because of its dependence on transmission from mosquito, temperature and geographical condition of the environment. Thus climate change can affect the malaria prevalence pattern by migration from lower latitudes to regions where the humans' population has not developed immunity to disease.

Malaria control is challenging due to many factors. The complexity of the disease control process, cost of the control program and resistance of the parasite to anti-malarial drugs, and vectors to insecticides, are some of the challenges. Malaria has for many years been considered as a global issue, and many epidemiologists and other scientists invest their effort in learning the dynamics of malaria and to control its transmission. From interaction with those scientists, mathematicians have developed 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[21].

The mathematical models have played an important role in the development of malaria epidemiology. Analysis of mathematical models is important because they help in understanding the present and future spreads of malaria so that suitable control technique

can be adopted. [Abadi Abay Gebremeskel,Harald Elias Karogstad]proposed SEIR for the human and SEI for vector compartment , and show the existence and stability of the disease-free equilibrium and an endemic equilibrium.

However, in reality the recovered class is not removed or quaranted from the main population ,in fact they become part of the susceptible class. Thus our model is a modification of [Abadi Abay Gebremeskel,Harald Elias Karogstad] on this basis. Hence in this research work, we will try to understand the important parameters in the transmission and spread of malaria disease, try to develop effective solutions and strategies for its prevention and controls.

Therefore, this study is believed to provide recent and valuable information for who is working in the prevention and control of malaria, and also it can be used as a base line for further study on the transmission and control of malaria. In general this research work would try to address the following questions.

1. What are the basic assumptions to develop a mathematical model which describe the dynamics of malaria?
2. How can we investigate the dynamics of malaria with its control mechanism?
3. Are the systems both locally and globally stable?
4. What is the biological interpretation of the solution of the model equations?

## 1.3 Research objectives

Based on the statement of the problem, the objectives of the study are as follows.

### 1.3.1 General objective

The main objective of this study is to investigate the dynamics of malaria and its control mechanisms.

### 1.3.2 Specific objectives

The specific objectives of the study are to

1. Investigate the dynamics of malaria and its control mechanisms.
2. Construct a mathematical model which describes the dynamic of malaria and its control.
3. Establish the stability properties of the equilibrium points of the model equation and assess the effects of control measures
4. Interpret the biological meaning of the solution of the model equation.

## 1.4 Significance of the study

The health as well as the socioeconomic impacts of appearing and re-appearing vector borne diseases like malaria is significant. The disease is endemic and claims so many lives and consequently makes its study valuable. Therefore, the result of this study would add more knowledge on existing literature. Improve our understanding of the dynamics of malaria in population and its control mechanisms. The results on the effects of the prevention and control measures would provide relevant guidance for decision makers and rising of the relevant social awareness. Suggest the possible control mechanisms.

## Chapter 2

# LITERATURE- REVIEW

Malaria in humans is caused by five species of parasites belonging to the genus *Plasmodium*. Four of these *P. falciparum*, *P. vivax*, *P. malariae* and *P. ovale* are human malaria species that are spread from one person to another via the bite of female mosquitoes of the genus *Anopheles*. There are about 400 different species of *Anopheles* mosquitoes, but only 30 of these are vectors of major importance. In recent years, human cases of malaria due to *P. knowlesi* have been recorded. This species causes malaria among monkeys in certain forested areas of South-East Asia. Current information suggests that *P. knowlesi* malaria is not spread from person to person, but rather occurs in people when an *Anopheles* mosquito infected by a monkey then bites and infects humans [13,16].

*P. falciparum* is most prevalent on the African continent, and is responsible for most deaths from malaria. *P. vivax* has a wider geographical distribution than *P. falciparum* because it can develop in the *Anopheles* mosquito vector at lower temperatures, and in cooler climates[6]. It also has a dormant liver stage (known as a hypnozoite) that can activate months after an initial infection, causing a relapse of symptoms. The dormant stage enables *P. vivax* to survive for long periods when *Anopheles* mosquitoes are not present.

Although *P. vivax* can occur through Africa, the risk of infection with this species is quite low there because of the absence in many African populations of the Duffy gene, which produces a protein necessary for *P. vivax* to invade red blood cells. In many areas outside Africa, infections due to *P. vivax* are more common than those due to *P. falciparum* and

cause substantial morbidity [25]. *P.vivax* is estimated to have been responsible for 13.8 million malaria cases globally in 2015, and accounted for approximately half the total number of malaria cases outside Africa [8]. Most cases of *p.vivax* malaria in the South-East Asia Region 74%, followed by the WHO Eastern Mediterranean Region 11% and the WHO African Region 10%. More than 80% of *p.vivax* malaria cases are estimated to occur in three Countries (Ethiopia, India and Pakistan)[23].

Globally, 3.3 billion people or half of the world's population in 97 countries are at risk of getting infected by malaria disease and 1.2 billion are at high risk. It has been estimated that between 300 and 500 million individuals of all age are infected annually and between 1.3 and 2.5 million dies of malaria every year [24]. The most case and deaths occur in sub-Saharan Africa. In particular, thirty countries in sub-Saharan Africa account for 88% of global malaria deaths. Shockingly, the disease kills an African child every 2 minutes[25]. Malaria affects the health and wealth of nations and individuals. In Africa today, malaria is understood to be both a disease of poverty and a cause of poverty[12].

The Global malaria control strategy is a concerted effort meant to bring about changes in the way malaria problem is addressed [21]. As a result, this strategy stresses the selective use of preventive measures wherever they can lead to sustainable results [10]. Interventions to prevent or reduce the transmission of malaria are currently being used, with a degree of success, in some parts of the world. Some of the methods include the situation of irrigated lands far from residential areas and cities, house spraying with residual insecticides and most recently the use of mosquito treated bed nets. The methods operate by reducing the contact rates (and hence exposure to infection) between the mosquitoes and humans. Other measures that employ the use of anti malarial drugs as a control measure may not be very effective when compared with control a measure that directly affects the dynamics of transmission of the parasite (that is based on the human mosquito interaction). This is because in endemic areas, drug coverage can only be effective if permanent prophylaxis is employed across an entire endemic human population. In most developed countries, where malaria has been eradicated but the mosquito vector is still present, changes in world climate through global warming indicate that these malaria free zones

risk being re-colonized by malaria [13,17].

Given these challenges be it in endemic areas or otherwise, predictive mathematical modeling and computer simulations remain our greatest hope[11] The measures are aimed at halting the deteriorating effects of the malaria situation, minimizing the wasteful use of resources and contributing appropriately to the development of health services, intersect oral cooperation and community participation. The ultimate goal of malaria control will be to prevent mortality and reduce morbidity and social and economic loss through the progressive improvement and strengthening of local and national capacities [6].

Malaria is common in tropical and sub-tropical regions because rain full, warm temperatures and stagnant waters provide an environment ideal for mosquito larvae. Malaria is typically diagnosed by the microscopic examination of blood using blood films, or with antigen-based rapid diagnostic tests. Modern techniques that use the polymerase chain reaction to detect the parasite's DNA have also been developed, but these are not widely used in malaria-endemic areas due to their cost and complexity. Disease transmission can be reduced by preventing mosquito bites by using mosquito nets and insect repellents, or with mosquito-control measures such as spraying insecticides and draining stagnant water.

Mathematical modeling of the dynamic of malaria began in 1911 with Ross Model, and major extensions are described in Macdonald's book [22]. Because of the simplified nature of Macdonald's model, malaria eradication or prevention of his model for total eradications of malaria from the world was not achieved as he failed or did not adequately advertise the long term consequences of the simplifications made to his model. The model for malaria transmission analyzed is an extension of the equations introduced by [14]. In the Ngwa and Shu model, humans follow an SEIR-like pattern and mosquitoes follow a SEI pattern, similar to that described by [26] but with only one immune class for humans.

Humans move from the susceptible to the exposed class at some probability when they come in to contact with an infectious mosquito, and then to the infection class as in conventional SEIRS models. However, infectious people can then recover with, or without,

a gain in immunity; and either return to the susceptible class, or move to the recovered class. A new feature of this model is that although individuals in the recovered class are assumed to be “immune”, in the sense that they do not suffer from serious illness and do not contract clinical malaria, they still have low levels of plasmodium in their blood stream and can pass the infection to susceptible mosquitoes. After some period of time these recovered individuals return to the susceptible class.

Susceptible mosquitoes get infected and move to the exposed class, at some probability when they come into contact with either infectious humans or recovered humans (albeit at a much lower probability). They then pass on to the infectious class. Both humans and mosquitoes leave the population through a density dependent natural death rate. This allows the model to account for changing human and mosquito populations.

Variations in mosquito populations are crucial to the dynamics of malaria and constant population models do not account for this. The model also includes human disease - induced death as mortality for malaria in areas of high transmission can be high, especially in infants.

Ngwa and Shu analyze this model assuming a linear per capita death rate. They convert the system to dimensionless quantities and in these new variables, define a reproductive number. They show that when  $R_0 > 1$ , there exist an endemic equilibrium (non negative solution distinct from the disease - free equilibrium) and furthermore, with no disease-induced death, this endemic equilibrium is unique. Using linear analysis, they also show that the disease - free equilibrium is locally asymptotically stable when  $R_0 \leq 1$  and the unique endemic equilibrium (for no disease - induced death) is locally asymptotically stable when  $R_0 > 1$ . They conclude by using numerical simulations to support their proposition that the endemic equilibrium is stable for  $R_0 > 1$ .

## Chapter 3

# RESEARCH METHEDODOLOGY

We develop a mathematical model which describe the dynamic and control of malaria using a system of non-linear differential equations. We perform the qualitative analysis of the model. Both local and global stability of the equilibrium points of the model equations would be done using next generetion matrix and Lyapunove functions respectively. The analytical solutions of the model equations would be supplemented by numerical simulations by choosing appropriate system parameter using Matlab .

# Chapter 4

## THE BASIC MALARIA MODEL

We formulate our basic malaria model with the population under study being divided into compartments and with assumptions about the nature and time rate of transfer from one compartment to another. We consider the total population size denoted by  $N_h(t)$  and  $N_v(t)$  for the human hosts and female mosquitoes respectively. We would use the SEIS framework to describe a disease with temporary immunity on recovery from infection.

SEIS model indicates that the passage of individual is from the susceptible class,  $S_h$ , to the exposed class,  $E_h$ , then to infective class,  $I_h$ , and finally to the recovery class (in this case the susceptible class).  $S_h(t)$  represents the number of individuals not yet infected with the malaria parasite at time  $t$ , or those susceptible to the disease. Many diseases like malaria have what is termed a latent or exposed phase,  $E_h(t)$ , during which an individual is said to be infected but not infectious.  $I_h(t)$  denotes the number of individuals who have been infected with malaria and are capable of spreading the disease to those in the susceptible category. This is done through infecting the susceptible mosquitoes.

The dynamic transmission of the malaria parasite between and amongst individuals in both species is driven by the mosquito biting habit of the humans. The susceptible human population is increased by recruitment (birth and immigration) at a constant rate  $\Delta$ .

All the recruited individuals are assumed to be naive when they join the community. Infected immigrants are not included because we assume that most people who are sick would not travel. When an infectious female anopheles mosquito bites a susceptible hu-

man, there is some finite probability,  $\beta_{vh}$  that the parasite ( in the form of sporozoites) would be passed on to the human. The parasites then move to the liver where it develops into its next life stage. The infected person would move to the exposed class. After a certain period of time, the parasite (in the form of merozoites) enters the blood stream, usually signaling the clinical onset of malaria. Then the exposed individuals become infectious and progress to infected state at a constant rate  $\varphi$ .

After some time, individuals who have experienced infection may recover with natural immunity and move to the recovered class (in this case the susceptible class). Human leave the population through natural death  $\mu_h$  and the infected humans have an additional disease- related death rate  $\delta_h$ . The disease - induced rate is very small in comparison with the recovery rate.

We divided the mosquito population into two classes:- Susceptible,  $S_v$  and infectious,  $I_v$ . Female anopheles mosquitoes (male anopheles mosquito is not included in the model because only female mosquito bites animals for blood meals) enter the susceptible class through birth at a rate  $\Pi$ . Susceptible mosquitoes become infected by biting infectious human at rate,  $\alpha$  .

The parasites (in the form of gametocytes) enter the mosquito with probability,  $\beta_{hv}$  when the mosquito bites an infectious human, and after some period of time, dependent on the ambient temperature and humidity, the parasite develops into sporozoites and enters the mosquito's salivary glands, and the mosquito progresses to infectious class. We assume that the infective period of the vector ends with its death, and therefore vector does not recover from being infective[8]. Mosquitoes leave the population through natural death. Its per capita death rate is  $\mu_v$  .

The rate of infection of susceptible individual is,  $\lambda_h$  and the rate of infecting a susceptible mosquito is  $\lambda_v$  .

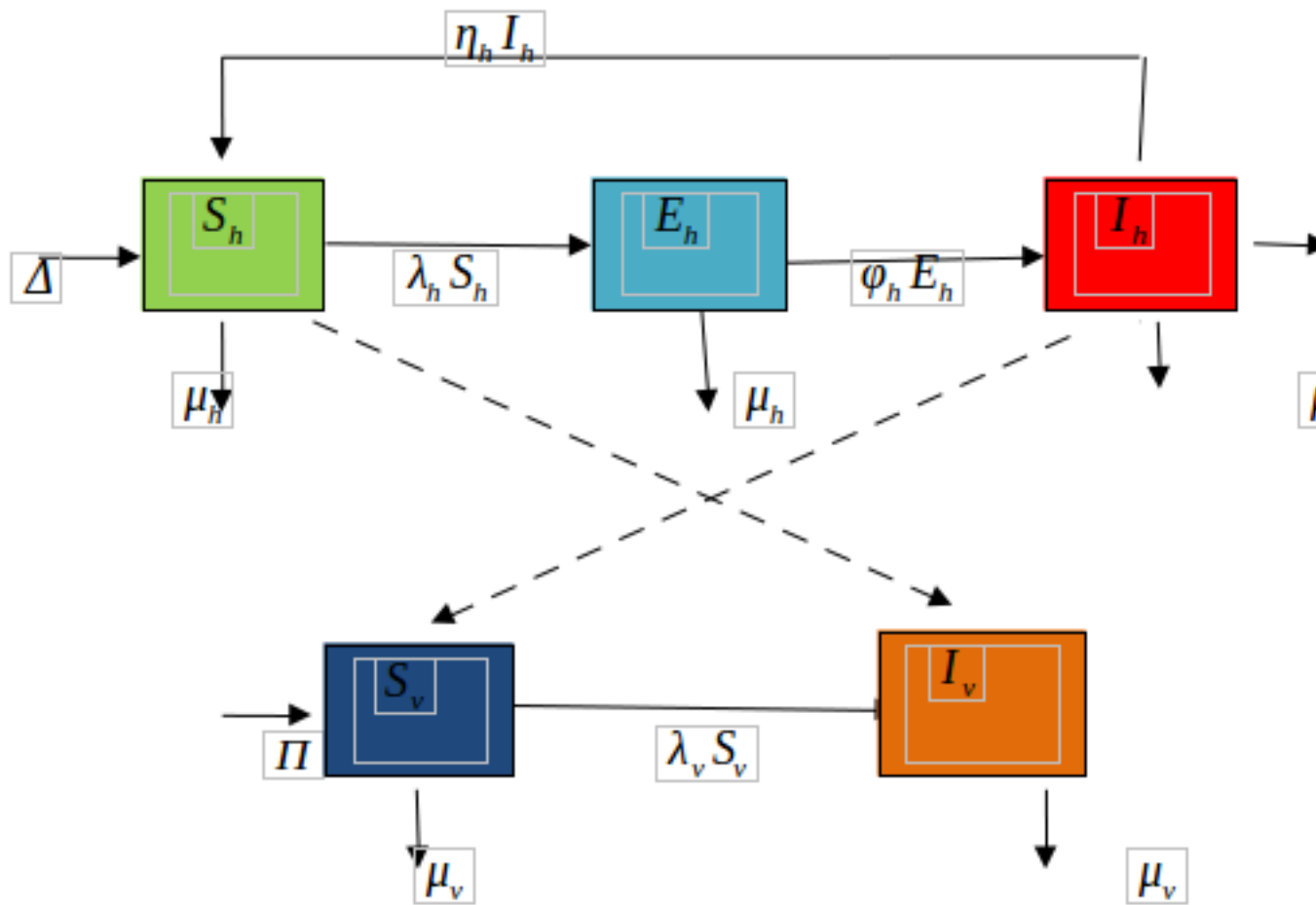


Figure 4.1: The model flow diagram is shown in figure one above

<i>Variables</i>	Descriptions
$N_h(t)$	The total human population at time t
$N_v(t)$	The total mosquitoes population at time t
$S_h(t)$	Number of susceptible humans at time t
$E_h(t)$	Number of exposed humans at time t
$I_h(t)$	Number of infectious humans at time t
$S_v(t)$	Number of susceptible mosquitoes at time t
$I_v(t)$	Number of infectious mosquitoes at time t

Table 1.1 State variables of the basic malaria model

<i>parameters</i>	Description
$\Delta$	Recruitment rate of humans
$\Pi$	Birth rate of mosquitoes
$\mu_h$	per capita natural death rate for humans
$\mu_v$	per capita natural death rate for mosquitoes
$\varphi$	progression rate of humans from the exposed state to the infected state
$\delta_h$	per capita disease-induced death rate for humans
$\lambda_h$	Force of infection for susceptible humans to exposed individuals
$\lambda_v$	Force of infection for susceptible mosquitoes to infected mosquitoes
$\alpha$	Biting rate of mosquitoes
$\beta_{vh}$	probability that a bite results in transmissions of infection to humans
$\beta_{hv}$	probability that a bite results in transmission of the parasite from humans to mosquitoes

Table 1.2 Parameters of the basic malaria model

## 4.1 Model Development

From the above model flowchart together lead to the following systems of ordinary differential equations which describe the progress of the disease.

$$\begin{aligned}
\frac{ds_h}{dt} &= \Delta - \lambda_h S_h - \mu_h S_h \\
\frac{dE_h}{dt} &= \lambda_h S_h - \mu_h E_h - \varphi E_h \\
\frac{dI_h}{dt} &= \varphi E_h - \mu_h I_h - \delta_h I_h \\
\frac{dS_v}{dt} &= \Pi - \lambda_v S_v - \mu_v S_v \\
\frac{dI_v}{dt} &= \lambda_v S_v - \mu_v I_v
\end{aligned}
\tag{4.1}$$

with initial condition  $S_h(0) = S_{ho}$ ,  $E_h(0) = E_{ho}$ ,  $I_h(0) = I_{ho}$ , and  $S_v(0) = S_{vo}$ ,  $I_v(0) = I_{vo}$ . And also, we have used in equation (4.1) that  $\lambda_h = \frac{\beta_{vh}\alpha I_v}{N_h}$  and  $\lambda_v = \frac{\beta_{hv}\alpha I_h}{N_h}$ . The term,  $\lambda_h$ , denotes the rate at which the susceptible humans become infected by infectious female mosquitoes. Similarly, the term,  $\lambda_v$ , denotes the rate at which the susceptible mosquitoes become infected by infectious humans. The rate of infection propagated to susceptible humans by infected mosquitoes is dependent on the total number of humans.

Similarly, the rate of infection propagated to susceptible mosquitoes by infected humans is dependent on the total number of humans [6].

## 4.2 Model Analysis

Now we consider the solutions of the system of non-linear differential equation (4.1).

We understand that the interpretations of these solutions must be biologically meaningful. Hence it is easy to identify that the feasible region of the system (4.1) is  $R^5$ . The five dimensional solution space shows that all the solutions are positive,  $\forall t \geq 0$ . Hence the feasible region containing all the solutions of the system of equations (4.1) is given by the set

$\Omega = (S_h, E_h, I_h, S_v, I_v) \in R^5$ . Here the quantities  $S_h, E_h, I_h, S_v, I_v$  are all non-negative.

The total population size  $N_h$  and  $N_v$  can be determined by  $N_h = S_h + E_h + I_h$  and  $N_v = S_v + I_v$

or from differential equation

$$\begin{aligned} \frac{dN_h}{dt} &= \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_h}{dt} \\ \frac{dN_h}{dt} &= \Delta - \lambda_h S_h - \mu_h S_h + \lambda_h S_h - \mu_h E_h - \varphi E_h + \varphi E_h - \mu_h I_h - \delta_h I_h \\ \frac{dN_h}{dt} &= \Delta - \mu_h S_h - \mu_h E_h - \mu_h I_h - \delta_h I_h \\ \frac{dN_h}{dt} &= \Delta - \mu_h (S_h + E_h + I_h) - \delta_h I_h \\ \frac{dN_h}{dt} &= \Delta - \mu_h N_h - \delta_h I_h \end{aligned} \tag{4.2}$$

We consider the solution of equation (4.2) when the term  $\delta_h I_h$  vanishes. In case if the death rate of humans due to malaria disease is considered to be free, i.e  $\delta_h = 0$  then we obtain

$$\frac{dN_h}{dt} = \Delta - \mu_h N_h \tag{4.3}$$

The solution of this differential equation is found to be  $N_h(t) = \frac{\Delta}{\mu_h} + [N_{h0} - \frac{\Delta}{\mu_h} e^{-\mu_h t}]$

This showing that  $N_h(t) \rightarrow \frac{\Delta}{\mu_h}$  as  $t \rightarrow \infty$

The term  $N_{h0}$  denotes the initial total human population. It can be interpreted that the total human population grows and asymptotically converge to a positive quantity given

by  $\frac{\Delta}{\mu_h}$ .

Under the condition that human do not die due to malaria infection.

Thus  $\frac{\Delta}{\mu_h}$  is an upper bound of the total human populations,  $N_h(t)$  i.e  $N_h(\infty) \leq \frac{\Delta}{\mu_h}$ .

Whenever the initial human population starts off below  $\frac{\Delta}{\mu_h}$  then it grows over time and finally reach the upper asymptotic value  $\frac{\Delta}{\mu_h}$ .

similarly whenever the initial human population starts off high above  $\frac{\Delta}{\mu_h}$  then it decays over time and finally reaches the lower asymptotic value  $\frac{\Delta}{\mu_h}$ .

Similarly the total population size for mosquitoes is  $N_v = S_v + I_v$  with their differential equation.

$$\begin{aligned}
 \frac{dN_v}{dt} &= \frac{dS_v}{dt} + \frac{dI_v}{dt} \\
 \frac{dN_v}{dt} &= \Pi - \lambda_v S_v - \mu_v S_v + \lambda_v S_v - \mu_v I_v \\
 \frac{dN_v}{dt} &= \Pi - \mu_v S_v - \mu_v I_v \\
 \frac{dN_v}{dt} &= \Pi - \mu_v (S_v + I_v) \\
 \frac{dN_v}{dt} &= \Pi - \mu_v N_v
 \end{aligned} \tag{4.4}$$

the solution of this differential equation is found to be

$$N_v(t) = \frac{\Pi}{\mu_v} + [N_{v0} - \frac{\Delta}{\mu_v} e^{-\mu_v t}]$$

This showing that  $N_v(t) \rightarrow \frac{\Pi}{\mu_v}$  as  $t \rightarrow \infty$

The term  $N_{v0}$  denotes the initial mosquito population. It can be interpreted that the total mosquitoes population grows and asymptotically converges to a positive quantity given by  $\frac{\Pi}{\mu_v}$ .

Thus  $\frac{\Pi}{\mu_v}$  is an upper bound of the total mosquito population  $N_v(t)$  i.e  $N_v(\infty) \leq \frac{\Pi}{\mu_v}$ .

Whenever the initial mosquito population starts off low below  $\frac{\Pi}{\mu_v}$ , then it grows over time and finally reaches the upper asymptotic value  $\frac{\Pi}{\mu_v}$ . Similarly, whenever the initial mosquito population starts off high above  $\frac{\Pi}{\mu_v}$  then it decays over time and finally reaches the lower asymptotic value  $\frac{\Pi}{\mu_v}$ .

### 4.2.1 Existence and Positivity of solution

Lemma(1) Let the initial data be  $S_h(0), S_v(0) > 0, (E_h(0), I_h(0), I_v(0)) \geq 0 \in \Omega$

Then the solution set  $S_h, E_h, I_h, S_v, I_v(t)$  of the model system (4.1) is positive for all  $t > 0$

Proof : The first equation model (4.1) gives

$$\frac{dS_h}{dt} = \Delta - \lambda_h S_h - \mu_h S_h \geq -\lambda_h S_h - \mu_h S_h$$

$$\frac{dS_h}{dt} = \Delta - \lambda_h S_h - \mu_h S_h \geq -(\lambda_h + \mu_h) S_h$$

$$\int \frac{1}{S_h} dS_h \geq -\int (\lambda_h + \mu_h) dt$$

$$S_h(t) \geq S_h(0) e^{-(\lambda_h t + \mu_h t)} \geq 0$$

From the second equation of (4.1) we have

$$\frac{dE_h}{dt} = \lambda_h S_h - \mu_h E_h - \varphi E_h$$

$$\frac{dE_h}{dt} = \lambda_h S_h - \mu_h E_h - \varphi E_h \geq -(\mu_h + \varphi) E_h$$

$$\int \frac{1}{E_h} dE_h \geq -\int (\mu_h + \varphi) dt$$

$$E_h(t) \geq E_h(0) e^{-(\mu_h + \varphi)t} \geq 0$$

Also we get the following from third equation of (4.1)

$$\frac{dI_h}{dt} = \varphi E_h - \mu_h I_h - \delta_h I_h$$

$$\frac{dI_h}{dt} = \varphi E_h - \mu_h I_h - \delta_h I_h \geq -\mu_h I_h - \delta_h I_h$$

$$\frac{dI_h}{dt} = \varphi E_h - \mu_h I_h - \delta_h I_h \geq -(\mu_h + \delta_h) I_h$$

$$\int \frac{1}{I_h} dI_h \geq -\int (\mu_h + \delta_h) dt$$

$$I_h(t) \geq I_h(0) e^{-(\mu_h + \delta_h)t} \geq 0$$

It follows also from fourth equation of (4.1)

$$\frac{dS_v}{dt} = \Pi - \lambda_v S_v - \mu_v S_v$$

$$\frac{dS_v}{dt} = \Pi - \lambda_v S_v - \mu_v S_v \geq -\lambda_v S_v - \mu_v S_v$$

$$\frac{dS_v}{dt} = \Pi - \lambda_v S_v - \mu_v S_v \geq -(\lambda_v + \mu_v) S_v$$

$$\int \frac{1}{S_v} dS_v \geq -\int (\lambda_v + \mu_v) dt$$

$$S_v(t) \geq S_v(0) e^{-(\lambda_v t + \mu_v t)} \geq 0$$

Solving for  $I_v(t)$ , we consider the fifth equation of (4.1) which gives

$$\frac{dI_v}{dt} = \lambda_v S_v - \mu_v I_v$$

$$\frac{dI_v}{dt} = \lambda_v S_v - \mu_v I_v \geq -\mu_v I_v$$

$$\int \frac{1}{I_v} dt \geq -\int \mu_v dt$$

$$I_v(t) \geq I_v(0)e^{-(\mu_v t)} \geq 0$$

Furthermore, we need to show that the region  $\Omega$  is positively invariant so that it suffices the dynamic of the above system.

The right hand sides of equation (4.3) and (4.4) are both bounded by

$\Delta - \mu_h N_h$  and  $\Pi - \mu_v N_v$  respectively, it follows that

$$\frac{dN_h}{dt} < 0 \text{ if } N_h(t) > \frac{\Delta}{\mu_h}$$

and

$$\frac{dN_v}{dt} < 0 \text{ if } N_v(t) > \frac{\Pi}{\mu_v}$$

Using a standard comparison theorem (Zhang, 1988) it has been shown above that

$$N_h(t) \leq \frac{\Delta}{\mu_h}(1 - e^{-\mu_h t}) + N_h(0)e^{-\mu_h t}$$

and

$$N_v(t) \leq \frac{\Pi}{\mu_v}(1 - e^{-\mu_v t}) + N_v(0)e^{-\mu_v t}$$

In particular, if  $N_h(0) < \frac{\Delta}{\mu_h}$  then  $N_h(t) \leq \frac{\Delta}{\mu_h}$

and

if  $N_v(0) < \frac{\Pi}{\mu_v}$  then  $N_v(t) \leq \frac{\Pi}{\mu_v}$ .

Therefore,  $\Omega$  is positively invariant.

If  $N_h(0) > \frac{\Delta}{\mu_h}$  and  $N_v(0) > \frac{\Pi}{\mu_v}$ , then either the solution enters  $\Omega$  in finite time or  $N_h(t)$  approaches  $\frac{\Delta}{\mu_h}$  and  $N_v(t)$  approaches  $\frac{\Pi}{\mu_v}$  asymptotically, and the infected state variable  $E_h, I_h$  and  $I_v$  approaches zero.

### 4.2.2 Existence of Disease-free Equilibrium point

Disease-free equilibrium points are steady state solution where there is no malaria in the human population and there is no plasmodium parasite in the mosquito population. That is, absence of malaria causing infections occurs in both populations at the disease free equilibrium point. The disease free equilibrium point is denoted by

$$E_0 = (S_h, E_h, I_h, S_v, I_v)$$

The equilibrium point is obtained on setting the right-hand side of the non-linear system (4.1) to zero. Thus, at the equilibrium the quantities satisfy the condition

$$I_h = E_h = I_v = 0, S_h = \frac{\Delta}{\mu_h}, \text{ and } S_v = \frac{\Pi}{\mu_v}$$

The disease free equilibrium point represents  $E_0$  is the disease free situation in which

there is no malaria infection either in the society or in the environment. Therefore the disease free equilibrium point is given by

$$E_0 = (S_h, E_h, I_h, S_v, I_v) = \left(\frac{\Delta}{\mu_h}, 0, 0, \frac{\Pi}{\mu_v}, 0\right)$$

Which represents the state in which there is no infection in society and is known as the disease-free equilibrium point (DFE).

### 4.2.3 Determination of the Basic reproduction number

Reproduction number, denoted by  $R_0$ , is the threshold or a level for many epidemiological models. It determines whether a disease can attack the populations or not. The threshold quantity  $R_0$  indicates the number of new infected individuals is produced by one infected individual. When,  $R_0 < 1$  each infected individual propagates the infection and produces on average less than one new infected individual so that the disease is expected to die out completely over time. On the other hand if,  $R_0 > 1$  each individual produces more than one new infected individual so we would expect the disease to spread more and grow in the population. This means that the value of the threshold quantity  $R_0$  in order to eradicate the disease must be reduced by less than one. We determine  $R_0$  using the next generation approach. The following steps are followed to compute the basic reproduction number  $R_0$ . The basic reproduction number cannot be determined from the structure of mathematical model alone but depends on the definition of infected and uninfected compartments. Assuming that there are  $n$  compartments of which the first  $m$  compartments to infected individuals.

$$V_i(x) = V_i^-(x) - V_i^+(x)$$

where  $V_i^+(x)$  is the rate of transfer of individual into compartment  $i$  by all other means and  $V_i^-(x)$  is the rate of transfer of individual out of the  $i^{th}$  compartment. It is assumed that each function is continuously differentiable at least twice in each variable. The disease transmission model consists of non-negative initial conditions together with the following equation.

$$\frac{dX_i}{dt} = f_i(x) - V_i(x), i=1,2,3,\dots,n$$

The next is the computation of the square matrices  $F$  and  $V$  of order  $(m \times m)$ . Where  $m$

is the number of infected classes, defined by

$$F = \left[ \frac{\partial f_i}{\partial x_j}(x_0) \right] \text{ and } V = \left[ \frac{\partial v_i}{\partial x_j}(x_0) \right] \text{ with } 1 \leq i, j \leq m$$

such that  $F$  is non-negative,  $V$  is a non-singular matrix and  $x_0$  is the disease free equilibrium point. 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 the next generation matrix for model. Finally the basic reproduction number  $R_0$  is given by  $R_0 = \sigma(FV^{-1})$  where  $\sigma(FV^{-1})$  denotes the spectral radius of matrix  $FV^{-1}$  and the spectral radius is the biggest non-negative eigenvalue of the next generation matrix. Rewriting model equation (4.1) starting with the infected compartments for both populations,  $I_h, E_h, I_v$  and then followed by uninfected classes,  $S_h, S_v$ , also from the two populations, then the model equation becomes

$$\begin{aligned} \frac{dE_h}{dt} &= \frac{\beta_{vh}\alpha I_v S_h}{N_h} - \mu_h E_h - \varphi E_h \\ \frac{dI_h}{dt} &= \varphi E_h - \mu_h I_h - \delta_h I_h \\ \frac{dI_v}{dt} &= \frac{\beta_{hv}\alpha I_h S_v}{N_h} - \mu_v I_v \\ \frac{dS_h}{dt} &= \Delta - \frac{\beta_{vh}\alpha I_v S_h}{N_h} - \mu_h S_h \\ \frac{dS_v}{dt} &= \Pi - \frac{\beta_{hv}\alpha I_h S_v}{N_h} - \mu_v I_v \end{aligned} \quad (4.5)$$

From the system(4.5)  $f_i$  and  $v_i$  are defined as follows respectively

$$f_i = \begin{bmatrix} \frac{\beta_{vh}\alpha I_v S_h}{N_h} \\ 0 \\ \frac{\beta_{hv}\alpha I_h S_v}{N_h} \end{bmatrix} \quad (4.6)$$

and

$$v_i = \begin{bmatrix} (\mu_h + \varphi)E_h \\ (\mu_h + \delta_h)I_h - \varphi E_h \\ \mu_v I_v \end{bmatrix} \quad (4.7)$$

The partial derivatives of (4.6) with respect to  $(E_h, I_h, I_v)$  and the Jacobian matrix of  $f_i$  at the equilibrium point is

$$F = \begin{bmatrix} 0 & 0 & \beta_{vh} \\ 0 & 0 & 0 \\ 0 & \frac{\beta_{hv}\alpha\Pi\mu_h}{\Delta\mu_v} & 0 \end{bmatrix} \quad (4.8)$$

Similarly the partial derivatives of (4.7) with respect to  $(E_h, I_h, I_v)$  and the Jacobian matrix of  $V_i$  is

$$V = \begin{bmatrix} \mu_h + \varphi & 0 & 0 \\ -\varphi & \mu_h + \delta_h & 0 \\ 0 & 0 & \mu_v \end{bmatrix} \quad (4.9)$$

The inverse of matrix V is given by

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu_h + \varphi} & 0 & 0 \\ \frac{\varphi}{(\mu_h + \varphi)(\mu_h + \delta_h + \eta)} & \frac{1}{\mu_h + \delta_h + \eta} & 0 \\ 0 & 0 & \frac{1}{\mu_v} \end{bmatrix} \quad (4.10)$$

Now we have to compute  $FV^{-1}$

$$FV^{-1} = \begin{bmatrix} 0 & 0 & \beta_{vh} \\ 0 & 0 & 0 \\ 0 & \frac{\beta_{hv}\alpha\Pi\mu_h}{\Delta\mu_v} & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\mu_h + \varphi} & 0 & 0 \\ \frac{\varphi}{(\mu_h + \varphi)(\mu_h + \delta_h)} & \frac{1}{\mu_h + \delta_h} & 0 \\ 0 & 0 & \frac{1}{\mu_v} \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} 0 & 0 & \frac{\beta_{vh}\alpha}{\mu_v} \\ 0 & 0 & 0 \\ \frac{\beta_{hv}\alpha\Pi\varphi\mu_h}{\Delta\mu_v(\mu_h + \varphi)(\mu_h + \delta_h)} & \frac{\beta_{hv}\alpha\Pi\varphi\mu_h}{\Delta\mu_v(\mu_h + \delta_h)} & 0 \end{bmatrix} \quad (4.11)$$

Let  $p = \frac{\beta_{vh}\alpha}{\mu_v}$

,

$q = \frac{\beta_{hv}\alpha\Pi\varphi\mu_h}{\Delta\mu_v(\mu_h + \varphi)(\mu_h + \delta_h)}$  and  $r = \frac{\beta_{hv}\alpha\Pi\varphi\mu_h}{\Delta\mu_v(\mu_h + \delta_h)}$

$$FV^{-1} = \begin{bmatrix} 0 & 0 & p \\ 0 & 0 & 0 \\ q & r & 0 \end{bmatrix} \quad (4.12)$$

From (4.12), we can now calculate the eigenvalues to determine the basic reproduction number  $R_0$  by taking the spectral radius of the matrix  $FV^{-1}$ . Thus it is computed by  $|FV^{-1} = \lambda I| = 0$ , where I is the identity matrix of order 3.

$$\text{we have } |FV^{-1} - \lambda I| = \begin{vmatrix} -\lambda & 0 & p \\ 0 & -\lambda & 0 \\ q & r & -\lambda \end{vmatrix} = 0$$

$$-\lambda \left[ \begin{vmatrix} -\lambda & 0 \\ r & -\lambda \end{vmatrix} + p \begin{vmatrix} 0 & -\lambda \\ q & r \end{vmatrix} \right] = 0$$

$$-\lambda(-\lambda^2) + p(\lambda q) = 0$$

$$\lambda^3 - pq\lambda = 0$$

$$\lambda(\lambda^2 - pq) = 0$$

$$\lambda = 0, \text{ or } \lambda^2 - pq = 0$$

$$\lambda = 0, \lambda = \pm\sqrt{pq} \text{ i.e } \lambda_1 = 0, \lambda_2 = \sqrt{pq}, \lambda_3 = -\sqrt{pq}$$

From the three values the dominant eigenvalue of the matrix is

$\lambda_3 = \sqrt{pq}$ . Therefore the basic reproduction number  $R_0$  is equal to,  $R_0$ . Thus

$$R_0 = \sqrt{\frac{\beta_{vh}\beta_{hv}\Pi\varphi\mu_h\alpha^2}{\Delta\mu_v(\mu_h + \varphi)(\mu_h + \delta_h)}} \quad (4.13)$$

Where  $\frac{\varphi}{\mu_h + \varphi}$  is the probability of survival of individuals from latent (exposed) stage into the infectious stage.

The term  $\frac{\beta_{vh}\alpha}{\mu_v}$  describes the number of humans that one mosquito infects (through contact) during the lifetime it survives as infectious, when all humans are susceptible. On the other hand,

the term  $\frac{\beta_{hv}\alpha\varphi}{(\mu_h + \varphi)(\mu_h + \delta_h)}$  is the number of mosquitoes that are infected through contact with one infectious human, while the human survives as infectious, assuming no infection among vectors.

The threshold parameter  $R_0$  can be defined as the square roots of the products of number of humans one mosquito infects during its infectious life time  $R_{0h}$  and the number of mosquito's one human infects during the duration of the infectious period  $R_{0v}$  provided all humans and mosquitoes are susceptible.

Therefore  $R_0 = \sqrt{R_{0h}R_{0v}}$

$$R_0 = \sqrt{\frac{\beta_{vh}\beta_{hv}\Pi\varphi\mu_h\alpha^2}{\Delta\mu_v(\mu_h + \varphi)(\mu_h + \delta_h)}}$$

$$R_0 = \sqrt{\frac{\beta_{vh}\varphi\alpha\mu_h}{\Delta(\mu_h + \varphi)(\mu_h + \delta_h)} \frac{\beta_{hv}\alpha\Pi}{\mu_v}}$$

Thus

$$R_{0h} = \frac{\beta_{vh}\varphi\alpha\mu_h}{\Delta(\mu_h + \varphi)(\mu_h + \delta_h)} \quad (4.14)$$

and

$$R_{0v} = \frac{\beta_{ov}\alpha\Pi}{\mu_v} \quad (4.15)$$

$\frac{\beta_{vh}\alpha\mu_h}{\Delta(\mu_h + \delta_h)}$  is the number of latent infections produced by a typical infectious individual during the mean infectious period.

$\frac{\beta_{hv}\alpha\Pi}{\mu_v\mu_v}$  is the number of latent infections produced by a typical infectious mosquito during the mean infectious period.

Notice that  $\alpha$  appears twice in the expression since the mosquito biting rate controls transmission from humans to mosquitoes and from mosquitoes to humans. Malaria infection exists in community due to contact between the humans and mosquitoes. Whether the disease becomes persistent or dies out depends on the magnitude of the basic reproduction number,  $R_0$ . Stability of the equilibrium points can be analyzed using  $R_0$ .

#### 4.2.4 Local Stability of the disease-free Equilibrium point

The local stability of the disease-free equilibrium can be discussed by examining system (4.1) at the steady state  $E_0$ .

Proposition(1). The disease-free equilibrium point  $E_0$  for the system (4.1) is locally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ .

Proof. The Jacobian matrix of system (4.1) with  $S_h = N_h - (E_h + I_h)$  evaluated at the disease-free equilibrium point is given by

$$\begin{bmatrix} -(\mu_h + \varphi) & 0 & 0 & \beta_{vh}\alpha \\ -\varphi & -(\mu_h + \delta_h) & 0 & 0 \\ 0 & -\frac{\beta_{hv}\alpha\Pi\mu_h}{\Delta\mu_v} & -\mu_v & 0 \\ 0 & \frac{\beta_{hv}\alpha\Pi\mu_h}{\Delta\mu_v} & 0 & -\mu_v \end{bmatrix} \quad (4.16)$$

The third columns have diagonal entries. Therefore, the diagonal entries is  $-\mu_v$  which is one of the eigenvalue of the Jacobian matrix . Thus, excluding these columns and the

corresponding rows, we can form a (3x3) matrix shown below.

$$\begin{bmatrix} -(\mu_h + \varphi) & 0 & \beta_{vh}\alpha \\ -\varphi & -(\mu_h + \delta_h) & 0 \\ 0 & -\frac{\beta_{hv}\alpha\Pi\mu_h}{\Delta\mu_v} & -\mu_v \end{bmatrix} \quad (4.17)$$

Thus from this resulted matrix we can calculate the remaining eigenvalues. These eigenvalues are the solutions of the characteristic equations of the reduced matrix is given by .

$$\det(A - xI) = 0$$

$$\begin{vmatrix} [x + (\mu_h + \varphi)] & 0 & \beta_{vh}\alpha \\ \varphi & [x + (\mu_h + \delta_h)] & 0 \\ 0 & -\frac{\beta_{hv}\alpha\Pi\mu_h}{\Delta\mu_v} & x + \mu_v \end{vmatrix} = 0$$

Thus

$$(x + \mu_h + \varphi)(x + \mu_h + \delta_h)(x + \mu_v) - \frac{\beta_{vh}\beta_{hv}\alpha^2\Pi\varphi\mu_h}{\Delta\mu_v} = 0 \quad (4.18)$$

To simplify the notation, we let

$$B_0 = \mu_h + \varphi, B_1 = \mu_h + \delta_h, \text{ and } B_2 = \mu_v$$

This reduces (4.13) to

$$R_0^2 = \frac{\beta_{vh}\beta_{hv}\alpha^2\varphi\Pi\mu_h}{\Delta\mu_v B_0 B_1 B_2} \text{ and (4.18) to}$$

$$(x + B_0)(x + B_1)(x + B_2) - \frac{\beta_{vh}\beta_{hv}\alpha^2\varphi\Pi\mu_h}{\Delta\mu_v} = 0$$

$$x^3 + (B_0 + B_1 + B_2)x^2 + (B_0B_1 + B_1B_2 + B_0B_2)x + B_0B_1B_2 - \frac{\beta_{vh}\beta_{hv}\alpha^2\varphi\Pi\mu_h}{\Delta\mu_v} = 0$$

$$x^3 + A_1x^2 + A_2x + A_3 = 0 \quad (4.19)$$

where  $A_1 = B_0 + B_1 + B_2$ ,  $A_2 = B_0B_1 + B_1B_2 + B_0B_2$  and  $A_3 = B_0B_1B_2 - \frac{\beta_{vh}\beta_{hv}\alpha^2\varphi\Pi\mu_h}{\Delta\mu_v}$

The Routh- Hurwitz condition , which usually have different forms are sufficient and necessary conditions on the coefficients of the polynomial(4.19). These conditions ensure that all roots of the polynomial given by (4.19) have negative real parts. For this polynomial, the Routh-Hurwitz conditions are

$A_2 > 0$ ,  $A_3 > 0$  and  $H_1 = A_1 > 0$

where the corresponding Routh-Hurwitz matrix

$$H_1 = A_1,$$

$$H_2 = \begin{bmatrix} A_1 & 1 \\ 0 & A_2 \end{bmatrix}$$

and

$$H_3 = \begin{bmatrix} A_1 & 1 & 0 \\ A_3 & A_2 & A_1 \\ 0 & 0 & A_3 \end{bmatrix}$$

with their corresponding determinants

$$\det(H_1) = A_1, \det(H_2) = \begin{vmatrix} A_1 & 1 \\ 0 & A_2 \end{vmatrix} > 0 \text{ and } \det(H_3) = \begin{vmatrix} A_1 & 1 & 0 \\ A_3 & A_2 & A_1 \\ 0 & 0 & A_3 \end{vmatrix} > 0$$

since  $B_0 > 0$ ,  $B_1 > 0$ , and  $B_2 > 0$  we have  $A_i > 0, i = 1, 2, 3$ .

Moreover, if  $R_0 < 0$ , it follows that  $A_3 > 0$

Thus it is enough to prove that  $H_2 > 0$ , it means  $A_1 A_2 > 0$  and  $H_3 > 0$  clearly  $H_2 = A_1 A_2$  and  $H_3 = A_3(A_1 A_3)$

Now  $H_2 = A_1 A_2$

$$H_2 = (B_0 + B_1 + B_2)(B_0 B_1 + B_1 B_2 + B_0 B_2)$$

$$H_2 = B_0 B_0 B_1 + B_0 B_1 B_2 + B_0 B_0 B_2 + B_0 B_1 B_1 + B_1 B_1 B_2 + B_0 B_1 B_2 + B_0 B_1 B_2 + B_1 B_2 B_2 + B_0 B_2 B_2$$

$$H_2 = B_0 B_0 B_1 + 3B_0 B_1 B_2 + B_0 B_0 B_2 + B_0 B_1 B_1 + B_1 B_1 B_2 + B_1 B_1 B_2 + B_1 B_2 B_2 + B_0 B_2 B_2$$

$$H_2 = B_0 B_1 (B_0 + B_1 + 3B_2) + B_0 B_2 (B_0 + B_2) + B_1 B_2 (B_1 + B_2)$$

which is positive.

Again to show  $H_3 > 0$ , since  $A_1 > 0$ ,  $A_3 > 0$  and also  $A_1 A_2 > 0$  it is enough to prove  $A_1 A_2 > A_3$  by Routh-Hurwitz conditions.

We know that  $A_3 = B_0 B_1 B_2 = \frac{\beta_{vh} \beta_{hv} \alpha^2 \varphi \Pi \mu_h}{\Delta \mu_h}$  which is greater than zero but

$$B_0 B_1 B_2 - \frac{\beta_{vh} \beta_{hv} \alpha^2 \varphi \Pi \mu_h}{\Delta \mu_v} > 0 \text{ if and only if } B_0 B_1 B_2 > \frac{\beta_{vh} \beta_{hv} \alpha^2 \varphi \Pi \mu_h}{\Delta \mu_v} \text{ But}$$

$H_2 = B_0 B_0 B_1 + B_0 B_1 B_2 + B_0 B_0 B_2 + B_0 B_1 B_1 + B_1 B_1 B_2 + B_0 B_1 B_2 + B_0 B_1 B_2 + B_1 B_2 B_2 + B_0 B_2 B_2$  Which is the sum of positive terms in addition to  $3B_0 B_1 B_2$  this implies that

$A_1 A_2 > A_3$  Therefore, all of the eigenvalues of the Jacobian matrix have negative real parts when  $R_0 < 1$  This concludes that the disease-free equilibrium point is stable. However,  $R_0 > 1$ , implies,  $A_3 < 0$ , ( $A_1$  and  $A_2$ ) and since all the coefficients of the polynomial (4.19) are positive, not all roots of this polynomial can have negative real parts. This means, when  $R_0 > 1$ , the disease-free equilibrium point is unstable. Note that the result in proposition (1) is local, that is, we can conclude that solutions with fairly small initial size in the invariant set  $\Omega$  are attracted to the disease-free equilibrium point.

### 4.2.5 Existence and Local Stability of the Endemic Equilibrium point

The Endemic equilibrium point  $E_1$ , is a steady - state solution where the disease persists in the population. For the existence and uniqueness of equilibrium

$$E_1 = (S_h^*, E_h^*, I_h^*, S_v^*, I_v^*)$$

Its coordinates should satisfy the conditions

$$S_h^* > 0, E_h^* > 0, I_h^* > 0, S_v^* > 0 \text{ and } I_v^* > 0$$

Now we need to solve the basic malaria model (4.1) by equating to zero, at an arbitrary equilibrium

$$E_1 = (S_h^*, E_h^*, I_h^*, S_v^*, I_v^*)$$

Solving the second equation of (4.1) for

$$E_h^* = \frac{\beta_{vh} \alpha I_v^* S_h^*}{N_h (\mu_h + \varphi)} \quad (4.20)$$

Again from fourth and fifth equation of (4.1) we solve for  $S_v^*$  and  $I_v^*$  respectively to get the following.

$$S_v^* = \frac{\Pi N_h}{\beta_{hv} \alpha I_h^* + N_h \mu_v} \quad (4.21)$$

$$I_v^* = \frac{\beta_{hv}\alpha I_h^* S_v^*}{N_h \mu_v} \quad (4.22)$$

Now substituting (4.21) into (4.22) for  $S_v^*$  gives

$$I_v^* = \frac{\beta_{hv}\alpha \Pi}{\mu_v(\beta_{hv}\alpha I_h^* + N_h \mu)} I_h^* \quad (4.23)$$

From equation (4.15) we have

$$R_{0v} = \frac{\beta_{hv}\alpha \Pi}{\mu_v}$$

Therefore

$$I_v^* = \frac{R_{0v}}{(\beta_{hv}\alpha I_h^* + N_h \mu_v)} I_h^*$$

$$I_v^* = \frac{R_{0v}}{(\beta_{hv}\alpha I_h^* + N_h \mu_v)} I_h^* \quad (4.24)$$

Furthermore , from the second equation of (4.1)

$$\frac{\beta_{vh}\alpha I_v^* S_h^*}{N_h} - (\mu_h + \varphi) E_h^* = 0 \quad (4.25)$$

we can substitute(4.24) into (4.25) to get

$$\frac{\beta_{vh}\alpha R_{0v} I_h^* S_h^*}{N_h(\beta_{hv}\alpha I_h^* + N_h \mu_v)} - (\mu_h + \varphi) E_h^* = 0 \quad (4.26)$$

But from the third equation of(4.1) we can get

$$E_h^* = \frac{(\mu_h + \delta_h)}{\varphi} I_h^* \quad (4.27)$$

and substitute (4.27) into (4.26) for  $E_h^*$  to get

$$\left( \frac{\beta_{vh}\alpha R_{ov} S_h^*}{N_h(\beta_{hv}\alpha I_h^* + N_h\mu_v)} - \frac{(\mu_h + \varphi)(\mu_h + \delta_h)}{\varphi} \right) I_h^* = 0 \quad (4.28)$$

clearly one can see that  $I_h^* = 0$  or

$$\frac{\beta_{vh}\alpha R_{ov} S_h^*}{N_h(\beta_{hv}\alpha I_h^* + N_h\mu_v)} - \frac{(\mu_h + \varphi)(\mu_h + \delta_h)}{\varphi} = 0$$

$$\beta_{vh}\alpha R_{ov}\varphi S_h^* - N_h(\mu_h + \varphi)(\mu_h + \delta_h)(\beta_{hv}\alpha I_h^* + N_h\mu_v) = 0$$

Now using the relation

$$N_h \leq \frac{\Delta}{\mu_h} \text{ and } R_0^2 = R_{0v}R_{0h}$$

we have the following equation

$$\frac{\beta_{vh}\varphi\alpha R_{ov} S_h^*}{N_h(\mu_h + \varphi)(\mu_h + \delta_h)} - (\beta_{hv}\alpha I_h^* + N_h\mu_v) = 0$$

$$\left( \frac{\beta_{vh}\alpha\varphi\mu_h}{\Delta(\mu_h + \varphi)(\mu_h + \delta_h)} \right) R_{0v}\mu_v S_h^* - (\beta_{hv}\alpha I_h^* + \mu_v \frac{\Delta}{\mu_h}) = 0 \quad (4.29)$$

from equation (4.14) we know that

$$R_{0h} = \frac{\beta_{vh}\alpha\varphi\mu_h}{\Delta(\mu_h + \varphi)(\mu_h + \delta_h + \eta)}$$

$$R_{0h}R_{0h}\mu_v S_h^* - \beta_{hv}\alpha I_h^* - \mu_v \frac{\Delta}{\mu_h} = 0$$

$$R_0^2\mu_v S_h^* - \beta_{hv}\alpha I_h^* - \mu_v \frac{\Delta}{\mu_h} = 0$$

From this we can solve  $S_h^*$  which gives

$$S_h^* = \frac{\beta_{hv}\alpha\mu_h I_h^* + \mu_v \Delta}{R_0^2 \mu_v \mu_h} \quad (4.30)$$

From the first equation of (4.1) we have

$$\Delta - \frac{\beta_{vh}\alpha I_v^* S_h^*}{N_h} - \mu_h S_h^* = 0 \quad (4.31)$$

Substitute equation (4.24) into (4.31) for

$I_v^*$  which gives

$$\Delta - \frac{\beta_{vh}\alpha\mu_v R_{0v} I_h^* S_h^*}{N_h(\beta_{hv}\alpha I_h^* + \mu_v N_h)} - \mu_h S_h^* = 0$$

$$\Delta N_h(\beta_{hv}\alpha I_h^* + \mu_v N_h) - \beta_{vh}\alpha\mu_v R_{0v} I_h^* S_h^* + N_h(\beta_{hv}\alpha I_h^* + \mu_v N_h) I_h^* - N_h \mu_h (\beta_{hv}\alpha I_h^* + \mu_v N_h) S_h^* = 0 \quad (4.32)$$

Now substituting equation (4.30) into (4.32) for  $S_h^*$  and with some algebraic manipulation, we get the following.

$$p(I_h^*)^2 + q(I_h^*) + r = 0 \quad (4.33)$$

where

$$p = N_h \beta_{hv} \alpha - \frac{\beta_{vh} \beta_{hv} R_{0v} \alpha^2 \mu_h}{R_0^2}$$

$$q = \Delta \beta_{hv} \alpha N_h + N_h^2 \mu_v - \frac{\beta_{vh} R_{0v} \alpha \mu_h \Delta \mu_v}{R_0^2} - T$$

where

$$T = \beta_{hv} \alpha \mu_h N_h$$

$$r = \Delta N_h^2 \mu_v \mu_h (1 - R_0^2)$$

$$I_h^* = \frac{\sqrt{q^2 - 4pr}}{2p} - q$$

$$I_h^* = k \quad (4.34)$$

where  $k = \frac{\sqrt{q^* - 4pr}}{2p} - q$

Now we can solve equation(4.30) using equation (4.34)

$$S_h^* = \frac{\beta_{hv}\alpha\mu_h k + \mu_v \Delta}{R_0^2 \mu_v \mu_h} \quad (4.35)$$

From the third equation of (4.1), and the substitution of(4.25) we get

$$E_h^* = \frac{(\mu_h + \delta_h)k}{\varphi} \quad (4.36)$$

In addition we are required to solve for  $S_v^*$  and  $I_v^*$  for the susceptible and infected mosquitoes in malaria endemic area using (4.36) and  $N_v \leq \frac{\Pi}{N_v}$ , we get

$$S_v^* = \frac{\Pi \Delta}{\beta_{hv}\alpha k + \Delta \mu_v} \quad (4.37)$$

$$I_v^* = \frac{R_{ov}k}{(\beta_{hv}\alpha\mu_v\mu_h k + \Delta\mu_v)} \quad (4.38)$$

For  $p > 0$  and  $q > 0$ , when  $R_0 < 1$  one can see that  $r > 0$ . Then the basic malaria model (4.1) has no positive solution. However, when  $R_0 > 1$  then  $r < 0$  and endemic equilibrium exists. This completes the proof.

### 4.2.6 Global Stability of the Disease- free equilibrium point

Let  $N_v(t) = S_v(t) + I_v(t)$ , then we have

$$\frac{dN_v(t)}{dt} = \frac{dS_v(t)}{dt} + \frac{dI_v(t)}{dt}$$

$$\frac{dN_v(t)}{dt} = \Pi - \mu_v N_v(t)$$

Therefore,

$$N_v(t) \longrightarrow \frac{\Pi}{\mu_v} \text{ as } t \longrightarrow \infty$$

Thus, in model (4.1) we can represent

$S_v(t)$  by  $(\frac{\Pi}{\mu_v} - I_v)$  and system (4.1) can be reduced to the following form with four equations

$$\begin{aligned} \frac{dS_h}{dt} &= \Delta - \lambda_h S_h - \mu_h S_h \\ \frac{dE_h}{dt} &= \lambda_h S_h - (\mu_h + \varphi) E_h \\ \frac{dI_h}{dt} &= \varphi E_h - (\mu_h + \delta_h) I_h \\ \frac{dI_v}{dt} &= \beta_{hv} \alpha \left( \frac{\Pi}{\mu_v} - I_v \right) \frac{I_h}{N_h} - \mu_v I_v \end{aligned} \tag{4.39}$$

In this subsection, we study the global stability of the disease-free equilibrium proposition (2)

If  $R_0^2 < p$ , then the disease-free equilibrium-point  $E_0$  of system (4.39) is globally asymptotically stable

$$\text{where } p = 1 - \frac{\beta_{vh} \beta_{hv} \Pi \alpha^2 \varphi \delta_h}{\mu_v^2 \Delta (\mu_h + \varphi) (\mu_h + \delta_h)}$$

proof

By proposition(1),  $E_0$  is locally stable when  $R_0 < 1$

$$\text{Let } R_1 = \frac{\beta_{vh} \beta_{hv} \alpha^2 \varphi \frac{\Pi}{\mu_v}}{\mu_v \frac{\Delta}{\mu_h + \delta_h} (\mu_h + \varphi) (\mu_h + \delta_h)}$$

we notice that  $R_1 = R_0^2 + 1 - p$ , thus  $R_1 < 1$

is equivalent to  $R_0^2 < p$ .

If  $R_1 < 1$ , then there is a sufficiently small constant  $\varepsilon > 0$

such that

$$\frac{\beta_{vh}\beta_{hv}\alpha^2\varphi(\frac{\Pi}{\mu_v}+\varepsilon)}{\mu_v(\frac{\Delta}{\mu_h+\delta_h}-\varepsilon)(\mu_h+\varphi)(\mu_h+\delta_h)} < 1$$

Let  $(S_h, E_h, I_h, I_v)$  be any positive solution of system (4.39), then there is a  $T_1$  such that

$$N_v(t) \leq \frac{\Pi}{\mu_v} + \varepsilon \text{ for all } t > T_1$$

since

$$\frac{dN_h(t)}{dt} = \Delta - \mu_h N_h(t) - \delta_h I_h(t)$$

$$\frac{dN_h}{dt} = \Delta - \mu_h N_h(t) - \delta_h I_h(t)$$

$$\frac{dN_h}{dt} \geq \Delta - (\mu_h + \delta_h)N_h(t)$$

We conclude that there is a  $T_2 > T_1$  such that  $N_h(t) \geq \frac{\Delta}{\mu_h+\delta_h} - \varepsilon$  for all  $t \geq T_2$

Consider the following Lyapunov function

$$V(I_h, I_v) = I_v + \frac{\mu_v(\mu_h+\varphi)}{\beta_{vh}\alpha\varphi} I_h$$

Now computing the derivative of  $V(I_h, I_v)$  along the solutions of system (4.39), we have

$$\frac{dV}{dt} = \frac{dI_v}{dt} + \frac{\mu_v(\mu_h+\varphi)}{\beta_{vh}\alpha\varphi} \frac{dI_h}{dt}$$

$$\frac{dV}{dt} = \beta_{hv}\alpha\left(\frac{\Pi}{\mu_v}\right)\frac{I_h}{N_h} - \mu_v I_v + \frac{\mu_v(\mu_h+\varphi)}{\beta_{vh}\alpha\varphi[\varphi E_h - (\mu_h+\delta_h)I_h]}$$

But

$$E_h = \frac{\beta_{vh}\alpha I_v S_h}{N_h(\mu_h+\varphi)}$$

$$\frac{dV}{dt} = \beta_{hv}\alpha\left(\frac{\Pi}{\mu_v} - I_v\right)\frac{I_h}{N_h} - \mu_v I_v + \frac{\mu_v(\mu_h+\varphi)}{\beta_{vh}\alpha\varphi}\left[\varphi\frac{\beta_{vh}\alpha I_v S_h}{N_h(\mu_h+\varphi)} - (\mu_h + \delta_h)I_h\right]$$

$$\frac{dV}{dt} = \beta_{hv}\alpha S_v \frac{I_h}{N_h} - \mu_v I_v + \frac{\mu_v I_v S_v}{N_h} - \frac{\mu_v(\mu_h+\varphi)}{\beta_{vh}\alpha\varphi}(\mu_h + \delta_h)I_h$$

$$\frac{dV}{dt} = \beta_{hv}\alpha \frac{S_v}{N_h} I_h - \frac{\mu_v(\mu_h+\varphi)}{\beta_{vh}\alpha\varphi}(\mu_h + \delta_h)I_h - \mu_v I_v + \frac{\mu_v I_v S_v}{N_h}$$

$$\frac{dV}{dt} = [\beta_{hv}\alpha \frac{S_v}{N_h} - \frac{\mu_v(\mu_h+\varphi)}{\beta_{vh}\alpha\varphi}(\mu_h + \delta_h)]I_h - \mu_v(1 - \frac{S_h}{N_h})I_v$$

$$\frac{dV}{dt} \leq [\beta_{hv}\alpha \frac{S_v}{N_h} - \frac{\mu_v(\mu_h+\varphi)}{\beta_{vh}\alpha\varphi}(\mu_h + \delta_h)]I_h$$

$$\frac{dV}{dt} \leq [\beta_{hv}\alpha \frac{\frac{\Pi}{\mu_v} + \varepsilon}{\frac{\Delta}{\mu_h+\delta_h} - \varepsilon} - \frac{\mu_v(\mu_h + \varphi)}{\beta_{vh}\alpha\varphi}(\mu_h + \delta_h)]I_h \quad (4.40)$$

for all  $t \geq T_2$

Obviously, we have

$$\frac{dV}{dt}(I_h, I_v) \leq 0 \text{ for all } I_h \geq 0 \text{ and } I_v \geq 0$$

let  $(S_h, E_h, I, I_v); \frac{dV}{dt}(I_h, I_v) = 0$ , then

$$M \subset S_h, E_h, I_h, I_v : I_h = 0$$

Let  $N \subset M$  be the largest invariant set with respect to system (4.39) and let

$(S_h, E_h, I_h, I_v)$  is defined and bounded on  $t \in R$ .

Since  $N \subset (S_h, E_h, I_h, I_v) : I_h = 0$ , we have  $I_h(t) = 0$

From equation (2) and (4) of (4.39) we obtain

$$\frac{dE_h}{dt} = -(\mu_h + \varphi)E_h$$

$$\frac{dI_v}{dt} = -\mu_v I_v$$

By solving these two equations, it is clearly equal to  $E_h(t) = 0$  and  $I_v(t) = 0$ . Furthermore,

from first equation of (4.39) we have  $\frac{dS_h}{dt} = \Delta - \mu_h S_h(t)$

Hence  $S_h(t) = \frac{\Delta}{\mu_h}$ . Thus, we obtain  $E_0 = (S_h, E_h, I_h, I_v)$

This shows that  $N = E_0$ . By Lasalle's invariance principle,  $E_0$  is globally attractive. Therefore, the disease-free equilibrium  $E_0$  is globally asymptotically stable when  $R_0^2 < p$ .

# Chapter 5

## NUMERICAL SIMULATION

The main aim is to verify some of the analytical results on the stability of system (4.1). The parameter values were obtained from literature and estimation. The figures are plotted using the parameter values in table (3). The rates are given per day.

Table 3 Model parameters and values in simulation

<i>Symbol</i>	Value
$\Delta$	0.000053
$\Pi$	0.073
$\mu_h$	0.00004
$\mu_v$	0.06
$\varphi$	0.073
$\delta_h$	0.00000025
$\alpha$	0.5
$\beta_{vh}$	0.0655
$\beta_{hv}$	0.445

After substituting the above values in table into the malaria model(4.1) we have the following system of non-linear differential equations

$$\frac{dS_h}{dt} = 0.000053 + 0.145I_h - 0.0325\frac{I_v S_h}{N_h} - 0.0000041S_h$$

$$\frac{dE_h}{dt} = 0.0325\frac{I_v S_h}{N_h} - 0.0000041E_h - 0.073E_h$$

$$\frac{dI_h}{dt} = 0.073E_h - (0.1450066)I_h$$

$$\frac{dS_v}{dt} = 0.073 - 0.22\frac{I_h S_v}{N_h} - 0.06S_v$$

$$\frac{dI_v}{dt} - 0.22\frac{I_h S_v}{N_h} = 0.06I_v \text{ with initial condition } S_{ho} = 400, E_{ho} = 280, I_{ho} = 200, S_{vo} = 2000$$

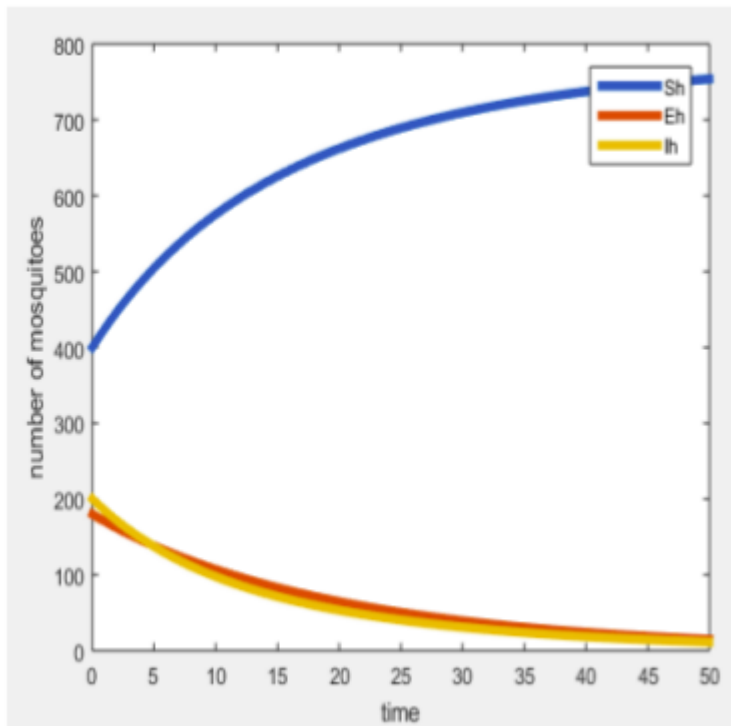


Figure 5.1: plot of the two state variables for mosquitoes against time

and  $I_{vo} = 700$

The reproduction number is less than one since less people from malaria and substituting the parameter values in the reproduction number gives;  $R_0 = 0.0277 < 1$ , hence malaria can be eliminated.

Our malaria model (4.1) was used for the spread dynamics of malaria. The compartment  $S_h$  contains individuals who were susceptible to malaria, compartment  $E_h$  contains individuals who were exposed to malaria and compartment  $I_h$ , contains individuals who were infectious to malaria. The computer software was used to solve the above ordinary differential equations known as Matlab which generated the graphs shown below.

The above figure is illustrating that the changes in the three state variables of malaria model showing the dynamics with time of susceptible humans, exposed humans and infectious humans.

The susceptible population increases and remained constant within a certain time interval. Exposed population initially was high but as the day progresses it reduces as some of the population enters infectious class and the other recover.

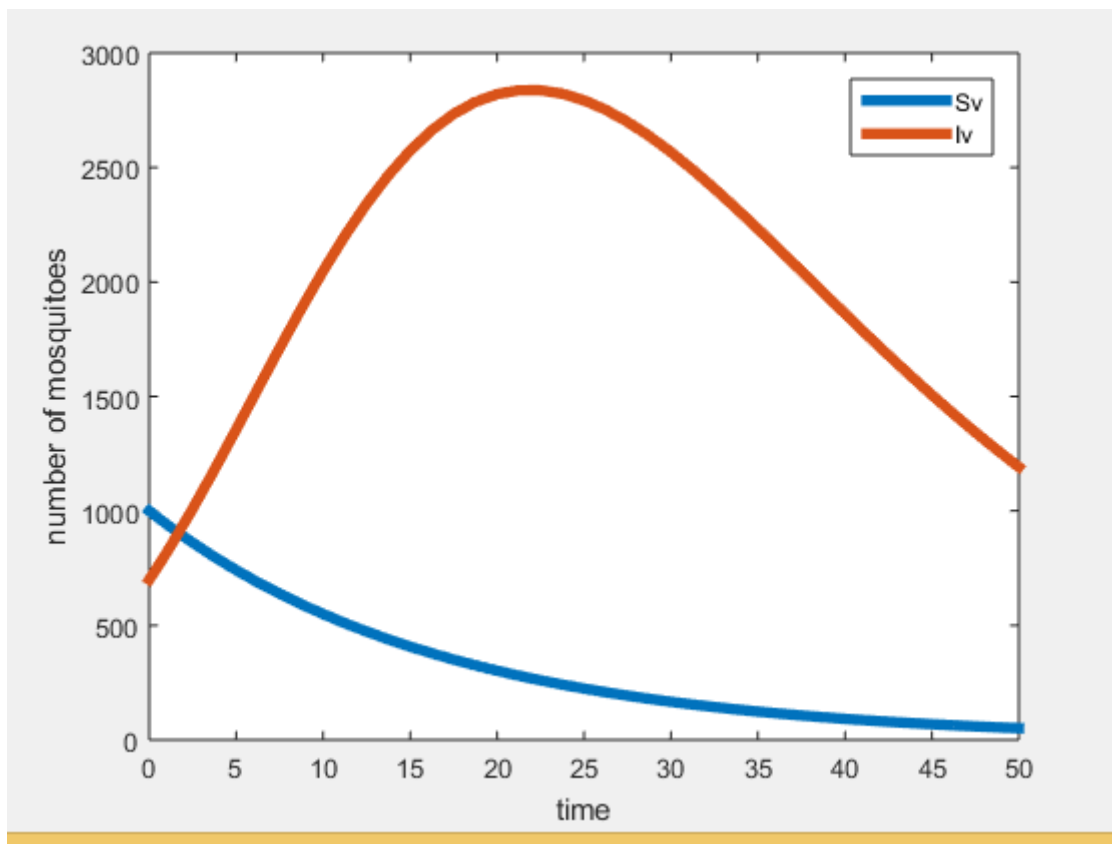


Figure 5.2: plot of the two state variables for mosquitoes against time

similarly the infectious populations reduced as some of them recovered and others die. Similarly as seen in fig. (2) the number of susceptible mosquito decreases with time since there is no recovered class for mosquito population.

In the some manner the number of infectious mosquito population increases due to increase in resistance to the malaria parasite.

## 5.1 Result and Discussion

The basic deterministic model of the transmission dynamics of malaria with a varying total human population that incorporated recruitment of new individuals in the susceptible class through recruitment has been formulated. Analysis of the model has showed that there exists a domain where there is epidemiologically meaningful and mathematically well-posed. The model has been qualitatively analyzed for the existence and stability of the disease-free equilibrium point and endemic equilibrium points. Hereafter the next generation method has been used to calculate the reproduction number,  $R_0$ , as an important parameter that plays a great role in the control of the malaria infection.

The stability of the equilibrium points has been analyzed using  $R_0$ . We have proved that the disease-free equilibrium  $E_0$  is locally asymptotically stable if  $R_0 < 1$  and globally asymptotically stable if  $R_0 > 1$  and also when  $R_0 > 1$  the endemic equilibrium  $E^*$  is appeared

## 5.2 Conclusion

We presented and analyzed a mathematical model to understand the transmission dynamics of malaria. The model considered a changing total population that incorporated recruitment of new individuals into the susceptible compartment through either birth or immigration. Mathematically, we modeled malaria as a 5-dimensional system of ordinary differential equations. We defined the basic reproduction number,  $R_0$ , which provides the expected number of new infections from one infectious individual over the duration of the infectious period given that all other members of the population are susceptible.

We proved that if  $R_O < 1$ , the disease cannot persist and when  $R_O > 1$ , the disease can persist. We have proved that the disease-free equilibrium point is locally asymptotically stable when  $R_O < 1$  and unstable when  $R_O > 1$ . Similarly we proved that if  $R_O > 1$  the disease-free equilibrium is globally asymptotically stable.

In view of the above, humans need to boost their antibodies production to be able to subdue the invasion of parasites in the bloodstream.

The immunity state of the individual, that is, the general health and nutritional status of the infected individual, is a factor for preventing or aiding the occurrence of malaria.

Thus, leading a healthy lifestyle and eating right foods can help boost the level of antibodies in humans. It is also important to note that reducing human-mosquito contact rate plays a big role in inhibiting the prevalence of malaria.

Hence, we can achieve a malaria-free state by scaling down mosquito biting rate through; insecticides, the use of insecticide-treated bed nets closing of doors and windows against mosquitoes, clearing of stagnant water and drainages ,and the use of mosquito repellent lotions, which are all regarded as vector control measures.

### 5.3 Recommendation

Malaria eradication remains a big challenge to National malaria control programme in most developing Countries thus there is need to strengthen the control strategies so that the spread dynamics is reduced. Hence from the results of this work it recommended that:-

- people who are ill should quickly seek treatment at health centers for effective diagnosis and treatment of the infected in order to avoid the spread of malaria within the population .
- Research institutions should start researching into genetically modified mosquitoes that would not be able to transmit malaria. When the probabilities of transmission of infection from an infectious humans to susceptible mosquito and vice versa are made permanently to zero then we can have genetically modified mosquitoes existing without malaria infection in human population.

- Vector control interventions such as insecticide-treated nets(ITNS) and indoor residual spraying (IRS) are proving effective to combat and prevent the disease.ITNS and IRS, with insecticidal and diversionary properties, would reduce the availability of hosts, and kill mosquitoes that are attempting to feed on human's blood, and reducing malaria transmission.
- Because of the complications of measuring malaria at different transmission levels with different immunological status prevalent in different age and gender groups, and across different locations, some guidelines should be developed to give researchers and health professionals a more accurate foundation on which to select their indicators.

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