Analyzing the Impact of Temperature and Rainfall on Malaria Transmission Dynamics: A Mathematical Modeling Approach

A Thesis Submitted to the Department of Mathematics, Jimma University in Partial Fulfillment for the Requirements of the Degree of Masters of Science (M.Sc.) in Mathematics

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Jimma, Ethiopia

Declaration

Here, I submit a thesis entitled "**Analyzing the Impact of Temperature and Rainfall on Malaria Transmission Dynamics: A Mathematical Modeling Approach."** for the award of degree of Master of Science in Mathematics. I, the undersigned declare that, this study is original and it has not been submitted to any institution elsewhere for the award of any academic degree or the like, where other sources of information have been used, they have been acknowledged.

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List of acronyms

DFEP - Disease Free Equilibrium point

EEP - Endemic Equilibrium point

ODE - Ordinary Differential Equation

SI - Susceptible - Infected

SIR - Susceptible - Infected - Recovered

WHO - World Health Organization

Abstract

This thesis explored how climate, particularly rainfall and temperature, affects malaria transmission. We used a compartmental mathematical model to analyze the relationship between these factors and disease progression. The study achieved several key results. Mathematically, we proved the model's solution is unique and has well-defined properties. It also determined the model's equilibrium points and a crucial parameter called the basic reproduction number (R_0). This number indicates how contagious the disease is. The stability of the model was investigated for both disease-free and endemic states (presence or absence of malaria). Stability analysis methods like Routh-Hurwitz criteria and Lyapunov theorems were employed. Additionally, sensitivity analysis identified parameters that significantly impact R_0. Finally, a computer simulation using MATLAB confirmed the model's accuracy. This research provides valuable insights for understanding and potentially controlling malaria outbreaks by highlighting the dependence of disease spread on climatic factors.

Key words: Mathematical model, equilibrium point, basic reproduction number, Local stability, global stability, sensitivity analysis.

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CHAPTER ONE

1. INTRODUCTION

1.1 Background of the Study

Malaria, a highly contagious disease caused by the Plasmodium genus of protozoan parasites, remains a significant global health burden (Paaijmans *et al*., 2007; Tumwiine *et al*., 2007; Tiu *et al*., 2021). Female *Anopheles* mosquitoes act as vectors, transmitting the parasite during blood meals (Bakary *et al.*, 2018). Despite global efforts, malaria claims countless lives yearly, with the majority of cases and fatalities concentrated in sub-Saharan Africa (WHO, 2022).

The distribution of the disease is also geographically different, with extensive prevalence in tropical and subtropical regions across Africa, Asia, Latin America, and parts of the Middle East and Europe (Diro Ana, 2019). Malaria is one of the most deadly diseases in Africa since it excessively affects pregnant women and children (WHO, 2022). A startling amount of the world's malaria cases is concentrated in Sub-Saharan Africa alone (WHO, 2022).

Ethiopia faces a particularly stark reality, with malaria estimated to encompass nearly 75% of the landmass and infect roughly 65% of the population. Especially, *Plasmodium falciparum* and *Plasmodium vivax* are the dominant strains, responsible for approximately 60% and 40% of cases, respectively (Gebremichael & Mekonnen, 2020; Alemu *et al*., 2011; Gizaw, 2024).

Using mathematical models to study the transmission dynamics of malaria can help understand the disease's behavior better. And the models have had a significant impact on how intervention methods for stopping and managing the spread of malaria are decided upon (Yiga *et al*., 2020; ul Rehman *et al*., 2021).

Ronald Ross (1911) started the first mathematical modeling study on malaria. He created a classical model that is now referred to as the traditional "Ross model", which clarified the connection between mosquito population and human malaria incidence.

Ross's model was modified by Macdonald (1957) by taking into account the latency period of the parasites in mosquitoes and their survival during that period. However, in this case, it was shown that reducing the number of mosquitoes is an ineffective control strategy that would have little effect on the epidemiology of malaria in areas of intense transmission.

Mathematical models of malaria often focus on environmental factors like temperature and rainfall, known to heavily influence the disease's spread (Yiga *et al.*, 2020; Agusto, 2020).

Temperature's impact is multifaceted. Warmer temperatures cause mosquitoes to feed more frequently while this boosts transmission (Gbenga J. Abiodun, 2018). higher temperatures (beyond 30-32°C) also shorten the mosquito's lifespan (Okuneye & Gumel, 2016). Interestingly, malaria burden peaks at a moderate temperature range (30-32°C) before sharply declining at extremes (>34°C) due to negative effects on both mosquitoes and parasites (Yiga *et al*., 2020; Agusto, 2020).

Rainfall similarly dances with malaria. Moderate amounts (32-110 mm) create ideal breeding grounds for mosquitoes, leading to increased abundance and higher transmission (Okuneye & Gumel, 2016). However, heavy rainfall can wash away these breeding sites, hindering mosquito populations (Okuneye & Gumel, 2016; Benedum *et al*., 2018). Therefore, understanding the intricate interplay of rainfall amount, frequency, and timing is crucial (Nissan *et al*., 2021). Additionally, the impact of rainfall can vary depending on the specific mosquito species involved (Okuneye & Gumel, 2016).

This study looks at how rainfall patterns, along with temperature, affect how malaria spreads. By figuring out this connection, we can better predict outbreaks and create better ways to control the disease. This builds on past research by Yiga et al. (2020) but focuses more on how rain affects malaria spread.

1.2 Statement of the Problem

Malaria remains a widespread and concerning disease, claiming countless lives across the nation (WHO, 2022). Its prevalence is particularly severe in tropical regions, where conducive environments favor the breeding of malaria mosquitoes (Gbenga J. Abiodun *et al.*, 2018). This pervasive spread underscores the urgent need for strategies to minimize disease transmission (Gbenga J. Abiodun *et al.*, 2018). Understanding the role of weather conditions, particularly temperature and rainfall, in influencing mosquito breeding is crucial to this effort (Yiga *et al*., 2020).

Temperature and rainfall play a significant role in malaria transmission dynamics (Okuneye & Gumel, 2016). Higher temperatures accelerate mosquito development, allowing them to mature faster and spread the disease more readily (Gbenga J. Abiodun *et al.*, 2018). Malaria parasites also thrive in warmer temperatures, although excessively high temperatures become detrimental to both mosquitoes and the parasite (Benedum *et al*., 2018). Similarly, rainfall directly impacts mosquito breeding (Okuneye & Gumel, 2016). Increased precipitation creates more breeding sites for mosquito larvae, leading to higher vector populations and consequently, greater disease transmission (Okuneye & Gumel, 2016). Understanding of how weather parameters, namely temperature and rainfall, influence malaria transmission dynamics is essential for informing effective control measures (Gbenga J. Abiodun *et al.*, 2018).

Therefore understanding the impact of temperature and rainfall on malaria transmission dynamics is crucial in reducing spread of malaria. This research expected to address this need by focusing on the following points:

- Developing a compartmental mathematical model that captures the effects of temperature and rainfall on malaria transmission dynamics.
- Well-posedness of the solution of the model.
- Equilibria and basic reproduction number of the model.
- Stability analysis of the model.
- Sensitivity analysis of the model parameters and their relative importance.

1.3 Objectives of the Study

1.3.1 General Objective

The general objective of this study was to analyze the impact of rainfall and temperature on malaria transmission dynamics.

1.3.2 Specific objectives

The specific objectives of the study were:

- To develop a compartmental mathematical model that captures the effects of temperature and rainfall in malaria transmission dynamics.
- To prove well-posedness of the model.
- To determine model equilibria and basic reproduction number.
- To show stability analysis.
- To carryout sensitivity analysis of the model parameters and determine their relative importance.

1.4 Significance of the Study

This study was believed to contribute to reducing malaria by identifying factors causing its spread and proposing effective strategies to protect community life.

1.5 Delimitation of the Study

This study was delimited to analyzing malaria disease transmission dynamics, focusing on temperature and rainfall as determinate factors, and employing compartmental mathematical study approaches.

CHAPTER TWO

2. LITERATURE REVIEW

Malaria is one of the world's most prevalent epidemics despite a series of control and eradication measures. It through mosquito bites, the Plasmodium parasite, which causes malaria, spreads the disease to humans. According to a 2017 World Health Organization report, there were an estimated 245 million malaria cases and 445 thousand malaria-related fatalities globally in 2016 (Gebremeskel, 2018). The number of cases of malaria worldwide in 2022 was 249 million and 608,000 malaria deaths in 85 countries (World Health Organization, 2023).

Plasmodium falciparum, *Plasmodium malariae*, *Plasmodium ovule*, *Plasmodium vivax* and *Plasmodium knowlesi* are the malaria parasites that infect humans (Bakary *et al*., 2018; Traoré *et al.*, 2021). Which are spread between humans through the bite of a young female *Anopheles* mosquito and in tropical regions and temperate regions, respectively, the most common species are *Plasmodium falciparum* and *Plasmodium vivax* (Yiga *et al.,* 2020).

The bulk of malaria-related deaths, particularly in sub-Saharan Africa, are caused by the deadly parasite *Plasmodium falciparum*, which also causes severe disease pathology. Although *Plasmodium vivax* has a far wider geographic range than *P. falciparum*, it usually causes illnesses that are milder (Sinden, 2017; Adegbite *et al*., 2023). A mosquito's life cycle starts with an egg, which hatches into a larva, which then becomes a pupa. After two to four days in the pupa stage, the mosquito becomes an adult (Yiga *et al.,* 2020).

The female Anopheles mosquito injects sporozoites into the blood of the human host when it bites the host. The Plasmodium parasite multiplies in the liver of the host as sporozoites before evolving into gametocytes, which are discharged into the bloodstream and consumed by a female Anopheles mosquito during a subsequent blood meal (Sinden, 2017). During the incubation period of malaria, symptoms may appear seven to thirty days after infection. Malaria symptoms include chills, fever, headaches, body aches, and vomiting (Yiga *et al*., 2020).

If the infection is not treated, severe malaria can develop, which can lead to organ failure or even death. Cerebral malaria, severe anemia, discomfort, kidney failure, acidosis, and hypoglycemia are some examples of severe malaria (Yiga *et al.,* 2020). Children under five

year's old and expectant mothers are the most susceptible populations to malaria (WHO, 2018).

A mosquito vector carries parasites that can cause malaria, an infectious disease that can be serious. In recent decades, the burden of malaria has decreased due to effective control techniques. It was projected that between 2010 and 2017, the incidence of malaria decreased by 18% worldwide (Kim *et al*., 2019). Due to temporal and regional heterogeneity, there is still a great deal of doubt regarding the drop in the malaria load, and high cases are still being recorded. Global estimates of 219 million new cases and 451,000 malaria-related fatalities were released in 2017 (Kim *et al*., 2019).

Mathematical models have been created to help control and eradicate malaria by providing insight into the mechanics of its transmission. The comprehension of intricate management tactics and diverse disease transmission characteristics has been significantly aided by mathematical modeling of infectious illnesses (Ul Rehman *et al*., 2021). Specifically, due to their higher predictive computing capabilities, mathematical models have proven to be more beneficial than statistical models for researching the variables impacting the dynamics of malaria transmission (Adegbite *et al*., 2023).

These days, mathematical models are widely used and valuable resources for understanding the dynamics of malaria transmission and providing insight into how to lessen the disease's impact on society. This is due to the fact that mathematical modeling can provide accurate answers to the following queries posed by public health authorities and decision-makers: So it has been proposed to analyze the dynamics of infectious diseases with the goal of reducing their transmission (Gebremeskel, 2018).

The life cycles of the Plasmodium malaria parasite and the Anopheles mosquito vector are strongly temperature-dependent (Yiga *et al*., 2020; Agusto, 2020; Eikenberry &Gumel, 2018). The aquatic immature Anopheles habitats are likewise highly reliant on local hydrodynamics and rainfall (Sharp, 1998). Changes in climate can create an environment that is conducive to the growth and reproduction of the malaria parasite and vector, causing malaria to appear in previously disease-free areas and they can also alter the intensity of malaria transmission by causing variations in biting patterns that are influenced by seasonal factors (Zhou *et al*., 2004). The high incidence of malaria in the tropics of Africa has been linked to favorable environmental factors for the development of larvae and the maturation of parasites within infected mosquitoes (Yiga *et al*., 2020).

It has long been known that malaria is a climate-sensitive illness, with summer time in temperate countries and wet lowlands in tropical regions historically being the times when it spreads. Unusual weather patterns have frequently sparked fatal epidemics. And all other things being equal, a range of statistical and dynamical transmission models of malaria transmission driven by climatic variables have been utilized in recent years to forecast likely changes in the disease's geographic distribution in a warmer world (Nissan *et al*., 2021).

Malaria is climate-sensitive (Gbenga J. Abiodun *et al.*, 2018). Temperature and rainfall are thought to be the primary meteorological variables that significantly influence malaria epidemics. It is crucial to comprehend which components of the relationship between climate and malaria transmission, morbidity, and death are foreseeable at various spatial and temporal scales in order to prevent climate change from derailing efforts to eradicate the disease in the future. Through its effects on the growth of vectors and parasites as well as the dynamics of transmission, climate can directly or indirectly affect malaria through a variety of channels, including the several socioeconomic factors that work together to determine (Nissan *et al*., 2021) Both the Plasmodium malaria parasite and the Anopheles mosquito vector depend heavily on temperature throughout their life cycles (Alemu *et al*., 2011; Yiga *et al*., 2020).

Excessive rainfall leads to an increase in mosquito breeding grounds and the spread of malaria (Kim *et al.*, 2019; Agusto, 2020; Oheneba-Dornyo *et al.*, 2022). However, some research suggests that heavy rains may short-term reduce mosquito populations by flushing early-stage larvae (Okuneye & Gumel, 2016; Benedum *et al*., 2018; Kim *et al*., 2019). The bulk of mosquito larvae were carried away by strong rains, which significantly increased larval mortality. The amount of rainfall, container size, and larval age all affected how much of an impact flushing had (Benedum *et al*., 2018). High temperatures decrease the time that parasites grow in mosquitoes, increasing the likelihood of transmission (Kim *et al.*, 2019; Alemu *et al*., 2011) Changes in temperature also affect mosquito development, reproduction, survival, and biting frequency (Kim *et al*., 2019; Loha & Ababa, 2015).

Thus, temperature and precipitation have a major influence on the dynamics of malaria transmission (Yiga *et al*., 2020; Okuneye & Gumel, 2016; Abdelrazec & Gumel, 2017; Benedum *et al*., 2018).

Studies show malaria, a global disease caused by the Plasmodium parasite, is strongly temperature-dependent and spreads through mosquito bites. The parasite and vector's life cycle are influenced by temperature and rainfall, with summer in temperate countries and wet lowlands in tropical regions being the most susceptible. High temperatures also decrease parasite growth, increasing transmission, and mathematical models have been developed to control and eradicate malaria by understanding transmission dynamics and providing accurate answers to public health authorities' questions. So it is very necessary to analyze the impacts of weather conditions, particularly temperature and rainfall, on malaria transmission dynamics.

CHAPTER THREE

3. METHODOLOGY

3.1. Study Area and Period

The study was conducted at Jimma University, in the Department of Mathematics, from September 2023 to June 2024.

3.2. Study Design

This study uses a mixed-design approach.

3.3. Source of Information

The relevant sources of information for this study were books, published articles, and related studies from the internet.

3.4. Mathematical Procedures

In order to achieve the stated objectives, the following procedures where implemented:

1. Formulating a mathematical model that encompasses temperature and rainfall.
2. Proving the well-posedness of the proposed model.
3. Determining model equilibria and the basic reproduction number.
4. Showing stability analysis of the equilibria.
5. Performing sensitivity analysis.
6. Analyzing the model outcomes.

CHAPTER FOUR

4 RESULT AND DISCUSSIONS

4.1 Preliminaries of the models

In this study, we used a non-linear deterministic model and the model is formulated by using system of ordinary differential equation. The stability analysis of the equilibrium points was done by classifying their steady states as Disease-free equilibrium point and Endemic equilibrium point. And also we used the next generation matrix to determine the basic reproduction number. The important terms that can serve as our methods relevant to the thesis are defined and explained in the following manner.

Definition 4.1: Ordinary differential equation (Ross, 2004)

An ordinary differential equation, denoted by ODE, is a differential equation involving one or more derivatives of a dependent variable with respect to a single independent variable. A typical n th order linear non-homogeneous ordinary differential equation is given by:

$$\frac{d^n \mathrm{y}}{dt^n} + a_1(\mathrm{t})\frac{d^{n-1}\mathrm{y}}{dt^{n-1}} + a_2(\mathrm{t})\frac{d^{n-2}\mathrm{y}}{dt^{n-2}} \ \ldots + a_{n-1}\frac{(\mathrm{t})\mathrm{dy}}{dt} + a_n(\mathrm{t})\mathrm{y} = \mathrm{g(t)}. \quad (1)$$

And system of ordinary differential equation is defined as:

$$\frac{dx}{dt} = F(X(t), t), \text{ Where } X(t) = \left(x_1(t), x_2(t), x_3(t), \ldots, x_n(t)\right)^T \quad (2)$$

$F = (f_1, f_2, f_3, \ldots, f_n)^T$ and $F_i = f_i(x_1(t), x_2(t), x_3(t), \ldots, x_n(t), i = 1,2,3, \ldots, n.$

Definition 4.2: Equilibrium point (Martcheva, 2015)

The equilibrium points to a system of first order differential equations are the points at which each differential equation is equal to zero. A point (x^*, y^*) is said to be the equilibrium point of the two dimensional dynamical system if:

$$\frac{dx}{dt} = f(x, y), \frac{dy}{dt} = g(x, y). \quad (3)$$

If it satisfies $f(x^*, y^*) = (0, 0)$ and $g(x^*, y^*) = (0, 0)$.

Definition 4.3: Stability of equilibrium points

1 Linearization Technique: for most dynamical systems the equilibrium point of the system of non-linear differential equations plays an important role in the analysis of a model.

Let $f: R^n \rightarrow R^n$ be a mapping and suppose that $x^* \in R^n$ is a point which makes $f(x^*) = 0$. That is x^* is said to be an equilibrium point for the system of differential equation $\frac{dx}{dt} = f(x(t))$. The linear part of f at x^* denoted by $Df(x^*)$ is the matrix of the partial derivative at x^*. That is, for any x $\in R^n$ we write:

$$f(x) = \begin{pmatrix} f_{1(x)} \\ f_{2(x)} \\ . \\ . \\ . \\ f_{n(x)} \end{pmatrix}. \tag{4}$$

The function f_i where $i = 1, 2, 3, \ldots, n$ are called the component of f. We define as:

$$Df(x^*) = \begin{bmatrix} \frac{\partial f_{1(x^*)}}{\partial x_1} & \frac{\partial f_{1(x^*)}}{\partial x_2} & . & . & . & \frac{\partial f_{1(x^*)}}{\partial x_n} \\ \frac{\partial f_{2(x^*)}}{\partial x_1} & \frac{\partial f_{2(x^*)}}{\partial x_2} & . & . & . & \frac{\partial f_{2(x^*)}}{\partial x_n} \\ . & . & . & . & . & . \\ . & . & . & . & . & . \\ . & . & . & . & . & . \\ \frac{\partial f_{n(x^*)}}{\partial x_1} & \frac{\partial f_{n(x^*)}}{\partial x_2} & . & . & . & \frac{\partial f_{n(x^*)}}{\partial x_n} \end{bmatrix}. \tag{5}$$

Equation (5) is called the Jacobian matrix of the system.

To find the eigenvalues of $Df(x^*)$ we solve the characteristic polynomial equation $|Df(x^*) - \lambda I| = 0$, where I is $n \times n$ identity matrix.

Therefore, the characteristic polynomial equation is:

$$\begin{vmatrix} \frac{\partial f_{1(x^*)}}{\partial x_1} - \lambda & \frac{\partial f_{1(x^*)}}{\partial x_2} & . & . & . & \frac{\partial f_{1(x^*)}}{\partial x_n} \\ \frac{\partial f_{2(x^*)}}{\partial x_1} & \frac{\partial f_{2(x^*)}}{\partial x_2} - \lambda & . & . & . & \frac{\partial f_{2(x^*)}}{\partial x_n} \\ . & . & . & . & . & . \\ . & . & . & . & . & . \\ . & . & . & . & . & . \\ \frac{\partial f_{n(x^*)}}{\partial x_1} & \frac{\partial f_{n(x^*)}}{\partial x_2} & . & . & . & \frac{\partial f_{n(x^*)}}{\partial x_n} - \lambda \end{vmatrix} = 0. \tag{6}$$

Let λ_1, λ_2 ,λ_3,.... λ_n, be the distinct eigenvalues of the Jacobian matrix of the n-dimensional autonomous linear system with $Det(J) \neq 0$. Then the equilibrium point x^* is stable if all the eigenvalues have negative real part and unstable if any one or more of the eigenvalues have positive real parts (Khalil, 2002).

2 Routh-Hurwitz stability criterions (Martcheva, 2015).

To obtain sufficient requirements for the global and asymptotic stability of such a rest point, we shall use the so-called direct technique of Lyapunov and Routh-Hurwitz Criteria. The Routh-Hurwitz Criteria is an essential criterion that specifies the necessary and sufficient conditions for all of the roots of the characteristic equation to be found in the left side of the complex plane. As this thesis progresses, we will apply the Routh-Hurwitz Criteria to determine the local stability of an equilibrium point.

Given the polynomial:

$$P(\lambda) = \lambda^n + a_1\lambda^{n-1} + \ldots + a_{n-1}\lambda + a_n. \quad (7)$$

Where the coefficients a_i are real constants and $i = 1, 2, \ldots, n$, define the n Hurwitz matrices using the coefficients a_i of the characteristic polynomial:

$$H_1 = (a_1),\ H_2 = \begin{pmatrix} a_1 & 1 \\ a_3 & a_2 \end{pmatrix},\ H_3 = \begin{pmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ a_5 & a_4 & a_3 \end{pmatrix},\ H_n = \begin{pmatrix} a_1 & 1 & 0 & 0 & \cdots & 0 \\ a_3 & a_2 & a_1 & 1 & \cdots & 0 \\ a_5 & a_4 & a_3 & a_2 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 & 0 & \cdots & a_n \end{pmatrix}. \quad (8)$$

Where $a_j = 0$ if $j > n$ all of the roots of the polynomial $p(\lambda)$ are negative or have negative real part if the determinants of all Hurwitz matrices are positive.

$det(H_j) > 0, j = 1, 2, \ldots, n$. When $n = 2$ Routh-Hurwitz Criteria simplify to $det(H_1) = a_1 > 0$ and

$$det(H_2) = det\begin{pmatrix} a_1 & 1 \\ 0 & a_2 \end{pmatrix} = a_1a_2 > 0, \text{ or } a_1 > 0 \text{ and } a_2 > 0.$$

For polynomial of degree $n = 2, 3, 4$ and 5, we summarize the Routh-Hurwitz Criteria as follows:

$n = 2 : a_1 > 0$, and $a_2 > 0$,

$n = 3 : a_1 > 0, a_3 > 0$, and $a_1a_2 > a_3$,

$n = 4 : a_1 > 0$, and $a_2 > 0, a_4 > 0$, and $a_1 a_2 a_3 > {a_3}^2 + {a_1}^2 a_4$,

$n = 5 : a_i > 0,\ i = 1, 2, 3, 4, 5,\ a_1 a_2 a_3 > {a_3}^2 + {a_1}^2 a_4$, and

$$(a_1 a_4 - a_5)(a_1 a_2 a_3 - {a_3}^2 - {a_1}^2 a_4) > a_5 (a_1 a_2 - a_3)^2 + a_1 {a_5}^2. \quad (9)$$

Sufficient condition for the roots of the polynomial $P(\lambda)$ to be negative or have negative real part is that all coefficients be strictly positive.

Definition 4.4: Let V be a continuous scalar function, that is $V : R^n \to R$. The function V is called positive definite on the entire space if:

- $V(x^*) = 0$,
- $V(x) > 0$, for $x \neq x^*$, where x^* is an equilibrium of the autonomous system $x' = f(x)$.

We define the derivative of $V(x)$ along the solutions of the system of differential equations as:

$$V'(x) = \frac{d}{dt} V(x(t)) = \frac{\partial V}{\partial x} \frac{dx}{dt}. \quad (10)$$

3 Lyapunov's Stability Theorem: If a function $V(x)$ is globally positively definite and radially unbounded, and its time derivative is globally negative $V'(x) < 0$, for all $x \neq x^*$ then the equilibrium x^* is globally stable (Martcheva, 2015).

Definition 4.5: If a function $V(x)$ exists that satisfies the conditions of Lyapunov's Stability Theorem, then this function is called a Lyapunov function (Martcheva, 2015).

4 Krasovkil-LaSalle Theorem: Consider the autonomous system $x' = f(x)$, where x^* is an equilibrium point i.e. $f(x^*) = 0$. Suppose there exists, a continuously differentiable function $L : R^n \to R$ that is positive definite on the entire space, radially unbounded, and that satisfies: $L'(x) \leq 0$ and $\forall x \in R^n$. Define the invariant set $\Omega = \{x \in R^n | L'(x) = 0\}$. if Ω contains only the equilibrium x^*, then x^* is globally stable (Martcheva, 2015).

Definition 4.6: The disease-free equilibrium is defined as the point at which there is no infected population i.e., $I_h = I_v = 0$ (Peter *et al*., 2018).

Definition 4.7: Endemic equilibrium points are steady-state situations where the diseases persist in the population i.e., $I_h = I_v \neq 0$ (Peter *et al*., 2018).

Definition 4.8: The basic reproduction number (R_0) is a threshold value used in epidemiology models to determine if a disease can spread to a population (Van Den Driessche & Watmough, 2002). When R_0 is less than one, each diseased individual creates less than one new infected individual, implying that a disease-free equilibrium point (DFE) is locally asymptotically stable.

If R_0 is more than one, the illness has the potential to invade or spread in the susceptible population without the use of control measures (Heffernan *et al*., 2005).

Let us assume that there are n compartments of which m are infected. We define the vector $x = x_i$, where $i = 1,2, \dots, n$, x_i denotes the number or proportion of individuals in the i^{th} compartment. Let $f_i(x)$ be the rate of appearance of new infections in compartment i, ${V_i}^+$ be the rate of transfer of individuals into compartment i by all other means and ${V_i}^-$ be the rate of transfer of individuals out of compartment i.

$$\frac{dx_i}{dx} = f_i(x) = F_i(x) - V_i(x), \tag{11}$$

where $V_i = {V_i}^- - {V_i}^+, i = 1,2,3, \dots, n$. The computation of the square matrices F and V of order $m \times m$, where m is the number of infected classes, defined by:

$$F = \frac{\partial F_i(x_0)}{\partial x_j} \text{ and } V = \frac{\partial V_i(x_0)}{\partial x_j}, \tag{12}$$

where $1 \leq i, j \leq m$, such that F is nonnegative, V is a non-singular matrix and x_0 is the disease-free equilibrium point (DFE). Since F is nonnegative and Vnonsingular, then V^{-1} is nonnegative and also FV^{-1}. Hence the matrix of FV^{-1} is called the next generation matrix for the model and $R_0 = \rho(FV^{-1})$, where $\rho(A)$ denotes the spectral radius of A when *spectral radius* of a matrix A is defined as the maximum of the absolute values of the eigenvalues of A.

4.2 Formulation of Mathematical Model

Referring to the work of Yiga *et al*. (2020) the model is formulated for the spread of malaria in human and mosquito populations. The total population sizes at time t are denoted by $N_h(t)$ and $N_v(t)$ for humans and mosquitoes, respectively. The human population is further divided into epidemiological compartments: susceptible ($S_h(t)$), infected ($I_h(t)$), and recovered ($R_h(t)$). Similarly, the mosquito population is divided into compartments: aquatic stages (eggs, larvae, pupae) denoted by $M_A(t)$, and adult female Anopheles mosquitoes categorized as susceptible ($S_v(t)$) and infected ($I_v(t)$).

The vector component of the model does not include an immune class. Mosquitoes never recover from the infection, their infected period ending with death due to their relatively short lifecycle. Hence, the total human population is $N_h(t) = S_h(t) + I_h(t) + R_h(t)$, and the total mosquito population is $N_v(t) = S_v(t) + I_v(t)$.

The basic model is built on the following set of assumptions:

- The populations in compartments of both humans and vectors are non-negative.
- All newborns are susceptible to infection and the development of malaria starts when the infected female mosquito bites the human host.
- Individuals move from one class to the other as their status with respect to the disease evolves.
- Humans enter the susceptible class through recruitment rate λ_h, leave from the susceptible class through death rate μ_h and infected rate $\beta_h S_h$.
- Human enter the infected class through infected rate $\beta_h S_h$ and leave through the recovered rate $\gamma_h I_h$.
- All human individuals, whatever their status, are subject to a natural death, which occurs at a rate μ_h and disease induced death rate ρ_h.

This allows us to examine the effects of temperature and rainfall on malaria disease dynamics based on the following schematic flow chart:

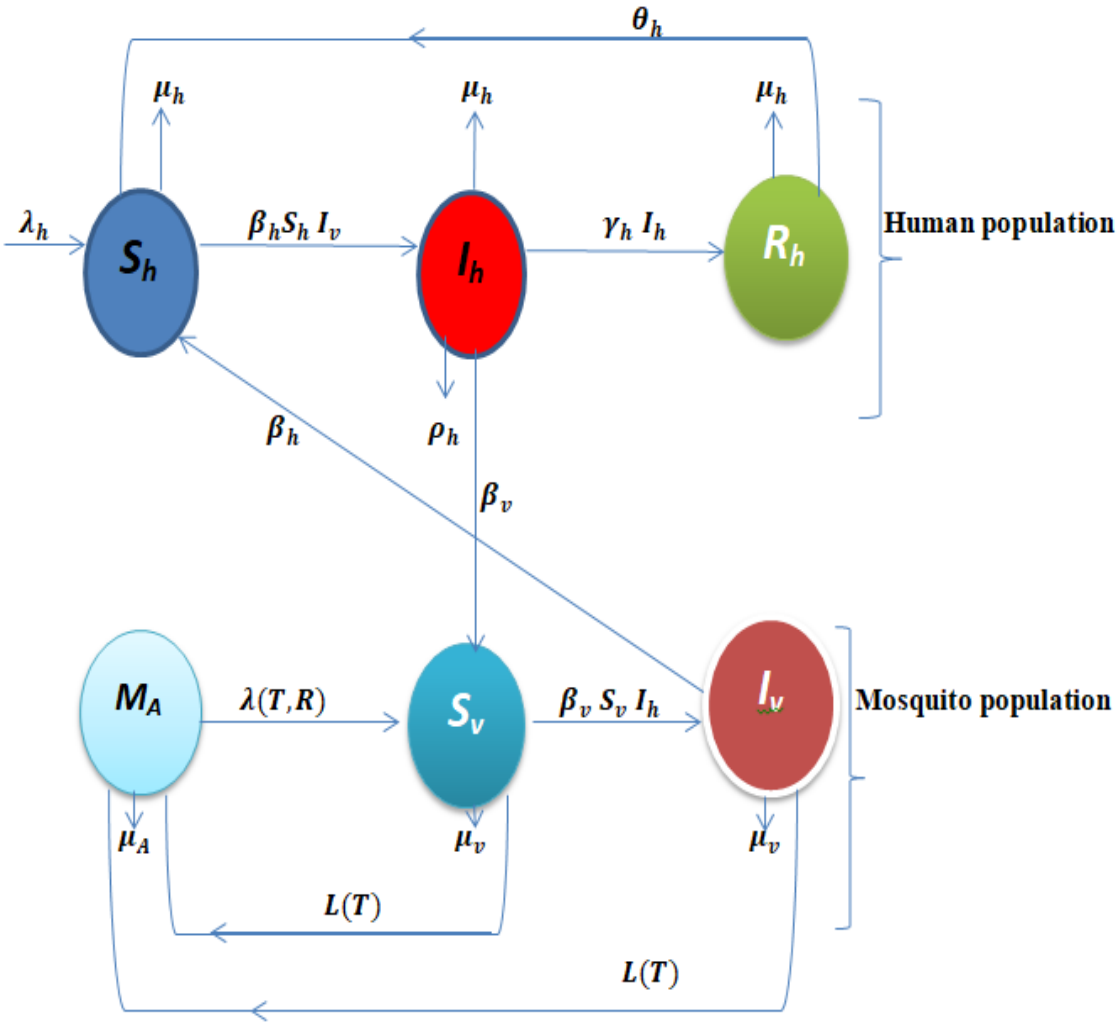

Figure 1: Schematic Flow Chart for the model.

Table 1: Descriptions of model variables

Variables	Descriptions of model variables
S_h	Number of susceptible humans
I_h	Number of infected humans
R_h	Number of recovered humans
S_v	Number of susceptible mosquitoes
I_v	Number of infected mosquitoes
M_A	Number of aquatic mosquitos
N_h	The total human populations
N_v	The total mosquito populations

Table 2: Descriptions of model parameters

Parameters	Descriptions of model parameters
λ_h	Recruitment rate of humans
μ_h	Natural death rate for humans
β_h	The humans contact rate
γ_h	Recovery rate for humans
θ_h	Humans loss of immunity rate
ρ_h	Disease induced death rate for humans
β_v	The mosquitos contact rate
$\lambda(T,R)$	Temperature and rainfall dependent maturity rate of aquatic mosquitoes
μ_A	Natural death rate for aquatic mosquitoes
μ_v	Natural death rate for adult mosquitoes
$L(T)$	Egg laying rate of adult mosquitos
K	Environment carrying capacity of female adult mosquitoes

From schematic flow diagram in Figure (1), we have the following model:

$$\left.\begin{aligned} \frac{dS_h}{dt} &= \lambda_h + \theta_h R_h - (\beta_h I_v + \mu_h) S_h \\ \frac{dI_h}{dt} &= \beta_h S_h I_v - (\rho_h + \mu_h + \gamma_h) I_h \\ \frac{dR_h}{dt} &= \gamma_h I_h - (\mu_h + \theta_h) R_h \\ \frac{dM_A}{dt} &= L(T)\left(1 - \frac{M_A}{K}\right)(S_v + I_v) - (\lambda(T,R) + \mu_A) M_A \\ \frac{dS_v}{dt} &= \lambda(T,R) M_A - (\beta_v I_h + \mu_v) S_v \\ \frac{dI_v}{dt} &= \beta_v S_v I_h - \mu_v I_v \end{aligned}\right\}. \tag{13}$$

$$\left.\begin{aligned} N_h(t) &= S_h(t) + I_h(t) + R_h(t) \\ N_v(t) &= S_v(t) + I_v(t) \end{aligned}\right\}. \tag{14}$$

4.3 Well-posedness of the model

4.3.1 Existence and uniqueness of solutions

This section identifies the region where the solution of the governing equation is both epidemiologically significant and mathematically well-defined. In other words, we establish that the solution of the proposed model in equation (13) exists within a specific feasible region.

Theorem 1: Existence and uniqueness of solutions. If $S_{h0}, I_{h0}, R_{h0}, M_{A0}, S_{v0}$ and I_{v0} are positive, then there exists a unique solution, $S_h(t), I_h(t), R_h(t), M_A(t), S_v(t)$ and $I_v(t)$ to the system (13) in R_+^6 for all $t \geq 0$.

Proof: To demonstrate the existence of a solution for system (13), we first rewrite the system in the form of:

$\frac{dx}{dt} = F(X(t), t)$, where $X(t) = \left(x_1(t), x_2(t), x_3(t), \dots x_n(t)\right)^T \in R_{+,}^6$

$F = (f_{1,}\ f_{2,}\ \ f_3, \dots, f_n)^T$ and $F_i = f_i(x_1(t), x_2(t), x_3(t), \dots, x_n(t), i = 1,2,3, \dots, n.$

$F(X(t), t)$ is given by:

$$\begin{bmatrix} f_1 \\ f_2 \\ f_3 \\ f_4 \\ f_5 \\ f_6 \end{bmatrix} = \begin{pmatrix} \lambda_h + \theta_h R_h - (\beta_h I_v + \mu_h) S_h \\ \beta_h S_h I_v - (\rho_h + \mu_h + \gamma_h) I_h \\ \gamma_h I_h - (\mu_h + \theta_h) R_h \\ L(T)\left(1 - \frac{M_A}{K}\right)(S_v + I_v) - (\lambda(T,R) + \mu_A) M_A \\ \lambda(T,R) M_A - (\beta_v I_h + \mu_v) S_v \\ \beta_v S_v I_h - \mu_v I_v \end{pmatrix}. \tag{15}$$

Note that, since f_1, f_2, f_3, f_4, f_5 and f_6 are all Continuous first derivative in R_+^6 existence of at least one solution for system (13) is guaranteed. By the well-known fundamental existence and uniqueness theorem (Perko, 2013).

4.3.2 Positivity of the solutions

Theorem 2: If the initial data $S_{h0}, I_{h0}, R_{h0}, M_{A0}, S_{v0}$ and I_{v0} are nonnegative, the solution $S_h(t), I_h(t), R_h(t), M_A(t), S_v(t), I_v(t)$ to the system (13) is nonnegative for all time $t \geq 0$.

Proof: We will demonstrate that the solutions of the system (13) are all positive. To achieve this, we will analyze each differential equation separately and prove the positivity of its solutions.

From the first equation in (13), we get:

$$\frac{dS_h}{dt} = \lambda_h + \theta_h R_h - (\beta_h I_v + \mu_h);\ \frac{dS_h}{dt} \geq -(\beta_h I_v + \mu_h) S_h, \tag{16}$$

Since λh and θ_h are a positive quantity, solving the inequality,

$$\frac{dS_h}{dt} \geq -(\beta_h I_v + \mu_h) S_h. \tag{17}$$

We obtain;

$$S_h(t) \geq S_{h0} e^{-\int_0^t (\beta_h I_v + \mu_h) ds} > 0. \tag{18}$$

Since the exponential term is always non-negative and $S_{h0} > 0$, we can deduce from equation (18) that $S_h(\mathrm{t}) > 0$.

From the second equation in (13), we get:

$$\frac{dI_h}{dt} = \beta_h S_h I_v - (\rho_h + \mu_h + \gamma_h) I_h;\ \frac{dI_h}{dt} \geq -(\rho_h + \mu_h + \gamma_h) I_h, \tag{19}$$

Solving the inequality,

$$\frac{dI_h}{dt} \geq -(\rho_h + \mu_h + \gamma_h) I_h. \tag{20}$$

We obtain;

$$I_h(t) \geq I_{h0}\, e^{-\int_0^t (\rho_h + \mu_h + \gamma_h) ds} > 0. \tag{21}$$

Because the exponential term is always non-negative and $I_{h0} > 0$, we can infer from equation (21) that $I_h(t) > 0$.

From the third equation in (13), we get:

$$\frac{dR_h}{dt} = \gamma_h I_h - (\mu_h + \theta_h)R_h; \frac{dR_h}{dt} \geq -(\mu_h + \theta_h)R_h, \quad (22)$$

Solving the inequality,

$$\frac{dR_h}{dt} \geq -(\mu_h + \theta_h)R_h, \quad (23)$$

We obtain:

$$R_h(t) \geq R_{h0}e^{-\int_o^t(\mu_h+\theta_h)ds} > 0. \quad (24)$$

Because the exponential term is always non-negative and $R_{h0} > 0$, we can conclude from equation (24) that $R_h(t) > 0$.

From the fourth equation in (13), we get:

$$\frac{dM_A}{dt} = L(T)\left(1 - \frac{M_A}{K}\right)(S_v + I_v) - (\lambda(T,R) + \mu_A)M_A; \frac{dM_A}{dt} \geq -(\lambda(T,R) + \mu_A)M_A, \quad (25)$$

Solving the inequality,

$$\frac{dM_A}{M_A} \geq -(\lambda(T,R) + \mu_A)dt, \quad (26)$$

We obtain:

$$M_A(t) \geq M_{A0}e^{-\int_0^t(\lambda(T,R)+\mu_A)ds} > 0. \quad (27)$$

Since the exponential term is always a non-negative and $M_{A0} > 0$, and we can deduce from equation (27) that $M_A(t) > 0$.

From the fifth equation in (13), we get:

$$\frac{dS_v}{dt} = \lambda(T,R)M_A - (\beta_v I_h + \mu_v)S_v; \frac{dS_v}{dt} \geq -(\beta_v I_h + \mu_v)S_v, \quad (28)$$

Solving the inequality,

$$\frac{dS_v}{dt} \geq -(\beta_v I_h + \mu_v)S_v, \quad (29)$$

We obtain:

$$S_v(t) \geq S_{v0} e^{-\int_0^t (\beta_v I_h + \mu_v) ds} > 0. \quad (30)$$

Since the exponential term is always a non-negative and $S_{v0} > 0$, we can deduce from equation (30) that $S_v(t) > 0$.

From the sixth equation in (13), we get:

$$\frac{dI_v}{dt} = \beta_v S_v I_h - \mu_v I_v; \ \frac{dI_v}{dt} \geq -\mu_v I_v, \quad (31)$$

Solving the inequality,

$$\frac{dI_v}{dt} \geq -\mu_v I_v, \quad (32)$$

We obtain:

$$I_v(t) \geq I_{v0} e^{-\int_0^t \mu_v ds} > 0. \quad (33)$$

Since the exponential term is always a non-negative and $I_{v0} > 0$, we can deduce from equation (33) that $I_v(t) > 0$.

4.3.3 Boundedness of the model solutions

All solutions $(S_h(t), I_h(t), R_h(t), M_A(t), S_v(t), I_v(t)) \in R_+^6$ of the malaria model Equation (13) are bounded, meaning that:

i. If $N_h(t) = S_h(t) + I_h(t) + R_h(t)$, then $lim_{t \to \infty} sup\, N_h(t) \leq \frac{\lambda_h}{\mu_h}$.

ii. If $N_v(t) = S_v(t) + I_v(t)$, then $\lim_{t \to \infty} sup\ N_v(t) \leq \frac{\lambda(T,R) M_A}{\mu_v}$.

Proof: (i) The sum of equations 1-3 in (13) gives:

$$\frac{dN_h}{dt} = \lambda_h - \mu_h N_h - \rho_h; \ \frac{dN_h(t)}{dt} \leq \lambda_h - \mu_h N_h. \quad (34)$$

Thus,

$$\frac{dN_h}{dt} \leq \lambda_h - \mu_h N_h. \quad (35)$$

From (35), we obtain:

$$N_h(t) \leq \frac{\lambda_h}{\mu_h} + [\, N_{h0} - (\frac{\lambda_h}{\mu_h})] e^{-\mu_h t}. \quad (36)$$

Showing that $N_h(t) \to \frac{\lambda_h}{\mu_h}\ as\ t\ \to\ \infty$ so $N_h(t) = S_h(t) + I_h(t) + R_h(t) \leq \frac{\lambda_h}{\mu_h}$.

At $t = 0, N_{h0} \geq 0$ and $N_{h0} \leq \frac{\lambda_h}{\mu_h}$ the bounded region of the system (13) for the human populations were $D_h = \left\{(S_h, I_h, R_h) \in R_+^3 : 0 \leq S_h + I_h + R_h \leq \frac{\lambda_h}{\mu_h}\right\}$.

Proof: (ii) The sum of equations (5) and (6) in (13), yields:

$$\frac{dN_v(t)}{dt} = \lambda(T,R)M_A - \mu_v Nv. \tag{37}$$

Solving equation (37) gives:

$$N_v(t) = \frac{\lambda(T,R)M_A}{\mu_v} + \left(N_{v0} - \frac{\lambda(T,R)M_A}{\mu_v}\right)e^{-\mu_v t}. \tag{38}$$

Letting t approach infinity in (38) implies that $N_v(t)$ asymptotically converges to $\frac{\lambda(T,R)M_A}{\mu_v}$. This signifies that the total adult mosquito population grows and reaches a stable value, $\frac{\lambda(T,R)M_A}{\mu_v}$. Therefore, $\frac{\lambda(T,R)M_A}{\mu_v}$ acts as an upper bound for the adult mosquito population $N_v(t)$ within the system (13). This upper bound is defined mathematically as:

$$D_v = \left\{(S_v, I_v) \in R_+^2 : 0 \leq S_v + I_v \leq \frac{\lambda(T,R)M_A}{\mu_v}\right\}.$$

4.4 Disease-free equilibrium point

Disease-free equilibrium points are steady-state solutions where there is no malaria in the human population. To find the disease-free equilibrium point of the proposed model (13), we set the right side of each equation in the model system (13) equal to zero. This inherently assumes that $I_h = R_h = I_v = 0$, since these variables represent infected humans, recovered humans, and infected mosquitoes, respectively, and in a disease-free state, they would all be zero.

Thus, Equation (13) takes the form:

$$\begin{cases} \lambda_h - \mu_h S_h = 0 \\ L(T)\left(1 - \frac{M_A}{K}\right)(S_v) - (\lambda(T,R) + \mu_A)M_A = 0. \\ \lambda(T,R)M_A - \mu_v S_v = 0 \end{cases} \tag{39}$$

Theorem 1 .The disease free equilibrium points of the proposed model (13) are:

1. $E_{01} = \left(\frac{\lambda_h}{\mu_h}, 0,0,0,0,0\right)$ whenever $\tau \leq 1$, and (40)
2. $E_{02} = \left(\frac{\lambda_h}{\mu_h}, 0,0, \frac{K\mu_v(\lambda(T,R)+\mu_A)[\tau-1]}{L(T)\lambda(T,R)}, \frac{K\mu_v(\lambda(T,R)+\mu_A)[\tau-1]}{L(T)\mu_v}, 0\right), \tau > 1,$ (41)

where $\tau = \frac{L(T)\lambda(T,R)}{\mu_v(\lambda(T,R)+\mu_A)}$.

Proof: From 2nd and 3rd equations in equation (39), we find either $M_A = 0$ or $M_A = \frac{K[L(T)\lambda(T,R)-\mu_v(\lambda(T,R)+\mu_A)]}{L(T)\lambda(T,R)}$.

Now, we apply different two cases to determine the disease free equilibrium points

Case 1: when $M_A = 0; I_v = I_h = R_h = 0$. Eq. (39) yields $S_h = \frac{\lambda_h}{\mu_h}$.

Therefore, the trivial disease free equilibrium point (E_{01}) of the model system (13) is

$$E_{01} = \left(\frac{\lambda_h}{\mu_h}, 0,0,0,0,0\right).$$

Case 2: When $M_A = \frac{K[L(T)\lambda(T,R)-\mu_v(\lambda(T,R)+\mu_A)]}{L(T)\lambda(T,R)}; I_v = I_h = R_h = 0$

From the 3rd equation in (39), we obtain:

$$S_v = \frac{\lambda(T,R)}{\mu_v} M_A = \frac{\lambda(T,R)}{\mu_v} \frac{K[L(T)\lambda(T,R)-\mu_v(\lambda(T,R)+\mu_A)]}{L(T)\lambda(T,R)}$$

Therefore, the nontrivial disease free equilibrium point when $\tau > 1$ is given by:

$$E_{02} = \left(\frac{\lambda_h}{\mu_h}, 0,0, \frac{K\mu_v(\lambda(T,R)+\mu_A)[\tau-1]}{L(T)\lambda(T,R)}, \frac{K\mu_v(\lambda(T,R)+\mu_A)[\tau-1]}{L(T)\mu_v}, 0\right).$$

4.5 Basic Reproduction Number

We employ the next-generation matrix method (Van Den Driessche & Watmough, 2002) to compute the basic reproduction number *(R_0)* of model (13) at the malaria disease-free equilibrium. To isolate the disease transmission dynamics, we focus only on the equations governing the infected sub-population, resulting in the following subsystem:

$$\frac{dI_h}{dt} = \beta_h S_h I_v - (\rho_h + \mu_h + \gamma_h) I_h. \quad (42)$$

$$\frac{dI_v}{dt} = \beta_v S_v I_h - \mu_v I_v. \quad (43)$$

Hence, the column matrices F_i and V_i are defined as:

$$F_i = \begin{bmatrix} \beta_h S_h I_v \\ \beta_v S_v I_h \end{bmatrix}. \quad (44)$$

$$V_i = \begin{bmatrix} (\rho_h + \mu_h + \gamma_h) I_h \\ \mu_v I_v \end{bmatrix}. \quad (45)$$

To calculate the matrices involved in the next-generation matrix method, we first compute the elements of the matrices F and V. F_{ij} represents the rate of appearance of infected individuals in compartment i due to new infections from compartment j, evaluated at the disease-free equilibrium (E_{01}). Similarly, V_{ij} represents the rate of transfer of individuals from compartment j to compartment i, excluding transitions due to infection, also evaluated at the disease-free equilibrium (E_{01}). These calculations involve taking partial derivatives of the model's functions (F_i and v_i) with respect to the state variables (x_j) and evaluating them at the disease-free equilibrium point.

We obtain:

$$F = \begin{bmatrix} 0 & \frac{\beta_h \lambda_h}{\mu_h} \\ 0 & 0 \end{bmatrix}. \quad (46)$$

$$V = \begin{bmatrix} (\rho_h + \mu_h + \gamma_h) & 0 \\ 0 & \mu_v \end{bmatrix}. \quad (47)$$

By inverting equation (47), we obtain:

$$V^{-1} = \begin{bmatrix} \frac{1}{(\rho_h + \mu_h + \gamma_h)} & 0 \\ 0 & \frac{1}{\mu_v} \end{bmatrix}. \quad (48)$$

From equations (46) and (48) we obtain:

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\beta_h \lambda_h}{\mu_v \mu_h} \\ 0 & 0 \end{bmatrix}. \quad (49)$$

The matrix on equation (49) has only one eigenvalue equal to zero; thus, the basic reproduction number is zero for this case. This implies that if an infective individual is introduced into the population at the steady-state E_{01}, the disease will not spread due to the absence of the parasite-transmitting mosquito vectors.

Similarly,

$$F = \begin{bmatrix} 0 & \frac{\beta_h \lambda_h}{\mu_h} \\ \frac{K\beta_v[L(T)\lambda(T,R) - \mu_v(\lambda(T,R)+\mu_A)]}{L(T)\mu_v} & 0 \end{bmatrix}. \quad (50)$$

Multiplying equation (50) with (48) we get:

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\beta_h \lambda_h}{\mu_v \mu_h} \\ \frac{K\beta_v[L(T)\lambda(T,R) - \mu_v(\lambda(T,R)+\mu_A)]}{L(T)\mu_v(\rho_h+\mu_h+\gamma_h)} & 0 \end{bmatrix}. \quad (51)$$

Now we can calculate the eigenvalues from equation (51) to determine the basic reproduction number R_0 by taking the spectral radius of the matrix. $|FV^{-1} - \lambda I| = 0$ (This equation computes the eigenvalues), and it yields:

$$\lambda = \pm\sqrt{\frac{\beta_h \lambda_h}{\mu_v \mu_h}\left(\frac{K\beta_v[L(T)\lambda(T,R) - \mu_v(\lambda(T,R)+\mu_A)]}{L(T)\mu_v(\rho_h+\mu_h+\gamma_h)}\right)}. \quad (52)$$

Thus, the dominant eigenvalue of the matrix on (51) or the basic reproduction number was given by:

$$R_0 = \frac{1}{\mu_v}\sqrt{\frac{\beta_h \lambda_h}{\mu_h}\left(\frac{K\beta_v[L(T)\lambda(T,R) - \mu_v(\lambda(T,R)+\mu_A)]}{L(T)(\rho_h+\mu_h+\gamma_h)}\right)}. \quad (53)$$

Note that: R_0 exists only if $L(T)\lambda(T,R) > \mu_v(\lambda(T,R) + \mu_A)$ implies $\tau > 1$ expressing R_0 in terms of τ gives as:

$$R_0 = \sqrt{\frac{\beta_h \lambda_h}{\mu_h}\left(\frac{K\beta_v[\mu_v(\lambda(T,R)+\mu_A)(\tau-1)]}{L(T)\mu_v{}^2(\rho_h+\mu_h+\gamma_h)}\right)}. \quad (54)$$

R_0 Exists only if the term under the square root is non-negative, that is $\tau \geq 1$; otherwise, if $\tau < 1$, there is no growth of the mosquito population, and malaria will not develop in the community since mosquito vectors are important for the spread of malaria.

4.6 Stability analysis of the Disease-Free Equilibrium

The Jacobian matrix of the system (13) was shown below:

$$J=\begin{pmatrix} \partial f1/\partial S_h & \partial f1/\partial I_h & \partial f1/\partial R_h & \partial f1/\partial M_A & \partial f1/\partial S_v & \partial f1/\partial I_v \\ \partial f2/\partial S_h & \partial f2/\partial I_h & \partial f2/\partial R_h & \partial f2/\partial M_A & \partial f2/\partial S_v & \partial f2/\partial I_v \\ \partial f3/\partial S_h & \partial f3/\partial I_h & \partial f3/\partial R_h & \partial f3/\partial M_A & \partial f3/\partial S_v & \partial f3/\partial I_v \\ \partial f4/\partial S_h & \partial f4/\partial I_h & \partial f4/\partial R_h & \partial f4/\partial M_A & \partial f4/\partial S_v & \partial f4/\partial I_v \\ \partial f5/\partial S_h & \partial f5/\partial I_h & \partial f5/\partial R_h & \partial f5/\partial M_A & \partial f5/\partial S_v & \partial f5/\partial I_v \\ \partial f6/\partial S_h & \partial f6/\partial I_h & \partial f6/\partial R_h & \partial f6/\partial M_A & \partial f6/\partial S_v & \partial f6/\partial I_v \end{pmatrix}.$$

As a result, the Jacobian of the system (13) takes the form:

$$J=\begin{pmatrix} -(\beta_h I_v+\mu_h) & 0 & \theta_h & 0 & 0 & -\beta_h S_h \\ \beta_h I_v & -(\rho_h+\mu_h+\gamma_h) & 0 & 0 & 0 & \beta_h S_h \\ 0 & \gamma_h I_h & -(\mu_h+\theta_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & -\frac{1}{K}L(T)(S_v+I_v)-(\lambda(T,R)+\mu_A) & L(T)\left(1-\frac{M_A}{K}\right) & L(T)\left(1-\frac{M_A}{K}\right) \\ 0 & -\beta_v S_v & 0 & \lambda(T,R) & -(\beta_v I_h+\mu_v) & 0 \\ 0 & \beta_v S_v & 0 & 0 & 0 & -\mu_v \end{pmatrix}. \quad (55)$$

Evaluating the Jacobian (J) matrix (55) at the disease-free equilibrium point (E_{01})yields:

$$J(E_{01})=\begin{pmatrix} -\mu_h & 0 & \theta_h & 0 & 0 & -\frac{\beta_h\lambda_h}{\mu_h} \\ 0 & -(\rho_h+\mu_h+\gamma_h) & 0 & 0 & 0 & \frac{\beta_h\lambda_h}{\mu_h} \\ 0 & 0 & -(\mu_h+\theta_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\lambda(T,R)+\mu_A) & L(T) & L(T) \\ 0 & 0 & 0 & \lambda(T,R) & -\mu_v & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_h \end{pmatrix}. \quad (56)$$

Similarly, the Jacobian (J) matrix (55) at the disease-free equilibrium point (E_{02}) yields:

$$J(E_{02})=\begin{pmatrix} -\mu_h & 0 & \theta_h & 0 & 0 & -\frac{\beta_h\lambda_h}{\mu_h} \\ 0 & -(\rho_h+\mu_h+\gamma_h-\delta_h) & 0 & 0 & 0 & \frac{\beta_h\lambda_h}{\mu_h} \\ 0 & 0 & -(\mu_h+\theta_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\lambda(T,R)+\mu_A) & \frac{\mu_v(\lambda(T,R)+\mu_A)}{\lambda(T,R)} & \frac{\mu_v(\lambda(T,R)+\mu_A)}{\lambda(T,R)} \\ 0 & \frac{-K\beta_v[L(T)\lambda(T,R)-\mu_v(\lambda(T,R)+\mu_A)]}{L(T)\lambda(T,R)} & 0 & \lambda(T,R) & -\mu_v & 0 \\ 0 & \frac{K\beta_v[L(T)\lambda(T,R)-\mu_v(\lambda(T,R)+\mu_A)]}{L(T)\mu_v} & 0 & 0 & 0 & -\mu_v \end{pmatrix}. \quad (57)$$

4.6.1 Local Stability of the Disease-Free Equilibrium

T**heorem 4:** The disease-free equilibrium point (E_{01}) of the model system (13) is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$.

Proof: The eigenvalues of Equation (56) take the form:

$$|J(E_{01})-\lambda I|=(-\lambda-\mu_h)(-\lambda-(\rho_h+\mu_h+\gamma_h))(-\lambda-(\mu_h+\theta_h))\begin{vmatrix} -\lambda-(\lambda(T,R)+\mu_A) & L(T) & L(T) \\ \lambda(T,R) & -\lambda-\mu_v & 0 \\ 0 & 0 & -\lambda-\mu_v \end{vmatrix}=0. \quad (58)$$

From (58), we obtain the eigenvalues; $\lambda_1 = -\mu_h, \lambda_2 = -(\rho h + \mu h + \gamma h), \lambda_3 = -(\mu h + \theta h)$ and all have negative real parts and the remaining eigenvalues are the roots of the equation:

$$(-\lambda - \mu_v)(\lambda^2 + \big(\mu_A + \mu_v + \lambda(T,R)\big)\lambda + \mu_A\mu_v - L(T)\lambda(T,R) + \mu_v\lambda(T,R)) = 0. \quad (59)$$

The roots of equation (59) are either $\lambda_4 = -\mu_v$, or

$$A\lambda^2 + B\lambda + \mathrm{C} = 0, \quad (60)$$

Where;

$$\begin{cases} A = 1 \\ B = \mu_A + \mu_v + \lambda(T,R) \\ C = \mu_v\big(\mu_A + \lambda(T,R)\big) - L(T)\lambda(T,R) \end{cases}. \quad (61)$$

By using Routh-Hurwitz criteria (Osman & Adu, 2017), Eq. (60) has a negative real root if and only if all the coefficients of Eq.(60) are positive. It is clear that both A and B are positive, as in (61) and $C = \mu_v\big(\mu_A + \lambda(T,R)\big) - L(T)\lambda(T,R) > 0$ implies that R_0 exists.

T**heorem 5:** The disease-free equilibrium point E_{02} of system (13) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: It has already been shown that the disease-free equilibrium E_{02} only exists if $\tau > 1$, If this condition is satisfied, then R_0 is real and positive. Thus, the basic reproduction number R_0 is biologically consistent. Using the theorem explained in (Van Den Driessche & Watmough, 2002) the disease-free equilibrium E_{02} is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

4.6.2 Global Stability of the Disease-Free Equilibrium

The global stability of the disease-free equilibrium is investigated using a theorem by (Castillo-Chavez *et al*., 2002).

System (13) can be expressed in terms of:

$$\frac{dX_1}{dt} = F(X_1, X_2). \quad (62)$$

$$\frac{dX_2}{dt} = G(X_1, X_2), G(X_1, 0) = 0. \quad (63)$$

The number of uninfected and infected population are represented in the system (13) by the variables X_1 and X_2 respectively where $X_1 = (S_h, S_v) \in R_+^2$ and $X_2 = (I_h, I_v) \in R_+^2$. E_0 Stands for disease-free equilibrium point and defined as $E_0 = (X^*, 0)$.

The two conditions listed below must be met for there to be global stability at the disease-free equilibrium point.

1. If $\frac{dX_1}{dt} = F(X^*, 0)$ then X^* is globally asymptotically stable.
2. $G(X_1, X_2) = AX_2 - \hat{G}(X_1, X_2)$ Where $\hat{G}(X_1, X_2) \geq 0$ for $(X_1, X_2) \in \Omega$.

At the second condition, $A = DX_2G(X^*, 0)$ is a Metzler matrix that is the off-diagonal entries are nonnegative, and Ω was the feasible region. Then, the following statement holds.

Lemma1 If $R_0 < 1$, then the equilibrium point $E_0 = (X^*, 0)$ of the system (13) is globally asymptotically stable, provided that condition 1 and 2 hold.

Theorem 6: for the system (13) the disease-free equilibrium point is globally asymptotically stable if $R_0 < 1$.

Proof: let $X_1 = (S_h, S_v) \in R_+^2$ and $X_2 = (I_h, I_v) \in R_+^2$ we group system (13) in to:

$\frac{dX_1}{dt} = F(X_1, 0)$, $\frac{dX_2}{dt} = G(X_1, X_2)$, where;

$$\frac{dX_1}{dt} = F(X_1, 0) = \begin{cases} \frac{dS_h}{dt} = \lambda_h - \mu_h S_h \\ \frac{dS_v}{dt} = \lambda(T, R)M_A - \mu_v S_v \end{cases}. \quad (64)$$

$$\frac{dX_2}{dt} = G(X_1, X_2) = \begin{cases} \frac{dI_h}{dt} = \beta_h S_h I_v - (\rho_h + \mu_h + \gamma_h) I_h \\ \frac{dI_v}{dt} = \beta_v S_v I_h - \mu_v I_v \end{cases}. \quad (65)$$

Now from equation (64) we obtain:

$$S_h(t) = \frac{\lambda_h}{\mu_h} + [S_{h0} - \frac{\lambda_h}{\mu_h}]e^{-\mu_h t}. \quad (66)$$

As t $\rightarrow \infty$ equation (66) approaches to $\frac{\lambda_h}{\mu_h}$ and;

$$S_v(t) = \frac{\lambda(T,R)M_A}{\mu_v} + \left[S_{v0} - \frac{\lambda(T,R)M_A}{\mu_v} \right] e^{-\mu_v t}. \quad (67)$$

As t $\rightarrow \infty$ equation (67) approaches to $\frac{\lambda(T,R)M_A}{\mu_v}$.

It has already been established that the threshold values for the human and mosquito populations are $\frac{\lambda_h}{\mu_h}$ and $\frac{\lambda(T,R)M_A}{\mu_v}$ respectively; thus, there is convergence in Ω. Therefore, X^* is globally asymptotically stable.

To investigate condition (2) for the disease-free equilibrium point E_{01}:

$$G(X_1, X_2) = AX_2 - \hat{G}(X_1, X_2) = \begin{pmatrix} -(\rho_h + \mu_h + \gamma_h) & \beta_h \frac{\lambda_h}{\mu_h} \\ 0 & -\mu v \end{pmatrix} \begin{pmatrix} I_h \\ I_v \end{pmatrix} - \begin{pmatrix} \beta_h I_v \left(\frac{\lambda_h}{\mu_h} - S_h\right) \\ -\beta_v S_v I_h \end{pmatrix}. \tag{68}$$

$$\hat{G}(X_1, X_2) = \begin{pmatrix} \beta_h I_v \left(\frac{\lambda_h}{\mu_h} - S_h\right) \\ -\beta_v S_v I_h \end{pmatrix}. \tag{69}$$

Where; $\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2)$

From equation (69) since $\hat{G}(X_1, X_2) < 0$, condition (2) is violated. Therefore, the disease-free equilibrium point E_{01} may not be globally asymptotically stable.

For the disease-free equilibrium point E_{02}:

$$A = \begin{pmatrix} -(\rho_h + \mu_h + \gamma_h) & \beta_h \frac{\lambda_h}{\mu_h} \\ \frac{K\beta_v[L(T)\lambda(T,R) - \mu v(\lambda(T,R) + \mu A)]}{L(T)\mu_v} & -\mu_v \end{pmatrix}. \tag{70}$$

By using similar process on equation (68) we obtain:

$$\hat{G}(X_1, X_2) = \begin{pmatrix} \beta_h I_v (\frac{\lambda_h}{\mu_h} - S_h) \\ \beta_v I_h (\frac{K[L(T)\lambda(T,R) - \mu_v(\lambda(T,R) + \mu_A)]}{L(T)\mu_v} - S_v) \end{pmatrix}. \tag{71}$$

It has already been established that the threshold value of the human population is $\frac{\lambda_h}{\mu_h}$ Therefore; $S_h \leq \frac{\lambda_h}{\mu_h}$ similarly, $S_v \leq \frac{K[L(T)\lambda(T,R) - \mu_v(\lambda(T,R) + \mu_A)]}{L(T)\mu_v}$ thus, equilibrium point E_{02} is globally asymptotically stable. Thus, by Lemma 1, the DFE E_{02} is globally asymptotically Estable for $R_0 < 1$.

4.7 Endemic Equilibrium point

The model allows for an equilibrium point, called the malaria endemic equilibrium point (E^*), denoted by $E^* = (S_h{}^*, I_h{}^*, R_h{}^*, M_A{}^*, S_v{}^*, I_v{}^*)$. This point signifies the presence of malaria within the population, as indicated by positive values for both infected sub-populations ($I_h > 0$, $I_v > 0$). to solve for this equilibrium point; we need to consider the following set of equations:

$$\begin{cases} \lambda_h + \theta_h R_h{}^* - (\beta_h I_v{}^* + \mu_h) S_h{}^* = 0 \\ \beta_h S_h{}^* I_v{}^* - (\rho_h + \mu_h + \gamma_h) I_h{}^* = 0 \\ \gamma_h I_h{}^* - (\mu_h + \theta_h) R_h{}^* = 0 \\ L(T)\left(1 - \frac{M_A{}^*}{K}\right)(S_v{}^* + I_v{}^*) - (\lambda(T,R) + \mu_A) M_A{}^* = 0 \\ \lambda(T,R) M_A{}^* - (\beta_v I_h{}^* + \mu_v) S_v{}^* = 0 \\ \beta_v S_v{}^* I_h{}^* - \mu_v I_v{}^* = 0 \end{cases} \quad (72)$$

Thus, the Endemic Equilibrium point of the model system (13) is:

$$E^* = (S_h{}^*, I_h{}^*, R_h{}^*, M_A{}^*, S_v{}^*, I_v{}^*), \quad (73)$$

Where;

$$S_h{}^* = \frac{\lambda_h(\lambda_h\beta_v(\mu_h+\theta_h)+\mu_v)}{\mu_h\lambda_h(\mu_h+\theta_h)\beta_v+\mu_h\mu_v R_0^2[(\rho_h+\mu_h)(\mu_h+\theta_h)+\mu_h\gamma_h)}, \quad (74)$$

$$I_h{}^* = \frac{\lambda_h\mu_v(\mu_h+\theta_h)({R_0}^2-1)}{\lambda_h\beta_v(\mu_h+\theta_h)+\mu_v{R_0}^2[(\rho_h+\mu_h)(\mu_h+\theta_h)+\mu_h\gamma_h)]}, \quad (75)$$

$$R_h{}^* = \frac{\gamma_h}{(\mu_h+\theta_h)} I_h{}^*, \quad (76)$$

$$M_A{}^* = \frac{{\mu_v}^2\mu_h(\rho_h+\mu_h+\gamma_h){R_0}^2}{\beta_v\beta_h\lambda_h\lambda(T,R)}, \quad (77)$$

$$S_v{}^* = \frac{{\mu_v}^2\mu_h(\rho_h+\mu_h+\gamma_h){R_0}^2}{\beta_v\beta_h\lambda_h(\beta_v I_h{}^*+\mu_v)}, \quad (78)$$

$$I_v{}^* = \frac{\mu_v\mu_h(\rho_h+\mu_h+\gamma_h){R_0}^2 I_h{}^*}{\beta_h\lambda_h(\beta_v I_h{}^*+\mu_v)}. \quad (79)$$

Solution: From the last two equations in (72), we obtain:

$$\lambda(T,R) M_A{}^* - \mu_v S_v{}^* - \mu_v I_v{}^* = 0,$$

$$(S_v{}^* + I_v{}^*) = \frac{\lambda(T,R)}{\mu_v} M_A{}^*. \quad (80)$$

By substituting equation (80) into the 4th equation of system (72), we get:

$$M_A{}^* = \frac{L(T)}{(\lambda(T,R)+\mu_A)}\left(1-\frac{M_A{}^*}{K}\right)\frac{\lambda(T,R)}{\mu_v}M_A{}^*,$$

Which yields either $M_A{}^* = 0$ or

$$M_A{}^* = \frac{K[L(T)\lambda(T,R)-\mu_v(\lambda(T,R)+\mu_A)]}{L(T)\lambda(T,R)}. \quad (81)$$

Writing (81) in terms of the basic reproduction number R_0, we obtain:

$$M_A{}^* = \frac{\mu_v{}^2\mu_h(\rho_h+\mu_h+\gamma_h){R_0}^2}{\beta_v\beta_h\lambda_h\lambda(T,R)}. \quad (82)$$

From the 6th equation in (72) and by using (82), we obtain:

$$S_v{}^* = \frac{\mu_v{}^2\mu_h(\rho_h+\mu_h+\gamma_h){R_0}^2}{\beta_v\beta_h\lambda_h(\beta_v I_h{}^*+\mu_v)}. \quad (83)$$

By adding the first three equations in (72), we obtain:

$$\lambda_h - \mu_h S_h{}^* - (\rho_h+\mu_h)I_h{}^* - \mu_h R_h{}^* = 0. \quad (84)$$

Solving for $R_h{}^*$ from the 3rd equation in (72), we obtain:

$$R_h{}^* = \frac{\gamma_h}{(\mu_h+\theta_h)}I_h{}^*. \quad (85)$$

By substituting (85) into (84) and solving for S_h^* in terms of I_h^* we obtain:

$$S_h{}^* = \frac{\lambda_h(\mu_h+\theta_h)-[(\mu_h+\theta_h)(\rho_h+\mu_h)+\mu_h\gamma_h]I_h{}^*}{\mu_h(\mu_h+\theta_h)}. \quad (86)$$

By solving for I_v^* from the 6th equation in (72) and writing interns of I_h^* and the basic reproduction number R_0, we obtain:

$$I_v{}^* = \frac{\mu_v\mu_h(\rho_h+\mu_h+\gamma_h){R_0}^2 I_h{}^*}{\beta_h\lambda_h(\beta_v I_h{}^*+\mu_v)}. \quad (87)$$

Using the 2nd equation in (72) and equations (86) and (87), we can solve for I_h^*. This gives:

$$I_h{}^* = \left(\frac{\beta_h}{(\rho_h+\mu_h+\gamma_h)}\right)\left(\frac{\lambda_h(\mu_h+\theta_h)-[(\rho_h+\mu_h)(\mu_h+\theta_h)+\mu_h\gamma_h)]I_h{}^*}{(\mu_h+\theta_h)\mu_h}\right)\left(\frac{\mu_v\mu_h(\rho_h+\mu_h+\gamma_h){R_0}^2 I_h{}^*}{\beta_h\lambda_h(\beta_v I_h{}^*+\mu_v)}\right),$$

$$I_h{}^* = \left(\frac{\lambda_h\mu_v(\mu_h+\theta_h){R_0}^2 I_h^* - \mu_v R_0^2[(\rho_h+\mu_h)(\mu_h+\theta_h)+\mu_h\gamma_h)](I_h^*)^2}{\lambda_h(\mu_h+\theta_h)(\beta_v I_h{}^*+\mu_v)}\right). \quad (88)$$

$$\lambda_h\mu_v(\mu_h+\theta_h){R_0}^2 I_h^* - \mu_v R_0^2[(\rho_h+\mu_h)(\mu_h+\theta_h)+\mu_h\gamma_h)](I_h^*)^2 = I_h^*\lambda_h(\mu_h+\theta_h)(\beta_v I_h^*+\mu_v). \quad (89)$$

Solving for I_h^* from Eq. (89), we get either $I_h^* = 0$ or

$$I_h^* = \frac{\lambda_h\mu_v(\mu_h+\theta_h)(R_0^2-1)}{\lambda_h(\mu_h+\theta_h)\beta_v+\mu_v R_0^2[(\rho_h+\mu_h)(\mu_h+\theta_h)+\mu_h\gamma_h)]}. \quad (90)$$

Thus, using equation (90) we obtain;

$${S_h}^* = \frac{\lambda_h(\lambda_h\beta_v(\mu_h+\theta_h)+\mu_v)}{\mu_h\lambda_h(\mu_h+\theta_h)\beta_v+\mu_h\mu_v R_0^2[(\rho_h+\mu_h)(\mu_h+\theta_h)+\mu_h\gamma_h)}. \quad (91)$$

Theorem 7: The endemic equilibrium point E^* of system (13) is globally asymptotically stable if $R_0 > 1$.

Proof: In the proof of the theorem, we consider the following Lyapunov function constructed as:

$$L(S_h, I_h, R_h, S_v, I_v) = \frac{1}{2}\left((S_h - S_h^*) + (I_h - I_h^*) + (R_h - R_h^*)\right)^2 + \frac{1}{2}\left((S_v - S_v^*) + (I_v - I_v^*)\right)^2. \quad (92)$$

From equation (92), we get:

$L(S_h, I_h, R_h, S_v, I_v) = 0$, iff $(S_h, I_h, R_h, S_v, I_v) = (S_h^*, I_h^*, R_h^*, S_v^*, I_v^*)$ and

$L(S_h, I_h, R_h, S_v, I_v) > 0$, otherwise, and

$$\frac{dL}{dt} = \left((S_h - S_h^*) + (I_h - I_h^*) + (R_h - R_h^*)\right)\frac{d(S_h+I_h+R_h)}{dt} + \left((S_v - S_v^*) + (I_v - I_v^*)\right)\frac{d(S_v+I_v)}{dt}. \quad (93)$$

Note that;

$$\begin{cases} \frac{dN_h}{dt} = \frac{d(S_h+I_h+R_h)}{dt} \leq \lambda_h - \mu_h N_h \\ \frac{dN_v}{dt} = \frac{d(S_v+I_v)}{dt} = \lambda M_A - \mu_v N_v \end{cases}. \quad (94)$$

Further;

$$\begin{cases} (S_h - S_h^*) + (I_h - I_h^*) + (R_h - R_h^*) = (S_h + I_h + R_h) - (S_h^* + I_h^* + R_h^*) = N_h - \frac{\lambda_h}{\mu_h} \\ (S_v - S_v^*) + (I_v - I_v^*) = (S_v + I_v) - (S_v^* + I_v^*) = N_v - \frac{\lambda {M_A}^*}{\mu_v} \end{cases}. \quad (95)$$

Using equations (94) and (95), equation (93) can be rewritten as:

$$\frac{dL}{dt} \leq \left(N_h - \frac{\lambda_h}{\mu_h}\right)(\lambda_h - \mu_h N_h) + \left(N_v - \frac{\lambda {M_A}^*}{\mu_v}\right)(\lambda {M_A}^* - \mu_v N_v). \quad (96)$$

Simplifying and rearranging (96), we obtain:

$$\frac{dL}{dt} \leq -\frac{1}{\mu_h}(N_h\mu_h - \lambda_h)^2 - \frac{1}{\mu_v}(N_v\mu_v - \lambda {M_A}^*)^2. \quad (97)$$

From (97), we conclude that:

$\frac{dL(S_h,I_h,R_h,S_v,I_v)}{dt} < 0$; And $\frac{dL(S_h,I_h,R_h,S_v,I_v)}{dt} = 0$ if and only if $S_h = S_h^*,\ I_h = I_h^*,\ R_h = R_h^*,$ $S_v = S_v^*,\ I_v = I_v^*$.

Thus, the largest closed and bounded invariant set in $\{(S_h, I_h, R_h, M_A, S_v, I_v) \in R_+^6 : \frac{dL}{dt} = 0\}$ is the singleton $\{E^*\}$, where E^* is the endemic equilibrium point. As a result, when $R_0 > 1$ in the region Ω, the unique equilibrium point E^* is globally asymptotically stable by La Salle (1976). This completes the proof of the theorem.

4.8 Sensitivity Analysis of the model

Sensitivity analysis aims to identify the model parameters that most significantly influence the model's output. This technique can be applied to both dynamic systems and static quantities like the basic reproduction number or equilibrium prevalence (Martcheva, 2015).

This section presents a sensitivity analysis of the basic reproduction number (R_0) with respect to several parameters influencing malaria transmission. The analysis aims to identify critical parameters that significantly impact R_0, thereby informing the development of more successful control and eradication strategies (Chitnis *et al*., 2008). To quantify the influence of each parameter, we will calculate the normalized sensitivity index

$$\pi_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}, \quad (98)$$

where the sensitivity index is denoted by $\pi_p^{R_0}$, and p represents the parameter.

Table 3: Parameter values of human and mosquito populations.

Parameter	Symbols	Values	Sources
Recruitment rate of humans	λ_h	10	(Alemu Geleta Wedajo, Boka Kumsa Bole, 2018)
Natural death rate of humans	μ_h	0.05	(Tilahun, 2017)
The human contact rate	β_h	0.01	(Tilahun, 2017)
Rate of loss of immunity	θ_h	0.9	(Tilahun, 2017)
Recovery rate	γ_h	0.9	(Tilahun, 2017)
Disease-induced death rate	ρ_h	0.01	(Tilahun, 2017)
Egg deposition rate	$L(T)$	$-0.153T^2 + 8.61T - 97.7$	(Mukhtar *et al*., 2019)
Aquatic mosquito death rate	μ_A	$1.0257 - 0.094T + 0.0025T^2$	(Mukhtar *et al*., 2019)
The mosquito contact rate	β_v	0.005	(Tilahun, 2017)
Adult mosquito death rate	μ_v	2	(Alemu Geleta Wedajo, Boka Kumsa Bole, 2018)
Environment carrying capacity of female adult mosquitoes	K	1,000,000	(Mukhtar *et al*., 2019)

Table 4 displays the sensitivity indices of R_0 to the parameters (independent of temperature and rainfall variations) that determine its value. The following parameter values are used: $\lambda_h = 10,\ \mu_h = 0.05,\ \beta_h = 0.01, \beta_v = 0.05,\ \gamma_h = 0.9,\ \rho_h = 0.01$ and $K = 1000000$. The MATLAB software is used to simulate the sensitivity of R_0 to temperature and rainfall. The temperature-rainfall-dependent parameters $L(T), \lambda(T, R)$, and μ_A are given by nonlinear functions.

Table4: Numerical values of sensitivity indices of R_0.

Parameter	Sensitivity index
μ_h	-0.5
β_h	0.5
β_v	0.5
λ_h	0.50345
γ_h	0.5
ρ_h	0.5

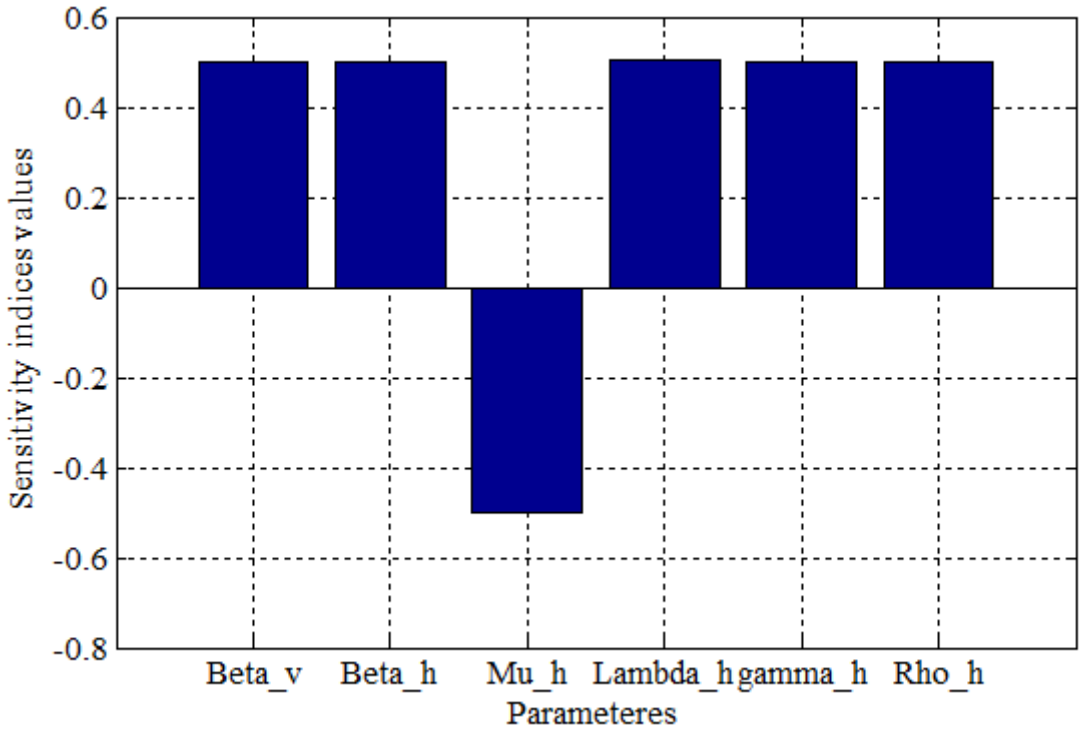

Figure 2: Graph of sensitivity indices vs. R_0.

4.8.1 Interpretation of Sensitivity Analysis

Our analysis reveals that some parameters have a greater impact on disease spread in the community. The parameters represented by β_v, β_h, λ_h, γ_h and ρ_h have positive sensitivity indices. As their values increase, so does the basic reproduction number (R_0), resulting in a higher spread of disease. Parameters with negative sensitivity indices μ_h have less impact on disease transmission as the value is increase. Figure 2 shows a visual representation of the sensitivity indices for R_0, according to Table 4. The parameters β_h, β_v, and λ_h seem to have the most impact on disease spread.

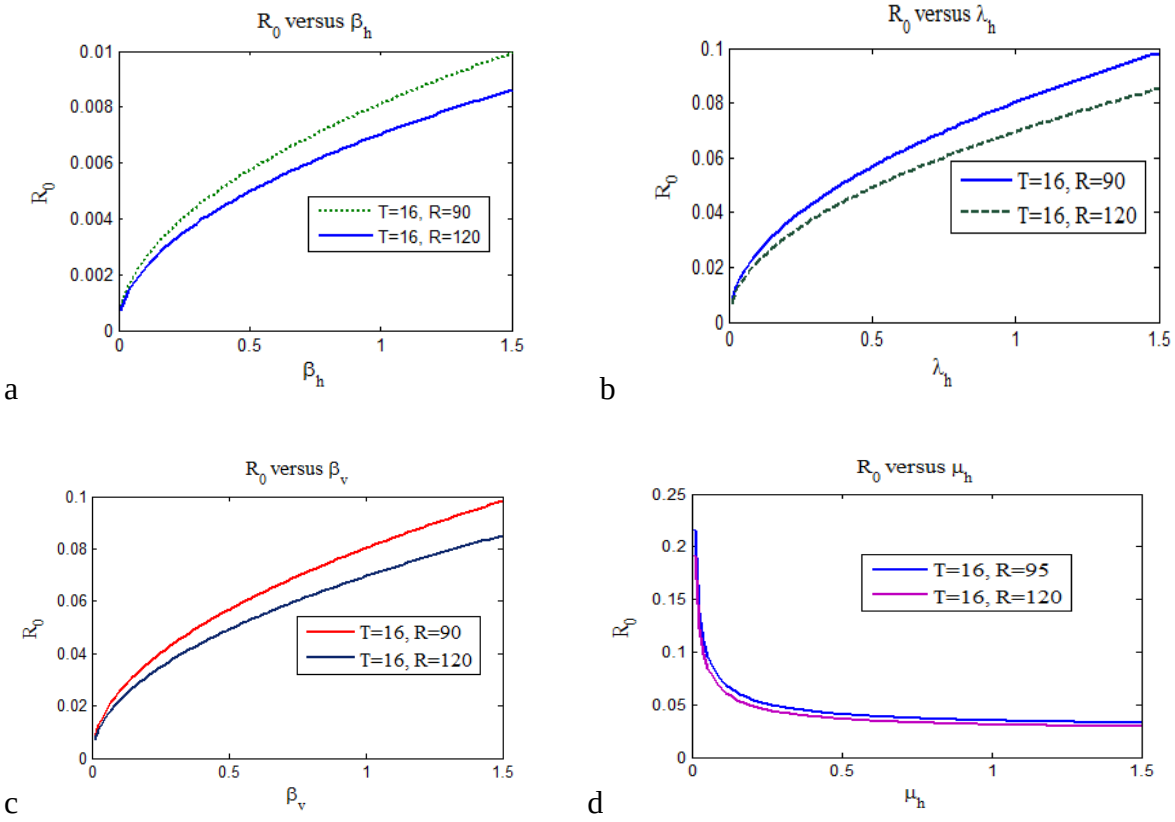

Figure 3: Graphs of the relationship between the basic reproduction number and various parameters.

Figure 3 shows a graph of the association between basic reproduction numbers and several parameters depending on temperature and rainfall. When we compare each of these parameters with R_0, the first is that, on the parameter β_h, as can be seen from the graph, R_0 increases as temperature and rainfall amount increase. This suggests that it has a significant impact on malaria transmission. Similarly, the parameters such as β_v and $\lambda_h$behave similarly with β_h. On the other hand, if we take μ_h its graph is falls down, which indicates that it, has a lesser effect on malaria transmission. This is based on a temperature of $16c^0$ and a rainfall of $90\ mm - 120\ mm$. The temperature was chosen because of the temperature blow 16°C is not favorable for mosquito a replication. And malaria transmission is maximized at the ranges between $90\ mm - 120\ mm$ (Okuneye and Gumel, 2016).

4.9 Numerical Simulations

To simulate the model system (13) using MATLAB, the following initial conditions were used: $S_h(0) = 5000000, I_h(0) = 500, R_h(0) = 300, M_A(0) = 200000, S_v(0) = 300000, I_v(0) = 1000$(Yiga *et al*., 2020).

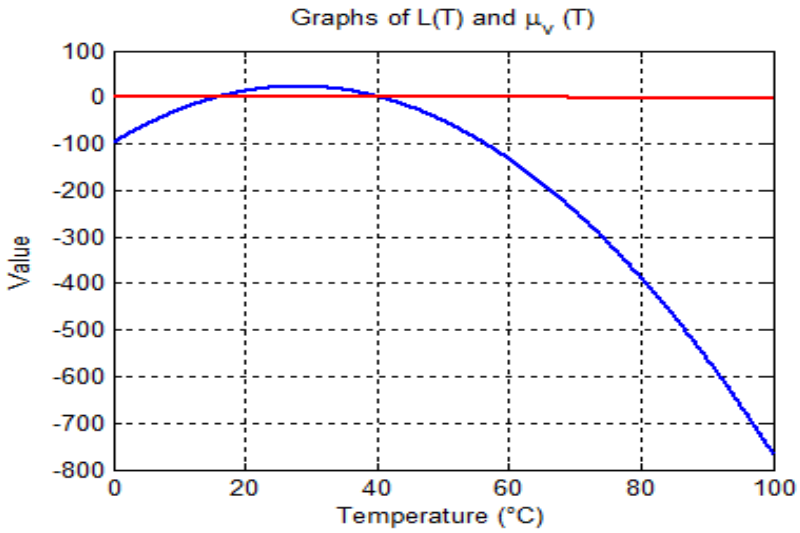

Figure 4: Egg deposition rate and adult mosquito death rate versus temperature

Figure 4 shows a graph of the link between temperature, mosquito egg laying (deposition) rate, and adult mosquito mortality rate. The graph indicates a clear tendency: as the temperature rises (between 16°C and 42°C), mosquitoes deposit more eggs. This observation is congruent with the findings of another investigation, Yiga et al. (2020). This study and the previous one show that a fundamental value known as the "basic reproduction number" exists, but only if a specific condition is met $(L(T) > \mu(v))$.

4.9.1 Parameters Determining the Aquatic Mosquito Maturation Rate

The mosquito maturation rate $\lambda(T, R)$ is affected by both temperature (T) and rainfall (R). It controls the rate at which juvenile mosquitos develop into mature adults, as described by the equation below (Mukhtar *et al*., 2019).

$$\lambda(T,R) = \frac{\omega(T)P_E(R)P_L(R)P_P(R)P_L(T)}{T_{MA}(T)}. \tag{99}$$

According to (Mukhtar et al., 2019), the formula for $\omega(T) = \frac{-0.153T^2 + 8.61T - 97.7}{-\ln(-0.000828T^2 + 0.0367T + 0.522)}$, this represents the lifetime number of eggs laid.

Rainfall has been proven to have a positive correlation with malaria incidence (Gbenga J. Abiodun *et al.*, 2018), but too much rain can flush away larvae and breeding grounds (Okuneye and Gumel, 2016). As a result, we assume a quadratic relationship between egg, larvae, and pupae survival rates and rainfall levels. We assume:

$$p(R) = [\frac{4{p_l}^*}{{R_l}^2}]R(R_l - R). \quad (100)$$

According to Parham and Michael (2010), R_l is the rainfall limit that flushes out breeding sites and prevents immature stages from surviving. The constant ${p_l}^*$ represents the greatest survival probability. It is considered that aquatic mosquitoes cannot tolerate a daily rainfall of more than 120mm (Okuneye and Gumel, 2016).

$P_E(R) = \frac{4*.93}{14400}R(120 - R)$,where $P_E(R)$ represents the daily survival probability of eggs (Parham & Michael, 2010).

$P_L(R) = \frac{4*.25}{14400}R(120 - R)$,where $P_L(R)$ represents the daily survival probability of larvae (Parham & Michael, 2010).

$P_P(R) = \frac{4*.75}{14400}R(120 - R)$, where $P_P(R)$ is the daily survival probability of pupae (Parham & Michael, 2010).

The temperature-dependent daily probability of larvae survival is denoted as $P_L(T) = e^{-(0.00554\mathrm{T} - 0.06737)}$, according to Parham and Michael (2010).

$T_{MA}(T)$, is the development time from egg to adult mosquito. Calculated as; $\frac{1}{-0.00094T^2 + 0.049T - 0.552}$ (Mordecai *et al.*, 2013).

$$\lambda(T,R) = \frac{-0.153T^2 + 8.61T - 97.7}{-\ln(-0.000828\mathrm{T}^2 + 0.0367\mathrm{T} + 0.522)}\frac{4*.93}{14400}R(120 - R)\frac{4*.25}{14400}R(120 - R)\frac{4*.75}{14400}R(120 - R)e^{-(0.00554\mathrm{T} - 0.06737)\frac{1}{-0.00094T^2 + 0.049T - 0.552}}.$$

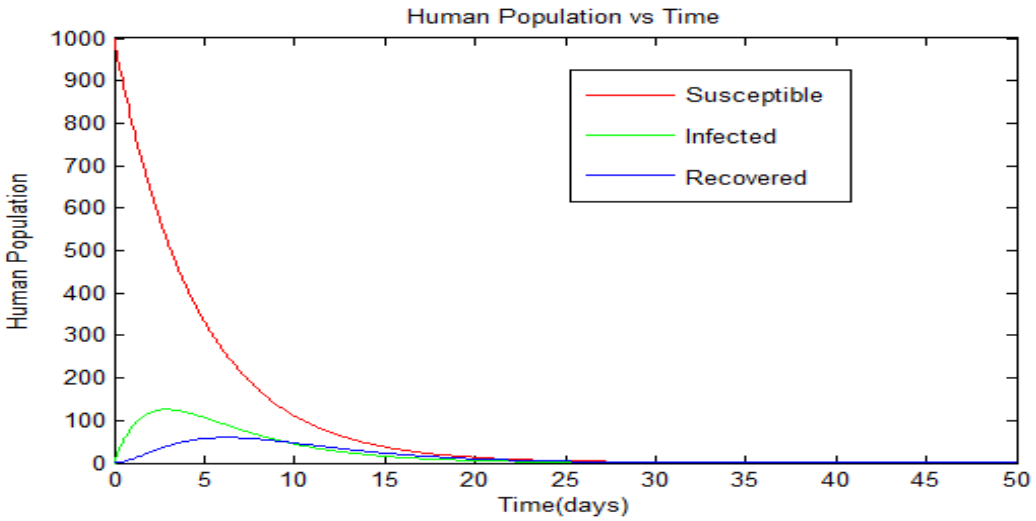

Figure 5: Change in the human population with time over a period of 50 days.

Fixing the temperature and rainfall Figure 5 shows that within the first 20 days, the susceptible human population decreased because the infectious human population increased. The infectious human population then declines as people recover and die.

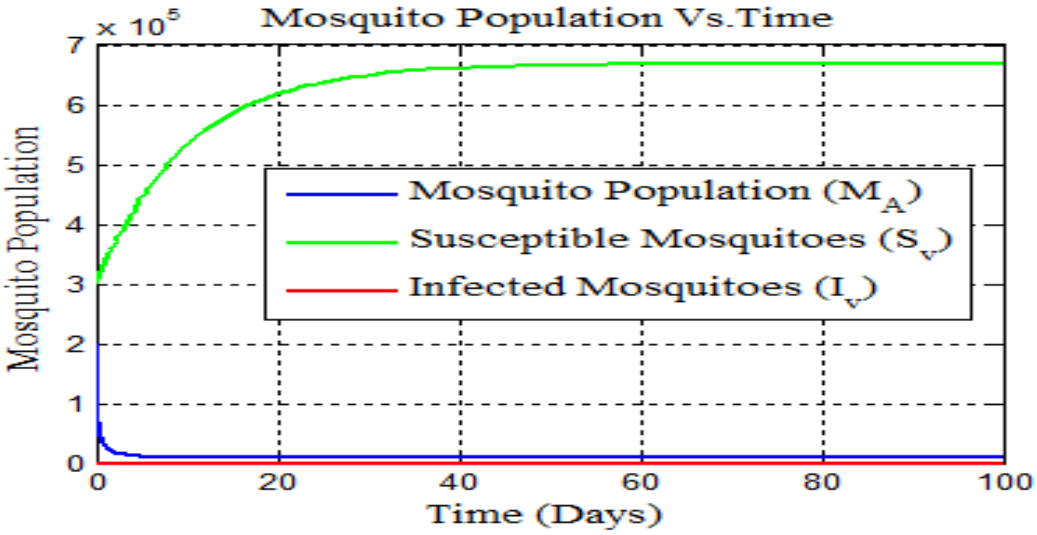

Figure 6: Change in the mosquito population with time over a period of 100 days.

By fixing the temperature and rainfall Figure 6 displays how the mosquito population changes over the course of 100 days. This indicates that fall in the aquatic mosquito population with susceptible mosquito population has increased.

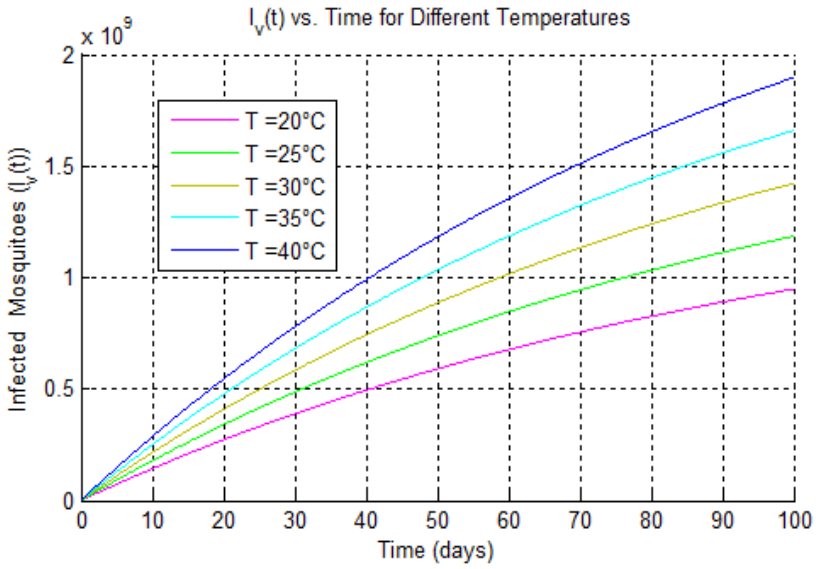

Figure 7: The number of infectious mosquitoes over a period of 100 days.

Figure 7 displays the development of infected mosquitoes over time at various temperatures. This shows that warm weather ecology has a significant influence on infected mosquito dynamics, which is consistent with earlier studies (Gbenga J. Abiodun, 2018). As a result, understanding the influence of climate change on malaria transmission dynamics is crucial for creating effective antimalarial treatments.

4.10: Discussion

In this study, a malaria transmission model with temperature and rainfall dependent parameters where developed. The model's study suggests that it was both mathematically and epidemiologically properly constructed. Further investigation reveals that there are two disease-free equilibrium points: the trivial disease-free equilibrium point (E_{01})(without mosquito population) and the nontrivial disease free equilibrium point (E_{02}) (with mosquito population).

The existence and stability of disease-free equilibria are determined by the vector reproduction number (τ). τ is a threshold parameter defined as the number of mosquitoes produced by a female Anopheles mosquito throughout its lifetime, which is completely governed by temperature-rainfall-dependent parameters (that is, egg deposition rate, maturation rate of aquatic mosquitoes to adulthood, and death rates of both adult and aquatic mosquitoes).

Seasonal conditions heavily influence the population size of mosquito vectors that transmit malaria, as mosquito replication is dependent on the value of τ. It was demonstrated that the mosquito population reproduce only when $\tau > 1$. The next-generation approach was used to calculate the model's fundamental reproduction number R_0, which was shown to exist only when $\tau > 1$. Malaria transmission relies on mosquito vectors, which only survive when $\tau > 1$. The disease-free equilibrium point E_{01} is stable if $\tau < 1$, which means that if an infected individual is introduced into the community at this point, malaria will not spread due to the absence of mosquito vectors.

The disease-free equilibrium point E_{02} exists if $\tau > 1$ and is stable if additionally $R_0 < 1$. this means that if an infected individual is introduced into the community, malaria will not spread if $R_0 < 1$; otherwise, there will be an outbreak. There is a unique endemic equilibrium if $R_0 > 1$. This would suggest that malaria remains in the community as long as $R_0 > 1$; thus, it is important to keep the basic reproduction number below unity.

To determine the optimal temperature range for mosquito reproduction, the threshold parameter τ was studied. The results showed that a replication range of $16c^0 - 42c^0$ is optimal. Temperatures outside of this range were not appropriate for mosquito reproduction.

CHAPTER FIVE

5 CONCLUSIONS AND FUTURE WORK

5.1 Conclusions

This study presents a compartmental mathematical model that incorporates climate variability (temperature and rainfall) to analyze the dynamics of malaria transmission. We developed SIR and SI systems of deterministic ordinary differential equations for human and adult mosquito populations, additionally accounting for the mosquito's aquatic stage**.**

Our study confirmed the mathematical and real-world validity of the malaria model (13). The model's operating domain is sound mathematically and reflects disease spread. We then investigated the stability of equilibria, which represent disease prevalence levels. We identified disease-free (no malaria) and endemic (consistent malaria) states.

A key factor is the basic reproduction number (R_0). It shows the average number of secondary infections caused by a single infectious individual in a healthy population. We used a method to determine R_0, with higher values indicating a more contagious disease.

We analyzed the stability of the disease-free state. For R_0 less than 1, the system is stable, meaning minor disruptions won't cause a permanent shift. Conversely, when R_0 is greater than 1, the disease-free state becomes unstable. Building on our understanding of how equilibria stability depends on R_0, this analysis offers valuable insights for controlling malaria outbreaks.

Next, the study analyzes the model's sensitivity to various parameters influencing malaria transmission. It aims to identify critical factors that significantly impact R_0, thereby informing the development of more effective control and eradication strategies. The analysis reveals that certain parameters have a stronger influence on disease spread. Here, positive sensitivity indices indicate a rise in transmission with increasing parameter values. Furthermore, the study highlights temperature and rainfall as key factors, with increasing transmission as these factors rise. Mosquito maturation rate is also affected by these climatic variables.

5.2 Future work

This study lays the foundation for further research in several key areas:

- **Making the model more detailed:** Researchers can add more things that affect malaria to the model, making it more realistic.
- **Optimal Control Strategies:** An ongoing investigation is focused on developing optimal control strategies for the disease. This approach aims to identify cost-effective combinations of prevention and treatment interventions that can significantly reduce or even eradicate the disease.
- **Fractional-Order Modeling:** Exploring modifications to the model by incorporating fractional-order derivatives holds promise for uncovering new insights and outcomes.

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