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A MATHEMATICAL MODEL FOR THE DYNAMICS OF DENGUE VIRUS DISEASE TRANSMISSION IN MANDERA COUNTY-KENYA

Research Report in Mathematics, Number 01, 2021

Abdiaziz Abdirashid

July 2021

Submitted to the School of Mathematics in partial fulfilment for a degree in Master of Science in Applied Mathematics

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Master Thesis

Submitted to the School of Mathematics in partial fulfilment for a degree in Master of Science in Applied Mathematics

Submitted to: The Graduate School, University of Nairobi, Kenya

Abstract

The aim of this project is to formulate a mathematical model for the dynamics of dengue virus disease transmission a case study of Mandera County Kenya.The compartmental model with 5-sate variables was developed to show the relationships that exist between the state variables.

The disease free equilibrium point was shown to be locally asymptotically stable when the basic reproduction number is less than unity and unstable if its greater than unity, in addition the global stability of the disease free equilibrium was determined by using the Lyapunov function and it was discovered that the disease free was globally asymptotically stable if the basic reproduction number is less or equal to unity.

The sensitivity index shows that the most sensitive parameters that affect the basic reproduction number are N_h and μ_v which has same degree of impact on R_0. Matlab version R2018a was used in performing the numerical simulations. We found out that the rate of susceptible human decreases from the rate of 1 and attain equilibrium at the rate of 0.4 while the rate of infected human increased from 0 and attain the equilibrium at the rate of 0.4.

The recovered human increased from 0 and attain equilibrium at the rate of 0.2. The susceptible Mosquito decline from the rate 1 and attain equilibrium after attaining the rate of 0.615 and finally the infected Mosquito increased from the rate of 0 and attain the equilibrium upon reaching the rate of 0.36.

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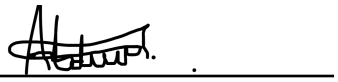
Declaration and Approval

I the undersigned declare that this dissertation is my original work and to the best of my knowledge, it has not been submitted in support of an award of a degree in any other university or institution of learning.



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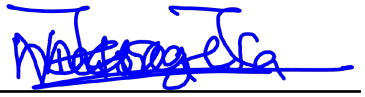
In my capacity as a supervisor of the candidate's dissertation, I certify that this dissertation has my approval for submission.



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Dedication

I dedicate this work to The Almighty, Blessed family , the School of Mathematics, University of Nairobi and to me for my time , sacrifice and research during the project

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First and foremost,I would like to thank the Lord Almighty for enabling me to complete this research work.

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Abdiaziz Abdirashid

Nairobi, 2021.

1 Introduction

1.1 Background

Dengue is a mosquito-borne viral infection that is usually found in tropical and sub-tropical regions around the world. In recent years, transmission has increased pre-dominantly in urban and semi-urban areas and has become a major public health concern[23].

There are four distinct but closely related serotypes of Virus that cause dengue (DEN-1, DEN-2, DEN-3 and DEN-4) and recovery from an infection by one provides lifelong immunity against that particular serotype. However, cross-immunity to the other serotypes after recovery is only partial and temporary. Subsequent infections by other serotypes increase the risk of developing severe Dengue[3].

World Health Organization (WHO) reported that over 2.5 billion peaople around the globe at risk of contacting dengue [23]. In addition WHO also estimated that there are approximately 50-100 million Dengue infections worldwide.The number of cases are not only increasing as the disease spreads to new areas,but explosive outbreaks are also occurring.

The history of Dengue virus disease date back to 1968 when it first started in Surabaya, East Java located in Indonesia [8]. There were 58 cases reported out of which 24 perished in that outbreak. Further, since that time, dengue disease tends to spread to the entire regions of Indonesia and by 1980 all provinces in Indonesia except East Timor have contracted the disease. This situation is closely related to the increasing mobility of the population and in line with the smooth transportation facilities. WHO revealed Indonesia as the country with the second highest dengue incidence in Southeast Asia with an average of approximately 130,000 cases over the period 2004-2010 and a maximum of 150,000 cases in 2007 [22].

In Kenya the first dengue outbreak was reported in 1982 in the coastal region. Dengue virus disease outbreak occurred almost 30 years later in the year 2011 in Mandera, northern Kenya and subsequently in Mombasa city in the coastal region (2013–2014) [18]. Dengue fever (DF) is presently the world's most important re-emerging arboviral disease with over 50 per cent of the world's population at risk of the disease and 50 per cent residing in dengue endemic countries [24].

In our world limited resources mathematical modeling is a powerful tool that is used to test and compare different intervention strategies that might be useful in controlling or eliminating Dengue. The formulation of the model and the possibility of a simulation with parameter estimation, allow tests for sensitivity and comparison of conjunctures[12]. In the case of dengue fever, the mathematical models we have found in the literature propose compartmental dynamics with Susceptible, Exposed, Infective and Removed (immunised). In particular, SEIRS models[19] and SIR models[17] with only one virus or two viruses acting simultaneously were considered [11].

1.2 Mode of transmission of Dengue Fever

Dengue is transmitted by species of mosquito known as Aedes aegypti, which transmit the dengue virus from person to person.The vector become infected after the female mosquito takes a blood meal from an infected human. Once the mosquito has been infected the virus incubates inside the mosquito host for approximately 8-10 days [13]. Upon completion of the incubation period the infected mosquito is capable of transmitting dengue to any human that it feeds on for the remainder of its life.

1.3 Symptoms of Dengue Fever

An individual infected with dengue virus exhibit the following symptoms.

- Fever
- Headache
- Muscle
- Joint pain
- Skin rashes
- Bleeding

1.4 Statement of the problem

Despite the documented presence of the species of Mosquito known as Aedes aegypti that transmit dengue virus and the rapid increase of dengue cases reported in Africa, most documented reports on dengue come from a small number of countries, with few prospective and population based studies[16]. With many competing public health problems, the clinical presentation of dengue is non-specific and difficult to distinguish from

other causes of febrile illness, especially with dengue diagnostic assays not widely available [27]. Also, unlike many countries in Asia and Latin America, most African countries lack systems of mandatory reporting of dengue cases [4].

In kenya despite reported cases of dengue in Mombasa,Mandera, Taita-Taveta and Urban slums of Kibera located in Nairobi their is no study that has been done to model the dynamics of Dengue virus disease transmission and this study aims at bridging the gap by formulating a mathematical model to determine the Dynamics of dengue Virus disease transmission in Mandera County.

1.5 Research Objectives

1.5.1 Objective of the Study

The general objective of this study is to develop and analyse a mathematical model for the dynamics of dengue virus disease transmission in Mandera County Kenya.

1.5.2 Specific Objectives

The specific objective of this study are;

- To determine the stability of the disease free equilibrium point.
- To determine the basic reproduction number.
- To determine the most sensitive parameter to R_0 .
- To obtain Numerical Simulation of the model variables.
- To give recommendations in mitigating dengue virus disease transmission.

1.6 Justification of the study

The study on the outbreak of Dengue virus disease among the residents living in Mandera county has never been done before despite reported cases of the disease in that region, Its against this backdrop we endeavour to model the dynamics of dengue virus disease outbreak in Mandera county to establish the behaviour of the model parameters and understand their meanings.

2 Literature Review

2.1 Introduction

2.2 Mathematical Modeling of Dengue Virus

In the existing literature mathematical models are widely used in describing and analyzing the behaviors of dengue virus disease transmission. Susceptible-Infected-Recovered(SIR) model is commonly used in mathematical model simulations of an infectious disease epidemiology.

Anderson and May (1992) [2] gave a comprehensive survey on the use of mathematics to study infectious diseases modeling, and since then there has been an increasing number of mathematical epidemiology papers published. Many infectious diseases are spread by biting insects and ticks or other organisms,collectively known as vectors, which transfer pathogens between humans or other animals. The emergence or reemergence of such vector-borne diseases seems especially to have stimulated recent interest. Rogers et al [21] reviewed the early vector-borne disease models, and many other authors have studied various particular vector-borne diseases, such as malaria [15], West Nile virus [7] and dengue fever [10].

The models by Derouich et al and by Syafruddin et al (2003)[9] are among the simplest since both of them used S-I-R Ordinary differential equations models of one strain of the virus and Iurii Bakach (2015) [3] used five different models in his study on survey of Mathematical Model of Dengue fever and observed that every Model was different.The model was used by Ganga Ram Phaijoo and Dil Bahadur Gurungs (2016) [25] in describing the transmission of dengue disease with constant human and vector populations. They divided the Human population into three compartment susceptible, infected and recovered and the Mosquito Population was divided into two compartment, susceptible and infected compartment, they found out that for higher level of awareness the disease was seen to affect less number of people and Large number of people is seen to be affected from the disease when there is no awareness on dengue in the host population.

Chanpra sopchai et al (2017) [5] proposed a SEIR(susceptible-exposed-infected-recovered) model for Thailand and the analysis was based on Routh-Hurwitz criteria to establish the local asymptotic stability of the equilibrium points. They established two equilibrium points a disease-free equilibrium point and an endemic equilibrium point. The disease-free equilibrium point is locally asymptotically stable

Esteva and Vargus (2021) proposed an SIR (susceptible-infected-recovery) model to describe the transmission of dengue disease with constant human and vector populations

2.2.1 SIR Model and Concepts

Infectious disease comprises of measles, chicken pox and to a deadly killer disease such as Acquired immune deficiency syndrome(AIDS).Infectious disease dynamics has been extensively studies for many years in the field of epidemiology.To model the progress of an outbreak of an infectious disease in a large population it is necessary to rediuce the population diversity a small number which corresponds to infectious disease under consideration.

W. O. Kermack and A. G. McKendrick (1927) [14] did a study on infected humans who were infected with a constagious disease. They used SIR model in their study and they assumed that the population is constant and Incubation of an infectious agent is instant, and the infection period is equal to the duration upon which the disease exist in the population, In addition they assumed that the population is homogeneous where no age, spatial, or social structure is considered. In their Model they divided the population into a number of compartments,each comprising of individuals that are similar in terms of status with respect to the disease.The human population was divided into three compartment, susceptible human to the disease at time t abbreviated as (S), Infected human with the disease at time t abbreviated as (I) and recovered individual from the disease at time t(R).

Traditionally in the research areas of an infectious disease epidemiology the compartments of host population is denoted as S(t),I(t) and R(t), respectively. The total population of human is shown in Equation (1)

$$S + I + R = N \tag{1}$$

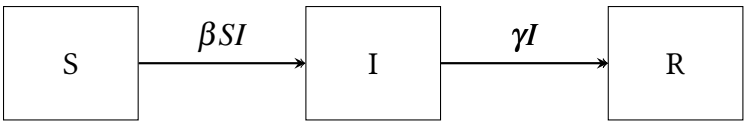

Figure 1. Diagrammatic representation of the Basic SIR model.

The model flow was considered as follows:Having compartmentalized the host population, Kermack and McKendrick derived the following set of equations (2a),(2b) and (2c) specified how the compartments size change over a period of time.

$$\frac{dS}{dt} = -\lambda S \tag{2a}$$

$$\frac{dI}{dt} = \lambda S - \gamma I \tag{2b}$$

$$\frac{dR}{dt} = -\gamma I \tag{2c}$$

λ is the rate of an infection and is a measure of how quickly a susceptible humans get infected. γ denoted the recovery rate and is a measure of how quickly the infected human recover from the disease.

The model assume the population is homogeneous with the same probability of transmuting the disease. Infected individual makes contact With a susceptible Mosquito and transmit the disease with λN per unit time. The proportion of contacts by an infected human with a susceptible mosquito is given by $\frac{S}{N}$.

The rate of new infections is given us λN($\frac{S}{N}$), and the rate of individuals leaving the susceptible class due to new infections is given us λN($\frac{S}{N}$)I=λSI.

Equation (2b) shows the infected class leaving the susceptible class category, a proportion of γ is exiting the infectious class and join the recovered class. These processes that occur at the same time are called the Law of Mass, which is defined as the rate of contact between two groups in a population is proportional to the size of each groups concerned.

If the SIR model is expanded to include births and deaths variables, then the model is as shown in Equation (3a),(3b) and (3c)

$$\frac{dS}{dt} = \mu N - \lambda SI - \mu S \tag{3a}$$

$$\frac{dI}{dt} = \lambda SI - \gamma I - \mu I \tag{3b}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{3c}$$

μ denotes the rate birth and mortality. The human recruitment rate due to birth is equal to the natural mortality given by

$$\mu N = \mu(S + I + R) \tag{4}$$

The model assume that the population is constant on the onset of the disease outbreak since the time for change in population is much longer than the period under infection.

2.2.2 SEIR Model

Incubation period occur when an individual human infected are unable to spread the disease to the susceptible vector . During this period the individual is in the category of compartment exposed denoted as (E). The diagram in Figure(2) is an extension of SIR Model to include the exposed class category and the model is then refereed to as SEIR Model.

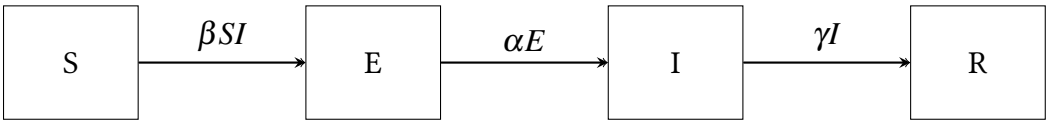

Figure 2. Diagrammatic representation of the SEIR model

Now considering a model which contains exposed compartment, an individual may leave the exposed class due to an infection and join the infected class category.in their Model the incubation period is assumed to be a random variable in the form of an exponential distribution having a parameter α and all the other assumptions and parameter sets are the similar based on the Model Equations (3a),(3b) and (3c). The SEIR model is given by Equation (5a),(5b),(5c) and (5d)

$$\frac{dS}{dt} = \mu N - \lambda SI - \mu S \tag{5a}$$

$$\frac{dE}{dt} = \lambda SI - \alpha E - \mu E \tag{5b}$$

$$\frac{dI}{dt} = \alpha E - \gamma I - \mu I \tag{5c}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{5d}$$

Since the Model assume total population is constant , we have the total population given as;

$$S + E + I + R = N \tag{6}$$

2.2.3 Force of Infection

In Equation (5a) λSI is the rate at which susceptible individuals contract the disease and is refereed to as force of infection. For a bigger classes of an infection , it is imperative to consider a force of infection that are independent on the absolute number of individual who are infectious, but on their proportion with regards to the total human population N, therefore we let $F = \lambda \frac{1}{N}$

Then Equation (5a),(5b),(5c) and (5d) becomes;

$$\frac{dS}{dt} = \mu N - \lambda S\frac{1}{N} - \mu S \tag{7a}$$
$$\frac{dE}{dt} = \lambda S\frac{1}{N} - \alpha E - \mu E \tag{7b}$$
$$\frac{dI}{dt} = \alpha E - \gamma I - \mu I \tag{7c}$$
$$\frac{dR}{dt} = \gamma I - \mu R \tag{7d}$$

The exposed class are non-infectious and therefore does not lead to the new infections and do not affect in the decline of the susceptible class category. Their is a delay between when an individual is infected and when they become infectious. The effect of the exposed class category can be included into expressions for other class category, which will then make the model simpler.

if we let u(t) to be the proportion of an infected human compartment at time t who are non-infectious,Then the proportion of becoming ineffective at infection duration t is given by $\mu u(t)$, and we have;

$$\frac{du}{dt} = -\mu u(t), u(0) = 1 \tag{8a}$$

The solution of (8a) is

$$u(t) = e^{-\mu t} \tag{9a}$$

if we assume that the incubation period is a constant τ . The individuals changing from exposed to infected are given as τ days, and the recruitment number of infected at time t is related to the number of susceptibles and infecteds at time (t), subject to surviving the incubation period, whose proportion is given by $e^{-\mu\tau}$

Then Equation (7a),(7b), (7c) and (7d) becomes;

$$\frac{dS}{dt} = \mu N - \lambda \frac{S(t)I(t)}{N} - \mu S(t) \tag{10a}$$

$$\frac{dI}{dt} = e^{-\mu\tau}\lambda \frac{S(t-\tau)I(t-\tau)}{N} - \gamma I(t) - \mu I(t) \tag{10b}$$

$$\frac{dR}{dt} = \gamma I(t) - \mu R(t) \tag{10c}$$

This SEIR model plays an important role in the field of infectious disease epidemiology, especially when the incubation period consume a lot of individual's life span is put into consideration.The SEIR model does not only consider the spread of an infectious disease in human but also animals. Mosquito is a carrier that transmit infectious disease to human. The infectious disease transmitted by Mosquito to human comprises of Malaria, Dengue fever and the west Nile Virus. Once the Mosquito become infected the disease stays in their body for a long time and transmit the disease for the reminder of its lifetime. SEIR model is mainly used to help in the developments of Mosquito and human interaction that results in the transmission of an infectious disease.

3 Mathematical model formulation

3.1 Introduction.

In these chapter,we intends to come up with a compartmental diagram representing the human and mosquito population, develop a mathematical model of the dynamics of dengue fever transmission based on the compartmental diagram and formulate model assumptions, define the variables and parameters of the model, check for the positivity and boundedness of the model solution, determine the basic reproduction number and finally the steady state will be analysed to determine the local and Global stability.

3.2 Model formulation and assumption

After reviewing the Mathematical models in the existing literature, these study intends to use Susceptible, Infected, and Removed (SIR) model of infectious disease epidemiology, which was adopted by [9, 20, 26] in order to formulate a mathematical model that describes the dynamics of dengue virus disease transmission.

The populations is divided into two categories, a human population (N_h) and a vector population (N_v). The human population (N_h) is divided into three compartment:people who may potentially get infected with dengue virus (susceptible; S_h), people who are infected with dengue (infected; I_h) and people who have recovered (removed; R_h). The vector population of mosquitoes (N_v) is divided into two compartment: mosquitoes that may potentially become infected with dengue virus (susceptible; S_v) and mosquitoes that are infected with dengue virus (infected; I_v) and the mosquitos are the carriers of the four viruses that cause Dengue fever but not negatively affected by it, and hence there is no need to consider the recovered mosquito population.

The model assumptions are;

- The model assume that a number of human population have already been infected by the virus while others have not been infected.
- The population is homogeneous with the same probability of transmitting the disease.
- The recruitment rate of both the Human and Mosquito population is equal to the Mortality rate.

- The mosquitoes and human populations birth enter into the susceptible class category.
- The bite rate of an infected mosquito is higher than the suspected mosquito.
- Each of the recovered individual has the possibility of being reinfected and enter the susceptible class category.

3.3 Compartmental Model of the dengue virus transmission.

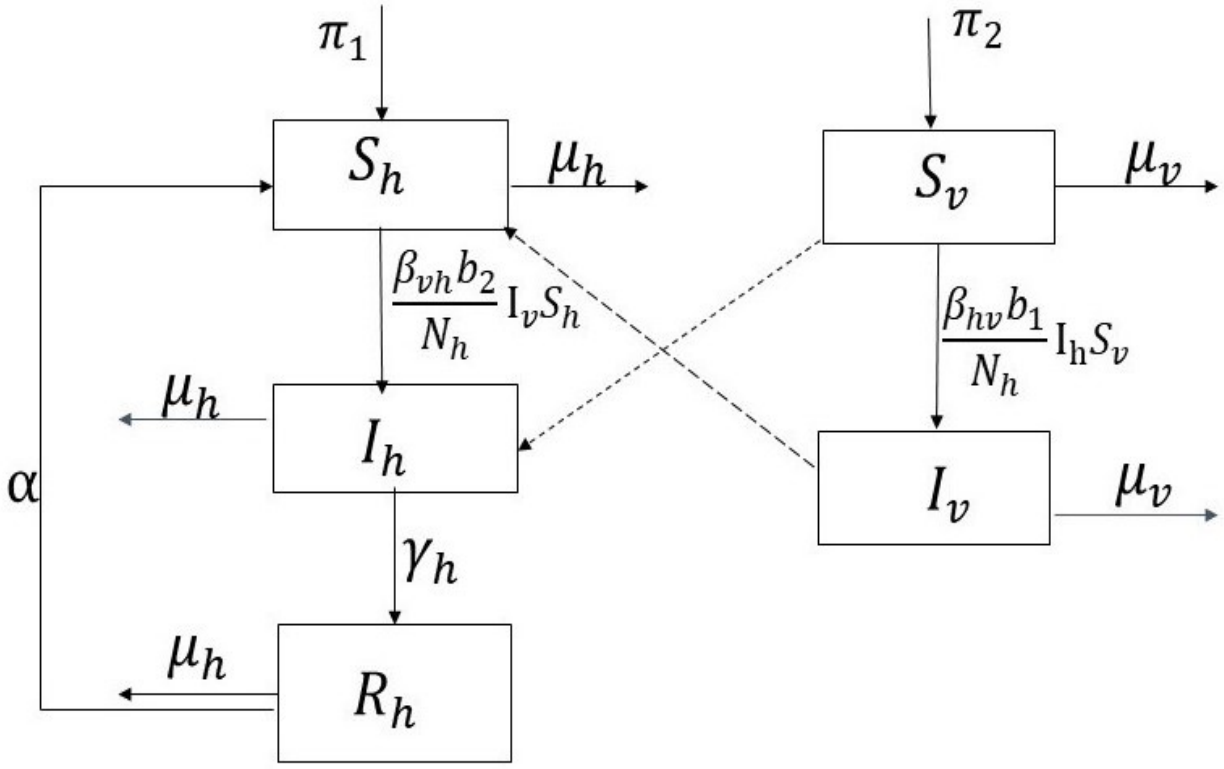

Figure 3. SIR Model diagram of human and Mosquito population

Figure 3 shows the SIR model diagram of human and Mosquito population.

3.4 Model Equation

The Model equation was developed by modifying the model equations used by [26]. The compartmental diagram in Figure 3 can be converted into a Mathematical model based on the interaction between the host and vector model which is in the form of non-linear differential equations as shown below.

$$\frac{dS_h}{dt} = \pi_1 - \frac{\beta_{v_h} b_2}{N_h} I_v S_h - \mu_h S_h + \alpha R_h \tag{11a}$$

$$\frac{dI_h}{dt} = \frac{\beta_{v_h} b_2}{N_h} I_v S_h - (\mu_h + \gamma_h) I_h \tag{11b}$$

$$\frac{dR_h}{dt} = \gamma_h I_h - (\mu_h + \alpha) R_h \tag{11c}$$

The vector population model becomes;

$$\frac{dS_v}{dt} = \pi_2 - \frac{\beta_{h_v} b_1}{N_h} I_h S_v - \mu_v S_v \tag{12a}$$

$$\frac{dI_v}{dt} = \frac{\beta_{h_v} b_1}{N_h} I_h S_v - \mu_v I_v \tag{12b}$$

with the condition;

$$S_h + I_h + R_h = N_h \tag{13a}$$

$$S_v + I_v = N_v \tag{13b}$$

The model for the human and Mosquito population can be combined to form Equations (14a), (14b),(14c),(14d) and (14e) as shown below,

$$\frac{dS_h}{dt} = \pi_1 - \frac{\beta_{v_h} b_2}{N_h} I_v S_h - \mu_h S_h + \alpha R_h \tag{14a}$$

$$\frac{dI_h}{dt} = \frac{\beta_{v_h} b_2}{N_h} I_v S_h - (\mu_h + \gamma_h) I_h \tag{14b}$$

$$\frac{dR_h}{dt} = \gamma_h I_h - (\mu_h + \alpha) R_h \tag{14c}$$

$$\frac{dS_v}{dt} = \pi_2 - \frac{\beta_{h_v} b_1}{N_h} I_h S_v - \mu_v S_v \tag{14d}$$

$$\frac{dI_v}{dt} = \frac{\beta_{hv} b_1}{N_h} I_h S_v - \mu_v I_v \tag{14e}$$

3.5 Explanation of the Model Variable and Parameters

- For the human population:
  The rate of change in the total host population which may easily be infected over the time due to host population birth rate is π_1. Deaths of the susceptible host are represented by $\mu_h S_h$. The rate of change in the number of the infected host depends on the

host infected population. A death among the infected host population is represented by $\mu_h I_h$, while members of the host population that recover their health after infection is $\gamma_h I_h$. In addition, the total host population that has recovered R_h will change according to changing times. The rate of changes for a healthy population is the difference between the host that has recovered from an infection $\gamma_h I_h$ with the total death in the number of healthy host population $\mu_h R_h$

- For the Mosquito population:
  The rate of change in the total Mosquito population which may easily be infected over time due to mosquito population birth rate is given by π_2 The number of deaths among the susceptible mosquito population is $\mu_v S_v$, while $\mu_v I_v$ is the total mortality of the infected mosquito population at any given time.

  Table 1 shows the definition of variables of the dengue virus model while Table 2 shows the definition of the model parameters.

Variable	Description
S_h	Susceptible human at time t
I_h	Infected human at time t
R_h	Recovered Human at time t
S_v	Susceptible Mosquito at time t
I_v	Infected Mosquito at time t

Table 1. Variable defination

Model Parameter	Defination	Units
N_h	Total number of Human Population.	Dimensionless
N_v	Total number of Mosquitoes.	Dimensionless
π_1	Human recruitment rate	Time
π_2	Mosquito recruitment rate	Time
μ_h	Death rate for human population	Time
μ_v	Death rate for Mosquito population	Time
γ_h	Recovery rate of of an infected humans.	Time
β_{v_h}	probability of viral transmission from an infected Mosquito to susceptible human.	Time
β_{h_v}	probability of viral transmission from an infected human to a susceptible mosquito	Time
b_1	The bite rate by suceptible mosquito	Time.
b_2	infected mosquito bite rate	Time.
α	Decline rate in human imunity to desease	Time

Table 2. Parameter Definition

3.6 Positivity and Boundedness of a solution

Theorem 3.6.1. *Let $(S_h(t)>0, I_h(t)>0, R_h(t)>0, S_v(t)>0, I_v(t)>0)$ be the solution of the Model Equations* (14a),(14b),(14c),(14d) *and* (14e) *on the compact set*
$\omega = \left\{ (S_h, I_h, R_h, S_v, I_v) \in \mathbb{R}^5_+, \omega_h = S_h + I_h + R_h \leq N_h \leq \frac{\pi_1}{\mu_h}, \omega_v = S_v + I_v \leq N_v \leq \frac{\pi_2}{\mu_v} \right\}$.
for all t>0. We show that all the feasible solutions are uniformly bounded in a proper subset $\omega = \omega_h + \omega_v$.Thus the set ω is positively invariant.

Proof. In order to show that the solutions are uniformly bounded in a proper subset ω,the model Equation (14a),(14b),(14c),(14d) and (14e) are divided into the human compartment N_h, and the mosquito compartment N_v

We let $\omega_h = \left\{ (S_h, I_h, R_h) \in \mathbb{R}^3_+ \right\}$ be the solution of the system of the Model Equation (14a),(14b),(14c),(14d) and (14e)

Obtaining the derivative of N_h along a solution path of the model Equations (14a),(14b),(14c),(14d) and (14e) gives

$$\frac{dN_h}{dt} \leq \pi_1 - \mu_h(S_h + I_h + R_h) \tag{15a}$$

Simplifying Equation (15a) by using (13a) we obtain

$$\frac{dN_h}{dt} + \mu_h N_h \leq \pi_1 \tag{16a}$$

The integrating factor for Equation (16a) is $e^{\int \mu_h dt}$

Multiplying both sides of Equation (16a) by $e^{\int \mu_h dt}$

$$e^{\int \mu_h dt} \frac{dN_h}{dt} + \mu_h N_h e^{\int \mu_h dt} \leq \pi_1 e^{\int \mu_h dt} \tag{17a}$$

We obtain;

$$\frac{d}{dt}(N_h e^{\int \mu_h dt}) \leq \pi_1 e^{\int \mu_h dt} \tag{18a}$$

Integrating both sides of Equation (17a) we have;

$$N_h e^{\int \mu_h dt} \leq \frac{\pi_1}{\mu_h} e^{\int \mu_h dt} + C \tag{19a}$$

C is the constant of integration. if Equation (19a) is divided by $e^{\int \mu_h dt}$

We obtain;

$$N_h \leq \frac{\pi_1}{\mu_h} + Ce^{\int -\mu_h dt} \tag{20a}$$

By using the initial condition t=0, $N_h(0) = N_{ho}$ we get

$$N_{ho} - \frac{\pi_1}{\mu_h} \leq C \tag{21}$$

Equating for the value of C obtained in (21) into Equation (20a) we get;

$$N_h \leq \frac{\pi_1}{\mu_h} + (N_{ho} - \frac{\pi_1}{\mu_h})e^{\int -\mu_h dt} \tag{22}$$

By applying differential inequality theorem we obtain;

$0 \leq N_h \leq \frac{\pi_1}{\mu_h}$ as $t \to \infty$

This shows that N_h is bounded and all the feasible solutions of the human component of the system of Equations (14a),(14b),(14c),(14d) and (14e) of the dengue fever model starting in the region ω_h approach,enter or stay in the region where;

$$\omega_h = \left\{ (S_h, I_h, R_h) \in \mathbb{R}^3_+ : N_h \leq \frac{\pi_1}{\mu_h} \right\}$$

Similarly the feasible solution set for the mosquito population

$$\omega_v = \left\{ (S_v, I_v) \in \mathbb{R}^2_+ : N_v \leq \frac{\pi_2}{\mu_v} \right\}$$

It follows from above that N_h and N_v are positively bounded and all the possible solutions of the model starting in ω will stay in the region ω = ω_h x ω_v for all t >0.Thus ω is positively invariant and thus the system of Equation (14a),(14b),(14c),(14d) and (14e) of the dengue fever model is biologically meaningful and mathematically well posed in the domain ω □

3.7 Stability Analysis

3.7.1 Desease Free Equilibrium

Total human population is given by

$$N_h = S_h + I_h + R_h \tag{23}$$

Therefore on simplifying we obtain;

$$\frac{dN_h}{dt} = \pi_1 - \mu_h N_h \tag{24a}$$

and the Mosquito population N_v calculated as

$$N_h = S_v + I_v \tag{25}$$

On simplification we obtain;

$$\frac{dN_v}{dt} = \pi_2 - \mu_v N_v \tag{26a}$$

The desease free equilibrium is obtained by setting the infectious and recovered classes equal to zero (I_h,R_h,I_V) and substitute in the model of dengue fever.

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dR_h}{dt} = \frac{dS_v}{dt} = \frac{dI_v}{dt} = 0 \tag{27}$$

Thus the system reduces to;

$$\pi_1 - \mu_h S_h = 0 \tag{28a}$$

$$\pi_2 - \mu_h S_v = 0 \tag{29a}$$

Disease free equilibrium(DFE) point is given by;

$$E_0 = (S_h, 0, 0, S_v, 0) = \left(\frac{\pi_1}{\mu_h}, 0, 0, \frac{\pi_2}{\mu_v}, 0\right) \tag{30}$$

3.7.2 The Basic Reproduction number (R_0)

R_0 is defined as the expected number of secondary cases produced by a single (typical) infection in a completely susceptible population [1]. The next generation matrix is a systematic way to calculate R_0. R_0 is the spectral radius of the next generation matrix. We calculate the basic reproduction of the system using the next generation operator approach. To achieve that we form an equations using the infected class category from the systems of the model of the dengue fever as shown in Equation (31a) and (31b) below;

$$\frac{dI_h}{dt} = \frac{\beta_{v_h} b_2}{N_h} I_v S_h - (\mu_h + \gamma_h) I_h \tag{31a}$$

$$\frac{dI_v}{dt} = \frac{\beta_{h_v} b_1}{N_h} I_h S_v - \mu_v I_v \tag{31b}$$

We define the vector valued function f as the rate of appearance of new infection in the human and vector model

$$f = \begin{bmatrix} \frac{\beta_{v_h} b_2}{N_h} I_v S_h \\ \frac{\beta_{h_v} b_1}{N_h} I_h S_v \end{bmatrix} \tag{32}$$

Evaluating the Jacobian matrix F for the rate of apperance of the new infection at the the disease free equilibrium gives;

$$F = \begin{bmatrix} 0 & \frac{\beta_{v_h} b_2}{N_h} S_h \\ \frac{\beta_{h_v} b_1}{N_h} S_v & 0 \end{bmatrix} \tag{33}$$

$$F = \begin{bmatrix} 0 & \frac{\beta_{v_h} b_2}{N_h} \frac{\pi_1}{\mu_h} \\ \frac{\beta_{h_v} b_1}{N_h} \frac{\pi_2}{\mu_v} & 0 \end{bmatrix} \tag{34}$$

The transfer of individual out of an infectious class is given by;

$$v = \begin{bmatrix} (\mu_h + \gamma_h) I_h \\ \mu_v I_v \end{bmatrix} \tag{35}$$

The Jacobian Matrix for the transfer of individual out of an infections class is given us;

$$V = \begin{bmatrix} (\mu_h + \gamma_h) & 0 \\ 0 & \mu_v \end{bmatrix} \tag{36}$$

Getting the inverse of V gives;

$$V^- = \frac{1}{\mu_v(\mu_h + \gamma_h)} \begin{bmatrix} \mu_v & 0 \\ 0 & (\mu_h + \gamma_h) \end{bmatrix} \tag{37}$$

$$V^- = \begin{bmatrix} \frac{1}{\mu_h + \gamma_h} & 0 \\ 0 & \frac{1}{\mu_v} \end{bmatrix} \tag{38}$$

$$FV^- = \begin{bmatrix} 0 & \frac{\beta_{v_h} b_2 \pi_1}{N_h \mu_h \mu_v} \\ \frac{\beta_{h_v} b_1 \pi_2}{N_h \mu_v (\mu_h + \gamma_h)} & 0 \end{bmatrix} \tag{39}$$

The Basic reproduction number R_0 is the equal to the spectral radius(the greatest eigenvalue) of FV^-

$$\begin{bmatrix} 0-\lambda & \frac{\beta_{v_h} b_2 \pi_1}{N_h \mu_h \mu_v} \\ \frac{\beta_{h_v} b_1 \pi_2}{N_h \mu_v (\mu_h + \gamma_h)} & 0-\lambda \end{bmatrix} \tag{40}$$

Hence;

$$\lambda^2 - \frac{\beta_{v_h} b_2 \pi_1 \beta_{h_v} b_1 \pi_2}{N_h^2 \mu_h \mu_v^2 (\mu_h + \gamma_h)} = 0 \tag{41}$$

The greatest eigenvalue will be the positive value of the square root and its the Basic reproduction number R_0;

$$R_0 = \sqrt{\frac{\beta_{v_h} b_2 \pi_1 \beta_{h_v} b_1 \pi_2}{N_h^2 \mu_v^2 \mu_h (\mu_h + \gamma_h)}} \tag{42}$$

R_0 is a measure of the average number of secondary dengue virus infections in human or mosquito population caused by a single infective human or mosquito introduced into an entirely susceptible population. The square root in the computation of the reproduction number means that dengue virus transmission is a two step process where an infected individual to infect another individual a mosquito must transmit the disease and our R_O is given us .

$$R_0 = \frac{\beta_{v_h} b_2 \pi_1 \beta_{h_v} b_1 \pi_2}{N_h^2 \mu_v^2 \mu_h (\mu_h + \gamma_h)} \tag{43}$$

3.8 Local Stability analysis of the Disease free equilibrium point

Theorem 3.8.1. *The disease free equilibrium is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$

Proof. The Jacobian matrix for the Model Equation (14a),(14b),(14c),(14d) and (14e) is given by;

$$J = \begin{bmatrix} \frac{-\beta_{v_h} b_2 I_v}{N_h} - \mu_h & 0 & \alpha & 0 & \frac{-\beta_{v_h} b_2 S_h}{N_h} \\ \frac{\beta_{v_h} b_2 I_v}{N_h} & -(\mu_h + \alpha) & 0 & 0 & \frac{\beta_{v_h} b_2 S_h}{N_h} \\ 0 & \gamma_h & -(\mu_h + \alpha) & 0 & 0 \\ 0 & \frac{-\beta_{h_v} b_1 S_v}{N_h} & 0 & \frac{-\beta_{h_v} b_1 I_h}{N_h} - \mu_v & 0 \\ 0 & \frac{\beta_{h_v} b_1 S_v}{N_h} & 0 & \frac{\beta_{h_v} b_1 I_h}{N_h} & -\mu_v \end{bmatrix} \tag{44}$$

By evaluating the Jacobin Matrix at the disease free equilibrium values given by $E_0 = \left(\frac{\pi_1}{\mu_h}, 0, 0, \frac{\pi_2}{\mu_v}, 0\right)$, We obtain the matrix as shown below.

$$J = \begin{bmatrix} -\mu_h & 0 & \alpha & 0 & -\frac{\beta_{v_h} b_2 \pi_1}{N_h \mu_h} \\ 0 & -\mu_h - \gamma_h & 0 & 0 & \frac{\beta_{v_h} b_2 \pi_1}{N_h \mu_h} \\ 0 & \gamma_h & -(\mu_h + \alpha) & 0 & 0 \\ 0 & \frac{-\beta_{h_v} b_1 \pi_2}{N_h \mu_v} & 0 & -\mu_v & 0 \\ 0 & \frac{\beta_{h_v} b_1 \pi_2}{N_h \mu_v} & 0 & 0 & -\mu_v \end{bmatrix} \tag{45}$$

We then compute the eigenvalues of the matrix evaluated at J(E_0)

$$|J(E_0) - \lambda I| = |(-\mu_h - \lambda)| \begin{bmatrix} -\mu_h - \gamma_h - \lambda & 0 & 0 & \frac{\beta_{v_h} b_2 \pi_1}{N_h \mu_h} \\ \gamma_h & -\mu_h - \alpha - \lambda & 0 & 0 \\ \frac{-\beta_{h_v} b_1 \pi_2}{N_h \mu_v} & 0 & -\mu_v - \lambda & 0 \\ \frac{\beta_{h_v} b_1 \pi_2}{N_h \mu_v} & 0 & 0 & -\mu_v - \lambda \end{bmatrix} | \tag{46}$$

$$(-\mu_h - \lambda)(-\mu_v - \lambda)(-\mu_h - \alpha - \lambda)((-\mu_h - \gamma_h - \lambda)(-\mu_v - \lambda) - \frac{\beta_{v_h} b_2 \pi_1}{N_h \mu_h} \frac{\beta_{h_v} b_1 \pi_2}{N_h \mu_v} = 0 \tag{47a}$$

The eigenvalues obtained from Equation (47a) is

$$\lambda_1 = -\mu_h \tag{48a}$$
$$\lambda_2 = -\mu_v \tag{48b}$$
$$\lambda_3 = -\mu_h - \alpha \tag{48c}$$

and the eigenvalue Equation is

$$\lambda^2 + (\mu_h + \mu_v + \gamma_h)\lambda + (\mu_h + \gamma_h)\mu_v - \frac{\beta_{v_h} b_2 \pi_1}{N_h \mu_h} \frac{\beta_{h_v} b_1 \pi_2}{N_h \mu_v} = 0$$

$$\lambda^2 + (\mu_h + \mu_v + \gamma_h)\lambda + (\mu_h + \gamma_h)\mu_v (1 - \frac{\beta_{v_h} b_2 \pi_1}{N_h \mu_h} \frac{\beta_{h_v} b_1 \pi_2}{N_h \mu_v} \frac{1}{\mu_v(\mu_h + \gamma_h)}) = 0$$
$$\lambda^2 + (\mu_h + \mu_v + \gamma_h)\lambda + (\mu_h + \gamma_h)\mu_v (1 - R_0) = 0 \tag{49}$$

From the eigenvalue Equation (49) we obtain eigenvalues 4 and 5 as shown in Equation (50)

$$\lambda_{4,5} = \frac{-(\mu_h + \mu_v + \gamma_h) \pm \sqrt{(\mu_h + \mu_v + \gamma_h)^2 - 4(\mu_h + \gamma_h)\mu_v (1 - R_0)}}{2} \tag{50}$$

We analyse the sign of eigenvalues, since all the eigenvalues are negative as shown in Equation (48a),(48b) and (48c) and when $R_0 < 1$ the real part of eigenvalues 4 and 5 in Equation (50) are negative and when $R_0 > 1$ we have an eigenvalue with positive real part hence the disease free equilibrium point is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$ This proves theorem 3.8.1. □

3.9 Global Stability of the Disease free equilibrium point

Theorem 3.9.1. *if* $R_0 < 1$ *then the free disease equilibrium* $E_0 = (S_h^*, 0, 0, S_v^*, 0) = \left(\frac{\pi_1}{\mu_h}, 0, 0, \frac{\pi_2}{\mu_v}, 0\right)$ *in the global stage is asymptotically stable in the set* ω

Proof. A Lyapunov functions constructed for the system is

$$M(t) = (S_h - S_h^* \ln S_h) + I_h + R_h + (S_v - S_V^* \ln S_v) + I_v \tag{51}$$

The derivative of M(t) with respect to time that satisfies Equation (51) is

$$M(t)^{'} = S_h^{'}(1-\frac{S_h^*}{S_h}) + I_h^{'} + R_h^{'} + S_v^{'}(1-\frac{S_v^*}{S_v}) + I_v^{'} \tag{52}$$

$$= \pi_1 - \frac{\beta_{v_h} b_2}{N_h} I_v S_h - \mu_h S_h + \alpha R_h (1-\frac{S_h^*}{S_h}) \tag{53}$$

$$+\frac{\beta_{v_h} b_2}{N_h} I_v S_h - (\mu_h + \gamma_h) I_h + \gamma_h I_h - (\mu_h + \alpha) R_h \tag{54}$$

$$+\pi_2 - \frac{\beta_{h_v} b_1}{N_h} I_h S_v - \mu_v S_v (1-\frac{S_v^*}{S_v}) + \frac{\beta_{hv} b_1}{N_h} I_h S_v - \mu_v I_v \tag{55}$$

$$= \pi_1 (1-\frac{S_h^*}{S_h}) + \mu_h S_h^* (1-\frac{S_h}{S_h^*}) - \frac{\beta_{v_h} b_2}{N_h} I_v S_h \tag{56}$$

$$+\frac{\beta_{v_h} b_2}{N_h} I_v S_h (\frac{S_h^*}{S_h}) - \alpha R_h (\frac{S_h^*}{S_h}) + + \frac{\beta_{v_h} b_2}{N_h} I_v S_h - \mu_h I_h \tag{57}$$

$$-\mu_h R_h + \pi_2 (1-\frac{S_v^*}{S_v}) + \mu_v S_v^* (1-\frac{S_v}{S_v^*}) - \frac{\beta_{v_h} b_2}{N_h} I_v S_h \tag{58}$$

$$+\frac{\beta_{hv} b_1}{N_h} I_h S_v (\frac{S_v^*}{S_v}) + \frac{\beta_{hv} b_1}{N_h} I_h S_v - \mu_v I_v \tag{59}$$

$$= \pi_1 (1-\frac{S_h^*}{S_h}) + \mu_h S_h^* (1-\frac{S_h}{S_h^*}) \tag{60}$$

$$-\alpha R_h (\frac{S_h^*}{S_h}) - \mu_h R_h + \pi_2 (1-\frac{S_v^*}{S_v}) \tag{61}$$

$$+\mu_v S_v^* (1-\frac{S_v}{S_v^*}) \tag{62}$$

$$+(\frac{\beta_{vh} b_1}{N_h} S_v^* - \mu_h) I_h + (\frac{\beta_{hv} b_2}{N_h} S_h^* - \mu_v) I_v \tag{63}$$

Considering $S_h^* = \dfrac{\pi_1}{\mu_h}$ and $S_v^* = \dfrac{\pi_2}{\mu_v}$ Equation (52) can be compressed as;

$$M(t)' = \pi_1 (2 - \frac{S_h^*}{S_h} - \frac{S_h}{S_h^*}) - \alpha R_h (\frac{S_h^*}{S_h}) - \mu_h R_h + \pi_2 (2 - \frac{S_v^*}{S_v} - \frac{S_v}{S_v^*}) \tag{64a}$$

$$= -\pi_1 \frac{(S_h - S_h^*)^2}{S_h S_h^*} - \alpha R_h - \pi_2 \frac{(S_v - S_v^*)^2}{S_v S_v^*} - \mu_h R_h \tag{64b}$$

Equation (64) shows that $M(t)' \leq 0$ using Lyapunov method , the finite sets applicable for the solutions are those contained in the largest invariant set where $S_h = S_h^*$, $R_h = 0$, $S_v = S_v^*$ that is the singletone set $\{(S_h^*, I_h^*, R_h^*, S_v^*, I_v^*)\}$. This implies that the disease free equilibrium is globaly asymptotically stable in ω. This proves theorem 3.9.1 □

4 Numerical Simulation

4.1 Introduction

In this chapter we carried out the numerical simulations for the dynamics of the state variables. The parameter values used in the simulations were obtained from the literature while total human population was taken from the KNBS population projection of 2011 based on the fact that the outbreak of dengue fever was more severe in Mandera county in the year 2011 and the total mosquito population was used as proxy variables from [6]. The initial values of state variables were then assumed. We used Matlab R2018a to generate the numerical simulations.The model equations are coded in Matlab together with initial values and Parameters and the required output generated by representing the human and vector compartments variables as a ratio of their populations respectively.The findings were then discussed based on the results.

4.2 Variables/Parameter Values

Table 3 below shows the initial values of the state variables and parameters values used in the model.The source of the initial values of Model parameters as obtained from the literature was cited.

Model Variables/Parameters	Initial Values	Source
N_h	1,165860.	KNBS-2011 population projection
S_h	1,165859	Assumed
I_h	1	Assumed
R_h	0	Assumed
N_v	600	[6]
S_v	599	Assumed
I_v	1	Assumed
π_1	46.63	Computed
π_2	150	Computed
μ_h	0.00004	[3]
μ_v	0.25	[3]
γ_h	0.2857.	WHO-2021
β_{v_h}	0.75	[3]
β_{h_v}	0.75	[3]
b_1	0.5	[3]
b_2	1	[3]
α	0.575000	[26]

Table 3. Variables/Parameter Values

4.3 Sensitivity Analysis of the Basic Reproduction Number

In this section we study how variation of the parameters in the expression of basic reproduction number can contribute to the change in the value of the basic reproduction number hence affecting the spread of the disease. By definition, if we denote basic reproduction number by R_0 , then the sensitivity is given by the scaled partial derivatives with respect to the parameters appearing in the expression of R_0 . That is,

$$s_p = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0} \tag{65}$$

where p is any parameter appearing in the expression of R_0 and the scaling product $\frac{p}{R_0}$ is a normalization for the sensitivity s_p. From the basic reproduction number which we have

obtained, the parameters appearing in its expression are $\beta_{v_h}, b_2, \pi_1, \beta_{h_v}, b_1, \pi_2, N_h^2, \mu_v^2,$ and μ_h. We carry out sensitivity analysis of these parameters to study the impact of their variations in the spread of the disease. Starting with β_{v_h}, we have

$$\begin{aligned} s_{\beta_{v_h}} &= \frac{\partial R_0}{\partial \beta_{v_h}} \frac{\beta_{v_h}}{R_0} \\ &= \frac{b_2 \pi_1 \beta_{h_v} b_1 \pi_2}{N_h^2 \mu_v^2 \mu_h (\mu_h + \gamma_h)} \beta_{v_h} \frac{N_h^2 \mu_v^2 \mu_h (\mu_h + \gamma_h)}{\beta_{v_h} b_2 \pi_1 \beta_{h_v} b_1 \pi_2} \\ &= 1 \end{aligned} \tag{66}$$

This implies that, a unit increase in the contact rate (β_{v_h}) would lead to a linear increase in the value of the basic reproduction number.

Similarly, we obtain that $s_{b_2} = s_{\pi_1} = s_{\beta_{hv}} = s_{b_1} = s_{\pi_2} = 1$. This means that, a unit increase of any numerator parameter appearing in the expression of basic reproduction number would lead to a linear increase in the basic reproduction number and thus increasing the transmission of the disease. Similarly, a unit decrease in these values would lead to linear decrease in the value of basic reproduction number and thus lowering the spread of the disease.

Now, we perform sensitivity analysis of the denominator parameters appearing in the expression of the basic reproduction number. We have;

$$\begin{aligned} s_{N_h} &= -2 \frac{\beta_{v_h} b_2 \pi_1 \beta_{h_v} b_1 \pi_2}{N_h^3 \mu_v^2 \mu_h (\mu_h + \gamma_h)} N_h \frac{N_h^2 \mu_v^2 \mu_h (\mu_h + \gamma_h)}{\beta_{v_h} b_2 \pi_1 \beta_{h_v} b_1 \pi_2} \\ &= -2 \end{aligned} \tag{67}$$

This means that a decrease in the population size of humans by 2 units would lead to increased value of the basic reproduction number. Similarly, $s_{\mu_v^2} = -2$, meaning that is decrease would lead to increased value of the basic reproduction number.

$$\begin{aligned} s_{\mu_h} &= -\frac{\beta_{v_h} b_2 \pi_1 \beta_{h_v} b_1 \pi_2}{N_h^2 \mu_v^2 \mu_h^2 (2\mu_h + \gamma_h)} \mu_h \frac{N_h^2 \mu_v^2 \mu_h (\mu_h + \gamma_h)}{\beta_{v_h} b_2 \pi_1 \beta_{h_v} b_1 \pi_2} \\ &= -\frac{\mu_h + \gamma_h}{2\mu_h + \gamma_h} \end{aligned} \tag{68}$$

This implies that decreasing μ_h which lead to increased basic reproduction number leading to more infections.

$$s_{\gamma_h} = \frac{\beta_{v_h} b_2 \pi_1 \beta_{h_v} b_1 \pi_2}{N_h^2 \mu_v^2 \mu_h (\mu_h - \gamma_h^2)} \gamma_h \frac{N_h^2 \mu_v^2 \mu_h (\mu_h + \gamma_h)}{\beta_{v_h} b_2 \pi_1 \beta_{h_v} b_1 \pi_2} \tag{69}$$

$$= \gamma_h \frac{\mu_h + \gamma_h}{\mu_h - \gamma_h^2} \tag{70}$$

This means that an increase in γ_h will lead to an increase in the basic reproduction number.

The bar graph in Figure 4 shows the sensitivity index of the parameters affecting the basic reproduction number(R_0).

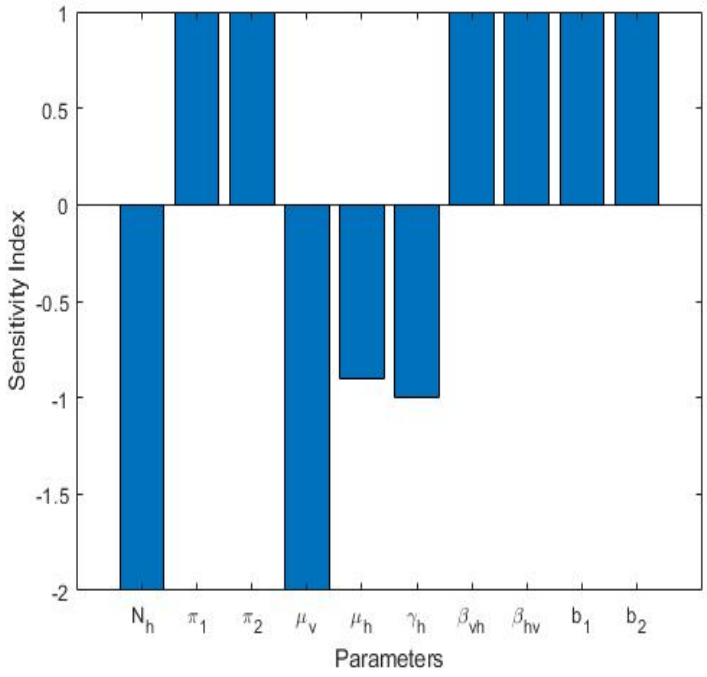

Figure 4. Sensitivity index against Parameters of the Basic Reproduction numbers.

4.4 Simulations of model state variables

The simulations on Figure 6,7,8,10 and 11 shows the variation of the model state variables for a period of 100 days upon which the equilibrium point is achieved.

4.5 Discussions

The graph given in Figure 5 shows the dynamics of transmission of dengue virus disease in population. It shows the rate at which susceptible humans decrease over time due to

infections. As the susceptible humans decline in number, infected individuals increase, and the recovered individuals increases simultaneously. Initially, we have only 1 infected individual, this individual starts spreading the disease in the population un-noticeably, however, when the number of infectious individuals start to increase in the population, we are clearly able to see how susceptible humans reduce rapidly because we have more people who are infectious. Having high number of infected individuals in the population increases the transmission of dengue virus disease because the chances of vector coming into contact with infected individuals are increased.As humans get infected, after some times they recover. This explains why the rate of recovered humans increased over time.Since we assumed demographic turn over, after a while we obtain an endemic equilibrium, where the number of susceptible individuals, infected, and recovered individuals remain constant as time changes

As the number of susceptible Mosquito come into contact with infected human the susceptible mosquito get infected and decrease over time as shown in Figure 10 this expains why the rate of infected mosquitos increases over time as shown in Figure 11

Figure 6 shows separately what happens to the susceptible humans over time, Figure 7 shows the dynamics of the infected humans over time, how initially they increase, then after an outbreak we have the equilibrium. Figure 8 shows the dynamics of the recovered individuals. Initially no one is recovered from the disease. After sometime, when infections start to increase, more people start recovering. They increase until we obtain an equilibrium.

Figure 9 shows the dynamics of the vectors. The number of infected individuals depends on the number of infectious vectors. When we have high number of infectious vectors, we have higher number of infected humans and vise verser.

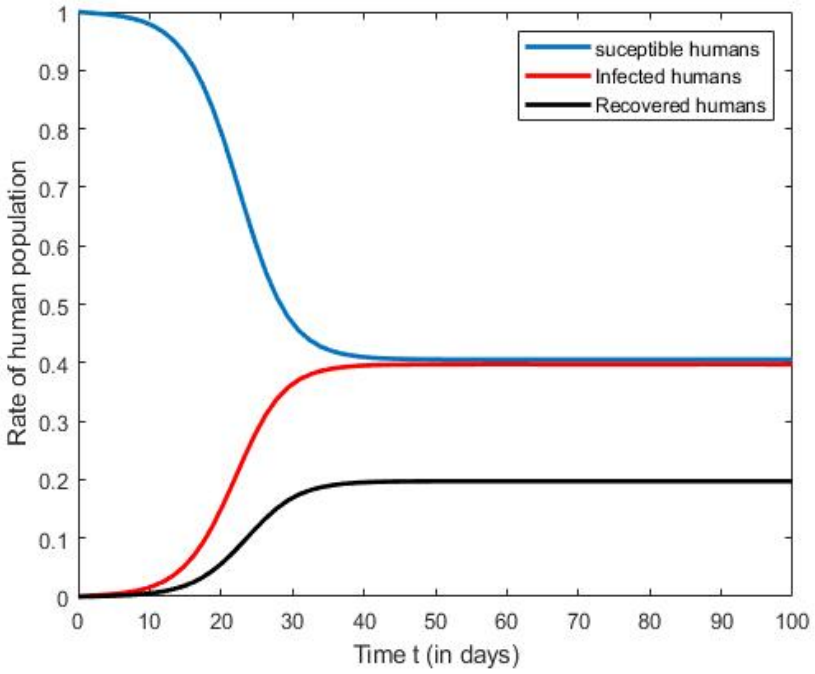

Figure 5. Rate of Susceptible, infected and recovered Human Population over time

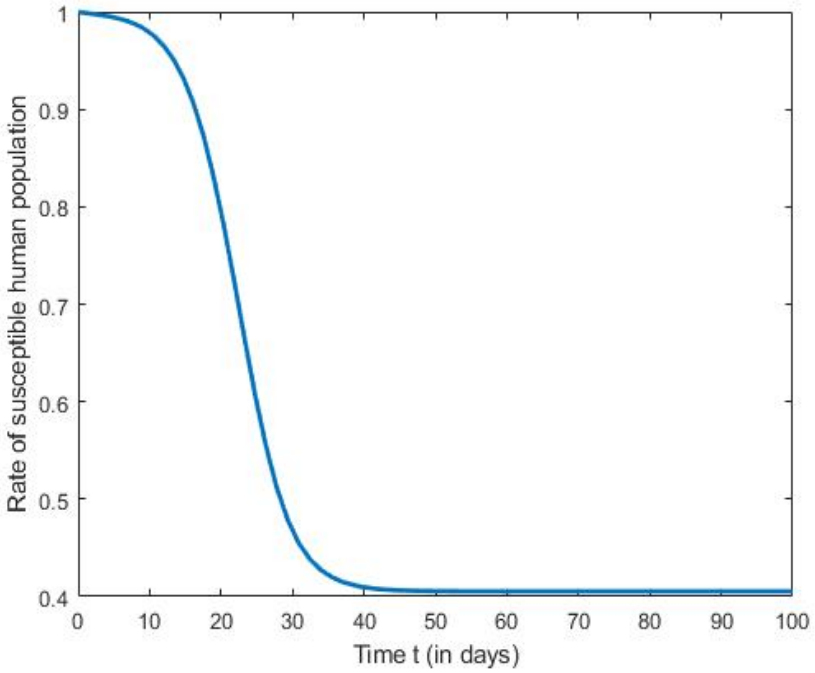

Figure 6. Rate of Susceptible Human Population with time

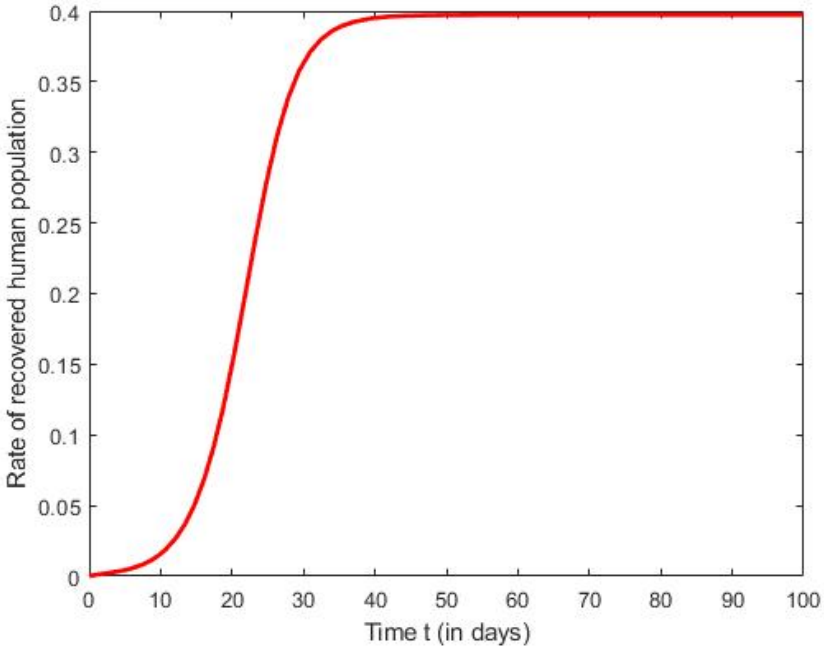

Figure 7. Rate of Infected Human population over time

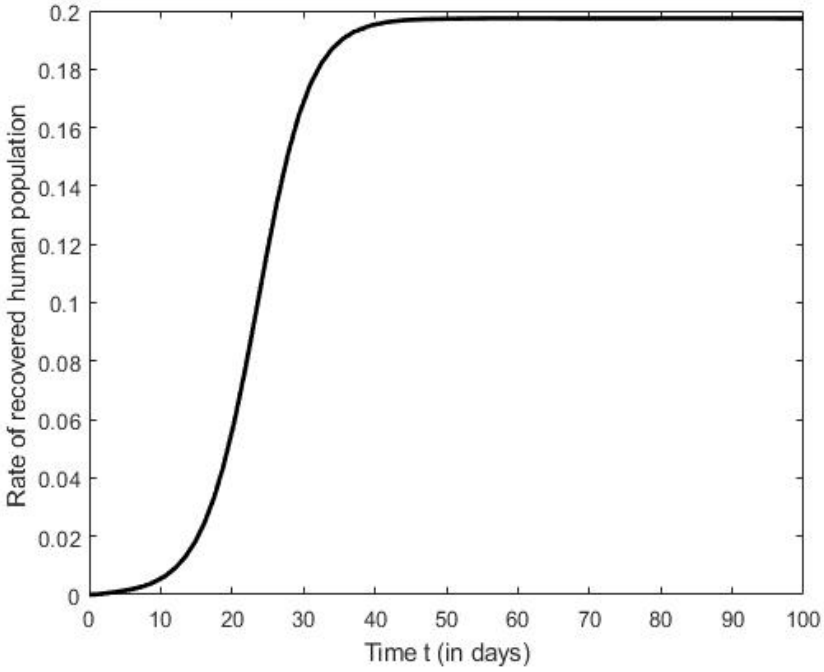

Figure 8. Variation of recovered human population over time

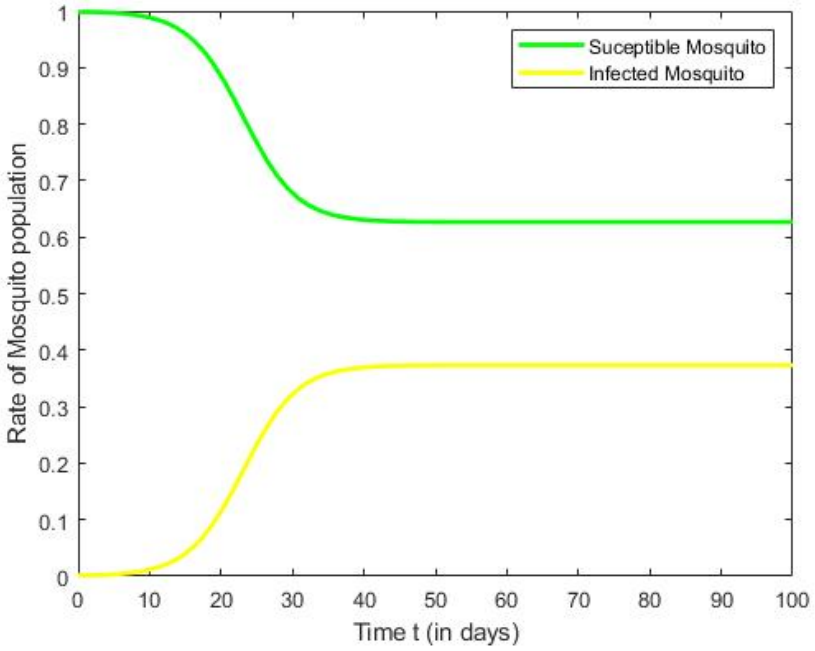

Figure 9. Variation of susceptible and Infected vector over time

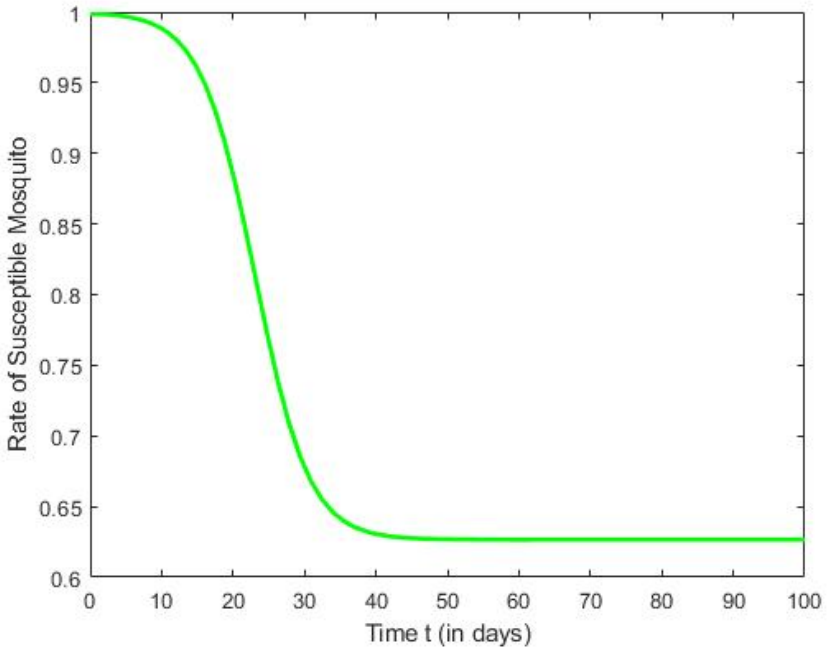

Figure 10. Variation of susceptible vector over time

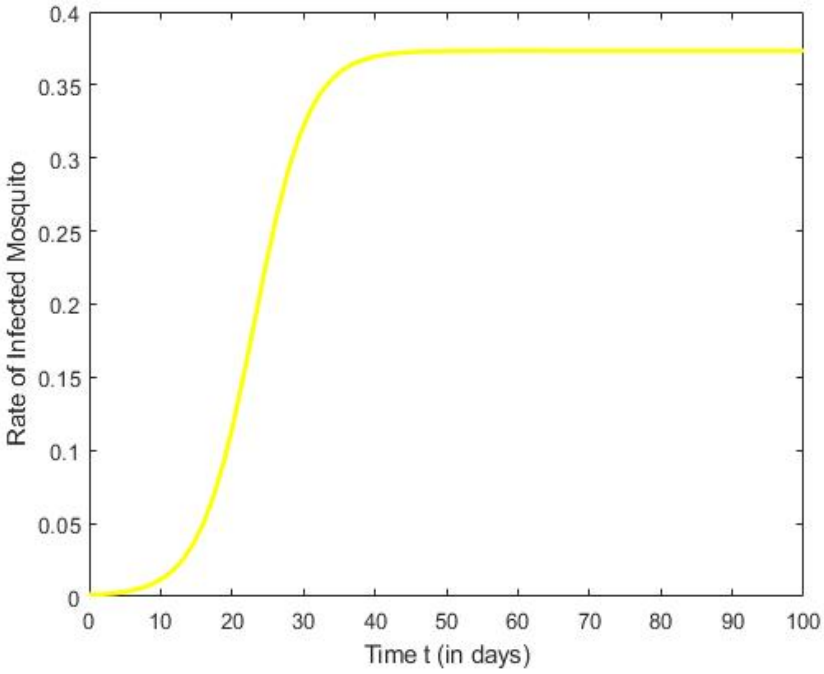

Figure 11. Variation of infected vector over time

5 Conclusion & Recommendation

5.1 Introduction

In this chapter the conclusions are obtained from the findings and the recomendation therein given at the end of the chapter. The recommendations will include policy and further research recommendations.

5.2 Conclusion

In this study a mathematical model for the dynamics of dengue virus disease transmission is formulated and analysed at the disease free equilibrium points. We derived the basic reproduction number using the next generation matrix as the dominant eigenvalue of the Jacobian matrix of the infectious clases of Infected humans and infected vectors It was discovered that when $R_0 < 1$ then the disease free equilibrium is both locally and globally stable.

Numerical analysis shows that the rate of susceptible humans to dengue fever infections decreases from 1 and attain equilibrium when they attain the rate of 0.4. The decline is attributed to the number of susceptible becoming infected with the dengue virus.The increase in infected humans is due new infections on susceptible humans, which leads to their decrease in number because they are added to the infectious stage. In addition, the growth of the infected vectors as well as recovered humans becoming susceptible contribute to this increase since we assumed the bite rate by an infected mosquito is higher than the susceptible mosquito, the infected human attain equilibrium when they attain the rate of 0.4.

The increase in the number of recovered humans is as result of the infected human leaving the infectious stage after some duration and Joining the recovered class, where we assumed that recovery rate is higher than natural mortality of the human population.

The decrease in susceptible mosquitoes is as result of an infection by the the infected humans. As the infected human increases, more susceptible mosquitoes come into contact with infected humans which makes them get infections and this explains why the graph of infected vectors increases.

Therefore, in conclusion, since we have seen that the number of infections depend on the interactions between mosquitoes and human beings, measures need to be put into place

to control these interactions. Some of the measures that can be put into place include ensuring that everyone sleeps under treated mosquito net, or reducing the population of the mosquitoes by constructing good drainage systems which would ensure that mosquitoes have no breading sites, hence leading to the decline of their population.

5.3 Recommendation

Dengue can be seen to pose a challenge in Mandera, Northern part of Kenya. Thus their is need to safeguard against Mosquito by sleeping under the treated mosquito net since the disease is spread by Mosquito, In addition a proper drainage system need to be put in place to allow the rain water to follow so as to mitigate against swampy areas where the mosquito breads.

5.4 Future Work

Accurately projecting the future of dengue under the context of climate change would help governments and public health officials take timely and preemptive actions to protect the public from dengue in the future.

There is a greater need to look at more prevention measures and control measures while clearly educating the public on the same. Furthermore, there should be in-communication between the reseachers and policy makers to ensure that there is more positive goodwill even the public domain.There is also a need for the study to be carried out to study the impact of the implemented control measures in the spread of the disease.

There is need for researchers to collect primary data from the field and draw accurate results based on the data collected .There is need to also look into to other demographic parameters such as age structure and population density as well as other regions of Kenya where dengue infection is prevalent.

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