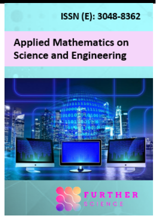
Applied Mathematics on Science and Engineering, 2(1):57-82, 2025.

Applied Mathematics on Science and Engineering

Journal homepage: https://furthersci.org/journal-details.php?journal-id=49



Research Article

Optimizing Control Measures for a Vector-Host Epidemic Model: A Mathematical Analysis

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Article Info

Keywords: *Malaria, Rift Valley Fever, Dengue Fever, West Nile Fever, vector-host diseases, Pontryagin's Maximum Principle, Optimality conditions.*

Received: *25.03.2025*
Accepted: *03.06.2025*
Published: *22.06.2025*

Abstract

Malaria, Rift Valley Fever, Dengue Fever and West Nile Fever are vector-host infections that are endemic among most of the populations mainly across the tropical regions. As much as these diseases are treatable and preventable they have proved to be among the greatest attributes to mortality among the populations of the world. This could be attributed to the fact that planning and measures that are proper and timely have not been put in place by the governments.To curb the effects of such diseases mathematical models can be used to study the dynamics of these infections and the effectiveness of various controls towards them including prevention and treatment.

In this paper, we come up with an appropriate system of equations to represent the dynamics of Malaria, Dengue Fever, Rift Valley Fever and West Nile Fever infections. In particular, we have developed a mathematical model for the transmission and control of Dengue Fever, incorporating *prevention* and *treatment* as control parameters. We have further shown that our model has a unique *disease-free* equilirium point which is locally and globally asymptotically stable when $R_0 < 1$. The model also have a unique *endemic equilibrum* point, which is locally and globally asymptotically stable when $R_0 > 1$.

We determined the parameters of the model, using Data from [1] while some of the data was approximated. To determine an optimal combination of *prevention* and *treatment* that effectively reduces the transmission, we formulated an optimal control problem, with an appropriate *cost* function. We then used Pontryagin's Maximum Principle to determine the optimal control functions. Simulations of our model using various combinations of treatment and prevention indicate that infected vector and human populations can be drastically reduced, thus effectively controlling the transmission of Dengue Fever. In addition, effective treatment reduces the infected human population. The results also showed that when more resources are channeled towards the treatment and prevention of vector-host infections then these diseases can easily be eradicated within the shortest time possible. This implies the designing of proper policies and strategies to fully render a population disease-free.

How To Cite: W. Kulei, A.K. Langat, D. Chepkonga and J.K. Mutinda. Optimizing Control Measures for a Vector-Host Epidemic Model: A Mathematical Analysis. *Applied Mathematics on Science and Engineering*, 2(1):57-82, 2025

1. Introduction

According to [2–4], Vectors are living organisms that can transmit infectious diseases between humans or from animals to humans. Vector-host diseases are diseases that are contracted by human beings or other organisms after adequate contact or interaction with vectors for example mosquitoes, birds, ticks, and rodents which carry with them pathogens such as viruses, plasmodia, and bacteria. These types of infections account for at least 17% of all infectious diseases globally and cause more than 700 000 mortalities per annum.
Examples of such diseases include but are not limited to

1. Malaria. This is a deadly disease that is caused by a parasite which mainly infects a given type of mosquitoes that feeds on human blood. Malaria is caused by *Plasmodium* parasite and is attributed to more than 405 000 deaths in the world, [5–7].
2. Dengue fever. This is a mosquito-borne infectious disease found in the tropical and sub-tropical regions in the world. It is the most common viral infection that is transmitted by *Aedes* mosquitoes. In Kenya, Dengue fever is mainly common in the coastal regions. According to WHO (2020), it is estimated that this disease leads to 40 000 deaths per annum with 3.9 billion cases over the world. [Tran et al, 2018] state that there has been a 7-time increase in dengue fever incidences from 8.3 million in the year 1990 to 58.4 million cases in the year 2018. This disease mainly affects the population that occupies the tropical and the sub-tropical regions of the earth [8, 9].
3. Rift Valley Fever. This viral infection, primarily impacting animals, can also be transmitted to humans through infected mosquitoes [10]. According to WHO (2018), the virus was first identified in 1931 in the Kenyan Rift Valley. Afterwards, subsequent infections have been experienced in Sub-Saharan Africa though it is postulated that the disease can eventually invade ecologically diverse regions. Despite this, the virus has never been observed in the Western Hemisphere, and it is feared that introduction into this region could have a significant deleterious impact on human and agricultural health. A worldwide potential outbreak of Rift Valley Fever especially in geographical regions with temperate climate is predicted. This is according to [10, 11]. In Africa, the disease is transmitted by mosquitoes to livestock while some of these mosquito species are infected only directly through feeding on infectious livestock others get infected at birth by vertical transmission.
4. West Nile Fever. It is a rare event for us to see a huge number of dead birds outdoors in an urban setting. However, if one lived in New York City in the summer of 1999, then one would testify to the unusually high number of dead birds found in backyards and parks [12]. Later in the same year, it would have dawned on us that the cause of death was a newly introduced disease, West Nile virus, that was carried by mosquitoes and was killing birds and humans. With those initial reports, we might have begun to ask any number of important questions: How would the disease affect bird populations?; How infectious would it be in humans?; How fast would it spread from New York to other locations?; Would it spread to other animals as well?; Was it carried by all mosquito species? Did they transmit it in every bite? How could the disease be controlled?; Would mosquito spraying help?; Would culling the bird population help?; Some of these questions would best be addressed in field and laboratory studies, others with mathematical modeling, and yet others with both approaches. According to [13, 14], nearly 80% of people who contract this disease are asymptomatic and it causes in humans a fatal neurological disease. For now, we will focus on the key question of how best to control a West Nile Fever outbreak and take advantage of empirical studies to inform and test our mathematical modeling [15–17].

Other vector-host diseases transmitted by mosquitoes include Yellow Fever, Zika Virus Fever, and Chikungunya Fever. Chagas disease, Bilharzia, and tick-borne encephalitis are also vector-borne diseases but are not transmitted by mosquitoes [18].

According to a world brief on vector-borne diseases that was released by WHO in the year 2014, World Health Organization [19], at least 1 billion individuals are infected with vector-borne diseases while more than 1 million of the infected succumb to them [20].

Economic stagnation in Africa is majorly attributed to such kinds of diseases and statistics from a study by [21–24] showed that a Dengue instance leads to 14.8 lost days for outpatients which cost US$514 on average and 18.9 days for the patience who are non-fatal and have been admitted at an average cost burden of US$ 1491.

According to WHO (2020) Malaria required a total funding of US$ 2.7 Billion as of the year 2018.
In Malawi [25], for example, the total annual household cost of treating and preventing malaria is estimated to be 7% of household income while 9-18% of the annual income for small farmers in Kenya is spent in Malaria according to [26]. It is also stated by WHO (2000) that malaria slows down the economic growth in Africa by upto 1.3% per annum.

Mathematical models have played an important role at the understanding of the dynamics of vector-host disease transmission and in the evaluation and comparison of treatment and prevention strategies. These roles have been analyzed by several researchers and authors and so we review some of the mathematical models with intervention that have so far been worked on.

Lourdes [27], worked on the analysis of the transmission model of a dengue disease. Dengue disease is a vector-host disease just like malaria. The model used several hosts as the source of food blood to the vector. They noted that during periods of low transmission, the human population loses interest in mosquito control and this results in the upsurge of the number of mosquitoes. Afeez [28] employed Lyapunov stability analysis and optimization measures in their study on a dengue disease transmission model. They investigated the stability of disease dynamics and explored optimization strategies for controlling the spread of dengue.

Gabriel Otieno [29], wrote a paper that proposed and analyzed mathematical model for transmission dynamics of malaria with four-time dependent control measures in Kenya that included Intermittent Preventive Treatment in Pregnancy (IPTp), Insecticide-Treated Nets (ITNs), Indoor Residual Spraying (IRS), and treatment. They then suggested an optimal control strategy which is hoped to effectively reduce the spread of malaria transmission in Kenya. Gbenga [30] developed a mathematical model for malaria transmission dynamics in humans, incorporating factors such as mobility and control states. Their study, published in Infectious Disease Modelling, delves into understanding how these variables influence the spread of malaria and the effectiveness of control measures.

Bakare E. [31], determined an optimal control of malaria transmission dynamics with seasonality in rainfall in the mosquito birth rate in the presence of multiple control strategies and by the use of Pontryagin's Maximum Principle. This is because they noted that malaria is a major contributor to mortality and morbidity in Nigeria. In Nigeria, Malaria results in 60% outpatients and 30% inpatients. In their study they showed that an optimal control exists for the disease and this led to data simulations and strategy formulations.

Donald [32], developed an optimal control problem to reduce the number of livestock at the final time while minimizing the effects of mosquitoes infected with Rift Valley Fever and the cost of the process of giving out vaccines. They noted that the unique optimal vaccination strategy is obtained for a given large spread rate. In their numerical simulations the results showed that vaccination depended on the level of effectiveness of the protocol to be followed while carrying out vaccination.

Tran [33], looked into the illness' cost and the quality of life with regards to health of the infected in dengue fever outbreak in the year 2017. They noted that a huge cost and acute health degeneration were associated with dengue fever among the sick. This requires health services to be solidly established and more public campaigns to be carried out. All this also posses an economic burden to a government.

Rowthorn [34], through their optimal control study in epidemiological model which factored in economic theory addressed the use of a blend of optimization methods for economic theory with models from the theory of epidemiology that involved meta-populations. They concluded that the control strategy chosen should be determined by key epidemiological indicators such as basic reproduction number and the rate of treatment. Further in [35], they conducted a study in heterogeneous population that experienced symmetric and asymmetric spread of infection. They established that in the asymmetric case it was only optimal if treatment priority was given to the more infectious species and in case the resources were left out then the less infectious species are treated. For the case of symmetric they advised that treatment should be prioritized to those species with a low susceptibility to the infection.

Clearly, many studies have been carried out on concerning vector-host infections that are already endemic within our communities. Having seen from the literature above the cost effects of prevalence of various vector-host diseases, the optimal control with regards to cost of prevention and in other cases treatment, the various studies on symmetric and asymmetric transmission of infections and even the expectation of emergence of such diseases in new areas then it is worth carrying out further studies on how costs can be limited while trying to prevent the spread and effects of such diseases while treating those who are already infected with these diseases.

Therefore, in this work we shall first formulate a model that factors in saturation incidence and from this model a basic reproduction number is evaluated from the equilibrium points of our model and then we shall carry out an optimal control study that will seek to reduce the cost of employing prevention and treatment of such diseases on the population using the Pontryagin's maximum principal. Finally, we seek to use data based in Thailand on Dengue Fever to do numerical simulations which shall be the basis for our conclusion.

2. Data and Model Formulation

We come up with a vector-host model where we have two populations, the host which includes human beings and the vector which includes mosquitoes. We also assume that the sources of food blood for mosquitoes is not only human beings but also other animals such as dogs, chickens,water buffaloes and pigs. At time t, we consider the following compartments

1. Susceptible human host, S_h.
2. Infected human host, I_h.
3. Recoverd human host, R_h.
4. Susceptible vector, S_v.
5. Infected human vector, I_v.

N_h and N_v denotes the total population of human host and vector populations respectively at a given time t and we have that

$$N_h = S_h + I_h + R_h$$

and that

$$N_v = S_v + I_v.$$

Let us now discuss the dynamics behind the spread of a vector host infection within these two populations. Our human population is recruited into the susceptible compartment at the rate of Λ which means that this population includes both the newborns and the immigrants [36].

Through adequate contact with an infected mosquito a given proportion,

$$\frac{\beta_h b}{N_h + m}$$

of this population will move to the infected class while some of this population will move out of this compartment through a natural death at the rate of μ_h, and prevention against the infection at the rate of α. A natural death is a death that results from all the other causes of death apart from the disease in question.

Once in the infected compartments, the infected and infectious individuals after being put on treatment recover at the rate of γ and goes to the recovered compartment while others suffer either a natural death at the rate of μ_h or a disease induced death at the rate of δ. This explains the population out to the recovered compartment. On recovery, individuals do not get a permanent immunity and hence after sometime they lose their immunity against the infection and move to the susceptible compartment at the rate of ω. Also into the recovery compartment we have those individuals who took prevention measures and hence move directly from the susceptible to the recovered compartments at the rate of α.

We also have that the mosquitoes are recruited at the rate of A into the susceptible compartment and get out of this compartment via natural death at the rate of μ_v or by sufficient contact with an infected human host the mosquitoes move into the infected compartment with a ratio of

$$\frac{\beta_v b}{N_h + m}.$$

The infected mosquitoes only suffer a natural death at a rate of μ_v.

Since we are working with multiple sources of blood for the mosquitoes it is worth explaining that

$$\frac{N_h}{N_h + m}$$

is the probability that a human host is chosen over any other alternative host by a mosquito and this translates to

$$b\frac{N_v}{N_h}\frac{N_h}{N_h+m}$$

bites per unit time.

The proportion

$$b\frac{N_h}{N_h+m}$$

is blood meals from humans per unit time that a vector feeds on. Therefore, the infection rates per susceptible human is given as

$$\frac{\beta_h b I_v}{N_h+m}$$

and for the susceptible vector is given as

$$\frac{\beta_v b I_h}{N_h+m}.$$

Assumptions For us to come up with our model we make the following assumptions

1. The host population is recruited at a given constant rate and that means susceptible populations consists of both newborns and immigrants.
2. The vector population is recruited at a given constant rate which is not dependent on the actual size of the adult population. This is because just a given fraction of the large deposit of eggs eventually mature to larvae. This is independent of the number of adult mosquitoes.
3. All the populations is susceptible to the infection.
4. The vector population is not affected by the infection and does not die from the infection.
5. The infected hosts once recovered only gain a temporary immunity.
6. The mosquitoes do not recover from the disease.
7. There is no latent period and so all the infected populations are able to transmit the disease once they get infected.
8. All the parameters used in the model are all ≥ 0.

As a result of the dynamics of malaria transmission and the assumptions above, we conclude that we are working with a $SIRS-SI$ epidemiological model where $SIRS$ is for our host population and SI represents the vector population. Therefore we have the following flowchart and consequently, the model equations as below.

Flow Chart.

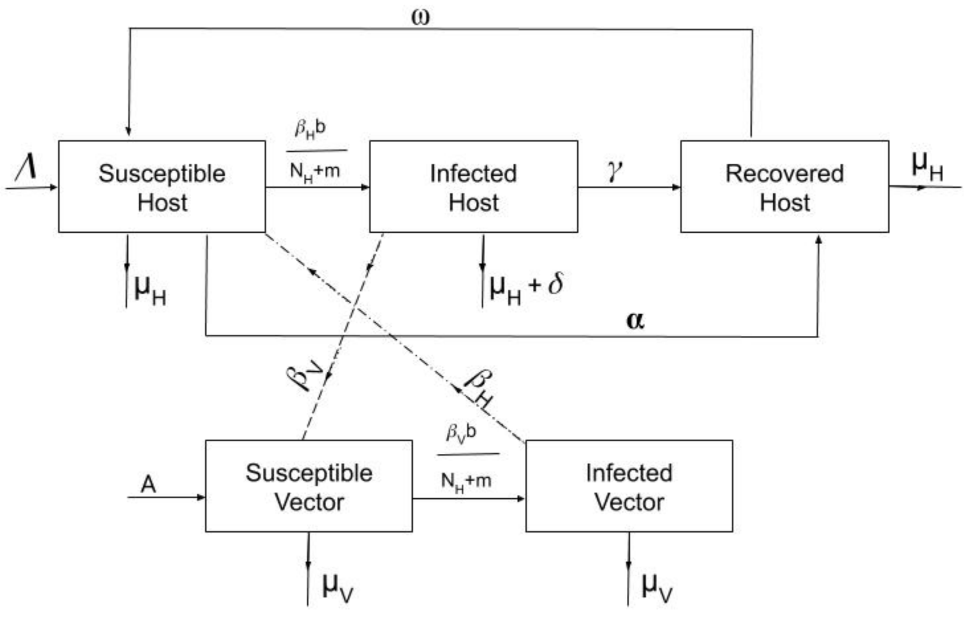

Figure 1: The Compartmental Model of the SIRS-SI Model.

Model equations

$$
\begin{aligned}
S_h' &= \Lambda - \frac{\beta_h b}{N_h+m} S_h I_v - (\mu_h+\alpha)S_h + \omega R_h. \\
I_h' &= \frac{\beta_h b}{N_h+m} S_h I_v - (\mu_h+\gamma+\delta) I_h. \\
R_h' &= \gamma I_h + \alpha S_h - (\mu_h+\omega) R_h. \\
S_v' &= A - \frac{\beta_v b}{N_h+m} S_v I_h - \mu_v S_v. \\
I_v' &= \frac{\beta_v b}{N_h+m} S_v I_h - \mu_v I_v.
\end{aligned} \tag{1}
$$

Let

$$c_1 = \mu_h + \alpha, \quad c_2 = \mu_h + \gamma + \delta \quad \text{and} \quad c_3 = \mu_h + \omega.$$

Then the equations in the system (1) becomes

$$
\begin{aligned}
& S_h' = \Lambda - \frac{\beta_h b}{N_h+m} S_h I_v - c_1 S_h + \omega R_h. \\
& I_h' = \frac{\beta_h b}{N_h+m} S_h I_v - c_2 I_h. \\
& R_h' = \gamma I_h + \alpha S_h - c_3 R_h. \\
& S_v' = A - \frac{\beta_v b}{N_h+m} S_v I_h - \mu_v S_v. \\
& I_v' = \frac{\beta_v b}{N_h+m} S_v I_h - \mu_v I_v.
\end{aligned} \tag{2}
$$

Subject to the initial conditions below

$$S_h(0) \geq 0, \quad I_h(0) \geq 0, \quad R_h(0) \geq 0, \quad S_v(0) \geq 0 \quad \text{and} \quad I_v(0) \geq 0. \tag{3}$$

A table describing the state variables.

Table 1: A table describing the state variables.

N_h	The total human population.
m	The total alternative blood hosts population.
S_h	The total susceptible hosts population.
I_h	The total infected and the infectious hosts population.
R_h	The total recovered human hosts population.
S_v	The total number of the susceptible vectors.
I_v	The total population of the infected and infectious vectors.

A table describing the parameters of the model.

Note that the transmission of the disease from an infected and infectious species to a susceptible species only occurs whenever there is adequate contact between the two species per unit time.

Positivity and Invariant Regions as Basic Properties of Malaria Model

Suppose the initial value $S_h(0) \geq 0, I_h(0) \geq 0, R_h(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0$
then the solutions $(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t))$ of the malaria model 2 are non-negative for all $t > 0$.

Table 2: A table describing the parameters of the model.

Λ	The rate of recruitment of the host population.
β_h	The likelihood of the transfer of an infection from an infectious mosquito to a susceptible host.
β_v	The likelihood of the transfer of an infection from an infectious host to a susceptible mosquito.
b	The rate per day of the mosquito bites per mosquito.
γ	The rate at which an infectious human host recovers from malaria due to treatment.
$\frac{1}{\gamma}$	The average period of infectiousness.
δ	The disease-induced death rate.
ω	The per capita loss of immunity by an initially recovered human host.
$\frac{1}{\omega}$	The period of immunity.
α	The prevention rate of the infection.
μ_h	The host's natural death rate.
μ_v	The vector's natural death rate.
A	The rate of recruitment of the vector population.

Proof. 1. We first seek to show that $S_h(t) \geq 0$.

$$\beta_h b N_h + m S_h I_v - c_1 S_h + \omega R_h$$

$$\frac{dS_h}{dt} = \Lambda - \left(\frac{\beta_h b}{N_h+m} I_v + c_1\right) S_h + \omega R_h$$

$$\frac{dS_h}{dt} + \left(\frac{\beta_h b}{N_h+m} I_v + c_1\right) S_h = \Lambda + \omega R_h. \quad (4)$$

Clearly, 4 can be solved using the Integrating Factor method as shown below. We let

$$\frac{\beta_h b}{N_h+m} I_v + c_1$$

to be represented by y and therefore our integrating factor is $e^{\int y dt} = e^{yt}$.
Multiplying through 4 with e^{yt} we have

$$\frac{dS_h}{dt} e^{yt} + e^{yt} y S_h = e^{yt}(\Lambda + \omega R_h).$$

We then integrate both the right and the left hand side of the above equation to get

$$\begin{aligned} e^{yt} S_h &= (\Lambda + \omega R_h) \int e^{yt} \\ &= (\Lambda + \omega R_h) \frac{e^{yt}}{y}. \end{aligned}$$

Dividing through e^{yt} then we have that

$$S_h = \frac{\Lambda + \omega R_h}{y}. \quad (5)$$

But $y = \frac{\beta_h b}{N_h+m} I_v + c_1$ and so we have that 5 becomes

$$\begin{aligned} S_h &= \frac{\Lambda + \omega R_h}{\frac{\beta_h b}{N_h+m} I_v + c_1} \\ &= \frac{\Lambda + \omega R_h}{\frac{\beta_h b}{N_h+m} I_v + \mu_h + \alpha} \end{aligned}$$

In the same way, we solve for I_h, R_h, S_v and I_v to get that

2. Solving for I_h,

$$I_h = \frac{\beta_h b S_h I_v}{c_2(N_h+m)}$$

but $c_2 = \mu_h + \delta + \gamma$ and so we have that

$$I_h = \frac{\beta_h b S_h I_v}{(\mu_h + \delta + \gamma)(N_h+m)}.$$

3. The solution for R_h,

$$R_h = \frac{\gamma I_h + \alpha S_h}{c_3}$$

but $c_3 = \mu_h + \omega$ and so we have that

$$I_h = \frac{\gamma I_h + \alpha S_h}{\mu_h + \omega}.$$

4. The solution for S_v,

$$S_v = \frac{A}{\frac{\beta_v b I_h}{N_h + m} + \mu_v}.$$

5. Finally, the Solution for I_v,

$$I_v = \frac{\beta_v b S_v I_h}{\mu_v (N_h + m)}.$$

In conclusion, since we know that all our coefficients are non-negative then we have shown that S_h, I_h, R_h, S_v, I_v are all non-negative. □

Given the model 1 with initial conditions that are all non-negative, we say that the region $\Omega = \Omega_h \cap \Omega_v \subset \mathbb{R}_+{}^3 \times \mathbb{R}_+{}^2$ is positively invariant for this model.

Proof. From the model that we have formulated, we want to determine if the host and vector populations actually change with time due to the rate of recruitment of the population. Here we check the behavior of N_h and N_v with regards to time.
We know that the total population of the human host and the vector is given by the relationship below respectively.

$$N_h = S_h + I_h + R_h$$

and

$$N_v = S_v + I_v.$$

This implies that

$$N_h' = S_h' + I_h' + R_h'$$

and

$$N_v' = S_v' + I_v'.$$

Solving for the human host population we have the following

$$\begin{aligned} N_h' &= S_h' + I_h' + R_h' \\ &= \Lambda - \frac{\beta_h b}{N_h + m} S_h I_v - (\mu_h + \alpha) S_h + \omega R_h + \frac{\beta_h b}{N_h + m} S_h I_V \\ &- (\mu_h + \gamma + \delta) I_h + \gamma I_h + \alpha S_h - (\mu_h + \omega) R_h \\ &= \Lambda - \mu_h S_h - \mu_h I_h - \delta I_h - \mu_h R_h \\ &= \Lambda - \mu_h (S_h + I_h + R_h) - \delta I_h. \end{aligned}$$

But $S_h + I_h + R_h = N_h$ and so,

$$N_h' = \Lambda - \mu_h N_h - \delta I_h$$

which is a first order Ordinary Differential Equation.
The above differential equation takes the form of

$$\frac{dN_h}{dt} + p(t) N_h = g(t)$$

whose general solution is found using the integrating factor which is $e^{\mu_h t}$. Multiplying our equation through by $e^{\mu_h t}$ we get

$$\frac{dN_h}{dt} e^{\mu_h t} = \Lambda e^{\mu_h t}. \tag{6}$$

Integrating both sides of 6 with respect to variable t we get our general solution below,

$$N_h(t) = \frac{\Lambda}{\mu_h} + k e^{-\mu_h t}.$$

Where k is a constant.

Taking our limit $t \to \infty$ we have that

$$\lim_{t \to \infty} N_h(t) = \frac{\Lambda}{\mu_h}.$$

Hence we see that the host population varies with time.
We now check for the vector population as below

$$\begin{aligned} N_v' &= S_v' + I_v' \\ &= A - \frac{\beta_v b}{N_h + m} S_v I_h - \mu_v S_v + \frac{\beta_v b}{N_h + m} S_v I_h - \mu_v I_v \\ &= A - \mu_v S_v + - \mu_v I_v \\ &= A - (S_v + I_v)\mu_v. \end{aligned}$$

But $S_v + I_v = N_v$ and so,

$$N_v' = A - \mu_v N_v$$

which is a first order Ordinary Differential Equation.
Using the method of Integrating Factor we solve our Ordinary Differential Equation and later taking the limit $t \to \infty$ then we have that

$$\lim_{t\to\infty} N_v(t) = \frac{\Lambda}{\mu_v}.$$

Again here we conclude that the vector population varies with time. □

Therefore its is clear that

$$\Omega = \{(S_h, I_h, R_h, S_v, I_v) \in \mathbb{R}_+{}^5 : (S_h, I_h, R_h, S_v, I_v) \geq 0; N_h \leq \frac{\Lambda}{\mu_h}, N_v = \frac{\Lambda}{\mu_v}\}$$

is positively invariant and so we conclude that our model is both Biologically and epidemiological meaningful and mathematically well-posed.

3. Equilibrium Points

Equilibrium points are points where our variables do not vary with time such that

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

In epidemiological models we normally have two non-negative equilibrium points which include

1. Disease-Free Equilibrium Point(DFE).
2. Endemic Equilibrium Point(EEP).

1. **Disease-Free Equilibrium Point.**
   In DFE there is no infection within the human population neither is their a pathogen within the vector population. Here we shall denote DFE as E^0. Therefore in this case we expect that the Infected compartments, I_h and I_v will be equal to 0. For the rest of the compartments, to solve for the values of our non-linear system 2 we equate them to 0 as below

$$\begin{aligned} \Lambda - \frac{\beta_h b}{N_h + m} S_h I_v - c_1 S_h + \omega R_h &= 0. \\ \gamma I_h + \alpha S_h - c_3 R_h &= 0. \\ A - \frac{\beta_v b}{N_h + m} S_v I_h - \mu_v S_v &= 0. \end{aligned}$$

   An on solving for S_h, R_h, S_v we find that

$$S_h = \frac{c_3 \Lambda}{c_1 c_3 - \alpha\omega},$$

$$R_h = \frac{\Lambda\alpha}{c_1 c_3 - \alpha\omega},$$

   and

$$S_v = \frac{A}{\mu_v}.$$

   Therefore we deduce that the disease free equilibrium point is

$$\begin{aligned} E^0 &= (S_h^0, I_h^0, R_h^0, S_v^0, I_v^0) \\ &= \left(\frac{c_3\Lambda}{c_1 c_3 - \alpha\omega},\ 0,\ \frac{\Lambda\alpha}{c_1 c_3 - \alpha\omega},\ \frac{A}{\mu_v},\ 0 \right) \\ &= \left(\frac{\Lambda(\mu_h + \omega)}{\mu_h(\mu_h + \omega + \alpha)},\ 0,\ \frac{\Lambda\alpha}{\mu_h(\mu_h + \omega + \alpha)},\ \frac{A}{\mu_v},\ 0 \right). \end{aligned}$$

2. **Endemic Equilibrium Point.**
In EEP the disease persists just within the human population and the pathogens persists within the vector population. Therefore we expect that $(S_h, I_h, R_h, S_v, I_v)$ will be positive values. Here our EEP is denoted as E^*.

Through algebraic manipulations we solve for our system of equations 2 to find that our EEP is

$$E^* = (S_h^*, I_h^*, R_h^*, S_v^*, I_v^*).$$

For

$$w = (\mu_h+\delta+\gamma)\mu_v(N_h+m) \\ ((\mu_h+\delta+\gamma)(\mu_h+\omega)\mu_v(N_h+m)+(\mu_h+\omega)\Lambda b\beta_v-\gamma\mu_v\omega(N_h+m))$$

$$x = b\beta_v((Ab\beta_h((\mu_h+\delta+\gamma)(\mu_h+\omega)-\gamma\omega))+ \\ (\mu_h+\alpha)(\mu_h+\delta+\gamma)(\mu_h+\omega)\mu_v(N_h+m)-(\mu_h+\delta+\gamma)\alpha\mu_v\omega(N_h+m))$$

$$y = A\Lambda b^2\beta_h\beta_v\gamma+(N_h+m)(\mu_h+\delta+\gamma)\mu_v[\mu_v(N_h+m)((\mu_h+\delta+\gamma)\alpha- \\ (\mu_h+\alpha))+\Lambda\alpha b\beta_v]$$

$$z = (N_h+m)(Ab\beta_h((\mu_h+\delta+\gamma)(\mu_h+\omega)-\gamma\omega)+ \\ (\mu_h+\alpha)(\mu_h+\delta+\gamma)(\mu_h+\omega)\mu_v(N_h+m)-(\mu_h+\delta+\gamma)\alpha\mu_v\omega(N_h+m))$$

then we have that the components of E^* are

$$S_h^* = \frac{c_2\mu_v(N_h+m)(c_2c_3N_h\mu_v+c_2c_3m\mu_v+c_3\Lambda b\beta_v-N_h\gamma\mu_v\omega-\gamma m\mu_v\omega)}{b\beta_v(Ac_2c_3b\beta_h-Ab\beta_h\gamma\omega+c_1c_2c_3N_h\mu_v+c_1c_2c_3m\mu_v-c_2N_h\alpha\mu_v\omega-c_2\alpha m\mu_v\omega)} = \frac{w}{x}.$$

$$I_h^* = \frac{Ac_3\Lambda b^2\beta_h\beta_v-c_1c_2c_3\mu_v^2(N_h^2+2N_hm+m^2)+c_2\alpha\mu_v^2\omega(N_h^2+2N_hm+m^2)}{b\beta_v(Ac_2c_3b\beta_h-Ab\beta_h\gamma\omega+c_1c_2c_3N_h\mu_v+c_1c_2c_3m\mu_v-c_2N_h\alpha\mu_v\omega-c_2\alpha m\mu_v\omega)} = \frac{A(\mu_h+\omega)\Lambda b^2\beta_h\beta_v-(\mu_h+\delta+\gamma)\mu_v^2(N_h+m)^2[(\mu_h+\alpha)(\mu_h+\omega)-\alpha\omega]}{x}.$$

$$R_h^* = \frac{A\Lambda b^2\beta_h\beta_v\gamma-c_1c_2\mu_v^2\gamma(N_h+m)^2+c_2^2\mu_v^2\alpha(N_h+m)^2+c_2\mu_v\Lambda\alpha b\beta_v(N_h+m)}{b\beta_v(Ac_2c_3b\beta_h-Ab\beta_h\gamma\omega+c_1c_2c_3N_h\mu_v+c_1c_2c_3m\mu_v-c_2N_h\alpha\mu_v\omega-c_2\alpha m\mu_v\omega)} = \frac{y}{x}.$$

$$S_v^* = \frac{(N_h+m)(Ab\beta_h(c_2c_3-\gamma\omega)+c_2\mu_v(N_h+m)(c_1c_3-\alpha\omega))}{b\beta_h(c_2c_3N_h\mu_v+c_2c_3m\mu_v+c_3\Lambda b\beta_v-N_h\gamma\mu_v\omega-\gamma m\mu_v\omega)} = \frac{z}{b\beta_h((\mu_h+\delta+\gamma)(\mu_h+\omega)\mu_v(N_h+m)+(\mu_h+\omega)\Lambda b\beta_v-\gamma\mu_v\omega(N_h+m))}.$$

$$I_v^* = \frac{Ac_3\Lambda b^2\beta_h\beta_v-c_1c_2c_3\mu_v^2(N_h^2+2N_hm+m^2)+c_2\alpha\mu_v^2\omega(N_h^2+2N_hm+m^2)}{b\beta_h\mu_v((\mu_h+\delta+\gamma)(\mu_h+\omega)\mu_v(N_h+m)+(\mu_h+\omega)\Lambda b\beta_v-\gamma\mu_v\omega(N_h+m))} = \frac{A(\mu_h+\omega)\Lambda b^2\beta_h\beta_v-(\mu_h+\delta+\gamma)\mu_v^2(N_h+m)^2[(\mu_h+\alpha)(\mu_h+\omega)-\alpha\omega]}{b\beta_h\mu_v((\mu_h+\delta+\gamma)(\mu_h+\omega)\mu_v(N_h+m)+(\mu_h+\omega)\Lambda b\beta_v-\gamma\mu_v\omega(N_h+m))}.$$

Basic Reproduction Number

We now find the Basic Reproduction Number which is a threshold for majority of the epidemiological models. The Basic Reproduction Number denoted by R_0 gives us the expected number of secondary infections recorded in case one infected and infectious individual is introduced into a population of hosts where each and every host in that population is susceptible to the disease [36, 37]. R_0 has a rich history dating back to the bubonic plague epidemic of $1905-1906$ that hit Mumbai in India. The development of R_0 is well explained in the article [38].

The R_0 depends on

1. The contact rate in the population of the host.
2. The likelihood of the transmission of the infection during contact.
3. The infectiousness duration once the person has been infected with the disease.

Since we are dealing with a multi-host infection then it suffices that we use the Next Generation Matrix in place of the commonly used Jacobian matrix to finding our R_0. The spectral radius of the Next Generation Matrix gives us our R_0.

If $R_0 \leq 1$ then we expect that the disease will die out.

If $R_0 > 1$ then we expect that the disease will recur into the population.

We consider our population of humans and vectors which is subdivided into m compartments with n infected compartments. Let the total population proportion in the i^{th} compartment be denoted by x_i. Then we define the matrices below

$$\mathbf{F} = \left(\frac{\partial \mathscr{F}_i}{\partial x_j}(E^0)\right)$$

$$\mathbf{V} = \left(\frac{\partial \mathscr{V}_i}{\partial x_j}(E^0)\right)$$

For $i, j = 1, \cdots, m$ then $\mathscr{F}_i$ represents the appearance rate into compartment i of new infections and $\mathscr{V}_i = \mathscr{V}_i^- - \mathscr{V}_i^+$ where $\mathscr{V}_i^-$ represents the transfer rate of individuals or vectors into compartment i by all other means and $\mathscr{V}_i^+$ the transfer rate of individuals or vectors out of compartment i by all other means. The Next Generation Matrix denoted by, G, is given as

$$G = \mathbf{F}\mathbf{V}^{-1}$$

and our Basic Reproduction Number denoted as R_0 is

$$R_0 = \rho(G).$$

Using our model and the above steps we calculate our R_0 as below.
Our infected compartments bear the following equations

$$I_h' = \frac{\beta_h b}{N_h + m} S_h I_v - c_2 I_h.$$

$$I_v' = \frac{\beta_v b}{N_h + m} S_v I_h - \mu_v I_v.$$

and so

$$\mathscr{F}_i = \begin{pmatrix} \frac{\beta_h b}{N_h + m} S_h I_v \\ \frac{\beta_v b}{N_h + m} S_v I_h \end{pmatrix}$$

and

$$\mathscr{V}_i = \begin{pmatrix} c_2 I_h \\ \mu_v I_v \end{pmatrix}.$$

Finding the respective Jacobian matrices of the above matrices with respect to

$$Mat = \begin{pmatrix} I_h \\ I_v \end{pmatrix}$$

we have

$$\mathbf{F} = \begin{pmatrix} 0 & \frac{\beta_h b}{N_h + m} S_h \\ \frac{\beta_v b}{N_h + m} S_v & 0 \end{pmatrix}$$

and

$$\mathbf{V} = \begin{pmatrix} c_2 & 0 \\ 0 & \mu_v \end{pmatrix}.$$

Our Next Generation Matrix is goven as below

$$\begin{aligned} G &= \mathbf{F}\mathbf{V}^{-1} \\ &= \begin{pmatrix} 0 & \frac{\beta_h b}{\mu_v (N_h + m)} S_h \\ \frac{\beta_v b}{c_2 (N_h + m)} S_v & 0 \end{pmatrix}. \end{aligned}$$

On evaluating our G at DFE we have

$$G_{(S_h^0, I_h^0, R_h^0, S_v^0, I_v^0)} = \begin{pmatrix} 0 & \frac{c_3 \Lambda \beta_h b}{\mu_v (N_h + m)(c_1 c_3 - \alpha \omega)} \\ \frac{A \beta_v b}{c_2 \mu_v (N_h + m)} & 0 \end{pmatrix}$$

and its spectral radius is

$$\rho(G_{(S_h^0,I_h^0,R_h^0,S_v^0,I_v^0)}) = b\frac{\sqrt{\frac{Ac_3\Lambda\beta_h}{c_2(c_1c_3-\alpha\omega)}}}{\mu_v(N_h+m)}.$$

Therefore we conclude that our Basic Reproduction Number is

$$R_O = b\frac{\sqrt{\frac{A(\mu_h+\omega)\Lambda\beta_h}{(\mu_h+\delta+\gamma)(\mu_h^2+\mu_h\alpha+\mu_h\omega)}}}{\mu_v(N_h+m)}.$$

Expressing our Endemic equilibrium Point, E^{**}, in terms of our Basic Reproduction Number

We have that

$$R_O = b\frac{\sqrt{\frac{A(\mu_h+\omega)\Lambda\beta_h}{(\mu_h+\delta+\gamma)(\mu_h^2+\mu_h\alpha+\mu_h\omega)}}}{\mu_v(N_h+m)}$$

and so

$$R_O^2 = \frac{Ab^2(\mu_h+\omega)\Lambda\beta_h}{\mu_v^2(N_h+m)^2(\mu_h+\delta+\gamma)(\mu_h^2+\mu_h\alpha+\mu_h\omega)}$$

which can be expressed in terms of c_1, c_2 and c_3 as

$$R_O^2 = \frac{Ab^2(c_3)\Lambda\beta_h}{\mu_v^2(N_h+m)^2c_2(c_1c_3-\alpha\omega)}.$$

From the EEP we first express S_h^* in terms of R_0 as follows.
We have that

$$S_h^* = \frac{c_2\mu_v(N_h+m)(c_2c_3N_h\mu_v+c_2c_3m\mu_v+c_3\Lambda b\beta_v-N_h\gamma\mu_v\omega-\gamma m\mu_v\omega)}{b\beta_v(Ac_2c_3b\beta_h-Ab\beta_h\gamma\omega+c_1c_2c_3N_h\mu_v+c_1c_2c_3m\mu_v-c_2N_h\alpha\mu_v\omega-c_2\alpha m\mu_v\omega)}.$$

Factoring both the numerator and the denominator and multiplying through what we get by $c_3\Lambda$ we have

$$S_h^* = \frac{c_3\Lambda c_2\mu_v^2(N_h+m)^2(c_2c_3-\gamma\omega)+c_3^2\Lambda^2b\beta_vc_2\mu_v(N_h+m)}{Ac_3\Lambda b^2\beta_v\beta_h(c_2c_3-\gamma\omega)+c_3\Lambda b\beta_vc_2\mu_v(N_h+m)(c_1c_3-\alpha\omega)}.$$

Dividing through the numerator and the denominator by $c_2\mu_v^2(N_h+m)^2$ and $c_1c_3-\alpha\omega$ respectively we get

$$S_h^* = \frac{\frac{c_3\Lambda(c_2c_3-\gamma\omega)}{c_1c_3-\alpha\omega}+\frac{c_3^2\Lambda^2b}{c_1c_3-\alpha\omega}}{\frac{Ac_3\Lambda b^2\beta_v\beta_h(c_2c_3-\gamma\omega)}{c_2\mu_v^2(N_h+m)^2}+\frac{c_3\Lambda b\beta_v}{(c_2c_3-\gamma\omega)\mu_v(N_h+m)}},$$

$$S_h^{**} = \frac{\frac{c_3\Lambda(c_2c_3-\gamma\omega)}{c_1c_3-\alpha\omega}+\frac{c_3^2\Lambda^2b}{c_1c_3-\alpha\omega}}{R_0^2(c_2c_3-\gamma\omega)+\frac{c_3\Lambda b\beta_v}{(c_2c_3-\gamma\omega)\mu_v(N_h+m)}}.$$

Which can be reduced to

$$S_h^{**} = \frac{\Lambda\mu_v(N_h+m)[c_1(c_2c_3-\gamma\omega)-c_3^2\Lambda b]}{R_0^2(c_2c_3-\gamma\omega)(c_1c_3-\alpha\omega)\mu_v(N_h+m)+c_3\Lambda b\beta_v}.$$

We solve for I_h^* as below

$$I_h^* = \frac{Ac_3\Lambda b^2\beta_h\beta_v-c_1c_2c_3\mu_v^2(N_h^2+2N_hm+m^2)+c_2\alpha\mu_v^2\omega(N_h^2+2N_hm+m^2)}{b\beta_v(Ac_2c_3b\beta_h-Ab\beta_h\gamma\omega+c_1c_2c_3N_h\mu_v+c_1c_2c_3m\mu_v-c_2N_h\alpha\mu_v\omega-c_2\alpha m\mu_v\omega)}.$$

Factoring the numerator,N, we have

$$\begin{aligned}
N &= Ac_3\Lambda b^2\beta_h\beta_v-c_1c_2c_3\mu_v^2(N_h^2+2N_hm+m^2)+c_2\alpha\mu_v^2\omega(N_h^2+2N_hm+m^2)\\
&= -c_1c_2c_3\mu_v^2(N_h+m)^2(N_h+m)^2+c_2\alpha\mu_v^2\omega(N_h+m)^2+Ac_3\Lambda b^2\beta_h\beta_v\\
&= 1+\frac{Ab^2(\mu_h+\omega)\Lambda\beta_h}{\mu_v^2(N_h+m)^2(\mu_h+\delta+\gamma)(\mu_h^2+\mu_h\alpha+\mu_h\omega)}\\
&= R_0^2-1.
\end{aligned}$$

So, I_h^{**} is given as

$$I_h^{**} = \frac{R_0^2-1}{b\beta_v(Ac_2c_3b\beta_h-Ab\beta_h\gamma\omega+c_1c_2c_3N_h\mu_v+c_1c_2c_3m\mu_v-c_2N_h\alpha\mu_v\omega-c_2\alpha m\mu_v\omega)}.$$

We now work on expressing R_h^* in terms of R_0.

We first factorize both the numerator and the denominator then we multiply through by $\frac{c_3}{c_1c_3-\alpha\omega}$. We then divide through both the numerator and the denominator by $c_2\mu_v^2(N_H+m)^2$ to get.

$$R_h^* = \frac{\frac{b^2Ac_3\Lambda\beta_h\beta_v\gamma}{(c_1c_3-\alpha\omega)c_2\mu_v^2(N_h+m)^2} + \frac{c_3(c_1\gamma-c_2\alpha)}{c_1c_3-\alpha\omega} + \frac{c_2\Lambda\alpha b\beta_v}{(c_1c_3-\alpha\omega)\mu_v(N_h+m)}}{\frac{c_3Ab^2\beta_v\beta_h(c_2c_3-\gamma\omega)}{(c_1c_3-\alpha\omega)c_2\mu_v^2(N_h+m)^2} + \frac{c_3b\beta_v}{\mu_v(N_h+m)}}.$$

This is then reduced to

$$R_h^{**} = \frac{\mu_v(N_h+m)c_2[R_0^2\gamma\mu_v(N_h+m)(c_1c_3-\alpha\omega) - \mu_v(N_h+m)c_3(c_1\gamma-c_2\alpha) + \Lambda\alpha b\beta_v c_3]}{c_3b\beta_v[Ab\beta_h(c_2c_3-\gamma\omega) + c_2(c_1c_3-\alpha\omega)\mu_v(N_h+m)]}.$$

We now express S_v^* in terms of R_0 as below.
We have that

$$S_v^* = \frac{(N_h+m)\,(Ab\beta_h(c_2c_3-\gamma\omega) + c_2\mu_v(N_h+m)(c_1c_3-\alpha\omega))}{b\beta_h\,(c_2c_3N_h\mu_v + c_2c_3m\mu_v + c_3\Lambda b\beta_v - N_h\gamma\mu_v\omega - \gamma m\mu_v\omega)}.$$

Factorizing both the numerator and the denominator and multiplying through by $A\mu_v$ we get

$$S_v^* = \frac{Ac_2\mu_v^2(N_h+m)^2(c_1c_3-\alpha\omega)\left[\frac{Ab\beta_h(c_2c_3-\gamma\omega)}{c_2\mu_v(N_h+m)(c_1c_3-\alpha\omega)} + 1\right]}{Ab\beta_h(N_h+m)\mu_v^2(c_2c_3-\gamma\omega) + Ac_3\Lambda b^2\beta_v\beta_h\mu_v}.$$

This is then reduced to

$$S_v^* * = \frac{A\left[\frac{Ab\beta_h(c_2c_3-\gamma\omega)}{c_2\mu_v(N_h+m)(c_1c_3-\alpha\omega)} + 1\right]}{\frac{Ab\beta_h(c_2c_3-\gamma\omega)}{c_2(N_h+m)(c_1c_3-\alpha\omega)}} + R_0^2\mu_v.$$

And therefore we have that

$$S_v^{**} = \frac{A[Ab\beta_h(c_2c_3-\gamma\omega) + c_2\mu_v(N_h+m)(c_1c_3-\alpha\omega)]}{Ab\beta_h\mu_v(c_2c_3+\omega) + R_0^2\mu_v c_2(N_h+m)(c_1c_3-\alpha\omega)}.$$

Since the numerator of I_v^* is the same as the numerator of I_h^* then expressing I_v^* in terms of R_0 yields

$$I_v^{**} = \frac{R_0^2-1}{b\beta_h\mu_v\,(c_2c_3N_h\mu_v + c_2c_3m\mu_v + c_3\Lambda b\beta_v - N_h\gamma\mu_v\omega - \gamma m\mu_v\omega)}.$$

Therefore we have expressed the elements of the Endemic equilibrium Point,E^{**}, in terms of our Basic Reproduction Number as the following.

$$E^{**} = (S_h^{**}, I_h^{**}, R_h^{**}, S_v^{**}, I_v^{**}).$$

Where

$$S_h^{**} = \frac{\Lambda\mu_v(N_h+m)[c_1(c_2c_3-\gamma\omega) - c_3^2\Lambda b]}{R_0^2(c_2c_3-\gamma\omega)(c_1c_3-\alpha\omega)\mu_v(N_h+m) + c_3\Lambda b\beta_v}.$$

$$I_h^{**} = \frac{R_0^2-1}{b\beta_v\,(Ac_2c_3b\beta_h - Ab\beta_h\gamma\omega + c_1c_2c_3N_h\mu_v + c_1c_2c_3m\mu_v - c_2N_h\alpha\mu_v\omega - c_2\alpha m\mu_v\omega)}.$$

$$R_h^{**} = \frac{\mu_v(N_h+m)c_2[R_0^2\gamma\mu_v(N_h+m)(c_1c_3-\alpha\omega) - \mu_v(N_h+m)c_3(c_1\gamma-c_2\alpha) + \Lambda\alpha b\beta_v c_3]}{c_3b\beta_v[Ab\beta_h(c_2c_3-\gamma\omega) + c_2(c_1c_3-\alpha\omega)\mu_v(N_h+m)]}.$$

$$S_v^{**} = \frac{A[Ab\beta_h(c_2c_3-\gamma\omega) + c_2\mu_v(N_h+m)(c_1c_3-\alpha\omega)]}{Ab\beta_h\mu_v(c_2c_3+\omega) + R_0^2\mu_v c_2(N_h+m)(c_1c_3-\alpha\omega)}.$$

$$I_v^{**} = \frac{R_0^2-1}{b\beta_h\mu_v\,(c_2c_3N_h\mu_v + c_2c_3m\mu_v + c_3\Lambda b\beta_v - N_h\gamma\mu_v\omega - \gamma m\mu_v\omega)}.$$

3.1. Stability Analysis and Sensitivity Analysis

3.1.1. Stability Analysis.

We first define the following two critical terms.

Local Stability of an equilibrium point implies that in case you put the system anywhere nearby that point then in some time the system will move itself to the equilibrium point.

Global Stability means that the system will come to the equilibrium point when put anywhere within the domain of the system and not necessarily near the point. When a point is globally stable then it implies that it is locally stable but the vice versa is not true.

Theorem 3: The disease-free equilibrium point for system 2 is locally asymptotically stable if $R_0 < 1$. For us to study if our Disease Free Equilibrium Point is stable then we employ Lyapunov's Indirect method for local stability.

Here we evaluate the Jacobian matrix of our system 2 at Disease free equilibrium point and then we find the eigenvalues to determine if the points are stable or not.

DECISION RULE:

1. If the eigenvalues or the real part of the eigenvalues are all < 0 then we say that the equilibrium point is Locally Asymptotically Stable.
2. Else, we say that the point is unstable.

NOTE: Any value under a root such as square root, cube root ecetera when solved is assumed to be > 0 and that c_1, c_2 and c_3 are all positive.

On doing the above, we find that we have the following eigenvalues.

1.
$$-\frac{c_1}{2} - \frac{c_3}{2} - \frac{\sqrt{c_1^2 - 2c_1c_3 + c_3^2 + 4\alpha\omega}}{2}.$$

2.
$$-\frac{c_1}{2} - \frac{c_3}{2} + \frac{\sqrt{c_1^2 - 2c_1c_3 + c_3^2 + 4\alpha\omega}}{2}.$$

3.
$$-\mu_v.$$

4.
$$-\frac{c_1c_2c_3\mu_v(N_h+m) + c_1c_3\mu_v^2(N_h+m) - c_2\alpha\mu_v\omega(N_h+m) - \alpha\mu_v^2\omega(N_h+m) - \sqrt{x}}{2c_1c_3N_h\mu_v + 2c_1c_3m\mu_v - 2N_h\alpha\mu_v\omega - 2\alpha m\mu_v\omega}.$$

5.
$$-\frac{c_1c_2C_3\mu_v(N_h+m) + c_1c_3\mu_v^2(N_h+m) - c_2\alpha\mu_v\omega(N_h+m) - \alpha\mu_v^2\omega(N_h+m) + \sqrt{x}}{2c_1c_3N_h\mu_v + 2c_1c_3m\mu_v - 2N_h\alpha\mu_v\omega - 2\alpha m\mu_v\omega}.$$

Where

$$\begin{gathered}
x = 4Ac_1c_3^2\Lambda b^2\beta_h\beta_v\mu_v - 4Ac_3\Lambda\alpha b^2\beta_h\beta_v\mu_v\omega + c_1^2c_2^2c_3^2N_h^2\mu_v^2 + 2c_1^2c_2^2c_3^2N_hm\mu_v^2 + c_1^2c_2^2c_3^2m^2\mu_v^2 \\
-2c_1^2c_2c_3^2N_h^2\mu_v^3 - 4c_1^2c_2c_3^2N_hm\mu_v^3 - 2c_1^2c_2c_3^2m^2\mu_v^3 + c_1^2c_3^2N_h^2\mu_v^4 + 2c_1^2c_3^2N_hm\mu_v^4 + c_1^2c_3^2m^2\mu_v^4 \\
-2c_1c_2^2c_3N_h^2\alpha\mu_v^2\omega - 4c_1c_2^2c_3N_h\alpha m\mu_v^2\omega - 2c_1c_2^2c_3\alpha m^2\mu_v^2\omega + 4c_1c_2c_3N_h^2\alpha\mu_v^3\omega \\
+8c_1c_2c_3N_h\alpha m\mu_v^3\omega + 4c_1c_2c_3\alpha m^2\mu_v^3\omega - 2c_1c_3N_h^2\alpha\mu_v^4\omega - 4c_1c_3N_h\alpha m\mu_v^4\omega \\
-2c_1c_3\alpha m^2\mu_v^4\omega + c_2^2N_h^2\alpha^2\mu_v^2\omega^2 + 2c_2^2N_h\alpha^2m\mu_v^2\omega^2 + c_2^2\alpha^2m^2\mu_v^2\omega^2 \\
-2c_2N_h^2\alpha^2\mu_v^3\omega^2 - 4c_2N_h\alpha^2m\mu_v^3\omega^2 - 2c_2\alpha^2m^2\mu_v^3\omega^2 + N_h^2\alpha^2\mu_v^4\omega^2 + 2N_h\alpha^2m\mu_v^4\omega^2 + \alpha^2m^2\mu_v^4\omega^2.
\end{gathered}$$

Clearly, we can observe that all the eigenvalues above are all < 0 apart from 2). which is shown below that it is also < 0.

We have that $c_1 = \mu_h + \alpha$ and $c_3 = \mu_h + \omega$ and so replacing above into

$$-\frac{c_1}{2} - \frac{c_3}{2} + \frac{\sqrt{c_1^2 - 2c_1c_3 + c_3^2 + 4\alpha\omega}}{2}$$

we have

$$\frac{-2\mu_h - \alpha - \omega}{2} + \frac{\sqrt{(\mu_h+\alpha)^2 - 2(\mu_h+\alpha)(\mu_h+\omega) + (\mu_h+\omega)^2 + 4\alpha\omega}}{2} = -\mu_h. \tag{7}$$

Therefore we conclude that the Disease Free Equilibrium point is Locally Asymptotically Stable.

Theorem 4: If $R_0 < 1$ the DFE point is globally asymptotically stable in Ω and if $R_0 > 1$ then the DFE is unstable in Ω.

Proof. We only consider the infected compartments. Let

$$V(S_h, I_h, R_h, S_v, I_v) = I_h + I_v = 0$$

be our Lyapunov function.

Then when $R_0 < 0$, $V(E^0) = 0$. Finding the derivative of $V(.)$ with respect to time we get

$$\begin{aligned}
\dot{V} &= \dot{I_h} + \dot{v} \\
&= \frac{\beta_h b}{N_h + m} S_h I_v - (\mu_h + \gamma + \delta) I_h \\
&+ \frac{\beta_v b}{N_h + m} S_v I_h - \mu_v I_v
\end{aligned}$$

But remember that $\gamma = \delta = 0$ and so we have

$$\begin{aligned}
&< \frac{\beta_h b}{N_h + m} S_h I_v - \mu_h I_h + \frac{\beta_v b}{N_h + m} S_v I_h - \mu_v I_v \\
&< (\frac{\beta_h b}{N_h + m} S_h - \mu_v) I_v + (\frac{\beta_v b}{N_h + m} S_v - \mu_h) I_h.
\end{aligned}$$

Therefore we make the assumptions that, if

$$\frac{\beta_h b}{N_h + m} S_h \leq \mu_v$$

and

$$\frac{\beta_v b}{N_h+m}S_v \leq \mu_h$$

then

$$\dot{V} \leq 0.$$

In conclusion, we say that the DFE is Globally Asymptotically Stable by Lyapunov Asymptotic Stability theorem since V is positive definite and $\dot{V}$ is negative definite. □

If $R_0 > 1$ then the Endemic Equilibrium point of our model (2) is globally asymptotically stable.

Proof. Let us consider a Lyapunov function candidate $V(S_h, I_h, R_h, S_v, I_v) : \mathbb{R}^5 \to \mathbb{R}^+$ defined as

$$V = \frac{1}{2}(S_h - S_h^*)^2 + \frac{1}{2}(I_h - I_h^*)^2 + \frac{1}{2}(R_h - R_h^*)^2 + \frac{1}{2}(S_v - S_v^*)^2 + \frac{1}{2}(I_v - I_v^*)^2$$

where $S_h^*, I_h^*, R_h^*, S_v^*, I_v^*$ is the endemic equilibrium state.

We differentiate our function V to end up with

$$\dot{V} = (S_h - S_h^*)\dot{S}_h + (I_h - I_h^*)\dot{I}_h + (R_h - R_h^*)\dot{R}_h + (S_v - S_v^*)\dot{S}_v + (I_v - I_v^*)\dot{I}_v.$$

Setting the following conditions from our system of equation (2)

$$\begin{aligned}
\Lambda &= \frac{\beta_h b}{N_h+m}S_h I_v + c_1 S_h - \omega R_h. \\
\frac{\beta_h b}{N_h+m}S_h I_v &= c_2 I_h. \\
\gamma I_h + \alpha S_h &= c_3 R_h. \\
A &= \frac{\beta_v b}{N_h+m}S_v I_h + \mu_v S_v. \\
\frac{\beta_v b}{N_h+m}S_v I_h &= \mu_v I_v. \\
\Lambda - \delta I_h &= \mu_h N_h. \\
A &= \mu_v N_v.
\end{aligned}$$

And imposing them on our $\dot{V}$, we have

$$\begin{aligned}
\dot{V} = (S_h - S_h^*)[\frac{\beta_h b}{N_h+m}S_h^* I_v^* + c_1 S_h^* - \omega R_h^* - \frac{\beta_h b}{N_h+m}S_h I_v + c_1 S_h - \omega R_h] \\
+(I_h - I_h^*)[c_2 I_h^* - c_2 I_h] + (R_h - R_h^*)[c_3 R_h^* - c_3 R_h] + (S_v - S_v^*)[\frac{\beta_v b}{N_h+m}S_v^* I_h^* + \\
\mu_v S_v^* - \frac{\beta_v b}{N_h+m}S_v I_h + \mu_v S_v] + (I_v - I_v^*)[\mu_v I_v^* - \mu_v I_v] + (N_h - N_h^*)[\mu_h N_h^* - \mu_h N_h] \\
+(N_v^* - N_v)[\mu_v N_v^* - \mu_v N_v] \\
= (S_h - S_h^*)[S_h^*(\frac{\beta_h b}{N_h+m}I_v^* + c_1) - S_h(\frac{\beta_h b}{N_h+m}I_v + c_1)] - (I_h - I_h^*)^2 c_2 - (R_h - R_h^*)^2 c_3 - \\
(S_v - S_v^*)[S_v^*(\frac{\beta_v b}{N_h+m}I_h^* + \mu_v) - S_v(\frac{\beta_v b}{N_h+m}I_h + \mu_v)] - (I_v - I_v^*)^2 \mu_v \\
-(N_h - N_h^*)^2 \mu_h - (N_v^* - N_v)^2 \mu_v.
\end{aligned}$$

Next we make the assumptions below

1. If $I_v^* < I_v$ then $S_h^*(\frac{\beta_h b}{N_h+m}I_v^* + c_1) - S_h(\frac{\beta_h b}{N_h+m}I_v + c_1)$ and $(I_v - I_v^*)^2 \mu_v$ are both < 0.
2. If $I_h^* < I_h$ then $S_v^*(\frac{\beta_v b}{N_h+m}I_h^* + \mu_v) - S_v(\frac{\beta_v b}{N_h+m}I_h + \mu_v)$ and $(I_h - I_h^*)^2 c_2$ are both < 0.
3. Since $(R_h - R_h^*)^2, (N_h - N_h^*)^2$ and $(N_v - N_v^*)^2$ is always ≥ 0 and $c_3, \mu_h, \mu_v > 0$ then we will make $-(R_h - R_h^*)^2 c_3, -(N_h - N_h^*)^2 \mu_h$ and $-(N_v^* - N_v)^2 \mu_v$ take 0.

Therefore provided the above assumptions hold then $\dot{V}$ is negative definite and since V is positive definite then we say that the function V is a Lyapunov function for our system 1 and we conclude that E^* is globally asymptotically stable by Lyapunov asymptotic stability theorem.

Since E* is globally asymptotically stable then it is in order to state that it is also locally asymptotically stable. □

3.1.2. Sensitivity Analysis.

We perform sensitivity analysis of the Basic Reproduction number and this tells us how important each parameter is to the transmission of the disease. This analysis helps us to to determine which parameters should be considered by intervention strategies [39].

Here we use the normalized forward sensitivity index,ε, of a variable with respect to a given parameter which is defined as the ratio of the relative change in the variable to that in the parameter. A parameter that is not sensitive does not produce a significant change in the variable and thus is not needed to be estimated [40].

We shall analyze the sensitivity of the parameters: biting rate, prevention rate and recovery rate after treatment on our variable, R_0. On evaluating the we find the following sensitivity indices.

$$\begin{aligned}
\varepsilon_b^{R_0} &= 1, \\
\varepsilon_\alpha^{R_0} &= \frac{-b(A\Lambda\beta_h\mu_h(\mu_h+\omega))^{\frac{1}{2}}}{\mu_v(N_h+m)(\mu_h^2+\omega\mu_h+\alpha\mu_h)^{\frac{3}{2}}(\mu_h+\delta+\gamma)^{\frac{3}{2}}}, \\
\varepsilon_\gamma^{R_0} &= \frac{-b(A\Lambda\beta_h\mu_h(\mu_h+\omega))^{\frac{1}{2}}}{\mu_v(N_h+m)(\mu_h^2+\omega\mu_h+\alpha\mu_h)^{\frac{1}{2}}(\mu_h+\delta+\gamma)^{\frac{3}{2}}},
\end{aligned}$$

From the above we can see that mosquito biting rate affects our R_0 most.

3.2. Optimal Control Model Application

Dengue fever is transmitted to humans by mosquitoes which carry the infection without getting affected by the infection themselves. Recently, dengue fever has become a great public health hazard and it is endemic in most countries all around the world.

Flavivirus is the virus that causes dengue fever. There exist four different serotypes of flavivirus. In dengue-endemic areas, a person can be infected with more than one of these serotypes and this does not guarantee them lifelong immunity instead it only confers one with long-term protection against infection by the particular serotype one got infected with [41]. In a paper by Xiao and Tang [42], it was shown that vaccination alone can lead to a further spread of a disease rather than leading the community to a disease-free situation. It is also shown that reducing the basic reproduction number, R_0, to a value that is less than one is not sufficient to bring about the elimination of a disease.

In this chapter, we wish to come up with an optimal control model for the dengue fever disease to come up with the optimal control strategies that present the minimal implementation cost then we apply Pontryagin's Maximum Principle [43] to select the optimal parameters that will minimize the Cost function. Our focus is on reducing the number of the mosquito population and the infected humans while keeping in check the cost that comes along with these interventions.

Our controls shall comprise of the following:

1. Prevention measures including but not limited to vaccines, use of Insecticide-treated Nets, use of Indoor Residual Spraying, outdoor spraying, community education and awareness and Larval Source Management [44].
2. Treatment efforts. This is through the use of the recommended chemoprophylaxis.

3.2.1. Analysis of Optimal Control of a Vector-Host Model.

Let $u_1(t)$ to represent the ratio of the susceptible individuals that take preventive measures per unit time and $u_2(t)$ to represent ratio of the infected individuals treated for a vector-host disease per unit time. Then our model equation 1 becomes

$$\begin{aligned}
S_h' &= \Lambda - \frac{\beta_h b}{N_h+m}S_hI_v - (\mu_h+u_1(t))S_h + \omega R_h. \\
I_h' &= \frac{\beta_h b}{N_h+m}S_hI_v - (\mu_h+\delta+u_2(t))I_h. \\
R_h' &= u_2(t)I_h + u_1(t)S_h - (\mu_h+\omega)R_h. \\
S_v' &= A - \frac{\beta_v b}{N_h+m}S_vI_h - \mu_vS_v. \\
I_v' &= \frac{\beta_v b}{N_h+m}S_vI_h - \mu_vI_v.
\end{aligned} \tag{8}$$

Then 8 is the state system with initial conditions

$$S_h(0) \geq 0, I_h(0) \geq 0, R_h(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0.$$

Now, where we have control variables that are bounded and measured then, we consider the following control variables $u(t) = (u_1(t), u_2(t)) \in U$ with regards to the state variables $S_h(t), I_h(t), R_h(t), S_v(t), I_v(t)$ where U is a lebesgue measurable as shown by (Jung et al.,2002; Yan & Zou et al, 2008) on $[0,1], 0 \leq u_i(t) \leq u_{imax}(t) \leq 1, t \in [0,T], i = 1,2$.

We define an objective function as

$$J(u_1(t),u_2(t)) = \int_0^T (A_1I_v(t)+A_2I_h(t)) + \frac{1}{2}(B_1u_1^2(t)+B_2u_2^2(t))dt \quad \text{subject to}$$

$$\dot{S}_h(t),\dot{I}_h(t),\dot{R}_h(t),\dot{S}_v(t),\dot{I}_v(t) \quad \text{such that} \quad 3 \quad \text{holds.}$$

Where A_1,A_2 are weights constants and B_i are weights constants for our controls respectively.

A linear function is chosen for the cost experienced by $A_1I_v(t)$ and $A_2I_h(t)$ while a quadratic function is chosen for the controls since there is no linear relationship between interventions and their cost.

Therefore, we look for an optimal control $u_1^*(t),u_2^*(t)$ such that $J(u_1^*(t),u_2^*(t)) = \min_{u_i(t)\in U} J(u_1(t),u_2(t))$.

We assume that the controls have the following property.

If $u(t)$ defined on $t_0 \le t \le t_1$ is in U where U is a set of pairwise continuous function and for $i = 1,\cdots,p, V_i \in U$ and $\tau_i - \Delta_i < t \le \tau_i$ are non-overlapping intervals intersecting $[t_0,t_1]$ then

$$\bar{u}(t) = \begin{cases} V_i & ;\tau_i - \Delta_i < t \le \tau_i. \\ u(t) & ;t \in [t_0,t_1]\text{and} \notin \text{one of the intervals } \tau_i - \Delta_i < t \le \tau_i. \end{cases}$$

is in U.

3.2.2. Statement of Maximum Pontryagin's Principle.

For a set of equations that are differentiable and with minimization side conditions, the conditions of Maximum Pontryagin's Principle help reduce the computations required for an optimal control solution to a two-point boundary problem [43].

Definition: The necessary conditions that $(x^*(t),u^*(t))$ be an optimal initial condition for the optimal control problem are the existence of a non-zero k-dimensional vector λ with $\lambda_1 \le 1$ and an n-dimensional vector function $\lambda(t)$ such that for $t \in [t_0,t_1]$ then

$$\dot{\lambda}(t)^T = -\lambda(t)^T f_x(t,x^*(t),u^*(t)) \quad \text{for} \quad t \in [t_0,t_1]$$

and

$$\lambda(t)^T[f(t,x^*(t),u(t)) - f(t,x^*(t),u^*(t))] \quad \le \quad 0 \tag{9}$$

Let $H(t,x(t),u(t),t) = \lambda(t)^T f(t,x(t),u(t))$ and so $H(t,x(t),u(t),t)$ is a Hamiltonian which helps at the solving of the adjoint variable. Therefore 9 can be written as

$$\max_{u(t)\in U} \{H(t,x^*(t),u(t),t)\} = H(t,x^*(t),u^*(t),t)$$

and is called **Pontryagin's Maximum Principle.**

3.2.3. Existence of a solution.

A solution exists if

1. $\exists$ a non-empty state variable and control set. Since our coefficients are all bonded then this holds.
2. We have a closed and convex control set U. Our solutions are bounded and thus this holds.
3. The state system equations on their right hand side are bounded, continuous and can be expressed as a linear function of u where the coefficients are time and state-dependent. This is true since our solution is bounded.
4. $\exists$ constants $k_1,k_2 > 0$ and $\rho > 0$ in such a way that the integrand, $L(x(t),u(t),t)$ of the objective function, J is convex and satisfies

$$L(x(t),u(t),t) \ge k_1(|u_1(t)|^2 + |u_2(t)|^2)^{\frac{\rho}{2}} - k_2.$$

This holds since the integrand of J is convex.

Theorem 6: Given the objective function

$$J(u_1(t),u_2(t)) = \int_0^T (A_1I_v(t)+A_2I_h(t)) + \frac{1}{2}(B_1u_1^2(t)+B_2u_2^2(t))dt \quad \text{for}$$

$$U = \{u_1(t),u_2(t) \quad \text{such that} \quad 0 \le u_1(t) \le u_{1max}(t) \le 1, 0 \le u_2(t) \le u_{2max}(t) \le 1, t \in [0,T], i = 1,2\}$$

where $u_{1max}(t)$ and $u_{2max}(t)$ are the respective maximum values of $u_1(t)$ and $u_2(t)$ that can be attained subject to (4) with initial conditions (3) then $\exists$ an optimal control

$$u^*(t) = (u_1^*(t),u_2^*(t))$$

such that

$$J(u_1^*(t),u_2^*(t)) = min_{u(t)}J(u_1(t),u_2(t)).$$

Proof. We first find the Lagrangian,L, of the optimal problem which is defined as

$$L(I_v(t), I_h(t), u_1(t), u_2(t)) = A_1 I_v(t) + A_2 I_h(t) + \frac{1}{2}(B_1 u_1^2(t) + B_2 u_2^2(t)).$$

This helps us find the first order necessary optimality conditions for optimal solutions.

We then define the Hamiltonian,H, for the control problem since we aim at finding the minimal value of L. Let

$$\begin{aligned} x(t) &= (S_h, I_h, R_h, S_v, I_v), \\ \lambda(t) &= (\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t), \lambda_5(t)), \end{aligned}$$

where $(\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t), \lambda_5(t))$ is the adjoint variable then

$$H(x(t), u(t), \lambda(t), t) = L(I_v(t), I_h(t), u_1(t), u_2(t)) + \lambda_1(t)\dot{S}_h(t) + \lambda_2(t)\dot{I}_h(t) + \lambda_3(t)\dot{R}_h(t)$$
$$+\lambda_4(t)\dot{S}_v(t) + \lambda_5(t)\dot{I}_v(t).$$

Theorem 7: Given the optimal controls $u_1^*(t), u_2^*(t)$ and solutions $S_h^*(t), I_h^*(t), R_h^*(t), S_v^*(t), I_v^*(t)$ subject to the initial conditions of the corresponding state system 8, there exists adjoint variables $\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t), \lambda_5(t)$ satisfying

$$\frac{d\lambda_1}{dt} = \frac{-\partial H}{\partial S_h}, \frac{d\lambda_2}{dt} = \frac{-\partial H}{\partial I_h}, \frac{d\lambda_3}{dt} = \frac{-\partial H}{\partial R_h}, \frac{d\lambda_4}{dt} = \frac{-\partial H}{\partial S_v}, \frac{d\lambda_5}{dt} = \frac{-\partial H}{\partial I_v}$$

with transversality or boundary conditions

$$\lambda_1(T) = \lambda_2(T) = \lambda_3(T) = \lambda_4(T) = \lambda_5(T) = 0.$$

Proof. We have our state system together with its initial conditions as

$$\begin{aligned} S_h' = \frac{-\partial H}{\partial \lambda_1} &= \Lambda - \frac{\beta_h b}{N_h + m} S_h I_v - (\mu_h + u_1(t))S_h + \omega R_h, \quad S_h(0) \geq 0. \\ I_h' = \frac{-\partial H}{\partial \lambda_2} &= \frac{\beta_h b}{N_h + m} S_h I_v - (\mu_h + \delta + u_2(t))I_h, \quad I_h(0) \geq 0. \\ R_h' = \frac{-\partial H}{\partial \lambda_3} &= u_2(t)I_h + u_1(t)S_h - (\mu_h + \omega)R_h, \quad R_h(0) \geq 0. \\ S_v' = \frac{-\partial H}{\partial \lambda_4} &= A - \frac{\beta_v b}{N_h + m} S_v I_h - \mu_v S_v, \quad S_v(0) \geq 0. \\ I_v' = \frac{-\partial H}{\partial \lambda_5} &= \frac{\beta_v b}{N_h + m} S_v I_h - \mu_v I_v, \quad I_v(0) \geq 0. \end{aligned} \tag{10}$$

Then

$$H(x(t), u(t), \lambda(t)) = L(S_h, I_h, R_h, S_v, I_v, u_1, u_2) + \lambda_1 \dot{S}_h + \lambda_2 \dot{I}_h + \lambda_3 \dot{R}_h + \lambda_4 \dot{S}_v + \lambda_5 \dot{I}_v$$
$$= (A_1 I_v + A_2 I_h) + \frac{1}{2}(B_1 u_1^2 + B_2 u_2^2) + \lambda_1(\Lambda - \frac{\beta_h b}{N_h + m} S_h I_v - (\mu_h + u_1)S_h + \omega R_h)$$
$$+\lambda_2(\frac{\beta_h b}{N_h + m} S_h I_v - (\mu_h + \delta + u_2)I_h) + \lambda_3(u_2 I_h + u_1 S_h - (\mu_h + \omega)R_h) +$$
$$\lambda_4(A - \frac{\beta_v b}{N_h + m} S_v I_h - \mu_v S_v) + \lambda_5(\frac{\beta_v b}{N_h + m} S_v I_h - \mu_v I_v).$$

The co-state equations together with their transversality conditions are ,

$$\begin{aligned} \frac{d\lambda_1}{dt} = \frac{-\partial H}{\partial S_h} &= -(\lambda_2 - \lambda_1)\frac{\beta_h b}{N_h + m} I_v + (\mu_h + u_1)\lambda_1 - \lambda_3 u_1, \quad \lambda_1(T) = 0. \\ \frac{d\lambda_2}{dt} = \frac{-\partial H}{\partial I_h} &= -A_2 + (\delta + u_2 + \mu_h)\lambda_2 - u_2\lambda_3 - (\lambda_5 - \lambda_4)\frac{\beta_v b}{N_h + m} S_v, \quad \lambda_2(T) = 0. \\ \frac{d\lambda_3}{dt} = \frac{-\partial H}{\partial R_h} &= -\lambda_1\omega + (\mu_h + \omega)\lambda_3, \quad \lambda_3(T) = 0. \\ \frac{d\lambda_4}{dt} = \frac{-\partial H}{\partial S_v} &= \mu_v\lambda_4 - (\lambda_5 - \lambda_4)\frac{\beta_v b}{N_h + m} I_h, \quad \lambda_4(T) = 0. \\ \frac{d\lambda_5}{dt} = \frac{-\partial H}{\partial I_v} &= -(\lambda_2 - \lambda_1)\frac{\beta_h b}{N_h + m} S_h + \mu_v\lambda_5, \quad \lambda_5(T) = 0. \end{aligned} \tag{11}$$

□

The optimality condition as described by [45] is given as

$$\frac{\partial H}{\partial u_1} = \frac{\partial H}{\partial u_2} = 0$$

and so we have that

$$\begin{aligned} \frac{\partial H}{\partial u_1} = B_1 u_1^* + (\lambda_3 - \lambda_1) S_h' &= 0. \\ \frac{\partial H}{\partial u_2} = B_2 u_2^* + (\lambda_3 - \lambda_2) I_h' &= 0. \end{aligned}$$

From the above equations then we have that

$$\begin{aligned} u_1^* &= \frac{S_h'(\lambda_1 - \lambda_3)}{B_1}. \\ u_2^* &= \frac{I_h'(\lambda_2 - \lambda_3)}{B_2}. \end{aligned}$$

The maximality condition ensures that the extremal control is given by

$$u_i^* = \min\{\max(0, u_i), u_{imax}\}.$$

When we apply the boundary condition of each control and so we have

$$\begin{aligned} u_1^* &= \min\left\{\max\left(0, \frac{S_h'(\lambda_1 - \lambda_3)}{B_1}\right), u_{1max}\right\}. \\ u_2^* &= \min\left\{\max\left(0, \frac{I_h'(\lambda_2 - \lambda_3)}{B_2}\right), u_{2max}\right\}. \end{aligned} \tag{12}$$

□

3.3. The Composition of the Optimality System

The first order necessary condtions are given by

1. The state System 10 with its initial conditions.
2. The adjoint or co-state system 11 together with its boundary conditions.
3. The characterization of the optimal control as given by 12
   The optimality system is then solved using the forward-backward sweep method in OCTAVE. The method is such that we solve the state equations and the co-state equations forward in time and backward in time respectively and at the same time. The process occurs repeatedly until a convergence is achieved.

4. Results and Discussion

4.1. Parameters values Simulation

The values for our parameters, initial conditions and weights were chosen such that the population does not go to extinction at any given time and also such that our value reflects that the disease is endemic to the population.

The following values were extracted from [1].

$$N_v = 700000, \mu_h = 0.0000391\backslash day, A = 50000 \beta_h = 0.5 \quad \text{and} \quad \beta_v = 0.3.$$

We estimated the following values

$$N_h = 100000, \delta = 0.0089\backslash day, m = 70000, \omega = \frac{1}{180}\backslash day \quad \text{and} \quad b = 0.4.$$

From the above values of the total population of the hosts and the vectors, it is clear that we are dealing with a limiting population.
We then find that $\Lambda = 3.91\backslash day$ and $\mu_v = 0.0714\backslash day$ through calculations.
The following weights were also adopted

$$A_1 = 1, A_2 = 1, B_1 = 0.4 \quad \text{and} \quad B_2 = 0.6.$$

We tried out various combinations of u_{1max} and u_{2max} which include

1. $(u_{1max}, u_{2max}) = (0, 0)$.
2. $(u_{1max}, u_{2max}) = (0.2, 0.2)$.
3. $(u_{1max}, u_{2max}) = (0.4, 0.4)$.
4. $(u_{1max}, u_{2max}) = (0.55, 0.55)$.

4.2. simulation process

We now check how the human host population and the vector population behaves when we have the above different values of controls.

4.2.1. when $(u_{1max}, u_{2max}) = (0.00, 0.00)$

When we neither have treatment nor prevention for the dengue disease in the population, then we notice that the population of the infected human host will continue rising as most of those in the susceptible class move into the infected compartment as shown in figure 2. We also observe in figure 3, that the population of the infected vector increases to a vector population that is 70 times the size of the initial population of the infected class of the vectors and in figure 5 the susceptible population decreases to 0 at $t = 40$.

4.2.2. when $(u_{1max}, u_{2max}) = (0.20, 0.20)$

It is observed in figure 2, the infected host population rises during the first 10 days and gradually levels down to 0 at T while in figure 5 susceptible population decreases continuously from its initial value to 0 at time T. We observe that the recovered population also monotonically increases to a population of 100 000 which represents the total population as shown in figure 4. while the infected vector population increases to a value that is greater than $I_h(0)$ before it decreases to 0 at T. The susceptible vector population also decreases to 0 at time T.

4.2.3. when $(u_{1max}, u_{2max}) = (0.40, 0.40)$

In this scenario, the figure 2 shows that the infected host population continuously decreases from $t = 0$ and gets to 0 when $t = 20$ while the recovered population gets to its maximum at $t = 15$ as shown in figure 4 and the susceptible host population gets to its minimum at $t = 15$. as indicated in figure 5. Studying the graph of the vector population, we notice that the infected vector population first rises to 1900 and starts decreasing to 0 at $t = 10$. This infected population gets to 0 at T. Also, the susceptible vector population decreases to 0 monotonically at T.

4.2.4. when $(u_{1max}, u_{2max}) = (0.55, 0.55)$

Using these values for our control then we notice that our infected host population decreases to 0 at $t = 12$ as shown in figure 2 and 3. Total population recovery is achieved at $t = 10$ and this is also when we have no susceptible population. For the infected vector population, we notice that this population increases to 1600 and starts decreasing to 0 from time $t = 5$. The susceptible vector population as shown in figure 5 at the last graph also decreases to 0 at T.

It is evident that whenever the values of u_{1max}, u_{2max} are increased then the disease becomes easily controlled by eliminating all the infected human hosts and vectors within a shorter period. However, it is typically difficult for a government to afford the use of high values of controls. Therefore, based on our controls we can see that at $(u_{1max}, u_{2max}) = (0.55, 0.55)$ the dengue disease is easily controlled and the infection is eliminated within the shortest time than when we used the other values for (u_{1max}, u_{2max}).

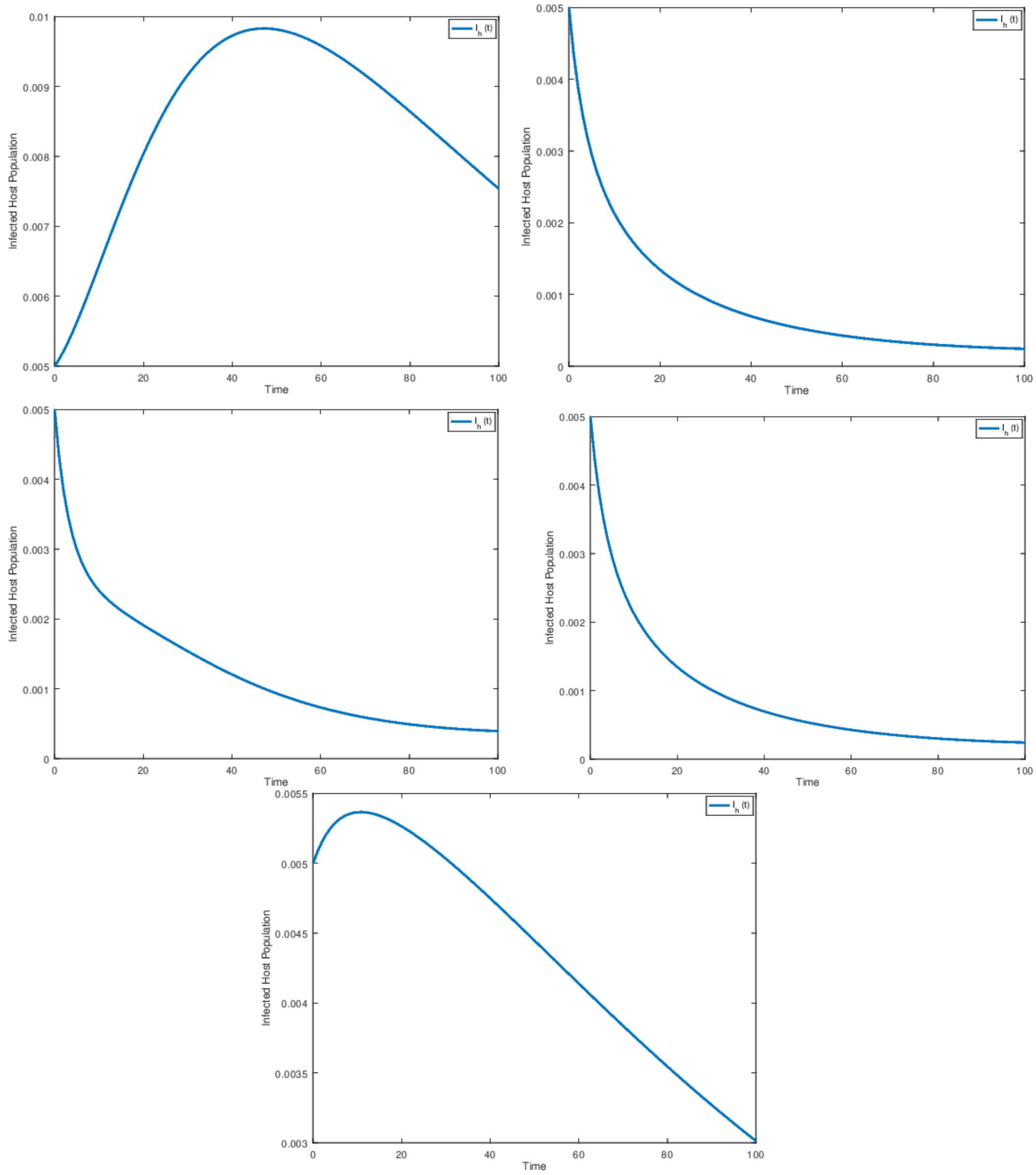

Figure 2: Infected Host Population

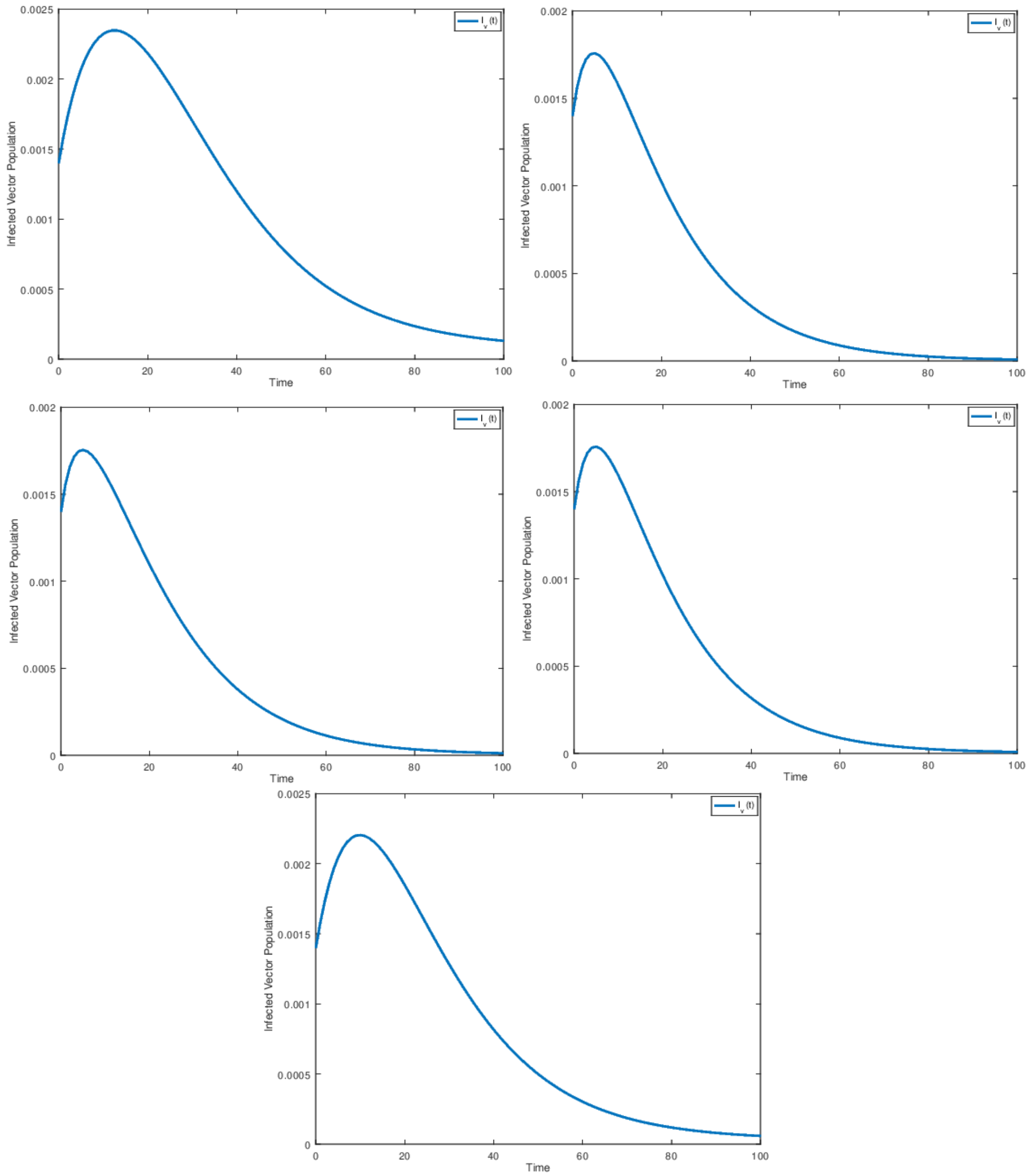

Figure 3: Infected Vector Population

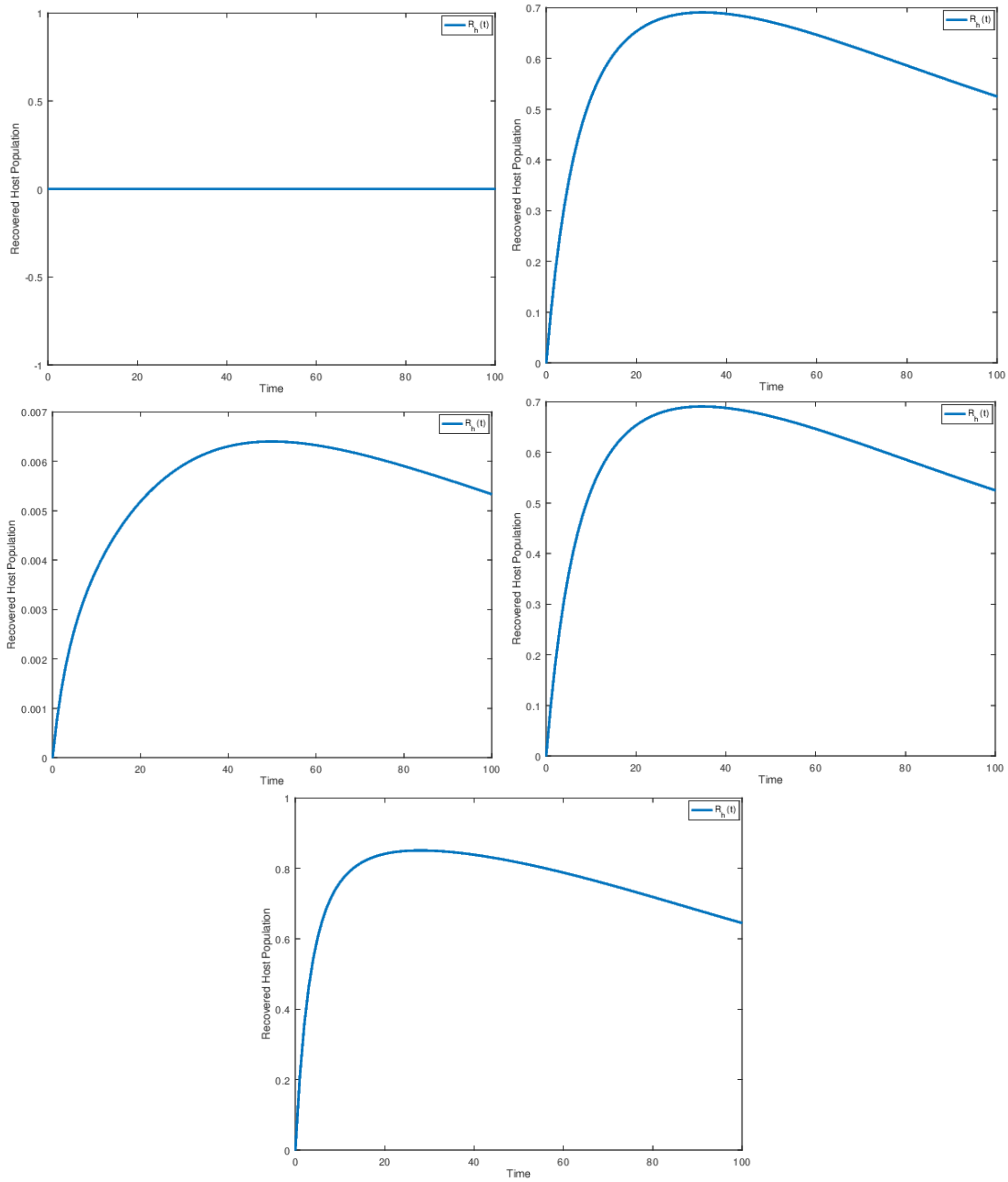

Figure 4: Recovery Host Population

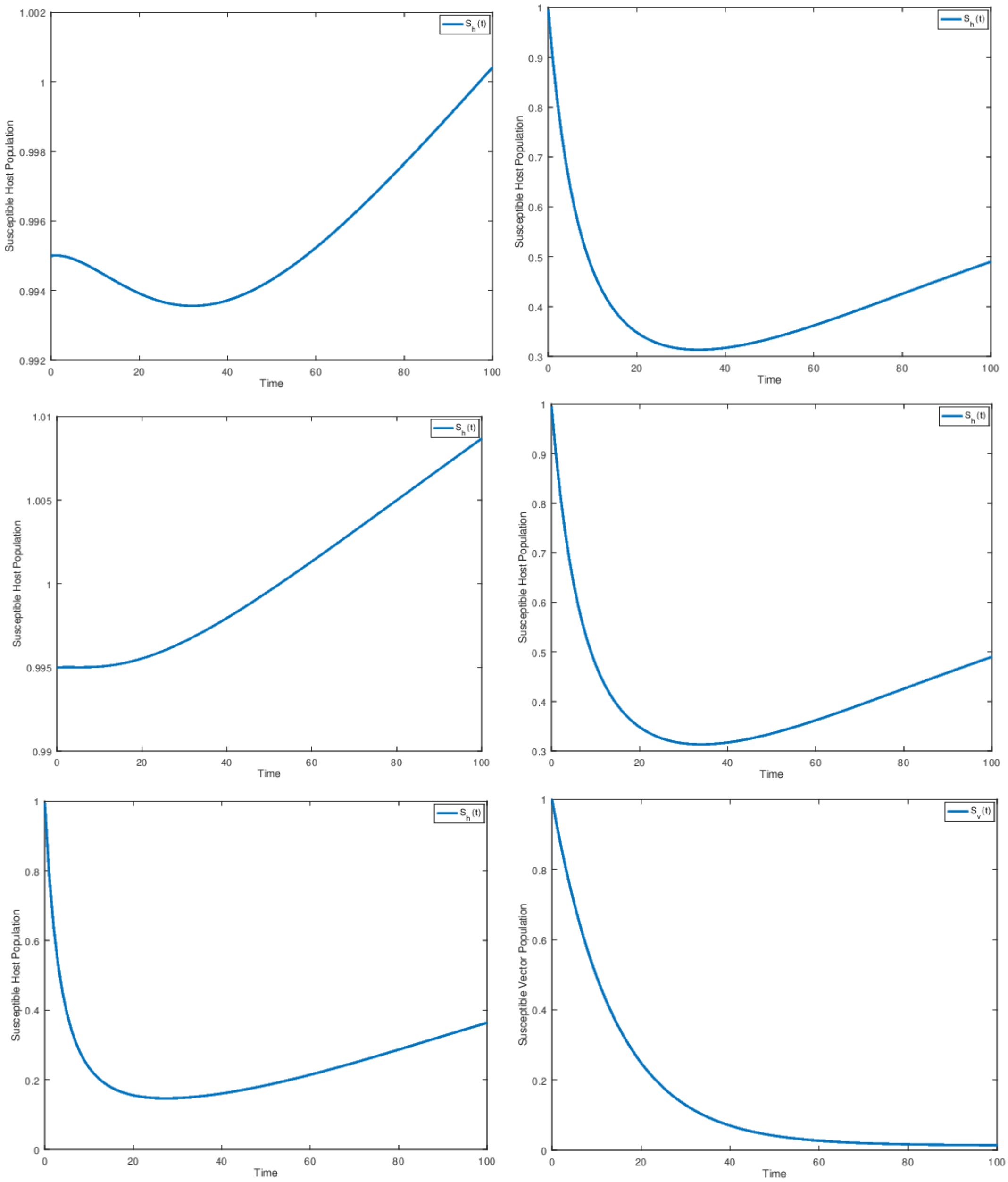

Figure 5: Susceptible Host Population

5. Conclusion

In this paper, we first came up with a model that could best generalize vector host diseases including malaria, West Nile Fever, Dengue Fever, and Rift Valley Fever. We went further to check if our model was epidemiological mathematically meaningful and well-posed before we studied the stability of the system's equilibrium points.

Sensitivity analysis was also conducted and this led us to the study of the optimal control of the system. Here, we employed two controls which included treatment and prevention. Applying Pontryagin's maximum principle we were able to come up with the first-order necessary conditions to be used for optimization.

We then used OCTAVE to do the numerical solutions and to obtain our results. Here we gave the same weights to the infected host and infected vector populations and also the two controls were given the same weight when it comes to the cost of implementing treatment and prevention strategies.

The results proved that we can eliminate dengue fever from a population and this will only occur if efficient strategies are taken into consideration. This also holds for the various vector-host diseases that are endemic within our communities. These strategies will be effective if and only if we channel sufficient resources into them.

Funding
The research got funding from the Pan African University Institute for Basic Sciences, Technology and Innovation

Conflict of Interest
The authors declare that they have no conflicts of interest related to this research. All funding sources supporting this study are acknowledged, and no financial or personal relationships with organizations that could potentially influence the work have been disclosed.

Data Availability
The data generated and analyzed during the current study are available from the corresponding author upon reasonable request.

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